INVITED REVIEW

ABSTRACT: The brachial plexus, which is the most complex structure of the peripheral nervous system, supplies most of the upper extremity and shoulder. The high incidence of brachial plexopathies reflects its vulnerability to trauma and the tendency of disorders involving adjacent structures to affect it secondarily. The combination of anatomic, pathophysiologic, and neuromuscular knowledge with detailed clinical and ancillary study evaluations provides diagnostic and prognostic information that is important to clinical management. Since most brachial plexus disorders do not involve the entire brachial plexus but, rather, show a regional predilection, a regional approach to assessment of plexopathies is necessary.

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BRACHIAL PLEXOPATHIES: CLASSIFICATION, CAUSES, AND CONSEQUENCES

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The brachial plexus, which supplies most of the upper extremity and shoulder, is the most complex structure in the peripheral nervous system (PNS). Its vulnerability to trauma reflects its large size, superficial location, and position between two highly mobile structures (neck and upper extremity).[121,144] Also, it may be affected secondarily by pulmonary, vascular, or skeletal disorders involving neighboring structures. Hence, most physicians encounter patients with brachial plexopathies. In addition to a comprehensive clinical evaluation, optimal assessment requires the performance of ancillary studies. Of these, electrodiagnostic examination is by far the most helpful. Although an extension of the neurologic examination, it has several advantages over the latter, including the ability to localize and characterize the lesion, evaluate muscles not easily assessed clinically (e.g., anconeus), recognize minimally affected muscles that seem normal clinically, prove continuity when visible muscle movement is lacking, recognize remote lesions no longer appreciable clinically, and estimate lesion severity for current and future comparative studies.

By integrating requisite anatomic, pathophysiologic, and neuromuscular knowledge with detailed clinical assessment and the results of ancillary studies, the examining physician can make an accurate diagnosis and prognosis. The lesion must be localized and characterized. This ability requires an understanding of the relevant anatomy, as well as a familiarity with disorders affecting the brachial plexus. This review details a regional approach to assessment of the brachial plexus and discusses certain plexopathies, especially those with a regional proclivity. Pertinent aspects of the anatomy, pathology, pathophysiology, electrodiagnosis, and injury classification of these disorders are reviewed.

Abbreviations: ADM, abductor digiti minimi; AHC, anterior horn cell; APB, abductor pollicis brevis; CMAP, compound muscle action potential; CSF, cerebrospinal fluid; CT, computerized tomography; DRG, dorsal root ganglion; DUC, dorsal ulnar cutaneous; EDC, extensor digitorum communis; EIP, extensor indicis proprius; EPB, extensor pollicis brevis; FDI, first dorsal interosseous; LABC, lateral antebrachial cutaneous; MABC, medial antebrachial cutaneous; MR, magnetic resonance; MUAP, motor unit action potential; NA, neuralgic amyotrophy; NCS, nerve conduction study; NEE, needle electrode examination; PNS, peripheral nervous system; SNAP, sensory nerve action potential; TOS, thoracic outlet syndrome

Key words: brachial plexus, classic postoperative paralysis, electrodiagnostic evaluation, iatrogenic plexopathy, medial brachial fascial compartment, neoplastic plexopathy, neuralgic amyotrophy, obstetric plexopathy, plexopathy, Pancoast syndrome, postmedian sternotomy, radiation plexopathy, root avulsion, rucksack, thoracic outlet syndrome, trauma

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ANATOMY

The brachial plexus is a triangular-shaped structure that extends from the spinal cord to the axilla. Its average extraforaminal length is 15.3 cm.[117] It is composed of connective and neural tissue in a 2 to 1 ratio,[9,117,154] and contains several elements: (1) five roots (classically, C5 through T1); (2) three trunks (upper, middle, and lower); (3) six divisions (three anterior, three posterior); (4) three cords (lateral, posterior, and medial); and (5) several terminal nerves (Fig. 1). The C6, C7, and C8 roots each

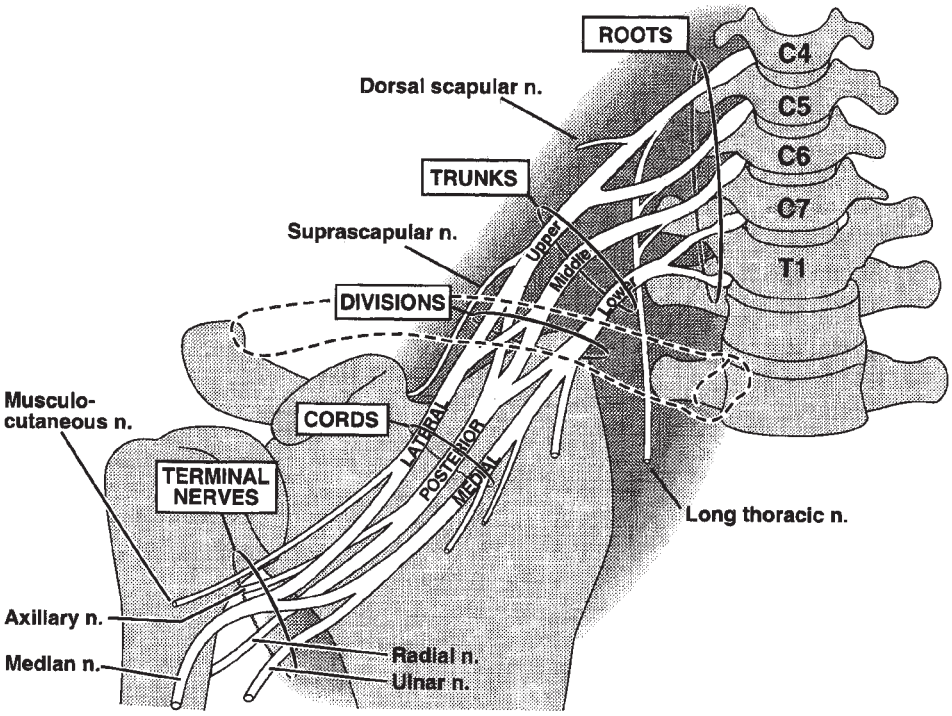

FIGURE 1. The brachial plexus.

provide about 25% of its nerve fibers, and the C5 and T1 roots provide the remainder.[117] The percentage of sensory and motor fibers composing each root varies. The largest percentage of motor fibers is found in the C5 and C6 roots; C7 and T1 have the least.[46,154] The greatest number of sensory fibers is found in the C7 root, followed, in descending order, by C6, C8, T1, and C5.[154] The brachial plexus also carries sympathetic fibers.

Roots. The dorsal and ventral rootlets exit the spinal cord and fuse, forming the dorsal and ventral roots, respectively. The latter enter the intervertebral foramen and fuse in the distal foramen, just beyond the dorsal root ganglion (DRG), creating a spinal nerve. (The latter are also referred to as mixed spinal nerves because they contain both sensory and motor nerve fibers.) After exiting the foramen, these nerves give off posteriorly directed branches, the posterior primary rami, and continue as anterior primary rami (Fig. 2). The anterior primary rami emerge from between the anterior and middle scalene muscles. The long thoracic nerve (serratus anterior) is derived via branches from the C5–C7 anterior primary rami, the C5 ramus contributes to the phrenic (diaphragm) and dorsal scapular (levator scapulae; rhomboids) nerves, and the C5–C8 rami supply the scalene and longus colli muscles. Preganglionic sympathetic fibers leave the spinal cord and exit the anterior primary rami, via white rami communicantes, to reach the sympathetic ganglia. The sympathetic ganglia send postganglionic fibers, via gray rami communicantes, to the C5 through T1 spinal nerves. Although anatomists define the anterior primary rami as the roots of the brachial plexus, much of the surgical literature defines them as those PNS elements proximal to the trunks.[144] Because of its clinical utility, the latter approach is used in this article.

Trunks. The trunks are located in the posterior cervical triangle, behind the clavicle and sternocleidomastoid. Trunk anomalies are infrequent.[75] Typically, the C5 and C6 anterior primary rami unite, the C7 anterior primary ramus continues, and the C8 and T1 rami coalesce to become the upper, middle, and lower trunks, respectively (named for their relationship to each other). The upper trunk gives off the suprascapular nerve and the nerve to the subclavius muscle. The lower trunk lies adjacent to the subclavian artery and the apex of the lung.

Divisions. Each trunk divides into anterior and posterior divisions, all of which are retroclavicular. The anterior and posterior divisions primarily supply flexor and extensor muscles, respectively. Although

the anterior and posterior divisions of the upper trunk are similar in caliber, the posterior division of the middle trunk is much larger (C7 extensors) than its anterior division,[154] whereas the posterior division of the lower trunk is much smaller (C8–T1 extensors) than its anterior counterpart. When present, less than 5% of posterior cord fibers are T1-derived.[52,117] Nerves usually do not arise from the divisions.

Cords. The cords are named for their relationship to the second segment of the axillary artery, to which typically they are bound (Fig. 3). They form at or just beyond the clavicle, below the pectoralis minor, and lie in the proximal region of the axilla, near the axillary lymph node chain and major blood vessels to the arm.[16,20,47,138,148] The lateral cord, formed from the anterior divisions of the upper and middle trunks, contains C6–C7 sensory and C5–C7 motor fibers. No C5 sensory fibers exist in the lateral cord, since the C5 dermatome is subserved by the upper and lower lateral cutaneous nerves, which derive from the axillary and radial nerves, respectively; these nerves exit from the posterior cord. The lateral cord gives off the lateral pectoral and musculocutaneous nerves and terminates as the lateral head of the median nerve. The posterior cord, formed by union of the three posterior divisions, contains C5–C7 sensory and C5–C8 motor fibers; it does not contain C8 sensory fibers.[27] It gives off the subscapular and thoracodorsal nerves before terminating as the axillary and radial nerves. The medial cord, a direct continuation of the anterior division of the lower trunk, contains C8 and T1 sensory and motor fibers. It gives off the medial pectoral, medial brachial cutaneous, medial antebrachial cutaneous (MABC), and ulnar nerves, and terminates as the medial head of the median nerve. When the lateral cord or C7 root sends nerve fibers to the ulnar nerve, C7 radiculopathies can produce abnormalities in ulnar-innervated muscles (e.g., flexor carpi ulnaris).[20,45,62,117]

Terminal Nerves. These elements are located in the distal axilla and, depending on the author, number from three (median, ulnar, and radial) to five (inclusion of musculocutaneous and axillary). Except for the median nerve (derived from lateral and medial cords), these nerves originate from a single cord: the ulnar nerve from the medial cord, the axillary and radial nerves from the posterior cord, and the musculocutaneous nerve from the lateral cord. It is unclear at which point the terminal nerves of the brachial plexus become the peripheral nerves of the upper extremity. Narakas defined that point at 3 cm beyond the cord, but Wilbourn prefers to consider the transition site as the point where they exit the axilla.[95,148]

Classically, the brachial plexus is defined as consisting of sensory and motor nerve fibers derived from neurons located in the C5–T1 DRG and anterior horn cells (AHCs), respectively.[16,125] However, vertical variations in its composition are not uncommon. When adjacent roots contribute (e.g., C4, T2), it is "expanded." Vertical shifts result when its formation is shifted one level upward or downward. When the C4 contribution is large and the T1 contribution is small, the brachial plexus is said to be "pre-fixed," and when the C5 contribution is minimal and the T2 contribution is large it is "post-fixed". Since these one-segment shifts do not affect the plexus arrangement itself, they do not affect lesion localization by either clinical or electrodiagnostic examination.

CLASSIFICATION OF BRACHIAL PLEXOPATHIES

Brachial plexopathies can be classified in several ways. They are best classified according to the region involved, such as supraclavicular (root and trunks), retroclavicular (divisions), and infraclavicular (cords and terminal nerves) sites. (Isolated retroclavicular plexopathies are rare.) Although this approach is anatomically simple, it has considerable clinical utility because the incidence, severity, prognosis, and lesion type vary among these regions.[144] In general, supraclavicular plexopathies are more common, more frequently due to closed traction (which can produce lengthy lesions), usually more severe (since greater force is required to produce them), and typically associated with a worse outcome.[1,7,66,144] The supraclavicular plexus is further divided into upper (C5 and C6 roots and upper trunk), middle (C7 root and middle trunk), and lower (C8 and T1 roots and lower trunk) portions and, again, this is a clinically relevant distinction. Patients with upper plexopathies tend to recover more completely because, in general, these lesions are more commonly due to demyelinating conduction block, located closer to the muscles they innervate, and extraforaminal (i.e., surgically accessible). This classification system facilitates communication among physicians since it is easier to discuss a patient with an upper plexopathy than to commit to one of its elements before diagnostic testing has been performed or when there are examination limitations (e.g., pain, mental status changes, or nonneural injuries, such as fractures or dislocations). The infraclavicular

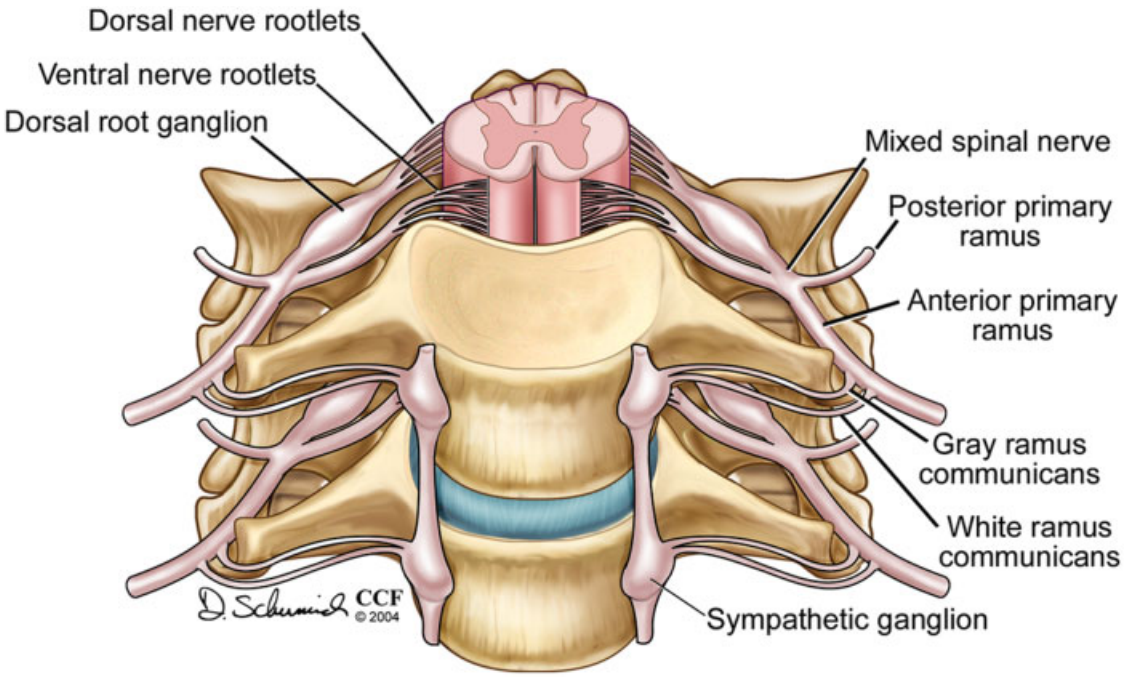

FIGURE 2. The relationship between the more proximal elements of the brachial plexus and the spinal column.

plexus is not subdivided because lesions affecting it do not show significant regional differences in incidence, severity, prognosis, or lesion type.

ASSESSMENT OF THE BRACHIAL PLEXUS

Clinical Assessment. A detailed clinical evaluation is vital for determining lesion localization (especially

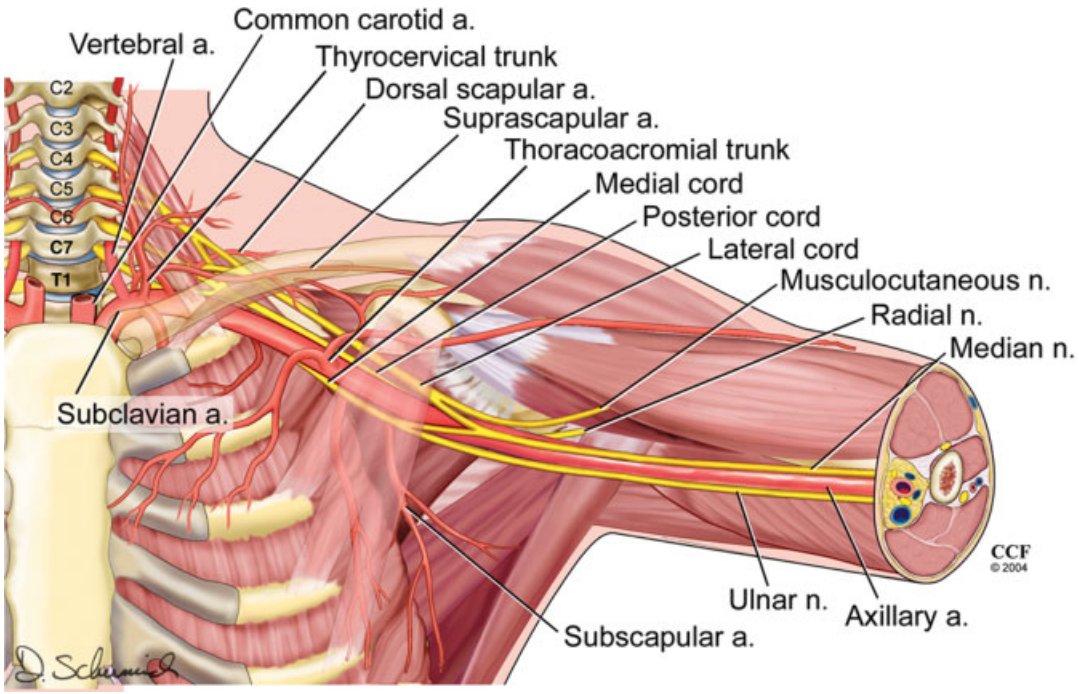

FIGURE 3. The relationship between the brachial plexus and its neighboring arteries.

its proximal extent) and severity (complete or incomplete), both of which have diagnostic and prognostic implications that contribute to clinical management. The initial and subsequent symptoms, the circumstances surrounding their onset (e.g., backpack usage; severe shoulder pain, followed by muscle weakness and wasting; postmedian sternotomy; axillary or scalene block anesthesia), and the past medical history are reviewed. In the setting of trauma, arm position at impact suggests the fibers most likely affected, as may concomitant injuries (e.g., scapular, clavicular, or humeral fracture; glenohumeral dislocation; scapulothoracic dissociation).[156] Since most brachial plexopathies are axon loss in nature, neurologic examination frequently discloses weakness and sensory loss. With supraclavicular lesions, the pattern of sensory and motor loss is segmental—dermatomal and myotomal, respectively—whereas infraclavicular plexopathies produce nonsegmental patterns that resemble those observed with involvement of one or more terminal nerves. The presence of a Horner's syndrome or involvement of the phrenic, dorsal scapular, or long thoracic nerve indicates a proximal process and portends a worse prognosis. Dysautonomic features, such as cutaneous trophic changes, sudomotor abnormalities, and vasomotor abnormalities, may also be noted. Clinical features strongly correlated with root avulsion include severe pain in an anesthetic limb and Horner's syndrome. When traumatic plexopathies are encountered, spinal accessory nerve, cervical plexus, and phrenic nerve function require assessment.

Radiologic Assessment. The radiologic procedures employed reflect the circumstances (e.g., urgency, suspected etiology and lesion site, availability). Plain films of the cervical spine, scapula, clavicle, humerus, shoulder, and chest assess for concomitant injuries and, with open injuries, for foreign bodies.[75] Signs of phrenic nerve dysfunction (e.g., elevated diaphragm), vascular trauma (e.g., mediastinal widening), or lung breach (e.g., pneumothorax, hemothorax) are sought. Radiologic features associated with brachial plexus injury include lateral tilt of the cervical spine and fractures of the transverse process, proximal first rib, or neighboring bones with root avulsion injuries; nonunion or excessive callus formation with inadequately treated midshaft clavicular fractures; humeral fracture or glenohumeral dislocation with infraclavicular plexopathies; bone or lung abnormalities with neoplastic or radiation damage; and rudimentary cervical ribs or elongated C7 transverse processes with true neurogenic thoracic outlet syndrome (TOS).[150]

Despite its drawbacks (monoplanar imaging; beam-hardening artifacts; poor tissue differentiation), computerized tomography (CT) scanning is useful for identifying bony changes and acute collections of blood.[35] Very thin slice (2-mm) CT-myelography images the axially oriented preganglionic root elements when nerve root avulsion is suspected.[119] When the meninges are pulled through the neural foramen, a contrast-filled meningeal diverticulum may be observed. The width of the dye column in the cervical gutter is assessed for narrowing (spinal cord edema) and thickening (spinal cord atrophy), and the intraspinal canal is assessed for masses. Deformed dural pouches, poor root sleeve filling, and cord edema or atrophy have strong correlations with root avulsion.[2,66,119,130] To lessen the chance of arachnoiditis, these studies usually are performed 4–6 weeks after symptom onset in those patients with persistent deficits.[119] As with other studies, falsely positive (e.g., extraforaminal injuries, meningeal tearing without root damage) and negative (e.g., after healing and scarring of the dural pouch) results occur.[20,56,63,86,93,107,108,130,156,158] The reliability of CT-myelography is greatest for C8 and T1 avulsions.[50]

Its noninvasiveness, lack of radiation, multiplanar imaging, lack of degradation by bone, and, especially, its tissue differentiating ability make magnetic resonance (MR) imaging the modality of choice for more distal brachial plexus imaging. Although it is becoming more widely used for proximal brachial plexus assessment, a recent study comparing it to CT-myelography found it less sensitive for avulsion injuries.[11] Unfortunately, when multiple slices and planes are required, acquisition time can be considerable. Magnetic resonance myelography is a newer technique that generates myelogram-like images of the intraspinal canal and intervertebral foramina via the three-dimensional reconstruction of T2-weighted images of the cerebrospinal fluid (CSF).[70,129] Traumatic meningoceles and injuries involving the C5 or C6 spinal nerves can be visualized. Since it is noninvasive, contrast-free, relatively quick, and multiplanar, this technique could become a useful adjunct for assessing proximal brachial plexus elements.[150]

Magnetic resonance neurography can image peripheral nerves using diffusion neurography or T2-based neurography.[31] With diffusion neurography, tissue differentiation reflects water diffusion differences rather than T1 or T2 differences. Tissue brightness is determined by the extent to which protons in that tissue are able to spin at exactly the same rate and in phase with one another. Since the water molecules within a nerve diffuse longitudi-

nally, application of a perpendicular magnetic field gradient allows these molecules to experience a uniform and unchanging field strength, thereby causing the nerve to appear increasingly bright in relation to any surrounding tissue. Unfortunately, this technique is very sensitive to patient motion.[31] In addition, since sagittal images are not truly perpendicular to the plexus elements, identification and evaluation of the latter can be difficult.[85] With T2-based neurography, T2-weighting and both fat and blood suppression, as well as voxel shortening, permit intraneural fascicles to be imaged. Although these techniques can localize neural lesions, they work best when they are directed to a specific region by clinical or electrodiagnostic findings. In the proper setting, MR neurography may be able to recognize nerve discontinuities and ball neuromas (e.g., upper trunk disruption), nerve deflections (e.g., lower trunk angulation caused by a fibrous band), and primary nerve tumors (e.g., schwannomas).[31]

Vascular Assessment. Brachial plexus damage may follow subclavian or axillary vessel damage (e.g., aneurysms, pseudoaneurysms, hematomas). Thus, arteriographic studies often are required, especially when the plexopathy follows a penetrating injury, coexists with or was caused by a primary vessel injury, or when examination discloses absence of the radial or carotid pulse or an expanding mass, bruit, or thrill near the injury site.[66,150] Since the distractive force required to produce neurovascular injury is greater than that to produce isolated neurologic damage, the prognosis for nerve recovery is less favorable when it is associated with concomitant vascular involvement[66]

Electrodiagnostic Assessment of the Brachial Plexus. Electrodiagnostic assessment of brachial plexopathies is invaluable for determining lesion severity and location, which have important diagnostic and prognostic implications. In general, extensive nerve conduction study (NCS) and needle electrode examination (NEE) evaluations are required, in addition to contralateral comparison studies. When approached regionally, however, the requisite number of studies is reduced. Sensory NCS, motor NCS, and NEE are all required because each yields information not discerned by the other two. On NCS, the sensory nerve action potential (SNAP) amplitudes are the most useful indicators of an axon-loss brachial plexopathy. In addition to differentiating pre- and postganglionic lesions, the pattern of SNAP abnormalities has localizing value. And, since many plexopathies have a regional predilection, lesion localization may have diagnostic implications. Thus, whenever a plexopathy is suspected, extensive sensory NCS are performed. Conversely, since motor NCS are quite insensitive to axon loss and are normalized by reinnervation, they are not useful for screening purposes. During the first week, before compound muscle action potential (CMAP) amplitudes reach their nadir, motor NCS can localize both axonal and demyelinating conduction-block lesions; subsequently, they can only localize the latter, which permits differentiation between the two processes. Another use of motor NCS is for estimating lesion severity. Before reinnervation, the relationship between CMAP amplitude and the number of motor fibers is nearly linear. This allows side-to-side CMAP amplitude comparisons to estimate the percentage of motor fibers affected. Due to the large number of fibrillation potentials generated per disrupted axon, the NEE is the most sensitive indicator of motor axon loss. This sensitivity is invaluable for determining the proximal extent of a lesion. Also, since it studies individual motor unit action potentials (MUAPs), it can determine continuity and identify early reinnervation when there is no muscle movement clinically. Moreover, when discordance between temporal and spatial MUAP recruitment is noted, it can identify the presence of a more proximally located demyelinating conduction-block lesion.

Since brachial plexus elements are composed of nerve fibers derived from different spinal cord segments, lesions involving individual elements have different electrodiagnostic features. The muscle domain of a brachial plexus element is defined as the muscles innervated by the motor fibers contained within it. These domains are easily calculated from standard myotomal charts (Table 1).[27,28,30,61,82,83,102,121,149] The CMAP and SNAP domains of an element are determined by the sensory and motor fibers contained within that element and whether they are assessable by NCS.[27,30] Thus, the CMAP domains are a subset of the muscle domains (Table 2).[28,30] Since the sensory nerve fibers subserving the various sensory NCS do not necessarily arise from the same DRG, the pathways through the plexus traversed by these fibers varies. For that reason, the SNAP domains of the brachial plexus elements also vary. These pathways and the frequency with which brachial plexus elemental lesions affect the various SNAPs have been described elsewhere (Table 3).[27,28,30]

Table 1. Muscle domains of the brachial plexus elements.*

Upper trunk	Middle trunk	Lower trunk
Supraspinatus	Pronator teres	Abductor pollicis brevis
Infraspinatus	Flexor carpi radialis	Flexor pollicis longus
Biceps	Triceps	Pronator quadratus
Deltoid	Anconeus	Extensor indicis proprius
Teres minor	Extensor carpi radialis	Extensor pollicis brevis
Triceps	Extensor digitorum communis	Extensor carpi ulnaris
Pronator teres		First dorsal interosseous
Flexor carpi radialis		Abductor digiti minimi
Brachioradialis		Adductor pollicis
Extensor carpi radialis		Flexor digitorum profundus 4,5
Brachialis		Flexor carpi ulnaris
Lateral cord	**Posterior cord**	**Medial cord**
Biceps	Latissimus dorsi	Abductor pollicis brevis
Brachialis	Deltoid	Opponens pollicis
Pronator teres	Teres minor	Flexor pollicis longus
Flexor carpi radialis	Triceps	First dorsal interosseous
	Anconeus	Adductor pollicis
	Brachioradialis	Abductor digiti minimi
	Extensor carpi radialis	Flexor carpi ulnaris
	Extensor digitorum communis	Flexor digitorum profundus 4,5
	Extensor pollicis brevis	
	Extensor carpi ulnaris	
	Extensor indicis proprius	

**Only muscles easily assessed by needle electrode examination are listed.*

Sensory Fiber Pathways. The lateral antebrachial cutaneous (LABC) nerve, which exits from the lateral cord, is the terminal portion of the musculocutaneous nerve. Its sensory fibers derive from the C6 DRG.[27] Thus, based solely on anatomy, in addition to the LABC and musculocutaneous nerves, the LABC sensory NCS assesses the lateral cord, upper trunk, and the C6 anterior primary ramus, spinal nerve, and DRG (Fig. 4). The sensory fibers of the median nerve have particularly complicated pathways through the brachial plexus. Those innervating the thumb emanate from the C6 DRG.[27] Thus, the median sensory NCS, recording from the thumb, assesses the median nerve, lateral cord, upper trunk, and the C6 anterior primary ramus, spinal nerve, and DRG (Fig. 5). Those fibers innervating the index finger derive from the C6 and C7 DRG about 20% and 80% of the time, respectively.[27] Hence, the median sensory NCS, recording from the index finger, assesses the median nerve and lateral cord consistently; the upper trunk and the C6 anterior primary ramus, spinal nerve, and DRG in 20% of instances; and the middle trunk and the C7 anterior primary ramus, spinal nerve, and DRG in 80% of instances (Fig. 6). Those fibers innervating the mid-

Table 2. CMAP domains of the brachial plexus elements.*

Upper trunk	Lateral cord
Musculocutaneous (biceps)	Musculocutaneous (biceps)
Axillary (deltoid)	
Middle trunk	Posterior cord
Radial (anconeus)	Axillary (deltoid)
	Radial (extensor digitorum communis)
	Radial (extensor indicis proprius)
	Radial (anconeus)
Lower trunk	Medial cord
Ulnar (abductor digiti minimi)	Ulnar (abductor digiti minimi)
Ulnar (first dorsal interosseous)	Ulnar (first dorsal interosseous)
Median (abductor pollicis brevis)	Median (abductor pollicis brevis)
Radial (extensor indicis proprius)	

CMAP, compound muscle action potential.
**The recording sites are shown in parentheses.*

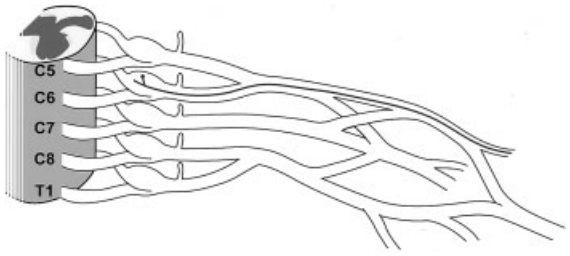

FIGURE 4. Proposed brachial plexus pathway for the sensory fibers assessed by the LABC SNAP.

Table 3. SNAP domains of the trunk and cord elements.*

Upper trunk	Lateral cord
LABC (100%)	LABC (100%)
Median (thumb) (100%)	Median (thumb) (100%)
Superficial radial (60%)	Median (index finger) (100%)
Median (index finger) (20%)	Median (middle finger) (80%)
Median (middle finger) (10%)	
Middle trunk	Posterior cord
Median (index finger) (80%)	Superficial radial (100%)
Median (middle finger) (70%)	
Superficial radial (40%)	
Lower trunk	Medial cord
Ulnar (little finger) (100%)	Ulnar (little finger) (100%)
MABC (100%)	MABC (100%)
Median (middle finger) (20%)	Median (middle finger) (20%)

LABC, lateral antebrachial cutaneous; MABC, medial antebrachial cutaneous; SNAP, sensory nerve action potential.
**The percentages shown in parentheses represent the frequency with which the sensory nerve fibers subserving the listed SNAPs traverse the different trunk and cord elements. The recording sites are shown in parentheses.*

dle finger arise from the C6, C7, and C8 DRG about 10%, 70%, and 20% of the time, respectively.[27] Thus, the median sensory NCS, recording from this finger, assesses the lateral cord in about 80% of instances and the medial cord in about 20% of instances. More proximally, it assesses the upper trunk and the C6 anterior primary ramus, spinal nerve, and DRG in 10% of instances; the middle trunk and the C7 anterior primary ramus, spinal nerve, and DRG in 70% of instances; and the lower trunk and the C8 anterior primary ramus, spinal nerve, and DRG in 20% of instances (Fig. 7).

The cell bodies of origin of the sensory nerve fibers assessed by the superficial radial sensory NCS reside in the C6 and C7 DRG about 60% and 40% of instances, respectively.[27] Thus, this study assesses the superficial radial nerve, radial nerve, and posterior cord consistently; the upper trunk and the C6 anterior primary ramus, spinal nerve, and DRG in about 60% of instances; and the middle trunk and the C7 anterior primary ramus, spinal nerve, and DRG in about 40% of instances (Fig. 8). Based on SNAP

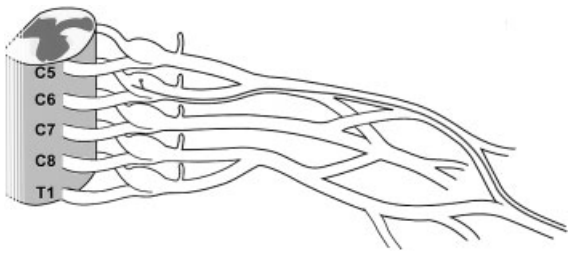

FIGURE 5. Proposed brachial plexus pathway for the sensory fibers assessed by the median SNAP recording from the thumb.

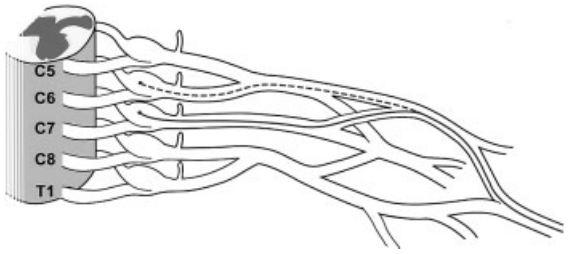

FIGURE 6. Proposed brachial plexus pathway for the sensory fibers assessed by the median SNAP recording from the index finger.

abnormalities noted among patients with plexopathies related to median sternotomy, the cell bodies of origin of the sensory fibers assessed by the ulnar sensory NCS, recording from the little finger—or from the dorsal aspect of the hand, as studied by the dorsal ulnar cutaneous (DUC) nerve—are primarily located in the C8 DRG.[27,84,96] Thus, these SNAPs always depend on the integrity of the ulnar nerve, medial cord, lower trunk, and the C8 anterior primary ramus, spinal nerve, and DRG (Fig. 9). Based on SNAP abnormalities noted among patients with true neurogenic TOS, as well as cadaver dissections, the cell bodies of origin of the sensory fibers assessed by the MABC sensory NCS reside predominantly in the T1 DRG.[27,37,84,96,117,138] Thus, this study assesses the MABC nerve, medial cord, lower trunk, and the T1 anterior primary ramus, spinal nerve, and DRG (Fig. 10). The incidence of SNAP abnormalities associated with individual trunk and cord lesions is shown in Table 3.

Electrodiagnostic Assessment of Individual Regions. Typically, lesions involving the brachial plexus do not affect all of its elements (i.e., they are regional) and, consequently, the entire plexus does not require exhaustive electrodiagnostic testing. Regarding the NCS, one approach is to screen the brachial plexus using just five sensory NCS (LABC; median recording from the thumb and index fingers; super-

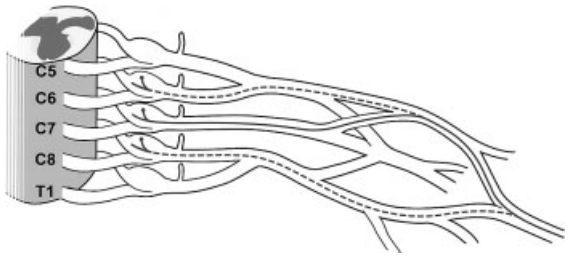

FIGURE 7. Proposed brachial plexus pathway for the sensory fibers assessed by the median SNAP recording from the middle finger.

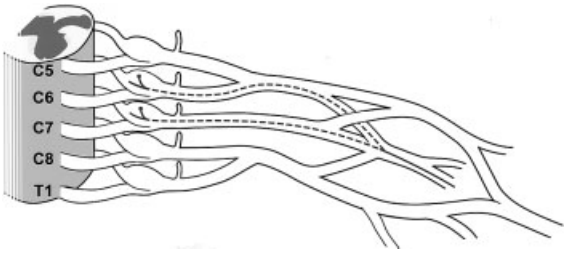

FIGURE 8. Proposed brachial plexus pathway for the sensory fibers assessed by the superficial radial SNAP.

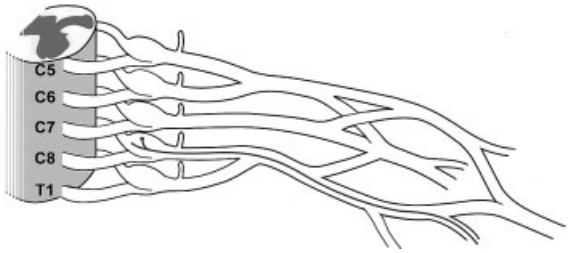

FIGURE 9. Proposed brachial plexus pathways for the sensory fibers assessed by the ulnar SNAP recording from the little finger.

ficial radial; and ulnar, recording from the little finger). Whenever a specific region of the brachial plexus requires assessment, additional sensory NCS, motor NCS, and NEE of muscles belonging to that particular region are added (see Tables 4 and 5).

Electrodiagnostic Assessment of the Supraclavicular Plexus. *Upper plexus.* The upper plexus contains nerve fibers from C5 and C6. Table 4 details its electrodiagnostic assessment. Regarding the sensory NCS, although no studies assess the C5 DRG or its postganglionic fibers, the other elements of the upper plexus are assessable. The median NCS, recording from the thumb, and the LABC NCS both reliably assess the C6 DRG, its postganglionic fibers, and the upper trunk. In general, upper plexopathies tend to affect these two studies equally. These studies may need to be performed contralaterally to identify relative abnormalities (i.e., side-to-side differences exceeding 50%). The superficial radial NCS and the median NCS, recording from the index finger, also assess these upper-plexus elements, albeit less reliably (i.e., in 60% and 20% of instances, respectively).[27] The musculocutaneous (recording biceps) and axillary (recording deltoid) motor NCS assess all of the upper-plexus elements. To avoid relative abnormalities, these studies are performed bilaterally in the presence of upper-plexus SNAP abnormalities or whenever the recorded CMAP values are near or below their lower limit of normal. NEE of the shoulder girdle, C5,6–radial, C5,6–axillary, and C6–median innervated muscles is helpful, and evaluation of levator scapulae, rhomboids, serratus anterior, and spinati muscles helps to define the proximal extent of the lesion.

Middle plexus. The middle plexus (Table 4) contains nerve fibers from C7. The sensory nerve fibers subserving the median NCS, recording from the index and middle fingers, traverse the middle plexus in approximately 80% and 70% of instances, respectively; whereas those subserving the superficial radial NCS traverse it in 40% of instances.[27] Contralateral studies help identify relative abnormalities. A radial motor NCS, recording from extensor digitorum communis (EDC) or anconeus, can be added, though neither assesses solely the middle plexus. NEE of selected muscles (Table 4) is useful. Since isolated middle plexopathies are rare,[1,27,75] their identification should always prompt screening of the adjacent upper and lower plexuses.

Lower plexus. The lower plexus (Table 4) contains fibers from C8 and T1. The ulnar sensory NCS, recording from the little finger, assesses the C8 DRG, its postganglionic fibers, and the lower trunk. The MABC study assesses the corresponding T1 structures. Thus, these two studies are complementary at the pre-trunk level. Typically, with lower-trunk lesions, both are equally affected, whereas their involvement is more discordant with more proximally situated lesions. The DUC sensory NCS typically is superfluous, since it assesses the same brachial plexus elements as the ulnar study.[27] The ulnar [recording from the abductor digiti minimi (ADM)] and median [recording from the abductor pollicis brevis (APB)] motor NCS assess the lower plexus, as does the radial motor NCS [recording from the extensor indicis proprius (EIP)]. Although the latter may be spared with partial lower-trunk lesions, its involvement excludes a medial cord lesion. These three motor NCS assess the pre-trunk level of the lower plexus differentially—the radial NCS assesses solely the C8 root; the ulnar, the C8 root predominantly; and the median, almost solely the T1

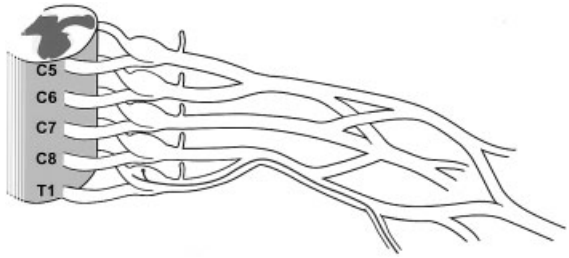

FIGURE 10. Proposed brachial plexus pathway for the sensory fibers assessed by the MABC SNAP.

Table 4. Electrodiagnostic assessment of the supraclavicular plexus.*

Upper plexus	
Sensory NCS	Needle electrode examination
LABC	Spinati muscles
Median (thumb)	Deltoid
Superficial radial	Biceps
Median (index finger)	Brachioradialis
	Pronator teres
Motor NCS	Extensor carpi radialis
Axillary (deltoid)	Triceps (lateral head)
Musculocutaneous (biceps)	
Radial (extensor digitorum communis or anconeus)	
Middle plexus	
Sensory NCS	Needle electrode examination
Median (index finger)	Triceps (lateral head)
Median (middle finger)	Anconeus
Superficial radial	Pronator teres
	Flexor carpi radialis
Motor NCS	
Radial (anconeus)	
Lower plexus	
Sensory NCS	Needle electrode examination
Ulnar (little finger)	Abductor pollicis brevis
Dorsal ulnar cutaneous	Flexor pollicis longus
MABC	First dorsal interosseous
Ulnar (ring finger)	Adductor pollicis
	Abductor digiti minimi
Motor NCS	Flexor carpi ulnaris
Ulnar (abductor digiti minimi)	Flexor digitorum profundus 4,5
Ulnar (first dorsal interosseous)	Extensor indicis proprius
Median (abductor pollicis brevis)	Extensor pollicis brevis
Radial (extensor indicis proprius)	

LABC, lateral antebrachial cutaneous; MABC, medial antebrachial cutaneous; NCS, nerve conduction study.
**The entire muscle domain of the individual supraclavicular plexus regions is not shown; only those muscles considered most helpful are included. Other helpful upper-plexus muscles include the serratus anterior, rhomboids, teres minor, brachialis, flexor carpi radialis, and, to a lesser degree, pectoralis major and levator scapulae. Other helpful middle-plexus muscles include the extensor digitorum communis, extensor carpi ulnaris, and extensor carpi radialis; lower-plexus muscles include the extensor carpi ulnaris, extensor digitorum communis, pronator quadratus, and pectoralis minor. The recording sites are shown in parentheses.*

root.[27,78] Although the ulnar-elicited CMAP from the first dorsal interosseous (FDI) reflects the same lower-plexus elements as that from ADM, lower plexopathies may affect these two CMAPs differently and, thus, both often are required. With lower-plexus SNAP abnormalities, CMAPs should be recorded bilaterally. On NEE, it is useful to study muscles innervated via C8,T1–median, C8,T1–ulnar, and C8–radial motor nerve fibers.

Electrodiagnostic Assessment of the Infraclavicular Plexus. *Lateral cord.* Typically, lateral cord lesions affect the three median and LABC SNAPs uniformly (Table 5). Although upper-trunk lesions may also affect these four SNAPs, only about 1 in 50 (2%) simultaneously involves all four studies and, when this occurs, their degree of involvement tends to be dissimilar—the median recording from the thumb and LABC SNAPs are affected to a greater extent than the median SNAPs recorded from the index and middle fingers.[27] On motor NCS, the musculocutaneous CMAP may be abnormal, but the axillary CMAP is spared. On NEE, abnormalities are restricted to muscles innervated via musculocutaneous and C6,7–median nerve fibers. Muscles innervated by C5,6–radial and –axillary nerve fibers and by nerve derived from the anterior primary rami (long thoracic and dorsal scapular nerves) and upper trunk (suprascapular nerve) help to differentiate an upper plexopathy.

Posterior cord. The only sensory NCS assessing this element is the superficial radial NCS (Table 5). On motor NCS, the axillary CMAP and one of the radial CMAPs are recorded. On NEE, muscles inner-

Table 5. Electrodiagnostic assessment of the cords.*

Lateral cord	
Sensory NCS	Needle electrode examination
LABC	Biceps
Median (thumb)	Brachialis
Median (index finger)	Pronator teres
Median (middle finger)	Flexor carpi radialis
Motor NCS	
Musculocutaneous (biceps)	
Posterior cord	
Sensory NCS	Needle electrode examination
Superficial radial	Deltoid
Motor NCS	Triceps (lateral head)
Axillary (deltoid)	Anconeus
Radial (extensor digitorum communis)	Brachioradialis
	Extensor carpi radialis
Radial (extensor indicis proprius)	Extensor digitorum communis
	Extensor indicis proprius
Radial (anconeus)	Extensor pollicis brevis
Medial cord	
Sensory NCS	Needle electrode examination
Ulnar (little finger)	Abductor pollicis brevis
Dorsal ulnar cutaneous	Flexor pollicis longus
MABC	First dorsal interosseous
Ulnar (ring finger)	Adductor pollicis
Motor NCS	Abductor digiti minimi
Ulnar (abductor digiti minimi)	Flexor digitorum profundus 4,5
Ulnar (first dorsal interosseous)	Flexor carpi ulnaris
Median (abductor pollicis brevis)	

LABC, lateral antebrachial cutaneous; MABC, medial antebrachial cutaneous; NCS, nerve conduction study.
**The muscle domains of the posterior and medial cords are not shown in their entirety. Rather, only those muscles considered most helpful are listed. Other helpful posterior-cord muscles include the latissimus dorsi, extensor carpi ulnaris, and teres minor. The pronator quadratus is a useful assessor of the medial cord. The recording sites are shown in parentheses.*

vated via axillary, radial, and thoracodorsal nerve fibers are helpful. To differentiate a middle plexus lesion, median SNAPs from the index and middle fingers and NEE of muscles innervated via C6,7–median nerve fibers are included.

Medial cord. Ulnar and MABC SNAPs and ulnar and median CMAPs are recorded (Table 5). The radial CMAP, recording from the EIP, is used to screen for a more proximal process. NEE of muscles innervated via C8,T1–median and C8,T1–ulnar nerve fibers is helpful. To screen for a more proximal process, muscles innervated via C8–radial nerve fibers [EIP; extensor pollicis brevis (EPB)] are added. Importantly, their normalcy does not exclude a more proximal process, as partial lower-trunk lesions may spare them.

Terminal nerves. Reliable sensory and motor NCS are available to assess the median, radial, ulnar, musculocutaneous, and axillary nerves. On NEE, muscles innervated by these nerves are sampled. The lack of proximal branches emanating from these terminal nerves means that it is impossible, by clinical or electrodiagnostic means, to differentiate a terminal nerve lesion of the brachial plexus from a more distal, proximally located peripheral nerve lesion.

Concluding Remarks. Typically, the five screening sensory NCS listed in the introduction to this section are generally required, with additional studies based on the particular region under study or on any identified SNAP abnormalities. Patients with a flail arm due to diffuse involvement of the distal supraclavicular plexus (pan-trunk plexopathy) or diffuse involvement of the proximal infraclavicular plexus (pan-cord plexopathy) can be difficult to differentiate by electrodiagnostic studies. Regarding the sensory and motor NCS, only an abnormal suprascapular motor NCS can identify a diffuse supraclavicular plexopathy. When normal, this study does not distinguish a distal supraclavicular plexus lesion (since the suprascapular nerve leaves the upper trunk proximally) from a diffuse infraclavicular one. Although NEE of the muscles innervated by the dorsal scapular, long thoracic, and suprascapular nerves can be helpful, when normal the same distal pan-trunk versus pan-cord localization dilemma remains. In this setting, examination of the pectoralis major muscle can be helpful, since it receives its innervation via motor nerve fibers exiting from the infraclavicular plexus so proximally that it tends to be affected by supraclavicular plexopathies and spared by infraclavicular ones. Clinically, diffuse supraclavicular plexopathies are much more common than diffuse infraclavicular ones. In one report of 78 patients with flail arms, 75 were due to diffuse supraclavicular lesions; only 3 reflected a diffuse infraclavicular plexopathy.[5]

SELECTED SITE-SPECIFIC DISORDERS OF THE BRACHIAL PLEXUS

Supraclavicular Plexopathies with Regional Predilections. ***Upper Plexus.*** The most commonly injured brachial plexus region is the upper plexus, usually from closed traction.[144,150] Brachial plexopathies with a predilection for the upper plexus include burner syndrome, rucksack paralysis, and classic postoperative paralysis.

Burner syndrome. When sudden, forceful shoulder contact produces separation of the shoulder and head, upper plexus traction may occur; if associated with pain and paresthesias, the term *burner* or *stinger* is applied. As expected, these injuries are more common among males involved in contact sports and are the most common of all sports-related injuries. In one report, they accounted for 38% of 190 sports-related injuries.[69] In general, the pain is abrupt in onset, sharp and burning in quality, and, with the paresthesias, extends distally into the upper extremity, often to the thumb. These symptoms usually persist for a few minutes, longer in the presence of weakness. Their distribution implicates the C6 nerve fibers, but whether they are affected at a pre- or postganglionic level is debated. Although some players experience numerous burners, permanent neurologic dysfunction is rare. Whenever the symptoms are prolonged, electrodiagnostic assessment is indicated. With burners, the electrodiagnostic abnormalities, when present, typically are limited to sparse fibrillation potentials in an upper-plexus distribution.

Rucksack paralysis (cadet palsy; pack palsy). This typically unilateral upper plexopathy usually presents with painless weakness following or during the course of wearing a rucksack or similar device (e.g., backpack, child-carrying harness). Sensory involvement, in the same distribution, often is present. Its pathogenesis is likely nerve fiber compression related to direct pressure from the rucksack. Risk factors include the weight of the load transported, characteristics of the device itself (i.e., the presence of a metal frame or waist belt), and the duration worn.[20] It may be more common in the presence of an underlying abnormality (e.g., vertebral anomaly, cervical rib) or previous local injury.[155] A history of transient weakness following rucksack usage may be elicited.[144] In two-thirds of patients, the lesions are predominantly demyelinating conduction block.[144]

In this setting, the sensory NCS are normal and lesion localization and severity are determined by the motor NCS and NEE. As expected with a demyelinating process, treatment is conservative and recovery occurs within a few months. In the one-third in whom axon loss predominates, recovery is more prolonged and may be incomplete.

Classic postoperative paralysis. Classic postoperative paralysis, which was initially described in 1894, is a traction or pressure injury that characteristically presents in the immediate postoperative setting as a unilateral upper plexopathy or, much less frequently, as a more diffuse supraclavicular plexopathy that involves the upper plexus disproportionately.[144] When involved, the middle and lower plexuses recover quicker, leaving an isolated upper plexopathy. Clinically, the primary complaint is painless weakness; paresthesias also may be noted. This entity is related to multiple factors, including patient positioning, loss of muscle tone from anesthesia, and unconsciousness, which blocks weight-shifting ability. Predisposing factors include the Trendelenburg position; upper-extremity abduction beyond 90 degrees; arm board restraint in an abducted, extended, and externally rotated position; and contralateral deviation and rotation of the head.[146] There is no gender or age group susceptibility. The underlying pathophysiology typically is demyelinating conduction block; much less frequently, axon loss predominates.[144,146] Thus, SNAPs assessing the upper plexus (Table 4) usually are normal unless there is concomitant axon loss. CMAP amplitude discrepancies between axillary (normal) and supraclavicular fossa (absent or low amplitude) stimulation sites indicate a demyelinating conduction-block along upper-plexus fibers (musculocutaneous, recording from biceps; radial, recording from brachioradialis). An absent or low-amplitude axillary CMAP is seen with supraclavicular fossa stimulation but, since this nerve cannot be stimulated infraclavicularly, an amplitude discrepancy cannot be sought. With demyelinating conduction block lesions, rapid and full recovery is expected and, thus, conservative treatment is employed.[144,146,150]

Middle Plexus. Isolated middle plexopathies are rare.[1,27,75] In one review of 417 brachial plexopathies, only one (surgically verified) was noted.[27] More typically, middle-plexus involvement occurs with concomitant upper or lower plexus involvement. Similar to upper plexopathies, the most common cause of middle plexopathies is closed traction.[144]

Lower Plexus. Among supraclavicular plexopathies, lower plexopathies are less common than upper plexopathies.[29] Disorders with a predilection for the lower plexus include true neurogenic TOS, following surgery for disputed neurogenic TOS, postmedian sternotomy brachial plexopathy, and Pancoast syndrome. The percentage of closed traction injuries involving the lower plexus is less than with upper and middle plexopathies.[150] Avulsion injuries are more common in this region of the supraclavicular plexus.

True neurogenic thoracic outlet syndrome. The brachial plexus and subclavian vessels traverse the thoracic inlet, which lies between the first rib and clavicle and is commonly referred to as the thoracic outlet. When one of these structures is compressed in that space, the general term TOS is applied. More specific terms include arterial TOS, venous TOS, and neurogenic TOS. The latter is divided into true neurogenic and disputed neurogenic TOS. True neurogenic TOS, which is more common among younger women and has an incidence of approximately 1 per million, is also called the cervical rib and band syndrome because the affected C8 and T1 fibers of the lower plexus are stretched and angulated by a taut band that extends from a rudimentary cervical rib or elongated C7 transverse process to the first rib.[37] Since the T1 fibers lie below the C8 fibers, they are deflected to a greater extent and, consequently, sustain greater injury.[37,38] This has both clinical and electrodiagnostic ramifications. Clinically, patients present with T1 more than C8 weakness and thenar muscle wasting, as well as paresthesias and pain along the medial aspects of the arm, forearm, and hand. Likewise, the MABC SNAP and median CMAP (i.e., studies that primarily assess T1 fibers) are more affected than the ulnar SNAP and CMAPs (i.e., studies that primarily assess C8 fibers). The NEE indicates a slowly progressive axon-loss process with a lower-plexus distribution that is most pronounced in the APB muscle. This pattern of clinical and electrodiagnostic abnormalities is essentially pathognomonic for true neurogenic TOS.[27] Although radiographic studies visualize associated bony changes, they do not visualize the band. Surgical division of the band typically relieves the pain and paresthesias and arrests the muscle weakness and wasting.[38,150] A single case with manifestations similar to true neurogenic TOS was reported in a competitive swimmer in whom the lower trunk was compressed by a fibrous band located within a hypertrophied scalene muscle.[59]

Unlike true neurogenic TOS, which has a clear pathogenesis, objective clinical and electrodiagnostic features, and a good response to surgical intervention, disputed neurogenic TOS has an unclear

pathogenesis, lacks objective clinical and electrodiagnostic features, and does not reliably respond to surgical intervention.[143] Although some of its proponents believe that disputed neurogenic TOS is a common and underdiagnosed disorder,[109,110] many physicians do not even consider it a distinct entity. Oddly, among 174 patients in Colorado undergoing surgery for TOS in 1989, almost all of them had either private insurance or worker's compensation; Medicaid patients almost never underwent surgery.[13]

Postoperative disputed neurogenic thoracic outlet syndrome. When patients with disputed neurogenic TOS are treated surgically, especially by transaxillary first rib resection, some develop severe brachial plexopathies, a condition termed postoperative disputed neurogenic TOS.[142] Most of these are incomplete, axon-loss lower plexopathies, though more extensive damage may occur. These patients often present with severe causalgic hand pain and clinical deficits in the distribution of the affected brachial plexus elements. Although the pain may improve after surgical repair of the surgically traumatized elements, the hand weakness typically does not do so.[14,66,81,141,142,146,147,150]

Postmedian sternotomy plexopathy. This term applies to brachial plexopathies following operations requiring median sternotomy, the most common of which is coronary artery bypass surgery.[72] The clinical and electrodiagnostic manifestations of this type of plexopathy suggest C8 anterior primary ramus involvement; less commonly, adjacent elements also are affected. Postulated etiologies incriminate the first thoracic rib. Either chest wall retraction (1) pushes the clavicle into the retroclavicular space, rotating the first rib into the C8 anterior primary ramus, or (2) fractures the first rib and the fractured segment impinges upon the C8 anterior primary ramus.[64,78,136] In either case, a lower-plexus traction injury results that, by clinical and electrodiagnostic assessments, affects the C8 anterior primary ramus disproportionately. Although this ramus contains motor fibers destined for the median, radial, and ulnar nerves, its sensory fibers are destined solely for the ulnar nerve. Thus, the associated paresthesias suggest an ulnar neuropathy. Misdiagnosis is avoided by clinical assessment of muscles supplied by C8–median nerve (e.g., flexor pollicis longus) and C8–radial nerve fibers (e.g., EIP, EPB). On sensory NCS, an absent or low-amplitude ulnar SNAP recording from the little finger and a normal MABC SNAP usually are seen, implying an ulnar neuropathy or a ganglionic or postganglionic C8 root lesion. (Although a fascicular process cannot be excluded, the normal MABC SNAP argues against a lower trunk or medial cord process, since lesions at these two sites tend to affect these two SNAPS more uniformly.) On motor NCS, isolated ulnar CMAP abnormalities may be seen, accompanied infrequently by radial or median CMAP abnormalities (recording from EIP and APB, respectively). Thus, localization typically rests on the NEE. When present, abnormalities in muscles supplied by C8–median nerve fibers indicate that the lesion lies at or proximal to the medial cord, whereas abnormalities in muscles innervated by C8–radial nerve fibers place it at or proximal to the lower trunk. Unless significant axon loss involves the dominant hand or causalgic pain develops, permanent disability is unexpected and, thus, conservative treatment usually is employed.[29,41,54,78,136,137,144,150]

Pancoast syndrome. Since only the pleura separates the lung from the T1 anterior primary ramus and lower trunk, lung diseases may involve the lower plexus. In 1924, Pancoast described the direct extension of cancer from the lung apex to the lower plexus.[98,99] This syndrome occurs in about 3% of lung cancer patients and, thus, is most frequently observed among men with a heavy smoking history.[104] Pancoast syndrome may also be observed among patients with lower plexopathies related to other tumors (both benign and malignant), tumor recurrences, and infectious or inflammatory disorders.[51,150] With lung cancer, shoulder-region pain typically is the initial and most pronounced symptom. It may reflect pleural, rib, spinal column, or brachial plexus involvement. Interscapular pain may be present when the cancer involves the posterior primary rami.[51] The shoulder pain tends to be burning or boring in character, worse at night, and tends to radiate along the medial aspect of the arm to the elbow, and, less commonly, to the 4th and 5th digits.[51] When present, clinical deficits are in a lower plexus distribution. When the cancer involves the T1 root or the inferior cervical sympathetic ganglion, a Horner's syndrome may appear. Pancoast syndrome is often the first manifestation of the neoplasm, commonly a non–small cell carcinoma; early recognition and treatment are associated with a higher cure rate.[71] Electrodiagnostic studies can localize the process, thereby directing imaging studies. With severe pain, narcotics and radiation therapy may be required.[68]

Other Supraclavicular Plexopathies. *Avulsions.* Rootlets are not surrounded by connective tissue and, hence, are easily avulsed from the spinal cord by stretch (traction). Since the torn roots cannot regenerate or be surgically repaired, avulsions represent the most serious complication of traction injuries.

The ventral roots are more easily avulsed because they are of lesser caliber, have thinner dural sacs, and are more dispersed along the spinal cord than the dorsal roots.[124] Regarding the brachial plexus, the lower two roots more commonly avulse, whereas the upper two roots more commonly rupture extraforaminally. This reflects anatomic differences in their proximal anchorage sites, angles of exit from the intervertebral foramina, and lengths. The C5–T1 spinal nerves traverse grooves in their respective transverse processes that lie between the intertransversalis muscles. Since the C5 and C6, and variably the C7, spinal nerves are securely anchored by fascia at this point, their anchorage sites are extraforaminal, whereas the C8 and T1 spinal nerves are anchored at the spinal cord.[125] In addition, the oblique course of the upper cervical roots makes them more likely to tear extraforaminally than to avulse, whereas the short length of the T1 root renders it more susceptible to avulsion.[1] Upper-extremity position at the time the traction force is applied also plays a role. The C5–C6 fibers are most susceptible with the upper extremity alongside the torso, the C7 fibers when it is oriented parallel to the floor, and the C8–T1 fibers when it is in an above-shoulder position. Strong enough traction forces avulse all roots, regardless of limb position. Ruptures may be incomplete, with one or more fascicles remaining in continuity. Approximately 15% of supraclavicular lesions are two-level processes (preganglionic and postganglionic), especially with upper cervical root involvement.[1] Concomitant axillary, musculocutaneous, and suprascapular nerve injuries may occur at their anchorage sites (i.e., quadrangular space, coracobrachialis, and suprascapular or spinoglenoid notch, respectively). When the entire upper extremity is paralyzed, including the long thoracic, dorsal scapular, thoracodorsal, and pectoral nerve–innervated muscles, especially in the presence of a Horner's syndrome, complete avulsion is likely. Other indicators of possible avulsion include bony injury, especially a transverse process fracture (spinal nerve anchorage site); long-tract signs (damage severe enough to injure the spinal cord); and severe burning pain, with shooting pain in the anesthetic area.[155] Although extraforaminal ruptures may be amenable to surgical repair, root avulsions are not. Moreover, most avulsion injuries are associated with severe pain (especially hand pain), the incidence of which increases with the number of avulsed nerve roots.[148]

Obstetric Brachial Plexopathy. Obstetric brachial plexopathy follows a type of traction injury that typically occurs when shoulder dystocia impedes vertex delivery, thereby prompting excessive lateral deviation of the head and neck in order to free the shoulder.[21,135] When this plexopathy follows a breech delivery, the risk of avulsion (usually of the C5 and C6 roots; less frequently of the C5–C7 roots) and bilateral involvement (22% in one series) is increased.[36] The fact that this type of plexopathy also follows deliveries by cesarean section implies that it does not simply follow poorly performed deliveries.[36,57,58,133,135,146] Reported risk factors include infantile macrosomia (common with maternal diabetes), short mothers, low or midforceps delivery, vacuum extraction, second-stage labor exceeding 60 min, passive head rotation with the shoulders fixed, multiparity, ethnic background, and delivery of a previous infant with an obstetric brachial plexopathy; fetal growth restriction and prematurity are considered protective.[20,21,79,135,153] The incidence of this type of plexopathy ranges from 0.5–2.6 per 1,000 full-term live births [135] and reportedly is declining.[20] Five patterns of nerve fiber involvement have been described: (1) C5–C6 (Erb's palsy; about 50%); (2) C5–C7 (Erb's-plus palsy; waiter's tip position, with adduction and internal rotation of arm, extension and pronation of forearm, and flexion of wrists and fingers; about 35%); (3) C5–T1 with some finger flexion sparing; (4) C5–T1 with flail arm and Horner's syndrome; and (5) C8–T1 with isolated paralysis of the hand and Horner's syndrome (Klumpke's palsy; almost never seen).[135] Concomitant postganglionic lesions are more common with injuries involving the C5–C7 fibers (anchored extraforaminally), whereas avulsion is more common with lesions involving the C8–T1 fibers (spinal cord anchoring).[119] Although obstetric brachial plexopathy was first described in 1764, its management remains controversial.[87] These lesions range from mixed demyelination and axonal to pure axon loss (avulsion) but, in general, are less severe than traction injuries occurring among adults. Although many reviews suggest that some spontaneous recovery occurs in over 90% of instances, its natural history is unknown.[135] Two Swedish studies, in which surgical intervention was not employed, reported that 20 to 25% of patients are significantly impaired in later life.[3,116,135] Unfortunately, neither clinical nor electrodiagnostic assessments can unequivocally identify this latter group. Hence, watchful waiting for evidence of recovery usually is employed, though the duration of such an approach is controversial. Since surgical repair yields the best results when performed within the 1st year, the observation period usually ranges from 3 to 9 months, or slightly longer.[119] During this time, physical therapy is employed. As expected, the

prognosis for C8–T1 nerve fiber involvement is poor due to the greater incidence of avulsion injury.

Infraclavicular Plexopathies with Regional Predilections. Disorders of the infraclavicular plexus have much less regional proclivity. At the cord level, radiation directed at the axillary lymph nodes (mostly women with breast cancer) tends to involve the infraclavicular plexus, especially the lateral cord, whereas midshaft clavicular fractures more commonly affect the medial cord.[18,24,60,88] At the terminal nerve level, the median terminal nerve characteristically is first and most affected with medial brachial fascial compartment syndrome, and the radial terminal nerve is more frequently affected with crutch palsies. Of the five terminal nerves, the musculocutaneous terminal nerve is more often affected by operative procedures to correct recurrent anterior shoulder dislocation or by other procedures performed near the coracoid process.[12,32,150] Glenohumeral dislocations and proximal humeral fractures most commonly involve the axillary terminal nerve, because of the short distance between its anchorage site and its point of origin,[76] yet the nerve involvement frequently goes unnoticed. In one electrodiagnostic study, performed 6 weeks after dislocation, 35 of 65 (55%) patients had axillary terminal nerve involvement, 8 of whom had a normal clinical examination.[128] Suprascapular neuropathies are less frequent and, when present, often coexist with axillary neuropathies; musculocutaneous neuropathies are least common.[148] These traction injuries range from focal demyelination to total axon loss (e.g., nerve rupture, avulsion from the brachial plexus).

Other Infraclavicular Plexopathies. Staal et al. first described medial brachial fascial compartment syndrome in 1966, and it has recently been reviewed.[120,132] The medial brachial fascial compartment, which extends from the clavicle to the elbow and houses the terminal nerves of the brachial plexus and the axillary vessels, is formed by the medial intermuscular septum, medially, when it divides into two fascial extensions that extend to the brachial fascia that surrounds the arm. The five terminal nerves of the brachial plexus exit from this compartment in the following order: musculocutaneous, axillary, radial, ulnar, and median. The median nerve, characteristically, is affected first, most severely, and most often in isolation. Isolated ulnar or radial neuropathies are rare, and other mononeuropathies have not been described.[118,132] When two nerves are involved, the median and ulnar nerves are the most common combination.[132] The radial, axillary, and musculocutaneous nerves are affected less frequently. Reportedly, lesions located within the compartment (e.g., hematomas, aneurysms, pseudoaneurysms, and other lesions with mass effect) cause the intracompartmental pressure to rise, thereby impeding nerve fiber microcirculation and inducing clinical dysfunction. If the pressure increment were uniform, it should affect intracompartmental nerves uniformly, whereas with this syndrome, the median nerve is affected disproportionately. However, compartment syndromes associated with fractures are associated with pressure gradients that are greatest near the fracture site,[48] and nerves located near a hematoma may be exposed to greater pressures.[42] Regarding axillary arteriograms, since the median and ulnar nerves lie near the axillary artery at the point of cannulation,[97] the occurrence of a hematoma involves these two nerves out of proportion to more distant ones, and the high pressures associated with the hematoma may account for the high incidence of these neuropathies. Clinically, patients present with pain or paresthesias in the distribution of the affected nerves, followed shortly thereafter by weakness in a similar or wider distribution. Without prompt surgical intervention, the likelihood of recovery is poor. Thus, these lesions must be recognized early and decompressed urgently.[40,115,118,120,132,145,157] A recent study reported that complete recovery was 8.3 times more likely when surgical exploration occurred within the first 4 h of symptom onset.[15] Electrodiagnostic testing, although useful for localizing and characterizing neuropathies resulting from this syndrome, has no role in the acute setting.

SELECTED SITE-NONSPECIFIC BRACHIAL PLEXOPATHIES

Neuralgic Amyotrophy (Parsonage-Turner Syndrome). Although many names have been coined for this disorder, the term neuralgic amyotrophy (NA) conveys its two quintessential features and thus is preferred. Since NA has a predilection for predominantly motor nerves, especially more proximally located ones, it frequently involves the long thoracic, suprascapular, and axillary nerves. The anterior interosseous and musculocutaneous nerves, and nerve branches to individual muscles (e.g., infraspinatus, pronator teres) also are frequently affected. The phrenic and medullary cranial nerves (especially the spinal accessory), as well as individual nerve roots, may be involved.[17,55,91,103,112] Proximally, the nerve fibers have a somatotopic arrangement. Therefore, although NA most commonly presents as a mono-

neuropathy or a multiple mononeuropathy, this may sometimes reflect a proximal lesion within the brachial plexus.[122] Within the brachial plexus, the upper trunk is most commonly affected.[23] When biceps weakness is associated with musculocutaneous CMAP and LABC SNAP abnormalities and the median SNAP recorded from the thumb is normal, a musculocutaneous neuropathy is more likely than an upper plexopathy.[27] Conversely, when abnormal LABC and median (recording from the thumb) SNAPs are recorded and a median neuropathy is excluded (by normal median SNAPs from the index and middle fingers and a normal median CMAP), an upper plexopathy is more likely. Bilateral NA may be simultaneous or sequential and either symmetric or asymmetric. When it recurs in a previously affected limb, it may involve the same or different nerves.[144,148]

Clinically, abrupt and excruciating shoulder or upper-extremity pain, often with a nocturnal onset, is the presenting feature. Most commonly, the pain is located at the lateral aspect of the shoulder or in the periscapular region, but its location varies with the involved nerve and can be most pronounced at the shoulder (axillary nerve), scapula (suprascapular nerve), lateral thorax (long thoracic), antecubital fossa (anterior interosseous nerve), or lateral arm and forearm (musculocutaneous nerve). Although the pain may extend proximally or distally, shoulder movement rather than neck movement intensifies it. When its nature is unrecognized, unnecessary procedures may be performed.[6] The severe pain typically abates after 7–10 days or is replaced by a more persistent dull ache. At this point, true weakness becomes apparent, as may significant muscle wasting. About 50% of affected individuals report antecedent events, such as recent infection, unaccustomed exertion, childbirth, trauma, or an invasive medical or dental procedure. Although this triad (antecedent event, severe pain, and weakness and wasting) generally is observed, considerable individual variation exists.[22] Sensory NCS help to localize the lesion. The motor NCS define the severity of the affected nerves and thus are useful as baseline prognosticators and for subsequent comparative measurements. Consistent with an axon-loss process, the NEE shows findings indicative of acute and chronic motor axon loss, the combination of which reflects the timing of the study.[101,105,144] Rarely, early in its course, demyelinating conduction block may predominate, as evidenced by full and rapid recovery from severe weakness or by the electrodiagnostic study itself.[139] Tsairis et al. reported recovery rates of 36% by 1 year, 75% by 2 years, and 89% by 3 years.[131] Recovery reflects lesion severity, lesion location, and the degree of connective tissue involvement, and is best determined by serial clinical and electrodiagnostic assessments. Analgesics, including narcotics, may be required for the initial pain. At that time, a short course of corticosteroids may be helpful.[74] With chronic pain, neuropathic pain medications (e.g., gabapentin, tricyclics) are added. Strengthening and stretching exercises are indicated. Unlike sporadic NA, the extremely rare familial form, which has been localized to chromosome 17, is associated with dysmorphic features (e.g., hypotelorism, high-arched palate, syndactyly) and commonly recurs.[19,134,140,152]

Primary Neoplastic Brachial Plexopathies. Neoplastic brachial plexopathies can be divided into primary (of brachial plexus origin) or secondary (originating outside the plexus). Primary brachial plexus tumors are rare and usually benign. Of these, nerve sheath tumors predominate. Most are solitary schwannomas or neurofibromas involving the upper or middle plexus, proximally.[148] Solitary schwannomas are slow-growing, encapsulated tumors that, at the root level, more commonly affect the sensory roots. When they grow through the neural foramen and expand at both ends, they appear dumbbell-shaped.[43] Most patients present with a painless mass and may have paresthesias, sometimes exacerbated by motion or palpation. Motor symptoms follow ventral root or spinal cord compression. On MR imaging, these lesions appear elliptical or spherical, isointense to muscle on T1 and hyperintense on T2, brightly enhance, and are often associated with entering, exiting, or displaced fascicles.[80] Pathologically, they arise from a single fascicle (plexiform schwannomas arise from multiple fascicles) and are thickly encapsulated. Once enucleated, they seldom recur. Solitary intraneural neurofibromas are benign, slowly growing, nonencapsulated tumors that originate from the neural sheath. Following excision, recurrence is unusual, even when incomplete.[43] When these tumors occur as part of neurofibromatosis type 1, they do not have a regional predilection and more frequently are multiple and plexiform, recur following excision, and, like malignant nerve sheath tumors, present with pain or clinical deficits.[119,150] On MR imaging, they appear fusiform or plexiform in shape, isointense to muscle on T1 and hyperintense on T2, and enhance. Unlike schwannomas, displaced fascicles are rare.[80]

Malignant nerve sheath tumors arise de novo or via malignant transformation—usually from a plexiform neurofibroma, less commonly from a solitary intraneural neurofibroma, and rarely from a schwan-

noma.[100] Patients often present with painful, enlarging masses associated with appropriately distributed clinical deficits. Their highly malignant nature is reflected by their 5-year survival rate (10 to 50%).[100] On MR imaging, these tumors are less circumscribed and may be observed to extend along fascial planes.[80] Complementary studies include CT scans, angiograms, and myelograms.[80,100] In the future, MR neurography may be helpful in demonstrating these lesions.[85]

Secondary Neoplastic Brachial Plexopathies. Neoplastic processes that involve the brachial plexus secondarily (usually breast or lung cancers) do so by means of extrinsic compression or infiltration from adjacent structures or spread from distant sites (metastases). When cancer involves the axillary lymph nodes, it may infiltrate the medial cord or nearby nerves (medial brachial cutaneous, MABC, ulnar, or median). Most patients present with severe and persistent shoulder and upper-extremity pain, followed by appropriate clinical deficits. With sympathetic involvement, the upper extremity may become warm and dry and a Horner's syndrome sometimes develops. With spread throughout the brachial plexus, symptom distribution increases.[68,71,104] Although plain films, bone scans, and CT-myelography (when epidural metastases are present) may demonstrate evidence of malignancy or metastatic disease, MR imaging is the radiographic procedure of choice for evaluating neoplastic plexopathies. Through lesion localization, electrodiagnostic testing can direct these studies. Since neoplastic processes frequently invade the brachial plexus from below, electrodiagnostic assessment of T1 fibers is mandatory. Initially, MABC SNAP abnormalities may be the only electrodiagnostic manifestation of T1 sensory fiber infiltration.[114]

Radiation-Induced Brachial Plexopathy. Although the PNS is relatively resistant to radiation damage, the incidence of damage increases with higher total dose, larger fraction sizes, and application times of shorter duration. Thus, lower doses administered over longer periods are safer.[43,77,123,155] The first cases of radiation-induced brachial plexopathy were reported in 1964.[92] These plexopathies are more often observed among women with breast cancer who received axillary lymph node chain radiation therapy months to decades earlier.[126] Radiation-induced brachial plexopathies typically are painless lesions that usually present with paresthesias involving one or more of the lateral three digits (i.e., lateral cord distribution), followed by extension of the paresthesias and, later, weakness.[151] Typically, these lesions are relentlessly progressive.[39] Because the paresthesias involve the lateral digits, carpal tunnel syndrome may be suspected unless electrodiagnostic testing is performed. When related to radiation, the initial electrodiagnostic manifestations typically include demyelinating conduction block on motor NCS, and myokymic discharges and fasciculation potentials on NEE.[25,39,68,73] The paraspinal muscles are studied, especially if tumor recurrence is being considered, because paraspinal fibrillation potentials are more common with radiation therapy than tumor recurrence.[44] Unlike most demyelinating conduction-block lesions, those associated with radiation therapy are prolonged and typically convert to axon loss, with electrodiagnostic features that reflect the timing of the study and the severity of the loss. As axon loss progresses, the limb eventually becomes nonfunctional, accounting for the dismal prognosis of these lesions.[151] The delayed effects of radiation therapy likely reflect ischemic damage related to microcirculatory impairment from radiation-induced fibrosis.[20] Rarely, radiation therapy generates nerve sheath tumors, typically malignant ones.[33]

It is important to distinguish neoplastic from radiation-induced plexopathy. Clinically, with neoplastic plexopathies, the incidence of pain at onset and Horner's syndrome is much higher, whereas with radiation plexopathies, isolated paresthesias predominate.[44,68,73] Although the electrodiagnostic abnormalities typical of radiation therapy may be observed, their presence does not exclude concomitant tumor recurrence. On MR imaging, nodular enhancement is highly suggestive of a neoplastic process,[100] as is the presence of a mass.[127] Unfortunately, even surgical exploration with biopsy may be nondiagnostic.[68,73] In the future, it may be possible to differentiate recurrent cancer from radiation changes using MR neurography.[31,85] Reversible brachial plexopathies also follow radiation therapy.[111] Although there is no effective treatment for plexopathies following radiation, neurolysis may ameliorate severe pain, when present, although this may result in greater clinical deficits.[66,144,148]

Traumatic Brachial Plexopathies. These typically result from closed traction, most of which follow high-velocity injuries (e.g., motor vehicle accidents, especially those involving motorcycles), or sports and occupational injuries. Most closed-traction injuries produce supraclavicular plexopathies, typically upper plexopathies that follow elongation of the brachial plexus by forceful separation of the head and

shoulder.[34,89,90] Lower-plexus traction injuries are less common and usually follow forceful separation of the upper extremity from the torso. Traction injuries involving the infraclavicular plexus, as with infraclavicular plexopathies in general, more commonly are associated with bone and blood vessel injury.[65] Open traction injuries are less common and usually result from gunshot or chainsaw accidents.[148] Penetrating injuries most commonly involve the infraclavicular plexus. Low-velocity insults (e.g., knife blade, low-velocity round) produce damage by direct contact, unless the injury occurs in a delayed manner (e.g., hematoma or pseudoaneurysm formation following vessel injury).[66,148] The pressure waves and cavitation created by the passage of a high-velocity round can indirectly damage the brachial plexus by contusive and traction forces related to the cube of its velocity.[8,10,66] Although these forces infrequently disrupt the brachial plexus, they may be quite lengthy.[66] Transecting injuries may be either sharp (knives, glass) or blunt (metal fragments, fan or motor blades, chain saws, animal bites). Compression injuries usually occur between the clavicle and first rib. Since the clavicle overlies the divisions, these injuries tend to involve the distal trunk, division, and proximal cord elements. In the compressed state, contralateral head movement produces traction forces on the roots and trunks, whereas downward or lateral arm movement places traction forces on the cords.

The treatment of traumatic brachial plexopathies reflects the type of injury, its location, its severity, and its chronicity.[119] Most closed injuries are associated with lesions in continuity and are treated conservatively since it is impossible at that time to determine the likelihood of recovery or the type of surgical repair necessary.[119] Conservative management includes physical therapy, and serial clinical and electrodiagnostic assessments. When patients with focal lesions show no signs of recovery after 2–3 months (4–5 months for lengthier lesions), they are candidates for surgical exploration.[119] Shorter observation periods (3 weeks to 3 months) may be considered for high-energy injuries or those associated with total or near-total paralysis.[49] Most infraclavicular plexopathies following humeral fracture or glenohumeral dislocation (mostly axillary neuropathies) are treated conservatively, unless a severe lesion in continuity or a rupture is present.[150] Sharp lacerations usually are repaired acutely (within 72 h), by primary end-to-end neurorrhaphy, because they are easier to assess prior to the onset of scarring and because their length is not lost to retraction.[49,66,119] Other indications for acute intervention include worsening pain or neurologic dysfunction, hematoma formation, concomitant bone or vascular injuries, and compartment syndrome.[119] With blunt lacerations, repair is delayed until the proximal and distal extents of the neuroma are appreciable.[66,119] Since the affected brachial plexus elements injured by high-velocity rounds are infrequently disrupted, these lesions usually are initially (2–4 months) managed conservatively, with surgical intervention being employed for those individuals not showing improvement. Resection with graft placement usually is required.[66]

Iatrogenic Brachial Plexopathy. Iatrogenic brachial plexopathies constitute about 7 to 10% of brachial plexopathies, the majority of which are blunt lesions in continuity.[66,67,94,146] Most follow operations and other medical procedures and are related to a number of factors, including patient position (e.g., classic postoperative paralysis) and the particular procedure performed (e.g., median sternotomy). Some iatrogenic brachial plexopathies have predispositions for particular brachial plexus regions or elements, as discussed earlier. The frequent location of the musculocutaneous nerve within the so-called conjoint tendon renders it susceptible to procedures performed in this area.[12,32,53] Like other traumatic plexopathies, iatrogenic brachial plexopathies may affect the plexus directly (e.g., suture or hardware misplacement, element transection, injection injury) or indirectly (e.g., pseudoaneurysm or hematoma formation) and, hence, do not always present acutely. With midshaft clavicular fractures, the plexus can be acutely damaged by sharp bony edges or subsequently damaged by hypertrophic callus, nonunion, or a subclavian pseudoaneurysm.[4,18,22] When surgically placed screws subsequently loosen, detach, and damage nearby vascular structures, producing a hematoma or pseudoaneurysm, the plexus injury can develop long after screw placement.[26,106,146] Treatment, like that of other traumatic brachial plexopathies, reflects the type of injury and its location, severity, and chronicity. With misplaced sutures or hardware, the offending item is removed and the element repaired; with sharp transections, acute end-to-end repair typically is employed; and with blunt transections, treatment usually is delayed long enough to permit neuroma formation.[66] Injuries related to malpositioning or following procedures involving sternal splitting typically are treated conservatively. With sharp injuries, surgical repair typically is performed within the first 72 h, whereas with blunt injuries, it typically is delayed for several weeks.[66]

PROGNOSIS

The prognosis for a brachial plexus injury reflects lesion localization and severity. Disorders producing isolated demyelination recover following remyelination, a process that typically occurs within 2 to 8 weeks. However, in certain settings, demyelination more commonly converts to axon loss (e.g., radiation plexopathies) or is prolonged and frequently unresponsive to therapy (e.g., multifocal motor neuropathy). The majority of brachial plexopathies are related to isolated axon loss and the prognosis for their recovery reflects their potential for reinnervation, which in turn reflects lesion completeness and distance from the denervated muscle fibers, as well as the degree of associated connective tissue involvement. Complete lesions are not amenable to reinnervation via collateral sprouting because the latter require unaffected nerve fibers from which to sprout. Lesions located more than 20–24 inches from the denervated muscle fibers are not amenable to reinnervation via proximodistal axon advancement, which occurs at a rate of 1 inch per month, because muscle fibers cannot survive in the denervated state for more than 20 to 24 months. Connective tissue (endoneurium, perineurium, and epineurium) disruption at the lesion site leads to fibrosis, which impedes axon advancement.[113,125] Unfortunately, there are no electrodiagnostic manifestations by which to judge the degree of fibrotic impediment, and the only clinical manifestation is the passage of time (i.e., failure to recover).

Even when sensory and motor functions are recaptured, persistent and unresponsive pain may occur. Not infrequently, analgesics, neuropathic pain medications, transcutaneous electical nerve stimulation (TENS) units, stellate ganglion blocks, dorsal column stimulation, and various surgical procedures (e.g., neurolysis, sympathectomies, stellate ganglionectomies, amputation, cordotomies, and dorsal root entry zone ablations) are ineffective.[148]

The operative outcomes associated with supraclavicular lesions typically are most favorable for upper plexopathies (more likely extraforaminal; closer to denervated organs), least favorable for lower plexopathies (more likely avulsion; further from denervated organs), and intermediate for mixed upper and middle plexopathies. Regarding infraclavicular processes, surgical outcomes are better for lateral and posterior cord repairs than for medial cord repairs.[66]

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