Chapter 38

Grading of Nerve Injuries

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INTRODUCTION

The type of initial nerve injury significantly alters the functional prognosis with or without nerve repair. Many stages of nerve damage, from simple block of conduction to complete nerve transection, have been described (Seddon, 1942; Sunderland, 1951), resulting in permanent paralysis in the absence of microsurgical repair. The goal of this chapter is to outline the main types of nerve injuries and then describe the main classifications of nerve injuries and evaluate their clinical and prognostic value. We propose a severity scale for nerve damage to supplement the Sunderland classification to which we add certain factors to improve prognosis of nerve recovery.

CLASSIFICATION BY TYPE OF NERVE DAMAGE

There are three main types of nerve injuries: section, stretching, and compression.

Nerve Sections

Nerve sections can be partial or complete, sharp or blunt. They often result from sharp wounds by glass, firearms, or knives (Figure 38.1). The wounds involve the upper limb in 74% of cases and males in 73.5% of cases (Kouyoumdjian, 2006). The wounds are isolated in more than 80% of cases. In lesions of ulnar and median nerves, associated vascular or tendon lesions affect the functional outcome (Kouyoumdjian, 2006).

Nerve Stretching

Stretching can occur with greatly displaced fractures. The extent of the neurological deficit depends on the degree of stretching. During traction, the perineurium elongates, and the axon, which was undulant, straightens out. Intraneural pressure exacerbates lesions of the axon and vascular elements. These lesions can lead to a neuroma in continuity (Alant et al., 2012) (Figure 38.2). If the stretching continues, the axon stretches and tears, as do the perineurium and epineurium. Stretching, such as that after resection of the radial head where cubitus valgus develops, may also be chronic, causing ulnar nerve stretch neuropathy (Mansat, Bonnevialle, Fine, Guiraud, & Testut, 1983).

Nerve Compression

Compression can be extrinsic or intrinsic. Extrinsic compression is more common in the median nerve in the carpal tunnel, ulnar nerve at the elbow, and common peroneal nerve at the knee. Intrinsic compression is caused mainly by nerve tumors.

Two mechanisms of peripheral nerve injury result from compression (Sunderland, 1976):

- Indirect mechanism through vascular compression. Acute or repeated prolonged compression may cause vascular stasis with increased vascular permeability and endoneurial edema formation. This fascicular edema impairs nerve function either directly by modifying the axonal flow or indirectly by increasing endoneurial fluid pressure (as in compartment syndrome). Persistent edema and obliteration of the intraneural microcirculation associated with chronic irritation can lead to fibroblast formation, fibrosis, and endo- or extraneural scarring.
- A direct mechanical damage to the myelin sheath, or the axon itself, thus limiting nerve conduction.

Other Mechanisms

Nerve friction: Continuous or intermittent dynamic compression involves friction of the nerve against adjacent structures. Nerve irritation stimulates fibroblast proliferation in the epineurium, which may lead to the compression of fascicles within the epineurium.

Pressure modifications in anatomical tunnels: At the elbow, the change in the diameter of the ulnar tunnel during

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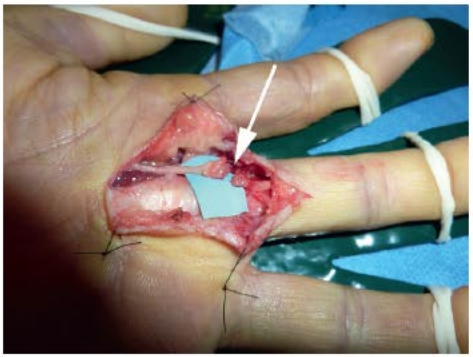

FIGURE 38.1 Section of a digital nerve (white arrow) secondary to knife wound (type V Sunderland lesion).

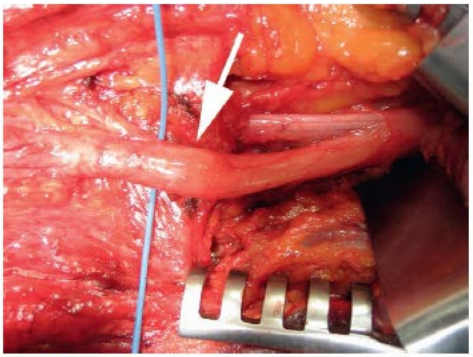

FIGURE 38.2 Neuroma in continuity (white arrow) of the common peroneal nerve after knee dislocation (type IV Sunderland lesion).

elbow flexion is associated with increased pressure. Similarly, pressure changes at the wrist are now well documented. However, the balance between pressures is necessary to maintain adequate intrafascicular circulation.

Nerve constriction (Figure 38.3): This mechanism is rare. The pathophysiology is unclear, but the nerve lesion is similar to complete section, Sunderland stage V (Omura et al., 2001; Umehara et al., 2003; Vigasio & Marcoccio, 2009).

Thermal, electrical, or radiation burns cause ischemia of the nerve and the surrounding tissues and must be considered separately (Moradzadeh et al., 2010).

ANATOMICAL CLASSIFICATION OF NERVE LESIONS

Description of Classifications

The two main classifications of nerve lesions are Seddon's classification of (Seddon, 1942, 1965) in three stages, and Sunderland's in five stages (Sunderland, 1951). These two

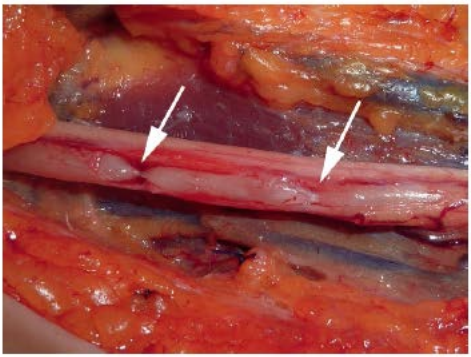

FIGURE 38.3 Hourglass constriction of the nerve (white arrows). This is a type IV Sunderland lesion and should lead to the same therapeutic decision as complete section of the nerve.

classifications are based on the involvement of various parts of the nerve and are perfectly correlated (Table 38.1). We describe their different stages from no nerve injury (Figure 38.4a) to Sunderland VI nerve injury (Figure 38.4g).

Stage I of Sunderland (Neuropraxia of Seddon)

Stage I is a lesion of the myelin sheath or only transient ischemia with no detectable histological changes leading to interruption of nerve conduction (Figure 38.4b). Axonal continuity is respected. There is no nerve fiber degeneration. The excitability of the nerve distal to the lesion is maintained. According to the classification of Seddon, neuropraxia causes complete motor paralysis although some sensory and sympathetic function is preserved (Seddon, 1942). This is because of the differences in nerve susceptibility to compression according to its diameter. Complete recovery is the rule if the cause is removed and when the myelin sheath is totally restored. Neuropraxia of ionic or vascular origin can recover within a few hours or a few weeks. Mechanical neuropraxia can cause a localized myelin degeneration, which requires 1-3 months for regeneration. Typical examples of these lesions are high compression of the radial nerve and positional or "Saturday night palsy" (Spinner, Poliakoff, & Tiel, 2002).

Stage II of Sunderland (Axonotmesis of Seddon)

The injury causes axonal interruption at the site of the lesion, but the endoneurial tube remains intact (Figure 38.4c). Hyperemia is present with endoneurial edema at the lesion. These lesions are caused by prolonged nerve compression or stretching. Axonal degeneration of the distal end occurs (Wallerian degeneration) (Waller, 1850). Recovery depends on the severity and duration of the compression. It has been shown experimentally that compression causes intraneural ischemic lesions that may

TABLE 38.1 Correlations Between Sunderland and Seddon Classifications and Estimation of Recovery with Respect to the Lesion

Sunderland Classification	Seddon Classification	Injury	Clinical Signs	Recovery
Sunderland I	Neuropraxia	Intrafascicular oedema Segmental demyelinization	Paresthesia, partial or total palsy	Full (1 day-3 months)
Sunderland II	Axonotmesis	Axon injured Endoneurial tubes normal	Paresthesia, partial or total palsy	Generally full (1-6 months)
Sunderland III		Endoneurial tubes injured	Paresthesia, dysesthesia, partial or total palsy	Partial (12-24 months)
Sunderland IV		Only epineurium normal (neuroma in continuity)	Hypoesthesia, dysesthesia, total palsy	None without repair
Sunderland V	Neurotmesis	Loss of continuity	Anesthesia, total palsy	None without repair
Sunderland VI		Combination of previous injuries	Paresthesia, partial or total palsy	None or partial

In both classifications, some important prognostic factors are not taken into account.

impair nerve regrowth (Matsumoto, 1983). The regeneration period corresponds to the time of axonal regrowth (from 1 to 2 mm per day) and varies according to the patient's age, associated lesions, the number of axons involved, and the existence of factors limiting nerve regrowth (age, smoking) (Steinberg & Koman, 1991). As the endoneurial tubes guiding regrowth are respected, prognosis of reinnervation is good provided that the distance to reach the endplates is not too great.

Stage III of Sunderland (Axonotmesis of Seddon)

The endoneurial tubes are destroyed (Figure 38.4d). Nerve regeneration is possible with the risk of axonal disorientation because the endoneurial tube guide is lost. Sunderland stage III lesions are often associated with bleeding and intrafascicular edema leading to intrafascicular fibrosis, which may further limit nerve regeneration. They often result from severe compression or traction lesions. Without repair, sensory and motor recovery is always incomplete because of errors of orientation and intraneural fibrosis. Recovery depends on the number of endoneurial tubes damaged.

Stage IV of Sunderland (Axonotmesis of Seddon)

Only the epineurium is intact in this stage (Figure 38.4e). Scarring and fibrosis limit nerve regeneration and may cause the formation of a neuroma in continuity (Figure 38.2). Without repair, functional recovery is poor or nonexistent.

Stage V of Sunderland (Neurotmesis of Seddon)

All parts of the nerve are injured or there is complete nerve division (Figures 38.1 and 38.4f). There can be no functional recovery without repair.

Stage VI of Sunderland

These are lesions of different stages affecting the same nerve with incomplete recovery without repair (Figures 38.4g and 38.5). Repair is difficult in these cases because microsurgical exploration is tricky and does not show all the fascicles and their integrity. If the contused or divided fascicles need trimming before suture, the healthy ones must be spared so as not to aggravate the motor or sensory deficit.

Clinical Correlation of the Sunderland Classification

Clinical assessment of the different stages of nerve lesions depends on the mechanism of injury (stab, compression, stretching). In open injuries and with motor or sensory deficit, diagnosis is easy. Exploration under the microscope is the rule, and the fascicles can be seen after opening the epineurium microsurgically.

Complete division (Sunderland V lesion) is immediately visible, indicating the need for immediate repair. When the nerve appears to be intact, it could be a Sunderland I or II lesion and nerve exploration may well be sufficient. However, in Sunderland III lesions, the nerve is often severely contused with a perfectly visible hematoma, and microscopic exploration becomes essential. In complete paralysis with large visible zones of injury, resection of the damaged zone and an epiperineural microsuture may

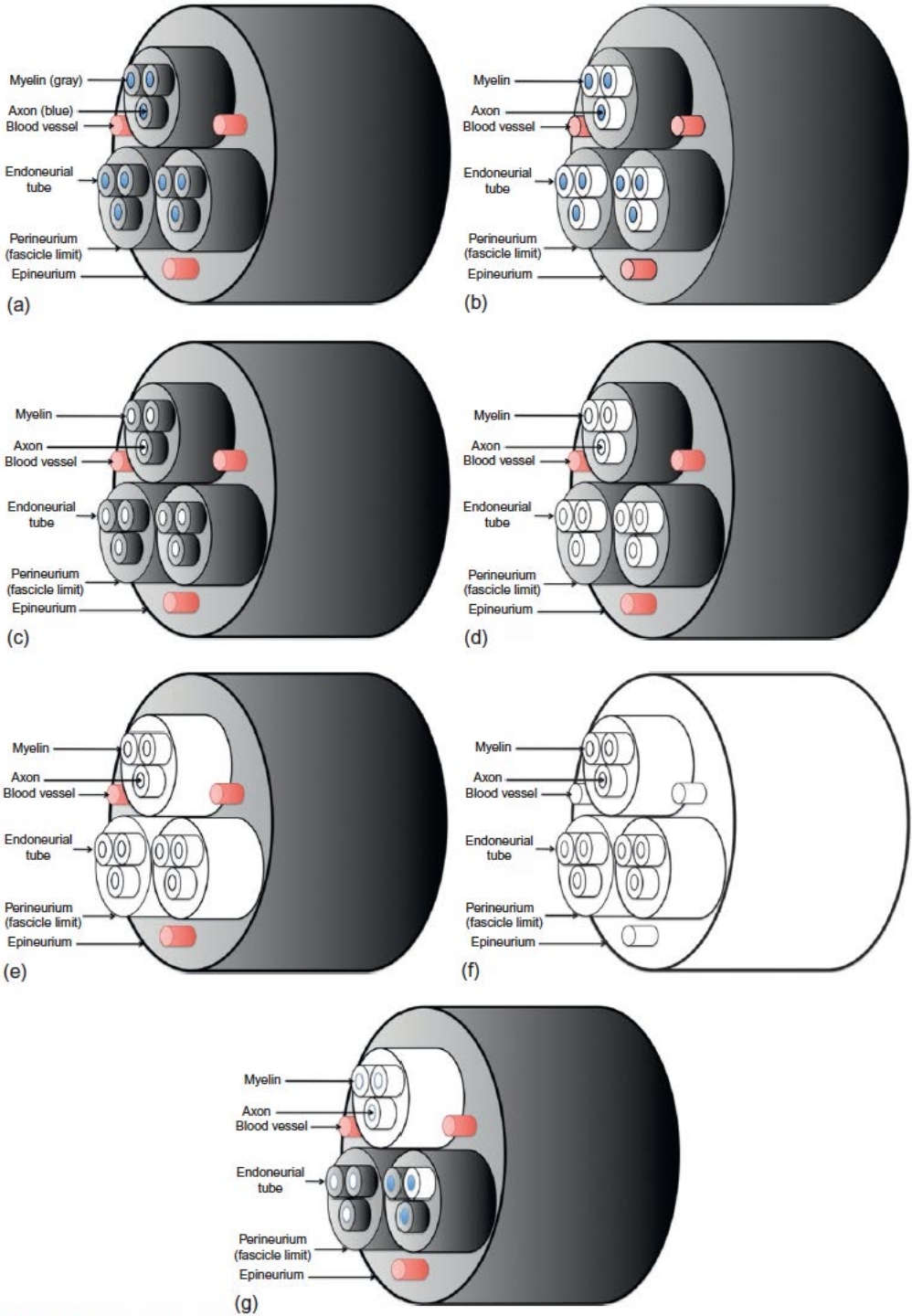

FIGURE 38.4 See legend on opposite page.

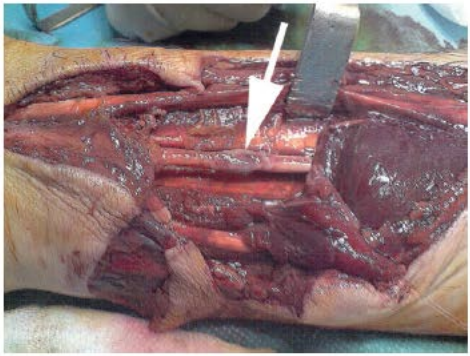

FIGURE 38.5 Median nerve lesion (white arrow) (type VI of Sunderland) secondary to open grossly displaced anterior radius fracture. Recovery is less rapid and complete than it would be in a clean section of the median nerve by a sharp object.

be necessary for any hope of recovery (Bourrel, Ferro, & Lorthioir, 1981). In Sunderland IV lesions, a hematoma is visible, and the nerve is empty on palpation, thus requiring microscopic exploration to show complete transection of the fascicles. Resection of the zone of injury to a healthy zone followed by microsuture is necessary.

In closed nerve injury, clinical diagnosis becomes difficult, and the severity of the initial lesion is diagnosed only by its evolution. If recovery is rapid and complete in the first 3 months, a Sunderland I lesion is diagnosed. If recovery is almost complete but delayed 3-6 months, it is a Sunderland II lesion. If recovery is minimal or nonexistent, it is a type III, IV, or V Sunderland injury. However, waiting too long (more than 6 months) prior to exploration is controversial. Many current series show that better functional recovery is achieved if nerve surgery is performed within 6 months (Bonnard, Anastakis, van Melle, & Narakas, 1999; Goubier & Teboul, 2007; Niver & Ilyas, 2013). Therefore, many authors propose exploration of closed injuries between 3 and 6 months in case of minimal or no recovery (Teboul, Kakkar, Ameur, Beaulieu, & Oberlin, 2004).

Signs of nerve regeneration can detect the type of nerve lesion. A Tinel sign (Tinel, 1915) progressing regularly, 1-2 mm per day, toward the endplates confirms nerve regeneration and evokes a type I or II Sunderland lesion. Persistent irritative syndrome evokes a type III, IV, or V Sunderland lesion.

In lesions affecting several nerves, rapid and progressive recovery of the proximal part of the limb toward the distal end gives a better prognosis and evokes a type I or II lesion. Chaotic and incomplete recovery denotes lesions of types III-VI Sunderland.

The severity of injury can be used to predict the type of nerve injured. Severe trauma (paralysis of the peroneal nerve in knee dislocations, complete brachial plexus palsy in motorcycle accidents) is generally associated with lesion type III, IV, or V.

OTHER NERVE INJURY CLASSIFICATIONS

Yang recently published a classification of brachial plexus nerve lesions in five stages (Yang et al., 2014). It is correlated with an anatomical classification using magnetic resonance imaging (MRI) for evaluating lesions before surgical exploration. MRI is used to locate brachial plexus injuries in relation to the preganglionic nerve root and postganglionic spinal nerve to guide the surgical strategy and make a provisional prognosis.

Lorei distinguished acute and chronic nerve injuries in athletes to guide treatment (Lorei & Hershman, 1993). These classifications are less accurate than Sunderland's, focus on an anatomical region, and are rarely used clinically for prognosis and treatment. They do not take into account the many factors affecting nerve injury.

Some authors have proposed classifications based on clinical symptoms emphasizing their importance (Giannini et al., 2002). These classifications generally consider sensory, motor, or mixed impairment. Some, such as the classification of MacGowan modified by Goldberg for ulnar nerve compression at the elbow, may have a prognostic or therapeutic value in posing surgical indication (Goldberg, Light, & Blair, 1989).

However, the prognostic value of these simple classifications is questionable because it is possible to have a pure myelin injury (Sunderland I) with advanced clinical lesions and complete recovery after surgical release, and low axonal impairment (Sunderland II) with minimal clinical signs and incomplete recovery after surgical release.

Finally, Padua proposed an electromyographic classification reserved for carpal tunnel syndrome (Padua et al., 1997), used to evaluate the severity of the compression of

FIGURE 38.4 Diagram explaining the classification of Sunderland based on damage to myelin, axon, endoneurium, and perineurium. (a) Normal peripheral nerve structure. (b) Type I Sunderland lesion (Seddon neuropraxia). Only the myelin sheath is affected, temporarily blocking nerve conduction. (c) Type II Sunderland lesion (Seddon axonotmesis). This is axonal damage only; endoneurial tubes are preserved, allowing nerve regeneration in good condition. (d) Type III Sunderland lesion (Seddon axonotmesis). The perineurium is intact, but endoneurial tubes are damaged. Nerve regrowth is no longer guided, leading to partial or no recovery, depending on the number of tubes injured. (e) Type IV Sunderland lesion (Seddon axonotmesis). Only the epineurium is intact; all endoneurial tubes are damaged. There is no spontaneous recovery in the absence of microsurgical repair. (f) Type V Sunderland lesion (Seddon neurotmesis). All structures are damaged. There is no spontaneous recovery in the absence of microsurgical repair. (g) Type VI Sunderland lesion. Structural damage is anarchic, leading to partial or no recovery.

the median nerve according to different parameters such as nerve conduction velocity. However, it only gives a retrograde prognosis and cannot determine whether recovery is complete or incomplete after nerve injury. Electromyography is also operator-dependent (Sohn, Whittle, Pezzin, Miao, & Dillingham, 2011).

LIMITS OF CLASSIFICATIONS AND PROPOSAL OF A PROGNOSTIC SCALE

Sunderland's classification is fundamental and perfectly describes anatomical nerve lesions; however, it does not take into account certain preoperative factors affecting functional outcome. Loss of nerve substance, local ischemia, and extensive tissue damage (burns, infection, injury ballistic blast) alter nerve regeneration (Steinberg & Koman, 1991). General factors (age above 50, smoking, and lengthy procedure time) can significantly slow regeneration for the same nerve damage.

We decided to introduce these factors (type of injury, delay of motor nerve repair, level of motor nerve injury, age of patient, perioperative smoking and management in microsurgical unit) into a classification or a scale to predict the final prognosis of nerve lesions (Table 38.2). The more disadvantageous the factors are, the higher the number of points allocated. Currently, it is difficult to determine the number of points for different factors and weigh them against each other. However, this classification has the merit of taking into account the most important factors.

TABLE 38.2 Nerve Injury Severity Scale

Nerve Injury	
– Sunderland I	1
– Sunderland II	2
– Sunderland III	3
– Sunderland IV	4
– Sunderland V/VI	5
– Motor nerve grafting	6
Type of Injury	
– No associated injuries	0
– Crush, avulsion injury	1
– Associated injuries (fracture, ischemia)	2
Delay of Motor Nerve Repair (months)	
– <6 months	0
– 6< delay <12 months	1
– >12 months	2
Level of Motor Nerve Injury	
– Distal injury	0
– Proximal injury	1
Age of Patient (years)	
– age< 20	0
– 20< age< 50	1
– age> 50	2
Perioperative Smoking	
– No	0
– Yes	1
Management in Microsurgical Unit	
– Yes	0
– No	1
Total/15	

Prognostic scale takes into account the anatomical classification of Sunderland and the most important factors affecting the prognosis of nerve recovery. This scale consists of 15 points. The more points, the poorer the prognosis. A nerve injury scoring 1 point has excellent prognosis, whereas a lesion with 15 points has a very poor prognosis.

Age of Patient

It is always difficult to give an age at which the prognosis in terms of motor and sensory recovery is altered. However, recovery is usually slow and partial in patients older than 50 years (al-Ghazal, McKiernan, Khan, & McCann, 1994; Mermans, Franssen, Serroyen, & Van der Hulst, 2012; Mondelli, Padua, & Reale, 2004; Sullivan, 1985). Sensory or motor recovery is better in children than in adults (Mailander, Berger, Schaller, & Ruhe, 1989). It thus seems logical to propose three different age groups for prognosis (less than 20 years, 20-50 years, and more than 50 years) (Mailander et al., 1989).

Mechanism of Injury (Type of Injury)

Severe trauma causes more extensive tissue and nerve damage (Bonnard et al., 1999) and more intraneural structures damaged in case of violent trauma (crush, avulsion, blast), and associated lesions are generally more frequent (vascular injury, tissue surrounding contusion), thus worsening conditions surrounding nerve healing (Lundborg & Danielsen, 1991). The experience gained during World War II has shown the importance of these factors (Woodhall, 1947). Finally, clean-cut injuries give better sensory or motor nerve recovery than crush or avulsion injuries (Gelberman, Urbaniak, Bright, & Levin, 1978; Jaeger, Tsai, & Kleinert, 1981).

Motor Nerve Graft

The need for a nerve graft resulting from loss of nerve substance is a fundamental consideration, especially for motor nerves (Steinberg & Koman, 1991) for which the results are usually worse than after direct nerve suture (Seidel, Koenig, Antoniadis, Richter, & Kretschmer, 2008). This factor is related to the type of nerve damage and operative time. As a matter of fact, if nerve damage and/or operative time

are significant, a nerve graft is often justified. There appears to be no significant difference between direct suture and nerve graft in sensory recovery after nerve repair (Mermans et al., 2012).

Operative Delay (Delay to Repair)

If an intervention is necessary (mainly for Sunderland III-V lesions), the operating time is an important factor in terms of motor nerve recovery. Experimental studies have shown that the shorter the delay between injury and nerve repair, the better the motor recovery (Fu & Gordon, 1995; Jonsson et al., 2013). Most clinical studies show better motor recovery if the repair takes place within 6 months of injury (Goubier & Teboul, 2007; Jivan, Kumar, Wiberg, & Kay, 2009), even though recent publications report satisfactory motor recovery after 12 months (Khalifa, Belkheyar, Diverrez, & Oberlin, 2012). Sensory recovery results following a delay of up to 48 months have been reported (Mailander et al., 1989; Weinzweig et al., 2000).

The Level of Injury (Level of Injury)

Distal lesions have a better prognosis than proximal lesions because the distal ones are located near the innervated muscles (Omer, 1974; Sakellarides, 1962). Nerve regeneration time is shorter with faster access to the endplates, thus limiting their destruction before muscle fiber reinnervation. In addition, the risk of fascicular switch error (fascicular misalignment) is greater in proximal lesions; 66% of median nerve sensory fibers are above the elbow and 94% are at the wrist. For the ulnar nerve, 35% of the fibers are sensory above the elbow and 56% at the wrist (Sunderland, 1978).

Perioperative Smoking

The influence of tobacco on sensorimotor recovery after nerve injury has not been specifically studied in humans. However, tobacco limits scarring and tissue microvascularization, thus altering nerve healing (Abate, Vanni, Pantalone, & Salini, 2013; Lau, Berger, Khullar, & Maa, 2013; Lee, Patel, Biermann, & Dougherty, 2013). This is why we have introduced smoking in our prognostic scale.

Management in a Specialized Unit

The management of the patient with a nerve injury in a specialized microsurgical unit is essential for recovery (Merle, 1994). This has not been clearly demonstrated in the literature; however, this factor is sufficiently obvious to be included in our prognostic scale.

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Chapter 38

Grading of Nerve Injuries

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INTRODUCTION

The type of initial nerve injury significantly alters the functional prognosis with or without nerve repair. Many stages of nerve damage, from simple block of conduction to complete nerve transection, have been described (Seddon, 1942; Sunderland, 1951), resulting in permanent paralysis in the absence of microsurgical repair. The goal of this chapter is to outline the main types of nerve injuries and then describe the main classifications of nerve injuries and evaluate their clinical and prognostic value. We propose a severity scale for nerve damage to supplement the Sunderland classification to which we add certain factors to improve prognosis of nerve recovery.

CLASSIFICATION BY TYPE OF NERVE DAMAGE

There are three main types of nerve injuries: section, stretching, and compression.

Nerve Sections

Nerve sections can be partial or complete, sharp or blunt. They often result from sharp wounds by glass, firearms, or knives (Figure 38.1). The wounds involve the upper limb in 74% of cases and males in 73.5% of cases (Kouyoumdjian, 2006). The wounds are isolated in more than 80% of cases. In lesions of ulnar and median nerves, associated vascular or tendon lesions affect the functional outcome (Kouyoumdjian, 2006).

Nerve Stretching

Stretching can occur with greatly displaced fractures. The extent of the neurological deficit depends on the degree of stretching. During traction, the perineurium elongates, and the axon, which was undulant, straightens out. Intraneural pressure exacerbates lesions of the axon and vascular elements. These lesions can lead to a neuroma in continuity (Alant et al., 2012) (Figure 38.2). If the stretching continues, the axon stretches and tears, as do the perineurium and epineurium. Stretching, such as that after resection of the radial head where cubitus valgus develops, may also be chronic, causing ulnar nerve stretch neuropathy (Mansat, Bonnevialle, Fine, Guiraud, & Testut, 1983).

Nerve Compression

Compression can be extrinsic or intrinsic. Extrinsic compression is more common in the median nerve in the carpal tunnel, ulnar nerve at the elbow, and common peroneal nerve at the knee. Intrinsic compression is caused mainly by nerve tumors.

Two mechanisms of peripheral nerve injury result from compression (Sunderland, 1976):

- Indirect mechanism through vascular compression. Acute or repeated prolonged compression may cause vascular stasis with increased vascular permeability and endoneurial edema formation. This fascicular edema impairs nerve function either directly by modifying the axonal flow or indirectly by increasing endoneurial fluid pressure (as in compartment syndrome). Persistent edema and obliteration of the intraneural microcirculation associated with chronic irritation can lead to fibroblast formation, fibrosis, and endo- or extraneural scarring.
- A direct mechanical damage to the myelin sheath, or the axon itself, thus limiting nerve conduction.

Other Mechanisms

Nerve friction: Continuous or intermittent dynamic compression involves friction of the nerve against adjacent structures. Nerve irritation stimulates fibroblast proliferation in the epineurium, which may lead to the compression of fascicles within the epineurium.

Pressure modifications in anatomical tunnels: At the elbow, the change in the diameter of the ulnar tunnel during

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