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

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Financial Disclosure

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
  - o Obtain waveform free of artifact
- Machine settings
  - o Gain (sensitivity)
  - o Sweep speed
  - o Filter settings
- Stimulation (red anode and black cathode)
  - o Orthodromic vs. antidromic (for sensory)
  - o Stimulation duration usually 0.1-0.2 ms
  - o Intensity (usually supramaximal stimulation)

BASICS OF NCS AND APPROACH TO STUDY

Electrodiagnostic Studies

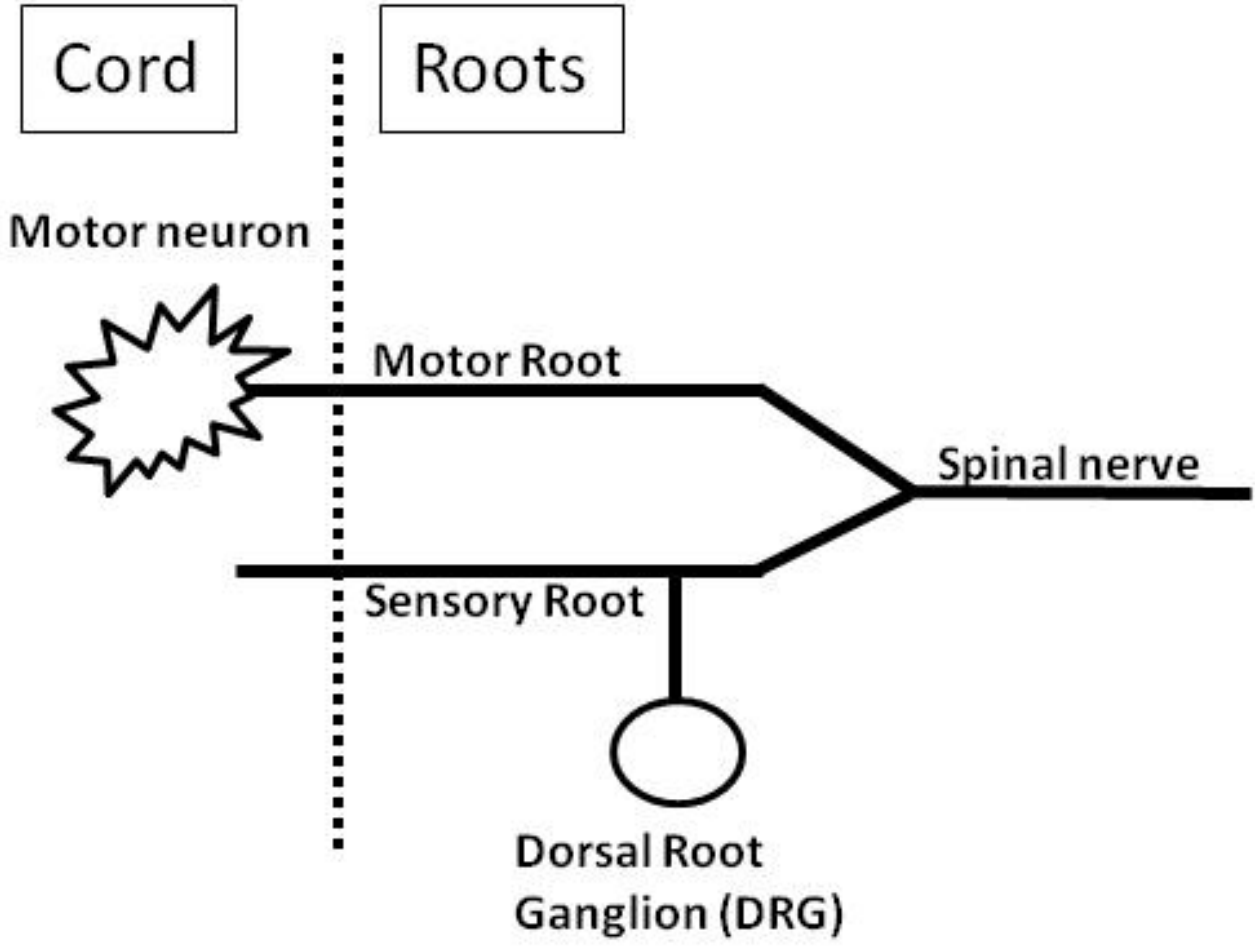
- Critical tools for the assessment of patients with neuromuscular disorders
- Mainly extensions of the clinical evaluation
  - o Obtain focused history and exam
  - o Form clinical impression with differential Dx
  - o Design study NCS > Needle EMG
  - o Report exam with clinical impression in mind

Nerve Anatomy & Physiology

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

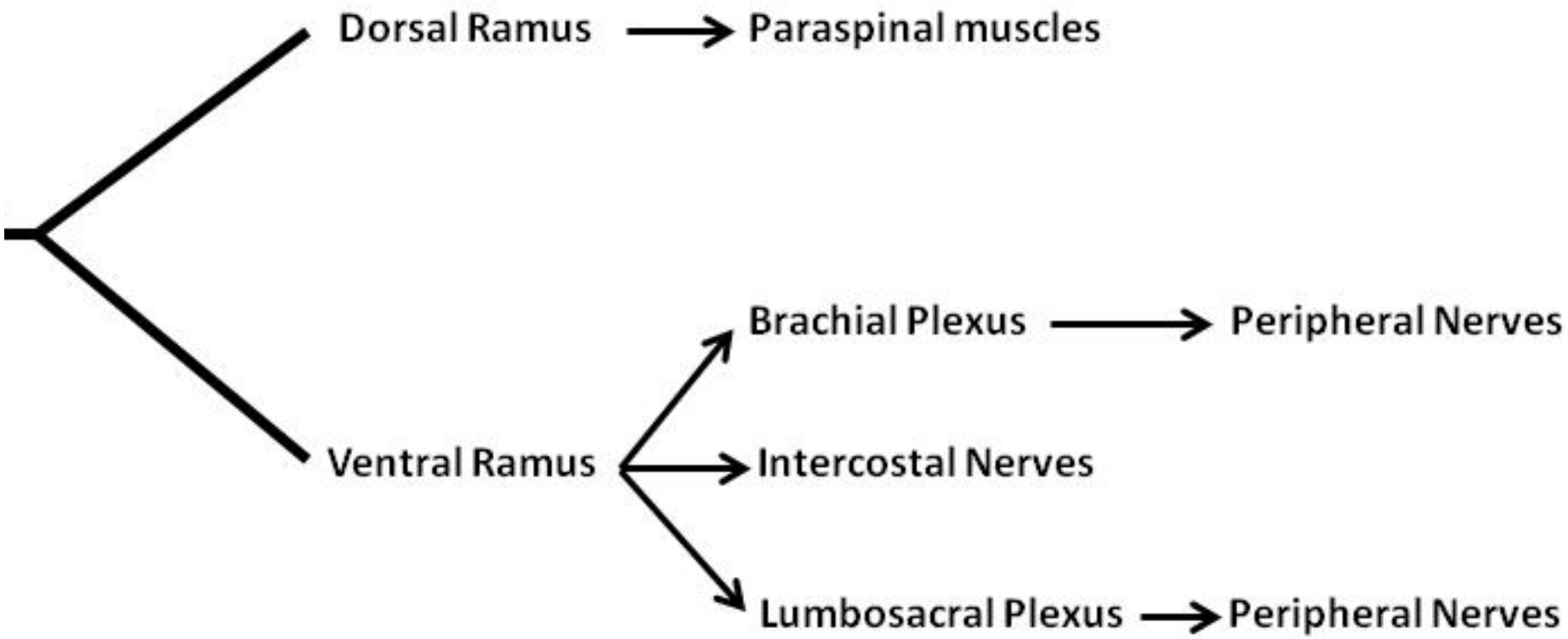
- Knowledge of nerve and muscle physiology important to determine type and timing of lesion

Peripheral Nervous System



31 pairs:
8 cervical
12 thoracic
5 lumbar
5 sacral
1 coccygeal

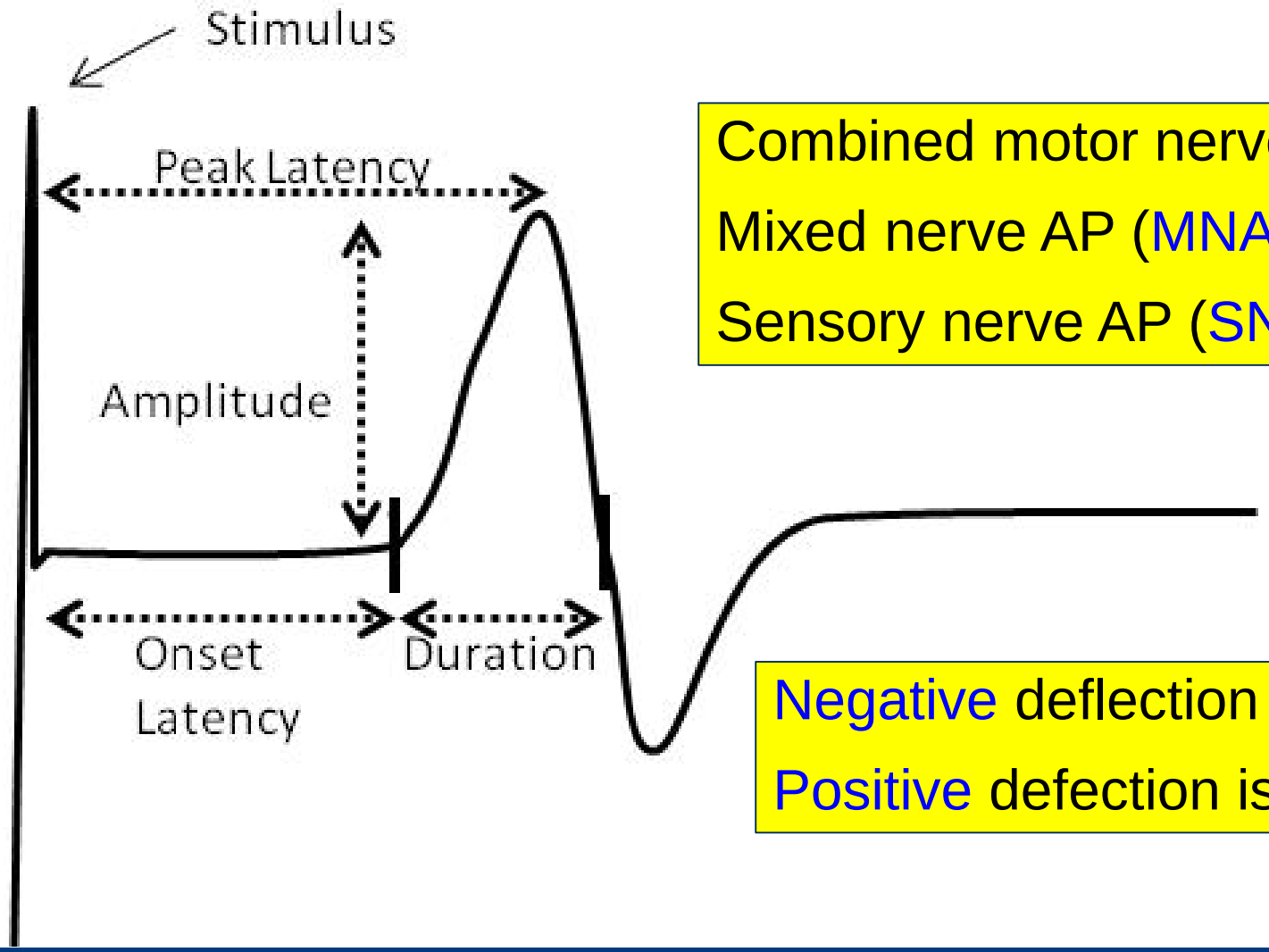
Peripheral Nervous System



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)

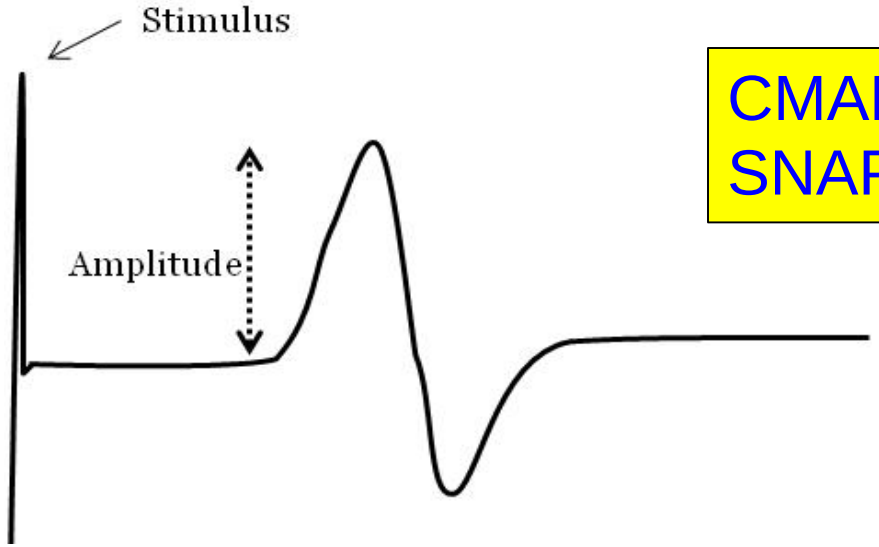
Mixed nerve AP (MNAP)

Sensory nerve AP (SNAP)

Negative deflection is upwards

Positive defection is downwards

Combined AP - Amplitude

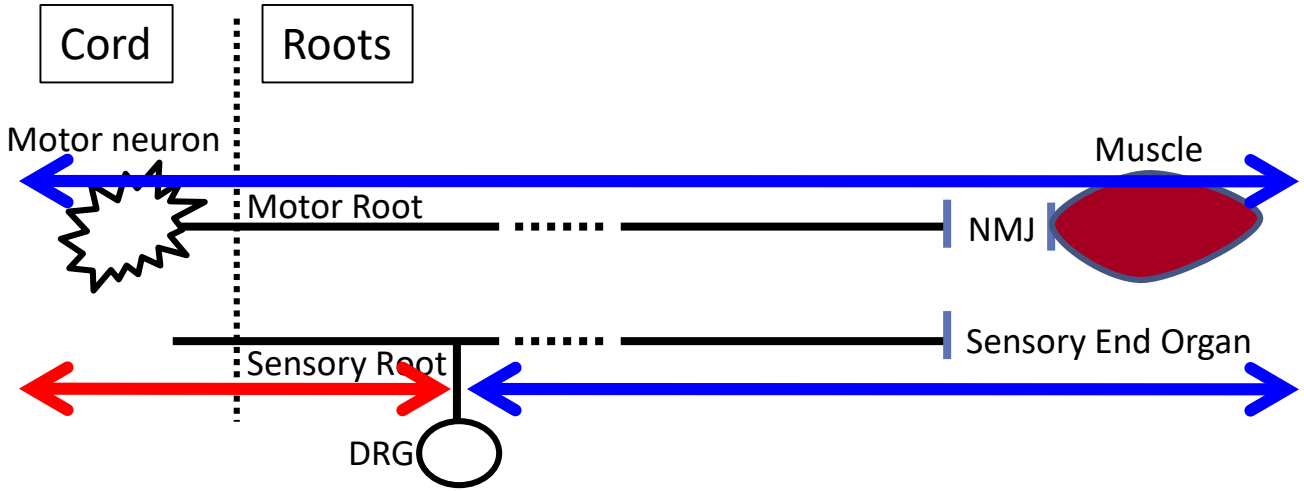


CMAP - in mV
SNAP & MNAP - in μV

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

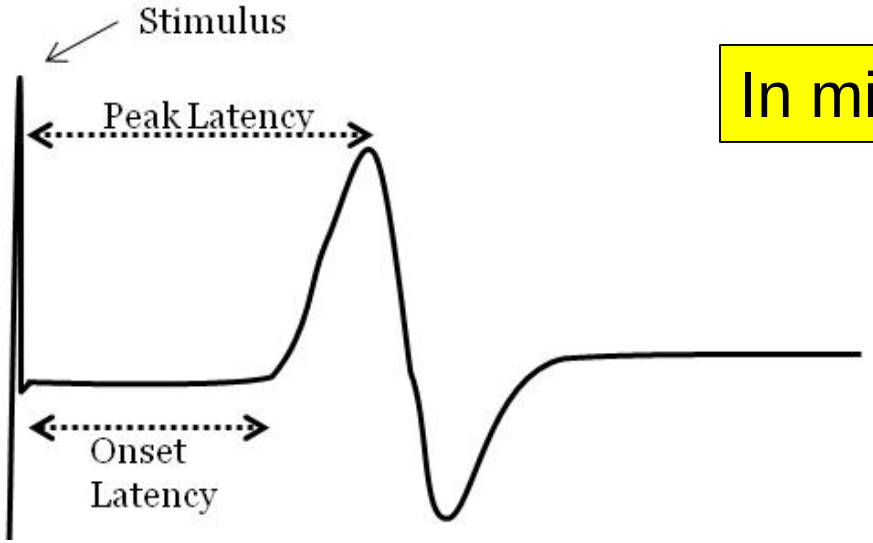
Combined AP - Amplitude

- Amplitude reduction
  - o SNAP & MNAP: loss of nerve fibers
  - o 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



In milliseconds (ms)

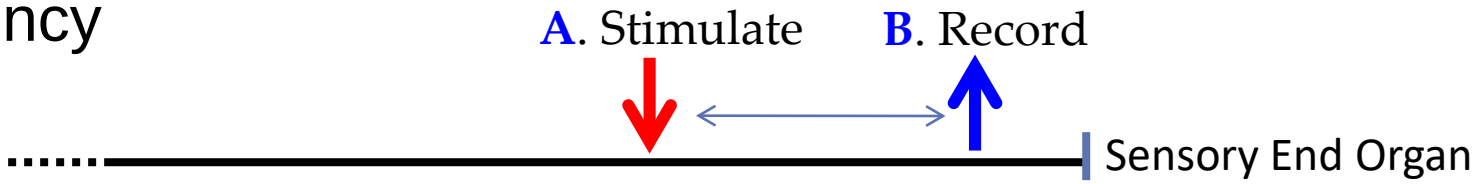
- Latency (time)
  - o Peak: Stimulus to first negative peak
  - o Onset: Stimulus to initial negative deflection
    - Reflective of fastest conducting fibers
    - Used for calculation of conduction velocity

Conduction velocity (m/s) = Distance/Latency

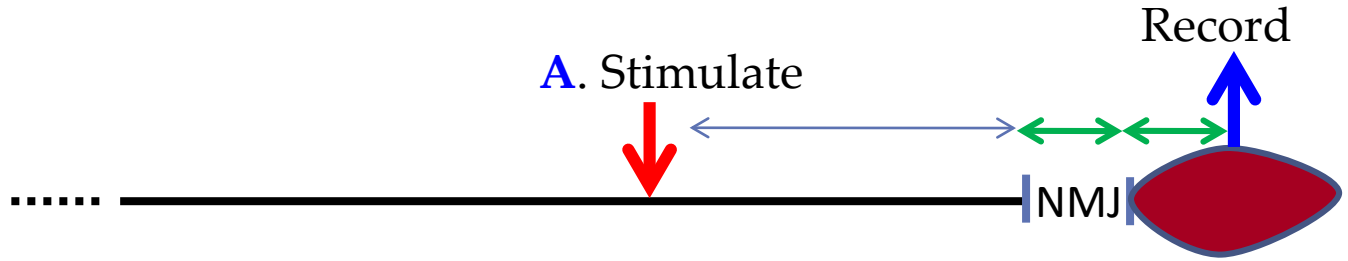
CV - CMAP vs SNAP

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

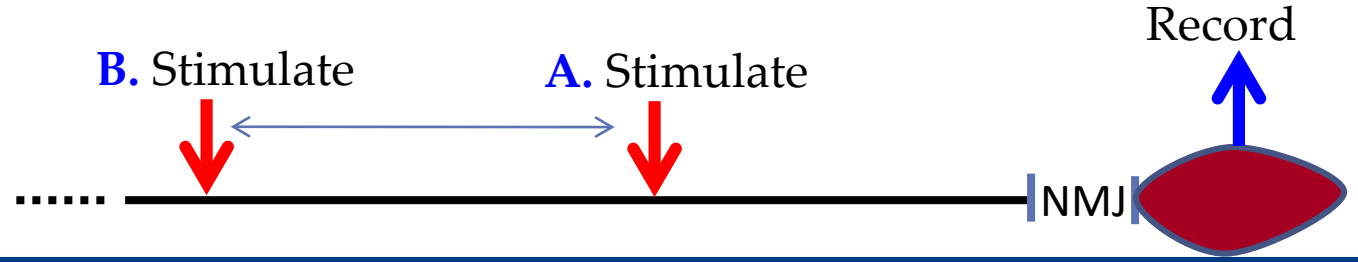
latency



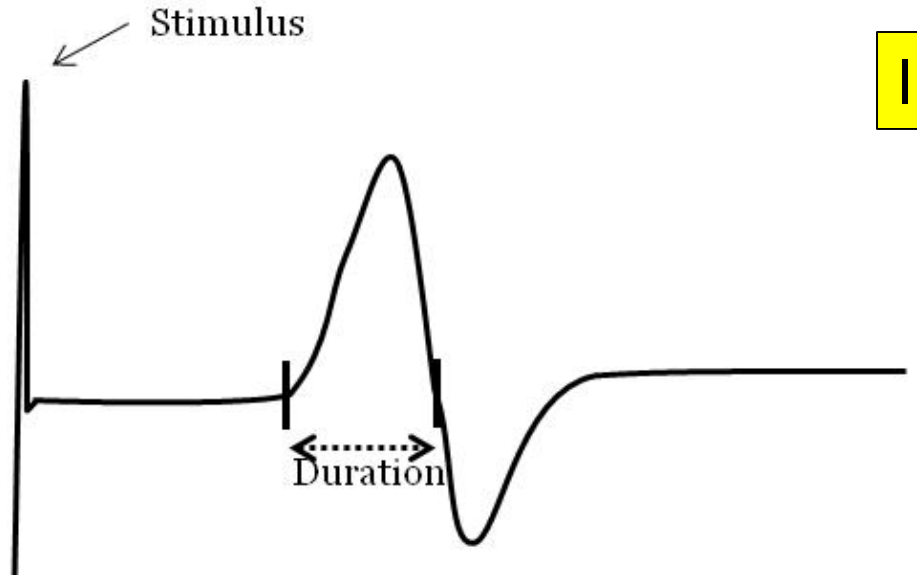
CMAP: Onset latency includes NMJ and muscle conduction



CMAP: CV=Distance A-B/latency A-B



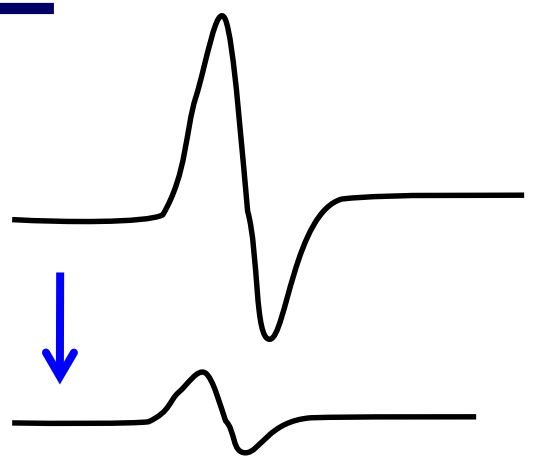
Combined AP - Duration



In milliseconds (ms)

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

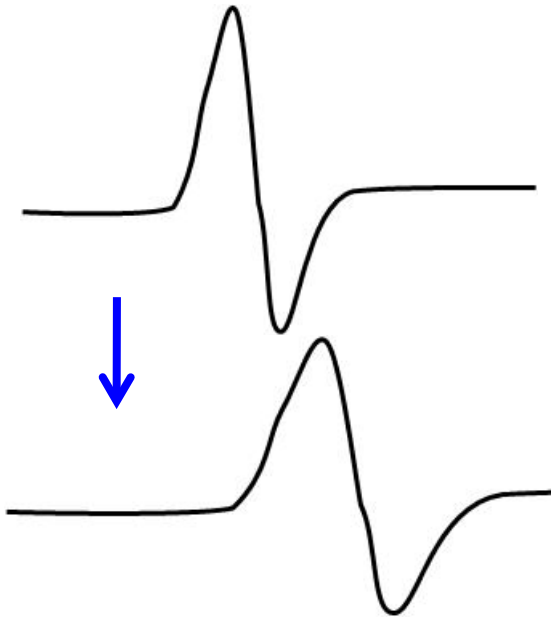
EDX Changes in NMD Disorders

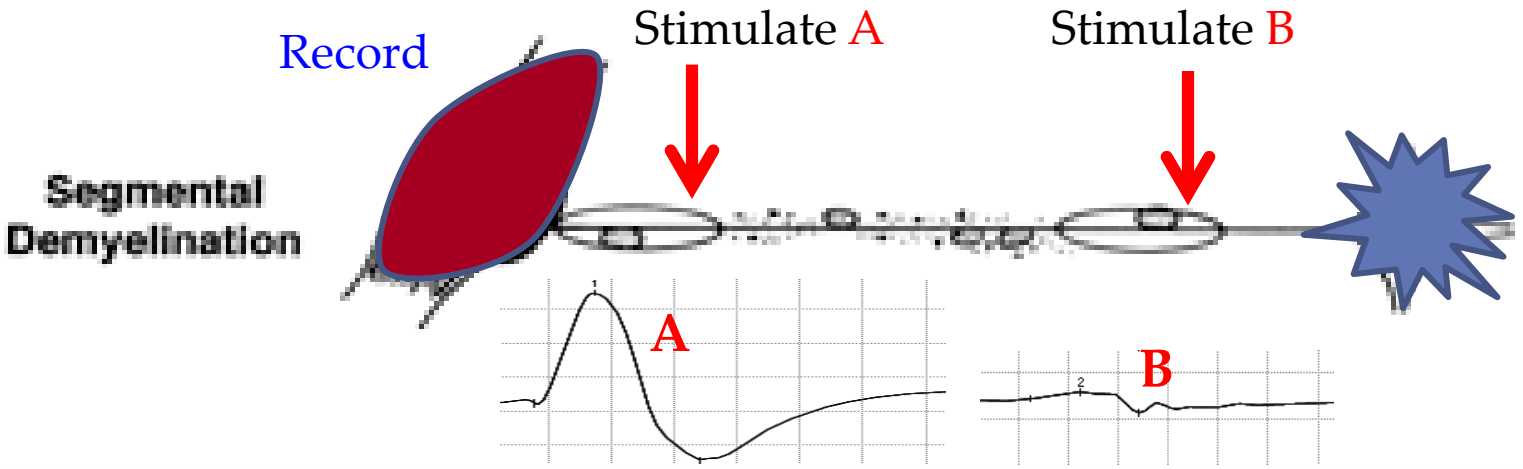


- Axonal loss (NCS)
  - o Reduction in amplitude
    - CMAP 3–5 days
    - SNAP 6-10 days
  - o Preserved latency and CV
    - Mild reduction with loss of fastest conducting fibers (not within demyelinating range)
- Axonal loss (EMG)
  - o Acute: reduced recruitment pattern
  - o Subacute: appearance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

EDX Changes in NMD Disorders

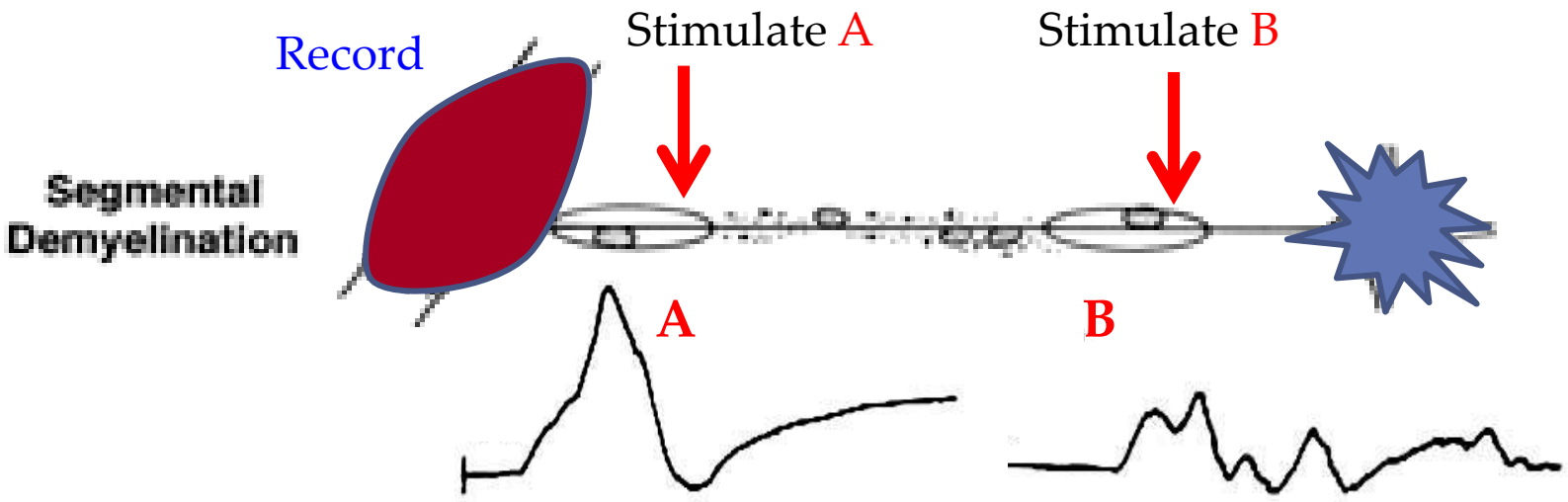
- Demyelination (NCS)
  - o Reduced CV <75% LLN
    - ~ 35 m/s UE and 30 m/s LE
  - o Prolonged latency >130% ULN
  - o Preserved amplitude except for
    - Conduction block





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
  - **Roots (dorsal)**
  - Dorsal sensory ganglion
  - **Nerve (sensory fiber)**
- Motor
  - Motor neuron (anterior horn)
  - **Roots (ventral)**
  - **Nerve (motor fiber)**
  - Neuromuscular junction
  - Muscle

NMD Presentation Sensorimotor

- Radiculopathy
  - o Disc
  - o Clinical
    - Radicular pain
    - Weakness in myotome, sensory change in dermatome
- Polyradiculopathy
  - o Diffuse meningeal process
  - o 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)

NCS/EMG
Similar to MND except for
- Demyelination in MMN
- Denervation in distribution of nerves or distal>proximal rather than myotomal

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
  - o 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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