Surgical Treatment of Permanent Diaphragm Paralysis after Interscalene Nerve Block for Shoulder Surgery

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UNILATERAL diaphragm paralysis after interscalene nerve block can result in respiratory disturbances that may have a substantial impact on quality of life and increased prevalence of respiratory infections.[1,2] Several reports have estimated the incidence of transient diaphragm paralysis after routine interscalene blocks for shoulder surgery to be 100%,[3] however, with modified local anesthetic dosing and ultrasound-guided needle placement, more recent data suggest this rate to be lower.[4,5] Of greater concern is permanent diaphragm paralysis after interscalene nerve block for shoulder surgery. There are isolated reports in the literature regarding long-standing postprocedural diaphragm paralysis,[6,7] yet the underlying causative mechanism has not been previously sought. Peripheral nerve injury may occur from a variety of mechanical causes, including: transection, piercing, stretching, thermal injury, and compression. Alternatively, a nonmechanical injury can result from the toxic or ischemic effects of pharmacologic agents, such as local anesthetics, epinephrine, or chemotherapeutic agents. Phrenic nerve injury from many of these causes may be repaired using nerve-reconstruction techniques. We report our experience with 14 patients suffering permanent diaphragm paralysis after interscalene nerve blocks evaluated and treated between 2009 and 2012 at a tertiary referral center for peripheral nerve injuries with a catchment area that includes the entire United States. Parameters for review included: results of comprehensive evaluation, intraoperative findings during phrenic nerve surgery, and outcomes of surgical intervention (using our previously reported surgical treatment algorithm and outcomes study[8]). Successful treatment of the paralyzed diaphragm was based on improvements on: sniff testing, spirometry, nerve conduction testing, electromyography, and patient reporting.

CASE REPORTS

Case 1

The patient is a 58-yr-old man (patient 4; table 1) with a 2-yr history of chronic dyspnea and orthopnea noted immediately after an interscalene nerve block administered for rotator cuff repair. Left diaphragm paralysis was confirmed on chest fluoroscopy, and a phrenic neuropathy was documented based on a prolonged conduction latency and reduced motor amplitude on nerve-conduction testing. Given the significant impact on his quality of life, and the absence of any spontaneous improvement, the patient desired definitive repair of his phrenic nerve.

During surgery, the phrenic nerve was found to be adherent to dense scar tissue consistent with chronic inflammation. A complete neurolysis was performed from the nerve root through the level of the subclavian vessels. A partial scalene muscle excision was also performed to permit complete decompression. Before decompression of the nerve, stimulation at 2 mA did not present a clear compound muscle action potential. However, after decompression was performed, a clear potential was elicited at 1 mA. Direct observation of the phrenic nerve showed an area of decreased diameter in the proximal portion. A 5-cm sural nerve graft was harvested and was used to bypass this area of compression by suturing it into the side of the phrenic nerve. Kenalog was placed in the area of

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Table 1. Demographics of Patients Diagnosed with Permanent Diaphragm Paralysis after Interscalene Nerve Block

Patient	Age	Sex	BMI	Side	Type of Surgery	No. of Total Blocks	Hx Trauma/ Surgery	SSM vs. Catheter	Neuropathy	Successfully Treated	Pathology
1	45	M	36.8	R	RC × 2	2	C5-6 fusion	Catheter	Y	Y	Scar
2	73	M	28	R	RC	1	N	SSM	Y	N	SA
3	51	M	27.8	R	RC	1	N	Catheter	N	Y	Scar/SA
4	58	M	31.4	L	RC	1	N	SSM	N	Y	Scar
5	41	M	26.2	L	RC	1	N	Catheter	N	Y	VC
6	71	M	31.3	R	RC	1	N	SSM	Y	Y	Scar
7	60	M	30.8	R	RC	1	N	Catheter	N	Y	Scar
8	71	M	31.5	L	BS	1	N	SSM	N	NS	—
9	67	M	27.9	R	RC	1	N	Catheter	N	N	Scar/SA
10	56	M	27.9	L	RC	1	N	Catheter	N	N	Scar/SA
11	50	M	29.7	R	RC × 4	4	N	Catheter	N	Y	Scar/SA
12	73	M	34.4	R	RC	1	N	SSM	Y	N	Scar
13	39	M	31.7	L	RC	1	MVA	SSM	N	Y	Scar
14	56	M	27.9	L	RC	1	N	Catheter	N	Y	VC

BMI = body mass index; BS = bone spur removal; NS = no surgery performed; RC = rotator cuff repair; SA = segmental atrophy (narrowing) of phrenic nerve; SSM = single-shot method; VC = vascular compression of phrenic nerve.

the nerve graft anastomoses to minimize postoperative inflammation, and a collagen nerve conduit was used to "wrap" both the phrenic nerve and nerve graft to prevent recurrence of inflammatory adhesions.

The patient has had an uneventful recovery and has begun to notice an improvement in respiratory ability at 6 months, which has been corroborated by a 10% improvement in forced vital capacity and 12% improvement in forced expiratory volume in 1 s.

Case 2

The patient is a 41-yr-old man (patient 5; table 1) who presented with an 11-month history of exertional dyspnea and orthopnea that started immediately after rotator cuff surgery performed using regional anesthesia. Left-sided diaphragm paralysis was confirmed on chest fluoroscopy and nerve-conduction testing revealed a phrenic neuropathy. The patient is a drill instructor at a state police academy and was on restricted duty because of the symptoms.

He underwent phrenic nerve decompression and sural nerve interposition grafting. The prevertebral fascia was thickened and fibrotic, and a large, dilated transverse cervical vein was noted to be adherent to the phrenic nerve, creating an obvious vascular compression. The fascia and adherent vein were gently released from the nerve, revealing an obvious narrowing in this segment. There was minimal improvement in nerve conduction after decompression and an interposition nerve graft was placed end-to-side to bypass the narrowed segment. Kenalog and a nerve conduit were used to prevent adherence and inflammation.

The patient reported improvement in respiratory function at 6 months, and postoperative nerve-conduction testing and chest fluoroscopy confirmed recovery of diaphragmatic activity. The patient has returned to full, active duty.

Case 3

The patient is a 45-yr-old man (patient 1; table 1) who was admitted to the hospital with pneumonia 2 weeks after revision rotator cuff surgery during which a repeat interscalene nerve block was performed. Right diaphragm paralysis was confirmed and the patient was monitored for 8 months. Interval testing failed to reveal improvement in diaphragm function, and nerve-conduction testing demonstrated a phrenic neuropathy.

Phrenic nerve decompression was performed to reverse a clinically apparent nerve entrapment. Intraoperative nerve-conduction testing demonstrated a substantial improvement after release of inflammatory adhesions.

Follow-up radiographic imaging and nerve-conduction testing at 3 months revealed a reversal of the diaphragm paralysis. The patient has reported improvements in respiratory function and has begun a program of pulmonary rehabilitation to optimize muscle recovery.

Additional Patients

Demographic data and surgical outcomes on the remaining patients can be found in table 1. Technical details of the interscalene nerve blocks used on the patients in this series can be found in table 2.

Discussion

The phrenic nerve originates from the third through fifth cervical roots and course inferiorly toward the clavicle interposed between the anterior scalene muscle beneath it and the prevertebral fascia as its "roof." The brachial plexus is in close proximity, emerging from between the anterior and middle scalene muscles, and coursing obliquely from the phrenic nerve toward the upper extremity. Nerve blocks of the brachial plexus will undoubtedly impact the phrenic nerve in a

Table 2. Specifics of the Interscalene Nerve Block in Patients Who Developed Postprocedure Phrenic Nerve Injury

Patient	SSM *vs.* Cath	US/NS/0	Local	Needle/Gauge	Cath. Duration, d
1	Cath	US	Ropivicaine	Tuohy/18	3
2	SSM	US	Lidocaine	b-bev./20	—
3	Cath	NS	Lido/Epi	Tuohy/18	4
4	SSM	0	Lido/Marc	b-bev./22	—
5	Cath	US	Lidocaine	Tuohy/18	3
6	SSM	US	Lidocaine	b-bev./22	—
7	Cath	0	Lido/Marc	Tuohy/18	2
8	SSM	US	Lido/Epi	b-bev./22	—
9	Cath	US	Lido/Epi	Tuohy/18	3
10	Cath	US	Lido/Marc	Tuohy/18	3
11	Cath	US	Lido/Epi	Tuohy/18	1
12	SSM	US	Lidocaine	b-bev./22	—
13	SSM	NS	Bupiv/Lido/Epi	b-bev./20	—
14	Cath	0	Lido/Epi	Tuohy/18	2

0 = no guidance.
b-bev. = ; Bupiv = ; cath =; Epi =; Lido = lidocaine; Marc =; NS = nerve stimulator; SSM = single-shot method; US = ultrasound-guided.

significant proportion of cases due to the anatomic proximity of these two structures,[3,6] yet should be transient, and reversal of the diaphragm paralysis should occur concomitantly with resolution of the peripheral nerve blockade.

In patients who suffer an unresolved diaphragm paralysis, there must be a pathophysiological process that progresses despite resolution of the anesthetic effect. It seems possible that a mechanism of nerve compression may develop in the hours and days after the intervention, leading to a prolonged or permanent diaphragm muscle weakness. Compression neuropathies are thought to result from isolated or combined effects of ionic, mechanical, and vascular insults to the involved nervous structure.[9] Most notably, any interruption to the venous outflow from the small blood vessels within the nerve sheath surrounding the axons can lead, first to a slowing of nerve conduction and subsequently, to progressive endoneurial edema.[9] Without prompt resolution of this edema fibroblasts will proliferate, resulting in the formation of scar tissue.[9] Perineural and intrafascicular scarring will further inhibit nerve transmission, altering both the mechanical and ionic activities in the nerve structures. Similar to nerve entrapment syndromes in the upper or lower extremity, there is the possibility that a preceding "event" or series of events can lead to edema in either the nerve directly, or in the anatomical structures surrounding the nerve. Regardless of a direct or indirect insult to the nerve, the pathophysiological process leading to this disorder may be initiated. The phrenic nerve may be vulnerable to compression, being snugly interposed between the anterior scalene muscle and the prevertebral fascia. Interscalene blocks that require multiple needle advances, or have difficulty with placement of in-dwelling catheters for infusion, may cause tissue damage to adjacent muscle and fascia making the phrenic nerve vulnerable to injury. Alternatively, in patients who have a mild, subclinical phrenic neuropathy from previous neck surgery or trauma,[10,11] or in those with systemic peripheral neuropathy,[12] it is possible that the phrenic nerve may be more susceptible to injury from procedural intervention, such as an interscalene nerve block.

The intraoperative findings we have identified in this patient population have been consistent with a compression neuropathy. In two patients we confirmed the nerve entrapment to be caused by adhesions between the phrenic nerve and a torturous, dilated transverse cervical artery. In a recent publication we described how ligation of the artery and release of the adhesions resulted in reversal of the ischemic neurapraxia and restoration of diaphragmatic function.[13] Alternatively, in other cases the compression has likely been due to thickening and fibrosis in the prevertebral fascia and anterior scalene muscle, narrowing or eliminating the anatomical space in which the phrenic nerve courses. Dense adhesions in this region, through a possible tethering effect, have also appeared to alter the normal course and caliber of the nerve. In two cases we observed anomalous relocation of the nerve, either transposed medially along the carotid sheath, or laterally adherent to the brachial plexus.

There is also the possibility that toxic or ischemic effects of the anesthetic could have contributed to, or have been a critical factor in the postoperative chronic neuropathy. Whereas nerve-conduction testing, electromyography, and intraoperative findings supported a compression neuropathy over toxic/ischemic injury (axonal *vs.* demyelinating pattern), it is very difficult to completely separate the two processes based on electrical studies alone. A pathological analysis of the phrenic nerve would be the only conclusive way to assess the difference. Regardless, the nerve surgery techniques performed in this series are established methods of functional restoration and can be appropriately applied for toxic/ischemic neuropathy as well.

All patients in the series were men, and all but one received the nerve block for repair of a torn rotator cuff. Rotator cuff injury is the most common diagnosis requiring shoulder surgery and is twice as common in men than in women, especially in those who are more than 40 yr of age.‖ Thus, it is not surprising that in our patient population that comprised men with a mean age of 58 yr; all but one received the nerve block for repair of a torn rotator cuff. It is important to identify that all 14 men patients were classified as overweight or obese according to their body mass index (>25 kg/m²), and the mean body mass index for the group (body mass index = 30.2 kg/m²) was in the obese range. Anatomical landmarks may be more difficult to palpate in heavier patients, forcing the physician to perform a less precise needle-stick(s) even with ultrasound guidance. Schroeder et al.[14] reviewed 528 consecutive patients undergoing ultrasound-guided interscalene nerve blocks, concluding a successful block is more anatomically challenging in this patient population, requiring increased procedural times and greater opioid administration to compensate for insufficient regional anesthesia. Overweight and obese patients may also be more likely to have metabolic syndrome or prediabetic conditions that include early, undiagnosed neuropathy. Other nerve entrapment conditions, such as: meralgia paresthetica, carpal tunnel syndrome, and sciatica, also have obesity as a risk factor.[15–17] Open or arthroscopic repair of a torn rotator cuff does not subject the phrenic nerve to any appreciable chance of injury and therefore cannot be considered a risk factor.[18] Anatomically, the course of the phrenic nerve in the neck and upper chest is well apart from the shoulder capsule and even adjacent tissue swelling would spare this region.

Large-scale, prospective assessments are necessary to determine the possible risk factors and better define appropriate candidates for regional anesthesia and postoperative infusions. Until those studies are undertaken, current practice of regional anesthetic blocks should continue to focus on technical accuracy, including use of ultrasound guidance, especially in patients with "difficult" anatomy.

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