Anaesthesia, 2000, **55**, pages 980–991

REVIEW ARTICLE

Peripheral nerve injuries associated with anaesthesia

R. J. Sawyer,[1] M. N. Richmond,[2] J. D. Hickey[3] and J. A. Jarrratt[4]

1 Specialist Registrar in Anaesthesia, and 2 Consultant Anaesthetist, Department of Anaesthesia, and 4 Consultant Clinical Neurophysiologist, Department of Clinical Neurophysiology, Royal Hallamshire Hospital, Glossop Road, Sheffield S10 2JF, UK
3 Medical Director, Medical Protection Society, 33 Cavendish Square, London W1M 0PS, UK

Summary

Peripheral nerve injuries can occur at any time during the peri-operative period. The long-term disability that results may have serious consequences for a patient. The incidence of peri-operative nerve injuries can be reduced by anaesthetists being aware of their causes and pathophysiology. This review article aims to explain the incidence, pathophysiology and medicolegal implications of peri-operative nerve injury and provides suggestions as to how they may best be avoided.

Keywords *Anaesthesia. Complications*: neurological. *Nerve*: damage.

Correspondence to: Dr R. J. Sawyer
Accepted: 4 March 2000

Peri-operative nerve lesions can mar a successful surgical procedure, handicap a patient with a severe functional disability and leave the medical team facing possible protracted and unpleasant litigation. The true incidence of peri-operative nerve damage remains unclear and, as a complication, it is probably under-reported. The presenting neurological symptoms may appear minor in nature to the attending physician, when compared with the clinical disorder that led to the initial surgical procedure, and, as such, may be dismissed. There is a belief that the lesion will be self-limiting with a full and spontaneous recovery; however, this is often not the case.

It is our impression that the incidence of these lesions is sufficiently great to warrant further study into their causation and prevention. In this paper we review their incidence, aetiology, pathology, pathophysiology and medicolegal implications, and offer some advice as to their avoidance

Incidence

Nerve palsies following anaesthesia have been observed since shortly after the introduction of ether [1]. Early writers blamed toxic properties of the anaesthetic agent, but Budinger [2] was the first to recognise that the majority were secondary to malpositioning of the patient on the operating table with consequent stretching and/or compression of nerves [3]. Since then, many publications have confirmed this view [4, 5], and suggested a far greater range of causes, including direct needle trauma and chemical irritation.

In 1950, a retrospective review of more than 30 000 cases found an incidence of approximately one per 1000 cases, of which 83% involved the ulnar nerve [4]. In 1987, a prospective study of 6538 patients showed an incidence of postoperative ulnar neuropathy of one per 350 patients [5].

Examination of the American Society of Anesthesiologists (ASA) Closed Claims Study showed that 15% of all claims were related to nerve injury. Ulnar neuropathy comprised one third of the injuries, the brachial plexus nerves 23% and lumbosacral roots 16% [6]. Although the mechanism of injury is uncertain, it is often suspected to be excessive pressure or undue stretching of the nerve. In 60 consecutive case reports dealt with by the Medical Protection Society since 1982 concerning peripheral nerve palsies associated with general anaesthesia, 75% involved a claim of medical negligence. The principal lesions were ulnar nerve palsies (43%) and common peroneal nerve injuries (11%) (Medical Protection Society, personal communication).

© 2000 Blackwell Science Ltd

Aetiology

There are many possible ways in which nerves can be damaged in the peri-operative phase. Events such as direct injury by needles, instruments, suturing, or the injection of neurotoxic material, or even thermal insults from diathermy, can cause nerve damage. The relationship between the agent and its effect is usually unclear. Less obvious, but more frequent adverse events, involve mechanical factors such as compression, stretch [3, 7], angulation, percussion or transection. Ischaemia is a crucial element in many of these injuries [8, 9]. The interdependence between mechanical and ischaemic factors is well recognised, but which is the more important factor in individual cases remains controversial.

Predisposing factors

Nerves may be unduly susceptible to trauma as a result of a pre-existing generalised peripheral neuropathy [10, 11], or a local compression neuropathy, whether overt or subclinical, or rarely as a hereditary predisposition [8, 12]. Nerve lesions are more common in diabetic patients than in the general population and, indeed, the development of a peri-operative nerve lesion should be an indication for further investigation of a possible underlying disease process. Patients who develop a postoperative ulnar neuropathy on one side often have abnormal ulnar nerve conduction on the other side [13].

Other structural causes include congenital abnormalities in the region of the thoracic outlet [8] and arthritis or instability of the elbow joint. Superficial nerves, such as the ulnar and common peroneal nerves, are especially vulnerable in patients who are thin [14].

Among the systemic factors potentially involved in the pathogenesis of peri-operative nerve lesions are hypovolaemia [13], dehydration, hypotension [14], hypoxia and electrolyte disturbances [15]. A high incidence of postoperative neuropathies has been reported after induced hypothermia [16, 17].

Surgical factors

The surgical procedure itself is relevant. All surgical disciplines employ patient positioning techniques, which may predispose to particular nerve injuries.

General surgical procedures on the lower abdominal or inguinal region may give rise to damage to the ilioinguinal, iliohypogastric or genito-femoral nerves either as a result of direct trauma or from excessive flexion of the thigh onto the abdomen [18]. Post vasectomy neuralgia affecting the scrotum occurs as a result of damage to the ilioinguinal nerve. The Lloyd-Davies position for rectal or anal surgery, or the exaggerated lithotomy position for radical prostate surgery both carry a particularly high risk because the patient's legs are flexed and abducted in supports for a prolonged period.

During cardiac surgery, median sternotomy is associated with a relatively high risk of peri-operative injury to nerves of the brachial plexus. The reported incidence ranges from 6 to 38% [19, 20]. The frequency of this procedure ensures that this type of complication is one of the most common. Possible causes include traction on the brachial plexus, compression of its medial cord by the first rib, or fracture of the first rib leading to a hyperabduction thoracic outlet compression syndrome [21]. Coronary artery bypass surgery has also been associated with a high incidence (15%) of postoperative ulnar neuropathies [22, 23].

The sciatic, femoral or obturator nerves may be damaged during total hip arthroplasty, the risk being higher in female patients and in operations requiring significant lengthening of the extremity stretching the sciatic nerve [24]. Rose *et al*. presented 23 cases of peroneal nerve palsy (0.88%) in 2600 knee arthroplasties [25].

The harvesting of bone or vein grafts may be followed by damage to nerves in the vicinity of the donor site. For example, meralgia paraesthetica caused by injury to the lateral femoral cutaneous nerve has been reported following iliac bone procurement [26].

Neurosurgery and the use of the sitting position for approaches to the posterior fossa is associated with nerve injuries. The common peroneal nerve is frequently involved, with an incidence of approximately 1% from a series of 488 [27]. Recurrent laryngeal nerve injury has been described after the use of transoesophageal echocardiography in two patients who underwent craniotomy in the sitting position. It is possible that the large size of the probe, tracheal intubation and excessive neck flexion resulted in pressure on the laryngeal nerve [28].

Anaesthetic factors

Peripheral nerve damage is associated with regional anaesthesia and nerve damage as a result of direct needle trauma and, although relatively rare, is avoidable with a meticulous technique. Peripheral nerve damage is not as dramatic as central neural damage, which may occur with spinal or extradural techniques. Neurological damage may only become apparent up to 1 week after the procedure. In normal clinical practice, an anaesthetist may not see patients after a nerve block, when the neurological signs will have become manifest [29].

The type of needle used in performing a regional technique has been a topic of debate for many years. Needle trauma may damage neural blood vessels causing extra- or intraneural haematoma, and possibly direct trauma to neural fascicles. Either of these may result in a

© 2000 Blackwell Science Ltd

discontinuity of fibres and subsequent pain. An immobile needle technique as described by Winnie [30] is still to be recommended. However, the debate as to whether to elicite paraesthesia or not is still contentious. The use of blunt atraumatic block needles has been suggested because they may help in identifying tissue planes [29]. Animal studies using long-bevelled needles demonstrated less neural damage than short-bevelled needles [31]. However, the optimum design of block needles to be used in humans still needs to be established. Epidemiological data and prospective, randomised clinical trials are not available and there are immense practical difficulties in conducting such trials.

Local anaesthetics used in clinical concentrations are not neurotoxic when applied extraneurally. There may be differences in neurotoxicity in substances injected intraneurally and this has been demonstrated with epinephrine [32]. It is theoretically possible that any preservative in a solution could be neurotoxic and it would therefore be wise to use preservative-free solutions for nerve blocks. The introduction of chemical agents used in skin preparation is a theoretical complication and has not been implicated in peripheral nerve damage.

The use of pneumatic tourniquets, to produce a bloodless field, has greatly reduced the risk of nerve injury as a result of better control of the compressive pressure [33, 34]. There is still a significant risk of compression nerve damage and tourniquet time should be kept as short as possible.

Lingual nerve damage has been associated with tracheal intubation and the use of the laryngeal mask [35, 36]. Irregular and over inflation of the cuff and nitrous oxide distension of an air-containing cuff are factors thought to be contributory.

Pathology and pathophysiology

Peripheral nerves comprise an extruded portion of the nerve cell body, the axon, encased by a series of Schwann cells, which form myelin segments. Each nerve is enclosed within three layers of connective tissue, the inner endoneurium, the perineurium and the outer epineurium.

The relatively simple structure of peripheral nerve results in a relatively small repertoire of responses to injury. These are focal conduction block, with or without detectable minor structural change, demyelination, and degeneration.

Focal conduction block

This is thought to be due to ischaemic anoxia. In animal experiments, modest degrees of tourniquet pressure to a nerve cause conduction block initially and, if the pressure is maintained, conduction block associated with oedema of the nerve. At either of these stages, cessation of pressure will reverse the block, either in a matter of minutes or hours, or over a period of days or weeks, respectively [33, 37].

Demyelination

In acute or chronic compression, Schwann cells underneath the compressing force are damaged leading to loss of the associated myelin segments. This process is referred to as segmental demyelination [38]. At the edges of the cuff in tourniquet compression, mechanical changes corresponding to displacement of soft tissue cause intussusception of internodes leading to paranodal demyelination [39]. If the pressure is high enough and prolonged, degeneration of the nerve distal to the compressing force occurs, producing axonal or Wallerian degeneration [38]. The degree of damage is proportional to the degree and duration of pressure applied [38, 39].

Degeneration

If a nerve is severely damaged, whether by compression, stretch or other means, there is distal degeneration of the axon and, with it, the myelin, over a period of 2–7 days [40]. In crush injuries which spare the endoneurium, the residue of macrophage activity on Schwann cells and other debris forms the so-called bands of Bungner.

Remyelination and regeneration

Remyelination is achieved by Schwann cells wrapping around an axon. Each cell produces a segment of myelin. The segments of myelin, or internodes, formed by this process are shorter than normal internodes [41–43]. Compared with regeneration, remyelination is a faster process and, in general, a more effective one.

In regeneration after lesions such as severe crush or nerve section, several neurofibrils emerge from the normal proximal portion of the nerve to form a growth cone. Eventually one axon predominates and, if there has been no disruption of the endoneurium, it may grow down the endoneurial tube to the end organ, the process being guided by the bands of Bungner. The regenerated nerve is usually smaller in diameter than normal [44, 45].

Regeneration after section is less effective than when continuity of the nerve has been preserved. The gap contains exudate and fibroblasts which interfere with the attempts of regenerating axons to find a way through, and there may be severe malalignment between the opposing nerve ends. It has been estimated that in these conditions only one in seven axon sprouts will reach an end organ [46]. These features are often expressed

 © 2000 Blackwell Science Ltd

clinically in the form of a neuroma at the site of the growth cone.

The rate of regeneration varies from 1 to 4 mm.day^{-1} [47]. It is slower over distal than proximal segments because of the increasing burden on axoplasmic flow as the distance of the growth cone from the cell body becomes greater [44]. Regeneration across scar tissue is about a tenth of this speed [46]. Remyelination of the regenerated axons lags behind by 9–20 days [48–50]. It also proceeds in a proximal to distal direction.

If no regeneration occurs within 1–1.5 years the prognosis is poor because Schwann cells tend to become replaced by fibrous tissue [44]. Regeneration also tends to be less effective as the age of the patient increases.

When there is less severe degeneration of motor nerves, particularly in chronic cases, a process of collateral regeneration frequently coexists. In this, nerve sprouts from a normal motor unit grow across to innervate muscle fibres which have lost their nerve supply. As a result they are incorporated into the donor's motor unit. This has important implications in diagnosis (see below, 'Electromyography and nerve conduction studies').

Classification of nerve injury

The degree to which a nerve is damaged has implicatons with respect to its function and potential recovery. There are essentially two general classification systems. Seddon's classification of nerve injuries describes three groups: neurapraxia, axonotmesis and neurotmesis [48]. Sunderland's classification describes five types of injury and depends exclusively upon which connective tissue components are disrupted [44].

Neurapraxia describes a mild degree of neural insult that results in impulse conduction failure across the affected segment. It is reversible. **Axontomesis** occurs when only the axon is physically disrupted with preservation of the endoneurial and other supporting connective tissue structures. Recovery of function depends upon time for the process of Wallerian degeneration and neural regeneration to occur. **Neurotmesis** is the greatest degree of disruption a nerve can incur and is complete disruption of all supporting connective tissue structures. The nerve is completely severed and there is no continuity and this carries a very poor prognosis for complete functional recovery. Table 1 succinctly compares Seddon's and Sunderland's classifications.

Clinically, the prognosis for spontaneous recovery is good in Sunderland's Type 1 and fairly good in Type 2 injuries. In the remaining groups the prognosis is poor, and surgery will usually be required in Type 4 or 5 nerve injuries.

Electromyography and nerve conduction studies

Electromyography (EMG), which involves the examination of electrical activity in a muscle at rest and during volition, provides valuable clinical information about the state of its nerve supply. Similarly, the recording of propagated action potentials following stimulation of motor or sensory nerves allows conduction velocities to be assessed. Amplitude measurements of action potentials may also permit inferences to be made about the number of nerve fibres participating in conduction.

The majority of peri-operative peripheral nerve lesions cause axonal degeneration. A few, notably chronic compression lesions or acute-on-chronic compression lesions such as carpal tunnel or cubital tunnel syndrome, produce demyelination.

The electrophysiological diagnosis of degeneration is based on the demonstration of reduced numbers of functioning axons. The compound sensory action potential (SAP) represents the summation of individual action potentials from single sensory nerves. The amplitude of the SAP will therefore be reduced in sensory degeneration if the stimulating and/or recording electrodes overlie an affected portion of the nerve. Technical factors limit the application of a similar method to motor nerves. However, on EMG, the number of motor units recruited during maximal voluntary effort (which is equal to the number of active motor nerve fibres) may be gauged from an inspection of the pattern of accompanying motor unit potentials or so-called interference pattern. In motor nerve degeneration, the pattern is less dense or 'reduced'. Spontaneously occurring EMG potentials are not normally present but after severe and acute lesions especially, and after nerve section in particular, denervation potentials such as fibrillation potentials and/or positive sharp waves may be recorded. They are generated by muscle fibres that have lost their nerve supply.

Diagnosis of the site of the lesion then depends on the distribution of the abnormal findings. A similar process applies when clinical signs, rather than electrophysiological abnormalities, are being interpreted.

The degree to which the amplitudes of SAPs and the patterns of EMG activity at maximal voluntary effort are reduced is proportional to the degree of degeneration causing the changes.

A caveat must be introduced here; namely that 10–14 or more days may be required for the process of degeneration to be completed. Prior to this, it is possible to obtain false reassurance if normal SAPs are recorded from nerves which will ultimately degenerate, but have not yet done so. Similarly, EMG may show no signs of denervation potentials during the early stages of motor nerve degeneration.

© 2000 Blackwell Science Ltd

Table 1 A Classification of nerve injuries.

Type				
Seddon	**Sunderland**	**Function**	**Pathological basis**	**Prognosis**
Neurapraxia	Type 1	Focal conduction block	Local myelin injury, primarily larger fibres. Axonal continuity, no Wallerian degeneration.	Recovery in weeks to months
Axontmesis	Type 2	Loss of nerve conduction at injury site and distally	Disruption of axonal continuity with Wallerian degeneration	Axonal regeneration required for recovery. Good prognosis since original end organs reached.
	Type 3	Loss of nerve conduction at injury site and distally.	Loss of axonal continuity and endoneurial tubes. Perineurium and epineurium preserved.	Disruption of endoneurial tubes, haemorrhage and oedema produce scarring. Axonal misdirection. Poor prognosis. Surgery may be required.
	Type 4	Loss of nerve conduction at injury site and distally.	Loss of axonal continuity, endoneurial tubes, and perineurium. Epineurium remains intact.	Total disorganisation of guiding elements. Intraneural scarring and axonal misdirection. Poor prognosis. Surgery necessary.
Neurotmesis	Type 5	Loss of nerve conduction at injury site and distally.	Severance of entire nerve.	Surgical modification of nerve ends required. Prognosis guarded and dependent upon nature of injury and local factors.

In demyelination, there is focal slowing of sensory and/or motor conduction across the affected portion of the nerve in the context of normal conduction over segments of the nerve distal or proximal to the lesion. This is a very valuable finding since it localises the lesion. The degree of slowing is proportional to the degree of demyelination. If the lesion is sufficiently severe it produces focal conduction block, and ultimately will lead to degeneration distally [51, 52].

In some circumstances, electrophysiological changes may be used to monitor recovery. Thus, as a demyelinated nerve remyelinates, so the conduction velocity over the affected segment improves.

Similarly, as a degenerated sensory nerve regenerates, so the amplitude of the SAP increases, but unfortunately this is usually such a late sign as to be of extremely limited clinical value. Even when the course of regeneration is completed, the nerve conduction velocity seldom returns to normal. This is because the restoration of maximal fibre diameter, upon which, inter alia, the maximal conduction velocity depends, is not usually achieved. Also, the segments of myelin now covering the axon are shorter than normal and so the number of nodes is increased. Since nerve depolarisation occurs at the internodes in saltatory conduction, the effect can be a measurable, cumulative delay in conduction time [53–55].

Because the assessment of the EMG pattern at maximal voluntary effort is subjective, a definite improvement can only be identified with confidence when it is very substantial. Loss of denervation potentials can be an encouraging trend, but it may be difficult to be certain that their absence does not, instead, reflect muscle fibre atrophy. After a severe motor nerve lesion, such as section, very small motor unit potentials, sometimes called nascent motor unit potentials, may be seen, as axonal sprouts begin to make contact with muscle fibres. Bitter experience teaches that these apparently encouraging signs should not be over-interpreted; all too frequently the final degree of recovery remains confined to one or two such motor unit potentials with little or no evidence of restoration of muscle power.

The increased size of the motor unit in collateral regeneration leads to an increase in the duration of the motor unit potential [56]. Measurements of motor unit potential durations can be relatively easily performed, and since they imply regeneration and thus prior motor nerve degeneration, they are widely used to aid the diagnosis of the latter.

This type of electrophysiological assessment should only be carried out by a clinical neurophysiologist. The results must be interpreted in the context of the clinical scenario.

Specific forms of nerve injury

Brachial plexus

These are most frequently related to excessive stretching of the cord. In general terms, stretch of the brachial plexus is particularly induced by arm abduction, external rotation and posterior shoulder displacement [4]. Circumstances in which this may occur include extension and lateral flexion of the head to one side with the patient

© 2000 Blackwell Science Ltd

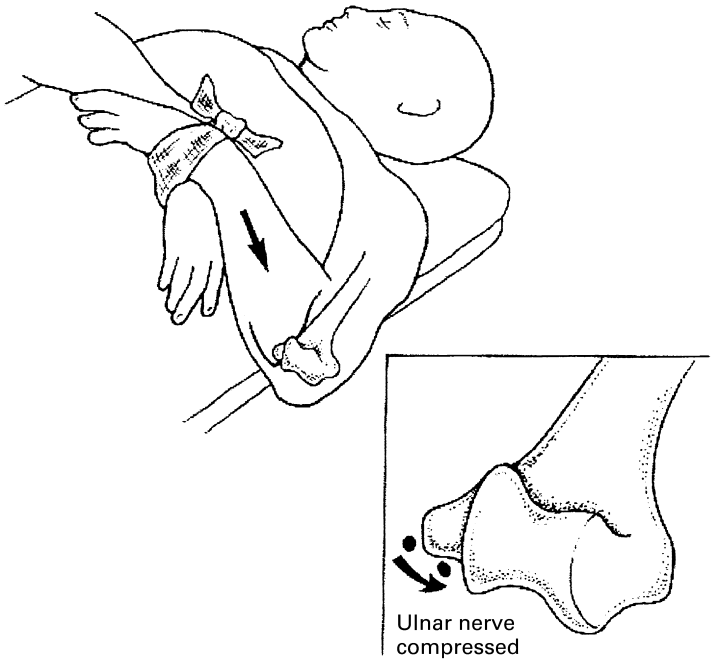

Figure 1. Ulnar nerve compression.

supine, and then abduction, external rotation and extension of the arm by allowing it to drop away from the side of the body. Extreme abduction of the arm so that the hand rests above the head also causes considerable stretch on the brachial plexus roots [3].

Compression of the brachial plexus can also be implicated in brachial plexus injury. This has been described with upward movement of the clavicle secondary to sternal retraction and median sternotomy [17, 19, 21]. Compression also plays a predominant role in injury with the patient in the lateral decubitus position when the plexus is compressed against the thorax by the humeral head. Anatomical variation of the thoracic outlet, in particular the presence of an extra rib on the seventh cervical vertebra, may provide a predisposing cause [57].

Brachial plexus lesions most frequently involve the upper nerve roots with corresponding symptoms and signs affecting that distribution. Lower brachial plexus nerve lesions are also associated with median sternotomy.

Ulnar nerve

Ulnar nerve injury is more common than brachial plexus injury possibly because of its superficial path along the medial epicondyle of the humerus [3].

The ulnar nerve is particularly vulnerable to compression against the operating table, especially if the forearm is extended and pronated (Fig. 1). Injuries may also occur when the nerve is stretched around the medial epicondyle during extreme flexion of the elbow across the chest [5].

There may be certain predisposing factors as suggested by the predominance of this problem in males (5 : 1), where the cubital tunnel may be narrower, or the ulnar nerve may be unusually mobile [6, 18, 58]. Subclinical lesions may be compounded during surgery as suggested by the finding of abnormal ulnar nerve conduction across the elbow in the unaffected arm [19].

Intra-operative ulnar nerve compression can result in lesions of quite remarkable severity and recovery can be slow and often incomplete. Nevertheless, it seems quite likely that postoperative ulnar nerve palsy can occur without apparent cause and despite accepted methods of positioning and padding [12].

Radial nerve

This lesion usually occurs as a result of compression of the nerve between the edge of the operating table and the humerus. It is also the classical 'Saturday night palsy'. It may occur when the patient is in the lateral position and the uppermost arm is abducted beyond 90° and suspended from a vertical screen support [11].

Sciatic nerve

The sciatic nerve is especially at risk if the patient is thin, the table hard, the operation long and when the opposite buttock is elevated as in the hip pinning position. In the lithotomy position maximal external rotation of the flexed thigh may damage the nerve by stretch [59].

© 2000 Blackwell Science Ltd

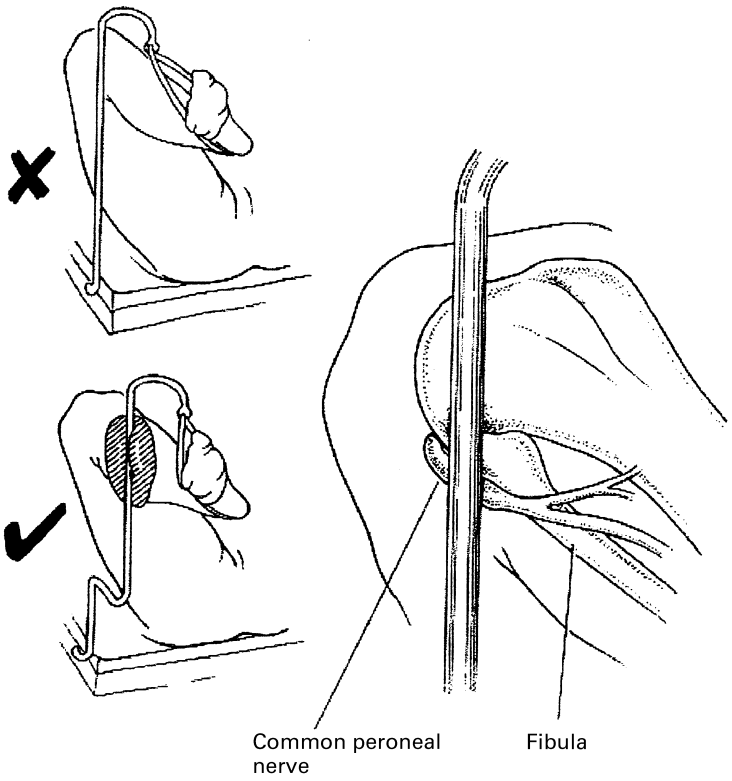

Figure 2. Common peroneal nerve compression.

Sciatic nerve palsies have also been reported following coronary artery bypass graft (CABG) surgery, probably as a result of prolonged nerve pressure compounded by the lowered arterial perfusion pressure provided by cardiopulmonary bypass [58].

Common peroneal nerve

This is the most frequently damaged nerve in the lower limb. It may be compressed against the head of the fibula in the lithotomy position or between the fibula and the operating table, a particular risk associated with the lateral position [60] (Fig. 2).

Less common nerve injuries

These include compression of the following nerves: tibial nerve in the popliteal fossa, saphenous nerve between the medial condyle of the tibia and a lithotomy pole, the supraorbital nerve by a tracheal tube or a tight head harness [61]; and of the facial nerve [62], which may be compressed against the ascending ramus of the mandible whilst the anaesthetist holds the jaw forward to maintain an airway.

Clinical features of nerve injuries

Nerve damage may be apparent immediately after recovery from anaesthesia or may occur only several days later.

Clinical features include anaesthesia, paraesthesia, hypaesthesia, hyperaesthesia, and pain in the areas supplied by the affected nerves, and there may be paresis or even paralysis of affected muscles. Sometimes, disabling autonomic dysfunction occurs. Ultimately, in extreme cases, there is muscle wasting, joint stiffening and demineralisation of bone (see Table 2)

Prevention of injuries to peripheral nerves in an anaesthetised patient

Prevention of injuries requires an awareness of the potential dangers of the various surgical positions utilised. Careful positioning of every patient on the operating table with proper padding will reduce, but not eliminate, injuries to peripheral nerves. In positions in which strain or pressure on the neurovascular system is possible, the pressure should be alleviated with padding such that the risk of nerve injury is reduced.

In preventing brachial plexus lesions, abduction of the arm should preferably be limited to 90° or less. Even with the use of lockable armboards, plexus injury has been reported with the arm abducted to as little as 60° [63]. If 90° abduction is necessary, the elbow should not be fully extended. External rotation of the abducted arm, and especially posterior displacement of the shoulder, should be avoided because this

© 2000 Blackwell Science Ltd

Table 2 Simplified clinical identification of major peripheral nerve injuries.

(I) Arm	
Median Nerve	Numbness over the index finger. Weakness of abduction of the thumb.
Ulnar Nerve	Numbness over the little finger. Weakness of abduction and/or adduction of the fingers. Also, weakness of flexion at the distal interphalangeal joints of the little and ring fingers if the lesion is at the elbow.
Radial Nerve	Weakness of extension at the distal interphalangeal joint of the thumb, and of the wrist and finger extensors.
Musculocutaneous Nerve	Weakness of flexion of the elbow.
Circumflex Nerve	Weakness of abduction of the shoulder.
Brachial Plexus	Various combinations of lesions within the median, ulnar, radial, musculocutaneous, and circumflex nerve territories.
(II) Leg	
Femoral Nerve	Weakness of flexion of the hip. Numbness over the thigh
Obturator Nerve	Weakness of adduction of the hip.
Sciatic Nerve	Weakness of ankle dorsiflexion and plantar flexion. Also, weakness of knee flexion, if the lesion is proximal. Numbness below the knee.
Common Peroneal Nerve	Weakness of dorsiflexion of the ankle and toes
Tibial Nerve:	Weakness of plantarflexion of the ankle and toes.

can stretch and compress the plexus. Rotation and lateral flexion of the neck towards the opposite side should also be avoided because this increases the tension on the brachial plexus (Fig. 3). Central position of the head is ideal.

The severity and permanence of many postoperative lesions of the ulnar nerve make it imperative that every precaution be taken to prevent their occurrence. Positioning the forearm and hand in pronation places the ulnar nerve at the elbow at risk. Placing the arm in supination and avoiding elbow flexion will free the cubital tunnel [64]. Extremes of elbow flexion should be avoided because this too may cause compression of the ulnar nerve by the medial and arcuate ligaments leading to a neurological deficit. Finally, it seems possible that the ulnar nerve could receive a severe blow from the end of the suspension pole passed up the lateral sleeve of the canvas sheet during patient transfer from operating table to bed (Fig. 4).

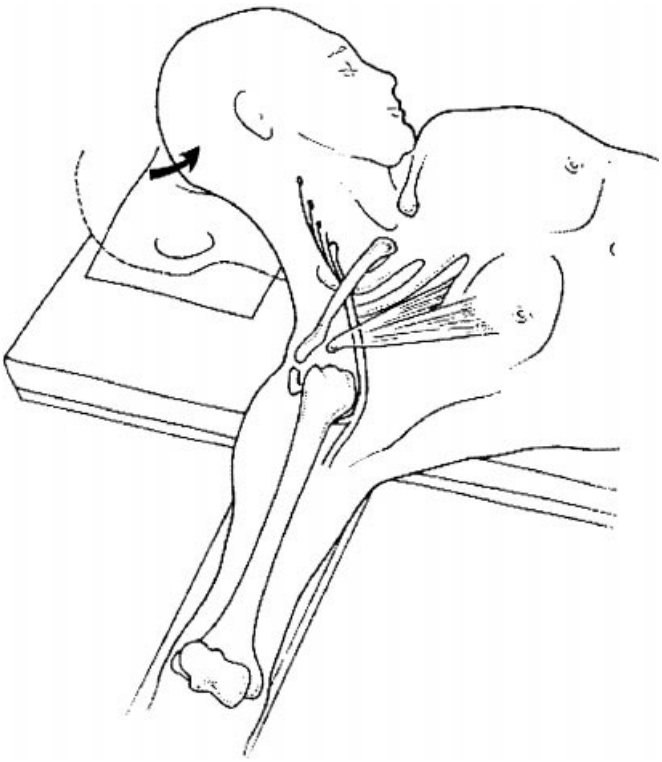

Figure 3. Head rotation causing excessive traction on the brachial plexus.

© 2000 Blackwell Science Ltd

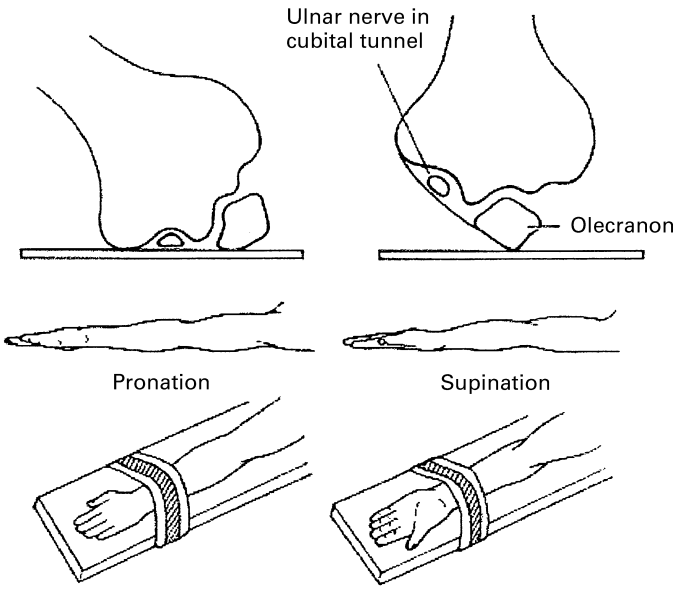

Figure 4. Ulnar nerve compression within the cubital tunnel.

Since nerve damage to the upper limb can be caused by even minimal compression, the safest position is for the arm to be wrapped in padding down by the patient's side [65]. If this is not possible, maximal padding in the desired position is indicated, with avoidance of other predisposing factors, such as hypothermia and dehydration. This does, however, present a practical problem in having access to an intravenous cannula sited in the hand or arm. We would suggest a free-flowing infusion with a three-way tap sited in the intravenous line to allow rapid administration of intravenous drugs and regular inspection of the cannula site to exclude fluid extravasation.

Adequate padding is also required with the patient in the lithotomy position when the saphenous, sciatic and peroneal nerves are vulnerable [5] (Fig. 5).

If the patient is in the prone position, the arm should ideally be placed down by the patient's side. If the arms are placed above the patient's head, this can cause a stretch injury to the lower trunks of the brachial plexus. If this is unavoidable, the arm should be placed on an arm board, with the arm abducted to less than 90° with the elbows flexed and the palms facing downwards.

Since the commonest injury in the lateral position is to the peroneal nerve, as a result of compression of the lower leg on the table mattress, the nerve must be protected with padding [66]. If the arm is to be suspended, then limitation of arm abduction to 90° is essential. The dependent arm should be anterior to the thorax to avoid compression of the brachial plexus on that side [63]. In the lateral position the head must be properly supported so that the cervical and thoracic portions of the vertebral column are kept in the same horizontal position [63]. Again, this avoids any unnecessary stretch of the brachial plexus.

A sound knowledge of nerve anatomy, a cautious injection technique during the block procedure and the judicious use of a nerve stimulator to elicit paraesthesia will also aid in reducing the incidence of nerve injuries.

Culpability and medicolegal implications

For nearly a century the literature has contained reports of damage to the peripheral nerves of patients undergoing general anaesthesia. Although these incidents are, potentially, anaesthesia-related injuries, the anaesthetist has not always been to blame, and although purported aetiological factors have been well described, these injuries continue to result in litigation for pain, suffering and economic consequences [65, 67]. In some cases there has been a pre-existing lesion or a general increase in susceptibility to damage, and nerve injury occurs despite conventionally accepted methods of positioning or padding. In other cases, the damage has been sustained after the patient has left the operating theatre [6].

The ASA Closed Claims Study implies that factors other than standards of care are involved in many cases. Thus, payment was made in 58% of claims for ulnar nerve damage in which care was considered to be adequate [6].

From unpublished data of the Medical Protection Society, it appears that although claims arising from peripheral nerve palsy associated with anaesthesia are rare, when they do occur and the patient suffers permanent and severe neurological deficit, the costs of settling these claims are now well above average anaesthetic claims.

The average current cost of each settled claim involving peripheral nerve injury is just under £4000 excluding costs, and the highest settled claim is £49 000 for an ulnar

 © 2000 Blackwell Science Ltd

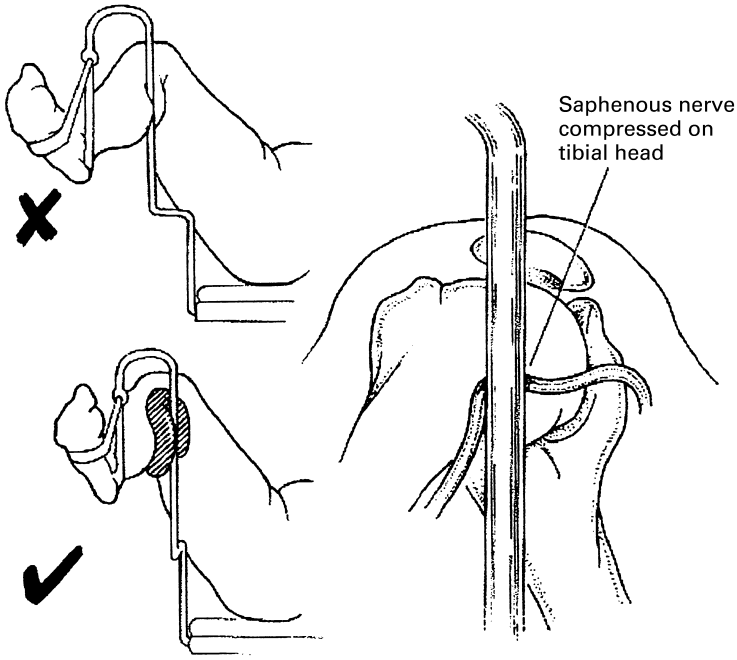

Figure 5. Saphenous nerve compression.

nerve injury (Medical Protection Society, personal communication). The highest unsettled and ongoing claim is for £120 000 for a common peroneal nerve palsy, although special circumstances apply in this claim which relate to loss of earnings. The same data revealed that proceedings were discontinued in 50% of cases and the cases closed. Many claims are not pursued by the claimant in the face of a steadfast defence.

In defending substantial claims, detailed investigations should be undertaken to establish the precise cause of the lesion. This would exclude cases in which 'intra-operative' nerve lesions are caused by a coexisting carpal tunnel syndrome or neurological disease.

The apportionment of liability between anaesthetist, surgeon and theatre staff is not usually an issue insofar as ulnar nerve palsy is concerned because the anaesthetist is most often considered to be liable. The uncertain mechanism of ulnar nerve injury implies that the anaesthetist must have done something wrong if the injury occurred in temporal proximity to anaesthetic care [64]. Responsibility for nerve injuries affecting the lower limbs is difficult to prove. Surgeons, nursing staff and operating department assistants are all involved in manoeuvring the patient into position and pressure or stretching nerve damage can occur at any moment. The apportionment of blame would be a very contentious issue in terms of liability and any subsequent disciplinary procedures. In a claim involving a common peroneal nerve injury, joint responsibility was accepted by surgeon, anaesthetist and theatre staff (MPS, personal communication). However, the onus of responsibility tends to default to the anaesthetist.

Very often in these claims the principle of *res ipsa loquitur* is pleaded by the plaintiff. This legal principle shifts the burden of proof from the plaintiff to the defendant (the reversal of which is normally the case) once certain facts are established:

1 the episode which caused the damage must be under the control of the defendant;

2 the episode would not have ordinarily occurred had proper care been applied.

If the defendant is able to demonstrate that, because of a predisposition, the patient could have suffered injury in the normal course of events, or if the episode was related to a coincidental disease process, then the principle of *res ipsa loquitur* is inapplicable. This supports the need to protect peripheral nerves whilst the patient is anaesthetised and accurately document such care. This will assist in refuting an allegation of *res ipsa loquitur.*

As with all anaesthetic procedures, meticulous attention to detail is a prerequisite, and an accurately completed anaesthetic chart documenting the peri-operative care is of the utmost importance. While these principles should merely be a reflection of good practice rather than defensive medicine, it is very difficult to defend a claim involving peripheral nerve injury in their absence.

Conclusion

Peripheral nerve lesions are a complication of operative procedures under both general and regional anaesthesia. Although they account for a small proportion of medicolegal claims, they are difficult to defend, being

© 2000 Blackwell Science Ltd

essentially avoidable. Avoidance involves awareness of the problems associated with operative positions and careful positioning of the patient with appropriate padding. This should form part of standard anaesthetic care.

Once damage to nerves has occurred it can take several forms, ranging from a mild, reversible neurapraxia to a permanent sensorimotor deficit. The neurophysiological investigations can often provide useful diagnostic and prognostic information, and the assistance of a trained neurophysiologist should be sought in these cases. A carefully completed anaesthetic record documenting limb position and appropriate protective measures should be standard for all cases.

Acknowledgment

We thank Mr P. Elliot, Medical Artist, of the Medical Illustration Services, Central Sheffield University Hospitals for his assistance in preparing the anatomical line drawings.

References

1 Slocum HC, O'Neal KC, Allan CR. Neurovascular complications from malposition on the operating table. *Surgery, Gynecology & Obstetrics* 1948; **86**: 729–34.

2 Budinger K. Veber lahmungen nach chloroform – narkosen. *Archive Fur Klinische Chirurgie* 1894; **47**: 121–45.

3 Britt BA, Gordon RA. Peripheral nerve injuries associated with anaesthesia. *Canadian Anaesthetists' Society Journal* 1964; **11**: 514–36.

4 Dhuner KG. Nerve injuries following operations: Survey of cases occurring during a 6 year period. *Anesthesiology* 1950; **11**: 289–93.

5 Alvine FG, Schurrer ME. Post operative ulnar nerve palsy. Are there predisposing factors? *Journal of Bone and Joint Surgery* 1987; **69**: 255–9.

6 Kroll DA, Caplan RA, Posner K, Ward RJ, Cheney FW. Nerve injury associated with anaesthesia. *Anesthesiology* 1990; **73**: 202–7.

7 Denny-Brown D, Doherty MM. Effects of transient stretching of peripheral nerves. *Archives of Neurology and Psychiatry* 1945; **54**: 116–29.

8 Clausen EG. Post operative ('anesthetic') paralysis of the brachial plexus; review of literature and report of 9 cases. *Surgery* 1942; **12**: 933–42.

9 Bonner SM, Pridie AK. Sciatic nerve palsy following uneventful sciatic nerve block. *Anaesthesia* 1997; **52**: 1205–7.

10 Gilliatt RW, Harrison MJG. Nerve compression and entrapment. In: Asbury AK, Gilliatt RW, eds. *Peripheral Nerve Disorders, a Practical Approach*. London: Butterworth, 1984: 245.

11 Nicholson MJ, Eversole UH. Nerve injuries incident to anaesthesia and operations. *Anesthesia and Analgesia* 1957; **36** (4): 19–32.

12 Earl CJ, Fullerton PM, Wakefield GS, Schutta HS. Hereditary neuropathy with liability to pressure palsies; a clinical and electrophysiological study of four families. *Quarterley Journal of Medicine* 1964; **33**: 481–98.

13 Stoelting RK. Post operative ulnar nerve palsy – Is it a preventable complication? *Anaesthesia and Analgesia* 1993; **76**: 7–9.

14 Garriques HJ. Anaesthesia paralysis. *American Journal of Medical Science* 1897; **113**: 81–9.

15 Bartholomew LG, Scholz DA. Reversible post operative neurological symptoms; report of 5 cases secondary to water intoxication and sodium depletion. *Journal of the American Medical Association* 1956; **162**: 22–6.

16 Delorme EJ. Hypothermia. *Anaesthesia* 1956; **11**: 221–31.

17 Swan H, Virtue RW, Blount SG, Kircher LT. Hypothermia in surgery: analysis of 100 clinical cases. *Annals of Surgery* 1953; **142**: 382–400.

18 Dawson DM, Krarup C. Peri-operative lesions. *Archives of Neurology* 1989; **46**: 1355–60.

19 Casscells CD, Lindsey RW, Ebersole J, Li B. Ulnar neuropathy after median stenotomy. *Clinical Orthopaedics & Related Research* 1993; **291**: 259–65.

20 Vander Salm TJ, Cereda JM, Cutler BS. Brachial plexus injury following median sternotomy. *Journal of Thoracic Cardiovascular Surgery* 1980; **80**: 447–52.

21 Seyfer AE, Grammer NY, Bogumill GP, Provost JM, Chaudry U. Upper extremity neuropathies after cardiac surgery. *Journal of Hand Surgery* 1985; **10**: 16–19.

22 Watson BV, Merchant RN, Brown WF. Early post operative ulnar neuropathies following coronary artery bypass surgery. *Muscle & Nerve* 1992; **15**: 701–5.

23 Merchant RN, Brown WF, Watson BV. Peripheral nerve injuries in cardiac anaesthesia. *Canadian Journal of Anaesthesia* 1990; **37**: S152.

24 Edwards BN, Tullos HS, Noble PC. Contributory factors and aetiology of sciatic nerve palsy in total hip arthroplasty. *Clinical Orthopaedics* 1987; **218**: 136–41.

25 Rose HA, Hood RW, Otis JC, Ranawat CS, Insall JN. Peroneal nerve palsy following total knee arthroplasty. A review of The Hospital for Special Surgery Experience. *Journal of Bone and Joint Surgery of America* 1982; **64**: 347–51.

26 Weikel AM, Habel MB. Meralgia parasthetica; a complication of iliac bone procurement. *Plastic and Reconstructive Surgery* 1977; **60**: 572–4.

27 Keykhah HM, Rosenberg H. Bilateral footdrop after craniotomy in the sitting position. *Anesthesiology* 1979; **51**: 163–4.

28 Cucchiara RF, Nugent M, Seward JB, Messick JM. Air embolism in upright neurosurgical patients: detection and localisation by two-dimensional echocardiography. *Anesthesiology* 1984; **60**: 353–5.

29 Chambers WA. Editorial 1: Peripheral nerve damage and regional anaesthesia. *British Journal of Anaesthesia* 1992; **69**: 429–30.

 © 2000 Blackwell Science Ltd

30 Winnie AP. An 'immobile needle' for nerve blocks. *Anesthesiology* 1969; **31**: 577–8.
31 Rice AS, McMahon SB. Peripheral nerve injury caused by injection needles used in regional anaesthesia: influence of bevel configuration, studied in a rat model. *British Journal of Anaesthesia* 1992; **69**: 433–8.
32 Selander D, Brattsand R, Lundborg G, Nordborg C, Olsson Y. Local anaesthetics: importance of mode of application, concentration and adrenaline for the appearance of nerve lesions. An experimental study of axonal degeration and barrier damage after intrafascicular injection or topical application of bupivicaine (Marcain). *Acta Anaesthesiologica Scandinavica* 1979; **23**: 127–36.
33 Eckhoff NL. Tourniquet paralysis; plea for extended use of pneumatic tourniquet. *Lancet* 1931; **2**: 343–5.
34 Dumitru D. *Electrodiagnostic Medicine*. Philadelphia: Hanley & Belfus Inc., 1994.
35 Ahmad NS, Yentis SM. Laryngeal mask airway and lingual nerve injury. *Anaesthesia* 1996; **51**: 707–8.
36 Laxton CH, Kipling R. Lingual nerve paralysis following the use of the laryngeal mask airway. *Anaesthesia* 1996; **51**: 869–70.
37 Lundborg G. *Nerve Injury and Repair.* Edinburgh: Churchill Livingstone, 1988.
38 Sunderland S. *Nerve Injuries and Their Repair.* Edinburgh: Churchill Livingstone, 1991.
39 Denny-Brown D, Brenner C. Paralysis of nerve induced by direct pressure and by tourniquet. *Archives of Neurology and Psychiatry* 1944; **51**: 1–26.
40 Ochoa J, Fowler TJ, Gilliatt RW. Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. *Journal of Anatomy* 1972; **113**: 433–55.
41 Hiscoe HB. Distribution of node and incisures in normal and degenerated nerve fibres. *Anatomical Record* 1947; **99**: 447–75.
42 Janz D, Neundorfer B. Klinische und electromygraphische untersuchungen nach triarylphosphat-polyneuropathie. *Deutsche Zeitschrift Fur Nervenheilkunde* 1962; **183**: 268–304.
43 Lee JC. Electron microscopy of Wallerian degeneration. *Journal of Comparative Neurology* 1963; **120**: 65–79.
44 Trojaborg W. Early electrophysiological changes in conduction block. *Muscle and Nerve* 1978; **1**: 400–3.
45 Pollock M. Nerve regeneration. *Current Opinion in Neurology* 1995; **8**: 354–8.
46 Sunderland S. *Nerves and Nerve Injuries*, 2nd edn. Edinburgh: Churchill Livingstone, 1978.
47 Ramon Y, Cajal S. *Degeneration and Regeneration of the Nervous System*. New York: Hafner, 1959.
48 Dustin AP. Les lesions posttraumatiques des nerfs. *Ambulance de L'ocean* 1917; **1**: 71–162.
49 Hentowa F. Regeneration der peripherischen nerven. *Z Ges Neurol Psychiat* 1933; **147**: 791–806.
50 Quilliam TA. Growth changes in sensory nerve fiber aggregates undergoing remyelination. *Journal of Anatomy* 1958; **92**: 383–98.
51 Mayer RF, Denny-Brown D. Conduction velocity in peripheral nerve during experimental demyelination in the cat. *Neurology* 1964; **14**: 714–26.
52 Seddon H. Three types of nerve injury. *Brain* 1943; **66**: 237–88.
53 Brill MH, Waxman SG, Moore JW, Joyner RW. Conduction velocity and spike configuration in myelinated fibers: computed dependence on internode distance. *Journal of Neurology, Neurosurgery and Psychiatry* 1977; **40**: 769–74.
54 Fowler TJ, Danta G, Gilliatt RW. Recovery of nerve conduction after a pneumatic tourniquet: observations on the hind limb of the baboon. *Journal of Neurology, Neurosurgery and Psychiatry* 1972; **35**: 638–47.
55 Sanders FK, Whitteridge D. Conduction velocity and myelin thickness in regenerating nerve fibres. *Journal of Physiology* 1946; **105**: 152–74.
56 Erminio F, Buchthal F, Rosenfalck P. Motor unit territory and muscle fibre concentration in paresis due to peripheral nerve injury and anterior horn cell involvement. *Neurology* 1959; **9**: 657–71.
57 Gilliatt RW, Harrison MJG. Nerve compression and entrapment. In: Asbury AK, Gilliant RW, eds. *Peripheral Nerve Disorders: a Practical Approach*. London: Butterworth, 1984: 245–55.
58 Cameron MGP, Stewart OJ. Ulnar nerve injury associated with anaesthesia. *Canadian Anaesthetists' Society Journal* 1975; **22**: 253–64.
59 Buckhart FL, Daly JW. Sciatic and peroneal nerve injury; a complication of vaginal operations. *Obstetrics and Gynecology* 1966; **28**: 99–102.
60 Garland H, Moorhouse D. Compressive lesions of the external popliteal (common peroneal) nerve. *British Medical Journal* 1952; **2**: 1373–8.
61 Barron DW. Supraorbital neuropraxia. *Anaesthesia* 1955; **10**: 374.
62 Fuller JE, Thomas DV. Facial nerve paralysis after general anaesthesia. *Journal of the American Medical Association*. 1956; **162**: 645.
63 Raffan AW. Postoperative paralysis of the brachial plexus. *British Medical Journal* 1950; **2**: 149.
64 Perreault L, Drolet P, Farny J. Ulnar nerve palsy at the elbow after general anaesthesia. *Canadian Journal of Anaesthesia* 1992; **39**: 499–503.
65 Britt BA, Joy N, McKay MD. In: Orkins FK, Cooperman LH, eds. *Positioning Trauma, Complications in Anaesthesiology.* Philadelphia: JB Lippincott, 1983: 646–70.
66 Sullivan D. Complications of intra operative positioning. *Orthopaedic Nursing* 1985; **4**: 56–9.
67 Dornette WHL. Compression neuropathies. Medical aspects and legal implications. *International Anaesthesiology Clinics* 1986; **24**: 201–968.

© 2000 Blackwell Science Ltd