Peripheral Nerve Injuries and Repair

Tim Hems

Contents

T. Hems
Department of Orthopaedic Surgery, The Victoria Infirmary, Glasgow, UK
e-mail: t.e.j.hems@doctors.org.uk

G. Bentley (ed.), *European Surgical Orthopaedics and Traumatology*,
DOI 10.1007/978-3-642-34746-7_82, © EFORT 2014

Abstract

Peripheral nerves may be injured in isolation or in combination with skeletal and other soft tissue structures. After treatment of trauma, impairment of nerve function often has an important influence on overall recovery and use of a limb. Management depends on early diagnosis of nerve injury based primarily on history and examination findings and supported by investigations. Minor compression or stretch injuries to nerves may recover spontaneously. Injuries where nerves have been transected or are under continuing compression will not recover. Surgery is required for decompression or repair. Early repair gives better results in most circumstances. Clean lacerations can be repaired by direct suture. Nerve grafts are required when a segment of nerve has been lost. Patterns of nerve injury which occur in association with specific skeletal injuries in the upper limb will be reviewed.

Keywords

Injuries • Peripheral Nerves • Repair

Introduction

Injury to peripheral nerves causes loss of motor function and sensation and may be complicated by severe pain. Although nerves have some capacity to regenerate, recovery is never perfect after transection of a nerve, even when the best surgical repair is performed. Therefore it is important to minimise damage to nerves, provide the best conditions to allow spontaneous recovery in favourable cases, and carry out optimal surgical repair as early as possible when necessary.

Anatomy of the Peripheral Nerve Trunk

Nerve impulses in the peripheral nerve are conducted by axons which extend from the central nervous system to a muscle or sense organ. Neurones consist of a cell body, associated dendrites and usually one axon. The cell bodies of the motor spinal nerves are located in the anterior horn of the spinal cord, while the cell bodies of the sensory neurones are located in the dorsal root ganglia. In order to remain viable an axon must stay connected to its cell body. Axons are surrounded by Schwann cells. In myelinated axons the Schwann cells form an insulating myelin sheath, the combination of the axon and myelin sheath being termed a nerve fibre. Each Schwann cell is associated with only one axon. The myelin sheath must be intact for conduction of nerve impulses in myelinated nerve fibres. In unmyelinated fibres a single Schwann cell wraps around several axons.

Nerve fibres are supported by connective tissue structures (Fig. 1). The Schwann cell basement membrane together with endoneurial collagen fibres forms an endoneurial tube. Large numbers of nerve fibres are gathered into fascicles surrounded by a connective tissue sheath called a perineurium. Fascicles are held together and the whole nerve trunk is ensheathed by another connective tissue layer called the epineurium. The fascicles divide and join along the length of the nerve forming a plexus within the nerve trunk. Hence the fibres destined for a particular branch of the nerve are mixed within the fascicles of the nerve trunk in the proximal part of the nerve and only become located in a separate fascicle or fascicular bundle a few centimetres before a branch is given off.

Response of a Nerve to Injury

Trauma to nerves may include laceration or blunt trauma such as stretch or compression. If the nerve is divided or blunt trauma is sufficient to disrupt axons, then nerve fibres in the distal part of the nerve undergo Wallerian degeneration (Fig. 2). In this process there is lysis of the axoplasm and fragmentation of the myelin sheaths leaving endoneurial tubes containing Schwann cells. In response to the injury there is some loss of neurones in the proximal segment of the nerve. Schwann cells react rapidly to injury and express factors important in supporting axonal regeneration. If the ends of a divided nerve are

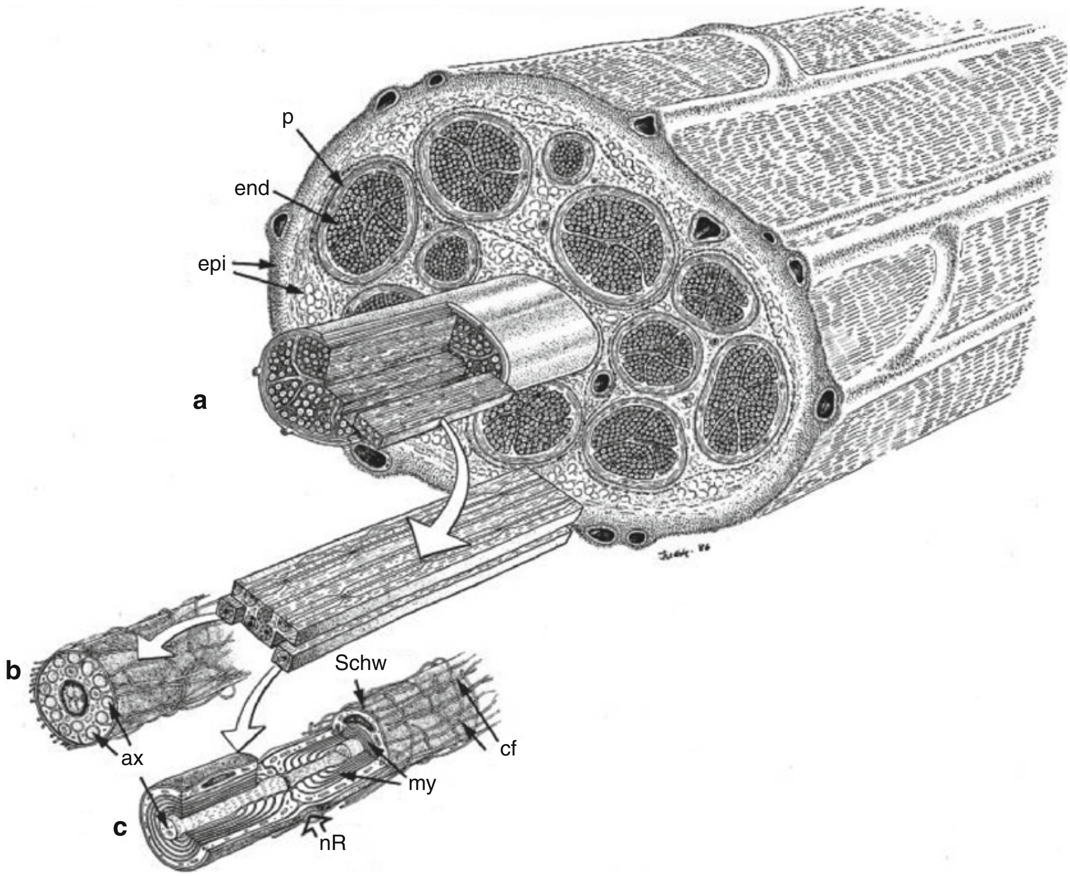

Fig. 1 Micro-anatomy of a peripheral nerve trunk. (**a**) Fascicles surrounded by perineurium (*p*) are bound together by loose connective tissue, the epineurium (*epi*). (**b**) and (**c**) show the appearance of unmyelinated and myelinated fibres respectively. Schw, Schwann cell; my, myelin sheath; ax, axon; nR, node of Ranvier (From Nerve Injury and Repair, G Lundborg, Churchill Livingstone, 1988. Reproduced with permission)

approximated, or if the connective tissue structure of the nerve remains intact, the remaining axons have a potential to regenerate into the endoneurial tubes of the distal segment. Schwann cells reform the myelin sheaths and the axons can make connections with target organs. Regeneration of axons proceeds slowly at only 1–2 mm per day in humans. The extent of damage to the supporting connective tissue layers of the nerve influences the quality of recovery and is hence the basis of classification of nerve injuries.

Classification of Nerve Injury

The most widely used classification of focal injury to peripheral nerves in the UK was described by Seddon [30] after a study of large numbers of casualties during the second world war. There are three types of injury of increasing severity (Table 1):

Neurapraxia
Axonotmesis
Neurotmesis

Neurapraxia (Nerve not Working)

Neurapraxia is a comparatively mild injury caused by moderate compression or stretch such as that caused by tourniquet or passage of a missile near to the nerve trunk. There is a block to conduction of nerve impulses across the damaged segment of the nerve. Neurapraxia may be termed "*local conduction block*". Nerve modalities are not equally

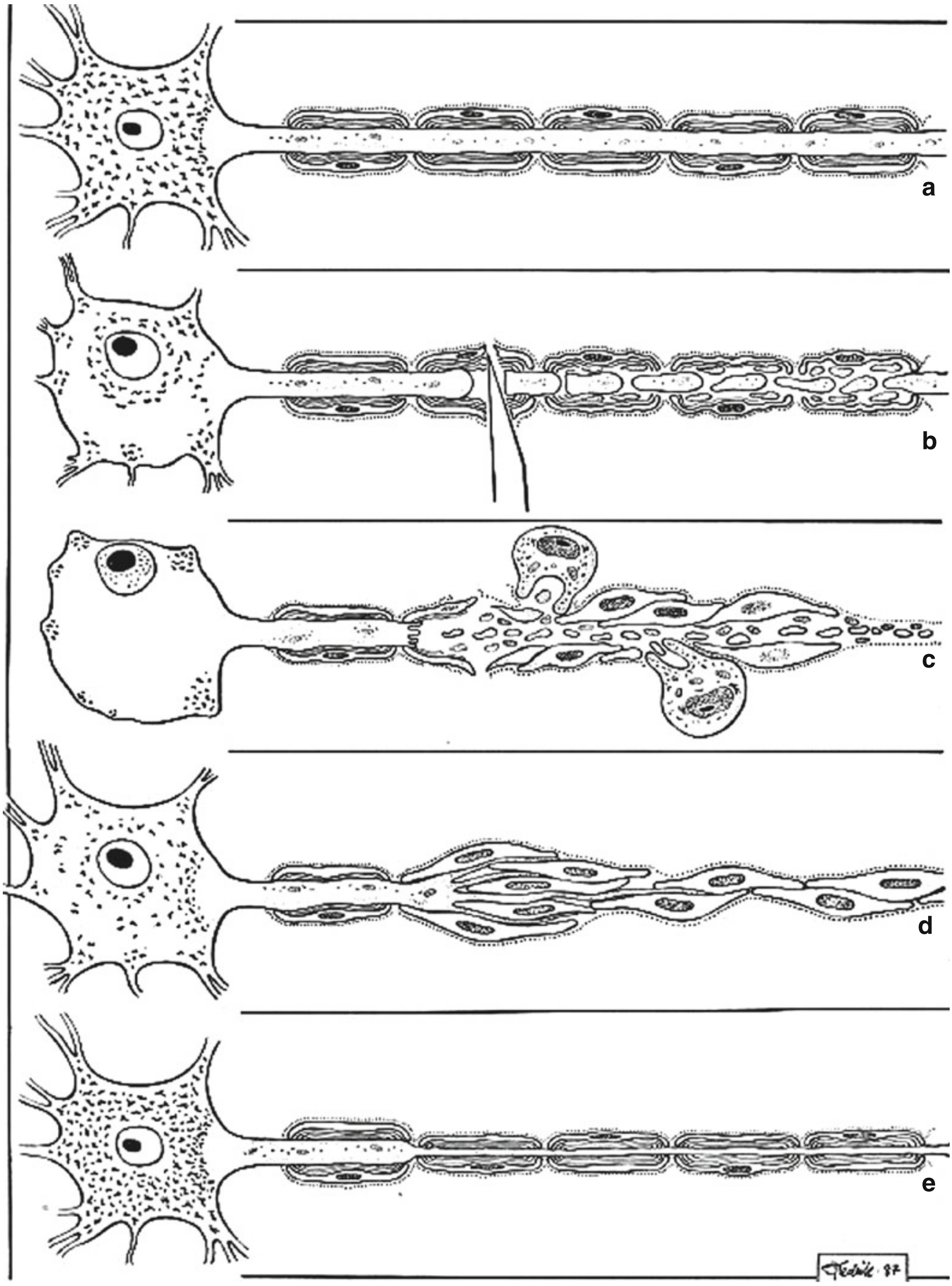

Fig. 2 The process of axonal degeneration and regeneration following injury to a myelinated peripheral nerve fibre. (**a**) Normal appearance. (**b**) Distal fragmentation of the axon and myelin after transection of the fibre (Wallerian degeneration). (**c**) In the distal segment Schwann cells proliferate. Macrophages and Schwann cells phagocytose debris material. (**d**) Sprouting occurs from the cut axonal stump. (**e**) Reconnection of the axon with the periphery. The myelin sheath is reformed around the regenerating axon (From Nerve Injury and Repair, G Lundborg, Churchill Livingstone, 1988. Reproduced with permission)

Table 1 Table showing the clinical features and neurophysiology findings of the three types of nerve injury defined in Seddon's classification. (From Table 42.2, Bailey & Love's Short Practice of Surgery, Editors: RCG Russell, NS Williams, CJK Bulstrode, Arnold, 24th Edition, 2004. Reproduced with permission.)

	Neurapraxia	Axonotmesis	Neurotmesis
Motor loss	Complete	Complete	Complete
Sensory Loss	Partial sparing	Complete	Complete
Autonomic function	Spared	Absent	Absent
Nerve conduction distal to injury	Present	Absent	Absent
Fibrillation on EMG	Absent	Present	Present
Recovery	Rapid	1 mm/day	1 mm/day
	Complete	Good	Always imperfect

affected. Motor paralysis is usually complete but sensory loss may be only partial. The axons are in-continuity and therefore Wallerian degeneration does not occur. Nerve conduction distal to the site of injury remains intact. Recovery is complete providing the cause is removed, for example, relief of any on-going compression on the nerve. However, the time for recovery may vary from days to several weeks. Recovery does not follow a proximal to distal pattern as occurs during axonal regeneration. Experimental work suggests that the conduction block results from demyelination of the nerve fibres in the damaged segment of the nerve.

Axonotmesis (Axons Divided)

Axonotmesis results from a more severe blunt injury to a nerve where the axons and their myelin sheaths are disrupted but the supporting connective tissue structures, including the endoneurial tubes, perineurium and epineurium, are still intact. Wallerian degeneration occurs distal to the zone of injury and hence conduction of the nerve is lost both at and distal to the injury. There is complete loss of motor, sensory and autonomic function on clinical examination. Radial nerve palsy associated with fracture of the humerus is usually an axonotmesis. Provided any on-going compression of the nerve is relieved, axons will regenerate distally at a rate of 1–2 mm per day. A Tinel's sign may be elicited at the level of the injury and will advance distally with time. The recovery of nerve function will follow a progression from proximal to distal. Axons are able to regenerate along the same endoneurial tube, in which they were previously located, and therefore connect with the same end-organ as before the injury. Hence recovery is usually good restoring near-normal sensory and motor function.

Neurotmesis (Whole Nerve Divided)

Neurotmesis is the term used to describe the state in which the nerve has been completely severed or so seriously disorganised that spontaneous recovery cannot occur. The axons and the supporting connective tissue layers of the nerve are disrupted and Wallerian degeneration occurs distal to the injury. Neurotmesis often occurs as a result of open injuries such as stab wounds but high energy traction can rupture a nerve and injection of noxious drugs or ischaemia can destroy a nerve.

If appropriate surgical repair is carried out then recovery may occur by axonal regeneration. The quality of recovery is never perfect after neurotmesis. This is probably as a result of failure of correct "re-wiring". Because the endoneurial tubes and fascicles have been disrupted even with the best surgical repair regenerating fibres connect with muscles or sense organs which they did not previously innervate. Repair of a nerve ruptured by severe traction, with damage over a long segment, is likely to have a worse outcome than repair of a sharp laceration.

Limitations of Seddon's Classification

The major limitation of Seddon's classification is that it does not distinguish between all grades

Table 2 Table showing the anatomical basis of Sunderland's and Seddon's classifications of nerve injuries. (+ = intact, – = severed). (From Table 42.1, Bailey & Love's Short Practice of Surgery, Editors: RCG Russell, NS Williams, CJK Bulstrode, Arnold, 24th Edition, 2004. Reproduced with permission.)

Sunderland grade	Axon	Endoneurial tube	Perineurium	Epineurium	Seddon group
1st degree	+	+	+	+	Neurapraxia
2nd degree	–	+	+	+	Axonotmesis
3rd degree	–	–	+	+	
4th degree	–	–	–	+	Neurotmesis
5th degree	–	–	–	–	Neurotmesis

of intraneural damage. Seddon himself noted variation in the appearance at operation and subsequent recovery, among nerves with injuries classified as axonotmesis. In some cases of "lesion-in-continuity" there is damage to the endoneurium and perineurium as well as disruption of the axons.

Sunderland's Classification

Sunderland [34] described five degrees of nerve injury on the basis of increasing anatomical disruption of the nerve trunk (Table 2). Although Seddon's classification is simpler, when exploring a damaged nerve Sunderland's classification is useful in its distinction between third and fourth degree injury. If the nerve fascicles are in-continuity and, hence the injury is not worse than third degree, then spontaneous recovery is possible. If the fascicles are disrupted indicating at least a fourth degree injury then spontaneous recovery will not occur and immediate repair should be considered. Some injuries are mixed with different parts of a nerve trunk being affected to a varying degree. This type of injury combines some or all of the five degrees of injury and the pattern of recovery will be mixed. This situation is not uncommon and has been classified as a "sixth degree injury" [23].

Summary of Classification

Although the distinction between nerve lesions where there is conduction block (neurapraxia) and axonal degeneration (axonotmesis and neurotmesis) is important in predicting prognosis, the most important issue in defining management is identifying injuries where the damage is sufficiently severe to preclude spontaneous recovery (Neurotmesis or Sunderland 4th or 5th degree). The latter require surgical repair to give any chance of recovery.

Anatomy of Arm Nerves

Median Nerve (C6, 7, 8, T1)

The median nerve is formed in the axilla from terminal branches of the medial and lateral cords of the brachial plexus. It runs along the medial aspect of the arm lying first anterior and then medial to the brachial artery. In the *antecubital* fossa it lies deep to the bicipital aponeurosis. The nerve then passes between the origins of the two heads of pronator teres to enter the deep *plane* of the forearm. Muscular branches to the forearm muscles are given off as it leaves the cubital fossa These include the anterior interosseous nerve which supplies the flexor pollicis longus, the flexor digitorum profundus to the index and middle fingers, and pronator quadratus. The main trunk of the median nerve passes beneath the fibrous arch of origin of the flexor digitorum superficialis and then runs down the forearm on the deep surface of this muscle. The median nerve emerges from behind the lateral edge of the flexor digitorum superficialis about 5 cm. proximal to the wrist and then lies between the flexor digitorum superficialis tendons and flexor carpi radialis. It then passes into the carpal canal deep to the flexor retinaculum to reach the palm of the hand. The palmar sensory branch given off above the wrist then passes through the sheath of the FCR tendon at the level of the wrist crease just medial to the tendon. As the median nerve emerges from the carpal canal it gives off branches to the radial two lumbrical muscles, the motor branch to the thenar muscles, and the digital nerves which supply sensation to the skin on the volar aspects of the thumb, index, middle and radial half of the ring fingers. The thenar motor branch leaves the radial side of the median nerve in the carpal canal and curves sharply back over the distal edge of the flexor retinaculum before entering the muscles. It may penetrate the flexor retinaculum more proximally.

Ulnar Nerve (C7, C8, T1)

The ulnar nerve is one of the terminal branches of the medial cord of the brachial plexus. It runs along the medial side of the arm slightly behind the brachial artery penetrating the medial intermuscular septum in the middle of the arm to descend on the medial side of the triceps muscle. At the elbow it passes through the cubital tunnel on the posterior aspect of the medial epicondyle of the humerus crossing the medial ligament of the elbow joint. It enters the forearm between the ulnar and humeral heads of flexor carpi ulnaris and then lies between the flexor carpi ulnaris and flexor digitorum profundus giving branches to both muscles. It is accompanied by the ulnar artery in the distal two-thirds of its course. Approximately 5 cm. above the wrist it gives off a dorsal sensory branch which passes dorsal to the flexor carpi ulnaris tendon and then runs subcutaneously over the ulnar styloid to the dorsum of the hand where it supplies sensation on the dorsum of the ring and little fingers. The ulnar nerve enters the hand through Guyon's canal superficial to the flexor retinaculum and lateral to the pisiform. In the canal it divides into three branches one supplying the muscles of the hypothenar eminence, including the abductor digiti minimi and flexor digiti minimi. The deep motor branch passes round the hook of the hamate deep to the opponens digiti minimi and then radially across the anterior surface of the interosseous muscles, all of which it supplies as well as the ulnar two lumbricals, before ending in the substance of adductor pollicis. It is not uncommon that some of the thenar muscles are supplied by the deep branch of the ulnar nerve although it is rare for the abductor pollicis brevis to be innervated. The sensory branch runs distally on the surfaces of abductor digiti minimi dividing into digital nerves which supply the sensation on the palmar aspect of the little finger and the ulnar aspect of the ring finger.

Radial Nerve (C6, 7, 8, T1)

The radial nerve forms in the axilla as the main terminal branch of the posterior cord. It then runs behind the brachial artery and then passes with the brachial artery through the triangular space formed by the humeral shaft, the long head of triceps and the teres major to reach the spiral groove deep to the lateral head of the triceps muscle. It gives off branches to the long, medial, and lateral heads of triceps as well as the posterior cutaneous nerves of the arm. At the junction of the middle and distal thirds of the arm it pierces the lateral intermuscular septum to reach the anterior compartment of the arm where it runs in the groove between brachialis and brachioradialis. Branches to brachioradialis and the radial wrist extensors are given off at the elbow. It enters the forearm anterior to the lateral epicondyle and divides into superficial radial nerve and the posterior interosseous nerve. The superficial radial nerve runs distally on the lateral side of the forearm deep to brachioradialis before passing superficially on the radial side of the wrist to supply sensation on the dorsum of the thumb, index and middle fingers. The posterior interosseous nerve passes round the proximal radius in the substance of the supinator, supplying this muscle, and then divides to supply all the finger and thumb extensors and extensor carpi ulnaris.

Clinical Features of Nerve Injuries

Diagnosis of nerve injuries is based largely on history and examination. The mechanism of injury, including sharp or blunt trauma, is important. High velocity and open injuries cause more

Table 3 MRC classification of motor nerve dysfunction

Grade	Clinical features
M0	Complete paralysis
M1	Flicker of muscle activity
M2	Power insufficient to overcome gravity
M3	Movement against gravity
M4	Movement against resistance
M4+	Strong movement, but not normal
M5	Normal, full power

Table 4 MRC classification of sensory nerve dysfunction

Grade	Clinical features
S0	No sensation
S1	Deep pain sensation
S2	Skin touch, pain and thermal sensation, i.e., protective sensation
S3	S2 also with accurate localisation but deficient stereognosis. Cold sensitivity and hypersensitivity are often present
S3+	Object and texture recognition, but not normal sensation. Good but not normal, two-point discrimination
S4	Normal sensation

severe nerve injuries. The history should also establish if the deficit occurred immediately after injury or if onset was delayed. Associated skeletal and soft tissue injury should be noted.

Examination of the nerve function includes:

- Motor power
- Sensation
- Autonomic Function

It is useful to grade the level of nerve function particularly in monitoring recovery. Muscle power is usually graded using the Medical Research Council system (MRC) (Table 3). The grading system is widely used and easy to apply in the clinical situation although rather a coarse measure. A simple assessment of sensation as normal, altered, or absent is useful. Sensory function may also be graded using the MRC classification (Table 4). Two- point discrimination is particularly useful for assessing sensation in the hand following injury as it is an objective measurement and normality (Approximately 4 mm. on the finger pulps) indicates continuity of the nerve. Lack of sweating in the distribution of a nerve indicates loss of autonomic function. This is an objective sign which does not rely on co-operation of the patient.

Special Investigations

Neurophysiology

Clinical assessment is paramount in the diagnosis of nerve injury. However, some additional information may be obtained from neurophysiological studies or imaging.

It is necessary to wait at least 2–3 weeks after a nerve injury before neurophysiological investigations can be performed, therefore limiting their application in many injuries. Interpretation of the results relies on the experience and skill of the neurophysiologist. Two types of test are available:

1. *Nerve conduction studies*. These involve recording the presence, amplitude, and conduction velocity of motor and sensory nerve action potentials.
2. *Electromyography (EMG)*. This test involves inserting a recording electrode into a muscle and recording the muscle action potentials in response to voluntary activity. Abnormalities indicating denervation and re-innervation may be observed. The presence of fibrillation potentials is associated with degeneration of the axons innervating the muscle.

Neurophysiology tests can distinguish between non-degenerative nerve lesions (neurapraxia) or degenerative lesions (axonotmesis and neurotmesis). While complete axonotmesis and neurotmesis cannot be distinguished, detection of the presence of some intact fibres in a nerve in a mixed lesion implies that the nerve is in continuity.

Magnetic Resonance Imaging (MRI)

MRI is a useful method of imaging for peripheral nerve tumours [13] and defining avulsion of nerve roots in brachial plexus injuries [14]. The technique might be used to confirm continuity of a nerve affected by a closed injury. Normal nerves can be demonstrated on MRI but usually have similar signal to surrounding tissues. Filler et al. [6] reported a technique, termed magnetic resonance neurography, for enhancing neural tissue on images. Early results were promising in defining nerve pathology. However, our experience suggests that it is difficult to consistently demonstrate nerves on MRI, using the scanners available for routine clinical cases, particularly in a zone of injury where signals are altered by oedema and haemorrhage in surrounding tissues.

Some information may be obtained with MRI of the muscles innervated by a damaged nerve. Changes in the signal from skeletal muscles are seen as early as 2 weeks after denervation on T2-weighted or stir sequences [38]. Later wasting and fatty infiltration is seen on T1-weighted images [7]. However, the exact relationship between the early signal changes in muscles and the severity of nerve injury is not clear.

Ultrasound

Over recent years there has been improvement in ultrasound machines such that the resolution is now greater than MRI. This imaging modality is showing promise in assessment of nerve injuries with the potential to confirm continuity of a nerve or demonstrate rupture or entrapment. It may be possible to demonstrate fascicular disruption within a nerve trunk. Bodner et al. [3] reported useful results when assessing radial nerve injuries in association with fractures of the humerus. Ginn et al. [8] reported diagnosis of median nerve entrapment in the forearm.

Management of Nerve Injuries

Open Nerve Injuries

If there is evidence of a nerve injury on clinical examination associated with a wound then it should be assumed that the nerve is divided. Partial laceration of a nerve will cause partial loss of function in that nerve. Surgical exploration of the affected nerves should be carried out in most circumstances once life-threatening haemorrhage has been controlled and if the patient is fit for operation. When wounds occur in areas where important nerves are vulnerable to damage these nerves should normally be explored even in the absence of obvious neurological deficit. This applies to much of the flexor compartment of the forearm and the hand. Early repair of divided nerves is indicated in most circumstances providing appropriate surgical expertise is available. Nerve repair is usually carried out simultaneously with the repair of other structures including vessels and tendons, providing there is adequate soft tissue cover. There should be vascularised full-thickness skin cover over a nerve repair. In complex injuries local or distant flaps may be needed. If the skin cover cannot be achieved early then nerve repair should be delayed. Split skin graft does not provide adequate cover over a nerve repair (R Dunn, personal communication).

Closed Nerve Injuries

Management of closed injuries, where nerves have been subjected to stretch or compression, is difficult as the severity of the nerve injury may not be clear. Lesions in continuity, neurapraxia and axonotmesis, will recover spontaneously providing there is no ongoing compression. However surgical repair of the nerve is necessary if there is to be any recovery after neurotmesis. Results are substantially improved if this repair is carried out soon after the injury.

In general, an assessment of the probability that a nerve has been disrupted should be made based on history and clinical findings. In high energy injuries where neurotmesis is more likely, early exploration of the involved nerves should be carried out together with repair if necessary. If operation is being carried out in any case, for example, for fracture fixation, then affected nerves should be explored at the same time. In cases of low energy trauma it is reasonable to observe the nerve injury initially. However cases managed non-operatively should be monitored carefully. If there is not clear evidence of recovery after 2–3 months then surgical exploration should be considered without further delay. Neurophysiology may be helpful in making a decision in some cases.

On-Going Nerve Compression

Following injury nerves may be compressed by dislocated joints, fragments of a displaced fracture, or expanding haematoma. In such cases the onset of nerve palsies may be delayed after the injury. There is often severe pain. Dislocations and displaced fractures should be reduced as soon as possible and operative fixation of fractures is usually indicated. Haematoma sufficient to cause nerve compression is often associated with arterial injury. Emergency angiography, followed by decompression of the haematoma or false aneurysm, and repair of the vessel is necessary.

There are some sites, for example, the carpal tunnel, where nerves run in confined spaces and are more at risk of compression by local swelling. Acute carpal tunnel syndrome may develop following a variety of wrist injuries or surgery on the wrist. Emergency decompression is usually indicated.

Surgical Repair of Nerves

Anaesthesia

Although some nerve repairs distal in the upper limb can be carried out under local or regional anaesthesia, most cases require a general anaesthetic. This is essential if a long procedure is expected or it is possible that grafts may be needed from other limbs. If nerve stimulation is being used then neuromuscular blocking agents must be avoided.

Exposure

Since nerves are longitudinal structures in the limbs large exposures are usually needed to adequately display the nerve proximal and distal to the injury together with adjacent structures including blood vessels. In delayed cases it is important to identify the nerve in unscarred tissue proximally and distally and then trace it into the zone of injury. If a tourniquet can be used dissection is easier in a bloodless field. Magnification using loupes is necessary for most operations on nerves.

Intra-Operative Neurophysiology

The simplest and indeed the most useful intraoperative test is direct stimulation of a nerve and observation of the motor response. This can be performed with a disposable sterile battery-powered stimulator. A neurophysiology unit is required for more complex investigations. If there is no muscle response, recording nerve action potentials can be useful. Bipolar electrodes are placed on the surface of the nerve above and below the site of injury for stimulation and recording. The presence of a nerve action potential indicates that axons are regenerating across the damaged section of nerve but have not yet reached the target muscles.

The time from injury has to be considered when interpreting the response to stimulation. The distal segment of a degenerating nerve will continue to conduct for 2–4 days, which may give a falsely optimistic assessment of a lesion in continuity. If there is clear loss of continuity, stimulation of the distal stump can help define the functional topography of the nerve when early operation is performed. It should also be remembered that nerves stop conducting about 30 min after application of a tourniquet as a result of ischaemia.

Neurolysis

Neurolysis is a procedure where a nerve is freed from constriction or scarring. Release of a nerve from external compression such as in the carpal canal or a displaced fracture is termed external neurolysis. The epineurium is not opened. External neurolysis can be an effective procedure providing the compression does not recur. Internal neurolysis involves opening the epineurium and dissecting fascicles from interfascular scar tissue. This is not often useful as recurrence of scarring is almost inevitable.

Assessment of the Lesion in Continuity

If a damaged nerve is found to be in continuity at operation then it may be difficult to decide if spontaneous recovery is likely or whether a better result will be obtained with resection and nerve grafting. Demonstration of intact fascicles crossing the lesion suggests the injury is no worse than Sunderland 3rd degree and hence recovery is possible. The larger and firmer the swelling of the nerve the less likely recovery is to occur. If a double bulb swelling is found this suggests disruption of fascicles within the epineurium (Sunderland 4th degree).

If exploration is carried out at 2–3 months recording of nerve action potential is very useful. Kline and Hudson [19] have reported that the presence of an action potential across the lesion is strongly predicative of eventual spontaneous recovery of the nerve.

Requirements for Nerve Repair

Nerve repair requires a healthy well vascularised tissue bed. Adequate debridement should be performed so that the wound is free of contamination. Associated skeletal, vascular and tendon injuries should be addressed. There should be adequate soft tissue cover with full thickness vascularised skin.

Direct nerve repair can be carried out when a nerve has been cleanly divided and the ends can be approximated with little tension. However, if the nerve ends are ragged or the injury has been caused by blunt trauma it is necessary to trim the ends back until healthy nerve fascicles are seen. If there is a delay between injury and repair the nerve ends tend to retract and there is scarring. It is again necessary to trim nerve back to normal tissue. Where nerve tissue has been lost there will be a gap between the nerve ends. Some strategies are available to reduce tension and allow direct nerve repair. These include mobilisation of the nerve ends, re-routing or transposition of the nerve, and splinting joints in flexion. However, these manoeuvres seldom gain much length and excessive mobilisation may impair blood supply to the nerve. If there are associated fractures then bone shortening may be a good option, with several centimetres possible in the humerus and some shortening being routine in digital replantation. In the forearm the radius and ulna must be shortened by exactly the same amount and it is less well tolerated.

Direct Nerve Suture

Nerve repair should be carried out under magnification using loupes or a microscope to aid accurate alignment of the nerve and placement of sutures. Methods of repair include epineurial repair, group fascicular repair, and fascicular repair. In most circumstances epineurial repair is used. Group fascicular repair is possible at distal sites where the nerve is dividing in terminal branches (Fig. 3). It is important to orientate the nerve ends as accurately as possible. The pattern of nerve fascicles and blood vessels is used to guide alignment. If possible sutures are placed in adjacent tissue to the nerve, for example, mesoneurial attachments, in order to approximate the nerve ends and reduce tension during the repair. Monofilament non-absorbable sutures should be used. While 6/0 may be used for large nerves such the sciatic, 8/0 is used for median nerve in the forearm with 9 or 10/0 for digital nerves. Sufficient sutures are inserted to provide epineurial cover around the nerve bundles. Fibrin glue may be used as an adjunct to repair once orientating sutures have been placed.

Nerve Grafting

If direct nerve repair is not possible then the gap between the nerve ends has to be bridged with a nerve graft. This is a technique where a length

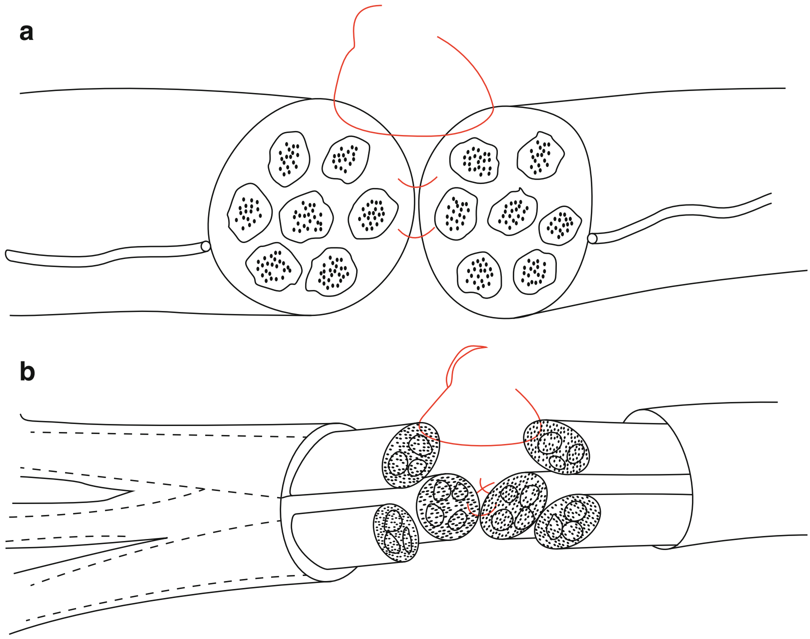

Fig. 3 (**a**) Epineurial and (**b**) fascicular suture repair of a nerve

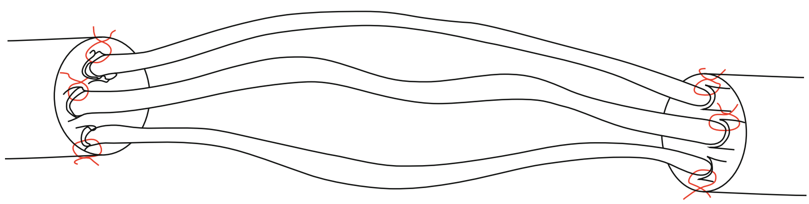

Fig. 4 Repair using cables of nerve graft to bridge a gap in a peripheral nerve. Grafts should always be longer than the defect being repaired so that there is no tension

of an expendable, usually sensory, nerve is used to reconstruct a major nerve trunk [26, 31]. Nerves used for grafts are thin nerves. They are cut up into a number of strands to build up a similar thickness to the trunk of the nerve being repaired (cable grafting, Fig. 4).

The most commonly used donor nerve is the sural nerve. However, nerves which can be used in the upper limb include the lateral cutaneous nerve of the forearm, the medial cutaneous nerve of the forearm, and the superficial sensory branch of the radial nerve. Use of these is particularly attractive if damage to the donor nerve has already occurred as a result of the initial injury. Function has then already been lost. The superficial radial nerve would not usually be used except when there has been disruption of the radial nerve more proximally.

The nerve stumps are prepared by serial section until normal-looking nerve tissue is seen. If a secondary repair is being carried out, once disruption of the nerve has been confirmed, it is not always necessary to resect the proximal and distal neuromas. A longitudinal incision may be made in the epineurium proximal and distal to the neuroma and healthy fascicles identified. By reducing mobilisation of the nerve ends, this technique reduces tension on the graft (Fig. 5).

The nerve grafts should be placed without tension being at least 10 % longer than the gap in the nerve. When possible equivalent fascicles in a nerve trunk should be matched with grafts. In reality this is usually only possible when a short segment of nerve has been lost and at more distal site. In this situation fascicular bundles may be

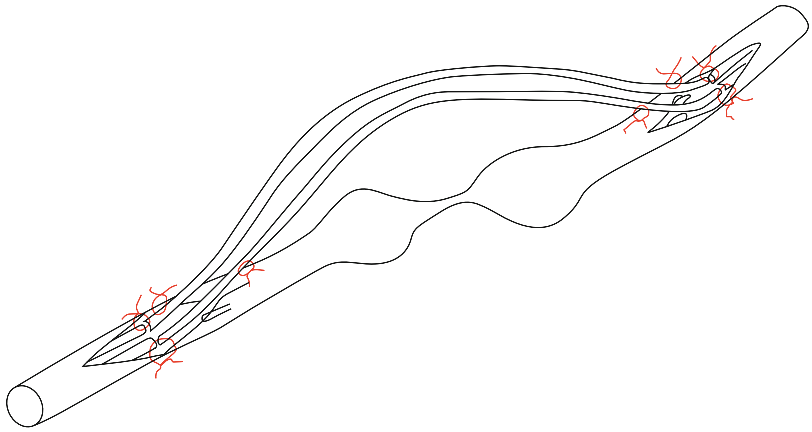

Fig. 5 Nerve grafts used to bridge a damaged segment of a nerve without resection of the neuroma

transected at different levels so that the suture lines for each bundles are separated.

Each strand of nerve graft is approximated with one or two lateral sutures (8/0 or 9/0) attaching the epineurium of the graft to the perinerium of the fascicular bundle or epineurium of the repaired nerve. In addition side-to-side sutures can be placed away from the point of co-aptation to reduce tension. Fibrin glue is often used to provide additional stability of the repair.

Technique of Harvesting Nerve Grafts

Sural Nerve

The sural nerve can be obtained from one or both lower limbs depending on the availability and length required. The nerve is harvested using a short incision behind the lateral malleolus and then 2 or 3 more proximal incisions. The nerve is easily identified first distally adjacent to the short saphenous vein. If gentle traction is then applied to the nerve its course can be palpated more proximally and incisions placed over it. Proximally the nerve is identified between the heads of gastrocnemius. It should always be divided at the level of the popliteal fossa so that the proximal stump and neuroma are deeply placed. The nerve can then be delivered from proximal to distal. Difficulty may be encountered if there is a communicating branch from the peroneal nerve and additional exposure may be needed in the proximal calf. Typically 45 cm. of graft can be obtained from each sural nerve in an adult. The patient should be warned of altered sensation on the lateral border of the foot. This rarely causes functional problems.

Lateral Cutaneous Nerve of Forearm (LCNF)

The LCNF is the terminal sensory branch of the musculocutaneous nerve which mostly receives nerve fibres from C5 and C6. About 25 cm. can be harvested from the mid arm level, below the motor branches, to mid-forearm, working through medial arm and anterior forearm incisions.

Superficial Radial Nerve (SRN)

The SRN is the terminal sensory branch of the radial nerve receiving fibres mainly from C6. A length of about 25 cm. can be obtained from the forearm. The nerve is identified and divided on the lateral side of the wrist. An incision is made on the anterior aspect of the proximal forearm and the radial nerve identified on the deep surface of brachioradialis. The SRN is separated from the posterior interosseous branch and can then be delivered into the proximal incision and removed.

Medial Cutaneous Nerve of Forearm (MCNF)

The MCNF receives fibres from C8 and T1 and can be harvested from the full length of the upper arm. Medial arm incisions are used but it can be traced up to its branch from the medial cord if the infraclavicular brachial plexus has been exposed giving as much as 30 cm of graft.

Alternative Techniques For Nerve Repair

Nerve Conduits

An alternative to suture of a nerve is to place the ends in a tube which may be a vein graft or synthetic material. Lundborg et al. [22] reported a prospective randomized trial comparing the use of silicone tubes for repair of the median and ulnar nerves with direct nerve suture. There was no significant difference between the two methods of repair. Silicone tubes have the disadvantage that they do not resorb and may require removal. Bioabsorbable nerve tubes are now available including polyglycolic acid, caprolactone, and collagen. Tubes may provide a simple method of nerve repair when microsurgical expertise and equipment is not available. In addition they appear to allow regeneration of a nerve across a short gap and therefore may be used for secondary repair or when there has been some loss of nerve so avoiding the use of a nerve graft. Mackinnon and Dellon [24] reported the use of PGA tubes for repair of gaps in digital nerves averaging 17 mm. Weber et al. [40] reported equal or superior results with PGA conduits for short or moderate defects in digital nerves compared to direct nerve suture or autologous nerve graft.

Bioabsorbable nerve conduits have gained popularity for repair of digital nerve particularly when tension free direct suture cannot be performed. Recent review concluded that the current evidence does not conclusively show that they are equivalent to existing techniques [4].

End-to-Side Repair

In end-to-side repair the distal stump of a damaged nerve is sutured into an epineurial/perineurial window of an adjacent nerve. Axons may sprout from the donor nerve into the recipient nerve. The technique proposed by Viterbo et al. [39] potentially allows some reinnervation when the proximal end of a damaged nerve is not available. Recent evidence suggests opening the perinerium will result in some limited collateral sprouting of sensory axons. However, regeneration of motor axons will only occur if there is axonal injury in the donor nerve [12].

The clinical applications of end-to-side repair are limited and remain to be fully defined.

Nerve Transfers

In some situations an alternative to repair of an injured nerve is to reinnervate the distal stump using a nerve transfer. A nerve transfer is a procedure where an expendable nerve is connected to a more important nerve. Nerve transfer may be possible when the proximal stump of a disrupted nerve in not available or the gap in the nerve is very long. Most nerve transfers are carried out to restore motor function. Donor nerves have to be selected with care to avoid any significant functional deficit resulting from the procedure. It should be appreciated that function after a nerve transfer cannot be independent of that of the donor nerve. Therefore there may be problems with coordination and co-contraction of muscle groups. If possible the donor nerve should innervate a synergistic muscle function. Although an interpositional nerve graft may be used between the donor and recipient nerves, results are generally better if a donor nerve is selected which allows tension free direct suture of the transfer.

Nerve transfers are most commonly used in reconstruction of brachial plexus injuries, for example, transfer of the distal accessory nerve to the suprascapular nerve. Some transfers have been described for reinnervation of forearm and hand nerves, for example, for treatment of radial nerve palsy. Tung and Mackinnon [36] recommended transfer of flexor digitorum superficialis and flexor carpi radialis branches of the median nerve to the extensor carpi radialis brevis nerve and posterior interosseous nerve respectively. The terminal branch of the anterior interosseous nerve may be transferred to the motor branch fascicle of the ulnar nerve to restore intrinsic function. Improved lateral pinch and grip strength has been reported by Novak and Mackinnon [27].

Sensory transfer may be performed using a nerve branch that supplies noncritical sensation to a hand or digital nerve. Transfer of the ulnar nerve branch to the fourth web to the median nerve branches to the thumb and first web space has been described for treatment of persisting median nerve palsy. While protective sensation may be obtained it is likely to be poorly localised. End-to-side repair may be used for sensory transfers therefore preserving sensation in the donor nerve territory [36].

Post-Operative Management

Usually some form of immobilisation is used to reduce tension on a nerve repair. However, the period of immobilisation is controversial. Currently for the median and ulnar nerves at wrist I recommend splinting the wrist at 30° flexion for 3 weeks and blocking extension for a further 3 weeks.

For digital nerves extension block splinting is traditional. However, nerve recovery appears to be similar for early and delayed mobilisation [5]. Therefore I use splintage only until skin sutures are removed. If there are associated fractures or tendon injuries then the appropriate rehabilitation program for that injury is followed.

A paralysed limb is prone to stiffness as a result of loss of muscle power, injury to other structures, and swelling. Therefore early routine physiotherapy instruction to mobilise joints is essential, while waiting for nerve recovery to occur.

Monitoring Recovery

After nerve repair or an injury in-continuity patients should be monitored clinically to check that nerve recovery is occurring at the expected rate. Hoffman-Tinel sign [16, 35] is an important sign in assessing early regeneration. Percussion is performed along the course of a nerve from distal to proximal. At the level of regenerating axons tingling is perceived in the sensory area of the nerve. The sign progresses distally at a rate of 1–2 mm per day after successful nerve repair or recovering *axonotmesis*. Neurophysiology does not usually add significant information when assessing recovery after repair. Muscle recovery and return of sensation can be recorded using the MRC scales.

General Factors Affecting Prognosis After Nerve Injury and Repair

The most important factor affecting prognosis for recovery is the severity of the injury to a nerve. Severity of injury is the basis of classifications described earlier in the chapter, with the outcome after lesions in-continuity, neurapraxia and axonotmesis, being superior to that after neurotmesis even when optimal surgical repair has been carried out. However it should be appreciated that there are varying grades of neurotmesis that will affect the quality of recovery after repair. A clean laceration has the best prognosis whereas high energy traction injuries or gunshot wounds, which damage a greater length of nerve will have a worse outcome. Associated vascular and soft tissue injury and fractures generally adversely affect outcome.

Other factors that affect the prognosis for recovery after nerve repair include.

Delay

Early repair of nerves gives the best results and this is one of the main factors that the surgeon can influence. Early operation for clean lacerations of nerves facilitates repair by direct suture. More complex injuries are also likely to benefit from repair within the first 1–2 weeks after injury before significant scarring has occurred, whenever the need for operation is clear, appropriate expertise is available, the patient is fit for surgery, and the condition of the wound satisfactory. While the evidence for the effect of delay is variable between individual nerves, in general, there is a deterioration in outcome if repair is delayed for a period greater than 3 months following injury.

There are deleterious effects of delay in repair at all levels of the neural pathways from cerebral cortex to end organs. Axonal degeneration is associated with death of some neurones in a nerve. Early repair reduces the neuronal loss [11] probably as a result of neurotrophic factors produced by cells in the distal segment as a response to the process of Wallerian degeneration. With time there is increased endoneurial fibrosis in the distal segment making it a less favourable environment for regenerating axons. Denervated muscles undergo rapid atrophy and increasing fibrosis with time. After 1–2 years the changes become irreversible so that recovery will not occur even if regenerating axons reach the muscle.

Age

Nerve recovery is generally less good with increasing age, with children sometimes recovering particularly well. However, there is no clear cut off for the age at which nerve repair is not worthwhile. Secondary consequences of paralysis in children may be worse because of associated growth abnormalities.

The Level of Injury

Usually proximal nerve injuries do worse than distal lesions, presumably because axons have a greater distance to regenerate. There do, however, appear to be some exceptions to this rule.

The Type of Nerve

The classic view is that mixed nerves recover worse than pure sensory or motor nerves. However, pure motor nerves do not truly exist as all motor nerves contain some afferent fibres from muscle spindles. Motor nerves that innervate large muscle groups, not requiring fine control, have a better prognosis following repair than motor nerves supplying smaller muscles which control fine movements, for example, the small muscles of the hand. While some sensory recovery is possible following nerve repair sensation never recovers to normal.

Injuries to Specific Nerves

Digital Nerves

Lacerations to digital nerves are common. Typically sensation and sweating are lost distal to the wound. There is often associated injury to digital arteries and tendons. Usually operation is recommended to confirm the extent of the injury and repair the damaged structures. Clean lacerations of digital nerves can be repaired by direct suture. If there has been damage to a longer segment of nerve, repair with a nerve graft can be considered but the morbidity from harvest of the nerve graft and the extent of the procedure should be weighed against the likely improvement in sensation as a result of the repair.

Birch et al. [2] reported only one excellent result in 102 digital nerve repairs, with 42 good, 35 fair, and 24 poor. Results were better in children with 17 of 27 gaining an excellent result. In a detailed study of 27 patients Goldie et al. [9] found that two-point discrimination was regained in 37 % of fingers but only 27 % of patients graded their overall result as excellent. 40 % complained of persistent hyperaesthesia for up to 2 years. None of the patients regained normal sensation. It has been the authors experience that normal two-point discrimination is rarely regained. It is difficult to exclude the possibility that much of the improvement in sensation after repair of a single digital nerve is the result of crossover from the adjacent nerve territory.

Lacerations of the Median and Ulnar Nerves

The median and ulnar nerves are most commonly damaged by lacerations on the volar aspect of the wrist and distal forearm. The neurological function should be carefully evaluated distal to the injury. Lacerations in the palm of the hand may cause division of individual branches. Isolated injury to the deep motor branch of the ulnar nerve produces a particularly subtle deficit,

Table 5 Method of grading results of repair of the median and ulnar nerves in the forearm [1]

Grade	Motor	Sensory
Excellent	Power, MRC 5	Function indistinguishable from normal hand
	No wasting or deformity	Good stereognosis, no hypersensitivity
	No trophic changes	2PD equivalent to uninjured digits
Good	Power, MRC 4–5	Accurate speedy localisation. Can recognise texture or objects. Minor cold sensitivity and hypersensitivity. 2PD < 8 mm at tips of fingers
	Abolition of paralytic deformity	
	Minimal pulp wasting	
Fair	MRC 3 or more	Accurate localisation to digit. No stereognosis. 2PD > 8 mm. Significant cold sensitivity and hypersensitivity
	Some sweating	
	Pulp wasted	
Poor	MRC 3 or less	No sensation
	No sweating	Severe cold sensitivity or hypersensitivity
	Trophic changes	

Table 6 Results of repair of: **a**. 119 median nerves and **b**. 145 ulnar nerves, in tidy wounds from distal wrist crease to elbow crease (Adults, age 16–65) ([2])

a.				
	Repair type (number of cases)			
Outcome	Primary repair	Delayed suture	Graft	Total
Excellent	5	1	0	6
Good	27	9	12	48
Fair	12	14	25	51
Poor or Bad	2	7	5	14
Total	46	31	42	119
b.				
	Repair type (number of cases)			
Outcome	Primary repair	Delayed suture	Graft	Total
Excellent	8	1	2	11
Good	25	6	22	53
Fair	13	16	27	56
Poor or Bad	0	9	16	25
Total	46	32	67	145

where the interossei and adductor pollicis are paralysed but the hypothenar muscles are preserved and there is no sensory loss. Lacerations associated with any neurological deficit should be explored, and indeed most lacerations which penetrate the deep fascia or where there is evidence of tendon or vascular injury. The laceration will usually require extension to give adequate exposure. Distally extension in the midline into the carpal canal usually gives the best exposure of the median nerve and the ulnar nerve can also be exposed through the same incision if necessary. Most nerve lacerations can be repaired by direct suture using standard techniques or nerve grafts if there has been a more extensive injury.

Birch and Raji [1] reported a system for grading outcome for repair of the median and ulnar nerves (Table 5). Birch et al. [2] reported the results shown in Table 6a and b for the median and ulnar nerves. The authors experience has generally been less favourable. While correct localisation of sensation to each finger may occur, two point discrimination is rarely regained. For the median nerve there is a good chance of recovery in the thenar muscles but control is often impaired by the sensory deficit. Finger abduction and thumb adduction may achieve good power after ulnar nerve repair but finger adduction is rarely regained.

Lacerations of the Radial Nerve

Lacerations of superficial radial nerve may occur in the distal forearm. While surgical repair is possible the recovery of sensation is often poor with hyperaesthesia, and associated with a tender neuroma at the site of repair. The alternative is to bury the proximal stump in muscle deep in the forearm.

Lacerations of the posterior interosseous nerve occur typically as a result of a fall on to glass causing a laceration on the dorsum of the proximal forearm. There is resulting loss of finger and thumb extension with preservation of the radial wrist extensors. Exploration and repair of the nerve is indicated. Recovery is often rewarding as this is a largely motor nerve close to its target muscles, Shergill et al. [33] reporting 16 good results in 18 repairs carried out either early or delayed.

Nerve Injury Associated with Fractures and Dislocations of the Wrist

The following nerve problems may arise in association with fractures of the distal radius, dislocations of the wrist, or carpal injuries:

- Direct median nerve injury
- Acute carpal tunnel syndrome (CTS)
- Superficial radial nerve injury.

Direct median nerve injury is associated with displaced fractures and high energy trauma (Fig. 6). Median nerve deficit is present at the time of presentation but it may not be possible to differentiate from acute CTS. Management involves emergency reduction of the fracture or dislocation to relieve ongoing pressure on the nerve, followed by emergency carpal tunnel release and nerve exploration. Operative fixation of the fracture is usually necessary. Localised bruising of the median nerve is often seen at exploration but loss of continuity is very rare. Recovery is variable. Unfortunately long term sensory impairment and pain in the median nerve distribution is not unusual.

CTS results from swelling or haematoma in the carpal canal. If evident early after injury it is an indication for emergency reduction of the fracture. Carpal tunnel release should be performed if pain and sensory impairment do not improve within a few hours. The operative release should extend proximal to the wrist skin crease. CTS may develop later in management of a fracture. Urgent carpal tunnel release should be performed unless there is improvement with simple measures including elevation and splitting the cast.

Although the superficial radial nerve is not often damaged by the injury it is vulnerable to damage during treatment of wrist trauma as a result of insertion of K-wires, external fixator pins and bone plates or from pressure inside a cast. Although the area of sensation supplied by the superficial radial nerve is less important than the median and ulnar nerves, damage to this nerve contributes to ongoing pain. If possible incisions should be avoided over the course of the nerve, but if necessary, then adequate incisions should be used to visualise the nerve when inserting or removing pins and wires.

Nerve Injury Associated with Fractures and Dislocations of the Elbow

Nerve injuries may occur in association with fractures and dislocations of the elbow. The following scenarios will be considered in more detail:

- Nerve injuries associated with supracondylar fracture of the distal humerus
- Ulnar neuropathy after distal humeral fractures
- Nerve injuries associated with dislocation of the elbow including entrapment of the median nerve in the elbow after reduction of dislocation
- Posterior interosseous nerve palsy after radial head dislocation.

In supracondylar fractures of the humerus, which occur mainly in children, the distal humerus usually displaces posteriorly and the distal end of the shaft may directly injure the median nerve and or the brachial artery which lie anteriorly. Other nerves may also be affected by the injury or associated with treatment. Neurological complications are not uncommon with Louahem et al. [21] reporting 60 (28 %) in 20 children with displaced supracondylar fractures. Two nerves were affected in 6 patients. The median nerve was affected in 28 cases (the anterior interosseous branch only in 18), the ulnar nerve in 25 and the radial nerve in 13. All cases made a full recovery spontaneously or after neurolysis. Ramachandran et al. [28] reported on 37 neuropathies in 32 children with displaced supracondylar fracture of the humerus. The ulnar nerve was injured in 19, the median nerve in 10 and the radial nerve in 8 cases. Surgical exploration of the nerve was carried out in 10 children with neurolysis being carried out in 6 cases for entrapment in scar tissue or the fracture site. Excision of a neuroma and nerve grafting was necessary in four cases, 1 median, 1 ulnar, and 2 radial nerves. Ulnar nerve injury may also occur iatrogenically during medial percutaneous pin placement.

When assessing median nerve function it is important to differentiate loss of function in the anterior interosseous nerve only from complete median nerve palsy. Some injuries at the elbow cause stretch or compression predominantly of the anterior interosseous nerve and spontaneous recovery likely. Complete median nerve palsy is more likely to result from nerve transection or ongoing compression.

Ulnar neuropathy has been reported after operative treatment of distal humeral fractures

Fig. 6 Radiograph of a displaced fracture of the distal radius. The median nerve was contused on the bone spike on the volar aspect of the shaft of the radius

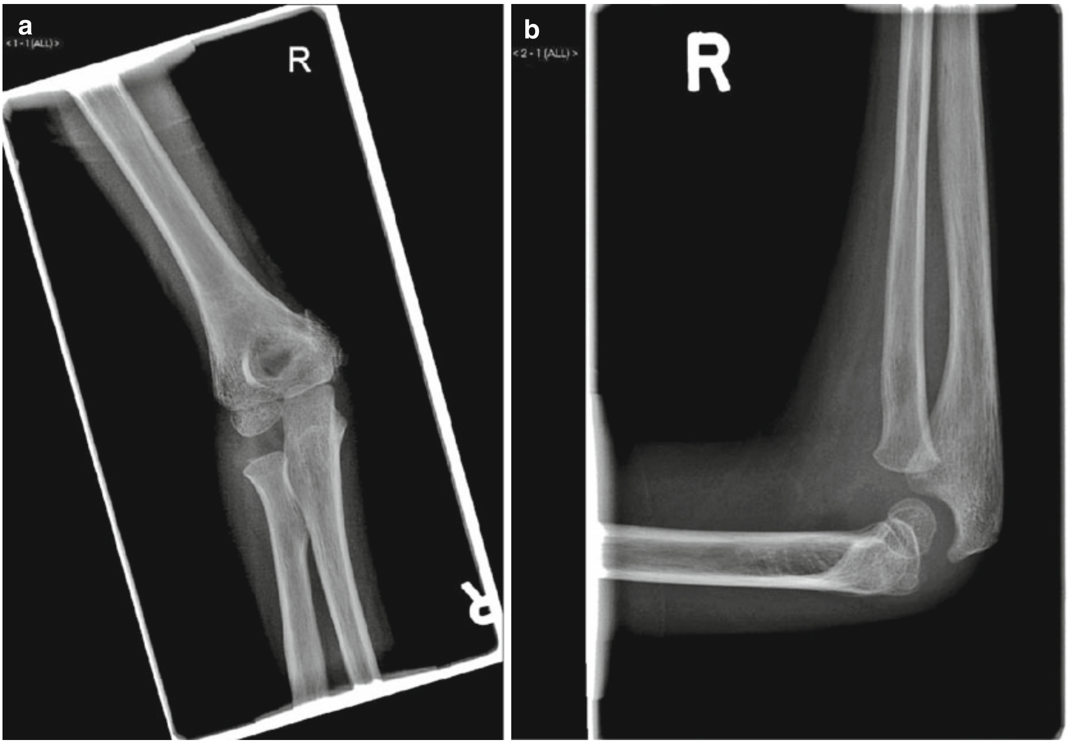

Fig. 7 Radiograph of a child's elbow after reduction of a dislocation. There is separation of the articular surfaces caused by entrapment of the median nerve in the joint

in adults. The cause is not always clear but manipulation of the nerve during surgery, inadequate release, impingement of bone fragments or metalwork, and post-operative fibrosis may all contribute. Good results have been reported when neurolysis of the ulnar nerve is carried out at the time of secondary reconstructive procedures [25].

Elbow Dislocation

The median, ulnar, and radial nerves can be injured by dislocation of the elbow. The radial nerve seems to be the least vulnerable. The median nerve can be injured by stretch or compression at the time of dislocation, during reduction by entrapment in the joint, or by expanding haematoma resulting from arterial injury. It is therefore important to examine the neurovascular function before and after manipulation. Entrapment usually results from the median nerve being displaced behind the medial epicondyle when the flexor origin and medial collateral ligament are ruptured or the medial epicondyle is fractured. In this position it may be entrapped in the joint at the time of reduction (Fig. 7, [10]). Most reported cases have occurred in children but one case in an adult has been managed in our unit. As well as the impaired nerve function entrapment is usually associated with severe pain. Operation is indicated as soon as possible to explore the nerve and extract it from the joint. In cases of arterial injury distal pulses will be absent although perfusion may be satisfactory. Emergency exploration, nerve decompression, and usually repair of the artery is necessary.

Elbow dislocation, fracture of the distal humerus, or humeral shaft fracture, caused by high energy trauma, may be associated with an extensive pattern of neurovascular injury where there is hyperextension of the upper limb [15]. This injury is characterised by rupture of the musculocutaneous nerve at the shoulder with additional injury to the median and/or the radial nerves. The ulnar nerve appears to be the least vulnerable to stretch probably because it lies behind the axis of the elbow joint.

Posterior Interosseous Nerve

The posterior interosseous nerve may be injured in association with dislocation of the radial head associated with fracture of the shaft of the ulna (Monteggia lesion) or olecranon. There is a high chance of spontaneous recovery. Jessing [18] recommended exploration and decompression of the nerve if there was no evidence of recovery after 8 weeks.

Radial Nerve Injury and Humeral Fractures

It is well known that the radial nerve may be injured in association with fractures of the shaft of the humerus. Shao et al. [32] reported an incidence or 11.8 %. The nerve may be subject to stretch or compression by fracture fragments. Injury may occur where the nerve is closely related to the humeral shaft in the spiral groove. Holstein and Lewis [17] reported an association with fractures at the junction of the middle and distal thirds of the humerus with entrapment of the nerve in the fracture where it passes though the lateral intermuscular septum. Transverse and spiral fractures are more likely to injure the nerve than comminuted or oblique. Nerve transection is associated with open fractures or complex upper limb trauma [29].

Clinical assessment should include establishing the mechanism of injury, whether the fracture is open or closed, and associated injuries. Typically examination reveals loss of function of the radial nerve below the triceps branches with absence of wrist, finger and thumb extension. Sensation is impaired on the dorso-radial aspect of the hand. Monitoring the advancement of Tinel's sign from the level of injury along the course of the nerve is important in assessing the progress of recovery.

As with other nerves the injury may be neurapraxia, with recovery occurring within in weeks, axonotmesis, with recovery taking months, or neurotmesis. Overall spontaneous recovery occurs in about 70 % of cases with a mean time to onset of recovery of 7 weeks (range 2 weeks to 6 months) [32]. Axonotmesis occurs in many cases.

Ultrasound has shown promise in demonstrating entrapment of nerve in the fracture or loss of continuity [3].

The difficulty in management lies in selecting the cases where operation is necessary to relieve entrapment of the nerve or repair rupture of the nerve. In general delay in these cases will adversely affect outcome. However, evidence suggests that a limited period of waiting before nerve exploration appears to have no effect on final recovery [32, 37].

In cases of low energy trauma where the fracture is being managed non-operatively I recommend observation in the first instance. After 3 months careful assessment is made to look for evidence of recovery and a neurophysiology assessment may be requested. Exploration is considered if there is no evidence of recovery. In cases where operative fracture management is selected, open fractures, associated vascular injury, or floating elbow, I recommend exploration and, when necessary, repair of the nerve at same time as fracture fixation. Nerve graft is almost always required for repair of a ruptured nerve. If neurology develops while the fracture is being managed non-operatively then the nerve is likely to be under continuing stretch or compression. Urgent exploration and fixation of fracture should be performed.

In delayed cases consideration should be given to a number of factors including whether the fracture has united, the age of the patient, and the speed functional improvement is likely to be regained after surgery. If tendon transfer is possible, the patient may be better served by this option, which will give earlier functional improvement compared with nerve repair.

Shergill et al. [33] reported the results of 242 repairs of the radial nerve. Ninety-one percent of nerve were grafted with a mean delay of 90 days (0–440). Violence of the injury was important in outcome. There were no good results if delay was more than 1 year. Overall there were 30% good results, 28% fair, and 42% failed. The outcomes may have been adversely affected by a high proportion of complex injuries with multiple nerves involved. Lee et al. [20] reported excellent or good results in 5 of 6 cases of radial nerve repair using grafts 9 cm or more in length after a mean delay of 6 months.

Nerve Injury Associated with Surgical and Medical Interventions

Unfortunately nerve injury sometimes occurs in association with medical or surgical procedures. A new nerve palsy following a procedure can be a disastrous complication and represents a difficult situation which is often poorly managed. It is of course important to document neurological function before a procedure so that it is clear if a change has occurred, although this can be difficult when dealing with acute limb trauma. Following a procedure it is equally important to check and document the function of nerves which may have been affected. If a new nerve palsy is identified after a procedure, then careful, urgent, and realistic assessment should be made. Judgement of the clinician who has performed the procedure may be affected by an emotional attachment to the case and unfounded optimism that serious nerve damage has not occurred. Therefore early involvement of another clinician, preferably with experience of nerve injury, is desirable.

The following factors should be considered:

- Was the nerve palsy apparent immediately after the procedure or was onset delayed? Often the patient will be able to give a useful history of events.
- Was the nerve seen and protected during the operation?
- Could the nerve have been divided during the procedure?
- Could the nerve be under continuing stretch or compression, for example, against an implant or expanding haematoma? Ongoing compression is often associated with severe pain and the impairment of nerve function may be progressive.

Investigations such as ultrasound to look for continuity of the nerve or compressing haematoma may be helpful. Neurophysiology tests cannot contribute to early decision making since useful results cannot be obtained until 2–3 weeks after injury.

In general there should be a low threshold for exploration of the affected nerve within days, before scarring has occurred, to confirm continuity and freedom from compression. Involvement of a surgeon experienced in nerve repair is desirable. This approach offers the best chance of recovery of the nerve. In many cases the patient and the surgeon can be reassured that recovery is likely. If the nerve has been divided then early repair can be performed. As with any surgical intervention, the potential risks and benefits of re-operation should be carefully considered and discussed with the patient. A second procedure may risk a new and serious complication such as introduction of infection to a joint replacement. The likelihood of useful recovery after repair of the nerve, if it is found to be divided, should be considered together with the effect of delay in intervention. Therefore in an elderly patient, who has a high risk from anaesthesia and a low chance of success from nerve repair, an active decision not to intervene may be justified.

References

1. Birch R, Raji ARM. Repair of median and ulnar nerves. Primary suture is best. J Bone Joint Surg. 1991;73-B:154–7.
2. Birch R, Bonney G, Wynn Parry CB. Surgical disorders of the peripheral nerves. Livingstone: Churchill; 1998.
3. Bodner G, et al. Radial nerve palsy associated with humeral shaft fracture: evaluation with US – initial experience. Radiology. 2001;219:811–6.
4. Cheng CJ. Synthetic nerve conduits for digital nerve reconstruction. J Hand Surg. 2009;34A:1718–21.
5. Clare TD, de Haviland Mee S, Belcher HJCR. Rehabilitation of digital nerve repair: is splinting necessary? J Hand Surg. 2004;29B:552–6.
6. Filler AG, Kliot M, Howe FA, et al. Application of magnetic resonance neurography in the evaluation of patients with peripheral nerve pathology. J Neurosurg. 1996;85:299–309.
7. Fleckenstein JL, Watumull D, Conner KE, et al. Denervated human skeletal muscle: MR imaging evaluation. Radiology. 1993;187:213–8.

8. Ginn SD, et al. Ultrasound in the diagnosis of median neuropathy in the forearm: case report. J Brachial Plexus and Peripheral Nerve Inj. 2007;2:23. doi:10.1186/1749-7221-2-23
9. Goldie BS, Coates CJ, Birch R. The long term result of digital nerve repair in no-man's land. J Hand Surg. 1992;17B:75–7.
10. Hallet J. Entrapment of the median nerve after dislocation of the elbow. A case report. J Bone Joint Surg. 1981;63-B:408–12.
11. Hart AM, Branstrom T, Wiberg M, Terenghi G. Primary sensory neurons and satellite cells after peripheral axotomy in the adult rat Time course of cell death and elimination. Exp Brain Res. 2002;142:308–18.
12. Hayashi A, Pannucci C, Moradzadeh A, Kawamura D, Magill C, Hunter D, Tong AY, Parsadanian A, Mackinnon S, Myckatyn TM. Axotomy or compression is required for axonal sprouting following end-to-side neurorrhaphy. Exp Neurol. 2008; 211:539–50.
13. Hems TEJ, Burge PD, Wilson DJ. The role of magnetic resonance imaging in the management of peripheral nerve tumours. J Hand Surg. 1997;22B:57–60.
14. Hems TEJ, Birch R, Carlstedt T. The role of magnetic resonance imaging in the management of traction injuries to the adult brachial plexus. J Hand Surg. 1999;24B:550–5.
15. Hems TEJ, Mahmood F. Injuries of the terminal branches of the Infraclavicular brachial plexus: patterns of injury, management, and outcome. J Bone Joint Surg Br Vol. 2012;94-B:799–804.
16. Hoffman P. Concerning a method for assessing the success of a nerve suture. Medizinische Klinik. 1915;13:359–60 (Translation: J Hand Surg. 2005; 30B:85–6).
17. Holstein A, Lewis GB. Fractures of the humerus with radial nerve paralysis. J Bone Joint Surg. 1963;45-A:1382–484.
18. Jessing P. Monteggia lesions and their complicating nerve damage. Acta Orthop Scand. 1975;46:601–9.
19. Kline DG, Hudson AR. Nerve injuries. Operative results for major nerve injuries, entrapments, and tumors. Philadelphia: WB Saunders; 1995.
20. Lee JH, et al. Sural nerve autografts for high radial nerve injury with 9 cm or greater defects. J Hand Surg. 2008;33A:83–6.
21. Louahem DM, Nebunescu A, Canavese F, Dimeglio A. Neurovascular complications and severe displacement in supraconylar humerus fractures in children: defensive or offensive strategy? J Pediatr Orthop B. 2006;2006(15):51–7.
22. Lundborg G, Rosen B, Dahlin L, Danielson N, Holmberg J. Repair of median and ulnar nerves in the human forearm: early results from a prospective, randomized, clinical study. J Hand Surg. 1997;22A:99–106.
23. Mackinnon SE, Dellon AL. Surgery of the peripheral nerve. New York: Thieme; 1988.
24. Mackinnon SE, Dellon AL. Clinical nerve reconstruction with bioabsorbable polyglycolic acid tube. Plast Reconstr Surg. 1990;85:419–24.
25. McKee MD, Jupiter JB, Bosse G, Goodman L. Outcome of ulnar neurolysis during post-traumatic reconstruction of the elbow. J Bone Joint Surg. 1998;80-B:100–5.
26. Millesi H, Meissl G, Berger A. The interfascicular nerve-grafting of the median and ulnar nerves. J Bone Joint Surg. 1972;54-A:727–50.
27. Novak CB, Mackinnon SE. Distal anterior interosseous nerve transfer to the deep branch of the ulnar nerve for reconsruction of high ulnar nerve injuries. J Reconstr Microsurg. 2002;18:459–64.
28. Ramachandran M, Birch R, Eastwood DM. Clinical outcome of nerve injuries associated with supracondylar fractures of the humerus in children. The experience of a specialist referral centre. J Bone Joint Surg. 2006;88-B:90–4.
29. Ring D, Chin K, Jupiter JB. Radial nerve palsy associated with high-energy humeral shaft fractures. J Hand Surg. 2004;29A:144–147.
30. Seddon HJ. A classification of nerve injuries. Br Med J. 1942;ii:237–9.
31. Seddon HJ. The use of autogenous grafts for repair of large gaps in peripheral nerves. Br J Surg. 1947;35:151–67.
32. Shao YC, et al. Radial nerve palsy associated with fractures of the haft of the humerus. A systematic review. J Bone Joint Surg. 2005;87-B:1647–52.
33. Shergill G, Bonney G, Munshi P, Birch R. The radial and posterior interosseous nerves: results of 260 repairs. J Bone Joint Surg. 2001;83-B:646–9.
34. Sunderland S. A classification of peripheral nerve injuries producing loss of function. Brain. 1951; 74:491–516.
35. Tinel J. "Tingling" signs with peripheral nerve injuries. La Presse Médicale. 1915;47:388–9 (Translation: J Hand Surg. 2005;30B:87–9).
36. Tung TH, Mackinnon SE. Nerve transfers: indications, techniques, and outcomes. J Hand Surg. 2010; 35A:332–41.
37. Verga M, et al. Delayed treatment of persistent radial nerve paralysis associated with fractures of the middle third of humerus: review and evaluation of the long-term results of 52 cases. J Hand Surg. 2007;32E:529–33.
38. Uetani M, Hayashi K, Matsunaga N, Imamura K, Ito N. Denervated skeletal muscle: MR imaging. Radiology. 1993;189:511–5.
39. Viterbo F, Tridade JC, Hoshino K, Mazzoni NA. End-to-side neurorrhaphy with removal of the epineurial sheath: an experimental study in rats. Plast Reconstr Surg. 1994;94:1038–47.
40. Weber R, Breidenbach WC, Brown RE, Jabaley ME, Mass DP. A randomized prospective study of polyglycolic acid conduits for digital nerve reonstruction in humans. Plast Reconstr Surg. 2000;106:1036–45.