CHAPTER **52**

NERVE INJURY

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Nerve injuries are often complex in nature and difficult to assess. Classifying the nerve injury enables the physician to better understand the degree of damage and determine the probable course of recovery. The degree of injury is further ascertained by an accurate history and physical examination, as well as through diagnostic tests. The information compiled is utilized to determine the treatment plan.

ANATOMY

The peripheral nerve trunk is composed of nerve fibers, endoneurium, fasciculi, perineurium, and epineurium. Nerve fibers vary from 0.5 to 20 μm in diameter to 0.5 mm to 1 m in length.[1] Nerve fibers are classified as myelinated or unmyelinated, with fibers less than 2 μm generally being unmyelinated.[1] Schwann cells are wrapped circumferentially around the axons in both types, with the primary difference being that one axon in the myelinated fiber is surrounded by only one Schwann cell, whereas multiple axons can be surrounded by a Schwann cell in the unmyelinated fiber. The myelinated fiber is interrupted by areas of no myelin, where Schwann cells meet, called nodes of Ranvier. Conduction along the myelinated fibers is by skip depolarization from node to node by a process known as saltatory conduction. The conduction along unmyelinated fibers is by continuous depolarization. Therefore, large myelinated fibers have higher conduction rates.

Each nerve fiber is surrounded by a loose connective tissue layer called the endoneurium, a group of nerve fibers with each having its own endoneurium form a fasciculus. Each fascicular group is surrounded and bound by dense fibrous tissue called the perineurium, which serves to protect the nerve fibers and maintain the intrafascicular pressure. Rupture of the perineurium leads to herniation of the intrafascicular tissue. The perineurium has a significant role in maintaining the integrity of the nerve trunk under tension.[2] The perineurium is then surrounded by a thick, loose areolar connective tissue called the epineurium. The epineurium constitutes approximately 30% to 75% of a peripheral nerve's cross-sectional area.[1] The epineurium affixes loosely or firmly to the surrounding tissues, allowing for some mobility as the nerve courses through the extremity.

The micro-vascular system of the peripheral nerve, the vasa nervorum, is a complex network composed of an intrinsic and extrinsic vascular system. The extrinsic system is constituted by regional vessels in the surrounding tissues that penetrate the epineurium along the nerve course. The intrinsic supply consists of longitudinally directed arterioles that course through the perineurium anastomosing with the capillary beds within the endoneurium. The extensive anastomoses within the nerve trunk allow the nerve to function, even if one or more nerve arteries is interrupted. However, compression can obstruct the vascular supply, leading to ischemic changes that produce failure in conduction and possible nerve damage if the intensity and duration are prolonged (Table 1). Compression can also impair venous outflow, producing edema within the nerve.

Table 1

COMPRESSION INJURY OF NERVES

EXTERNAL COMPRESSION

1. Tourniquet Palsy
2. Habitual Postures
3. Compression against a firm object (cast, shoes, ski boots, roller blades, etc.)

INTERNAL COMPRESSION

1. Fracture Callus
2. Constricting Fibrous Tissue
3. Enlarging Tumor, Cyst, Aneurysm
4. Compartment Syndrome

CLASSIFICATION

During World War II, Seddon classified nerve injury according to the degree of damage to the axon, 1) neuropraxia, 2) axontemesis, and 3) neurotemesis.[3,4] The classification scheme is based on the specific cause of injury (Table 2).

Neuropraxia was originally described by Seddon as a transient cessation in nerve function.[3] The injury produces a local conduction block with no axonal damage. The injury is usually minor, with compression and mild contusion causing a conduction deficit without anatomic disruption. This may often involve motor paralysis, sparing sensory and sympathetic function. The larger and more myelinated fibers are affected most commonly. Recovery from neuropraxia is complete after hours to several weeks.

Seddon described axontemesis as a lesion in continuity.[3,4] Injury to the nerve fiber produces loss of axonal continuity and Wallerian degeneration, but the endoneurial tubes are intact. Degeneration occurs distal to the site of injury with absence of nerve conduction. The quality of recovery is usually perfect due to the intact endoneurial tubes serving as a guide for the axonal regeneration. Recovery is complete in days to months with the regeneration of the fiber at 1 to 2 mm per day.

Neurotemesis was described by Seddon as a division of nerve.[3,4] Complete disruption of the nerve occurs with or without severance of the epineurium. There is complete sensory and motor paralysis. Surgical repair is a necessity for the best chance of recovery. The recovery from neurotemesis is always imperfect.

Seddon's classification was modified by Sunderland. (Table 3) He classified nerve injuries into five degrees incorporating neuropraxia, axontemesis, and neurotemesis.[5] Sunderland's classification is more practical with the advancements in surgical technique.

A first degree injury occurs with interruption of axonal conduction corresponding to neuropraxia. Axonal continuity is preserved with no Wallerian degeneration. First degree injury results from compression and contusion. Recovery is complete in hours to weeks.

A second degree injury corresponds to axontemesis with disruption of the axon and presence of Wallerian degeneration. The rate of recovery is determined by the rule of axon regeneration, usually 1 mm to 2 mm per day. The endoneurial tube is intact, allowing for complete recovery in days to weeks.

A third degree injury occurs with disruption of the axons and severance of the endoneurial tubes,

Table 2

THREE TYPES OF NERVE INJURY

FEATURE	NEUROPRAXIA	AXONTEMESIS	NEUROTEMESIS
ANATOMY	Preserved	Preserved	Disrupted
PATHOLOGY	Myelin sheath distortion	Wallerian degeneration Endoneurial tube preserved	Complete disorganization
MOTOR PARALYSIS	Complete	Complete	Complete
MUSCLE ATROPHY	Minimal	Progressive	Progressive
SENSORY PARALYSIS	Usually intact	Complete	Complete
AUTONOMIC PARALYSIS	Usually intact	Complete	Complete
QUALITY RECOVERY	Perfect	Perfect	Imperfect
RATE RECOVERY	Days to weeks	1-2 mm/day	1-2mm/day if surgery
TREATMENT	Conservative	Conservative	Surgery

Table 3

CLASSIFICATIONS OF NERVE INJURY: SEDDON AND SUNDERLAND

Type/Degree	First	Second	Third	Fourth	Fifth
Neuropraxia	■				
Axontemesis		■	■		
Neurotemesis				■	■

similar to a combination of axontemesis and neurotemesis injuries. The disorganization within the fasciculus leads to fibrosis, complicating regeneration. Regeneration is irregular with predictable deficits. The pattern of recovery should be evident in six months. Surgical intervention must be considered for recovery.

A fourth degree injury results from disruption of the fascicles and perineurium though the epineurium is intact. Wallerian degeneration is present with the probability of scarring greatly increased from third degree injury. Spontaneous recovery is rare and surgical intervention is indicated.

A fifth degree injury implies complete loss of continuity of the nerve trunk, similar to neurotemesis (Table 4). Motor, sensory, and sympathetic function are not present. The injury occurs commonly in lacerations and gunshots. Surgical intervention is required for the possibility of any recovery.

Table 4

TRAUMATIC INJURIES TO NERVE

DIRECT

Laceration
Crush or Contusion
Traction
Thermal Injury: cold, heat
High Velocity: gunshot
Radiation
Injection of Noxious Agent

EVALUATION

Peripheral nerve injury can produce symptoms of sharp, burning, or stabbing pain, tingling, numbness, and weakness. The involved extremity is best stabilized before evaluation to prevent further injury. A careful history can provide significant information as to the nature and severity of injury. The physical examination may involve an evaluation of the central, peripheral, and autonomic nervous systems. Motor and sensory evaluation may include manual muscle testing, deep tendon reflexes, sharp/dull, light touch, proprioception, two-point discrimination, vibratory perception, and temperature.

Tinel's sign is an accurate indicator of sensory nerve regeneration because the ends of the regenerating axons are extremely sensitive to pressure. The level of regeneration can be determined by percussion along the nerve course until paresthesias appear. Nerve regeneration should progress distally at 1 mm to 2 mm per day in the lower leg and 1 mm per day in the foot. Once the rate of recovery slows or ceases, or motor recovery does not proceed with sensory improvement, surgical exploration of the nerve may be indicated. Valleix's sign may also be seen with radiating pain proximally with percussion.

Light touch sensation is difficult to quantitate. The use of Semmes-Weinstein esthesiometer is a useful means of testing for light touch in a clinical setting. Two-point discrimination can be quantitated by means of a Boley gauge, caliper, two pins, or paper clip. The pressure should be firm, but should not blanch the skin. Two-point discrimination below the knee is normally 40 mm to 50 mm, diminished at 50 mm to 80 mm, and absent at greater than 80 mm.[6]

Vibratory sensation may be assessed with a tuning fork. using tuning forks with different frequencies may be of some value. A 30-cps (cycles per seconds) tuning fork is useful in evaluating the Meissner's corpuscles (corpuscles of light touch), and a 256-cps tuning fork is for evaluating the pacinian corpuscles (corpuscles of pressure).

Temperature is often difficult to accurately assess, due to the calibration of the thermometers, and is not practical in a normal office environment.

The patient's joints are used to assess proprioceptive by moving the metatarsophalangeal joint or the ankle joint dorsally or plantarly, and asking the patient the position of the joint. Manual muscle testing is necessary to assess level of injury. Circumferential measurements of muscles at known levels may be used to monitor muscle atrophy.

Sympathetic denervation leads to absence of sweating, poor response to cold and heat, and perhaps to reduced resistance to physical stress. A psychiatric evaluation may be employed to evaluate possible unforeseen difficulties adding to recovery. The information obtained may greatly aid in the treatment plan.

There are a number of neurophysiologic tests to aid in the diagnosis and evaluation of nerve injury. These tests include electromyography, nerve conduction, H-reflex, F-wave, and somatosensory evoked potentials. EMG changes will not be evident with a neuropraxia, but will become apparent two weeks following axontemesis. Nerve conduction velocity will be slowed in neuropraxia, significantly slowed in axontemesis, and absent in neurotemesis. There also have been studies utilizing MRI, specifically short-tau inversion recovery (STIR) images. MRI utilizing STIR is being used to assess denervation of muscle. The amount of increase in the signal correlates with the degree of denervation seen on EMG and weakness found on clinical examination. The STIR signal appears greatest with the clinical manifestation of maximum injury and normalizes with clinical recovery of function.[7]

TREATMENT

The goal of any treatment is to prevent further injury and sequelae, i.e., chronic pain symptoms. Patients with nerve injuries should have the involved extremity immobilized and the degree of injury assessed. Conservative treatment is indicated in acute and low velocity types of nerve injury. Surgical intervention may be necessary in open wounds, severe dislocated fractures, and chronic nerve symptoms.

Conservative treatment is indicated in first and second degree injuries. After the extent of injury is assessed, the injured extremity is immobilized via a posterior splint or Jones compression cast. A Jones compression cast is the most advantageous way of controlling edema and preventing further injury. Rest, ice, compression, and elevation are also indicated. Nonsteroidal anti-inflammatory drugs may be helpful.

Physical therapy modalities such as, stretching exercises, ultrasound, and TENS are at times helpful for maintaining or increasing joint mobility, and preventing contractures. Regional nerve blocks can aid in breaking the pain cycle. In cases where there is residual deformity from nerve injury, bracing and/or surgical stabilization procedures may be of benefit.

Surgical intervention is indicated in all third degree injuries or when the rate of recovery does not coincide with the type and usual course of injury. Appropriate surgical technique involves the use of magnification via loupes or a microscope for better visualization. In acute injuries neurorraphy, primary repair, or nerve graft reconstruction are indicated. In injuries producing chronic nerve symptoms, neurolysis, releasing of the nerve, or neurectomy may be required.

CONCLUSION

The classification schemes developed by Seddon and Sunderland allow the physician to better understand the degree of nerve injury and probable course of recovery. Nerve injuries are often challenging due to the frequency of mixed nerve lesions. In instances where one witnesses an unusual course of recovery or with possible non-compliance, a psychiatric evaluation may be indicated. Various neurophysiological tests can aid in the determination of the degree of nerve injury.

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