# Decreasing the Local Anesthetic Volume From 20 to 10 mL for Ultrasound-Guided Interscalene Block at the Cricoid Level Does Not Reduce the Incidence of Hemidiaphragmatic Paresis

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**Background and Objectives:** This prospective, randomized, double-blind study was designed to determine whether reduction in volume from 20 to 10 mL of ropivacaine 0.5% for ultrasound-guided interscalene block might decrease the incidence of diaphragmatic paresis and preserve pulmonary function.

**Method:** Thirty patients scheduled for arthroscopic shoulder surgery were randomized to receive either 10 or 20 mL of ropivacaine 0.5% for interscalene block at the level of the cricoid cartilage. General anesthesia was administered for surgery, and the surgeon infiltrated lidocaine at the port sites. Hemidiaphragmatic excursion and pulmonary function tests were measured before block, 15 mins after block, and at the time of discharge from recovery room. Onset and duration of sensory dermatomal spread, motor block, pain scores, and analgesic consumption were recorded.

**Results:** Hemidiaphragmatic paresis occurred 15 mins after block performance in 14 of 15 patients in each group. At postanesthesia care unit discharge, 13 of 15 patients in each group continued to demonstrate hemidiaphragmatic paresis. Significant reduction of spirometric values (forced vital capacity, forced expiratory volume at 1 sec, and peak expiratory flow) occurred to a similar degree in both groups after block. Sensory dermatomal spread, motor block, pain scores, and analgesic consumption were not significantly different between groups.

**Conclusions:** Decreasing the volume for interscalene block from 20 to 10 mL did not reduce the incidence of hemidiaphragmatic paresis or impairment in pulmonary function, which persisted at discharge from recovery room. No significant differences in quality or duration of analgesia were observed.

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nterscalene block (ISB) results in 100% ipsilateral phrenic nerve block, leading to impaired hemidiaphragmatic motion and decreased pulmonary function. The cause of phrenic nerve block after ISB includes cephalad spread of local anesthetic

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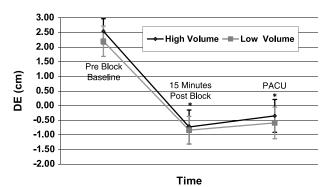
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(LA) solution to the cervical roots or spread of LA across the surface of the anterior scalene muscle over which the phrenic nerve courses. Attempts to prevent phrenic nerve block have included ISB with lower volumes of LA solution and application of digital pressure to prevent cephalad spread of LA solution or both.<sup>2,3</sup> None of these techniques have prevented the phrenic nerve from being blocked. Riazi et al<sup>4</sup> evaluated ultrasound-guided ISB (USISB) with 5 mL of ropivacaine 0.5% and found that, in 55% of the patients, phrenic nerve block did not occur.

This study was designed to compare the incidence of hemidiaphragmatic paresis using a low volume (10 mL) versus a normal volume (20 mL) of ropivacaine for USISB performed at the level of the cricoid cartilage. In addition, pulmonary function tests (PFTs) and hemidiaphragmatic paresis were evaluated at the time of discharge from the post anesthesia care unit (PACU). In this study, we hypothesized that reducing the volume of ropivacaine 0.5% from 20 to 10 mL may decrease the incidence of hemidiaphragmatic paresis. Block characteristics and postoperative analgesia was also evaluated.

## **METHODS**

This study was approved by the institutional review board of Saint Francis Hospital and Medical Center, and written informed consent was obtained from all subjects. Thirty patients scheduled for outpatient arthroscopic shoulder surgery under general anesthesia (GA) with ISB for postoperative analgesia were studied. Exclusion criteria included age younger than 18 years or older than 70 years, allergy to any medications used in the study, chronic lung disease, pregnancy, neuropathy, neurologic disease, and diabetes. The patients were randomized by sealed envelopes either to a low- (L) or to high- (H) volume group. Baseline PFTs including forced vital capacity (FVC), forced expiratory volume at 1 sec (FEV1), and peak expiratory flow rate (PEF) were recorded using a hand-held spirometer (SpiroPro; Viasys Healthcare, Inc, Conshohocken, Pa). Spirometry was performed 3 times, and best effort was recorded. The primary end point of hemidiaphragmatic paresis was determined as follows. Preblock hemidiaphragmatic excursion (DE) with a forceful sniff was measured using a 5-2 MHz 60-mm broadband curved array transducer (C60; Sonosite, Inc, Bothell, Wash) and M-mode ultrasonography in the anterior axillary line at the upper margin of the zone of apposition of the diaphragm to the thoracic wall as described by Urmey et al.1 Normal caudad motion of the diaphragm with inspiration was designated as positive (+) and cephalad, paradoxical motion as negative (-). The zero line was the diaphragmatic position at the end of a tidal breath. With routine monitors in place, patients were lightly sedated, and supplemental oxygen was administered by nasal cannula. The maximum sedation administered was midazolam 2 mg and fentanyl 100 µg, and verbal

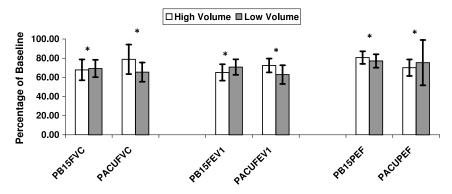


**FIGURE 1.** Hemidiaphragmatic excursion with forceful sniff. Ipsilateral diaphragmatic excursion (DE) in high-volume (20 mL) and low-volume (10 mL) group measured in centimeters, before block (baseline), 15 mins after block, and at the time of discharge from post anesthesia care unit (PACU). Normal caudad motion with sniff is (+) and paradoxical cephalad motion with sniff is (-). Data are shown as mean with 95% CI. Hemidiaphragmatic excursion shows significant difference (\*P < 0.001) after block and in PACU from baseline values but no difference between groups.

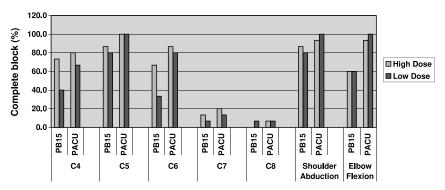
contact was maintained with the patient throughout the block procedure. Under sterile conditions, an in-plane USISB was performed with a 22-gauge, 50-mm insulated short bevel needle (StimuQuick, Arrow, Pa) at the level of the cricoid cartilage. A short-axis view of the sternocleidomastoid, scalene muscles, and brachial plexus nerves was first obtained with a 13-6 MHz linear transducer (HFL38; Sonosite). The block needle was inserted in plane from posterior to the probe, with the needle tip positioned between the 2 most lateral hypoechoic nerve structures of the brachial plexus between the anterior and middle scalene muscles (likely the C<sub>5</sub>-C<sub>6</sub> roots or the upper trunk-C<sub>7</sub> root). Nerve stimulation was used to confirm that the needle was adjacent to brachial plexus elements, but motor response was not recorded or optimized to any predetermined current. The LA was injected incrementally after negative aspiration without needle repositioning, and patients received either 10 mL of ropivacaine 0.5% with epinephrine 1:400,000 in the low-volume group (L) or 20 mL of ropivacaine 0.5% with epinephrine 1:400,000 in the highvolume group (H). The end of ropivacaine injection was denoted time 0, and the secondary end point of onset of sensory (pinprick with 21-gauge needle) and upper trunk motor blockade was assessed at 5, 10, and 15 mins using a 3-point scale (0 = no block – normal sensation to pin prick, 1 = partial block – touch without sharpness to pin prick, 2 = complete block - absence of sensation to pin prick). Sensory block was evaluated in the  $C_4$  to  $C_8$  dermatomes ( $C_4 = \text{top of shoulder}$ ,  $C_5 = \text{skin over the deltoid muscle}$ ,  $C_6 = \text{tip of thumb}$ ,  $C_7 = \text{tip of middle finger}$ , and  $C_8 = \text{tip of little finger}$ ). Motor block was evaluated by determining patients' ability to abduct the shoulder and flex the elbow against gravity (0 = no block - full strength, 1 = partial block - weak but able to abduct or flex against gravity, 2 = complete block - no activity of muscle group). Assessment of PFT and DE was repeated 15 mins after block placement and at PACU discharge. The sensory dermatomal spread, motor block, PFT, and DE testing were performed by an investigator blinded to study group.

All patients received GA for surgery. Anesthesia was induced with propofol, intubation was facilitated by rocuronium, and the anesthetic was maintained with desflurane, oxygen, and nitrous oxide. Fentanyl was the only opioid allowed without any specific limitation. The surgeon infiltrated all the portal sites used for accessing the shoulder joint with arthroscopic instruments with a total of 20 mL of lidocaine 1% with epinephrine 1:200,000. Neuromuscular blockade was reversed, if necessary, before extubation and transport to the PACU. Patient's numeric rating scale (NRS) pain score (0–10) and fentanyl used in the PACU for pain scores greater than 4 were recorded. Before discharge from the PACU, patient's motor and sensory block, PFT, and DE were reassessed by a blinded investigator. Patients were discharged with a prescription for either Vicodin (Abbott Laboratories; hydrocodone 5 mg/acetaminophen 500 mg) or Percocet (Endo Pharmaceutical, Inc; oxycodone 5 mg/acetaminophen 325 mg) based on the surgeon's preference. A PACU nurse, also unaware of group assignment, contacted the patient on postoperative day 1 and recorded secondary end points of block duration (defined as resolution of numbness, or the onset of pain in the shoulder, whichever occurred first), and home analgesic consumption since discharge. (Number of tablets converted to mg of hydrocodone equivalent and acetaminophen.)

Sample size calculations were based on the expected difference in proportion of patients who developed paradoxical hemidiaphragmatic motion after ISB in patients receiving a high and a low dose of LA solution. Al-Kaisy et al<sup>7</sup> showed that, by reducing the dose of bupivacaine by 50% for ISB, the risk of developing hemidiaphragmatic paresis decreased from 80% to 17%. With a 2-tailed  $\alpha$  of 0.05, a sample of 30 patients (15 per group) was associated with a power of 97% to detect a difference of this magnitude (Z test with pooled variance). Statistical significance of differences in high-dose versus low-dose groups with respect to nominal variables, including sensory block were



**FIGURE 2.** Pulmonary function tests as a percentage of preblock baseline values. Values of forced vital capacity (FVC), forced expiratory volume at 1 sec (FEV1) and peak expiratory flow (PEF) achieved by patients in high-volume (20 mL) and low-volume (10 mL) group with respect to baseline (preblock) expressed as percentage with 95% CI, 15 mins after block (PB15), and in PACU. \*P < 0.001 compared with baseline values but no significant difference between groups.



**FIGURE 3.** Sensory dermatomal spread and motor block. Percentage of patients with complete (score 2) sensory block by dermatome, and complete (score 2) motor block by muscle group, in high-volume (20 mL) and low-volume (10 mL) groups 15 mins after block (PB15) and at the time of discharge from PACU.

analyzed with  $\chi^2$  or Fisher exact test, as appropriate. For non-parametric variables (analgesic consumption), the Mann-Whitney U test was applied. Mean differences in continuous, normally distributed variables comparing these groups, were explored with Student t tests. P values less than 0.05 were considered statistically significant, except for a set of 6 paired t tests used to assess significance of changes from baseline to postblock lung function data. Using the Bonferroni correction, the significance criterion was set at 0.008 for these comparisons.

#### RESULTS

Hemidiaphragmatic paresis occurred in 93% of patients in each group 15 mins after block. Before block, all patients had normal (caudad, positive) diaphragmatic excursion of +2.5 cm (95% confidence interval [CI], 2.1-2.9 cm) in group H and +2.2 cm (95% CI, 1.7-2.7 cm) in group L (Fig. 1). Fifteen minutes after block, 14 of 15 patients in each group developed paradoxical (cephalad, negative) DE with a mean of -0.7 cm (95% CI, −1.3 to −0.1 cm; group H) and −0.9 cm (95% CI, -1.3 to -0.3 cm; group L). At the time of discharge from PACU, all but 2 patients in each group continued to demonstrate paradoxical diaphragmatic motion with a mean of -0.4 cm (95% CI, −0.09 to −0.2 cm; group H) and −0.6 cm (95% CI, -1.1 to -0.05 cm; group L). Spirometric measurements showed significant and equivalent reduction in FVC, FEV1, and PEF in both groups 15 mins after block, which persisted until PACU discharge (Fig. 2; P < 0.001). Compared with preblock baseline, the postblock FVC, FEV1, and PEF were 68% (95% CI, 57%-79%; group H) and 69% (95% CI, 60%-78%; group L), 65% (95% CI, 56%–74%; group H) and 71% (95% CI, 63%–79%; group L), and 81% (95% CI, 74%–87%; group H) and 77% (95% CI, 70%–84%; group L), respectively.

There were no significant differences in the demographic variables between the groups (Table 1). The extent of sensory and motor block was not significantly different between the groups (Fig. 3). At the time of PACU discharge, all patients had complete sensory block in C<sub>5</sub> dermatome, whereas 93% of patients in group H and 80% of patients in group L had complete block in C<sub>6</sub> distribution (not statistically significant). Although the percentage of patients with complete sensory block in C<sub>4</sub> dermatome was lower in group L 15 mins after block placement (40% versus 73.3%) and in PACU (66.7% versus 73.3%), the difference did not reach statistical significance. No patient received more than 100 μg of fentanyl for block placement and intraoperative use. Only 1 patient (group H) received 50 µg of fentanyl in PACU for pain. The time from block performance to discharge from PACU was similar in both groups. There were no differences in the duration of block 777 ± 120 min (H), 745 ± 173 min (L), and home analgesic consumption between the groups.

## **DISCUSSION**

This study demonstrates that reducing the volume of ropivacaine 0.5% from 20 to 10 mL for USISB at the level of the cricoid does not reduce the incidence of hemidiaphragmatic paresis. The decrease in the measured PFT variables (FVC, FEV1, and PEF) was not statistically different between the high-volume and low-volume groups and was consistent with the reduction in PFT values reported by others. Importantly, the impaired diaphragmatic function and PFT persisted at discharge of these patients from the recovery room  $236.6 \pm 50.5 \, \text{mins}$  (group L) and

**TABLE 1.** Patient Characteristics

	High-Volume Group (H)	Low-Volume Group (L)	P
No. patients	15	15	
Age, mean $\pm$ SD, y	$49 \pm 9.7$	$50 \pm 12.6$	NS
Weight, mean ± SD, kg	$84 \pm 21.1$	$81 \pm 16.5$	NS
Height, mean ± SD, cm	$173 \pm 10.9$	$172 \pm 8.5$	NS
Sex (M/F)	5/10	5/10	NS
Time from block to PACU discharge, mean ± SD, min	$246.0 \pm 54.4$	$236.6 \pm 50.5$	0.76
Block duration, mean ± SD, min	$777.1 \pm 120.5$	$744.9 \pm 173.2$	0.56
Fentanyl PACU-median (min-max), µg	0 (0–0)	0 (0-50)	0.78
Hydrocodone home, median (min-max), mg	25 (5–64)	30 (5–64)	0.94
Acetaminophen home, median (min-max), mg	975 (0–3750)	1000 (0-3750)	0.97

 $246 \pm 54.4$  mins (group H) after the block. Our study reaffirmed that reduction of volume of LA did not significantly reduce the quality or duration of postoperative analgesia after shoulder surgery with ISB.

Interventions to avoid phrenic nerve block while performing USISB block have included decreasing the volume of LA. An ultra-low-volume study of USISB performed using ropivacaine 0.5% 5 mL resulted in 45% of patients having hemidiaphragmatic paresis.<sup>4</sup> This is lower than our results using a similar technique with a larger volume of 0.5% ropivacaine 10 mL in which 93% of patients showed hemidiaphragmatic paresis. Local anesthetic volumes as low as 3 mL for ISB have been implicated in causing respiratory distress from concurrent phrenic nerve block. 10 However, ISB has also been performed on patients bilaterally, and in a patient with a previous contralateral thoracotomy in which a phrenic nerve block could have potentially compromised pulmonary function but did not result in any respiratory distress. 11-13 The inability to predict which patients will develop hemidiaphragmatic paresis or who may have earlier recovery of diaphragmatic function after using a low-volume of LA solution is an important consideration when performing ISB in patients with preexisting lung disease.

Injection of LA farther from the C<sub>3-5</sub> roots has been another strategy proposed to reduce phrenic block. Renes et al<sup>14</sup> demonstrated that USISB performed at the root of C<sub>7</sub> with ropivacaine 0.75% 10 mL resulted in the preservation of diaphragmatic function in 93% of patients. This approach requires identification and approximation of the needle tip to the posterior tubercle of the C<sub>7</sub> transverse process, closer to the vertebral vessels than the technique described for USISB at the cricoid level. Injection of LA at this site may decrease the incidence of hemidiaphragmatic paresis because the injection site is farther away from the upper cervical roots and also farther from the course of the phrenic nerve on the ventral surface of anterior scalene muscle. Kessler et al15 used ultrasound to identify the phrenic nerve and measure the increasing distance from the phrenic nerve to the C<sub>5</sub> root as the transducer was moved caudad in the neck. At the cricoid level, the mean distance was 1.8 mm, whereas 3 cm caudad, the mean distance was 10.8 mm. The authors suggested that phrenic block was independent of LA volume at the cricoid level because of the proximity of the phrenic nerve to the C5 root, and this is consistent with our results.

The extent of sensory dermatomal spread showed some delay in the spread to the C4 and C6 dermatomes at 15 mins in the low-volume group, but no significant difference in the ultimate sensory or motor block in the PACU. In addition, the duration of analgesia and analgesic consumption was not statistically different in the low-volume group, consistent with other studies showing satisfactory analgesia after ISB for shoulder surgery with even 5 mL of long-acting LA.4 Of interest, the percentage of patients with complete sensory block of C4 in the PACU (73% in group H and 67% in group L) was lower than the percentage of patients with hemidiaphragmatic paresis in the PACU (87% in each group). This finding, coupled with the studies by Kessler et al and Renes et al, suggests that hemidiaphragmatic paresis after ISB is less likely due to direct blockade of the C4 root and more likely due to spread of LA solution to the phrenic nerve on the anterior scalene muscle. The actual spread of LA over the anterior scalene was not assessed, however, nor was there any attempt to identify the phrenic nerve in this study.

A limitation of this study is that patients were not followed until resolution of their phrenic nerve block to ascertain the duration of pulmonary dysfunction or whether the analgesia persisted beyond hemidiaphragmatic dysfunction. Because patients were all recruited from an ambulatory surgical population with discharge to home within a few hours of surgery, it was considered impractical to keep them hospitalized until full diaphragmatic recovery. Another limitation of the study is that lidocaine was injected by the orthopedic surgeons, at portal sites used for arthroscopic instruments, which could potentially mask an insufficient USISB and improve NRS in the PACU. The short duration of lidocaine should not, however, have influenced analgesic consumption after discharge nor would it have affected the primary end point of hemidiaphragmatic paresis.

In summary, this study shows that reducing the volume of LA for USISB performed at the level of the cricoid cartilage from 20 to 10 mL of ropivacaine 0.5% with 1:400,000 epinephrine produced equivalent analgesia after shoulder surgery, but hemidiaphragmatic paresis occurred in 93% of patients.

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