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Electromyography in Shoulder Disorders

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Introduction

This article reviews some of the disorders that can be investigated in the electromyography (EMG) laboratory. EMG is defined here as nerve conduction studies (NCS) and needle electromyography (NEMG). NCS determines the integrity of the myelin sheath of a nerve, the amount of axons present, and the integrity of synaptic transmission between nerve and muscle. NEMG can detect disturbances in various parts of the motor unit, which consists of the anterior horn cell, its axons, and the skeletal muscle fibers innervated by those axons. Therefore, EMG is an extension of the clinical examination of the peripheral nervous system; it can detect disease in the lower motor system that may not be symptomatic or visible to the naked eye.

Demyelination and axonal degeneration

Mild nerve injury often results in focal demyelination causing paralysis, such as wrist drop in Saturday night palsy or footdrop in peroneal nerve palsy at the fibular head. Paralysis is due to failure of electrical impulses to proceed across the segment of demyelination, producing a conduction block. Although nerve injury is more severe, Wallerian degeneration occurs; the axons that are covered by the myelin sheath are damaged, resulting in death of the axon and ultimately retrograde degeneration of the cell body. Weakness occurs when there is a significant reduction in the number of axons (and by extension, a reduction in the number of motor units) available to cause muscular contraction. A clinical feature of demyelination is weakness without muscle atrophy, whereas axonal degeneration, especially when severe, will cause weakness and severe muscle atrophy.

Nerve entrapment refers to a nerve that is free in a compartment but compressed by neighboring tissues. The term also refers to a nerve that is pulled while being tethered to other tissue. In progressive entrapment neuropathies, demyelination precedes axonal degeneration; in fact, nerves are relatively resistant to axonal degeneration [1].

NCS and NEMG

An extensive description of this topic is beyond the scope of this paper but can be found elsewhere [2,3]. NCS are used to detect demyelination and axonal degeneration in a nerve, to localize the site of the lesion, and to quantify the degree of injury. The electrical features of demyelination are slowing of the conduction velocity, especially when it is diffuse along the length of a nerve. When only a segment of nerve is demyelinated, there is failure of conduction of the electrical signal across that segment. The exact electrical criteria needed to make this diagnosis remain somewhat controversial [4–6].

In axonal degeneration, the conduction velocity is nearly normal, but there is a reduction in the size of the compound muscle action potential (CMAP). When axons die, their numbers dwindle and become insufficient to result in a forceful muscular contraction. Therefore, the muscle contraction is weak and its signal output is low as recorded by NCS.

NEMG detects abnormal spontaneous muscle contractions. When abnormal contractions occur in shoulder muscles, NEMG provides incontrovertible objective evidence of neuromuscular disease. The two most important types of contractions are fasciculations and fibrillations. Fasciculations are contractions of a part or all of a muscle. They can be seen with the naked eye and are identified by the NEMG as the firing of part or all of a motor unit. Fasciculations are believed to arise in the distal axon or even in the nerve terminals in most neuromuscular conditions [7].[a] Fibrillations are contractions of individual muscle fibers that can be detected by the needle electrode. Like fasciculations, fibrillations are a hallmark feature of ongoing axonal degeneration. Both fasciculations and fibrillations can be seen in neurogenic disorders, but fasciculations can occur in healthy individuals.

In addition to spontaneous activity, NEMG detects the architecture of the motor unit. That is to say, a motor unit that has been denervated has characteristics that distinguish it from one suffering from primary muscle disease. In a weak muscle, a normal NEMG suggests a mechanical or nonphysiological cause (i.e., malingering). Similarly, muscle atrophy without abnormalities on NEMG examination suggests disuse.

Uses and limitations of EMG

In disorders of the shoulder, EMG studies can identify and characterize nerve injury and locate the site of injury

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[a]Eisen and Krieger [8] suggest that some of the fasciculations in amyotrophic lateral sclerosis (ALS) are produced in the upper motor system.

along the nerve or one of its components. EMG can determine if a nerve is in continuity and the degree of demyelination or axonal degeneration present. It is also helpful in making a prognosis and determining treatment. For example, in traumatized nerves, axonal degeneration does not recover as well as demyelination, especially in very long nerves. Acute wrist drop (Saturday night palsy), when mild, recovers quickly. Acute wrist drop is an example of focal demyelination of the radial nerve. The weakness is due to conduction block. Treatment here is conservative. By contrast, if there is persistent evidence of axonal loss (see below under axillary neuropathy), an axillary neuropathy occurring as a complication of shoulder surgery may require surgical exploration and neurolysis.

The EMG can help determine prognosis. In some cases, weakness is due to a combination of focal demyelination and axonal degeneration (also known as denervation). The initial EMG in such cases cannot determine prognosis. However, serial studies may be useful. For example, if the patient is weak and fibrillation potentials are seen on the initial study (which should not be performed sooner than three weeks from the onset of injury to allow fibrillation potentials to appear), a follow-up study showing diminution or disappearance of fibrillation potentials, or other evidence of denervation, would suggest a good prognosis for neurologic recovery. Because newly formed axons do not conduct electrical impulses as efficiently as the native axons, the persistence of significant denervation (i.e., axonal loss) is a poor prognostic sign.

There are several limitations to EMG studies. The test is uncomfortable and at times painful. Obesity limits the accuracy of nerve conduction in the brachial plexus. Surgery with injury to muscles limits the NEMG examination. Muscle injury results in fibrillation potentials, making it impossible to distinguish between postsurgical changes and those caused by disease or surgical trauma. Most importantly, the sensitivity and specificity of EMG relative to other testing modalities including radiographic imaging has been studied in only a few disorders affecting the shoulder (vide infra).

EMG and Entrapment of Some Nerves of the Shoulder

This section deals with some of the most commonly encountered entrapment neuropathies in the shoulder.

Suprascapular nerve entrapment

Suprascapular nerve entrapment is probably an underdiagnosed disorder in EMG laboratories. The suprascapular nerve originates from the C4,5,6 nerve roots and exits the posterior aspect of the brachial plexus. It passes (posterior) to the trapezius muscle and is in close proximity to the distal clavicle and acromioclavicular joint [9]. When traversing the scapular notch, the nerve is prone to entrapment from the superior transverse scapular ligament above it. This may cause pain in the shoulder because the nerve sends sensory branches to the acromioclavicular and glenohumeral joints. It may also cause weakness in shoulder abduction, especially during the first 30 degrees of movement. Because the short leverage of the deltoid is not sufficient, there may also be significant weakness between 70 and 120 degrees of abduction.

After passing through the scapular notch, the suprascapular nerve descends anteriorly to the scapular spine and through the spinoglenoid notch (notch of the scapular spine), passing under the inferior transverse scapular ligament to innervate the infraspinatus. The nerve is relatively fixed in this segment. Injury at this point has resulted in isolated infraspinatus neuropathy with weakness of external rotation of the shoulder [10,11]. A study of 23 cadaveric shoulders has demonstrated that the spinoglenoid ligament is present 60.8% of the time [12]. The ligament tightens and presumably entraps the suprascapular nerve during adduction and internal rotation of the glenohumeral joint. This may be the mechanism of isolated weakness of the infraspinatus. It may not be noticed by the patient as the infraspinatus muscle is aided by the posterior belly of the deltoid and the teres minor during external rotation of the shoulder.

EMG is useful in detecting injury to the suprascapular nerve. NCS are difficult to perform because of the deep location of this nerve. Electrical stimulation of the nerve is likely to spread to nearby nerves and muscles, causing contraction of many muscles, rather than simply to the supraspinatus and infraspinatus. A side-to-side comparison of the NCS is mandatory. However, subtle abnormalities from demyelination will probably be missed. Especially if there is axonal degeneration leading to the appearance of fibrillation potentials, the needle examination is likely to be more useful [13].

The Axillary Nerve

The axillary nerve can be involved in shoulder trauma or it may be entrapped. Trauma can occur during contact sports [14] or during surgery of the inferior aspect of the shoulder. Entrapment occurs in the quadrilateral space. This space is bordered by the capsule of the shoulder and the tendons of the teres minor and subscapularis above, laterally by the surgical neck of the humerus, medially by the tendon of the long head of the triceps, and below by the tendon of the teres major. After passing through this space, the nerve supplies the teres minor (via the posterior branch) before innervating the posterior, middle, and anterior (in that order) bellies of the deltoid.

Axillary nerve injury resulting in paralysis of the deltoid muscle causes weakness of shoulder abduction, particularly after 30 degrees of abduction. The supraspinatus initiates abduction of the shoulder in the first 30 degrees of movement. Therefore, this part of shoulder abduction is spared.

The teres minor and infraspinatus contribute to external rotation of the shoulder, so this movement is less affected by axillary neuropathies. This is especially true if the lesion is distal to the innervation of the teres minor or if the lesion is in the proximal segment of the nerve but is mild. Rarely in axillary neuropathies is there a patch of numbness over the

deltoid, the region of sensory innervation of the axillary nerve. More commonly, the patient has no sensory complaints.

Case Report

A 60-year-old man dislocated his shoulder while pulling a heavy object. He underwent surgical repair of a rotator cuff tear followed by two additional shoulder procedures. Postoperatively, he developed severe shoulder pain, deltoid muscle atrophy, and weakness.

Abundant fibrillation potentials and a neurogenic firing pattern (a discrete number of motor units fired rapidly) were seen on EMG. The pain persisted. Ten months later, his surgeon contemplated surgical exploration of the axillary nerve. This was deferred when a repeat EMG showed minimal fibrillation potentials and many more motor units than previously recorded from the deltoid. These changes were more prominent in the posterior than in the anterior belly of the deltoid, consistent with the anatomy of the nerve. This case illustrates the utility of EMG in determining the need for surgical intervention. If inappropriate, surgery may damage a healing nerve.

The most common type of axillary nerve palsy occurs following shoulder dislocation or humeral fracture. This type of axillary nerve injury has a good prognosis. Another form is seen after blunt trauma to the shoulder region without associated fracture or dislocation. Berry and Brill [15] reviewed 13 patients with this type of palsy after blunt shoulder trauma. Seven patients showed minimal or no recovery of deltoid muscle function and six patients showed complete or near complete recovery. Serial EMG examinations usually revealed the lesion to be in continuity. The mechanism of the palsy appeared to involve a stretch injury; this was confirmed at operation in two patients. Glenohumeral fixation was a troublesome complication that limited recovery of function in four patients.

Winging of the Scapula

The scapula is kept in place, against the chest wall, by two main muscles—the serratus anterior and the upper portion of the trapezius (the other muscles involved in movements of the scapula are beyond the scope of this discussion and can be found elsewhere [15]). The serratus anterior pulls the medial border of the scapula anteriorly and laterally during forward extension of the arm and the trapezius pulls the superior border of the scapula up and medially during lateral movement of the arm [16].

Weakness of the serratus anterior, innervated by the long thoracic nerve, causes posterior projection of the medial border of the scapula or "winging." The angle of the scapula moves up and medially during forward extension of the arm. When winging is due to weakness of the upper portion of the trapezius, the scapula moves down and laterally during lateral movement of the arm. The causes of winging of the scapula secondary to involvement of the serratus anterior or its nerve are many and include sports injury [17,18] and other trauma [19] and various neuromuscular diseases [20,21]. C7 radiculopathy should be considered in the differential diagnosis of scapular winging [22].

The accessory nerve innervates two muscles, the sternocleidomastoid (SCM) and the trapezius. The most common cause of accessory neuropathy is surgical trauma in the posterior triangle of the neck, especially during lymph node dissections. If the lesion is proximal to the innervation of the SCM, there may be weakness in turning the head because of paralysis of the SCM and winging of the scapula. If the lesion is distal to the innervation of the SCM, then there will be isolated winging of the scapula. An unusual cause of scapular winging is spontaneous accessory neuropathy [23]. This may be a variant of brachial amyotrophy (see below) and its prognosis is very good.

In accessory nerve palsy, whether from surgical trauma or idiopathic, one electrodiagnostic study of 16 patients found excellent nerve regeneration after severe denervation [24]. The authors suggest delaying surgical intervention given the good clinical prognosis. NCS of the trapezius is easily performed and the amplitude of the CMAP will be reduced, especially with lesions affecting the upper fibers of the muscle [25]. NEMG will show fibrillation potentials when the lesion is acute.

Cervical Radiculopathies and Brachial Plexopathies

There are several clinical features that help distinguish lesions of the cervical nerve roots from those of the brachial plexus (Table 1). Cervical radiculopathies cause paresthesias in dermatomal distribution (C5,6, thumb and index finger; C7, middle finger; C8, T1, fourth and fifth fingers) and simultaneous weakness in a myotomal pattern. Brachial plexopathies cause sensory symptoms and weakness in the distribution of individual nerves or cords or trunks. The pain of a cervical radiculopathy is intermittent and can often be reproduced by hyperextension of the neck. Unlike cervical radiculopathy, coughing or sneezing cannot reproduce the pain of brachial plexopathy.

Brachial plexopathy should be suspected when there is antecedent trauma, especially from contact sports. The upper trunk of the plexus is most commonly involved, although the damage is seldom restricted to one spot and a variety of combinations of nerves arising from the plexus, cords, and trunks are affected [26]. Likewise, radiation-induced plexopathy usually involves the upper trunk [27]. This is not always the case. Others have found the lower

Table 1. Symptoms of cervical radiculopathy versus brachial plexopathy

Symptom	Cervical radiculopathy	Brachial plexopathy
Weakness	Myotomal	Nerve/upper trunk
Atrophy	Unusual	Common
Sensory loss	Dermatomal	30–70%* and focal
	Unusual	

*Figures refer to patients [35].

trunk to be equally affected [28]. To distinguish between malignant infiltration and radiation plexopathy can be notoriously difficult. If more than 6,000 rads are given, 73% patients develop radiation plexopathy. The duration of onset ranges from several months up to 20 years [29].

Weakness and atrophy are more commonly seen in plexopathies. The sine quo non of NCS in brachial plexopathies are abnormal sensory nerve responses. These are invariably normal in radiculopathies because the dorsal root ganglion cells are distal to the site of injury, thus sparing the sensory nerve.

In the only study comparing EMG to magnetic resonance imaging (MRI) in radiculopathy, Nardin et al. [29] found the sensitivity of EMG to be 72% and MRI to be 60% at best. The two tests were in agreement 60% and in disagreement 40% of the time. The authors correctly conclude that the two examinations are complementary.

Brachial Amyotrophy

This section discusses some of the disorders that affect primarily the brachial plexus. These include brachial amyotrophy and its differential diagnosis and thoracic outlet syndrome. Brachial amyotrophy is also known as idiopathic brachial neuritis, neuralgic amyotrophy, and Parsonage-Turner syndrome, whose reports firmly established this entity [31,32]. The term *brachial* refers to the arm rather than the brachial plexus.

The typical presentation is the sudden onset of severe arm or shoulder pain. Narcotics are required in some cases. The pain resolves within one to three weeks. This is then followed by weakness and muscle atrophy in the shoulder or arm muscles. The weakness is usually quite focal and can be traced to an individual nerve (mononeuropathy) or to several nerves (mononeuropathy multiplex) such as the long thoracic, antebrachial cutaneous, suprascapular, and phrenic nerve [33,34]. An EMG will show evidence of axonal degeneration (fibrillation potentials, sharp positive waves). England and Sumner [32] have postulated that focal trauma occurs to some nerves as they cross joint. The cause of this disorder is unknown. An antecedent event occurs in 28–83% of cases [35]. These include trauma and surgery in areas remote from the shoulder, infection, childbirth, and immunization. Postsurgical brachial neuritis occurred in 10% of Parsonage and Turner's original and in more recent reports [36]. Recognition of shoulder surgery as a risk factor is important to prevent unnecessary exploration of the brachial plexus. Treatment is supportive as 90% of patients recover by three years [37].

The differential diagnosis in this disorder is a cervical radiculopathy, thoracic outlet syndrome, or more widespread disorders like amyotrophic lateral sclerosis (ALS), multifocal motor neuropathy (MMN), or hereditary acute brachial plexopathy. Hereditary acute brachial plexopathy is a well-described disease. Transmission is autosomal dominant. A deletion of the peripheral myelin protein-22 (PMP22) gene on chromosome 17p11.2- 12 has been found [38]. The clinical tipoff to the presence of this condition is recurrent painless brachial plexopathy, sometimes associated with a more widespread polyneuropathy [39–41]. There are no convincing data associating diabetes mellitus with brachial plexopathy [37]; diabetes is much more commonly associated with a lumbosacral plexopathy.

Delayed Costoclavicular Syndrome and Other Types of Thoracic Outlet Syndrome

The alert physician should also consider delayed compressive brachial plexopathy after fractures of the clavicle, the costoclavicular syndrome [42]. Only 3–5% of fractures of the clavicle result in nonunion. A brachial plexopathy is even rarer. Nonunion most commonly occurs after fractures of the middle portion of the clavicle [43], the site most commonly associated with a costoclavicular syndrome [44].

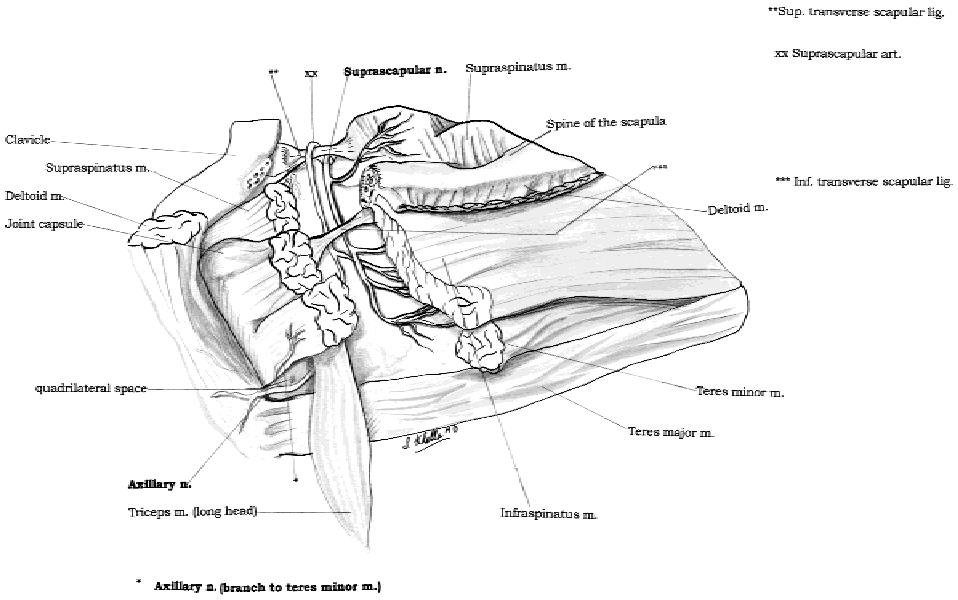

Fig. 1. Dorsal Aspect Left Shoulder.

The middle cord of the plexus is most often compressed, probably by the dorsal tug of the trapezius and deltoid. Other factors contributing to a delay in the compressive plexopathy can include exuberant callus formation or expanding false aneurysms of the subclavian artery and subsequent narrowing of the costoclavicular space [42]. Surgical decompression is the treatment of choice.

The thoracic outlet comprises a series of passages in which the neurovascular bundle may be entrapped. These passages include the interscalene triangle, the costoclavicular space, and the axilla. Compression in any of these passages can lead to thoracic outlet syndrome (TOS). There are currently five groups that make up TOS: one is traumatic; two are vascular, arterial, and venous; one is neurogenic; and the fifth is designated disputed neurogenic [45].

Neurogenic TOS is a rare entity. The lower trunk of the brachial plexus is the usual site of injury, resulting in thenar more than hypothenar muscle atrophy and sensory loss along the medial hand and forearm. The diagnosis is supported by EMG localizing the lesion to the lower trunk and a cervical rib is most often seen on C-spine x-rays [45]. By contrast, the disputed TOS is a syndrome of fleeting hand and arm pain or paresthesias, a variety of unexpected symptoms including headache and low back pain, no intrinsic hand muscle atrophy, and normal EMG and radiographic studies [46]. Often, these are work-related complaints but no specific job or limb activity has been proved to be causal. Its proponents have refused to participate in blinded testing [47]. Kasdan et al. [47] have suggested that misdiagnosing patients can be detrimental. Therefore, I strongly urge physicians not to diagnose a disorder that has no typical or agreed upon symptoms and no clear cause; one that is impossible to support by objective tests; and the main proponents of which refuse to test its validity and yet claim that surgery is the best possible means of treatment [46].

ALS and MMN

Lastly, this section covers ALS and MMN. MMN may present with painless shoulder weakness. The patient does not complain of numbness or tingling. The disease often affects the hands more than the legs. Muscle atrophy may be present and the disorder can be confused with ALS [49]. A careful history and examination will reveal that the weakness is confined to the territory of individual nerves rather than the more widespread weakness across many myotomes seen in ALS [8]. Like ALS, fasciculations and muscle atrophy are present in MMN but are not as widespread as in ALS. The deep tendon reflexes in MMN are diminished whereas those in ALS are brisk. In MMN, the EMG shows the changes of demyelination (reduced conduction velocity and conduction block with few fibrillation potentials). In ALS, the changes are those of axonal degeneration (near normal conduction velocities, abundant fibrillation potentials, and fasciculations even in clinically normal limbs). ALS should thus be considered in the differential diagnosis of painless shoulder muscle atrophy, especially of older adults. MMN is treatable and may improve with intravenous immunoglobulin [50], whereas treatment for ALS is very discouraging. Space does not permit discussion of a variety of other neuromuscular disorders that can present in the shoulder and the reader is referred to several recent texts [51–53].

In summary, EMG is a technique that can quantify nerve or muscle disorders causing shoulder pain or weakness. It may be abnormal when clinical signs are absent. It may detect more generalized disorders than clinically suspected. In some instances, it is useful in helping to decide the need for surgical intervention.

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