



Peripheral Neuropathy: Clinical Approach to Diagnosis and Management

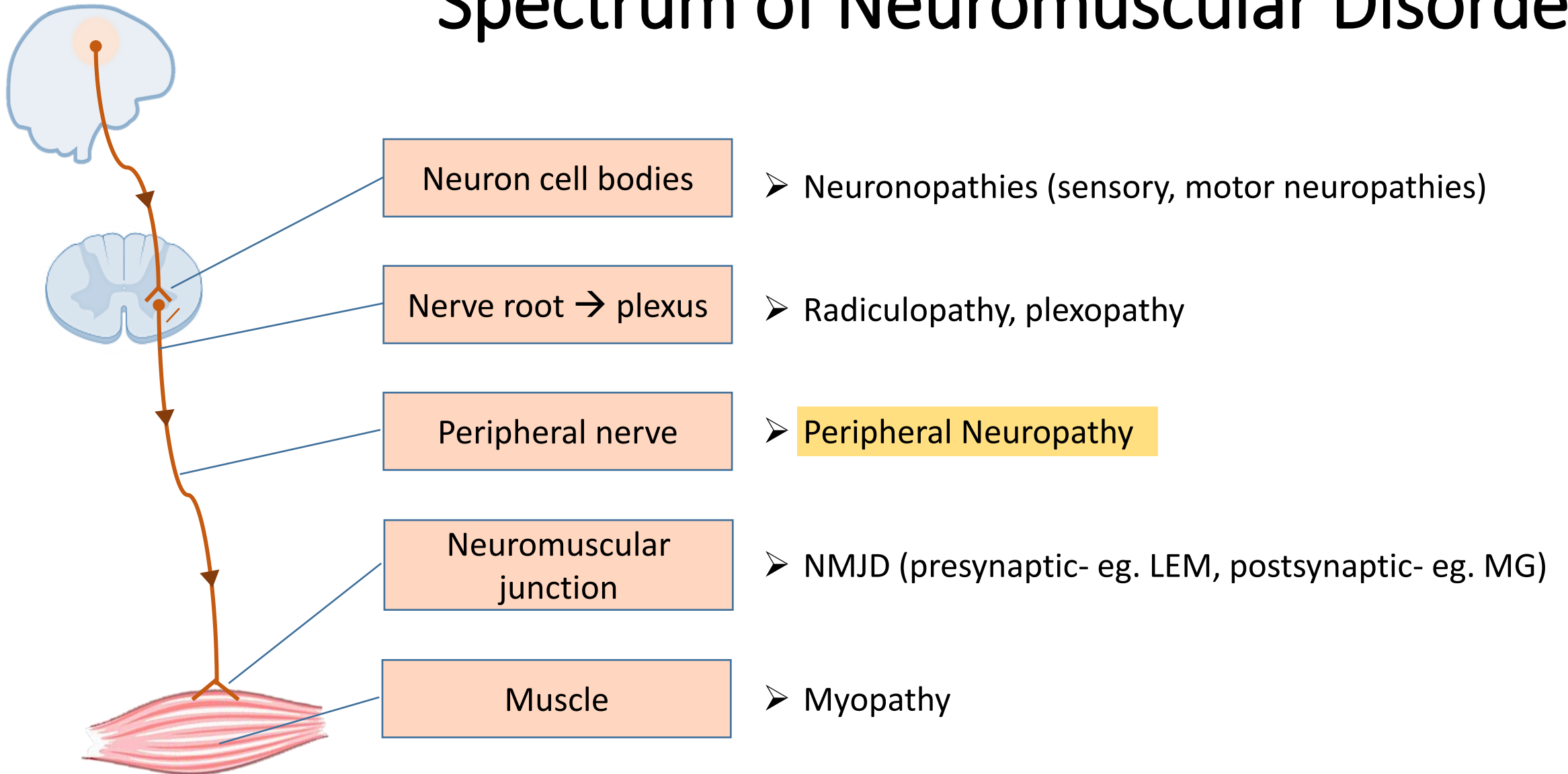
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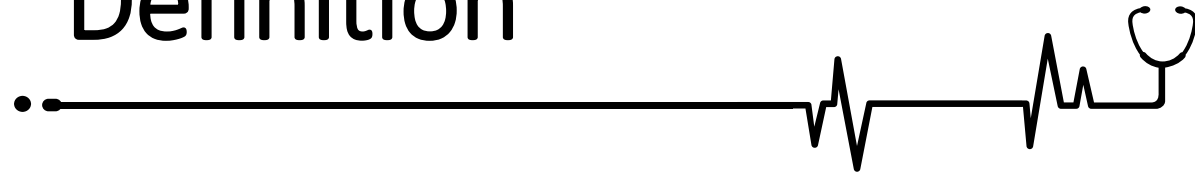
Outline

- Definition of peripheral neuropathy
- Types
- Etiology
- Clinical Approach
- Common Cases

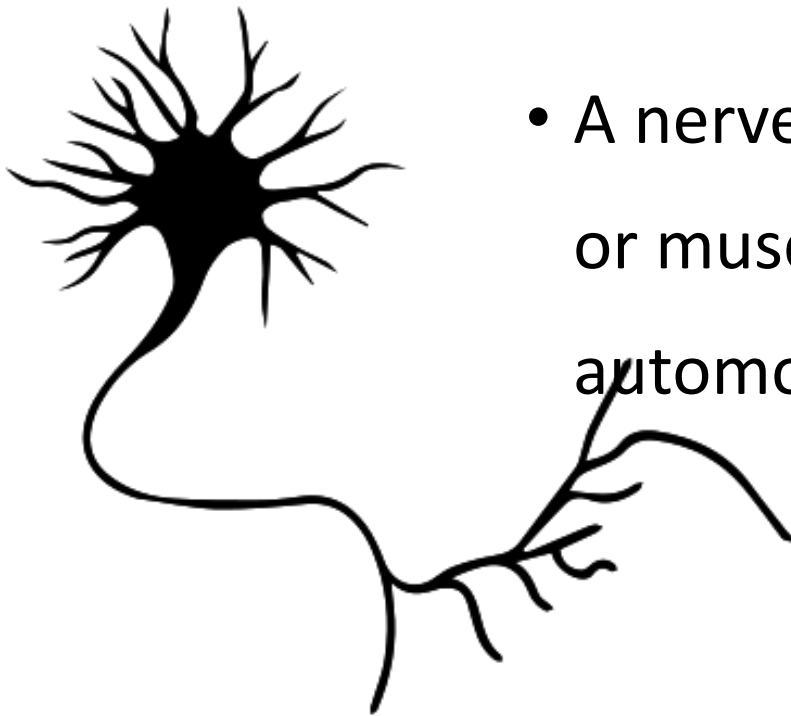
Spectrum of Neuromuscular Disorders



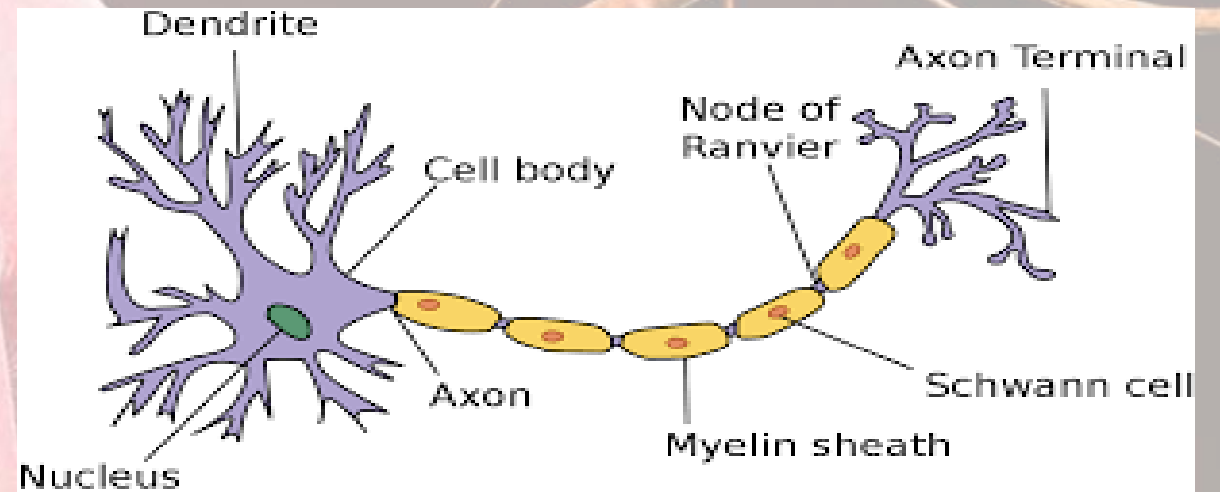
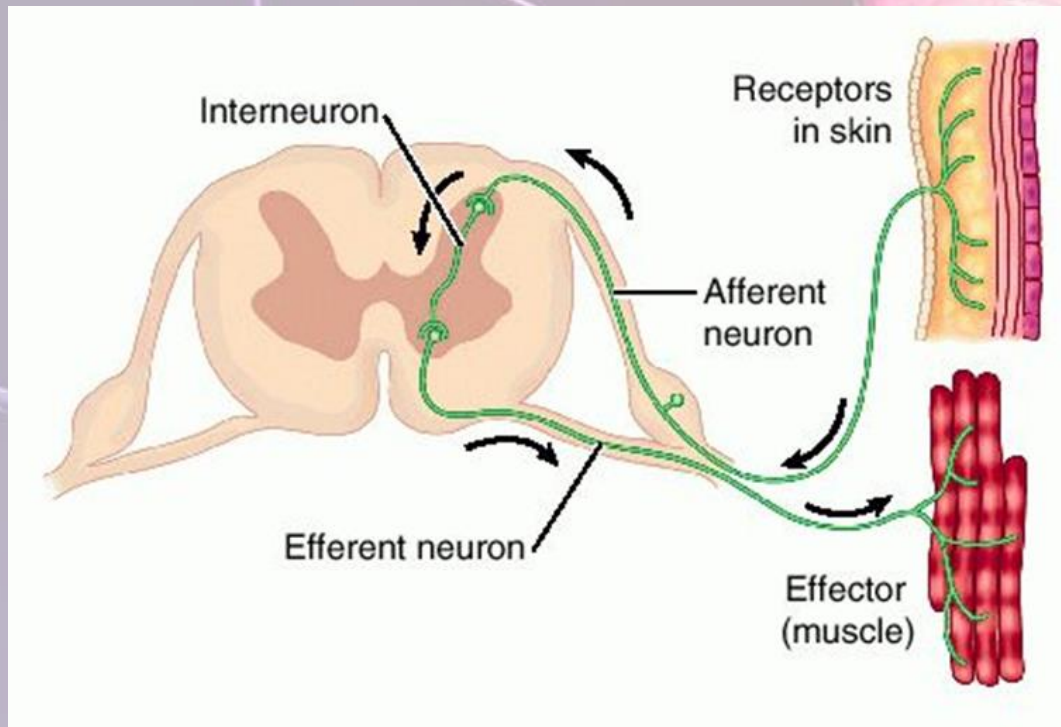
Definition



- Peripheral nerve damage causing sensory, motor, autonomic impairment
- A nerve problem that causes pain, numbness, tingling, or muscle weakness in different parts of the body or autonomic dysfunction (postural hypotension)



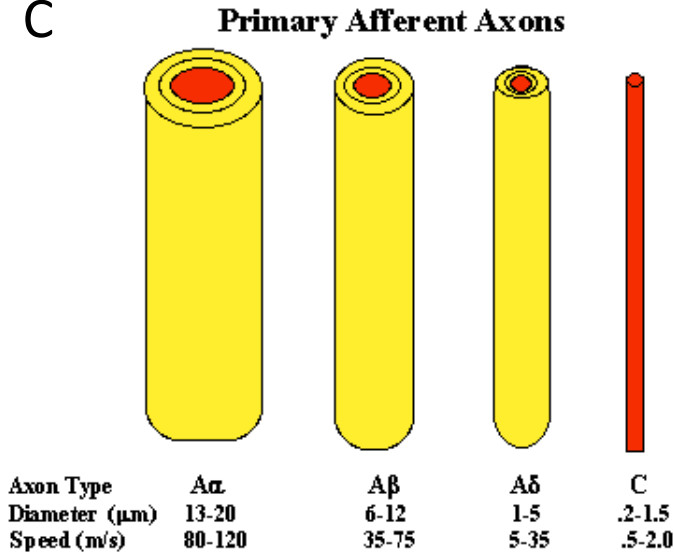
Anatomy



Nerve fibres

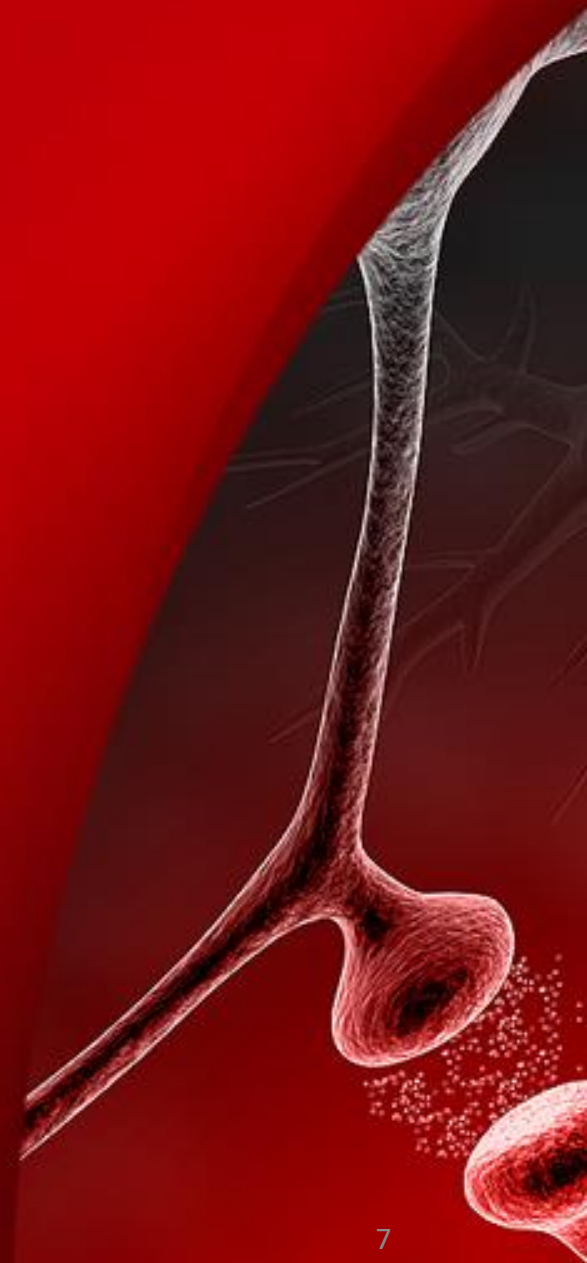


- Large nerve fibres
 - A α
 - A β
 - A γ
- Small nerve fibres
 - A δ
 - C
- B

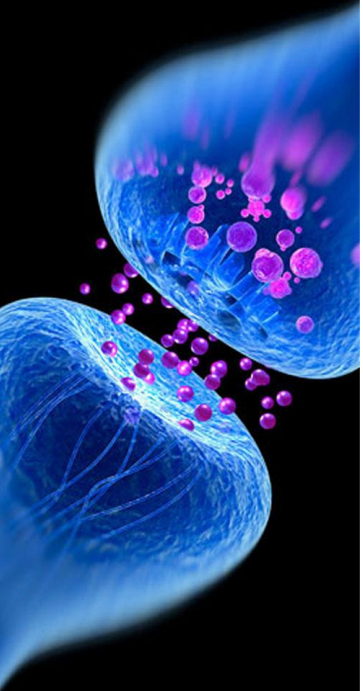
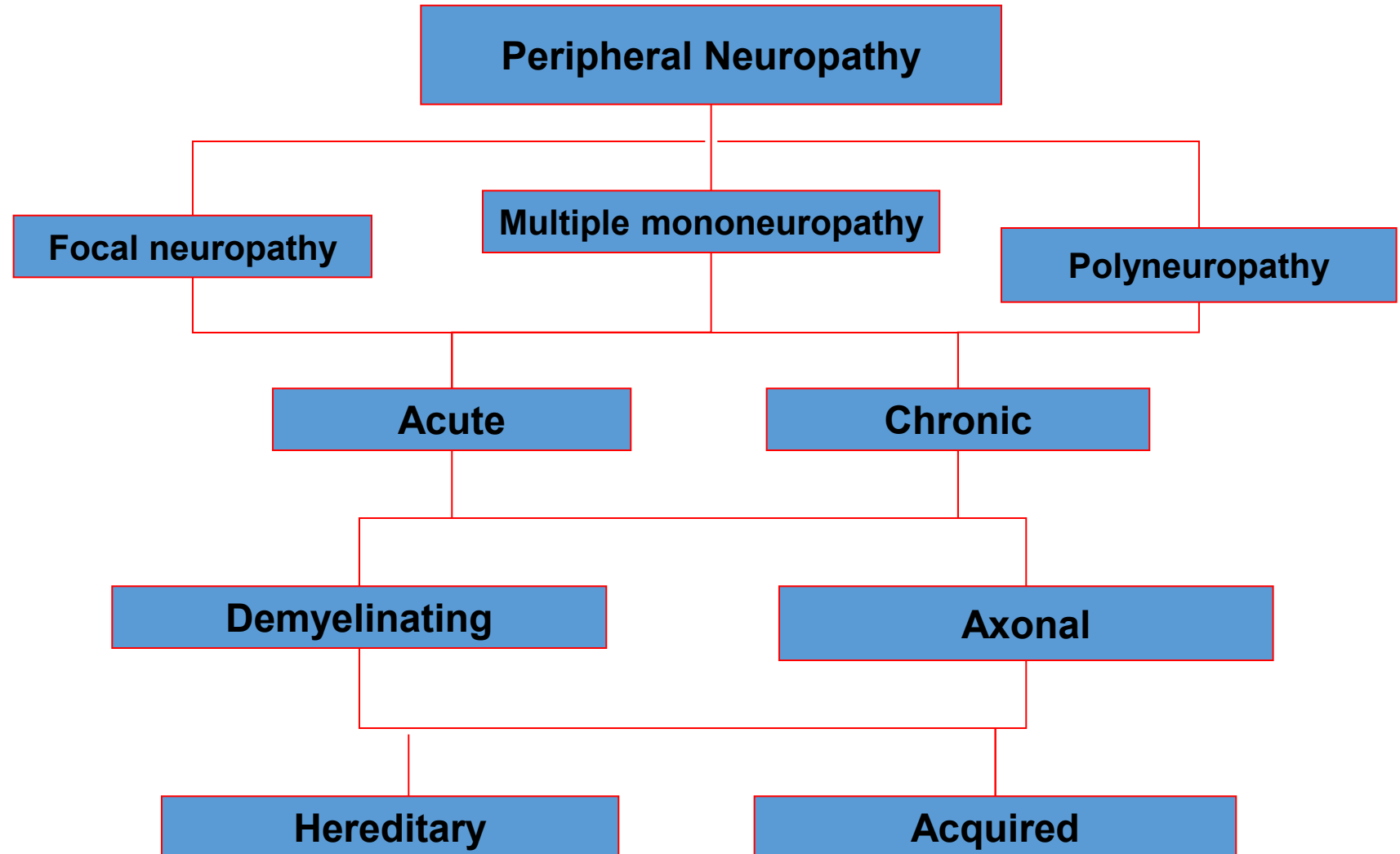


Diameter(μm)	Conduction Velocity m/s	Function
A α (1-20)	c.70	Motor: Proprioception
A β (5-10)	30-60	Touch
A γ (3-6)	20-30	Fusimotor,to spindles
A δ (2-5)	20-30	Sharp pain
B (< 3)	5-15	Autonomic, preganglionic
C (<1.3)	0.5-2	Slow pain

Types of Peripheral Neuropathy



Types of Peripheral Neuropathy



Types of Peripheral neuropathy

- Mononeuropathy (e.g., Ulnar neuropathy)
- Multiple mononeuropathies (mononeuritis multiplex)
- Symmetrical polyneuropathy (e.g., DM)
- Small fiber neuropathy (painful neuropathy)

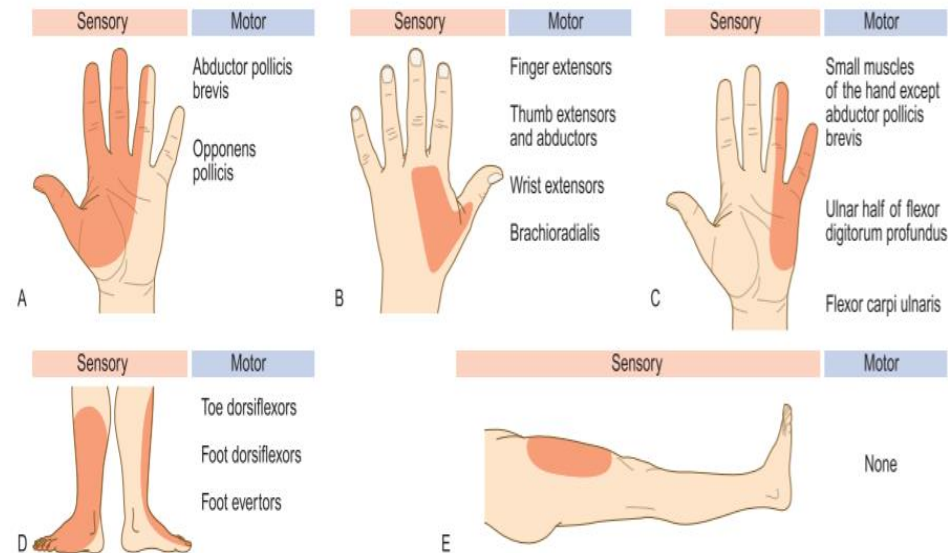
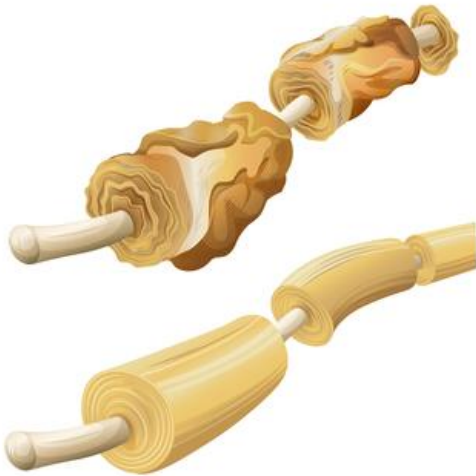


Fig. 7.27 Sensory and motor deficits in nerve lesions. **A** Median. **B** Radial. **C** Ulnar. **D** Common peroneal. **E** Lateral cutaneous of the thigh.



Acute or Chronic?

Acute polyneuropathy (days to 4weeks)

- GBS
- Acute porphyria
- Toxic neuropathy
- Critical illness neuropathy
- Vasculitis
- Infectious neuropathy (Botulism, Tick paralysis)

Chronic polyneuropathy (> 8 weeks)

- Inherited neuropathies
- Immune- CIDP, Paraproteinemia-related
- Metabolic (diabetes, hypothyroid, celiac disease)
- Alcohol, toxic (drugs, heavy metals-zinc, lead, arsenic)
- Medication (nitrofurantoin, lithium, phenytoin, B6 etc.)
- Vitamin B1, B6, B12, E, copper deficiency
- Connective tissue diseases
- Infectious (Lyme, HIV, leprosy, brucellosis)
- Sarcoid neuropathy
- Cryptogenic polyneuropathy
- Mitochondrial neuropathy

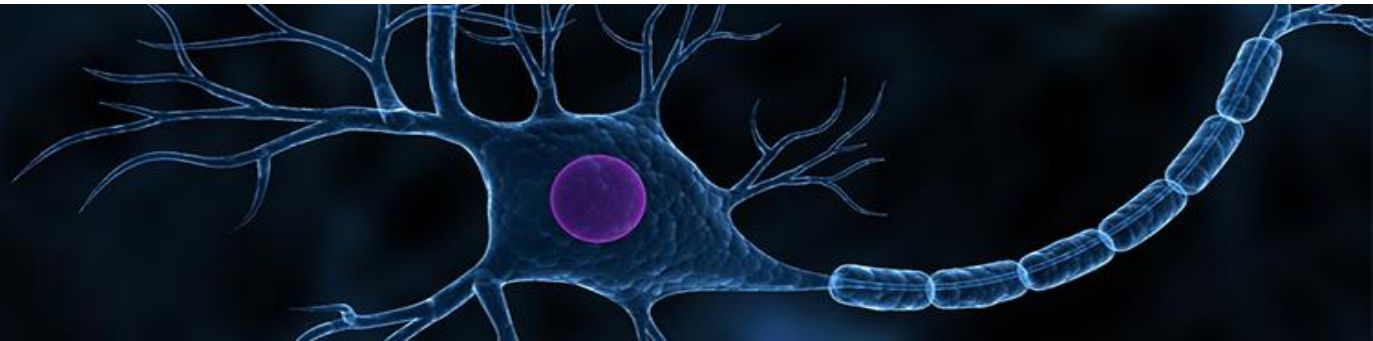
Sensory or Motor predominant?

Motor Dominant (GOLD Platinum)

- G- Guillain-Barre\$
- O- Organophosphate poisoning
- L- Lead poisoning
- D- Diphtheria
- P- Porphyria, Polio

Sensory Dominant

- A- Alcohol, Amyloid
- B- vit: B deficiency
- C- CKD
- D- Diseases(DM, HIV, Vasculitis, Sjögren's syndrome, paraneoplastic(anti-Hu))
Drugs (Cisplatin, INH)



Association

Autonomic Involvement

- GB\$
- DM
- Amyloid

Neuropathy with facial palsy

- GBS
- Familial amyloidosis
- Infection (HIV, Leprosy, Lyme disease)
- Infiltration (Sarcoidosis, Lymphoma)
- Vasculitis



Etiology



Mechanism of peripheral neuropathy

- **Axonal** degeneration e.g., systemic diseases, toxins and drugs
- **Demyelination**: damage myelin sheath (Schwann cell) e.g., GBS, CIDP, paraproteinemic
(Requires neurophysiology NCS±EMG to differentiate.)
- Wallerian degeneration (occur in axon distal to site of injury)
- Compression/entrapment e.g., Carpal tunnel syndrome
- Infarction e.g., Diabetic ischemic microvascular
- Infiltration e.g., Leprosy, sarcoid

Symmetrical Diffuse polyneuropathy

Congenital / Inherited

CMT, familial amyloid polyneuropathy

Metabolic / Endocrine

DM, CRF, B1/B12/Vitamin E deficiency, Hypothyroidism

Neoplastic / Paraneoplastic

Anti-Hu antibody associated sensory neuropathy, infiltration

Inflammatory / Infectious

GBS/CIDP, Vasculitis, HIV

Human activity

Drugs: Cisplatin, Vincristine, Taxol (S>M), Thalidomide, Pyridoxine, ddI, ddC, d4T, Chloroquine, Amiodarone, dapsone, INH, ETB, Linezolid, Metro, etc. **Toxins:** Alcohol, lead, Arsenic, Mercury, organophosphorates, Nitrous oxide



MULTIFOCAL NEUROPATHY/MONONEURITIS MULTIPLEX

i

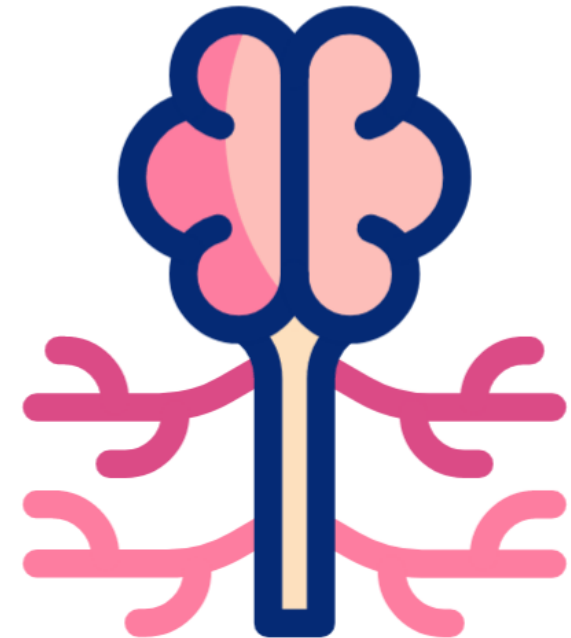
28.87 Causes of multifocal mononeuropathy

Axonal (defined on nerve conduction studies)

- Vasculitis (systemic or non-systemic)
- Diabetes mellitus
- Sarcoidosis
- Infection (HIV, hepatitis C, Lyme disease, leprosy, diphtheria)

Focal demyelination with/without conduction block

- Multifocal motor neuropathy
- Multiple compression neuropathies (usually in association with underlying disease, such as diabetes or alcoholism)
- Multifocal acquired demyelinating sensory and motor neuropathy (MADSAM)
- Hereditary neuropathy with a predisposition to pressure palsy (autosomal dominant, peripheral myelin protein 22 gene)
- Lymphoma



Mononeuropathy: Common entrapment neuropathies

- Focal compression
- Predisposing causes: diabetes, excess alcohol or toxins, or genetic syndromes, obesity, hypothyroid, Hereditary neuropathy with liability to pressure palsies (HNPP)
- Common entrapment neuropathies
 - Carpal tunnel syndrome
 - Ulnar neuropathy at elbow
 - Peroneal neuropathy at fibula neck
 - Radial neuropathy at spiral groove (Saturday night palsy)
 - Lateral femoral cutaneous nerve of thigh (Meralgia paresthetica)
 - Hereditary neuropathy with liability to pressure palsies (HNPP)
- Can recover by avoiding the precipitation of activity or by surgical decompression
- If not at common entrapment sites, alert to other causes such as neuroma, external compression, infection, infiltration, immune including vasculitis

Common Entrapment Neuropathies

Nerve	Symptoms	Muscle weakness/ muscle wasting	Area of sensory loss
Median (at wrist) (carpal tunnel syndrome)	Pain and paraesthesia on palmar aspect of hands and fingers, waking patient from sleep. Pain may extend to arm and shoulder	Abductor pollicis brevis	Lateral palm and thumb, index, middle and lateral half fourth finger
Ulnar (at elbow)	Paraesthesia on medial border of hand, wasting and weakness of hand muscles	All small hand muscles, excluding abductor pollicis brevis	Medial palm and little finger, and medial half fourth finger
Radial	Weakness of extension of wrist and fingers, often precipitated by sleeping in abnormal posture, e.g. arm over back of chair	Wrist and finger extensors, supinator	Dorsum of thumb
Common peroneal	Foot drop, trauma to head of fibula	Dorsiflexion and eversion of foot	Nil or dorsum of foot
Lateral cutaneous nerve of the thigh (meralgia paraesthetica)	Tingling and dysaesthesia on lateral border of thigh	Nil	Lateral border of thigh

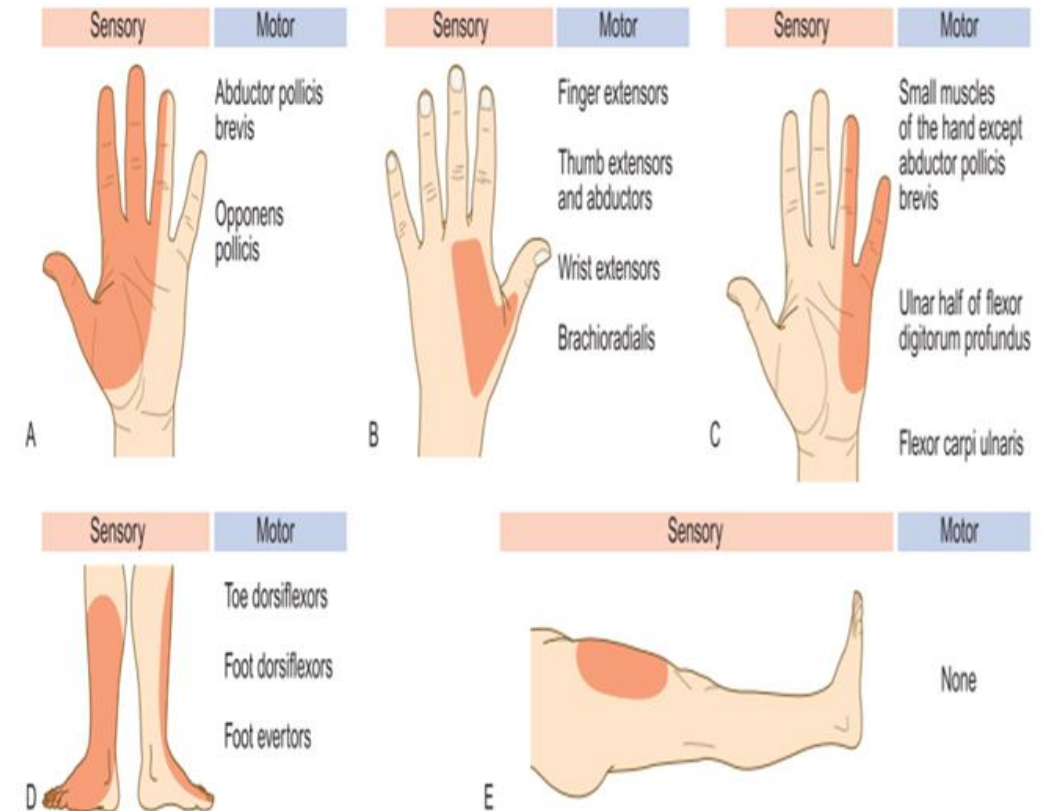
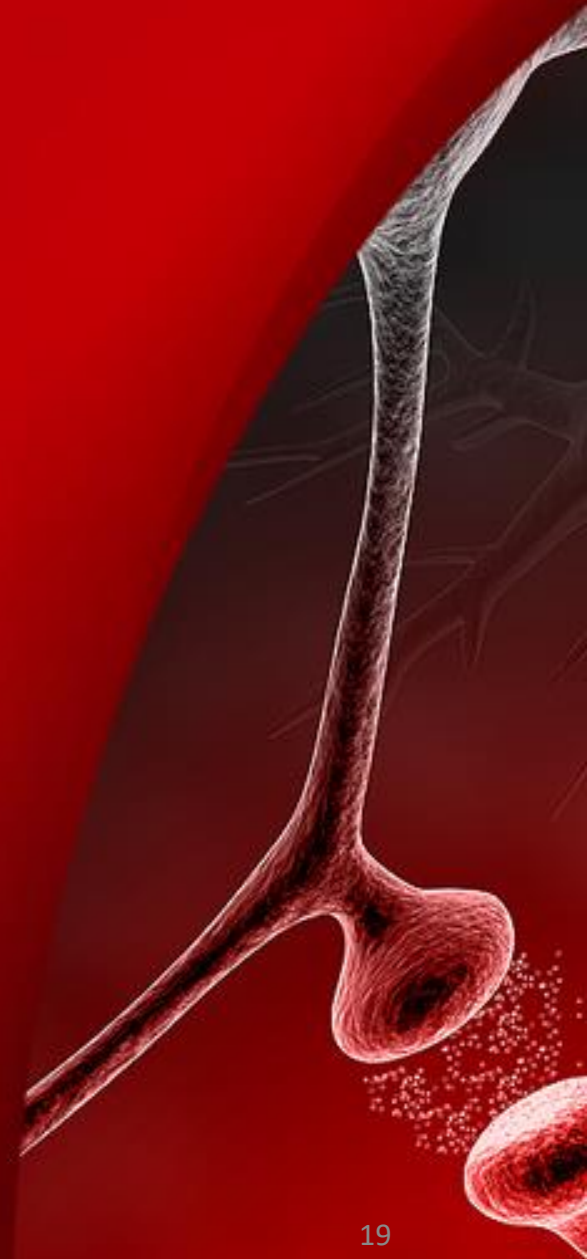


Fig. 7.27 Sensory and motor deficits in nerve lesions. **A** Median. **B** Radial. **C** Ulnar. **D** Common peroneal. **E** Lateral cutaneous of the thigh.

Clinical Approach: Peripheral Neuropathy



- Photos and videos – with the permission of the patients and their relatives
- Requested not to take photos and videos to protect the privacy of the patients



Comprehensive History Taking



- Symptom Onset: Acute (days to 4weeks), Subacute (4 to 8weeks), Chronic (> 8 weeks)
- Pattern/Distribution: Focal, Multifocal, Symmetric, Ascending vs. Descending
- Sensory Symptoms: Positive (burning, tingling), Negative (numbness, proprioception loss)
- Motor Symptoms: Weakness, cramps, atrophy
- Autonomic Symptoms: Orthostatic hypotension, bowel/bladder dysfunction
- Risk Factors: Diabetes, thyroid disorders, alcoholism, malignancy, infections

History Taking

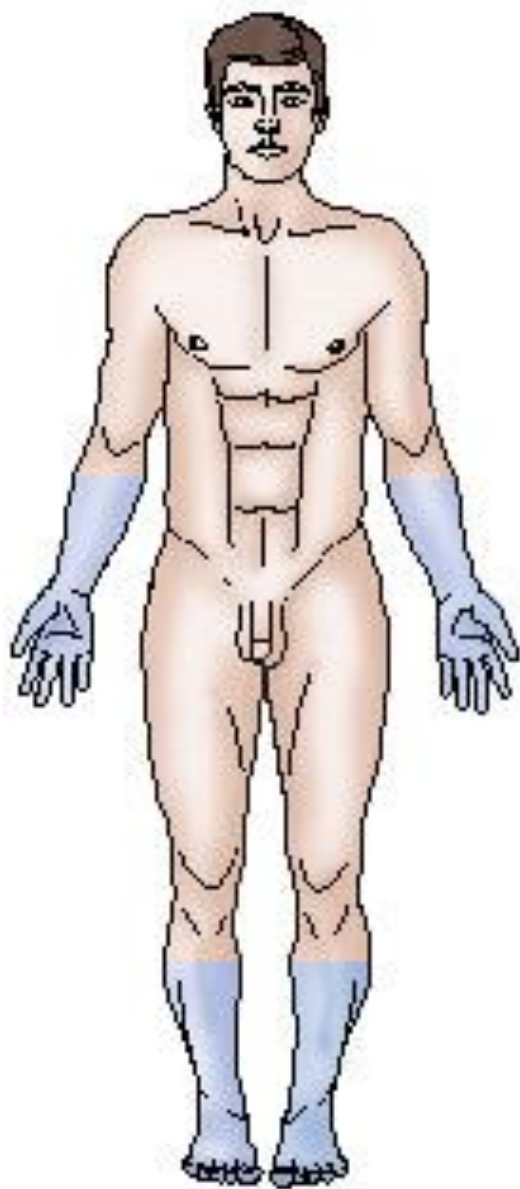
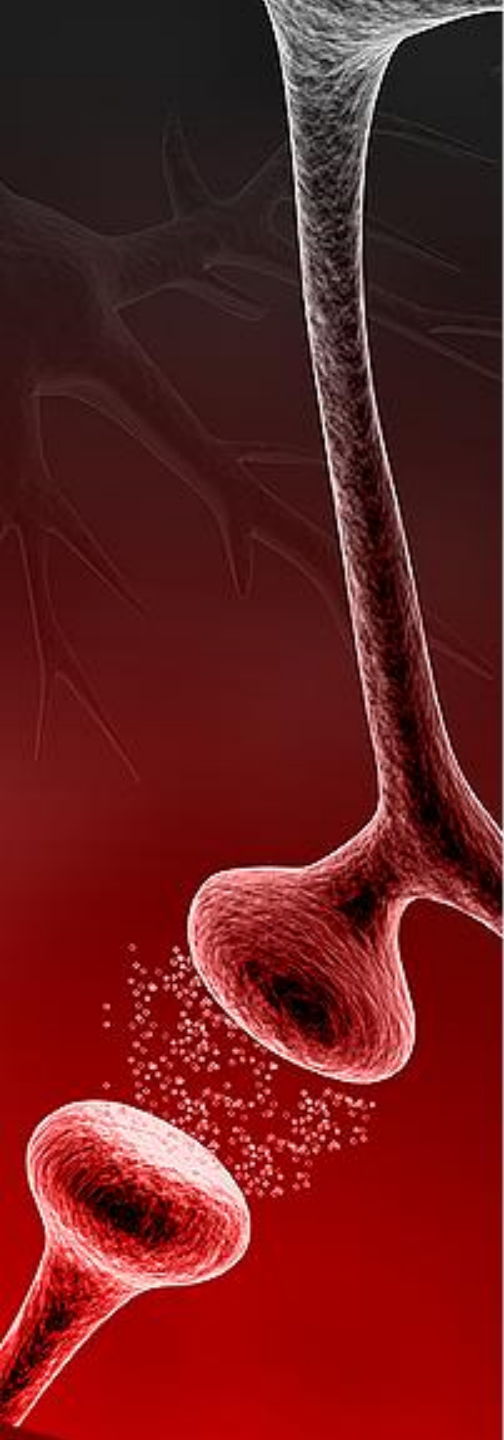
- **Medical history (complete list of medications)**
- **Detailed family history** - should include ethnic background, consanguinity, and details about parents and siblings such as the presence of (skeletal deformity)high-arched feet, flatfeet, hammertoes, “skinny legs,” gait dysfunction, and other signs of possible inherited neuropathies.
- It is helpful to examine family members for signs of polyneuropathy and associated features.
- **Social history** includes the patient’s country of origin, occupation and potential occupational exposure, sexual history, as well as alcohol and drug use.
- **Travel history** (infectious neuropathy).



Physical Examination

- General : Skin changes, muscle atrophy, fasciculations
- Neurological : Sensory, motor, reflex testing
- Autonomic : Blood pressure,
- Special Tests : Tinel's sign (Carpal-Tunnel \$), Romberg test





Distribution



Clinical examination

- Presence of **Charcot joints** (loss of pain sensation, leading to chronic trauma → Enlargement and deformities of the joints).
- **Nerve enlargement**: Ulnar nerve at the elbow, the radial nerve at the radial wrist, and the greater auricular nerve behind the ear, peroneal nerve at fibula neck, sural nerve at lateral foot)
- **Muscle atrophy**
- Joints **contractures** (clues to the chronicity and anatomic localization of the nerve dysfunction).



Wrist Drop



Foot Drop/High stepping gait,distal weakness



Distal muscle weakness/Toes weakness



Sensory examination

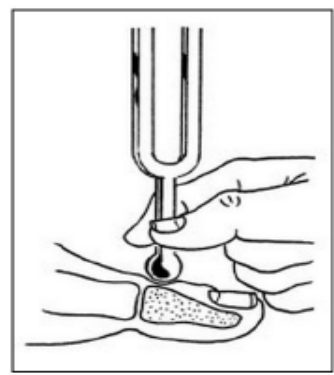
- Touch



- Temperature

- Pain

- Vibration (128 Hz tuning fork)
 - start at top of DIP and going up as necessary
 - time the duration of vibration.



- Proprioceptive sensation

Vibration perception	Result	Score
Felt > 10 seconds	Normal	0
Felt 6-10 seconds	Mild loss	1
Felt <5 seconds	Moderate loss	2
Not felt	Severe loss	3

Power Examination

Movement	Muscle	Nerve and root
Shoulder abduction	Deltoid	Axillary C5
Elbow flexion	Biceps ^a Brachioradialis (supinator reflex) ^a	Musculocutaneous C5 ^a /6 Radial C6 ^a
Elbow extension	Triceps ^a	Radial C7
Wrist extension	Extensor carpi radialis longus	Posterior interosseous C6
Finger extension	Extensor digitorum communis	Posterior interosseous C7
Finger flexion	Flexor pollicis longus (thumb) Flexor digitorum profundus (index and middle fingers) Flexor digitorum profundus (ring and little fingers)	Anterior interosseous C8 Ulnar C8
Finger abduction	First dorsal interosseous	Ulnar T1
Thumb abduction	Abductor pollicis brevis	Median T1
Hip flexion	Iliopsoas	Iliofemoral nerve L1/2
Hip extension	Gluteus maximus	Sciatic L5/S1
Knee flexion	Hamstrings	Sciatic S1
Knee extension	Quadriceps ^a	Femoral L3 ^a /4
Ankle dorsiflexion	Tibialis anterior	Deep peroneal L4/5
Ankle plantar flexion	Gastrocnemius and soleus ^a	Tibial S1 ^a /2
Great toe extension (dorsiflexion)	Extensor hallucis longus	Deep peroneal L5
Ankle eversion	Peronei	Superficial peroneal L5/S1
Ankle inversion	Tibialis posterior	Tibial nerve L4/5
^a Indicates nerve root innervation of commonly elicited deep tendon reflexes.		

Deep tendon Reflexes

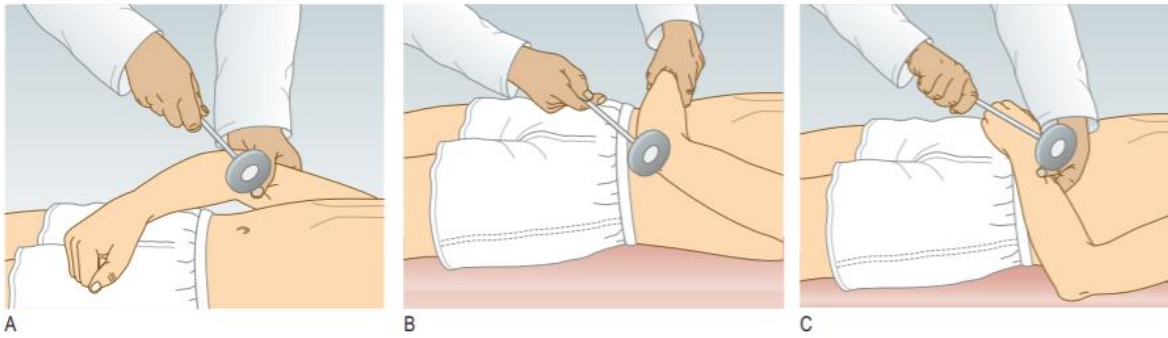


Fig. 7.19 Testing the deep tendon reflexes of the upper limb. **A** Eliciting the biceps jerk, C5. **B** Triceps jerk, C7. **C** Supinator jerk, C6.

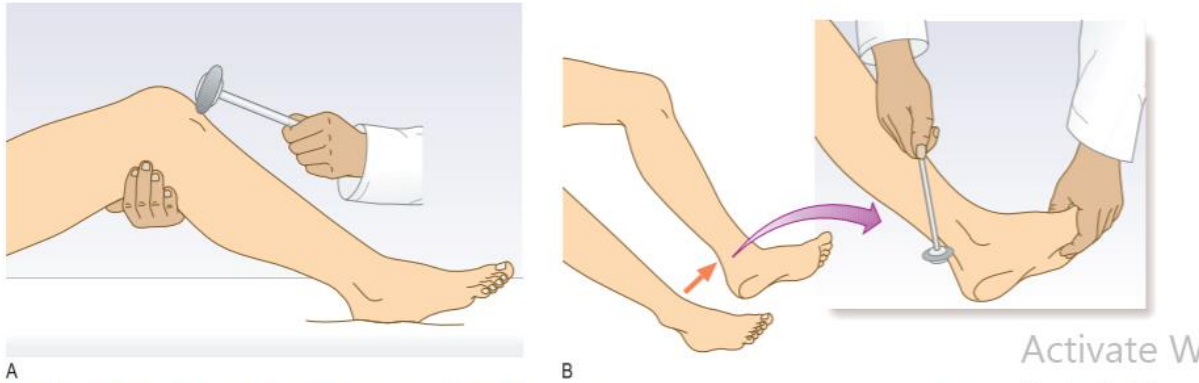
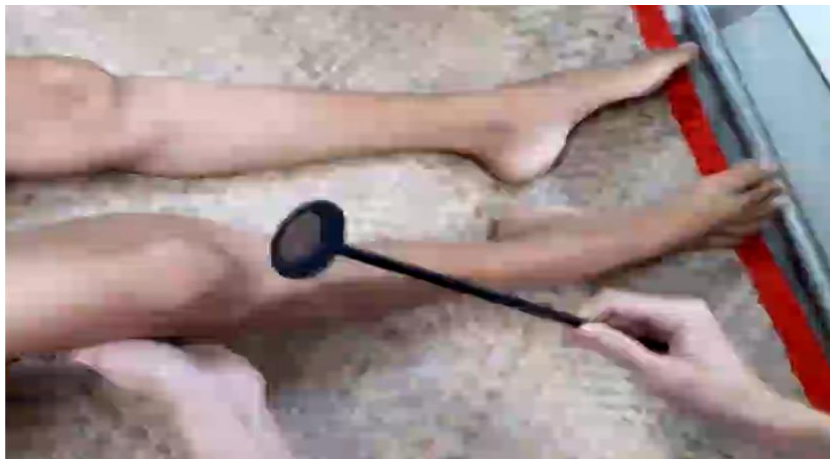


Fig. 7.20 Testing the deep tendon reflexes of the lower limb. **A** Eliciting the knee jerk (note that the patient's legs should not be in contact with each other), L3, L4. **B** Ankle jerk of the recumbent patient, S1.

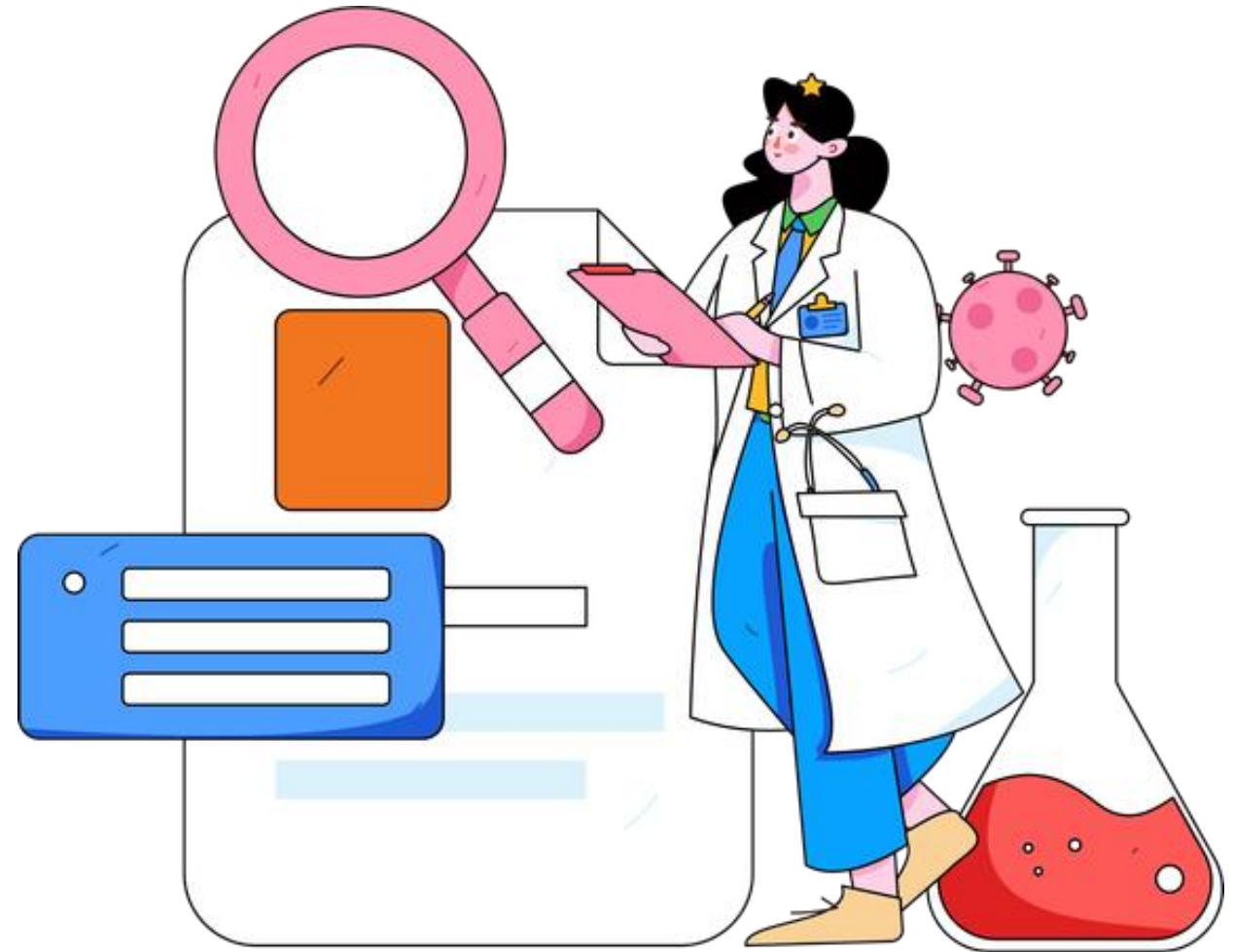


Record the response as:

- Clonus (++++)
- Increased (+++)
- **Normal (++)**
- **Decreased (+)**
- **Present only with reinforcement (+/-)**
- **Absent (0)**
- Biceps jerk -C5/6
- Supinator jerk -C5/6
- Triceps jerk -C7
- Finger jerk -C8
- Abdominal reflexes-T8-12
- Knee jerk -L3/4
- Ankle jerk -S1/2



Investigations



Investigations of Peripheral neuropathy

Initial tests

- Glucose (fasting)
- Erythrocyte sedimentation rate, C-reactive protein
- Full blood count
- Urea and electrolytes
- Liver function tests
- Serum protein electrophoresis
- Vitamin B₁₂, folate
- ANA, ANCA, ENA
- Chest X-ray
- HIV testing

If initial tests are negative

- Nerve conduction studies
- Vitamins E and A
- Genetic testing (see Box 28.83)
- Lyme serology
- Serum angiotensin-converting enzyme
- Serum amyloid

(ANCA = antineutrophil cytoplasmic antibody; ANA = antineutrophil antibody; ENA = extractable nuclear antigen)



Neurophysiology

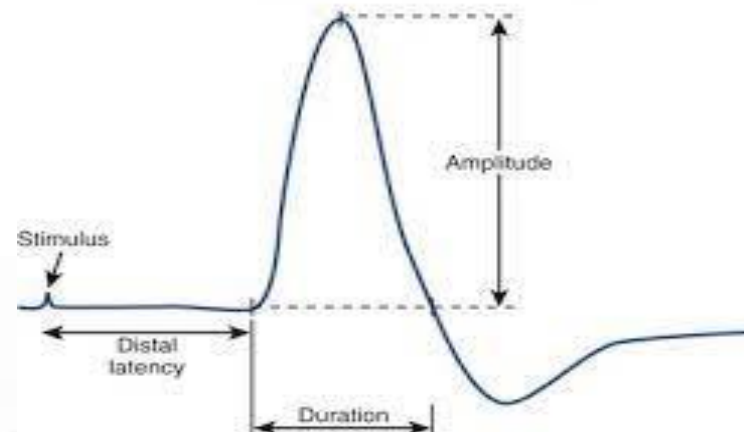


NCS machine



Role of Nerve conduction study/Electromyogram (NCS/EMG)

1. To confirm the presence or absence of large-fiber polyneuropathy
2. To identify the predominant fiber involved (sensory, motor, or sensorimotor)
3. To detect superimposed compression neuropathies or radiculopathies
4. To demonstrate the distribution (distal symmetric, multiple mononeuropathy, polyradiculoneuropathy, predominant involvement of upper or lower extremities)
5. To show symmetry or asymmetry
6. To determine the underlying pathophysiology (primary axonal or demyelinating)
7. To determine the severity
8. To monitor: stability, worsening, or improvement of the polyneuropathy
9. To monitor the effects of therapeutic interventions on the polyneuropathy



Nerve Conduction Studies

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	Norm Onset (ms)	O-P Amp (µV)	P-T Amp (µV)	Stim Site	Dist (cm)	Latency (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Anti Sensory (2nd Digit)												
Digit 2(M)		3.1	<3.6		24.8	37.6	Digit 2(M)	2nd Digit	3.1	13.0	42	
Right Median Anti Sensory (2nd Digit)												
Digit 2(M)		3.4	<3.6		23.2	34.9	Digit 2(M)	2nd Digit	3.4	13.0	38	
Left Ulnar Anti Sensory (5th Digit)												
Wrist		2.9	<3.7		17.1	25.3	Wrist	5th Digit	2.9	11.0	38	
Right Ulnar Anti Sensory (5th Digit)												
Wrist		3.0	<3.7		15.1	28.0	Wrist	5th Digit	3.0	11.0	37	
Left Sup Fibular Anti Sensory (Ant Lat Mall)												
14 cm		2.3	<4.4		15.3	11.0	14 cm	Ant Lat Mall	2.3	10.0	43	>32
Right Sup Fibular Anti Sensory (Ant Lat Mall)												
14 cm		2.2	<4.4		14.0	7.0	14 cm	Ant Lat Mall	2.2	10.0	45	>32
Left Sural Anti Sensory (Lat Mall)												
Calf		2.8	<4.0		14.8	18.3	Calf	Lat Mall	2.8	14.0	50	>35
Right Sural Anti Sensory (Lat Mall)												
Calf		2.8	<4.0		18.4	17.1	Calf	Lat Mall	2.8	14.0	50	>35

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Full Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poll Brev)													
Wrist		3.3	<4.2	10.2	>5	6.41	28.83	Elbow	Wrist	4.7	27.0	57	>50
Elbow		8.0		9.7		6.72	34.53						
Right Median Motor (Abd Poll Brev)													
Wrist		3.5	<4.2	9.7	>5	7.11	30.16	Elbow	Wrist	4.7	27.0	57	>50
Elbow		8.2		9.0		7.34	18.98						
Left Ulnar Motor (Abd Dig Minimi)													
Wrist		2.4	<4.2	8.9	>3	7.58	30.86	B Elbow	Wrist	4.1	24.5	60	>50
B Elbow		6.5		7.7		7.81	34.22	A Elbow	B Elbow	1.5	10.0	67	>53
A Elbow		8.0		7.3		7.97	25.63						
Right Ulnar Motor (Abd Dig Minimi)													
Wrist		2.7	<4.2	8.5	>3	6.33	29.38	B Elbow	Wrist	4.3	25.0	58	>50
B Elbow		7.0		8.4		6.33	22.34	A Elbow	B Elbow	1.7	10.0	59	>53
A Elbow		8.7		7.7		6.95	32.11						
Left Fibular Motor (Ext Dig Brev)													
Ankle		5.1	<6.1	3.1	>2.5	5.39	13.13	B Fib	Ankle	8.5	35.0	41	>38
B Fib		13.6		2.8		7.03	10.78	Poplt	B Fib	2.0	10.0	50	>40
Poplt		15.6		2.7		7.19	10.78						
Right Fibular Motor (Ext Dig Brev)													
Ankle		5.2	<6.1	3.3	>2.5	5.63	13.20	B Fib	Ankle	7.8	33.0	42	>38
B Fib		13.0		3.2		6.56	33.28	Poplt	B Fib	1.9	10.0	53	>40
Poplt		14.9		3.1		6.17	32.27						

Autonomic Function test

Phase I. Lying to standing

Baseline BP – 106/72 (89/min)

1 mins post standing – 63/50 (97/min) (Symptomatic)

5 mins stand - can't tolerate

30:15 heart rate ratio - cannot calculate due to frequent ectopics

Phase II. Deep breathing - performed adequately

HR on inspiration – 91, HR on expiration – 89

Phase III. Isometric exercise

Baseline – 107/75 (89/min), Post – 113/80 (90/min)

Phase IV. Valsava manœuvre – performed adequately

Phase 2 HR 88, Phase 4 HR – 91

Ratio – 1.02

Phase V. Cutaneous cold

Baseline – 107/72 (86/min)

Post – 111/72 (87/min)

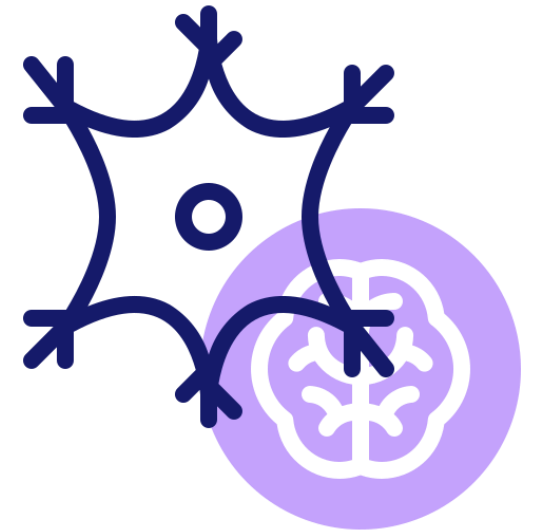
Sympathetic skin response to deep breathing, pain, noise, cough

all absent on palm and sole



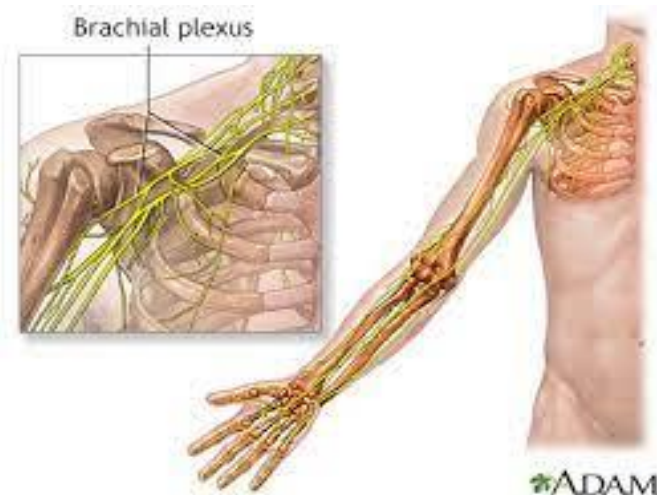
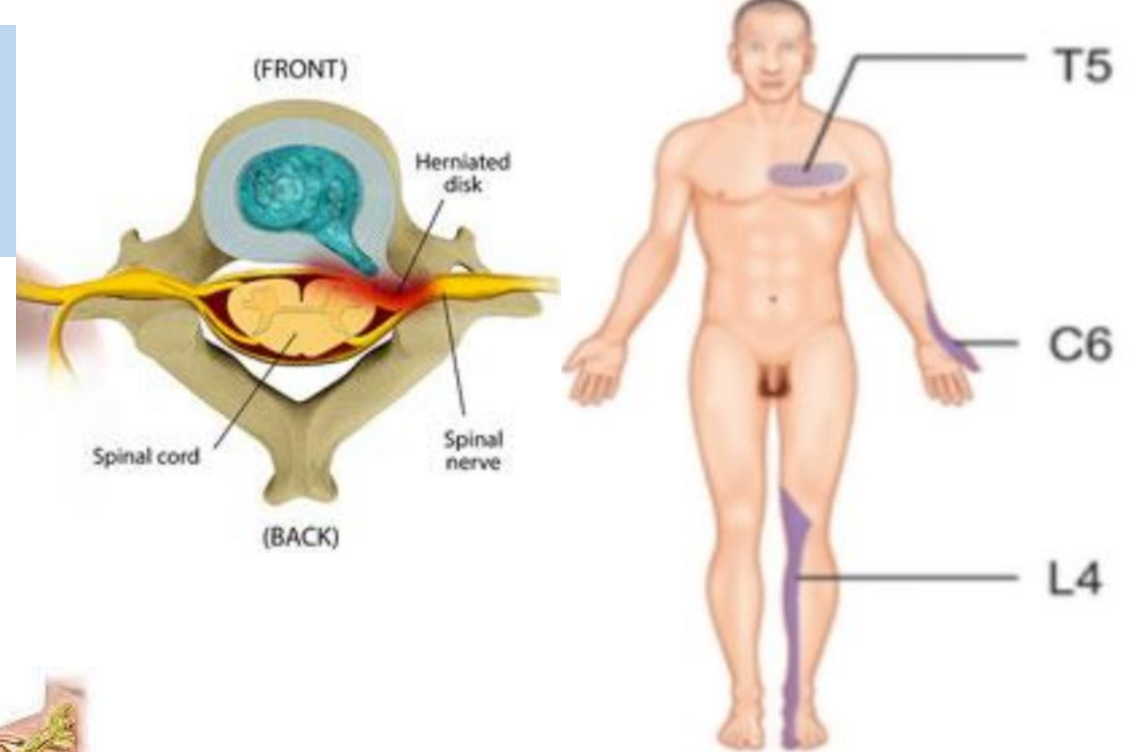
Role of Nerve biopsy

- With the current advances in neurophysiologic, immunologic, and genetic testing in peripheral neuropathy, the use of nerve biopsy is now primarily limited to patients with possible
 - Vasculitic neuropathy
 - amyloid neuropathy
 - Lymphoma
 - Hensen disease



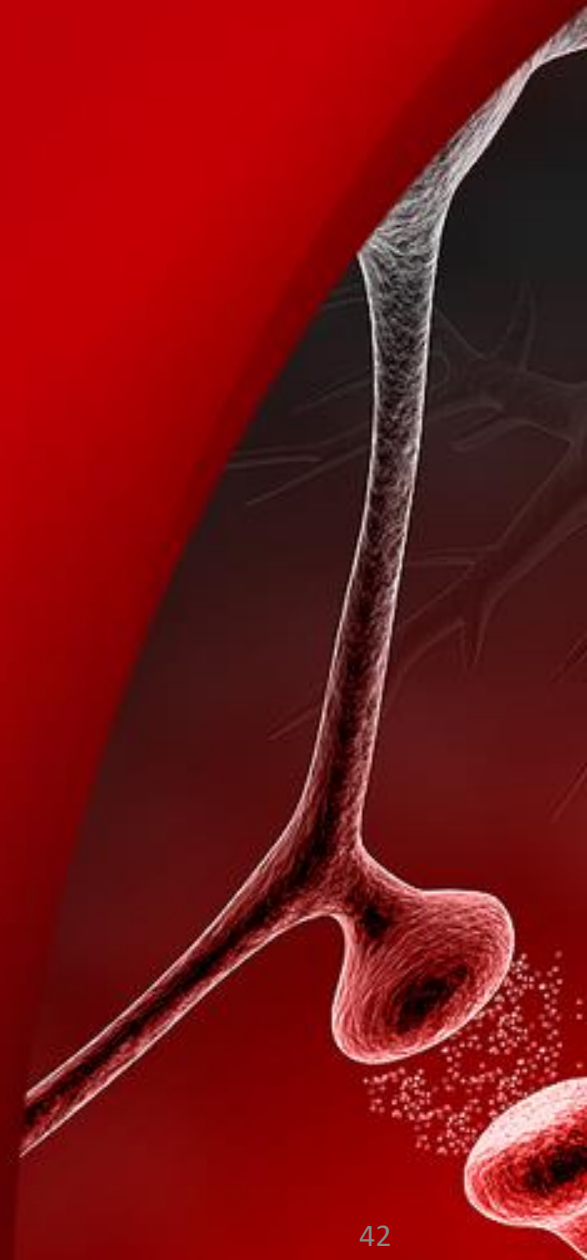
Differential diagnosis

- Bilateral lumbosacral radiculopathy
- Plexopathy
- Cervical myelopathy
- Motor neuron disease
- Myopathy



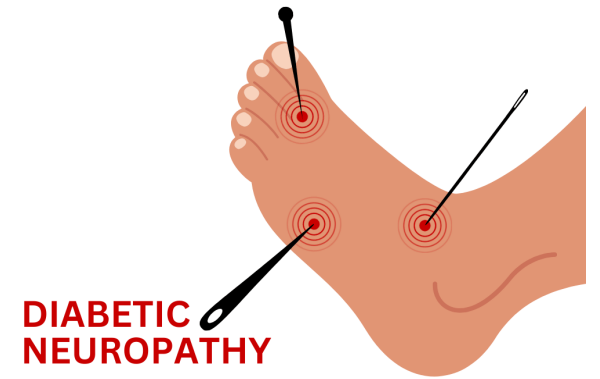
	Upper motor neurone lesion	Lower motor neurone lesion
Inspection	Usually normal (may be disuse wasting in longstanding lesions)	Muscle wasting, fasciculations
Tone	Increased with clonus	Normal or decreased, no clonus
Weakness	Preferentially affects extensors in arms, flexors in leg	Usually more focal, in distribution of nerve root or peripheral nerve
Deep tendon reflexes	Increased	Decreased/absent
Plantar response	Extensor (Babinski sign)	Flexor

Common Peripheral Neuropathy Cases



Diabetic Neuropathy

- Commonest: Distal symmetrical sensory predominant neuropathy
- Risk Factors
 - Poor blood glucose control
 - Long duration of diabetes
 - High blood pressure or cholesterol
 - Smoking or alcohol use
 - Kidney disease



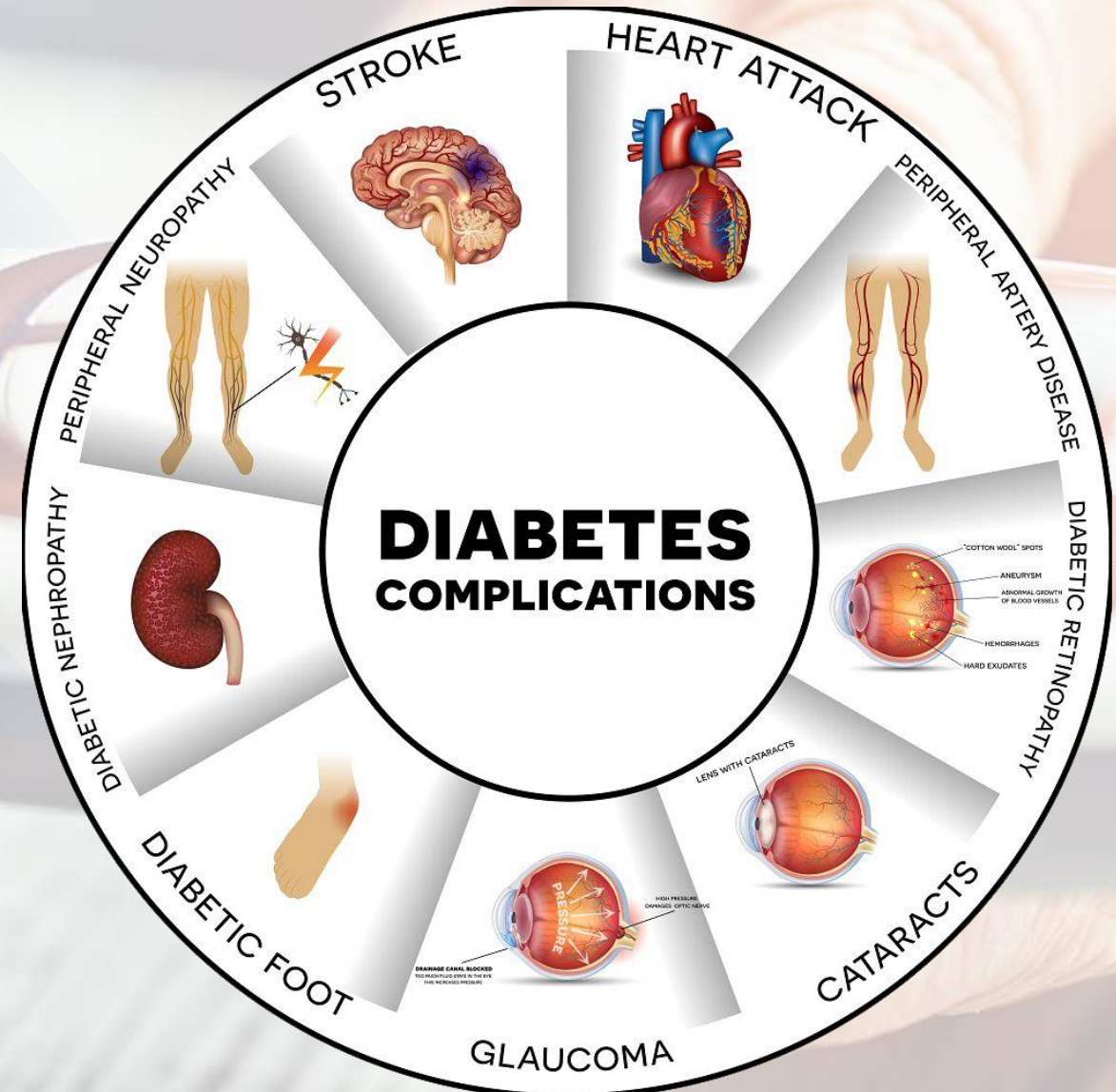
Complication of DM

Macrovascular Complication

- ✓ CVA
- ✓ IHD
- ✓ Peripheral arterial diseases

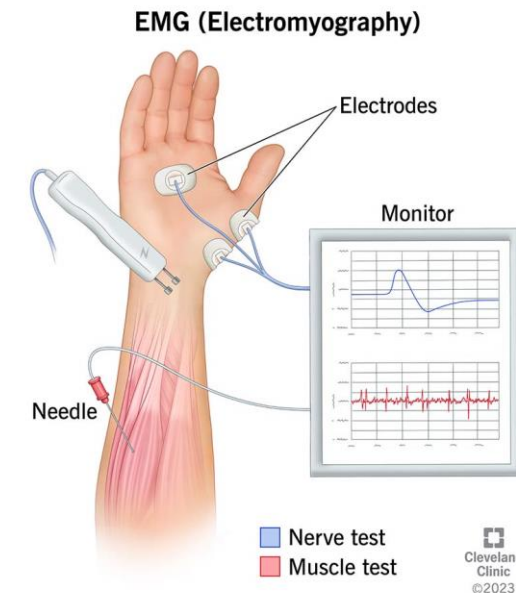
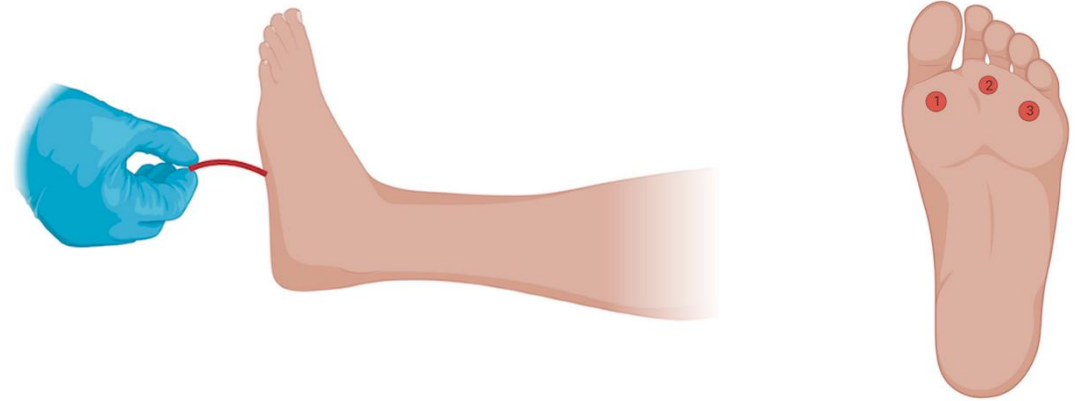
Microvascular Complication

- ✓ Retinopathy
- ✓ Nephropathy
- ✓ Neuropathy



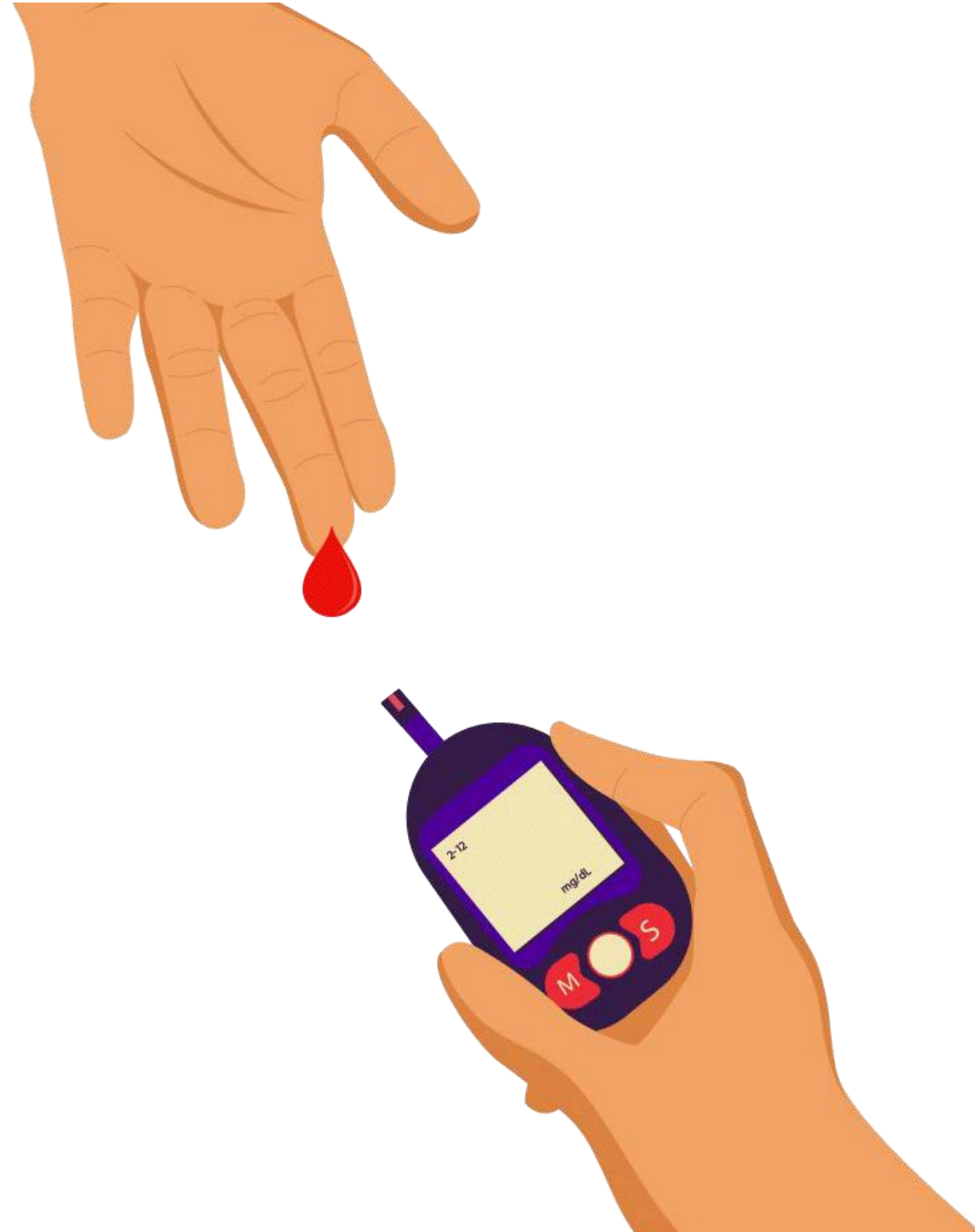
Diagnosis

- Monofilament test for touch sensation
- Vibration sense
- Nerve conduction studies(Axonal Neuropathy)



Treatment & Management

1. Blood Sugar Control: Tight glucose management slows damage
2. Pain Relief: Antidepressants, anticonvulsants, topicals, opioids



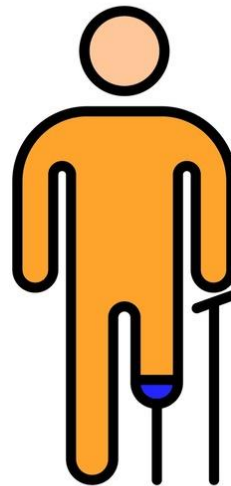
Foot Care & Lifestyle

- Foot Care: Daily inspection, proper shoes, podiatrist visits



Complications if Untreated

- Non-healing foot ulcers
- Infections and gangrene
- Risk of amputation
- Reduced quality of life and mobility





Alcoholic Neuropathy



- Chronic alcohol consumption (usually >10 years)
- Direct toxic effect of alcohol and alcohol induced malnutrition (thiamine deficiency)
- Association: Wernicke-Korsakoff syndrome, delirium tremens, alcoholic dilated cardiomyopathy
- Treatment: stop alcohol(Essential), B1 supplementation



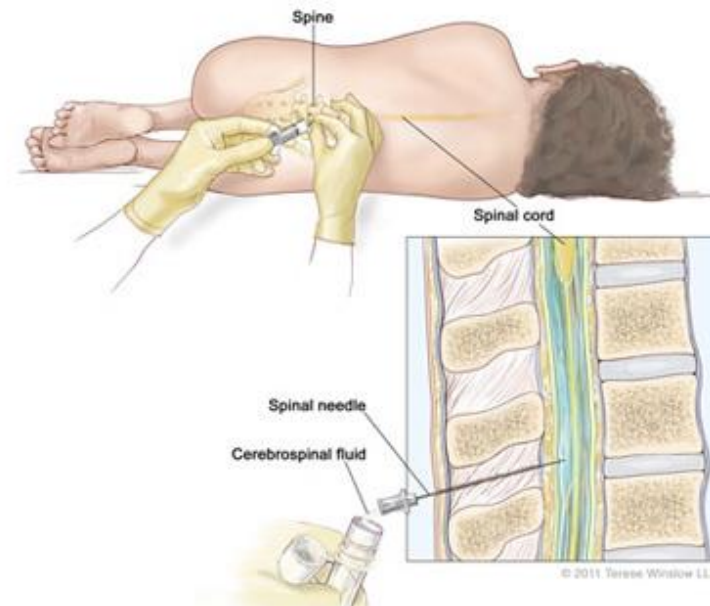
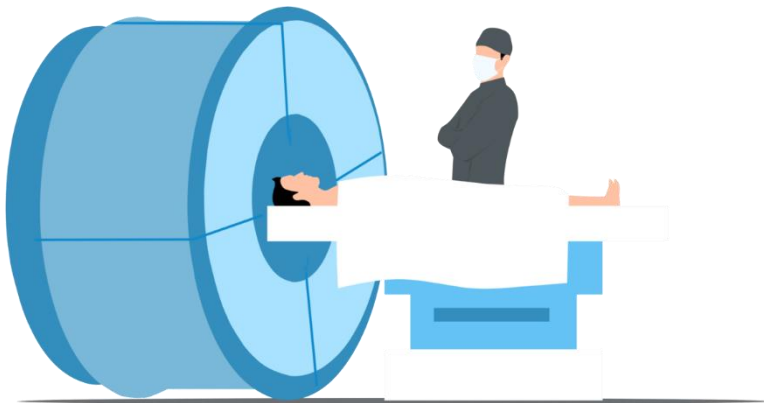
Guillain-Barre Syndrome

- Acute post infectious, immune-related inflammatory polyneuropathy (Monophasic)
- Causes rapid-onset muscle weakness, can lead to paralysis
- Often follows a viral or bacterial infection[Rarely triggered by vaccinations]
- Demyelinating or axonal
- Acute flaccid quadripareisis
- Glove and stocking sensory symptoms
- Facial, ocular, bulbar, respiratory muscles, autonomic
- Progress in days, then plateau and improves



Diagnosis

- Clinical examination (symmetry, areflexia or reduced reflexes)
- Lumbar puncture: High protein, normal WBC count
- Nerve conduction studies/EMG
- MRI: May show nerve root enhancement



Treatment

- Early recognition and treatment are crucial
- Hospitalization for monitoring and supportive care
- First-line: IVIG or plasmapheresis
- Supportive care: Ventilation (lifesaving)
- physiotherapy, pain management

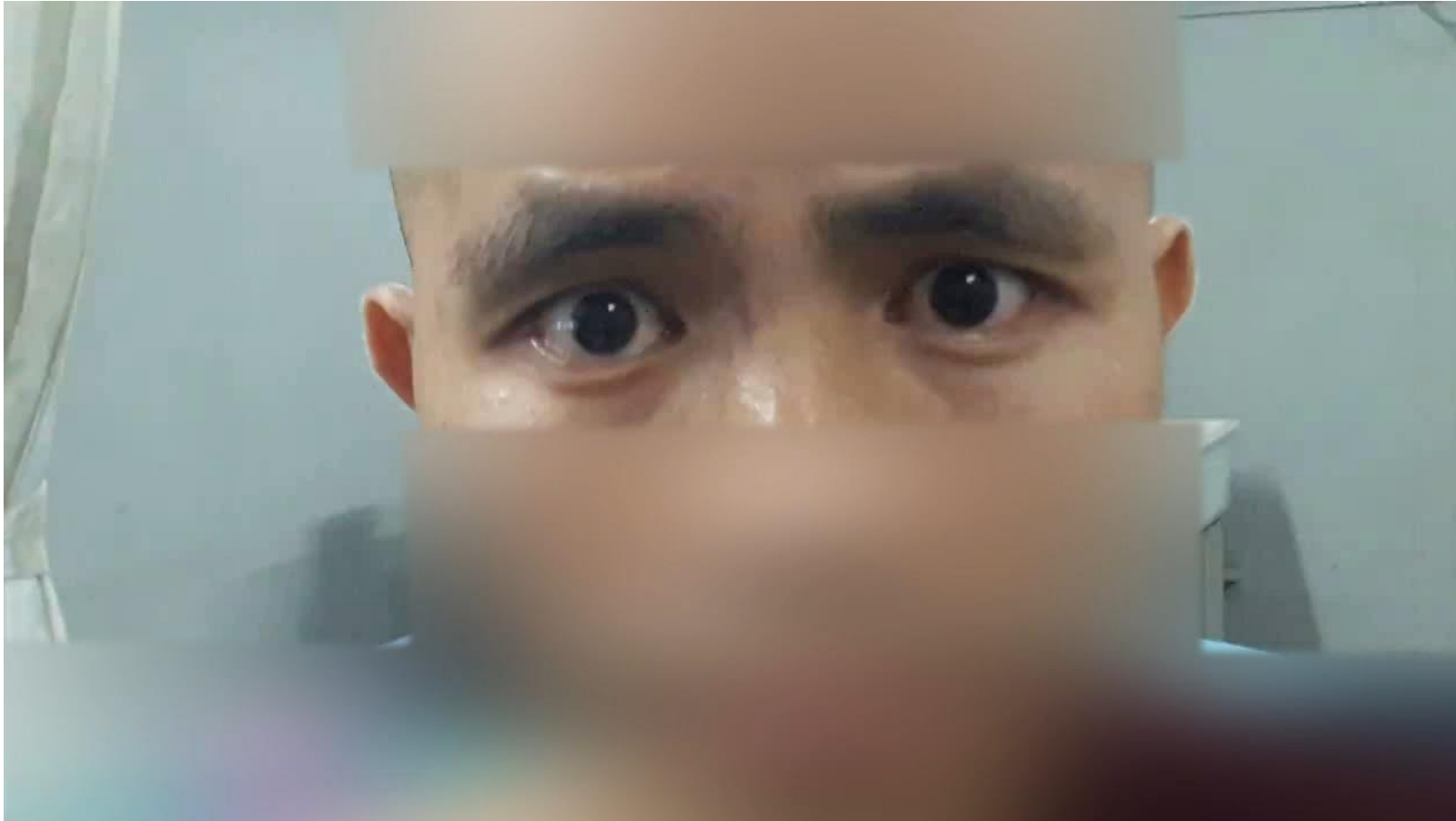


Case 1

- 26 years old monk
- C/O weakness of all 4 limbs for 10 days
- Tingling sensation in tips of toes on both sides
- Facial weakness on both sides (difficulty in closing of eyes)
- History of viral infections (fever with dry cough) 5 days ago before the onset of weakness
- Can swallow well
- No urinary and bladder dysfunction



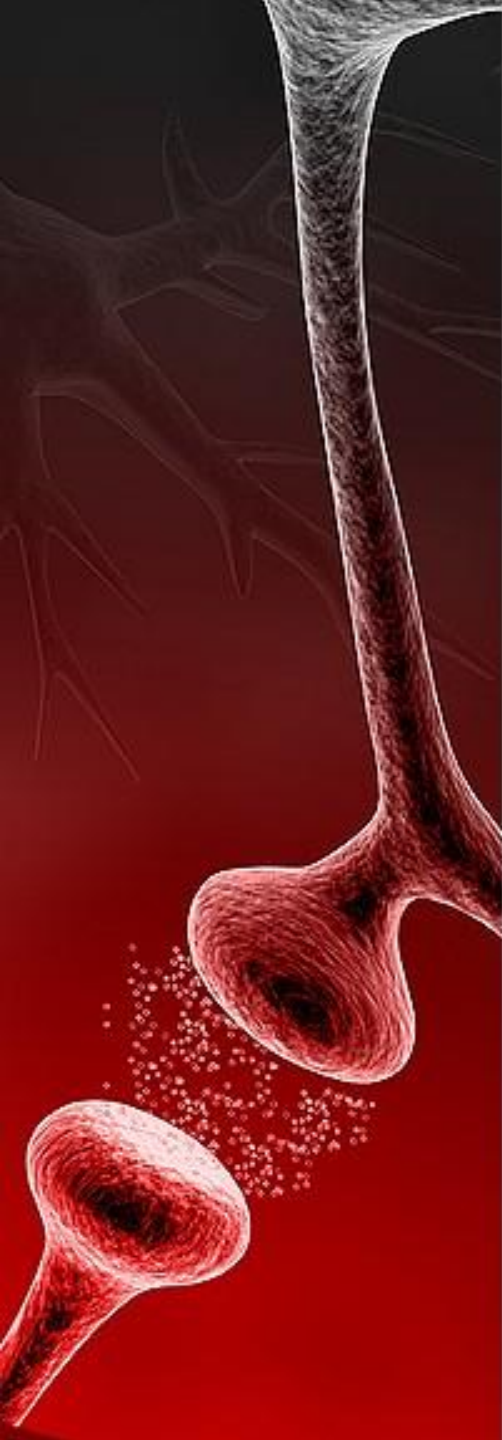
On Examination



On examination

- GCS – 15/15, afebrile
- Bilateral facial palsy(LMN type)
- No ophthalmoplegia
- Flaccid quadriparesis (UL: 3-3-3, LL: 3-2-2)
- Areflexic





CSF RE:

Pressure 12 cmH2O

CSF Protein: 142 mg/dl

Cells 4 (all lymphocytes)

Sugar 70%

Nerve Conduction Studies

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	Norm Onset (ms)	O-P Amp (µV)	P-T Amp (µV)	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Anti Sensory (2nd Digit)												
Digit 2(M)		3.4	<3.6		17.3	42.9	Digit 2(M)	2nd Digit	3.4	13.0	38	
Right Median Anti Sensory (2nd Digit)												
Digit 2(M)		3.6	<3.6		10.0	32.9	Digit 2(M)	2nd Digit	3.6	13.0	36	
Left Ulnar Anti Sensory (5th Digit)												
Wrist		3.7	<3.7		15.3	27.9	Wrist	5th Digit	3.7	11.0	30	
Right Ulnar Anti Sensory (5th Digit)												
Wrist		3.6	<3.7		8.9	9.2	Wrist	5th Digit	3.6	11.0	31	
Left Sural Anti Sensory (Lat Mall)												
Calf		2.7	<4.0		35.5	27.4	Calf	Lat Mall	2.7	14.0	52	>35
Right Sural Anti Sensory (Lat Mall)												
Calf		2.6	<4.0		36.0	36.0	Calf	Lat Mall	2.6	12.0	46	>35

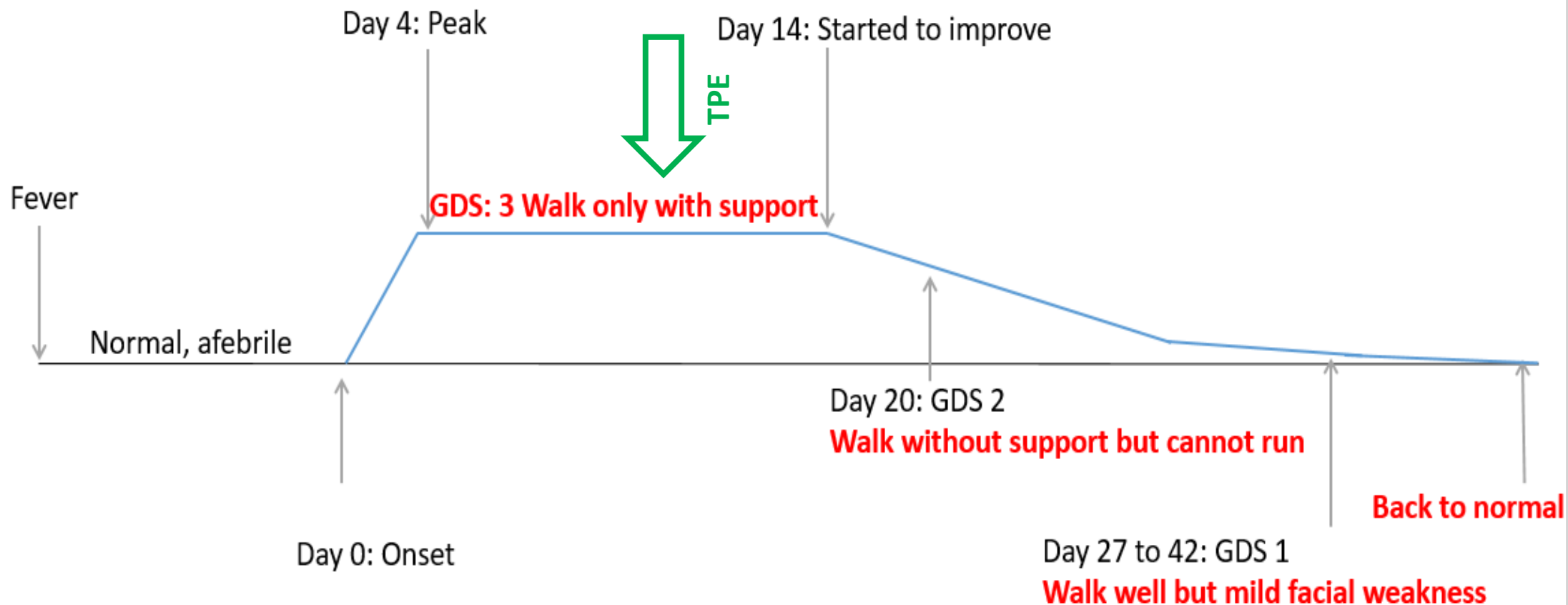
Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Full Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Polli Brev)													
Wrist		5.3	<4.2	4.4	>5	15.00	34.45	Elbow	Wrist	4.8	22.0	46	>50
Elbow		10.1		4.1		15.31	20.70						
Right Median Motor (Abd Polli Brev)													
Wrist		8.9	<4.2	2.5	>5	8.83	15.08	Elbow	Wrist	4.2	23.0	55	>50
Elbow		13.1		2.3		9.45	15.39						
Left Ulnar Motor (Abd Dig Minimi)													
Wrist		4.5	<4.2	4.5	>3	9.69	31.64	B Elbow	Wrist	3.9	20.0	51	>50
B Elbow		8.4		4.0		10.70	31.72	A Elbow	B Elbow	2.7	10.0	37	>53
A Elbow		11.1		3.2		8.59	26.02						
Right Ulnar Motor (Abd Dig Minimi)													
Wrist		4.7	<4.2	5.0	>3	7.42	9.45	B Elbow	Wrist	3.6	20.0	56	>50
B Elbow		8.3		3.7		8.13	33.98	A Elbow	B Elbow	2.6	10.0	38	>53
A Elbow		10.9		3.1		7.81	35.00						
Left Fibular Motor (Ext Dig Brev)													
Ankle		8.4	<6.1	2.0	>2.5	6.72	15.31	B Fib	Ankle	5.2	27.0	52	>38
B Fib		13.6		1.9		8.05	13.91	Poplt	B Fib	2.3	10.0	43	>40
Poplt		15.9		1.8		8.52	15.16						
Right Fibular Motor (Ext Dig Brev)													
Ankle		7.0	<6.1	1.4	>2.5	9.61	16.72	B Fib	Ankle	6.0	28.0	47	>38
B Fib		13.0		1.1		12.11	21.02	Poplt	B Fib	2.5	10.0	40	>40
Poplt		15.5		1.1		12.03	15.94						
Left Fibular TA Motor (Tib Ant)													
Fib Head		3.3	<4.2	5.2		13.52	36.41	Poplit	Fib Head	1.9	10.0	53	>40.5
Poplit		5.2	<5.7	5.1		13.83	20.55						
Right Fibular TA Motor (Tib Ant)													
Fib Head		1.8	<4.2	5.6		13.44	38.05	Poplit	Fib Head	1.9	10.0	53	>40.5
Poplit		3.7	<5.7	4.9		12.50	41.64						
Left Tibial Motor (Abd Hall Brev)													
Ankle		4.5	<6.1	8.6	>3.0	9.22	21.33	Knee	Ankle	10.6	38.0	36	>35
Knee		15.1		7.1		8.28	23.52						
Right Tibial Motor (Abd Hall Brev)													
Ankle		4.9	<6.1	9.1	>3.0	8.36	16.17	Knee	Ankle	8.5	38.0	45	>35
Knee		13.4		6.1		9.30	16.95						

Case 1

Acute flaccid quadriparesis with bilateral LMN type Facial palsy

Timeline/Disease course of a GBS case



Case 2

Slowly progressive symmetrical distal predominant weakness with glove and stocking sensation loss

Nerve Conduction Studies
Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	O-P Amp (µV)
Left Median Anti Sensory (2nd Digit)				
Digit 2 (Median)			<3.6	Absent
Right Median Anti Sensory (2nd Digit)				
Digit 2 (Median)			<3.6	Absent
Left Ulnar Anti Sensory (5th Digit)				
Digit 5 (Ulnar)			<3.7	Absent
Right Ulnar Anti Sensory (5th Digit)				
Digit 5 (Ulnar)			<3.7	Absent
Left Radial Anti Sensory (Base 1st Digit)				
Wrist			<3.1	Absent
Right Radial Anti Sensory (Base 1st Digit)				
Wrist				
Left S				
Calf				
Right				
Calf				



Toxic peripheral neuropathy due to chronic Arsenic poisoning

Motor Summary Table

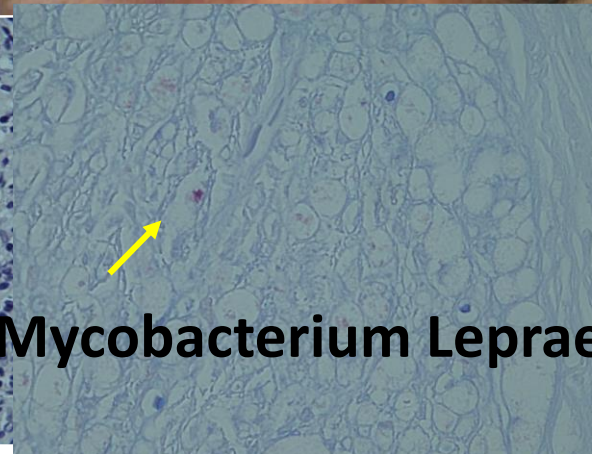
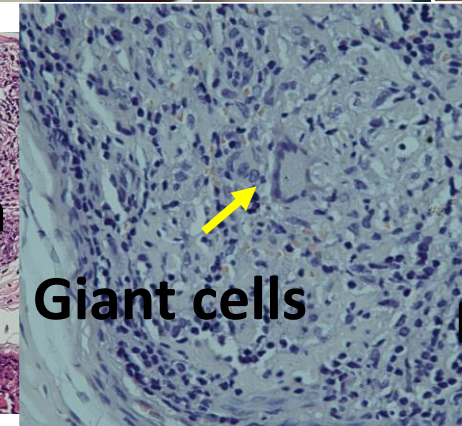
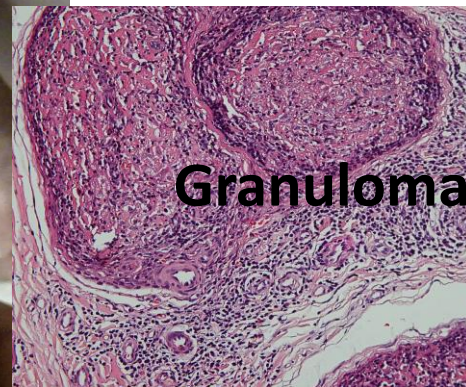
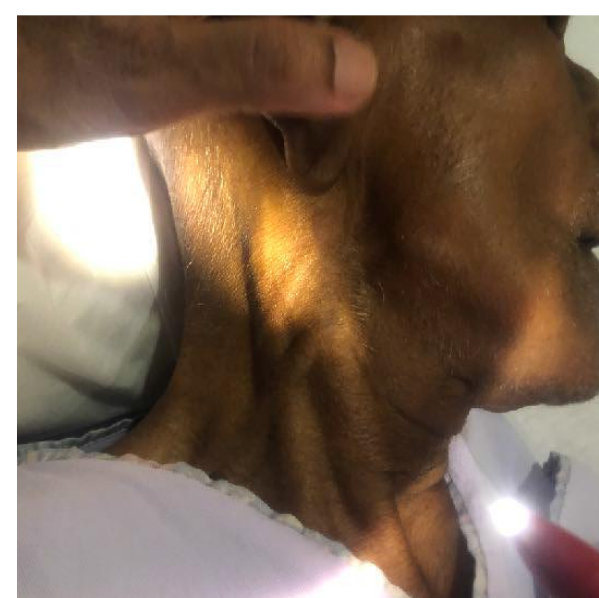
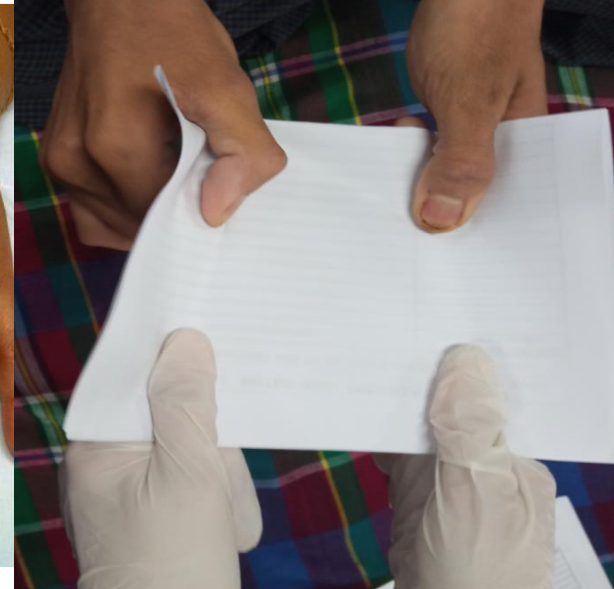
Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp
Left Median Motor (Abd Poll Brev)					
Wrist		3.2	<4.2	6.4	>5
Elbow		7.3		4.2	
Right Median Motor (Abd Poll Brev)					
Wrist		3.1	<4.2	6.7	>5
Elbow		7.8		4.7	
Left Ulnar Motor (Abd Dig Minimi)					
Wrist		2.9	<4.2	5.8	>3
B Elbow		7.0		4.3	
A Elbow		8.8		3.8	
Right Ulnar Motor (Abd Dig Minimi)					
Wrist		3.0	<4.2	4.7	>3
B Elbow		6.6		3.4	
A Elbow		8.4		2.8	
Left Fibular Motor (Ext Dig Brev)					
Ankle		3.8	<6.1	0.9	>2.5
B Fib		10.2		0.4	
Poplit		12.3		0.3	
Right Fibular Motor (Ext Dig Brev)					
Ankle		3.9	<6.1	0.8	>2.5
B Fib		11.2		0.5	
Poplit		13.4		0.4	
Left Fibular TA Motor (Tib Ant)					
Fib Head		2.9	<4.2	2.3	
Poplit		4.9	<5.7	1.9	
Right Fibular TA Motor (Tib Ant)					
Fib Head		3.3	<4.2	2.3	
Poplit		5.2	<5.7	2.0	
Left Tibial Motor (Abd Hall Brev)					
Ankle		5.3	<6.1	3.1	>3.0
Knee		15.0		1.5	
Right Tibial Motor (Abd Hall Brev)					
Ankle		4.9	<6.1	3.0	>3.0
Knee		14.8		1.6	



Hair : 46.12
mg/kg As level in
drug
-65.8 x 10³mg/kg
(Ref;value:
<5mg/kg)
Test powder with
honey 58
-365mg/kg

Case 3

Multiple mononeuropathy one limb after another



Multiple mononeuropathy due to
leprosy/Hansen's disease

Courtesy Prof T Umapathi

An abstract illustration featuring a vibrant red background. On the left side, there are several branching, tree-like structures. One structure is rendered in a light, almost white, translucent style, while others are in a darker, more textured red. These structures resemble biological forms like neurons or dendrites. A prominent, thicker, and more detailed red structure with a bulbous, rounded end is positioned in the lower center. From this bulbous end, a fine spray of small, bright red particles or droplets is being emitted, directed towards the right. The overall composition is dynamic and visually striking due to the high contrast and organic forms.

Management

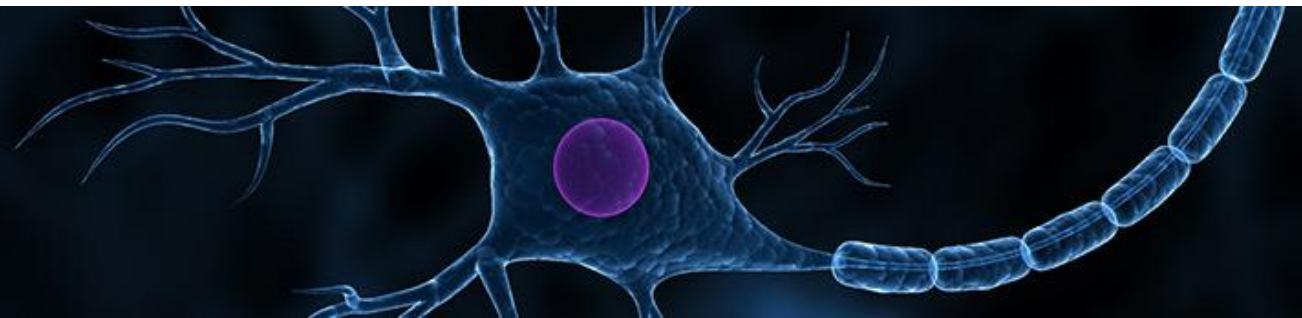
General Management

- Find underlying cause and treat
- If painful → management of neuropathic pain
 - Tricyclic antidepressants
 - Anti-epileptics
 - Tramadol
- Numbness
 - Foot care to prevent ulcers
- Weakness
 - Orthoses and walking aids
- Management of Autonomic symptoms
- Physiotherapy



Treatable Neuropathies

- Compressive neuropathies (by avoiding the precipitation of activity or by surgical decompression)
- Immune mediated neuropathies
 - GBS
 - CIDP
 - MMN
 - Vasculitic neuropathy



Immunomodulatory Therapy

- Corticosteroids
- Immunosuppressive agents
- Intravenous immunoglobulin
- Plasma exchange

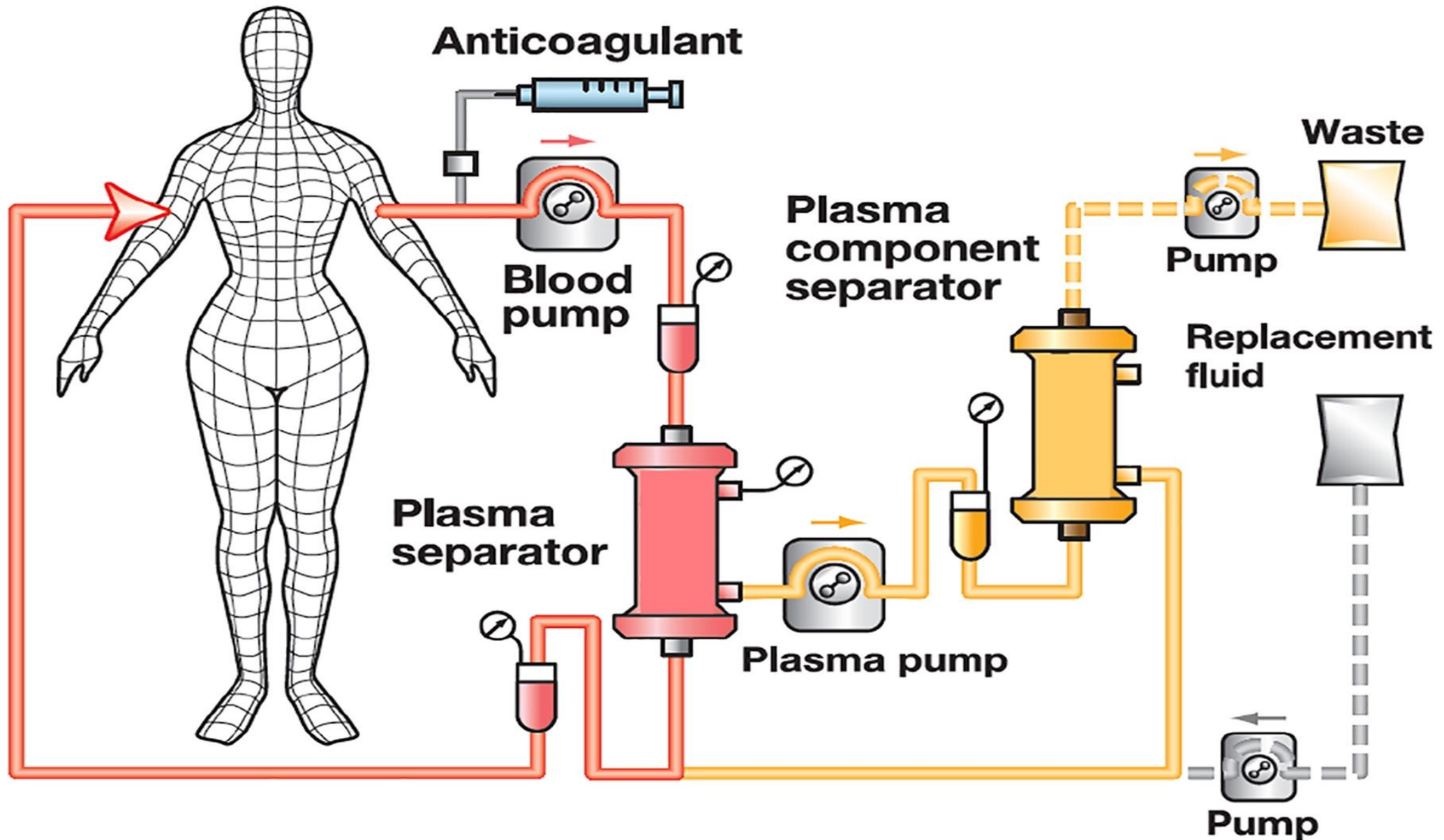


DFPP-Double Filtration Plasmapheresis

Plasmapheresis (**DFPP**) is a technique used in the treatment of some neurological autoimmune disorders.



Principles and Procedure





Take Home Message



- The diagnosis of peripheral neuropathy is not always straightforward.
- Symmetrical numbness of the feet +/- hands → make sure the patient's symptoms are due to peripheral neuropathy → search for the etiology of peripheral neuropathy.
- Peripheral neuropathy can be a manifestation of a wide range of pathologies that requires further evaluation and management.
- Early diagnosis and treatment of certain polyneuropathy can prevent further worsening of neuropathy.



Thank You

