Peripheral nerves have low metabolic demands and extensive collateral circulation:
- invariable to occlusion of large peripheral arteries,
- susceptible to small blood vessel diseases (focal circulation interruption in vasa nervorum - individual nerve fascicles) - many types of systemic vasculitis affect peripheral nerves!

**Etiology**
1) polyarteritis nodosa (nerves are most frequently damaged organs!)
2) RA, SLE, Sjogren syndrome, systemic sclerosis
3) vasculitides associated with infections (hepatitis B, Lyme disease, HIV).
4) Churg-Strauss syndrome
5) Wegener granulomatosis
6) VASCUITIS RESTRICTED TO PNS - special diagnostic challenge, because footprints of systemic inflammatory disease (e.g. ESR1) are often absent.

**Clinical Features**
- reflect patchiness of underlying disease; characteristically - MONONEUROPATHY MULTIPLEX
- asymmetry & length-independence.
- evolves in stepwise fashion (e.g. wristdrop → contralateral footdrop → patchy areas of subjective numbness or sensory loss elsewhere on extremities).
- cranial nerve involvement, respiratory complications, and sphincter dysfunction are uncommon.

**Diagnosis**
- In absence of diabetes mellitus, vasculitis becomes prime diagnostic consideration!
- screening to detect systemic vasculitis.
- vasculitis is histologic diagnosis - if no other organ involvement is identified → combined nerve and muscle biopsy (axon loss).
- CSF typically is normal (except with SLE).

**Treatment**
- treatment of underlying vasculitis.
- VASCUITIS RESTRICTED TO PNS - corticosteroids, but most patients require CYTOTOXIC therapy (as in polyarteritis).

**CRITICAL ILLNESS POLYNEUROPATHY**
- occurs in critically ill patients (sepsis, multiple organ failure, etc).
- pathophysiology unknown (diabetic deficiency is not considered candidate).
- severe MONONEUROPATHY MULTIPLEX (axon loss).
- patients experience difficulty being weaned from ventilators.
- complete recovery may occur if underlying cause of multiple organ failure is successfully treated.

**TOXIC NEUROPATHIES**
- persons with pre-existing nerve disease are unusually susceptible to neurotoxins!
- most, although not all, neurotoxins produce distal axonal degeneration – distal sensory loss, loss of ankle tendon reflexes, distal weakness.
- sensory component suffers most, toxins that produce predominantly motor neuropathy:
  1) lead
  2) α-hexane (glue sniffer's neuropathy)
  3) tri-ortho-cresyl phosphate ("ginger jake") - adulterant in illegal liquor (moonshine)
  4) dapsone (leprosy treatment)
- with continued exposure, symptoms may progress proximally. COASTING - continuing progression even after offending agent is withdrawn.
- key to treatment - prompt recognition and withdrawal.
- specific therapy for metal poisoning - DI-PENICILLAMINE.

**BIBLIOGRAPHY** for ch. “Peripheral Neuropathies” — follow this LINK >>