

cytokine production [3,5], up-regulation of TrkA mRNA in PBMC might play a role in the modulation of the immune system after induction of anaesthesia. Further study is needed to investigate the cause of change in TrkA expression in PBMC induced by general anaesthesia.

K. Tsugita, M. Hirose, E. Murata
Department of Anaesthesiology and Reanimatology
Faculty of Medical Sciences
University of Fukui, Yoshidagun
Fukui, Japan

S. Ibo
Department of Pathological Sciences
Faculty of Medical Sciences
University of Fukui, Yoshidagun
Fukui, Japan

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Perioperative ulnar neuropathy following shoulder surgery under combined interscalene brachial plexus block and general anaesthesia

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EDITOR:

Permanent neurological deficit, as a direct result of regional nerve blocks, is rare. Some physicians remain sceptical concerning the benefits of regional anaesthesia, and there is a tendency to instinctively attribute the development of a perioperative neuropathy to the performance of a contemporaneous regional nerve block. If other remedial causes are not considered, a delay in appropriate management may lead to a permanent, devastating injury. We describe a case of debilitating, permanent ulnar neuropathy that occurred following routine arthroscopic shoulder surgery performed under combined interscalene brachial plexus block and general anaesthesia. A diagnosis of ulnar neuropathy localized

to the elbow secondary to postoperative immobilization of the limb in elbow flexion was eventually made following imaging and electrodiagnostic studies.

Interscalene brachial plexus block affords effective anaesthesia and analgesia for both arthroscopic and open shoulder surgery. Unease concerning the risk of neurological injury may result in this technique being underused. When a neurological complication develops after a regional technique has been performed, there is an instinct to attribute the injury to the anaesthetic technique and other causes may be overlooked. This can result in a delay in remedial management, apprehension on behalf of the anaesthetist and an unnecessary reform in anaesthetic practice.

This article uniquely highlights the importance of prompt investigation and accurate identification, where possible, of the aetiology, type and site of nerve injury, as timely surgical intervention for some lesions can effect a significant improvement in functional outcome and prognosis. Furthermore,

Correspondence to: Hervé Bouaziz, Department of Anaesthesia and Intensive Care Medicine, Hôpital central, 29 Avenue du Maréchal de Lattre-de-Tassigny, 54035 Nancy Cedex, France. E-mail: h.bouaziz@chu-nancy.fr; Tel: +33 383858585; Fax: +33 383858559

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identification of causal factors can facilitate the appraisal of current practices and contribute to the future development and implementation of preventative strategies.

Case report

A 55-yr-old, 71 kg male, ASA Grade I, presented for arthroscopic repair of a rotator cuff muscle tear involving the left shoulder. He had no medical history, was not receiving any medication and had no allergies. He was premedicated with 0.5 mg of oral alprazolam on the morning of surgery. He underwent a left-sided interscalene brachial plexus block with a 25-G, 25 mm insulated needle (Stimuplex; B. Braun Medical Inc, Bethlehem, PA, USA) using 30 mL of 0.75% ropivacaine. Needle placement was confirmed by elicitation of a biceps twitch at 0.5 mA and no adverse effects were experienced during the procedure. After confirming complete sensory and motor block, general anaesthesia with orotracheal intubation was instituted. The patient was placed in the beach chair position and underwent routine arthroscopic repair of the rotator cuff muscles. During the operation, the patient's left arm was manually supported by the surgeons, and at the end of the procedure, was immobilized in elbow flexion using a Velpeau-type padded sling. The operative time was 3 h.

At 3 h postoperatively, in the post-anaesthesia care unit, the patient started to complain of a painful left elbow that was initially relieved by removal of the sling and extension of the elbow. Within 24 h, the patient started to experience pain, paraesthesia and motor weakness localized to the ulnar nerve distribution of his left hand. His symptoms persisted, and treatment with gabapentin and laroxyll was commenced. Clinical examination at 5 weeks postoperatively demonstrated obvious clawing of the hand and severe allodynia. Magnetic resonance imaging (MRI) of both brachial plexuses confirmed normal anatomy. Electrodiagnostic studies demonstrated a significant lesion of the ulnar nerve localized to the elbow. Nerve conduction at the level of the brachial plexus was unremarkable. In an attempt to alleviate his distress, the patient underwent simple ulnar nerve exploration and decompression at the elbow 7 weeks after his shoulder surgery. The macroscopic appearance of the nerve at this time was unremarkable. Repeat electrophysiologic studies performed 4 months after the primary surgery reaffirmed the presence of an axonal injury of the ulnar nerve at the elbow. There was minimal improvement in motor function following surgery and months of extensive multidisciplinary therapy, and more than 3 yr later, the patient

remains afflicted with a debilitating, painful sensorimotor deficit of his left hand. He is currently unable to work.

Discussion

Perioperative neuropathy is a recognized, serious complication of surgery and regional nerve blocks that can result in debilitating functional impairment, significant financial burden and medicolegal consequences. Perioperative neuropathies are not unusual. The most common perioperative neuropathies involve the ulnar nerve (0.26–0.5% of adult patients undergoing non-cardiac surgery; 28% of all nerve injury claims in the ASA Closed Claims Study database) and the brachial plexus (20%) [1].

Numerous mechanisms contribute to the development of a perioperative neuropathy including direct nerve trauma, nerve ischaemia, nerve compression (external or internal) and excessive nerve stretch. Both shoulder surgery and brachial plexus anaesthesia are independent risk factors for brachial plexus injury, whilst tourniquets, excessive manipulation and inappropriate arm positioning during and after surgery may contribute to compressive or entrapment neuropathies [2].

Most peripheral nerves are intolerant of traction beyond 110%, and patient positioning with the elbow in extreme flexion can stretch and compress the ulnar nerve as it passes deep to the cubital tunnel retinaculum at the elbow. Prelipp and colleagues have demonstrated that supination of the forearm minimizes the direct pressure applied to the ulnar nerve, whereas pronation incurs the greatest pressure regardless of arm abduction between 30° and 90°. With the forearm in neutral position, pressure over the ulnar nerve decreases as the arm is abducted from 30° to 90° [3]. Males develop perioperative ulnar neuropathies more frequently than females. Anatomic differences in the size of the bony tubercle of the coronoid process combined with a reduction in fat content on the medial aspect of the elbow increase the vulnerability of the nerve to ischaemic and compressive injury in males [4].

Brachial plexus injuries following regional nerve blocks can be a result of direct needle or catheter trauma, local anaesthetic neurotoxicity, microhaematoma formation, perineural oedema or vasoconstrictor-induced nerve ischaemia. A recent meta-analysis estimated a 2.84% incidence of transient neuropathy following interscalene brachial plexus block. Only one case of permanent neuropathy (>9 months) was identified amongst an estimated 5700 interscalene brachial plexus blocks [5]. Interestingly, the study documenting this particular case reported that electrodiagnostic studies established that

independent processes, including carpal tunnel syndrome and sulcus ulnaris syndrome, accounted for a significant number of symptoms persisting for 1 month or more following an interscalene block. The authors concluded: 'Interscalene brachial plexus block performed with a standardized technical approach, material, and drugs is associated with an incidence of short- and severe long-term complications of 0.4%' [6].

Numerous drugs and medical conditions predispose to the development of perioperative neuropathies and some patients may have a pre-existing, sub-clinical neuropathy that becomes symptomatic consequent to the manoeuvres and manipulations during and after surgery [7]. Another mechanism is that of the 'double-crush' phenomenon, which describes the increased vulnerability of a nerve to injury at a second site when already compromised at a different, pre-existing locus [8]. Frequently, the aetiology is multifactorial, and the exact mechanism of injury cannot be identified.

Nerve conduction studies (NCS) and needle electromyography (EMG) do not specify the exact mechanism of nerve injury but are used to establish the type of injury, its severity, distribution and temporal profile. NCS can localize a focal nerve lesion, differentiate between axon loss and demyelinating processes, and identify the presence of a pre-existing, sub-clinical polyneuropathy. EMG provides additional information regarding the distribution, severity and time course of *axonal* injuries when there is motor involvement. Mild compressive neuropathies usually result from segmental demyelination that typically manifests as conduction block. In these cases, complete and rapid recovery can be expected. In severe, acute processes, axonal degeneration may occur and changes of an acute axonal neuropathy may appear on NCS as early as 3–5 days after its onset. EMG evidence of denervation (increased insertional and spontaneous activity) appear approximately 3 weeks after the injury. The presence of neurogenic Motor Unit Action Potentials indicates chronicity as they do not appear for at least 2–3 months following injury because of the time required to develop collateral reinnervation [9].

Most perioperative neuropathies are transient, and complete recovery the rule [5]. Enduring symptoms (>6 weeks), motor involvement and electrophysiological changes of axonal degeneration tend to indicate a more severe lesion and are associated with a poorer prognosis [10].

Typically, perioperative ulnar neuropathy does not become symptomatic until at least 2 days after surgery [3]. The presence of neurological symptoms in our patient within the first 24 h could have been

misconstrued as a brachial plexus injury caused by the interscalene block. However, the distribution of neurological deficit indicated the involvement of the ulnar nerve, which is frequently spared following the performance of an interscalene block [11]. MRI ruled out any anatomical abnormalities of the brachial plexus, and electrophysiological studies performed 5 weeks after the onset of symptoms demonstrated changes consistent with a severe axonal injury localized to the elbow, which when interpreted in the clinical context from which they were obtained, strongly implicated a compressive process. Factors predisposing our patient to the development of a perioperative neuropathy included his gender, upper limb surgery and postoperative immobilization of the limb in extreme elbow flexion. It is also possible that he may have had a pre-existing, sub-clinical, compressive neuropathy that was aggravated by handling of his arm during surgery and/or prolonged immobilization of the limb in elbow flexion postoperatively. Early electrophysiological studies, performed within 48–72 h of the onset of symptoms may have acknowledged the presence of a chronic, underlying condition. Surgical decompression of the ulnar nerve at the elbow undertaken 7 weeks after the onset of symptoms provided negligible relief, and we can only speculate as to the improvement in outcome if performed earlier.

In conclusion, whilst the development of a perioperative ulnar neuropathy secondary to inappropriate arm positioning has been well described, this case serves to underscore the importance in challenging the tendency to casually attribute a perioperative neuropathy to a contemporaneous regional nerve block. Permanent neurological deficit is rare following peripheral nerve block and other, potentially reversible aetiologies must be rigorously excluded [5]. Prompt differentiation between a brachial plexus injury and compressive mononeuropathy is crucial, as there is potential for significant symptom relief and recovery of neurological function following timely surgical exploration and decompression of the latter. Prolonged duration of symptoms has been associated with poorer outcomes, and preoperative identification of the high-risk patient coupled with strategies designed to prevent peripheral nerve injury both intra- and postoperatively therefore assumes paramount importance.

T. O'Neill, E. Cuignet-Royer, M. Lambert
C. Cornet, H. Bouaziz

Department of Anaesthesia and Intensive Care Medicine
Hôpital central
CHU Nancy, France

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Potential for foreign body going unnoticed with a disposable fibreoptic laryngoscope

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EDITOR:

Endotracheal intubation is regarded as the gold standard in advanced airway management. In cases where intubation is foreseen to be straightforward, practitioners would use a Macintosh laryngoscope [1,2] for the purpose of visualization of the larynx and vocal cords. With the inherent risk of transmission [3] of diseases via saliva and lymphatic tissue, the use of 'single use only' [4] laryngoscope blades has become common practice in many hospital departments. We report a case where the routine use of such a blade lead to an unsuspected breakage in the fibreoptic light source with the potential for serious consequences.

Case report

A 58-yr-old female, ASA Grade II was scheduled for elective mastectomy and sentinel node biopsy as an elective procedure. During preoperative assessment of the airway she was noted to have prominent top incisors, malocclusion of jaws with the upper teeth overriding the bottom set and a thyromental distance less than 6 cm. Mouth opening and neck extension were full. Although some difficulty at

intubation was anticipated, it was not judged to be impossible with routine equipment.

Standard recommended monitoring was applied. After pre-oxygenation, fentanyl 100 µg, propofol 200 mg and atracurium 35 mg were administered. After 3 min, intubation was attempted using a Timesco Surgical and Medical, London, Callisto Macintosh size 3 disposable laryngoscope. Laryngoscopy revealed a Cormack & Lehane grade III view [5] and tracheal intubation could not be performed. The laryngoscope blade was removed under direct vision with the intention of trying a McCoy blade. At this point, a glistening object was noted in the pharynx. This was retrieved using Magill's forceps. On checking the laryngoscope, it became apparent that the light source had fractured and the distal one-third had fallen into the patient's pharynx (Fig. 1).



Figure 1.
The laryngoscope blade with its fractured fibreoptic light path.

Correspondence to: Sam George, Department of Anaesthesia, Good Hope Hospital, Rectory Road, Sutton Coldfield, Birmingham B75 7RR, UK. E-mail: drsamgeorge@yahoo.com; Tel: +121 424 7428; Fax: +121 424 7407

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