

COMPRESSIVE NEUROPATHIES AND TENDINOPATHIES IN THE ATHLETIC ELBOW AND WRIST

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Optimal function of the upper extremity is vital to success in most sporting events. The time and effort put forth in training and conditioning the upper extremities for athletic performance can become a double-edged sword. The benefits of rigorous training or recreational sports frequently are negated by the occurrence of an overuse injury.

Overuse is defined as the level of repetitive microtrauma sufficient to overwhelm the tissues' ability to adapt.⁹⁶ This microtrauma leads to persistent, and at times disabling, injuries for the athlete. These injuries commonly involve prolonged symptoms and recovery time. Most of these injuries heal with conservative treatment but require many athletes to consider an unacceptable period of inactivity, thus encouraging all sports enthusiasts to train wisely to avoid overuse injuries.

This article reviews the applied anatomy, pathomechanics, and the relationship to athletics of compressive neuropathies and tendinopathies of the upper extremity. Treatment and preventative rehabilitation programs are discussed. The time lost to the athlete and cost to our work force make it imperative for the physician charged with the care of these patients to be aware of the wide range of possible injuries related to overuse.

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COMPRESSIVE NEUROPATHIES

Compressive neuropathies can be subtle and are often overlooked in the athletic upper extremity. The incidence of peripheral nerve injury in the athlete differs from that in the general population.⁵⁴ Nerve injury may result from a single, violent force or from repetitive stress.³ Mechanisms of injury most commonly involve repetitive compression, contusion, or traction. Anatomic structures, such as anomalous muscles or vessels, fibrous bands, osteofibrous tunnels, or simple muscular hypertrophy, as well as pathologic structures including ganglia, lipomas, osteophytes, aneurysms, and local inflammation of any type, all can lead to a compressive neuropathy.^{96, 116}

The degree of injury in compressive neuropathy may vary. The two most common classification schemes used to describe these injuries were devised by Seddon¹¹⁴ and Sunderland.¹²⁵ Seddon's classification (the most commonly used) combines clinical and pathologic findings. Seddon attempted to classify nerve injuries into three types: (1) neurapraxia, (2) axonotmesis, and (3) neurotmesis. Few lesions, however, fit exclusively into one category.

Neurapraxia is the mildest form of a nerve injury and implies a decrease in function in the presence of anatomic continuity. A loss of conduction along the course of the nerve is caused by a loss of axon excitability or segmental demyelination. Prognosis for complete recovery is good, and improvement may be swift depending on duration of injury and the need for remyelination.⁷⁶ This lesion is the most common nerve lesion in the athletic population.¹¹⁶

Axonotmesis implies axonal injury and distal degeneration with the alignment and the connective tissue supporting the structure of the nerve intact. Axonotmesis represents a more chronic form of injury. Prognosis for recovery is good, but the timing depends on the amount of axonal regeneration required from the injury site to the end organ.⁷⁶

Neurotmesis, less common in the athlete and usually associated with a more severe injury, represents a complete disruption in the structure of the nerve. Recovery is usually slower, often less complete, and depends on anatomic reapposition of the separated nerve segments.⁷⁶

The pathophysiology of the compressive neuropathy remains an area of active investigation.⁹⁶ Attention has focused recently on the ischemia induced by compression. Lundborg has shown that endoneurial pressure increases after acute compression and that this increase in pressure may lead to venous congestion, relative ischemia, increased vascular permeability, and a change in local ionic composition, which may alter conduction.⁷⁹ Patients with hypertension have been found to have a lower incidence of compressive neuropathies, giving support for this theory.²⁰ Rapid reversal of symptoms and improvement in nerve conduction following operative decompression also are cited frequently as support for the theory of ischemia-induced compressive neuropathy.^{79, 96, 127} The importance of the pathophysiology of nerve compression is in its relation to

diagnosis and treatment. Delays in diagnosis may allow treatable lesions to become irreversible. Chronic compression can cause demyelination followed by axonal loss and, finally, intraneural fibrosis. The large, myelinated fibers and peripheral fibers are most susceptible, but at 1 year, all fascicles are affected equally.^{80, 96}

Radial Nerve

The radial nerve is a continuation of the posterior cord of the brachial plexus and receives contributions from spinal roots C5–C8 (Fig. 1). The first branches occur in the axilla and upper arm, including the posterior cutaneous nerve and the motor supply to the triceps and anconeus. The nerve travels in the spiral groove of the humerus, then passes beneath the lateral head of the triceps, and penetrates the lateral intermuscular septum traveling anteriorly. Cutaneous branches to the lateral arm and posterior forearm and nerves to the brachioradialis and extensor carpi radialis longus (ECRL) arise proximal to the lateral epicondyle, whereas the branch to the extensor carpi radialis brevis (ECRB) arises at or just beyond the epicondyle. The radial nerve bifurcates near the radiocapitellar joint to become the posterior interosseous and superficial radial nerve. The posterior interosseous nerve continues between the two heads of the supinator, around the proximal radius, and under the forearm extensors to supply the terminal articular branches to

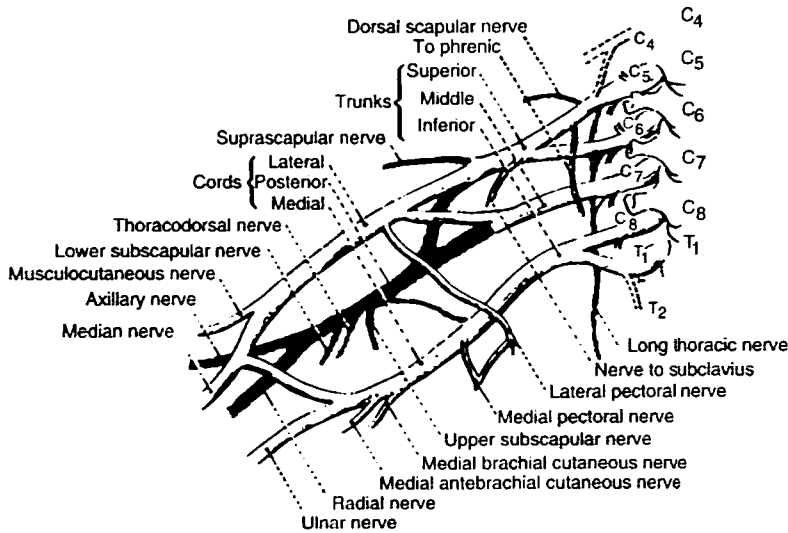


Figure 1. Anatomy of the brachial plexus. (From Rockwood CA, Matson FA III: The Shoulder. Philadelphia, WB Saunders, 1990, p 68.)

the wrist. The superficial radial nerve travels underneath the brachioradialis to become subcutaneous in the distal third of the forearm (three branches) to supply sensation to the dorsoradial portion of the hand, including the first web space and the proximal phalanges of the first three digits.

The radial nerve is susceptible to a compressive neuropathy at three levels. Above the elbow, the radial nerve may be compressed in the spiral groove or as it passes from the lateral border of the triceps through the lateral intermuscular septum. It may be compressed at the level of the elbow, producing either a radial tunnel syndrome or posterior interosseous syndrome. Compression of either the superficial radial nerve or the distal posterior interosseous nerve may occur at the wrist.

High Radial Nerve Palsy

Injury to the nerve above the elbow is rare and usually secondary to trauma, humeral fractures, crutch use, tourniquet use, "Saturday night palsy," and other sources of traction or torsional injury.³⁴ Nontraumatic compression is rare and usually is related to strenuous activity.³⁴ Compression or injury in the spiral groove may be seen with activities that require forceful adduction of the shoulder, as seen in gymnastics⁷⁶ and wrestling.⁸³ Compression may occur at the fibrous arcade at the origin of the lateral head of the triceps⁷⁸ within the triceps,⁸³ or below the lateral intermuscular septum.³⁴ Activities that require forceful extension of the elbow against resistance, such as throwing (e.g., discus, javelin, baseball) and weightlifting, are associated with a high radial nerve entrapment.^{18, 87, 116} "Runner's radial palsy," an unusual and distinct entity that presents with numbness in the distal forearm and dorsum of the hand of runners who keep their elbow acutely flexed while running, is another example of a high radial nerve lesion. The nerve in this injury is compressed between the humerus and the triceps. Treatment is an alteration of the running position, resulting in complete resolution of symptoms.⁹⁵

A mixed motor and sensory involvement is seen in the high radial nerve palsy. The triceps may or may not be involved depending on the level of the lesion. Conservative treatment often will result in complete recovery, although the time required varies.³⁴ Surgical exploration of the radial nerve is indicated only if there is a persistent paralysis or electrodiagnostic evidence of denervation.^{34, 87}

Radial Tunnel Syndrome

The most common compressive neuropathy of the radial nerve occurs at the radial tunnel. There are five sites of compression in the radial tunnel³⁹: (1) the fibrous bands lying anterior to the radial head at the entrance to the tunnel; (2) the group of vessels known as the leash of Henry that supply the brachioradialis and ECRL; (3) the ulnar half of the tendon and fascia of the ECRB; (4) under the arcade of Fröhse, the

fibrous arch at the proximal edge of the supinator; and (5) below the fascial arcade, at the distal lateral border of the supinator (Fig. 2).¹²²

The radial tunnel syndrome (RTS) often can be confused with recalcitrant lateral epicondylitis. These authors believe that they represent separate entities, although this judgment is controversial. They both may be related as a by-product of local inflammation but almost always can be distinguished with a meticulous physical examination. Heyse-Moore⁵⁶ thought that the mechanism of these two pathologic conditions may be related to the blended origin of the ECRB and supinator from the lateral epicondyle, elbow capsule, and orbicular ligament. Heyse-Moore noted that dividing the arcade of Fröhse would relieve tension at the lateral epicondyle and that the treatment of lateral epicondylitis with ECRB lengthening could decompress the radial tunnel at the same time. Because the two muscles are essentially inseparable, division of the arcade of Fröhse would relieve tension at the lateral epicondyle, but ECRB lengthening would not relieve the nerve compression. These authors and others¹³² could not find any objective evidence of radial nerve entrapment.

Roles and Maudsley¹¹⁰ thought that RTS mimics and may coexist with lateral epicondylitis in up to 10% of patients. Werner¹³⁷ found an 8% coincident lesion in 203 patients. Although some still feel there is a

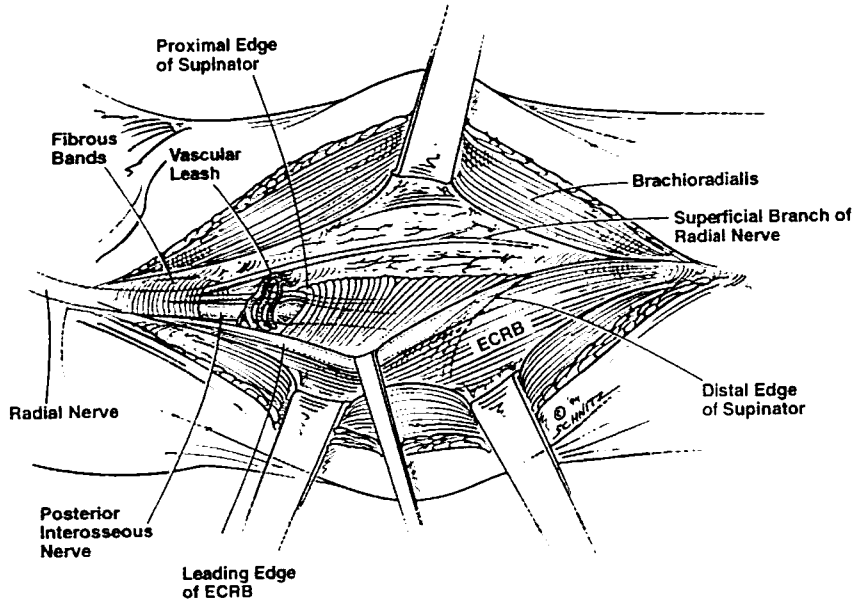


Figure 2. Anatomy of the radial tunnel and common compression site of the radial nerve in the proximal forearm. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

lack of empiric data against this relationship, most authors now recognize the importance of the radial nerve entrapment syndrome.¹⁰⁹ Considering the senior author's (JBS) experience with 200 patients over 15 years, these authors believe this syndrome to be a separate entity with real pathologic indications found at operation.

RTS may be caused by compression or contusion. A single strenuous muscular effort alone may result in transient deficits. The syndrome is seen often in those athletes who repetitively pronate and supinate and in those who are stressed in wrist extension, as in weightlifting, bowling, rowing, discus, racquet sports, swimming, and golf.^{18, 102, 116}

Clinically, this entity must be differentiated from lateral epicondylitis, wrist extensor tendinitis, stenosis of the orbicular ligament, fibrillations of the radial head, and extensor tendon ruptures.¹³⁶ The patient with RTS often will complain of soreness and aching just distal to the lateral epicondyle over the extensor muscle mass. This chronic, deep ache is common at night. There is no true sensory involvement, although the nerve does supply nociceptive fibers to the wrist and recurrent fibers to the radiocapitellar joint. Motor weakness is uncommon. It generally is thought that a patient with RTS complains of a dull ache, whereas patients with lateral epicondylitis complain of sharp or knifelike pains.³⁴

It is critical to determine the exact location of pain. The patient with RTS classically shows tenderness directly over the extensor muscle mass approximately four fingerbreadths distal to the lateral epicondyle. Pain with resisted extension of the middle finger with the elbow in extension is a helpful physical sign. The nerve in this test is irritated by the tendinous edge of the ECRB. The patient with RTS often exhibits pain with resisted supination of the extended forearm, made worse with wrist flexion.

Treatment consists of rest, anti-inflammatory medication, and splinting for 3 to 6 months. If there is no improvement, surgical decompression may be performed on the radial tunnel (Figs. 3 and 4).¹⁴² Results vary widely with good to excellent reports from 51% to 80% of patients.³⁴ Studies have shown men are affected twice as often as women. The dominant hand also is involved twice as often as the nondominant.³⁴ This wide variety of success rates may be a product of the uncertainty of the diagnosis or the chronicity of the process.

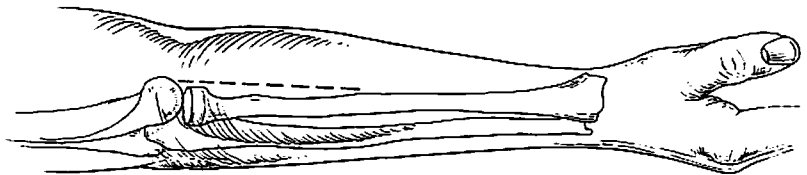


Figure 3. Incision used by the authors to release radial tunnel. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

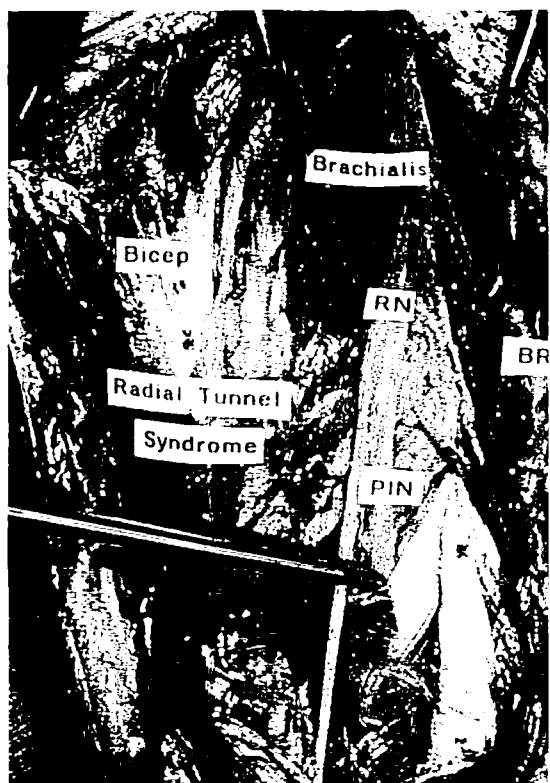


Figure 4. Anatomic probe in the arcade of Fröhse prior to radial tunnel release. RN = radial nerve; PIN = posterior interosseous nerve; BR = brachioradialis. (Courtesy of Kevin D. Plancher, MD, MS.)

Posterior Interosseous Nerve Syndrome (PINS)

There is some confusion in the literature regarding the difference between this entity and RTS. Most think that these two syndromes represent two ends of a spectrum with overlapping anatomic features and symptoms.¹²⁰ The main difference is in the clinical presentation. The patient with PINS has weakness in wrist extension and finger and thumb extension with no sensory changes, whereas in RTS, pain is the pre-eminent symptom and weakness plays a lesser role. The difference may reflect a longitudinal variation in intraneural organization of proprioceptive and motor fibers or a differential response to crushing versus ischemic mechanisms.¹²⁰ The compression of the nerve in PINS is under the fibrous arch at the proximal edge of the supinator (the arcade of Fröhse). In addition to this site, the syndrome may be caused by anatomic abnormalities, such as a bifid ECRB or vascular anomalies; synovitis; edema; or structural lesions, such as lipoma, ganglion, or bursa.^{34, 89, 120}

The compression may be secondary to overuse of the extensor musculature or from repetitive pronation and supination. The space-occupying lesion more commonly causes PINS than RTS.

Anatomic studies have shown the proximal supinator to be membranous in 70% and fibrous in 30% of cadavers at the arcade of Fröhse.¹¹⁷ This structure is not seen in a full-term fetus, suggesting that it is developmental and dependent on use. This structure in patients with refractory PINS is found in 80% of those taken to the operating room.¹³⁶ Treatment is identical to that for RTS.

Distal Posterior Interosseous Nerve Syndrome

This syndrome is a compression and irritation of the deep terminal sensory branch of the posterior interosseous nerve as it passes dorsally over the distal radius and enters the wrist capsule.¹⁹ Athletes subjected to repetitive and forceful wrist dorsiflexion, especially gymnasts,⁷³ are prone to this injury.

Clinically, the patient presents with a deep, dull ache in the wrist and may have reproduction of the pain with forceful wrist extension or deep palpation of the forearm with the wrist in flexion. This syndrome is a diagnosis of exclusion, and the examiner should look for masses and clicking or snapping on wrist motion, and should perform provocative tests for carpal instability. The differential diagnosis includes occult fracture, carpal instability, ganglions, wrist sprains, and radial carpal impaction syndrome.³⁴

Radiographs may reveal static carpal instability. Anesthetic injections can be helpful diagnostically as well as provide some relief if combined with a steroid. Surgical excision of the nerve under the extensor digitorum communis at the distal wrist is indicated if conservative therapy is ineffective. The nerve is exposed on the radial aspect of the floor of the fourth extensor compartment, and a segment of the nerve is excised.²⁶

Superficial Radial Nerve Syndrome

Cheiralgia paresthetica,¹³⁴ or Wartenberg's syndrome, is a compression or irritation of the superficial radial nerve. The nerve is susceptible to injury as it pierces the deep fascia to become subcutaneous between the tendons of the ECRL and brachioradialis. Dellon et al²⁷ think the nerve is tethered causing a "scissoring" effect as it becomes superficial. This tight construction is made worse by sports that include pronation and supination,²⁷ such as batting, throwing, and rowing.⁷⁶ In nonathletic endeavors, this compression can occur from the so-called handcuff neuropathy.^{30, 84} In sports, this equivalent may be seen with tight wristbands, tape, watches, archery guards, gloves, or the straps of a racquetball racquet.^{76, 116} Direct trauma also may be responsible for this syndrome in contact sports, such as hockey, football, and lacrosse.

The athlete often notes numbness or paresthesias over the dorsoradial aspect of the wrist, hand, dorsal thumb, and index finger. There

may be pain in the area of the wrist with flexion of the thumb and ulnar deviation of the wrist, but this is unusual and may represent concurrent de Quervain tenosynovitis. Eaton and Lister³⁴ stress that the distinguishing characteristic of Wartenberg's syndrome is that, when performing a Finkelstein test, and carefully observing the thumb position, the pain is present regardless of the position of the thumb.

Typically the Finkelstein test is negative, but a Tinel's sign over the wrist is positive. The athlete denies any pain with motion of the wrist, which rules out a tendinitis-, arthritis-, or impingement-type syndrome. The sensory changes must be separated from those involving the lateral antebrachial cutaneous nerve of the proximal forearm, which may show some overlapping innervation in this area.³¹ Dellon²⁷ has suggested that when lidocaine injections are used diagnostically the lateral antebrachial cutaneous nerve be injected first to help confirm the diagnosis.

Electrodiagnostic findings are inconsistent but will aid in the diagnosis if a decrease in sensory nerve action potentials or altered distal latencies can be demonstrated.¹³⁶ Conservative therapy consists of rest, ice, nonsteroidal anti-inflammatory medication, splinting, padding, and avoidance of any irritating devices that may be worn on the wrist or distal forearm. Surgical treatment may be considered if there is no improvement after 6 to 12 months, although Dellon²⁷ has shown that after 18 months surgical treatment offers little benefit. Surgery involves an extensive release of the deep fascia along the course of the superficial radial nerve (Fig. 5). Results have been 37% excellent and 45% good, with 43% able to return to manual work.²⁷

Median Nerve

The median nerve is derived from the spinal roots of C6–T1 arising from both medial and lateral cords of the brachial plexus (see Fig. 1). The nerve travels along the medial side of the upper arm, accompanying the brachial artery to the level of the elbow. The nerve lies medial to the artery in the cubital fossa and passes under the lacertus fibrosis and distally between the two heads of the pronator teres. At the distal margin of the pronator teres, the nerve divides to form the anterior interosseous nerve to supply the flexor pollicis longus, the flexor digitorum superficialis to the index and middle digits, and the pronator quadratus. The anterior interosseous nerve has small articular branches to several intercarpal joints of the wrist. The main trunk of the median nerve continues distally beneath the fibrous arch of the flexor digitorum superficialis. The palmar cutaneous branch of the median nerve, which supplies sensation to the volar wrist, thenar eminence, and palm, arises approximately 5 cm proximal to the radial styloid and continues distally, ulnar to the flexor carpi radialis to pass superficially through the flexor retinaculum. The median nerve continues through the carpal tunnel beneath the flexor retinaculum. The nerve supplies sensation to the palm and radial three and one half digits. The deep motor branch typically

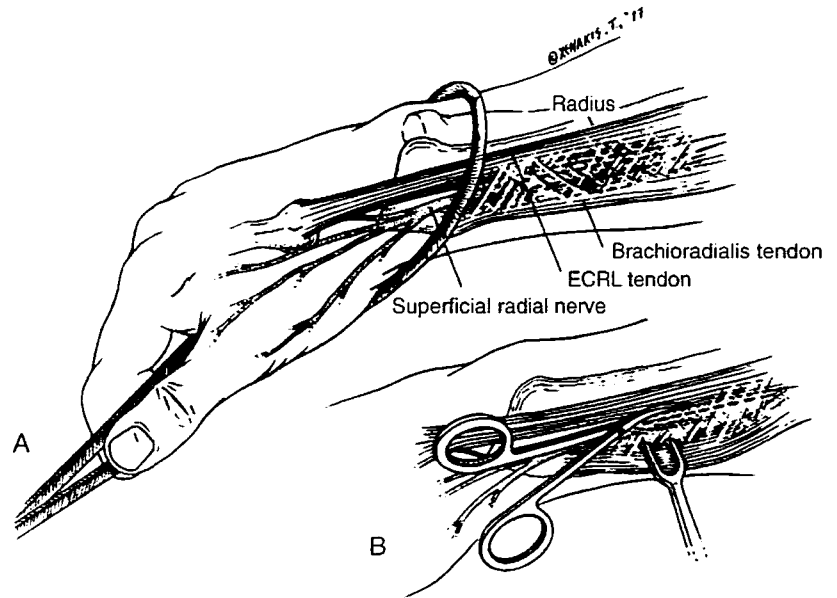


Figure 5. Anatomy (A) and surgical release (B) of cheiralgia paresthetica. B, Fascial sheath is divided and released distally to proximally to free constriction. (Courtesy of Thomas Xenakis.)

arises in the distal carpal tunnel and supplies the abductor pollicis brevis, opponens pollicis, superficial head of the flexor pollicis brevis, and the two lateral lumbricales.

Pronator Syndrome

Pronator syndrome is compression of the median nerve at any one of several sites at the elbow and proximal forearm (Fig. 6). There are three common sites of compression. The first area is beneath the bicipital aponeurosis (lacertus fibrosus) as its fascia is confluent with the pronator teres. This site of compression is seen more commonly when the pronator teres origin is high^{102, 120} or when there is fibrous thickening of the lacertus fibrosus. Second, at the level of the pronator teres as the nerve passes between the humeral and ulnar heads, compression may be caused by pronator hypertrophy, fibrous bands within the muscle,⁶² or an anomalous course of the nerve, as it has been shown that 15% of the time it passes below a solitary humeral head and 5% of the time may pierce the humeral head.⁹ The third site of compression for the median nerve is when it passes below a thickened fibrous arch of flexor digitorum superficialis.³⁸ Less common sites of compression include beneath the ligament of Struthers,⁸ which connects a supracondylar process on the distal humerus to the medial epicondyle in less than 1% of the

population, or by compression from a persistent median artery,⁶³ seen in 5% of the population. A very uncommon cause of compression is an enlarged bursa at the insertion site of the bicipital tuberosity.^{64, 120}

This syndrome is seen in sports that require repetitive, forceful pronation and gripping.^{76, 116} Most commonly seen in throwers, pronator syndrome has been reported in weightlifting, archery, tennis, arm wrestling, and rowing.^{18, 138} The clinical presentation consists of an insidious onset of volar forearm pain, most often related to activity (especially repetitive pronation and wrist flexion), and is relieved by rest. Hand pain may occur but is variable. Sensory symptoms have been reported but not as commonly as in carpal tunnel syndrome. In contradistinction to carpal tunnel syndrome, nocturnal symptoms are uncommon.

There are several clinical indicators that may help to confirm diagnosis of a pronator syndrome. A Tinel's sign in the proximal forearm may be positive. Indentations of the pronator teres, increased by pronation, suggest a tightened lacertus fibrosus.^{68, 126} Unilateral hypertrophy of the pronator musculature, as seen in pitchers and athletes who participate in racquet sports, may predispose to compression.

Symptoms of pain may be reproduced by provocative tests. Pain with resisted elbow flexion and supination with the elbow flexed to 120 to 130 deg imply that the lacertus fibrosus or ligament of Struthers is

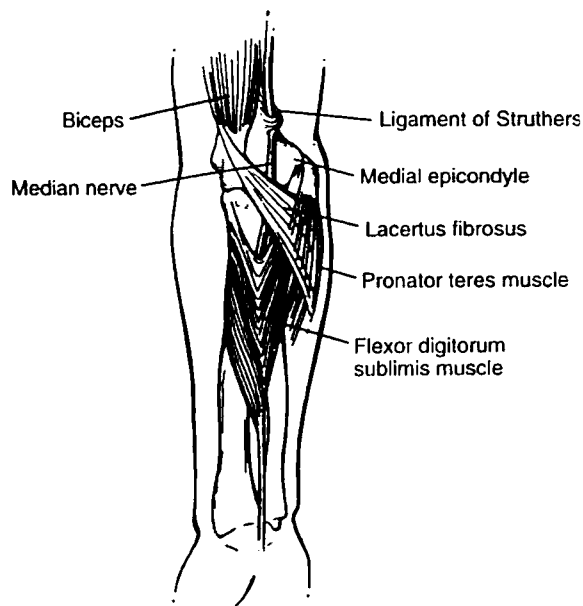


Figure 6. Anatomy of the median nerve in the proximal forearm. (From Butiers KP, Singer KM: Nerve lesions of the arm and elbow. In DeLee JC, Drez D Jr (eds): *Orthopaedic Sports Medicine: Principles and Practice*. Philadelphia, WB Saunders, 1994, p 807.)

the site of compression.³⁸ Pain with resisted pronation with the elbow extended and the wrist flexed (to eliminate the flexor digitorum superficialis) suggests localization of compression within the pronator teres. Reproduction of symptoms with the resisted flexion of the middle finger suggests the arch of the flexor digitorum superficialis as the site of compression. Passive stretching of the proximal forearm may reproduce symptoms, and muscle weakness involving the median nerve may be detected.^{38, 97, 120} Spinner¹²⁰ believes that the most sensitive test for pronator syndrome is deep, direct palpation of the proximal forearm over the pronator teres with reproduction of symptoms.

Further work-up may include radiographs to look for a supracondylar process and electrodiagnostic studies. Electromyography (EMG) and nerve conduction velocity testing for pronator syndrome have yielded disappointing results. Buchthal et al¹³ found that only 10% of the results supported the diagnosis, and Hartz et al⁵² found that only 2 of 39 operative cases showed definitive findings with a focal conduction block or delay across the elbow. It generally is thought that for these reasons pronator syndrome remains a clinical diagnosis.

Initial management is rest of the limb, especially from weight training, anti-inflammatory medications, and occasional splinting. Results have been very favorable with this conservative treatment regime. Surgical treatment is only necessary with refractory symptoms after 6 months, axonal loss demonstrated by EMG, or persistent decrease in function. Surgery involves exploration of the median nerve in the forearm from above the medial epicondyle to the arch of the flexor digitorum superficialis (Fig. 7).

Anterior Interosseous Nerve Syndrome

An uncommon entity, the anterior interosseous nerve (AIN) syndrome, is seen sporadically in athletes.⁴⁸ It can be seen after a single



Figure 7. The median nerve proximal to the pronator teres. (Courtesy of Kevin D. Plancher, MD, MS.)



Figure 8. Typical pinch sign with flattening of index pulp and classic palsy of the anterior interosseous nerve. (Courtesy of Kevin D. Plancher, MD, MS.)

violent muscle contracture from aggressive forearm exercises.³⁸ Repetitive trauma to the forearm and structural anomalies are other known causes of AIN syndrome. Isolated AIN syndrome also may be seen in acute brachial neuritis caused by sports (e.g., stingers) or postanesthesia from an interscalene block.¹⁰³

Structurally, there may be compression of the nerve by fibrous bands from the deep head of the pronator teres or flexor digitorum superficialis (FDS); aberrant or anomalous vessels¹¹⁸ or muscles,⁶⁹ including Gantzer's muscle (an accessory slip from the origin of flexor pollicis longus [FPL]; flexor carpi radialis brevis; or palmaris profundus.¹¹⁸ Spinner has reported an isolated compression of the branch to the flexor pollicis longus.¹¹⁸ This syndrome has been reported in "junk" baseball pitchers, weightlifters, gymnasts, tennis players, swimmers, and in a football player after receiving a direct blow to the forearm.^{17, 76, 138}

Clinically, the patient may present after a single episode of vague pain or with activity-related proximal forearm pain with atrophy but no sensory changes. Motor weakness usually begins 12 to 24 hours after the pain is noted. The characteristic motor examination finding is the inability to pinch the ends of the thumb and index finger secondary to weakness of the FPL and index flexor digitorum profundus (FDP) (Fig. 8). The pronator quadratus may be evaluated by testing pronation in full elbow flexion.¹³⁶ Unexpected findings, such as intrinsic weakness, may be seen with a Martin-Gruber anastomosis, an anomalous median-ulnar anastomosis seen in up to 15% of the population.

Electrodiagnostic studies represent the gold standard for diagnosis of this entity.⁷⁶ Electrodiagnostic studies are difficult and demand an

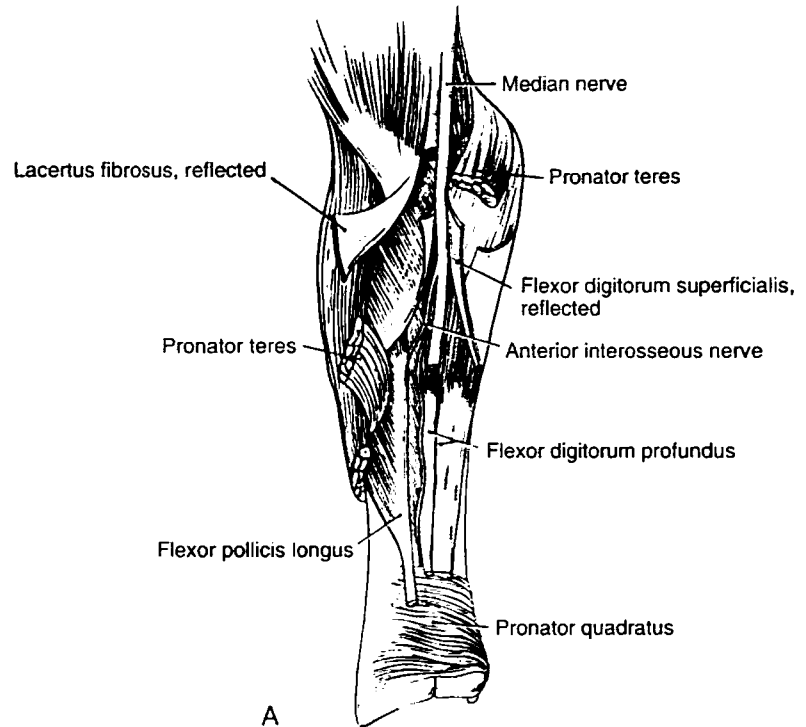


Figure 9. A, Course of the median nerve and origin of the anterior interosseous nerve in the proximal forearm. (From Butters KP, Singer KM: Nerve lesions of the arm and elbow. In Delee JC, Drez D Jr (eds): Orthopaedic Sports Medicine: Principles and Practice. Philadelphia, WB Saunders, 1994, p 996.)

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exact technique.⁹⁷ Denervation limited to the FPL/FDP index and pronator quadratus is diagnostic, but nerve conduction velocities are often normal.¹⁷

Conservative treatment with rest, ice, and nonsteroidal or tapering steroid anti-inflammatory medication is continued for approximately 8 weeks before surgery is considered. A postanesthetic acute brachial neuritis of the AIN should be given at least 4 months of conservative treatment before discussing surgical options. Surgical decompression is indicated if there is no response to conservative treatment, if progression of symptoms after 6 to 8 weeks exists, or if EMG indicates axonal denervation. Surgery entails exploration of the median nerve from the proximal forearm following the AIN to all its branches (Fig. 9).



Figure 9 (Continued). B, Photograph of the anterior interosseous nerve illustrating arch of the flexor digitorum superficialis prior to release. C, Anterior interosseous nerve following decompression. (Courtesy of Kevin D. Plancher, MD, MS.)

Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) is the most common example of a compressive neuropathy,¹²⁸ although it is not seen commonly in the athletic population.¹³⁶ CTS is a compression of the median nerve in the osteofibrous carpal tunnel created by the transverse carpal ligament and carpal bones (Fig. 10). CTS may be caused by direct trauma, repetitive use, or anatomic anomalies. CTS occurs more often in the dominant upper extremity, suggesting that use is a contributory factor. Repetitive flexion and extension of the wrist, as seen in lacrosse and gymnastics, and grip-intensive activities, such as cycling, are known to aggravate the condition.^{116, 128} Studies have shown that the pressure inside the



Figure 10. Compressed median nerve with typical hourglass appearance. (Courtesy of Kevin D. Plancher, MD, MS.)

carpal tunnel is elevated at the extremes of flexion and extension and that carpal canal pressures are elevated at rest in symptomatic patients.¹¹⁶

Sporting activities that predispose athletes to CTS include those that involve repetitive or continuous flexion and extension of the wrist, as seen in cycling, throwing sports, racquet sports, archery, and gymnastics.^{104, 116, 136} Other causes of CTS include hypertrophy of the lumbrical muscles, as is seen often in weightlifters; flexor tenosynovitis; anatomic anomalies, including palmaris longus inside the carpal tunnel; palmaris profundus; hypertrophic FDS; accessory lumbricales; and a persistent median artery.¹³⁶

Clinically, patients with CTS have the classic symptoms of pain and paresthesias, especially at night, in the radial three and one-half digits. Athletes frequently complain of clumsiness and weakness when performing grip-related activities. Phalen and Tinel's tests are variably positive. Measured grip and pinch strengths are asymmetric (except in patients with bilateral involvement).¹²⁸ Electrodiagnostic studies vary but frequently demonstrate conduction delays across the wrist.⁷⁶

Initial treatment consists of activity modification, rest, anti-inflam-

matory medication, and splinting at night. Corticosteroid injections provide relief in up to 80% of patients, but this relief is often temporary.⁴⁵ Surgical decompression of the carpal tunnel is best undertaken early in its course (Fig. 11).¹²⁸ There are various methods to decompress the transverse carpal ligament, including newer endoscopic and limited open incision techniques. The pros and cons of the various releases will be discussed in the May 1996 issue of *Hand Clinics* on carpal and cubital tunnel surgery.

Ulnar Nerve

The ulnar nerve receives contributions from the spinal roots of C8-T1 and represents the terminal branch of the medial cord of the brachial plexus (see Fig. 1). In the arm, the nerve travels lateral to the brachial artery until it passes posteriorly under the medial triceps and through the medial intermuscular septum, the confluence of which forms the arcade of Struthers approximately 8 to 10 cm proximal to the medial epicondyle. The nerve passes through the ulnar groove posterior to the medial epicondyle, bordered medially by the ulnar collateral ligament and laterally by the medial epicondyle with the arcuate ligament forming its roof. The nerve then enters the cubital tunnel formed by the aponeurosis and two heads of the flexor carpi ulnaris. The nerve continues distally between the FDP dorsally and the flexor carpi ulnaris palmarly.

The first branch of the ulnar nerve is articular to the elbow joint. Branches to the flexor carpi ulnaris arise near the elbow followed by branches to the FDP. The palmar cutaneous nerve arises in the midforearm and supplies the proximal hypothenar eminence. The dorsal cutaneous nerve arises 5 to 8 cm proximal to the ulnar styloid and supplies the dorsum of the ulnar side of the hand. At the wrist, the ulnar nerve and artery lie between the flexor carpi ulnaris and the FDP and pass above the flexor retinaculum and below the volar carpal ligament and palmaris brevis. This is the ulnar tunnel, also known as Guyon's canal. It is bordered laterally by the hook of the hamate and medially by the pisiform. In zone 1 of Guyon's canal, the nerve divides into its deep and superficial branches. The superficial branch supplies the overlying palmaris brevis, then becomes entirely sensory to supply the hypothenar eminence and ring and small fingers. The deep branch curves around the hook of the hamate to supply the ulnar intrinsics ending its terminal branch in the first dorsal interossei.

Cubital Tunnel Syndrome

Ulnar nerve entrapment near the elbow is the second most common compressive neuropathy in the upper extremity.⁸⁶ The nerve in this location is vulnerable to injury by compression, traction, and friction. Entrapment is common at several locations (Fig. 12). The first is below the arcade of Struthers, approximately 8 cm proximal to the medial epicondyle. The second area is in the ulnar groove of the medial epicondyle. The third, and most common, is in the cubital tunnel formed distal to the medial epicondyle as the nerve passes between the two heads of

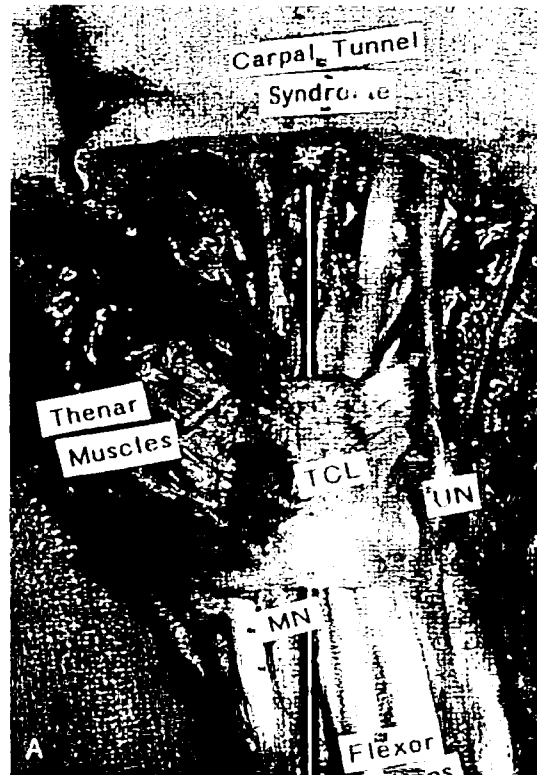


Figure 11. Anatomy of the volar wrist before (A) and after (B) transverse carpal ligament release. TCL = transverse carpal ligament; UN = ulnar nerve; MN = median nerve. (Courtesy of Kevin D. Plancher, MD, MS.)

Illustration continued on opposite page

the flexor carpi ulnaris (Fig. 13). Less common causes of this syndrome include direct trauma, hypermobility of the nerve, cubitus valgus, degenerative or post-traumatic changes near the medial elbow,⁶⁶ inflammatory conditions, ganglia, lipomas, and anatomic anomalies, such as an anconeus epitrochlearis muscle or the ligament of Spinner.^{38, 76, 120}

The act of throwing is often responsible for ulnar nerve entrapment at the elbow in the athlete. Tremendous forces develop at the elbow with angular velocities up to 7000 deg per second.¹¹⁶ Studies have shown that the position of elbow flexion and wrist extension creates a threefold increase in ulnar nerve pressure in the cubital tunnel, and when the upper extremity is placed in the cocking position of a throw, the pressure increases to six times the resting pressure.⁹⁴ Biomechanically, several processes cause an elevation of pressure. The arcuate ligament lengthens with elbow flexion until the proximal edge becomes taut at 90 deg of flexion. At the same time the ulnar collateral ligament relaxes and may bulge inward, potentially decreasing the space available to the nerve.¹⁷

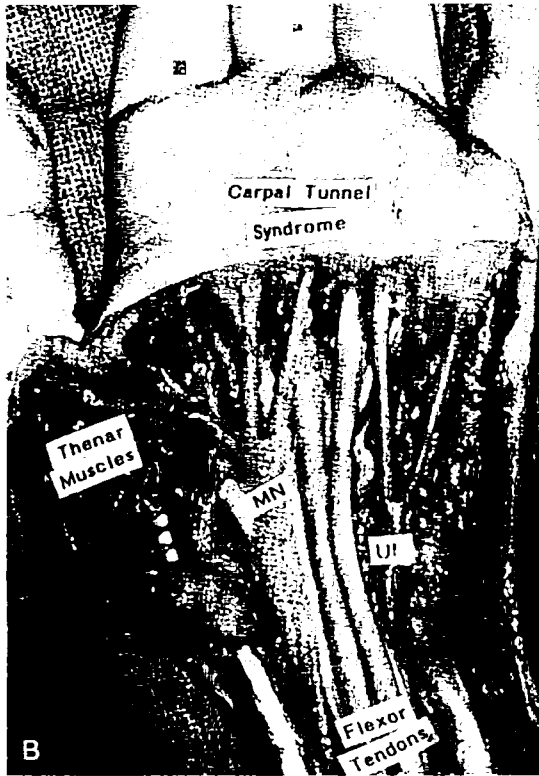


Figure 11 (Continued).

During elbow flexion, the ulnar nerve has been shown to elongate an average of 4.7 mm.^{2, 136} These factors all can contribute to the athletes' symptoms. Jobe and colleagues⁶¹ believe that throwers develop a progressive flexionvalgus deformity with attenuation of the ulnar collateral ligament. This exacerbates the athletes' symptoms by increasing the magnitude of the tensile forces already at work.

Cubital tunnel syndrome is found more often in the throwing athlete but can be seen in the recreational skier, weightlifter, and racquet sport enthusiast.⁴⁶ There has been a case report of cubital tunnel syndrome involving a cross-country skier. The mechanism of injury was extension and pronation required for poling.⁴³

Patients classically present with medial elbow and forearm pain and paresthesias radiating into the ring and little fingers and the dorsoulnar hand. Motor findings may be subtle, but patients may note a weak grip, clumsiness, or early fatigue of the hand. The elbow flexion test may be useful in reproducing symptoms.¹⁵ A Tinel's sign over the cubital tunnel is inconsistent. Hypermobile nerves are present in 16% of the asymptomatic population²³ and may cause pain on examination with

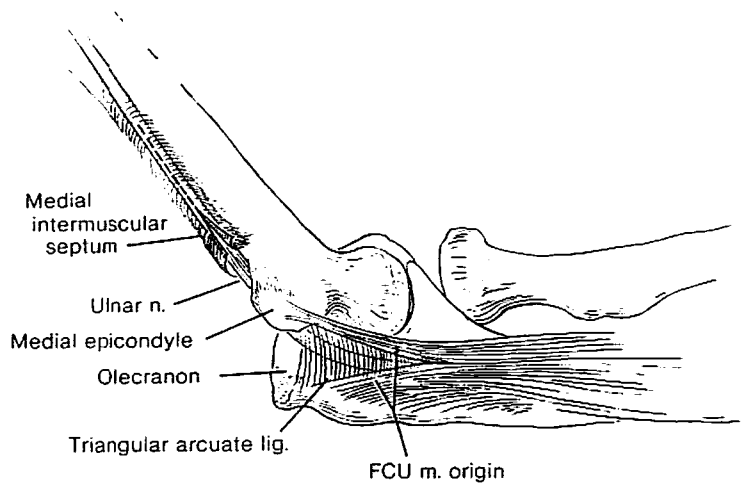


Figure 12. Anatomy of the ulnar nerve in the proximal forearm. FCU = flexor carpi ulnaris. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

range of motion. One should note the presence or absence of valgus stability, cubitus valgus, or flexion contractures. A careful motor examination must be documented. Electrodiagnostic studies demonstrating a significant loss of conduction velocity across the elbow can help to localize the lesion, but false negatives are not uncommon.

Nonoperative treatment consists of rest, ice, anti-inflammatory medication, and night splinting with the elbow in 45 deg of flexion and the

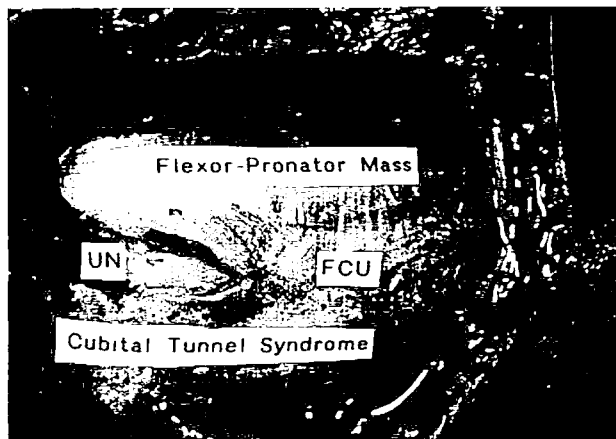


Figure 13. Cadaver showing the ulnar nerve (UN) passing between the two heads of the flexor carpi ulnaris (FCU) after release of the cubital tunnel. (Courtesy of Kevin D. Plancher, MD, MS.)



Figure 14. The course of the ulnar nerve at the elbow; the vessel loop is around the ulnar nerve proximally, and the retractor is holding the lateral head of the extensor carpi ulnaris. Note the course of the medial antebrachial cutaneous nerve. (Courtesy of Kevin D. Plancher, MD, MS.)

forearm in neutral rotation. If this treatment regime is unsuccessful or tests demonstrate a significant neuropathy, surgery may be indicated (Fig. 14). There are several options: in situ decompression; anterior transposition; or either subcutaneous, intramuscular, submuscular, or in situ decompression with medial epicondylotomy.⁸⁶ A thorough discussion of each of these surgical techniques will be covered in the May 1996 issue of *Hand Clinics*.

Ulnar Tunnel Syndrome

Compression of the ulnar nerve at the level of the wrist may occur as the nerve enters the ulnar tunnel or as the deep branch curves around the hook of the hamate and traverses the palm (Fig. 15). Compression may be caused by ganglia, lipomas, anatomic anomalies, carpal fractures (most notably of the hook of the hamate), local inflammation, or an ulnar artery thrombosis.^{76, 116, 136} The predominant mechanism in sports involves direct compression or trauma.¹¹⁶ This injury has been reported widely in cyclists and referred to as the handlebar or cyclist's palsy (Fig. 16).³⁶ Ulnar tunnel syndrome commonly is seen in baseball catchers, hockey goalies, handball players, and racquet sports and golf enthusiasts.^{5, 76} Distal ulnar nerve palsy also may be seen as a push-up palsy,¹³³ following fractures of the hook of the hamate, or from a missed golf shot or baseball swing,^{93, 131} or, secondarily, from inflammation in the so-called racquet player's pisiform.⁵³ Degenerative changes in the pisotriquetral joint causing local inflammation also have been known to create an ulnar tunnel syndrome.

The lesion may present with motor, sensory, or mixed symptoms. Coincident involvement of the median nerve is common. Fixed motor

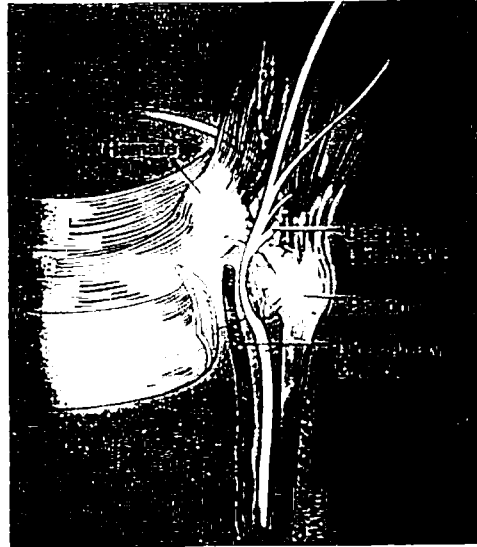


Figure 15. The ulnar nerve in the canal of Guyon. Br = brachial. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

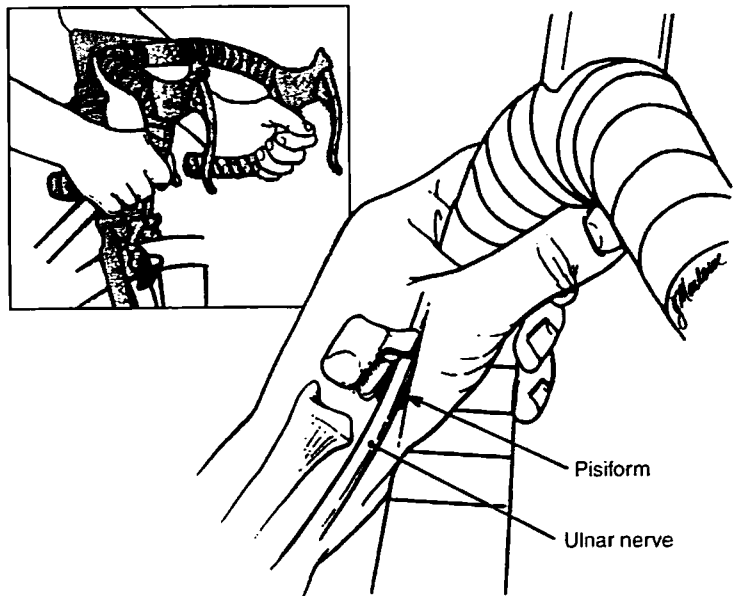


Figure 16. The mechanism of injury for cyclists' palsy. (Courtesy of J. Marlowe as published in Long RR: Nerve anatomy and diagnostic principles. In Pappas AM (ed): Upper Extremity Injuries in the Athlete. New York, Churchill Livingstone, 1995, p 484.)

deficits are fortunately rare but are the characteristic lesion in the ulnar claw hand. This posture is caused by the unopposed action of the extensor digitorum communis causing hyperextension of the ring and small fingers and hyperflexion of the proximal interphalangeal joint by the unopposed FDP. Anomalous motor and sensory findings may occur secondary to the Martin-Gruber anastomosis, an ulnar to median anastomosis seen in 15% of the population, or the Riche-Cannieu anastomosis involving the deep branch of the ulnar nerve and the median nerve in the hand.

EMG of both ulnar and median nerves may be helpful, although most symptomatic patients exhibit normal electrodiagnostic findings.⁴² Treatment is initially nonsurgical, involving adjusting hand positions on the bicycle or increasing padding over the bars, mitts, or gloves. Rest, anti-inflammatory drugs, local corticosteroid injections, and splinting may help.

Some authors feel that surgical decompression rarely is required.⁸⁵ When surgery is necessary, release of Guyon's canal is done in an open fashion under local anesthesia with excellent results.

Digital Nerves

Injury to the digital nerves is often the result of repetitive trauma over the palm or digits. The most common example, as described by Dobyys et al²⁸ in 1972, is the bowler's thumb. The term *bowler's thumb* describes an injury to the ulnar digital sensory nerve of the thumb caused by direct trauma from the edge of the thumb hole of a bowling ball (Fig. 17). The radial digital nerve of the index finger is similarly at risk in racquet sports.^{29, 91} Digital nerve symptoms can be seen in baseball, handball, gymnastics, and the martial arts.⁷⁶ The chronic nature of the injury was demonstrated by Buckhout et al,¹⁴ who showed that digital nerve involvement in handball players occur in those with more than 2 years or 200 hours of play.

Patients with a compression neuropathy of the digit usually present with paresthesias and altered sensation in the affected area. Though there is no true motor involvement, grip strength may be decreased secondary to pain. There may be a firm, palpable mass or tender cord on examination of the digit. Bowler's thumb has been demonstrated pathologically to represent perineural fibrosis of the digital nerve.¹³³ Digital neuropathies also may be caused by ischemia from cold intolerance, and atrophy of the overlying affected skin may be seen. There are reports in the literature of pitchers experiencing digital ulcers not responsive to basic wound care that heal after surgical exploration of the involved digital nerves, which were reported to have been compressed at Cleland's ligament and in the lumbrical canal.^{60, 104}

Digital neurapraxias usually will respond to avoidance of the inciting activity, splinting, and protective padding. Sport-specific changes are appropriate, such as alterations in the grip or handle size or padding

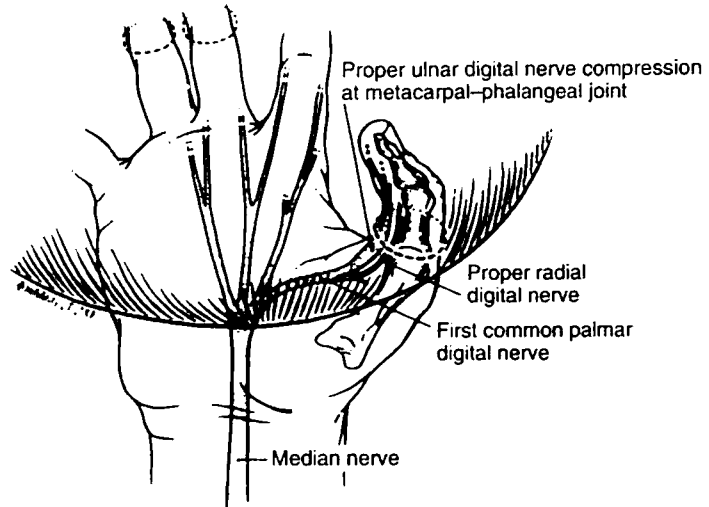


Figure 17. The compression of the ulnar digital nerve of the thumb in bowler's thumb. (Courtesy of Thomas Xenakis.)

in racquet sports, changes in the padding of a baseball glove, or changes in the size, spacing, or depth of the finger holes of a bowling ball.^{98, 116}

Surgical management may be required in lesions not responsive to conservative care. Surgical management consists of decompression and internal neurolysis. Neuromas may be resected with primary nerve repair.¹⁰⁴ Some surgeons advocate relocation of the nerve for bowler's thumb to allow it to sit in a more dorsolateral position out of harm's way.³³

TENDINOPATHIES

Tendons transmit the force of a muscular contraction to bone. The demands placed on the musculotendinous unit during athletic activity may overwhelm the capabilities of the tissues. The tendon injury may be macrotraumatic, involving acute tissue destruction, or microtraumatic, from chronic loading.⁷⁰ Earlier in this article, overuse was defined as the level of repetitive microtrauma sufficient to overwhelm a tissue's normal adaptive capability.⁹⁶ Overuse often leads to derangement of both the mechanic and physiologic components of the normal tendon and is recognized clinically as tendinitis.¹³⁰ This section discusses these overuse tendinopathies after reviewing some basics about tendon pathophysiology.

A tendon loaded in tension undergoes an initial nonlinear elongation as the collagen fibers are stretched from their crimped resting

configuration to become taut. This phenomenon occurs during the first 4% of elongation. If the force that is applied is removed, the fibers will return, unchanged, to their resting state. When a force is applied that produces a strain greater than 4%, collagen cross-links begin to break, allowing the collagen fibrils to slide past each other. As the strain increases, individual fibers will rupture. At strains of 10% to 12%, macroscopic failure occurs.^{90, 96, 130}

Tendons also exhibit the time-dependent deformation in response to a load, i.e., creep. Goldstein and colleagues⁴⁷ have demonstrated that the application of a physiologic load will cause fibrillar creep if the time between loads does not allow for elastic recovery to a resting length. Most agree that a tendon is at its greatest risk for injury when tension is applied rapidly and at an oblique angle. A tendon already under tension or a muscle maximally innervated or stretched are other risk factors for rupture.⁶

The physiologic response to injury begins with an inflammatory reaction. The clinical signs are pain, swelling, erythema, and warmth. Treatment during this phase of injury consists of rest, ice, elevation, and compression. Nonsteroidal anti-inflammatory medications are used to limit the inflammatory response.^{65, 96}

The proliferative phase begins when collagen and ground substance production is initiated. Pitner⁹⁶ stresses that these products are immature, disorganized, and easily disrupted. Treatment during this injury phase should focus on minimizing the forces acting on the injured tendon. Repetitive disruption during this phase may lead to chronic changes including fibrosis.⁵⁵

The maturation phase begins 1 to 2 weeks postinjury. This phase involves a progressive restructuring of the disorganized, healing tendon to resemble more closely the normal tendon.⁹⁶ Studies have shown that the strength of healing tendons increases faster if controlled forces are applied.^{65, 96} Strenuous activity still should be avoided, but range-of-motion and isometric exercises may begin. Tendons are able to respond to increasing forces by increasing their cross-sectional area and tensile strength. During the maturation phase, increases in collagen content, degree of cross-linking, and mucopolysaccharide content all have been observed.^{4, 96} This process allows for the healing to begin. Understanding tendons and their response to injury helps us to treat the common tendinopathies discussed in the following section.

Extensor Compartment Tendinopathies

De Quervain's Tenosynovitis

De Quervain's is a stenosing tenosynovitis of the abductor pollicis longus (APL) and extensor pollicis brevis (EPB). Athletes considered to be at risk are those who require a forceful grasp, combined with repeti-

tive use of the thumb and ulnar deviation.^{65, 140} Sports such as racquet sports, golf, fly fishing, javelin, and discus throwing are all good examples.¹⁴⁰ Rettig et al¹⁰⁵ noted that the left thumb of the right-handed golfer is susceptible to this overuse injury due to hyperabduction during the swing.

The APL and EPB tendons pass through a fibro-osseous tunnel formed by a groove in the radial styloid and overlying extensor retinaculum (Fig. 18). The tendons deviate as they pass through the tunnel, and this angle increases with ulnar deviation of the wrist.⁹⁹ Whether the pathologic condition originates in the tenosynovium¹⁴⁰ or the tendon sheath^{25, 39} remains unclear.⁶⁵

Patients typically present with pain over the radial styloid, which worsens with thumb or wrist motion. Examination reveals tenderness over the radial styloid, swelling, and occasionally crepitus or triggering.^{24, 41} A positive Finkelstein test, that is, reproduction of pain with ulnar deviation of the wrist while the thumb is adducted, is typical but not pathognomonic.^{41, 112, 130} The differential diagnosis of pain in this region includes scaphoid fracture, flexor carpi radialis tendinitis, thumb carpometacarpal arthritis, intersection syndrome, and Wartenberg's syndrome.^{41, 123, 130}

Initial management on confirmation of the diagnosis includes

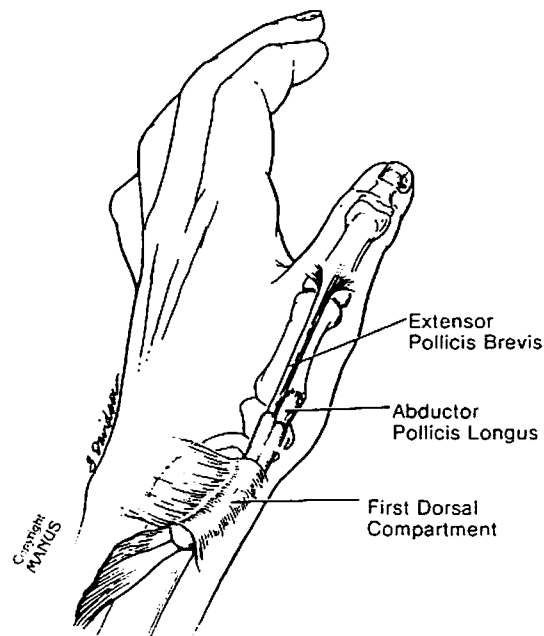


Figure 18. Anatomy of the first dorsal compartment. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

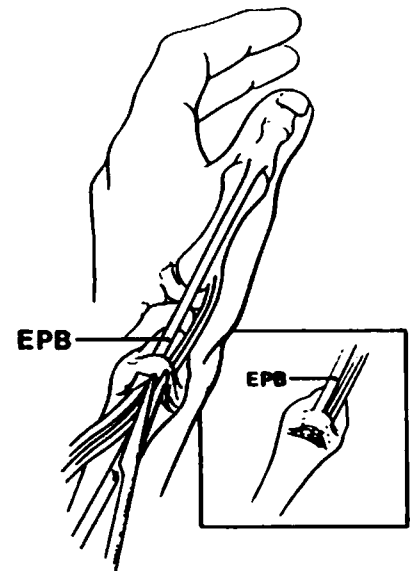


Figure 19. First dorsal compartment release illustrating longitudinal septum separating the APL and EPB. (From Stern PJ: Tendinitis, overuse syndrome and tendon injuries. *Hand Clin* 6:469, 1990.)

thumb-spica splinting, anti-inflammatory medication, and a corticosteroid injection into the first dorsal compartment. Anatomic studies have demonstrated that a longitudinal septum separates the APL and EPB in 20% to 30% of reported cases (Fig. 19).⁴¹ This may be one reason for a poor response to injection and an explanation for recurrence of symptoms. For this reason, Leslie and colleagues⁷¹ recommend a second, more dorsal injection.

Surgical management is pursued early if there is no resolution with conservative care.⁴¹ Surgery involves decompression of the first dorsal compartment with care taken to divide any septation present in the compartment. Confirmation at surgery of the multiple slips of the APL is essential, along with identification of EPB in the depths of the wound (Fig. 20).¹²⁴ Recognized complications of surgical decompression include injury to the superficial radial nerve, volar tendon subluxation, hypertrophic scarring, tendinous adhesions, and persistence of symptoms (possibly due to inadequate decompression).^{10, 41, 123}

Intersection Syndrome

Athletes with intersection syndrome have pain in the dorsoradial wrist where the APL and EPB cross over the wrist extensors (ECRL and ECRB) (Fig. 21). The syndrome has been reported in weightlifters, rowers, canoeists, and recreational tennis enthusiasts, all of whom are exposed to repetitive wrist motion.¹⁴⁰

Several theories have been put forth to explain the pathophysiology of intersection syndrome. Previous studies have suggested inflammatory

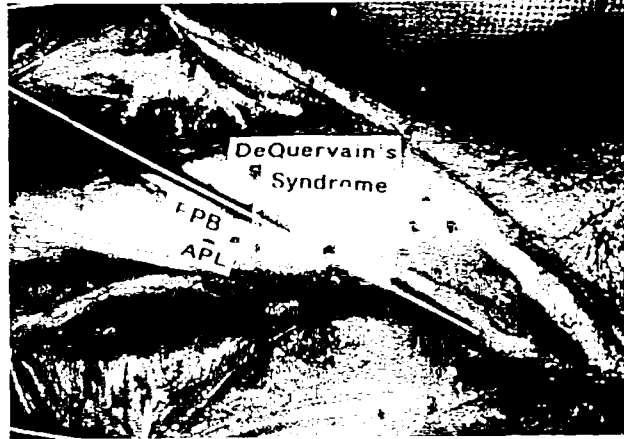


Figure 20. Cadaver with anatomic probe under extensor retinaculum overlying first dorsal compartment. (Courtesy of Kevin D. Plancher, MD, MS.)

peritendinitis,⁵⁵ development of an adventitial bursa,¹⁴¹ and muscular hypertrophy.¹³⁹ Grundberg et al⁵⁰ found that tenosynovitis of the second dorsal compartment was present in all operative specimens studied and thought that this represented the basic pathologic origin.

Clinically, athletes have swelling, tenderness, and often crepitus found 4 to 6 cm proximal to Lister's tubercle. Initial treatment includes rest, splinting, anti-inflammatory medication, and a corticosteroid injection.^{41, 50} Conservative treatment is successful 95% of the time. Surgical treatment for resistant cases consists of release and tenosynovectomy of the second dorsal compartment, exploration of the intersection zone

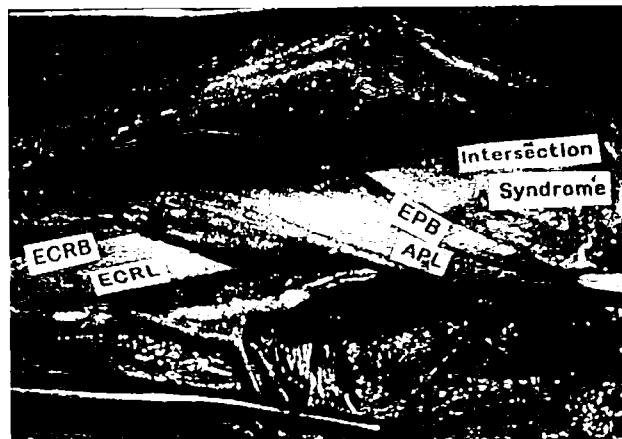


Figure 21. Anatomic dissection illustrating the intersection of the APL, EPB, and the wrist extensors. (Courtesy of Kevin D. Plancher, MD, MS.)

with debridement of any inflammatory or bursal tissue, and release of the fascial sheaths of the APL and EPB.^{50, 65}

Extensor Pollicis Longus (EPL) Tendinitis

EPL tendinitis has been reported in athletes who participate in racquet sports.¹⁰⁵ It was described originally as a disorder among Prussian drummers referred to as "drummer boy palsy."³² Anatomic studies have demonstrated the muscle belly of the EPL inside the third dorsal compartment in some cadavers.⁸⁸ EPL tendinitis commonly is seen in patients with rheumatoid arthritis and in those with recent distal radius fractures.¹²³ Early diagnosis and treatment is necessary to prevent tendon rupture at Lister's tubercle.⁴¹

Athletes complain of swelling, tenderness, and occasionally crepitus localized to the third dorsal compartment where the EPL curves around Lister's tubercle. The pain often is aggravated by thumb motion. Initial treatment consists of thumb-spica splinting, rest, and anti-inflammatory medication. Corticosteroid injections are not recommended as increased local tissue pressure may heighten the risk of tendon rupture.¹²³

Surgical treatment of EPL tendinitis consists of release of the third dorsal compartment and transposition of the EPL tendon. Froimson⁴¹ advocates a translocation of the tendon to the radial side of Lister's tubercle and closure of the extensor retinaculum to prevent relocation (Fig. 22).

Extensor Indices Proprius (EIP) Syndrome

First described in two athletes in 1969,¹⁰⁸ the EIP syndrome can be precipitated by direct trauma or overuse. Ritter and Inglis¹⁰⁸ noted that

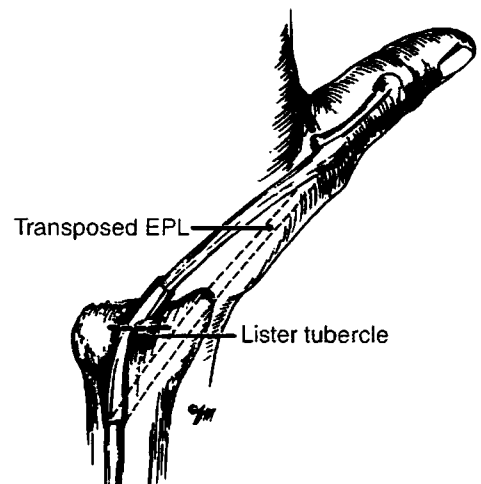


Figure 22. Surgical translocation of the EPL tendon. (Illustration by Elizabeth Roselius, © 1993. From Froimson AI: Tenosynovitis and tennis elbow. In Green DP (ed): Operative Hand Surgery. New York, Churchill Livingstone, 1993, p 1999; with permission.)

on surgical exploration, the muscle belly of the EIP entered the fourth dorsal compartment. Anatomic studies have shown that the musculotendinous junction of the EIP enters the fourth dorsal compartment in 75% of 263 cadavers studied.²² The cause of discomfort is thought to be muscular hypertrophy following exercise or synovitis from repetitive motion.

Pain and swelling over the fourth dorsal compartment is the hallmark of this disorder. Resisted extension of the index finger with the wrist in flexion is a reliable provocative test.¹²¹ If swelling appears distal to the extensor retinaculum, then the differential diagnosis of tenosynovitis of the extensor digitorum communis, dorsal carpal ganglion, or hypertrophy of an anomalous extensor digitorum manus should be considered.^{65, 107}

Conservative treatment consists of anti-inflammatory medication, corticosteroid injection, and splinting of the wrist and metacarpophalangeal joints to rest the digital extensors. Kiefhaber and Stern⁶⁵ note that resolution may be slow and recommend continuation of nonoperative treatment for 8 to 12 weeks. If conservative methods fail, division of the extensor retinaculum over the fourth dorsal compartment and synovectomy usually produce prompt resolution of symptoms.

Extensor Digiti Minimi (EDM) Tendinitis

Tenosynovitis of the fifth dorsal compartment is rare and has been associated with trauma³¹ and overuse.⁵⁷ Anatomic anomalies also have been implicated¹ as the cause of this syndrome, but anatomic studies have shown multiple tendon slips in 93% of the cadavers studied.¹¹³

Pain and swelling just distal to the ulnar head often is found on examination. Treatment involves the use of an ulnar gutter splint, anti-inflammatory medication, and corticosteroid injection into the fifth dorsal compartment. Surgical division of the retinaculum over the fifth dorsal compartment may be required for resistant cases.

Extensor Carpi Ulnaris (ECU) Tendinitis

ECU tendinitis is the second most common stenosing tenosynovitis of the hand following de Quervain's.¹⁴⁰ Tendinitis involving the ECU is seen in athletes participating in sports that require repetitive wrist motion, such as racquet sports, rowing, baseball, and golf.¹⁰⁵ ECU tendinitis also may occur secondary to post-traumatic ECU subluxation. The ECU is unique among extensor tendons because it passes through its own fibro-osseous tunnel, separate from the overlying extensor retinaculum (Fig. 23).¹¹⁹ The fibrous sheath overlying the ECU may be ruptured in forced supination, flexion, and ulnar deviation of the wrist, as seen in the trailing hand following a baseball swing.^{16, 35, 65} This rupture may occur even with an intact extensor retinaculum.¹⁶ Other causes of secondary ECU tendinitis include tears of the triangular fibrocartilage complex⁹¹ and anatomic variants.⁷

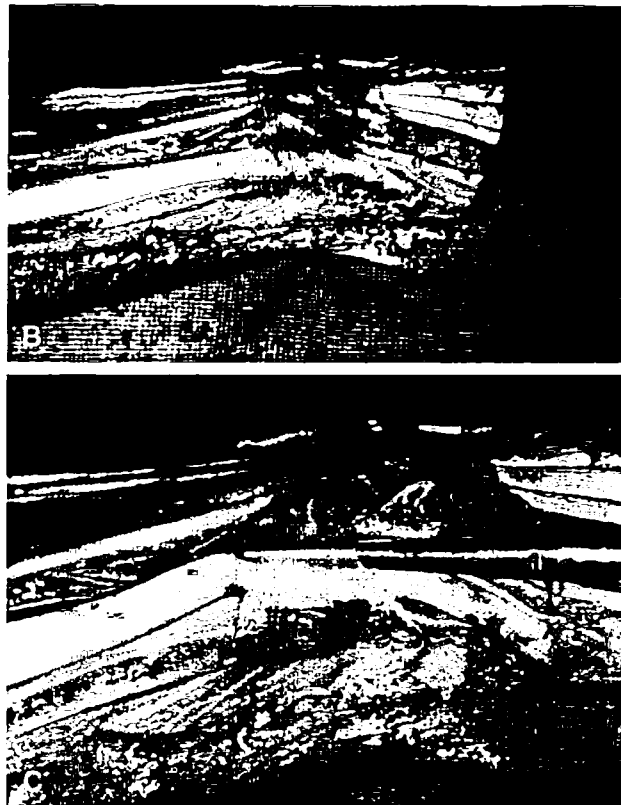
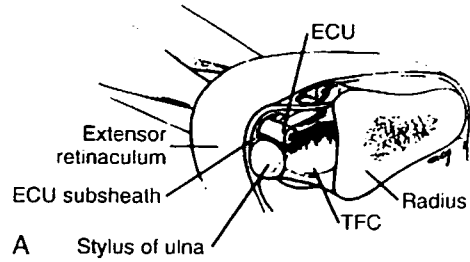


Figure 23. A, The extensor retinaculum, ECU subsheaths, and tendons. (From Kieffhaber TR, Stern PJ: Upper extremity tendinitis and overuse syndrome in the athlete. *Clin Sports Med* 11:49, 1992.) B, Intact extensor retinaculum over sixth dorsal compartment. C, Underlying ECU subsheaths well visualized. TFC = triangular fibrocartilage complex. (Courtesy of Kevin D. Plancher, MD, MS.)

Athletes with ECU tendinitis have pain and swelling distal to the ulnar head, exacerbated by resisted wrist extension. ECU subluxation may be elicited with supination and ulnar deviation of the wrist. This deviation can result in a painful snap over the dorsal ulnar wrist.¹²³ In

pronation, the ECU is palpated just distal to the ulnar head on the dorsum of the wrist, whereas in supination it is palpated along the ulnar border of the wrist. Conservative therapy is indicated in acute stages of symptoms.⁹¹ Patients not responding to conservative care require surgical decompression of the sixth dorsal compartment. Hajj and Wood⁵¹ suggest radial release of the fibro-osseous tunnel to prevent postoperative subluxation of the tendon, followed by repair of the extensor retinaculum. ECU subluxation may be treated acutely in a long-arm cast with the wrist in full pronation and slight dorsiflexion,¹⁴⁰ but it rarely responds to nonoperative care.^{35, 111} A retinacular sling¹¹⁹ or free retinacular graft³⁵ may be used to stabilize the ECU.

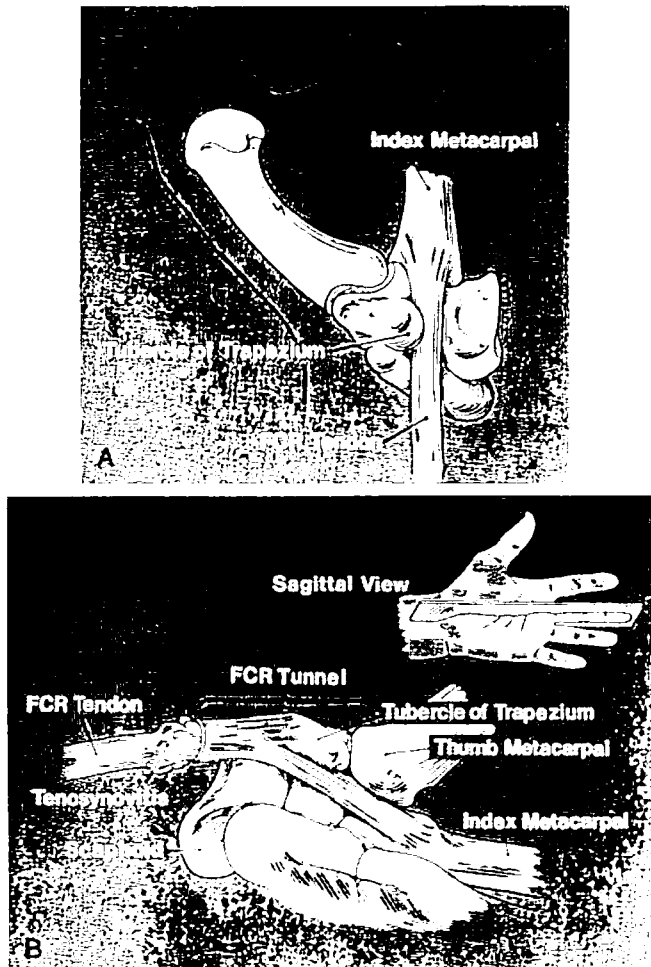


Figure 24. Anterior (A) and sagittal (B) views of the FCR tunnel at the wrist. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)

Flexor Compartment Tendinopathies

Flexor Carpi Radialis (FCR) Tendinitis

Repetitive wrist motions experienced by some athletes can lead to a stenosis and synovitis within the fibro-osseous tunnel containing the FCR.⁴¹ Entities that may coexist and confuse the clinical picture include scaphotrapezial arthritis,⁴⁰ scaphoid nonunions, basal joint arthritis of the thumb,⁹⁹ volar ganglia,¹²³ and Linburg's syndrome.⁷²

The FCR passes through a synovial tunnel bordered by the scaphoid tuberosity, trapezium ridge, and the transverse carpal ligament (Fig. 24). Within the tunnel, the tendon occupies 90% of the available space.¹¹ As the tendon enters this tunnel, it deviates 30 deg dorsally over the volar pole of the scaphoid to insert at the base of the second and third metacarpals and provides a slip to the trapezium ridge.¹¹ Weeks¹³⁵ has suggested that this angulation may create mechanical irritation and predispose the athlete to tenosynovitis.

Athletes with FCR tendinitis have pain over the volar aspect of the wrist, proximal to the wrist crease and overlying the FCR tendon. Lister⁷⁵ described a provocative test performed by abruptly extending a relaxed wrist to reproduce the pain. Pain also may be elicited on resisted flexion and radial deviation. Chronic synovitis and tendon ruptures may not allow active testing in some patients.¹³⁵

Immobilization, anti-inflammatory medication, and a corticosteroid injection may provide relief in the setting of a primary tendinitis. Chronic processes may be resistant and often require decompression of the fibro-osseous tunnel.⁴⁴ In the setting of an FCR rupture, simple debridement of the stump can provide effective pain relief.⁶⁵

Flexor Carpi Ulnaris (FCU) Tendinitis

Most golf and racquet sports athletes experience FCU tendinitis at some point in their career due to the chronic repetitive wrist motions needed in their sports.⁵³ The differential diagnosis for pain at the volar ulnar wrist is pisotriquetral arthritis. This phenomenon is found more commonly in young gymnasts. Pisotriquetral instability caused by the repetitive pronation and supination of racquet sports has been found to be a mechanism predisposing this small joint to arthritic changes.⁵³ Pain and crepitus produced by moving the pisiform radially and ulnarly upon the triquetrum is indicative of pisotriquetral arthritis.⁹²

FCU tendinitis presents clinically with pain and swelling localized just distal to the pisiform. Passive wrist extension and resisted flexion and ulnar deviation exacerbate the pain. Radiographs are usually negative, though calcific deposits may be seen occasionally on a 20-deg supinated oblique lateral radiograph.²¹

Conservative treatment consists of dorsal splinting, anti-inflammatory medication, and a corticosteroid injection. If surgical intervention is required, Palmieri⁹² recommends pisiform excision and a 5-mm Z-plasty

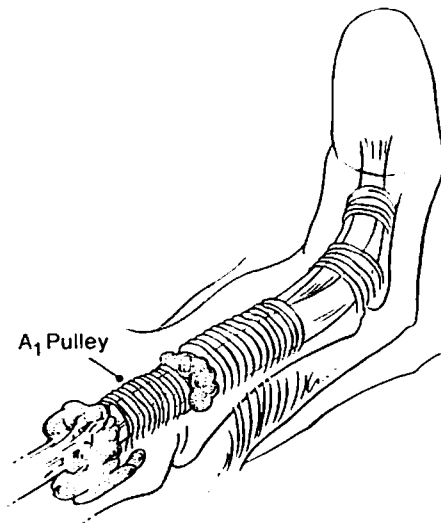


Figure 25. Stenosing tenosynovitis of the digit at the A₁ pulley. (Courtesy of The Indiana Hand Center, Indianapolis, Indiana.)



Figure 26. A, pulley intact (A) and following surgical release (B). Note the course of digital vessels and nerves. (Courtesy of Kevin D. Plancher, MD, MS.)

Illustration continued on opposite page

lengthening of the FCU tendon. Pisotriquetral arthritis, when found as the diagnosis, can be treated with pisiform excision.⁵³

Trigger Digits

Stenosing tenosynovitis of the digits causes snapping or triggering with flexion and extension of the involved digit due to a disproportion between the flexor tendon and its sheath (Fig. 25).^{41, 59} Most commonly idiopathic,⁵⁹ it may be caused by blunt trauma or mechanical irritation. Sports requiring the repetitive, forceful grip of a tool or direct trauma to the region of the A₁ pulley may predispose the patient to this condition.^{12, 74, 91} This condition is seen in handball players, baseball catchers, gymnasts, weightlifters, and golfers.¹⁸ Trigger finger has a higher incidence in the dominant hand.^{129, 130} This finding has convinced most that stenosing tenosynovitis is a cumulative trauma disorder.

Symptoms include local pain, triggering, and occasionally locking. A nodule is often palpable at the level of the metacarpal head with triggering occurring at the A₁ pulley. Nonoperative care with splint-



Figure 26 (Continued).

ing and the injection of corticosteroids into the tendon sheath has been reported to provide successful outcomes ranging from 36% to 84%.^{41, 67, 82, 100, 106}

Surgical release of the A₁ pulley under local anesthesia is successful in relieving the symptoms of a trigger digit. This may be done openly or percutaneously (Fig. 26).^{41, 77} Though surgery is generally successful, residual symptoms and complications do occur.^{12, 129} Major complications include digital nerve injury, painful scarring, and bowstringing of the tendon.^{41, 130} Recurrence of triggering and loss of full flexion usually is due to enlargement of the tendon distal to the A₂ pulley. Recurrence may be treated with surgical release of the A₃ pulley¹⁰¹ or a reduction flexor tenoplasty.¹¹⁵

SUMMARY

Overuse syndromes of the upper extremity in the athletic population are a common and often difficult problem for physician and patient alike. Optimal function of the upper extremity is tied intimately to success in many sporting activities. Correct diagnosis and proper care require a thorough knowledge of the pertinent anatomy, pathophysiology, and pathomechanics involved in each disorder. Conservative care with rest, activity modification, and medication is adequate for most athletic injuries. Surgical intervention may be indicated for continuing pain, decreased performance, or to prevent chronic changes. Surgery must be followed by thoughtfully prepared training and rehabilitation programs to optimize the chances of a successful outcome.

References

1. Ambrose J, Goldstone R: Anomalous extensor digiti minimi proprius causing tunnel syndrome in the dorsal compartment. *J Bone Joint Surg Am* 57:706-707, 1975
2. Apfelberg DB, Larson SJ: Dynamic anatomy of the ulnar nerve at the elbow. *Plast Reconstr Surg* 51:76-81, 1973
3. Armstrong TJ, Chaffin DB: Some biomechanical aspects of the carpal tunnel. *J Biomech* 2:567, 1979
4. Armstrong TJ, Fine LJ, Goldstein SA, et al: Ergonomic considerations in hand and wrist tendinitis. *J Hand Surg [Am]* 12:830-837, 1956
5. Aulicino PL: Neurovascular injuries in the hands of athletes. *Hand Clin* 6:455, 1990
6. Barfred T: Experimental rupture of the Achilles tendon: Comparison of various types of experimental rupture in rats. *Acta Orthop Scand* 42:528-543, 1971
7. Barfred T, Adamsen S: Duplication of the extensor carpi ulnaris tendon. *J Hand Surg [Am]* 11:423-425, 1986
8. Barnard LB, McCoy SM: The supracondylar process of the humerus. *J Bone Joint Surg Am* 28:845, 1946
9. Beaton LE, Anson BJ: The relation of the median nerve to the pronator teres muscle. *Anat Rec* 75:23-26, 1929
10. Belsole RJ: De Quervain's tenosynovitis: Diagnostic and operative complications. *Orthopedics* 4:899-903, 1981

11. Bishop AT, Gabel G, Carmichael SW: Flexor carpi radialis tendinitis: I. Operative anatomy. *J Bone Joint Surg Am* 76:1009-1014, 1994
12. Bonnici AV, Spencer JD: A survey of trigger finger in adults. *J Hand Surg [Br]* 13:202, 1988
13. Buchthal F, Rosenfalck A, Trojaberg W: Electrophysiologic findings in entrapment of the median nerve to wrist and elbow. *J Neurol Neurosurg Psychiatry* 37:340, 1974
14. Buckhout BC, Warner MA: Digital perfusion of handball players. *Am J Sports Med* 8:206, 1980
15. Buehler MJ, Thayer DT: The elbow flexion test: A clinical test for cubital tunnel syndrome. *Clin Orthop* 233:213, 1988
16. Burkhart SS, Wood MB, Linscheid RL: Posttraumatic recurrent subluxation of the extensor carpi ulnaris tendon. *J Hand Surg [Am]* 7:1-3, 1982
17. Butters KP, Singer KM: Nerve lesions of the arm and elbow. In Deter JC, Drez DJ (eds): *Orthopaedic Sports Medicine: Principles and Practice*. Philadelphia, WB Saunders, 1994, pp 802-871
18. Cabrera JM, McCue FC: Nonosseous athletic injuries of the elbow, forearm and hand. *Clin Sports Med* 5:681-700, 1986
19. Carr D, David P: Distal posterior interosseous nerve syndrome. *J Hand Surg [Am]* 10:873, 1985
20. Carragee EJ, Hentz VR: Repetitive trauma and nerve compression. *Orthop Clin North Am* 19:157, 1988
21. Carrol RE, Coyle MP: Dysfunction of the pisotriquetral joint: Treatment by excision of the pisiform. *J Hand Surg [Am]* 10:703, 1985
22. Cauldwell EW, Anson BJ, Wright RR: The extensor indicis proprius muscle: A study of 263 consecutive specimens. *Quart Bull Northwestern Univ M School* 17:267-279, 1943
23. Childress HM: Recurrent ulnar nerve dislocation at the elbow. *J Bone Joint Surg Am* 38:978, 1956
24. Chow SP: Triggering due to de Quervain's disease. *Hand* 11:93-94, 1979
25. Conklin JE, White WL: Stenosing tenosynovitis and its possible relation to the carpal tunnel syndrome. *Surg Clin North Am* 40:531-540, 1960
26. Dellon AL: Partial dorsal wrist denervation: Resection of the distal posterior interosseous nerve. *J Hand Surg [Am]* 10:527-533, 1985
27. Dellon AL, Mackinnon SE: Radial sensory nerve entrapment in the forearm. *J Hand Surg [Am]* 11:199-205, 1986
28. Dobyms JH, O'Brien ET, Linscheid RL, et al: Bowler's thumb, diagnosis and treatment: Review of 17 cases. *J Bone Joint Surg Am* 54:751-755, 1972
29. Dobyms JH, Sim FH, Linscheid RL: Sports stress syndromes of the hand and wrist. *Am J Sports Med* 6:236, 1978
30. Dorfman LF, Jaepnam P: Handcuff neuropathy. *JAMA* 239:957, 1958
31. Drury BJ: Traumatic tendovaginitis of the fifth dorsal compartment of the wrist. *Arch Surg* 80:554, 1960
32. Dums F: Uber trommlerlahmungen. *Deutsche Militarztliche Zeitschrift* 25:145-155, 1896
33. Dunham W, Haynes G, Spring JM, et al: Bowler's thumb. *Clin Orthop* 83:99-101, 1972
34. Eaton CJ, Lister GD: Radial nerve compression. *Hand Clin* 8:343-357, 1992
35. Eckhardt WA, Palmer AK: Recurrent dislocation of the extensor carpi ulnaris tendon. *J Hand Surg [Am]* 6:629-631, 1981
36. Eckman PB, Perlstein G, Altrocchi PH: Ulnar neuropathy in the bicycle rider. *Arch Neurol* 32:130, 1975
37. Eversmann WW Jr: Compression and entrapment neuropathies of the upper extremity. *J Hand Surg [Am]* 8:759-766, 1983
38. Eversmann WW Jr: Entrapment and Compression Neuropathies. In Green DP (ed): *Operative Hand Surgery*, ed 3. New York, Churchill Livingstone, pp 1341-1385, 1993
39. Finkelstein H: Stenosing tendovaginitis at the radial styloid process. *J Bone Joint Surg Am* 12:509-540, 1930
40. Fitton JM, Shea FW, Goldie W: Lesion of the flexor carpi radialis tendon and sheath causing pain in the wrist. *J Bone Joint Surg Br* 50:359-363, 1968

41. Froimson A: Tenosynovitis and tennis elbow. *In* Green DP (ed): *Operative Hand Surgery*, ed 3. New York, Churchill Livingstone, 1992, pp 1989–2006
42. Frontera WR: Cyclists's palsy: Clinical and electrodiagnostic findings. *Br J Sports Med* 17:91, 1983
43. Fulkerson J: Transient ulnar neuropathy from nordic skiing. *Clin Orthop* 153:230–231, 1980
44. Gabel G, Bishop AT, Wood MB: Flexor carpi radialis tendinitis: II. Results of operative treatment. *J Bone Joint Surg Am* 76:1015–1018, 1994
45. Gelberman RH, Aronson D, Weisman MH: Carpal tunnel syndrome: Results of a prospective trial of steroid injection and splinting. *J Bone Joint Surg Am* 62:1181, 1980
46. Glousman RE: Ulnar nerve problems in the athlete's elbow. *Clin Sports Med* 9:365–377, 1990
47. Goldstein SA, Armstrong TJ, Chaffin DB: Analysis of cumulative strain in tendons and tendon sheaths. *J Biomech* 20:1–6, 1987
48. Goodman CE: Unusual nerve injuries in recreational athletes. *Am J Sports Med* 11:244–277, 1983
49. Gross MS, Gelberman RH: The anatomy of the distal ulnar tunnel. *Clin Orthop* 196:238–247, 1985
50. Grundberg AB, Reagan DS: Pathologic anatomy of the forearm: Intersection syndrome. *J Hand Surg [Am]* 10:299–302, 1985
51. Hajj AA, Wood MB: Stenosing tenosynovitis of the extensor carpi ulnaris. *J Hand Surg [Am]* 11:519–520, 1986
52. Hartz CR, Linscheid RL, Gromse RR, et al: Pronator teres syndrome: Compressive neuropathy of the median nerve. *J Bone Joint Surg Am* 63:885, 1981
53. Helal B: Racquet player's pisiform. *Hand* 10:87, 1978
54. Herasawa Y, Sakaheda M: Sports and peripheral nerve injury. *Am J Sports Med* 11:420–426, 1983
55. Herring SA, Nilson KL: Introduction to overuse injuries. *Clin Sports Med* 6:225–239, 1987
56. Heyse-Moore EH: Resistant tennis elbow. *J Bone Joint Surg Br* 9:64–66, 1984
57. Hooper G, McMaster MJ: Stenosing tenovaginitis affecting the tendon of the extensor digiti minimi at the wrist. *Hand* 11:299–301, 1979
58. Howard NJ: Peritendinitis crepitans. *J Bone Joint Surg Am* 19:447–459, 1937
59. Hueston JT, Wilson WF: The aetiology of trigger finger. *Hand* 4:257, 1972
60. Itoh Y, Wakano K, Takeda T, et al: Circulatory disturbances in the throwing hand of baseball pitchers. *Am J Sports Med* 15:264, 1987
61. Jobe FW, Stork H, Lombardo ST: Reconstruction of the ulnar collateral ligament in athletes. *J Bone Joint Surg Am* 68:1558–1563, 1986
62. Johnson RK, Spinner M, Shrewsbury MM: Median nerve entrapment syndrome in the proximal forearm. *J Hand Surg [Am]* 4:48–51, 1979
63. Jones NF, Ming NL: Persistent median artery as a cause of the pronator syndrome. *J Hand Surg [Am]* 13:728–732, 1988
64. Karanjia ND, Stiles PJ: Cubital bursitis. *J Bone Joint Surg Br* 70:832–833, 1988
65. Kiefhaber TR, Stern PJ: Upper extremity tendinitis and overuse syndromes in the athlete. *Clin Sports Med* 11:39–55, 1992
66. King JW, Brelstard HJ, Tullos HS: Analysis of the pitching arm of the professional baseball pitcher. *Clin Orthop* 67:116–123, 1969
67. Kolin-Sorensen V: Treatment of trigger fingers. *Acta Orthop Scand* 41:428, 1970
68. Laha RK, Lunsford LD, Dujovny M: Lacertus fibrosus compression of the median nerve: A case report. *J Neurosurg* 48:838, 1978
69. Lahey MD, Aulicio PL: Anomalous muscles associated with compression neuropathies. *Orthop Rev* 15:199–207, 1986
70. Leadbetter WB: Cell matrix response in tendon injury. *Clin Sports Med* 11:533–578, 1992
71. Leslie BM, Ericson WB, Morehead JR: Incidence of a septum within the first dorsal compartment of the wrist. *J Hand Surg [Am]* 15:88, 1990
72. Linburg RM, Comstock BE: Anomalous tendon slips from the flexor pollicis longus to the flexor digitorum profundus. *J Hand Surg [Am]* 4:79–83, 1989

73. Linscheid RL, Dobyns JH: Athletic injuries of the wrist. *Clin Orthop* 198:141-151, 1985
74. Lipscomb PR: Tenosynovitis of the hand and wrist: Carpal tunnel syndrome, de Quervain's disease, trigger digit. *Clin Orthop* 13:164, 1959
75. Lister G: *The Hand*, ed 2. Edinburgh, Churchill Livingstone, 1984, p 244
76. Long RR: Nerve anatomy and diagnostic principles. *In* Pappas AM (ed): *Upper Extremity Injuries in the Athlete*. New York, Churchill Livingstone, 1995, pp 53-54
77. Lorthior J: Surgical treatment of trigger-finger by a subcutaneous method. *J Bone Joint Surg Am* 40:793-795, 1958
78. Lotem M, Fried A, Levy M: Radial palsy following muscular effort. *J Bone Joint Surg Br* 53:500, 1971
79. Lundborg G, Meyers R, Powell H: Nerve compression and increased fluid pressure: A "miniature compartment syndrome." *J Neurol Neurosurg Psychiatry* 46:119-124, 1983
80. Mackinnon SE, Dellon AL: Experimental study of chronic nerve compression. *Hand Clin* 2:639-650, 1986
81. Mackinnon SE, Dellon AL: The overlap pattern of the lateral antebrachial cutaneous nerve and the superficial radial nerve. *J Hand Surg [Am]* 10:522-526, 1985
82. Magill RM, Bassini-Lipson L, Patel MR: Digital stenosing tenosynovitis, comparison of treatment with splinting and injections. *In* American Society for Surgery of the Hand Proceedings, Toronto, 1990
83. Manske FR: Compression of the radial nerve by the triceps muscle. *J Bone Joint Surg Am* 59:835, 1977
84. Massey EW, Pleet AB: Handcuffs and cheiralgia paresthetica. *Neurology* 28:1312-1313, 1978
85. McCue FC III, Bruce JF Jr: Hand and Wrist. *In* Delee JC, Drez D Jr (eds): *Orthopaedic Sports Medicine: Principles and Practice*. Philadelphia, WB Saunders, 1994, pp 913-1017
86. McPherson SA, Meals RA: Cubital tunnel syndrome. *Orthop Clin North Am* 23:111-123, 1992
87. Mitsunga MM, Nakano K: High radial nerve palsy following strenuous muscular activity. *Clin Orthop* 234:39, 1988
88. Mogensen BA, Mattson HS: Stenosing tendovaginitis of the third compartment of the hand: Case report. *Scand J Plast Reconstr Surg Hand Surg* 14:127, 1980
89. Moss SH, Switzer HE: Radial tunnel syndrome: A spectrum of clinical presentations. *J Hand Surg [Am]* 8:414-420, 1983
90. O'Brien M: Functional anatomy and physiology of tendons. *Clin Sports Med* 11:505-520, 1992
91. Osterman AL, Moskow L, Low DW: Soft-tissue injuries of the hand and wrist in racquet sports. *Clin Sports Med* 7:329-348, 1988
92. Palmieri TJ: Pisiform area pain treatment by pisiform excision. *J Hand Surg [Am]* 7:477-480, 1982
93. Parker RD, Berkowitz MS, Brahams MA, et al: Hook of the hamate fracture in athletes. *Am J Sports Med* 14:517, 1968
94. Pechan J, Julis I: The pressure measurement in the ulnar nerve: A contribution to the pathophysiology of the cubital tunnel syndrome. *J Biomech* 8:75-79, 1975
95. Pickering TG: Runner's radial palsy. *N Engl J Med* 304:768, 1981
96. Pitner MA: Pathophysiology of overuse injuries in the hand and wrist. *Hand Clin* 6:355-364, 1990
97. Posner MA: Compressive neuropathies of the median and radial nerves at the elbow. *Clin Sports Med* 9:343-363, 1990
98. Press JM, Weisher SL: Prevention: Conditioning and orthotics. *Hand Clin* 6:383, 1990
99. Pyne JIB, Adams BD: Hand tendon injuries in athletics. *Clin Sports Med* 11:833-850, 1992
100. Quinnell RC: Conservative management of trigger finger. *Practitioner* 224:187-190, 1980
101. Rayan GM: Distal stenosing tenosynovitis. *J Hand Surg [Am]* 15:973-975, 1990
102. Regan WD, Morrey BF: Entrapment neuropathies about the elbow. *In* Delee JC, Drez D Jr: *Orthopaedic Sports Medicine: Principles and Practice*. Philadelphia, WB Saunders, 1994, pp 844-859

103. Rennels GD, Ochoa J: Neuralgic amyotrophy manifesting as anterior interosseous nerve palsy. *Muscle Nerve* 3:160, 1980
104. Rettig AC: Neurovascular injuries in the wrists and hands of athletes. *Clin Sports Med* 9:389-417, 1990
105. Rettig AC, Patel DV: Epidemiology of elbow, forearm, and wrist injuries in the athlete. *Clin Sports Med* 14:289-297, 1995
106. Rhoades C, Gelberman R, Manjarrin J: Stenosing tenosynovitis of the fingers and thumb. *Clin Orthop* 190:236, 1987
107. Riordan DC, Stokes HM: Synovitis of the extensors of the fingers associated with an extensor brevis manus muscle. *Clin Orthop* 95:278-280, 1973
108. Ritter MA, Inglis AE: The extensor indicis proprius syndrome. *J Bone Joint Surg Am* 51:1645-1650, 1969
109. Ritts GD, Wood MB, Linscheid RL: Radial tunnel syndrome: A ten-year surgical experience. *Clin Orthop* 219:201, 1987
110. Roles N, Maudsley R: Radial tunnel syndrome: Resistant tennis elbow as nerve entrapment. *J Bone Joint Surg Br* 54:499-508, 1972
111. Rowland SA: Acute traumatic subluxation of the extensor carpi ulnaris tendon at the wrist. *J Hand Surg [Am]* 11:809-811, 1986
112. Saplys R, Mackinnon SE, Dellon LA: The relationship between nerve entrapment versus neuroma complications and the misdiagnosis of de Quervain's disease. *Contemporary Orthopedics* 15:51, 1987
113. Schenk RR: Variations of the extensor tendons of the finger. *J Bone Joint Surg Am* 46:103-110, 1964
114. Seddon JH: Three types of nerve injury. *Brain* 66:237, 1943
115. Seradge H, Kleinert HE: Reduction flexor tenoplasty. Treatment of stenosing flexor tenosynovitis distal to the first pulley. *J Hand Surg [Am]* 6:543-544, 1981
116. Sicuranza MJ, McCue FC: Compressive neuropathies in the upper extremity of athletes. *Hand Clin* 8:263-273, 1992
117. Spinner M: The arcade of Fröhse and its relationship to posterior interosseous nerve paralysis. *J Bone Joint Surg Br* 50:809-812, 1968
118. Spinner M: The anterior interosseous nerve syndrome with special attention to its variations. *J Bone Joint Surg Am* 52:84-94, 1970
119. Spinner M, Kaplan EB: Extensor carpi ulnaris: Its relationship to the stability of the distal radioulnar joint. *Clin Orthop* 68:124-129, 1970
120. Spinner M, Linscheid RL: Nerve entrapment syndrome. In Morrey BF (ed): *The Elbow and its Disorders*. Philadelphia, WB Saunders, 1993, pp 813-832
121. Spinner M, Olshansky K: The extensor indicis proprius syndrome: A clinical test. *Plast Reconstr Surg* 51:134-138, 1973
122. Sponseller PD, Engber WD: Double entrapment radial tunnel syndrome. *J Hand Surg [Am]* 8:420-423, 1983
123. Stern PJ: Tendinitis, overuse syndromes, and tendon injuries. *Hand Clin* 6:467-476, 1990
124. Strandell G: Variations of the anatomy in stenosing tenosynovitis at the radial styloid process. *Acta Chir Scand* 113:234-240, 1957
125. Sunderland S: *Nerves and Nerve Injuries*. Baltimore, Williams & Wilkins, p. 749, 1968
126. Swiggett R, Ruby LK: Median nerve compression of the lacertus fibrosus: Report of three cases. *J Hand Surg [Am]* 11:700, 1986
127. Szabo RM, Gilberman RH: The pathophysiology of nerve entrapment syndromes. *J Hand Surg [Am]* 12:880, 1987
128. Szabo RM, Madison M: Carpal tunnel syndrome. *Orthop Clin* 23:103-109, 1992
129. Thorpe AP: Results of surgery for trigger finger. *J Hand Surg [Br]* 13:199, 1988
130. Thorson E, Szabo RM: Common tendinitis problems in the hand and forearm. *Orthop Clin North Am* 23:65-74, 1992
131. Torisu T: Fracture of the hook of the hamate by a golf swing. *Clin Orthop* 83:91, 1972
132. Van Rossum J, Buruma OJS, Kamphuisen HAC, et al: Tennis elbow—A radial tunnel syndrome? *J Bone Joint Surg Br* 60:197-198, 1978
133. Walker PO, Troost BT: Push-up palmar palsy. *JAMA* 259:48, 1988