Peripheral Nerve Injuries: A Clinical Guide

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Chapter 1
The Peripheral Nervous System: Anatomy and Function

> Restatement of the facts appears to be warranted by the misconceptions shown by many post graduate students [21].

The nervous system is the mechanism through which the organism is kept in touch with its internal structures and external environments and reacts to changes in them. *The central nervous system* – the brain and its caudal prolongation the spinal cord – is connected to the periphery by the *peripheral nervous system*. The latter includes the cranial nerves, the spinal nerves with their roots and rami, the peripheral nerves and the peripheral components of the autonomic nervous system, the sympathetic, parasympathetic and enteric divisions [16]. The peripheral nerves contain motor fibres (to end plates in skeletal muscle), sensory fibres (from organs and endings in skin, muscle, tendon, periosteum, and bone and joint), efferent autonomic fibres (to blood vessels, sweat glands and arrectores pilarum muscle), and visceral afferent fibres. In no other system is so much functional and relay capacity concentrated in so small a volume of tissue. The cervical spinal cord, with a width of about 2 cm and a depth of about 1.5 cm, contains all the apparatus transmitting control of somatic function from the neck down, together with that of control of much visceral function. Because of their greater content of connective tissue, the peripheral nerves have proportionately a lesser functional content, yet severance in an adult's arm of the median nerve of 5 mm diameter effectively ruins the function of the hand and forearm.

Twelve pairs of cranial nerves arise from the brain and brain stem. The second of these, the optic nerves, are in fact prolongations of the central nervous system. Thirty one pairs of spinal nerves – eight cervical, twelve thoracic, five lumbar, five sacral and one coccygeal – arise from the spinal cord. Each spinal nerve leaves or enters the cord by ventral, largely motor, root, and a dorsal sensory root (Figs. 1.1 and 1.2). Each sensory root splits into several rootlets as it approaches the spinal cord; these enter the cord along the line of the posterolateral sulcus. The division of the anterior roots into rootlets is less obvious and takes place nearer the cord. Because in the adult the spinal cord extends caudally only so far as the first lumbar

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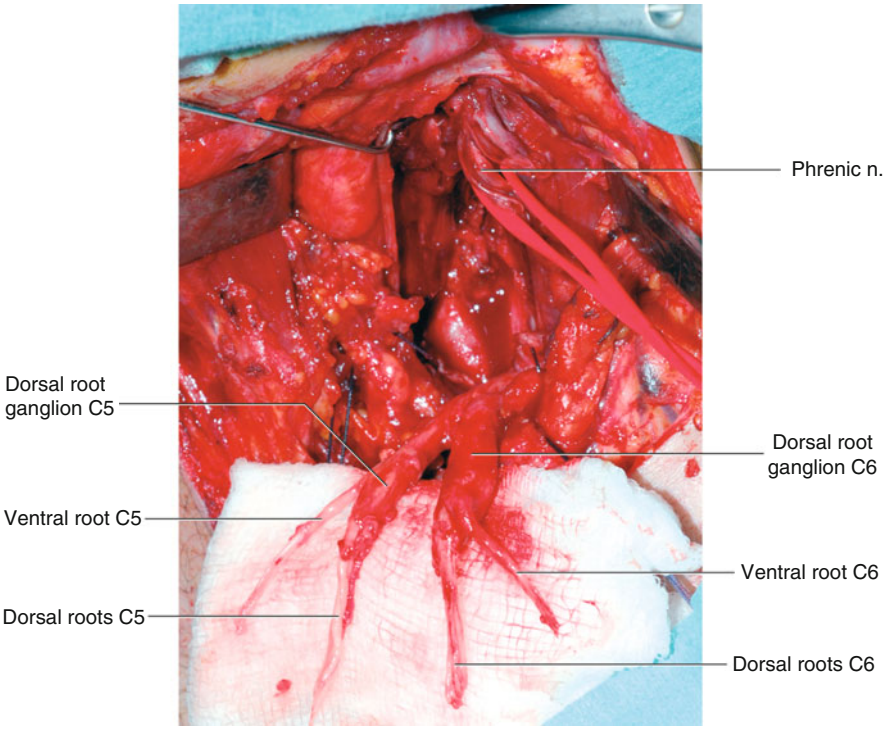

Fig. 1.1 The fifth, sixth cervical nerves avulsed from the spinal cord. The ventral root is easily distinguishable from the dorsal rootlets. Note the dorsal root ganglion, the dural sleeve merging into the epineurium and the spinal nerve itself. The small pieces of tissue on the proximal ends of the dorsal rootlets (*below*) are probably portions of the spinal cord

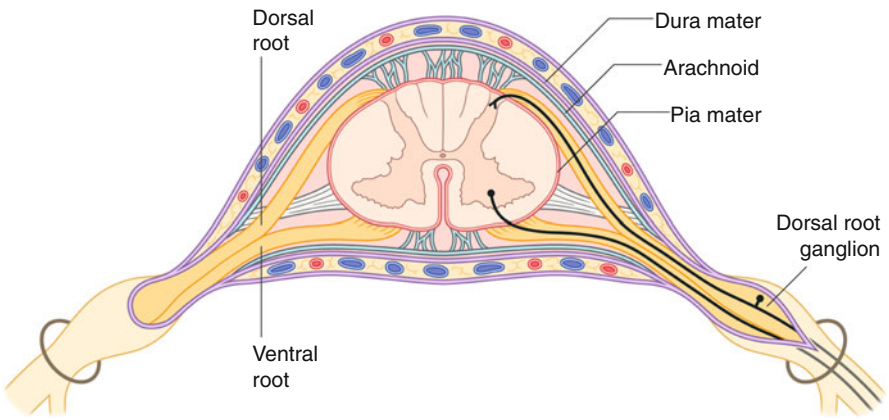

Fig. 1.2 The origin of the roots from the cord, their junction just distal to the dorsal root ganglion, and the emergence of the nerve from the spinal cord

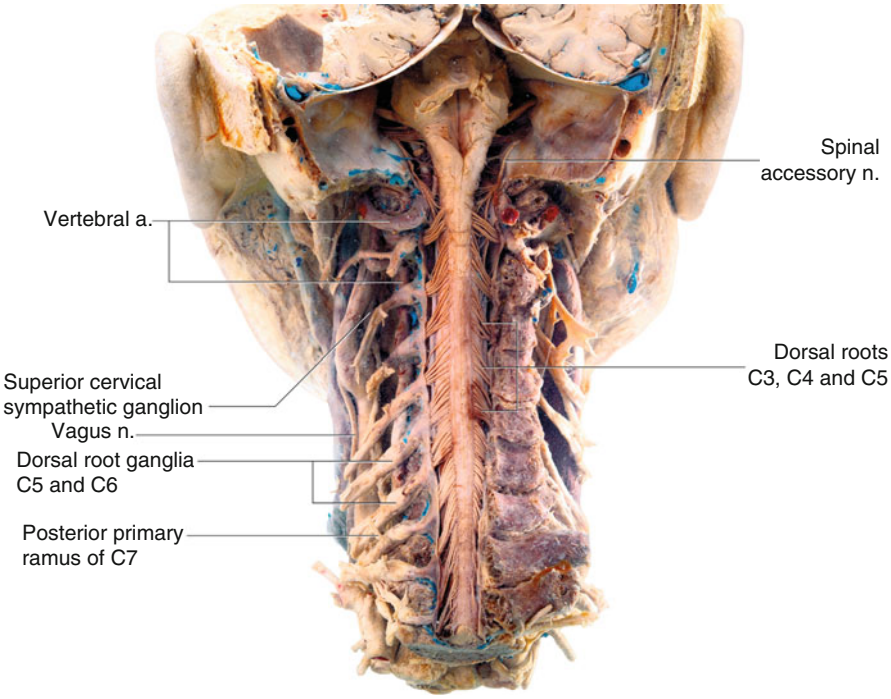

Fig. 1.3 The brainstem and cervical cord exposed by laminectomy. The spinal accessory nerve passes anterior to the dorsal roots, and emerges through the jugular foramen accompanied by the vagus and glosso-pharyngeal nerves. The vertebral artery courses anteriorly to the spinal nerves

vertebral level, the obliquity of the emerging and entering roots in the theca increases from above downwards. The theca below the first lumbar level is occupied by the lumbar, sacral and coccygeal roots forming a leash whose appearance has been likened to that of a horse's tail (cauda equina).

The cell bodies of the fibres forming the anterior roots are mostly situated in the anterior horn of the grey matter of the spinal cord; those of the fibres of the dorsal root are in the dorsal root ganglion, situated in or near the intervertebral foramen. As they approach the foramen, the two roots join to form the spinal nerve, which outside the foramen divides into anterior and posterior primary rami (Fig. 1.3).

Three divisions of the autonomic nervous system – the sympathetic, the parasympathetic and the enteric – are usually described [16]. In each, pre-ganglionic fibres arise from cells in the brain stem or spinal cord. These relay in ganglia to post-ganglionic fibres innervating cardiac muscle, smooth muscle and glands. Most viscera are supplied by both sympathetic and parasympathetic divisions; the cell bodies of the enteric system are confined to the wall of the bowel.

1.1 The Cranial Nerves

The first, olfactory, mediates the sense of smell; the second, optic, mediates that of sight. The latter nerve is a prolongation of the central nervous system. The third, fourth and sixth nerves control the muscles concerned with movement of the eye. The fifth, (trigeminal) nerve has an extensive motor and sensory function, controlling the muscles of the jaw and conveying sensation from the skin of the face and the mucosa of the mouth and nose, and probably from the superficial muscles of the face. The lingual branch which conveys sensation from the tongue and buccal mucosa is, with the inferior alveolar nerve, particularly at risk during operations upon the mouth and jaws. The seventh (facial) nerve innervates the superficial muscles of the face and neck. It is remarkable for its vulnerability to damage in each of the three parts of its course – intra-cranial, intra-osseous (in the petrous part of the temporal bone) and extra-temporal. The eighth (auditory) nerve mediates the senses of hearing and of balance. The ninth (glosso-pharyngeal) nerve conveys sensibility from the pharynx and from the back of the tongue and has a small motor function. The tenth (vagus) nerve has, as its name suggests, wide ranging branches and functions, most of the latter being parasympathetic. Motor branches innervate the muscles of the larynx, and sensory branches convey sensation from it. Its recurrent laryngeal branch is, in the ascending part of its course, in close relationship with the trachea and oesophagus and with the thyroid and parathyroid glands.

The spinal part of the eleventh (accessory) nerve arises from cells in the accessory nucleus, a column of cells extending from the second to the fifth and sixth cervical segments of the cord [5]. These cells are in the dorsolateral part of the anterior horn of the grey matter. The fibres emerge segmentally from each side of the cord, to unite to form on each side a nerve which passes rostrally, posterior to the denticulate ligament, into the cranial cavity through the foramen magnum (Fig. 1.4). In the cranial cavity the nerve unites briefly with its cranial part, derived mainly from the cells in nucleus ambiguus, before passing out of the skull with it through the jugular foramen. Outside the skull the two parts separate, the cranial portion going to join the vagus nerve and the spinal part passing obliquely down the neck to innervate the sternocleidomastoid and trapezius muscles. The spinal accessory nerve is particularly at risk to the activities of surgeons in the posterior triangle of the neck yet its course here is consistent. It emerges from beneath the sternocleidomastoid muscle at about 5 mm cephalad to the point where the greater auricular nerve begins its upward course over the anterior face of the muscle.

The twelfth (hypoglossal) nerve leaves the skull through the hypoglossal canal in the occipital bone to supply the intrinsic and all but one of the extrinsic muscles of the tongue. Although there are receptor organs in the muscles of the human tongue it is likely that most of the impulses from them travel in the lingual nerve. The sensation of taste is mediated by fibres in the facial nerve (anterior two thirds of the tongue) and by fibres in the glossopharyngeal nerve (posterior one third of the tongue). In the upper part of the neck the hypoglossal nerve is joined by fibres from the anterior rami of the uppermost two cervical nerves. These soon leave the nerve to form the ansa hypoglossi from which the infrahyoid muscles are supplied.

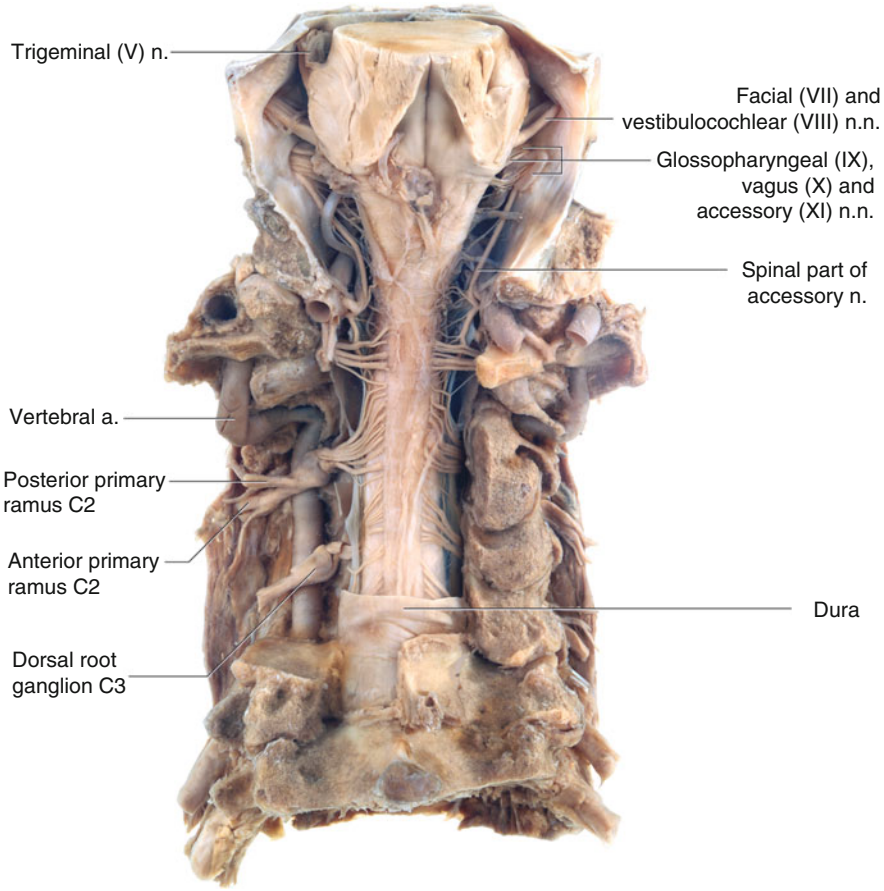

Fig. 1.4 The junction between the spinal cord and the brain stem shown by excision of posterior bony elements. The first cervical nerve passes away from the spinal cord at almost a right angle

1.2 The Spinal Nerves

1.2.1 The Anterior Primary Rami

The anterior primary rami of the uppermost four cervical nerves unite and branch to form the cervical plexus, through which the skin of the neck and part of the face and some of the muscles of the neck are innervated. A branch of the fourth cervical anterior ramus, with contributions from the third and fifth rami, passes caudally into the thorax as the phrenic nerve, to supply motor fibres to the diaphragm and sensory fibres to the related pleura, fibrous pericardium and peritoneum (Figs. 1.5 and 1.6).

The anterior primary rami of the lowest four cervical nerves and most of that of the first thoracic nerve unite and branch to form the brachial plexus in the lower

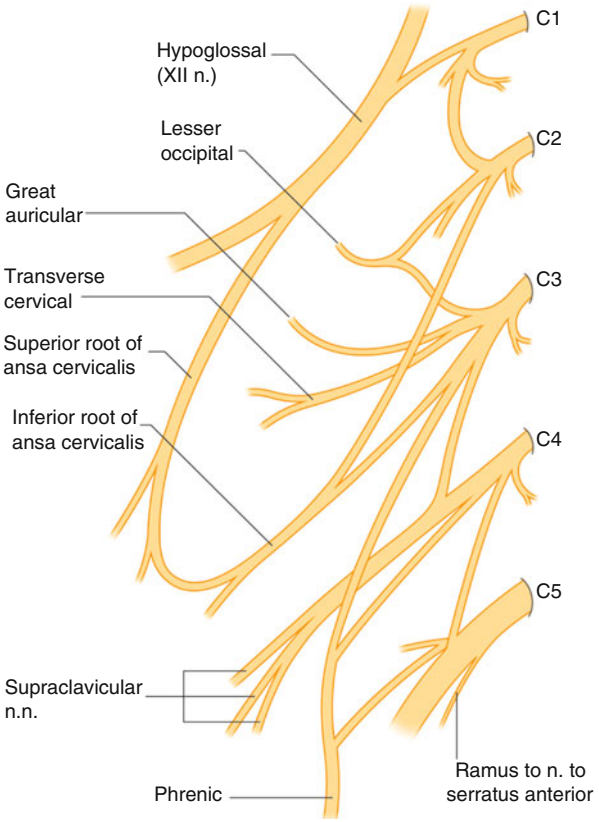

Fig. 1.5 The right cervical plexus and its (mainly sensory) branches

part of the neck and behind the clavicle (Fig. 1.7). The upper limb receives its innervation through the branches of this important plexus. The most proximal muscles are supplied by branches from the rami; the intermediate muscles by branches from the trunks and cords; the muscles of the limb itself by branches from the main terminal nerves – the median, ulnar, musculo-cutaneous, radial and circumflex (axillary). There is a segmental pattern to this innervation: the most proximal muscles are supplied by branches of the uppermost rami; the most distal muscles are supplied by branches derived from the eighth cervical and first thoracic nerves. The segmental pattern of innervation is shown more clearly in the cutaneous supply (Figs. 1.8 and 1.9) [28]. The cervical root supply has been, as it were, extruded from the supply to the trunk. Thus, in the transition of innervation from the skin of the neck to that of the trunk there is anteriorly a change from the fourth cervical to the second thoracic segment; posteriorly. from the fifth cervical to the first thoracic segment.

An important anatomical and functional differentiation of the plexus takes place with the division of the trunks into anterior and posterior divisions. From the anterior divisions the lateral and medial cords are formed; from the posterior divisions the posterior cord is formed. The lateral and medial cords innervate pre-axial (flexor)

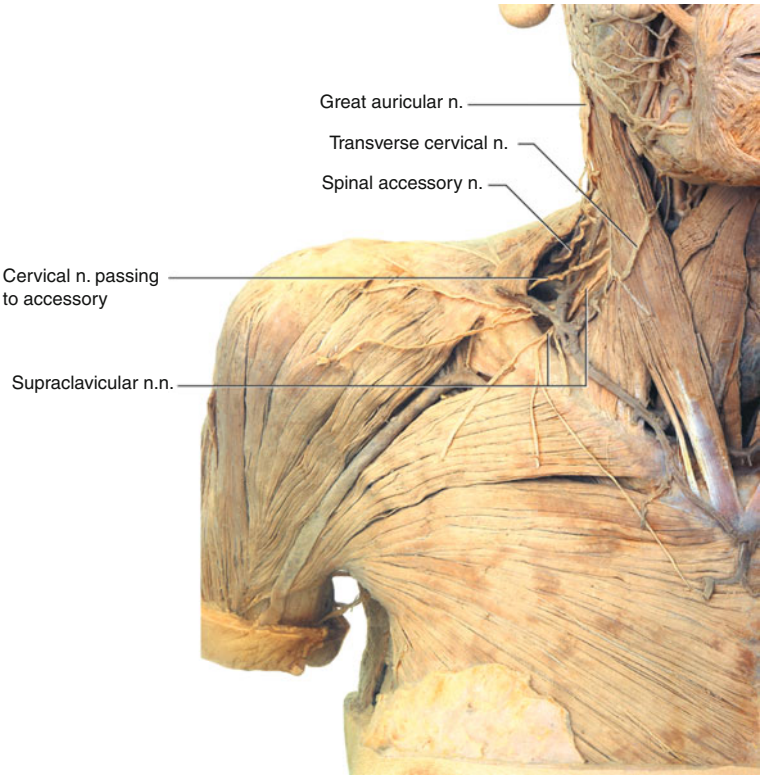

Fig. 1.6 The relations of the right cervical plexus

musculature; the posterior cord innervates post-axial (extensor) musculature (Figs. 1.10, 1.11, 1.12, 1.13, 1.14 and 1.15).

The plexus and the distribution of its nerves vary considerably from one individual to another: the contributions made by the component nerves vary; the origin and method of formation of the main nerves vary; in some cases the contribution of the fifth nerve is large and that of the first thoracic nerve is small, while in others the reverse is the case. The truly autonomous area of cutaneous supply of each main component nerve is small and variable in extent and location. The contributions made by the fourth cervical and second thoracic nerves vary: usually their contributions are small, but occasionally the fourth cervical nerve makes an important contribution to the innervation of scapulo-humeral muscles [5].

The supply of the skin and of the hand is divided between the median, ulnar and radial nerves. The first two supply the palmar aspect; all supply the dorsal aspect. The median nerve supplies the skin of the radial side of the palm, the palmar aspects of the thumb, index and middle fingers and of the radial part of the ring finger, and the terminal parts of the dorsal aspect of these digits. The ulnar nerve supplies the

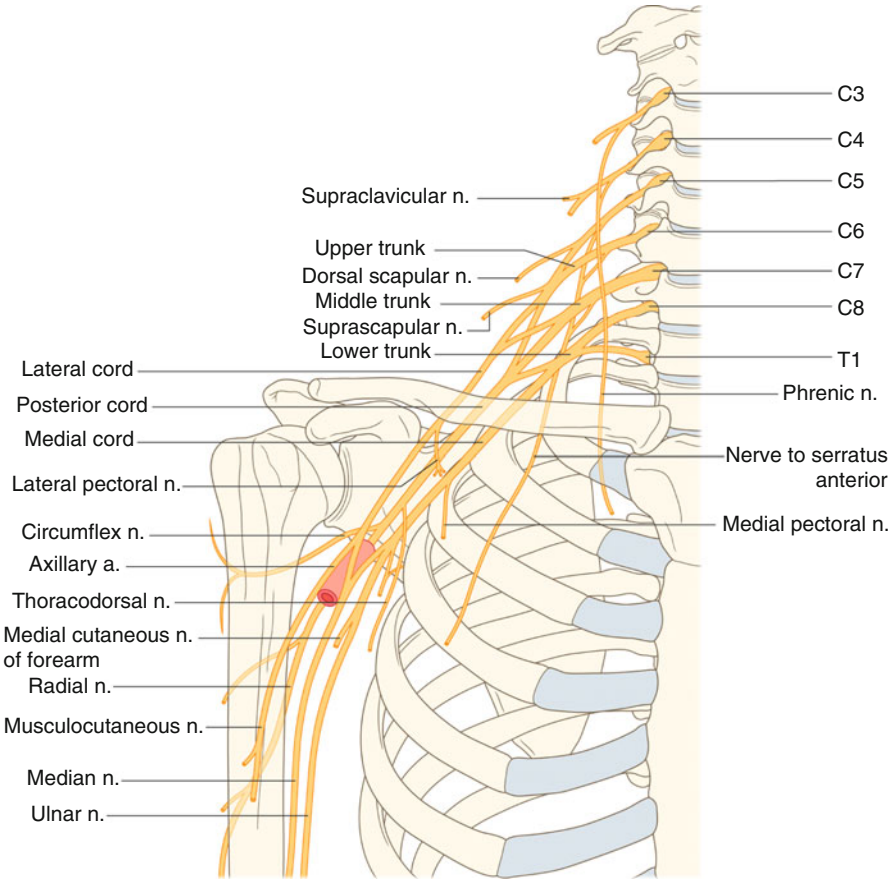

Fig. 1.7 The right brachial plexus. Note the sequence: the anterior primary rami; trunk; divisions; cords; nerves. Note that the trunks are upper, middle and lower, and that the cords are lateral, medial and posterior from their position in relation to the axillary artery which is, in fact, variable

skin of the ulnar side of the palm, the palmar aspects of the little finger and the ulnar part of the ring finger, the dorsal aspect of the ulnar half of the hand, the little and ring fingers and the ulnar side of the proximal part of the middle finger. The radial nerve supplies the radial side of the dorsum of the hand, of the proximal parts of the thumb and index fingers and of the radial side of the middle finger. Damage to the terminal branches of these nerves of cutaneous sensation which is usually caused by a needle or scalpel often leads to pain which is quite out of proportion to the functional importance of the nerve (Figs. 1.16 and 1.17).

Whilst the variations of the distribution of the peripheral nerves are significant, the variations in the distribution of the spinal nerves forming the brachial plexus are much more important. At least one third of patients with complete lesions of C5, C6 and C7 retain powerful extension of the digits and this is seen in some patients in whom only the first thoracic nerve has survived.

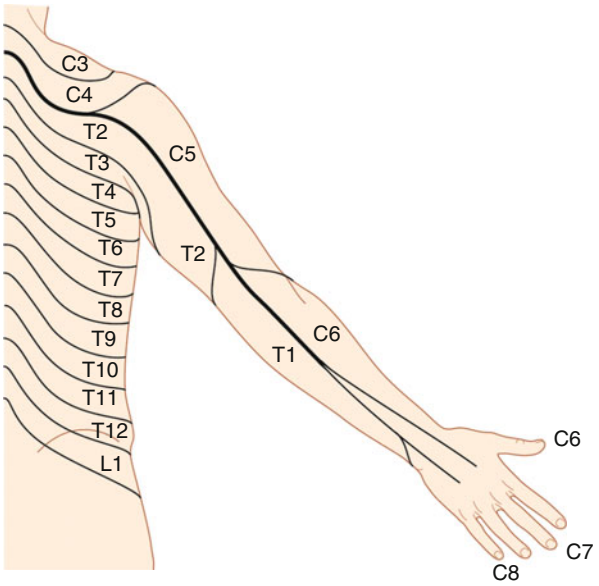

Fig. 1.8 Approximate distribution of dermatomes on the posterior aspect of the right upper limb (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

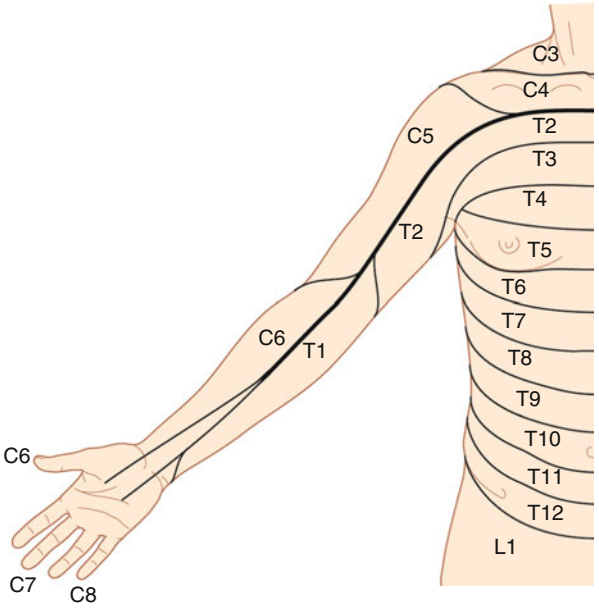

Fig. 1.9 Approximate distribution of dermatomes on the anterior aspect of the right upper limb (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

1.2.2 Thoracic Anterior Primary Rami

The second to the sixth thoracic anterior primary rami innervate the intercostal muscles and the skin of the anterior and lateral chest wall. Most of the first nerve goes

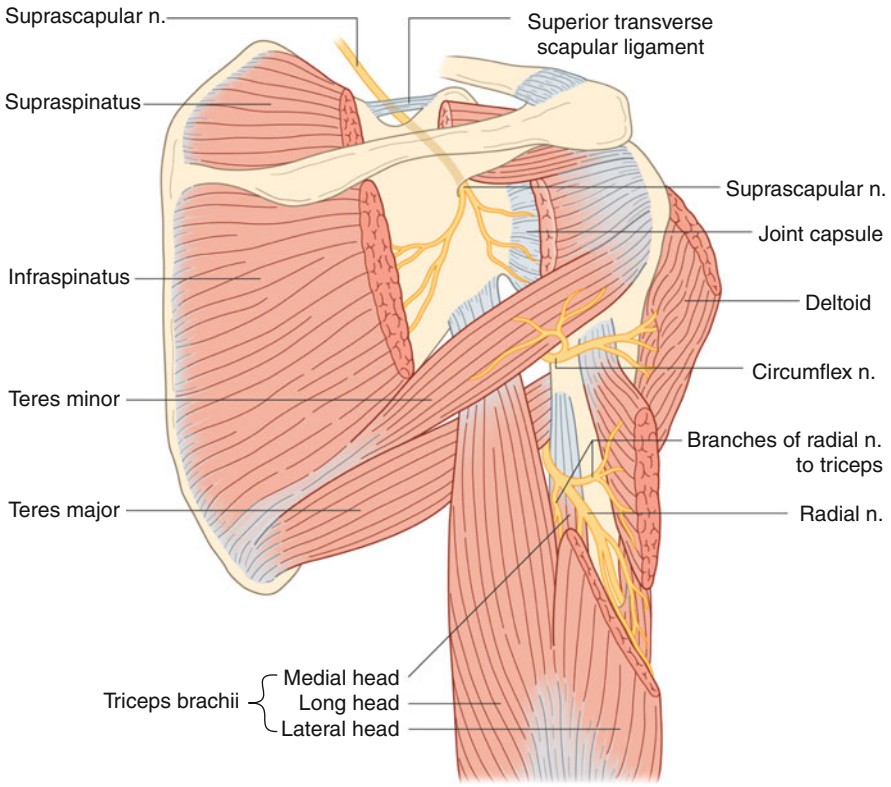

Fig. 1.10 The right circumflex (axillary) and suprascapular nerves

to join the brachial plexus; most of the second goes as the intercosto-brachial nerve to innervate the skin of the axilla and of the medial side of the arm. The lower six thoracic anterior rami continue from the intercostal spaces to the anterior wall of the abdomen, innervating its skin and muscles. The lowest nerves supply sensory fibres to the lateral part of the diaphragm. The lowest (12th) thoracic ventral ramus, sometimes called the subcostal nerve, is larger than the others and connects with the iliohypogastric branch of the first lumbar nerve.

1.2.3 Lumbar and Sacral Anterior Primary Rami (Fig. 1.18)

The first lumbar anterior primary ramus gives rise to two mainly cutaneous nerves and part of a third. The iliohypogastric, iloinguinal and genitofemoral nerves supply respectively the skin of part of the buttock, of the groin and the greater part of the external genitalia (Figs. 1.19 and 1.20). The second, third and fourth lumbar anterior rami unite and branch to form the lumbar plexus from which arise the nerves

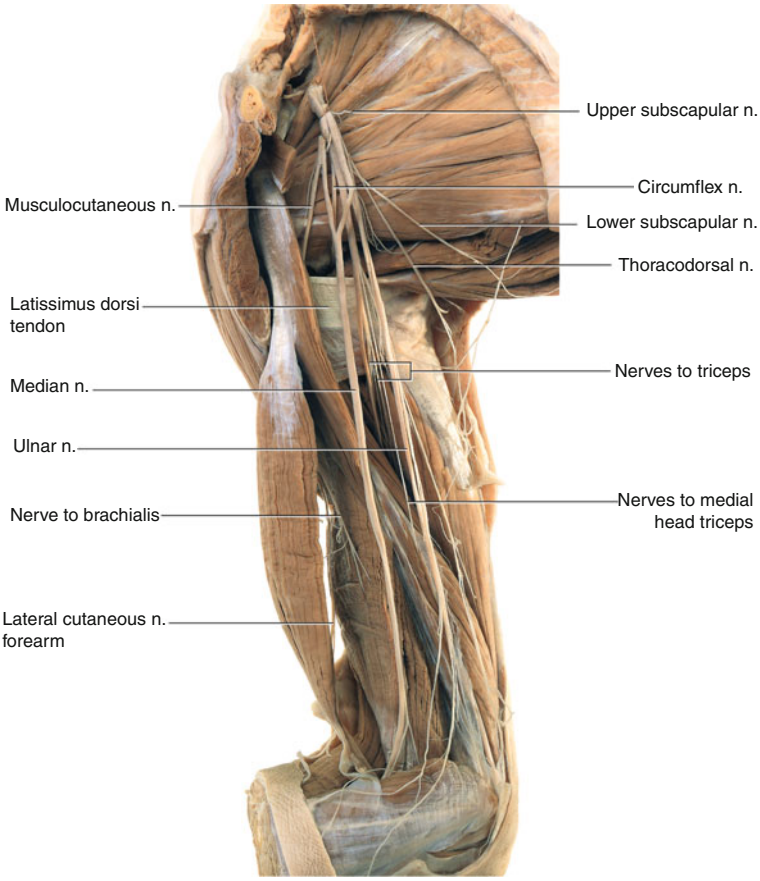

Fig. 1.11 The main nerves in the right axilla and arm

innervating the skin of the thigh and its anterior and medial muscles. The plexus is formed in the anterior part of the psoas major muscle, in the posterior wall of the abdomen. Its terminal branches lie under the parietal peritoneum. Some of these emerge lateral and some medial to the psoas major. The most important terminal branch is the femoral nerve, which passes, lateral to the psoas major and femoral vessels, under the inguinal ligament to reach the upper part of the thigh. Through its anterior and posterior divisions it supplies the skin of the anterior surface of the thigh and the quadriceps and sartorius muscles. The saphenous branch of the posterior division descends with the femoral artery to emerge from the femoral canal above the knee and supply the skin of the medial side of the leg and foot. The obturator nerve emerges medial to the psoas major and, passing along the lateral wall of the pelvis, emerges into the thigh through the obturator foramen. Through anterior and posterior branches the adductor muscles and the skin of the medial side of the thigh are supplied.

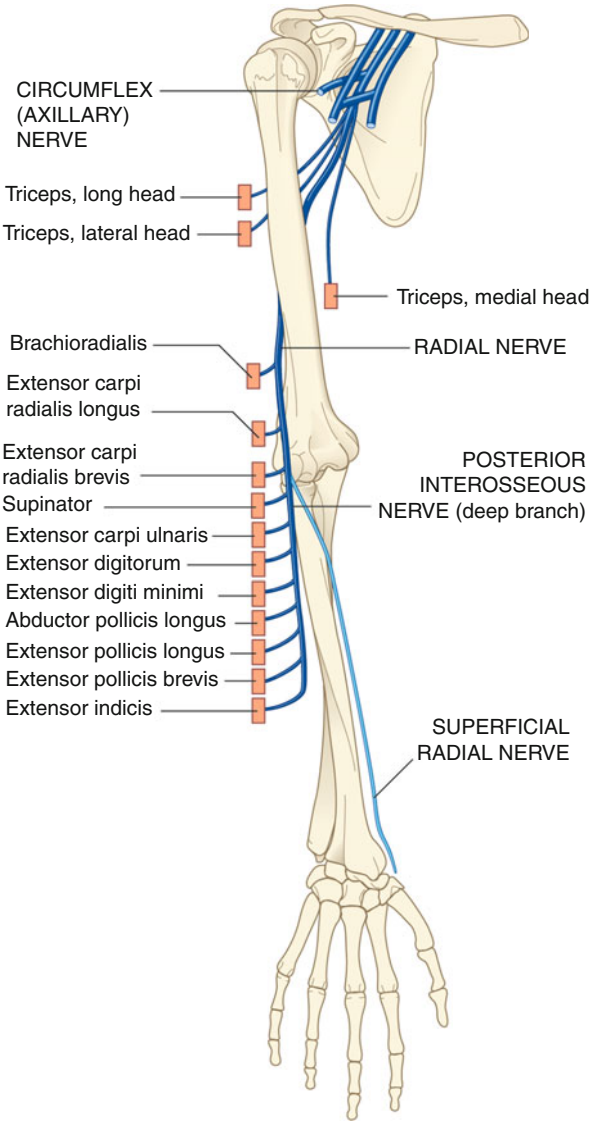

Fig. 1.12 The right radial nerve (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

Part of the fourth lumbar ramus and all the fifth ventral ramus join to form the lumbo-sacral trunk, which emerges medial to the psoas major to enter the pelvis and join the first, second and third sacral nerves to form the sacral plexus on the postero-lateral wall of the pelvis (Figs. 1.21 and 1.22).

The innervation of the perineum and most of the lower limb is derived from the branches of this plexus. The sciatic nerve, the largest in the body, leaves the pelvis through the greater sciatic foramen and passes behind the hip joint into the back of

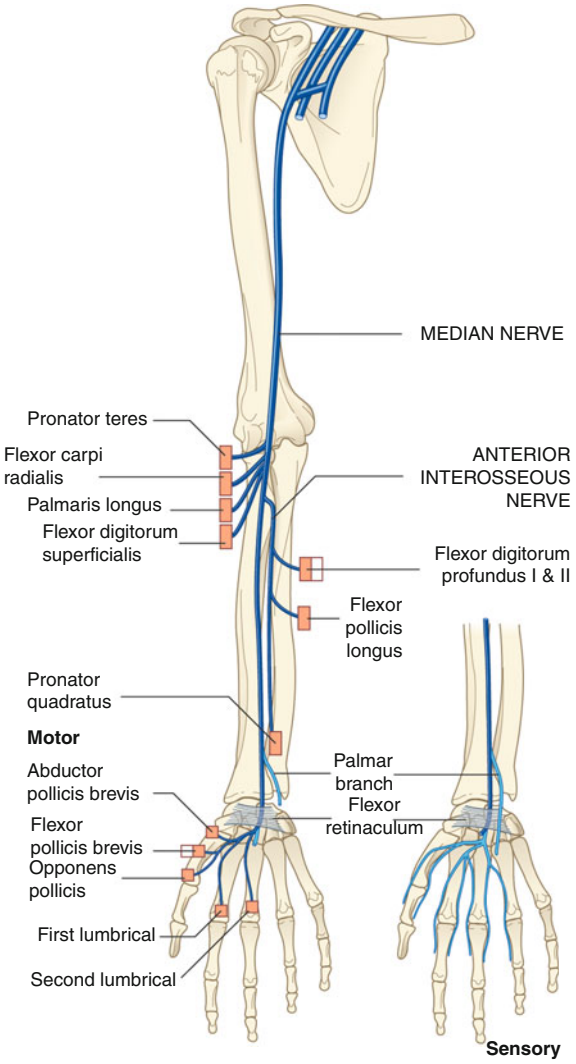

Fig. 1.13 The right median nerve (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

the thigh. This great trunk has two main components, which are functionally and often anatomically quite distinct (Figs. 1.23, 1.24, 1.25, 1.26, 1.27 and 1.28). The tibial nerve innervates the medial hamstrings, it descends in the midline through the popliteal fossa into the back of the leg, to supply its superficial and deep muscles. It has an important and frequently useful branch, the sural nerve, which arises in the upper part of the popliteal fossa, descends between the two heads of gastrocnemius and pierces the deep fascia in the proximal part of the leg. Usually, a branch from the common peroneal (or fibular) nerve joins the sural nerve; at times, it is larger than the contribution from the tibial nerve. Rarely, the sural nerve arises wholly

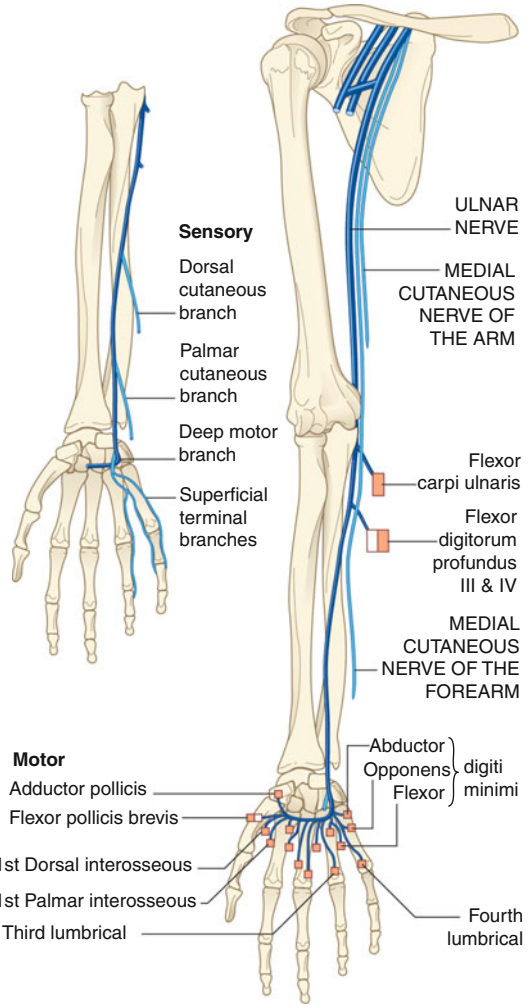

Fig. 1.14 The right ulnar nerve (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

from the common peroneal division. The nerve then descends to pass lateral to the tendo Achilles to supply the skin on the lateral side of the foot. The tibial nerve continues into the foot behind the medial malleolus and through its terminal medial and lateral plantar branches supplies the intrinsic muscles of the foot and the skin of the sole. The common peroneal nerve innervates the lateral hamstring muscles in the thigh. It diverges laterally from the mid line to pass behind the head of the fibula and lateral to its neck. Here it divides into deep and superficial peroneal nerves. The former passes into the anterior compartment of the leg to innervate the anterior muscles and finally to supply the extensor digitorum brevis and the skin of the dorsum of the first inter-digital space (Figs. 1.29 and 1.30). The superficial peroneal (musculocutaneous) nerve passes deep to the upper part of the peroneus longus

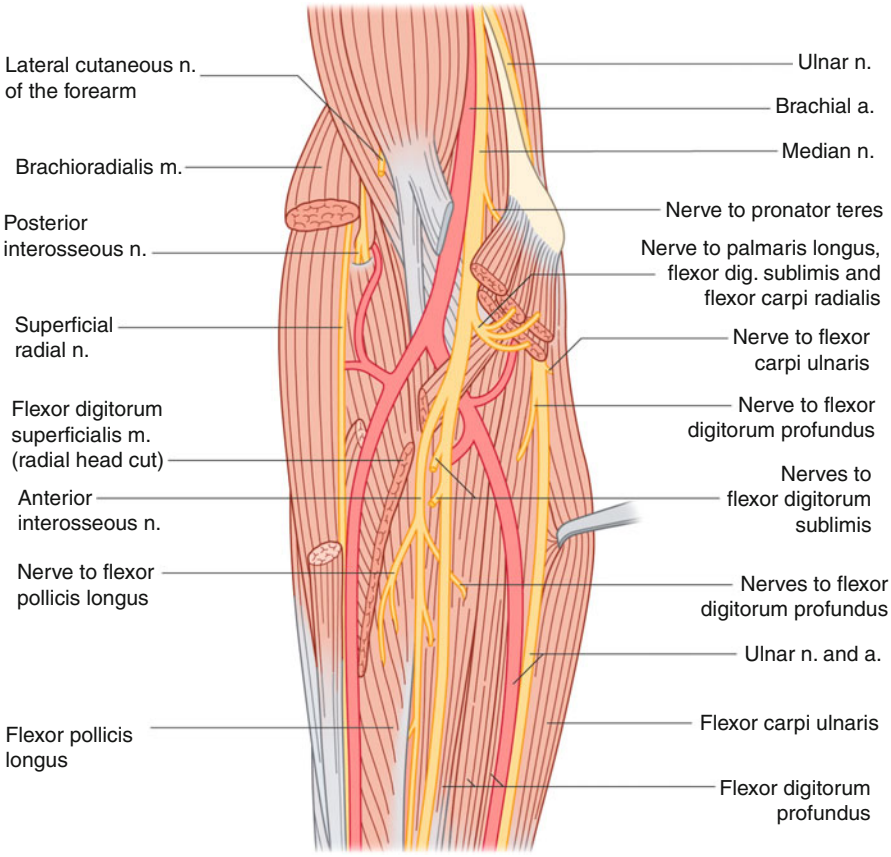

Fig. 1.15 The anterior aspect of the right elbow

muscle to supply both peronei. Its continuation pierces the deep fascia in the distal part of the leg to supply the skin of the dorsum of the foot and anterolateral part of the ankle.

The more proximal branches of the sacral plexus supply the gluteal muscles and the skin and muscles of the perineum. The superior gluteal nerve, emerging above the piriformis muscle, supplies the short gluteus medius and minimus and the tensor fasciae latae. The inferior gluteal nerve, emerging below the piriformis muscle, supplies the gluteus maximus muscle. The pudendal nerve leaves the pelvis through the greater sciatic foramen and, entering the pudendal canal through the lesser sciatic foramen, passes into the perineum to innervate its skin and muscles. As in the case of the upper limb, there is a segmental innervation of the muscles and, more easily seen, of the skin. Again, the segments innervating the limb have been extruded from the innervation of the trunk and perineum, so that in the transition from trunk to perineum there is posteriorly a segmental change

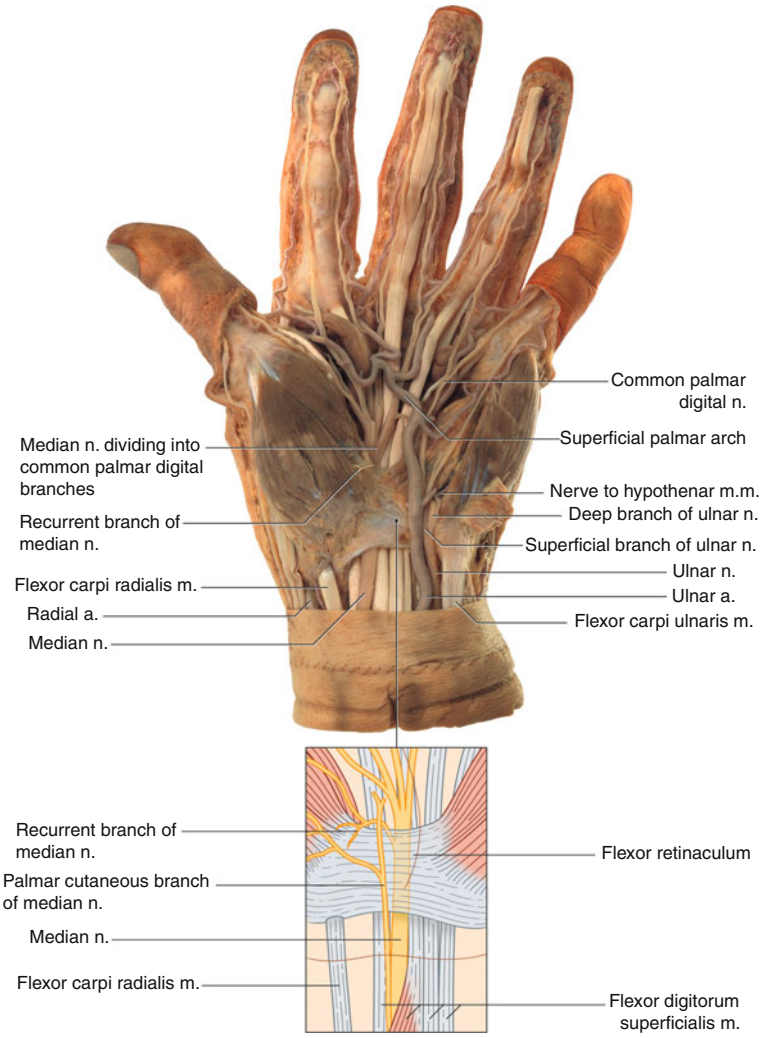

Fig. 1.16 The median and ulnar nerves in the left hand. *Inset* shows the normal course of median nerve at the wrist and also the palmar cutaneous nerve

from the third lumbar to the third sacral dermatome. The skin of the foot is supplied by all the main nerves of the lower limb save the obturator (Figs. 1.31 and 1.32). The plantar surface is supplied by the tibial nerve through its plantar branches; the medial side by the saphenous branch of the femoral nerve; the lateral side by the sural branch of the tibial nerve, and the dorsum by the superficial and deep divisions of the common peroneal nerve. Apparently trivial injuries to the terminal branches of the nerves of cutaneous sensation sometimes cause even more trouble in the lower than in the upper limbs.

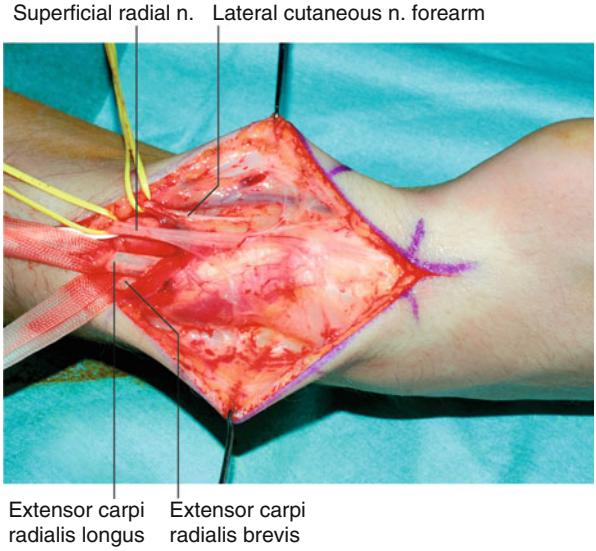

Fig. 1.17 The terminal branches of the superficial radial and lateral cutaneous nerves of forearm seen during operation at the right wrist

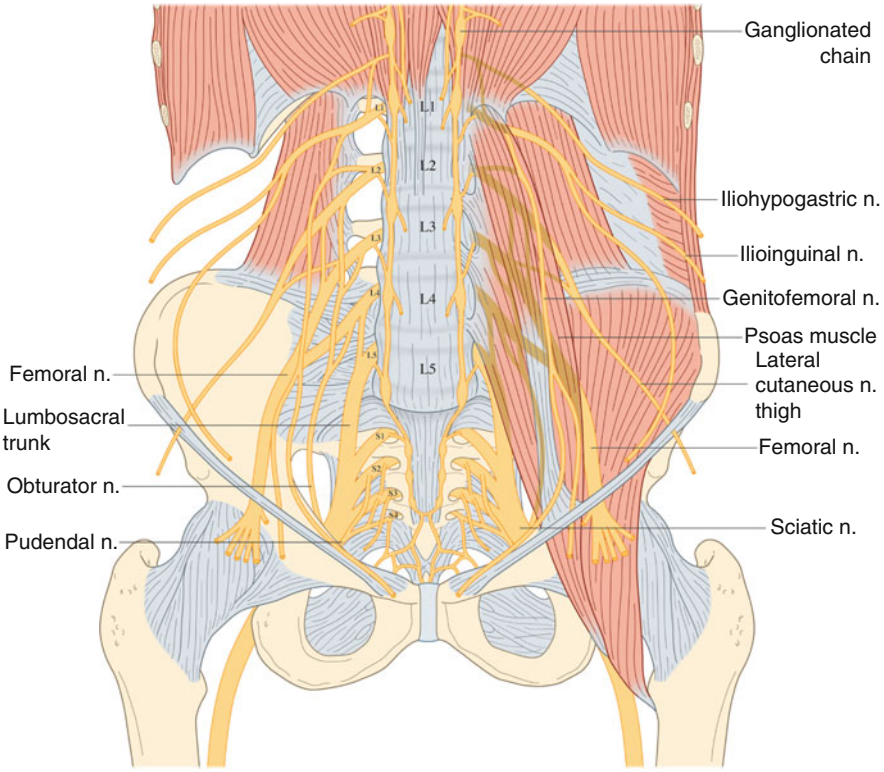

Fig. 1.18 The femoral and sacral plexuses and the ganglionated sympathetic chain

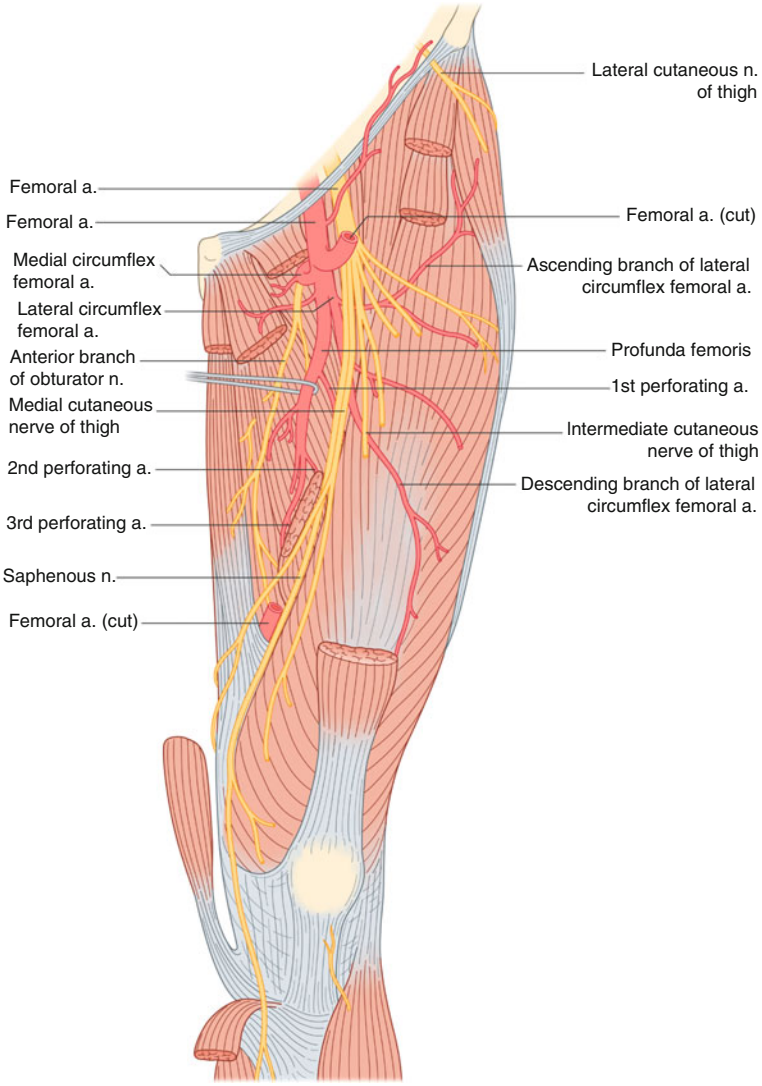

Fig. 1.19 The left femoral nerve

1.2.4 The Posterior Primary Rami

The posterior primary rami are usually smaller than the anterior. Most divide into medial and lateral branches to supply the muscles and skin of the posterior part of the neck and trunk. They do not enter the limbs.

The posterior primary rami of the uppermost three spinal nerves differ from those of the lowest five in extending their supply to the back of the head. The posterior

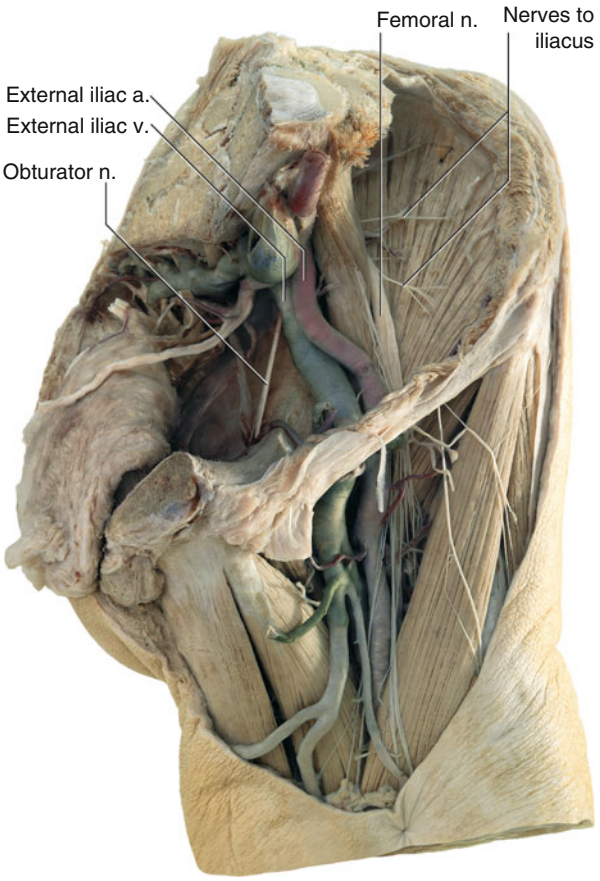

Fig. 1.20 The left femoral nerve

ramus of the first nerve, actually larger than the anterior ramus, chiefly supplies muscles between the atlas and occiput. The posterior ramus of the second cervical nerve is the largest of the cervical posterior rami and larger than its anterior ramus. Emerging between the posterior arch of the atlas and the lamina of the axis, it divides into a large medial and a smaller lateral branch. The former goes on as the great occipital nerve to innervate the skin of the back of the scalp. At its beginning, it is in close relationship with the back of the atlanto-axial joint. The third cervical posterior ramus provides a third occipital branch. The posterior rami of the lowest five cervical nerves innervate the posterior vertebral muscles; the medial branches of the fourth and fifth rami also innervate the skin.

The thoracic posterior primary rami similarly pass posteriorly close to the posterolateral (zygapophyseal) intervertebral joints to supply posterior vertebral muscles and the skin of the back of the chest. The lumbar posterior rami are similarly disposed, but only the uppermost three reach the skin. The sacral posterior rami are small, having a small distribution to muscle and to the skin over the sacrum.

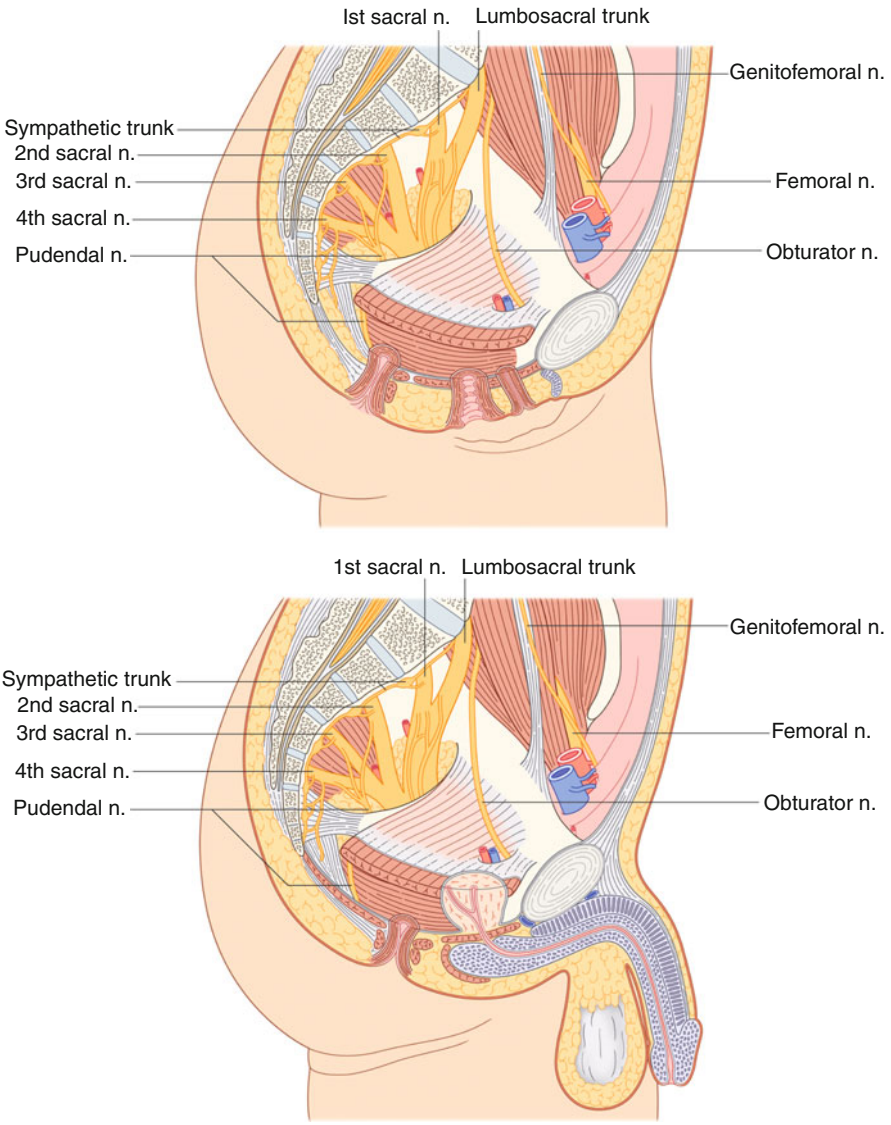

Fig. 1.21 The relations of the left sacral plexus. (**a**) *Above*: the female pelvis. (**b**) *Below*: the male pelvis

1.3 The Autonomic Nervous System

The sympathetic and parasympathetic systems are characterised by having relays between their cells of origin and their terminations: in the case of the sympathetic system the relays are in paravertebral or axial ganglia; in the case of the parasympathetic system they are in or near the organs innervated (Fig. 1.33).

Fig. 1.22 The left sacral plexus

1.3.1 The Sympathetic System

The pre-ganglionic cells of the efferent fibres of the sympathetic system are in the lateral horn of the grey matter of the spinal cord from the first thoracic to the second lumbar level. Most of the ganglia are in the paravertebral sympathetic chains extending from the top to the bottom of the spinal column; others lie in autonomic plexi in the abdominal cavity. Usually there are on each side two cervical, one cervico-thoracic (stellate), eleven thoracic, four lumbar and five sacral or pelvic ganglia. Pre-ganglionic myelinated fibres enter the cervico-thoracic, thoracic and upper two lumbar ganglia in white rami from the first thoracic to the second lumbar spinal nerve. These fibres relay in the corresponding ganglia or proceed up or down the chain to relay in other ganglia of the chain or in one of the ganglia of the autonomic plexi. The distribution to the spinal nerves is by way of grey rami, which contain unmyelinated fibres, from the corresponding paravertebral ganglia. Fibres pass directly from the autonomic plexi to their destinations. Afferent fibres have their cells in the posterior root ganglia; their sites of relay are not clearly identified.

The sympathetic supply to the head and neck arises mainly from the uppermost three thoracic segments, passes cranially, and relays in the cervical ganglia to be

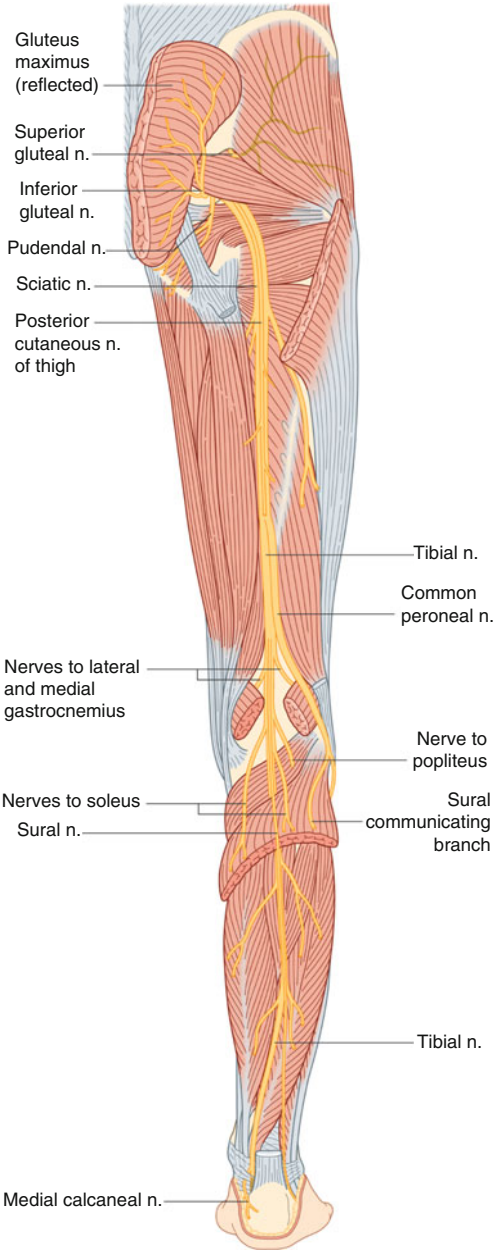

Fig. 1.23 The right sciatic nerve and its major components in the lower limb

distributed to vessels and sweat glands and in particular to the dilator of the pupil, and the smooth muscle fibres in orbitales and levator palpbrae superioris muscles. Most of the supply to these muscles of the eye arises from the first thoracic segment (Fig. 1.34).

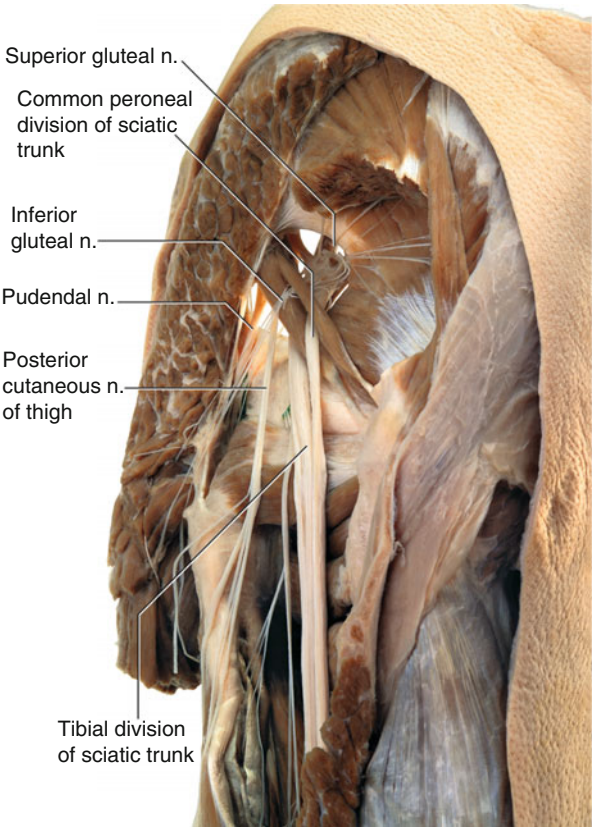

Fig. 1.24 The right sciatic nerve and its two main components, the common peroneal and tibial nerves. The CPN is quite distinct and in this specimen it passes from the tibial nerve and passes separately through piriformis muscle, a not uncommon variation

The sympathetic supply to the upper limb arises principally from the second to the sixth thoracic segments. Fibres pass up the chain to the middle cervical and cervicothoracic stellate ganglia, where they relay to be distributed by grey rami to the nerves of origin of the brachial plexus.

The sympathetic supply to the lower limbs arises from the lowest three thoracic and uppermost two lumbar segments, fibres enter the first and second lumbar ganglia by white rami, descend in the chain, relay in the lumbar and sacral ganglia and are distributed by grey rami to the lumbar and sacral nerves (Fig. 1.35).

1.3.2 The Parasympathetic Nervous System

The efferent outflow of the parasympathetic nervous system arises from nuclei in the mid-brain and part of the hind brain and the sacral part of the spinal cord. The pre-ganglionic fibres are distributed by the third, seventh, ninth and tenth cranial nerves and by the second to the fourth sacral spinal nerves. From the last arise the pelvic splanchnic nerves (nervi evigentes) which supply the ganglia in which the

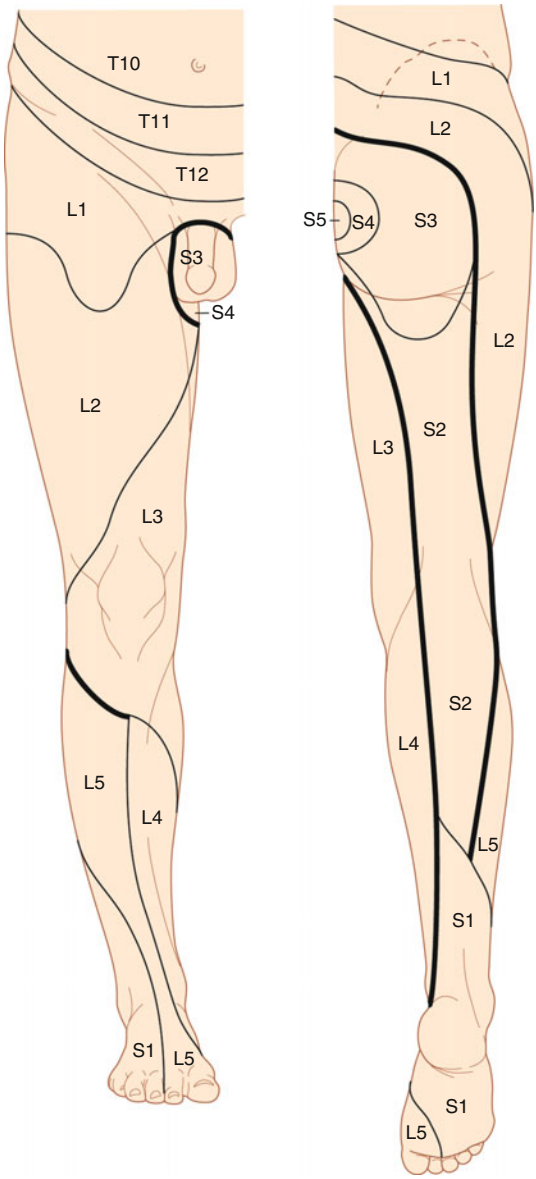

Fig. 1.25 Approximate distribution of dermatomes on the right lower limb (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

pre-ganglionic fibres relay are in or near the organs supplied. The effect of parasympathetic activity is inhibitory in the heart, motor to the muscle of the bladder and bowel and dilator in small vessels. The central control of both sympathetic and parasympathetic function is exercised from nuclei in the hypothalamus which themselves receive input from higher centres. The fibres from the hypothalamus almost certainly descend in a column in the lateral part of the white matter of the spinal

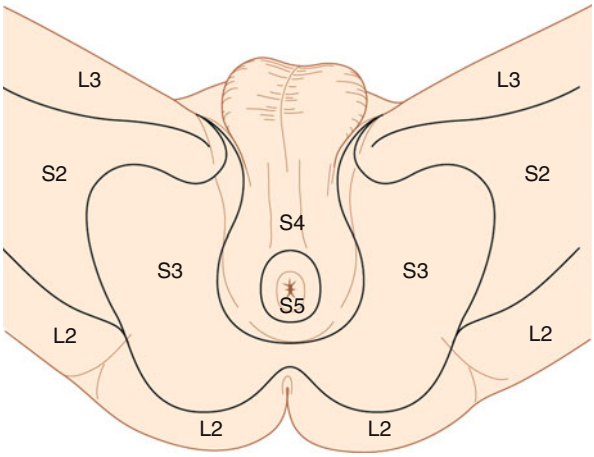

Fig. 1.26 Approximate distribution of dermatomes on the perineum (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

cord [27]. Parasympathetic fibres to the papillary and ciliary muscles pass with the oculomotor (III) cranial nerve via the ciliary ganglion, those to the lacrimal, submandibular and sublingual salivary glands travel with the facial (VII) cranial nerve via the submandibular ganglion. Those to the parotid gland are conveyed by the glossopharyngeal (IX) cranial nerve.

The main visceral plexi are the cardiac, pulmonary oesophageal, coeliac, mesenteric and hypogastric. They are fed from the cervical and cervico-thoracic ganglia, from the middle and lower thoracic ganglia (the thoracic splanchnic nerves), from the lumbar ganglia (the lumbar splanchnic nerves) and from the sacral ganglia. Both sympathetic and parasympathetic systems contribute to these plexi, the vagus (tenth cranial) nerve being the principal source of parasympathetic fibres to the chest and abdomen, and the second, third and fourth sacral nerves to the pelvis. The effect of efferent sympathetic activity is to cause sweating, to constrict small vessels and to cause contraction of the arrectores pilarum muscle. The visceral actions are to stimulate the action of the heart and to cause sphincteric contraction (see Sect. 1.10.6).

1.4 Nerves at Risk from Musculo Skeletal Injury

The anatomical arrangements of some of the peripheral nerves make them particularly vulnerable to damage from skeletal injury. The sacral nerves are particularly at risk in fractures involving the sacral foraminae. The proximity to bone of the main nerves at the elbow render all three vulnerable to skeletal injury. The sciatic trunk is damaged by the posterior displaced head of the femur.

The circumflex nerve runs in loose fatty tissue in the axilla, but then is captured, with the posterior circumflex vessels, in a tunnel formed by the fascia of the subscapularis muscle cranially, the teres major muscle caudally and the coraco-brachialis muscle laterally, at the entrance to the quadrilateral tunnel. The neurovascular

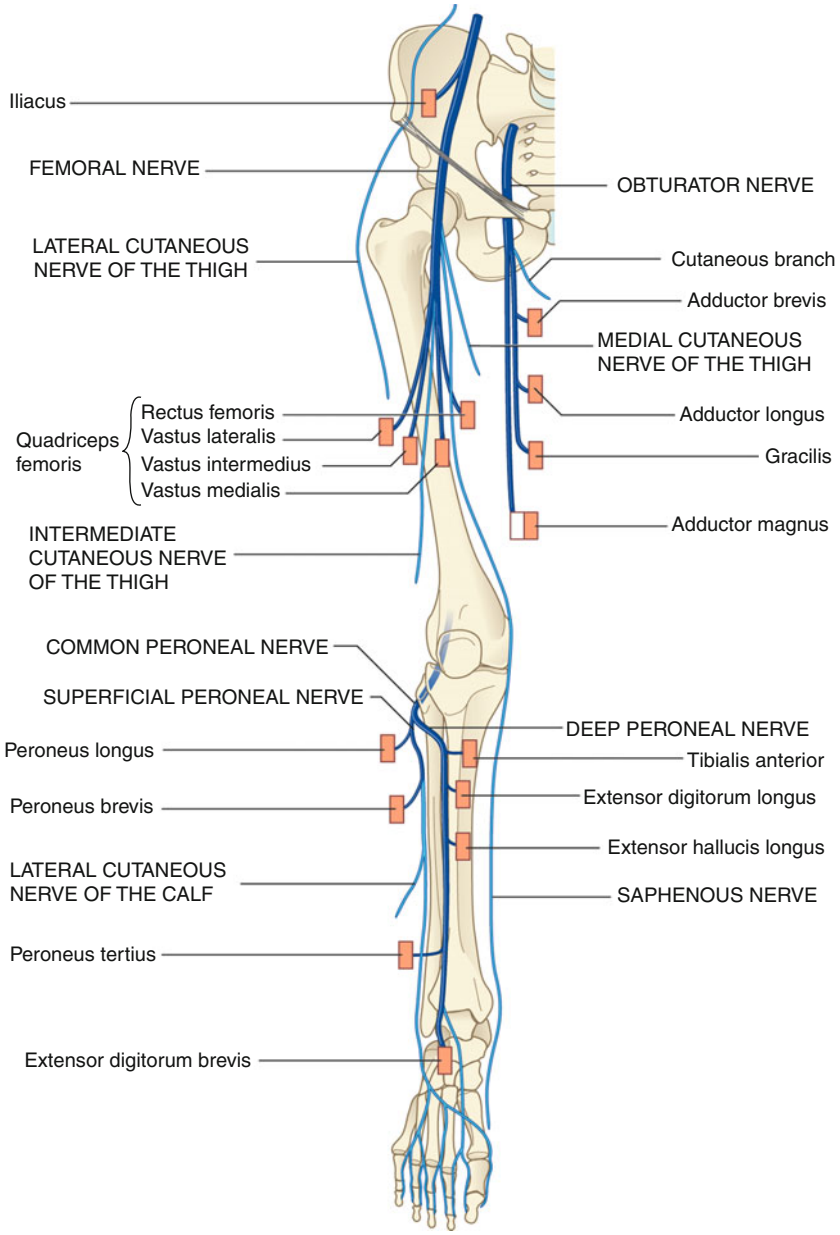

Fig. 1.27 The nerves on the anterior aspect of the right lower limb (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

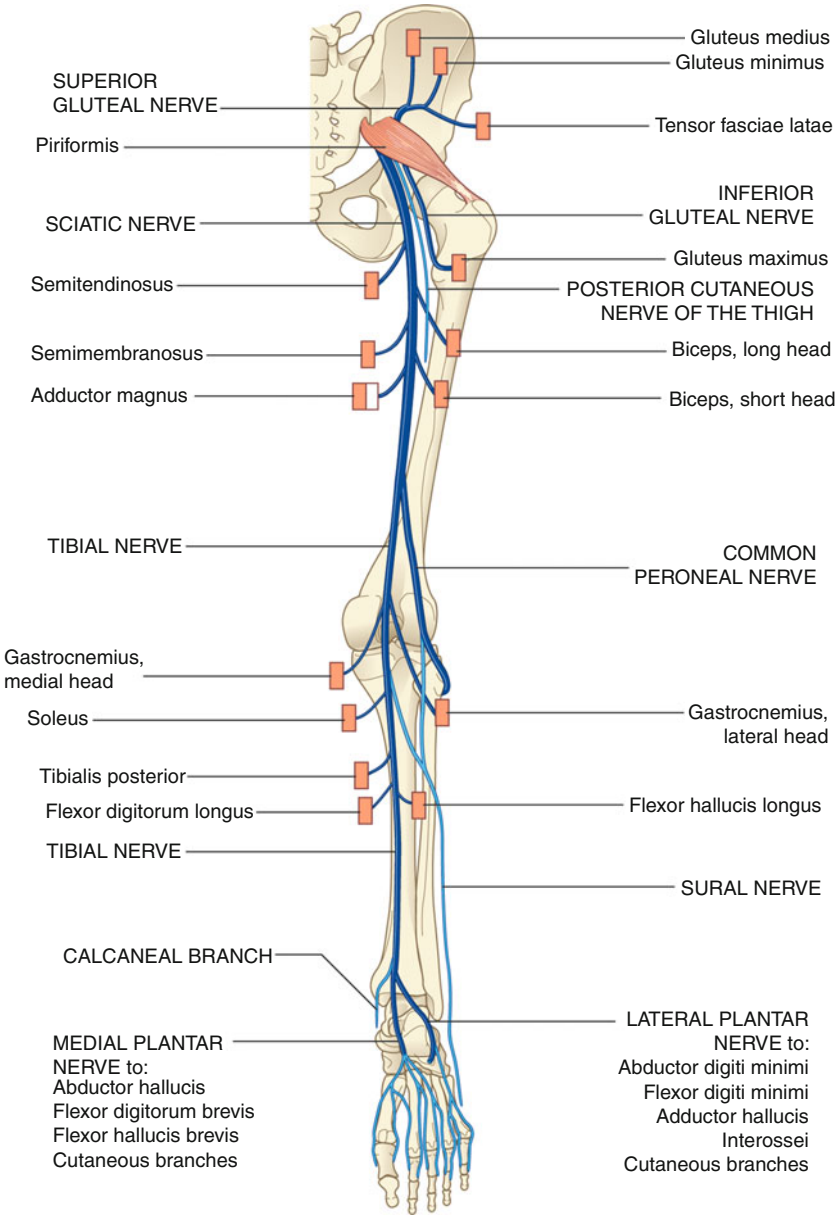

Fig. 1.28 The nerves on the posterior aspect of the right limb (From *Aids to the Examination of the Peripheral Nervous System*. 4th ed. By kind permission of Dr. Michael O'Brian and Elsevier Ltd)

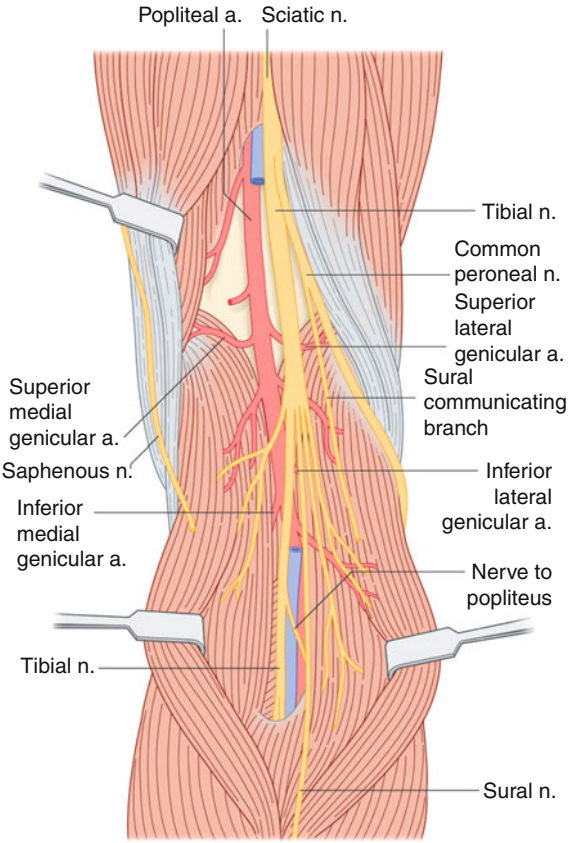

Fig. 1.29 The right popliteal fossa

bundle is relatively fixed here and is at risk by forward displacement of the head of humerus and bleeding from the posterior circumflex vessels strangles the nerve. The radial nerve is at risk from fractures of the shaft of the humerus between the two relatively fixed points of the nerves to the lateral head of triceps and the tunnel through the inter muscular septum. The common peroneal nerve, which passes above or through the piriformis in as many as 30 % of cases, is tethered above in relationship to the piriformis and below at the neck of the fibula. The fascia surrounding the biceps femoris muscle and tendon sweeps around to embrace the nerve. The deep peroneal nerve passes rather acutely forward to enter the anterior compartment of the leg.

Sleeves of fascia surround main nerves and main vessels in some regions, an arrangement which predisposes the nerve to injury from ischaemia or compression or both, from bleeding. The anterior primary rami of C7, C8 and T1 are enclosed in quite a rigid space after they enter the posterior triangle of the neck. This is bounded, posteriorly, by the dorsal part of the first rib, the transverse processes of the cervical vertebrae and by the fascia of the levator scapulae muscle. The nerves are embraced by the scalenus anterior and scalenus medius muscles, both of which are invested in

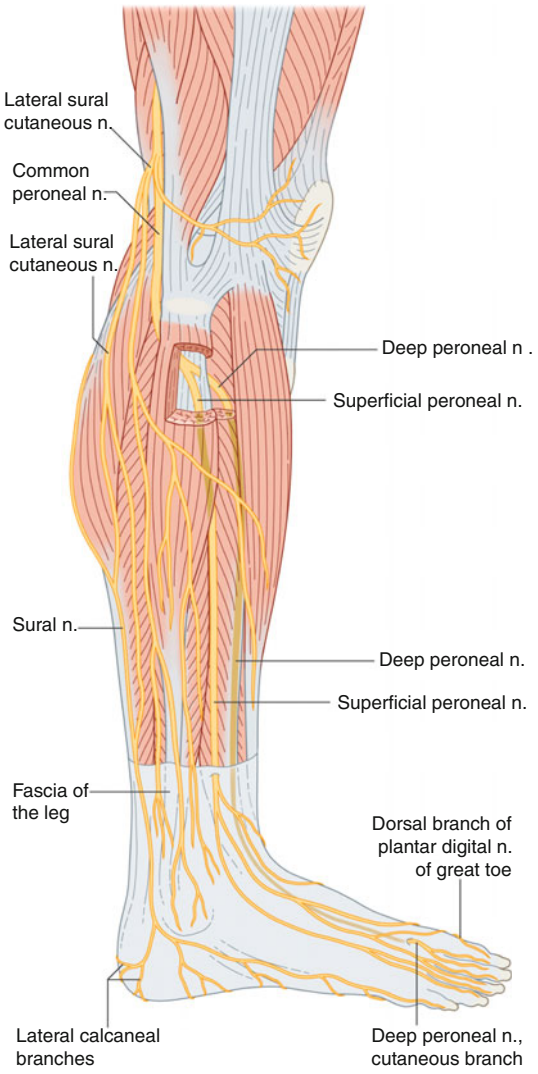

Fig. 1.30 The course relations and branches of the right common peroneal nerve

an unyielding fascia. This is one envelope of the prevertebral fascia which also serves to bind the phrenic nerve down to the anterior face of scalenus anterior. The pre vertebral fascia is particularly well developed in front of the vertebral column and also at the base of the posterior triangle where it envelops C7, C8 and T1, the phrenic nerve, the cervical sympathetic chain, and the subclavian and vertebral arteries. Infusion of relatively large volumes of fluid, from 10 to 20 mL, deep to the prevertebral fascia for the purpose of inducing regional block may cause tamponade of the radicular vessels which enter the spinal canal and contribute to the anterior spinal artery.

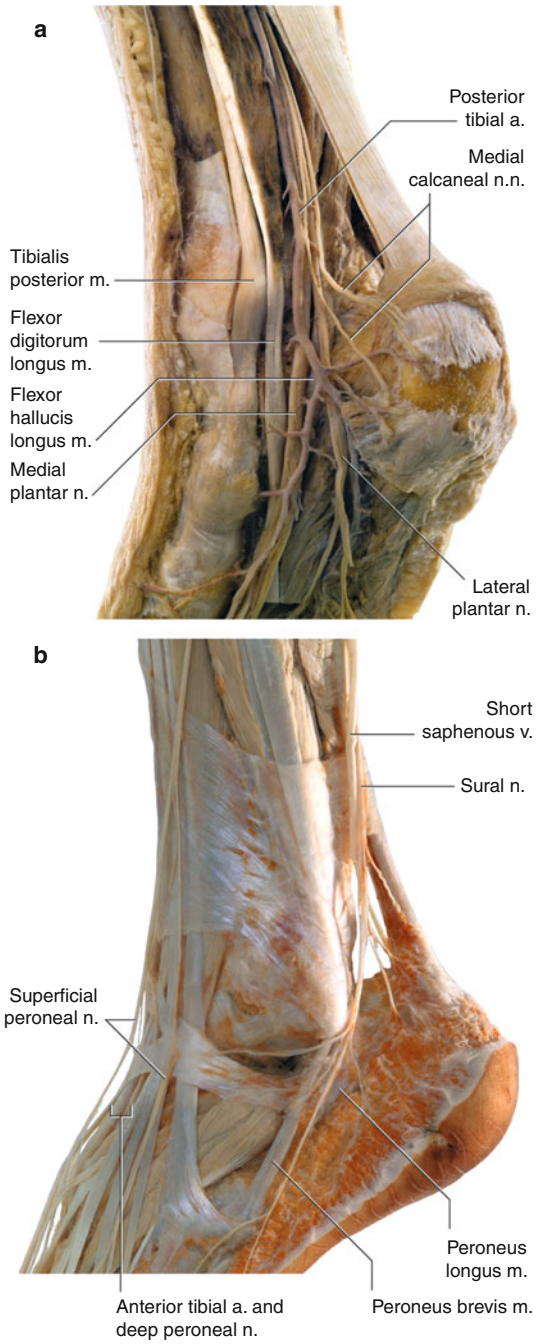

Fig. 1.31 (**a**) *Above*: the medial aspect of the right ankle. (**b**) *Below*: the lateral aspect of the left ankle and heel

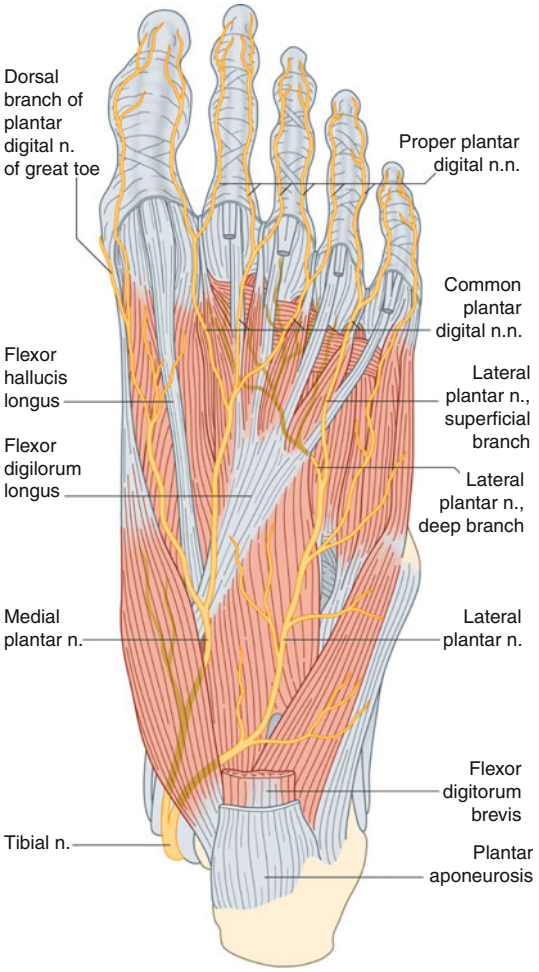

Fig. 1.32 The sole of the left foot

The medial brachial fascial compartment extends from the axilla to the elbow, and is bounded by the tough medial intramuscular septum and the axillary sheath. Bleeding into this compartment is responsible for the majority of infraclavicular plexopathies following regional block and possibly for many of the neurovascular injuries which result from closed or penetrating missile injuries into this region. The anterior interosseous nerve and its accompanying artery may be damaged by compression because of swelling in the deepest part of the flexor compartment of the forearm (Fig. 1.36). The ulnar nerve is accompanied by the ulnar artery, in a discrete fascial compartment in the distal two thirds of the forearm. The deep peroneal nerve is accompanied by the anterior tibial artery, an end artery, throughout most of

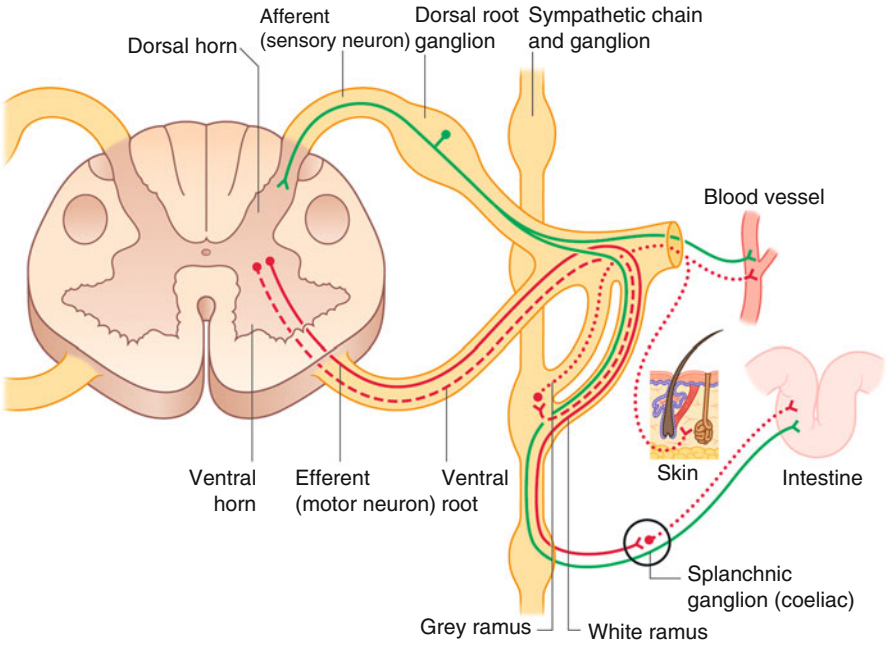

Fig. 1.33 Efferent (*red*) and afferent (*green*) autonomic paths in the spinal cord and ganglionic chain

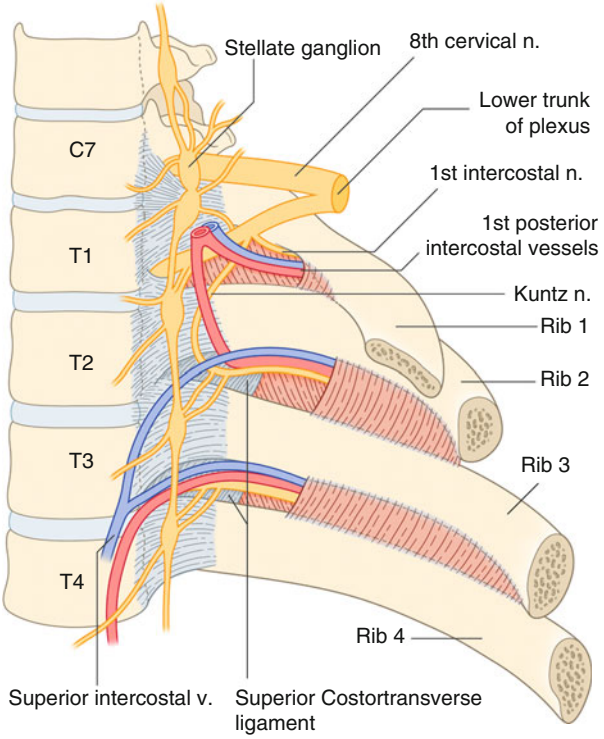

Fig. 1.34 The relations of the cervico thoracic (stellate) ganglion

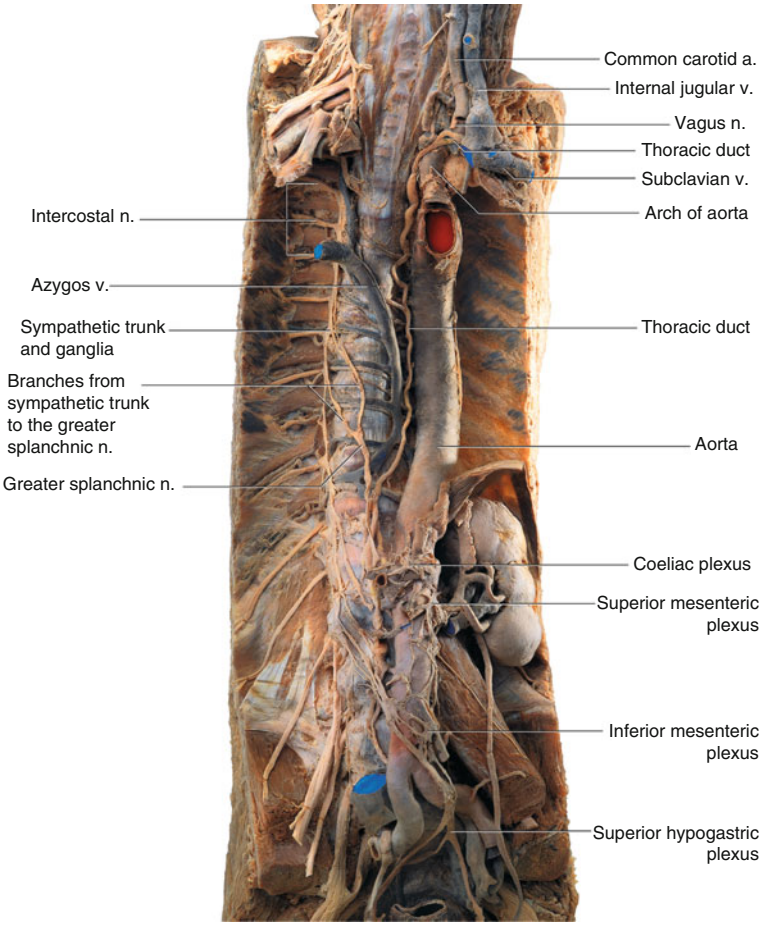

Fig. 1.35 The autonomic nerves in the chest and abdomen

the anterior compartment of the leg (Fig. 1.37). The tibial nerve is accompanied by the posterior tibial artery in the distal one half of the leg in a sheath of fascia similar to the arrangements for the ulnar vessels and nerve. The femoral nerve is damaged by haematoma where it passes deep to the thick fascia over the iliacus muscle. The nerve is especially at risk from bleeding into the femoral triangle (Fig. 1.38).

1.5 The Neurone

The essential component of the system is the neurone, the nerve cell body with its dendrites and its prolongation, the axon. The neurones are the only elements in the nervous system which conduct nervous impulses. There is no continuity between

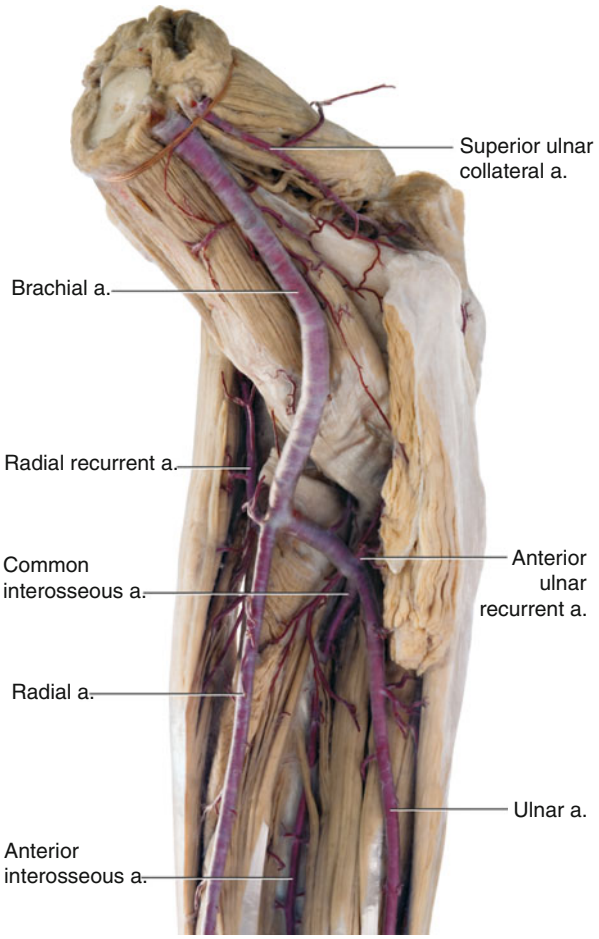

Fig. 1.36 The collateral vessels at the elbow accompany the nerves. The specimen is the left elbow

nerve cells: the termination of the axon on a cell is no more than a contact, the synapse (Fig. 1.39). The neurone is a structural unit; it also behaves as a trophic unit; and it is dependant upon trophic support during maturation and after injury to the nerve. This support is provided by the neurotrophins.

1.5.1 The Neurotrophins

Windebank and MacDonald [45] defined growth factors as "soluble extracellular macromolecules that influence the proliferation, growth and differentiation of target cells by a cell surface receptor mediated mechanism". Most neurotrophins are polypeptides which are produced in tissues such as skin or muscle from whence they are transported to the neuronal cell body by the fast centripetal component of axonal transport. Interruption of this system by division of the nerve contributes to cell

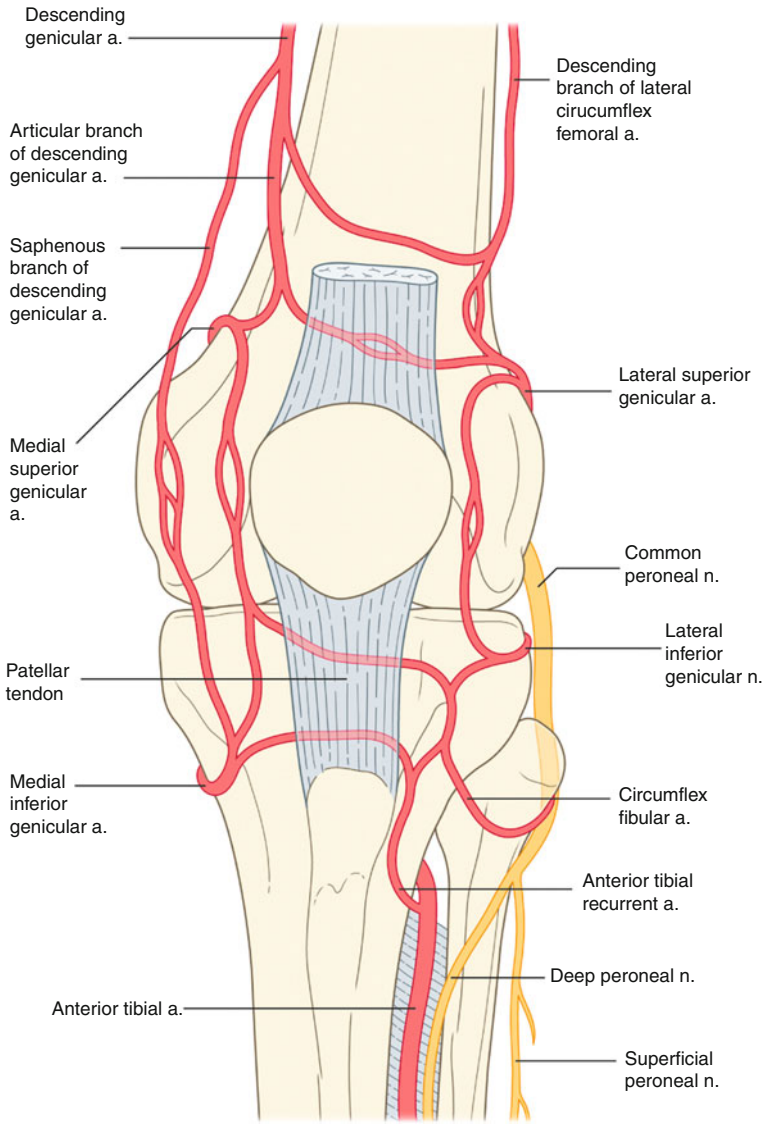

Fig. 1.37 The collateral circulation about the knee is poor. The drawing shows the left knee

death amongst central neurones, an effect which is more severe in the immature nervous system and after axonotomy close to the neuronal cell bodies. Three major families of growth factors are recognised [6, 45].

1. The classic neurotrophins include nerve growth factor (NGF), brain derived nerve factor (BDNF) and the neurotrophins 3–7 (NT3, 4, 5, 6, 7) (Fig. 1.40). NGF is produced by cells including keratinocytes, melanocytes, vascular and smooth muscle cells, testis and ovarian cells, and endocrine and exocrine tissue.

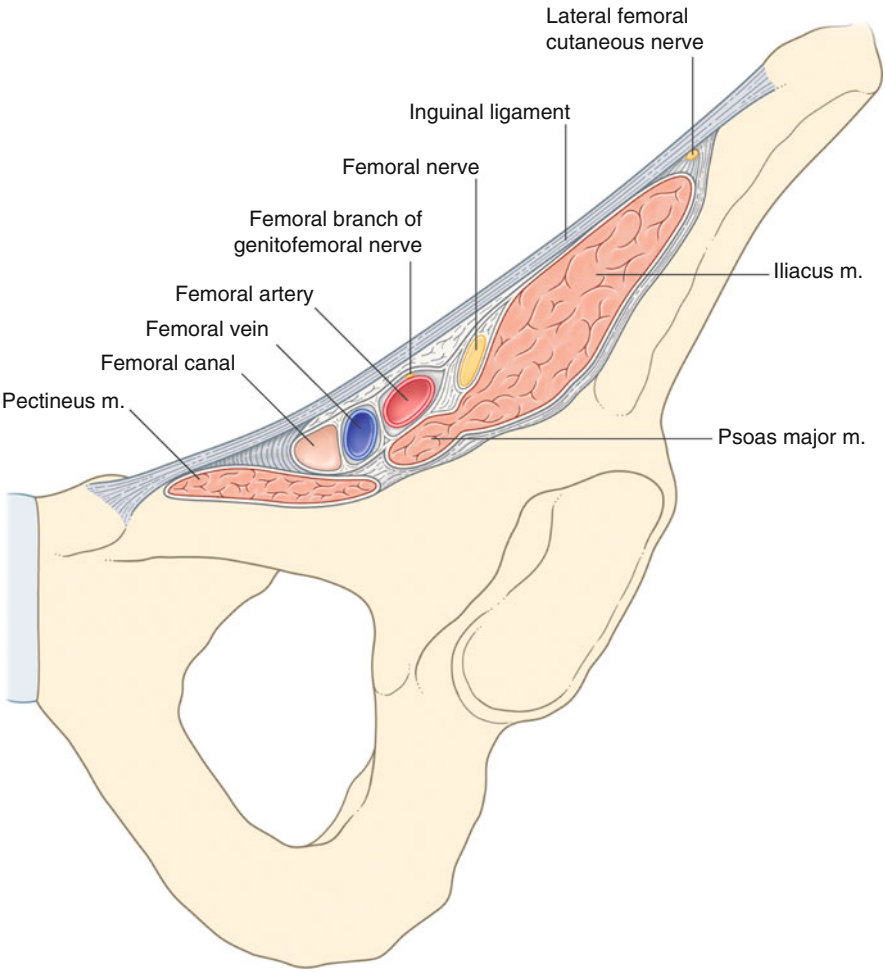

Fig. 1.38 The femoral nerve and vessels deep to the inguinal ligament. The drawing shows the left inguinal region

NGF interacts with the high affinity receptor p140 tyrosine receptor kinase (TrkA) which is expressed by sympathetic neurones and by small diameter neurones in the dorsal root ganglia. After nerve injury, cells in other tissues, including Schwann cells and fibroblasts, synthesise and release NGF. Mice experimentally engineered to be deficient in TrkA do not develop thermoceptive or nociceptive neurones. Administration of NGF induces thermal and mechanical hyperalgesia, it is upregulated by inflammation and plays a key role in the pathophysiology of nociception [2]. BDNF apparently supports the development of motor neurones and their survival after axonotomy. Neurotrophin 3(NT3) is mainly expressed in muscle spindles, Merkel cells and the Golgi tendon organs.

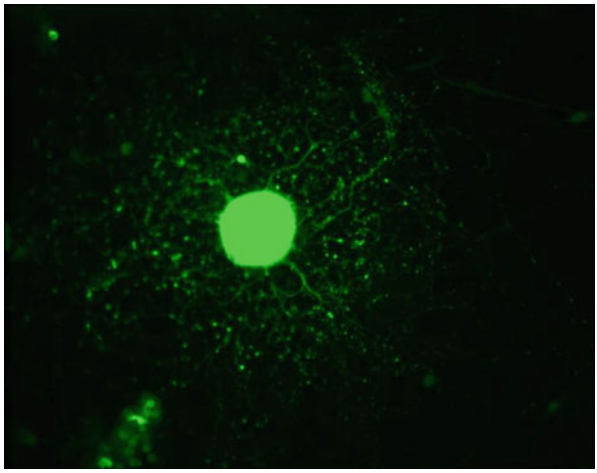

Fig. 1.39 Cultured human dorsal root ganglion neurone immunostained for Gap 43 (growth associated protein) showing the cell body and neurites arising from the cell body ×40

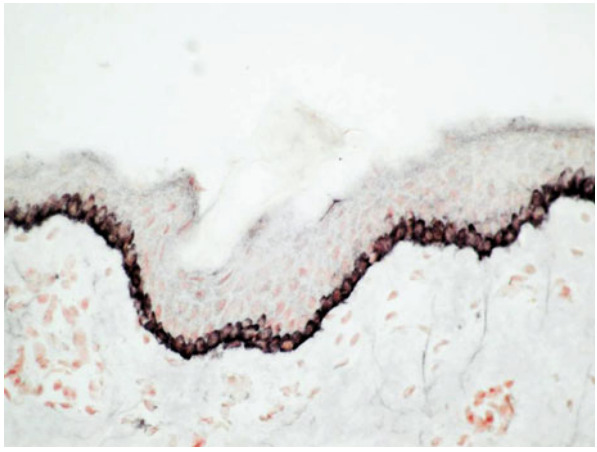

Fig. 1.40 Nerve growth factor (NGF) immunostaining of basal epithelial cells in human glabrous arm skin ×40

This neurotrophin specifically binds to tyrosine receptor kinase C (TrkC). Mice genetically engineered to lose this receptor lack proprioceptive organs.

2. Other neurotrophins are synthesised by the glial cells. It is likely that these factors support the embryonic midbrain and motor neurones in the spinal cord. The glial derived nerve factor (GDNF) binds with its high affinity receptor and also the tyrosine kinase receptor c-Ret (Fig. 1.41). The ciliary neurotrophic factor (CNTF) binds to its receptor and also the leukaemia inhibitory factor receptor beta (LIFR β). CNTF supports neurones in the ciliary ganglion, dopaminergic neurones, retinal rods and sympathetic and motor neurones.
3. The third family include the insulin growth factor (Igf) which structurally resembles insulin and binds with the tyrosine kinase IGF-I receptor, which is itself homologous to the insulin receptor. This receptor is expressed throughout the nervous system.

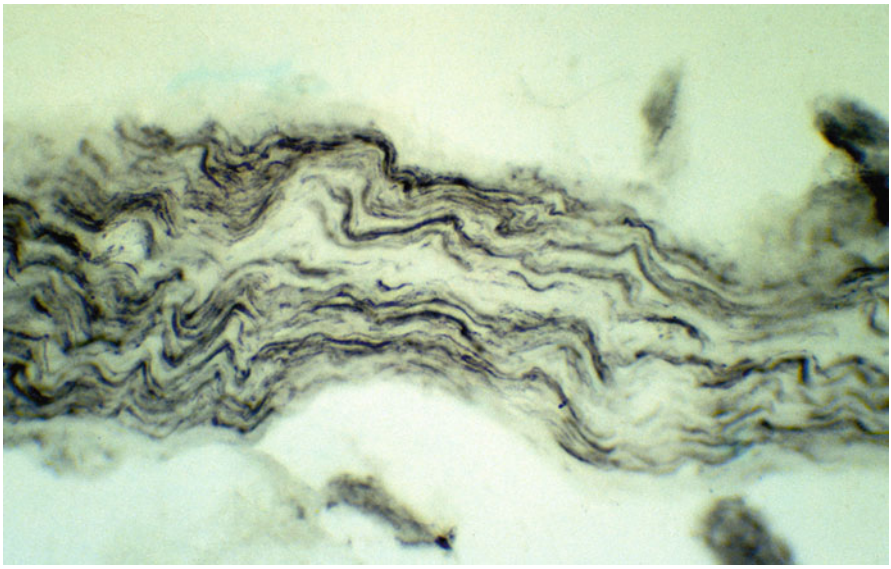

Fig. 1.41 GDNF immunostaining in Schwann cells of healthy human sural nerve ×150

1.6 The Nerve Fibre

In the central nervous system the neurons are supported in a network of oligodendrocyte and astrocyte processes, with very little extracellular space. The structure of peripheral nervous tissue is one of nerve fibres (axons – Schwann cell units) suspended in a collagen rich extra cellular space. The transition from central to peripheral nervous structures takes place in the rootlets or less often in the roots of the spinal nerves. This is the *transitional region* or *transitional zone* (*TZ*). The extension of CNS structure into the base of the rootlet is cone-shaped [14] (Fig. 1.42). The axolemma and the basal lamina surrounding the Schwann cell – axon unit extends into the spinal cord and remains continuous through the TZ. In preganglionic injuries of the brachial plexus the spinal nerves are usually torn at the level of the roots or rootlets [32].

Although most of the fibres of the ventral roots have their cells in the ventral horn of the grey matter there are also myelinated afferent fibres which have cell bodies in the dorsal root ganglion (DRG) [31] (Fig. 1.43). The fibres of the dorsal roots have their cell bodies in the dorsal root ganglion and are unipolar in form with a single axon and no true dendrites. Each axon bifurcates into peripherally and centrally directed axons after leaving the cell body. The centrally directed branches are of smaller calibre than the peripheral ones and enter the spinal cord along the posterolateral sulcus. In the cord the fibres bifurcate into ascending and descending branches. Both branches of the smaller fibres in the lateral part of the root reach the dorsal horn of the grey matter, where they terminate having traversed between three and five segments. The branches of the larger fibres in the medial part of the root, similarly bifurcate after entering the white matter just medial to the dorsal horn. Some ascending fibres reach as high as the gracile and cuneate nuclei in the caudal

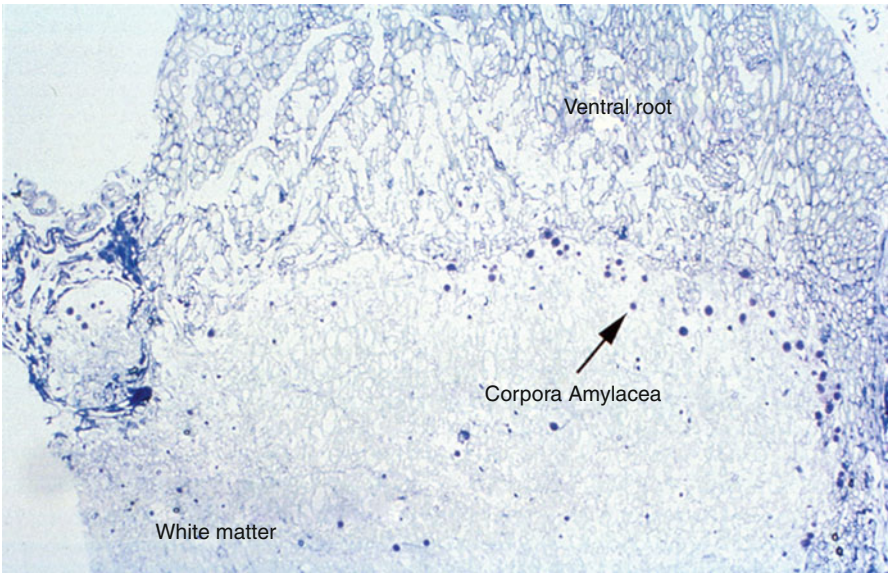

Fig. 1.42 Morphology of the normal human spinal cord. A transverse light microscopic section at C7 showing a ventral root. A single large root in transition is demonstrated with central islands of autolysed glia. Numerous corpora amylacia are seen on the central side of the transitional zone. *Toluidine blue* ×100 (Courtesy of Editor *Journal of Bone and Joint Surgery* (British))

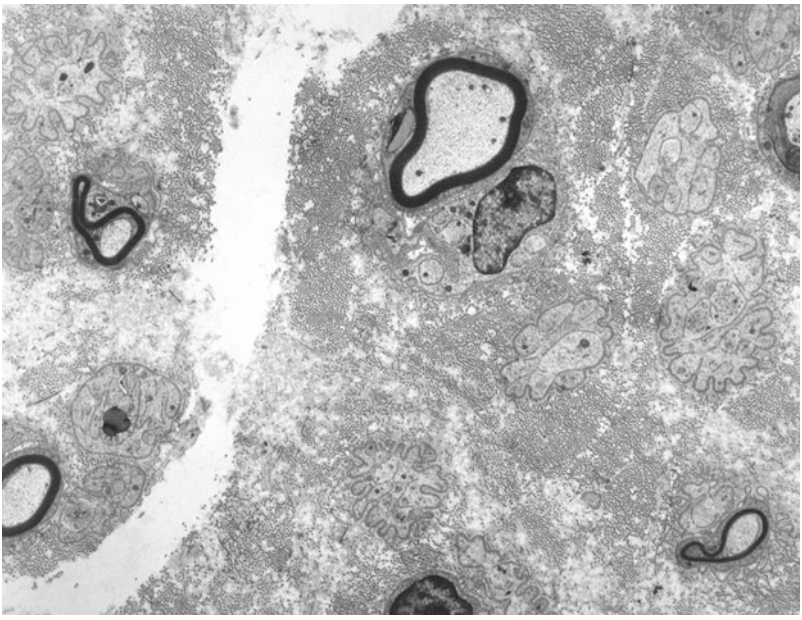

Fig. 1.43 Afferent and efferent fibres in the ventral root. Large healthy myelinated and unmyelinated fibres in the ventral root of the eighth cervical nerve avulsed from the spinal cord 6 weeks previously. The myelinated efferent fibres have undergone Wallerian degeneration and there is much collagenisation ×5,000 (Electronmicroscopic study EM)

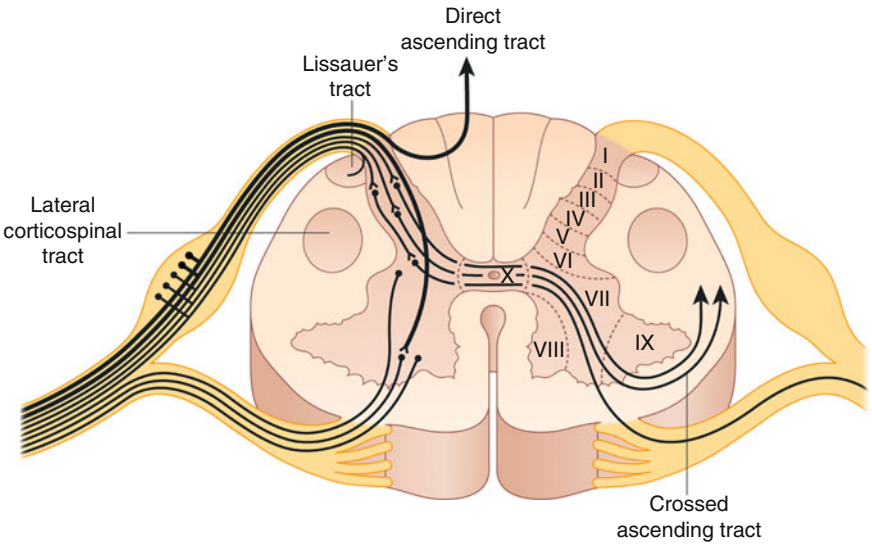

Fig. 1.44 The paths of the afferent fibres entering and efferent fibres leaving the spinal cord. Note (*right*) the laminae of the grey matter

part of the medulla. Other fibres of this group have short ascending and descending branches which enter the grey matter of the dorsal horn to establish synapses with nerve cells in its different laminae (Fig. 1.44).

1.6.1 The Axon

The axon is a column of neuronal cytoplasm, the axoplasm enclosed by a cell membrane, the axolemma. The axoplasm is a fluid cytosol containing formed elements, notably the cytoskeleton consisting of neurotubules, neurofilaments and matrix. In addition, there are mitochondria, axoplasmic reticulum, lamellar and multivesicular bodies, and membranous cisterns, tubes and vesicles. The cytoskeleton provides the apparatus for axoplasmic transport. The axolemma is a three-layered unit membrane about 8 nm thick. It conveys signals between the neurone and its Schwann cells which control their proliferative and myelin producing functions [4] (Fig. 1.45).

1.6.2 Axonal Transport

Axonal transport is a system of intra cellular motility which enables nerve cells to deliver proteins membrane components and neurotransmitters, to the periphery,

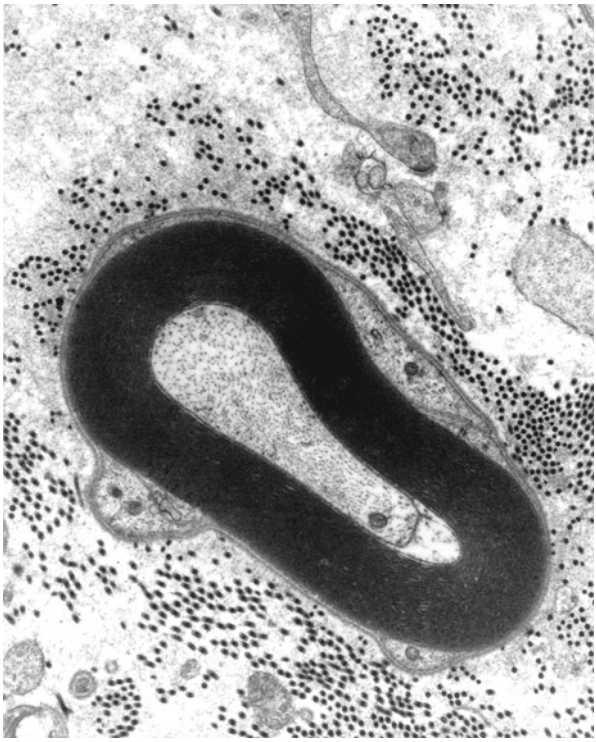

Fig. 1.45 A large myelinated nerve fibre. It lies within the posterior root of the seventh cervical nerve which had been avulsed from the spinal cord 6 weeks previously. The axoplasm contains neurofilaments and a few neurotubules. The Schwann cell cytoplasm it is enveloped by a well defined basal lamina. There are processes from fibroblasts from within the endoneurium ×16,200 (EM)

and to return chemical signals, materials for recycling and some neurotrophins to the cell body. Two forms of transport, fast and slow, are recognised [7]. All systems are ATP dependant, and the microtubules are critical for fast axonal transport. The process is sensitive to temperature; it is sensitive to deprivation of oxygen.

The fast retrograde (centripetal) component conveys material to the cell body in microvesicles at the rate of 150–300 mm a day. The fast orthograde (centrifugal) component transports materials from the cell body at a rate of 200–400 mm/day.

Slow transport is uni-directional, orthograde (centrifugal). Rates of transport are from 1 to 4 mm daily; it is concerned with the transport of the neurotubule-neurofilament network of the cytoskeleton. There are two distant components [7].

1. Slow component A (SCa averaging about 1 mm a day)
2. Slow component B (SCb averaging 2–10 mm/day).

The rate of transport of the SCa component is about the same as the rate of peripheral regeneration after axonotomy. The significance of axonal transport in disorders of peripheral nerves is plain: interference with the centrifugal process is likely to lead to defect or cessation of conduction; interference with the centripetal process will ultimately lead to degeneration of the nerve cell.

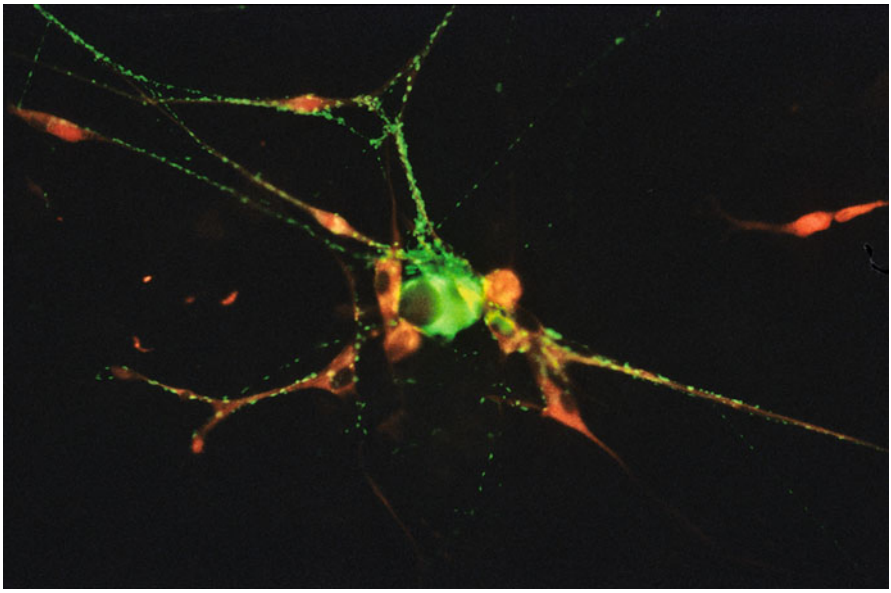

Fig. 1.46 In vitro cultures of DRG neurones and Schwann cells. The Schwann cells are immunostained for S100 (*red*) the neurones for nerve growth factor (*green*) ×50

1.6.3 The Glial Cells of the Peripheral Nervous System

The glial cells of the peripheral nervous system are essential for the development, maturation, survival and regeneration of the neurone. The myelinating and non myelinating Schwann cells are the main peripheral glial cells. Others include the satellite cells surrounding cell bodies in the dorsal root and autonomic ganglia, the glia of the enteric system, the teloglia (terminal Schwann cells) at the terminals of somatic motor axons and the glia associated with sensory terminals such as the Pacinian corpuscle. About 80 % of nuclei within the endoneurium of a normal peripheral or spinal nerve root are Schwann cells, 10 % are fibroblasts, endogenous macrophages account for between 2 and 9 %. Whilst mast cells are also seen their function is not well understood.

The Schwann Cells arise from the neural crest, from the same tissue that differentiates into peripheral neurones; they provide essential trophic support to the neurone during development and regeneration [18] (Fig. 1.46).

The most important component of the basal lamina is laminin which interacts with receptors including the integrins in the plasma membrane of the Schwann cell. Mice genetically engineered to produce defective laminin or a defective receptor for laminin develop profound nerve pathology and muscle dystrophy. During myelination Schwann cells radically transform their phenotype in response to signals from the larger axons.

The smaller non myelinated fibres are contained in bundles by columns of Schwann cells (Fig. 1.47).

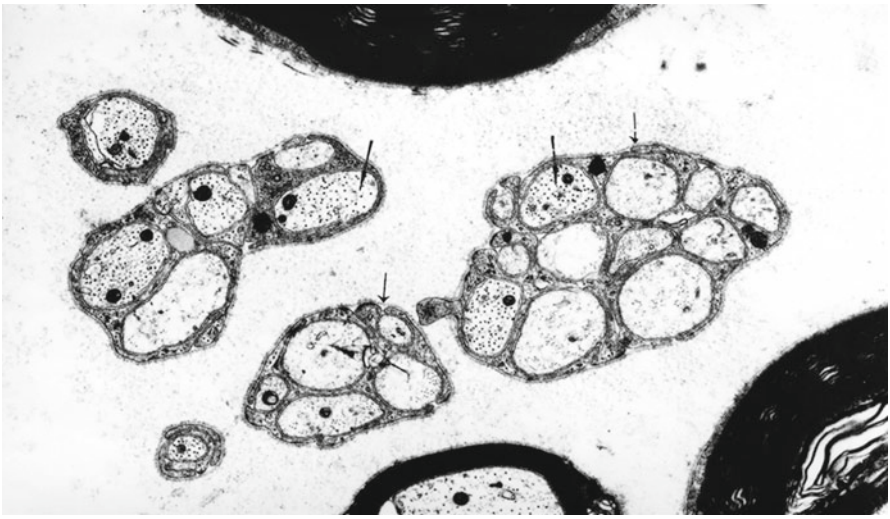

Fig. 1.47 Clusters of unmyelinated axons (*bold arrows*), enveloped by Schwann cell cytoplasm. *Light arrows* indicate basal lamina and Schwann cells ×26,220 (EM)

The larger axons are enwrapped along their length by a continuous series of contiguous Schwann cells into which they are invaginated. The *nodes of Ranvier* represent the points of contiguity of adjacent Schwann cells. The fibre is contained within a basal lamina. The basal lamina separates nerve fibres from the endoneurial space and it runs without interruption from the central nervous system to the termination of the axon. It is approximately 250 Å in thickness which is separated from the plasma membrane of the Schwann cell by a gap of 250 Å. The endoneurium is organised in two layers which surround the basal lamina. The inner layer is composed of collagen fibres of smaller diameter than those in the outer layer which run longitudinally, circularly and obliquely. This layer is inflected at the nodes with the basement membrane. The outer layer consists solely of longitudinal collagen fibres and it is not inflected at the node.

1.6.4 The Myelin Sheath

The diameter of the axon is one important factor which determines whether the Schwann cells will lay down a myelin sheath around it. The multilamellar sheath has a high lipid content and some protein components [40]. The major component of the protein components of myelin is Po myelin protein zero, (MPZ) which accounts for 50–60 % of all myelin protein. Peripheral myelin protein 22 (PMP 22) comprises from 2 to 5 % of myelin proteins and mutations of the controlling gene lead to inherited myelin disorders. Myelin basic protein (MBP) accounts for 5–15 % of myelinated proteins. The myelin associated glycoprotein (MAG), which forms

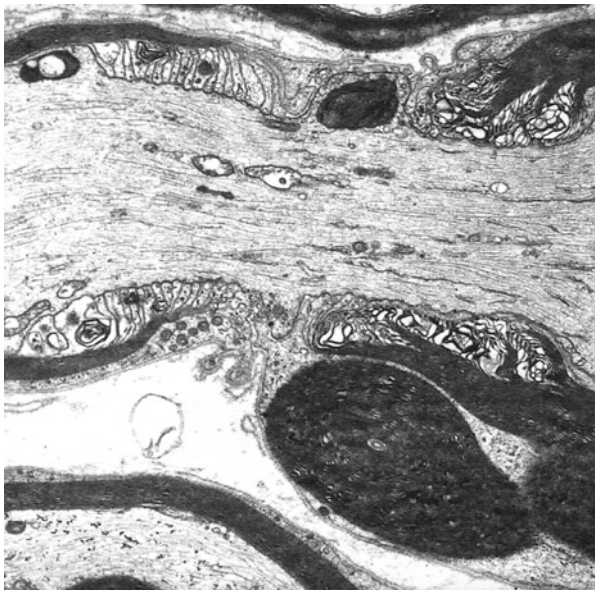

Fig. 1.48 Longitudinal section through a node of Ranvier, showing a remyelinated heminode (*left*) adjacent to a normal heminode (*right*). Compare the complexity of the paranodal fingers of the normal myelin sheath with the simple arrangement of the paranodal loops of the thinner remyelinated sheath ×5,000 (EM)

no more than 0.1 % of the myelin proteins, may play a pivotal role during myelination because of its early expression and because of its location to the axon-Schwann cell interface. The myelin sheath is traversed by cytoplasmic channels – the incisures of Schmidt-Lanterman. *The nodes of Ranvier* are short, about 1 μm in length and the axon here is constricted, free of myelin but enveloped by projections of Schwann cytoplasm. The node is bordered by an adjacent paranode, which is dilated and which contains an increasing amount of mitochondrial rich Schwann cell cytoplasm outside a more or less crenated myelin sheath. Berthold, King and Rydmark [4] characterise the node thus: "these parts of the myelinated nerve fibres; the paranode-node-paranode (PMP) regions; constitute, structurally as well as functionally, the most spectacular parts of a myelinated nerve fibre". The PMP regions are responsible for the generating and propagation of the action potential and they are the centres for activity in the early phases of Wallerian degeneration and collateral sprouting (Figs. 1.48 and 1.49).

1.6.5 Conduction

The special property of the nerve fibre is that of conducting a signal in the form of a propagated action potential This a brief, self propagating reversal of membrane polarity and it depends on an initial influx of sodium ions which cause a reversal of polarity to about +40 mV followed by a rapid return towards the resting potential, −80 mV, as potassium ions flow out. The action potential is evoked by a stimulus

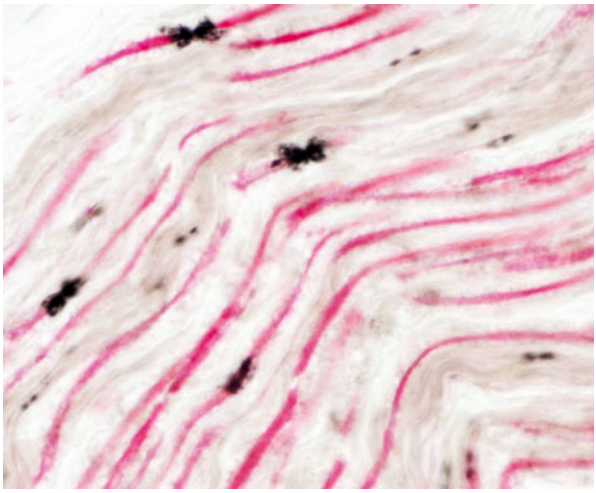

Fig. 1.49 Double immunostaining of sural nerve showing nodes of Ranvier (*black*) stained with antibodies to junction adhesion molecule (JAM-c) and axons (*red*) stained with antibodies to neurofilaments ×40

which exceeds threshold by the all or nothing law; the cell body, on the other hand, responds in a graduated manner to stimuli transmitted across synapses which either inhibit or facilitate by raising or lowering the threshold respectively. In the unmyelinated fibre, a wave of depolarization spreads continuously along the axon, attenuated by the large capacitance of the axolemma, which limits the velocity of conduction to about 1 m/s. Standring [37] likens this to: "a flame moving along a fuse. Just as each segment of the fuse is ignited by its upstream neighbour, each segment of axon membrane is driven to threshold by the depolarization of neighbouring membrane. Sodium channels within the newly depolarized segment open and positively charged sodium ions enter, driving the local potential inside the axon towards positive values. This inward current in turn depolarizes the neighbouring, downstream, non depolarized membrane, and the cyclic propagation of the action potential is completed".

In the myelinated fibre the high resistance and lower capacitance of the myelin sheath limits depolarisation to the membrane of the axon at the node, so that current is directed towards the next node, exciting it in turn. The action potential jumps from node to node (saltatory conduction) which greatly increases the conduction velocity. The calibre of myelinated axons varies from 0.4 to 1.25 μm that of myelinated fibres from 2 to 22 μm. The largest, fastest conducting elements are the myelinated fibres of around 20 μm diameter concerned with somatic afferent and efferent activity; the smallest and slowest conducting are the fibres of around 1 μm diameter that subserve autonomic activity and delayed pain sensibility.

Conduction velocity ranges from about 0.7 m/s in small unmyelinated fibres to about 80 m/s in the largest myelinated fibres. The electrical changes associated with the wave of depolarisation can be measured through electrodes placed on the skin over the nerve, on the nerve, in the nerve or in individual fibres. These reactions form the basis for electrophysiological examination and for microneurography.

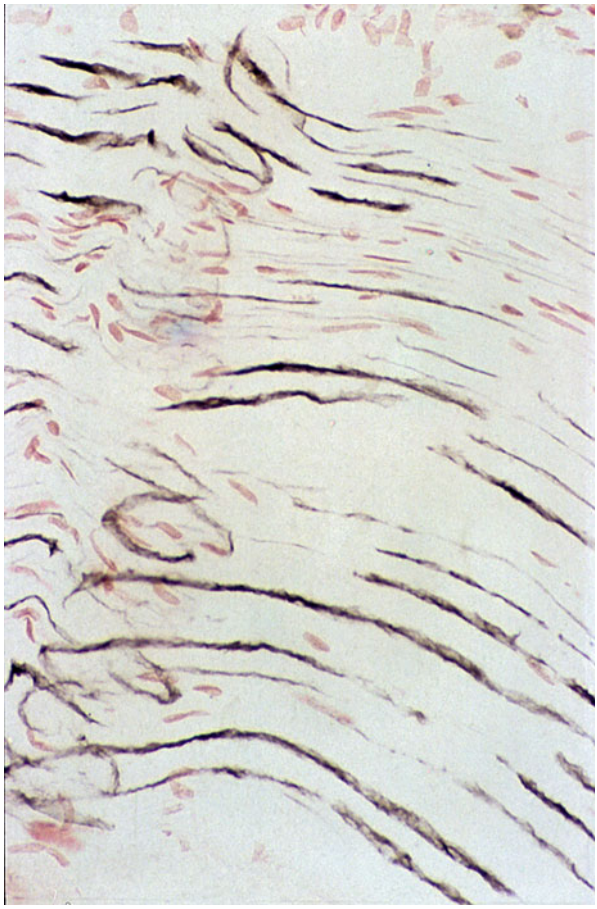

Fig. 1.50 Sodium channel staining normal sural nerve ×40

1.6.6 The Basis of the Action Potential: Ion Channels

In 1952 Hodgkin and Huxley [19] described the cycle of depolarisation and repolarisation which underlies the high speed transmission of nerve action potentials and showed that the reversal of polarity was brought about by the influx and efflux of sodium and potassium ions across the axon membrane through individual parallel pathways which are controlled by independent gating particles or charges, now known as voltage gated ion channels. These are like membrane lodged proteins that mediate rapid ion flux (10^6 ions/s) across cell membranes [9]. Sodium and potassium ion channels are fairly evenly distributed along the axon membrane of nonmyelinated fibres. The sodium channels are densely concentrated at the nodes of Ranvier in the myelinated nerve fibres, whereas the potassium channels are concentrated in the axon membrane at the juxta paranode (Fig. 1.50). Ion channel function is energy dependent, it is ATP driven and this function is curtailed or altogether blocked by anoxia. Distortion of the myelin sheath adjacent to the node of Ranvier

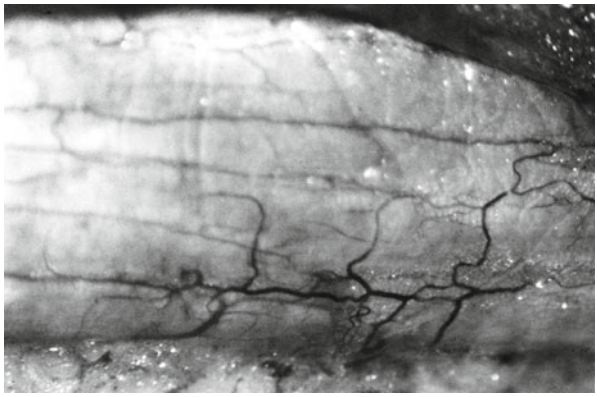

Fig. 1.51 The extrinsic epineurial vessels of the ulnar nerve ×40

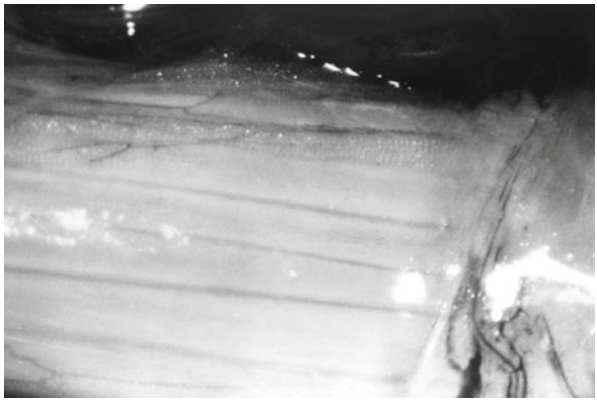

Fig. 1.52 The bundles and epineurial vessels of the ulnar nerve after displacing the adventitia ×40

may unmask the potassium ion channels to such an extent that prolonged conduction block ensues. Demyelination is bound to lead to slowing of conduction and eventually to conduction block. Anoxia blocks conduction within 1 h.

1.7 The Peripheral Nerve Trunk

A normal peripheral nerve trunk exposed at operation is enveloped in a well defined translucent envelope. This is the external epineurium (Fig. 1.51). Normal nerve trunks are easily distinguished from other longitudinal structures by the appearance of white spiral bands on their surface, the spiral bands of Fontana. The individual bundles or fascicles are seen within. These are enclosed by the perineurium with some condensation of the innermost epineurium forming a white, opaque layer (Fig. 1.52). The tissues surrounding the bundle form the epineurium, rather loose in texture, and rich in blood vessels which pass longitudinally along the axis of the nerve. However, the observer will see adventitial material outside the epineurium

which is more clearly defined in some nerves than in others and in different locations within the limb. There are, for example, translucent connective tissue arcades accompanying the median nerve in the forearm where it passes between the superficial and the deep flexor muscles of the fingers. Such vessels provide an alternative collateral pathway to the part; they also supply the nerve trunk so permitting the use, for example, of the ulnar nerve as a free vascularised graft. This tissue plane not only conveys vessels to the nerve but it also permits gliding of that nerve across joints and against the adjacent tissues.

1.7.1 The Connective Tissue Sheaths

The axon-myelin sheath – Schwann cell complexes (nerve fibres) are arranged in bundles otherwise known as fascicles (Fig. 1.53). In so small a nerve as the fourth cranial there may be as many as 3,400 fibres. In the roots inside the spinal canal endoneurial collagen is scanty in contrast with the abundant content in the nerves outside the foramen. The surgeon who has had dealings with nerves inside and outside the spinal canal will appreciate the distinction: the spinal roots and rootlets are fine and fragile and very susceptible to trauma; the peripheral nerves are strong and have much greater resistance to handling. Outside the intervertebral foramina the three supporting structures, epi, peri, and endoneurium are clearly established. The epineurium, in effect the prolongation of the dural sleeve of the nerve roots, is composed of longitudinally directed collagen fibres, fibroblasts and fat cells. The more compact inner layer containing collagen and elastic fibres which are arranged in a wavy pattern. The perineurium, which ensheaths the bundles, is composed of flattened cell processes arranged in a wavy manner alternating with layers of collagen. It provides a barrier to diffusion. The perineurium is strong; the intrafascicular pressure can be raised to more than 300 mmHg before it ruptures The contents of the perineurium are under tension so that when it is cut they are extruded, rather like toothpaste. This is most clearly seen on the day of injury in nerves which have been transected or ruptured and it is one of the indications that the level of section of the stump is adequate. The outflow rapidly diminishes over the course of several days. In the endoneurium, supporting the fibres themselves, there is a return to longitudinal direction of cells and fibres; there are abundant collagen fibrils. The spiral bands of Fontana represent the wavy organisation of nerve fibres. These arrangements provide a degree of protection to the nerve against traction. The nerve can be stretched by as much as 20 % before the wavy arrangement is converted into a linear array.

1.7.2 Topographical Organisation

Sunderland [39] mapped the arrangement of bundles along the course of nerve trunks, showing branching, fusion and changes in number. He also showed the

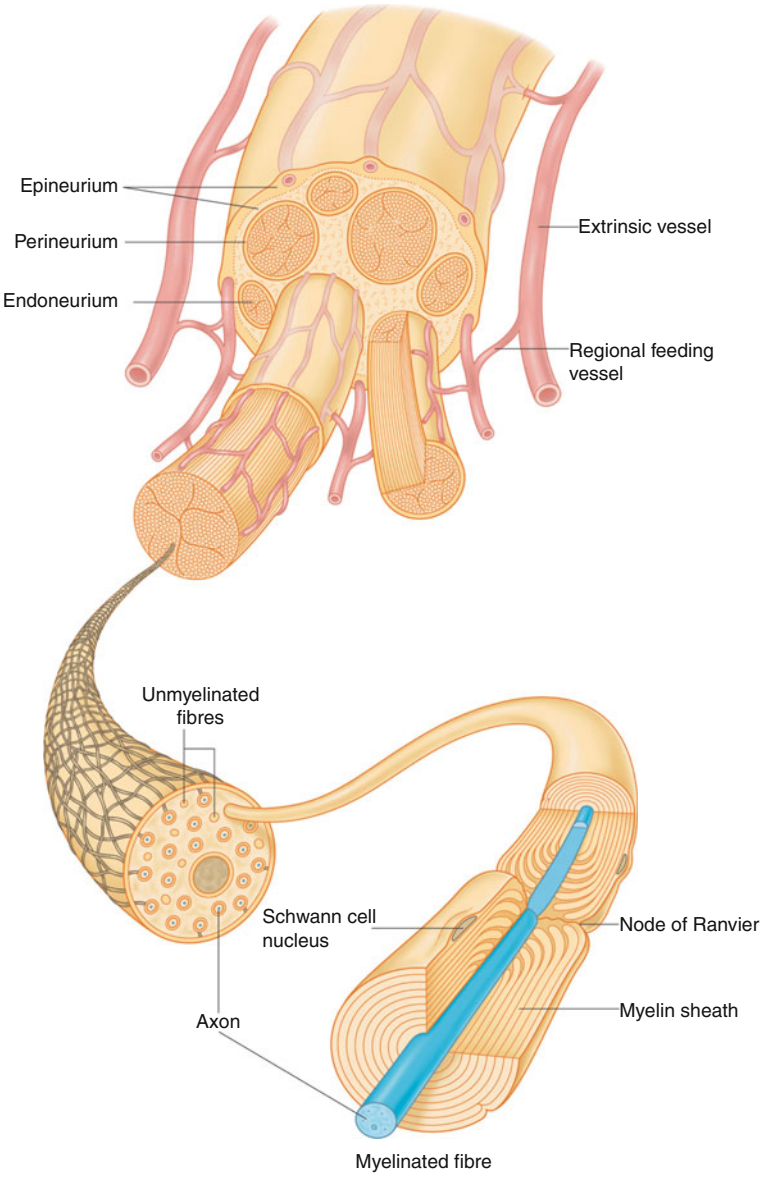

Fig. 1.53 Fascicular arrangement of nerve fibres and their supporting structures, the vascular systems of the peripheral nerve

cross-sectional area of the nerve occupied by connective tissue was variable, ranging from 60 to 85 %. These findings have raised doubts about the feasibility of achieving accurate co-aptation of the ends of divided nerves. However topographical organisation is one essential quality of the nervous system and this is shown by the considerable topographical segregation of neurones involved in the somatic afferent pathways in the dorsal root ganglia, dorsal horn of the spinal cord, the thalamus and

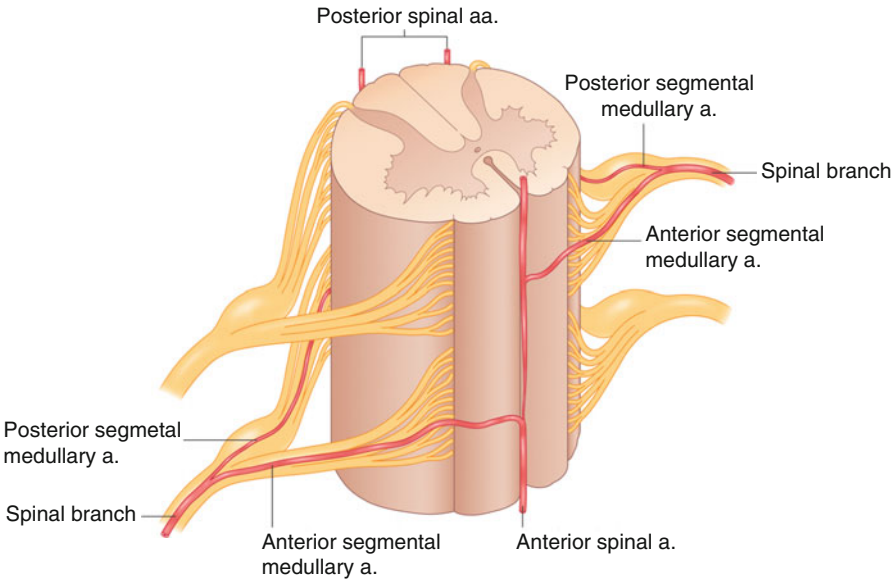

Fig. 1.54 The segmental medullary (radicular) arteries and the anterior spinal artery in the lower cervical cord

the sensory cortex. There is extensive functional topographical segregation of fibres according to function over considerable lengths of the main trunks which permits nerve transfers. The ability to "map" the stump of a divided nerve allied to the ease of matching individual bundles by their size and disposition is but one of the great advantages of urgent repair of nerves.

1.7.3 The Blood Supply of Nerves

Nerves have a very good blood supply: they need it. There are indeed [25] intrinsic epineurial, perineurial and endoneurial plexuses, and extrinsic regional vessels in the "paraneurium". These form "separate but extensively interconnected microvascular systems" providing a wide margin of safety. The richness of the extrinsic supply varies along the course of a nerve and also between nerves [6].

The blood supply to the roots of the spinal nerves is much less robust. Woollam and Millen [46] studied the anterior spinal artery, in the foetus and in the guinea pig and rat. Relatively few radicular arteries survived into adult life. Two of these seemed to be particularly important: a cervical vessel, arising from the vertebral artery and entering into and sustaining the anterior spinal artery at C6, C7 or C8, and the artery of Adamkievicz in the upper lumbar region (Fig. 1.54). Dommisse [13] confirmed that the number of radicular arteries (which he termed the medullary feeders) reinforcing the anterior longitudinal arterial channel was eight and that

those reinforcing the dorsal arterial columns were 17. Only 8 % of those passing to the cervical spinal cord arose from the vertebral artery. The pattern was variable: "but the principle of a rich supply for the cervical and lumbar enlargements was confirmed". The anterior spinal artery is the most important of the longitudinal channels its central branches, which are end arteries, supply about two thirds of the cross sectional area of the cord. The rest of the dorsal grey and the white columns are supplied by branches arising from the dorsal arterial system. Disruption, or occlusion of the radicular arteries entering the spinal canal with the spinal nerves or occlusion of the anterior spinal artery leads to the catastrophe of infarction of the anterior cord, the anterior spinal cord syndrome.

1.7.4 The Nervi Nervorum

The nervi nervorum curiously but perhaps predictably, nerves have their own nerve supply in the shape of the nervi nervorum, derived from their own fibres. There are free endings in the epi-, peri- and endoneurium, and some encapsulated endings of Pacinian type in the endoneurium. These are probably one factor underlying the exquisite sensitivity of nerves trapped in fibrosis.

1.8 Changes in Nerves with Aging

Myelination continues for the first 3 years of life and conduction velocity reaches adult levels at 5 years. In infancy there is a higher density of nerve fibres and higher blood flow to that tissue. The neurones in infancy are more vulnerable to the effects of axonotomy or avulsion. With increasing age there is increasing endoneurial collagen with demyelination and degeneration accompanied by remyelination and regeneration affecting the larger, longer MNF and larger sensory neurones. There is a slow decline in conduction velocity after the fourth decade [11]. These changes must evidently concern clinicians treating the very young and the rather old: in the former, they may have a bearing on diagnosis; in the latter, they may be relevant to the susceptibility of a nerve or nerves to damage by pressure or traction.

1.9 The Somatic Motor System

The motor pathway begins in the neurones in the pre-central gyrus of the cerebral cortex. Their axons pass by the internal capsule to the mid-brain and to the pyramids of the medulla. From each side most fibres cross the mid line at the decussation of the pyramids to descend in the lateral part of the white matter of the cord as the

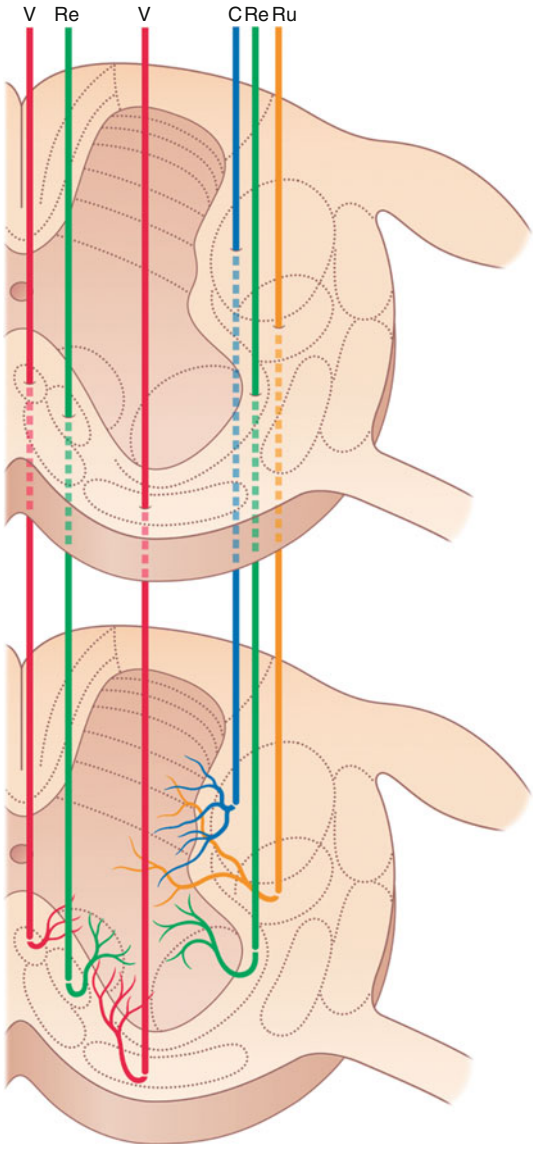

Fig. 1.55 The major descending tracts in the spinal cord and their overlapping zones of termination in the grey matter. *C* corticospinal, *V* vestibulospinal, *Re* reticulospinal, *Ru* ruprospinal

lateral corticospinal tract. At various segmental levels impulses from this tract activate, through internuncial neurons, the motor cells in the anterior part of the grey matter (Fig. 1.55). "Extrapyramidal" tracts from the red nucleus, the vestibular nuclei and the reticular formation also influence the activity of the ventral horn neurones.

The cell bodies of the motor neurons are in Lamina IX [30] of the ventral horn of the grey matter. There are large (alpha) and small (gamma) cells. They are acted on

by primary sensory fibres and by fibres descending from the cortex and from nuclei in the brain stem. The axons from the large cells are destined for the extrafusal fibres. By correlating the distribution of paralysis with the sites of loss of cells in the ventral horn, Sharrard [35] was able to show how the cells were grouped in the grey matter. Broadly, the medial group supply the muscles of the trunk and neck; the lateral group supply the muscles of the limbs. Thus, cells of the latter group are present chiefly in the cervical and lumbar enlargements, whereas those of the medial group are found throughout the length of the cord.

The distribution of nerves within the muscles of the upper limb has been described by Lim and his colleagues [24]. In flat, triangular or trapezoid muscles (class 1) the main nerve runs perpendicular to the muscle fibres giving off side branches that run parallel with them. The spindle shaped or fusiform muscles (class 2) were subdivided into unipennate or bipennate muscles. In the bipennate muscles the aponeurosis of the tendon splits the muscle into two compartments and in these the primary nerve divided into two secondary branches passing each side of the tendon. In muscles with more than one head of origin (class 3) the pattern of innervation is more complex. These findings support the idea of transfer of part of a muscle and they emphasise the requirement for the repair of intramuscular nerves in lacerated muscles.

Contact with, and transmission to muscle is effected through the motor end-plates (see Fig. 1.60) There are two components of each end plate: neural and muscular. They are separated by a cleft of about 30 nm. The muscular sole-plate contains a number of muscle cell nuclei; it is not itself contractile. There are two types of neural endings: the en plaque terminal on extrafusal (alpha nerve fibre) muscle fibres, and the plate endings on intrafusal (gamma nerve fibre) muscle fibres. Transmission at en plaque endings initiates action potentials which are rapidly conducted to all parts of the muscle fibres, whereas transmission at plate endings excites the fibres at several points. Acetylcholine released at the nerve ending interacts with receptors to produce depolarisation of the muscle membrane and trigger the action potential in the muscle.

The ventral roots from the first thoracic to the second lumbar segments of the spinal cord contain also the efferent pre-ganglionic fibres of the sympathetic nervous system: those of the second to fourth sacral nerves contain the efferent pelvic parasympathetic outflow.

1.10 The Somatic Sensory System

The afferent pathways of the peripheral nervous system considerably exceed the efferent pathways in numbers and in complexity of organisation. By no means all lead to conscious sensation. Amongst the somatic afferents the Golgi organs and the muscle spindles are examples; the whole array of the visceral afferents is one more.

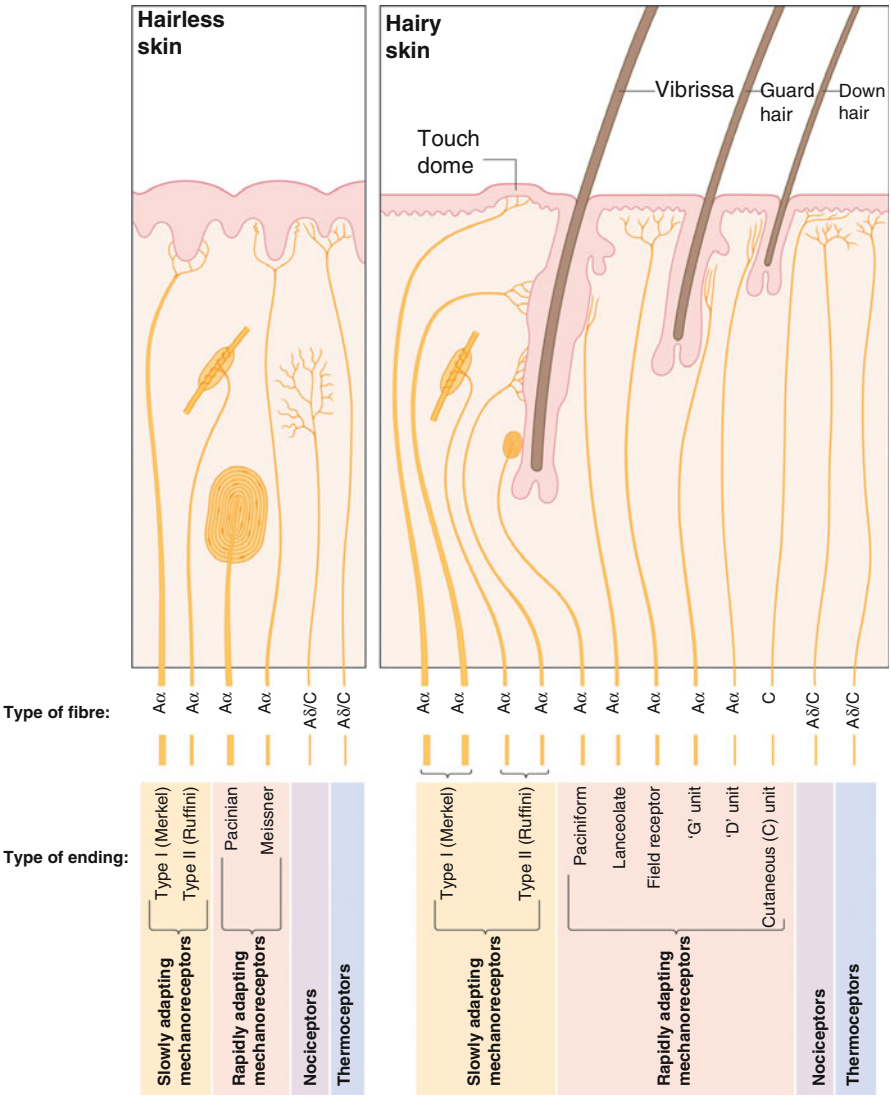

Fig. 1.56 Cutaneous sensory receptors with nerve fibres. (*Left*) glabrous skin; (*right*) hairy skin

1.10.1 Cutaneous Sensibility

There is general agreement that cutaneous receptors have a high degree of selective sensitivity rather an absolute specificity (Fig. 1.56). Adrian and Zotterman [1] pioneered methods in Cambridge in the 1920s which enabled electrophysiological studies of single afferent units. This led to extensive work with microelectrodes and with intraneural microstimulation [42]. No specialised mechanoreceptor transducers transmitting through unmyelinated fibres have been identified yet; the same is not so for nociceptors and most thermoreceptors, where the transient receptor

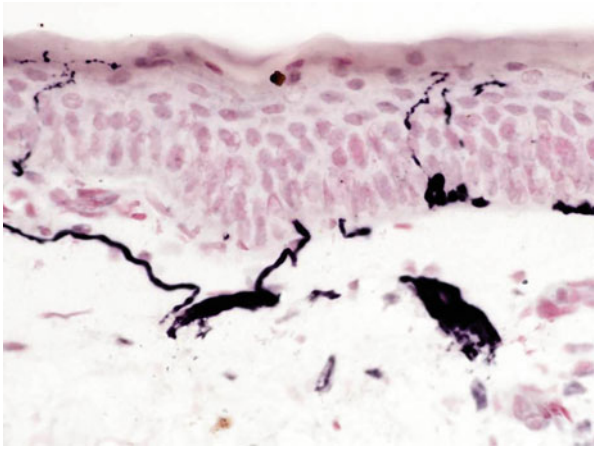

Fig. 1.57 PGP9.5 immunoreactive nerves in skin of a patient with small fibre neuropathy Mag ×40

potential (TRP) channels have been identified as transducing specific ranges of temperature. All these receptors seem to be represented in fine branching unmyelinated nerve endings in the cell layers of the epidermis (Fig. 1.57). The basis of stereognosis is a combination of stimuli from skin, tendons, muscles and joints relaying sensory information where comparison is made from memories of movement. The role of movement is vital: a blindfolded person cannot identify the nature of a material if it is simply placed on the finger. Identification is aided if the material is drawn across the finger tip. Recognition is, however, immediate if the subject is allowed to create temporal and spatial patterns by feeling the texture between the moving finger and thumb.

The fibres of afferent neurones are classed by their conduction velocity. Afferent fibres from the skin are divided into A-αβ, A-δ and C; muscle afferents are classed I, II, III and IV [22, 23]. There is some correlation between fibre diameter and the characteristics of the soma within the dorsal root ganglion. These are classed as large light (neurofilament rich) and small dark (neurofilament poor) neurones. The neurones of C-fibres are small; those with Aδ fibres are small to medium size; those with A-αβ fibres are medium to large (Fig. 1.58).

1.10.2 The Skin

The introduction of immunohistochemical staining of nerve antigens has provided new insights into innervation of the skin described by Kennedy et al. [20] “bundles of nerves enter the skin deep in the dermis and course towards the skin surface, giving off axons to innervate the associated end organs. Unmyelinated nerve fibres comprise the vast majority of cutaneous innervation to the above dermal structures. The few myelinated nerve fibres terminate at hair follicles, Meissner corpuscles and Merkel complexes. The vertically orientated nerve bundles form a horizontal sub epidermal neural plexus. Epidermal nerve fibres branch from this plexus and, while penetrating the dermal-epidermal basement membrane to enter the epidermis, they

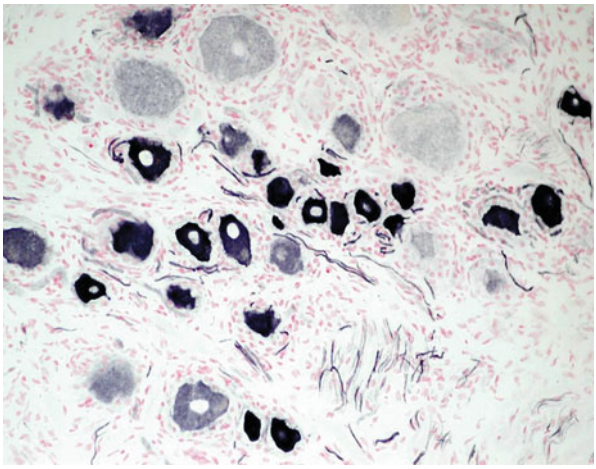

Fig. 1.58 Small diameter nociceptor cell bodies and axons in the human dorsal root ganglion immuno reactive for the heat and capsaicin receptor TRPV1 2 weeks after avulsion injury ×20

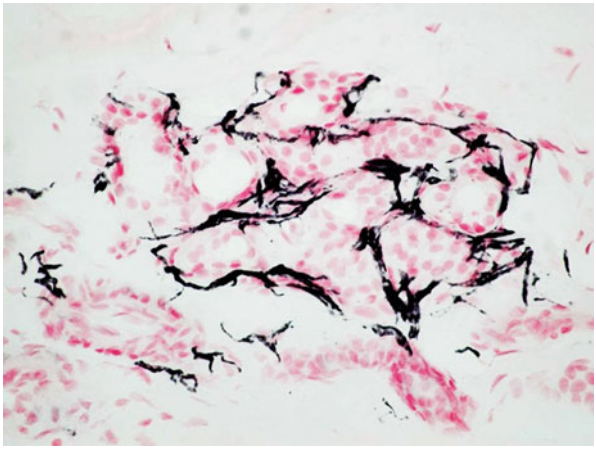

Fig. 1.59 PGP9.5 immunoreactive autonomic nerve fibres surrounding sweat glands in the skin of a patient with small fibre neuropathy ×40

lose their Schwann cells ensheathment and collagen collar". The sweat glands are carpeted by a dense pattern of autonomic nerves (Fig. 1.59). Fine unmyelinated nerve endings form a network covering larger arteries in the deep dermis. The density of innervation of the epidermis is greatest in the proximal segment of the limb. There is little change between the 20th and 80th year.

1.10.3 Cutaneous Sensory Receptors

Three types of cutaneous receptor are defined: low threshold mechanoreceptors; thermoreceptors; nociceptors.

Low threshold mechanoreceptors: The distinction is made between slowly adapting receptors responding to sustained displacement such as continuous pressure;

rapidly adapting receptors responding to the beginning or withdrawal of a stimulus or by a moving stimulus, and receptors responding to brief mechanical disturbances such as vibration and tapping.

The first group includes the Merkel cells; the second includes the Meissner corpuscles and the third, the Pacinian corpuscles. Most are innervated by large and medium sized fibres conducting at rates of from 20 to 90 m/s. The principal mechano-receptor in hairy skin is the hair follicle receptor; in hairless (glabrous) skin the two principal types are the Meissner corpuscle, rapidly adapting, and the Merkel receptor, slowly adapting. Beneath the skin the rapidly adapting organ is the Pacinian corpuscle; the slowly reacting organ is the Ruffini's corpuscle which is found in deep dermal layers and is characterised by large diffuse receptive fields. The Ruffini corpuscles provide information about finger position by responding to stretching of the skin.

Thermoreceptors: Cooling receptors are served by unmyelinated and fine myelinated fibres, usually serving receptor fields about 100 mm in diameter [23]. They are very sensitive to decrease in skin temperature from the normal or "neutral" level of 30–35 °C. Davis and Pope [12] found that the sensation of cooling is replaced by an ache below 17.5 °C and by pain below 14 °C.

Warming receptors, less common than cooling receptors, have receptive fields of less than a millimetre in diameter. Warm sense is a function of unmyelinated fibres within the epidermis Temperatures above 43 °C induce firing in C-fibre polynociceptors. Temperatures above 53 °C evoked responses in fast conducting myelinated mechano-heat fibres.

Nociceptors: The term is applied to primary afferent units which "uniquely signal stimuli intense enough to threaten physical damage to the tissue" [23]. Some respond to intense mechanical stimuli; some to strong thermal stimulation, and some are polymodal. Impulses travel in myelinated fibres in the Aδ to Aαβ ranges and in unmyelinated C fibres.

Nociceptor fibres are widely distributed in the skin, muscle, joints, the epineurium of trunk nerves and the wall of blood vessels as an extensive plexus of free nerve endings. These pass to fine myelinated and non myelinated fibres and also to the largest (Aαβ) fibres [22]. Aδ nociceptors are high threshold mechano-receptors. Some respond to damaging heat. They conduct impulses from receptive fields of about 5 mm^2 at about 20 ms. Many of the C-fibres are polymodal, responding to a range of noxious stimuli including histamine and other chemicals, heat, cutting and crushing. C-fibres are responsible for the triple response of Lewis and they are the basis of the axon reflex. They are less than 2 μm in diameter and conduct at between 0.5 and 2 m/s from fields which range from 1 to 10 mm^2. Microneurography has clarified the physiological characteristics of the nociceptors in humans. Sharp, well localised pain follows stimulation of Aδ afferents. Stimulation of single C-afferents induces dull, burning, poor localised and delayed pain. The reader can experience the two modalities of pain by stimulating the skin on the front of the wrist with a sharp pin. First, and almost immediately, a sharp, well localised pain is experienced. A little later the delayed response – slightly unpleasant, a little longer lasting and a little diffuse – is felt. The A-β and A-δ nociceptors have

punctate superficial receptive fields and respond to noxious mechanical or noxious mechanical and thermal stimuli (Mechano-heat units) [22].

1.10.4 Deep Sensibility

Sensation is conveyed from muscles, ligaments and tendons from specialised receptors and from free nerve endings in those structures. The receptor organs are: in muscle, muscle spindles and free nerve endings; in tendons, the Golgi organs, and in capsules and ligaments various endings, some similar to Ruffini endings, Pacinian corpuscles and Golgi organs. There are also plexuses of unmyelinated fibres (Fig. 1.60).

Joints are innervated by a network of rapidly conducting myelinated fibres some of which are associated with encapsulated mechanoreceptors, by high threshold, slowly conducting fibres many of which are perhaps nociceptors, and by sympathetic afferents [41].

The muscle spindles: It is over a hundred years since Sherrington [36] demonstrated by ventral root section that "in a muscular nerve-trunk from one-third to one-half of the myelinated fibres are from cells of the spinal root-ganglion". The size of these fibres was from 1.5 to 20 μm; they were not the largest fibres in the nerve; the largest came from the ventral root. On the other hand, the largest of these fibres were larger than any fibres in the cutaneous nerves. Muscle spindles signal the length of extrafusal muscle at rest, during contraction and relaxation and the speed of change. Most spindles lie deep in muscle near branches of its nerve or blood vessels. Each contains small muscle fibres (intrafusal fibres) within a cellular and connective tissue capsule. Innervation by large afferent fibres is copious: the muscle spindle and the Golgi tendon organ account for nearly all group 1 afferent axons from muscles. There are three types of specialised intrafusal muscle fibres: the nuclear bag fibres (bag 1, bag 2), with a central accumulation of nuclei, and nuclear chain fibres, smaller and with a single row of nuclei [3]. The spindles vary in length from a few millimetres to a centimetre. Each spindle receives up to 25 terminal branches of motor and sensory axons, together with autonomic innervation. The motor axons are the β and γ axons of the ventral root; the sensory axons are myelinated fibres of groups I and II. The combination of motor and sensory innervation is reflected in the complexity of the function of the spindles. The conception of a servo action is evidently too simple; rather, it is evident that the spindles play several roles in the "feedback" mechanism for regulating muscle contraction and for appreciation of body position. Banks [3] studied the nerve to the soleus muscle of the cat. The nerve contains 180 myelinated sensory and 270 myelinated motor fibres. Most of the myelinated afferents arose from 56 muscle spindles and 45 Golgi tendon organs. There were 115 fusimotor gamma efferent fibres, which means that the 25,000 extrafusal skeletal muscle fibres are innervated by only one third of the total of myelinated nerve fibres. The human longissimus capitis is the most densely spindled muscle and the density of muscle spindles is 25 times more in the lumbrical muscles than in the gastrocnemius [10].

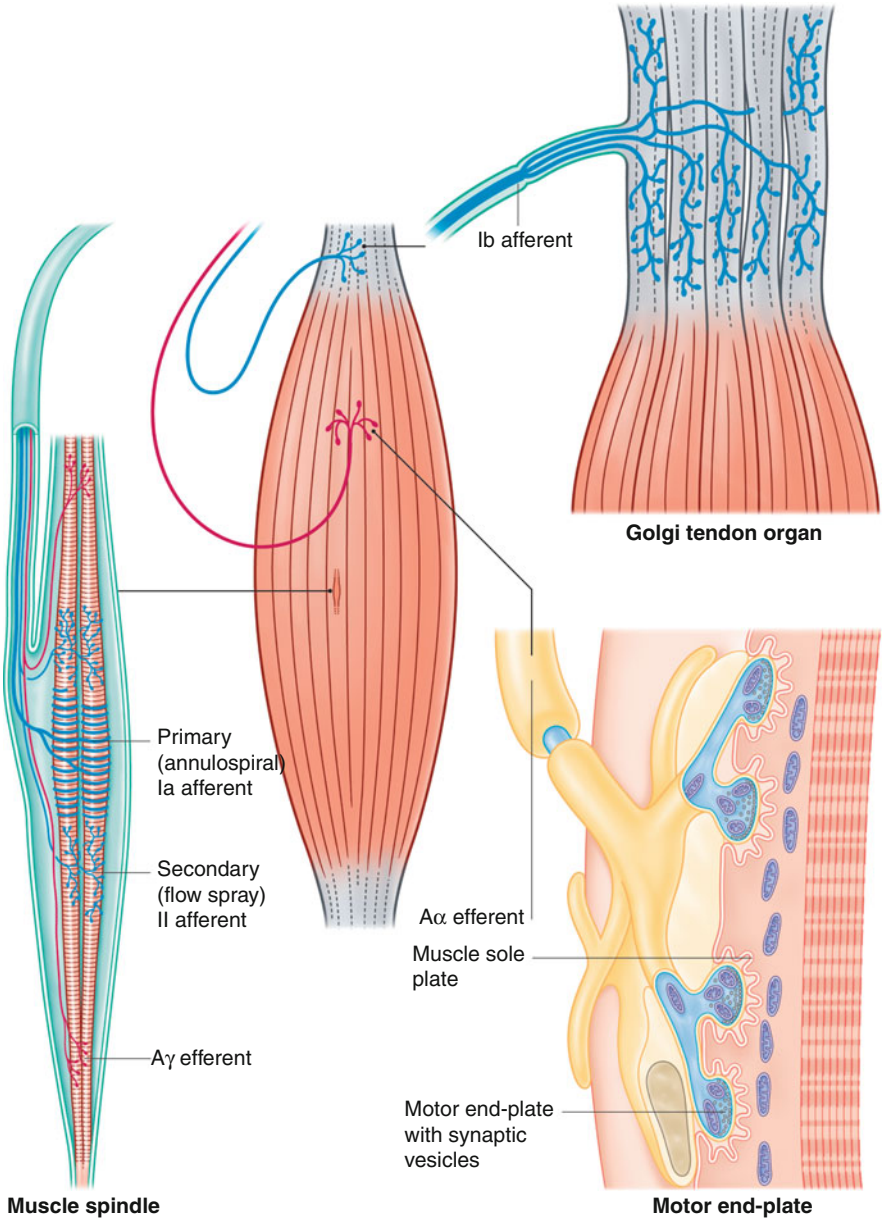

Fig. 1.60 The afferent and efferent innervation of skeletal muscle

The Golgi tendon organs: Scott [34] characterises the second of the two encapsulated mechano-receptors in muscle. The Golgi organ senses maintained tension in muscle and impulses project from it to the cerebellum and cortex. Stimulation of the fibres from the Golgi apparatus in hand muscles causes cortical potentials and "illusions of muscle stretch". The Golgi organ is about 0.1 mm in diameter and between

0.2 and 1.5 mm in length. It contains collagen strands which continue into muscle fibres at one end and into the tendon at the other. There are between 3 and 50 of these organelles in each muscle, and Scott says that the ratio of Golgi organs to spindles is less than 0.3. The myelinated afferent fibre is a little smaller than the largest afferent from the muscle spindle, and in the cat it conducts at the rate of 60–110 m/s. The terminals interweave amongst the collagen strands as sprays or spirals. The capsule contains capsular cells which are continuous with the Schwann cells. The receptor is slowly adapting; it responds to the whole range of muscle contraction and the firing rate is proportionate to active tension. In humans the fibres are silent at physiological rest and there are progressive steps in the firing rate with increasing steps in muscle contraction. Recovery is virtually complete after a crush lesion inducing axonotmesis, it is very much worse after repair of divided nerves. The poor recovery of the two main encapsulated mechanoreceptors in muscle after repair of divided nerves may account for the common complaints of weakness, lack of stamina, and poor coordination and also for the failure of musculotendinous transfer using reinnervated muscles.

Up to now, most clinical work on sensation and on recovery of sensibility after nerve injury and repair has been directed to cutaneous sensibility. Yet function such as stereognosis and proprioception must depend principally on signals from endings in muscle, tendons and ligaments. It is perhaps inadequately appreciated that there may be good recovery of sensory function of the hand with very imperfect cutaneous reinnervation, and that pain is just as likely to follow damage to a "purely motor" nerve as it is to follow damage to a "mixed" or "sensory" nerve. There is in fact no such entity as a "purely motor" nerve, except perhaps for the hypoglossal or facial. The signals from the muscles supplied by those nerves probably proceed by other cranial nerves: the lingual in the case of the hypoglossal nerve and the trigeminal and the auricular branch of the vagus in the case of the facial nerve. There are indeed a few peripheral nerves without a cutaneous sensory component: the spinal accessory; the phrenic; the anterior and posterior interosseous; the deep branch of the ulnar; the suprascapular. The content of afferent fibres in all such nerves is about 30 % [6] (Fig. 1.61). It is perhaps best to drop the terms "purely motor" and "purely sensory", and even drop the term "mixed" applied to nerves with both motor and cutaneous sensory components. The terms "nerves with motor and cutaneous sensory components" and "nerves without somatic motor components" are, unfortunately, cumbersome, but they do say what they mean.

1.10.5 Central Connections

The great array of sensory receptors in the skin and deep tissues sends back to the centre the signals of the stimuli received. Most afferent fibres, with their cells in the dorsal root ganglia, enter the cord by the dorsal roots. Others, with cells in the dorsal root ganglia or actually in the ventral roots, enter the cord by the latter (Fig. 1.62).

The first analysis of incoming signals takes place in the spinal cord and medulla where all fibres terminate. Most of the large myelinated fibres ascending in the

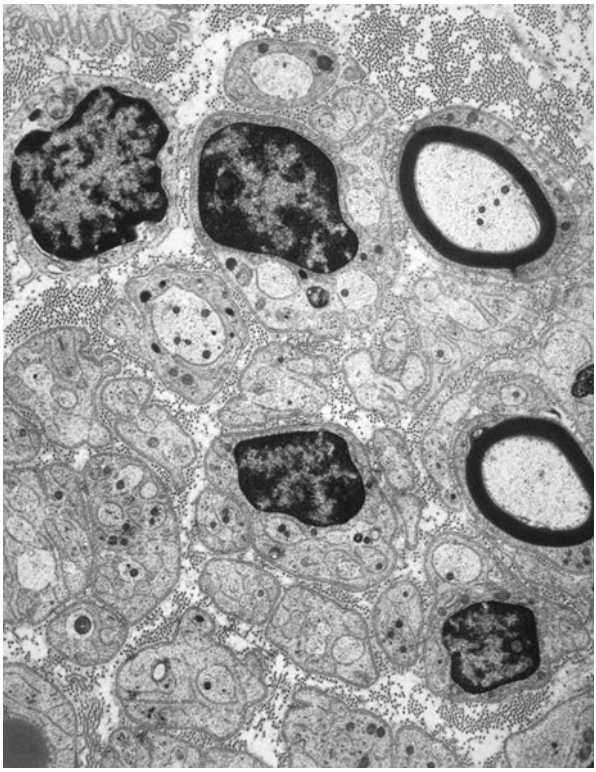

Fig. 1.61 Intact (afferent) myelinated and unmyelinated fibres in the suprascapular nerve after avulsion lesion of the brachial plexus. Specimen taken 6 weeks after injury, when efferent fibres had degenerated ×6,600 (EM)

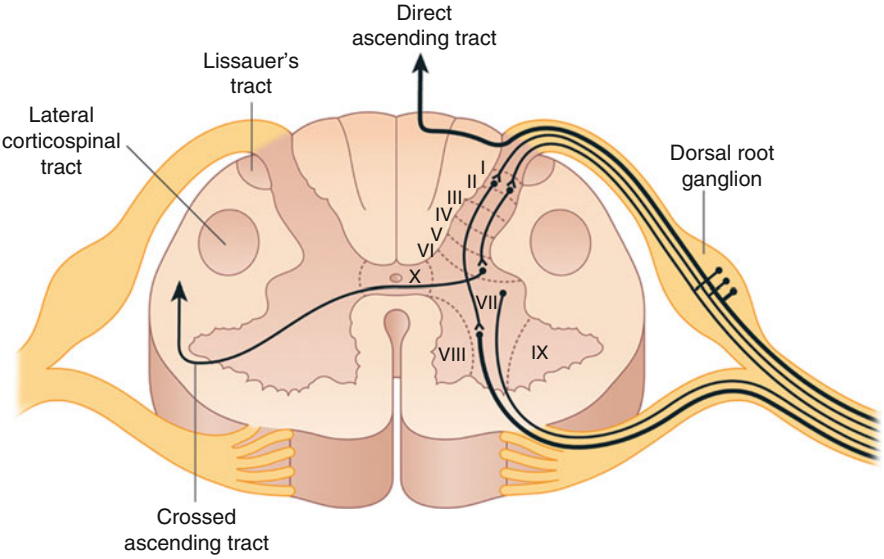

Fig. 1.62 The laminae of the grey matter with direct ascending, crossed ascending and internuncial tracts

dorsal columns terminate in the gracile and cuneate nuclei in the medulla. Some, smaller fibres of the dorsal columns terminate and relay in the cord: these are the propriospinal fibres. Although the classical view of the function of the dorsal column has been challenged [43], it is broadly true that, as Brodal [8] states, they "mediate sensory signals necessary for rather complex discrimination tasks" [8].

Other afferent fibres terminate and relay in the grey matter of the dorsal horn. Each lamina of the grey matter receives afferents of specific functional modalities; each has a particular neuronal structure [29, 30]. Small myelinated nociceptor and thermoreceptor fibres terminate in lamina I; C fibres, nocithermo- and mechanoreceptors, in lamina II (substantia gelatinosa). Larger mechanoreceptor fibres terminate in laminae III and IV. These relay to cells whose axons either ascend in the dorsal columns or reach the dorsal column nuclei by the dorsolateral fasciculus. Some fibres pass through the dorsal horn to relay with the large cells in the motor apparatus in lamina IX. Some unmyelinated and small myelinated fibres enter the dorsolateral fasciculus (Lissauer's tract) just lateral to the tip of the dorsal horn to join fibres from cells in the substantia gelatinosa. Some fibres cross the midline to terminate in laminae I and V of the contralateral dorsal horn. There is a complex network of interconnecting fibres in the dorsal horn and in the substantia gelatinosa in particular. Sensory input is first analysed and modified here. Secondary neurons in the dorsal horn give rise to fibres which ascend or descend for a few segments in the cord. They give rise principally to the fibres that, crossing the midline, ascend in the long tracts in the anterolateral segment of the spinal cord (Fig. 1.63).

The transmission of impulses from the nuclei of the dorsal column is influenced by fibres descending from the sensory motor cortex [17]. This influence is predominantly inhibitory. The ascending fibres of those nuclei form the medial lemniscus of the brainstem, crossing the midline in the medulla to end in the thalamus. The final resolution of sensory impulses takes place in the somatosensory areas of the cerebral cortex [26]. Not even in this last analysis are afferent functions separated completely from motor function: stimulation of any of these areas produces motor effect.

1.10.6 Afferent Autonomic Pathways

The first evidence for the presence of a system for conveying from all viscera sensations both perceived and unperceived rested largely on indirect observations: the truly autonomic functioning of viscera; the production of pain by mechanical stimulation of peripheral arteries and veins; the operation of "referred pain" mechanisms; pain after operations on the sympathetic chain; the lack of "visceral sensibility" when the function of visceral afferents is impaired by age [33]. Autonomic afferent fibres have their cells of origin in the dorsal root ganglia and in some cranial ganglia. Their peripheral processes run with efferent fibres, terminating in receptor endings in the walls of viscera and vessels. The cell bodies of afferent fibres in the enteric division lie in the wall of the gut (Fig. 1.64). Some

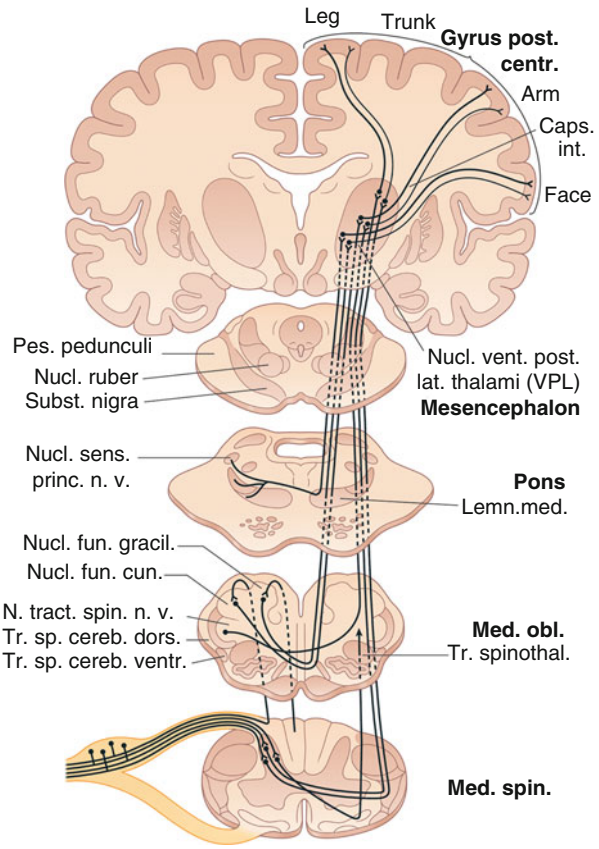

Fig. 1.63 Ascending tracts in the cord, brain stem and cortex. Note the relay of the directly ascending tracts in the gracile and cuneate nuclei in the medulla

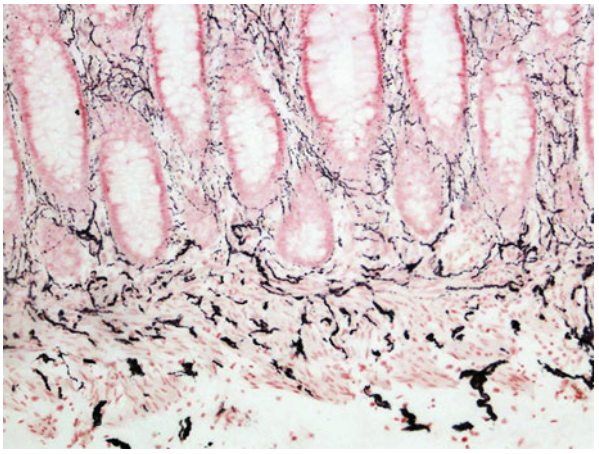

Fig. 1.64 Afferent fibres in the enteric division of the autonomic system. Nerve fibres within the mucosa and submucous plexus of human rectum stained with antibodies to sensory sodium channel (Nav1.7) ×10

impulses from these endings mediate sensations such as hunger, distension of the bladder and perhaps pain. Most, presumably, are concerned with the initiation of visceral reflexes. The afferent component of the enteric division is described by Furness [15]. Sugiura et al. [38] found that visceral afferents terminate in laminae IV, V and X of the dorsal horn as well as in laminae I and II, and suggested that "the somato-visceral convergence could occur in the superficial dorsal horn of the spinal cord", and that "the scattered and extensive distribution of the terminal fields of single visceral C-afferent fibres may be one basis for the poor localisation of visceral sensation".

1.11 Synaptic Activity

The transmission of impulses at synapses is chemical, by the release of neurotransmitters causing a change in the permeability and hence the electrical polarisation of the post-synaptic membrane. Such changes may be excitatory or inhibitory; they are usually short-lived, because of early inactivation of the neurotransmitter. This is not of course the whole process: the effect of some neurotransmitters may be more prolonged or even permanent. In addition, some substances released at synapses may simply modify the response of the post-synaptic membrane to neurotransmitters. The general term "neuromediators" has been applied to substances released at synaptic endings; "neurotransmission" implies a direct effect on post-synaptic membrane; "neuromodulation" implies alteration of its response to a neuromediator [44].

The best-known mediator and the one that has longest been known, is of course acetylcholine, synthesised by motor neurons and released at the motor terminals in skeletal muscle and at the synapses in sympathetic and parasympathetic ganglia. The other well-known mediators belong to the monoamine group: they are noradrenaline, adrenaline, dopamine, serotonin and histamine. Noradrenaline is the chief transmitter at the endings of sympathetic ganglionic neurons. Adrenaline too is present in peripheral neural pathways. Nitric oxide (NO) mediates smooth muscle relaxation at autonomic synapses. The other monoamines are chiefly present in the central nervous system. Gamma amino butyric acid (GABA) is a major inhibitory transmitter which is released at the terminal of such local circuit neurone systems as the inhibitory Renshaw loop. Glycine is another example of an inhibitory transmitter which is particularly prominent in the lower brain stem and spinal cord. Glutamate and aspartate are widely distributed excitatory transmitters. The range of neuropeptide modulators is very wide, including those associated with the function of the hypothalamus and hypophysis, corticotrophin, beta-endorphin, the enkephalins, calcitonin-related gene peptide and nerve growth factors. In the field of peripheral nerves, the last is of great and growing importance; the beta-endorphins and enkephalins are important in the consideration of the mechanism and treatment of pain.

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Chapter 2
Reactions to Injury

The conducting element of the nerve fibre, the axon, responds to focal injury in a number of ways. There are two essential lesions. In the first, the axon is intact but conduction at the level of lesion is blocked. This is *conduction block* (*CB*). There is no Wallerian degeneration; this is the *non degenerative lesion*. Recovery will be complete if the cause is removed. In the second, the axon is transected. Wallerian degeneration ensues; this is the *degenerative lesion.* There are two types of degenerative lesion. In the first, the basal lamina of the Schwann cell envelope is intact and the axon can regenerate in an orderly fashion into the distal Schwann cell tube. In the second type the basal lamina has been interrupted and spontaneous regeneration will be imperfect, disorderly and may not occur at all. Seddon [32] introduced the terms *neurapraxia* for CB, *axonotmesis* for the degenerative lesion of favourable prognosis because of the intact basal lamina, and *neurotmesis* for the degenerative lesion of unfavourable prognosis because the basal lamina has been interrupted. In clinical practice neurotmesis usually represents severance not only of the axon and its Schwann cell envelope but also of the perineurium and the epineurium.

The difference between CB and the unfavourable degenerative lesion, neurotmesis, is exemplified by two clinical situations which are common enough.

Case report: Pure CB: A fit 23 year old woman fell deeply asleep lying on her left side for about 2 h and awoke with a complete left sided radial palsy. She had no pain, there was no Tinel sign and there was some preservation of cutaneous sensibility within the distribution of the nerve. She was fitted with a dynamic extension splint and at 6 weeks the first evidence of recovery into the extensor muscles of the wrist was apparent. Neurophysiological investigations were performed 9 weeks later which revealed normal conduction and a normal recruitment to a full pattern of motor units of normal appearance. Her recovery was complete by 12 weeks from the incident.

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Case report: unfavourable degenerative lesion, neurotmesis: A 32 year old tiler wounded his left wrist with a tile knife. He experienced shooting pain in the hand and noticed a sudden rush of warmth especially into the thumb and index finger. On examination the skin of the thumb, index, middle and radial side of the ring finger was red, warm and dry. He was able to recognise a sort of light touch in the median territory but it was abnormal. The wound was less than 1 cm in length. Gentle percussion over the skin just proximal to it elicited painful pins and needles in the thumb. A diagnosis of partial neurotmesis was made but at operation later that day the nerve was found wholly transected. There was little retraction of the severed bundles. The patient himself experienced the sudden vasomotor paralysis and this, with the sudomotor paralysis demonstrated that the axons, at least, had been severed.

Most injuries of nerves short of transection inflict damage of all three grades of severity. This is the usual pattern for the sciatic nerve stretched but not ruptured by dislocation of the femoral head and for the cords of the brachial plexus stretched but not ruptured by anterior dislocation of the head of the humerus. The decision about exploration in cases such as these is never easy but the difficulties may be eased if the clinician bears in mind these facts.

- removal of the cause of the lesion may prevent deepening or deterioration
- removal of the cause may convert a situation inimical to spontaneous recovery to one that is more favourable.
- the persistence of pain is an important indication for exploration of the nerve [6, 24].
- a nerve which has been transected or ruptured cannot recover until it is repaired;
- a lesion of a peripheral nerve which remains in continuity but which continues to be subjected to the cause of that lesion will deepen until the cause is removed (Figs. 2.1 and 2.2).

The speed of deepening of lesion is related to the cause.

- a nerve crushed by a plate or by an encircling suture may recover if that cause is removed within a minute or two; it is unlikely to recover if it is not relieved for more than 2 or 3 h.
- the nerve subjected to compression and ischaemia within a swollen ischaemic limb will almost certainly recover if the cause is corrected within 3 h. The chances of full spontaneous recovery diminish with the passage of every hour after that time.
- A nerve entrapped within a fracture or dislocation will usually recover if it is extricated within a few days but it will be destroyed if a compression device has been used.
- It may be years before the situation becomes irretrievable for a nerve subjected to radiotherapy or exposed to continuing traction from a malunited fracture.
- *The cardinal symptom of the persistence of a noxious agent is pain*

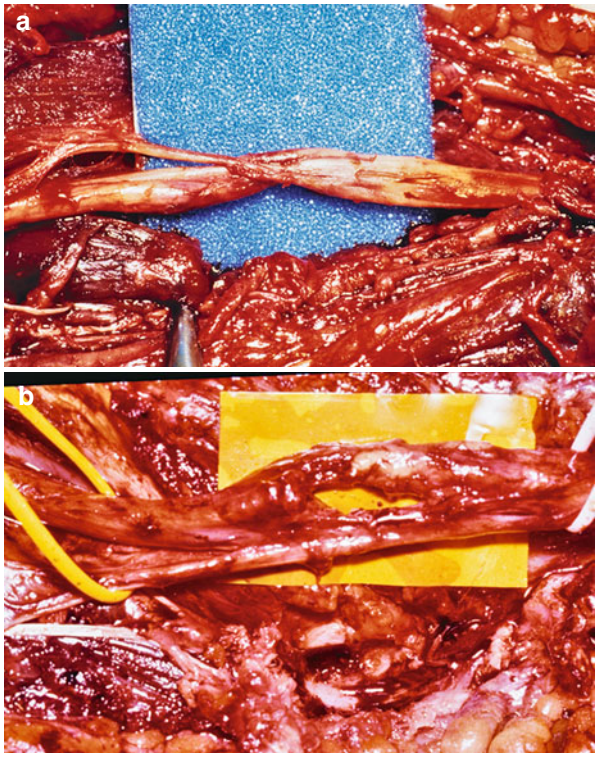


Fig. 2.1 Deepening of lesion. (**a**) Median nerve extricated from supracondylar fracture in a 9 year old girl at 3 days from injury. There was complete recovery. (**b**) Median nerve extricated from supracondylar fracture in a 13 year old girl 8 weeks after injury. There was no recovery

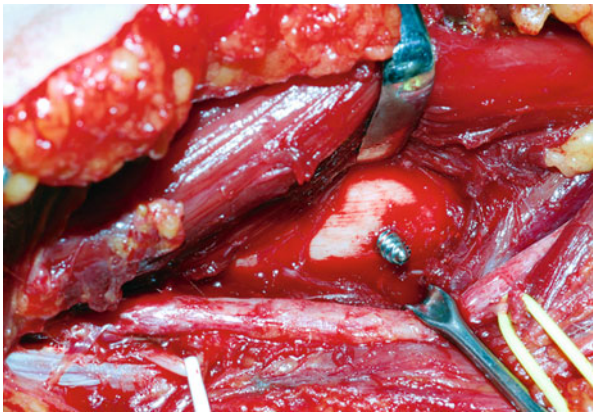


Fig. 2.2 Conduction block. The radial nerve shown tented over the tip of the screw 10 days after operation for fracture. There was a painful deep radial palsy. Conduction in the distal segment was preserved but, there was no conduction across the lesion. Conduction in the nerve to brachioradialis (*left hand sling*) was preserved. Stimulation of the radial nerve above the level of lesion did not evoke response through the nerve to extensor carpi radialis longus (*right hand sling*). The screw was shortened. Recovery was complete by 24 h

2.1 Conduction Block: Neurapraxia

There are a number of distinct patterns of CB. Anoxia is a dominant factor in several of these and it is important in all. There may be an elements of mechanical deformation from forces acting upon the nerve from outside or from within it as in tumour.

2.1.1 Transient Ischaemia

The first effect of ischaemia upon peripheral nerves is the loss of conduction caused by anoxic block of fast axoplasmic transport systems and the paralysis of ion channel function. This is seen during exposure of limb nerves with an inflated cuff in position. For about the first 20 min stimulation of the nerve evokes a brisk muscular response by transmission through the neuromuscular junction, which diminishes and disappears after about 30 min. Conduction within the nerve itself can still be detected for about another 30 min. On the other hand direct stimulation of the muscle provokes a twitch which can be elicited for up to several hours. Indeed, it is the loss of that direct response which signifies impending death of the muscle, and with it, the death of the limb.

This form of centripetal paralysis is experienced by an observer following the classical experiment of application round the arm of a cuff inflated to suprasystolic pressure. First, there is loss of superficial sensibility. This is succeeded by a gradual loss of motor power. The first pain response is lost soon after superficial sensibility fails, but the delayed pain response can still be elicited after 40 min of ischaemia. Pilomotor and vasomotor functions are scarcely affected. Large myelinated fibres (Mnf) are first affected; non myelinated C fibres (nMnf) and autonomic fibres escape. Recovery of all modalities occurs within a few minutes of release of the cuff. The experience of the unpleasant quality of the residual delayed pain sensation gives a good insight into the feelings of patients affected by dysaesthesia.

This pattern of CB caused by transient ischaemia offers a valuable insight into the effect of anoxia on nerve function but it is uncommon in clinical practice. It is especially significant as a prelude to something much worse if perfusion is not restored.

The slowly evolving anoxic CB of progressing ischaemia is more common. Compression of nerves by haematoma or aneurysm produces a characteristic pattern: autonomic paralysis is early and deep; loss of power extends over hours or days; deep position sense and limited joint position sense persist. Slower still is the CB brought about by compression or strangulation from scar tissue. The patient usually experiences pain, neurostenalgia, an important indication for operation [6, 7].

Case report: Phang et al. [30] described the case of a 26 year old woman who developed pain in her left hip. This was attributed to previously undiagnosed bilateral hip dysplasia. Pelvic osteotomy of both hips was done. The pain in the left hip became much worse and both hips were resurfaced 6 years later. The pain in her left

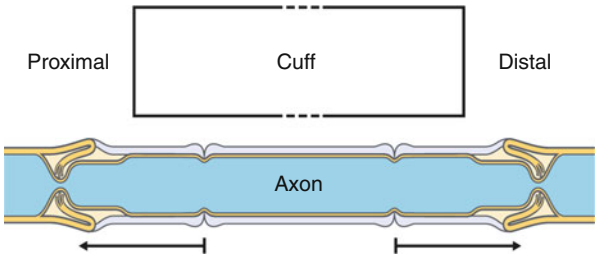


Fig. 2.3 Effect of pressure on nerve: squeezing of myelin with invagination at the node of Ranvier. Narrowing of axon with extrusion of its contents

hip deepened so that she could walk only with crutches. When she was reviewed 10 years after the onset of her symptoms it was clear that there was a focal lesion of the left femoral nerve. There was a strong and painful Tinel sign over the nerve at the groin crease, motor and sensory conduction was impaired and electromyography confirmed a mild degree of degenerative lesion of the fibres to the quadriceps muscle. The femoral nerve was exposed. It was tethered and sharply compressed by scar tissue over a 4 cm segment. The nerve was liberated. Her pain was improved, improvement in the range of movement at the hip and power of extension of the knee. By 1 year she was able to walk freely without any aid. Hindsight is always easy but it does seem that the lesion of the nerve occurred during the operation of pelvic osteotomy and her description of the new symptoms which occurred after that operation indicated neuropathic pain.

Important examples of prolonged and very painful CB caused by the scarring associated with split skin grafts are seen in war wounds [7] (see Sect. 5.1.1).

2.1.2 Conduction Block in Myelin Deformation and Focal Demyelination

Severe prolonged pressure causes local demyelination and more prolonged CB [15]; (Fig. 2.3). The myelin is squeezed proximally and distally from underneath the tourniquet so as to invaginate into the proximal and distal sheaths of the nodes of Ranvier. The structural effects of focal compression include [14]:

- The endoneurial fluid is squeezed out, nerve fibres and cells became more closely apposed;
- Fluid is squeezed from out of the axon leading initially to compaction of the formed elements and later to their being expressed from out of the axon at the margins of the zone of compression.
- The internodes become lengthened with shearing between the lamellae of the myelin sheath and there is swelling of the nodes of Ranvier adjacent to the zone of compression.

A conduction block which may last for weeks or months results. If, in the clinical situation, the cause of the local demyelination persists in the form of (say) a bony

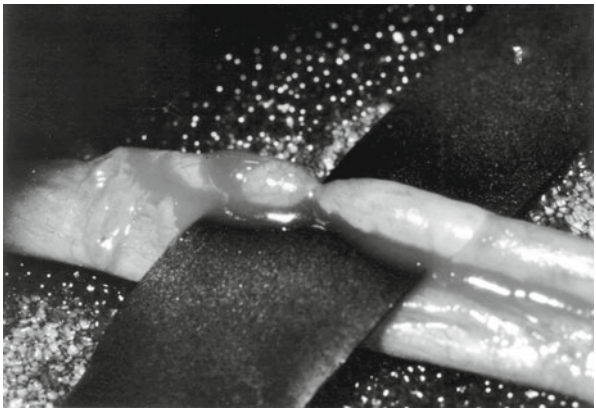


Fig. 2.4 Hour glass "constriction" of the lateral root of the median nerve, the result of traction injury. There was full spontaneous recovery

projection causing pressure and distortion the block persists. This was so in the three cases reported by Birch and St Clair Strange [1]. In these removal of the external pressure was rapidly followed by relief of pain and recovery in lesions which had persisted for up to 3 years.

Mechanical deformation is the likely explanation for the CB in cases of "hour glass" constriction of the fascicles within a main nerve trunk. It is possible that there is an element of constriction of the axon itself. Recovery after internal (interfascicular) neurolysis is generally good (Fig. 2.4).

2.1.3 The Conduction Block of War Wounds

Seddon [32] observed the characteristic features: paralysis exceeds loss of sensation; the nerves responsible for proprioception are more deeply affected than those conveying light touch sensation; vasomotor and sudomotor function is least affected. It is likely that this lesion is provoked by a momentary displacement, or stretching, of the nerve trunks. This explanation cannot account for more frequent CB of blast injuries in which the patient is exposed, at close range, to the shock wave of an explosion without any wound, or fracture and with no signs of significant injury to the soft tissues. Often the smallest fibres are most deeply affected and they may not recover. The mean time to recovery was 3.8 months (0.6–6) in 45 cases of CB caused by penetrating missile wounds; it was 47 months (2.5–10.2) in the 71 cases caused by explosion [7].

A diagnosis of conduction block (neurapraxia) is established by the demonstration of persisting conduction in the nerve distal to the level of lesion after an interval of about 5 days. It is unwise to make this diagnosis in the following circumstances.

- Where there is a wound over the course of a nerve. This error is all too common where nerves have been injured during operation.
- With persisting pain for this signifies that the noxious agent is continuing to act.

- In the presence of a strong Tinel sign for this indicates that axons have been ruptured.
- In limbs rendered pulseless by injury: conduction block may prove to be but the first step towards something much worse.

The early disappearance of conduction is, of course, the hall mark of impending or actual "critical" ischaemia.

2.2 The Degenerative Lesion

Wallerian degeneration affects not only the axon but also the cell body; not only the neurone but also its Schwann cell ensheathment and its myelin sheath. There are changes too in the endoneurial cells and, over longer periods of time, in the motor and sensory end-organs. Distal to the site of injury the axon degenerates; there is a granular disintegration of the cytoskeleton and axoplasm, which are converted over succeeding days into amorphous debris (Figs. 2.5 and 2.6). Observations on motor conductivity after pre-ganglionic injury to the brachial plexus suggest that the motor response ceases between 3 and 5 days after injury [5].

So long as the lesion is not severe enough to interrupt the continuity of the Schwann cell basal laminae from proximal to distal segment the original pathways for re-growth of axons remain. It is the difference between preservation and destruction of continuity *of the basal lamina* that underlies the division of degenerative lesions between those with the potential for spontaneous recovery (*axonotmesis*) and those that will not recover unless action is taken (*neurotmesis*) (Fig. 2.7). The processes of Wallerian degeneration are the same in both types of lesion but they are much less severe in those where the basal lamina is intact because the regenerating axon is able rapidly to re-establish contact with the distal Schwann cells, so restoring the flow of neurotrophins from these and from the target tissues. Above all the incidence of central cell death is far lower after axonotmesis than it is after neurotmesis.

2.2.1 The Cell Body and Proximal Stump

The central and the peripheral effects of Wallerian degeneration are profound and, in neurotmesis, ultimately irreversible. The cell body is separated from the supply of neurotrophins and it may be drained or exhausted by the process of regeneration. Proximal to the lesion changes occur in the axon, the myelin sheath, and in the nerve cells.

- Within a few days there is a reduction in the calibre of the proximal axon; nerve conduction velocity in the proximal segment falls.
- In the cell body itself there may be chromatolysis, a process characterised by Groves and Scaravilli [16] as one associated with a regenerative and not a

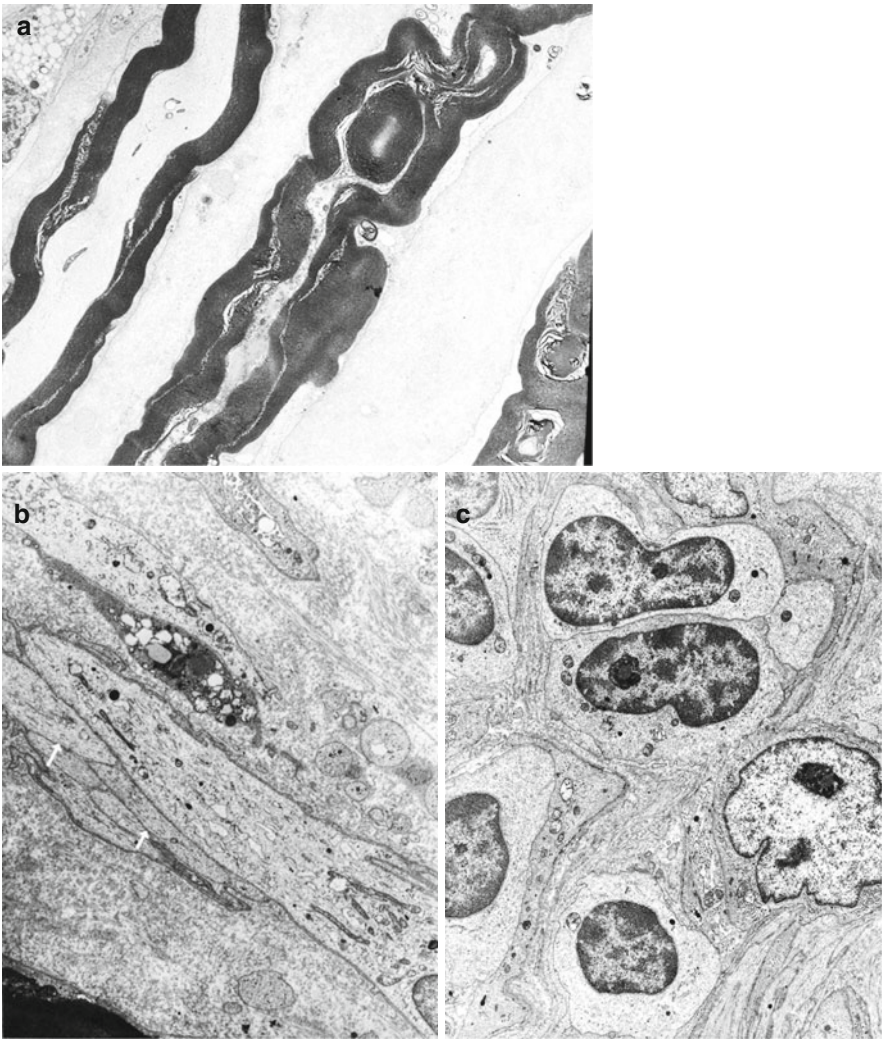


Fig. 2.5 Changes in the distal stump of ulnar nerve transected 3 weeks previously. (**a**) Disintegration of the axon and the myelin sheath. Axoplasm and neurofilaments are seen in the lower fibre ×2,210. (**b**) Another part of the same specimen. Myelin debris within a macrophage (*asterisk*), probable Schwann cell processes (*arrows*) ×5,525. (**c**) Another part of the same specimen. Many Schwann cells, some with active nuclei ×5,525 electronmicroscopic studies (EM)

degenerative response to an insult. Chromatolysis may continue to actual dissolution of the cell body. The nucleus becomes unidentifiable, all basophilia has disappeared and what remains is a seemingly empty sac containing the condensed remnants of neuronal DNA, a so-called ghost cell. Curiously enough, transection of the central branches going to the central nervous system (*rhizotomy*) does not produce such clear cut changes in the cell bodies in the dorsal root ganglia.

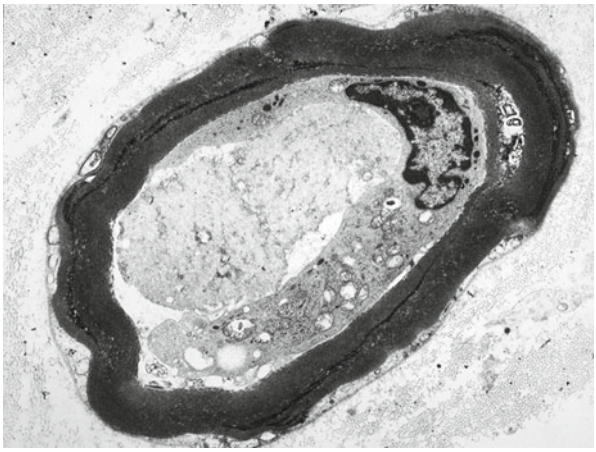


Fig. 2.6 Wallerian degeneration in the distal stump of fifth cervical nerve ruptured 3 weeks previously. The axon is collapsing and it is surrounded by a macrophage ×11,000 (EM)

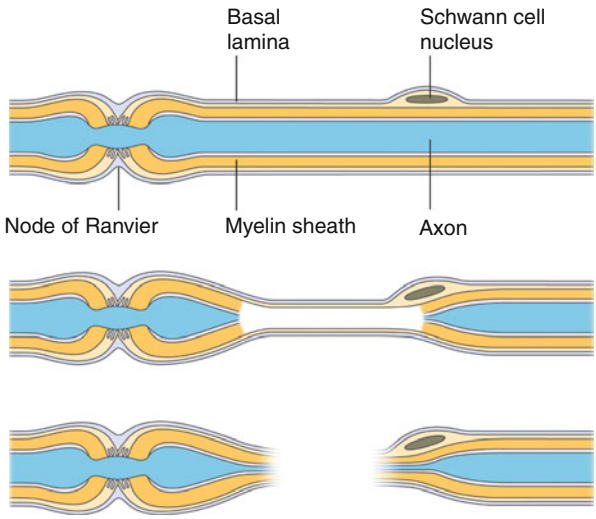


Fig. 2.7 Axonotmesis (*centre*) and neurotmesis (*bottom*) at the moment of injury. Note the preservation of the Schwann cell basement membrane in axonotmesis

- Changes in the expression of ion channels and receptors in the dorsal root ganglion (DRG) neurone can be detected within a few minutes of an injury to the nerve [21].
- Studies of neurones within adult human dorsal root ganglia in cases of avulsion lesions of the brachial plexus have revealed dramatic changes in the expression of genes involved in neurotransmission, trophism, cytokine function, signal transduction, myelination, transcription regulation and apoptosis [31] (Fig. 2.8).
- The loss of cells is more severe in more proximal neurotmesis. Neurotmesis in the neonate produces a more rapid and much greater incidence of sensory and motor neurone death than in the adult. Motor neurone cell death is particularly severe after avulsion of the ventral root.

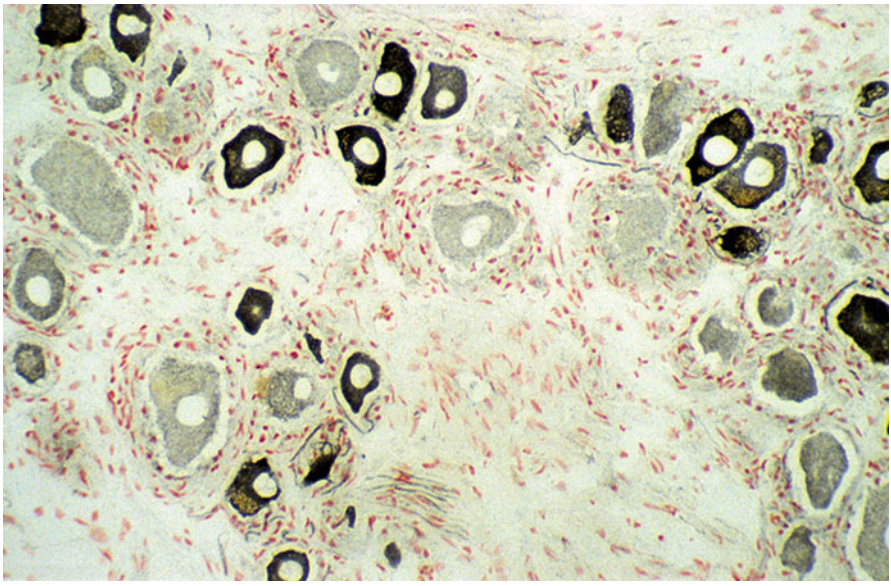


Fig. 2.8 TRPV1 (the heat and capsaicin receptor) in a human dorsal root ganglion 6 weeks after avulsion showing immunostaining in small diameter neuronal cell bodies and axons ×40

Amputation provides a model of the effect of permanent axonotomy on the spinal cord. There is a loss of neurones in the DRG, in the anterior horn, and a diminution of the large myelinated nerve fibres in the ventral and dorsal roots [35].

2.2.2 The Distal Stump

Hall [18] conceives Wallerian degeneration as an active process in which the environment of a normal nerve so inimical to regeneration of axons is transformed into one which is actively receptive to that regeneration, at least for a limited period. The earliest changes affect the cytoskeleton. There is dissolution and clumping of the neurofilaments and microtubules [14]. Schwann cells in the distal nerves, both in myelinated and unmyelinated fibres, begin a process of proliferation. Within 48 h of injury, denervated myelinating Schwann cells down regulate expression of those genes encoding myelin associated proteins and other proteins which are important for maintaining the organisation of the nodes and paranodes. The denervated Schwann cell columns lie within the original basal lamina forming Schwann tubes, formerly known as bands of Büngner. There is proliferation of endoneurial fibroblasts in the distal nerve. The last important feature is the increase of macrophages; some derived from resident cells, others recruited from the circulation. Macrophages clear the debris of myelin and axoplasm during which process a Schwann cell mitogen is liberated, they remove proteins such as the myelin associated glycoprotein (MAG) which normally inhibit axonal growth [18]. As time passes the endoneurial tubes shrink, more collagen is deposited within the endoneurium and there is progressive

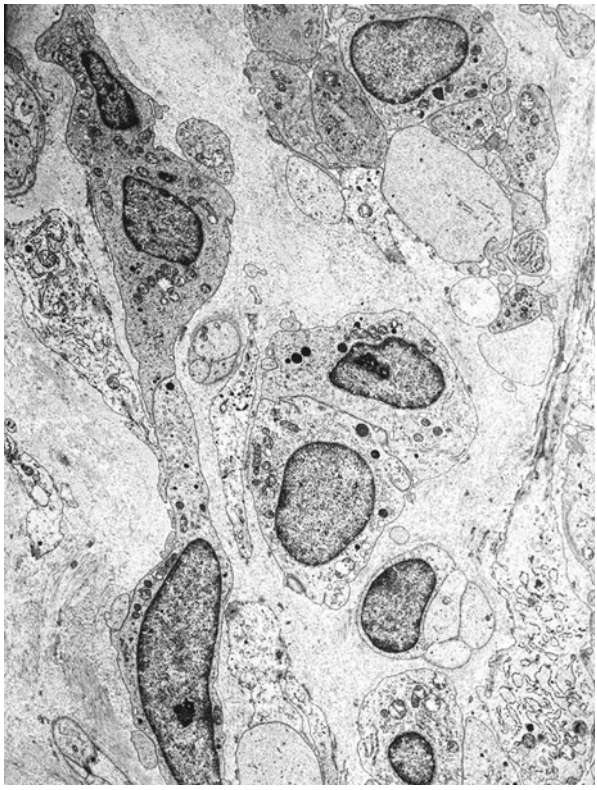


Fig. 2.9 Distal stump of median nerve in a 25 year old man transected 6 months previously by bullet from military rifle. Numerous pale processes of Schwann cell cytoplasm with occasional axonal sprouts. Extensive endoneurial collagenisation ×3,245 (EM)

fibrosis within the distal stump. With delay, the number of Schwann cells in the distal stump diminishes and they become less receptive to regenerating axons because of the decrease in the expression of receptors which are normally important in Schwann cell-axon signalling, effects which are worsened by ischaemia and by sepsis [3] (Figs. 2.9 and 2.10).

Wallerian degeneration is inevitable after opening of the perineurium as in "end to side" repair. Perineurial cells separate from one another and also from their basal lamina and come to resemble fibroblasts. Regrowth occurs by bundles of axons and Schwann cells surrounded by fibroblasts which later develop into perineurial cells leading to the formation of many small fascicles, (mini fascicles). This process occurs when the perineurium is lacerated by the tip of a needle and it leads to the formation of a small neuroma within the nerve trunk (Fig. 2.11).

2.2.3 Contralateral Effects

A dramatic example of the involvement of regions beyond the area of injury is provided by Suzuki et al. [35] who, in addition to the ipsilateral effects already described, demonstrated atrophy of the contralateral anterior horn, where there was a loss of the medium size cells and a reduction in the numbers of medium and small

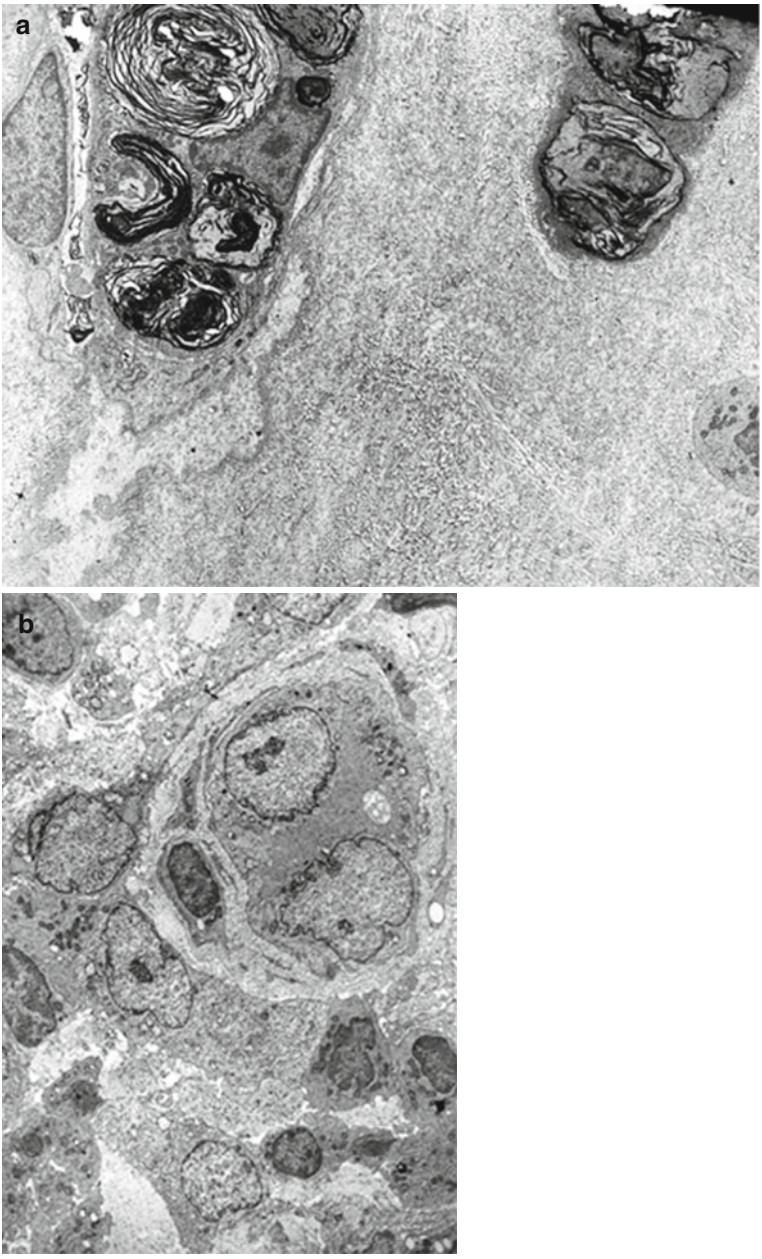
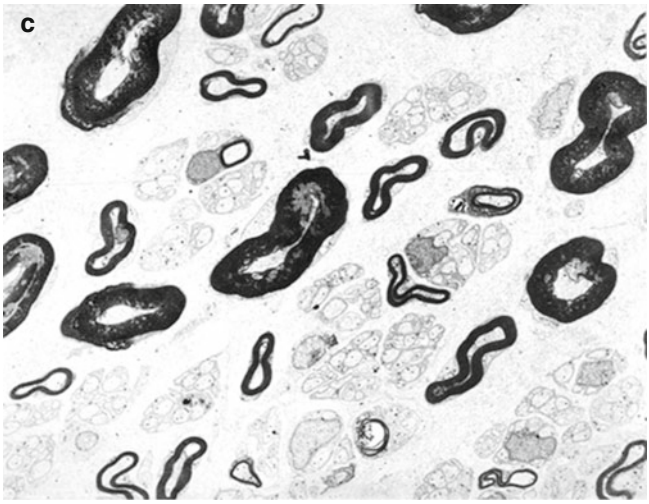


Fig. 2.10 The results of rupture complicated by sepsis. Electronmicroscopic study of serial (2 cm interval) biopsies of proximal stump of sciatic nerve of 34 year old man 8 months after rupture by fracture and subsequent sepsis. (**a**) 2 cm from the tip of the proximal stump ×2,340. Relics of myelin persist amidst massive collagenisation. (**b**) 4 cm from the tip of the proximal stump ×3,000. Many fibroblasts with Schwann cells. (**c**) 6 cm from the tip of the proximal stump ×1,500. Myelinated and non myelinated fibres (EM)

Fig. 2.10 (continued)



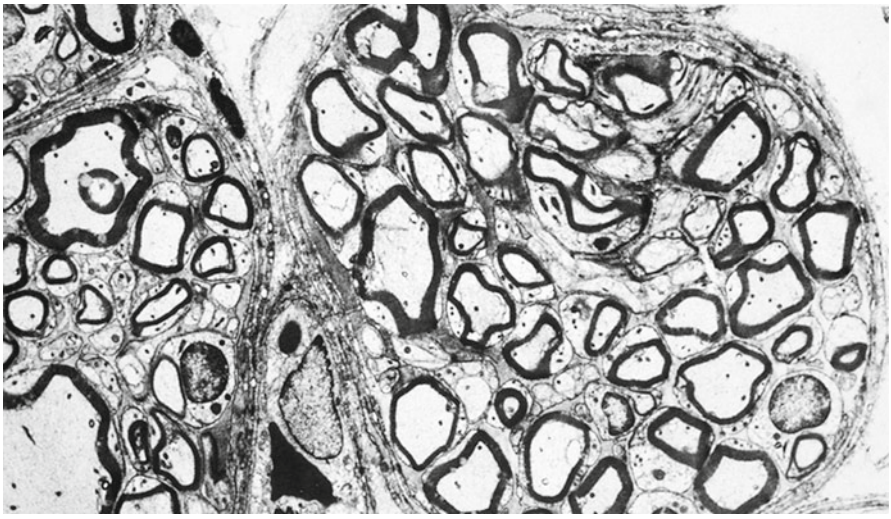


Fig. 2.11 Injection injury. The tip of the needle lacerated the lateral part of median nerve at the elbow and a neuroma occupied about four of the bundles. Mini fascicles in the proximal stump of one bundle, with myelinated fibres and Schwann cells ×2,000 (EM)

myelinated nerve fibres in the contralateral ventral roots. Oaklander and Brown [27] used the pan neuronal marker protein (PGP9.5) to measure the density of innervation in the skin of the paws of the rat after transecting one tibial nerve. There was almost complete loss of innervation within the plantar skin in the injured limb. However, a persisting loss of innervation, in excess of 50 %, was noted in the skin of the contralateral hind paw.

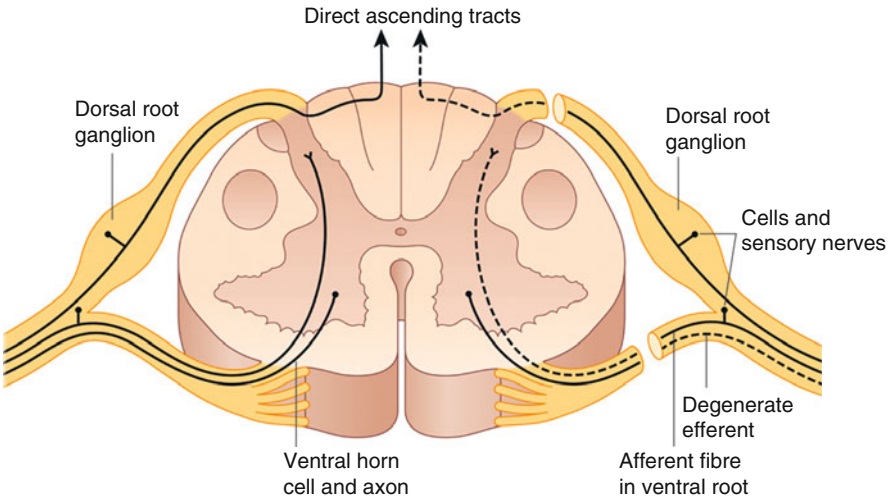


Fig. 2.12 The nerve and roots detached from the spinal cord. Note the intact dorsal root ganglion cell, with healthy axons in the detached parts of the roots, and the degeneration of the efferent fibre in the ventral root and of the central projections of the afferent fibres

2.3 The Special Case of the Brachial Plexus

A wholly different system of thought has to be employed in lesions of the brachial plexus, or for that matter of the lumbo-sacral plexus, in which there is intradural damage to the roots (Figs. 2.12 and 2.13). In spite of the damage to the proximal branches, the axons, whose cells of origin are outside the cord in the posterior root ganglion, remain healthy for a long time when they are avulsed from the cord or ruptured intradurally [3, 9] (Fig. 2.14). Such axons include all those in the dorsal root; also, of course, many "recurrent" fibres in the anterior root whose cells of origin are in the dorsal root ganglion (Fig. 2.15). These axons, their Schwann cells and myelin sheaths remain intact and functional, detached not only from central connection but also from the various systems of classification. This is in fact, so far as afferent neurones are concerned, a lesion of the central nervous system. Somatic efferent fibres undergo degeneration, being separated from their cells; post ganglionic autonomic efferent fibres also degenerate, because of damage to their grey rami communicantes. The effect of severe intradural (pre ganglionic) injury to the brachial plexus is severe. At delayed operation (hemi laminectomy) the ipsilateral cord is seen to be atrophic. Magnetic resonance imaging plainly shows atrophy of the cord after birth injury of the brachial plexus. Carlstedt [11] reckons that about one half of all motor neurones in the affected spinal cord segment have disappeared by 2 weeks after avulsion of the ventral root and he urges "a swift intervention to re establish contact between the injured nerve cells and the periphery with its supply of neurotrophic substances to counteract nerve cell loss".

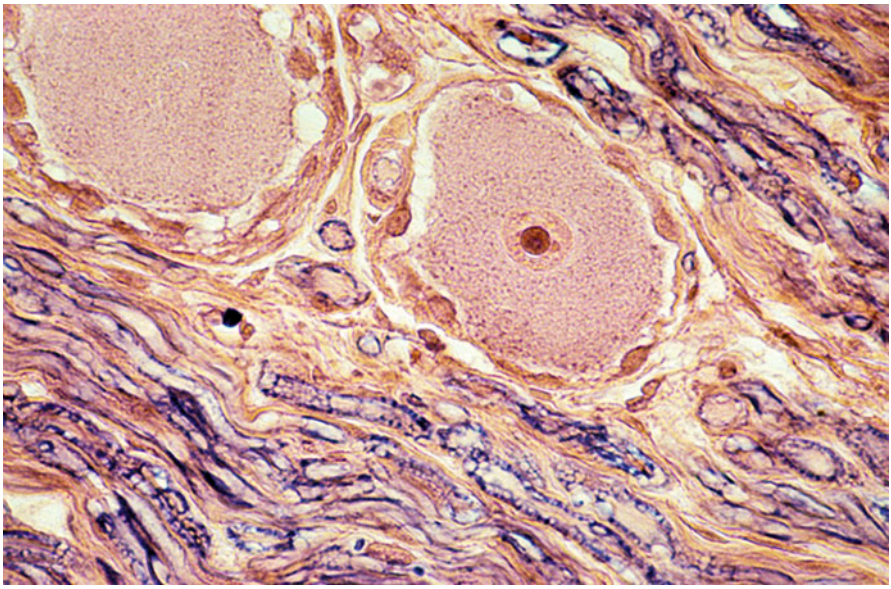


Fig. 2.13 Dorsal root ganglion 6 months after avulsion from the spinal cord. The two cell bodies appear healthy and there are numerous myelinated fibres. Solochrome cyanin ×960

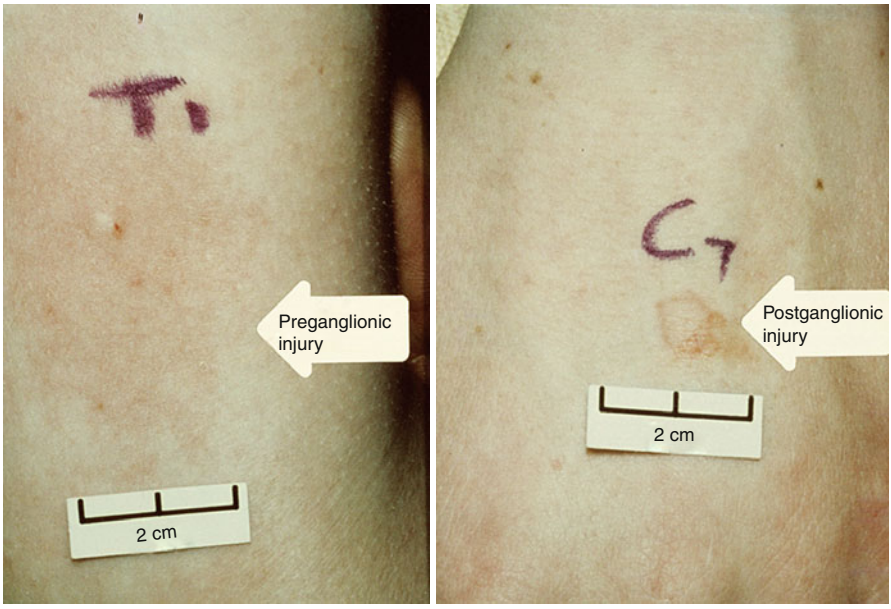


Fig. 2.14 Rupture and avulsion of the spinal nerves forming the brachial plexus: response to intradermal injection of histamine. *Left*. There is a flare, mediated by the axon reflex in the territory of the avulsed first thoracic nerve. *Right*. There is a wheal but no flare in the dermatome of C7 which was ruptured

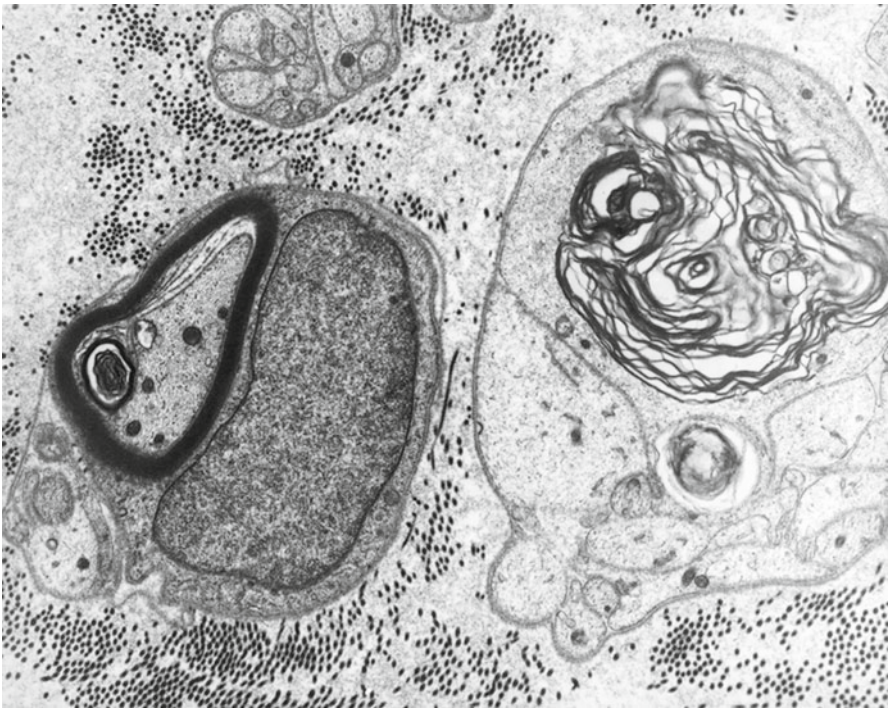


Fig. 2.15 Wallerian degeneration in the ventral root of the eighth cervical nerve 6 weeks after avulsion from the spinal cord. A degenerate efferent myelinated fibre (*right*) compared with a healthy myelinated afferent fibre (*left*) ×11,115 (EM)

2.4 Types of Lesion Produced by Different Physical Agents

Physical agents rarely act in isolation. Radiation induces changes not only in the neurone but also in the extrinsic and intrinsic circulation to the nerve and also to the surrounding tissues. "Entrapment" neuropathy is much more than simple focal compression. Tethering of, for example, the ulnar nerve at the elbow or the lower trunk of the brachial plexus at the first rib indices traction upon the nerve. There is compression and traction, both leading to anoxia. There is, too, mechanical deformation. These factors combine to produce ischaemic and focal demyelinating CB and then to degeneration with disorderly regeneration in the untreated case.

The difficulties of disentangling the various agents at work are exemplified by the common and serious problem of nerve injury incurred during arthroplasty of the major joints.

Total arthroplasty of the hip is a difficult operation, one in which there is very little room for error and the difficulties are more severe in cases where the joint is greatly distorted or when revision proves necessary. The complication of a painful

Table 2.1
The predominant lesion revealed at operation in 147 nerves (116 patients) operated between 1979 and 2007

Cement burn	3
Laceration by knife or scissors	11
Crush by suture or wire	10
Crush by retractor	10
Entrapment and immobilisation by fibrosis	23
Tethering by fibrosis to adjacent structures	27
Traction, with or without tethering	39
Ischaemia (rupture of femoral vessels)	1
Intraneural bleeding	3
Extraneural bleeding (haematoma)	20

[a]The sciatic trunk is considered as its two component nerves, the tibial and common peroneal
[b]Some cases of fibrosis probably represent initial crushing or organised haematoma

and deep lesion of a main nerve is extremely disheartening for patient and surgeon alike and it is important to emphasise that much can be achieved by urgent reexploration and relief of the cause (Table 2.1).

Setting aside cases of complete transection or the transient conduction block from concussion or compression the lesion is invariably mixed so that some fibres are intact, others recover as conduction block or favourable degenerative lesions whilst yet others never recover. It is because of this that neurophysiological investigations (NPI), which are helpful in confirming the level and the extent of lesion, cannot reliably indicate prognosis. The behaviour of the Tinel sign is less reliable than it is in nerves injured by fracture or dislocation. Two features are important. Severe pain indicates that the cause of the lesion remains active and in these cases urgent reexploration is indicated. Delayed onset of pain and lesion strongly suggests bleeding. Bleeding is the most likely explanation when the lesion of the nerve deepens whilst under observation and when there is worsening pain. If decompression of the nerve is done within 3 h, patient and surgeon alike can confidently expect abolition of the pain and speedy recovery. After that interval it is likely that pain will be improved but recovery will probably be incomplete.

The response of pain to reexploration is often gratifying and this is an important reason for considering re operation upon the nerves even in late cases [24]. It must be said that no patient should be sent to a Pain clinic until a clear diagnosis of the cause of the pain has been made.

2.4.1 Acute Ischaemia

Whilst it is hard to separate the effects of acute ischaemia from those of other physical agents there are examples of the effects of ischaemia alone upon conducting tissue. Harriman [19] examined the lower limb soon after amputation in a case

where femoral embolectomy successfully restored flow but failed to relieve severe pain. There were areas of muscle infarction but these were confined to the thigh, the muscles below the knee were normal in colour. The nerves in the leg were infarcted. The stump of the sciatic nerve appeared normal but as the nerves passed distally they became soft and grey and sections showed swelling of the myelin sheaths and axons with only a scanty cellular reaction in the epineurium.

2.4.2 Ischaemia from Tamponade

The most dramatic examples are provided by the catastrophic cases of infarction of the spinal cord after interscalene block or by the injection of local anaesthetic and other agents into the invertebral foramina. Flow through radicular vessels passing to the anterior spinal artery is occluded. One such case [10] was caused by a diagnostic block of the right sixth cervical nerve in a 58 year old man. Under radiological control a 22 gauge needle was positioned in the posterior caudal corner of the foramen of C6 so that the tip of the needle lay well within the foramen. No cerebrospinal fluid was aspirated and radio contrast medium showed spreading alongside the nerve root. A mixture of 0.5 ml of bupivicaine and 0.5 ml triamcinolone was injected around the nerve root over a 1 min period. At about 1 min after this the patient suddenly developed flaccid paralysis and severe breathing difficulties. It soon became clear that there was a complete lesion of the cord from C3. An MR scan at 6 h showed increased signal intensity from C2 to T1 and a further MR scan at 24 h confirmed infarction of the spinal cord. The patient later died. Nash [25] described other cases of severe cord ischaemia following radio frequency lesioning of dorsal root ganglia and root sleeve injection and he emphasised the importance of the blood vessels passing with the spinal nerves through the intervertebral foramina. A similar mechanism underlies the cases of anterior cord infarction following interscalene block in which the infusion of relatively large volumes of fluid deep to the unyielding prevertebral fascia disturbs flow within the radicular vessels accompanying the spinal nerves. The permanent defects in eight patients adequately followed are set out in Table 2.2 and illustrated in one patient in (Fig 2.16).

Case report: A 48 year old woman underwent arthroscopic decompression for her painful left shoulder. An interscalene block was performed after induction of anaesthetic using 20 ml of 0.5 % bupivicaine under stimulator control. She developed hypotension and was slow to breathe spontaneously. On awakening she had numbness and weakness in all four limbs. There was flaccid paralysis in the left upper limb and of the C5/6 and C7 muscles in the right upper limb. There were severe defects in light touch and temperature sense in the left forequarter with lesser abnormalities in the right hand. Joint position and vibration sense were maintained. There was vasomotor and sudomotor paralysis in the right hand. There was no Bernard Horner syndrome. There was some affection of both lower limbs with weakness of the muscles about the hip and defects in light touch and temperature

Table 2.2 Permanent defects in eight patients after interscalene block, followed for at least 3 years

				Lower motor neurone				Spinothalamic tract		
Age	Sex	Side	Phrenic palsy	Ipsilateral	Contralateral	Cervical sympathetic	Upper limb sympathetic	Ipsilateral	Contralateral	Pain
52	M	R	Yes	C4, 5, 6	C5	No	No	C5, 6, 7		Neck and shoulders PNI 3: VAS 8
60	F	R	No	C5, 6, 7, 8, T1	C6, 7	No	No	C7, 8, T1	Lower limb	Resolved by day 7
55	F	L	Yes	C5, 6, 7, 8, T1	–	Yes	Bilateral	C6, 7, 8, T1	Upper and lower limbs	PNI 3: VAS 8 neck and shoulders
46	F	R	No	C7, 8	–	No	No	–	–	No
46	F	L	No	C5, 6, 7, 8, T1	–	No	No	–	–	PNI 3: VAS 8 neck and shoulder
60	F	R	Yes	C7, 8, T1	T1	No	Bilateral	C5, 6, 7, 8, T1	Upper limb C7, 8, T1 Lower limb	PNI 3: VAS 8 neck, shoulders and right hand
4[a]	M	R	Yes	C7, 8, T1		Yes	No	C5, 6, 7, 8, T1	T3, T4	No
48	F	L	No	C7, 8, T1	C8, T1	No	Bilateral	C5, 6, 7, 8, T1	C7, 8, T1	PNI 3: VAS 8 shoulders and neck

[a]The bulbar palsy had recovered by 5 months in case 4

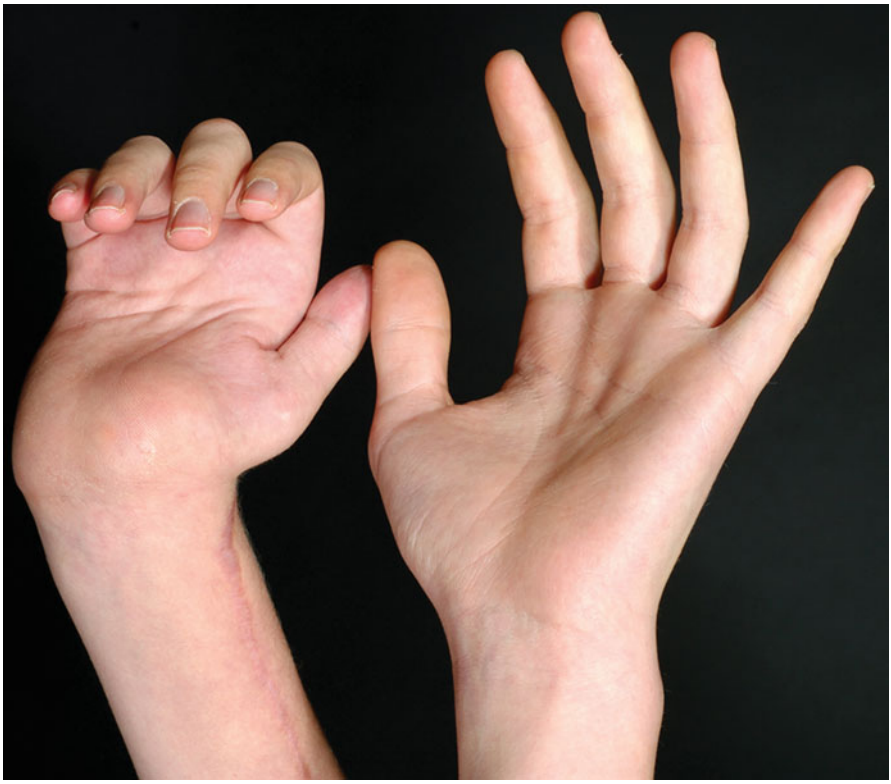


Fig. 2.16 The hands of an 11 year old boy 7 years after anterior cord infarction caused by interscalene block

sense in the right lower limb. Her reflexes were brisk but there was no clonus. A dissociated sensory loss was evident on the right side of the trunk, the level for light touch was T4, that for pinprick sense was T6. An MR scan done on the day after injury was reported as normal but when this was repeated at 5 days bilateral linear patchy areas of high signal were seen, extending from C3 to T5. Somatosensory evoked potentials (SSEPs) at 11 days were normal; sensory action potentials were maintained in all four limbs and motor conduction in both lower limbs was preserved. Motor conduction was absent in the median and ulnar nerves in both upper limbs. By 6 months the lower limbs had recovered. There was complete paralysis of C8 and T1 muscles on the right and of C7, C8 and T1 muscles on the left. By now she experienced severe burning pain in the shoulder girdles and chest. Quantitative sensory testing revealed normal vibration sense in all four limbs, a marked elevation of thermal thresholds especially so in the left forequarter and reduced sweating in both hands. It seems that motor neurones in the anterior horns of the segment C7, C8 and T1 were infarcted, that there was some involvement of the sympathetic outflow to both of the upper limbs and that there was a primary spinothalamic syndrome. She remains in severe pain.

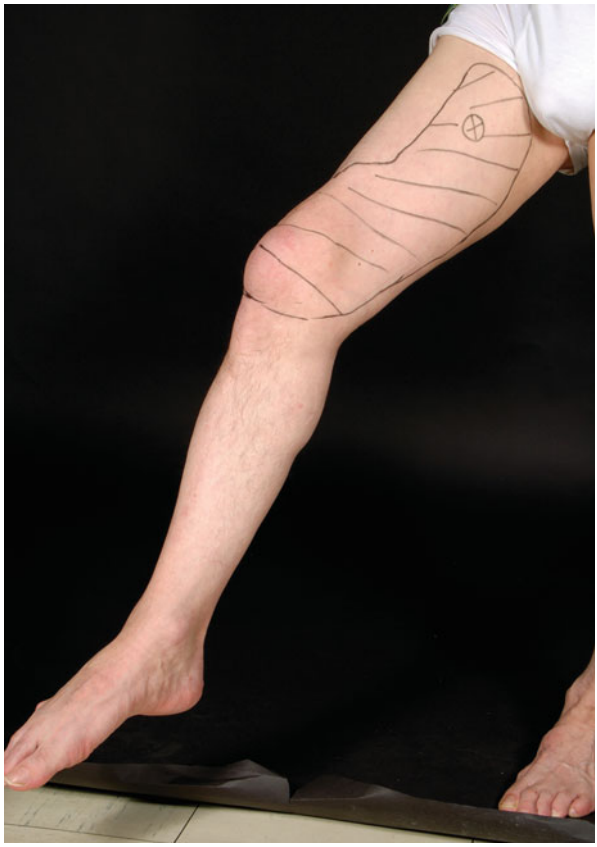


Fig. 2.17 Recovering femoral palsy from haematoma in the femoral triangle in a 69 year old farmer who was taking warfarin after aortic valve replacement. Two months previously he had injured his thigh whilst vaulting a gate. He experienced severe pain for 24 h but this recovered spontaneously. The area of sensory loss is outlined and the site of the Tinel sign marked. Recovery was good but not complete by 6 months

2.4.3 Ischaemia and Acute Compression Within Neurovascular Fascial Compartments

This is caused by bleeding or infusion of fluid into a fascial compartment which encloses the nerve and axial vessels but not muscle. Nerves especially at risk include the femoral nerve in the groin, the ulnar nerve in the forearm and the tibial nerve in the leg (Fig. 2.17). The syndrome is a common complication of skeletal injury, of nerve blocks and of vessel puncture. The medial brachial fascial compartment syndrome described by Wilbourn [38] is probably responsible for the majority of infraclavicular plexopathies following axillary regional block and also for many of the neurovascular injuries which result from closed or penetrating injuries in this region. The progression of the lesion is characteristic: there is, almost always, pain accompanied by dysaesthesiae; loss of sensation soon follows and then, over the next 2–3 h, paralysis ensues. Wilbourne's comment bears repeating: "distal pulses are normal as they are with most compartment syndromes because the elevated pressure, although sufficient to collapse the vasae nervori, is far below mean arterial pressure. Ultrasound, MR and CT may reveal the vascular lesion, but, considering the very brief time

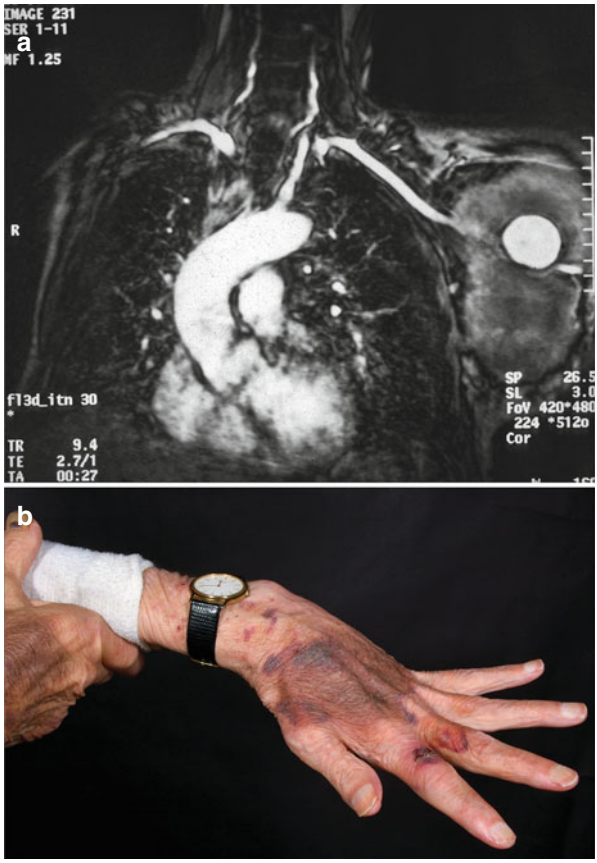


Fig. 2.18 Fracture of left proximal humerus was complicated by expanding haematoma in a 63 year old man. Two attempts to occlude the torn posterior circumflex artery by interventional radiology failed. He was seen at 8 weeks by which time he was in right heart failure, in great pain and he had a complete infraclavicular plexopathy on the left side. Six litres of altered blood were removed from the axilla. His pain was relieved. Recovery was particularly poor in the radial and median nerves. (**a**) MR angiogram before operation. (**b**) The left hand 4 years after operation

available for surgical decompression before irreversible nerve damage occurs, obtaining these is rarely justified". The 16 patients described by Stenning, Drew and Birch [33] exemplify the syndrome. There was, in all of these cases, an injury to the axillary artery or one of its offsets caused by dislocation of the shoulder or fracture of the proximal humerus. The diagnosis of continuing bleeding into the axillary sheath was made by the delayed onset of nerve palsy or the deepening of the lesion whilst under observation. There were 87 nerve palsies. A favourable outcome was seen in all cases where urgent repair of the artery and decompression of the axillary sheath was performed. The outcome for function within the hand in another patient in whom the diagnosis was delayed by 8 weeks is shown in (Fig. 2.18).

2.4.4 Ischaemia by Acute Compression from Swollen Muscle

The effect upon the nerves is at least as rapid as it is in cases of compression within a neurovascular sheath. The response to correction within 3 h is almost

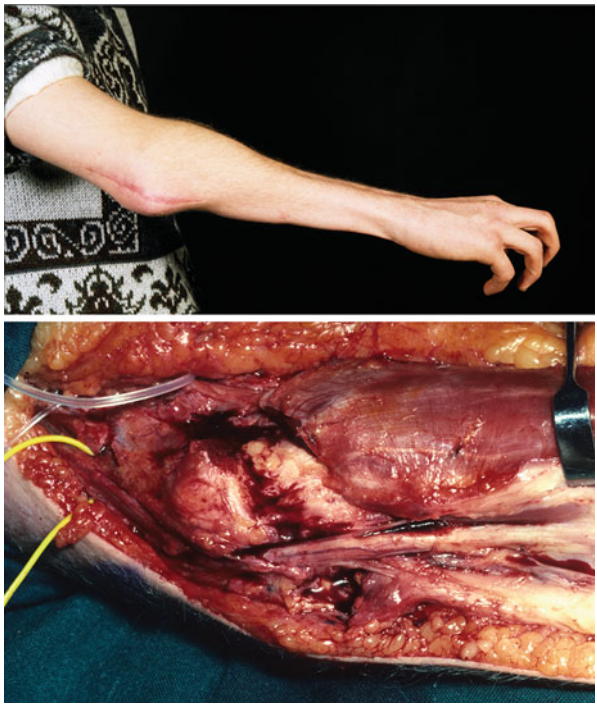


Fig. 2.19 Volkmann's ischaemic contracture. *Below*: the ulnar nerve exposed during flexor muscle slide 8 weeks after supracondylar fracture. The epineurial vessels and also the ulnar recurrent collateral vessels are occluded and the nerve is compressed by the swollen infarcted muscle. *Above*: the appearance of the hand 14 years later

always gratifying and the consequences of delay before that correction are particularly severe. The vascular arrangements of nerves are such that injuries of main arteries are more likely to produce infarction of muscle than necrosis of nerve trunks. The evidence is distorted by the circumstance that ischaemia is rarely complete. Even nerves which have been seriously ischaemic for 36 h have been seen to recover adequate function. In one case of ischaemia after supracondylar fracture of the humerus complicated by thrombosis of the brachial artery, the median nerve was seen at operation to lack all vascular pedicles from elbow to wrist. It lay in the middle of the completely infarcted flexor muscles of the forearm. Three years later there was recovery of sweating and of impaired sensation in its area of distribution. The effect of increasing pressure within the osseo-fascial compartment upon the vessels running with the nerves at the elbow is illustrated in (Fig. 2.19). These vessels provide the main pathway for collateral circulation at the elbow after cessation of flow through the axial artery [8]. Wajcberg et al. [37] used high resolution ultrasonography to measure the rate of flow through the brachial artery in adults and in children. Flow was calculated by multiplying the velocity-time interval of the Doppler flow signal by the heart rate and the cross-sectional area of the vessel according to Laplace equation: $BF(\text{blood flow}) = [\pi \times (D/2)]^2 \times FV(\text{flow velocity})$. The mean diameter of the brachial artery in children aged between 4 and 5 years is 2.7 mm, which provides a resting flow of about 200 ml/min. The reader will at once note the significance of the diameter of the vessel from the equation of Laplace and

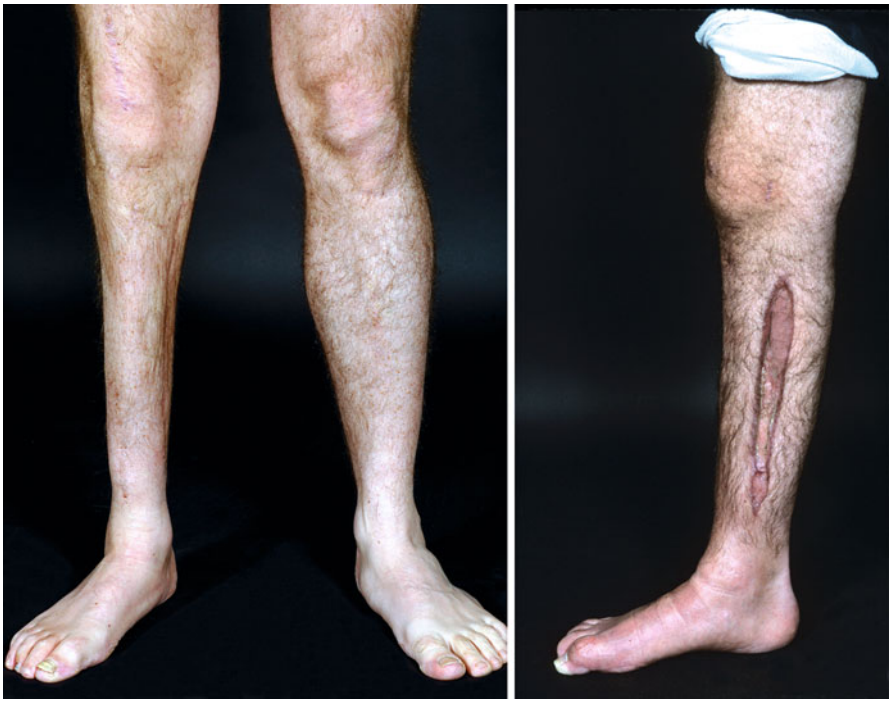


Fig. 2.20 The popliteal artery was lacerated during arthroscopic reconstruction of the posterior cruciate ligament. There was 24 h delay before the vessel was repaired. This was followed by rhabdomyolysis and acute renal failure. Most of the muscle in the leg was excised. Nine months later step elongation of the flexor tendons improved the posture of the foot and the decompression of the tibial nerve was followed by considerable recovery of sensation and some recovery into the small muscles of the foot

this factor is emphasized by Poiseuille's law which states that flow through a vessel is affected by three variables; the radius of the cylindrical vessel, the total tension in the wall and the pressure gradient. Poiseuille's law is the physical law describing the volume of flow (Φ) of an incompressible uniform viscous liquid, where R is the internal radius of the tube, P the pressure difference between the two ends, the dynamic fluid viscosity and L the total length of the tube.

$$\Phi = \frac{\pi R^4}{8\eta} \frac{[\Delta P]}{L}$$

The diameter of the superior ulnar collateral artery at the elbow in children is no more than 1 mm. This calibre provides flow of about 20 ml/min assuming that the pressure gradient is the same as that in the brachial artery itself. These facts must be borne in mind by any clinician inclined to the view that cessation of flow through the brachial [8], or other main, artery is a matter of little consequence (Fig. 2.20).

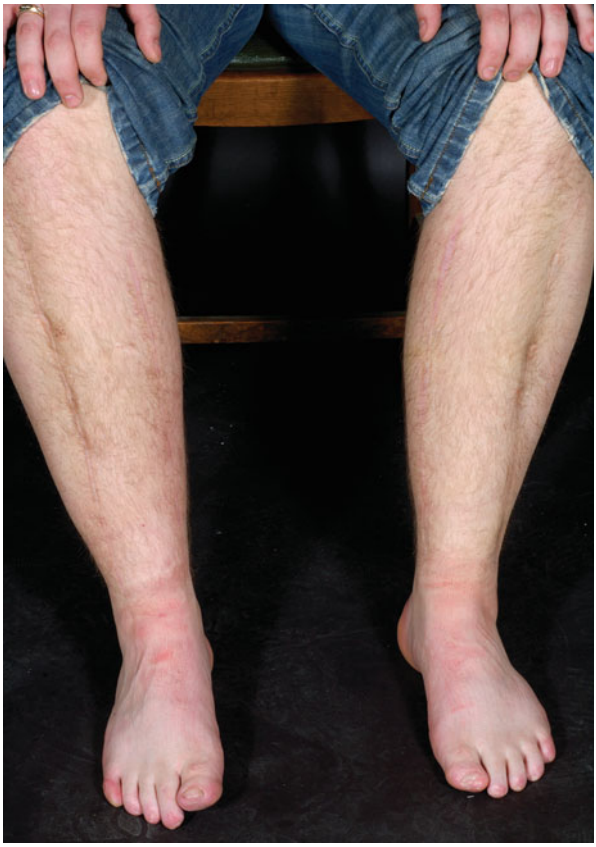


Fig. 2.21 A 20 year old man developed staphylococcal septicaemia after an operation for pilonidal sinus. Rhabdomyolysis led to multiple organ failure. Early fasciotomy and wide excision of the muscles of the anterior compartment of both legs was performed. There was recovery of sensation and sympathetic function in the foot: the small muscles also recovered

Critical illness neuropathy may develop in patients who develop multi organ failure or sepsis. It is possible that the intense compression of nerve trunks within oedematous and swollen limbs contributes to this disorder (Fig. 2.21).

2.4.5 Ischaemia Caused by Traction

Lundborg and Rydevik [22] showed that 8 % elongation of a segment of a nerve could cause impairment to vascular flow and that an elongation of 10–15 % could arrest all blood flow. Relaxation within 30 min would, in most cases, lead to restoration of flow and conductivity. Nerves which are stretched over an expanding haematoma sustain severe lesions, especially so when the false aneurysm is pulsatile. This type of injury is associated with causalgia. Recovery for nerves embedded within the wall of the sac is generally poor (Fig. 2.22). The situation can usually be retrieved by urgent and accurate action. The recovery of some function through nerves strangled by fibrosis for months or even years, provides an indication of their

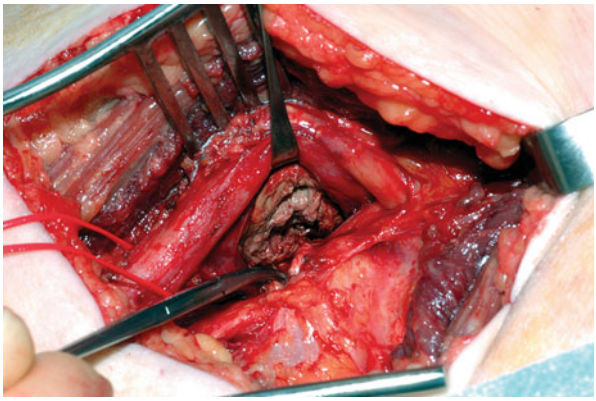


Fig. 2.22 A 68 year old woman developed an intensely painful and rapidly deepening lesion of the sciatic nerve on the evening of operation for total hip arthroplasty. The nerve was exposed 3 months later and it was found stretched over an organised haematoma. Her pain improved but the nerve did not recover

resilience: on the other hand relief of the cause within 3 or 4 h offers the only real prospect for complete recovery. The different responses evoked by acute compression of a nerve trunk within a fascial sheath versus the insidious effect of a haematoma is illustrated by the following example.

Case report: A healthy 28 year old woman suffered a fracture of her tibia and fibula. This was treated by open reduction, internal fixation and bone graft from the ipsilateral iliac crest 7 days later. A femoral nerve block was given before induction of general anaesthesia. In spite of her complaints of intense searing, shooting pain radiating down the front of her thigh and leg the anaesthetist persisted with the infusion of local anaesthetic into the groin crease. Upon awakening the patient continued to experience intense pain which required several hundred grams of morphine during the next 3 days. She became aware that she could not feel the sole of her foot and could not move her ankle. Her haemoglobin was measured at less than 8 g/dl on the third post operative day. By 7 months it was appreciated that she had developed not only a significant femoral neuropathy but also a profound sacral plexopathy. The femoral neuropathy remained very painful and she continued to experience dynamic mechanical allodynia to stimulation of the skin of the front of the thigh. She was unable to walk because of pain, weakness and loss of position sense throughout the left lower limb.

NPI were performed 9 months after her injury. Sensory and motor conduction studies were normal. Electromyography revealed a few small polyphasic motor units in the left vastus medialis, otherwise the motor units were normal. Quantitative sensory testing (QST) found that joint position sense was very poor throughout the left lower limb, indeed it could not be detected for the hip. On the other hand vibration thresholds at the left great toe were within normal limits. The thresholds to monofilament stimulation and warm sensation were increased throughout the left lower limb and there was a rather patchy loss of pinprick sense. The pain was exacerbated by extension at the hip. The femoral nerve was explored 11 months after her injury. The fascia surrounding the nerve was greatly thickened. The nerve was narrowed, and inflamed with a much diminished epineurial circulation. There was no sign of injury to any of the individual bundles within the trunk. The nerve was

decompressed over some 10 cm. A tissue catheter was placed for the infusion of local anaesthetic about the proximal part of the femoral nerve for 48 h. There was considerable improvement in her pain and in her ability to walk. By 9 months after operation the muscles about both hips and knees were graded 5 by the MRC system but she still had extremely poor joint position sense throughout the left lower limb.

The strangulation of the femoral nerve was provoked by injection of bupivicaine into the epineurium and by haematoma within the enveloping fascial sheath. Pain was so severe that the more insidious and painless lesion of the sacral plexus caused by continuing bleeding from the donor site at the ipsilateral iliac crest escaped attention. The effect of the haematoma was particularly severe for the largest myelinated afferent fibres: some never recovered. The myelinated efferent fibres and the smaller myelinated afferent fibres recovered over the course of 3 months: in these the lesion was one of conduction block. *The severe pain at the beginning of the femoral nerve block should have led to instant cessation of the injection.*

2.4.6 Compression: Acute

Closed compression lesion, in which an external force is applied to the limb of a conscious patient usually leads to a conduction block. The lesion in the more severe compression injuries is usually much more than a conduction block. The tempo of recovery varies from one population of nerve fibres to another and it is not unusual to see poor recovery for somatic unmyelinated fibres. As a rule, the sympathetic efferent fibres suffer least. Some nerves never recover. More sustained compression leads to deeper lesions.

Case report: A slim healthy 22 year old woman sustained a mid shaft right femoral fracture through an area of fibrous dysplasia. The operation of internal fixation was difficult and lasted for 7 h during which time the contralateral left leg was kept in a flexed abducted position to permit the use of an image intensifier. A muscle relaxant (Rocuronium) was used. She awoke with a complete left sciatic palsy. There was no bruising in the left thigh or the buttock. NPI at 2 months after operation showed a complete degenerative lesion of the nerves and muscles of the leg and foot with extensive denervation in the hamstring muscles. By 8 months after the incident she reported increasingly severe pain but some recovery of feeling into her foot. Recovery of power of the knee flexor muscles was measured at MRC Grade 4 and there was perceptible activity in the flexor muscles of the heel. There was still complete vasomotor and sudomotor paralysis in the foot. There were strong Tinel signs for both divisions of the sciatic nerve in the leg indicating a rate of regeneration of a little more than 2 mm/day. By 14 months all muscle groups in the leg and foot were recovering and she could localise light touch to the skin of the foot. There was still sympathetic paralysis in the sole of the foot and she still had pain. NPI were repeated; no sensory conduction could be demonstrated in the nerves of the leg nor was motor conduction demonstrable for the tibial nerve. Motor conduction in the common peroneal nerve was reduced and slowed. Electromyography of the leg

muscles revealed persisting denervation with reinnervation by collateral sprouting with many wide polyphasic units and irregular recruitment. By 18 months she showed further recovery and some improvement of her pain. Quantitative sensory testing showed elevated thermal thresholds in the plantar skin but the threshold to cooling fell within normal limits. The threshold to monofilament sensation was elevated and pinprick was felt as an unpleasantly sharp sensation from the mid calf down. Sweating in the left sole was reduced to about one half of that on the right. The threshold to vibration was markedly elevated. It seems that the largest and the smallest fibres suffered most.

Such lesions can prove very serious in the growing child because of the disturbance of growth

Case report: A 12 year old boy fell from a swing and experienced much pain. There was deep bruising in the left buttock and he presented with a high sciatic palsy which involved the superior gluteal nerve. The common peroneal nerve was more severely affected, NPI at 6 weeks revealing loss of conduction and denervation of all of the muscles innervated by that nerve. Sensory conduction was diminished in the tibial division and there were signs of partial denervation of all tibial muscles. There was considerable muscular recovery by 1 year. Sensory conduction in the tibial nerve had recovered. There was no sensory conduction in the common peroneal nerve and motor conduction was slowed and reduced in amplitude to just under 10 % of the uninjured side. At 2 years the nerve had recovered but the left lower limb was shorter by 1.5 cm and the heel was fixed in equino varus.

2.4.7 Crush

Crush injuries stand with inadvertent transection, traction or neglected ischaemia as one of the four main causes of iatrogenous lesions. Although a crush injury might be considered as an extreme form of compression the clinical evidence suggests that it is rather more than that. Unless the cause is relieved swiftly the lesion of the nerve rapidly becomes a neurotmesis. The most extreme examples of this of course is when a nerve is encircled by a suture. Much depends on the material used and the tightness of the strangulation. Nerves have been divided by stainless steel wire used for the fixation of displaced fragments of bone. Whilst recovery might be anticipated after removal of a braided suture, within an hour or two, nerves do not recover after this time (Fig. 2.23). Not only do catgut sutures crush, they also provoke a brisk inflammatory response. Not only does an encircling suture strangle a nerve it also tethers it so that there is the added element of stretch. Severe pain is usual and it is often related to posture. Attempted stretching of the limb against the tethered nerve provokes intense pain (Fig. 2.24). Forty patients in whom main nerves had become inadvertently strangled by a suture passed around, or through, the nerve have been operated. When the suture was removed within hours of the operation there was instant relief of pain and a high level of recovery. Relief of pain was usual after removal of the suture up to about 2 weeks. In these cases recovery was always

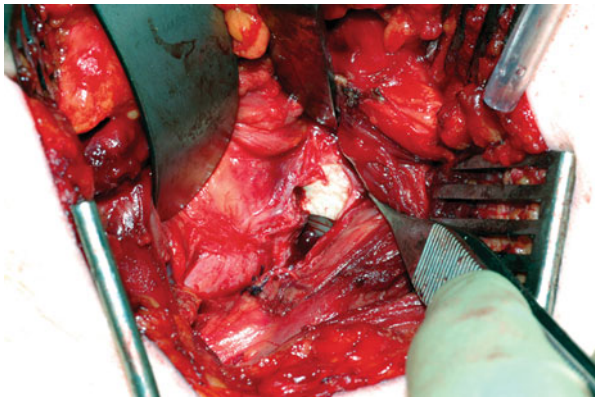


Fig. 2.23 A 48 year old woman experienced severe pain and an incomplete lesion of the sciatic nerve after operation of total hip arthroplasty. The nerve was exposed 8 weeks later. It had been transfixed by a braided suture. There was some moderation in her pain but little recovery for the nerve

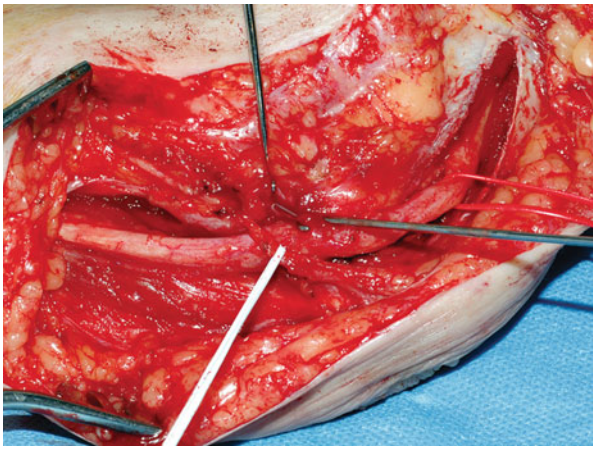


Fig. 2.24 A 38 year old woman experienced intense pain and a partial lesion of the ulnar nerve after repair of the capsule of the joint using an arthroscope. Her pain was worsened by attempted extension at the elbow. The nerve was exposed 4 days later, it had been caught by a clip. The clip was removed and her pain was relieved. There was considerable recovery in the ulnar nerve but she did not recover vasomotor and sudomotor control and there was lasting weakness of the small muscles

incomplete and when the ligature was removed later there was very little useful recovery. Hindsight suggests that that the better course might have been resection and suture of the relatively short damaged segment.

Nerves entrapped within fractures or joints certainly pass through a period of ischaemic conduction block which may last for as long as 2 or 3 days. After that there is demyelination but recovery may be anticipated if the nerve is set free within 7–10 days. The situation is made very much worse if a compression plate or tension band is applied to the fracture without extricating the nerve (Fig. 2.25). The resected material in such cases shows transection of normal structures with an interposed zone of dense fibrosis. The effect of compression between the bone and the plate is illustrated by a case treated by an alert surgeon who, drawing to the close of a

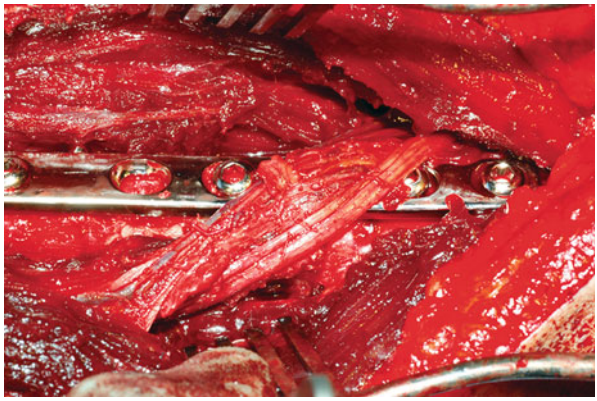


Fig. 2.25 The appearance of a radial nerve after extrication from beneath a compression plate 48 h after first operation. There was relief of pain but only incomplete recovery so that later flexor to extensor transfer was necessary

difficult operation of internal fixation of a fractured shaft of humerus, realised that the hitherto protected radial nerve had inadvertently slipped between the plate and the bone. The nerve was at once extricated. The duration of compression was, at the most, 5 min. The nerve was re-explored at that surgeon's insistence some 6 weeks later even though an advancing Tinel sign offered the prospect of spontaneous recovery. The nerve had reconstituted, the epineurium was thickened but the epineurial vessels were patent and the bundles within had not been severed. Recovery proceeded uneventfully as an axonotmesis.

2.4.8 Traction

Peripheral nerves outside the spinal canal have considerable tensile strength, but their function is damaged by an elongation of 12 % or more, the extent of damage varying with the suddenness and the length of time during which that elongation is maintained (Fig. 2.26). It is difficult to separate the effects of stretch upon the conducting tissue from those imposed upon the vessels. Haftek [17] described how, at first, elongation is permitted by the elongation of the epineurium and the straightening of the irregular course of the fibres within the fascicles. The spiral bands of Fontana disappear, confirming that the banding appearance of the peripheral nerve is due to the wave-like alignment of its individual nerve fibres. Haftek added the observation that "before rupture of the perineurium the damage to the nerve fibres is either neurapraxia or axonotmesis, because the endoneurial sheaths and Schwann fibres remain intact". Ochs et al. [28] studied the effects of stretch upon isolated segments of nerve placed within an oxygen chamber so that anoxia was prevented. A very light stretch straightened out the zig zag disposition of the nerve fibres. The spiral bands of Fontana were erased when the nerve was elongated to about 15 %. An applied tension of 2 g or more induced rapid beading of the nerve fibres which was rapidly reversible. The compound nerve action potential was actually augmented in the earliest stages before it fell away.

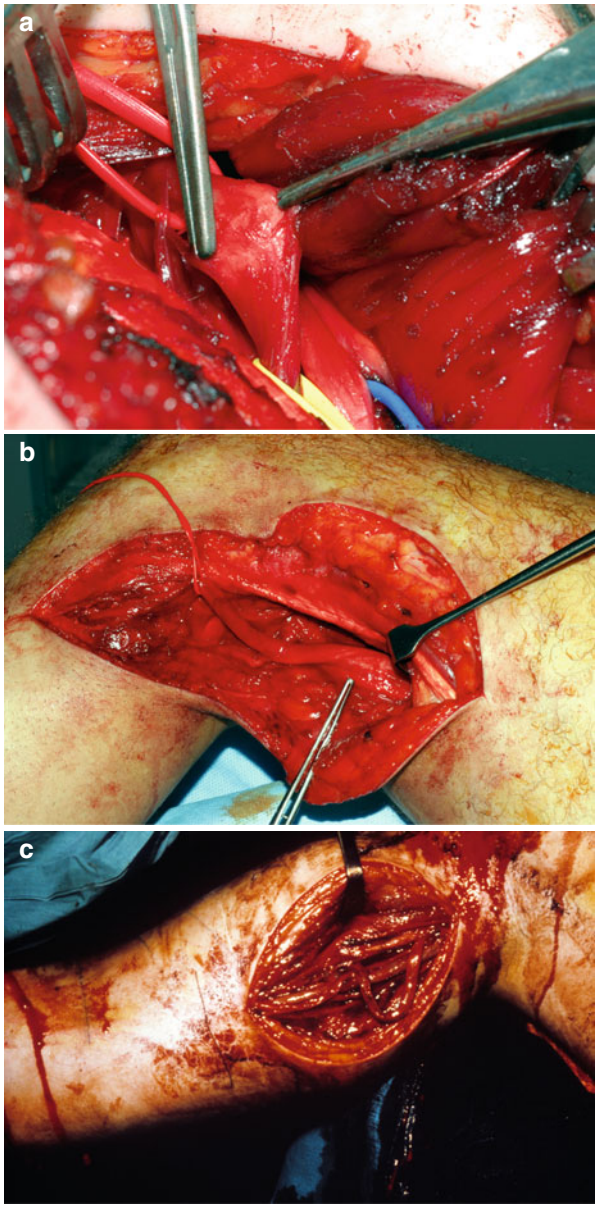


Fig. 2.26 Traction injury. (**a**) The circumflex nerve exposed 4 days after anterior dislocation of shoulder. The bundles had ruptured and retracted within the intact epineurium. (**b**) Traction injury of the common peroneal nerve from varus injury at the knee. There was extensive recovery over the course of 9 months (axonotmesis). (**c**) A more violent traction injury of the right upper limb. The median nerve was elongated by more than 100 %. Over the course of the next 3 years there was recovery of cutaneous sensation in the hand, and some recovery of the flexor muscles of the forearm. The sympathetic fibres never recovered

It is common, in closed traction lesion, to see that the epineurium has ruptured but the perineurium within remains intact, albeit stretched. These injuries are usually complicated by bleeding into the epineurium, extending over many centimetres. However cases have been encountered where the perineurium was ruptured even though the epineurium remained intact in nerves sharply angulated over a fragment of bone. It is much easier to recognise this pattern when the nerve is explored within

24 h of the injury (Fig. 2.26). In the extreme traction injury the nerve is ruptured or avulsed from muscle. The wide recoil of the stumps can only be reduced by urgent operation. Nerves have been destroyed over a length of 15 cm by the action of a drill during operation.

The manipulation of any joint which has been fixed for some time in a position of deformity always carries the risk of damage to nerves and vessels passing across it and accustomed to the position of deformity. In one case of attempted correction of long standing flexion deformity of both knees in an adult with cerebral palsy loss of pulses at the ankles was recognised by the staff of the recovery ward. No action was taken and above knee amputation of one lower limb proved necessary.

Case report: A fit man, in age 64 years, developed severe osteoarthritis in his right knee complicated by severe fixed flexion deformity. Capsulotomy was done to regain extension as the first step in total arthroplasty of the joint. A complete lesion of the tibial and common peroneal nerves was recognised on the evening of operation. Neurophysiological investigations at 22 months revealed no sensory or motor conduction. Considerable reinnervation of the tibial muscles was demonstrated and the distal muscles of the anterior compartment were also recovering (EHL, EDC) However, tibialis anterior was fibrosed, the muscle was silent and the concentric needle met with the characteristic, gritty resistance. In addition to the traction injury of the nerves it is likely that flow through the anterior tibial artery was interrupted.

Case report: A 13 year old girl with severe cerebral palsy was treated by anterior transfer of the hamstring muscles of the left knee with the object of correcting flexion deformity. On the day of operation she developed intense pain in the leg and foot. This did not respond to opiates and neither the child nor her mother were able to sleep for 8 weeks. The child lost a good deal of weight. We saw her at 8 weeks when it was clear that the nerve lesion, although deep, was not complete. The child had causalgia. The extent of discoloured skin matched the areas of intense mechanical allodynia. At operation both tibial and common peroneal nerves were found stretched and compressed, indeed strangled, by fascia and by scar in the popliteal fossa. The common peroneal nerve was reduced to about one half of normal diameter and the epineurial vessels were obliterated. The tibial nerve was inflamed and embedded in vascular adhesions. An external neurolysis was done, and a tissue catheter placed to permit infusion of local anaesthetic for 48 h after operation. The pain from the tibial nerve was improved, the pain from the common peroneal nerve persisted. Both nerves recovered. By about 9 months the child was able to tolerate shoes and weight bearing. Her mother described how vasomotor and sudomotor disturbance in the foot persisted even after pain had improved. In this case stretching of the nerves damaged the epineurial vessels, the myelin sheath and the axons, and this was compounded by compression from bleeding and by the persistence of inflammation induced by haematoma. A wide range of drugs had been used in an attempt to control the pain without considering the possibility of a persisting focal noxious agent at work.

There may be a case, not only for monitoring of nerve conduction, but also the flow through adjacent main arteries before, during and after operations for the correction of severe flexion deformity at the knee and other joints [23].

Somewhat similar circumstances obtain in limb lengthening operations, even though it is usual to effect a change slowly, over a period of weeks. Nogueira et al. [26] used a pressure sense monitoring device during 814 limb lengthening procedures. Seventy six (9.3 %) nerve lesions occurred. Nerves were most at risk in double level lengthening of the tibia and in skeletal dysplasia. Most of the affected nerves were decompressed; 74 of the 76 recovered. Clear conclusions are drawn from this admirable work: the rate of lengthening should be slow; affected nerves should be decompressed as soon as possible; monitoring of nerve function by a pressure sense device is more sensitive than clinical examination and the largest myelinated fibres are the most vulnerable. This work provides sound advice about prevention. Analysis of the deformity provides information about the potential distortion of neurovascular bundles; the drill hole for the frame screws should be made opposite to the bundle and neuromuscular blocking agents should not be used. Severe pain indicates damage to a nerve or vessel or both. If a Tinel sign is evoked by tapping the transfixion wire then it has certainly passed through, or close to, a trunk nerve.

Case report: A 35 year old woman with severe deformity at the knee and in the leg after operations for Blount's disease, was treated by two level osteotomy of the tibia and fibula stabilised by an Ilizarov frame. This was followed by a deep, but painless, palsy of the tibial and common peroneal nerves. The nerves were exposed at 14 days. They were unblemished. There was little recovery for either nerve at 18 months.

2.4.9 Thermal Injury

The effects of cold have been studied extensively. In the First World War "trench foot" was a common cause of disablement; in the Second World War "immersion foot" was a more common occurrence. Donaghy [13] describes the freezing injury of frost bite, in which there is tissue necrosis often with a clear demarcation between living and dead tissue. Wallerian degeneration is an early feature of these injuries. The second type of cold injury recognised by Donaghy follows prolonged immersion in cold water or prolonged exposure to cold around freezing point. There is damage to both myelinated and unmyelinated nerve fibres, possibly in a cycle of ischaemia and reperfusion.

In civil practice at the present time, it is rather the effects of heat that concern the clinician, principally because of damage to main nerves by the heat of polymerising cement during replacement arthroplasty. Nerves can be destroyed by extremes of heat, or by diathermy during operation. Xu and Pollock [39] examined physiologically and morphologically the effect of heat ranging from 47 to 58 °C on rat sciatic nerve. Unmyelinated fibres showed a greater direct vulnerability to hyperthermia, first manifest as a reversible conduction block and at higher temperatures by immediate axonal degeneration. Lower grade thermal injury caused a delayed selective loss of myelinated fibres secondary to a heat-induced angiopathy. It is necessary in considering risks to nerves during joint replacement,

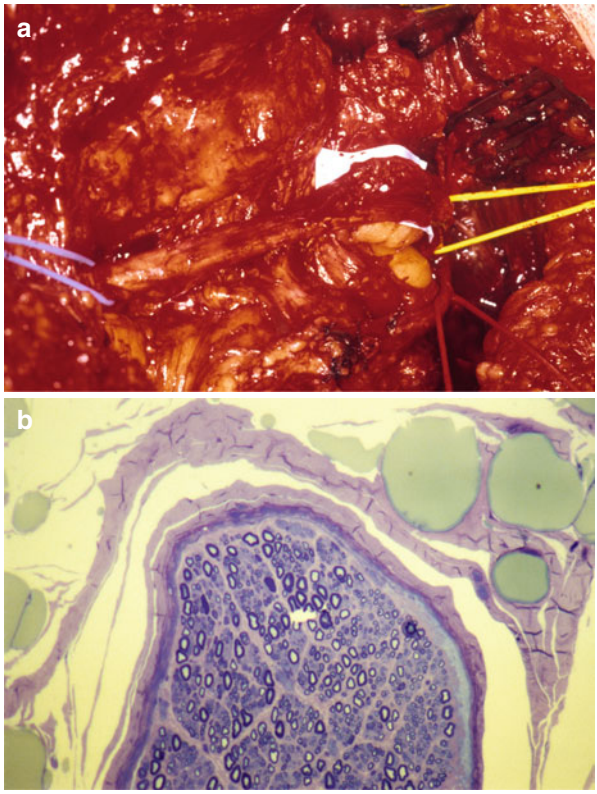


Fig. 2.27 A case of damage to the sciatic nerve by cement during operation for arthroplasty of the hip. (**a**) The sciatic nerve seen at operation a year after initial operation. The extruded cement can be seen in relation to the nerve. (**b**) The severely damaged common peroneal component was resected and grafted. The proximal stump appeared to be relatively healthy at 1 cm proximal to the cement. Toluidine blue ×100

to recall that the temperature of polymerising cement rises to 95 °C about 15 min after mixing, and remains above 70° for another 12 min. Birch et al. [2] had the opportunity of examining a length of sciatic nerve damaged by the heat of polymerising cement. They later took the opportunity of studying the effect of the heat of polymerising cement on the median nerve of an arm recently amputated because of a complete pre-ganglionic injury of the brachial plexus. The remarkable feature was the localised nature of the lesion: although at the site of burning there was destruction of axoplasm and disruption of myelin, a normal pattern of myelinated and unmyelinated fibres was found 10 mm from the margin of the cement (Figs. 2.27 and 2.28).

Flame burns cause extensive fibrosis of the nerves and adjacent tissues. Emergency incision of the encircling eschar must be considered (Fig. 2.29).

2.4.10 Electric Shock

Hobby and Laing [20] recognised four groups of electrical injury in 169 cases from a total of 3,300 patients: true electrical injury, from current passing from the

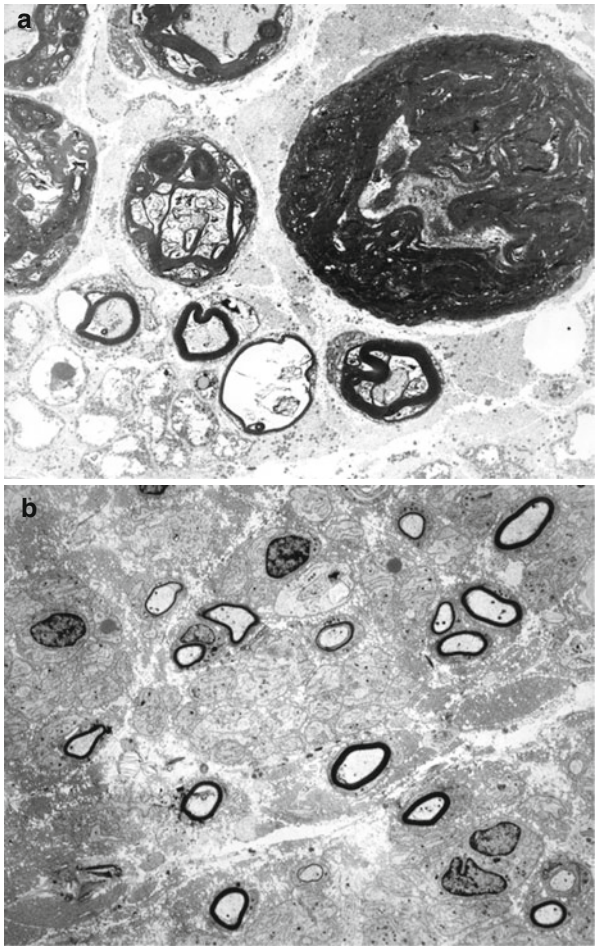


Fig. 2.28 Thermal damage to the median nerve after exposure to the heat of setting cement, showing the limited longitudinal extent of the neural lesion. (**a**) Virtual destruction of axoplasm and cellular elements at the site of the lesion ×3,600. (**b**) Healthy axons and collagen 10 mm from site of injury ×3,000 (EM)

conductor through the skin to the tissues; "arc" burn, where a current passes external to the body to the ground; secondary flame burn, from ignited clothing; and direct burn, from a hot electrical element. Reasonable spontaneous recovery occurred in those cases where the blood supply to the adjacent tissues was not destroyed. Clifton et al. [12] advise immediate repair of trunk nerves charred in electrical burns using nerve grafts which are covered by free full thickness skin flaps or by a vascularised nerve graft, such as the lateral cutaneous nerve of forearm within a free lateral arm flap.

When electrical contact is brief there may be little burning but the passage of electrical current causes break down of the cell membranes. Muscle and nerve cells are particularly susceptible to this process, termed electroporation, which is responsible for some of the immediate clinical signs such as muscle spasms and episthotonus.

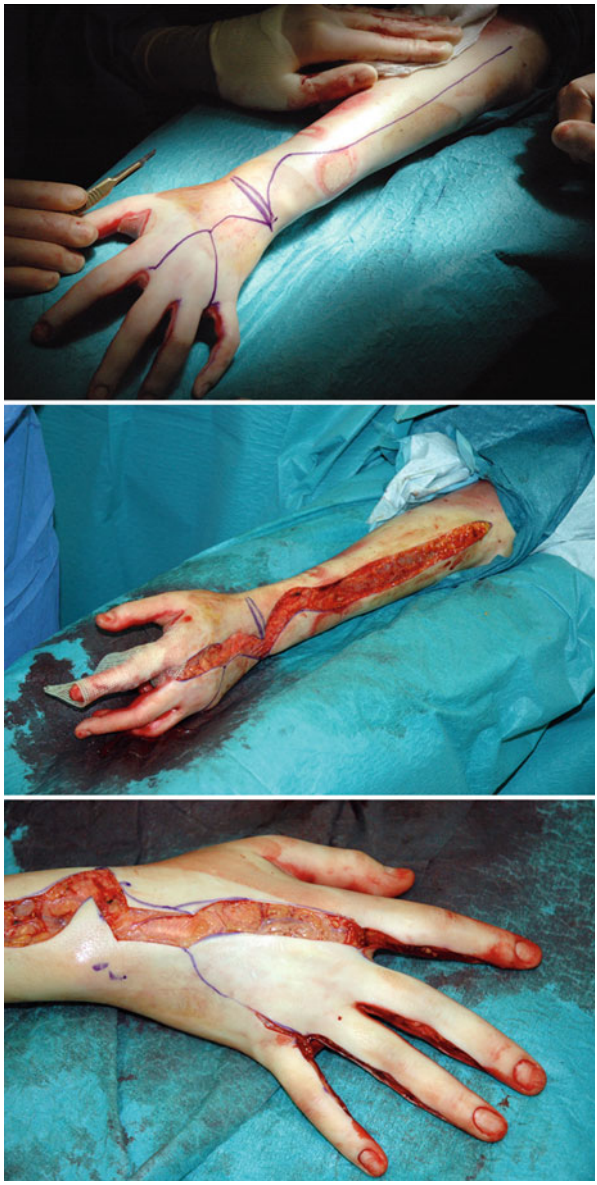


Fig. 2.29 Urgent and extensive division of the skin and fascia in a full thickness burn from petrol fire

Case report: A 34 year old man was struck by lightning. He suffered immediate loss of consciousness and cardiac arrest but was successfully revived. He was wearing a heavy gold chain at the time and a deep entry burn was seen on the right side of his neck with an exit burn on the left side. There were second degree flash burns to the chest, groin, upper and lower legs and there were superficial burns over

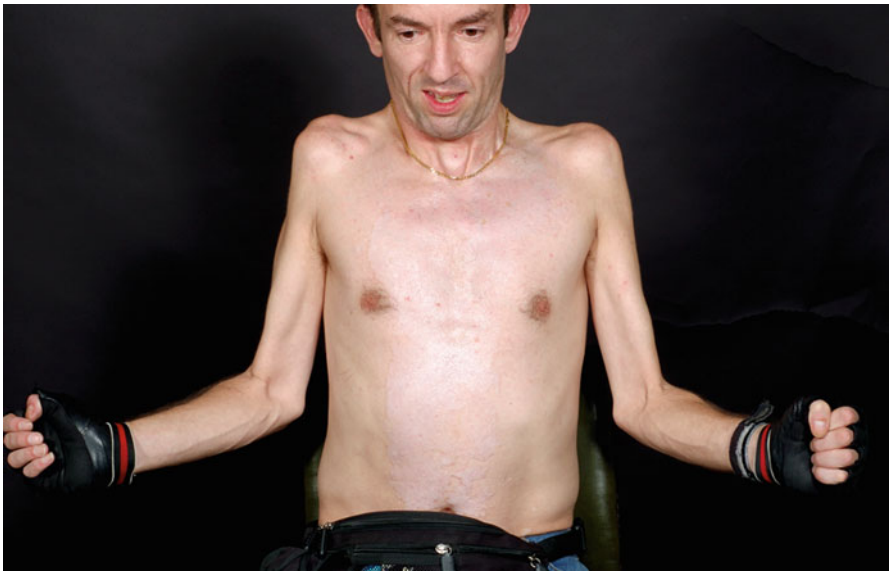


Fig. 2.30 Function in the upper limbs 2 years after lightning strike. Note the atrophy of the muscles of the shoulder and in the arm

the whole of his torso which formed a feathery or fern leaf pattern. He had a C5 tetraplegia with both urinary and faecal incontinence. By 2 years after injury he had regained considerable recovery of power in all four limbs so that he could walk and he had regained urinary and faecal control. QST showed normal sensory thresholds for all modalities in all four limbs except for an elevated vibration threshold in the right lower limb. NPI confirmed normal sensory and motor conduction but persisting denervation of the C5 and C6 myotomes. The initial lesion was severe, diffuse sensory motor and autonomic neuropathy. The recovery unmasked a permanent defect in the anterior horn C5 and C6. It is possible that the site of the entry and exit burns in the neck is relevant (Fig. 2.30).

2.4.11 Injection Injury

The perineurium may be lacerated by the point of a needle an event which causes severe pain in the conscious patient. If that complaint of pain is ignored and there is an injection into the nerve, the consequences may be severe. The damaging substances commonly injected into nerves are: steroid preparations; anaesthetic agents for intravenous use such as thiopentone; non steroidal anti-inflammatory drugs; anxiolytic agents such as diazepam; antibiotics; and local anaesthetics.

The nerves most commonly affected are the brachial plexus in the neck and axilla, the radial nerve in the arm, the median at the elbow and the sciatic in the buttock. Usually, the occurrence of severe local and radiated pain makes it plain that the drug has been injected into the nerve. Delayed onset probably arises from injecting near the nerve and later diffusion. Persistence of pain following an injection of steroid for carpal tunnel syndrome led to exploration of the median nerve in four patients at intervals ranging from 2 to 8 weeks after injection. The nerves were found inflamed and swollen over a length of some 3 cm and they were surrounded by filmy adhesions, but there were no signs of penetration of the trunk by the needle.

Experience with injection injuries in human beings does not always match with that of injection into the nerves of rats, just as the behaviour of the former is not regularly matched by that of the latter. Full recovery is by no means invariable; noxious injection is often followed by epineurial fibrosis and sometimes by dense intraneural scarring. The varying responses are illustrated by the following examples.

Case report: A 40 year old man of slender physique came to operation which required exposure of the common peroneal nerve at the knee. After induction of general anaesthesia the line of incision was infiltrated with 0.25 % bupivicaine. This provoked a twitch in the extensor muscles of the ankle indicating that the needle had been passed too deeply. The nerve was then exposed and the epineurium was greatly distended by the injected fluid. The bundles within were intact but the epineurial circulation had disappeared over a segment of some 4 cm. The epineurium was incised to decompress the nerve; the operation was then completed. On awakening he had a foot drop with sensory loss but no pain. By 6 h, sensation had recovered and there was the first evidence of recovery into the dorsiflexor muscles. Recovery was complete by 36 h. It is likely that the swift decompression saved the nerve.

Case report: A 45 year old woman with rheumatoid arthritis, for which she was taking prednisolone, experienced sudden severe shooting pain into her hand during venepuncture at the elbow. Pain persisted and sensation remained abnormal in the index and middle fingers whereas muscle power and sympathetic function remained intact. At 24 months the amplitude and velocity of sensory conduction from the affected digits was reduced to less than one half of normal. The median nerve was explored at the level of lesion which was marked by a strong Tinel sign. The epineurium was thickened and adherent to adjacent structures; epineurotomy revealed a neuroma of two bundles. After external neurolysis local anaesthetic was infused about the nerve by catheter for 24 h. Her pain improved.

Case report: A 6 year old boy was given an injection of antibiotics into the buttock and he experienced instantaneous and severe pain. When he was seen 2 years later there was a severe equinovarus deformity with shortening of the femur, the tibia and the foot on the ipsilateral side. Neurophysiological investigations at that

time revealed an extensive degenerative lesion. There was no motor or sensory conduction in the common peroneal nerve. Denervation of the muscles was widespread, but rather deeper for the common peroneal nerve than it was for the tibial division. The posture of the foot was improved by an extensive operation which included elongation of the heel cord, slide of the plantar muscles, shortening of the lateral column of the foot, calcaneal osteotomy and anterior transfer of tibialis posterior.

Pandian et al. [29] followed 65 lesions of sciatic nerve and radial nerves caused by intramuscular injections of various drugs. Axonopathy was confirmed in all cases, and reinnervation was demonstrated in only one third. Pain was usual. The consequences for the growing limb were particularly severe.

2.4.12 Vibration Injury

Stromberg [34] has improved understanding of this difficult and controversial field. The regular use of hand held vibrating tools may lead to a complex of symptoms, the hand–arm vibration syndrome. There are three groups of symptoms: sensory neural; vasospastic, and a combination of both. Cold intolerance presents as a significant symptom in one half of the patients with sensory neural symptoms. Impairment of nerve conduction, vibro tactile sense, and temperature sense in all patients occurs, more strongly expressed in the median nerve. Biopsies of the dorsal interosseous nerve revealed demyelination, endoneurial and perineurial fibrosis, and loss of axons.

2.4.13 Radiation and Peripheral Nerves

Vujaskovic [36] found that peripheral nerves were damaged by exposure exceeding 20 Gy. The more deeply seated larger fibres were worst affected: the first changes occurred within the axon, where there was increased density of microtubules and neurofilaments. The lesion is perhaps best considered as (1) a lesion from external compression exerted by fibrosis of soft tissue and (2) an intrinsic lesion of the nerve. The latter affects the axon, the Schwann cells and the myelin sheath; it is associated with vasculitis, which leads eventually to fibrosis (Fig. 2.31). The affection may extend to the main vessel [4, 13] (Fig. 2.32). It seems likely that the dose of radiation tolerated by neural tissue depends broadly on the total dose and the period of time over which it is given, but evidently there are individual variations and there may indeed be individual susceptibilities.

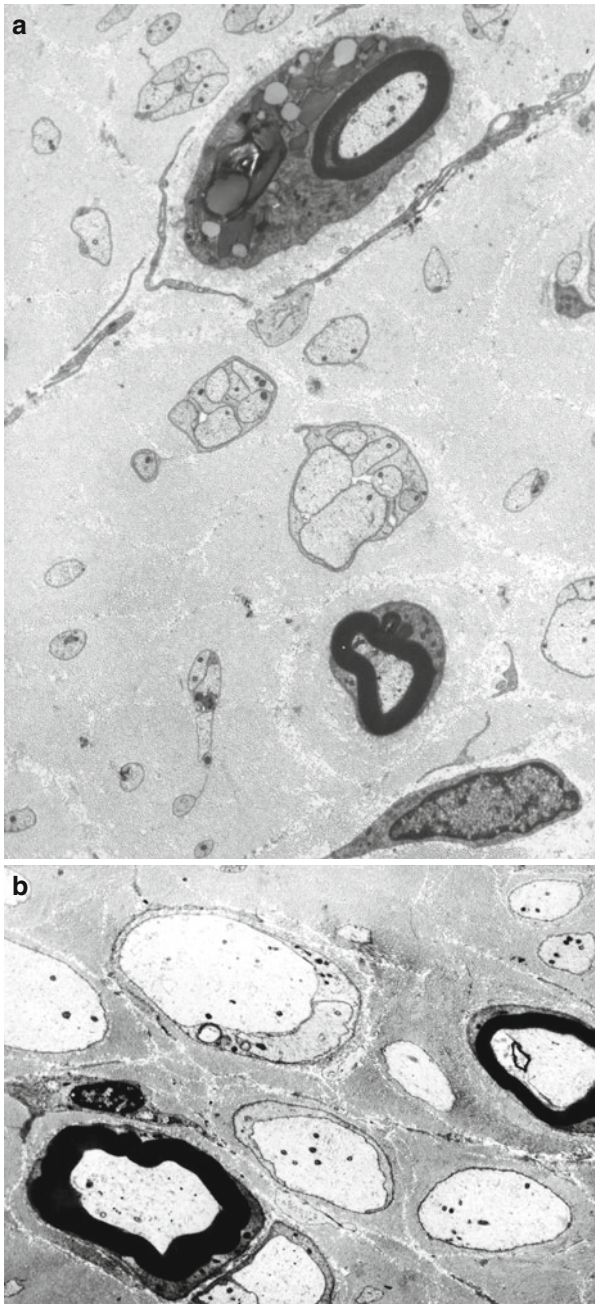


Fig. 2.31 Radiation neuropathy. Electron microphotographs of specimens from biopsy of the lateral trunk of the brachial plexus taken two and a half years after radiotherapy for cancer of the breast. (**a**) Extensive collagenisation, loss of axons and myelin ×4,125. (**b**) Extensive demyelination ×4,125 (EM)

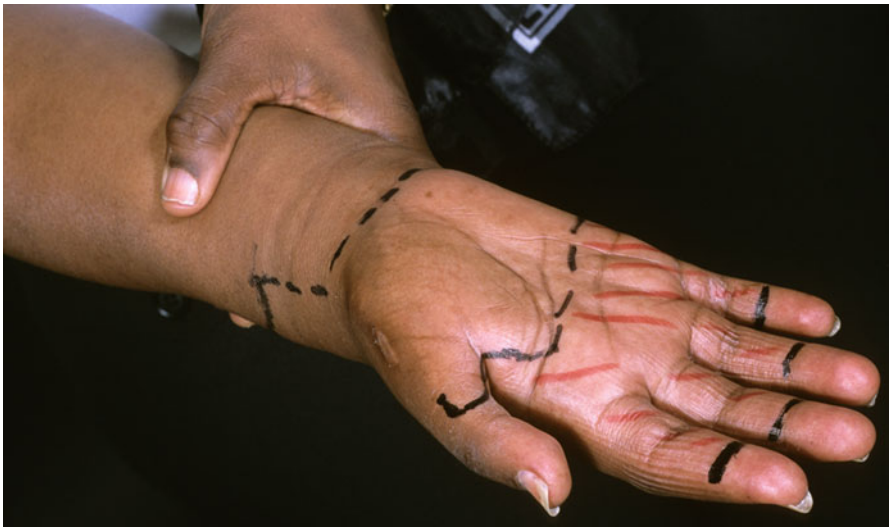


Fig. 2.32 Radiation induced thrombosis of the third part of subclavian artery. A 38 year old woman developed severe pain, swelling and paralysis 6 weeks after completing a course of radiotherapy for breast cancer. She lost all sensation in the tips of her fingers. She could recognise light touch but could not localise it in the palm of the hand. Sensation was normal proximal to the wrist crease. There was no recovery of skeletal muscle or of smooth muscle function in her forearm and hand

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Chapter 3
Regeneration and Recovery of Function

> But the journey of the axon tip to an end organ is only the most dramatic of the phases in the process of regeneration, and its arrival is alone no guarantee of the return of useful function [26].

The fundamental cellular processes which underlie regeneration are similar in the laboratory, in the injuries of civilian practice and in the wounds of war, but they are modified by the violence of the injury, by the effects of injury on associated tissues and in particular by ischaemia and by delay before repair. It is important to remember the differences between the laboratory investigation, in which a controlled, precise and limited lesion is inflicted upon a nerve and the situation faced by the clinician presented with a patient with a massive wound involving the soft tissues, the skeleton, the vessels and sometimes by other injuries which threaten life and limb. The demonstration of regenerating axons across a lesion of a nerve inflicted in the laboratory does not necessarily translate to the recovery of function in the human. The phenomena of pain and recovery of sensation are rather poorly revealed by experiments upon small mammals or for that matter by barely justifiable experimentation upon primates.

Some general conclusions may be drawn from extensive clinical and laboratory studies [7].

- The concept of retrograde degeneration of the axon extending to the first internode applies only to the most benign lesions, that of crushing the nerve between the tips of a jeweller's forceps. The extent of longitudinal damage of the nerve is greater in a rupture caused by traction that it is in "tidy" transections caused by knife or glass and this effect worsens with increasing delay before repair. It is worse still when healing has been complicated by sepsis and worst of all in neglected ischaemia.
- Failure to repair main vessels and failure to ensure perfusion of tissues is profoundly deleterious to regeneration and rules out worthwhile recovery of function.

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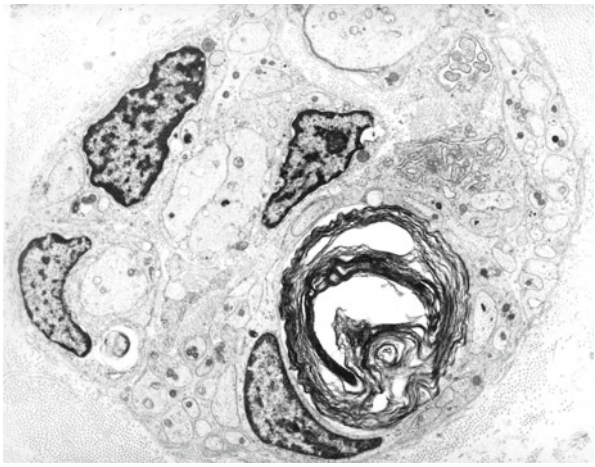

Fig. 3.1 Regeneration in the ulnar nerve at 3 weeks after section. Proximal stump. Retrograde degeneration of one large myelinated axon, proliferating Schwann cells and sprouting axons, forming a regenerating unit ×6,960. Electronmicroscopic (EM) study

- As time goes by the cellular response in both stumps changes from one friendly to regeneration to one less favourable. Dense collagenisation and a profusion of fibroblasts are characteristics of the distal stump in late cases.
- The normal architecture of the nerve is most closely restored to normal in a well executed primary suture.
- Regeneration through a graft falls away along its length and not solely at the suture lines.
- Delay before repair leads to increasing fibrosis and to shrinking of the distal segment so that it becomes impossible to ensure an accurate topographical match.
- Destruction of the target tissues, of muscle and skin, limits function even when there is strong regeneration.

3.1 The Response of the Nerve and Axon to Transection

Wallerian degeneration transforms the environment of the peripheral nerve from one which is actively hostile to the sprouting and growth of axons into one which actively supports that process [15, 16]. Within a few hours the transected axon seals off. The proximal end is transformed into a growth cone: “a swelling at the tip of the regenerating axon and possesses multiple needlelike extensions, filopodia and broader sheet like extensions, lamellopodia” [20]. The filopodia are rich in actin, and they may extend or retract within a matter of minutes. The axon forms new branches or sprouts: collateral sprouts arise from nodes of Ranvier at levels at which the axons are still intact; terminal sprouts arise from the tip of the surviving axon. The axon response is followed, within a few days, by a dramatic increase in the numbers of supporting cells. The influx of haematogenous macrophages into the endoneurium is accompanied by intense mitotic activity within the Schwann cells and the fibroblasts so that the number of nuclei increases by as much as six times or more (Fig. 3.1).

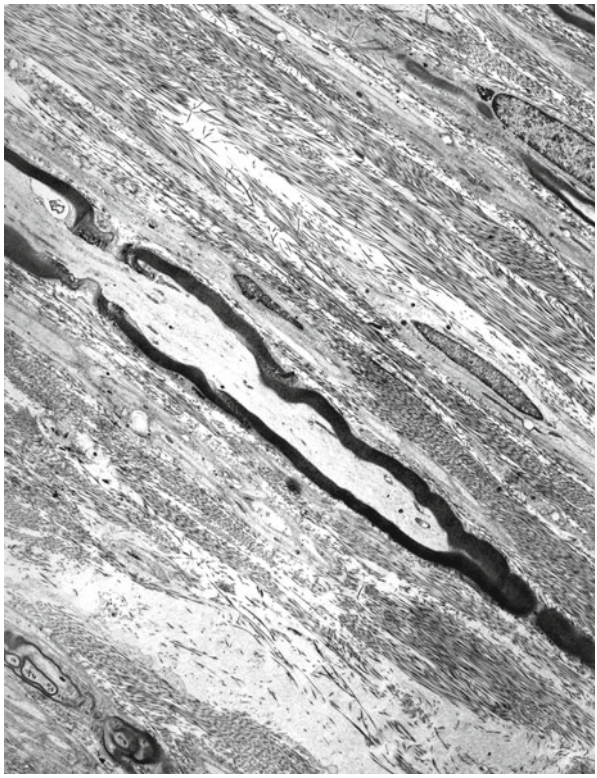

Fig. 3.2 The proximal stump of the median nerve 3 months after rupture of the nerve and of the axillary artery, which was not repaired. 2 cm proximal to the rupture. One myelinated fibre surviving embedded within dense collagen. There is very scanty evidence of regeneration ×3,400 (EM)

3.1.1 The Schwann Cells

The Schwann cells promote, sustain and guide regenerating axons. Regeneration in the peripheral nervous system is possible only because of the interaction between the Schwann cell and the axon. The proliferating Schwann cells express phenotypes which are specific to myelinated and non myelinated axons and also to motor and sensory axons [15, 16]. The proliferation, indeed the survival, of Schwann cells is modified by the severity of the injury to the distal trunk. Examination of biopsies taken during late repairs, especially when the injury was complicated by arterial injury or by sepsis, often show remarkably few Schwann cells. Instead it seems that the cellular population is largely composed of fibroblasts, surrounded by much collagen. Myelin fragments are detectable in such cases many months after the injury (Fig. 3.2).

3.1.2 The Axon

The axon prouts form clusters, *the regenerating units*, which are surrounded by the cytoplasm of one Schwann cell and its basal lamina. Sprouts within one regenerating unit represent the regenerative effort of one neurone and its axon (Fig. 3.3). The

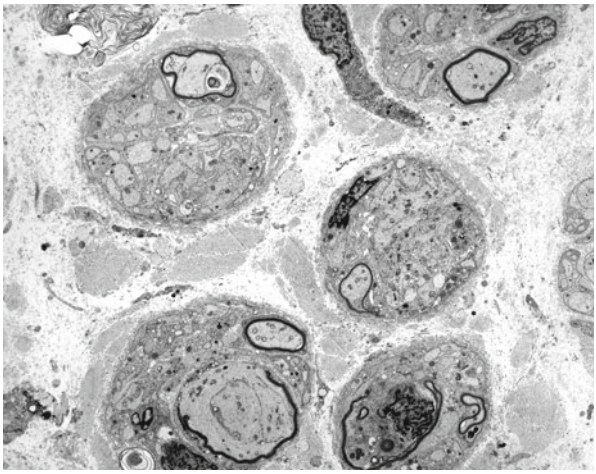

Fig. 3.3 Regenerating units. Proximal stump of ulnar nerve at 3 weeks after section. Numerous axon sprouts, Schwann cell processes, commencing remyelination with some retrograde regeneration ×2,784 (EM)

proximal segments of regenerating axons become ensheathed by Schwann cells. Myelination is determined by the axon. The regenerating sprouts of unmyelinated fibres remain so. The ensheathment of the growing axon is brought about by complex interactions involving the laminin component of the basal lamina and receptors such as the integrins in the plasma membrane of the Schwann cell and also between the adjacent plasma membranes of the axon and the Schwann cell [15, 16].

3.1.3 Reorganisation into "Mini Fascicles"

Even after an ideal laboratory repair, immediate suture after clean transection, regenerating nerve fibres burst out from the perineurium at the suture line and form mini fascicles in the epineurium. This pattern is usual in neuromas, in many grafts in small mammals and in failed grafts coming to revision in the human. The neuroma is a common example of the fate of the regenerating axons which are unable to form any connections with original targets. It is an expression of the vitality of the neurones producing new axons. After limb amputations it is common to find neuromas of the main nerves 3 cm or more in length, which contain abundant myelinated nerve fibres rather chaotically organised into mini fascicles (Fig. 3.4). Perhaps the process of compartmentation into mini fascicles should be seen as an aberrant or incomplete form of regeneration?

3.1.4 Guidance and Selection

In a carefully performed crush by jeweller's forceps only the axon is interrupted and growth cones elongate along former Schwann tubes. Unfortunately this most

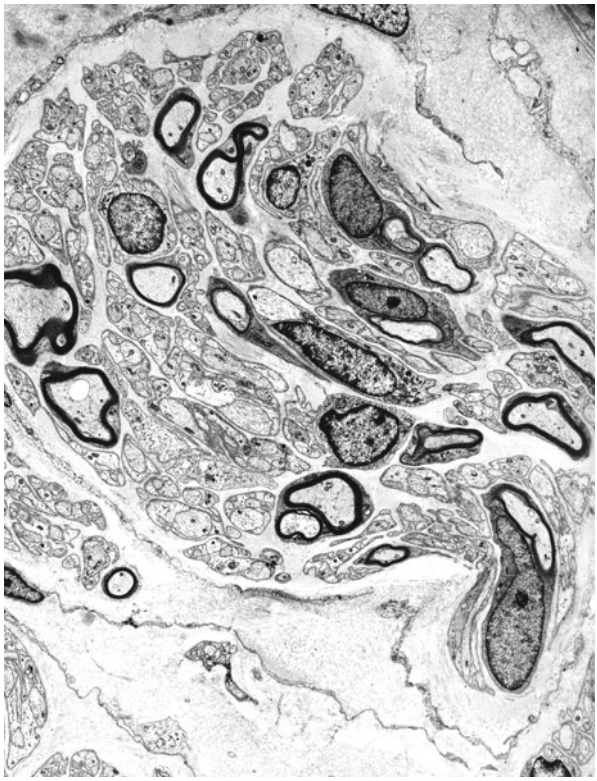

Fig. 3.4 Compartmentation or mini fascicle formation in the proximal stump of the common peroneal nerve ruptured 9 weeks previously in an 8 year old boy. Myelinated and unmyelinated fibres with Schwann cells surrounded by a perineurium composed of fibroblasts and their processes ×3,400 (EM)

favourable situation is rarely encountered in clinical practice. Other mechanisms become important in the control of the exuberant but chaotic regeneration which occurs after nerve repair. If the growth cone encounters non neural tissue, axonal elongation and myelination still occurs. The neuroma is one obvious example. Lundborg [20] distinguishes between *trophic* and *tropic* influences, between those which support and maintain the neurones during development and after injury, and those which act to guide regenerating axons. Some neurotrophins, including NGF, exert a *tropic* influence in addition to their main *trophic* action. Experiments were undertaken in which axons growing from the proximal stump were "offered" a choice between nerve, tendon and granulation tissue as distal targets. Nerves almost exclusively grew towards nerves. An even further degree of specificity may lead motor axons to motor endings and sensory axons to sensory endings. Redundant axon sprouts atrophy [10].

There are limits to neurotropism: in experimental repairs where the stumps were deliberately malaligned axonal dispersion was determined by fascicular size [15], not by fascicular identity. Clinicians must strive for accurate topographical alignment during repair whether by suture or by graft, and must accept the limitations of neurotropism exerted by a cutaneous nerve graft.

3.1.5 Maturation

Initially, there are more axonal sprouts than there were axons in the intact nerve, but only those that establish connections with end organs survive. As connections are established and the regenerating nerve matures, the original profusion of Schwann cells gives way to the more orderly arrangement characteristic of the healthy nerve. Studies of mature laboratory repairs of the sciatic nerve of the rat [6] showed that the numbers of MNF in the distal trunk were reduced by about 25 % in the primary sutures, by 33 % in the secondary sutures and primary grafts and by 40 % in the delayed and predegenerate grafts. These differences corresponded to such functional measures of outcome as power and weight of the reinnervated muscles.

The production of nerve growth factors in the distal nerve by endoneurial fibroblasts, macrophages, Schwann cells and by muscle and skin provide an environment friendly to regenerating axons. When there is a lesion in continuity or when healthy nerve end has been opposed to healthy nerve end, the elongating axon enters the environment most favourable to it: the denervated distal stump. Early and accurate suture of a divided nerve is followed by reconstitution of the trunk which is closest to normal. On two occasions the suture lines of the median and ulnar nerves 12 months after reattachment of the amputated hand were inspected during operations of tenolysis. The perineurium surrounding the bundles had reformed, there was only slight thickening of the epineurium and there was no visible neuroma. One indication about the accuracy of repairs completed within 3 or 4 h of the transection is the temporary restoration of conduction; the appropriate muscular response is evoked by stimulating the nerve trunk proximal to the repair.

After even the most careful primary suture of a cleanly divided nerve it seems that there is:

- a diminution in the number of nerve fibres which have made successful reconnection with their target tissues;
- reduction in the calibre of those nerve fibres;
- shortening of the internode and
- slowing of conduction.

These facts should not permit any sense of therapeutic nihilism: the best chance for the restoration of useful function rests on following the fundamental principles in the treatment of wounds by prevention of sepsis, restoration of flow through an injured axial artery, restoration of perfusion of the tissues, stabilisation of any fracture and providing full thickness skin cover over the repair. The nerve itself should be repaired as soon as reasonably possible. Whilst there may be a limit to technique much more needs to be done to improve the organisation of the handling of severe nerve injuries.

3.1.6 The Rate of Regeneration

The rate of peripheral outgrowth of regenerating fibres is reckoned at about 1 mm a day in the human adult: that is about the rate of slow axoplasmic transport. The rate

is substantially faster in children. It is almost certainly faster after primary than after secondary repair and is faster proximally than distally. A rate of 3 mm a day after suture, or even after graft of ruptures the brachial plexus or high sciatic nerve *performed within* 24 h of injury is common. Why not? Such wounds are nearer to the nerve cell than are more distal injuries [6, 8].

3.2 Regeneration of End-Organs

Regeneration in the deep afferent pathway from the muscle spindle and the Golgi tendon receptor is generally good after crush lesions (axonotmesis) although there is some reduction in the number of afferent endings [4, 24]. Regeneration after repair of a divided nerve is much less orderly. A muscle spindle may become reinnervated by afferents normally destined for the Golgi tendon organ. Many tendon organs remain uninnervated and the regenerated endings are frequently abnormal in appearance. Scott identified three deleterious factors:

- the effects of denervation on the afferent axon;
- the consequences of reinnervation by inappropriate axons, and
- the effects caused by the reorganisation of the motor units after repair causing significant alterations in the mechanical input to an individual tendon organ.

Muscles usually exhibit impaired coordination and reduce stamina after nerve repair. Some reasons for this were defined by Fullerton et al. [14] who found: “a selective failure of regeneration of the largest diameter fibres…. it seems more likely that the failure of recovery of fine movements is due to the fact that the proprioceptive pathway involving 1α, 1β and group 2 fibres on the afferent side and the process of α and γ coactivation on the efferent side, is lost”.

However there is undoubtedly considerable adaptation of the central receptor and effector mechanisms after nerve transfer (Fig. 3.5). Independent flexion of the elbow without associated activity in the flexor muscles of the forearm is usual by 24 months after transfer from the ulnar nerve to the nerve to biceps. Reinnervation of the muscle spindle and with that reorganisation and remapping of the somatosensory cortex has been confirmed after transfer of intercostal nerves to the musculocutaneous nerve in adult patients with severe lesions of the brachial plexus [21, 22].

3.2.1 The Cutaneous Sensory Receptors

The cutaneous sensory receptors similarly undergo a slow degenerative change after denervation. After 3 years they may disappear. Reinnervation tends to reverse these changes, though the longer the period of denervation has been, the less complete will be the regeneration.

The recovery of sensation within the hand after repair of severe birth lesions of the brachial plexus is remarkably good. Indeed, the sensory recovery is far better

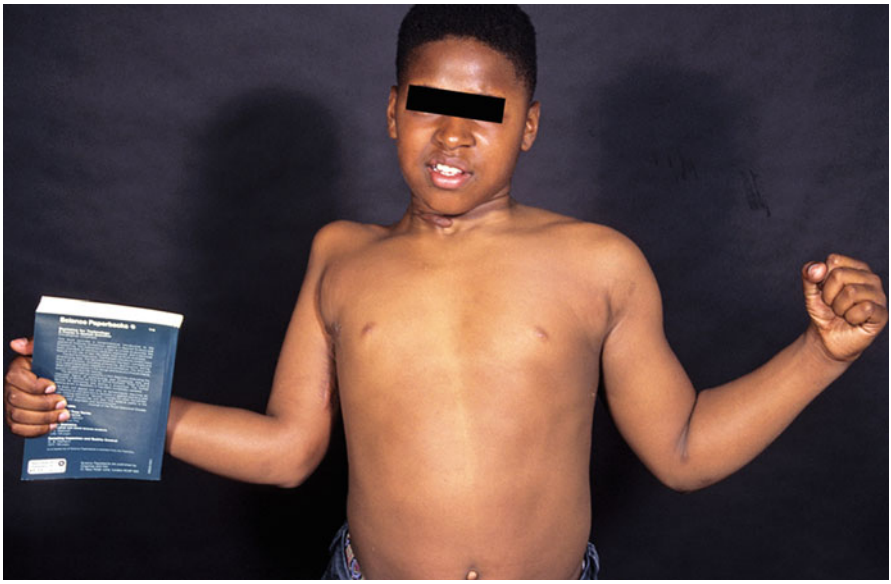

Fig. 3.5 A 13 year old boy. Right sided lesion. Avulsion of C5, C6 and C7. Repair at 2 months: accessory to suprascapular transfer, and one bundle of the ulnar nerve to the nerve to biceps and the nerve to the medial head of triceps; the medial cutaneous nerve of the forearm was transferred to the lateral root of the median nerve. Results at 18 months: full range of lateral rotation; abduction to 60°; power of elbow flexion MRC grade 4; and power of elbow extension, MRC Grade 3+. There was a full range of active flexion and extension without cocontraction

than that of skeletal muscle and sympathetic function. There was accurate localisation to the dermatomes of avulsed spinal nerves which had been reinnervated by intercostal nerves transferred from remote spinal segments [1]. The situation in the adult is very different. After transfer of the intercostal nerves to the lateral or medial cords it is usual to find that stimulation of the reinnervated skin is referred to the chest wall and only rarely to the skin of the hand [18].

3.3 Regeneration After Intradural Injury

There can be no doubt that the motor neurones in the anterior horn are capable of regenerating new axons through repair of the ventral root by suture, transfer or by direct reimplantation into the spinal cord. Nor can it, any longer, be a matter of, doubt that this regeneration is capable of restoring useful function within the upper limb [8, 12] (Fig. 3.6). Regeneration after reattachment of the dorsal root is blocked by proliferation of the asytrocytes which form a glial scar but it has been enhanced by combining olfactory ensheathing cells with re-attachment [19] (Fig. 3.7).

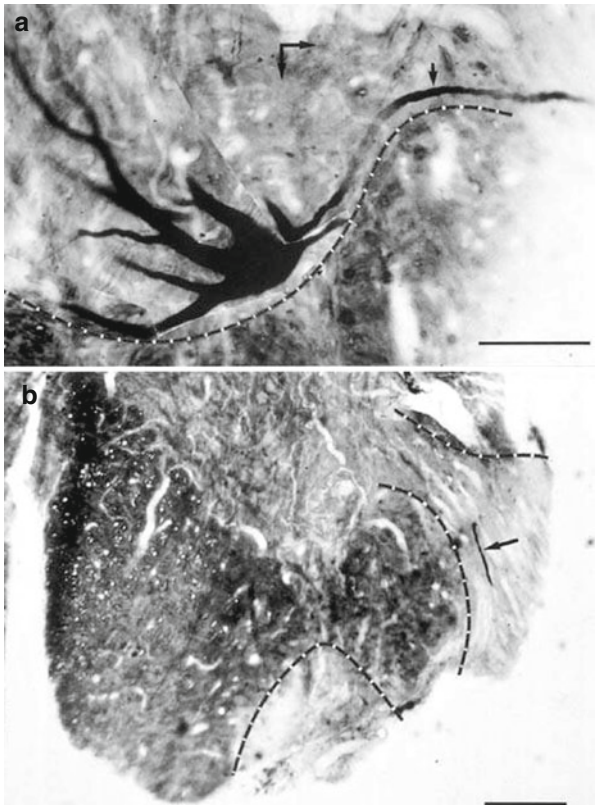

Fig. 3.6 A horse radish peroxidise (HRP) filled motor neurone regenerated into an avulsed and implanted ventral root. *Above*. Scale bar 20 μm. *Below*. Scale bar 200 μm

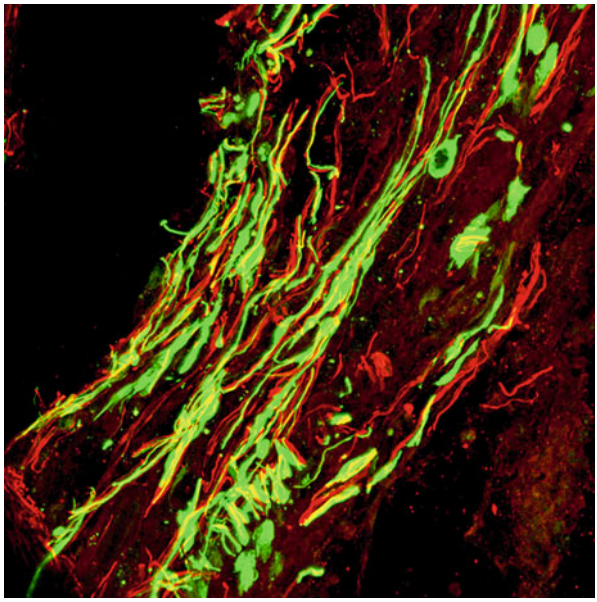

Fig. 3.7 Olfactory ensheathing cells (*green*) in the proximal part of the dorsal root. *Red* stained regenerating nerve fibres passing between the root and the spinal cord. Original magnification ×180

Avulsion or rupture of the roots has a devastating effect on cells within the dorsal and ventral horns. Motor neurones can be rescued by reconnection with the periphery an effect which may be enhanced by combining neurotrophins and neuroprotective agents with repair [5]. Hart et al. [17] showed that dorsal horn neurones were protected by repair of the nerve within 24 h combined with the administration of neuroprotective agents. It is difficult to explain the level of function that has been seen in some successful cases after reimplantation without some elements of recovery of the muscle afferent pathway. The restoration of the biceps tendon reflex and of the H reflex was demonstrated in a man who had undergone reimplantation of the ventral root of C6 10 years previously [13]. Perhaps this indicates a role for the myelinated afferent axons in the ventral root.

3.4 The Repair of Large Gaps

Direct apposition may be impossible because of the fixed retraction of stumps in delayed cases or because so much of the nerve has been destroyed by the injury. Methods of bridging this gap have been extensively studied for more than 100 years. They include:

- Autogenous grafts of cutaneous nerves
- Autogenous grafts of main nerves. These may be vascularised as a pedicle or as a free graft
- Homografts or allografts
- Non neural material.

Sanders [23] recognized the essential role of Schwann cells when he classed operations for the repair of large gaps in the peripheral nerves into two groups: "those which provide *orientated live Schwann cell columns, down which large numbers of fibres can grow and become mature, … and those which provide some form of artificial scaffolding down which… the new fibres and Schwann cells can grow in a regular manner*". The italics are original.

The advantages and the significant limitations of these methods are outlined in Chap. 5.

The chief advantage of grafting is that it overcomes tension, a nerve suture under tension develops a slow traction injury and ischaemia after removing protecting splints. However wide retraction of the stumps in closed traction lesion is usual and it is a simple matter to draw the nerves back to their normal position during operation performed within the first 1 or 2 days. Wherever possible the stumps of transected or ruptured nerves should be approximated at the first operation in cases where circumstances prevent primary repair. The shortage of available donor graft means that the sciatic nerve should be sutured wherever possible, even though the patient must endure a hip spica with gradual release for some weeks. Reasonable methods of diminishing the gap between nerve stumps include their careful

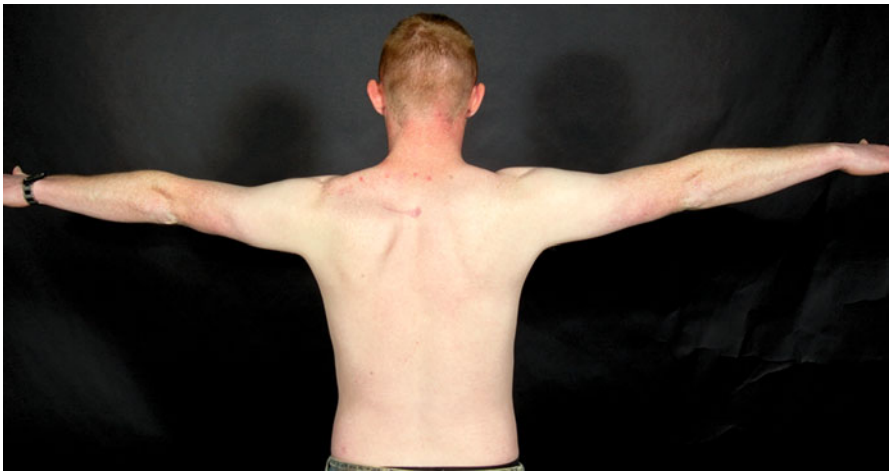

Fig. 3.8 A 25 year old man. Penetrating missile wound to the left shoulder, the exit wound close to the upper medial border of the scapula damaging the spinal accessory nerve at the level of spine of the scapula. There was cocontraction between trapezius, levator scapulae and the rhomboid muscles

mobilization and appropriate positioning of adjacent joints. The gap is always longer in delayed repairs.

3.5 Complications of Regeneration

3.5.1 Muscle Function

Weakness, reduced stamina, imbalance of muscle forces acting across joints and impaired coordination are usual after nerve repair. Cocontraction is a frequent complication of the disorderly regeneration across traction lesions. The shoulder girdle is particularly vulnerable (Fig. 3.8). The normal movement of joints is brought about by the smoothly coordinated and controlled activity in muscles and precise and delicate regulation by inhibition and facilitation of the motor neurones. The conversion of an antagonist to an agonist is the basis of musculotendinous transfer and it is common to see patients actively extending the knee or the ankle and toes as soon as the post operative splint is removed after hamstring to quadriceps transfer or anterior transfer of tibialis posterior. This control is damaged in lesions in continuity, especially of the brachial plexus; once again, the joints of the shoulder girdle are especially vulnerable. Reinnervated muscles usually fail to convert after muscle transfer irrespective of their power. Perhaps the defective reinnervation of the deep afferent pathways from the muscle spindles and the tendon receptors blinds muscles, which are, after all, sensory as well as effector organs.

3.5.2 Pain

Pain is, of course, a common feature of nerve injury particularly when the agent remains active but nerve regeneration itself may be associated with pain, particularly in adults.

Many patients experience flitting, deep, cramping pain in muscle, months after repair of the brachial plexus or high lesions of the sciatic nerve and shortly before obvious recovery of muscle function. This is a reassuring symptom, perhaps the only "good" form of neuropathic pain. The close relation to return of muscle activity suggests some reintegration of the deep afferent pathway.

It is usual, after repair of a nerve with a large cutaneous component, for the patient to experience over reaction. Such normal stimuli as cool or warm or pin prick are perceived as unpleasant cold or heat or stabbing. This "over reaction" generally improves over a period of 2–5 years but it may persist and prove disabling. Cold affects healthy nerves: it affects even more severely those in which the number of functioning axons is reduced, so, conditions of cold which would not seriously affect healthy nerves are liable badly to affect damaged nerves in which regeneration has been imperfect. Both motor and sensory function are affected. The cold affects not only nerve function; it affects too the skin and deep tissues of the affected limb, which is quick to cool and slow to warm. Sensitivity to cold may prevent return to work in the cold room of a butcher or fishmonger, or to outdoor work with machinery, even when recovery after repair of a median nerve at the wrist has been otherwise good.

The exaggerated response to pin prick is an example of *hyperalgesia*, a heightened response to a stimulus which is normally painful. This is bad enough, but the perception of stimuli which are normally not painful as painful ones is even worse. Light touch is painful: warmth often felt as burning, coolness as a painful icy sensation. This is *allodynia*, a most important symptom which usually indicates impending or actual damage to the nerve. It is uncommon in the later stages of regeneration but it is crippling when it occurs. Some of the worse examples are seen after repair of the tibial nerve; patients feels as if they are walking on hot coals or broken glass and they may be forced to request amputation.

It is important to explain to the patient what is going on, that there are reasons for these bizarre phenomena. It is as well for the clinician to be aware of these reasons:

- The density of innervation in the skin is reduced.
- The regenerated fibres are smaller in calibre, the intermodal length is reduced and conduction is slowed.

There are changes in the phenotype of the cell body and axon of the sensory neurone

- There are changes in the expression of the volted gated ion channels in the axon membrane and there may be a relative increase in the sodium channels associated with nociception. Indeed one form of the channel NAV 1.9 is not expressed

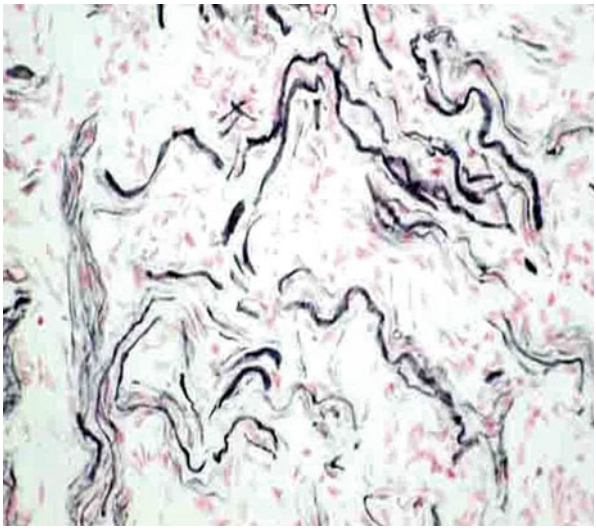

Fig. 3.9 NGF fibres in a human painful neuroma, ×40

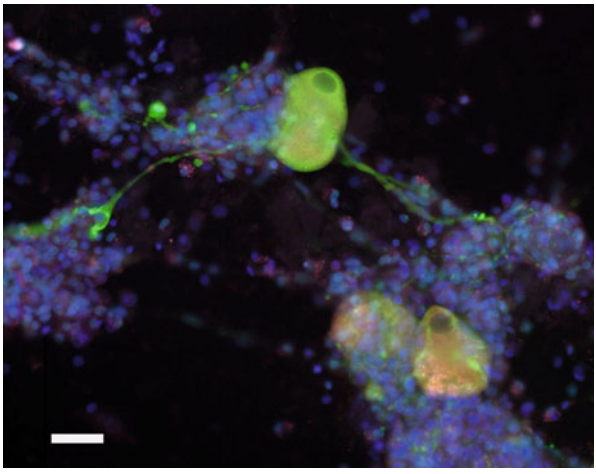

Fig. 3.10 Cultured human DRG neurones immunostained for Gap 43 (*green*) and for the vanilloid receptor TRPV1 (*red*), the nuclei of satellite cells are stained *blue*. Bar = 25 μm

in infant nerves [25], a possible explanation for the apparent absence of pain behaviour in BLBP (Fig. 3.9).

- Cultured neurones from the dorsal root ganglia of human spinal nerves avulsed from the spinal cord in severe traction lesion to the brachial plexus provide an important experimental model. There are, after injury, radical changes in the morphology of the cell body and in the expression of receptors. Those associated with nociception may increase [2, 3] (Fig. 3.10).
- Changes in the phenotype of regenerating axons provides an explanation for the pain experienced by some patients who are diagnosed, incorrectly, as suffering from so-called "complex regional pain syndrome Type 1" (Figs. 3.11 and 3.12). This diagnosis should not be considered unless or until focal axonopathy and subsequent regeneration has been excluded.

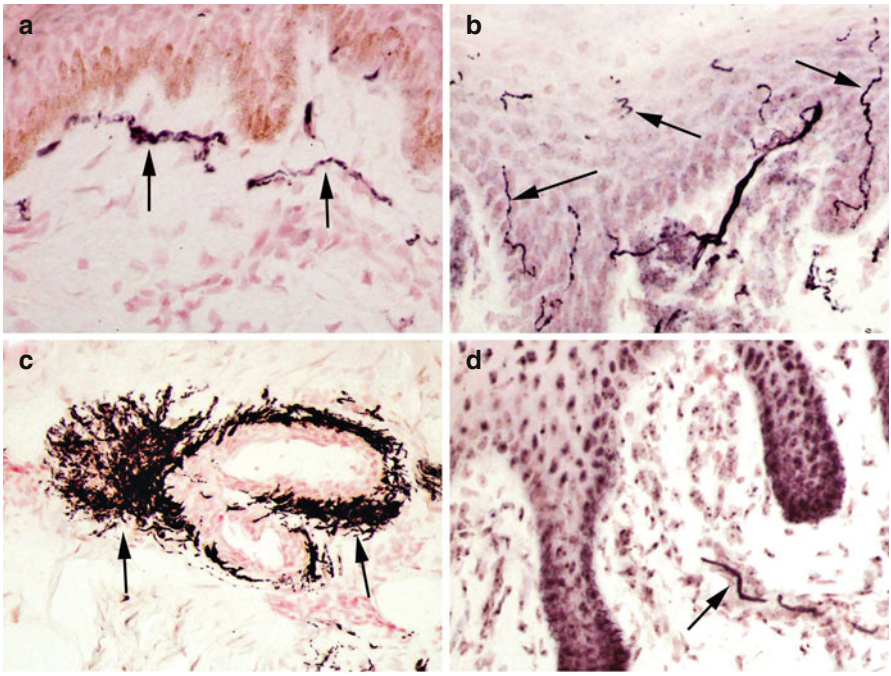

Fig. 3.11 A 26 year old woman developed back pain radiating to the left foot and ankle after a sprain whilst training for a run. Over a few weeks she developed allodynia to touch, changes in colour and sweating and an abnormal foot posture. Clinical examination and quantitative sensory testing revealed a mechanical allodynia, elevated cold threshold and reduced heat pain threshold over the affected limb. Below knee amputation was performed. Examples of various nerve markers in the skin of the calf close to the ankle. (**a**) Preserved sub-epithelial nerve fibres stained with the marker PGP9.5 (*arrowed*) ×40. (**b**) Unusually dense PGP9.5 fibres around blood vessels in the skin (*arrowed*) ×40. (**c**) Increased intra-epithelial TRPV1 (heat receptor) fibres (*arrowed*) ×40. (**d**) A few sub-epithelial TRPM8 (cool receptor) fibres, (*arrowed*) ×40

3.6 Recovery of Function After Nerve Repair

The quality of recovery after neurotmesis and repair depends chiefly on the number of axons reaching their correct targets and on the later development and myelination of those axons. Factors influencing such re-innervation are:

- the promptitude of repair;
- the quality and viability of the opposed nerve ends;
- The quality and accuracy of fascicular matching;
- the degree of damage to the nerve ends during suture;
- the length of gap after resection;
- the number of channels provided by the interposed graft for the regenerating columns;

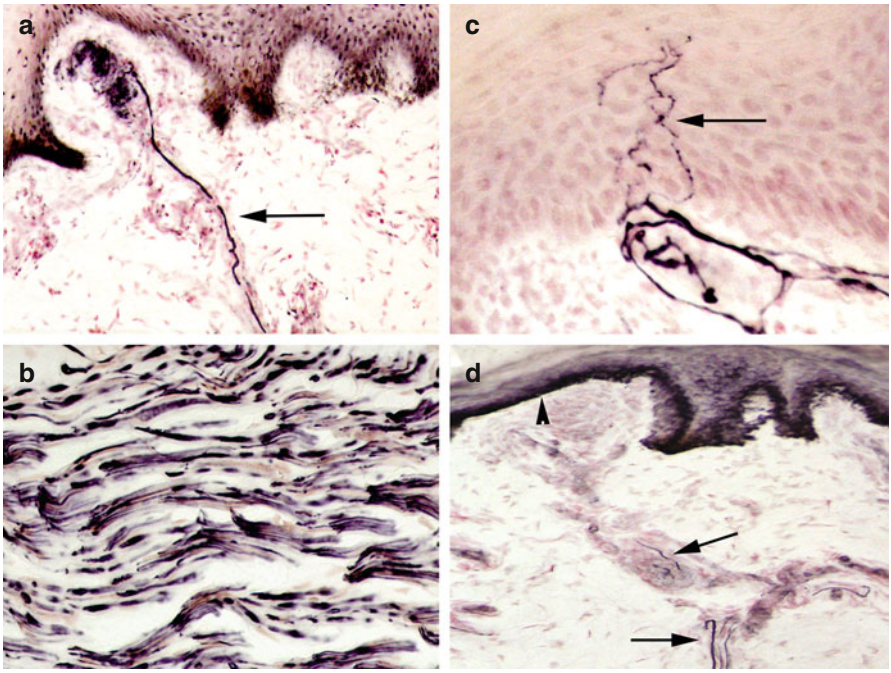

Fig. 3.12 Further examples of skin staining with various nerve markers in the patient illustrated in Fig. 4.26. (**a**) Sub-epithelial TRPM8 (cool and menthol receptor) fibres (*arrowed*) ×40. (**b**) PGP9.5 (structural marker) staining of injured nerve. (**c**) Increased intra-epithelial (*arrowed*) and sub-epithelial TRPV1 (heat and capcaisin receptor) fibres ×40. (**d**) Basal keratinocytes (*arrow head*) and sub-epithelial NGF fibres (*arrowed*) ×40

- the extent of fibroblastic infiltration of the stumps and of the interposed grafts;
- the state of the target muscle and skin;
- the condition of the tissues at the site of repair.

It is usual to find numerous axons, some of them myelinated, in the tissues bridging nerve stumps in the human. Such findings illustrate the difference between regeneration and recovery (Figs. 3.13 and 3.14).

3.6.1 Duration of Study: Records

Recovery of nerves after repair is prolonged particularly so for sensation.

Case report: Primary repair of median and ulnar nerves was performed in a 22 year old staff nurse. Recovery was quite good, but marred by cold sensitivity, so that her result was considered only fair at 3 years after operation. She came to see

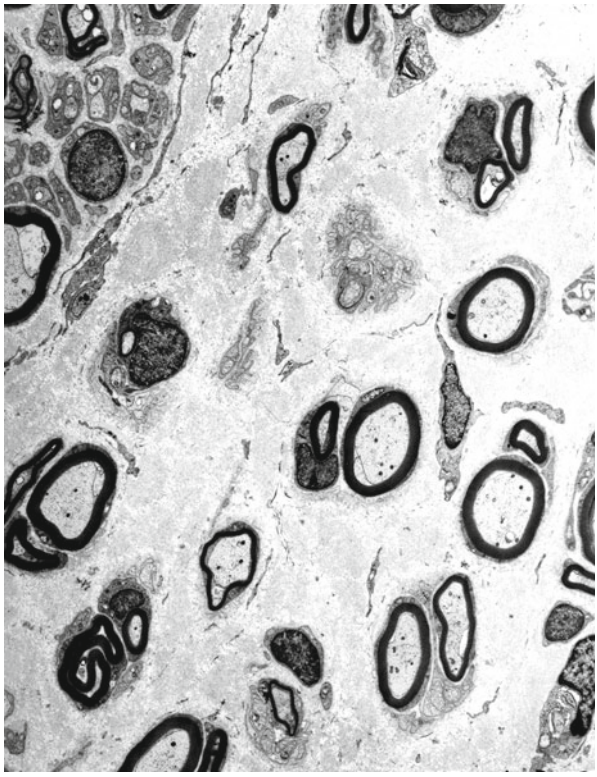

Fig. 3.13 Useless regeneration. Failure of recovery in a median nerve sutured 3 years previously. Distal to the suture line. There was no recovery of function in spite of regeneration of myelinated fibres ×4,300 (EM)

us 7 years after operation to report a striking improvement over the course of the previous few months. Over-sensitivity had disappeared, the final result for the ulnar nerve was considered excellent – and it was very good for the median. Her case is by no means unusual.

Adequate assessment of outcome after severe injuries to the brachial and lumbosacral plexus in the adult requires at least 5 years of study. In some patients even this is inadequate. The length of the follow up must be longer still in children with birth lesions of the brachial plexus (BLBP) or in children with severe injuries to the main nerves of the lower limb because of the ever present risk of secondary deformity from muscular imbalance or impaired growth and because of the high incidence of posterior dislocation at the shoulder in BLBP.

3.6.2 Grading of Results

In some nerves, such as the radial muscular function is a good deal more important than recovery of cutaneous sensation and for these little significance is attributed to

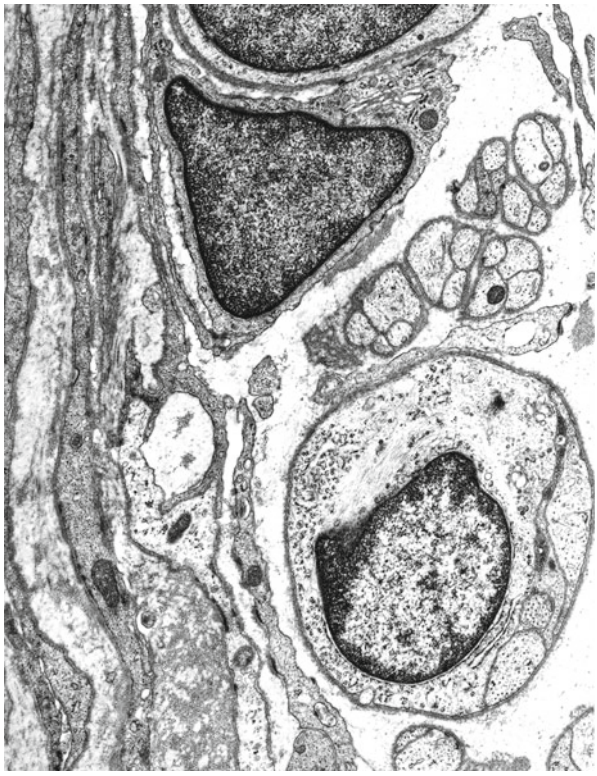

Fig. 3.14 Useless regeneration. Failure of recovery in a median nerve sutured 3 years previously. Biopsy of the skin of the index finger. Schwann cell processes and some unmyelinated axons ×18,000 (EM)

return of cutaneous sensation *except when recovery is complicated by significant pain when the result is considered poor*. Conversely sensibility is the most important function of the median and tibial nerves. The requirements for a good result are higher in the lower limb. Recovery of power to MRC grade 2 in the small muscles of the hand is sufficient to overcome much paralytic deformity. Such recovery in the muscles of the leg is of no functional worth.

Systems have been developed which record the progress after nerve injury, associated injuries and their consequences, and after "reconstruction" operations [7]. Information is collected about the effects on daily life, on work or training or study. The evolution of pain, its response to treatment or to recovery is recorded. The progress for the nerve is recorded sequentially for motor, sensory, and sympathetic function. Suitably modified forms are used for war wounds, for injuries to the brachial plexus in the adult and the infant, to the shoulder girdle and gleno-humeral joint, and to the lower limb (Fig. 3.15). It is particularly important to be on the look out for fixed deformity, which is a serious nuisance in the hand but much worse in the ankle and foot.

All systems of measurement have their defects and it seems the more closely one looks at the result of a nerve repair the more defects are revealed.

Part of a study? Please indicate (examples: Radial, CPN, ...) :-

Headley Court patient? ☐

Compound Nerve

NOTE - See also: IATROPATHIC ☐ RECONSTRUCTION ☐ BP ☐

Summary

Tinel static.....☐
Tinel advancing ☐
Tinel consistent ☐
Tinel misleading☐
Significant pain? (Y / N): .....
Onset pain date: ..../..../.....
Onset recovery: ..../..../.....
Plateau recovery: ..../..../.....

Tidy ..... ☐
Untidy ..... ☐
Traction ... ☐
Arterial ..... ☐
Delay ..... ☐
Repair ..... ☐
Neurolysis . ☐
Non-op ..... ☐
Compressn.☐
Compartmt. ☐
RESULT: G☐ F☐ P☐

Predominantly conduction block ☐	Axonotmesis ☐	Neurotmesis ☐	Mixture of all three ☐

Surname ..... Hospital No. .....

Forename ..... Sex M/F .....

Date of Birth ...../...../.....

Dominance ..... Side of Injury .....

Occupation (original) .....

Work at first attendance Y/N .....

Occupation (final) .....

significant associated disease .....

Pain, at first attendance ..... at final attendance .....

Pain diagnosis: Date of injury or diagnosis ..../..../.....

Referring doctor: Date of referral ..../..../.....

Date seen (PNI) ..../..../..... Date operation (PNI) ..../..../.....

Interval (in weeks) from incident to PNI operation:

N.P.I.

Rehabilitation, O.P. I.P

Splinting, before operation ..... after operation .....

Sensory retraining: Y / N Helpful?: Y / N

Desensitisation: Y / N Helpful?: Y / N

RNOH: Y / N Elsewhere: Y / N

Comment on rehabilitation process .....

.....

Compound Nerve.doc *(modified: 30/06/2008 15:00:37)* Page 1 of 6

Fig. 3.15 The document used for recording data in patients with compound nerve injury

Energy transfer

Massive ☐
High ☐
Low ☐

Cause

RTA:

Motorcycle ☐
Vehicle ☐
Cycle ☐
Pedestrian ☐

Industrial ☐
Agriculture ☐
Sport ☐

PMW ☐
Knife / glass ☐
Fall ☐
Other assault ☐
Other .....

Tidy ☐ Untidy ☐ .....

Associated			Treatment referrer	Treatment PNI
Head				
Spine				
Chest				
Abdomen				
Pelvis	a	#		
	b	Visceral		
At level of nerve injury	Fracture longbone			
	Dislocation or fracture disclocation			
	Arterial			
	Venous			
	Muscle loss			
	Skin loss			

Comments .....

.....

.....

Fig. 3.15 (continued)

Muscle fibrosis (or loss of muscle)

None ☐
Mild ☐
Moderate ☐
Severe ☐

comment .....

Notes for table completion and interpretation

mild	MRC 5. Correctible
moderate	MRC 3-4. Range greater than ½
severe	Range less than ½. No useful function. Rigid contracture

Joint contracture

None ☐
Mild ☐
Moderate ☐
Severe ☐

comment .....

Notes for table completion and interpretation

mild	Loss of less than ¼ range
moderate	Loss of ¼ to ½ range
severe	Loss of more than ½ range

Skin loss

None ☐
Mild ☐
Moderate ☐
Severe ☐

comment (e.g. skin graft, flap, z plasty) .....
.....

Notes for table completion and interpretation

mild	Cosmetic
moderate	Loss of up to ¼ range
severe	Tethering to bone and muscle Loss of more than ¼ range

PNI Operations

Not done – recovery ☐
Not done – too late ☐
Not done – too severe ☐

Reconstruction operation

- to correct deformity ☐
- to restore function ☐

Nerve operation method

NAP ☐
Graft ☐
Suture ☐
Transfer ☐
Neurolysis ☐
Irreparable ☐
Other ☐ .....

Comments .....
.....

Fig. 3.15 (continued)

Nerve and level

- Circ. ☐
- SS. ☐
- M C ☐
- Radial:
  - High: Above Triceps ☐
  - Inter: Above PIN ☐
  - Low: PI N ☐
- Median:
  - High: Above superficial flexor branch ☐
  - Inter: Between superficial flexor branch and wrist crease ☐
  - Low: Wrist and hand ☐
- Ulnar:
  - High: Above FCU ☐
  - Inter: Above wrist ☐
  - Low: Wrist and hand ☐
- Lumbosacral plexus ☐
- Femoral, above Psoas ☐ below Psoas ☐
- Superior gluteal ☐
- Inferior gluteal ☐
- Obturator ☐
- Tibial:
  - Buttock ☐
  - Thigh ☐
  - Knee ☐
  - Leg ☐
  - Ankle & foot ☐
- CPN:
  - Buttock ☐
  - Thigh ☐
  - Knee ☐
  - Leg ☐
  - Ankle & foot ☐

Lesion

- Lesion In Continuity ☐
- Partial transection ☐
- Transection ☐
- Nerve in fracture or joint ☐
- Nerve avulsed from muscle ☐

Comment (re lesion).....

.....

Fig. 3.15 (continued)

Recovery

Date		Good	Fair	Poor
..... /.... /.....	Motor			
	Sensory			
	Sympathetic			
..... /.... /.....	Motor			
	Sensory			
	Sympathetic			
..... /.... /.....	Motor			
	Sensory			
	Sympathetic			
..... /.... /.....	Motor			
	Sensory			
	Sympathetic			
..... /.... /.....	Motor			
	Sensory			
	Sympathetic			
..... /.... /.....	Motor			
	Sensory			
	Sympathetic			

Response of pain to nerve operation .....

.....

.....

.....

Comment on recovery .....

.....

.....

Now attach printed sheets Surgical Disorders & JBJS about recovery for:

Radial	☐
Median	☐
Ulnar	☐
Femoral	☐
Tibial	☐
CPN	☐
Musculocutaneous	☐
Goldie & Coates	☐

where their nerves have been injured, and, sheet, shoulder, for Circ. & SS.

Fig. 3.15 (continued)

Shoulder form

Date	Mallet	Narakas points	Myometry (as % of opposite)
pre-op			
final			

Range of Motion (by date)

NOTE: Insert normal in brackets at pre-op and final	Date	Forward flexion		Abduction		Lateral Rotation		Medial Rotation		Inferior SH Angle		Post SHA
		Passive	Active	Passive	Active	Passive	Active	Passive	Active	Passive	Active	Passive
	pre-op											
	final											

Comments .....

.....

.....

Fig. 3.15 (continued)

Table 3.1 Grading of recovery of the common peroneal nerve

	Motor recovery	Sensory recovery
Good	Dorsiflexion and eversion ≥ MRC 4	No spontaneous pain; no hypersensitivity
Fair	Dorsiflexion MRC 3+ to 4 or Eversion MRC 3+ to 4 or Dorsiflexion and eversion MRC 2–3	No spontaneous pain, no, or only mild, hypersensitivity
Poor	Less than the above	Spontaneous pain or significant sensitivity

Table 3.2 Grading of recovery for the tibial nerve: all levels

Good	Heel flexors MRC 4 or better. Tibialis posterior, flexor digitorum longus, and flexor hallucis longus MRC 3. Return of sweating. No fixed deformity, no trophic disturbance. Normal Shoes	Accurate localisation, pain and temperature sense, no hypersensitivity; no spontaneous pain
Fair	Heel flexors MRC 3+ or better. No useful recovery in long flexor muscles. Incomplete return of sweating. No serious trophic disturbance, no fixed deformity. Normal shoes with or without foot drop splint for CNS palsy	Protective sensation, hypersensitivity not interfering with daily activities
Poor	No useful motor recovery; and/or significant fixed deformity; and/or trophic ulceration	No return of sensation; and/or significant hypersensitivity interfering with daily activities

3.7 Factors Governing Prognosis

The prognosis for recovery after nerve repair is determined by a number of factors. Two of these are particularly important. First is the *severity of injury,* and next is *delay* before the repair. Taking all in all, these two factors are, by far and away, the most important. The quality of the first operation and the timing of repair are factors within the control of the surgeon.

3.7.1 Severity of Injury

Results of repairs are consistently better in the open tidy wounds from knife, scalpel or glass, than they are in the open untidy wound of compound fracture or penetrating missile. They are worse still in the ruptures of traction lesion. Worst of all is the effect of failure to repair main vessels and the failure to restore perfusion of the tissues. Useful recovery in function cannot be expected if the fundamental principles of treatment of a wound are neglected. Arteries must be repaired, the tissues of the limb must be perfused, the skeleton must be stabilised and repaired nerves and vessels must be covered by full thickness skin [9]. The results of repairs of the common peroneal and tibial nerves exemplify these facts (Tables 3.1, 3.2 and 3.3).

Table 3.3 319 repairs of the common peroneal and tibial nerves 1979–2006 in adults and children (%)

	Common peroneal nerve				Tibial nerve			
	Tidy wound	Untidy wound	Closed traction		Tidy wound	Untidy wound	Closed traction	
Good	29 (60.4)	18 (38.3)	40 (33.3)	87 (40.5)	17 (68)	17 (37)	6 (18.2)	40 (38.5)
Fair	12 (25)	15 (32)	28 (23.3)	55 (25.6)	6 (24)	20 (43.5)	16 (48.5)	42 (40.4)
Poor	7 (14.6)	14 (29.7)	52 (43.4)	73 (33.9)	2 (8)	9 (19.5)	11 (33.3)	22 (22.1)
Total	**48**	**47**	**120**	**215**	**25**	**46**	**33**	**104**

Table 3.4 Grading of results of median and ulnar nerves injured below the elbow (low)

Grade	Motor	Sensory	Equivalent on Seddon's grading [6, 7]
Excellent	Power MRC 5 No wasting or deformity No trophic changes	Function indistinguishable from normal hand. Good stereognosis, no hypersensitivity. 2PD[a] equivalent to uninjured digits	Good M5, S4
Good	Power MRC 4–5 Abolition of paralytic deformity Minimal pulp wasting	Accurate speedy localisation to digital segment. Can recognise texture or objects. Minor cold sensitivity and hypersensitivity. 2PD < 8 mm at tips of fingers	Good M5, S3+
Fair	MRC 3 or more Some sweating Pulp wasted	Accurate localisation to digit. Stereognosis slowed and sometimes inaccurate. 2PD > 8 mm. Cold sensitivity and hypersensitivity sufficient to interfere with some activities	Fair M3, S3
Poor	MRC 3 or less No sweating Trophic changes	No sensation or severe cold sensitivity and hypersensitivity	Bad M0, 1, S0, 1 or 2

2PD two point discrimination

3.7.2 Delay

Some reasons for the deleterious effects of delay have been outlined in Chap. 2. Repair of the median and ulnar nerves divided in "tidy" wounds in the wrist and forearm provides one useful example (Tables 3.4 and 3.5). The average delay in the delayed suture or graft group was 10 weeks. The incidence of arterial and tendon injury was much higher in the primary suture group. The effect of delay is particularly dramatic in the cases of supraclavicular lesions of the brachial plexus where it seems that every day counts [8] (Table 3.6). Results of repairs performed within 7 days were decisively better than later ones and this in spite of excluding repairs of the ventral root (usually by nerve transfer/see Sect. 5.4.3), a method which is, for technical reasons, generally possible only within a few days of injury. There is one

Table 3.5 Repair of 264 median and ulnar nerves in tidy wounds injured between the distal wrist crease and elbow in adults aged between 16 and 65 years: 1979–2004

	Repair Type (number of cases)			
Outcome	Primary repair	Delayed repair	Graft	Total
Excellent	13	2	2	**17**
Good	52	15	34	**101**
Fair	25	30	52	**107**
Poor	2	16	21	**39**
	92	**63**	**109**	**264**

Table 3.6 Results of repairs in 585 elements in 228 patients operated between 2000 and 2004 – by interval between injury and operation

		Results of repairs		Results (excluding ventral root repairs)		Average number of elements repaired in each patient	Average number of functions regained in each patient
Interval in days	Number of patients	Good/total	%	Good/total	%		
0–7	52	114/175	65.1	86/40	61	3.4	5.4
8–14	25	41/72	57	21/45	46.7	2.9	3.8
15–28	31	48/87	50.1	34/73	46.6	2.8	3.3
29–56	32	25/74	33.8	21/68	30.1	2.3	1.6
57–84	31	31/67	46.2	30/65	46.2	2.2	1.8
85–112	16	13/35	37.1	12/33	36.4	2.2	1.9
113–182	22	8/34	23.5	7/33	21.2	1.5	1.1
More than 182	19	12/41	29.3	11/39	28.2	2.2	1
	228	288/585	49.2	222/496	44.8		

1. The average numbers of repairs for each patient was 2.6
2. The average number of functions regained in each patient was 2.9; the total of functions regained was 658

exception to the general rule and it is an important one. Alone amongst the peripheral nerve it seems that the spinal accessory nerve is almost immune to the effects of delay before repair [11].

3.7.3 Other Factors in Prognosis

3.7.3.1 Age

Recovery of function after repair of a nerve in a child is, on the whole, better than in the adult, but it is not as good as has sometimes been assumed. This comes as no surprise if we take into consideration the increased vulnerability of the immature nervous system to axonotomy. Some of the most difficult problems in reconstruction follow failure of recovery of either of the divisions of the sciatic nerve in the growing child. The recovery of skeletal muscle and of sympathetic efferent function

Table 3.7 Recovery of sensation after repair of 129 digital nerves

	Adults		Children (aged 1 years or less)	
Grade	Repair within 48 h of injury	Repair 2 weeks or more after injury	Repair at varying intervals after injury	
Excellent	1	0	17	**18**
Good	33	9	8	**50**
Fair	24	11	2	**37**
Poor	16	8	0	**24**
	74	**28**	**27**	**129**

Table 3.8 Grading of outcome in lesions of the radial nerve

	For high lesions above nerves to triceps	For intermediate lesions, between medial head of triceps and brachio radialis	For low lesions, the posterior interosseous nerve
Good	Elbow extension ≥ MRC grade 4	Wrist extension ≥ MRC grade 4	Full independent extension of the digits ≥ MRC grade 4
	Wrist extension ≥ MRC grade 3	Finger and thumb extension ≥ MRC grade 3	
Fair	Elbow extension ≥ MRC grade 3	Wrist extension ≥ MRC grade 3	Extension of digits ≥ MRC grade 3
	Wrist extension MRC grade 2	Finger and thumb extension ≥ MRC grade 2	
Poor	Less than above	Less than above	Less than above

after injuries to the brachial or lumbosacral plexus in the growing child is generally inferior to that seen following urgent repair in adults. There is often severe shortening of the limb and atrophy of the hand or foot. On the other hand the recovery of cutaneous sensation in the infant or child is often remarkably good. The results after repair of digital nerves provides one example of the difference between the adult and the child (Table 3.7).

The changes in the peripheral nerves with aging (see Sect. 1.8) suggest that the older patient may be more likely to develop severe pain after injury to a nerve. Striking results have been seen after repair of the brachial plexus in patients aged over 65 years. The relief of pain and with it the improvement in the patient's mental well being, were remarkable [8]. It may be administratively convenient to ignore the elderly but they are, after all, still human beings and the risk of losing independence calls for a far more rational and vigorous approach than is often, too often, seen.

3.7.3.2 Level of Injury

This is undoubtedly important for the median, ulnar and radial nerves, a fact reflected in the grading of results in the radial nerve (Tables 3.8). It seems to be less important for the sciatic nerve and its divisions. Only rarely is extension of the digits regained after injuries to the brachial plexus, the posterior cord or the radial nerve in the axilla. Recovery of the small muscles of the hand is unusual after high repairs of the

median or ulnar nerves in the axilla. Recovery was seen in only 27 of 216 high repairs. Twenty of the 27 good results followed repair within 48 h of injury and 7 were in children [7].

There seems little doubt that repair of the recurrent motor branch of the median nerve at the wrist, of the posterior interosseous nerve and of the deep branch of the ulnar nerve is followed by a return of function which is scarcely ever seen after high repairs. On the other hand lesions of the distal branches of such cutaneous nerves as the sural, the medial cutaneous of forearm and the superficial radial nerve have deservedly gained an ill reputation. It is worth noting that urgent repair of lesions of C5 and C6 is often followed by recovery to a level far higher than that seen after repair of combined lesions of the circumflex, the suprascapular and the musculocutaneous nerves.

3.7.3.3 The Nerve

Whilst there is no such thing as a purely "motor" or "sensory" nerve in the peripheral nervous system, it appears that recovery after repair of nerves which have no cutaneous distribution is generally better than after repair of main nerves. The spinal accessory and the nerve to serratus anterior generally recover well after repair. The suprascapular fares better than the circumflex. The blood supply to the most proximal segment of the radial nerve and to the common peroneal nerve is rather poor, so that transection at this level may worsen ischaemic changes in the distal segment.

3.8 Conclusion

The clinician must always bear in mind that the sooner the distal segment is connected to the cell body and proximal segment the better the result will be. The quality of treatment of the wound at first operation and the timing of repair of the nerves are factors within the control of clinicians. The last 20 years has seen extensive laboratory work which confirms the view, long held by many clinicians, that the central nervous system suffers after interruption of a peripheral nerve. The rapidity and the severity of that response to violent proximal nerve injury is the over arching biological imperative which must guide action.

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Chapter 4
Clinical Aspects of Nerve Injury

In the acute injury the object of the clinician must be to recognise the fact of injury as soon as possible after the event, and later to go on to determine the nerve or nerves affected, the level or levels of injury and the extent and depth of the lesion or lesions.

4.1 The History: Characteristics of the Wound

The history is important: high injury, compound fracture and wounding, accidental, criminal, surgical or all three, are likely to mean that there has been a serious lesion. The use of a knife, often enough in the hand of a surgeon, is an indication that a nerve is likely to have been partly or completely severed. Advice from witnesses or emergency paramedical staff is always valuable. Potentially life or limb threatening injuries complicate closed traction lesion of the supraclavicular brachial plexus in at least 20 % of cases. Even more patients with injuries to the lumbo-sacral plexus are so threatened. The subclavian artery is ruptured in 10 % of complete lesions of the brachial plexus and in as many as 30 % of cases of violent traction injury of the infraclavicular portion of the brachial plexus. The incidence of arterial lesion is high after fracture dislocations of the shoulder and elbow, higher still after fracture dislocations of the knee. It is important always to search with diligence for occult injuries to the head, the spine, the chest, the abdomen and pelvis before embarking upon treatment of the nerve lesion, both at the first hospital but also after transfer to another unit (Fig. 4.1).

The site and nature of the wound or wounds must be observed. In closed injuries the presence of swelling and bruising may give some indication of severity. In all cases of limb injury the adequacy of perfusion as judged by the state of the pulses, by colour and by temperature must be observed. Indications of associated fracture must be sought (Fig. 4.2). It is useful to distinguish between the tidy

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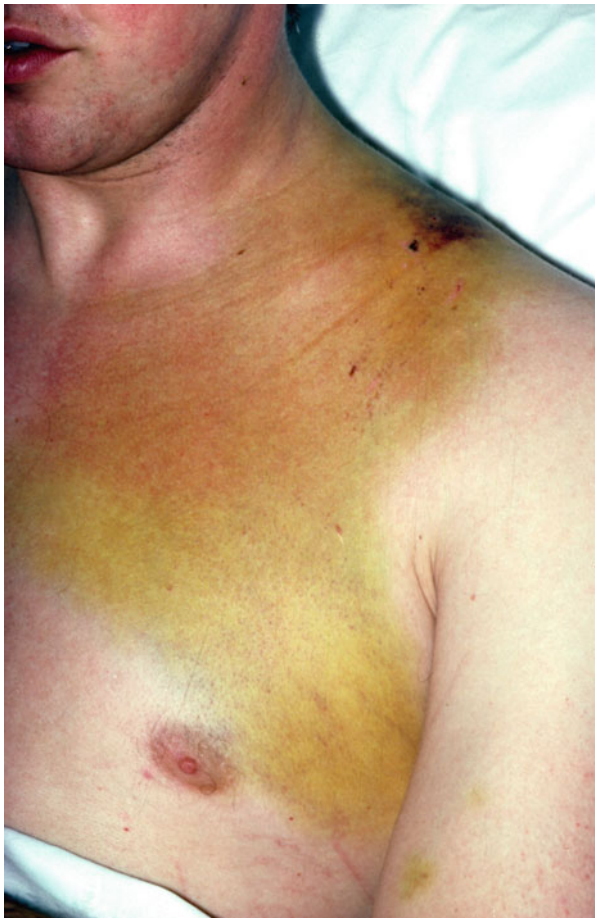

Fig. 4.1 This motor cyclist struck his shoulder against a traffic bollard. There is bruising and swelling of the left shoulder, neck and upper arm. Total avulsion

wound caused by a knife and the untidy wound of open fracture. Soft tissue damage is worse in the latter; nerves and vessels are often subjected to traction. In penetrating missile wounds it is important to distinguish between the shot gun, the hand gun or rifle and the fragment (Fig. 4.3). The immensely destructive effect of a close range shotgun injury is much more than that of wounds from more distant discharge. The International Committee of the Red Cross (ICRC) wound classification [4, 7] scores certain features of a wound: the maximum diameter, in cm, of the entry (E) and of the exit (X) wounds; the presence and the size of the cavity (C); the presence of a fracture (F) and the extent of comminution of that fracture; injury to a vital structure which may be the dura, the pleura, the peritoneum, or a major vessel; and the retention of metallic fragments. The wounds are graded according to the amount of tissue damage by the E, X, C, and F scores into low energy transfer, high energy transfer and massive wounds, and then typed according to the structures injured. The wound is then placed in 1 of 12 categories by grade and type (Fig. 4.4).

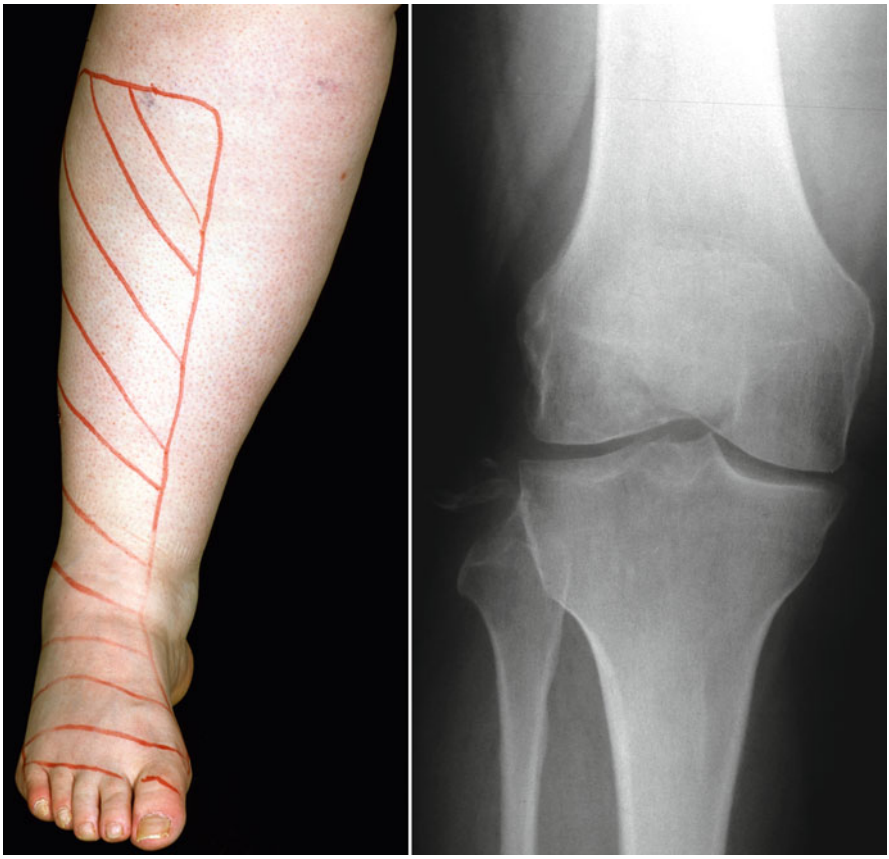

Fig. 4.2 The Platt lesion. A 64 year old woman avulsed the fibular styloid standing up from a chair. The common peroneal nerve was ruptured

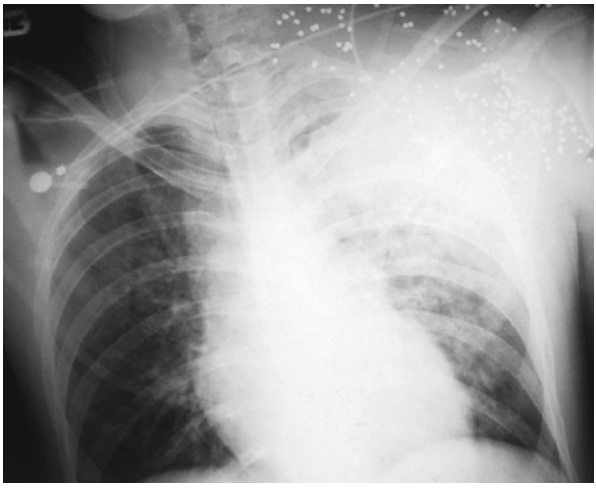

Fig. 4.3 Close range shot gun blast to the posterior triangle of the neck. There was rupture of the first part of the subclavian artery

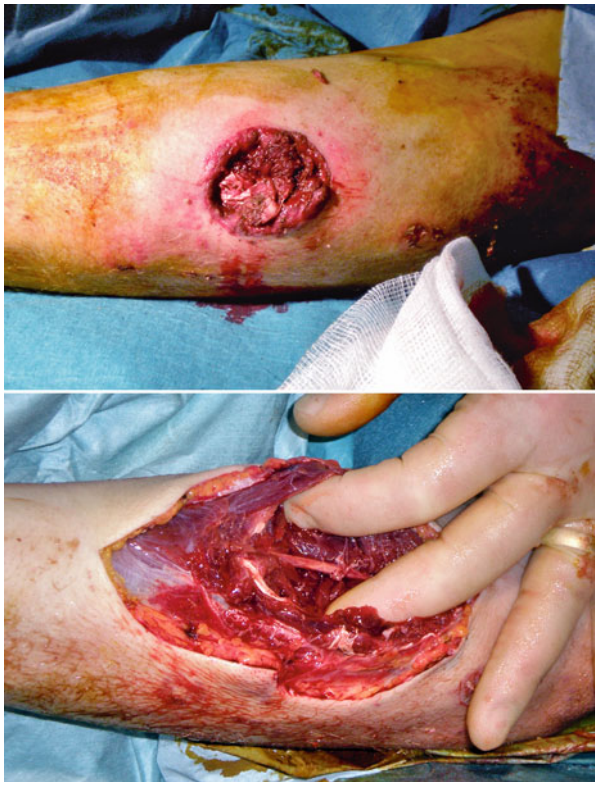

Fig. 4.4 Military rifle bullet, before debridement (*above*) and after debridement (*below*). Type 3 F wound: E3, X8, C2, F2. The common peroneal nerve recovered (axonotmesis)

4.2 Associated Symptoms and Signs

The early symptoms of acute nerve injury include:

- Abnormal spontaneous sensations,
- Alteration or loss of sensibility,
- Weakness and paralysis,
- Impairment of function and sometimes pain.
- Sometimes the patient is aware of warming and dryness of all or part of an extremity. The patient's failure to observe warming and anhidrosis is, regrettably, often shared by the examining clinician (Fig. 4.5).

Neuropathic pain is never easy to recognise in the injured patient who is probably confused, distressed and in pain. It can be distinguished from the pain of fracture or dislocation by loss of sensation, by painful, spontaneous sensory symptoms, expressed throughout the territory of the nerve, and by lancinating or shooting pain irradiating into the distribution of the nerve. In some patients neuropathic pain is so severe that it overwhelms the pain from a fracture. Mothers may advise the clinician that the pain is worse than that of child birth. A constant crushing, bursting or

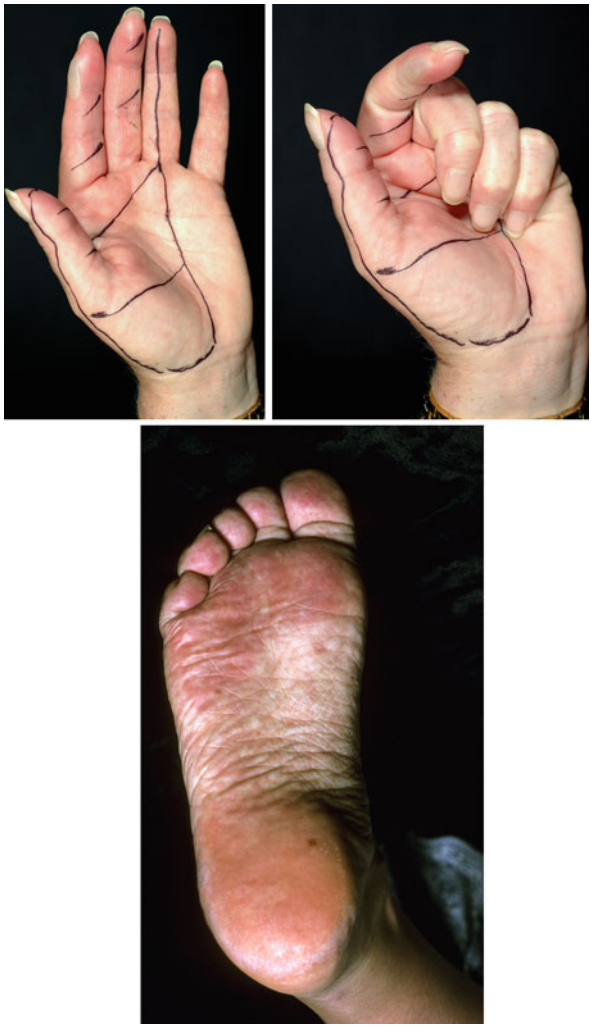

Fig. 4.5 Sympathetic paralysis seen within a few days of transection of (*above*) the median nerve at the elbow and (*below*) the tibial nerve in the thigh

burning pain in the otherwise undamaged hand or foot indicates serious and continuing injury to major trunk nerves more proximally. Progression of sensory loss with a deep bursting or crushing pain within the muscles of the limb signifies critical ischaemia (see Sect. 4.10).

4.2.1 Examination

Examination should enable the clinician to extend the knowledge afforded by the history and the narrative of symptoms to permit accurate diagnosis to be made.

All findings should be recorded in such a manner that the record will be intelligible later not only to the examiner but also to others. Unfortunately, the signs of acute nerve injury have to be sought at a time when the patient may be the least able to co-operate in an examination; soon after wounding, when there is likely to be distress and when the general condition may be affected by loss of blood and other injuries. The examination often has to be done in the often unfavourable surroundings of an accident department. The patient may be a distressed child; an older child, an adolescent or an adult patient who may be affected by drink or drugs or by both. When the lesion has been inflicted by a surgeon or anaesthetist, the patient's response is likely to be distorted by post operative pain, by the effects of recent general anaesthesia or by sedative or analgesic drugs. These are no conditions for a quiet and comprehensive "neurological examination", yet this is the time when the fact of nerve injury must at least be recognised if the best result is to be obtained from treatment. The examiner should at all times bear in mind that if there is a wound over the line of a main nerve and if there is any suggestion of loss of sensibility or impairment of motor function in the distribution of that nerve, it must be regarded as having been cut until *and unless* it is proved otherwise.

Sensory loss is determined by response to light touch and pin prick and if circumstances permit, the patient outlines the area of sensory loss which is marked by a black skin marker pen. The surrounding zone of incomplete sensory loss can be similarly marked in red, and the limb then photographed (Figs. 4.6, 4.7, 4.8, 4.9, 4.10, 4.11, 4.12, 4.13, 4.14, 4.15, 4.16, 4.17, 4.18, 4.19, 4.20, 4.21, 4.22, 4.23 and 4.24).[1] *Selected muscles* are examined. The patient lying supine is usually able to demonstrate activity in serratus anterior by lifting the shoulders away from the couch, by "forward shrugging". It is usually possible to observe the presence of flexion and abduction at the shoulder, flexion and extension of the elbow and wrist and flexion and extension of the fingers. The radial, median and ulnar nerves are tested by asking the patient to form an "O" between the thumb and little finger, to give the "thumbs up", and to open and close the fingers like a fan. It should be possible, by gentle persuasion, to observe active flexion and abduction at the hip, extension at the knee, and extension and flexion at the heel and toes. The palmar and plantar skin is scrutinised for changes in colour and in sweating. Although this may be more difficult in pigmented skin such changes are detectable. The standard tendon reflexes are examined.

A more detailed examination is possible when the patient's condition is stable, and when pain has been controlled. Limb dominance, occupation, marital status, underlying disease or continuing medication are recorded if this has not already been done. Neuropathic pain is by now somewhat easier to recognise, for this is less responsive to analgesics than is pain from skeletal injury

[1] Show the sensory loss after transection, rupture, or avulsion of spinal and peripheral nerves.

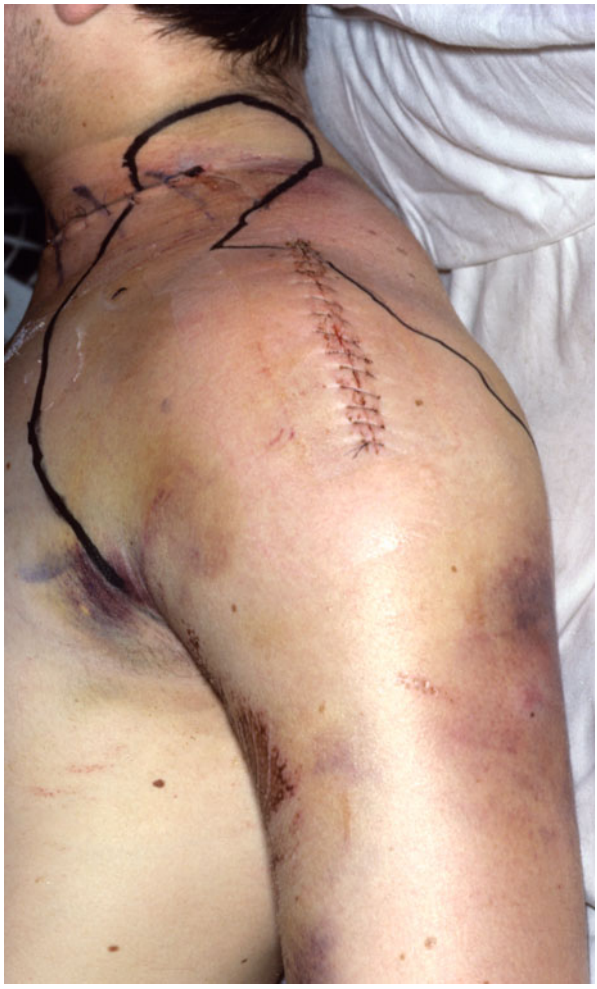

Fig. 4.6 Sensory loss in a case of preganglionic injury C5, C6, C7 and C8 with involvement of C4. The ipsilateral hemi diaphragm was paralysed

4.3 Recognition of the Level and the Depth of Injury

4.3.1 Level

In the absence of wounding clinicians should be able to arrive at an accurate diagnosis of the level of a lesion by clinical examination. A sound grasp of the level of the branches of the trunk nerves and of the contribution to those nerves coming from individual spinal nerves is a prerequisite. "Aids to Examination of the Peripheral Nervous System", [13] originally produced by the Medical Research Council and now in its fourth edition under the direction of Michael O'Brian (2000), is essential

Fig. 4.7 Sensory loss in avulsion of C5-T1. C4 innervates the skin of the outer aspect of the shoulder; T2 innervates the skin of the inner aspect of the arm

reading. This slim volume should be in the possession of all doctors engaged in injury work. It easily fits into a pocket, but now that white coats have been abolished perhaps nurses and therapists who, of course, continue to wear their uniforms, might be invited to carry the volume.

To take one example, the level of injury to the posterior cord and the radial nerve can be determined by examining teres major (inferior subscapular nerve), latissimus dorsi (thoraco dorsal nerve), and deltoid (circumflex nerve). The nerves to the long head of triceps leave the main trunk proximal to the spiral groove. Those innervating the medial head of triceps pass away from the radial nerve at the entrance to and

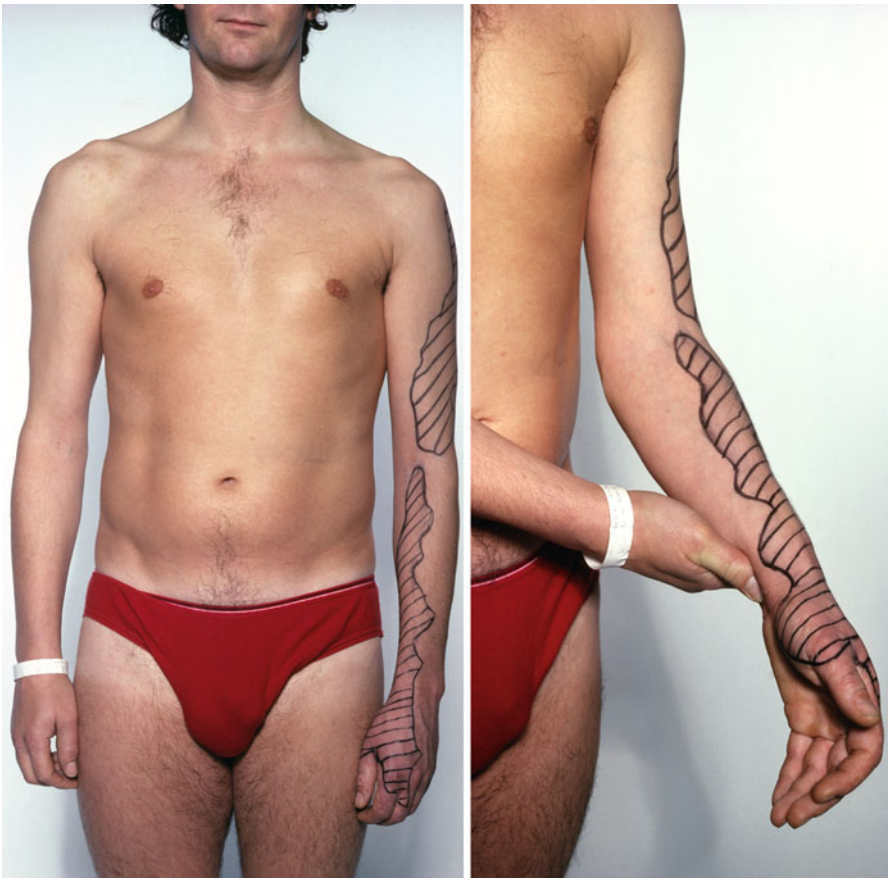

Fig. 4.8 Rupture of C5 and C6. Sensory loss does not extend to the thumb and the index finger

in the first part of the spiral groove whereas those innervating the lateral head of the muscle leave the main nerve still more distally. Paradoxically, the contribution from the spinal nerves is in reverse order: the medial head is usually innervated by the eighth cervical nerve, the long head by the seventh cervical nerve and the lateral head by the sixth cervical nerve. The nerve to brachioradialis consistently passes away from the trunk about three finger breadths above the lateral epicondyle; the nerve to extensor carpi radialis longus comes off about a centimetre more distally. One nerve to extensor carpi radialis brevis leaves the main nerve about 1 cm above the lateral epicondyle and another at the level of the branching into superficial radial and posterior interosseous nerves. Lesions of the sciatic nerve are often, incorrectly, placed at the knee, to the common peroneal nerve. These errors are prevented by examining gluteus medius, gluteus maximus and biceps femoris.

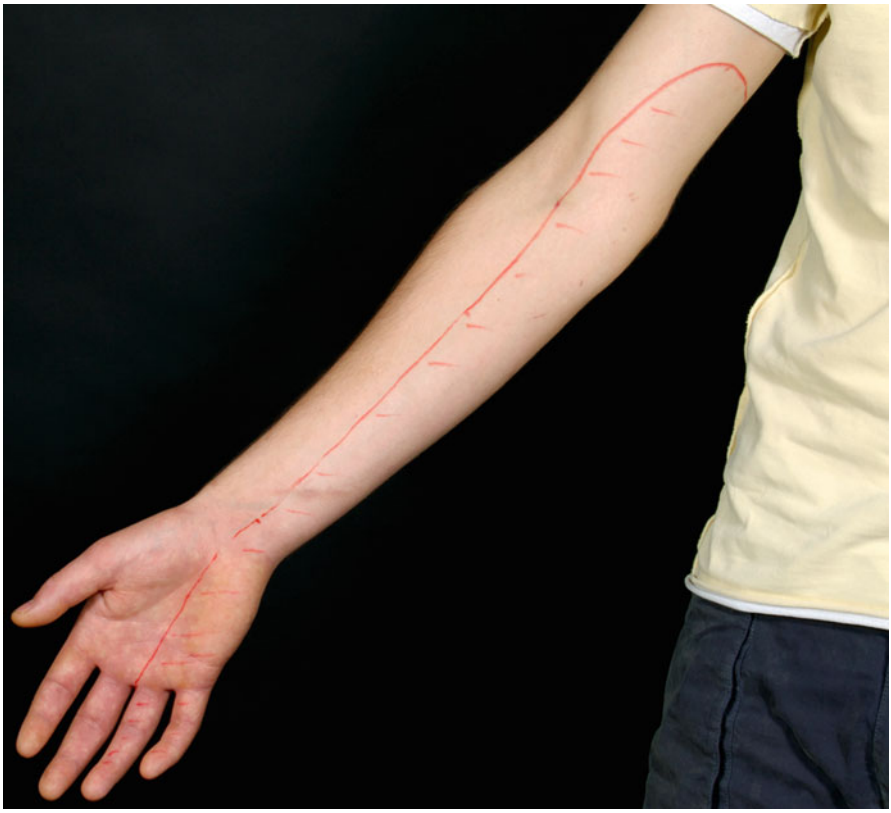

Fig. 4.9 Transection of C8 and T1. The area of sensory disturbance extends into the arm (medial cutaneous nerve of arm)

4.3.2 Depth

Some of the most serious mistakes in the diagnosis and treatment of patients with injured nerves are made because the examiner fails accurately to assess the depth of injury, failing to distinguish between degenerative and non-degenerative injury and to estimate the extent in the nerve of each type of lesion. Some atavistic urge seems to cause clinicians to play down the severity of nerve injury. Perhaps beneath this urge there is a feeling that if there is a serious injury, much hard and possibly unrewarding work is going to be required. The tendency is of course particularly marked in cases of closed injury and of injury during operation. Too often the mantra "Neuropraxia" is pronounced: too often the soothing words "just some bruising of the nerve" are uttered.

The diagnosis of the depth of the injury depends on the history and signs and on the simplest electrical examination. Serious injuries are likely to cause serious lesions of nerves. Severance of a nerve with a cutaneous sensory component will lead to

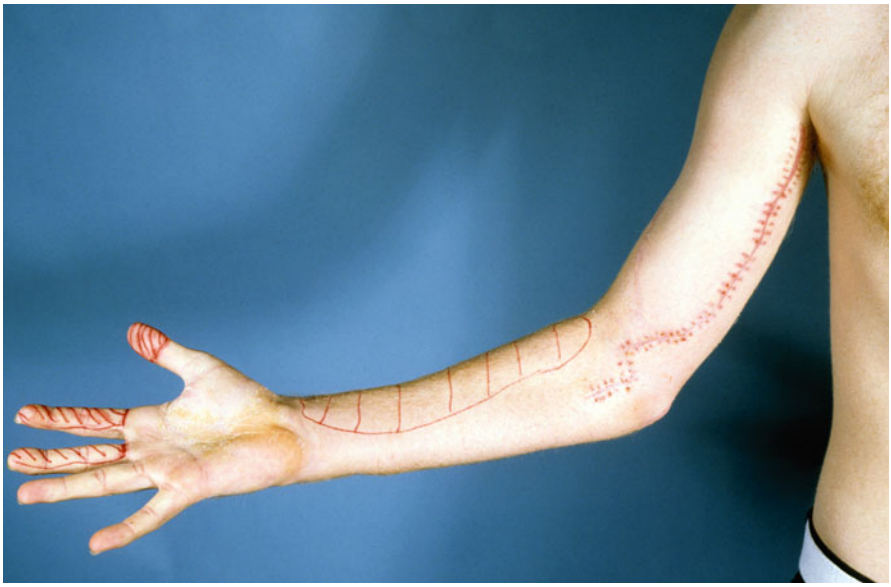

Fig. 4.10 The area of sensory abnormality after section of the lateral cord in the axilla. As in Fig. 4.9, there was no complete loss of sensation.

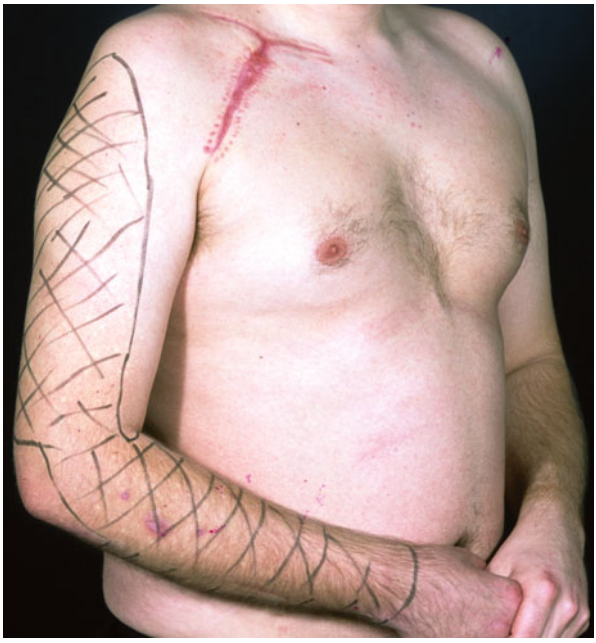

Fig. 4.11 Rupture of the posterior divisions of the trunks of the brachial plexus deep to the clavicle

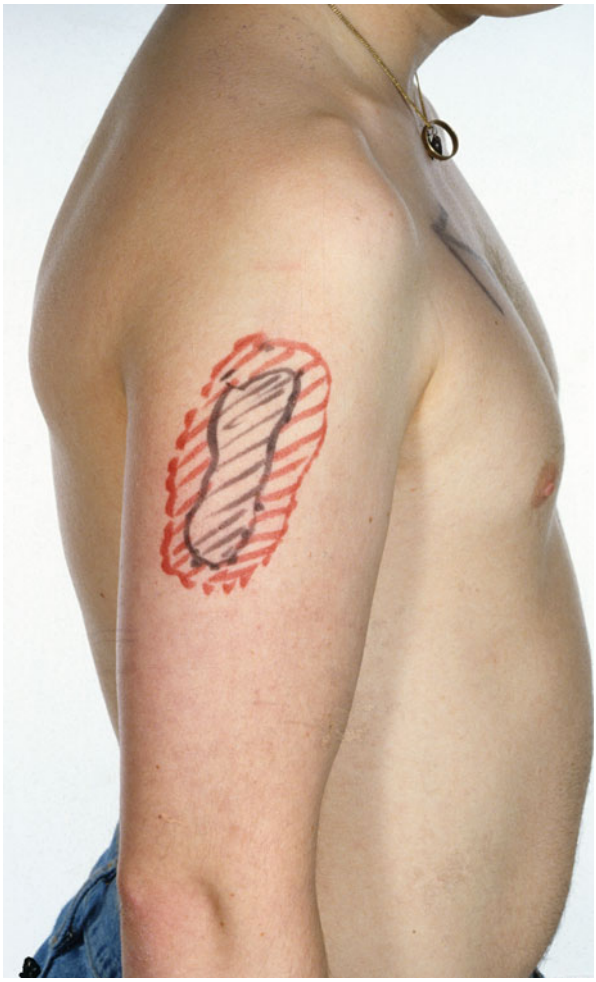

Fig. 4.12 Rupture of the circumflex nerve: the sensory loss is usually smaller than in lesions of C5

well-defined loss of sensibility and to complete motor, sudomotor and vasomotor paralysis in the distribution of the nerve. Simple conduction block is likely to produce a patchy loss of sensibility and a patchy motor loss. Further, it is likely to bear more heavily on the large axons than on the small ones: vibration sense and sensibility to light touch are likely to be impaired, whereas pain sensibility may be unaffected.

4.4 Signs

The early signs of nerve injury are:

- Alteration or loss of sensibility,
- Weakness or paralysis of muscles,

Fig. 4.13 Sensory loss in two cases of rupture of the musculocutaneous nerve. Both patients were able to supinate the forearm fully; the power of elbow flexion was around 30 %

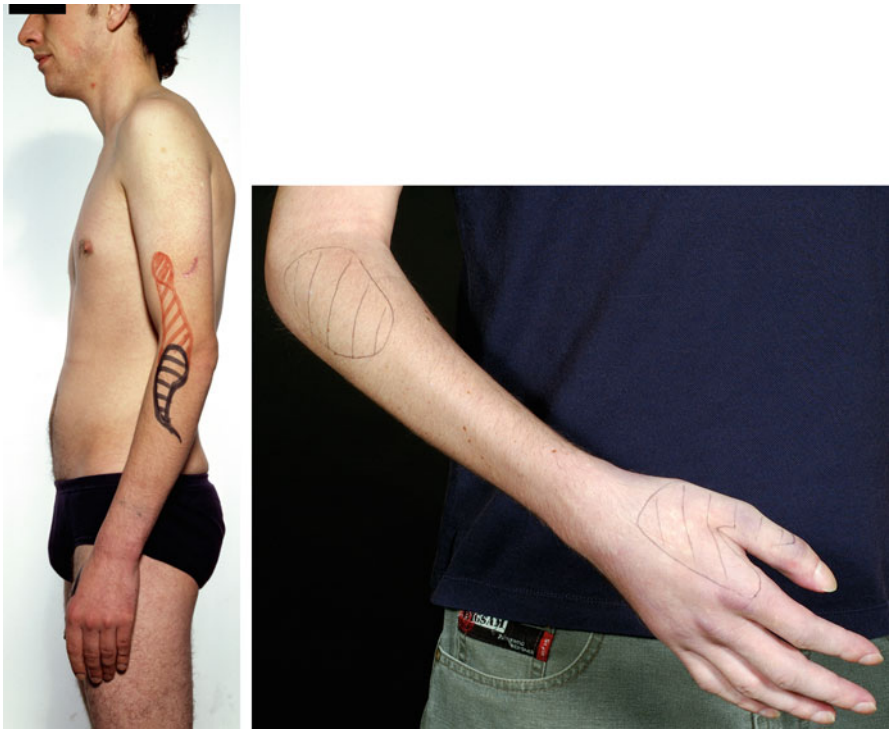

Fig. 4.14 High lesion of the radial nerve. *Right*: there is early recovery into the wrist extensors after repair

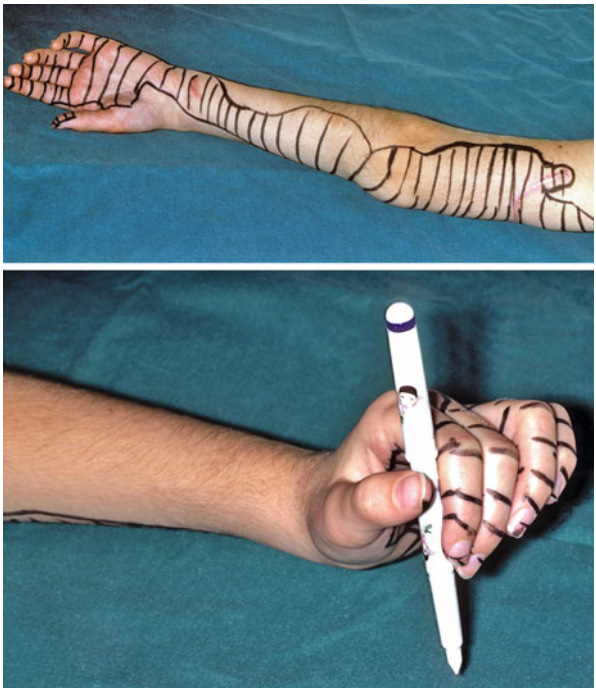

Fig. 4.15 Transection of median, ulnar, medial cutaneous nerve of forearm, and brachial artery in the arm of a 14 year old boy. Note the extent of skin innervation provided by the superficial radial and lateral cutaneous nerves of forearm. The intact radial nerve permits a sort of grasp

- Vasomotor and sudomotor paralysis in the distribution of the affected nerve or nerves,
- Abnormal sensitivity over the nerve at the point of injury.

Testing of sensibility is often difficult soon after wounding, or when nerve injury is associated with fracture of a long bone. The actions of some muscles can be simulated by the actions of others, so that the fact of paralysis can be missed in the early stages after nerve injury. However, one almost infallible sign is always present in the first 48 h after deep injury of a nerve with a cutaneous sensory component: because of the affection of small as well as of large fibres, *the skin in the distribution of the affected nerve is warm and dry*. In the small child, there may be an abnormal posture of the denervated digits. Another test for nerve injury in small infants is the "immersion test": the injured hand or foot is placed, for a few minutes, in warm water. The skin of the denervated digits fails to wrinkle (Fig. 4.25). Other early signs which indicate a deep injury to a nerve include changes in texture of the skin rather like "goose pimples," development of a skin rash, and hypersensitivity surrounding the area of anaesthesia.

When there is no breach of the skin and the injury of the nerve is caused by pressure or distortion, there is usually differential affection of fibres. Peripheral ischaemia is usually signalled by pain, but in cases in which the vascular injury is associated with fracture, the significance of that pain may not be recognised. Ischaemia affects first the large fibres: discriminative sensibility and vibration sense are first affected

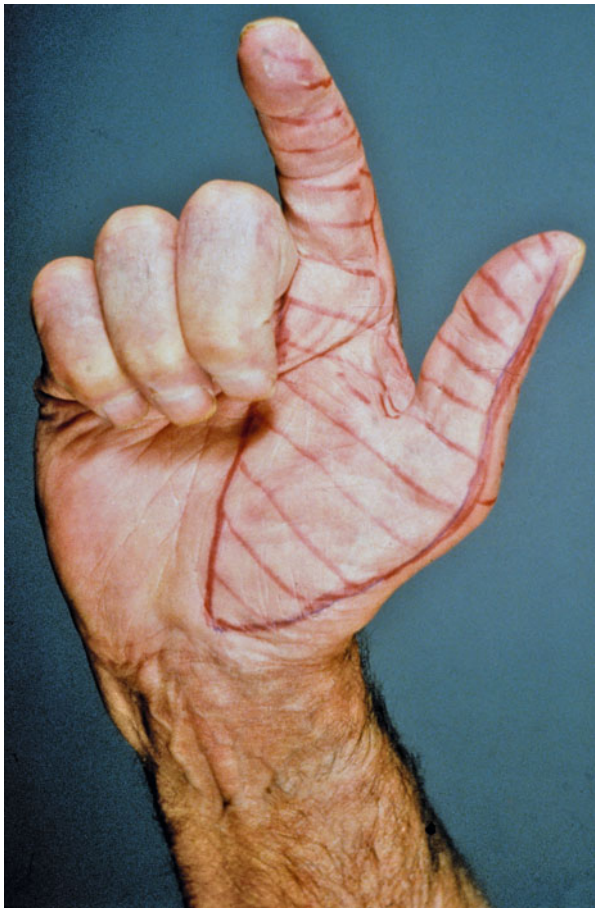

Fig. 4.16 High median nerve injury: no active flexion of the index finger and thumb

It is not easy to test these modalities when ischaemia is developing because of damage to a main vessel associated with a fracture of a long bone, but if action is not taken until superficial sensibility is lost, it will come too late.

4.5 Tinel's Sign

In closed injuries percussion of the skin over a nerve which has sustained a degenerative lesion, either axonotmesis or neurotmesis or a mixture of both, evokes sensations usually described as a wave or surge of pins and needles into the cutaneous distribution of the nerve. This is Tinel's sign and it is a most useful aid to diagnosis. The sign is elicited on the day of injury in most conscious patients. It indicates not only where the nerve has been injured but also the fact that at least some axons have been ruptured. Tinel's sign can be detected over such "motor" nerves, as the posterior

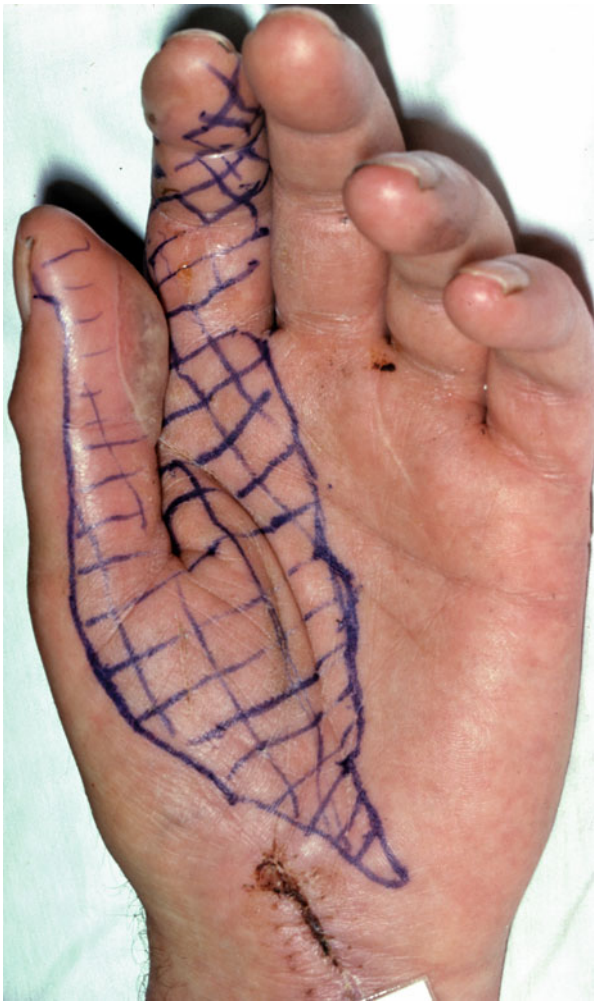

Fig. 4.17 Section of the median and the palmar cutaneous nerve at wrist

interosseous. The sensory symptoms radiate into the muscular territory rather than into the skin. It is more difficult to elicit the sign over deep seated nerves such as the circumflex, the eighth cervical or first thoracic nerves.

The Tinel-like sign elicited by percussion over schwannoma or over nerves in the early stages of entrapment neuropathy does not indicate that axons have been ruptured, rather that nerve fibres have become sensitised because of focal demyelination and changes in the expression of voltage gated ion channels at the level of lesion.

These points can be stated:

- a strongly positive Tinel sign over a lesion soon after injury indicates rupture of axons or severance of the nerve;
- A positive sign means the lesion is degenerative, not a conduction block, for at least a significant number of axons.

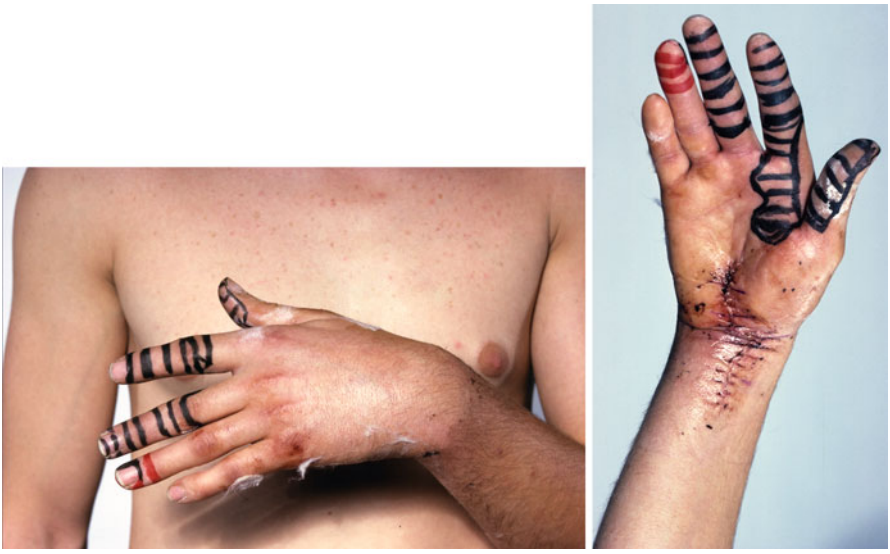

Fig. 4.18 A typical area of loss of sensibility after division of the nerve at the wrist, sparing the palmar cutaneous branch

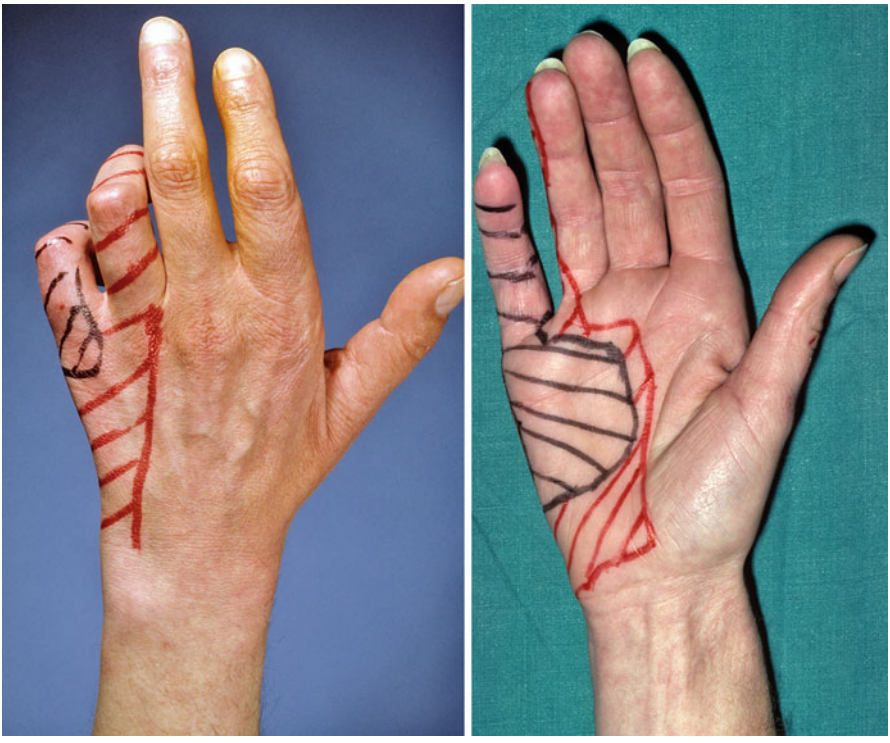

Fig. 4.19 The area of sensory loss and "clawing" of little and ring fingers after division of ulnar nerve in the forearm

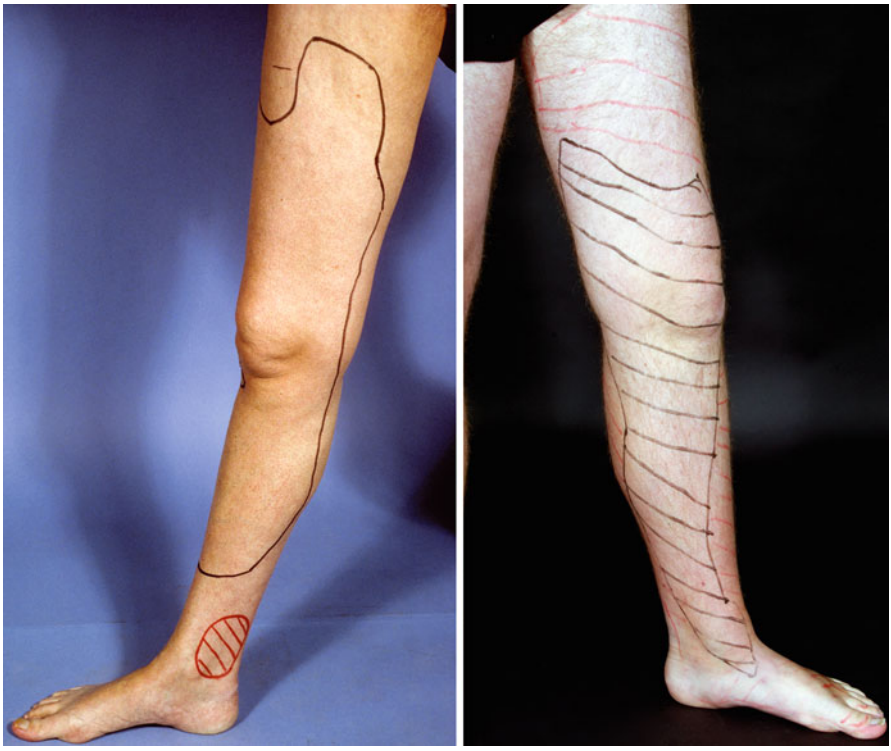

Fig. 4.20 The area of loss of sensibility in two cases of injury to the femoral nerve at, or proximal to the groin crease

- Failure of distal progression of the sign in a closed lesion indicates rupture or other injury not susceptible of recovery by natural process.
- in favourable degenerative lesions (axonotmesis) or after repair which is going to be successful, the centrifugally moving sign is persistently stronger than that at the suture line;
- after repair which is going to fail, the sign at the suture line remains stronger than that at the growing point;

4.5.1 Eliciting the Tinel Sign in Closed Lesions

The examiner's finger percusses along the course of the nerve from DISTAL to PROXIMAL starting well below the presumed level of lesion. The patient is asked to say when the advancing finger elicits a wave or a surge of pins and needles or abnormal sensations, which may be painful, into the distribution of the nerve which must be clearly indicated by the examiner. The level of the sign should be measured

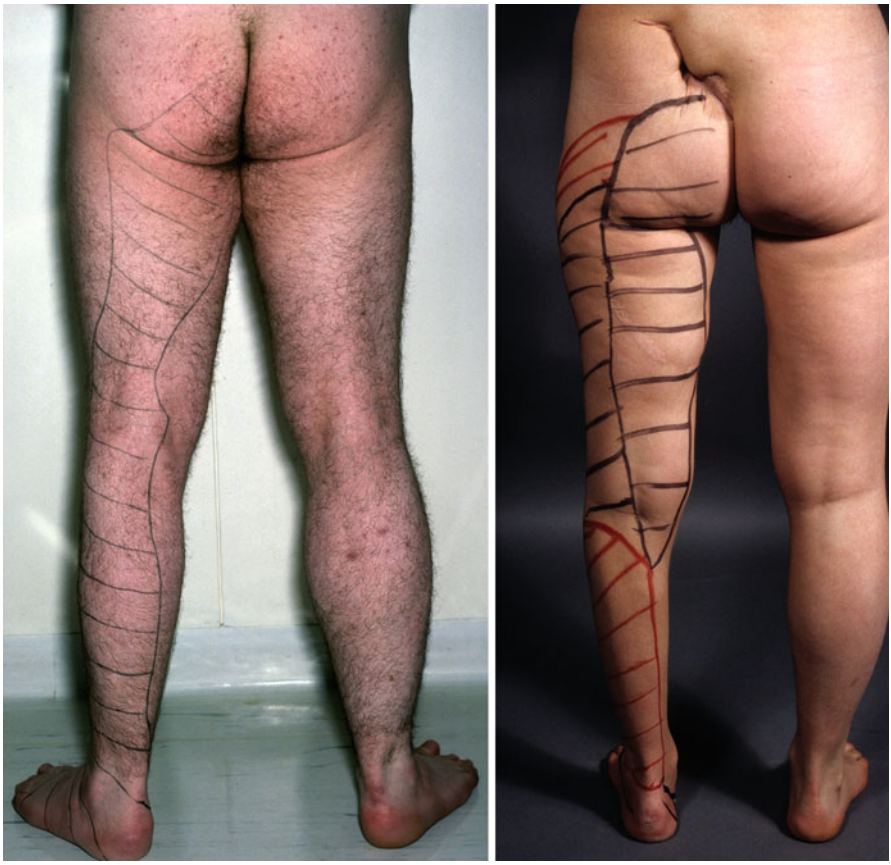

Fig. 4.21 Cutaneous distribution of the sacral plexus. *Left* showing area of sensory loss after closed fracture/dislocation of sacro-iliac joint. *Right* showing area of loss of sensibility after open fracture/dislocation of the pelvis. The muscles of the buttock are wasted

from a fixed point and the distance entered into the records. At times the examination is painful and patients need to be warned about that. Tinel's sign is valuable in the diagnosis of post ganglionic rupture of the spinal nerves of the brachial plexus. If percussion in the posterior triangle induces radiation as far as the elbow then rupture of C5 is likely; rupture of C6 is anticipated when radiation extends to the lateral forearm and thumb and when radiation extends to the whole hand, especially to the dorsum, then rupture of C7 is expected. Percussion over the swollen posterior triangle of the neck in cases of multiple avulsion usually elicits painful sensory phenomena which do not radiate into the dermatomes of the injured nerves. A Tinel sign which remains static at the level of lesion strongly suggests rupture of the nerve or persisting local conditions inimical to spontaneous regeneration. Operation is indicated [10] (Fig. 4.26). Table 4.1 shows the value of a static or advancing Tinel sign in predicting recovery in degenerative lesions after closed injury to the common

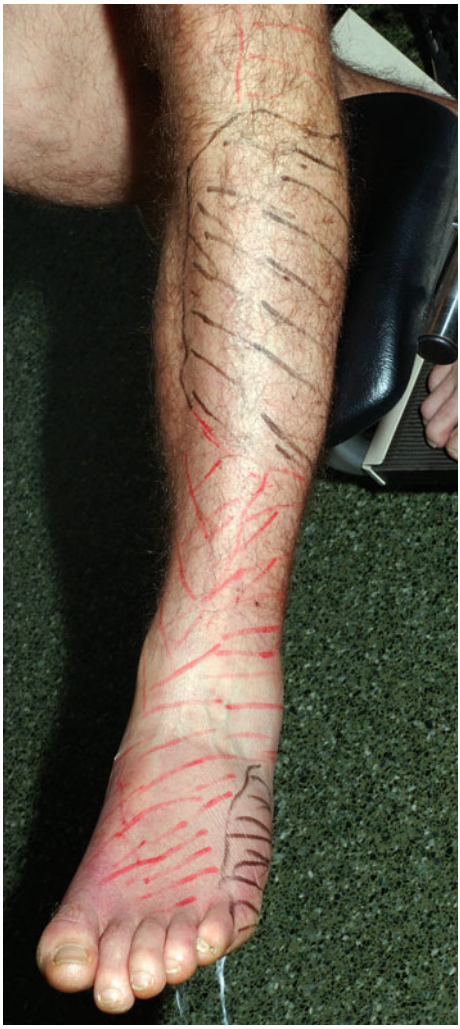

Fig. 4.22 The area of sensory loss after transection of sciatic nerve in the thigh is confined to the leg and foot

peroneal, the radial and tibial nerves. An advancing sign proved misleading in 18 nerves. In most of these the distal muscles had been damaged by ischaemia so that the regenerating axons arrived at target organs which were irredeemably fibrosed.

4.5.2 Tinel's Sign and Recovery

By between 4 and 6 weeks from the day of the injury it is usually possible to distinguish between axonotmesis and neurotmesis in closed lesions of the radial, median, ulnar, common peroneal and tibial nerves. However an advancing sign may also be found when only a few nerve fibres are regenerating as in cases where trunk nerves

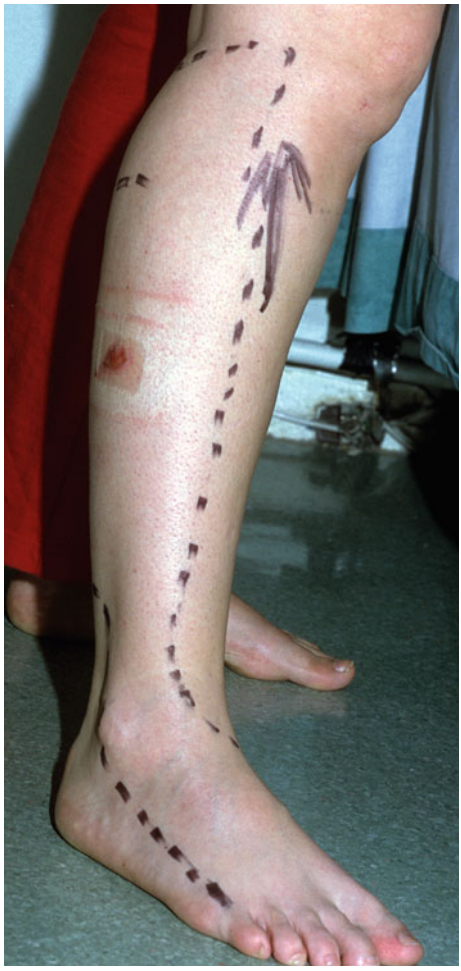

Fig. 4.23 Area of sensory loss after high division of common peroneal nerve during operation of knee ligament reconstruction. Note the pressure sore caused by a conventional ankle-foot orthosis

are entrapped within a fracture or joint. Centrifugal progress of the sign is often unreliable in predicting recovery of lesions of the sciatic nerve incurred during arthroplasty of the hip. Most of these are mixed lesions, some nerve fibres are intact, others sustain conduction block whilst many more have sustained degenerative lesions which may or may not be naturally favourable (see Sect. 2.4).

4.6 Examination of Sensibility

The Medical Research Council [8] method of recording sensibility offers a reasonable method for recording and measuring progress. It has obvious disadvantages, but no comprehensive method has yet been devised that does not have the overwhelming disadvantage of extreme complication (Table 4.2).

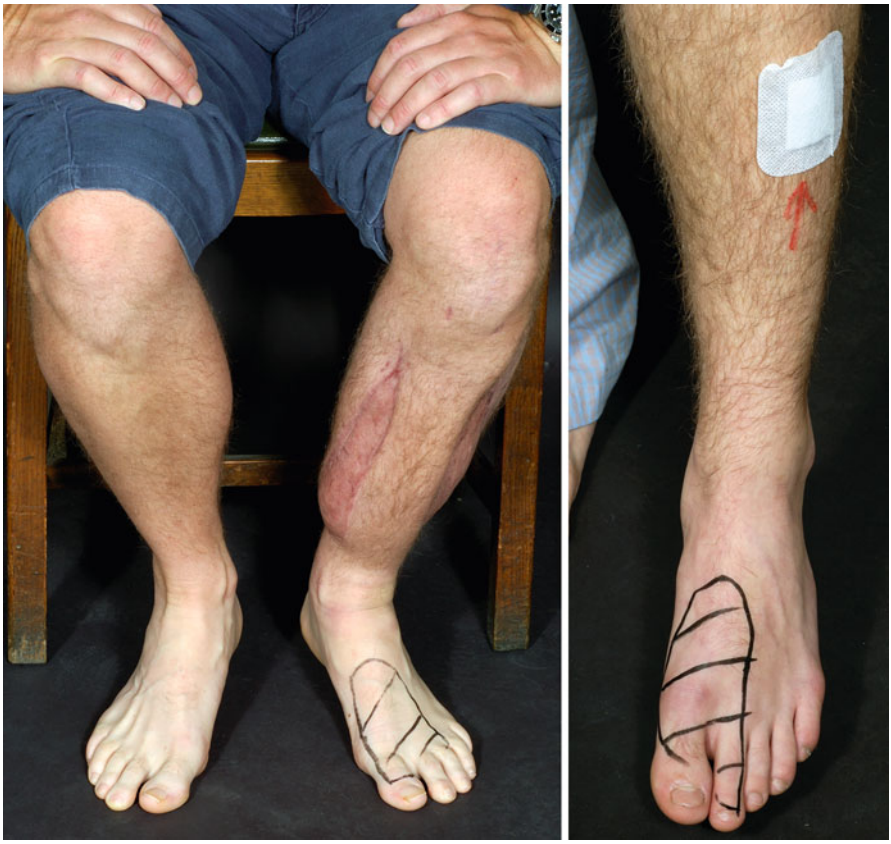

Fig. 4.24 Area of sensory loss after interruption of the deep division of the common peroneal nerve. *Left*, showing the leg of a 29 year old man in whom severe "compartment syndrome" was overlooked after intramedullary nailing of closed fracture of tibial shaft. The anterior compartment was infarcted and it was excised. *Right*, showing the area of sensory loss after transection of the deep division of the common peroneal nerve by a knife

The modalities routinely tested are light touch, localisation, temperature, position sense, pain and, sometimes, two point discrimination.

Light touch and localisation: The examiner's finger or a wisp of cotton wool is moved lightly across the area under test. With the eyes closed the patient is asked to say *where* the stimulus is applied to the limb and to say yes if the stimulus is appreciated but not localised. Sennes Weinstein hairs (made by A. Ainsworth, University College, London) are more sensitive and provide a measure of pressure sense. The localisation chart [20] is used for recording sensation in the hand (Fig. 4.27).

Two point discrimination [8]: This is done with the blunted points of a compass or the ends of a paper clip or with a special device. The patient is first instructed: "I shall touch your finger now with one point; now with two. If you feel one, say

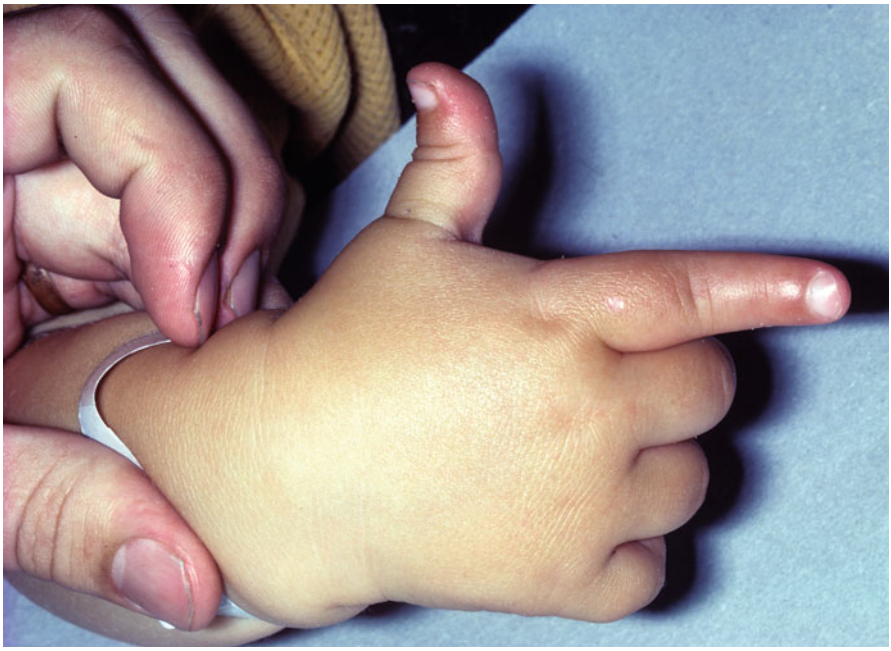

Fig. 4.25 Infant's hand 24 h after section of the palmar nerves to the index finger and thumb. Note that the anaesthetic digits are held out from the others. There was no tendon injury

"one"; if you feel two, say "two"; if you are in doubt, say "one" ". Then, with closed eyes, the patient attempts distinction between one and two points. The test indicates the degree of reinnervation of slowly adapting receptors. The patient easily gets confused; it is difficult or impossible to ensure that the same pressure is used throughout the test.

Temperature: Plastic tubes are used: one contains cold water, the other, warm at about 35 °C. These are applied alternately to the area under test.

Position sense: All nearby joints, other than the one being tested, must be stabilised. The patient is first shown the direction in which the joint is being moved. Then, with eyes closed, he or she is asked to indicate the direction in which the joint is being moved.

Pin prick: A bluntened pin is lightly applied to the skin and the patient is asked to say whether it feels sharp or blunt

Recognition of textures and shapes: The methods and charts developed by Wynn Parry and Salter [20] are used. The blindfolded patient is presented with a series of objects of differing shape, texture and surface character, and asked to distinguish them. The number correctly identified and the time taken are recorded. Later, common textures and small and large objects in daily use are presented for recognition. These tests provide a better measure of regeneration and function than two point discrimination [9].

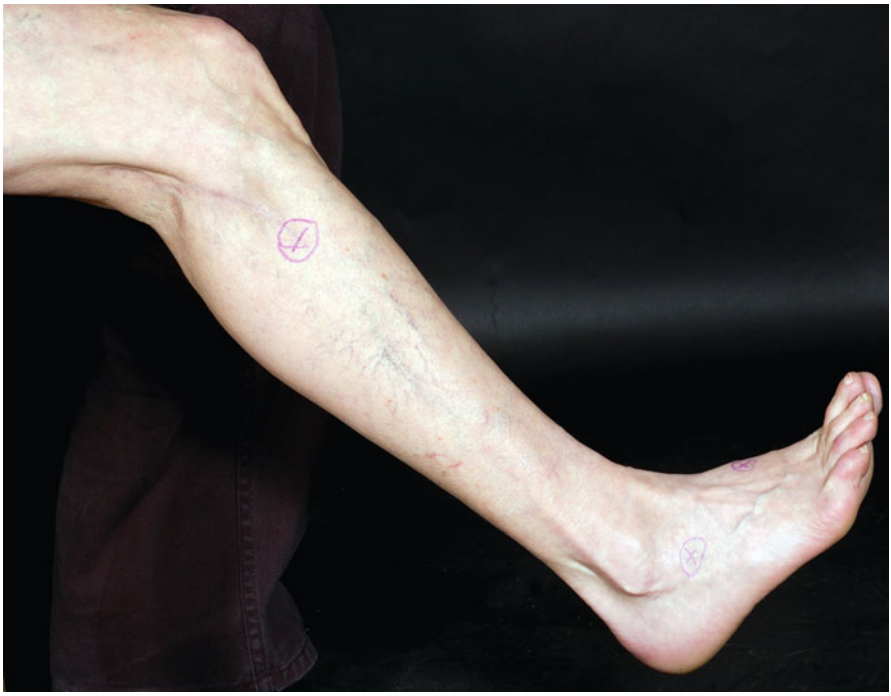

Fig. 4.26 Static and progressing Tinel's signs. A 43 year old woman sustained a complete and very painful, lesion of the common peroneal nerve from the kick of a horse. There was a strong, painful, Tinel sign at the level of lesion at the time of exploration 10 weeks after injury. The nerve was deeply compressed by scar from which it was removed. Her pain was abolished. The rate of progress of the Tinel sign for the superficial and the deep divisions of the nerve was about 2 mm a day. There was complete recovery

Table 4.1 Tinel's sign as a guide to prognosis in 339 consecutive cases of degenerative lesions in closed injuries to the common peroneal (171 cases), the radial (139 cases) and the tibial (29 cases) nerves examined 2000–2007

	Tinel sign – progressing		Tinel sign – static	
	Spontaneous recovery	Misleading – no, or poor, spontaneous recovery	Spontaneous recovery	Rupture or other lesion not susceptible to recovery by natural process
Common peroneal nerve with divisions	84	12	0	75
Radial nerve	103	5	0	31
Tibial nerve	16	1	0	12
Total	203	18	0	118

Table 4.2 Sensory recovery

The original grading by Highet 1941 [1]	
Stage 0	Absence of sensibility in the autonomous zone of the nerve
Stage 1	Recovery of deep cutaneous pain sensibility within the autonomous zone
Stage 2	Return of some degree of superficial pain and tactile sensibility within the autonomous zone
Stage 3	Return of superficial pain and tactile sensibility throughout the autonomous zone with the disappearance of over-response
Stage 4	Return of sensibility as in Stage 3 with the addition that there is recovery of two-point discrimination within the autonomous zone
The Medical Research Council System 1954 [1]	
S0	Absence of sensibility in the *autonomous area*
S1	Recovery of deep cutaneous pain sensibility within the *autonomous area* of the nerve
S2	Return of some degree of superficial cutaneous pain and tactile sensibility within the *autonomous are*a of the nerve
S3	Return of some degree of superficial cutaneous pain and tactile sensibility within the *autonomous area* with disappearance of any previous over-reaction
S3+	Return of sensibility as in Stage 3 with the addition that there is some recovery of two-point discrimination within the *autonomous area*
S4	Complete recovery

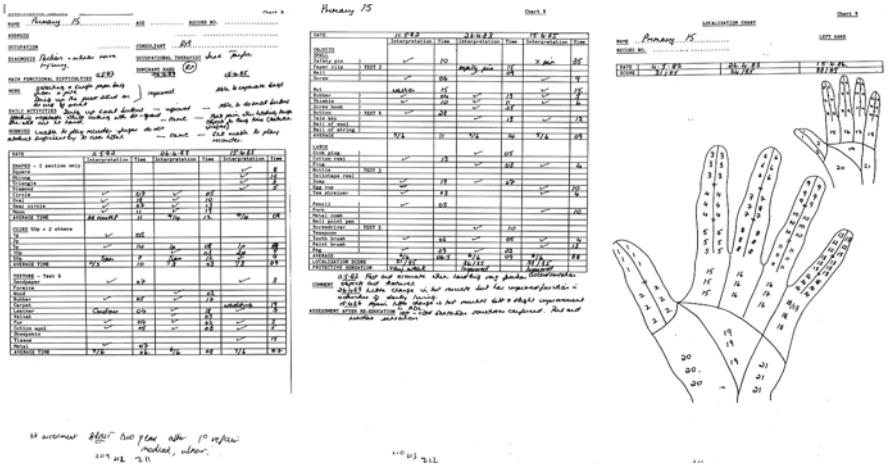

Fig. 4.27 Charts recording recovery of localisation and recognition after repair of median nerve

4.6.1 Quantitative Sensory Testing (QST)

QST is the use of stimuli which are more precisely quantitative and more rigorously controlled. The technique is not generally available but it is an important development. Some findings from the use of QST include:

- focal, or generalised neuropathy in patients wrongly labelled as "complex regional pain syndrome Type 1" (CRPS Type 1);
- differential susceptibility of nerve fibres to different lesions;
- differential rates of recovery in different populations of nerve fibres

The methods include measurement of.

- such subjective senses as thermal threshold, light touch and vibration;
- Sweating and the histamine induced flare response;
- studies of conduction within small sensory fibres (Aδ and C fibres) by Contact Heat Evoked Potential Stimulator (CHEPS);
- Conduction in somatic efferent pathways by transcranial electromagnetic evoked potentials (TCEMEP). For further discussion see Birch [1].

4.7 Examination of Muscles

The examination of muscles in the normal subject is illustrated in the CD or video accompanying this book.

4.7.1 Some Pitfalls

Substitution. It is important for the examiner to palpate the belly of the muscle under examination and at the same time, to palpate the tendon of that muscle. Brachioradialis alone is a powerful flexor of the elbow. The power of gravity suffices to extend the elbow in the absence of triceps. Paralysis of the extensor and flexor muscles of the wrist may be masked by the extensor and flexor muscles to the digits. Extensor pollicus longus is capable of extending the wrist, abductor pollicus longus can mimic this action if the wrist is partially pronated. The abductor and flexor brevis Pollicis have insertions to the extensor expansion, so that adduction of the thumb extends the interphalangeal joint, even when the extensor muscles are paralysed. Strong extension of the fingers can give an impression of an abducting action in the interossei, whilst strong flexion can give the appearance of an adducting action. The interosseous muscles extend the proximal interphalangeal joints whilst the metacarpophalangeal joints are flexed. The peroneal muscles can produce dorsi flexion at the ankle.

"*Tenodesis*" *action.* When the long flexors of the fingers are paralysed, extension of the wrist produces in them sufficient tension to cause flexion of the interphalangeal joints. A similar response is seen in the toes when the ankle is dorsiflexed. The "tenodesis" effect underlies most muscle transfers (Figs. 4.28 and 4.29). The effect is increased by moderate post ischaemic fibrosis which causes the fixed length deformity (Fig. 4.30).

Rebound. When the antagonist to a paralysed muscle contracts strongly and relaxes quickly it may appear as a contraction of paralysed muscles. In paralysis of the common peroneal nerve, the patient can mimic active extension of the toes

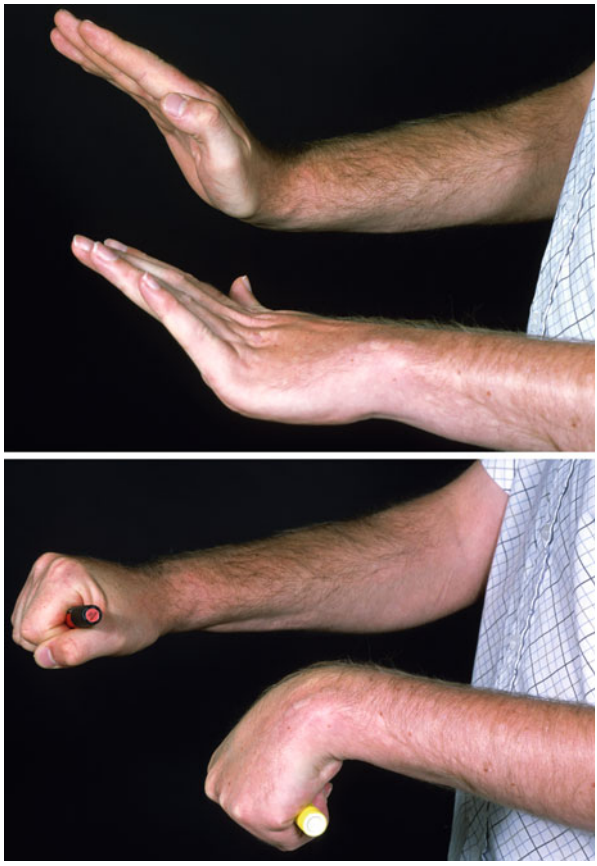

Fig. 4.28 The importance of wrist extension. This 31 year old graphic designer sustained bilateral lesion of the brachial plexus. Avulsion of C7 on the right was treated by flexor to extensor transfer, achieving a power grip at 50 % of estimated normal. On the left C6, C7 and C8 were avulsed and only one FDS muscle was available for transfer to EDC and EPL. Power grip was negligible.

or active extension at the ankle by strong contraction and sudden relaxation of the flexors.

4.7.2 Measurement of Muscle Power

No system for recording of motor power has really superseded that proposed in 1941 by Highet [8] to the Nerve Injuries Committee of the Medical Research Council (Table 4.3). This is modified for individual muscles (see Table 4.4). The scale is non linear and the individual grades represent a wide range of actual power [11, 12, 17].

Muscle power may be measured more accurately by using instruments which also provide information about stamina. In the hand those devised by Mannerfeldt and made by HC Ulrich (Ulm) show that power of pinch grip is reduced by about one third in low median palsy, and by nearly three quarters in low ulnar palsy. Power

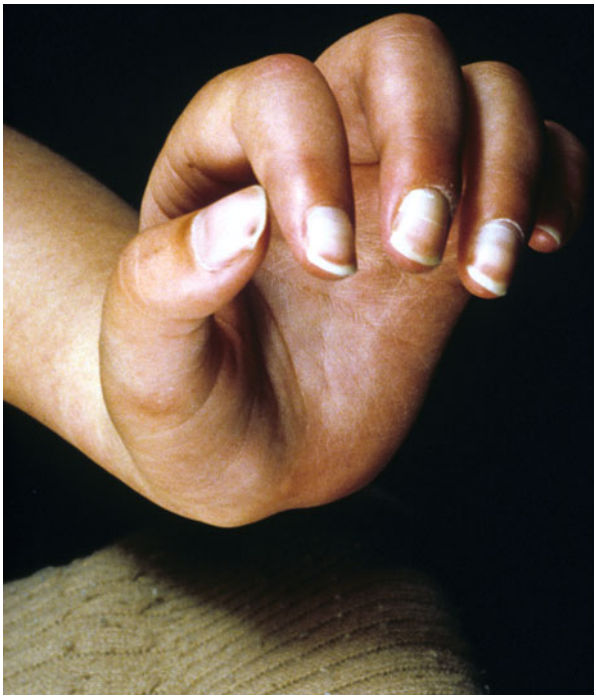

Fig. 4.29 "Tenodesis" effect of the wrist extensor muscles in a 13 year old child with high median and ulnar palsy

grip is reduced by about one-half in high ulnar palsy and it is as low as 20 % in radial palsy, such is the importance of extension of the wrist [1].

A myometer (model D60107MK1. Penny and Giles Transducers, Christchurch, Hampshire) is used for the examination of more proximal muscles. For the shoulder and arm, the patient is seated comfortably with their back erect against the upright of a chair, both upper limbs are held in the same position. The examiner applies force against the arm using the appropriate cup. The amount of force required to overcome the patient's resistance is noted and recorded as a percentage of the opposite limb. For hip flexion, the patient lies supine, on their side for abduction and for extension prone. Power of extension of the knee is best measured with the patient sitting with the legs over the side of the couch (Fig. 4.31).

Although many patients with isolated paralysis of deltoid show a complete range of active movement at the shoulder, the power of forward flexion and abduction is reduced to about 40 % of the uninjured side. The power of extension of the shoulder, measured at 90° of abduction, is reduced to as little as 5 %. The power of abduction after a "good" result of repair of the circumflex nerve reaches about 60 % of the uninjured side. The power of elbow flexion after musculocutaneous palsy is reduced to between 20 and 40 % of the uninjured side. It approaches 60–80 % of normal after successful repair of the nerve. The power of dorsiflexion of the ankle after "good" results of repair of the common peroneal nerve is around 50 % of normal, that of extension of the knee after successful repair of the femoral nerve, about 60 %

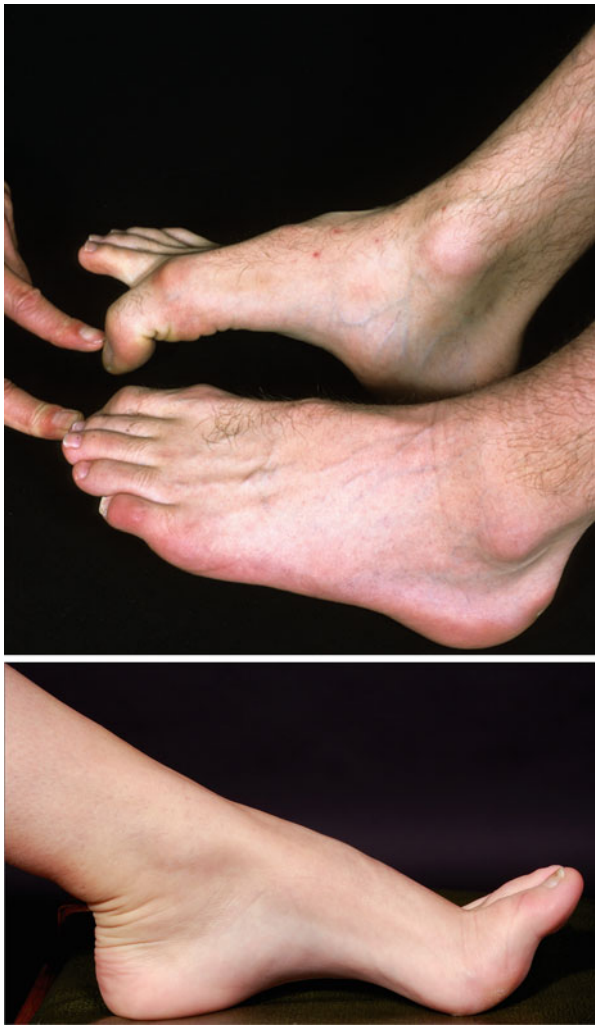

Fig. 4.30 Examples of post ischaemic fibrosis of the anterior compartment of the leg complicating intramedullary nailing. *Above*, showing contracture of extensor hallucis longus; *below*, the contracture involves extensor digitorum longus and extensor hallucis longus

of normal. Although these figures fall short of normal, they are, of course, far superior to the power restored by muscle transfers [1].

4.8 Some Difficulties in Diagnosis

It is with the large proximal muscles, about the shoulder girdle and about the hip, that serious mistakes are most common. Delay before diagnosis of nerve injury is, in many cases, quite alarming despite the reliability of precise but elementary clinical examination. Some areas of particular difficulty are described.

Table 4.3 Motor recovery

The original grading proposed by Highet 1941 [1]	
Stage 0	No contraction
Stage 1	Return of perceptible contraction in proximal muscles
Stage 2	Return of perceptible contraction in both proximal and distal muscles
Stage 3	Return of function in both proximal and distal muscles to such an extent that all important muscles are of sufficient power to act against resistance
Stage 4	Return of function as in Stage 3 with the addition that all synergic and isolated movements are possible
Stage 5	Complete recovery
The Medical Research Council System 1954 [1]	
M0	No contraction
M1	Return of perceptible contraction in the proximal muscles
M2	Return of perceptible contraction in both proximal and distal muscles
M3	Return of perceptible contraction in both proximal and distal muscles of such degree that all *important* muscles are sufficiently powerful to act against resistance
M4	Return of function as in Stage 3 with the addition that all *synergic* and independent movements are possible
M5	Complete recovery

Table 4.4 Grading of power of individual muscles or muscle groups

0	Complete paralysis
1	"Flicker": visible and palpable contraction in the muscle
2	Management of joints possible *with gravity eliminated*
3	Movement of joint *against gravity*
4	Movement of joint against resistance
5	Full power

4.8.1 Thoraco Scapular, Thoraco Humeral, and Scapulo Humeral Muscles

The inferior scapulo-humeral angle (ISHA), is helpful in the analysis of injuries to the nerves to these muscles. The ISHA is subtended by the long axis of the humerus and the lateral border of the scapula. The tip of that angle is centred over the gleno-humeral joint. It is measured at rest and then with the arm in full active elevation (Fig. 4.32). This simple investigation measures the respective contributions to elevation provided by the thoraco-scapular and the gleno-humeral joints. The *active* ISHA in the normal limb lies between 150° and 170°. Stiffness of the joints is detected by measuring the *passive* range which, in the normal limb lies between 170° and 180°.

Transection of the spinal accessory nerve, usually at the apex of the posterior triangle and usually caused by surgeons, is crippling. Most patients experience immediate pain and demonstrate remarkable loss of function (Fig. 4.33). The scapula drops downwards, and away from the spine. The average *active* ISHA is about 50°. The "winging" is often wrongly attributed to paralysis of serratus anterior muscle (Fig. 4.34).

Fig. 4.31 Measuring power using a myometer, in this case of the muscles at the shoulder. The patient is seated

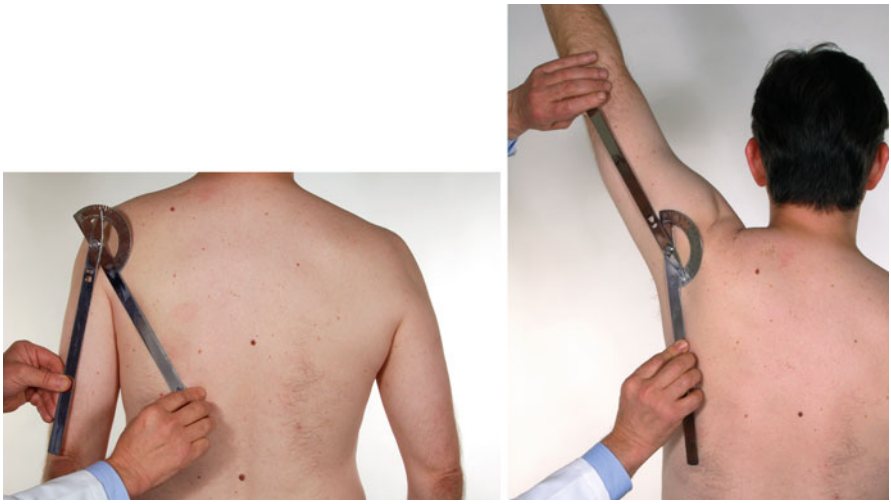

Fig. 4.32 The active inferior scapula-humeral angle (ISHA) in a normal shoulder is about 170°

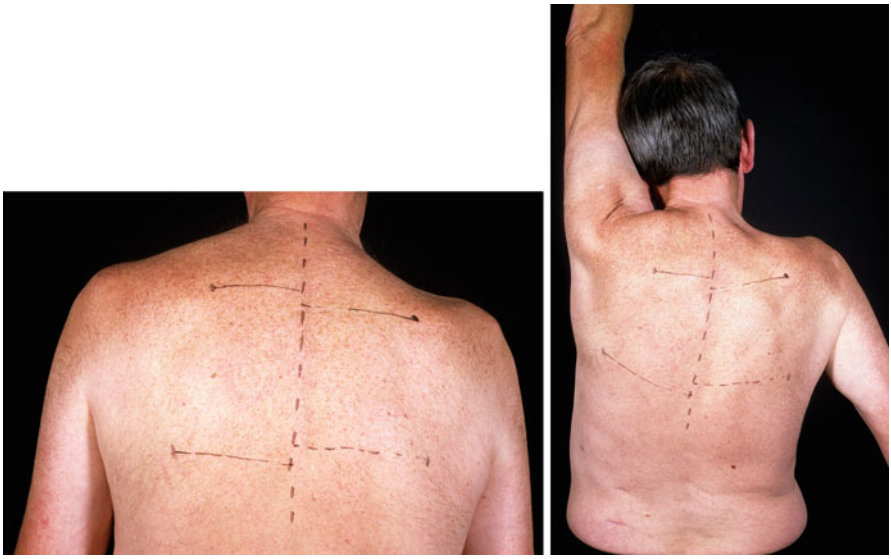

Fig. 4.33 Right spinal accessory palsy. The scapula drops down and away from the spine. The active ISHA is 30°

The nerve to serratus anterior is a frequent victim of the attentions of surgeons and transection is associated with pain and loss of function only slightly less than that seen after accessory palsy. The nerve is particularly susceptible to involvement in neuralgic amyotrophy. The *active* ISHA is, on average, 130°. This is the only nerve lesion in which the active ISHA actually exceeds the total range of abduction. The scapula is elevated and approaches the spine (Fig. 4.35).

The circumflex and suprascapular nerves: the rotator cuff.

Recognition of rupture of the circumflex nerve can be very difficult. One reason for this is the widely held (and erroneous) view that the deltoid muscle is the abductor of the gleno humeral joint (Figs. 4.36, 4.37, and 4.38). Wynn Parry [19] examined 145 patients with paralysis confined to the deltoid muscle. He found that the range of abduction was full, or nearly so, and described a system of training compensatory movements which enabled most of his patients to return to full military duties: "it must be stressed that these movements providing full abduction and elevation are not trick actions in the sense usually associated with this word; all the muscles involved normally help to abduct the shoulder. The scapulo-humeral rhythm is quite normal and in the later stages of re-education the patient does not even need to rotate the humerus externally to initiate the movement". Seddon [14] was a little more cautious: "this perfect abductor action of the supraspinatus is rare; it is more usual to find abduction to about 155°, with the arm a little in front of the coronal plane of the body". Curiously, the loss of abduction caused by lesions of the suprascapular nerve and/or of rupture of the rotator cuff is frequently and wrongly attributed to a lesion of the circumflex nerve (Figs. 4.39 and 4.40). In 63 cases the active ISHA was diminished by about 20° in uncomplicated ruptures of the circumflex

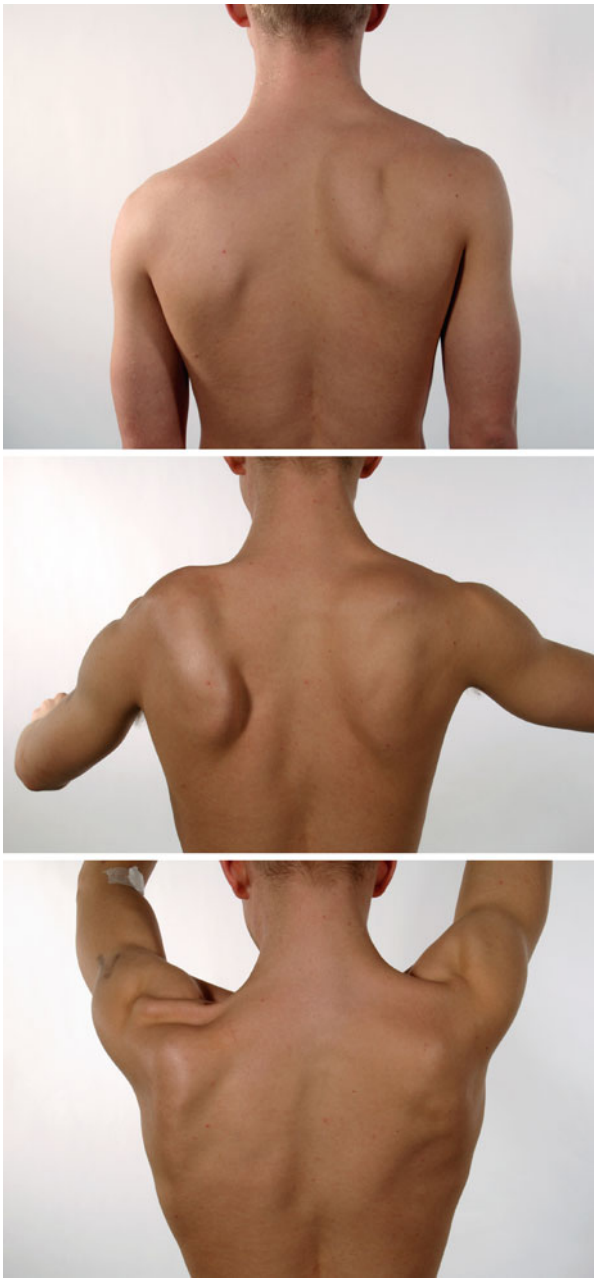

Fig. 4.34 Left spinal accessory palsy. Scapular winging, without prominence of the lower fibres of the trapezius, in a case where there is some early recovery into the upper fibres after repair of the spinal accessory nerve. At rest, the scapula is displaced downwards and away from the spine

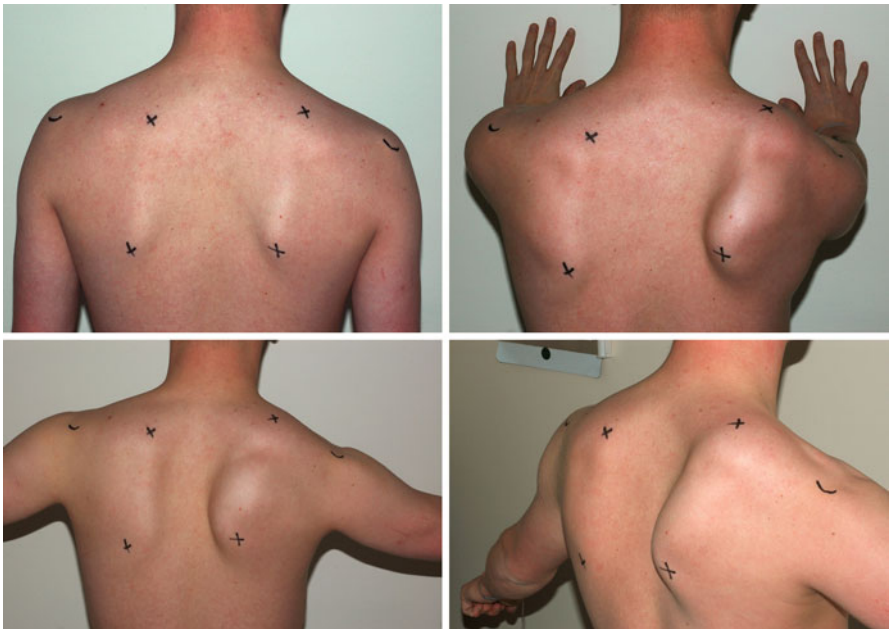

Fig. 4.35 Scapular winging in nerve to serratus anterior lesion. This is easily distinguishable from the winging provoked by accessory palsy by the position of the scapula which is drawn upwards and towards the spine by the unopposed action of trapezius, levator scapulae, and rhomboids

when there was no stiffness of the shoulder [1]. The angle is reduced to less than 30° in most cases of suprascapular palsy or in complete ruptures of the rotator cuff (78 cases) [1]. Perhaps the most reliable sign of circumflex palsy is weakness of extension. The power of extension at the shoulder abducted to 90° is as little as 5–10 % of normal when the deltoid is paralysed. The diagnosis of rupture of the circumflex nerve is only easy when it is too late to do anything about it, that is, when the atrophy of the muscle is all too plain. It is a very hard matter for the clinician treating a patient with fracture/dislocation of the shoulder to examine function in the muscles. The area of loss of sensation is inconsistent and some patients will describe sensation of the skin over the muscle as abnormal rather than absent. In the example of the patient after successful reduction of a dislocated shoulder or fracture the arm will be supported in a sling and three simple tests can be done:

- Initiation of abduction indicates that the suprascapular nerve is working and that the rotator cuff is not ruptured;
- *Abnormal* sensibility in the skin over the deltoid indicates a lesion of the circumflex nerve;
- The patient is able gently to extend the shoulder supported as it is in a sling enabling the examiner to palpate activity in the posterior deltoid.

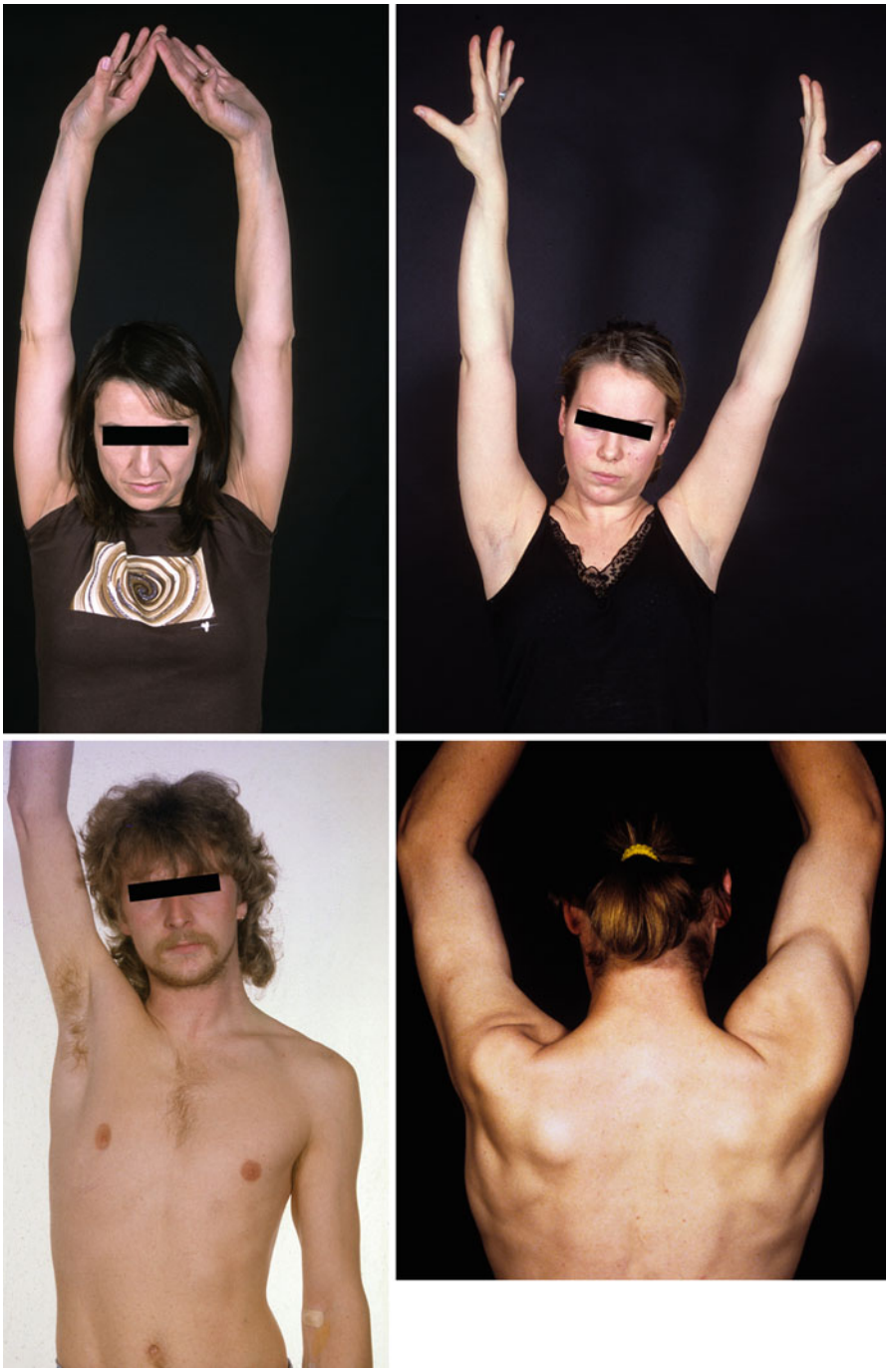

Fig. 4.36 Elevation of the upper limb in full medial rotation by the supraspinatus in the absence of deltoid in four cases of proven rupture of the circumflex nerve. *Bottom left* and *right*: note the activity in the clavicular head of pectoralis major

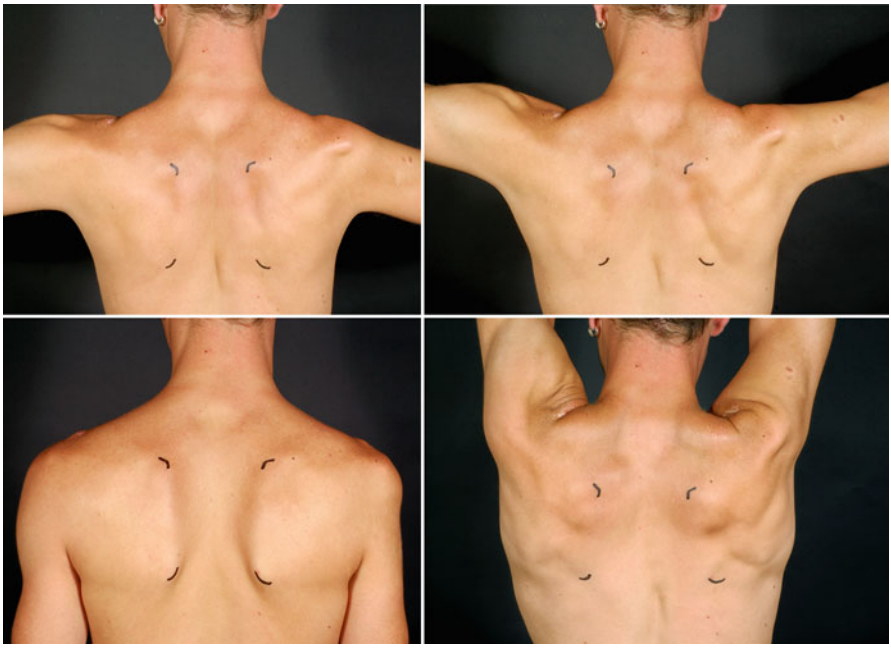

Fig. 4.37 The movement of the scapula in another case of rupture of the right circumflex nerve. The active ISHA on the side of the injury (*bottom right*) is reduced by 20° showing that 20° of the range of elevation is provided by extra movement at the thoraco-scapular joint

4.8.2 The Hand

When the median nerve has been cut the opposing action of the thenar muscles can be mimicked by the combined action of an ulnar-innervated flexor brevis and the abductor longus muscle. Comparison with the intact side will usually show that this combined action does not reproduce the rotational action of the opponens. Similarly, the abducting action of the abductor brevis can in the absence of median nerve function be imitated by the action of the abductor longus muscle. These points are important in the early stages when there is no wasting to guide the examiner.

Loss of the intrinsic muscles innervated by the ulnar nerve causes abnormality of pinch grip and imbalance between the long extensor and flexor muscles. The clawing is worst in the little and ring fingers (Fig. 4.41). The power of the ulnar-innervated muscles of the hand may be tested by examining the power of abduction and adduction of the fingers. The ease with which a sheet of paper may be pulled from between two adducted fingers gives some indication of this power.

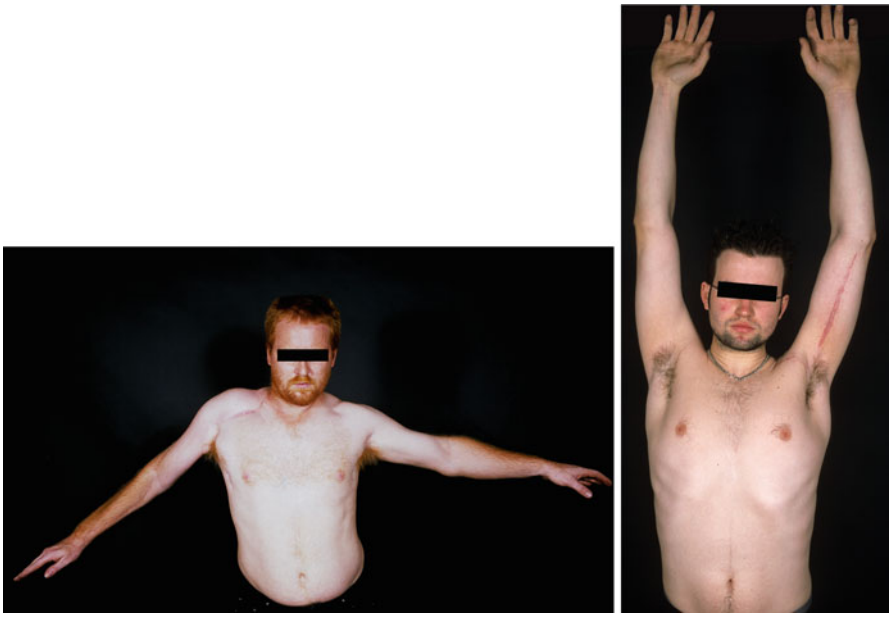

Fig. 4.38 Combined injuries to the suprascapular and circumflex nerves. *Left*: showing the range of elevation in a patient with irreparable injury to the right suprascapular nerve but with a good result after repair of the circumflex nerve. *Right*: showing the elevation in another patient in whom repair of the left suprascapular nerve was successful but whose circumflex nerve injury was irreparable

4.8.3 The Lower Limb

There should be no great difficulty in testing the muscles connecting the pelvis to the femur in the healthy subject, but things are different when this has to be done soon after replacement arthroplasty. It appears that in this situation there is also quite often a certain reluctance to look. In one case, a "drop foot" was observed soon after arthroplasty, but it was not until a year later that another examiner found paralysis of most of the muscles of the buttock. Superior gluteal palsy is crippling, yet delay in diagnosis is common. Much can be learnt from watching the patient stand and walk. The integrity of the smaller glutei is tested with the patient standing or lying supine; that of the rotators of the hip with the patient seated, and that of the gluteus maximus with the patient prone. It was common experience at times when poliomyelitis was common to see children and young adults walking quite well even though their quadriceps muscles were paralysed. They did this by a form of adaptation, a substitution movement, in which the tensor fascia lata was responsible for stabilisation of the knee. In many cases there was the added factor of a hyperextension deformity of the knee. It is, however, quite wrong to assume that an adult with a deep femoral nerve lesion could walk comfortably and without risks. In six of our cases of femoral palsies incurred during total hip arthroplasty, the diagnosis

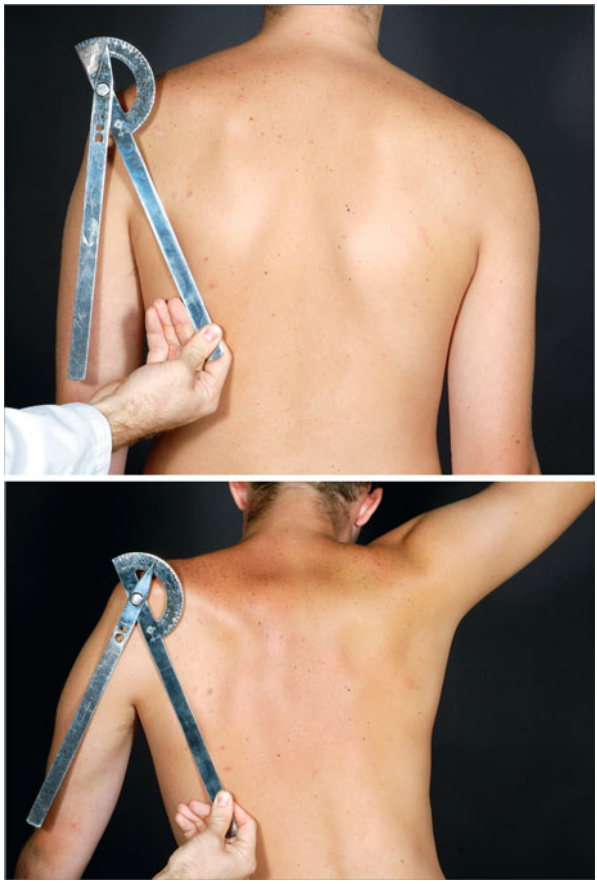

Fig. 4.39 Initiation of abduction, with opening of the active ISHA was the first sign of recovery into supraspinatus after repair of the suprascapular nerve. The lesion of the circumflex nerve was irreparable

was recognised only after the patients fell and damaged themselves. A lesion of the femoral nerve high enough to paralyse both hip flexors and extensor muscles of the knee is crippling.

4.9 Late Signs of Nerve Injury

Two weeks after a complete degenerative lesion, the area of loss of sensibility is well defined; the beginning of wasting indicates the extent of the motor affection. Anhidrosis is still present, but with the degeneration of peripheral fibres the warm isothermia of the skin gives way to poikilothermia and later to cold isothermia (Figs. 4.42).

As time goes by, the changes of disuse appear: thinning of the skin; even ulceration from accidental injury; loss of substance in the tips of the digits; loss of skin markings; constant coldness and cyanosis; stiffness of joints; contractures;

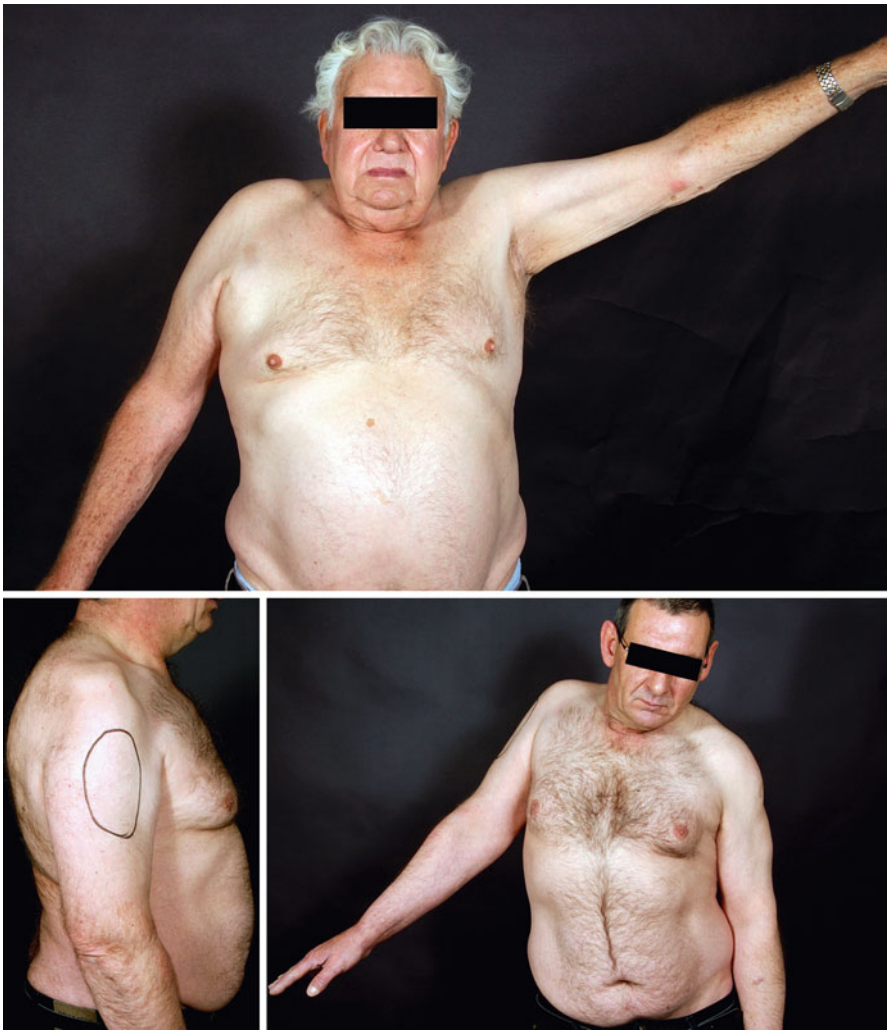

Fig. 4.40 Rupture of rotator cuff with lesions of the suprascapular and circumflex nerves from fracture/dislocation of the shoulder. *Above* showing the range of elevation at the right shoulder in 74 year old ex-paratrooper in whom there was clear evidence of recovery for both of the nerves. *Below*: this shipwright held onto a cable to rescue a man from the Thames. The weight of the man and the force of the current was such that he felt the muscles tearing in his right shoulder, then he felt the head of the humerus pulling out from the socket and then his arm went dead. Rupture of the rotator cuff was confirmed by MR scan. Electromyography showed that the suprascapular nerve was intact and that there was, at 8 weeks, reinnervation of the posterior deltoid. A Tinel sign was detectable at the posterior aspect of the shoulder. A subsequent repair of the rotator cuff was successful

unmistakable wasting. Nails become brittle and discoloured and are prone to infection. Hair growth is disturbed, hairs are often coarse (Figs. 4.43 and 4.44). These changes occur more rapidly in the ischaemic limb. Prolonged denervation of a growing limb

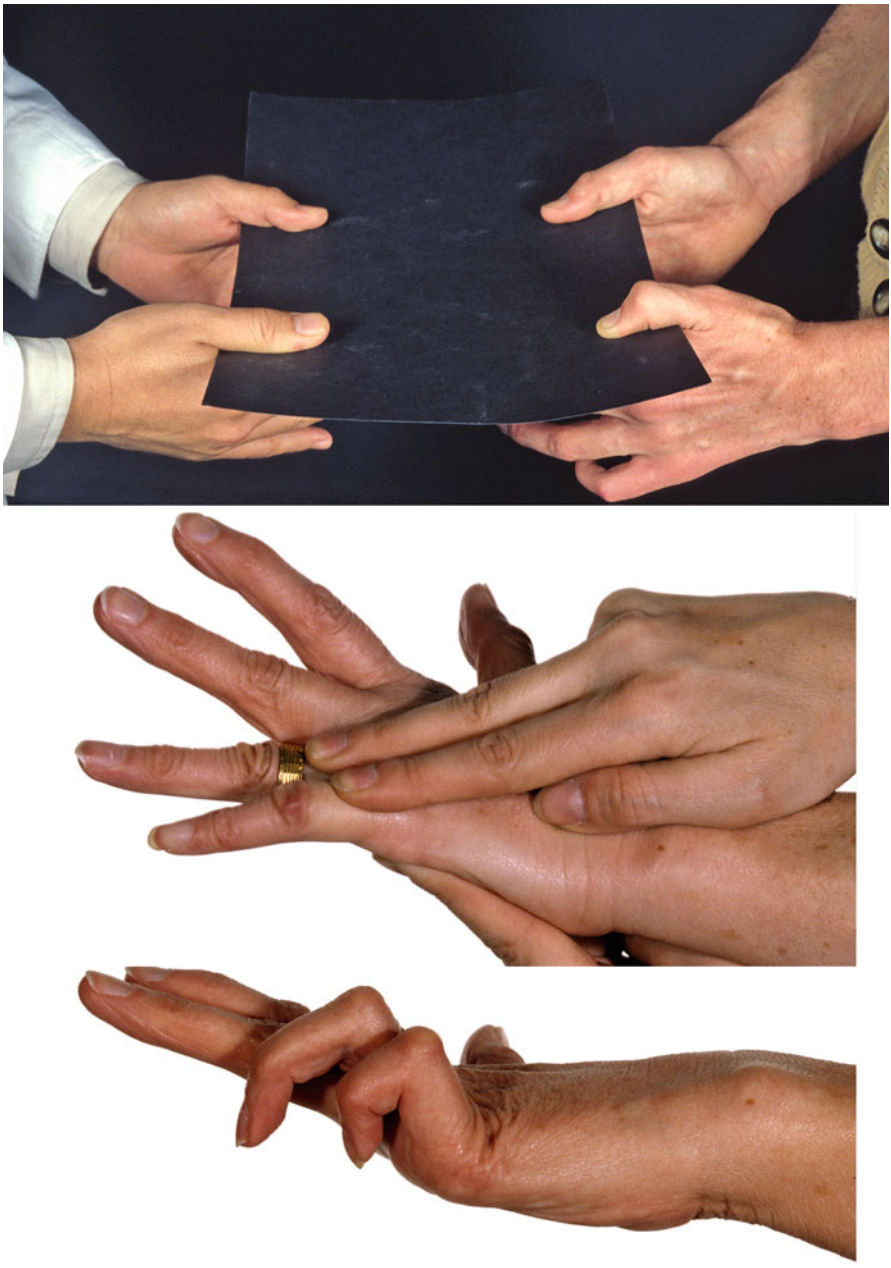

Fig. 4.41 The small muscles of the hand in lesions of the ulnar nerve. *Above*: "Froment's sign" is positive in the patient's left hand. *Below*: clawing is corrected by passive flexion at the metacarpophalangeal joints

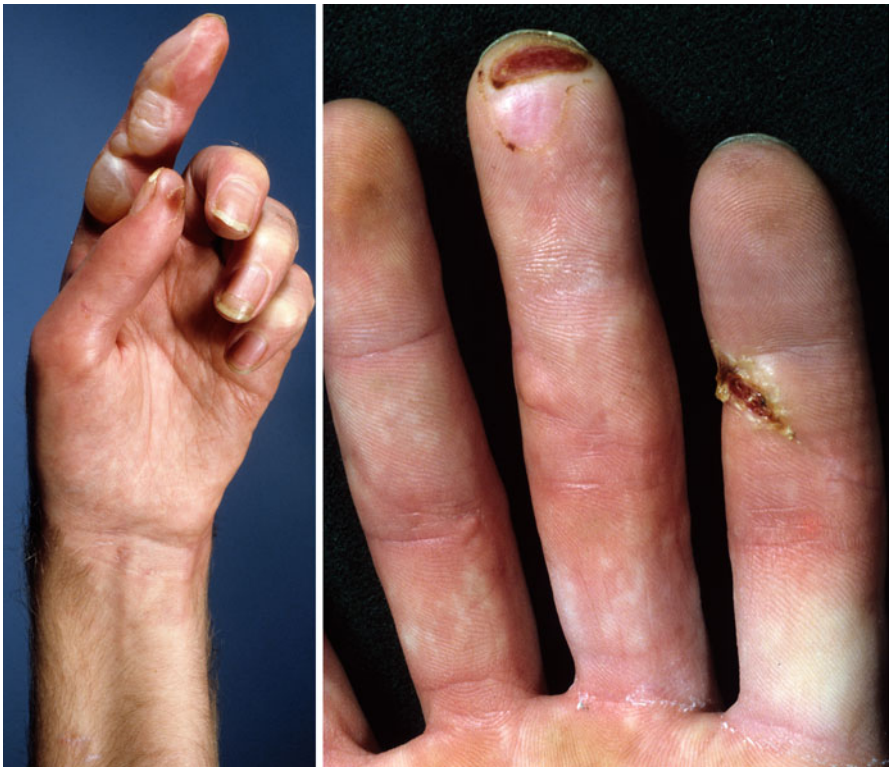

Fig. 4.42 Late changes after nerve injury. *Left*: there is sympathetic paralysis and unnoted burns after high lesion of the median nerve. *Right*: wasting and ulceration of the skin of middle finger and accidental injury to index finger after median nerve injury. The patient was working as a stone mason

leads to defective growth: this is of course well seen after birth injury of the brachial plexus (Figs. 4.45 and 4.46). In cases of greatly prolonged conduction block, the changes are always far less than they are in degenerative lesions. In degenerative lesions profound changes take place in both motor and sensory end-organs. The distal axon normally maintains a dense population of end-organs in skin and sweat glands and in the muscular component of arterioles. It is hard to resist the conclusion that the changes of "disuse" are at least in part due to the loss of distal axons, their end-organs, and to the effects of that loss on target tissues.

By the time the changes of degeneration are present, the patient is a better candidate for the examination halls than for restorative treatment. The object of the clinician must be to make the diagnosis before the signs of peripheral degeneration have appeared; before the best time for intervention has passed. Unfortunately, the peripheral neurologist is still likely to be presented with cases in which delay in diagnosis has permitted the development of these signs. The last are at this stage well marked; their absence in association with persistent partial motor and sensory paralysis almost certainly means that the lesion is partly or wholly a conduction block.

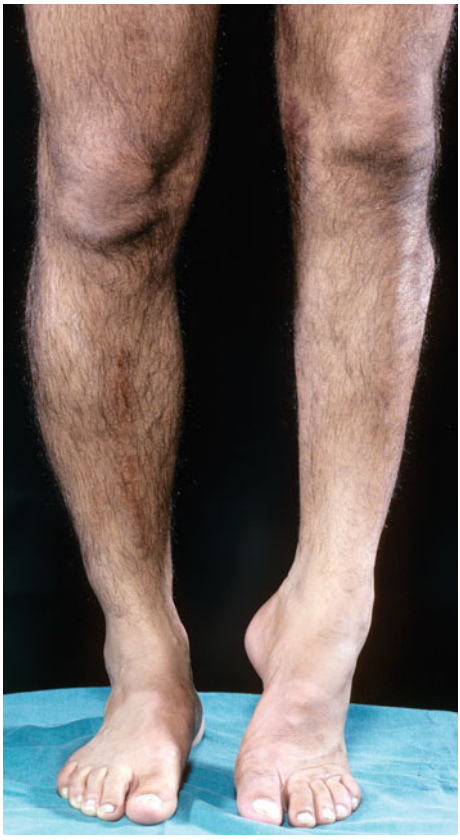

Fig. 4.43 Post ischaemic fibrosis. The lateral geniculate artery was lacerated during arthroscopic meniscectomy. The false aneurysm which ensued remained undetected for 4 days in spite of his intense causalgia. The position of the leg, ankle and foot 3 years after injury

4.10 The Diagnosis of Neuropathic Pain After Injury to a Nerve

Severe neuropathic pain is a common complication of injuries to peripheral nerves especially in those caused by surgeons or anaesthetists. Diagnosis rests on a careful history, and gentle accuracy during examination. Precision in classifying symptoms and signs and in the use of terms is essential [3].

- Nociceptors are those neural structures which detect the existence of a noxious event: nociceptive pathways or tracts inform the mind – brain of the event which may there be perceived as pain.
- Paraesthesiae – spontaneous abnormal sensations
- Dysaesthesiae – spontaneous, unpleasant abnormal sensations
- Hyperalgesia – increased perception of a stimulus which is normally painful
- Allodynia – the perception of a stimulus which is not normally painful as a painful event.
- Hyperpathia – a state of exaggerated, prolonged and very painful perception of stimulation.

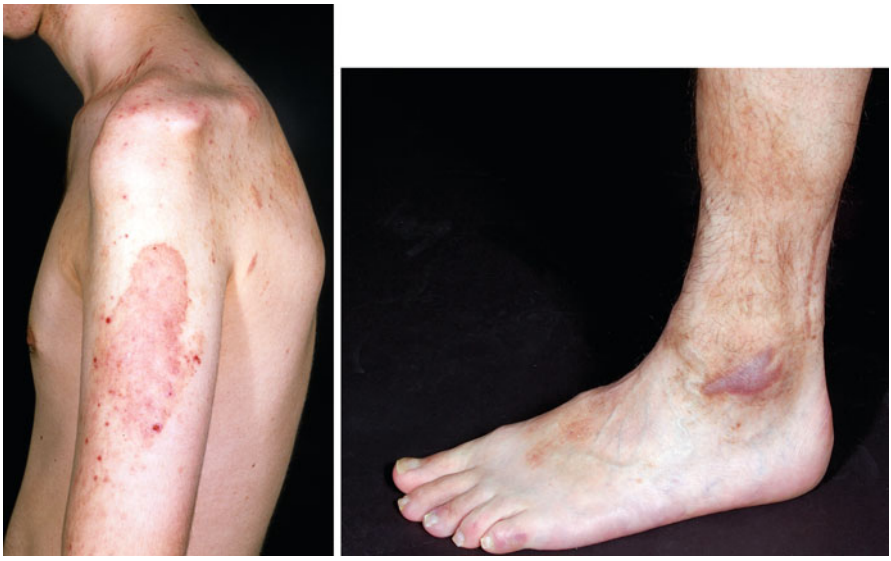

Fig. 4.44 Late skin changes after nerve injury. *Left*: a skin rash in the distribution of C5, 6 months after rupture. *Right*: skin rash in the distribution of the common peroneal nerve 1 year after rupture at the knee

It is important to distinguish between the spontaneous symptoms of paraesthesiae and dysaesthesiae, which arise from injured axons without external stimulation from evoked symptoms such as allodynia, which signify that fast conducting mechanoreceptor fibres are conveying impulses which are being interpreted as pain. The spread of spontaneous and evoked sensory symptoms beyond the distribution of the injured nerves indicates that there is *central sensitisation* involving other neurones in the dorsal horn (Fig. 4.47).

There are obvious examples in everyday practice of these different types of sensory disturbance. Patients with entrapment or other irritative lesions of peripheral nerves volunteer sensations of cold water or (for the select) cold champagne trickling down underneath the skin. These are paraesthesiae. Sometimes these spontaneous sensations have an unpleasant quality, they are described as if there are ants crawling under the skin. These are dysaesthesiae. The patient who cannot tolerate light touch on the afflicted skin is describing allodynia. More severe injuries to proximal nerves brings on a state of constant racking pain, often described as burning; the part cannot be examined and moved only with difficulty. This is hyperpathia.

4.10.1 Allodynia

Allodynia is one of the most important of clinical signs in medicine and it must be sought for and interpreted with precision. It is evoked by applying stimuli which are

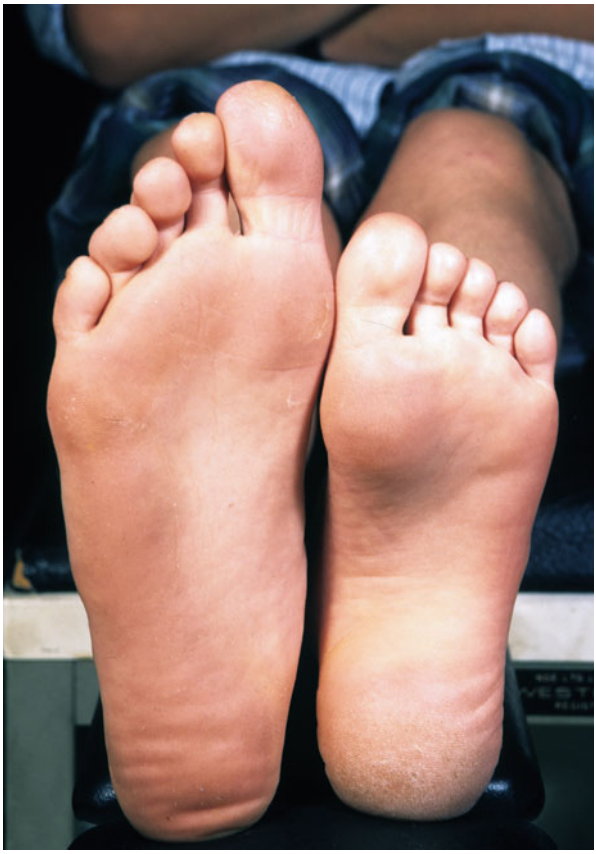

Fig. 4.45 Atrophy of the left foot in an 11 year old boy 4 years after transection of the tibial and common peroneal nerves at the knee. The repair of the common peroneal nerve was successful, that of the tibial nerve failed

normally not painful but which the patient interprets as pain and it can be found only when there is some innervation remaining in the skin. Allodynia signifies that mechano receptor and other fibres have begun to signal pain because of events at their terminals, in their parent cell bodies, and in the second order neurones of the dorsal horn. Although it may seem reasonable to use the term for the overreaction so often seen in the earlier stages of regeneration after nerve repair it is more precisely restricted to pain induced by gentle stimulation of the skin after injury to a nerve. Allodynia maybe dynamic, when it is elicited by moving touch, by a draught or breeze, or by contact with a sheet or clothing or it may be static when it is elicited by pressure. There is also warm or cool allodynia, when a normally non painful warm or cool stimulus is perceived as pain. There may be a paradoxical interpretation of a cool stimulus as one which is painfully hot or vice versa. The extension of allodynia beyond the distribution of the injured nerve is common, another example of *central sensitisation*.

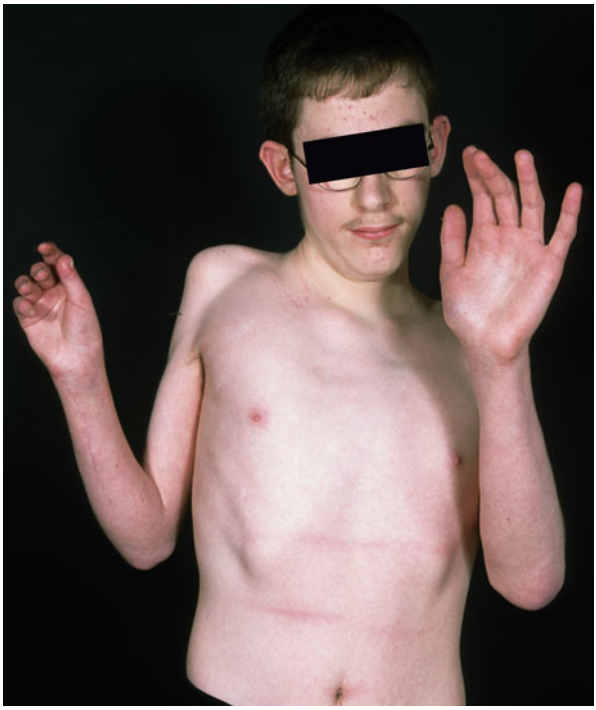

Fig. 4.46 Bilateral lesion from breech delivery. On the right – the lesion was complete and complicated by phrenic nerve palsy. Recovery in C5 and C6 was poor. At the age of 7 years, accessory to suprascapular nerve transfer restored some lateral rotation. On the left there was avulsion of C5 and rupture of C6. Transfer of latissimus dorsi at the age of 4 failed. The discrepancy in growth is particularly severe in the forearm and hand

4.10.2 Hyperpathia

Hyperpathia is the deep seated, burning and poorly localised pain extending beyond the distribution of the injured nerve, evoked by the palpation of the muscles of the limb it is common in ischaemia. Hyperpathia is analogous to cutaneous allodynia, but one which involves the deep afferent pathways.

4.10.3 Deafferentation Pain

Deafferentation pain is used when the injury has interrupted the pathway between cell bodies in the dorsal root ganglion and those in the dorsal horn. After all, any lesion severe enough to inflict interruption upon the axons must lead to deafferentation and to use the term in these situations renders it virtually meaningless. This pain is pathognomonic of intradural, preganglionic injuries to the brachial or lumbo sacral plexuses.

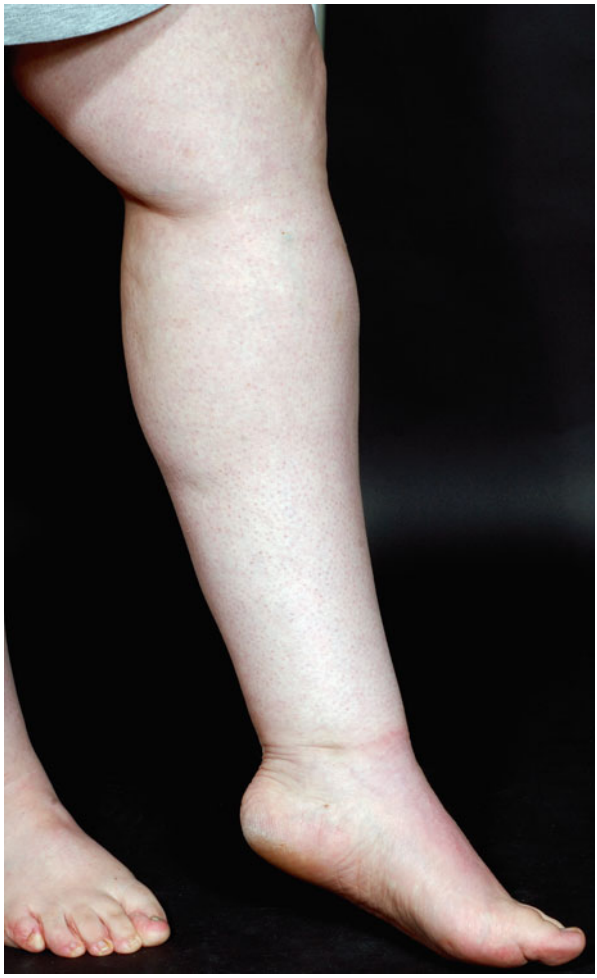

Fig. 4.47 Extreme central sensitisation in post traumatic neuralgia. Intra operative incomplete section of the medial plantar nerve in a 51 year old woman. She was treated for many months by drugs and blocks for an incorrect diagnosis of CRPS1. By 3 years she could not walk because of intense allodynia in the leg and foot, where there was profuse sweating and discolouration. She experienced great improvement after repair of the nerve combined with local anaesthetic blockade of the tibial nerve which was maintained for 3 days through an indwelling catheter

4.10.4 Clinical Assessment

The history is essential and the patient must be given ample time to tell their story. In late cases there may be diffidence about expressing symptoms which seem so bizarre and which may already have been dismissed by others. It may prove necessary to put leading questions which indicate that the clinician is indeed listening to the patient, believes what they are saying and has an understanding of what is being said. Certain features are particularly important.

- Onset. The immediate onset of pain after a wound or on awakening from an operation implies that the lesion has already been inflicted whereas delayed onset suggests a later event such as haematoma.

- Distribution. The patient is asked where the pain started and where it went to. Did the pain spread beyond an earlier well defined area and if so, over what period of time?
- Qualities. Was the pain there all the time, was it episodic, was it constant or intermittent? Many different terms are used, burning, bursting, crushing, compressing, "the hand in a vice", "the bones of the foot are coming out of my skin", "a hot needle or a hot file rasping on the skin" are common descriptions. Was the pain on the surface or was it deep? Episodic or convulsive pain is often described as lightning like, electrical, shooting or lancinating.
- Aggravating and relieving factors. Many patients give a clear description of the phenomenon of allodynia. One which is commonly related is the increasing intolerance of a sheet on the leg and the foot after compression of the sciatic nerve by haematoma in the thigh or buttock. The effect of changes in temperature or the weather or of associated illness are important and frequently described features.
- The effect of the pain upon life, upon work or study, on social activities and on sleep provide an insight into the severity of the pain.

Examination must be done with gentleness, and in some patients no more than inspection is possible. Important features include trophic changes, vaso and sudomotor abnormality, the posture of the part, and the presence of spontaneous movements. After this allodynia, in its various forms, must be sought before deep palpation of the muscle compartments. Tinel's sign is sought. There are three characteristics of pain caused by injury to a nerve which are extremely important for the clinician. These are:

- dysaesthesiae,
- allodynia, and
- Tinel's sign.

With the evidence provided so far the clinician ought to be able to arrive at an accurate diagnosis about what nerve has been injured, where it was injured, and have a view about the cause of that injury and of the underlying mechanisms.

4.10.5 Neuropathic Pain Syndromes

There are four main syndromes [3].

1. *Causalgia* is usually caused by high partial injury to the median, ulnar or tibial nerves or the trunks of origin, with an associated arterial injury. Pain is spontaneous, persistent, often with a burning feeling extending throughout the limb and is worsened by physical and emotional stimuli. There is excessive sweating and vasomotor disturbance. Allodynia and hyperpathia are intense (Fig. 4.48).

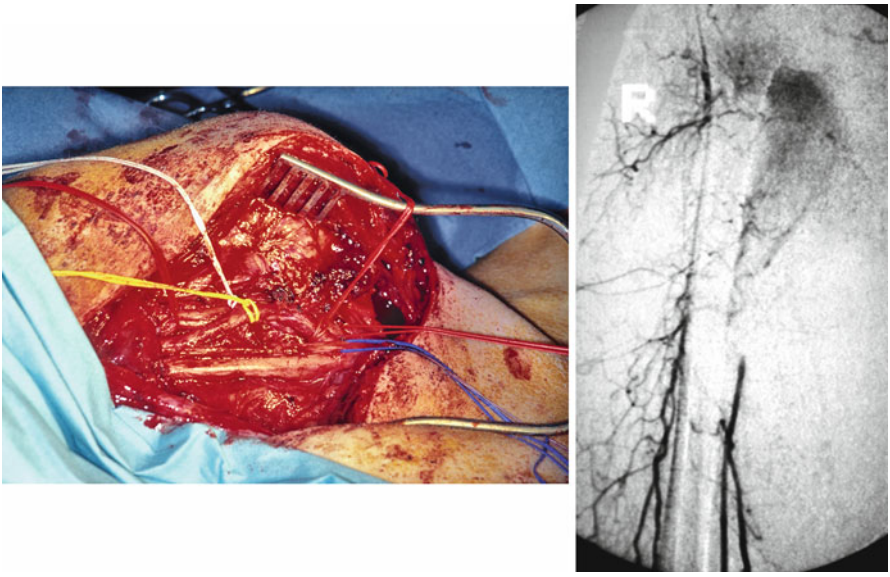

Fig. 4.48 Causalgia in a 55 year old woman after removal of a lipoma in the axilla. At operation, 3 weeks later, an iatrogenous false aneurysm of the brachial artery was displayed. The median and ulnar nerves were displaced and compressed. Her pain was relieved by correction of the aneurysm, decompression of the nerves which were blocked by local anaesthetic for 3 days through an indwelling catheter

2. *Neurostenalgia* is caused by persistent tethering, compression, distortion or ischaemia of an intact or repaired nerve. Pain is confined to the distribution of the nerve: sympathetic over activity is rare.
3. *Post traumatic neuralgia.* Pain is initially confined to the territory of the nerve without florid sympathetic overactivity. Dysaesthesiae and lancinating pain are usual in nerves with a cutaneous component, whereas pain is deep, boring and poorly localised from nerves passing to muscles.

   A strong, painful Tinel's sign is almost always present at the level of lesion in these three syndromes. It is absent in the fourth, deafferentation pain.
4. *Deafferentation pain in preganglionic injury to the brachial or lumbo sacral plexusus*. Most patients experience characteristic, indeed pathognomonic pain, within 24 h of injury [2]. There is, first, a constant crushing bursting or burning pain felt in the anaesthetic areas: next, there is superimposed convulsive, lightning like shooting pains which are felt within the dermatomes of the avulsed spinal nerves (Fig. 4.49).

Willner and Low [18] set out some principles governing the treatment of neuropathic pain. They include:

1. Removal of the cause.
2. Promotion of healing or regeneration.
3. Correction of the microenvironment of the nerve.

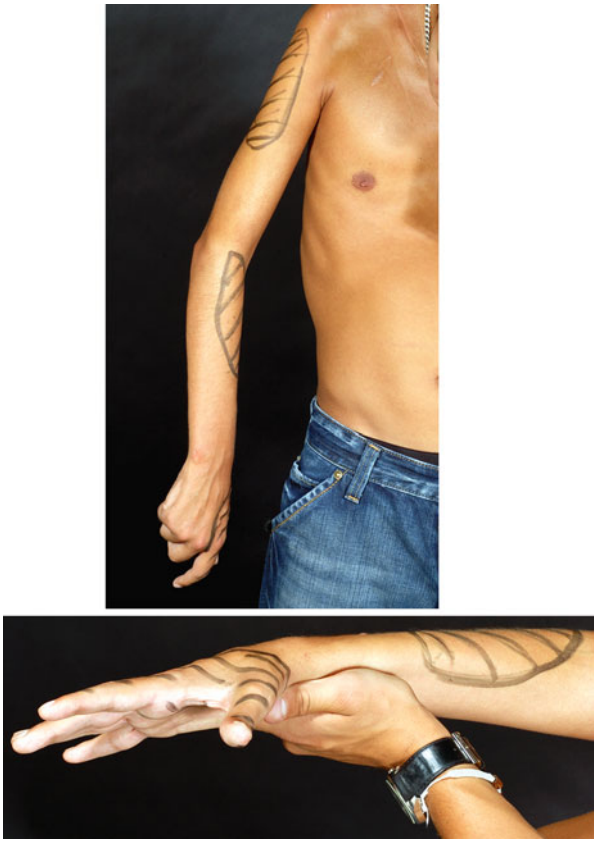

Fig. 4.49 Intense shooting, lightning like pain was felt in the dermatomes of C5, C6 and C7 in this patient on the day of injury. These three nerves were avulsed from the spinal cord in a motor cycle accident

4. Restoration of afferent pathways.
5. Modulation of central inhibitory pathways.
6. Reduction of sympathetic over activity, and
7. Changing pain thresholds by modification of emotional or behavioural components of pain interpretation. These are excellent principles. Neuropathic pain following a focal injury of a nerve will require operation in 1, 2, 3, 4, and, sometimes, 6 whereas drugs and other measures short of operation, have a part to play in the last three.

4.11 Aids to Diagnosis

4.11.1 Neurophysiological Investigations (NPI)

Neurophysiological examination is certainly the foremost aid to diagnosis, though in the acute stage the process is often hampered by pain and by local conditions. It

must be done properly and results must expertly be interpreted. It is no substitute for clinical observation; it must not be used as device for deferring decision and delaying action. The reader is referred to the comprehensive discussion by Smith and Knight [15] who emphasise that: "the term EMG is often used colloquially to refer to electrodiagnostic studies that incorporate nerve conduction with or without electromyography and other investigative techniques. This is misleading and should be avoided. Nerve conduction studies and electromyography, whilst inter related, are distinct procedures". NPI in nerve injury is helpful in :

- Localisation of the lesion.
- Determination of pathophysiology.
- Detection of conduction *across* the lesion, at any time after injury which shows that some fibres are intact and working.
- Establishing severity of lesion.
- Identification of reinnervation.
- The extension of the process to the detection and measurement of potentials evoked from the cortex provides valuable evidence in the case of suspected avulsion of the roots of the brachial plexus.
- Perhaps the simplest, yet often neglected, technique of electrophysiological examination is that of stimulating the nerve below the level of the lesion and observing the motor response. If, 3 days after injury, stimulation below the level of the lesion produces a normal response in the muscles supplied by the nerve, the odds are that the lesion is a conduction block. If there is no motor response or if the response is much subdued, then the lesion is degenerative.

The introduction of NPI during operation has brought massive advantages, in particular in:

- determining neural continuity across a lesion in continuity;
- determining the site of a conduction block;
- determining which part of a nerve has suffered axonal interruption;
- determining whether an apparently intact component of the brachial plexus has intact central connections.

4.11.2 High Resolution Ultrasonography

It seems likely that ultrasonography, in skilled hands, has great potential in the early detection of ruptures or other serious injuries to nerves. A number of orthopaedic and fracture surgeons are already well versed in the technique and it is probable that the very real difficulty of recognition of rupture of the nerve trunk in a closed fracture will be overcome by the widespread use of this method by interested clinicians. Cokluk and Aydin [5, 6] examined 58 patients using a Tosbee ultrasound (Toshiba Inc. Tokyo) with a 5–7.5 MHz linear probe. The patients with injuries in the upper limb were placed supine. Ultrasound gel was plastered on the probe surface and the

skin to enhance visualisation of peripheral nerves and the musculo-skeletal structures. The examination commenced about 10 cm proximal to the suspected region and continued 10 cm distally. Bone, muscles, tendons, vascular structures, and peripheral nerves were identified and distinguished: "Continuity, architecture, shape, calibration and integrity of the involved nerve and peripheral tissues were examined in the perpendicular and transverse planes". The femoral nerve was examined with the patient supine, the sciatic nerve was examined with the patient placed prone.

Sixteen of these patients were examined within 3 days of injury. In most cases the diagnosis made by ultrasound was matched with the findings at subsequent operation. The investigation proved reliable in identifying the nerve, in localising the level of injury and in the recognition of the nature of that injury. Toros et al. [16] provide further valuable information about the technique.

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Chapter 5
Operating on Peripheral Nerves

5.1 Indications and Objects of Intervention

An experimental study is one: "in which the investigator intervenes in some way to affect the outcome. Such studies are longitudinal and prospective; the investigator applies the intervention and observes the outcome some time later" Petrie [23].

Operation is indicated to:

- to confirm or establish diagnosis;
- to restore continuity to a severed or ruptured nerve;
- to remove a noxious agent compressing or distorting or occupying a nerve.

It is difficult to overstate the significance of worsening of pain and deepening of nerve lesion caused by expanding haematoma or ischaemia. Clinicians must never forget that nerves compressed in a swollen ischaemic limb or in a tense compartment progress from conduction block to much deeper and much less favourable degenerative lesions.

Pain persisting after a focal injury to a nerve is an indication for operation at almost any interval after injury [4]. In the remarkable case described by Camp, Milano and Sinisi [7], the patient suffered intractable and increasing pain for 18 years. Pain was abolished by neurolysis of the ulnar nerve which had become adherent to the pulsatile vein graft used to repair the brachial artery. That lesion was a conduction block, prolonged by external causes: the pain was neurostenalgia.

5.1.1 The Lessons of War

Surgeons at the London Hospital firmly advocated urgent (primary) over delayed (secondary) suture in the decades before the First World War. During that conflict the principles governing the repair of war nerve wounds were established. These include: the proper treatment of the wound by debridement, excision, and delayed

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closure; excision of scar until a healthy bed is secured; excision of damaged nerve until healthy stumps are reached; and tension free suture by adequate mobilisation and flexion of adjacent joints or grafting. The imperative of rehabilitation was emphasised [3].

The work of the Medical Research Council Nerve Injuries Committee during the Second World War marked a most important stage in improving understanding of regeneration and of applying those lessons to clinical practice. Nerve grafting in all its forms was studied. The first evidence of axonal transport was revealed. The rate of nerve regeneration, the effects of disuse, the regeneration of proprioceptors, the maturation of regenerated fibres and the significance of retrograde influence were amongst the fields of enquiry [3].

Modern war wounds reinforce earlier and costly lessons but also provide new ones [6]. Most wounds are caused by explosive devices; they are multiple and complex. Avulsion, laceration, blast and crush combine to tear and shred tissues of all compartments. Heavy contamination with dirt and debris is usual and meticulous debridement is the essential first step in time-limited resuscitative surgery. The field hospital policy of emergency restoration of arterial flow and extensive decompression led to a remarkably low incidence of ischaemic fibrosis; the sole case occurred in a patient in whom the brachial artery was ligated.

The severity of damage at the level of nerve lesion is probably unparalleled; fracture in 50 %, arterial injury in 32 %, moderate or severe muscle loss in 28 % and moderate or severe skin loss in 50 %. Two of more nerves were injured in 70 % of patients. Such massive damage to the skin and muscle is inimical to nerve regeneration and function. The resultant fibrosis is an important cause of pain and loss of function. Thirty six patients came to secondary operations because of persisting severe neuropathic pain. Thirty experienced such relief after operation that analgesic medication was considerably reduced or abandoned. Some patients experienced relief when they awoke from the anaesthetic. The operations included six revision repairs, 11 neurolyses of repaired nerves, and neurolysis in the other 19 patients. The causes of persisting pain included displaced bone fragments, heterotopic bone, retained metallic fragments or suture material, and most commonly, scar tissue which enveloped and constricted the nerve. Resurfacing by free fascio cutaneous flaps were used in 15 patients, to relieve pain and enhance nerve regeneration. No case of false aneurysm or arterio-venous fistula was encountered.

5.1.2 Timing: Nerve Lesions in Fractures and Dislocations

Some important practical advantages of urgent exploration include the ease of recognising a rupture and the ease with which the stumps can be approximated. The best time to explore such injuries is before distal conduction is lost (Figs. 5.1, 5.2 and 5.3). Primary repair is defined as one performed within 5 days of injury and delayed primary repair for one completed at intervals from 5 days to 3 weeks after

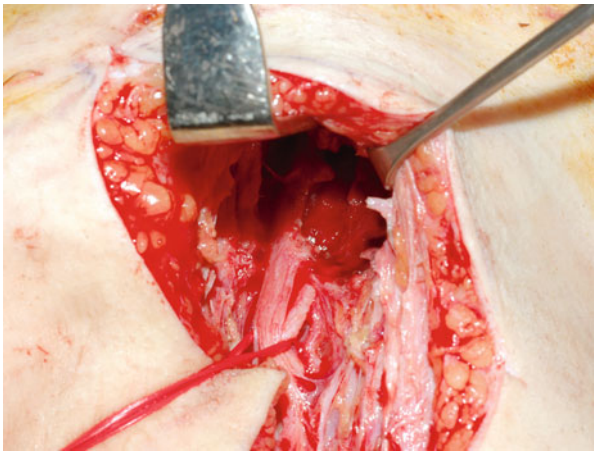


Fig. 5.1 Ease of diagnosis at operation done soon after injury. Incomplete transection of the femoral nerve occurred during a difficult pelvic operation. The surgeon recognised the event and enabled display and repair 24 h after wounding

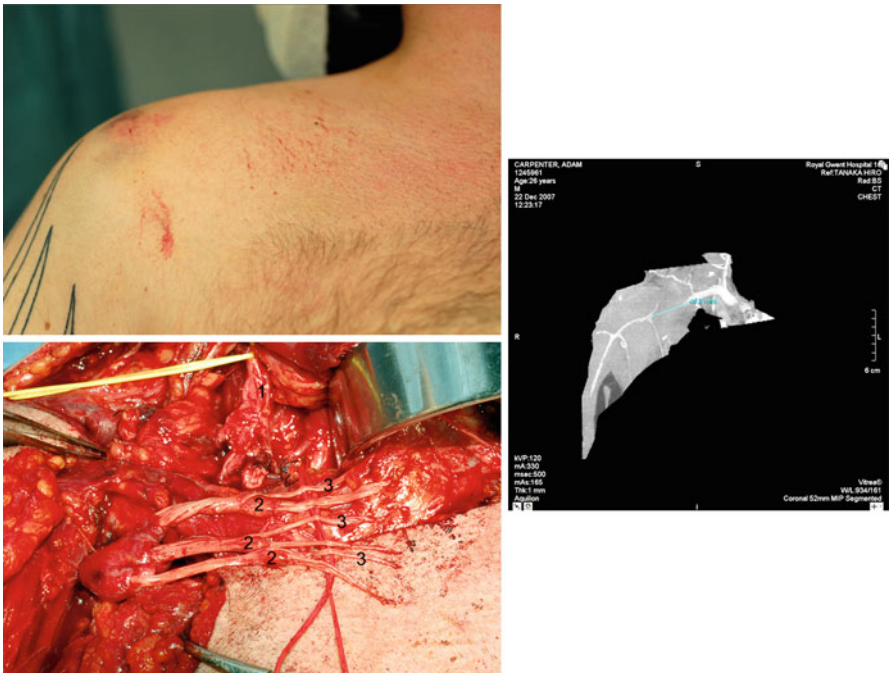


Fig. 5.2 Ischaemia and conduction. Traction lesion of the brachial plexus was accompanied by rupture of the subclavian artery. There was a weak pulse. At operation, 54 h after injury, stimulation of the avulsed ventral roots of C7, C8 and T1 evoked strong contraction in the relevant muscles distally. This showed that there was neither critical ischaemia within the limb nor that there was a second, more distal, lesion. Strong SSEP's were recorded from the stumps of C5 and C6 (*1*). The dorsal root ganglia of C7, C8 and T1 (*2*) and their ventral roots (*3*) are shown. An extensive repair was done

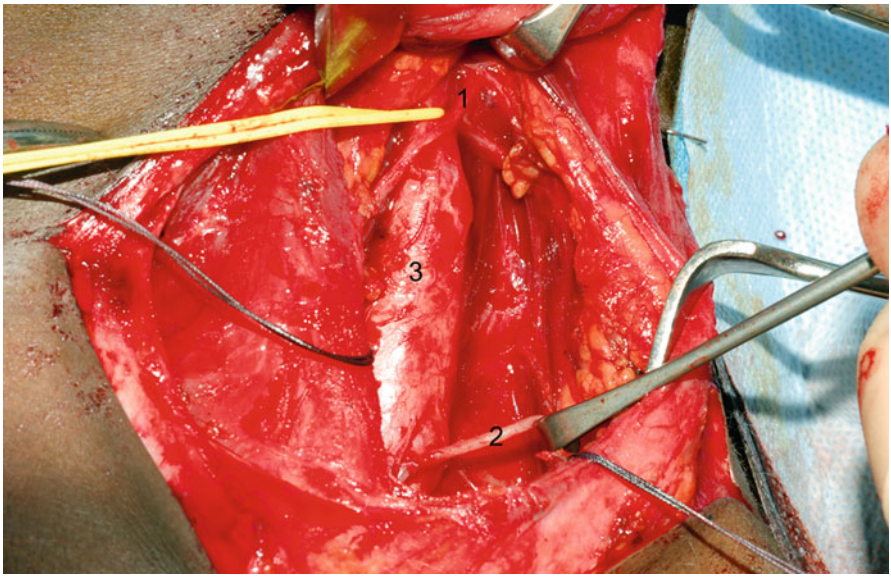


Fig. 5.3 It is often very hard to determine from inspection alone the nature of the injury in delayed or neglected cases. Ten weeks after a severe lesion of C5, C6 and C7 it was not possible to distinguish between rupture and avulsion. The phrenic nerve (*1*) suprascapular nerve (*2*) and the neuroma (*3*) are seen

injury. Secondary repair is performed at between 3 weeks and 3 months after injury. Late, or neglected, repair is reserved for those cases where delay exceeds 3 months.

Indications for exploring nerves injured by fractures and dislocations include:

- the fracture needs internal fixation;
- there is associated vascular injury
- wound exploration of an open fracture is necessary
- a fracture or dislocation is irreducible.
- the lesion deepens while it is under observation,
- the lesion occurred during operation for internal fixation.
- If the clinician elects to convert a closed fracture to an open one by internal fixation, then it is wisest to expose nerves which are not working. The fracture surgeon who does not do this is asking for trouble (Fig. 5.4).

5.2 General Principles of Operation

5.2.1 Control of Bleeding

The emergency control of bleeding by pressure is an art to practise for we have seen cases where patients were close to exsanguination following stab wounds to the

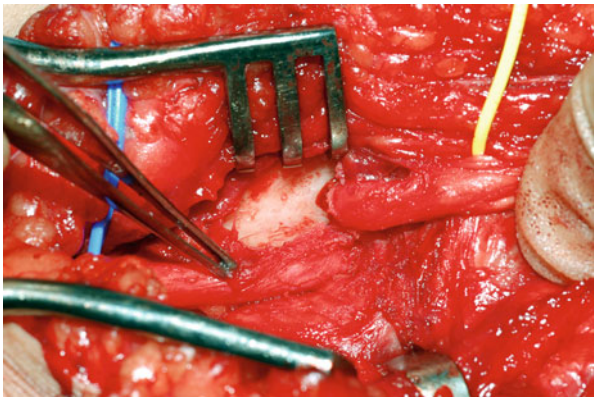


Fig. 5.4 The explanation for persisting painful median palsy in a 7 year old child after fracture of distal humerus, which had been treated by "closed" wiring, was revealed when the nerve was found embedded within the bone 11 months later

axillary, brachial, or femoral arteries. Firm digital compression of the subclavian artery against the first rib of the femoral artery at the groin which enabled successful disarticulation at the shoulder or the hip on the battle fields of the Napoleonic wars [12]. Accidental damage to an artery during operation should be treated by firm compression at the point of bleeding, NEVER by hurried, blind clamping. With this local control the incision may be extended to expose the artery above and below allowing the accurate use of slings and clamps.

The tourniquet is potentially dangerous. It is absolutely contraindicated in a limb in which an arterial prosthesis has been inserted. It seems that the implant is insufficiently elastic to dilate after release of the cuff; also, collateral circulation is likely to be defective. Tourniquet times must be reduced in patients with rheumatoid arthritis, diabetes mellitus, alcohol addiction or other possible causes of neuropathy. In many such patients it is best to avoid altogether the use of a tourniquet. Klenerman [14] provides an important manual about tourniquet use. Post operative pain is worsened by the tourniquet by longer periods of ischaemia and in older patients. The duration of application, inflation pressure and site of application should be always recorded in an ordered operating note and the times of application and release should be written on board in the operating theatre and in the case notes. The duration of tourniquet ischaemia is reduced by inflating the cuff after preparation and towelling of the limb. Elevation of the limb before inflation of the cuff is preferred to the exsanguinating bandage.

5.2.2 Preparation

Because these operations are usually time-consuming, it is especially important adequately to protect the pressure points at the knee, the elbow and elsewhere with suitable padding. In operations on the nerves of the neck in particular, special care must be taken to avoid air embolism, to recognise it if it does occur and to be prepared to deal effectively with any such occurrence. In the case of severe injuries

to the brachial plexus, when there may be a sudden release of spinal fluid, it is necessary to be prepared quickly to alter the position of the patient to avoid "coning" of the medulla. If it seems likely that nerve or vein grafts are going to be needed, the donor sites should be prepared and monitoring equipment, arterial and venous lines and the drapes should be placed accordingly. It is all too easy to get lost especially in a swollen, deformed or scarred limb. Inclusion of joints above and below the presumed site of injury within the area of skin preparation helps to prevent this.

5.2.3 Prevention of Pain

Surgeons can and should do more to prevent post operative pain. The complications of blind nerve or regional blocks can be severe. Infarction of the spinal cord is a catastrophic complication of interscalene or intervertebral block; laceration of a nerve trunk by a needle can cause severe intractable pain; laceration of a vessel adjacent to a nerve may lead to a haematoma. Henry [13] describes his technique of thigh amputation without tourniquet or general anaesthetic by a method first described before the Second World War: "so, after infiltrating areas of flap or cuff, and all the operative field – most thoroughly- with procaine (0.5 %), we can in comfort see and block the great sciatic trunk". Then follows ligation of main vessels and then: "during these activities, however, the great sciatic trunk will rest in peace – until the time comes to remove the limb. With procaine infiltration, not less than 20 minutes must elapse before injecting and dividing this capacious conduit of shock impulse. And that, indeed, is little time enough". Surgeons can and should do a great deal more to diminish postoperative pain by the simple and safe expedient of infiltration of the line of incision and the skin on either side, with local anaesthetic use a preparation of Levobupivicaine 0.25 %, with adrenalin 1:200,000. The maximum dose is 2 mg/kg body weight. It is a simple matter to infiltrate the tissues around the supraclavicular nerves and to inject local anaesthetic into the joint itself for operations at the shoulder. If amputation of the lower limb is indicated because of a deformed and painful foot, a circumferential block of the skin of the mid thigh is made before exposing the sciatic nerve. This is bathed in local anaesthetic infused through an epidural catheter, with its tip adjacent to the nerve, before proceeding to amputation. The infusion is maintained for 48 h after operation. Other nerves are infiltrated with local anaesthetic before cutting them.

5.2.4 Apparatus and Instruments

Most operations on injured peripheral nerves are best done with general anaesthesia. The anaesthetist using a muscle relaxant must be prepared to reverse its effect when the nerve stimulator/recorder is being used.

Special apparatus required includes bi-polar diathermy, stimulating and recording apparatus and instruments for magnification.

Neurophysiological examination: Nerve stimulation and the recording of nerve conduction is used to (1) identify the nerve, (2) to identify individual bundles within a nerve or in a prepared stump, (3) to demonstrate conduction across a lesion and (4) to record continuity between the central and peripheral nervous systems. For simple stimulation and observation of motor response only the simplest uni- or bi-polar stimulator is necessary. For stimulation and recording from muscle and nerve, more elaborate apparatus is required. The Medelec synergy monitoring system (Vaisys Health care, Madison, Wisconsin, USA) (Fig. 5.5). The electrodes are provided by Ambu, Ballerup, Denmark. In measuring conduction across a lesion, hand held

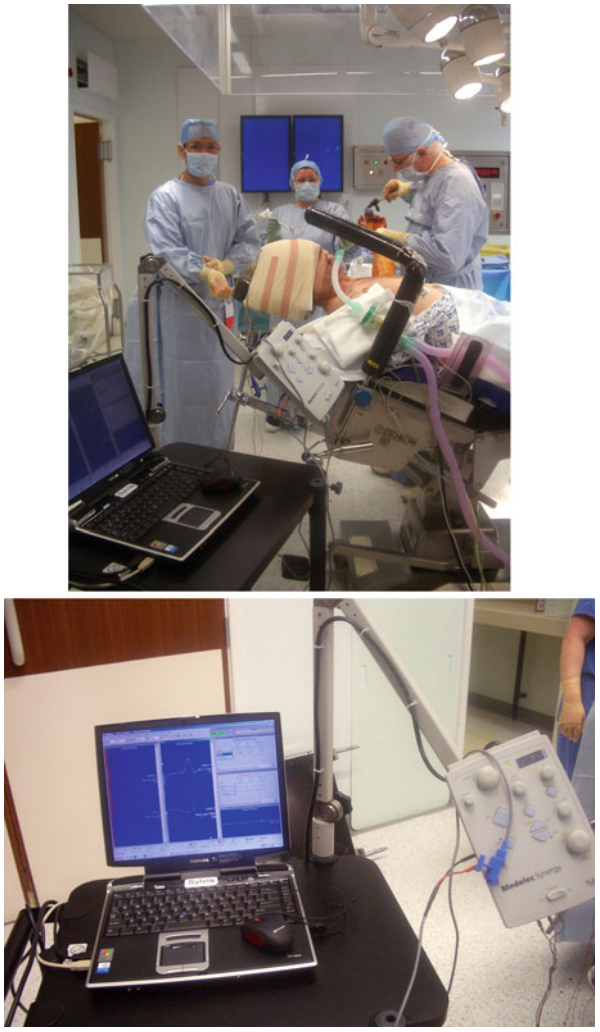


Fig. 5.5 The apparatus for recording sensory evoked potentials

bi-polar stimulators are placed on either side of the lesion. The ground electrode can be placed in a convenient adjacent area. The interval between the electrodes and between each electrode and the lesion is measured (Fig. 5.6).

For recording somatosensory evoked potentials (SSEP's) the reference electrode is placed on the forehead, the ground electrode at the temple, and the recording electrode on the skin overlying the second or third cervical intervertebral space. The skin is prepared with abrasive paste and alcohol wipes to lower resistance with the object of balancing impedance between reference and recording electrodes, at a level less than 2.0 kΩ. Once the surface electrodes are positioned they are secured with tape. A hand held stimulator is used to record signals from the median and ulnar nerves before preparing and towelling the upper limb, using the uninjured side as a control. A sterile hand held bi-polar stimulator is used to stimulate the nerve directly. Somato-sensory evoked potentials (SSEP) are recorded using a stimulus rate of 3–5 pulses/s, of duration 2.0 ms, and intensity 150–300 V. Signals are averaged from between 50 and 200 sweeps. The stimulus rate for the hand held bi-polar stimulator is 3–5 pulses/s.

The quality of the traces may be adversely affected by: (1) ambient noise interference from electrical equipment in the operating theatre; (2) when nerves are embedded in dense fibrosis; (3) when the wound is permitted to become too wet or too dry; (4) compression of nerves by haematoma causes anoxic conduction block; (5) SSEP's are relatively unaffected by most anaesthetic agents, but muscle relaxants block neuromuscular conduction (Fig. 5.7).

Magnification: the operating microscope was used by ophthalmic and ENT surgeons for decades before its regular use in orthopaedic and plastic surgery. The discipline of microsurgery seems to have acquired a mystique which is not altogether justified. The elements of microsurgical technique are no more than the application of basic surgical skills. They are acquired by practice. For magnification we use loupes or the operating microscope. The microscopes are OPMI 6SD FC and OPMI 6 (both Carl Zeiss, Oberkochen): the stand is the universal S3B (Carl Zeiss, Oberkochen).

Instruments: The Joll's thyroid retractor is excellent for reflection of skin flaps in the neck. The small Deaver's retractor is useful in the neck. A set of malleable retractors are essential. Conventional toothed self retaining retractors are avoided, because of the risk to nerves and vessels. Three sizes of vascular clamps are used. The Satinsky clamp is especially useful in end to side anastomosis. DeBakey's scissors and forceps are used for both arterial and nerve work. The range of sutures includes: 6/0, 8/0 nylon on a 6 mm or 8 mm vascular needle and 8/0, 9/0, 10/0 and 11/0 which are used with the appropriate needle holders.

Howarth's dental elevator and Lemperts raspatory are very good for fine bone work. A range of spinal punches, angled bone nibblers and bone cutters are needed. Fibrin clot "glue" [28] is now available commercially. (Tisseel (TM) Immuno Ltd, Arctic House, Rye Lane, Dunton Green, Sevenoaks, TN14 5HB). The aprotinin must be diluted with sterile water: otherwise there is a risk of inducing fibrosis. The undiluted preparation is reserved for haemostasis. The needle tip should be directed away from the repair to avoid disruption. A steady gentle pressure is exerted so that

Royal National Orthopaedic Hospital NHS
NHS Trust

Department of Clinical Neurophysiology
Peripheral Nerve Exploration

Patient:
Patient ID:
Sex:
Date of Birth:
Age: Years Months
Ward:
Surgeon: Prof Birch
Technician: Karen Holmes/Catherine Jones
Notes: Exploration of R Brachial Plexus. Fall at home Dec 2006. Dislocation of the R shoulder, altered sensation, loss of power.

Surgeon requires across lesion traces only.

Intra-op

Nerve / Sites	Lat. ms	Amp.2-3 µV
1Musculocutaneous cord – Lateral cord *(present)- distance* ***5cms****- distal - prox*	0.75	16.5
2 repeat *(present)- distance* ***5cms***	1.05	9.3
3 Radial nerve – Posterior cord *(absent)- distance* ***5cms***		
4 Circumflex nerve – Posterior cord *(present)- distance* ***4cms***	1.05	4.0
5 Medial cord *(present)- distance* ***3cms***	0.75	13.9

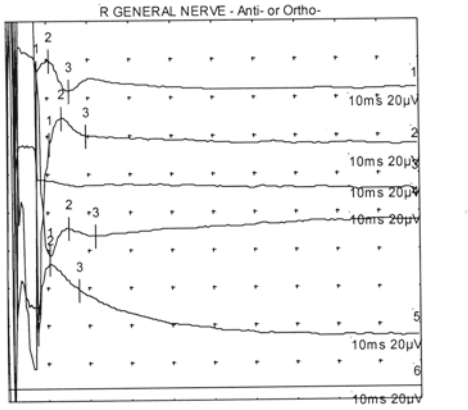


Fig. 5.6 Severe pain and deep paralysis complicated closed dislocation of the gleno-humeral joint in a 78 year old woman. The nerves were exposed 3 months later; there were no ruptures. CNAP and SSEP were recorded traversing the lesions of median, ulnar and musculocutaneous nerves. They were absent across the radial nerve. Recovery through the radial nerve extended only to the triceps and flexor to extensor transfer was performed later. The other nerves recovered, there was early relief of pain

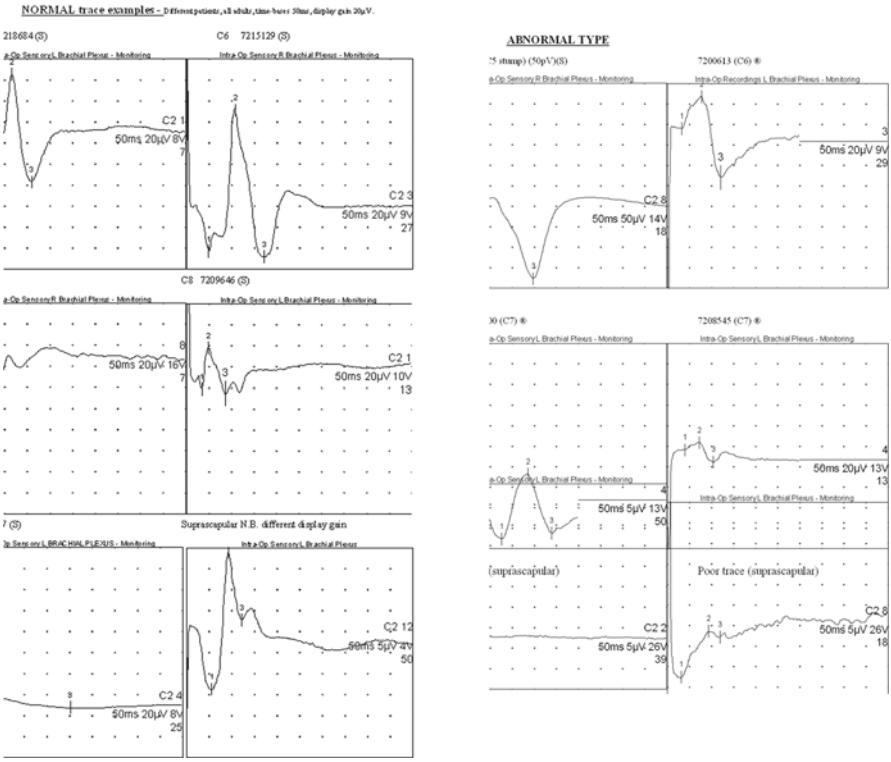


Fig. 5.7 Examples of normal and abnormal SSEP traces

a film of the fluid bathes the repair and seals it. Fibrin clot glue acts as an envelope around the nerve but offers no resistance to tension.

5.2.5 Incisions: Handling of Tissues

This need to be adequate and, where possible, extensile. There is no place for short incisions. Main nerves and major vessels are exposed first proximal to the wound or lesion and then distal to it. The lesions are displayed by dissection from above and below. All tissues must be treated gently. They are, after all, alive, and on their continued viability depend the healing of the wound without infection and the recovery of the nerve lesion. Indeed, avoidance of infection probably has more to do with tender handling of the tissues and accurate haemostasis than has the administration of antibiotics. The line of the proposed incision should be marked and cross-hatched and be planned with cutaneous innervation in mind. Lasting trouble can follow wounding of an apparently trivial cutaneous nerve; it is an ill matter for a surgeon purporting to assist healing of lesions of peripheral nerves to damage one set of

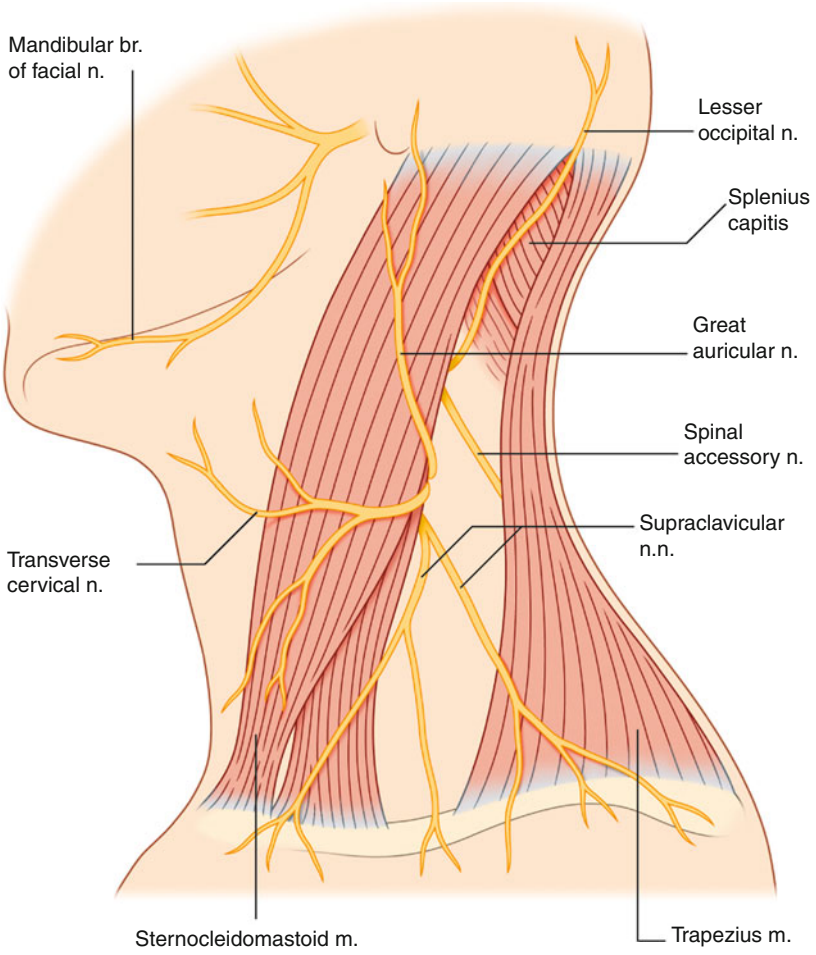


Fig. 5.8 Some of the more important superficial nerves in the neck. Note that the mandibular branch of the facial nerve comes at one point below the lower margin of the mandible. The cervical branch has been omitted

nerves on the way to repairing another (Figs. 5.8 and 5.9). In the neck, the skin flaps should include the platysma; elsewhere, they should be cut to full thickness. Skin flaps should be held with fine skin hooks; if the procedure is going to take a long time, the flaps should be sutured back to the surrounding skin. So far as possible, dissection should be done with the knife or with sharp blunt-ended scissors. Although in the limbs the pneumatic tourniquet can be used during dissection and exposure, it must be released for stimulation, repair and closure. The best possible haemostasis must be secured after the cuff has been deflated, by diathermy, ligation and haemostatic sponge. Cut bone surfaces should be sealed with wax or similar preparation.

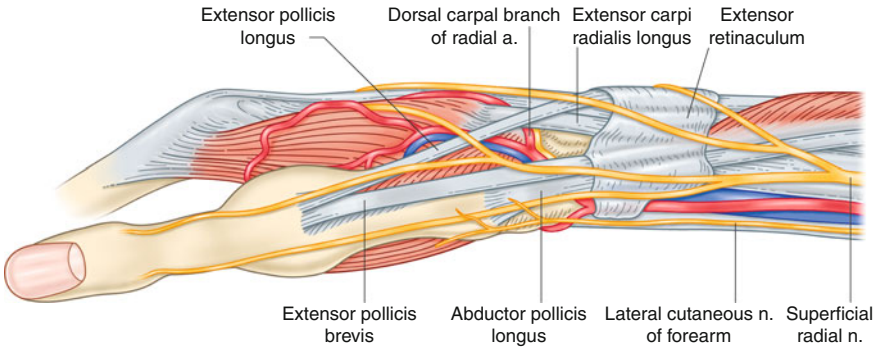


Fig. 5.9 Radial side of the hand, warning about the terminal branches of the radial nerve and lateral cutaneous nerve of forearm

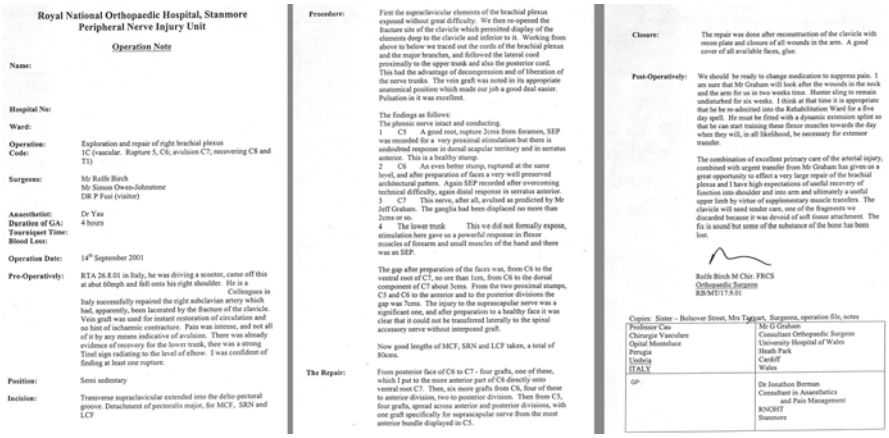
Once the flaps are raised, the field should be kept as free of blood as possible, but it must be kept moist by regular irrigation. Even well shielded operation lamps generate enough heat to accelerate desiccation of the tissues. When muscles have to be divided, their ends should be marked with sutures and if necessary labelled, so that when the procedure is completed they can accurately be re-united. Nerves should be handled with extreme care; retracted with very fine skin hooks in the epineurium or with plastic slings. Colour coding of the slings adds pleasing variety to the proceedings and, more importantly, permits the surgeon to identify to the assistant the nerve to be retracted. They should not be mobilised over such a length as to impair their blood supply.

The wound should not be closed before bleeding points have been checked; even with good haemostasis it is wise to use a suction drain in most wounds. However, the business end of the drain should not be placed near the site of repair, for fear of damage to the anastomosis by the suction or by later withdrawal of the drain. Divided muscle layers should be repaired accurately and securely. In the neck the platysma should carefully be closed with interrupted sutures.

Although with careful handling of tissues wound infection is rare, the exposure is often so much prolonged that prophylactic use of antibiotics is advisable. Such cover should always be used if there is any liability to ischaemia or if there is an associated fracture.

5.2.6 The Record

This should carefully be maintained: it is best to follow a standard form and to supplement this with a diagram and photographs. Under no circumstances must descriptions of the lesion, of the state of the stumps after resection or of the gap after resection be omitted. The operation record should be written or dictated by the operating surgeon as soon as possible after completion of the procedure. The first copy is retained with the medical case notes, a copy is sent to the family practitioner,



Royal National Orthopaedic Hospital, Stanmore
Peripheral Nerve Injury Unit

Operation Note

Name:

Hospital No:

Ward:

Operation: Exploration and repair of right brachial plexus
Code: 1C (vascular. Rupture 5, C6; avulsion C7; recovering C8 and T1)

Surgeons: Mr Rolfe Birch
Mr Simon Owen-Johnstone
DR P Fusi (visitor)

Anaesthetist: Dr Yau
Duration of GA: 4 hours
Tourniquet Time:
Blood Loss:

Operation Date: 14th September 2001

Pre-Operatively: RTA 26.8.01 in Italy, he was driving a scooter, came off this at abut 60mph and fell onto his right shoulder. He is a Collengues in Italy successfully repaired the right subclavian artery which had, apparently, been lacerated by the fracture of the clavicle. Vein graft was used for instant restoration of circulation and no hint of ischaemic contracture. Pain was intense, and not all of it by any means indicative of avulsion. There was already evidence of recovery for the lower trunk, thre was a strong Tinel sign radiating to the level of elbow. I was confident of finding at least one rupture.

Position: Semi sedentary

Incision: Transverse supraclavicular extended into the delto-pectoral groove. Detachment of pectoralis major, for MCF, SRN and LCF

Procedure: First the supraclavicular elements of the brachial plexus exposed without great difficulty. We then re-opened the fracture site of the clavicle which permitted display of the elements deep to the clavicle and inferior to it. Working from above to below we traced out the cords of the brachial plexus and the major branches, and followed the lateral cord proximally to the upper trunk and also the posterior cord. This had the advantage of decompression and of liberation of the nerve trunks. The vein graft was noted in its appropriate anatomical position which made our job a good deal easier. Pulsation in it was excellent.

The findings as follows:
The phrenic nerve intact and conducting.
1 C5 A good root, rupture 2cms from foramen, SEP was recorded for a very proximal stimulation but there is undoubted response in dorsal scapular territory and in serratus anterior. This is a healthy stump.
2 C6 An even better stump, ruptured at the same level, and after preparation of facets a very well preserved architectural pattern. Again SEP recorded after overcoming technical difficulty, again distal response in serratus anterior.
3 C7 This nerve, after all, avulsed as predicted by Mr Jeff Graham. The ganglia had been displaced no more than 2cms or so.
4 The lower trunk This we did not formally expose, stimulation here gave us a powerful response in flexor muscles of forearm and small muscles of the hand and there was an SEP.

The gap after preparation of the faces was, from C6 to the ventral root of C7, no ore than 1cm, from C6 to the dorsal component of C7 about 3cms. From the two proximal stumps, C5 and C6 to the anterior and to the posterior divisions the gap was 7cms. The injury to the suprascapular nerve was a significant one, and after preparation to a healthy face it was clear that it could not be transferred laterally to the spinal accessory nerve without interposed graft.

Now good lengths of MCF, SRN and LCF taken, a total of 80cms.

The Repair: From posterior face of C6 to C7 - four grafts, one of these, which I put to the more anterior part of C6 directly onto ventral root C7. Then, six more grafts from C6, four of these to anterior division, two to posterior division. Then from C5, four grafts, spread across anterior and posterior divisions, with one graft specifically for supracapular nerve from the most anterior bundle displayed in C5.

Closure: The repair was done after reconstruction of the clavicle with recon plate and closure of all wounds in the arm. A good cover of all available faces, glue.

Post-Operatively: We should be ready to change medication to suppress pain. I am sure that Mr Graham will look after the wounds in the neck and the arm for us in two weeks time. Hunter sling to remain undisturbed for six weeks. I think at that time it is appropriate that he be re-admitted into the Rehabilitation Ward for a five day spell. He must be fitted with a dynamic extension splint so that he can start training these flexor muscles towards the day when they will, in all likelihood, be necessary for extensor transfer.

The combination of excellent primary care of the arterial injury, combined with urgent transfer from Mr Graham has given us a great opportunity to effect a very large repair of the brachial plexus and I have high expectations of useful recovery of function into shoulder and into arm and ultimately a useful upper limb by virtue of supplementary muscle transfers. The clavicle will need tender care, one of the fragments we discarded because it was devoid of soft tissue attachment. The fix is sound but some of the substance of the bone has been lost.

Rolfe Birch M Chir. FRCS
Orthopaedic Surgeon
RB/MD/17.9.01

Copies: Sister – Bolsover Street, Mrs Tarquet, Surgeons, operation file, notes

Professor Cau Chirurgia Vascolare Opital Monteluce Perugia Umbria ITALY	Mr G Graham Consultant Orthopaedic Surgeon University Hospital of Wales Heath Park Cardiff Wales
GP	Dr Jonathon Berman Consultant in Anaesthetics and Pain Management RNOHT Stanmore

Fig. 5.10 An example of an operating record

another to the referring clinician, and the final copy is stored with files of coded operating records (Fig. 5.10).

5.3 Methods of Repair

There are remarkable similarities between the technique of repair of nerves and vessels. In arterial injury the first principle is rapid control of the vessels proximal and distal to the wound. Whilst the situation is less urgent in nerve repair, the nerve should be exposed first in healthy tissue above and below the level of lesion. Damaged vessel and nerve must be resected, the repair will fail unless healthy tissues are coapted. Undue tension guarantees failure. Adventitial tissue must be resected to expose the media or the epineurium (Fig. 5.11).

5.3.1 The Vascular Repair

Of combined venous and arterial injury Barros d'Sa [2] says: "ligation (of the vein) should be avoided at all costs. A vein tolerates lateral suturing much better than an artery. At least one major channel of satisfactory calibre must be restored so as to avoid a serious rise in peripheral venous resistance and pressure which reduces arterial flow to the limb and can lead to thrombosis at the site of the arterial repair with disastrous consequences. In combined venous and arterial injury, the vein should be repaired first".

Having secured proximal and distal control of the injured vessel any associated fracture or dislocation must be neutralised as rapidly as possible. A Rush nail,

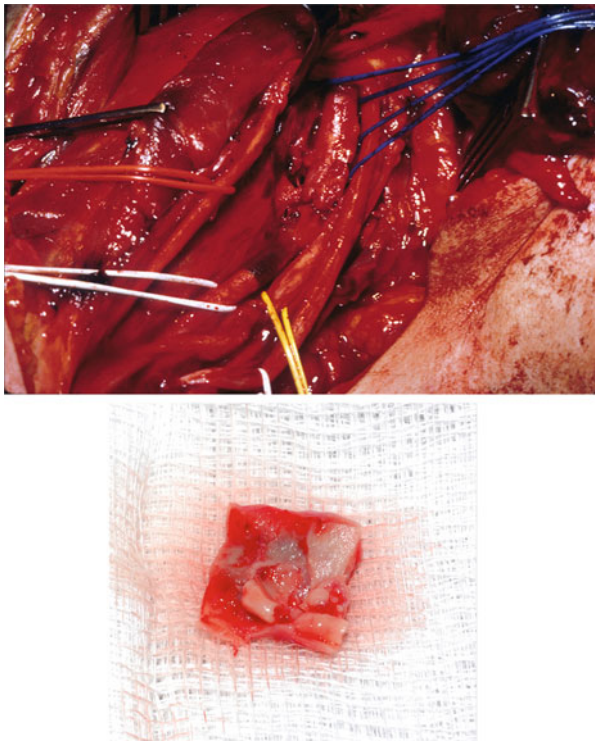


Fig. 5.11 Rupture of axillary artery from fracture/dislocation in a 68 year old man. The atheromatous intima was fractured. A reversed vein graft was necessary after resection back to healthy intima

passed from proximal to distal provides rapid and adequate stability of the humerus before repair of the axillary or brachial artery. Direct suture is sometimes possible in the fresh stab wound; a vein patch is better than lateral suture at the mouth of a false aneurysm. Interrupted sutures are preferred to continuous suture to reduce the risk of stenosis at the suture line, ease co-aptation of vessels of different diameter, and facilitate end to side suture.

Division of the axillary and brachial sheath and of the deep fascia of the forearm is essential in all cases save those where a simple wound is successfully sutured within three hours of injury. Subcutaneous fasciotomy of the forearm is adequate if there is no distal injury of elbow and forearm. The deep fascia of the forearm is exposed through a short incision on the medial side of, and parallel to, the biceps tendon. The skin is retracted and the plane between it and the deep fascia is gently developed by blunt dissection. The fascia is now incised and the plane deep to it opened out in a similar manner so that the fascia can be split safely using blunt tipped scissors. In more severe cases a fasciotomy should include the skin: this is usually susceptible to delayed primary closure at about 48 h. The indications for fasciotomy are more stringent in the lower limb. Decompression of all four compartments of the leg is essential in missile wounds. This principle should be followed in closed lesions complicating fractures of the tibia or deep contusion of the muscles. Two incisions may be used. That over the fibula exposes the deep fascia

enclosing the anterior and lateral compartments. The medial incision starts just above the midpoint between the medial malleolus and the Achilles tendon and extends to the upper leg. It is very important to identify and to open the fascia over the deep flexor compartment.

Repair of artery or vein: Both proximal and distal vessels can be infused with heparinised saline but systemic anti-coagulation is not used. The proximal and distal stumps of the vessels are securely controlled by plastic slings and appropriate clamps. A skilful and patient assistant is charged with controlling the clamps and adjusting their position. Adventitia is removed after confirming back flow from the distal stump of the vessel. Repair is by interrupted sutures, 5/0 or 6/0 nylon for the subclavian, axillary, femoral or popliteal arteries, 7/0 or 8/0 for brachial, radial, ulnar and tibial arteries. The first two sutures are placed at the equator. It is often easier to repair the posterior wall first before turning the clamps to expose the anterior wall. The sutures pass through the media and the intima at intervals of about three quarters of a millimetre.

The reversed vein graft: The graft is taken from an uninjured limb where possible. The long saphenous vein in the leg is best for larger arteries. The prepared stumps of the artery are gently drawn together and the gap between them measured, then the vein graft is prepared to match that gap. A very light touch must be used in handling the vein. Diathermy should not be used on the small branches. The proximal vessel is tied off leaving a long strand of suture to indicate that this must be placed distally during the repair. A flexible cannula, mounted on a syringe, is introduced into the distal stump of the vein and secured with another tie. The segment of the vein is distended with heparinised saline. This reduces spasm. End-to-side anastomosis is used when there is disproportion between the stumps of the vessels (Fig. 5.12). Bleeding from the suture lines is best controlled by pressure for several minutes before inserting further sutures. The repaired vessel should be kept exposed for as short a time as possible and kept moist at all times. Nerve grafts must be elevated as swiftly as possible and much of this can be done whilst the arterial repair is underway. Fibrin clot glue is invaluable in these cases, for its saves a great deal of time.

5.3.2 The Nerve Operations

Neurolysis: A good deal of the argument about the value of "neurolysis" arises from imperfect definition of the term [3].

External neurolysis is the freeing of the nerve from a constricting or distorting agent by dissection outside the epineurium and it is especially valuable when the nerve is intact but tethered, strangled or immobilised by scar. Pain (neurostenalgia) is usual in such cases and relief of pain with improvement in function is regularly seen after external neurolysis and decompression.

External neurolysis after repair or amputation: Neurolysis is usually fruitless in a nerve which has been repaired. The decision to revise that repair is governed by failure to progress, persisting pain and a static Tinel sign (Fig. 5.13). However, it is

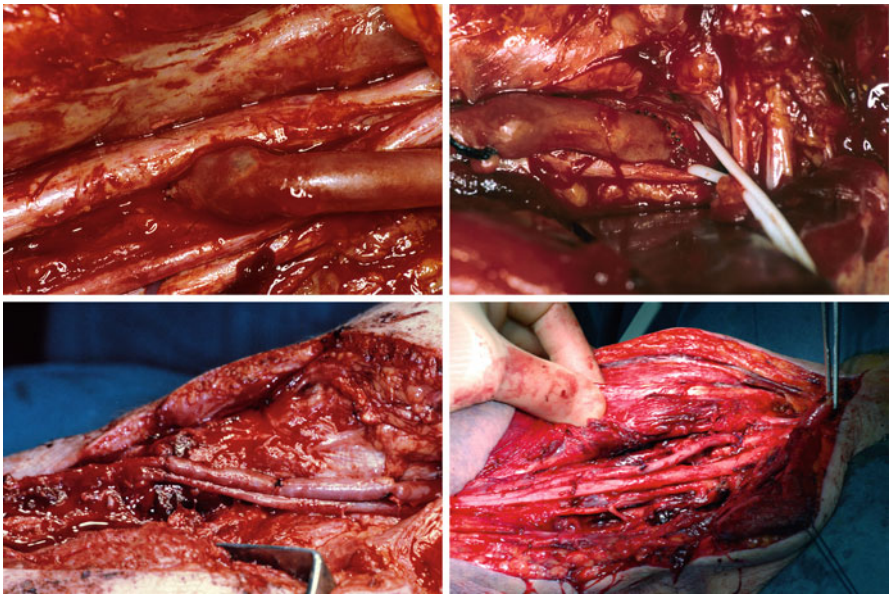


Fig. 5.12 Reversed vein grafting in the axilla: end-to-side (*above*) and end-to-end (*below*)

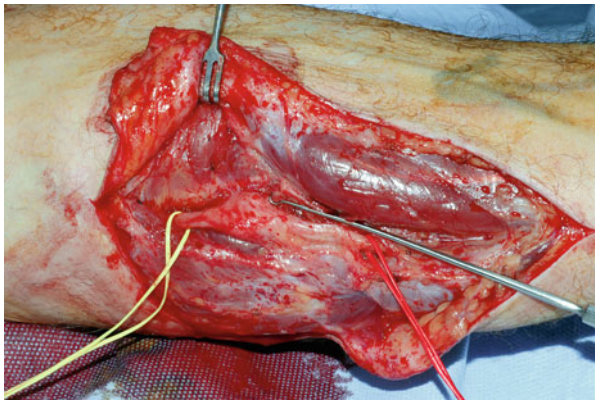


Fig. 5.13 Futile neurolysis. Poor recovery after primary suture of an ulnar nerve. The nerve was re-explored at 8 months. A CNAP was detectable and neurolysis was done. There was little recovery

not uncommon to find that the function regained after successful suture of the median or ulnar nerves at the wrist is marred by pain because the nerve has become adherent to the adjacent flexor tendons. Pain is worsened by movements of the digits and the neuroma can be seen moving up and down during flexion and extension. Liberating the nerve by incision of adhesions is often successful but a bed of healthy synovium must be restored. A neuroma which has become adherent to the scar over an amputation stump is often extremely painful. Again, liberation of the nerve from scar, which may require cutting the nerve again so that the stump lies in healthy tissue, usually relieves the pain.

Epineurotomy: means the simple division of the epineurium in the line of the nerve and it probably has a limited place in cases of damage from injection of a noxious substance near a nerve or by localised pressure on a nerve, which has produced localised thickening and fibrosis of the epineurium. Unhappily, the fibrosis is unlikely to be limited to the peri-fascicular epineurium: there is likely to be fibrosis of the interfascicular epineurium too. The place of epineurotomy in radiation neuropathy is uncertain. The most that can at present be said for it is that when done carefully it is unlikely to do any harm.

Internal neurolysis or *inter fascicular neurolysis* is the exposure of the bundles by epineurotomy and their separation by dissection between them or by the removal of interfascicular scar tissue. It is an essential part of three important procedures: (1) separation of intact from damaged fascicles in nerves which have suffered partial damage; (2) separation of a motor fascicle of a nerve for transfer; (3) separation of intact fascicles during removal of a benign but infiltrative tumour.

The traction lesion in continuity is a common finding at exploration and it is very difficult. The nerve, which is usually in the axilla or at the knee, is exposed after a severe closed traction injury, and is found elongated by one third or more. The epineurial blood vessels are torn, but the perineurium and individual bundles appear intact. The damaged segment may exceed 25 cm. in length and resection and repair of such extensive lesions is scarcely feasible. Useful recovery occurs naturally in-between one third and one half of these injuries. It seems that the damaged segment acts as an imperfect graft because the perineurium is in continuity and some, at least, of the Schwann cells survive.

5.3.3 Biopsy

Biopsy of a nerve requires removal of portions of conducting tissue and it necessitates at least epineurotomy and excision of one or more bundles so that the perineurium and its contents are made available for examination. Biopsy may extend to the entire nerve. Nerve biopsy is neither trivial nor without risk and it should never be a matter of unthinking routine.

Case report: A 52 year old woman with chronic inflammatory peripheral neuropathy experienced increasing pain and a deepening sciatic neuropathy raising the possibility of plasmacytoma. One fascicle of the nerve was biopsied. On the following day she noted a virtually complete but painless, sciatic palsy. Haematoma was excluded by ultrasonography and by reexploration. The biopsy excluded plasmacytoma and it seems that an apparently relatively innocuous intervention in which a segment of one fascicle of the sciatic nerve was removed induced a dense ischaemic lesion of the whole trunk.

It is in the investigation of nerve tumours that the greatest errors are seen.

Case report: A 43 year old woman presented with a 3 year history of intermittent abdominal symptoms; a mass was palpable. MRI showed a large tumour in the retroperitoneum, extending from L1 to the sacrum. A diagnosis of soft tissue sarcoma

was made on the basis of a needle biopsy and the mass was excised, including a segment of the lumbo sacral plexus L4, L5 and S1. Chemo-and radiotherapy followed, complicated by a massive pulmonary embolism. The histological material was reviewed: the diagnosis was revised to a benign schwannoma. The patient now has profound weakness of the abductors and extensors of the hip, paralysis of the dorsi flexors of ankle and foot, weakness of quadriceps and other muscles of the lower limb. She is able to walk 50 yards with two sticks.

Knight et al. [17] described the complications attending biopsy, usually by fine needle, in 53 patients with benign solitary schwannoma.

- Eight biopsies failed to yield diagnostic material
- Ten biopsies removed portions of normal nerve
- An erroneous diagnosis of a soft tissue sarcoma was made in two core needle biopsies
- Most patients experienced significant or severe pain as a result of the biopsy and there was significant loss of function in 22 of them.
- Fibrosis induced by the biopsy distorted the tissue planes of the nerve and added greatly to the difficulties of later enucleation of this benign tumour.

It is important for the surgeon who comes to treat such lesions to ask to see the sections taken from the material removed at the primary operation. Often enough, a cross section of a trunk nerve is included in the specimen (Fig. 5.14). There is no justification for biopsy in tumours of nerves which are clearly benign. The clinical

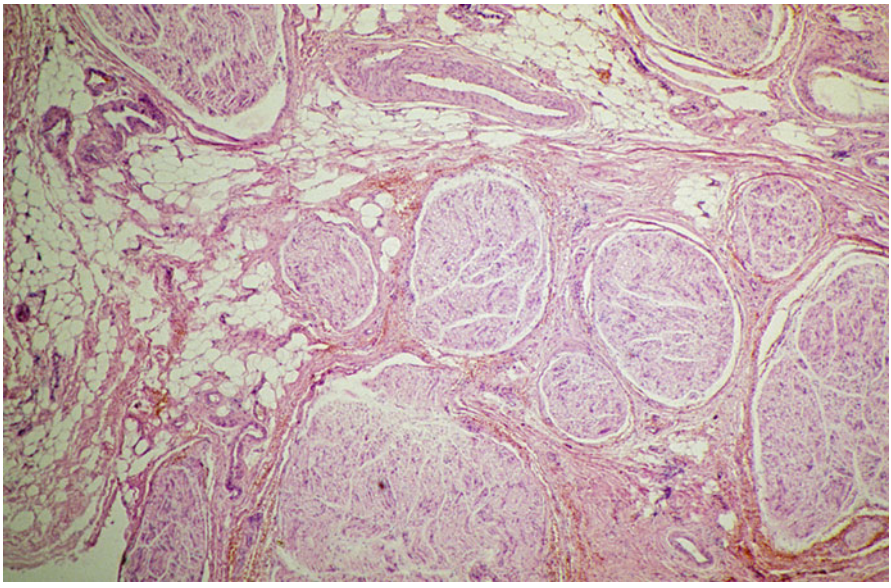


Fig. 5.14 Biopsy of a benign lesion of the femoral nerve caused severe pain and complete paralysis of the extensor muscles of the knee. No action was taken for 10 months. The first surgeon did not examine the specimen. Histological findings: fascicles of a main nerve

features supported by the findings from magnetic resonance imaging and ultrasonographic scanning enable the accurate diagnosis of nearly all cases of schwannoma and intraneural ganglion.

An incorrect diagnosis of a benign lesion in malignant peripheral nerve sheath tumour (MPNST) may cost the patient his or her life because of dissemination of tumour from the nerve into adjacent soft tissues or because of the continuing extension of the tumour during the months before the correct diagnosis becomes all too clear (Fig. 5.15). Ten such cases have been seen. In five of these the initial error, compounded by delay, lost the chance of adequate surgical excision. Whilst biopsy is essential in cases in which there is any doubt about diagnosis and in particular in those where there is a possibility of primitive neurectodermal tumour, neuro-epithelioma or extra osseous Ewing's tumour, that biopsy is best performed within the Institution where definitive treatment will take place. The possibility that the tumour is catecholamine secreting should be considered in adrenal and extra-adrenal retroperitoneal lesions.

It is for the responsible surgeon to decide whether biopsy is needed and which method is used. It is the responsibility of that surgeon to examine the biopsy tissue with an experienced pathologist. Surgeons do not always get things right, neither do radiologists or pathologists. A surgeon encountering an unexpected tumour within a nerve, one which is not evidently a benign schwannoma, will do no harm in leaving the nerve well alone, closing the wound and referring the patient to an interested

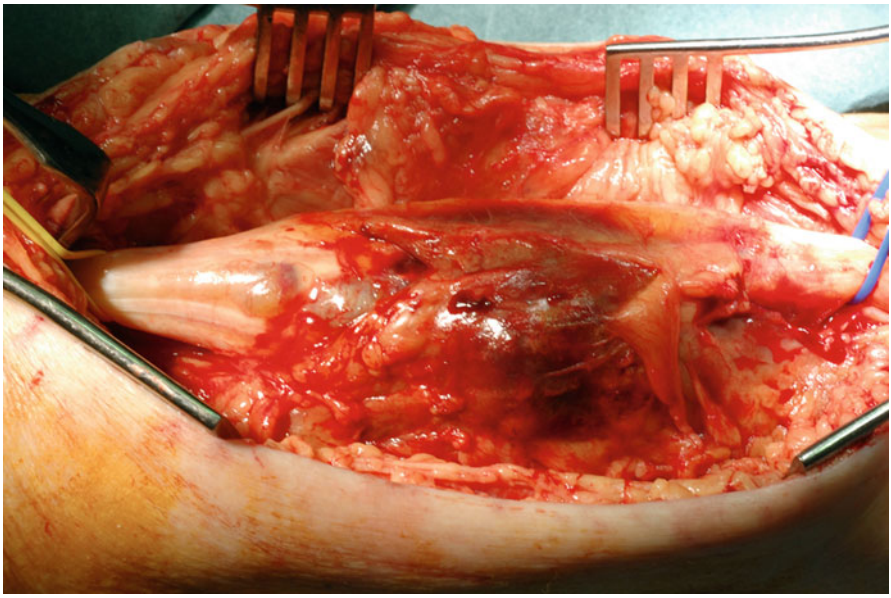


Fig. 5.15 Open biopsy was performed on a lump in the thigh in an 87 year old woman. She experienced intense pain and raid progression of neural deficit. The lesion was re explored 1 month later, the tumour had burst out of the epineurium and infiltrated the muscles of the thigh and had extended to the skin

colleague. We emphasise the importance of frozen section biopsies during resection of MPNST which are essential in proving an adequate margin of resection.

5.4 The Nerve Repair

- The object of nerve repair is the accurate coaptation of healthy conducting elements without tension. In practical terms this means accurate coaptation of the bundles (Fig. 5.16).
- This is done after skeletal injuries have been stabilised and vessels repaired, and after muscles, tendons, joint capsule and synovium have been drawn together so restoring gliding planes.
- Cover by healthy full thickness skin is essential (Fig. 5.17).

Once the decision is made for or by the surgeon, the nerve ends are cut back progressively until healthy pouting bundles show in the cut surfaces (Fig. 5.18). Resection is less in urgent repairs. No more than 1–2 mm of nerve is removed in tidy wounds. Finding the right level of section in the closed traction rupture or in the untidy wound is much easier in urgent repairs where there is still conduction in the distal trunk. In ruptures of the spinal nerves, the stimulator is moved slowly from the rupture towards healthy tissue until an SSEP becomes detectable (proximal stump), or, until muscular activity returns (distal stump). This usually coincides with a healthy looking nerve face. In traction ruptures, the amount of tissue resected

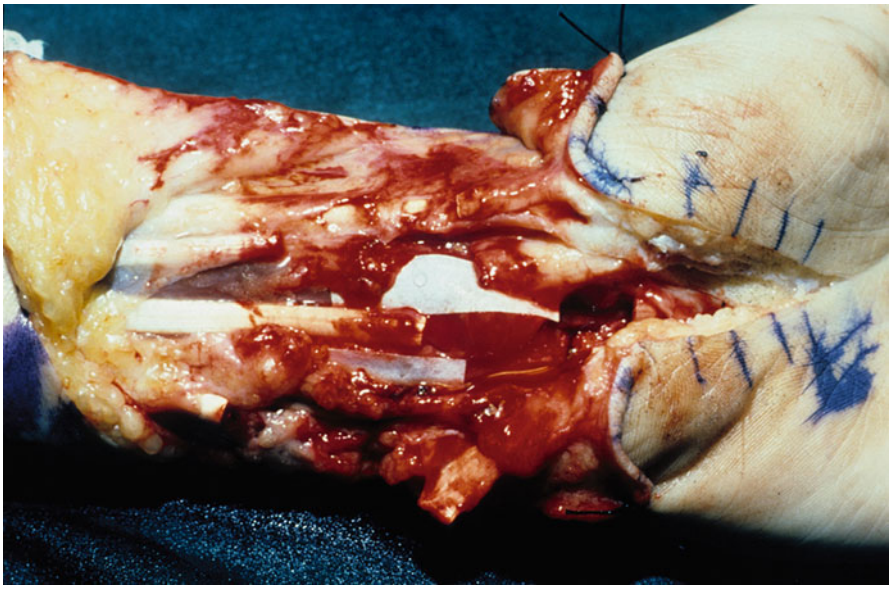


Fig. 5.16 A tidy wound. Primary repair of all divided structures is indicated

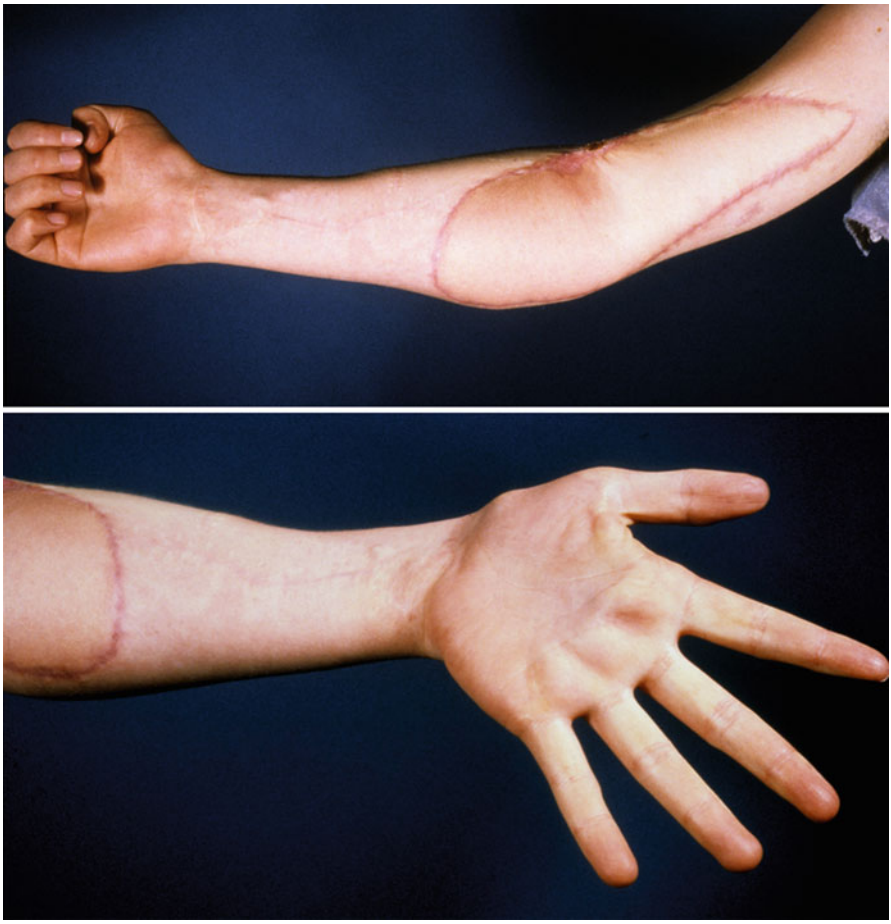


Fig. 5.17 A 28 year old woman suffered a gunshot wound to the elbow destroying the brachial artery, median nerve, and overlying skin. These were repaired and skin cover provided by a free latissimus dorsi myocutaneous flap

usually lies between 5 and 10 mm. More nerve must be resected in late cases. When the case has been complicated by infection as much as 4 cm of proximal and distal stumps are irretrievably fibrosed. Palpation of the nerve detects the difference between soft, healthy tissue from the firm or hard, scarred segment.

Then the ends must be united, preferably by end to end suture. So long as the gap after resection is small, little mobilisation of the nerve is needed to close it, and the repaired nerve lies without tension, without excessive flexion of adjacent joints. One simple test as to the advisability of direct suture of a nerve trunk at the wrist or in the forearm involves passing an epineurial suture of 7.0 nylon, with the wrist flexed to no more than 30°. If this suture will draw the stumps together without tearing the epineurium and without causing blanching of the epineurial vessels, then

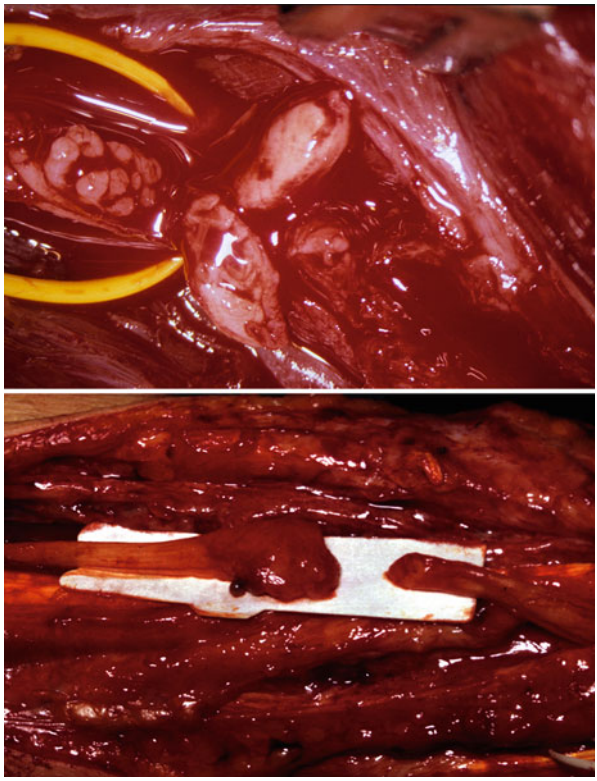


Fig. 5.18 Good stumps after resection in traction injury of the radial nerve. *Below*: the rupture displayed. *Above*: resection is done back until clearly separated "pouting" bundles are visible

suture is reasonable. Failing that, grafting is necessary. It will be seen that in many circumstances it is better to bridge a gap with a graft than to force direct suture; it is better to resect to healthy bundles and create a wide gap than to resect too little in order to facilitate direct suture. We take as guides the following principles:

- End to end suture of the nerves of the brachial plexus above the clavicle or of the accessory nerve is never practicable.
- It is impracticable to bridge with grafts gaps in lesions of the whole sciatic nerve. Sufficient graft material is not available. The gap has to be closed by flexion of the knee and extension of the hip and later maintenance of that position for the appropriate time.
- Anterior transposition of ulnar or radial nerves gives at most 3 cm.
- No gaps in the median nerve in the forearm can be closed by end to end suture.

Ideally, it is best to match bundle to bundle, sensory fibres to sensory fibres and motor fibres to motor fibres. It is often easy to match bundle to bundle simply by looking at the nerve end under the microscope or with the loupe, though the changing architecture of the nerve along its length makes this difficult when a long gap has to be bridged. When operation is done soon after injury it is easy to determine the sites of motor fascicles in the distal stump and to effect an electrophysiological orientation.

5.4.1 Methods of Suture

Orgell [21] described a modified fascicular suture: "group fascicular suture" and he concluded that since there was little difference between the results of epineurial and perineurial suture, epineurial suture was "the technique of choice for most acute nerve lacerations." He pointed out that it was easier and faster and entailed less manipulation of the internal structure of the nerve than did fascicular suture. Spinner [25] thinks that "fascicular" suture is useful in distal median and ulnar repairs and he emphasises that the most important cause for failure of suture is "inadequate resection of injured nerve back to healthy tissue".

During urgent or emergency operation the surgeon should indeed proceed to a neat epineurial suture using 6/0 or 7/0 sutures or to immediate grafting if circumstances permit. Many excellent results have been seen by this approach in severe cases of injury or when the nerve has been inadvertently divided during an operation.

Accurate matching is assisted by the orientation of epineurial vessels, and by making a sketch of the prepared faces indicating the size and the disposition of different bundles.

The preferred method is bundle (fascicular) suture, combined with epineurial suture for most main nerves, with the exception of the sciatic. In the early days after division of a nerve bundles are mobile within the epineurium, and epineurial suture increases the risk of mal alignment. Dissection within the nerve is avoided as this surely leads to fibrosis. The needle is passed through the condensed inner epineurium and the perineurium to secure accurate coaptation of larger bundles. The repair is completed by epineurial suture (Fig. 5.19). In delayed repair, fibrosis within the epineurium stabilises the bundles so that they cannot rotate within the epineurium. In these, epineurial suture may be adequate. The atrophy of the distal stump and the extent of fibrosis in both proximal and distal stumps increases the difficulties in neglected cases (Fig. 5.20).

In both primary and secondary suture, the areolar adventitial tissue is pushed back from each stump to expose the true epineurium. In fascicular suture, matched bundles, identified by size and by position in the nerve, are united by perineurial sutures of 9/0 or 10/0 nylon. Once these "key" bundles have been united, the union is completed by passing sutures of 8/0 or 9/0 nylon through perineurium and epineurium The nerve can be rotated on a saline-soaked dental swab, first from one side and then from the other, so that the whole circumference is accessible. Between 18 and 25 sutures are used to repair the adult median nerve at wrist level. In epineurial repair, orientation of bundles is achieved as well as is possible, and the epineurium is united with two lateral sutures of 8/0 nylon, the ends of which are left long. The repair of the anterior face is completed with sutures of 8/0 or 9/0 nylon, and the nerve is then rotated by manipulation of the lateral sutures so that the posterior epineurium can be united. If fibrin clot glue is available this reduces the number of sutures required. The glue is applied after suture. Heavier epineurial sutures (6/0 or 7/0) are used for suture of the sciatic nerve.

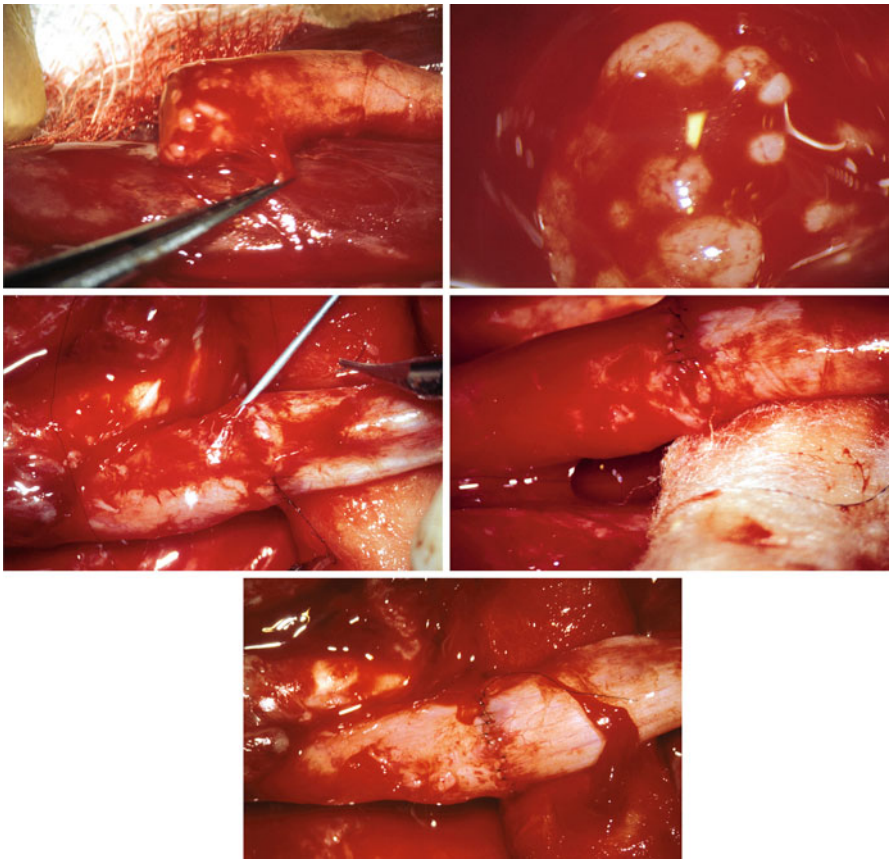


Fig. 5.19 Primary suture of median nerve at the wrist. *Top right*: the pattern of the bundles is examined. *Top left*: the first sutures pass through the perineurium of the larger bundles then follows epi-perineurial suture. *Middle*: the nerve is rolled on a swab for access to the posterior aspect. The completed repair is seen *below*

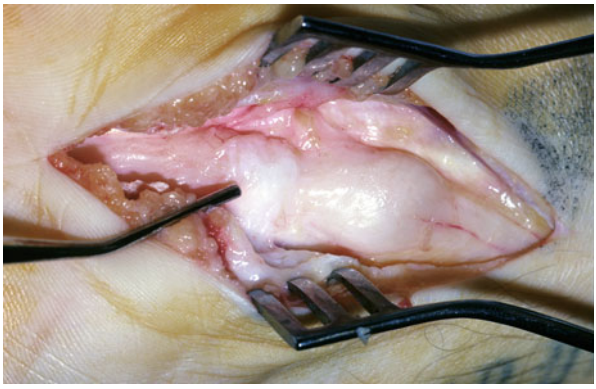


Fig. 5.20 The opportunity for primary suture was lost in this transection of median nerve displayed 3 months after wounding

5.4.2 *Grafting*

Clinicians must never forget that nerve grafts obtain their blood supply from the bed in which they lie. “Since the wounds of nerves that call for repair by grafting are usually extensive the need for the replacement of skin scars by healthy tissue arises with corresponding frequency. Nothing less than a full thickness flap or tube pedicle graft will suffice since it is important the graft should lie, so far as is possible, in healthy well vascularised tissue” [24]. Leaving a nerve graft within scarred muscle or underneath split skin graft just will not do.

The limitations of conventional grafting.

- There is only a limited amount of cutaneous nerve available. In an adult with a complete lesion of the brachial plexus it is possible to collect about 180 cm of nerve by using both sural nerves with the cutaneous nerves of sensation from the injured limb.
- The added defect imposed upon the patient may be too severe. The lateral cutaneous nerve of forearm and the superficial radial nerve provide significant innervation to the skin of the thumb, the thenar eminence and the palm of the hand. They are used only when the parent nerve is irreparably damaged. The supraclavicular nerves provide important sensation from the skin above the clavicle, over the shoulder and the upper part of the chest. The sural nerve innervates the heel and it should not be used for repair of low lesions of the ipsilateral tibial nerve. Pain is a common complication after deliberate wounding of the terminal branches of the nerves of cutaneous sensation and it is advisable always to section the donor nerve proximally, deep to the deep fascia.
- The architecture of a cutaneous nerve bears little resemblance to that of a main nerve trunk. The fifth cervical nerve contains between four and eight bundles. The largest of these requires one strand of cutaneous nerve which may contain between 8 and 20 such bundles.
- There is much disproportion in the volumes occupied by conducting tissue and the calibre of nerve fibres between the proximal segments of main nerves and cutaneous nerves. Myelinated nerve fibres (MNF) account for between 46 and 70 % of the cross sectional area of the ventral root of L5, and between 35.8 and 50.1 % of the dorsal root. The area falls to between 23.8 and 34.5 % in the proximal sural nerve. The median diameter of MNF in the ventral root of L5 is 12 μm. It is about 5 μm in the tibial nerve and just under 4 μm in the sural nerve [9] The problem worsens when regenerating axons having traversed the graft reach the distal trunk of the nerve for here, particularly in late or neglected cases, the Schwann tubes are embedded in dense collagen.
- The recognition that Schwann cells may be specific either to motor or to sensory axons casts a shadow over the use of cutaneous nerves for the repair of main nerves. Regeneration of motor axons is better promoted by a graft of a “motor” nerve whilst the regeneration of sensory axons is better through a graft from a cutaneous nerve [11].

Wherever possible cutaneous nerves from the damaged limb are used. The medial cutaneous nerve of forearm (MCNF) is best, if it is available. The ipsi lateral sural nerve should never be used for the repair of a low lesion of the tibial nerve for this adds to the denervation of the skin of the heel. These patients are better off losing one of the medial cutaneous nerves of forearm.

No graft should be elevated until the injured nerves have been exposed and the extent of lesion defined and the gap between the prepared faces measured. This is a good time for the surgeon to pause for a few minutes of reflection, so that the repair can be properly planned. This is particularly important when repairs of several main nerves are necessary. A map of the proximal and distal faces is prepared outlining the pattern of the bundles in each stump and measuring the length of the gap between the prepared stumps. Then, a calculation is made of the number of grafts required for each nerve, and the disposition and length of the grafts which are cut to about 15 % more than the measured gap.

Elevation and preparation of the graft: The MCNF is taken from the arm through a straight incision. One anterior branch will be seen in the middle of the arm, lower down the nerve divides into two branches which straddle the main brachial vein. The SRN is delivered through separate incisions. The nerve is identified where it emerges from deep to the brachioradialis. The terminal branches are divided at the wrist. The radial nerve is displayed between brachioradialis and brachialis, and the superficial branch identified by gentle traction. It is delivered into the elbow wound, a manoeuvre which has the advantage of stripping it of much of its adventitia. Up to 30 cm of nerve are available in the adult. The LCNF is found just lateral to the biceps tendon and it can be displayed, in the lower part of the arm, between the biceps and brachialis muscles. About 15 cm of graft is available. The terminal 4 cm of the posterior interosseous nerve provides useful material for grafting palmar digital nerves (Fig. 5.21).

The sural nerve(s) is almost always required in repair of lesions of the brachial plexus in the adult and in cases where more than one main nerve must be repaired. The patient is prone for repair of the sciatic nerve and its divisions, otherwise they are placed supine. The lower limb(s) is elevated by a stoical assistant or the knee is flexed to about 70° with the foot resting on the table. The nerve is exposed through a long midline incision which moves laterally in the distal one third of the leg to a point midway between the posterior aspect of the lateral malleolus and the lateral margin of the Achilles tendon. The incision may be extended into the popliteal fossa as a Z. Up to 50 cm of graft is available.

The grafts, tenderly handled, are laid between saline soaked swabs and cut to appropriate length with a new blade or with vascular scissors. The nerve ends are prepared so that the fascicles protrude, and are laid in the prepared bed which must be healthy and unscarred. Fixation is usually by fibrin clot glue but the grafts can be sewn into place, two lateral sutures of 9/0 nylon being used for each. The sutures unite the fascicles of the stumps with the grafts. Because the individual strands of the grafts are likely to be around the same size as the individual bundles, the suture unites epineurium of graft to perineurium of bundle (Fig. 5.22).

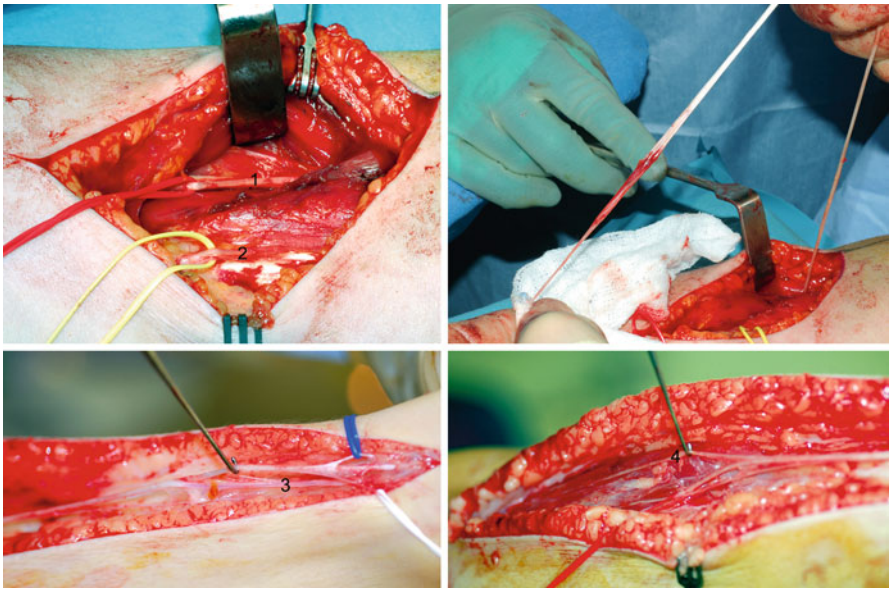


Fig. 5.21 Nerve grafts. *Above left*: the superficial radial nerve (*1*) and lateral cutaneous nerves of forearm (*2*) are displayed at the elbow. *Above right*: the radial nerve is drawn into this wound after section of the distal branches at the wrist. *Below*: the communicating branches of the sural nerve are variable. This nerve is best elevated through a long incision. *Below left*: the sural nerve divides into its terminal branches close to the short saphenous vein (*3*) above the ankle. *Below right*: note the communicating branch from the common peroneal nerve in the upper leg (*4*)

The proximal stump of the nerve is grafted first, leaving the distal ends of the grafts laid out across the bed before proceeding to the distal stump, matching as far as is possible, fascicle to fascicle. It is very important, once both ends have been anastomosed, carefully to inspect the grafts and the suture lines to make certain that during the union of one end the union of the other end has not been disturbed.

- It is better to bridge the gap with grafts if intact fascicles have been separated from the lesion.
- All nerve repairs must be secluded from naked bone, tendon, or lacerated muscle by healthy tissue.
- Grafts are stabilised by closing healthy synovium or fat, or muscle, over them. The fat pad is a valuable shield in the posterior triangle of the neck and it should be carefully apposed over the repair.
- It is necessary to restate that repairs of nerves and vessels must be covered by healthy, full thickness skin. Split skin grafts induce severe fibrosis which strangles.

Vascularised grafts: the free vascularised ulnar nerve graft in repair of the brachial plexus was a development of the ingenious operations described by Strange [26] and MacCarty [19]. In Strange's operation the ulnar nerve is used to

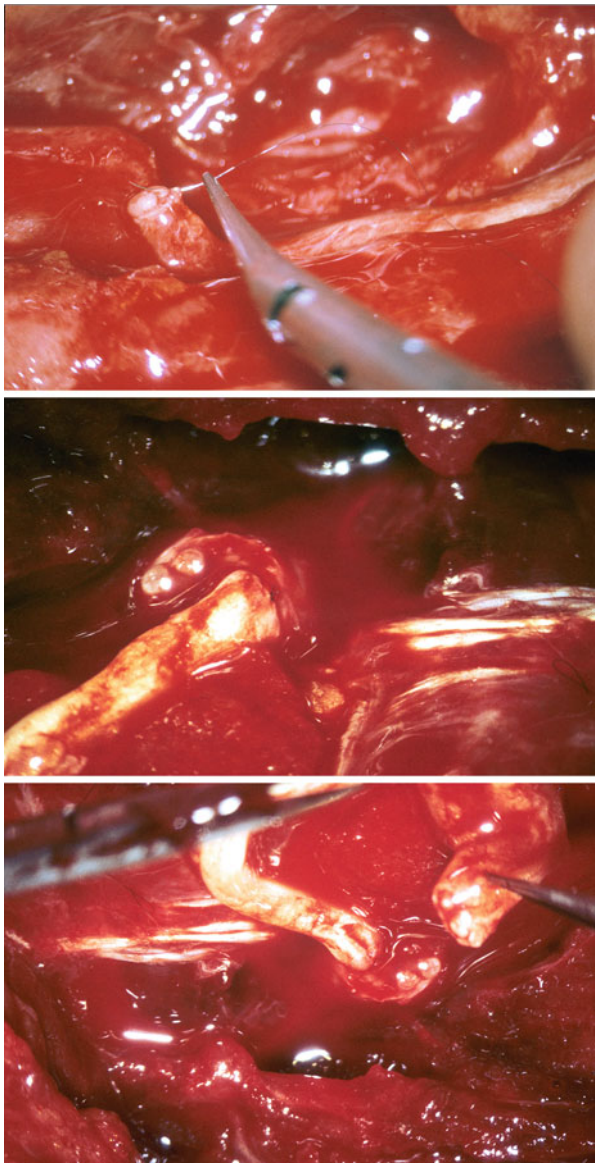


Fig. 5.22 In grafting the suture unites epineurium of graft to perineurium of the bundle

repair the median nerve in cases where both are otherwise irreparable and in MacCarty's technique the common peroneal nerve is used to bridge long gaps in the tibial nerve in otherwise hopeless injuries of the sciatic nerve. These techniques provide a full calibre graft which is not only vascularised but also predegenerate, and they remain valuable in the most severe injuries. The steps required are described using the sciatic nerve injured in the mid or lower part of the thigh as an example.

- At the first operation the proximal and distal stumps are identified and the gap between them measured.
- The proximal stumps of the tibial and common peroneal nerves are prepared and then sutured together.
- The proximal segment of the common peroneal nerve is traced and then the bundles within the trunk are cut across at a distance from the suture line equivalent to the gap already measured. *The blood vessels in the mesoneurium and in the epineurium are preserved. In effect a vascularised graft has been prepared.*
- At the second operation which is performed no sooner than 3 weeks after the first the proximal segment of the common peroneal nerve is sectioned and brought down to the distal stump of the tibial nerve to which it is sutured. Bleeding, which is at times quite copious, is seen at the face of the common peroneal graft when it is apposed to the distal tibial nerve.

Case report: An 18 year old woman sustained open fracture of mid shaft of femur in a rod traffic accident. Most of the skin of the lower part of the thigh was avulsed and there was much destruction of muscles. Extensive skin grafting proved necessary. The tibial nerve was repaired by the pedicle technique: a 24 cm long segment of the proximal common peroneal nerve was prepared and it proved a simple matter to separate this from the tibial division as far as the level of the neck of the femur. The proximal stumps of the tibial and of the common peroneal nerves were prepared and sutured. The bundles within the common peroneal nerve were sectioned 25 cm proximal to this suture line. Four weeks later the common peroneal graft was mobilised and sutured to the distal stump of the tibial nerve in the upper part of the leg. She regained flexion of the heel and of the toes to power MRC grade 4, warm and cool sensation in the sole of the foot and accurate localisation to the plantar skin without over reaction. There was recovery of sweating.

5.4.3 Indications for and Methods of Nerve Transfer

Nerve transfer, also known as neurotisation, or nerve crossing, involves the passing of nerve fibres from a healthy nerve to the distal stump of an injured nerve or directly to the target tissue. This principle can be applied in a number of ways.

- End to side transfer by suture of the distal stump of the injured nerve onto or within the epineurium of the healthy donor.
- Transection of a healthy donor nerve and transfer onto the distal stump of the injured nerve.
- Transfer of one or more healthy bundles within an uninjured donor to the recipient. This important technique rests on the functional segregation and topographical organization of nerve fibres within the donor nerve trunk so that it is possible to take one bundle, say, from the ulnar nerve to reinnervate the nerve to biceps without inflicting any significant loss of function within the hand. The method has wide application.

- Transfer of the proximal stump of a divided nerve onto the distal stump of another divided nerve.
- Direct muscular neurotisation. Sometimes a nerve is avulsed from the muscle. The musculocutaneous and the circumflex nerves are most commonly affected. The technique of implanting the working proximal stump of the nerve directly into the target muscle has considerable support from laboratory and clinical study [3]
- Nerve transfers work best when a healthy nerve, or a portion of a healthy nerve, is transferred to a nerve to muscle of roughly equivalent size without any interposed nerve graft. They cannot be asked to do too much; a rivulet cannot feed the Nile. Addas and Midha [1] provide a valuable review of this field. They say "nerve transfers tend to take the surgeon away from exploring the injury site, the brachial plexus, which carries the potential for surgeons to not even offer an anatomic nerve reconstruction, even in situations when these are perfectly appropriate…. With the increasing use of transfers, newly trained peripheral nerve surgeons are less likely to have exposure to the brachial plexus and they will be increasingly unfamiliar with the detailed anatomy and intraoperative electrophysiology assessment of the lesion".

  These are important warnings. Here are two more.

- There are far fewer MNF in the donor nerves than in the main trunks. The number of MNF in the spinal accessory nerve at the base of the posterior triangle is about 1,500; that number in C5, usually the smallest of the spinal nerves forming the brachial plexus, is at least 25,000.
- The deficit imposed on the patient must not be too severe. No nerve of vital function should be used for the sake of regaining a non vital function. Phrenic nerve palsy at birth is life threatening. Transfer of the hypoglossal nerve in infancy causes a high morbidity and disturbance of speech. Some adults experience serious ventilatory impairment following injury to the phrenic nerve complicating lesions of the brachial plexus. In one adult patient where both hypoglossal nerves were used there was very serious disturbance of speech and swallowing for as long as 6 months after operation.

The intercostal nerves: The patient is placed in a semi-sedentary position with preparation including the forequarter and the chest wall from the mid line to the iliac crest. An incision is made below the fold of pectoralis major extending to the axilla as a "Z". The serratus anterior is exposed, and the neurovascular pedicle to that muscle identified and protected.

The lateral perforating branches of the intercostal nerves are identified. The upper four digitations of the serratus anterior are released from the ribs, leaving a sufficient cuff of muscle for later repair. The muscle is reflected so opening the plane between the muscle and the rib cage. The cutaneous branch of the intercostal nerve is traced back to a narrow foramen in the external intercostal muscle. The muscle is released from the rib above as far as the posterior angle.

The deep division of the intercostal nerve is found by dividing the attachment of the middle intercostal muscle from the rib above. Gentle traction with a phrenic

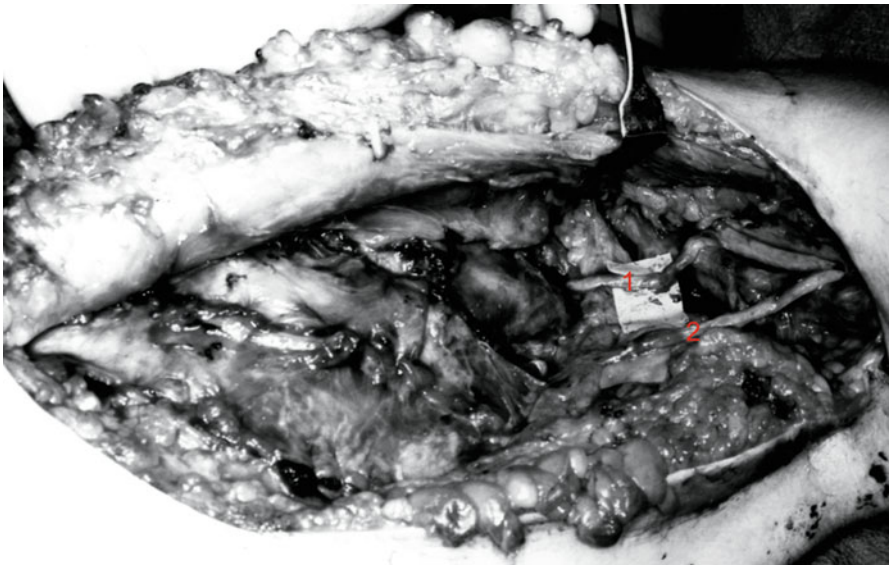


Fig. 5.23 Nerve transfer. Upper left intercostal nerves T2, T3, T4 (*1*) raised and united to the lateral cord of the plexus (*2*)

hook, underneath the upper rib, reveals the deep branch which is divided anteriorly. A Gelpi knee retractor can now be inserted to spread the rib cage. The deep division is traced back to its junction with the perforating branch. Elevation of the nerve becomes progressively easier. Bi-polar diathermy must be used throughout (Fig. 5.23).

The intercostal nerves from T3 to T6 are available by this method. These are brought to the anterior surface of serratus anterior through a tunnel in the upper part of the muscle. The serratus anterior is repaired.

If only one or two deep divisions of the intercostal nerves are being used for the nerve to serratus anterior, one of the most effective of all nerve repairs, then a suitably placed transverse incision in that muscle is adequate. It is wise always to see a radiograph of the chest before the patient leaves the operating theatre.

The spinal accessory nerve: This is a powerful motor and it should be used with discrimination. *The innervation of the upper one third of trapezius must not be compromised*. Even when the nerve is divided deep to the clavicle, the loss of function is less but it is not insignificant. The nerve is found at the lateral end of the transverse supraclavicular incision in the plane between the fat pad and the deep face of trapezius. The nerve passes down it in a rather sinuous fashion and is accompanied by a longitudinal artery and vein. These can cause troublesome bleeding. The nerve is joined by a branch from the cervical plexus just above the level of the clavicle and intra-operative stimulation of this branch only occasionally evokes a muscle twitch. The nerve is sectioned distal to that junction (Fig. 5.24).

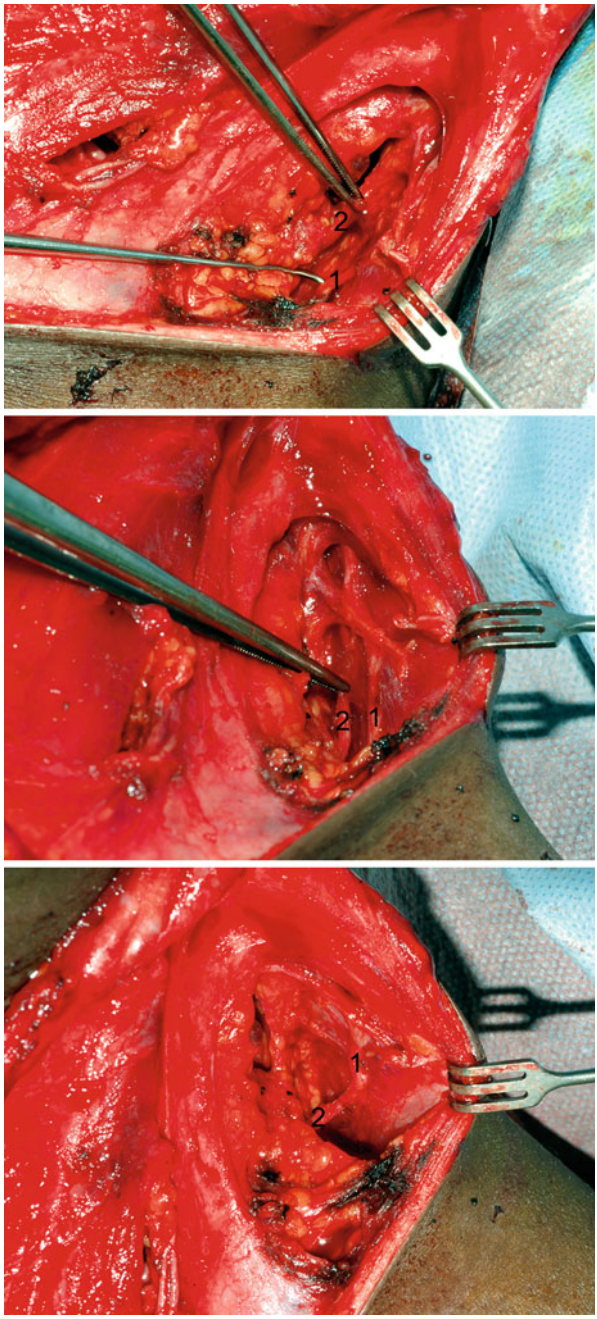


Fig. 5.24 Accessory (*1*) to suprascapular (*2*) transfer. *Above*, seen from the head: the spinal accessory nerve is displayed on the inner face of the trapezius at the base of the posterior triangle, *middle* and *below*, the view from the shoulder: the suprascapular nerve is passed deep to the fat pad and united to the spinal accessory nerve

Ulnar to biceps transfer is regularly effective in cases of avulsion of C5 and of C6. With care, it can be extended to cases where C5, C6, C7 or even C5, C6, C7 and C8 have been avulsed. Fastidious dissection of the bundles within the ulnar nerve is

necessary. The nerves are exposed through an incision along the length of the brachial bundle. The musculocutaneous nerve is identified. It is not uncommon to find that the nerves to biceps and brachialis arise directly from the median nerve. The nerve to biceps is accompanied by a sizeable artery and vein. It is traced proximally and separated from the main nerve. It is sectioned here so that it drops down onto the ulnar nerve. The epineurium of the ulnar nerve is incised and the bundles exposed (Fig. 5.25). Nerve stimulation used at very low intensity leads the surgeon to a bundle in the antero-lateral quadrant of the nerve passing to the flexor muscles of the forearm but not to the muscles of the hand. This is divided and an end-to-end suture is done. This valuable principle has been extended widely, using bundles from the median or the ulnar nerve to nerves to triceps, to the nerve to extensor carpi radialis brevis, or to reinnervate free functioning muscle grafts. It has proved particularly valuable in the repair of injuries to the brachial plexus in which some spinal nerves are intact whilst others are avulsed. In these a bundle from an intact nerve may be used to reinnervate the suprascapular nerve or an avulsed ventral root (Figs. 5.26, 5.27 and 5.28).

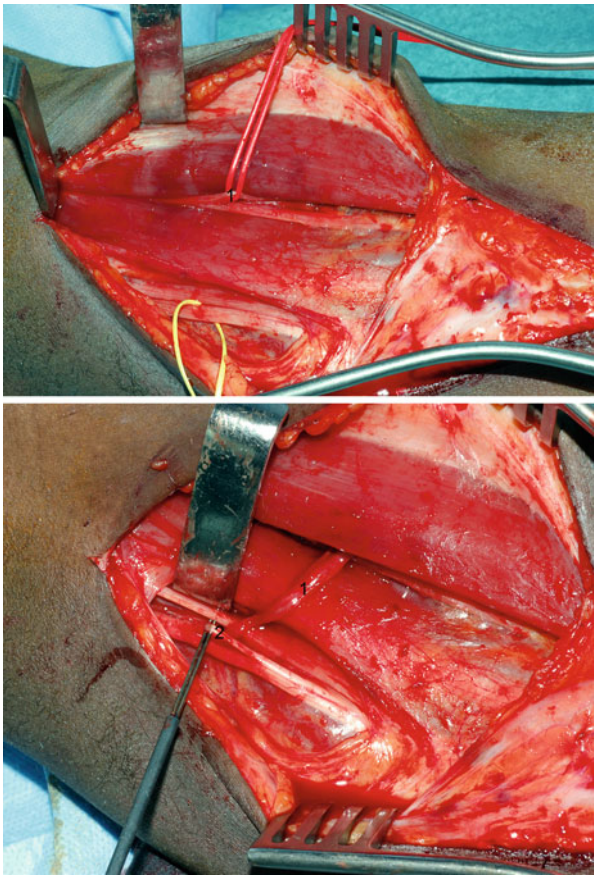


Fig. 5.25 Transfer of a bundle from the ulnar nerve (*2*) to the nerve to biceps (*1*)

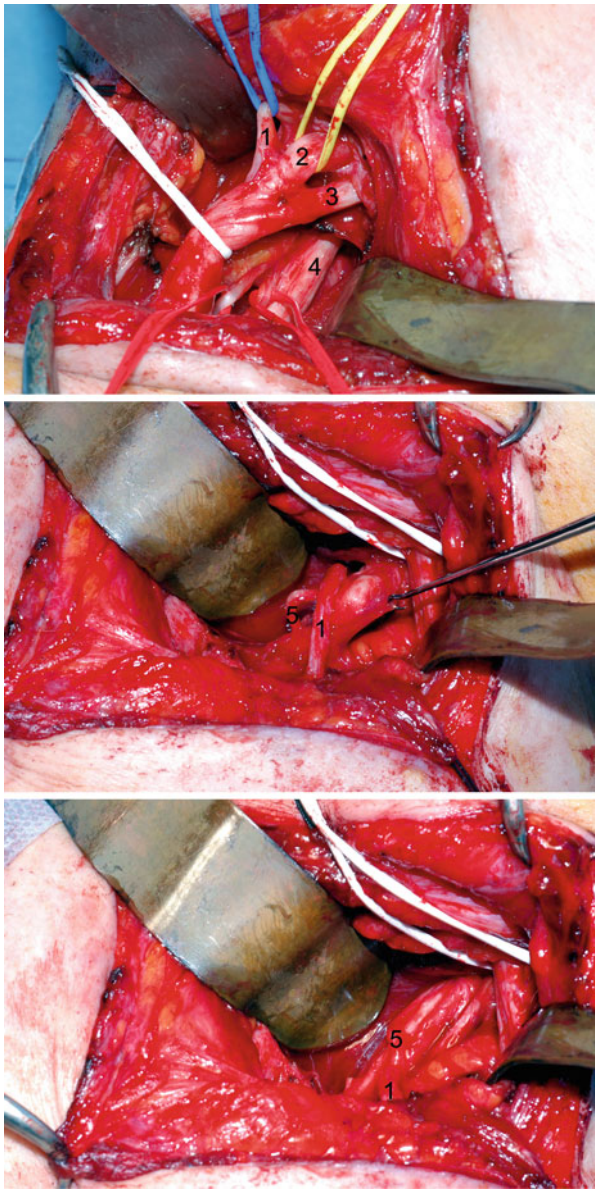


Fig. 5.26 Avulsion of C5 and of C6 with dorsal scapular palsy. The suprascapular nerve was reinnervated by one bundle from C7. The suprascapular nerve (*1*) posterior division of upper trunk (*2*) anterior division of upper trunk (*3*) C7 (*4*) and the selected bundle from C7 (*5*) are displayed

Direct muscular neurotisation is useful in lesions of the circumflex or musculocutaneous nerves in which the nerve has been avulsed directly from the muscle or, in the case of circumflex, the distal stump of the nerve is wrecked by fibrosis. The proximal stump is prepared in the usual way and two lengths of graft, usually the MCNF, are united to the proximal stump and then implanted into the muscle through short incisions through its sheath. The grafts are passed subcutaneously, and the

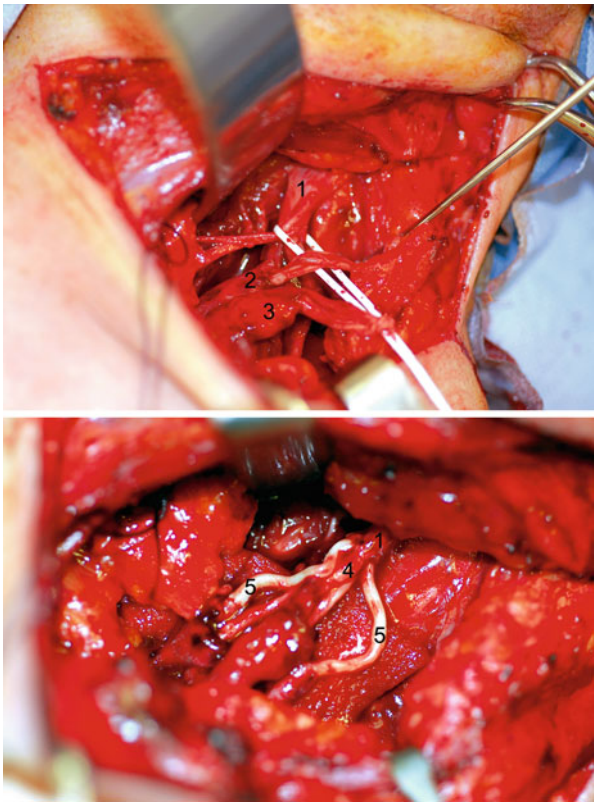


Fig. 5.27 Reinnervation of the avulsed ventral roots of C6 and C7 from the proximal stump of C5. The ventral root of C6 is apposed to the stump of C5. Grafts were passed to the ventral root of C7 and to suprascapular nerve. C5 (*1*), C6 (*2*), C7 (*3*), the ventral root of C6 (*4*) and the grafts (*5*) are seen

portal of entry is exposed by a short incision of the skin at those points. About ten portals of entry are fashioned using the terminal branches of the graft or by subdivision of the distal stump. The entry points of the implanted nerve are sealed with fibrin clot glue (Fig. 5.29).

5.4.4 Other, Non Neural, Material for Grafts: Entubation

Surgeons have for many years sought a source of material for graft to supplement or to replace the rather meagre stock provided by dispensable cutaneous nerves.

The freeze thawed muscle graft (*FTMG*) has been used extensively in laboratory and clinical studies. It has a place in the treatment of painful cutaneous neuromas [27]. Pereira et al. [22] treated the leprotic hand and foot by replacing the damaged segment of the median or tibial nerve with FTMG. Most patients recovered protective sensation; their ulcers healed. Doubtless, a number of amputations were prevented. One intriguing finding was the improvement in the condition of the skin in one third of the contralateral feet.

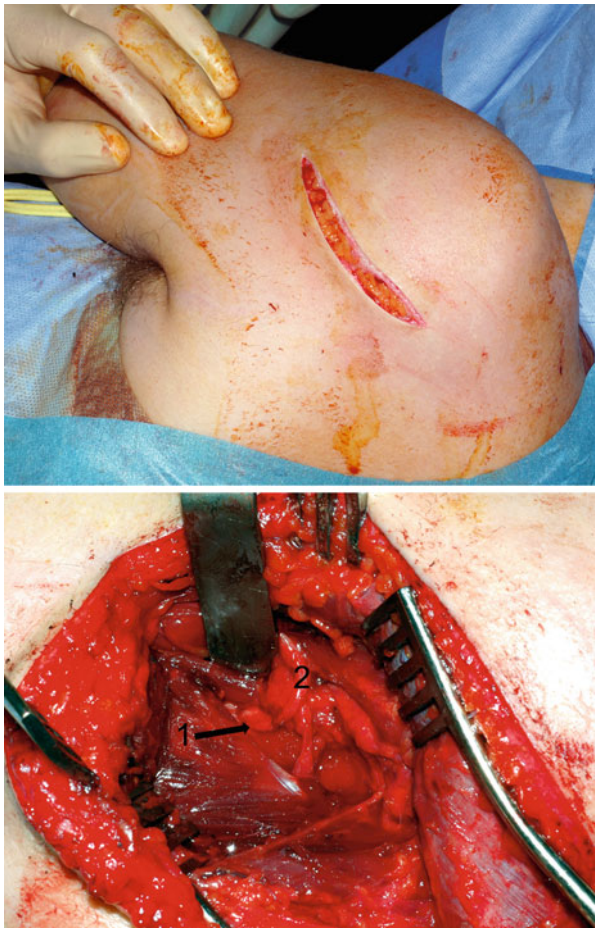


Fig. 5.28 Reinnervation of part of the circumflex nerve (*1*) by a nerve to long head of triceps (*2*)

The stumps of the nerve are prepared and the gap measured. A segment of adjacent muscle, of equivalent calibre, is excised. This should be at least two and a half times the length of the measured defect in the nerve. The muscle is then enveloped by a piece of aluminium foil and immersed in liquid nitrogen for about 60 s. The packet is then placed in distilled water for a couple of minutes. The prepared muscle graft can then be trimmed to appropriate length and breadth and it may be secured to the nerve stumps by suture and then sealed by fibrin clot glue. Fragmentation is usual with muscle grafts more than 3 cm long.

Entubation: In 1997 Lundborg and his colleagues [18] published a significant paper describing a prospective randomised trial comparing silicone entubation and suture of median and ulnar nerves in the forearms of 18 patients. Recovery was studied most thoroughly and no significant difference in outcome between the two groups was noticed. The proposed advantages following placing of nerve stumps within a silicone tube, so providing a "chamber" separate from the surrounding tissues,

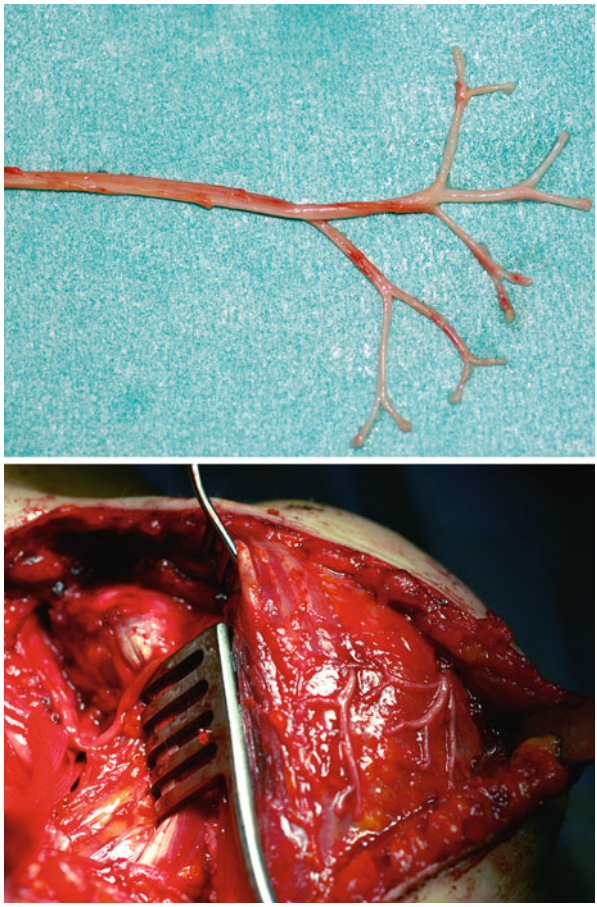


Fig. 5.29 Muscular "neurotisation". *Above*: the nerve graft is prepared. *Below*: it is passed from the proximal stump of the circumflex nerve and implanted into the muscle

include: the local accumulation of neurotrophic factors; the longitudinal orientation of fibrin matrix within the tube; and the possibility for the regenerating axons to be better guided into distal Schwann tubes across the gap. The tube must not be too tight. Silicone entubation may cause constriction and fibrosis of a sutured nerve.

It seems that the upper limit of a defect reparable by these methods is 3 cm. Repair of the long gap by means other than autogenous graft remains elusive. De Ruiter et al. [8] conclude that there is, as yet, little evidence demonstrating the superiority of empty, hollow biodegradable nerve tubes over suture or autografting.

5.4.5 Immobilisation

Repaired nerves need protection during at least the first 3 weeks after repair. In most cases, a simple plaster slab gives sufficient protection. Only in the case of extensive

Fig. 5.30 Sling for control of the fore quarter after repair of the brachial plexus. Note the check on lateral rotation at the gleno-humeral joint

proximal lesions of the whole sciatic nerve is elaborate protection required. Narrowing of the gap to permit end to end suture or adequate repair by graft requires flexion of the knee and avoidance of flexion of the hip. The necessary position of immobilisation in a hip spica is awkward and uncomfortable; further, gradual extension of the knee after 3 weeks may be necessary in order to protect the line of the repair. Usually, it is sufficient when the splint is removed to warn the patient about the danger of excessive movement of related joints, and to rely on the patient steadily to restore movement of those joints. There is particular difficulty in protecting repairs of the brachial plexus. At present, in adults, a sling with straps is used, which secures the arm across the body supplemented with a soft cervical collar. A plaster of Paris "cocoon" is applied after repair of the plexus in infants (Fig. 5.30).

The method used in the post operative care of severe wounds at the wrist is as follows (Fig. 5.31).

- The splint holds the elbow at 90° of flexion, the wrist at between 30 and 40° of flexion, the metacarpophalangeal (MCP) joints at about 70° of flexion, and the proximal interphalangeal (PIP) joints at no more than 30° of flexion. The dorsal splint extends to the tips of the fingers and the palmar splint to the PIP joints only. The splints are bandaged so that there is restriction but not rigid immobilization. Gentle, active flexion of the fingers and the thumb is encouraged from the

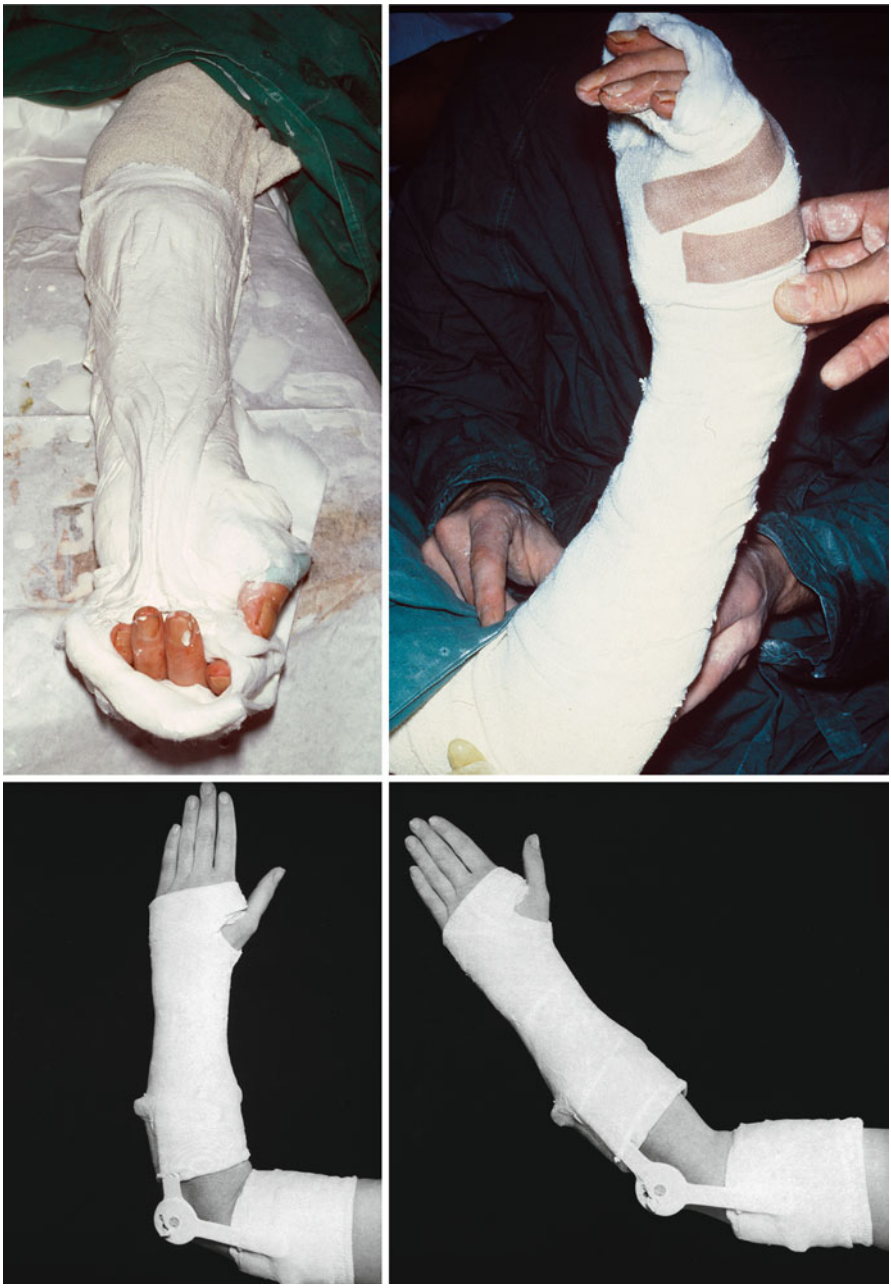


Fig. 5.31 Protection after repair. *Above*: plaster after repair of both nerves and flexor tendons at the wrist. *Below*: hinged plaster with adjustable check

outset. The arm is supported in a sling, but there should be encouragement of gentle active lateral rotation and of elevation of the shoulder to 90° from the first postoperative day.

- At 3 weeks the splints and the sutures are removed. The next splint does not restrict the elbow. The wrist is splinted to prevent extension beyond 20°. The dorsal hood, which again extends to the tips of the fingers, blocks the MCP joints to 30° of flexion and the PIP joints to 30° of flexion. Increasingly, vigorous active flexion of the fingers and the thumb is now permitted and gentle active flexion at the wrist is also encouraged within the confines of the splint, bandaged as it is to the forearm and hand.
- In direct sutures of nerves in the elbow region a hinged splint is applied at 3 weeks from operation (Fig. 5.31). This permits active flexion but blocks extension. The range of permitted movement is increased at weekly intervals by adjusting the hinge.
- By 6 weeks, splints are discarded and vigorous active flexion work against resistance is introduced. Now, gentle passive stretching work can be introduced for the fingers and the thumb.

It is of course very important during the period of recovery to maintain the mobility of the joints some or all of whose governing muscles are paralysed and to warn the patients of the danger, especially in cold weather, of accidental damage to the anaesthetic skin. Most of the work on passive movements of the joints can be done by the patient or by his or her parents, but weekly supervision by a physiotherapist is useful in keeping the mind concentrated on the work during the long march towards recovery. In the case of the metacarpo-phalangeal joints of the fingers and the carpo-metacarpal joint of the thumb, "lively" splints are useful, but the problem of the stiff metacarpo-phalangeal joint in the paralysed hand persists. Accurate diagnosis is the foundation of rehabilitation and operations for repair of the nerves should be seen as but the first stage in that process. It should be superfluous to add that the sooner the diagnosis is established the better, and that urgent efforts must be made to improve the prognosis.

5.5 Approaches to Individual Nerves: Neck and Upper Limb

5.5.1 The Transverse Supraclavicular Approach: (Anterior, or Anterolateral)

This is used for exposure of the supraclavicular part of the brachial plexus. Its disadvantage is that the vertebral artery stands between the operator and the most proximal parts of the nerves. The risk of skin necrosis is negligible. The scar is reasonable. The length and the level of the incision are modified according to the lesion. In the urgent case the incision extends from beyond the mid line to beyond the anterior fold of the trapezius and is placed just above the clavicle. For lesions of the upper

trunk of the brachial plexus the incision is shorter and is placed about two fingers breadth above the clavicle. The exposure of Fiolle and Delmas [10] is achieved by adding a vertical limb to the transverse supraclavicular wound. The transclavicular approach [5] is, in effect, a medial extension of the transverse supraclavicular approach and it gives excellent access to the first part of the subclavian artery, the first part of the vertebral artery and the whole of the brachial plexus.

The operation: The patient lies supine in a semi-sedentary position, with the head elevated to about 30° and the neck in extension. After placing recording electrodes to the scalp and the skin of the neck, the head is bandaged to a neurosurgical rest. The neck is extended but not rotated. The area of skin preparation includes the whole of the forequarter, extending to the jaw line and the ear, beyond the mid line to the inferior rib margin.

The skin flap is developed deep to the platysma (Fig. 5.32). This is easier to identify at the posterior margin of the sternocleidomastoid muscle. Below, the flap is developed to reveal the inferior margin of the clavicle; above as far as the greater auricular and transverse cervical nerves crossing the anterior face of ster-

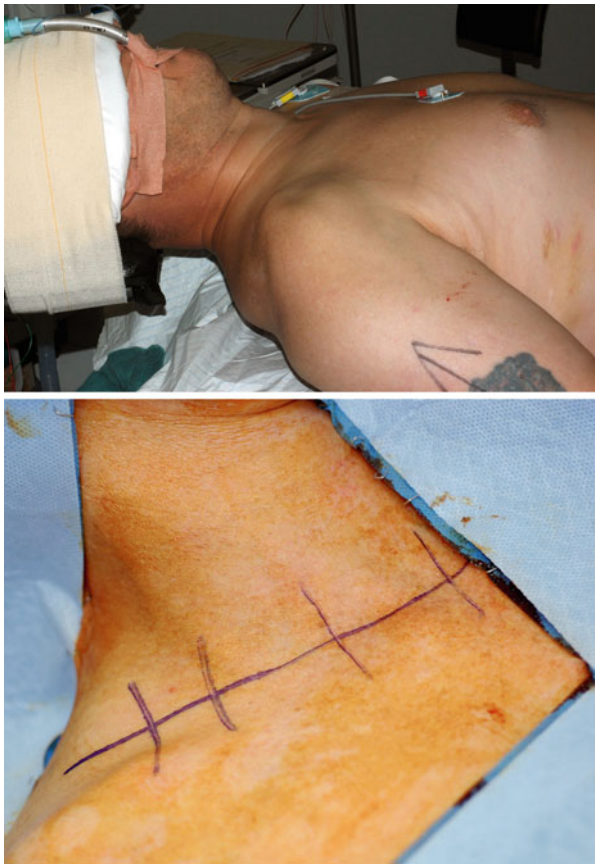


Fig. 5.32 The transverse supraclavicular approach to the brachial plexus. Note the position of the patient and the line of incision

nocleidomastoid (SCM). The incision is deepened in the plane between external jugular vein and the SCM, displacing the supraclavicular nerves posteriorly. The lateral part of the insertion of the muscle can be elevated from the clavicle. The fat pad and the omohyoid muscle are now seen. The omohyoid is divided between stay sutures and reflected. The fat pad may present as two leaves, split by branches of the transverse cervical vessels. These must be preserved in cases of rupture of the subclavian artery because of their contribution to the collateral circulation (Fig. 5.33).

The scalenus anterior is exposed and the phrenic nerve is seen coursing obliquely across it. It is mobilised and held in nerve sling. Access to bony structures behind the plexus should be achieved by separating the component nerves rather than by forward retraction of the plexus. Following the phrenic nerve cranially brings the surgeon to the fifth cervical nerve and it is sometimes helpful to work on the medial side of the phrenic nerve to expose a ruptured stump at C5 or C6. The upper trunk and the suprascapular nerve are traced. After traction injury it may be easier to identify the suprascapular nerve first and follow it back to the upper trunk. The seventh cervical nerve can now be seen behind the upper trunk. Division of scalenus anterior

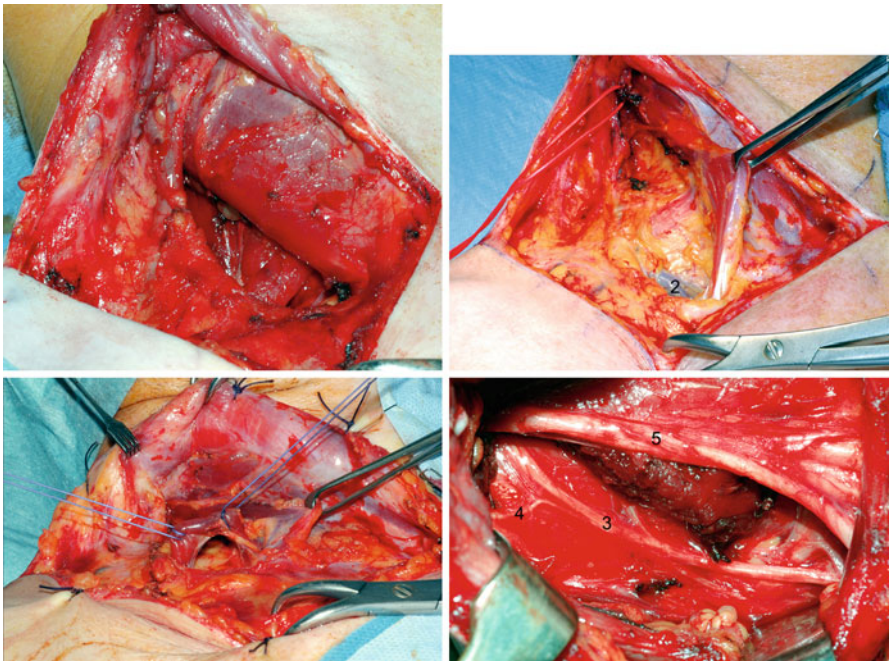


Fig. 5.33 *Above left*: the posterior margin of sternocleidomastoid is defined, anterior to the external jugular vein and supraclavicular nerves (*1*). *Above right*: this is retracted to display the fat pad and transverse cervical vessels (*2*). *Below left*: the omohyoid is divided between stay sutures. *Below right*: the nerve to serratus anterior (*3*) is displayed by section of scalenus medius deep to the upper trunk (*5*). The largest ramus comes from C6. There is often a communicating branch from the dorsal scapular nerve (*4*)

reveals the subclavian artery; the plane between the muscle and the subclavian artery requires careful definition. We have seen the subclavian artery crossing anterior to scalenus anterior in three cases and through it in seven more. The eighth cervical and first thoracic nerves are traced by following the plane between the artery and the lower trunk. The nerve to serratus anterior lies lateral and deep to the upper trunk, behind the suprascapular nerve and posterior to scalenus medius. The rami forming this nerve are displayed by dividing scalenus medius. There are usually three, usually that from C6 is the largest. The spinal accessory nerve, is identified running in a vertical and sinuous manner on the deep face of trapezius. The dorsal scapular nerve can be found passing posteriorly away from C5.

Some key points:

- Neck and head should be extended but not rotated
- Place towels to expose the whole of the posterior triangle, including manubrium, the mandible, the lower part of the ear, the upper margin of trapezius and the whole of the upper limb.
- Skin flaps include the platysma
- Define the posterior margin of SCM and work between it and the fat pad. This protects the supraclavicular nerves
- Identify and reflect omohyoid and ligate the transverse cervical vessels.
- Identify the phrenic nerve in front of scalenus anterior.
- For the exposure of C8, T1, and the proximal subclavian artery divide scalenus anterior WITH GREAT CARE

Extension by the operation of Fiolle and Delmas [10]: This affords full display of the supra, retro and infra clavicular plexus, of the second part of the subclavian to the terminal axillary artery and of the subclavian and axillary veins deep to the clavicle. The originators said "the exposure of the vessels can be achieved in three minutes" and this is possible in an emergency. There is no doubt that the operation secures rapid access to and control of major vessels from the scalenus anterior to the lowest part of the axilla. The plexus itself is exposed from the spinal nerves above to the terminal branches below. It is especially valuable in fresh cases of laceration or rupture of the great vessels deep to or below the clavicle and in later cases of false aneurysm, and is often necessary for the delayed exposure of ruptured nerves after primary vascular repair. It is the exposure of choice in the closed infraclavicular rupture of vessels and nerves. The incision is T-shaped, the vertical limb running in the delto-pectoral groove, retracting the cephalic vein medially, then curving into the apex of the axilla underneath the fold of pectoralis major. In urgent cases a Gigli saw is used to divide the clavicle at the lateral edge of SCM: otherwise, a hole is drilled and prepared for a compression screw, inclined at 45° to the long axis of the clavicle. Subsequent fixation is made easier by contouring a plate to the bone and drilling and preparing one hole in the lateral fragment. The clavicle is cut at 90° to the axis of the hole for the compression screw. It is possible, in some cases, to avoid cutting the clavicle. The bone can be drawn upwards or downwards by a nylon tape (Figs. 5.34 and 5.35).

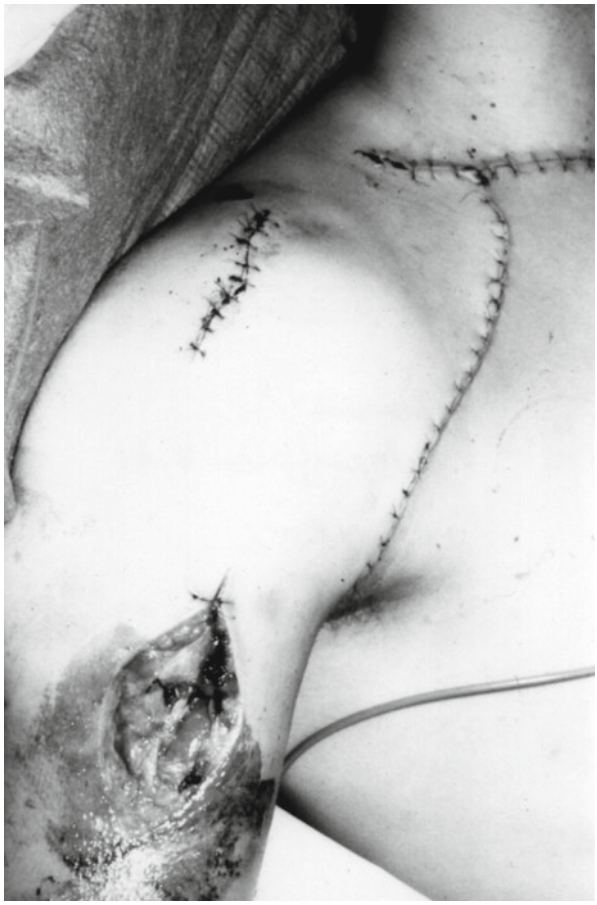


Fig. 5.34 The Fiolle Delmas exposure. The incision at the shoulder was made for the purpose of inserting an intramedullary nail. The wound, caused by the fracture, has been left open

The fascia at the root of the axilla is divided and the finger develops a plane between pectoralis major and minor in front, and the axillary bundle behind. Pectoralis major is detached from the humerus. Pectoralis minor is reflected taking care for the musculocutaneous nerve. The limb is rolled into lateral rotation so that the trunks, divisions and cords of the brachial plexus are displayed with their accompanying vessels. The exposure has been used in more than 250 cases. There have been nine cases of non union of the clavicle; infection complicated three of these.

Some key points:

- Identify and preserve the cephalic vein
- Identify the groove between pectoralis major and deltoid which is often easier close to the clavicle
- Use a malleable retractor between the clavicle and subclavius muscle before dividing it.
- The subclavius muscle is cut between clamps. LOOK OUT FOR THE SUPRASCAPULAR VESSELS

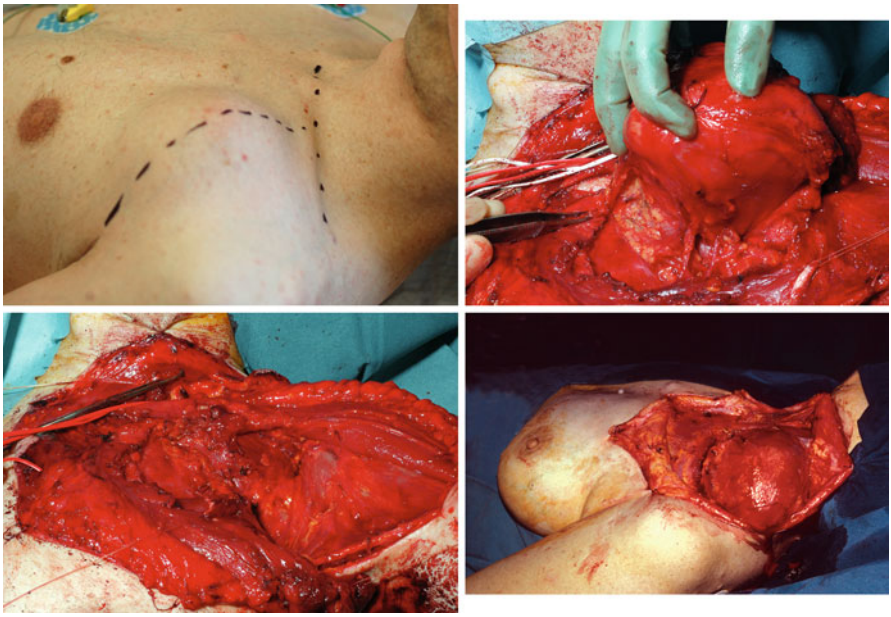


Fig. 5.35 Two large neurofibromata exposed through the approach of Fiolle and Delmas. *Top left*: the skin incision. *Bottom left*: the tumour exposed and, *top right*: removed. There was no loss of function. *Bottom right*: another neurofibroma arising in the posterior triangle of the neck. It was not necessary to divide the clavicle in either of these cases

- The easiest way to define the interval between the pectoralis minor and the coraco brachialis is to pass the index finger behind the upper margin of pectoralis minor and feel for the interval.

5.5.2 The Transclavicular Exposure

The approach was designed to give adequate exposure and control of the venous trifurcation, the first part of subclavian artery, the vertebral artery and the recurrent laryngeal nerve. the intervention remains an extensive and difficult one calling for a high degree of anatomical knowledge and practical versatility. The exposure rests on the elevation of the osseo-muscular flap comprising the medial portion of clavicle with the sterno clavicular joint based on the sternocleidomastoid muscle (SCM). The whole of the brachial plexus can be displayed after some lateral work; the cervico dorsal spine can be seen from C3 to T3 by developing the plane between the carotid sheath and the visceral axis (Fig. 5.36).

The transverse limb of the incision runs from the fold of the trapezius on the side of operation to the mid point of the opposite posterior triangle. This limb can be extended to increase the exposure. The vertical limb runs to the sternal angle.

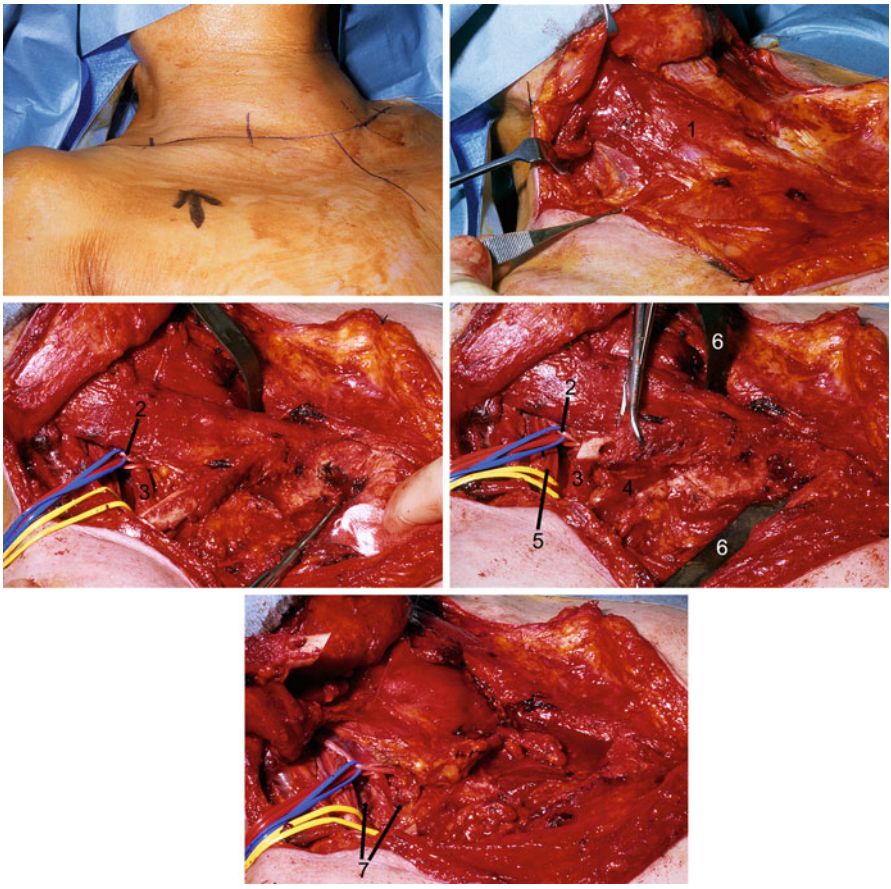


Fig. 5.36 The transclavicular exposure. *Top left*: the position of the patient and line of incision. *Top right*: elevation of the skin flaps with platysma and definition of sternocleidomastoid muscle (*1*). *Middle left*: the clavicle exposed and the phrenic nerve (*2*) and subclavian artery (*3*) traced. *Middle right*: the bone flap is elevated, taking care for the subclavian vein (*4*). The trunks of the brachial plexus (*5*) are seen. Malleable retractors (*6*) are inserted deep to the manubrium before section of the first costochondral junction and division of the manubrium. *Bottom*: the tumour (*7*) is seen deep to the subclavian artery, where it enveloped the vertebral artery

The flaps are widely elevated to include platysma. The SCM is defined anteriorly and posteriorly from its insertion below to the uppermost limit of the wound above. The accessory nerve is identified. The clavicle is displayed subperiosteally at its middle point. The subclavius muscle and suprascapular vessels are divided. The omohyoid muscle is divided and reflected. Pectoralis major is detached from the inferior portion of the medial clavicle revealing the subclavian vein. The strap muscles are released from the notch of the manubrium and the plane deep to this developed carefully using a dental Howarth elevator and a curved Adson's dissector. The plane is enlarged with the finger and a malleable retractor passed. The first

costochondral junction is defined after detaching pectoralis major from the adjacent manubrium and the interval between the first and second sternocostal junction is developed deep to the sternum by the means outlined above. A malleable retractor is passed to meet its fellow from above. The first costal cartilage is cut with a scalpel, a fine osteotome is used to divide the manubrium with an L-shaped cut. The clavicle is now divided and the medial clavicle, sternoclavicular joint and upper corner of the manubrium are elevated on the SCM. The residual strap muscles are released. The internal jugular vein is now seen and traced to its junction with the subclavian vein. The brachiocephalic vein is traced and lightly held in a vascular sling. The phrenic nerve is elevated from scalenus anterior and the muscle divided. The first part of subclavian artery, the vertebral artery and the recurrent laryngeal and vagus nerves are seen. To display the whole of the plexus pectoralis major and pectoralis minor are detached from the humerus. When it comes to closure, the manubrio-clavicular fragment is reattached to the manubrium with wires, and to the clavicle with a plate and screws. The soft tissue layers are carefully closed. This exposure has been used on 80 occasions, chiefly for tumours of the brachial plexus. One patient, a 62 year old man, with a malignant neurofibroma complicating neurofibromatosis type 1 died of consumptive coagulopathy. Osteomyelitis of the clavicle occurred in two cases, and non union in four more. The recurrent laryngeal nerve is more vulnerable on the right side. Damage to the thoracic duct can usually be avoided, except in a scarred field and is usually recognised because of the leak of milky fluid. It is best to identify and repair the lesion, because if the pleura has been opened, there is a real danger of formation of a chylothorax. If the hole cannot be repaired, it can be plugged with a muscle graft.

Some key points:

- This is a major intervention and each step should be carried out with care under direct vision.
- Use malleable retractors behind the clavicle and deep to the manubrium.
- Detach pectoralis major from inferior clavicle with care, to expose subclavian vein crossing the first rib.

5.5.3 The Postero-Lateral Route

This, the posterior subscapular route, provides very good access to the most proximal parts of the nerves and in particular to the nerves in the intervertebral foramina [15]. The patient is almost supine, with the limb on the affected side resting on a separate table. The incision is convex medially, truly parascapular. The scapula is freed by the division of the trapezius and rhomboid muscles and, if necessary, the levator scapulae and it is retracted laterally so that the upper ribs are exposed. The first rib is defined and removed extra-periosteally, if necessary after the second rib has been removed sub-periosteally to facilitate exposure. The scalenus medius muscle is partly liberated during the removal of the first rib, and further mobilisation

permits its upward retraction to display the brachial plexus. It is necessary to avoid damage to the nerve to serratus anterior during this process. By medial retraction of the posterior paravertebral muscles the foramina can be opened by hemi-laminectomy. Closure after completion of the procedure is by re-uniting the muscles carefully over a drain or drains.

There are three *potential* complications:

- winging of the scapula,
- instability of the cervical spine if more than two facet joints are removed,
- damage to various related structures.

The approach is firmly indicated in:

- the thoracic outlet syndrome: prior operation by the transaxillary or supraclavicular routes for removal of the first thoracic or seventh cervical rib, when the posterior third of the rib remains;
- tumours of the plexus: tumours with intraforaminal and extra foraminal lateral components;
- radiation neuropathy when there is extensive change in the skin and deep tissues of the neck and chest wall;
- traumatic lesions when the evidence is that a reparable lesion is in or near the foramen.

The approach described affords a less extensive view to the more distal (lateral) part of the supraclavicular plexus than does Kline's [15] (Fig. 5.37). The patient is put in the lateral position, the affected side uppermost, and the limb is included in the field. The incision, convex laterally, is centred over the seventh cervical vertebral spine. The flaps are raised. The trapezius is divided close to the midline and the muscle is retracted laterally. The next layer then comes into view: the upper part of the rhomboids, and the lower part of the splenius capitis. The levator scapulae, running from the scapula to the upper cervical transverse processes, and the splenius cervicis, running from the third to the sixth thoracic vertebral spines to the upper cervical transverse processes, are rather lateral. The upper part of the rhomboids and the lower part of the splenius capitis are divided near the mid line and the erector spinae group is exposed. Lateral to this, the transverse processes of the first thoracic and lowest four cervical vertebrae can be felt. The transverse processes and lateral masses are exposed by blunt dissection, with medial retraction of the erector spinae group. The back of the first rib is exposed at the lower end of the field, and the scalenus medius is cleared from its upper surface. That muscle is detached from its origin on the posterior tubercles of the lowest three of four cervical transverse processes. The nerves are then shown distal to the transverse processes, the proximal branches of the fifth, sixth and seventh nerves going into the scalenus medius to form the nerve to the serratus anterior. Now the posterior tubercles of the fifth and sixth vertebrae and part of the transverse process of the seventh are nibbled away to show the most proximal parts of the nerves. If necessary, the first thoracic transverse process and the first rib can be removed to increase the exposure of the lowest part

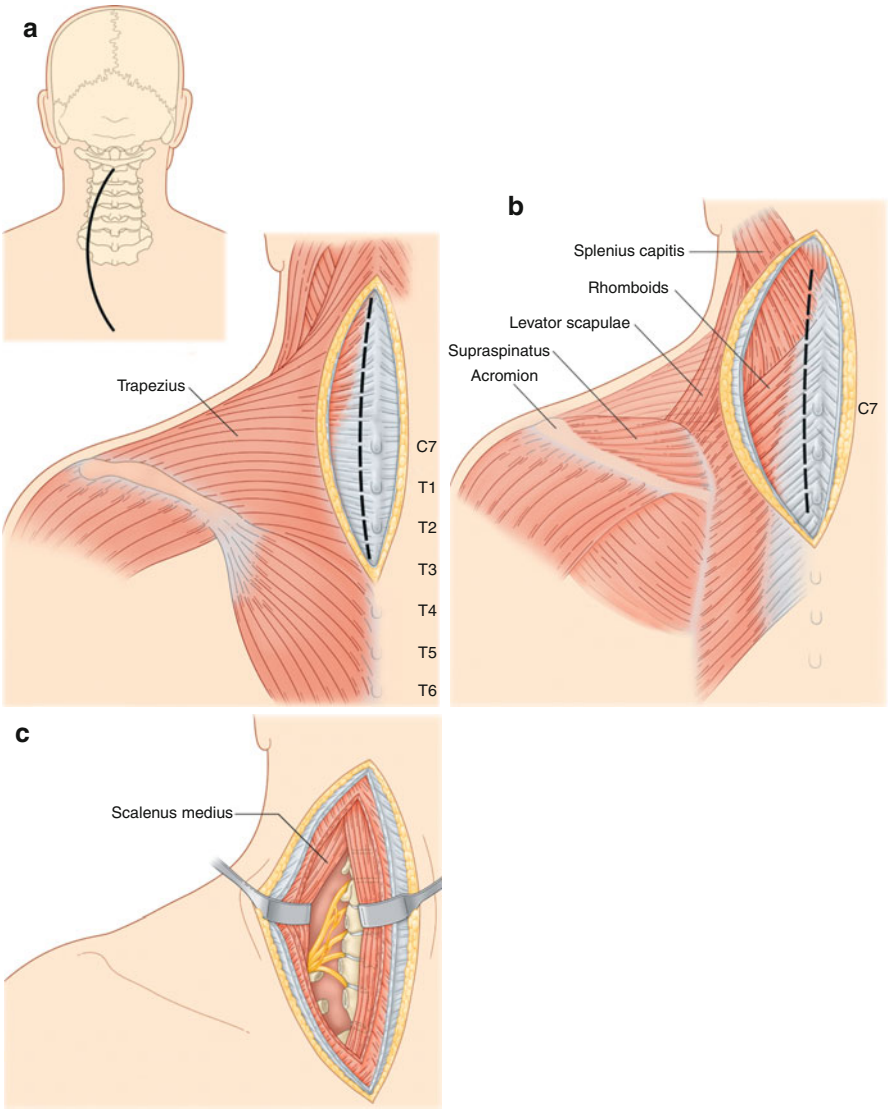


Fig. 5.37 Posterior approach to the left brachial plexus. (**a**) The incision. (**b**) The division of the trapezius. (**c**) Removal of posterior elements and detachment of scalenus medius to expose the proximal part of the plexus. Note that the nerve to serratus anterior is shown. In many cases of proximal injury to the plexus, it will have been damaged with the main nerves

of the plexus. Later, if necessary, one or two interfacetal joints can be removed to show the nerves in their dural sheaths in the foramina.

It is necessary to proceed carefully and methodically in this exposure, securing good haemostasis at each stage. It is not an easy exposure, but it is the preferred route when there is a very proximal lesion and access by the anterior route is barred

by the sclerosis of a previous intervention. Two muscle layers and one subcutaneous layer are closed over a suction drain. A description of the approaches for the roots of the spinal nerves after avulsion is available [3].

5.5.4 *The Spinal Accessory Nerve*

The incision is often dictated by the site through which the nerve was originally damaged. Often enough, the original incision can be extended either as a "Z" or in the direction of the lines of skin tension. The usual site of injury is in the posterior triangle between the sternocleidomastoid muscle (SCM) and trapezius muscles, though occasionally the nerve is wounded in its course through the former muscle or proximal to it. The greater auricular nerve is the key to the exposure of the proximal stump which emerges from behind the SCM about 5 mm cephalad. The nerve emerges from deep to the muscle as one trunk, and at this point a fine branch is seen which innervates the uppermost part of the upper one third of the trapezius. This must be respected. The course and relations of the nerve are remarkably constant (Fig. 5.38).

The incision extends to the anterior face of the SCM, displaying the greater auricular and transverse cervical nerves. Exposure of the accessory nerve should begin in unscarred tissue, and the lesion defined after proximal and distal trunks have been found. The proximal stump may have retracted deep to the SCM. It can be identified anterior to the muscle or branches to the SCM are identified with a nerve stimulator. The distal part is found beneath the anterior part of the upper fibres of the trapezius where it must not be confused with branches of the supraclavicular plexus which pass obliquely or horizontally in front. Atrophy of the distal trunk is a common finding because of the long delay before diagnosis. There is no point in attempting end-to-end suture after resection: the gap is usually too great, and in any case, the mobility of the forequarter on the axial skeleton forbids the use of such a

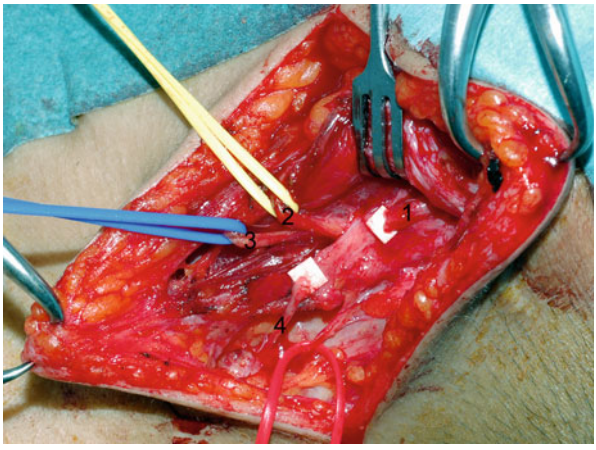


Fig. 5.38 The spinal accessory nerve emerges, as one trunk (*1*), from deep to sternocleidomastoid about 5 mm cephalad to the greater auricular (*2*) and transverse cervical (*3*) nerves. In this case the nerve had been transected 15 months earlier and there is atrophy of the distal stump (*4*)

method because of the hazards of disruption. If the proximal stump is inaccessible part of the lateral pectoral nerve may be used to reinnervate the distal trunk.

Some key points:

- Find the anterior face of SCM and identify the greater auricular and transverse cervical nerves.
- Inferiorly the nerve has a characteristic sinuous course, deep the fascia covering the inner face of the trapezius. It is accompanied by a vein which can prove troublesome.

5.5.5 The Suprascapular Nerve

The lateral position and a transverse incision is preferred. The flaps are raised, and the supraspinous part of the trapezius muscle is detached from its origin and raised to show the supraspinatus muscle, or the fibres of trapezius can be split to show the underlying supraspinatus, which is lifted from its scapular origin to reveal the suprascapular nerve traversing the notch. The accompanying artery is usually superficial to the transverse ligament or bony bridge. Ochiai et al. [20] showed that the suprascapular nerve might be seriously damaged in several places and describe an exposure which exposes the nerve along its entire course as far as the infraspinatus and which can be extended to display the circumflex nerve anteriorly.

5.5.6 The Infraclavicular Part of the Brachial Plexus

With the full opening of the delto-pectoral interval the pectoralis minor muscle is exposed crossing the field to its attachment on the coracoid process. Below and above it is the neurovascular bundle, covered by a layer of fascia and, in the lower part of the field, by the flat reflected tendon of the sternal part of the pectoralis major. The easiest way to define the interval between the pectoralis minor and the coraco brachialis is to pass the index finger behind the upper margin of pectoralis minor and feel for the interval. The tendon of pectoralis minor is divided. The muscle is drawn medially, care being taken of the medial pectoral nerve piercing it and going on to the pectoralis major. Now the fascia over the plexus is divided above and below the former site of the pectoralis minor. If it is absolutely necessary, the flat reflected tendon of the pectoralis major too can be divided. Thus the whole neurovascular bundle is displayed. The lateral cord is the most prominent component, the axillary artery is behind it and the axillary vein medial to it. In the lower part of the field the median nerve is formed from the contributions from the medial and lateral cords. Just at the formation of the lateral cord the lateral pectoral nerve arises to pierce the clavipectoral fascia and enter the pectoralis major. The posterior cord lies deep to the lateral cord and axillary artery; the medial cord is deep to the axillary vein. Some mobilisation of both great vessels is necessary for the full display of the cords (Fig. 5.39).

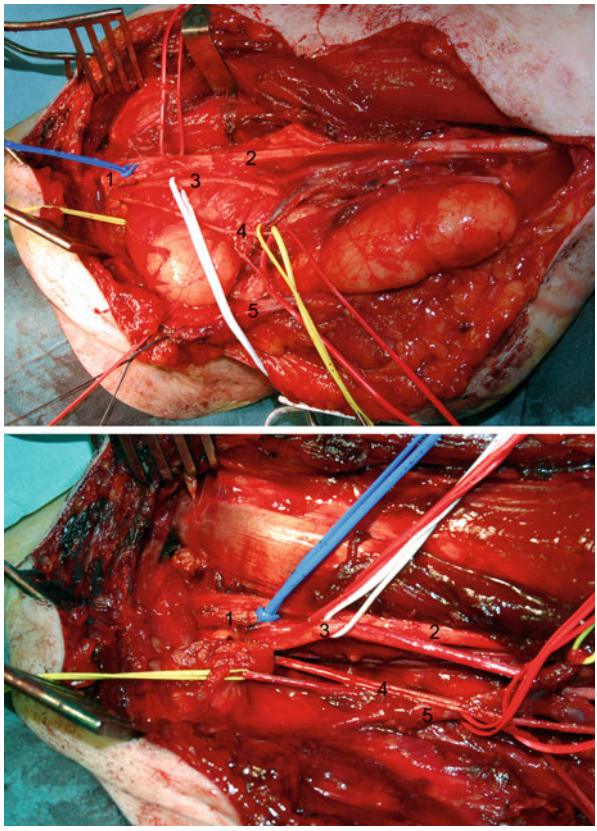


Fig. 5.39 The infraclavicular brachial plexus splayed over a massive lipoma. The axillary artery (*1*) is marked by the blue sling, the median nerve (*2*) by a red sling and the ulnar nerve (*3*) by a white sling. The medial cutaneous nerve of forearm (*4*) is seen and the radial nerve (*5*) lay deep to the tumour. The appearances before (*above*) and after (*below*) removal of the tumour

The musculocutaneous nerve arises from the lateral cord above the level of the coracoid process and runs laterally into the coraco-brachialis muscle. It may be a single branch but can consist of several branches arising at different levels from the cord.

The posterior cord and its lateral and terminal branches are exposed between the lateral cord and the axillary artery. The three subscapular nerves are seen, and in the lower part of the field the separation of the trunk into radial and circumflex nerves is visible. Reflection of coracobrachialis from the tip of the coracoid process improves exposure of the anterior "door" of the quadrilateral space and permits exposure of the distal stump of a ruptured circumflex nerve. This display may be extremely difficult in the late case, especially after arterial injury.

Some key points:

- Identify and preserve the cephalic vein.
- Definition of the delto pectoral groove may be easier just below the clavicle.
- Variations in the course of the axillary artery and the musculocutaneous nerve are common.
- This exposure can be extremely difficult in delayed cases with arterial injury. Do not hesitate to extend the incision.

5.5.7 The Circumflex Nerve

In cases of rupture the anterior (proximal) stump is almost always found just below the coracoid process and repair sometimes requires exposure of the posterior (distal) stump through a separate posterior incision. Repair has to be effected by graft, and it is certainly easier to secure good placement and attachment through two incisions than through one giving limited access. The posterior circumflex artery is often ruptured by the head of humerus, in anterior dislocation. This can lead to serious bleeding, or to a false aneurysm. The distal stump of the nerve may be strangled in fibrosis and in these cases direct muscular neurotisation is useful as it is when the nerve has been avulsed from the muscle (Fig. 5.40).

5.5.8 Median and Ulnar Nerves in the Arm and the Axilla

The incision crosses the anterior axillary fold and the axilla, and descends the medial side of the arm. The flaps are raised and the axillary fat displaced downwards. The neurovascular bundle is found in its sheath in the lower part of the axilla: most medial is the axillary/brachial vein; the nerves are grouped around the lower part of the axillary and the brachial arteries. In the upper arm, the medial cutaneous nerve of forearm (MCNF) lies anterior to the brachial vein and is the most superficial of the nerves within the neurovascular bundle. The nerve perforates the deep fascia in the middle part of the arm. The slender medial cutaneous nerve of the arm (MCA) runs outside and posterior to the neurovascular sheath. Communicating branches between the MCNF and MCA are common. The axillary artery is embraced by the two roots of the median nerve, which starts on its lateral side and crosses it in the arm. The musculocutaneous nerve most commonly arises as a single branch from the lateral cord at or below the level of the coracoid process, but it may consist of several branches arising at intervals along the line of the lateral root of the median nerve. It passes laterally into the coracobrachialis muscle and the flexors of the elbow. The ulnar nerve and the medial cutaneous nerve of the forearm are on the medial side of the artery. The former passes posteriorly about half way down the arm to pierce the medial intermuscular septum and to lie between it and the medial part of the triceps muscle, to which it usually gives a branch. Deepest of all is the radial nerve, which crosses anterior to the tendon of latissimus dorsi and then passes posteriorly between the long and medial heads of triceps. The nerve runs behind the humerus deep to the lateral head of the triceps and gains the lateral aspect of the lower part of the arm by piercing the lateral intermuscular septum. The neurovascular bundle can be traced up into the axilla to expose the cords and the origin of the circumflex and radial nerves.

Some key point:

- Take time to identify the medial cutaneous nerve of forearm, an important guide.
- If there is difficulty with the median nerve trace the brachial artery.

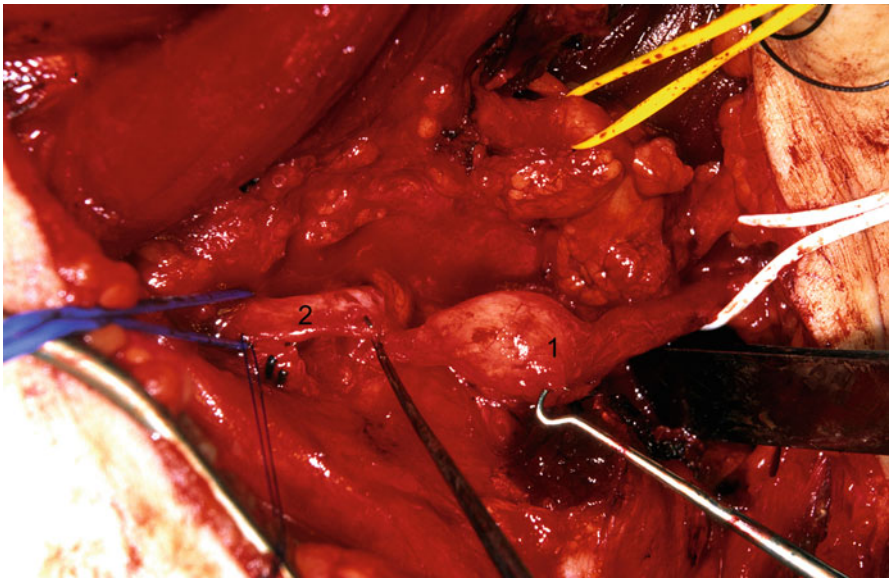


Fig. 5.40 Rupture of the circumflex nerve at the entrance to the quadrilateral tunnel. The proximal stump (*1*) and, somewhat unusually the distal stump (*2*) are visible

5.5.9 The Radial Nerve

The proximal part of the radial nerve is quite easily displayed in the upper part of the axillo-brachial incision; the distal part is easily found through an anterolateral incision in the lower part of the arm and by entering the interval between the biceps and brachialis medially and the brachioradialis and extensor carpi radialis longus laterally (Fig. 5.41). Finding the middle part – the part most likely to be in trouble – is rather more difficult. It is to be found through a posterior incision, and by separation of the superficial part of the triceps (the long and lateral heads) from its deep part (the medial head). The flaps are raised and the lower part of the deltoid muscle and the superficial part of the triceps muscle are exposed**.** The V-shaped interval between the upper parts of the long and lateral heads is now defined, by locating the upper part of the long head and following its lateral border distally. The "seam of the half sleeve" [13] – that is, the junction of the long and lateral heads of the triceps – is now opened from the top towards the olecranon (Fig. 5.42). Further exposure of the radial nerve is anterior to the lateral head of the triceps muscle, by entering the interval between the biceps/brachialis and the brachioradialis/extensor carpi radialis longus. If there is difficulty in bridging a gap after resection, some length can be obtained by re-routing the distal stump towards the upper medial aspect of the arm deep to the biceps and brachialis muscles. For exposure of the more distal parts of the nerve the incision may be extended round the side of the arm to the medial side of the brachioradialis. Thence it runs across the anterolateral aspect of the elbow into the forearm. With

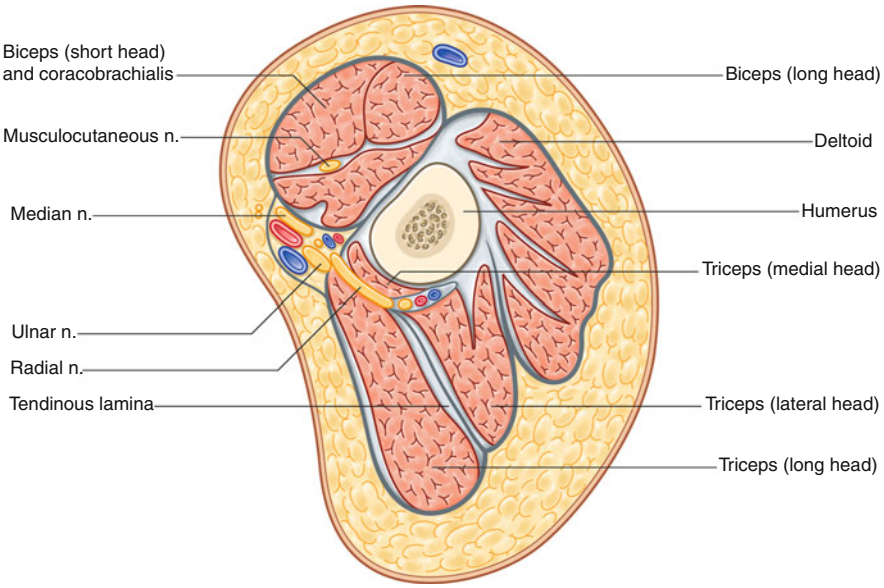


Fig. 5.41 Transverse section through the arm above the level of the insertion of the deltoid muscle, just below the level of the posterior movement of the radial nerve

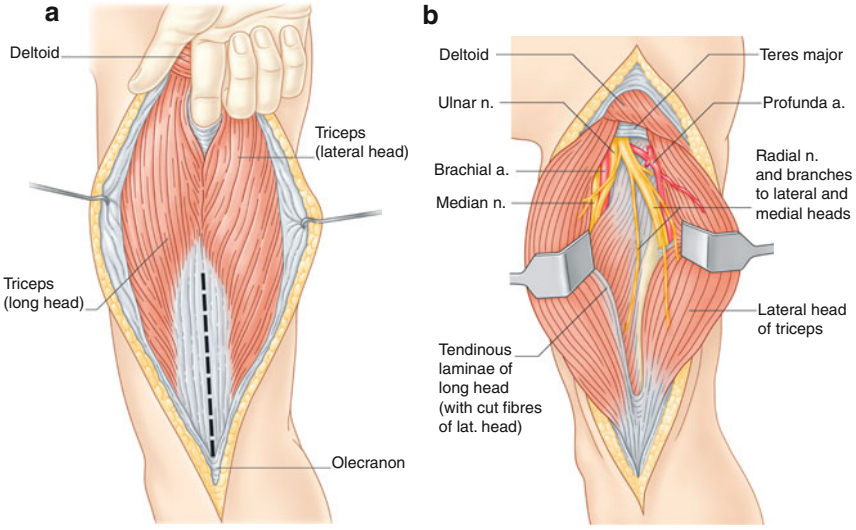


Fig. 5.42 The exposure of the radial nerve in the arm. (**a**) The "seam of the triceps". The finger is introduced between the long and lateral heads which are separated. (**b**) The opening of the "seam" reveals the radial nerve from the start of its course on the back of the humerus to the piercing of the lateral intermuscular septum

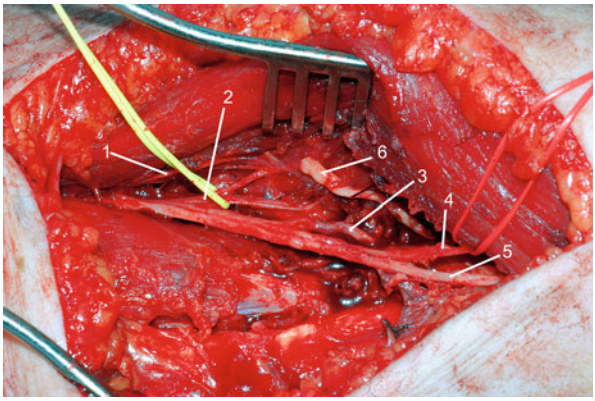


Fig. 5.43 The radial nerve at the elbow exposed during repair of posterior interosseous nerve cut during operation for fracture of radial head. *Above*: the nerve is seen in the valley between brachioradialis and biceps lying on fibres of brachialis. *Left*: the nerves to brachioradialis (*1*) to extensor carpi radialis longus and brevis (*2*) the proximal part of the posterior interosseous nerve (*3*) a further branch to extensor carpi radialis brevis (*4*) and the superficial radial nerve (*5*) are shown. A segment of the lateral cutaneous nerve of forearm (*6*) has been prepared as a graft

this anterior approach the radial nerve and its terminal branches are found in the interval between the biceps/brachialis and the brachioradialis/extensor carpi radialis longus. There are important branches from this segment of the nerve. The nerve to brachialis, passing antero-medially is often seen about five finger breadths above the lateral epicondyle. The nerve to brachioradialis passes postero-laterally about 2 cm more distal. More distally still, is the branch to extensor carpi radialis longus and then, after that, branches, usually two, to extensor carpi radialis brevis (Fig. 5.43).

Some key points:

- Note the posterior cutaneous nerves of arm and forearm emerge from the interval between lateral head of triceps and brachioradialis.
- The plane between brachioradialis and brachialis is sometimes obscured: do not hesitate to extend distally to the cubital fossa and work from below.

5.5.10 The Posterior Interosseous Nerve

In the Henry [13] approach, the incision is made on the posterolateral aspect of the upper forearm between the mobile mass of the brachioradialis and the radial extensors of the wrist, and the extensor communis digitorum. The interval between the extensor carpi radialis brevis and the extensor communis digitorum is then opened and the supinator muscle exposed. The posterior interosseous nerve passes between the superficial and deep parts of this muscle, to emerge at the lower margin of the superficial part and to run for about 4–5 cm before breaking up into its terminal (motor) branches. The point of emergence can quite easily be found, and the

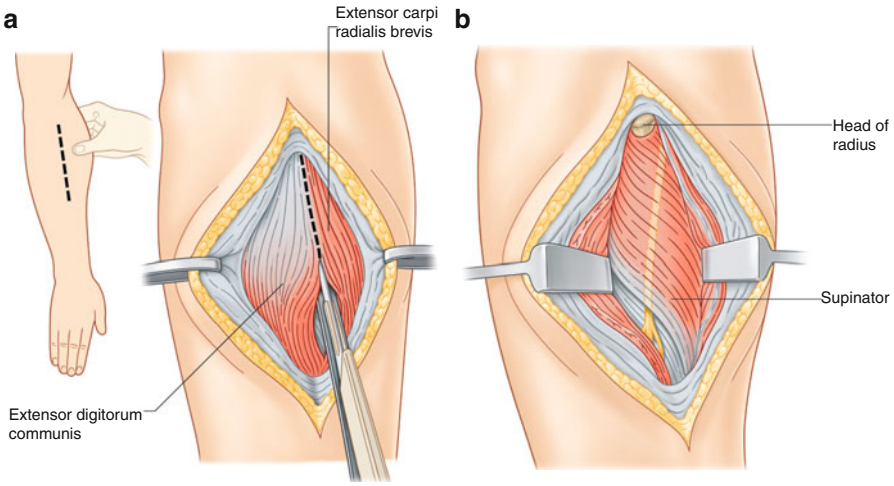


Fig. 5.44 The exposure of the posterior interosseous nerve. (**a**) The incision (*left*) and the separation of the extensor carpi radialis brevis from the extensor communis (*right*). (**b**) The posterior interosseous nerve and supinator exposed

nerve then followed proximally by division of the superficial part of the muscle along its line (Fig. 5.44).

5.5.11 The Lower Part of the Median Nerve

The median nerve is easily displayed at and just below the elbow and in the lower part of the forearm through a sinuous incision winding down from the elbow. At elbow level it is found medial to the brachial artery (Fig. 5.45). It then descends between the superficial and deep heads of the pronator teres The median nerve can be traced down into this tunnel, and can to some extent be mobilised by division of the deep (ulnar) head of the pronator teres. Having negotiated the pronator teres, the nerve runs down the forearm between the deep and superficial flexor muscles, loosely attached by areolar tissue to the deep surface of the flexor digitorum superficialis. It can be exposed in this part of its course by separating the flexor superficialis from its radial origin and retracting the muscle. The anterior interosseous nerve is now displayed. The nerve enters the hand just deep to and between the tendons of the palmaris longus and flexor carpi radialis.

The carpal tunnel is formed posteriorly and laterally by the concavity of the carpus; in particular, the scaphoid, lunate, hamate, and pisiform bones. It is roofed by the flexor retinaculum and anchored medially to the pisiform and hamate bones and laterally, to the scaphoid and the trapezium. The width of the retinaculum is about 2.5 cm; its length is about the same. Proximally it blends with the ante-brachial fascia, and distally with the palmar aponeurosis. The tendon of the palmaris longus is attached to it anteriorly. A deep lamina on the radial side is attached to the medial

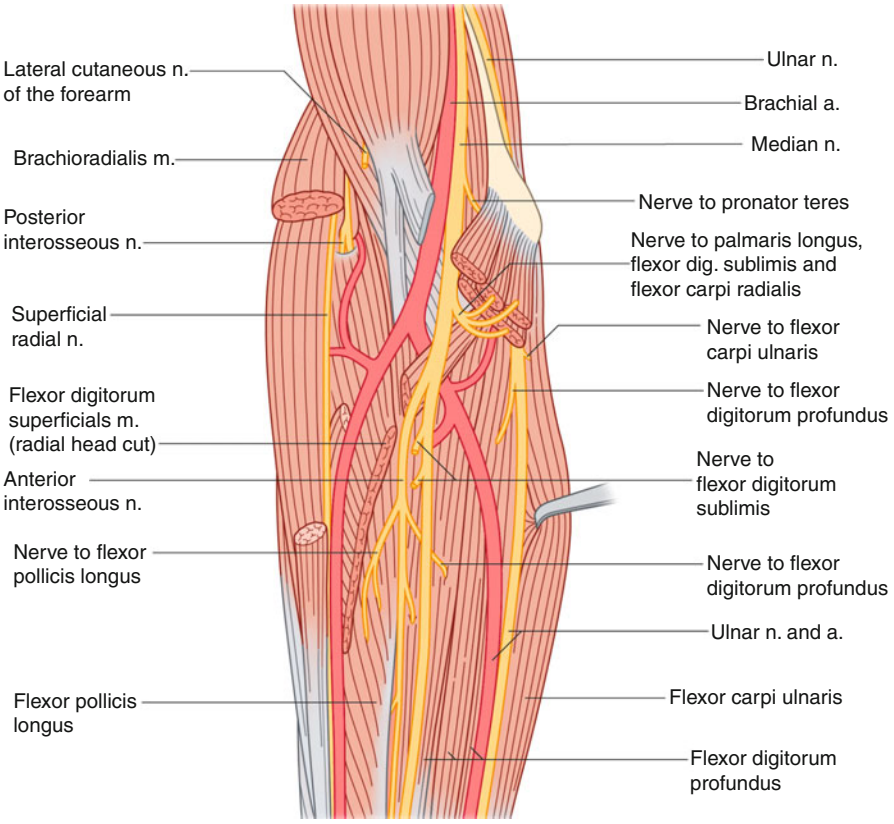


Fig. 5.45 The anterior aspect of the right elbow (see Fig. 1.15)

lip of a groove on the trapezium. Between this layer and the more superficial part lies the tendon of the flexor carpi radialis muscle in its synovial sheath. On the ulnar side a localised thickening of the ante-brachial fascia extends laterally from the pisiform bone as a superficial part of the retinaculum, passing superficial to the ulnar nerve and vessels to blend with the retinaculum lateral to them.

The positions of the motor and the palmar cutaneous branches of the median nerve are very important for the surgeon. In most cases the motor branch arises from the radial side of the nerve in the carpal tunnel and runs laterally, soon branching to supply the opponens and short abductors of the thumb, and often part of the flexor brevis. A particular hazard for the surgeon is produced by an origin from the ulnar side of the nerve and a course across it, either deep or superficial, to the retinaculum. The palmar cutaneous branch arises from the median nerve about 7 cm above the wrist crease and runs distally, medial to the tendon of the flexor carpi radialis to innervate the skin over the thenar eminence and the radial side of the proximal part of the palm. Damage to this can cause a profound, persistent painful state, with very troublesome hyperaesthesia and hyperalgesia, and even hyperpathia and allodynia (Fig. 5.46).

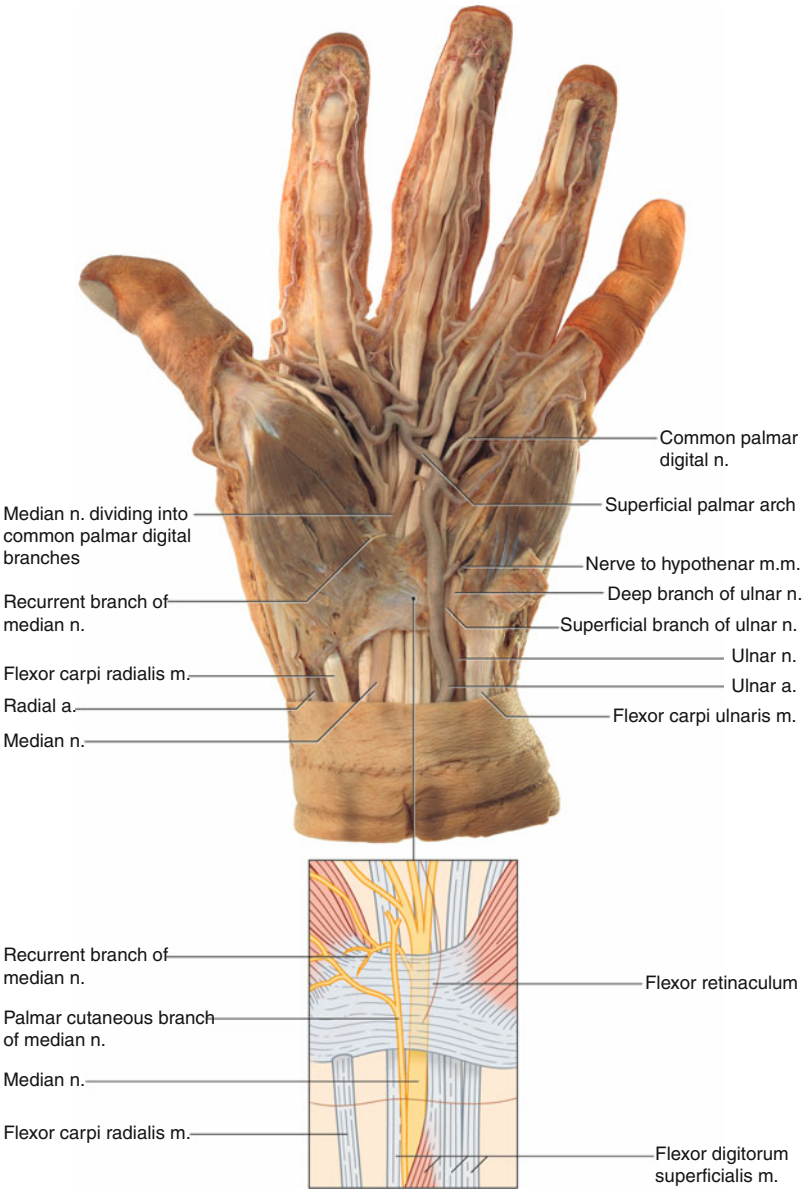


Fig. 5.46 The median and ulnar nerves in the left hand. *Inset* shows the normal course of median nerve at the wrist and also the palmar cutaneous nerve (see Fig. 1.16)

Two key points:

- Beware the palmar cutaneous nerve which arises from the radial side of the median nerve.
- Variations in the course of the motor branch are frequent.

5.5.12 The Lower Part of the Ulnar Nerve

As the ulnar nerve approaches the elbow, it runs behind the medial intermuscular septum and then passes subcutaneously behind the medial epicondyle of the humerus in the retro-condylar groove. It passes through the arcade of Struthers, a fibrous canal about 6 cm in length which terminates at about 4 cm proximal to the medial epicondyle [3]. The components of the arcade include not only the tough medial intermuscular septum of the arm but also the sheath of the medial head of triceps, which can be seen enveloping the nerve, as well as the internal brachial ligament. The ulnar nerve then passes superficial to the medial capsule of the elbow joint and deep to the arcade, joining the two origins of the flexor carpi ulnaris muscle (the arcuate ligament). This is the "cubital tunnel". A branch of the medial cutaneous nerve of forearm passes superficially here. The nerve then runs between the flexor carpi ulnaris and flexor digitorum profundus muscles, joining the ulnar artery in its course down the forearm. The muscular branches arise just below the elbow. The nerve is vulnerable to external pressure. It may be compressed between the arcuate ligament and the medial capsule and with relaxation of this ligament it may become hypermobile, dislocating forward when the elbow is flexed. Because of the excursion required of it during movement of the elbow, it may be stretched during flexion if its movement is restricted by changes due to damage by external pressure.

The ulnar nerve and artery pass down the forearm within a well defined sheath which is found deep to the anterior margin of flexor carpi ulnaris. The nerve and artery may become tightly compressed by swollen anoxic muscle and, later, by post ischaemic fibrosis. Arterial bleeding from a wound on the ulnar aspect of the forearm indicates damage to the ulnar nerve.

The ulnar nerve divides into its superficial (sensory) and deep (motor) components at about wrist level. Both components run into Guyon's space (Fig. 5.47). The superficial branch passes superficially to supply the palmaris brevis and the skin of the medial two digits; the deep branch runs between the abductor and the flexor of the little finger to pierce the opponens and then runs across the deep palmar space with the deep palmar arch, ending by supplying the adductor and flexor brevis of the thumb and the first palmar interosseous muscle. Deep as it is, the deep branch is vulnerable to wounds from glass or knife. The nerve is best followed by an incision beginning above the wrist, entering the ulnar side of the palm and curving across the distal palm in a crease. It is isolated in the upper part of the incision lateral to the tendon of flexor carpi ulnaris and medial to the ulnar artery and followed to its bifurcation. Then the deep branch is followed between the hypothenar muscles. Its deep course has to be revealed by mobilisation of the deep and superficial flexor tendons. The multitude of the motor branches of this nerve, to most of the intrinsic muscles of the hand, has to be borne in mind during this exposure, though it is impossible to avoid some of these in a scarred field.

Two key points:

- Beware the medial cutaneous nerve of forearm around the elbow. It is always in the way and damage causes intractable pain.
- Look out for the dorsal cutaneous branch of the ulnar nerve. It is best to formally expose this when dealing with fractures of the shaft of the ulna.

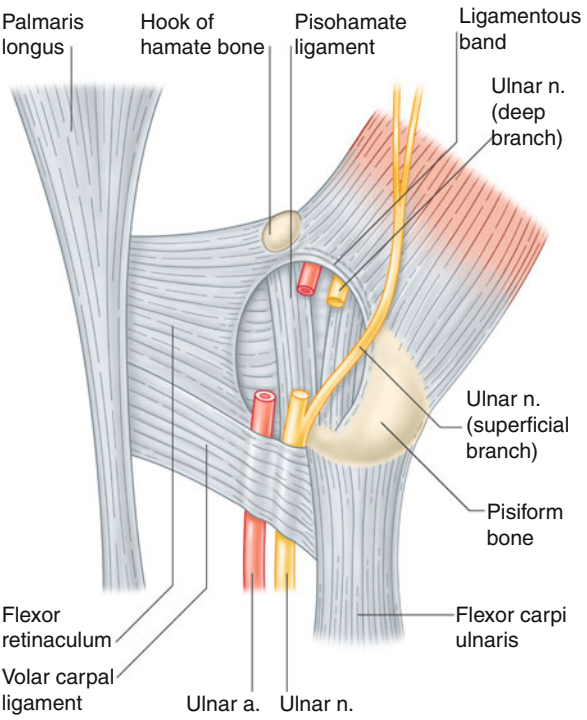


Fig. 5.47 Guyon's space. Note the possibilities for entrapment at, and distal to, the volar carpal ligament

5.6 Approaches to Individual Nerves: Abdomen, Pelvis and Lower Limb

5.6.1 The Lumbar Plexus

This is accessible through the lower quadrant of the abdomen, by the same transverse muscle cutting incision and extra-peritoneal approach that is used for lumbar sympathectomy [16]. The exposure may be helped by placing the patient in the lateral or semi-lateral position. The incision is made between the rib cage and the iliac crest. The external oblique split and the deeper muscle is cut, with opening of the lateral part of the rectus sheath. The underlying fascia is delicately opened and the peritoneum is swept away from the abdominal wall and vertebral column. The lateral cutaneous nerve of thigh, the ilio-inguinal nerve and the ilio-hypogastric nerve are encountered approaching the lateral and posterior margin of the psoas muscle. The femoral nerve lies in a gutter between the iliacus and the psoas major (Figs. 5.48 and 5.49). The obturator nerve runs medial to the psoas muscle. The lumbo-sacral trunk is closer to the mid line and deep to the great vessels. The spinal nerves passing to the femoral nerve run deep to the psoas muscle in much the same way as the cervical spinal nerves run deep to scalenus anterior and the muscle must be cut or reflected to display them. This approach gives good access to the lumbar plexus in the psoas muscle and to the femoral and obturator nerves on each side of the lower part of the psoas. Access to the lumbosacral plexus is rather restricted

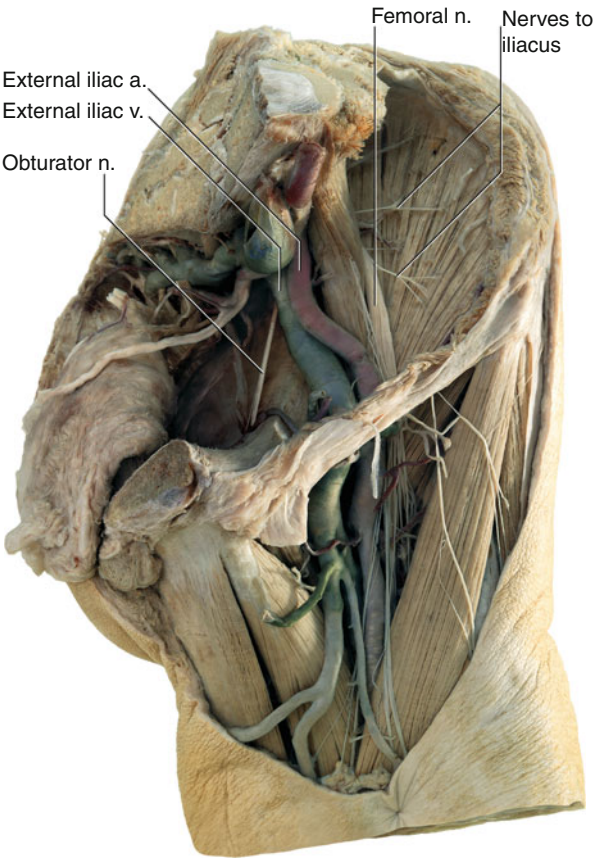


Fig. 5.48 The left femoral nerve (see Fig. 1.20)

through this extra-peritoneal approach. A trans-peritoneal approach makes access easier, but the viscera have to be mobilised with the consequent risks of ileus after operation and of late complications from adhesions. Injuries to the lumbosacral plexus are often associated with forbidding scarring.

5.6.2 The Femoral Nerve

This can be traced down to the level of the inguinal ligament from above through an abdominal incision and exposed again in the thigh through a separate anterior crural incision. Kline, Hudson and Kim [16] suggest that the crural incision can be extended laterally above the inguinal ligament and the lower abdominal muscles split to give an extraperitoneal approach.

Key point:

- Exposure of the nerve at the groin crease is enabled by identifying the common femoral artery and working lateral to it. The nerve lies in a slightly deeper plane.

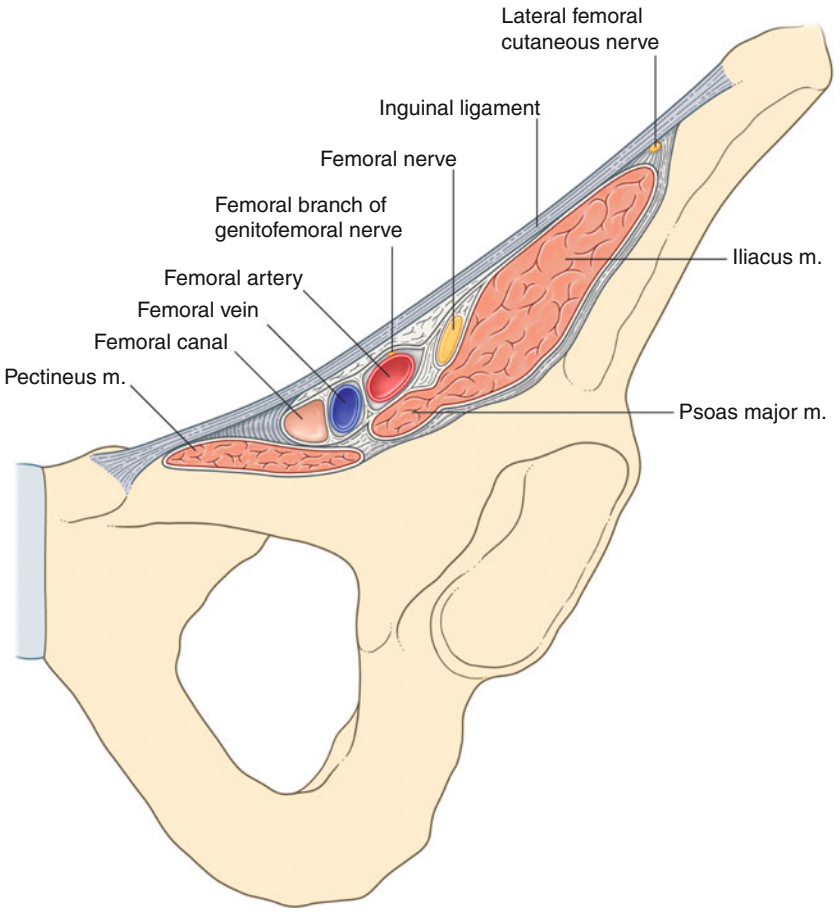


Fig. 5.49 The femoral nerve and vessels deep to the inguinal ligament. The drawing shows the left inguinal region (see Fig. 1.38)

5.6.3 The Sciatic Nerve

Two exposures are regularly used. In the first, the gluteus maximus muscle is split as in the posterior approach to the hip. This provides an adequate exposure of the sciatic nerve where it traverses the neck of the femur. Up to 15 cm of the trunk can be seen. A second, more extensive, approach is described by Henry [13]. The prone position is used for both.

Splitting the gluteus maximus: The incision runs obliquely about two finger breadths above the line drawn from the ischeal tuberosity to the tip of the greater tuberosity. The gluteus maximus is exposed and gently split along the line of its fibres noting and preserving branches of the inferior gluteal nerve and securing careful haemostasis. The muscle flaps are held apart with the large Deavers retractor.

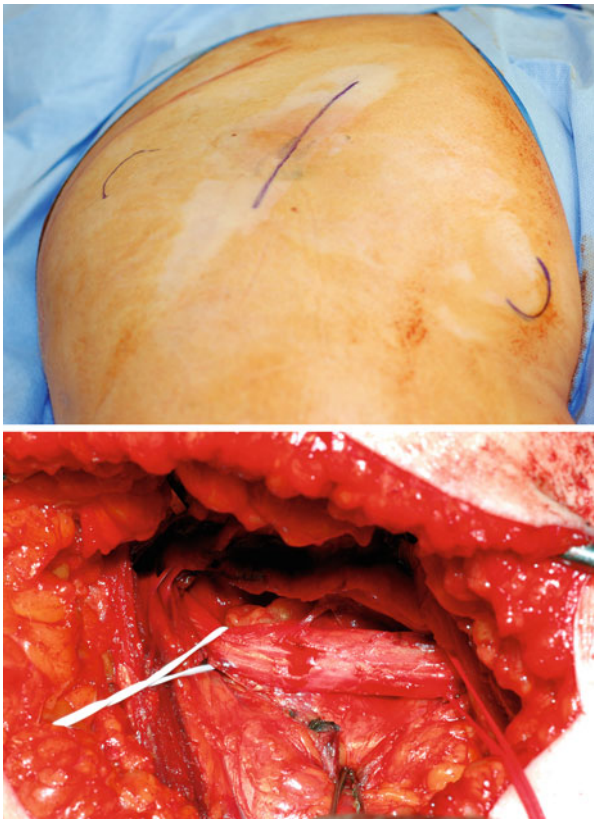


Fig. 5.50 Exposure of sciatic nerve by transgluteal approach. *Above*: the patient lies prone and the incision is made between the tip of the greater trochanter and the ischial tuberosity. *Below*: the nerve is exposed as it crosses the neck of the femur

The nerve is seen crossing the posterior aspect of the neck of the femur, and it can be traced up to the sciatic notch if necessary by division of the piriformis (Fig. 5.50).

The wound is closed by repair of the sheath of gluteus maximus, the fat and the skin. This approach is very useful in exploration of lesions of the sciatic nerve incurred during arthroplasty of the hip.

Key points:

- Haemostasis must be meticulous.
- The trunk is easily demonstrated by rolling the tip of the finger over it.

Full exposure of the sciatic nerve and of the superior and inferior gluteal nerves and vessels is achieved through the operation described by Henry [13], who compares the "gluteal lid" with a parallelogram whose shorter sides are almost longitudinal and whose longer (upper and lower) sides are oblique. The caudal edge is virtually unattached.

The incision can follow the "question-mark" shape advised by Henry, or can run from the iliac crest at the junction of the gluteus maximus and the iliotibial tract,

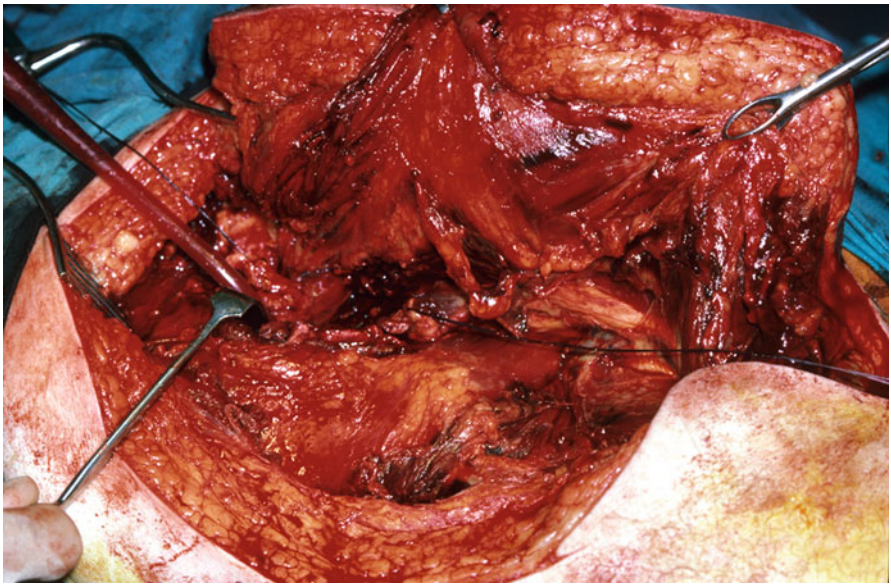


Fig. 5.51 The extended exposure of the sciatic nerve damaged by fracture/dislocation of the hip

obliquely down the back and lateral side of the buttock to reach the great trochanter and then turn medially to descend in the mid line of the thigh. It is necessary to look out for the posterior cutaneous nerve of the thigh, piercing the deep fascia in the upper part of the thigh and descending in the midline. It should be identified under the fascia below the lower border of gluteus maximus. Now, the gluteus maximus is detached from its insertion to the femur; the cephalic side of the muscle is separated from the ilio-tibial tract, and both tendinous and muscular insertions to the femur are divided**.** Then, with continued care for the posterior cutaneous nerve of the thigh, the gluteal lid is hinged back on its pelvic origin. Excessive traction upon the superior gluteal vessels must be avoided. The structures displayed are the gluteus medius and the short lateral rotators of the hip, the superior and inferior gluteal vessels and nerves, the pudendal nerve and vessels and the sciatic nerve. The sciatic nerve can be followed into and through the great sciatic notch by division of the tendon of the piriformis and retraction of the muscle. Downward extension of the vertical limb of this incision permits display of the whole of the sciatic nerve (Fig. 5.51).

Careful closure of the wound is essential. In particular, the gluteus maximus must be reattached laterally and cephalad. One or two suction drains should be used. The subcutaneous tissues should be closed.

Key points:

- Haemostasis must be meticulous.
- Avoid traction upon the formidable superior gluteal vessels.
- Reattachment of the muscle must be secure.

5.6.4 The Tibial and Common Peroneal Nerves in the Popliteal Fossa and Below

The incision starts above the back of the knee and skirts the crease to return to the midline below the knee, to descend in the midline for about 10cms. The flaps are raised. Care must be taken of the sural nerve arising from the tibial nerve and descending in the midline, at first just under the deep fascia and piercing that in the proximal part of the leg The tibial nerve and popliteal artery and vein are found above in the mid-line; the common peroneal nerve has at this level deviated laterally to lie close to the tendon of the biceps femoris. The gastrocnemius is split in the midline to expose the underlying soleus muscle, which also is split to show the nerve and vessels below the knee.

The tibial nerve in the leg and behind the ankle is exposed through a straight or sinuous incision over the medial head of the gastrocnemius and medial to the tendo calcaneus. The medial head of the gastrocnemius is exposed, freed and retracted laterally to expose the medial part of the soleus. Then, the medial part of the soleus is mobilised by division of the medial "pier" of its tendinous arch and of its medial origin. The soleus is then retracted laterally, to expose the deep compartment of the leg with the tibial nerve and vessels (Fig. 5.52).

Exposure of the common peroneal nerve necessarily takes the popliteal incision laterally, to descend on the lateral side of the leg and permit exposure of the deep peroneal (anterior tibial) and superficial peroneal divisions in which the nerve terminates just below the neck of the fibula. The operator must remember that the common peroneal nerve is very close to the surface behind the head of the fibula and lateral to its neck. It has been partly divided by the initial skin incision. The nerve is embraced by fascia enveloping the biceps femoris and is displaced with that muscle in dislocation of the knee.

Some key points:

- The sural and common peroneal nerves are the signposts in this exposure.
- The common peroneal nerve is close to the skin and will be displaced anteriorly by the biceps femoris after knee dislocation.
- As with all nerve work it is much easier to find these nerves in proximal healthy tissue.

5.6.5 The Lower Tibial Nerve and the Plantar Nerves

The lower part of the tibial nerve is easily found on the medial side of the ankle, under the flexor retinaculum, between the flexor tendons of the hallucis and the flexor digitorum. The terminal plantar branches are traced into the foot by division of the retinaculum and then by bringing back the abductor hallucis muscle after defining its superior edge and detaching it from its origin on the distal part of the

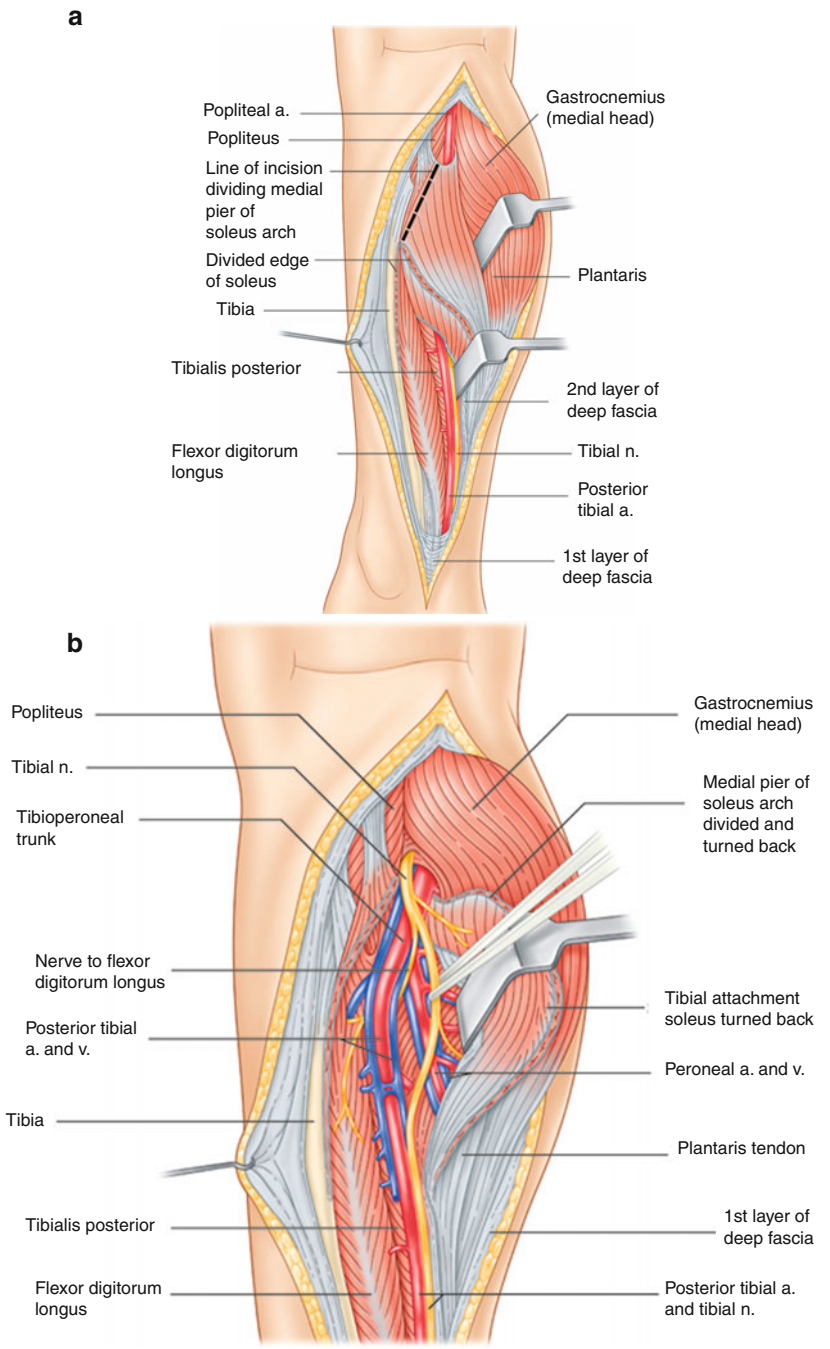


Fig. 5.52 Exposure of the tibial nerve in the leg. (**a**) The soleus exposed and the line for its detachment shown. (**b**) The tibial nerve and vessels revealed by the retraction of the soleus

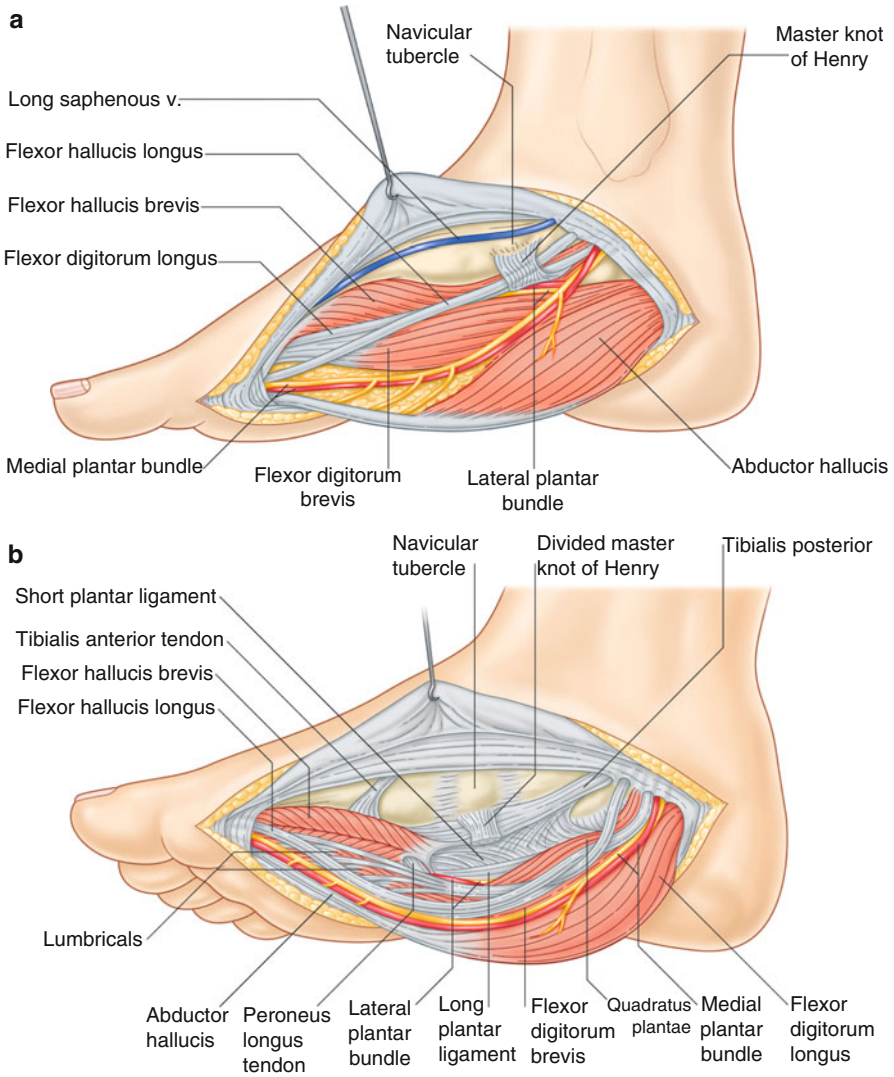


Fig. 5.53 Exposure of the plantar nerves through a medial incision. (**a**) Retraction of abductor hallucis reveals the medial plantar nerve. (**b**) Release of the tendon of the flexor longus hallucis gives access to the whole of the deep compartment of the sole

retinaculum. The plantar nerves are found between the deep and superficial layers of the plantar muscles in the plane between, on the one hand, the two abductors and the flexor brevis digitorum and on the other, the flexor accessorius and the tendons of the long flexors (Fig. 5.53).

Key point:

- Take care for the calcaneal nerves. Wounding these can prove most troublesome.

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