I. Median Nerve Entrapment at the Wrist: Carpal Tunnel Syndrome (CTS)

A. Anatomy

1. The carpal bones and the carpal ligament create a confined space.
2. The median nerve and 10 tendons transverse the carpal tunnel {FDS (4), FDP (4), FP, and FCR}. (See Figure 1)
3. Any condition that reduces the cross-sectional area may cause CTS.
   a. Distortion of the bony or ligamentous elements (arthritis, old fracture, ganglion cyst, etc.)
   b. Increasing the content volume (edema, lipoma, anomalous tendon/muscle).
   c. Increased susceptibility of nerve to pressure (diabetes mellitus, hereditary neuropathy, HNPP, or other polyneuropathy).
4. Wrist configuration may predispose to CTS
   a. Calculate (AP diameter)/(side-to-side width)
   b. Values > 0.7 (more “box-like”) diagnostic of CTS
   c. Values of 0.55-0.75 strongly correlate with increasing latency

B. Clinical Syndrome

   1. Epidemiology
      a. Women: Men = 3:1
      b. Most common ages 40-60 with second peak in 70’s where gender ratio equal
      c. Prevalence 2.7% (clinically and electrophysiology confirmed)
2. Symptoms
   a. Numbness, tingling, burning, or deep pain in the hand
   b. Sensory loss most prominent in digits 2 and 3
   c. Complain of loss of control of the hand (sensory and opposition loss)
   d. Weakness may be noted but usually mild
   e. Pain may be poorly localized, even with radiation into arm and shoulder
   f. Neck pain should suggest other etiology
   g. Symptoms usually worse at night or early morning
   h. Morning hand stiffness, maybe mistaken for arthritis
   i. Reported relief by shaking hands
   j. Symptoms increased by activities using fingers and gripping
      (1) Knitting
      (2) Driving car
      (3) Hand tool use (hammering, painting)
      (4) Typing
      (5) Holding a book, newspaper or phone

3. Signs
   a. Sensory loss (touch, pin-prick) digits 1, 2, 3 (half of 4). 20-50% may be normal
   b. Weakness APB
      (1) Test abduction, no opposition.
      (2) Opposition uses other (ulnar) muscles
   c. Positive Tinel sign with percussion over nerve
      (1) Paraesthesias in distribution of nerve
      (2) Reproduction of pain
   d. Positive Phalen maneuver (sign)
      (1) Wrists flexed 90 degrees for 30-60 seconds
      (2) Reproduces symptoms
      (3) Reverse Phalen’s test: both hands dorsiflexed 30-60 sec
   e. Negative examination for localized musculoskeletal/radicular disease.

C. Associated disorders
   1. Mechanical factors as above
   2. Repetitive wrist flexion
   3. Local inflammation or swelling (acute wrist fracture, hematoma, suppurative tenosynovitis, rheumatoid arthritis exacerbation, episode of unaccustomed manual work)
   4. Pregnancy (18-62% of pregnant women develop symptoms with 7-10% objective signs and/or EMG abnormalities)
   5. Polyneuropathy (susceptible nerve as above)
   6. Other systemic diseases
      a. Hypothyroidism (30-38% have nerve conduction studies consistent with CTS, and CTS may be presenting feature of diagnosis)
      b. Amyloidosis (CTS symptoms often antedate other symptoms in primary amyloid from plasma cell proliferative disorder, and synovial tissue taken at time of CTS surgery will stain for amyloid)
c. Renal dialysis (more common in hand with AV fistula, but found to be associated with increased levels of beta-2-microglobulin, poorly cleared by dialysis)

d. Obesity (relationship between BMI and CTS)

e. Wheelchair use (40-55% of paraplegic wheelchair users, likely from weight-bearing on wrists in maximum extension with transfers)

f. Acromegaly (often bilateral)

D. Conventional Electrodiagnosis

1. Sensory studies measuring 14 cm from digit 2. (Sensitivity 65%, Specificity 98%)
   a. Absolute limit of 3.7 msec DL (may be normal in 25% with CTS)
   b. Side-to-side, Median DL difference of 0.5 msec significant
   c. Median-Ulnar latency difference of 0.5 msec
   d. ALWAYS check other sensory to exclude polyneuropathy
   e. Normal in digit 2 but abnormal in digit 3 in 5% of patients with CTS

2. Motor studies measuring 7 cm from motor point. (Sensitivity 63%, Specificity 98%)
   a. DL longer for median than ulnar is a possible artifact
      (1) Nerve doesn’t follow straight line
      (2) Median-Ulnar latency difference greater than 1.7 msec significant
   b. Be suspicious of motor only CTS (? MND v root)
   c. Side-to-side, Median DL difference of 0.5 msec significant
   d. Side-to-side, Median Amplitude difference of 50% significant

3. Needle electromyography
   a. Examine APB, FDIH-ulnar nerve, FPL-anterior interosseous nerve:
      (same root, different nerve)
   b. Decreased recruitment correlates with weakness
   c. Amount of fibrillation (1+ to 4+) relates to axonal degeneration
      (1) Not helpful in predicting prognosis
      (2) Sometimes used to recommend surgery
   d. ALWAYS do root screen, C5-T1 (biceps, pronator, triceps)

E. Refinements of Electrodiagnosis: Per 2002, AAN & AAPMR Practice Parameter, for electrodiagnostic studies for CTS when initial median sensory NCS across the wrist is normal then:

1. Mid-palmer stimulation
   a. Stimulate median and ulnar nerves in the palm, orthodromic “bar” electrode, recording over wrist 8 cm distance
   b. Absolute latency of >2.1 msec significant (Sensitivity 74%, Specificity 97%)
   c. Relative difference median to ulnar DL of > 0.5 msec significant
      (Sensitivity 71%, Specificity 97%)

OR:

2. Difference of sensory latency to digit 4 (ring finger) (median and ulnar nerve)
   a. Antidromic stimulation of (1) median nerve at wrist and (2) ulnar nerve at wrist (same distance) recording digit 4, ring electrodes
b. 95% of normal subjects within 0.3 msec

OR:

3. Difference of sensory latencies to thumb (median and radial nerve)
   a. Antidromic stimulation of (1) median nerve and (2) radial nerve 10 cm from active recording ring electrode around base of thumb
   b. 95% of normal subjects within 0.2 msec (Sensitivity 65%, Specificity 98%)
   c. Cathode over distal lateral volar forearm stimulates both median and radial nerves simultaneously with a double hump NAP in CTS (Dr. E Johnson’s “Bactrian Camel” sign)

OR:

4. Comparison of antidromic sensory peak latency to digit 3 when stimulating at wrist (14 cm) and then mid-palm (7 cm) (wrist and palm segment compared to digit segment) (Sensitivity 85%, Specificity 98%)
   a. Normal palmar latency 1.0 to 2.1 and normal amplitude 30-70 microvolts
   b. Normal ratio (%), palm/wrist: latency= 50% and in CTS palm/wrist latency ratio decreased as latency prolonged with stimulation at wrist because response slowed across carpal tunnel; amplitude =135%, greater if conduction block
   c. Compare both amplitude and duration of SNAP. If amplitude reduced, duration must be prolonged or the low amplitude may not be significant. Normal duration 1.1 msec
   d. In polyneuropathy palm/wrist latency ratio would be unchanged as no focal slowing at the wrist

F. Treatment

1. Treat underlying disease, if any
   a. If considering space-occupying mass, Ultrasound or MRI can be done

2. Conservative treatment
   a. Avoidance of aggravating activities (no data/studies), natural history study of 441 hands demonstrated 21% improved at 10-15 months with no treatment, but patients did alter occupational and recreational activities
   b. Splint (neutral) (after six weeks symptoms improved or absent in 54%, and in 37% after 3 and 18 month assessments)
   c. Prednisone (20mg daily for 2 weeks then 10 mg daily for 2 weeks improved symptoms in one study), and two other studies report improvement with brief, oral steroids. Diuretics and NSAID are ineffective.
   d. Intracarpal steroid injection (one study reported equivalent to surgery at 1 yr)
      (1) May have relapse, optimal dose and frequency of injections unknown.
(2) Good evidence for effectiveness and low risk, less costly and less time off work than surgery.

3. Surgical decompression
   a. Randomized controlled study of splints vs surgery demonstrate 72% of splinted patients had symptoms controlled at 12 months vs 92% of surgical
   b. 75% (range 27-100%) are cured or improved to satisfactory state

II. Pronator Syndrome
   A. Anatomy (see Figure 2)
      1. Compression of proximal nerve by pronator muscle
         a. Pronator hypertrophy
         b. Local hematoma, etc.
      2. Constriction where median nerve passes between two heads of pronator teres muscle and under the fibrous arch of the flexor digitorum sublimis
      3. Fibrous band from ulnar head of pronator teres to sublimis bridge
      4. Ligament from medial epicondyle to radius

![Figure 2](image-url)
B. Clinical syndrome- true neurogenic, pronator syndrome is fairly rare
   1. Symptoms
      a. Pain in flexor surface of the proximal forearm (likely reflects muscular effort, not nerve damage)
      b. Paresthesias of the hand (aggravate with forced pronation)
      c. Onset related to forceful pronation
      d. Weakness of grip is not a common complaint
      e. Nocturnal paraesthetic discomfort is uncommon
   2. Signs
      a. Tenderness to deep pressure over pronator teres in virtually all cases
      b. Tinel’s sign over site of entrapment
      c. Weakness, often only slight, in median innervated muscles of both forearm and hand
      d. Sensory deficit over cutaneous distribution of median nerve including thenar eminence (usually none to mild)
      e. Aggravate paresthesias by forced pronation

C. Electrophysiologic studies
   1. Needle electrode examination
      a. Sparing of pronator teres
      b. Abnormalities of other median innervated forearm muscles and abnormalities of thenar muscles
   2. Conduction studies
      a. Slowing of conduction through forearm
      b. Distal studies normal, motor and sensory
      c. Local site of conduction block can be demonstrated; localized slowing and amplitude drop (conduction block)
   3. Differentiation from AINS may be difficult but treatment is the same

D. Treatment
   1. Period of rest
   2. ? steroids
   3. Exploration of nerve, particularly with progressive weakness

III. Anterior Interosseus Nerve Syndrome
     A. Anatomy
        1. FPL
        2. Pronator quadratus
        3. Radial part of flexor profundus (particularly part serving index finger)
     B. Clinical syndrome
        1. Symptoms
           a. Pain over proximal flexor surface of forearm
              (1) May be severe, more often with inflammatory or non-compressive etiology
              (2) Often worse at night
              (3) May be painless
           b. Onset usually spontaneous or associated with vigorous exercise; often sudden but may begin during sleep
c. Weakness is a common complaint, and may be the presenting complaint

2. Signs
   a. Tenderness over proximal flexor surface of forearm
   b. Weakness is the prominent feature-FPL most consistently. No weakness of thenar muscles.
   c. Minimal limitation of elbow extension has been reported
   d. No sensory deficit
   e. Circle test. Pulp of thumb to index finger; requires strong flexion at the terminal IP joint of both digits (FP & FPL).

![Figure 3](image)

C. Etiological factors
   1. Chronic compression: Accessory head of FPL, Tendinous origin of FDS to long finger, Fibrous origin of FDS
   2. Acute Injury: Direct trauma, midshaft radius fracture
   3. Inflammatory: in isolation or associated with brachial plexopathy
   4. Pseudo-AIN: really a partial proximal median mononeuropathy: neuroma, venous or arterial catheterization/puncture, proximal radius fracture, supracondylar fracture or multifocal motor neuropathy

D. Anatomical anomalies
   1. Martin-Gruber anastomosis (about half arise from the AIN)
      a. Link AIN and ulnar nerve
      b. Carry motor fibers from AIN to AP, FDI, ? ADQ)
   2. The FDP to the long finger may be supplied by ulnar nerve
   3. The FPL may receive a branch from the main trunk of median nerve

E. Electrophysiologic studies
   1. Needle electrode examination
a. Abnormalities in FPL, FDP 1 & 2, and PQ sparing of all other muscles, exp. thenar  
b. Primarily a needle EMG diagnosis

2. Conduction studies  
a. Latency from elbow to PQ may be prolonged  
b. Analysis of evoked CMAP for conduction block

F. Treatment  
1. Inflammatory or traumatic injury type often improve spontaneously with complete or partial recovery as long as 12 months after onset  
2. If progressive worsening and concern for compressive lesion then imaging and surgical exploration maybe indicated

IV. **Humeral Supracondylar Spur Syndrome (Ligament of Struthers)**  
A. Presents clinically like a pronator syndrome  
B. Aggravated by forearm supination and elbow extension which may obliterate the radial pulse  
C. Spur may be palpable  
D. EMG abnormalities of all median nerve innervated muscles including pronator teres  
E. Supracondylar conduction block can be demonstrated

REFERENCES

MONONEUROPATHY II
PERONEAL, ULNAR AND RADIAL NERVES

I. DEFINITIONS
A. Compressive neuropathy - damage to a nerve from pressure at any point along the nerve
B. Entrapment neuropathy - a form of compressive neuropathy due to pressure occurring where the nerves are normally confined to narrow anatomic passageways

II. FACTORS INFLUENCING DEGREE OF DAMAGE
A. Generalized peripheral neuropathy - acquired; familial
B. Anatomy
   1. Peripheral nerve fibers are more vulnerable than central ones with damage to selective fascicles only.
   2. Nerves with large amounts of epineurial tissues are less susceptible than those with less epineurial tissue.
   3. Relationship of vasa nervorum to compressive structures

III. EVALUATION
A. Clinical
   1. Localized pain at rest; may radiate.
   2. Paresthesias in cutaneous distribution of nerve
   3. If mild, may have normal motor and sensory exam.
   4. Rule out plexus or radicular lesion.
   5. Examine course of nerve for thickening, musculoskeletal abnormalities, and Tinel's sign.
B. Electrodiagnostic evaluation

1. Focal demyelination as shown by slowed conduction or conduction block across abnormal nerve segment.

2. May see decreased amplitude CMAP and SNAP from axonal loss or conduction block

3. F-wave latency probably not significantly changed as damage is over a relatively short segment.

4. EMG evidence of axonal loss and reinnervation; if early, decreased numbers of voluntarily recruited motor unit potentials.

C. Other

1. Intradermal histamine injection - flare component dependent on intact C-fibers

2. X-ray evidence of bone, joint or soft tissue abnormality in area where suspected lesion is located

3. Laboratory or electrodiagnostic evidence of generalized neuropathy

IV. PERONEAL NEUROPATHY

A. Anatomy (Figure 1)
1. Posterior division of sciatic nerve with L4-S2 root contributions

2. Common peroneal nerve arises mid-thigh and branches in the following order:
   a) To short head of biceps femoris proximal to fibular head
   b) To deep and superficial branches "near" fibular head through attachment of superficial head of peroneus longus muscle.

   (1) Deep - to anterior tibialis, extensor hallucis longus, extensor digitorum longus, peroneus tertius, extensor digitorum brevis with sensory fibers to first web space
   (2) Superficial - to peroneus longus and brevis with sensory to anterolateral leg and dorsum of foot and toes

3. Deep branch runs beneath extensor retinaculum at ankle.

4. Accessory peroneal, branch of superficial peroneal, may innervate extensor digitorum brevis.

B. Clinical syndrome

1. Symptoms
   a) Pain and numness in distribution of the nerve
   b) Anterior compartment syndrome - deep peroneal nerve
   c) Anterior tarsal syndrome
   d) History of leg crossing, bed rest, trauma, recent surgery, "hunkering"

2. Signs
   a) Weak ankle dorsiflexion and/or eversion
   b) Sensory loss over dorsum of foot or web space

C. Electrodiagnosis

1. Peroneal motor amplitude - anterior tibial recording - most important (Wilbourn)
   a) Stimulate proximal and distal to fibular head
b) Three combinations of findings:

(1) Low amplitude responses at either point-axonal loss; most common presentation (60%).
(2) Low amplitude response at proximal stimulation only - demyelination.
(3) Very low amplitude response with proximal stimulation and low amplitude on distal stimulation - axonal loss and demyelination.

c) With extensor digitorum brevis recording, should show at least 20% drop in amplitude comparing proximal to distal recordings.

2. Comparison of conduction velocity across fibular head versus distal segment - 10 m/s or 20% slower across fibular head

   a) Cannot determine CV with complete conduction block
   b) May be normal if fastest fibers intact

3. Recordings from EDB

   a) EDB may be injured due to local trauma or peripheral neuropathy.
   b) EDB may have accessory innervation.
   c) Chief complaint is "foot drop" not "toe drop".

4. Superficial peroneal sensory recording - unilateral absence in 6-10% of normal limbs

   a) Decreased amplitude due to axonal loss
   b) Some authors assert that slowed conduction of sensory nerve across fibular head is most sensitive indicator of peroneal neuropathy at fibular head; distal superficial peroneal sensory recording may be normal.
   c) If normal with abnormal motor studies, suggests lesion of deep peroneal nerve alone or L5 radiculopathy.

5. Determine location of lesion with EMG of muscles innervated by both deep and superficial branches of peroneal nerve; study short
head of biceps femoris to determine if lesion is proximal to fibular head.

6. NCS and EMG of sciatic and tibial-innervated muscles to rule out generalized neuropathy, radiculopathy, plexopathy, sciatic nerve lesion.

V. ULNAR NEUROPATHY

A. Anatomy (Figures 2 and 3)

Figure 2. Locations for Ulnar neuropathy at the wrist or Guyon’s canal

Figure 3. Canal of Guyon.
1. Roots: C8-T1; lower trunk, medial cord

2. Passes with subclavian artery through triangle composed of anterior and posterior scalene muscles with first rib as base of triangle, then passes beneath clavicle.

3. Passes between biceps and triceps then shifts posteriorly in upper arm to become superficial behind medial epicondyle.

4. Enters forearm (figure 4) through flexor carpi ulnaris (cubital tunnel) branching to flexor carpi ulnaris (FCU), flexor digitorum profundus III and IV, runs along medial aspect of forearm lateral to flexor carpi ulnaris giving cutaneous branch to medial palm and dorsum of hand.

5. Enters wrist through Guyon's canal (figure 2), under pisohamate ligament and divides into deep and superficial branches. Superficial is pure sensory, and deep, which hooks around hook of hamate, is pure motor and innervates hand intrinsics, hypothenar, and opponens, adductor pollicis, and flexor pollicis brevis muscles.

B. Entrapment at the elbow - Tardy Ulnar Palsy and Cubital Tunnel Syndrome

1. Clinical syndrome
   a) Symptoms - elbow pain, numbness and weakness in ulnar distribution; pain radiating proximally or distally
   b) Signs - Tinel's at medial epicondyle; tenderness in groove; hand intrinsic weakness with sensory loss over medial aspect of hand and 4th and 5th digits

2. Electrophysiologic studies
   a) Ulnar sensory - mildly slowed conduction velocity; drop in amplitude or no response
   b) Ulnar motor - drop in amplitude of at least 25% across elbow; slowed conduction velocity across the elbow; only significant if difference exceeds 10 m/s comparing with more distal segment; distal segment may also show mild slowing; amplitude may be reduced if conduction block present; prolonged distal latency.
   c) Needle exam - may see sparing of FCU since branch to FCU is proximal to cubital tunnel, however FDP and hand muscles will be involved.
3. Treatment

a) Avoid compression through excessive flexion of elbows during sleep or leaning on elbows.

b) Surgical transposition - excellent results if only mild to moderate damage exists; usually have persistent symptoms of weakness if moderate to severe damage present; some prefer "simple" decompression - transposition may cause secondary compression; medial epicondylectomy.

C. Compression at Guyon's Canal

1. Clinical syndrome - similar to above but no sensory loss on dorsum of hand - only palm.

2. Electrophysiologic studies - denervation in hand intrinsics only; prolonged mid-palmar studies; reduced or absent sensory responses.

3. Usually associated with ganglion - resect ganglion.
D. Other ulnar entrapments

1. Palmer branch - deep branch, pure motor (see Fig 2 area 1); weakness without sensory symptoms; due to trauma, bicycling, crutch use; spares hypothenar muscles but can see prolonged latency if recording from FDIH and compare with opposite side

2. Digital nerve – due to trauma or inflammation with pain on hyperextension of digits and sensory loss in digits only

VI. RADIAL NERVE ENTRAPMENT

Anatomy (Figures 5-7)

Figure 5
1. Roots: C5-T1; posterior cord

2. Passes through axilla posterior to axillary artery and posterior cord sends branches to deltoit and teres minor as axillary nerve and continues as radial nerve to send branches to triceps and anconeus before reaching spiral groove of humerus.

3. Winds from medial to lateral and posterior to anterior in spiral groove through lateral intermuscular septum, then sends branches to brachioradialis and ECRL and enters forearm lateral to biceps between brachialis and brachioradialis at level of lateral epicondyle (fig 4)

4. At this point, divides into motor branch (fig 6) - posterior interosseous nerve and innervates supinator, AP, ECRB, ECU, EDC, EDM, EI, EPL, EPB.

5. Sensory branch (fig 6) - C6-C7 roots, supplies dorsum of hand laterally
B. Entrapment at the spiral groove - Saturday Night Palsy

1. Clinical syndrome
   a) Symptoms and signs - wrist and finger extensor weakness with variable sensory loss over dorsum of hand laterally and first two digits; triceps spared; occurs after falling asleep with arm over back of chair or other compression at spiral groove; frequently occurs with fractures of humerus.

2. Electrophysiologic studies
   a) Radial sensory - reduced or absent response
   b) Radial motor - reduced or absent response; may demonstrate slowing across spiral groove
   c) Needle exam - denervation with triceps spared; check triceps, brachioradialis, and wrist or finger extensor; note recruitment abnormalities and rule out root lesion - check PRT

3. Treatment - may resolve in 6-8 weeks depending on degree of axonal loss; may require surgical reanastomosis

C. Posterior Interosseous Syndrome

1. Clinical syndrome
   a) Weakness in wrist and finger extensors without sensory loss; ECRL and ECRB spared (fig 6) as well - gives characteristic radial deviation of wrist on extension
   b) Compression in four anatomic areas:
      (1) Fibrous bands anterior to radial head (fig 5); aggravated by full flexion of elbow with forearm supinated
      (2) Vessels, fan-shaped, crossing radial nerve, that supply brachioradialis and ECRL
      (3) Tendinous margin of the extensor carpi radialis brevis; aggravated by pronation
      (4) Arcade of Frohse; aggravated by pronation
c) Sunderland has shown 5 out of 6 branching patterns in which posterior interosseous branch innervates supinator after the bifurcation and may or may not be involved.

2. Electrophysiologic studies - may see reduced distal motor and sensory amplitudes and prolonged latencies; demonstrate sparing of triceps, brachioradialis, ECR, and supinator with involved ECU on needle exam.

3. Treatment - surgical exploration and decompression

D. Radial sensory nerve entrapment

1. Clinical syndrome – discomfort over dorsoradial aspect of hand; worsened by ulnar – volar flexion of wrist; false positive Finkelstein’s test; seen in occupations with repetitive supination/pronation of forearm – movement associated with brachioradialis and extensor carpi radialis tendons

2. Decreased radial sensory amplitude or prolonged latency

3. Diagnose using nerve block

4. Treatment – neurolysis

REFERENCES

Entrapment Neuropathy


Peroneal Nerve


**Ulnar Nerve**


**Radial Nerve**


I. THORACIC OUTLET SYNDROME

A. Anatomy

1. Lower trunk, medial cord of brachial plexus passes with subclavian artery through triangle composed of the anterior and posterior (middle) scalene muscles with the first rib as the "base" of the triangle, and then passes beneath clavicle (Figure 1).

![Figure 1](image-url)
B. Historical Aspects

1. Wilson (1913) - first described syndrome of vague upper extremity numbness, pain due to compression by cervical rib; probable carpal tunnel syndrome

2. 1930-1940s - widely diagnosed

3. 1950s - recognition of extreme frequency of carpal tunnel syndrome and cervical spondylosis and radiculopathy accounting for majority of patients with upper extremity sensory complaints

4. Diagnostic criteria have changed making literature review difficult.

C. Clinical Features

1. Neurogenic
   a) Paresthesias in ulnar and medial cutaneous nerve of forearm distribution, later accompanied by pain, poorly localized.
   b) Sensory symptoms worse with carrying suitcase, heavy object or when arms held overhead.
   c) Sense of weakness or clumsiness of hand; selective thenar atrophy

2. Vascular
   a) Signs of vascular occlusion – Adson maneuver, costoclavicular maneuver, hyperabduction, supraclavicular bruit – positive in many who do not have symptoms, so are probably not helpful
   b) Coldness, achiness, loss of strength on continued use; hand pale or cyanotic
   c) Rare to see vascular lesion alone

D. Electrodiagnosis

1. Nerve conduction studies
   a) Decreases ulnar sensory amplitude and mildly slowed conduction velocity
   b) May see reduced mean median motor amplitude more commonly than reduced ulnar motor amplitude
c) Slowed proximal ulnar nerve conduction - difficult to assess since actual location of lesion unclear and difficult to know where to measure distances; ideally, see slowing across Erb's point (if that is where the lesion is).

d) F-wave may have prolonged latency; however, lesion is typically across very small segment and will not significantly alter F-latency

e) Rule out ulnar or median or other mononeuropathy.

2. EMG - chronic denervation of lower trunk muscles with minimal spontaneous activity.

E. Differential Diagnosis

1. Intramedullary or extramedullary spinal cord processes
   a) Syringomyelia
   b) Glioma
   c) Foramen magnum meningioma
   d) Cervical spondylosis
   e) Extramedullary tumor

2. Radiculopathy or mononeuropathy

3. Vascular insufficiency
   a) Atherosclerosis
   b) Systemic lupus erythematosus
   c) Aortic dissection
   d) Granulomatous angiitis

4. Reflex sympathetic dystrophy

F. Treatment

1. Non-surgical - avoid activities and postures requiring abduction of arms or arms raised overhead; night splints; stretching of shoulder girdle and postural exercises to correct slumped posture.

2. Surgical
a) Indicated when sign of muscle wasting appears or pain incapacitating.

b) First rib resection, claviculectomy, anterior scalene section, supraclavicular exploration

II. DIGITAL NERVE ENTRAPMENT IN THE HAND

A. Clinical Features

1. Numbness in distribution of nerve - may mimic forearm mononeuropathy; may be painful.

2. Nerve tenderness, positive Tinel's, palpable nerve

B. Etiology

1. Acute trauma – fracture, dislocation

2. Chronic compression – tailors from scissors, harp and other stringed instrument players, staple gun users, excessive pressure from pencil writing

3. Bowler’s thumb – painful ulnar side of thumb with low evoked sensory response; also seen in tennis players, woodchoppers, baseball players

4. Cysts, osteophytes, tumors – Schwannomas

C. Electrodiagnosis

1. Rule out more proximal lesion
III. SIATIC NERVE ENTRAPMENT

A. Anatomy

1. Roots L4-S2; exits pelvis through greater sciatic foramen beneath piriformis muscle and over obturator internus (Figure 2)
B. Clinical Features

1. Weakness of knee flexion, ankle dorsiflexion or plantarflexion with numbness posterior leg, dorsum and sole of foot (see Fig 3); rare to see complete lesion

C. Etiology

1. Entrapment of piriformis muscle - usually peroneal fibers; other fibrous bands

2. Pentazocine injection
D. Electrodiagnosis

1. Nerve conduction studies may show reduced or absent sensory and motor responses, tibial and peroneal
2. Rarely slowed F-wave or H-reflex latencies
3. Localization with EMG of posterior thigh muscles
4. Rule out radiculopathy, plexopathy, mononeuropathy.

E. Treatment

1. Surgical release or stretching of piriformis
2. Avoid compression

IV. TARSAL TUNNEL SYNDROME

A. Anatomy (Figure 4)

![Figure 4]
B. Etiology

1. Ankle sprain or fracture
2. Tenosynovitis or rheumatoid arthritis
3. Hyperpronation of foot
4. Thrombophlebitis and resultant ankle swelling
5. Posterior tibial nerve passes behind the medial malleolus, gives off a branch to the calcaneus, and passes under the flexor retinaculum (tarsal tunnel) and branches into the medial and lateral plantar nerves. (see fig 4)

6. Medial plantar nerve innervates the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis, and gives sensation to the medial anterior sole and first three toes.

7. Lateral plantar branch innervates the abductor digiti minimi, flexor digiti minimi, abductor hallucis, interossei, and gives sensation to the lateral sole and 4th and 5th toes.

8. Tunnel contains posterior tibial nerve, artery, flexor digitorum longus and flexor hallucis longus tendons.

C. Clinical Features

1. Pain in sole of foot and like carpal tunnel syndrome, pain is worse at night and has a burning quality
2. Positive Tinel's behind medial malleolus
3. Sensory loss of sole of foot - usually excludes heel
D. Electrodiagnosis

1. Motor nerve conduction recording from medial (AHB) and lateral (ADQ) plantar innervated muscles across tarsal tunnel
2. Rarely bilateral, so side-to-side comparison used for diagnosis
3. Can also use ring electrodes to stimulate great or little toes and record behind medial malleolus.

V. FEMORAL AND LATERAL FERMORAL CUTANEOUS NERVE ENTRAPMENT

A. Anatomy

1. Lateral femoral cutaneous nerve is first sensory branch of lumbosacral plexus; roots L2-3; courses along psoas to brim of pelvis, through tunnel formed by lateral inguinal ligament and ASIS, and supplies sensation to anterior, lateral and posterior thigh (figure 5)

2. Femoral nerve - roots L2-4; passes along psoas and supplies psoas and iliacus; leaves pelvis passing under inguinal ligament (figure 5) lateral to femoral artery and vein; sensory (figure 6)- anterior thigh and medial calf; motor - innervates pectineus, sartorius, and quadriceps femoris (figure 7).

3. Saphenous nerve (figure 7) - branches from femoral nerve at femoral triangle and descends medially passing anterior to the medial malleolus; supplies sensation to medial thigh, leg, foot, with infrapatellar branch supplying medial aspect of knee.

B. Meralgia Paresthetics

1. Clinical syndrome

   a) Anatomy - compression of lateral femoral cutaneous nerve at the ASIS from corsets, seatbelts, tight belts

   b) Symptoms and signs - pain, paresthesias, with sensory loss over anterolateral thigh
2. Electrophysiologic studies - rule out high lumbar radiculopathy; needle exam normal; slowed conduction across ASIS and decreased or absent evoked sensory response; technically difficult

3. Treatment - conservative - usually resolves within 2 years; may surgically decompress through cutting portion of inguinal ligament but usually leaves persistent paresthesia.

C. Femoral Neuropathy

1. Clinical syndrome
   a) True entrapment rare - usually due to hematoma or tumor
   b) Usually see diabetic amyotrophy
   c) Weakness of hip flexion and knee extension with variable sensory loss along thigh

2. Electrophysiologic studies - prolonged femoral motor distal latency with reduced CMAP amplitude; rule out root lesions - screen adductors, quads, gluteus medius, anterior tibialis, and gastroc.

3. Treatment - if compression, surgically decompress

VI. MISCELLANEOUS MONONEUROPATHIES

A. Other Shoulder Girdle Nerve Entrapments

1. Long thoracic nerve - from heavy shoulder bag, trauma; scapular winging more pronounced with forward flexion of arm; serratus anterior weakness

2. Dorsal scapular nerve - abnormal EMG in rhomboids and levator scapulae

3. Suprascapular nerve - from heavy shoulder bag or stab wound; weak initiation of abduction and external rotation; posterior shoulder pain (sensory component); EMG abnormal in supra- and infraspinatus only with other C5-6 muscles spared.

4. Musculocutaneous nerve - fractures or dislocations of humerus, gunshot or stab wounds, numb along lateral aspect of forearm; absent biceps reflex with partial weakness of elbow flexion; prolonged distal latency and decreased or absent musculocutaneous CMAP with denervation in biceps, brachialis, coracobrachialis.
B. Other Pelvic Girdle Nerve Entrapments

1. Ilioinguinal nerve: L1-3 roots; winds around to medial ASIS; pain with standing referred inguinal area; associated with direct inguinal hernia.

2. Genitofemoral nerve: L1-2 roots; cremasteric muscle and skin of penis, labium, upper medial thigh; sensory deficits and absent cremasteric reflex; groin trauma or surgical adhesions.

3. Saphenous nerve – compressed on exit from Hunter’s subsartorial canal with femoral vessels; usually pain at medial aspect of knee and medial foot and worsens with exercise, particularly climbing stairs.

4. Obturator nerve: roots L2-4; compression due to pregnancy and during prolonged labor; weakness of adduction and internal and external rotation of thigh; pain and dysesthesia radiating to medial thigh common.

5. Superior and inferior gluteal nerves - pass directly behind hip joint; superior L4-S1, gluteus medius and minimus; inferior - L15-S2, gluteus maximus; pyriforms muscle may compress superior gluteal with pain in sciatic notch.

REFERENCES

5. Refer to Compressive Neuropathy I reference list.