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Phrenic Nerve Palsy and Regional Anesthesia for Shoulder Surgery

Anatomical, Physiologic, and Clinical Considerations

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ABSTRACT

Regional anesthesia has an established role in providing perioperative analgesia for shoulder surgery. However, phrenic nerve palsy is a significant complication that potentially limits the use of regional anesthesia, particularly in high-risk patients. The authors describe the anatomical, physiologic, and clinical principles relevant to phrenic nerve palsy in this context. They also present a comprehensive review of the strategies for reducing phrenic nerve palsy and its clinical impact while ensuring adequate analgesia for shoulder surgery. The most important of these include limiting local anesthetic dose and injection volume and performing the injection further away from the C5–C6 nerve roots. Targeting peripheral nerves supplying the shoulder, such as the suprascapular and axillary nerves, may be an effective alternative to brachial plexus blockade in selected patients. The optimal regional anesthetic approach in shoulder surgery should be tailored to individual patients based on comorbidities, type of surgery, and the principles described in this article. **(Anesthesiology 2017; 127:173-91)**

SURGERY for shoulder pathology is increasingly common,[1,2] with regional anesthesia playing an important role in multimodal analgesia for these painful procedures.[3] Interscalene brachial plexus block is the most common regional anesthetic technique; however, phrenic nerve palsy and hemidiaphragmatic paresis have traditionally been inevitable consequences, which limit its utility in the population of patients at high risk of respiratory complications. A range of modifications and alternatives to interscalene block have been proposed to minimize the respiratory impact of phrenic nerve palsy, but to date there has been no thorough assessment of the clinical value offered by each of these strategies. In this article, we aim to describe the anatomical, physiologic, and clinical principles governing phrenic nerve palsy in the context of regional anesthesia for shoulder surgery. We also review the various techniques that seek to provide adequate regional anesthesia of the shoulder while minimizing the risk of phrenic nerve palsy, as well as methods for assessing their impact on diaphragmatic function, and thus provide a comprehensive narrative of their value in achieving these two objectives.

Materials and Methods

For this narrative review, we systematically searched electronic databases including MEDLINE, PubMed-not-MEDLINE, Excerpta Medica database (Embase), Cochrane Central Controlled Trials Database Register, and Cumulative Index to Nursing and Allied Health Literature (CINAHL), supplemented by a manual search. Search terms in medical subject headings, text words, and controlled vocabulary terms were used in permutations relevant to the components of this review. Search terms included (1) regional anesthesia; (2) local anesthesia; (3) shoulder; (4) surgery; (5) phrenic; (6) nerve; (7) diaphragm; and (8) diaphragmatic. Filters applied included (1) publication date January 1, 1946, to November 1, 2016; (2) English language; (3) human studies; and (4) adult studies. Eligible trials included randomized or quasirandomized controlled trials, controlled trials, case series, or pertinent correspondence that were deemed relevant or providing new knowledge on the subject in question. Trials were excluded if they produced no original empirical data, or if they were not directly relevant to phrenic nerve palsy related to regional anesthesia for shoulder surgery (fig. 1).

Studies were supplemented qualitatively with an informal literature search for relevant articles describing anatomical, physiologic, clinical, and diagnostic concepts so as to provide a comprehensive insight into the subject.

This article is featured in "This Month in Anesthesiology," page 1A. Figure 1 was enhanced by Annemarie B. Johnson, C.M.I., Medical Illustrator, Vivo Visuals, Winston-Salem, North Carolina.

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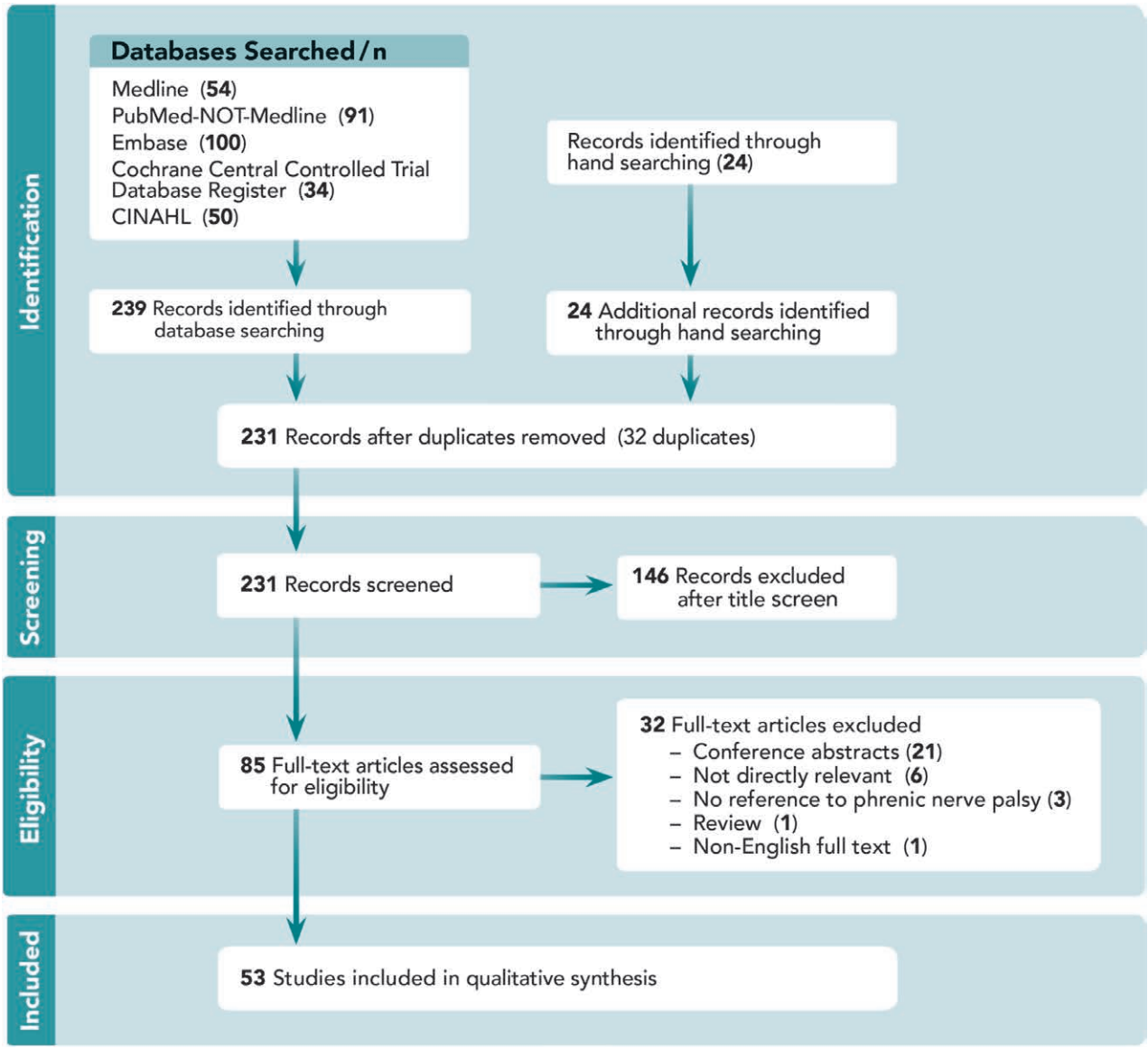

Fig. 1. Flowchart of study selection. CINAHL = Cumulative Index to Nursing and Allied Health Literature.

Discussion

Regional Anesthesia Targets for Shoulder Analgesia

Innervation of the cutaneous, muscular, bony, and capsular components of the shoulder is complex. Cutaneous innervation is provided by the axillary (C5–C6), suprascapular nerve (C5–C6), and supraclavicular nerves of the cervical plexus (C3–C4). Bony and capsular components are innervated by the suprascapular, axillary, lateral pectoral (C5–C7), musculocutaneous (C5–C7), and long thoracic (C5–C7) nerves (fig. 2). The suprascapular nerve provides up to 70% of the innervation to the glenohumeral joint,[4] with the axillary nerve supplying the majority of the remaining joint capsule. Sensory contributions to the muscles of the shoulder comprise the following: the ventral rami of the third and fourth cervical nerves to the trapezius muscle, the pectoral nerves (C5–C7) to the pectoral muscles, the dorsal scapular nerve (C5) to the levator scapulae and rhomboid muscles, and the axillary nerve (C5–C6) to the deltoid muscle. The rotator cuff muscles are innervated by the suprascapular, upper and lower subscapular (C5–C6), and axillary nerves.

The clinical aim of regional anesthesia or analgesia is to deliver local anesthetic to some or all of these key nerves that contribute to pain after shoulder surgery. The specific nerves to be targeted will depend in part on the surgical approach that is used (fig. 2). This traditionally has been achieved by performing an interscalene block, which targets the C5 and C6 roots of the brachial plexus in the interscalene region. However, conventional interscalene block is associated with several complications, the most common of which is phrenic nerve palsy with ensuing hemidiaphragmatic paresis, and this has driven the development of modifications to the interscalene block as well as alternative techniques that target the peripheral sensory supply to the shoulder at sites distal to the C5 and C6 roots.

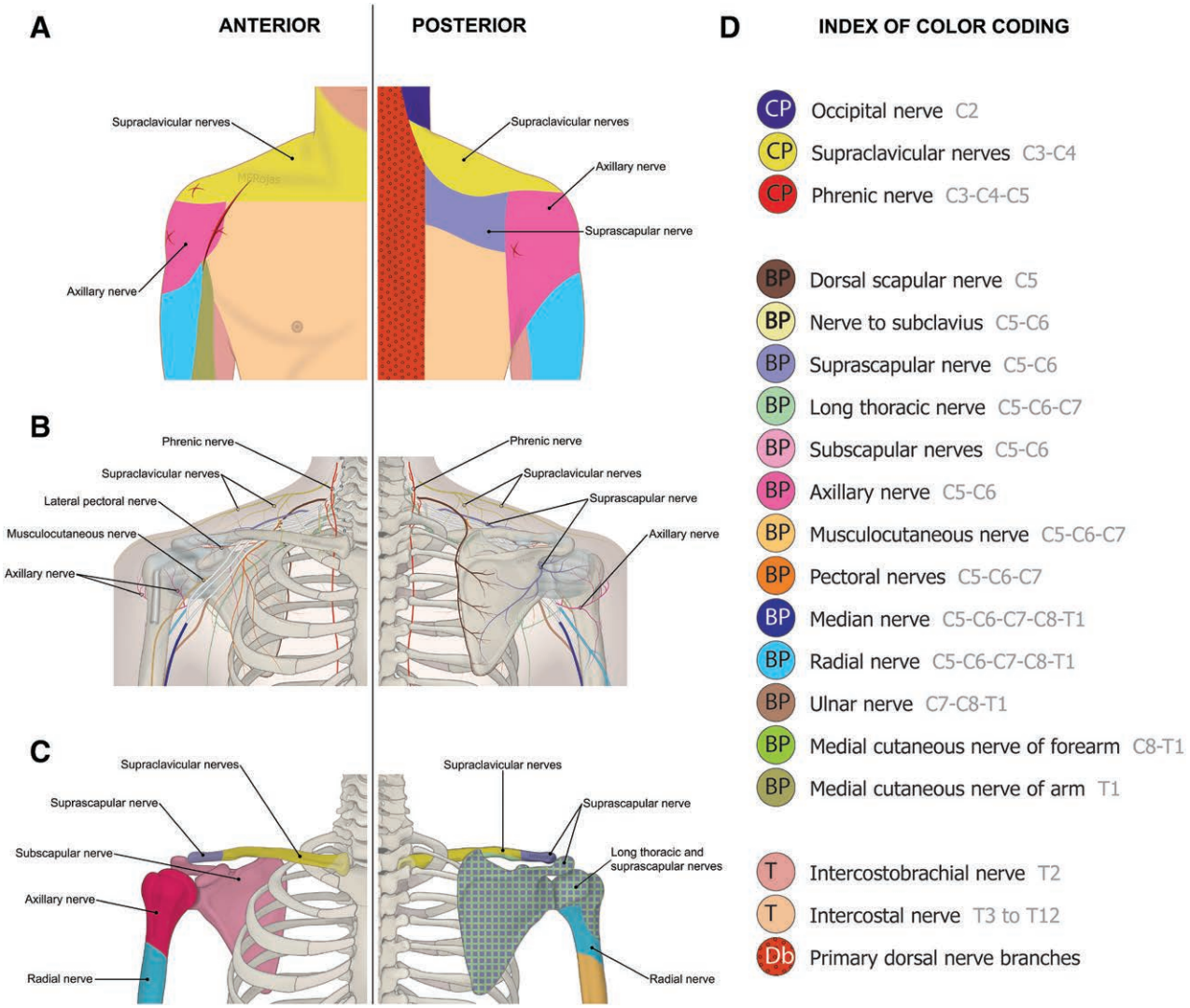

Fig. 2. Anterior (*left*) and posterior (*right*) innervation of the shoulder. (*A*) The distribution of cutaneous innervation of the shoulder. Common port-hole incisions (*red crosses*), including superior, anterior, lateral, and posterior incisions made for arthroscopic shoulder surgery and the deltopectoral incision (*red line*) for open shoulder surgery are represented. (*B*) The route taken by nerves to supply both skin and bone in the shoulder. (*C*) The osteotomal supply of the shoulder. (*D*) An index of color coding of dermatomes, nerves, and osteotomes. Images adapted with permission from Maria Fernanda Rojas Gomez and reproduced with permission from Ultrasound for Regional Anesthesia (USRA; http://www.usra.ca).

Anatomy of the Phrenic Nerve

The anatomy of the phrenic nerve is key to understanding the basis for the strategies to reduce the risk of phrenic nerve palsy. The phrenic nerve originates primarily from the fourth cervical ventral ramus but also receives contributions from both third and fifth ventral rami, as well as the cervical sympathetic ganglia or thoracic sympathetic plexus.[5] This small nerve forms at the upper lateral border of the anterior scalene muscle and descends obliquely across the anterior surface of the muscle toward its medial border (fig. 3). The phrenic nerve lies deep to the prevertebral fascia here and remains posterior to the sternocleidomastoid muscle, the inferior belly of the omohyoid, the internal jugular vein, the dorsal scapular and transverse cervical arteries, and the thoracic duct on the left. The phrenic nerve courses in close proximity to the brachial plexus, initially lying 18 to 20 mm medial to the C5 nerve root at the level of the cricoid cartilage (C5/C6) but diverging an additional 3 mm further away for every centimeter that it descends over the anterior scalene muscle (fig. 3).[6] As it approaches the root of the neck, the phrenic nerve usually lies between the subclavian artery and vein, before coursing medially in front of the internal thoracic artery (fig. 4).

An accessory phrenic nerve is present in 60 to 75% of individuals and provides an independent contribution to the phrenic nerve. The fibers of the accessory phrenic nerve arise primarily from C5 and run within the nerve to subclavius, the ansa cervicalis, or the nerve to sternohyoid.[7] These fibers then emerge from any one of these nerves to form the accessory phrenic nerve, which then joins the phrenic nerve

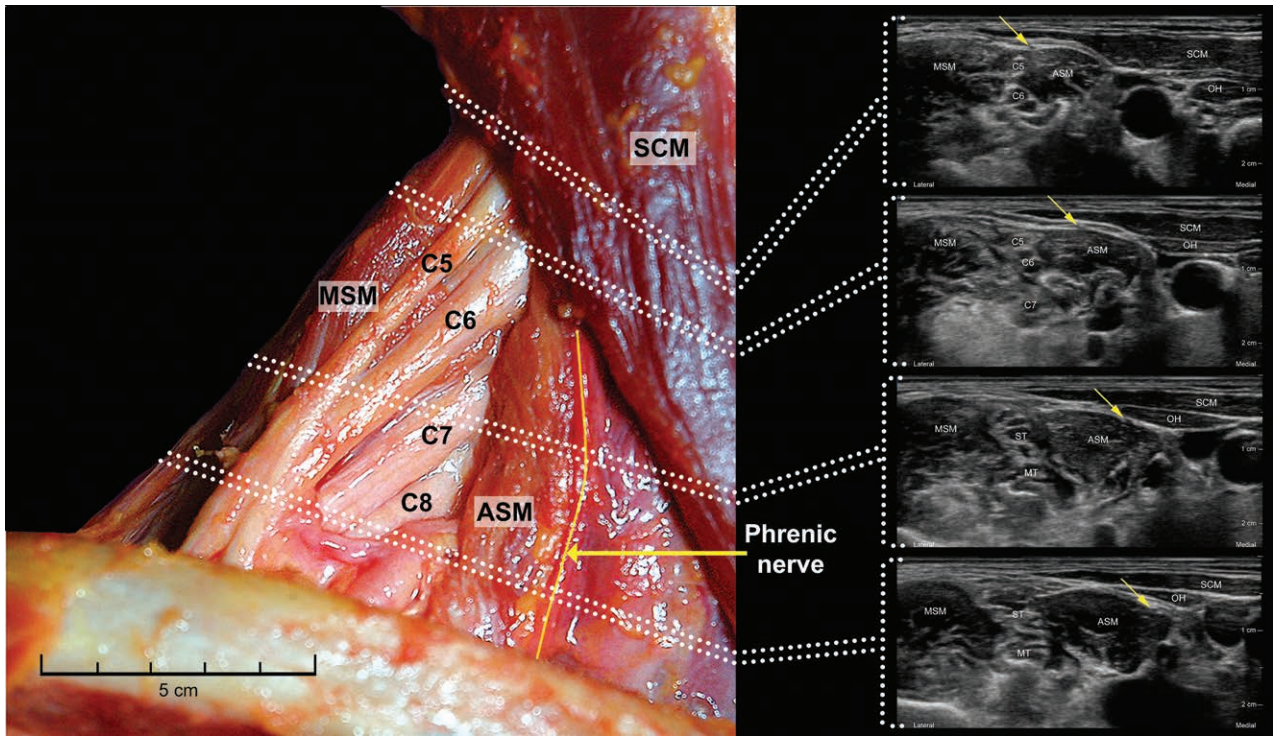

Fig. 3. Cadaveric (*left*) and corresponding sonographic images (*right*) demonstrating the course of the right phrenic nerve as it emerges beneath the lateral margin of the sternocleidomastoid muscle (SCM), between the middle scalene muscle (MSM) and the anterior scalene muscle (ASM). It begins near to the roots of the brachial plexus, then travels inferomedially away from the brachial plexus. The C5–C7 roots of the brachial plexus emerge deep to the ASM, coursing laterally, where C5 and C6 roots merge to form the superior trunk. The sonographic images of the right interscalene area descending sequentially caudally, with the brachial plexus found between the MSM and the ASM. In the *upper image*, the phrenic nerve (*yellow arrow*) can be seen above (superficial to) the ASM in close proximity to the C5 nerve root. More caudally, the phrenic nerve (*yellow arrow*) can be seen to travel medially over the ASM and beneath the omohyoid (OH) muscle until it lies nearly 2 cm away from the brachial plexus. MT = middle trunk; ST = superior trunk. *Left image* adapted with permission from Danilo Jankovic and reproduced with permission from Ultrasound for Regional Anesthesia (USRA; http://www.usra.ca).

at a variable location along its course.[8,9] Isolated damage to the accessory phrenic nerve is associated with diaphragmatic dysfunction,[10] and similarly, reports suggest that local anesthetic blockade of the accessory nerve also may lead to diaphragmatic paresis.[11,12]

Mechanisms of Phrenic Nerve Palsy after Regional Anesthesia

Transient Phrenic Nerve Palsy. Phrenic nerve palsy leading to hemidiaphragmatic paresis may be a temporary or persistent phenomenon after interscalene block or other injections of local anesthetic in the neck. Transient phrenic nerve palsy is caused by local anesthetic spreading directly to the phrenic nerve and its contributing nerves (including the accessory phrenic nerve) or proximally to the roots of the phrenic nerve. The duration of phrenic nerve palsy is determined by the duration of local anesthetic effect, which in turn is related primarily to the type and mass of local anesthetic administered. The incidence of transient phrenic nerve palsy is virtually 100% after landmark- and paresthesia-guided interscalene block techniques that use a large-volume injection of 20 ml or greater.[13,14]

Despite this, the vast majority of patients in clinical trials of interscalene block exhibit few symptoms and require no specific treatment.[15–17] Thus, on the surface, transient phrenic nerve palsy appears to have little clinical significance in terms of both objective (respiratory support) and subjective (dyspnea) features. However, randomized controlled trials generally exclude patients with pulmonary disease, obesity, or obstructive sleep apnea, and this therefore hinders the generalizability of the results reported in the literature. A significant proportion of these subgroups of patients are likely to develop symptoms or require treatment after phrenic nerve palsy, but unfortunately data on these high-risk populations usually are confined to the realm of case reports.

There is also a lack of studies formally examining clinical predictors of symptomatic phrenic nerve palsy after interscalene block, and thus it remains difficult to determine which patients, healthy or otherwise, will benefit most from avoidance of phrenic nerve palsy. It therefore falls to the individual anesthesiologist to assess the likely impact of phrenic nerve palsy in any given patient undergoing shoulder surgery and to select the appropriate regional anesthetic technique accordingly.

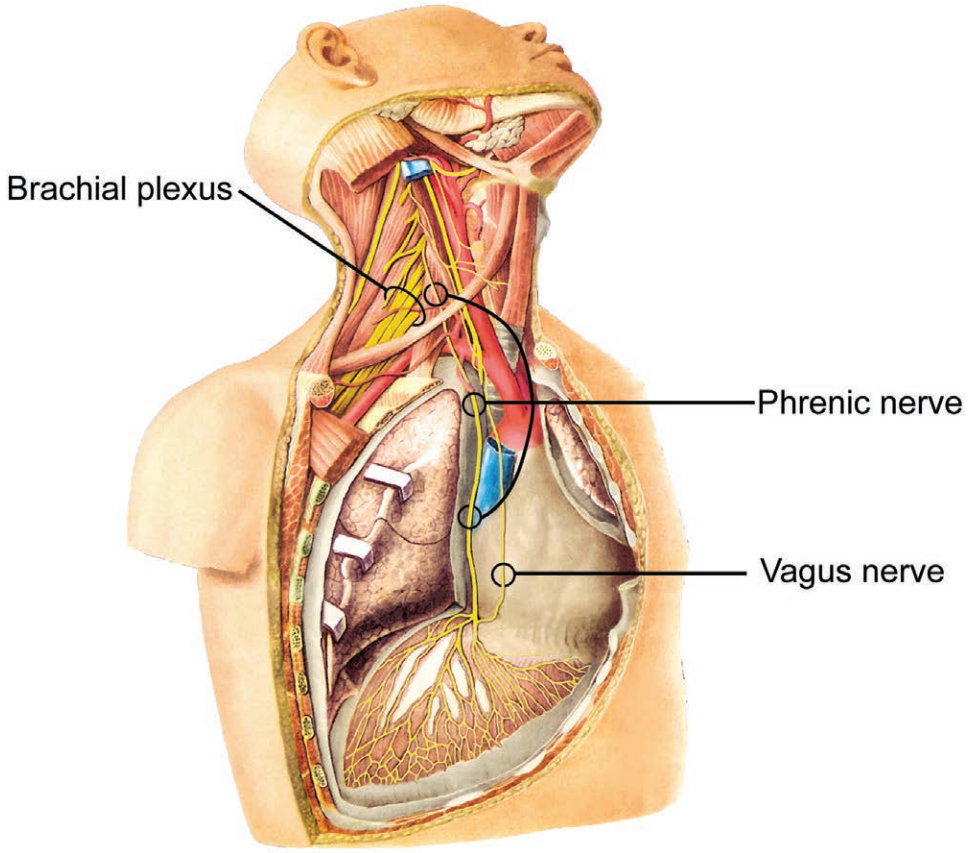

Fig. 4. Illustration demonstrating the course of the phrenic nerve from the root of the neck, through the thorax, and terminating at the diaphragm. Image adapted with permission from Danilo Jankovic and reproduced with permission from Ultrasound for Regional Anesthesia (USRA; http://www.usra.ca).

Persistent Phrenic Nerve Palsy. Persistent phrenic nerve palsy after interscalene block is a complication that has recently gained wider recognition, and its incidence has been estimated from case series data to range from 1 in 2,000[18] up to 1 in 100.[19] There are several potential causes of persistent phrenic nerve palsy that have been put forth in the literature. Nerve damage due to direct needle trauma or intraneural injection has been implicated in case reports of persistent phrenic nerve palsy after landmark-guided interscalene block techniques,[20–23] but not so far with ultrasound-guided interscalene block. Inflammatory scarring causing nerve entrapment has been reported with both landmark-guided and ultrasound-guided interscalene block, and although it has been suggested that this scarring may be related to local anesthetic myotoxicity,[24,25] these are postulated mechanisms without direct supporting evidence at present. A "double crush" syndrome[26] due to previous cervical spine stenosis along with nerve trauma also may contribute to persistent phrenic nerve palsy.[18] Finally, a "triple crush" mechanism that includes pressure ischemia resulting from high volumes of local anesthetic injected within the tight confines of the interscalene sheath also has been postulated.[27] It must be noted that these causes of persistent phrenic nerve palsy differ from those implicated in transient phrenic nerve palsy, and thus it cannot be assumed that strategies to reduce the risk of the latter will also reduce the risk of the former.

Physiologic Effects of Phrenic Nerve Palsy

The diaphragm is the most important inspiratory muscle, accounting for 75% of the increase in lung volume during quiet inspiration; intercostal, scalene, and sternocleidomastoid muscles contribute the remaining 25%. There is little crossover innervation of the right and left hemidiaphragms, and each can contract independently of the other in the event of unilateral phrenic nerve palsy. In the presence of diaphragmatic paresis, inspiration is achieved largely by contraction of intercostal and accessory muscles and expansion of the rib cage.[28] Pleural pressure is reduced, which leads to air intake and expansion of intrathoracic volume.[29] However, this reduction in pleural pressure during inspiration also causes the paralyzed diaphragm to move cephalad and the abdominal muscles inward. Consequently, there is reduced lung ventilation on the affected side, particularly of the lower lobe.[28,30] In healthy individuals, however, tidal volumes remain unchanged due to a greater contribution from the rib cage.[11,28] In higher-risk patient groups, hypoxia and dyspnea may ensue and require treatment by sitting the patient upright and administering supplemental oxygen

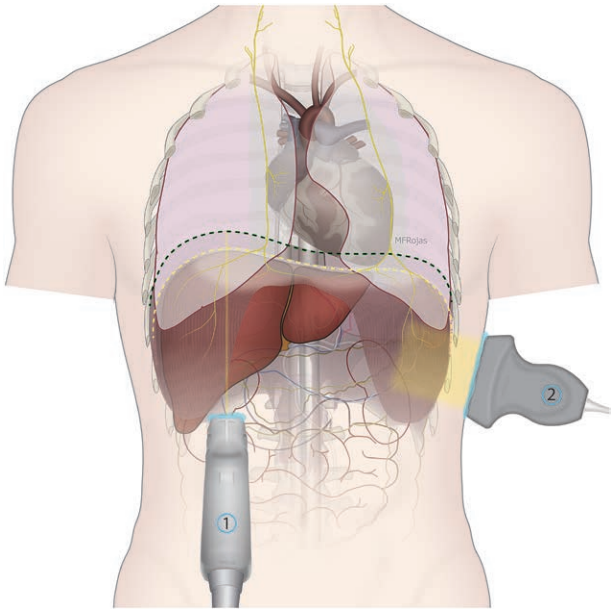

Fig. 5. Diaphragmatic ultrasound. Curved array transducer (1) position for scanning the diaphragm in the midclavicular, right subcostal margin using the liver as an acoustic window, and linear array transducer (2) on the left in the midaxillary line at the level of ribs eight to nine. Image reproduced with permission from Maria Fernanda Rojas Gomez and Ultrasound for Regional Anesthesia (USRA; http://www.usra.ca).

therapy or, in severe cases, instituting noninvasive or invasive ventilatory support to augment tidal volumes.

Subjectively, dyspnea is the cardinal symptom of phrenic nerve palsy after interscalene block. However, just as phrenic nerve palsy does not always result in dyspnea, dyspnea may also be experienced in the absence of phrenic nerve palsy.[31–38] Although up to 40% of patients complain of dyspnea after interscalene block or supraclavicular block,[14,17,39] only one third[40] to three quarters[41] of these patients have objective evidence of phrenic nerve palsy. Patients who are obese are more likely to experience dyspnea in association with phrenic nerve palsy.[42] Thus, although dyspnea clearly is more prevalent in the presence of phrenic nerve palsy,[42] it is neither sensitive nor specific for phrenic nerve palsy. Dyspnea after interscalene block might not be related to the block itself, and other causes must be sought and excluded.

Assessing the Severity of Phrenic Nerve Palsy

The impact of phrenic nerve palsy on respiratory function may be quantified by several bedside methods, including pulse oximetry, pulmonary function tests, and sonographic evaluation of the diaphragm.

Oxygen Saturation. Hypoxemia secondary to unilateral phrenic nerve palsy after regional anesthesia has a low diagnostic sensitivity due to the mechanics of respiratory compensation. Accessory muscles and the contralateral diaphragm both contribute to maintaining gas exchange.

There are conflicting data regarding the incidence and extent of hypoxemia after unilateral phrenic nerve palsy, which probably reflects its multifactorial etiology. Contemporary studies in healthy patients with unilateral phrenic nerve palsy suggest that oxygen saturations may remain unchanged[40] or decrease by less than 7%.[16,43,44] The limited extent of this change correlates with a reduction in Pa_{O_2} of 6 to 7 mmHg and an increase in Pa_{CO_2} of only 3 mmHg.[28] In contrast, hypoxemia may be more significant after interscalene block in patients with multiple comorbidities and who receive higher volumes and/or concentrations of local anesthetic.[33,34,45] In one study of patients with chronic renal failure undergoing arteriovenous fistula surgery, 10% had oxygen saturations less than 85% on room air after a high-volume (30 ml) interscalene block.[45] In another study, brief episodes of oxygen saturations less than 85% after interscalene block with 20 to 28 ml bupivacaine, 0.75%, were seen in 4 of 10 patients, three of whom were obese.[33]

Pulmonary Function Tests. Pulmonary function tests using bedside spirometry to assess diaphragmatic function should be performed with the patient in the semirecumbent position, with the head up at 45°. Baseline pulmonary function tests ideally should be performed before block performance to place postblock values into context and more accurately quantify any deterioration. However, isolated testing after block performance may be compared with predicted values based on patient demographics, although this is less accurate than a comparison with baseline values. Unilateral phrenic nerve injury not related to regional anesthesia reduces the total lung capacity (by 14 to 29%), forced vital capacity (by 23 to 27%), and inspiratory capacity (by 10 to 20%) compared with baseline or predicted parameters.[46–49] Unilateral phrenic nerve palsy after interscalene block reduces the forced expiratory volume in 1 s (FEV_1) by 16 to 40%,[17,50] the forced vital capacity by 13 to 40%,[36,50] and the peak expiratory flow rates by 15 to 43% (tables 1 and 2).[36,40]

Ultrasound. The assessment of phrenic nerve palsy using ultrasound relies on visualizing the diaphragm and quantifying the magnitude and direction of its movement with respiration. The most common method involves placing a 3- to 5-MHz curved array transducer inferior to the costal margin and in a longitudinal parasagittal orientation in the anterior axillary line on the left or in the midclavicular line on the right (fig. 5). The ultrasound beam is directed medially and cephalad to visualize the posterior third of the hemidiaphragm by using either the spleen or the liver as an acoustic window (fig. 6) in a two-dimensional B-mode. Visualization on the left often is technically more challenging due to the smaller acoustic window of the spleen and the presence of the air-filled stomach. Once a view of the curved, hyperechoic diaphragmatic line has been obtained, M-mode sonography is used to quantify the extent of diaphragmatic excursion. In men, the normal displacement of an unaffected diaphragm is 1.8 ± 0.3, 7.0 ± 0.6, and 2.9 ± 0.6 cm in quiet breathing, deep breathing, and sniffing, respectively,

Table 1. Characteristics of Relevant Studies of Regional Anesthesia Techniques for Shoulder Surgery

Study	Characteristics						
	Interscalene Block Technique	Comparison	Target	Drug	Concentration	Volume	Dose
Noncomparative studies							
Urmey *et al.*,[14] 1991, n = 13	SS, LM	—	IS (Winnie approach, C5, C6)	Mepivacaine	1.5%	34–52 ml	510–780 mg
Urmey and McDonald,[13] 1992, n = 8	SS, LM	—	IS (Winnie approach, C5, C6)	Mepivacaine	1.5%	45 ml	675 mg
Neal *et al.*,[11] 1998, n = 8	SS, LM	—	SC (plumb-bob technique)	Lidocaine	1.5%	30 ml	450 mg
Volume effect							
Urmey and Gloeggler,[50] 1993, n = 20	SS, LM	LA volume 20 ml *vs.* 45 ml	IS (Winnie approach, C5, C6)	Mepivacaine	1.5%	45 ml	675 mg
						20 ml	300 mg
Sinha *et al.*,[51] 2011, n = 30	SS, USG	LA volume 10 ml *vs.* 20 ml	IS, intraplexus C5, C6, or C7 roots	Ropivacaine	0.5%	20 ml	100 mg
						10 ml	50 mg
Riazi *et al.*,[16] 2008, n = 40	SS, USG	LA volume 5 ml *vs.* 20 ml	IS, intraplexus C5, C6 roots	Ropivacaine	0.5%	20 ml	100 mg
						5 ml	25 mg
Lee *et al.*,[52] 2011, n = 60	SS, USG	LA volume 5 ml *vs.* 10 ml	IS, intraplexus C5, C6 roots	Ropivacaine	0.75%	10 ml	75 mg
						5 ml	37.5 mg
Concentration effect							
Al-Kaisy *et al.*,[36] 1991, n = 11	SS, NS	LA concentration 0.25% *vs.* 0.5%	C5, C6	Bupivacaine	0.5%	10 ml	50 mg
					0.25%	10 ml	25 mg
Casati *et al.*,[53] 1999, n = 30	SS, NS	LA concentration ropivacaine 0.5% *vs.* ropivacaine 0.75% *vs.* mepivacaine 2%	IS (Winnie approach, C5, C6)	Mepivacaine	2%	20 ml	400 mg
				Ropivacaine	0.75%	20 ml	150 mg
				Ropivacaine	0.5%	20 ml	100 mg
Wong *et al.*,[43] 2016, n = 50	SS, USG	LA concentration 0.1% *vs.* 0.2%	IS, intraplexus around trunks	Ropivacaine	0.2%	20 ml	40 mg
					0.1%		20 mg
Al-Kaisy *et al.*,[54] 1998, n = 30	SS, NS	LA concentration 0.125% *vs.* placebo	C5, C6	0.9% saline	Placebo	10 ml	0 mg
				Bupivacaine	0.125%	10 ml	12.5 mg
Concentration and volume effect							
Pippa *et al.*,[55] 2006, n = 60	SS, NS	LA concentration and volume 0.25/1% *vs.* 0.5/2% 60 (30/30) ml *vs.* 30 (15/15) ml	IS (Winnie approach, C5, C6)	Bupivacaine/ lidocaine	0.25/1%	60 (30/30) ml	75/300 mg
					0.5/2%	30 (15/15) ml	75/300 mg
Zhai *et al.*,[56] 2016, n = 95	SS, USG	LA concentration and volume 0.25% *vs.* 0.5% *vs.* 0.75% 20 ml *vs.* 10 ml *vs.* 6.7 ml	IS, intraplexus C5, C6 roots	Ropivacaine	0.25%	20 ml	50 mg
					0.5%	10 ml	
					0.75%	6.7 ml	
Injection below C6							
Renes *et al.*,[15] 2009, n = 30	SS, USG/NS	Localization method USG *vs.* NS	IS, intraplexus C7 root	NS ropivacaine	0.75%	10 ml	75 mg
				US ropivacaine		10 ml	75 mg
Petrar *et al.*,[41] 2015, n = 64	SS, USG	Injection site supraclavicular *vs.* infraclavicular	IS, intraplexus	SC ropivacaine	0.5%	30 ml	150 mg
			IC, posterior and lateral cords	IC ropivacaine		30 ml	150 mg

(*Continued*)

Table 1. (Continued)

Study	Characteristics						
	Interscalene Block Technique	Comparison	Target	Drug	Concentration	Volume	Dose
Extrafascial injection							
Palhais *et al.*,[17] 2016, n = 40	SS, USG	Injection site extrafascial *vs.* intraplexus	IS, intraplexus C5, C6 roots	Bupivacaine	0.5%	20 ml	100 mg
			IS, extrafascial 4 mm lateral to C5, C6 roots	Bupivacaine		20 ml	100 mg
Catheter technique							
Renes *et al.*,[57] 2010, n = 20	Catheter, USG	Minimum effective volume	IS, intraplexus C7 root	Ropivacaine	0.75%	2–6 ml	15–45 mg
Stundner *et al.*,[58] 2016, n = 30	Catheter, USG	LA volume 5 ml *vs.* 20 ml	IS, intraplexus C5, C6 roots	Ropivacaine	0.75%	20 ml	150 mg
						5 ml	37.5 mg
Hartrick *et al.*,[42] 2012, n = 36	Catheter, USG	LA volume 20 ml *vs.* 10 ml *vs.* 5 ml	IS, intraplexus C5 root	Ropivacaine	0.75%	20 ml	150 mg
						10 ml	75 mg
						5 ml	37.5 mg
Thackeray *et al.*,[44] 2013, n = 30	Catheter, USG	LA concentration 0.125% *vs.* 0.25%	IS, intraplexus C6	Bupivacaine	0.25%	20 ml	50 mg
					0.125%		25 mg
Koh *et al.*,[59] 2016, n = 75	Catheter, USG	Injection site interscalene *vs.* supraclavicular	IS, intraplexus C5, C6 roots	Ropivacaine	0.375%	20 ml	75 mg
			SC, intraplexus	Ropivacaine		20 ml	75 mg
Wiesmann *et al.*,[40] 2016, n = 114	Catheter, USG	Injection site interscalene *vs.* supraclavicular	SC, between upper and middle trunks	Ropivacaine	0.2%	10 ml + 4 ml/h + 4-ml bolus	20 mg
			IS, between upper and middle trunks				

Studies are divided into those assessing outcomes noncomparatively, as well as those describing outcomes by comparing volume effect, concentration effect, injection below C6, extrafascial injection, and catheter techniques.

IC = infraclavicular; IS = interscalene; LA = local anesthetic; LM = landmark; NS = nerve stimulator; SC = supraclavicular; SS = single shot; US = ultrasound; USG = ultrasound-guided.

Table 2. Incidence and Magnitude of Phrenic Nerve Palsy and Analgesic Outcomes of Relevant Studies of Regional Anesthesia Techniques for Shoulder Surgery

Study	Monitoring for PNP	Incidence/Magnitude						Analgesic Outcomes		Remarks
		US Evidence of PNP	FEV_1 Change	FVC Change	PEFR Change	Oxygen Saturation	Dyspnea	Pain Scores	Analgesic Consumption	
Noncomparative studies										
Urmey *et al.*,[14] 1991, n = 13	US	Sniff +5.96 cm to –4.53 cm (176% reduction)	—	—	—	—	38%	—	—	—
Urmey and McDonald,[13] 1992, n = 8	US, PFTs	100%	–26.4%	–27.2%	–15.4%	—	—	—	—	Magnetometry demonstrating increased chest wall movement (indicating use of intercostal and accessory muscles) with reduced ipsilateral abdominal movement
Neal *et al.*,[11] 1998, n = 8	US, PFTs	50%	Paralyzed: +6% Nonparalyzed: 0%	+2% +2%	+7% +6%	0%	0%	—	—	—
Volume effect										
Urmey and Gloeggler, [50] 1993, n = 20	US, PFTs	100%	–39.9%	–40.9%	–35.9%	—	—	—	—	—
		100%	–31.6%	–32%	–32%	—	—	—	—	
Sinha *et al.*,[51] 2011, n = 30	US, PFTs	93%	–35%	–32%	–19%	–	—	—	PACU fentanyl, 0 μg; home hydrocodone, 25 mg	No significant difference in analgesic consumption
		93%	–29%	–31%	–23%	–	—	—	0 μg ,30 mg	
Riazi *et al.*,[16] 2008, n = 40	US, PFTs	100%	–1.23 l	–1.59 l	–2.50 l/min	–5.85%	5%	VAS at 30 min, 0.3; 60 min, 1; 120 min, 1.3; 12 h, 3.1; 24 h, 4.7	Intraoperative fentanyl, 107.5 μg; PACU MEQ, 1.3 mg; 24-h MEQ, 26.5 mg	No significant difference in pain scores or analgesic consumption
		45%	–0.83 l	–0.7 l	–0.83 l/min	–1.5%	0%	1.1, 1.1, 0.5, 3.4, 3.6	140.3 μg, 2.9 mg, 23.3 mg	
Lee *et al.*,[52] 2011, n = 60	CXR	—	—	—	—	—	—	—	—	CXR diaphragmatic paresis 60% *vs.* 33%
Concentration effect										
Al-Kaisy *et al.*,[36] 1999, n = 11	US, PFTs	100%	–21.8%	–25.4%	–15.2%	—	0%	—	—	Volunteer study
		16%	–9.4%	–13.4%	–16.7%	—	0%	—	—	
Casati *et al.*,[53] 1999, n = 30	US, PFTs	100%	–40%	–39%	—	—	33%	—	No analgesia, 0 patients	No difference in pain scores. Longer time to first analgesic with both ropivacaine groups.
		100%	–38%	–41%	—	—	0%	—	No analgesia, 0 patients	
		100%	–30%	–40%	—	—	8.3%	—	No analgesia, 2 patients (20%)	

(*Continued*)

Table 2. (Continued)

Study	Monitoring for PNP	Incidence/Magnitude: US Evidence of PNP	Incidence/Magnitude: FEV_1 Change	Incidence/Magnitude: FVC Change	Incidence/Magnitude: PEFR Change	Incidence/Magnitude: Oxygen Saturation	Incidence/Magnitude: Dyspnea	Analgesic Outcomes: Pain Scores	Analgesic Outcomes: Analgesic Consumption	Remarks
Wong *et al.*,[43] 2016, n = 50	US, PFTs	66%	−28%	−33%	−34%	No desaturations	0%	NRS at 30 min, 0; 60 min, 0; AUC POD 0-3, 8.4	PACU fentanyl, 18 μg; 72-h codeine, 55 mg	Only difference in 72-h codeine requirement, no other pain score or analgesic consumption differences
		35%	−20%	−22%	−27%	No desaturations	0%	0, 0, 12.8	25, 102	
Al-Kaisy *et al.*,[54] 1998, n = 30		—	—	—	—	—	—	VAS at 20 min, 8.1; 30 min, 6.9; 60 min, 5.0; 120 min, 3.6	PACU morphine, 9.5 mg	—
		—	—	—	—	—	—	3.4, 3.5, 2.5, 2.2	PACU morphine, 2.7 mg	
Concentration and volume effect										
Pippa *et al.*,[55] 2006, n = 60	US	0%	—	—	—	—	—	—	—	Significantly better clinical outcomes and degree of analgesia in high-volume, low-concentration group in all regions of upper limb except for lateral neck and arm
		27%	—	—	—	—	—	—	—	
Zhai *et al.*,[56] 2016, n = 95	US	70%	—	—		—	—	NRS at PACU, 0; 4 h, 0; 8 h, 0; 24 h, 0; worst, 3	—	No significant difference in diaphragmatic paresis, pain scores, or satisfaction
		69%	—	—		—	—	0, 0, 0, 1, 3	—	
		58%	—	—		—	—	0, 0, 0, 1, 3	—	
Injection below C6										
Renes *et al.*,[15] 2009, n = 30	US, PFTs	Sigh, −95%; sniff, −87%	−0.9 l (−33%)	−1.2 l (−36%)	−105.1 l/min (−28%)	—	—	NRS at 30 min, 2	13% needing morphine	No significant difference in pain scores or analgesic consumption
		−11%, + 60%	−0.1 l (−4%)	−0.1 l (−3%)	−8 l/min (−2%)			NRS at 30 min, 2	13% needing morphine	
Petrar *et al.*,[41] 2015, n = 64	US	Partial, 9%; complete, 34%	—	—	—	—	25%	—	—	Supraclavicular *vs.* infraclavicular blocks
		Partial, 9%; complete, 3%	—	—	—	—	16%	—	—	
Extrafascial injection										
Palhais *et al.*,[17] 2016, n = 40	US, PFTs	90%	−0.9 l (−28%)	−1.2 l (−28%)	−2.1 l/s (−24%)	—	30%	NRS at PACU, 0.5; 24 h, 1.6	PACU rescue (n), 0; 24-h MEQ, 5 mg; 48-h MEQ, 7.5 mg	No significant difference in analgesic consumption after PACU
		21%	−0.6 l (−16%)	−0.8 l (−17%)	−0.7 l/s (−8%)	—	0%	0.4, 1.6	5, 5, 10 mg	

(*Continued*)

Table 2. (Continued)

Study	Monitoring for PNP	Incidence/Magnitude: US Evidence of PNP	Incidence/Magnitude: FEV_1 Change	Incidence/Magnitude: FVC Change	Incidence/Magnitude: PEFR Change	Incidence/Magnitude: Oxygen Saturation	Incidence/Magnitude: Dyspnea	Analgesic Outcomes: Pain Scores	Analgesic Outcomes: Analgesic Consumption	Remarks
Catheter technique										
Renes *et al*.,[57] 2010, n = 20	US, PFTs	0% in all patients at 2 h	0%	0%	0%	—	—	—	—	No changes in respiratory function with ≤6 ml injection, but changes after 24 h of infusion of 6 ml/h 0.2% ropivacaine in 100% of patients
Stundner *et al*.,[58] 2016, n = 30	US, PEFR	53%	—	—	−2.66 l/min	—	—	—	Intraoperative fentanyl, 200 μg	Nonsignificant difference in PNP. No significant difference in rest or dynamic pain scores or analgesic consumption.
		27%	—	—	−1.69 l/min		—	—	150 μg	
Hartrick *et al*.,[42] 2012, n = 36	US	−65% excursion	—	—	—	—	33%	NRS at PACU D/C, 0.62; 24 h, 2.54; 48 h, 2.15; 12 wk, 1.15	24-h MEQ, 13.4 mg	Only difference at PACU D/C between 5 ml and 20 ml, no other pain score or analgesic consumption differences
		−60% excursion	—	—	—	—	0%	1.58, 3.33, 2.08, 2.42	24-h MEQ, 29.8 mg	
		−66% excursion	—	—	—	—	8.3%	2.67, 3.67, 2.67, 1.5	24-h MEQ, 27.7 mg	
Thackeray *et al*.,[44] 2013, n = 30	US, oxygen saturations	78%	—	—	—	−4.3%	0%	0	Fentanyl, 0 μg	
		21%	—	—	—	−2.6%	0%	1	Fentanyl, 7 μg	
Koh *et al*.,[59] 2016, n = 75	US	Partial, 32%; complete, 63%	—	—	—	—	—	Mean NRS, 2.84	PACU morphine, 1 mg; 24-h MEQ, 8 mg	No significant difference in pain scores or analgesic consumption
		Partial, 26%; complete, 24%	—	—	—	—	—	Mean NRS, 2.57	0 mg, 7 mg	
Wiesmann *et al*.,[40] 2016, n = 114	US, PFTs	PACU, −82%; POD1, −46%	−33%	−38%	−32%	+1%	8%	0–1	Ward rescue opioids, 16%	No significant difference in pain scores or analgesic consumption
		PACU, −55%; POD1, −34%	−32%	−30%	−43%	+1%	7%	0–0	Ward rescue opioids, 5.2%	

Studies are divided into those assessing outcomes noncomparatively, as well as those describing outcomes by comparing volume effect, concentration effect, injection below C6, extrafascial injection, and catheter techniques.

CXR = chest X-ray; D/C = discharge; FEV_1 = forced expiratory flow rate in 1 s; FVC = forced vital capacity; MEQ = morphine equivalents; NRS = numerical rating scale; PACU = postanesthetic care unit; PEFR = peak expiratory flow rates; PFTs = pulmonary function tests; PNP = phrenic nerve palsy; POD = postoperative day; VAS = visual analog score; US = ultrasound.

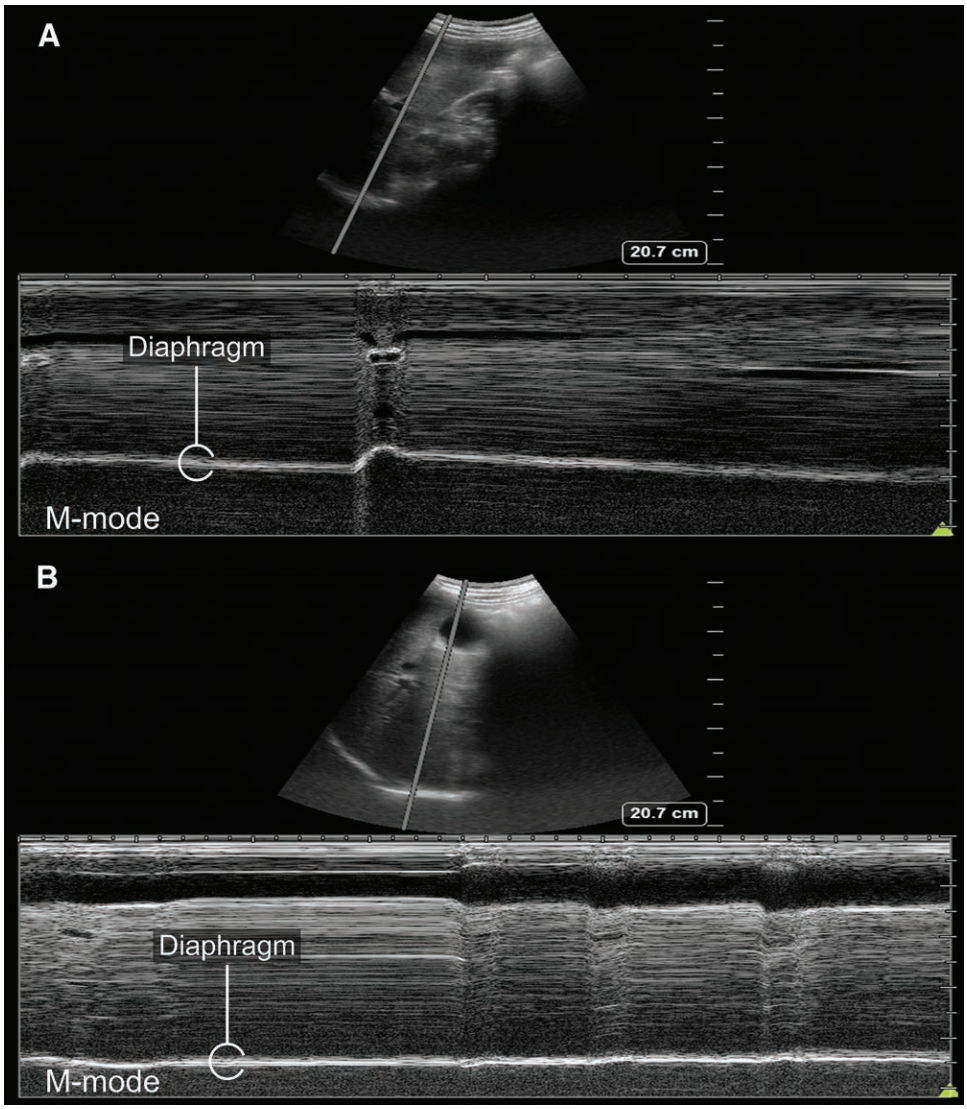

Fig. 6. Curved array transducer ultrasound image of the right diaphragm using the liver as an acoustic window in two-dimensional B-mode and M-mode. (*A*) Preblock sniff test assessment for phrenic nerve palsy. The diaphragm (*white circle*) is seen to move caudally, toward the probe, in M-mode. (*B*) Postblock sniff test assessment for phrenic nerve palsy. There is no movement of the diaphragm seen in M-mode, indicating phrenic nerve palsy.

and 1.6 ± 0.3, 5.7 ± 1.0, and 2.6 ± 0.5 cm in women.[60] Once again, it is ideal to obtain baseline measures of diaphragmatic excursion before block performance. Although evidence of hemidiaphragmatic paresis may be seen within 5 min of local anesthetic injection, measurement should be repeated 15 to 30 min after block completion to allow time for the full extent of phrenic nerve palsy to develop.[14,50] In the presence of partial phrenic nerve palsy, a forceful rapid sniff (the sniff test) can demonstrate partial diaphragmatic paresis with a 25 to 75% reduction in caudal movement (toward the transducer) of the diaphragm. Complete phrenic nerve palsy may be diagnosed by paradoxical cephalad movement of the diaphragm[61,62] or a 75% or greater reduction in diaphragmatic movement.[15,41] Diaphragmatic ultrasound has been shown to have high sensitivity (93%) and specificity (100%) in diagnosing phrenic nerve dysfunction.[63]

An alternative, simpler ultrasound approach that may be used involves placing a high-frequency (10 to 15 MHz) linear array transducer in the coronal plane at the midaxillary line to obtain an intercostal view.[64] At the level of ribs eight to nine on the left and seven to eight on the right, the spleen or liver are centered with the rib shadows on either side (fig. 5). On deep inspiration, caudal descent of the liver or spleen precedes descent of the bright pleural line (fig. 7). The transducer is then moved in both caudal and cephalad directions to visualize the end-inspiratory and end-expiratory levels of the pleural line, respectively, which are then marked on the patient's skin. This process is repeated before and after the chosen regional anesthetic technique with the patient in

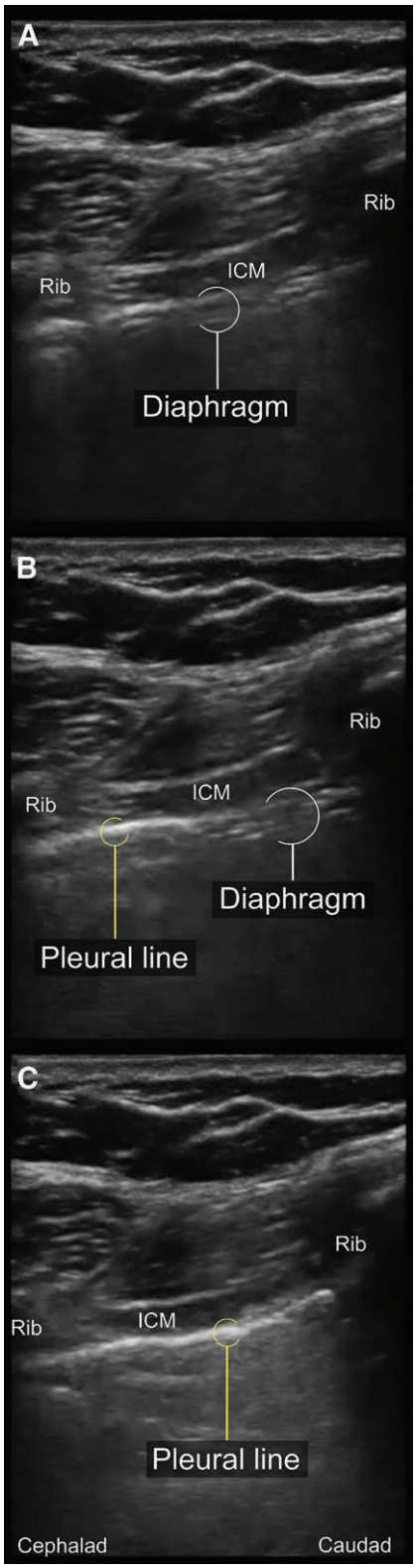

Fig. 7. Linear array transducer ultrasound image of the pleura in the right midaxillary line at the level of the seventh and eighth ribs in (*A*) early inspiration; (*B*) mid-inspiration; and (*C*) end-inspiration. The pleural line (*yellow circle*) can be seen progressively descending with inspiration. The intercostal muscles (ICM) lie superficial to the diaphragm (*white circle*).

the same position. Minimal change signifies no block, but a reduction in this distance represents phrenic nerve palsy.[65] Although this technique is yet to be validated, it is a simple qualitative assessment that relies on the gross caudad movement of the pleural line during inspiration representing diaphragmatic excursion and thus phrenic nerve function.

Correlation between Parameters

Quantified sonography of the diaphragm is more sensitive to changes in unilateral diaphragmatic dysfunction than pulmonary function tests and oxygen saturation because the latter two variables assess bilateral pulmonary function simultaneously, including the use of accessory muscles and contralateral diaphragmatic activity. However, there may be some correlation between these parameters. Borgeat *et al.*[66] demonstrated that a 60 to 80% reduction in unilateral diaphragmatic excursion with forced respiration was associated with a 30 to 40% reduction in both vital capacity and FEV_1. Similarly, our group has demonstrated that hemidiaphragmatic paresis resulted in a decrease in forced vital capacity and FEV_1 to 75 and 78% of baseline, respectively.[36] However, these patients remain asymptomatic and require no treatment. Although there is clearly some correlation between pulmonary function test changes and ultrasound evidence of unilateral diaphragmatic paresis, no study has explicitly and specifically assessed the correlation between ultrasound, pulmonary function test, oxygen saturations, and subjective symptoms of dyspnea.

Strategies to Reduce Phrenic Nerve Palsy in Regional Anesthesia of the Shoulder

Transient phrenic nerve palsy after regional anesthesia for shoulder surgery results from a direct inhibitory effect of local anesthetic on the phrenic nerve or its roots (C3–C5), and thus minimizing its occurrence depends on reducing the dose of local anesthetic reaching these neural structures. This can be achieved by modifying the local anesthetic dose (volume and concentration),[54] injection site and technique in interscalene block, or by modifying the location of local anesthetic injection and using a different regional anesthetic technique altogether. Ultrasound has been instrumental in the development of these modifications: the increased accuracy of local anesthetic deposition allows the use of lower doses, and direct visualization increases the range of available sites for injection. In the following section, we review the evidence for the effectiveness of these various modifications in minimizing the risk of phrenic nerve palsy while preserving analgesic efficacy.

Modifications of Interscalene Block

Local Anesthetic Volume. There is a clear relationship between the volume of local anesthetic injected during interscalene block and the occurrence of phrenic nerve palsy. This is likely to be related to the greater extent of spread that occurs with larger volumes. An injection around the C5–C6 nerve roots with

volumes of 20 ml or greater inevitably produces phrenic nerve palsy, regardless of localization technique.[16,50,51,53] When an ultrasound-guided technique is used, a volume of 10 ml reduces the incidence of phrenic nerve palsy to as low as 60%,[52] whereas a volume of 5 ml reduces it still further to between 27[58] and 45%,[16] without compromising analgesic efficacy up to 24 h postoperatively.[16,52] Although McNaught *et al.*[67] determined that the minimum effective volume for achieving analgesia for shoulder surgery with ultrasound-guided interscalene block at the C5–C6 nerve root level is as low as 0.9 ml ropivacaine, 0.5%, it must be noted that the duration of analgesia was not assessed formally beyond the first 30 min after surgery. The incidence of respiratory compromise was not reported in this study, so it is unclear whether there are further reductions in the incidence of phrenic nerve palsy at volumes less than 5 ml.

Local Anesthetic Concentration. Several studies have shown that reducing local anesthetic concentration independent of volume, thus reducing the dose of drug delivered, also produces a significant decrease in the incidence of phrenic nerve palsy and an improvement in pulmonary function after landmark- or ultrasound-guided interscalene block.[36,43,44] With a nerve stimulator-guided interscalene block, halving the concentration of a 30-ml mixture of 0.5% bupivacaine and 2% lidocaine but doubling the volume led to a reduction in phrenic nerve palsy from 27% to 0%.[55] Halving the concentration of bupivacaine from 0.5% to 0.25% reduced the incidence of phrenic nerve palsy from 100% to 17% when 10 ml was administered *via* a landmark approach[36] and from 78% to 21% when 20 ml was administered with nerve-stimulator localization.[44] Similarly, the incidence of phrenic nerve palsy was reduced from 71% to 42% by halving the concentration of 20 ml ropivacaine from 0.2% to 0.1% in an ultrasound-guided interscalene block.[43] Unfortunately, this reduction in phrenic nerve palsy generally appears to come at the expense of reduced analgesic efficacy. The reduction in local anesthetic concentration and dose decreased duration of sensory blockade by 34% and increased postoperative opioid requirements by up to 50%.[43,44] Zhai *et al.*[56] demonstrated that there is no significant difference in the incidence of phrenic nerve palsy with a fixed 50-mg dose of ropivacaine for ultrasound-guided interscalene block using concentrations of 0.25, 0.5, or 0.75%, with minimal effect on analgesic outcomes.

Site of Injection.

Periplexus Injection. Recently, the concept of ultrasound-guided periplexus (between the interscalene muscles and brachial plexus nerve sheath) injection of local anesthetic has been introduced for interscalene block. Palhais *et al.*[17] recently reported that an ultrasound-guided extrafascial (periplexus) injection of 20 ml bupivacaine 0.5%, performed 4 mm lateral to the brachial plexus sheath not only provided similar analgesia compared with an intraplexus injection between the C5 and C6 roots but also reduced the incidence of diaphragmatic paresis from 90% to 21%. In addition, FEV_1, forced vital capacity, and peak expiratory flow rates were less affected in the extrafascial group compared with an intraplexus injection, decreasing by 16 *versus* 28%, 17 *versus* 28%, and 8 *versus* 24%, respectively.[17]

Intrafascial Injection Below C6 Level. Another strategy to avoid phrenic nerve palsy involves injecting local anesthetic further away from the C5 and C6 roots and phrenic nerve. Renes *et al.*[15] showed that ultrasound-guided injection of 10 ml ropivacaine 0.75%, around the C7 nerve root resulted in similar analgesia, but only a 13% incidence of phrenic nerve palsy compared with 93% with a neurostimulation-guided interscalene block using the same dose of local anesthetic. Recovery of diaphragmatic function also was faster in the patients who received the C7 root injection. In a subsequent study, the same authors reported that the minimum effective anesthetic volume to achieve complete sensory block of C5 and C6 dermatomes within 30 min in 50% of patients using this technique was 2.9 ml ropivacaine 0.75%. They noted that none of the 20 patients who received 6 ml ropivacaine 0.75%, or less had any evidence of diaphragmatic paresis up to 2 h after injection.[57]

Injection Posterior versus Interior to the C5–C6 Nerve Roots. A recent study compared the effect of performing an ultrasound-guided intraplexus injection on the anterior *versus* posterior aspect of the C5–C6 nerve roots with 15 ml ropivacaine 1%.[35] There was a similar reduction of 12 to 28% in all pulmonary function parameters in both groups. Once again, this suggests that the dose and volume of local anesthetic and the caudocephalad level at which it is injected are the most significant factors affecting incidence of phrenic nerve palsy.

Injection Method. There are no studies reporting the impact of injection dynamics on phrenic nerve palsy. It is possible that a slower, lower-pressure, titrated injection of low-volume aliquots also may limit spread of injectate to the phrenic nerve, but this is yet to be supported by published evidence. Interestingly, injection of local anesthesia through a catheter appears to produce a less dramatic change in diaphragmatic sonographic excursion than if the same large-volume bolus was injected directly through a needle, possibly supporting a benefit of titrated injection.[66] This suggests that injection dynamics may play an important role in development of diaphragmatic dysfunction and should be investigated further.

Alternatives to Interscalene Block

The conventional ultrasound-guided interscalene block is a direct carryover from the landmark-guided approach, which relied on the interscalene groove and the anterior tubercle of the C6 transverse process as key landmarks, and thus necessitated a needle approach to the brachial plexus at the root level. This restriction no longer exists; ultrasound allows visualization of the entire brachial plexus and its individual branches, and thus similar analgesic effects can be achieved with more selective injection further away from the phrenic nerve and the C5 and C6 roots.

Superior Trunk Block. The superior trunk is formed by the union of C5 and C6 roots and is an appealing alternative target for local anesthetic injection, given that the phrenic nerve has diverged a considerable distance away from the brachial plexus at this level. All the terminal nerves supplying the shoulder arise distal to the origin of the superior trunk and hence analgesic efficacy is not compromised. However, to date, only two case reports of this technique have been published,[68,69] and data supporting its effectiveness in minimizing phrenic nerve palsy are still awaited.

The superior trunk also can be targeted at the supraclavicular brachial plexus level. Injection with 20 to 30 ml local anesthetic is associated with a 25 to 51% incidence of phrenic nerve palsy in both landmark- and ultrasound-guided studies.[11,40,41,59] Nonetheless, inferior sensory block and greater analgesic requirements in shoulder surgery have been reported with supraclavicular brachial plexus block compared with interscalene block.[37,59] This may reflect incomplete blockade of the suprascapular nerve, which has left the brachial plexus at this point. Related to this, placement of a supraclavicular brachial plexus catheter just proximal to the exit of the suprascapular nerve from the common superior trunk sheath has been described; there were apparently no episodes of phrenic nerve palsy with this technique reported in published correspondence.[70] Clinical trials to verify its efficacy are awaited.

Suprascapular and Axillary Nerve Block. The risk of phrenic nerve palsy might be eliminated by avoiding injection around the brachial plexus and performing a suprascapular nerve and axillary nerve block instead. The suprascapular nerve provides sensory fibers to approximately 70% of the shoulder joint capsule, and blocking this peripheral nerve can be performed with either a landmark-guided[71–73] or ultrasound-guided technique.[4,74,75] The suprascapular nerve can either be blocked in the suprascapular fossa or in the root of the neck distal to where it arises from the superior trunk of the brachial plexus.[76] However, large volumes of injection in the latter approach may still potentially lead to local anesthetic spread to the phrenic nerve and its roots.

The axillary nerve is a terminal branch of the posterior cord of the brachial plexus. It may be blocked in the anterior chest where it arises from the posterior cord of the brachial plexus in the infraclavicular and proximal axillary area[77] or posterior to the humerus as it emerges from the quadrangular space.[74,75] This latter approach may occasionally miss the articular branches of the axillary nerve and may be responsible for inferior analgesic outcomes.[78]

In arthroscopic shoulder surgery, suprascapular nerve block alone or combined with an axillary nerve block has been shown to provide superior analgesia compared with placebo or subacromial local anesthetic infiltration[79–81] but is less effective compared with interscalene block.[78,79] Because this peripheral nerve block technique primarily targets the capsular innervation of the shoulder, it also may be less useful in open or extensive shoulder surgery.[82] Nevertheless, this technique has a good safety record in chronic pain practice[83] and has not been associated with any reported episodes of phrenic nerve palsy to date. In view of the trade-off in analgesic efficacy, suprascapular and axillary nerve blocks are probably best reserved for patients with preexisting respiratory dysfunction or who have other comorbidities (*e.g.*, obesity) that are likely to lead to clinically significant dyspnea and hypoxemia in the presence of unilateral phrenic nerve palsy.

Catheter Techniques

The risk of phrenic nerve palsy appears to be different between single-shot and continuous interscalene block. Infusion rates of 4 to 6 ml/h will invariably lead to phrenic nerve palsy over the first 24 h, regardless of the concentration of the local anesthetic.[84] Similarly, Renes *et al.*[15] found that although there was no evidence of phrenic nerve palsy 2 h after a small bolus injection of 0.75% ropivacaine at the C7 nerve root, all patients developed either partial or complete phrenic nerve palsy after a 24-h infusion of 0.2% ropivacaine at 6 ml/h.

Strategies that have been proposed to reduce phrenic nerve palsy while preserving analgesic efficacy include limiting infusion rates to 2 ml/h.[85] In a letter to the editor, Tsui and Dillane[86] reported that using an intermittent bolus regimen reduces phrenic nerve palsy; however, this has yet to be confirmed in a formal clinical trial. Another approach is to use a short-acting agent such as lidocaine instead of ropivacaine or bupivacaine. In the event of respiratory compromise due to phrenic nerve palsy, cessation of the infusion should result in a more rapid return of phrenic nerve function.[87] It also may be possible to speed up the resolution of phrenic nerve palsy by administering a bolus of 0.9% sodium chloride through the catheter to "wash off" residual local anesthetic.[86]

Future Trends

As discussed previously, the use of ultra-low volumes and doses of local anesthetic will minimize the risk of phrenic nerve palsy but at the expense of reduced duration of analgesia. The use of intravenous dexamethasone or perineural local anesthetic adjuvants that prolong the duration of sensory-motor blockade and analgesia[88–90] are a promising way to address this issue and should be specifically studied in this context. However, the potential risk and impact of a prolonged phrenic nerve block must still be considered, because none of the described techniques to date guarantee that this can be avoided completely. Liposomal bupivacaine[91,92] is not approved presently for perineural injection, but using it in local wound infiltration may be an alternative worthy of further study.[93,94] Continued investigation also is needed into the optimal dosing strategy in continuous catheter techniques, as well as the impact of different injection methods, including titrated dosing, the use of low injection pressures, as well as the concept of reversal of phrenic nerve palsy by local anesthetic washout.[86]

Conclusions

Regional anesthesia continues to be of value in providing analgesia for shoulder surgery, but its benefits must be weighed against the risks, including phrenic nerve palsy. The high incidence of phrenic nerve palsy associated with the conventional technique of interscalene block have led some to propose that "the safest option to avoid phrenic nerve block would be to avoid performing an interscalene block" altogether.[3] However, the evidence indicates first that temporary phrenic nerve palsy is inconsequential in the vast majority of healthy patients and, second, that relatively simple modifications such as minimizing local anesthetic doses and injection volumes (to less than 10 ml), as well as performing injection further distal to the C5–C6 nerve roots (*e.g.*, at the level of the superior trunk or supraclavicular brachial plexus), will significantly reduce the incidence of phrenic nerve palsy. Combined suprascapular and axillary nerve blocks are another alternative to consider in scenarios in which avoiding phrenic nerve palsy is critical, particularly in arthroscopic shoulder surgery. We encourage practitioners to use the principles and methods outlined in this article to refine and tailor their regional anesthetic strategy to each patient in their care, taking into account all the medical and surgical considerations pertinent to that individual.

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Competing Interests

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ANESTHESIOLOGY REFLECTIONS FROM THE WOOD LIBRARY-MUSEUM

William B. Quinn, M.D., Eclectic Physician and Occasional "Anaesthetist"

In Springfield, Ohio, on St. Patrick's Day of 1920, a female patient paid $15 to Clayton W. Russell, M.D. (1866 to 1922). Her receipt from the surgeon (*above*) allocated $5 to his surgical assistant, Howard H. Austin, M.D. (1880 to 1915). The remaining $10 was designated for "Dr. Quinn, Anaesthe[ti]st." So who was Doctor Quinn? A Kentucky native, William Babbit Quinn, M.D. (1892 to 1970), was raised by his Eclectic-physician mother after she was widowed during William's first week of life. Young William followed in his mother's footsteps, graduating in 1913 from her alma mater, the Eclectic Medical Institute of Cincinnati, Ohio. He trained and practiced in Springfield, Ohio, Blackwell's Island, New York, and then Hollywood, California, before settling back in Springfield. The depicted receipt was likely issued from the surgeon's office in the Fairbanks Building, where all three of these Eclectic alumni, Drs. Russell, Austin, and Quinn, maintained professional offices. In this time period, surgeons could charge the equivalent of 5 to 10% of their surgeon's fee from patients to pay for services rendered by the anesthetist. (Copyright © the American Society of Anesthesiologists' Wood Library-Museum of Anesthesiology.)

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