Brachial Plexus Injuries and the Electrodiagnostic Examination

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Brachial plexus injuries represent a complex clinical challenge from both a diagnostic and treatment standpoint. Given the potential etiologic mechanisms of injury, as well as the varied number of anatomic sites vulnerable to such injury, it is essential that a precise diagnostic approach be utilized. From a neurophysiologic perspective, the electrodiagnostic examination has been shown to be the most powerful tool to evaluate brachial plexus injuries in all of their complexity. This article summarizes the more recent literature with regard to the electrodiagnostic approach to brachial plexus injuries, and discusses its clinical relevance.

Introduction

Delineation of brachial plexus injuries (both traumatic and nontraumatic) remains one of the most challenging diagnostic tasks for the sports medicine physician. Evaluation of the athlete requires not only a detailed history and physical examination, but a comprehensive knowledge of brachial plexus anatomy and the potential mechanisms of injury it may befall. Having said this, it still remains for the electrodiagnostician to make the definitive diagnosis as to the location and severity of the lesion. This article reviews the current state of the literature and field of electrodiagnostics with regard to brachial plexus injuries. Historically, the literature has pursued this problem by means of different conceptual paradigms, including the categorizing of injuries anatomically according to their location within the brachial plexus, as well as the mechanism of injury, or the injury's anatomic relationship to contiguous structures (eg, supraclavicular vs infraclavicular injuries) [1•,2,3••,4••,5–7,8•,9,10]. Whatever the etiology, anatomic profile, or mechanism of injury, analysis of brachial plexus injuries assumes a detailed knowledge of brachial plexus anatomy.

Brachial Plexus Anatomy

Given the complexity of the brachial plexus, knowledge of its contents and anatomic relationships to adjacent structures is necessary in order to properly plan the electrodiagnostic study. The ventral or anterior primary rami of C5 to T1 represent the initial precursors of the plexus itself. The anterior rami of the C5 and C6 roots coalesce to form the upper trunk. The C7 anterior ramus evolves into the middle trunk, and the anterior rami from C8 to T1 combine to form the lower trunk. At the clavicular level, the respective trunks bifurcate into their anterior and posterior divisions. The anterior divisions of the upper and middle trunks coalesce to form the lateral cord. The anterior component or division of the lower trunk becomes the medial cord. The posterior cord is formed by all three posterior divisions of the respective trunks. The cords, in turn, project from the midpoint of the clavicle to the inferomedial portion of the coracoid process of the scapula. At this juncture, the cords give rise to their respective peripheral nerve branches.

It is within this anatomic context that the electromyographer must analyze the brachial plexus injury and plan the electrodiagnostic study. Injuries can be categorized as occurring either proximal or distal (superior or inferior) to the clavicle. Supraclavicular brachial plexus lesions tend to involve both flexor and extensor musculature, whereas infraclavicular lesions tend to involve either flexor or extensor musculature, but not both. It is for this reason that plexus lesions involving the proximal components (ie, anterior primary rami and trunks) tend to be dissimilar to lesions involving the more distal elements (ie, cords and terminal peripheral nerve lesions). However, "mixed" nerve lesions may occur, making anatomic localization more difficult.

Brachial Plexus Lesions

The classification of brachial plexus injuries into either supraclavicular or infraclavicular injuries is more than just an anatomic ordering, but rather implies certain mechanisms of injury, different degrees of severity, specific types of pathology, and varying overall prognosis [11].

Supraclavicular lesions tend to be caused by closed, downward traction mechanisms or a widening of the shoulder-cervical spine angle. This mechanism tends to involve the upper and middle trunks, along with transmitted traction to the C5 and C6 roots. Infraclavicular plexus injuries commonly occur with the upper extremity abducted and extended anterior to the frontal plane, with stretching of the infraclavicular plexus in the region of its distal components. Mechanisms of this type have been shown to involve prima-

rily the posterior cord, axillary, and musculocutaneous nerves [12,13••,14].

Although the primary purpose of the electrodiagnostic examination in a patient with a suspected brachial plexus lesion is the anatomic localization of the pathology, it is also important to determine the type and severity of neuropathology present. Nerve injuries essentially fall within two categories that are not mutually exclusive: axonal loss lesions and demyelinating nerve lesions.

Axonal Loss

Axonal loss implies the disruption of axonal continuity to varying degrees with different prognostic outcomes. Axonal loss with the preservation of supporting connective tissue structures (ie, endoneurium, perineurium, and epineurium) is termed *axonotmesis*. In this instance, axonal regrowth is possible along the endoneurial tube with subsequent reinnervation of its denervated muscle tissues. In contrast, axonal injury that involves significant disruption of its supporting structural elements (endoneurium, perineurium, or epineurium) is termed *neurotmesis*. Functional return in nerve injuries of this type is generally poor due to the breach of endoneurial continuity and the potential for the misalignment of neural tube regeneration.

Brachial plexus injuries characterized by axonal loss are the most common pathologic category in plexus injuries. Additionally, they generally represent a greater degree of morbidity than do demyelinating injuries. From an electrodiagnostic standpoint, their characterization requires both a thorough nerve conduction examination as well as a comprehensive needle examination; accurate identification of the location of the nerve injury and its severity can only be accomplished with the aid of needle electromyography (EMG). This process includes the observation of spontaneous electrical activity and the analysis of voluntary motor unit action potential recruitment.

A detailed discussion of the electrodiagnostic needle examination is beyond the scope of this review, and may be found elsewhere [1•,3••]; however, the following points should be emphasized:

1. The needle evaluation for spontaneous activity in the form of positive sharp waves and fibrillation potentials provides a subjective appraisal of the degree of spontaneous muscle fiber denervation.

2. In order to obtain an accurate portrayal of the extent and location of the plexus lesion a pattern of EMG findings must be procured. This can only occur if a sufficient number of muscles are studied. Failure to do so may lead to an inability to make an accurate diagnosis secondary to a paucity of data. This remains one of the most common errors in the electrodiagnostic examination in a patient with a suspected brachial plexopathy. To reiterate a well-known dictum, "the absence of evidence is not the evidence of absence."

3. In evaluating motor unit recruitment on needle examination, one is engaged in the analysis of motor unit morphology (including amplitude, duration, and phase number) and firing rate (the sequential addition of motor units in response to increasing resistance, that is, the "size principle"). Decreased motor unit recruitment as an indication of axonal loss requires significant nerve damage before becoming observable.

4. Evaluation of motor unit morphology may be of some benefit in that incomplete nerve injuries may result in the loss of functioning motor units giving rise to collateral sprouting. The result is the creation of reinnervated muscle fibers which are added to already viable motor units. This in turn generates polyphasic motor units with increased amplitudes and durations, as well as increased phases.

Demyelinating Injuries and Nerve Conduction Studies

Demyelination in brachial plexus injuries most often takes the form of a focal lesion. The electrodiagnostic investigation of such injuries requires the performance of nerve conduction studies [15]. We discuss both sensory and motor nerve conduction studies, although there remains a role for other techniques, such as nerve root stimulation, F-waves, and somatosensory evoked potentials.

Depending upon the severity of the demyelinating lesion, the nerve conduction examination may reveal either conduction block or conduction slowing. Conduction block secondary to demyelination implies destructive alteration in the neural structure of the myelin but not the axon itself. Impulse propagation across the lesion site is prevented. Incomplete demyelination may result in only a partial block, in which impulses are capable of crossing the lesion site although at a less than normal rate. What follows is a summation of the respective importance of both sensory and motor nerve conduction studies [1•,3••,4••,5,6,8•,10,13••,16].

Sensory Nerve Conduction Studies

Only sensory nerve conduction studies allow for the determination of whether or not the plexus injury is proximal or distal to the dorsal root ganglion. This is especially important in detecting nerve root avulsions, because such injuries carry a poor prognosis for neurologic recovery. Preganglionic lesions result in an intact sensory nerve action potential (SNAP), because the lesion is proximal to the dorsal root ganglion, which is the origin of the sensory nerve cell body. Proximal, postganglionic lesions, however result with either an impaired or absent SNAP response because the lesion is distal to the dorsal root ganglion.

A limitation in the evaluation of brachial plexus lesions is the lack of singular sensory responses for delineating C5 and T1 roots. For this reason, motor conduction studies may need to be performed examining the axillary and musculocutaneous nerves for evaluation of the C5 anterior primary ramus. Additionally, the medial antebrachial cutaneous nerve (with T1 innervation) is studied in order to evaluate the T1 anterior primary ramus.

Another limitation of sensory studies in the evaluation of brachial plexus lesions is their intrinsic quality of phase cancellation with temporal dispersion. This results in deterioration and drops in SNAP amplitudes on sequential proximal studies across the presumed plexus lesion site. This is not a problem with compound motor unit action potential (CMAP) amplitudes on motor nerve conduction studies.

Motor Nerve Conduction Studies

Motor nerve conduction studies and CMAP evaluation have limited usefulness in the evaluation of brachial plexus injuries. This is due to the fact that most upper extremity motor conduction study techniques are performed distal to the location of most brachial plexus injuries. However, by stimulating proximally (*ie*, in the supraclavicular region at Erb's point) and distally (infraclavicular) to the presumed site of the brachial plexus lesion, one can attempt to determine whether or not conduction block or slowing exists. Motor conduction velocities and latencies are of limited value, even in the instance of studies in the opposite extremity for purposes of comparison. From the standpoint of the CMAP, this may result in an abnormal waveform on the affected side, characterized by a reduced amplitude, temporal dispersion, and possible "multiphasicity" in appearance, especially when compared with the comparable CMAP in the opposite extremity.

It has been shown that the most salient measurement to be obtained in the assessment of the CMAP is its amplitude [3••]. The CMAP amplitude is an indication of the number of viable motor nerve axons that can be elicited by proximal stimulation across the presumed brachial plexus lesion site. If on comparison with the comparable opposite extremity CMAP response there is a relative decrease in amplitude and morphologic asymmetry, one may infer a certain degree of conduction block (clearly implied in the absence of a response) or axonal loss.

We may conclude that in evaluating brachial plexus lesions, the needle examination provides the opportunity to localize the lesion, especially in the more proximal segments of the brachial plexus. With this in mind, recent authors have sought to provide further specific guidelines in planning the electrodiagnostic examination in order to maximize the acquisition of potential obtainable information from the patient [3••,4••,8•].

Brachial Plexus Analysis

In planning the electrodiagnostic examination, it is important to view the EMG examination as an extension of the physical examination, along with the patient's clinical history and the radiographic work-up (*eg*, radiographs, computed tomography scan, magnetic resonance imaging). To reiterate, the components of the brachial plexus can be divided anatomically based on its relative position to the clavicle. In this way, a general preliminary classification of brachial plexus pathology may be constructed in the form of supraclavicular and infraclavicular brachial plexopathies. This in no way, however, implies pathologic regional exclusivity. With this in mind, numerous authors have provided overview tables to help organize and compartmentalize the various root, trunk, cord, and peripheral nerve components, as well as their respective innervated musculature (Table 1) [1•,3••,4••,5–7,9,10,16].

Supraclavicular Plexopathies
Root lesions

Root lesions (*ie*, cervical anterior primary rami) may occur in combination with trunk and peripheral nerve injuries. The most commonly found lesions involve C5 to C6 or C5 to C7 [3••]. Moreover, because the dorsal scapular and long thoracic nerves originate at the root level, needle evaluation of their respective innervated musculature (*ie*, the rhomboids and serratus anterior) can assist in the overall evaluation of potential root pathology. The cervical paraspinal muscles should always be studied as well, to rule out a concomitant radiculopathy [17].

C5 root

Evaluation of the C5 root remains somewhat problematic from a nerve conduction standpoint, because it lacks an exclusive sensory nerve conduction technique specific to C5. Moreover, it remains difficult to differentiate between a C5 injury and an upper trunk lesion. The musculocutaneous CMAP, however, can be studied with recording at the biceps, in addition to the axillary CMAP with recording at the deltoid. The needle examination should include C5 innervated musculature and the cervical paraspinal musculature. It is within this context that root avulsions should be considered; it represents a potentially catastrophic injury and may occur with other coincident brachial plexus trauma. Cervical myelography can assist in making this diagnosis [18]. If the root avulsion occurs distal to the dorsal root ganglion, one would expect to find intact SNAP responses where technically feasible (sensory studies to the thumb, including the median and radial d1 sensory studies, as well as the lateral antebrachial cutaneous nerve, can be utilized for evaluation of the C5 and C6 roots). Additionally, one may find significant abnormalities on the CMAP studies listed above, including decreased amplitudes with temporal dispersion and multiphasicity. The needle examination can show ongoing denervation (*ie*, fibrillation potentials, positive sharp waves) in the C5 musculature.

Table 1. Upper extremity motor innervation at the root, trunk, and cord levels

Upper trunk (C5–C6)	Middle trunk (C7)	Lower trunk (C8–T1)
Supraspinatus	Triceps	Triceps
Infraspinatus	Pronator teres	Extensor digitorum
Deltoid	Flexor carpi radialis	Flexor carpi ulnaris
Biceps	Flexor carpi ulnaris	Flexor digitorum profundus
Supinator	Extensor carpi radialis	Flexor pollicis longus
Pronator teres	Extensor digitorum	Abductor pollicis brevis
Flexor carpi radialis	Extensor carpi ulnaris	First dorsal interosseous
Brachioradialis		Abductor digiti quinti
Extensor carpi radialis		
Triceps		
Lateral cord	Medial cord	Posterior cord
Biceps	Flexor carpi ulnaris	Deltoid
Pronator teres	Flexor digitorum profundus	Triceps
Flexor carpi radialis	Abductor digiti quinti	Brachioradialis
	First dorsal interosseous	Extensor carpi radialis
	Flexor pollicis longus	Extensor carpi ulnaris
	Abductor pollicis brevis	

C6 root

Sensory evaluation of the C6 root can be accomplished by performing sensory nerve conduction studies of the radial d1 (thumb), median d1, and lateral antebrachial nerves. Additionally, the superficial radial nerve may also be studied; however, its C6 innervation is less than the others listed. The needle examination should include all C6-innervated muscles.

C7 root

Isolated C7 root lesions are very rare. C7 root injury generally occurs in conjunction with trauma to other portions of the brachial plexus. Its sensory evaluation can be performed via the median d2 or d3 sensory studies. The motor study to the triceps may be of some benefit with proximal stimulation at Erb's point. This may reveal findings suggestive of conduction block or slowing when compared with the contralateral side. The needle examination should incorporate the investigation of C7 musculature.

C8 root

The ulnar d5 sensory study is used to evaluate the C8 root. The motor study generally utilized for the C8 root also includes the ulnar motor study to the abductor digiti quinti. Other potential studies that may be utilized include the median motor study to the abductor pollicis brevis and the radial motor study to the extensor indicis proprius. The needle examination should include C8-innervated muscles.

T1 root

The medial antebrachial cutaneous sensory study may be utilized for T1 root evaluation. The motor study most often utilized is the median motor study to the abductor pollicis brevis (generally more T1 innervation than the ulnar inner-

vated abductor digiti quinti). The needle examination should include all T1-innervated musculature.

With regard to the evaluation of the brachial plexus at the root level (ie, the anterior primary rami component), C5 and C6 root involvement tends to be more common in acute brachial plexitis/Parsonage Turner syndrome [19,20,21•,22], whereas C8 to T1 involvement tends to be more common in neurogenic thoracic outlet syndrome [23], metastatic plexopathies, Pancoast tumor syndrome, and complications from median sternotomy.

Brachial plexus: proximal peripheral neuropathies
Long thoracic neuropathy

The long thoracic nerve is innervated via the C5, C6, and C7 anterior primary rami as a purely motor nerve. It supplies motor innervation to the serratus anterior. The C5 to C7 anterior primary rami join to form the long thoracic nerve before progressing to create the upper and middle trunks of the brachial plexus [24–26].

On one level, because of its long anatomic course, injuries to the long thoracic nerve are more likely than if its terminal muscle innervation were more proximally located. We see this in the relatively common stretch or traction injuries that occur with athletic endeavors. Additionally, because the long thoracic nerve possesses multi-level innervation (ie, C5–C7), it has a greater statistical likelihood for involvement in a wide variety of pathologic and biomechanic mechanisms involving the proximal region of the brachial plexus.

The clinical presentation of a long thoracic neuropathy is well known (ie, scapular "winging"), with altered glenohumeral biomechanics. Additionally, a C7 radiculopathy may also present in this manner. It should be remembered that scapular winging as a clinical entity may be caused b

any combination of long thoracic, dorsal scapular, or spinal accessory nerve injury [27]. A spinal accessory nerve conduction study to the trapezius has been described, which may assist in the evaluation of scapular winging [28]. The electrodiagnostic evaluation of the long thoracic nerve does not include a sensory nerve conduction study, because it is a pure motor nerve. A motor nerve conduction study has been described, however, but there is some debate as to its actual clinical usefulness [3••]. In contrast, the needle examination remains the most important component of the EMG study. The presence of axonal loss findings and decreased motor unit recruitment allow one to make the diagnosis.

Dorsal scapular nerve

The dorsal scapular nerve originates within the body of the scalenus medius muscle and supplies the levator scapulae and the rhomboids during its course. Trauma to the dorsal scapular nerve may result in lateral winging of the scapula, but unlike long thoracic neuropathies, significant dysfunction with regard to shoulder girdle mechanics is rare. As a pure motor nerve, sensory studies are not applicable; motor studies to the rhomboids are not felt to be clinically helpful. The diagnosis of dorsal scapular pathology is therefore dependant on the needle examination.

Suprascapular nerve

The suprascapular nerve is the only peripheral nerve that originates from the upper trunk with primarily C5 innervation, and some C6 contribution. It is susceptible to injury during its course as it passes through the posterior triangle of the neck to the superior border of the scapula, where it passes through the suprascapular notch and supplies the supraspinatus. It then passes around the spinoglenoid notch and terminates in the infraspinatus. There are no cutaneous nerve branches that can be studied electrodiagnostically [29–36].

The clinical presentation of suprascapular neuropathies includes poorly localized shoulder girdle pain, weakness in shoulder abduction, and external rotation along with intact sensation. The presence of deltoid pathology and/or scapular winging should alert the physician to a more generalized process such as acute brachial plexitis (Parsonage Turner syndrome). In this instance, however, the involvement of the suprascapular nerve merely represents a component of a more generalized neuropathic condition with an autoimmune etiology, which may simultaneously involve multiple brachial plexus components at the root and trunk levels.

It remains the task of the electrodiagnostic examination to determine the anatomic level (posterior triangle, suprascapular notch and spinoglenoid notch as potential entrapment sites), and degree and type of suprascapular nerve injury (axonal loss and/or demyelinating pathology). As with other peripheral nerve injury evaluations (*ie*, long thoracic and dorsal scapular nerves), motor nerve

conduction studies have limited diagnostic efficacy. The needle examination remains the most important diagnostic tool for delineating the extent of nerve trauma. Finally, other C5 and C6 musculature, along with the cervical paraspinal musculature, should also be examined to rule out a more diffuse process.

Axillary nerve

Axillary nerve innervation is supplied by the C5 to C6 roots (anterior primary rami) via the posterior cord. As a terminal branch of the posterior cord, it supplies the deltoid and teres minor muscles. The greatest frequency of axillary nerve trauma occurs with blunt trauma, for example, shoulder dislocation. Contact sports such as football also possess a high incidence of injury [37,38].

The electrodiagnostic examination of the axillary nerve should include a needle examination in all three components of the deltoid, because the nerve injury may not be uniform in all three branches. The teres minor should be examined along with the triceps to help rule out a posterior cord lesion. The differential diagnosis should include C5 and/or C6 radiculopathies, as well as an upper trunk brachial plexopathy. The axillary motor study can be performed with side-to-side comparison of the CMAPs, which assists in evaluating axonal loss severity [3••,15]. There are no sensory studies available to assess the sensory branch of the axillary nerve.

Thoracic outlet syndrome

The term *thoracic outlet syndrome* (TOS) refers to a symptom complex that actually represents more than one clinical entity [21•,22,23]. It continues to remain a point of controversy in the medical literature. As a syndrome, it incorporates a constellation of clinical findings and complaints that can challenge even the most astute diagnostician. From the standpoint of etiology, it has been subdivided into vascular and neurogenic subcategories. Whereas the former is characterized by compression of the subclavian blood vessels, the later is characterized by compression to the brachial plexus itself. The clinical conundrum occurs because of the relative similarity of their clinical presentations.

The neurogenic form of TOS is a rare entity as confirmed on electrodiagnostic testing. It generally involves the lower trunk and/or the C8 to T1 anterior primary rami. Upper trunk involvement is less frequent (unlike acute brachial plexitis; Parsonage Turner syndrome).

The electrodiagnostic evaluation of the neurogenic form of TOS remains the lynch-pin in the diagnosis of this entity [21•]. Electrodiagnostic findings may include 1) decreased median motor CMAP (the EMG parameter most affected); 2) ulnar SNAP less than 10 uV in 50% of patients; and 3) axonal loss findings in the lower trunk musculature (especially abductor pollicis brevis and first dorsal interosseus).

Trunk lesions

The electrodiagnostic evaluation of trunk lesions requires the differentiation between the more proximal brachial plexus elements listed above (eg, the respective anterior primary rami and the more proximal individual peripheral nerves) and the trunks themselves (including their terminal branches).

Upper trunk lesions

Electrodiagnostic evaluation of the upper trunk incorporates sensory and motor conduction studies as well as the needle examination. There is general agreement that the most optimal sensory studies include the lateral antebrachial cutaneous and the median d1 studies [4••,8•]. Potential motor studies include both the axillary (to the deltoid) and the musculocutaneous (to the biceps) nerves. One must be aware, however, that peripheral nerve entrapments or injuries to these nerves may provide misleading information, giving the impression of an upper trunk lesion instead of a peripheral nerve injury. The needle examination includes all of the upper trunk innervated muscles listed above, as well as the cervical paraspinal musculature.

Potential etiologies causing neurologic lesions involving the upper trunk include acute brachial plexitis (Parsonage Turner syndrome), obstetric palsies, postoperative paralytic syndromes, and the burner/stinger syndrome.

Burner or stinger syndrome

From the standpoint of the sports medicine physician, the burner or stinger represents a ubiquitous neurologic injury to the brachial plexus that is very common in football players. There has been considerable debate regarding the exact nature of the burner syndrome from the standpoints of its injury mechanism, pathophysiology, and anatomic location. This is partly due to the current limitations in the field of electrodiagnostics to examine the proximal regions of the brachial plexus. Essentially, the burner represents a traumatic proximal injury to the plexus primarily involving the cervical roots and the trunks. It still remains within the capability of the EMG examination to obtain a significant amount of information that will further define the injury to a relatively high degree of sensitivity and specificity. In considering the evaluation of a patient who may be suffering a burner, it may be helpful to view the clinical presentation as a symptom complex characterized by a sharp, burning pain radiating from the supraclavicular region, extending distally down the arm [39•,40–43].

The localization of the site of injury may be related to the injury mechanism itself. Supraclavicular injuries tend to be secondary to traction mechanisms, with multiple involvement at both the root and trunk levels. The upper and middle trunks tend to have a higher incidence. Infraclavicular injuries tend to be secondary to shoulder abduction/extension mechanics, which tend to involve cords and terminal nerves generally, and the posterior cord and axillary nerve more specifically. In both types of biomecha-

nism, the suprascapular nerve is commonly involved. Distal peripheral nerves may also be involved at their sites of origin, including the musculocutaneous, radial, median, and ulnar nerves.

Middle trunk lesions

As an isolated brachial plexus lesion, middle trunk lesions are considered extremely rare. Generally, the middle trunk represents an unlikely site for a traction injury although it can be affected by penetrating injuries. The middle trunk represents the continuation of the C7 anterior primary ramus. Care must be taken to distinguish middle trunk lesions from C7 radiculopathies and posterior cord lesions.

The evaluation of the middle trunk may incorporate the median sensory study to d3 (middle finger) with radial motor study to the extensor digitorum communi muscle. The needle examination includes C7 innervate musculature (eg, triceps, anconeus, pronator teres, flexo carpi radialis, extensor carpi radialis, extensor digitorum with the exception of the serratus anterior.

Lower trunk lesions

The lower trunk is formed via the fusion of the C8 and T anterior primary rami. Trauma to the lower trunk ofte represents a problematic situation; reinnervation is mo difficult given the relative distance of the innervation to i respectively assigned musculature in the brachial plexu anatomic paradigm [44]. The differential diagnosis extensive and should include C8 and T1 radiculopathie as well as median and ulnar peripheral neuropathies, ar focal lesions at the root level. From a technical standpoi it can be very difficult to distinguish a lower trunk lesic from a medial cord lesion.

The lower trunk can be evaluated electrodiagnostica with the use of an ulnar sensory nerve conduction study d5 (little finger), the dorsal cutaneous ulnar nerve, and t medial antebrachial cutaneous nerve. The motor nerve cc duction studies may include the ulnar motor study to t first dorsal interosseous and the abductor digiti minir Other motor options include the median motor study to t abductor pollicis brevis or the radial motor study to t extensor indicis proprius. The needle examination inclu the C8 and T1 innervated musculature (eg, flexor ca ulnaris, flexor digitorum profundus 4/5, extensor digiton communis, extensor carpi ulnaris, abductor digiti mini first dorsal interosseous, and abductor pollicis brevis).

Brachial Plexus Cord Lesions

Because of the relative proximity of the respective cord their terminal peripheral nerves, cord injuries tenc present with more exclusive axonal loss findings on r dle examination with regard to individual periph nerves. This represents a significant difference from r and trunk lesions.

Medial Cord Lesions

The medial cord is the continuation of the anterior division of the lower trunk. Its electrodiagnostic evaluation includes the medial antebrachial cutaneous sensory study (to evaluate C8 and T1 roots), along with the ulnar sensory study to d5. Motor studies include the thenar and hypothenar musculature. It should be noted that lesions involving the lower trunk as well as the medial cord may present in the same manner with regard to either absent or decreased SNAPs or CMAPs. The needle examination is important in differentiating lower trunk from medial cord lesions. Specifically, medial cord lesions will present with axonal loss findings in C8- to T1-innervated thenar and hypothenar musculature, whereas C8-innervated radial musculature will be spared. If both groups of muscles are found to harbor axonal loss pathology, then a lower trunk lesion should be suspected.

Lateral Cord Lesions

The lateral cord represents the continuation of the anterior primary rami of the upper and middle trunks. It is important in lateral cord lesions to distinguish between proximal and distal lesions. This is accomplished by evaluating the pectoralis major muscle (superior and middle components) because its innervation (lateral pectoral nerve) is supplied from the proximal part of the cord [3••]. The terminal portion of the cord supplies the lateral half of the median nerve (*eg*, pronator teres innervation). The remainder of the median innervated musculature is supplied by the medial cord.

The electrodiagnostic evaluation of the lateral cord includes sensory and motor conduction studies as well as a detailed needle examination. The sensory exam may include the lateral antebrachial cutaneous nerve and the median d1 to d3 sensory studies. The motor studies include the musculocutaneous study to the biceps. The needle examination should focus on the biceps, brachialis, pronator teres, and flexor carpi radialis. Upper trunk lesions can be ruled out by the absence of axonal loss findings in the deltoid, brachioradialis, supraspinatus, and infraspinatus.

It is important to note that the musculocutaneous nerve, because it arises from the lateral cord, should be carefully evaluated for the presence of an isolated peripheral neuropathy [45,46]. Although musculocutaneous nerve injuries are uncommon, they may occur secondary to acute shoulder dislocations and present with biceps and brachialis weakness, along with sensory dysesthesias in the lateral forearm. The EMG evaluation, while including all of the guidelines above in the assessment of the lateral cord, may also utilize bilateral musculocutaneous motor studies for comparison purposes. C5-6–innervated muscles should also be studied on the needle examination to assess for the possibility of a cervical radiculopathy at these levels.

Posterior Cord Lesions

The posterior cord is formed by the joining the posterior segments of all three trunks. It terminates as the radial nerve. As with the lateral cord, it may be possible to differentiate between proximal and distal lesions. This requires needle examination of the latissimus dorsi muscle (proximal innervation by the thoracodorsal nerve) and the teres major muscle (proximal innervation by the lower subscapular nerve). The deltoid may be examined as a more distal muscle (axillary nerve innervation), along with the radial innervated musculature.

The nerve conduction examination in the evaluation of potential posterior cord lesions includes the radial d1 sensory study, as well as motor studies to the deltoid (axillary nerve) and the extensor indicis proprius (radial nerve).

Conclusions

The evaluation of a brachial plexus lesion requires the integration of a detailed history and physical examination, along with a comprehensive electrodiagnostic examination. Mechanisms of injury and anatomic relationships must be integrated into the planning of the EMG study. A "cookbook approach" will not suffice given the potential complexity and singularity of each individual case. Moreover, as one proceeds through the electrodiagnostic study, the acquisition of information may require an alteration in its original design. The EMG diagnostic process remains, therefore, an active, dynamic endeavor, with exceptional potential for unraveling the mysteries of the brachial plexus.

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