Compressive Neuropathies (s. Entrapment Neuropathies, Tunnel Syndromes)

Last updated: February 6, 2023

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. Intrinsic compressions - tumors, bony hyperostosis, inflammatory edema of adjacent structures, infiltrating 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.

Entrapment neuropathies may be associated with:
1. Diabetes mellitus
2. Hypothyroidism: due to glycogen deposition in schwann cells
3. Acromegaly
4. Amyloidosis: primary or secondary (as in multiple myeloma)
5. Carcinomatosis
6. Polymyalgia rheumatica
7. Rheumatoid arthritis: 45% incidence of 1 or more entrapment neuropathies
8. Gout

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 mobile* – 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 nodes of Ranvier → focal segmental ***demyelination*** (still reversible with treatment) → ***axonal loss*** → epineurial fibrosis-neuroma (constant feature!)

ISCHEMIA + EDEMA

Pressure	Result
30 mm Hg	impaired axonal transport
40 mm Hg	paresthesias and neurophysiologic changes
50 mm Hg	axonal block
60 mm Hg	complete intraneural ischemia (sensory and motor block)

- **brief** compression primarily affects myelinated fibers, and classically spares unmyelinated fibers
- **chronic** compression affects both myelinated and unmyelinated fibers → demyelination, axon loss, fibrosis-neuroma.

- 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

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

- first postulated by Upton and McComas in 1973.
- mechanism: impaired axonal flow, ischemia, and altered nerve elasticity, which lessen nerve's resiliency. Intrinsic neuropathies additionally affect nerve's susceptibility to injury.
- common examples: cervical radiculopathy and CTS, thoracic outlet syndrome and CTS, 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 (mimics radiculopathy)!!!
Referred pain is so common that Frank Mayfield once said that *patients with nerve entrapment don't know where the problem is located*

DIAGNOSIS

Diagnosis of most entrapment neuropathies is **clinical**!

Always check Tinel sign!

Always order EDX

May add US (not pricy MRI)*

*uncommon entrapments (e.g. Guyon, PIN) – order MRI!

- **nerve conduction** abnormalities across entrapment tunnel.
- **EMG** - signs of *denervation* (but only after > 3 weeks)
- **ultrasound** – swollen nerve
- **MRI** using short inversion imaging recovery technique (**STIR**) - *high signal intensity* in affected nerve segment at site of compression (due to presence of edema in myelin sheath and perineurium).
  Uncommon entrapments (e.g. Guyon, PIN) – order MRI!
- **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

Laboratory tests - recommended only in cases where an underlying peripheral neuropathy is suspected (i.e. unclear etiology in a young individual with no risk factors such as repetitive hand use):
1. HgA1c (DM)
2. BMP (uremic neuropathy)
3. Thyroid hormone levels (myxedema).
4. Vit. B12 levels
5. Multiple myeloma: anemia, 24 hour urine for kappa Bence-Jones protein, SPEP with reflex IFE and FLC, skeletal radiologic survey.

TREATMENT

Conservative therapy should be tried first.

- mainly consists of educating patient to adopt ***avoidance behaviors***.
- various ***splints*** and ***paddings***.
- steroid injections.
- PT, TENS
- Botox injections
- local measures – lidocaine patch, capsaicin cream, ice applications.
- NSAIDs, antiepileptic, antidepressant, and narcotic pain medications.

Surgical decompressions, s. **external neurolysis** (incl. endoscopic techniques).

- low risk for serious morbidity and high success rates

N. OCCIPITALIS

- greater occipital nerve (nerve of Arnold) - sensory branch of C2.

- entrapment presents as occipital neuralgia: pain in occiput usually with a trigger point near superior nuchal line → pain radiating up along back of head towards vertex.
- more common in women.
- traumatic cervical extension may crush C2 root and ganglion between C1 arch and C2 lamina.

TREATMENT

- PT, TENS
- Botox injections
- **trigger point injection** with steroids and local anesthetics - only temporary relief.
- surgical nerve root **decompression or neurectomy** may provide effective pain relief for some patients.
    - occipital nerve usually pierces the cervical muscles ≈ 2.5cm lateral to midline, just below inion.
    - palpation or Doppler localization of the pulse of the accompanying greater occipital artery sometimes helps to locate the nerve
- decompression of C2 nerve root if compressed between C1 and C2 ± atlanto-axial fusion.
- occipital nerve **stimulators**

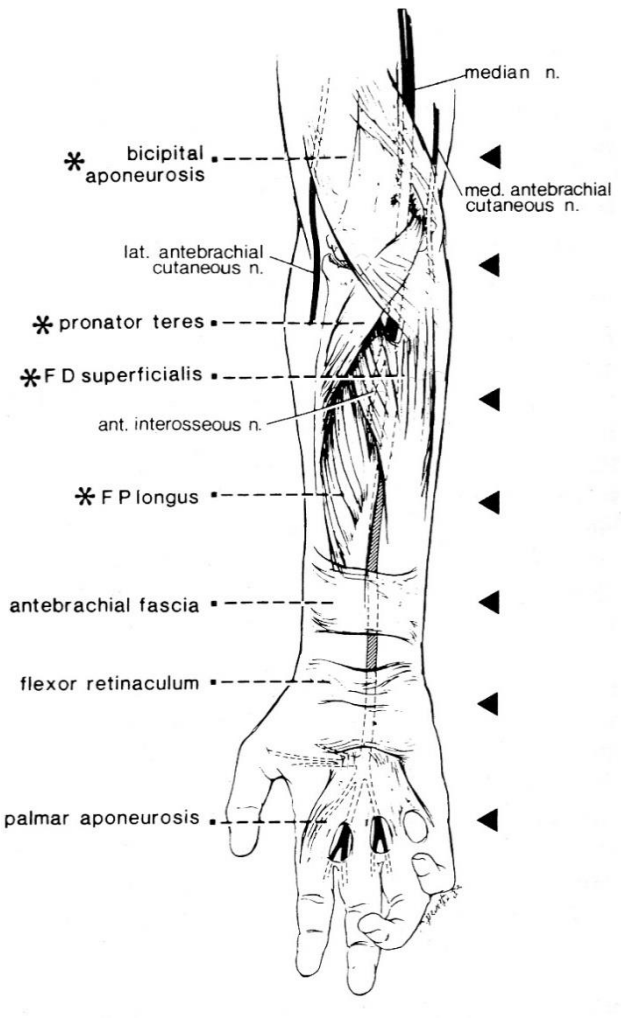
N. MEDIANUS

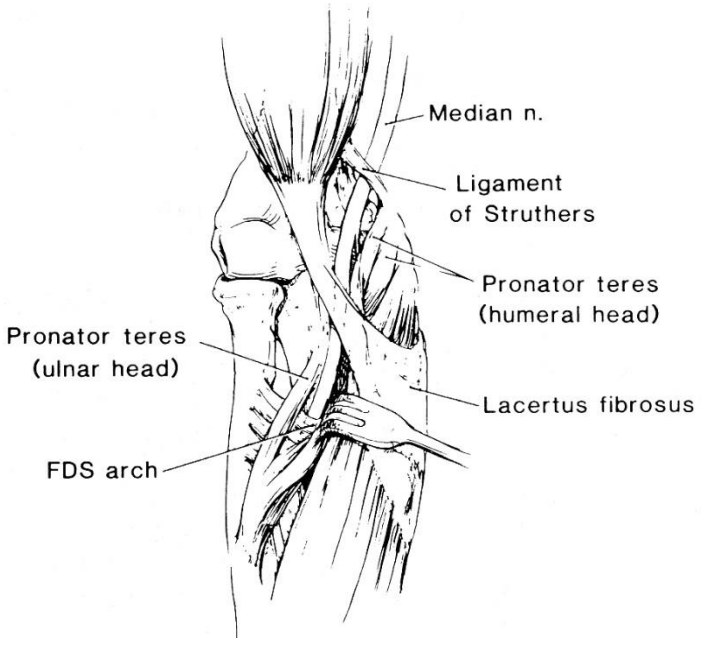
full anatomy of median nerve → see p. A20 (12) >>

PLACES OF COMPRESSION

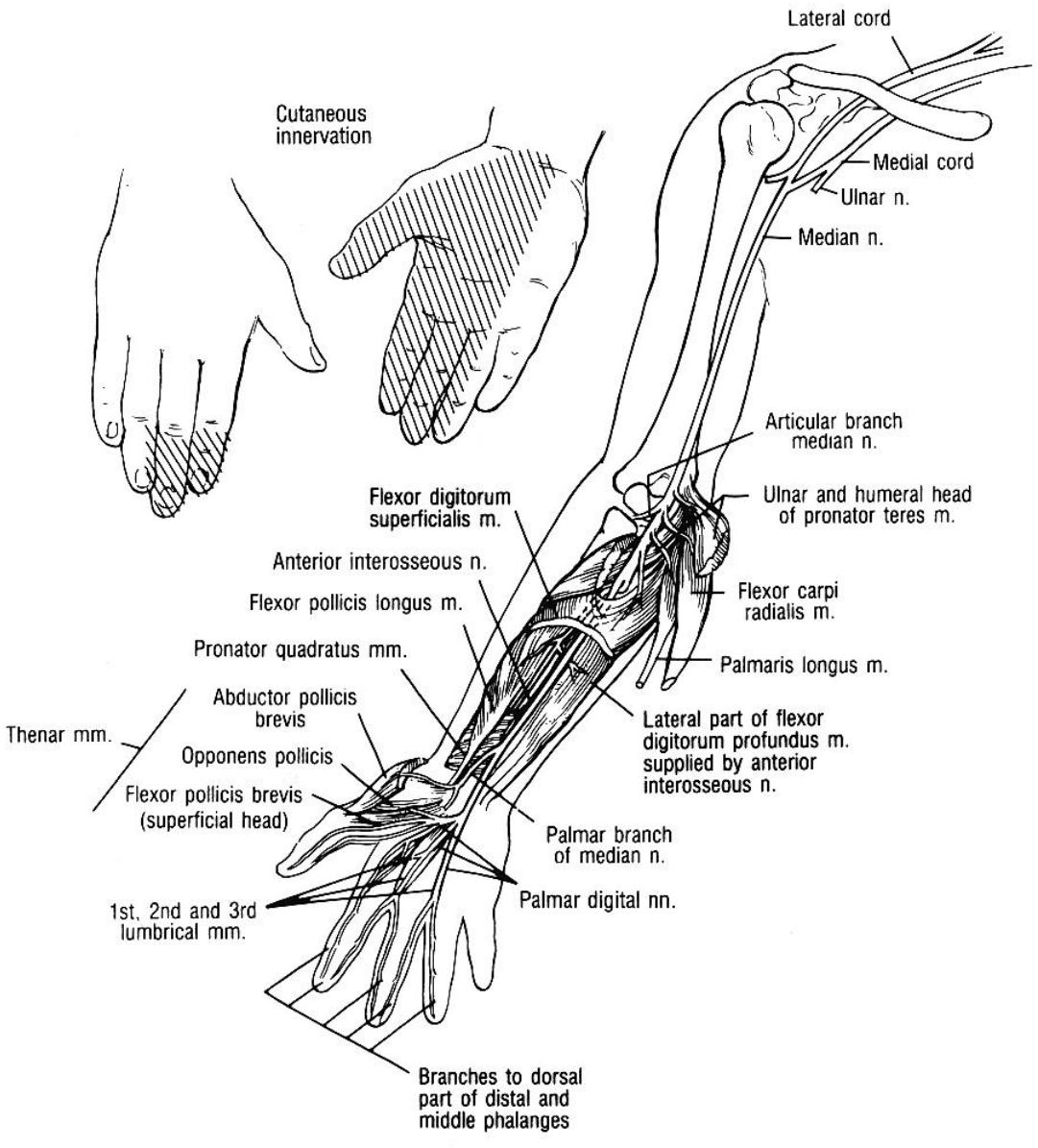
1. ***Within carpal canal*** (**carpal tunnel syndrome**); anatomy → *see below >>*

2. ***Near elbow*** (**proximal median neuropathy**):
    1) ligament of Struthers / supracondylar process of humerus – those are abnormal structures (vs. Struthers arcade)
    2) lacertus fibrosus (bicipital aponeurosis)
    3) between two heads of hypertrophied pronator teres (pronator teres syndrome)
    4) flexor digitorum superficialis fascial arch (sublimis bridge)
    5) direct external compression ("honeymoon palsy"), needle injury during cubital phlebotomy



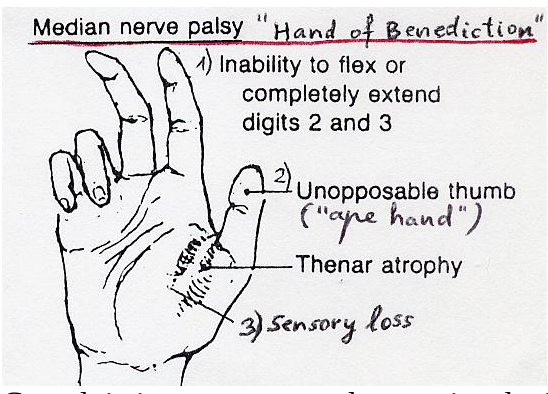


Source of pictures: David C. Sabiston "Sabiston Textbook of Surgery: the Biological Basis of Modern Surgical Practice", 15th ed. (1997); W.B. Saunders Company; ISBN-13: 978-0721658872 >>



Source of picture: Paul W. Roberts "Useful Procedures in Medical Practice" (1986); Lea & Febiger; ISBN-13: 978-0812109856 >>

- upon attempt to make a fist (only flexor digitorum profundus IV-V works – ulnar nerve):



Causalgia is most commonly associated with lesions of median nerve!

N.B. "benediction" vs "claw" – depends what you are asking patient to do!

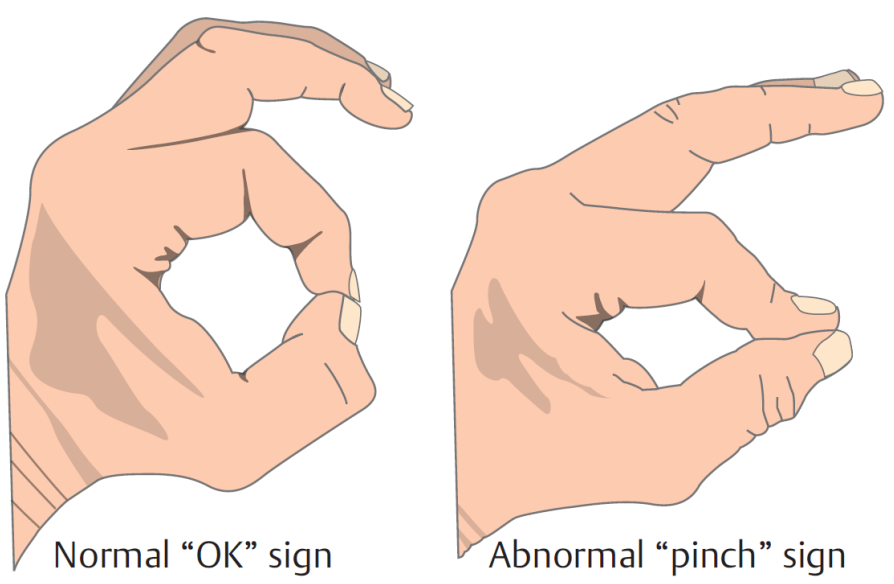
ANTERIOR INTEROSSEOUS NEUROPATHY

- **purely motor branch** of median nerve that arises in upper forearm (same as PIN of radial nerve)

No sensory loss + weakness of 3 forearm muscles:

1) flexor digitorum profundus (FDP) - flexion of distal phalanx of digits 2 & 3
2) flexor pollicis longus (FPL): flexion of distal phalanx of thumb
3) pronator quadratus (in the distal forearm): difficult to isolate clinically (H: EMG)

Summary – distal phalanx of I-III digits → abnormal "OK" sign:



Important to evaluate pronator teres (abnormalities suggest involvement more proximal than forearm)!

MANAGEMENT

- in absence of identifiable cause of nerve injury, expectant management is recommended for 8–12 weeks, following which exploration is indicated, which may reveal a constricting band near the origin.

CARPAL TUNNEL SYNDROME (CTS)

- most common compressive neuropathy!

- PREVALENCE: 3% in women and 2% in men
- PEAK PREVALENCE - women > 55 years.
- 50% ***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), osteoarthritis, 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** (esp. **hemodialysis** - deposition of β-microglobulin derived amyloid, vascular steal from AV fistula)
10) **anatomic predispositions**: persistent median artery, anomalous tendons or muscles, congenital stenosis of carpal tunnel

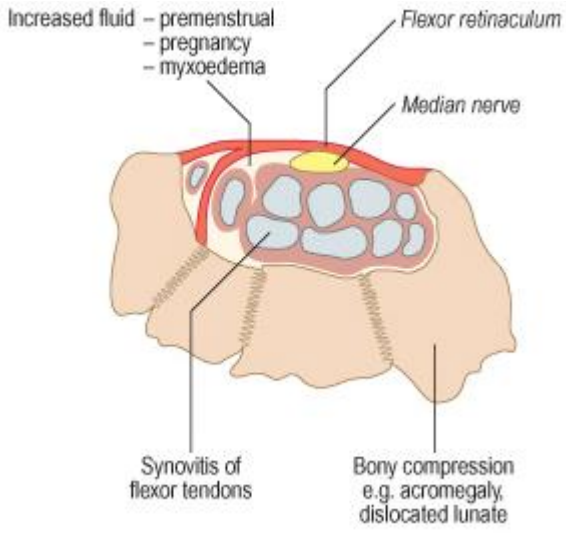


Table 30.1 Pressure within carpal tunnel

Pressure (mm Hg)	Description
<20	normal
20–30	venular flow retarded
30	axonal transport impaired
40	sensory & motor dysfunction
60–80	blood flow ceases

CLINICAL FEATURES

Referred pain with entrapment neuropathy can ***radiate proximally*** - to the arm and even neck! (mimics C6-7 radiculopathy)

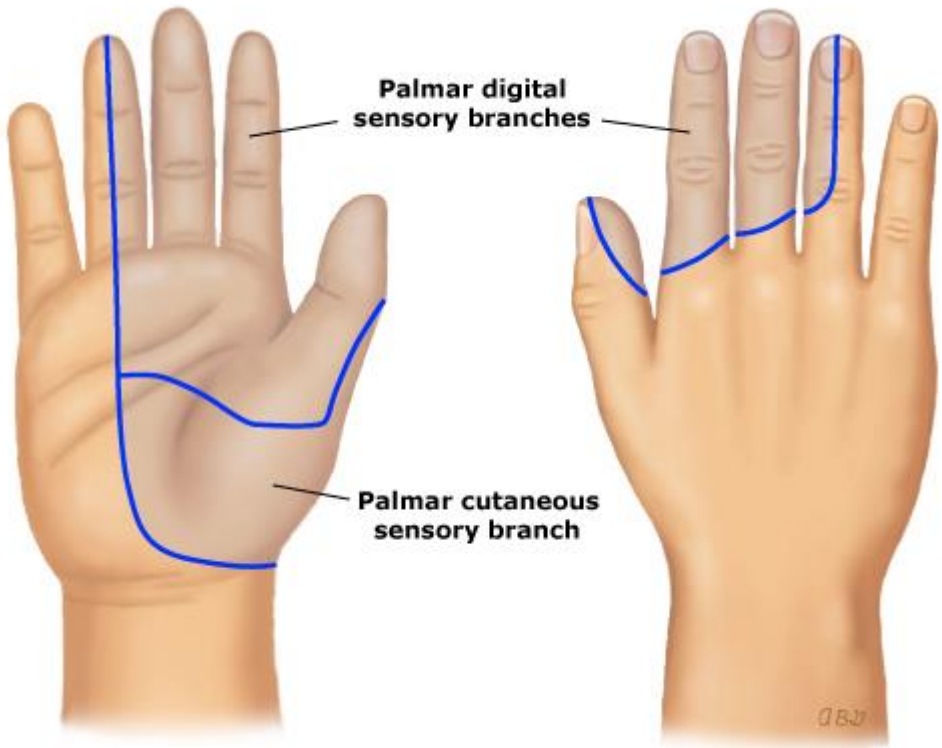
Mild disease: **paresthesias** & **pain** in median nerve distribution (after strenuous wrist movements or nocturnal*).

*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 with painful "hand falling asleep"); exacerbated by hand elevation.
  - patients often seek relief by: shaking or dangling or swinging the hand, opening and closing or rubbing the fingers, running hot or cold water over the hand, or pacing the floor
- sometimes pain radiates *proximally* to forearm or even 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).

Median palmar cutaneous nerve arises from radial side of median nerve approximately 5 cm proximal to TCL and travels superficial* to carpal tunnel to provide sensory innervation to thenar eminence.

*thus, preserved in carpal tunnel syndrome!



Carpal Tunnel Syndrome

Section through wrist at distal row of carpal bones shows carpal tunnel. Increase in size of tunnel structures caused by edema (trauma), inflammation (rheumatoid disease), ganglion, amyloid deposits or diabetic neuropathy may compress median nerve

More severe disease: **sensory loss & weakness** (with THENAR **atrophy***):

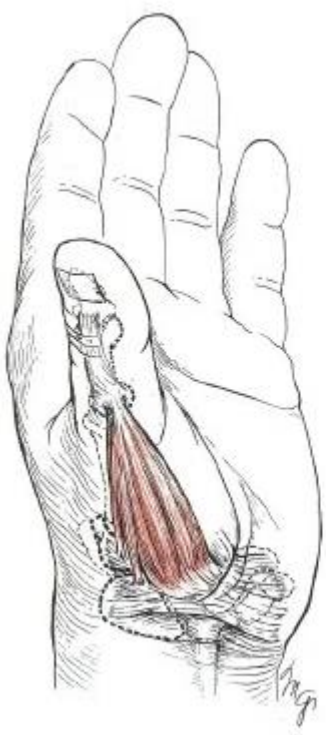
*may be absent in patients with Riche-Cannieu anastomosis. see p. A20 (12) >>

- hand grip weakness, especially opening the jar.
- hand clumsiness is more related to numbness than motor deficit

1) **opponens pollicis** – ability of thumb to move toward little finger against resistance:

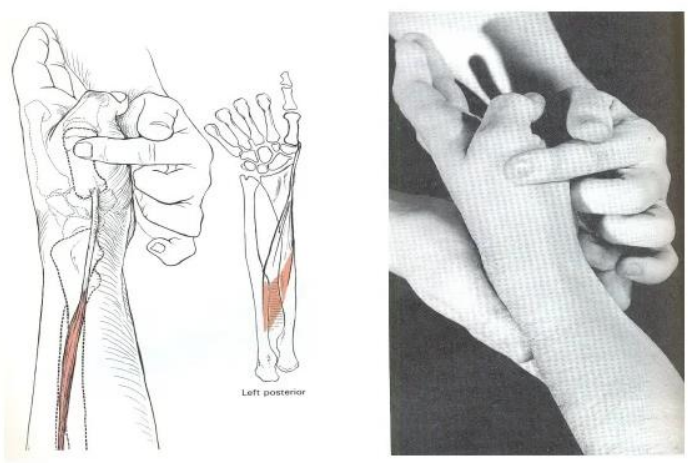
Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

2) **abductor pollicis brevis (APB)** (most reliably affected muscle!) – ability to lift thumb proximal phalanx against resistance:



quantitative testing:

vs. abductor pollicis longus (***radial n.***):



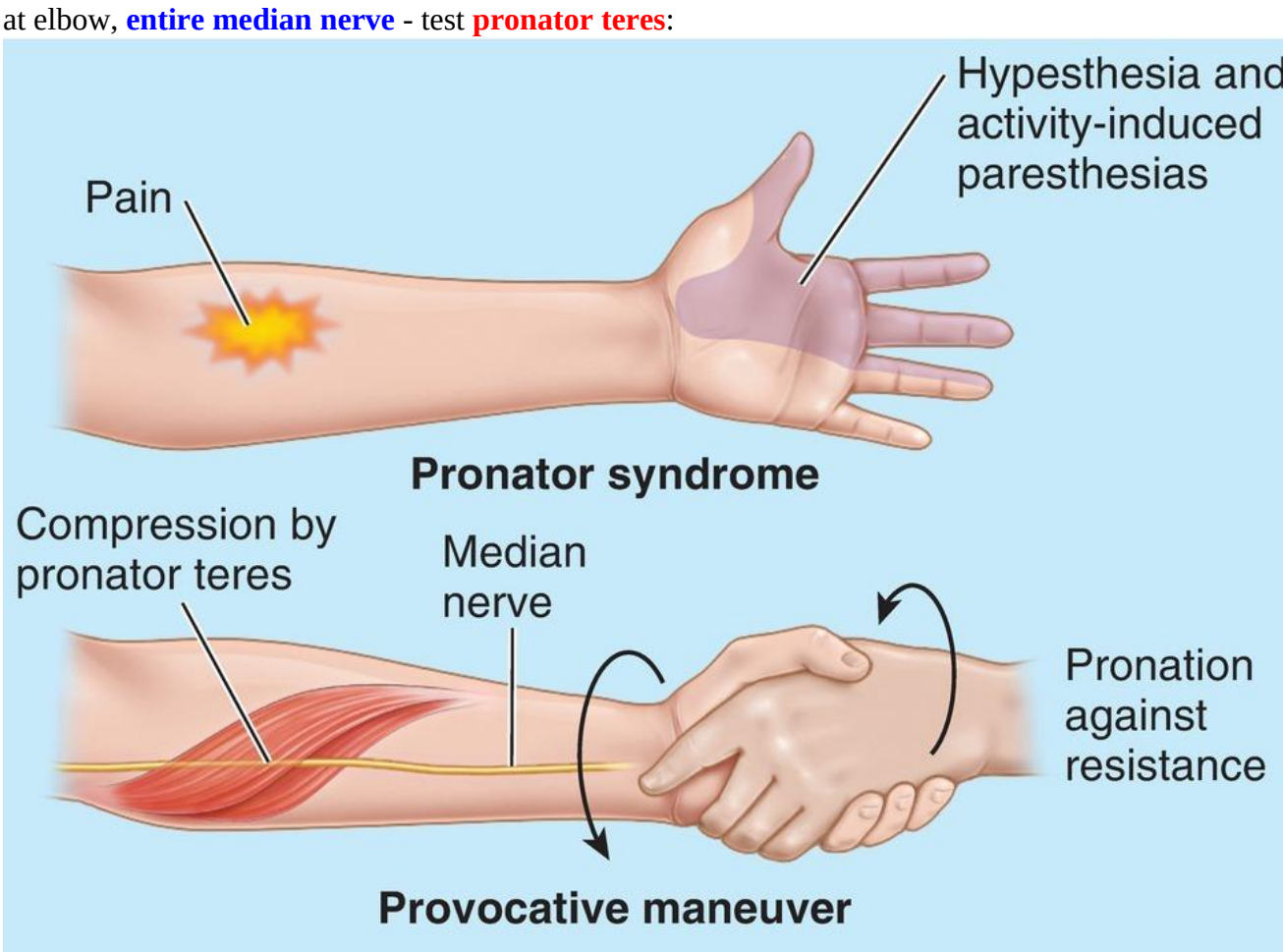
No weakness in more proximal muscles (difference from radiculopathy)

vs.

Proximal median neuropathy – tenderness along nerve course, motor deficit more widespread:

below elbow, only **AIN** branch (test distal I-II finger flexion - "OK" sign).

at elbow, **entire median nerve** - test **pronator teres**:



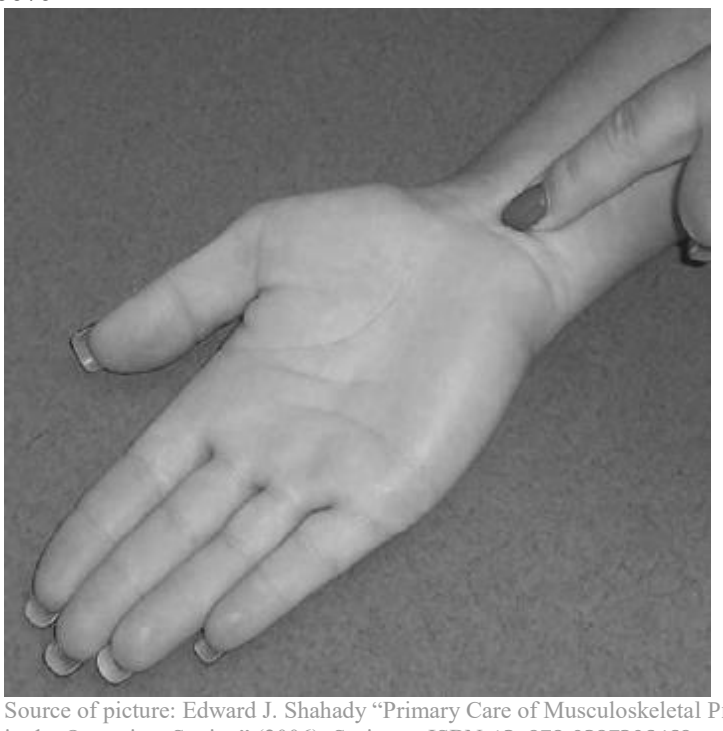
Location	Muscles Affected	Action	Sensory Loss
At wrist	Abductor pollicis	Abduction	Palmar and dorsal surfaces of thumb, index, middle fingers
	Opponens pollicis	Opposition	
Near elbow (pronator syndrome)	Abductor pollicis	Abduction	Palm, palmar and dorsal surfaces of thumb, index, middle fingers (no loss on forearm)
	Opponens pollicis	Opposition	
	Pronator quadratus	Pronation	
	Pronator teres	Pronation	
	Flexor pollicis longus	Flex thumb, distal joints	
	Flexor digitorum superficialis sublimis bridge	Flex fingers	
	Flexor digitorum profundus	Flex fingers, median side	
	Flexor carpi radialis	Wrist flexion	
	Lumbricales (two radial)	Extend MP joint	
	Flexor pollicis longus	Flex thumb, distal joint	None

Below elbow (anterior interosseus branch)	Flexor digitorum profundus II	Flex index finger, distal joint	

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 ≈ 50-60%



Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

2) **PHALEN sign** (hold forcedly patient's wrist in acute 90 degree flexion for 30-60 seconds → paresthesias; sensitivity 80%):

Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

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) **sensory nerve conduction slowing** (> 3.7 msec) across carpal tunnel (focal ***demyelination***; rarely can progress to axonal loss)

   Palmar sensory latency (stimulating sensory fibers in palm and recording over wrist) is most sensitive test!!!

   Distal motor latency may be normal in 25% of patients!!!, i.e. sensory latencies are more sensitive than motor.

   - electrodiagnostic studies are also helpful in ***grading severity*** of CTS:
     mild: prolonged (relative or absolute) median nerve sensory latencies.
     moderate: + prolongation of motor distal latency.
     severe: + axonal loss

Table 30.2 Distal conduction latencies through *carpal tunnel*[a]

Degree of involvement[b]	Sensory		Motor	
	latency[c] (mSec)	amplitude (mcV)	latency[d] (mSec)	amplitude (mV)
normal	<3.7	>25	<4.5	>4
mild[b]	3.7–4.0		4.4–6.9	
moderate[b]	4.1–5.0		7.0–9.9	
severe[b]	>5 or unobtainable		>10	

[a]assumes normal proximal NCV
[b]severity does not reliably correlate with latency (see text)
[c]to index finger. Sensory latency is measured to the peak of the waveform
[d]to abductor pollicis brevis

   - for uncertain cases compare median nerve sensory conduction velocity to that of the ulnar nerve (or radial nerve): normal median nerve should be at least 4 m/sec faster than the ulnar; reversal of this pattern suggests median nerve injury.

6) **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).

7) **MRI** of wrist (very sensitive test but only indicated if mass is suspected) – nerve thickening, increased signal intensity within inflamed peripheral nerve.

   - MRI is not cost-effective but may be useful in complicated cases.

8) **ultrasonography** with 18 MHz probe (highly sensitive and specific even in patients with negative electrodiagnostic studies) - entrapped peripheral nerve may appear hypoechoic, swollen, or flattened.

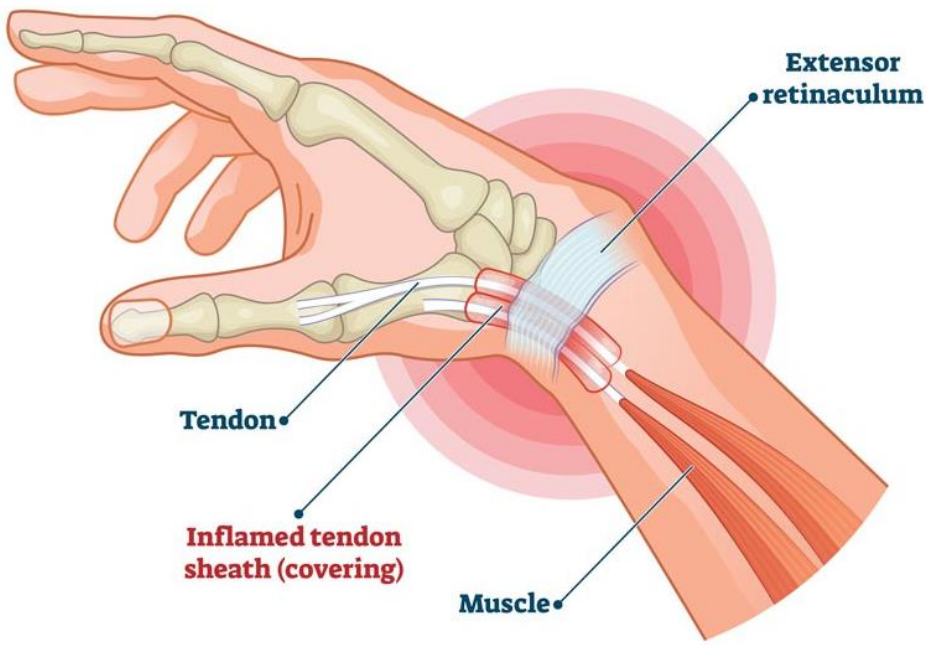
9) lab tests (thyroid, DM, uremia, multiple myeloma) – if systemic disease suspected

DIFFERENTIAL

1) Main mimicker – C6 radiculopathy! CTS pain sometimes radiates *proximally* to forearm or even shoulder

   Double-crush syndrome – C6 root compression may interrupt axoplasmic flow and predispose nerve to compressive injury at carpal tunnel

2) **de Quervain's syndrome** - tenosynovitis of the abductor pollicis longus and extensor pollicis brevis tendons often caused by repetitive hand movements
   - NCVs normal
   - pain and tenderness in wrist near the thumb.
   - **Finkelstein's test**: thumb is passively abducted while thumb abductors are palpated, positive if this aggravates pain.
   - H: wrist splints and/or steroid injections.



TREATMENT

American Association of Orthopedic Surgeons (AAOS) Clinical Practice Guideline (2010) endorsed by AANS, CNS, American Society of Plastic Surgeons, American Academy of PM&R and AANEM
Grade C, Level V: course of non-operative treatment is an option in patients diagnosed with CTS. Early surgery is an option when there is clinical evidence of median nerve denervation or the patient elects to proceed directly to surgical treatment.
Grade B, Level I and II: another non-operative treatment or surgery is suggested when the current treatment fails to resolve the symptoms within 2–7 weeks.
Insufficient evidence to provide specific treatment recommendations for CTS when found in association with diabetes*, coexisting cervical radiculopathy, hypothyroidism, polyneuropathy, pregnancy, rheumatoid arthritis, and CTS in the workplace.
Management specifics:
- local steroid injection or splinting is suggested before considering surgery (***Grade B, Level I and II***).
- oral steroids or ultrasound are options (***Grade C, Level II***).
- carpal tunnel release is recommended (***Grade A, level I***)

*notwithstanding the AAOS recommendations, multiple studies report that the results of carpal tunnel release in diabetics are good even when polyneuropathy is present.

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

1. **Ergonomic** corrections (do not return to heavy manual labor) and **rest**
2. **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 (usually results seen within few days; try at least for 4 weeks):

Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

   – **hand–wrist exercises** and **ultrasound** do not provide additional benefit beyond that offered by splinting alone.
3. **Ultrasound** therapy
4. Injection of depot **corticosteroids** into carpal tunnel (medial to m. palmaris longus tendon, just proximal to distal wrist crease) - significant, but temporary improvement:

   N.B. aim to inject **tendon sheaths**; injection adjacent or into nerve is to be avoided! – all *steroids are neurotoxic* upon intrafascicular injection, and so are some of the carrier agents!
   - 10–25 mg HYDROCORTISONE.
   - *avoid local anesthetics* (may mask symptoms of intra-neural injection)
   - 3-cc syringe with 25G 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:

Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

   - appropriate needle location can be assessed by moving ring (IV) 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.

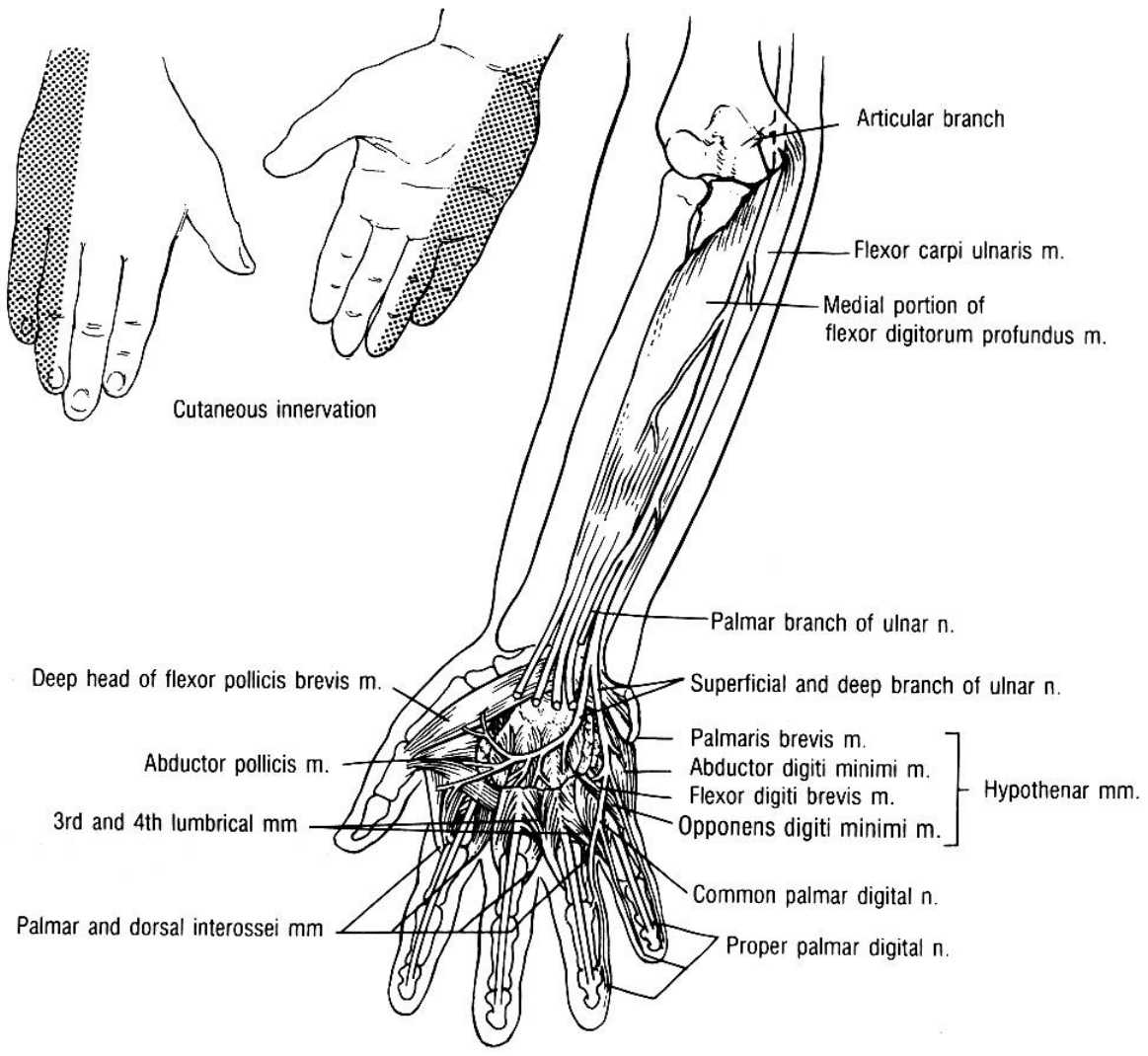
- advice patient that there will be some mild soreness and it may require 24 h to feel full effect.
- symptoms improve in > 75% of patients; 33% relapse within 15 mos (repeat injections are possible, but most clinicians limit to 3/year).

5. NSAIDs, diuretics, vit. B_6 – ineffective!

SURGERY

- see p. Op450 >>

N. ULNARIS



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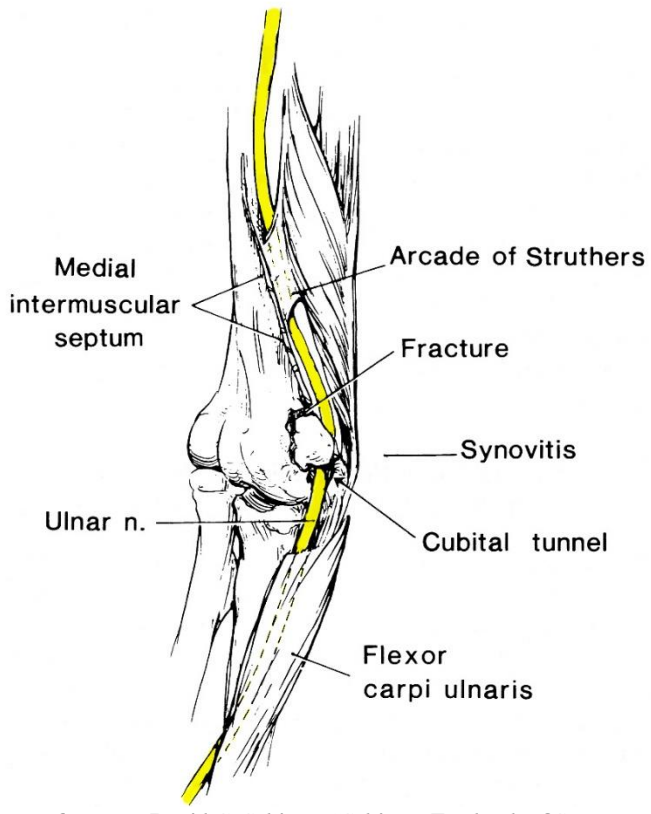
For anatomy – see p. A20 (10-11) >>

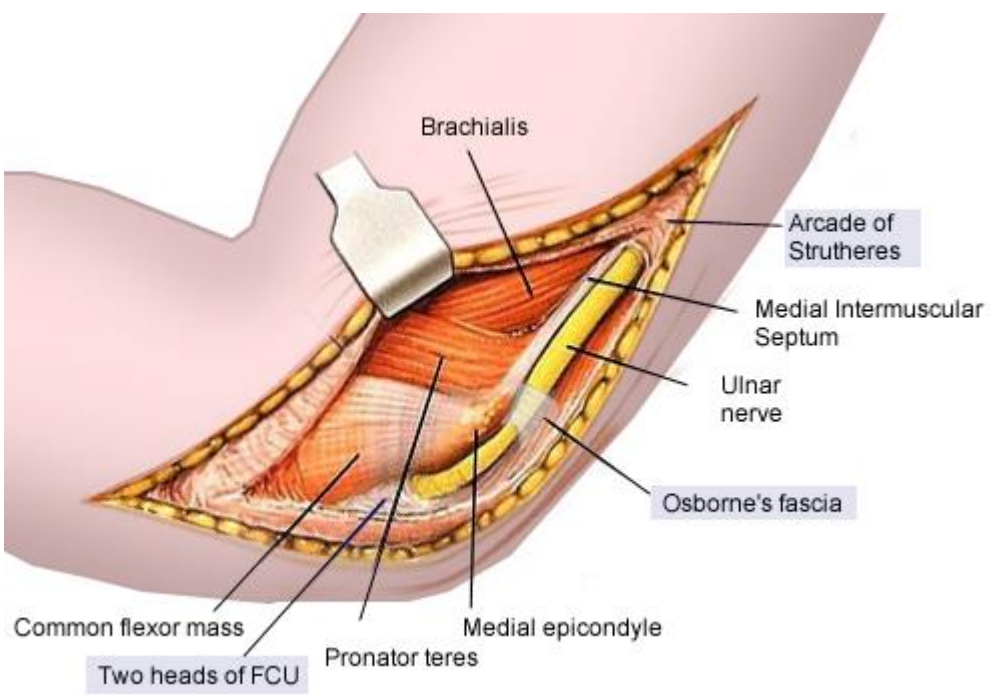
N. ULNARIS AT ELBOW

Places of compression (proximal to distal):

1. **Medial intermuscular septum** - sharp edge that can indent nerve (esp. after anterior transposition where nerve may be kinked).
2. **Arcade of STRUTHERS** (hiatus 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.
3. **ULNAR GROOVE (S. RETRO-EPICONDYLAR GROOVE)** - between the medial epicondyle and olecranon process - compression by fascia or by dynamic compression or repetitive trauma or during anesthesia (most common *anesthesia-related* compressive neuropathy!!!) or prolonged resting of elbow on hard surface.
4. **CUBITAL TUNNEL** just distal to the ulnar groove (e.g. cubitus valgus, medial condyle fracture, RA synovitis, osteophytes) – compression between cubital tunnel retinaculum (**OSBORNE'S ligament**) and ***medial collateral ligament (MCL)***.
5. **Between two heads of flexor carpi ulnaris** (aponeurosis of flexor carpi ulnaris also referred to as **OSBORNE'S fascia**; 3-5 cm distal to cubital tunnel) – e.g. in pianists (repeated forceful wrist flexion).



Source of picture: David C. Sabiston "Sabiston Textbook of Surgery: the Biological Basis of Modern Surgical Practice", 15th ed. (1997); W.B. Saunders Company; ISBN-13: 978-0721658872 >>



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** - little finger and ulnar half of the ring finger; exacerbating activities include:

N.B. sensory only IV-V fingers (vs. C8 – also ulnar forearm)

   - peripheral nerves have much more precise sensory and motor borders (vs. radiculopathies): sensory loss at V and ulnar half of IV digit – ulnar neuropathy (not C8 radiculopathy).
   - cell phone use (excessive flexion)
   - sleeping with elbow in flexion → nocturnal paresthesia and pain.

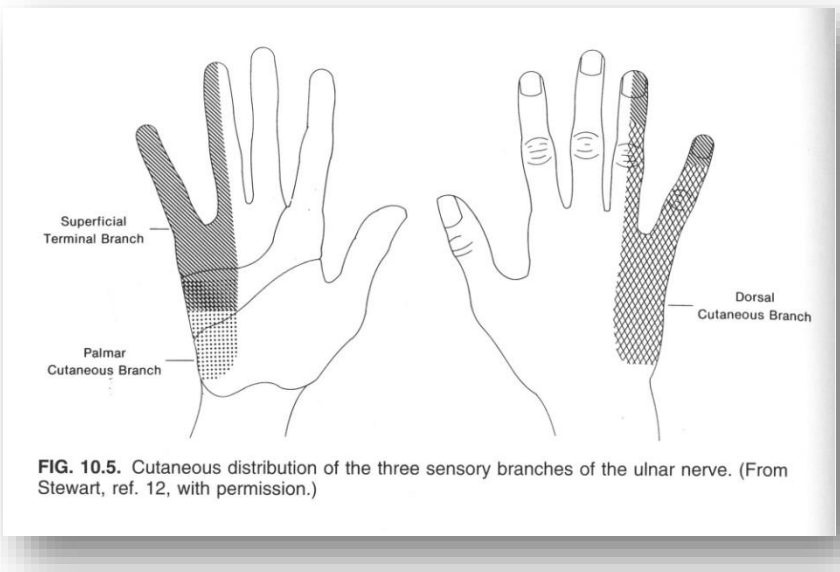
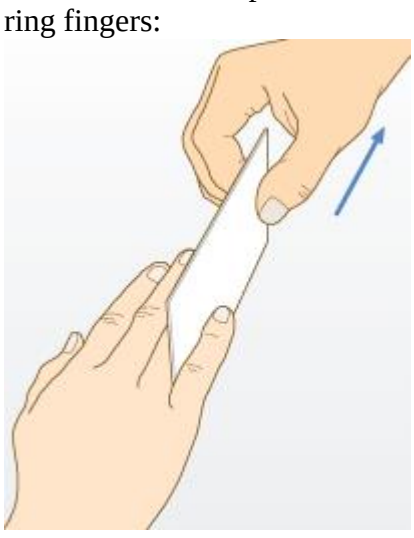


FIG. 10.5. Cutaneous distribution of the three sensory branches of the ulnar nerve. (From Stewart, ref. 12, with permission.)

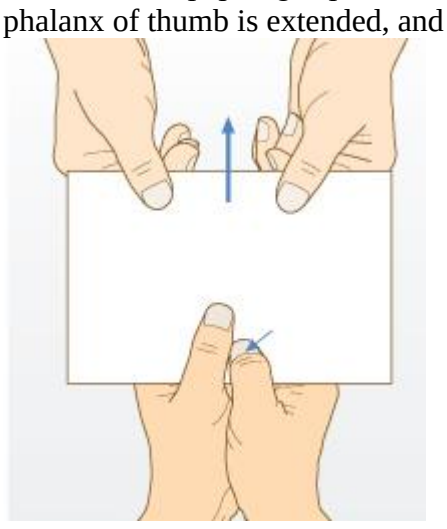
2) attempt to extend fingers → only MCP IV-V joints extend, IP joints do not extend - **"claw hand" (main en griffe)**; hand clumsiness, dropping objects; hypothenar + interossei **weakness** and **atrophy**. see p. D1 >>

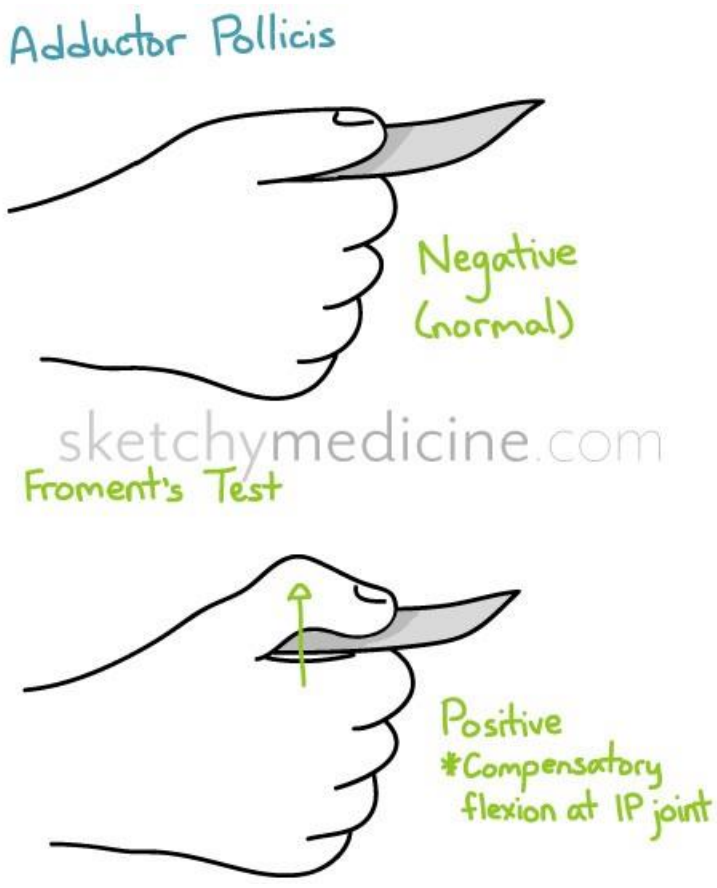
N.B. "benediction" (median) vs "claw" (ulnar) – depends what you are asking patient to do!

   - ulnar neuropathy may start with motor signs before sensory (opposite to CTS) - because of predominance of motor fibers within UN!
   - atrophy is most evident in the first dorsal interosseous (in thumb web space).
   - test *interossei*: ask patient to hold sheet of light card between fully extended little and ring fingers:



   - *m. adductor pollicis* weakness **FROMENT prehensile thumb sign (signe du journal)** - when sheet of paper, grasped between thumb and index finger, is pulled → proximal phalanx of thumb is extended, and distal phalanx is flexed:





– fifth finger may be abducted away from other fingers at rest (**Wartenberg sign**); patients complain of catching pinky finger when placing hand in pocket

A, Interosseous atrophy resulting in prominent metacarpal bones. **B,** Atrophy of the first dorsal interosseous muscle. **C,** Abduction at rest of the fifth digit (Wartenberg's sign).

- 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.

Location	Muscles Affected	Action	Sensory Loss
At elbow (cubital tunnel syndrome)	Flexor digitorum profundus V	Flexes little finger, distal joint	Medial side of hand and fingers to wrist crease
	Interossei	Adducts and abducts	
	Flexor pollicis brevis	Adducts thumbs	

Motor (differential from C8):

ulnar nerve innervates all intrinsic hand muscles, except LOAF (5 muscles) : *abductor - opponens - flexor pollicis brevis, and lateral two lumbricals* ← innervated by C8 and T1 (recurrent motor branch of median nerve);
ulnar nerve does not innervate *flexor digitorum superficialis* and *first two flexor digitorum profundus muscles* ← innervated by C8 (median nerve)

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
   N.B. in contrast to CTS, which is predominantly demyelinating, UNE has more ***axonal loss***! – surgery results worse than with CTR
   N.B. in contrast to CTS, ***motor NCS findings*** are more useful for localization for site of entrapment than sensory abnormalities!
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

DIFFERENTIAL

Referred pain with entrapment neuropathy can ***radiate proximally*** (mimics C8 radiculopathy)

N.B. *sensory testing of **dorsal** ulnar hand 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).

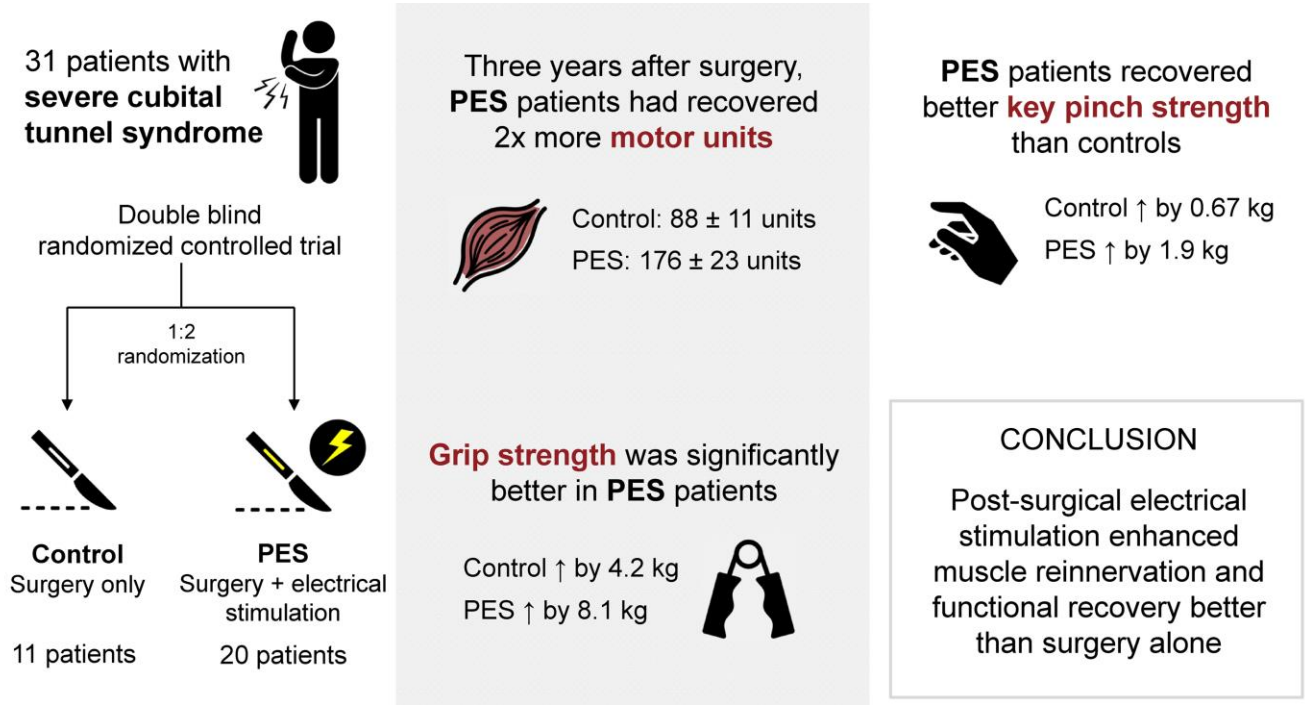
TREATMENT

No guidelines or consensus!

1. **Half-splint with elbow pad** (elbow in gentle extension) at nighttime ± daytime.
2. **NSAIDs**
   N.B. *steroid injections* have no role in treatment!
   If treating conservatively, follow patient at 1-2 month intervals as long as stable or improving; if worsening → surgery
3. **Surgery** – see p. Op450 >>

Postsurgical Electrical Stimulation Enhances Recovery

Postsurgical Electrical Stimulation Enhances Recovery Following Surgery for Severe Cubital Tunnel Syndrome: A Double-Blind Randomized Controlled Trial. Hollie A Power et al. Neurosurgery, Volume 86, Issue 6, June 2020, Pages 769–777



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.

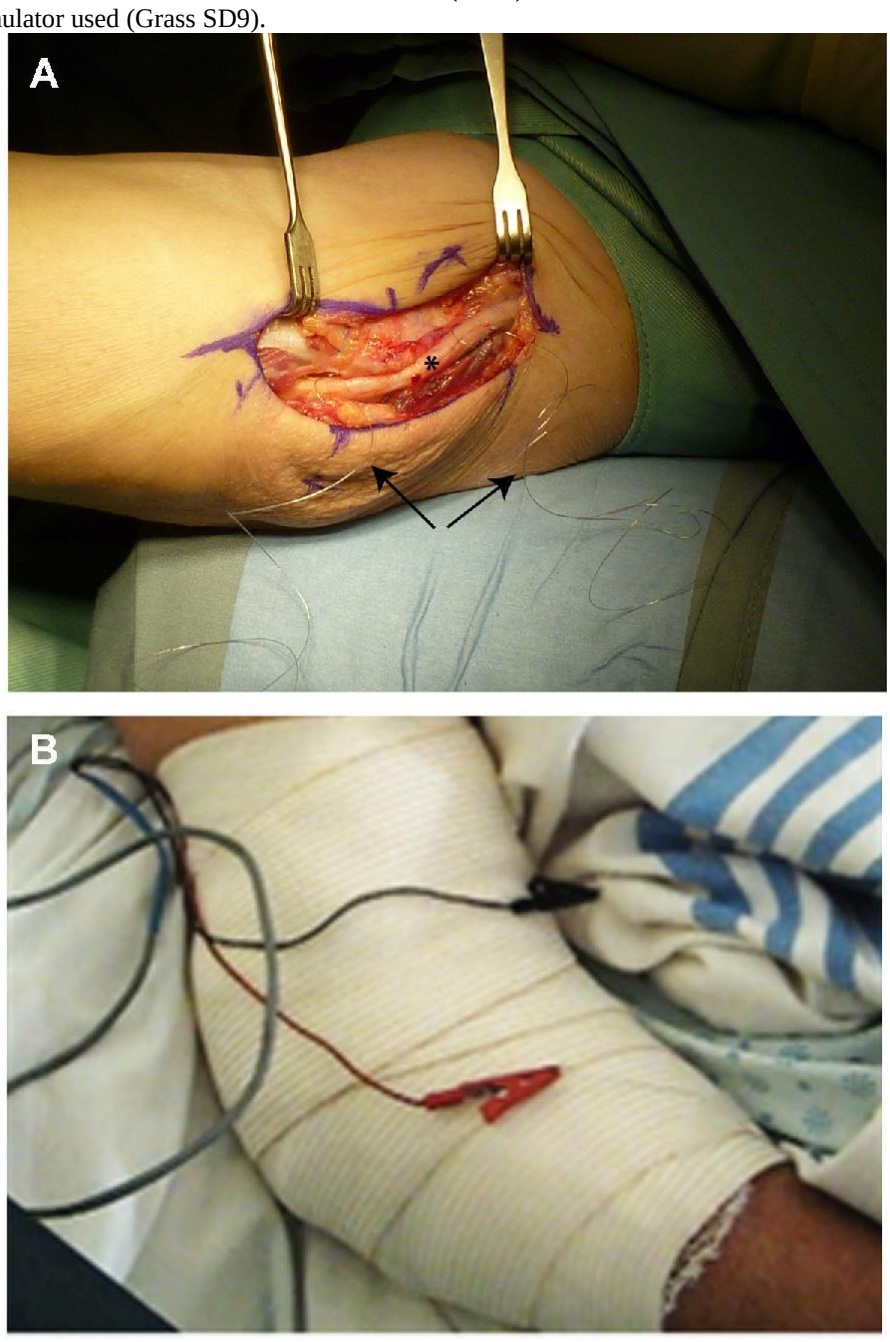
Stimulation group received **1 hour of stimulation** as a continuous **20-Hz** train of balanced biphasic pulses. The stimulation intensity was set at the tolerance limit (<30 V, 0.1 ms pulse duration).

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** (only 1% of all ulnar neuropathies):

1) paraplegics using hand crutches with horizontal bar across palm.
2) motorcyclists who firmly grasp hand bar control.
3) 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) >>

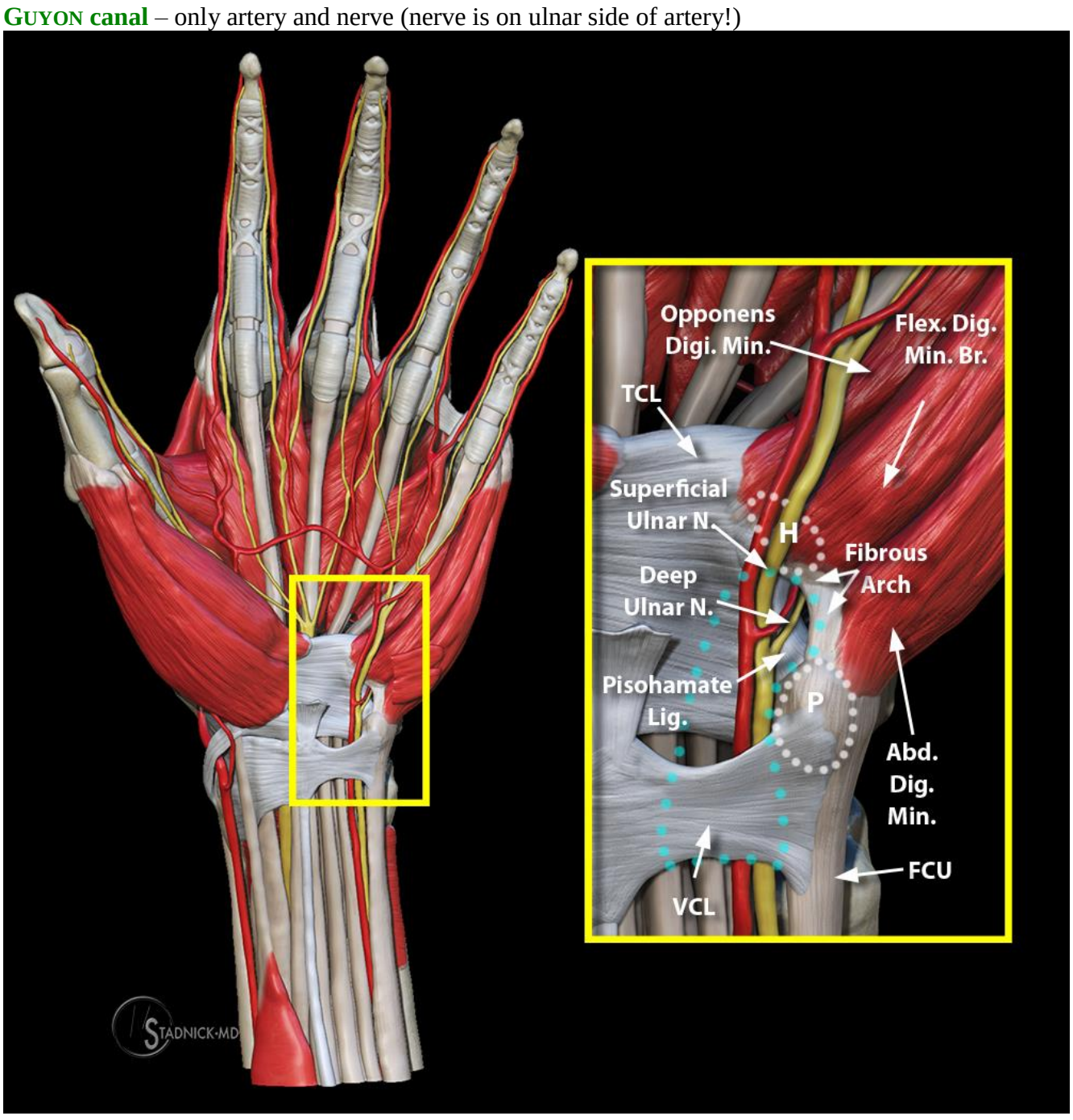
- ulnar nerve runs above flexor retinaculum (lateral to flexor carpi ulnaris tendon and medial to a. ulnaris).
- at proximal carpal bones, it dips between pisiform and hamate at entrance to Guyon canal, roofed over by extension of transverse carpal ligament between these 2 bones.
- superficial hypothenar sensory branch (hypothenar 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 hypothenar atrophy).
- in other 35% individuals, some *pain* and hypothenar *numbness* is expected.
- after entering Guyon canal, deep motor branch first supplies abductor digiti minimi (ADM), then crosses under one head of flexor digiti minimi (FDM), supplies this muscle, and crosses over to supply opponens digiti 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; opponens always is affected, together with interossei, ulnar lumbricales, and adductor pollicis.

Location	Muscles Affected	Action	Sensory Loss
At wrist	Interossei	Adducts and abducts	Palmar medial hand and finger
	Flexor pollicis brevis	Adducts thumb	
	Opponens V	Adducts little finger	

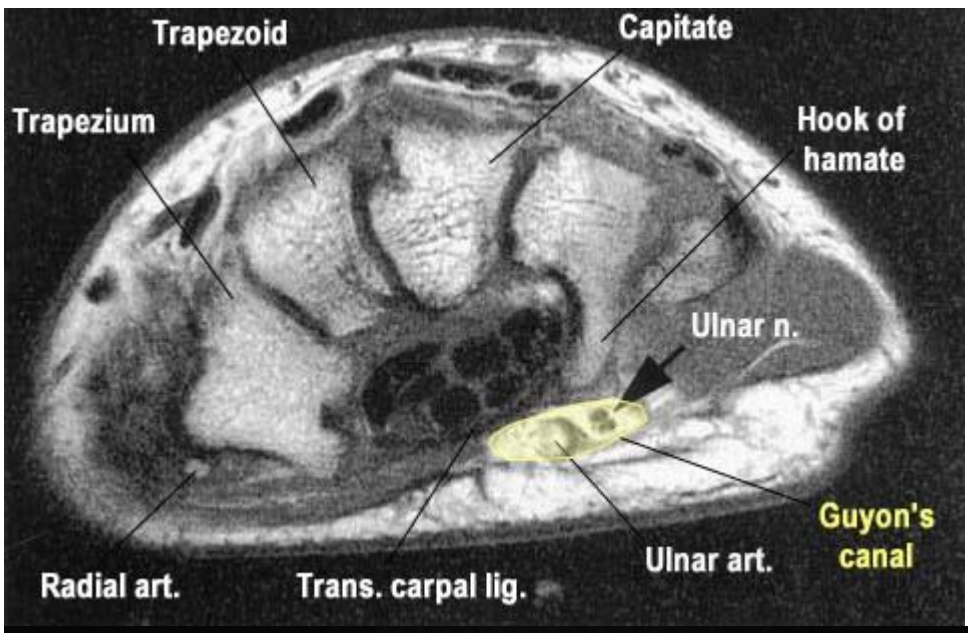
Wasted 1st dorsal interosseus:

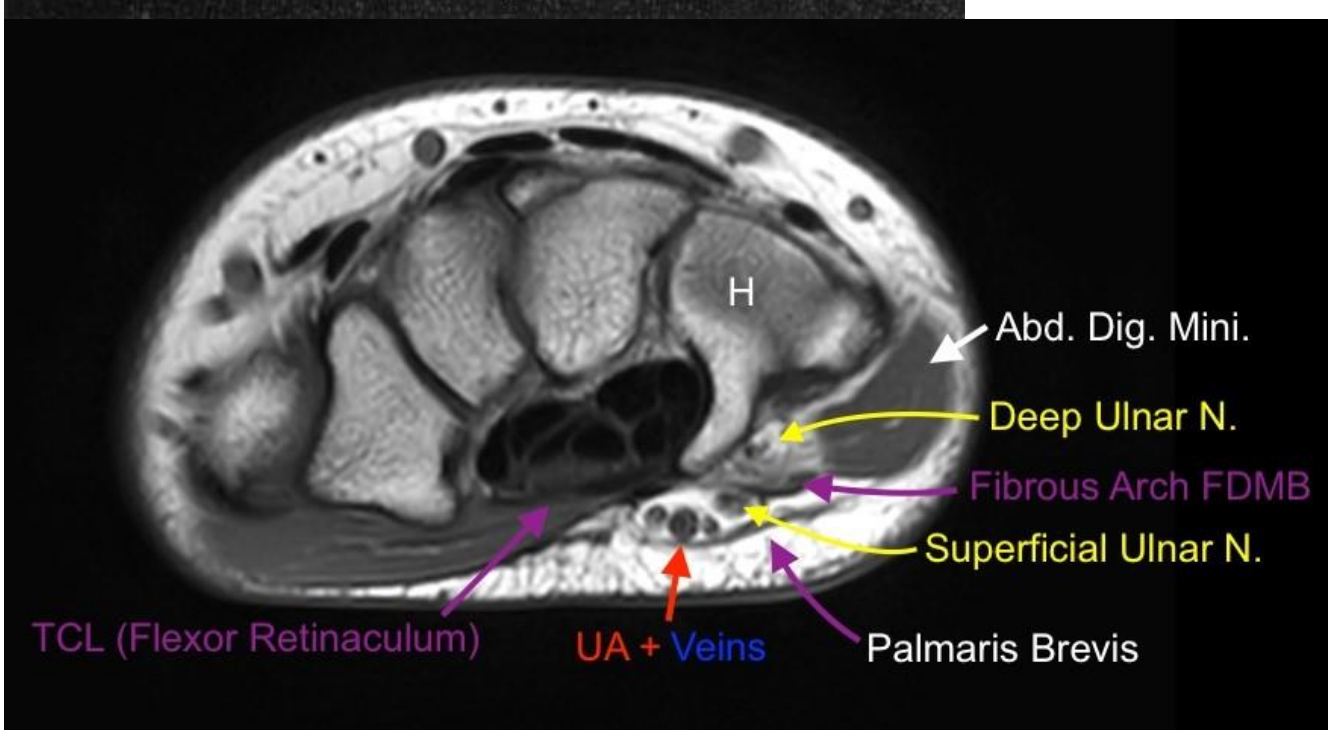
N.B. no sensory loss in dorsal ulnar side of hand!!!!
If no sensory loss at all – either ALS or only deep branch!

GUYON canal – only artery and nerve (nerve is on ulnar side of artery!)



Diagnosis – EDX and MRI:





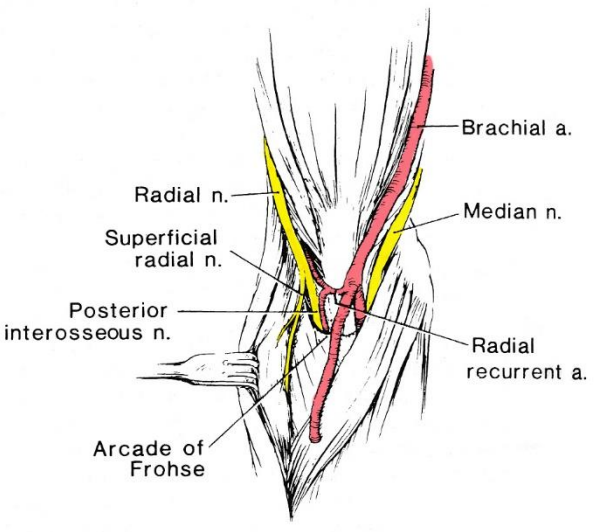
TREATMENT

- ***avoidance*** & use of ***palmar padding***.
- preop order MRI! (ganglion cyst?)
- ***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 PALSY.
2. HUMERUS SHAFT FRACTURES (***spiral groove*** between medial and lateral heads of triceps).
3. Underneath **arcade of FRÖHSE** (musculotendinous arcade, formed by upper free border of superficial head of m. supinator) → ***radial tunnel*** (under m. extensor carpi radialis, 3-4 cm distal to lateral epicondyle; within tunnel, nerve rests on deep head of m. supinator) - RADIAL TUNNEL (S. POSTERIOR INTEROSSEUS NERVE, PIN) SYNDROME; no sensory loss!

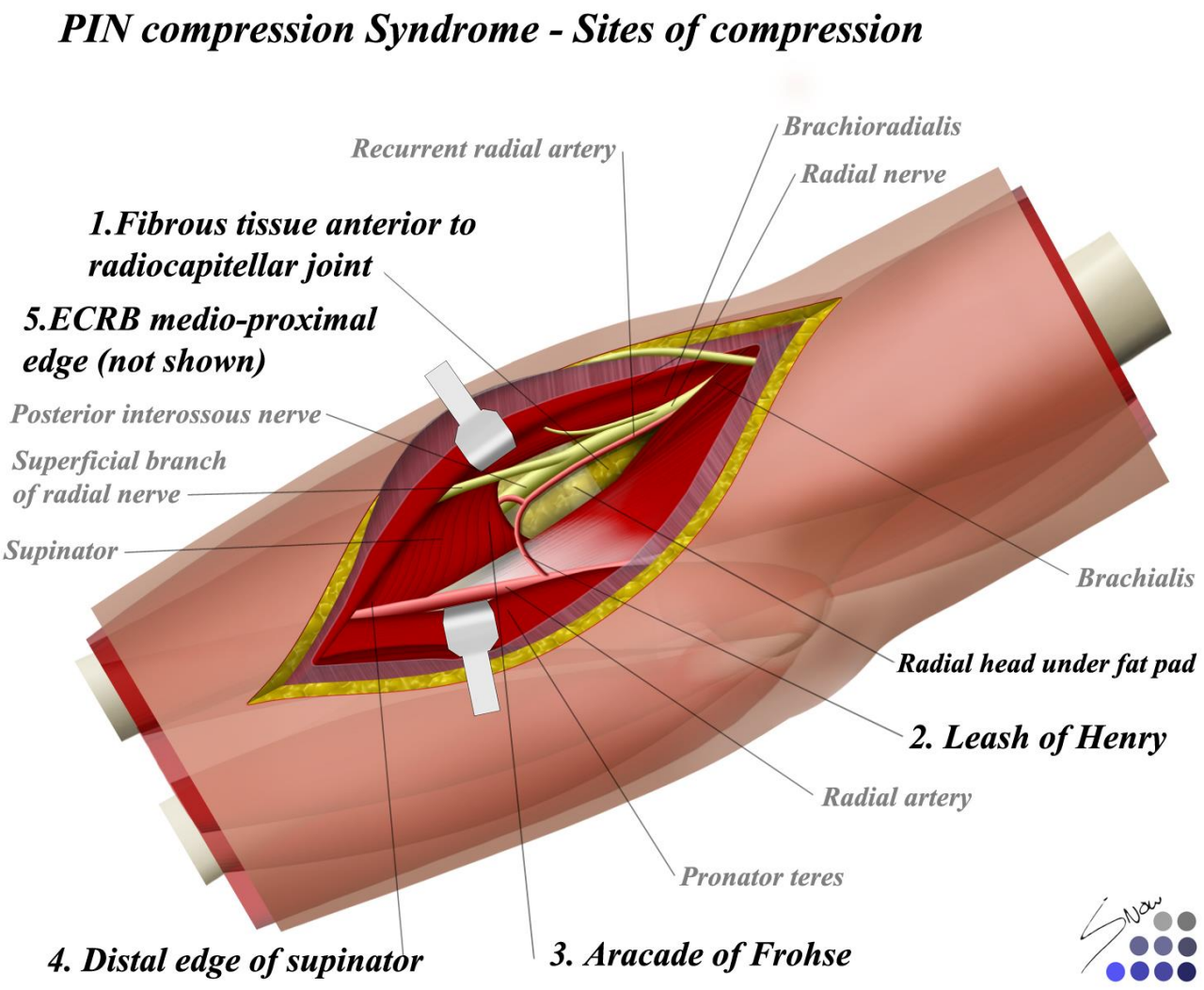


Source of picture: David C. Sabiston "Sabiston Textbook of Surgery: the Biological Basis of Modern Surgical Practice", 15th ed. (1997); W.B. Saunders Company; ISBN-13: 978-0721658872 >>

   - 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's 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. D1 >>
   1) **paralysis of finger extension at MCP joints** (IP joints extension – action of mm. lumbricales – ulnar and median nerves).
      - 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 by radial nerve*), wrist extension weakness usually is undetectable in spite of weakness of ulnar wrist extensor.
   2) *pseudo-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).

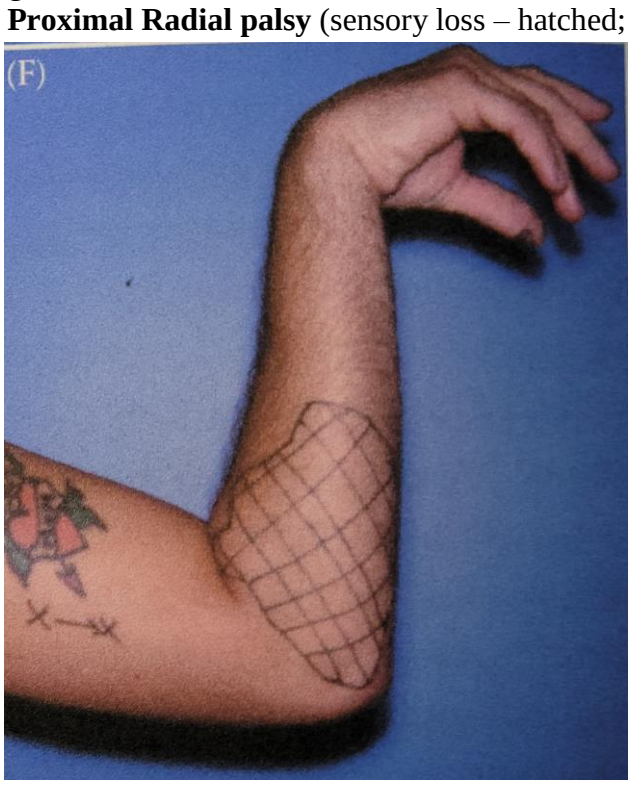
Location	Muscles Affected	Action	Sensory Loss
At elbow (POSTERIOR INTEROSSEUS SYNDROME)	Extensor carpi ulnaris	Extends wrist	None
	Extensor digitorum communis	Extends fingers	
	Extensor pollicis	Extends thumb	
	Abductor pollicis	Extends, abducts	
Below elbow (SENSORY SUPERFICIAL RADIAL BRANCH)	None		Lateral side of forearm and hand

*extensor carpi ulnaris – PIN; extensor carpi radialis – proximal radial nerve

RADIAL TUNNEL (S. POSTERIOR INTEROSSEUS NERVE, PIN) SYNDROME:
1) mm. extensor digitorum → ↓finger extension at MCP joints
2) m. extensor carpi ulnaris → wrist radial deviation (no wrist drop!) – wrist extension weakness in neutral position (but normal wrist extension in radial deviation – no need for extensor carpi ulnaris!)

PROXIMAL RADIAL NERVE – add wrist drop (at spiral groove), triceps weakness (proximal to spiral groove)

Attempt to extend wrist and fingers:
PIN palsy (no sensory loss): **Proximal Radial palsy** (sensory loss – hatched; x-x – Tinel area):



DIAGNOSIS

1) ***TINEL sign*** at radial tunnel.
2) **nerve conduction studies** - conduction block (locating exact site of compression).
3) **EMG**
4) **MRI**!

DIFFERENTIAL

1) lead poisoning - isolated wrist and finger extensor weakness (usually bilateral)
2) C7 radiculopathy: triceps will be weak
   N.B. takeoff of nerve to triceps is proximal to spiral groove

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 (PIN) SYNDROME (excellent outcome in 90-95% cases)

RADIAL TUNNEL SYNDROME is motor neuropathy - diagnosis mandates **surgical decompression**; conservative treatment has no place! (preop needs MRI, EDX – to rule out brachial plexitis, ect)

See p. Op450 >>

THORACIC OUTLET SYNDROME (TOS)

- **compression** of BRACHIAL PLEXUS or SUBCLAVIAN VESSELS in their passage from cervical and upper thoracic area toward axilla and proximal arm – between clavicle and 1st rib.

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 TOSs do not coexist!

- < 1% of all TOS cases

NEUROGENIC TOS

1. **TRUE (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 scalenus ant.)

Syndrome is very rare!

- compresses / irritates lower trunk of brachial plexus (C_8-T_1).

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) ***scalenus muscle spasm*** (*scalenus anticus syndrome*) – due to minor cervical or shoulder trauma – very controversial syndrome and surgery is controversial.
   2) ***abnormal shoulder posture***:
      a) "*droopy shoulder syndrome*" - tall, slender, and round-shouldered person.
      b) occupational arms *above head*.

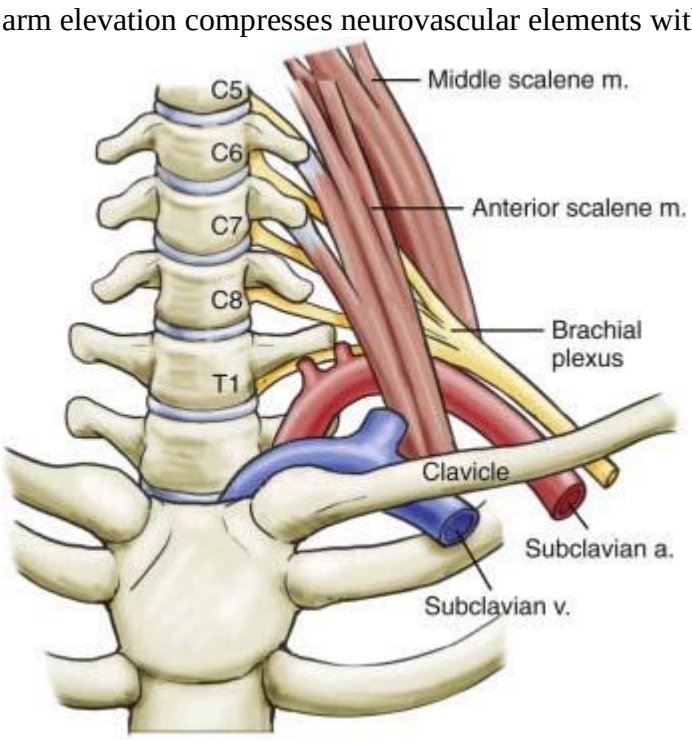
Three sites within thoracic outlet where neurovascular compression may occur – going from proximal to distal:

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** (middle third of clavicle anteriorly, first rib posteromedially, upper border of scapula posterolaterally) - immediately distal to interscalene triangle.

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

3. **SUBPECTORAL TUNNEL, S. SUBCORACOID SPACE, S. RETROPECTORALIS MINOR SPACE** (deep to the pectoralis minor tendon) - distal to costoclavicular space.

- arm elevation compresses neurovascular elements within subcoracoid space.



Source of picture: Huang JH, Zager EL. Thoracic outlet syndrome. Neurosurgery. 2004;55:897

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 ***C_8-T_1 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 (C_8-T_1 myotomes) → muscle atrophy
   - classic **GILLIATT-SUMNER hand** - dramatic atrophy in abductor pollicis brevis and lesser atrophy in interosseous 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.
3) ***vasomotor 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
- 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! (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!).

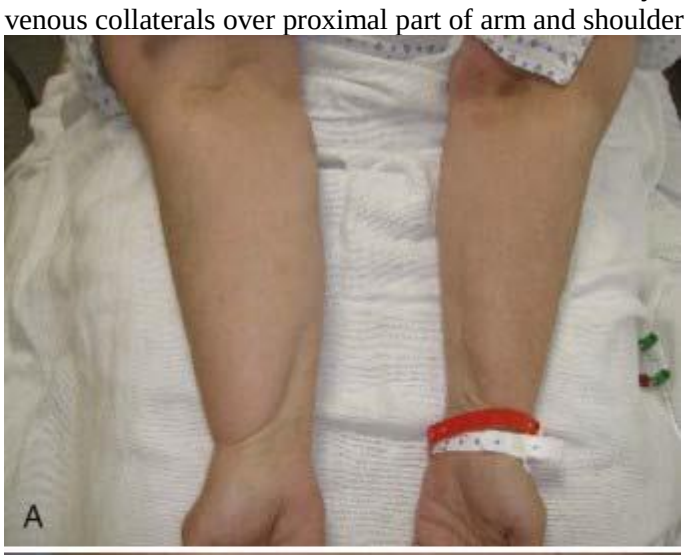
VASCULAR TOS

– ***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 SCHROTTER 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

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** - compression site and cause.
- **chest XR** – cervical rib + to rule out Pancoast tumor.

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

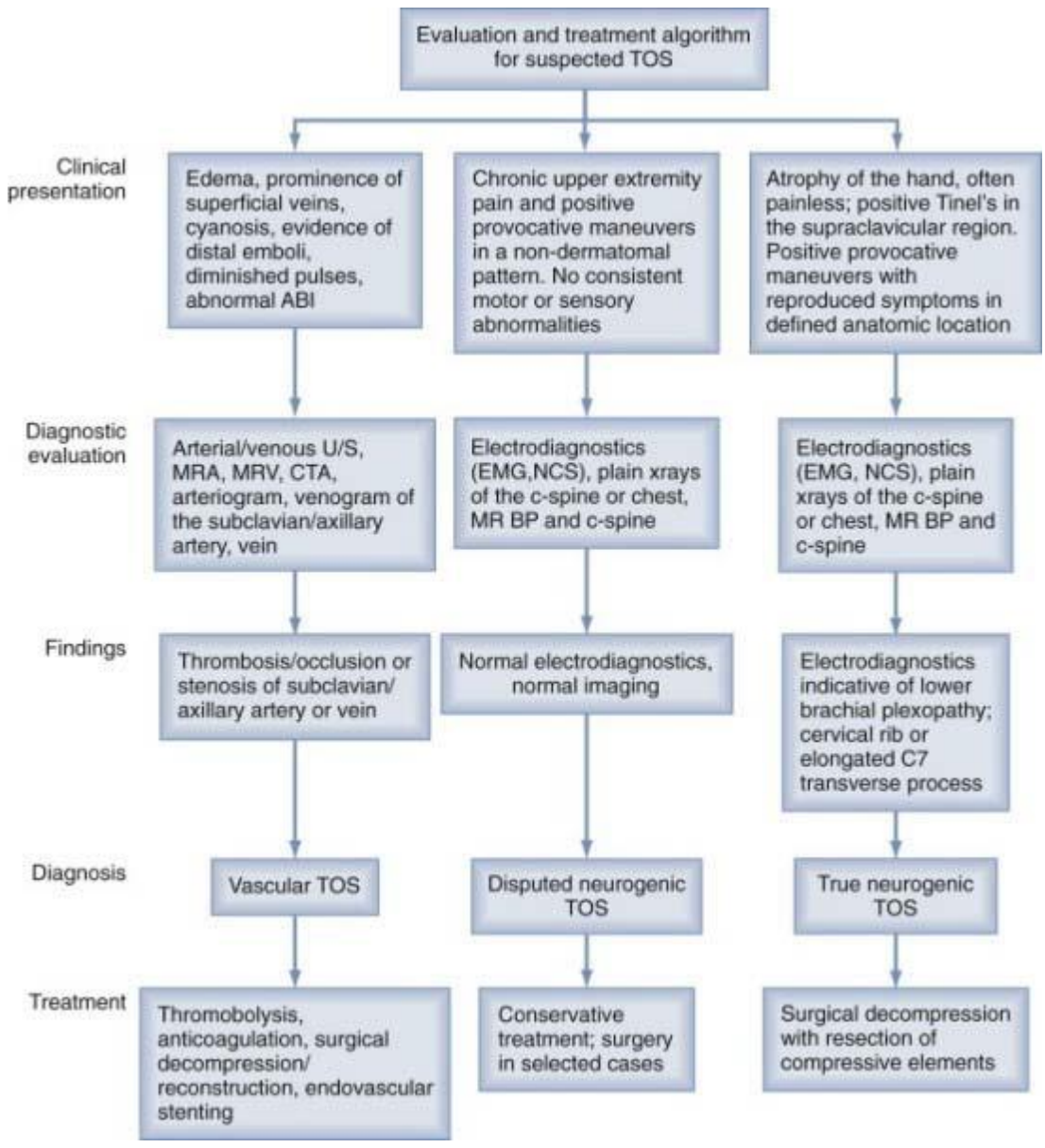
Orthopedic
- Shoulder abnormalities (rotator cuff injury)

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

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

VASCULAR TOS

- usually easy to detect on **clinical examination** or **vascular imaging** modalities (US, MRA – with/without arm elevation)



TREATMENT

NEUROGENIC TOS

Most patients deserve trial of (and only need) 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 correcting poor posture and improving cervical and periscapular mobility.

SYMPTOMATIC (SECONDARY) NEUROGENIC TOS – maximal conservative therapy (PT) 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: PT + scalene Botox → surgical release (transection of aberrant bundle, removal of cervical rib*, scalenotomy at insertion):

*until the 1930s, first rib resection was mainstay of treatment

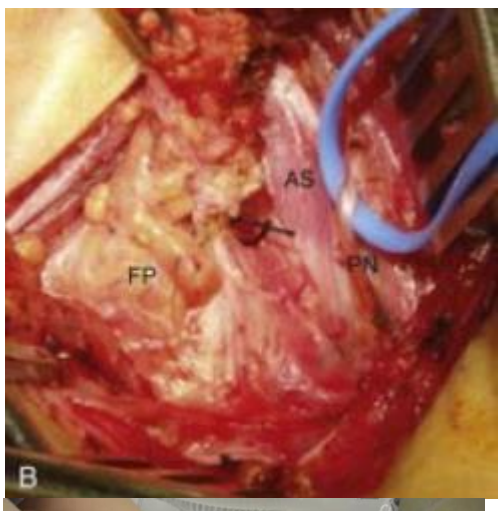
a) *anterior supraclavicular approach*
b) *Roos's transaxillary approach* (with first rib removal) - has many complications (neurovascular injures).
c) *posterior subscapular approach*

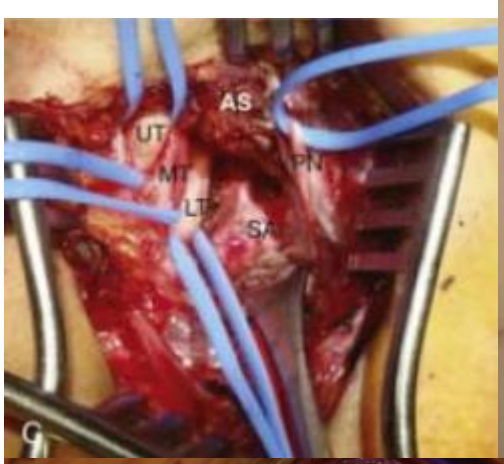
- 15-20% of patients experience recurrence of symptoms after either transaxillary rib resection or scalenectomy; recurrence rate is lowered to 5-10% when a combination of transaxillary rib resection and supraclavicular scalenectomy is used as primary surgery.

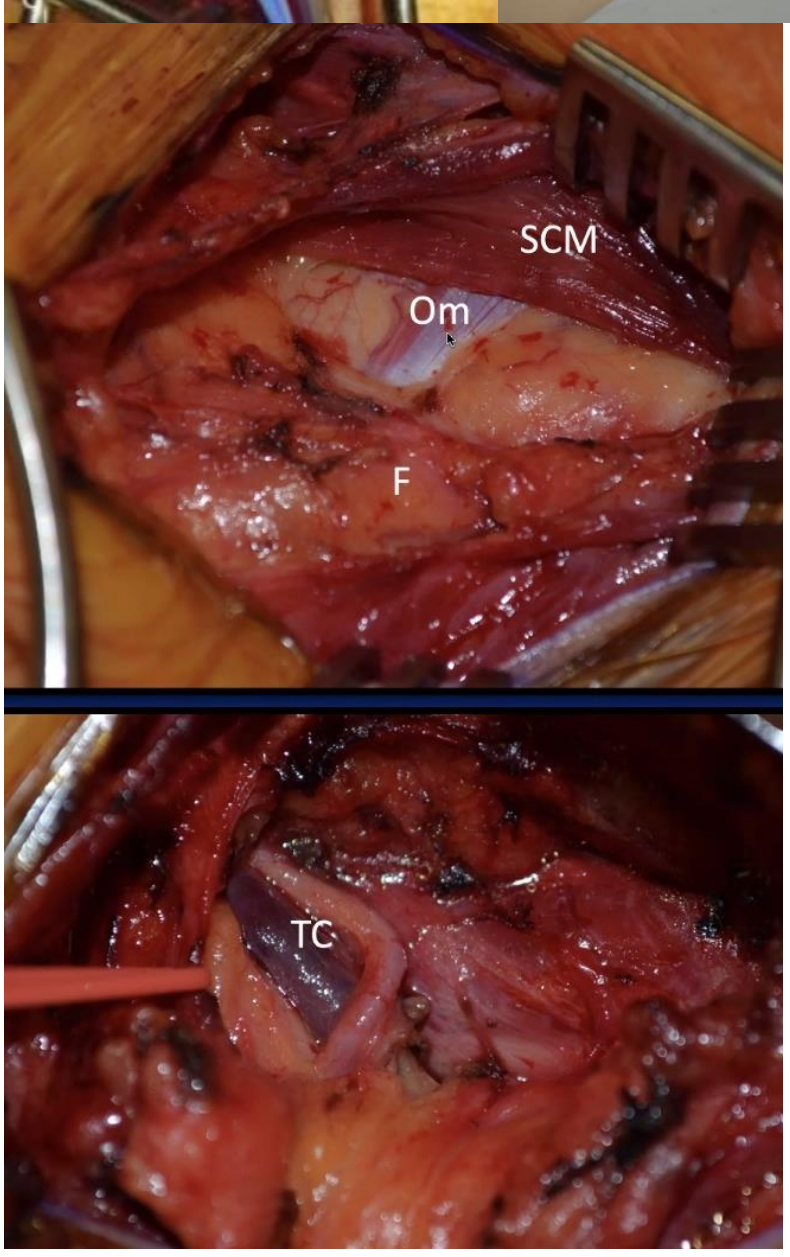
Anterior Supraclavicular Approach

- 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 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 a 3-0 silk tie and divided. **C,** 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 phrenic nerve (PN) is gently retracted medially.







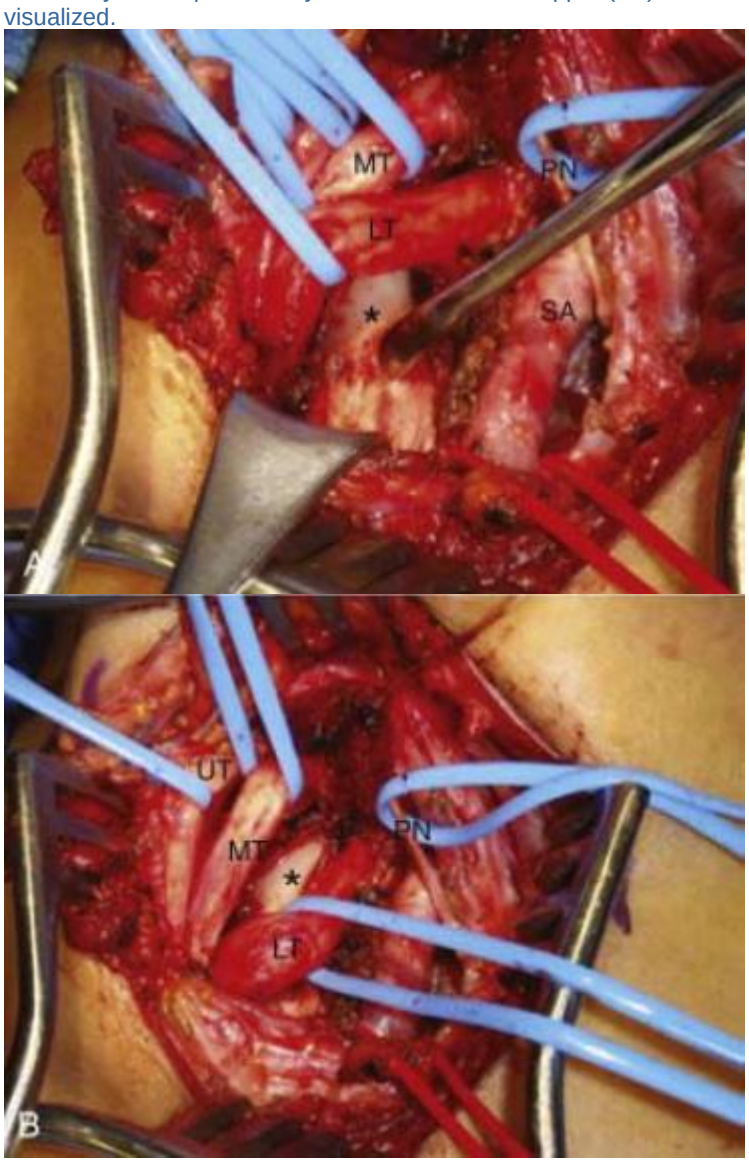
During exposure, important anatomic landmarks to identify are the posterior border of the sternocleidomastoid muscle, the omohyoid muscle, the supraclavicular fat pad, the transverse cervical artery and vein, the phrenic nerve, and the anterior scalene muscle. Our preferred technique is to make a 6- 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 supraclavicular 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.

- **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.
- 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 found by palpation and visual inspection running inferiorly 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.

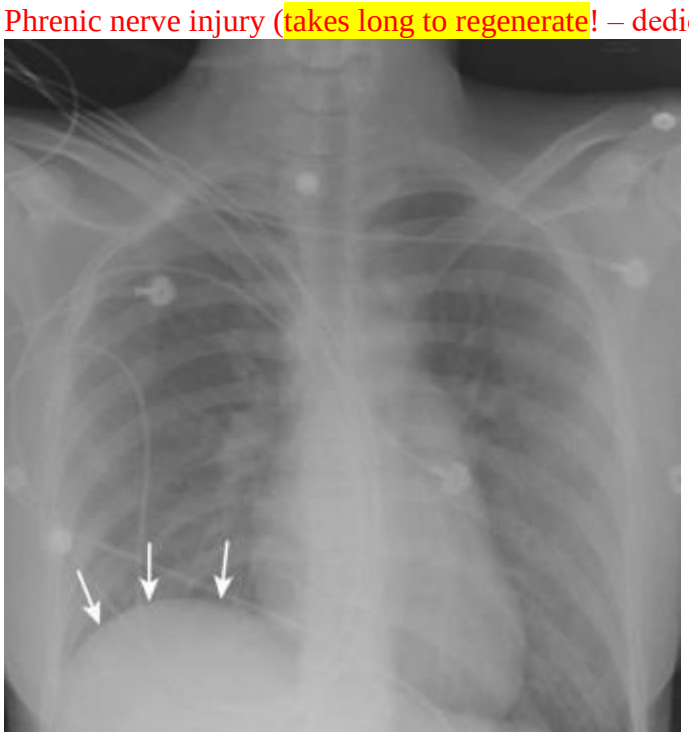
- neural elements are inspected in circumferential fashion, and any compressive bands or anomalous structures are resected.

Intraoperative demonstration of a right-sided cervical rib.
A, The middle (MT) and lower (LT) trunk is gently retracted superiorly to show the distal aspect of the cervical rib (*asterisk*). Also seen are the phrenic nerve (PN) and the subclavian artery (SA). **B,** The lower trunk (LT) is retracted inferiorly to demonstrate the proximal aspect of the cervical rib (*asterisk*). A Penfield No. 4 dissector is placed on the cartilaginous portion of the cervical rib near its articulation with the first thoracic rib. Note the swollen appearance of the lower trunk secondary to compression by the cervical rib. The upper (UT) and middle (MT) trunks and the phrenic nerve (PN) are also visualized.



Occasionally, the **suprapleural 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 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. Numbness 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 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 supraclavicular approach. Transient paresthesias or weakness in the arm or hand is seen occasionally and generally resolves within days to a few weeks.

Phrenic nerve injury (takes long to regenerate! – dedicated respiratory PT → diaphragm plication):



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 arm 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-to-cranial 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 paraspinous 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 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 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.

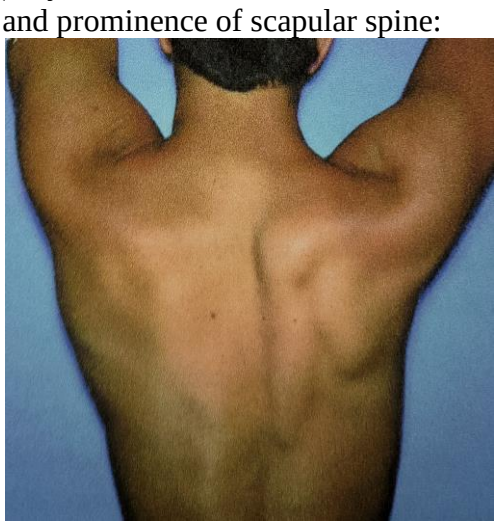
Transaxillary Approach

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 posterolateral

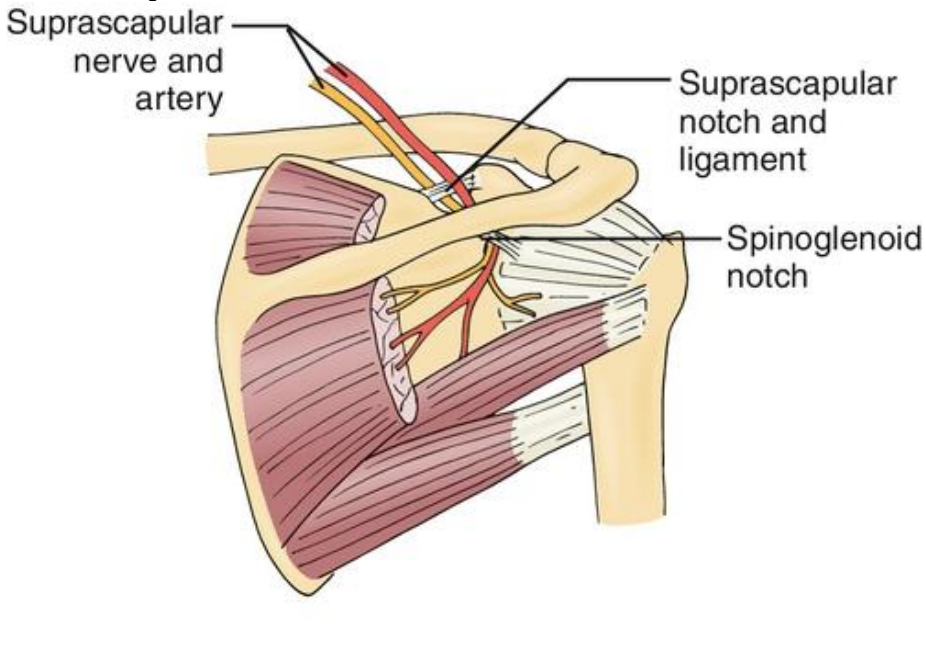
position with the arm elevated above the head. An incision is made over the first palpable rib (usually the third rib) in the axillary fossa. The axillary fat, lymph nodes, and vessels are dissected away, and the anterior and middle scalene muscles are divided. The first rib is identified and resected. 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 elements, behind which congenital 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

- motor nerve (C_{5-6}) → 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 infraspinous fossa and prominence of scapular spine:



Two areas of entrapment:



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

Clinical Features

- only sensory fibers in suprascapular nerve supply posterior aspect of 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.
MRI may show ganglion cyst.

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).
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 are atraumatically split to expose constant fat pad separating trapezius from supraspinatus 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 widely decompressed by clearing off encasing fibrofatty tissue.

ILIOHYPOGASTRIC NERVE

- may cause lower abdominal musculature weakness with bulging ("pseudohernia syndrome")

GENITOFEMORAL NERVE

- may be injured during psoas muscle retraction during LLIF surgery – nerve exits at medial edge of psoas (other nerves – at lateral edge) → burning pain in genitalia

OBTURATOR NERVE

- may be compressed by pelvic tumors, fetal head or forceps.
- sensation to inner thigh, and motor to thigh adductors (gracilis and adductors longus, brevis, and magnus).

FEMORAL NERVE

ETIOLOGY

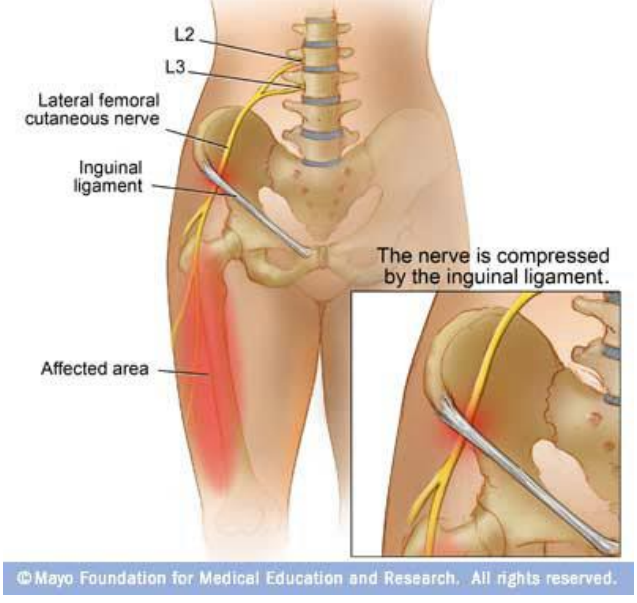
1. **Diabetes** - most frequent cause! (e.g. plexopathy)
2. Entrapment (rare) - secondary to inguinal hernia or its repair (deep sutures placed during herniorrhaphy), prolonged pelvic surgery (retractor compression)
3. DSA with femoral arterial catheterization
4. Intraabdominal tumor, retroperitoneal hematoma
5. Pelvic fracture

CLINICAL FEATURES

- motor deficits - quadriceps femoris (knee extension)
  N.B. ***weakness of iliopsoas (hip flexion) indicates very proximal pathology*** (lumbar root or plexus lesion) as branches to iliopsoas arise just distal to neural foramina!
- patellar (knee jerk) reflex↓
- sensory loss and pain over anterior thigh and medial calf (saphenous nerve)
- positive femoral stretch test.

MERALGIA PARESTHETICA, s. Bernhardt-Roth syndrome, "swashbuckler's disease"

(Greek: *meros* – thigh, *algos* – pain)
- entrapment of purely sensory *lateral femoral cutaneous nerve* (L_{2-3}) where it passes ***beneath inguinal ligament*** at its attachment to the anterior superior iliac spine:



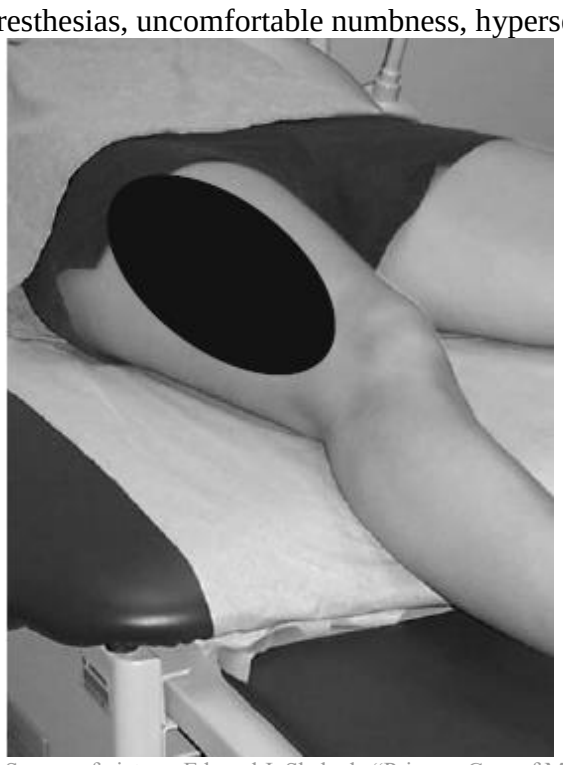
ETIOLOGY

protruding, pendulous abdomen (pregnancy, obesity, ascites), tight belt or corset, excessive walking or marathon running*; also may be initial manifestation of diabetic neuropathy; may also occur post-op in slender patients positioned prone.

*nerve angulation is exaggerated with thigh extension.

CLINICAL FEATURES

- burning paresthesias, uncomfortable numbness, hypersensitivity, hyperpathia:



Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

- patient learns to <u>relieve symptoms</u> by:
  - placing pillow behind thighs;
  - sitting or lying prone helps;
  - assuming slightly hunched posture while standing.

  - spontaneous rubbing or massaging the area in order to obtain relief is very characteristic!
- deep digital pressure 1 cm medial to anterior superior iliac spine (ASIS) may set off shooting paresthesia down lateral thigh.

DIAGNOSIS

- diagnosis is confirmed with **nerve block** - 0.5% BUPIVACAINE injected finger's breadth medial to ASIS → anesthesia + complete cessation of pain and tingling (may be long lasting).
- imaging (18 MHz US, MRI) – only for select cases.

DIFFERENTIAL DIAGNOSIS

1. Femoral neuropathy: sensory changes more anteromedial, extend to anteromedial lower leg (saphenous nerve!)
2. L2 or L3 radiculopathy: motor weakness (thigh flexion or knee extension)
3. Nerve compression by abdominal or pelvic tumor (concomitant GI or GU symptoms)

TREATMENT

1) weight loss, avoidance of all constrictive garments, and postural modification (avoiding hip extension).
2) serial injections of **local anesthesia** and **steroid**.
3) **local measures** – ice applications, capsaicin ointment, lidocaine patches.
4) centrally acting pain medications (e.g. gabapentin, carbamazepine) - rarely effective

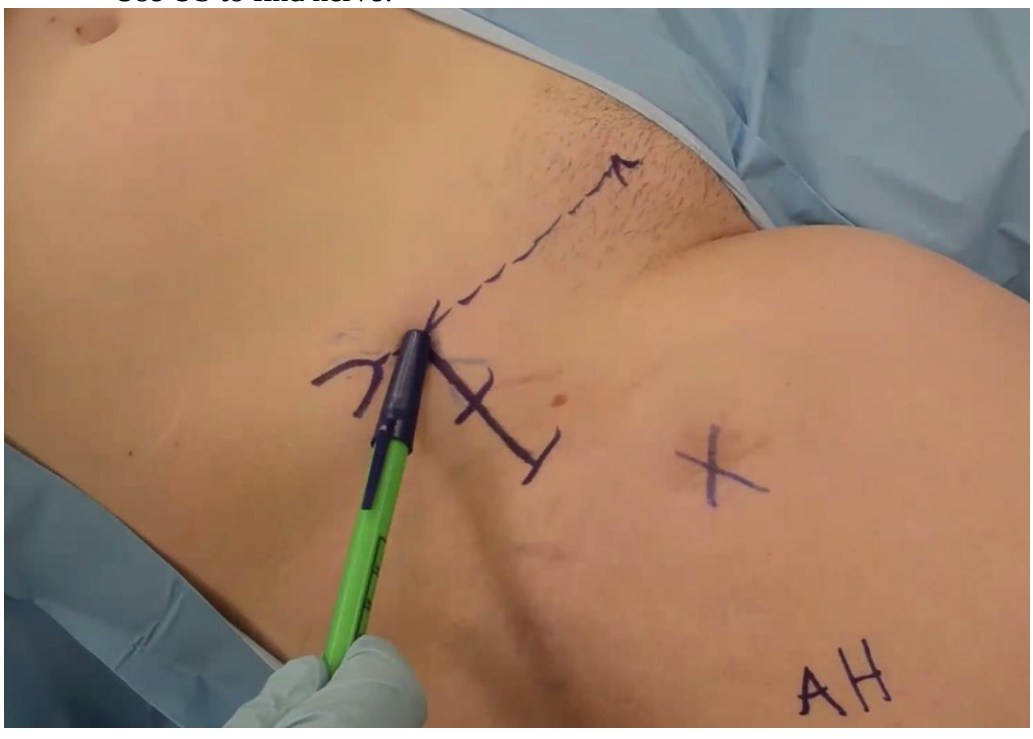
N.B. anatomic variation is common – LFCN may actually pass through the ligament, and as many as four branches may be found.

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 2-3 cm to ensure clearance of other iliacus fascial bands.

N.B. operation is exploratory in nature - generous exposure is required; if nerve can't be located, it is usually because the exposure is too superficial. if nerve still cannot be found → small abdominal muscle incision and nerve located in the retroperitoneal area.

Use US to find nerve!



15-20% cases recur → **nerve transection (neurectomy)** – more effective than neurolysis:

- after freeing nerve at ASIS and proximally toward pelvis, ligature is tied tightly around nerve.
- nerve is ***firmly tugged downward*** → 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 (but risk of denervation pain).

CAUTION: cases have occurred where the femoral nerve has erroneously been divided! – always stimulate before cutting!

SCIATIC NERVE (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 adductor magnus)
4) ***trauma*** (fractures of hip, surgical trauma from hip replacement).

Piriformis syndrome

- main symptom - pain aggravation by sitting on hard surface.
- diagnostic provocative maneuvers – FAIR test. see p. D1 >>
- treatment:
  1) stop aggravating activity
  2) stretching exercises
  3) corticosteroid injection (decreases fat amount around m. piriformis – more room for nerve).

Sciatica - loosely used term - pains in low back and along n. ischiadicus course - caused by involvement of any portion of nerve, including intraspinal L4-S3 roots.

N.B. most common cause is ruptured intervertebral disc! see p. Spin11 >>

N. PERONEUS

Anatomy – see p. A22 (8) >>

Common peroneal nerve is more frequently subjected to trauma / compression than is 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 almost as a rule injures peroneal but spares tibial divisions of sciatic nerve)

Just distal to fibular head, CPN divides into:

1. **Deep peroneal nerve** (AKA anterior tibial nerve) - primarily motor:
   - motor: foot and toe extension (extensor hallucis longus (EHL), anterior tibialis (AT)*, extensor digitorum longus (EDL)). *also inverts foot (in concert with tibialis posterior)
   - sensory: very small area between great toe and second toe.
2. **Superficial peroneal nerve** (AKA musculocutaneous nerve):
   - motor: foot eversion (peroneus longus and brevis).
   - sensory: lateral distal leg and dorsum of foot.

Mechanism:

1) damage at fibular head (fractures, bandages, stockings, crossing knees while sitting).
2) forcible foot inversion (nerve stretching).

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) *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 – **steppage gait** with exaggerated thigh & knee flexion) ± ***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.
- chronic foot drop may produce Achilles tendon contracture (**talipes equinus**).

Diagnosis

1. **Electrophysiologic evaluation** (after > 3 weeks of symptoms) 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.

DIFFERENTIAL OF FOOT DROP

- ***deep peroneal nerve*** → weak **anterior tibialis** (L4 > L5), **EHL & extensor digitorum longus** (L5)
  N.B. foot drop is L5 > L4

1) **L4/L5 radiculopathy** – also affects posterior tibialis (foot inversion) and gluteus medius (internal rotation of flexed hip); pain!
2) **sciatic nerve** palsy (hip fracture-dislocation, IM injection) - **flail foot** (paralysis of dorsiflexors + plantarflexors)
3) **common peroneal nerve** palsy
4) **Charcot-Marie-Tooth**
5) **heavy metal** poisoning (esp. *lead*)
6) **diabetic** neuropathy
7) **Hansen's disease** (leprosy)
8) lesion anywhere along **pyramidal tract**, **motor neuron disease** - **spastic foot drop** (Babinski sign, hyperactive Achilles reflex).
9) anterior compartment syndrome, severe ankle inversion sprains
10) muscular dystrophy
11) popliteal fossa cysts (Baker cyst)
12) anterior tibial artery aneurysm

Foot drop → get **MRI** to rule out mass effect – either **L-spine** or **peroneal** ← decide clinically

N.B. painless foot drop is unlikely to be due to radiculopathy!

N.B. L5 affects both foot inversion and eversion (deep peroneal nerve – only partial inversion; superficial peroneal nerve – only eversion)

TREATMENT

CONSERVATIVE THERAPY

- effective for most cases of CPN entrapment:
  Complete or partial recovery is rule when paralysis is caused by transient pressure!

1. **PT** to prevent Achilles contractures (heel cord), which would impair ankle dorsiflexion if nerve function returns.
2. **Ankle-foot orthosis (AFO) –** inserts unobtrusively into a shoe - to protect ankle joint and improve gait.

SURGERY

Peroneal Nerve Decompression – for patients who show little or no improvement after 3 months.
See p. Op450 >>

N.B. operate for foot drop early (maximum wait – 3 months)

N.B. peroneal injuries above knee usually do not regenerate enough!

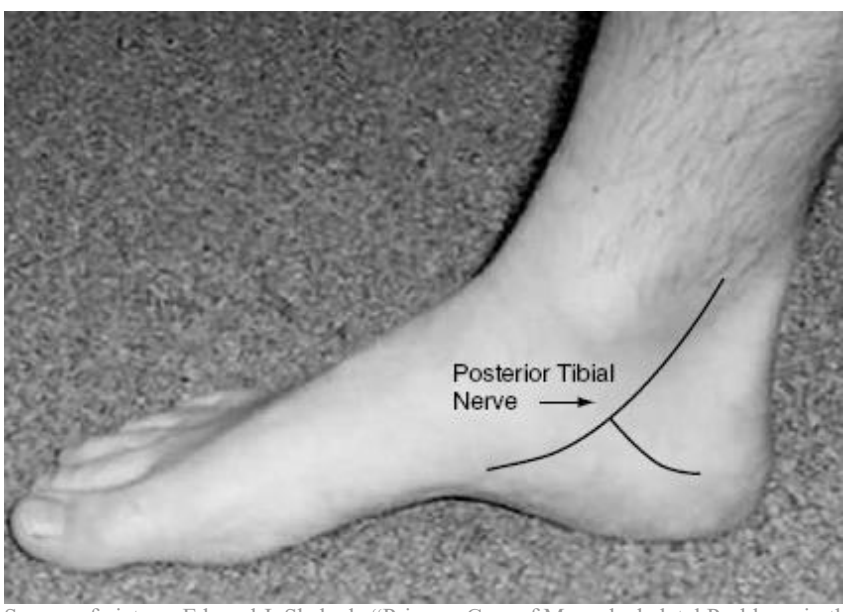
Persistent footdrop after surgery → **TP tendon transfer** - highly effective for footdrop caused by CPN injury, particularly in men < 30 years

N. TIBIALIS POSTERIOR / TARSAL TUNNEL SYNDROME

- *posterior tibial nerve* entrapment posterior-inferior to medial malleolus at flexor retinaculum or more distally.

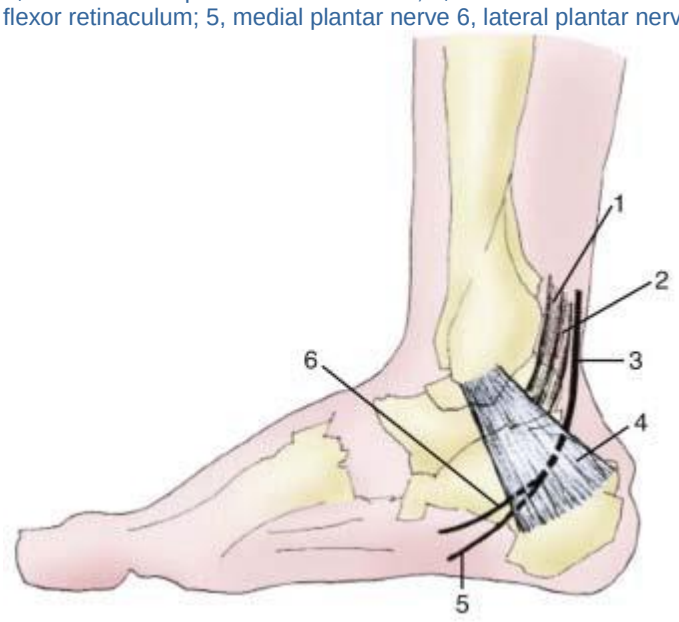
Tarsal tunnel (TT) anatomy

- TT is covered by **flexor retinaculum (laciniate ligament)** which extends downward from the medial malleolus to the tubercle of the calcaneus.
- 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.
- TT is made up of two main compartments: an upper (tibiotalar) and a lower (talocalcaneal) 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.
- 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.
- 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.
- medial and inferior calcaneal nerves may arise proximal to, within, or distal to the TT.



Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

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



Picture source: Fernandez E, Pallini R, Lauretti L, et al. Neurosurgery of the peripheral nervous system: Entrapment syndromes of the lower extremity. Surg Neurol. 1999;52:449

- etiology (quite rare): bony impingement (ankle trauma), space-occupying lesions (ganglion cysts, schwannomas, RA tenosynovitis, hypertrophic muscles, or varicosities, gout, diabetes, and myxedema).
- clinical features:
  1) burning, unpleasant poorly localized pain and paresthesias in medial heel* + sole (down to first, second, and third toes)
     *__calcaneal branch__ (sensation to heel) often is spared because of its proximal takeoff.
     - pain is set off by pressing or rubbing over plantar skin, sometimes with after-discharge phenomenon.
     - some patients experience ***nocturnal exacerbations***
     - pain *reminds plantar fasciitis*, but positive **Tinel sign** (tapping* over area posterior to medial malleolus → numbness, tingling) is present.
       *or compression with finger for 30 s
  2) intrinsic toe flexors are weak and atrophied → hollowing of instep, **toe clawing**.
  3) provocative testing: foot dorsiflexion-eversion – examiner maximally everts and dorsiflexes the ankle while dorsiflexing the toes at the MTP joints for 5–10 seconds - positive test reproduces pain.

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, antidepressant, and narcotic pain medications.

Surgical Decompression (75% patients enjoy significant improvement)

- **incision** begins 2 cm proximal to medial malleolus to pick up neurovascular bundle above flexor retinaculum.

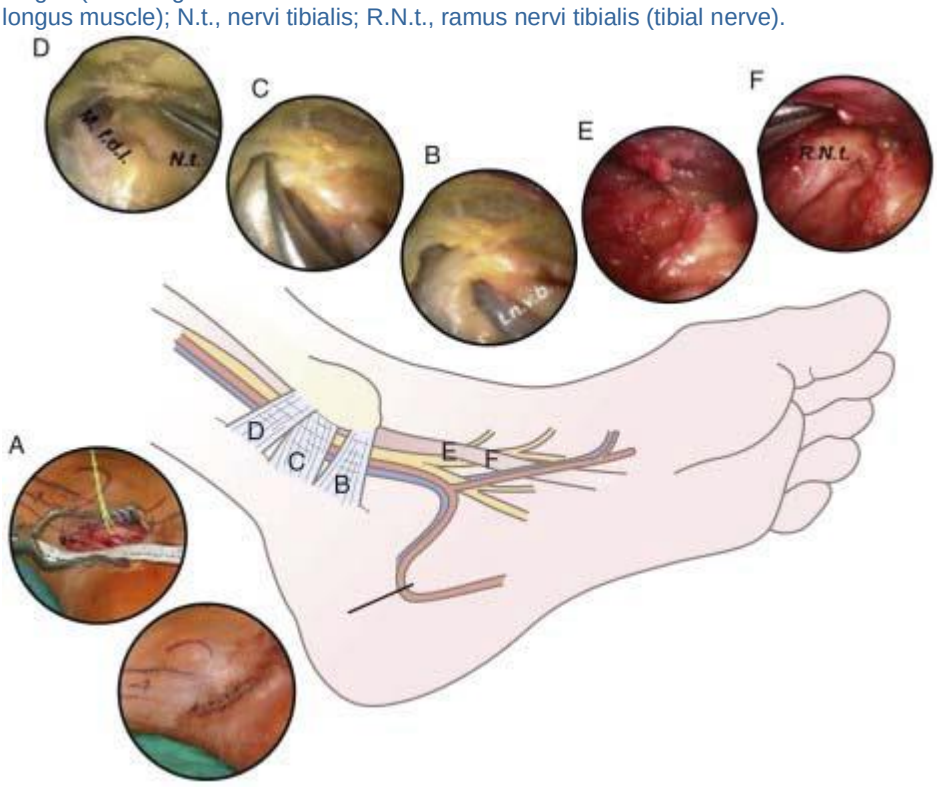
- nerve is followed distally with **release of retinacular fibers**.
- mass lesions or fibrous septae are identified and removed.
- each of plantar nerve canals is opened into plantar surface.
- tight fascial band arising from border of m. abductor hallucis and roofing over plantar canals is divided.
- 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 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.

Schematic representation of the course of the tibial nerve (*central sketch*) and the various endoscopic (*top*) and macroscopic (*bottom*) views. The steps of in situ decompression of the tibial nerve follow the alphabetical labeling order. The labels on the endoscopic
snapshot insets correspond to the anatomic region represented by the lettering on the sketch. **A,** The tibial nerve is openly dissected under loupe
magnification behind the medial malleolus. **B,** The ligaments roofing the tarsal tunnel are seen here. **C,** The ligaments are split. **D,** Proximal
release of the nerve is performed up to the distal third of the leg. **E,** Distal release of the tarsal tunnel. **F,** The distal dissection reaches well into th
plantar region, where the nerve is seen to bifurcate. t.n.v.b., tibial neurovascular bundle; M.f.d.l., musculus flexor digitorum longus (flexor digitorum
longus muscle); N.t., nervi tibialis; R.N.t., ramus nervi tibialis (tibial nerve).



Picture source: Krishnan KG, Pinzer T, Schackert G. A novel endoscopic technique in treating single nerve entrapment syndromes with special attention to ulnar nerve transposition and tarsal tunnel release: Clinical application. Neurosurgery. 2006;59:ONS89

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 neuroma, 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 Murovic reported a series of patients who underwent revision surgery for TTS at LSUHSC. 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 neurectomy 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 at the anterior aspect of the ankle, and the posterior tibial nerve along with calcaneal, medial, and lateral plantar branches at the TT (the Dellon triple decompression technique). A multicenter prospective study of this technique in diabetic patients reported a reduction in the prevalence of foot ulceration in 665 patients without previous ulceration from 15% to 0.6%; in 44 patients with a previous history of foot ulceration, the prevalence of ulceration was reduced from 50% to 2.2%. The authors claim that this triple 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.

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

Source of picture: Edward J. Shahady "Primary Care of Musculoskeletal Problems in the Outpatient Setting" (2006); Springer; ISBN-13: 978-0387306469 >>

- 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	Compressive Sites & Causes
Axillary (C_{5-6}): m. deltoideus, teres minor; C_5 sensory	*Near shoulder joint*: fractures / dislocation of humerus head; neuritis after serum (esp. antitetanus) therapy
Long thoracic (C_{5-7}): see p. D1 >> m. serratus anterior; not sensory	Surgery
Femoral (L_{2-4}): see p. D1 >> m. iliopsoas, quadriceps femoris; anterior thigh sensory	*Proximal to inguinal ligament*: idiopathic, iatrogenic, retroperitoneal hemorrhage, tumor
Saphenous	Iatrogenic (surgery, scar after surgery)
Ilioinguinal, **iliohypogastric**	
Obturator (L_{3-4}): thigh adductors; medial thigh sensory	Pelvic tumor, hematoma, obturator hernia, difficult labor

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 >>