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Surgical Treatment of Peripheral Nerve Injury

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1. Introduction

Peripheral nerves were first distinguished from tendons by Herophilus in 300 BC. By meticulous dissection he traced nerves to the spinal cord, demonstrating the continuity of the nervous system⁽¹⁾. In 900 AD, Rhazes made the first clear reference to nerve repair. However, not until 1795 did Cruikshank demonstrate nerve healing and recovery of distal extremity function after repair. In the early 1900s, Cajal pioneered the concept that axons regenerate from neurons and are guided by chemotrophic substances. In 1945, Sunderland promoted microsurgical techniques to improve nerve repair outcomes ⁽¹⁾. Since that time, there have been a number of advances and new concepts in peripheral nerve reconstruction.

Research regarding the molecular biology of nerve injury has expanded the available strategies for improving results. Some of these strategies involve the use of pharmacologic agents, immune system modulators, enhancing factors, and entubulation chambers. A thorough understanding of the basic concepts of nerve injury and repair is necessary to evaluate the controversies surrounding these innovative new modalities ^(1,2).

Treatment of peripheral nerve injuries is considered as challenge procedure. In the past there is no definite line of treatment. A lot of cases with peripheral nerve injuries either missed the diagnosis or found no treatment. With the advent of microscope and development of microsurgical instrument, the era of microsurgical nerve reconstruction has been developed. Two lines of treatment for peripheral nerve injuries have been discussed in the literatures and specialized books. The conservative treatment is one line and it was widely used in the past. This line of treatment is mainly described for the non surgical causes of peripheral nerve injuries. There are numerous causes of non surgical peripheral nerve injuries as, metabolic, collagen diseases, malignancies, endogenous or exogenous toxins, thermal, chemical, or nutritional. Surgical causes of peripheral nerve injuries include acute and chronic causes. The acute surgical causes of peripheral nerve injuries mostly due to simple bone fracture, open fracture, cut wound, traction injuries, firearm injuries (either thermal effect or direct injury), crushed injuries, or animal bite injuries. The chronic surgical causes of peripheral nerve injuries include either acute nerve injuries with formation of painful neuroma or entrapment neuropathies.

1.1 Peripheral nerve injury classification

Classification of peripheral nerve injury assists in prognosis and determination of treatment strategy. Classification of nerve injury was described by Seddon in 1943⁽³⁾. The classification was based on Neuropraxia

- The lowest degree of nerve injury in which the nerve remains intact but signaling ability is damaged.
- There is physiological loss of nerve conduction. The nerve is temporarily blocked.
- There are sensory-motor problems distal to the site of injury.
- The endoneurium, perineurium, and the epineurium are intact.
- There is no wallerian degeneration.
- In neurapraxia, conduction is intact in the distal segment and proximal segment, but no conduction occurs across the area of injury.
- Common causes are Saturday night palsy, honey moon palsy, simple fractures, mild degree of tourniquet paralysis.
- Recovery of nerve conduction deficit is full, and requires days to weeks.
- EMG shows lack of fibrillation potentials (FP) and positive sharp waves.

1.1.1 Axonotmesis

It involves loss of the relative continuity of the axon and its covering of myelin, but preservation of the connective tissue framework of the nerve (the encapsulating tissue, the epineurium and perineurium are preserved).

- Wallerian degeneration occurs below to the site of injury.
- There are sensory and motor deficits distal to the site of lesion.
- There is not nerve conduction distal to the site of injury (3 to 4 days after injury).
- Traction injuries, simple fracture with high energy trauma, severe degree of tourniquet paralysis, are the most common causes.
- EMG shows fibrillation potentials (FP) , and positive sharp waves (2 to 3 weeks postinjury).
- Axonal regeneration occurs and recovery is possible without surgical treatment. Sometimes surgical intervention because of scar tissue formation is required.

1.1.2 Neurotmesis

Neurotmesis is a total severance or disruption of the entire nerve fiber. A peripheral nerve fiber contains an axon (Or long dendrite), myelin sheath (if existence), their schwann cells, and the endoneurium. Neurotmesis may be partial or complete.

Other characteristics:

- Wallerian degeneration occurs below to the site of injury.
- There is connective tissue lesion that may be partial or complete.
- Sensory-motor problems and autonomic function defect are severe.
- There is not nerve conduction distal to the site of injury (3 to 4 days after lesion).
- Cut wounds, crushed injuries, most of open fractures with nerve injuries, iatrogenic nerve injury, gunshot injury, firearm injury are the most common causes.

- EMG and NCV findings are as axonotmesis.
- Because of lack of nerve repair, surgical intervention is necessary.

1.1.3 Sunderland's ⁽¹⁾ classification

In 1951, Sunderland⁽¹⁾ expanded Seddon's classification to five degrees of peripheral nerve injury:

First-degree (Class 1): Seddon's neuropraxia and first-degree are the same.

Second-degree (Class 2): Seddon's axonotmesis and second-degree are the same.

Third-degree (Class 3): Sunderland's third-degree is a nerve fiber interruption. In third-degree injury, there is a lesion of the endoneurium, but the epineurium and perineurium remain intact. Recovery from a third-degree injury is possible, but surgical intervention may be required.

Fourth-degree (Class 3): In fourth-degree injury, only the epineurium remains intact. In this case, surgical repair is required

Fifth-degree (Class 3): Fifth-degree lesion is a complete transection of the peripheral nerve. Recovery is not possible without an appropriate surgical treatment.

Although Sunderland's classification provides a concise and anatomic description of nerve injury, the clinical utility of this system is debatable. Many injuries cannot be classified into a single grade. Mixed nerve injuries, in which all fibers are affected but to varying degrees, are common among peripheral nerve injuries. Furthermore, although Sunderland's classification accurately describes the pathoanatomy of nerve injury, it is seldom possible to accurately subclassify an axonotmetic nerve injury on the basis of preoperative clinical and electromyographic data. The subtype is usually discernible only by histologic examination of the injured nerve ⁽²⁾.

Non Degenerative

1. neuropraxia

Degenerative nerve injury

2. axonotmesis
3. neurotmesis

1.2 Manifestations of peripheral nerve injury

1.2.1 Motor

1. Muscle wasting, muscle atrophy and even muscle paralysis
2. Loss of reflex according to the affected muscle e.g. loss of biceps reflex in case of musculocutaneous nerve injury, loss of triceps reflex in case of radial nerve injury, loss of knee reflex in case of femoral nerve injury and loss of ankle reflex in case of posterior nerve injury.
3. Joint contracture and fixed deformity, this can be happened in longstanding nerve injury due to muscle imbalance.

1.2.2 Sensory

1. Loss of superficial sensation including pain (anesthesia, hyposthesia, parathesia), touch, temperature.
2. Loss of deep sensation including sense of position, sense of movement and stereognosis (identification of object by touch with closed eyes).

1.2.3 Vasomotor

The skin supplied by the injured nerve become pale and dry (anhidrosis) with crust formation.

1.3 Indications for nerve surgery

1. Cases with neuropraxia that failed to recover within the first 3 months.
2. Cases with definite nerve injuries as, open fracture, crushed injuries, firearm and gunshot injuries, cut wounds, iatrogenic nerve injuries, postreduction injuries, animal bite injuries.
3. Chronic nerve lesions with painful neuroma.
4. Entrapment neuropathies.

There is a lot of debate for surgical treatment of nerve injuries especially those associated with simple fracture. One school of surgeons prefers treatment of fracture and wait for months to give chance for nerve to be recovered spontaneously. The other school advises to explore the nerve and treat both the fracture and injured nerve simultaneously. Actually I prefer early surgical exploration and surgical treatment of the fracture at the same time. The chance of simultaneously nerve recovery is decreased with surgical maneuver around the injured nerve, the time elapsed between nerve injury and exploration is sufficient enough for nerve regeneration after surgical treatment even after nerve repair, to explore the injured nerve acutely passed the chance for nerve graft which is more complex of course than neurolysis or nerve repair.

1.4 Timing of nerve surgery

It has been the time-honored policy to advise primary suture when possible. This recommendation is logical when one considers what happens to the distal end of the nerve, motor end plates, sensory nerve endings, muscles, joints, and other tissues of the denervated extremity. The controversy concerning whether primary or secondary nerve repair is better is unresolved. Primary repair done in the first 6 to 8 hours or delayed primary repair done in the first 7 to 18 days is appropriate when the injury is caused by a sharp object, the wound is clean, and there are no other major complicating injuries. Ideally, such repairs should be performed by an experienced surgeon in an institution where adequate equipment and personnel are available. The development of magnification devices, new instruments, and new techniques and the modification of a variety of small instruments for use in nerve surgery have improved the technique of early repair. Primary repair should shorten the time of denervation of the end organs, and fascicular alignment should be improved because minimal excision of the nerve ends is required. For several reasons a primary peripheral nerve repair is favourable. Technically, a primary repair of a sharp injury is easier. The rotation of the nerve segment can be easily judged since the epineurial

blood vessels can be identified on the surface of the nerve trunk. The nerve stumps can also easily be cleaned, i.e. excision of nonviable tissue, to help coaptation ⁽⁴⁾.

When the diagnosis of division of a peripheral nerve has been made, if conditions are suitable and repair is indicated, one should not delay repair in anticipation of spontaneous regeneration. Only if the patient's life or limb is seriously endangered should the operation be long postponed. However, an important part of nerve repair is to judge the extent of necrotic tissue in specific injuries. In severe cases, caused by for example a gun shot wound, there may be widespread injury to the peripheral nerve components. It can be difficult in the fresh case to evaluate how much of the nerve ends needs to be resected. In such injuries it is of extreme importance to tidy up the wound and remove all other necrotic tissues to avoid infection. In some cases with severe nerve injuries it may be advisable to delay the nerve reconstruction until all other tissues, such as muscle, have healed properly. However, this is not the case in a sharp transection injury where a primary suture is the best alternative. A fracture is not a contraindication for operation. Operation before the fracture becomes united may be advantageous for two reasons: (1) If bone shortening is necessary, resection of an ununited or partially united fracture is a much less formidable procedure than resection of a fully united bone; and (2) restriction of joint motion is minimal if the nerve is repaired soon after the injury; later, motion would be more limited, perhaps so severely as to prevent flexing the joint enough to overcome a gap between the nerve ends.

Another interesting issue is exploration of the radial nerve in patients with radial nerve dysfunction in connection with a humeral shaft fracture. The radial nerve can in such cases be injured or even ruptured. If the humeral shaft fracture is to be repaired with plates and screws one may consider exploring the dysfunctional radial nerve at the same time. In case one is convinced that the radial nerve is severely lacerated an early repair should be considered based on the neurobiological alterations and impaired axonal outgrowth with time described earlier. However, one can not as a general rule recommend a generous exploration of all radial nerves that may have some dysfunction after humeral shaft fracture ⁽⁵⁾.

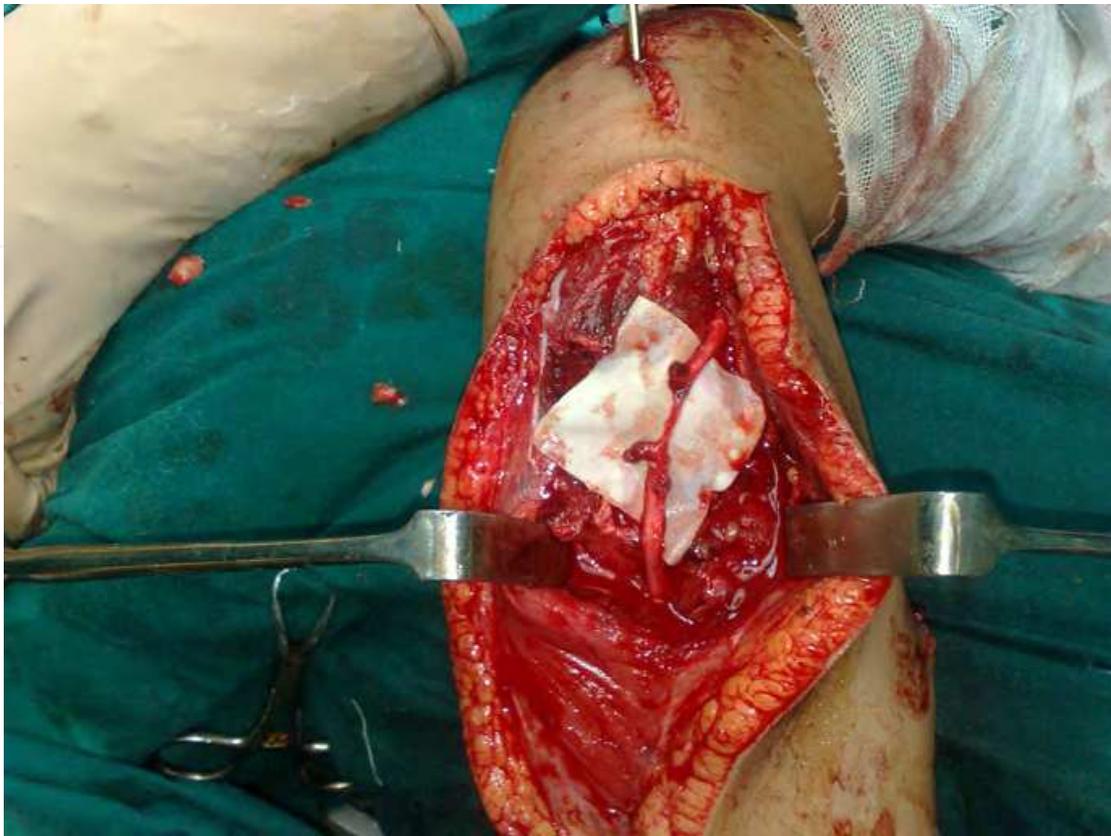
1.5 Author preferred method

A radial nerve injury associated with a humeral shaft fracture is an important injury pattern among trauma patients ⁽⁶⁾. It is the most common peripheral nerve injury associated with fracture of long bones ⁽⁷⁻¹⁰⁾. As our understanding of the pathoanatomy of the humerus and surrounding neurovascular structures has evolved, surgeons have adapted their strategies to improve outcome. There are differences in opinion regarding the treatment of choice. Early exploration of the radial nerve claims a variety of advantages. It is technically easier and safer than the delayed procedure. Direct examination of the nerve clarifies the diagnosis and the extent of the lesion. Early stabilization of the fracture reduces the chance of the nerve being enveloped by scar tissue and callus. Reduction of the open fracture helps lessen the risk of further neural damage from mobile bone ends. Shortening of the humerus to facilitate nerve repair is better done before healing of the fracture is complete ⁽¹¹⁻¹³⁾. However, opponents of early exploration have observed high rate of spontaneous recovery and have advised a policy of expectancy, ⁽¹⁴⁻²¹⁾ believing that this approach mitigates an unnecessary complications attendant upon exploration. Thickening of the neurilemmal sheath during waiting helps to define the extent of nerve damage and facilitates repair. It is easier to treat the nerve when the fracture is healed. Most of these papers describe small

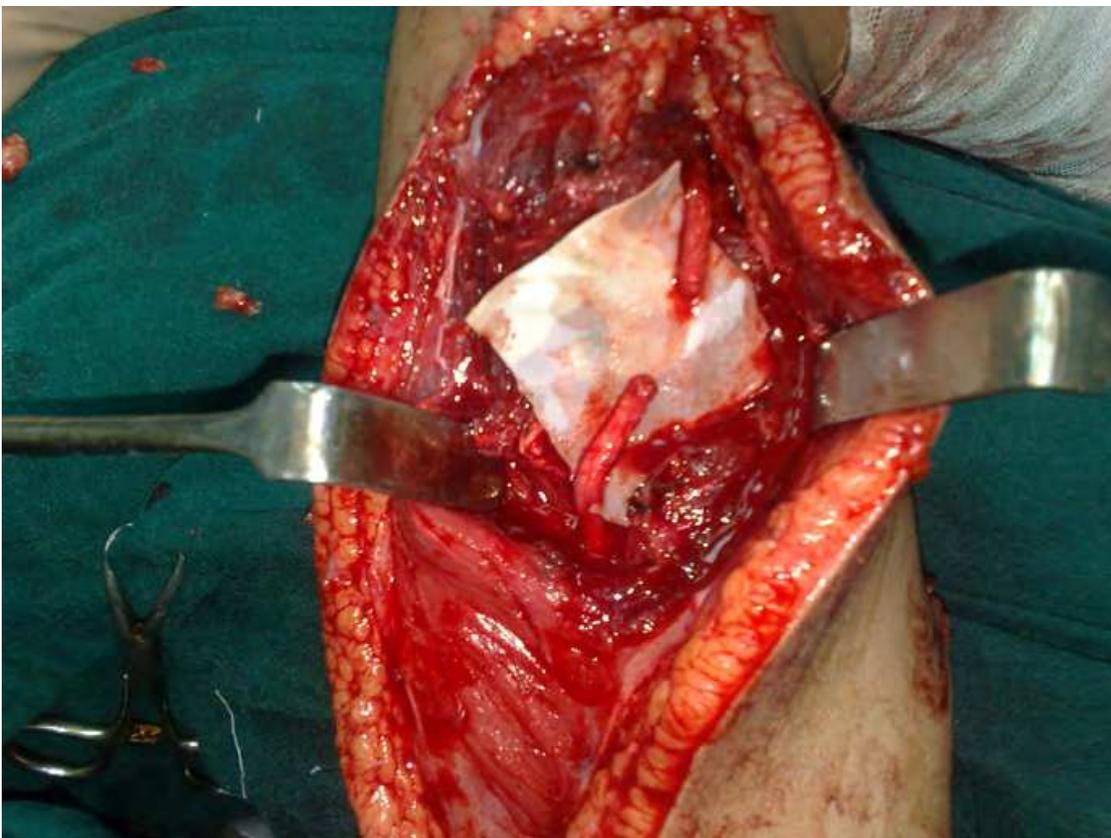
numbers of patients and all are uncontrolled retrospective case series. Although treatment for this injury pattern is a controversial subject among upper-extremity surgeons, certain principles of management need to be applied in all cases. We limited our analysis to post-humeral fracture radial nerve palsies, which were operated due to the presence of neurological deficits after the fracture. We recorded the type of fracture, treatment used to achieve bone healing, surgical approach, and type of radial nerve surgery. Between April 2001 and April 2007, 36 patients with fractures of the shaft of the humerus with palsy of the radial nerve were treated by early exploration of the radial nerve and internal fixation by narrow DCP plate. Twenty four were male and 14 female, with a mean age of 30.3 years (8-53 years). The most common cause of injury was motor car accident in 25 patients, falling from height in 5 patients, fire arm injury in 4 patients, and machine injury in 2 cases. A lesion of the radial nerve had occurred at the time of injury in 27 patients, 7 of these patients had open fracture, and post reduction injury occurred in 9 cases. The fracture patterns were varied. The most common pattern of fracture was transverse pattern involved the distal third occurred in 16 cases, oblique fracture in the distal third (Holstein-Lewis fracture) in 7 cases, spiral fracture involved the middle third in 8 cases, transverse fracture involved the middle third in 3 cases and fracture involved the junction of middle and upper third of the humerus in 2 cases. Twelve patients had surgery on the day of injury and the other 26 at a mean of 8 days later (3 to 14). The mean follow up was 28 months (9 to 72). The anesthesia was general, the position was laterally and the approach was posterior approach. Exploration of the radial nerve demonstrated compression at the lateral intermuscular septum in 19 cases, entrapment in the fracture site in 9 cases and loss of its continuity in 8 cases. Neurolysis was required in 20 cases, epineurorrhaphy in 9 cases, nerve grafts in five, and first-intention tendon transfer in two. Plate fixation was generally used for fixation. Results of nerve surgery were assessed used the MRC (Medical Research Council) at a mean follow-up of 8.2 years. Outcome was rated good to excellent in 28 patients, fair in one and poor (failure) in three. First-intention tendon transfers were performed in two patients and two patients were lost to follow-up. Mean delay to recovery was five months after neurolysis, eight months after nerve repair and fifteen months after nerve grafts. The fracture was united in all cases. The mean time of union was 5 months.

2. The technique of nerve repair

The anaesthesia have to be tailored according to the site, the affected nerve, the age and the need of the patient. The operation is performed under tourniquet, and wide exposure is used. Dissection is carried out from normal to abnormal tissue, with an attempt not to disturb the local blood supply (Fig. 1a). The procedure of repairing a nerve trunk can be divided into four steps. Initially the nerve ends are prepared to get a viable nerve end without necrotic tissue (preparation) (Fig. 1b). The nerve ends are handled with care using microsurgical instruments. A pair of sharp micro scissors or a surgical blade can be used to remove the necrotic part of the nerve ends. The extent of resection can be difficult to judge if there is a laceration or a contusion by for example a gun shot. After the nerve ends are prepared, they should be approximated keeping in mind the importance of adjusting the length of the gap and the tension of the nerve segments (approximation). During the approximation the nerve ends can be slightly mobilised by dissection but one should avoid extensive intrafascicular dissection. The nerve ends are coaptated. The nerve repair is maintained by stitches (maintenance) (Fig. 1c); 9-0 or 10-0 nylon (sometimes thicker suture materials can be suitable in specific cases) is inserted into the epineurium.



(a)



(b)



(c)

Fig. 1. Male patient 9 years old subjected to firearm injury caused open fracture humerus with radial nerve injury, (a) Intraoperative photo showing laceration of both ends of the radial nerve. (b) Intraoperative photo after preparation of both stumps. (c) Intraoperative photo after maintenance of the repair by 10/0 Nylon.

Thus, interrupted epineurial sutures maintain the repair. In a digital nerve it may be enough with three 9-0 sutures, while in a larger ulnar or median nerve several interrupted sutures are applied, sometimes with thicker suture material. When the sutures are placed one should try to avoid malrotation of the nerve ends. Identification of the longitudinal intraneural blood vessels may help in this. In specific cases it is possible to identify individual fascicular groups for attachment (group fascicular nerve repair), predominantly at a distal level where fascicles with specific targets are well defined. The ulnar nerve at wrist level is one nerve where such a repair technique can be used since it contains two separate motor and sensory components. All nerve repairs are explored by the use of surgical loupe (magnification $\times 3.5$), but a microscope is routinely used during repair. Nerve suturing is the key of success for nerve surgery. The aim is to convert the distal nerve stump into patent tube to receive the growth cone of the axons. The regenerating nerve axons have to cross smoothly through the site of nerve anastomosis (neurorrhaphy).

3. Neurorrhaphy (direct nerve repair)

Historically, it was thought best to wait 3 weeks before repair to allow the conclusion of wallerian degeneration. However, Mackinnon ⁽²²⁾ and other authors have shown that immediate primary repair is associated with better results. Prerequisites are a clean wound, good vascular supply, no crush component of the injury, and adequate soft-tissue coverage. Skeletal stability is paramount, and there should be minimal tension on the nerve repair. Although the classic technique of neurorrhaphy is devoid of tension, Hentz et al ⁽²³⁾ studied a primate model and showed that a direct repair under modest tension actually does better than a tension-free nerve graft over the same regenerating distance. With the advent of microsurgical instrumentation and technique, attempts at group fascicular repair, rather than simple epineurial coaptation, have been attempted. Proponents argue that group fascicular repair is better because axonal realignment is more accurate with this technique. However, others have shown that there is no functional difference in outcome between epineurial and group fascicular repair. Furthermore, group fascicular repair has the potential disadvantage of increased scarring and damage to the blood supply as a result of the additional dissection. Lundborg et al ⁽²⁴⁾ concluded that although this technique purportedly ensures correct orientation of regenerating axons, there is little evidence that it is superior to the less exact but simpler epineurial repair.

Monofilament nylon suture is the preferred suture type because of its ease of use and minimal foreign body reactivity. Using a cadaveric median nerve model, Giddins et al ⁽²⁵⁾ demonstrated that 10-0 nylon failed under tension; that 9-0 nylon withstood the greatest distractive force before repair gapping; and that 8-0 nylon had a tendency to pull out of the repaired nerve ending.

A number of techniques are available to facilitate fascicular matching. Visual alignment may be aided by topographic sketches of both cut ends. With this method, it can be determined which fascicular group of the proximal stump corresponds to the fascicular group of the distal stump. Electrical stimulation can be used to identify sensory fascicles in the proximal stump in an awake patient, but because wallerian degeneration of the distal axon begins within 2 to 4 days after transection, motor fascicles can be identified reliably only by direct

nerve stimulation in fresh injuries. Nerve ends can also be stained to differentiate between motor and sensory axons. Initially, staining was too time-consuming to be clinically useful, but recent advances have been made. Gu et al ⁽²⁶⁾ reported on a 30-minute technique for blue- SAb staining of sensory fascicles and showed that staining does not affect the growth and metabolism of neurons. Sanger et al ⁽²⁷⁾ have reported on carbonic anhydrase staining and cholinesterase staining of sensory and motor neurons, respectively. Carbonic anhydrase staining took 12 minutes, and cholinesterase staining took 1 hour. The stain persisted for 35 days in the proximal stump and 9 days in the distal stump. These techniques may aid in both immediate and delayed primary nerve repair. Direct nerve repair (neurorrhaphy) can be performed in different ways:

1. Epineurorrhaphy this means the stitches passed through the epineurium. This is the most common types of nerve suturing. Its main drawback is malalignment of the different nerve fascicles. The incidence of mismatching is higher than the other types of nerve suturing. Its advantages are less time consuming, avoid intraneural dissection and intraneural fibrosis. There is significant difference in the results when compared by other types of nerve suturing.
2. Perineurorrhaphy, this is means the suturing is passed through the perineurium. It decreases the incidence of the mismatching of nerve fascicles. It gives good nerve alignment. It is used in cases with deficient nerve sheath (epineurium).
3. Epiperineurorrhaphy, the needle passed through both the epineurium and perineurium. It gives strong coaptation. It provides better nerve alignment. It can be widely used in frersh cases (primary nerve repair).

3.1 Author preferred method

There is no significant difference in the result of nerve repair when the principles of neuromicsurgery have been taken. The above 3 types of nerve repair can be used in one nerve repair. I prefer to do epineurorrhaphy in primary nerve repair, specially if the epineurium is intact (Fig 2). In cases with injured epineurium and the nerve is divided into group of fascicles, perineurorrhaphy is the good solution. In cases with delayed primary or even secondary nerve repair, the use of epiperineurorrhaphy is the method of choice to have good suturing strength because of the fragility of nerve sheath.

4. Specific nerve repair

4.1 Radial nerve repair

The radial nerve arises from the posterior cord of the brachial plexus. It receives contributions from C5-8 spinal roots. It runs medial to the axillary artery. At the level of the coracobrachialis, it courses posteriorly to lie in the spiral groove of the humerus. In the lower arm, it pierces the lateral intermuscular septum to run between the brachialis and the brachioradialis. It divides 2 cm distal to the elbow into a superficial sensory branch and a deep motor branch, the PIN. The radial nerve gives off branches to the extensor carpi radialis longus and brevis, brachioradialis, and anconeus before giving off the PIN branch. The PIN continues on between the superficial and deep head of the supinator muscle, to exit on the dorsal forearm. After it emerges from the distal border of the supinator, the PIN sends branches to the extensor digitorum communis, extensor carpi ulnaris, extensor digiti

quinti, extensor pollicis longus and brevis, and extensor indicis proprius in descending order, although there may be considerable variation.

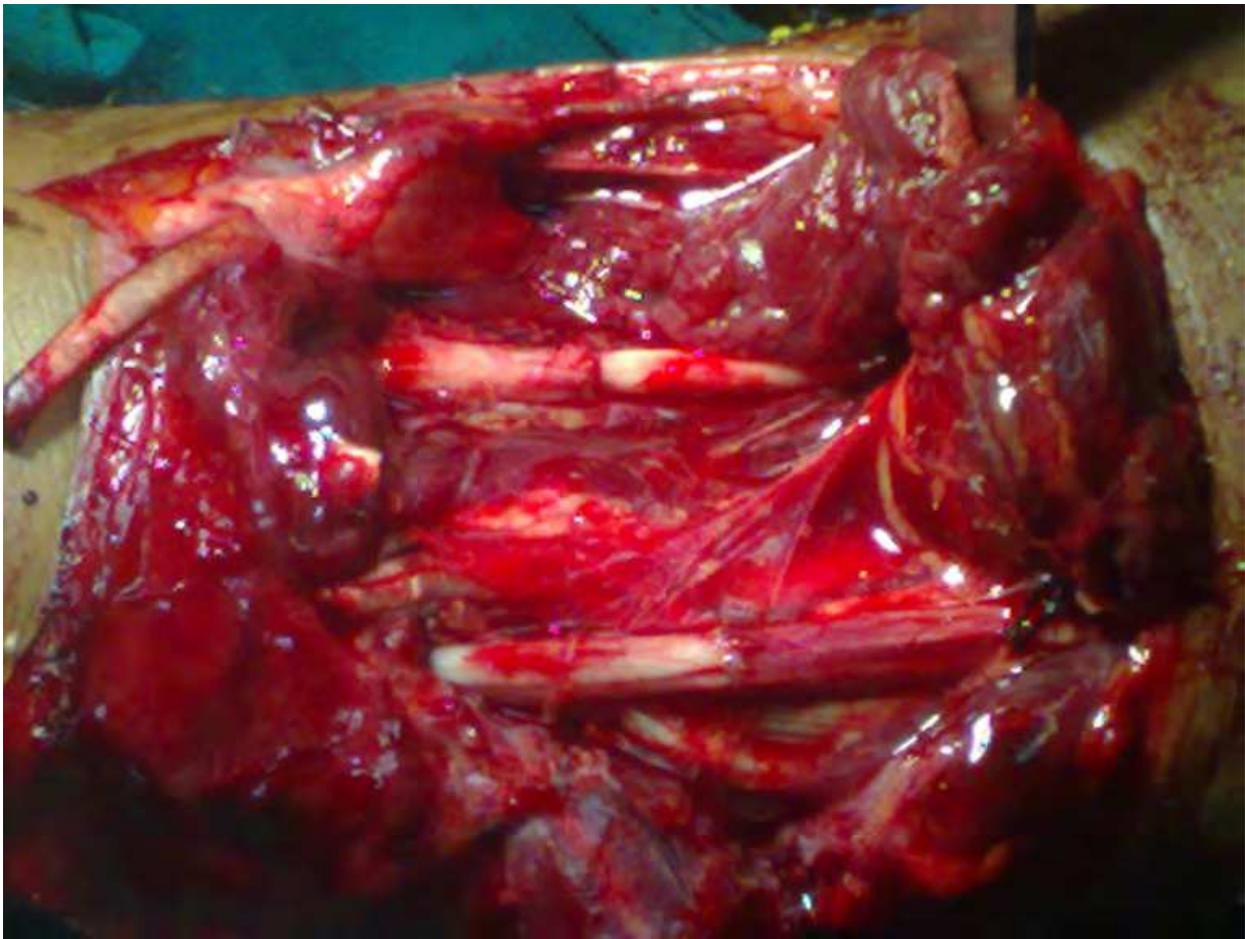


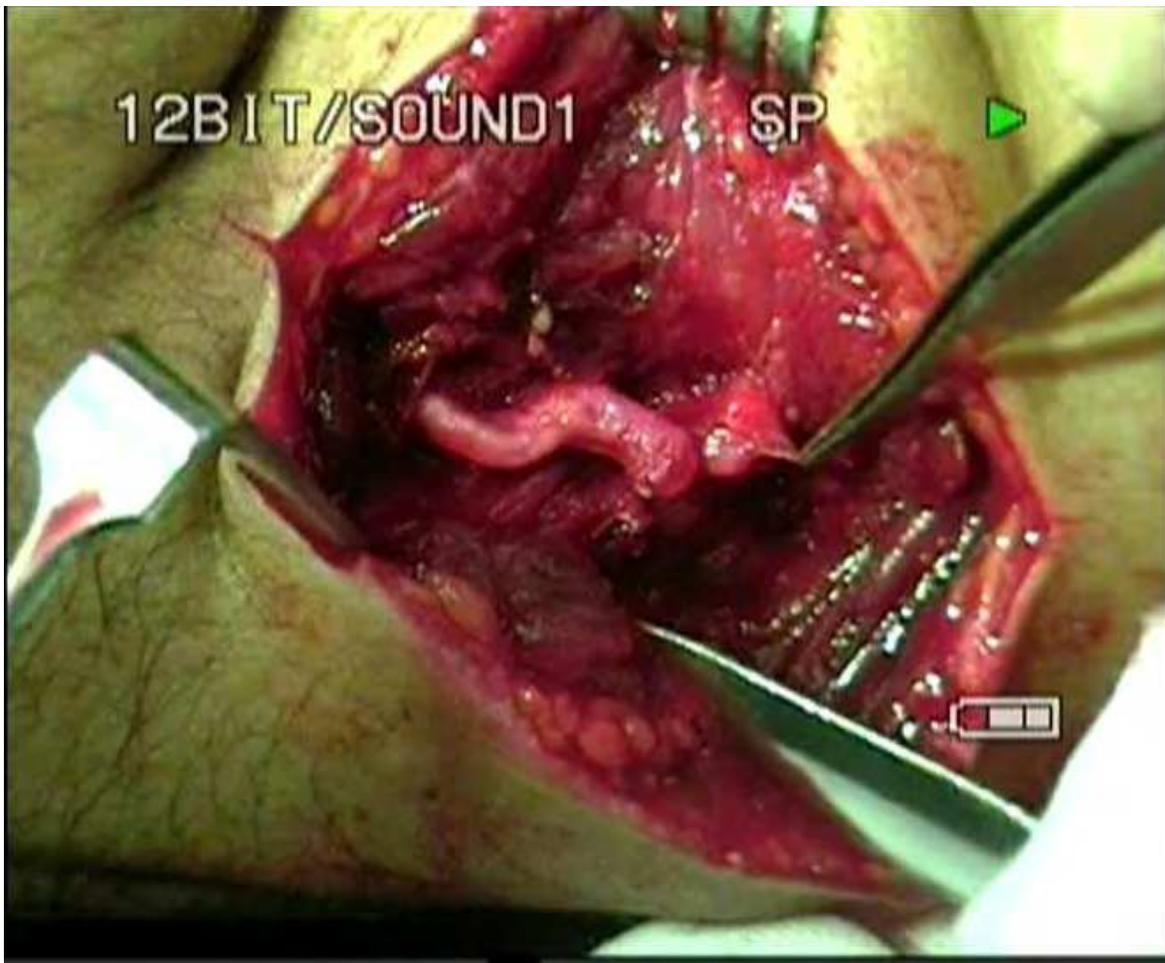
Fig. 2. Intraoperative photo showing epi-neuroorrhaphy of both median and ulnar nerves at the musculotendinous junction of the forearm with cut of all the flexor tendons.

Radial nerve is the most common peripheral nerve to be injured. It travels for long distance so different levels of injuries are well known. It may vulnerable for injury in the axilla, along the arm, spiral groove posteriorly, lateral intermuscular septum laterally, between brachioradialis and brachialis anteriorly, opposite to the radial head laterally, in the proximal forearm posteriorly and finally its sensory part in the forearm anteriorly. In this study, we present 62 patients who had radial nerve injuries. Forty eight were male and 14 were females. The average age of the patient was 26 years (ranged between 6-42 years). The right side was involved in 37 cases while 25 cases had left radial nerve injury. Motor car accident was responsible for the majority of lesion (32 patients), fire arm injuries was the next common cause of injury (15 patients), cut wound caused injury of 8 cases, while machine injury of the radial nerve occurred in 7 cases. Radial nerve injuries associated with fracture humerus occurred in 42 patients while isolated radial nerve injuries happened in 20 cases. The indication for radial nerve exploration were cut wound, firearm injuries, open fracture, postreduction injuries and cases with transverse fracture of the distal humerus. The mean time elapsed between injury and exploration was 12 hours (ranged between 2- 76 hours). The average surgical time 2.5 hours (ranged between 2-6 hours). The surgical procedures

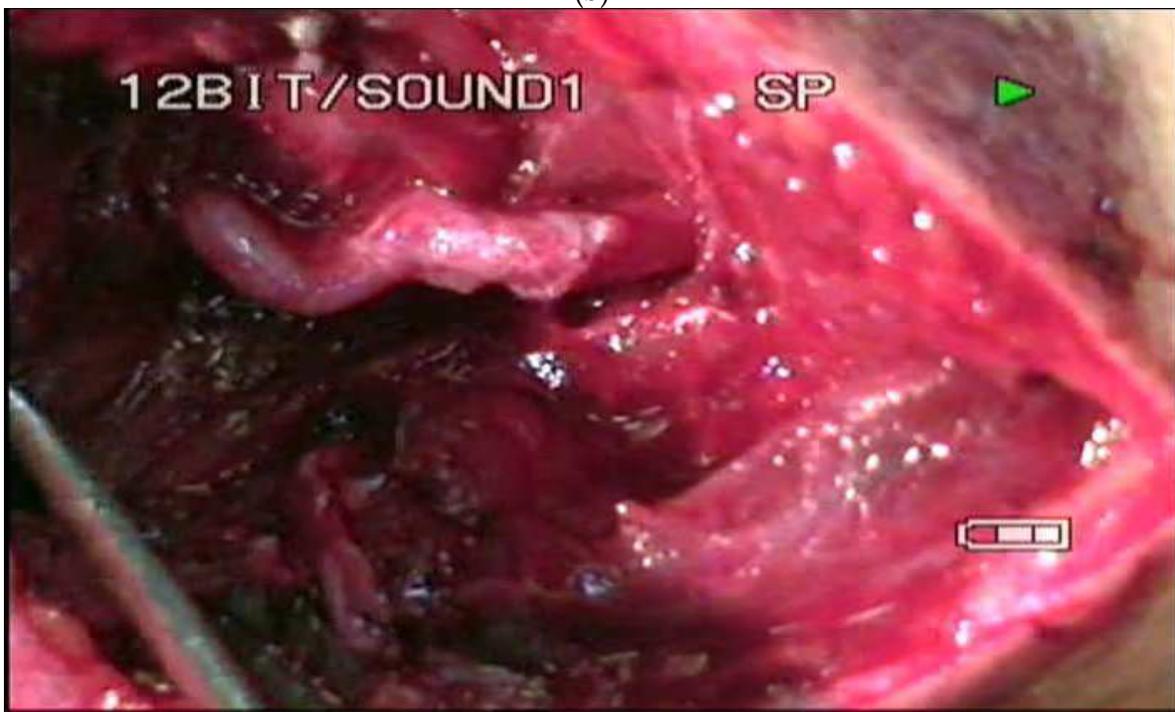
were neurolysis in 16 patients, epineurorrhaphy in 33 patients and nerve graft in 13 patients. We used nylon 10/0 for suturing under microscope in all cases of repair or graft. We used Medical research council grading for evaluation of wrist and finger extensors and sensation along the radial nerve sensory supply. Motor recovery of wrist and finger extensors was M5 in 60 patients (97%), M4 in one case and M3 in one patient. Sensory recovery was S3+ in 52 Patients, S3 in 7 patients and S2+ in 3 patients. The hand grip, hand pinch were the same as the non injured side. All the patients returned to their original activities. The time of recovery after surgery depend on the level of injury, the age of the patient and the type of surgical treatment. The average time of recovery was 7 month (ranged from 4 to 24 month). The average follow up was 12 years (Fig. 3,4).



(a)



(b)



(c)



(d)

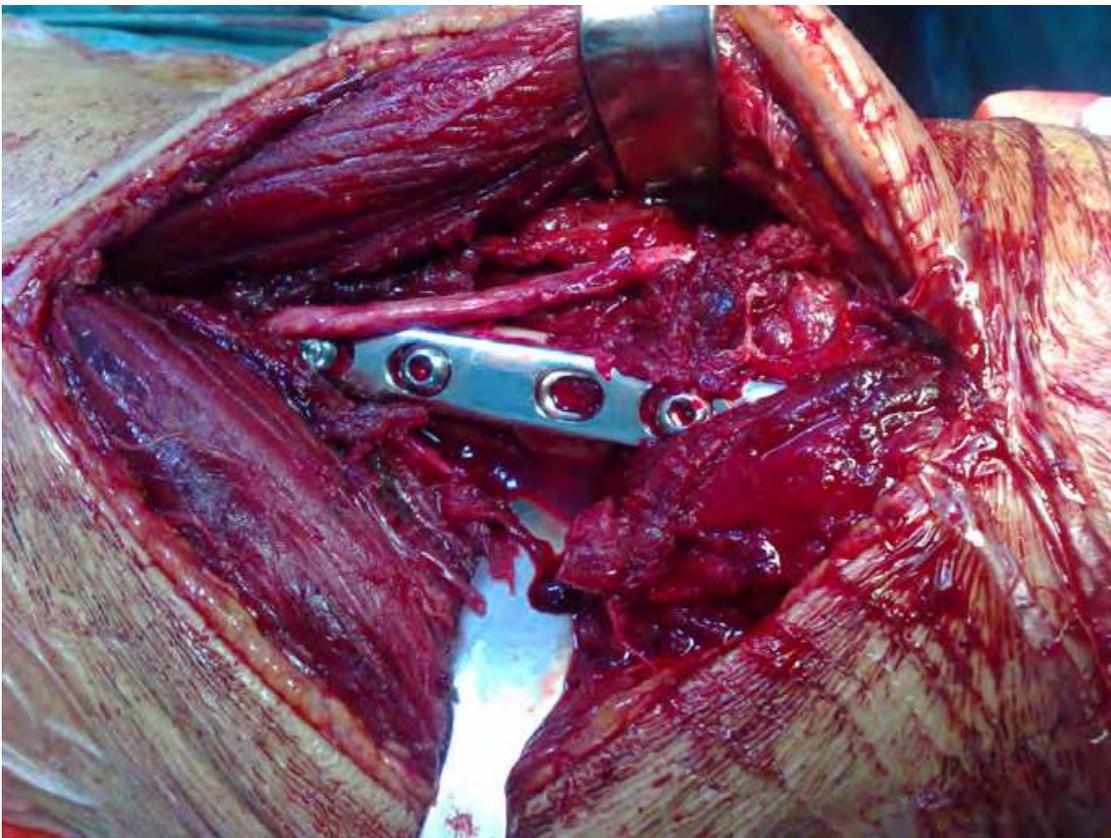


(e)

Fig. 3. Male patient 28 years old subjected to stab wound at distal left arm caused radial nerve injury, (a) Site of the stab wound with stitches. (b) Intraoperative photo showing complete transaction of the radial nerve. (c) Micro-epi-neurorrhaphy of the nerve. (d) 12 months of follow up with complete wrist extension. (e) 12 months of follow up with complete finger extension.



(a)



(b)



(c)



(d)

Fig. 4. Male patient 40 years old sustained motor car accident presented by fracture humerus with radial nerve injury, (a) early exploration revealed complete transection of the radial nerve. (b) Micro-epi-neurorrhaphy after fixation of the humerus by plate and screws. (c) 9 months of follow up with complete wrist extension. (d) 9 months of follow up with complete finger extension.

5. Median nerve

5.1 Anatomy

The median nerve arises from the medial and lateral cords of the brachial plexus. It contains the nerve root fibers from C6-T1. It provides the motor supply to the pronator teres, the flexor digitorum sublimus, the palmaris longus, the flexor carpi radialis, the thenar muscles, and the radial two lumbricals. Its anterior interosseous branch supplies the flexor pollicis longus, the pronator quadratus, and the flexor digitorum profundus to the index and middle fingers. Its sensory distribution includes the palmar surface of the thumb, index, middle, and radial half of the ring finger. It lies lateral to the axillary artery, but then crosses medial to it at the level of the coracobrachialis. At the elbow, the median nerve travels behind the bicipital aponeurosis but in front of the brachialis. It enters the forearm between the two heads of the pronator teres and is adherent to the undersurface of the flexor digitorum sublimus muscle until it becomes superficial, 5 cm proximal to the wrist. It then passes underneath the carpal transverse ligament, giving off the recurrent motor branch and sensory branches to the thumb and fingers.

5.2 Above elbow

Median nerve injury is uncommon above the elbow. We present 12 patients who have median nerve injury above the elbow. Three patients had broken glass injury, 4 cases had firearm injuries and 5 cases associated with fractures around elbow. Exploration was performed for 10 cases while two patients recovered spontaneously. Primary repair was performed for 8 cases while 2 cases were repaired within one week of the trauma. The mean follow up was 7 years. The time of recovery was ranged from 6-12 month postoperatively. We used both Medical Research Council Grading and Noaman scoring system for evaluation of wrist and finger flexors, intrinsic muscles supplied by the median nerve, hand grip, lateral key pinch, opposition, hand deformity, and sensation. All patients had wrist and finger flexors M5, good opposition, normal hand grip, lateral key pinch and S3.

5.3 Below elbow

The most common cause of median nerve injury at our locality is broken glass; the next cause is firearm injuries while motor car accident with crushed forearm is not uncommon. We treated 40 patients who had median nerve injuries below elbow from 1999 to 2010. Twenty six were male and 14 were females. Twenty two patients presented by cut wound caused by broken glass associated with muscles and musculotendinous injuries. Primary repair was performed in all cases. Time elapsed between injury and exploration was ranged from 2 to 6 hours. Eight cases had firearm injury. Open fracture radius was involved in 4 cases while fracture proximal ulna and radius was involved in one case. Debridement, open reduction and internal fixation and median nerve epineurorrhaphy were performed for 5 cases while debridement and epineurorrhaphy were performed for 3 cases. Seven cases had median nerve injury below the elbow caused by motor car accident. There were forearm complex injuries, i.e injuries of bone, muscles tendons, nerves, vessels and skin. Debridement, bone stabilization, vascular repair, nerve repair, and tendon repair were performed consequently. Machine injury was the leading cause in the remaining 2 cases of median nerve injury with complex forearm injuries.

Wrist and finger flexors were M5 in 28 (70%) patients (Fig. 5), M4 in 10 (25%) and M3 in 2 patients. S3 was gained in 32 (80%), and S2+ in 8 patients. Hand grip was 90% in 32 (80%), and 75% in 8 (20%) patients.

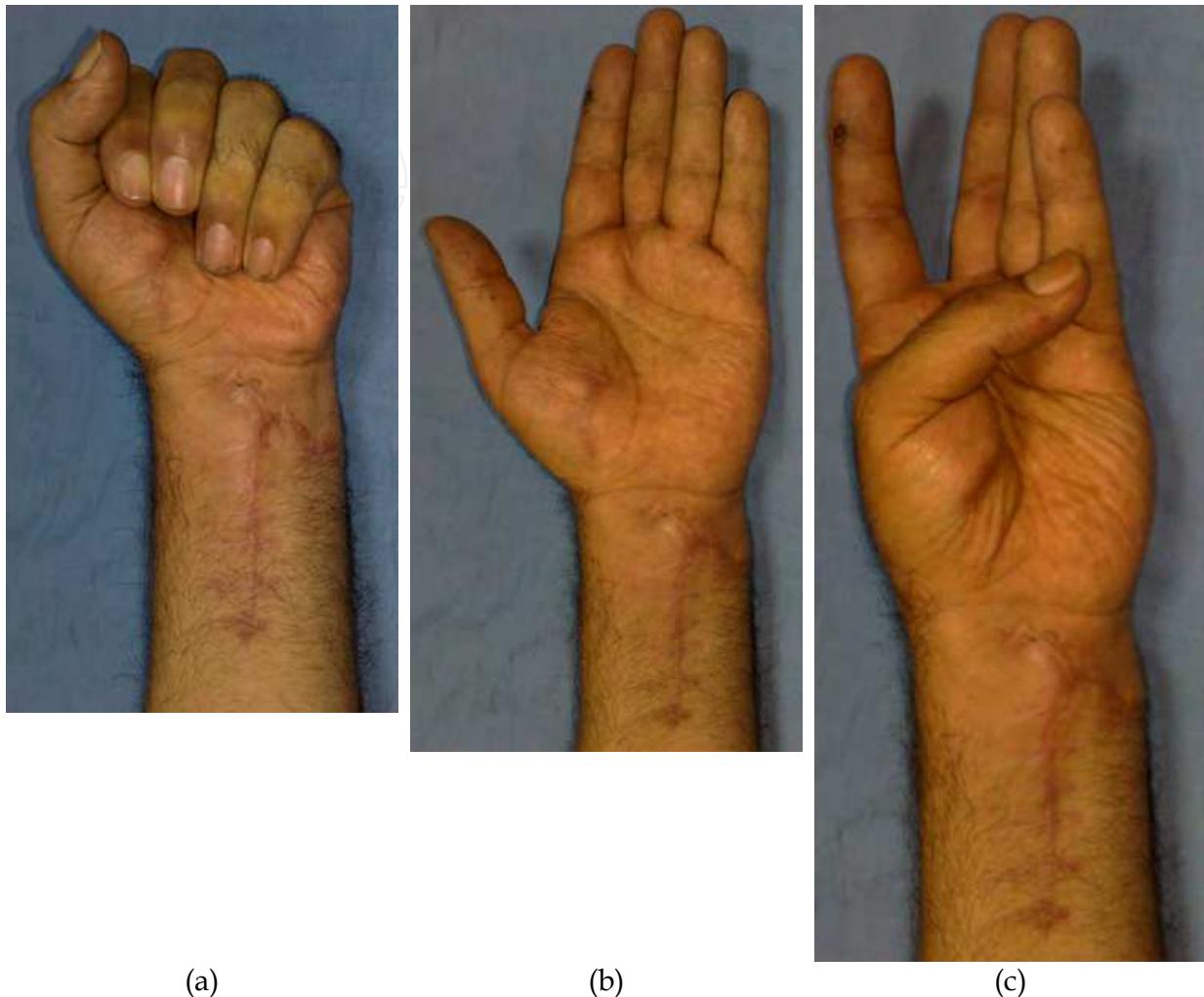


Fig. 5. Male patient 32 years old had cut wound at mid- forearm presented by median nerve injury. Primary exploration of the median nerve and repair were done. Post operative follow up after 24 month showed: (a) Complete finger flexion. (b) Complete finger extension. (c) Normal thumb opposition.

5.4 Median nerve injury at wrist

It is rarely to have isolated median nerve injury at the wrist. It is usually associated with injuries of one or more of the following structures; flexor tendons, radial artery, ulnar nerve, ulnar vessels and bone (distal radius and/or carpal bones). A retrospective review of 42 patients with spaghetti wrist lacerations operated on by the author between June 1997 and May 2005 was completed. A total of 31 males and 11 females, average age of 17.1 years (range, 2–40 years), sustained spaghetti wrist injuries. The most frequent mechanisms of injury were accidental glass lacerations (55%), knife wounds (24%), and electrical saw injuries (11%). An average of 9.16 structures was injured, including 6.95 tendons, 1.4 nerves,

and 0.8 arteries. The most frequently injured structures were median nerve (83%), flexor digitorum superficialis 2-4 tendons (81%), flexor digitorum profundus 2-4 tendons (66%), ulnar nerve and ulnar artery (57%), and flexor pollicis longus (40%). Combined flexor carpi ulnaris, ulnar nerve, and ulnar artery (ulnar triad) injuries occurred in 31%, while combined median nerve, palmaris longus, and flexor carpi radialis injuries (radial triad) occurred in 43%. Simultaneous injuries of both median and ulnar nerves occurred in 40.5%. Simultaneous injuries of both ulnar and radial arteries occurred in 14%. Neither artery was injured in 30.9%. Follow-up has ranged from 1 to 8 years, with an average of 46 months. Only four patients have been completely lost to follow-up. Range of motion of all involved digits (tendon function) was excellent in 34 patients, good in 3 patients, and poor in only one patient. Opposition was excellent in 31 patients, good in 5 patients, and poor in 2 patients. Intrinsic muscle recovery was subjectively reported to be excellent in 29 patients, good in 7, and fair to poor in 2 patients. Minor deformity (partial clawing) was reported in 4 patients and one patient has major deformity (total clawing). Sensory recovery was reported, excellent in 32 patients, good in 5 patients, and fair in only one patient (Table 1) ⁽²⁸⁾.

	Tendon function	Opposition	Intrinsics	Deformities	Sensation
Excellent	Individual tendon function was evident with 85% to full range of motion or finger flexion to 1.0 cm or less from the distal palmer crease	When the tip of the thumb moves freely over the three phalanges of the other four fingers	When the patient can do both finger abduction and adduction with -ve froment sign	Major if there is both clawing and ape hand	When the two point discrimination is less than 10 mm
Good	70-84% total normal range of motion or 2.0 cm from the distal palmer crease	When the tip of the thumb touches only the tip of the other four fingers	When the patient can do both finger abduction and adduction with +ve froment sign		When the two point discrimination is 10-20 mm
Fair	50-69% total normal range of motion	When the tip of the thumb cannot reach the tip of the other four fingers	When the patient can do either finger abduction or adduction with +ve froment sign	Minor if there is either clawing or ape hand	When the two point discrimination is more than 20 mm with light touch and pain prick sensation
Poor	Fixed contractures or adhesions		When the patient cannot do finger abduction or adduction with +ve froment sign		When there is trophic changes or skin ulceration

Table 1. Noaman's evaluation system for follow up results of spaghetti wrist

6. Ulnar nerve

The ulnar nerve arises from the medial cord of the brachial plexus. It contains the nerve root fibers from C8-T1. It provides the motor supply to the hypothenar muscles, the ulnar two lumbricals, the interosseous muscles, the adductor pollicis, the FCU, and the profundus to the ring and small fingers. Its sensory distribution includes the palmar surface of the small finger, the ulnar half of the ring finger, and the dorsoulnar carpus. It lies medial to the axillary artery and continues distally to the midarm, where it pierces the medial intermuscular septum. The nerve often is accompanied by the superior ulnar collateral artery. At the elbow, it lies between the medial epicondyle and the olecranon, where it is covered by Osborne's ligament. It enters the forearm between the two heads of the FCU covered by a fibrous aponeurosis (the cubital tunnel). It runs deep to the FCU until the distal forearm. At the wrist, it passes over the transverse carpal ligament, medial to the ulnar artery through Guyon's canal. The deep motor branch is given off at the pisiform and passes underneath a fibrous arch to lie on the palmar surface of the interossei. It crosses the palm deep to the flexor tendons, to terminate in the adductor pollicis and ulnar head of the flexor pollicis brevis.

6.1 Ulnar nerve injury above elbow

It is manifested by partial claw hand, loss of finger adduction and abduction, positive Froment test, loss of sensation over the medial one third of the palm and medial one and half fingers (Fig. 6). Retrospectively, we treated 22 cases of ulnar nerve injury above the elbow by microneurorrhaphy. Median, radial and musculocutaneous nerve injuries were also involved in 7 cases. Cut wound was responsible for 10 cases, firearm injuries in 4 cases, post reduction injury of supracondylar fracture humerus in 4 cases, machine injury in 3 cases, and motor car accident with open fracture humerus in one patient. The mean age was 23 years. Right side was injured in 14 patients. Primary repair was performed for all patients. The mean follow up was 46 months. We used British medical research council grading system for both motor and sensory evaluation and Noaman⁽²⁸⁾ evaluation system for intrinsic function. Wrist and finger flexors were M5 in 7 cases, M4 in 9 cases and M3 in 6 cases. Sensation was S3 in 10 patients, S2+ in 9 patients and S2 in 3 patients. Hand grip was 80% in 15 patients, 70% in 5 patients and 60% in 2 patients.

6.2 Below elbow

Retrospectively, we treated 27 cases of ulnar nerve injury below elbow by microneurorrhaphy. Median nerve was involved in 12 patients. Cut wound was responsible for 15 cases, firearm injuries in 6 cases, machine injury in 3 cases, and motor car accident with open fracture radius and ulna in three patients. The mean age was 23 years. Right side was injured in 11 patients. Primary repair was performed for all patients. The mean follow up was 42 months. We used British medical research council grading system for both motor and sensory evaluation and Noaman evaluation system for intrinsic function. Wrist and finger flexors were M5 in 15 cases, M4 in 8 cases and M3 in 4 cases. Sensation was S3 in 18 patients, S2+ in 7 patients and S2 in 2 patients. Hand grip was 80% in 16 patients, 70% in 9 patients and 60% in 2 patients.



(a)



(b)



(c)

Fig. 6. Male patient 9 years old presented by ulnar nerve injury after percutaneous pinning of supracondylar fracture humerus, (a) Clinical manifestation of partial claw hand with trophic ulcer of the little finger. (B) And (C) antero-posterior and lateral views of the x-ray elbow with complete union of the supracondylar fracture humerus.

6.3 At the wrist

The ulnar nerve usually associated with other soft tissues (including tendons, vessels, and median nerve) at the wrist.

7. Sciatic nerve

Fortunately, the incidence of sciatic nerve injury is uncommon because its recovery (conservatively or after surgery) does not like the recovery of other peripheral nerves. This is most likely due to highly mixed nerve, thick nerve and very long nerve. The causes of sciatic nerve injury are variable. It is vulnerable for injury in cases of fracture acetabulum specially posterior column and posterior wall acetabulum fracture. Posterior hip dislocation, central hip fracture dislocation can be associated with sciatic nerve injury. These can be induced by motor car accident, fire arm injuries and falling from height. Iatrogenic sciatic nerve injury is the next common cause of sciatic nerve injury. It can be happened during posterior exposure of the hip e.g. (open reduction and internal fixation of fracture acetabulum, total hip replacement and even hemiarthroplasty). I noticed sciatic nerve injury after plate and screw fixation for fracture shaft femur. Sciatic nerve injury from intramuscular (gluteal) injection also is not uncommon.

The manifestations of sciatic nerve injury are variable. Usually the branches to the hamstring muscles (knee extensors) take off from the sciatic nerve before its vulnerable for injury. This is mean that the manifestations of sciatic nerve injury appear below the knee (flail ankle and loss of superficial and deep sensation over the leg and foot).

7.1 Tibial nerve injury

The tibial nerve is the larger of the two terminal branches of the sciatic nerve which separate about the middle of the back of the thigh. It supplies the muscles of the back of the leg and the sole of the foot, and the skin of the lower half of the back of the leg and the lateral side and sole of the foot. It is vulnerable to injury in cases with fracture lower femur and proximal tibia caused by high energy trauma e.g. motor car accident, falling from height, fire arm injury also caused tibial nerve injury without fracture. Cut wound in the popliteal fossa also can cause tibial nerve injury which is usually associated with popliteal artery injury.

The incidence of tibial nerve injury is not common. We treated 7 cases of tibial nerve injury from 1998 to 2010. Five cases were male and 2 cases were female. The average age was 13 years. The affected site was right side in 5 cases and left in 2 cases. The causative agent was fire arm injury in 3 cases, motor car accident in 2 cases and cut wound in 2 cases. Neurolysis was performed in 3 cases, repair in 3 cases and nerve graft in one case. The average follow up was 124 month. Plantar flexors of the ankle and toes were G5 in 2 cases of neurolysis, G4 in 3 cases, one of them had neurolysis and 2 had nerve repair, G3 in 2 cases (one treated by nerve repair and one treated by nerve graft). Sensation was gained in 7 cases.

7.2 Common peroneal nerve

This nerve is smaller than the tibial nerve. It supplies the muscles on the lateral and anterior surfaces of the leg and the dorsum of the foot and skin on the lateral side of the leg and the greater part of the dorsum of the foot.

It is usually vulnerable for injury because it is superficial and runs around the neck of the fibula. The causes of injury are the same like tibial nerve injury in addition to causes of entrapment neuropathy. It is common to be injured during squatting position so common in painters, farmers, and carpenters.

We treated 12 cases of common peroneal nerve injuries in the last 12 years. Six cases had entrapment neuropathy which did not recovered conservatively. Neurolysis was performed and full motor and sensory recovery were gained 36 month postoperatively. Six cases treated by nerve repair, followed up to 54 month. Motor recovery was G5 in 2 cases, G4 in 3 cases and G3 in one case. Sensory recovery was satisfactory.

8. Methods to overcome nerve defect

There are several methods of closing gaps between nerve ends without appreciable damage to the nerve itself. The methods most often used are mobilization of the nerve ends and positioning of the extremity. Other methods include nerve transplantation, bone resection, bulb suture, nerve grafting, and nerve crossing (pedicle grafting).

1. Mobilization

It means lysis of the nerve through its course and to cut off small insignificant branches which tend to fix it through its pathway. It can be done to proximal and distal stump. It provides sufficient length in some cases. It needs wide nerve exposure. Most small gaps can be closed by mobilizing the nerve ends for a few centimeters proximal and distal to the point of injury. Mobilization of both nerve ends to some degree is required in all peripheral neurorrhaphies⁽⁴⁾. The exact amount of mobilization a peripheral nerve can tolerate before its regenerating potential is compromised is unknown; however, extensive dissection of a nerve from its surrounding tissues does disrupt the segmental blood supply, causing subsequent ischemia and increased intraneural scarring. Starkweather et al⁽²⁹⁾ showed that mobilization is more detrimental to the distal nerve segment, as manifested by increased intraneural collagen production and collagen cross-linking. Seddon⁽³⁾ suggested that extensive mobilization adversely affects recovery after median nerve repairs in the forearm. They found that if the gap was less than 2.5 cm, motor recovery was to the M3 level or better in 70%, whereas if the gap was 2.6 to 5 cm and required extensive mobilization, recovery was to the M3 level or better in only 50%. Large gaps require extensive dissection of the nerve from its adjacent tissues for a relatively tension-free epineurial repair. Before subjecting a peripheral nerve to extensive dissection, the surgeon should have some idea of the maximal nerve gap over which mobilization may become a futile endeavor. The nerve gap is determined at the time of surgery with the extremity in the anatomical position and after distal and proximal neuroma excision. Certain guidelines, although extremely variable, are found in the literature. Zachary⁽³⁰⁾ showed that the median and ulnar nerves can be mobilized 7 to 9 cm, and with anterior transposition an ulnar nerve gap of 13 cm can be overcome. Wilgis⁽³¹⁾ cautioned against extensive mobilization to overcome gaps greater than 4 cm. Millesi⁽³²⁾ recommended that defects greater than 2.5 cm be treated by interfascicular nerve grafting rather than by extensive mobilization and positioning.

2. Positioning

Relaxing nerves by flexing various joints and occasionally by other maneuvers, such as abducting, adducting, rotating, and elevating the extremity, is as important as mobilization

in closing large gaps in nerves. Through use of both methods, long gaps can be closed in nearly all of the peripheral nerves, and many unsatisfactory neurorrhaphies result from failure to make the most of their possibilities. When joints that are excessively flexed or awkwardly positioned are mobilized later, tension on the neurorrhaphy may be too great and may cause intraneural fibrosis that compromises axonal regeneration. Consequently, a joint should never be flexed forcibly to obtain end-to-end suture. It is a reasonable policy to flex the knee and elbow no more than 90 degrees. Also, flexion of the wrist more than 40 degrees is probably unwise. After the wound has healed sufficiently, the joint can be extended about 10 degrees per week until motion is regained. Flexing joints is most important in repairing gaps in the long nerves of the extremities. External rotation and abduction are helpful when repairing radial and axillary nerves, as in elevation of the shoulder girdle in brachial plexus injuries. Rarely, extension of a joint can be helpful, as in extension of the hip in sciatic injuries. Strong consideration should be given to nerve grafting in preference to drastic positioning of the extremity to produce a tension-free neurorrhaphy ⁽⁴⁾.

3. Nerve transposition

The anatomical course of some nerves can be changed to shorten the distance between severed ends. This is true especially of the ulnar nerve at the elbow. The median nerve also can be transposed anterior to the pronator teres if the lesion is distal to its branches to the long flexor muscles of the forearm, and the tibial nerve can be placed superficial to the soleus or gastrocnemius in the leg if the lesion is distal to its branches to the calf muscles. Most surgeons recommend transposition of the proximal end of the radial nerve anterior to the humerus and deep to the biceps to obtain needed length. Considerable length can be gained in most patients by the simpler maneuver of externally rotating the arm, provided that the mobilization has been carried into the axilla, and that the branches of the radial nerve to the triceps muscle have been dissected well up the nerve ⁽⁴⁾.

4. Bone shortening

In civilian injuries, bone resection almost never should be necessary to accomplish neurorrhaphy. Even in war wounds, it rarely was employed, and when it was used, it usually was because the joints of the extremity had become so stiff from immobilization caused by fracture or injudicious use of casts that limited flexion. Intact long bones and most bones in children rarely if ever should be shortened to aid in nerve repair. Bone resection is of particular value in the upper arm for closing large gaps in the ulnar, radial, or median nerves when the humerus already has been fractured. If early delayed suture is done in such patients before the fracture has healed, shortening the bone if necessary is not difficult. After the fracture has healed, however, osteotomy is more difficult. It rarely is worthwhile to shorten the femur in injuries of the sciatic nerve, unless this bone already has been fractured; shortening of the bone can be helpful. Both bones of the forearm or leg in the absence of a fracture should never be shortened ⁽⁴⁾.

5. Nerve graft

Interfascicular nerve grafting as described by Seddon ⁽³⁾ (and later by Millesi ⁽³²⁾) is indicated when primary nerve repair cannot be done without excessive tension. In general, a nerve gap that is caused simply by elastic retraction usually can be overcome with local nerve mobilization, limited joint positioning, and primary repair. If the defect is caused in part by

loss of nerve tissue, however, nerve grafting is our procedure of choice. When primary repair cannot be performed without undue tension, nerve grafting is required. Autografts remain the standard for nerve grafting material. Allografts have not shown recovery equivalent to that obtained with autogenous nerve and are still considered experimental. The three major types of autograft are cable, trunk, and vascularized nerve grafts. Cable grafts are multiple small-caliber nerve grafts aligned in parallel to span a gap between fascicular groups. Trunk grafts are mixed motor-sensory whole-nerve grafts (e.g., an ulnar nerve in the case of an irreparable brachial plexus injury). Trunk grafts have been associated with poor functional results, in large part due to the thickness of the graft and consequent diminished ability to revascularize after implantation. Vascularized nerve grafts have been used in the past, but with conflicting results. They may be considered if a long graft is needed in a poorly vascularized bed. Because donor-site morbidity is an issue, vascularized grafts have been most widely utilized in irreversible brachial plexus injuries. Autogenous sural nerve is the preferred source of graft. We do not use vascularized nerve grafts, trunk grafts, or allografts. The most common source of autograft is the sural nerve (Fig. 7), which is easily obtainable, the appropriate diameter for most cable grafting needs, and relatively dispensable. Other graft sources include the anterior branch of the medial antebrachial cutaneous nerve, the lateral femoral cutaneous nerve, and the superficial radial sensory nerve. The technique of nerve grafting involves sharply transecting the injured nerve ends to excise the zone of injury (Fig. 8). The nerve ends should display a good fascicular pattern. The defect is measured, and the appropriate length of graft is harvested to allow reconstruction without tension. If the injured nerve has a large diameter relative to the nerve graft, several cable grafts are placed in parallel to reconstruct the nerve. The grafts are matched to corresponding fascicles and sutured to the injured nerve with epineurial sutures, as in the primary neuroorrhaphy technique (Fig. 9). Fibrin glue may be used to connect the cable grafts, thus decreasing the number of sutures and minimizing additional trauma to the nerve grafts. The surgeon can make fibrin glue intraoperatively by mixing thrombin and fibrinogen in equal parts, as originally described by Narakas⁽³³⁾. Although nerve grafts have not generally been considered polarized, it is recommended that the graft be placed in a reversed orientation in the repair site. Reversal of the nerve graft decreases the chance of axonal dispersion through distal nerve branches. A well-vascularized bed is critical for nerve grafting. The graft should be approximately 10% to 20% longer than the gap to be filled, as the graft inevitably shortens with connective tissue fibrosis. From their clinical experience, Millesi et al⁽³⁴⁾ reported good results using an interfascicular nerve autografting technique to close gaps without undue tension. In the upper extremity especially, good results were achieved in repairing injuries to the digital, median, ulnar, and radial nerves. Of 38 patients with median nerve grafts, 82% achieved useful motor recovery (M3 or better), and all but 1 regained protective sensibility. Of 39 patients with ulnar nerve grafts, all achieved useful motor recovery (M2+ or better), and 28% regained 2-point discrimination. Of 13 patients with radial nerve grafts, 77% achieved an M4 or M5 level of function.

9. Allografts

Allografts have several potential clinical advantages: (1) grafts can be banked; (2) there is no need for sacrifice of a donor nerve; and (3) surgical procedures are quicker without the need to harvest a graft. However, allografts are not as effective as autografts, mainly due to the

immunogenic host response. Anselin and Pollard ⁽³⁵⁾ studied rat allograft nerves and found an increase in helper T cells and cytotoxic/suppressor T cells, implying immunogenic rejection. The cellular component of allografts, and with it, their immunogenicity, can be destroyed by freeze-thawing. This leads to the production of cell debris, which in turn impairs neurite outgrowth. Dumont and Hentz ⁽³⁶⁾ reported on a biologic detergent technique that removes the immunogenic cellular components without forming cell debris. Their experiments in rats have shown that allografts processed with this detergent had equivalent post repair results compared with autografts.

9.1 Role of the nerve graft

The nerve graft acts to provide a source of empty endoneurial tubes through which the regenerating axons can be directed. Any tissue that contains a basal lamina, such as freeze-dried muscle or tendon, can be substituted ⁽³⁷⁾, but only the autogenous nerve graft also provides a source of viable Schwann cells. To be effective, the graft must acquire a blood supply. If the nerve graft survives, the Schwann cells also survive ⁽³⁸⁾.



Fig. 7. Post operative photo showing a nice scar after sural nerve taken from both legs.



Fig. 8. Intra-operative photo showing a long median nerve defect at the forearm.



Fig. 9. Intraoperative photo showing 2 cable grafts bridging the median nerve defect with micro-epi-neurorrhaphy at both sides of the graft.

9.2 Graft incorporation

When separated from its blood supply, the graft undergoes wallerian degeneration. Schwann cells can survive 7 days, depending purely on diffusion⁽³⁹⁾. By 3 days after implantation, there is invasion of the nerve graft by endothelial buds from the surrounding tissue bed, with evidence of high nerve blood flows by 1 week^(40,41). This segmental vascular sprouting from extraneural vessels is not limited by the length of the graft^(42,43). The length of the graft is, within certain limits, of no significance to the end result, provided that there is a tension-free anastomosis.

The ingrowth of vessels from the ends of the graft (inosculation) does not seem to be of major importance, unless the recipient bed is poorly vascularized. The late phase of nerve graft incorporation shows migration of Schwann cells from the proximal nerve end into the graft and from the graft into both host nerve ends⁽⁴⁴⁾.

9.3 Graft diameter

Small-diameter grafts spontaneously revascularize, but large-diameter grafts do so incompletely⁽⁴⁵⁾. Thick grafts undergo central necrosis with subsequent endoneurial fibrosis. This fibrosis ultimately impedes the advancement of any ingrowing axon sprouts. Cable nerve grafts are similar to thick grafts. They consist of numerous nerve grafts that are sutured or glued together to match the caliber of the recipient nerve. Because a large percentage of the surface is in contact with another graft and not in contact with the recipient bed, the central portions may not revascularize. With large-diameter recipient nerves, it is preferable to use multiple smaller caliber grafts to bridge fascicular groups in the proximal and distal stumps to increase the surface area that is in contact with the recipient bed⁽⁴⁵⁾.

9.4 Nerve biomechanics

A normal nerve has longitudinal excursion, which subjects it to a certain amount of stress and strain in situ. Peripheral nerve is initially easily extensible. It rapidly becomes stiff with further elongation as a result of the stretching of the connective tissue within the nerve. Chronically injured nerves become even stiffer. Elasticity decreases by 50% in the delayed repair of nerves in which wallerian degeneration has occurred. Experimentally, blood flow is reduced by 50% when the nerve is stretched 8% beyond its *in vivo* length. Complete ischemia occurs at 15%. Suture pullout does not occur until a 17% increase in length; this suggests that ischemia and not disruption of the anastomosis is the limiting factor in acute nerve repair. This observation also is applicable to nerve grafting. Nerve is a viscoelastic tissue in that when low loading in tension is applied over time, the nerve elongates, without deterioration in nerve conduction velocities. Stress relaxation results in recovery of blood flow within 30 minutes at 8% elongation. Intriguing experimental work has been done with gradual nerve elongation to overcome nerve gaps using tissue expansion and external fixation, but this cannot be considered an accepted standard of treatment as yet.

A normal nerve can compensate for the change in length with limb flexion and extension because it is surrounded by gliding tissue that permits longitudinal movement. The change

in length is distributed over the entire nerve so that the elongation of each nerve segment is small. A nerve graft becomes welded to its recipient bed by the adhesions through which it becomes vascularized. As a consequence, the nerve graft is exquisitely sensitive to tension because it has no longitudinal excursion. The harvested length of the graft must be long enough to span the nerve gap without tension while the adjacent joints are extended; this is also the position of temporary immobilization. If the limb or digit is immobilized with joint flexion, the graft becomes fixed in this position. When the limb is mobilized at 8 days, the proximal and distal stumps are subject to tension even though the graft initially was long enough. Early attempts at lengthening the graft lead to disruption of the anastomosis ⁽⁴⁶⁻⁵³⁾.

9.5 Grafting versus primary repair

A tension-free repair is the goal for any nerve anastomosis. When there is a clean transaction of the nerve and the gap is caused by elastic retraction, an acute primary repair is indicated. When treatment of a nerve laceration is delayed, fibrosis of the nerve ends prevents approximation, and nerve grafting is indicated even though there is no loss of nerve tissue. As a general rule, primary nerve repair yields superior results to nerve grafting, provided that there is no tension across the anastomotic site. Grafting can obtain similar results to primary repair under ideal conditions. If a nerve is repaired under tension, however, the results are superior with an interpositional graft. Axon sprouts are able to cross two tension-free anastomotic sites more easily than crossing one anastomosis that is under tension. Nerve grafting is indicated to bridge a defect when greater than 10% elongation of the nerve would be necessary to bridge the gap. This is a better indication for grafting than the nerve gap per se, although 4 cm is often used as the critical defect for grafting in the limb. Defects less than this may be overcome by nerve rerouting and transposition in some instances ⁽⁵⁴⁻⁵⁸⁾.

9.6 Considerations for donor nerve grafts

Many conditions must be met for a nerve to be considered as a potential graft. First, the relationship between the surface area and the diameter of the graft must be optimal to allow rapid revascularization. The donor site defect from sacrifice of any given nerve must be acceptable for the patient. The harvested nerve must be long enough to ensure a tension-free anastomosis with the adjacent joints in full extension. Finally, the cross-sectional area and number of fascicles should match those of the recipient nerve at the level of injury as closely as possible. For these reasons, most of the available grafts are cutaneous nerves. Most donor grafts are imperfect matches of the recipient nerve. The fascicular arrangement of the nerve graft is dissimilar to the nerve being repaired in size, number, and fascicular topography. The branching pattern of the grafts usually changes from an oligofascicular pattern proximally to a polyfascicular pattern distally, which typically corresponds to the branching pattern of the recipient nerve. There may be some loss of axon sprouts owing to growth down peripheral branches that leave the nerve graft. Some authors have recommended inserting the grafts in a retrograde manner for this reason, but others believe this is not warranted ⁽³¹⁾. The choice of nerve graft is dictated by the length of the nerve gap, the cross-sectional area of the recipient nerve, the available expendable donor nerves for that particular nerve injury, and the surgeon's preference.

9.7 Donor nerve grafts

As a general rule, it is good practice to divide the donor nerve in an intermuscular plane rather than in the subcutaneous tissue to diminish the risk of a painful neuroma. Some commonly used donor nerves are summarized here. Sunderland ⁽¹⁾ emphasized that a cutaneous nerve for nerve grafting should be selected with great care. The sural nerve is the most commonly used, and in most situations it is recommended. From each leg, 40 cm of graft material can be obtained. McFarlane and Mayer (1976) ⁽⁵⁹⁾, Wilgis and Maxwell (1979) ⁽⁶⁰⁾, and Tenny and Lewis (1984) ⁽⁶¹⁾ used the lateral antebrachial cutaneous nerve for digital nerve grafts so that another limb would not be involved in the surgical procedure. Anatomical studies have shown no significant difference in fascicular area, area of the entire nerve bundle, and percentage of the nerve bundle occupied by the actual nerve fascicles. The lateral antebrachial cutaneous nerve is found most easily just lateral to the biceps tendon alongside the cephalic vein. Through a longitudinal incision, 20 cm of graft material can be obtained. The medial antebrachial cutaneous nerve, the terminal articular branch of the posterior interosseous nerve, and the dorsal sensory branch of the ulnar nerve also have been used for digital nerve grafting. The medial antebrachial cutaneous nerve is found adjacent to the basilic vein. The posterior interosseous nerve is located at the wrist just ulnar to the extensor pollicis longus tendon lying on the interosseous membrane. The superficial radial nerve is an excellent source of graft material when used in grafting a high radial nerve laceration because the neurological deficit that otherwise would be created already exists. It is not recommended as a routine source because its sensory contribution to the hand is significant, especially when the median nerve is deficient ^(4, 62).

9.8 Motor sensory differentiation

The use of intraoperative motor and sensory nerve differentiation can diminish the risk of fascicular mismatch when grafting a nerve. Available methods are the anatomic method, based on separate identification of groups of fascicles; the electrophysiologic method, using awakened stimulation and histochemical methods, which rely on staining for enzymes specific to motor or sensory nerves.

Electrical fascicle identification awakened stimulation requires the cooperation of the anesthesiologist and the patient. It is based on the observation that motor and sensory fascicles can be differentiated by direct stimulation. The median and ulnar nerves in the distal forearm are most amenable to this technique. It is especially useful when there is a nerve defect, owing to the dissimilar fascicular pattern between the proximal and distal nerve ends. The initial nerve dissection is performed under a regional block with tourniquet control. The wound is infiltrated with local anesthetic before release of the tourniquet. After 20 minutes, the patient is awakened. A lowamperage stimulator is applied to the major fascicles of the proximal nerve end in a systematic manner, starting at 0.2 to 0.5 mA. Sensory fascicles elicit pain and may be localized to a specific digit. Motor fascicles elicit no response at lower intensities and poorly localized pain at higher intensities. A cross-sectional sketch of the proximal stump is made. The sensory fascicles are tagged with 10-0 nylon, and the patient is placed under general anesthesia. The distal stump is stimulated in a similar fashion. The reverse picture is seen, with motor fascicles eliciting a muscle twitch and sensory fascicles being silent. A cross-sectional map is made again and used to match the proximal and distal motor and sensory fascicles ⁽⁶³⁻⁶⁹⁾.

9.9 Nerve lesions in continuity

Electrical stimulation is useful to determine if there are any intact fascicles in a neuroma in continuity. Bipolar hook electrodes are used with the stimulating and recording electrodes separated by at least 4 cm. The stimulus frequency is two to three times per second with pulse duration of less than 0.1 ms. The intensity is slowly increased to the range where a response is expected (3–15 V). The recorder sensitivity is increased to a maximum of 20 $\mu\text{V}/\text{cm}$. The nerve is stimulated proximal to, across, and below the lesion. It is estimated that there must be at least 4000 myelinated axons for a recordable nerve action potential to conduct through a neuroma. A neurolysis is performed to single out any normal-appearing fascicles; this is confirmed electrically. Non conducting fascicles are excised and grafted ⁽⁷⁰⁾.

- Grafting specific nerves
- Median nerve
- Injury at the elbow
- The Ulnar nerve

10. Factors affecting the results of nerve surgery

Few worthwhile reports have been published on the results of neurorrhaphy and the factors that influence them, first because few investigators have had access to a large enough group of patients to make evaluations statistically significant, and second because reports have only rarely been based on sound criteria of regeneration. Valuable reports have been compiled from studies of such injuries incurred in World War II and later conflicts. As a result of these studies, the influence of many factors on regeneration after nerve suture is now better understood. Rarely should a fracture interfere with nerve repair. In the usual situation, a nerve may be explored if the fracture requires open reduction. In many open injuries, the nature of the wound may be such that early repair of the nerve cannot be done satisfactorily. Every effort should be made by repeated débridement of necrotic material to promote rapid healing of any open wounds without sepsis. Nerves may be repaired successfully during a second débridement, followed by closure and healing. Associated vascular injury can adversely affect nerve regeneration because of tissue ischemia ⁽⁴⁾.

Several important factors that seem to influence nerve regeneration are (1) the age of the patient, (2) the gap between the nerve ends, (3) the delay between the time of injury and repair, (4) the level of injury, (5) the condition of the nerve ends, (6) type of nerve, (7) specific nerve involved, (8) associated vascular injury, and (9) the experience and techniques of the surgeon.

10.1 Age

Age undoubtedly influences the rate and degree of nerve regeneration. All other factors being equal, neurorrhaphies are more successful in children than in adults and are more likely to fail in elderly patients; why this is true has not been completely explained, but it may relate to the potential for central adaptation to the peripheral nerve injury. We do not

know precisely what results can be expected in either of the extremes of age, for practically all significant studies have dealt with military personnel, whose average age was 18 to 30 years. Omer ⁽⁷¹⁾, in reviewing peripheral nerve injuries in upper extremities incurred in the Vietnam War, found the most successful results after neuroorrhaphy in patients younger than 20 years. The work of Onne ⁽⁷²⁾ suggests a close correlation between the age at the time of neuroorrhaphy and the 2-point discrimination obtained after median and ulnar nerve repairs. Most of his patients between the ages of 20 and 40 years were found to have 2-point discrimination values in the range of 30 mm. During the teens, the values did not exceed 15 mm, and in patients 10 years old or younger, the values, with one exception, were less than 10 mm. Onne ⁽⁷²⁾ observed, however, that after digital nerve repair the final 2-point discrimination was not as closely related to age. Kankaanpää and Bakalim ⁽⁷³⁾, in studying sensory recovery after 137 peripheral neuroorrhaphies in 96 patients, found that a higher percentage of patients younger than 20 years at the time of repair had 2-point discrimination of less than 6 mm than did patients older than 20.

10.2 Gap between nerve ends

The nature of the injury is the most important factor in determining the defect remaining between the nerve ends after any neuromas and gliomas are resected. When a sharp instrument, such as a razor or knife, severs a nerve, damage is slight proximally and distally, and although the nerve ends inevitably do retract, the gap usually can be overcome easily. Conversely, when a high-velocity missile severs a nerve, proximal and distal nerve damage is extensive. Ultimately, both ends must be widely resected to expose normal funiculi, producing a larger gap. The gap is increased further if part of the nerve is carried away by a missile, as in shrapnel injuries. Methods of closing troublesome gaps include (1) nerve mobilization, (2) nerve transposition, (3) joint flexion, (4) nerve grafts, and (5) bone shortening. The greater the defect, the more dissimilar the funicular pattern of the two ends because of the constantly changing arrangement of fibers within the nerve as it progresses distally. This is particularly important in the more proximal portion of peripheral nerves. Agreement is widespread that excessive tension on a neuroorrhaphy harms nerve regeneration. Brooks advised nerve grafting if, after the nerve is mobilized, the gap cannot be closed by flexing the main joint of the limb 90 degrees. Sunderland cautioned against excessive tension on the line of suture after surgery to avoid excessive fibrosis. He advised a combination of transplantation, transposition, and mobilization of the nerve to close gaps. After experimental and clinical observations, Millesi ⁽³²⁾ concluded that tension at the line of suture is the most important factor influencing the results of neuroorrhaphy. He advised intrafascicular nerve grafting to close the large gaps. Nicholson and Seddon ⁽³⁾ and Sakellarides ⁽⁷⁵⁾ observed that the upper limit of a gap beyond which results deteriorate is approximately 2.5 cm. The observations of Kirklin, Murphey, and Berkson ⁽⁷⁶⁾ in 1949 that recovery is slightly better when the gap is relatively small remain valid.

10.3 Delay between time of injury and repair

Delay of neuroorrhaphy affects motor recovery more profoundly than sensory recovery. Scarff suggested that this is related to the survival time of denervated striated muscle.

Sunderland reported that satisfactory reinnervation of human muscle can occur after denervation of 12 months. The observations of patients with peripheral nerve injuries during World War II revealed that for every delay of 6 days between injury and repair, there is a variable loss of potential recovery that averages about 1% of maximal performance; after 3 months, this loss increases rapidly. In addition, return of function in distal muscles is poor when suture is late. The influence of delay on sensory return is unclear; in the Veterans Administration study, little influence could be found, and useful sensation returned in a few patients when suture was performed 2 years after injury. The critical limit of delay beyond which sensation does not return is unknown ⁽⁴⁾.

The British found early suture important in reducing the number of painful paresthesias and in regaining a useful degree of sensation. Kankaanpää and Bakalim ⁽⁷³⁾ studied sensory return after 137 neurorrhaphies and found that, if done within 3 months after injury, the results usually were better after secondary repair than after primary repair; 21% of the 85 primary repairs and 38% of the 52 secondary repairs regained 2-point discrimination of 6 mm or less. The difference in return of sensation was most marked in digital nerve repairs. No difference in return between the primary and secondary repairs was noted in the nerve injuries at the wrist or in the forearm. The experimental work of Ducker et al ⁽⁷⁷⁾ revealed a consistent timetable for intracellular metabolic events after nerve injury. They found that between 3 and 6 weeks the degenerative and reparative changes within the nerve cell body and the proximal and distal nerve trunks were well established. Kleinert et al ⁽⁷⁸⁾ reported their clinical impression that a delayed primary repair done 7 to 18 days after injury is best for return of satisfactory function. In Omer's ⁽⁷¹⁾ study, 70% of successful repairs of lacerated nerves in the upper extremity had been done within 6 weeks after injury, and all successful repairs had been done within 3 months. Our practice is to perform neurorrhaphies in clean, sharp wounds immediately or during the first 3 to 7 days. In the presence of extensive soft-tissue contusion, laceration, crushing, or contamination in which the proximal and distal extent of the nerve injury is impossible to delineate, a delay of 3 to 6 weeks is preferred.

10.4 Level of injury

The more proximal the injuries, the more incomplete the overall return of motor and sensory function, especially in the more distal structures. Sunderland observed that conditions are more favorable for recovery in the more proximal muscles because (1) the neurons that innervate the distal portions of the limb are more severely affected by retrograde changes after proximal injury, (2) a greater proportion of the cross-sectional area of the nerve trunk is occupied by fibers to the proximal muscles, and (3) the potential for disorientation of regrowing axons and for axon loss during regeneration is greater for the distal muscles than for the muscles more proximally situated after a proximal injury. Boswick et al ⁽⁷⁹⁾ in a review of 102 peripheral nerve injuries in 81 patients, found that of injuries below the elbow, 87% regained protective sensation, and 14% regained normal 2-point discrimination. Sakellarides ⁽⁷⁵⁾ found that after closed peripheral nerve injuries above the elbow, return of function was delayed compared with such injuries below the elbow. Of patients treated surgically, 143 were followed for 12 months or more after neurorrhaphy. Recovery of good clinical function was found in 13 (27%) of 48 lesions

above the elbow and in 37 (39%) of 95 lesions below the elbow. Except for parts of the brachial plexus, useful function at times returns regardless of the level of injury if the critical limit of delay has not passed.

10.5 Condition of nerve ends

Sunderland ⁽¹⁾ stressed the importance of the condition of the nerve ends at the time of neurorrhaphy. He suggested that meticulous handling of the nerve ends, asepsis, care with nerve mobilization, preservation of neural blood supply, avoidance of tension, and provision of a suitable bed with minimal scar all exert favorable influences on nerve regeneration. Distal stump shrinkage has been found maximal at about 4 months, leaving the distal fascicular cross-sectional area diminished to 30% to 40% of normal size. Intra-neural plexus formation and fascicular dispersal make accurate fascicular alignment and appropriate axonal regeneration more difficult. Edshage ⁽⁸⁰⁾ showed that a neurorrhaphy with a satisfactory external appearance is no guarantee of optimal internal fascicular alignment. Fascicular malalignment was a common finding in his specimens taken from human nerve repairs. He used, in addition to a special miter box for nerve trimming, a variety of special knives and scissors designed to ensure satisfactory fascicular identification during neurorrhaphy. It is generally agreed that the nerve ends should be prepared in such a way that a satisfactory fascicular pattern is apparent in the proximal and distal stumps. No scar, foreign material, or necrotic tissue should be allowed to remain around the ends to interfere with axonal regeneration. Sometimes resection of the nerve ends so that satisfactory fasciculi are exposed leaves a gap that cannot be closed by end-to-end repair. As noted previously, clinical and experimental evidence indicates that excessive tension on the neurorrhaphy at the time of repair and when an acutely flexed limb is mobilized later causes excessive intra-neural fibrosis. These findings and the promising results achieved after the interfascicular nerve grafting technique advocated by Millesi ⁽³²⁾ and by Millesi, Meissl, and Berger ⁽³³⁾ suggested that such a technique is preferable to repair of nerves under too much tension or with limbs in acutely flexed or awkward positions.

10.6 Type of nerve

The type of nerve injured (pure motor, pure sensory, sudomotor, or mixed) has same bearing on outcome. Repair of a pure motor or sensory nerve will lead to better results than mixed nerves, due to a decreased chance of fascicular mismatching (sensory axon growing into motor endoneurial tubes and vice versa). The specific prognosis for motor VS sensory recovery is more controversial.

10.7 Specific nerve involved

The specific peripheral nerve involved affects functional recovery. Radial nerve injuries are more likely to have functional motor return in the forearm than median or ulnar nerve lesions of similar level. As discussed before, if all other factors are comparable, a mixed motor-sensory nerve (e.g. ulnar) demonstrate inferior recovery compared to a primarily motor (e.g. radial) or sensory (e.g. digital) nerve.

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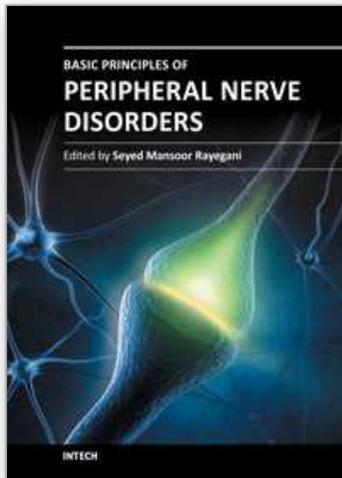
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Peripheral nerve disorders are comprising one of the major clinical topics in neuromusculoskeletal disorders. Sharp nerve injuries, chronic entrapment syndromes, and peripheral neuropathic processes can be classified in this common medical topic. Different aspects of these disorders including anatomy, physiology, pathophysiology, injury mechanisms, and different diagnostic and management methods need to be addressed when discussing this topic. The goal of preparing this book was to gather such pertinent chapters to cover these aspects.

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