Compressive Neuropathies (s. Entrapment Neuropathies, Tunnel Syndromes)

Last updated: August 8, 2020

ETIOLOGY

1. Pathophysiologic

1. Double Crush Syndrome

2. Clinical Features

3. Diagnosis

2. Treatment

N. Medians

3. Places of compression

CARPAL TUNNEL SYNDROME (CTS)

3. Provoking factors

3. Clinical Features

5. Diagnosis

5. Treatment

Surgery

N. Ulnaries

6. N. Ulnaries at elbow

Clinical Features

Diagnosis

Treatment

N. Ulnaries at wrist

Treatment

N. Radialis

9. Clinical Features

9. Diagnosis

9. Treatment

THORACIC OUTLET SYNDROME (TOS)

10. Classification & Causes

10. Vascular TOS

10. Neurogenic TOS

10. Neurogenic TOS

10. Vascular TOS

11. Diagnosis

11. Neurogenic TOS

11. Vascular TOS

12. Treatment

12. Neurogenic TOS

N. Suprascapularis

14. N. Iliohypogastricus

14. N. Pernoneus

15. Conservative therapy

15. Surgery

N. TIBialis posterior

15. Treatment

MELASIA PARESTHETICA

17. MORNON’S NEUROMA

18. OTHER NERVES

18.

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. Extensive compressions - casts, crutches.

4. Intrinsic compressions - tumors, benign hyperostosis, inflammatory edema of adjacent structures, infuriating substances (e.g. amyloid, hypothyroidism, 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 anesthesia) in thin-cachectic persons (esp. alcoholics)
  b) nerves at narrow osseoligamentous canals (e.g. carpal tunnel).

- in all cases, at least one side of compressive surfaces is movable – allows chronic injury: either repetitive “slapping” insult or “rubbing/sliding” 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 bones of Ranvier → local segmental demyelination (still reversible with treatment) → axonal disruption, epineurial fibrosis (constant feature?)

<table>
<thead>
<tr>
<th>Pressure</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 mm Hg</td>
<td>unpaired 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 mononeural ischemia (sensory and motor block)</td>
</tr>
</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
Compressive Neuropathies

Clinicopathies

Coexistent compressive lesions in series along course of peripheral nerve, with one lesion rendering nerve susceptible to distal or proximal compression.

- mechanisms: 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 (12)

Places of Compression

1. Near elbow (proximal median neuropathy):
   1) ligament of Struthers / supracondylar process of humerus
   2) Lacertus fibrosus (bicipital 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.
   Certain sports are associated: wheelchair athletes, archers, bicyclers, bodybuilders, football players, golfers, wrestlers

2) pregnancy (esp. 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 neck1 (mimics C6-7 radiculopathy)

Mild disease: paresthesias & pain in median nerve distribution (after strenuous wrist movements or nocturnal)1

- because of venous stasis (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).

**COMPRESSION NEUROPATHIES**

**PN5 (4)**

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>Palmar and dorsal surfaces of thumb, index, middle fingers</td>
</tr>
<tr>
<td>Near elbow (pronator syndrome)</td>
<td>Opponens pollicis</td>
<td>Opposition</td>
<td>Palmar, palmar and dorsal surfaces of thumb, index, middle fingers (no loss on forearm)</td>
</tr>
<tr>
<td>Pronator teres</td>
<td>Pronation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor pollicis longus</td>
<td>Flex thumb, distal joints</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor digitorum sublimis</td>
<td>Flex fingers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor digitorum profundus</td>
<td>Flex fingers, median side</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor carpi radialis</td>
<td>Wrist flexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lumbricales (two radial)</td>
<td>Extensor MP joint</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Below elbow (anterior interosseous branch)</td>
<td>Flexor pollicis longus</td>
<td>Flex thumb, distal joint</td>
<td></td>
</tr>
<tr>
<td>Flexor digitorum profundus II</td>
<td>Flex index finger, distal joint</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Severity assessment:

Carpal Tunnel Syndrome Assessment Questionnaire (CTSAQ) - 9-item functional status scale.
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) **Durrant 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).

6) other provocative maneuvers - reverse Phalen test, Gilliat (tourniquet) test, ultrasonic stimulation test.

7) MRI of wrist - nerve thickening, increased signal intensity within inflamed peripheral nerve.

8) **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:

2. **Ergonomic corrections**

3. **Ultrasound 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:

7. **Therapeutic endpoints**:

   a. 1 cc of local anesthetic and 1 cc of long-acting corticosteroid.
COMPRESSIVE NEUROPATHIES

1. Arcade of STRUTHERS (diatia 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. CUBITAL TUNNEL SYNDROME at elbow groove (e.g. cubitus valgus, medial condyle fracture, RA synovitis, osteophytes) - compression between cubital tunnel retinaculum (OSBORN’s ligament) and medial collateral ligament (MCL).

3. External 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 OSBORN’s fascia; 3.5-5 cm distal to cubital tunnel) – e.g. in pianists (repeated forceful wrist flexion).

5. Medial intermuscular septum - sharp edge that can indent nerve (esp. after anterior transposition where nerve may be kinked).
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 suggests entrapment at Guyon’s canal (spared dorsal cutaneous branch distribution).

Referred pain with entrapment neuropathy can radiate proximally to the site of entrapment (mimics CR radiculopathy).

2) **"CLAW HAND"**, hand clumsiness, dropping objects; hypothenar + interossei weakness and atrophy.

- 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.

**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 suggests 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!

**Postsurgical Electrical Stimulation Enhances Recovery**

Stimulation Protocol

Prior to skin closure, 2 sterile Teflon-coated stainless-steel electrodes were placed transcutaneously immediately adjacent to the ulnar nerve proximal to the site of compression. These were secured to the surgical dressing using tape. In the PACU, a research assistant administered PES using a Grass SD9 stimulator (Grass Technologies, Warwick, Rhode Island). The proximal wire electrode was connected to the cathode and the distal to the anode.

Patients in the stimulation group received 1 hour of stimulation as a continuous 20-Hz train of balanced biphasic pulses. The stimulation intensity was set at the tolerance limit (<30 V, 0.1 ms pulse duration).

Patients in the control group received 5 s of similar-intensity PES before the stimulator was turned off for the remainder of the hour.

Because none of the patients had previously received PES, it was difficult for them to guess which group they had been randomized to. This was further aided by sensory accommodation following repetitive stimulation and that the patients were still groggy under the influence of general anesthetics and opioid analgesics in the recovery unit.

The stimulation electrodes were removed and discarded at the end of the stimulation session.

A. Electrode placement, intraoperative view of the right arm. The black asterisk (*) marks the decompressed ulnar nerve, and the black arrows mark the stimulating electrode wires that were lay immediately adjacent to the ulnar nerve proximal to the site of compression. B. Electrode placement for stimulation of the right arm in the postanesthesia recovery room. The proximal wire electrode was connected to cathode (black), whereas the distal electrode was connected to anode (red). C. The stimulator used (Grass SD9).

- outcomes for sensation and pain were not studied.
- potential barrier to clinical implementation is the requirement for general anesthesia (not to have interference of local anesthetic).
- in rats, 1 h of 20 Hz PES produced the same beneficial results as week-long continuous stimulation in motor nerves, whereas stimulation durations longer than 1 h (ie, 3 h, 7 d, and 14 d) were harmful for regenerating sensory nerves.

N.B. carpal tunnel release: although motor reinnervation was significantly better in patients who received carpal tunnel release and PES, there was no significant functional improvement compared to surgery alone (due to the short regeneration distance and that fine dexterity can be compensated for by the ulnar-innervated muscles).

N. ULNARIS AT WRIST
- compression at ulnar GUYON canal:
  1) repetitive and forceful actions (e.g., Elbow dislocations, tennis, squash, tennis racket, throwing sports)
  2) paraplegics, using hand controls with horizontal bar across palm.
  3) motorcyclists who firmly grasp hand bar control.
  4) operators of pneumatic drills.

- compression at radial (antero-posterior) 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).
- at proximal carpal bones, it dips between pisiform and hamate to entrance to Guyon canal, roofed over by extension of transverse carpal ligament between these 2 bones.
- superficial hypohyponean sensory branch (hypohyponean skin ulnar to vertical line at base of ring finger) supplies this muscle, and crosses after entering Guyon canal, deep motor branch first supplies abductor digiti minimi (ADM), then crosses the angle where nerve turns radially.
- in other 35% individuals, some pain and hypohyponean numbness is expected.
- after entering Guyon canal, deep motor branch first supplies adductor digitii minimi (ADM), then crosses under one head of flexor digiti minimi (FDM), supplies this muscle, and crosses over to supply opponens digitii minimi 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, opposedness always is affected, together with interossei, ulnar lumbricates, and adductor pollicis.

N. RADIUS.

Places of compression.

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

2. **HUMERUS SHAFT TRAUMA** (surgical groove)
   - between medial and lateral heads of triceps.

3. Underneath arcade of FROHSE (musculotendinous arcade, formed by upper free arterial branches (of Henry) arising from radial recurrent artery cross over nerve just distal to spiral groove)
   - radial tunnel (p. to 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!
   - leash 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 radiocapitellar joint capsule.

b) Monteggia fracture-dislocation.

c) vascular compression by hypertrophic leash of Henry.

d) synovial cyst, rheumatoid synovitis.

e) tenosynovitis of arcade of Frohse.

f) chronic trauma to flexion surface of forearm (e.g. constriction rings of Canadian crutches in paraplegics).

**TREATMENT**

- **avoidance & use of palmar padding.**

- **surgery** — see p. A3450

**CLINICAL FEATURES**

1. **Motor** - see p. D113

   1) **WRIST DROP** with paralysis of finger extension at MCP joints (IP joints extension — action of mm. lumbricalis).

   - 2nd and 5th fingers receive both their own extensor tendon 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 weakness of ulnar wrist extensor.

   - pseudomega — weakness 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) **TENSE** sign at radial tunnel.

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

3) **EMG**

**TREATMENT**
Three sites within thoracic outlet where neurovascular compression may occur:

1. **Upper Thoracic Area Toward Axilla and Proximal Arm.**
   - 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 brachioradial 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.
   - Fissural thickening associated with joint capsule also is divided, as is arterial leash of PIN within radial tunnel.

2. **Costoclavicular Space.**
   - Brachial plexus runs anterior to 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.

3. **Subclavian Space (Costoclavicular Triangle).**
   - Anterior scalene (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!

**Symptomatic (Common, Secondary, Disputed) Neurogenic TOS:**
- No identifiable anatomical structure causing nerve compression! ("wastebasket" diagnostic group that includes chronic pain syndromes of multiple causes)
- Preputating factors:
  1. **Scalenus Muscle Spasm (Scalenus Anticus Syndrome).**
     - Due to minor cervical or shoulder trinitia.
  2. Abnormal Shoulder Posture:
     - "Droopy Shoulder Syndrome" - tall, slender, and round-shouldered person.
     - 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. **Costoclavicular Space.**
   - Medial third of clavicle anteriorly, first rib posteriorly, and upper border of scalpula posteromedially.
   - Immediately distal to interscalene triangle.

3. **Subclavian Space (Subclaviocoracoid Space).**
   - Deep to the pectoralis minor tendon - distal to costoclavicular space.

**Arm hyperabduction and external rotation produces compression of neurovascular elements within costoclavicular space.

**Clinical Features:**

- **Neurogenic TOS:**
  - Various 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 C5-T, 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 scapular musculature.
  - Weakness of all intrinsic hand muscles (C5-T, myotomes) → muscle atrophy
    - Classic Gilliatt-Sumner hand - dramatic atrophy in abductor pollicis brevis and lesser atrophy in interosseus and hypothenar muscles.
2) numbness, pain, sensory loss (lateral aspect of neck, shoulder, axilla, parascapular region, and ulnar side of hand and forearm)
   - pain is aggravated by pulling arm down or repetitive overhead arm use; arm "fatigue" is often prominent.
   - other classic provocative maneuvers (sensitivity 72% and specificity 53%; false-positives 45-77%):
     1. **Roos 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. **Wright 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!
- 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-Schroetter syndrome) distal to constriction.
- in later stages, 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**

**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 (Perry's exercises) and scalene musculature, plus, focused toward correcting poor posture and improving cervical and pectoralis 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 supraspinous fascia and pectoralis major over the clavicular head of the first rib. C. The anterior scalene muscle is retracted medially. D. The transverse cervical vessels are ligated with 0 silk ties and divided. E. After division of the anterior scalene muscle, the upper (UT), middle (MT), and lower (LT) trunks of the brachial plexus and the subclavian artery (SA) are identified. The plexus nerve (PN) is gently retracted medially.
During exposure, important anatomic landmarks to identify are the posterior border of the sternocleidomastoid muscle, the omohyoid muscle, the supraclavicular 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 sizable cutaneous nerves to avoid a painful neuroma. 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 suprascapular 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. Typically, we perform the transection in piecemeal fashion with bipolar coagulation and scissors while carefully protecting the overlying phrenic nerve. The upper, middle, and lower trunks of the brachial plexus are running laterally and inferiorly deep to the lateral edge of the anterior scalene. An identifying loop is placed around each trunk. The subclavian artery is identified running inferomedially in the plane of the brachial plexus and is controlled with a vessel loop. Frequently, glistening white fascial bands are seen within the anterior and middle scalene muscles and, in many cases, are the presumed culprits in compression/irritation of the plexus elements. The neural elements are inspected in circumferential fashion, and any compressive bands or anomalous structures are resected.

Occasionally, the supracleural 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 than the first rib. The lower trunk is more likely to contact the plexus than the upper or middle trunks. 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. This procedure can be performed with minimal morbidity by surgeons experienced in this approach. Numberous over the supraclavicular region, lasting approximately 6 weeks, may occur as a result of manipulation of or injury to the supraclavicular nerve during the approach; in certain circumstances, painful neuromas or neurotopathic pain, or both, may form at the site of the nerve injury. Major complications from this approach include pneumonathorax (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 approach is used. Transient paresthesias or weakness in the arm or hand is seen occasionally and generally resolves within days to a few weeks.
Posterior Subscapular Approach

The posterior subscapular approach as described by Kline and associates provides excellent exposure of the C8 and T1 spinal nerves and the lower trunk of the brachial plexus.[10,11] This approach is particularly useful in patients who have previously undergone anterior approaches or received radiation therapy to the area. The posterior subscapular approach is performed with the patient in the prone position and the arms of the affected side abducted at the shoulder and flexed at the elbow. A curvilinear incision is centered between the upper thoracic spinous processes and the medial border of the scapula. The first muscular layer, the trapezius, is split along the incision in a caudal direction with care taken to preserve the spinal accessory nerve in this layer. The next layer, composed of the levator scapulae and the rhomboid muscles, is divided in similar manner. The scapula is retracted into an abducted and externally rotated position with the use of a chest retractor placed between the medial border of the scapula and the paraspinal musculature. The first rib is exposed and removed from the costotransverse articulation to the costoclavicular ligament. The posterior and middle scalene muscles are divided to expose the spinal nerves and trunks of the brachial plexus. The long thoracic nerve should be identified and protected. Careful and complete external neurolysis of 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’s maneuver performed to identify potential pleural injury. Each muscle layer should also be reaproximated 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 hemothorax 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.

Transaxillary Approach

The transaxillary approach with resection of the first rib was popularized by Roos in 1966 and is still commonly used by many thoracic and vascular surgeons. The patient is placed in the posteroanterior position with the arms 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 anterior and middle scalene muscles are divided. The first rib is identified and resected. The advantage with this approach is that it allows easy and almost complete access to the first rib, unhindered by adjacent neurovascular structures. The posterior third of the first rib may, in large patients, be difficult to excise with this approach. The major shortcoming of this approach is limited exposure of the neurovascular structures, behind which bony con genital bands or a cervical rib may be located. Endoscopically assisted transaxillary techniques have recently been developed and are reported to be safe and effective. Major complications with this approach include brachial plexus injury (1% to 3%), venous injury (2%), and pneumothorax (9%). Other reported complications have included Horner’s syndrome and damage to the thoracic duct.[1] Patients may also experience paresthesias or hypersensitivity in the distribution of the intercostobrachial nerve because this nerve is vulnerable to injury with this approach.

N. SUPRASCAPULARIS

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Weakness</th>
</tr>
</thead>
<tbody>
<tr>
<td>m. infraspinatus (only muscle for external rotation of humerus)</td>
<td>hollowing of infraspinous fossa and prominence of scapular spine.</td>
</tr>
<tr>
<td>m. supraspinatus</td>
<td>hollowing of supraspinatus fossa and atrophy of m. supraspinatus</td>
</tr>
<tr>
<td>m. subscapularis</td>
<td>hollowing of subscapular fossa and atrophy of m. subscapularis</td>
</tr>
<tr>
<td>m. teres minor</td>
<td>atrophy of m. teres minor</td>
</tr>
</tbody>
</table>

Clinical Features

- only sensory fibers in suprascapular nerve supply posterior aspect of shoulder joint
- chief complaint is insidious onset of deep, dull aching pain
- EMG evidence of denervation of supraspinatus and infraspinatus muscles.

Treatment

a) If pain is only manifestation of syndrome
   - CONSERVATIVE MANAGEMENT: cessation of athletic activities, conditioning exercises of upper girdle, periodic injection of nerve (bupivacaine and dexamethasone).
   - surgery: anterior approach to suprascapular notch, posterior approach to suprascapular ligament.

b) Failure of pain control / severe weakness
   - SURGICAL DECOMPRESSION (symptomatic improvement is expected in 95% patients; some patients never regain full strength due to atrophy - early detection is most important predictor of outcome!):
   - patient is placed prone.
   - incision 2 cm above and parallel to scapular spine.
   - horizontal trapezial fibers areatraumatically split to expose constant fat pad separating trapezius from suprascapular muscle.
   - digital palpation along sharp, bony edge of superior scapular border detects abrupt change into rubbery springiness of suprascapular ligament.
   - blunt dissection by firm, sweeping motion using “peanut” dissector readily reveals glistening, taut ligament.
   - suprascapular artery, which crosses above ligament, is swept aside.
   - ligament is cut and bony notches enlarged with rongeur, if necessary.
   - nerve is exposed and widened by decompressing if encasing fibrofatty tissue.

N. ISCHIADICUS
Anatomy – see p. A22 (8)

Common peroneal nerve is more frequently subjected to trauma than is any other nerve of body (> 25% of all compression neuropathies): 1) superficial location and 2) higher fascicle number and lower connective tissue content at fibular neck than within popliteal fossa (t Farrer’s susceptibility to stretch or compression injury, e.g. gunshot wound in thigh almost as a rule injures peroneal but spares tibial divisions of sciatic nerve)

Mechanisms
1) forcible foot inversion (nerve stretching).
2) damage at fibular head (bandages, stockings etc).

Etiology:
1) thin individuals who habitually cross their legs
2) patients who lose significant amount of weight (space closure effect)
3) certain professions that require repetitive movements (e.g. roofers, carpet layers, strawberry pickers).
4) prolonged squatting during childbirth
5) adenoids while intoxicated
6) iatrogenic injury – 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) "n. pain" laterally in leg and foot. See p. D1 ++
- 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 tonal - 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 paralyis is caused by transient pressure!
1. PT to prevent contractures.
2. AFO with 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. Dp4720 ++

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

N. TIBIALIS POSTERIOR

TARSAL TUNNEL SYNDROME (posterolateral 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. See Fig. 236-7. The TT is made up of two main compartments: an upper (tibial) and a lower (talo-calcaneal) compartment. The floor of the upper compartment is formed by the posterior aspect of the tibia and the talus, 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. 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.
Surgical Decompression

1. Tendon of the posterior tibial muscle; 2, tendon of the flexor digitorum longus muscle; 3, tibial nerve; 4, flexor retinaculum; 5, medial plantar nerve; 6, lateral plantar nerve

Diagnosis

1. Electrophysiologic evaluation
   - tibial motor nerve conduction may exhibit prolonged distal onset latency when recorded over the abductor hallucis and abductor digiti 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, antiantidepressant, 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. [121,144] Succes 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 roof. 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.
Surgical decompression: a treatment of choice for patients with diabetic foot neuropathy. This approach has been stated to be of unproven value by the American Academy of Neurology. This controversial approach has not yet been subjected to a prospective, randomized trial and has been stated to be of unproven value by the American Academy of Neurology. 

- Decompression technique also improves sensation and reduces foot pain in diabetics with sensory neuropathy. [148] This controversial approach has not yet been subjected to a prospective, randomized trial and has been stated to be of unproven value by the American Academy of Neurology.

- Prevalence of ulceration was reduced from 50% to 2.2%. The authors claim that this triple decompression technique has advocated external neurolysis of the posterior tibial nerve, only 4 showed improvement (40%); of the 5 patients who underwent internal neurolysis of the posterior tibial nerve, 2 (40%) had satisfactory results. Seven patients from the series underwent neurectomy of the posterior tibial nerve, only 2 of these patients experienced ulceration of the sole at a mean follow-up time of 3.2 years. [114] For diabetic sensory neuropathy of the lower extremity, Dr. Dellon has advocated external neurolysis of the CPN at the knee, peroneal branches over the ankle, and fascial sheath of the soleus muscle; N.t., nervi tibialis; R.N.t., ramus nervi tibialis (tibial nerve).

- Surgical decompression: a treatment of choice for patients with diabetic foot neuropathy. This approach has been stated to be of unproven value by the American Academy of Neurology. This controversial approach has not yet been subjected to a prospective, randomized trial and has been stated to be of unproven value by the American Academy of Neurology.

- Multlick 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 surgery by neurolysis, resection of scar neureura, or occasional neurectomy, with a primary outcome measure of self-reported patient satisfaction. At a mean follow-up time of 2.2 years, 54% reported excellent results; 24%, good results; 13%, fair results; and 9%, poor results. [147] Kim and Munovic reported a series of patients who underwent revision surgery for TTS at LSUMHC. Of the 10 patients who underwent external neurolysis of the posterior tibial nerve, only 4 showed improvement (40%); of the 5 patients who underwent internal neurolysis of the posterior tibial nerve, 2 (40%) had satisfactory results. Seven patients from the series underwent neurolyctomy of the posterior tibial nerve, all of whom reported improvement in pain; none of these patients experienced ulceration of the sole at a mean follow-up time of 3.2 years. [114] For diabetic sensory neuropathy of the lower extremity, Dr. Dellon has advocated external neurolysis of the CPN at the knee, peroneal branches over the ankle, and fascial sheath of the soleus muscle; N.t., nervi tibialis; R.N.t., ramus nervi tibialis (tibial nerve).
COMPRESSIVE NEUROPATHIES

15-20% cases recur → nerve transection (neurectomy):
- after freeing nerve at ASIS and proximally toward pelvis, ligature is tied tightly around nerve.
- nerve is firmly nugged 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) of 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:
  - 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 (spinal segment): muscle, sensory</th>
<th>Compressive Sites &amp; Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axillary (C5-6): m. deltoideus, teres minor; C5 sensory</td>
<td>Shoulder joint fractures / dislocation of humerus head; neuritis after serum (esp. antitetanus) therapy</td>
</tr>
<tr>
<td>Long thoracic (C5-7): m. serratus anterior; not sensory</td>
<td>Surgery</td>
</tr>
<tr>
<td>Femoral (L2-4): m. iliopsoas, quadriceps femoris; anterior thigh sensory</td>
<td>Proximal to inguinal ligament: iatrogenic, iatrogenic, retroperitoneal hemorrhage; tumor</td>
</tr>
<tr>
<td>Saphenous</td>
<td>iatrogenic (surgery, scar after surgery)</td>
</tr>
<tr>
<td>Hip/groin, obturator (L1-2): m. obturator internus, adductors; medial thigh sensory</td>
<td>Pelvic tumor, hematoma, obturator hemi, difficult birth</td>
</tr>
</tbody>
</table>

**EXTRATROPHIC 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 >>