30 Brachial Plexopathy

The brachial plexus is a complicated anatomic structure formed by the ventral rami of the lower cervical and upper thoracic nerve roots. Different fascicles from those roots intermix widely within the plexus to ultimately form all the nerves of the upper extremity (Figure 30–1). In cases of suspected brachial plexopathy, nerve conduction studies and electromyography (EMG) often are used to localize the lesion accurately and to assess its severity. Notwithstanding its usefulness, the electrophysiologic evaluation of brachial plexopathy is demanding for the electromyographer. Detailed knowledge of the anatomy of the upper extremity roots, plexus, and peripheral nerves is required. Extensive bilateral studies, with emphasis on the sensory conduction studies and needle EMG, frequently are needed to localize the lesion. Proper localization is key, not only to exclude a disorder of the nerve roots, which may closely resemble brachial plexopathy clinically, but also to suggest possible etiologies, as certain disorders preferentially affect different parts of the brachial plexus. In addition, assessing the severity is important, especially in cases of trauma, where the results often help decide whether surgery should be considered.

ANATOMY

The brachial plexus is located between the lower neck and axilla, running behind the scalene muscles proximally and behind the bony clavicle and the pectoral muscles distally. The plexus is divided anatomically into *roots*,

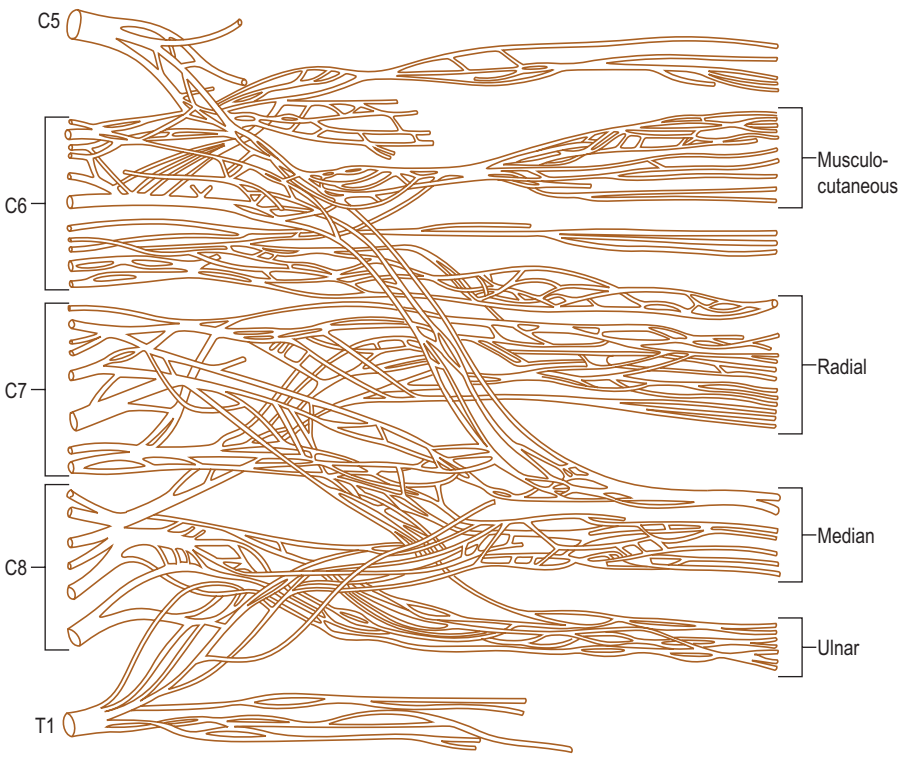

FIGURE 30–1 Microdissection of brachial plexus anatomy. The brachial plexus is a complicated anatomic structure, with nerve fibers from the lower cervical and upper thoracic roots intermixing widely to ultimately form the peripheral nerves. (From Kerr, A.T., 1918. Am J Anat 23, 285, with permission.)

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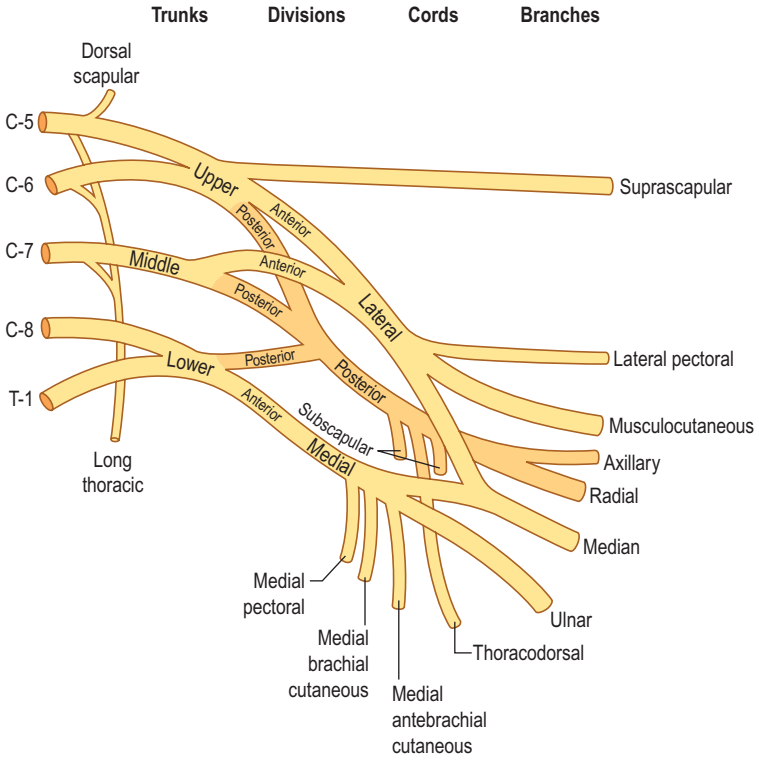

FIGURE 30–2 Brachial plexus anatomy. The brachial plexus is divided into roots, trunks, divisions, cords, and finally nerves.
(From Hollinshead, W.H., 1969. Anatomy for surgeons, volume 2: the back and limbs. Harper & Row, New York, with permission.)

trunks, *divisions*, *cords*, and finally *nerves* (Figure 30–2), although, strictly speaking, the roots and peripheral nerves are not considered part of the plexus proper. Two important nerves, the long thoracic and dorsal scapular, originate directly from the roots, proximal to the brachial plexus. The *long thoracic nerve* comes off the C5–C6–C7 roots, innervating only the serratus anterior muscle. The *dorsal scapular nerve* is formed primarily from the C5 root and less so from the C4 root, innervating the rhomboid muscles. After the take-off of these two nerves, the anterior rami of the C5–T1 nerve roots come together above the level of the clavicle to form the three trunks of the brachial plexus. The *upper trunk* is formed from the C5–C6 roots. The C7 root continues as the *middle trunk*, and the *lower trunk* is formed from the C8–T1 roots.

Each trunk then divides into an *anterior* and *posterior division*. From these six divisions, the cords are formed, located below the level of the clavicle. The three posterior divisions unite to form the *posterior cord*. The anterior divisions of the upper and middle trunks join to form the *lateral cord*. This leaves the anterior division of the lower trunk to continue as the *medial cord*.

All major nerves in the upper extremity originate either from the cords and trunks of the brachial plexus or, less commonly, directly from the roots (Table 30–1). Although the brachial plexus is generally formed from the C5–T1 nerve roots, anomalies are not infrequent. For example, in some individuals the brachial plexus is formed predominantly from the C4–C7 roots and is said to be *prefixed*. In others the plexus is *postfixed*, receiving most of its innervation from the C6–T2 roots.

Table 30–1. Innervation of Major Upper Extremity Nerves

Nerve	Innervation
Dorsal scapular	C4–C5 roots directly
Long thoracic	C5–C6–C7 roots directly
Suprascapular	Upper trunk
Radial	Posterior cord
Axillary	Posterior cord
Thoracodorsal	Posterior cord
Musculocutaneous	Lateral cord
Median	Lateral and medial cords
Ulnar	Medial cord
Medial antebrachial cutaneous	Medial cord
Medial brachial cutaneous	Medial cord

CLINICAL

Because the upper extremity receives its entire motor and sensory innervation from the brachial plexus, brachial plexopathies may present with a variety of clinical patterns,

depending on the part of the plexus affected. These are the same important patterns that form the basis of localization on nerve conduction studies and needle EMG as well.

Panplexus

A complete brachial plexopathy results in weakness, sensory loss, and decreased or absent reflexes in the entire arm. Provided the roots remain intact, the serratus anterior and rhomboids usually are the only muscles spared because they are innervated by nerves that come directly off the roots, proximal to the plexus. The assessment of these two muscles is key, both clinically and electrically, in differentiating a severe lesion at the level of the plexus from one originating at the roots.

Upper Trunk Plexopathy

The upper trunk is formed from the C5–C6 roots. Thus, upper trunk lesions result in weakness of nearly all muscles with C5–C6 innervation. Most affected are the deltoid, biceps, brachioradialis, supraspinatus and infraspinatus muscles. Muscles that receive partial upper trunk innervation, such as the pronator teres (C6–C7) and triceps (C6–C7–C8), may be partially affected. Sensory loss involves the lateral arm, lateral forearm, lateral hand, and thumb. This territory corresponds to the sensory distributions of the axillary and lateral antebrachial cutaneous nerves, as well as the median and radial sensory branches to the thumb and index finger (Figure 30–3). The biceps and brachioradialis tendon jerks are depressed or absent, but the triceps reflex is spared.

Middle Trunk Plexopathy

Middle trunk lesions are very rare. Because the middle trunk is formed directly from the C7 root, middle trunk lesions mimic C7 radiculopathies. Weakness involves primarily the triceps, flexor carpi radialis, and pronator teres muscles. Sensory abnormalities predominantly affect the middle finger, and less so the index and ring fingers (sensory branches of the median nerve) and posterior forearm (posterior cutaneous nerve of the forearm). Only the triceps reflex is abnormal on reflex testing.

Lower Trunk Plexopathy

The lower trunk is formed from the C8–T1 roots. The entire ulnar nerve, the medial brachial cutaneous nerve, and the medial antebrachial cutaneous nerve are ultimately supplied from fibers passing through the lower trunk. In addition, both the median and radial nerves receive partial motor innervation from the lower trunk. Accordingly, lower trunk lesions involve all ulnar muscles, in addition to median C8–T1-innervated muscles (e.g., abductor pollicis brevis [APB], flexor pollicis longus, flexor digitorum profundus) and radial C8-innervated muscles (e.g., extensor indicis proprius [EIP], extensor pollicis brevis). Sensory loss involves the medial arm, medial forearm, medial hand, and fourth and fifth fingers. This territory corresponds to the distribution of the medial brachial cutaneous, medial antebrachial cutaneous, ulnar sensory, and dorsal ulnar cutaneous sensory nerves (Figure 30–4). In pure lower trunk plexopathies, there are no reflex abnormalities.

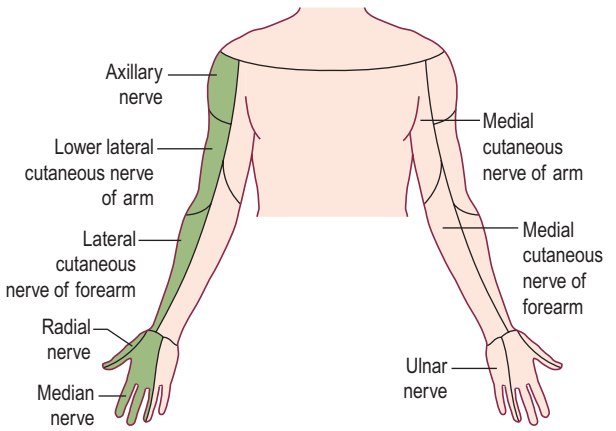

FIGURE 30–3 Upper trunk brachial plexopathy sensory loss. This territory corresponds to the sensory distributions of the axillary nerve, lower lateral cutaneous nerve of the arm (a.k.a. the lateral brachial cutaneous nerve), and lateral cutaneous nerve of the forearm (a.k.a. the lateral antebrachial cutaneous nerve), as well as the median and radial sensory branches to the index finger and thumb.
(Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

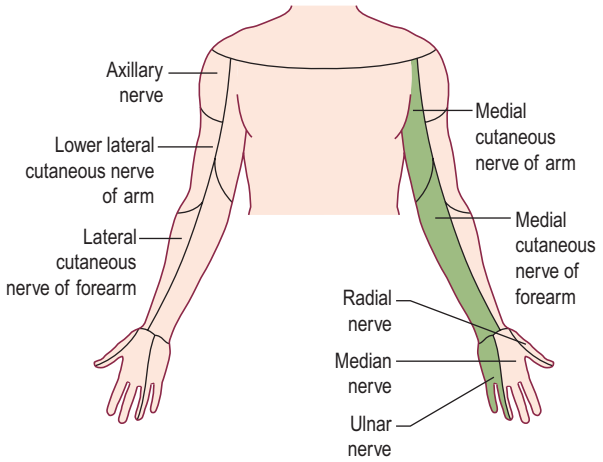

FIGURE 30–4 Lower trunk brachial plexopathy sensory loss. This territory corresponds to the distribution of the medial cutaneous nerve of the arm (a.k.a. the medial brachial cutaneous nerve), medial cutaneous nerve of the forearm (a.k.a. the medial antebrachial cutaneous nerve), ulnar sensory, and dorsal ulnar cutaneous sensory nerves.
(Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

Lateral Cord Plexopathy

The entire musculocutaneous nerve and the C6–C7 portion of the median nerve are derived from the lateral cord. Accordingly, lateral cord lesions result in median weakness of arm pronation (pronator teres) and wrist flexion (flexor carpi radialis) and musculocutaneous weakness of elbow flexion (biceps). Sensory loss involves the lateral forearm, lateral hand, and first three fingers. This territory corresponds to the distribution of the lateral antebrachial cutaneous and median sensory nerves. On reflex testing, the biceps reflex is abnormal, but the triceps and brachioradialis reflexes are preserved.

Posterior Cord Plexopathy

The radial, axillary, and thoracodorsal nerves are derived from the posterior cord. Accordingly, posterior cord lesions result in complete radial palsies (wristdrop and fingerdrop, arm extension weakness) in addition to weakness of shoulder abduction (deltoid) and adduction (latissimus dorsi). Sensory loss involves the lateral arm, posterior arm and forearm, and radial dorsal hand. This territory corresponds to the sensory distribution of the radial (superficial radial, posterior cutaneous nerve of the forearm) and axillary nerves. On reflex testing, the triceps and brachioradialis reflexes are abnormal.

Medial Cord Plexopathy

The medial cord is the direct continuation of the anterior division of the lower trunk. Thus, medial cord lesions are nearly identical to lower trunk plexopathies, except for intact radial C8 fibers, which pass through the posterior division of the lower trunk and then through the posterior cord. Medial cord lesions result in weakness of all ulnar muscles and C8–T1 median muscles (APB, flexor pollicis longus, flexor digitorum profundus – median). Notably, finger extensors, especially to the index finger (radial innervated), are spared. Sensory loss is identical to that seen in lower trunk lesions, involving the medial arm, medial forearm, medial hand, and fourth and fifth fingers.

ETIOLOGY

Traumatic Brachial Plexopathy

Traumatic injuries are the most common cause of brachial plexopathies. Most frequently, traumatic brachial plexopathies are the result of automobile, motorcycle, or bicycle accidents. Penetrating knife or gunshot wounds may injure the brachial plexus. Traumatic brachial plexopathies may occur in newborns, usually as a result of traction during delivery.

Most traumatic plexopathies are the result of traction and stretch injuries. Injuries in which the head is pushed away from the shoulder (e.g., the head and shoulder striking the pavement when a person is thrown from a moving vehicle) typically result in upper plexopathies, affecting the C5–C6 fibers (Figure 30–5). Such injuries result in characteristic weakness of shoulder abduction, elbow flexion, and arm supination, known as *Erb's palsy*. This is also the most common type of brachial plexopathy seen in newborns, presumably as a result of the head being delivered down, away from the shoulder. The most common risk factor for an Erb's palsy in a newborn is shoulder dystocia in a large infant. In contrast, injuries in which the arm and shoulder are pulled up typically result in lower plexopathies, affecting the C8–T1 fibers. Severe hand weakness, known as *Klumpke's palsy*, characteristically occurs in these latter injuries, with preservation of upper arm and shoulder girdle muscles. One of the most common scenarios in which this occurs is when an individual (often unconscious) is dragged by one arm.

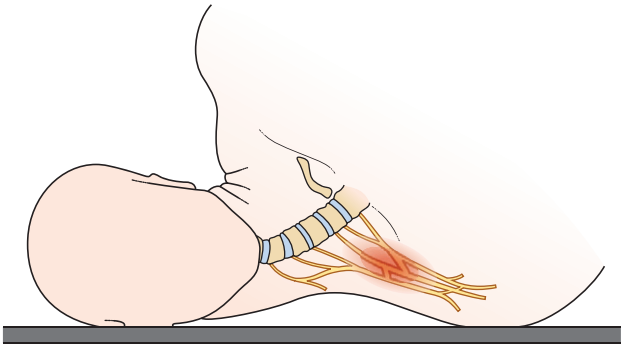

FIGURE 30–5 Traumatic upper trunk plexopathies. Most traumatic plexopathies are the result of traction and stretch injuries. Although the entire brachial plexus can be injured, the upper trunk is most commonly injured when the head is pushed away from the shoulder, as might occur when the head and shoulder strike the pavement after a person is thrown from a moving vehicle.

It is important to understand that severe traction injuries may result in damage to the roots as well as the plexus. A traction injury can cause frank root avulsion, in which the roots are physically separated from the spinal cord. This is the most serious type of injury, with no chance for recovery. Nerve conduction studies and needle EMG are useful in differentiating root avulsion from plexus lesions, or lesions that involve both the roots and the plexus.

Neoplasms and Other Mass Lesions

Brachial plexopathy may result from local tumor invasion. For example, Pancoast tumors of the lung may spread and invade the plexus directly. More commonly, tumors metastasize to nearby lymph nodes, where they grow and compress the plexus. Lymphomas, breast cancer, and lung cancer are the most frequent causes. Lymphomas and leukemia can also infiltrate nerve directly, in the absence of a mass lesion. Rarely, primary nerve sheath tumors (e.g., schwannomas, neurofibromas, or neurofibrosarcomas) may affect the brachial plexus. In unusual cases, non-neoplastic mass lesions, such as hematomas and unusual vascular anomalies (e.g., aneurysm, arteriovenous malformation), can compress the brachial plexus.

Characteristically, neoplastic brachial plexopathy results in a slowly progressive syndrome often associated with prominent pain. In some cases, it may be difficult or impossible to distinguish these lesions clinically from more proximal lesions of the cervical nerve roots. Nerve conduction studies and needle EMG often are very useful in distinguishing brachial plexus from cervical root lesions in these cases.

Neuralgic Amyotrophy (Brachial Plexitis)

Neuralgic amyotrophy is a common although underappreciated disorder that frequently affects individual upper extremity nerves or the brachial plexus. The condition is known by various names, including *Parsonage–Turner syndrome*, *brachial plexitis*, *idiopathic brachial plexopathy*, *and brachial amyotrophy*. In many but not all cases, the syndrome is preceded by an antecedent event that triggers the immune system, usually a viral illness or immunization or, occasionally, surgery. The onset of shoulder pain typically follows within several days to a few weeks. The pain is severe, often awakening the patient from sleep. Early on, muscle weakness may be difficult to detect on examination because of the prominent pain. However, as the pain subsides, typically after 1 to 2 weeks, significant underlying weakness becomes apparent. Muscle atrophy follows. Although paresthesias and sensory loss may also be present, it is not unusual to find only mild or minimal sensory abnormalities on examination.

In some cases, all or part of the brachial plexus may be affected. In others, individual upper extremity nerves, including nerves that come directly off the roots, may be affected in isolation, in a pattern that more resembles a mononeuropathy multiplex. Certain nerves, especially the long thoracic and anterior interosseous nerves, are frequently involved in neuralgic amyotrophy. A long thoracic nerve palsy results in characteristic winging of the scapula, due to weakness of the serratus anterior muscle. An anterior interosseous nerve palsy is recognized principally by weakness of the long flexors of the thumb and index finger (flexor pollicis longus and flexor digitorum profundus – median): the patient is unable to make an "OK" sign. In some cases, involvement of the phrenic nerve has been reported, either in isolation or in conjunction with other mononeuropathies. Exceptionally, lower cranial neuropathies (IX–XII) have accompanied otherwise classic presentations of neuralgic amyotrophy.

Most episodes of neuralgic amyotrophy are primarily unilateral. On close examination, however, especially with needle EMG, some abnormalities on the contralateral side are not unusual. Likewise, most cases are a one-time event. Recurrent episodes can occur but are distinctly uncommon. Recurrent episodes of painful brachial neuritis should raise the possibility of hereditary neuralgic amyotrophy, a rare, dominantly inherited disorder associated with mutations in the *SEPT9* (septin-9) gene on chromosome 17q25 that has a similar clinical presentation to the idiopathic cases. Minor dysmorphic features may be present on physical examination of these patients (i.e., hypotelorism, short stature, cleft palate, epicanthal folds, ring-shaped skin creases on limbs and neck, partial syndactyly).

Postoperative Brachial Plexopathy

Brachial plexopathy is the most common peripheral nervous system complication occurring after coronary artery bypass and other similar chest surgery. These lesions are thought to result from stretch injury following chest wall retraction or occur secondary to compression from hematomas associated with internal jugular catheters. Nearly all involve principally the lower trunk or medial cord of the plexus.

In lesions of the lower trunk, patients note sensory disturbance in the fourth and fifth fingers (ulnar distribution), which may continue up the medial forearm and arm (medial brachial and medial antebrachial cutaneous nerves). Weakness involves all C8–T1 muscles, including median and ulnar hand intrinsics, all forearm long finger flexors (Figure 30–6), and, less so, the finger extensors (principally the extensors to the thumb and index finger). In some cases, pain may be a prominent symptom. Because the presumed injury is secondary to stretch and compression, without any tearing or shearing of nerve and basement membrane, most patients make a good recovery over several months. Rarely, patients may not recover completely; occasionally, patients are left with chronic pain that is difficult to treat.

Delayed Radiation Injury

Radiation may result in a progressive brachial plexopathy, typically presenting years after the radiation exposure. Radiation ports often include the region of the brachial plexus, especially in the treatment of lymphomas and breast, lung, and neck cancers. The risk of radiation-induced plexopathy increases with the dose of radiation; it is more common after doses of more than 5700 rads.

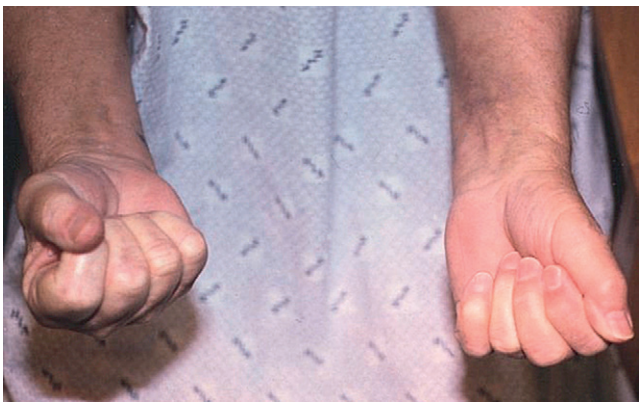

FIGURE 30–6 Postoperative brachial plexopathy. Postoperative brachial plexopathies characteristically affect C8–T1 fibers that travel through the lower plexus. Weakness of intrinsic hand muscles and the long finger flexors results. In the patient shown here, weakness of the long finger flexors on the left is recognized as the inability to make a grip and fully flex the fingers and thumb at the distal interphalangeal joints.

When a patient with a prior history of malignancy who has been treated with radiation develops a slowly progressive brachial plexus lesion, the differential diagnosis usually rests between radiation-induced brachial plexopathy and direct invasion from recurrent tumor. Several clinical and electrophysiologic findings may be of help in distinguishing between the two. First, pain is an earlier and more prominent finding in direct neoplastic invasion. Likewise, the presence of a Horner's syndrome is much more common in direct neoplastic invasion. In contrast, sensory symptoms (i.e., paresthesias and numbness) appear more commonly and earlier in cases of radiation damage. In addition, patients with radiation-induced plexopathy usually are symptomatic for a much longer time, often many years, before coming to medical attention.

On electrophysiologic testing, the presence of myokymic discharges and fasciculations is especially helpful in differentiating radiation-induced from neoplastic plexopathy. Myokymic discharges are characteristic of radiation-induced brachial plexopathy. They may be seen clinically but are more often appreciated on needle EMG. Although conduction block across the brachial plexus has been described in patients with radiation plexitis, it is a nonspecific finding that has also been reported, although less frequently, in plexopathy associated with neoplasm. Other findings on nerve conduction studies and EMG, including the region of the plexus involved, and the presence of clinical weakness are generally not helpful in differentiating radiation-induced from direct neoplastic brachial plexopathy.

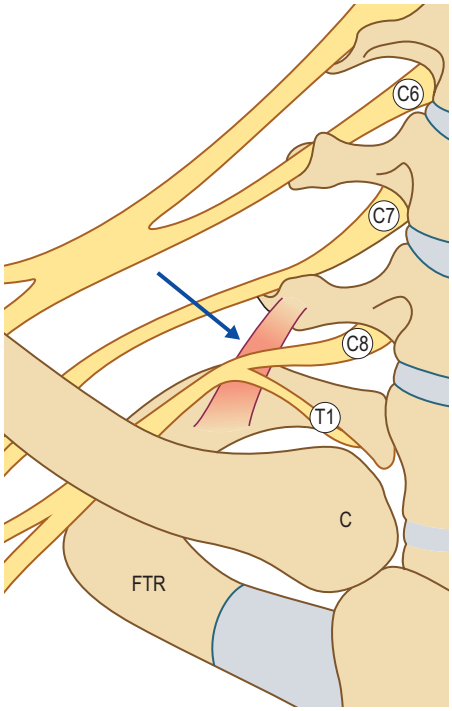

FIGURE 30–7 Neurogenic thoracic outlet syndrome anatomy. Most cases of true neurogenic thoracic outlet syndrome are caused by a fibrous band that runs from a rudimentary cervical rib to the first thoracic rib (arrow), entrapping the lower trunk of the brachial plexus. C, clavicle; FTR, first thoracic rib.
(Adapted from Levin, K.H., Wilbourn, A.J., Maggiano, H.J., 1998. Cervical rib and median sternotomy-related brachial plexopathies: a reassessment. Neurology 50, 1407–1413, with permission.)

Thoracic Outlet Syndrome

The term *thoracic outlet* refers to the exit of the brachial plexus and the major arteries and veins from the shoulder and axilla into the arm. Several types of thoracic outlet syndrome (TOS) occur, depending on which structure is entrapped. Impingement of the subclavian and axillary vessels may result in vascular TOS. Entrapment of the brachial plexus itself results in true neurogenic TOS.

In the past, the diagnosis of neurogenic TOS was made frequently, and many patients underwent surgical procedures to decompress the thoracic outlet. These procedures included removal of cervical ribs, first rib resections, lysis of fibrous bands, as well as sectioning of some of the scalene muscles. However, impingement of the cervical nerve roots at the intervertebral foramina and the common entrapment neuropathies in the arm were not well appreciated at that time. It has since become apparent that true neurogenic TOS is quite rare. Most patients diagnosed with TOS in the past actually had either a cervical radiculopathy or an entrapment of either the ulnar nerve at the elbow or the median nerve at the wrist.

Most cases of true neurogenic TOS are caused by a fibrous band that runs from a rudimentary cervical rib to the first thoracic rib, entrapping the lower trunk of the brachial plexus (Figure 30–7). Accordingly, sensory and motor loss develops in the C8–T1 distribution. Anatomically, the fibrous band most often preferentially affects the T1 fibers. This results in a characteristic pattern of signs and symptoms, including prominent wasting and weakness of the thenar and, less prominently, the hypothenar muscles (Figure 30–8). The explanation for the relative vulnerability of the thenar muscles is not completely clear, but it may be that the thenar muscles are more T1 innervated, whereas the hypothenar muscles receive more C8 innervation.

In addition to the median and ulnar intrinsic hand muscles, the long flexors to the fingers (i.e., flexor digitorum profundus) and thumb (flexor pollicis longus) also are C8–T1 innervated and may be affected. Radial C8 weakness (e.g., EIP) can occur but is less common. Paresthesias and sensory loss affect the fourth and fifth fingers, medial hand, and medial forearm. These sensory changes are in the distribution of the ulnar and the medial antebrachial cutaneous sensory nerves, both of which pass through the lower trunk of the brachial plexus.

Neurogenic TOS is most often confused clinically with the more common ulnar neuropathy at the elbow or C8–T1 radiculopathy. Several pieces of clinical information are helpful in differentiating among these conditions. A history of neck pain with radiation down the arm, provoked by neck movement, strongly favors the diagnosis of radiculopathy. Local tenderness and pain around the elbow commonly accompany ulnar neuropathy at the elbow. In all three

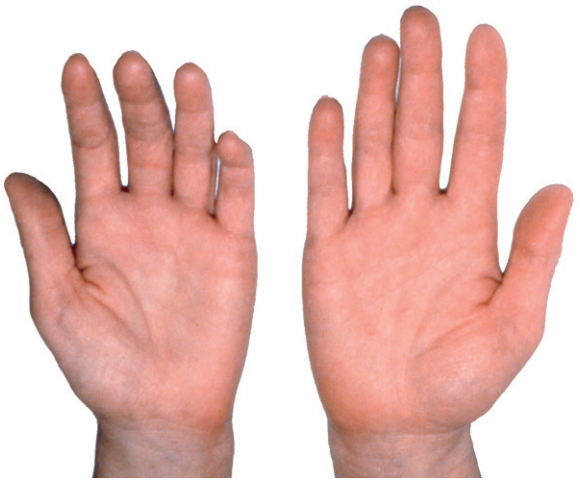

FIGURE 30–8 Hand atrophy and posture in a patient with neurogenic thoracic outlet syndrome. Neurogenic thoracic outlet syndrome preferentially affects the T1 fibers. This results in a characteristic pattern of wasting and weakness of the thenar and, less prominently, the hypothenar muscles (left hand). The explanation for the relative vulnerability of the thenar muscles is likely that they are more T1 innervated, whereas the hypothenar muscles receive more C8 innervation.

conditions, atrophy and weakness may affect both the thenar and hypothenar muscles. With ulnar neuropathy at the elbow, however, thumb abduction will be spared (median innervated). In neurogenic TOS, thumb abduction not only is involved but is often preferentially affected. In a C8–T1 radiculopathy, thumb abduction may be weak but is not out of proportion to weakness of the other C8–T1-innervated muscles. On sensory testing, abnormalities are restricted to the fifth and medial fourth fingers and medial hand in ulnar neuropathy at the elbow. In both neurogenic TOS and C8–T1 radiculopathy, sensory disturbance extends more proximally into the medial forearm, in the distribution of the medial antebrachial cutaneous sensory nerve.

ELECTROPHYSIOLOGIC EVALUATION

The principal goals of the electrophysiologic study in suspected brachial plexopathy are to localize the lesion accurately and to assess its severity. In addition, every study should exclude the possibility of radiculopathy or multiple peripheral nerve lesions mimicking a brachial plexus lesion. Before embarking on the electrophysiologic study, a firm understanding of normal brachial plexus anatomy is essential. Likewise, the electromyographer should have a good idea from the clinical examination where in the brachial plexus the pathology is likely to be.

The electrophysiologic evaluation of brachial plexopathy relies primarily on the sensory nerve action potentials (SNAPs) and a detailed needle EMG examination. Motor nerve conduction studies, although helpful in some cases, are generally not useful in differentiating between a plexopathy and radiculopathy.

Box 30–1. Recommended Nerve Conduction Study Protocol for Brachial Plexopathy

Routine sensory conduction studies:

1. Sensory potentials: lateral antebrachial cutaneous, radial, median, ulnar and medial antebrachial cutaneous (see Table 30–3)
2. Compare with the unaffected side, especially if potentials are low or near the lower limit of normal

Routine motor conduction studies:

1. Routine median motor study, recording abductor pollicis brevis, stimulating wrist and antecubital fossa
2. Routine ulnar motor study, recording abductor digiti minimi, stimulating wrist and below and above elbow

Special considerations:

- In suspected lower trunk/medial cord lesions, routine median and ulnar motor conduction studies can be performed, but also stimulating at the axilla and Erb's point. For proximal median motor conduction studies to be performed properly, collision studies are required to eliminate the confounding effect of co-stimulating the ulnar nerve.
- Comparison of the motor studies to the contralateral side may be helpful.
- In suspected posterior cord lesions, radial motor conduction studies should be performed to exclude radial neuropathy at the spiral groove.
- Stimulating Erb's point, recording the biceps, triceps, deltoid, or spinati can be performed bilaterally in suspected upper or middle trunk lesions, to assess the amount of axonal loss.

F responses:

1. Bilateral median and ulnar F responses, especially in suspected lower trunk or medial cord lesions.

Nerve Conduction Studies

Sensory nerve conduction studies play a central role in the assessment of possible brachial plexopathy (Box 30–1). All sensory nerve fibers in the plexus lie distal to the dorsal root ganglion. Thus, brachial plexus lesions often result in abnormal SNAPs, which are one of the most useful pieces of information to help differentiate plexus from root lesions. In the arm, the lateral antebrachial cutaneous, radial, median, ulnar, and medial antebrachial cutaneous sensory conduction studies are all easily performed. In brachial plexopathy, one or more of these SNAPs usually are abnormal, depending on the location and etiology of the lesion (Table 30–2). In some cases, an abnormal SNAP may be discovered only by comparing it with the contralateral side. Bilateral studies are most helpful when the SNAP amplitude on the symptomatic side is at or just below the lower limit of normal. In some cases, a SNAP may have an amplitude in the normal range, but side-to-side comparison reveals a clear asymmetry. In general, there must be a 50% difference in amplitude from side to side for a study to be considered abnormal (i.e., the abnormal side has a 50% or lower amplitude than the normal side).

Motor studies are less useful in the assessment of brachial plexopathy. Their usefulness lies primarily in excluding multiple entrapment neuropathies that can mimic a

Table 30–2. Sensory Potentials to Check in Brachial Plexopathy

SNAP	Cord	Trunk
Lateral antebrachial cutaneous	Lateral	Upper
Radial to the thumb	Posterior	Upper
Median to the thumb	Lateral	Upper
Radial to the snuffbox	Posterior	Upper/middle
Median to the index finger	Lateral	Upper/middle
Median to the middle finger	Lateral	Middle
Median to the ring finger	Medial	Middle/lower
Ulnar to the ring finger	Medial	Lower
Ulnar to the little finger	Medial	Lower
Dorsal ulnar cutaneous	Medial	Lower
Medial antebrachial cutaneous	Medial	Lower

SNAP, sensory nerve action potential.

Note: SNAPs are abnormal in lesions at or distal to the dorsal root ganglion including plexopathies. In the evaluation of a possible brachial plexopathy, the pattern of the abnormal SNAPs helps to localize the lesion.

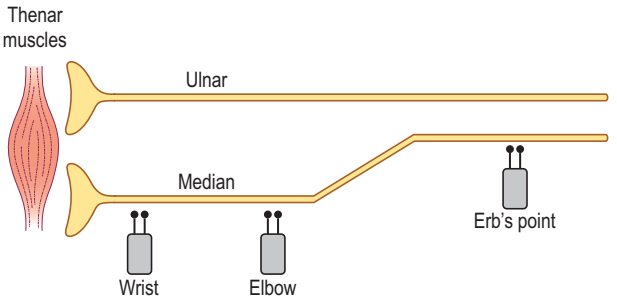

FIGURE 30–9 Co-stimulation of adjacent nerves at Erb's point. During routine median and ulnar motor conduction studies, co-stimulation occurs at the wrist and elbow sites only if an excessive stimulus is used. In contrast, co-stimulation of ulnar and median fibers occurs routinely at proximal stimulation sites (e.g., axilla and Erb's point). During ulnar motor conduction studies recording the abductor digiti minimi, co-stimulation is not a major problem because there are no median-innervated muscles in the hypothenar eminence. In contrast, during median motor conduction studies recording the thenar muscles, co-stimulation results in a median compound motor action potential contaminated by ulnar motor fibers in the thenar eminence.

brachial plexus lesion. The routine median, ulnar, and radial motor studies all record from distal C8- or C8–T1-innervated muscles. Accordingly, routine median and ulnar motor studies are useful only in assessing medial cord or lower trunk lesions. Likewise, radial motor studies are useful only in assessing posterior cord or lower trunk lesions. Lesions of the lateral cord or of the upper or middle trunks do not result in any abnormalities on routine motor studies.

If a brachial plexus lesion associated with axonal loss affects the lower trunk or medial cord, median and ulnar compound muscle action potentials (CMAPs) may have reduced motor amplitudes, with mild slowing of distal latency and conduction velocity. Median and ulnar F responses may be prolonged, especially when compared with the asymptomatic side. Radial motor nerve conduction studies may show similar findings in a lower trunk or posterior cord lesion.

Conduction studies can be performed across the brachial plexus but should be approached with caution. Most brachial plexopathies are primarily axonal loss lesions. Hence, no focal slowing or conduction block will be seen across the lesion in most cases. Conduction block and focal slowing typically are seen only in some cases of radiation plexitis and inflammatory demyelinating polyneuropathy. Motor conduction studies across the plexus require stimulation at the axilla and at Erb's point. In some individuals, it may be difficult or impossible to obtain supramaximal stimulation even with maximum machine output at proximal sites, especially at Erb's point. Submaximal stimulation, if not recognized, may give the mistaken impression of a conduction block.

The other major problem with proximal stimulation is co-stimulation of adjacent nerves (Figure 30–9). Co-stimulation often occurs at the axilla and always occurs at Erb's point. When routine ulnar motor conduction studies are performed, stimulating at the axilla or at Erb's point, this results in depolarization of both ulnar and median C8–T1-innervated muscles. This is not a major problem when the abductor digiti minimi (ADM) is used for recording because there are no median-innervated muscles in the hypothenar eminence that could potentially contaminate the ADM CMAP. However, when routine median motor nerve conduction studies are performed recording the APB, co-stimulation at the axilla or Erb's point is problematic because both median- and ulnar-innervated muscles are present in the thenar eminence, where the recording electrodes are placed. With co-stimulation, the median CMAP will be contaminated by the contribution from ulnar fibers, making the amplitude larger and perhaps also affecting the distal latency.

The problem of proximal co-stimulation during median motor studies can be eliminated only by the use of a collision study (Figure 30–10). The basic idea of a collision study is to collide out the ulnar fiber contribution from proximal stimulation by also stimulating the ulnar fibers distally. Collision studies require two separate stimulators that can be set to give their individual shocks at different times. The first stimulator is placed over the ulnar nerve at the wrist and the second over the proximal site (axilla or Erb's point). Recording electrodes are placed on the median-innervated APB as usual. The stimulators are individually set to give a supramaximal shock over the ulnar nerve at the wrist and at the proximal site. By subtracting the distal latency from the proximal latency, one can then calculate the amount of time in milliseconds it takes for the depolarization to travel from the proximal to the distal

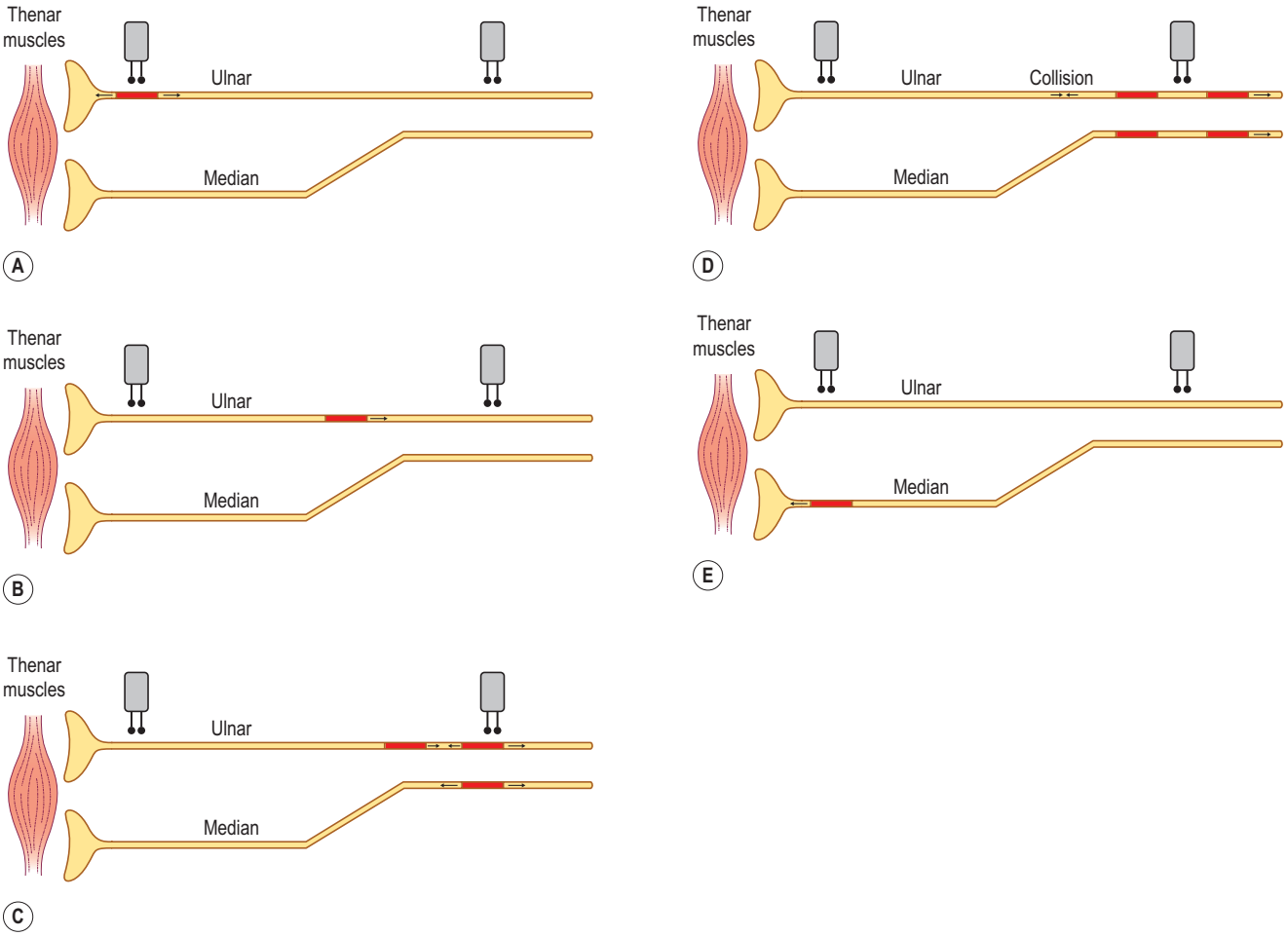

FIGURE 30–10 Collision study. Stimulation at Erb's point activates both median and ulnar nerve fibers. During median nerve conduction studies, co-stimulation of ulnar fibers can be eliminated with collision studies. Collision studies require two stimulators that are set to give their individual shocks at different times. The first stimulator is placed over the ulnar nerve at the wrist and the second over Erb's point. Individually, the stimulators are set to give a supramaximal shock to the ulnar nerve at the wrist and at Erb's point, respectively. A collision study then is performed, wherein a stimulus given at the first stimulator is followed by a delay before the second stimulator discharges. **A:** The first stimulus depolarizes the ulnar nerve and travels both distally and proximally. **B:** The distal pulse results in an ulnar compound muscle action potential (CMAP) from the ulnar-innervated thenar muscles. **C:** The second stimulus at Erb's point is given at a time before the first stimulus antidromically passes the proximal stimulation site. Co-stimulation of both median and ulnar fibers results. **D:** The depolarization in the ulnar fibers collides with the antidromic impulse from the first stimulus, resulting in elimination of the impulse. The median fibers are not affected by the collision, and continue to travel down the nerve. **E:** The true proximal median CMAP is then recorded; it can be used both to calculate a proximal conduction velocity and to look for evidence of conduction block.

stimulation site (and vice versa). The collision study is performed by giving a stimulus at the first stimulator (at the wrist) followed by a slight delay before the second stimulator (proximal site) discharges. Ideally, the delay should be as long as possible, but not longer than the time it takes for a depolarization to travel from the distal to proximal stimulation sites. The first stimulus depolarizes the ulnar nerve and the pulse travels both distally and proximally. The distal pulse results in an ulnar CMAP from ulnar-innervated thenar muscles. The second stimulus is slightly delayed but is given before the first stimulus passes the proximal stimulation site. The second stimulus, which is given at the proximal site (axilla or Erb's point), results in co-stimulation of both median and ulnar fibers. The depolarization in the ulnar fibers, which travels distally down the arm, collides with the proximally traveling impulse from the first stimulus and is thereby blocked, leaving only the impulse from the median fibers to continue to travel down the arm. The true proximal median CMAP is then recorded and can be used both to calculate a proximal median conduction velocity and to look for evidence of a conduction block (Figure 30–11). If collision studies are not performed for proximal median studies across the plexus, conduction block and focal slowing may be missed.

Erb's point stimulation also can be used to record other muscles. Either surface or needle electrodes can be used to record many of the major upper extremity muscles (e.g., deltoid, triceps, supra/infraspinatus, biceps). Stimulating at Erb's point, one can compare the amplitude and latency of the resultant CMAPs from side to side. Although a single stimulus site cannot be used to look for conduction block,

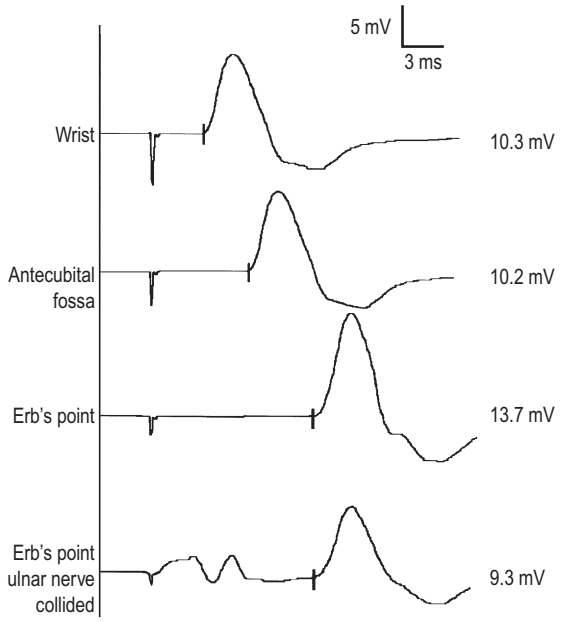

FIGURE 30–11 Median motor conduction studies and co-stimulation at proximal sites. Recording the abductor pollicis brevis, stimulating wrist **(top trace)**, antecubital fossa **(second trace)**, and Erb's point **(third trace)**. Note the higher amplitude at Erb's point from co-stimulation and co-recording of ulnar muscles in the thenar eminence. In the collision study **(bottom trace)**, a supramaximal stimulus is given over the ulnar nerve at the wrist 4 ms before the stimulation at Erb's point. An initial thenar ulnar compound muscle action potential (CMAP) is seen from the first stimulus, followed by the one from the collided Erb's point stimulus, which represents the true proximal median CMAP.

CMAP amplitude can be used to help assess the amount of axonal loss. In some instances, more distal sites such as the axilla can also be used to look for conduction block (see Chapter 31).

Electromyographic Approach

The EMG approach is straightforward in suspected brachial plexopathy (Table 30–3). An extensive EMG study of many muscles must be performed to tease out the correct pattern. Ideally, enough muscles to represent all the trunks, cords, and nerves should be studied. In addition, assessment of the most proximal muscles is of paramount importance to help differentiate a plexus from a root lesion. Because the paraspinal muscles, rhomboids, and serratus anterior come directly off the roots, they should be normal in plexopathies; however, they often are abnormal in root lesions. Nevertheless, it is important to remember that root avulsion can accompany brachial plexopathies, especially in the case of traumatic brachial plexus injuries. When EMG abnormalities are mild or borderline, comparison to the contralateral side is useful.

As in other neuromuscular conditions, the needle EMG is used to look for evidence of active denervation, motor unit action potential (MUAP) abnormalities, recruitment pattern abnormalities, and unusual spontaneous discharges. As mentioned earlier, the presence of myokymic discharges and fasciculations is especially important in differentiating radiation-induced plexopathy from direct neoplastic invasion. Myokymic discharges are recognized as the spontaneous bursting of single MUAPs, resulting in grouped repetitive discharges of the same MUAP. Usually, myokymic bursts fire every 0.5 to 2.0 seconds; typical firing frequencies range from 20 to 70 Hz within bursts.

The needle EMG is also used to evaluate the severity of the lesion. Most important is the assessment of axonal continuity following traumatic lesions. Absence of axonal continuity (absent CMAP, profuse denervation, no MUAPs activated despite good effort) is an ominous sign. If no axonal continuity can be demonstrated, especially in the case of suspected nerve root avulsion, consideration often is given to surgical exploration, nerve grafts, or tendon transfers, in an attempt to increase function. If the lesion is acute, it is useful to wait and repeat the needle EMG study 3 to 6 months later before considering surgical intervention. Often, evidence of early reinnervation (i.e., nascent MUAPs) can be detected on EMG a few months before clinical improvement is noted. In most cases, if there is any evidence of axonal continuity, further observation is indicated before surgical intervention.

Table 30–3. Recommended Electromyography Protocol for Brachial Plexopathy

1. Examine at least one muscle in each peripheral nerve distribution (median, ulnar, radial, anterior interosseous, posterior interosseous, axillary, musculocutaneous, suprascapular).
2. Sample muscles innervated by the same nerve but from different roots.
3. All clinically weak or paralyzed muscles should be examined.
4. Proximal muscles must be examined, including the paraspinal muscles. In suspected upper trunk lesions, examine the rhomboids and/or serratus anterior as well.
5. If findings are borderline or equivocal, compare with findings on the contralateral side.

Example Muscles	
Median	Pronator teres, abductor pollicis brevis
Anterior interosseous	Flexor pollicis longus
Posterior interosseous	Extensor indicis proprius, extensor digitorum communis
Ulnar	First dorsal interosseous, flexor digitorum profundus
Radial	Extensor carpi radialis, brachioradialis, triceps
Axillary	Deltoid
Musculocutaneous	Biceps brachii
Suprascapular	Supraspinatus , infraspinatus
Dorsal scapular	Rhomboids
Dorsal rami	Cervical paraspinals

COMMON ELECTROPHYSIOLOGIC PATTERNS OF BRACHIAL PLEXOPATHY

Upper Trunk Plexopathy

Upper trunk lesions may result in abnormal lateral antebrachial cutaneous sensory responses. In addition, radial and median sensory responses may be abnormal, especially when recording the thumb. Median and ulnar motor conduction studies and F responses are normal. Needle EMG abnormalities may involve the deltoid, biceps, brachioradialis, supraspinatus and infraspinatus muscles. The triceps, pronator teres, and flexor carpi radialis may be partially involved. Most important, the rhomboids, serratus anterior, and cervical paraspinal muscles are spared, unless the lesion also involves the nerve roots. One curious phenomenon which may be seen on needle EMG following an upper trunk plexopathy which also involves the C5 root proper is the "breathing arm." This phenomenon results from aberrant regeneration wherein fibers originally supplying the phrenic nerve grow back and innervate upper trunk muscles. This results in synkinesis, wherein some upper trunk muscles spontaneously fire in a rhythmic pattern. When closely observed, one will note that the firing only occurs when the patient breathes, specifically during inspiration. The phrenic nerve is derived from the C3, C4, and C5 roots. Any trauma which injures the C5 root before the branch to the phrenic nerve can result in this syndrome. This phenomenon was first described in Erb's obstetric brachial plexus, but it can be seen with other trauma or after surgery.

Middle Trunk Plexopathy

Middle trunk lesions may affect the median SNAP, especially when recording the middle finger. The radial SNAP also may be abnormal. Median and ulnar motor conduction studies and F responses are normal. Needle EMG may show abnormalities in C7-innervated muscles (e.g., triceps, pronator teres, flexor carpi radialis).

Lower Trunk Plexopathy

Lower trunk lesions affect the ulnar, dorsal ulnar, and medial antebrachial cutaneous SNAPs. Because the median- and ulnar-innervated hand muscles are derived from the lower trunk, their respective motor conduction studies and F responses may be abnormal as well. If axonal loss is present, CMAP amplitudes may be reduced, with mild prolongation of distal latency and mild slowing of conduction velocity. The vast majority of these lesions are axonal rather than demyelinating, so motor conduction studies across the brachial plexus (although theoretically appealing) most often are not helpful. Needle EMG may show abnormalities in all ulnar-innervated muscles as well as the median- and radial-innervated muscles that contain C8 or T1 fibers, including the flexor pollicis longus, APB, and EIP.

Lateral Cord Plexopathy

Lateral cord lesions affect the lateral antebrachial SNAP and the median SNAPs recording the thumb, index, or middle fingers. Median and ulnar motor conduction studies and F responses are normal. Needle EMG may show abnormalities in the biceps and proximal median forearm muscles (pronator teres, flexor carpi radialis). Distal median-innervated muscles in the forearm and hand, including the flexor pollicis longus and APB, are normal.

Posterior Cord Plexopathy

Posterior cord lesions result in abnormal radial SNAPs. Routine median and ulnar motor conduction studies and F responses are normal. Because the radial-innervated extensor indicus proprius muscle is derived from the lower trunk, the radial motor study recording the EIP may be abnormal. If axonal loss is present, CMAP amplitudes may be reduced, with mild prolongation of distal latency and mild slowing of conduction velocity. Needle EMG may show abnormalities in both distal and proximal radial-innervated muscles (e.g., extensor indicis proprius, extensor carpi radialis, brachioradialis, triceps). In addition, abnormalities may be seen in the deltoid, teres minor, and latissimus dorsi.

Medial Cord

Medial cord lesions are identical to lower trunk plexopathies, except that radial-innervated C8 muscles are normal on EMG. Medial cord lesions may affect the ulnar, dorsal ulnar, and medial antebrachial cutaneous SNAPs. Because the median- and ulnar-innervated hand muscles are derived from the medial cord, their respective motor studies and F responses may be abnormal as well. If axonal loss is present, CMAP amplitudes may be reduced with mild prolongation of distal latency and mild slowing of conduction velocity. Needle EMG abnormalities are limited to all ulnar-innervated muscles and the distal median-innervated muscles that contain C8–T1 fibers (e.g., APB, flexor pollicis longus).

Neurogenic Thoracic Outlet Syndrome

True neurogenic TOS is actually a lower trunk plexopathy. In this entrapment neuropathy, the T1 fibers tend to be preferentially affected, leading to a distinctive pattern on nerve conduction studies and EMG (Figure 30–12). An axonal loss pattern develops (low CMAP amplitude) in both the median and ulnar motor nerves, preferentially affecting the median-innervated thenar muscles. Median and ulnar distal latencies and conduction velocities may be slightly slowed. Stimulating more proximally at Erb's point

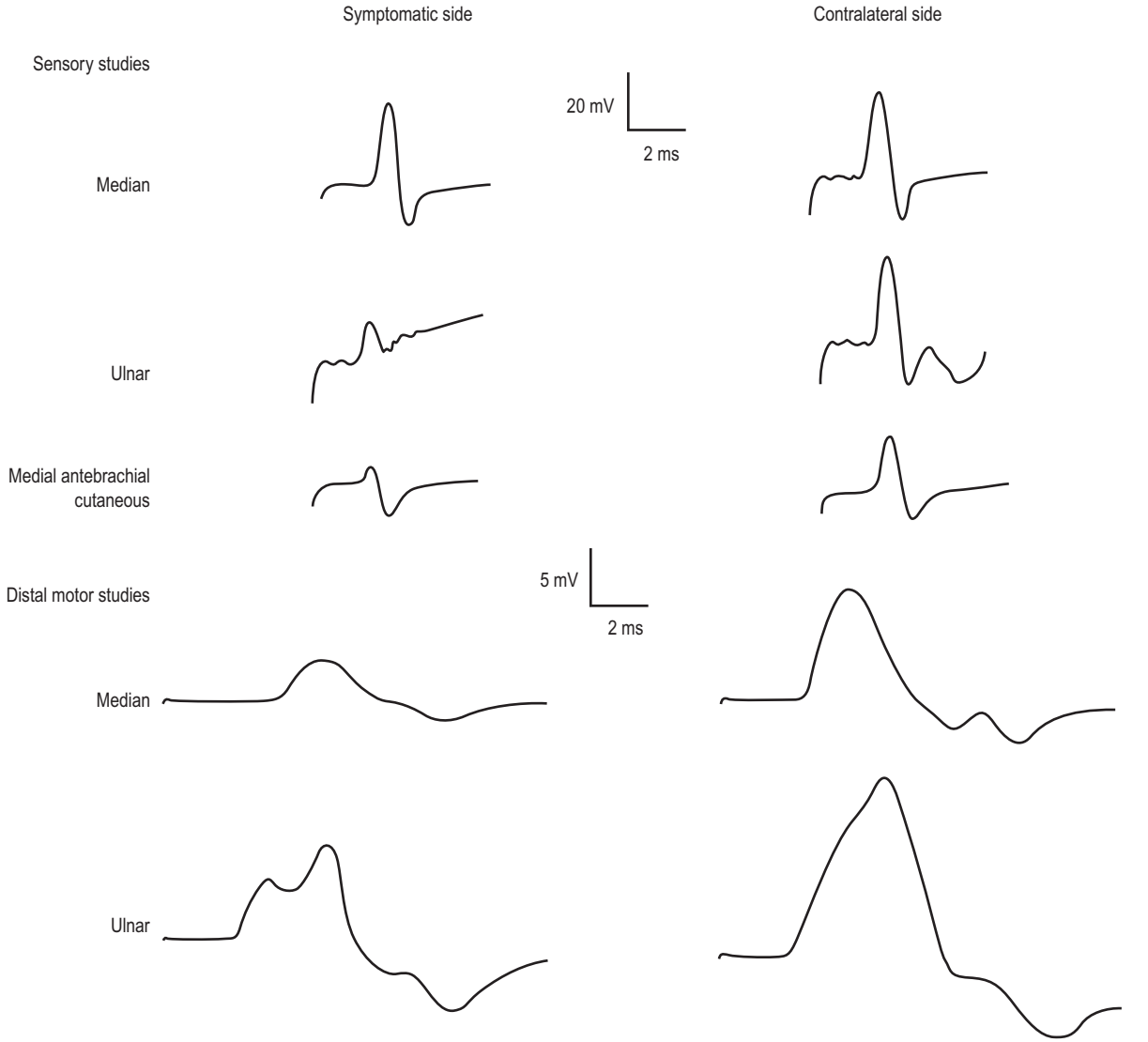

FIGURE 30–12 Nerve conduction studies in thoracic outlet syndrome (TOS). A distinctive pattern of motor and sensory conduction studies occurs in TOS. C8 and especially T1 fibers passing through the lower trunk are affected in true neurogenic TOS. On sensory conduction studies, ulnar and medial antebrachial cutaneous sensory nerve action potentials (SNAPs) are abnormal, but median SNAPs are spared. Whereas both ulnar and medial antebrachial cutaneous sensory fibers travel through the lower trunk, median sensory fibers are derived from the upper and middle trunks. A different pattern is seen on motor conduction studies. Although both median- and ulnar-innervated motor fibers travel through the lower trunk, the median-innervated thenar muscles typically are more affected than are the ulnar-innervated hypothenar muscles, probably reflecting greater T1 innervation of thenar compared to hypothenar muscles.

is of little use in true neurogenic TOS, where the lesion usually is due to axonal loss with no evidence of proximal conduction block.

The sensory nerve conduction studies also reveal a distinctive pattern. Despite the decreased median CMAP amplitude, the median SNAP is normal. This is because the median sensory fibers do not travel through the lower trunk but rather through the upper and middle trunks, which are not involved in neurogenic TOS. The ulnar sensory response, however, is abnormal because the ulnar sensory fibers travel through the lower trunk. In most cases, the ulnar SNAP is low in amplitude but not absent. The medial antebrachial cutaneous SNAP is also usually low in amplitude or absent in true neurogenic TOS (Figure 30–13). Because this nerve is predominantly T1 innervated and travels through the lower trunk and medial cord, it also is subject to damage in neurogenic TOS.

Needle EMG abnormalities are found in median- more than ulnar-innervated C8–T1 muscles and less so in radial-innervated C8 muscles.

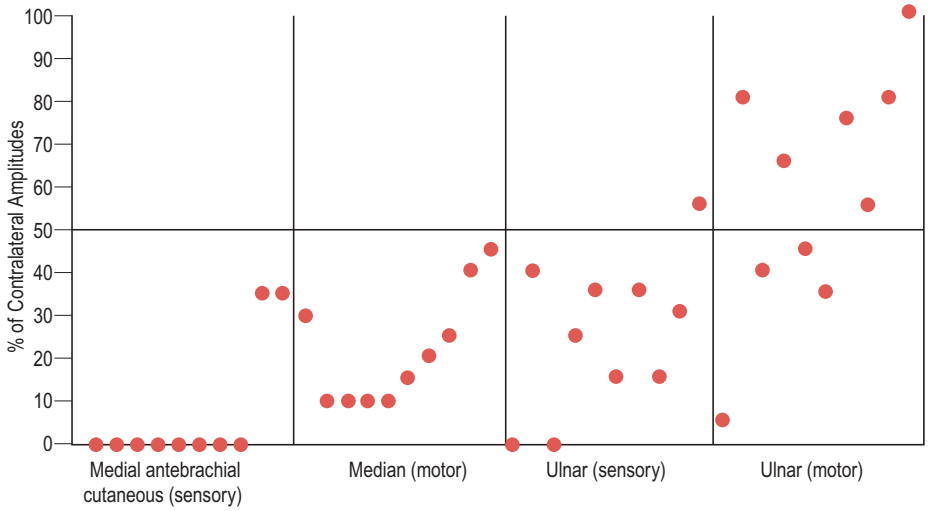

FIGURE 30–13 Nerve conduction studies in patients with neurogenic thoracic outlet syndrome. The sensory nerve action potential (SNAP) and compound muscle action potential amplitudes recorded on the affected side as a percentage of the amplitude on the contralateral side in 10 patients with neurogenic thoracic outlet syndrome. The sensory studies reveal a distinctive pattern. The ulnar sensory response is abnormal because the ulnar sensory fibers travel through the lower trunk. In most cases, the ulnar SNAP is low but not absent. The medial antebrachial cutaneous SNAP is also usually low in amplitude or absent in true neurogenic TOS. Because this nerve is predominantly T1 innervated and travels through the lower trunk and medial cord, it is preferentially subject to damage in neurogenic TOS.
(From Levin, K.H., Wilbourn, A.J., Maggiano, H.J., 1998. Cervical rib and median sternotomy-related brachial plexopathies: a reassessment. Neurology 50, 1407–1413, with permission.)

EXAMPLE CASES

Case 30–1

History and Physical Examination

A 68-year-old man developed numbness and weakness in the left hand after coronary artery bypass surgery. He had no history of weakness or numbness before the operation. On awakening from surgery, he noted numbness in the fourth and fifth fingers with loss of dexterity. There was no associated pain.

When the patient was examined 11 days after the operation, there was hypesthesia of the left fourth and fifth fingers and the hypothenar eminence. There was a suggestion of hypesthesia along the medial forearm. Motor testing showed normal muscle bulk throughout. All of the intrinsic hand muscles on the left were moderately weak, including the APB, interossei, and ADM. The left long finger and thumb flexors were moderately weak, and the wrist and finger extensors were mildly weak; the index finger extensor was the weakest. Strength was otherwise normal, as were the deep tendon reflexes.

CASE 30–1. Nerve Conduction Studies

			Amplitude Motor = mV; Sensory = µV			Latency (ms)			Conduction Velocity (m/s)			F-Wave Latency (ms)		
Nerve Stimulated	Stimulation Site	Recording Site	RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	10.3	8.2	≥4	3.2	3.5	≤4.4				24	26	≤31
	Antecubital fossa	APB	10.2	7.6		6.2	6.8		60	54	≥49			
Ulnar (m)	Wrist	ADM	9.5	6.6	≥6	2.9	3.3	≤3.3				25	28	≤32
	Below elbow	ADM	9.3	6.1		6.1	6.1		62	55	≥49			
	Above elbow	ADM	9.1	5.8		7.6	8.0		65	53	≥49			
Median (s)	Wrist	Index finger	22	20	≥20	3.5	3.3	≤3.5	48	50	≥50			
Ulnar (s)	Wrist	Little finger	18	8	≥17	2.7	2.9	≤3.1	52	48	≥50			
Radial (s)	Forearm	Snuffbox	19	15	≥15	2.3	2.3	≤2.9	56	59	≥50			
Lateral antebrachial (s)	Elbow	Lateral forearm	17	18	≥10	2.2	2.4	≤3.0	70	67	≥55			
Medial antebrachial (s)	Elbow	Medial forearm	16	NR	≥5	2.2	NR	≤3.2	70	NR	≥50			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; APB = abductor pollicis brevis; ADM = abductor digiti minimi.
Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 30–1. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Left first dorsal interosseous	NL	0	0	NL	↓	NL	NL	NL
Left APB	NL	0	0	NL	↓	NL	NL	NL
Left EIP	NL	0	0	NL	↓↓	NL	NL	NL
Left flexor carpi ulnaris	NL	0	0	NL	↓	NL	NL	NL
Left flexor digitorum profundus IV	NL	0	0	NL	↓↓	NL	NL	NL
Left biceps brachii	NL	0	0	NL	NL	NL	NL	NL
Left pronator teres	NL	0	0	NL	NL	NL	NL	NL
Left triceps brachii	NL	0	0	NL	NL	NL	NL	NL
Left C7 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Left C8 paraspinal	NL	0	0	NL	NL	NL	NL	NL

↓ = slightly reduced; ↓↓ = moderately reduced; NL = normal; APB = abductor pollicis brevis; EIP = extensor indicis proprius.

Summary

The history is that of an older gentleman who noted numbness and weakness of the left hand on awakening from coronary artery bypass surgery. The neurologic examination is notable for hypesthesia of digits 4 and 5 and the medial forearm, and weakness without wasting of the intrinsic hand muscles, long finger and thumb flexors, and wrist and finger extensors on the left side.

Nerve conduction studies done 11 days after the surgery show that the left ulnar CMAP amplitude is slightly low compared with the right side, although it is within normal limits. The median motor study is normal bilaterally, as are the median and ulnar F responses. The left ulnar SNAP is of low amplitude, and the medial antebrachial cutaneous SNAP is unrecordable. The remainder of the SNAPs on the left side, including the median, radial, and lateral antebrachial cutaneous SNAPs, and all of the SNAPs on the right side are normal. The abnormal ulnar and medial antebrachial cutaneous SNAPs correspond to the areas of hypesthesia found on the neurologic examination. Given that both of these sensory potentials are abnormal, wallerian degeneration with axonal loss must have taken place, and the lesion must be at or distal to the dorsal root ganglion, in nerve fibers that subserve the lower trunk or medial cord of the brachial plexus. The relatively low CMAP amplitude in the left ulnar nerve, which is mediated by fibers from the lower trunk and medial cord, also lends support to this localization.

The needle EMG examination shows no spontaneous activity in any of the left upper extremity or cervical paraspinal muscles examined. There is mildly reduced MUAP recruitment in the first dorsal interosseous, APB, and flexor carpi ulnaris muscles, and moderately reduced recruitment in the EIP and flexor digitorum profundus muscles. All of the MUAPs are normal in configuration. Note that the needle abnormalities are found in C8–T1-innervated muscles innervated by the median (APB), ulnar (first dorsal interosseous, flexor carpi ulnaris, flexor digitorum profundus IV), and radial (EIP) nerves, which are derived from the medial and posterior cords of the brachial plexus. The C6–C7-innervated muscles innervated by the median and radial nerves (pronator teres, triceps) are normal, as are the biceps and the C7 and C8 paraspinal muscles.

The abnormalities on the needle examination add several important pieces of information. First, the lesion must be fairly proximal along the C8–T1 fibers to involve muscles innervated by both the medial and posterior cords. However, the paraspinal muscles are normal, and the SNAPs are abnormal, placing the lesion at or distal to the dorsal root ganglion. The nerve conduction studies pointed to a lesion in either the medial cord or the lower trunk. Putting all this information together, the lesion must be in the lower trunk of the brachial plexus rather than at the level of the nerve roots. We can now form an electrophysiologic impression.

IMPRESSION: *There is electrophysiologic evidence of an acute axonal lesion of the lower trunk of the brachial plexus on the left.*

The history of numbness in digits 4 and 5 and weakness of the hand immediately after coronary artery bypass surgery should suggest a lesion of the brachial plexus, usually as a result of stretch injury from retraction of the

chest wall. In this case, the clinical history, examination, and electrodiagnostic study pointed toward an acute lesion of the lower trunk of the brachial plexus. The nerve conduction studies revealed the lesion to be axonal. The patient subsequently recovered full function over a period of 8 months.

This case raises several important questions.

If the Lesion is Axonal, Why are there no Fibrillation Potentials?

The abnormal SNAPs, low CMAP, and reduced recruitment of normal configuration MUAPs indicate that there has been enough time for wallerian degeneration to occur. However, active denervation potentials (i.e., fibrillation potentials and positive sharp waves) generally take 10 days to 2 weeks to appear in the most proximal muscles and even longer in more distal muscles. Consistent with the clinical history of 11 days, this lesion is acute. Note the importance of being able to accurately judge MUAP recruitment. In this case, the only abnormality noted on the needle examination was reduced recruitment of MUAPs, which was helpful in localizing the lesion to the lower trunk of the brachial plexus.

Could this be a Case of a Non-localizing Ulnar Neuropathy, with a Superimposed C8–T1 Radiculopathy?

Remember that the paraspinal muscles do not have to be abnormal in radiculopathies, especially in a case such as this one, in which the patient was studied just 11 days after developing weakness, by which time fibrillation potentials, for example, may not yet have developed. However, the absent SNAP from the left medial antebrachial cutaneous nerve, which comes directly off the medial cord of the brachial plexus, places the lesion at or distal to the dorsal root ganglion and outside the distribution of the ulnar nerve. The most parsimonious explanation of the data is a lower trunk brachial plexopathy. The importance of the abnormal medial antebrachial cutaneous SNAP is underscored here: without it, the results of the nerve conduction studies and needle EMG might be explained on the basis of an acute non-localizing ulnar neuropathy with a superimposed acute C8–T1 radiculopathy.

Case 30–2

History and Physical Examination

A 49-year-old woman was referred for evaluation of right hand numbness. She had noted slowly worsening numbness of the fourth and fifth digits of the right hand over 10 years, without pain. Symptoms initially were intermittent but had become more persistent in the last month. She also had noticed weakness of the right hand, especially when opening jars or turning the car key in the ignition.

Her past medical history was significant for Hodgkin's lymphoma 20 years ago that was treated with mantle radiotherapy. A recurrence in the right neck 14 years ago was treated successfully with local radiotherapy.

Examination was notable for normal cranial nerves, with no evidence of a Horner's syndrome. There was decreased bulk in the right thenar and hypothenar areas, with weakness of right thumb abduction and the interossei. Hypesthesia was present in the right fifth and medial aspect of the fourth fingers. Bulk, strength, and sensation were normal in the left arm. No reflexes were present in either arm. Strength and reflexes were normal and symmetric in the lower extremities. There were undulating,

CASE 30–2. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = µV			Latency (ms)			Conduction Velocity (m/s)			F-Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	5.4		≥4	4.7		≤4.4				33		≤31
	Antecubital fossa	APB	4.5			9.8			47		≥49			
Ulnar (m)	Wrist	ADM	6.5		≥6	3.0		≤3.3				30		≤32
	Below elbow	ADM	5.2			6.9			51		≥49			
	Above elbow	ADM	5.0			9.2			54		≥49			
Median (s)	Wrist	Index finger	7	12	≥20	3.2	2.8	≤3.5	54	62	≥50			
Ulnar (s)	Wrist	Little finger	38	13	≥17	3.0	3.0	≤2.8	47	58	≥50			
Radial (s)	Forearm	Snuffbox	8	10	≥15	2.7	2.5	≤2.9	57	61	≥50			
Medial antebrachial (s)	Elbow	Medial forearm	5	3	≥5	2.6	2.7	≤3.2	59	54	≥50			
Sural (s)	Calf	Posterior ankle	14		≥6	3.6		≤4.4	47		≥40			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; APB = abductor pollicis brevis; ADM = abductor digiti minimi.
Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 30–2. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right first dorsal interosseous	MK	0	0	NL	↓↓	+1	+1	+1
Right APB	MK	0	0	NL	↓↓	+2	+1	+1
Right EIP	MK	0	0	NL	↓↓	+1	+1	+1
Right pronator teres	NL	0	0	NL	NL	NL	NL	NL
Right biceps brachii	NL	0	0	NL	NL	NL	NL	NL
Right flexor digitorum profundus IV	MK	0	0	NL	NL	NL	NL	NL
Right triceps brachii	NL	0	0	NL	↓↓	+1	+1	+1
Right C6 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Right C7 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Right C8 paraspinal	NL	0	0	NL	NL	NL	NL	NL

↓↓ = moderately reduced; NL = normal; MK = myokymic discharges; APB = abductor pollicis brevis; EIP = extensor indicis proprius.

wormlike movements of several muscles in the distal right forearm and hand.

Summary

The history is that of a woman with the insidious onset over 10 years of right hand numbness affecting digits 4 and 5 and wasting and weakness of the intrinsic hand muscles on the right. Symptoms are not accompanied by pain. There is a remote history of Hodgkin's lymphoma treated with mantle radiation. Neurologic examination is notable for hypesthesia in the right fifth and medial aspect of the fourth digit, with weakness of the right intrinsic hand muscles and areflexia in the upper extremities bilaterally. Undulating, wormlike movements are noted in the distal right forearm and hand muscles. The remainder of the neurologic examination is normal.

Nerve conduction studies reveal that the right median distal motor latency and F response are slightly prolonged. The right ulnar motor CMAP amplitude is borderline low, and the F response is normal. The right ulnar SNAP is of normal amplitude and approximately three times the size of the left ulnar SNAP, although it is slightly slow. The SNAP from the medial antebrachial cutaneous nerve, which comes off the medial cord of the brachial plexus, is of normal amplitude on the right and of slightly low amplitude on the left. The median and radial SNAPs are of low amplitude bilaterally, as is the left ulnar SNAP. The right sural SNAP is normal. Note that the right ulnar SNAP, which is normal in amplitude, corresponds to the area of numbness noted on the neurologic examination. Remember that there are three possible explanations for finding a normal SNAP in an area of numbness: (1) the lesion is hyperacute (i.e., <6–10 days old for sensory fibers) and there has not been enough time for wallerian degeneration to occur; (2) the lesion is proximal to the dorsal root ganglion, either at the level of the nerve roots or more centrally located in the spinal cord or brain; or (3) the lesion is one of proximal demyelination, possibly conduction block, which leaves the axon relatively intact.

The needle examination should be helpful in distinguishing among these possibilities. Clearly the lesion is not hyperacute because the needle EMG shows large, long reinnervated MUAPs in several muscles subserved by the C7–T1 myotomes in the distribution of the ulnar, median, and radial nerves. The pronator teres, biceps brachii, and cervical paraspinal muscles are normal. The lesion is unlikely to be proximal to the dorsal root ganglion, given the normal paraspinal muscles in conjunction with the abnormal SNAPs. Finally, the finding of myokymic discharges in several of the limb muscles is a very helpful clue. Myokymic discharges are bursts of rhythmic, grouped repetitive discharges of single MUAPs that originate along segments of demyelinated nerve, presumably from spontaneous depolarization or ephaptic transmission along the areas of demyelination. These discharges seen on the needle examination correspond to the undulating, wormlike movements seen in the distal right forearm and hand on the clinical examination.

To summarize, most of the SNAPs in the upper extremities are of low amplitude bilaterally, with the exception of the right ulnar and medial antebrachial cutaneous SNAPs, which are normal, even though this is the area where the patient describes decreased sensation. Although one might suspect that these findings indicate a distal dying back generalized peripheral neuropathy, the fact that the sural SNAP is normal makes this possibility unlikely. One should next consider the possibility of a

brachial plexopathy, especially in light of the history of prior mantle radiation therapy. The needle examination is helpful in that it shows reduced recruitment of reinnervated MUAPs in several muscles subserved by the C7–T1 myotomes, but with normal cervical paraspinal muscles. Thus far, the electrophysiologic findings are consistent with a lesion primarily affecting the middle and lower trunks of the brachial plexus on the right. The needle examination points toward a lesion primarily affecting the lower trunk of the brachial plexus.

We can now form our impression.

IMPRESSION: *There is electrophysiologic evidence of a chronic brachial plexus lesion on the right, primarily affecting the middle and lower trunks. The myokymic discharges are consistent with radiation-induced brachial plexopathy. In addition, the abnormal sensory responses on the left suggest a similar asymptomatic process in the left brachial plexus.*

The history of insidious onset of numbness and weakness in the upper extremity in a patient who has received prior radiation therapy should suggest a delayed radiation-induced plexopathy. Prominent characteristics of delayed radiation-induced plexopathy include the insidious onset over several years, the lack of pain on presentation, and the undulating, wormlike movements on clinical examination, which suggest myokymia.

This case raises several important questions.

If there is Numbness over Digits 4 and 5 of the Right Hand, Why is the Ulnar SNAP Normal?

The ulnar and medial antebrachial cutaneous SNAPs are normal, even in the face of clinical numbness, because they are distal to a primarily demyelinating lesion. The myokymic discharges in several muscle groups belonging to the C7–T1 myotomes lend support to the demyelinating nature of the lesion. Myokymia is common in delayed-onset radiation-induced plexopathy, and its finding on EMG helps to distinguish between neoplastic and radiation-induced brachial plexopathy. The chronic changes in the MUAPs, the slightly prolonged distal median motor latency, and the abnormal sensory potentials in other nerves indicate that there also has been axonal loss. One might have performed more proximal ulnar nerve stimulation in the upper arm and at Erb's point to look for a conduction block across the brachial plexus; such has been reported in radiation-induced brachial plexopathy and would provide further evidence of proximal demyelination. However, one must be cautious with Erb's point stimulation because of the inherent technical difficulty of achieving supramaximal stimulation in that area. As already noted, the other possible explanations for a normal SNAP in an area of sensory loss include a hyperacute lesion or a lesion proximal to the dorsal root ganglion. However, both the clinical history and the finding of reinnervated MUAPs point toward a chronic rather than hyperacute lesion. Furthermore, the low-amplitude median and radial SNAPs and the normal cervical paraspinal muscles indicate that the lesion is at or distal to the dorsal root ganglion, leaving proximal demyelination as the most reasonable explanation.

Do the Abnormal SNAPs on the Left Side Indicate a Brachial Plexopathy on that Side?

The abnormal SNAPs on the left indicate that there may be a brachial plexopathy on that side as well, although the possibility was not fully investigated because the patient was asymptomatic on that side. The normal sural SNAP was important in ruling out a chronic generalized peripheral neuropathy. Deep tendon reflexes are absent in both upper extremities but present and normal in the lower extremities, a finding that also supports bilateral brachial plexus dysfunction, although it is asymptomatic on the left side.

Case 30–3

History and Physical Examination

A 15-year-old boy was referred for persistent weakness and numbness of the left arm 4 months after a traumatic injury from a bicycle accident. Examination showed marked wasting of the left shoulder girdle and upper arm. He was completely unable to abduct the shoulder or to flex the arm at the elbow. Arm extension was present but weak. Wrist flexion and extension as well as intrinsic hand function were relatively intact. The left biceps and brachioradialis deep tendon reflexes were absent. All other reflexes were present and normal. Sensation was diminished over the lateral arm and forearm.

Summary

The history is that of a young boy who sustained a traumatic injury from a bicycle accident, resulting in persistent and profound weakness and wasting of the left arm over 4 months, primarily affecting the shoulder girdle and upper arm musculature on the left. The neurologic examination is notable for weakness and wasting of shoulder abduction and arm flexion and extension, sensory loss over the lateral arm and forearm, and depressed biceps and brachioradialis reflexes.

On nerve conduction studies, the left median and ulnar motor conduction studies and F responses are normal. The median and ulnar SNAPs are normal and symmetric bilaterally. The left radial SNAP is just at the lower limit of normal and is clearly abnormal in comparison with the right side (less than half the amplitude). This finding emphasizes the need to perform bilateral sensory studies when a brachial plexus lesion is suspected; otherwise the left radial SNAP may have been considered normal. The left lateral antebrachial cutaneous SNAP is absent, with the right side normal.

The needle EMG study shows increased insertional activity and florid fibrillation potentials in muscles in the left C5–C6-innervated myotomes, spanning several nerves including the musculocutaneous (biceps), axillary

CASE 30–3. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μV			Latency (ms)			Conduction Velocity (m/s)			F-Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB		10.2	≥4		3.3	≤4.4					25	≤31
	Antecubital fossa	APB		9.6			6.9			58	≥49			
Ulnar (m)	Wrist	ADM		11.4	≥6		2.8	≤3.3					24	≤32
	Below elbow	ADM		11.0			6.3			62	≥49			
	Above elbow	ADM		10.9			9.7			61	≥49			
Median (s)	Wrist	Index finger	33	25	≥20	2.7	2.8	≤3.5	56	55	≥50			
Ulnar (s)	Wrist	Little finger	27	23	≥17	2.4	2.6	≤3.1	58	54	≥50			
Radial (s)	Forearm	Snuffbox	39	16	≥15	2.0	2.2	≤2.9	57	55	≥50			
Lateral antebrachial (s)	Elbow	Lateral forearm	14	NR	≥10	2.0		≤3.0	58		≥55			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; APB = abductor pollicis brevis; ADM = abductor digiti minimi.
Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 30–3. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Left biceps brachii	↑	+3	0	None				
Left medial deltoid	↑	+3	0	None				
Left brachioradialis	↑	+3	0	None				
Left infraspinatus	↑	+3	0	None				
Left triceps brachii	NL	0	0	NL	↓	+1	+1	+1
Left pronator teres	↑	+2	0	NL	↓↓↓	+2	+2	+2
Left first dorsal interosseous	NL	0	0	NL	NL	NL	NL	NL
Left extensor digitorum communis	NL	0	0	NL	NL	NL	NL	NL
Left extensor indicis proprius	NL	0	0	NL	NL	NL	NL	NL
Left serratus anterior	NL	0	0	NL	NL	NL	NL	NL
Left rhomboids	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

↑ = increased; ↓ = slightly reduced; ↓↓↓ = markedly reduced; NL = normal.

(medial deltoid), radial (brachioradialis), suprascapular (infraspinatus), and median (pronator teres) nerves. In addition, there are no MUAPs activated in these muscles, with the exception of the pronator teres, which also has some C7 innervation. There, the MUAPs are very large, long, and polyphasic, with markedly reduced recruitment. In the triceps (C6–C7–C8 innervation) there is mildly reduced recruitment of large, long, and polyphasic MUAPs. Of note, the serratus anterior, rhomboids, and C5 and C6 paraspinal muscles, which carry C5–C7 fibers but come directly off the nerve roots before the brachial plexus, are entirely normal.

In summary, the combination of the abnormal SNAPs and the abnormalities noted on EMG in the limb muscles with sparing of the serratus anterior, rhomboids, and upper cervical paraspinal muscles points toward a severe, chronic lesion primarily affecting the upper trunk of the brachial plexus.

IMPRESSION: *There is electrophysiologic evidence of a severe upper trunk brachial plexopathy on the left. There is no evidence of axonal continuity in muscles that are subserved entirely by the upper trunk. A follow-up study in 3 to 6 months may be helpful to determine whether there has been reinnervation of muscles of the upper trunk.*

This case raises several important questions.

Is the Lesion in the Brachial Plexus Itself, or is there Evidence of Avulsion of the Nerve Roots?

The abnormal radial and lateral antebrachial cutaneous sensory potentials indicate that the lesion is at or distal to the dorsal root ganglion, that is, distal to the nerve roots, in the brachial plexus itself. The sparing of the serratus anterior, rhomboids, and upper cervical paraspinal muscles is a key finding to corroborate that there has not been root avulsion. Because those muscles come directly off the roots, proximal to the brachial plexus, there should be abnormal EMG findings in some of these muscles if the roots had been avulsed. Note, however, that in rare cases of root avulsion the paraspinal muscles are normal on EMG, which may indicate relative sparing of the dorsal rami in some root injuries.

What is the Prognosis for Recovery?

This study was performed 4 months after the original injury. The presence of profuse fibrillation potentials with no activation of MUAPs in C5–C6-innervated muscles and reduced recruitment of large MUAPs in C6–C7-innervated muscles indicate that the lesion is severe and chronic, with extensive axonal loss. There is no evidence at this point of reinnervation in any muscles innervated solely by the C5–C6 roots. Although root avulsion carries a poorer prognosis than an injury to the plexus itself, this patient has a very severe lesion. A follow-up study in 3 to 6 months has been recommended in order to document whether and to what extent reinnervation may occur. At 4 months, it is still too early to make any definite statements about prognosis or what treatment options would be best, including possible muscle or tendon transfers if reinnervation remains poor.

If there is so Much Axonal Loss, Why are the CMAPs Normal?

The CMAPs are normal because recording was done from median- and ulnar-innervated hand muscles, which are subserved by the C8–T1 myotomes. Because those fibers were not affected, one would expect the CMAPs to be normal when recording from these muscles. If one were to record over the biceps or medial deltoid, stimulating the musculocutaneous or axillary nerves, respectively, one would expect to find a very low or unrecordable CMAP amplitude.

Suggested Readings

Dawson, D.M., Hallet, M., Wilbourn, A.J., 1999. Entrapment neuropathies, third ed. Philadelphia, Lippincott.

Harper, C.M., Juergen, E.T., Cascino, T.L., et al., 1989. Distinction between neoplastic and radiation-induced brachial plexopathy, with emphasis on the role of EMG. Neurology 39, 502.

Hollinshead, W.H., 1969. Anatomy for surgeons, volume 2: the back and limbs. New York, Harper & Row.

Lederman, R.J., Wilbourn, A.J., 1984. Brachial plexopathy: recurrent cancer or radiation? Neurology 34, 1331.

Lederman, R.J., Breuer, A.C., Hanson, M.R., et al., 1982. Peripheral nervous system complications of coronary artery bypass graft surgery. Ann Neurol 12, 297.

Levin, K.H., Lederman, R.J., Wilbourn, A.J., 1987. Spectrum of EMG changes in radiation brachial plexopathy. Muscle Nerve 10, 656.

Levin, K.H., Wilbourn, A.J., Maggiano, H.J., 1998. Cervical rib and median sternotomy-related brachial plexopathies: a reassessment. Neurology 50, 1407–1413.

Swift, T.R., 1994. The breathing arm. Muscle Nerve 17 (1), 125–129.

Vahl, C.F., Carl, I., Muller-Vahl, H., et al., 1991. Brachial plexus injury after cardiac surgery: the role of internal mammary artery preparation: a prospective study on 1,000 consecutive patients. J Thorac Cardiovasc Surg 102, 724.