

# Upper Extremity Nerve Entrapment Syndromes in Sports: an Update

Shane Cass, DO

## Abstract

Peripheral nerve entrapment syndromes are difficult diagnoses to make. Symptoms are often vague and mimic other musculoskeletal pathology. Clinicians' best diagnostic tools for entrapment neuropathies are a good history, physical examination, and anatomical knowledge. Neurodiagnostic testing and advanced imaging have some role to play in entrapment neuropathies, but these are not always necessary or helpful. Most entrapment neuropathies resolve with nonoperative treatment. This article will look at upper extremity entrapment neuropathies and review updated literature from the last decade.

## Introduction

Upper extremity nerve entrapment syndromes are a difficult diagnosis for clinicians to make, usually due to their vague and variable symptoms. The exact incidence of entrapment neuropathies in sports is not well known, but the upper extremity accounts for greater than 80% of the total amount (9). Entrapment neuropathies can be acute and caused by trauma or chronic due to overuse or sport-specific risk factors.

There are numerous articles that review specific entrapment neuropathies in detail (1,3,5–9,15,18,22,23). This article will review in brief the history, examination, diagnostic studies, and treatment of entrapment neuropathies while discussing new literature and findings within the last decade. This review will focus on mononeuropathies; therefore thoracic outlet syndrome and brachial plexus injuries will not be discussed.

## Spinal Accessory Nerve

Spinal accessory (SA) nerve injuries are considered rare in sports. Accessory nerve injuries have been reported in judo, karate, and kickboxing, usually due to direct trauma leading to a nerve contusion (26). The SA nerve is the lone motor innervation of the trapezius muscle. Upon leaving the

jugular foramen, the nerve has a relatively vulnerable subcutaneous course until reaching the trapezius. Patients often complain of pain, weakness, and deformity if scapular winging occurs. There is lack of ability to elevate or fully abduct the arm above the head. They may have a drooping shoulder, neckline asymmetry, and winging of the scapula. On gross examination, muscles might show asymmetry or atrophy. Active forward flexion should

be assessed for winging. Winging due to SA neuropathy appears on active shoulder abduction with winging of the superior medial angle. Classically there is no winging with forward flexion, differentiating it from a long thoracic nerve (LTN) injury (23). There may be a weakness with the shoulder shrug, but this can be overcome with the levator scapulae. Chan and Hems (3) conducted a six-person case study reporting resisted external rotation as a beneficial test to finding winging from SA palsy.

Imaging usually is not helpful. Neurodiagnostic tests are the most helpful studies for confirming diagnosis of trapezius dysfunction (28). Outcomes for nontraumatic SA neuropathies are generally good, despite severity of electrodiagnostic findings. Worse outcomes in one study were correlated more with traumatic neuropathies, involvement of the dominant limb, scapular winging, and impaired arm elevation (12). Treatment usually is attempted conservatively with rehabilitation to strengthen accessory muscles and improve drooping and forward flexion. Surgical indications are chronic pain and scapular instability for at least one year after injury or three months in iatrogenic trapezius paralysis. The most common surgical procedure is transfer of the levator scapulae or rhomboids to substitute for the trapezius. To date, there are no randomized trials, but case studies show promising results (23).

## Long Thoracic Nerve

The LTN travels subcutaneously along the chest wall to innervate the serratus anterior muscle. Injury can occur from trauma or due to overuse. In athletics, repetitive microtrauma due to nerve traction is a common cause of injury. LTN palsy has been seen in archery, bodybuilding

---

Sports Medicine, University of New Mexico, Albuquerque, NM 87131

Address for correspondence: Shane Cass, DO, Sports Medicine, University of New Mexico, Albuquerque, NM; E-mail: shanepcass@gmail.com, scass@salud.unm.edu.

1537-890X/1301/16–21

Current Sports Medicine Reports

Copyright © 2014 by the American College of Sports Medicine

(due to increased bulk in the serratus anterior), volleyball, tennis, football, wrestling, judo, karate, and kickboxing (26). Athletes often complain of pain in the shoulder, scapula, or neck, which is usually worsened with activity that would cause traction on the nerve. They may note scapular winging when sitting against hard chairs. Examination usually demonstrates decreased forward flexion strength and winging. The winging is usually at the inferior border and worsened with forward flexion. A good way to see the winging is with wall push-ups. Nerve conduction studies can be challenging, but electromyogram (EMG) is generally helpful in detecting problems in the serratus anterior (28). Plain films may be helpful to look for a cervical rib. The natural history for recovery of atraumatic LTN neuropathy is good with resolution often in 1 year (12). Therefore treatment should start conservatively. This includes rest, symptom management, reassurance, and rehabilitation. Rehabilitation includes maintaining range of motion of the shoulder and strengthening of scapular stabilizing and compensatory muscles (trapezius, rhomboids, and levator scapulae). Surgical indications include symptoms lasting longer than a year and no EMG improvement in serratus function. Although no randomized controlled trials have been done, case series have reported good clinical outcomes for pain, function, and range of motion with pectoralis major tendon transfer (23,25). A small retrospective analysis of patients undergoing neurolysis of the LTN showed quick and significant improvement in serratus anterior functioning, even at study follow-up 2 to 3 years later (19).

### **Axillary Nerve**

Axillary nerve injury in sports is fairly rare. It accounts for less than 1% of all nerve injuries. Traumatic football injuries accounts for the majority of injuries, with hockey, martial arts, rugby, and wrestling also reporting traumatic injuries. Baseball and volleyball also have been reported due to compression and stretching of the axillary nerve.

### **Traumatic Injury to Axillary Nerve**

Traumatic injuries occur from dislocations or fractures. Axillary nerve injury from dislocations occurs 19% to 55% of the time and up to 58% for proximal humeral fractures (22). Direct blunt trauma to the anterolateral shoulder also can cause injury. Athletes may have no symptoms at rest but notice they weaken and fatigue easily with overhead activity and lifting. There may be numbness on the lateral arm and reduced abduction strength. The examination should include range of motion and deltoid strength evaluation as well as a careful neurological examination. Plain radiographs can be sought to evaluate for fractures and confirm reduction. EMG/nerve conduction studies (NCS) are helpful to confirm the diagnosis and stage severity and recovery. It is important to realize that these studies will be normal until at least 3 wk after injury. If neurodiagnostic abnormalities are found in the deltoid, they should be repeated in 3 to 4 months to document improvement (28). Within 6 months to a year, most patients with this injury recover with conservative treatment. Reassurance, rest, range of motion, and symptom management are the usual treatment. Electrical

stimulation may be a promising intervention to delay or prevent muscle atrophy (22).

### **Quadrilateral Space Syndrome**

Quadrilateral space syndrome involves compression of the axillary nerve and/or the posterior circumflex artery within the quadrilateral space. This space is defined by the teres minor superiorly, long head of the triceps medially, humerus laterally, and teres major inferiorly. There are rare reported cases in the literature. Pathophysiology without trauma can occur from direct compression (most commonly due to fibrous bands), direct trauma to the posterior shoulder, or traction injury of the upper extremity. The presentation and examination are often vague and fairly nonspecific. Pain is usually dull, burning, or described as a deep ache localized to the lateral and posterior portion of the shoulder. It usually is worsened with activity, and there is weakness with overhead activity. On examination, there may be pain in the quadrilateral space, reproduction of symptoms with forward flexion, or atrophy. There may be weakness with deltoid and teres minor testing.

Safran (22) advocates an arteriogram with arm in abduction and external rotation to demonstrate lack of flow in the posterior circumflex humeral artery to confirm diagnosis. Due to the intermittent nature of compression, neurodiagnostic testing is often negative but can demonstrate denervation in chronic disease (28). Magnetic resonance imaging (MRI) may demonstrate a space-occupying lesion in the quadrilateral space or show signal alteration or atrophy of the teres minor or deltoid (10). Without a space-occupying lesion, it is believed this has a good chance of resolving nonoperatively. Rehabilitation should focus on stretching the posterior shoulder, concentrating on the teres minor. An evaluation of the athlete's biomechanics (throwing, serving) should be reviewed also. Consider surgery if symptoms last past six months despite aggressive conservative management and if confirmed by an arteriogram (22). Surgery involves decompression in the deltoid and excision of fibrous bands or space-occupying lesions. There are no large case series or randomized surgical trials relating to this syndrome.

### **Suprascapular Nerve**

Once thought to be an uncommon occurrence, suprascapular nerve (SSN) entrapment is increasingly being recognized as a cause of shoulder pain. Up to 2% of all shoulder disorders might be related to SSN injury (22). It is most common in volleyball players and also seen in baseball, notably pitchers. A study of international level volleyball players showed up to 45% prevalence of SSN injury with documented asymptomatic atrophy and weakness (26,17). It also has been seen in basketball, dance, bodybuilding, football, and wrestling (26). The most common areas of entrapment are at the suprascapular notch and the spinoglenoid notch. Narrow notches and pathologic transverse scapular or spinoglenoid ligaments contribute to causes of compression. One of the best described causes for compression arises from a paralabral cyst due to injury, usually at the spinoglenoid notch. Recently large retracted rotator cuff tears are being recognized also as causes for

SSN injury. This largely is due to traction injury on the nerve at the suprascapular notch (17).

Diagnosis can be difficult as symptoms are usually similar to common causes of shoulder pain, such as rotator cuff disease. Also there is often coexistent pathology. A high index of suspicion should be held for young overhead athletes and those with large rotator cuff tears. It is described as a deep, dull, chronic pain and is localized usually to the superior, posterior, and lateral aspects of the shoulder. Compression at the suprascapular notch is tolerated less with a significant loss of abduction and external rotation strength due to supraspinatus involvement. Compression at the spinoglenoid notch is tolerated better, likely due to compensation from the posterior deltoid and teres minor. Atrophy of the supraspinatus and infraspinatus muscles indicates entrapment at the suprascapular notch versus isolated infraspinatus atrophy due to spinoglenoid notch involvement.

When considering diagnostic studies, MRI can be useful to detect soft tissue masses and look for indirect signs of denervation (10). The most useful diagnostic examination for suspected SSN injury is EMG and NCS (28). EMG/NCS can be used to confirm the diagnosis, test the function of the supraspinatus/infraspinatus in those with atrophy, and monitor nerve function. Sensitivity and specificity vary in studies from 74% to 91% (17). A nerve block at the suprascapular or spinoglenoid notch with relief of pain also can help in the diagnosis.

The natural history of SSN injury is unknown, but in the absence of structural lesions, there is usually spontaneous resolution in a year with multiple case studies documenting good results with nonoperative treatment. NSAIDs, relative rest, activity modification, and rehabilitation are main stays of treatment. Rehabilitation should focus on rotator cuff, deltoid, and periscapular stretching and strengthening with scapular stabilization exercises (17). With structural causes of entrapment, such as paralabral cysts, surgery is advocated. Surgical repair of the labrum may alleviate the cyst and compression. Surgery should be considered in patients without a cyst who have continued pain and weakness after 6 months of conservative rehabilitation. Nerve decompression in the suprascapular or spinoglenoid notch shows promise in a recent case series of patients undergoing this surgery (24).

## **Median Nerve**

### **Pronator Syndrome**

The pronator syndrome (PS) is a compression neuropathy of the median nerve as it passes through the two heads of the pronator teres muscle or under the proximal edge of the flexor digitorum superficialis (FDS) arch. The sites of compression also can be from a ligament of Struthers or the lacertus fibrosus (bicipital aponeurosis) in the antebrachial fossa. PS has been seen in archers (from repetitive flexion of the bow), pitchers, tennis, and bodybuilders (26). Athletes often present with aching pain in the proximal volar forearm. Usually they have paresthesias in the thumb, index finger, middle finger, and radial half of the ring finger, similar to carpal tunnel syndrome (CTS). Often they report pain with pronation and supination activities. Their symptoms do not have a nocturnal component, as in CTS, and

they typically have numbness over the thenar eminence. (The palmar cutaneous branch of the median nerve arises proximal to the transverse carpal ligament.) There also should be no Tinel sign over the wrist or a Phalen sign (6). If resisting pronation of the forearm in a neutral position reproduces symptoms, there could be entrapment at the pronator teres. Another useful test is to apply direct pressure in the area of the pronator with the forearm supinated. Paresthesia in the median nerve distribution within 1 min is considered positive (20). Resisted contraction of the flexor digitorum superficialis in the middle finger with reproduction of symptoms may indicate compression at the fibrous arch. Lastly resisted flexion of the forearm in full supination may correspond to compression proximally at the lacertus fibrosus (7). Neurodiagnostic studies for PS are unreliable and normal in 50% of symptomatic patients with PS (11). MRI is usually not helpful but can demonstrate a pattern of muscle denervation or a mass (10). Small case series demonstrate up to 70% recovery with conservative management (21). This includes relative rest, NSAIDs, and therapy. The literature describes a local injection of corticosteroid but no studies demonstrate its efficacy. Surgical success rates are variable and include nerve decompression and removal of any space-occupying lesions (7,21).

### **Anterior Interosseous Nerve Syndrome**

Anterior interosseous nerve (AIN) syndrome is compression of the motor branch of the median nerve to the deep muscles of the hand, flexor pollicis longus (FPL), flexor digitorum profundus (FDP) to the index and middle fingers, and pronator quadratus. Compression may occur at the FDS origin, flexor carpi radialis origin, deep into the pronator teres head, or by an accessory head of the FPL (Gantzer's muscle). AIN syndrome is rare in sports with limited documentation in the literature. The AIN is purely motor, so there is no sensory loss. Patients notice a loss of FPL function and pain in the proximal volar forearm. A classic examination finding is inability to make an "OK" sign, due to lack of ability to flex thumb interphalangeal (IP) joint and index finger distal interphalangeal (DIP) joint. Symptoms may be provoked with resisted elbow flexion, resisted forearm pronation, and resisted finger flexion (20). Neurodiagnostic studies are often helpful with EMG showing irregularities in pronator quadratus, FPL, or median branch of FDP (11). MRI is usually not necessary but may show signal intensity changes related to muscle denervation (10). There is controversy regarding surgery and natural time course of spontaneous healing. It is thought that spontaneous recovery with nonoperative treatment should occur within 6 to 12 months, with literature describing up to 18 months for spontaneous recovery and better healing in patients younger than 40 years (5). Surgical literature consists of uncontrolled, case-based studies with varying results of median nerve decompression (5,7).

### **CTS**

CTS is the most common entrapment neuropathy in the normal population as well as sports. Incidence rates are very high in wheelchair athletes, in cyclists, in wrestlers, and in football from blocking technique (26). It is due to compression of the median nerve, which is deep into the

transverse retinacular ligament. Patients present with forearm, wrist, and hand pain as well as paresthesias in the median nerve distribution (sparing the thenar eminence). The paresthesias are worsened usually at night. Later in the disease course, there may be thenar atrophy and weakness of the grip or pinch. Traditionally Phalen sign (symptom reproduction with bilateral wrist flexion) and Tinel sign at the wrist have been used as physical examination findings for diagnosing CTS, with Phalen's being the most reliable clinical test. Electrodiagnostic studies have a reported 85% to 90% accuracy for CTS and can be useful for diagnosis (11). MRI can be useful in detecting a space-occupying lesion, but otherwise, it has fairly low sensitivity and specificity for CTS (10). The American Academy of Orthopedic Surgeons published recommendations for diagnosing CTS, the highest level of recommendation being electrodiagnostic testing if clinical evaluation is positive and surgery is being considered (14). Conservative treatment includes activity modification, volar splinting, rehabilitation for range of motion, and corticosteroid injection. A Cochrane review demonstrated greater improvement in symptoms after a corticosteroid injection compared to placebo after 1 month, but its benefits have not been demonstrated past that (16). Another Cochrane review, including four randomized controlled trials, looked at surgical versus nonsurgical treatment. Their pooled results showed that surgery improved symptoms better at 1 year than splinting (27). Surgical decompression is the surgery of choice, and typically the evidence shows no benefit over arthroscopic release versus open.

## **Ulnar Nerve**

### **Cubital Tunnel Syndrome**

Cubital tunnel syndrome is ulnar nerve entrapment at the elbow. Cubital tunnel syndrome is the second most common entrapment neuropathy, second to CTS, in both sports and the general population. It is thought to be caused by compression and traction of the ulnar nerve and is seen in baseball, bodybuilding, football, and wrestling (26). Compression can occur at the arcade of Struthers (present in 70% of patients), medial intermuscular septum, medial epicondyle, cubital tunnel, anconeus, and fibrous bands in the flexor carpi ulnaris. Patients present with a vague discomfort localized to the medial elbow, paresthesias in the ring and small fingers of the hand, and weakness in pinching or gripping. They may have fatigue with repetitive tasks and worsening symptoms with activities requiring elbow flexion. There are often nocturnal symptoms.

Physical examination may show atrophy of intrinsic hand muscles (especially the first dorsal interosseous muscle). Clawing of the small and ring fingers is found when denervation is severe. There may be sensory loss in the fourth and fifth finger. Strength should be assessed, being mindful of intrinsic muscles of the hand and the deep flexors to the ring and small finger. The Wartenburg sign is the inability to fully adduct the small finger with finger held slightly abducted and extended. With Froment sign, patients are asked to perform a pinch, with a positive test being flexion of the IP joint of the thumb to compensate for weakness of the adductor pollicis by using the FPL (median nerve innervated). Provocative testing can be done with Tinel testing over the course of the nerve, having patients hold their

elbow in maximal flexion while the examiner holds pressure on the ulnar nerve at the cubital tunnel and a test called the scratch-collapse test. With this test, both elbows are at the side and the patient externally rotates against resistance with elbows flexed. The examiner then gently scratches along the course of the ulnar nerve at the elbow and reapplies resistive force. A positive test happens when the patients have a momentary loss in ability to externally rotate. A controlled trial of 169 patients showed highest levels of negative predictive value (98%) and higher sensitivity and specificity with the scratch-collapse test compared to Tinel and elbow flexion/compression (4).

Electrodiagnostic testing is usually very helpful. EMG and NCS can be used to determine the precise site of the neuropathy and confirm localization (8). Nonoperative treatment should at first be attempted as up to an 89% improvement rate has been demonstrated (15). A Cochrane review demonstrated that giving information on avoiding prolonged movements or positions causing traction and compression on the cubital tunnel (avoid full flexion and direct pressure) was effective for mild to moderate symptoms (2). Night splinting (flexion-blocking splints) and referral to occupational therapy for nerve-gliding exercises have been shown to be helpful in some studies (15). For those who do not respond to conservative treatment, surgery can be considered, including decompression of the ulnar nerve and transposition. A Cochrane review found no difference between simple decompression and transposition of the ulnar nerve but higher rates of infections in the transposition cases (2).

### **Ulnar Tunnel Syndrome**

Ulnar tunnel syndrome refers to compression of the ulnar nerve in Guyon canal at the level of the wrist. Guyon canal is bordered by the volar carpal ligament, by the transverse retinacular ligament, by pisiform, and laterally by the hook of the hamate (9). In sports, it is most seen commonly in cyclists, especially long-distance cyclists due to compression and gripping of the handlebars. A cross-sectional study of 160 professional cyclists demonstrated a 30% incidence of ulnar neuropathy (26). It also has been seen in wheelchair sports, cross-country skiing, weight lifting (heavy bench presses), and racquet sports (tennis, baseball, and golf) from fractures to the hamate. Compression may occur from space-occupying lesions such as ganglion cysts, hook of the hamate and pisiform fractures, ulnar artery aneurysms, and deviant hypothenar muscles.

Presentation may include pure motor, sensory, or mixed findings, depending on the area of compression. Proximal compression before any nerve bifurcation causes motor weakness of ulnar innervated intrinsic muscles and sensory deficits over the hypothenar eminence and small and ring finger. Compression deep in Guyon canal causes pure muscle weakness, as the superficial sensory branch is spared. Pure sensory symptoms occur from compression of the superficial branch of the ulnar nerve (1). Examination should include good motor testing, paying attention to intrinsic muscles, and any potential atrophy, as well as a good sensory examination. Tinel and Phalen tests are used often as provocative tests, but their effectiveness has not been verified (1). Plain films may be helpful if there is suspicion of a

hamate fracture. MRI can be used to rule out space-occupying lesions and evaluate areas of muscle denervation (10). EMG can be helpful for confirmation and localization of the area of compression. Conservative treatment should first be tried including splinting, padding of gloves and handlebars, and activity modification (frequent changing of hand position on handlebars or grips). Surgery should be considered for conservative failures and includes removal of space-occupying lesions and decompression of the ulnar tunnel (1).

## Radial Nerve

### Radial Tunnel Syndrome

Radial tunnel syndrome (RTS) is a pure pain syndrome that refers to entrapment of the radial nerve in the radial tunnel. The radial tunnel is a potential space located anterior to the proximal radius. Its diagnosis is controversial, and many clinicians do not subscribe to its existence. This mainly is due to a lack of electrodiagnostic and pathophysiologic findings. This diagnosis is reported rarely in sports but has been seen in tennis and bodybuilding (26). The radial nerve splits into the radial sensory nerve and the posterior interosseous nerve (PIN) proximal to the supinator at the elbow. The PIN is the terminal motor branch of the radial nerve. It can be compressed at the elbow at the arcade of Frohse, the medial edge of the extensor carpi radialis brevis, the radial recurrent blood vessels, and the inferior margin of the supinator muscle (18). Patients present with pain along the dorsoradial aspect of the proximal forearm. They tend to have increased pain with activities causing supination and pronation. There may be muscle weakness, but this is thought to be due to pain, and there are no sensory symptoms. The hallmark diagnostic finding is focal tenderness over the anatomic landmark of the PIN. This area is 3 to 5 cm distal to the lateral epicondyle over the supinator mass, often mistaking RTS with lateral epicondylitis (18). Specificity and sensitivity of other provocative tests have not been established (18). MRI is typically not useful, but denervation or atrophy in the supinator muscle or extensor muscles was seen in one case series (18). Electrodiagnostic studies are frequently not helpful. NCS are usually normal. EMG can be helpful if positive for denervation changes in muscles. There are limited high-quality studies on RTS treatment. Conservative treatment may be considered initially and includes splinting, activity modification (avoiding prolonged elbow extension with forearm pronation and wrist flexion), and NSAIDs. The optimal period of time to try conservative treatment is not known but should be implemented for 3 to 6 months before considering surgery (18).

### PIN Syndrome

In contrast to RTS, PIN syndrome has clear motor loss and neurodiagnostic findings. It is thought to occur from compression of the PIN as it pierces the supinator at the arcade of Frohse. PIN syndrome has been reported in tennis players, bodybuilders, swimmers, and gymnasts (26). Patients present with dropped fingers and lack of thumb extension. Extensor carpi radialis longus is usually preserved (due to innervation proximal to PIN branching), so wrist extension and radial deviation will be present still (6). Examination findings show lack of strength with thumb ex-

tension and extension of the metacarpophalangeal (MCP) joints (20). Resisted middle finger extension can cause pain and should be differentiated from lateral epicondylitis. As above, MRI is most useful for finding compressive lesions. EMG is usually positive for diagnosing PIN syndrome (6). As with RTS, initial treatment is conservative and includes rest, activity modification, splinting, NSAIDs, and stretching. If there is no significant improvement in 3 months with conservative treatment, surgical consultation should be sought. A systematic review of treatment for PIN syndrome turned out two high-quality studies showing some effectiveness of nerve decompression for treatment of PIN (13).

## Conclusion

Peripheral nerve entrapment in sports is more common than once suspected. Clinicians should consider these diagnoses in their differential of an injured athlete. Diagnosis hinges on thorough knowledge of the anatomy and typical presentation of these syndromes and a good history and physical examination. Most patients will respond to conservative treatment, and this should be tried first in most cases of peripheral nerve entrapment. No improvement may necessitate surgical treatment.

There are no additional sources of funding for this article and no financial conflicts of interest.

## References

1. Bachoura A, Jacoby SM. Ulnar Tunnel Syndrome. *Orthop. Clin. North Am.* 2012; 43:467–74.
2. Caliendo P, La Torre G, Padua R, et al. Treatment for ulnar neuropathy at the elbow. *Cochrane Database Syst. Rev.* 2012, Issue 7. Art. No.: CD006839. DOI: 10.1002/14651858.CD006839.pub3.
3. Chan PK, Hems TEJ. Clinical signs of accessory nerve palsy. *J. Trauma.* 2005; 60:1142–4.
4. Cheng CJ, Mackinnon-Patterson B, Beck JL, Mackinnon SE. Scratch collapse test for the evaluation of carpal and cubital tunnel syndrome. *J. Hand Surg.* 2008; 33A:1518–24.
5. Chi Y, Harness NG. Anterior interosseous nerve syndrome. *J. Hand Surg.* 2010; 35A:2078–80.
6. Dang AC, Rodner CM. Unusual compression neuropathies of the forearm, part I: Radial nerve. *J. Hand Surg.* 2009; 34A:1906–14.
7. Dang AC, Rodner CM. Unusual compression neuropathies of the forearm, part II: Median nerve. *J. Hand Surg.* 2009; 34A:1915–20.
8. Dimberg EL. Electrodiagnostic evaluation of ulnar neuropathy and other upper extremity mononeuropathies. *Neurol. Clin.* 2012; 30:479–503.
9. Dimeff RJ. Entrapment neuropathies of the upper extremity. *Curr. Sports Med. Rep.* 2003; 2:255–61.
10. Dong Q, Jacobson JA, Jamadar DA, et al. Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. *Radiol. Res. Pract.* 2012; 2012:230679. doi: 10.1155/2012/230679. Epub 2012 Oct 17.
11. Freedman M, Helber G, Pothast J, et al. Electrodiagnostic evaluation of compressive nerve injuries of the upper extremities. *Orthop. Clin. North Am.* 2012; 43:409–16.
12. Friedenberg SM, Zimprich T, Harper MC. The natural history of long thoracic and spinal accessory neuropathies. *Muscle Nerve.* 2002; 25:535–9.
13. Huisstede BM, Miedema HS, Van Opstal T, et al. Interventions for treating the posterior interosseous nerve syndrome: a systematic review of observational studies. *J. Peripher. Nerv. Syst.* 2006; 11:101–10.
14. Keith MW, Masera V, Chung K, et al. Diagnosis of carpal tunnel syndrome. *J. Am. Acad. Orthop. Surg.* 2009; 17:389–96.
15. Kroonen LT. Cubital tunnel syndrome. *Orthop. Clin. North Am.* 2012; 43:475–86.

16. Marshall SC, Tardif G, Ashworth NL. Local corticosteroid injection for carpal tunnel syndrome. *Cochrane Database Syst. Rev.* 2007, Issue 2. Art. No.: CD001554. DOI: 10.1002/14651858.CD001554.pub2.
17. Moen TC, Babatunde OM, Hsu SH, *et al.* Suprascapular neuropathy: what does the literature show? *J. Shoulder Elbow Surg.* 2012; 21:835–46.
18. Nash NH, Nemani S. Radial tunnel syndrome. *Orthop. Clin. North Am.* 2012; 43:529–36.
19. Nath RK, Melcher SE. Rapid recovery of serratus anterior muscle function after microneurolysis of long thoracic nerve injury. *J. Brachial Plex. Peripher. Nerve Inj.* 2007; 2:4. doi:10.1186/1749-7221-2-4.
20. Popinchalk SP, Schaffer AA. Physical examination of upper extremity compressive neuropathies. *Orthop. Clin. North Am.* 2012; 43(4):417–30.
21. Presciutti S, Rodner CM. Pronator syndrome. *J. Hand Surg. Am.* 2011; 36A:907–9.
22. Safran MR. Nerve injury about the shoulder in athletes, part 1: suprascapular nerve and axillary nerve. *Am. J. Sports Med.* 2004; 32:803–19.
23. Safran MR. Nerve injury about the shoulder in athletes, part 2: Long thoracic nerve, spinal accessory nerve, burners/stingers, thoracic outlet syndrome. *Am. J. Sports Med.* 2004; 32:1063–76.
24. Shaw AA, Butler RB, Sung SY, *et al.* Clinical outcomes of suprascapular nerve decompression. *J. Shoulder Elbow Surg.* 2011; 20:975–82.
25. Streit JJ, Lenarz CJ, Shishani Y, *et al.* Pectoralis major tendon transfer for the treatment of scapular winging due to long thoracic nerve palsy. *J. Shoulder Elbow Surg.* 2012; 21:685–90.
26. Toth C, McNeil S, Feasby T. Peripheral nervous system injuries in sport and recreation. *Sports Med.* 2005; 35:717–38.
27. Verdugo RJ, Salinas RA, Castillo JL, Cea JG. Surgical versus non-surgical treatment for carpal tunnel syndrome. *Cochrane Database Syst. Rev.* 2008, Issue 4. Art. No.: CD001552. DOI: 10.1002/14651858.CD001552.pub2.
28. Williams FH, Kumiga B. Less common upper limb mononeuropathies. *PM R.* 2013; 5(Suppl 5):S22–30. Doi: 10.1016/j.pmrj.2013.03.021. Epub 2013 Mar 21.