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# Novel technique for reversing phrenic nerve paresis secondary to interscalene brachial plexus block

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Interscalene brachial plexus block is the reference analgesic technique for shoulder surgery. Phrenic nerve palsy with hemidiaphragmatic paresis is an established complication that results in symptomatic dyspnoea in a small number of subjects, and is poorly tolerated. Established management is supportive and assumes that, once administered, the duration of the block is unalterable. A case is presented of saline washout as a rescue measure for severe dyspnoea due to phrenic nerve palsy following interscalene brachial plexus block. To the authors' knowledge, this is the first case of this method used to reverse a single-injection brachial plexus block.

Keywords: brachial plexus block, diaphragm, local anaesthetics, phrenic nerve

# Introduction

Developments in diagnostic capability and arthroscopic techniques have triggered an increase in the number of operative shoulder procedures.<sup>1</sup> Interscalene brachial plexus block is a popular technique that has met surgical expectations for superior analgesia, patient satisfaction, early discharge and rehabilitation.<sup>2</sup> Temporary phrenic nerve palsy with ipsilateral hemidiaphragmatic paralysis is a well-recognised consequence of interscalene block. Patients with pre-existing pulmonary or neuromuscular disease may cope less well with acute changes in pulmonary mechanics than healthy counterparts. Efforts to identify techniques that preserve phrenic nerve function have produced inconsistent results.<sup>3–5</sup> and have not eliminated the complication of phrenic nerve block.

## **Case report**

Written consent for publication was obtained from the patient.

The setting was a small hospital with limited resources, specialising in elective day case procedures. A 63-year-old male (99 kg, body mass index 33 kg/m<sup>2</sup>) was scheduled for revision right shoulder arthroscopic repair. Past medical history included treated hypertension and type-II diabetes mellitus. The previous anaesthetic record detailed a one-hour surgical procedure under general anaesthesia with a laryngeal mask airway, coupled with an asleep interscalene block with 15 ml of ropivacaine 0.75%. The patient encountered rebound discomfort from the regional block wearing off after the initial surgery.

Accordingly, he requested a longer-lasting block, and preferred this performed asleep. Informed consent was obtained for general anaesthesia with asleep interscalene block. General anaesthesia was induced with propofol 240 mg, fentanyl 200 mcg and rocuronium 80 mg. Endotracheal intubation was performed with assistance of a bougie for a Cormack and Lehane grade 3 direct laryngoscopy view.

Under aseptic conditions, ultrasound guidance (Fujifilm SonoSite M-Turbo<sup>°</sup>, Bothell, WA, USA) was used to place a right interscalene block. An in-plane needle approach (Pajunk<sup>°</sup> SonoPlex 22G 50 mm, Geisingen, Germany) was used with a posterolateral to anteromedial direction through the middle scalene aponeurosis.

The needle tip was clearly visualised at all times. Following negative aspiration, 8 ml of 0.75% ropivacaine and 60 mcg of clonidine was deposited between the C5 and C6 nerve roots. Symmetrical spread was confirmed anterior and posterior to the plexus.

Surgery, lasting 2 hours, was performed in the lateral position with the operative arm in traction. No additional opioids or muscle relaxant were required intraoperatively. Full neuromuscular reversal with sugammadex 200 mg was confirmed on clinical assessment and nerve stimulation (train of four ratio > 0.9). Tracheal extubation was undertaken. On handover to post anaesthesia care unit (PACU) staff, vital signs were stable. The patient was drowsy but rousable, and comfortable from a demonstrable interscalene block.

Fifteen minutes later, an urgent review was requested in PACU. The patient was sitting upright, diaphoretic, tachypnoeic, complained of difficulty taking deep breaths, and was unable to talk in full sentences. Oxygen saturations were 96–99% via non-rebreathing facemask at a flow of 15 l/minute. Differential diagnoses included phrenic nerve block, pneumothorax, pulmonary lobar collapse, anaphylaxis, thromboembolism and laryngeal oedema secondary to tracheal intubation.

Clinical examination revealed reduced air entry at the base of the right lung. There was no wheeze, stridor or chest pain. Electrocardiogram showed sinus tachycardia. It was concluded that right phrenic nerve block with right diaphragmatic paresis was the reason for his respiratory distress. The on-call radiographer was contacted, but was off the premises and had an expected response time of 30 minutes.

There was no response to empirical treatment with an epinephrine 5 mg nebuliser. Contingency plans were made for emergency tracheal intubation. There was no intensive care facility onsite for ventilation. The decision was made to attempt rescue washout of the local anaesthetic effect with a 'sham' interscalene block using 0.9% saline.

Sonographic assessment revealed that no residual local anaesthetic reservoir remained in the interscalene groove.

Repeat injection of 30 ml of saline 0.9% was performed with ultrasound guidance, using the equipment and approach described previously. The anterior scalene aponeurosis was targeted preferentially, to ensure proximity to the phrenic nerve. A chest radiograph performed immediately post-procedure demonstrated ipsilateral raised hemidiaphragm and no pneumothorax (Figure 1), confirming the diagnosis. At 10 minutes post-procedure, there was a clear improvement in symptoms. Right basal air entry increased, the work of breathing was reduced and the patient could soon talk in full sentences. Respiratory function recovered steadily, and PACU discharge criteria were met within an hour. The patient remained comfortable and observations overnight were stable. He was discharged the following day with satisfactory analgesia on oral analgesics. At three-month follow up he was doing well with no residual problems from the block.

## Discussion

This report describes saline washout of local anaesthetic effect as a successful treatment for respiratory distress attributable to phrenic nerve paresis complicating interscalene block. Important questions are raised. What is the justification and evidence for washout of local anaesthetic effect? What is the mechanism? Why did the patient exhibit respiratory distress 15 minutes after PACU admission? Why did washout reverse phrenic nerve paresis whilst maintaining plexus analgesia? Should this approach be more widely considered as a method for managing iatrogenic harm from regional anaesthesia?

Irrespective of technique usSDed, hemidiaphragmatic paresis from phrenic nerve block is a frequent consequence of interscalene block. The close proximity of the origin of the phrenic nerve (C3-5) to the roots specifically targeted for interscalene anaesthesia (C5-6) results in an incidence of phrenic nerve involvement as high as 100%.6 Spread of injectate may be via contact of the phrenic nerve anterior with the anterior scalene muscle, proximal extension to involve the nerve roots or even via the epidural space.<sup>7</sup> In healthy individuals, hemidiaphragmatic paresis leads to a 25% reduction in forced vital capacity, yet the vast majority of patients are asymptomatic. Symptomatic patients are usually managed by providing supportive measures: reassurance, physiotherapy, semi-recumbent positioning, non-invasive and invasive ventilation are conventional strategies for managing respiratory embarrassment for the duration of the block.8-10 They do not, however, address the problem by targeting the cause directly.



Figure 1: Postoperative chest radiograph demonstrating an elevated right hemidiaphragm, consistent with phrenic nerve paresis.

The patient was in clear distress, necessitating prompt treatment. The clinical trajectory was towards emergency repeat tracheal intubation, followed by inter-hospital transfer to a facility with intensive care capability. This novel intervention averted this sequence of events and was, we believe, justified on balance of risk.

Evidence for washout of local anaesthetic is limited to case reports, suggesting it is rarely utilised and considered only in unusual circumstances. Cerebrospinal fluid lavage, where cerebrospinal fluid is aspirated and replaced with a similar volume of crystalloid, has been used to mitigate harm from medication errors. CSF lavage is recognised in oncology as an emergency measure for inadvertent subarachnoid administration of vincristine.<sup>11,12</sup> In anaesthesia, this manoeuvre has been advocated for managing intrathecal injection of potassium chloride<sup>13</sup> and excessive doses of opioids<sup>14</sup>. In neuraxial catheter techniques, saline injection modifies the spread and nature of epidural block.<sup>15,16</sup> In a volunteer study, Chan et al. reported that flushing an epidural catheter with 40 ml of saline promotes sensory and motor recovery from established epidural blockade.<sup>17</sup> More recently, the value of lavage for successfully reversing high spinal anaesthesia following inadvertent dural puncture complicating epidural anaesthesia has been described.<sup>18–20</sup> With reference to plexus blocks, Tsui and Dillane subsequently reported that reversal of unwanted diaphragmatic block with saline was a potential advantage of a continuous interscalene catheter technique.<sup>21</sup> To our knowledge, this is the first case of washout of local anaesthetic as a method to reverse a single-injection plexus block.

What is the mechanism for reversal? The technique relies on saline injection to reverse the established concentration gradients of local anaesthetic across the neuronal membrane. 'Washout' would be expected to reduce the intra-neural concentration of local anaesthetic. Once this falls below a threshold level, phrenic nerve transmission would be re-established, with recruitment of hemidiaphragmatic function and normalisation of respiratory mechanics. However, the exact mechanism for reversal is not certain and probably not so simple. Plausible variables include the location of initial local anaesthetic deposition and subsequent rescue injection, time interval between local anaesthetic and rescue, dose and type of local anaesthetic administered, dilutional volume, electrolyte composition and pH of crystalloid solution, in addition to local perfusion. Patient positioning and placebo effect may also play a role.

Why did the patient exhibit respiratory distress 15 minutes after PACU admission? It is possible that, as the patient became more lucid, there was a corresponding increase in his respiratory effort, specifically tidal volume. This would reveal the effect of phrenic nerve palsy.

In our case, despite washout, analgesia quality was satisfactory and with smooth transition to oral analgesics. The smaller size of the phrenic nerve compared with roots of the brachial plexus may make it more susceptible to washout, and account for the observed differential recovery. Though separated in time, addition of 30 ml of saline to 8 ml of 0.75% ropivacaine would be expected to result in a final concentration of 0.16%. Lowconcentration solutions, such as 0.2% ropivacaine, are used in some institutions in an attempt to provide analgesia whilst sparing phrenic nerve involvement. Opinion as to the ideal concentration is divergent since onset, efficacy, duration and the risk of adverse effects are interdependent. The fact that the patient also received clonidine suggests that the adjuvant may have conferred an analgesic quality and duration beyond that expected from low-concentration ropivacaine.

Preoperative risk appraisal is hindered by a lack of prospective research examining predictors for symptomatic phrenic nerve blockade. It is interesting to note that an interscalene block of 8 ml led to severe dyspnoea in a functionally well patient with no background of respiratory disease, especially given he had previously received an uncomplicated larger volume block (15 ml). The prior anaesthetic chart did not document the precise location of deposition of injectate or the pattern of spread. It is possible that 15 ml of injectate did not block the phrenic nerve at the time of initial surgery. Additionally, it is plausible that compensated respiratory pathology coexistent on the second occasion, but not present on the first, was unmasked by the block.<sup>22</sup>

A potential criticism of this case is that 'washout' of local anaesthetic is not substantiated by robust evidence. This technique is rarely considered for managing major complications of regional anaesthesia, since the strategy is not supported by high-level evidence. In this case, washout was performed in the absence of an available safer option to manage this iatrogenic complication at the time. The rare and emergent situations in which dilution has been undertaken to date means that high-level evidence is hard to achieve. Tsui<sup>23</sup> points to the parachute argument<sup>24</sup> as a criticism of over-reliance on evidence-based medicine, suggesting that there is a role for conceptually sound interventions based on anecdotal evidence. Though anecdotal reports are useful, formal examination of this intervention through prospective trials would be welcome.

A further potential criticism is that a post-procedural chest radiograph was not performed to demonstrate the response to saline washout. In this case, institutional constraints made imaging challenging. Staff rostering arrangements at the hospital late in the evening precluded the ongoing support of a radiographer to provide a follow-up image. During continuous assessment in PACU, there was subjective resolution of the respiratory distress experienced by the patient, normalisation of vital signs and ipsilateral lung re-expansion on serial chest examination. It was determined that there was sufficient clinical evidence for reversal of phrenic nerve blockade that an additional chest radiograph, though useful, was not essential to confirm that phrenic nerve function had been re-established by the 'sham' block.

Guidance on the management of respiratory failure from phrenic nerve paresis recommends supportive measures and, if required, tracheal re-intubation for the duration of the regional block. Prospective research is required to support widespread adoption of our approach. A trial is under way evaluating whether deliberate saline injection around the phrenic nerve has a protective effect in reducing the incidence of phrenic nerve palsy.<sup>25</sup>

Can phrenic nerve block be avoided in the first place? Technical modifications to interscalene approaches, including ultrasound guidance, reducing volume and/or dose of local anaesthetic, targeting lower nerve roots, or injection posterior to the plexus rather than anterior, have sought to reduce the incidence of phrenic nerve involvement. More distal approaches have been investigated for shoulder surgery, such as discrete axillary and suprascapular nerve blocks. Though these show promise, the burden of proof remains on these alternatives to demonstrate non-inferior analgesia compared with interscalene block, whilst avoiding associated complications. There is currently insufficient evidence to suggest a shift in practice away from interscalene approaches towards these alternatives.

## Conclusion

Our case details saline washout as a rescue technique to successfully reverse symptomatic phrenic nerve paresis complicating interscalene block. This challenges the notion that, once administered, the duration of single-shot blocks cannot be modified. More trials are warranted to evaluate this further.

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#### **Prior presentation**

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