Management of Missile Peripheral Nerve Injuries

Part I: History, Pathophysiology, Clinical Presentation, and Diagnostics

Kenan I. Arnautović, M.D., Ali F. Krisht, M.D., and B. Lee Ligon, Ph.D.

Learning Objectives: *After reading this article, the participant should be able to:*
1. *Recall the pathophysiology, classification, and ballistics of missile peripheral nerve injuries.*
2. *Describe the clinical presentation of missile peripheral nerve injuries.*
3. *Explain the basics of missile peripheral nerve injury diagnostics.*

Missile peripheral nerve injuries (MPNIs) usually receive secondary consideration, including deferred surgical treatment, mainly because they are not life threatening. Approximately 70% of missile wounds that require exploration include either a complete or partial section of nerve. Surgical management of these wounds is very demanding, requiring considerable attention during the initial examination and a lengthy, intricate surgical procedure, detailed knowledge of peripheral nerve anatomy, experience using microsurgical techniques, and support from a team of experts who provide various diagnostic, therapeutic, and rehabilitation services. Patients are followed for months and even years, at the end of which time the gain may be barely measurable, because the return of neurological function is a very slow process.

MPNIs are relatively uncommon injuries in peacetime. Current increases in violence and in the availability of firearms have, however, resulted in a rise in the incidence of MPNIs, even in children, with a much higher proportion of low-velocity injuries. During times of war, the incidence of these injuries increases significantly, providing opportunities to gain valuable surgical experience and to make contributions to the knowledge about their epidemiology, pathophysiology, and treatment.

Background

Galen of Pergamon (130–200) argued that nerve lesions lead to convulsions and are irreparable. Rhases the Experienced (Al Rhazi) (850–923) performed one of the first recorded nerve sutures, the results of which are not known. Some 300 years later, Salicetti from Bologna performed a surgical repair of an injured nerve that is described in the 13th-century text *Cyrurgia*.

Gunshot wounds were first recorded in Europe during the Hundred Years' War (1337–1453). In 1497, the Alsatian army surgeon Hieronymus Brunschwig pronounced these wounds "poisonous." In the 16th century, the French army surgeon Ambroise Paré (1510–1590), who described causalgia and phantom limb pain, was pessimistic about the idea of restoring nerve function after a surgical repair. During the same era, two Elizabethan surgeons, Thomas Gale (1563) and William Glovers (1588), advocated removal of badly traumatized and contaminated tissue. Ferrara performed the first detailed operative procedure for suturing severed nerves in 1608, with split tortoise tendons previously soaked in red wine used as suture material. In 1787, Arnemann of Germany performed the first "modern" suturing of a divided nerve; unfortunately, the distal segment had collapsed and the nerve did not regenerate. Principles of debridement devised by DeSault during the later 18th century were popularized by Larrey, military Surgeon-in-Chief to Napoleon. Gutrie voiced the opinions of British Army surgeons at the Battle of Waterloo when he advised no treatment for completely severed nerves.

Category: Peripheral nerves

Key Words: Peripheral nerves, Missile injuries, Pathophysiology, EMG

Surgeons during the Napoleonic Wars advised prompt amputation for all gunshot wounds of the thigh that were deemed particularly lethal.

During the 19th century, advances were made in the surgical treatment of nerves. In 1836, Baudens performed the first "modern" epineural nerve suturing, and in 1854 Von Langenbeck reported the first successful repair of a median nerve laceration, with complete return of function 1 year later. Subsequently, Silas Weir Mitchell, who after doing animal experiments recommended suture for completely severed nerves and also coined the term "phantom limb pain," undertook the first organized investigation of MPNIs at Turner Lane Hospital in Philadelphia during and after the American Civil War (1863–1872). Toward the end of the l9th century, the first nerve grafts were performed, unsuccessfully by Albert in 1885 and then successfully by Mayo-Robson in 1896. In 1891, Gardner transposed an ulnar nerve from its cubital canal. In 1898, Woodruff reported that when a missile passes through something, a cavity is created in the target medium into which air is sucked.

In World War I, Dejerines vigorously campaigned for resection of every cicatrix associated with MPNI as early as 3 weeks after the injury. Organized investigations of MPNIs were performed during and after World War I by two doctors on opposite sides of the battle line, Jules Tinel of France and Paul Hoffmann of Germany. Each doctor, working independently, developed a test for the purpose of examining the regeneration of an injured nerve and localizing the level of damage to a nerve. The test is known in most countries as Tinel's sign, after Tinel, who described it in October of 1915; in most German-speaking countries, it is called the Hoffmann-Tinel test, recognizing the contribution of Hoffman, who had described it earlier in March of the same year. In 1915, Henry Gray recommended the "debridement" technique, which gained popularity during World War I.

During World War II, Seddon (1945), one of the pioneers of nerve grafting techniques, continued the study of MPNIs and introduced a classification scheme that is still in use. Another study of MPNIs and classification of nerve injuries was introduced in 1951 by Sunderland. Smith, a plastic surgeon, and Kurze, a neurosurgeon, introduced the microscope into surgical practice in 1964. In 1967, Bora published his work on fascicular nerve sutures, and in 1972, Millesi reported his development of interfascicular nerve grafting and interfascicular neurolysis.

After slow but steady development in the treatment of nerve injuries, including MPNIs, the modern era of treatment of these wounds began less than 100 years ago. The vast majority of MPNIs occurred in the 20th century, accompanied by improvements in knowledge, surgical experience, and operative techniques. Generally, MPNIs have appeared more difficult and complex in each subsequent war.

Classification Schemes

Currently, two classification schemes of nerve injuries are used: Sunderland's (five degrees) and Seddon's (three degrees). We have found Seddon's scheme more practical, with nerve injuries classified as neurapraxia, axonotmesis, or neurotmesis. In the case of neurapraxia, the nerve elements remain intact, while a physiologic block in nerve conduction (usually in the motor fibers) occurs. In axonotmesis, the axon-Schwann cell complex is injured, but the endoneurium and other structures are not. Neurotmesis corresponds to the third, fourth, and fifth degrees of nerve injury in Sunderland's classification, as follows:

- 3rd degree: lesion of the endoneurium, all above (more superficial) structures spared;
- 4th degree: lesion of the perineurium, all above structures spared; and
- 5th degree: lesion of the epineurium, the nerve is divided.

Ballistic Features

During times of war, MPNIs are caused by mortar, tank, or cannon shells; grenade particles; and numerous types of bullets (including very dangerous exploding bullets that are analogous to artillery shells) that produce particles of various dimensions, shapes, and velocities. In peacetime, MPNIs

also can be caused by the impact of objects such as glass particles or irregular metal fragments traveling with considerable speed, as well as by handguns and other weapons that are commonly used in domestic violence and street and gang fighting. The extent of the injury varies according to several factors: the caliber of the weapon; the kinetic energy, which varies with the square of the velocity and weight; the mass, shape, and temperature of the missile or shrapnel; the angle at which the object enters the skin; and the deformation or fragmentation of the missile object on impact. Several traumatic mechanisms contribute in the etiology of MPNIs (Table 1). Factors that cause or complicate MPNIs (Table 2) also should be taken into consideration.

Although the speed of a missile decreases as the distance from the origin of discharge increases, the weapons of modern warfare have an extremely high initial velocity. When they explode, they produce a large number of metal particles and generate very high temperatures. Based on their velocities, missiles can be classified into four categories: (1) low velocity (<350 m/sec); (2) medium velocity (350–700 m/sec); (3) high velocity (700–1000 m/sec); and (4) ultra-high velocity (>1000 m/sec). Low-velocity missiles (seen in "peacetime" injuries) damage the tissues mainly by lacerating and crushing, whereas high-velocity missiles (seen in wartime) cause tissues to be compressed, expanding the missile track into a cavity.

Table 1. Traumatic Elements in Etiology of MPNI

Mechanical	Physical	Ischemia of Nerve
Compression, contusion, or laceration	Thermal radiation	Lesion of parent artery
Stretch or traction stretch secondary to hematoma		Lesion of segmental arterial perforators
Bone fractures and dislocations		Combination of the two

Table 2. Factors That Cause or Complicate MPNIs

Factors That Cause MPNIs	Factors That Complicate MPNIs
Direct tissue disruption and laceration	Destruction of neighboring tissues (e.g., vascular, bone, muscle)
Shock wave injury, with amplitudes up to 80 kg/cm^2	Development of traumatic or hemorrhagic shock
Temporary tissue cavitation	Wound contamination and the risk of wound infection (frequently with gram-negative and anaerobic bacteria)

Pathophysiology

Neural Injuries

Neural injuries occur at both macroscopic and microscopic levels. At the macroscopic level, the nerve itself can be either completely divided or connected by only a bridging track of fibrous tissue; partially divided, with a remaining track of nervous substance; or "in continuity," which usually accounts for the majority of serious injuries.

The microscopic and ultrastructural damage to the nerve usually is severe and extensive. Demyelination and wallerian degeneration occur distal to the site of injury. Destructive enzymes are released in the axonal segments, and myelin fragments are phagocytized by pluripotent Schwann cells. By the end of the third week postinjury, most of the cellular debris has been eliminated, the Schwann cells are ready for remyelination, and the injured site is prepared for neuronal regeneration. The nerve cell body enlarges in order to synthesize a large amount of structural material and transport it over a significant distance. The reconstruction of lost axoplasm and the recovery of peripheral connections are provided by the formation of ribonucleic acid, resulting in increased cytoplasmic protein synthesis. This accelerated activity also peaks by the end of the third week postinjury, at which time the supportive glial cells around the involved nerve cells provide the optimal environment for the regenerating neuron. Because of the excessive amount of foreign material, tissue debris, or accompanying ischemia, considerable inflammatory response occurs. This leads to fibrous proliferation of the internal epineurium, as well as of the muscular perimysium, bone periosteum, and muscular fascia, producing excessive scar formation, which is made even worse by inflammation and hypoxia.

Muscle Cell Changes

The postinjury changes of the muscle cells (as the important end organs) also are degenerative in nature. Because the damaged nerve does not provide the usual repetitive stimulation, the muscle cells atrophy and shrink. The endomysium and the perimysium thicken, and the muscle spindles undergo atrophy. If this status lasts longer than 24 months, complete, irreversible atrophy of the muscle occurs, precluding any improvement of motor function.

Clinical Presentation

The clinical presentation of MPNIs is not consistent because all of the degrees and various combinations of injuries can occur in any single patient. An accurate assessment of sensory loss requires the conscious cooperation of the patient.

Neurologic Status

The initial neurologic evaluation of a patient with MPNIs includes an assessment of the loss of motor, sensory, or autonomic functions. Motor paralysis and sensory loss appear immediately, followed within weeks by hypotonia and muscle atrophy. The borders of sensory innervations present different patterns in different patients, following the rule that the analgesic zone is narrower than the anesthetic one. For this reason, patients with similar MPNIs may present with different sensory losses. Thermanalgesia, loss of pressure and posture, vibration sensations, analgesia, and anesthesia also can be noted.

Autonomic nerve fibers of the skin run with the sensory fibers in the same distribution. Pseudomotor paralysis of

these nerve fibers may result from MPNIs, producing absence of sweating in the involved area. The skin temperature is hot at first, followed within several weeks by a change to cold: these changes usually are seen in patients with median, tibial, and ulnar nerve injuries. The skin is reddish or even cyanotic. Trophic changes occur after some time. The skin becomes smoother and shiny and, due to the lack of sweating, dry and thin; it also peels readily. The extent of skin keratinization increases. The nails become long, curved, and dry, and often have transverse ridges.

Pain

The pain that often accompanies MPNIs may have various manifestations (e.g., hyperalgesia, aching, spreading pain, tingling) and may be caused by a number of factors. A relatively rare phenomenon (2%–4% in World Wars I and II), is causalgia, which usually occurs in patients between 20 and 40 years of age who are prone to changes of mood and agitation. It may occur as early as 1 week following the injury. The pain is similar to that experienced with trigeminal neuralgia and usually is associated with median or sciatic-tibial nerve lesions. The patient complains of a severe, intense, diffuse, burning pain and refuses to allow anyone to touch or examine the affected extremity (usually the hand), which should be kept in a wet, cold wrap. Trophic changes usually occur, and partial muscle paralysis and rapid muscle wasting are present. The treatment options for this syndrome are surgical management of the lesion (external, internal neurolysis, resection, or suturing and grafting), local anesthetics, peripheral sympathetic blocking, sympathectomy, physiotherapy, exercise, and sedation.

Vascular Injuries

The possibility of associated vascular injuries, the effects of which may not appear until several days following the injury, must be considered. The rupture of a parent artery can lead to the formation of a hematoma, a pseudoaneurysm, spasm, or thrombosis. The segmented arterial blood supply of the nerve usually is severed, leading to ischemia of the nerve. The nerve also can be compressed by a hematoma or a pseudoaneurysm. Immediate vascular treatment and reconstruction are necessary to prevent further sequelae. If the clot that initially plugs the tear in the arterial wall breaks later, further hemorrhage can occur, with the risk of subsequent development of thrombosis or a pseudoaneurysm a couple of weeks later. Should a major arterial trunk injury (most common in the brachial artery) remain undetected, Volkmann ischemic contracture develops (a relatively rare occurrence—0.4%). It is caused by relatively short duration of intense ischemia, followed by ischemic infarction of the forearm muscles and subsequent development of contractures and fibrosis. Prevention is the key to treating this pathologic entity, but if it occurs, excision of infarcted muscles, repair of the nerve, or various orthopedic reconstruction procedures should be performed.

Diagnosis and Clinical Evaluation

A thorough neurological examination is a salient part of preoperative investigation. The exact time, condition, and type of injury are very important, as is information about the weapon caliber and the type of ammunition. A complete diagnostic work-up consists of physical, motor function, and sensory function examinations. During the physical examination, it is important to assess the mechanism of injury, the site and level of injury, the injured nerve(s), the severity of the injury, and the presence of other associated injuries (e.g., vascular, muscular, bony). Radiographs of the extremity in at least two projections can reveal retained foreign metal fragments or bone particles, which often accompany MPNIs. The motor function examination assesses the extent of the lesion—each nerve and corresponding muscle has a specific test for evaluating its function. The most widely used test is based on the following semiquantitative scale:

- M0: complete paralysis;
- Ml: palpable muscle contraction;
- M2: active joint motion with elimination of gravity;
- M3: contraction or full joint motion against gravity;
- M4: contraction or full joint motion against gravity and resistance; and
- M5: full range of motion—normal contraction.

The sensory function examination should include touch, pressure, pain, temperature, deep sensation, and two-point (static and movement) discrimination. The following scale can be used:

- S0: absence of sensation in an autonomous area;
- Sl: presence of deep cutaneous pain and sensation;
- S2: presence of some degree of superficial cutaneous pain, tactile sensation, and two-point discrimination;
- S3: presence of appreciable sensation, but no localization;
- S4: presence of sensation with diminished acuity; and
- S5: normal sensory function.

Tinel's sign, a simple sign for examining the regeneration of an injured nerve and localizing the level of damage to a nerve, remains a very important diagnostic factor. Proximal-to-distal digital or hammer percussion helps localize the site of nerve injury or repair. Distal-to-proximal percussion along the course of the nerve is performed until a tingling sign with distal sensation is obtained and continued further until tingling sensation is maximal. The degree of Tinel's sign "progression" or its absence has certain prognostic and operative indications.

The absence of sweating indicates the presence of injury (although the recovery of sweating does not predict the degree of useful motor or sensory function recovery). An evaluation of the patient's functional ability includes "pick-up" and "tactile cognition" tests that can help the physician determine the extent of damage to complex nerve functions. The full scale of clinical examinations should be performed at the initial assessment of the patient, during the course of treatment, and after the surgical repair.

Electrophysiological Testing

Electromyography (EMG) and nerve conduction studies provide the surgeon with significant information regarding the localization, severity, and pathophysiology of the nerve injury. An EMG recording is informative as early as 3 weeks after the injury. In addition to helping to define the extent and distribution of denervation and determine the length of time a muscle has been denervated, an EMG may detect signs of reinnervation such as a decrease in the intensity or the frequency of fibrillations and denervation potentials, a restored insertional activity, or the occurrence of regeneration potentials. A second EMG should be performed about 2 weeks after the first to evaluate the patient for possible surgical repair. It is important to emphasize that signs of regeneration do not guarantee useful functional recovery.

The outcome of nerve injury usually cannot be predicted without intraoperative determination of nerve viability. Intraoperative recording of nerve action potentials with operative microscope assistance can provide information about the transmission of impulses across the lesion site, which can help the surgeon manage the early exploration of the nerve, determine the type of lesion, and decide on the appropriate treatment (e.g., whether to perform a resection of a neuroma in continuity or to do interfascicular neurolysis alone). External neurolysis, which permits accurate placement of electrodes at sites proximal and distal to the lesion, usually is required before intraoperative stimulation and recording are performed. A bipolar electrode stimulates axons at one end of the operative field; simultaneously, a bipolar recording electrode senses conduction at another site along the nerve trunk, allowing the length of the regenerating axons to be traced and the length of the nonviable nerve to be identified. The nerve stimulation and conduction studies help determine the presence of neuropraxic injury if stimulation above the level of injury produces no motor activity, but stimulation below the level of injury does. Stimulation of a nerve that has undergone wallerian degeneration does not produce muscle contraction, whereas stimulation of regenerating nerves can produce muscle contraction several weeks before voluntary contraction returns. This delay is due to the time needed between the time at which the nerves reach the neuromuscular plate and that at which the neuromuscular plate matures. Recording of somatosensory evoked potentials can help localize preganglionic lesions of the brachial plexus.

Magnetic Resonance Imaging

Although the evaluation of peripheral nerve function traditionally has relied on the techniques described earlier, magnetic resonance imaging (MRI) recently has been tested to evaluate its efficacy for diagnosing both nerve and muscle disorders. The increased muscle signal was detected as early as 4 days after the onset of clinical symptoms, considerably earlier than the change on electromyography. Further, the MRI signal changes were reversible when the recovery of motor function occurred as a result of further innervation. These results indicate that MRI may play an important role in the prediction of clinical outcome and early determination of appropriate treatment after peripheral nerve injury.

Readings

Archibald KC: Clinical usefulness of EMG and nerve conduction tests in nerve injury and repair, in Jewett DL, McCarrol HR, Jr (eds): *Nerve Repair and Regeneration: Its Clinical and Experimental Basis.* St. Louis: CV Mosby, 1980, pp 209–212

Browne KM: Surgery of peripheral nerves, in Walker AE (ed): *History of Neurological Surgery.* Baltimore: Williams & Wilkins, 1951, pp 396–425

Daniel RK, Terzis JK: *Reconstructive Microsurgery.* Boston: Little Brown, 1977.

Dorfman LJ: Quantitative clinical electrophysiology in the evaluation of nerve injury and regeneration. *Muscle Nerve* 13:822, 1990

Franchetti MA: Trauma surgery during the Civil war. *Civil War Surg* 86:553, 1993

Friedman WA: The electrophysiology of peripheral nerve injuries. *Neurosurg Clin North Am* 2:43, 1991

Kraft GH: Fibrillation potential amplitude and muscle atrophy following peripheral nerve injury. *Muscle Nerve* 13:814, 1990

Lettes RM, Miller D: Gunshot wounds of the extremities in children. *J Trauma* 16: 807, 1976

McAllister RMR, Calder JS: Paradoxical clinical consequences of peripheral nerve injury: a review of anatomical, neurophysiological and psychological mechanisms. *Br J Plast Surg* 48:384, 1995

Parry GJ: Electrodiagnostic studies in the evaluation of peripheral nerve and brachial plexus injuries. *Neurol Clin* 10:921, 1992

Ragsdale BD: Gunshot wounds: a historical perspective. *Military Medicine* 149:301, 1984

Seddon H: *Surgical Disorders of the Peripheral Nerves.* Baltimore: Williams & Wilkins, 1972

Snyder CC: The history of nerve repair, in Omer GE, Spinner M (eds): *Management of Peripheral Nerve Problems.* Philadelphia: WB Saunders, 1980, pp 353-365

Spinner M: *Injuries to the Major Branches of Peripheral Nerves of the Forearm.* Philadelphia: WB Saunders, 1978

Sunderland S: *Nerves and Nerve Injuries.* 2nd ed. New York: Churchill Livingstone, 1978

Terzis JK, Dykes RW, Hakstian RW: Electrophysiological recordings in peripheral nerve surgery: a review. *J Hand Surg* 1:52, 1976

Terzis JK, Smith KL: *The Peripheral Nerve: Structure, Function, Reconstruction.* New York: Raven Press, 1990

West GA, Haynor DR, Goodkin R, et al: Magnetic resonance imaging signal changes in denervated muscles after peripheral nerve injury. *Neurosurgery* 35:1077, 1994