Timing and Appropriate Use of Electrodiagnostic Studies

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KEYWORDS

• Electrodiagnostic study • Nerve conduction study • Electromyography • Nerve injury
• Nerve compression • Carpal tunnel syndrome • Cubital tunnel syndrome • Indications

KEY POINTS

- Electrodiagnostic studies (EDS) consist of nerve conduction studies (NCS) and electromyography (EMG).
- Both NCS and EMG are needed to perform a complete electrodiagnostic study.
- Specific changes in EDS are used to characterize nerve pathology.
- Nerve injury and healing can be monitored with EDS, which provides variable information at differing time intervals.
- EDS is recommended by multidisciplinary groups in the diagnosis of carpal tunnel syndrome, yet this remains controversial.
- Prognostic information regarding mononeuropathy and polyneuropathy can be determined with EDS.
- EDS should not be used to diagnose radiculopathy.

Electrodiagnostic studies (EDS) are powerful tools used to objectively examine the physiologic status of a nerve. These consist of nerve conduction studies (NCS), which directly examine motor and sensory function of the nerve, and electromyography (EMG), which examines motor unit, action and spontaneous potentials in the muscle. Together these studies enable characterization, localization, and duration of nerve pathology and healing. An understanding of the appropriate timing and use of EDS is essential in the application of these diagnostic studies.

BASICS OF EDS

EDS consist of two components: NCS and needle EMG. These studies are used in concert to characterize and localize a pathologic lesion in a peripheral nerve. Each set of tests provides critical data, making both tests necessary to fully understand the condition.

Nerve Conduction Studies

NCS consists of stimulating a peripheral nerve and recording the response elsewhere on the nerve or associated muscle using a recording electrode.[1] Measurements are only as good as the examiner in making approximations of the locations of the nerve and its target. As a result, testing proximal and deeper structures is more challenging. When a nerve is stimulated, it is important that all elements of the nerve are depolarized. This is achieved by successively increasing the levels of current until the recorded potentials no longer increase despite an increasing stimulus. A continued increase in stimulus can result in stimulation of surrounding nerves and false recordings. The recording electrodes consist of an active and

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inactive electrode placed a known distance from each other. The recorded potentials are measured as a function of time and voltage, and compared with laboratory normative values or published normal values.[2]

NCS can be divided into motor and sensory studies. Motor conduction studies are performed by placing the active recording electrode over the muscle belly and inactive recording electrode over the insertion of the muscle with the stimulation occurring over at least two points on the motor nerve. The recorded potential in the muscle represents the summated depolarization of all the muscle fibers innervated by the nerve called the compound muscle action potential (CMAP). The CMAP waveform is broken into many parts, including amplitude, latency, and conduction velocity. The amplitude is measured from the baseline to the negative peak, time from stimulation to initial deflection of the waveform is latency, and the difference in latency between proximal and distal sites of stimulation divided by the distance between those sites is conduction velocity.

The deep nature of the brachial plexus and cervical spine makes NCS difficult to perform on nerves and nerve structures in this region, given the importance of precise placement of the stimulating and recording electrodes. One additional study can be performed examining for a second CMAP called the F wave. This CMAP is created by the stimulation of a small number of anterior horn cells from the antidromic (the action potential that runs in the opposite direction from native transmission) action potential created at the time of nerve stimulation. This CMAP is much smaller with a greater latency allowing for it to be differentiated from the initial CMAP. The characteristics of the F wave can be analyzed and compared with normative values identifying pathologic features of proximal nerves and nerve structures.

Sensory studies can be performed as isolated studies or as part of a mixed nerve study. In a mixed study, the nerve of investigation creates motor and sensory action potentials when stimulated. The sensory nerve action potentials (SNAP) are measured in a similar fashion to the CMAP, except the two recording electrodes are placed over the nerve of interest instead of a muscle belly. There are marked technical differences between these two studies. SNAPs can be performed in the orthodromic or antidromic direction. Sensory axons have greater variability in the size and myelination compared with motor axons, which are more uniform.[3] Additionally, the volume of tissue (the nerve as compared with a muscle belly) stimulated is much smaller making the amplitude much smaller, and thus a greater signal-to-noise ratio. This makes clear results more challenging to identify.

Needle EMG

Needle EMG records electrical signals in motor units within muscles by inserting a recording electrode into a muscle. EMG waveforms are divided into spontaneous and voluntary waveforms. Spontaneous waveforms are action potentials recorded on insertion of the needle, with needle movement, and at rest. Voluntary waveforms are created by voluntary muscle contraction. Differences in waveforms help to localize and characterize pathologic changes.[4]

Spontaneous waveforms relevant to hand surgery include insertional activity, fasiculation potentials, fibrillation potentials, myotonic discharges, complex repetitive discharges, myokymic discharges, neuromyotonic discharges, cramp potentials, and synkinesis. Each has a characteristic waveform and associations with normal function or pathology.

Voluntary motor unit potentials (MUP) are the action potentials recorded from motor units innervated by a single anterior horn cell. They occur in a semirhythmic pattern with specific identifiable characteristics to the appearance of the waveform. These include the duration, amplitude, and number of turns or phases (ie, the number of times the action potential crosses the baseline plus one). Multiple variables contribute to the appearance of the MUP other than pathology. These include the patient's age, muscles studied, needle position within the muscle, temperature, and strength of activation.

NERVE INJURY AND TRAUMA

Nerve repair occurs by three mechanisms: (1) remyelination, (2) collateral sprouting from preserved axons, and (3) regeneration from the site of injury. In a neuropraxic injury, the axon is intact but there is focal demyelination. Remyelination begins almost immediately on removal of the offending agent, with recovery ranging hours to months. If axonotmesis occurs, the severed axons seal over and the stump swells because of anterograde axonal transport within hours, and fragmentation and digestion of axons and myelin begin in the first few days. If less than 30% of the axons are injured, collateral sprouting occurs by Day 4 with axons traversing the segment and rearrange by Days 8 to 15. Complete healing takes 2 to 6 months. If more than 90% of axons are injured, the distal nerve segment disintegrates by wallerian degeneration and regeneration occurs from the proximal nerve end at the site of injury.[5] Injuries with 30%