THROWING ATHLETES AND THE ROLE OF NERVES IN SHOULDER PAIN

– Written by Frantzeska Zampeli, Greece, Theodorakys Marin Fermin, Venezuela, and Elisabet Hagert, Qatar

INTRODUCTION

Shoulder pain and dysfunction of the throwing athlete may be attributed to variable conditions, including glenohumeral pathology (capsulolabral or cartilage injuries), rotator cuff pathology, scapular dyskinesis, or pathology of neurovascular structures. When examining a thrower for shoulder pain, nerve pain etiologies, although rare, may cause profound morbidity and should be considered. Causes of nerve disorders include (but is not limited to): thoracic outlet syndrome, axillary-, suprascapular,- and long thoracic compression neuropathies. Differentiating between various types of nerve compressions and nerve-related conditions can be complex, but knowledge of their differences can facilitate the proper diagnosis and treatment. They often have similar clinical presentation such as fatigue, vague shoulder pain, decreased velocity or manual dexterity, weakness of grip, numbness and tingling, sense of heaviness, achiness, or cramping in the upper extremity.

The throwing motion is one of the fastest and most demanding maneuvers to which any joint in the body is subjected and facilitates the synchronization of the kinetic chain. Baseball pitchers are a classic paradigm of throwers, although similar throwing motions (with technical modifications depending on the specific sports demands and characteristics) are required in several sports such as handball, volleyball, tennis, water polo, and track and field throws (discus, shot put, hammer, and javelin). For each throw or pitch, the thrower must generate high levels of energy in the lower extremities and trunk allowing adequate production and efficient transfer of energy to accelerate the ball (or throwing object) to top velocity upon release. In elite pitchers, internal rotation of the humerus can reach velocities as great as 7000 deg/s. This coordinated function of the kinetic chain is essential to reduce the need for the shoulder to generate large forces. However, the structures of the shoulder still have to keep a delicate equilibrium ("throwers paradox") between necessary laxity to achieve an extreme range of motion and sufficient stability to inhibit subluxation and instability in order to maintain and maximize the force that can be generated and transferred to the ball. Finally, the muscle-tendinous and capsule-labral structures of the shoulder must absorb this force after release of the ball and during arm deceleration[1-5].

A brief review of the causes of neurological pain in the shoulder of the throwing athlete is presented in this article.

QUADRILATERAL SPACE SYNDROME

Anatomy

Quadrilateral space syndrome (QSS) is an infrequent condition that affects young active adults and was originally reported since 1983[6]. It results from compression of the axillary nerve or the posterior circumflex humeral artery (PCHA) within the quadrilateral space (QS). The quadrilateral space (occasionally mentioned as quadrangular space in the literature) is bounded by the teres minor superiorly, the teres major inferiorly, the long head of triceps medially, and the surgical neck of the humerus laterally[5]. The axillary nerve and PCHA transverse the QS. The axillary nerve innervates the deltoid and teres minor muscles, which are responsible for the abduction and external rotation (at 90 abduction) of the arm, respectively. The PCHA provides the main perfusion of the humeral head. (Figure 1)

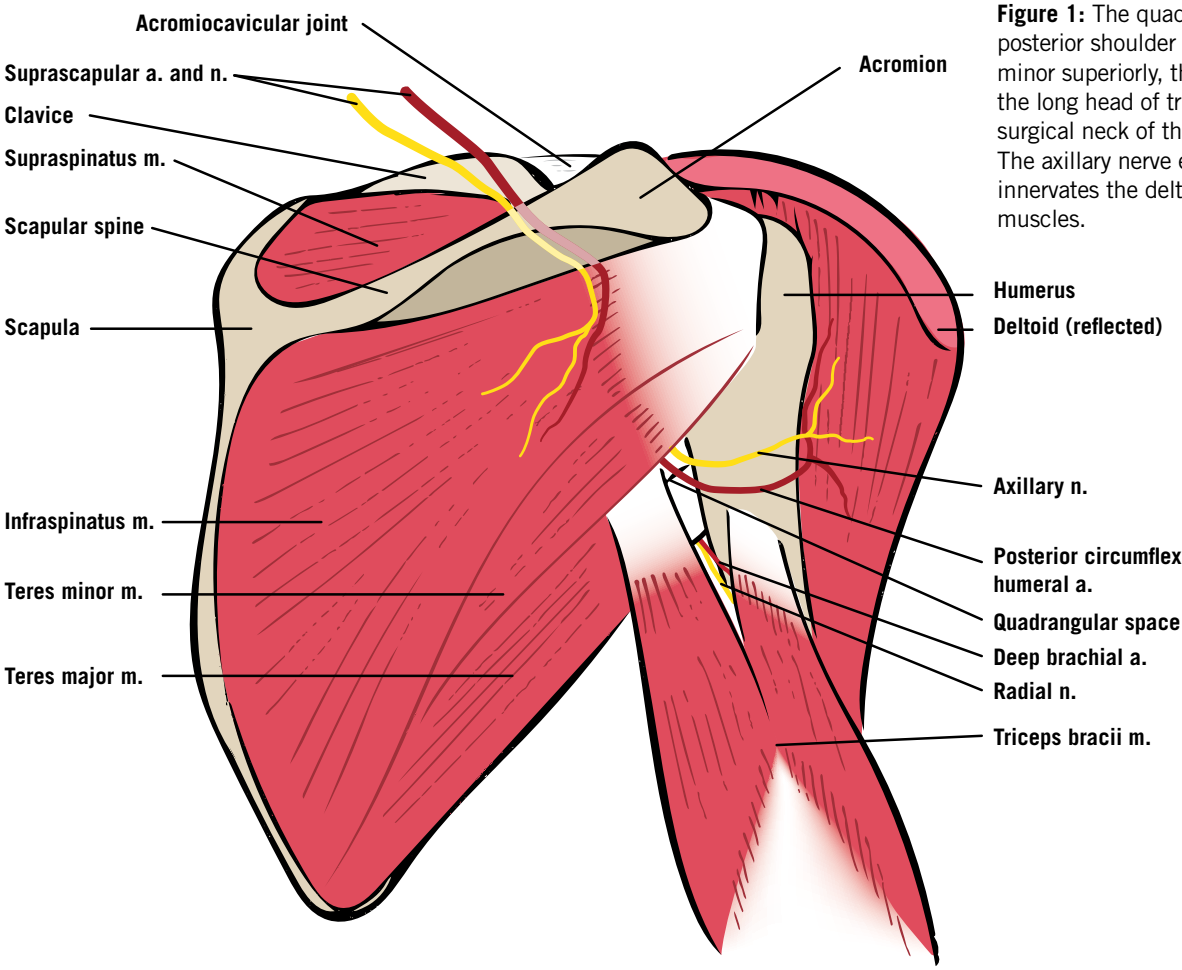

Figure 1: The quadrangular space of the posterior shoulder is bounded by the teres minor superiorly, the teres major inferiorly, the long head of triceps medially, and the surgical neck of the humerus laterally. The axillary nerve exists the QS to The innervates the deltoid and teres minor muscles.

Etiology

Variable etiology of QSS has been reported, but most commonly it is attributed to repeated overhead activity in throwers and overhead athletes (i.e. swimmers). In such cases fibrous bands or muscle hypertrophy is usually the specific pathoanatomy relating to the compression. However, other rare causes have also been reported including lipoma, hematoma, osteochondroma, axillary nerve sheath tumor, and labral cysts that may cause compression of the neurovascular bundle in the quadrilateral space. More rarely, anatomical variations such as an abnormal origin of the radial collateral artery from PCHA or an accessory subscapularis muscle can predispose throwers to QSS[1,7]. The Mayo group has suggested a more strict differentiation of QSS cases with respect to the pathogenesis where fixed structural impaction of the QS with space-occupying lesions or fibrous bands leads to neurogenic QSS (nQSS) presenting with non-dermatomal paresthesias and QS point tenderness. On the other hand, repetitive mechanical injury to the PCHA as it passes through the tight QS and wraps around the humeral neck during abduction and external rotation leads to a vascular type of QSS with PCHA thrombosis and/or aneurysm with distal embolization and digital ischemia[7].

Diagnosis

The relatively low frequency of the condition, combined with the nonspecific, clinical presentation of athletes with quadrilateral space syndrome make diagnosis challenging and require a high index of suspicion[7,8]. Although both features from nerve or vascular compression may be present, symptoms initially result from compression of the axillary nerve, not from PCHA thrombosis.

Symptoms

The thrower or overhead athlete with QSS may complain of intermittent, poorly localized anterior, lateral, or most often posterior (over QS) shoulder pain or tenderness on palpation, weakness and distally radiating non-dermatomal paresthesias to the arm or the forearm, aggravated by forward flexion, abduction, and external rotation of the arm. Secondarily, repeated abduction and external rotation may lead to injury of the PCHA within the QS, resulting in thrombosis and aneurysm formation, with distal emboli[1,6,7].

Clinical examination

Upon inspection, atrophy of the deltoid and teres minor can be noticed due to denervation. Fasciculation of the long head of the triceps has also been reported. The diagnosis is usually made based on the presence of Hagert's clinical triad of nerve compression diagnosis: (1) tenderness over the quadrilateral space (usually discrete point tenderness), (2) possible paresthesia over the lateral shoulder and upper posterior arm with positive scratch-collapse test at the QSS, and (3) deltoid weakness with decreased strength in shoulder abduction[1,5,6,9,10].

Differential diagnosis

Differential diagnoses include other sources of shoulder pain in the thrower such as rotator cuff and labral pathology, C5-C6 radiculopathy, thoracic outlet syndrome, brachial plexus neuritis, and suprascapular nerve injury. Conditions with axillary nerve injury other than compression at the QS

should be excluded such as blunt trauma, anterior shoulder dislocation, or proximal humerus fracture[11]. Although extremely rare, an uncommon compression site of the axillary nerve between the proximal humerus and the latissimus dorsi tendon has been described to appear with the same presentation of shoulder pain and upper limb numbness when throwing as in QSS[12].

Imaging

Upon clinical suspicion, an imaging workup can assist in diagnosis both by ruling out other diagnoses and confirming QSS. Although no "gold standard" diagnostic test for QSS exists, magnetic resonance imaging (MRI) is usually the first choice. MRI often demonstrates focal fatty infiltration and atrophy of the teres minor muscle and can exclude pathological causes of shoulder pain[11]. However, teres minor atrophy on MRI, in the absence of nQSS symptoms, is often nonspecific. The Mayo group has suggested searching for dynamic PCHA occlusion during abduction-external rotation maneuvers due to extrinsic compression. Digital subtraction angiography (DSA), computed tomography angiography (CTA), and magnetic resonance angiography (MRA) have all been used to visualize PCHA occlusion[7]. However, it should be noted that the accuracy of vascular studies alone for confirming the diagnosis of nQSS is unclear. Electrodiagnostic studies can be performed after vascular imaging studies suggestive of nQSS; however, there is low EMG sensitivity due to technical difficulties in targeting the teres minor, which might be improved by ultrasound guidance.

Treatment

Conservative treatment

First line of treatment is usually conservative, while operative management is reserved for selected patients. Conservative management include oral anti-inflammatory medications,

physical therapy, and limitation of activity. Physical therapy includes internal rotation stretching with posterior rotator cuff and periscapular muscle strengthening and stretching. Almost 30% of throwers will not respond to conservative measures and will require surgical intervention.

Surgical treatment

Surgical decompression involves the removal of fibrous bands or other space-occupying lesions and neurolysis. Usually obliquely oriented fibrous bands have been identified tethering the neurovascular bundle in the quadrilateral space. Regarding the best surgical approach, the anterior (deltopectoral) approach although more demanding, reduces postoperative morbidity. The posterior approach has been found as the easier and quicker method to identify the quadrangular space and secure its contents[7-9,11-13]. During this approach, a 10 cm posterior incision is done to approach the posterior deltoid and teres minor. The muscle do not need to be detached, rather the posterior edge of the deltoid can simply be elevated to allow identification of the axillary nerve branch to the teres minor, which is decompressed, followed by decompression of the fibrous bands overlying the axillary nerve in the deep quadrangular space[10].

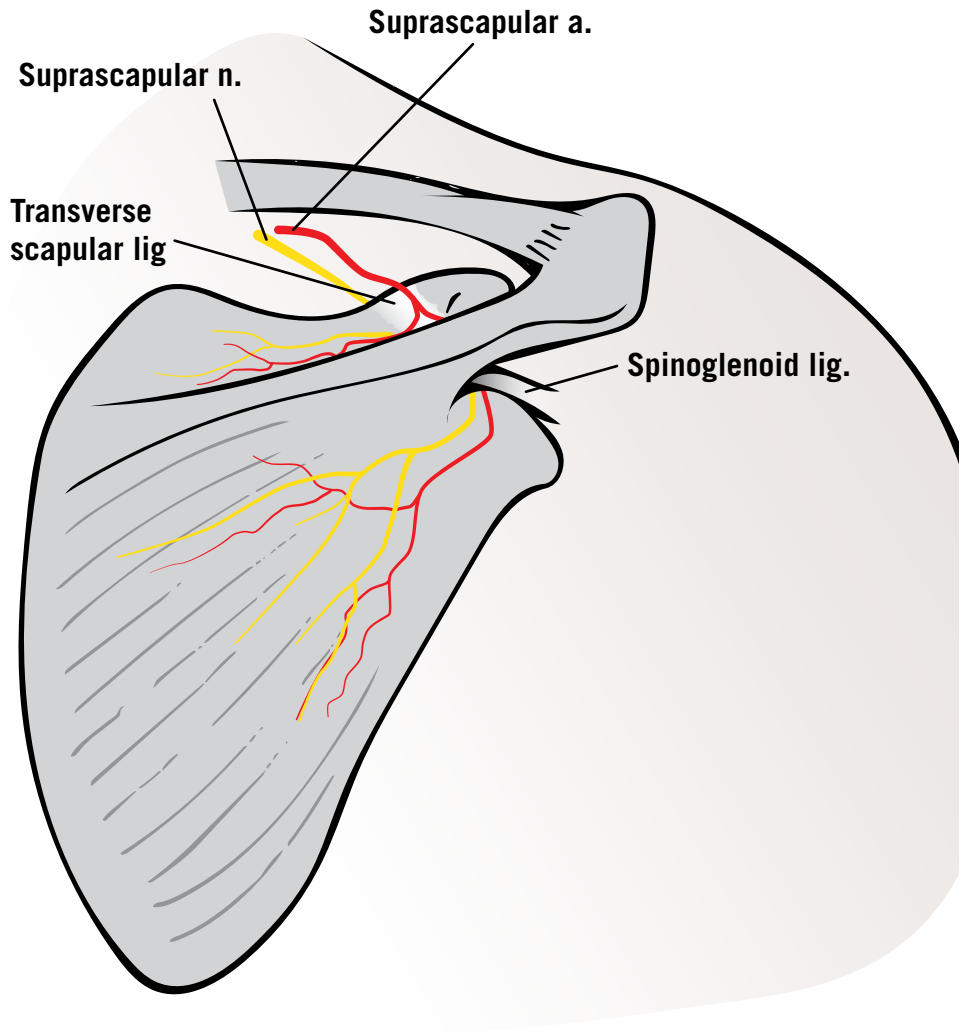

Figure 2: Anatomy of the suprascapular nerve, which passes through the suprascapular notch to provide motor branches to the supra- and infraspinatus muscles.

SUPRASCAPULAR NERVE COMPRESSION

Suprascapular nerve palsy has been reported in throwing and overhead athletes such as baseball players, tennis players, weightlifters, swimmers, handball and volleyball players. Although it is not a common injury, it is however the most frequently injured peripheral branch of the brachial plexus in athletes[14,15].

Anatomy

The suprascapular nerve is a mixed motor and sensory nerve that originates from the C5 and C6 roots (occasionally including C4) and arises from the upper trunk at Erb's point. To reach the supraspinatus and infraspinatus fossa at the posterior scapular surface, the suprascapular nerve crosses the posterior triangle of the neck, passes at the anterior border of the trapezius muscle along the posterior border of the clavicle

> ***The throwing motion is one of the fastest and most demanding maneuvers to which any joint in the body is subjected.***

and, when reaching the scapular border, passes beneath the transverse scapular ligament through the suprascapular notch to reach the supraspinatus fossa. The suprascapular notch can have variable shape and size. Typically, the suprascapular artery and vein pass above the ligament, while the nerve passes under the ligament. The suprascapular nerve then branches into 1-2 small and short motor branches to the supraspinatus muscle (in most cases just 1mm caudally to the ligament) and contributes terminal sensory nerve endings to the glenohumeral joint, the acromioclavicular joint, and the coracohumeral ligament. In about 15%, the nerve additionally provides cutaneous sensory fibers to the upper lateral arm (deltoid patch), and in 3% of patients, the nerve provides the motor branches proximal to the suprascapular notch which then pass above the transverse scapular ligament.

The suprascapular nerve then courses obliquely along the floor of the supraspinatus fossa, under the supraspinatus muscle, toward the rim of the glenoid, and continues around the lateral margin of the base of the scapular spine, also known as the spinoglenoid notch, to enter the infraspinatus fossa. There it provides 3-4 motor branches to the infraspinatus muscle, longer and larger than the motor branches to the supraspinatus muscle. The spinoglenoid ligament (or inferior transverse scapular ligament), may occasionally cover the nerve at the spinoglenoid notch although its existence has been debated. (Figure 2)

Pathophysiology

In the overhead-throwing athlete, the cause of suprascapular nerve injury is caused by repetitive microtrauma to the nerve through several mechanisms, including compression, traction, and friction, either directly or through microvascular injury.

Compression of the nerve can occur from space-occupying masses, in the notches and overlying ligaments, as well as from rotator cuff tendons. The suprascapular nerve is susceptible to compression by space-occupying lesions because of its relatively fixed position under the rotator cuff and the ligaments within the suprascapular and spinoglenoid notches. The fixed position of the nerve makes it susceptible to compression even by relatively small size lesions, such as ganglion cysts or lipomas. The ganglion cysts are usually found in relation to capsulolabral or SLAP tears and are created from a 1-way valve mechanism. The more frequent sites of compression are the suprascapular notch and the spinoglenoid notch. Compression of the nerve at the suprascapular notch may occur due to the overlying hypertrophic or calcified transverse scapular ligament. Also, the shape of the notch has been hypothesized as a risk factor for compression or injury of the nerve[16-18]. Compression at the spinoglenoid notch occurs from the spinoglenoid ligament tightening during cross-body adduction and internal rotation that occurs during the follow-through phase of throwing or serving[19]. During extreme abduction and full external rotation, the medial tendinous portions of the infraspinatus and supraspinatus muscles impinge against the lateral edge of the scapular spine, compressing the infraspinatus branch of the suprascapular nerve[20].

Another mechanism of injury is traction, or mechanical stretching of the nerve, with or without friction. The reason is that the suprascapular nerve displays several turns and sharp changes of direction around critical osseous points during its course while it is concomitantly fixated in some other points. Kinking of the nerve may be exacerbated by repetitive overhead activities and respectively the addition of more compressive forces. Again, the 2 common areas of potential fixation and kinking occur at the suprascapular and spinoglenoid notches. This stretching of the nerve can be further exacerbated by scapular protraction, or at the extremes of scapular motions such as scapular depression, retraction, or abduction. Cross-body adduction, forward flexion, and external rotation have been reported to place the nerve at risk for this mechanism of injury. Additionally, simultaneous contraction of the infraspinatus muscle, during which the contraction of the infraspinatus pulls the nerve medially while it is tethered laterally at the base of the spine of the scapula further exacerbates this stretching injury mechanism[21]. These traction forces are accentuated by the extreme torque and angular velocity placed on the shoulder during overhead activities, especially during the cocking, acceleration, and follow-through phases.

Nerve stretching 6% beyond resting length compromises nerve conduction while stretching the nerve more than 15% leads to irreversible nerve damage. Relevant findings have shown suprascapular neuropathy in professional volleyball players, which were otherwise asymptomatic but showed a 15-30% decrease of shoulder external rotation power[22]. Ferretti et al suggested that rapid, forceful external rotation entrapped the nerve at the spinoglenoid notch and that the cocked position of serving in volleyball caused increased tension on the nerve[21]. Similarly, Ringel et al showed slowing of nerve conduction velocities in baseball pitchers as the season progressed[23]. The spinoglenoid notch is the most common site of this type of injury in the throwing athlete and may present as a partial palsy, with retention of partial infraspinatus function despite visible atrophy, while also the thrower may usually compensate with the teres minor[21,24].

Diagnosis

Symptoms

The symptoms depend on the site of injury or compression and the chronicity of the condition. Symptoms tend to be less severe when the injury is localized to the spinoglenoid notch because this level of injury does not affect supraspinatus while spares 30-40% of the infraspinatus muscle. At the initial stage, the athlete may have poorly localized or even diffuse pain over the lateral and posterior shoulder, which is usually exacerbated by activity. The pain is characterized as a dull ache, or burning or deep pain and may radiate to the neck. The athlete may also experience weakness of external rotation and abduction especially during overhead activities. At later stage the pain may become constant and persist even at night.

Clinical examination

The clinical examination reveals tenderness at the location of nerve compression, usually over the suprascapular notch or the spinoglenoid notch. Tenderness over the acromioclavicular joint may also be present due to the sensory fibers. Atrophy of the infraspinatus and suprapsinatus or the infraspinatus alone will be seen depending on the location of injury in later stages, several months after the onset of the nerve compression.

Muscle testing will reveal weakness in shoulder external rotation, and positive scratch collapse test over the level of nerve compression.

Differential diagnosis

Differential diagnosis includes shoulder pathology such as rotator cuff tendinopathy or tear, labral tear, degenerative joint disease of glenohumeral and acromioclavicular joint and nerve related pain sources due to cervical radiculopathy, Parsonage-Turner syndrome, quadrilateral space syndrome, and thoracic outlet syndrome.

Imaging/nerve conduction studies

Electrodiagnostic studies (EDX) may be helpful if indicate either ischemia, conduction block from demyelination or axonal injury. However, it is sometimes difficult to localize the site of injury, and the reliability of EDX studies for supra-scapular pathology is low. An ultrasound-guided lidocaine injection in the suprascapular notch can help in diagnosis if pain is relieved; however, if not, it cannot preclude the diagnosis. An MRI can help in identifying soft tissue masses compressing the nerve and quantifying rotator cuff muscle atrophy

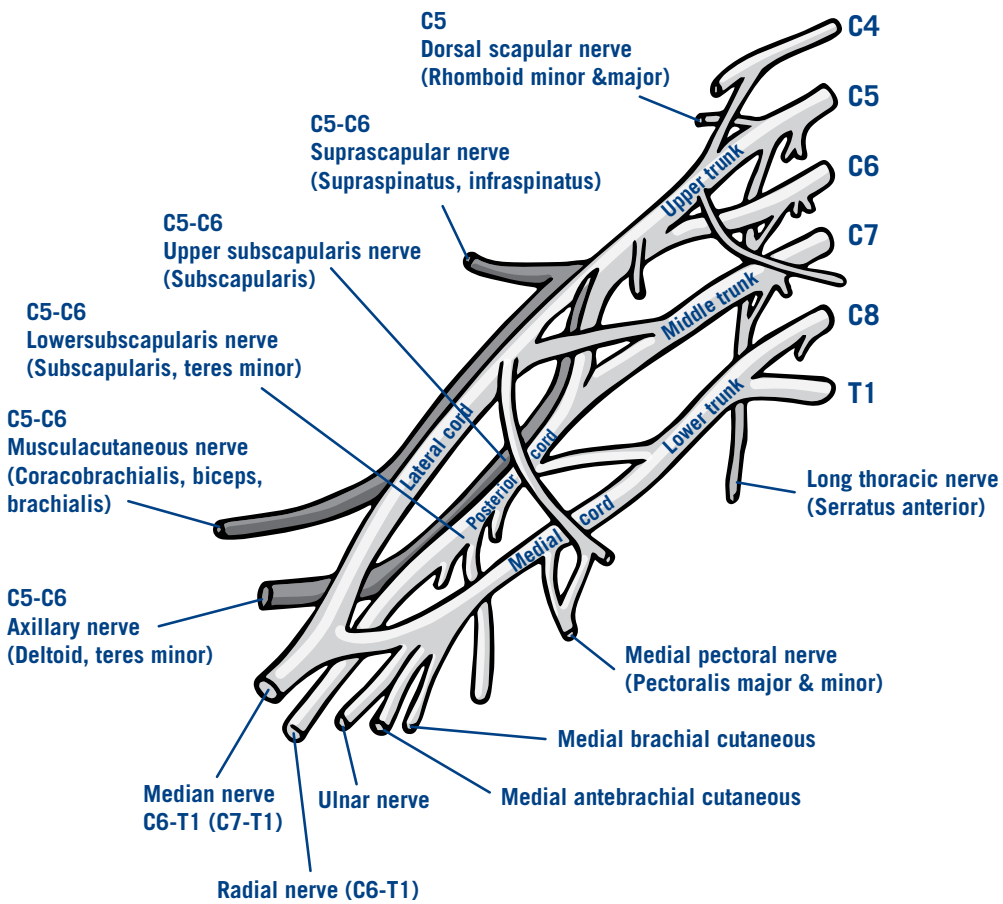

Figure 3: The anatomy of the brachial plexus.

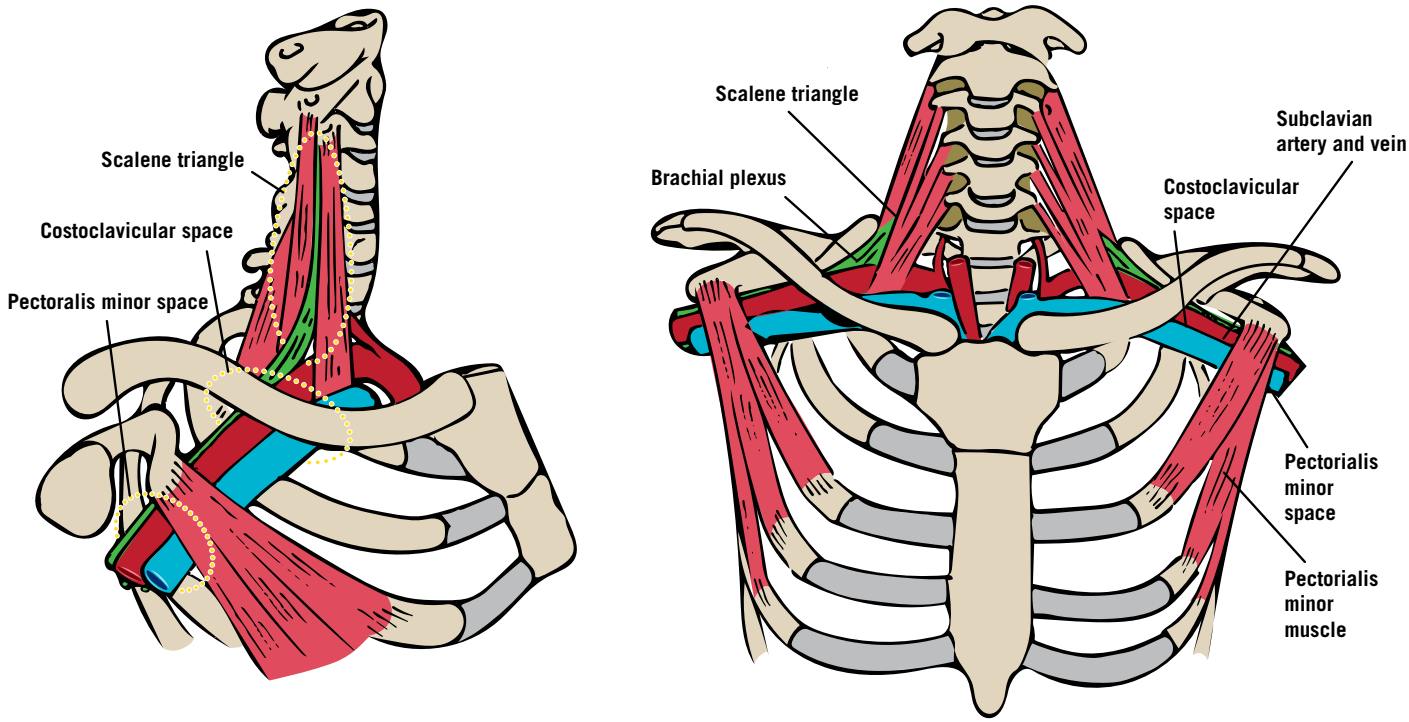

Figure 4: Neurogenic TOS is caused by dynamic compression of the brachial plexus nerves at the supraclavicular scalene triangle level, between the clavicle and first rib, or in the infraclavicular level posterior to the pectoralis minor.

and fatty infiltration. Other diagnoses of shoulder specific pathology can be ruled out.

Treatment

Conservative treatment

If there is no evidence of a space-occupying well-defined lesion, the initial treatment is nonsurgical management with activity modification and NSAIDs and analgesic medications, combined with strengthening exercises for the rotator cuff, deltoid, and periscapular muscles. Maintenance of shoulder range of motion to prevent stiffness is imperative to this nonoperative approach, along with posterior capsular stretching with the arm in abduction to relax the spinoglenoid ligament. Most patients have recovery of nerve function with this regimen[25].

Surgical treatment

Surgical intervention is warranted in recalcitrant cases when there is no improvement after 6 months of nonsurgical treatment or in cases with a compressive lesion. The superior (saber cut) approach is used to decompress the nerve at the suprascapular notch, where either a splitting of the trapezius in line with its fibers, or trapezius elevation from the scapula is used, to provide a clear view of the notch and resect the transverse scapular ligament. A posterior approach is used to decompress the nerve at the spinoglenoid notch. A posterior deltoid sparing approach is used followed by subperiosteal elevation of the infraspinatus to access the spinoglenoid notch. An arthroscopic approach is used in cases with soft tissue mass like paralabral cyst that is usually related to posterior labrum or SLAP tears, and in this way both the cyst is debrided and removed, and the intraarticular pathology is addressed[1,22].

THORACIC OUTLET SYNDROME

Thoracic outlet syndrome (TOS) describes a range of upper extremity neurovascular compressive conditions involving the brachial plexus (neurogenic TOS, NTOS) and the subclavian artery and vein (vascular TOS), as they course through the cervicoaxillary canal toward the upper extremities. Although rare, these neurovascular compressive conditions are often overlooked as an etiology for progressive limitation of high-level athletic performance in overhead athletes. Neurogenic TOS is more common in women than men by a ratio of 3.5:1.2

Neurogenic TOS

Anatomy

The C5 to T1 roots exit the intervertebral foramens and form the brachial plexus, which crosses and leaves the neck between the anterior and middle scalene muscles (Figure 3). The anterior and middle scalene muscles originate from the transverse processes of the upper cervical spine and insert on the first rib. The brachial plexus and subclavian artery course between these 2 muscles, over the first rib, and beneath the clavicle, while the subclavian vein travels over the first rib anterior to the anterior scalene before joining the subclavian artery and brachial plexus. Neurogenic TOS is caused by dynamic compression of the brachial plexus nerves at the supraclavicular scalene triangle level, between the clavicle and first rib, or in the infraclavicular level at the subcoracoid space posteriorly to the pectoralis minor (Figure 4).

Pathophysiology

The etiology of NTOS can be multifactorial and is usually the combined result of predisposing anatomic variations with local responses to tissue injury or trauma (either acute or repetitive), resulting in a narrowing of the thoracic outlet with compression

or angulation of the brachial plexus. Examples of underlying mechanisms include a repetitive injury leading to fibrosis and hypertrophy of the scalene or pectoralis minor muscles, creating scar deposition onto the brachial plexus nerves which can be further exacerbated by predisposing anatomical factors such as musculotendinous abnormalities, fascial bands, or cervical ribs. NTOS in throwers and overhead athletes such as baseball pitchers and tennis players may occur because of excessive scapular depression of the dominant arm due to inadequate scapular muscle stabilization or because of scalene, trapezius and pectorals minor muscles hypertrophy[1,26,27].

Diagnosis

Symptoms

There are no pathognomonic signs or symptoms for NTOS, and it is often a diagnosis of exclusion. The symptomatology of NTOS usually involves pain, numbness, or paresthesia/dysesthesia radiating from the neck to the shoulder and extending into the arm and hand. Paresthesias may involve the entire upper extremity and does not follow a single peripheral nerve or cervical nerve root distribution. However, most patients identify symptoms in the lower trunk or ulnar nerve distribution (C8 to T1). These symptoms may vary and may be exacerbated by overhead activities. Other complaints include weakness in up to half the patients, and clumsiness due to intrinsic muscle dysfunction. Throwers may complain of difficulty in gripping the racket, bat or throwing object because of intrinsic muscle weakness. However, in the high-level athlete, the often intermittent nature of symptoms may lead to a delay in diagnosis. Also, the periods that are free of symptoms due to lower activity levels (eg breaks in the competitive season) or rest periods should not be used to exclude NTOS diagnosis in these athletes. In contrast, significant arm fatigue, heaviness, and other symptomatology when throwing is useful, while exercise-enhanced, ultrasound-guided, local anesthetic anterior scalene and/or pectoralis minor muscle blocks can be used to uncover NTOS diagnosis even in athletes with intermittent symptoms[28-31].

Clinical examination

Clinical examination includes a careful neurovascular assessment of the upper extremity, particularly of the muscles (in NTOS with lower trunk involvement, weakness of the intrinsic muscles of the hand) and sensation supplied by the lower brachial plexus. Symmetrical pulses, extremity size, color, and skin temperature should be examined. Cervical spine examination is performed to rule out proximal causes and usually neck range of motion is unaffected. A valuable diagnostic finding is to elicit symptoms with overhead arm position and by direct palpation over the brachial plexus at the supraclavicular or infraclavicular space. Provocative maneuvers include the Adson maneuver, the Wright test, the Roos stress test, and costoclavicular maneuvers[1,22,27].

Standardized clinical diagnostic criteria for NTOS have been proposed to help unrevealing this difficult diagnosis in a recent prospective cohort study that validates a consensus-based standardized set of clinical diagnostic criteria for neurogenic TOS[29]. This facilitates the setting of NTOS diagnosis in cases with upper extremity symptoms extending beyond the distribution of a single cervical nerve root or peripheral nerve, that present for at least 12 weeks, are not adequately explained

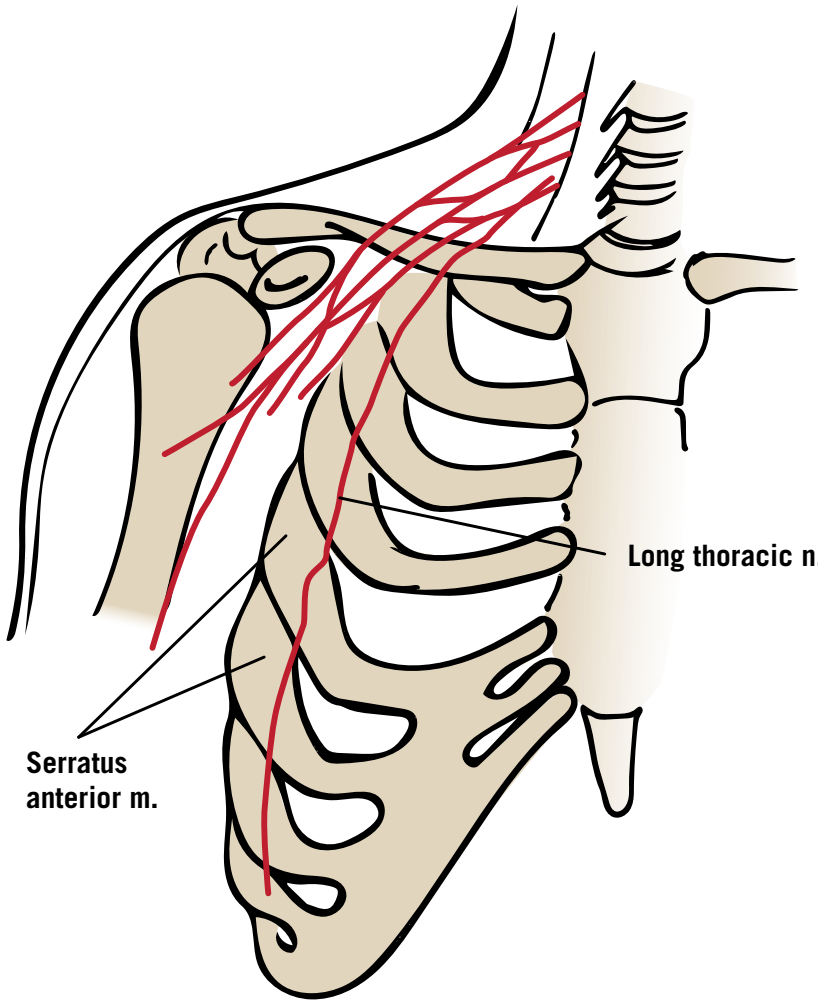

Figure 5: The long thoracic nerve can be compressed from repetitive traction in throwing athletes or from direct trauma to the lateral thorax.

by another pathology, and meet at least 1 criterion in at least 4 of 5 categories: principal symptoms, symptom characteristics, clinical history, physical examination and provocative maneuvers (elevated arm stress test, EAST and upper limb tension test, ULTT)[29].

Imaging

Imaging and electrodiagnostic studies are usually negative and serve to help exclude other diagnoses rather than confirm a diagnosis of NTOS.

Treatment

Conservative treatment

The first line of treatment of NTOS is nonoperative and relies on appropriate physical therapy and additional measures including relative rest of the affected upper extremity, nonsteroidal anti-inflammatory medications, and muscle relaxants. The main goal of physical therapy is to decrease external nerve compression resulting from muscle imbalance. This is achieved by restoring proper muscle balance in the cervical and thoracic regions via stretching, strengthening, and postural measures. Specific goals of the focused physiotherapy are to improve posture using postural feedback, to relax the scalene muscles and decompress the scalene triangle and subcoracoid spaces, as well as to restore muscle imbalance of the neck, upper back and shoulder by strengthening scapular stabilizers, especially the trapezius, rhomboid, and levator scapulae muscles.

Improvement may not be apparent until after the first 2 to 3 months of therapy and typically it takes 4 to 6 months of appropriate physiotherapy to realize significant improvement. Although no specific data for throwers exist, evidence shows that most patients (ranging from 50% up to more than 80%) with NTOS are effectively treated with appropriate physiotherapy alone and not require operative intervention[1,29,32-34].

Surgical treatment

Surgical treatment is recommended for patients with refractory symptoms and no sufficient improvement with conservative therapy alone and remain with substantial disability. Surgical decompression for NTOS can be achieved through either a supraclavicular or transaxillary incision, and includes anterior and middle scalene muscles release or resection, at times removal of the first rib, as well as brachial plexus neurolysis. During the same surgery, a second incision may at times be used to perform pectoralis minor tendon release from the coracoid process for patients with evidence of compression at that level[26]. A structured stepwise postoperative rehabilitation program is recommended after NTOS decompression. Any attempts to rush the rehabilitation process may lead to recurrence due to excessive muscle spasms with subsequent perineural scar tissue deposition. Full rehabilitation and return to high-level competition often take 9–12 months after surgical decompression. Outcomes after decompression for NTOS show almost 85–90% of the high-level overhead athlete population reporting significant symptom improvement[30,35].

LONG THORACIC NERVE PALSY

Anatomy

The long thoracic nerve is a motor nerve that innervates the serratus anterior muscle. It originates from the ventral rami of C5 to C7. It passes through the middle scalene muscle and courses behind the brachial plexus to penetrate the proximal serratus anterior muscle; therefore, the nerve is relatively tethered at these two points (middle scalene and serratus anterior). The serratus anterior muscle originates from the upper eight ribs and inserts on the medial border of the scapula. The actions of this muscle are to keep the scapula close to the thorax by drawing it forward and to stabilize it during arm motion (Figure 5).

Pathophysiology

Long thoracic nerve palsy in the thrower can occur by repetitive traction microtrauma and stretching during throwing with the arm in the overhead position and the head and neck being tilted away from the arm to

the contralateral direction[1,27,36]. The nerve can also be injured during maximal protraction of the scapula or after a direct blow to the shoulder or lateral thorax.

Diagnosis

Symptoms

The athlete may experience neck, shoulder, or scapular discomfort. Pain may be localized at the rhomboids, and the levator scapulae muscles. Loss of velocity and weakness may be noted during throwing motion. The thrower may also feel the popping of the scapula due to scapular dyskinesia.

Clinical examination

Clinical examination will reveal scapular dyskinesia especially during forward flexion of the arms and during wall push-up. Forward flexion range of motion and strength may be decreased.

Imaging

Imaging using MRI will rarely show fatty infiltration or atrophy of the serratus anterior. Electrodiagnostic studies may help to confirm the diagnosis, but are often inconclusive.

Treatment

Conservative treatment

Nonoperative treatment has been used initially for athletes with long thoracic nerve palsy. Range of motion exercises help to avoid further contracture of periscapular muscles, followed by periscapular and rotator cuff muscle strengthening. Maximal recovery may take as long as 2 years although a small amount of scapular dyskinesia may persist[36,37].

Surgical treatment

Surgical treatment may be offered for patients that show no improvement following conservative management. Nerve decompression surgery can be attempted at early stages of nerve compression, without significant nerve damage.

Nerve transfer of thoracodorsal to long thoracic nerve has provided good functional recovery and can be done few months (3-4) after the onset of weakness[38]. In later stages muscle transfers or, rarely, scapulothoracic fusion may be necessary. Muscle transfer of the sternal head of pectoralis major to the inferior angle of scapula provides a relatively stable, pain-free scapula and preserves scapulothoracic motion[39,40].

CONCLUSION

Treatment of the throwing athlete with shoulder pain remains among the more challenging aspects of orthopedic sports medicine. Nerve compressions and pain syndromes should be included in the differential diagnosis of these athletes. Early detection of symptoms, careful clinical examination, and exclusion of other usual structural pathologies around the shoulder may raise suspicion of possible nerve compression syndrome for the throwing athlete. A coordinated approach among trainers, therapists, and physicians is required for the comprehensive treatment of nerve compression symptoms and related shoulder pain in the throwing athlete.

References

Available at www.aspetar.com/journal

Frantzeska Zampeli MD PhD[1],

Theodorakys Marin Fermin MD[2],

Elisabet Hagert MD PhD[3,4]

1. *Hand, Upper Limb & Microsurgery Department, KAT Attica General Hospital*
   Athens, Greece
2. *Centro Médico Profesional Las Mercedes*
   Caracas, Venezuela
3. *Aspetar Orthopedic and Sports Medicine Hospital*
   Doha, Qatar
4. *Karolinska Institutet*
   Stockholm, Sweden

Contact: fzampeli@gmail.com