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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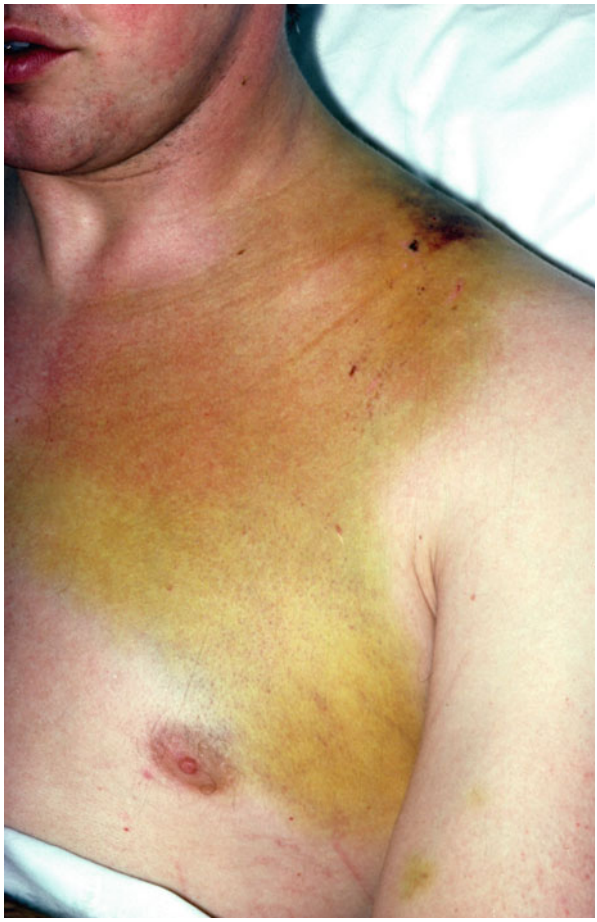

Fig. 4.1 This motor cyclist struck his shoulder against a traffic bollard. There is bruising and swelling of the left shoulder, neck and upper arm. Total avulsion

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).

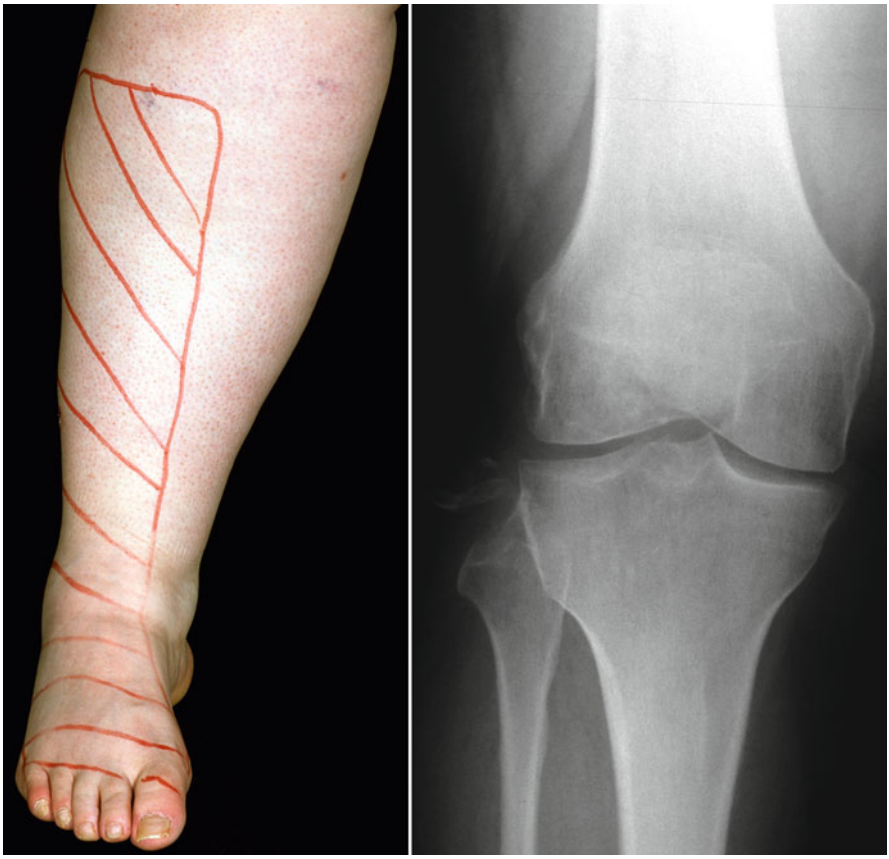

Fig. 4.2 The Platt lesion. A 64 year old woman avulsed the fibular styloid standing up from a chair. The common peroneal nerve was ruptured

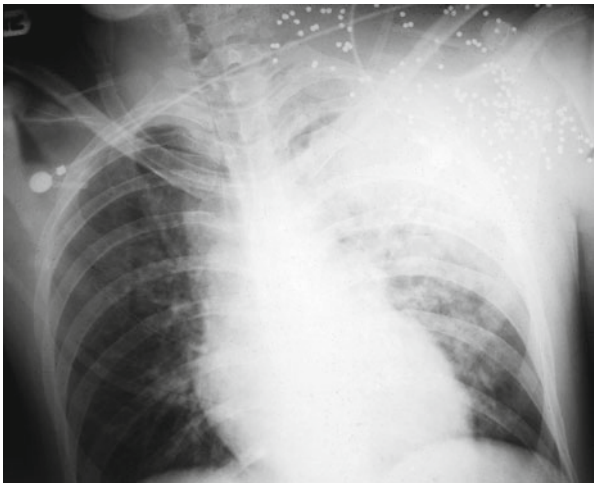

Fig. 4.3 Close range shot gun blast to the posterior triangle of the neck. There was rupture of the first part of the subclavian artery

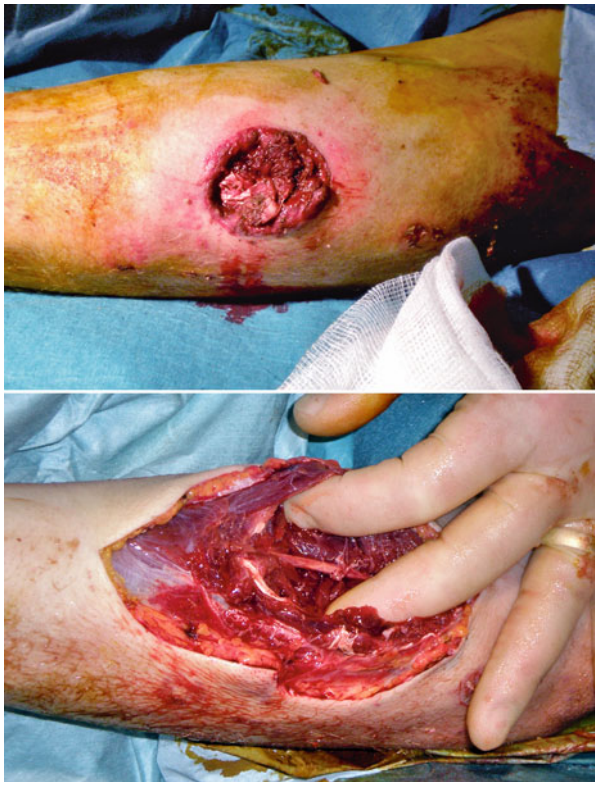

Fig. 4.4 Military rifle bullet, before debridement (*above*) and after debridement (*below*). Type 3 F wound: E3, X8, C2, F2. The common peroneal nerve recovered (axonotmesis)

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

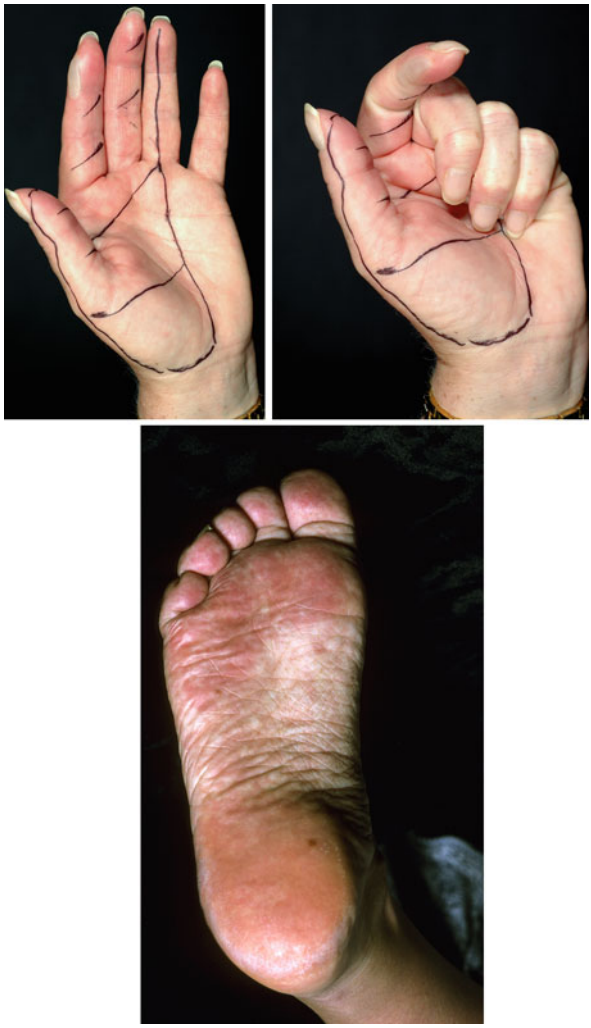

Fig. 4.5 Sympathetic paralysis seen within a few days of transection of (*above*) the median nerve at the elbow and (*below*) the tibial nerve in the thigh

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.

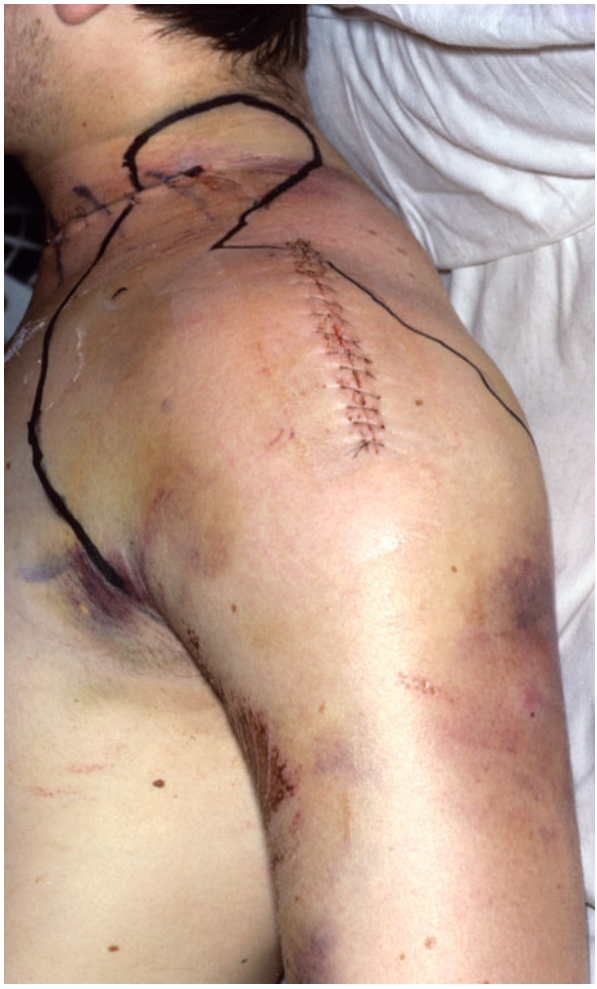

Fig. 4.6 Sensory loss in a case of preganglionic injury C5, C6, C7 and C8 with involvement of C4. The ipsilateral hemi diaphragm was paralysed

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

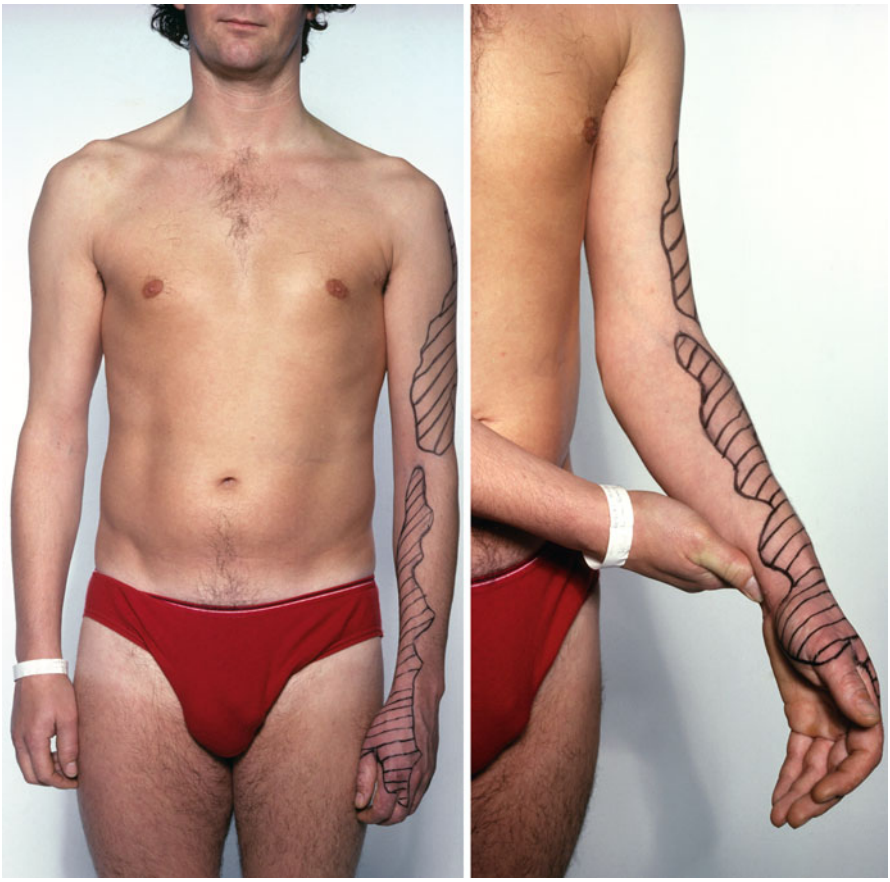

Fig. 4.8 Rupture of C5 and C6. Sensory loss does not extend to the thumb and the index finger

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.

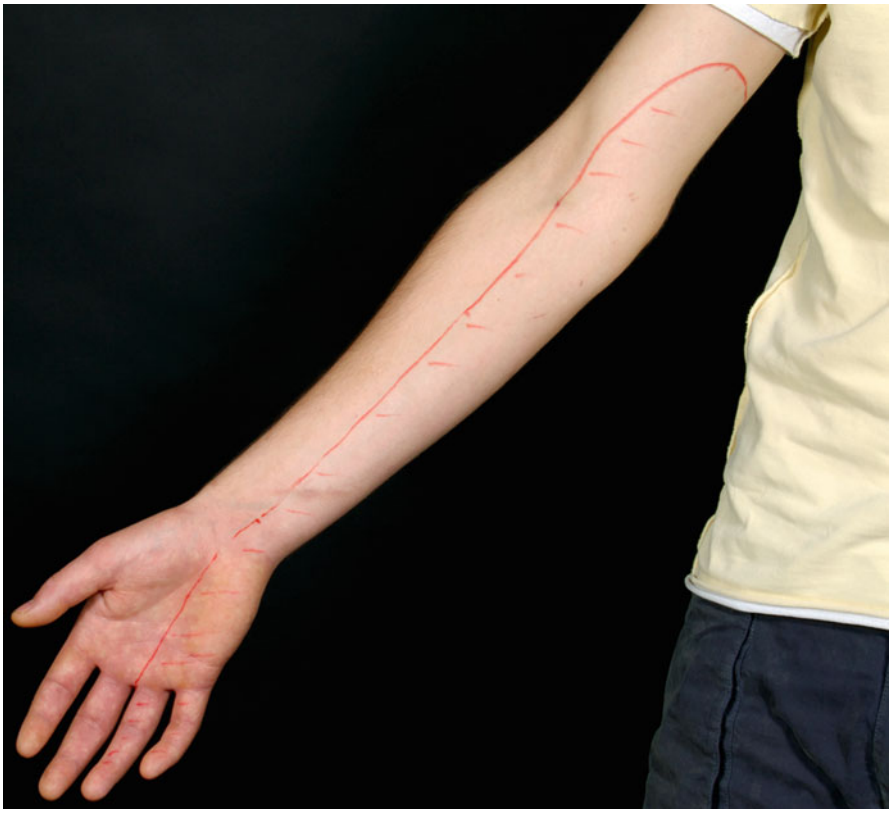

Fig. 4.9 Transection of C8 and T1. The area of sensory disturbance extends into the arm (medial cutaneous nerve of arm)

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

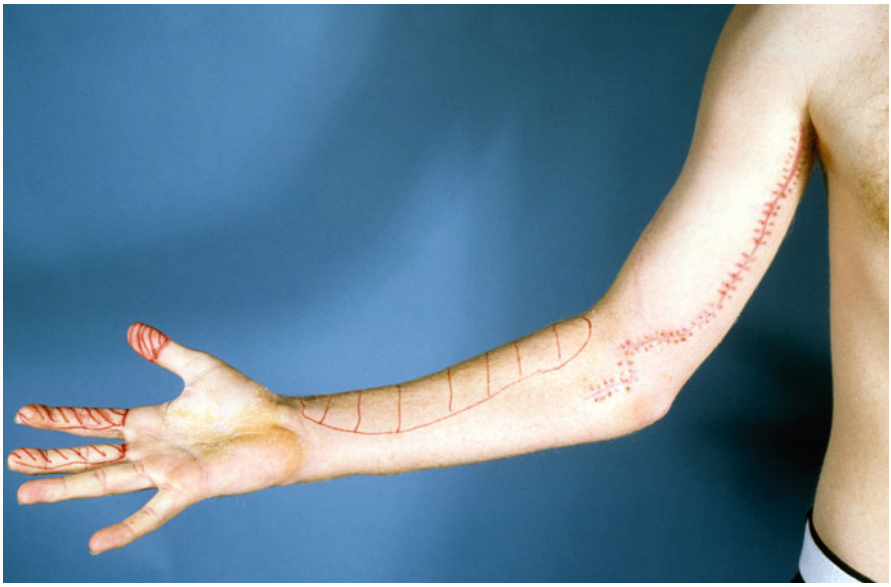

Fig. 4.10 The area of sensory abnormality after section of the lateral cord in the axilla. As in Fig. 4.9, there was no complete loss of sensation.

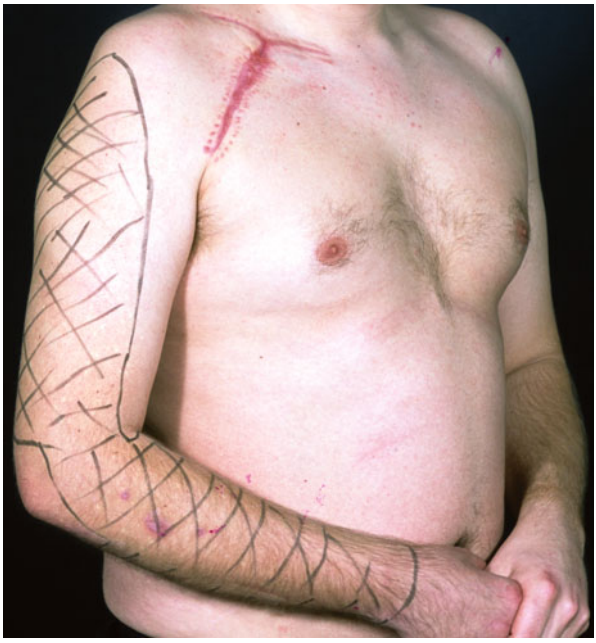

Fig. 4.11 Rupture of the posterior divisions of the trunks of the brachial plexus deep to the clavicle

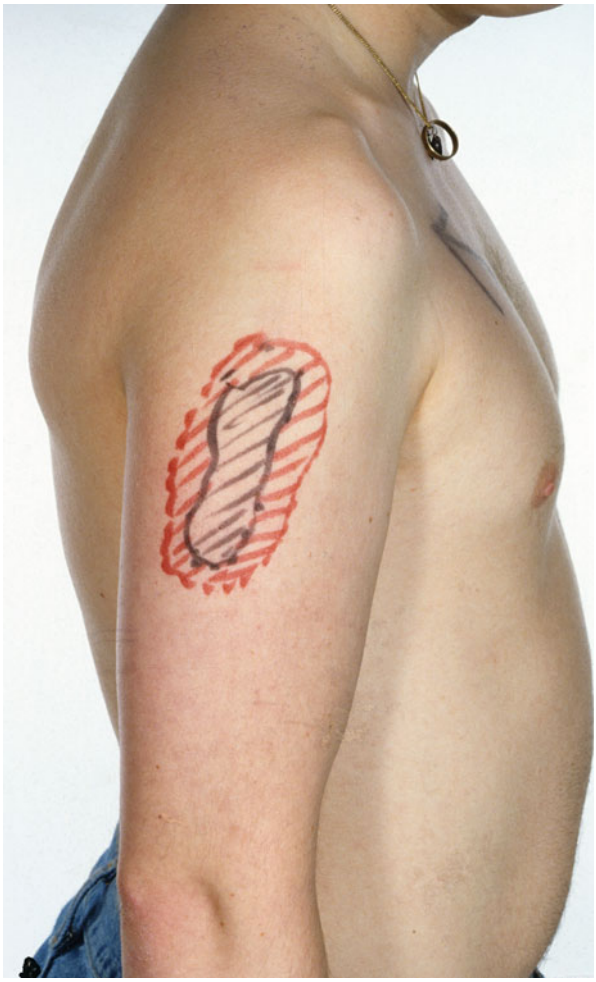

Fig. 4.12 Rupture of the circumflex nerve: the sensory loss is usually smaller than in lesions of C5

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 %

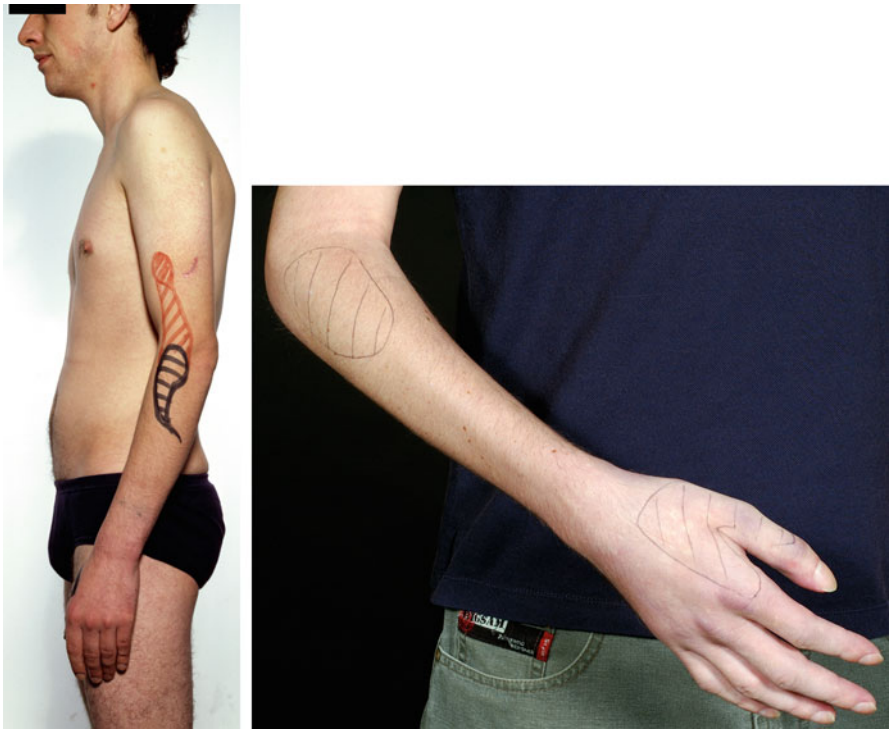

Fig. 4.14 High lesion of the radial nerve. *Right*: there is early recovery into the wrist extensors after repair

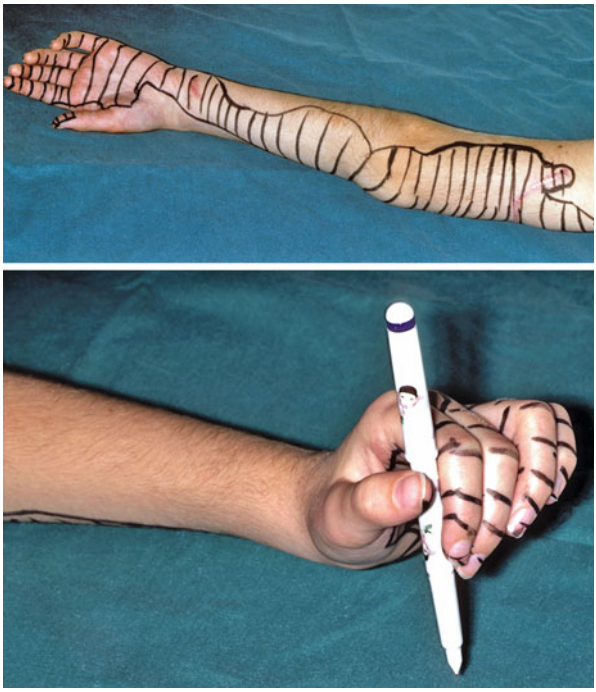

Fig. 4.15 Transection of median, ulnar, medial cutaneous nerve of forearm, and brachial artery in the arm of a 14 year old boy. Note the extent of skin innervation provided by the superficial radial and lateral cutaneous nerves of forearm. The intact radial nerve permits a sort of grasp

- 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

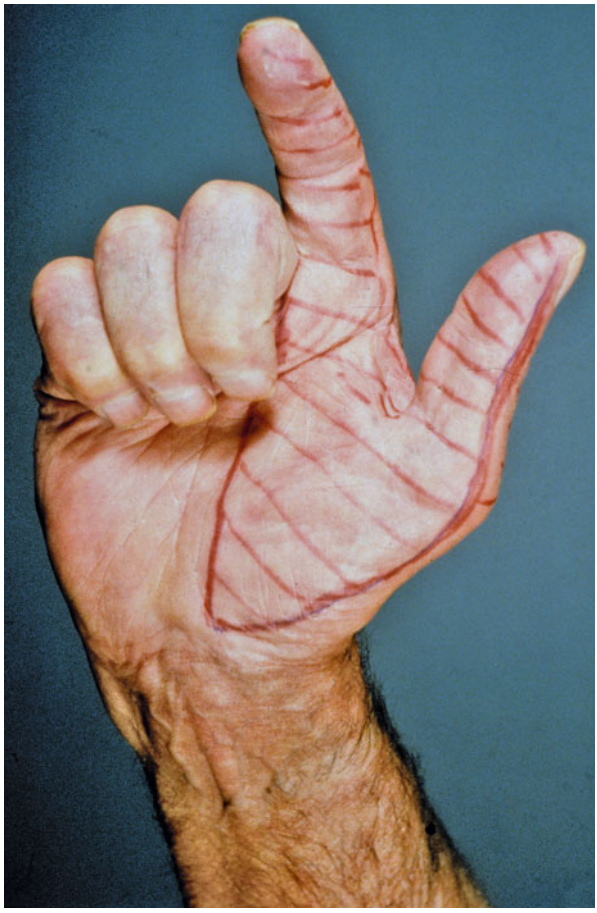

Fig. 4.16 High median nerve injury: no active flexion of the index finger and thumb

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

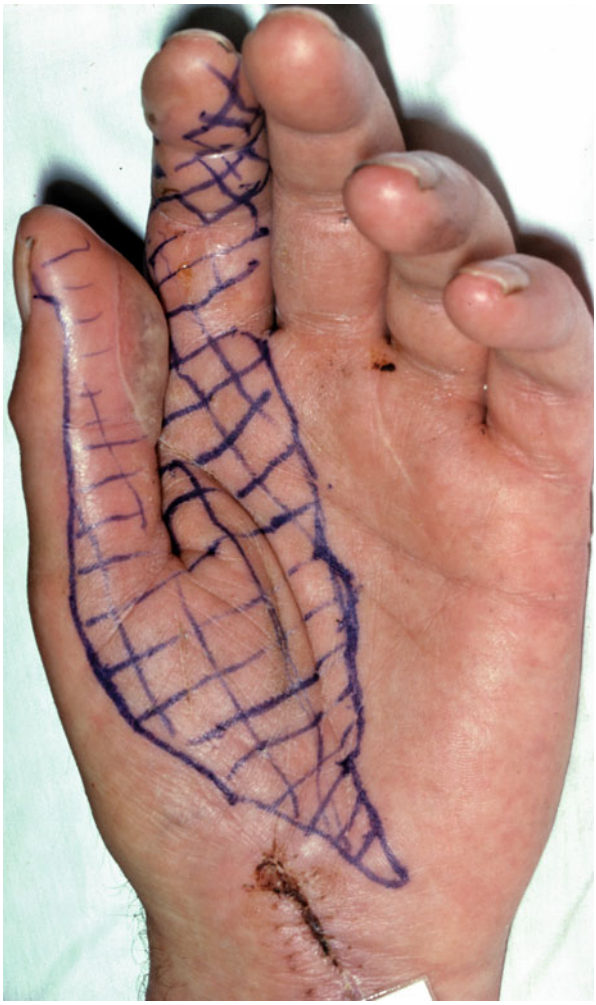

Fig. 4.17 Section of the median and the palmar cutaneous nerve at wrist

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.

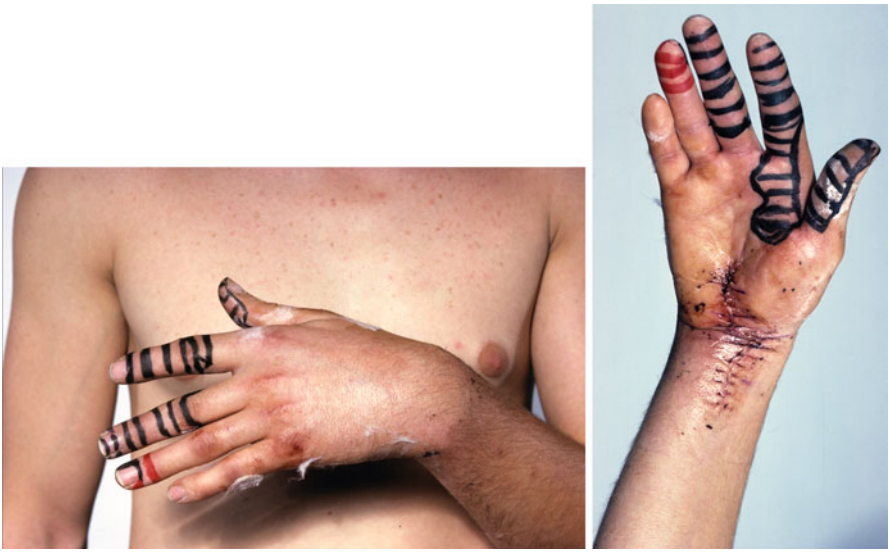

Fig. 4.18 A typical area of loss of sensibility after division of the nerve at the wrist, sparing the palmar cutaneous branch

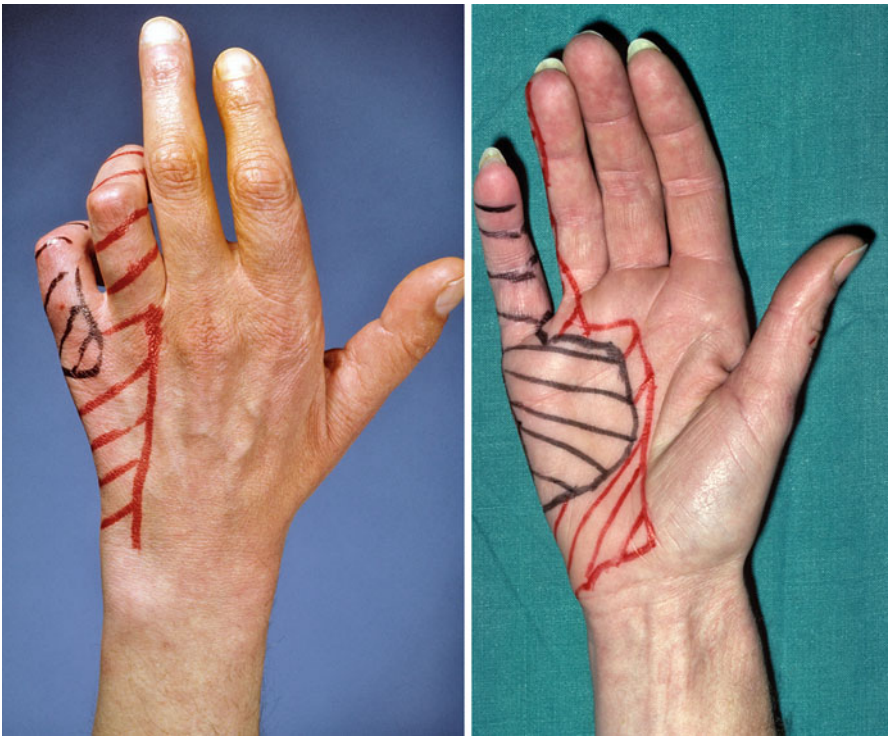

Fig. 4.19 The area of sensory loss and "clawing" of little and ring fingers after division of ulnar nerve in the forearm

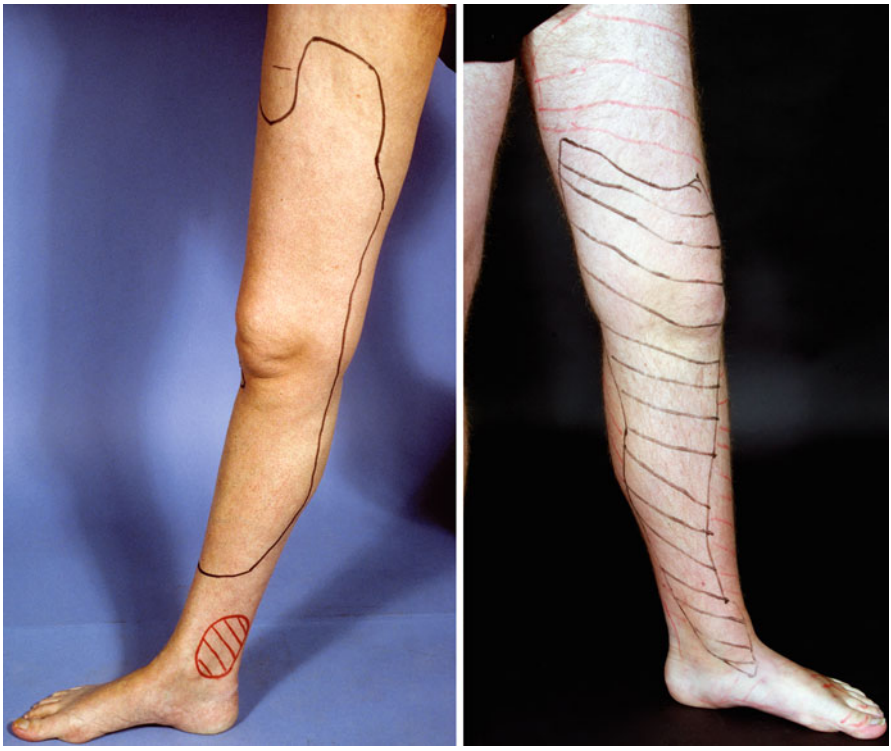

Fig. 4.20 The area of loss of sensibility in two cases of injury to the femoral nerve at, or proximal to the groin crease

- 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

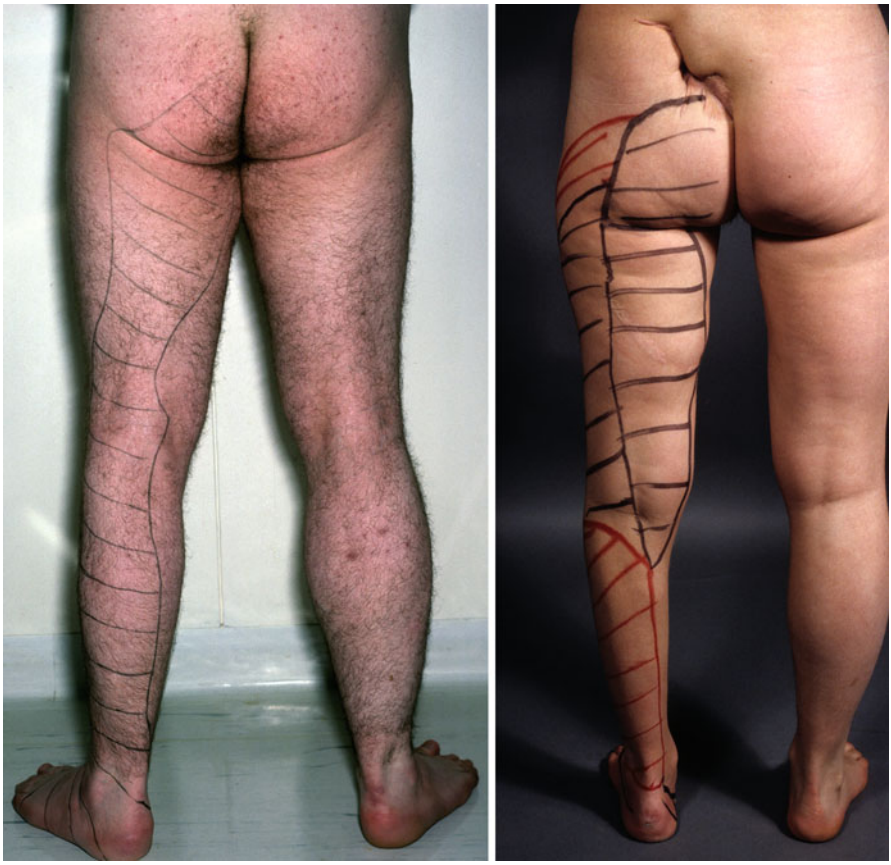

Fig. 4.21 Cutaneous distribution of the sacral plexus. *Left* showing area of sensory loss after closed fracture/dislocation of sacro-iliac joint. *Right* showing area of loss of sensibility after open fracture/dislocation of the pelvis. The muscles of the buttock are wasted

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

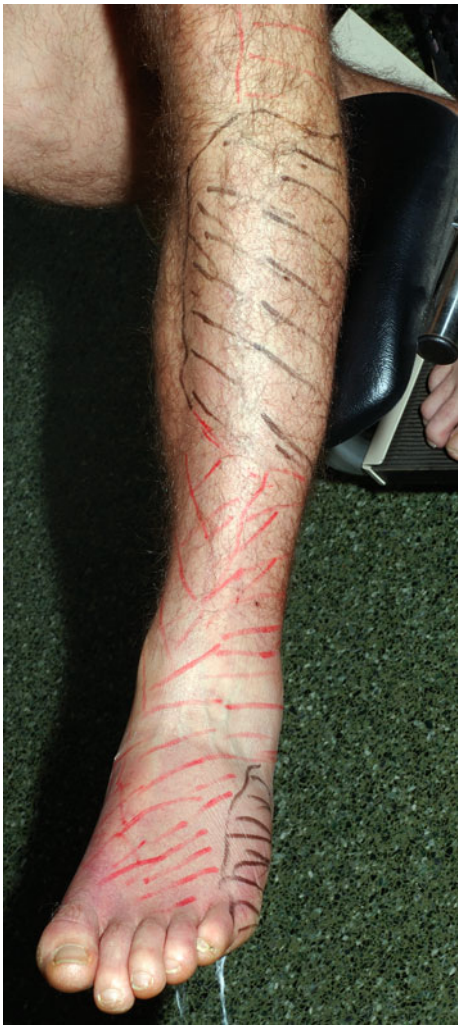

Fig. 4.22 The area of sensory loss after transection of sciatic nerve in the thigh is confined to the leg and foot

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

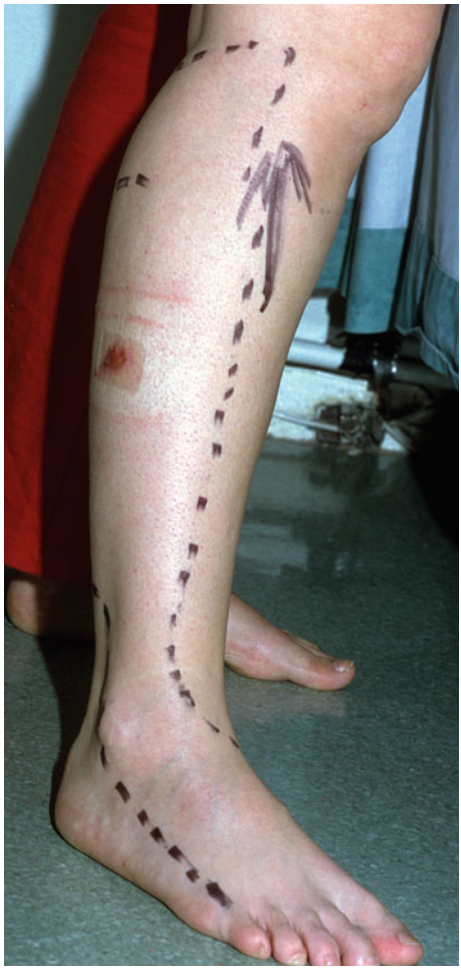

Fig. 4.23 Area of sensory loss after high division of common peroneal nerve during operation of knee ligament reconstruction. Note the pressure sore caused by a conventional ankle-foot orthosis

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).

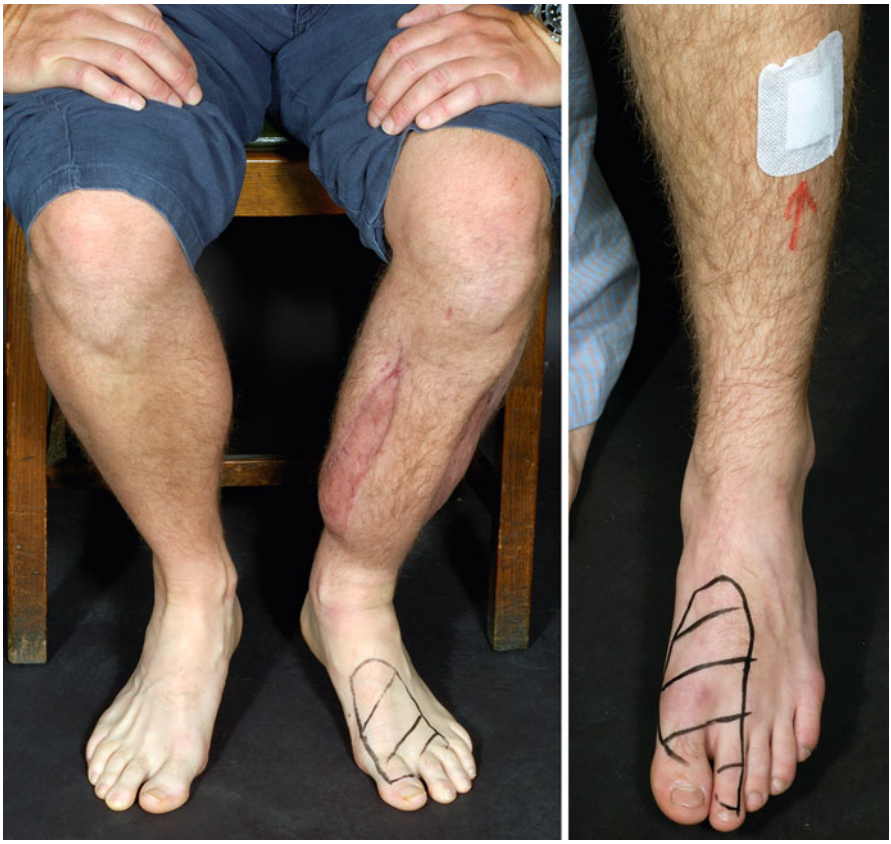

Fig. 4.24 Area of sensory loss after interruption of the deep division of the common peroneal nerve. *Left*, showing the leg of a 29 year old man in whom severe "compartment syndrome" was overlooked after intramedullary nailing of closed fracture of tibial shaft. The anterior compartment was infarcted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

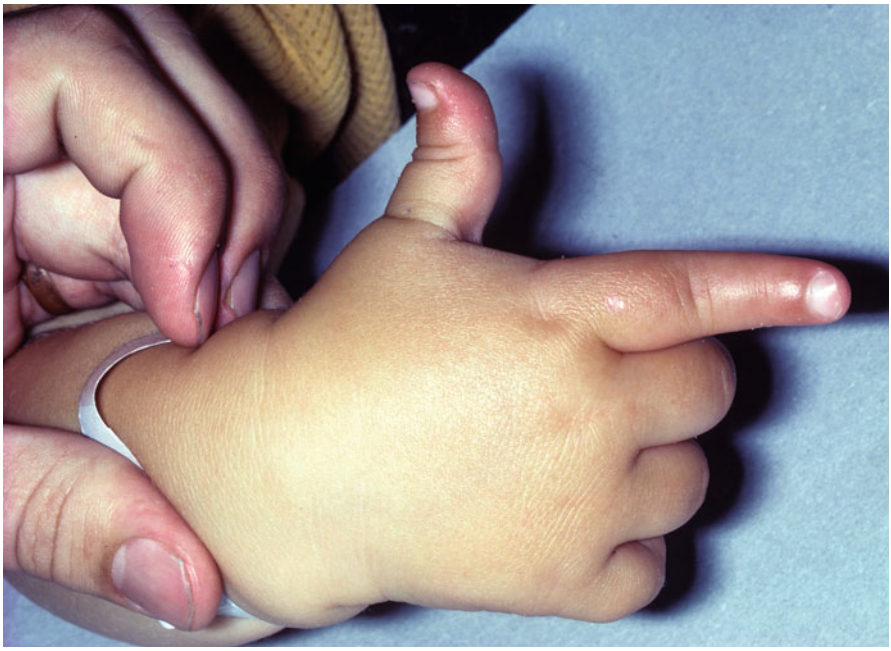

Fig. 4.25 Infant's hand 24 h after section of the palmar nerves to the index finger and thumb. Note that the anaesthetic digits are held out from the others. There was no tendon injury

"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].

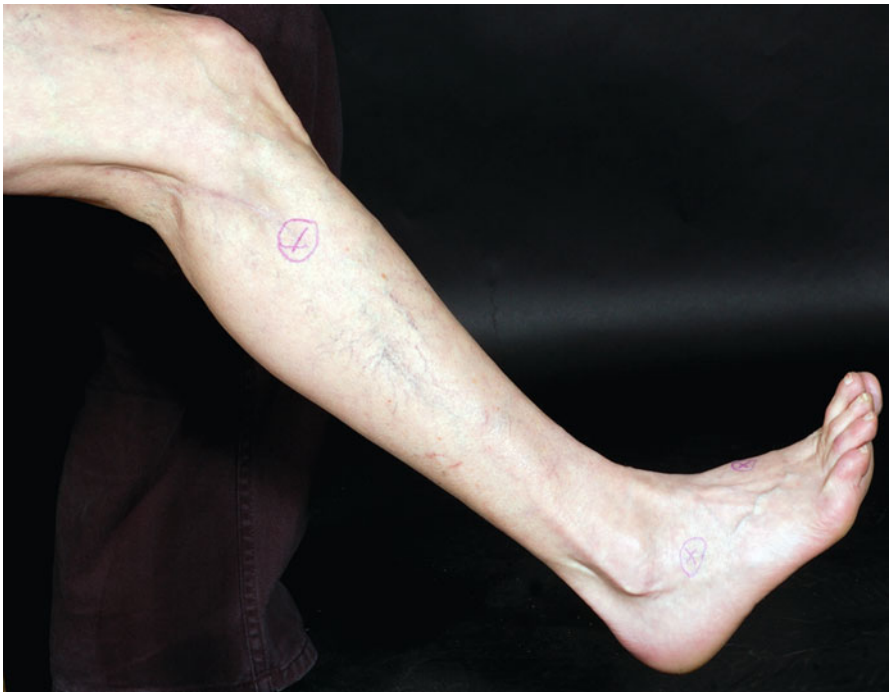

Fig. 4.26 Static and progressing Tinel's signs. A 43 year old woman sustained a complete and very painful, lesion of the common peroneal nerve from the kick of a horse. There was a strong, painful, Tinel sign at the level of lesion at the time of exploration 10 weeks after injury. The nerve was deeply 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

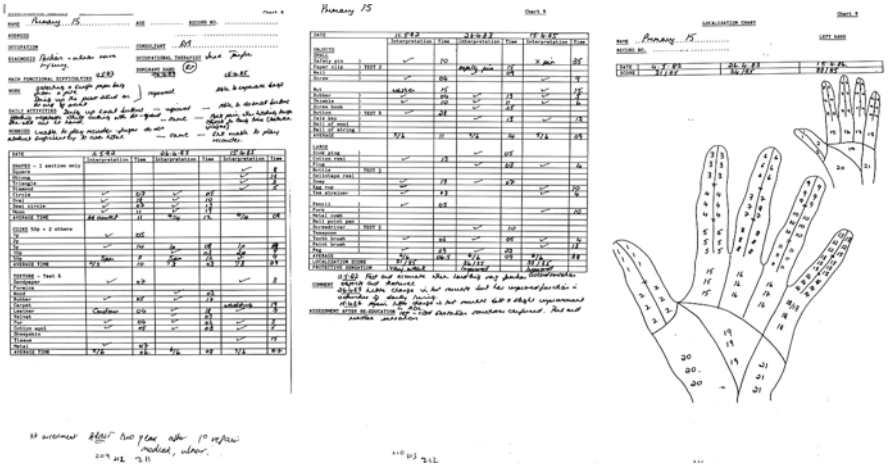

Fig. 4.27 Charts recording recovery of localisation and recognition after repair of median nerve

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

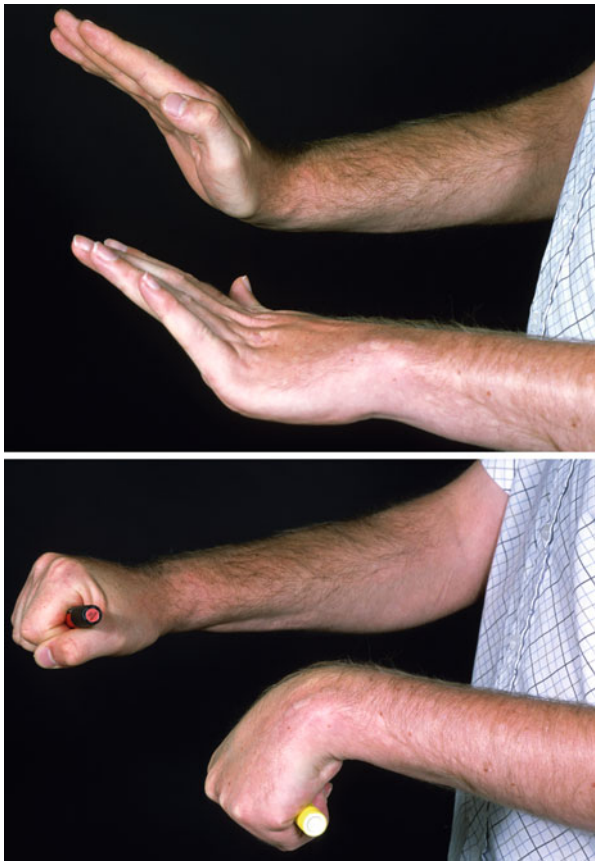

Fig. 4.28 The importance of wrist extension. This 31 year old graphic designer sustained bilateral lesion of the brachial plexus. Avulsion of C7 on the right was treated by flexor to extensor transfer, achieving a power grip at 50 % of estimated normal. On the left C6, C7 and C8 were avulsed and only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

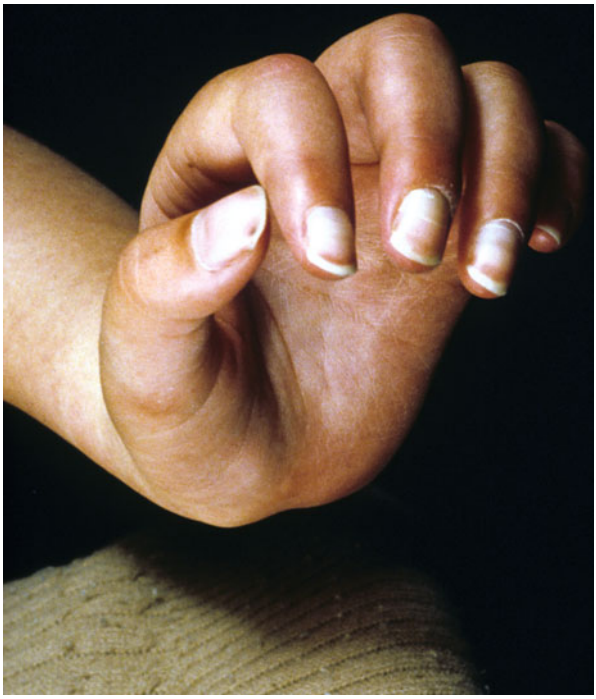

Fig. 4.29 "Tenodesis" effect of the wrist extensor muscles in a 13 year old child with high median and ulnar palsy

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 %

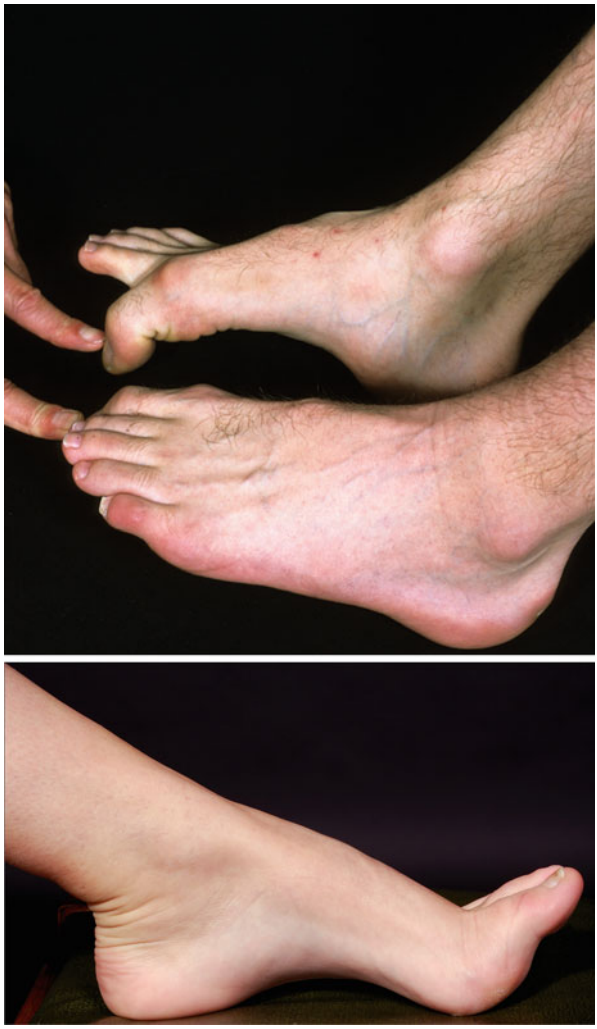

Fig. 4.30 Examples of post ischaemic fibrosis of the anterior compartment of the leg complicating intramedullary nailing. *Above*, showing contracture of extensor hallucis longus; *below*, the contracture involves extensor digitorum longus and extensor hallucis longus

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

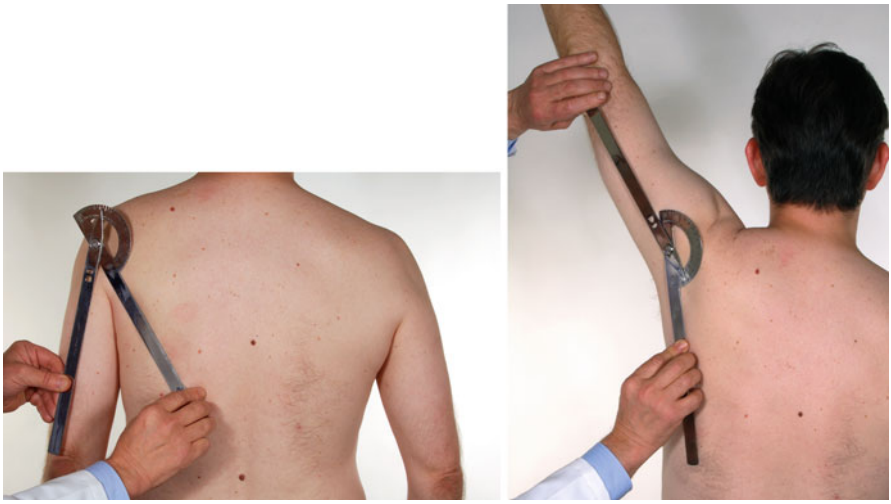

Fig. 4.32 The active inferior scapula-humeral angle (ISHA) in a normal shoulder is about 170°

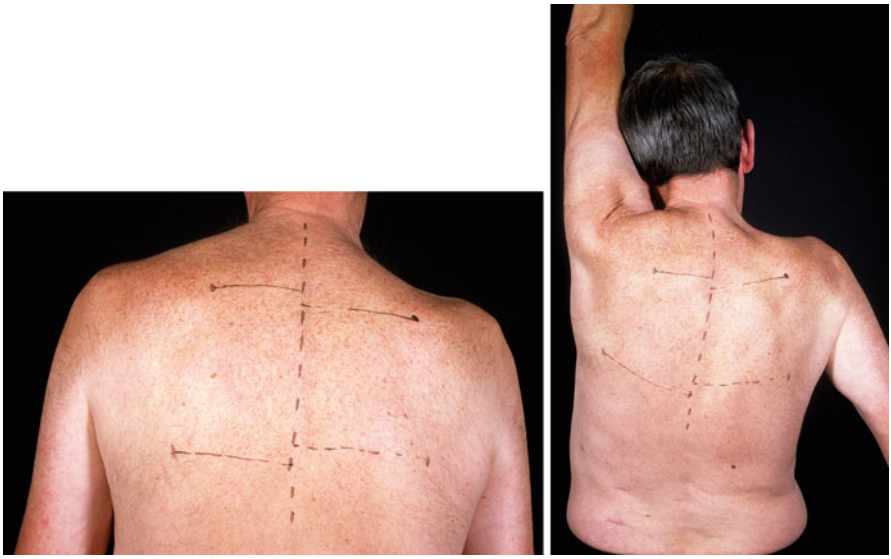

Fig. 4.33 Right spinal accessory palsy. The scapula drops down and away from the spine. The active ISHA is 30°

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

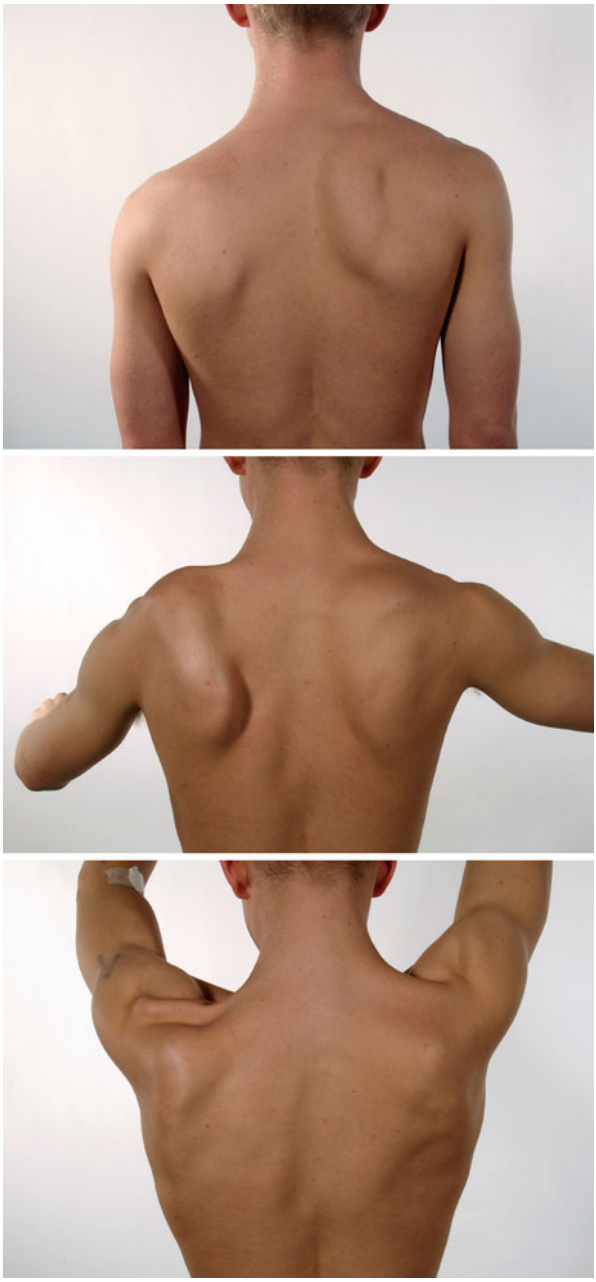

Fig. 4.34 Left spinal accessory palsy. Scapular winging, without prominence of the lower fibres of the trapezius, in a case where there is some early recovery into the upper fibres after repair of the spinal accessory nerve. At rest, the scapula is displaced downwards and away from the spine

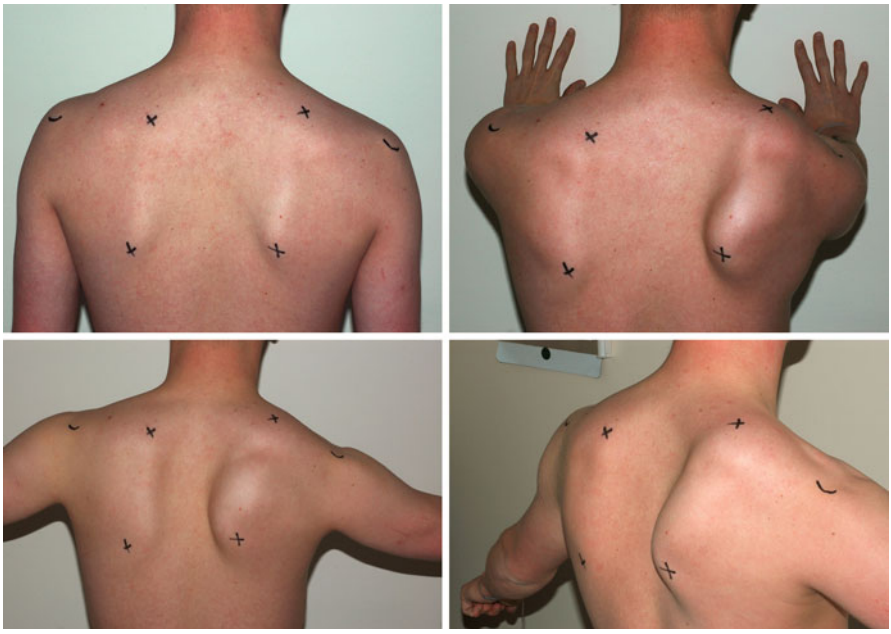

Fig. 4.35 Scapular winging in nerve to serratus anterior lesion. This is easily distinguishable from the winging provoked by accessory palsy by the position of the scapula which is drawn upwards and towards the spine by the unopposed action of trapezius, levator scapulae, and rhomboids

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.

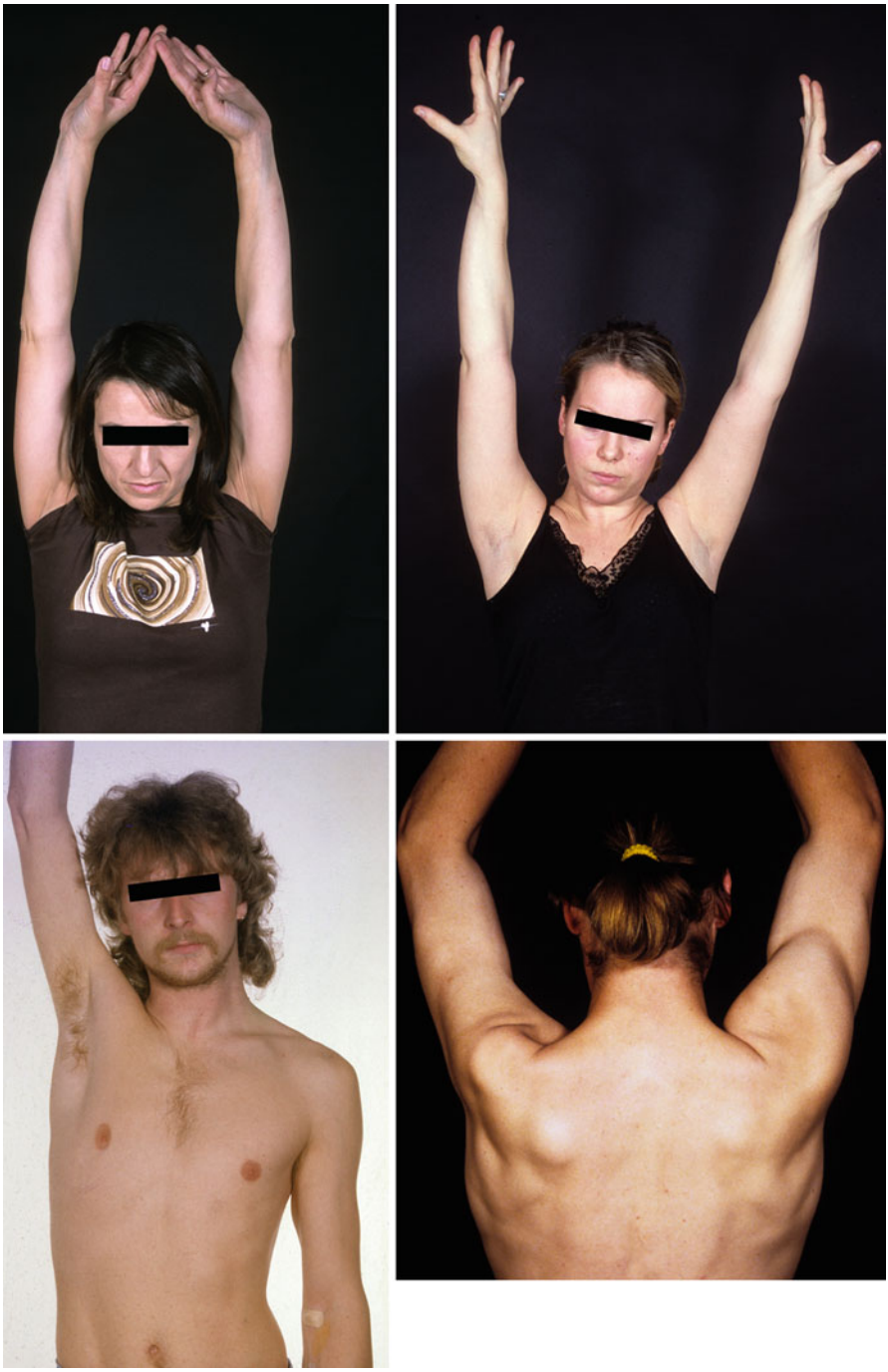

Fig. 4.36 Elevation of the upper limb in full medial rotation by the supraspinatus in the absence of deltoid in four cases of proven rupture of the circumflex nerve. *Bottom left* and *right*: note the activity in the clavicular head of pectoralis major

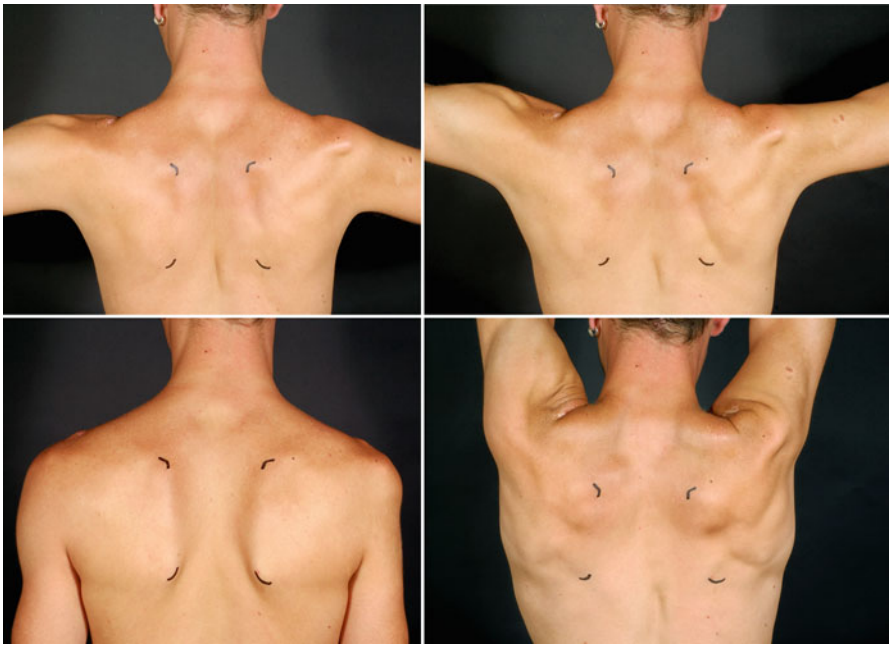

Fig. 4.37 The movement of the scapula in another case of rupture of the right circumflex nerve. The active ISHA on the side of the injury (*bottom right*) is reduced by 20° showing that 20° of the range of elevation is provided by extra movement at the thoraco-scapular joint

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.

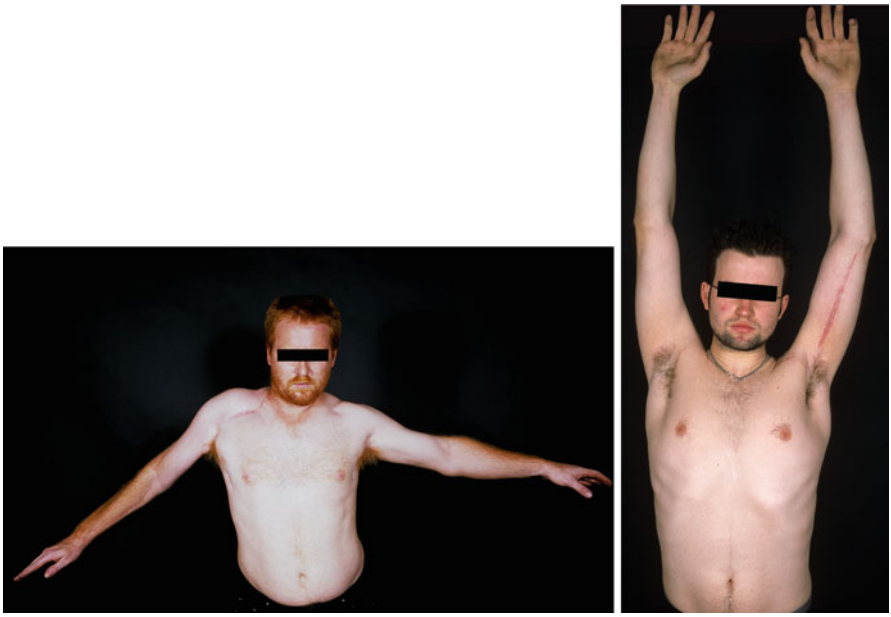

Fig. 4.38 Combined injuries to the suprascapular and circumflex nerves. *Left*: showing the range of elevation in a patient with irreparable injury to the right suprascapular nerve but with a good result after repair of the circumflex nerve. *Right*: showing the elevation in another patient in whom repair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

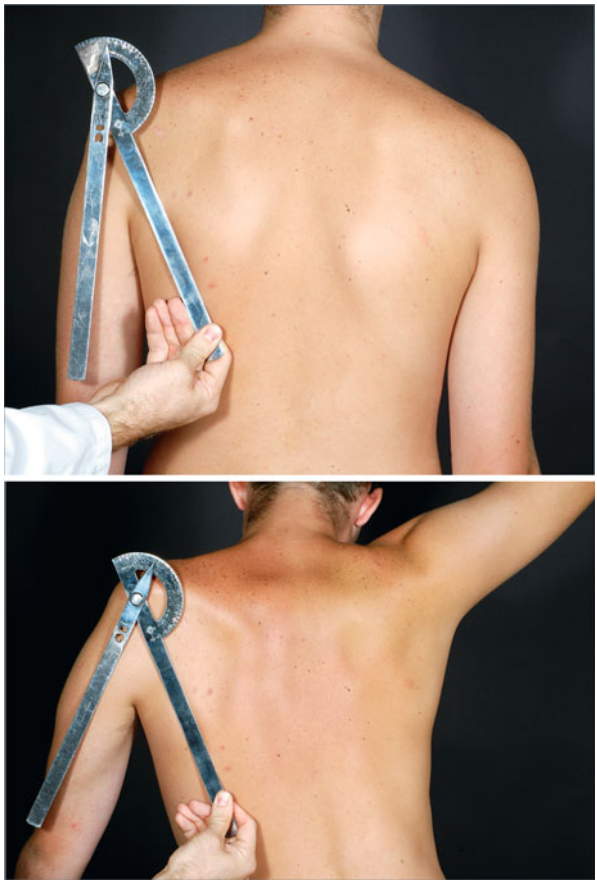

Fig. 4.39 Initiation of abduction, with opening of the active ISHA was the first sign of recovery into supraspinatus after repair of the suprascapular nerve. The lesion of the circumflex nerve was irreparable

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;

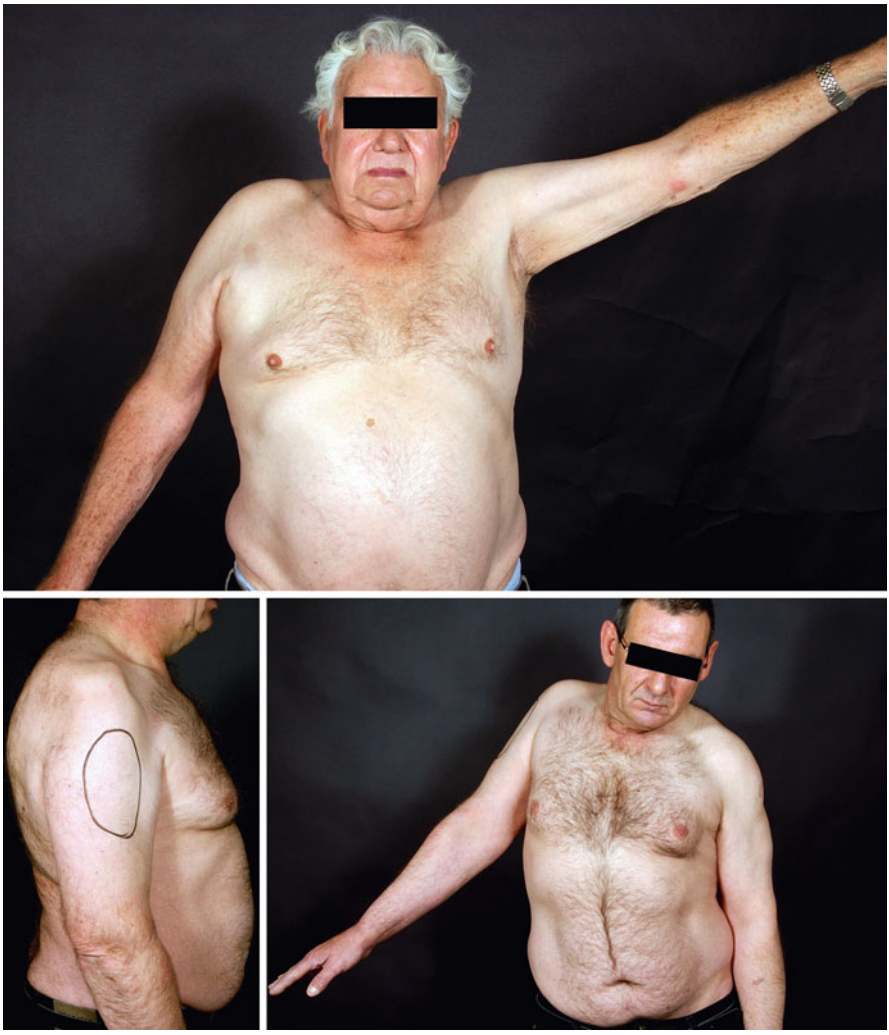

Fig. 4.40 Rupture of rotator cuff with lesions of the suprascapular and circumflex nerves from fracture/dislocation of the shoulder. *Above* showing the range of elevation at the right shoulder in 74 year old ex-paratrooper in whom there was clear evidence of recovery for both of the nerves. *Below*: 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

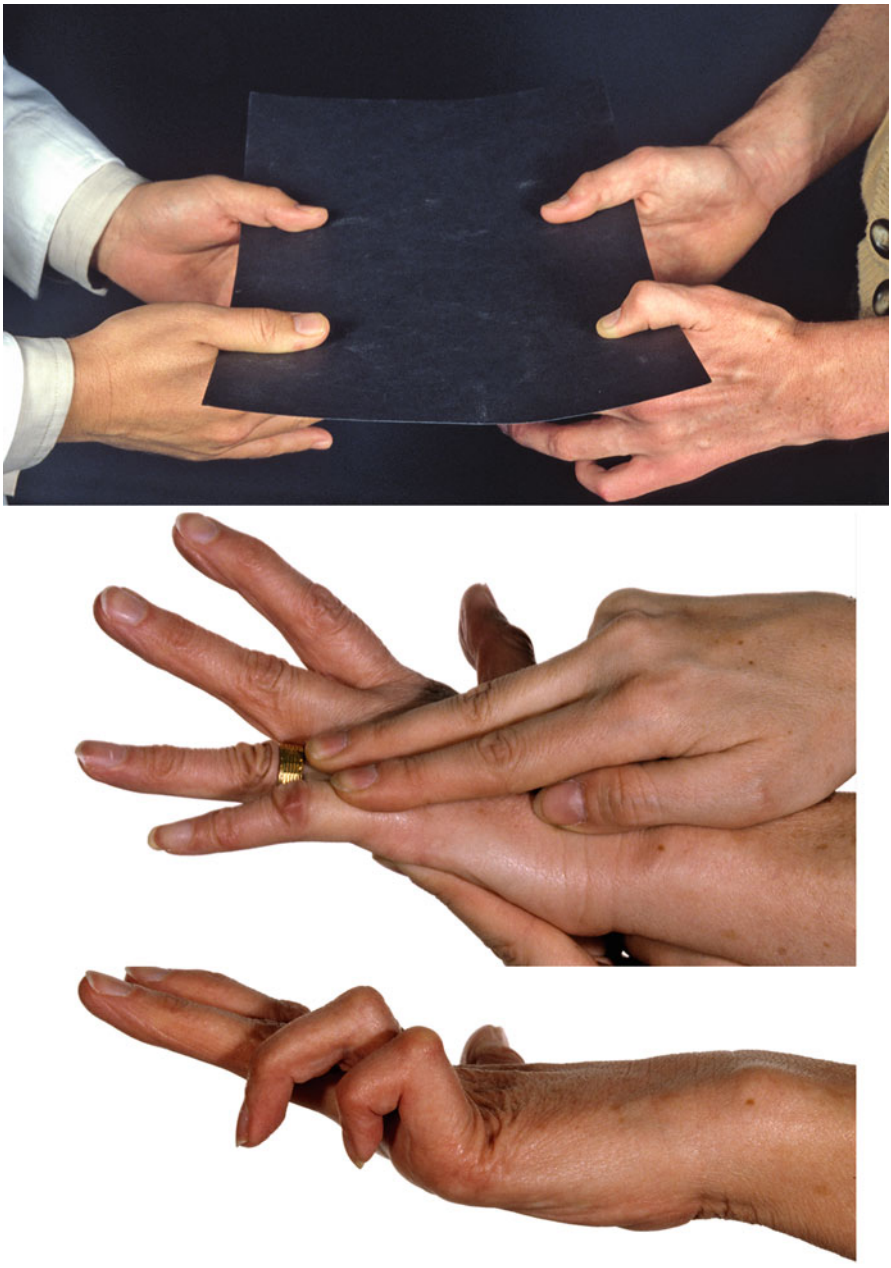

Fig. 4.41 The small muscles of the hand in lesions of the ulnar nerve. *Above*: "Froment's sign" is positive in the patient's left hand. *Below*: clawing is corrected by passive flexion at the metacarpophalangeal joints

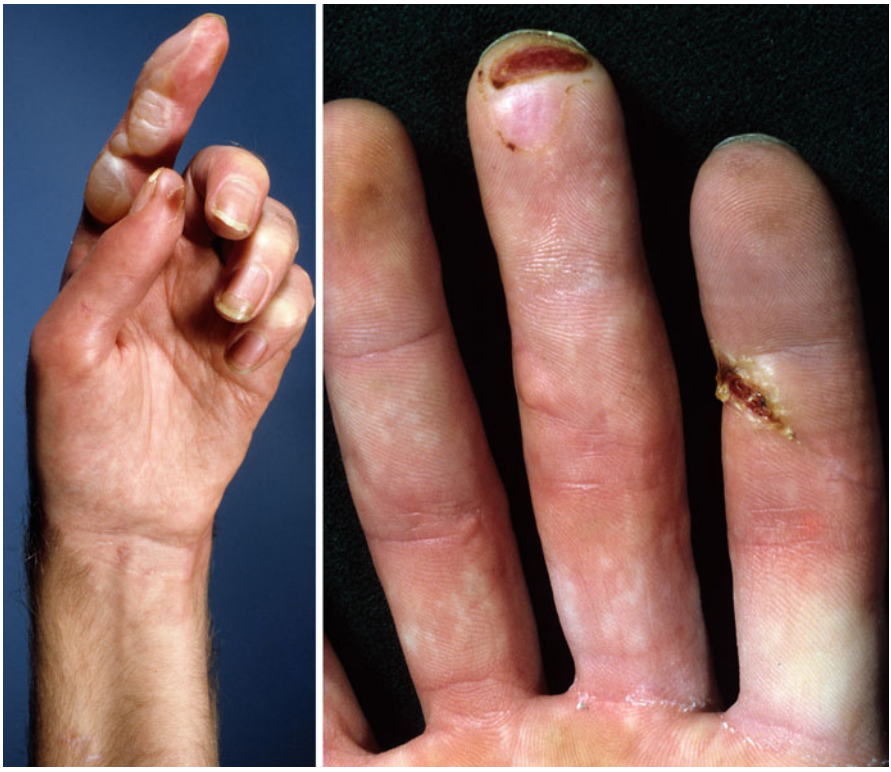

Fig. 4.42 Late changes after nerve injury. *Left*: there is sympathetic paralysis and unnoted burns after high lesion of the median nerve. *Right*: wasting and ulceration of the skin of middle finger and accidental injury to index finger after median nerve injury. The patient was working as a stone mason

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.

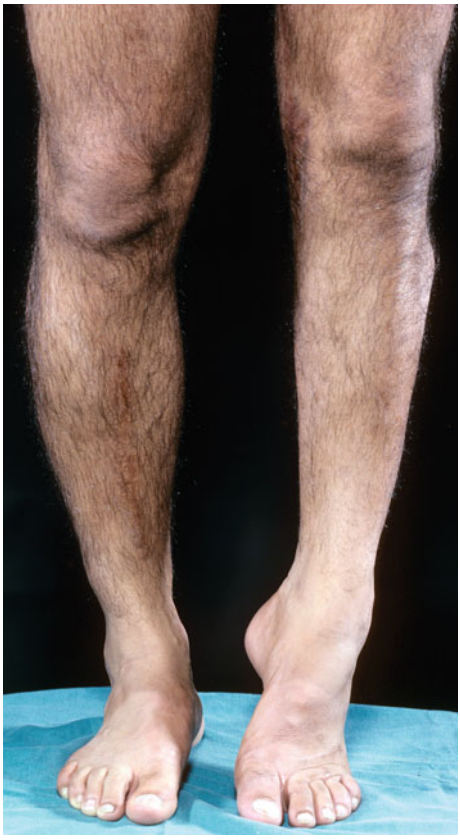

Fig. 4.43 Post ischaemic fibrosis. The lateral geniculate artery was lacerated during arthroscopic meniscectomy. The false aneurysm which ensued remained undetected for 4 days in spite of his intense causalgia. The position of the leg, ankle and foot 3 years after injury

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.

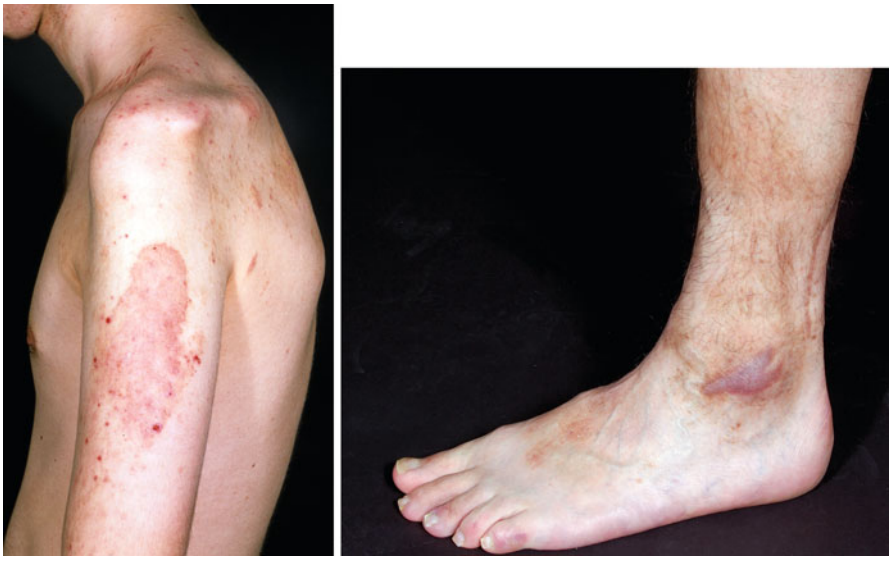

Fig. 4.44 Late skin changes after nerve injury. *Left*: a skin rash in the distribution of C5, 6 months after rupture. *Right*: skin rash in the distribution of the common peroneal nerve 1 year after rupture at the knee

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

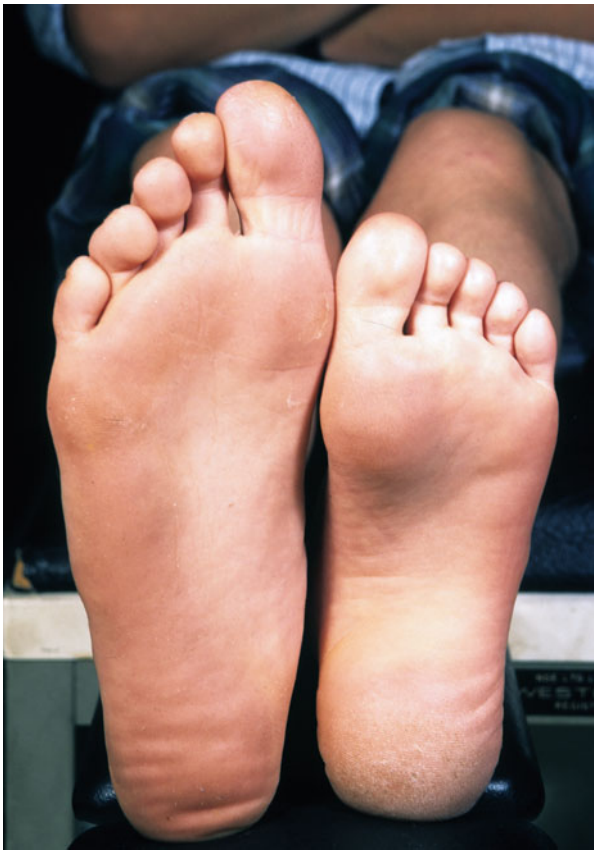

Fig. 4.45 Atrophy of the left foot in an 11 year old boy 4 years after transection of the tibial and common peroneal nerves at the knee. The repair of the common peroneal nerve was successful, that of the tibial nerve failed

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*.

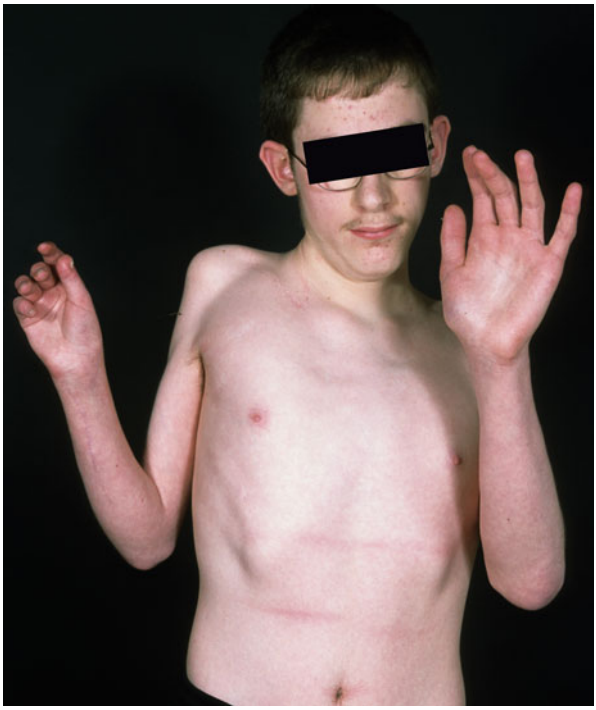

Fig. 4.46 Bilateral lesion from breech delivery. On the right – the lesion was complete and complicated by phrenic nerve palsy. Recovery in C5 and C6 was poor. At the age of 7 years, accessory to suprascapular nerve transfer restored some lateral rotation. On the left there was avulsion of C5 and rupture 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.

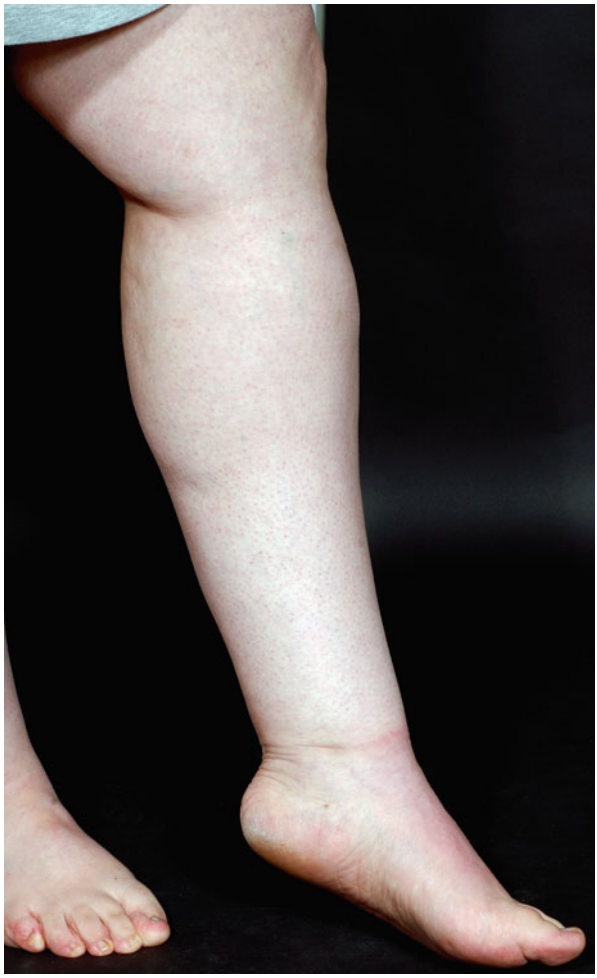

Fig. 4.47 Extreme central sensitisation in post traumatic neuralgia. Intra operative incomplete section of the medial plantar nerve in a 51 year old woman. She was treated for many months by drugs and blocks for an incorrect diagnosis of CRPS1. By 3 years she could not walk because of intense allodynia 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).

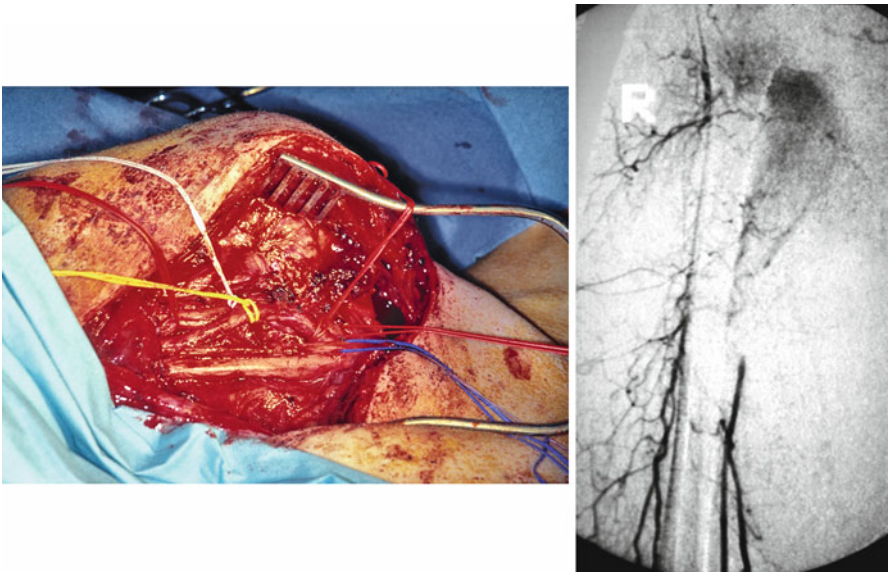

Fig. 4.48 Causalgia in a 55 year old woman after removal of a lipoma in the axilla. At operation, 3 weeks later, an iatrogenous false aneurysm of the brachial artery was displayed. The median and ulnar nerves were displaced and compressed. Her pain was relieved by correction of the aneurysm, decompression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

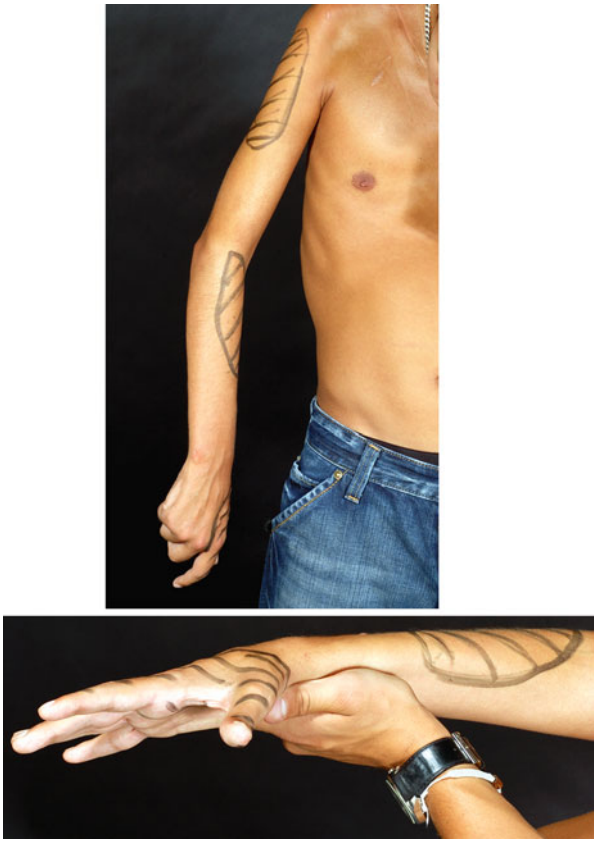

Fig. 4.49 Intense shooting, lightning like pain was felt in the dermatomes of C5, C6 and C7 in this patient on the day of injury. These three nerves were avulsed from the spinal cord in a motor cycle accident

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.

References

1. Birch R. Clinical aspects of nerve injury. In: Birch R. Surgical disorders of the peripheral nerves. London: Springer; 2011. p. 145–90, Chapter 5.
2. Birch R. The closed supraclavicular lesion. In: Birch R. Surgical disorders of the peripheral nerves. London: Springer; 2011. p. 375–428, Chapter 9.
3. Birch R. Pain. In: Birch R. Surgical disorders of the peripheral nerves. London: Springer; 2011. p. 527–62, Chapter 12.
4. Birch R, Eardley W, Ramasamy A, Brown K, Shenoy R, et al. War nerve injuries part 1 – epidemiology pp 523–528: part II – outcomes. J Bone Joint Surg Br. 2012;94B:529–35.
5. Cokluk C, Aydin K. Ultrasound examination in the surgical treatment for upper extremity peripheral nerve injuries: part I. Turk Neurosurg. 2007;17(4):277–82.
6. Cokluk C, Aydin K. Ultrasound examination in the surgical treatment for upper extremity peripheral nerve injuries: part II. Turk Neurosurg. 2007;17(3):197–201.
7. Coupland RM. War wounds of limbs. Oxford/London: Butterworth–Heinemann; 1993.
8. Highet WB. Memorandum to the nerve injuries committee of the Medical Research Council. In: Seddon HJ, editor. Peripheral nerve injuries Medical Research Council Special Report Series No. 282. London: HMSO; 1954. p. 355.
9. Jerosch-Herold C. Assessment of sensibility after nerve injury and repair: a systematic review of evidence for validity, reliability and responsiveness of tests. J Hand Surg Br. 2005; 30B:252–64.
10. Kato N, Birch R. Peripheral nerve palsies associated with closed fractures and dislocations. Injury. 2006;37:507–12.
11. Kaufmann KR. Quantitative muscle strength assessment. In: Dyck PJ, Thomas PK, editors. Peripheral neuropathy. 4th ed. Philadelphia: Elsevier Saunders; 2005. p. 1095–102, Chapter 43.
12. Mills K. Wasting, weakness and the MRC scale in the first dorsal interosseous muscle. J Neurol Neurosurg Psychiatry. 1997;62:541–2.
13. O'Brian M, editor. Aids to the examination of the peripheral nervous system. 4th ed. London: Elsevier; 2000.
14. Seddon HJ. Surgical disorders of the peripheral nerves. Edinburgh/London/New York: Churchill Livingstone; 1975. p. 177.
15. Smith SJM, Knight R. Clinical neurophysiology in peripheral nerve injuries. In: Birch R. Surgical disorders of the peripheral nerves. London: Springer; 2011. p. 191–230, Chapter 6.

16. Toros J, Karabay N, Őzaksar K, Sugun TS, Kayalar M, Bal E. Evaluation of peripheral nerves of the upper limb of the upper limb with ultrasonography. J Bone Joint Surg Br. 2009;91B:762–6.
17. Trumble TE, Kahn U, Vanderhooft E, Bach AW. A technique to quantitate motor recovery following nerve grafting. J Hand Surg Am. 1995;20A:367–72.
18. Willner C, Low PA. Pharmacologic approaches to neuropathic pain. In: Dyck PJ, Thomas PK, Griffin JW, Low PA, Poduslo JF, editors. Peripheral neuropathy. 3rd ed. Philadelphia: Saunders; 1993. p. 1709–20, Chapter 94.
19. Wynn Parry CB. Rehabilitation of the hand. 4th ed. London: Butterworth; 1981.
20. Wynn Parry CB, Salter RM. Sensory re-education after median nerve lesions. Hand. 1976;8:250–7.