PERIPHERAL NERVE INJURIES (PART 1)

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OBJECTIVES

- Classification and pathology


**ANATOMY**

- Anatomical organization of the peripheral nervous system:
  - 1) Cranial nerves
  - 2) Spinal nerves

  - which compose of:
    - Afferent and efferent connection
    - Sensory cells in the dorsal root ganglia
    - Motor cells in the anterior horn of the spinal cord
Connects the brain and the spinal cord with sensory receptors, muscles and glands.
NERVE FIBER COMPONENTS

- a layer of dense connective tissue, covers and holds together the outer surface of nerves, called the **epineurium**, which it have blood supply mainly.

- Axons, which are nerve fibers, organize into bundles known as fascicles with each fascicle surrounded by the **perineurium**.
Inside these fascicles, each nerve fiber enclosed by the endoneurium.

So, in compression neuropathy the most common causes is connective tissue syndrome.
Peripheral neuropathy: Damage to the peripheral nerves.

Primary injury: Results from same trauma that injures a bone or joint.

Secondary injury: Results from involvement of nerve by infection, scar, callous or vascular complications which may be hematoma, AV fistula, Ischemia or aneurysm.
The nerve is injured by:

1) Ischemia
2) Compression
3) Traction
4) Laceration
5) burning
TYPE OF INJURY:

- Transient ischemia
- Neurapraxia
- Axonotmesis
- Neurotmesis
These changes are due to transient endoneurial anoxia and leave no trace of nerve damage.

- **Stages:**
  a. numbness and tingling within **15 minutes**
  b. loss of pain sensibility after **30 minutes**
  c. muscle weakness after **45 minutes**
Relief of compression is followed by:

A) feeling is restored within 30 sec.

B) intense paraesthesia lasting up to 5 min.

C) full muscle power after 10 min.
NEURAPRAXIA (NON-DEG)

- **Mechanical pressure** (direct compression, traction, trauma) causing segmental demyelination.
- The axon still intact, transient loss of nerve function.
- Distal conduction preserved.
- Loss of some types of sensation and muscle power.
- Reversible physiological nerve conduction block followed by spontaneous recovery after a few days or weeks.
There is complete interruption of the axons in a segment of nerve. (demyelination and axon loss)

There is loss of conduction but the nerve neural tubes are intact.

It is seen typically after closed fractures and dislocations

( Wallerian degeneration )?
The denervated motor end plate and sensory receptors gradually atrophy.

If they are not re-innervated within 2 years they will never recover.

Axonal regeneration starts within hrs of nerve damage.

The new Axonal processes grow at a speed of 1-2 mm per day
WALLERIAN DEGENERATION

- It is a process that results when a nerve fibre is cut or crushed, in which the part of the axon separated from the neuron's cell body degenerates distal to the injury. This is also known as anterograde or orthograde degeneration.
Division of the nerve trunk, Neural tubes are destroyed

A Neuroma is formed (regenerating fibers + Schwann cells + fibroblasts)

Function may be adequate but is never normal even after surgical repair.
Spontaneous recovery cannot be expected unless surgically intervened.

There is rapid Wallerian degeneration.

Ex: Occurs in open wounds (knife cut, gunshot)
**HX AND PX:**

- **Always** examine for nerve injuries following any significant trauma, and again after manipulation or operation.
- Which nerve? What level? What is the cause? What degree of injury? Old or free injury?
- Ask patient if there is (1) numbness, (2) tingling or (3) muscle weakness in the target area.
- Ask about **onset** if it gradual or sudden? **Progression** rapid or slow? **Distribution** focal or generalized, distal or proximal? **Symmetry**? **Associated symptoms**?
• Signs: (1) Abnormal posture (wrist drop) (2) Atrophy of the muscles (3) Change in sensibility

• Sensory examination: light touch, pinprick, proprioception, vibration.

• Motor examination: muscle strength and power, muscle reflex.
Nerve loss in low-energy injuries is likely to be due to Neuropraxia.

And in high-energy injuries and open wounds to Axonotmesis or Neurotmesis
SPECIAL TESTS:

- **Tinel’s sign:**
  - Peripheral tingling provoked by percussing the nerve at the site of injury.
  - -ve in neuropraxia
  - +ve in neurotmesis due to regenerating sprouts

- **Electrodiagnostic tests:** (test the function of muscles and nerves)
  - Help to establish the **level** and **severity** of the injury, as well as the progress of nerve recovery
  - excludes neurapraxia ( Distal conduction preserved ) “No fibrillation”
  - does not distinguish between axonotmesis and neurotmesis.
PRINCIPLES OF TX:

Open injuries:
1. If the nerve is *cleanly divided*, end to end suture may be possible.
2. Paring of the stumps with a sharp blade, and if this leaves too large gap, nerve mobilizing can be done to prevent tension.
3. Nerve grafts can be used.

Closed injury:
More difficult to decide what to do…
Low degree injury: nerve sheath is likely intact we will wait to see if there is sign of recovery if not the nerve should be explored.
High degree force: early exploration.
Care of paralyzed parts:
While recovery is awaited:
1. **Skin** must be protected from friction damage and burns.
2. **Joints** must move twice daily to prevent stiffness.

- **Exploration is indicated:**
  - (1) if the nerve was seen to be divided and needs to be repaired;
  - (2) if the type of injury (e.g. a knife wound or a high energy injury) suggests that the nerve has been divided or severely damaged;
  - (3) if recovery is inappropriately delayed and the diagnosis is in doubt.
• It may be **best to leave the injured nerve alone** in case of:
  1. If the patient has adapted to the functional loss.
  2. If it is high lesion, and re-innervation is unlikely to occur within the critical 2 years.
  3. If there is pure motor loss which can be treated by tendon transfer.
BRACHIAL PLEXUS INJURIES:

**Causes**

1. **Closed injury**: Due to birth or due to bike trauma
2. **Open injury**: Due to penetrating or gunshot injuries
3. **Others (less common)**: Traction injuries, tumor removal, shoulder dislocations, surgical excision of cervical ribs, abnormal pressures due to faulty posture

- **Supraclavicular lesion**: In motorcycle accident
- **Infraclavicular lesion**: Fracture or dislocation of the shoulder
- **Clavicle fracture**: *rarely* causes damage to the plexus
CLINICAL FEATURES:

- According to the Level of lesion:

1) **upper plexus injury**: paralysis of shoulder abductors, external rotators and forearm supinators.
   - arms hangs close to the body and internally rotated and pronated.
   - sensation is lost along the outer aspect of arm and forearm.

2) **Lower plexus injury**: rare,
   - clawing due to intrinsic hand muscle paralysis.
   - sensation is lost along the inner aspect of arm.

3) **Total plexus lesion**(pan plexus)
   - paralysis and numbness of the entire limb.
ERB’S PALSY:

- Caused by injury of the nerves in the shoulder (C5-C6 axontmesis) at birth (during delivery).
- The baby lies one arm hangs close to the body and internally rotated and pronated. (paralysis of shoulder abductors, external rotators and forearm supinators)
- Reflux are absent
MANAGEMENT

Over the next few weeks:

1. Paralysis may recover completely: recover spontaneously by third month.
2. Paralysis may improve and then remain static.
3. Paralysis may remain unaltered: especially in the presence of Horner’s syndrome.

Physiotherapy while waiting for recovery.
Extend the child’s arm and turn the hand upwards.

Then raise the arm straight over the child’s head.
If there is no recovery by 3 months then operative intervention should be considered:

- If the root is not avulsed: nerve graft
- If the root are avulsed: advanced surgery

Types of surgery: Nerve graft, Nerve transfers, Muscle transfers, release of soft tissue contractures