Somatosensory Evoked Potentials

- Used to test the proximal axons of bipolar sensory ganglion cells and integrity of dorsal column & medial lemniscal pathways, to identify plexopathies, radiculopathies and nerve root avulsions.

- A mixed peripheral nerve is stimulated (e.g. radial, medial, common peroneal, or posterior tibial nerves). Evoked potentials generated in the CNS are detected at specific sites:
  - UL stimulation → check Erb’s point in supraclavicular fossa, or over C2/C7
  - LL stimulation → check over L1, C2 or C7 ; or over vertex
Electromyography

- Study of both voluntary and spontaneous electrical activity of muscle to identify neurogenic and myopathic conditions
- Motor unit = single motor nerve + neuromuscular end plate + muscle fibres it innervates
- Grounding is required to remove background noise and artefact. Surface electrodes used to stimulate motor nerve, and recording needles to assess muscle fibre response
  - Monopolar needles are narrower and better tolerated, but require surface reference electrodes which are prone to noise. Teflon coated apart from distal 0.5 mm of tip
  - Concentric needles are wider with insulated wires down the centre – active and reference needles can be close together allowing better noise reduction
- Motor unit potential (MUP) morphology:
  - Amplitude → muscle fibre density per axon at needle tip
  - Duration → indicator of motor unit territory
  - Phases → normally triphasic, but 20% of normal muscle have > 5 phases
  - Interference pattern occurs as a synchronous indistinguishable increase in amplitude of MUP as more separate motor units are recruited and existing active motor units fire more frequently to generate a greater contractile force.

- Recording parameters:
  - Insertional activity – brief burst of electrical activity representing an injury potential
    - Normal duration of 300-500 msec
    - Reduced after prolonged denervation, when muscle fibrosis occurs
    - Increased activity: nerve entrapment, anterior horn cell disease, axonal neuropathy (+ early sign of denervation)
  - Spontaneous activity:
    - Fibrillation potentials – short duration and low amplitude, seen early in partial/complete denervation at 21 days.
    - Positive sharp waves – [as above]
    - Fasciculations – spontaneous fibre contraction from loss of descending inhibition in LMN pathologies, anterior horn cell disease, neuropathies and MND
    - Complex repetitive discharges – earlier sign in denervation at 12 days (muscular dystrophy, MND, myositis, polyneuropathies)
    - Myokymic discharges – occur in chronic peripheral nerve disorders
  - Other pathological patterns:
    - Low amplitude, high frequency action potentials – reflect increased stimulation of poorly responding muscle group (e.g. myopathies)
    - Large amplitude, low frequency action potentials – reflect increased response of voluntary muscle to a decreased nerve stimulus (e.g. nerve compression syndromes)
    - MUP polyphasia – results from loss of synchronicity of nerve fibre conduction and muscle fibre discharge (e.g. radiculopathy, nerve entrapment, polyneuropathy)
    - Diminished interference and recruitment pattern – seen in neurogenic disorders.
Factors affecting NCV measurement:
- Specific nerve
- Patient age
- Temperature – NCV slows by 2 ms⁻¹ for every degree of cooling

Late Response

- Whereas standard NCS assess the distal third of a peripheral nerve, late responses assess the proximal portion of a peripheral nerve along with the nerve roots.
- H-reflex is the motor response produced by sub-maximal stimulation of 1a-afferent fibres from stretch receptors within muscle, that are involved in a monosynaptic reflex arc in the dorsal horn of the spinal cord, where they synapse onto alpha motor neurons
  - Elicited from soleus muscle on stimulation of the posterior tibial nerve in the popliteal fossa
  - Or from the FCR muscle
  - Delayed or absent in polyneuropathies or radiculopathy
- F-wave represents the small muscle response from retrograde conduction along motor nerves from a point of supra-maximal stimulation to the anterior horn cells, with secondary recurrent transmission back to the muscle
  - Normally an F-wave is 5% of the motor action potential.
  - Therefore prolongation of the F-wave latency with normal peripheral NCS implies a lesion causing slowing over the proximal motor fibres at the plexus or root level

Nerve Injuries

- Neuropraxia – a local myelin injury that manifests as a conduction block, with likely full recovery over a period of days to weeks
- Axonotmesis – axonal disruption with an intact endoneurium
  - Wallerian degeneration distal to point of injury
  - Antegrade re-inervation occurs slowly (1 mm/day or 1 inch/month) along the intact endoneural pathways.
- Neurotmesis – total disruption of the axons and endoneurium
  - Wallerian degeneration distal to injury, with no recovery
  - Requires anastomosis or autogenous nerve grafting to bypass disrupted segment
- In all three injury types there is a conduction block when measured proximal to the point of injury.
  - But distal conduction is possible in the acute stage in axonotmesis and neurotmesis, which is blocked once Wallerian degeneration has occurred
  - Distal conduction remains throughout in cases of neuropraxia
  - Axonotmesis distinguished from neurotmesis by return of partial motor or sensory function
  - (Complete absence of somatosensory evoked potentials usually indicates neurotmesis).
Neurophysiology for the Orthopaedic Surgeon
Taken from Woodruff M (2000), Current Orthopaedics, 14; 347-355

Nerve conduction studies

- A measure of distal peripheral nerve conduction, assessing:
  - Distal latency – time from stimulation to onset of action potential
  - Amplitude of evoked response
  - Conduction velocity

- Measured by stimulating a motor action potential (MAP) at 2 sites, with a supra-maximal stimulus. The calculation involves the latency as the time for the impulse to travel along the nerve and cross the neuromuscular junction (slower chemical transmission), where it can be detected in the muscle
  - The distance between the 2 sites measured divided by the difference in latencies gives the nerve conduction velocity
  - \[ nc = \frac{d}{(pl-dl)} \]

- The amplitude of the MAP (millivolts) is a measure of the active motor units within that muscle

- Sensory nerve action potentials can be measured using orthodromic pure sensory nerves (SNAP), or antidromic mixed nerves (compound, CNAP)
  - CNAPs allow larger amplitudes which are more reliably recorded
  - But, produce movement artefact and can be uncomfortable.
  - Sensory NCV is simply the distance between the stimulus and detection electrodes, divided by the latency between them.
  - SNAP amplitude is measured in microvolts (much smaller than motor APs) – often require needle electrodes positioned close to the nerve instead of surface electrodes.

- Normal values

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<thead>
<tr>
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<th>Arm</th>
<th>Leg</th>
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<tbody>
<tr>
<td>Motor NCV</td>
<td>$&lt;45 \text{ ms}^{-1}$</td>
<td>$&lt;40 \text{ ms}^{-1}$</td>
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<tr>
<td>Sensory NCV</td>
<td>$&lt;45 \text{ ms}^{-1}$</td>
<td>$&lt;35 \text{ ms}^{-1}$</td>
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- Sensory NCV is a more sensitive measure of a peripheral nerve lesion

- A 50% reduction in amplitude when comparing sides is considered significant – usually representing axonal loss. A 75% axonal loss is needed before NCV is affected

- Temporal dispersion occurs when there is reduction in conduction velocity by myelin pathology, where there is loss in fibre synchronicity leading to reduced amplitude with increased duration ($>4 \text{ ms}$).
  - The area under the curve remains the same, showing the pathology is in conduction and not axonal loss.
  - Conduction velocity drop $<60\%$ and terminal latency rise $>150\%$; (vice-versa in axonal loss)