Peripheral nerve injuries

Done by : Huda Etoom
Objectives

• Classification of nerves injury and pathology

• Upper limb- Brachial Plexus Injuries, Erbs Palsy, median nerve injuries, Unlnar Nerve injuries, Radial Nerve injuries, Digital nerves.

Anatomy of the PNS

• Composed of:
  Cranial nerves, Spinal nerves
  Connects the brain and the spinal cord with sensory receptors, muscles and glands.

Most Peripheral nerves contains both Afferent and Efferent neurons.

• Afferent Neurons: Sensory neurons
• Efferent Neurons: Motor Neurons.
Nerve fiber components
Structure of a nerve

- It has an outer covering which forms a sheath around the nerve, called the **epineurium**.

- Nerve fibers, which are axons, organize into bundles known as fascicles with each fascicle surrounded by the **perineurium**.

- Between individual nerve fibers is an inner layer of **endoneurium**.
Mechanism of injury:

- Damage to the peripheral nerves is called peripheral neuropathy.
- 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:

A. Ischaemia  
B. Compression  
C. Traction  
D. Laceration  
E. burning
Type of injury:

- Transient ischaemia
- Neurapraxia
- Axonotmesis
- Neurotmesis

Non degenerative
Transient ischemia

• 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

- mechanical pressure causing segmental demyelination (no loss of structural continuity)
- 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.
- The nerve is intact but mechanical pressure has caused demyelenation of axons in limited segment.
Axonotmesis

- There is complete interruption of the axons in a segment of nerve. (demylination 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.
Neurotmesis

• 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
• Seddon System and Sunderland System

• Sunderland system: expansion of Seddon’s system.

- Both are in common use -
Grades of Nerve Injury (Seddon 1942)

- **Neurapraxia**
  - Conduction block

- **Axonotmesis**
  - Axons divided

- **Neurotmesis**
  - Nerve divided
Sunderland classification:

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
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<tbody>
<tr>
<td>Type 1</td>
<td>Conduction block (neurapraxia)</td>
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<tr>
<td>Type 2</td>
<td>Axonal injury (axonotmesis)</td>
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<tr>
<td>Type 3</td>
<td>Type 2 + Endoneurium injury</td>
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<tr>
<td>Type 4</td>
<td>Type 3 + Perineurium injury</td>
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<tr>
<td>Type 5</td>
<td>Type 4 + Epineurium injury (neurotmesis)</td>
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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, proprioceptin, 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 explorition.

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.
Principles of Tx:

- 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 anatomy:
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 cause damage to the plexus
Clinical features: level of lesion, pre\post-ganglionic, type of injury?

• 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**: paralysis and numbness of the entire limb.
Clinical features:

• **Preganglionic or Postganglionic?**

  Preganglionic lesions: can't recover and surgically irreparable, Postganglionic lesions: can be repaired and capable for surgery

  1. **Preganglionic lesion**: Cause could be either birth or bike trauma  
  Characteristic feature: Presence of Horner's syndrome.

  2. **Postganglionic lesion**: absence of Horner's syndrome - prognosis is slightly better than the preganglionic lesion - positive Tinel's sign (tapping above the clavicle, produces tingling sensation in the anaesthetic limb

  Histamine test?

• **The type of damage?**

  neurapraxia and axontemesis show signs of recovery by 6 or 8 wks.  
  neurotemesis need early operation.
Management:

Emergency? Penetrating wounds, vascular injury, severe soft tissue damage.

All other closed injuries left until detailed physical examination and special investigation have been completed.
Obstetric brachial plexus injuries

- Caused by excessive traction on the brachial plexus (C5+C6+C7+C8+T1)
- during childbirth
- **Clinical features:**
  1. Difficult delivery
  2. Floppy or Flail arm.
- Further examination reveals one of the following: (day or two later)
  1. (A) Erb’s palsy C5 and C6
  2. (B) Klumpke’s palsy C8 and T1
  3. Total plexus injury.
Erb's palsy:

- Paralysis of the muscles in a baby's arm, caused by injury of the nerves in the shoulder 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
- Unilateral Horner syndrome (preganglionic lesion)
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.*
• *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.