Anatomical Variation Leads to Phrenic Nerve Palsy after Supraclavicular Block.

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Citation

Abstract
We are reporting a case of simultaneous diaphragmatic and brachial plexus stimulation followed by a successful nerve block using the supraclavicular approach followed by phrenic nerve paralysis. It can be due to anatomic variations of phrenic nerve.

INTRODUCTION
With the current trend in upper extremity surgery toward outpatient management, brachial plexus blocks have become valuable in providing effective anesthesia and analgesia to patient’s perioperatively. Intercalene and supraclavicular approaches have been widely studied and regarded as reliable and safe in ambulatory surgery. No major complications were identified in a review of 1001 supraclavicular blocks performed by staff and resident anesthesiologists in one study. Despite the popularity of brachial plexus blocks, the surgeon must not forget the rare, severe complications associated with regional anesthesia, including pneumothorax, neurologic injury, and vascular penetration.

Bigeleisen (2003) reported a case of simultaneous diaphragmatic and brachial plexus stimulation followed by a successful nerve block and demonstrated the necessity of a thorough knowledge of anatomical variations and standard anatomy for the safe and efficient practice for regional anesthesia. Brachial plexus block is used in surgeries of the upper limbs, both in hospitalized and outpatients. It is a good alternative for severely ill patients, who benefit from the blockade instead of general anesthesia. However, a few adverse events have been described like pneumothorax, nerve lesions, accidental intravascular injection, Horner's syndrome and phrenic nerve block.

Some authors observed that varying degrees of phrenic nerve block also occur with the supraclavicular and infraclavicular techniques with few clinical manifestations.

This article describes a case of a man who underwent a routine preoperative supraclavicular brachial plexus block and experienced acute dyspnea due to iatrogenic phrenic nerve palsy. The objective of this report was to present a case of phrenic nerve block without ventilatory compromise in a patient with hypertension posted for the implant removal.

CASE REPORT
A 67-year old male patient, 1.77 m, 76 kg, physical status ASA II, was scheduled for the implant removal from the right upper limb under supraclavicular brachial plexus block (Fig 1).
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Figure 1

Figure 1: X-rays showing implant in Right Ulna

Pre-anesthetic medication was not administered. In the operating room, venipuncture with a 20G catheter was performed in the left upper limb and normal saline (500 mL) was administered to keep the vein patent. Monitoring consisted of pulse oximetry, ECG and automatic non-invasive sphygmomanometer. The patient was sedated with intravenous fractionated doses of Midazolam (2 mg) and Fentanyl (50 µg). Oxygen (2 L.min⁻¹) was administered via a nasal cannula. The patient was calm, cooperative, with good ventilation, with SpO₂ of 98%. He was positioned for the blockade with his head rotated to the opposite side and right arm close to the body. The area was cleaned and sterile fields were placed around the area of the puncture.

The supraclavicular technique was used to approach the brachial plexus and the site of the injection was determined by the peripheral nerve stimulator (B Braun, Stimuplex, DIG RC) was set at a current of 0.5 mA, a frequency of 2 Hz and a prefixed pulse width of 0.1 ms. The patient’s head was turned toward the left and his sternocleidomastoid muscle was identified by having him lift his head off the pillow. The author’s index finger was placed posterior to the clavicular head of the sternocleidomastoid muscle and rolled laterally into the groove between the anterior and middle scalene muscles. This groove was traced distally to a position inferior to the omohyoid muscle. After identifying the appropriate landmarks, a 50 mm, 22 gauge insulated needle (B Braun, Stimuplex) was advanced in a coronal plane posterior to the subclavian artery until a motor response caused supination of the patient’s forearm. Simultaneously, the patient reported paraesthesia in his right thumb and the author noticed a motor response in the patient’s abdomen at 2 Hz.

The current was decreased to 0.2 mA. After this, the motor response in the patient’s forearm disappeared but the diaphragmatic twitch persisted. The latter was assumed to result from direct stimulation of the patient’s right phrenic nerve. It was assumed that the block needle was mistakenly placed anterior to the scalenus anticus muscle, and that the motor response in the patient’s forearm was from current leak across the plexus sheath rather than from direct stimulation of the plexus roots themselves. For this reason, the needle was removed. The landmarks in the patient’s neck were reassessed and the needle inserted several millimeters posterior to the original puncture site. The results were changed, a paraesthesia in the hand, supination of the forearm present but motor response of the diaphragm not present this time. Convinced that the plexus had been properly located, we injected 40 ml of bupivacaine (5 mg ml⁻¹) using an immobile needle technique.

Shortly after the administration of the local anesthetic was initiated, the stimulus evoked by the nerve stimulator was abolished. Five milliliters of the anesthetic solution were injected and after a 60 second waiting period the remainder of the anesthetic was injected. At the end of the administration, the patient was alert and oriented, but he complained of dyspnea and short respiratory incursion. On inspection, breathing was predominantly intercostal ipsilateral to the blockade, with retraction of the abdominal wall and tachypnea. Changes from normal to paradoxical motion of the ipsilateral hemidiaphragm were seen. On auscultation breath sounds were absent in the right base. The left hemithorax maintained the same pre-operative pattern. A hypothetic diagnosis of ipsilateral phrenic nerve block was made.

The dyspnea did not worsen and, therefore, non-invasive respiratory support (CPAP or BIPAP) or controlled mechanical ventilation was not necessary. A chest X-ray revealed the right hemidiaphragm at the level of the 6th rib, occupying the right 5th intercostal space. (Figure 2B)
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**Figure 2**

Figure 2: X-rays showing Diaphragms levels

A) Pre op B) On OT Table C) After 2hrs

Showing Preoperative Chest Xray With both diaphragms’ at same level.

Showing Chest Xray AP view with Right diaphragam at 5th Intercostal space.

Showing Chest Xray AP view with Right diaphragam at 7th Intercostal space.

The symptom eventually improved, the blockade was established and the surgery was performed without complaints of pain, discomfort or the need of supplemental analgesia. The surgery lasted approximately 75 minutes. The patient underwent an uneventful removal of implant without additional sedation or local anaesthetic.

At the end of the procedure the patient was transferred to the post-anesthesia recovery unit where there was complete remission of the dyspnea after approximately two hours, with SpO2 96% in room air. Pulmonary auscultation revealed the same preoperative pattern, with breath sounds present in the right base. The brachial plexus block was maintained and the patient did not complain of pain. The patient was kept under observation in the recovery room for three hours before being referred to the admission unit.

**DISCUSSION**

Phrenic nerve palsy is a frequent complication from both interscalene and supraclavicular brachial plexus blocks. 1, 6, 9-11

This is not surprising given that the roots of the cervical plexus are often anaesthetized with this block. The phrenic nerve, however, is also frequently anaesthetized (36–67%) when a supraclavicular block is performed. 10, 12, 13 This is surprising because the cervical roots are infrequently blocked when a supraclavicular block is performed. 13 Moreover, the quality of phrenic nerve block differs between the supraclavicular and interscalene approaches. When an interscalene block is used, there is a 100% incidence of diaphragmatic hemiparesis accompanied by a 25% reduction in forced vital capacity (FVC). 6 When a supraclavicular block is used, only 50% of patients have diaphragmatic paresis and there is no reduction in FVC in those who do. 14

The brachial plexus is formed by the ventral branches of the inferior cervical nerves, C5 to T1, with or without contributions from C4 and T2. The phrenic nerve originates from the deep cervical plexus, derived from the ventral branches of C2, C3, and C4, being located very close to the brachial plexus in the neck, in front of the anterior scalene muscle, separated from the plexus only by a thin fascia 6. Therefore, it can be reached by the diffusing anesthetic solution when the perivascular interscalene and supraclavicular approaches are used; this is not seen with the perivascular axillary techniques due to the distance between the site of injection and the phrenic nerve. Some authors admit that almost all adverse events resulting from phrenic nerve block are due to extravasation of the local anesthetic out of the musculoaponeurotic cuff or its dispersion towards the cervical plexus, which, in the cuff, is contiguous to the brachial plexus 3, 7. Thus, if the solution reaches the level between C3 and C5 it spurs the blockade. Not even digital compression above the site of the puncture prevents extravasation of the local anesthetic 10, 14.

Anatomical variations of phrenic nerve were observed on right side in the neck region of a middle aged cadaver. The phrenic nerve in its early course close to its origin was giving a communicating branch to C5 root of brachial plexus and at the level of the root of neck just before entering the thorax, the phrenic nerve was placed anterior to the subclavian vein. This unique case of phrenic nerve variation gains tremendous importance in context of subclavian vein cannulation, implanted venous access portals, and supraclavicular nerve block for regional anesthesia 15

Many variations of the phrenic nerve have been described. Rather than descending behind the subclavian vein, the phrenic nerve may also pass anterior to it. An accessory phrenic nerve may arise from roots C5 and C6 or from the nerve to the subclavius muscle. This variation is present in up to 75% of cadavers 16. The phrenic nerve may receive branches from the cervical or brachial plexus or arise entirely from the brachial plexus. Cranial nerves XI or XII may also contribute branches. These branches arise in close proximity to the site where supraclavicular block is
performed. Thus, when one considers the relatively high frequency of an accessory phrenic nerve or a branch from the brachial plexus itself, there is a significant possibility of anaesthetizing only part of the phrenic nerve with a supraclavicular block. This may lead to a partial block of the ipsilateral hemidiaphragm and is consistent with the outcomes of the study by Neal and colleagues, particularly the preservation of FVC with supraclavicular block even in those patients who have evidence of hemiparesis 14.

In reality, most healthy patients tolerate ipsilateral diaphragmatic paralysis without any symptoms. It is possible that the accessory musculature compensates the restriction imposed by the paralysis and that expansion of the contralateral lung is able to produce enough negative pressure to guarantee good ventilation. Development of dyspnea after brachial plexus block demands that other causes, such as pneumothorax, recurrent laryngeal nerve block, bronchospasm, allergic reaction, direct neurological lesion and injection in the neuro axis be ruled out. Phrenic nerve block can contribute to trigger the symptoms, respiratory effort and anxiety causing an increase in negative pressure in the upper airways but might not be the only cause of dyspnea.

The development of dyspnea is the result of the association of phrenic nerve block with other factors and despite studies that use ultrasound to evaluate diaphragmatic incursion inspiratory chest X-ray is a simple and useful exam to detect the problem and might be faster if it is available in the operating room. Ultrasound has the advantage of being able to determine the degree of diaphragmatic incursion and therefore demonstrate whether the patient has partial or total paralysis 6. Preoperative sedation with benzodiazepines does not seem to be related with the development of intraoperative dyspnea since in most cases patients seem to be cooperative and alert. Chronic obstructive pulmonary disease (COPD), such as emphysema, is an important co-factor for the development of symptoms. With the destruction of the pulmonary parenchyma, the diaphragmatic movement is important to guarantee the hematosis since a 50% loss in diaphragmatic function will result in dyspnea. In patients with a restrictive pulmonary pattern, the loss of diaphragmatic movements further impairs ventilation and might cause respiratory failure.

CONCLUSION

In summary, the author reports a case of simultaneous diaphragmatic and brachial plexus stimulation resulting in a successful nerve block. An explanation for the qualitative difference in phrenic nerve block between interscalene and supraclavicular block is postulated based on known anatomic variations. The case demonstrates the necessity of a thorough knowledge of anatomical variations and standard anatomy for the safe, efficient practice of regional anaesthesia. In this case, the patient developed complete paralysis of the phrenic nerve with respiratory symptoms. Although invasive treatment was not necessary, it is necessary to alert anesthesiologists to restrict the indication of this technique

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