▪ *Review Article*

Peri-operative peripheral nerve injury

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Abstract

Peripheral nerve injury in peri-operative period is the topic of interest especially for the anesthesiologists because the concentration of rest of the operation theatre team lies on the positioning of the patient required for the surgical exposure rather than towards the stretching or compression of the nerves. Etiology of peri-operative peripheral nerve injury (PPNI) is complex and multifactorial. Prevention of injury and its consequences to some extent can be done with the alertness of the concerned surgical team and the anesthesiologist.

Keywords: peri-operative peripheral nerve injury (PPNI), nerve injury prevention, surgical positioning, injury risk factors

Introduction

The peripheral nervous system consists of motor, sensory and mixed nerves. Signs and symptoms of nerve injury depend on the type of nerve involved. Involvement of sensory nerves may cause numbness, tingling, pain and inappropriate temperature sensations whereas motor nerve involvement may cause weakness or paralysis of the muscles innervated by that particular nerve. Autonomic dysfunction and tropic changes may occur, leading to severe neuropathic pain. The nerve injury not only leads to severe functional disability and a prolonged morbidity to the patient but leaves behind unpleasant litigation to the medical team.[1] During the effect of anesthesia, the patient is partially or totally insensitive to usual warning signals of pain of nerve injury.[2] The physician who delivers anesthesia, either regional or general, is responsible for the patient's welfare.

Incidence of PPNI and anesthesia

The exact incidence of PPNI is unknown because of the heterogeneity and quality of studies, also probably due to under reporting. Before, the cause of PPNI was thought to be toxic properties of anesthetic agent (ether) [3] but later on, the malpositioning on the operating table leading to stretching or compression of nerves, direct trauma and chemical irritation were recognized as risk factors for PPNI.[4,5] Retrospective studies have found that the occurrence of permanent nerve damage after a surgical procedure and anesthesia is 0.03% to 1.4% with or without excluding neuraxial or peripheral nerve block. Commonly injured nerves include the ulnar nerve (28%), brachial plexus (20%), lumbosacral root (16%), spinal cord (13%), sciatic, median, radial and femoral nerves. Generally, 91% of ulnar nerve injuries have no identifiable cause. Spinal cord and lumbosacral nerve root injuries are associated with paresthesia during regional anesthetic techniques or pain during injection of local anesthetic.[6] The incidence of neurological injury is not reduced by ultrasound-guided technique. It only reduces the risk of intravascular injection. After a peripheral nerve block, 15% of patients suffer from transient paraesthesia that can resolve within 1 year. In a prospective study, permanent neurological injury was found to have an incidence of 2.4 in 10000 peripheral nerve blocks.[7,8] Auroy et al in 2002 found 56 major complications in 158083 regional anesthesia procedures (50223 were peripheral blocks) with over all prevalence rate of 3.5/10000. After spinal anesthesia, peripheral nerve injury and cauda equina syndrome was observed in 9 and 3 patients respectively mainly with the use of lidocaine in spinal anesthesia and nerve stimulator stating paresthesia using low intensity.[9] Two ASA closed-claim analyses revealed that anesthesia-related nerve injury is found to be the second most common cause of anesthesia-related litigation comprising 16% of claims.[6,10,11] Poor peri-operative positioning and padding

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is considered as negligence of the anesthetist in most claims.

Pathophysiology and classification of peripheral nerve injuries

Seddon's classification[12] on the basis of nerve pathology

1. Neurapraxia: This is a condition when the myelin is damaged but the axon is intact. Impulse conduction across the affected segment fails. Recovery occurs in weeks to months and the prognosis is good.
2. Axonotmesis: In this condition, there is axonal disruption with preservation of endoneurium and supporting connective tissues. Recovery of function depends on Wallerian degeneration and neural regeneration. The prognosis is variable.
3. Neurotmesis: In this condition, nerve is completely severed with complete destruction of all supporting connective tissue structures. Surgery may be required and prognosis is poor.

Sunderland's classification[13] on the basis of disrupted connective tissue components

1. Type 1: Local myelin injury. Recovery in weeks to months. Good prognosis.
2. Type 2: Disruption of axonal continuity with Wallerian degeneration. Axonal regeneration is required for recovery. The prognosis is good.
3. Type 3: There is a loss of axonal continuity and endoneurial tubes. Perineurium and epineurium are preserved. Scarring can compromise recovery. Surgery may be required. The prognosis is guarded.
4. Type 4: Loss of axonal continuity with damaged endoneurial tubes and perineurium. Epineurium remains intact. Surgery necessary. Prognosis poor.
5. Type 5: Nerve entirely severed. Surgery required. Prognosis poor.

Etiology and mechanisms

The mechanisms that could lead to PPNI are

i. Direct nerve damage. During surgery and regional anesthetic techniques, there can be a direct damage to a nerve. Blunt needles are less likely to penetrate the tougher perineurium surrounding more widely dispersed fascicles and help in identifying tissue planes.[14] This anatomical arrangement which occurs in distal nerves may account for the fact that neural injury does not always result from epineurial injection. In infants, intragluetal injection may cause sciatic nerve injury.[15]

ii. Extravasation of injectates. Median neuropathy has been reported after Pentothial sodium extravasation in antecubital fossa.[16]

iii. Stretch and compression: Poor padding and positioning of limbs, the use of tourniquets and surgical equipments like retractors and theatre bed frames can result in stretching and compression of peripheral nerve.

iv. Ischemia may result by tourniquets with high pressures, prolonged immobility, haematoma surrounding a nerve and local anesthetic agents with epinephrine espesially in patient with micro-vascular disease.[17]

v. Toxicity of injected solutions: The local anesthetics can produce cytotoxic axonal damage particularly with intra-fascicular injection, high concentrations and prolonged exposure.

vi. Pre-existing nerve insult: Diseased nerves are more susceptible to injury than normal nerves. Coexisting conditions such as rheumatoid arthritis, diabetes, periarteritis nodosa, alcoholism and viral infectious neuritis are vulnerable to be injured peri-operatively as it renders the nerve less tolerant of compression at the same or a second locus known as Double crush syndrome.[18]

vii. Unknown factors

Predisposing factors

It can be classified as follows.

1. Surgical factors

Neurosurgery, cardiac surgery, gastrointestinal surgery, orthopaedic surgery and longer hospital stay (greater than 14 days) are associated with higher risk of PPNI.

In general surgery, ilioinguinal, iliohypogastric or genito-femoral nerves may be injured due to direct trauma or from excessive flexion of the thigh onto the abdomen. 15% incidence of postoperative ulnar neuropathies during coronary artery bypass surgery is recorded.[19,20] Median sternotomy is associated with injury to nerves of brachial plexus with reported incidence from 6 to 38%.[21,22] Pneumonectomy and axillary lymph node dissection can stretch the long thoracic nerve causing

motor deficit of the serratus anterior muscle and disrupted scapula movement.[23] Total shoulder arthroplasty have 3% incidence of neurologic complications. Total hip arthroplasty is associated with the sciatic, femoral or obturator nerves injury with higher incidence in female patients.[24] Rose et al. presented 23 cases of peroneal nerve palsy (0.88%) in 2600 knee arthroplasties.[25] Risk of PPNI is higher when surgery involves tourniquets use and tight fitting plaster casts.[26] Meralgia paraesthetica caused by injury to the lateral femoral cutaneous nerve has been reported following iliac bone procurement.[27] Neurosurgery in sitting position and posterior fossa surgery is associated mainly with common peroneal nerve injury.[28] Transoesophageal echocardiography in sitting craniotomy patients were suffered from recurrent laryngeal nerve injury and is possibly due to large probe size, tracheal intubation and excessive neck flexion causing pressure on the nerve.[29]

2. Patient related factors[23]

- Very thin or obese body habitus
- Male gender
- Old age
- History of vascular disease, hypertension, diabetes, smoking
- Coagulopathy or presence of haematoma near nerve
- Infection or presence of abscess near nerve
- Pre-existing generalized neuropathy
- Hereditary predisposition
- Structural anomaly/congenital abnormality (constriction at thoracic outlet or condylar groove, or arthritic narrowing of joint space)

3. Anesthetic factors: general and regional anesthesia are associated with a higher incidence of PPNI compared with monitored anesthetic care where the patient is able to change their position intraoperatively.

- Direct trauma by needle and catheter- nerve trauma may occur even with the insertion of intravenous cannula. Needle gauge, type and bevel configuration have conflicting results. The debate whether to elicit paresthesia or not is still contentious.[30]
- Local anesthetic toxicity- lidocaine and tetracaine are more neurotoxic than bupivacaine at clinical concentrations.
- Neural ischemia- intra-neural injection of volumes causes intra-neural pressure to exceed more than capillary perfusion pressure resulting in neural ischemia and endoneural hematomas formation. Use of epinephrine containing local anesthetics especially in micro-vascular disease can also produce nerve ischemia.[31]
- Tracheal intubation and Laryngeal Mask Airways has been associated with lingual nerve damage which is thought to be due to irregular and over inflation of the cuff and use of nitrous oxide.[32,33]

4. Peri-operative factors: hypo-volemia, dehydration, hypotension, hypoxia, electrolyte disturbances, and hypothermia have been implicated in the development of PPNI.[1]

5. Patient positioning during anesthesia

Diagnosis of PPNI

Patients should be thoroughly assessed with history and examination. Findings must be well documented and neurological review should be made early. Of the total, 95% of significant lesions can be revealed by preoperative assessment of peripheral nerve function by checking the most distal function of frequently injured 5 major nerves. Radial nerve can be tested by the ability to actively extend the distal phalanx of the thumb. The median and ulnar nerves can be tested by the finding of normal sensation in the distal volar phalanx of the index and little fingers respectively. Similarly, peroneal nerve dorsiflex and tibial division plantarflex the great toe.[34] Neurological deficits that arise within the first 24 hours allowing immediate diagnosis mainly represent extra or intra-neural hematoma, intra-neural edema or a lesion in sufficient number of nerve fibers. In cases with persistent paresthesias following regional anesthesia, the onset of symptoms of nerve injury may developed days or weeks later. The presentation of late disturbances in nerve function suggests an alternative etiology such as tissue reaction or scar formation.[35]

Electromyography (EMG): The EMG records the electrical activity of muscles at rest and during volition using a needle electrode. Reduced number of functional axons results in reduction of motor units. Abnormal spontaneous activity or fibrillation potentials due to denervated muscle takes 1-4 weeks to develop so early detection of these waves reflects pre-existing conditions. With re-enervation, the waves disappear. In infectious

neuritis like Guillain Barre Syndrome, EMG changes are seen in paraspinal muscles and in muscles enervated by peripheral nerves too.

NCS – It tells us the function of both motor and sensory nerves. The conduction velocity and the size of muscle response estimate the number of axons and muscle fibers activated. It evaluates the functional integrity and localizes the focal lesion. In demyelination, focal slowing of sensory or motor conduction across the injured nerve is seen. Compound sensory action potential is reduced in sensory axonal degeneration. It can differentiate subclinical neuropathy and underlying pathological process having implications for clinical courses and prognosis.

Magnetic Imaging (3 Tesla) - identify and confirm the site of the lesion if undetermined electrophysiologically.

Evoked potentials - reduced amplitude in evoked responses indicates axonal loss and increased latency indicates demyelination.

High Resolution Ultrasound - an adjunct to electrodiagnosis

Prevention of PPNI

The ASA has produced a practice advisory notice for the prevention of PPNIs.[36] Prevention of nerve injury at first is done by an awareness of the potential for injury. The patient's general condition and pre-existing neurologic lesions should be determined. Extreme care must be exercised in positioning the anesthetized patients aiming at good surgical access and to minimize risk of harm to the patient. Prevention is the realization of differential risk. For example, in a steroid-dependent patient, axillary roll for the lateral position is beneficial as there is more risk of fracture and skin tearing. Similarly in cases of fixed flexion disorders and preexisting joint or nerve pain, positioning prior to induction of anesthesia is useful to ensure comfortable position. Position should be checked at regular intervals (every 30 minutes) during surgery to identify malpositioning timely. Proper surgical planning should be made, like avoiding sitting position in patients with known intra-cardiac defects or performing the complex spine procedure in two stages to reduce operative time and optic ischemic neuropathy. Peri-operative nursing recommendations are also there to prevent PPNI. Nurses should collaborate with other surgical team members to establish a protocol for identifying patients at risk for peripheral nerve injury and determine a standard for their care and for the reporting of these injuries. These actions will improve the quality and safety of patient care.[37] Interactions with patients and their families should be empathic and supportive. Nerve injuries may not manifest themselves immediately on awakening. Patient complaining even days later must be taken seriously and investigated in light of recent surgery. Because the cause of many position related nerve injuries is unknown, caution is advised in categorically accepting responsibility for their occurrence except in entirely clear-cut circumstances.

Some preventable points are

- Intraoperative hypotension, hypothermia and dehydration to be avoided.
- Head and neck must be in a neutral position to avoid vascular or neural injury.
- Eyes should be protected to prevent ischemic optic neuropathy.
- Careful positioning of the patient with all limbs placed in natural positions without stretch to nerves/ muscles/tendons/vessels, protective padding, padded arm boards, and avoidance of contact with hard surfaces or support to be kept in mind.
- The superficial nature of the plexus, running between two fixed points (intervertebral foramen and the axillary sheath), and its way through a limited space between the clavicle and first rib and proximity to a number of mobile bony structures make it susceptible to injury. Lateral decubitus position is associated with compression of brachial plexus against the thorax and humeral head. Upper limb joints should not extend beyond ninety degrees. Shoulder abduction with lateral rotation should be avoided if possible to prevent stretching of brachial plexus. Avoid shoulder braces to support trendelenburg position.
- Prolonged pronation of the forearm can compress the superficial lying ulnar nerve in the cubital tunnel. Median nerve injury may occur in antecubital fossa, at mid arm or as a part of brachial plexus injury. Avoid steep trendelenburg position and great care exercised in abducting the arm for intravenous infusions and monitoring to prevent compression of the ulnar nerve against the table edge. Protection of the both nerves can be done by padding and keeping forearms in the supine or in neutral position.

- Tourniquets and arterial pressure cuffs, compression against a patient screen or an arm board positioned at an incorrect height creating a step compressing humerus in the radial groove are the most common peri-operative reasons for radial nerve injury.
- Stretching of the sciatic nerve occurs due to hyperflexion of the hip, abduction, and extension of the leg. Tibial branch may be affected due to compression in the popliteal space by hanging the leg over the bedrail. Common peroneal nerve is usually affected at the level of fibular head. Injury is reported to occur due to crossing the legs, kneeling for periods of time, the occupational wearing of knee pads and occlusive dressings. Prevention is done by adequate padding at the lateral aspect of upper fibula, avoiding extreme lithotomy position and avoiding prolonged (>2 hour) exposure to lithotomy position.[38]
- In infants and children, intramuscular injections should be given in anterior thigh rather than in the buttocks.
- More potent local anesthetics and vasoconstrictors should be avoided in patients with preexisting neurological disease or peripheral nerve injury. Ropivacaine is considered to be the least toxic. Pain or paraesthesia on injection potentially implies perineurial injection and the needle should be repositioned or the block abandoned.[39]

Management of position injuries

Once a position-related nerve injury is suspected, thorough physical examination and assessment should be done. Further documentation of nerve injuries and possible recommendations for intervention should be sought through neurologic or ophthalmologic consultations. Serious problems like epidural hematoma or cervical spine compression and immediate intervention (surgical decompression) if required should be kept in mind. Subacute and chronic treatment of nerve injuries is best accomplished in close collaboration with a neurologist and psychiatrist. It may include analgesics, medications effective in the treatment of neuropathic pain, splinting and physical therapy. Chronic pain syndromes may result requiring sympathetic blockade, surgical neurolysis and nerve grafting.

Performance of regional techniques in anesthetized patients

No nerve localization or monitoring technique is superior in terms of reducing the frequency of clinical injury in regional anesthesia.[40] It is better to avoid nerve blocks in anaesthetized or heavily sedated patients because they cannot respond to pain associated with needle or catheter induced paresthesias or intra-neural injections, a major risk factors for ensuing nerve injury. 4 cases were reported with permanent cervical spinal cord injury after interscalene block under general anesthesia or heavy sedation even with the use of nerve stimulator.[41] Careful interpretation in paediatric literature should be made for whom nerve blocks cannot be done without sedation or anesthesia.

Controversies

Studies have shown that the anesthetic technique is unrelated to incidence of ulnar nerve injury.

Unconscious patients are not at increased risk of ulnar nerve injuries compared to awake patients. This arise the question whether patient positioning, nerve compression and stretch are significant mechanisms in peri-operative ulnar nerve injury. Initial symptoms of ulnar nerve injury occur most frequently more than 24 hours after the surgical procedure. In addition, abnormal nerve conduction is often found in the contralateral unaffected arm, indicating likely predisposition to ulnar nerve symptoms preoperatively.[23] These findings and its delayed onset has lead to much debate regarding the etiology of the injury. Closed claims analysis has also demonstrated that nerve injury can occur even with optimal patient positioning and padding to risk areas like the elbow.

Outcomes

In most cases, injuries resolve within 6-12 weeks. More than 50% of patients typically regain full sensory and motor function within 12 months. Patients with poor recovery can have ongoing or permanent symptoms. Permanent injury may be minor (such as small area of sensory loss that is minimally inconvenient to the patient), or major and disabling (such as significant motor loss and chronic pain). Poor recovery can have a profound impact on quality of life for patients with ongoing nerve injury.[23]

Conclusion

Surgical awareness of the risk, proper positioning of the patient for surgery, good anesthetic techniques, choice of right drugs in right concentrations and following preventable recommendations can reduce the risk of most peri-operative peripheral nerve injuries.

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