Validation of the Peripheral Trigger Point Theory of Migraine Headaches: Single-Surgeon Experience Using Botulinum Toxin and Surgical Decompression

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Background: Migraine headache is a widespread neurovascular disorder that is often suboptimally or incompletely treated. This article confirms the efficacy of botulinum toxin treatment with surgical decompression as a deactivator of migraine headache trigger sites through the retrospective analysis of a single surgeon's experience.
Methods: A retrospective chart review was performed on 24 patients presenting with the diagnosis of migraine headache. Botulinum toxin type A injections were used to identify frontal, temporal, and/or occipital trigger points. The nasal trigger point was diagnosed with a decongestant trial, intranasal examination, and computed tomographic scan. Those patients with more than one trigger point underwent multiple surgical procedures, which were performed concomitantly during the same operation. All botulinum toxin injections, surgical procedures, and patient meetings were conducted by the principal investigator (J.E.J.), minimizing intrapatient treatment variability and multiprovider bias.
Results: Patient progress was tracked by consolidating migraine frequency, severity, and duration as a Migraine Headache Index. Nineteen patients (79.2 percent) benefited from surgery. Two patients (8.3 percent) reported migraine elimination and 17 patients (70.8 percent) reported significant improvement of their migraine symptoms. Among those patients who responded to surgery, average improvement from baseline levels was 96.9 percent. Among the entire patient population, average improvement was 78.2 percent from baseline. The mean postsurgical follow-up was 661 days.
Conclusion: This study found botulinum toxin treatment with surgical decompression to be a potent deactivator of migraine headache trigger sites, corroborating the findings of the current literature in the field and underlining the reproducibility of the treatment. (*Plast. Reconstr. Surg.* 128: 123, 2011.)
CLINICAL QUESTION/LEVEL OF EVIDENCE: Therapeutic, IV.

Migraine headache affects 28 million individuals in the United States,[1] 17.1 percent of women and 5.6 percent of men,[2] and is more prevalent than asthma and diabetes combined.[3] Direct medical costs for migraine headaches are approximately $1 billion annually, with an estimated $13 billion annually in lost productivity,[4] causing enormous economic difficulties and personal suffering.

Traditionally, abortive and preventative pharmaceuticals have been the preferred means of migraine management. Despite advancements in pharmaceutical treatment, medications are not always effective, often have severe side effects, and can be expensive.[5,6]

In 2000, Guyuron et al. first reported an association between corrugator supercilii muscle resection and elimination or significant improvement of migraine headache symptoms: 31 of 39 patients experienced elimination or significant improvement after corrugator resection, a 79.5 percent

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success rate.[7] Furthering this study in 2002, Guyuron et al. performed corrugator supercilii muscle resection alone or in conjunction with transection of the zygomaticotemporal nerve on 22 patients.[8] Twenty-one of 22 (95.5 percent) experienced elimination or considerable improvement.

Dirnberger and Becker used Guyuron's techniques in 2003 to corroborate these findings and add support to the "peripheral trigger point theory." They found elimination or significant improvement in 41 of 60 migraine headache patients (68.3 percent).[9]

In 2005, Guyuron et al. reported on the simultaneous treatment of four trigger points: corrugator supercilii muscle resection in the frontal region, zygomaticotemporal nerve avulsion in the temporal region, greater occipital nerve decompression in the occipital region, and septoplasty and inferior and/or middle turbinectomy to treat intranasal abnormality causing compression of the nasal trigeminal branches. The investigators reported elimination or significant improvement in 82 of 89 patients (92.1 percent).[10] The theory and technique were reaffirmed by Poggi et al. in 2008, showing elimination or significant improvement in 16 of 18 patients (88.9 percent) using Guyuron's protocols, even without addressing the septal trigger point.[11,12]

Despite success, investigators could not obtain complete patient response. This encouraged reinvestigations into the anatomy of known trigger points (e.g., supraorbital nerve, zygomaticotemporal nerve, and greater occipital nerve) and new studies into additional trigger points, such as the auriculotemporal nerve.[13–20]

The strongest support for the surgical treatment of migraine headaches was a sham-surgery controlled study that demonstrated statistically significant improvements in the surgery versus sham surgery groups. In the surgery group, 41 of 49 patients (83.7 percent) reported significant improvement, 28 (57.1 percent) with migraine headache elimination. In the sham-surgery group, significant improvement was seen in 15 of 26 patients (57.7 percent) and elimination was seen in one of 26 patients (3.8 percent).[21]

To demonstrate the reproducibility of botulinum toxin and surgery to treat migraine headaches, our study provides an analysis of a patient population that had undergone nerve avulsion and/or muscle resection at multiple trigger points, including the septum. The principal investigator (J.E.J.) conducted the entire treatment protocol, providing unparalleled consistency and minimized biases.

PATIENTS AND METHODS

From March of 2005 to March of 2009, 96 patients presented to the principal investigator's practice for migraine headache treatment. Of these, 24 (25.0 percent) underwent operative treatment, 11 (11.5 percent) are still undergoing the injection protocol, 10 (10.4 percent) opted to forgo treatment, seven (7.3 percent) saw no benefit from botulinum toxin, 19 (19.8 percent) were lost to follow-up, and 25 (26.0 percent) elected to stop the injection protocol despite reporting an improvement (likely for financial reasons, as treatment is considered "out-of-pocket" in the principal investigator's state).

The 24 patients who underwent operative treatment were included in the study, with a total of 63 surgical trigger point decompressions. Patients with multiple trigger points underwent multiple concomitant procedures. After institutional review board approval, data were obtained from chart review. All patients presented with diagnoses of migraine headache as outlined by the International Headache Society and were in good health; women were not pregnant or nursing. Before injection, patients were informed that the use of botulinum toxin type A to address migraine headaches was not U.S. Food and Drug Administration approved.

Patients completed a survey before injections were administered. An intranasal examination was performed on patients with morning migraines, seasonal allergies, nasal obstruction, or incomplete migraine headache elimination after injections to other trigger points.

After consent, botulinum toxin was injected into the suspected primary "trigger point." Patients with migraine headache pain originating primarily from the occipital region received 50 units (25 units per side), 3 cm inferior to the occipital protuberance and 1.5 cm lateral to the midline bilaterally, to the portion of the semispinalis muscle surrounding the greater occipital nerve. Those with migraine headaches originating from the frontal and/or temporal region received 62.5 units: the bilateral corrugators surrounding the supraorbital nerve and supratrochlear nerves were injected with 12.5 units each and the bilateral temporalis muscles surrounding the zygomaticotemporal nerve received 18.75 units each.

Patients were asked to keep track of their headache patterns until follow-up 30 days later. Progress was tracked with postinjection surveys. If the patient reported a 50 percent or greater improvement in migraine headache frequency, duration,

or severity, the site was confirmed to be a trigger point. If the patient presented with residual migraine headaches, other potential sites were injected in a systematic manner as outlined by Guyuron et al.[10] Patients with septal deformities and/or turbinate hypertrophy were treated with a decongestant in addition to a computed tomographic scan of the face and sinuses to look for concha bullosa, septal deviation or spurring, and contact points. The mean time to completion of the diagnosis protocol was 129 days. An average of 172.1 units of botulinum toxin over 4.8 injections was administered per patient.

Once the diagnosis protocol was complete, surgery was offered to patients who exhibited a positive response. An endoscopic bilateral corrugator supercilii muscle resection was performed on patients with frontal migraines. To protect the nerve and correct minor contour deformities caused by the procedure, the skeletonized supraorbital nerve and supratrochlear nerve were shielded with autologous fat grafts after corrugator supercilii muscle resection, as described by Guyuron et al.[8] The transpalpebral approach[8] was not used for any patient. The temporal trigger point was addressed with an endoscopic bilateral zygomaticotemporal nerve avulsion. Bilateral corrugator supercilii muscle resection and zygomaticotemporal nerve avulsion were performed concurrently in all instances except one, because of the association and proximity of the frontal and temporal trigger points and the common exposure of the two sites during the procedure. Decompression of the single arterial and five potential musculofascial points of greater occipital nerve entrapment were performed to address the greater occipital nerve trigger point.[16,17] Septoplasties and/or inferior and/or middle turbinectomies were performed on those patients who displayed intranasal abnormality (Table 1). At each follow-up, patients completed a postsurgery survey.

Table 1. Surgical Procedures Performed on the Patient Population*

Procedure Performed (n = 24)	No. of Patients (%)
SON/STN alone	1 (4.2)
SON/STN/ZTN alone	4 (16.7)
GON alone	4 (16.7)
SON/STN/ZTN + GON	7 (29.2)
SON/STN/ZTN + septoplasty	3 (12.5)
SON/STN/ZTN + GON + septoplasty	5 (20.8)

SON, supraorbital nerve; STN, supratrochlear nerve; ZRN, zygomaticotemporal nerve; GON, greater occipital nerve.
*Those patients with more than one trigger point underwent multiple procedures, which were performed concomitantly at the same operation.

Statistical analysis was performing using Analyze-it v2.21 (Analyse-it Software, Ltd., Leeds, United Kingdom) software. Migraine patterns were tracked using the Migraine Headache Index, defined as the product of migraine headache severity (scale of 1 to 10), migraine headache duration (fraction of 24 hours), and migraine headache frequency (days per month). With a small sample size and nonparametric data set, a two-tailed Wilcoxon signed ranks test was used to compare quality of life, migraine headache frequency, severity, duration, and Migraine Headache Index from baseline, post–botulinum toxin, and post–surgery levels. To assess overall improvement by trigger site, a two-tailed binomial test with a hypothesized proportion of 0.5 was used. A two-tailed p value was calculated using the McNemar test to see whether women with septal trigger points were more likely to have menstruation-related migraines. Values of $p < 0.05$ were considered significant.

RESULTS

Overview

The patient population consisted of 23 women and one man, with an average age of 44.4 years (range, 23.2 to 66.5 years). Mean follow-up was 661 days (range, 157 to 1615 days).

Patients were asked to rank on a scale ranging from 1 (mild) to 10 (severe) the effect that migraine headaches had on their quality of life and to compare the efficacy of botulinum toxin injections versus surgery. The average effect of migraine headaches on quality of life was 9.18 before treatment, 3.80 after botulinum toxin injection ($p < 0.0001$), and 3.11 after surgery ($p < 0.0001$) (Table 2). Three patients (14.3 percent) felt that botulinum toxin injection was the most effective treatment, 10 (47.6 percent) felt surgery was most effective, and eight (38.1 percent) said both were equal.

Post–Botulinum Toxin Results

All patients received benefit from botulinum toxin injection: six (25.0 percent) experienced migraine headache elimination, 17 (70.8 percent) reported significant (>50 percent) improvement, and one (4.2 percent) had only a modest improvement of 12.9 percent over baseline Migraine Headache Index but reported subjective benefits as the "longest migraine headache-free streak in 25 years." Of the 17 patients with significant improvement, the Migraine Headache Index improved an average of 87.5 percent from baseline levels (Table 2).

Table 2. Mean Pretreatment, Post–Botulinum Toxin Injection, and Postsurgery Quality of Life and Migraine Headache Frequency, Severity, and Duration for All Patients ($n = 24$)

					p†	
	Pretreatment	Post- Botulinum Toxin Injection	p (Comparison with Baseline)†	Postsurgery	Comparison with Baseline	Comparison with Post–Botulinum Toxin
MH frequency (days/mo)*	16.52 ± 11.11	4.24 ± 5.82	<0.0001	3.78 ± 6.45	<0.0001	0.731
MH severity (scale of 1 to 10)*	7.25 ± 2.08	3.66 ± 2.95	<0.0001	3.31 ± 2.50	<0.0001	0.427
MH duration as a fraction of 24 hr*	1.04 ± 0.89	0.50 ± 0.71	0.0391	0.47 ± 0.48	0.0002	0.754
MHI*	106.59 ± 89.72	13.44 ± 21.33	<0.0001	10.27 ± 28.17	<0.0001	0.520
Quality of life*‡	9.18 ± 1.01	3.80 ± 2.37	<0.0001	3.11 ± 2.59	<0.0001	0.268

MH, migraine headache; MHI, Migraine Headache Index.
*Data are expressed as mean ± SD.
†The p values were obtained using a two-tailed Wilcoxon signed ranks test.
‡For the quality of life row only, $n = 22$. Two patients could not be reached for these data.

Botulinum Toxin Injection Complications and Benefits

Of the 135 injections administered, 23 (17.0 percent) resulted in migraine headache, 14 (10.4 percent) resulted in neck soreness, one resulted in neck weakness (0.7 percent), three (2.2 percent) resulted in neck soreness and weakness, two (1.5 percent) resulted in diplopia, one resulted in ptosis (0.7 percent), and one (0.7 percent) resulted in mild mastication weakness. Five patients' (20.8 percent) migraine headache medicine took effect more quickly and/or could be administered in smaller doses. Five (20.8 percent) completely discontinued neurologist-prescribed migraine headache medication (despite requests to remain consistent throughout the treatment). Other benefits included decreased nausea and non–migraine headache pain.

Postsurgery Results

Nineteen of 24 patients (79.2 percent) benefited from surgery. Two (8.3 percent) experienced migraine headache elimination and 17 (70.8 percent) reported significant (>50 percent) improvement. Of the 17 patients with significant improvement, the Migraine Headache Index improved an average of 96.6 percent from baseline (Table 2).

Five patients (20.8 percent) failed surgery, defined as subjectively reporting failure of surgery or a Migraine Headache Index approaching baseline levels. On average, 148 days elapsed before patients experienced resurgence of nearly baseline migraine headache pain. One patient had a postoperative course complicated by the development of a hematoma in the occipital region, related to resumption of her preoperative warfarin for a history of deep venous thrombosis. One patient was diagnosed with fibromyalgia, a possible confounding factor.

Surgery Complications and Benefits

Eleven of 24 patients (45.8 percent) experienced immediate postoperative migraine headache. Surgery-site paresthesias lasted 163 days on average, with one patient (4.2 percent) experiencing paresthesias for over 1 year. Eleven patients (45.8 percent) experienced mild incisional alopecia, which resolved in five patients by the last follow-up. There were 16 instances (66.7 percent) of periorbital ecchymosis, most very mild. One patient (4.2 percent) continues to feel discomfort when touching her forehead.

After surgery, seven patients' (29.2 percent) migraine headache medication took effect more quickly and/or required smaller doses. Three patients (12.5 percent) were able to stop taking migraine headache medications and three (12.5 percent) reported decreased nausea. Eleven patients (45.8 percent) "challenged" themselves with known migraine triggers without instigating a migraine headache.

Postsurgery Results by Trigger Site

Five patients (20.8 percent) were diagnosed with one trigger point, four patients (16.7 percent) were diagnosed with two trigger points, 10 patients (41.7 percent) were diagnosed with three trigger points, and five patients (20.8 percent) were diagnosed with four trigger points. Greatest overall improvement was seen in the septal region, followed by the occipital region. Similar overall improvement was seen in the frontal and temporal sites (Table 3).

Postsurgery Migraine Headache Recurrence

Of the 22 patients without migraine headache elimination postoperatively, 17 (77.3 percent) had

Table 3. Postsurgery Results Stratified by Migraine Headache Trigger Points ($n = 24$)*

Trigger Site	No. of Patients (%)	Overall Improvement (%)†	Elimination of MH (%)	Improvement of MH (%)
Frontal	20 (83.3)	17 (85.0)	7 (35.0)	10 (50.0)
Comparison		17/20 vs. 3/20		
p		0.0026		
Temporal	19 (79.2)	17 (89.5)	10 (52.6)	7 (36.8)
Comparison		17/19 vs. 2/19		
p		0.0007		
Occipital	16 (66.7)	15 (93.8)	9 (56.3)	6 (37.5)
Comparison		15/16 vs. 1/16		
p		0.0005		
Nasal	8 (33.3)	8 (100)	5 (62.5)	3 (37.5)
Comparison		8/8 vs. 0/8		
p		0.0078		

MH, migraine headache.
*Overall improvement was defined as the postsurgical elimination or significant improvement of migraine pain in a trigger point that was diagnosed through the botulinum toxin injection protocol.
†The *p* values were obtained from a two-tailed binomial test with a hypothesized proportion of 0.5.

symptoms in the frontal (supraorbital nerve/supratrochlear nerve) area (including the upper cephalic and/or periocular region), the most common site of migraine pain recurrence. Thirteen (59.1 percent) reported residual postoperative migraine in the temporal region, eight (36.4 percent) reported residual postoperative migraine in the occipital region, and four (18.2 percent) reported residual postoperative migraine in the retroorbital region. Auriculotemporal pain, previously masked by symptoms in other areas, was uncovered in two patients (9.1 percent) (Table 4).

DISCUSSION

Despite being a widespread, debilitating, and costly neurovascular disorder, migraine headache continues to be underdiagnosed and suboptimally treated,[22] with 71 percent of migraine sufferers in the United States not completely satisfied with their usual acute treatment, citing such reasons as delayed and/or incomplete pain relief.[23] The pathophysiology of migraine is not completely understood. Of the various theories, central and peripheral activation[24] and sensitization[25] of the trigeminal nerve system is most pertinent to this study. Migraine headaches may be caused by the vasodilation of the meningeal blood vessels, which are innervated extensively by the trigeminal nerve system.[10,26–28] The terminals of these nerves contain the neuropeptides substance P, neurokinin A, and calcitonin gene-related peptide, which cause vasodilation of the meningeal vessels and the activation and degranulation of mast cells (neurogenic inflammation).[10,26,29]

Table 4. Areas of Migraine Pain Recurrence in the 22 Patients Who Did Not Experience Postoperative Migraine Headache Elimination ($n = 22$)*

Pain Site	No. of Patients (%)
SON/STN (frontal)	17 (77.3)
ZTN (temporal)	13 (59.1)
GON (occipital)	8 (36.4)
AT	2 (9.1)
Retroorbital	4 (18.2)

SON, supraorbital nerve; STN, supratrochlear nerve; ZTN, zygomaticotemporal nerve; GON, greater occipital nerve; AT, auriculotemporal.
*Patients presenting with pain in the upper cephalic (forehead and top of head) and/or periocular region were grouped in the supraorbital nerve/supratrochlear nerve category.

The frontal, temporal, and occipital trigger points are all characterized by the close association of branches of the trigeminal nerve with muscular, vascular, and/or fascial structures, often presenting multiple points of potential entrapment.[13–19] Intranasal contact points may trigger migraine headaches through the release of pain mediators found in the trigeminovascular system.[30] The physiologic mechanisms underlying chemodenervation and surgical migraine treatment are identical: botulinum toxin "chemically" decompresses nerves that are associated with surrounding musculature.[31,32]

Partial surgical release of existing trigger points and/or the existence of additional, untreated trigger points may cause incomplete migraine headache elimination. The frontal trigger point was the most common site of postoperative migraine pain recurrence. This may be because the corrugator supercilii muscle is more expansive than previously described[13] and was incompletely resected, leading to only partial decompression of the supraorbital nerve/supratrochlear nerve.

Roughly half of the patients experienced residual postoperative temporal migraine pain. A study of the anatomical variations of the zygoma-

ticotemporal nerve found intramuscular innervation in 50 percent of specimens and extramuscular coursing in the rest.[19] Perhaps nerve entry *and* exit from the muscle were not addressed, resulting in residual symptoms.

Eight patients reported postoperative migraine pain in the occipital area. Research into the course of the greater occipital nerve has uncovered six points of potential entrapment: five musculofascial[16] and one arterial.[17] Closely related to the greater occipital nerve are the lesser and third occipital nerves, which may be suspect in lateral and medial occipital pain, respectively.[33] Patients presenting with pain in the auriculotemporal region may have benefited from ligation of the superficial temporal artery proximal and distal to the point at which it transects the auriculotemporal nerve,[20] which was not performed in any patients in this study.

Our study found botulinum toxin treatment and surgical decompression to be potent deactivators of migraine headache trigger points. In the 19 patients who reported significant migraine headache improvement or elimination postsurgically, the Migraine Headache Index decrease from baseline levels was 96.9 percent on average. Even with inclusion of patients who did not respond to surgery, the Migraine Headache Index decreased 78.2 percent from baseline on average. Nonresponders to surgery saw a Migraine Headache Index decrease of 6.9 percent from baseline on average, which was not statistically significant.

Of patients reporting significant improvement, a greater average improvement over baseline levels was derived from surgery than from botulinum toxin. Furthermore, average Migraine Headache Index was lower after surgery than after botulinum toxin injection, even with the inclusion of surgery nonresponders. The trend suggests that surgery is the more effective and long-lasting means of addressing migraines, despite the fact that there was no statistically significant difference between post–botulinum toxin Migraine Headache Index and postsurgery Migraine Headache Index figures (Table 2). Three of five surgery failures were characterized by residual migraine headache pain in areas different from those the patient experienced preoperatively, whereas two patients experienced reoccurrence of pain in the regions of surgical intervention.

In addition to the five nonresponders to surgery, the small sample size, retrospective review, and low rate of postsurgical migraine headache elimination were additional limitations. Our migraine headache elimination rate of 8.3 percent was lower than that of Poggi et al. (16.7 percent) and substantially lower than that of Guyuron et al. (57.1 percent). This discrepancy may partially be explained by variations in surgical technique: both Poggi et al. and Guyuron et al. addressed the frontal trigger point by removal of the glabellar muscles (i.e., corrugator supercilii muscle, depressor supercilii, and procerus), whereas the principal investigator of the present study only resected the corrugator supercilii muscle. More complete supraorbital nerve/supratrochlear nerve decompression may be achieved by complete avulsion of the corrugator supercilii muscle origin medially, and resection to where the corrugator supercilii muscle passes into the frontalis/orbicularis muscles laterally, as recommended by Knize.[34]

Our data also yielded other associations: (1) postoperative residual aura (without migraine pain); (2) menstrual cycle–related septal triggers; (3) migraines beginning with a herpes zoster infection; (4) unusually high occurrence of head and neck trauma in the patient population; and (5) possible occurrence of referred pain. Four patients who only underwent greater occipital nerve release all reported postoperative migraine pain in the frontal and/or temporal region(s).

Five patients postoperatively reported continued aura without migraine pain. These patients appeared to have acephalgic migraine (aura without headache).[35] For these five, the decompression of peripheral trigeminal nerves did not seem to knock out the "first domino" in the cascade leading to a migraine headache. Some central mechanisms of migraine pathogenesis appear to be postsurgical, suggesting that this treatment falls "downstream" of these mechanisms. This is consistent with most current migraine theories, which point to cortical spreading depression, a diffusing decrease in cerebral electrical activity and blood flow,[29] as the primary trigger of trigeminovascular system activation.[26] Cortical spreading depression has been shown to be the phenomenon underlying aura.[36,37] These five patients may experience cortical spreading depression and the associated aura and trigeminovascular activation but escape migraine pain because of the release of the points of mechanical stimuli.

Six of seven female patients (85.7 percent) with septal triggers also had migraine headaches linked to their menstrual cycles. However, of the 13 female patients with menstruation-linked migraines, only six displayed septal triggers (46.2 percent). Though marginally statistically insignificant ($p = 0.0703$), there seems to be a trend between the presence of septal triggers and migraines linked to the menstrual

cycle. Studies have shown that hormonal fluctuations of the menstrual cycle lead to changes in nasal mucosa,[38,39] including increased vascularity and congestion.[38,40] These changes lead to increased friction between the septum and the enlarged turbinates, triggering migraine headache by means of irritation and/or compression of the sphenopalatine ganglia. Studies have documented the treatment of rhinologic migraine headache by addressing the compression of an enlarged turbinate against an often deviated septum.[41–45] Such contact results in the irritation of the maxillary and ophthalmic braches of the trigeminal nerve[46] and, ultimately, migraine headache, lending further support to peripheral migraine activation.

One patient reported that her migraines began when she contracted herpes zoster, and they were subsequently linked to episodes of that infection. The shingles presented only on the right side—the same side as her headaches. Cases of cluster-like headaches following herpes zoster infection of the ophthalmic division of the trigeminal nerve have been reported, with ipsilateral headache symptoms and infection.[47] This link between headache and herpes zoster can be expanded to include systemic and extracranial infections in general.[48] The infection and subsequent irritation of peripheral trigeminal nerves seems sufficient to trigger the chain of events leading to migraine headache.

There was an unusually high occurrence of head and neck trauma in our patient population. In a recent study, the prevalence of head and neck injury was found to be 37.2 percent in a group of women experiencing chronic daily headache versus 26.9 percent in a control group.[49] The 37.5 percent (nine of 24) prevalence of head and neck injury in our predominantly female patient population mirrors these findings, corroborating a higher prevalence of prior head and neck trauma in patients with migraine headache. Chronic common and classic migraine caused by head or neck trauma is termed "posttraumatic migraine."[50] A study into the neurochemical processes of mild head injury and migraine headache uncovered numerous similarities in the mechanisms underlying both afflictions.[51] These neurochemical processes may activate the trigeminovascular system through the cascade described previously. One patient with a greater occipital nerve trigger point successfully treated with surgery by the senior author (J.E.J.) experienced postsurgical recurrence of migraine headache after neck trauma resulting from an automobile accident. As opposed to classifying them as distinct entities, researchers are beginning to place migraine, chronic daily headache, posttraumatic migraine, and other such disorders along a multifactorial and intertwined continuum of headache pain.[52] Such a holistic perspective enables us to more readily appreciate the merit in sharing treatments and theories among the various forms of headache pain.

The four patients who underwent greater occipital nerve decompression experienced postsurgical migraine pain in the frontal and/or temporal region(s), despite having no presurgical diagnosis of triggers in those areas. One of the patients reported a Migraine Headache Index score of 0 after receiving an injection to the greater occipital nerve region despite presenting with pretreatment pain in the supraorbital nerve/supratrochlear nerve/zygomaticotemporal nerve areas and the occipital region. These trends may be attributed to (1) the failure to properly identify frontal triggers during the botulinum toxin injection protocol; or (2) pain referral, whereby pain originating from a given receptive field is perceived to be coming from a distant receptive region.[53] We suggest that physicians remain cognizant of referred pain when evaluating for migraine headache botulinum toxin injections and surgical intervention.

During botulinum toxin treatment, patients often reported worsening migraine headaches 2.5 to 3 months after injection. This corresponds to studies that have shown returning nerve functionality 63 days after injection of botulinum toxin and complete return of dynamic muscle contraction 91 days after injection.[31] These Migraine Headache Index parameters were used to calculate "rebound Migraine Headache Index" (Migraine Headache Index as the patient recovers from chemodenervation) for 18 patients. Fifteen of 18 patients (83.3 percent) had a rebound Migraine Headache Index somewhere in between baseline Migraine Headache Index and post–botulinum toxin injection Migraine Headache Index. As expected, patients displayed gradual botulinum toxin metabolization, as opposed to a sharp loss of chemodenervation, because of the exceptionally stable activity of botulinum toxin type A.[31] Although rebound Migraine Headache Index cannot be compared between patients, it does provide a means of establishing a control internal to each patient.

CONCLUSIONS

In this single-surgeon study, the principal investigator was able to significantly improve or elim-

inate the migraine symptoms in 19 of 24 patients. With the addition of these findings, three independent operators, Dirnberger et al., Poggi et al., and the principal investigator, have been able to successfully treat migraine headaches using the surgical and botulinum toxin injection protocols first recommended by Guyuron et al. This underscores the reproducibility of the treatment and lends further credibility to the peripheral trigger point theory of migraine headaches. Investigations into resting muscle tone, histologic and biochemical differences in trigeminal nerve branches relative to nonmigraineurs, anatomical variants, and new trigger points may help address symptoms in those patients with recalcitrant migraines.

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