***Peripheral nerve***

***disorders***

**NERVE STRUCTURE AND FUNCTION**

**Peripheral nerves are bundles of axons conducting**

**efferent (motor) impulses from cells in the anterior**

**horn of the spinal cord to the muscles, and afferent**

**(sensory) impulses from peripheral receptors via cells**

**in the posterior root ganglia to the cord. They also**

**convey sudomotor and vasomotor fibres from ganglion**

**cells in the sympathetic chain. Some nerves are**

**predominantly motor, some predominantly sensory;**

**the larger trunks are mixed, with motor and sensory**

**axons running in separate bundles.**

**Each axon is, in reality, an extension or elongated**

**process of a nerve cell, or neuron (see Chapter 10).**

**The cell bodies of the motor neurons supplying the**

**peripheral muscles are clustered in the anterior horn**

**of the spinal cord; a single motor neuron with its axon**

**may, therefore, be more than a metre long. The cell**

**bodies of the sensory neurons serving the trunk an**

***PATHOLOGY***

**Nerves can be injured by ischaemia, compression,**

**traction, laceration or burning. Damage varies in**

**severity from transient and quickly recoverable loss of**

**function to complete interruption and degeneration.**

**There may be a mixture of types of damage in the various**

**fascicles of a single nerve trunk.**

**Transient ischaemiaAcute nerve compression causes numbness and tingling within 15 minutes, loss of pain sensibility after**

**30 minutes and muscle weakness after 45 minutes.**

**Relief of compression is followed by intense paraesthesiae**

**lasting up to 5 minutes (the familiar ‘pins and**

**needles’ after a limb ‘goes to sleep’); feeling is**

**restored within 30 seconds and full muscle power**

**after about 10 minutes. These changes are due to**

**transient endoneurial anoxia and they leave no trace**

**of nerve damage.**

**Neurapraxia**

**Seddon (1942) coined the term ‘neurapraxia’ to**

**describe a reversible physiological nerve conduction**

**block in which there is loss of some types of sensation**

**and muscle power followed by spontaneous recovery**

**after a few days or weeks. It is due to mechanical pressure**

**causing segmental demyelination and is seen typically**

**in ‘crutch palsy’, pressure paralysis in states of**

**drunkenness *(‘Saturday night palsy’*) and the milder**

**types of tourniquet palsy.**

**Axonotmesis**

**This is a more severe form of nerve injury, seen typically**

**after closed fractures and dislocations. The term means,**

**literally, axonal interruption. There is loss of conduction**

**but the nerve is in continuity and the neural tubes**

**are intact. Distal to the lesion, and for a few millimetresretrograde, axons disintegrate and are resorbed by**

**phagocytes. This *wallerian degeneration* (named after**

**the physiologist, Augustus Waller, who described the**

**process in 1851) takes only a few days and is accompanied**

**by marked proliferation of Schwann cells and**

**fibroblasts lining the endoneurial tubes. The denervated**

**target organs (motor end-plates and sensory**

**receptors) gradually atrophy, and if they are not reinnervated**

**within 2 years they will never recover.**

**Axonal *regeneration* starts within hours of nerve**

**damage, probably encouraged by neurotropic factors**

**produced by Schwann cells distal to the injury. From**

**the proximal stumps grow numerous fine unmyelinated**

**tendrils, many of which find their way into the**

**cell-clogged endoneurial tubes. These axonal**

**processes grow at a speed of 1–2 mm per day, the**

**larger fibres slowly acquiring a new myelin coat. Eventually**

**they join to end-organs, which enlarge and start**

**functioning**

**Neurotmesis**

**In Seddon’s original classification, neurotmesis meant**

**division of the nerve trunk, such as may occur in an**

**open wound. It is now recognized that severe degrees**

**of damage may be inflicted without actually dividing**

**the nerve. If the injury is more severe, whether the**

**nerve is in continuity or not, recovery will not occur.**

**As in axonotmesis, there is rapid wallerian degeneration,**

**but here the endoneurial tubes are destroyed**

**over a variable segment and scarring thwarts any hope**

**of regenerating axons entering the distal segment and**

**regaining their target organs. Instead, regenerating**

**fibres mingle with proliferating Schwann cells and**

**fibroblasts in a jumbled knot, or ‘neuroma’, at the site**

**of injury. Even after surgical repair, many new axons**

**fail to reach the distal segment, and those that do may**

**not find suitable Schwann tubes, or may not reach the**

**correct end-organs in time, or may remain incompletely**

**myelinated. Function may be adequate but is**

**never normal.**

**The ‘double crush’ phenomenon**

**There is convincing evidence that proximal compression**

**of a peripheral nerve renders it more susceptible**

**to the effects of a second, more peripheral injury. This**

**may explain why peripheral entrapment syndromes**

**are often associated with cervical or lumbar spondylosis.**

**A similar type of ‘sensitization’ is seen in patients**

**with peripheral neuropathy due to diabetes or alcoholism**

****

**CLINICAL FEATURES**

**Acute nerve injuries are easily missed, especially if**

**associated with fractures or dislocations, the symptoms**

**of which may overshadow those of the nerve**

**lesion. *Always test for nerve injuries following any significant***

***trauma.* If a nerve injury is present, it is crucial**

**also to look for an accompanying vascular injury.**

**Ask the patient if there is numbness, paraesthesia or**

**muscle weakness in the related area. Then examine**

**the injured limb systematically for signs of abnormal**

**posture (e.g. a wrist drop in radial nerve palsy), weakness**

**in specific muscle groups and changes in sensibility.**

**Areas of altered sensation should be accurately**

**mapped. Each spinal nerve root serves a specific dermatome**

**(see Fig. 11.3) and peripheral nerves have**

**more or less discrete sensory territories which are**

**illustrated in the relevant sections of this chapter.**

**Despite the fact that there is considerable overlap in**

**sensory boundaries, the area of altered sensibility is**

**usually sufficiently characteristic to provide an**

**anatomical diagnosis. Sudomotor changes may be**

**found in the same topographic areas; the skin feels dry**

**due to lack of sweating. If this is not obvious, the**

**‘plastic pen test’ may help. The smooth barrel of the**

**pen is brushed across the palmar skin: normally there**

**is a sense of slight stickiness, due to the thin layer of**

**surface sweat, but in denervated skin the pen slips**

**along smoothly with no sense of stickiness in the**

**affected area.**

**The neurological examination must be repeated at**

**intervals so as not to miss signs which appear hours**

**after the original injury, or following manipulation or**

**operation.**

***In chronic nerve injuries* there are other characteristic**

**signs. The anaesthetic skin may be smooth and**

**shiny, with evidence of diminished sensibility such as**

**cigarette burns of the thumb in median nerve palsy or**

**foot ulcers with sciatic nerve palsy. Muscle groups will**

**be wasted and postural deformities may become fixed.**

**Beware of trick movements which give the appearance**

**of motor activity where none exists.**

**Assessment of nerve recovery**

**The presence or absence of distal nerve function can be**

**revealed by simple clinical tests of muscle power and**

**sensitivity to light touch and pin-prick. Remember that**

**after nerve injury motor recovery is slower than sensory**

**recovery. More specific assessment is required to answer**

**two questions: How severe was the lesion? How well is**

**the nerve functioning now?**

**THE DEGREE OF INJURY**

***The history* is most helpful. A low energy injury is**

**likely to have caused a neurapraxia; the patient should**

**be observed and recovery anticipated. A high energy**

**injury is more likely to have caused axonal and**

**endoneurial disruption (Sunderland third and fourth**

**degree) and so recovery is less predictable. An open**

**injury, or a very high energy closed injury, will probably**

**have divided the nerve and early exploration is**

**called for.**

***Tinel’s sign* – peripheral tingling or dysaesthesia**

**provoked by percussing the nerve – is important. In a**

**neurapraxia, Tinel’s sign is negative. In axonotmesis,**

**it is positive at the site of injury because of sensitivity**

**of the regenerating axon sprouts. After a delay of a**

**few days or weeks, the Tinel sign will then advance at**

**a rate of about 1 mm each day as the regenerating**

**axons progress along the Schwann-cell tube. *Motor***

***activity* also should progress down the limb. Failure of**

**Tinel’s sign to advance suggests a fourth or fifth**

**degree injury and the need for early exploration. If the**

**Tinel sign proceeds very slowly, or if muscle groups**

**do not sequentially recover as expected, then a good**

**recovery is unlikely and here again exploration must**

**be considered.**

***Electromyography (EMG) studies* can be helpful. If a**

**muscle loses its nerve supply, the EMG will show**

**denervation potentials by the third weekexcludes neurapraxia but of course it does not distinguish**

**between axonotmesis and neurotmesis; this**

**remains a clinical distinction, but if one waits too long**

**to decide then the target muscle may have failed**

**irrecoverably and the answer hardly matters**

**ASSESSMENT OF NERVE FUNCTION**

***Two-point discrimination* is a measure of innervation**

**density. After nerve regeneration or repair, a proportion**

**of proximal sensory axons will fail to reach their**

**appropriate sensory end-organ; they will either have**

**regenerated down the wrong Schwann-cell tube or**

**will be entangled in a neuroma at the site of injury.**

**Therefore, two-point discrimination (measured**

**with a bent paper clip and compared with the opposite**

**normal side) gives an indication of how completely**

**the nerve has recovered. Static two-point**

**discrimination measures slowly adapting sensors**

**(Merkel cells) and moving two-point discrimination**

**measures rapidly adapting sensors (Meissner corpuscles**

**and pacinian corpuscles). Moving two-point discrimination**

**is more sensitive and returns earlier.**

**Normal static two-point discrimination is about 6**

**mm and moving is about 3 mm.**

***Threshold tests* measure the threshold at which a sensory**

**receptor is activated. They are more useful in**

**nerve-compression syndromes, where individual receptors**

**fail to send impulses centrally; two-point discrimination**

**is preserved because the innervation density is**

**not affected. Fine nylon monofilaments of varying**

**widths are placed perpendicularly on the skin and the**

**size of the lightest perceptible filament is recorded.**

***Locognosia* is the ability to localize touch and can be**

**tested with a standardized hand map.**

***The Moberg pick-up test* measures tactile gnosis. The**

**patient is blindfolded and instructed to pick up and**

**identify nine objects as rapidly as possible.**

***Motor power* is graded on the Medical Research**

**Council scale as:**

**0 No contraction.**

**1 A flicker of activity.**

**2 Muscle contraction but unable to overcome gravity.**

**3 Contraction able to overcome gravity.**

**4 Contraction against resistance.**

**5 Normal power.**

**PRINCIPLES OF TREATMENT**

**Nerve exploration**

**Closed low energy injuries usually recover spontaneously**

**and it is worth waiting until the most proximally**

**supplied muscle should have regained function.**

**Exploration is indicated: (1) if the nerve was seen to**

**be divided and needs to be repaired; (2) if the type of**

**injury (e.g. a knife wound or a high energy injury)**

**suggests that the nerve has been divided or severely**

**damaged; (3) if recovery is inappropriately delayed**

**and the diagnosis is in doubt.**

**Vascular injuries, unstable fractures, contaminated**

**soft tissues and tendon divisions should be dealt with**

**before the nerve lesion. The incision will be long, as**

**the nerve must be widely exposed above and below**

**the lesion before the lesion itself is repaired. The**

**nerve must be handled gently with suitable instruments.**

**Bipolar diathermy and magnification are essential.**

**An operating microscope is ideal but magnifying**

**loupes are better than nothing. A nerve stimulator is**

**essential if scarring makes recognition uncertain. If**

**microsurgical equipment and expertise are not available,**

**then the nerve lesion should be identified and**

**the wound closed pending transferral to an appropriatea**

**facility.**

**Primary repair**

**A divided nerve is best repaired as soon as this can be**

**done safely. Primary suture at the time of wound**

**toilet has considerable advantages: the nerve ends**

**have not retracted much; their relative rotation is usually**

**undisturbed; and there is no fibrosis.**

**A clean cut nerve is sutured without further preparation;**

**a ragged cut may need paring of the stumps**

**with a sharp blade, but this must be kept to a minimum.**

**The stumps are anatomically orientated and**

**fine (10/0) sutures are inserted in the epineurium.**

**There should be no tension on the suture line. Opinions**

**are divided on the value of fascicular repair with**

**perineurial sutures.**

**Sufficient relaxation of the tissues to permit tension-**

**free repair can usually be obtained by positioning**

**the nearby joints or by mobilizing and re-routing the**

**nerve. If this does not solve the problem then a primary**

**nerve graft must be considered. A traction lesion**

**– especially of the brachial plexus – may leave a gap**

**too wide to close. These injuries are best dealt with in**

**specialized centres, where primary grafting or nerve**

**transfer can be carried out.**

**If a tourniquet is used it should be a pneumatic**

**one; it must be released and bleeding stopped before**

**the wound is closed.**

**The limb is splinted in a position to ensure minimal**

**tension on the nerve; if flexion needs to be excessive,**

**a graft is required. The splint is retained for 3 weeks**

**and thereafter physiotherapy is encouraged.**

**Delayed repair**

**Late repair, i.e. weeks or months after the injury, may**

**be indicated because: (1) a closed injury was left alone**

**but shows no sign of recovery at the expected time;**

**(2) the diagnosis was missed and the patient presents**

**late; or (3) primary repair has failed. The options must**

**be carefully weighed: if the patient has adapted to the**

**functional loss, if it is a high lesion and re-innervation**

**is unlikely within the critical 2-year period, or if there**

**is a pure motor loss which can be treated by tendon**

**transfers, it may be best to leave well alone. Excessive**

**scarring and intractable joint stiffness may, likewise,**

**make nerve repair questionable; yet in the hand it is**

**still worthwhile simply to regain protective sensation.**

**The lesion is exposed, working from normal tissue**

**above and below towards the scarred area. When the**

**nerve is in continuity it is difficult to know whether**

**resection is necessary or not. If the nerve is only**

**slightly thickened and feels soft, or if there is conduction**

**across the lesion, resection is not advised; if the**

**‘neuroma’ is hard and there is no conduction on**

**nerve stimulation, it should be resected, paring back**

**the stumps until healthy fascicles are exposed.**

**How to deal with the gap? The nerve must be**

**sutured without tension. The stumps may be brought**

**together by gently mobilizing the proximal and distal**

**segments, by flexing nearby joints to relax the soft tissues,**

**or (in the case of the ulnar nerve) by transposing**

**the nerve trunk to the flexor aspect of the elbow. In this**

**way, gaps of 2 cm in the median nerve, 4–5 cm in the**

**ulnar nerve and 6–8 cm in the sciatic nerve can usually**

**be closed, the limb being splinted in the ‘relaxing’ position**

**for 4–6 weeks after the operation. Elsewhere, gaps**

**of more than 1–2 cm usually require grafting.**

**Nerve guides**

**It is now apparent that nerve gaps can regenerate**

**through a tube which excludes the surrounding tissue**

**from each end. The tubes can be autogenous vein,**

**freeze-dried muscle, silicone or metal; soluble guides**

**(flexible at body temperature) which dissolve over**

**weeks or months are also used. This technology offers**

**a simple way of avoiding a nerve graft yet achieving**

**results which are at least as good in both digital nerves**

**and probably in main trunks.**

**Nerve grafting**

**Free autogenous nerve grafts can be used to bridge**

**gaps too large for direct suture. The sural nerve is**

**most commonly used; up to 40 cm can be obtained**

**from each leg. Because the nerve diameter is small,**

**several strips may be used (cable graft). The graft**

**should be long enough to lie without any tension, and**

**it should be routed through a well-vascularized bed.**

**The graft is attached at each end either by fine sutures**

**or with fibrin glue.**

**It is crucial that the motor and sensory fascicles are**

**appropriately connected by the graft. There are various**

**techniques which can help. Careful inspection of the**

**fascicular alignment, structure and vascular markings is**

**often helpful. Enzyme-staining techniques can be used.**

**Vascularized grafts are used in special situations. If**

**the ulnar and median nerves are both damaged (e.g.**

**in Volkmann’s ischaemia) a pedicle graft from the**

**ulnar nerve may be used to bridge the gap in the**

**median. It is also possible to use free vascularized**

**grafts for certain brachial plexus lesions.**

**Nerve transfer**

**In root avulsions of the upper brachial plexus, too**

**proximal for direct repair, nerve transfer can be used.**

**The spinal accessory nerve can be transferred to the**

**suprascapular nerve, and intercostal nerves can be**

**transferred to the musculocutaneous nerve. If biceps**

**has failed because too much time has passed since the**

**injury, an entire muscle (gracilis or latissimus dorsi)**

**can be transferred as a free flap, attached between**

**elbow and shoulder and then innervated by joining**

***PRINCIPLES OF TENDON TRANSFERANSFERINCIPLES* OF TENDON TRANSFER**

**Assess the problem**

**Which muscles are missing?**

**Which muscles are available?**

**The donor muscle should be:**

**expendable**

**powerful enough**

**an agonist or synergist**

**The recipient site should:**

**be stable**

**have mobile joints and supple tissues**

**The transferred tendon should be:**

**routed subcutaneously**

**placed in a straight line of pull**

**capable of firm fixation**

**The patient should be:**

**motivated**

**able to comprehend and attend hand therapy**

**intercostal nerves or the spinal accessory nerve to the**

**stump of the original nerve supplying that muscle.**

**Care of paralysed parts**

**While recovery is awaited the skin must be protected**

**from friction damage and burns. The joints should be**

**moved through their full range twice daily to prevent**

**stiffness and minimize the work required of muscles**

**when they recover. ‘Dynamic’ splints may be helpful.**

**Tendon transfers**

**Motor recovery may not occur if the axons, regenerating**

**at about 1 mm per day, do not reach the muscle**

**within 18–24 months of injury. This is most likely**

**when there is a proximal injury in a nerve supplying**

**distal muscles. In such circumstances, tendon transfers**

**should be considered. The principles can be summarized**

**in the Box on the previous page.**

**Recommended transfers are discussed under the**

**individual nerve lesions.**

**PROGNOSIS**

***Type of lesion* Neurapraxia always recovers fully;**

**axonotmesis may or may not; neurotmesis will not**

**unless the nerve is repaired.**

***Level of lesion* The higher the lesion, the worse the**

**prognosis.**

***Type of nerve* Purely motor or purely sensory nerves**

**recover better than mixed nerves, because there is less**

**likelihood of axonal confusion.**

***Size of gap* Above the critical resection length, suture**

**is not successful.**

***Age* Children do better than adults. Old people do**

**poorly.**

***Delay in suture* This is a most important adverse factor.**

**The best results are obtained with early nerve**

**repair. After a few months, recovery following suture**

**becomes progressively less likely.**

***Associated lesions* Damage to vessels, tendons and**

**other structures makes it more difficult to obtain recovery**

**of a useful limb even if the nerve itself recovers.**

***Surgical techniques* Skill, experience and suitable facilities**

**are needed to treat nerve injuries. If these are**

**lacking, it is wiser to perform the essential wound toilet**

**and then transfer the patient to a specialized centre.**

**REGIONAL SURVEY OF NERVE**