Upper Extremity Nerves

Learning Objectives

At the completion of the course, the learner will be able to
1. describe the neuroanatomy from brain to fingertip.
2. describe current concepts of peripheral nerve biology and healing.
3. collect a good history & exam for common peripheral nerve conditions.
4. educate patients regarding their condition and treatments.
5. participate more effectively in the patients’ care

METHODS

• 12 lectures
• 2 question and answer sessions
• networking

• some repetition between talks
  bad news: unavoidable because of multiple speakers
  good news: repetition is a good learning tool

Course Content Meets These
AOTA Classification Codes for CE activities

Category 1: Domain of OT
Areas of occupation: ADLs
Performance skills: motor skills
Activity demands: required actions, body functions and structures

Category 2: OT Process
Develop intervention plan/approaches
Implement intervention

Category 3: Professional Issues
OT education: teaching theory and methods
Supervision: competence
Contemporary issues/trends: professional
development/continuing competence

For Your Questions during the Discussion Sessions:
Write them on notecards as you think of them

Practical Matters
Can everybody see? Hear?
Room temperature?
Cell phones, beepers
Auditorium’s policy on food/beverage
Restrooms
Evaluation forms and certificates
Ready, set, go……

MACRO ANATOMY
From Brain to Fingertips

Naveed Nosrati, MD
Outline

- Motor pathways (cortex to finger tip)
- Sensory pathways (finger tip to cortex)
- 6 major nerves
- Anatomic anomalies
- Sympathetic nervous system

Motor Pathways: Major Elements

- Central Nervous System
  - cerebral cortex
  - brain stem
  - spinal cord
- Peripheral Nervous System
  - root
  - plexus
  - branch/ periph n.
  - end organ

Motor Strip  Sensory Strip

- Motor Impulses
  - originate in parietal cortex gray matter
  - originate in the “motor strip”

Motor Impulses

- travel through the internal capsule (white matter)
- cross in the brainstem — each side of the cortex controls the opposite side of the body

spinal cord (CNS) gives off nerve roots (PNS)

Anterior:
  - Motor Roots
    - Cell bodies in anterior horn of spinal cord

Dorsal:
  - Sensory Roots
    - Cell bodies in dorsal root ganglia

brachial plexus is formed from:
- inferior 4 cervical nerve roots (C5-C8)
- superior thoracic nerve root (T1)
  - posterior
  - anterior
The Brachial Plexus Has 5 Anatomical Parts

- **Roots**
- **Trunks**
- **Divisions**
- **Cords**
- **Branches**

Randy Travis Drinks Cold Beer

Bony Landmarks and the Parts of the Brachial Plexus

- **Roots**
- **Trunks**
- **Divisions**
- **Cords**
- **Branches**

Brachial Plexus
And
Muscles/Bones

- Emerges between anterior and medial scalene muscles
- Passes beneath clavicle
- Passes deep to pectoralis major and minor

Brachial Plexus and Axillary Artery

- Medial, lateral, and posterior cords
- Closely associated with AA
- Named for their relationships to the AA

The brachial plexus is complicated...

Its major components can be learned and drawn as easy as 1,2,3 (x8)...
It is important to know the nerves, their routes through the brachial plexus, AND from which spinal roots they arise...

How to begin making sense out of which nerve roots supply which muscles?

1. Know the sensory dermatomes
   - Start with C6
     - Bang!

   It works for biceps, brachialis and sort of for triceps

2. Make a “7” with your fingers, wrist and elbow
   - C7: elbow extension
   - wrist flexion
   - finger extension
16 nerves exit the brachial plexus
We will look at 6 major ones
(more detail to follow later today)

1) Long Thoracic
2) Axillary
3) Musculocutaneous
4) Radial
5) Median
6) Ulnar

Quiz follows!

2) Axillary Nerve
• exits brachial plexus posteriorly
• courses through “quadrilateral space”
• innervates DELTOID

3) Musculocutaneous Nerve
• Innervates the “BBC muscles”
  • BICEPS
  • BRACIHALIS
  • CORACOBRACHIALIS
• Continues as the lateral antebrachial cutaneous nerve

1) Long Thoracic Nerve
• courses inferiorly on anterior lateral chest wall
• innervates SERRATUS ANTERIOR
• prevents scapular winging
4) Radial Nerve
- Leaves brachial plexus posteriorly
- Passes laterally behind the humerus
- Crosses anterior to lateral epicondyle
- Pierces supinator
- Passes along dorsolateral forearm
- Innervates triceps, wrist and finger extensors

5) Median Nerve
- Arm: medial, with brachial artery
- Elbow: central, medial to biceps tendon
- Forearm: central, beneath fds
  innervates fds, ½ fdp, fcr, pronator teres
- Palm: central, through carpal tunnel
  innervates thenar muscles, 2 lumbricals

6) Ulnar Nerve
- Arm: medial; posterior to median nerve and brachial artery
- Elbow: posterior to medial epicondyle
- Forearm: medial, beneath fcu
  innervates fcu and ½ fdp
- Palm: Guyon’s canal; innervates hypothenars, interossei, add pollicis, 2 lumbricals

QUIZ!
Name that palsy!
Motor Impulses Terminate at the Neuromuscular Junction

Nerve

Muscle

On to sensory nerves.....

Sensory Impulses

- Motor impulses move distally away from CNS
- Sensory impulses move proximally toward the CNS
  - share nerves with motor system, use different fibers
- Sensory nerves innervate skin in the area of their motor counterpart
  - (i.e. axillary nerve transmits sensory information from the skin of the shoulder)

Sensation in the Hand

Median: palmar aspect of lateral 3 ½ digits + nail beds of same

Ulnar: palmar/dorsal aspects of medial 1 ½ fingers + hand continuous with same

Radial: volar aspect of lateral 3 ½ digits and hand continuous with same (excluding nail beds).

Differences in Sensory Nerve Pathways

- S. nerves enter spinal cord thru dorsal roots
- S. nerves cross to opposite side in brain stem or spinal cord
- Pass through the thalamus
- Terminate in the sensory strip

The Sympathetic Nervous System in the Upper Extremity

2 parallel chains of ganglia along sides of spinal cord.

Stellate Ganglion

= Inferior Cervical + Superior Thoracic Ganglia
Sympathetic Fibers in the Upper Extremity

- pass through stellate ganglion
- travel along arteries
- control vessel constriction/relaxation; sweat; piloerection
- implicated in some chronic regional pain syndromes

References

Outline
- Microanatomy
- Biology
- Nerve Injuries
  - Physiology
  - Classification
  - Nerve response to injury

Anatomy of Neuron

- Cell body
- Dendrites
  - Receive incoming impulses
- Axon
  - Conducts outgoing impulses
- Schwann Cells
Anatomy of a Peripheral Nerve

1. Axon: basic subunit
   - Nerve fiber = axon + Schwann cell + myelin
2. Fascicle: groups of axons
3. Peripheral nerve: groups of fascicles

Nerve Fiber (Axon) Counts
- Electric lamp cord: ~60 copper strands
- Digital nerve: ~1500 nerve fibers
- Median nerve: ~25,000 nerve fibers
- Brachial plexus: ~145,000 nerve fibers

Connective tissue layers
1. Epineurium
2. Perineurium
3. Endoneurium
   - Vascular supply
     - Intrinsic + extrinsic

Cross-section of peripheral nerve
- Epineurium
  - Cushions nerve
  - Nerve gliding
- Perineurium
  - Surrounds fascicle
  - Tight junctions
- Fascicle
- Endoneurium
  - Surrounds axons

Axon + Endoneurium
  - External epineurium
  - Internal epineurium
  - Fascicle
Nerve Physiology
- Axonal transport
- Protein synthesis in cell body
- Distance too long for diffusion
- Products motored along microtubules
- Retrograde transport
- Returns waste products for recycling
- Transports growth factors to nucleus
- Implications with injury

Nerve Injury
- Mechanical injuries
  - Compression
  - Traction
  - Laceration
  - Combination (e.g. GSWs)
- Secondary injury
  - Infection
  - Scarring
  - Fracture callus
  - Ischemia

Response to Injury
- Limited repertoire of responses to nerve injury
1. Mild injury → schwann cells/myelin sheath
   - Focal demyelination
2. Severe injury → axons
   - Axonal degeneration

Nerve Injury Classification
1. Neurapraxia
2. Axonotmesis
3. Neurotmesis

Neurapraxia
- Conduction block
- All supporting layers entirely intact
- Mild neurapraxia → transient ischemia
  - Impairs polarization of cell membrane
  - Unable to conduct action potential

Neurapraxia
- More severe injury → Demyelination
- Disrupts impulse conduction
- Axons are physically intact, physiologically out
Neurapraxia
- Prognosis for recovery excellent
  - Axons remain intact → no axonal degeneration
  - Mild cases: relief of temporary ischemia
  - Severe cases: Schwann cells remyelinate damaged segment
  - Restores conduction along axon
  - Symptoms may last moments to months

Axonotmesis
- Loss of axon continuity
  - Connective tissue envelope intact
  - Degeneration of injured axons distally

Axonal Injury: Wallerian Degeneration
- Axonal transport interrupted
- Survival of axonal segments connected to cell body
- Distal segments
  - Deprived of metabolic support
  - Degeneration of axons beyond point of injury

Axonotmesis
- Recovery requires axonal regeneration
  - Prolonged recovery
  - Prognosis for recovery good
  - Endoneurial tubes are intact → no miswiring
  - Limiting factor is the distance of regeneration required

Neurotmesis
- Complete nerve transection
- Degeneration of all axons distal to injury
- Separation of nerve ends → spontaneous recovery will not occur
- Surgical problem

Neurotmesis- End Result
- Neuroma
**Physiology of Injury**

- Laceration → Neurotmesis
  - Smaller zone of injury
- Traction → Axonotmesis
  - Axons most susceptible
  - Larger zone of injury
- Acute compression → Neurapraxia
  - Local contusion with 2nd circulatory changes
  - Inflammation → scarring and further compression

**Chronic compression**

- Ischemia + mechanical compression
  - At 30 mm Hg
    - Decreased intra-neural blood flow
    - Axonal transport disrupted
    - Progressive axonal loss (axonotmesis) and fibrosis
    - Damage often permanent

**Nerve regeneration**

- Wallerian degeneration
  - Distal axon fragmentation
  - Disintegration of myelin
  - Macrophage invasion
    - Clears endoneurial tubes of debris
  - Schwann cell proliferation
  - Pathway for later axonal regeneration

**Axonal Regeneration**

- Proximal effects
  - Loss of trophic support
  - Sometimes neuronal death
  - Survivors → alter metabolic activity in preparation
- Axonal sprouting
  - Growth cones
    - Samples environment
    - Seeks sensory or motor pathways
  - Guided by Schwann cells

**Nerve Regeneration**

- Axons that find distal tubules are guided along (1mm/day)
- Sprouts that reach distal connections mature
  - Increase in axon and myelin thickness
- Sprouts which fail to make connections die back

- If gap exists, axonal sprouts migrate aimlessly
  - No distal targets are reached
  - Neuroma formation
Nerve Regeneration: Molecular Level

- **Neurotrophic factors** ("fertilizer")
- **Neurotropic factors** (directional signals)
- **Matrix substrates**
- **Metabolic factors for nourishment**

### Neurotrophic Factors

- **Promote survival and maintenance** of neuron
- **Axonal injury** → disruption of retrograde supply of growth factors
- **Schwann cells** provide trophic support
  - Proteins presented along regenerating pathway
  - Picked up by axonal sprouts to sustain neuron
- **Examples**
  - Nerve Growth Factor → sensory neurons
  - Brain-derived neurotrophic factor → motor

### Neurotropic factors

- **Promote extension and branching** of axonal sprouts
- **Attract** sprouting axons toward distal nerve segment
- **2 mechanisms**: contact-guidance versus attraction at a distance
  - Laminin and fibronectin: matrix-bound glycoproteins
  - Soluble mediators

### Topographic Specificity

- **Accuracy** with which regenerating axons re-innervate appropriate targets
- **Dramatically affects outcomes** after nerve injury
- Axonotmesis >> neurotmesis
- **Miswiring**: axons wander into wrong tubules

### Clinical Perspective

- Axons regrow about ~1mm/day (1" / month)
- Motor end plates degrade ~1%/week
- **Need > 50%** of motor endplates for function
- **Maximum distance** to restore motor function is ~35 cm
- Sensory end-organs may survive much longer
- Late nerve reconstruction can offer recovery of protective sensation
Clinical Perspective

- Functional result following nerve injury dependent on numerous factors
  1. extent of neuron survival
  2. rate and quality of axonal outgrowth
  3. topographic specificity of regenerating axons
  4. survival of end organs
  5. cortical reorganizational
- Clinical recovery often incomplete

References

2. Eberlin KR, Treiser M. Anatomy and physiology of the peripheral nerve. ASSH Surgical Anatomy: Nerve Reconstruction, 2017

Diagnostic Testing for Nerves

Sensory, Motor, Electrical, Imaging

Roy A. Meals, MD

Burning Questions about Sensation

“You claim my nerve is recovering, why do I have pain every time I touch something?”

“So if I have carpal tunnel syndrome, why aren’t my thenar muscles weak?”

“You tell me I am maximally recovered from my nerve laceration, why can’t I feel my electric toothbrush vibrating?”

The Anatomy of Fingertip Skin and Specialized Sensory End Organs

- papillary ridges
- Meissner corpuscles
- Merkel cell neurite complex
- digital nerve
- A-β
- C
- A-Δ
- sweat pores
- sweat duct & gland
- epidermis
- dermis
- fat
## Nerve Fiber Types

<table>
<thead>
<tr>
<th>Fiber type</th>
<th>Myelin sheath?</th>
<th>Size in microns</th>
<th>Function</th>
<th>Meters per sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>No</td>
<td>1</td>
<td>Burning pain</td>
<td>2</td>
</tr>
<tr>
<td>A-Δ</td>
<td>Yes</td>
<td>2-5</td>
<td>Sticking &amp; temp</td>
<td>20</td>
</tr>
<tr>
<td>A-β</td>
<td>Yes</td>
<td>10-15</td>
<td>Light touch</td>
<td>60</td>
</tr>
<tr>
<td>A-α</td>
<td>Yes</td>
<td>15-20</td>
<td>Motor</td>
<td>60</td>
</tr>
</tbody>
</table>

## Specialized Sensory End Organs Supplied by A-β Fibers

<table>
<thead>
<tr>
<th>Pacinian corpuscle</th>
<th>Meissner corpuscle</th>
<th>Merkel cell neurite cplx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schematic</td>
<td></td>
<td></td>
</tr>
<tr>
<td># of nerve fibers/rcptr</td>
<td>1</td>
<td>&gt;1</td>
</tr>
<tr>
<td>Recovery after laceration</td>
<td>Poor</td>
<td>Excellent</td>
</tr>
</tbody>
</table>

## What constitutes normal sensation?

1. A multitude of A-beta fibers present, supplying various types of end organs
2. Each nerve fiber functioning properly

## What can go wrong?

1. A multitude of A-beta fibers present, supplying various types of end organs
   - Laceration (physical disruption of axon and entire sheath = neurotmesis)
   - Marked compression/stretch (physical disruption of axon but continuity of sheath = axonotmesis)
2. Each nerve fiber functioning properly
   - Compression (physiologic disruption, aka ischemia = neurapraxia)

## When something goes wrong, inquiring minds want to know:

- How many nerve fibers are present? (more is better) Innervation density
- How much energy does it take to stimulate a nerve fiber? (less is better) Threshold

**POP QUIZ!**
Matching:
1. Innervation density test
2. Threshold test

Matching:
1. Threshold test
2. Innervation density test

Two Point Discrimination (The Innervation Density Test)

How wide do I have to stretch to find 2 functioning nerve fibers?
Maybe if I move along the finger I can find 2 fibers more easily.
I go wide when nerve fibers are missing, e.g., lacerations, severe compressions

Monofilament Testing (The Threshold Test)

I can’t go wide. I am looking for a single nerve fiber to stimulate but I can push only so hard.
If I can’t push hard enough to make the nerve fire, maybe my big brother can!

For Your Reference:

<table>
<thead>
<tr>
<th>Mono Filament #</th>
<th>Force in grams</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.65</td>
<td>.01</td>
</tr>
<tr>
<td>2.36</td>
<td>.02</td>
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<td>2.44</td>
<td>.04</td>
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<td>2.83</td>
<td>.08</td>
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<td>3.22</td>
<td>.17</td>
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<td>3.61</td>
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<td>3.84</td>
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<td>.75</td>
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<td>4.17</td>
<td>.98</td>
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<td>4.31</td>
<td>2.4</td>
</tr>
</tbody>
</table>

Final Exam: MFT vs. TPD

1. What test is appropriate for testing nerve regeneration after laceration and repair?
2. What test is appropriate for testing sensory loss in early cubital tunnel syndrome?
3. What test is appropriate in severe carpal tunnel syndrome when MFT is markedly abnormal?

Inquiring minds want to know:

“You claim my nerve is recovering. Why do I have pain every time I touch something?”

Rephrased: Why is pain the first sensation to return after nerve repair?

The unsophisticated unmyelinated C fibers grow back the quickest.
Inquiring minds want to know:

“So if I have carpal tunnel syndrome, why aren’t my thenar muscles weak?”

Rephrased: Why does sensory loss occur before motor loss in carpal tunnel syndrome?

The motor fibers are larger and are more resistant to compression ischemia.

Inquiring minds want to know:

“You tell me I am maximally recovered from my nerve laceration, why can’t I feel my electric tooth brush is vibrating?”

Rephrased: Why does response to low frequency vibration return poorly after nerve repair?

Because the Pacinian corpuscles reinnervate poorly.

Scratch Collapse Test for Carpal Tunnel and Cubital Tunnel Syndromes

Patient

Examiner

1

2

3

See Ref 7

Muscle Testing

Quantitate where possible

Grip and pinch meters

Compare to opposite side (ideally without examiner in the middle)

For Reference: Muscle Testing

The well-known Medical Research Council 0-5 scale

0 = no function

1 = palpable contraction, no movement

2 = movement with gravity neutralized

3 = movement against gravity

4 = movement against some resistance

5 = normal strength

Muscle Testing, Reconsidered

• McAvoy and Green:

  — Cadaver study, elbow flexion
  — Only 4% of normal force required to flex against gravity

• MRC Grade 0 1 2 3 4 5

  % of normal <4 <4 4 >4 100

  95%

Grade 4 represents 95% of potential elbow flexion strength, therefore includes both weak and strong muscles. Better system needed!
Electromyography (EMG)

Nerve conduction velocity (NCV)
• Tests nerve

Needle electrode exam (NEE)
• Tests muscle

Nerve Conduction Velocity
• Affected by the room’s temperature, patient age, anomalous innervation patterns
• Usually with surface electrodes
  Stimulating
  Recording
  • Velocity = distance divided by time
  >50 meters/sec for all upper extremity nerves
• Latency (time in milliseconds) substitutes for velocity when distance is uncertain

NCV in Neurapraxia
• Ulnar nerve
  – 54 meters/sec
  – 42 meters/sec
  – Conclusion: cubital tunnel synd

• Median nerve
  – Sensory distal latency 4.0 millisec
    • (nl <3.6 millisec)
  – Motor distal latency 4.8 millisec
    • (nl <4.6 millisec)
  – Conclusion: carpal tunnel synd

NCV in Axonmetsis and Neurotmesis
remains normal for 2-3 days

Needle Electrode Exam
• Normal muscle
  – Electrically silent at rest
  – Electrical activity proportionate to force
  – of active contraction (recruitment)

• Denervated muscle
  – Fibrillations and sharp waves at rest begin 2-3 weeks after injury
  – No change on active contraction effort

• Reinnervating muscle
  – Fibrillations and sharp waves
  – Disorganized recruitment pattern—seen 1-2 months before clinical improvement noted

Nerve Imaging
• Nerves not seen on x-ray or CT scan
• Nerves not seen well on conventional MRI
  – Magnetic Resonance Neurography (MRN)
MRN: Cervical Spine

- BP Injury: Yoshikawa
- T.O.S.: Filler
- Neurofibromatosis: Filler

Magnetic Resonance Neurography: CTS

- C6 Compression: Aagaard
- Flat: normal

References


Introduction

Pain and paraesthesias of the upper extremity
- Cervical radiculopathy
- Brachial plexopathy
- Thoracic outlet syndrome

Anatomy is key
Cervical Spine

Dermatome Distribution

- C5: Deltoid, Biceps
- C6: Biceps, wrist extensor
- C7: Triceps, wrist flexors, finger extensors
- C8: Interossei, Finger flexors
- T1: Interossei

Motor Innervation

Reflexes

- Biceps – C5, C6
- Brachioradialis – C6
- Triceps – C7
Pathomechanics of Cervical Radiculopathy

- Compression of nerve root
  - Disc pathology or herniation
  - Arthritis
  - Congenital stenosis
  - Trauma
  - Tumor

Symptoms of radiculopathy

- Based on nerve root affected
- Radicular pain, numbness, or weakness
- Neck and shoulder pain are common
- Pain, paraesthesias, weakness in distribution of the affected nerve
- Anatomy is key

Diagnosis of radiculopathy

- History and physical
  - Spurling's test: extension, ipsilateral rotation, and downward pressure
- XR
- MRI
- Electrodiagnostics
  - Primarily to evaluate for other sites of compression

X-ray

Treatment of Radiculopathy

- Initial
  - Pain control, anti-inflammatories and analgesics, +/- steroid injections
  - Traction and manipulation: controversial
  - Neck strengthening
- Surgery
  - ACDF
  - Laminectomy
  - Foraminotomy

Brachial Plexus Injuries
Brachial Plexus Injuries
- Trauma
  - congenital
  - low energy
    - stinger
  - high energy
    - motorcycle
  - penetrating trauma

Brachial Plexus Injuries
- Upper trunk palsy
  - weakness in abduction and elbow flexion
  - radial sided hand numbness
- Lower Trunk
  - lose hand function
  - ulnar sided hand numbness

Brachial Plexus Treatment
- depends on etiology and status of nerve
- sharp laceration → direct repair
- nerve graft or transfer
  - intercostal to musculocutaneous
  - spinal accessory to suprascapular
  - radial N (triceps) to axillary
  - ulnar motor to musculocutaneous

Pathomechanics of TOS
- Poor posture and descent of shoulder girdle
- Congenital anatomic variations – cervical ribs, long transverse processes, congenital bands
- Apical lung tumors (Pancoast tumors)
- Malunion or non-union of the clavicle, sternoclavicular dislocations

TOS Symptoms
- Arterial
- Venous
- Neurogenic

TOS Symptoms
- Neurogenic TOS
  - Classic presentation with objective physical evidence is rare
  - Hypothenar atrophy, decreased grip strength, sensory (C8/T1) deficits
TOS Symptoms

• Disputed Neurogenic TOS
  – Chronic, insidious onset pain of neck, upper chest, and shoulder girdle
  – Radiates into ulnar side of arm and hand, and rarely into radial side
  – Worse with elevation of arm during sleep/activity, driving, or heavy objects
  – Subtle findings may be confined to ulnar intrinsics of hand, mimic ulnar neuropathy

• Upper trunk involvement mimics cervical radiculopathy or carpal tunnel syndrome
• Left shoulder and chest pain mimics angina or heart attack
• Pain with overhead activity mimics cuff tears
• Glenohumeral instability and global upper extremity symptoms

Diagnosis of TOS

• History and physical
• Diagnosis of exclusion
• Rule out distal sites of compression
• C8-T1 cervical radiculopathy is rare but should still be excluded (usually with MRI)

• Evaluate posture and head position
• Forward head position
• Shoulder droop
• Venous collaterals
• Muscle bulk and tone
• Arm swelling
• Hand atrophy, intrinsic muscle testing
• Sensory abnormalities

Diagnosis: Provocative Testing for TOS

• Look for reproduction of symptoms or loss of radial pulse
• Beware: many positions can cause diminution of radial pulse, and are not diagnostic in isolation
• 64 volunteers
  – 17% with symptoms on questionnaire
  – 58% had one provocative test positive

Roos test – “stick up test”

• 90 degree abduction and external rotation test
• Pump hand for 3 minutes
• Rapid fatigue or reproduction of symptoms
• Most sensitive and reproducible
**Diagnosis - Imaging**

- C-spine and chest films
- Consider CT scan if suspicion high.
  - Adventitious ribs, long transverse process, cervical ribs
  - Apical lung tumors
- MRI

**Diagnosis - Electrodiagnostics**

- Generally, EMG/NCS not useful in diagnosing TOS
  - Results do NOT predict thoracic outlet syndrome
  - Useful to RULE OUT carpal tunnel or ulnar neuropathy at the elbow

**TOS: Treatment**

- Therapy: focus on postural abnormalities, muscle imbalances, and neural mobility
- Nutritional counseling, diet, and exercise program

**TOS Treatment Overview**

- Key is to correct postural abnormalities
- Generic instructions from physicians may lead to treatment with stretching, soft tissue massage, nerve mobilization, or cervical traction – this does not help and may aggravate symptoms
- Limitation of aggravating activities (heavy loads, overhead work, backpacks)
- Identification of weakened muscles and strengthening of shoulder girdle to improve posture is key
- Bring shoulder girdle back posteriorly

**TOS Treatment**

- Risk factors for failure of therapy
  - Obesity
  - Poor cardiovascular condition
- Obesity, chronic pain and associated depression exacerbate poor posture
  - Antidepressants or psychotherapy can improve symptoms

**Treatment - Surgery**

- Vascular occlusion can be treated with thrombolytics and long term anticoagulation, or surgical decompression
- First rib resection
- Claviculectomy for malunions or hypertrophic callus
  - Loss of strut → shoulder droop → worsening symptoms
Complications of Surgery

- Pneumothorax
- Nerve injury – brachial plexus from traction, long thoracic N on middle scalene, phrenic N on anterior scalene
- Vascular injury – death from exsanguination has been reported.
- Persistent symptoms -- inadequate resection vs incorrect diagnosis

Prognosis

- Review of 10 studies of conservative treatment found no randomized trials, systematic reviews, or meta-analysis.
- Unclear if conservative treatment better than no treatment, or what type of treatment is best
  - Less than 20% of patients diagnosed progress to surgery
  - When appropriately done, 75% good to excellent results in some series

Summary

- ANATOMY IS KEY
- First line treatment is always conservative
- TOS is a diagnosis of exclusion with few reproducible objective tests or measurements. Treat with postural training
- Surgical decompression works well for cervical radiculopathy and for clear cases of TOS

References

- Kuhn, JE, Lebus V, Bible JE. Thoracic Outlet Syndrome. JAAOS, April 2015. 23(4) 222-32

Thank you

Adson’s test

- Arm at the side, hyperextend neck, turn toward affected side, and inhale deeply
- Pulse diminishes
- Symptoms reproduced
Halsted Maneuver
- Costoclavicular test/military brace test
- Arms at side
- Move shoulders down and back with protruding chest
- Clavicle closes on 1st rib
- Downward compression of clavicle with traction of affected extremity accentuates symptoms

Wright’s hyperabduction test
- External rotate and abduct arm 180 degrees
- Inhale deeply
- Symptoms worsen

TOS Treatment – Step 1
- Identify and treat myofascial trigger points, local spasm, tendonitis, bursitis
  - Use relaxants, mild narcotics, antidepressants, anti-inflammatories, TCA, tegretol, neurontin.
  - Modalities such as heat/ice, TENS, ultrasound are debatable (can promote dependence on therapy)
  - Trigger injections, botox injections, and epidural steroids

TOS Treatment – Step 2
- Concurrent postural training
- Stretching, relaxation, and myofascial manipulation restore posture in C-spine, shoulder girdle
- Upper trapezius, levator scapulae, scalenes, sternocleidomastoid, pectoralis minor, and suboccipitals
- Weight loss and CV conditioning
- Avoid pathologic postures - head-forward posture leads to decreased flexibility, scapular abduction, with tightening of anterior groups and elongation of posterior groups.

TOS Treatment – Step 3
- Muscle strengthening, increased endurance, and restoration to pre-symptomatic levels
  - Not started until patient has adequate pain-free ROM
  - Aggressive therapy at this time may exacerbate symptoms
  - Typically weak in middle and lower trapezius, and serratus anterior
TOS Treatment – Step 4

• Home program for maintenance followed by return to workplace
• Analysis of workplace and job site
• Scalene stretching, cervical protraction and retraction, diaphragmatic exercises, pectoralis stretching, and shoulder circumduction
• Orthotics for scapular retraction may help, but must be part of muscular strengthening plan

MEDIAN NERVE ENTRAPMENT

ALI GHIASSI, MD

Median Nerve Compression Syndromes

• Nerve Entrapment at the Wrist
  – Carpal tunnel syndrome
• Compressive Neuropathies in Proximal Forearm
  – Pronator syndrome
  – Anterior interosseous syndrome

CARPAL TUNNEL SYNDROME

• Epidemiology
  – Classically
    • Posttraumatic
    • Female
    • Middle age
  – More recently
    • Younger
    • Industrial worker
    • Repetitive motions

RISK FACTORS

• Clear intrinsic risk factors
  • Female
  • Pregnancy
  • Diabetes
  • Rheumatoid arthritis

RISK FACTORS

• Occupational factors
  • Task repetition
  • Posture
  • Mechanical stress
  • Force
  • Vibration
  • Temperature (cold)
CARPAL TUNNEL SYNDROME
• Median nerve entrapment in the carpal tunnel
• Chronic inflammation?
• Amyloid deposition?
• Repeated mechanical stress?
• Vascular sclerosis and ischemia?

CARPAL TUNNEL SYNDROME
• Nakamichi ‘98:
  – Looked at the histology of 166 patients with CTS
  – 74% normal histology of TCL
  – 25% had mucoid, amyloid deposits
  – Concluded: No typical histological changes noted

CARPAL TUNNEL SYNDROME
• Ikdea (06):
  – Increases in carpal canal pressure
  – 10 mm distal to distal wrist crease
  – Pressures correlated with nerve conduction results

CARPAL TUNNEL SYNDROME
• Szabo (‘89)
  • Increased carpal tunnel pressures with exercise in patients with CTS
  • Sustained and delayed recovery of normal pressures after exercise in CTS

Carpal Tunnel Syndrome
• History & examination are most important tools in diagnosis
  – Szabo (JHS 99)
    • Night pain in median nerve distribution
    • Sensory changes in median nerve
    • Median nerve compression test
    • EMG/NCS not helpful in diagnosis
Carpal Tunnel Syndrome

- X-rays, CT scan, MRI not useful
- EMG/NCS are helpful in confirming diagnosis
  - Double crush syndrome
  - Diabetes
  - Difficult exam

CARPAL TUNNEL SYNDROME

- Pain
  - Along median nerve
- Paresthesias in median nerve distribution
- Normal thenar sensation

CARPAL TUNNEL SYNDROME

- Symptoms worse at night (waking up)
- Extreme wrist positions
  - Talking on phone
  - Driving
- Dropping objects due to weakness or altered sensibility
  - Cups, dishes

PHALEN’S MANEUVER

- Wrist flexion with elbow on table
- Paresthesia in response to position
- Numbness and tingling in radial digits in 60 sec. = pos. test
- Probable CTS (sen.0.75, spec. 0.47)

TINEL’S SIGN

- Tap on median nerve at wrist
- Site of irritable nerve due to axonal injury
- Tingeling and shooting pain in nerve dist.
- Probable CTS (sen. 0.60, spec. 0.67)

CARPAL COMPRESSION TEST

- Direct compression of median nerve
- Paresthesia in response to pressure
- Paresthesia occur within 30 sec.
- Probable CTS (sen. 0.87, spec. 0.90)
SENSORY TESTING

- Static two point discrimination >6 mm = advanced nerve dysfunction or nerve laceration
- Rarely useful in CTS
- Monofilaments testing is better
- Value greater than .08 gm (monofilament 2.83) in radial 3 digits
- Probable CTS (sen. 0.83)

EMG/NCS

- It’s important to remember that CTS is a CLINICAL diagnosis
- Electrodagnostic tests should NOT be used independently in making diagnosis
- Glowaki (JHS ‘96)
  - 30% Patient with CTS and normal NCS responded to surgery
  - Concluded: EMG/NCS does not correlate with surgical outcome

CARPAL TUNNEL SYNDROME

- Early
  - Intermittent symptoms
  - No weakness of thumb abduction
  - No permanent numbness or paresthesias
  - No atrophy
  - Treatment = wrist splints, activity modification, limb positioning 6–8 weeks

STEROID INJECTIONS

Preoperative response to cortisone injection
- Diagnostic value
- Prognostic value

CARPAL TUNNEL SYNDROME

- Intermediate
  - Constant paresthesias, numbness
  - No atrophy
  - +/− Muscle weakness of thumb abduction
  - Pain with irritability of nerve
  - Treatment = surgical decompression
CARPAL TUNNEL SYNDROME

- Late
  - Sensory loss
  - Muscle atrophy
  - Weakness grasping objects
  - +/- Pain
  - Treatment = surgical decompression
  - Surgery will halt progression & pain

SURGICAL TECHNIQUES

- What is the better surgical technique?
  - Open release
  - Limited open release
  - Two incision release
  - Endoscopic release

OPEN CARPAL TUNNEL RELEASE

- Under direct vision release all structures
- Explore median nerve and other carpal pathology
- Safe and efficient under local
- Larger incision
- May have more incisional irritation

OPEN VS. ENDOSCOPIC

- MacDermitt (JHS ’02)
  - Randomized blinded prospective trail
  - Short and long term outcome measures
- Endoscopic group better short term outcome
- Endoscopic group lower long term satisfaction (higher re-operation rate)
- NO SUBSTANTIVE DIFFERENCE NOTED

OPEN VS. ENDOSCOPIC

- Cochrane Review 2006
  - No better alternative than standard open CTR
  - Earlier return to work with endoscopic: conflicting results
  - No strong evidence to replace standard open CTR

ENDOSCOPIC RELEASE

- Smaller incision
- May have improved short term recovery
- Avoid palmar incision
- Regional or general anesthesia
- Not able to visualize median nerve or intercarpal pathology
- Increased risk of NV damage
Surgeons: Shared decision making
Patients: Lead decision making
Non-Operative treatment = likely not to want an injection
Decision aid (evidence based medicine) maybe helpful
Both: Value in getting family support

HAND THERAPY

- Pomerance (JHS '07)
  - Prospective randomized study
  - Two week of post operative therapy vs. home therapy
  - No change in outcome noted
  - Therapy added $600 - $900

- However, there is a role for patients with
  - Limited digital motion
  - Edema
  - Incision tenderness

SPECIAL CIRCUMSTANCES

- WORKER’S COMPENSATION PATIENTS
- MORE MATURE PATIENTS
- DEPRESSION
- RE-OPERATION

MATURE PATIENTS

- Townshend (JHS ’94)
  - 83 CTR in patients over 70 yrs
  - 80% with severe changes
  - 94% satisfied at 1 year
- Weber (JHS ’04)
  - 105 CTR in patients over 65 yrs
  - 83% very satisfied with results at 6 months
  - Reduced paresthesia, night pain
  - Improved strength & sensibility

WORKER’S COMPENSATION

- Higgs (JHS ’94)
  - CTS outcome in worker’s compensation patients
  - Residual symptoms more common in WC patients
  - 73% of WC patients changed jobs due to residual symptoms
  - 2% non-WC changed jobs
MATURE PATIENTS

- Keep in mind that 5-10% not satisfied
- Pain relief is main goal
- Important to discuss goals and recovery before surgery
- Long term recovery to be expected in most patients

DEPRESSION

- Ring (JHS '07)
  - 82 Patients with CTR
  - Survey of outcome and satisfaction
  - Dissatisfaction correlates with depression and ineffective coping skills
  - More than a peripheral nerve problem

RE-OPERATION

- Cobb (JHS '96)
  - 113 patient with CTR re-operation
  - 15 failed surgeries needing 3rd operation
- Risk factors for failure
  - Worker’s Comp
  - Pain in ulnar nerve dist.
  - Normal Nerve studies
- 20% dissatisfied with final result

American Academy of Orthopedic Surgery Recommendations Following Literature Review (from aaos.org)

- Recommend carpal tunnel release for CTS
- Epineurotomy and skin preservation procedures not recommended
- Hand therapy is not among the options used for CTS
Treatments not Specifically Addressed or Advocated

Acupuncture, Steroids, Cold laser, Diuretics, Exercise, Yoga, Vitamin B6, Fitness, Iontophoresis, Laser, Stretching, Massage, Magnets, Manipulation, Activity modification, Medications (NSAIDS, etc.), Cognitive behavioral therapy, Nutritional supplements, Phonophoresis, Smoking cessation, Therapeutic touch, Electrical stimulation, Weight Reduction

Is there better evidence?

http://www.cochrane.org/

- Looked at all randomized trails for CTS
  - 4 main studies
  - Concluded surgery relieves symptoms better than splinting
  - Not conclusive for patients with mild symptoms
  - No comparison made to injections.

http://www.cochrane.org/

- Looked at 21 trials
  - Alternative non-surgical treatment
  - Excluded cortisone injections
  - Short term benefit
    - Oral steroids
    - Splinting
    - Ultrasound
    - Yoga

REFERENCES

- http://www.jhandsurg.org/
- http://www.cochrane.org/
- http://www.aaos.org/

Appendix for self-study

PRONATOR SYNDROME

- Entrapment of median nerve in proximal forearm
- Forearm pain along median nerve
- Sensory changes in median nerve distribution
- Rare if actually real

AIN PALSY

- AIN innervations
  - FPL
  - FDP IF (MF)
  - PQ
  - No sensory component
AIN PALSY

- Complete palsy or incomplete with weakness
- FPL, FDP IF
- Pinch causes IP hyperextension
- Weak pronation with elbow flexed

Complex Regional Pain Syndrome (CRPS)

- Chronic pain syndrome most often affecting one arm or one leg
- Pain out of proportion to initial injury
- Occurs after an injury, surgery, stroke or heart attack
- Incidence: 6-26/100,000 persons/year
- Females > Males at ratio of 3.4:1
- UE > LE
- Fracture = Most common precipitating event

CRPS Background

- Neurologist Silas Weir Mitchell: “Causalgia” seen after major nerve injuries in Civil War vets at Philadelphia VA Hospital
- Sudek, 1900: Localized bone atrophy on x-ray
- Leriche, 1916: Noted similarities to ischemia, treated with sympathectomy
- Evans, 1946: Coined “reflex sympathetic dystrophy” (RSD) for syndrome w/o major nerve injury
- Int'l Assn. Study of Pain (IASP), 1994: “CRPS” because “RSD” is not solely sympathetically-mediated pain

CRPS Overview

No single unifying explanation can account for the diverse features.
CRPS is probably a spectrum of disorders.

CRPS II (causalgia) least common
CRPS I (minor RSD) most common

CRPS

- **Type 1**: No identifiable nerve injury
  - Previously known as RSD
- **Type 2**: Identifiable nerve injury
  - Previously known as causalgia when following major nerve injury
CRPS Predisposing Factors

- Psychological factors? — Probably not
- Menopause
- History of migraines
- Osteoporosis
- Asthma
- ACE (Acetyl Choline Esterase)-inhibitor therapy

CRPS Predisposing Factors

- Elevated intra-cast pressure due to a tight cast or extreme positions
- Litigation
- Genetic
  - Siblings of CRPS patients under 50 years were at three times higher risk of developing the condition
  - HLA-B62 and HLA-DQ8 alleles

The Perfect Storm for CRPS

Patient with wrist injury…
Sling/tight bandage cause:
  - Immobilization
  - Ischemia
  - Edema

Features of CRPS

- Pain out of proportion to the inciting cause
- Non-dermatomal
- Skin dry or overly moist
- Skin color: Mottled or cyanotic
- Skin temperature: Cool
- Skin texture: Smooth, shiny, non-elastic
- Edema
- Soft tissue atrophy (especially fingertips)
- Sweat: Excessive, reduced, absent

Features of CRPS

- Hair changes: Loss, finer, longer
- Nails: Rridged, curved, thin, brittle
- Joints: Stiff
- Muscles: Wasted, weak, tremor, spasms
- Radiographs: Osteoporosis
- Bone scan: Findings consistent with CRPS

Features of CRPS

- Not all CRPS is sympathetically-mediated
- Not all sympathetically-mediated pain is CRPS

Diagrams:
- Herpes zoster
- Neuralgias
- Metabolic Neuropathies
- SMP
- CRPS
- Phantom Pain
Useful Definitions and a Conundrum

- **Hyperalgesia**: Increased pain response to a stimulus that is normally painful
- **Hyperesthesia**: Abnormal increase in sensitivity to stimuli of any of the senses
- **Alldynia**: Non-painful stimuli evoke pain

When does pain become abnormal? Who says so?

**Budapest Diagnostic Criteria** for CRPS

The following FOUR criteria must be met:

1. Pain out of proportion to inciting event
2. Must report at least 1 symptom in 3 of the following 4 categories:
   - Sensory: Hyperesthesia and/or allodynia
   - Vasomotor: Temperature asymmetry and/or skin color changes and/or skin color asymmetry
   - Sudomotor/edema: Edema and/or sweating changes and/or sweating asymmetry
   - Motor/trophic: Decreased ROM and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nails, skin)

**Budapest Diagnostic Criteria** (Cont.):

3. Must display at least 1 sign in 2 of the following categories:
   - Sensory: Hyperalgesia (to pinprick) and/or allodynia (to light touch and/or deep somatic pressure and/or joint movement)
   - Vasomotor: Temperature asymmetry and/or skin color changes and/or asymmetry
   - Sudomotor/edema: Edema and/or sweating changes and/or sweating asymmetry
   - Motor/trophic: Decreased ROM and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nails, skin)

4. There is no other diagnosis that better explains the signs and symptoms

**CRPS Pathophysiology**

- Exaggerated inflammatory response
- Altered cutaneous innervation
- Central & peripheral sensitization
- Altered sympathetic nervous system fxn
- Circulating catecholamines
- Autoimmunity
- Distorted sensory representation of affected limb in brain

**Causes of CRPS Type I**

- Peripheral insult: Limb trauma/surgery, electric shock
- Peripheral and central insult: Herpes zoster, brachial plexus avulsion
- Central insult: Stroke, MS, spinal cord injury, brain injury/tumor
- Drugs: Isoniazid, phenobarbital
- Heart/lung disorders: Myocardial infarction, post-op cardiac surgery, lung disease

**CRPS, Type I**

- 50% follow trauma
  - Possibly trivial
  - Distal radius fracture
  - Surgery: Including CTR, Dupuytren's
- Immobilization contributes
  - Self-induced and/or iatrogenic
  - Immobilization by itself causes stiffness, clumsiness, atrophy of muscle, skin, fat
- Tight bandage, cast, splint

  Was it the injury, the surgery, the bandage?
CRPS: Management

• Prevent it!
  – No slings for hand and wrist problems
  – Mobilize all joints possible
    • Minimally restraining bandages, splints, casts
    • Encourage functional use
  – Elevate, elevate, elevate!
  – Loosen/replace tight bandages/casts

CRPS: Management Overview

• A. Drugs
• B. Interruption of sympathetic supply
• C. Implanted nerve stimulators, pain pumps
• D. Psychological approaches
• E. Physical forms of treatment

Treatment is clearly multidisciplinary with treatment by hand surgeon, pain management specialist, therapist and psychologist

A: Drugs for CRPS

• Anti-inflammatory (NSAIDS, steroids)
• Vitamin C (preventative)
• Topical antioxidants (DMSO)
• Anti-convulsants (e.g., Gabapentin)
• Ketamine
• Antidepressants (Nortriptyline, Cymbalta)
• Narcotics
• Calcitonin
• Intravenous immunoglobulin (IVIG)

B: Interrupt the Sympathetic Supply

• Stellate ganglion block(s)
• Surgical sympathectomy
  • Peripheral blockade
    – Local anesthetics
    – Guanethidine

These treatments won’t work when the pain is not sympathetically mediated

C: Implanted Nerve Stimulators and Pain Pumps

• Spinal cord stimulation
• Peripheral nerve stimulation
• Spinal medication pumps
• Deep brain stimulation

D: Psychological Approaches

• Active self-management and participation in a care plan
• Assess & treat patients for concomitant axis I disorders (depression, anxiety, PTSD)
• Cognitive behavioral therapy
• Biofeedback
• Learning relaxation skills
**E: Therapy**
- First line of treatment
- Active movement, resume ADLs
- Passive motion, splinting after pain is controlled
- Posture normalization
- Edema reduction
- Desensitization
- Stress loading
  - Scrubbing
  - Carrying
- Aerobic conditioning

**E: Therapy**
- Overcome kinesophobia
- Massage
- Contrast baths
- Graded motor imagery (GMI)**
- Transcutaneous electrical nerve stimulation
- Isometric strengthening exercises
- Encourage use of affected limb in ADLs
- Mirror box therapy**
- Specialized garments or wrappings

**Surgery During/After CRPS**
- Relief of CTS, cubital tunnel syndrome may help treat CRPS >> Don’t delay
- Secondary surgery after CRPS is controlled, eg, distal ulna excision after distal radius fx
  - 47% recurrence rate
  - Make sure pain manager and therapist are in town to start treatment at earliest sign of recurrent CRPS
- Pain manager often recommends peri-operative stellate ganglion block

**CRPS: Prognosis**
- Within first year, 70% improved (Bean et al)
  - 25% still met Budapest Criteria
  - Only 5% without complaints
- Patients with higher levels of anxiety and pain-related fear at beginning of therapy have worse long-term outcomes
- CRPS present for >1 year rarely spontaneously resolves
- Prognosis poorer in smokers

**CRPS References**

**Shingles**
Resurgence of virus that causes chickenpox in immunocompromised patient
0.5% > Age 60, 1% > Age 70
- Pain and rash follow a dermatome
  - Face > Trunk > Limb
- Pain (post-herpetic neuropathy) can last for years, treated with meds
- Immunization now available, advised for > 60
Parsonage Turner Syndrome

- Idiopathic brachial plexopathy
- Etiology unclear
- Abrupt onset of shoulder pain (usually unilateral) followed by a progressive neurologic deficits of motor weakness, dysesthesias and numbness
- Pain may extend to trapezius, upper arm, forearm and hand
- Pain usually worse at night
- Pain usually lasts 1-2 weeks

Nerve Mobilization

- Not published, discussed or understood in hand surgery circles
- Recent review by Mark Walsh, PT, MS, CHT, ACT (J Hand Therapy 2005, 18:241-258):
  - “Neural mobilization…is based on an eclectic compilation of theoretical concepts. There is a paucity of reported clinical studies using neural mobilization for the treatment of neuropathic pain”

Targeted Muscle Reinnervation

- Novel approach to post-amputation neuroma pain
- Provides distal target and vascularized scaffold to guide sprouting nerve axons
- Improve functional prosthesis control
- Residual nerves from the amputated limb are transferred to reinnervate new muscle targets that have otherwise lost their function
- 0/26 patients developed neuroma pain after TMR (Souza et al, 2014)

Seddon Injury Classification

- Neuropraxia
  - Temporary paralysis
- Axonotmesis
  - Likely recovery
- Neurotmesis
  - No recovery without repair
Basic Injury Types
- Compression – “Saturday night palsy”
- Traction
- Laceration

Mechanism of Injury
- Tidy wounds: knife, glass, surgical
- Untidy wounds:
  - Open fracture
  - Penetrating missile
- Avulsion

Nerve repair

Indications for operative intervention
- Sensorimotor exam + mechanism
- Associated vascular/bony
- Failure of nerve recovery
- Worsening of nerve injury
- Persistent pain
- Painful neuroma

Reasons not to repair
- General condition of the patient
- Skill/availability of operating team and specialized equipment
- Uncertain viability or state of nerve trunks
- Local or systemic sepsis
- Tendon transfer or nerve transfer will give better results

Timing of Nerve Repairs
Open Injuries
- Early exploration
- Definitive repair if sharp laceration
- Crush injury – zone of injury not apparent
Timing of Repair
Closed Injuries
- Expectant observation
  - Expect complete recovery in 6 weeks
    - If none
      - 6 wks: baseline EMG/NCS
      - 12 wks – clinical exam, repeat EMG/NCS
      - 3 months – operative exploration

Classification of Nerve Repair
- Primary repair (<1 week)
- Delayed primary repair (after 3-4 days)
- Secondary repair (>1 week)


Principles of Repair
- Meticulous surgical technique
  - (microscope preferred to loupes)
- Debride to healthy nerve ends
- Primary tension-free repair
  - Graft if tension-free repair not possible
- Match motor and sensory fascicles

Nerve Repair Technique
- Correct alignment/orientation of fascicles
- Use external markings

Group Fascicular Nerve Repair
- Median nerve @ elbow

Epineural Nerve Repair
- Superficial radial nerve
Factors in Outcomes

- Age
- Nerve gap
- Delay to repair
- Level of Injury
- Condition of nerve ends

Management of Nerve Gaps

Nerve Grafts

- Graft: tissue that depends on recipient bed blood supply
- Indication: Cannot achieve tensionless direct repair

Donor Nerves

<table>
<thead>
<tr>
<th>Donor</th>
<th>Length</th>
<th>Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sural</td>
<td>30-40 cm</td>
<td>Lateral foot</td>
</tr>
<tr>
<td>Lateral antebrachial cutaneous</td>
<td>5-8 cm</td>
<td>Lateral forearm</td>
</tr>
<tr>
<td>Medial antebrachial cutaneous (ant. br.)</td>
<td>10-20 cm</td>
<td>Medial arm and elbow</td>
</tr>
</tbody>
</table>

Cable Nerve Grafting

- Autografts (same person)
- Allografts (same species, rendered immune inert)
- Pedicled nerve grafts (nerve flaps)
- Vascularized nerve flaps
Disadvantages of Autologous Grafting

- Prolonged operative time
- Limited supply of expendable donor nerve
- Donor site morbidity
- Axons must cross 2 suture lines
- Motor axons must growth through potentially inhibitory sensory nerve environment

Nerve Conduits

- Provide safe passage for regenerating nerves
- Localizes neurotrophic factors
- Protection from scarring
- Natural, synthetic, non/absorbable
- Vein, polyglycolic acid (PGA), collagen

Avance™

- AxoGen Inc.
- Human cadaveric nerve allograft
- 1.5, 3, 5, and 7 cm lengths
- 1-5mm diameters

Nerve Conduits

- Require population by Schwann cells from neural stumps to guide axonal growth
- Short (<3cm) nerve defects

Cho et al. JHS 2012

- Sensory Function
- Motor Function

TABLE 5. Comparison With Historical Reference Literature

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Gap (mm)</th>
<th>Nerve Injury</th>
<th>Repair Technique</th>
<th>Predicted Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miller et al.</td>
<td>77</td>
<td>&lt; 50</td>
<td>Digital</td>
<td>Autograft</td>
<td>60%</td>
</tr>
<tr>
<td>Plymate and Giannotta</td>
<td>15</td>
<td>Median</td>
<td>Autograft</td>
<td>87%</td>
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<tr>
<td>Knoop et al.</td>
<td>7</td>
<td>Ulnar</td>
<td>Autograft</td>
<td>37%</td>
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<tr>
<td>Plymate and Giannotta</td>
<td>14</td>
<td>&lt; 35</td>
<td>Ulnar</td>
<td>Autograft</td>
<td>60%</td>
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<tr>
<td>von der Heydt et al.</td>
<td>12</td>
<td>5-18</td>
<td>Digital</td>
<td>NeuroCon™ type I bovine collagen tube</td>
<td>75%</td>
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<tr>
<td>Wang et al. and Kallmes et al.</td>
<td>64</td>
<td>3-25</td>
<td>Digital and mixed</td>
<td>NeuroCon™ type I bovine collagen tube</td>
<td>47%</td>
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<tr>
<td>Chien et al.</td>
<td>16</td>
<td>2-25</td>
<td>Digital</td>
<td>Neurotact® poly(D, L-lactide-co-e-caprolactone) tube</td>
<td>64%</td>
</tr>
<tr>
<td>Cho et al.</td>
<td>12</td>
<td>3-25</td>
<td>Median and other</td>
<td>Neurotact® poly(D, L-lactide-co-e-caprolactone) tube</td>
<td>8%</td>
</tr>
</tbody>
</table>

Note: Data is based on individual study parameters for successful recovery. MUMI and 5/50 by MIECC. 
°Neurotact®, Medtronic, Minneapolis, MN 
°NeuroCon™, Nuvasive, San Diego, CA 
°GEM Neurotube, Graftlink, Glenview, IL 
°Integra, Integra LifeSciences, Plainsboro, NJ
What if there is no proximal nerve end that can be used in a repair?

Nerve Transfer
Healthy proximal donor → distal palsied nerve

Principles of Nerve Transfer
- Expendable donor
- Donor with large number of pure axons
- Donor near motor end plates of muscle or target sensory nerve
- Innervates a muscle that is synergistic to the target muscle (preferred)

Nerve Transfers
Historically used in brachial plexus injuries
- No viable proximal nerve

Oberlin Transfer
- Restoration of elbow flexion
- Ulnar nerve fascicle to musculocutaneous nerve

Indications for Nerve Transfer
- Brachial plexus avulsion injuries
- Avoid dissection through scar
- Major trauma with segmental nerve loss
- Alternative to grafting in older patients
- Proximal nerve injury

Nerve Transfers
- Convert high injury to low injury
- Recruiting redundant or unimportant fascicles

~35cm~/~14in ~12cm~/~4.7 in
High Ulnar Nerve Injury

- Missing:
  - intrinsic function
  - sensation to ulnar 1 ½ digits & ulnar dorsal hand

Median to ulnar transfers - Motor

- AIN (anterior interosseous n.) → motor branch ulnar nerve

Adapted from Dvali et al. Clin Plastic Surg 2003

Median to ulnar transfers - Sensory

- Median n. to 3rd webspace → ulnar sensory nerve
- End to side

Adapted from Dvali et al. Clin Plastic Surg 2003

Cortical Reorganization

- Sensory denervation of the hand → “black hole” in sensory cortex of brain
- Tactile input from the hand causes functional cortical reorganization
- Organization of the sensorimotor cortex is not fixed
- “Hand speaks a new language to the brain.”

J. Surg. Ortho Advances 2008; 17(3) 159-164.

References


References

References

07 Ulnar Nerve Compression Syndromes
PROSPER BENHAIM, MD
UCLA Department of Orthopaedic Surgery
Talk originally prepared by David J. Slutsky MD, FRCS(C)

Nerve Gliding
- Ulnar nerve
  - above elbow: 8-10 mm
  - below elbow: 3-6 mm
  - localized scarring tethers the nerve
    ▪ joint motion & muscle pull results in traction injury proximal and distal to site

Microcirculatory Effects of Compression
- increased permeability / edema
- increased endoneurial fluid pressure
- sub-perineurial demyelination

Nerve Compression Clinical Implications
- compressed nerves have a lower threshold to mechanical pressure
- provocative nerve tests reproduce paresthesias

Experimental Stretch Neuropathy
@ 6%, motor potential amplitude decreases by 70%, but returns to normal after 1 hour
@ 12% strain, conduction is blocked and shows minimal recovery after 1 hour
Experimental Stretch Neuropathy

- Continuous stretching (2N) of rat tibial nerve for 1 hour resulted in no histologic, EDX, or functional abnormality
- 2N load applied cyclically 60-120 times/hr lead to abnormalities
- This suggests that a small strain applied repeatedly might lead to nerve dysfunction

Watanabe et al. J Hand Surg 2001

Traction Neuropathy: Pathophysiology

- Injury/scarring of mesoneurium -> nerve adherence to surrounding structures
- Subsequent movement -> traction on the nerve

Lunborg G, Dahlin LB. Hand Clinics 1992

Traction Neuropathy: Pathophysiology

- Stretching injuries -> micro-lesions -> epineural/intraneural fibrosis and permanent nerve entrapment
- Thickening of epineurium/perineurium interfere with blood flow -> dynamic ischemia.

Lunborg G, Dahlin LB. Hand Clinics 1992

Proximal Ulnar Nerve Fascicular Topography

- Approximately 20 fascicles
- Motor fibers to FCU, FDP are deep
- Motor fibers to intrinsics, sensory fibers are superficial, hence more susceptible to early compression

CUBITAL TUNNEL SYNDROME Symptoms

- Aching discomfort
  - Medial aspect of elbow
- Pain, paresthesias, numbness
  - Ulnar forearm
  - Ulnar ½ ring finger
  - Small finger
  - Ulnar dorsal ½ of the hand
- Hand clumsiness and weakness
- Exacerbation by repetitive elbow flexion
  - Sleeping position
  - Holding a phone, reading a newspaper
  - Driving
  - Leaning on a flexed elbow

Cubital Tunnel Syndrome

Lunborg G, Dahlin LB. Hand Clinics 1992
CUBITAL TUNNEL SYNDROME

Signs

- Tinel's sign over ulnar nerve
- Elbow flexion test
- Decreased sensation in ulnar nerve distribution
- Weakness of 1st dorsal interosseous muscle and FDP to RF & SF
- Atrophy of intrinsic muscles and clawing

TINEL’S SIGN

ELBOW FLEXION TEST

Correct Incorrect

FIRST DORSAL INTEROSSEOUS STRENGTH

LIGHT TOUCH SENSATION TESTING

CUBITAL TUNNEL SYNDROME

Scratch Collapse Test

Cubital Tunnel Syndrome

- Mild neuropathy:
  - Positive elbow flexion test
    - (10% false positive)
  - ± Positive Tinel's sign over cubital tunnel
  - ± Scratch collapse test

- Severe neuropathy:
  - Abnormal 2pd of ring and small fingers
  - Weak intrinsics, clawing of ring, small
  - Positive Froment’s sign

Nonoperative Treatment

- Avoidance of elbow flexion
- Avoid direct pressure on the elbow
- NSAIDs (cortisone ineffective)
- Ergonomic workstation modification
- Nighttime elbow extension splint
  - Anterior elbow splint with elbow at 30° of flexion

CUBITAL TUNNEL SYNDROME Operative Procedures

- In situ release (open vs. endoscopic)
- Medial epicondylectomy
- Anterior transposition of ulnar nerve from behind the medial epicondyle:
  - subcutaneous
  - intramuscular
  - submuscular

CUBITAL TUNNEL RELEASE ± MEDIAL EPICONDYLECTOMY

Simple In-Situ Decompression
Medial Epicondylectomy

SUBCUTANEOUS TRANSPOSITION
SUBMUSCULAR TRANSPOSITION

Beneath the flexor-pronator muscle mass
Above the brachialis muscle

Outcomes
- Good results for in-situ release in 17/18 patients with McGowan stage I (paresthesias only with normal motor and sensory exam).
- When there are constant symptoms, demyelination is present and recovery may take 6-8 months.
- Residual sensory complaints are common.
- Intrinsic wasting rarely recovers in an adult.

Ulnar Tunnel Syndrome

Etiology
- Benign tumors (ganglion >> lipoma, GCT of tendon sheath)
- Trauma (hook of hamate fracture, cycling, wheelchair athletes)
- Anomalous muscles, thickened pisohamate lig.
- Ulnar artery aneurysms, thrombosis

Clinical Presentation
- Paresthesias of ring and small fingers
- ↓ Semmes Weinstein of ring and small
- Tinel’s sign over Guyon’s canal
- Weakness of intrinsics and ADM
- Froment’s sign
- Normal FCU, FDP
- Normal Dorsal Cutaneous Br. of Ulnar N.
- NEGATIVE ELBOW FLEXION TEST

Ganglion Neurilemmoma

ULNAR TUNNEL SYNDROME
Anatomy of Guyon’s Canal

Roof  volar carpal ligament
pisohamate ligament (motor branch)
Medial  platform
Lateral  hook of hamate
Green's Operative Hand Surgery

3 Zones:

- **Type I:** mixed motor and sensory
- **Type II:** pure sensory
- **Type III:** pure motor

Nonoperative Treatment

- Activity modification
- **Bicyclists:** avoid riding with hands low on handlebars
- Avoid repetitive percussion on ulnar border of palm
- **Wrist splinting/corticosteroid injections**

Surgical Treatment Required

- Intrinsics wasting and/or sensory loss
- Space-occupying lesion
- Ulnar artery thrombosis/aneurysm -> ulnar artery repair or ligation.

OUTCOMES

- Clinical recovery in majority of patients treated for a space-occupying lesion. [Foucher, 1993]
- Motor recovery less predictable than sensory recovery, especially when compression caused by longstanding fibrotic hypothenar arch [Zoch, 1990]

- **Type IV:** pure motor with sparing of hypothenars
  - motor branch distal to ADM
- **Type V:** distal motor
  - just proximal to FDI and Add. Pollicus

Suggested Reading


Radial, Axillary and Suprascapular Nerve Palsies

Add N Esmail, MD
Chief of Service
Permanente Medical Group
Panorama City
Assistant Clinical Voluntary Professor
UCLA: Department of Orthopaedic Surgery

Radial Nerve

- **High**
- **Elbow** Radial Tunnel and PIN
- **Wrist** Superficial radial nerve (SRN) entrapment (Wartenberg’s Syndrome)

Anatomy

- **Origin**
  - Derived primarily from C6, C7 and C8. T1 contribution in ~ 11% of population
  - All three trunks (upper, middle, lower) contribute to posterior cord
  - Posterior branches into radial and axillary nerves

Anatomy

- Triceps
- Anconeus
- Brachioradialis
- ECRL
- ECRB
- Supinator
- Divides into PIN and Superficial Branch

High Radial Neuropathy: Etiology

- Humeral shaft fx, 14% incidence
- Tourniquet palsy
- “Sat. night palsy”, crutch injuries
- Anomalous muscle, rare
- Penetration by an aberrant artery
- Tumors
- Strenuous muscle activity
- Windmill pitching
High Radial Neuropathy

**Clinical Features**
- ± triceps dysfunction depending on level of lesion
- Wrist drop with inability to extend fingers or thumb
- Decreased sensation SRN distribution
- ± decreased sensation posterolateral aspect arm and forearm

**Management**
- Spontaneous onset, no mass, nl imaging
  - dynamic wrist splint, passive ROM exercises
  - if no evidence of recovery at 3-6 months, surgical exploration
    - Neurolysis
    - If humeral nonunion, shorten humerus

**Humerus Shaft Fracture**
- Somewhat controversial
- Usually injury due to contusion or mild stretch
- Most advocate non-operative management with exploration at 4 months if persists
- Exceptions: open fracture; loss of function after closed manipulation

**Tendon Transfers**
- If neuropathy has lasted longer than 12-18 months, tendon transfer indicated
  - Common tendon transfers:
    - palmaris longus to EPL
    - pronator teres to ECRB
    - FCR to EDC
PIN Compression Syndrome: Etiology
– Anatomic constraints
  • Fibrous bands, crossing veins, ECRB
  • Supinator, prox. edge (Arcade of Frohse)
    – Distal edge
  • Tumor
  • Proximal 1/3 radius fx
  • Radial head dislocation
  • Surgical retraction

PIN Compression: Management
– Treat known compressor (eg mass, radial head)
– If new onset, not progressive, and images nl:
  – 6-8 weeks long arm splint
  – Digital extnsr tenodesis splint
  – If no improvement
    - Surgical decompression

PIN Compression: Management
– If progressive or open injury:
  • Surgical exploration
  – If palsy for greater than 18 months,
    tendon transfers

Radial Tunnel Syndrome: Etiology
– Same as PIN compression syndrome
– Tumors less common
– Most commonly due to musculotendinous structures

Radial Tunnel Syndrome: Symptoms
Pain
  • deep aching pain over lateral aspect of elbow and dorsal proximal forearm,
    commonly at night
  • can radiate proximally or distally
  • pain often aggravated by activity

Radial Tunnel Syndrome: Exam
– Tender over radial tunnel
– Provocative maneuvers
  • Resisted supination
  • Resisted middle finger extension
  • Passive pronation and wrist flexion
– Weakness
  • uncommon
Radial Tunnel Syndrome

- **Differential Diagnosis**
  - Lateral epicondylitis (may coexist in about 5% of cases)
  - Parsonage-Turner Syndrome
  - Cervical radiculopathy
  - Systemic disorders
  - Chronic compartment syndrome

Radial Tunnel Syndrome: Management

- Initially, conservative
  - NSAID, rest, avoid provocative activities
  - Long arm splint with elbow in flexion and supination, wrist in extension
  - Treat lateral epicondylitis as needed
- Recalcitrant after 3 months
  - Surgical exploration
  - Results mixed

Wartenberg’s Syndrome

- Also known as:
  - Superficial radial nerve entrapment
  - Radial sensory nerve entrapment
  - Cheiralgia paresthetica
  - Handcuff neuropathy
  - Wristwatch neuropathy

Wartenberg’s Syndrome: Etiology

- Entrapment between BR and ECRL, esp with repetitive movement
- Anatomic anomalies (e.g., conjoined BR/ECRL)
- Compression over radial styloid
- Surgical injury
- Tumors
- Trauma- crush injuries, fractures, lacerations
- Synovial rupture in RA

Wartenberg’s Syndrome: Clinical Features

Paresthesias or dysesthesias dorsoradial forearm, wrist and hand
- Increased with wrist flexion, thumb flexion and ulnar deviation (false + Finkelstein test)
- Decreased sensation first web space
- Positive Tinel’s at BR tendon
- Pain may lead to decreased grip strength, although no motor denervation

Wartenberg’s Syndrome: Differential Dx

- De Quervain’s extensor tenosynovitis
- Lateral antebrachial cutaneous nerve injury
- Cervical radiculopathy
- Scaphoid fracture
- OA of thumb CMC joint
Wartenberg’s Syndrome: Management

- Initial treatment- conservative
  - NSAIDS, rest, activity modification
  - Thumb spica splint with wrist extended
  - Eliminate external compression
- If persists greater than 3 months, surgical exploration

Axillary Nerve

- C5 and C6 -> posterior cord
- Gives branches to shoulder capsule
- Goes through quadrilateral space with posterior humeral circumflex artery

Quadrilateral Space Syndrome

- Occlusion of posterior humeral circumflex artery (and axillary nerve)
- Occurs with arm in abduction, extension, and external rotation
- Shoulder pain and distal paresthesias, worse with overhead activity
- MRI: atrophy of the teres minor
- Arteriogram, Doppler studies
- EMG
- Surgery

Axillary Nerve after the quadrilateral space

- Posterior division– teres minor, posterior deltoid and terminates superior lateral cutaneous nerve
- Anterior division– middle and anterior deltoid
- Average 6cm lateral edge of acromion (can be as little 3.1cm)

Axillary Nerve Injury

- Fracture (greater tuberosity)
- Dislocations (5-33%)
- Iatrogenic
- Blunt trauma

Axillary Nerve Injury

- Weakness of abduction
- Weakness in external rotation (teres minor)
- Atrophy
- Anesthesia over lateral shoulder not reliable
Axillary Nerve Injury: Treatment

• Conservative if not transection (can wait 6-12 months)
• Nerve grafting
• Tendon transfers (latissimus, trapezius)

Suprascapular Nerve Entrapment

• Motor and sensory nerve
• Upper trunk
• Dull aching pain in post lateral aspect
• Sports, weight lifting, volleyball, baseball
• Masses, ganglion
• Iatrogenic, mobilizing large rotator cuff tears

Suprascapular Nerve Palsy: Anatomy

• Suprascapular notch
  – suprascapular ligament or shoulder joint cyst -> paralysis of supraspinatus and infraspinatus
• Spinoglenoid notch palsy of infraspinatus

Suprascapular Nerve Palsy: Etiology

• Trauma
• Iatrogenic
• Activity (volleyball, stretching at spinoglenoid notch)
• Mass effect (ganglion)

Suprascapular Nerve Injury: Clinical Findings

• Pain and weakness (RC)
• Dull aching pain in post lateral aspect
• More common in males
• Worse with arm elevation and exercise
• Depending on site either supra/infra vs infraspinatus
• Atrophy (infraspinatus more superficial)
• Diagnosis of exclusion
• EMG

Suprascapular Nerve Injury: Treatment

• Conservative
• If no improvement in 2 months consider decompression
• Good results with early decompression
• Need to know where compression is and decompress there
References


Nerve Tumors

Gina Farinas-Eisner, MD
No vested interests, No FDA off-label uses presented
Talk originally prepared by Kodi K. Azari, MD, FACS and Roy A. Meade, MD

CLINIC PATIENT #1

HPI: 51 year old LHD female incurs a 2 cm laceration to the right index finger. The laceration is repaired in the ED. The patient notices loss of sensation in the radial digital nerve distribution of the index finger. Weeks later she notices severe electrical pain with pinch.

CASE #1: NEUROMA

HPI: 51 year old LHD female incurs a 2 cm laceration to the right index finger. The laceration is repaired in the ED. The patient notices loss of sensation in the radial digital nerve distribution of the index finger. Weeks later she notices severe electrical pain with pinch.
What is a neuroma?

A group of regenerating nerve fibers which fail to reach distal targets or end organs.

Pathophysiology

- Wallerian degeneration after injury
- Sprouts emerge from proximal stump of the axon to find the distal end

Physical Exam

- The growing point of regenerating axons can produce paresthesias when tapped
- Tinel sign

Treatment

- Surgical Exploration
- Serial section until healthy fascicles
- Primary repair
- Nerve grafting

HPI: 42 year old RHD male presents with a painless mass along the flexor surface of his right small finger that has been growing increasingly larger over the past year. On exam, the lesion is mobile transversely but not longitudinally.
CASE #2: Schwannoma

What is a Schwannoma?

• Most common benign nerve tumor of the UE
• Arises from Schwann cells

Treatment

• May be “shelled out” microsurgically
• Very rare reports of malignant transformation

CLINIC PATIENT #2

HPI: 42 year old RHD male presents with a painless mass along the flexor surface of his right small finger that has been growing increasingly larger over the past year. On exam, the lesion is mobile transversely but not longitudinally.

CLINIC PATIENT #3

HPI: 20 year old RHD female presents with multiple painful subcutaneous masses located on the volar and dorsal aspect of the right hand and forearm. On exam, the lesions were varying in size, firm, and tender. The patient had faint café au lait macules and freckling in the axillary region.
CLINIC PATIENT #3

HPI: 20 year old RHD female presents with multiple painful subcutaneous masses located on the volar and dorsal aspect of the right hand and forearm. On exam, the lesions were varying in size, firm, and tender. The patient had faint café au lait macules and freckling in the axillary region.

CASE #3: Neurofibroma

What is a Neurofibroma?

• Benign nerve tumors that arise within nerve fasciculi

Physical Exam

• Solitary lesions may be seen
• Multiple neurofibromas common in Neurofibromatosis (Von Recklinghausen’s disease)
• Café au lait macule
• Axillary freckling
• Lisch Nodules

Treatment

Excision likely to require segmental nerve resection and reconstruction

• Median Nerve
• Ulnar Nerve
CLINIC PATIENT #4

HPI: 22 year old RHD male presents with gradually enlarging nontender lesion and complaints of numbness and tingling along the median nerve distribution of the thumb, index and middle fingers

CASE #4: Lipofibromatous Hamartoma

What is a Lipofibromatous Hamartoma?

Treatment

- Simple decompression
- Interfascicular resection is not possible and contraindicated
- Can have gradual deterioration of nerve function
  - Resection and nerve grafting

• Fibro fatty infiltration of nerve
• Part of DDx in child with CTS
HPI: 45 year old RHD female presents with painful subungal nodule of the left middle finger. The patient complains of cold hypersensitivity, intermittent severe pain, point tenderness.

CASE #5: Glomus Tumor

What is a Glomus Tumor?

- A rare benign neoplasm
- Arising from neuromyoarterial apparatus

Imaging

- MRI
  - High signal intensity oval shaped lesion
- Plain Film
  - Scalloped osteolytic defect
Numbness AKA Peripheral Neuropathy

- At any time 2-4% population affected
- Most common cause in USA is DM
- Hanson's disease (Leprosy) is most common in Southeast Asia
- Must determine
  - Distribution
  - Symptoms
  - Duration
  - Course

References

**Definitions**

<table>
<thead>
<tr>
<th></th>
<th>Ability to perceive stimuli</th>
<th>Sensitivity to stimuli</th>
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</thead>
<tbody>
<tr>
<td>Hypesthesia</td>
<td>Diminished</td>
<td>Intact</td>
</tr>
<tr>
<td>Anesthesia</td>
<td>Absent</td>
<td>Intact</td>
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<tr>
<td>Hypalgesia</td>
<td>Intact</td>
<td>Diminished</td>
</tr>
<tr>
<td>Analgesia</td>
<td>Intact</td>
<td>No sensitivity</td>
</tr>
</tbody>
</table>

**Definitions cont**

- Hyperpathia, hyperesthesia, and allodynia all refer to increased sensitivity to sensory stimuli
- In peripheral neuropathies, patients can vacillate between periods of hyperesthesia and hypesthesia – Don't be fooled

**Sensory Exam**

- **Peripheral Nerve Distribution**

**Sensory Exam**

- **Dermatome like distribution**

**Sensory exam**

- Complete spinal cord injury, appears easy to diagnose; however this presentation is rare

**Sensory exam**

- Instead, a brain stem injury could have this pattern of paresthesia
- Whereas a central cord syndrome would present with this pattern
Sensory exam

- Common in axonal neuropathies present in a stocking glove distribution
  - Feet affected first!

Sensory testing

- Follow the patterns
  - Confined to distal aspect of limb
  - Confined to discrete area of a limb
  - Confined to the whole limb, side of body
  - Involves reflexes
  - Involves cognitive deficits

Clinical Pearls

- Distribution of peripheral nerve; think of that nerve – Carpal tunnel, median nerve
- Distribution of a dermatome; think of the corresponding nerve root – C5 radiculopathy
- Stocking-glove sensory loss; think axonal neuropathies - diabetes

Axonal Neuropathies

- Diabetes mellitus
- Alcohol
- Vitamin B12 deficiency
- HIV
- Lyme Disease
- Uremia
- Chemotherapy
- Leprosy
- Syphilis
- Vasculitis
- Amyloidosis
- Multiple sclerosis
- Idiopathic
- Paraneoplastic neuropathy

Diabetes

- Neuropathy secondary to microvascular injury to the vasa nervorum which supplies the nerves
- Occurs in 20% of DM patients
- Implicated in 50-75% of nontraumatic amputations
- Durations of DM, tobacco use, age, HTN and hyperlipidemia are also risk factors

Diabetic Neuropathy

- Clinical manifestations
  - Numbness
  - Hypalgesia
  - Diarrhea
  - Erectile dysfunction
  - Dizziness
  - Vision changes
Diabetic Neuropathy

- Pearls to spot DM
  - Classic Triad
    - Polyuria, polydipsia, polyphagia
    - Increased urination, drinking, and eating
  - Acanthosis nigricans
  - Loss of proprioception
  - Stocking glove distribution
  - Ketone (Fruity/Alcoholic) odor in breath

Diabetic Neuropathy

- In both diabetes and syphilis, patients may present with a Neuropathic or Charcot joint
- Due to the insensate nature of the joint, it gradually degenerates and deforms
- This is uncommon in nonweightbearing joints

Alcoholic Neuropathy

- Characterized as gradually progressive affecting sensory, motor, and autonomic nerves.
- Starts symmetrically and distally (feet first)
- Associated with loss of reflexes
  - Loss of ankle jerk first

Alcohol Neuropathy Treatment

- Stop drinking
- Thiamine supplementation
- Better nutrition
- Complete recovery is uncommon
- Some medications can help with burning dysesthesias

Alcohol

- 32% of heavy users have peripheral neuropathy
- Confounded by nutritional deficiency, as well as being a risk factor for compression neuropathies
- Associated with “Saturday night palsies”

HIV Neuropathy

- Hypothesized to be secondary to dorsal root ganglion damage by the virus
- Occurs in 30% of HIV/AIDS pts
- Normally occurs in the feet

HIV Viral Load

- Infection
- Weeks
- Years
- Neuropathy
- Death
**HIV Neuropathy**

- **Main complaints**
  - Bizarre burning
  - Hyperalgesia
  - Allodynia

- **Risk factors**
  - DM
  - Nutritional deficiencies
  - HIV Medications

**Multiple Sclerosis**

- Idiopathic CNS inflammatory disease
- Demyelination and axonal degeneration
- Young adults
- 2 Women: 1 Man
- MRI diagnostic for lesion
- Differential includes spinal cord compression, vitamin deficiency,
  - Clinical and radiographic lesions separated in time and space

**Multiple Sclerosis**

- Where is the neuropathy?
  MS is a constellation of symptoms;
  - Depression
  - Fatigue
  - Numbness/pain
  - Urinary problems
  - Nystagmus
  - Red color perception
  - All of which wax and wane

**Schwann cell**

Photomicrograph of gradient
Of healthy and diseased myelin

**Syphilis**

- "Great imitator"
- First outbreak in 1494
  - French besieging Naples
- Primary syphilis
  - Skin lesion 21 days after exposure – chancre
  - Caused by Treponema pallidum (bacteria)
Secondary Syphilis

- Secondary syphilis
  - 6-8 weeks after exposure
  - Rash in palms and hands
  - Most contagious

Tertiary Syphilis

- Occurs 1-10 years after initial infection
- Changes in personality
- Tabes dorsalisis
  - Dorsal column disease causing a shuffling gait
- Neuropathic/Charcot joint disease
  - Loss of proprioception

Lyme Disease

- Emerging infectious disease caused by tick bites – 1% of bites
- Bacterial infection easily treated by antibiotics
- When untreated can cause a variety of joint, heart, and nervous system disease

Lyme Disease

- Incubation period 1-2 weeks
- Initial erythema chronicum migrans
- Then, headache, fever, malaise
- Late persistent infection after months - polyneuropathy

Lyme Disease

- Polyneuropathy symptoms include
  - Numbness
  - Shooting pain
  - Difficulty with concentration
  - Depression
  - Bell’s palsy

Chemotherapy Neuropathy

- Chemotherapeutic Culprits
  - Methotrexate (encephalopathy)
  - Rheumatoid Arthritis
  - Taxanes, ie Paclitaxel or Taxol (burning paresthesias and loss of reflexes)
  - Breast Cancer
Chemotherapy
- Vincristine (both motor/sensory fibers, virtually all patients)
  - Leukemias
- Propylthiouracil (dose dependent)
  - Thyroid disease
- Thalidomide (75% get sensory neuropathy but reversible with dosage adjustment)
  - Myeloma, Ovarian

Hansen’s Disease (Leprosy)
- Leprosy – Greek for scales on a fish
- Caused by Mycobacterium leprae
- Granulomas of the nerves, respiratory tract, skin, and eyes
- Since 600 BC
- Still leper colonies today
- Not actually highly contagious

Hansen’s Disease
- Incubates either for weeks or as long as 30 years (average 3-5 years)
- Affects 3 million/year (30% get neuropathy)
- Loss of sensation -> repeated injuries, infection due to unnoticed wounds -> loss of body parts/deformity
- Affects Schwann cell
  - Causes inflammation/edema
  - Yet scientists do not understand how unmyelinated fibers are affected

Summary
- A numb finger is not always carpal tunnel
- Review the distribution of altered sensation with patient
- What are the other symptoms
- Look at the big picture
  - Past medical history, travel history, habits, diet of patient

Be a patient advocate, early diagnosis is always key

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- Robinson, LR. Role of Neurophysiologic Evaluation in Diagnosis. JAAOS. 2000 May;8(3): 190-199

References
- Calabresi, PA. Diagnosis and Management of Multiple Sclerosis. Am Fam Physician. 2004 Nov 15;70(10):1935-44
References


11 Weakness/Spasticity

Roy A. Meals, MD

no conflicts, no off-label FDA treatments

Talk originally prepared by Lucie Krenek MD

Stroke

- Leading cause of hemiplegia in adults
- Most recovery in first 6 months
- Functional recovery can continue
- Upper extremity < lower extremity recovery
- Flaccid paralysis immediately
- Spasticity over several weeks
- Associated perceptual/cognitive/visual losses

Stroke – Treatment

- Goal - ↑ hygiene, ↓ pain, ↑ function
- Acute
  - Prevention of contractures
  - Brachial plexus and phenol nerve blocks
  - Botulinum (Botox)
- Chronic treatment of contracture
  - Antispasmodic medication
  - Surgery +/- after 6 months

Stroke - Shoulder Adduction/IR Contracture

- Spastic subscapularis, pectoralis major, latissimus dorsi, teres major
- Release pectoralis major and subscapularis, or all in nonfunctional extremity
- Pain
  - Complex regional pain syndrome
  - Spasticity
  - Inferior subluxation

Stroke - Elbow

- Flexion contracture
  - Biceps, brachialis, brachioradialis spasticity
  - Release some or all elbow flexors depending on severity
  - Severe contracture may require serial postoperative casting
Stroke - Forearm
- Pronation contracture
  - Pronator teres spasticity
  - Proximal pronator teres advancement
  - Tendon lengthening in nonfunctional upper extremity

Stroke – Wrist and Fingers
- Flexion contractures
- FCR, FCU, PL spasticity
  - With functional hand – cut PL, lengthen FCU and FCR
  - Nonfunctional hand – lengthen/cut long digital flexors

Stroke - Thumb
- Thumb-in-palm deformity
  - Adductor pollicis/first dorsal interosseous/thenar spasticity
  - Release muscle origins

Stroke - Weakness
- Wrist extension
  - FCU transfer to ECRB
- Intrinsic muscle imbalance/spasticity
  - Intrinsic plus deformity can occur if only hand extrinsics are lengthened

Charcot-Marie-Tooth (CMT) Disease
aka
Peroneal Muscular Atrophy
Hereditary Motor-Sensory Neuropathy

CMT
- Most common hereditary neuromuscular disorder
- Usually autosomal dominant
- Chronic distal sensory and motor neuropathy
- Symmetric involvement
- Parasthesias and cramps
- Onset: late childhood or adolescence
- Normal life expectancy
- Different types, affecting either axon or myelin
CMT – Symptoms

- Lower extremity – begins first
  - Gait problems
  - Distal weakness legs and feet
  - Inability to heel walk
  - Foot drop, ankle instability
  - Pes cavus deformity, hammer toes
  - Reduction in distal tendon reflexes

CMT – Symptoms

- Upper extremity
  - Involvement milder than lower extremity
  - Rarely extends above elbow
  - Atrophy of hand and forearm muscles
  - Intrinsic-minus hand deformity
  - Difficulty with fine motor function and strength

CMT – Treatment

- Stabilization of ankles with orthoses, tendon transfers, fusion
- Moderate aerobic exercise
- Muscle stretching/strengthening
- Excessive weight training not recommended

CMT – Treatment

- Intrinsic minus hand most debilitating
  - MP extension block splint
  - Volar MP joint capsulodesis
  - Surgical – FDS\textsuperscript{R} -> proximal phalanges

Amyotrophic Lateral Sclerosis

Lou Gherig’s Disease

Amyotrophic Lateral Sclerosis

- Loss of upper and lower motor neurons
- No sensory or autonomic involvement
- Prevalence 3-5 per 100,000
- Etiology unknown
  - Increased risk - pesticides, smoking, military, familial
ALS – Treatment

- Controversy regarding exercise
- Strengthening and moderate endurance exercise may result in better function
- Little can be done to improve quality of life, fatigue, or muscle strength
- Ventilatory support when respiratory muscles become affected

Guillain-Barre Syndrome (GBS)

- Acute autoimmune polyradiculopathy
- Demyelination of peripheral nerves
- Recovery occurs with remyelination
- 70% preceded by acute infection
  - usually respiratory or gastrointestinal
  - Flu shot/other vaccines
- Plateau by 4 weeks

GBS - Symptoms

- Rapid onset ascending motor paralysis
- May have sensory/autonomic symptoms
- Generalized muscle weakness in lower extremities, progresses to upper extremities
- Back, shoulder and neck pain
- Later: muscle atrophy, and potential for contractures due to lack of motion
- Severity of weakness highly variable

GBS – Treatment

- Acute phase
  - Gentle strengthening
  - Maintenance of range of motion
  - Prevention of contractures
- Medical treatment crucial - IV immunoglobulin

GBS - Treatment

- Recovery phase
  - Retraining necessary for many daily activities, ie grooming, gait
  - Muscle strengthening
  - Supervised exercise programs help overcome fatigue and improve function

GBS - Prognosis

- Most patients need hospitalization
- Up to 30% require ventilatory support
- <5% mortality, usually due to pulmonary complications
- 85% achieve full recovery by 1 year
- Poorer prognosis for older patients, late treatment, severe attacks
Acute Flaccid Myositis (AFM)

- On CDC’s radar since 2014
- Sudden arm/leg weakness
  - May also affect eye/face/throat/sphincters
- Mostly affects young children
- Incidence 1-2/million
- Cause: viral? 90% preceded by mild respiratory illness/fever
  - Not polio

10 patients:
- 4-35 day hospitalization
- one on ventilator
- no deaths
- 7 received OT/PT (4 inpt, 3 outpt)
- 6-12 weeks later: 5/9 normal, 3/9 residual weakness, 1/9 ambulation with assistance


References

- Adams and Victor’s Neurology, AH Ropper, RH Brown. 8th Edition
- Current Orthopedics, AE Keenan, S Mehta

http://www.ninds.nih.gov/disorders/charcot_marie_tooth/detail_charcot_marie_tooth.htm

Supplementary Material
Polio

- Lower motor neuron lesion
- Decreased reflexes and muscle wasting
- Proximal muscles affected
- Sensory loss rare
- Viral infectious disease
- Fecal-oral transmission
- Usually infects children under 5 years
Polio
- Rare in developed countries
- Afghanistan, Nigeria, Pakistan, India
- Less than 2000 cases per year
- 95% of infected patients asymptomatic
- 0.5% irreversible paralysis, usually legs
- Of those with paralysis, 5-10% die due to involvement of respiratory muscles

Polio – Treatment
- Acute
  - Range of motion/splinting
- Subacute
  - Prevent deformity and preserve function
  - Splinting/bracing to maintain joint position and supplement function
- Residual stage
  - Surgical intervention if needed

Post Polio Syndrome
- Muscle pain, severe fatigue, cramping, fasciculations
- Occurs years after acute illness
- Overuse of muscles that were originally affected
- Slowly progressive weakness in already weak muscles
- Treatment - limited exercise with frequent rest

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**Reinforcement Quiz**

1. The cell bodies of peripheral sensory nerves are in the
   A. anterior horn of spinal cord
   B. dorsal root ganglia
   C. white matter of brain
   D. gray matter of brain
   E. white matter of spinal cord

2. What is the correct relationship of nerve injury severity?
   A. axonotmesis > neuropraxia > neurogenesis
   B. neurotmesis > axonotmesis > neuropraxia
   C. neuropraxia > neurotmesis > axonotmesis
   D. sensory > motor > autonomic
   E. men > women > children

3. In a patient with recent onset of mild carpal tunnel syndrome symptoms
   A. two-point discrimination will be altered
   B. monofilament testing may be normal
   C. thenar muscle wasting will precede monofilament changes
   D. Meissner corpuscles will be atrophic
   E. loss of vibratory sensation to 256 cycles/second will be present

4. A successful nerve transfer requires
   A. a healthy proximal donor nerve and a palsied distal recipient nerve
   B. a distal palsied donor and a healthy recipient nerve
   C. complete absence of donor muscles for tendon transfers
   D. more rehab than comparable tendon transfers
   E. more post-op immobilization than comparable tendon transfers

5. The Roos “stick up test” for thoracic outlet syndrome consists of
   A. shoulder abduction and internal rotation
   B. turning head away from the side being tested
   C. externally rotating and abducting shoulder 180 degrees
   D. checking for radial pulse with patient seated
   E. shoulder abduction to 90 degrees and external rotation
Reinforcement Quiz

6. Which diagnostic test for carpal tunnel syndrome has the highest sensitivity and specificity?
   A. Phalen test
   B. Tinel test
   C. Adson maneuver
   D. median nerve compression test
   E. Allen test

7. In cubital tunnel syndrome, why do sensory changes and intrinsic wasting occur before forearm muscle weakness?
   A. some nerve fibers have more myelin covering
   B. the intrinsic muscles are smaller than the flexor carpi ulnaris
   C. the sensory and motor fibers to hand are superficial in the ulnar nerve at the elbow
   D. the sensory and motor fibers to hand are superficial in the ulnar nerve at the wrist
   E. the fibers affected early originate solely from the C8 nerve root

8. Which nerve is most likely to be injured with a humeral shaft fracture?
   A. median nerve
   B. ulnar nerve
   C. suprascapular nerve
   D. radial nerve
   E. medial pectoral nerve

9. A nerve sheath tumor (Schwannoma) in a digital nerve
   A. will move more medially laterally than proximally distally
   B. will move more proximally distally than medially laterally
   C. is far less common than a neurofibroma
   D. will cause sensory loss if excised
   E. will cause motor loss if excised

10. Neuropathy occurs in what percent of patients with diabetes?
    A. 1
    B. 5
    C. 20
    D. 50
    E. 95

11. “Upper motor neuron” disorders include conditions such as
    A. stroke, spinal cord injury
    B. brachial plexus palsy
    C. Parsonage-Turner syndrome
    D. Shingles
    E. proximal median nerve lacerations

12. Chronic regional pain syndrome is
    A. Usually preceded by a viral infection
    B. Managed best by the patient’s personal physician
    C. Probably a spectrum of disorders
    D. Fully responsive to stellate ganglion blocks
    E. Relieved by tight serial casting

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