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Medico-legal aspects of peripheral nerve injury

MEDICO-LEGAL ASPECTS OF PERIPHERAL NERVE INJURY

Iatrogenic nerve injuries are always a matter for concern. This article will address the common causes, diagnosis and management of iatrogenic peripheral nerve injuries.

The most widely used classification of peripheral nerve injury is that described by Seddon in 1943.¹ He divided nerve injuries into three types: neurapraxia, axonotmesis and neurotmesis.

Neurapraxia (inactivity of the nerve) is a non-degenerative lesion of a nerve characterised by a complete or partial failure to propagate an action, potentially resulting in motor and/or sensory loss. It is usually caused by compression or ischaemia, resulting in ischaemia of the myelin sheath. The nerve remains intact and Wallerian degeneration does not occur. It is reversible if the injurious agent is removed. If the distal segment of the nerve is stimulated, there is a motor response. The lesion recovers by remyelination of the distal segment and takes between two and 12 weeks, depending on the age of the patient and the site of the injury.

In practice, it is unwise to assume that a lesion is a neurapraxia rather than a more severe injury because this will lead to delay in diagnosis and a poorer outcome. The presence of persistent pain suggests that the injurious agent is continuing to act. The diagnosis should not be

made in the presence of a strong Tinel test which indicates that axons have been ruptured.

An axonotmesis (cutting of the axon) is the result of disruption of the axon and its myelin sheath. The supporting structures, Schwann cells, endoneurium, perineurium and epineurium remain intact. It is usually the result of severe compression or a crush injury. Wallerian degeneration occurs distally, and proximally to the closest node of Ranvier. Repair is by a combination of collateral sprouting in lesser injuries and axonal regeneration in more severe injuries. The latter occurs at approximately 1 mm to 2 mm per day and the time to recovery can vary between two and six months. Nerve conduction studies show a loss of conduction in the distal segment three to four days after injury due to demyelination. There are also small or absent compound muscle or sensory nerve action potentials as a result of axonal loss. Electromyogram (EMG) studies show fibrillation potentials and sharp waves two to three weeks after injury as a result of axonal loss. The degree of recovery depends on the age of the patient, the site of injury and the amount of fibrosis that occurs.

Neurotmesis (cutting of the nerve) is the complete disruption of a peripheral nerve by any means. Wallerian degeneration occurs distal to the lesion, and nerve conduction studies show loss of conduction in the distal segment three to four days after injury. Electromyogram

studies will show fibrillation potentials and sharp waves two to three weeks after injury. Surgical intervention is always required to repair the nerve, whether by direct suture or by grafting. The subsequent recovery can take anywhere from two to 18 months depending on the age of the patient and site of the lesion.

The causes of peripheral nerve injury can be broadly divided into closed and open. Closed injuries include ischaemia, crush, compression, traction, thermal injury, electric shock, vibration and radiation whereas open injuries are usually caused by laceration or injection.

In this article, we are principally concerned with what are generally known as iatrogenic injuries, although perhaps more properly termed iatrogenous or iatropathic. Whichever terminology is used, they are injuries caused by a doctor or, increasingly, by others who treat patients.

The late George Bonney,² in a masterly paper in *The Journal of Bone and Joint Surgery [Br]*, stated that “when a patient enters hospital without a nerve lesion and emerges with one, it is seldom possible to resist an allegation of negligence.” He continued: “if there is an incision over the line of a main nerve and if, after operation, there is complete paralysis (including vasomotor and sudomotor paralysis) in the distribution of that nerve, speculation is unnecessary: the nerve has been cut, and there will be no recovery unless it is explored and repaired.”

He also noted: “when pressure on a nerve has been followed by partial paralysis in its distribution, but stimulation below the level of the lesion produces a motor response, it is reasonable to assume that there has been a conduction block which will recover.” Importantly, he added: “Between these extremes, precise diagnosis is much more difficult.”

The American plastic surgeon Lee Dellon³ has added: “Failure to make the diagnosis of a nerve injury, and failure to treat that complication of the first surgery, the iatrogenic nerve injury, is as much a cause for concern as the initial injury to the peripheral nerve.”

So, what is the overall incidence of iatrogenic peripheral nerve injury? The simple answer is that it is unknown. The NHS Litigation Authority does not specifically record cases under the heading ‘peripheral nerve injury’. There are, however, two considerable series from the 1990s which are of assistance. Khan and Birch⁴ reviewed 612 cases between 1991 and 1998, of which 291 were subsequently explored, and 144 were the subject of litigation. Many of these followed excision of a tumour or cyst (67), biopsy of a lymph node (52) or internal fixation of a fracture (48). A second paper, by Kretschmer et al,⁵ studied 722 consecutive cases of peripheral nerve injury, of which 17.5% (126) were iatrogenic. The pattern of injury was much the same as in the paper of Khan and Birch. Orthopaedic and general surgeons were by far the greatest culprits.

Anaesthesia-related peri-operative peripheral nerve injury is relatively uncommon. Welch et al⁶ found an overall incidence of 0.03% in a retrospective study of 380 680 cases. The principal causes were: poor padding and positioning of limbs; needle trauma secondary to regional anaesthesia; haematoma surrounding a nerve; and toxicity and direct damage from intraneural injection of local anaesthetic agents. Additionally, they noted that pre-existing disease such as diabetes, smoking, hypertension and pre-existing neuropathy, as well as intra-operative problems with hypovolaemia, dehydration and hypotension, were also contributory.

The inappropriate use of a tourniquet also causes problems. Braithwaite and Klenerman modified Bruner’s ten rules for the safe use of a tourniquet in 1996.⁷ They remain as appropriate today as they were 20 years ago.

The diagnosis of a peripheral nerve injury must be the responsibility of the treating clinician. Nerve repair, however, is the business of a

subspecialist. There is no doubt that the earlier a peripheral nerve injury is diagnosed and treated, the better. In the case of neurotmesis, the injury is more easily recognised, and the nerve stumps can be mobilised and approximated without difficulty. The best results are achieved by early direct repair. The reasons for this are well summarised in a British Orthopaedic Association (BOA) ‘blue book’ of 2011.⁸ A delay of two months halves the number of axons crossing a repair and halves their rate of growth. Longer delay causes further deterioration. Motor endplate loss progresses even after repair and is near total after one year (for a proximal repair). Central cord changes also progress prior to repair and become less reversible with time.

If a complete or partial laceration of a nerve is identified at operation, whether as the result of trauma or iatrogenic injury, a surgeon trained in nerve repair should undertake a primary epineural suture. An untrained surgeon, however, should attempt to oppose the ends of the nerve, gently mobilising it if necessary. The nerve ends should then be tagged with coloured epineural sutures. In either case, there should be clear, accurate documentation of the injury and the action taken. The situation should be discussed with and explained to the patient, and the resultant neurological deficit recorded. The case should be discussed with and, if necessary, referred to an experienced peripheral nerve surgeon. If the nerve injury is identified post-operatively, similar steps should be taken. However, under these circumstances the nature of the lesion will be unclear. If the nerve is thought to have been divided, that is if Bonney’s criteria apply, the wound should be re-explored with a view to primary nerve repair. If not, EMG and nerve conduction studies (NCS) should be carried out at between two and three weeks. These will distinguish lesions with predominant demyelination from those with substantial axonal loss, and assess the extent of that loss.⁹

Other indications for surgery are, as Birch¹⁰ has recommended: failure of recovery of a presumed axonotmesis at the predicted time; deterioration of a lesion while under observation; or persistent, intractable pain. The aim of surgery in these cases is to establish the diagnosis, relieve pain and improve function.

Non-operative treatment is indicated if the patient is clearly improving, if there has been a prolonged delay in presentation, if, for

whatever reason, palliative treatment is to be preferred, or if the predominant symptom is pain which can be relieved by non-operative means. Whether or not it is indicated where there is an ongoing claim for compensation, is undoubtedly a matter for debate.

The commonest causes of an allegation of mismanagement of a peripheral nerve injury are: inadequate informed consent; avoidable damage to a nerve or nerves; delay in diagnosis, referral or treatment; and a misdiagnosis and inappropriate subsequent treatment. The matter of informed consent is now subject to the Montgomery ruling but I would suggest that there should be a detailed discussion if the proposed operation puts a named nerve at more than a remote risk of damage. This would include, for example, the excision of a lump from the posterior triangle of the neck, fracture fixation or the removal of metalwork, and the excision of any lump from, or from close to, a nerve. This discussion should be clearly recorded.

The BOA ‘blue book’ of 2011 also states: “It must be accepted that despite careful surgery nerve injury during operation may occur. Recognition and prompt remedial action after the event are the keys.”⁸

In conclusion, properly informed consent to any orthopaedic procedure, particularly those in which peripheral nerves may be at risk, is more important than ever. A thorough knowledge of the local anatomy reduces the risk of inadvertent nerve damage. Any peripheral nerve injury should be promptly identified, documented and discussed with the patient. Early discussion with and, if necessary, referral to an expert in these injuries is always advisable.

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