Disease	Feature	Cause	Sign & Symptoms	Histology & Microscopically	Other
0 3	Demyelinating neuropathies Polyneuropathies Acute Inflammatory STUDIES CONDUCTION STUDIES CHYERAL NERVES Mycoplasma pneumoniae CAMPHIDRACTER CA	Postinfectious ,immunemediated disease. Cellular and humoral immune mechanisms Autoimmune response: Infection with → Campylobacter jejuni ,CMV , Epstein-Barr virus & , Mycoplasma pneumoniae Acute ,influenza-like illness from which the affected individual has recovered by the time the neuropathy becomes symptomatic	Presents 2 - 4 weeks following a relatively benign respiratory or gastrointestinal illnes finger dysesthesias and proximal muscle weakness of the lower extremities. The weakness may progress over hours to days to involve the arms, truncal muscles, cranial nerves, and muscles of respiration Weakness beginning in the distal limb→ rapidly advances to proximal muscle function"→ ascending paralysis["motor] CSF protein levels are elevated ↑↑ Altered permeability of the microcirculation within the spinal roots.	1. Segmental demyelination 2. inflammation of peripheral nerves , (perivenular and endoneurial mononuclear cell infiltrates rich in macrophages & lymphocytes) GUILAIN-BARKÉ SYNDROME SUM ENDONEURIS SONTHORDING CONTROLLED PROJECTION OF THE CONTROLL	Acute Inflammatory Rapidly progressive most common life- threatening diseases of PNS, may lead to death from failure of respiratory muscles in days. Patients who survive the initial acute phase of the disease usually recover with time Treatments Plasmapheresis intravenous immunoglobulin supportive care
Chronic Inflammatory Demyelinating Poly (radiculo) neuropathy (CIDP)	Demyelinating neuropathies Polyneuropathies Chronic inflammatory	Acquired Immune mediated	Symmetrical mixed sensorimotor polyneuropathy that persists for 2 months or more. Abnormalities include weakness ,difficulty in walking ,numbness, and pain or tingling sensations.	Segmental demyelination & remyelination. Repeated activation and proliferation of Schwann cells result in concentric arrangement of multiple Schwann cells around individual axons to produce multilayered structures → onion bulbs.	Most common Chronic acquired Inflammatory Follows a chronic relapsing- remitting, or progressive course . Treatments Plasmapheresis IVlg treatment. physical and occupational therapy with orthotic devices
Diabetic Peripheral Neuropathy	Demyelinating neuropathies Axonal neuropathies Polyneuropathies Distal symmetric sensorimotor polyneuropathy	Nutritional & metabolic Hyperglycemia accumulation of advanced glycosylation end products(AGEs), increased levels of reactive oxygen species, microvascular injuries & ,changes in axonal metabolism avonal & myelin injuries	Sensory axons are more severely affected than motor axons paresthesias & numbness. Forms: Autonomic neuropathy is involve the cardiovascular, gastrointestinal, and genitourinary systems Sensory neuropathy: numbness, tingling in stocking-and-glove distribution Sensorimotor neuropathy: impaired fine hand coordination, e.g difficulty with tasks such as opening jars or turning keys		Most common Treatments: glycemic control.

Disease	Feature	Sign & Symptoms	Histology & Microscopically	Other
Schwannomas	 Benign Encapsulated Occur in soft tissues ,internal organs, or spinal nerve roots. Well circumscribed nerve sheath tumor arising from differentiated Schwann cells. 	Most commonly affected cranial nerve is the vestibular portion of the eighth nerve, symptoms related to nerve root compression, which includes hearing loss here.	 Grossly: 1. Circumscribed masses 2. Solitary 3. Completely encapsulated Histology: Spindle cell proliferation, arranged in hypo/hypercellular pattern. 	 Most are sporadic More common in 30 – 60 years of age May occur spontaneously or associated with familial Neurofibromatosis type 2 (NF2) Pain and neurological symptoms are uncommon unless the tumor is large Treatment: Surgical excision Local recurrence is uncommon Most cases have an indolent course → good prognosis
Neurofibromas	Not Encapsulated Presence of a neuronal component comprising transformed Schwann cells and a nonneoplastic fibrous component that includes fibroblasts	 Localized neurofibromas are superficial and evenly disturbed over the body surface Diffuse neurofibromas are usually in the head and neck region Presented as Painless, slowly growing, solitary, skin colored, soft mass. 	Proliferation of all elements of peripheral nerves including schwann cells with wire-like collagen fibrils and fibroblasts	 Malignant Peripheral Nerve Sheath Tumors can arise from them (50%NF1) Treatment: Superficial neurofibromas → marginal excision deep-seated neurofibromas → treated conservatively