Anatomical deliberation for prevention strategy of Peri-Operative nerve injuries

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Abstract
Nerve injuries can happen whenever amid the peri-operative period. The long haul handicap that outcomes may have genuine results for a patient. The frequency of peri-operative nerve wounds can be lessened by anesthetists monitoring their causes and pathophysiology. This article means to clarify the anatomical thought, occurrence and pathophysiology of perioperative nerve damage and gives recommendations with reference to how they may best be maintained a strategic distance from.

Keywords: insentience, drawback, neurological, Nerve, adulteration

Introduction
Peri-operative nerve lesions can spoil a successful surgical procedure, handicap a patient with a severe functional disability and leave the medical team facing possible protracted and unpleasant litigation. 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 anesthesia have been observed since shortly after the introduction of ether [1]. Early writers blamed toxic properties of the anesthetic agent, but Budinger [2] was the first to recognize that the majority were secondary to 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 anesthesia, 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).

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, mechanical factors such as compression, stretch [3, 7], angulations, percussion or transection. Ischemia is a crucial element in many of these injuries [8, 9].

Predisposing factors
Nerves may be unduly susceptible to trauma as a result of a pre-existing generalized 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 exaggerated lithotomy position for radical prostate surgery 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. 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 hyper abduction 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 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 clinically in the form of a neuroma at the site of the growth cone. The rate of regeneration varies from 1 to 4 mm.day21 [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.

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 fibers 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 fibers 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.

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 localizes the lesion. The degree of slowing is proportional to the degree of demyelization. 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 fiber 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 fibers. 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 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. 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 908 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]. 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].

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

Prevention of injuries to peripheral nerves in an anaesthetized patient
Prevention of injuries requires an awareness of the potential dangers of the various surgical positions utilized. 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 908 or less.

Even with the use of lockable arm boards, plexus injury has been reported with the arm abducted to as little as 608 [63]. If 908 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 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. 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.

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]. 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 908 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 908 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.

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 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.

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