Compressive Neuropathies
(s. Entrapment Neuropathies, Tunnel Syndromes)

Last updated: April 17, 2019

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Other Nerves


*Pressure-induced injury to segment of peripheral nerve secondary to anatomic / pathologic structures

- account for 10-20% of all neurosurgery cases!

Most frequent:
1. Carpal tunnel syndrome
2. Ulnar nerve compression at elbow

Etiology

1. Violent muscular activity, forcible joint overextension, prolonged cramped postures (e.g. in gardening).
2. Repeated small traumas (e.g. tight gripping of small tools, excessive vibration from air hammers).
3. Extrinsic compressions - casts, crutches.
4. Intrinsinc compressions - tumors, bony hyperostosis, inflammatory edema of adjacent structures, infiltrating substances (e.g. amyloid, lymphoidomas, mucopolysaccharidosis, acromegaly).

- patients with any polyneuropathy are more vulnerable to mechanical injury of nerves!!!
- patients with congenital narrowing of osseous tunnel or thickening of overlying retinaculum have predilection.

Pathophysiology

- usually affects:
  a) superficial nerves (ulnar, radial, peroneal) at bony prominences (e.g. during sleep or anesthesias) in thin-catchic persons (esp. alcoholics).
  b) nerves at narrow osseodermal tunnels (e.g. carpal tunnel).
- in almost all cases, at least one side of compressive surface is mobile – allows chronic injury: either repetitive “slipping” in a tunnel or “rubbing/slidig” against sharp, tight edges with motion at adjacent joint – this explains beneficial effect of splinting.
- chronic blunt injury / pressure (above perfusion pressure) to nerve → disruption of blood-nerve barrier → microvascular (ischemic) changes, edema → dislocation of nodes of Ranvier → local segmental demyelination (still reversible with treatment) → axonal disruption, epineural fibrosis (constant feature)?

<table>
<thead>
<tr>
<th>Pressure</th>
<th>Result</th>
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<tbody>
<tr>
<td>30 mm Hg</td>
<td>impaired axonal transport</td>
</tr>
<tr>
<td>40 mm Hg</td>
<td>paresthesias and neurophysiologic changes</td>
</tr>
<tr>
<td>50 mm Hg</td>
<td>axonal block</td>
</tr>
<tr>
<td>60 mm Hg</td>
<td>complete microvascular ischemia (sensory and motor block)</td>
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</tbody>
</table>

- recovery:
  a) complete - reflects remyelination.
  b) incomplete - due to Wallerian degeneration and permanent fibrotic changes.

- nerve compression affects myelinated fibers first (A type > B type > C type) - nerve conduction studies & EMG are usually diagnostic.

N.B. larger fibers are more susceptible than small fibers

DOUBLE CRUSH SYNDROME
COMPRESSIVE NEUROPATHIES

(2)

- coexistence of compressive lesions in series along course of peripheral nerve, with one lesion rendering nerve susceptible to distal or proximal compression.
- mechanism: impaired axonal flow, ischemia, and altered nerve elasticity, which lessen the nerve's resiliency. Intrinsic neuropathies additionally affect nerve's susceptibility to injury.
- common examples: cervical radiculopathy and CTS, thoracic outlet syndrome and CTS, and cubital tunnel syndrome and Guyon's canal syndrome.

CLINICAL FEATURES
- temporal sequence of neurological manifestations: irritative sensory symptoms (pain, paresthesia) → ablative sensory symptom (numbness) → ablative motor signs (weakness and atrophy).
- sensory loss is less extensive than anatomic distribution of nerve.
- in major mixed nerve (e.g. sciatic, median) sympathetic dystrophy may be prominent.
- palpate entire length of affected nerve to check for masses, points of tenderness, adjacent bony abnormalities.
- N.B. referred pain with entrapment neuropathy can radiate proximally to the site of entrapment (mimics radiculopathy)!!!

DIAGNOSIS
- Diagnosis of most entrapment neuropathies is clinical!
- MRI using short inversion recovery technique (STIR) - high signal intensity in affected nerve segment at site of compression (due to presence of edema in myelin sheath and perineurium).
- MR neurography - only large nerves (ulnar, median, sciatic) are reliably identifiable.
- demonstrates nerve position in relation to adjacent joint placed in varying degrees of flexion - may suggest adhesion of nerve to surrounding tissue.
- nerve conduction abnormalities across entrapment tunnel.
- EMG - signs of denervation.

TREATMENT
- Conservative therapy should be tried first.
  - mainly consists of educating patient to adopt avoidance behaviors.
  - various splints.
- Surgical decompressions, s. external neurolysis (incl. endoscopic techniques).
- low risk for serious morbidity and high success rates.

N. MEDIANUS
full anatomy of median nerve → see p. A20

PLACES OF COMPRESSION
1. Near elbow (proximal median neuropathy):
   1) ligament of Struthers / supracondylar process of humerus
   2) lacertus fibrosus (bicapital aponeurosis)
   3) between two heads of hypertrophied pronator teres
   4) flexor digitorum profundus fascial arch (sublimis bridge)

2. Within carpal canal (carpal tunnel syndrome); anatomy → see below

Causalgia is most commonly associated with lesions of median nerve!
CARPAL TUNNEL SYNDROME (CTS)

- most common tunnel syndrome

- PREVALENCE: 3% in women and 2% in men

- PEAK PREVALENCE - women > 55 years

- frequently bilateral, dominant side being affected more severely.

PRECIPITATING FACTORS

1) overuse - repetitive motion of fingers (frequent prolonged wrist flexion, especially with force) - often occupational; prevention - ergonomic redesign of work stations and tools.

2) pregnancy (especially fluid retention in 3rd trimester; resolves spontaneously after birth’) = 1%

3) nonspecific tenosynovitis (found in up to 75% cases); rheumatoid arthritis (synovial hypertrophy), osteoarthrosis, gout

N.B. arthritis per se may cause thenar pain but no numbness (numbness is a must for CTS)

4) trauma: wrist fractures, lunate dislocation

5) ganglionic cysts

6) nerve sheath tumor

7) hypothyroidism, mucopolysaccharidosis, acromegaly, sarcoidosis

8) diabetes mellitus (microvascular injury)

9) amyloidosis (e.g. hemodialysis - deposition of β-microglobulin derived amyloid)

10) anatomic predispositions: persistent median artery, anomalous tendons or muscles, congenital stenosis of carpal tunnel

CLINICAL FEATURES

Referred pain with entrapment neuropathy can radiate proximally to the site of entrapment; carpal tunnel syndrome may cause referred pain to the arm and even to the neck (mimics C6-7 radiculopathy)

Mild disease: paresthesias & pain in median nerve distribution (after strenuous wrist movements or nocturnal) *because of venous stagnation (Sunderland hypothesis: pain is characteristically relieved by hand shaking or elevating) related to hypotonia during sleep or because wrist falls into flexion with sleep

- pain is burning and may be severe (awakening from sleep).

- sometimes pain radiates proximally to forearm and shoulder.

- grasping objects is painful and patients may report dropping cups and glasses

- sensation in thenar eminence is not affected (palmar cutaneous nerve emerges from median nerve before carpal tunnel).
**Compressive Neuropathies**

More severe disease: sensory loss & weakness (with thenar atrophy). *May be absent in patients with Riche-Cannieu anastomosis, see p. A20 (12).*

N.B. most reliably affected muscle is abductor pollicis brevis – ability of thumb to move toward little finger against resistance.

### Proximal Median Neuropathy
- **Deficit**: More widespread, tenderness along nerve course.

<table>
<thead>
<tr>
<th>Location</th>
<th>Muscles Affected</th>
<th>Action</th>
<th>Sensory Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>At wrist</td>
<td>Abductor pollicis</td>
<td>Abduction</td>
<td>Palm and dorsal surfaces of thumb, index, middle fingers</td>
</tr>
<tr>
<td></td>
<td>Opponens pollicis</td>
<td>Opposition</td>
<td>None</td>
</tr>
<tr>
<td>Near elbow (pronator syndrome)</td>
<td>Abductor pollicis</td>
<td>Abduction</td>
<td>Palm, palma and dorsal surfaces of thumb, index, middle fingers (less loss on forearm)</td>
</tr>
<tr>
<td></td>
<td>Opponens pollicis</td>
<td>Opposition</td>
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</tr>
<tr>
<td></td>
<td>Pronator quadratus</td>
<td>Pronation</td>
<td></td>
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<tr>
<td></td>
<td>Pronator tereus</td>
<td>Pronation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flexor pollicis longus</td>
<td>Flex thumb, distal joints</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flexor digitorum sublimis</td>
<td>Flex fingers</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flexor digitorum profundus</td>
<td>Flex fingers, median side</td>
<td></td>
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<tr>
<td></td>
<td>Flexor carpi radialis</td>
<td>Wrist flexion</td>
<td></td>
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<tr>
<td></td>
<td>Lumbricales (two radial)</td>
<td>Extensor MP joints</td>
<td></td>
</tr>
<tr>
<td>Below elbow (anterior interosseous branch)</td>
<td>Flexor pollicis longus</td>
<td>Flex thumb, distal joints</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flexor digitorum profundus II</td>
<td>Flex index finger, distal joint</td>
<td>None</td>
</tr>
</tbody>
</table>

### Severity assessment
- **Carpal Tunnel Syndrome Assessment Questionnaire (CTSAQ)** - 9-item functional status scale.
DIAGNOSIS

1) **Tinel sign** – tapping carpal tunnel (esp. with reflex hammer, wrist extended) elicits paresthesias – only ≈ 50%

2) **Phalen sign** (hold forcibly patient’s wrist in acute flexion for 60 seconds → paresthesias):

3) **Durkan compression test** - performed by examiner placing thumb over carpal tunnel and exerting downward pressure for 30 seconds - best sensitivity (82-89%) and specificity (90-99%)

4) **other provocative maneuvers** – reverse Phalen test, Gilliat (tourniquet) test, ultrasonic stimulation test

5) **EMG** (abductor pollicis brevis or opponens pollicis) - spontaneous fibrillation potentials and positive sharp waves, increased terminal latencies (norma - 3.5 ms) or significant asymmetry (but opposite side may be affected subclinically – compare also with ipsilateral ulnar and radial nerves). Sensory nerve conduction slowing across carpal tunnel (focal demyelination).

6) **sensory nerve conduction slowing** across carpal tunnel (local **denervation**)

7) **Electrodiagnostic studies** are also helpful in grading severity of CTS:

   - **mild CTS** – SNAP or mixed nerve action potential (NAP) is often prolonged, and SNAP amplitude may be below the lower limit of normal: moderate CTS - there are findings of mild CTS plus prolongation of median motor distal latency. severe CTS - median motor and sensory distal latencies are prolonged, with absent SNAPs or mixed NAPs or absent or reduced thenar compound motor action potentials, or both. Fibrillations, reduced recruitment, and changes in motor unit potential are often seen in severe cases.

8) **ultrasonography** (highly sensitive and specific even in patients with negative electrodiagnostic studies) - entrapped peripheral nerve may appear hypoechoic, swollen, or flattened.

9) **thyroid testing** (TSH)

TREATMENT

CTS is usually progressive condition, but course of conservative therapy should be completed before surgical intervention:

1. **Splinting** of wrist in neutral / slight dorsiflexion (cross-sectional area↑ of carpal tunnel) - splint should be worn at night and if needed during day for weeks:

   - hand–wrist exercises and ultrasound do not provide additional benefit beyond that offered by splinting alone.

2. **Ergonomic corrections**

3. **Ultrasonic therapy**

4. **NSAIDs** - short course (7-10 days)

5. **Potassium-sparing diuretics**

6. Injection of depot corticosteroids into carpal tunnel (medial to m. palmaris longus tendon, just proximal to distal wrist crease) - significant, but temporary improvement:

   - 1 cc of local anesthetic and 1 cc of long-acting corticosteroid.
- 3-cc syringe with 25-G needle.
- Flex wrist and identify wrist flexion crease and palmaris longus tendon - needle will be inserted on ulnar side of palmaris longus about 1 cm proximal to wrist crease.
- Ask patient to fully flex fingers; advance needle at 45° angle for ≈ 1 cm until you feel resistance.

- Appropriate needle location can be assessed by moving ring finger (this should produce movement of needle); ask patient to now extend fingers to bring needle into carpal tunnel and slowly inject 1-2 cc of steroid–anesthetic solution.
- Advice patient that there will be some mild soreness and it may require 24 h to feel full effect.

N.B. aim to inject tendon sheaths; injection adjacent or into nerve is to be avoided!

7. Exercises, vit. B6 – ineffective!

**SURGERY**

- See p. 650 >>

**N. ULNARIS AT ELBOW**

**Pla**ces of compression

1. **A**rcade of **S**TRUTHERS (diastasis in medial intermuscular septum; tense sheet of fascia stretching from medial head of triceps to insert into medial intermuscular septum) 6-8 cm above cubital tunnel.

2. **C**UBITAL TUNNEL SYNDROME at elbow groove (e.g. cubitus valgus, medial condyle fracture, RA synovitis, osteophytes) - compression between cubital tunnel retinaculum (OSBORNE'S ligament) and medial collateral ligament (MCL).

3. **E**xternal compression at elbow groove; e.g. during anesthesia (most common anesthesia-related compressive neuropathy?!); prolonged resting of elbow on hard surface.

4. Between two heads of flexor carpi ulnaris (aponeurosis of flexor carpi ulnaris also referred to as OSBORNE'S fascia; 3.5-5 cm distal to cubital tunnel) – e.g. in pianists (repeated forceful wrist flexion).

5. **M**edial intermuscular septum - sharp edge that can indent nerve (esp. after anterior transposition where nerve may be kinked).
COMPRESSIVE NEUROPATHIES

N.B. elbow flexion narrows cubital tunnel (flexion can cause anterior subluxation of nerve).
Spontaneous ulnar nerve subluxation out of cubital tunnel occurs in 15% population - rubbing action by bony surfaces aggravates entrapment.
- asymptomatic (or minimally symptomatic) ulnar neuropathy is very common, approaching incidence of carpal tunnel syndrome.
- musicians who use one arm in flexed position (cellists, violinists) commonly develop ulnar neuropathy.

CLINICAL FEATURES

1) paresthesias, pain, sensory loss; exacerbating activities include:
   - cell phone use (excessive flexion)
   - sleeping with elbow in flexion → nocturnal paresthesia and pain.
N.B. sensory testing of dorsal medial hand portion is important – preserved sensation in this area with sensory deficits in ulnar distribution of fingers suggest entrapment at Guyon's canal (spared dorsal cutaneous branch distribution).
Referred pain with entrapment neuropathy can radiate proximally to the site of entrapment (mimics C8 radiculopathy)

2) "CLAWHAND": hand clumsiness, dropping objects; hypothenar + interossei weakness and atrophy.
see p. D1 >>
- fifth finger may be abducted away from other fingers at rest (Wartenberg sign); patients complain of catching fifth finger when placing hand in pocket

- weakness may occur quickly and may precede sensory disturbances because of predominance of motor fibers within UN
- course can be prolonged – e.g. due to asymmetric bone growth after childhood fracture (tardy ulnar palsy).
- old, "burnt out" neuropathic hand is atrophic, thin-skinned but, surprisingly, painless and free of other sensory phenomena.

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<td>Flexor digitorum profundus</td>
<td>Flexes little finger, distal joint crease</td>
<td>Medial side of hand and fingers to wrist crease</td>
</tr>
<tr>
<td>Interossei</td>
<td>Adducts and abducts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor pollicis brevis</td>
<td>Adducts thumbs</td>
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DIAGNOSIS

1. Nerve percussion (Tinel sign) → paresthesias
2. Elbow flexion test - positive when flexion elbow for > 60 seconds → paresthesias
3. Elbow pressure-flexion test (sensitivity 91%) - elbow is flexed and pressure applied over cubital tunnel for 30 seconds → paresthesias
4. Nerve conduction studies (motor conduction < 50 m/sec across elbow suggest entrapment)
5. EMG - signs of denervation
6. Plain radiographs of elbow - search for fracture / deformity when there is history of trauma.
7. MRI - increased T2 nerve signal; nerve subluxation / dislocation can be seen on axial images acquired during elbow flexion

TREATMENT

1. Half-split with elbow pad (elbow in gentle extension) at nighttime ± daytime.
2. NSAIDs
   - N.B. steroid injections have no role in treatment!

N. ULNARIS AT WRIST

- compression at ulnar Guyon canal:
1) paraplegics using hand crutches with horizontal bar across palm.
2) motorcyclists who firmly grasp hand bar control.
- operators of pneumatic drills.
- compression within proximal Guyon canal often is attributed to thickening of tendinous arch stretched between pisiform and hamate; hook of hamate may be sharp-edged and forms acute angle where nerve turns radially.
- compression within distal Guyon canal may be accentuated by fibrous bands; distal canal also is common site for ganglions.

Short anatomy: also see p. A20 (10)
- ulnar nerve runs above flexor retinaculum (lateral to flexor carpi ulnaris tendon and medial to a. ulnaris).

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Short anatomy: also see p. A20 (10)
- ulnar nerve runs above flexor retinaculum (lateral to flexor carpi ulnaris tendon and medial to a. ulnaris).
at proximal carpal bones, it dips between pisiform and hamate at entrance to Guyon canal, roofed over by extension of transverse carpal ligament between these 2 bones.
- superficial hypotthenar sensory branch (hypotenar skin ulnar to vertical line at base of ring finger and ends as 2 ulnar digital nerves for little finger and ulnar half of ring finger) comes out just outside Guyon canal in 65% population, compression at Guyon canal spares sensory branch; damage to deep palmar motor branch
- weakness of small hand muscles but no sensory loss (i.e. painless hypotenar atrophy).
- in other 35% individuals, some pain and hypotenar numbness is expected.
- after entering Guyon canal, deep motor branch first supplies abductor digiti minimi (ADM), then crosses over to supply opponens pollicis before rounding hook of hamate to enter mid palmar space depending on exact site of compression, ADM or both ADM and FDM may be spared; opponens always is affected, together with intrinsic, ulnar micrhalicades, and adductor pollicis.

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<td>Opponens V</td>
</tr>
<tr>
<td>Palm and radial hand and finger</td>
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### TREATMENT

- avoidance & use of palmar paddling.
- surgery — see p. Op450 ->

**N. RADIALIS**

Places of compression.

1. *Distal brachial plexus* - when patient falls asleep with arm draped over chair - nerve is acutely compressed against humerus - *SATURDAY NIGHT PALPES*.

2. *HEMORRHUS SHAFT FRACTURES* (spiral groove)
   - between medial and lateral heads of triceps.

3. Underneath arch of *FROHSE* (musculotendinous arcade, formed by upper free border of superficial head of m. supinator) ⇒ radial tunnel (p. m. extensor carpi radialis, 3-4 cm distal to lateral epicondyle); within tunnel, nerve rests on deep head of m. supinator - *RADIAL TUNNEL SYNDROME: no sensory loss!*
   - lesion of *arterial branches* (of Henry) arising from radial recurrent artery cross over nerve just before arcade of Frohse.

4. Wrist (sensory superficial radial branch).

Causes of *RADIAL TUNNEL SYNDROME:*

- a) tendinous hypertrophy of arcade of Frohse and fibrous thickening of radiocarpal joint capsule.
- b) Monteggia fracture-dislocation.
- c) vascular compression by hypertrophic leash of Henry.
- d) synovial cyst, rheumatoid synovitis.
- e) repetitive and forceful supination.
- f) chronic trauma to flexion surface of forearm (e.g. constricting rings of Canadian crutches in paraplegics).

### CLINICAL FEATURES

1. Motor — see p. DH 11>
   1) Wrist drop with paralysis of *finger extension* at MCP joints (IP joints extension — action of mm. lumbricales).
   - 2nd and 5th fingers receive both their own extensor tendons and tendon branch from common extensor - they are less affected — in early entrapment, characteristic finger posture - middle 2 fingers fail to extend, while index and little fingers hold erect!
   - since radial wrist extensors are spared (because of their proximal innervation), wrist extension weakness usually is undetectable in spite of ulnar wrist extensor weakness.

2) pseudo-weakening of finger abduction - intrinsic hand muscles are weak in semiflexed finger position; this can be corrected by supporting fingers.

2. Sensory: pain (exacerbated by wrist extension).

### DIAGNOSIS

1) **TENZ sign at radial tunnel.**

2) nerve conduction studies — conduction block (locating exact site of compression).

3) **EMG**

### TREATMENT

- **spring-loaded brace** for finger and wrist extension.
- **acute radial palsy patients usually recover completely within 4-6 weeks**; even after severe injury full late recovery can occur.

- no improvement within 3 to 4 months following humeral fracture ⇒ *surgical exploration*.

**SURGICAL EXPLORATION** – for *RADIAL TUNNEL SYNDROME: excellent outcome in 90-95% cases*

- **RADIAL TUNNEL SYNDROME** is motor neuropathy — diagnosis mandates surgical decompression; conservative treatment has no place!
- **incision** - lateral side of biceps muscle; extended across elbow and along border of brachioradialis.
- **radial nerve is picked up within groove made by biceps and forearm extensor group (groove is held open by self-retaining retractors).**

radial nerve is traced toward upper border of supinator; bifurcation into PIN and SRN are readily seen just above and in front of radiocapitellar joint (SRN courses deep to brachioradialis and may be picked up first, in which case it is traced backward to locate much deeper PIN).

- arcade is divided, together with fibers of superficial supinator muscle, to expose entire length of PIN within radial tunnel.
- fascial thickening associated with joint capsule also is divided, as is arterial leash of Henry.

**THORACIC OUTLET SYNDROME (TOS)**

- compression of brachial plexus of subclavian vessels in their passage from cervical and upper thoracic area toward axilla and proximal arm.

**CLASSIFICATION & CAUSES**

**VASCULAR TOS**

- affect subclavian artery or vein --> neurological symptoms by ischemia of nerves / muscles.

N.B. brachial plexus is not directly affected!
Neurogenic and vascular TOS do **not** coexist!

- < 1% of all TOS cases

**NEUROGENIC TOS**

1. **TINEL (CLASSIC) NEUROGENIC TOS** - caused by structural anomalies: congenital aberrant band between prominent C7 transverse process (or rudimentary cervical rib) and 1st rib (behind tubercle of scalene ant.)

Syndrome is very rare!

- compresses / irritates lower trunk of brachial plexus (C8-T1).

2. **SYMPTOMATIC (COMMON, SECONDARY, DISPUTED) NEUROGENIC TOS** - no identifiable anatomical structure causing nerve compression! (“wastebasket” diagnostic group that includes chronic pain syndromes of multiple causes)

Precipitating factors:

1) **scalene muscle spasm** (scalene anticus syndrome) – due to minor cervical or shoulder trauma.
2) **abnormal shoulder posture:**
   a) “droopy shoulder syndrome” - tall, slender, and round-shouldered person.
   b) occupational arms above head.

Three sites within thoracic outlet where neurovascular compression may occur:

1. **INTERSCALENE TRIANGLE** (anterior scalene muscle anteriorly, middle scalene muscle posteriorly, and medial surface of first rib inferiorly) contains trunks of brachial plexus and subclavian artery (subclavian vein runs anterior to anterior scalene muscle) - vast majority of neurogenic TOS cases!!

2. **COSTOTRUNCAL (spinal) SPACE** (midspace third of clavicle anteriorly, first rib posteroinferiorly, upper border of clavicle posterolaterally) - immediately distal to interscalene triangle.

- arm hyperabduction and external rotation produces compression of neurovascular elements within costotransverse space.

3. **SUBPECTORAL TUNNEL, S. SUBCOSTAL SPACE, S. RETROPECTORAL MUSCLE** (deep to the pectoralis minor tendon) - distal to costotransverse space.

- arm elevation compressed neurovascular elements within subcostal space.


**CLINICAL FEATURES**

**NEUROGENIC TOS**

- wide variety of clinical manifestations; two extremes:
  a) **painless form** - neurological and electrodiagnostic findings are quite dramatic
  b) **chronic pain syndrome** - few, if any neurological and electrophysiologic abnormalities.

**TRUE (CLASSIC) NEUROGENIC TOS** - stereotyped clinical picture in C8-T1 distribution:

N.B. **motor findings** include both median and ulnar nerve distributions whereas **sensory findings** are confined to ulnar nerve distribution!

- typical patients:
  a) young, thin female with long neck and drooping shoulders
  b) athlete with overdeveloped scalene musculature

1) weakness of all intrinsic hand muscles (C8–T1 myotomes) → muscle atrophy
   a) **classic GILLIATT-SUMNER hand** - dramatic atrophy in abductor pollicis brevis and lesser index muscle.
   b) **atrophy in interosseus muscles** (lateral aspect of neck, shoulder, axilla, parascapular region, and ulnar side of hand and forearm) / muscles
   c) **pain is aggravated by pulling arm down or repetitive overhead arm use; arm “fatigue” is*** often prominent.

3) **sustained motor disturbances** (changes in skin color and temperature) - in advanced cases related to compression of sympathetic fibers

Various provocative maneuvers have high false positive rate - no diagnostic value!

- **two best tests** (best predictive value):
  1. 90-degree shoulder abduction and external rotation
  2. Tinel sign over supraclavicular brachial plexus

**COMPREHENSIVE NEUROPATHIES**

PN5 (9)
other classic provocative maneuvers (sensitivity 72% and specificity 53%; false-positives 45.77%):
1. Rosom test (elevated arm stress test to induce reproduction of neurological symptoms)
2. Adson test (full neck extension and head rotation toward the side being examined; with deep inspiration → diminution (or total loss) of radial pulse on the affected side)
3. Wriscott test (progressive shoulder abduction to reproduce symptoms)

**SYMPTOMATIC (SECONDARY) NEUROGENIC TOS** - chronic pain / positional numbness that may or may not follow dermatomal pattern.
- no neurological deficit! (but due to pain patient may demonstrate give-way type of weakness)
- radial pulse may diminish with arm abduction (it is present in 15% of normals!)

**VASCULAR TOS** – ischemic symptoms in young adults with history of vigorous arm activity:
1) ischemic muscular pain - cold, pale, diffusely painful arm that is easily fatigued with activity.
2) distal pulse↓ (pulse may even disappear on arm elevation and turning head toward affected side; see Adson test above).
- some develop aneurysm (supraclavicular mass or bruit) distal to constriction
- some develop subclavian vein thrombosis (Paget-Von Schrötter syndrome) distal to constriction.
- some develop gangrene of digits may occur.

Subclavian vein occlusion in venous thoracic outlet syndrome - upper extremity edema (A) and superficial venous collaterals over proximal part of arm and shoulder (B).

**DIAGNOSIS**

**NEUROGENIC TOS**
In TRUE (CLASSIC) NEUROGENIC TOS injury is axonal:
1) nerve conduction studies:
   - ulnar sensory action potentials↓ but normal in median nerve
   - median motor conduction velocity↓ but normal in ulnar nerve
2) EMG findings in C8-T1 myotomes (reduced compound motor action potentials over thenar muscles, whereas normal over hypothenar muscles)
- MRI (cervical spine, brachial plexus, MR neurography) can demonstrate compression site and cause.

Cervical ribs bilaterally (larger on right)

Differential Diagnoses for Neurogenic TOS

**Spinal**
- Cervical disk disease or foraminal stenosis
- Cervical spinal cord tumor
- Cervical syrinx

**Peripheral nerve**
- Brachial plexitis
- Median nerve entrapment neuropathy
- Ulnar nerve entrapment neuropathy
- Nerve sheath tumor

**Orthopedic**
- Shoulder abnormalities (rotator cuff injury)

**Other**
- Complex regional pain syndrome
- Fibromyalgia
- Apical lung lesion (Pancoast's tumor)

**SYMPTOMATIC (SECONDARY) NEUROGENIC TOS** - electrophysiologic studies are usually normal.
TREATMENT

NEUROGENIC TOS

Most patients deserve trial of conservative therapy:
1. Lifestyle modification - avoidance of activities that provoke symptoms (overhead activities, arm hyperabduction, carrying of heavy bags over shoulder, sleeping in positions with arms overhead).
2. Physical therapy directed at strength of shoulder girdle (PEET's exercises) and scalene musculature, plus, focused toward correct posture and improving cervical and scapular mobility.

SYMPTOMATIC (SECONDARY) NEUROGENIC TOS – maximal conservative therapy for at least 3-6 months is mainstay (no risk involved - syndrome does not transform into or progress to true neurogenic TOS)
- scalene muscle denervation (injection of botulinum toxin) has been reported to result in improved pain
- surgery is often offered only as a last resort (patients who respond to scalene muscle blocks are more likely to respond to surgery) - significant chance that the patient will not improve!!!

TRUE (CLASSIC) NEUROGENIC TOS - surgical release (transsection of aberrant bundle, removal of cervical rib, scalenectomy at insertion):
- until the 1930s, first rib resection was mainstay of treatment
  a) anterior supraclavicular approach
  b) Roos's transaxillary approach (with first rib removal) - has many complications (neurovascular injuries).
  c) posterior subscapular approach

- 15-20% of patients experience recurrence of symptoms after either transaxillary rib resection or scalenectomy; recurrence rate is lowered to 5-10% when a combination of transaxillary rib resection and supraclavicular scalenectomy is used as primary surgery.

Anterior Supraclavicular Approach

- favored by most neurosurgeons, who frequently use this exposure to treat traumatic or neoplastic lesions of the brachial plexus. This approach allows wide exposure of the supraclavicular plexus and the middle two thirds of the first rib, where most potential anomalous fibrous bands are attached [21,45]. The incision is either transverse within a skin crease (our preference for cosmesis) or L shaped and centered on the posterior cervical triangle.

Supraclavicular approach for the treatment of neurogenic thoracic outlet syndrome. A, Proposed skin incision along an anterior skin crease. B, Reflection of the supraclavicular fat pad (FP) superolaterally and exposure of the phrenic nerve (PN) overlying the anterior scalene muscle (AS). The transverse cervical vessels were ligated with 3-0 silk tie and divided. C, After division of the anterior scalene muscle, the upper trunk (UT), middle (MT), and lower (LT) trunks of the brachial plexus and the subclavian artery (SA) are identified. The phrenic nerve (PN) is gently retracted medially.

- usually easy to detect on clinical examination or vascular imaging modalities.
During exposure, important anatomical landmarks to identify are the posterior border of the sternocleidomastoid muscle, the omohyoid muscle, the supravacular fat pad, the transverse cervical artery and vein, the phrenic nerve, and the anterior scalene muscle. Our preferred technique is to make a 6- to 8-cm transverse incision approximately one to two fingerbreadths above the clavicle, preferably along a preexisting skin crease. The medial extent of the incision is the midpoint of the sternocleidomastoid. Sharp dissection down to the platysma muscle is performed. We attempt to preserve sural cutaneous nerves to avoid a painful neurora. The platysma muscle is opened parallel to the incision, with the intent of reapproximating its edges on closure. Next, the omohyoid is identified running transversely across the exposure and is retracted laterally (it may be divided with impunity, but this is not usually necessary; it may serve as a guide to the supravacular nerve more distally). The supravacular fat pad is then identified and reflected carefully in an inferomedial-to-superolateral direction. Frequently, sizable lymphatic channels are encountered within the fat pad, and they must either be preserved or, more likely, dissected with bipolar electrocautery. The transverse cervical vessels are deep to or within the fat pad, and they are usually ligated and divided. The phrenic nerve has a unique course; it runs superolaterally to inferomedially on the anterior surface of the anterior scalene muscle, beneath its investing fascia. The identity of the phrenic nerve is confirmed by stimulating it and feeling contraction of the ipsilateral hemidiaphragm. The nerve is then gently mobilized and a vessel loop is placed. The medial and lateral margins of the anterior scalene muscle are identified and bluntly dissected. Once the anterior scalene is isolated, the muscle is transected. Numbness over the supraclavicular region, lasting approximately 6 weeks, may occur as a result of injury to the supraclavicular nerve. This procedure can be performed with minimal morbidity by surgeons experienced in this approach.

The lower trunk in particular is dissected proximally until the C8 and T1 spinal nerves are identified. Significant traction must be applied to the trunks to safely resect the first rib, and thus we rarely do this. Intraoperative EMG is used to confirm the identities of the neural elements, and nerve action potentials may also be recorded to assess damaged nerve segments. Before closure, the wound cavity is filled with saline and a Valsalva maneuver is performed to check for a pleural leak. A chest radiograph is always obtained postoperatively to check for pneumothorax, hemothorax, or hemidiaphragm elevation. Occasionally, the supraclavicular membrane (Sibson's fascia) is prominent and may need to be divided. The lower trunk in particular is dissected proximally until the C8 and T1 spinal nerves are identified. The first rib can be identified and resected as well, although we generally find that the soft tissue elements are much more likely to contact the plexus. Significant retraction must be applied to the trunks to safely resect the first rib, and thus we rarely do this. Intraoperative EMG is used to confirm the identities of the neural elements, and nerve action potentials may also be recorded to assess damaged nerve segments. Before closure, the wound cavity is filled with saline and a Valsalva maneuver is performed to check for a pleural leak. A chest radiograph is always obtained postoperatively to check for pneumothorax, hemothorax, or hemidiaphragm elevation.

This procedure can be performed with minimal morbidity by surgeons experienced in this approach. Numberness over the supravacular region, lasting approximately 6 weeks, may occur as a result of manipulation of or injury to the supravacular nerve during the approach; in certain circumstances, painful neuritis or neuropathic pain, or both, may form at the site of the nerve injury. Major complications from this approach include pneumothorax (1% to 2%), phrenic nerve injury (3% to 6%), and chylothorax (1% to 2%). Vascular injury occurs in approximately 1% to 2% of patients in whom the first rib is removed via the supravacular approach. Transient paresthesias or weakness in the arm or hand is seen occasionally and generally resolves within days to a few weeks.
Sciatica

- there is no consistent area in lower extremity where entrapment occurs!
  - pain / weakness
  - diagnostic provocative maneuvers
  - treatment

Etiology

- myofascial band
  - traumatic (fractures of hip, surgical trauma from hip replacement).
  - degenerative (e.g., in distal portion of thigh (between biceps femoris and abductor magnus)
- compressive
  - retroperitoneal bleeding
  - failure of pain control / severe weakness (bupivacaine and dexamethasone).
- neuropathies
  - chronic compression at suprascapular notch of scapula (not in posterior scapular notch)

Treatment

- surgical decompression
- PMR injection of nerve root

Transaxillary Approach

The transaxillary approach with resection of the first rib was popularized by Ross in 1966 and is still commonly used by many thoracic and vascular surgeons. The patient is placed in the posterolateral position with the arm elevated above the head. An incision is made over the first palpable rib (usually the third rib) in the axillary fossa. The axillary fat, lymph nodes, and vessels are dissected away, and the posterior and middle scalene muscles are divided. The first rib is identified and resected. The exposed neural elements may then be performed. From this exposure, dissection can be carried proximally in the neural foramen. Before closure, the operative field should be filled with saline and a Valsalva maneuver performed to identify potential pleural injury. Each muscle layer should also be reapproximated and the skin closed according to the surgeon’s preference. A soft compressive dressing is then applied. A chest radiograph should be performed to look for evidence of hemotthorax or pneumothorax. Higher rates of injury to the long thoracic, dorsal scapular, and spinal accessory nerves are seen in this procedure, along with a 5% incidence of scapular winging.

SUPRASCAPULARIS

- motor nerve (C5) – weakness of:
  1) m. supraspinatus (initiation of shoulder abduction); atrophy is not obvious due to overlying m. trapezius.
  2) m. infraspinatus (only muscle for external rotation of humerus) – hollowing of infraspinatus fossa and prominence of scapular spine.

Etiology – athletes (esp. basketball, volleyball, weight lifting, gymnastics) – compression at suprascapular notch of scapula (strut, strong suprascapular ligament).

Clinical Features

- only sensory fibers in suprascapular nerve supply posterior shoulder joint – chief complaint is insidious onset of deep, dull aching pain in posterior part of shoulder and upper periscapular region.
- deep pressure over midpoint of superior scapular border may produce discomfort.

Best diagnosis – EMG evidence of denervation of supraspinatus and infraspinatus muscles.

N. ISCHIADICUS

There is no consistent area in lower extremity where entrapment occurs!

1) retroperitoneal bleeding
2) course of sciatic nerve between parts of piriformis muscle (PIRIFORMIS SYNDROME)
3) myofascial band in distal portion of thigh (between biceps femoris and abductor magnus)
4) root-neural entrapment of hip, surgical trauma from hip replacement.

PIRIFORMIS SYNDROME

- diagnosis / provocative tests
- treatment

Sciatica – loosely used term - pains in low back and along n. ischiadicus course - caused by involvement of the piriformis muscle, including intraspinal / extraforaminal

N.B. most common cause is impinged intervertebral disc! see p. S21
Common peroneal nerve is more frequently subjected to trauma than any other nerve of body [25% of all compression neuropathies]: 1) superficial location; 2) higher fascicle number and lower connective tissue content at fibular neck than within popliteal fossa (nerve's susceptibility to stretch or compression injury, e.g. gunshot wound in thigh as almost a rule injures peroneal but spares tibial divisions of sciatic nerve).

**Mechanism:**
1. Forceful foot inversion (nerve stretching).
2. Damage at fibular head (bandages, stockings, crossing knees while sitting).

**Etiology:**
1. Thin individuals who habitually cross their legs.
2. Patients who lose significant amount of weight, as in case of cancer or eating disorder, (slimmer's palsy).
3. Certain professions that require frequent sitting, squatting, or kneeling (e.g. roofers, carpet layers, strawberry pickers).
4. Prolonged squatting during childbirth.
5. Asleep while intoxicated.
6. Intravenous injection improper cushioning or positioning of leg under anesthetic (esp. in dorsal lithotomy or lateral decubitus positions), improperly applied casts.
7. Any contact sport.
8. Ganglion cysts.

**Clinically- foot drop** (analogous to wrist drop with n. radialis damage; patients compensate for footdrop by lifting leg higher: 1) pain laterally in leg and foot. See p. 111)
- ask to heel walk.
- Tinel sign is frequently present at site of compression.
- Coexistent foot inversion weakness may suggest either L5 radiculopathy or sciatic nerve injury.
- Biceps femoris weakness - CPN injury above knee.

**Diagnosis:**
1. Electrophysiologic evaluation to exclude other conditions (esp. L5 radiculopathy or more proximal CPN lesion) - record from extensor digitorum brevis or tibialis anterior while stimulating CPN above and below fibular neck to look for focal slowing, temporal dispersion, or conduction block. EMG - on both peroneal-innervated muscles and non - peroneal, L5-innervated muscles. N.B. Short head of biceps femoris is the only peroneal-innervated muscle proximal to peroneal tunnel!
2. Imaging - plain films, MRI, ultrasound.

**CONSERVATIVE THERAPY**
- Effective for most cases of CPN entrapment: Complete or partial recovery is rule when paralysis is acute and duration is less than 2 weeks.
1. PT to prevent contractures.
2. Ankle-foot orthosis (to protect ankle joint and improve gait).

**Surgery**

Peroneal Nerve Decompression - for patients who show little or no improvement after 3-4 months. See p. Op450 +

Persistent footdrop after surgery → TP tendon transfer, highly effective for footdrop caused by CPN injury, particularly in men < 30 years

**N. TIBIALIS POSTERIOR**

**TIBIAL TUNNEL SYNDROME** (posterior tibial nerve entrapment behind medial malleolus at flexor retinaculum or more distally)

**Tarsal tunnel (TT) anatomy:**
TT is a continuation of the deep posterior compartment of the calf into the posteromedial aspect of the ankle and the medial plantar aspect of the foot (Fig. 236). The TT is made up of two main compartments: an upper (tibiotalar) and a lower (talo-calcaneal) compartment. The floor of the upper compartment is formed by the posterior aspect of the talus and the tibia, and the roof is formed by a deep aponeurosis. The posterior tibial neurovascular bundle (including the posterior tibial nerve) runs through this space with the tendons of the TP, FDL, and flexor hallucis longus. The lower compartment of the TT contains the abductor hallucis muscle [136]. The tibial nerve passes within the upper compartment of the TT posterior to the tendons of the TP and FDL and the posterior tibial artery and vein. The medial and inferior calcaneal nerves may arise proximal to, within, or distal to the TT.

**Figure 236:**
- 1, Tendons of the posterior tibial muscle; 2, tendon of the flexor digitorum longus muscle; 3, tibial nerve; 4, flexor retinaculum; 5, medial plantar nerve; 6, tibial posterior nerve.

Source of picture: Edward J. Shahady, "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN 0387306469

1. Tendons of the posterior tibial muscle. 2, tendon of the flexor digitorum longus muscle. 3, tibial nerve. 4, flexor retinaculum. 5, medial plantar nerve. 6, tibial posterior nerve.
etiology (quite rare): bony impingement (ankle trauma), space-occupying lesions (ganglion cysts, neurolemomas, RA tenosynovitis, hypertrophic muscles, or varicosities, gout, diabetes, and myxedema).

- clinical symptoms:
  1. burning, unpleasant poorly localized pain and paresthesias in medial heel* + sole (down to first, second, and third toes)
  2. calcaneal branch (sensation to heel) often is spared because of its proximal takeoff
  3. pain is set off by pressing or rubbing over plantar skin, sometimes with after-discharge phenomenon.
  4. pain aggravated by forced dorsiflexion and eversion of ankle.
  5. some patients experience nocturnal exacerbations
  6. pain reminds plantar fasciitis, but positive Tinel sign (tapping* over area posterior to medial malleolus = numbness, tingling) is present.

- treatment:
  1. pain compression with finger for 30 s
  2. intrinsic toe flexors are weak and atrophic → hollowing of instep, toe clawing.

Diagnosis
1. Electrophysiologic evaluation
   - tibial motor nerve conduction may exhibit prolonged distal onset latency when recorded over the abductor hallucis and abductor digit minimi.
   - mixed nerve conduction studies of medial and lateral plantar nerves may demonstrate prolonged peak latency or slowed velocity; sensory nerve conduction of two nerves may be slowed or absent across tarsal tunnel
2. Imaging - plain films, MRI, ultrasound.
   - differential diagnosis: plantar fasciitis, stress fractures, bursitis, diabetic neuropathy, posterior tibial tendonitis.

TREATMENT
Period of conservative therapy should be attempted before surgical intervention.
- lifestyle modification (weight loss and avoidance of ill-fitting shoes or high heels).
- trial of immobilization
- orthotics (medial arch support - avoids extreme ankle eversion and dorsiflexion)
- corticosteroid injections.
- nerve blocks
- antiepileptic, antianxiety, and narcotic pain medications may help with chronic pain

Surgical Decompression (75% patients enjoy significant improvement)
- incision begins 2 cm proximal to medial malleolus to pick up neurovascular bundle above flexor retinaculum.
- nerve is followed distally with release of retinacular fibers.
- mass lesions or fibrous septae are identified and removed.
- each of plantar nerve canals is opened into plantar surface.
- tight fascial band arising from border of m. abductor hallucis and roofing over plantar canals is divided.
- all intersecting septae are cut to convert tunnels into single cavity.
- ankle is placed in soft splint and elevated for 3 days → minimal weight-bearing for additional week.

From Youmans
Open exploration of the TT is the preferred surgical technique, but endoscopic techniques have been developed. [112,144] Success rates for surgical decompression of the TT have been reported to be between 44% and 93%, with success being defined as resolution or improvement of symptoms, no requirement for pain medications, and the ability to return to work.

Curvilinear incision is started 4 cm proximal to the medial malleolus while staying posterior to the medial malleolus, extends distally toward the midaspect of the plantar surface of the foot, and curves anteriorly at the heel. The deep fascia over the neurovascular bundle is divided proximal to the TT, and division is continued distally as the fascia thickens to form the flexor retinaculum. The fascia covering the abductor hallucis brevis signifies the end of the TT. The medial and lateral plantar nerves are identified and followed into their two separate tunnels. Both tunnels are released by dividing the fascial origin of the abductor hallucis brevis, which forms their root. Any calcaneal branches are identified and decompressed. The posterior tibial vessels are elevated and the tibial nerve and its branches are inspected. Complete external neurolysis is usually performed.

Schematic representation of the course of the tibial nerve (central sketch) and the various endoscopic (left and right sketches) approaches to the tarsal tunnel are depicted by the sketches on the right. A. The flexor retinaculum is sharply divided along its entire length in line with the central sketch; the tarsal tunnel is released proximally and distally. B, C. A parallel incision is made along the distal portion of the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. D, E. A parallel incision is made along the proximal portion of the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. F, G. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. H. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. I. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. J. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. K. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. L. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. M. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. N. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. O. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. P. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. Q. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. R, S. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. T. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. U. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. V. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. W. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. X. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum. Y. A parallel incision is made along the posterior tibial vessels, and the tibial nerve is freed from the vessels and the posterior tibial vessels and flexor retinaculum. Z. A parallel incision is made along the anterior tibial vessels, and the tibial nerve is freed from the vessels and the anterior tibial vessels and flexor retinaculum.
Mullick and Dellon recently reported their long-term outcomes after decompression of the TT. The series included 87 procedures with a mean follow-up of 3.6 years. Significant improvement was seen in motor and sensory function. Using unspecified postoperative assessment techniques, there were 82% excellent (resolution of symptoms), 11% good (slight residual numbness and tingling, able to return to work, no pain medications), 5% fair (residual symptoms requiring pain medications, unable to return to work), and 2% poor results (no improvements). [139] Revision surgery for TTS carries a less favorable outcome. Barker and coauthors reported a series of 44 patients who underwent revision by neurolysis, resection of scar neurapraxia, or occasional neurorrhaphy with special mention to either nerve transposition and surgical tunnel release. Clinical application. Neurosurgery. 2006;59:ONS89

MERALGIA PARESTHETICA

- entrapment of purely sensory lateral femoral cutaneous nerve (L-2) where it passes beneath inguinal ligament → uncomfortable numbness, tingling, painful hypersensitivity.

- patient learns to relieve symptoms by:
  - placing pillows behind thighs;
  - assuming slightly hunched posture while standing.
- deep digital pressure 1 cm medial to anterior superior iliac spine (ASIS) may set off shooting paresthesia down lateral thigh.
- diagnosis is confirmed with nerve block - 0.5% BUPIVACAINE injected finger's breadth medial to ASIS → anesthesia + complete cessation of pain and tingling.
- treatment:
  1) weight loss, avoidance of all constrictive garments, and postural modification.
  2) serial injections of local anesthesia and steroid.

Surgical decompression:

- Incision - along medial border of sartorius, 2 cm below ASIS; extends 6-7 cm.
- fascia over sartorius is opened carefully.
- nerve is located at medial muscle border or just behind it (also may be attached to underside of fascial sheath - careful handling to avoid cutting nerve).
- nerve is traced proximally - toward exit site just medial to ASIS.
- bands of inguinal ligament over nerve are divided (hernia is extremely rare after this procedure!).
- if sharp ridge is palpable just below nerve, it also should be divided to completely free nerve of sharp surfaces.
- nerve is followed into pelvis for 2 cm, ligature is tied tightly around nerve.
- nerve is firmly tugged downward while cut is made just proximal to tie.

15-20% of cases recur → nerve transection (neurectomy):

- after freeing nerve at ASIS and proximally toward pelvis, ligature is tied tightly around nerve.
- nerve is firmly tugged downward while cut is made just proximal to tie.
• upper cut end of nerve springs back and disappears into pelvic cavity - this prevents painful neuroma formation on surface of thigh.

• pain is gone, and patient usually adjusts well to numbness.

MORTON’S NEUROMA

Benign perineurium thickening (fibrosis, not true neuroma!) at 3rd interdigital nerve due to pinching between heads of 3rd and 4th metatarsals; 2nd and 3rd is next most common site.

• most often unilateral.

• women > men.

• causes:
  1) tight shoes (compress toes)
  2) loss of fat-pad of ball

• clinical features
  - pain (metatarsalgia), tenderness, paresthesias along nerve (sometimes patient takes off shoe to decrease pain)
  - patient may feel “mass” between metatarsal heads.
  - long-standing cases will have decreased sensation in web space.

• diagnosis: tenderness between 3rd and 4th metatarsal heads; compressing metatarsal heads between examiner’s thumb and fifth digit will accentuate pain.

• treatment:
  1) comfortable shoes, orthotics (metatarsal pad).
  2) lidocaine + corticosteroid infiltration - given dorsally (top of foot) so that it is less painful.
  3) surgical excision

OTHER NERVES

about motor and sensory signs → see p. D1 >>

Nerve (spinal segment): muscle, sensory

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Compressive Sites &amp; Causes</th>
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<tbody>
<tr>
<td>Axillary (C5-6)</td>
<td>m. deltoideus, teres minor; C5 sensory</td>
</tr>
<tr>
<td></td>
<td>Near shoulder joint: fractures / dislocation of humerus head; neuritis after serum (esp. antitetanus) therapy</td>
</tr>
<tr>
<td>Long thoracic (C5-7)</td>
<td>m. serratus anterior; not sensory</td>
</tr>
<tr>
<td></td>
<td>Surgery</td>
</tr>
<tr>
<td>Femoral (L1-2)</td>
<td>m. iliopsoas, quadriceps femoris; anterior thigh sensory</td>
</tr>
<tr>
<td></td>
<td>Proximal to inguinal ligament: idiopathic, iatrogenic, retroperitoneal hemorrhage, tumor</td>
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<tr>
<td>Saphenous</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Iatrogenic (surgery, scar after surgery)</td>
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<tr>
<td>Obturator (L3-4)</td>
<td>m. obturator; medial thigh sensory</td>
</tr>
<tr>
<td></td>
<td>Pelvic tumor, hematoma, obturator hemia, difficult labor</td>
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</tbody>
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EXERTIONAL COMPARTMENT SYNDROMES

Deep posterior compartment syndrome (n. tibialis) → see p. A22 (7), p. 1226a
Anterior compartment syndrome (n. peroneus profundus) → see p. A22 (9), p. 1226a

BIBLIOGRAPHY for ch. “Peripheral Neuropathies” → follow this LINK >>