

全国高等医药院校规划双语教材

Bailey & Love

外科学

SHORT PRACTICE OF SURGERY


第24版

原著 R.C.G. Russell
Norman S. Williams
Christopher J.K. Bulstrode

主编 陈孝平 刘允怡
主审 裘法祖 吴孟超

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神经外科、泌尿外科和
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LEARNING OBJECTIVES

- To understand the structure of a peripheral nerve and its response to injury
- To know the clinical features of nerve disorders
- To be familiar with the principles of management of open and closed injuries to peripheral nerves and of nerve compression syndromes

INTRODUCTION

The nervous system is divided into the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS consists of the brain, spinal cord and the first two cranial nerves, while the remaining cranial nerves and the spinal nerves constitute the PNS. The CNS and PNS have fundamentally different responses to injury, the CNS having little ability to regenerate, whereas the PNS has considerable potential for recovery if conditions are favourable. This chapter deals mainly with surgical disorders of the PNS. Injuries to peripheral nerves have a major effect on the functional outcome after trauma and therefore careful management from an early stage is important to obtain the best results possible.

PRINCIPLES OF PERIPHERAL NERVE SURGERY

Structure of the peripheral nerve trunk

The anatomy of a peripheral nerve trunk is shown in Fig. 57.1. Nerve impulses are conducted by axons. The nerve contains many axons that are supported by connective tissue structures. Neurones consist of a cell body, associated dendrites and usually one axon. In order to remain viable, an axon must be connected to its cell body. All axons are surrounded by Schwann cells. In myelinated fibres, the Schwann cells form an insulating sheath, each Schwann cell being associated with only one axon. Integrity of the myelin sheath is necessary for conduction of nerve impulses in myelinated fibres. Unmyelinated fibres are composed of several axons wrapped by a single Schwann cell. The Schwann cell basement membrane, together with endoneurial collagen fibres, forms the *endoneurial tube*. Large numbers of nerve fibres are gathered in fascicles surrounded by a connective tissue sheath called the *perineurium*. The fascicles are bound together and the whole trunk is ensheathed by a further connective tissue layer called the *epineurium*.

Theodor Schwann | 1810–1882. Professor of Anatomy, Liège, Belgium. Described the neurilemma in 1839.

Response of a nerve to injury

If trauma to a nerve is sufficient to disrupt axons then the distal part of the nerve undergoes Wallerian degeneration. In this process, there is lysis of the axoplasm and fragmentation of myelin sheaths, leaving an endoneurial tube containing Schwann cells. The axons in the proximal part of the nerve have the potential to regenerate into the endoneurial tubes of the distal segment and subsequently make connections with target organs. The regeneration proceeds slowly with axons growing at only 1–2 mm per day in humans. The extent of damage to the supporting connective tissue layers influences the quality of recovery and, hence, is the basis of the classification of nerve injuries.

Classification of nerve injuries

The most widely used classification of nerve injuries in the UK was described by Seddon (1942) after the study of a large number of battle casualties during the Second World War. There are three types of injury of increasing severity (Box 57.1):

- neurapraxia;
- axonotmesis;
- neurotmesis.

Box 57.1
Types of nerve injury

Neurapraxia	Axons are intact. Spontaneous recovery is complete
Axonotmesis	Axons divided. Connective tissue intact. Wallerian degeneration occurs. Axons then regenerate slowly
Neurotmesis	Whole nerve severed. Recovery may occur if cut ends are apposed

Augustus Volney Waller | 1816–1870. General practitioner, Kensington, London, England (1842–51), who subsequently worked as a physiologist in Bonn, Germany, Paris, France, Birmingham, UK, and Geneva, Switzerland.
Herbert John Seddon | 1903–1977. Professor of Orthopaedic Surgery, Royal National Orthopaedic Hospital, London, England.

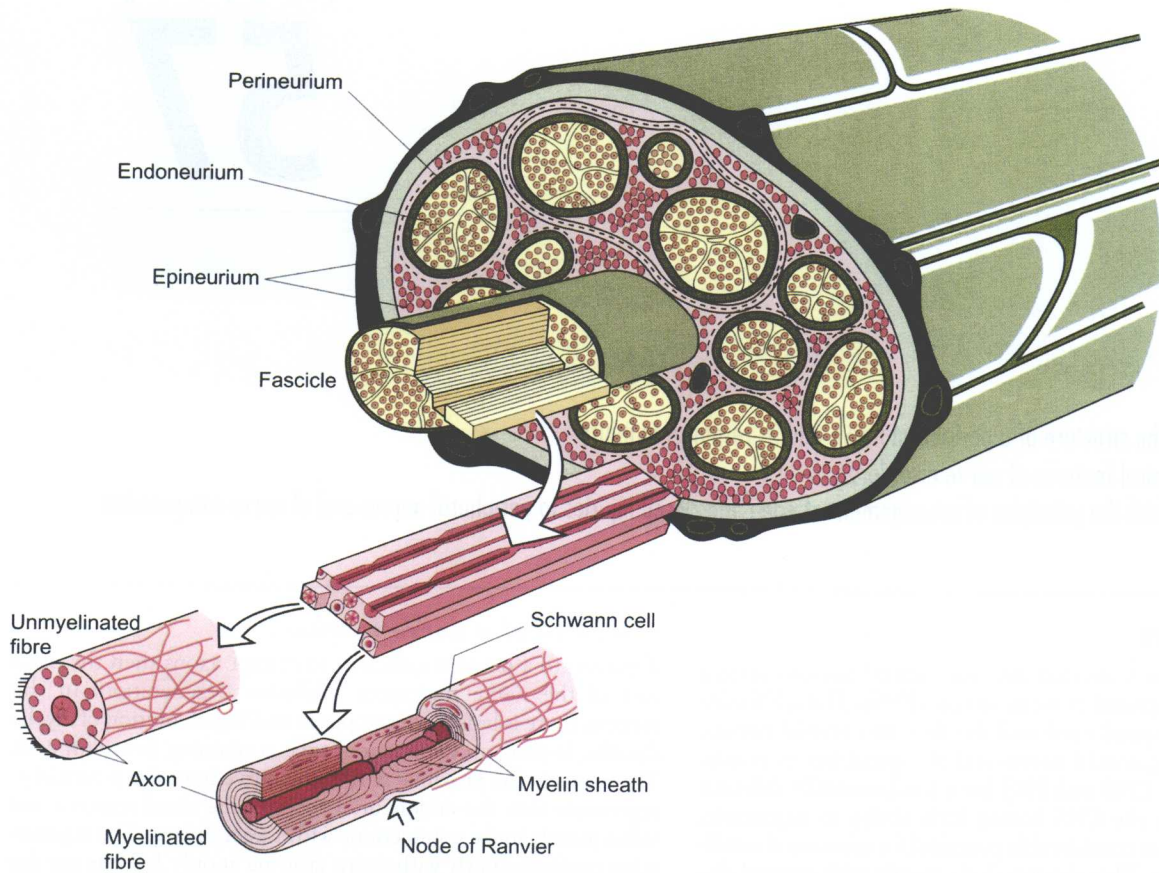


Figure 57.1 Microanatomy of a peripheral nerve trunk and its components. Fascicles surrounded by a multilaminated perineurium are embedded in a loose connective tissue, the epineurium. The outer layers of the epineurium are condensed into a sheath. The appearance of unmyelinated and myelinated fibres respectively is shown in detail. [Reproduced with permission from Lundborg, G. (1988) *Nerve Injury and Repair*. Churchill Livingstone, Edinburgh.]

Neurapraxia (nerve not working)

This is a local block to conduction of nerve impulses at a discrete area along the course of a nerve. The axons are in continuity and therefore Wallerian degeneration does not occur. Nerve conduction distal to the site of injury remains normal. Experimental work suggests that the conduction block results from localised demyelination of fibres in the damaged segment of nerve. Neurapraxia is a relatively mild injury typically caused by moderate compression such as that caused by a tourniquet, slight stretching, or the passage of a missile close to a nerve. Recovery is complete providing the cause is removed, but the time varies from days to several weeks.

Axonotmesis (axons divided)

This represents an anatomical disruption of the axons and their myelin sheaths. However, the supporting connective tissue structures including the endoneurial tubes, perineurium and epineurium, are still intact. Wallerian degeneration occurs distal to the site of injury and hence distal conduction is lost. Axonotmesis results from a more severe blow or stretch injury to a nerve. For example, radial nerve palsy associated with fracture of the humerus is usually an axonotmesis. Recovery occurs by

axon regeneration proceeding at a rate of 1–2 mm per day. Axons regenerate along the same endoneurial tube and therefore connect with the same end organ as before injury. The prognosis is good, restoring near normal sensory and motor function.

Neurotmesis (whole nerve divided)

This is the state in which the nerve has been completely severed or is so seriously disorganised that spontaneous recovery is not possible. The axons and the supporting connective tissue structures are disrupted and Wallerian degeneration occurs distal to the site of injury. Typically neurotmesis occurs as a result of an open injury such as a stab wound, but high-energy traction, injection of noxious drugs and ischaemia can destroy a nerve in this way.

If appropriate surgical repair is carried out then recovery may occur by axonal regeneration at a rate of 1–2 mm per day. In contrast with axonotmesis, the quality of recovery is never perfect after neurotmesis. This is probably the result of the failure of correct 'rewiring'. As the endoneurial tubes and other connective tissue structures have been disrupted, even with the best repair, regenerated nerve fibres connect with muscles or sensory organs which they did not previously innervate.

Table 57.1 Anatomical basis of Sunderland's and Seddon's classifications of nerve injuries

Sunderland grade	Axon	Endoneurial tube	Perineurium	Epineurium	Seddon group
First-degree	+	+	+	+	Neurapraxia
Second-degree	–	+	+	+	Axonotmesis
Third-degree	–	–	+	+	
Fourth-degree	–	–	–	+	Neurotmesis
Fifth-degree	–	–	–	–	Neurotmesis

+ , Intact; – , severed.

Sunderland's classification

Sunderland (1951) defined five degrees of nerve injury on the basis of increasing anatomical disruption of the nerve trunk (Table 57.1). Although Seddon's classification (Table 57.2) is simpler and more widely used in the UK, when exploring a damaged nerve, Sunderland's classification is useful in its distinction between third- and fourth-degree injuries. If the fascicles are in continuity (not worse than third-degree injury) then spontaneous recovery is possible, whereas if the fascicles are disrupted (fourth-degree injury) then spontaneous recovery will not occur and immediate nerve grafting may be considered.

Clinical features of nerve disorders

As with other medical and surgical problems, diagnosis of conditions affecting nerves is based upon history, examination and special investigations. The history should elucidate the nature of the deficit, its onset and severity and the causative factors. In the case of trauma, it is particularly important to establish the mechanism. High-velocity and open injuries produce more severe nerve injuries.

Examination of patients with nerve disorders includes:

- motor function;
- sensory function;
- autonomic function.

It is useful to *grade* the level of dysfunction of nerves, particularly in the assessment of recovery and the results of treatment (Box 57.2). The system widely used is the 1975 update of the 1954 Medical Research Council (MRC) classification (Tables 57.3 and 57.4). This grading system is good, and widely used, but

Table 57.2 Clinical features and neurophysiology findings of the three types of nerve injury defined in Seddon's classification

	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 per day	1 mm per day
	Complete	Good	Always imperfect

Sydney Sunderland | b. 1910, Melbourne. Published the 'bible' of peripheral nerve injuries in 1978.



Box 57.2

Diagnosis of nerve injury

Type of nerve damaged	Clinical finding
Motor	Weakness – measured using Medical Research Council (MRC) scale
Autonomic	Loss of sweating
Sensory	Two-point discrimination is very sensitive

Table 57.3 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 57.4 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

is still a rather coarse measure of muscle function and sensation. Loss of autonomic function is most easily shown by lack of sweating in the distribution of the nerve. This is a useful objective sign as it does not require a cooperative patient, as with testing sensation and muscle function.

The level of injury and the nerve injured can normally be deduced by careful physical examination and knowledge of the anatomical distribution of the nerves. Nonetheless, there is significant crossover in sensory function and also in some motor function, particularly when considering nerve roots (Table 57.5 and Fig. 57.2).

Clinical examination needs to be conducted with care, particularly with those patients who may have difficulty cooperating.

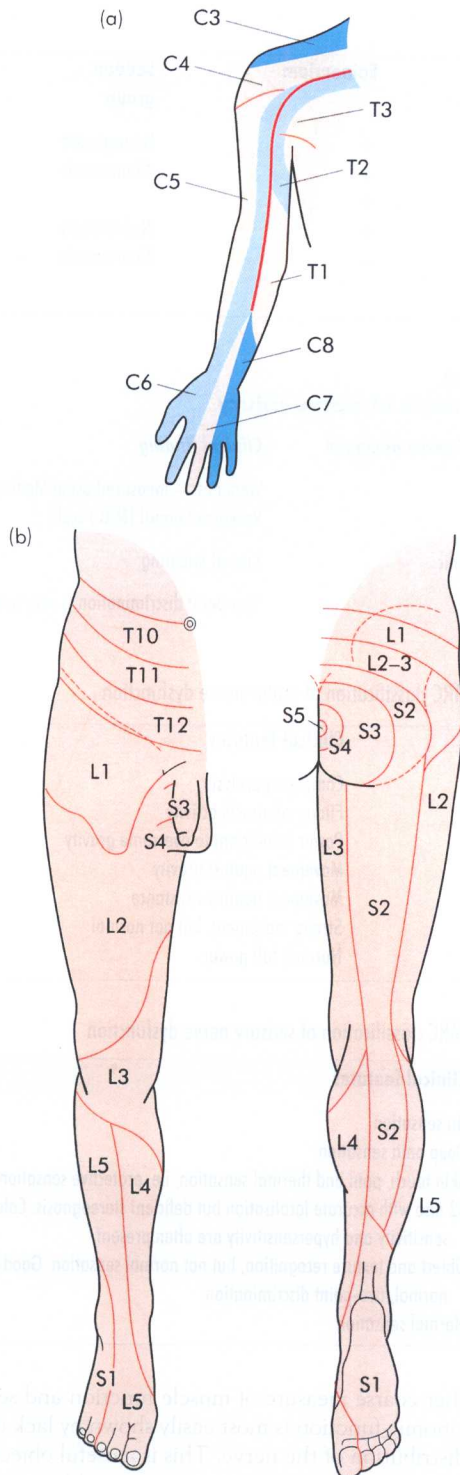


Figure 57.2 Dermatomes in the upper limb (a) and lower limb (b).

The findings should be accurately documented. Two-point discrimination is particularly useful for assessing sensation in the hand as it is an objective measurement and normality (approximately 4 mm on the finger pulps) excludes significant nerve injury. If there is any doubt, initial first aid should be carried out and the patient re-examined within 48 hours. Diagnosis can be difficult and severe nerve injuries are regularly missed.

Table 57.5 Myotomes

Arm	
Shoulder abduction	C5
Elbow flexion	C5/6
Elbow extension	C7/8
Finger flexion	C8
Small muscles of hand	T1
Leg	
Hip flexion	L2/3
Hip extension	L5/S1
Knee flexion	L5/S1
Knee extension	L3/4
Ankle inversion	L4
Ankle eversion	L5/S1
Plantar flexion	S1/2
Dorsiflexion of foot and toes	L4/5

Nerve dysfunction depends, in part, upon the grade of injury. With neurapraxia there is usually complete paralysis of the appropriate muscle groups but some sensation and autonomic function is preserved. In cases of axonotmesis and neurotmesis, there is complete loss of muscle power, sensation and autonomic function (Table 57.2).

Investigation

Although the most important assessment of nerve pathology is undoubtedly clinical, useful additional information can sometimes be obtained from neurophysiological studies or imaging.

Neurophysiological investigations require complex stimulation and recording apparatus. Interpretation of the results requires experience and is reliant upon the skill of the neurophysiologist. One must wait at least 2–3 weeks after a nerve injury before neurophysiological assessment can be performed. Two types of test are available:

- 1 *Nerve conduction studies.* These involve recording sensory or motor nerve action potentials and calculating the conduction velocity for given anatomical segments. Slowing of conduction, such as that resulting from compression neuropathy, can be identified.
- 2 *Electromyography (EMG).* In this test, muscle action potentials are recorded in response to voluntary activity. Abnormalities that are typical of denervation and reinnervation can be observed.

Using these tests, it is possible to distinguish between a nerve injury in which axons have not degenerated distal to the lesion (neurapraxia) and one in which Wallerian degeneration has occurred (axonotmesis or neurotmesis). Axonotmesis and neurotmesis cannot be distinguished.

Magnetic resonance imaging (MRI) is showing some promise in displaying peripheral nerve pathology and is likely to become more routine in the future. Currently its main application is in imaging cervical nerve roots after brachial plexus injuries.

Treatment

Open injuries

If there is clinical evidence of a nerve injury associated with a wound, then it should be assumed that the nerve is divided until

proven otherwise. It should be remembered that partial loss of function of a nerve may result from partial division of the nerve. *Surgical exploration of the nerves is advisable*, once life-threatening haemorrhage has been controlled and providing the general condition of the patient allows operation (Box 57.3). In addition, wounds in areas where important nerves are vulnerable to damage should be routinely explored even in the absence of obvious neurological deficit, for example the flexor compartment of the forearm. Early repair of any divided nerves is indicated in most circumstances. The operation should therefore be carried out by a surgeon with appropriate experience to carry out repair of nerves if necessary. If a vascular repair is required then nerve repair should normally be carried out at the same time. If the injury is in a site that is difficult to expose surgically, for example the brachial plexus, then transfer to a specialist unit should be arranged as soon as possible.

Box 57.3

Treatment of potential nerve injuries

- If a nerve's function is abnormal in association with a wound, it should be explored
- Important nerves in the area of a wound should always be explored
- Closed injuries should be explored if fractures are being fixed
- A nerve should be explored if there is no recovery after some months
- The earlier the repair (if needed) the better the prognosis

Closed injuries

Management of closed injuries in which nerves have been subjected to stretch or compression is more difficult as the severity of nerve injury may not be clear. Neurapraxia and axonotmesis will recover spontaneously providing the cause, for example compression, is removed. However, surgical repair of the nerve is necessary for there to be any chance of recovery after neurotmesis. Results are markedly better if this repair is carried out early after injury.

In high-energy injuries in which neurotmesis is more likely, the nerves involved should be explored as soon as possible and repair carried out if necessary. The procedure may be combined with fixation of any associated skeletal injury. In cases of low-energy injury, it is reasonable to observe the nerve injury initially, unless operation is being carried out for fracture fixation, in which case the opportunity should not be missed to confirm nerve continuity. Cases managed conservatively should be followed up carefully. If there is clear evidence of recovery after 2–3 months then conservative management can continue. If not, then surgical exploration should be considered without further delay. Neurophysiology may be helpful in making the decision in some cases as the tests may detect early recovery that is not evident clinically.

Surgical repair

The essence of a good surgical repair of a nerve is accurate coaptation of the nerve ends without tension in a healthy bed of tissue (Box 57.4). At operation the nerve ends are exposed, carefully avoiding further injury. If there has been a clean division of a nerve, then little dissection is usually required and *direct suture* can be carried out. However, if the nerve ends are ragged or the disruption has been caused by blunt trauma, it is necessary to trim the nerve back to healthy tissue with bulging nerve bundles. In

Box 57.4

Surgical repair of nerves

- Accurate coaption of the ends is crucial
- A microscope should be used to align fascicles and place sutures
- If ends are ragged, damaged tissue needs to be excised
- Grafts should be used to avoid repairs under tension
- Repair should be delayed only if the wound is contaminated or expertise is not available

delayed repairs, significant scarring and retraction of the nerve ends may have occurred, and it is again important to trim the nerve back to normal tissue. In these circumstances there will be a gap between the nerve ends. In the past, extensive mobilisation of some nerves was recommended to allow direct suture. However, it is seldom possible to get much length by this manoeuvre, and the repair is invariably under tension. Thus, if a significant gap is present it is usually better to perform *nerve grafting*. Occasionally, when a nerve injury is combined with a fracture, bone shortening may be justified to allow direct nerve suture.

Timing of nerve repair

Early nerve repair provides the best chance of satisfactory recovery and should be carried out providing that a well-trained surgeon and suitable equipment is available. Occasionally, if a wound is very contaminated, primary nerve repair may not be appropriate. In most circumstances it is possible to carry out early repair if sufficient wound debridement is carried out and plastic surgical expertise is available to provide flap cover of soft-tissue defects. If primary repair is not carried out then it is useful to apply one or two non-absorbable sutures, either to hold nerve ends together or to suture a nerve end to local soft tissue, thereby minimising retraction and aiding identification at later surgery.

Direct nerve suture

When repairing a nerve, a microscope should be used to aid accurate alignment of the nerve and placement of sutures, which range in the order of 6/0 for large nerves, such as the sciatic, 8/0 for the median nerve in the forearm and 9/0 or 10/0 for digital nerves. It is important to orientate the nerve ends with the correct rotation in order to minimise crossover during recovery. The pattern of nerve fascicles and surface blood vessels can be used as guides for alignment. Sutures are usually placed in the epineurium (*epineurial repair*). There is probably not any advantage in performing interfascicular repair except at distal sites where the nerve is dividing into terminal branches. Sufficient sutures are inserted to provide epineurial cover for all nerve bundles (Fig. 57.3).

Nerve grafting

When direct nerve suture is not possible, an interpositional nerve graft is necessary. This involves harvesting a length of an 'expendable' nerve trunk, such as the sural nerve or the medial cutaneous nerve of the forearm. These are long slender nerves that supply only small areas of sensation. The nerve graft is usually cut up so that a number of strands can be used to build up a similar thickness to that of the nerve trunk being repaired (see *Cable grafting*, Fig. 57.4).

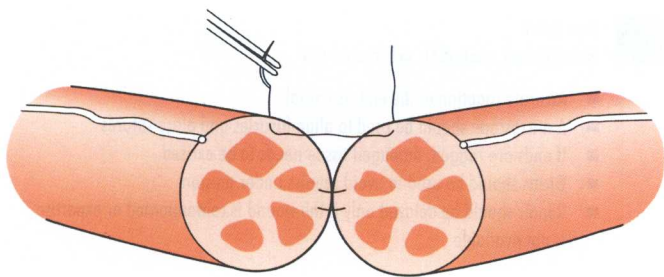


Figure 57.3 Direct epineurial suture of a nerve.

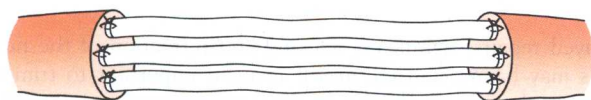


Figure 57.4 Cable grafting a large nerve trunk. The strands of nerve graft should connect matching groups of fascicles in the proximal and distal stumps if these can be identified.

Postoperative management

After wound closure, the limb is immobilised to minimise any tension on the suture line, with appropriate flexion of proximal and distal joints (Box 57.5). These are held in a cast for a minimum of 3 weeks. It may then be appropriate to progressively extend the proximal and distal joints, thus gradually restoring normal tension to the nerves. After 6 weeks, free mobilisation is permitted, assisted by appropriate physiotherapy.



Box 57.5

Nerve repair post operation

- The repair should be splinted for 3 weeks to avoid tension
- Physiotherapy is needed for joint mobility
- Regular monitoring is needed for signs of nerve recovery
- Re-exploration is needed if no recovery is noted
- Tendon transfers and joint fusions are needed for permanent nerve deficit

Follow-up management

After nerve repair, patients should have regular physiotherapy to maintain the passive range of movement in all joints prior to muscle recovery. In addition, patients should be monitored clinically to check that nerve recovery is occurring at the expected rate. Tinel's sign is useful in monitoring axon regeneration and should advance at about 1 mm per day (elicited by percussing over the course of the nerve). The patient feels paraesthesia in the distribution of the nerve. In the event of nerve recovery not progressing, re-exploration of the nerve may be justified.

Secondary reconstruction

If a nerve fails to recover, then in the long term other reconstructive procedures, including tendon transfers and joint fusions, may be indicated to improve function.

General factors affecting prognosis

The factors governing the prognosis of nerve repair are both general to all nerves and specific to certain nerves.

Age

Children recover much better than adults, and this is probably the most important prognostic factor. Nonetheless, the secondary consequences of paralysis in children may be worse because of the associated growth abnormalities. Age over 50 years is a particularly poor prognostic factor for proximal nerve injuries.

The severity of injury to the nerve

Seddon's classification of nerve injuries relates the severity of injury to the prognosis. However, there are varying grades of neurotmesis that affect the chance of recovery after repair. A clean-cut nerve injury has the best prognosis, whereas high-energy injuries, such as high-velocity gunshot wounds or severe traction injuries, damage a greater length of nerve and have a much worse prognosis.

The level of injury

In general, proximal lesions do worse than distal lesions although there do appear to be some exceptions to this rule.

The type of nerve

The classic teaching is that mixed nerves fare worse than pure sensory or motor nerves, although, of course, the latter do not truly exist, as all motor nerves have some afferents from muscle spindles. However, it is certainly true that motor nerves to large muscle groups not requiring fine control have a better prognosis than motor nerves supplying the small muscles of the hand (Box 57.6).



Box 57.6

Factors affecting recovery

Factor	Effect
Age	Children recover better than adults
Severity	Clean cuts do best
Level	Distal lesions do better than proximal
Type	Motor nerves to large muscles do best
Other injuries	Soft-tissue damage reduces healing potential
Delay	The earlier the repair, the better

Associated injuries

Nerve repairs with associated vascular injuries, soft-tissue damage and fractures are generally less successful. It is important that associated vessels and other structures are repaired as far as possible.

Delay

Early repair of nerves, that is within a few days of injury, gives the best results and is one of the main factors that the surgeon can influence.

Special types of nerve injury

Compression neuropathy

This is the term used to describe chronic dysfunction of a nerve as a result of local compression at some point along its course. Compression neuropathy is one of the most frequent single

conditions presenting to orthopaedic and hand clinics. The most common sites of compression are the median nerve at the carpal tunnel and the ulnar nerve at the cubital tunnel or Guyon's canal. These are anatomical sites where the nerve is surrounded by unyielding bone and ligaments. Sometimes there is an identifiable cause, such as tenosynovitis within the carpal tunnel, but, in many cases, there is no obvious cause. Compression of a nerve has a direct effect on the myelin sheaths as well as causing ischaemia of the nerve with consequent fibrosis. It also appears that damage results from loss of mobility of the nerve at the point of entrapment. The patient initially complains of pain and altered sensation in the distribution of the affected nerve. In more advanced cases, there is loss of sensory and motor function. Most compression neuropathies respond to surgical decompression of the affected nerve.

Irradiation

Radiation neuritis can occur up to 20 years after the radiation, the classic site being the infraclavicular brachial plexus following radiation for breast cancer but, fortunately, is rare. Characteristically, there is severe pain with some motor and sensory changes. The pain is very difficult to treat.

Injection injuries

Injection of toxic substances directly into a nerve does occur. This typically leads to a very intense fibrotic reaction in the nerve. Immediate exploration, incision of the epineurium and irrigation of the nerve trunk is recommended, but the outcome is poor.

Pain

The pain following nerve injuries or other nerve pathology can be of the most severe intractable type, leading to requests for amputation and even depression and suicide. The cause of this pain is poorly understood. Local non-operative treatment includes encouraging use and movement of the limb and transcutaneous electrical nerve stimulation (TENS), which works as a counter-stimulus to the pain (Box 57.7). In addition to simple systemic analgesics, anticonvulsant medication to suppress nerve excitability, such as carbamazepine or gabapentin, and tricyclic antidepressants, such as amitriptyline, can be useful. There are certain occasions when pain of nerve origin may be influenced by surgical intervention.

Box 57.7 Treatments for pain

Treatment	Action
Transcutaneous nerve stimulators	Act as a counter-stimulant
Anticonvulsants	Suppresses nerve excitability
Surgical exploration	Excision of neuroma
Surgical release	Treats pain from compression

Neuroma

If a nerve is wholly or partially divided, then axons attempting to regenerate form a neuroma on the nerve end. The neuroma may be exquisitely sensitive to any pressure, particularly if it is tethered in scar tissue or situated at a prominent point. Surgery may be helpful to restore continuity of the nerve by repair or grafting if possible or to move a neuroma to a less prominent position.

Nerve compression

Nerve compression may cause severe pain (neurostenalgia) as in compression neuropathies.

Complex regional pain syndrome type 1 (causalgia)

This is pain as a result of an injury (often partial) to a major nerve. The pain typically has an intense burning character. This pain tends to improve with nerve recovery, and therefore any surgical measures should be aimed at facilitating this.

Complex regional pain syndrome type 2 (reflex sympathetic dystrophy, algodystrophy)

This is a specific syndrome that can occur after trauma or surgery, when a cycle of pain and dysfunction is set up and leads to a chronic state associated with sympathetic overactivity. The limb involved becomes painful and tender to normal stimuli. This leads to disuse, stiffness and trophic changes. It is important to recognise the condition as early as possible and to break the vicious cycle. Analgesia and vigorous physiotherapy are the mainstays of treatment.

Avulsion of spinal nerve roots

Avulsion of spinal nerve roots from the spinal cord, such as may occur with traction injuries to the brachial plexus, is a complex pain syndrome involving damage to the spinal cord and loss of the normal afferent signals from the limb. Re-establishment of some neurological input by transfer of intercostal nerves may prove useful.

SPECIFIC NERVE INJURIES

Brachial plexus lesions

Damage to the brachial plexus (Box 57.8) is caused by:

- traction as a result of violent displacement of the shoulder girdle and cervical spine;
- open, penetrating injuries by knife or missile;
- operation in the area (for example, for removal of lymph nodes);
- malignant infiltration at the base of the neck, for example Pancoast syndrome.

Traction injury is particularly associated with road traffic accidents to motorcyclists. Any component of the plexus (Fig. 57.5) can sustain any grade of injury (neurapraxia, axonotmesis, neurotmesis). In addition, nerve roots may be avulsed from the spinal cord (preganglionic injury). Depending on the degree of injury, the paralysis varies from a completely flail and useless arm and hand to paralysis of groups of muscles and anaesthesia according to the roots affected. Injuries are usually divided into supraclavicular and

Box 57.8 Brachial plexus lesions

- Ruptures are most likely after high-energy trauma
- The most common injury is to the C5 and C6 roots
- Preganglionic injuries have a poor prognosis

Henry Khunrath Pancoast | 1875–1939. Professor of Radiology, University of Pennsylvania, Philadelphia, PA, USA.

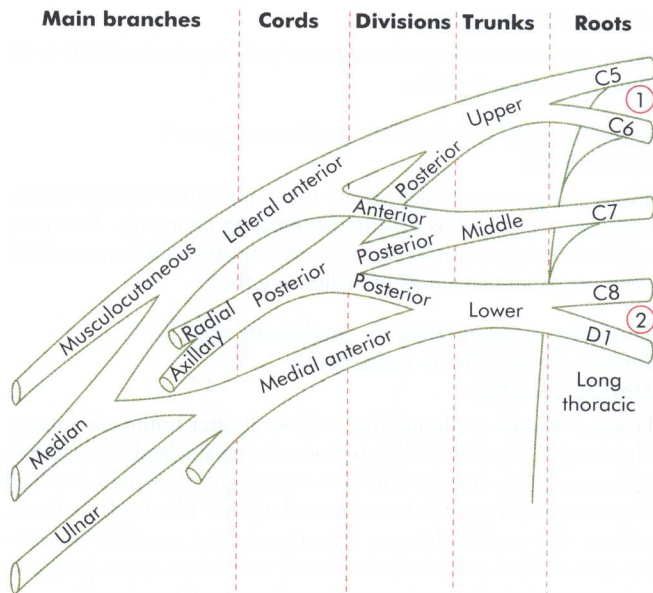


Figure 57.5 The brachial plexus, showing two classic sites of injury, ① and ②.

infraclavicular, although it is possible for there to be damage both above and below the clavicle.

The most common pattern of injury to the supraclavicular plexus is a lesion of the C5 and C6 roots and upper trunk (Erb–Duchenne palsy). There is loss of shoulder abduction and external rotation, elbow flexion and forearm supination. Sensation is absent on the outer aspect of the arm and hand. Klumpke described a lesion of the lower roots, but this is rare on its own. Injuries to all the roots are more common.

Management of brachial plexus injuries

It is important to establish the mechanism of injury as nerve ruptures are more likely after high-energy trauma. The nerves affected can largely be deduced from careful clinical examination. Signs of preganglionic injury include:

- Horner syndrome;
- paralysis of the thoracoscavular muscles (innervated by branches near the roots);
- swelling in the posterior triangle of the neck;
- severe pain in the anaesthetic arm.

Investigation includes radiographs of the chest and cervical spine to look for vertebral or rib fractures and to assess phrenic nerve function. In cases of supraclavicular injury, myelography, preferably combined with computerised tomography (CT) scanning or MRI, gives useful additional information on the integrity of the roots before surgery (Fig. 57.6). Neurophysiological assessment is not usually helpful until 2–3 weeks after injury.

Wilhelm Erb | 1840–1921. Professor of Medicine, Heidelberg, Germany.
 Guillaume Benjamine Amand Duchenne (Duchenne de Boulogne) | 1806–1875. Neurologist, successively in Boulogne and Paris, France, but never held a hospital appointment.
 Augusta Marie Dejerine-Klumpke | 1859–1927. Neurologist who practised in Paris, France.
 Johann Friedrich Horner | 1831–1886. Professor of Ophthalmology, Zurich, Switzerland. Described this syndrome in 1869.

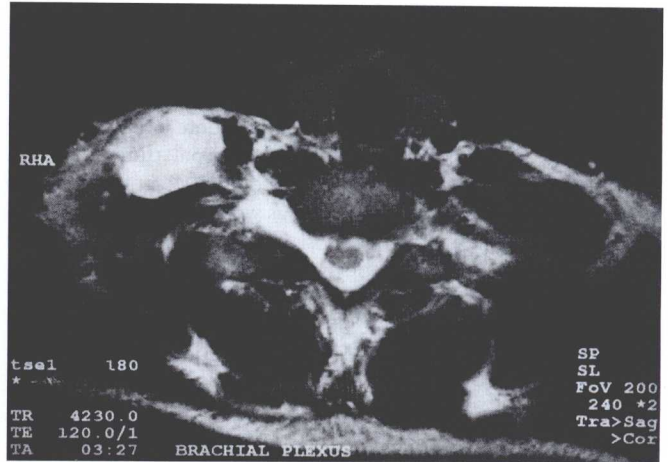


Figure 57.6 Magnetic resonance image (axial, T2 weighting) showing a right-sided pseudomeningocele resulting from traumatic avulsion of one of the roots of the brachial plexus.

Open injuries (e.g. stab wounds)

Any sharp injury to the brachial plexus should be explored as soon as possible and repaired using nerve grafts (Box 57.9).



Box 57.9

Management of brachial plexus lesions

- Associated vascular injuries: repair at the same time
- Sharp lacerations: repair with grafts
- Low-energy closed injuries: treat expectantly
- High-energy closed injuries: explore either in first days or within 3 months
- Obstetric injuries: treat initially with physiotherapy and monitor recovery

Injuries with associated arterial damage

Damage to the *subclavian or axillary vessels* may occur with injuries to the brachial plexus. Urgent vascular repair combined with exploration of the plexus by a specialist team is necessary.

Closed injuries resulting from low-energy trauma

As spontaneous recovery is likely, these cases should be managed non-operatively for 2–3 months and surgery considered only if improvement does not occur.

Closed injuries resulting from high-energy trauma

Referral to a specialist unit should be arranged as soon as possible. Operation within days of injury may be advantageous in some cases if the general condition of the patient allows but otherwise should not be delayed more than 3 months, as the results of repair are undoubtedly worse with increasing delay. At operation, the damage to the plexus is defined and nerve grafting is carried out for nerve ruptures. Until recently, repair of avulsed roots was thought not to be possible, but some recovery has now been obtained after repair of ventral (motor) roots within the spinal canal. Alternatively, nerve transfers are performed to restore the most important functions of avulsed roots as far as possible.

Obstetric brachial plexus palsy

Injury to the brachial plexus can result from traction on the shoulder girdle during birth. The upper roots are most commonly affected (Erb–Duchenne palsy). The prognosis is good with conservative management in most cases. Physiotherapy is necessary to prevent joint contractures, particularly shoulder, developing during the recovery period. The prognosis is improved by surgery to the brachial plexus in approximately 10% of cases. Operation is considered if there is:

- failure to regain elbow flexion by 3–6 months;
- complete paralysis of the limb.

Branches of the brachial plexus

Axillary or circumflex nerve

This is most commonly injured in association with dislocation of the shoulder joint. The deltoid muscle is paralysed and there is a patch of anaesthesia over the outer side of the arm. The majority of cases recover spontaneously but rupture of the nerve does sometimes occur and then recovery is only possible if nerve grafting is carried out.

The long thoracic nerve

The long thoracic nerve to serratus anterior (nerve of Bell) may be injured by operations on the breast or chest wall or is occasionally involved in neuropathies. Paralysis of serratus anterior results in ‘winging’ of the scapula and difficulty in elevating the arm above a right angle (Fig. 57.7).

Radial nerve

This nerve is most commonly injured in the radial groove in association with fracture of the shaft of the humerus or as a result of pressure as in ‘Saturday night’ palsy due to a heavy sleep with the arm over the sharp back of a chair. Clinical features include:

- **Motor.** Paralysis of brachioradialis, the wrist extensors, and extensor digitorum. It should be remembered that extension of the interphalangeal joints will still be present if the hand is supported because of the action of the lumbricals and interossei, which are inserted into the extensor expansions. In higher lesions, the triceps will also be affected.
- **Sensory.** Loss of sensation over the dorsum of the thumb and the first web space. In higher lesions, sensation is also lost on the dorsum of the forearm (Fig. 57.8).

Recovery of the radial nerve is usually good either after conservative management or repair if appropriate. If not, then good results can be obtained by tendon transfer (Box 57.10).

Median nerve

The median nerve is classically injured at the elbow or wrist. Injuries at the elbow are due to fractures of the distal humerus or dislocation of the elbow joint. Clinical features include:

- **Motor.** Paralysis of the pronators of the forearm and flexors of the wrist and fingers, with the exception of the flexor carpi ulnaris and the medial part of the flexor digitorum profundus. The index finger and thumb cannot be flexed at the interphalangeal joints, but flexion of the other fingers is performed by the portion of the flexor digitorum profundus, which is supplied by the ulnar nerve. The thenar muscles are paralysed with resulting loss of abduction and opposition of the thumb.



Figure 57.7 Winging of the scapula resulting from long thoracic nerve palsy; the patient is pushing against a wall.



Box 57.10 Upper limb nerve damage

Nerve	Problem
Axillary	Associated with dislocated shoulder. Most recover spontaneously
Long thoracic nerve	Injured during surgery to chest wall. Leads to winging of scapula
Radial nerve	Injured in fractured shaft of humerus. Usually recovers well
Median nerve	Can be injured or trapped at elbow or wrist. Has profound effect on sensation and mobility of hand
Ulnar nerve	Can be injured or trapped at elbow or wrist Needed for normal hand function

- **Sensory.** Sensation is lost over the palmar aspect of the thumb, index, middle, and the radial half of the ring fingers, as well as part of the palm.

Damage to the median nerve at the wrist is comparatively common as a result of lacerations, fractures of the distal radius or compression in the carpal canal (see Chapter 39). Clinical features include paralysis of the thenar muscles and loss of sensation on the palmar aspect of the radial three and a half fingers (Fig. 57.9).

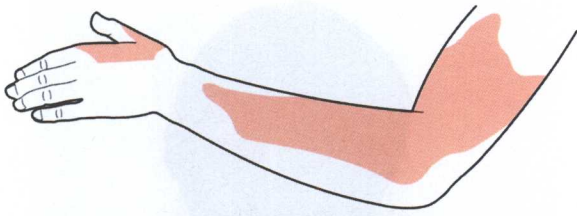


Figure 57.8 Areas of anaesthesia after a high complete lesion of the radial nerve. If damage is below the origin of posterior cutaneous nerve of the forearm then the sensory loss is limited to the dorsum of the first web space.

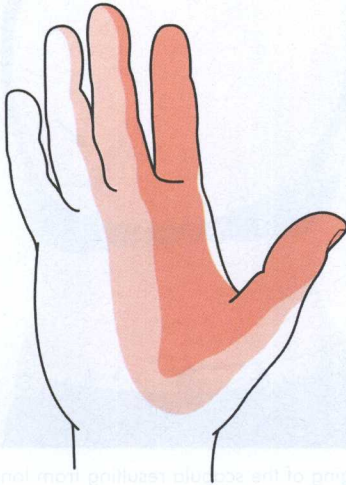


Figure 57.9 The area of sensory loss in the hand involving the radial three and a half digits after damage to the median nerve proximal to the wrist.

Ulnar nerve

The ulnar nerve is most commonly damaged by lacerations in the forearm or entrapment as it passes behind the medial epicondyle of the humerus, in which case decompression or anterior transposition may be indicated. Clinical features include:

- *Motor.* Paralysis of the small muscles of the hand with the exception of the thenar muscles and lateral two lumbricals. Inability to abduct and adduct the fingers results, and the patient cannot grip a piece of paper between the fingers (Fig. 57.10). Weakness of flexion of the metacarpophalangeal joints and extension of the interphalangeal joints results in a claw-type deformity. If the patient pinches a piece of paper between the thumb and the index finger, the distal phalanx of the thumb assumes a flexed position, as weakness of the adductor pollicis permits overaction of flexor pollicis longus (Froment's sign; Fig. 57.11). In longer-standing cases, muscle wasting will be evident in the interosseus spaces and along the medial border of the hand. Lesions proximal to the elbow also cause paralysis of the flexor carpi ulnaris and medial half of the flexor digitorum profundus.

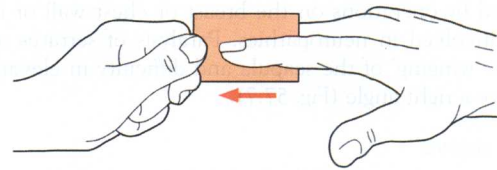


Figure 57.10 Ulnar nerve injury. Test for weakness of the interosseus muscles.



Figure 57.11 Froment's sign for right ulnar nerve paresis.

Jules Froment | 1878–1946. Professor of Clinical Medicine, Lyons, France.

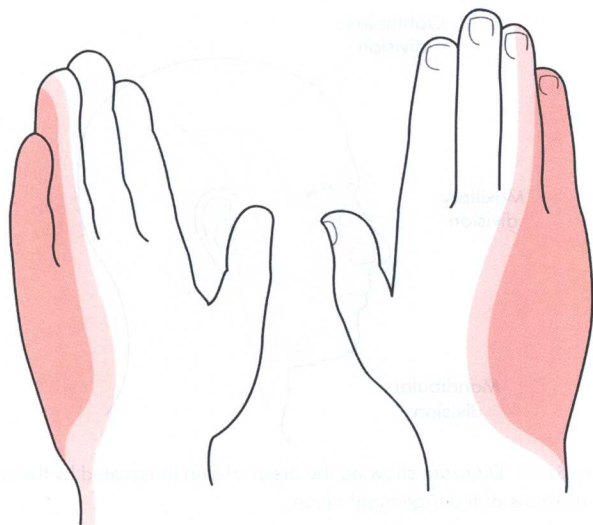


Figure 57.12 Ulnar nerve injury. The area of sensory loss affecting the ulnar one and a half digits.

- **Sensory.** Sensation is lost on the medial one and a half fingers (Fig. 57.12).

Lower limb nerves

Sciatic nerve

The sciatic nerve is occasionally injured by wounds, fractures of the pelvis, posterior dislocation of the hip, operation for hip replacement, or tumours. The prognosis for recovery is poor, particularly in proximal injuries. If the lesion is above the origin of branches to the hamstrings, the following features will be present:

- **Motor.** The flexors of the knee are paralysed but some degree of flexion is possible owing to the action of the sartorius and gracilis muscles. Complete paralysis exists below the knee and the pull of gravity therefore causes foot drop.
- **Sensory.** Complete loss below the knee, with the exception of the skin supplied by the saphenous nerve, i.e. the medial border of the foot.

Causalgia may complicate partial lesions of the sciatic nerve.

Common peroneal (lateral popliteal) nerve

Partial lesions of the sciatic nerve affect the peroneal division much more commonly than the tibial division. The common peroneal nerve itself is quite sensitive to injury by fractures or dislocations around the knee, pressure from plasters or splints, and operations around the knee. Complete lesions will cause:

- **Motor.** Complete paralysis of the extensor muscles of the ankle and toes and the peroneal muscles, with resulting foot drop and tendency to inversion of the ankle.
- **Sensory.** Anaesthesia of the dorsum of the foot and toes.

The prognosis depends on the severity of injury but is certainly poor even after repair for neurotmesis. Function is improved use of an ankle-foot orthosis or by tendon transfer at the ankle (Box 57.11).

Box 57.11

Lower limb nerve damage

Nerve	Problem
Sciatic nerve	Injured in posterior dislocation of hip and following hip surgery
Common peroneal nerve	Can be mimicked by partial lesions of sciatic nerve. Can be injured by pressure or blows around the knee. Prognosis is poor. Foot-drop splint will help function
Femoral nerve	Sometimes injured in stabbings. Prognosis is good if repaired

Femoral nerve

The femoral nerve is occasionally injured by stab wounds or operations on the groin. Paralysis of the quadriceps results. The prognosis is good if a laceration of the nerve is repaired early.

Cranial nerves (Box 57.12)

Olfactory nerve (I)

The fine olfactory filaments pass through the cribriform plate to join the olfactory bulb, which runs on the undersurface of the frontal lobe. Damage can result from acceleration–deceleration

Box 57.12

Cranial nerve damage

Nerve	Problem
Olfactory (I)	Damaged when ethmoid bone fractured
Optic (II)	Can be damaged by external pressure or by glial tumours internally
Oculomotor (III)	Damage produces ptosis, and the eye points down and laterally. The pupil is fixed and dilated
Trochlear (IV)	Damage produces diplopia. It is rarely damaged alone
Trigeminal (V)	Supplies sensation to the face and the chewing muscles. Pressure on the nerve may produce trigeminal neuralgia
Abducens (VI)	Can be damaged by pressure or fractured base of skull. Damage produces diplopia
Facial (VII)	Main motor supply to muscles of facial expression. It can be damaged by viral infection (Bell's palsy) or surgery to the parotid
Vestibulocochlear (VIII)	Carries nerves of hearing and balance. Affected by trauma or acoustic neuromas
Glossopharyngeal (IX)	Mediates the sensory part of the gag reflex. May be affected by fractured base of skull
Vagus (X)	Supplies the vocal cords through the recurrent laryngeal, which may be damaged at surgery
Accessory (XI)	Supplies trapezius. Damaged in surgery in the posterior triangle of the neck
Hypoglossal (XII)	Supplies muscles to tongue. Can be damaged during surgery to the submandibular glands

injuries, causing shifts in the position of the brain, fractures of the ethmoid bone and meningioma arising from the floor of the anterior cranial fossa. The sense of smell is impaired and because of the strong relationship between smell and taste this can considerably affect the enjoyment of food and drink. These injuries are likely to leave a permanent deficit.

Optic nerve (II)

The optic nerve is an outgrowth from the cerebrum and has an investing nerve sheath, enclosing cerebrospinal fluid (CSF), which allows intracranial hydrostatic pressure to be transmitted to the optic fundus. A rise in intracranial pressure may be manifest by swelling of the optic disc (papilloedema). The optic nerve may be damaged as it leaves the skull, and glial tumours may arise within the substance of the optic nerve, particularly in children. By testing the visual field, it may be possible to infer the site of intrinsic or extrinsic lesions affecting the optic pathways.

Oculomotor nerve (III)

A complete lesion of this cranial nerve causes total paralysis of the levator palpebrae superioris, resulting in ptosis. Owing to the unopposed action of the fourth and sixth cranial nerves, the eye is deviated downwards and outwards, and, when the lid is lifted, diplopia will occur. Because of the unopposed action of sympathetic fibres, there is dilatation of the pupil, which is unresponsive to both light and accommodation. The length of the intracranial course of the third cranial nerve exposes the nerve to damage either intracranially, as it leaves the skull, or within the orbit. In circumstances of raised intracranial pressure, herniation of the uncus of the temporal lobe through the tentorial notch leads to pressure on the third cranial nerve and a dilated pupil. This is a late and serious sign of raised intracranial pressure.

Trochlear nerve (IV)

The fourth cranial nerve supplies the superior oblique muscle and is rarely involved by itself. It is associated with diplopia.

Trigeminal nerve (V)

This nerve has a sensory portion, conveying sensation from the face and a motor root, supplying the muscles of mastication. There are three divisions of the nerve: the ophthalmic, maxillary and mandibular (Fig. 57.13). The most common clinical manifestation of trigeminal nerve dysfunction is trigeminal neuralgia. This condition occurs predominantly in the middle-aged and elderly, with a female predominance. It is characterised by severe, dagger-like pain within one or more divisions of the trigeminal nerve. Frequently, the pain is triggered by any movement or stimulus to the face. It is becoming increasingly recognised that ectatic vascular loops may cause compression of the fifth nerve, producing these symptoms. Management of trigeminal neuralgia is, in the first instance, with medication (see Pain). However, if this fails, surgery may be considered to relieve vascular compression in the posterior fossa or to disrupt the trigeminal ganglion using percutaneous thermocoagulation.

Abducens nerve (VI)

This nerve supplies the lateral rectus muscle and, when it occurs in isolation, results in diplopia due to the unopposed action of the medial rectus muscle. Because of its long intracranial course, the sixth nerve may be affected by fractures of the skull base or, alter-

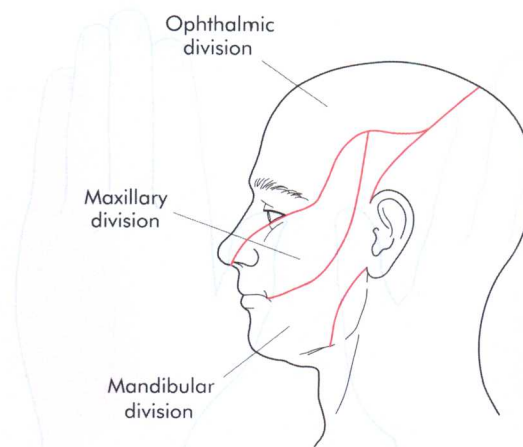


Figure 57.13 Diagram showing the areas of skin innervated by the sensory divisions of the trigeminal nerve.

natively, a supratentorial mass lesion may result in traction of the nerve as it passes over the petrous tip.

Facial nerve (VII)

The seventh cranial nerve gives a motor supply to the muscles of facial expression, and its sensory branch, the chorda tympani, carries taste from the anterior two-thirds of the tongue.

In clinical practice, the motor supply is of most importance. Paralysis of the facial muscles may result from upper or lower motor neurone lesions. Because of bilateral cortical representation of motor supply to the upper half of the face, upper motor neurone lesions, such as those caused by cerebrovascular events, will result in weakness of the face with preservation of eye closure and forehead movement. In a lower motor neurone lesion, all muscles innervated by the facial nerve will be affected, and this results in complete facial weakness, with loss of resting tone and of facial expression (Fig. 57.14).

The causes of facial nerve damage include:

- cerebellopontine angle lesions, such as an acoustic neuroma;
- Bell's palsy, a mononeuritis that may be related to viral infection;
- trauma to the nerve during surgery on the parotid gland.

Damage to the nerve in the face should be repaired as with other peripheral nerves. If damage or a defect occurs during the course of a cerebellopontine angle operation, such as removal of an acoustic neuroma, then the facial nerve may be reconstituted using either a nerve graft or a piece of freeze-thawed skeletal muscle. When repair is not possible, cross-face nerve transfer may be carried out. Alternatively, surgical procedures may be used to improve the resting state of the face. Maintenance of eye closure is paramount to protect the cornea. This can be achieved surgically by tarsorrhaphy.

Vestibulocochlear nerve (VIII)

The eighth cranial nerve carries information from the vestibular apparatus and organ of Corti (hearing). The surgical significance

Charles Bell | 1774–1842. Surgeon, Middlesex Hospital, London, England (1812–35), and later, Professor of Surgery, University of Edinburgh, Scotland (1836–42).
Alfonso Corti | 1822–1888. A histologist who worked in Vienna, Austria, Berlin, Germany, Utrecht, The Netherlands, and Turin, Italy, but who never held an academic post.