**Trauma of Peripheral Nerves**

**Important quality of PNS (vs. CNS)** is

**Neurapraxia**

Examples: Saturday night palsy, tourniquet paralysis

**Electrophysiologic Testing**

**Seddon (1943)**

**Seddon** damage,

**Traumatic Neuroma**

Mechanisms of injury

**TREATMENT**

**SURGICAL**

**atrophy**

**Clinical Features**

**Pathophysiology, Classification**

**Neurotization** - nerve regeneration after its division

- most common injury is from blunt trauma and penetrating missiles.

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**Seddon (1943)**

**Seddon** damage,

**Traumatic Neuroma**

Mechanisms of injury

**TREATMENT**

**SURGICAL**

**atrophy**

**Clinical Features**

**Pathophysiology, Classification**

- nerves that do not regenerate well:
  1) long nerves
  2) nerves to fine muscles (high axon-to-myocyte rate)

Classification according to Seddon (1943):

A. **Neurapraxia** - myelin damage, axon intact - conduction block at site of lesion**;

B. **Axonotmesis** - axons are interrupted but endoneurium, Schwann cell tubes, connective tissue are intact

C. **Neurotmesis** - axon, myelin, and connective tissue components are damaged:

- preserved continuity of epineurium (afferent and efferent, NEUROMA IN CONTINUITY).
- nerve severed completely (Stump neuroma).
- at site of injury – traumatic degeneration; distally wallerian degeneration occurs; recovery occurs only as long as nerve ends are brought together.
N.B. unlike cellular repair in other areas, response of peripheral nerve to injury does not involve mitosis and cellular proliferation.

Sunderland (1951) further categorized nerve injuries according to degree:

1° injury = NEURAPRAXIA
2° injury = AXONOTMESIS
3° injury - some disorganization of internal structure of fascicles (loss of continuity of ENDONEURAL tubes, perineurium intact) - some regenerating axons are no longer confined to tubes they originally followed → new anomalous patterns of innervation, intrafascicular fibrosis; recovery may be incomplete.
4° injury - continuity of nerve trunk (epineurium persists), but its internal structure (PERINEURIN) is severely disrupted - organized regeneration is unlikely (involved segment is converted into tangled strap of connective tissue, Schwann cells, and regenerating axons → neuroma in continuity).
5° injury - EPINEURIR disrupted → perineural scarring, stump neuroma.
6° category (MaxKinnon and Dellon, 1988) - combination of above injuries.

Caricature of nerve injury levels related to train (rails = nerve fiber, track = endoneural tube, train = electric impulse traveling along fiber, electric wire = micro-vessels providing blood supply to nerve):

- physiological conduction block - local energy supply is interrupted: train cannot move in spite of intact nerve fiber; moment energy supply is restored (electric wire repair), train starts moving again; if electric wire system is more severely damaged (falling tree) - repair takes longer, still, rail is intact.
- neurapraxia - train is stopped because of local damage to rail (demyelinating block), while more distal parts of rail, as well as energy supply system, remain intact; local repair takes up to 6-8 weeks.
1. Loss of function supplied by nerve.
   N.B. deficits after injury may not be present immediately (e.g. may present months after crush injury - when scattering renders nerve dysfunctional).
2. Painful sequelae:
   a) pressure-sensitive traumatic NEUROMA.
   b) ENTRAPMENT syndromes.
   c) CAUSALGIA (after incomplete nerve injury). see p. S20 >>
   d) NEUROTOMIC DEAFFERENTATION PAIN.

**Clinical Features**

Injury to peripheral nerve may result in:

1. Loss of function supplied by nerve.
   N.B. deficits after injury may not be present immediately (e.g. may present months after crush injury - when scattering renders nerve dysfunctional).
2. Painful sequelae:
   a) pressure-sensitive traumatic NEUROMA.
   b) ENTRAPMENT syndromes.
   c) CAUSALGIA (after incomplete nerve injury).
   – see p. S20 >>
   d) NEUROTOMIC DEAFFERENTATION PAIN.

**Time Course**

All degrees of injury initially clinically appear the same:

- if neurological deficit is incomplete - injury is most likely neurapraxic.
- if neurological deficit is complete - injury may be neurapraxic, axonotmetic, or neurotmetic.

**Neurapraxia** - complete recovery in hours + weeks.

Recovery:

- pure motor or sensory nerves recover better than mixed nerves.
- recovery is better in radial and musculocutaneous nerves (course muscles) than in median or ulnar nerves (fin muscles); tubial division fares better than personal division.
- high injury of sciatic nerve – muscles always degenerate before reinervation – functional results are always bad!
- sign of recovery - distally migrating Tinel's sign (light tapping along nerve → paresthesia in sensory distribution of nerve) is evidence of functional recovery of C fibers (but does not guarantee good functional recovery).
  - if distal aspect of nerve is thinned progressively proximally, level at which sign is first elicited marks most distal point of small fiber regeneration.
  - absence of Tinel's sign distal to injury site 3-4 months post-injury suggests need for nerve exploration.
  - Tinel's sign demonstrates unstable regenerating axon.
- N.B. tapping over injury site itself is meaningless!
- N.B. distally migrating Tinel's sign or recovery of autonomic function in absence of sensory or motor recovery requires surgical exploration!!!

**Evaluation**

Determine:

1) Type of injury
2) Time injury occurred
3) Clinical condition at time of examination - legal and clinical implications (did nerve lesion occur at time of accident, or was it iatrogenic lesion that occurred during repair of patient's other injuries?).
- sensory and motor components must be evaluated separately.
- motor function suffers most!
- damaged nerve initially may appear normal in neurological examination!

**ELECTROPHYSIOLOGIC TESTING:**
- can support clinical suspicion of nerve injury or to evaluate nerve function if reliable neurological examination is impossible.

**EMG:**
- the only clinically useful diagnostic test!

- *denervation* changes (fibrillations and positive sharp waves) appear only after 2-5 weeks.
- *early EMG signs of reinnervation* - decreased insertional activity, decreased fibrillation, nascent polyphasic potentials.
- *nascent polyphasic potentials* - early return of solitary/few MUPs (indicative of muscle reinnervation), can be seen with few muscle fibers reinnervated, but clinical recovery requires reinnervation of many to several hundred motor units.

**NERVE CONDUCTION STUDIES:**
- not helpful clinically!

- *initially, all injuries have conduction block and intact distal portion*

  - **Early:**
    - Proximal stimulation: *neurapraxia* - slowing or conduction block.
    - Distal stimulation: normal (intact axons distal to any injury site - normal amplitude of compound muscle action potential CMAP).

  - **Late:**
    - Proximal stimulation: *neurapraxia* - normal (correspond to clinical recovery).
    - Distal stimulation: *neurapraxia* - normal (correspond to clinical recovery).

  - Reduced CMAP amplitude is observed by 7 days (wallerian degeneration).

**OPTIONAL TESTING:**
- to rule out bony and ligamentous injuries → radiographs.
- for fine anatomic detail of soft tissue, MRI is much more effective than CT.
- MRI can detect signal changes in denervated muscle as early as 4 days after injury! (useful differentiation of neurapraxic from axonotmetic injury).
- MR neurography can visualize both normal and abnormal peripheral nerves.

**MEDICAL TREATMENT**
- initial treatment of choice in all cases except when nerve discontinuity is known (→ surgery).
- analgesics to control pain.
- measures to decrease endoneurial edema:
  1) antivirals
  2) steroids
  3) hyperbaric oxygen (HBO)
- ciliary neurotrophic factor (CNTF) enhances motor neuron survival - continues to undergo research.

**SURGICAL TREATMENT**

**TREATMENT OF (PAINFUL) TRAUMATIC NEUROMA**

**STUMP NEUROMA:**
- daily ultrasound for 5-10 sessions
- injection of corticosteroids or analgesics into neuroma or surrounding area
- cryotherapy
- continuous tight bandaging of stump.
- sharply sectioning nerve proximal to neuroma → embedding freshly sectioned nerve end in adjacent deep soft tissue (surrounded by muscle).

*N.B. most common cause of stump pain is poorly fitted prosthetic socket; other common cause is spur formation at amputated end of bone; diagnosed by palpation and x-ray; H: surgical resection.*

**NEUROMA IN CONTINUITY:**
- A) complete loss of motor function of 3-12 mo duration + intraoperative nerve action potentials show no regeneration across site of injury: neuroma excised → primary neurorrhaphy (or cable grafting).
- B) intraoperative nerve action potential show recovery of function: external or internal (interfascicular) neurolysis.

**RESEARCH ASPECTS**

Experimental Lesion Paradigms:
- **axonotomy** - crush injury without discontinuing nerve; easy to perform; main disadvantage: fastness of regeneration process in animals.
B. NEUROTOMY - complete transaction of whole nerve; need for technically challenging microsurgical nerve reconstruction.

**BRACHIAL PLEXUS TRAUMA**

**BRACHIAL PLEXUS BIRTH TRAUMA** → see p. Ped9 >>

**ETHOLOGY**

1) clavicle fractures with disruption of the peripheral brachial plexus
2) avulsion of cervical roots by traction on the brachial plexus

**CLINICAL FEATURES**

- intractable long-term pain of the upper limb in 20-30% of patients (much more common [up to 90%] in avulsions than in peripheral injuries).
  - pain may begin immediately after the trauma or can be delayed up to months later.
  - pain projection depends primarily on the extent of the injury and the number of avulsed roots involved; it may include the whole upper limb.
  - pain is debilitating, burning, electrical, or sharp.
  - pathophysiology of pain: afferent input deprivation, caused by the lack of inhibitory effects of the large-caliber sensory fibers after division of the dorsal rootlets, results in spontaneous discharges in DREZ – rationale for DREZ lesioning procedures. see p. S20 >>

**BIBLIOGRAPHY** for ch. “Peripheral Neuropathies” → follow this [LINK] >>