

## 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.

*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.

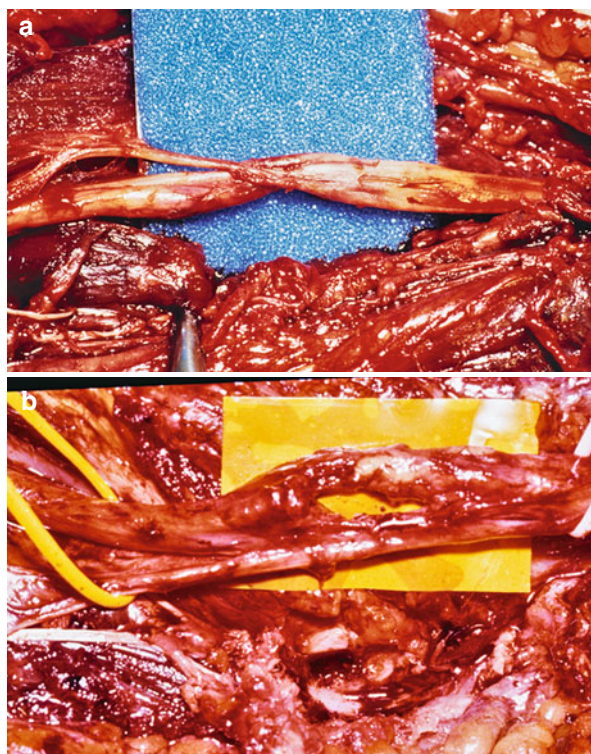
Most injuries of nerves short of transection inflict damage of all three grades of severity. This is the usual pattern for the sciatic nerve stretched but not ruptured by dislocation of the femoral head and for the cords of the brachial plexus stretched but not ruptured by anterior dislocation of the 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.

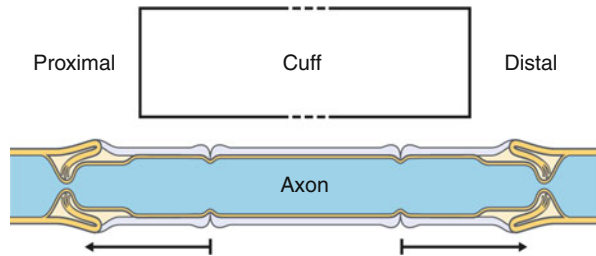
This form of centripetal paralysis is experienced by an observer following the classical experiment of application round the arm of a cuff inflated to suprasystolic pressure. First, there is loss of superficial sensibility. This is succeeded by a gradual loss of motor power. The first pain response is 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).

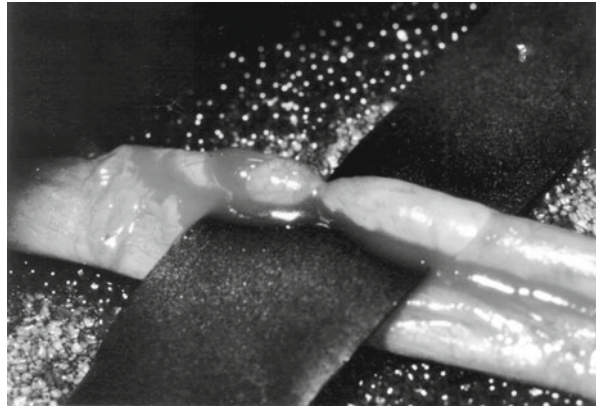
### 2.1.2 Conduction Block in Myelin Deformation and Focal Demyelination

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

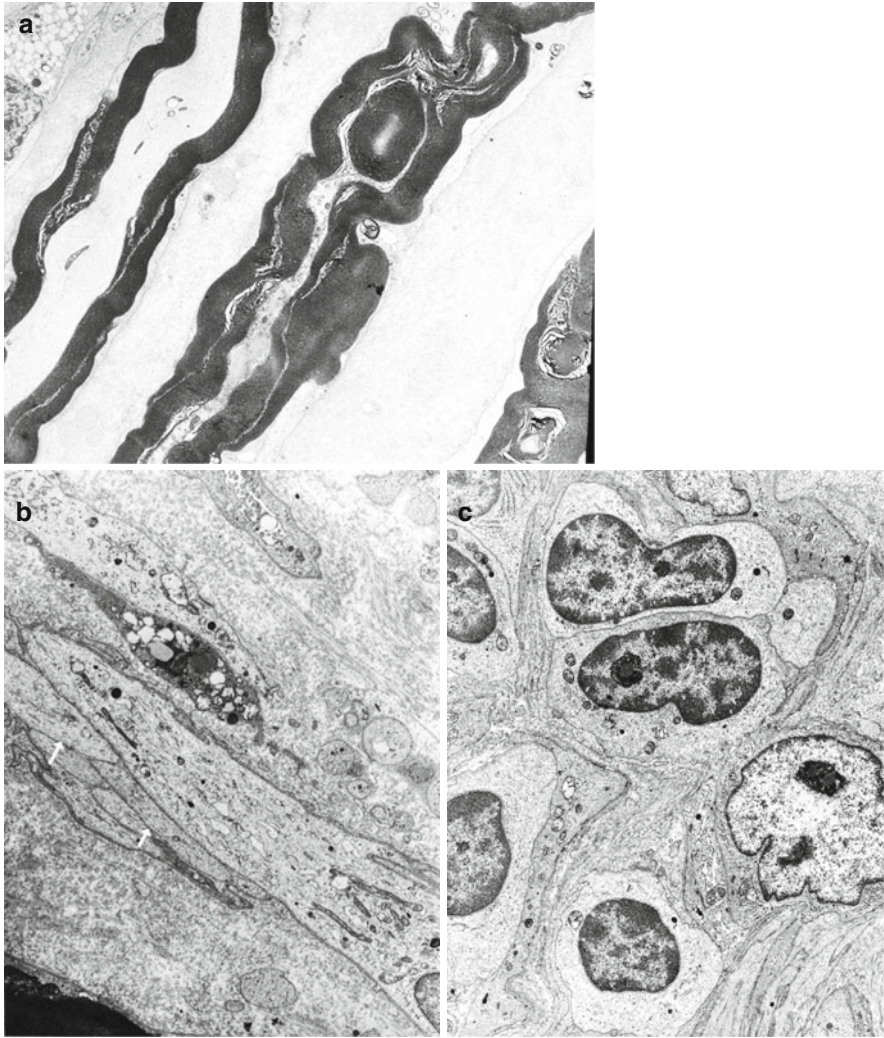
Wallerian degeneration affects not only the axon but also the cell body; not only the neurone but also its Schwann cell ensheathment and its myelin sheath. There are changes too in the endoneurial cells and, over longer periods of time, in the motor and sensory end-organs. Distal to the site of injury 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

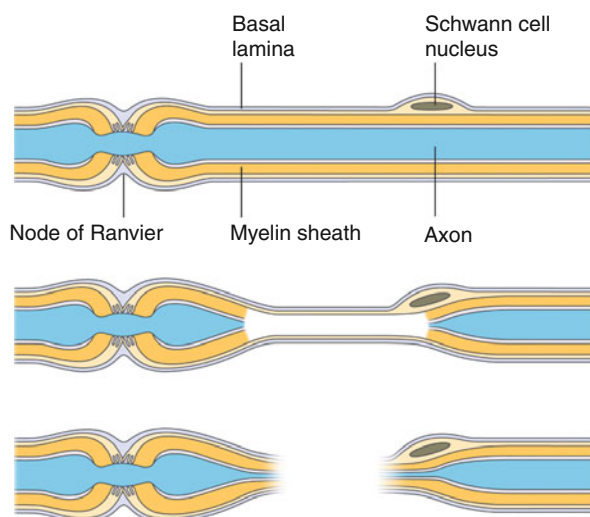
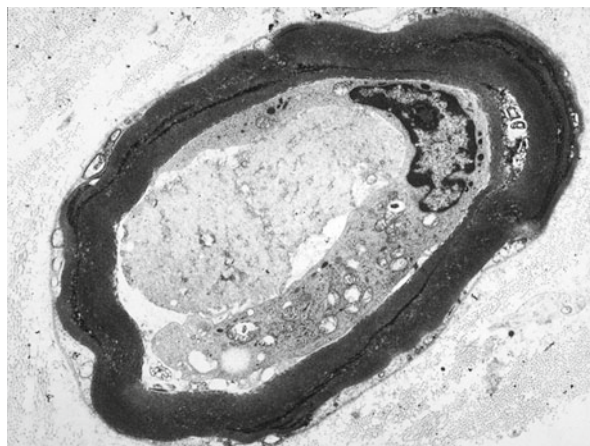


**Fig. 2.5** Changes in the distal stump of ulnar nerve transected 3 weeks previously. (a) Disintegration of the axon and the myelin sheath. Axoplasm and neurofilaments are seen in the lower fibre  $\times 2,210$ . (b) Another part of the same specimen. Myelin debris within a macrophage (*asterisk*),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

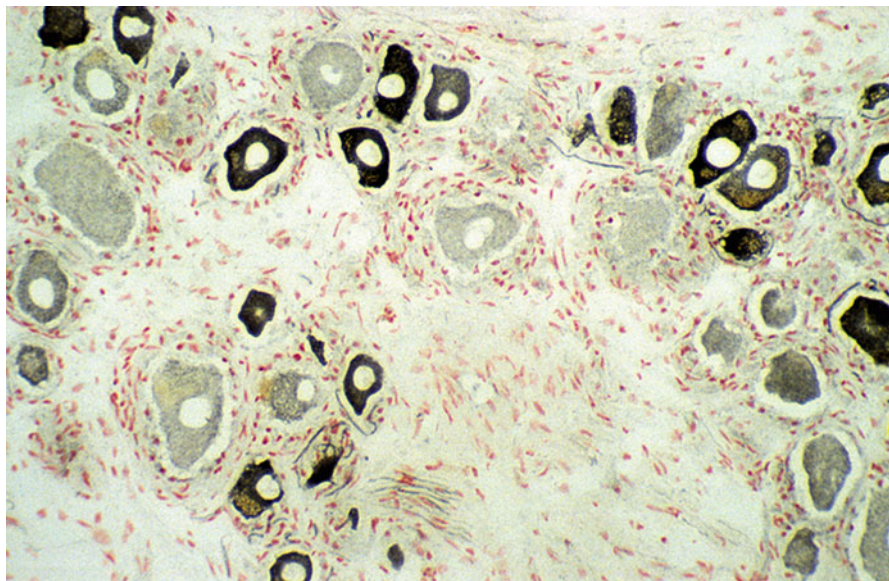


**Fig. 2.6** Wallerian degeneration in the distal stump of fifth cervical nerve ruptured 3 weeks previously. The axon is collapsing and it is surrounded by a macrophage  $\times 11,000$  (EM)



**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.



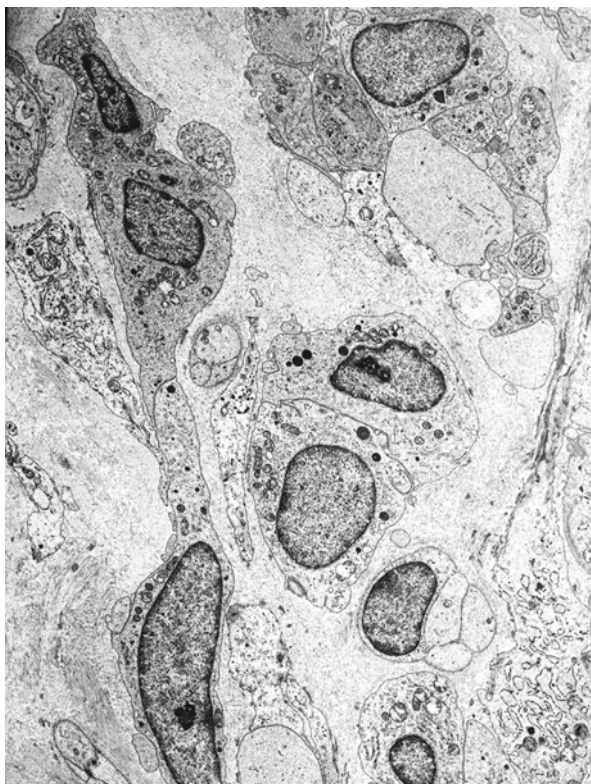
**Fig. 2.8** TRPV1 (the heat and capsaicin receptor) in a human dorsal root ganglion 6 weeks after avulsion showing immunostaining in small diameter neuronal cell bodies and axons  $\times 40$

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  $\times 3,245$  (EM)



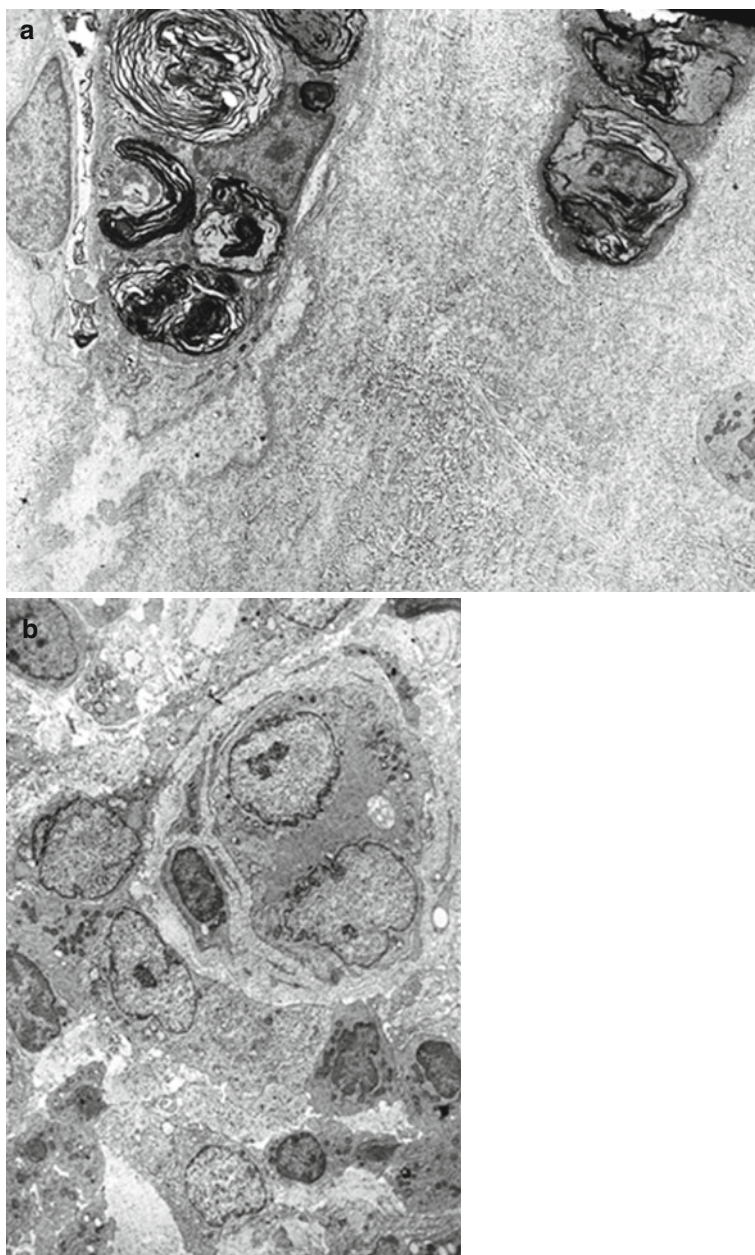
fibrosis within the distal stump. With delay, the number of Schwann cells in the distal stump diminishes and they become less receptive to regenerating axons because of the decrease in the expression of receptors which are normally important in Schwann cell-axon signalling, effects which are worsened 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).

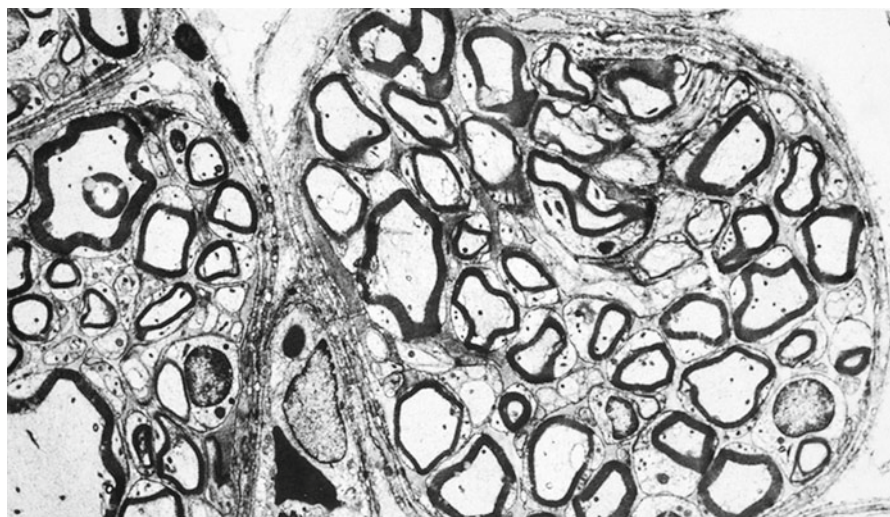
### 2.2.3 *Contralateral Effects*

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





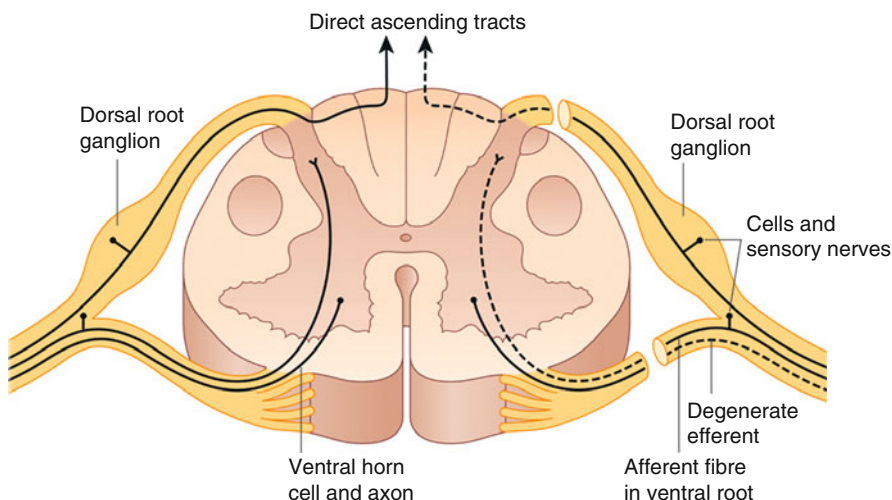
**Fig. 2.10** The results of rupture complicated by sepsis. Electronmicroscopic study of serial (2 cm interval) biopsies of proximal stump of sciatic nerve of 34 year old man 8 months after rupture by fracture and subsequent sepsis. (a) 2 cm from the tip of the proximal stump  $\times 2,340$ . Relics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

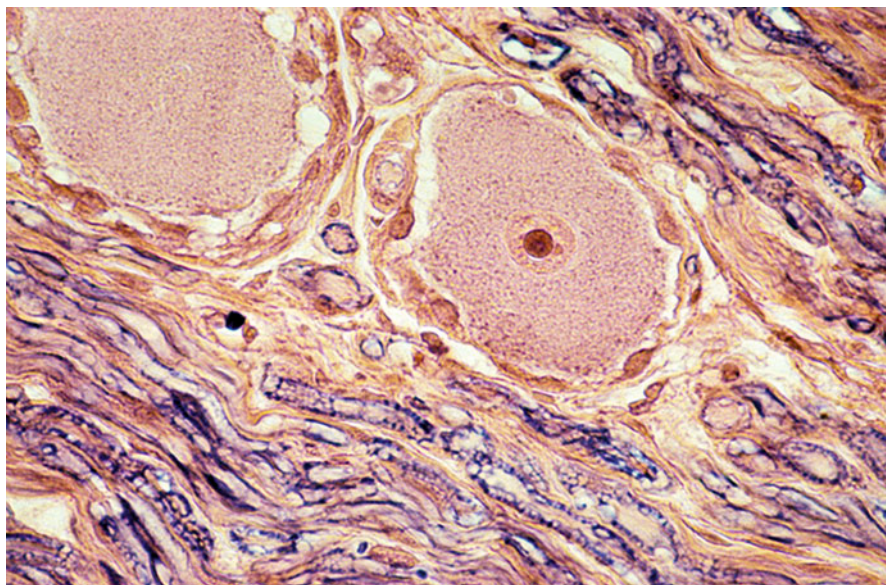




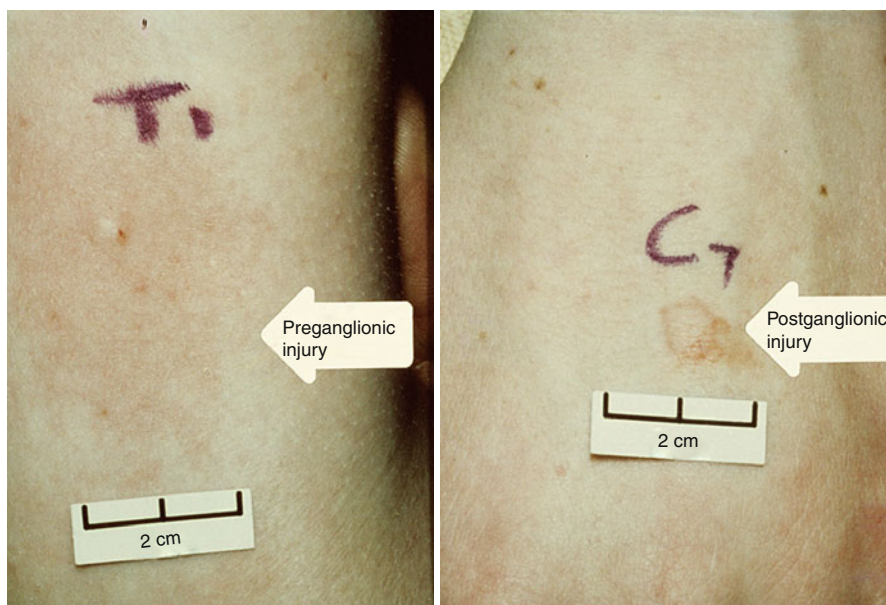
**Fig. 2.12** The nerve and roots detached from the spinal cord. Note the intact dorsal root ganglion cell, with healthy axons in the detached parts of the roots, and the degeneration of the efferent fibre in the ventral root and of the central projections of the afferent fibres

### 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  $\times 960$



**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*)  $\times 11,115$  (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 induces 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

<sup>a</sup>The sciatic trunk is considered as its two component nerves, the tibial and common peroneal

<sup>b</sup>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 reoperation 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 intervertebral 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 bupivacaine 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 % bupivacaine 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

Age	Sex	Side	Phrenic palsy	Lower motor neurone		Cervical sympathetic	Upper limb sympathetic	Spinothalamic tract		Pain
				Ipsilateral	Contralateral			Ipsilateral	Contralateral	
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	PNI 3: VAS 8 neck, shoulders and right hand
									Lower limb	
4 <sup>a</sup>	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

<sup>a</sup>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.

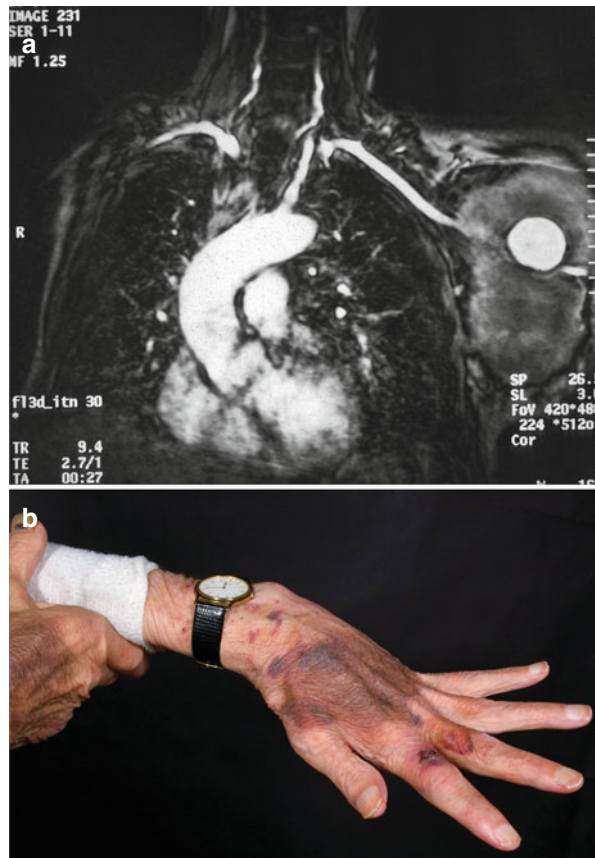
**Fig. 2.17** Recovering femoral palsy from haematoma in the femoral triangle in a 69 year old farmer who was taking warfarin after aortic valve replacement. Two months previously he had injured his thigh whilst vaulting a gate. He experienced severe pain for 24 h but this recovered spontaneously. The 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 infra-clavicular 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. Wilbourn's comment bears repeating: "distal pulses are normal as they are with most compartment syndromes because the elevated pressure, although sufficient to collapse the vasa nervosa, 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

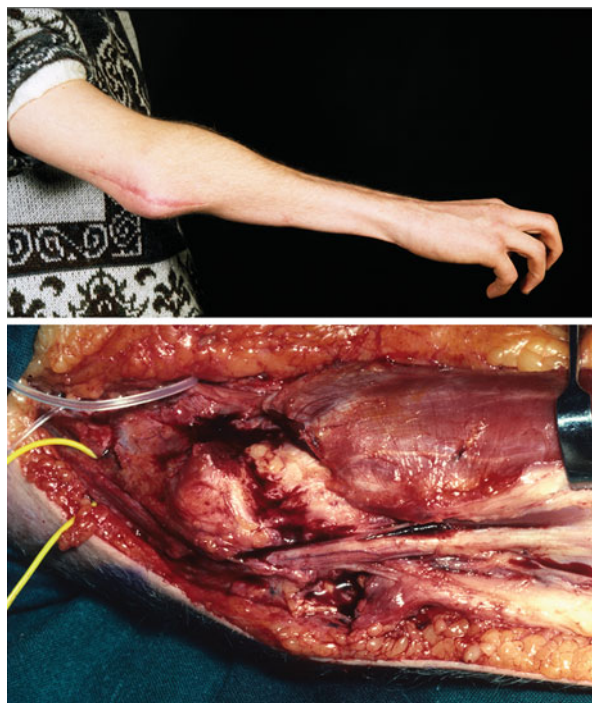


available for surgical decompression before irreversible nerve damage occurs, obtaining these is rarely justified". The 16 patients described by Stenning, Drew and Birch [33] exemplify the syndrome. There was, in all of these cases, an injury to the axillary artery or one of its offsets caused by dislocation 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]. Wajsborg 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 ( $\Phi$ ) 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

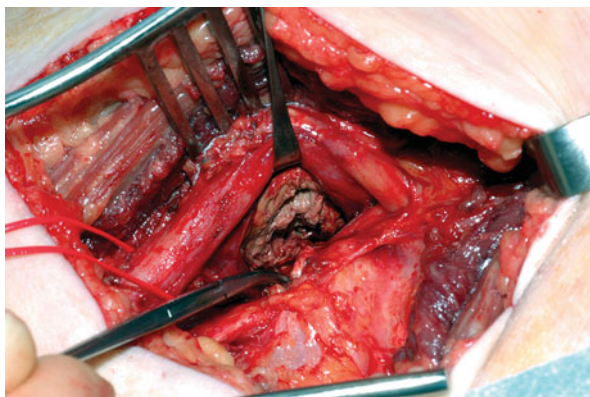


Critical illness neuropathy may develop in patients who develop multi organ failure or sepsis. It is possible that the intense compression of nerve trunks within oedematous and swollen limbs contributes to this disorder (Fig. 2.21).

### 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 bupivacaine 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

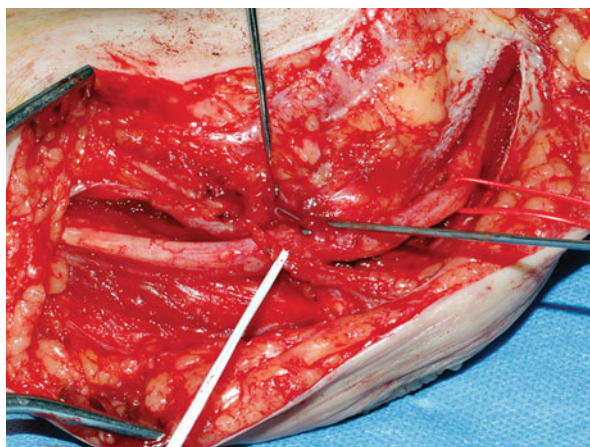
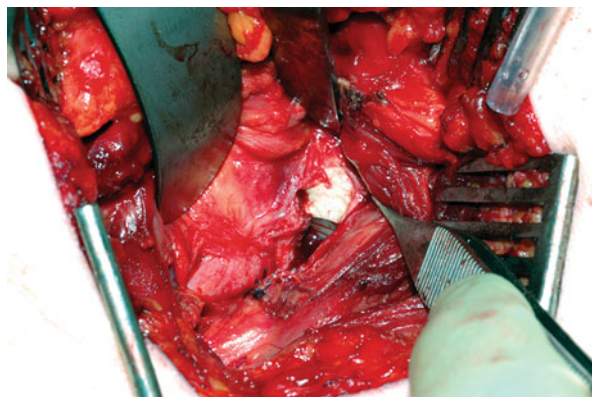
*Case report:* A 12 year old boy fell from a swing and experienced much pain. There was deep bruising in the left buttock and he presented with a high sciatic palsy which involved the superior gluteal nerve. The common peroneal nerve was more severely affected, NPI at 6 weeks revealing loss of conduction 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 equinovarus.

### 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 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



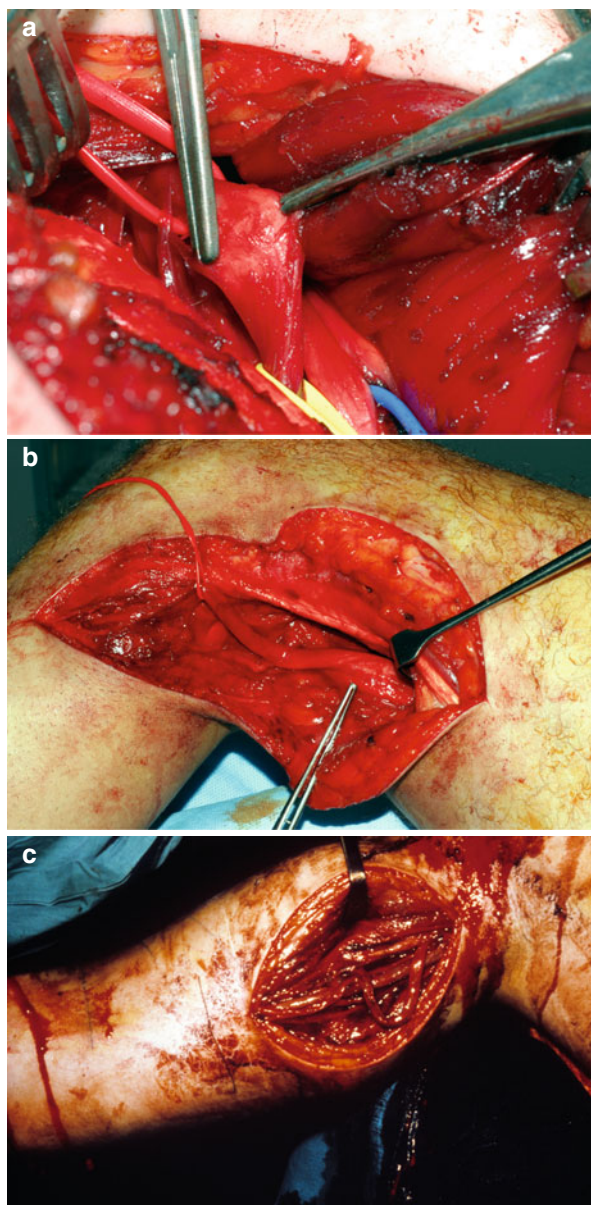
difficult operation of internal fixation of a fractured shaft of humerus, realised that the hitherto protected radial nerve had inadvertently slipped between the plate and the bone. The nerve was at once extricated. The duration of compression was, at the most, 5 min. The nerve was re-explored at that 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.

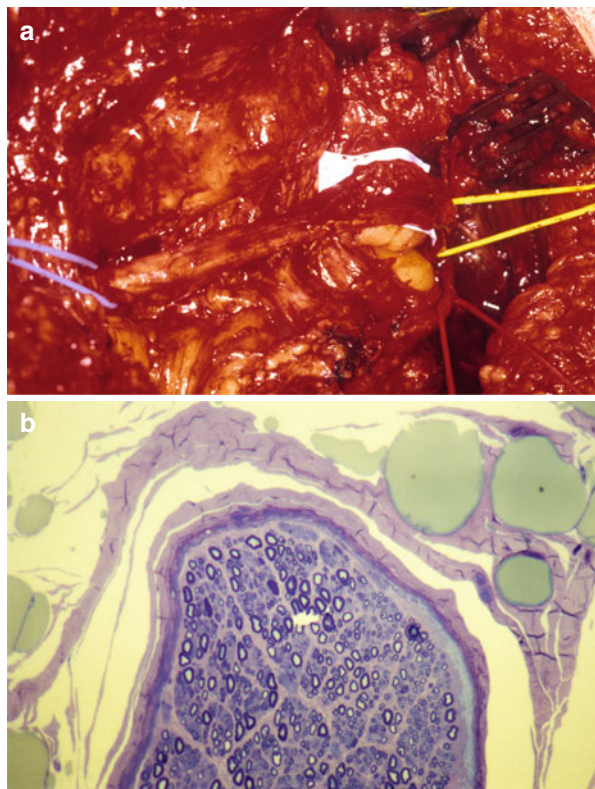
### 2.4.9 Thermal Injury

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  $\times 100$



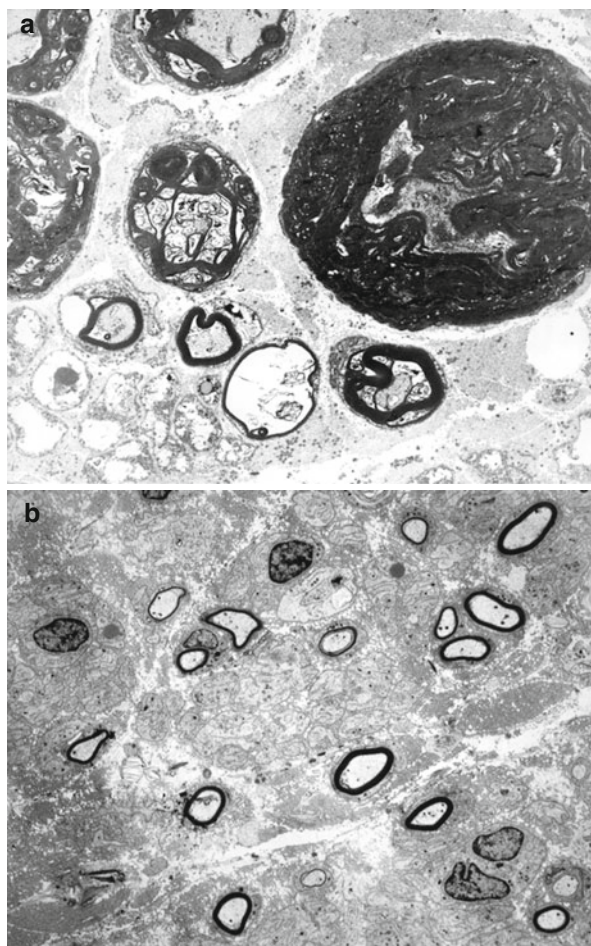
to recall that the temperature of polymerising cement rises to  $95^{\circ}\text{C}$  about 15 min after mixing, and remains above  $70^{\circ}$  for another 12 min. Birch et al. [2] had the opportunity of examining a length of sciatic nerve damaged by the heat of polymerising cement. They later took 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

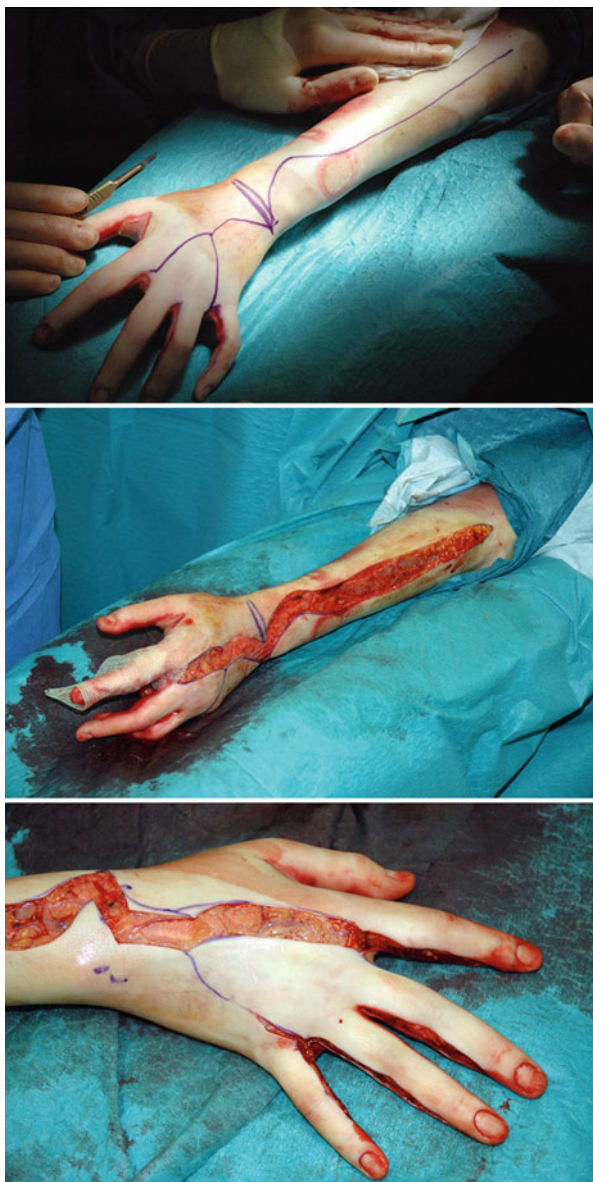
**Fig. 2.28** Thermal damage to the median nerve after exposure to the heat of setting cement, showing the limited longitudinal extent of the neural lesion. (a) Virtual destruction of axoplasm and cellular elements at the site of the lesion  $\times 3,600$ . (b) Healthy axons and collagen 10 mm from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



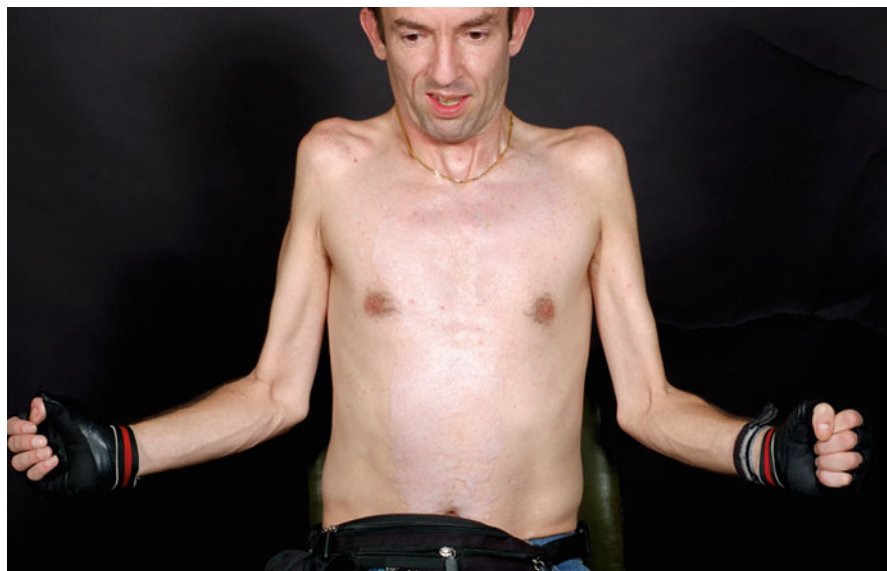
conductor through the skin to the tissues; “arc” burn, where a current passes external to the body to the ground; secondary flame burn, from ignited clothing; and direct burn, from a hot electrical element. Reasonable spontaneous recovery occurred in those cases where the blood supply to the adjacent 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 % bupivacaine. 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.

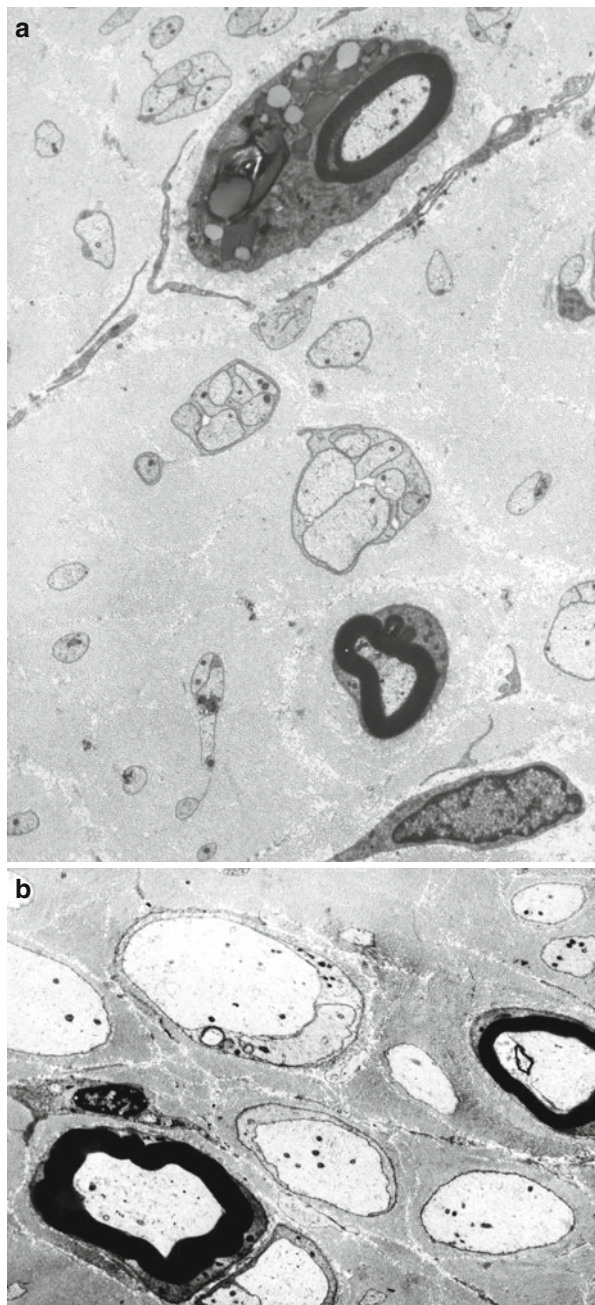
### **2.4.12 *Vibration Injury***

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  $\times 4,125$ . **(b)** Extensive demyelination  $\times 4,125$  (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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Birch, R.

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