433 Teams

MEDICINE

18| Peripheral Neuropathy

Part 2

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Peripheral Neuropathy

C. lesions according to number of affected nerves :

1) **(mononeuropathy).**
2) **(mononeuropathy multiplex).**
3) **(polyneuropathy).**

- Weakness is more prominent distally at the outset (as opposed to muscle myopa- thy **usually asymmetric).**
- Presents with diminished deep tendon reflexes, may include sensory changes (numbness, paresthesias, tingling), muscle atrophy, and fasciculations.
- Can be due to diabetes (nerve infarction), trauma, entrapment, or vasculitis.
- (The most common cause of peripheral neuropathy is diabetes mellitus).
- Common neuropathies include radial/ulnar/median/musculocutaneous nerves, long thoracic nerve, axillary nerve, common peroneal nerve, and femoral nerve.

1) mononuropathy:

These neuropathies are recognized largely by clinical features. Diagnosis is confirmed by nerve conduction studies.

People with these conditions can experience both weakness and numbness.

Common causes: Diabetes, Vasculitis, Infection, Leprosy, Neoplasm, Brachial or lumbarplexopathy, Radiation, Sarcoidosis andRadiculopathies.

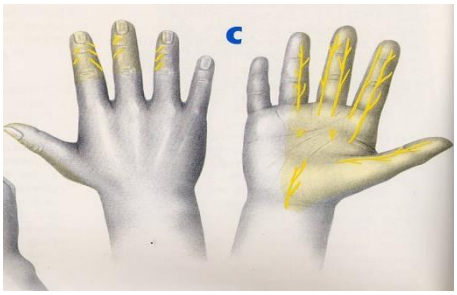
In the nexst few slides will go through these nerves

Table 21.52 Nerve compression and entrapment

Nerve	Entrapment/compression site
Median	Carpal tunnel (wrist)
Ulnar	Cubital tunnel (elbow)
Radial	Spiral groove (of humerus)
Posterior interosseous	Supinator muscle (forearm)
Lateral cutaneous of thigh	Inguinal ligament
Common peroneal	Neck of fibula
Posterior tibial	Tarsal tunnel (flexor retinaculum – foot)

Median nerve compression:

- The median nerve is derived from the **medial** and **lateral** cords of the brachial plexus. It contains fibres from all five roots (C5-T1).
- Three separate syndromes are recognised: Carpal tunnel syndrome, Pronator syndrome, and Anteriorinterosseous syndrome.



• Carpal tunnel syndrome:

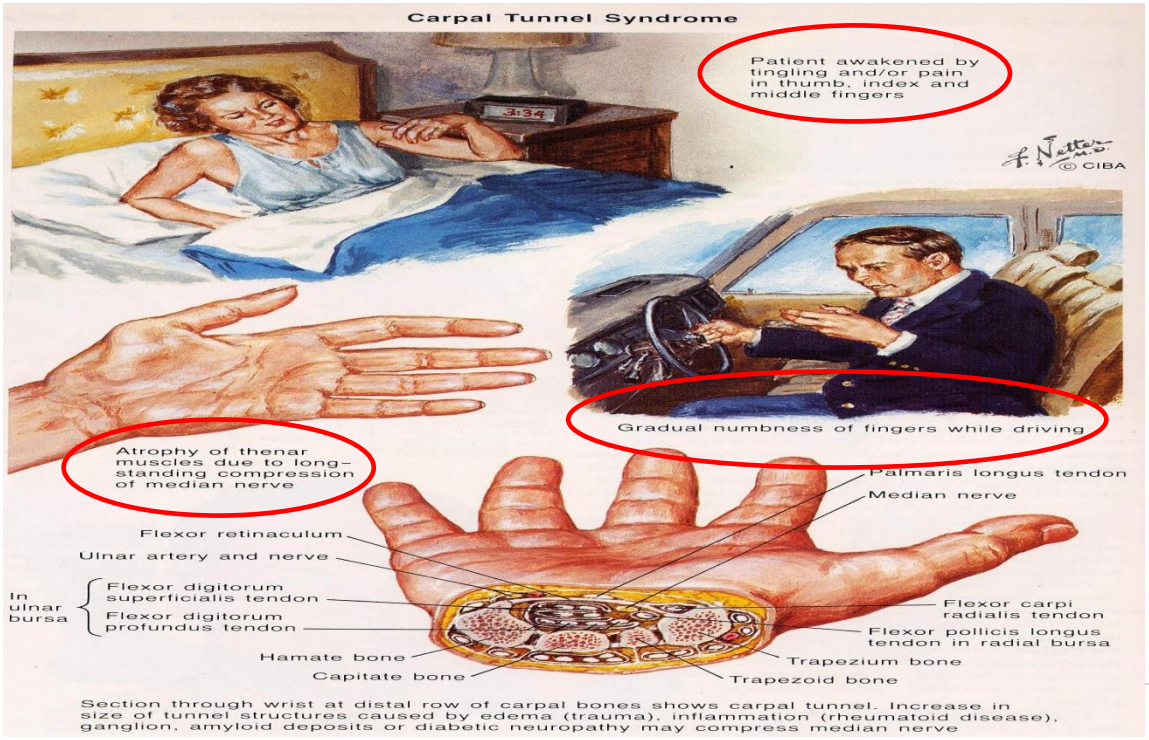
❖ Clinical features:

1. Nocturnal pain or paresthesia in the hand and/or forearm, thenar atrophy will be only in the hand (unlike Radiculopathy of C8 and T1)
2. Sensory loss in the palm and radial three-and-a-half fingers develops, followed by wasting of abductor pollicisbrevis.
3. Tinel's sign :Tinel's is elicited by tapping the flexor aspect of the wrist: this causes tingling and pain (https://www.youtube.com/watch?v=VtrC9dnVrrQ)
4. Phalen's test positive: In Phalen's, symptoms are reproduced on passive maximal wrist flexion. (https://www.youtube.com/watch?v=DZ9UGuA8oAE)

❖ Treatment:

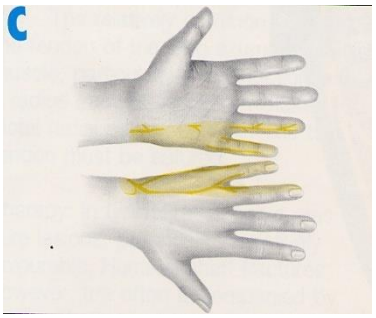
- Splinting – prevent wrist flexion.
- Corticosteroid/anesthetic injection – give temporary relief.
- Surgical decompression.

Hand of Benediction or Pop's Blessings (APE HAND) will result from median nerve injury.



Ulnar Nerve:

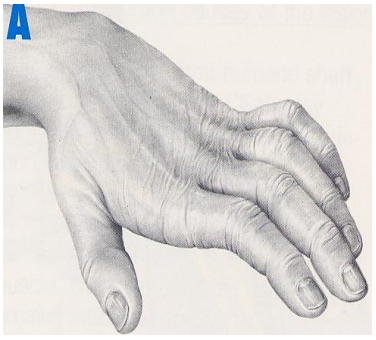
- The ulnar nerve is derived from the brachial plexus. It is a continuation of the **medial cord,** containing fibres from spinal roots C8 and T1.
- Three lesions are recognized: Lesion at condylar groove, lesion at Guyon's canaland Lesion at wrist and hand.



Lesion at condylar groove, which lead to (cubital tunnel)

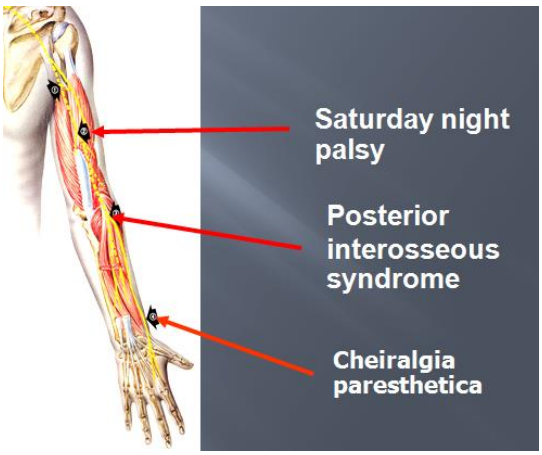
Cubital tunnel:

This follows prolonged or recurrent pressure and elbow fracture. Weakness and wasting of ulnar innervated muscles leads to clawing of the hand – hypothenar muscles, interossei and medial two lumbricals – with sensory loss in the ulnar one and a half fingers. Decompression and transposition of the nerve at the elbow is sometimes helpful.



Radial Nerve:

The radial nerve is a continuation of the posterior cord of the brachial plexus, containing nerve fibres from all 5 roots (C5 – T1).



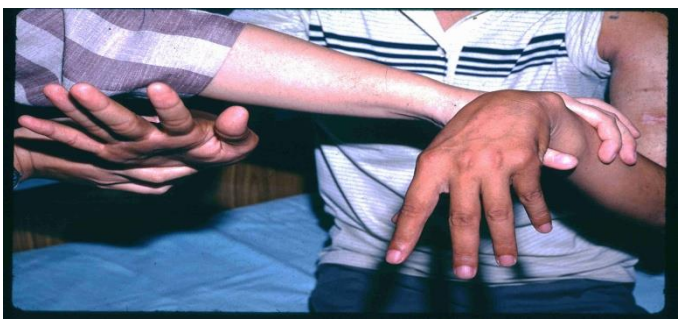
❖ 3 main lesions :

i. **Saturday night palsy:** The terms Saturday night palsy and honeymooner's palsy refer to the concept of placing one's arm over another chair, with the resultant pressure causing injury to the radial nerve.
ii. **Posterior interosseous syndrome:** (A pure motor branch of the radial nerve, no sensory loss)
iii. **Cheiralgiaparesthetica:** (Sensory only).

❖ specific sites of lesions and its affects:

- ✓ **Axillary lesion:** weak triceps and radial innervated muscles.
- ✓ **Mid-upper arm lesion:** 'Saturday night palsy' (spiral groove or intermuscular septum), wrist drop, variable motor and sensory deficits (weakness of Brachioradialis and finger extension).
  -with normal triceps, why ?
  becausethe affected part is always distal to the lesion site
- ✓ **Posterior interosseous :** weak extensor of thumb and other fingers, (wrist drop), no sensory loss
  -without weakness of Brachioradialis, why ?
- ✓ **Superficial radial nerve:** terminal cutaneous branch:

a) Wrist drop of posterior interosseous nerve injury, the sensory supply will be intact.
b) Wrist drop of the spiral groove injury both sensory and motor of wrist and fingers will be affected.
c) In lower part only sensory part of the radial will be affected at dorsum of the hand.



Wrist drop

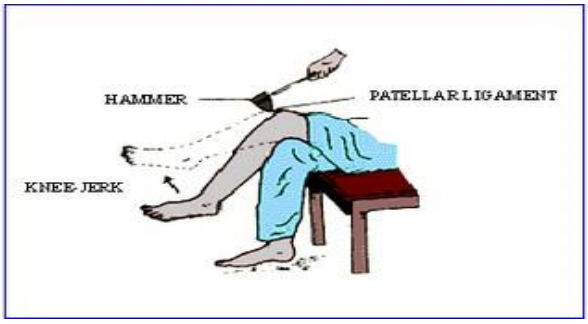
Femoral Nerve (L2,3,4)

- Mix sensorimotor.
- Quadriceps femoris or knee extensor.

❖ The lesions

1- Motor effect:

a) Wasting of quadriceps femoris.
b) Loss of extension of knee Weak flexion of hip. (opposite to sciatic nerve).
c) Weakness of hip flexor in intraabdominal lesion



2- Sensory deficit: over anteromedial aspect of thigh and perhaps leg.

3- Absent or diminished knee jerk

Lateral femoral cutaneous:

Meralgiaparesthetica: A painful mononeuropathy of the lateral femoral cutaneous nerve (LFCN), meralgiaparesthetica is commonly due to focal entrapment of this nerve as it passes through the inguinal ligament. And it's pure sensory common condition seen in clinics "Pure sensory loss to the inferior iliac spine area"

Notes:

- If it's sensory and motor defect at the same area (inferior iliac spine): It can't be femoral because the sensory branch of femoral which is the saphenous nerve extends till down so the sensation should be affected all the way down.
- But you think of radiculopathy (L2-L3) because they supply the motor and sensory part of that area but it should be asymetrical to say it's radiculopathy. And if it was pure sensory it's meralgiaparesthetica.
- confirm by EMG.
- Whenever you have a problem in sensation in one area check it from other ways, it can be different nerve, it can radiculopathy, it can't be polyneuropathy here cause it's proximal defect not distal, neither mononeuritis multiplex because only one area is involved.

Sciatic nerve (L4, S3):

- The sciatic nerve is most frequently injured
- Composed of 2 main nerves of leg: common peroneal and tibial nerve.
- Paralysis of all muscles below knee plus hamstrings and for high lesion, external rotators of thigh.
-

❖ SCIATIC NERVE INJURY:

Motor effect:

paralysis of hamstrings and all muscles of legs and foot.

movement affected: Weak flexion of the knee, weak extension of hip and all movements of leg and foot.

Sensory effect:loss below knee except anteromedial aspect of leg and foot.

-Foot Drop(High stoppage gait): which innervated by branch of femoral nerve.

A. Common peroneal nerve:

Also called lateral popliteal nerve, affected by fracture or compressed against the head of the fibula after prolonged squatting, yoga, pressure from a cast, prolonged bed rest or coma, or for no apparent reason.

- ✓ Foot-drop and weakness of ankle eversion.
- ✓ Numbness develops on the anterolateral border of the dorsum of the foot and/or lower shin.
- ✓ Paralysis of anterior and lateral compartment of leg.
- ✓ Sensory loss over dorsum of foot and toes and anterolateral aspect of leg.
- ✓ Loss of dorsiflexion of ankle.
- ✓ The ankle jerk (S1) is preserved.
- ✓ Recovery is usual, though not invariable, within several months.

B. Tibial nerve

- Medial division of sciatic nerve.
- Lesions at ankle.

The lesions:

❖ *Tarsal tunnel syndrome:*

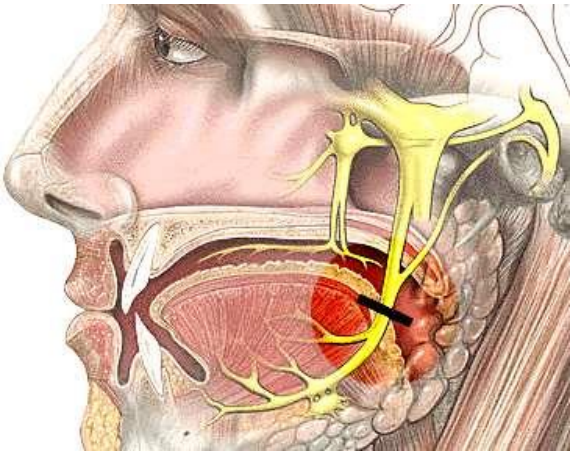
Tarsal tunnnel syndrome: when the tibial branch reaches the foot passing behind the medial malleulous and branches to medial and lateral tarsal nerves, there happens the common compression which causes burning and pain and discomfort. It can happen in both feet.

- ❑ Pain and paresthesia in sole.
- ❑ Paralysis of intrinsic muscles of foot.
- ❑ Tenderness of Tinel's sign at flexor retinaculum.

❖ *Sural nerve compression syndrome*

- ❑ Pure sensory.
- ❑ Numbness on lateral aspect of foot.

Facial nerve:



The lesions:

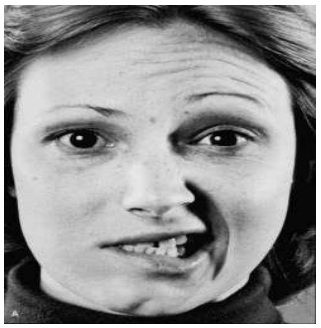
- Bell's palsy: idiopathic, HSV1
- Ramsay Hunt syndrome : external ear pain with presence of herpes zoster vesicles in auditory canal and pinna, VZV
- Trauma: blunt impact to temporal bone.
- Middle ear infection: otitis media, mastoid pain persist after acute infection resolved.
- Neoplasm: rarely compressed by CPA (Cerebellopontineangle) tumor but due to surgery for tumor removal.

❖ Bell's palsy: (idiopathic facial paralysis)

Always remember it's a mononeuropathy.

Facial nerve is LMN nerve (differentiate between upper and lower facial weakness).

Bell's palsy is not the result of a stroke or a transient ischemic attack (TIA). While stroke and TIA can cause facial paralysis, there is no link between Bell's palsy and either of these conditions. But sudden weakness that occurs on one side of your face should be checked by a doctor right away to rule out these more serious causes.



Clinical features:

- Postauricular pain (few days).
- Lower motor neuron facial weakness (where all muscles of facial expression are affected on the side of the lesion).
- Impaired taste.
- Hyperacusis: caused by paralysis of the stapedius muscle, defined as intolerance of loud or high pitch sounds.
- Difficulty with speaking.
- Dryness of the eyes and mouth (decrease lacrimation and salivation).

Diagnostic Tests:

No test is usually done because of the characteristic presentation of paralysis of half of the face. The most accurate test (if asked) is electromyography and nerve conduction studies.

Management:

60% of patients have full recovery even without treatment. The best initial therapy is prednisone. Acyclovir does not help.

2) Mono-neuropathymultiplex :

- ✓ Mononeuritis multiplex: sometimes they're not explained by compression. Not only involve compressive nerves, but also the non-compressive.
- ✓ put in mind systemic diseases.
- ✓ This occurs in:DM, Leprosy, Vasculitis, Sarcoidosis.
- ✓ Diagnosis is largely clinical, supported by electrical studies.
- ✓ Several nerves become affected sequentially or simultaneously, e.g. ulnar, median, radial and lateral popliteal nerves. When multifocal neuropathy is symmetrical, there is difficulty distinguishing it from polyneuropathy. Where leprosy is prevalent, e.g. in India, a single nerve lesion can be the presenting feature.

3) Polyneuropathy:

Polyneuropathy is a generalized pathological process occurring in the longest peripheral nerves first, affecting the distal lower limbs before the upper limbs, with sensory symptoms and signs of an ascending 'glove and stocking' distribution. This is particularly true with axonal neuropathies where the disorder affects the metabolic processes required for axonal transport in the peripheral nerves. In inflammatory demyelinating neuropathies, the pathology may be patchier and variations from this ascending pattern occur.

1- Axonal.
2- Demyelinating.

Q How can we differentiate between axonal and demyelination?

A: by EMG nerve conduction study

1- Axonal:

Acute:

- AXONAL VARIANT OF GBS "Guillain-Barre Syndrome"
- PORPHRIA" disease of hemoglobin metabolism lead to sever abdominal pain and neuropathy"
- Medication : NITROFURONOIN

Sub-acute:

- Toxins.
- Malnutrition.
- Systemic disease : DM, hypertension, HIV.

Chronic:

- HMSN-II ' Hereditary Motor Sensory Neuropathy'

-Most common reason causes polyneuropathy is diabetes, next medication induced (antibioctic).

-Toxic neuropathy is one of the commonest reason in polyneuropathy, lead and arsenic are common in old people.

2-Demyelination:

A) Uniform.

-All nerve demyelinated .

-Genetic\Hereditary diseases mainly cause UNIFORM.

B) Non-uniform.

-Patchy demyelinating.

-Acute→ AIDP 'acute inflammatory demyelinating polyneuropathy' Diphtheri 'serious bacterial infection.

-Chronic→ CIDP 'chronic inflammatory demyelinating polyneuropathy'

Q: How can we differiniate between them ?
A: By EMG.electromyograpgies

Q:Patient come polyneuropathy, What is the most important test?
A: EMG.electromyograpgies.

Acute inflammatory demyelinating polyneuropathy (AIDP):

❖Guillain-Barre Syndrome "in acute only" :

- ✓ Guillain-Barré syndrome (GBS) is an acute, frequently severe, and fulminant, polyradiculoneuropathy that is autoimmune in nature.
- ✓ Males are at slightly higher risk for GBS than females, and in Western countries adults are more frequently affected than children.
- ✓ The most common acute polyneuropathy. Acute inflammatory polyneuropathy is most commonly demyelinating (acute inflammatory demyelinating neuropathy, AIDP), but occasionally axonal and probably has an autoallergic basis.
- ✓ There is a predominantly cell-mediated inflammatory response directed at the myelin protein of spinal roots, peripheral and extra-axial cranial nerves. The resulting release of inflammatory cytokines blocks nerve conduction and is followed by a destruction of the myelin sheath and the axon.
- ✓ Paralysis follows 1–3 weeks after an infection that is often trivial and seldom identified. Campylobacter jejuni and cytomegalovirus infections are well-recognized causes of severe GBS. Infecting organisms induce antibody responses against peripheral nerves.
- ✓ The patient complains of weakness of distal limb muscles and/or distal numbness. These symptoms progress proximally, over several days to 6 weeks. In some cases respiratory and facial muscles become weak, sometimes progressing to complete paralysis. Autonomic features sometimes develop.
- ✓ GBS (AIDP) is the most common condition we see.
- ✓ Ten years later, it'll affect all the nerves even the proximal ones, CSF proteins will be high because it'll affect the roots and the roots are covered by protein which will reach the CSF in case of inflammation.
- ✓ Characteristics of GBS: cells will be normal and CSF proteins will be high.

Subtype	Features	Electrodiagnosis	Pathology
Acute inflammatory demyelinating polyneuropathy (AIDP)	Adults affected more than children; 90% of cases in western world; recovery rapid; anti-GM1 antibodies (<50%)	Demyelinating	First attack on Schwann cell surface; widespread myelin damage, macrophage activation, and lymphocytic infiltration; variable secondary axonal damage
Acute motor axonal neuropathy (AMAN)	Children and young adults; prevalent in China and Mexico; may be seasonal; recovery rapid; anti-GD1a antibodies	Axonal	First attack at motor nodes of Ranvier; macrophage activation, few lymphocytes, frequent periaxonal macrophages; extent of axonal damage highly variable
Acute motor sensory axonal neuropathy (AMSAN)	Mostly adults; uncommon; recovery slow, often incomplete; closely related to AMAN	Axonal	Same as AMAN, but also affects sensory nerves and roots; axonal damage usually severe
M. Fisher syndrome (MFS)	Adults and children; uncommon; ophthalmoplegia, ataxia, and areflexia; anti-GQ1b antibodies (90%)	Demyelinating	Few cases examined; resembles AIDP

A. Clinical features:

1. Abrupt onset with rapidly ascending weakness/paralysis of all four extremities; frequently progresses to involve respiratory, facial, and bulbar muscles.

   a. Usually symmetric (but not always).

   b. Weakness may be mild or severe.

   c. Weakness usually progresses from distal to central muscles.

   d. If generalized paralysis is present, it can lead to respiratory arrest

2. Extremities may be painful, but sensory loss is not typical.

3. Sphincter control and mentation are typically spared.

4. Autonomic features (e.g., arrhythmias, tachycardia, postural hypotension) are dangerous complications.

B. Diagnosis:

- Diagnostic Tests.

- The most specific diagnostic test is nerve conduction studies/electromyography. These will show a decrease in the propagation of electrical impulses along the nerves, but it takes 1–2 weeks to become abnormal.

- CSF shows increased protein with a normal cell count.

- Tests of Respiratory Muscle Involvement,when the diaphragm is involved, there is a decrease in forced vital capacity and peak inspiratory pressure. Inspiration is the “active” part of breathing and the patient loses the strength to inhale. PFTs tell who might die from GBS.

C. Treatment:

- Carefully monitor pulmonary function. Mechanical ventilation may be necessary.
- Intravenous immunoglobulin (IVIG) or plasmapheresisare equal in efficacy.
- Do not give steroids(Prednisone). They are usually harmful and never helpful in Guillain–Barré syndrome.

Chronic inflammatory demyelinating polyneuropathy (CIDP):

- ✓ Multifocal motor neuropathy (MMN).
- ✓ Lewis-Sumner syndrome, also known as multifocal acquired demyelinating sensory and motor neuropathy (MADSAM).
- ✓ Distal demyelinating neuropathy with IgMparaprotein, with or without anti-myelin associated glycoprotein (anti-MAG).
- ✓ Demyelinating neuropathy with IgG or IgA paraprotein.
- ✓ POEMS syndrome (osteosclerotic myeloma: Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal protein, Skin changes).
- ✓ Sensory predominant demyelinating neuropathy.
- ✓ Demyelinating neuropathy with central nervous system demyelination.
- ✓ Demyelinating neuropathy associated with systemic disorders, including: Hepatitis B or C, HIV infection, lymphoma, diabetes mellitus, SLE/CTD, thyrotoxicosis, organ or bone marrow transplants, inherited neuropathies, nephrotic syndrome, inflammatory bowel disease.
- ✓ Presents with a relapsing or progressive generalized neuropathy. Sensory, motor or autonomic nerves can be involved but the signs are predominantly motor; a variant causes only motor involvement (multifocal motor neuropathy, MMN).
- ✓ CIDP usually responds to immunosuppressive treatment, corticosteroids, methotrexate or cyclophosphamide, or to immunomodulatory treatments (plasma exchange or intravenous immunoglobulin, IVIg); MMN is best treated by IVIg.
- ✓ Some 10% of patients with acquired demyelinating polyneuropathy have an abnormal serum paraprotein, sometimes associated with a lymphoproliferative malignancy.
- ✓ Symmetrical numbness and tingling occurs in hands and feet, spreading proximally in a glove and stocking distribution. Distal weakness also ascends. Tendon reflexes are lost.
- ✓ Nerve conduction studies show either axonal degeneration or demyelination, or features of both, and no underlying cause is found.
- ✓ Steroids isn't recommended except in CIDP.

TABLE 4-3 Chronic Inflammatory Demyelinating Polyneuropathies: Classification By Weakness and Sensory Loss Patterns

Clinical Features	Classic Chronic Inflammatory Demyelinating Polyneuropathy	Distal Acquired Demyelinating Symmetric Neuropathy	Multifocal Acquired Demyelinating Sensory and Motor Neuropathy	Multifocal Motor Neuropathy
Weakness and sensory loss	Symmetric, distal and proximal	Symmetric, mild distal	Asymmetric, mostly distal	Purely motor, asymmetric, mostly distal
Elevated CSF protein	Yes	Yes	Yes	No
M protein	Uncommon, may have IgG or IgA	Usually IgM, rarely IgG	Uncommon	Uncommon, rarely IgM
Antineural antibodies	Uncommon	Anti–myelin-associated glycoprotein (MAG) in 50% of patients	Uncommon	Anti-GM_1
Motor nerve conduction studies	Demyelination	Demyelination	Demyelination	Demyelination
Sensory nerve conduction studies	Absent or small sensory nerve action potentials (SNAPs)	Absent or small SNAPs	Absent or small SNAPs	Normal
Treatments	Prednisone, IV immunoglobulin (IVIg), plasmapheresis, immunosuppressants	Possibly rituximab Some patients without anti-MAG may respond to IVIg	IVIg, immunosuppressants	IVIg, possibly cyclophosphamide

Electrodiagnostic Criteria for CIDP:

These criteria are applied by testing the median, ulnar (stimulated below the elbow), peroneal (stimulated below the fibular head), and tibial nerves on one side of the body. During testing, limb temperature should be no less than 33°C at the palm and no less than 30°C at the external malleolus.

Definite CIDP
At least one of the following demyelinating parameters are necessary:
≥51 percent prolongation of motor distal latency above the ULN in two nerves
≥31 percent reduction of motor conduction velocity below the LLN in two nerves
≥21 percent prolongation of F-wave latency above the ULN in two nerves, or>50 percent if the amplitude of the distal negative peak CMAP is <80 percent of the LLN.
Absence of F-waves in two nerves, if these nerves have amplitudes of distal negative peak CMAPs ≥21 percent of the LLN, plus at least one other demyelinating parameter (meeting any of the definite criteria) in at least one other nerve
Partial motor conduction block, defined by a ≥51 percent amplitude reduction of the proximal negative peak CMAP relative to distal, if distal negative peak CMAP is ≥21 percent of the LLN, in two nerves, or in one nerve plus at least one other demyelinating parameter (meeting any of the definite criteria) in at least one other nerve.
Abnormal temporal dispersion, defined by a >30 percent duration increase between the proximal and distal negative peak CMAP in at least two nerves.
Distal CMAP duration (interval between onset of the first negative peak and return to baseline of the last negative peak) increase in at least one nerve (median ≥6.6 ms, ulnar ≥6.7 ms, peroneal ≥7.6 ms, tibial ≥8.8 ms) plus at least one other demyelinating parameter (meeting any of the definite criteria) in at least one other nerve.
Probable CIDP
≥31 percent amplitude reduction of the proximal negative peak CMAP relative to the distal, excluding the posterior tibial nerve, if the distal negative peak CMAP is ≥21 percent of LLN, in two nerves, or in one nerve plus at least one other demyelinating parameter (meeting any of the definite criteria) in at least one other nerve
Possible CIDP
As in "Definite CIDP" but in only one nerve.

❖ *Neuropathies with HIV infection:*

A.Seroconversion:

- GuillainBarre syndrome

- Chronic inflammatory demyelinating polyneuropathy (CIDP)

B.Symptomatic stage:

-Mononeuritis multiplex axonal type subacute or chroni

C.Late symptomatic stage:

- distal symmetrical sensory polyneuropathy, most commonneuropathy frequently coexists with symptomatic encephalopathy and myelopathy

-toxic polyneuropathy.

-subacute asymmetrical polyneuropathy of caudaequina, caused by cytomegalovirus

lesions localization

- 3 Functions of spinal cord : Motor, Sensory and Autonomic.

- What's unique about spinal cord lesion localization?
- What's the hallmark When you bring a patient with a spinal cord injury?
- What is the most important question?

✓ Sensory level or the motor level (the level of injury).

✓ So the hallmark for the spinal cord injury to have a motor, sensory and autonomic deficit, this is the complete classic picture.

✓ Anything affecting the legs and sparing the arm, or it has a sensory level to the nipple it means spinal cord injury.

✓ Whenever you examine a patient and you are suspecting a spinal cord injury, try to identify the spinal level of injury, whether it is sensory level, anterior or posterior.

✓ Suppose a patient have weakness in both legs and normal upper, he also has spasticity in the legs with hyper-reflexia, where is the lesion? Dorsal or thoracic. It is not lumbar, because there is no spinal cord in the lumbar area, it is caudaequina (it is nerve roots so no spinal cord injury), so it will not cause hypertonia, it will cause hypotonia – nerve root injury.

✓ If you see the legs are affected with spasticity, then you know it is CNS lesion.

✓ If the arms are spared, then you know that the C spine is spared.

✓ What's the part of the CNS is below the C spine and above the lumbar? Dorsal or thoracic spine.

✓ You can identify it by the sensory examination, you do the sensory examination and you go anterior posterior for like pinprick and temperature until you identify the level.

✓ You have to go by the map for the dermatomal and sensory level.

- The level for the nipple is T4 – T5.
- The level for the umbilicus is T10.

-What about anterior horn cells, where are they located?

Anterior horn of the spinal cord.

-What's the function? Motor.

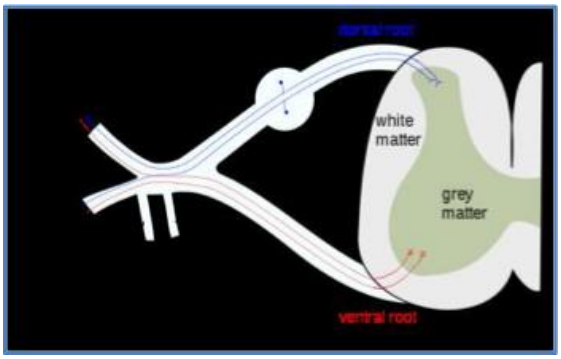
-Is it upper motor neuron or lower motor neuron?

Of course lower motor neuron.

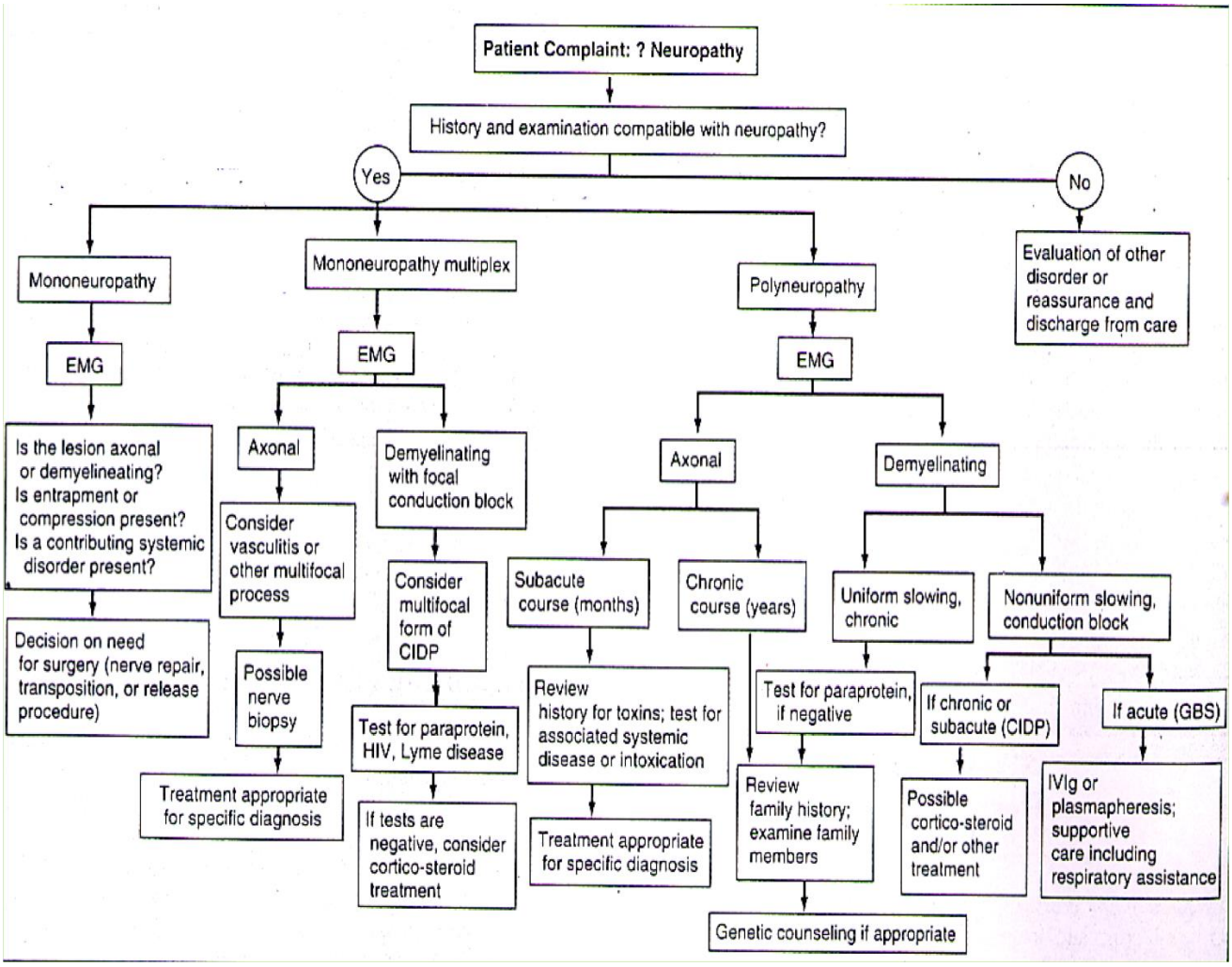
-When we say first and second order neuron, suppose I want to move my thumb here, the order has to come from the motor cortex in my right hemisphere, it will go down through the brainstem until it reaches C6 in the anterior horn level in the c spine (neck), then the anterior horn cell from there will make the radial nerve to the extension of the thumb. So all what I need is two levels, only single synapse in the spinal cord.

-What's the hallmark of anterior horn cell lesion or pathology like lower ALS (Amyotrophic lateral sclerosis) – common disease in the states- ?Flaccidity and muscle atrophy and sensory examination will be normal.

This is the way you can differentiate between anterior horn cells or motor neuron disease from peripheral neuropathy. The sensory examination is normal, but in different neuropathy we will see motor and sensory deficit going with single peripheral nerve distribution or multiple nerve distribution. In anterior horn cells you will see pure weakness, fasciculation and atrophy, but sensory examination is totally normal, why? (see the pic.).



Summery


Patient Complaint: ? Neuropathy
History and examination compatible with neuropathy?
Yes
No
Evaluation of other disorder or reassurance and discharge from care
Mononeuropathy
Mononeuropathy multiplex
Polyneuropathy
EMG
EMG
EMG
Is the lesion axonal or demyelineating? Is entrapment or compression present? Is a contributing systemic disorder present?
Decision on need for surgery (nerve repair, transposition, or release procedure)
Axonal
Consider vasculitis or other multifocal process
Possible nerve biopsy
Treatment appropriate for specific diagnosis
Demyelinating with focal conduction block
Consider multifocal form of CIDP
Test for paraprotein, HIV, Lyme disease
If tests are negative, consider cortico-steroid treatment
Axonal
Demyelinating
Subacute course (months)
Chronic course (years)
Review history for toxins; test for associated systemic disease or intoxication
Treatment appropriate for specific diagnosis
Uniform slowing, chronic
Nonuniform slowing, conduction block
Test for paraprotein, if negative
If chronic or subacute (CIDP)
If acute (GBS)
Review family history; examine family members
Possible cortico-steroid and/or other treatment
IVIg or plasmapheresis; supportive care including respiratory assistance
Genetic counseling if appropriate

MCQs

1- A 38 year-old carpenter comes with pain near his ear that is quickly followed by weakness of one side of his face. Both the upper and lower parts of his face are weak, but sensation is intact.

What is the most common complication of his disorder?

A. Corneal ulceration
B. Aspiration pneumonia
C. Sinusitis
D. Otitis media
E. Deafness
F. Dental caries

2- During an industrial accident, a sheet metal worker lacerates the anterior surface of his wrist at the junction of his wrist and hand. Examination reveals no loss of hand function, but the skin on the thumb side of his palm is numb. Branches of which nerve must have been severed?

A. Lateral antebrachial cutaneous
B. Medial antebrachial cutaneous
C. Median
D. Radial
E. Ulnar

3- In a patient with Erb-Duchenne palsy, a nerve arising from the superior trunk of the brachial plexus is nonfunctional. This nerve is the:

F. Suprascapular
G. Dorsal scapular
H. Long thoracic
I. Lateral pectoral
J. Medial pectoral

4- The following may be features of a Bell's palsy, except:

A. Pain
B. Hyperacusis
C. Loss of taste in anterior part of the tongue
D. Ptosis

1-A
2-C
3-A
4-D

The explinations

1- Answer: (A) Corneal ulceration occurs with seventh cranial nerve palsy because of difficulty in closing the eye, especially at night. This leads to dryness of the eye and ulceration. This is prevented by taping the eye shut and using lubricants in the eye. Dental caries don't happen because although there is drooling from difficulty closing the mouth, saliva production is normal. Rather than deafness, sounds are extra loud. Aspiration doesn't occur because gag reflex and cough are normal.

2- The correct answer is: (C) median nerve
The median nerve provides sensory innervation to the skin of the radial 3.5 fingers of the palm. So, the patient's loss of cutaneous sensation is suggestive of a median nerve injury. The location of the injury also implies that there has been an injury to the median nerve--this nerve enters the hand by crossing over the anterior side of the wrist.

3- The correct answer is: (A)Suprascapular Remember -- Erb-Duchenne palsy is the avulsion of the C5 and C6 roots of the brachial plexus. The suprascapular nerve comes from the superior trunk of the brachial plexus to innervate supraspinatus and infraspinatus. Since the superior trunk is made entirely from the C5 and C6 nerve roots, it makes sense that the suprascapular nerve would be damaged in Erb-Duchenne palsy. The dorsal scapular nerve comes off of the C5 root to innervate levator scapulae and the rhomboids. This nerve would also be damaged in a case of Erb-Duchenne palsy, but it is not coming off of the superior trunk. (Remember: the question specified the superior trunk!) The long thoracic nerve comes off the roots of C5, C6, and C7 to innervate serratus anterior. This nerve would be affected by Erb-Duchenne palsy, but it's not from the superior trunk. Finally, the lateral and medial pectoral nerves come off the lateral and medial cords of the brachial plexus, respectively. Lateral pectoral nerve would be affected in Erb's palsy, but not medial pectoral.

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