

Proximal Neuropathies of the Shoulder and Arm

31

Electromyographers occasionally are called on to evaluate the proximal nerves in the shoulder and arm. Isolated lesions of these nerves, including the suprascapular, axillary, musculocutaneous, long thoracic, and spinal accessory, are far less frequent than the common entrapment and compressive neuropathies of the median, ulnar, and radial nerves. The electrophysiologic evaluation of proximal neuropathies in the shoulder and arm relies principally on needle electromyography (EMG). Nerve conduction studies of these nerves are limited and are complicated by technical factors. In addition, nearly all lesions in these nerves are axonal loss, and cannot be localized by focal slowing or conduction block. Similar to other mononeuropathies, the goals of the electrophysiologic study are to localize the lesion as accurately as possible, to exclude a more widespread lesion or proximal radiculopathy, and to assess the underlying severity.

SUPRASCAPULAR NEUROPATHY

Anatomy

The suprascapular nerve comes off the upper trunk of the brachial plexus, receiving innervation from both the C5 and C6 roots. The nerve runs posteriorly under the trapezius, passing through the *suprascapular notch* of the scapula to enter the supraspinous fossa (Figure 31–1). The suprascapular notch is U shaped, located along the superior border of the scapula, and covered by the *transverse scapular ligament*. The suprascapular nerve first supplies motor fibers to the supraspinatus muscle, a shoulder abductor, before proceeding laterally to supply deep sensory fibers to the glenoacromial and acromioclavicular joints, and the coracoacromial ligament. It then wraps around the *spinoglenoid notch* of the scapular spine under the spinoglenoid ligament to enter the infraspinous fossa, where it supplies motor fibers to the infraspinatus muscle, an external rotator of the shoulder. The suprascapular nerve usually carries no cutaneous sensory fibers, although rare anomalous innervations have been reported. In these rare cases, the suprascapular nerve carries cutaneous sensation to the proximal lateral arm, the area usually supplied by the axillary nerve.

Clinical

Suprascapular entrapment most commonly occurs at the suprascapular notch, under the transverse scapular ligament. Less frequently, the nerve can also be entrapped distally at the spinoglenoid notch. The suprascapular nerve is relatively immobile both at its origin at the upper trunk and at the suprascapular notch. Because both the shoulder and scapula are quite mobile, movement, especially repetitive movement, results in stretch and nerve injury (Figure 31–2). Also, like most of the major proximal upper extremity nerves, the suprascapular nerve is often prominently involved in neuralgic amyotrophy (see Chapter 30).

Rare cases of suprascapular nerve entrapment have been reported secondary to a variety of mass lesions, including

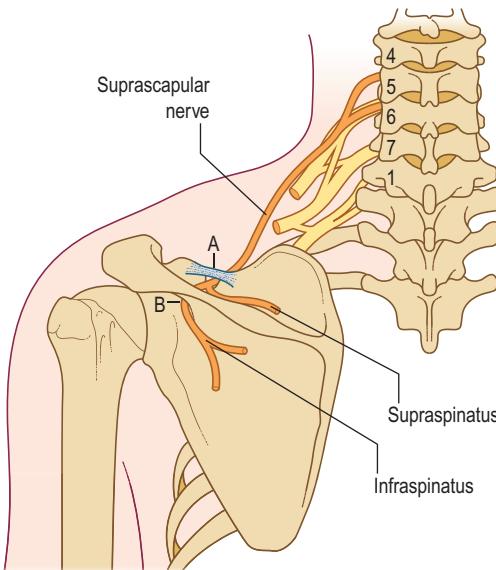


FIGURE 31–1 Anatomy of the suprascapular nerve. The suprascapular nerve originates from the upper trunk of the brachial plexus. The nerve first runs under the suprascapular notch (A) to innervate the supraspinatus muscle. Sensory fibers then are given to the shoulder joint before the nerve wraps around the spinoglenoid notch (B) to supply the infraspinatus muscle.

(Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia. With permission.)

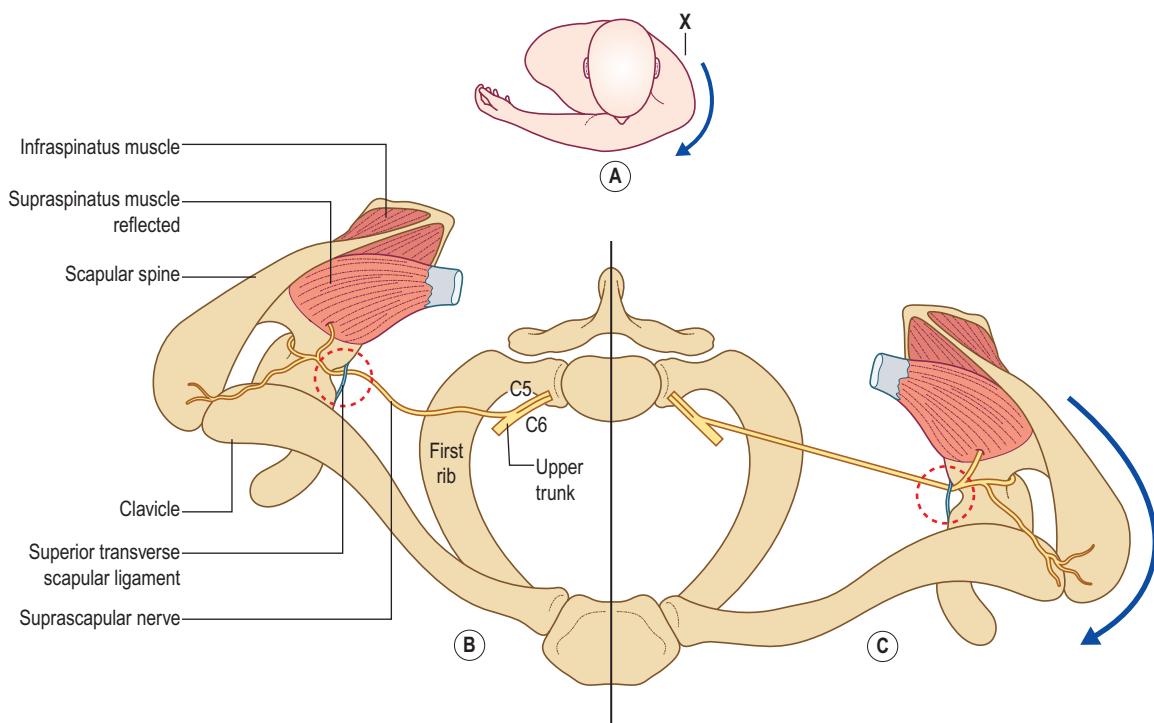


FIGURE 31–2 Suprascapular neuropathy. Suprascapular neuropathy may occur from repetitive protraction of the scapula and tethering of the nerve between the suprascapular notch and the upper trunk of the brachial plexus. Coronal view of the suprascapular nerve from above: (B) normal position, (C) nerve stretch produced by arm posture in (A).

(Reprinted from Kopell, H.P., Thompson, W.A.L., 1959. Pain and the frozen shoulder. *Surg Gynecol Obstet* 109, 92. By permission of Surgery, Gynecology & Obstetrics, now known as the Journal of the American College of Surgeons.)

ganglion cysts, sarcomas, and metastatic carcinomas. Ganglion cysts are especially common at the spinoglenoid notch. In addition, certain activities, positions, and professions are associated with suprascapular entrapment. For example, weight lifting has been implicated in several reports as a provocative factor in suprascapular entrapment, likely as a consequence of repetitive movement of the scapula, especially during lifts that involve shoulder abduction and protraction. Suprascapular neuropathy has also been reported as a consequence of positioning during surgical procedures, when patients are placed in a knee-chest position with the scapula protracted. Of interest, several professions put patients at risk for suprascapular entrapment. These include professional volleyball players, baseball pitchers, and dancers. In these professions, the clinical and electrophysiologic findings most often suggest a distal lesion at the spinoglenoid notch.

In addition, suprascapular neuropathy, which is sometimes confused clinically with a rotator cuff injury, may also accompany a rotator cuff injury. One might initially assume that both have a common traumatic etiology. However, *a suprascapular neuropathy may actually occur as a result of a rotator cuff tear*, usually a large and full thickness tear. Following a rotator cuff tear, there may be medial retraction of the tendons to the supraspinatus and infraspinatus muscles. This may result in increased tension on the

suprascapular nerve both at the suprascapular notch and the spinoglenoid notch (Figure 31–3).

Symptoms and signs depend on the site of nerve entrapment. At the most common site of entrapment, the suprascapular notch, shoulder pain may be prominent. Indeed, there is anatomic and clinical evidence that the suprascapular nerve supplies the majority of deep sensory fibers (including pain fibers) to the shoulder joint. The pain typically is described as deep and boring, occurring along the superior aspect of the scapula and radiating to the shoulder, but usually not more distally. The pain may be exacerbated by shoulder movements, especially adduction of the extended arm. This movement results in protraction of the scapula, which increases the nerve tethering between the upper trunk and the suprascapular notch. Occasionally, the suprascapular notch may be tender to palpation. Weakness involves shoulder abduction (supraspinatus) and external rotation (infraspinatus). Impairment of these motions may or may not be noticed by the patient, because both functions are subserved by other muscles as well. Atrophy may be recognized, especially over the infraspinatus muscle, which is only partially covered by the trapezius muscle (Figure 31–4).

If the entrapment occurs more distally at the spinoglenoid notch, the syndrome is limited to atrophy and weakness of the infraspinatus muscle. Pain usually is absent

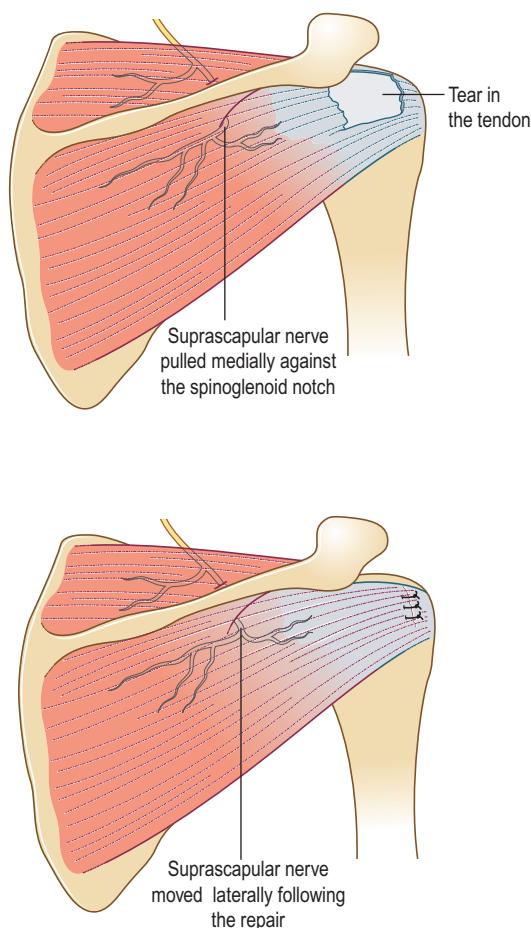


FIGURE 31-3 Suprascapular neuropathy and rotator cuff tear.
Top: Following a massive rotator cuff tear involving the tendons of the supraspinatus and infraspinatus, the muscles retract medially and can result in traction of the suprascapular nerve at the spinoglenoid notch.
Bottom: Following repair of the tendon, the traction is relieved.
 (Adapted with permission of Elsevier, from: Costouros, J.G., Porramatikul, M., Lie, D.T., Warner, J.J.P., 2007. Reversal of suprascapular neuropathy following arthroscopic repair of massive supraspinatus and infraspinatus rotator cuff tears. *Arthroscopy* 23, 1152–1161.)

because the deep sensory fibers to the shoulder joint have exited more proximally.

Several conditions may be confused with suprascapular neuropathy, including cervical radiculopathy, rotator cuff injury and other orthopedic conditions, and neuralgic amyotrophy. In contrast to suprascapular neuropathy, a C5–C6 radiculopathy may have radiating pain from the neck into the shoulder and arm, associated with sensory abnormalities in the lateral arm, forearm, and thumb. Often, the biceps and brachioradialis tendon reflexes are depressed or absent. Higher cervical radiculopathies (e.g., C3 or C4) may have a similar pain distribution to suprascapular neuropathy but are not associated with significant weakness of the shoulder or arm.

Local orthopedic conditions may be difficult to differentiate clinically from suprascapular neuropathy. Although weakness should not be present, pain often prevents full muscle activation. Exacerbation of pain by palpation (other than at the suprascapular notch) or by passive shoulder movement (other than protraction of the shoulder) would be unusual for suprascapular entrapment.

Lastly, neuralgic amyotrophy often presents with severe proximal arm and shoulder pain and later weakness (see Chapter 30). In some cases, the suprascapular nerve may be primarily involved. However, close clinical and electrodiagnostic evaluation usually reveals evidence of more widespread involvement of other nerves.

Electrodiagnosis

The goal of electrodiagnosis is to demonstrate abnormalities of the suprascapular-innervated muscles and exclude cervical radiculopathy, brachial plexopathy, or involvement of other proximal nerves. Because the suprascapular nerve has no cutaneous distribution, there is no corresponding sensory nerve to be recorded. However, as the suprascapular nerve originates from the upper trunk of the brachial plexus, studies of the sensory nerves that pass through the upper trunk should be performed to help exclude a more widespread plexus lesion. These studies should include the

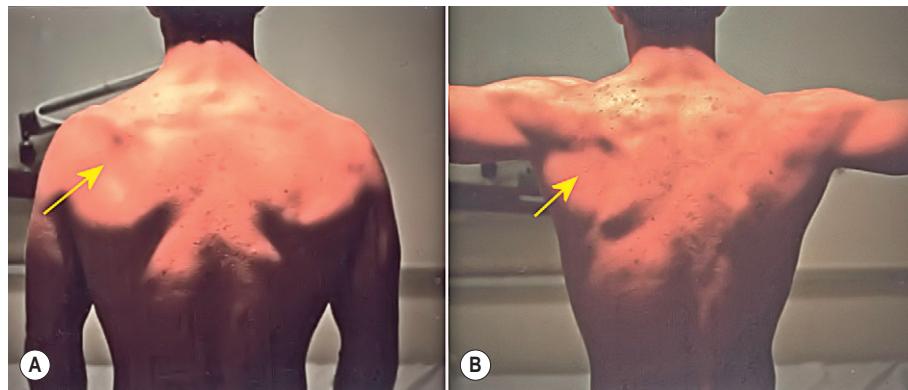


FIGURE 31-4 Suprascapular neuropathy. **A:** Shoulders relaxed; **B:** Shoulders abducted. Note the prominent atrophy of the left inferior scapular area (yellow arrows). Suprascapular neuropathy results in weakness of shoulder abduction and external rotation, without any cutaneous sensory loss.

Table 31–1. Major Upper Extremity Motor Latencies from Erb's Point Stimulation

Nerve	Muscle	Latency (ms) Upper Limit Normal	Distances (cm)*
Axillary [†]	Deltoid	4.9	15–21
Musculocutaneous [†]	Biceps	5.7	23–29
Suprascapular	Supraspinatus	3.7	7–12
Suprascapular	Infraspinatus	4.3	10–15

*Distances measured with calipers.
†The axillary and musculocutaneous nerves can also be stimulated in the axilla, with typical distal motor latencies ≤ 3.3 ms. Both axillary and Erb's point stimulation often are technically difficult. In patients with symptoms limited to one side, comparing latencies and amplitudes from side to side is always preferable to using normal value tables.
Source: From Kraft, G.H., 1972. Axillary, musculocutaneous, and suprascapular nerve latency studies. Arch Phys Med Rehabil 53, 382; and Currier, D.P., 1971. Motor conduction velocity of axillary nerve. Phys Ther 51, 503.

lateral antebrachial cutaneous nerve and the median and radial sensory nerves, especially when recording from the thumb. Often, comparison with the contralateral asymptomatic side can be useful in identifying a mild abnormality, even if the studies are normal on the symptomatic side. Any abnormality present in these sensory studies suggests a more widespread brachial plexopathy. Of course, an abnormality found in the median sensory nerve may indicate a superimposed median neuropathy at the wrist, which may need to be studied further.

Motor conduction studies can be performed, stimulating Erb's point and recording with a monopolar needle electrode in either the supraspinatus or infraspinatus muscle, or both, simultaneously (Table 31–1). A surface recording electrode should not be used to record from the spinati muscles, especially the supraspinatus, because they are covered by the trapezius. A surface reference electrode is placed distally over the shoulder joint. Compound muscle action potential (CMAP) amplitude and latency are measured. Comparing amplitude side to side can give an estimate of the amount of axonal loss present. However, these studies generally do not increase the yield over conventional EMG in terms of localizing the lesion. Typically, the pathophysiology of these entrapment neuropathies is axonal loss. Thus, although motor nerve conduction studies may show reduced amplitudes and slightly prolonged latencies, there really is no information gained over needle EMG, which more easily demonstrates axonal loss. When Erb's point stimulation is performed, high stimulating currents often are required, and supramaximal stimulation can be difficult to ensure.

During needle EMG, both the supraspinatus and infraspinatus muscles should be sampled. Care must be taken to ensure that the EMG needle is not in the more superficial trapezius muscle, by checking that no motor unit action potentials (MUAPs) are activated with a shoulder shrug. In lesions at the suprascapular notch, both the supraspinatus and infraspinatus are abnormal. With spinoglenoid lesions, however, only the infraspinatus is involved. If either of these muscles is abnormal, it is essential to sample other C5–C6 innervated muscles (e.g., deltoid, biceps, brachioradialis), as well as the cervical paraspinal muscles, to

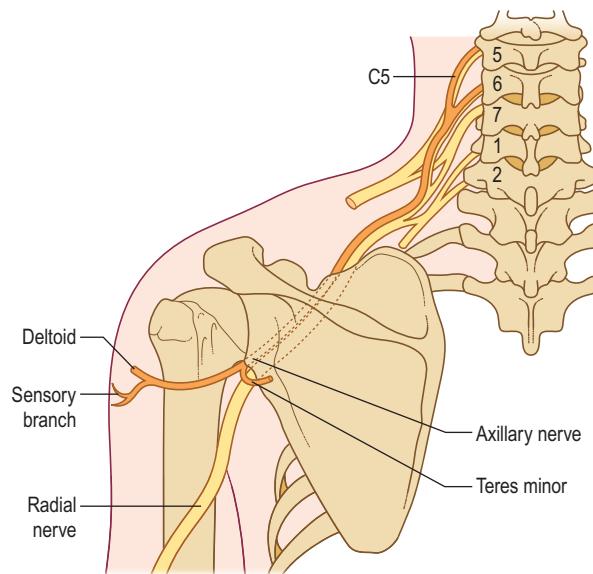


FIGURE 31–5 Anatomy of the axillary nerve. The axillary nerve originates from the posterior cord of the brachial plexus. The nerve innervates the teres minor and deltoid muscles and supplies sensation to the lateral shoulder.

(Adapted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia. With permission.)

exclude a cervical radiculopathy or more widespread brachial plexus lesion.

AXILLARY NEUROPATHY

Anatomy

Along with the radial nerve, the axillary nerve originates from the posterior cord of the brachial plexus (Figure 31–5). The axillary nerve is composed primarily of C5–C6 fibers, running through the upper trunk and posterior cord of the plexus. The nerve leaves the axilla through the *quadrilateral space*, which is formed by the humerus and the teres minor, teres major and long head of the triceps muscles (Figure 31–6). Posteriorly in the quadrilateral

space, it often divides into two major trunks. The posterior trunk always supplies the teres minor before terminating as the superior lateral brachial cutaneous nerve (i.e., axillary sensory nerve). The teres minor aids in external rotation of the shoulder while the deltoid is principally a shoulder abductor. The axillary sensory nerve supplies an oval-shaped area over the lateral shoulder. The anterior trunk travels deep to the fascia of the deltoid and always supplies the middle and anterior heads of the deltoid as well as a deep sensory branch to the shoulder joint. The posterior head of the deltoid is most commonly supplied by the posterior trunk, but some variations exist wherein it is supplied by the anterior trunk alone, and in others by a combination of the anterior and posterior trunks.

Clinical

Axillary neuropathies typically result from trauma, especially dislocation of the shoulder and fracture of the

humerus. Less commonly, athletes participating in contact sports have developed axillary neuropathies as a result of injury, typically a direct blow to the anterolateral deltoid area. Similar to suprascapular neuropathy, axillary neuropathies have been reported in professional volleyball players. Rare cases of entrapment in the quadrilateral space have been reported but are exceptional. Quadrilateral space syndrome results from compression of the axillary nerve and posterior humeral circumflex artery.

Patients with axillary neuropathies have a well-defined circular area of numbness over the lateral shoulder, along with partial weakness of shoulder abduction and external rotation (Figure 31–7). The degree of weakness varies from patient to patient. The weakness is only partial, because other muscles also contribute to shoulder abduction (i.e., the supraspinatus) and external rotation (i.e., the infraspinatus).

Electrodiagnosis

The major goal of electrodiagnosis is to demonstrate abnormalities of axillary-innervated muscles and rule out cervical radiculopathy, brachial plexopathy, or involvement of other proximal nerves. Unfortunately, there is no routine sensory nerve conduction study for the axillary nerve. However, because the axillary nerve originates from the posterior cord and upper trunk, sensory nerves that run through the posterior cord or upper trunk of the brachial plexus should be studied. These include the radial and lateral antebrachial cutaneous sensory nerves and the median sensory nerve, especially when recording the thumb. To detect mild abnormalities, comparison with the contralateral asymptomatic nerve is suggested, even if the studies are normal on the symptomatic side. Abnormalities of any of these sensory studies suggest a more widespread brachial plexopathy.

Axillary motor nerve conduction studies can be performed, stimulating the axilla and Erb's point and recording with a monopolar needle or surface electrode over the deltoid (Table 31–1). A surface reference electrode is placed distally over the deltoid tendon. To calculate a conduction velocity, distances must be measured with calipers.

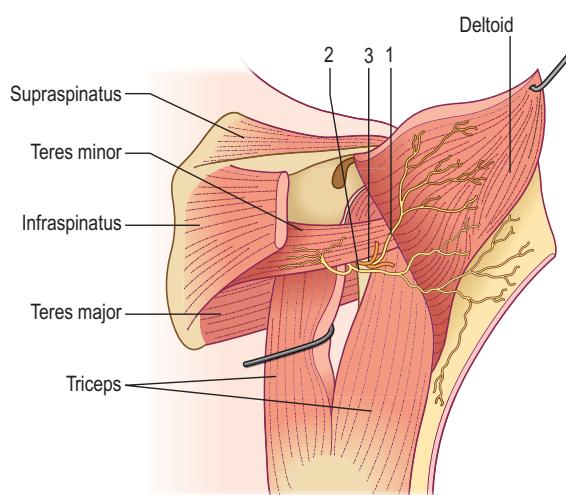


FIGURE 31–6 Posterior view of the quadrilateral space. Anterior (1) and posterior (2) branches of the axillary nerve and circumflex artery (3).

(From Paladini, D., Dellantonio, R., Cinti, A., et al., 1996. Axillary neuropathy in volleyball players: report of two cases and literature review. *J Neurol Neurosurg Psychiatry* 60, 345–347. With permission.)

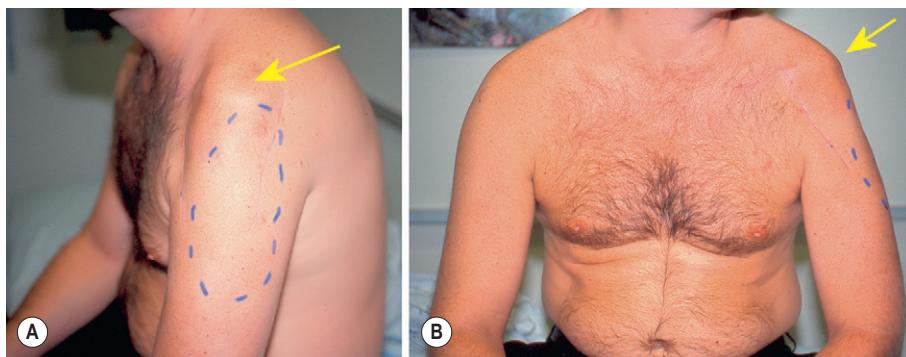


FIGURE 31–7 Axillary neuropathy in a patient following humeral fracture. **A:** Lateral view; **B:** Anterior view. Axillary neuropathy results in atrophy of the lateral shoulder girdle (yellow arrow), weakness of shoulder abduction and external rotation, and sensory loss in an oval area over the lateral shoulder (blue dashed oval).

CMAP amplitude can be compared both from side to side, to assess the amount of axonal loss, and between the axilla and Erb's point on the symptomatic side, to look for conduction block. These studies can be technically difficult to perform, however, especially obtaining supramaximal stimulation, and are best used to assess axonal loss by comparing the symptomatic side to the asymptomatic side. Because these usually are axonal loss lesions, motor studies generally do not increase the yield of localizing the lesion beyond what is obtained from routine needle EMG.

In axillary neuropathies, needle EMG is used to demonstrate denervation, reinnervation, or both in the two axillary innervated muscles, the deltoid and the teres minor. All three heads of the deltoid are easily accessible to needle EMG; the teres minor is more difficult to study. If abnormalities are found in any of these muscles, it is essential to sample other muscles innervated by the upper trunk and posterior cord of the brachial plexus to ensure that the abnormalities found in the axillary-innervated muscles are not part of a more widespread brachial plexus lesion or cervical radiculopathy. Muscles that are important to check include the biceps, supraspinatus, infraspinatus, triceps, and brachioradialis. In addition, the cervical paraspinals should be sampled to help exclude a C5–C6 radiculopathy.

MUSCULOCUTANEOUS NEUROPATHY

Anatomy

The musculocutaneous nerve arises directly from the lateral cord of the brachial plexus (Figure 31–8). In the upper arm, it pierces the coracobrachialis muscle to run in the fascia between the biceps and brachialis muscles. It innervates all three of these elbow flexor muscles, including the biceps, brachialis, and coracobrachialis. The brachialis muscle also commonly receives some innervation from the radial nerve nearby, although clinically, this is of little or no importance. In the region of the elbow, the musculocutaneous nerve runs deep to the brachial fascia, over the brachialis muscle. Past the elbow, its terminal extension continues as a pure sensory nerve, known as the *musculocutaneous sensory* or *lateral antebrachial cutaneous sensory nerve*. In the forearm, the nerve becomes subcutaneous and separates into two terminal divisions (anterior and posterior) to supply sensation to the lateral half of the forearm.

Clinical

Isolated musculocutaneous neuropathies are rare. Patients with nontraumatic musculocutaneous neuropathies have been reported due to strenuous physical activity (e.g., weight lifting, rowing, throwing a football), surgery, and pressure during sleep. There is a single report of a musculocutaneous neuropathy associated with repetitive carrying of items on the shoulder with the arm curled around the object (labeled the “carpet carrier’s palsy”). Similarly, there

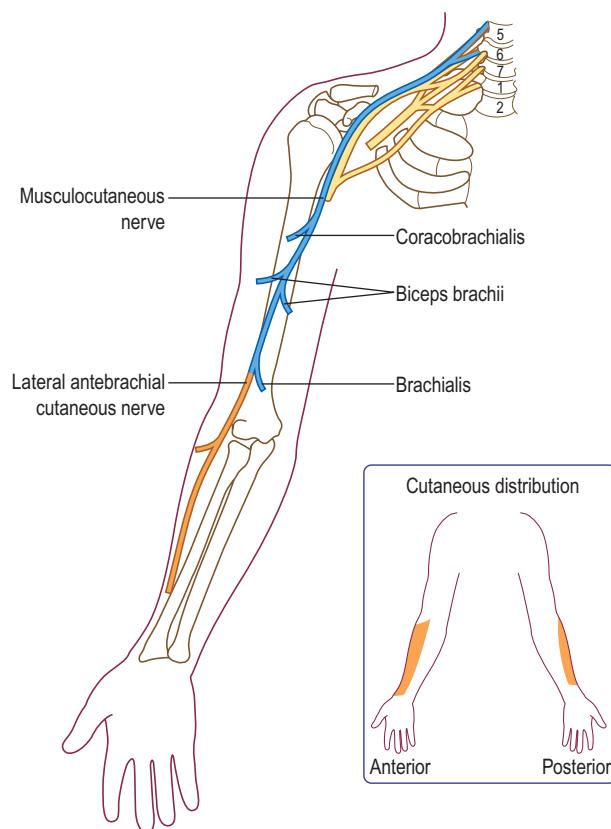


FIGURE 31–8 Anatomy of the musculocutaneous nerve. The musculocutaneous nerve originates from the lateral cord of the brachial plexus. The nerve innervates the biceps, brachialis, and coracobrachialis muscles. It then continues past the elbow as a pure sensory nerve, the lateral antebrachial cutaneous nerve, to supply sensation to the lateral forearm.

(Reprinted from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia. With permission.)

is one report of osteochondroma of the humerus compressing the musculocutaneous nerve.

More commonly, musculocutaneous neuropathies occur as part of more widespread traumatic lesions of the shoulder and upper arm, especially fractures of the proximal humerus. Clinically, musculocutaneous neuropathies result in weakness of elbow flexion, an absent biceps reflex, and sensory loss in the lateral forearm.

More common is entrapment of the distal musculocutaneous sensory nerve. This occurs at the elbow, where the nerve can become entrapped between the biceps tendon or fascia and the brachialis muscle. Characteristically, patients report worsening pain or paresthesias, or both, when the arm is pronated and extended, a position that increases the pressure on the nerve at the elbow site. A hyperextension injury of the elbow, such as may occur during sports-related activities such as tennis, also may cause musculocutaneous sensory neuropathy. Examination in these cases shows isolated altered sensation in the lateral forearm, with normal muscle strength and reflexes. There may be tenderness to palpation over the nerve at the elbow.

Electrodiagnosis

The aim of the electrophysiologic exam is to demonstrate isolated involvement of the musculocutaneous nerve and to exclude a brachial plexopathy, cervical radiculopathy, or involvement of other proximal nerves. The most important nerve conduction study to perform is the lateral antebrachial cutaneous sensory study. This sensory potential can be easily elicited by stimulating just lateral to the biceps tendon at the elbow and recording over the nerve 12 cm distally, on a line connecting the stimulation point to the radial pulse. Comparison with the contralateral side is useful in cases where symptoms are limited to one side. Musculocutaneous neuropathies, both distal and proximal, result in abnormal lateral antebrachial cutaneous sensory nerve action potentials (SNAPs). When an abnormal potential is found, it is important to check other sensory potentials, especially those that pass through either the lateral cord or the upper trunk of the brachial plexus (e.g., median and radial SNAPs). Abnormalities found in these nerves suggest a more widespread brachial plexopathy. As noted earlier, comparison with the asymptomatic side is helpful, especially if the studies are at the lower limits of normal.

Similar to axillary motor studies, proximal motor nerve conduction studies can be performed stimulating the axilla and Erb's point and recording with either a monopolar needle or surface electrode over the biceps (Table 31-1). A surface reference electrode is placed distally over the biceps tendon. The CMAP amplitude can be compared both from side to side, to assess the amount of axonal loss, and between the axilla and Erb's point, to look for a conduction block. A conduction velocity can be calculated but requires calipers to measure the distance accurately. In contrast to the sensory studies, these motor studies are more technically difficult, especially obtaining supramaximal stimulation, and are best used to assess the degree of axonal loss by comparing the symptomatic side with the asymptomatic side. Similar to axillary and suprascapular neuropathies, musculocutaneous neuropathies usually are axonal loss lesions. Accordingly, motor studies generally do not increase the yield of localization over performing the needle EMG alone.

In distal musculocutaneous neuropathies at the elbow, the needle EMG is normal. In proximal lesions, EMG demonstrates denervation or reinnervation, or both, with decreased recruitment of motor unit action potentials (MUAPs) in the biceps. The brachialis and coracobrachialis can also be sampled but are more difficult than the biceps and offer no additional information. If abnormalities are found in the biceps, it is essential to sample other upper trunk and lateral cord innervated muscles to ensure that the abnormalities found are not part of a more widespread brachial plexus lesion or cervical radiculopathy, especially if the lateral antebrachial cutaneous SNAP is normal. Important muscles to check include the pronator teres and flexor carpi radialis (lateral cord) and deltoid, brachioradialis, supraspinatus, and infraspinatus (upper trunk). In

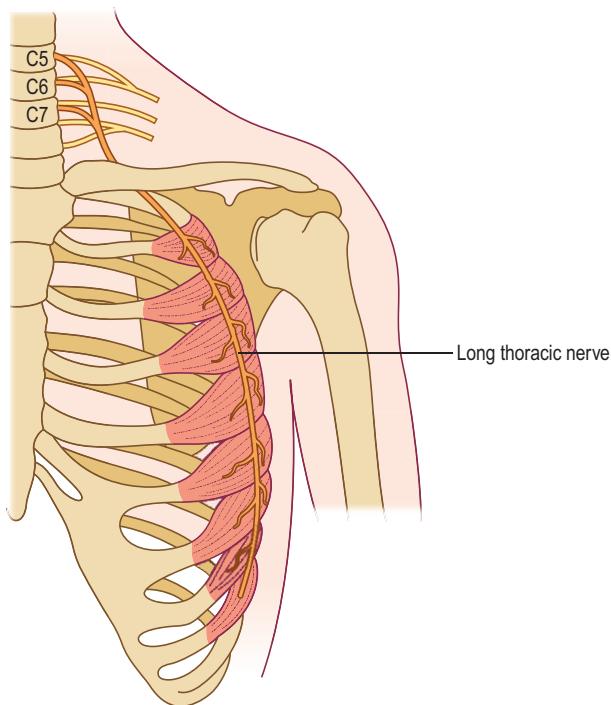


FIGURE 31-9 Anatomy of the long thoracic nerve. The long thoracic nerve originates directly from the C5, C6, and C7 roots, proximal to the brachial plexus proper. It runs inferiorly to supply the serratus anterior muscle. There is no cutaneous sensory innervation. (Reprinted from Fisher, M., 1993. Other mononeuropathies of the upper extremity. In: Brown, W.F., Bolton, C.F. (Eds.), Clinical electromyography, second ed. Butterworth, Boston, p. 271. With permission.)

addition, the cervical paraspinals need to be sampled to help exclude a C5–C6 radiculopathy.

LONG THORACIC NEUROPATHY

Anatomy

The long thoracic nerve arises directly from the C5–C6–C7 roots, before the brachial plexus proper (Figure 31-9). The nerve runs inferiorly to innervate only one muscle, the serratus anterior. The serratus anterior arises from the first 8 to 10 thoracic ribs and inserts on the costal margin of the scapula. Anatomically, the serratus anterior muscle can be thought of as having an upper portion, supplied by C5–C6 fibers, and a lower portion, supplied by C7 fibers. The upper portion is responsible principally for scapular protraction, and the lower portion for scapular stabilization. Protraction is the movement of the scapula forward along the chest wall.

Clinical

Long thoracic nerve palsies may occur as part of a more widespread traumatic lesion affecting the cervical roots. Although isolated long thoracic palsies have also been reported as a consequence of external compression and stretch, most result from neuralgic amyotrophy (see Chapter 30). Indeed, in some attacks of neuralgic

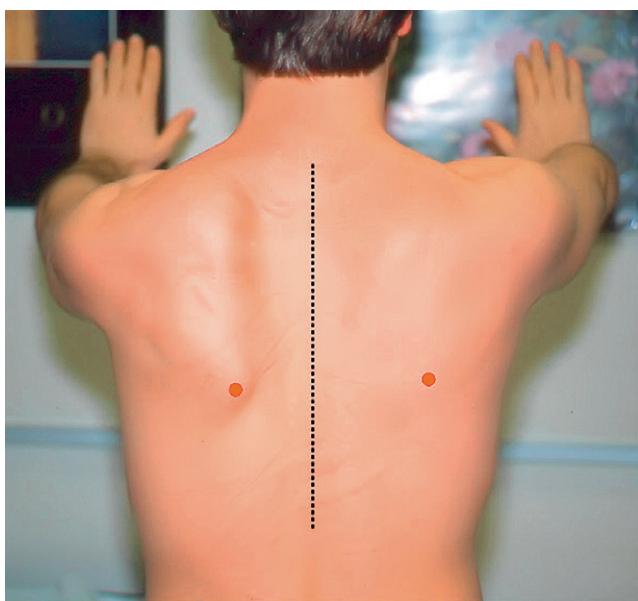


FIGURE 31-10 Long thoracic neuropathy. Isolated weakness of the serratus anterior occurs in long thoracic neuropathies. Serratus anterior weakness results in “winging” of the scapula. Winging becomes most pronounced with the arm extended in front of the body. As the serratus anterior normally pulls the scapula forward, weakness of the serratus anterior results in the scapula being displaced closer to the midline. In this figure, a red circle marks the tip of each scapula. Note the left scapula is displaced closer to the midline than the right scapula.

amyotrophy, the long thoracic nerve is affected in isolation. Patients describe severe pain in the shoulder region that lasts several days to weeks. As the pain abates, patients note difficulty with shoulder movement. Weakness or paralysis of the serratus anterior characteristically results in “winging” of the scapula (Figure 31-10). Winging from serratus anterior dysfunction becomes most pronounced when the arm is extended in front of the body. As the serratus anterior normally pulls the scapula forward against the ribs, weakness of the serratus anterior results in the inferior tip of the scapula being displaced closer to the spine. Because the serratus anterior is a shoulder stabilizer, other shoulder muscles may also appear weak (e.g., deltoid, supraspinatus, infraspinatus). If these muscles are tested with the examiner’s hand pressed against the scapula, however, much of the “weakness” will disappear. EMG is especially useful in trying to differentiate true neurogenic weakness from poor shoulder fixation and functional weakness. As the long thoracic nerve has no cutaneous distribution, there is no area of altered sensation or numbness in isolated lesions of the long thoracic nerve.

Electrodiagnosis

The electrodiagnosis of long thoracic nerve palsy is challenging. There is no reliable way to study this nerve with nerve conduction studies. Although Erb’s point stimulation can be attempted, with monopolar needle recording, these studies are seldom of practical use and are potentially hazardous because of the risk of pneumothorax. To look for

evidence of a more widespread brachial plexus lesion, sensory nerve conduction studies should be performed, studying especially those nerves that travel through the upper and middle trunks of the brachial plexus, and which have the same root innervation as the long thoracic nerve. These studies include the lateral antebrachial cutaneous, median, and radial sensory nerves.

The electrodiagnosis relies on the needle EMG. In long thoracic nerve palsy, abnormalities are limited to the serratus anterior muscle. Unfortunately, the serratus anterior is a difficult muscle to study. Although it can be sampled under the inferior angle of the scapula, it is most approachable with a needle where it arises from the mid-thoracic ribs in the mid-axillary line. Caution must be taken to insert the needle over the rib proper and not into the inter-space, where there is a risk of pleural puncture and pneumothorax.

Other C5–C6–C7-innervated limb muscles (e.g., biceps, deltoid, supraspinatus, infraspinatus, triceps, pronator teres) should be sampled to exclude a cervical radiculopathy, brachial plexopathy, or involvement of other proximal nerves. In addition, the cervical paraspinal muscles should be checked to help exclude a more proximal lesion at the roots.

SPINAL ACCESSORY NEUROPATHY

Anatomy

The spinal accessory nerve is a pure motor nerve, with no cutaneous sensory fibers. The spinal accessory nerve is derived from the C1–C4 cervical segments (Figure 31-11). The nerve ascends through the foramen magnum, to return through the jugular foramen. It first supplies motor innervation to the sternocleidomastoid muscle and then runs superficially in the posterior cervical triangle to innervate the trapezius muscle. It is at this latter location where the nerve is most susceptible to injury. Some branches from the cervical plexus may also contribute to the innervation of the upper trapezius directly.

Clinical

Often, spinal accessory nerve palsies occur in the region of the posterior cervical triangle, resulting in isolated weakness of the trapezius. This may occur from stretch or external compression, but most commonly occurs after local surgical procedures. Cervical lymph node biopsy is the most common procedure that injures the spinal accessory nerve, reported to occur in 3–10% of all such procedures. The trapezius is the major suspensory muscle of the shoulder. The upper fibers of the trapezius elevate the scapula and rotate its lateral angle upward; the intermediate fibers adduct and retract the scapula; and the lower fibers depress and rotate the scapula downward.

In distal spinal accessory palsies, atrophy and weakness of the trapezius occur, resulting in a shoulder drop (Figure 31-12). The destabilized scapula moves downward from

the weight of the limb. It also moves laterally away from the spine as a result of the unopposed action of the serratus anterior. In this posture, the head of the humerus cannot articulate properly with the glenoid, resulting in impaired shoulder abduction. Mild scapular winging may also be seen, especially during attempted arm abduction. Indeed, an intact trapezius is needed for proper shoulder fixation, and essentially all movements around the shoulder. A destabilized shoulder from trapezius weakness often results in apparent weakness of other shoulder movements as well. Thus, it is not uncommon for patients with a spinal accessory neuropathy to be misdiagnosed clinically as a brachial

plexopathy or other proximal neuropathy, in addition to primary orthopedic problems of the shoulder. Indeed, patients with a spinal accessory neuropathy commonly go many months before the correct diagnosis is reached. Adding to the confusion is that pain and paresthesias may occur, presumably from traction on the brachial plexus as a result of the dropped shoulder. The dropped shoulder can also cause similar symptoms on a vascular basis, from compression of the axillary artery, resulting in pain and paresthesias (Figure 31-13).

In the less common proximal lesions of the spinal accessory nerve, weakness of the sternocleidomastoid muscle, in addition to trapezius weakness, occurs. This manifests as weakness of neck flexion, as well as contralateral turning of the head and neck.

Electrodiagnosis

The spinal accessory nerve is easy to study, especially compared with the other proximal nerves in the upper extremity. This nerve is also used for routine repetitive nerve stimulation studies. Motor studies can be performed with surface recording electrodes over the upper trapezius. The active recording electrode is placed over the muscle belly, with the reference electrode placed distally over the shoulder joint. Stimulation is performed just posterior to the middle of the sternocleidomastoid muscle. The nerve is superficial at this point and can be stimulated with low current intensities. Supramaximal stimulation can easily be achieved here, as opposed to Erb's point stimulation.

The CMAP from the upper trapezius can be compared with the contralateral side. Because the posterior sternocleidomastoid is the only easily accessible stimulation site, the major use of this study is to measure the distal CMAP amplitude and compare it to the contralateral side, in order to estimate the amount of axonal loss. More proximal studies of the spinal accessory nerve are not easily performed.

Because the spinal accessory nerve carries no sensory fibers, there is no corresponding sensory nerve conduction study to perform. In patients who appear to have shoulder weakness from poor fixation, however, it is reasonable to

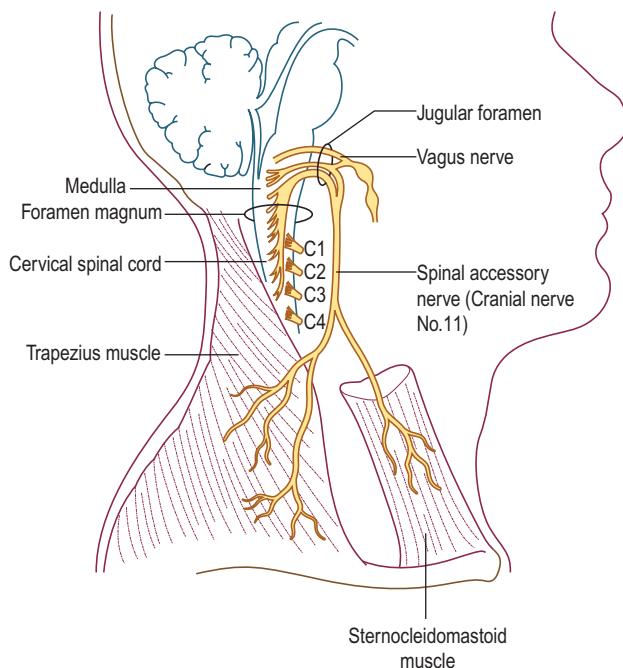


FIGURE 31-11 Anatomy of the spinal accessory nerve. The spinal accessory nerve originates from the C1–C4 roots, ascending through the foramen magnum and then returning via the jugular foramen. The nerve first innervates the sternocleidomastoid muscle before running over the posterior cervical triangle to innervate the trapezius muscle. There is no cutaneous sensory innervation.

(Reprinted from Spence, A., 1982. Basic human anatomy, second ed. Benjamin Cummings, San Francisco. With permission.)

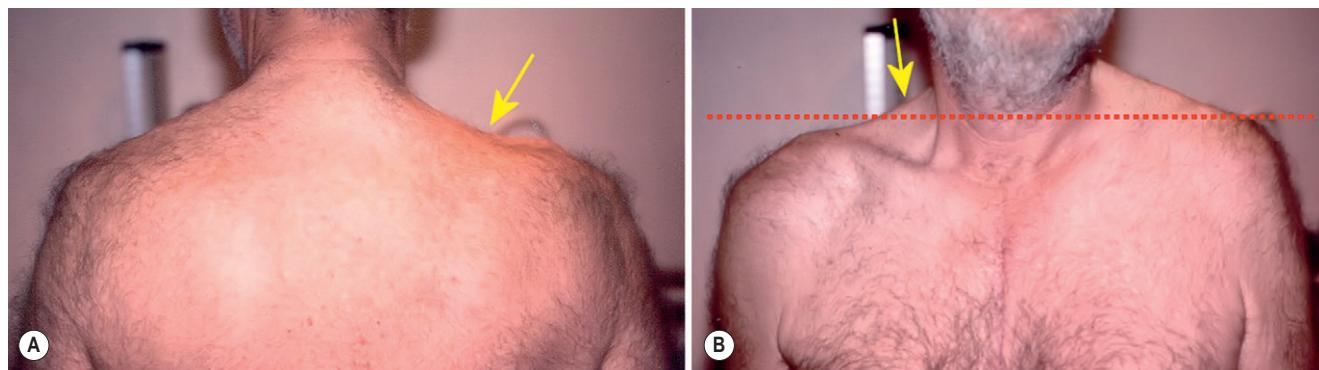


FIGURE 31-12 Distal spinal accessory neuropathy. **A:** Posterior view; **B:** Anterior view. The most common site of injury to the spinal accessory nerve occurs in the region of the posterior cervical triangle, where the nerve runs superficially. A lesion at this site results in shoulder drop (note red dashed line) and atrophy of the trapezius muscle (yellow arrows), but with sparing of the sternocleidomastoid muscle. Note the prominent trapezius atrophy on the patient's right side.

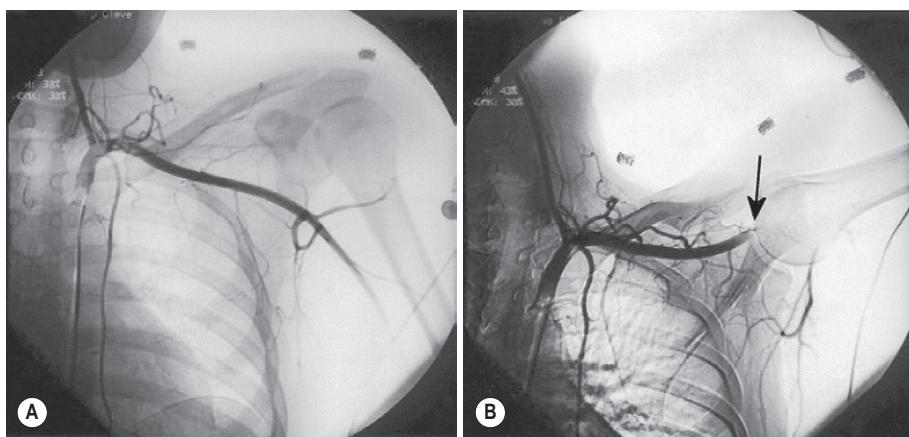


FIGURE 31-13 Neurovascular thoracic outlet syndrome following spinal accessory neuropathy. Shoulder drop from spinal accessory neuropathy may result in traction on the brachial plexus. Rarely, it also may result in frank vascular compression at the thoracic outlet. Axillary artery arteriography in a patient with a spinal accessory neuropathy and shoulder drop, showing normal blood flow with the left arm adducted (A) and complete occlusion of the axillary artery with the arm abducted to 90° (B).

(From Al-Shekhhlee, A., Katirji, B., 2003. Spinal accessory neuropathy, droopy shoulder, and thoracic outlet syndrome. *Muscle Nerve* 28, 383–385. With permission.)

study the sensory nerves that travel through the upper trunk of the brachial plexus. These studies, including the lateral antebrachial cutaneous, radial, and median SNAPs, should be sampled bilaterally to help exclude a more widespread lesion affecting the upper brachial plexus.

Needle EMG can be used to assess the trapezius (upper, middle, and lower fibers) as well as the sternocleidomastoid muscle. One must be cautious in studying the trapezius using EMG. If the trapezius is severely atrophied, it is easy to inadvertently pass through this muscle with the needle and thus actually sample underlying muscles (e.g., supraspinatus, rhomboids). The best way to check that the EMG needle is actually in the trapezius muscle is to have the patient shrug his or her shoulder (trapezius action) and see if MUAPs are activated. If this potential problem is not appreciated, one may mistakenly sample a muscle beneath the trapezius and interpret it as normal, when indeed the trapezius muscle would be very abnormal on EMG, if it had been correctly sampled.

Along with checking the spinal accessory-innervated muscles, needle EMG should be used to sample other proximal muscles, especially those that control the shoulder. Because spinal accessory neuropathies may result in apparent weakness of the shoulder, it is essential to confirm that other shoulder girdle muscles are normal on EMG. At a minimum, the supraspinatus, infraspinatus, deltoid, and rhomboids should be sampled. Lastly, similar to all other proximal neuropathies, the cervical paraspinal muscles should be sampled to help exclude a radiculopathy.



EXAMPLE CASES

Case 31-1

History and Physical Examination

A 33-year-old man was referred for progressive atrophy of the left posterior shoulder. For the past year, he noted deep pain in the region of his left posterior shoulder. This

was followed by slowly progressive wasting over the scapula. The patient frequently lifted weights at the gym, and he was aware that the lifting and external rotation power of his left shoulder was reduced. There was no history of acute pain, sensory loss, or previous episodes of pain or weakness. There was no family history of similar problems.

Examination showed prominent atrophy of the posterior inferior left scapular area. Otherwise, the patient was quite muscular. On muscle strength testing, external rotation of the shoulder was moderately weak. There was only a suggestion of scapular winging. Shoulder abduction was normal, as were all other upper extremity muscles. Reflexes and sensation were intact.

Summary

The history is that of a male weight lifter who has noted the insidious onset of muscle wasting over the left inferior scapula, with reduced ability to externally rotate the shoulder. There is pain in the posterior shoulder, but no neck pain or sensory loss. Neurologic examination is notable for prominent wasting of the left posterior inferior scapular area, with moderate weakness of external rotation of the shoulder, and a suggestion of scapular winging. Otherwise, strength, deep tendon reflexes, and sensation are intact throughout.

Note that the motor and sensory nerve conduction studies were specifically tailored to evaluate the C5–C6 spinal segments because these are the clinically affected segments in this case. The differential diagnosis of shoulder weakness rests between a cervical radiculopathy, a lesion of the upper trunk or lateral or posterior cord of the brachial plexus, and a lesion isolated to one of the nerves that come off the upper trunk or lateral or posterior cord of the brachial plexus.

Therefore, rather than perform routine median and ulnar motor and sensory nerve conduction studies, the motor conduction studies were limited to stimulation of

CASE 31-1. Nerve Conduction Studies											
Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μ V			Latency (ms)			Conduction Velocity (m/s)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL
Suprascapular (m)	Erb's point	Infraspinatus	10.2	0.4		3.2	3.6	\leq 4.2			
Median (s)	Wrist	Thumb	23	24	\geq 10	3.2	3.1	\leq 3.5	52	54	\geq 50
Radial (s)	Forearm	Thumb	24	20	\geq 15	2.3	2.3	\leq 2.9	56	57	\geq 50
Lateral antebrachial (s)	Elbow	Lateral forearm	19	20	\geq 10	2.3	2.4	\leq 3.0	65	63	\geq 55

m = motor study; s = sensory study; RT = right; LT = left; NL = normal.
Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies.

CASE 31-1. Electromyography									
Muscle	Insertional Activity	Spontaneous Activity			Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Duration	Amplitude	Configuration	Polyphasia
Left supraspinatus	↑	+2	0	NL	↓↓	+2	+1	+1	
Left infraspinatus	↑	+3	0	NL	↓↓	+2	+2	+2	
Left medial deltoid	NL	0	0	NL	NL	NL	NL	NL	
Left biceps brachii	NL	0	0	NL	NL	NL	NL	NL	
Left brachioradialis	NL	0	0	NL	NL	NL	NL	NL	
Left C5 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Left C6 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Left C7 paraspinal	NL	0	0	NL	NL	NL	NL	NL	

↑ = increased; ↓↓ = moderately reduced; NL = normal.

the suprascapular nerve at Erb's point bilaterally, with the infraspinatus (the area of muscle wasting) recorded, and sensory conduction studies, with the radial-to-digit 1, median-to-digit-1, and lateral antebrachial cutaneous SNAPs recorded bilaterally. All of these conduction studies evaluate the C5–C6 spinal segments, including the upper trunk and lateral and posterior cords of the brachial plexus. The CMAP amplitude recording the left infraspinatus is markedly reduced, with a normal distal motor latency. The low CMAP amplitude with a normal distal latency implies axonal loss. Note that the median sensory conduction study was performed stimulating the wrist and recording digit 1, although traditionally digit 2 is used as the recording site for median sensory nerve studies. This is because the innervation to digit 1 is derived from the C6 spinal segment, whereas innervation to digit 2 is derived from the C6–C7 segments. The sensory conduction studies are normal and symmetric bilaterally. The normal SNAPs suggest that this is not a lesion of the brachial plexus. On needle EMG examination, abundant fibrillation potentials are noted in the left supraspinatus and infraspinatus, with reduced recruitment of large, long, polyphasic MUAPs. Needle

examination of other muscles in the C5–C6 myotomes, including the medial deltoid, biceps, brachioradialis, and upper cervical paraspinal muscles, is entirely normal. The normal needle examination of other muscles subserved by the C5–C6 myotomes, including the paraspinal muscles, suggests that this is not a cervical radiculopathy.

In summary, the CMAP to the left infraspinatus is markedly reduced, with active denervation and reinnervation restricted to the left supraspinatus and infraspinatus muscles. The remainder of the study is normal. The lesion appears to be restricted to the suprascapular nerve. The presence of a low CMAP amplitude and fibrillation potentials in a patient who has had symptoms over the course of 1 year suggest that the lesion is axonal and severe. The presence of reinnervated MUAPs indicates that the lesion is chronic.

IMPRESSION: There is electrophysiologic evidence consistent with a chronic axonal lesion of the left suprascapular nerve at the suprascapular notch, affecting both the supraspinatus and infraspinatus

This case raises several important questions.

What is the Most Likely Clinical Diagnosis?

The most likely clinical diagnosis is that of suprascapular nerve entrapment at the suprascapular notch, because there was involvement of both the supraspinatus and infraspinatus muscles and the patient experienced deep shoulder pain. The pain likely is secondary to involvement of the deep sensory branches that supply the glenoacromial and acromioclavicular joints. The likely etiology was the chronic repetitive movements of the scapula associated with his weight lifting. The patient subsequently underwent surgery, with exploration of the suprascapular notch. At surgery, the nerve was released from the notch, and the patient experienced subsequent relief of the shoulder pain. Follow-up at 1 year revealed near-complete recovery of muscle bulk and strength of both the supraspinatus and infraspinatus muscles.

Why were the Sensory Nerve Action Potentials Normal?

Note that although normal SNAPs may suggest that a lesion is acute, proximal to the dorsal root ganglion, or secondary to proximal demyelination, in this particular case there is no reported numbness or sensory loss in the areas subserved by the sensory nerves studied. Thus, the normal SNAPs simply suggest that the lesion is outside the distribution of the sensory nerves tested, although they subserve the same spinal segments (C5–C6). Remember that there is no cutaneous sensory nerve associated with the suprascapular nerve.

Case 31–2

History and Physical Examination

A 28-year-old man fell while skiing. He sustained significant trauma to his left neck and shoulder, with a fracture and dislocation of the left mid-humerus. His fracture was reduced and casted for 8 weeks. Upon removal of the cast, he underwent several months of intensive physical therapy but continued to have difficulty with his left

shoulder. Although his daily activities were not affected, his basketball game had not returned to normal. In addition, he was concerned about the lack of muscle bulk around the left shoulder.

On examination, there was marked atrophy of the lateral left shoulder girdle. On muscle testing, there was mild-to-moderate weakness of left shoulder abduction and external rotation. Otherwise, strength was normal, and deep tendon reflexes were intact throughout. On sensory testing, there was a well-circumscribed area of decreased sensation over the left proximal lateral arm.

Summary

The history is that of a young man who fell while skiing, sustaining significant trauma to the left neck, shoulder, and arm, complicated by a fracture and dislocation of the left mid-humerus. Two months later, on removal of the cast, he noticed loss of muscle bulk around the left shoulder and difficulty with his basketball game. Neurologic examination was notable for marked atrophy of the left lateral shoulder girdle, weakness of shoulder abduction and external rotation, and decreased sensation over the left proximal lateral arm. The remainder of the neurologic examination, including strength, deep tendon reflexes, and sensation, was intact.

Note that, as in the previous case, the motor and sensory nerve conduction studies were specifically tailored to evaluate the C5–C6 spinal segments, because these are also the clinically affected segments in this case. The differential diagnosis of weakness of shoulder abduction and external rotation rests between a cervical radiculopathy, a lesion of the upper trunk, lateral cord, or posterior cord of the brachial plexus, and a lesion isolated to one of the nerves that comes off the upper trunk, lateral cord, or posterior cord of the brachial plexus. Therefore, motor conduction studies were limited to stimulation of the axillary nerve bilaterally, with the deltoid (the area of muscle wasting) recorded. Sensory conduction studies were performed recording the radial,

CASE 31–2. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μ V			Latency (ms)			Conduction Velocity (m/s)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL
Axillary (m)	Axilla Erb's point	Deltoid Deltoid	10.2 10.1	0.4 0.3		2.5 4.2	2.8 5.0	\leq 3.3	62	46	\geq 50
Median (s)	Wrist	Thumb	33	34	\geq 10	3.1	3.0	\leq 3.5	53	55	\geq 50
Radial (s)	Forearm	Snuffbox	29	25	\geq 15	2.4	2.4	\leq 2.9	55	56	\geq 50
Lateral antebrachial (s)	Elbow	Lateral forearm	17	19	\geq 10	2.2	2.3	\leq 3.0	64	62	\geq 55

m = motor study; s = sensory study; RT = right; LT = left; NL = normal.

Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies.

CASE 31-2. Electromyography									
Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials					Configuration
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Duration	Amplitude	Polyphasia	
Left medial deltoid	↑	+2	0	NL	↓↓	+2	+1	+1	
Left teres minor	↑	+3	0	NL	↓↓	+2	+2	+2	
Left Infraspinatus	NL	0	0	NL	NL	NL	NL	NL	
Left biceps brachii	NL	0	0	NL	NL	NL	NL	NL	
Left brachioradialis	NL	0	0	NL	NL	NL	NL	NL	
Left triceps	NL	0	0	NL	NL	NL	NL	NL	
Left C5 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Left C6 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Left C7 paraspinal	NL	0	0	NL	NL	NL	NL	NL	

↑ = increased; ↓↓ = moderately reduced; NL = normal.

median-to-digit-1, and lateral antebrachial cutaneous SNAPs bilaterally, all of which evaluate the C5–C6 spinal segments, including the upper trunk and lateral and posterior cords of the brachial plexus. The CMAP amplitude recording the left deltoid is markedly reduced, with a normal distal motor latency. The low CMAP amplitude with normal distal latency implies axonal loss. Note that, as in the previous case, the median sensory nerve study was performed stimulating the wrist and recording digit 1, as innervation to digit 1 is derived from the C6 spinal segment, whereas sensation to digit 2, which is traditionally used, is derived from the C6–C7 segments and is not as relevant to this case. The sensory studies are normal and symmetric bilaterally. The normal SNAPs imply that this is not a lesion of the brachial plexus. On needle EMG examination, abundant fibrillation potentials are noted in the left deltoid and teres minor, with reduced recruitment of large, long, polyphasic MUAPs. Needle examination of other muscles with C5 or C6 innervation, or both, including the infraspinatus, biceps, brachioradialis, triceps, and upper cervical paraspinal muscles, is entirely normal. The normal examination of other muscles, including the paraspinal muscles, in the same myotomes as the deltoid and teres minor suggests that this is not a cervical radiculopathy.

In summary, there is a low CMAP recording the left deltoid, with active denervation and reinnervation restricted to the left deltoid and teres minor muscles. Needle examination of other muscles in the same myotomes is normal, including the cervical paraspinal muscles, and all of the SNAPs are normal. The lesion appears to be restricted to the axillary nerve. The presence of a low CMAP and fibrillation potentials in a patient who has had symptoms for several months suggests that the lesion is axonal and severe. The presence of reinnervated MUAPs indicates that the lesion is chronic.

IMPRESSION: There is electrophysiologic evidence consistent with a chronic axonal lesion of the left axillary nerve.

This case raises several important questions.

What is the Most Likely Etiology of this Patient's Injury?

The most likely etiology of the patient's injury is that of an axillary nerve injury secondary to fracture and dislocation of the mid-humerus. Axillary neuropathies most often occur as a result of trauma, especially dislocation of the shoulder and fracture of the humerus.

Can One be Absolutely Certain that there is not a Cervical Radiculopathy or Lesion of the Brachial Plexus?

The electrodiagnostic abnormalities are limited to the deltoid and teres minor. Although one cannot say with absolute certainty that the lesion is restricted to the axillary nerve, given the clinical history of a mid-humeral fracture and the findings of weakness of shoulder abduction and external rotation and sensory loss over the lateral arm, this is the most likely diagnosis. The possibility of a lesion of the brachial plexus or cervical roots primarily affecting the axillary fibers may be considered, but the clinical context and the electrophysiologic findings make this very unlikely.

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