NCS & EMG Guide

Nerve conduction studies and needle EMG — normal values and pathological findings. Ahmed Koriesh, MD · neuro.wiki. Reference values are laboratory/temperature-dependent.

Nerve conduction studies (NCS) and needle electromyography (EMG) together make up the **electrodiagnostic (EDX) study** — an extension of the neurological exam that localizes and characterizes disorders of the peripheral nervous system: anterior horn cell, root, plexus, peripheral nerve, neuromuscular junction (NMJ), and muscle.

Normal values are laboratory- and temperature-dependent. The reference ranges below are widely used adult values (after Preston & Shapiro); always interpret against your own lab's limits, and warm a cool limb — low temperature falsely prolongs latencies, slows conduction velocity, and increases amplitudes (rule of thumb: ~1.5–2.5 m/s slowing per °C below 32°C at the skin).

- **Motor study:** stimulate the nerve at two (or more) points and record the compound muscle action potential (CMAP) over the belly of the target muscle (belly-tendon montage). Use **supramaximal** stimulation (increase current until the response no longer grows, then add ~20%).
- **Sensory study:** stimulate and record along a pure sensory nerve (orthodromic or antidromic) to obtain the sensory nerve action potential (SNAP).
- Measure off the same landmarks each time; keep the limb warm; mark distances accurately (CV = distance ÷ proximal-minus-distal latency).

- **Amplitude** — number of functioning axons/fibers. Low = *axon loss* (or conduction block proximally). CMAP in mV, SNAP in µV.
- **Distal latency** — slowest distal conduction; for motor it also includes NMJ transmission and muscle activation (ms).
- **Conduction velocity (CV)** — speed of the fastest fibers; reflects *myelination* (m/s); needs two stimulation sites for motor nerves.

Nerve (record)	Distal latency	Amplitude (CMAP)	Conduction velocity
Median (APB)	≤ 4.4 ms	≥ 4 mV	≥ 49 m/s
Ulnar (ADM)	≤ 3.3 ms	≥ 6 mV	≥ 49 m/s
Peroneal / fibular (EDB)	≤ 6.5 ms	≥ 2 mV	≥ 44 m/s
Tibial (AH)	≤ 5.8 ms	≥ 4 mV	≥ 41 m/s

Nerve	Peak latency	Amplitude (SNAP)	Conduction velocity
Median (digit II)	≤ 3.5 ms	≥ 20 µV	≥ 50 m/s
Ulnar (digit V)	≤ 3.1 ms	≥ 17 µV	≥ 50 m/s
Radial (snuffbox)	≤ 2.9 ms	≥ 15 µV	≥ 50 m/s
Sural (lateral ankle)	≤ 4.4 ms	≥ 6 µV	≥ 40 m/s

SNAP and the DRG: the sensory cell body sits in the dorsal root ganglion, *outside* the cord. A lesion **proximal** to the DRG (e.g., radiculopathy) leaves the SNAP **normal**; a lesion at or distal to the DRG (plexopathy, peripheral neuropathy) reduces it — the key to separating radiculopathy from plexopathy.

Study	What it tests	Normal / key abnormality
F-wave	Proximal motor segment (antidromic to the anterior horn and back)	Upper limb ≤ ~31 ms, lower limb ≤ ~56 ms (height-dependent). Prolonged/absent early in acquired demyelination (GBS) and proximal lesions.
H-reflex	S1 reflex arc (tibial → soleus); the electrical ankle jerk	≤ ~34 ms (height-dependent); side-to-side difference >1.5 ms abnormal. Prolonged/absent in S1 radiculopathy and proximal tibial/sciatic lesions.
RNS — slow (2–3 Hz)	NMJ safety factor	Decrement >10% → postsynaptic defect (myasthenia gravis).
RNS — fast / post-exercise	Presynaptic NMJ reserve	Increment >60–100% → presynaptic defect (LEMS, botulism).

A concentric needle samples muscle in three phases — at **insertion**, at **rest**, and during **voluntary activation**.

Finding	Significance
Fibrillations & positive sharp waves	Active denervation (axon loss) or active myopathy (esp. inflammatory/necrotizing). Appear ~2–3 weeks after axon injury.
Fasciculation potentials	Spontaneous motor-unit discharges — prominent in motor neuron disease; also benign, and in radiculopathy/neuropathy.

Finding	Significance
Myotonic discharges	Waxing-and-waning “dive-bomber” sound — myotonic dystrophy, myotonia congenita, Pompe, some channelopathies.
Complex repetitive discharges (CRDs)	Chronic neuropathic or myopathic processes.
Myokymic / neuromyotonic discharges	Radiation plexopathy, demyelination, peripheral nerve hyperexcitability (Isaacs).
Reduced insertional activity	Fibrosis or fatty replacement (end-stage muscle).

Process	MUAP morphology	Recruitment
Neuropathic (chronic)	Large amplitude, long duration, polyphasic (reinnervation)	Reduced — few units firing fast
Myopathic	Small amplitude, short duration, polyphasic	Early / rapid — many small units for little force
NMJ disorder	Unstable / varying MUAPs (moment-to-moment)	Normal to early

Pattern	NCS	EMG
Axonal neuropathy	Low amplitude (CMAP/SNAP); CV and latencies relatively preserved	Fibrillations/PSWs; large, long, polyphasic MUAPs; reduced recruitment
Demyelinating neuropathy	Slow CV, prolonged distal latencies, prolonged/absent F-waves, conduction block & temporal dispersion (acquired)	Less denervation unless secondary axon loss
Myopathy	Usually normal (CMAP may be low in severe/distal myopathy; SNAPs normal)	Small, short, polyphasic MUAPs; early recruitment; ± fibs in inflammatory/necrotizing myopathy
Motor neuron disease	Low CMAP amplitudes; normal sensory studies	Widespread fibrillations + fasciculations; large MUAPs; reduced recruitment across multiple regions/segments
NMJ — postsynaptic (MG)	Normal routine NCS; decrement on slow RNS	Unstable MUAPs, ± short-duration MUAPs
NMJ — presynaptic (LEMS)	Low CMAP amplitudes; marked post-exercise increment	Unstable MUAPs
Radiculopathy	Normal SNAP (lesion proximal to DRG); may have low CMAP if severe	Denervation in a myotomal distribution incl. paraspinals

Diagnosis	Hallmark findings
Carpal tunnel syndrome	Prolonged median sensory (then motor) distal latency; median-vs-ulnar or median-vs-radial comparison across the wrist is most sensitive. EMG of APB only in moderate-severe cases.
Ulnar neuropathy at the elbow	Focal CV slowing >10 m/s and/or conduction block across the elbow segment; reduced ulnar SNAP; EMG of FDI & FCU.
Peroneal neuropathy (fibular head)	Focal slowing/conduction block across the fibular head; EMG of tibialis anterior, sparing the short head of biceps femoris.
Cervical / lumbosacral radiculopathy	Normal SNAPs; needle denervation in ≥2 muscles of one myotome (incl. paraspinals) supplied by different peripheral nerves.
Length-dependent axonal polyneuropathy	Low/absent sural & distal SNAPs, low distal CMAPs, distal-predominant denervation (feet first). Diabetes the most common cause.
GBS (AIDP)	Acquired demyelination: prolonged/absent F-waves early, conduction block, temporal dispersion, prolonged distal latencies, slow CV.
CIDP	Same demyelinating features, chronic/relapsing, with secondary axon loss over time.
ALS / motor neuron disease	Normal sensory NCS; widespread active + chronic denervation across ≥3 spinal regions (bulbar/cervical/thoracic/lumbosacral).
Myasthenia gravis	Decrement on slow RNS; single-fiber EMG shows increased jitter/blocking (most sensitive).
LEMS	Low resting CMAP; >60–100% increment after brief exercise / on fast RNS.

Root	Representative muscles	Peripheral nerve(s)
Cervical / upper limb		
C5–C6	Deltoid, biceps, brachioradialis	Axillary, musculocutaneous, radial
C6–C7	Pronator teres, flexor carpi radialis, triceps	Median, radial
C8–T1	First dorsal interosseous, abductor pollicis brevis, FDP	Ulnar, median
Lumbosacral / lower limb		

Root	Representative muscles	Peripheral nerve(s)
L2–L4	Vastus medialis/lateralis, iliopsoas, adductors	Femoral, obturator
L4–L5	Tibialis anterior, extensor hallucis longus, gluteus medius	Peroneal/fibular, superior gluteal
S1–S2	Gastrocnemius, soleus, abductor hallucis	Tibial

Always sample paraspinal muscles and confirm a level with ≥2 muscles from different peripheral nerves sharing the same root before calling a radiculopathy.

1. Is the process **neuropathic, myopathic, NMJ, or anterior-horn**?
2. If neuropathic: **axonal vs demyelinating**, and **focal vs generalized** (sensory, motor, or both)?
3. **Localize**: nerve, plexus, or root — use the SNAP (normal in radiculopathy) and the pattern of muscle involvement (single nerve vs myotome vs diffuse).
4. Correlate with the clinical exam — EDX is an *extension* of the exam, not a substitute for it.