Frequency of Peripheral Nervous System Trauma

- Of patients admitted to Level 1 trauma centers:
  - 2-3% have peripheral nerve injuries
  - 2-3% have brachial plexus injuries
- Of those with PNS injuries, 60% have TBI
- Of those with traumatic brain injury:
  - 10-30% have PNS injuries

Classification of Nerve Injuries (Seddon)

- Neurapraxia
- Axonotmesis
  - Sunderland subdivides axonotmesis into 3 anatomically based categories depending on the degree of intraneural disorganization
- Neurotmesis

Nerves Most Often Affected

- Upper limb > Lower limb
- Upper Limb:
  - Radial
  - Ulnar
  - Median
- Lower Limb:
  - Sciatic
  - Peroneal
Neurapraxia

- Comparatively mild injury
- Motor and sensory loss
- No axonal (Wallerian) degeneration
- Nerve conducts normally distally
- Focal demyelination and/or ischemia
- Recovery within hours to a few months

Axonotmesis

- Commonly seen in crush injuries
- Axon and myelin sheaths are broken
- Surrounding stroma partially intact
- Wallerian degeneration occurs
- Recovery depends upon axonal regrowth, internal disorganization, and distance to muscle

Neurotmesis

- Nerve is completely severed, or so scarred (endoneurium and perineurium) that regrowth does not occur
- Sharp injury, traction and intraneural injection
- Prognosis is very poor without surgery

Classifying the Nerve Injury

- Neuropraxia
  - Distal CMAP and CNAP maintained
- Axonotmesis/Neurotmesis
  - CMAP and CNAP drop in rough proportion to degree of axon loss
  - Drop is complete by day 9 for CMAP and day 11 for SNAP
**Mixed Lesions**  
(axon loss and conduction block)

- Percentage of axon loss best estimated by distal CMAP  
- Percentage of conduction block by examining loss of amplitude from stimulation above and below the lesion

**Needle EMG in Neurapraxia**

- Reduced recruitment  
  - (Increased recruitment ratio >7)

**Needle EMG in Axonotmesis/Neurotmesis**

- Length-dependent onset of fibrillations and positive sharp waves  
  - Proximal muscles 10-14 days  
  - Distal muscles 3-4 weeks  
  - Fibrillation amplitudes decrease over time  
- Sensitive indicator of axon loss, but does not quantify  
- Beware of mixed lesions  
- Beware of muscle trauma

**Timing of the Electrodiagnostic Study Depends on the Question**

- 7-10 days for localization and sorting neurapraxia from axonotmesis  
- 3-4 weeks for most diagnostic information  
- 3-4 months for detecting reinnervation
Localization of Nerve Injuries

- Focal slowing of conduction
  - Only with demyelination or conduction block
  - Not seen in pure axonal lesions
- SNAP amplitude
  - Helps with pre- vs post-ganglionic lesions
  - Normal SNAP in presence of complete denervation usually indicate root avulsion
  - Reduced amplitude indicates some post-ganglionic axon loss

Localization using SNAP’s

- Upper trunk
  - Median to thumb
  - Lat. Antebrachial Cutaneous
- Middle trunk
  - Median to long finger
- Lower trunk
  - Ulnar to small finger
  - Dorsal Ulnar Cutaneous

Localization of Root vs Plexus using paraspinal EMG

- Denervation suggests pre-ganglionic lesion
- Cannot differentiate between complete and incomplete lesions due to segmental overlap

Estimation of Prognosis using the CMAP Amplitude

- Much data comes from the study of facial nerve lesions
- Comparing CMAP on involved to uninvolved side:
  - > 30% - excellent outcome
  - 10-30% - good but incomplete recovery
  - <10% - poor recovery (insufficient overlap of intact axons for optimal terminal reorganization)
Immediate Surgical Reconstruction

- Sharp lacerations
- Complete nerve section
- Nerve ends are intact
- Minimal local tissue trauma

Delayed Surgical Reconstruction

- Nerve continuity unclear
- Natural recovery could be better than surgery
- Wait to see if there is clinical or EMG evidence of reinnervation
- Operate on those without ongoing recovery
- Usually intervene by 6 months to prevent end organ deterioration

Top 10 Entrapments

1. CTS
2. CTS
3. CTS
4. Ulnar Elbow
5. Radial Spiral Groove
6. Anterior interosseous
7. Ulnar Wrist
8. Radial Wrist
9. Fibular Knee
10. Tarsal Tunnel Syndrome
The best electrodiagnostic testing for CTS should be:

- In descending order of priority:
- Specific (few false positives)
- Sensitive (few false negatives)
- Reliable (get the same results on repeat testing)
- Resistant to Temperature Effects
- Efficient

Median motor conduction in CTS

- Prolonged distal latency
  - less sensitive than sensory prolongation
- Motor amplitude
  - low amplitude may indicate axon loss or conduction block
  - palm stimulation unreliable
- Martin-Gruber anastamosis

Repeated Measures in CTS

There are 3 good sensory tests for CTS
The problem is how to interpret them! More tests = more false positives

Combined Sensory Index (CSI)

Add 3 sensory latency differences

- Median – radial thumb difference
- Median – ulnar ring finger difference
- Median – ulnar mid-palmar difference

= CSI

Sensory Reference Values

<table>
<thead>
<tr>
<th>Reference Value</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palm: Med - Uln</td>
<td>≤ 0.3</td>
</tr>
<tr>
<td>Ring: Med - Uln</td>
<td>≤ 0.4</td>
</tr>
<tr>
<td>Thumb: Med - Rad</td>
<td>≤ 0.5</td>
</tr>
<tr>
<td>CSI</td>
<td>≤ 0.9</td>
</tr>
</tbody>
</table>
Combined Sensory Index (CSI)

- Improved Sensitivity in mild CTS
- High Specificity
- Improved test-retest reliability

Sensitivity and Specificity of CSI

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>MidPalm</td>
<td>≤ 0.3</td>
<td>70%</td>
<td>97%</td>
</tr>
<tr>
<td>Ring</td>
<td>≤ 0.4</td>
<td>74%</td>
<td>97%</td>
</tr>
<tr>
<td>Thumb</td>
<td>≤ 0.5</td>
<td>76%</td>
<td>97%</td>
</tr>
<tr>
<td>CSI</td>
<td>≤ 0.9</td>
<td>83%</td>
<td>95%</td>
</tr>
</tbody>
</table>

Results: Palmdiff

Results: CSI
Reliability

<table>
<thead>
<tr>
<th>Test</th>
<th>Spearman Rho</th>
</tr>
</thead>
<tbody>
<tr>
<td>MidPalmar</td>
<td>.74</td>
</tr>
<tr>
<td>Ring</td>
<td>.67</td>
</tr>
<tr>
<td>Thumb</td>
<td>.75</td>
</tr>
<tr>
<td>CSI</td>
<td>.95</td>
</tr>
</tbody>
</table>

Effect of temperature on latency

- 8 cm Median: 0.06 msec/degree Celsius
- 10 cm Median: 0.11 msec/degree Celsius
- 14 cm Median: 0.14 msec/degree Celsius
- All Latency Diff: not affected by temperature

Single Test vs. CSI
With very small or very large differences
CSI may not be necessary
Perform Needle EMG?

- Debated
- My practice:
  - Signs or symptoms of cervical radiculopathy
  - History of Trauma
  - Abnormal Median Motor Response

Ulnar Neuropathy at the Elbow

Motor Conduction Studies

- Elbow should be flexed
- Stimulate at wrist, below elbow, above elbow, and axilla
- At least 10 cm across elbow is advocated
- NCV < 48 m/s is likely slowing
- Less useful to compare with forearm NCV
- Beware of Martin Gruber!

Ulnar Neuropathy at the Elbow

FDI recording

- Active over bulk of the muscle
- Reference proximally over the dorsal CMC joint of the thumb
- (Don’t place over index finger MCP.)
- 2 Channel recording
How much slowing is abnormal?

- **Segmental Difference**
  - Difference of 11 - 15 m/s often used
  - Assumes normal forearm conduction
  - But the forearm doesn’t stay normal, with axon loss!

- **Absolute CV**
  - < 48 m/s used
  - Does not depend on assuming forearm conduction is normal
  - Better sensitivity!

Comparison of Methods

(Specificty = 95% for all, Shakir & Robinson 2004)

<table>
<thead>
<tr>
<th>Method</th>
<th>Reference</th>
<th>Value m/s</th>
<th>Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADM CV</td>
<td></td>
<td>48</td>
<td>80%</td>
</tr>
<tr>
<td>FDI CV</td>
<td></td>
<td>49</td>
<td>77%</td>
</tr>
<tr>
<td>ADM Difference</td>
<td></td>
<td>10</td>
<td>51%</td>
</tr>
<tr>
<td>FDI Difference</td>
<td></td>
<td>15</td>
<td>38%</td>
</tr>
</tbody>
</table>
Ulnar Neuropathy at the Elbow

Motor Conduction Studies

- Inching studies may pick up more mild cases
- Stimulate at 2 cm increments along nerve
- > 0.7 msec latency change over 2 cm is suspicious
- More convincing if accompanied by sudden change in amplitude or shape

“Inching” Technique

- Avoid excessive stimulation intensity
- Move stimulator following submaximal stimulation to localize the nerve path prior to supramaximal stimulation

Ulnar Neuropathy at the Elbow

Sensory Conduction Studies

- Distal sensory amplitude is reduced early.
  - Reflects loss of sensory axons
  - Compare with the opposite side
- Don’t over-interpret drop in sensory amplitude from proximal stimulation
  - Expect 50% drop from wrist to elbow
  - Temporal dispersion and phase cancellation
**Ulnar Neuropathy at the Elbow**

**Needle EMG**

- May show abnormalities in Ulnar distribution
  - even in a few cases with normal NCS
- FCU and FDP are often not involved
  - rarely, branches come off proximal
  - intraneural topography spares these branches

**Ulnar Neuropathy at the Wrist**

- Ganglions
- Rheumatoid Arthritis
- Lacerations
- Fractures
- Occupational
  - Pipe cutters
  - Metal polishers
  - Mechanics

**Ulnar Neuropathy at the Wrist**

- Mixed Motor and Sensory (30%)
- Pure Motor (52%)
- Pure Sensory (18%)
- Dorsal Ulnar Cutaneous Branch

**Anterior Interosseus Lesions**

- Supplies FPL, FDP (digits 2 & 3), PQ;
  No sensory fibers
- Clinical Presentation:
  forearm pain, weakness of FPL FDP
difficult to isolate PO
- EMG of affected muscles is abnormal
  - beware of Martin Gruber (50% from AIN)
  - beware of FDS supply from AIN (30%)
- NCS - conventional studies normal
- **Etiology**
  - anomalous muscles
  - neuralgic amyotrophy
  - partial higher median neuropathy
High Radial Nerve Lesions

Etiology
– almost always traumatic
– crutch palsy (axilla, triceps often weak)
– Saturday night or honeymoon palsy
  (triceps usually spared)

EMG and NCV
– EMG of triceps, brachioradialis, forearm extensors most useful.
– Radial SNAP reduced in amplitude
– Motor studies may show focal slowing or conduction block, but these are not optimal.

Electrodiagnostic Exam
• Needle EMG is most useful
  – Work down radial nerve
    • Triceps
    • (Anconeous same as branch to medial triceps)
    • Brachioradialis (important for prognosis)
    • ECR
    • EDC
    • ECU
    • EIP
  – Non radial muscles by same roots

Superficial Radial Nerve Lesions
• Reduced sensation in radial distribution
  – pain is often most disabling
• Etiologies include:
  – wristwatch, handcuffs, casts
  – laceration during IV or deQuervain’s surgery
• Only electrodiagnostic finding is abnormal SNAP.
Fibular Neuropathy

- There is no more Peroneal Nerve
  - Anatomists killed it
  - Worried about confusion with perineum
  - If you leg and perineum confused, you have big problems

- Important to record from Tibialis Anterior
  - EDB has no useful function
  - EDB is often absent in Fibular Neuropathy
  - Tib Anterior more helpful for prognosis

Fibular Motor NCS

- Record EDB and Tibialis Anterior
  - provides confirmatory results
  - helpful when EDB response is absent

- Stimulate
  - ankle, below fibular head, lateral popliteal fossa
  - beware of volume conduction from tibial nerve

- Inching across fibular head often helpful

Recording Site for Tibialis Anterior

- Active over motor point
- Reference distal over tendon
  - Not over muscle
- Better for prognosis and localization
- Can do 2 channels

Beware of Accessory Fibular Nerve

- Originates from Superficial Fibular Nerve
- Supplies Lateral Head of EDB
- Fibular Motor NCS to EDB
  - small CMAP at ankle
  - larger CMAP at fib head
  - CMAP present behind lateral malleolus
Fibular Nerve EMG

- Deep Fibular Distribution
  - Tibialis Anterior, EHL
- Superficial Fibular Distribution
  - Peroneus Longus
- Tibial Distribution
  - Gastroc, Soleus (to rule out higher lesion)
- Short Head of Biceps Femoris
  - to rule out higher lesion

Nerve Conductions for TTS
Are there more techniques than patients?

- Motor latencies to AH and ADQP
- Sensory latencies from 1st and 5th toes
  - surface recording
  - near nerve recording
- Mixed nerve latencies with plantar stim
  - recording over medial malleolus
- Variations of the above

Nerve Conduction in TTS
Galardi et al 1994 (14 Pts, 12 Nmls)

<table>
<thead>
<tr>
<th>Technique</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMAP latency</td>
<td>22%</td>
<td>100%</td>
</tr>
<tr>
<td>SNAP Med Plantar</td>
<td>93%</td>
<td>92%</td>
</tr>
<tr>
<td>SNAP Lat Plantar</td>
<td>100%</td>
<td>83%</td>
</tr>
<tr>
<td>CNAP Med Plantar</td>
<td>64%</td>
<td>100%</td>
</tr>
<tr>
<td>CNAP Lat Plantar</td>
<td>71%</td>
<td>100%</td>
</tr>
</tbody>
</table>

“Causes” of Tarsal Tunnel Syndrome

- Post-traumatic fibrosis
- Ganglion
- Tumor
- Fracture
- Thrombophlebitis / varicosities
- Anomalous muscle - FDAL
- Rheumatoid arthritis
- Joint hypermobility / hyperpronation
MRI in Tarsal Tunnel Syndrome
(33 cases Kerr et al 1991)

- Varicosities 8
- FHL Tenosynovitis 6
- Normal 6
- Fracture / soft tissue injury 5
- Mass lesions 5
- Fibrous scar 2
- Abductor Hallucis Hypertrophy 1
- 17 / 19 confirmed surgically

Other Foot Neuropathies

- Medial plantar neuropathy behind the navicular tuberosity (jogger’s foot)
  - Needle exam of the flexor hallucis brevis
- Lateral plantar neuropathy near the insertion of the plantar fascia (Baxter’s neuropathy)
  - Needle exam of the ADQ
  - Motor latency to the ADQ

Thanks for being entrapped in this lecture for so long!