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NARRATIVE REVIEW

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A review of upper extremity peripheral nerve injuries in throwing athletes

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Abstract

Peripheral nerve injuries in the upper extremities may be common in throwing athletes as the throwing motion places extreme stress on the dominant arm. The combination of extreme stress along with repetitive microtrauma from throwing uniquely places the throwing athlete at elevated risk of upper extremity peripheral nerve injury. However, because symptoms can be nonspecific and frequently coexist with pathology in the upper extremity, the diagnosis of peripheral nerve injury is often delayed. Diagnosis of peripheral nerve injuries may require a combination of history and physical examination, diagnostic imaging, electrodiagnostic testing, and diagnostic ultrasound-guided injections. The primary management should include physical therapy focusing on throwing mechanics and kinetic chain evaluation. However, some athletes require surgical intervention if symptoms do not improve with conservative management. The purpose of this focused narrative review is to highlight upper extremity peripheral neuropathies reported in throwing athletes and to provide an overview of the appropriate clinical diagnosis and management of the throwing athlete with a peripheral nerve injury.

INTRODUCTION

Throwing-related sports are popular throughout the United States and other countries, but the high rate of popularity also comes with increased injuries.[1-8] The frequency of injury may be quite common, with one report identifying an injury rate of 1.26 injuries per 1000 athletic exposures in high school baseball players over a 3-year period.[9] The most common activities that resulted in injury in this study were fielding (21.6%), running (14.4%), and pitching (13.2%).[9] The shoulder was found to be the most commonly injured area in pitchers, with reports as high as 31%, and composed of muscle strain and ligament sprains in the throwing arm.[9,10]

Although structural soft tissue and bony stress injuries may represent more common forms of injury, physicians who care for throwing athletes must be aware of signs and symptoms of peripheral nerve (PN) injuries. Athletes may present with a combination of symptoms such as numbness, decreased motor function, pain, or even functional decline that may be attributed to a PN injury.[11] The rate of PN injuries has not been established in throwing athletes in any specific sport, but Hirasawa and Sakakida reported an overall rate of 5.7% across sports.[12] Treatment for these injuries includes a combination of rest, anti-inflammatories, and physical therapy. Surgery is often reserved for recalcitrant cases.[11] However, the impairment that follows an undiagnosed PN injury can be detrimental, with one study reporting 9.4% of PN injuries were season ending at the high school level.[13]

Compared to other athletes, those in sports involving the throwing motion possess an inherent risk of increased injuries due to their respective biomechanics.[14-16] The throwing motion places significant stress on the throwing arm. For example, as the thrower progresses through late cocking and into early acceleration, there is a valgus force of over 500 N about the elbow and a torque of 64 Nm.[17,18]Because of coexisting pathology, such as rotator cuff tendinopathy

or internal impingement, the diagnosis of upper extremity neuropathies in throwing athletes is often delayed.[19] Furthermore, upper extremity neuropathies in throwers are often dynamic with specific activity dependent onset and resolution, which lack standardized diagnostic protocols, further delaying diagnosis. This is problematic given that an alteration in the kinetic chain of a thrower because of a compressive neuropathy will affect performance and increase stress on surrounding anatomy, thus increasing the risk for further injury.[12,13,20] The clinician must be cognizant of potential upper extremity nerve injury locations in throwers extending from the brachial plexus into the individual PN branches.

Despite the growing knowledge regarding etiology and associations of PN injuries in overhead throwing athletes, there are still important questions about these injuries including inherent risk factors, mechanisms, and optimal management. The purpose of this narrative review is to discuss the biomechanics of the throwing athlete, understand the presentation of upper limb PN injuries as it relates to throwing, and present available evidence regarding the best approach to evaluating these disorders and guide treatment.

LITERATURE SEARCH

A literature search was conducted by R.L.B. and C.C. in the database Medline (Pubmed) from October 2020 to February 2021. A total of 574 articles were initially identified from the search combinations. A total of 177 articles were determined to be appropriate for this review. Search term combinations included: "peripheral nerve injury," OR "peripheral neuropathy" AND "overhead athlete" OR "throwing athlete" OR "athlete' OR "baseball" OR 'sports'.

Types of literature included in this review were expert opinions, case reports, cohort studies, narrative reviews, systematic reviews, meta-analyses, retrospective cohort studies, prospective comparative studies, and randomized controlled trials. A search for the general diagnosis "peripheral neuropathy" and "peripheral nerve injury" was also completed. Such articles were reviewed by the authors only for information or further literature pertinent to peripheral neuropathies in throwing athletes and were otherwise excluded. The results and discussion of this review are presented in a format familiar to clinicians to help readers synthesize current data in a clinical context.

BIOMECHANICS OF THE THROWING ATHLETE

Throwing is a complex, multisegmental process that relies on whole body kinetics. The lower limb, pelvis, and trunk initiate kinetic force development while serving as a base of support that facilitates the transfer of potential energy to the shoulder complex and elbow and ultimately the ball.[21] There are various phases of throwing that are important to recognize when considering PN injuries in baseball and softball athletes (Figure 1). Windup begins when the pitcher initiates motion and ends when removing the ball from the glove while the thrower has maximum knee lift of the stride leg. Stride (early cocking) typically coincides with the end of the windup phase and ends when the stride leg contacts the ground. Late cocking begins after the stride leg contacts the ground and ends when the throwing shoulder reaches the point of maximum external rotation. Acceleration is an explosive phase in which the arm is at maximum external rotation followed by powerful internal rotation of the shoulder to generate increased peak angular velocities. This is followed by deceleration when the ball is released and ends when the throwing arm reaches 0° of internal rotation. Follow-through is the last phase in which the body's motion continues to move forward until it catches up with the throwing arm.

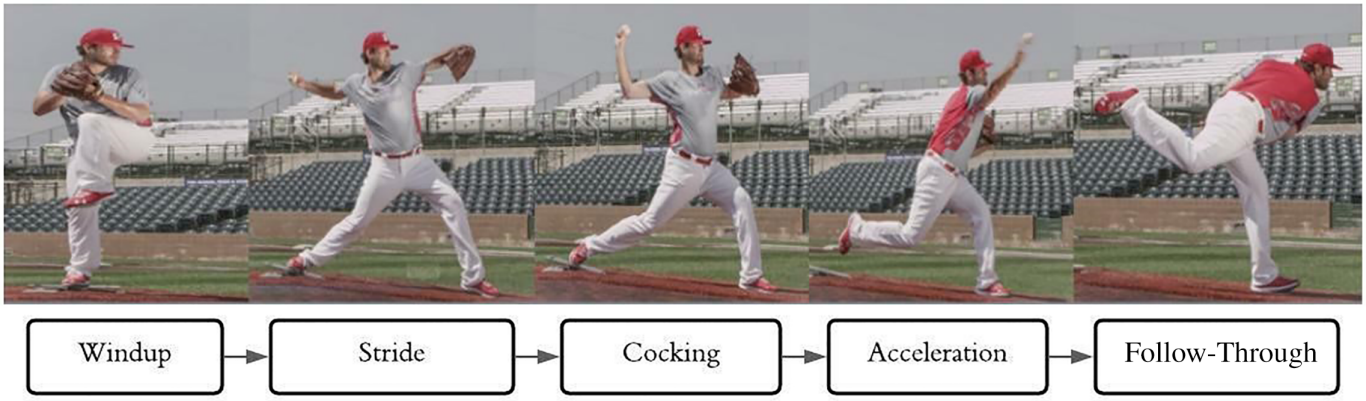

FIGURE 1 Phases of throwing. Image adapted from Zaremski JL, Wasser JG, Vincent HK. Mechanisms and Treatments for Shoulder Injuries in Overhead Throwing Athletes. *Curr Sports Med Rep*. 2017;16(3):179–188

EXAMINATION, EVALUATION, AND MANAGEMENT

History and physical examination

A thorough physical examination should always be conducted as an extension of the history obtained. Throwing athletes may endorse symptoms such as pain, weakness, paresthesia, numbness, or even functional decline. Careful attention should be paid to the location of the symptoms, alleviating or aggravating factors or arm positions, and when in the throwing cycle the symptoms are present.[22] Pitch count, style, changes in velocity, player position, and types of pitches thrown should also be noted.[23]

The medical provider should inspect for any obvious area of atrophy and asymmetry from side to side. Palpation of upper extremities pulses (radial, ulnar, brachial, etc.) should be performed to ensure there is not a vascular etiology to the presenting symptoms. The examination process should follow a systematic routine related to the initial history including inspection, palpation, active and passive joint range of motion, strength testing, special appropriate dynamic tests, and a comprehensive neurologic examination. In particular, delineation of upper limb dermatomes and myotomes identifying involvement of specific PNs and spinal level involvement. Specific exam maneuvers that may help with assessment are listed in Table 1.

Diagnostic workup

Diagnostic evaluation after history and physical examination may begin with radiographs useful to evaluate for causes such as undiagnosed fractures and/or cervical spondyloarthropathies. At a minimum, views should include an anterior–posterior in internal rotation (AP–Int Rot), anterior–posterior in external rotation (AP–Ext Rot), and a true lateral/axillary view.[24] Other views include West Point, a modified axillary designed to enable review of the anterior/inferior glenoid rim,[24,25]as well as Stryker, an anterior-posterior with arm in 45° of external rotation and 90° of abduction. This view helps expose Hill-Sachs lesions associated with an instability event.[24,26] If the radiographs are unremarkable, consideration should be given to obtaining advanced imaging such as magnetic resonance imaging (MRI) which can help in the assessment of soft tissue and bony structures while not exposing the patient to radiation. MRI can be augmented with intra-articular contrast, known as magnetic resonance arthrography (MRA). The use of MRA may increase sensitivity for identifying articular cartilage lesions or labral defects.

Identifying the location of imaging is also important in formulating differential diagnoses. For example, an MRI of the medial elbow can aid in diagnosis by differentiating an ulnar collateral ligament injury from ulnar neuritis. This also applies in the situation of a type II Superior Labrum Anterior Posterior tear contributing to dead arm syndrome versus possible brachial plexopathy.[15] Clinical scenarios also exist when there is pathology from more than one source contributing to the patient's presenting symptoms. Suprascapular neuropathies present in throwing athletes may be a consequence of traction injury to the nerve.[27] Labral tears leading to paralabral cysts can also be problematic as they can cause compression of the suprascapular nerve at the spinoglenoid notch.[28] Similarly, an anatomical variant such as an anconeus epitrochlearis has been reported as a space-occupying lesion leading to compression of the ulnar nerve at the cubital tunnel.[29]

The use of ultrasound can provide diagnostic value when evaluating potential PN abnormalities.[30-35] Jacobson and colleagues noted that the hallmark of PN entrapment is hypoechoic enlargement of the nerve at and proximal to the area of entrapment.[35] Moreover, sonopalpation with an ultrasound transducer over the area of enlargement can provide the examiner valuable information on location of entrapment if the patient's symptoms are reproduced. Further, sonographic evaluation of the muscle involved may appear abnormal with increased echogenicity and subsequent atrophy if denervation has occurred.[35]

Along with imaging, the diagnostic workup for possible neuropathies should also include electrodiagnostic testing (as an extension of the physical exam) to include both nerve conduction studies (NCS) and electromyography (EMG).[36] NCS assess the integrity of sensory and motor nerves by stimulating a segment of a PN and can elicit information about the latency, action potentials, and conduction velocity. EMG involves the examiner inserting a needle into the muscle to investigate the electrical activity. EMG testing should be performed after approximately 3 weeks to allow for Wallerian degeneration to occur and therefore, abnormalities to be measurable if present on testing. Both tests can help provide key pieces of information to assess for any neuropathic or myopathic conditions at play that may contribute to the patient's symptoms. Electrodiagnostic testing may have further clinical utility to assess for any space occupying lesions or areas of abnormal physiological responses that may indicate PN compression. The utility of these tests can help differentiate a PN compression from brachial plexopathy and from radiculopathy. In addition, the severity of nerve injury (such as neurapraxia, axonotmesis, or neurotmesis) may lead to changes within the muscle that might be appreciated on MRI.[37] However, neuropraxic injuries with conduction block may not reveal any abnormalities on MRI, increasing the value of the EMG and NCS to localize and determine the extent of the injury.

TABLE 1 Upper limb peripheral nerve specific exam maneuvers[a22]

Physical exam maneuver	Peripheral nerve	Description of maneuver
Carpal compression	Median nerve	Examiner holds the patient's symptomatic hand in a manner where he/she places pressure on the anatomical course of the median nerve with their own thumb for period of 15 s up to 2 min. This is located just distal to the flexor retinaculum at the carpal tunnel. The exam is considered positive if the patient confirms reproduction of symptoms.
Phalen's test – wrist flexion	Median nerve	The patient is asked to fully flex both of their wrists by holding the dorsum of their hands together for at least 1 min. The maneuver is considered positive if there is reproduction of symptoms in the symptomatic hand.
Reverse Phalen's test – wrist extension	Median nerve	The patient is asked to fully extend both of their wrists by holding the palms together for at least 1 min in a prayer like pose. The maneuver is considered positive if there is reproduction of symptoms in the symptomatic hand.
Tinel's sign at the wrist	Median nerve	Patient is asked to extend the symptomatic wrist and the examiner will tap along the anatomical course of the median nerve, proximally to distally, from wrist crease to palmar area over lying the carpal tunnel.
Froment's sign	Ulnar nerve	The patient is requested to pinch a piece of paper between their thumb and index finger. The examiner will attempt to pull the paper from the patient while watching the patient's thumb and index finger. The test is considered positive if the patient flexes the thumb instead of keeping the interphalangeal joint straight, indicative of weakness of the adductor pollicis and possible ulnar nerve injury.
Tinel's sign at the elbow	Ulnar nerve	The examiner identifies the ulnar groove between the olecranon process and medial epicondyle of the affected limb. The examiner then taps within the groove to assess if patient's symptoms can be reproduced.
Spurling's neck compression test	Cervical radiculopathy	Examiner extends the patients neck and rotates the head while applying a downward pressure on the head. The test is noted to be positive when there is reproduction of pain into the ipsilateral limb that the head is turned towards.
Shoulder abduction relief test	Cervical radiculopathy	This test is performed when the examiner actively (or passively) abducts the patient's ipsilateral shoulder. If there is relief of the patient's cervical radicular symptoms in this ipsilateral limb, it is considered a positive test.
Adson's test	Thoracic outlet syndrome	The patient is asked to sit upright, with arms resting on their knees. The patient then takes a deep breath, extends the neck, and turns the head toward the symptomatic side. A disruption in the radial pulse or blood pressure in the ipsilateral arm is considered a positive test.
Roos test	Thoracic outlet syndrome	The patient is asked to raise both arms to an abducted, externally rotated position. The patient is then asked to open and close their fist at a rapid pace. The examiner then observes whether the patient indicates early fatigue or heaviness in the affected arm and/or increased neuropathic symptoms.
Scapular winging	Long thoracic nerve, spinal accessory nerve, or dorsal scapular nerve	The patient is seated with both scapulae observed. The patient is then asked to forward flex their arm followed by abduction, as well as push against a wall to observe the movement of the scapula. Prominence of the medial border of the scapula may indicate an injury to the long thoracic nerve. Likewise, a prominence of the lateral border of the scapula may suggest possible spinal accessory nerve injury versus dorsal scapular nerve.

[a]Adapted from Malanga et al.

General treatment considerations

Management of suspected PN abnormalities should be conducted in a systematic, stepwise manner. Conservative management is an appropriate starting point in the setting of suspected PN entrapment. Oral medication may be used as a short-term treatment for pain associated with nerve entrapment. Neuropathic agents have shown efficacy at low doses for nerve entrapment syndromes such as carpal tunnel syndrome.[38] However, side effects (such as lethargy and dizziness) when using these medications in student-athletes must be considered when determining appropriateness of use. At times, physicians may prescribe an oral nonsteroidal anti-inflammatory drug (NSAID), but a Cochrane review noted very low-quality evidence with no indication of any significant neuropathic pain and symptom reduction with NSAIDs.[39] Along with medication, the athlete should be encouraged to begin a targeted rehabilitation program. This can include massage, modalities such as electrical nerve and muscle stimulation, splinting, passive range of motion stretching, strengthening, sensory reeducation, and education on the importance of joint mobilization and protection.[40-42]

An adjunct may also include corticosteroid injection as a temporary nerve block to provide analgesic relief from potential entrapment. Eker and colleagues conducted a randomized, double-blind control trial in nonathletic injuries comparing 0.5% lidocaine to a group receiving 80 mg depo-methylprednisolone with 0.5% lidocaine for various PN blocks depending on dermatomal spread of pain and sensory symptoms that identified the injured PN.[43] At the 3-month mark, the group receiving methylprednisolone had superior improvement in numbness and burning pain compared to the lidocaine group. A systematic review by Bhatia et al.[44] evaluated the evidence of perineural steroids for chronic peripheral neuropathic pain of traumatic or compressive origin. Authors reached a similar conclusion that the injections may provide analgesic relief for 1 to 3 months, but evidence to support use of injections was weak. It should be noted that the number of participants in the randomized controlled trials used were small and important secondary measures of pain, such as anxiety, depression, and patient satisfaction, were not mentioned nor were adverse events of the interventions.

Some clinicians may opt not to use a corticosteroid but rather turn to an alternative injectate such as dextrose as part of a hydrodissection procedure. Nerve hydrodissection is a procedure in which an injectate is used to separate the nerve from entrapments, such as fascia, under ultrasound guidance.[45] More common injectates used for hydrodissection procedures can include normal saline, dextrose solutions, and corticosteroids.[45] The premise of the procedure is to release pressure on the nervi nervorum, which are smaller nerves that supply the main nerve, as well as the vasa nervorum (small blood vessels), thereby decreasing neuropathic symptoms and pain.[46] The authors go on to describe both in-plane and out-of-plane approaches with ultrasound guidance using 5% dextrose in water that has shown superior results compared to hydrodissection with normal saline or triamcinolone. Normal saline used for hydrodissection of the median nerve has provided an improved therapeutic effect compared to normal saline subcutaneous injection for carpal tunnel syndrome.[47] A randomized controlled trial compared perineural injection of normal saline, dextrose (D5W), and corticosteroid for carpal tunnel syndrome.[48] The authors noted that at the 6-month mark, the D5W group had the greatest reduction in pain and disability, most improvement on electrophysiological response measures, and largest decreased cross-sectional area of the nerve. A systematic review was conducted to evaluate the body of evidence using hydrodissection for carpal tunnel syndrome.[49] The authors identified six randomized controlled trials using various injectates for the procedure. Although the adverse effects of the procedure under ultrasound guidance were not significant, the lack of homogeneity in procedure-related parameters introduced bias and weak support. Applying findings from prior work,[46] hydrodissection of any PN may be appropriate to consider depending on technical expertise of the provider to help with pain relief and isolate location of nerve entrapment. However, the lack of high-level randomized controlled trials and low biased studies in the current literature lends limited support as a treatment for peripheral neuropathies in athletes.

If the athlete's neuropathy is recalcitrant to nonoperative measures, surgical intervention may be indicated. Surgical intervention can include decompression to release fascial bands or other anatomical structures that may be contributing to entrapment and possibly neuropraxia.[50-52] If an athlete has neuropathic symptoms from axonotmesis or neurotmesis, then direct nerve repair with a graft may be indicated.[53] If there is a laceration where there is clear transection of the nerve, surgery is indicated within 72 hours.[54] In the instance that there is a closed nerve injury but still likely in continuity, it is recommended that the athlete be followed by electrodiagnostic studies for 2 to 5 months.[55] If there are early signs of spontaneous recovery, nonoperative treatment can be continued.[54]

PERIPHERAL NEUROPATHIES IN THROWING ATHLETES

It is important to understand the functional movements required for a given athlete, as there may be clues as to what PN injury the athlete may have. Table 2 is provided as a summary for common signs and symptoms, etiology, and diagnosis of the respective PN injuries.

Suprascapular nerve

The suprascapular nerve (SSN) originates from the upper trunk of the brachial plexus with root supply from C5 to C6. It descends laterally along the trapezius and then down through the suprascapular and spinoglenoid notches. It terminates in the infraspinatus muscle. The nerve innervates the supraspinatus and infraspinatus and provides sensory articular branches to the capsule, glenohumeral joint, and acromioclavicular joint. The SSN does not provide cutaneous innervation.[56]

Suprascapular neuropathy presents as vague, deep posterior shoulder pain with possible weakness of shoulder abduction and external rotation, which can mimic rotator cuff pathology. There is often a component of scapular dyskinesia as well. In more advanced cases, there may be atrophy of the supraspinatus and/or infraspinatus. There may also be pain with cross-arm adduction. With injuries at the spinoglenoid notch distal to the branching of the sensory fibers, the athlete will present primarily with weakness and

TABLE 2 Summary of common signs and symptoms, etiology, and diagnosis of upper extremity PN injuries in throwing athletes

	Common signs and symptoms	Etiology	Diagnosis
Suprascapular nerve	• Vague, deep posterior shoulder pain • Shoulder abduction & ER weakness • Scapular dyskinesis	• Repetitive overhead motion in throwing athletes • Impaired scapular motion	• EMG/NCS • MRI & musculoskeletal ultrasound • Diagnostic ultrasound-guided injection
Axillary nerve & Quadrilateral Space Syndrome (QSS):	• Pain and possible decreased sensation over the lateral shoulder after a direct trauma • Arm fatigue ("dead arm") with overhead activity as well as vague, dull posterior shoulder pain that can radiate into the axilla • Weakness with shoulder flexion, abduction, or ER of the shoulder	• Sequela to anterior shoulder dislocation, humerus fracture, or direct blow to the shoulder • Overuse from in abduction and ER such as in pitching and volleyball serving	• EMG/NCS • MRI & musculoskeletal ultrasound • Diagnostic ultrasound-guided injection
Ulnar nerve	• Decreased velocity, accuracy, and durability • Medial elbow pain with or without sensory radiation into the ulnar hand • Possible loss of grip strength or clumsiness of the hand • Most commonly during the late cocking and early acceleration phases as maximum elbow torque occurs at 90–100° of elbow flexion • Snapping or popping in cases of ulnar nerve subluxation with elbow flexion	• Traction and compressive forces at the elbow in the setting of overuse • Excessive valgus forces and rapid elbow extension during throwing • Repetitive elbow valgus forces, compression at the cubital tunnel, or osteophyte formation in the ulnar groove with subsequent nerve compression	• EMG/NCS • Diagnostic ultrasound possibly with subluxation with dynamic imaging
Radial nerve (PIN)	• Lateral elbow and radial/dorsal forearm pain that is worsened with supination resistance with the elbow at 90°. • Tenderness distal and just anterior to the lateral epicondyle extending into the supinator musculature. • Weakness with finger extension and with radial wrist deviation upon extension • No sensory loss with PIN entrapment	• PIN due to overuse or entrapment at the supinator muscle due to repetitive supination and pronation • Entrapment sites include the fibrous bands from the radiocapitellar joint and the tendinous origin of the extensor carpi radialis brevis	• H&P • EMG/NCS, but can often be normal • Diagnostic ultrasound-guided injection
Neurogenic thoracic outlet syndrome	• Vague pain, numbness, and paresthesias of the throwing arm	• Compression or irritation of the brachial plexus as it traverses the scalene triangle at the base of the neck or in the pectoralis minor space below the clavicle	• H&P including provocative maneuvers (see Table 1) • Clinical as imaging and EMG/NCS studies have variable utility • MRI of the brachial plexus • Diagnostic ultrasound-guided injections at pectoralis minor and/or scalenes
Long thoracic nerve	• Vague shoulder pain and decreased endurance with overhead activities • Decreased overhead activity power or decreased throwing velocity • Medial scapular winging	• Overuse traction injury traction injury with the head rotated away from the throwing arm	• On PE, bringing the arm into the overhead position while rotating the head to the contralateral side • Medial scapular winging • EMG/NCS

(Continues)

TABLE 2 (Continued)

	Common signs and symptoms	Etiology	Diagnosis
Musculocutaneous nerve	• Combination of elbow/forearm pain, weakness with elbow flexion, and numbness/paresthesias over the volar forearm	• Overuse due to forceful pronation of an extended elbow • Overstretching of the nerve may occur at ball release through deceleration	• H&P • EMG/NCS
Median nerve (pronator syndrome)	• Vague volar proximal forearm and/or antecubital pain • Numbness in the median nerve distribution and/or weakness with forearm pronation, wrist flexion, or grip strength	• Entrapment at the elbow and proximal forearm caused by overuse involving elbow and forearm flexion during the late cocking and early acceleration phases of throwing • Compression at ligament of Struthers (supracondylar process), between the two heads of the pronator teres, at the lacertus fibrosus, or at the FDS arch	• H&P • Diagnostic ultrasound, diagnostic ultrasound-guided injection, and/or EMG/NCS

Abbreviations: EMG, electromyography; ER, external rotation; FDS, flexor digitorum superficialis; H&P, history and physical examination; MRI, magnetic resonance imaging; NCS, nerve conduction studies; PE, physical examination; PIN, posterior interosseous nerve; PN, peripheral nerve.

isolated atrophy of the infraspinatus without pain.[19] Overhead throwing athletes are more susceptible to SSN injuries at this location with isolated infraspinatus muscle atrophy noted in 4% of major league pitchers. Furthermore, isolated infraspinatus muscle atrophy was more prevalent in higher volume pitchers.[19,57-63]

The SSN is most often injured through repetitive overhead motion in throwing athletes with pain that will frequently be worsened during the cocking phase of the throwing motion.[56,64] Furthermore, faulty scapular motion with overhead activity can predispose to repetitive stretch and compression of the nerve.[64] Overall, the mechanism of injury can be structural compression from a paralabral cyst or ligament hypertrophy or repetitive traction. Throwing athletes can develop superior and posterior labral tears, which can lead to paralabral ganglion cyst development and possible eventual compression of the SSN.[19] Several case reports suggest that ultrasound-guided aspiration +/− corticosteroid injection of the paralabral cyst is an efficacious treatment option to relieve SSN compression and improve pain.[65,66]

Electrodiagnostic studies remain the most common means of confirming and localizing a SSN lesion. If the site of compression is the suprascapular notch, shoulder abduction and external rotation may be weak. Impairment at the spinoglenoid notch will only result in the infraspinatus being affected and result in weakness with external rotation.[67] Imaging such as MRI and musculoskeletal ultrasound can identify ganglion cysts and provide detail of muscle atrophy/local anatomy. However, imaging and electrodiagnostic studies are often normal.[68] Therefore, in the author's clinical experience, ultrasound-guided diagnostic SSN block is a very useful tool to quickly confirm the diagnosis.

Management initially involves rest from throwing followed by structured rehabilitation. Rehabilitation should address the overhead athlete's biomechanics and functional movement including scapulothoracic motion and stabilization. In refractory cases, surgery is warranted. The operation for SSN includes release of the transverse scapular ligament and less commonly release of the spinoglenoid ligament based on the site of compression. Surgery may be recommended earlier if there is a compressive lesion, such as a ganglion cyst. Regardless of whether operative intervention is pursued, rehabilitation should address biomechanics and functional movement to reduce the risk of an incomplete or poor recovery.[64,69]

Axillary nerve/quadrilateral space syndrome

The axillary nerve is the terminal branch of the posterior cord of the brachial plexus with fibers arising from C5 to C6. The nerve courses posteriorly under the axillary recess of the glenohumeral joint and exits with the posterior humeral circumflex artery through the quadrilateral space, which is bordered by the teres minor superiorly, teres major inferiorly, long head of the triceps medially, and surgical neck of the humerus laterally. The axillary nerve provides motor supply to the deltoid and teres minor and supplies sensation to the lateral aspect of the shoulder via the superior lateral brachial cutaneous nerve.[19,64]

Axillary nerve injuries are uncommon and typically occur as a sequela to anterior shoulder dislocation, humerus fracture, or direct blow to the shoulder.[70] Injury to the axillary nerve in throwing athletes may

occur because of direct trauma or quadrilateral space syndrome (QSS) as a result of overuse. Intermittent compression of the axillary nerve at the quadrilateral space occurs when the shoulder is in abduction and external rotation as in the late cocking phase of pitching where the quadrilateral space closes because of contraction of the teres major and minor.[19] QSS is most common in patients younger than 40 years old who participate in repetitive overhead activities.[71,72]

Typically, athletes with QSS present with pain, shoulder abduction weakness, and possible decreased sensation over the lateral shoulder after a direct trauma. However, this is uncommon in throwers.[56] Athletes with QSS will often deny trauma as an inciting event and will report arm fatigue ("dead arm") with overhead activity such as lifting as well as vague, dull posterior shoulder pain that can radiate into the axilla.[19,64,71,73,74] On clinical examination, there may be weakness with flexion, abduction, or external rotation of the shoulder. However, with chronic QSS, weakness is often subtle. In these cases, there may be tenderness over the quadrilateral space. Diagnostic ultrasound-guided injection is very useful in this scenario as tenderness and pain should improve.[19,52,71,75,76] Otherwise, from a diagnostic standpoint, EMG/NCS remains the study of choice, although it is less sensitive in evaluating for QSS because of significant dynamic and positional aspects.[52] MRI may be helpful in the evaluation of QSS through detection of ganglion cysts and/or mass lesions compressing the nerve and muscular denervation changes.[77] Ultrasound-guided injection of local anesthetic into the quadrilateral space performed by an experienced physician can be a useful diagnostic modality as data have indicated that this procedure improves tenderness to palpation at site of injury and decreases pain with active throwing.[52,75,76] Although there is no literature on a true gold standard for the diagnosis of QSS, a lidocaine block test seems to be the best current diagnostic tool.[78]

Nonoperative management is the initial treatment for axillary nerve injuries and QSS. This includes rest from throwing and physical therapy. Physical therapy for the throwing athlete with this injury typically involves range of motion with posterior capsule stretching, scapular stabilization, posterior rotator cuff strengthening, and an interval throwing program.[71,79] Throwing mechanics are also addressed in the rehabilitation period. An ultrasound-guided injection of anti-inflammatory medication into the quadrilateral space can also be employed for pain relief although literature on this topic is limited.[75,76,80] Only case reports and series have been published as randomized controlled trials have not been performed at this time. Indications for surgery include space occupying lesions and cases with a positive diagnostic injection that are refractory to 3–6 months of nonoperative management.[78] Literature on surgical outcomes is limited because most of the data are derived from smaller case series. Nonetheless, decompression of the quadrilateral space after failed nonsurgical treatment is noted to be successful with complete or near complete resolution of symptoms in most cases. Furthermore, very few complications have been reported after surgical treatment for QSS.[19,52,69,78,81,82]

Ulnar nerve

Ulnar neuropathy is the most common peripheral mononeuropathy in throwing athletes given the degree of ulnar nerve mobility and the extreme biomechanics associated with throwing.[19,56,83-86] The ulnar nerve is derived from the C8-T1 roots, which then form the medial cord of the brachial plexus. The ulnar nerve descends down the upper arm in the anterior compartment and then transverses posteriorly at the middle third of the upper arm. The ulnar nerve then passes through the arcade of Struthers approximately 6–8 cm proximal to the medial epicondyle and subsequently passes posterior the medial epicondyle and enters the cubital tunnel. The borders of the cubital tunnel are the posterior ulnar collateral ligament and capsule laterally, the medial epicondylar groove anteriorly, and the arcuate ligament medially. The nerve then descends between the two heads of the flexor carpi ulnaris under a fascial band known as Osborne's ligament and ultimately into the distal aspect of the forearm, wrist, and hand. At the wrist, the nerve enters the hand via Guyon's canal and terminally divides into superficial and deep branches. The motor innervation from the ulnar nerve includes the majority of the intrinsic muscles of the hand including the palmaris brevis, abductor digiti minimi, flexor digiti minimi, opponens digiti minimi, third and fourth lumbricals, palmar interossei, dorsal interossei, and adductor pollicis; the flexor carpi ulnaris; and the medial aspect of the flexor digitorum profundus. The sensory innervations are the dorsal and palmar aspects of the fourth and fifth digits and the ulnar aspect of the hand.

Throwing athletes are particularly susceptible to ulnar neuropathy because of traction and compressive forces at the elbow during the late cocking and acceleration phases of throwing in the setting of overuse.[87] The combination of excessive valgus forces and rapid elbow extension with throwing places high tensile stress along the medial side of the elbow and the ulnar nerve is one of the structures that can be injured because of these forces.[19] Ulnar nerve dysfunction with throwing is caused by significant repetitive elbow valgus forces, compression at the cubital tunnel due to repetitive stress, or osteophyte formation in the ulnar groove with subsequent nerve compression.[11,88] Other potential factors affecting the ulnar nerve in throwers are loose bodies, traction spurs, hypertrophy of the

triceps and anconeus epitrochlearis muscles, synovitis, thickened retinaculum, scar tissue, and calcification of the ulnar collateral ligament.[19] Flexor carpi ulnaris contraction with the elbow flexed in the throwing motion can increase pressure in the cubital tunnel from 6 to 20 times baseline, which can further lead to ulnar nerve compression.[89,90] Furthermore, it has been reported that the ulnar nerve elongates 4.7 mm with elbow flexion, which can also stress the nerve during the throwing motion.[91,92]

The athlete's presenting symptoms with ulnar neuropathy can be decreased performance including decreased velocity, accuracy, and durability rather than the typical nerve symptoms such as pain and paresthesias.[86] Once neurological symptoms do develop, the thrower will report medial elbow pain with or without sensory radiation into the hand. The athlete may also report numbness/tingling in the fourth and or fifth fingers with possible loss of grip strength/clumsiness of the hand. These symptoms initially occur with throwing alone most commonly during the late cocking and early acceleration phases as maximum elbow torque occurs at 90–100° of elbow flexion.[86] As the nerve injury progresses, symptoms may also develop at rest. A feeling of snapping or popping may be present in cases of ulnar nerve subluxation with elbow flexion. The clinical diagnosis is often complicated by the presence of concomitant musculoskeletal pain in the elbow. For example, 42% of throwing athletes with ulnar collateral ligament injuries also have ulnar neuritis and 53% of throwers with medial epicondylitis have concomitant ulnar neuritis.[93] However, ulnar nerve symptoms in throwing athletes can occur with or without other musculoskeletal injury.

The diagnosis of ulnar neuritis in the overhead throwing athlete is often based on history and physical examination.[94] Physical exam of the ulnar nerve should include palpation along the course of the nerve at the medial elbow from just proximal to the medial epicondyle down to the flexor carpi ulnaris musculature in the forearm. Tinel's sign can be elicited along the length of the nerve as well. The elbow should be ranged into flexion and extension to determine if there are any signs of nerve subluxation. Typically, the unstable ulnar nerve will sublux or dislocate anterior to the medial epicondyle while the elbow is moved from extension into flexion. It should be noted if subluxation reproduces symptoms as asymptomatic subluxation has been reported in 16% of individuals.[95] Diagnostic ultrasound is also very helpful to identify nerve displacement on dynamic imaging with elbow flexion. More recently, Kawabata and colleagues found ultrasound evidence of dynamic ulnar nerve displacement in one third of asymptomatic youth baseball players examined.[96] Diagnostic ultrasound is also helpful in identifying morphological nerve abnormalities, such as increases in nerve cross sectional area.[30] MRI is not routinely used for the evaluation of ulnar neuropathy. However, because ulnar neuropathy often occurs with other medial elbow pathology, such as ulnar collateral ligament injury, MRI with or without contrast may be used to evaluate the ulnar collateral ligament or other soft tissue lesions that can cause nerve compression.[85] Electrodiagnostic studies may also be helpful in the diagnosis of ulnar neuropathy; however, abnormal changes may not be found until more advanced stages of pathology. For example, one study did not find any difference in ulnar nerve conduction velocities across the elbow when comparing dominant versus nondominant arms of injured baseball pitchers.[97] Therefore, a normal electrodiagnostic study does not rule out the presence of ulnar neuropathy.

Initial treatment of ulnar neuropathy in the overhead throwing athlete should typically be nonsurgical. This involves rest from throwing, avoidance of other aggravating activities, oral anti-inflammatory medications, ultrasound-guided perineural injection, and physical therapy. If symptoms occur at night or are more severe, nighttime splinting can also be beneficial.[98] This can be particularly helpful in athletes with symptomatic nerve subluxation.[99] The athlete should refrain from throwing until they are asymptomatic. Physical therapy should focus on strengthening the dynamic stabilizers of the elbow and stretching the posterior capsule. Finally, an interval throwing program should be initiated with a focus on throwing mechanics.[64,85,100] An extended period of nonoperative management of 3–6 months is generally recommended before surgery is considered. However, it is common that ulnar nerve symptoms recur once throwing is resumed.[85]

Indications for surgery in the throwing athlete with ulnar neuropathy include failure of nonoperative management, persistent ulnar nerve instability, or concomitant elbow pathology, such as ulnar collateral ligament injury, requiring surgical intervention. Earlier operative management is also considered for athletes with more severe nerve compression/pathology including axonal loss, profound sensory deficits, and/or motor weakness.[19] Surgical treatment options include simple in situ decompression with neurolysis or surgical decompression with subcutaneous or submuscular transposition.[85] However, simple in situ decompression of the ulnar nerve is generally not sufficient for throwers given traction forces placed on the nerve with overhead throwing, which can lead to increased failure rates. Two potential complications after in situ decompression that occur more frequently in young, active males are ulnar nerve instability/subluxation and ulnar neuritis due to adhesion formation.[101-106] Even in nonthrowers, one report identified that in situ decompression required revision surgery in 19% of patients.[107] Given these issues with simple in situ decompression, decompression with transposition has become the preferred surgery for throwing athletes.[85] Regarding transposition, both

subcutaneous and submuscular transposition have demonstrated favorable outcomes in the overhead throwing athlete.[108-112] However, subcutaneous transposition has become preferred recently because of decreased rates of continued postoperative ulnar nerve symptoms.[108,113,114] Moreover, there is concern in performing submuscular transposition in throwers given potential damage to the flexor-pronator mass, which can significantly affect throwing performance.[85,86] Nicholson and colleagues reported that in 26 overhead athletes who underwent anterior/subcutaneous ulnar nerve transposition, 92% returned to their sporting activity along with a low rate (4%) of symptomatic recurrence of preoperative ulnar nerve symptoms.[115] Furthermore, one study found that in 21 subcutaneous transpositions in athletes, Rettig and Ebben reported excellent outcomes and return to sports occurred on average at 12.6 weeks.[112] Conversely, a separate investigation reported a 31% incidence of postoperative ulnar nerve dysfunction after ulnar collateral ligament reconstruction with submuscular transposition of the ulnar nerve.[113] Although decompression with subcutaneous transposition seems to be the current preferred surgical technique in throwing athletes, there are no prospective randomized controlled trials or meta-analyses available to guide treatment.

Radial nerve

The radial nerve is derived from the C5-C8 roots and the posterior cord of the brachial plexus. The nerve descends in the posterior compartment of the upper arm and then turns around the humerus at the spiral groove between the medial and long heads of the triceps. During late acceleration and follow-through in the thrower, the triceps brachii is contracting, and the radial nerve may be compressed.[86] The nerve then moves to the forearm where it splits into the posterior interosseous nerve (PIN) and superficial radial nerve just distal to the elbow. The PIN moves through the supinator muscle through the arcade of Frohse, a potential site of entrapment.

The radial nerve innervates the triceps, anconeus, brachioradialis, extensor carpi radialis longus, and supinator muscles before it splits. In the forearm, the PIN innervates the extensor carpi radialis brevis, extensor carpi ulnaris, abductor pollicis longus, and the finger extensors. The radial nerve provides sensory cutaneous innervation to the dorsolateral hand including the first three and a half digits.

Radial nerve injury proximal to the elbow is rare in throwing athletes. More commonly, the PIN is susceptible to repetitive overuse and entrapment at the supinator muscle because of repetitive supination and pronation. Other entrapment sites include the fibrous bands from the radiocapitellar joint and the tendinous origin of the extensor carpi radialis brevis.[11,64,88] Near ball release and into follow-through, the forearm pronates and the supinator is eccentrically contracting, with potential PIN compression.[86] Athletes with PIN entrapment will generally present with lateral elbow and radial/dorsal forearm pain that is worsened with supination resistance with the elbow at 90°. There is usually tenderness to palpation distal and just anterior to the lateral epicondyle extending into the supinator musculature. There may also be weakness with finger extension and with radial wrist deviation upon extension. It is important to remember that there is no sensory loss with PIN entrapment. Electrodiagnostic testing may be helpful in confirming the diagnosis; however, this is not necessary as EMG/NCS can often be normal.

Management is typically nonsurgical. Treatment includes rest and physical therapy focusing on mechanics and the kinetic chain. Physical therapy should avoid wrist extensor and supinator strengthening to lessen PIN irritation.[86] Furthermore, corticosteroid injection in the area of the PIN may be helpful, though data are limited. One report identified that 40 patients with radial tunnel syndrome experienced significant improvements in pain and function at 12 weeks and 1 year after a single corticosteroid injection to the proximal forearm at the point of maximum tenderness.[116] Furthermore, Sarhadi et al. reported long-term relief of pain in 16 of 25 patients after a single localized corticosteroid injection.[117] Theoretically, and in the authors' clinical experience, ultrasound-guided PIN perineural injection/hydrodissection is also a reasonable nonsurgical treatment option, although there is no published data in the literature specifically addressing ultrasound-guided hydrodissection of the PIN.[118] As such, ultrasound-guided radial nerve block may be helpful to be both diagnostic and therapeutic in the treatment of radial tunnel syndrome.[119] For athletes who do not respond to conservative treatment, surgical decompression may be indicated.[64]

Neurogenic thoracic outlet syndrome

Thoracic outlet syndrome (TOS) is a relatively uncommon cause of shoulder pain characterized by pain, numbness, and paresthesias in the shoulder and arm, especially with activities involving raising the arms above the head, such as throwing. These symptoms manifest as a result of the compression of neurovascular structures as they pass through the thoracic outlet, the space between the clavicle and first rib.[120] The most common cause of TOS is neurogenic thoracic outlet syndrome (nTOS) due to compression or irritation of the brachial plexus as it traverses the scalene triangle at the base of the neck or in the pectoralis minor space below the clavicle.[121] Though nTOS is a relatively uncommon cause of shoulder pain and injury, it should always be considered in throwing and

overhead athletes presenting with pain, numbness, and paresthesias of the upper extremity.

The biomechanics of throwing is a central consideration in discussing nTOS in throwing athletes. Previous studies in healthy adults have shown that maximal shoulder abduction and external rotation, such as in the late cocking and early acceleration phases of throwing, significantly reduce the space between both the clavicle and scalene and clavicle and first rib, key structures associated with compression and nTOS.[122] Immediately distal, the neurovascular bundle must traverse the posterior aspect of the pectoralis minor muscle, or retropectoralis minor space. During upper limb elevation the cords may be pressed tightly against the pectoralis minor musculature and predispose to compression.[123] Biomechanical studies have shown that throwing athletes tend to have a throwing arc that is shifted further toward external rotation compared to their nonthrowing arms and the general population, increasing their predisposition for compression of the thoracic outlet.[124] Additionally, the number of the overhead repetitions required for high-level play further increases the risk for symptomatic nTOS.[125] These repetitive motions in the throwing athlete can result in chronic inflammation, muscle spasms of the affected musculature, and even fibrosis that exacerbates compression of the neurovascular bundle.[126]

Prevalence studies of nTOS in throwing athletes compared with the general population support the anatomical and biomechanical risk factors that throwers are at increased risk for nTOS. One cross-sectional study of 1288 Japanese high school baseball players demonstrated a 32.8% prevalence of TOS, of which the majority was hypothesized to be nTOS.[127] This figure is three times higher than the prevalence found in industrial workers.[128] A study analyzing U.S. collegiate athlete injury data found that baseball and softball were the sports most commonly associated with nTOS, followed by swimming and water polo.[125]

The physical exam to identify nTOS should begin with a thorough inspection of the shoulder girdle and upper extremities for gross deformity or asymmetry. Strength and sensation of the affected and contralateral extremity should be examined to identify asymmetry with particular attention to musculature innervated by the upper versus lower brachial plexus. Next, palpation and provocative maneuvers should be performed over pertinent structures to measure reproducible pain and paresthesia. A key finding is the ability to provoke symptoms with direct pressure placed over the brachial plexus at the areas of nerve compression including the interscalene triangle or below the pectoralis minor or with overhead arm use.[129-131] However, although positive provocative maneuvers are supportive of a diagnosis of nTOS, there is a high false-positive rate.[132-134]

Currently, there is no gold standard for diagnosis of nTOS and it largely remains a clinical diagnosis of exclusion as imaging and electrodiagnostic studies have variable utility. Imaging starts with ruling out other causes of nerve entrapment, such as cervical pathology that can present with similar symptoms. MRI of the brachial plexus remains ideal in the visualization of neural structures that may contribute to symptoms as it provides superior localization of compression suspected on physical examination. The evidence for electrodiagnostic studies in the diagnosis of nTOS remains controversial. The most recent TOS guidelines from the Society for Vascular Surgery in 2018 do not recommend NCSs as they can often be normal in those without prolonged symptoms.[130] Diagnostic ultrasound may also aid in diagnosis of nTOS by visualizing key structures. One proposed technique suggests dynamic evaluation with the arm abducted to measure the "pectoral bowing ratio" measured as a percentage of the vertical deformation of the pectoralis minor muscle compared to its length.[32] An important limitation of this technique is that formal training is not readily available for this advanced ultrasound evaluation. Lastly, performing ultrasound-guided diagnostic injections at common sites of compression such as the anterior scalene or pectoralis minor is becoming a more common in the workup for nTOS. Alleviation of symptoms with these injections would suggest a diagnosis of nTOS.[135-139]

Treatment for nTOS begins with rest from throwing and physical therapy. Physical therapy should focus on pectoralis minor and scalene stretching as well as scapular retraining and scapulohumeral motion. Care should be taken with strengthening exercises, especially those involving the pectoralis minor and scalenes, as these can often exacerbate symptoms.[126,134,140] Therapeutic ultrasound-guided injections with anesthetic agents and botulinum toxin are also a treatment option, although they are not demonstrated to provide long term symptomatic improvement.[141-144] There are not any trials published evaluating the therapeutic utility of corticosteroid injections into the pectoralis minor or scalenes for the treatment of nTOS. Botulinum toxin injections have come into question because a randomized controlled trial of botulinum toxin injection to the anterior and middle scalenes did not demonstrate benefit compared to placebo.[143] However, this study has significant limitations. Most notably, these patients did not have a confirmed positive response to a diagnostic lidocaine block into the scalenes prior to the botulinum toxin treatment. Therefore, it remains to be seen whether the nTOS symptoms were secondary to compression at the scalenes versus the pectoralis minor. Furthermore, a number of other trials, although not randomized or controlled, have demonstrated improvement after botulinum toxin injections into the scalenes and pectoralis minor in patients with nTOS.[142,145-150] There is scant evidence in the literature regarding ultrasound-guided brachial plexus hydrodissection for the treatment of nTOS. One report described multiple

hydrodissection techniques and reported symptomatic improvement at 2 months after ultrasound-guided brachial plexus hydrodissection with 5% dextrose in water in patients with neuropathic upper extremity pain, some with nTOS.[151] For those athletes who do not improve after 3–6 months of focused conservative management, surgery is often required for symptomatic resolution. Surgical treatment of nTOS entails decompression of the thoracic outlet by some combination of scalenectomy and/or pectoralis minor release, brachial plexus neurolysis, and first rib resection.[152] Outcomes after surgical management of nTOS in throwing athletes tend to be successful. A recent outcome report for professional baseball players with nTOS followed key performance metrics in 10 Major League Baseball (MLB) pitchers and showed essentially no decrease in performance following decompression and rehab for nTOS. Metrics measures included maximal and average pitch velocity, earned run average, and strikeouts-to-walks ratio.[153] A similar, but larger study of MLB pitchers over 2001–2017 showed that 20 pitchers with nTOS underwent surgical decompression. Of those, fast ball velocity and strike percentage were equivalent post-operatively, but pitcher earned run average remained inferior to preoperative baseline.[154]

Long thoracic nerve

Long thoracic nerve injury is uncommon in throwing athletes; however, it should be considered as a potential source of pain. The nerve is derived from C5 to C7 and travels over a long course beneath the scapula to innervate the serratus anterior. The etiology of long thoracic nerve injury in throwing athletes is generally an overuse traction injury with the head tilted or rotated away from the throwing arm as it is overhead. In this position, the long thoracic nerve can undergo significant traction/stretch.[155] These athletes typically present with vague shoulder pain and decreased endurance with overhead activities. They will also report decreased overhead activity power or decreased throwing velocity. Symptoms may be recreated by bringing the arm into the overhead position while rotating the head to the contralateral side. Furthermore, the athlete will likely have medial scapular winging due to lack of full enervation of the serratus anterior. From a diagnostic standpoint, imaging studies are generally unhelpful outside of demonstrating bony abnormalities of masses that may compress the nerve.[156] Electrodiagnostic studies are more helpful in confirming the diagnosis and in determining the severity.

Overuse long thoracic nerve injuries in throwing athletes typically spontaneously resolve with nonsurgical management in 6–18 months.[71] A shorter recovery time course of 6 to 9 months has been reported in athletes.[157,158] Nonoperative management will consist of rest from throwing and physical therapy. Physical therapy will initially focus on maintaining shoulder range of motion in the throwing arm as well as working on strengthening of compensatory musculature. Scapular bracing to improve scapular position and help with pain and range of motion is a treatment option as well although data regarding efficacy is conflicting.[159] Early surgery is not indicated unless there is penetrating trauma. If symptoms persist for 1–2 years despite conservative management, then these athletes may benefit from surgical intervention.[160] However, the most common surgeries performed in this situation come with significant restrictions, which limit return to high-level sport and overhead activity.[158,161-165]

Musculocutaneous nerve

Musculocutaneous nerve injury is relatively uncommon in throwing athletes, but this form of neuropathy has been documented in the literature.[166-169] This nerve contains fibers from the C5-C6 roots, arises from the lateral cord of the brachial plexus, passes underneath the coracoid and moves distally medial to lateral along the anterior aspect of the arm between the biceps and brachialis. The terminal portion of the nerve becomes the lateral antebrachial cutaneous nerve. Motor innervation includes the biceps, brachialis, and coracobrachialis. Sensory innervation includes the radial/volar aspect of the forearm via the lateral antebrachial cutaneous nerve of the forearm.

The nerve is more commonly injured in the distal arm owing to overuse that includes forceful pronation of an extended elbow, which is a motion that occurs with throwing. More specifically, overstretching of the nerve may occur at ball release through deceleration.[170,171] The presenting symptoms include some combination of elbow/forearm pain, weakness with elbow flexion, and numbness and paresthesias over the volar forearm. Imaging studies are most likely to be normal with EMG/NCS being more helpful for definitive diagnosis. Treatment is generally nonsurgical including rest from throwing and physical therapy. Physical therapy should focus on sports specific mechanics including scapular mechanics and stabilization and the kinetic chain with attention turned to deriving power from the kinetic chain as opposed to an overreliance on power generation from the throwing arm.[64,166,172] Initially, biceps strengthening exercises should be avoided to minimize further irritation of the nerve. Rest from throwing should continue until the restoration of elbow flexion strength, decreased neural tension symptoms, and improved sensation in the lateral antebrachial cutaneous nerve distribution.[166] Of note, a specific rehabilitation and return to throwing program in a collegiate baseball player with musculocutaneous nerve injury has been described.[166] Reported return to play for

musculocutaneous nerve injury in throwing athletes varies between 2 and 6 months.[167-169]

Median nerve

Median nerve injury is another less common peripheral neuropathy in throwing athletes. The median nerve is formed from both the medial and lateral cords of the brachial plexus and is supplied by the C5-T1 nerve roots. The nerve travels along the medial aspect of the upper arm and passes between the two heads of the pronator teres as it enters into the forearm. More distally, the median nerve branches into the anterior interosseous nerve, which innervates the flexor pollicis longus, pronator quadratus, and flexor digitorum profundus. The main branch of the median nerve continues distally through the carpal tunnel, formed by the carpal bones and the transverse carpal ligament, and terminates in the hand. The median nerve innervates all flexors of the forearm except the flexor carpi ulnaris and the flexor digitorum profundus to the fourth and fifth digits. In the hand, the median nerve innervates the first and second lumbricals, opponens pollicis, abductor pollicis brevis, and flexor pollicis brevis. Sensory innervation is provided to the lateral palmar aspect of the hand and to the first three and a half digits.

The median nerve can be compressed at multiple locations. Carpal tunnel syndrome (aka median neuropathy at the wrist) is the most common overall peripheral neuropathy in the upper extremity. It is common in both athletes and nonathletes; however, it is less commonly attributed to the results of throwing in overhead athletes.[11,64] Sports with greater incidence of carpal tunnel syndrome caused by their sport are cycling and wheelchair sports where direct pressure is placed over the palmar aspect of the hand. These athletes often present with pain at the wrist and numbness, tingling, and pain over the palmar aspect of the first three and a half digits. Diagnosis can be made clinically, with the assistance of electrodiagnostic studies, or diagnostic ultrasound measuring nerve cross-sectional area.[31] Treatment includes splinting, local corticosteroid injection, and occupational therapy. Carpal tunnel release surgery may be necessary for more refractory cases.[64]

Pronator syndrome is much less common than carpal tunnel syndrome overall but is more likely to occur as a direct cause of throwing overuse. Pronator syndrome is synonymous with median nerve entrapment at the elbow/proximal forearm caused by overuse involving elbow and forearm flexion, repeated pronation, and gripping.[92,173] The entrapment can occur at the ligament of Struthers (supracondylar process), between the two heads of the pronator teres, at the lacertus fibrosus, or at the flexor digitorum superficialis arch. Athletes will present with vague volar proximal forearm and/or antecubital pain. They may also have numbness in the median nerve distribution or weakness with forearm pronation, wrist flexion, or grip strength. Just as with the ulnar nerve, the median nerve is at risk of compression at the elbow and/or forearm during the late cocking and early acceleration phases of throwing. This can most likely occur due to compression under the ligament of Struthers (supracondylar process) or the lacertus fibrosus as they can compress the median nerve secondary to biceps contraction during the early acceleration phase.[86] Diagnosis is often clinical as electrodiagnostic studies are frequently normal.[174] Clinical tests include provocation on examination with resisted pronation of the extended forearm (pronator teres entrapment), with resisted elbow flexion and forearm supination (lacertus fibrosus), and with resisted flexion of the third proximal interphalangeal joint (flexor digitorum superficialis arch).[64] Diagnostic ultrasound can also be helpful in localizing the site of compression with or without a diagnostic injection.[175,176] Treatment most frequently involves rest and physical therapy focusing on appropriate throwing mechanics and kinetic chain optimization. Ultrasound-guided injections can also help with pain relief.[176] Surgical decompression is warranted in refractory cases.[64,92] However, there are no randomized controlled trials indicating treatment outcomes in pronator syndrome or to guide the duration of conservative management before pursuing surgical intervention.[92]

CONCLUSION

Peripheral neuropathies are common in throwing athletes and may present differently than in other patients. Unrecognized PN injuries can result in worse outcomes and lead to significant impairment in these athletes' ability to perform at their optimal levels.

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