Marc R. Safran
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Nerve injuries about the shoulder in athletes are being recognized with increasing frequency. Prompt and correct diagnosis of these injuries is important to treat the patient and to understand the potential complications and natural history, so as to counsel our athletes appropriately. This 2-part article is a review and an overview of the current state of knowledge regarding some of the more common nerve injuries seen about the shoulder in athletes, including long thoracic nerve, spinal accessory nerve, burners and stingers, and thoracic outlet syndrome. Each of these clinical entities will be discussed independently, reviewing the anatomy, mechanism of injury, patient presentation (history and examination), the role of additional diagnostic studies, differential diagnosis, and management.

**Keywords:** nerve; long thoracic nerve; spinal accessory nerve; burner; stinger; thoracic outlet syndrome; winging; scapular winging; trapezius palsy; serratus anterior palsy

LONG THORACIC NERVE INJURY

Traction injury to the long thoracic nerve has been identified in the players of many sports, including tennis, volleyball, archery, golf, gymnastics, bowling, weight lifting, soccer, hockey, and rifle shooting.\(^9^9\) This injury, although uncommon, can be quite disabling to athletes.

**ANATOMY.** The long thoracic nerve arises from the anterior branches of C5 through C7, and occasionally C8 (in 8%). In about 20% of patients, there is a contribution from the intercostal nerves. The C5 to C6 nerve roots pass through or on the scalenus medius muscle, whereas the C7 nerve passes between the anterior and middle scalene muscles. The C5 and C6 nerves unite with the C7 nerve distal to the scalene muscles to form the long thoracic nerve. The nerve travels anteriorly to the scalenus posterior muscle and traverses distally and laterally, going below the clavicle and under the first or second rib. The nerve then travels along the chest wall in the midaxillary line to the outer border of the serratus anterior, sending branches to each of the serratus anterior muscle digitations (Figure 1). The long thoracic nerve is 22 to 24 cm in length.\(^3^5,3^6\)

The serratus anterior muscle arises from the first through ninth ribs and inserts onto the costo-medial border of the scapula. The upper fibers of the serratus anterior insert onto the superior angle of the scapula and act to stabilize the scapula during the initial stages of abduction.
The middle fibers insert onto the vertebral border of the scapula and are instrumental in protraction of the scapula. The lower fibers of the serratus anterior insert onto the inferior angle of the scapula. They are a primary upward rotator of the scapula during abduction. The middle fibers insert onto the vertebral border of the scapula and are instrumental in protraction of the scapula. The lower fibers of the serratus anterior insert onto the inferior angle of the scapula. They are a primary upward rotator of the scapula during abduction.

PATHOPHYSIOLOGY. The proposed origin of long thoracic nerve palsy can be divided into 2 main categories: traumatic and nontraumatic. Acute or recurrent trauma has been identified as the most common cause of injury to this nerve. Repetitive microtrauma is a frequent cause of long thoracic nerve injury, especially in athletes. This usually occurs as a result of traction to the nerve. With the athlete’s head tilted or rotated laterally away from the affected extremity and the arm raised overhead, the nerve may become stretched. In fact, the length of the nerve may double between points of relative fixation. The points of fixation include the scalene medius muscle and the superior aspect of the serratus anterior. It has been shown that, in general, neurapraxia can occur as a result of an increase in length of only 10%. So doubling nerve length will likely result in neurapraxic lesion at the least and in a more severe injury at worst. This position of traction is common in throwing a baseball, football, or javelin and spiking or serving a volleyball or tennis ball. However, minor stretching, such as with yoga, has also been reported as a cause of this injury. More severe traction may occur when there is shoulder depression in conjunction with contralateral neck bending, such as seen with tackling. Regardless of the mechanism of stretching, it is unclear where along the course of the nerve that the injury occurs. Repetitive microtrauma with repeated stretch injury may result in a vascular intimal injury, as outlined in part 1 of this study (see “Suprascapular Nerve Pathophysiology”).

The long thoracic nerve is quite susceptible to direct trauma because of its subcutaneous location as it exits the pectoralis muscle at the fourth or fifth rib. Nontraumatic causes include compression at one of many sites, including within the scalene muscles (medius and posterior), 3 cm proximal to the first rib, between the clavicle and the second rib, between the second rib and the coracoid, by compression at the inferior angle of the scapula, or by compression by inflamed bursae. Traction over a fascial band from the inferior aspect of the brachial plexus extending to the proximal aspect of the serratus anterior has been postulated as another potential cause, as the nerve has been shown in cadavers to bow-string over this band with the arm in abduction and external rotation, accentuated by proximal and medial migration of the scapula. There are many bursae along the course of the long thoracic nerve, including the subsacral, accessory subscapular, subcoracoid, and supracoracoid bursae, which may compress this nerve.

PRESENTATION AND CLINICAL EXAMINATION. Athletes with a long thoracic nerve injury will usually complain of pain or discomfort about the shoulder, neck, and/or scapular area, which is exacerbated by activity and/or by tilting head away from and elevating the affected arm. Often, especially early in the process, the pain may not be severe enough to stop sports. The pain may only last 2 to 3 weeks. Athletes may note a loss of throwing or tennis serving speed or power. Weakness may be noted during overhead activity, followed by difficulty with forward elevation and overhead motions. The person will complain most often of an insidious onset of weakness. The patient may complain of winging; particularly, some will note a protuberance of the scapula when sitting in a high-back chair. Other symptoms include complaints of glenohumeral instability and subacromial bursitis as well as a history of being involved in sports with repetitive shoulder use, such as baseball, tennis, or volleyball, or contact sports such as football.

On physical examination, there will often be decreased active forward elevation and loss of power/strength. On examination from behind, the subject will have winging, especially at the inferior border and particularly with forward elevation and/or wall push-ups (Figure 2). The subject may also have diastrophism scapulohumeral rhythm. In a thin or muscular subject, there may be wasting or atrophy of the serratus anterior. The differential diagnosis of long thoracic nerve injury includes cervical disk disease, brachial plexitis (Parsonage-Turner syndrome), rotator cuff tendinitis or tear, adhesive capsulitis, degenerative arthritis of the glenohumeral joint, degenerative arthritis of the acromioclavicular joint,
There are several causes of scapular winging (Table 1). It is important to differentiate the cause of winging from the other common neurologic cause—palsy of the trapezius. Spinal accessory nerve injury (CN XI) may result in atrophy or paralysis of the trapezius muscle, producing scapular winging clinically. However, in addition to atrophy of the trapezius, spinal accessory nerve palsy results in winging with the arms in abduction and not with forward elevation (Figure 3). Furthermore, the winging associated with spinal accessory nerve injury usually involves the superomedial scapula.

![Figure 2. Long thoracic nerve palsy. Winging of an 18-year-old woman with a 3-year history of left shoulder pain due to a long thoracic nerve palsy sustained while involved in a waterskiing accident.](image)

**TABLE 1**

<table>
<thead>
<tr>
<th>Causes of Winging of the Scapula</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary scapula winging</strong></td>
</tr>
<tr>
<td>Neurologic types</td>
</tr>
<tr>
<td>Long thoracic nerve/serratus</td>
</tr>
<tr>
<td>anterior palsy</td>
</tr>
<tr>
<td>Spinal accessory nerve/trapezius</td>
</tr>
<tr>
<td>palsy</td>
</tr>
<tr>
<td>Dorsal scapular nerve/</td>
</tr>
<tr>
<td>rhomboideus palsy</td>
</tr>
<tr>
<td>Bony types</td>
</tr>
<tr>
<td>Osteochondroma</td>
</tr>
<tr>
<td>Fracture malunion</td>
</tr>
<tr>
<td>Soft tissue types</td>
</tr>
<tr>
<td>Contractural winging</td>
</tr>
<tr>
<td>Muscle avulsion or muscle</td>
</tr>
<tr>
<td>agenesis</td>
</tr>
<tr>
<td>Scapulothoracic bursitis</td>
</tr>
<tr>
<td><strong>Secondary scapular winging</strong></td>
</tr>
<tr>
<td>Voluntary scapular winging</td>
</tr>
<tr>
<td>Voluntary scapular winging</td>
</tr>
</tbody>
</table>

![Figure 3. Spinal accessory nerve palsy. A 17-year-old male patient with a 1-year history of shoulder pain. A, winging of the superior medial angle of the scapula with abduction. A line drawn along the medial border of the normal left shoulder is to be compared with the symptomatic right shoulder. Forward elevation does not result in winging in this patient, differentiating spinal accessory nerve palsy winging from long thoracic nerve palsy winging (B).](image)
Tests used to confirm the diagnosis, rule out other causes, and evaluate for correctable lesions that may have caused the long thoracic nerve palsy include neurodiagnostic tests and radiographic evaluation. Nerve conduction velocity and EMG are helpful to confirm the diagnosis, to document the severity of nerve injury, and to follow recovery. Plain radiographs are usually unremarkable but should be obtained to assess for a cervical rib, which may be a cause of long thoracic nerve injury, and for calcifications. Other imaging studies, such as CT scans and MRIs, are not particularly helpful.

**NATURAL HISTORY.** The natural history of atraumatic long thoracic nerve palsy is resolution within 1 year. However, cases due to Parsonage-Turner syndrome (brachial plexitis) may take as long as 2 to 3 years to resolve. It has been reported that nonoperative treatment will not be successful in approximately one quarter of patients affected by long thoracic nerve injury.

**TREATMENT.** Because of the relatively good prognosis for spontaneous recovery, the mainstay of initial management is a nonoperative program. Nonoperative treatment for this problem includes relative rest, symptomatic management, reassurance, maintaining shoulder range of motion, strengthening of compensatory muscles, and possibly bracing. With regard to relative rest, the athlete should avoid lifting heavy objects or participating in activities that exacerbate symptoms and place the nerve at risk. Pain should be managed symptomatically by use of nonsteroidal anti-inflammatory drugs or other medications for neurogenic pain. Athletes should be reassured and informed of the fact that most cases of atraumatic long thoracic nerve injury subside within 6 to 9 months, and almost all cases resolve satisfactorily within 12 months. It should be stressed to the patient to maintain full glenohumeral range of motion, actively, passively, and actively assisted. Furthermore, strengthening of the scapular stabilizers, particularly the trapezius, rhomboids, and levator scapulae, should be instituted because these are stressed more in the face of a nonfunctioning or dysfunctional serratus anterior. Use of an orthosis has been recommended to help hold or support the scapula to the chest wall. This has been reported to relieve pain, control the stability of the scapula, and prevent overstretching of the serratus anterior. However, it has been the author’s experience, as well as the experience of others, that success with this custom brace for this injury is inconsistent.

The indications for surgery for patients with a long thoracic nerve injury include symptoms that persist beyond 1 to 2 years despite nonoperative management, and no improvement as assessed by EMG. Traditionally, surgical options include muscle transfers, scapulopexy, and scapulothoracic fusion, although none of these procedures will allow return to most competitive sports that require arm strength and motion. Options for muscle transfers include using the pectoralis major, serratus minor, rhomboids, and teres minor. Recently published series of the transfer of the sternal head of the pectoralis major tendon for serratus anterior paralysis have reported mostly good to excellent results, with consistent improvement in function, resolution of winging, and relief of pain (Table 2). In 1979, Gozna and Harris reviewed 14 patients with serratus anterior palsy. Three of the 14 patients had nonoperative treatment failure and ultimately needed surgery. All 3 recovered satisfactory shoulder function after transfer of the sternal head of the pectoralis major, although 1 patient underwent reoperation. In 1995, Post presented 8 patients treated with sternal head transfer and a reinforced, single-limb fascia lata autograft extension, with an average 2-year follow-up. All patients had excellent results. Most recently, Noerdlinger et al updated Post’s series, reporting on 15 patients with a follow-up of 64 months. Twelve patients (75%) reported that they would undergo the procedure again. Pain decreased in 11 patients, whereas function improved in 10 patients. The results were excellent in 2 patients, good in 5, fair in 4, and poor in 4. Better results occur in patients attaining at least

<table>
<thead>
<tr>
<th>Author</th>
<th>No. of Patients</th>
<th>No. of Surgeries</th>
<th>Follow-up</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gozna and Harris</td>
<td>14</td>
<td>3</td>
<td></td>
<td>All 3 had satisfactory function; 1 reoperation</td>
</tr>
<tr>
<td>Post</td>
<td>8</td>
<td>8</td>
<td>Average 2 y</td>
<td>All excellent</td>
</tr>
<tr>
<td>Noerdlinger et al</td>
<td>15</td>
<td>15</td>
<td>64 mo</td>
<td>12 would undergo the procedure again; pain decreased in 11 patients; function improved in 10 patients; excellent in 2 patients, good in 5, fair in 4, poor in 4; better results when at least 60° of external rotation postoperatively; most returned to preoperative level of activity</td>
</tr>
<tr>
<td>Connor et al</td>
<td>11</td>
<td>11</td>
<td>41 mo</td>
<td>10 (91%) had improvement in motion, function, reduction of pain, and elimination of scapular winging; 1 unsatisfactory, recurrence of winging secondary to noncompliance postoperative</td>
</tr>
<tr>
<td>Warner and Navarro</td>
<td>8</td>
<td>8</td>
<td></td>
<td>7 had satisfactory results; 1 unsatisfactory, deep infection and graft removal</td>
</tr>
</tbody>
</table>

TABLE 2

Results of Pectoralis Major Transfer for Long Thoracic Nerve Palsy
60° of external rotation postoperatively. Most patients returned to their preoperative levels of activity with minor adaptations. In 1997, Connor et al reported on 11 patients treated with fascial autograft reinforcement of the attachment of the transferred tendon. At an average follow-up of 41 months, 10 of 11 patients (91%) had significant improvement in motion and function, reduction of pain, and elimination of scapular winging. One patient had an unsatisfactory result, with recurrence of winging secondary to noncompliance with the postoperative rehabilitation program. Warner and Navarro reviewed their experience with sternal head transfer and autogenous semitendinosus and gracilis tendon augmentation. Seven of 8 patients had satisfactory results. The 1 unsatisfactory result was attributable to deep infection necessitating graft removal.

It is this author's preference to perform a transfer with the sternal head of the pectoralis major because of its substantial excursion, similar electromyographic activity, and power as the serratus anterior, and because the orientation of its fibers approximates that of the serratus anterior after transfer. Preservation of the clavicular head of the pectoralis major rather than use of the entire tendon maintains internal rotation strength and provides a cosmetically favorable breast contour. The author prefers the use of the semitendinosus-gracilis graft because of its strength, minimal morbidity, and ease of harvest and handling.

Recently, there have been reports of surgical management of long thoracic nerve dysfunction without muscle transfer or fusion. One study has reported results of supraclavicular neurolysis of the long thoracic nerve that was dysfunctional because of compression within the scalene muscles. These authors noted that neurolysis 10 to 35 months after the onset of symptoms resulted in excellent results in all 4 patients. Other investigators have reported nerve transfer of the thoracodorsal nerve or medial pectoral nerve to the long thoracic nerve, with good functional results in patients with symptomatic long thoracic nerve injury.

**SPINAL ACCESSORY NERVE INJURY**

The spinal accessory nerve, although not a true peripheral nerve, can also be considered in the discussion of nerve injuries about the shoulder because it supplies the trapezius muscle, an important muscle for shoulder and scapular function. Injury to the spinal accessory nerve is rare in sports, although it may be injured with surgery about the neck. The spinal accessory nerve is injured less frequently than one would expect based on its relatively vulnerable course superficially in the neck.

**ANATOMY.** The spinal accessory nerve (cranial nerve 11) is the sole motor innervation of the trapezius muscle. The spinal accessory nerve exits the base of the skull at the jugular foramen and passes obliquely through and penetrates the sternocleidomastoid muscle in its upper third. The nerve then runs subcutaneously at the floor of the posterior cervical triangle as it courses to supply the trapezius muscle. The trapezius muscle is a large flat muscle that covers the back of the neck and upper half of the trunk and has 3 main sets of fibers that allow for shrugging of the shoulders, retraction of the scapulae, scapular stabilization, and scapular rotation. More specifically, the upper fibers upwardly rotate and elevate the scapula, the middle fibers stabilize and retract it, and the lower fibers downwardly rotate and depress it. The trapezius is the only muscle in the body that elevates the lateral tip of the scapula and with it the rest of the upper extremity. Thus, one of its most important functions is to resist drooping of the shoulder, assist in abduction of the arm, and allow for the arm's use for overhead activities.

**PATHOPHYSIOLOGY.** Because of its subcutaneous location, the spinal accessory nerve is quite susceptible to injury. Mechanisms of injury include penetrating injury, such as a stab wound or gunshot; blunt trauma, such as with a direct blow or stretching of the nerve during a fall or motor vehicle accident; and iatrogenic injury, such as with a radical neck dissection for tumor, carotid endarterectomy, subcutaneous mass or cyst excision, or inadvertent sectioning during a cervical lymph node biopsy. In sports, spinal accessory nerve injury may be the result of a blow from a hockey stick or lacrosse stick. The spinal accessory nerve has also been sacrificed to use as a cable graft.

**PRESENTATION AND CLINICAL EXAMINATION.** Patients with injury to the spinal accessory nerve often complain of disabling pain, weakness, and deformity. In addition to loss of function of the trapezius muscle, the sternocleidomastoid is also involved. They complain of inability to fully elevate or abduct their upper extremity overhead. Injury to this nerve results in drooping of the shoulder; asymmetry of the neckline; winging of the scapula; weakness of forward elevation, particularly above the horizontal; weakness in abduction due to the resultant paralysis of the trapezius; and loss of its normal functions as noted above. Patients may complain of a dull ache and/or heavy feeling about the shoulder. The loss of normal scapulohumeral rhythm and downward droop of the scapula and acromion may result in subacromial impingement, causing pain. Pain may also develop from coexistent adhesive capsulitis, muscle spasm (usually involving the levator scapulae or rhomboids major and minor), or radicular pain from traction on the brachial plexus from the drooping shoulder. Many patients can compensate and live with the inability to use their arms overhead, although athletic function is frequently significantly impaired if the sport involves upper extremity strength, particularly at or above eye level.

Examination of the patient starts with evaluation of the shoulders from behind with the shoulders exposed (Figure 3A). With the patient at rest and the arms at the side, the contour of the normal shoulder should be compared with that of the shoulder in question, while looking for asymmetry. The muscles are assessed for atrophy or spasm. Patients typically have an asymmetric neckline...
because of the atrophic trapezius and drooping of the shoulder girdle. The patient is asked to actively elevate both arms in the forward plane, and the relationship between the scapula and chest wall is observed. Winging of the scapula occurs as it is displaced laterally, rotating downward and outward (Figure 3B). The patient is often unable to shrug the affected shoulder, although rested levator scapulae can produce a normal shrug test. Difficulty abducting the extremity above the horizontal plane and weakness elevating against resistance are characteristic. Neer's weight duration test, in which the patient holds a heavy object such as a chair at arm's length, is helpful for reporting the presence of fatigue pain.

Plain radiographs of the cervical spine, chest, and shoulder, although rarely diagnostic, are obtained; CT or MRI scans are not necessary unless other diagnoses, such as mass lesions or disk disease, are suspected. The most helpful studies in confirming trapezius dysfunction are electromyography and nerve conduction studies. Specifically, the condition of the trapezius, sternocleidomastoid, and potentially transferable muscles (levator scapulae, rhomboideus major, and rhomboideus minor) is assessed. Periodic EMGs follow recovery of the injured nerve and help in the decision of nerve exploration versus muscle transfer. Ultrasound has also been found to be useful in the study of the spinal accessory nerve.

**NATURAL HISTORY.** The natural history of spinal accessory nerve injury depends on the cause of the nerve palsy. Blunt trauma or traction injury may recover within 12 months. Because pain is often activity related, those with lesions of the nondominant extremity or those who can successfully alter their lifestyles may not need surgical intervention. However, penetrating trauma or laceration of the nerve generally does not recover and requires early surgical intervention.

**TREATMENT.** Nonoperative treatment that has been described to treat paralysis of the trapezius includes modalities, electrical stimulation, heat and cold, transcutaneous electrical nerve stimulation, nerve blocks, and acupuncture. Resistance exercises are performed in an attempt to strengthen the accessory muscles to eliminate pain and improve drooping of the shoulder and weakness in elevation. Braces have also been used to support the scapula against the chest wall, but often this is of little use, particularly in the long term. These forms of nonoperative treatment are generally unsuccessful, especially in active patients.

Several reconstructive procedures have been proposed to substitute for the paralyzed trapezius muscle, including (1) static stabilization of the medial border of the scapula to the vertebræ, although this does not allow for compensation of the complex muscular function of the trapezius; (2) transfer of the levator scapulae with fascia lata sling; (3) dynamic muscle transfer using the levator scapulae and rhomboid muscles, which is termed the Eden-Lange procedure; and (4) surgical intervention. The indications and technique of tendon transfer for trapezius muscle palsy have been described by Bigliani et al. They include chronic pain and scapular instability for at least 1 year after injury or confirmed loss of trapezius muscle function in a healthy, active patient for whom nonoperative treatment has been unsuccessful. For those patients with iatrogenic trapezius muscle paralysis, nerve exploration, lysis, or repair—particularly if there is persistent and complete paralysis of the trapezius after 3 months—is indicated. Exploration, neurolysis, and grafting have variable outcomes, with the best results noted within 6 months after injury. Patients who have failed nerve repair are also candidates for tendon transfer. For those patients who have failed to obtain adequate stability or range of motion from the above procedures, scapulothoracic fusion is the salvage procedure of choice. Patients who have heavy demands for their shoulders may benefit from scapulothoracic fusion as the primary procedure of choice. Surgery for spinal accessory nerve injury, particularly tendon transfer, is generally for functional activities of daily living and is generally not adequate for return to sports activity.

**RESULTS.** The largest published series in the English literature using this transfer was reported by Bigliani et al., although their study did not describe whether these patients were athletes or discuss athletic function. In those authors’ original series of 18 patients, 8 did not have sufficient disability to undergo surgical treatment of this problem. They reported their results for 22 patients who underwent levator and rhomboid transfer (Eden-Lange procedure) for trapezius paralysis, with an average 7.5-year follow-up. Overall, 13 patients (59%) had excellent results, 6 (27%) had satisfactory results, and 3 (14%) had unsatisfactory results. Postoperatively, 19 of the 22 patients were able to elevate the extremity above the horizontal plane with enough strength for functional activity and had no pain or only slight pain after strenuous activity, whereas none were able to do this preoperatively. Adequate pain relief was noted in 91% of patients undergoing this transfer, and significant functional improvement was noted in 87%. Sakellarides also published his series of 10 patients who underwent a similar procedure with 3- to 22-year follow-up, noting 2 excellent, 4 good, and 4 fair results.

**BURNERS/STINGERS**

The burner/stinger syndrome is one of the most common injuries seen in sports medicine, particularly American football, in which up to 65% of collegiate football players note having had a burner or stinger during their 4-year college careers. This syndrome was named for the temporary burning or stinging pain or tingling that radiates from the shoulder to the hand of one upper limb that is frequently described by the affected athlete. This syndrome is so common and familiar to athletes that most do not report it to sideline medical personnel or coaches. Although symptoms from this syndrome last seconds to minutes, in 5% to 10% of cases, neurologic deficit may last hours, days, or even weeks.

**ANATOMY.** Understanding the neuroanatomy and distinguishing between the cervical nerve roots from the...
brachial plexus can be important in differentiating the more common and frequently benign burner from the less common but more often serious cervical nerve root or spinal cord injury. Based solely on physical examination, this differentiation may be difficult.

Each cervical nerve root is formed from the confluence of segmental dorsal and ventral roots exiting the spinal cord. The ventral roots consist of the motor nerve fibers leaving the anterior horn cells of the anterior gray area of the spinal cord. Cell bodies of the sensory fibers form the dorsal root ganglia and lie close to the vertebral foramen. Just distal to the foramen, the cervical root divides into a posterior primary ramus and anterior primary ramus. The posterior primary ramus division innervates the paraspinal muscles, posterior elements of the spine, and overlying skin. The anterior primary ramus division of the C5 to T1 (and sometimes C4 and/or T2) nerve roots continues for a short course. Small branches coming off the spinal nerves form the dorsal scapular nerve (from the C5 spinal nerve), the long thoracic nerve (C5 to C7), and the phrenic nerve (C3 to C5), before the first part of the brachial plexus is formed. The brachial plexus then supplies the sensation and motor function of the upper limbs.

There are several anatomical reasons the cervical nerve roots are more susceptible to injury than the brachial plexus as a result of traction injury (Table 3).

**PATHOPHYSIOLOGY.** Burners and stingers are the result of nerve traction or compression, particularly involving C5 and C6. Several mechanisms have been reported to cause this unilateral shoulder/arm pain and dysesthesia. Controversy still exists regarding whether a burner is primarily a brachial plexus or cervical root injury, which is compounded by the lack of clear demarcation about where the cervical nerve root ends and the brachial plexus begins. Players complain of symptoms of the upper brachial plexus or cervical roots C4 and C5, and occasionally C6. It is uncommon for the athlete to complain of lower cervical or trunk symptoms. The mechanisms frequently described as the cause of this syndrome result in upper trunk or cervical root symptoms. Lower cervical root symptoms are evident with the shoulder abducted or fully extended.

There are 3 main mechanisms described that may result in a burner or stinger. When combined with neck extension, a compression injury may result on the side the head and neck are tilted toward due to narrowing of the intervertebral foramen. This narrowing is most pronounced at the C4 through C5 and C5 through C6 levels. The narrowing occurs with neck extension and with lateral bending and is narrowed even further when the 2 motions are combined. It is this mechanism that predominated (83%-85%) as the cause of burners in published series attempting to identify the mechanism of injury. It is thought that this mechanism occurs in more mature athletic populations (collegiate and professional). Burners associated with this mechanism are seen in athletes with preexisting (although perhaps asymptomatic) cervical spine conditions, such as cervical disk disease or degenerative change.

Forces that lead to distraction of the shoulder from the head and neck can result in this injury. This can occur during blocking or tackling in football or landing on the shoulder with the head tilted away in wrestling, the second most common sport in which this injury occurs. The shoulder is driven downward and the head and neck are driven in the opposite direction (Figure 4). This leads to nerve traction as the shoulder is being depressed and the nerves are fixed proximally. The traction forces are transmitted to the upper trunk of the brachial plexus to the upper cervical nerve roots (C5-C6), stretching and injuring these structures. It is suggested that these occur more frequently in the younger athletes without cervical stenosis or arthritic change. This is the mechanism most frequently cited in the literature as the cause of burners.

Another mechanism of injury not commonly described in the literature is a direct blow to the supraclavicular region. It is at this point that the brachial plexus is most superficial and, thus, most vulnerable to direct trauma. The plexus can get compressed when an opposing player’s helmet or shoulder hits the shoulder pad, which is then driven toward the superior medial scapula, causing compression of the nerve between the helmet/shoulder pad and the bony scapula.

**PRESENTATION AND EXAMINATION.** The history from an athlete with a burner usually comprises a traumatic episode with transient (seconds to minutes) numbness, weakness, and/or electrical pain that shoots down the arm to the hand. Their complaints are typically unilateral. Although most burners are probably cervical nerve root injuries, rarely will athletes with this problem complain of neck pain. It is most commonly associated with blocking or tackling, and thus, it more commonly affects those athletes participating in American football who play defensive back, linebacker, and/or lineman.

| 1. The cervical roots lack protective epineurium, perineurium, fascicular structure, and fascicular plexiform arrangement that protect the brachial plexus from stretch and compression. |
| 2. The dural ligaments have an anchoring effect on the cervical roots and create a countertraction force proximally when there is traction from the brachial plexus distally. |
| 3. The roots pass through a rigid bony canal, the intervertebral foramen, which may be narrowed by various anatomical factors. |
| 4. The cervical roots are pressed against the vertebral transverse processes by the adventitia of the vertebral artery. |
| 5. Hypertrophy of the scalene muscles can lead to tearing of the cervical roots, and as they tighten, additional compression against the transverse process occurs. |
| 6. The plexiform structure of the brachial plexus tolerates a greater amount of tension than the cervical nerve roots and is surrounded by more compliant soft tissue structures. |
Most athletes with burners and stingers have a normal examination by the time they reach the sideline. The examination of the athlete with a burner begins with observation. Some may come off the field shaking their heads and hands. They may hold their necks flexed to alleviate pressure on the cervical root. If significant neck pain exists or when neurologic symptoms involve 2 or more extremities, cervical spine injury should be ruled out and the athlete initiated on cervical spine precautions. The examination should begin with palpation of the neck region to assess for tenderness, swelling, or deformity. The athlete then should actively range the neck through full motion—flexion, extension, lateral bending, and rotation. This may be difficult with helmet and shoulder pads on; thus, the athlete should remove any headgear, shoulder pads, and shirt, if necessary and appropriate. If there is not significant restriction in range of motion or significant pain at the end range of motion, a Spurling maneuver may be performed to rule out radiculopathy. Neurologic testing should be performed, including evaluation of strength, reflexes, and sensation. Special attention should be taken to assess upper trunk function (deltoid, rotator cuff, and biceps) to evaluate the burner and recovery from the injury, in addition to lower trunk (ulnar nerve) to rule out other significant injuries that may be masquerading as a burner. Next, the examination should proceed to the shoul-

der to rule out other injury, including evaluating the clavicle and acromioclavicular joint for deformity and swelling as well as the supraclavicular and glenohumeral regions. A Tinel sign may be elicited by tapping in the region of Erb's point (superior and deep to the medial clavicle, just lateral to the sternocleidomastoid muscle), although the sensitivity and specificity of this test have not been reported.

The differential diagnosis of burners and stingers includes cervical spine fracture, herniated disk, cervical spine instability, cord contusion, transient quadriparesis, brachial plexitis, rotator cuff tear, rotator cuff contusion, clavicle fracture, acromioclavicular joint sprain/separation, shoulder instability, TOS, other peripheral nerve injury about the shoulder (axillary nerve, long thoracic nerve, spinal accessory nerve) or distally (median nerve, ulnar nerve, radial nerve), scapula fracture (including coracoid fracture), and proximal humerus fracture. Some of these problems may occur in conjunction with brachial plexus injury.

**DIAGNOSTIC TESTS.** A complete cervical spine series of radiographs, including AP, lateral, bilateral obliques, as well as lateral flexion and extension views, is important. Radiographs should be evaluated for loss of cervical lordosis, spinal stenosis, foraminal stenosis, and instability. Meyer and others have reported those players with a Pavlov ratio of less than 0.8 have an increased risk of experiencing a burner. Castro et al found that those athletes with a Pavlov ratio of less than 0.75 had a higher risk of recurrent stingers but not first-time stingers and, interestingly, showed the level of stenosis did not correlate with the level of stinger symptoms.

An MRI can be helpful by directly visualizing a nerve root injury while also allowing for inspection for structural injuries, such as herniated disks, ligament injuries, facet injuries, and nondisplaced fractures; MR neurography is still investigational and being evaluated for usefulness in this scenario.

Electrodiagnostic studies may be useful to localize the site of nerve injury and quantify the degree of neurologic damage. Because burners and stingers last seconds to minutes, and because EMG studies take 3 weeks for a significant injury to become positive, electrodiagnostic studies are generally reserved for severe, prolonged burners. However, continued muscular weakness at 72 hours has been shown to correlate with positive results from electrodiagnostic testing performed at 4 weeks. There have been cases reported in which the EMG results may remain abnormal for more than 4 years after the injury and after full clinical recovery. Thus, electrodiagnostic studies are not helpful in decision making with regard to return to play.

Electrodiagnostic studies may help differentiate between cervical root injury and brachial plexus injury, based on the presence or absence of cervical paraspinal abnormalities and sensory amplitudes. As compared with cervical root injury, brachial plexus injuries are characterized on neurodiagnostic studies as having normal paraspinal EMG results and decreased sensory amplitudes; EMGs otherwise show normal results with classic short-duration burners and stingers.
TREATMENT. Because most athletes with this syndrome have normal examination results by the time they reach the sideline, most can return to play that same game. The natural history of athletes who have sustained burners has not been studied.

The key to management of the athlete with a burner is prevention of recurrence. Prevention is initiated by reviewing and modifying tackling technique to avoid dropping the shoulder and rotating the head and neck resulting in lateral bending and extension. The proper technique involves tackling or blocking from a more vertical, upright position. Spearing (tackling with the crown of the helmet), as always, should be discouraged. Furthermore, appropriate fit of shoulder pads is important. Lifting the pads and using a cervical collar, a neck roll, and other devices to limit excessive neck extension (although they usually are not as helpful in reducing bending) are used by many players, coaches, and trainers; however, these have not been shown to prevent burners or recurrence of burners.

Those with prolonged burner symptoms are managed with rest and removal from play, and with nonsteroidal anti-inflammatory medications and physical therapy. Physical therapy includes neck range of motion and strengthening of the neck, shoulder, and periscapular muscles, in addition to stretching and soft tissue myofascial techniques. Ice and electrical stimulation to reduce inflammation and postural exercises and traction seem to be of benefit.

Returning to full contact depends on pain-free, full passive and active range of motion of the neck and shoulder and normal neurologic examination results, including full and symmetric strength of the shoulders and neck.

THORACIC OUTLET SYNDROME

Also known as neurovascular compression syndrome, TOS refers to a symptom complex of upper extremity pain and paresthesias that actually represents more than one clinical entity.54,123 It has been subdivided into vascular and neurologic categories. The neurologic TOS is compression of the brachial plexus, whereas the vascular TOS is the result of compression of the subclavian blood vessels as they emerge from the thorax and enter the upper limb.106 The clinical conundrum occurs because of the relative similarity of their clinical presentations. The neurogenic form is a rare entity, involving the lower trunk and/or C8 to T1 anterior primary rami. The upper trunk is less frequently affected.

Thoracic outlet syndrome is a controversial diagnosis because some clinicians doubt its existence,23 whereas others find that its constellation of clinical findings and complaints can challenge even the most astute diagnostician. Wood et al most aptly stated that TOS is “incompletely understood, difficult to diagnose and often poorly managed.”126

ANATOMY. The C5 to T1 nerves exit the intervertebral foramen to form the brachial plexus, which then leaves the neck between the anterior and middle scalene muscles. The subclavian artery exits the chest off the aortic arch, joining the trunks of the brachial plexus that lie on the anterior surface of the middle scalene muscle. The anterior and middle scalene muscles take their origin from the transverse processes of the upper cervical spine and insert on the first rib. The brachial plexus and subclavian artery travel between these 2 muscles, over the first rib, and beneath the clavicle. The subclavian vein travels over the first rib anterior to the anterior scalene before joining the subclavian artery and brachial plexus (Figure 5).

The thoracic outlet may be considered a space bounded by the relatively fixed immobile thorax, particularly the first rib and clavicle below, the pectoral tendon and coracoid process above, and the anterior and middle scalene muscles medially and laterally, respectively.

PATHOPHYSIOLOGY. Any factor that increases angulation of the neurovascular structures as they pass through the thoracic outlet or causes narrowing of the outlet may result in this syndrome. Abducting the arm to 180°, pulling the shoulders down and back (such as with backpacking), or muscle swelling from trauma, exercise, or hypertrophy may initiate TOS. Postulated contributors to this syndrome include muscle weakness, trauma, arteriosclerosis, abnormal anatomy, and poor posture.65
The brachial plexus and/or the subclavian vasculature may become compressed by the scalene muscles or by a cervical rib. Most patients note some history of trauma temporarily associated with the onset of symptoms. It has been hypothesized that fibrosis of the scalene muscles as a result of trauma to the neck, or compression due to carrying a heavy backpack, may produce TOS. Thoracic outlet syndrome may occur in athletes with excessive shoulder girdle depression or overly developed trapezius and neck musculature, as seen in weight lifters. The development of TOS in tennis players and baseball pitchers may be a result of greater muscle development of the dominant arm or increased scapular depression due to inability to maintain adequate scapular stabilization with repetitive motions. It has also been identified in other athletes as well, including swimmers (presumably due to hypertrophy of the pectoralis minor), long-distance runners, football players, and wrestlers. Compressing the neurovascular structures may also occur as the sequelae of a clavicle fracture due to abundant callus formation or nonunion or malunion, narrowing the space between the clavicle and first rib. Accessory neck muscles and fibrous bands have also been identified as causes of TOS at the interscalene region. One variation of TOS has been called the hyperabduction syndrome, which may result from compression/traction of the neurovascular structures under the pectoral tendon and coracoid with the arm in abduction.

**PRESENTATION AND EXAMINATION.** Because TOS may be a spectrum of disorders to the neurovascular structures at the thoracic outlet, clinical examination and presentation will vary. There are no pathognomonic presenting signs or symptoms to confirm this syndrome, and thus, it is often a diagnosis of exclusion. The subjective complaints and objective findings depend on which structures are being compressed—arterial, venous, or neurologic. It is more prevalent in women, particularly those with poor muscular development. It is reported that regardless of the origin, 94% to 97% of patients show some neurologic symptoms. Although patients with TOS may complain of paresthesias involving the entire upper extremity, most identify symptoms in the lower trunk or ulnar nerve distribution (C8 to T1). These paresthesias may be exacerbated by overhead activities. Other complaints include weakness in up to half the patients, pain, paresthesias, and clumsiness due to intrinsic muscle dysfunction. Tennis players and baseball players may complain of difficulty gripping a racket or bat because of the intrinsic muscle weakness. Those with the particularly rare arterial form may complain of numbness, tingling, aching, heaviness, pain, and coolness of the involved extremity and may even note the arm is paler than the contralateral arm. Those with venous compromise may complain of swelling; inability to place or remove a ring from their fingers; heaviness; mottled, blotchy, or purple discoloration; and numbness and/or tingling, particularly after exercise or activity. The affected athlete may also note venous engorgement of the superficial veins.

Examination of patients with possible TOS includes a careful neurovascular examination of the entire upper extremity, particularly of the muscles and sensation supplied by the lower brachial plexus. Inspection of the arm for symmetry with regard to pulses, extremity size, color, and skin temperature is important. Cervical spine examination to rule out more proximal causes is also important. Provocative maneuvers have been described to test for TOS, including the Adson maneuver, the Wright test, and the Roos stress test. The Adson maneuver consists of neck extension and turning of the head toward the shoulder being tested while that shoulder is slightly abducted and extended. The subject inhales while the examiner palpates the ipsilateral radial pulse. Diminution or elimination of the pulse and reproduction of the paresthesias are positive test results, provided that they do not occur on the asymptomatic contralateral side. The Wright test is performed with the subject’s arm progressively hyperabducted and externally rotated while assessing for ipsilateral radial pulse diminution and reproduction of paresthesias. The Roos stress test is performed with the patient holding his/her shoulders in abduction and external rotation of 90° while maintaining elbow flexion at 90°. Then, the patient repeatedly opens and closes his/her hands for several minutes. Reproduction of symptoms or a sensation of heaviness and fatigue is consistent with the diagnosis of TOS. Because of the variability of structures compromised, many other tests have been described. Having the patient perform a shoulder shrug will produce symptoms in patients with narrowing of the costoclavicular interval. Patients with ptosis of the scapula as a cause of the compression will exacerbate their symptoms with downward pressure on...
the shoulder girdle, such as with pulling on the arms.\textsuperscript{65} Other clinicians have described compression of the anterior cervical triangle to reproduce the symptoms in TOS. Auscultation of the subclavian artery, particularly during these stress maneuvers, is important because a bruit may indicate vascular TOS.\textsuperscript{65}

The list of problems that are in the differential diagnosis of TOS is long. The differential includes herniated cervical disk; cervical nerve root impingement; brachial neuritis; carpal tunnel syndrome; cubital tunnel syndrome; arterial insufficiency secondary to vascular occlusive disease; malignant tumors about the head, neck, and lung; reflex sympathetic dystrophy; and angina.

**DIAGNOSTIC TESTS.** Radiographs are useful to rule out a cervical rib,\textsuperscript{96} long transverse process of C7, or sequelae of a prior clavicle fracture. Accessory ribs are found in approximately 0.5\% of the general population.\textsuperscript{38} Cervical spine radiographs are useful to rule out cervical spondylolisthesis, narrowed intervertebral space, and osteophytes impinging on neural foramina. Chest radiographs may also be useful to rule out a Pancoast tumor. An MRI can be useful to rule out nerve or cord compression from degenerative cervical spine changes or herniated disks; MR angiography has been found to be useful in the diagnosis of the rare vascular compromise subset of TOS.\textsuperscript{31}

Electrodiagnostic studies, such as EMG, nerve conduction velocity, and somatosensory evoked potentials, have been used and may show abnormal results in more clear-cut or classic cases.\textsuperscript{66,64,122,124} However, many cases of neurogenic TOS have intermittent symptoms and no fixed neurologic deficits that can be identified with electrodiagnostic testing.\textsuperscript{65} Thus, the electrodiagnostic testing is useful when there are positive results, but a negative electrodiagnostic test result does not rule out TOS. It is also helpful to rule in or rule out other neurologic problems.

**TREATMENT.** The initial treatment is almost exclusively nonoperative, provided there is no severe vascular compromise. Nonoperative management includes relative rest, nonsteroidal anti-inflammatory medications, shoulder girdle (focusing on scapular stabilizers) strengthening exercises, and modalities. One goal of the exercise program is to improve posture using postural feedback, as slouching may decrease the space available for the neurovascular structures.\textsuperscript{65} Rest reduces possible repetitive irritation, whereas the medications may reduce swelling about the nerve that may be causing the symptoms. Modalities, including ultrasound and transcutaneous nerve stimulation and biofeedback, have been recommended.\textsuperscript{54,64,65,90} The success of nonoperative treatment has been reported to be 67\% to 90\%,\textsuperscript{64,65,90} The success rates range from 70\% to 90\%, with poorer results in patients with work-related TOS.\textsuperscript{51,56,103,112}

Surgery is indicated immediately for acute vascular insufficiency and progressive neurologic dysfunction. Surgery is otherwise indicated for those patients with refractory pain that fails to respond to nonoperative treatment. The surgical technique involves the release or removal of the structures that cause compression and can involve scalene muscle release, first rib resection, cervical rib excision, claviclectomy, resection of anomalous fibromuscular bands, or a combination of these procedures. The surgical approach can be supraclavicular, subclavicular, transaxillary, posterior, or combined. Because of the highly variable causes and approaches, lack of standardized evaluation, and lack of information about athletes in the treatment of TOS, a detailed review of results is not useful. Success rates range from 70\% to 90\%, with poorer results in patients with work-related TOS.\textsuperscript{51,56,103,112}
SUMMARY

Nerve injuries about the shoulder should be considered in the differential diagnosis of shoulder pain, especially when the other common causes of shoulder pain have been excluded. Clinical examination of the shoulder should include direct inspection of the entire shoulder musculature (including from the back) and strength testing of selected muscle groups. This article is a review of nerve injuries about the shoulder to remind the clinician of these problems when evaluating the athlete with shoulder pain that does not fit more common diagnoses.

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