LETTER TO THE EDITOR

Nerve Conduction Studies: What an Intensivist should Know?

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Dear Editor,

In clinical scenarios with acute-onset neuromuscular weakness, reports of nerve conduction studies (NCS) form one of the crucial diagnostics, aiding in treatment decisions like administration of intravenous immunoglobulin therapy or therapeutic plasma exchange, as these interventions reduce mortality significantly.[1,2] There might be a situation during that period, wherein the intensivist may need to take an early decision based on the available clinical scenario with the NCS report in hand.[2,3] There is a lack of simplified rules or algorithms in critical care literature that can help the intensivist in such decision-making.[4,5] In the intensive care unit, multiple factors like age, sex, height, and other physical characteristics and ongoing treatment like sedatives, analgesics, muscle relaxants, and antimicrobials influence the NCS.[1,5] Through this letter, we provide approximate cutoff values for an adult in a single image based on which bedside interpretation can be done.

This algorithm (Fig. 1) even provides information relating to prerequisites to be considered before shifting for such studies such as the absence of peripheral edema, pacemaker, and hypothermia, which can influence the report. It provides three simple rules by looking at conduction velocity (CV), amplitude, and onset latency. Sensory nerves have highest CV with upper limb approximately 60 meters/second (UL) > lower limb (LL) with approximately 50 meters/second (m/s). Motor nerves have approximately 50 m/s in UL and 40 m/s in LL, respectively, creating rule I as of the 60-50-40 rule.[4] Sensory nerves have higher amplitude and peak latency than their motor components. After 20 years, the CV reduces by 2–4 m/s in sensory nerves and 0.4–1.7 m/s per decade in motor nerves respectively. The CV reduces by more than two-thirds of cutoff values (COV) in demyelinating pathology and less than two-thirds of COV in axonal pathology. Apart from this, in axonal pathology,

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NERVE CONDUCTION STUDIES: What an intensivist should know

Rule I: Conduction velocity (m/s): 60-50-40 rule: 60 → UL sensory; 50 → LL sensory
50 → UL motor; 40 → LL motor

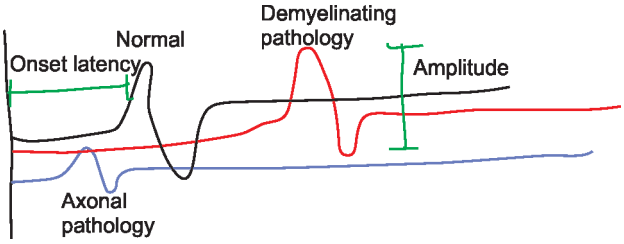
Rule II: Amplitude (mV): 6-4-2 rule: ulnar (UL), median (UL), and common peroneal (LL), respectively

Rule III: Onset latency (ms): 3-6 rule: radial (UL) and tibial (LL), respectively

Nerve (motor)	*Onset latency (ms)*	*Amplitude (mV)*	*Velocity (m/s)*
Median (APB)	≤4.4	≥4	≥50
Ulnar (ADM)	≤3.3	≥6	≥50
Radial (EIP)	≤2.9 (~3)	≥2	≥50
CPN (EDB)	≤6.5	≥2	≥40
Tibial (AHB)	≤5.8 (~6)	≥2	≥40
Nerve (sensory)	*Peak latency (ms)*	*Amplitude (mV)*	*Velocity (m/s)*
Median (Digit II)	≤3.5	≥20	≥60
Ulnar (Digit V)	≤3.1	≥17	≥60
Radial (snuff box)	≤2.9	≥15	≥60
Sural (postankle)	≤4.4	≥6	≥50

When to shift for NCS:

1. No or minimal peripheral edema
2. No hypothermia (1m/s ↓ /1°C ↓)
3. No pacemaker (if so programming)



Pathology:

Demyelination: ≥2/3 reduction in velocity (legs <30; arms <40); onset latency ↑↑ in very proximal pathology/early-stage F-wave minimum latency—↑↑
Axonal loss: ↓↓ amplitude; CV in severe: <2/3 reduction in velocity (legs >30; arms >40); onset latency N/↑; F-wave minimum latency—N/↑

Fig. 1: Approach to interpretation of nerve conduction study (NCS) in an adult. m/s, meters/second; mV, millivolt; UL, upper limb; LL, lower limb; NCS, nerve conduction study; APB, abductor pollicis brevis; ADM, abductor digiti minimi; EIP, extensor indicis proprius; EDB, extensor digitorum brevis; AHB, abductor hallucis muscle; CV, conduction velocity

amplitude of motor nerves reduces significantly with COV being 6-4-2 millivolts (mV) in ulnar, median, and common peroneal nerves, respectively (rule II). Onset latency is approximately 3 milliseconds (ms) in radial (UL) and 6 ms in tibial (LL), respectively, labeled as the 3-6 rule as rule III.[1,6] Apart from this, in axonal pathology, the onset latency in milliseconds will be normal or slightly increased and so is F-wave minimum latency (late motor response testing in the proximal part of the nerve which indicates proximal demyelination), whereas these two are significantly affected in demyelinating pathology.[4] F-wave abnormality is useful as this is the first affected in the early stages of acute inflammatory demyelinating polyneuropathy (AIDP), in which even the CV and onset latency can be normal.

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