A Review of Current Evidence in the Surgical Treatment of Migraine Headaches

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Summary: Migraines affect 18% of women and 6% of men and result in an estimated $1 billion in medical costs and $16 billion productivity loss in the United States annually. Migraine headaches persist as a problem of this scale because pharmacologic treatments for migraines are frequently incompletely effective, resulting in a population of patients with significant residual disability. In the last decade, novel approaches to the treatment of migraines have been developed based on the theory that extracranial sensory branches of the trigeminal and cervical spinal nerves can be irritated, entrapped, or compressed at points throughout their anatomic course, ultimately leading to the cascade of physiologic events that results in migraine. Botulinum toxin (Botox) injection and surgical decompression of these trigger points have been shown to reduce or eliminate migraines in patients who are incompletely treated by traditional medical management. Despite the recent advances made with Botox, this treatment strategy most commonly results in only temporary migraine prevention. However, the evidence supporting the efficacy and safety of permanent surgical decompression of peripheral trigger points is accumulating rapidly, and the overall success rate of surgery has approached 90%. In addition, an abundance of literature investigating the precise anatomical dissections associated with trigger points has been published concurrently. This article reviews the most up-to-date clinical and anatomic evidence available and seeks to provide a comprehensive, concise resource for the current state of the art in the surgical treatment of migraine headaches. (*Plast. Reconstr. Surg.* 134: 131S, 2014.)

Migraine headache is a widespread neurovascular disorder that is often inadequately treated by existing medical therapies, resulting in a population of patients with significant residual disability. Migraines affect over 35 million Americans a year, with a striking preponderance in women (18% of women versus 6% of men), with a cumulative lifetime risk of 43% in women and 18% in men, most before the age of 35.[1,2] Migraine symptoms commonly interfere with daily function and result in an estimated $1 billion in medical costs and $16 billion in lost productivity per year in the United States alone[3] and are the 12th leading cause of disability among women worldwide.[4] Most migraine sufferers require a combination of pharmacological treatment and avoidance of common environmental triggers to manage their symptoms, oftentimes with variable effectiveness. In addition, standard pharmacologic therapies, including prophylactic, acute abortive, and acute analgesic therapy, are accompanied by numerous side effects that can preclude their use.[5] The etiology of migraine headache has been classically described as a central

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phenomenon. However, extracranial trigger sites have been recently identified, and decompression of these peripheral trigger sites has been shown to have significant impact on symptomatology.[6–13] There is growing evidence within the field of neurobiology, which supports the concept of a peripheral nerve trigger in the initiation of migraine headaches.[14] A recent study also suggests, through electron microscopy and proteomic analysis, that there are biostructural differences in myelin found in peripheral nerves surgically excised from patients with migraines compared with peripheral nerves from patients without migraines (Guyuron et al, manuscript in preparation, 2014). This study may further support the role of a peripheral mechanism in the complex migraine cascade. Botulinum toxin-A (Botox) injections have been recognized and Food and Drug Administration–approved as an effective temporary preventative therapy for chronic migraine headaches,[15–17] and surgical decompression or neurectomy of select injection sites has been used to achieve long-term improvement of patients with diagnosed peripheral nerve compression.[6–13]

SURGICAL TECHNIQUE

The surgical treatment of migraine headaches currently involves operative decompression of 4 major peripheral trigger sites although there are other more infrequent potential sites of compression. Typically, patients are selected after having a confirmed diagnosis of migraine headache or chronic daily headache by a neurologist and after failure of conservative medical management. Early data have advocated for the use of chemodenervation by Botox to identify and verify which trigger sites are affected in a patient-specific, step-wise progression.[7,9,10,12,13] Although response to Botox is a positive prognostic indicator for surgical success,[18] recent data also suggest that a constellation of symptoms obtained through a thorough history and physical is equally as efficacious in predicting which sites will be amenable to surgical decompression.[19] As an alternative, many surgeons also use a diagnostic peripheral nerve block with a local anesthetic to identify trigger sites that will be amenable to surgical decompression. The 4 sites addressed include a frontal trigger, temporal trigger, occipital trigger, and a nasoseptal trigger in the setting of septal deviation or turbinate hypertrophy, which results in aberrant contact points.

The frontal trigger site is thought to exist as a result of compression or irritation of the supraorbital nerve (SON) and supratrochlear nerve (STN) branches of the ophthalmic division of the trigeminal nerve (V1). This site is addressed by decompression of the corrugator myofascial unit through subtotal resection of the corrugator supercilli muscles (CSM) or by resection of the entire glabellar muscle group including the corrugator supercilii, depressor supercilii, and procerus muscles. This is achieved through either a transpalpebral approach (**See Video 1, Supplemental Digital Content 1**, which demonstrates decompressing the frontal trigger site using the transpalpebral approach, available in the "Related Videos" section of the full-text article on PRSJournal.com or, for Ovid users, available at ***http://links.lww.com/PRS/B93***.) or with an endoscopic approach through small hairline incisions.[10] (**See** Video 2**, Supplemental Digital Content 2**, which demonstrates decompressing the frontal and temporal trigger sites using the endoscopic approach, available in the "Related Videos" section of the full-text article on PRSJournal.com or, for Ovid users, at ***http://links.lww.com/PRS/B94***.) Additionally, the focal anatomical site at the bony orbital

Video 1. Supplemental Digital Content 1. Decompressing the Frontal Trigger Site: Transpalpebral Approach. This video demonstrates an incision made in the upper tarsal crease after injection of xylocaine containing 1:100,000 epinephrine. Electrocautery is used to incise the orbicularis oculii, and the dissection is carried cephalically in a plane between the orbicularis muscle and the orbital septum to expose the depressor supercilii and corrugator muscles. The depressor supercilii muscle is then resected followed by the corrugator muscles. The fascia of the superorbital notch is then released followed by resection of the procerus muscle along with resection of a portion of the supratrochlear artery. A piece of fat which is harvested from the medial compartment of the upper eyelid is used to replace the removed volume of the muscle and secured with a 6-0 monocryl suture. A 6-0 fast absorbable catgut suture is used in a subcuticular fashion to repair the skin. This video is available in the "Related Videos" section of the full-text article on PRSJournal.com or, for Ovid users, at ***http://links.lww.com/PRS/B93***. Copyright Bahman Guyuron, MD.

Video 2. Supplemental Digital Content 2. Decompressing the Frontal and Temporal Trigger Sites: Endoscopic Approach. This video demonstrates the endoscopic approach performed through five incisions. The scalp is injected with xylocaine containing 1:200,000 epinephrine and the forehead is injected with xylocaine containing 1:100,000 epinephrine. After the incisions are made, baby Metzenbaum scissors are used to dissect the soft tissues down to the deep temporal fascia. The soft tissues are dissected off the deep temporal fascia using the Obwegeser periosteal elevator until the zygomaticotemporal branch of the trigeminal nerve is exposed and then is gently avulsed. The deep temporal fascia is pierced medially, immediately above the zygomatic arch, and a piece of fat is harvested to be applied in the corrugator muscle site. The depressor supercilii and corrugator muscles are then resected, and the harvested fat is placed to replace the muscle volume. This video is available in the "Related Videos" section of the full-text article on PRSJournal.com or, for Ovid users, at ***http://links.lww.com/PRS/B94***. Copyright Bahman Guyuron, MD.

Video 3. Supplemental Digital Content 3. Decompressing the Occipital Trigger Point. This video demonstrates a vertical incision made in the midline of the occiput. The dissection is deepened and shifted laterally, and the trapezius fascia is incised. While protecting the nerve, a rectangular segment of the muscle is isolated medial to the nerve and then transected caudally. The muscle is then reflected caudally and a 2 cm length of the muscle is removed en bloc. The fascial bands over the lateral portion of the nerve are released similar to carpal tunnel surgery. A subcutaneous flap is elevated, and then is passed under each greater occipital nerve and sutured to the midline. The two flaps are sutured to the midline raphe by passing the suture through the raphe and catching the contralateral flap, bringing it back, and tying it on the ipsilateral side, preventing the flap from retracting laterally. This video is available in the "Related Videos" section of the full-text article on PRSJournal.com or, for Ovid users, at ***http://links.lww.com/PRS/B95*.** Copyright Bahman Guyuron, MD.

rim can be addressed as well, and depending on symptoms and computed tomography findings, foraminotomy or fasciotomy of the supraorbital notch and arterectomy can be added to the approach to achieve superior results.

The temporal trigger site is attributed to compression of the zygomaticotemporal nerve (ZTN) branch of the maxillary division of the trigeminal nerve (V2). To address this trigger site, a small extension of the transpalpebral incision or an endoscopic approach is used. A segmental neurectomy of the ZTN below the level of the temporal fascia is then performed. The ZTN is a nerve that is commonly transected during forehead rejuvenation procedures without consequence. A current study evaluating the efficacy of decompression versus resection of this nerve has demonstrated no statistical difference between these 2 techniques (Guyuron et al, manuscript in preparation).

The occipital trigger point is addressed by decompression of the greater occipital nerve (GON), which is a terminal sensory branch of the dorsal rami of the second cervical spinal nerve (C2). Recent hypotheses suggest that compression of the lesser occipital nerve (LON) and third occipital nerve (TON) may also contribute to the initiation of migraine headache.[20] However, decompression of the TON has not demonstrated robust clinical benefit.[21] The GON is approached on the posterior scalp at the occiput, either through a 4-cm vertical midline incision or transverse incision(s), depending on surgeon's preference. The predominant area of compression was originally described at the intersection of the GON and the semispinalis capitis muscle although a total of 6 areas of compression have subsequently been elucidated.[22] Decompression should be performed proximally to at least the level of the oblique capitis inferioris muscle. A segment of the semispinalis capitis is removed, approximately 1 cm wide and 2.5 cm in length, medial to the GON. A segment of autologous fat is then placed between the GON and the muscle to prevent further entrapment postoperatively. (**See Video 3, Supplemental Digital Content 3**, which demonstrates decompressing the occipital trigger point, available in the "Related Videos" section of the full-text article on PRSJournal.com or, for Ovid users, at ***http://links.lww.com/PRS/B95***.)

Finally, for patients with intranasal abnormalities such as a deviated septum, septal spurring, concha bullosa, or turbinate hypertrophy, septoplasties with or without turbinectomies (inferior, middle or superior) are typically performed for elimination of aberrant contact points.

CLINICAL EVIDENCE

Like many impactful scientific discoveries, the treatment of migraine headaches through surgical intervention was a serendipitous finding. After anecdotal reports of migraine symptom improvement in patients who underwent CSM resection as part of an endoscopic browlift procedure for forehead rejuvenation, Guyuron et al[11] surveyed a population of patients to quantify the observation. In this study, which examined 39 patients with preexisting migraine who underwent CSM resection, 31 (79.5%) experienced elimination or significant improvement in their migraines. In response to these findings, Guyuron et al[10] piloted a prospective cohort study where 22 patients underwent either transpalpebral CSM resection or endoscopic CSM resection with concurrent ZTN resection in response to trigger points identified by a preoperative series of Botox injections. In this study, the authors found that 21 of the 22 patients (95.5%) experienced complete elimination or significant improvement in their migraine headaches, as assessed by a greater than 50% reduction in symptoms, with a mean follow-up of 347 days. Since the original reports, a total of 17 clinical studies have now been published evaluating the efficacy and safety of the surgical treatment of migraine headaches (Table 1).

Dirnberger and Becker[6] were the first to describe the reproducibility of Guyuron's technique in another prospective cohort study that demonstrated elimination or significant improvement in migraine headaches for 41 of 60 (68.3%) patients who underwent surgical decompression of the frontal trigger point with a mean follow-up of 12.8 months. In 2005, Guyuron et al[7] performed the first randomized controlled trial by enrolling 125 patients with a 4:1 allocation for the treatment and control arms, respectively. This trial investigated the simultaneous decompression of all 4 trigger points in a patient-specific manner which depended on the results of a Botox injection series. Of the 4 trigger points, 80 patients, 71 patients, 34 patients, and 62 patients underwent decompression of the frontal, temporal, occipital, and septal trigger points, respectively. In this study, 82 of 89 (92%) patients who met the appropriate follow-up criteria experienced elimination or significant improvement in their migraine headaches, with a mean follow-up of 396 days. Migraine symptom severity was also tracked through the Migraine Headache Index, which is an assessment tool that combines migraine frequency, severity, and duration. In 2008, Poggi et al[13] reported a retrospective case series in which 16 of 18 patients (92.1%) benefited from surgical decompression of the frontal, temporal, and occipital triggers sites after identification with a Botox injection series with a mean follow-up of 16 months. Then, in 2009, Guyuron et al[9] performed the true gold standard for a surgical randomized controlled trial with a sham surgery or placebo-controlled trial. Here, 41 of 49 patients (83.7%) experienced migraine elimination or significant improvement, with 28 patients (57.1%) reporting complete elimination. This was significantly different than the sham-surgery group where 15 of 26 patients (57.7%) ($P < 0.05$) reported improvement, with only 1 patient reporting migraine elimination (3.8%) ($P < 0.001$) after 1-year follow-up.

In 2011, Janis et al[12] corroborated the use of these techniques by reporting a case series where 24 patients underwent decompression of peripheral trigger points guided by the results of their individualized Botox injection series. In this study, 19 of 24 patients experienced either complete elimination or significant improvement of migraine headaches with a mean follow-up of 661 days. Finally, in 2011, Guyuron et al[8] reported the long-term outcomes for patients who had undergone decompressive surgery by describing the 5-year results of the first randomized controlled trial. Here, 69 of 89 patients who underwent surgical decompression were available for follow-up after 5 years, and 61 of 69 patients (88%) experienced either complete elimination or substantial improvement of migraine headaches. That same year, Larson et al[23] reported factors that predicted surgical failure versus success in outcomes in a retrospective chart review of 169 patients who had undergone decompressive surgery. Importantly, the authors found that surgery was more successful when all 4 trigger sites were addressed and, conversely, that surgery was more likely to fail with fewer trigger sites included. Increased intraoperative bleeding was also associated with surgical failure, which may have caused increased inflammation and scarring of tissues, resulting in persistent nerve impingement postoperatively.

Several recent studies have provided details that are helpful in fine-tuning the toolbox of surgical procedures for migraine trigger point decompression.

Table 1. Summary of Clinical Studies of the Surgical Treatment of Migraine Headache

References	Study Design	No.	Months Follow-up	% Migraine-free	% With Improvement	Trigger Points Addressed	Adverse Events
Guyuron et al[11]	Case series	39	46.5	38.5%	79.5%	F	Not reported
Guyuron et al[10]	Cohort-prospective	22	11.4	45.5%	95.5%	F, T	Transient surgery site numbness
Dirnberger and Becker[6]	Cohort-prospective	60	12.8	28.3%	68.3%	F	Transient surgery site paresthesia
Guyuron et al[7]	RCT	89	13	34.8%	92.1%	F, T, O, N	Temporary nasal dryness, rhinorrhea, slight recurrence of septal deviation, intense itching, minor hair loss, temporary neck stiffness
Poggi et al[13]	Case series	18	16	16.7%	94.4%	F, T, O	Itching, numbness, scar alopecia
Guyuron et al[9]	RCT (sham-controlled)	49	12	57.1%	83.7%	F, T, O	Numbness, temporal hollowing, temporary itching, uneven brow movement, temporary hair loss or thinning, residual corrugator function, neck stiffness
Guyuron et al[8]	RCT	69	60	29.0%	88.4%	F, T, O, N	Neck stiffness or weakness, numbness, hypersensitivity
Janis et al[12]	Case series	24	21.7	8.3%	79.2%	F, T, O, N	Immediate post-op headache, transient surgery site paresthesia, incisional alopecia, periorbital ecchymosis
Larson et al[23]	Cohort-retrospective	169	>11	39.1%	78.7%	F, T, O, N	Not reported
Ducic et al[26]	Cohort-prospective	25	—	—	—	O	Not reported
Chepla et al[28]	Cohort-retrospective	86	12	—	100.0%	F	Not reported
Liu et al[19]	Cohort-retrospective	335	12	35.2%	83.3%	F, T, O, N	Not reported
Liu et al[30]	Cohort-retrospective	253	>12	63.2%	86.6%	F	Scalp paresthesia, forehead asymmetry, frontalis paralysis, eyebrow elevation, dimpling on animation
Chmielewski et al[25]	Cohort-retrospective	170	>12	55.9%	87.6%	O	Not reported
Lee et al[18]	Cohort-retrospective	188	>12	34.6%	86.2%	F, T, O, N	Not reported
Lee et al[21]	Cohort-retrospective	229	>6	27.5%	80.5%	O	Not reported
Ducic et al[27]	Cohort-retrospective	71	33	—	70.4%	O	Numbness, hypersensitivity

Trigger points: F, frontal; T, temporal; O, occipital; N, nasal. Adverse events listed were found in at least 5% of patients when incidence was reported.

Migraine headache is commonly described as a pulsatile phenomenon with a strong vascular component to its etiology, and there is a known intimate anatomical relationship between the GON and the occipital artery (OA).[24] It was hypothesized that OA irritation of the GON could be one factor contributing to migraine pathogenesis. In 2013, Chmielewski et al[25] described a series of patients who underwent GON decompression with or without OA ligation as a component of the procedure. The authors found that OA ligation was not contributory to the success of GON decompression and that this step was likely not necessary. It is also now known that OA vasculitis is not contributory to the pathogenesis of headaches caused by occipital neuralgia. In 2011, Ducic et al[26] described a series of 25 patients who underwent GON decompression where 15 patients also underwent simultaneous OA resection. Histologic analysis of the resected arterial segments provided no evidence for vasculitis.

Frequently, the TON is sacrificed during decompression of the GON, and a retrospective review by Lee et al[21] in 2013 investigated the impact of TON resection on patient outcomes. In this study, 111 patients with TON resection in the setting of GON decompression and 118 patients without TON resection were compared. The authors found that removing the TON did not affect surgical success, and there were no differences in patient outcomes between the groups, although the limitations of a retrospective review, including variation in technique and patient selection, might warrant further prospective analysis. A subset of patients who undergo occipital decompression because of migraines caused by occipital neuralgia do not respond to typical decompressive procedures. In this setting, Ducic et al[27] described in 2014 that GON resection, as opposed to decompression, is a valid treatment option with surgical success and relief of migraines in 70.4% of patients.

The frontal trigger site is the most commonly reported trigger point among patients seeking treatment for migraine headaches by surgical decompression. In 2012, Chepla et al[28] hypothesized that incomplete decompression of the frontal trigger site may be related to the presence of a supraorbital foramen, as opposed to a supraorbital notch, in a percentage of patients. Indeed, as many as 25% of people possess a supraorbital foramen.[29] In this study, 2 groups were compared: glabellar muscle group resection alone versus glabellar muscle group resection with concurrent supraorbital foraminotomy. The authors demonstrated that muscle resection with supraorbital foraminotomy was the superior approach, with a significant reduction in Migraine Headache Index compared with muscle resection alone. In 2012, Liu et al[30] compared the success rate of endoscopic versus transpalpebral approaches and found that the endoscopic approach has a higher success rate. The authors postulate that this may result from more complete resection of the CSM, especially the lateral component, and easier visualization and identification of supraorbital foramen and accessory SON branches.

ANATOMICAL STUDIES

In addition to the abundance of clinical data supporting the surgical treatment of migraine headaches, complimentary anatomical studies have been published in parallel detailing dissections pertinent to migraine trigger sites (Table 2). The frontal trigger site was first analyzed by Janis et al[31,32] in a 2-part cadaver dissection series in 2007 and 2008. In this report, the authors described the dimensions of the CSM in relation to clinically relevant bony landmarks. These findings are pertinent for CSM injection and subsequent resection because lower early surgical success rates in some studies were attributed to failing to appreciate the lateral extent of the glabellar complex which resulted in incomplete resection. In the second part of the series, Janis et al[32] described 4 branching patterns of the SON in relation to the CSM, with a 78% incidence of nerve/muscle interactions and a 22% incidence of nerve branching cephalad to the CSM, which may have implications on the success of decompression. Fallucco et al[29] subsequently described the presence of supraorbital foramina as potential sites for nerve compression and also provided a classification system for 4 types of fascial band constriction that can occur at the supraorbital notch. To further investigate anatomical structures implicated in the frontal trigger point, Janis et al[33] described 3 potential points of compression of the STN, as well. These included compression of the STN as it enters the brow through a frontal notch or foramen, where it can be compressed by a fibrous band, and at both the entrance and exit of the nerve through the CSM. These detailed descriptions have provided the tools necessary for surgeons to ensure complete decompression of the frontal trigger point.

Two studies have detailed the anatomy pertinent to the temporal trigger point. In 2005, Totonchi et al[34] described the course of the ZTN as it emerges from the temporalis muscle, its relative distance to the lateral canthus, and provided a

Table 2. Key Findings from Anatomical Studies

References	Anatomy	Source	Key Findings
Mosser et al[36]	GON	20 cadavers (28 nerves)	The GON emerges 3 cm below the occipital protuberance and 1.5 cm lateral to midline.
Dash et al[20]	LON, TON	16 and 13 cadavers (30 and 22 nerves)	The LON emerges 65 ± 12 mm lateral to midline and 53 ± 16 mm below the line between the EACs. The TON is 13 ± 5 mm from midline and 62 ± 20 mm below the line between the EACs.
Totonchi et al[34]	ZTN	20 patients undergoing endoscopic forehead surgery	The ZTN emerges on average 17 mm from the palpebral fissure in the posterolateral direction and 6.5 mm in the cephalad direction; three accessory branch patterns (cephalad, lateral, and immediate vicinity) are described.
Janis et al[31]	Corrugator	25 cadavers (50 corrugators)	The CSM origin begins 3 ± 1 mm medial to the nasion and extends 14 ± 3 mm laterally. It inserts 43 ± 3 mm from the nasion or 8 ± 3 mm medial to the LOR. The muscle apex is 33 ± 3 mm cephalad to the nasion-LOR plane and 18 ± 4 mm medial to the LOR.
Janis et al[32]	SON	25 cadavers (50 supraorbital nerves)	There are 4 branching patterns of the deep and superficial divisions of the SON: in type I (40%) the deep division sends branches into the CSM; in type II (34%) both the superficial and deep divisions enter; in type III (4%) only the superficial enters; in type IV (22%) the branches occur cephalad to the CSM.
Ducic et al[37]	GON, LON	112 patients and 13 cadavers	The GON pierces the semispinalis; it branches in the semispinalis or in the trapezial tunnel in 6% of patients; 44% have GON asymmetry; the LON follows the posterior border of the SCM.
Janis et al[24]	GON, OA	25 cadavers (50 nerves)	The GON and occipital artery intersect 54% of the time either at a single point or with helical intertwining.
Janis et al[42]	GON	25 cadavers (50 nerves)	The GON has 6 potential compression points: (1) between the semispinalis and the obliquuscapitis inferior; (2) at the entrance to and (3) exit from the semispinalis; (4) at the entrance into the trapezius and (5) exit from the trapeziusfascial insertion into the nuchal line; (6) at the intersection with the occipital artery.
Janis et al[35]	ZTN	25 cadavers (50 nerves)	The ZTN has an intramuscular course 50% of the time; the nerve foramen on average is 7 mm lateral to the LOR and 8 mm cranial to the nasion-LOR line.
Chim et al[43]	ATN	10 cadavers (20 nerves)	The ATN has 3 potential compression points: two preauricularfascial bands and a nerve-superficial temporal artery investment found in 80% of nerves.
Fallucco et al[29]	Supraorbital rim	30 cadavers (60 nerves)	A supraorbital notch occurs 83% of the time; 86% of notches are encircled by a fascial band in one of four patterns.
Janis et al[33]	STN	25 cadavers (50 nerves)	The STN has 3 potential compression sites: (1) the frontal notch or foramen, (2) its entrance into the corrugator, and (3) its exit from the corrugator; it branches within the retro-orbicularis oculi fat pad and enters the corrugator in one of four patterns.
Junewicz et al[38]	GON	272 patients	The trapezius extends to midline in 67% of patients; therefore, muscle fiber orientation better distinguishes the trapezius (oblique fibers) from the semispinalis (vertical). The GON branches in 7% of patients; occipital arterectomy was required in 64% of patients and resection of a lateral segment of semispinalis was required in 11% of patients.

EAC, external auditory canal; LOR, lateral orbital rim; SCM, sternocleidomastoid muscle; TON, third occipital nerve. Measurements rounded to the nearest millimeter.

description of accessory branching patterns. The authors found that the main branch of the ZTN emerges at approximately 17 mm posterolateral and 6.5 mm cephalad to the lateral canthus and that there are 3 accessory branching patterns: a lateral accessory branch, a cephalad accessory branch, or immediate branches in the vicinity of the main ZTN. In 2010, Janis et al[35] provided additional information detailing the muscular course of the ZTN and the location of the nerve's foramen. In this study, the authors found that 50% of the time the ZTN had an intramuscular course while 50% was extramuscular. When intramuscular, 22% of the time the ZTN had a short, straight

trajectory while the remaining 28% were long and tortuous. Additionally, the location of the nerve foramen was elucidated to be on average 6.70 mm lateral to the orbital rim and 7.88 mm cranial to the nasion-lateral orbital rim line. The data presented in these studies provide highly useful landmarks for chemodenervation of the temporal trigger point and the information needed to achieve adequate surgical decompression.

Several studies have addressed the anatomy pertinent to the occipital trigger point. In 2004, Mosser et al[36] described the course of the GON from the semispinalis to the superior nuchal line in a cadaveric dissection study. Using the midline and the occipital protuberance as landmarks, the emergence of the GON from the semispinalis was found to occur at 1.5 cm lateral to the midline and 3 cm below the occipital protuberance. In 2009, Ducic et al[37] confirmed these findings by describing the same location of emergence from the semispinalis, but also commented that the GON course is asymmetric in 43% of people. The authors described the average diameter of the GON at approximately 4 mm, while the LON was approximately 1 mm in diameter on average, thus allowing for reliable distinction between the structures. Further, the LON was found to be reproducibly located along the border of the sternocleidomastoid. In 2010, Janis et al[24] described the relationship between the OA and GON and found that the 2 structures interacted in 54% of specimens, in either a simple intersection pattern or with helical intertwining. That same year, Janis et al[22] also described a total of 6 potential points of compression of the GON, which included compression between the semispinalis and obliquus capitis inferior, at both the entrance and exit from the semispinalis, followed by the entrance into the trapezius muscle and exit from the trapezius fascia and, finally, at its intersection point with the OA. Later, Junewicz et al[38] reported on intraoperative variations in GON anatomy and described branching patterns of the GON.

The TON and LON have also been studied anatomically. In 2005, Dash et al[20] defined the location of the TON and LON with respect to external landmarks in a cadaveric dissection study. Subsequently, Lee et al[39] described the topography of the LON including the location of its emergence from the sternocleidomastoid, interactions with branches of the OA, and the presence of fascial band compression in some specimens.

The nasoseptal trigger point is thought to be related to both anatomical and functional components. Mucosal contact points such as septal deviation, spurs, concha bullosa, turbinate hypertrophy, or irritated or inflamed paranasal sinus linings may either directly trigger V2 irritation, which is the primary innervation to the nasal mucosa, or may indirectly trigger through air turbulence created by these entities. Anatomic studies on rhinogenic headaches have demonstrated this.[40] Further radiologic studies have also demonstrated a connection, where 74.3% of "sinus headache" patients actually satisfied International Headache Society for migraine headaches.[41]

Finally, the auriculotemporal nerve (ATN), which is a branch of the mandibular division of the trigeminal nerve (V3), has recently gained attention as a potential trigger site. Of particular interest is the relationship between the ATN and the superficial temporal artery (STA), which was described by Janis et al[42] in 2010. In this cadaveric dissection study, the authors reported an interaction or crossing of the ATN and STA in 34% of specimens. To complement these findings, Chim et al[43] subsequently described 3 potential compression sites of the ATN. Two occurred as a result of preauricular fascial bands and the third corresponded to an interaction with the STA, which was present in 80% of specimens in this study.

DISCUSSION

To date, there have been 17 clinical studies reporting the outcomes of surgical peripheral nerve decompression for the treatment of migraine headaches with level I to level IV evidence supporting its efficacy. A recent critique suggests that there is insufficient clinical evidence to support this treatment strategy with too few trials, a lack of appropriate follow-up, and a lack of evidence regarding safety and adverse events.[44] Questions that challenge the practice of the surgical treatment of migraine headaches are welcomed and are essential as they provide substrate for further discovery and opportunities to raise the standard of care. Unfortunately, no critique, to date, has examined the available evidence in its entirety, perhaps because the evidence supporting the surgical treatment of migraine headaches is evolving quickly and accumulating rapidly. There are currently 3 case series, 8 retrospective cohort studies, 3 prospective cohort studies, and 2 randomized controlled trials that address the efficacy of peripheral nerve decompression for the treatment of migraine headaches. Among these trials, the average success rate of surgery, quantified as either migraine headache elimination or at least 50% reduction in symptoms, has approached nearly 90%. These findings have been reproduced

by multiple surgeons at multiple institutions. For all but one retrospective study, follow-up has exceeded 1 year for patients who underwent surgical decompression, and all studies have demonstrated sustained patient benefit. In addition, 5-year follow-up data have been published for the first randomized controlled trial. These data show both sustained benefit and a lack of long-term major complications.[8] In 10 of the 17 studies, adverse events have been reported in detail. The most common adverse events have included transient numbness or paresthesias at the surgical site, incisional alopecia, controlled intraoperative bleeding, and transient uneven brow movement. No major side effects requiring return to the operating room have been reported. These data may suggest that the adverse effects related to surgical decompression are less frequent and less problematic for patients than the side effects that can be observed with traditional medical therapy.

In 2011, Kung et al[45] reviewed the current medical management for migraine headaches, including medication efficacy and side effects. In this review, the authors highlighted that although some patients do achieve sustained relief of migraines through medical management, a substantial number of patients do not or are not eligible for medication use because of comorbid conditions. It is this subset of patients that often benefits the most from surgical decompression of peripheral trigger points. In an effort to raise the standard of care, future research should be dedicated to earlier identification and stratification of this patient population. To achieve this goal, a collaboration between physicians specializing in both the medical and surgical arms of treatment will be essential. Fortunately, the concept of multispecialty care is already emerging as a priority in the treatment of migraine patients.[46]

In addition to the dramatic improvement of quality of life for this patient population, surgical treatment of migraines is also cost-effective. Although the initial costs of surgery may be higher, Faber et al[47] demonstrated that, ultimately, both direct and indirect costs are reduced for patients after surgery with a median total cost reduction of $3949 per year. This is contributed to by decreased medication costs, fewer primary care visits, and by reducing the number of work days missed with regained productivity time. Because the average surgical cost was $8378 in this study, the expense of medical management for migraine headaches exceeded the up-front cost of surgery shortly after 2 years postoperatively.

Although surgical deactivation of peripheral trigger sites is not the only procedural treatment available for migraine headaches, it is the most rigorously studied with well-demonstrated efficacy and sustainability. Unfortunately, this technique is often broadly categorized with other procedural modalities, and this allows for misrepresentation and underestimation of the clinical and anatomical expertise that has been gained in the past decade. Ducic et al[48] recently provided a systematic review examining the prevalence, efficacy, and complication rates of some of the available procedural treatments for migraine headache including peripheral nerve decompression, radiofrequency therapy, and peripheral nerve stimulation and found that peripheral nerve decompression is overall the most efficacious strategy (with an 86%, 55%, and 68% success rate, respectively) with the fewest complications.

The data supporting surgical deactivation of peripheral nerve triggers for migraine headaches are now well described in the plastic surgery literature. This is reflected by the fact that more surgeons are adopting these techniques into their practice, as reported by Kung et al[49] in 2012. We are encouraged that the overwhelming evidence reviewed here will promote the emergence of new, multidisciplinary teams between plastic surgeons, neurologists, and pain management specialists for the benefit of patients who often have too few options for achieving a pain-free, meaningful quality of life. We hope that this open collaboration and mutual critique will continue to facilitate innovative solutions and new ideas for research. Despite the significant progress made, the pursuit to refine a surgical approach for the treatment of migraine headaches through technique modification, patient stratification, identification of additional trigger points, and an increased anatomical knowledge continues. Currently, we are aware of several additional studies in progress, including a multicenter randomized controlled trial, a study on supraorbital rim anatomy and potential proximal points of compression, and several studies investigating the molecular mechanisms behind extracranial sensory nerve compression in migraine initiation. It is our hope that through continued discovery, innovation, and dissemination of knowledge, an increasing number of patients will benefit from this technique.

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