Perioperative peripheral nerve injuries

Abdul Ghaaliq Lalkhen FRCA FFPMRCA DPMed (CARCSI)
Kailash Bhatia FRCA DA DNB

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Key points

- Perioperative peripheral nerve injury (PPNI) causes significant patient morbidity and is a leading cause of anaesthesia-related litigation.
- Mechanisms of injury include compression, stretch ischaemia, direct nerve trauma, and local anaesthetic toxicity.
- Hypertension, smoking, and diabetes mellitus are associated with PPNIs and may explain why some patients develop nerve injury, despite adequate precautions.
- Clinical examination, electromyography, nerve conduction studies, and magnetic resonance imaging enable the nature of the injury to be defined.
- Knowledge regarding the anatomical vulnerability of peripheral nerves enables empiric precautions to be taken to prevent injury in the absence of robust evidence-based recommendations.

Abdul Ghaaliq Lalkhen FRCA FFPMRCA DPMed (CARCSI)
Consultant in Anaesthesia and Pain Medicine
The Manchester and Salford Pain Centre
Salford Royal NHS Foundation Trust
Stott Lane
Eccles
Salford M6 8HD
UK
Tel: +44 161 2064103
Fax: +44 161 2061929
E-mail: abdul.lalkhen@srft.nhs.uk
(for correspondence)

Kailash Bhatia FRCA DA DNB
Regional Anaesthesia and Pain Medicine Fellow
Salford Royal NHS Foundation Trust
Salford
UK

Perioperative peripheral nerve injuries (PPNIs) complicate both general and regional anaesthesia. Two ASA closed-claim analyses conducted a decade apart reveal that anaesthesia-related nerve injury (ulnar nerve 28%, brachial plexus 20%, lumbosacral nerve roots 16%, and spinal cord 13%) constituted 15% and 16% of total claims, respectively, making it the third most common cause of anaesthesia-related litigation.[1] A PPNI potentially results in significant morbidity for the patient and considerable distress to the medical team facing potentially protracted litigation. Peripheral nerves may be injured as a direct result of the anaesthetic technique in the case of regional anaesthesia.[2] However, diseases affecting the microvasculature of nerves, poor perioperative positioning and padding (considered the responsibility of the anaesthetist in most claims of negligence), tourniquets, and the nature of surgery may also contribute to or compound PPNI.[3] The aetiology of PPNI is further complicated by the fact that prolonged hospital stay has been associated with peripheral nerve injury. The clinical manifestations of PPNI may only become apparent 48 h post-surgery which may cast doubt on the cause of the injury.

Incidence

The exact incidence of PPNI is difficult to define because of the heterogeneity and quality of studies. A retrospective review of general surgical patients excluding those who had a neuraxial or peripheral nerve block quotes an incidence of 0.14%. An incidence of 0.11% was found in a study that did not exclude these patients. A retrospective review considering a broad surgical population quotes an incidence of PPNI of 0.03% (112 patients out of 380 680).[3] The incidence of ulnar neuropathy has been quoted as 0.037% and lower extremity neuropathy in the lithotomy position as between 0.028% and 1.5%. Confounding factors include the definition of a PPNI (permanent *vs* transient and time of onset in relation to surgery), the nature of the surgery, patient factors, anaesthetic technique, and the fact that retrospective reviews suffer from under-reporting while medicolegal databases may overestimate the incidence of PPNI due to over-reporting.

Permanent neurological injury after a peripheral nerve block regardless of the technique (ultrasound-guided or traditional techniques) is rare.[4] Ultrasound-guided techniques reduce the risk of intravascular injection but do not reduce the incidence of neurological injury. Transient paraesthesia may occur in up to 15% of patients after a peripheral nerve block with 99% resolving within 1 yr. In a prospective study, permanent neurological injury was found to have an incidence of 2.4 in 10 000 peripheral nerve blocks. The question, however, in cases where PPNI occurs when a nerve block has been performed is whether the anaesthetic intervention is the causative factor in the injury.[5]

Aetiology and predisposing factors

Peripheral nerves consist of the axons of neurones that reside in the central nervous system. Individual axons have a myelin sheath and Schwann cells that are surrounded by the endoneurium, which forms an unbroken tube around the axon from its origin in the spinal cord to the point where it synapses. A number of axons (nerve fibres) are organized into fascicles surrounded by a connective tissue layer, the perineurium. The number of fascicles increases and their diameter decreases from proximal to distal with an increase in stromal tissue more distally. The perineurium is itself surrounded by the epineurium. The endoneurium acts as a selective barrier and produces endoneurial fluid (similar to cerebrospinal fluid) that surrounds the axon. A peripheral nerve has an extrinsic plexus of vessels in the epineurial space that crosses the perineurium to anastomose with the intrinsic circulation of the endoneurium (Fig. 1).

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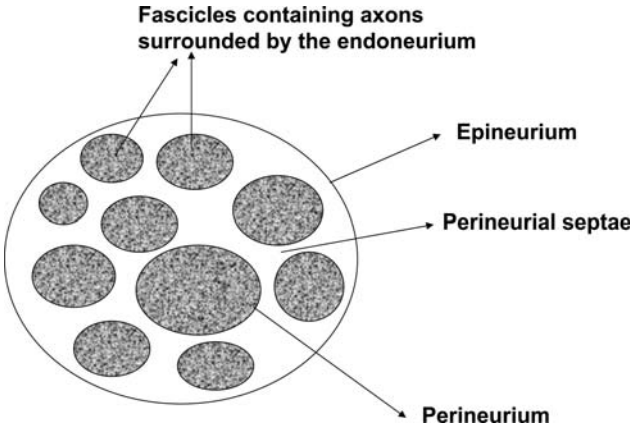

Fig 1 Diagrammatic representation of a peripheral nerve.

The mechanisms that could lead to PPNI are:

(i) Direct nerve damage—from surgery, needle trauma secondary to a regional anaesthetic technique or a peripheral nerve catheter. Blunt needles are less likely to penetrate the tougher perineurium surrounding more widely dispersed fascicles. This anatomical arrangement which occurs in distal nerves may account for the fact that neural injury does not always result from epineurial injection.
(ii) Stretch and compression—poor padding and positioning of limbs, the use of tourniquets, and surgical retractors.
(iii) Ischaemia—this is often the final common pathway of nerve injury and may be caused primarily by tourniquets, prolonged immobility, haematoma surrounding a nerve, and local anaesthetic agents.
(iv) Toxicity of injected solutions—local anaesthetics can produce cytotoxic axonal damage particularly if the solution is injected intrafascicularly. Highly concentrated solutions and prolonged exposure predispose to nerve injury.
(v) Double crush syndrome—a compressive lesion occurring along a nerve renders the nerve less tolerant of compression at the same or a second locus. Hence, nerves with a pre-existing injury or compression (e.g. patients with diabetes mellitus or patients suffering with rheumatoid arthritis with unstable joints) are at greater risk of a second, possibly subclinical insult resulting in a permanent nerve injury.
(vi) Unknown factors
(vii) A combination of the above factors

Predisposing factors can be classified as follows:

Surgical factors: neurosurgery, cardiac surgery, gastrointestinal surgery, and orthopaedic surgery are associated with a higher incidence of PPNI.

Patient factors: hypertension, diabetes mellitus, and smoking cause microvascular changes that may render these patients more susceptible to PPNI. The 'double crush' syndrome through alteration in neuronal homeostasis may also play a role in these patients. Pre-existing peripheral neuropathies may predispose to PPNI. Anatomical abnormalities especially in the thoracic outlet and at the elbow may also predispose patients to PPNI.

Anaesthetic factors: general and epidural anaesthesia are associated with a higher incidence of PPNI compared with monitored anaesthetic care in sedated patients where the patient was able to change their position intraoperatively.

Perioperative factors: hypovolaemia, dehydration, hypotension, hypoxia, electrolyte disturbances, and hypothermia have been implicated in the development of PPNI.

Table 1 Classification of peripheral nerve injuries

Seddon	Sunderland	Pathophysiology
Neuropraxia (compression)	Type 1	Local myelin damage with the nerve still intact
Axonotmesis (crush)	Type 2	The continuity of axons is lost. The endoneurium, perineurium, and epineurium remain intact. Loss of continuity of axons with Wallerian degeneration due to disruption of axoplasmic flow
	Type 3	Type 2 with endoneurial injury
	Type 4	Type 2 with endoneurial and perineurial injury but an intact epineurium
Neurotmesis (transection)	Type 5	Complete physiological disruption of the entire nerve trunk. Early surgical intervention necessary. Prognosis guarded

Classification of peripheral nerve injuries

The most widely accepted classification of nerve injuries are those described by Seddon[6] (neuropraxia, axonotmesis, and neurotmesis) and by Sunderland[7] (Grade 1–5 nerve injuries). The pathophysiological features are described in Table 1.

Clinical presentation of peripheral nerve injuries

A PPNI may lead to sensory (anaesthesia, paraesthesia, hypoaesthesia, hyperaesthesia, and pain in the areas supplied by the affected nerves) and/or motor deficit (paresis or even paralysis of the affected muscles). Autonomic dysfunction and trophic changes may occur leading to severe neuropathic pain. The evidence suggests that upper limb nerve injuries occur more commonly than lower limb injuries (60/40). The symptoms and signs depend on the particular nerve injured.

Upper limb peripheral nerve injuries

Ulnar nerve injury (C7, C8–T1)

Incidence = 0.037%.

Predisposing factors

The superficial nature and close proximity of the ulnar nerve to the medial condyle make this the most common perioperative nerve injury. It is three times more common in men, suggesting that

anatomical predisposition could play a role in this nerve injury (men have a larger tubercle and less adipose tissue protecting the nerve). Often patients have a subclinical neuropathy, which becomes exaggerated in the postoperative period. This has been demonstrated by finding abnormal nerve conduction studies (NCSs) in the contralateral arm. The median age is 50 yr and the symptoms may be delayed by up to 28 days after surgery.

Mechanism of injury

Direct pressure on the ulnar groove in the elbow and prolonged forearm flexion are cited as the most common causes of injury. In some cases, the injury can occur, despite the elbows being padded perhaps as a result of a subclinical injury to an already compromised nerve.

Clinical presentation

It is usually characterized by tingling or numbness along the little finger and weakness of abduction, adduction of the fingers, or both. Examination of the hand reveals hyperextension of the metacarpo-phalangeal joints and flexion at the distal and the proximal interphalangeal joints of the ring and the little finger (ulnar claw).

Brachial plexus injury (C5–T1)

Incidence = 0.2–0.6%.

Predisposing factors

The superficial nature of the plexus, running between two fixed points (intervertebral foramen and the axillary sheath), and its course through a limited space between the clavicle and first rib and proximity to a number of mobile bony structures make it susceptible to injury.

Mechanism of injury

This results from compression, stretching, or a direct injury as a result of a regional technique. Compression against the clavicle may occur during retraction of a median sternotomy or in the lateral decubitus position with compression against the thorax and humeral head. Arm abduction, external rotation with posterior shoulder displacement, causes considerable stretch on the upper brachial plexus roots. The same effect is seen when there is extreme abduction of the arm (>90°).

Clinical presentation

If the C5–6 nerve roots are affected in a high lesion leading to involvement of the musculocutaneous, axillary, and suprascapular nerves, the arm hangs by the side, medially rotated, and pronated described as the 'Waiter's Tip' position. C8–T1 lesions mainly affect the small muscles of the hand, resulting in a claw hand and numbness in the ulnar distribution.

Radial nerve injury (C5–T1)

Predisposing factors

Arising from the posterior cord, the radial nerve tracks along the spiral groove of the humerus at which point it is most commonly injured.

Mechanism of injury

Tourniquets/arterial pressure cuffs, compression against a patient screen or an arm board positioned at an incorrect height creating a step, are the most common perioperative reasons cited for this nerve injury.

Clinical presentation

This injury classically manifests as wrist drop and numbness along the posterior surface of the lower part of the arm, posterior surface of the forearm, and a variable small area on the dorsum of the hand and lateral three-and-a-half fingers.

Median nerve injury (C5–T1)

Mechanism of injury

Direct nerve damage from regional techniques, invasive procedures around the elbow, and compression in the carpal tunnel are usually responsible for injury to this nerve.

Clinical presentation

Median nerve injury results in paraesthesia along the palmar aspect of the lateral three-and-a-half fingers. Motor manifestations include: weakness of abduction and opposition of the thumb, weak wrist flexion, and the forearm being kept in supination. The muscles of the thenar eminence become wasted and the hand appears flattened.

Axillary (C5, 6) and musculocutaneous nerve injury (C5–7)

These injuries mostly result from shoulder surgery or shoulder dislocations. They result in weakness of shoulder abduction and anaesthesia along the upper lateral border of the arm (axillary nerve), whereas weakness of flexion of the elbow and numbness along the lateral border of the forearm are seen with the involvement of the musculocutaneous nerve.

Lower limb peripheral nerve injuries

Sciatic nerve injury (L4–S3)

Mechanism of injury

Stretch, compression, ischaemia, and direct damage are the primary mechanisms. The lithotomy, frog leg, and sitting positions have been implicated in perioperative injury to this nerve (hyperflexion of the hip, abduction, and extension of the leg causes stretching). Regional anaesthetic techniques and hip arthroplasty may also cause injury. A higher incidence is seen in men aged

45–55 yr and in patients suffering with diabetes mellitus. The common peroneal component is usually affected, as this is more superficial compared with the tibial component.

Clinical presentation

Injury manifests as paralysis of the hamstring muscles and all the muscles below the knee leading to weak knee flexion and foot drop. All sensation below the knee except the medial aspect of the leg and foot is impaired.

Femoral nerve injury (L2–4)

Mechanism of injury

This nerve can be injured with compression at the pelvic brim by retractors as seen in abdomino-pelvic surgery, ischaemia associated with aortic cross-clamp, and the lithotomy position with extreme abduction of the thighs and external rotation of the hips. It is also associated with invasive procedures to access the femoral vessels and hip arthroplasty.

Clinical presentation

Loss of sensation at the front of the thigh and medial aspect of the leg occurs (saphenous nerve). Weak hip flexion and loss of extension of the knee will cause difficulty in climbing stairs. Decreased or absent knee jerk reflexes confirm the diagnosis.

Superficial peroneal nerve injury (L4–5 S1–2)

Incidence

An incidence of 0.88% in 2600 total knee arthroplasties attributed mainly to surgical factors is quoted.

Mechanism of injury

Lithotomy and the lateral position are the common risk factors as the nerve is potentially compressed at the fibular head. Length of time in lithotomy has not been associated with an increased risk of developing a PPNI.

Clinical presentation

There is loss of dorsiflexion and eversion of the foot (equinovarus deformity). Sensory manifestations are described along the anterolateral border of the leg and the dorsum of the digits except those supplied by saphenous and sural nerves.

Diagnosis of PPNI

A thorough history and clinical examination are essential to localize the lesion and to identify a pre-existing peripheral neuropathy. The clinical examination serves to identify whether single or multiple nerves are involved and the severity of the motor, sensory, or both impairment which is of prognostic value. If a deficit is found, electrophysiological and imaging studies are performed and early consultation with a neurologist is advisable.

Electromyography and NCSs

Electromyography (EMG) involves recording the electrical activity of a muscle at rest and during volition from a needle electrode inserted within it. The presence and nature of abnormalities depend on the affected component of the motor unit with the distribution of abnormalities indicating the likely source of injury. Reduced numbers of functioning axons, which is the pathophysiology in the majority of PPNI, results in a reduction in the number of motor units recruited. Abnormal spontaneous activity (fibrillation potentials; positive sharp waves due to denervated muscles) takes 1–4 weeks to develop and disappears with reinnervation. Their presence early on implies a pre-existing condition and a specific aetiological diagnosis cannot be made.

NCSs permit the assessment of function in motor and sensory nerves. The conduction velocity and the size of the muscle

Table 2 Summary of electrophysiology findings and prognosis of nerve injury

Seddon's classification	Neuropraxia		Axonotmesis		Neurotmesis	
Nerve conduction studies	Impulse conduction failure across the affected segment. Conduction is normal distal to the lesion		Until Wallerian degeneration occurs, the NCS resembles that of neuropraxia		Resembles axonotmesis but recovery does not occur	
	Conduction block		Conduction failure		Conduction failure	
Action potential in relation to the lesion	Distal	Proximal	Distal	Proximal	Distal	Proximal
Immediate	+	−	+	−	+	−
2 weeks	+	−	−	−	−	−
Weeks to months	+	+	+	+	−	−
EMG	Normal or decreased recruitment		May be normal early on. Abnormal activity after 10–14 days		Abnormal activity (fibrillation potentials and positive sharp waves)	
Prognosis	Recovery within months		Poor prognosis if the endoneurium is breached and surgery may be required. Nerve conduction returns to normal only with regeneration		Surgery required—guarded prognosis	

response estimate the number of axons and muscle fibres activated. NCSs evaluate the functional integrity of peripheral nerves and enable localization of focal lesions. The compound sensory action potential will be reduced in sensory axonal degeneration if the electrodes overlie the affected portion of the nerve. In demyelination, there is focal slowing of sensory conduction or sensory and motor conduction across the injured portion of the nerve (proportional to the severity of the demyelination). NCSs are able to reveal the presence of a subclinical neuropathy predisposing nerves to injury and may also suggest the underlying pathological process (axon loss *vs* demyelination), which has implications for the clinical course and prognosis.

NCSs and EMG are complementary and can help to determine whether a lesion is complete or incomplete; determine the basis of the clinical deficit; localize the lesion; define the severity and age of the lesion; and guide prognosis and course of recovery. Table 2 illustrates the results of EMG and NCSs performed in different lesions at different times and the prognosis of injury.

Imaging studies

Three tesla magnetic resonance imaging provides adequate resolution to visualize peripheral nerves and may be used to identify and confirm the site of the lesion particularly if localization is undetermined by electrophysiological testing. High-resolution ultrasound has also been proposed as an adjunct to electrodiagnosis.

Prevention of PPNI

The ASA has produced a practice advisory notice for the prevention of PPNIs.[8] A sound knowledge of anatomy and the risks posed by positioning patients for surgery is essential in understanding the preventive strategies. The task force emphasizes in their report the paucity of evidence for the recommendations, which are largely based on empiric knowledge and consensus opinion.

A thorough preoperative history and examination is important to identify conditions that predispose the patient to nerve injury and existing neurological dysfunction. Intraoperative hypotension, hypothermia, and dehydration should be avoided. Careful positioning of the patient, protective padding, padded arm boards, and avoidance of contact with hard surfaces or supports that may apply direct pressure to susceptible peripheral nerves are important components of perioperative care.

Arm abduction should be limited to $<90^{\circ}$ in the supine position to prevent brachial plexus lesions. Protection of the ulnar and median nerves: padding should be mandatory and the forearm should be kept in the supine position or in the neutral position. Flexion/extension of the elbow should be $\leq 90^{\circ}$. Preventative measures for lower limb nerve injuries include adequate padding with the patient in the lithotomy, prone, and lateral positions with hip flexion $\leq 120^{\circ}$. Appropriate documentation of specific perioperative positioning actions is essential and has been highlighted as an area of poor practice. A simple postoperative assessment of extremity nerve function may lead to early recognition of peripheral nerve injury.

No nerve localization or monitoring technique has been shown to be clearly superior in terms of reducing the frequency of clinical injury in regional anaesthesia.[9] Consideration should be given to avoiding more potent local anaesthetics (ropivacaine is considered to be the least toxic) and vasoconstrictors in patients with pre-existing neurological disease or peripheral nerve injury. Pain or paraesthesia on injection potentially implies perineurial injection and the needle should be repositioned or the block abandoned.[10]

Declaration of interest

None declared.

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Please see multiple choice questions 29–32.