Diaphragmatic Paralysis Following Thoracic Outlet Decompression: An Evaluation of Secondary Phrenic Nerve Reconstruction for Salvage

Matthew R. Kaufman,[1,2] Ajul Shah,[1] Robert T. Nevitt,[1] Reza Jarrahy,[1,2] and Cameron Santoro,[3,] Red Bank, New Jersey, and Los Angeles, California*

Background: Thoracic outlet syndrome (TOS) involves the compression of neurovascular structures within the thoracic outlet, defined as a space at the base of the neck, superior to the first rib and posterior to the clavicle. Surgical management most commonly requires a supraclavicular approach to allow for anterior and middle scalene muscle resection, cervical and first rib resection, and brachial plexus decompression. Pectoralis minor tenotomy and vascular decompression may also be indicated. While these interventions may successfully relieve compression within the thoracic outlet, they carry a risk of iatrogenic phrenic nerve injury (PNI). PNI-induced diaphragm paralysis often results in symptomatic pulmonary dysfunction. Phrenic nerve reconstruction may offer an effective treatment for functional diaphragm salvage after PNI following TOS surgery.

Methods: We performed a retrospective review of 17 patients with PNI following TOS surgery referred for management at a tertiary referral center who underwent phrenic nerve reconstruction to restore functional diaphragm activity.

Results: There were 17 patients (10 females and 7 males with a mean age of 47) who were treated for chronic, symptomatic PNI following TOS surgery. Two patients were lost to follow-up. The mean interval from PNI to secondary phrenic nerve reconstruction was 19 months. Following phrenic nerve intervention, 12 of 15 patients (80%) reported improvement, 10 of whom demonstrated partial or complete functional diaphragm recovery on objective analyses.

Conclusion: Surgical treatment for TOS has demonstrable therapeutic benefits to patients suffering from this disease. However, PNI is a potential complication associated with TOS surgery. Secondary phrenic nerve reconstruction to achieve salvage of diaphragm function can successfully address this risk and reduce or eliminate the morbidity associated with TOS-related PNI.

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[1]*The Plastic Surgery Center and Institute for Advanced Reconstruction, Red Bank, NJ.*

[2]*Division of Plastic and Reconstructive Surgery, David Geffen UCLA Medical Center, Los Angeles, CA.*

[3]*Advanced Reconstructive Surgery Alliance, Red Bank, NJ.*

Correspondence to: Cameron Santoro, BA, Advanced Reconstructive Surgery Alliance, 200 Schulz Dr, Red Bank, NJ 07701, USA; E-mails: mkaufmanmd@tpscnj.com or csantoro@arsahealth.com

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INTRODUCTION

Thoracic outlet syndrome (TOS) refers to a constellation of symptoms caused by compression of the neurovascular bundle located within the supraclavicular fossa or the costoclavicular space.[1] The disease was first described in 1956 as a thrombogenic vascular entity caused by arterial compression from a cervical rib (''cervical rib syndrome''). Since then, the clinical understanding of TOS has evolved to recognize a contributory neurogenic component to the development of symptoms.[2] Initial surgical approaches were focused on first rib resection. High recurrence rates associated with this limited intervention, however, eventually led to the integration of scalenectomy into primary surgical protocols.[2]

Despite decades of collective clinical experience, TOS remains a diagnostic and therapeutic challenge for physicians.[2,3] Although published evidence supports the neurogenic basis of the disease, many clinicians remain hesitant to recommend surgical intervention due to the intricate anatomy of the involved region and the inherent risks associated with surgical decompression, such as phrenic nerve injury (PNI).[4,5] Phrenic nerve dysfunction—whether resulting from chronic compression within the thoracic outlet or iatrogenic injury during scalenectomy—can lead to diaphragmatic paralysis, which significantly impacts respiratory mechanics and quality of life.[6] PNI has been reported in multiple reports evaluating TOS surgery with an incidence range of 1−11%, including transient and permanent PNI.[2,3,7,8] Although the risk of permanent PNI is 1−5% in most series, TOS surgery performed using a supraclavicular approach is likely associated with a higher occurrence of PNI than alternate techniques, particularly when scalenectomy is performed.[9]

The purpose of this study is to evaluate the efficacy of phrenic nerve reconstruction in TOS-related PNI to restore diaphragm function and alleviate respiratory deficits.

METHODS

Clinical Review

We identified 17 patients with chronic, symptomatic diaphragm paralysis secondary to permanent PNI following TOS surgery who underwent secondary phrenic nerve reconstruction. These patients presented through direct physician referrals or online inquiries to our tertiary referral center for diaphragm paralysis treatment between 2010 and 2026. They were selected from a larger cohort of over 900 patients who were evaluated and underwent phrenic nerve reconstruction for chronic, symptomatic diaphragm paralysis resulting from a variety of etiologies. We performed a comprehensive retrospective review of their medical records, collecting demographic data and documenting subjective and objective preoperative evaluations. These included chest fluoroscopy, pulmonary function testing (PFT) to identify restrictive pulmonary deficits, and electrodiagnostic evaluation (electromyography [EMG] and nerve conduction studies [NCSs]) to confirm and quantify the degree of PNI. Additional radiographic imaging (e.g., computed tomography [CT] or magnetic resonance imaging) was performed as indicated to exclude alternative etiologies of the (PNI). Patients who had undergone diaphragm plication were excluded from review ($n = 5$). Additional exclusion criteria included: asymptomatic diaphragm paralysis, BMI > 35, age > 70, absence of motor unit potentials on EMG testing, and major cardiovascular or metabolic risk factors.

Institutional Review Board approval was granted for this study and informed consent was obtained from each patient in accordance with study metrics. Assessment of the surgical technique included: procedures performed, operative time, length of hospital stay, and recorded complications. Patients were evaluated postoperatively for functional diaphragm recovery using chest fluoroscopy, PFT, EMG, and subjective reporting. Patient-reported outcomes were assessed using a previously published questionnaire that was based upon a validated evaluation of sleep-disordered breathing in patients with diaphragm weakness[10,11]:

Compared to preoperatively please assess the following:

1. Ability to bend at the waist
2. Comfort with lying flat
3. Ascending stairs
4. Exercise capacity
5. Sleep efficiency

For a successful result following phrenic nerve reconstruction, it was necessary to identify improvements in patient reported outcomes and objective diagnostic testing (i.e., recovery of diaphragm movement on chest fluoroscopy, improvement of PFT parameters, or improvement in diaphragm motor amplitude on EMG testing).

Phrenic Nerve Reconstruction Technique

Phrenic nerve reconstruction was performed using the supraclavicular scar from prior TOS surgery. The phrenic nerve and peripheral cervical root contributions (C3−C5) were explored throughout their entire

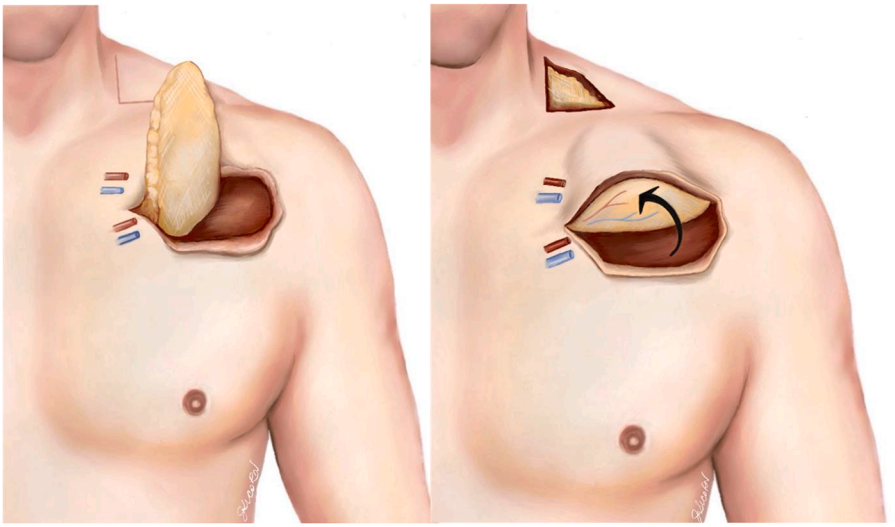

Fig. 1. Elevation and transfer of a pedicled adipo-fascial deltopectoral flap during phrenic nerve reconstruction.

course in the neck and decompressed via microsurgical neurolysis. Dense scar tissue from musculofascial fibrosis and vascular adhesions were meticulously separated from the cervical roots and the phrenic nerve. Sural nerve interposition grafting and/or neurotization (nerve transfer) from a regional donor nerve using end-to-side grafting methods was performed distal to the area of grossly identifiable nerve compression. Distal anastomosis of the sural nerve graft was performed in the thoracic cavity with a thoracoscopic or minithoracotomy approach when cervical adhesions precluded isolating healthy appearing phrenic nerve distal to the zone of injury. Collagen nerve protectors (Axogen, Tampa, FL) were placed around the phrenic nerve and nerve grafts to mitigate against the recurrence of adhesions postoperatively. If intraoperative assessment determined that the degree of devascularization of the wound bed and loss of domain in the supraclavicular space due to prior scalene muscle resection and scarring was severe, a deltopectoral adipo-fascial flap was harvested via a separate horizontal infraclavicular incision and transposed into the wound to cover the phrenic nerve and cervical roots with vascularized tissue and to eliminate dead space (Fig. 1). The flap was based medially on the upper internal mammary perforators to include the subcutaneous tissues superficial to the pectoralis major muscle.

Rehabilitation Protocol and Follow-Up Testing

Patients were directed to follow a specific protocol of diaphragm physical therapy and cardiovascular exercise beginning 4 weeks postoperatively. One year following surgery patients were requested to undergo repeat diagnostic testing, including: chest fluoroscopy, PFT, and EMG.

RESULTS

Between 2010 and 2026, 17 patients with post-TOS PNI and chronic, symptomatic diaphragm paralysis underwent phrenic nerve reconstruction (Table I). There were 10 females and 7 males in the cohort. Average patient age was 47 years (range: 21–60). All patients reported chronic, nonremitting respiratory symptoms—including exertional dyspnea, orthopnea, and sleep-disordered breathing—beginning immediately following TOS surgery and persisting without relief for at least 10 months following that index procedure. Ipsilateral diaphragm paralysis was confirmed on chest fluoroscopy in all patients. In all 17 patients, EMG/NCS testing demonstrated phrenic neuropathy and intact voluntary motor unit potentials, without any evidence of reinnervation or recovery. Eleven patients presented with right-sided PNI, whereas left-sided PNI was identified in 6 patients. In 1 patient there was a concomitant long thoracic nerve injury in addition to PNI. Five patients presented with symptoms consistent with residual TOS and/or brachial plexopathy (e.g., deltoid weakness, neuropathic extremity pain, and paresthesia).

All patients had previously undergone decompression surgery via a supraclavicular approach for TOS of neurogenic and/or vasculogenic origin. In 4

Table I. Demographic data and surgical details of patients who developed PNI post-TOS surgery

Patient	M/F	Age	Side	TOS subtype	TOS approach	Scalene resection	PNI duration (mos.)	Other post-TOS morbidity
1	F	55	L	nTOS	SC	A	12	+TOS
2	M	47	R	nTOS	SC	A	10	-
3[a]	F	59	R	nTOS	SC	A	34	-
4	M	44	L	nTOS	SC	A	24	-
5	M	60	R	nTOS, vTOS	SC, TA	A, M	22	+TOS, +Plexus
6	F	40	R	nTOS	SC	A, M	10	-
7	F	56	L	nTOS	SC	A, M	11	-
8	F	48	R	nTOS	SC	A, M	15	-
9	F	42	R	nTOS	SC	A, M	14	-
10	M	33	L	nTOS, vTOS	SC	A, M	12	+Plexus
11	F	44	R	nTOS	SC	A, M	26	-
12[a]	M	52	R	nTOS, vTOS	SC	A	10	-
13	F	51	R	nTOS	SC, IC, TA	A, M	10	+TOS, +Plexus
14	F	44	L	nTOS	SC	A, M	24	+TOS
15	F	50	R	nTOS	SC	A, M	60	-
16	M	21	L	nTOS	SC, IC	A, M	11	+LTN
17	M	54	R	nTOS	SC, IC	A, M	17	
	10 F, 7 M	Mean = 47	6 L, 11 R				Mean = 19	

nTOS, neurogenic thoracic outlet syndrome; vTOS, vasculogenic TOS; SC, supraclavicular; IC, infraclavicular; TA, transaxillary; A, anterior scalenectomy; M, middle scalenectomy; +TOS, residual TOS; +Plexus, brachial plexopathy; +LTN, long thoracic nerve injury; PNI, phrenic nerve injury; TOS, thoracic outlet syndrome.
[a]Patients 3 and 12 were lost to long-term follow-up.

patients an additional transaxillary or infraclavicular approach was used for neurovascular decompression. Anterior scalenectomy and brachial plexus decompression were performed in all patients along with first or cervical rib resection. In 12 patients, a middle scalenectomy was performed and in 2 patients a release of the pectoralis minor was part of the procedure.

The mean time interval from PNI to secondary phrenic nerve surgery was 19 months (range: 10–60 months). Phrenic nerve reconstruction was performed in all 17 patients. Four patients required thoracic access to facilitate distal nerve graft anastomosis and 6 patients underwent simultaneous adipo-fascial flap reconstruction due to severe supraclavicular scarring and loss of domain (Tables II and III). The average surgical time was 275 minutes. Patients were hospitalized for an average of 2 days. Two of the 17 patients were lost to follow-up.

In 12 of the 15 patients (80%) for whom long-term (>1 year) follow-up data were available there was patient-reported subjective symptomatic improvement. Partial or complete functional diaphragm recovery was confirmed on objective diagnostic testing in 10 of these patients (Tables II and III). Of the remaining 2 patients who reported improvement, one refused additional testing, and another was too early for follow-up evaluation. In 8 patients the subjective improvement was confirmed on chest fluoroscopy. PFT was evaluated in 5 patients and demonstrated improvements in percent predicted values of forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC). Diaphragm contractility improved in 7 patients that underwent postoperative EMG testing. Complications included surgical site seromas in 2 patients managed with percutaneous drainage, and a chyle leak in 1 patient that was resolved with conservative management.

DISCUSSION

The surgical management of TOS is primarily dictated by clinical subtype (neurogenic, venous, or arterial) and surgeon preference.[4] Neurogenic TOS (nTOS) is the most frequently encountered subtype, comprising 90–95% of cases, while vasculogenic TOS (5%) and arterial thoracic outlet syndrome (1%) are significantly less common.[7] The reported incidence of nTOS varies widely, ranging from 3 per 10,000 to 3 per 100,000 persons.[12] Traditionally, this subtype is managed initially through nonoperative means, specifically physical therapy

Table II. Surgical techniques and patient reported outcomes in patients undergoing secondary phrenic nerve reconstruction for chronic, symptomatic PNI following TOS surgery

Patient	Thoracic access	Adipofascial flap	Nerve reconstruction techniques	Operative complication	PRO
1	+		D, S		+
2			D, S		+
3[a]	+		D, S		N/A
4			D, S		+
5	+		D, S		
6			D, S	Seroma	+
7			D, S, T		+
8	+		D, S		+
9		+	D, S		
10		+	D, S		+
11		+	D, S		+
12[a]			D, S		N/A
13		+	D, S, T	Seroma	+
14		+	D, S	Chyle leak	+
15			D, S		+
16		+	D, S		+
17			D, S		
Totals	4/17 (24%)	6/17 (35%)		3/17 (18%)	12/15 (80%)

D, neurolysis; S, sural nerve interposition (''jump graft''); T, nerve transfer (end-to-side); PRO, patient-reported outcome; PNI, phrenic nerve injury; TOS, thoracic outlet syndrome.
[a]Patients 3 and 12 were lost to follow-up.

and muscle relaxant medications. While conservative management remains the first choice, particularly for nTOS, surgical intervention is indicated for patients with persistent symptoms despite nonsurgical efforts, or in cases involving thromboembolic events, chronic vascular occlusion, or progressive neurologic deficits.[12]

The supraclavicular approach has proven effective for managing nTOS.[9] This technique offers substantial exposure of the middle and upper brachial plexus trunks under direct visualization of surrounding critical neurovascular structures with a low risk of injury to the C8 and T1 roots, the lower plexus trunk, and the long thoracic nerve. Furthermore, this approach avoids trauma to the intercostobrachial cutaneous nerve and permits a comprehensive anterior and middle scalenectomy, including excision of the scalene minimus or other compressive anatomical anomalies detected intraoperatively. The phrenic nerve requires exposure and manipulation during anterior and middle scalenectomy, likely resulting in a higher incidence of PNI.

The phrenic nerve provides the sole motor innervation to the diaphragm, the primary muscle of respiration. During inspiration, diaphragmatic contraction and descent facilitate passive lung expansion. Unlike skeletal muscles used for intermittent locomotion, the diaphragm must maintain near-constant activity to support functional respiratory cycles. Any iatrogenic interference—whether through traction, thermal injury, or transection—may disrupt this critical electromechanical coupling.[6] Iatrogenic PNI is reported in up to 11% of surgical treatments for thoracic outlet obstruction, compared to 10% after cardiac surgery and 1% after interscalene blocks.[7,8,13] Compromise of phrenic nerve anatomy and conduction culminates in diaphragmatic paralysis or paresis, precipitating significant pulmonary dysfunction and a diminished quality of life.[6]

PNI typically arises from iatrogenic, traumatic, or idiopathic origin.[13–16] Iatrogenic causes are the most prevalent etiology, frequently occurring during cardiac or mediastinal surgeries, as well as interscalene blocks for orthopedic procedures.[17] Traumatic mechanisms often involve traction injuries where the neck is forcefully distracted from the shoulder. While such mechanisms are traditionally associated with brachial plexus avulsions, they have become increasingly recognized as a cause of isolated or concurrent diaphragmatic paralysis without other plexus-related deficits.[17]

Cases of phrenic nerve dysfunction lacking a definitive iatrogenic or traumatic cause are classified as idiopathic. Many of these cases, however, are being increasingly attributed to Parsonage-Turner syndrome, an inflammatory plexopathy that can

Table III. Diagnostic evaluation of diaphragm recovery in patients undergoing secondary phrenic nerve reconstruction after PNI following TOS surgery who self-reported improvement

Patient	PRO	Chest fluoroscopy	PFT (ΔFEV1%, ΔFVC%)	EMG (Δ%)
1	+	+		
2	+	+	+(45%, 39%)	
4	+	+		+(334%)
6	+		+(19%, 30)	+(100%)
7	+			
8	+	+	+(29%, 15%)	+(45%)
10	+	+		+(100%)
11	+	+		+(200%)
13	+	+	+(12%, 39%)	
14	+	+	+(14%, 6.5%)	+(100%)
15	+			+(80%)
16	+			
	N = 12	N = 8	N = 5	N = 7

Patient 7 reported improvement but refused testing. Patient 16 reported improvement but was too early for evaluation as of most recent follow-up.

ΔFEV1%, ΔFVC%: Percent improvement in percent predicted value for forced expiratory volume in 1 second and forced vital capacity following phrenic nerve reconstruction.

EMG (Δ%): Percent improvement in diaphragm motor amplitude following phrenic nerve reconstruction.

PRO, patient-reported outcome; PFT, pulmonary function testing; PNI, phrenic nerve injury; TOS, thoracic outlet syndrome; EMG, electromyography.

manifest as sudden onset diaphragmatic weakness.[18]

Intraoperative findings in our cohort of patients during phrenic nerve reconstruction after TOS surgery with PNI consistently identified dense scar compressing the phrenic nerve and C4-C5 cervical root contributions, adherent fibrotic remnants of anterior and middle scalene muscle, and nerve traction due to loss of domain in the thoracic outlet. These observations suggest that while complete scalenectomy aims to decompress the thoracic outlet, it may inadvertently introduce new compressive forces or scarring that negatively impacts phrenic nerve electrophysiology.

The pathological factors that were identified as contributing to loss of phrenic nerve conduction and diaphragm paralysis after TOS surgery included compressive forces impacting the C4 and C5 motor contributions to the phrenic nerve in addition to the phrenic nerve itself. In normal anatomy, the proximal muscular and tendinous portions of the anterior and middle scalene muscles are in close adjacency to the C4 motor root contribution to the phrenic nerve, whereas the C5 contribution is along the mid or distal portion of the anterior scalene muscle. The C4 motor root provides most of the phrenic nerve axonal density with the remainder provided mainly by the C5 root.[19] Transection and excision of the anterior and middle scalene muscles appears to result in cephalic and caudal contraction, bunching, and fibrosis of the musculotendinous remnants resulting in severe compressive adhesions along the C4 motor root and traction between the phrenic nerve and the C5 root contribution. Nerve transfers were required in 2 patients in whom severe, proximal C4 motor root compression was accompanied by visual and electrodiagnostic evidence of neuroforaminal stenosis.

Another site of significant nerve pathology was identified at the base of the neck, approaching the mediastinal inlet. Severe compressive adhesions ramifying around the phrenic nerve were seen from thickened prevertebral fascia, fibrotic remnants of the anterior scalene muscle, and vascular adhesions from scarred transverse cervical vessels. In this series, 4 patients required thoracic exposure for distal phrenic nerve grafting due to severe neural pathology along the phrenic nerve that extended into the upper mediastinum.

The clinical spectrum of PNI ranges from mild diaphragmatic paresis to complete paralysis.[6] While bilateral injury typically requires supplemental oxygen therapy or mechanical ventilatory support, unilateral paralysis—the most common phenic morbidity associated with TOS surgery—results in ipsilateral inactivity of the lower lung and reduced gas exchange.[6] This physiological deficit manifests clinically as exertional dyspnea and orthopnea. Over time, chronic hypoinflation can lead to secondary obstructive lung disorders and an increased susceptibility to respiratory infections.[6,20]

Functional paralysis is confirmed through a fluoroscopic ''sniff test,'' which is a reliable radiographic method for documenting absence of diaphragmatic excursion.[21] It is often associated with paradoxical diaphragm movement during rapid inspiration. Additionally, PFT is utilized to measure the severity of the restrictive ventilatory deficit.

NCS and EMG remain essential for quantifying neuromuscular dysfunction. EMG identifies whether voluntary motor potentials are preserved, which determines the feasibility of functional restorative procedures such as nerve grafting or nerve transfers.

Historically, adult diaphragmatic paralysis was managed conservatively. In the absence of effective surgical treatments, patients were often advised to simply adapt to their respiratory limitations. The introduction of diaphragmatic plication to the treatment protocol of paralysis offered a surgical option;

it remains the standard ''static'' treatment option.[22] By securing the diaphragm in a permanent inspiratory position, the procedure increases lung volume, minimizes paradoxical motion, and corrects ventilation-perfusion mismatch.[22,23] While plication improves total lung capacity and arterial PaO_2, it unfortunately does not restore active contraction, and recurrent diaphragm elevation is reported in up to 19% of adult cases.[23] Even with the application of minimally invasive video-assisted thoracic surgery or robotic approaches to plication surgery, patients may experience postoperative discomfort for up to 6 months.[23,24]

Phrenic nerve reconstruction that combines neurolysis, nerve grafting, and neurotization has emerged as an alternative surgical approach to diaphragm paralysis that results in functional restoration of spontaneous diaphragm excursion.[25] In a landmark series of 400 patients, Kaufman et al. (2021)[13] demonstrated that phrenic nerve reconstruction is an effective treatment for symptomatic PNI. At 2-year follow-up, patients showed a 22% improvement in FEV1, an 18% improvement in FVC, and an 82% improvement in diaphragm motor amplitude on EMG. Furthermore, an 83% rate of favorable patient-reported outcomes supports reconstruction to restore physical activity and improve sleep quality.[13] Significantly, patients who fail to improve after reconstruction may still undergo diaphragm plication, whereas a prior plication typically serves as a contraindication for nerve reconstruction. Unlike the immediate mechanical stabilization that characterizes plication, reconstruction relies on gradual neuromuscular functional restoration as the phrenic nerve regenerates.

The risk of PNI following TOS surgery as reported in this series may prompt a discussion regarding technique modification. Considerations for future analysis may include segmental scalenectomy instead of comprehensive muscle removal to avoid cephalic and caudal contraction, and prophylactic application of a deltopectoral adipo-fascial flap to reduce the postoperative dead space.

CONCLUSION

The surgical management of TOS is complex. TOS surgery is associated with potential risk to the phrenic nerve due to proximity of the nerve to the structures that are targeted in thoracic outlet decompression. If PNI occurs, phrenic nerve reconstruction has emerged as a promising functional alternative to diaphragm plication to restore diaphragm function and address pulmonary morbidity. Future studies should evaluate whether modification of TOS surgical techniques could minimize the risk of PNI.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Matthew R. Kaufman: Writing — review & editing, Validation, Supervision, Resources, Investigation, Formal analysis, Data curation. **Ajul Shah:** Writing — review & editing, Validation, Formal analysis. **Robert T. Nevitt:** Writing — review & editing, Validation, Formal analysis. **Reza Jarrahy:** Writing — review & editing, Validation, Formal analysis. **Cameron Santoro:** Writing — review & editing, Writing — original draft.

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