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## Traction injury of the brachial plexus confused with nerve injury due to interscalene brachial block: A case report

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## ABSTRACT

**INTRODUCTION:** Shoulder surgery is often performed with the patient in the so called “beach-chair position” with elevation of the upper part of the body. The anesthetic procedure can be general anesthesia and/or regional block, usually interscalenic brachial plexus block. We present a case of brachial plexus palsy with a possible mechanism of traction based on the electromyographic and clinical findings, although a possible contribution of nerve block cannot be excluded.

**PRESENTATION OF THE CASE:** We present a case of a 62 year-old female, that suffered from shoulder fracture-dislocation. Open reduction and internal fixation were performed in the so-called “beach-chair” position, under combined general-regional anesthesia. In the postoperative period complete motor brachial plexus palsy appeared, with neuropathic pain. Conservative treatment included analgesic drugs, neuromodulators, B-vitamin complex and physiotherapy. Spontaneous recovery appeared at 11 months.

**DISCUSSION:** in shoulder surgery, there may be complications related to both anesthetic technique and patient positioning/surgical maneuvers. Regional block often acts as a confusing factor when neurologic damage appears after surgery. Intraoperative maneuvers may cause eventual traction of the brachial plexus, and may be favored by the fixed position of the head using the accessory of the operating table in the beach-chair position.

**CONCLUSION:** When postoperative brachial plexus palsy appears, nerve block is a confusing factor that tends to be attributed as the cause of palsy by the orthopedic surgeon. The beach chair position may predispose brachial plexus traction injury. The head and neck position should be regularly checked during long procedures, as intraoperative maneuvers may cause eventual traction of the brachial plexus.

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## 1. Introduction

Shoulder surgery is often performed with the patient in the so called “beach-chair position” with elevation of the upper part of the body and with the aid of an accessory for the patient’s head to make the procedure easier.

The anesthetic procedure can be general anesthesia or regional block, usually interscalene brachial plexus block. The combination of both types of anesthesia is theoretically associated with less postoperative pain (due to regional block) [14,19] with high intraoperative comfort (due to general anesthesia).

Interscalene block can be performed by mechanical feeling of passing through the fascia (click) or by nerve identification (paresthesia, neurolocalization) in order to apply enough amount of local anesthesia to the area around the nerves that must be blocked [4,17,19].

There may be complications related to both anesthetic technique and patient position/surgical maneuvers. We present a case of a patient that suffered from brachial plexus palsy after open reduction and internal fixation of proximal humeral fracture, in which the anesthetic procedure was a combination of general anesthesia with interscalene plexus block.

The aims of this report are to identify the possible causes of the neurological injury and to discuss the mechanisms that may have been involved in the lesion based on the clinical and electromyographic findings.

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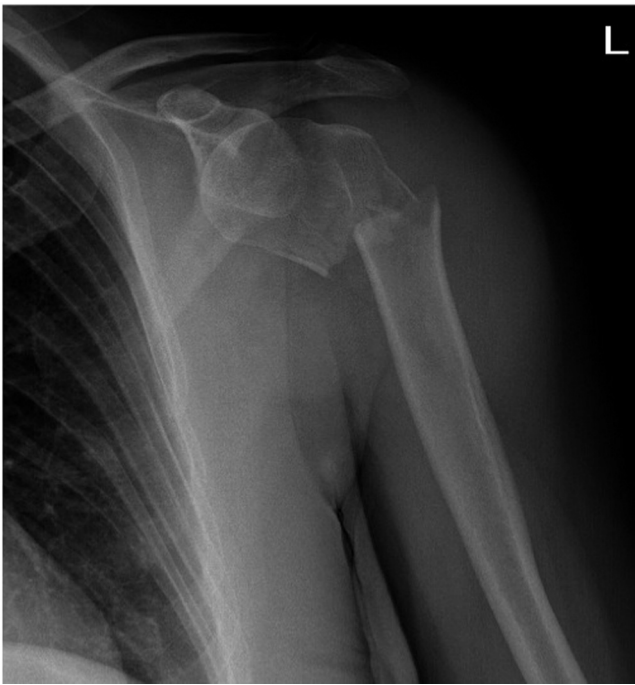


Fig. 1. Radiological image of fracture-dislocation of the shoulder.

## 2. Presentation of the case

A 62 year-old female, with no medical history, moderate smoker, who was admitted into Casualty after falling off her motorcycle while it was not moving and suffering traumatism in the left shoulder. The initial physical examination revealed severe acute pain, loss of mobility and deformity of the left shoulder, without neurovascular damage. The radiology showed a proximal humeral neck fracture with anterior glenohumeral dislocation (Fig. 1).

Reduction of dislocation was performed by manipulation under general anesthesia and assessed by x-ray. After manipulation, the neurovascular examination was still normal.

The definitive treatment (open reduction and internal fixation) was performed after 48 h. In order to improve postoperative pain relief and early mobilization, ultrasound-guided interscalene block (with neurostimulation) was performed in combination with general anesthesia. Nerve block was done with the patient in supine position with lateralization of the head 45° to the contralateral side. A mild sedation with midazolam 0.01 mg/kg was performed. The blockade was performed with the patient awake, using neurostimulation of an intensity of 1.0 mA, a frequency of 2 Hz and impulse duration of 0.1 m/s (TOF Watch® SX). A scanner with a 7.5 MHz linear probe was used (Stimuplex, B. Braun Medical). The plexus divisions were localized with the aid of the probe. Once twitch of the deltoid, bicipital, tricipital and forearm muscles had been obtained, the intensity was lowered to 0.4 mA. A 45 mm 20-gauge 17°-bezel needle (Locoplex) was used. Local anesthetics were injected (10 cc of 0.5% bupivacaine plus 10 ccc of 2% lidocaine) while applying slow and constant pressure, always after a negative aspiration test. There was no pain or paresthesia during the procedure. After injection, muscular activity ceased. The anesthetist scanned the nerves proximally and distally during injection to verify that there was no intraneural injection. After assessing the quality of the blockade, general anesthesia was induced with 150 mg propofol, 150 µg fentanyl and 50 mg rocuronium. Endotracheal intubation proceeded uneventfully. Anesthesia was maintained with continuous IV infusion of propofol (6 mg/kg/h) and remifentanyl (0.10 µg/kg/min). Mechanical ventilation was done with a mixture of oxygen and air

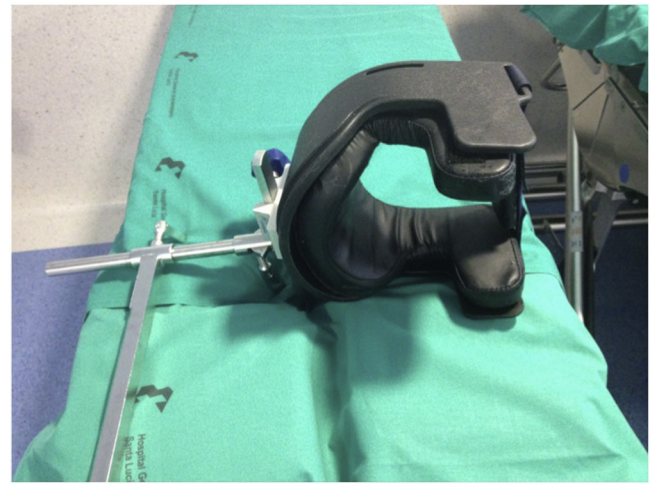


Fig. 2. Operating table accessory for the head used in the beach-chair position.

in a 1:2 ratio. After spontaneous recovery (TOF-ratio >90%) neuromuscular blockade was reversed by the IV administration of 2.0 mg/kg sugammadex.

The patient was placed on a Maquet operating table in the “beach-chair” position, with the head steadied by means of its specific accessory (Fig. 2). Deltopectoral approach was used. The fracture was difficult to reduce due to the associated instability. The bone fragments were temporarily fixed by Kirschner wires and then stabilized using a proximal locking plate (Philos, Synthes). The position of the arm was in slight abduction for most of the time. Pulling and rotating manoeuvres were applied gently. Surgery took 170 min. Submuscular drainage was used and the superior limb was immobilized in a sling. The postoperative x-ray showed good reduction of the fracture with adequate joint congruity (Fig. 3).

After surgery, physical examination showed complete motor brachial plexus palsy (deltoid M0/5, biceps M0/5, forearm muscles M0/5, hand muscles M0/5), with maintenance of tactile and thermoalgesic sensitivity. There were also paresthesias in the forearm (lateral part) and thumb. The patient felt diffuse and severe neuropathic pain. Neither hematoma nor external signs of compartment syndrome were observed at that time.

The patient was treated with pregabalin (initial dose of 75 mg orally per day for the first week, which was progressively increased until a maximal dose of 300 mg per day), B vitamin complex and tapentadol retard (50 mg orally per day).

An orthesis type Winn-Parry was used to avoid stiffness of the hand. One month after surgery, the patient had undergone partial motor recovery, as she was able to adduct her thumb.

Acute electromyography (EMG) was not performed under the assumption that the results would not be reliable until Wallerian degeneration occurs. Tests that are non-sensitive to the time after injury (somatosensory-evoked potentials, motor-evoked potentials) were not performed at that time either.

EMG was performed at 3-week follow-up. It showed fibrillations and positive sharp waves in left deltoid, biceps and palmaris brevis muscles without voluntary trace in left deltoid and bicipital muscles, loss of motor units in left palmaris brevis, decrease in sensory-evoked potential width of left radial and cubital nerves, absence of motor-evoked potential in left axillary nerve (Erb-deltoid), increase in motor-evoked potential latency of left musculocutaneous nerve (Erb-biceps), width asymmetry of motor-evoked potential of right and left cubital nerve.

The lesion was described as a brachial plexus injury with pre- and postganglionic damage (severe partial axonotmesis of upper

**Table 1**  
electromyography (table and graphics). MUAP: motor unit action potential duration.

Electromyography	Spontaneous muscle activity				MUAP			Voluntary trace
	Fibrillations.	Positive Sharp waves	Fasciculations.	High Frequency	Amplitude	Duration.	Polyphasia	Pattern
I. DELTOID	N 3+	3+	No	No	N	N	N	No trace
I. BICEPS	N 2+	2+	No	No	N	N	N	No trace
I. FLEX CARPI RAD	N 1+	No	No	No	N	N	N	Simple accelerated
I. FIRST D INTEROSS	N No	No	No	No	N	N	N	Simple non accelerated
I. ABD POLL BREVIS	N No	No	No	No	N	N	N	Simple non accelerated

Consent to publish: written consent to publish has been obtained from the participant to report individual patient data.

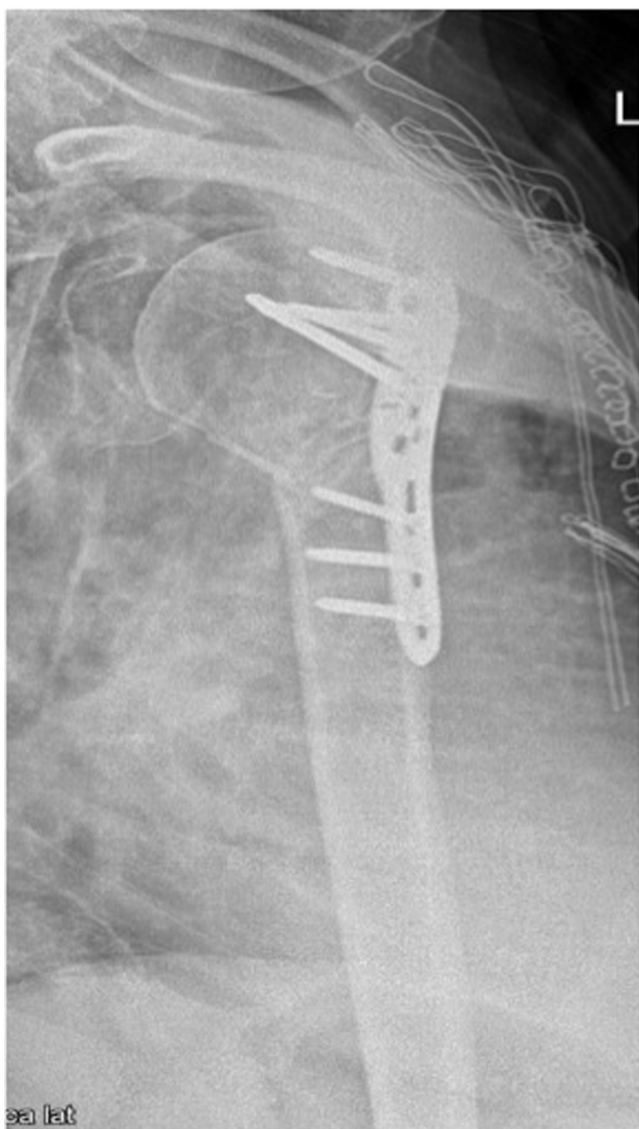


Fig. 3. Postoperative radiological imaging.

trunk, moderate partial axonotmesis of middle trunk and slight partial axonotmesis of lower trunk, in acute phase) (Table 1).

The patient began physical therapy, which included electrotherapy for muscle stimulation and kinesitherapy to preserve joint balance.

At 10 weeks, electromyographic study showed improvement of electric conduction, with signs of nerve regeneration in the abductor muscles of the shoulder, and collateral reinnervation in the upper and middle trunk territories. The patient evolved favorably with complete recovery of muscular tone at 11 months and absence

of neuropathic pain. However, she had a limited range of motion of the shoulder (maximal abduction 90°, maximal flexion 100°, active and passive) that was attributed to retractile capsulitis. Her finger flexion was also slightly limited.

The diagnosis was idiopathic partial axonotmesis of brachial plexus pre- and postganglionic (no avulsion) without associated vascular injury.

**3. Discussion**

When neurological damage appears after shoulder surgery, it may be complex and affect different types of nerve fiber and be of variable extension. It is of paramount importance to differentiate between whether the injury is central (spinal cord) or peripheral (nerve roots, plexus and peripheral nerves) [4]. It is also important to determine whether the lesion is preganglionic (proximal to the spinal ganglion) or postganglionic (distal to the foramen). Preganglionic lesions are often a consequence of nerve root avulsion and eventually cause death of spinal neurons, so no spontaneous recovery is possible (worst prognosis) [7] (Fig. 4C). However, there may be preganglionic lesions of less intensity such as, for example, radiculopathies caused by disk herniation without avulsion of nerve roots.

Postganglionic lesions may involve variable damage in accordance with the Seddon classification, and they also may be classified as supraclavicular and infraclavicular [7].

Interscalene block provides proper anesthesia for most types of shoulder surgery, including arthroplasty and fracture fixation [3]. However, the procedure is not completely safe, with a wide range of possible neurological complications, from slight brachial plexus damage [5,8] to severe spinal medullar lesions due to the needle entering the spinal canal [2]. As brachial plexus is closely related to arterial and venous systems, there may also be local anesthetic toxicity when a significant volume of local anesthetic is used, with the possibility of developing loss of consciousness and seizures [9]. Other possible complications include high spinal block, hematoma, pneumothorax, phrenic nerve palsy and respiratory distress [3].

Brachial plexus injury after interscalenic block is infrequent and may be of variable intensity [4,8]. The incidence of postoperative sensory-motor disorder of more than 6 months after isolated interscalene block has been evaluated by prospective electromyographic studies with a result of 0.2–0.4% [5,6,11,16]. However, the orthopedic literature has reported a higher incidence in patients that undergo general anesthesia without interscalene block [8,18]. Based on this epidemiologic data, a number of cases of brachial plexus palsy that are attributed to interscalene block may have a different etiology. Perioperative risk factors for peripheral nerve injury include paresthesia with needle placement, pain with injection, prolonged tourniquet time, compression or stretch related to position, sedated patient during regional block, hypothermia and prolonged hospitalization. There are also patient related factors such as diabetes, pre-existing neurologic disease, smoking, extreme body mass index, and patients being male or elderly [15].

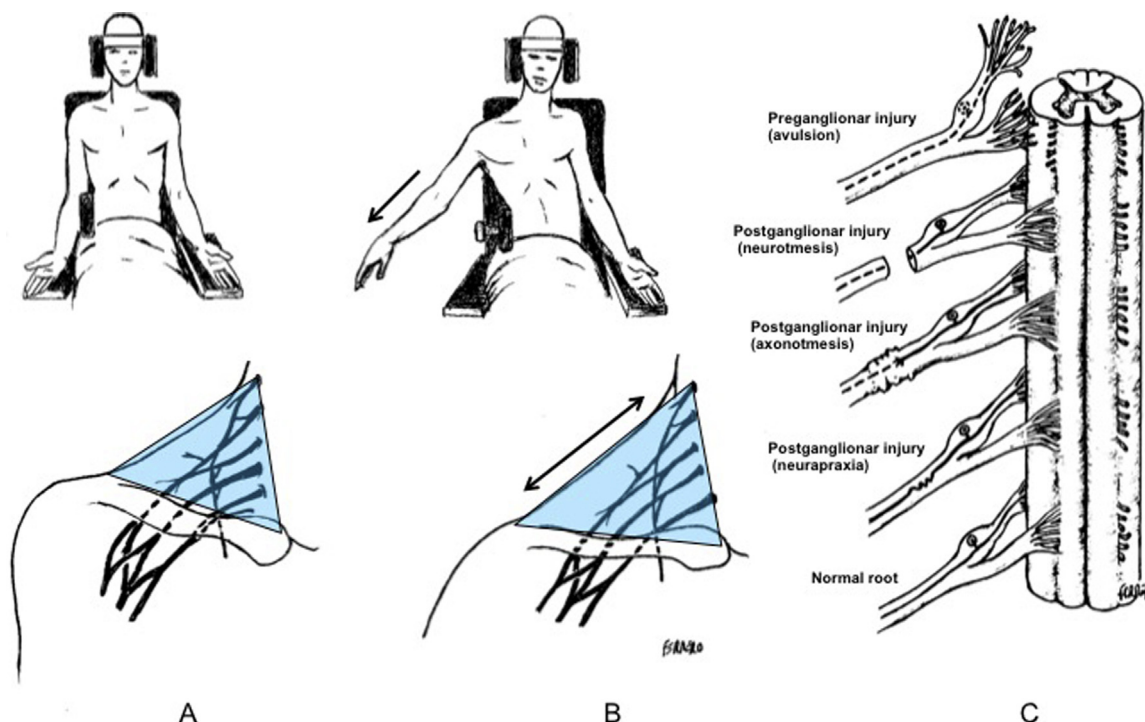


Fig. 4. schematic representation of the stretching of nerve roots when traction is applied with the head fixed at the operative time (A,B) and different degrees of nerve root injury.

The pathogenesis of brachial plexus palsy in this case remains unclear. Although the patient’s symptoms were initially attributed to the confusing anesthetic nerve block, the position of our patient’s head in extension with a slight degree of rotation may have caused traction of the brachial plexus nerve roots [1,12,13] (Fig. 4A and B). The electromyographic finding of decreasing intensity of lesion from superior to inferior nerve trunks may indicate a traction mechanism. Due to the specific anatomy of brachial plexus (triangular with its base at the spine and the vertex at the armpit), sudden stretching that increases the acromio-mastoid distance may lead to a supraclavicular plexus disorder (superior trunks). The superior nerve trunks are more strongly fixated at the spinal neural foramina than the inferior, making them more prone to postganglionic stretching, which agrees with the EMG findings [7]. Considering patient position as a possible cause of lesion, the prolonged time of surgery may have contributed to the development of plexus palsy.

The traction mechanism could have been increased by manipulation of the upper limb at the time of surgery (pulling maneuvers) as well as mobilization of the patient’s trunk. It could have been promoted by the existence of osteoarthritis at the cervical spine segment, which was assessed by x-ray. Moreover, the initial traumatism was a fracture-dislocation of the humeral head that could have caused traction of the nerve roots at that time, and therefore made it more vulnerable to injury during surgery (“double crush syndrome”) [10]. Smoking is another factor that may have contributed to nerve vulnerability. The cumulative effect of all these factors and the nerve blockade seem to be the perfect explanation for the onset of this complication.

**4. Conclusion**

When postoperative brachial plexus palsy appears, nerve block is a confusing factor that tends to be attributed as the cause of palsy by the orthopedic surgeon. The beach chair position with the head fixed may predispose brachial plexus traction injury. The head and neck position should be regularly checked during long procedures,

as intraoperative maneuvers may cause eventual traction of the brachial plexus.

**Conflict of interest**

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**Consent**

None.

**Author’s contribution**

The authors have:

- 1) Written the initial draft: FFM, RLP (orthopedic part), JRBB, AIOP (anesthetic part), RLB (electromyographic part).
- 2) Gathered the data: FFM, AIOP, RLB.
- 3) Ensured the accuracy of the data and analysis: FFM, RLP.
- 4) Conceived of the study: FFM, RLP, JRBB.
- 5) Read and approved the final manuscript: all authors.

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