Basics with the Experts - Basics of Nerve Conduction Study and Needle EMG

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Nothing to disclose

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Outline of Session

8:00-8:05 am  5 min  Introduction

8:05-8:25 am  20 min  Nerve Conduction Basics & Approach to Study

8:25-8:55 am  30 min  Nerve Conduction Study Methods

8:55-9:25 am  30 min  Needle EMG Basics and Specialized Studies

9:25-9:30 am  5 min  Discussion, questions, and answers
Basics of Nerve Conduction and Approach to Study

- Speaker: M. Kian Salajegheh, MD

- Anatomy and physiology of the PNS
- Measurement of combined action potentials
- EDX changes in disease states
- Approach to the study
Upper and Lower Extremity Nerve Conduction Studies

• Speaker: Kelly G. Gwathmey, MD

• Review anatomy of nerves and their study technique
• Anomalous innervations
• Alternative studies
Specialized Studies and Basics of Needle EMG

- Speaker: Aaron Izenberg, MD
- Repetitive nerve stimulation
- Blink reflex
- Needle EMG basics
INTRODUCTION
Nerve Conduction Study & EMG

• Electrodiagnostic (EDX) studies are valuable tools for the assessment of neuromuscular disorders

• Numerous studies and various techniques are used today
  o Nerve conduction studies (NCSs)
  o Late responses (F response and H reflex)
  o Repetitive nerve stimulation (RNS)
  o Needle electromyography (EMG)
  o Specialized examination
EDX technical aspects

• Recording electrodes types
  o Ring or clamp electrodes
  o Bar or disk electrodes
  o Needle electrodes

• Recording
  o Active (G1) and reference (G2) electrodes
    • CMAP: mostly belly (G1) and tendon (G2) montage
    • SNAP: usually 3-4 cm apart
    • Needle usually concentric
EDX technical aspects

• **Grounding**
  - Obtain waveform free of artifact

• **Machine settings**
  - Gain (sensitivity)
  - Sweep speed
  - Filter settings

• **Stimulation** (red anode and black cathode)
  - Orthodromic vs. antidromic (for sensory)
  - Stimulation duration usually 0.1-0.2 ms
  - Intensity (usually supramaximal stimulation)
BASICS OF NCS AND APPROACH TO STUDY
Electrodiagnostic Studies

• **Critical tools** for the assessment of patients with neuromuscular disorders

• Mainly **extensions** of the **clinical evaluation**
  - Obtain focused **history and exam**
  - Form clinical impression with **differential Dx**
  - **Design** study NCS > Needle EMG
  - Report exam with clinical impression in mind
Nerve Anatomy & Physiology

• Knowledge of peripheral nervous system anatomy key to optimal localization (main role of EDX)

• Knowledge of nerve and muscle physiology important to determine type and timing of lesion
Peripheral Nervous System

31 pairs:
- 8 cervical
- 12 thoracic
- 5 lumbar
- 5 sacral
- 1 coccygeal
Peripheral Nervous System

Dorsal Ramus $\rightarrow$ Paraspinal muscles

Ventral Ramus
  $\rightarrow$ Brachial Plexus $\rightarrow$ Peripheral Nerves
  $\rightarrow$ Intercostal Nerves
  $\rightarrow$ Lumbosacral Plexus $\rightarrow$ Peripheral Nerves
Nerve & Muscle Physiology

• Axonal membrane is electrically active with resting membrane potential

• Depolarization leads to an action potential propagating in both directions away from sight of depolarization

• We record only what we stimulate and capture – importance of correct stimulation and recording sites
Combined Action Potentials (AP)

- Combined motor nerve AP (CMAP)
- Mixed nerve AP (MNAP)
- Sensory nerve AP (SNAP)

Negative deflection is upwards
Positive deflection is downwards
Combined AP - Amplitude

- **Amplitude**
  - Usually *baseline-peak* but also peak-peak
  - Reflective of
    - **SNAP** and **MNAP**: all *nerve fibers* stimulated and captured
    - **CMAP**: all *nerve fibers* stimulated and *muscle fibers* stimulated and captured

**Graph:**
- Stimulus
- Amplitude

**Legend:**
- **CMAP** - in mV
- **SNAP & MNAP** - in μV
Combined AP - Amplitude

- Amplitude reduction
  - SNAP & MNAP: loss of nerve fibers
  - CMAP: loss of motor nerve or muscle fibers or neuromuscular junction (NMJ) block

- Lesion location (after Wallerian degeneration)

Lesions proximal to the DRG not measured by the SNAP
Combined AP - Latency

- Latency (time)
  - Peak: Stimulus to first negative peak
  - Onset: Stimulus to initial negative deflection
    - Reflective of fastest conducting fibers
    - Used for calculation of conduction velocity

Conduction velocity (m/s) = \( \frac{\text{Distance}}{\text{Latency}} \)

In milliseconds (ms)
**CV - CMAP vs SNAP**

**SNAP & MNAP:** CV = Distance stimulus to recording/onset latency

**CMAP:** Onset latency includes NMJ and muscle conduction

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**A. Stimulate**

**B. Record**

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**SNAP & MNAP:** CV = Distance A-B / latency A-B

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**CMAP:** CV = Distance stimulus to recording/onset latency
Combined AP - Duration

- Duration
  - Negative peak onset to first baseline crossing (i.e. negative peak duration)
  - Marker of synchrony of action potentials
  - SNAP duration typically 1.5 ms

In milliseconds (ms)
EDX Changes in NMD Disorders

- Axonal loss (NCS)
  - Reduction in amplitude
    - CMAP 3–5 days
    - SNAP 6-10 days
  - Preserved latency and CV
    - Mild reduction with loss of fastest conducting fibers (not within demyelinating range)
- Axonal loss (EMG)
  - Acute: reduced recruitment pattern
  - Subacute: appearance of membrane irritability
  - Chronic (months): large and long MUAP
EDX Changes in NMD Disorders

• Demyelination (NCS)
  o Reduced CV <75% LLN
    • ~ 35 m/s UE and 30 m/s LE
  o Prolonged latency >130% ULN
  o Preserved amplitude except for
    • Conduction block
EDX Changes in NMD Disorders

- Temporal dispersion (Increased CMAP duration)

Both conduction block and temporal dispersion are markers of acquired demyelination.

Within few days from lesion onset may be due to pseudo-conduction block (repeat study ~1 week)
EDX Changes in NMD Disorders

• Demyelination (EMG)
  o Should only show reduced recruitment pattern
  o Most lesions have some axonal damage as well

• Late responses
  o Important to look for lesions proximal to the usual sites of stimulation in motor studies
  o Evaluate the entire length of the nerve
    • F waves: motor nerve
    • H reflex: sensory and motor nerves
APPROACH TO EDX:
CLINICAL LOCALIZATION IN THE PNS
NMD Presentation

• Sensory
  ▫ Roots (dorsal)
  ▫ Dorsal sensory ganglion
  ▫ Nerve (sensory fiber)

• Motor
  ▫ Motor neuron (anterior horn)
  ▫ Roots (ventral)
  ▫ Nerve (motor fiber)
  ▫ Neuromuscular junction
  ▫ Muscle
NMD Presentation Sensorimotor

• Sensory
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NMD Presentation Sensorimotor

- **Radiculopathy**
  - Disc
  - Clinical
    - Radicular pain
    - Weakness in myotome, sensory change in dermatome

- **Polyradiculopathy**
  - Diffuse meningeal process
  - Clinical
    - Diffuse pain and motor/sensory change
    - May involve cranial nerves and cognition
NMD Presentation Sensorimotor

- Radiculopathy/polyradiculopathy

NCS/EMG:
- Normal SNAP (lesion central to DRG)
- Possible low CMAP amplitude (depending on level)

- Denervation in muscles sharing myotome (s)
- Denervation of parapsinal muscles
NMD Presentation Sensorimotor

• Mononeuropathies
  o Initial demyelinating: “Compressive”
    • Median at wrist “carpal tunnel syndrome”
    • Ulnar at elbow “cubital tunnel syndrome”
    • Peroneal (fibular) neuropathy at fibular head
  o Axonal
    • Traumatic
  o Clinical
    • Weakness and sensory change in distribution of a single nerve
NMD Presentation Sensorimotor

- Mononeuropathies

**NCS/EMG:**
- Focal demyelination and/or low SNAP and CMAP amplitude in single nerve territory

  - Denervation in muscles innervated by the nerve (if axonal)

  - Exact localization only possible with demonstration of focal demyelination
NMD Presentation Sensorimotor

• Multiple mononeuropathies
  o Demyelinating and/or axonal
    • Vasculitis “mononeuritis multiplex”
    • Infiltration (lymphoma)
    • Infection (Lyme, leprosy)
    • Granulomatous (sarcoid)
    • CIDP variants
    • Hereditary (HNPP)
  o Clinical
    • Weakness and sensory change in distribution of multiple individual nerves or asymmetric polyneuropathy (due to lesion confluence)
NMD Presentation Sensorimotor

• Generalized polyneuropathy
  o Distal symmetric length dependent sensory > motor
    • Diabetic
    • Toxic (alcohol and drugs)
  o Non-length dependent sensory < or = motor
    • Autoimmune (GBS or CIDP)
    • Hereditary
  o Clinical
    • Diffuse weakness and sensory changes
    • Look for signs of chronicity (pes cavus and hammertoes)
    • Family history important (most AD)
NMD Presentation Sensorimotor

• Generalized polyneuropathy

NCS/EMG:
- **Study enough nerves** and **muscles** to establish pattern and symmetry
- Determine axonal vs demyelinating
- If demyelinating determine hereditary vs acquired
- Consider proximal stimulation and late responses if suspicion for demyelinating

NCS and EMG are **normal** in **small fiber sensory neuropathy**

**Cold nerves are slow** so pay attention to limb temperature and warm them up
NMD Presentation Sensorimotor

• Plexopathies
  o Brachial
    • Brachial plexitis “Parsonage-Turner syndrome”
    • Radiation (may have myokymia)
  o Lumbosacral
    • Radiation
    • Retroperitoneal process

  o Clinical
    • May start with shoulder or hip pain
    • Diffuse weakness and sensory changes in single limb
NMD Presentation Sensorimotor

• Plexopathies

**NCS/EMG:**
- **Expanded study** of nerve and muscle in involved limb
- Consider **side to side** comparison
- **Myokymia** on exam and EMG suggestive of **radiation**
NMD Presentation Sensory

• Sensory
  ▫ Roots (dorsal)
  ▫ **Dorsal sensory ganglion**
  ▫ **Nerve (sensory fibers)**

• Motor
  ▫ Motor neuron (anterior horn)
  ▫ Roots (ventral)
  ▫ Nerve (motor fiber)
  ▫ Neuromuscular junction
  ▫ Muscle
NMD Presentation Sensory

• Ganglionopathy
  o Sjogren’s syndrome
  o Drugs (B6 toxicity, thalidomide)
  o Paraneoplastic (anti-Hu)
  o Autoimmune

  o Clinical
    • Non-length dependent, focal or generalized
    • All sensory modalities with severe sensory ataxia
NMD Presentation Sensory

• Ganglionopathy

**NCS/EMG**
- Non length dependent or asymmetric loss of SNAP amplitude
- Normal CMAP and EMG studies
NMD Presentation Motor

• Sensory
  ▫ Roots (dorsal)
  ▫ Dorsal sensory ganglion
  ▫ Nerve (sensory fiber)

• Motor
  ▫ Motor neuron (anterior horn)
  ▫ Roots (ventral)
  ▫ Nerve (motor fiber)
  ▫ Neuromuscular junction
  ▫ Muscle
NMD Presentation Motor

• Motor neuron disease
  o ALS
  o Spinal muscular atrophy (SMA)
  o Infections (polio, WNV)
  o Clinical
    • Muscle weakness and atrophy (regional, diffuse)
    • Bulbar and/or respiratory symptoms
    • Cramps and fasciculations
    • Upper motor neuron symptoms and signs in ALS
NMD Presentation Motor

• Motor neuron disease

NCS/EMG
- Normal SNAP amplitudes (usually)
- Reduced CMAP amplitude (usually asymmetric) with normal latency and CV
- Important to consider proximal stimulation and late responses if no upper motor neuron findings

- Denervation changes in muscles sharing myotome(s) and also bulbar/cranial
NMD Presentation Motor

• Neuropathy (motor fibers)
  o Demyelinating (multifocal mononeuropathy; MMN)
  o Axonal (GBS variant)
  o Hereditary

  o Clinical
    • Muscle weakness and possible atrophy
    • Distribution of multiple nerves or generalized distal>proximal (not myotomal)
NMD Presentation Motor

- Neuropathy (motor fibers)

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<tr>
<th>NCS/EMG</th>
<th>Similar to MND except for</th>
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<tbody>
<tr>
<td></td>
<td>- Demyelination in MMN</td>
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<td></td>
<td>- Denervation in distribution of nerves or distal&gt;proximal rather than myotomal</td>
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NMD Presentation Motor

• Neuromuscular junction disorder
  o Pre-synaptic
    • Lambert Eaton (LEMS)
    • Botulism
  o Post-synaptic
    • Myasthenia gravis

  o Clinical
    • Fluctuating and fatigable muscle weakness
    • Ocular and bulbar involvement
    • LEMS and botulism with autonomic symptoms
NMD Presentation Motor

- Neuromuscular junction disorder

NCS/EMG
- Slow repetitive nerve stimulation (RNS)
- Fast RNS for presynaptic (or check for abnormal post 10-second exercise facilitation)
- Routine NCS and needle EMG to exclude underlying muscle denervation/reinnervation or myopathy
NMD Presentation Motor

- Myopathy
  - Proximal > distal
    - Endocrine and Toxic
    - Polymyositis and dermatomyositis
  - Distal > or = proximal
    - Myotonic dystrophy type 1 and other inherited myopathies
    - Inclusion body myositis
  - Diffuse
    - ICU myopathy
    - Periodic paralysis
NMD Presentation Motor

- Myopathy
  - Clinical
    - Progressive muscle weakness and atrophy
    - Some episodic/periodic +/- triggers
    - Some with myotonia

NCS/EMG
- Normal SNAP
- Normal CMAP (unless distal myopathy)
- Myopathic units
- Membrane irritability and myotonic discharges in some (inflammatory, toxic,...)
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