Peripheral Nerve Injury

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Objectives

► Review the different neural structures involved in Seddon’s classification of traumatic peripheral nerve injuries

► Discuss the clinical support needed to allow for optimal functional recovery following a significant peripheral nerve injury

► Describe the processes and the time course for peripheral nerve regeneration

► Clinically relate to the timing of electrodiagnostic studies
Anatomy
Anatomy
Anatomy
Seddon’s Classification of Traumatic Peripheral Nerve Injury

- Neuropraxia
- Axonotmesis
- Neurotmesis
Neuropraxia

- Manifestation of conduction block where nerve impulses cannot be transmitted across a lesion site even though axon continuity remains intact.
Axonotmesis

- A state where there is axonal loss with subsequent Wallerian degeneration. The connective tissue infrastructure however remains relatively intact.
Neurotmesis

- Condition where axonal loss occurs in combination with prominent disruption of the connective tissue infrastructure of the endoneurium, perineurium and/or epineurium
Supportive Care Needs
Skin Integrity and Edema Control

- Loss of natural lubrication
- Pressure monitoring
- Positioning
- Compression
- DVT prevention
Soft Tissue Management

Contracture prevention
- ROM program for muscle and tendon
- Resting splints

Contracture management
- ROM program
- Dynamic splinting/serial casting
- Manipulation/capsular expansion
- Capsular release
The majority of traumatic nerve injuries are relatively non-painful. Of those that are painful, the majority improve with time. Management is to get to tolerable status. Psychological support.
Functional Independence

► Self care

► Mobility

► Adaptive environment

► Social and work considerations
Psychological Response

- Knowledge and independence brings optimism
- Most improve in time functionally
- Most improve in time for pain control
- Patience
Ulnar Nerve Injury
Motor Recovery Post Peripheral Nerve Injury

18 year old female injured the ulnar nerve at the elbow during a fall

20% of the motor axons remain in continuity yet 50% have neuropraxic lesions

80% axonotmesis injury with 20% of the motor axons able to achieve axonal terminal regeneration to the target muscle – 1st DI
Recovery from Neuropraxia
Remyelination

- 20% in continuity yet 50% have neuropraxia
- 80% axonotmesis with 20% of the motor axons able to achieve terminal regeneration
Axonal/Nodal Sprouting Potentiation
Axonal/Nodal Sprouting

- 20% in continuity yet 50% have neuropraxia
- 80% axonotmesis with 20% of the motor axons able to achieve terminal regeneration

Recovery of Motor Function

Weeks from Injury

% Function

Recovery of Motor Function
Muscle Hypertrophy
Muscle Atrophy/Hypertrophy

- 20% in continuity yet 50% have neuropraxia
- 80% axonotmesis with 20% of the motor axons able to achieve terminal regeneration
Axonal Terminal Regeneration
Axonal Terminal Regeneration

- 20% in continuity yet 50% have neuropraxia
- 80% axonotmesis with 20% of the motor axons able to achieve terminal regeneration
Alternative Options for Function

- Nerve grafting/transfer procedures
- Free muscle transfers
- Tendon transfers
Summary

► Tremendous capacity for recovery of function over time

► Tremendous capacity for improvement of pain over time

► Knowledge and independence brings optimism
We've got to work on our timing.

Stop global warming!
Electrophysiologic Testing

Physiologic response to injury

Processes of neural regeneration
Premorbid Conditions

- PNI solely related to the current injury, an old injury, or acute on chronic injury
- Spontaneous activity of the sarcolemma
  - Occurs beginning 3 weeks post injury
  - In partial injuries, potentially gone at 3.5 months
- Motor unit morphology
  - Polyphasia present at 8 weeks
  - Maturing parameters at 5-6 months
Localization of Injury

► Nerve conduction studies
  - If complete functional loss, prior to wallerian degeneration (study preferably before day 4)
  - Neuropraxia resolves over a 2-4 month time frame dependent upon the degree of injury

► Distribution of involvement on needle exam (study between 4-6 weeks post injury)
  - Spontaneous activity
  - Motor unit morphology/recruitment

► Ultrasound Imaging
"Give me the bad news, Doc. Am I going to live?"
Determination of Severity

- Wallerian degeneration occurs by day 8 for motor axons and day 10-11 for sensory axons. (study post day 12 but prior to week 6 when nodal sprouting occurs)

- Motor unit estimation
  - Neuropraxia resolves over 2-4 months
  - Motor unit recruitment

- Nodal sprouts mature by 3-4 months post injury with muscle fiber hypertrophy occurring by 5-6 months post injury
Final Functional Recovery

- **Terminal regeneration (completed by 2 yr)**
  - Never complete regeneration in severe PNI
  - Proximal more typical 30%, distal 10-15%

- **Sensory function**
  - 20% in continuity usually normalizes gross sensations
  - 5% allows for protective sensation
  - Motor control

- **Motor nodal sprouting**
  - Dependent upon age (4 fold tapering to 1-1.5)
Age Happens
Aging and Sarcopenia

-Lexell et al 1988
-Campbell et al 1973
-Faulkner et al 2007
Conclusion

► Early decision within days of injury
  ▪ Premorbid conditions
  ▪ Wallerian degeneration

► 4-6 weeks post injury
  ▪ Degree of axonal continuity for NCV studies/twitch force
  ▪ Localization by the needle exam

► 4-5 months post injury
  ▪ Degree of motor axonal continuity on needle exam

► 2 years post injury
  ▪ Quantify degree of final neural continuity after terminal regeneration
  ▪ Discussion of functional losses with aging.