Infectious/para-infectious (poly-)radiculoneuritis

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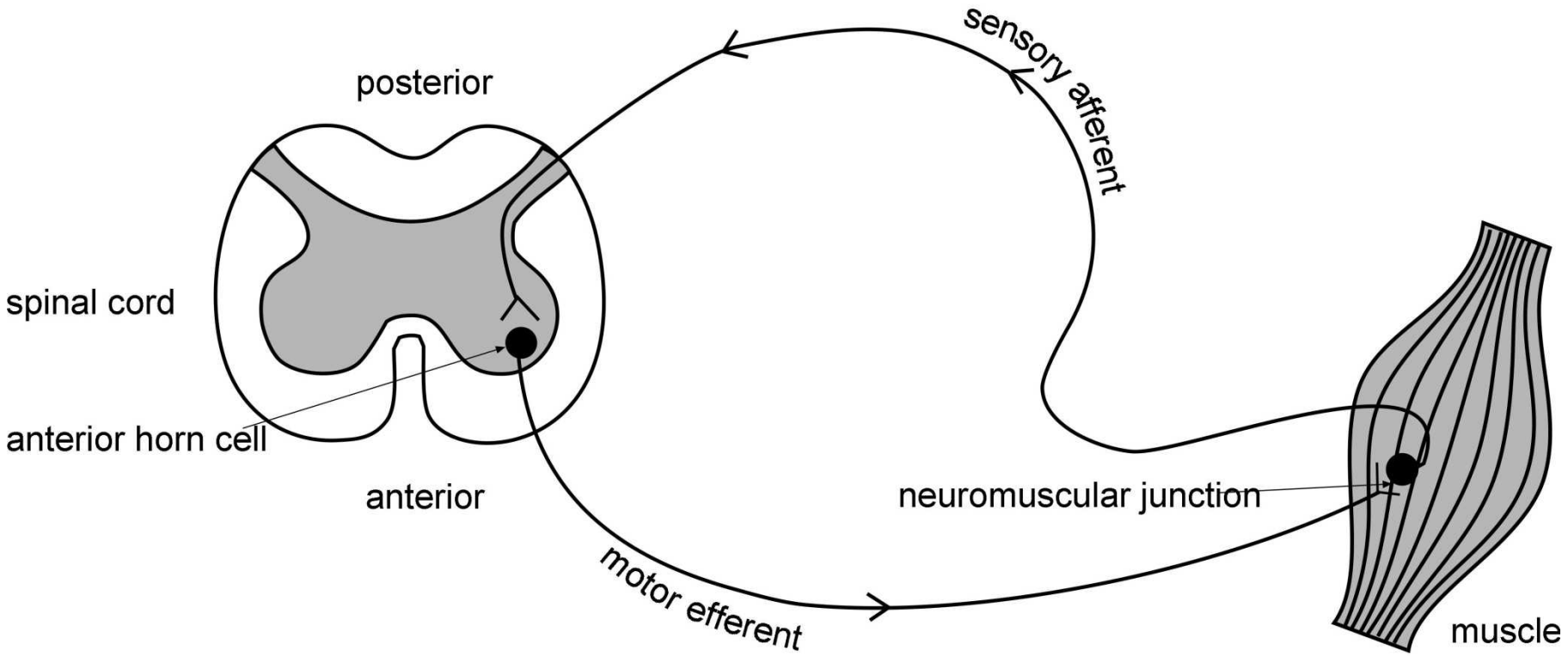
Overview

- **Anatomy** of anterior horn-radix-plexus-nerve-neuromuscular junction
- Infectious/inflammatory **causes** in Africa
- Symptoms, signs and **patterns**
- **Investigating** neuropathies
- Selected **conditions**
- **Differentials**

Types of nerve fibres

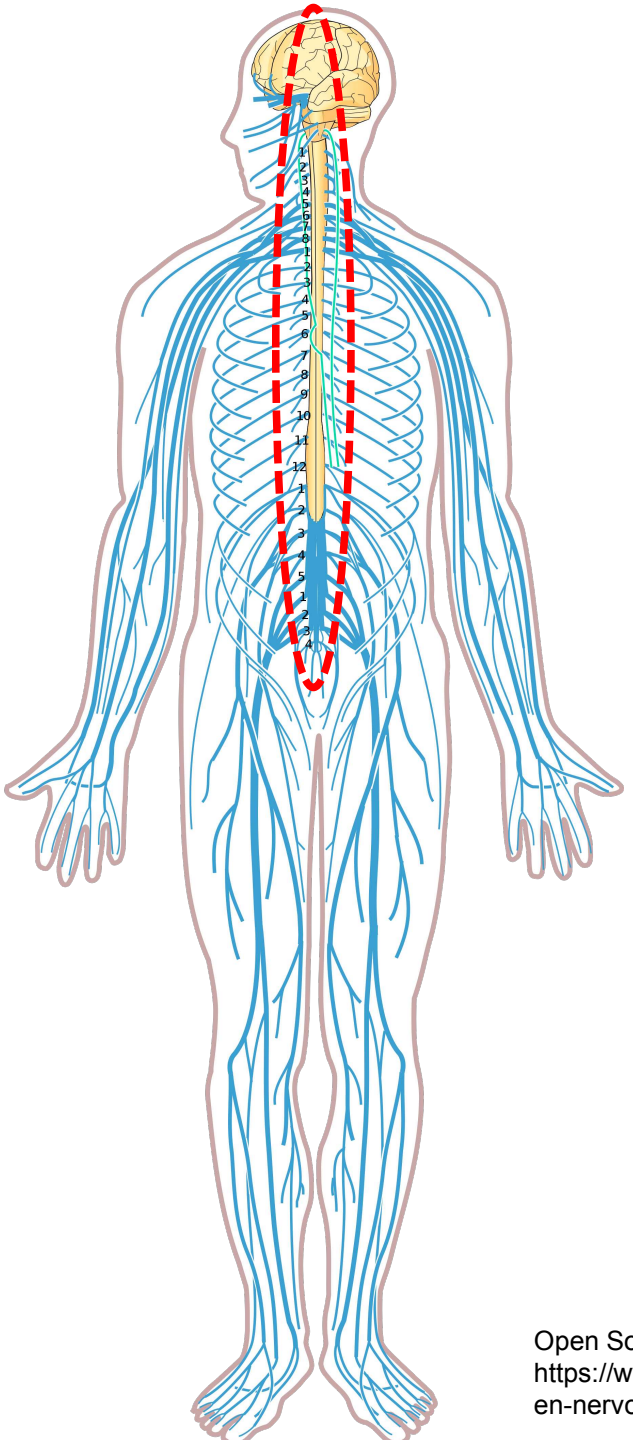
- Classified according to axonal Ø & speed of conduction

  A fibres – large myelinated ***(fast)*** fibres – *motor & sensory*
  B fibres – myelinated – pre-ganglionic autonomic fibres
  C fibres – non-myelinated ***(slow)–*** post-ganglionic autonomic & visceral & somatic *afferents for pain & temperature* – 'small fibres'



The peripheral reflex pathway

“radiculitis”



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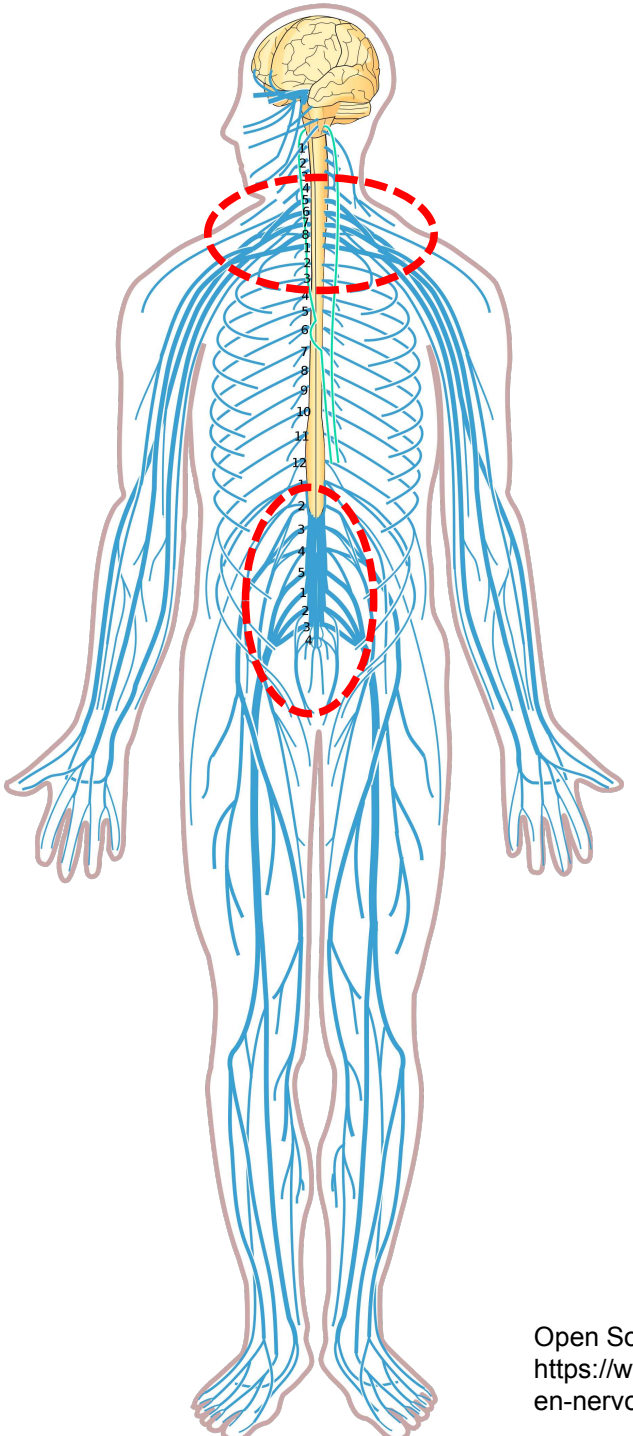
"plexitis"



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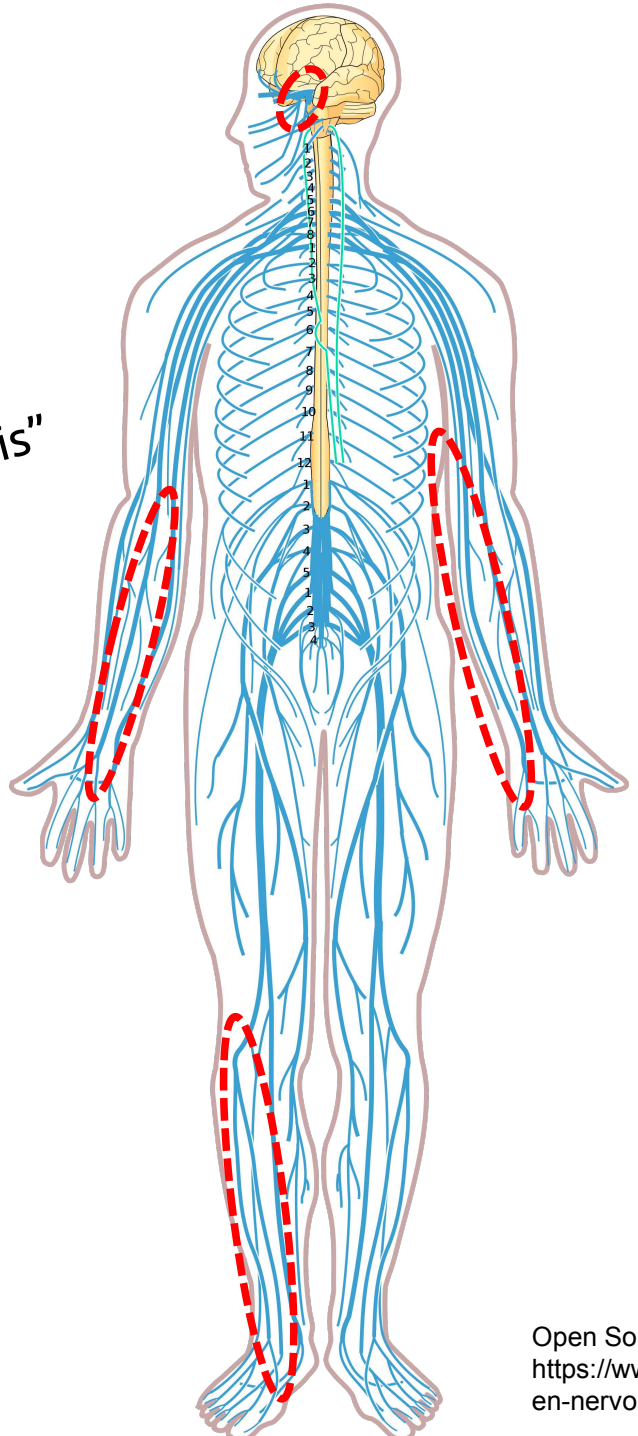
"(multiple) mononeuritis"



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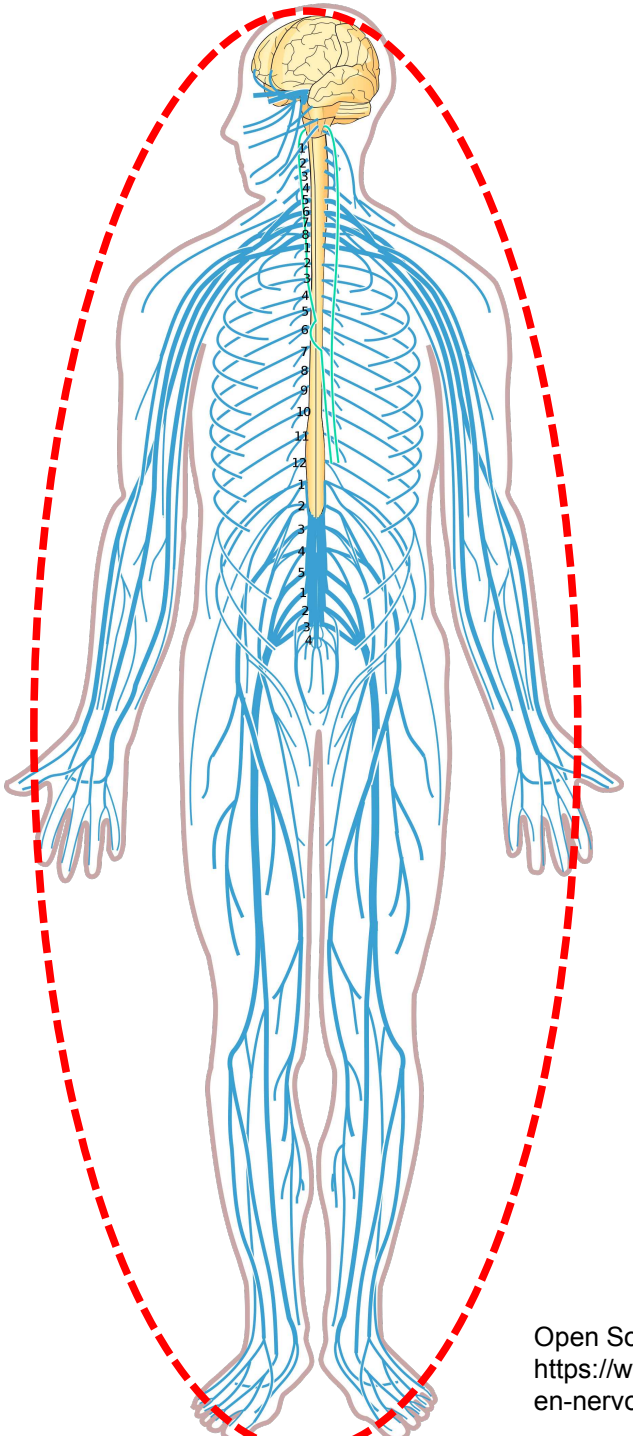
“polyneuropathy”



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Terminology

Polyneuropathy or peripheral neuropathy: generalised disorder of peripheral nerves usually in a length dependant manner (types: axonal or demyelinating)

Mononeuropathy: localised disorder as above, can occur isolated or multiple

Radiculopathy, plexopathy and combinations: involving rootlets, brachial or lumbosacral plexus or combinations

Nerve damage in general

- **Infective**
- **Metabolic (diabetes)**
- Endocrine
- Trauma
- Toxic
- Vasculitis
- Inflammatory/Immune mediated
- (Para)neoplastic
- Hereditary

Questions to ask

- Age at onset of symptoms
- Time Course & Duration of symptoms
- Provoking factors
- Concurrent systemic symptoms *e.g* HIV/diabetes
- Autonomic: Bladder function, visual acuity, postural hypotension symptoms
- Medication use and intoxications, profession, travel history, diet
- Family history (“difficult feet”)

Main causes of neuropathy Africa

Diabetes

HIV, leprosy and other infections

Inflammation/postinfectious

Alcohol

Drugs: eg TB/HIV treatment

B12 deficiency

Hereditary

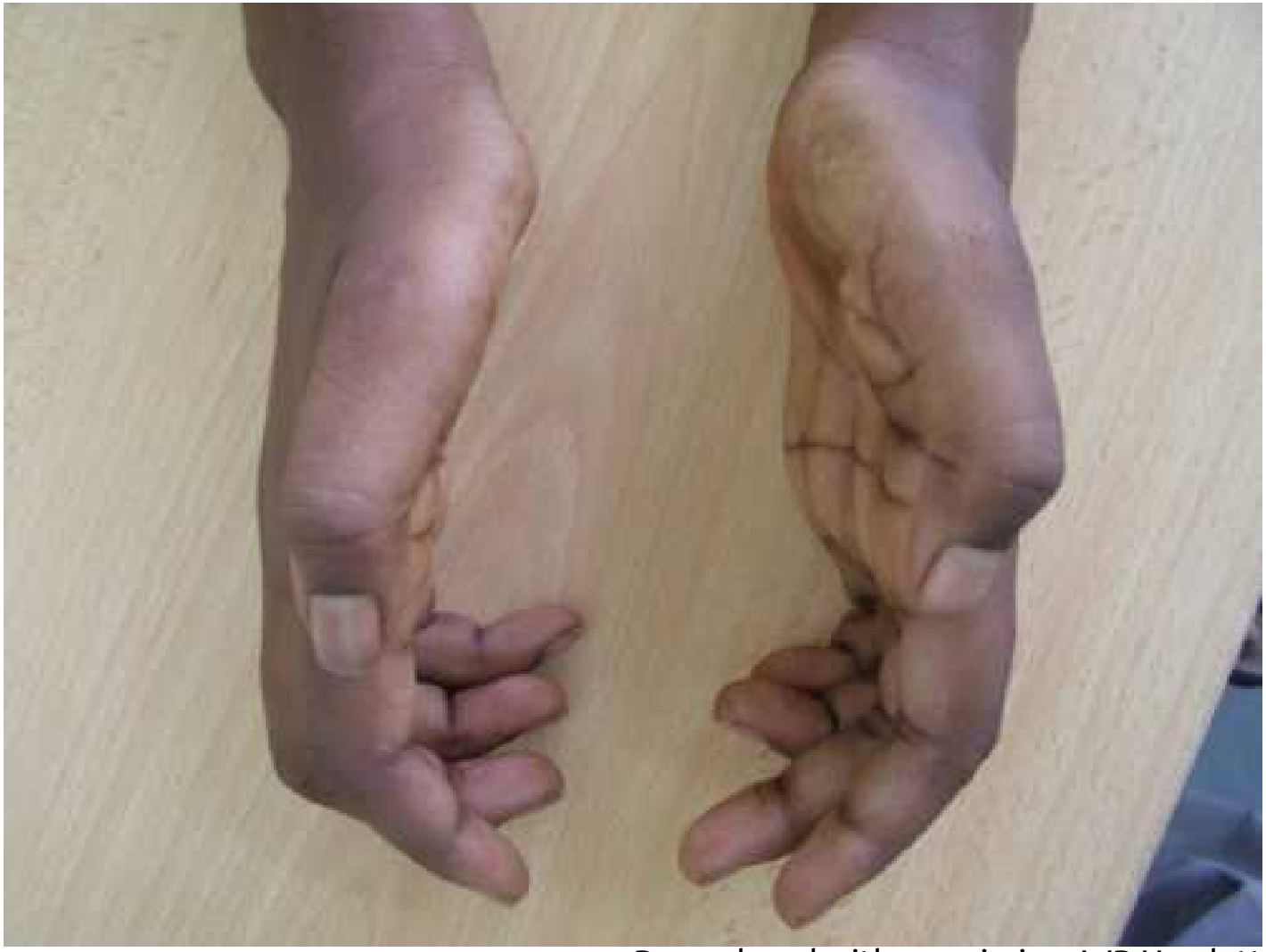
Toxins: lead, pesticides

Trauma/compression

Patterns 1:
Signs of peripheral nerve disease

- Weakness (gait: high stepping due to foot drop)
- Muscle wasting
- Loss of reflexes
- Hypesthesia/anesthesia/paresthesia
- Ataxia (due to loss of position sense)
- Skin, nail changes, hair loss, ulcers

Distal wasting



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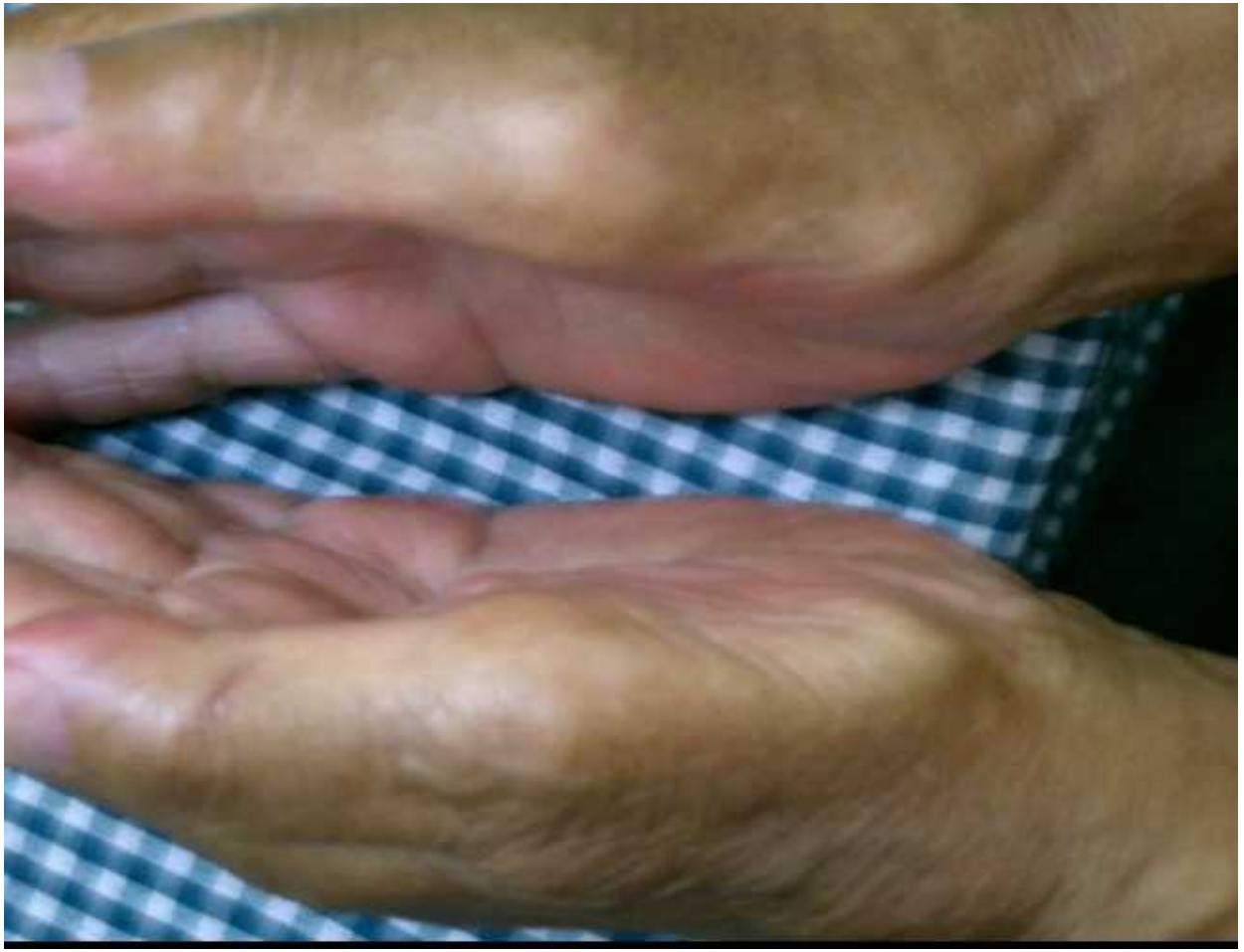
Bilateral CTS



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Ulnar neuropathy

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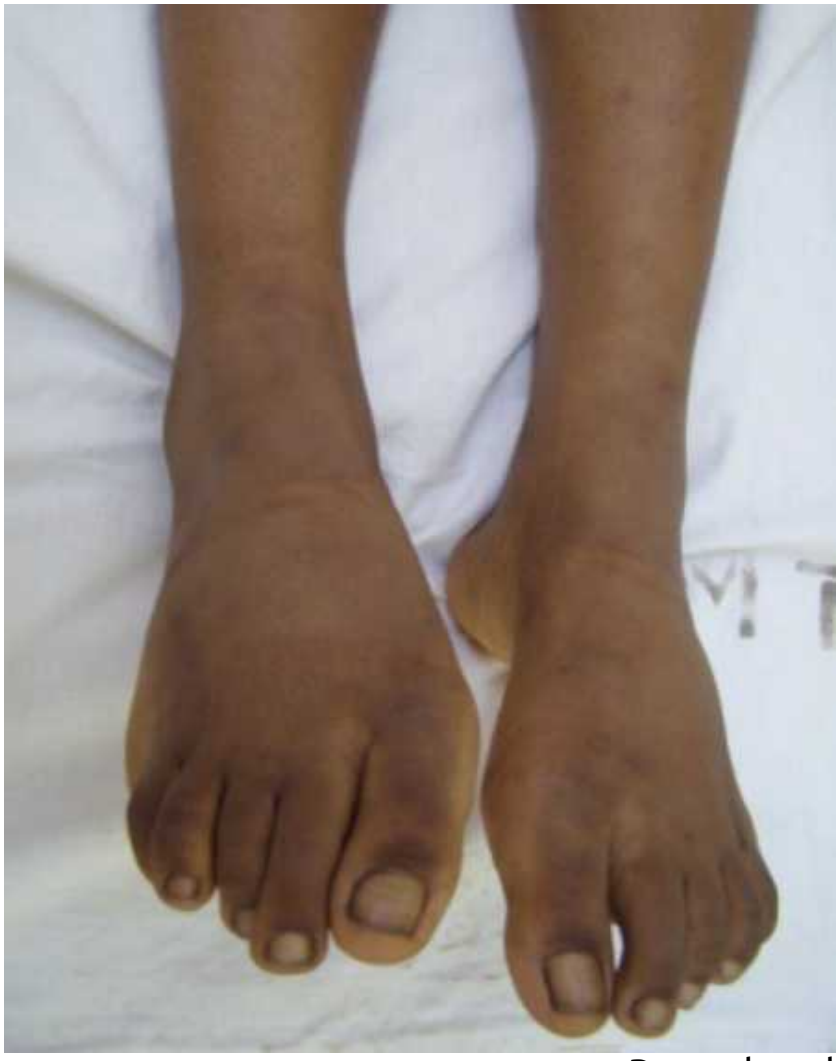
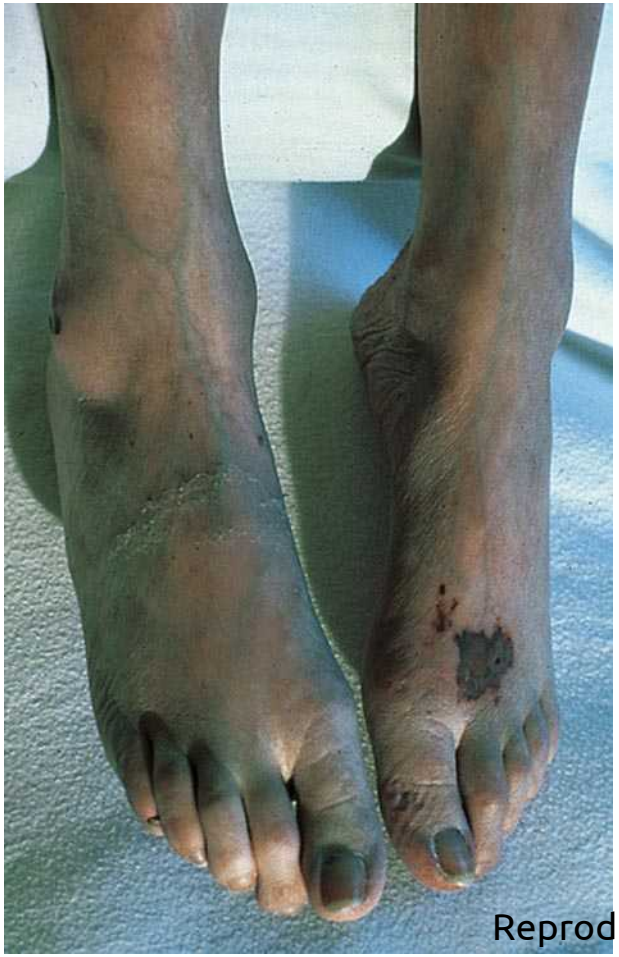
Bilateral Foot Drop



Vasculitis – cutaneous infarcts and bilateral foot drop



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Patterns 2:
Terminology according to location

Mononeuropathy – one peripheral nerve affected

Mononeuritis multiplex – more than one individual nerve affected

Plexopathy – brachial & lumbosacral

Generalised (peripheral) neuropathy

Patterns 2:
Axonal or Demyelinating?

Demyelinating more likely to have proximal weakness

More widespread reflex loss than other symptoms might predict may suggest **demyelination**

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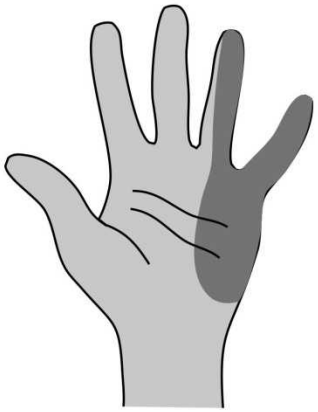
Patterns 3: distribution

ulnar nerve

sensory



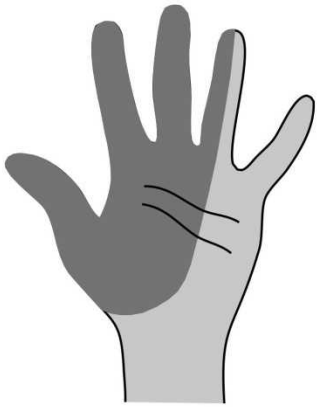
motor

small muscles of the hand except abductor pollicis brevis

ulnar half of flexor digitorum profundus
flexor carpi ulnaris

median nerve

sensory

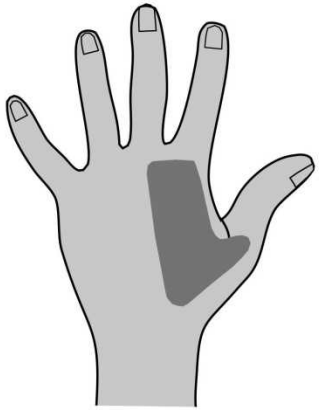


motor

abductor pollicis brevis

radial nerve

sensory



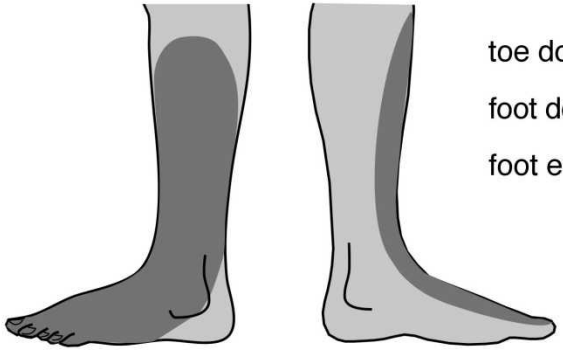
motor

finger dorsiflexors

thumb dorsiflexors
and abductors
wrist dorsiflexors
brachioradialis

common peroneal nerve

sensory



motor

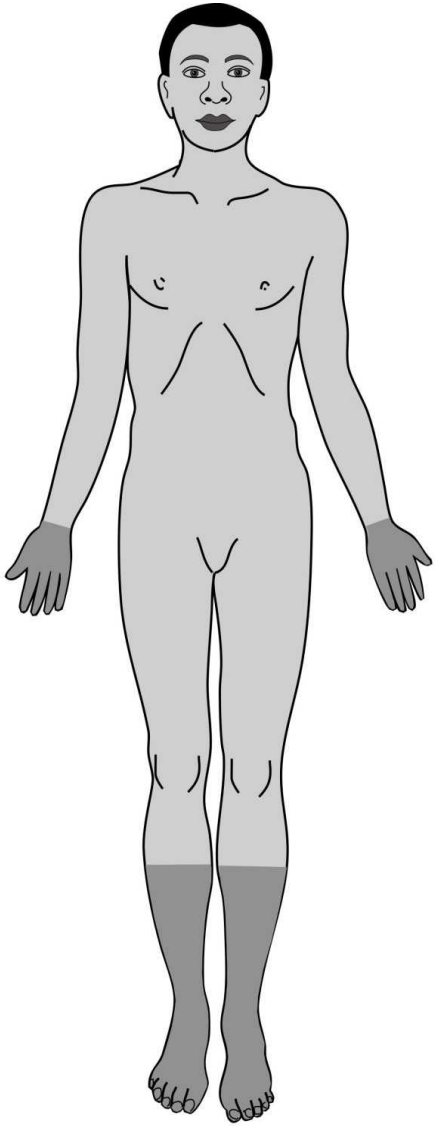
toe dorsiflexors

foot dorsiflexors

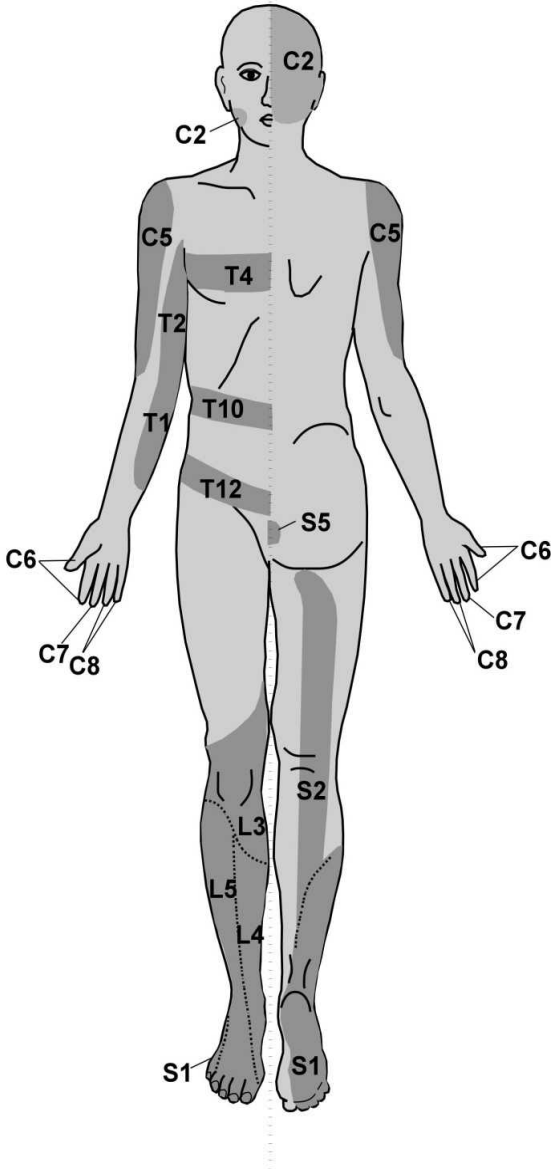
foot evertors

Sensory and motor loss in peripheral nerves

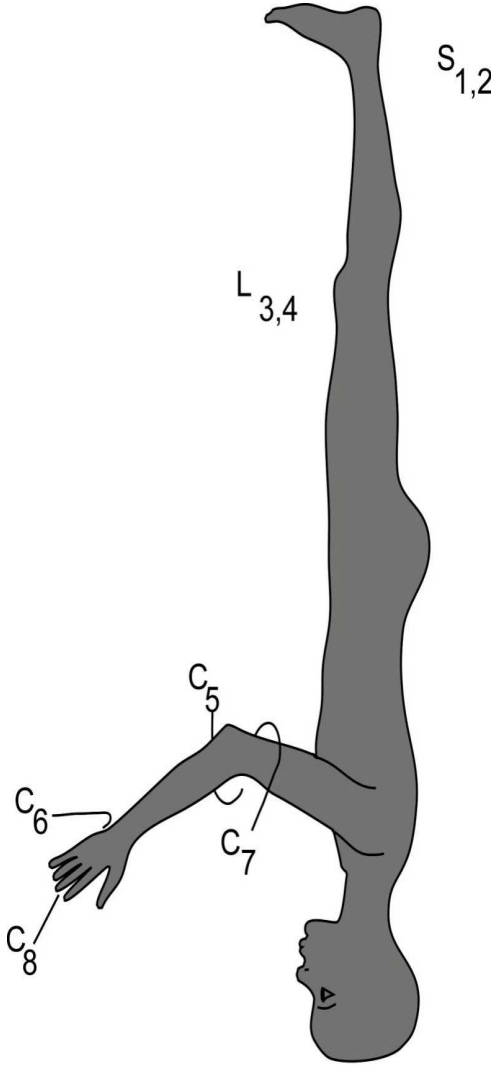
Patterns 4: distribution



Sensory loss in peripheral neuropathy, glove and stocking distribution



Skin territories of landmark nerve roots



Reflexes
Count from the ankle

Investigating neuropathies 1
General workup

- Full blood picture, ESR
- Blood glucose, HbA1c
- HIV test
- Liver function tests/ renal function
- Vitamin B12
- Thyroid function
- Vasculitis screen (*ANA, Rheuma Factor, paraproteins)*

Investigating neuropathies 2
Narrowing down

- **Nerve conduction studies/Electromyography**
- **MRI plexus** (root enhancement)
- **Further blood tests –** *infection screens, toxins, antibody tests, genetic PNP panel*
- **Urine –** evidence of (renal) vasculitis, Bence-Jones protein
- **Cerebrospinal fluid (CSF)** - raised protein, pleocytosis, cytology on cells/cytospin
- **Nerve biopsy** (sural), skin or muscle biopsy

Neurophysiology testing 1

- **Nerve conduction studies (NCS):** Stimulating peripheral nerves & recording impulses generated

  Can measure

  - sensory & motor nerve action potential amplitudes
  - conduction velocities

- **Electromyography (EMG):**

  Can show if muscles are (being) denervated

Neurophysiology testing 2

Studies can give information on extent of disease & symmetry

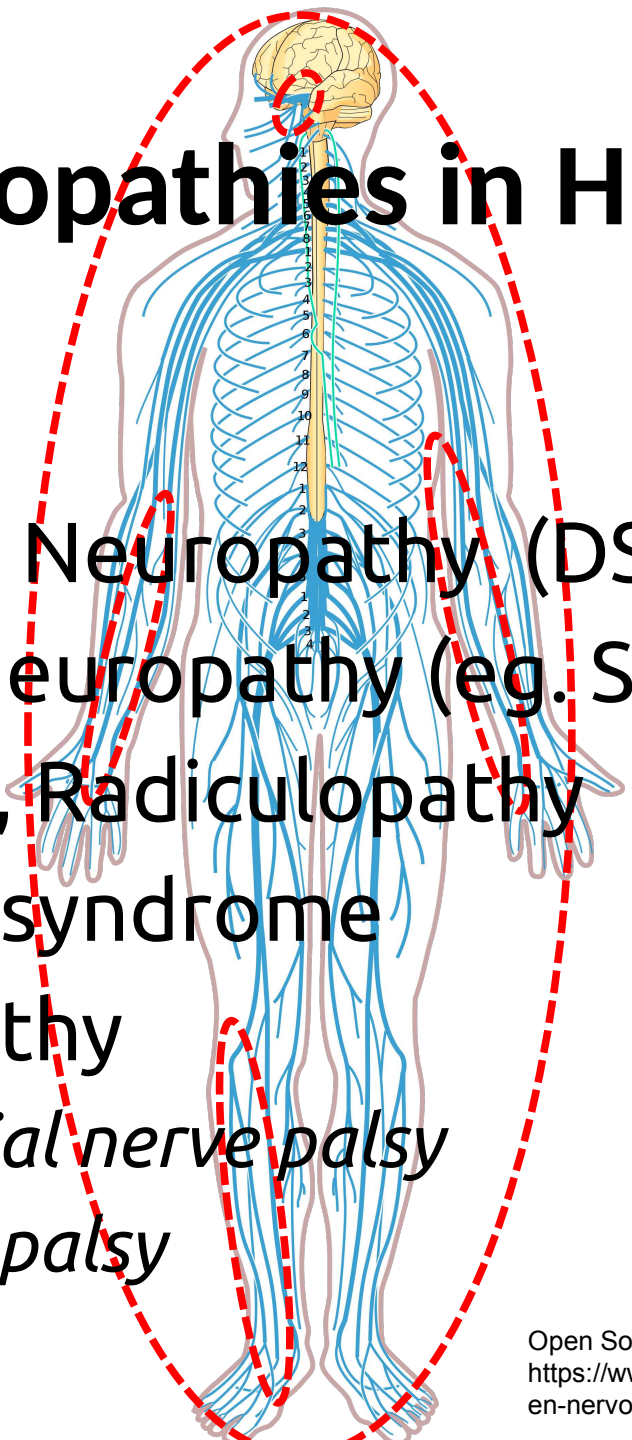
Measurements allow peripheral neuropathies to be separated into **axonal *or* demyelinating varieties**

If clinically large fibre sensory disturbance is present but *sensory nerve action potentials are normal* this suggests a central or root disorder

Main Conditions

- HIV neuropathies
- Leprosy
- Neuralgic Amyotrophy (Brachial Plexus Neuritis/Parsonage Turner syndrome)
- Guillain-Barré syndrome (Acute Inflammatory Demyelinating Polyneuropathy)
- Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)
- Other infectious or inflammatory oligo/polyradiculopathies

Main Neuropathies in HIV Disease



- Distal Sensory Neuropathy (DSN)
- ART induced neuropathy (eg. Stavudine)
- Herpes Zoster, Radiculopathy
- Guillain-Barré syndrome
- Mononeuropathy
  - *Idiopathic facial nerve palsy*
  - *Cranial Nerve palsy*
  - *Mononeuritis*

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Key Points

Neuropathy: very common in HIV disease

Most common is: **Distal Sensory Neuropathy** in **40%** of patients

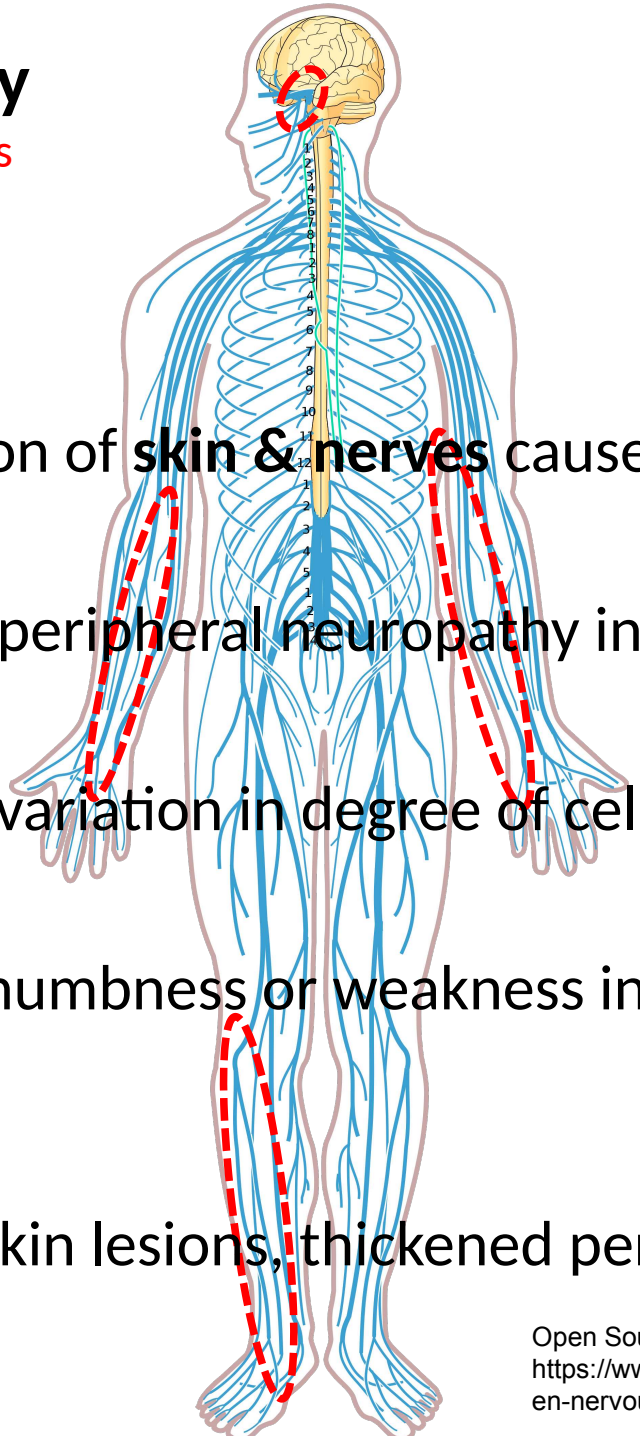
Mechanisms: HIV infection, ART toxicity & autoimmunity

Management: starting ART & reducing or stopping offending drug (might require second line ART)

Frequency of GBS & Bell's palsy: increased in HIV infection *so always test for HIV*

Leprosy

Key points



Leprosy: chronic infection of **skin & nerves** caused by M. leprae

Major cause: disabling peripheral neuropathy in Africa

Findings explained: by variation in degree of cell mediated immunity

Neurological features: numbness or weakness in individual **nerves & polyneuropathy**

diagnosis: anaesthetic skin lesions, thickened peripheral nerves & AFBs in skin snips

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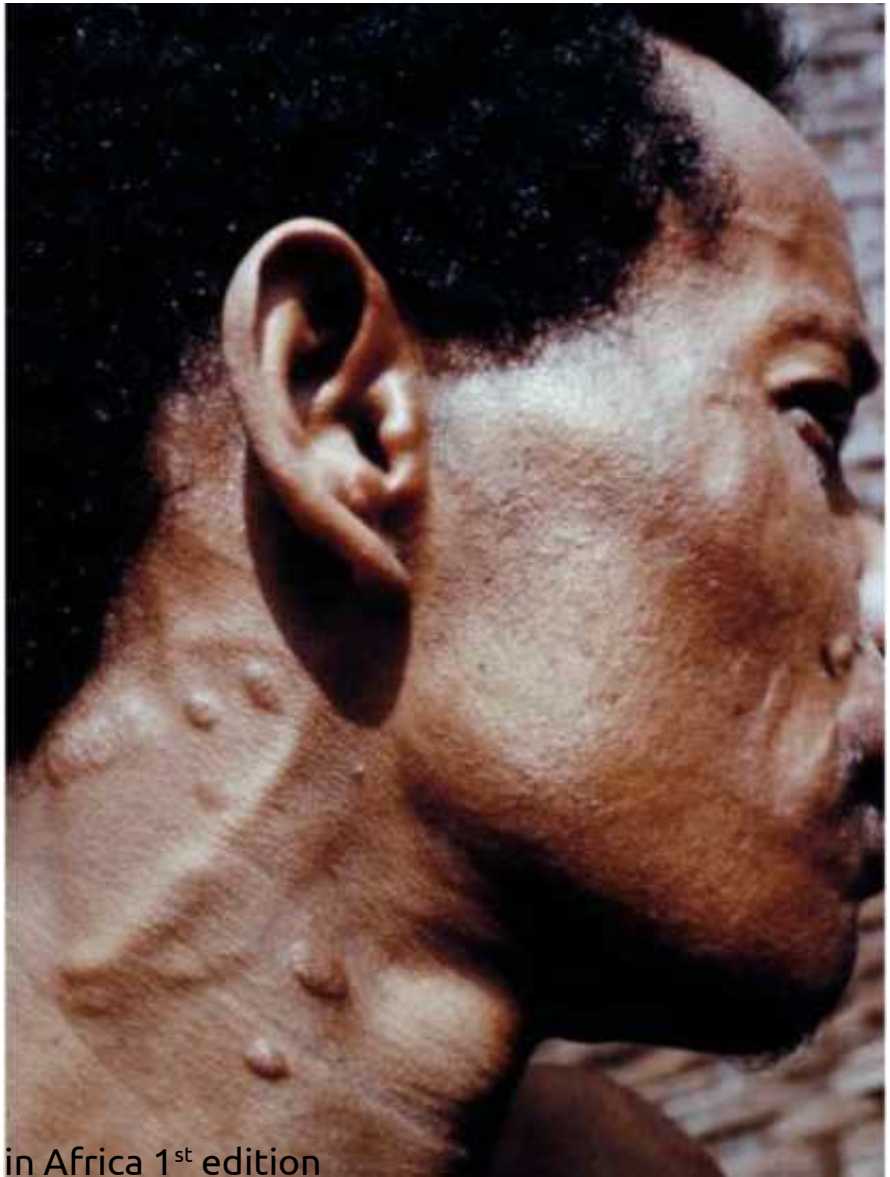
Thickened Nerves



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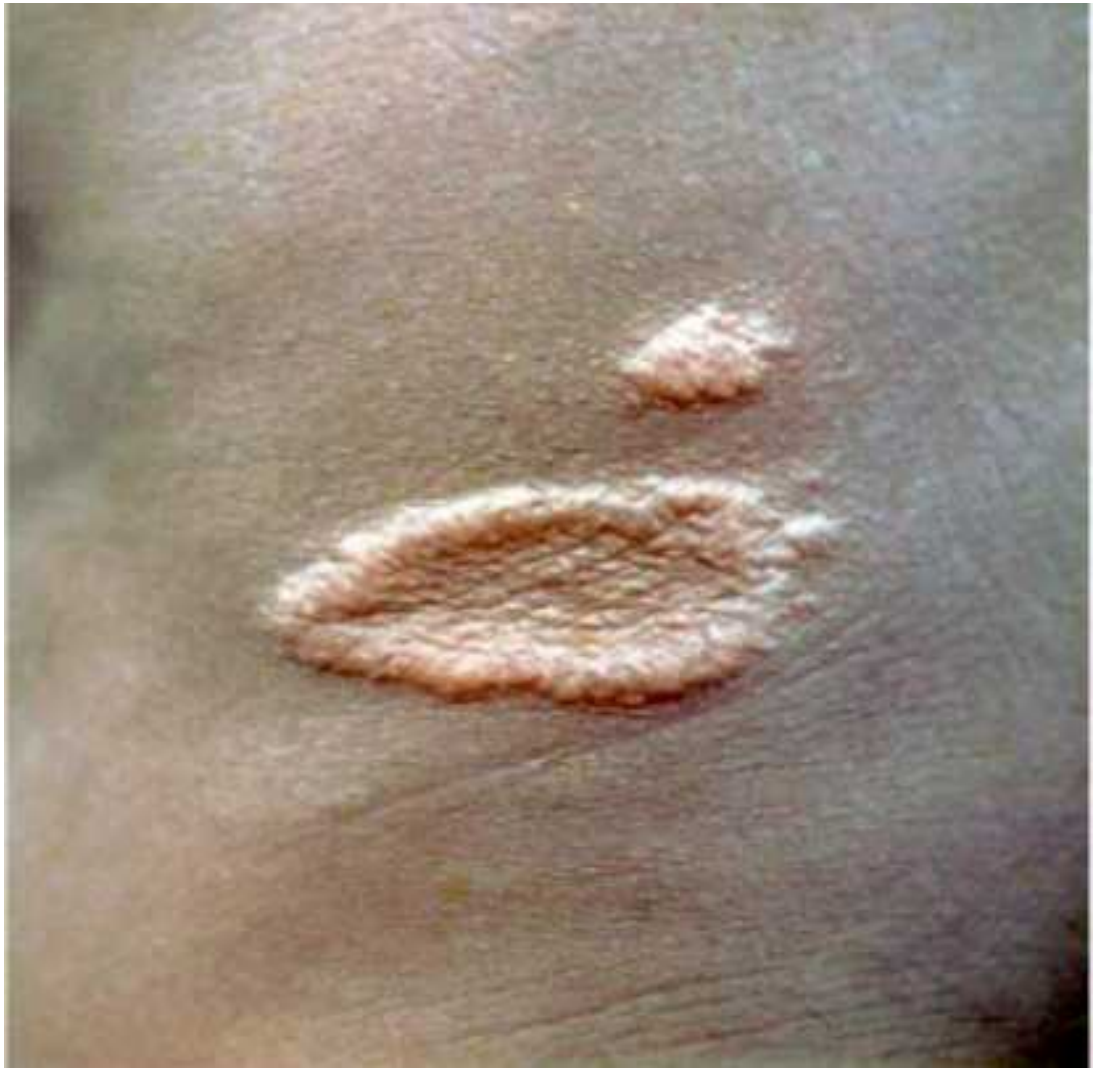
Tuberculoid



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Borderline

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Lepromatous (leonine facies)

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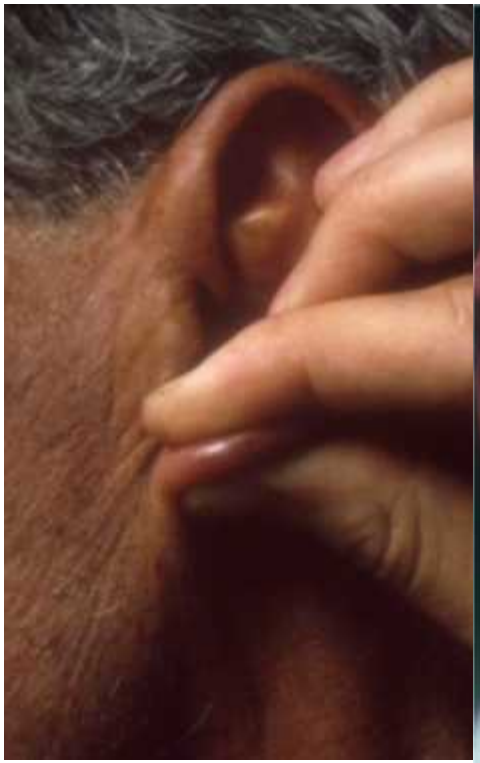
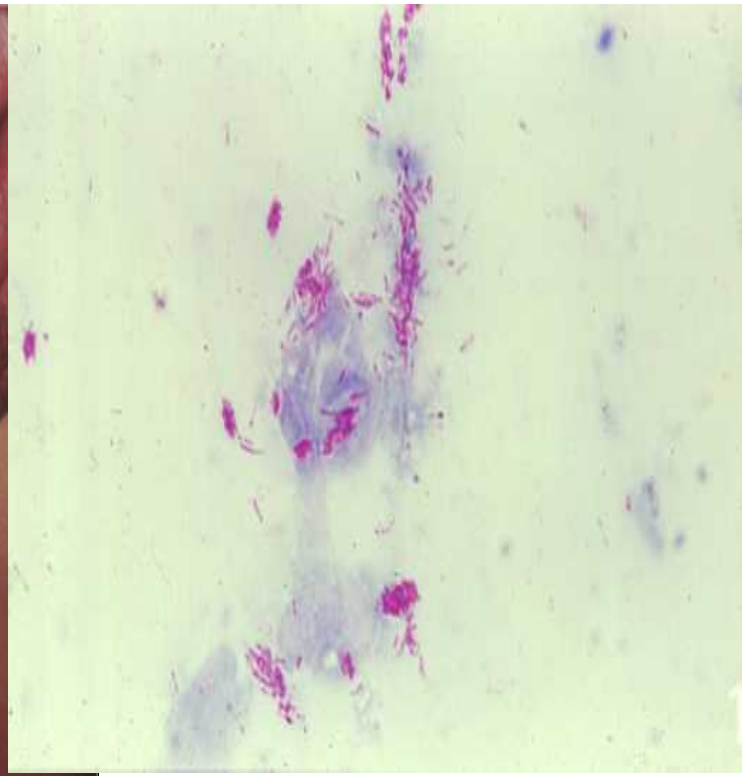
Skin Snip/Smear





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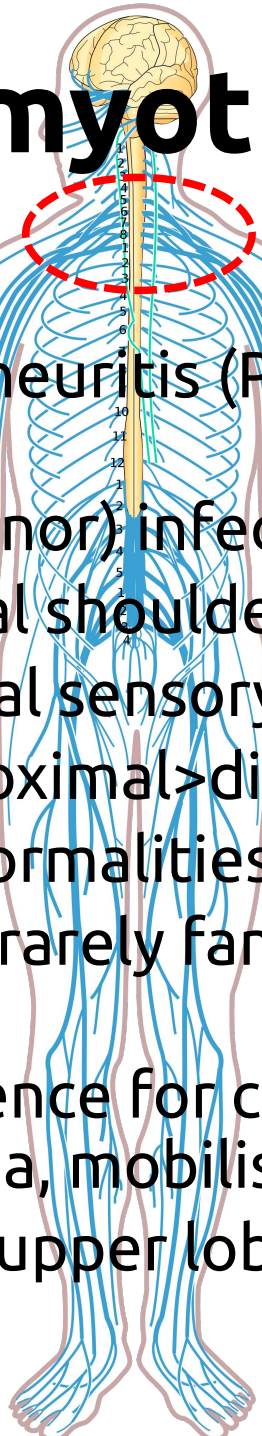
Neuralgic amyotrophy



- Acute brachial plexus neuritis (Parsonage Turner syndrome)
- Often preceded by (minor) infection
- Severe, often nocturnal shoulder/arm pain
- Not strictly dermatomal sensory deficits and weakness
- Scapula alata, early proximal>distal atrophy
- NCS, EMG: patchy abnormalities
- Occasionally bilateral, rarely familial (genetic predisposition)
- Treatment: weak evidence for course of oral steroids as in Bell's palsy, analgesia, mobilisation
- To rule out: ipsilateral upper lobe lung malignancy

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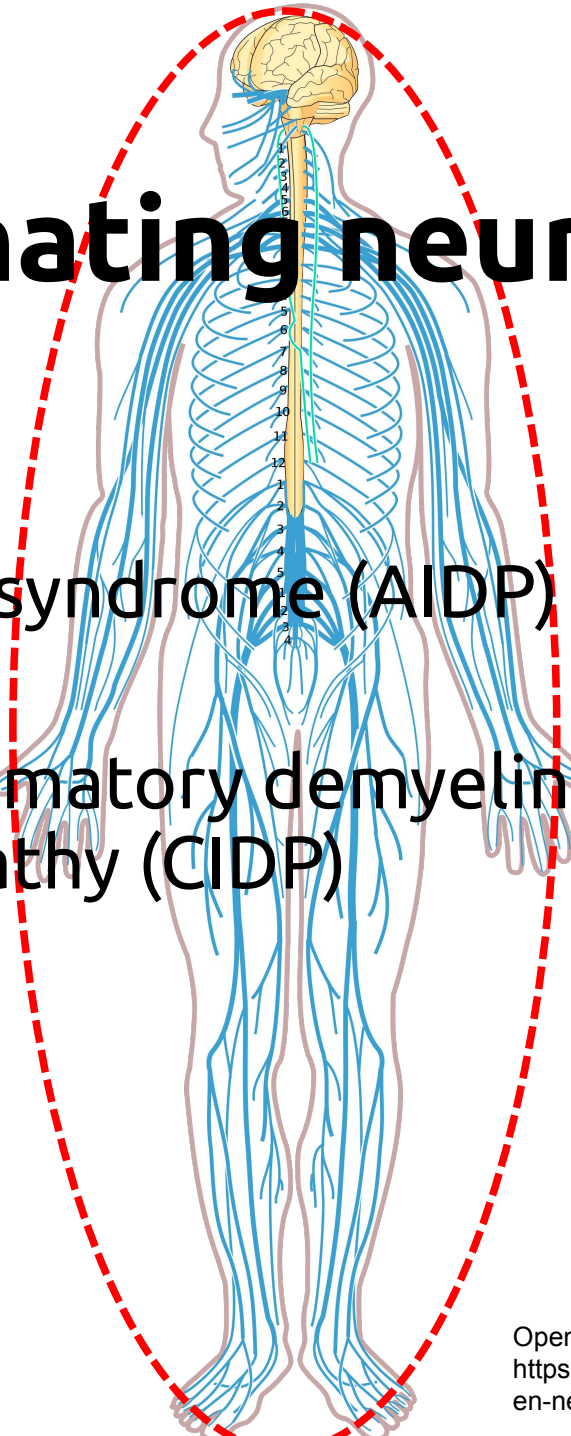
Demyelinating neuropathies



- Guillain-Barré syndrome (AIDP)
- Chronic inflammatory demyelinating polyradiculopathy (CIDP)

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Guillain-Barré Syndrome

- **Progressive weakness** of 2 or more limbs reaching a peak within **4 weeks**
- Reduced *or* absent reflexes
- Cranial nerves: **> 50% 7th Nerve palsies**
- Exclusion of other causes
- 2/3 of cases preceded by infections
- – *eg Campylobacter jejuni, CMV, EBV, Mycoplasma pneumoniae* – Infection has usually already resolved before neurological symptoms start

Guillain-Barré Syndrome investigations

- Usually an acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
- Investigations can be normal early in disease, optimal window for LP: 10-14 days post onset
- Usually raised CSF protein and normal cell count ('albumino-cytologic dissociation'), though up to 50 lymphocytes allowed
- **Nerve Conduction Studies:** can be characteristic with long DML, low v, partial blocks, absent Fs, but normal early in disease

Guillain-Barré Syndrome treatment

- Supportive measures are the most important
- **Intravenous immunoglobulin** and Plasmapheresis (plasma exchange) both hasten recovery and reduce long term disability. SVPE a low resource option!
- **Cardiac monitoring** for arrhythmias, monitoring **respiratory function** with FVC

  Measures to reduce the risk of thromboembolism & constipation
- **Physiotherapy**

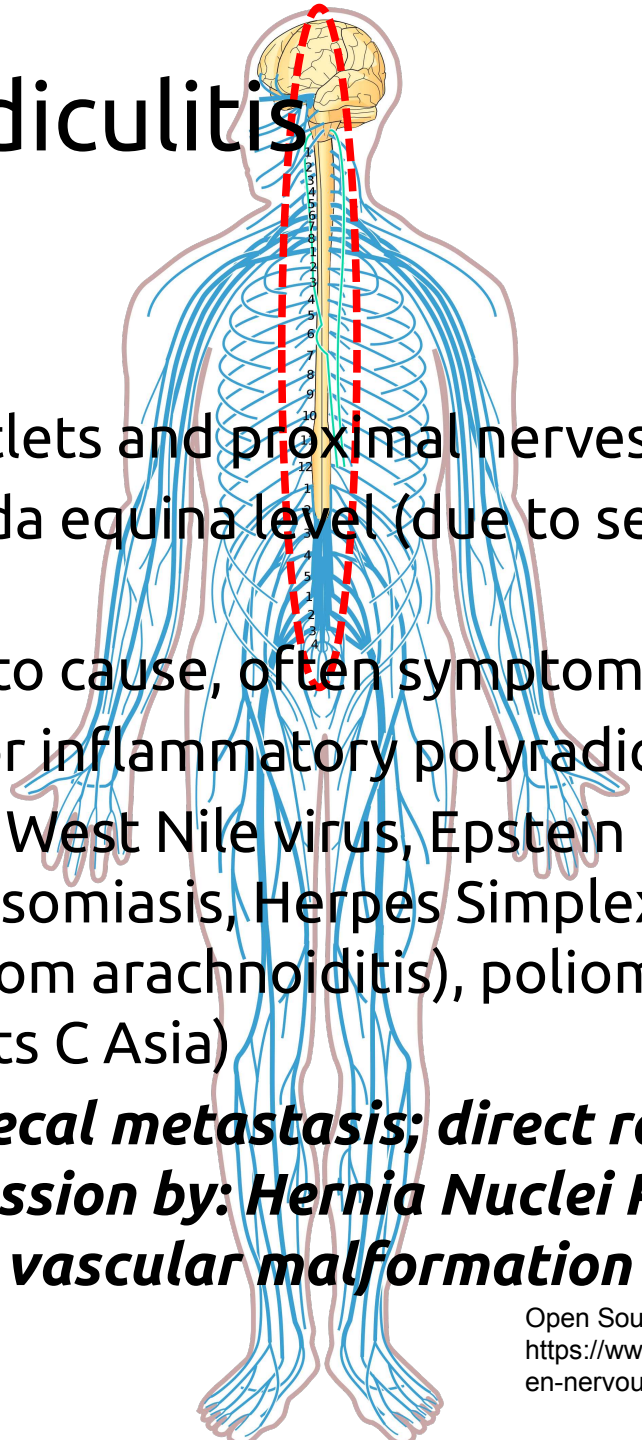
Guillain-Barré Syndrome
Differential Diagnosis

- Disorders of neuromuscular junction – myasthenia gravis, botulism
- Disorders or muscle - eg inflammatory myopathy (periodic paralyses)
- Acute myelopathy
- *Acute poliomyelitis*
- Other axonal neuropathies

Chronic Inflammatory Demyelinating Polyneuropathy

- Chronic ‘counterpart’ of GBS
- Association: HIV, hepatitis, puerperium
- Similar CSF and NCS/EMG profile as in GBS. MRI LS plexus: root enhancement
- Presents with distal sensory loss or tingling, weakness and loss of deep tendon reflexes
- Treatment: ivIg, plasmapheresis or dexamethasone pulse course (40mg/day, 4 days/month, during 6 months)

Poly-/oligoradiculitis



- Localisation: CSF, rootlets and proximal nerves
- Predisposition to cauda equina level (due to settling protein)
- Treatment according to cause, often symptom relief only
- Causes of infectious or inflammatory polyradiculitis:
- HIV, neuroborreliosis, West Nile virus, Epstein Barr virus, Neurosyphilis, Schistosomiasis, Herpes Simplex Virus 2, Tuberculosis (often from arachnoiditis), poliomyelitis WT1 (outbreak pockets C Asia)
- ***-Differential: intrathecal metastasis; direct radicular or cauda equina compression by: Hernia Nuclei Pulposi, tumour, Spinal-Dural vascular malformation***

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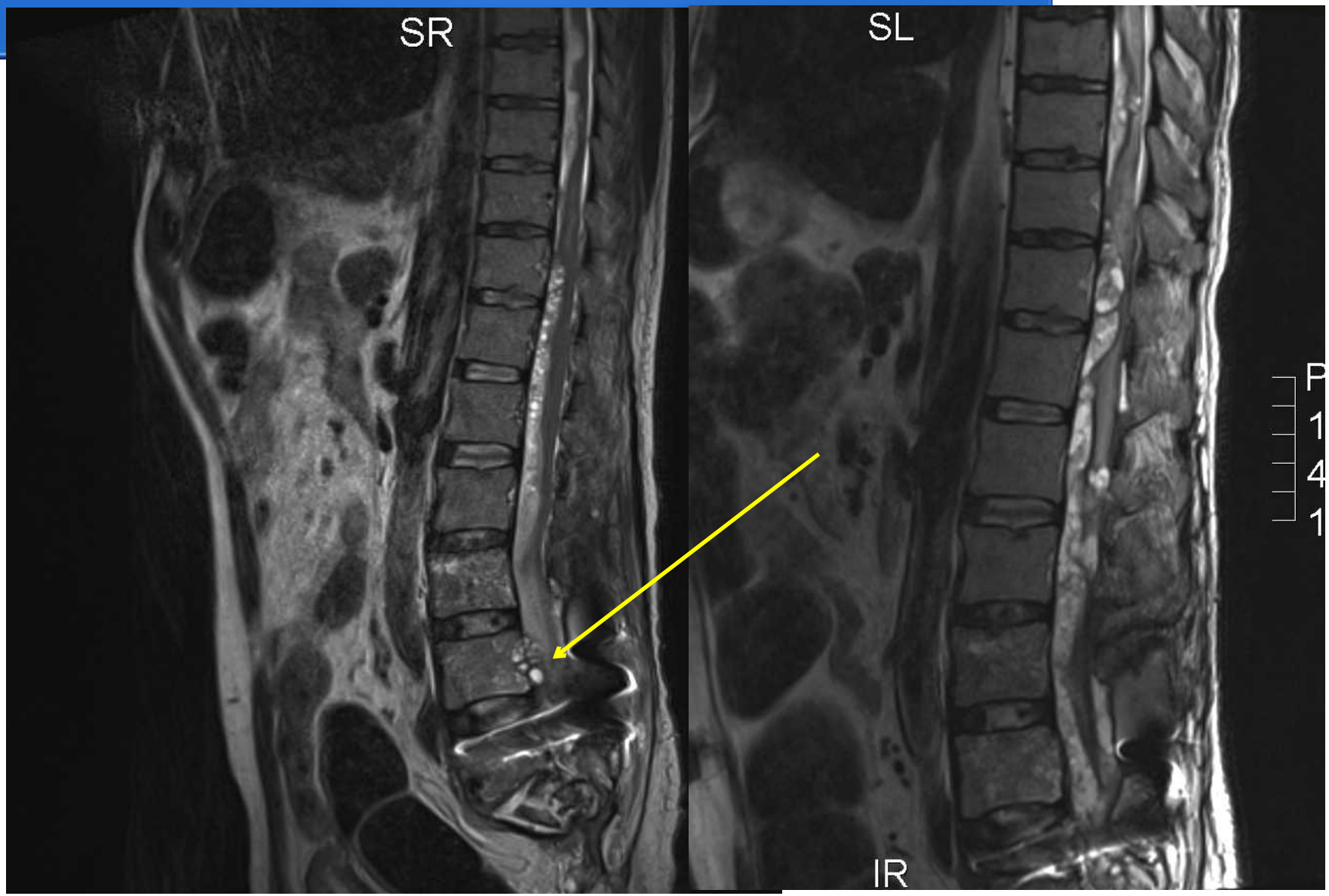
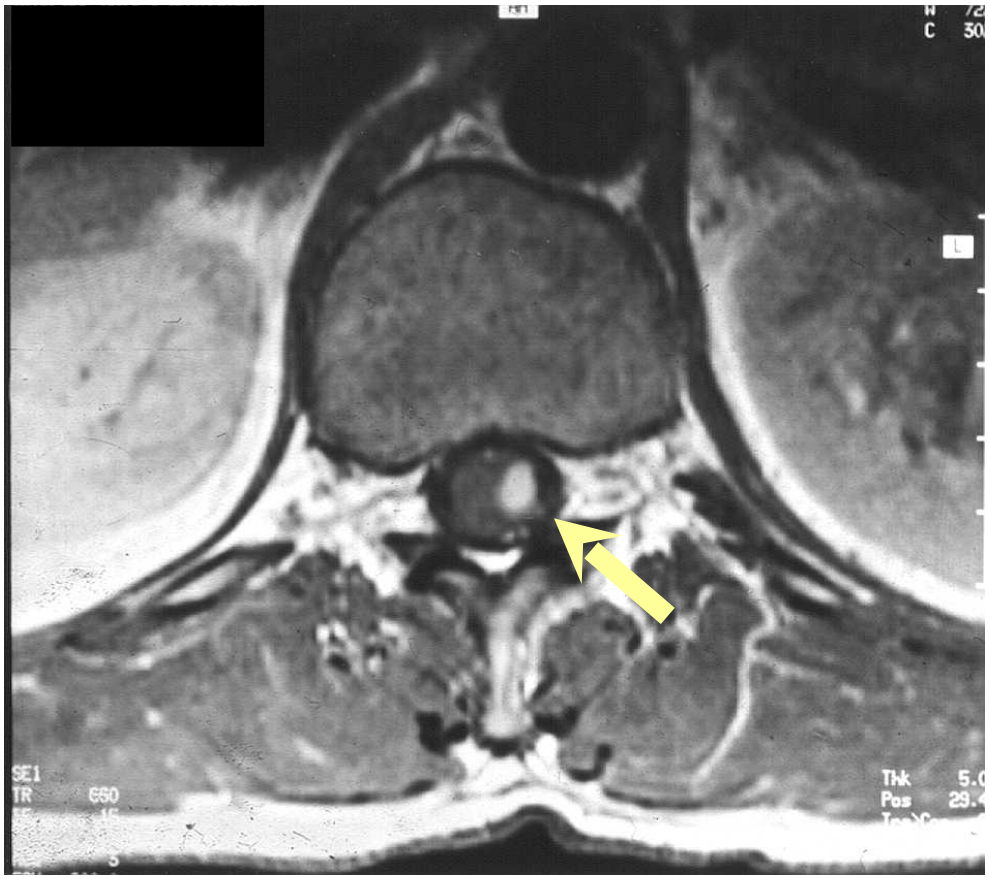
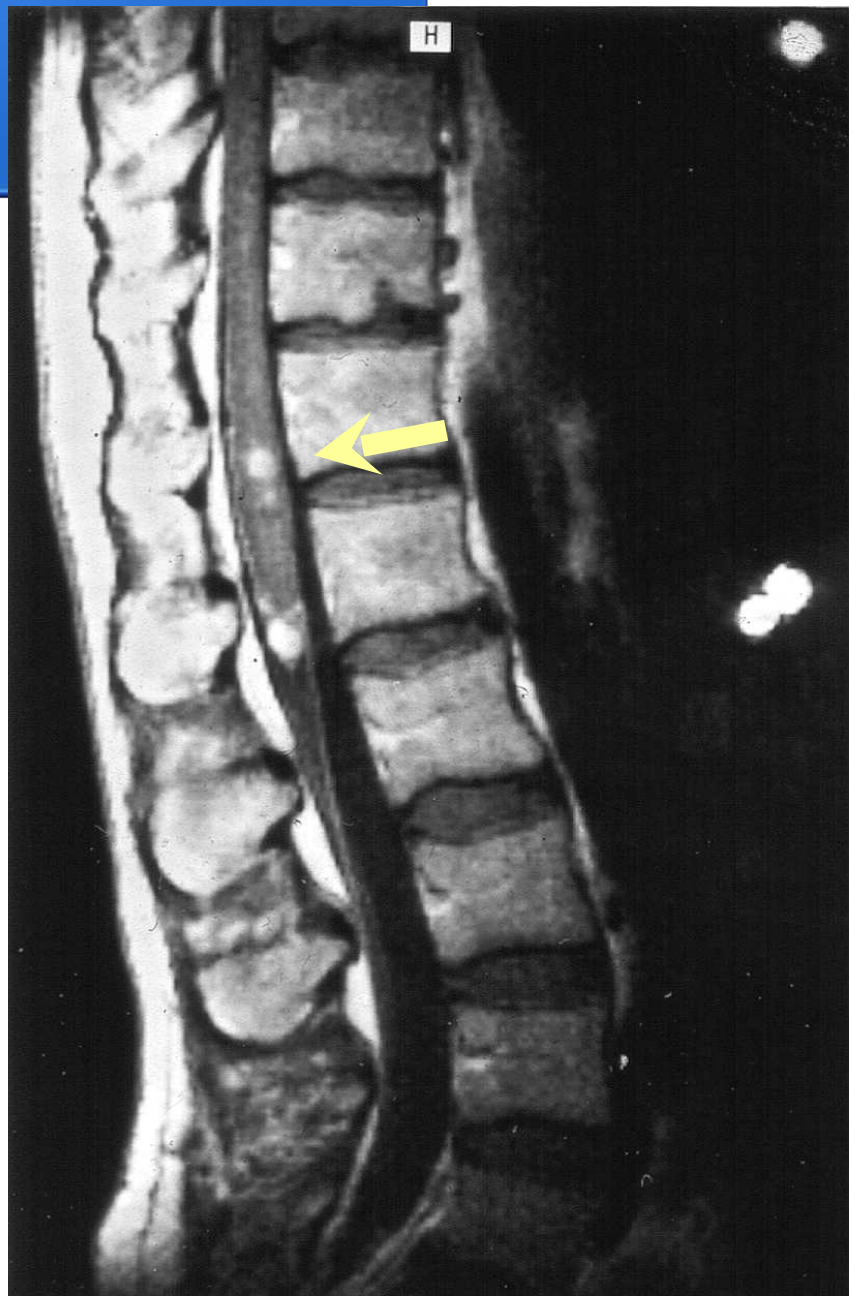
Echinococcosis



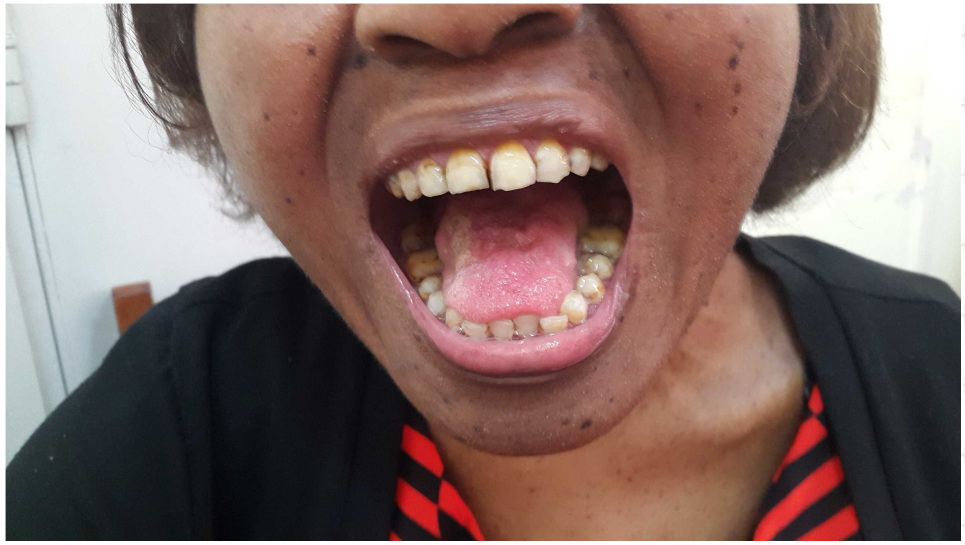
Schistosomiasis



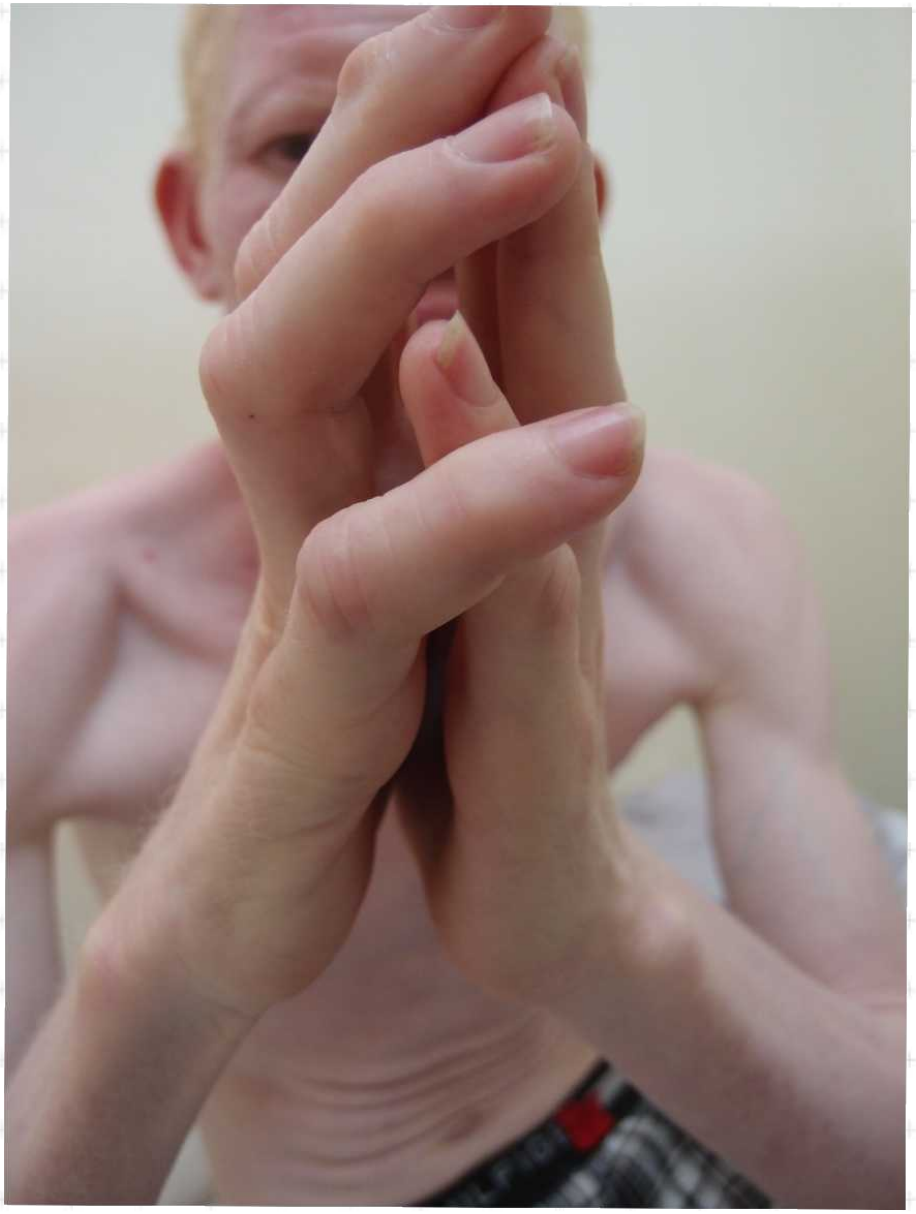


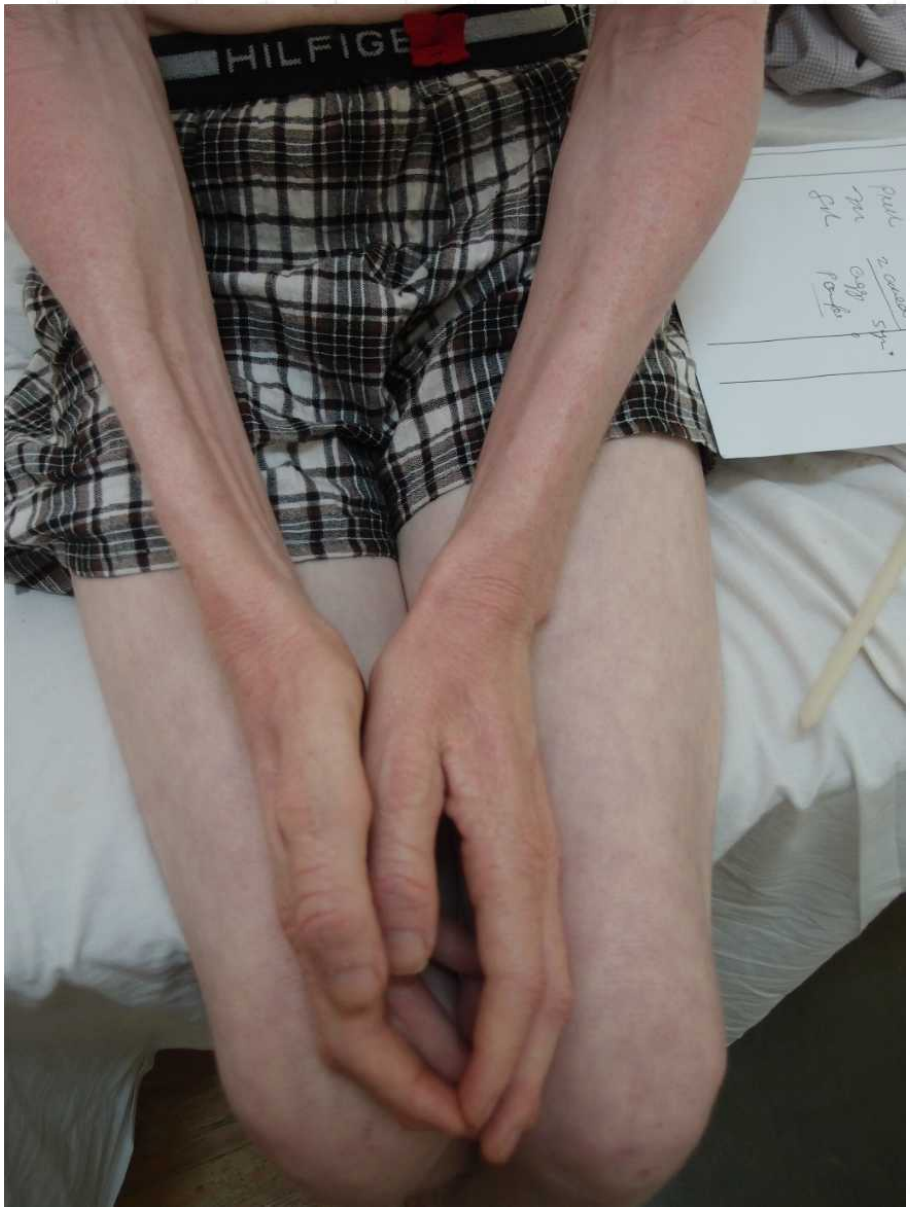
General Differentials

- *Diabetic neuropathies*: more prone to cranial neuropathies and entrapment neuropathies; furthermore truncal radiculoneuropathy and diabetic amyotrophy (lumbosacral radiculoplexoneuropathy)
- *Intoxications* (Pb), drug induced (ARTs, chemo Rx)
- Myelitis (inflammatory or demyelinating)
- Myopathy and myositis (infectious/inflammatory/auto immune/paraneoplastic)
- *Myasthenia gravis; Motor neurone disease*
- *Botulism* (generalised neuromuscular junction impairment due to clostridium botulinum infection and release of botulin toxin type B)



Today's Clinic
Lady, 1974
Clinical course 6 months

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