

“EMG” for thoracic outlet syndrome

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The use of electrodiagnostic studies (EDX) in the work-up of thoracic outlet syndrome (TOS) is said to be controversial. We believe it is essential.

Neurogenic TOS refers to dysfunction of various components of the brachial plexus at various points between the neck and axilla [1]. For the purposes of this article, the author uses the term neurogenic to refer to those cases in which objective electrophysiologic evidence is obtained. How that evidence is obtained and its validity are discussed.

TOS presents with a variety of causative factors, symptoms, and physical findings; as a result, there is no gold standard for diagnosing it [2]. The diagnosis of neurogenic/neurologic TOS elicits a response of controversy, and so do the various methods of arriving at that diagnosis. To some, TOS is a diagnosis of exclusion that incorporates electrodiagnostic testing for exclusion purposes only [3]. To others, various electrodiagnostic techniques are a means of documenting the compromise of the brachial plexus or even to localize the problem to a specific segment of the brachial plexus [1,2,4–24].

Although few would dispute the value of a good clinical diagnosis, the virtues of EDX lead to considerably more contention when discussing TOS. The rationale for EDX has been well stated as a logical “extension of the history and physical exam, but it can provide reproducible objective data on the severity, progression over time, and extent of recovery after surgery for nerve entrapment” [4].

There are two major categories of EDX; they are electromyography (EMG) and nerve conduction studies (NCS). Some examiners seem to

weigh either the EMG or the nerve conduction data more heavily, or sometimes exclusively, in arriving at their diagnosis. Others seem to take a more balanced approach, weighing these findings more as an indicator of involvement or a way of determining specific location within the brachial plexus and level of severity.

Some suggest or outright state that EMG (axonal/neurogenic) changes are of primary importance and apparently believe that EMG findings, including denervation, polyphasia, and decreased motor unit activity are necessary for a definitive diagnosis [5–7]. Those who rely on NCS more heavily do not seem to totally discount EMG evidence of TOS, but believe that NCS may be an earlier and more sensitive diagnostic tool [8].

Diagnostic theories and early detection

A review of the literature reveals four primary groups of diagnostic theory.

1. Clinical diagnosis of inclusion (ie, history, signs, symptoms, and provocative testing, as are noted elsewhere in this issue)
2. Clinical diagnosis of exclusion (ie, radiographs, CT scans, MRI, vascular studies); electrodiagnostic testing is used to rule out distal and systemic neuropathies
3. Primary diagnosis by electrodiagnostic studies (electromyography or NCS)
4. An eclectic approach incorporating all of the above with various levels of reliance on these individual techniques

When there is EMG evidence of TOS/brachial plexus neuropathy, there is little controversy. There is, however, considerable disagreement with regard to its ability to detect earlier/less severe

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states of pathology before the evolution of axonal change. NCS detect segmental demyelination and reflect these early changes.

Several investigators suggest that only axonal EMG changes are of diagnostic significance, including denervation activity, increased polyphasia, and decreased interference patterns [5,7]. Others suggest that, although EMG evidence is certainly significant, it is usually associated with more severe states of pathology; as a result, EMG is not sufficiently sensitive to detect early/less severe involvement. Furthermore, it is generally agreed that detection at an earlier, less severe state might allow more timely treatment and might result in increased incidence of recovery or improvement.

Early detection seems therefore to be the primary rationale for the use of NCS [8]. These studies fall into the primary categories: motor and sensory. Each of these categories includes multiple specific techniques.

The motor category includes but is not necessarily limited to brachial plexus cord level testing, brachial plexus trunk level testing, and late response testing (primarily F-wave responses). The sensory category includes evaluation of sensory amplitudes of response for the median, ulnar, lateral antebrachial cutaneous, and medial antebrachial cutaneous nerves, as they reflect brachial plexus dysfunction. Various investigators preach the superiority of one, some, or all of the above. Somatosensory evoked potentials (SSEPs) also fall into this category.

Motor testing

Cord level testing of the brachial plexus consists of stimulation at the axilla and supraclavicular areas with the shoulder in 90° of abduction and full external rotation. The motor distal latency and amplitude of response is recorded from a distal muscle innervated by the appropriate cord of the plexus (upper/lateral, middle/posterior, and lower/medial). Examples of these would be recording with surface recording electrodes over the biceps for the lateral cord of the upper plexus, the extensor carpi radialis, or abductor pollicis longus for the posterior cord of the middle plexus and the adductor digiti minimi for the medial cord of the lower plexus.

Sequential motor distal latencies are recorded from the axilla and the supraclavicular area, with supramaximal level stimulation. The distance between the stimulation sites is measured accu-



Fig. 1. Positioning of patient and measurement with a caliper to obtain accurate distance between axilla (first stimulation point) and Erb's point (second stimulation point). Recordings are obtained with electrodes over biceps motor point and abductor digiti minimi motor point. (From Pennsylvania Hand Center LTD; with permission.)

rately with a caliper (Fig. 1). The motor nerve conduction velocity can be calculated subsequently (velocity = distance/time). At this point the responses also may be evaluated for possible loss of motor amplitude crossing the specific segment.

Responses not complying with the chosen statistical or comparative norms are assessed as abnormal and suggestive of two possible pathologies: a demyelinating process (slowing of nerve conduction velocity) or loss of axons (loss of motor units resulting in decreased amplitude of response). Each cord segment (lateral, posterior, or medial cords) can be examined in this fashion.

Cervical root stimulation

Specific evaluation at trunk level of the brachial plexus is, on the other hand, more controversial. This technique consists first of testing the cord levels, as described earlier. Next stimulation is attempted at the C5–C6/upper trunk, C7/middle trunk, or C8–T1/lower trunk, root levels as appropriate [9]. This requires the insertion of a monopolar needle electrode to a site near the appropriate nerve root in the neck. The needle is connected to the negative/active pole of the stimulator. Next, either a surface electrode or a second needle electrode is placed at a site near the first needle electrode and is attached to the positive/passive pole of the stimulator. Stimulation is performed at that root level as usual, using a supramaximal stimulus to obtain a maximal amplitude response. Motor distal latency and

amplitude of response are recorded at the appropriate distal muscle.

Latency for that segment is obtained by subtracting the supraclavicular latency from the root level latency. This is sometimes known as differential motor distal latency. The differential latency and the motor amplitudes of response are compared with the statistical norms and the contralateral side for that segment. Responses not complying with these norms are assessed as abnormal and are suggestive of segmental demyelination, axonal loss, or both, for the tested trunk level segment (upper/middle/lower).

The cervical nerve root stimulation technique has significant limitations. Patient tolerance and safety (risk versus gain) are two of them. Many patients cannot tolerate this procedure because of severe pain and passing out. Consideration of patient safety also exists; in attempting to insert the needle close to the nerve root, the root is at risk for being impaled and the nearby apex of the lung can be punctured. Although these dangers can be limited by proper technique, neither risk can be eliminated completely.

Other factors deter from the value of cervical root stimulation. The actual small and variable length of each component of the plexus, and in particular the trunks, makes it uncertain what exactly is being measured. In addition, the high level of stimulation required leads to “volume conduction” (the higher the electrical stimulus intensity, the further it spreads). Due to the spread of the stimulus, the measured length of the nerve segment may be inaccurate.

F-wave

Because of the technical and anatomic difficulty in studying proximal nerve lesions, the use of F-waves has been proposed as a solution. The F-wave is believed to result from the “backfire” of antidromically activated anterior horn cells [10].

The F-wave is recorded by placing the active (anode) of a surface-recording electrode over the motor point and the reference (cathode) lead over the tendon of that muscle (Fig. 2). This represents the neural pathway to be examined (eg, ulnar nerve/medial cord/lower plexus). A supramaximal stimulus is applied to a point along the course of the nerve. The M response and F-wave are recorded and latencies are measured from the stimulus artifact to the onset of the evoked potential. Generally the shortest latency after several responses (usually more than 10) is recorded as the

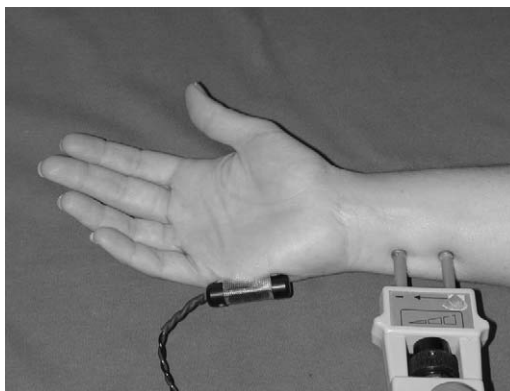


Fig. 2. Set-up for F-wave testing of ulnar nerve/lower trunk. Same electrode position over abductor digiti minimi as Fig. 1 is used for recording. Stimulation is over ulnar nerve at the wrist. (From Pennsylvania Hand Center LTD; with permission.)

F-wave response; this minimizes time-related variability. Latencies also should be compared with the contralateral limb or other nerves in the same limb to increase sensitivity of this test.

The F-wave is proposed to access motor conduction along the most proximal segment of the nerve, however, “The inherent variability of the latency and configuration makes the use of F-wave less precise than that of direct compound muscle action potentials or M-response determination” [10]. The shortcomings of F-wave testing include the following:

1. By definition, the F-wave is the shortest latency measured. In other words, one measures the response from only one single motor neuron. In segmental neuropathies in which all neurons are not affected equally, therefore, one may in theory be measuring the latency of the only healthy neuron in the entire nerve.
2. F-wave represents motor component fibers only.
3. Much of the literature on the use of F-wave testing for TOS finds these studies to be of “limited utility” [11].

For these reasons, F-wave measurements are not as useful in the diagnosis of brachial plexus problems as may seem on theoretic grounds.

Sensory testing

Sensory testing is accomplished in the traditional manner for that component of mixed nerves

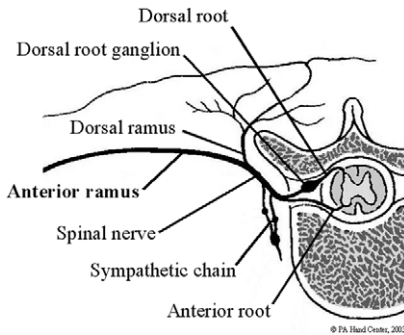


Fig. 3. Cross-section of spinal cord and spinal nerve. (From Pennsylvania Hand Center LTD; with permission.)

(median, ulnar, and radial) and purely sensory nerves (medial and lateral antebrachial cutaneous nerves), except the interpretation of these results differs from what might be considered standard testing.

At the cervical spine level, the dorsal sensory root ganglia are located within the neural foramina (Fig. 3). The nerve structures located proximal to the dorsal root ganglia are referred to as preganglionic, whereas the structures located distal to the dorsal root ganglia are referred to as postganglionic. The nerve roots (anterior and posterior), which are within the vertebrae, are therefore preganglionic; the spinal nerve and rami (anterior and posterior) are postganglionic [12]. Confusing as it may be, the most proximal structures in the brachial plexus are rami but are generally referred to as nerve roots.

Differential assessment of preganglionic (root avulsion) versus postganglionic (brachial plexus) pathology is possible with sensory nerve conduction studies (S-NCS). These also can differentiate between complete and incomplete brachial plexus lesions [10,11,13–17].

S-NCS is an indirect method that depends only on axonal changes (decreased amplitude). This is in contrast to motor nerve conduction studies (M-NCS), in which direct changes of demyelination (slowing) can be detected in addition to axonal changes (decreased amplitude).

The sensory nerve action potential (SNAP) amplitudes measured by S-NCS are generally diminished in postganglionic, incomplete lesions (ie, brachial plexus) and are present and normal in preganglionic lesions (ie, complete nerve root avulsion lesions). Because none of the distal sensory nerve fibers travels through all of the

proximal segments of the brachial plexus, however, no single sensory nerve response can assess all of the brachial plexus components.

Some investigators have suggested the use of typical/routine sensory NCS (ie, median, ulnar, and radial nerves) for the diagnosis of TOS [18,19]. Others propose less typical S-NCS to more thoroughly assess lesions within the brachial plexus [8,11,20–22]. Ferrante and Wilbourn tried to define more specifically distal sensory nerve conduction response parameters as (indirect) indicators of proximal sensory nerve axonal loss lesions within the brachial plexus [23]. They recommend testing of the lateral antebrachial cutaneous nerve (LAC) and thumb median and radial nerves for suspected upper plexus lesions. For suspected middle plexus lesions, they use the median nerve in the long finger. For the lower plexus they focus on the ulnar nerve in the small finger and on the ulnar dorsal sensory and medial antebrachial cutaneous nerves (MAC) [23].

A caveat to indirect sensory testing is that the sensory component testing of the median, ulnar, and radial nerves must be clear of distal segment involvement; axonal loss distally otherwise could be misinterpreted as a decrease in amplitude at the more proximal plexus level. The high frequency of distal neuropathy makes these procedures unreliable if the above precautions are not observed. This would not be a likely factor in testing the more proximal sensory nerves as discussed earlier (MAC and LAC). Difficulties in recording these responses, however, can be expected in patients with obesity or lymphedema [24].

Somatosensory evoked potentials

Somatosensory evoked potentials (SSEPs) measure sensory conduction latencies and amplitudes in the peripheral nerves through the brachial plexus, cervical roots, posterior spinal cord, and the subcortical sensory neural pathways. Recording electrodes are placed at specific standard locations, such as the elbow, axilla, supraclavicular space, spinous process of the second cervical vertebra, and scalp. Distal stimulation of the median, ulnar, and radial nerves is performed at the wrist/forearm. Sensory nerve latencies and amplitudes of response are recorded; inter-site times can be calculated by subtraction. These data are evaluated statistically and relatively (side to side). Abnormalities are correlated with the appropriate segment and likely location of a lesion [7].

The primary negative factor regarding SSEP seems to be the lack of research showing its usefulness in differentiation or localization of lesions in the brachial plexus. In fact, Aminoff et al describe SSEPs (and F-waves) as being of “limited utility” [11]. Also, “There would likely be a tremendous dilution effect by many centimeters of normal functioning nerve” [4] as compared with a small length of compression at the thoracic outlet level. Other technical considerations, such as very small working amplitudes, large margin of error caused by factors such as skin resistance, and variation in electrode placement further add to the “limited utility” of this procedure.

Author’s preference

At the Pennsylvania Hand Center, we choose an eclectic approach. We favor procedures and techniques that seem most justified by several criteria: foundation in laboratory and clinical research, correlation with clinical complaints and signs, medical ethics (testing what is needed based on clinical assessment), patient safety versus possible gain from results, and common sense considerations of physics and physiology and their limitations.

Our most consistently productive (correlation with clinical picture) and efficacious (medically and with regard to patient safety) protocol for TOS is as follows.

1. Routine sensory testing of the distal components of the median, ulnar, and radial nerves to rule out distal segmental neuropathy. If negative for distal neuropathy, these findings then may be applied toward possible correlation with proximal, TOS/brachial plexus level pathology in evaluation of sensory nerve action potential (SNAP) amplitudes. If clinically indicated, medial antebrachial cutaneous (MAC) and lateral antebrachial cutaneous (LAC) also may be examined.
2. Routine motor testing of the distal components of the median and ulnar nerves (and the radial nerve, if indicated), the lateral cord of the upper plexus, the medial cord of the lower plexus, and, if indicated, the posterior cord of the middle plexus.
3. Routine EMG of the muscle groups of both upper extremities representative of the C4 through T1 nerve root distributions and the median, ulnar, and radial nerve distributions. When indicated, the study includes the

musculocutaneous, axillary, and proximal shoulder girdle musculature.

4. In a patient willing and desirous of surgery with a strongly suggestive history or clinical findings but with no objective electrophysiologic evidence, cervical nerve root level testing may be considered.
5. SSEPs also are considered if already performed. We do not obtain SSEPs routinely because of the unreliability of this procedure.
6. We then grade our findings in severity and relate them to previous electrodiagnostic studies (Table 1).

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