

A Placebo-Controlled Surgical Trial of the Treatment of Migraine Headaches

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Background: Many of the nearly 30 million Americans suffering with migraine headaches are not helped by standard therapies, a proportion of which can harbor undesirable side effects. The present study demonstrates the efficacy of independent surgical deactivation of three common migraine headache trigger sites through a double-blind, sham surgery, controlled clinical trial.

Methods: Seventy-five patients with moderate to severe migraine headache who met International Classification of Headache Disorders II criteria were studied. Trigger sites were identified (frontal, temporal, and occipital), and patients were randomly assigned to receive either actual or sham surgery in their predominant trigger site. Patients completed the Migraine Disability Assessment, Migraine-Specific Quality of Life, and Medical Outcomes Study 36-Item Short Form Health Survey health questionnaires before treatment and at 1-year follow-up.

Results: Of the total group of 75 patients, 15 of 26 in the sham surgery group (57.7 percent) and 41 of 49 in the actual surgery group (83.7 percent) experienced at least 50 percent reduction in migraine headache ($p < 0.05$). Furthermore, 28 of 49 patients in the actual surgery group (57.1 percent) reported complete elimination of migraine headache, compared with only one of 26 patients in the sham surgery group (3.8 percent) ($p < 0.001$). Compared with the control group, the actual surgery group demonstrated statistically significant improvements in all validated migraine headache measurements at 1 year. These improvements were not dependent on the trigger site. The most common surgical complication was slight hollowing of the temple in the group with temporal migraine headache.

Conclusion: This study confirms that surgical deactivation of peripheral migraine headache trigger sites is an effective alternative treatment for patients who suffer from frequent moderate to severe migraine headaches that are difficult to manage with standard protocols. (*Plast. Reconstr. Surg.* 124: 461, 2009.)

Nearly 30 million Americans suffer from migraine headaches.¹⁻³ Many of the available prophylactic medications harbor side effects such as sedation, paresthesias, weight gain, cognitive impairment, and sexual dysfunction.⁴⁻⁶ The cost of migraine treatment and loss of time from work associated with migraine headaches impose a major economic burden on the patient and society, collectively exceeding \$13 billion.⁷ Multiple studies by our research team have demonstrated a response rate (i.e., at least 50 percent

reduction in intensity, frequency, and duration of migraine headaches) of over 90 percent when migraine trigger sites are surgically deactivated.⁸⁻¹⁰ Other researchers have demonstrated almost similar results.^{11,12} The purpose of this placebo-controlled (sham surgery) prospective study was to investigate the efficacy of this surgical treatment in patients with a single or predominant trigger site. The trigger site is where the migraine headache

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begins and settles and corresponds to the anatomical zone of potential irritation of the trigeminal nerve, based on our studies.

PATIENTS AND METHODS

Research Design and Methods

Institutional review board approval was obtained from two institutions. Patients with frequent moderate to severe migraine headaches triggered from a single or predominant site were interviewed by a physician member of the research team. After completing a daily migraine headache diary for a 1-month period and a comprehensive migraine headache information form, patients were examined by one of two board-certified headache neurologists (D.R. or J.S.K.). The diagnosis of migraine headache was confirmed using the International Classification of Headache Disorders II criteria.¹³ Patients who met study criteria were offered participation. All patients signed a written informed consent form on study enrollment. Patients were asked to complete previously validated questionnaires, including the Medical Outcomes Study 36-Item Short Form Health Survey, Migraine-Specific Quality of Life, and Migraine Disability Assessment questionnaires before treatment.^{14–17} For both the Medical Outcomes Study 36-Item Short Form Health Survey and Migraine-Specific Quality of Life questionnaires, a higher score indicates better general health and quality of life; for the Migraine Disability Assessment, a lower score indicates less headache-related disability. Guided by the most prevalent site from which the migraine pain started and settled consistently and a positive response (i.e., at least 50 percent decrease in the migraine headache intensity, duration, frequency, and the migraine index, all four being considered the endpoint) to the injection of 25 units of botulinum toxin type A (Botox; Allergan, Inc., Irvine, Calif.) at the site, the patients were assigned into one of three groups: frontal (F), temporal (T), and occipital (O). In each group, approximately one-third of the patients were assigned randomly to undergo a sham surgical procedure (control group) and the other two-thirds were randomly assigned to undergo the actual operation. Operations were performed only when the migraine headaches recurred following the disappearance of the Botox effect. Random assignment was accomplished by drawing an instruction card from a serially numbered, opaque, sealed envelope prepared by the biostatistician of the research team. The medical and surgical team played no role in the assignment of patients to the treatment or control groups. The patients remained blinded as to whether their sur-

gery was actual or sham. The data were collected and tabulated, and statistical analyses were carried out independent of the surgical team, who remained blinded to the outcome until data collection was completed. A total of 317 patients were initially screened for inclusion in the study, and 130 underwent injection of Botox to determine study eligibility. Based on the response to Botox, 76 patients were deemed eligible and were included in the study.

Surgical Treatment

Under monitored anesthesia care, patients in treatment group F underwent removal of the glabellar muscles (corrugator supercilii, depressor supercilii, and procerus) encasing the supraorbital and supratrochlear nerves through an upper eyelid incision. The supraorbital and supratrochlear nerves were preserved. A small amount of fat from the medial compartment of the upper eyelid was used to fill the defect of the excised muscles and to shield the nerves. The patients with frontal headaches assigned to sham surgery group F underwent exposure of the muscles and nerves through a similar incision, but the integrity of these structures was maintained.

Patients in treatment group T underwent endoscopic removal of a segment of the zygomaticotemporal branch of the trigeminal nerve. Two 1.5-cm incisions were made at approximately 7 and 10 cm from the midline of the scalp in the region of the right and left hair-bearing temples. Approximately 2.5 cm of this nerve was removed. In sham surgery group T, the nerve was exposed in a similar fashion but was left intact. This nerve is commonly transected during forehead rejuvenation and other craniofacial surgery.

Surgery in treatment group O was performed under general anesthesia, with the patient in prone position, using a 4-cm incision in the midline occipital area. A segment of the semispinalis capitis muscle medial to the greater occipital nerve, approximately 1 cm wide and 2.5 cm long, was removed. A subcutaneous flap was then interposed between the nerve and the muscle to isolate it from the surrounding muscles and avoid impingement of the nerve. In sham surgery group O, the surgery was limited to the exposure of the nerve and the muscle was left intact.

All procedures were performed in an ambulatory surgery center with an average surgery time of less than 1 hour. Patients were permitted to resume ordinary activities in 1 week and heavy exercise in 3 weeks.

Data Collection

All patients maintained a daily headache diary and completed migraine headache questionnaires assessing the frequency (number of headaches per month), intensity (rated on a scale of 1 to 10), and duration (days) of their headaches on a monthly basis. Patients were seen after initial recovery, at 1 month, and every 3 months thereafter for 1 year. Postoperative complications were recorded. At the end of 1 year, patients were again asked to complete the Medical Outcomes Study 36-Item Short Form Health Survey, Migraine-Specific Quality of Life, and Migraine Disability Assessment questionnaires.

Statistical Analysis

Statistix Version 8 (Analytical Software, Inc., Tallahassee, Fla.) and StatView Version 5 (SAS Institute, Inc., Cary, N.C.) were used for statistical analysis. Descriptive statistics were computed for all variables. Reduction of at least 50 percent in migraine headache frequency, intensity, or duration compared with baseline values was used as the criterion for significant improvement. A migraine headache index was calculated by multiplying the frequency, intensity, and duration of migraine headaches, and this was compared with the baseline migraine headache index. A repeated measures analysis of variance was used to compare the mean frequency, intensity, and duration of migraine headaches over time (0, 3, 6, 9, and 12 months) and included parametric and nonparametric techniques. When comparing the data at 12 months to baseline measures, paired analyses were used. A two-way analysis of variance was used to compare the type of surgery and trigger site for each outcome measure at baseline and at 12-month follow-up. Multivariable logistic regression analyses were used to assess factors that influenced significant overall improvements in migraine headache status. Fisher's exact test was used to examine the association between categorical variables such as surgery type and the patient's improvement status. A value of $p < 0.05$ was considered significant.

RESULTS

Baseline Information

Only one of 76 patients failed to complete the 1-year follow-up. The patients' baseline information is listed in Table 1. The mean age was 44.9 years, ranging from 26 to 76 years. The mean \pm SD age was 45.1 ± 9.5 years for patients undergoing actual surgery and 44.6 ± 8.3 years for patients in the sham surgery group ($p = 0.83$). In the actual surgery group, 29 of 49 patients (59 percent) ex-

perienced migraines with no aura, 11 of 49 (22 percent) experienced migraines with aura, and nine of 49 (18 percent) experienced migraines both with and without aura. In the sham surgery group, 17 of 26 patients (65 percent) experienced migraines with no aura, seven of 26 (27 percent) experienced migraines with aura, and two of 26 (8 percent) experienced migraines both with and without aura.

Of 49 patients who underwent actual surgery, 19 underwent the procedure at the frontal trigger site, 19 at the temporal site, and 11 at the occipital site. Of the 26 patients who underwent sham surgery, 10 had the procedure at the frontal trigger site, nine at the temporal site, and seven at the occipital site. All preoperative baseline scores were comparable between the actual and sham surgery groups.

Patients in the sham surgery group were offered the opportunity to undergo actual surgery at the completion of the 1-year follow-up. Of the 26 patients in the sham surgery group, 22 have undergone the actual surgery for their migraine headaches after serving as a control for 1 year.

Follow-Up Information

All patients were followed up at 3, 6, 9, and 12 months after surgery. Four types of outcome measures were considered: (1) complete elimination of migraine headaches; (2) significant improvement in patients' migraine headache frequency, intensity, duration, or migraine index; (3) the difference between the baseline and 12-month follow-up measures; and (4) the difference between the average measures at 1 year and baseline.

Complete Elimination

Twenty-eight of 49 patients (57.1 percent) in the actual surgery group reported complete elimination of migraine headaches at 12 months, compared with only one of 26 patients (3.8 percent) in the sham surgery group ($p < 0.0001$, Fisher's exact test). Eleven of 26 patients (42.3 percent) in the sham surgery group reported no change in migraine headaches at 12 months, which was significantly higher than the eight of 49 patients (16.3 percent) in the actual surgery group ($p = 0.02$, Fisher's exact test).

Significant Improvement

Forty-one of 49 patients (83.7 percent) in the actual surgery group reported significant improvement at 12 months compared with 15 of 26 patients (57.7 percent) in the sham surgery group ($p = 0.014$). Patients who experienced migraine headaches both with and without aura had a greater

Table 1. Baseline Data by Location and Type of Surgery

Variable	Actual Surgery*	Sham Surgery*	<i>p</i>
No. of patients	49	26	
No. of patients/trigger site			
Frontal	19	10	
Temporal	19	9	
Occipital	11	7	
Age, years	45.1 ± 9.5	44.6 ± 8.3	<i>p_G</i> = 0.79
Frontal	43.8 ± 9.9	42.2 ± 4.6	<i>p_L</i> = 0.37
Temporal	44.6 ± 7.8	46.2 ± 11.7	
Occipital	47.9 ± 11.4	45.9 ± 7.7	
Frequency, MH/mo	9.9 ± 6.0	9.5 ± 4.4	<i>p_G</i> = 0.82
Frontal	9.8 ± 7.7	7.6 ± 3.2	<i>p_L</i> = 0.59
Temporal	10.2 ± 4.7	11.6 ± 4.6	
Occipital	9.5 ± 5.4	9.6 ± 5.0	
Intensity (visual analogue scale, 1–10)	6.2 ± 1.7	5.5 ± 1.4	<i>p_G</i> = 0.06
Frontal	5.9 ± 1.6	6.1 ± 1.5	<i>p_L</i> = 0.76
Temporal	6.3 ± 1.6	4.8 ± 1.1	
Occipital	6.5 ± 2.0	5.5 ± 1.4	
Duration, days	0.54 ± 0.55	1.74 ± 5.6	<i>p_G</i> = 0.16
Frontal	0.56 ± 0.57	1.1 ± 2.4	<i>p_L</i> = 0.30
Temporal	0.56 ± 0.57	0.43 ± 0.4	
Occipital	0.62 ± 0.51	4.3 ± 10.5	
Migraine headache index	29.3 ± 30.8	27.0 ± 28.3	<i>p_G</i> = 0.71
Frontal	24.3 ± 25.9	27.5 ± 31.9	<i>p_L</i> = 0.43
Temporal	28.4 ± 22.7	23.1 ± 26.3	
Occipital	39.7 ± 47.4	31.3 ± 29.0	
MSQEM	41.1 ± 27.5	39.2 ± 25.9	<i>p_G</i> = 0.15
Frontal	48.8 ± 28.8	37.2 ± 33.2	<i>p_L</i> = 0.17
Temporal	52.9 ± 46.9	43.8 ± 31.9	
Occipital	78.1 ± 67.8	50.7 ± 30.7	
MSQPRE	61.6 ± 21.6	62.1 ± 18.5	<i>p_G</i> = 0.84
Frontal	42.6 ± 19.6	49.7 ± 16.2	<i>p_L</i> = 0.37
Temporal	47.8 ± 17.9	48.7 ± 9.5	
Occipital	37.8 ± 18.1	46.1 ± 17.2	
SFPH	45.0 ± 7.1	44.3 ± 9.3	<i>p_G</i> = 0.77
Frontal	45.4 ± 6.7	46.7 ± 7.6	<i>p_L</i> = 0.16
Temporal	44.6 ± 7.6	47.8 ± 7.3	
Occipital	45.0 ± 7.4	36.3 ± 10.3	

p_G, comparison between actual and sham surgery (*p* value computed from two-sample *t* test and verified by Wilcoxon rank test); *p_L*, comparisons among the trigger sites; MH, migraine headaches; MIDAS, Migraine Disability Assessment Score; MSQEM, Migraine-Specific Quality of Life, emotional; MSQPRE, Migraine-Specific Quality of Life, preventive; MSQRES, Migraine-Specific Quality of Life, restrictive; SFPH, Medical Outcomes Study 36-Item Short Form Health Survey, physical.

*Continuous data are represented as mean ± SD.

improvement in intensity scores at 12 months (mean difference at 12 months versus baseline, 4.7) compared with those who always experienced migraine headaches with aura (mean difference at 12 months versus baseline, 1.95) or migraine headaches without aura (mean difference at 12 months versus baseline, 2.09). This bordered statistical significance (*p* = 0.057). Otherwise, there was no significant difference between the groups on any of the other variables.

Comparison of Baseline to 12-Month Data

Changes in the various migraine headache measures from baseline were calculated at 12 months. Table 2 outlines the overall changes from baseline by trigger site (location) and surgical group and compares the changes between the actual and sham surgery groups. Compared with baseline values, all of the migraine headache mea-

asures were significantly improved at 1 year in the actual surgery group, whereas only some of the migraine headache measures were significantly improved in the sham surgery group. In addition, the extent of improvement in frequency, intensity, Migraine Disability Assessment Score, and Migraine-Specific Quality of Life scores was significantly higher in the actual surgery group compared with the sham surgery group (*p* < 0.05).

The effect of trigger site and surgery type on the differences between 12-month and baseline data were further analyzed using two-way analysis of variance. The improvements were not dependent on the trigger site.

Mean Change at 1 Year versus Baseline

The outcome measures obtained at 3, 6, 9, and 12 months were averaged and compared with the baseline values. The average of the outcome mea-

Table 2. Overall Change from Baseline to 12 Months by Location and Type of Surgery

Variable	Actual Surgery*	Sham Surgery*	<i>p</i>
No.	49	26	
Elimination	28/49 (57.1%)	1/26 (3.8%)	<0.001
Significant improvement	41/49 (83.7%)	15/26 (57.7%)	0.014
Frequency, MH/mo	7.4 ± 5.8 (<0.001)	3.5 ± 5.4 (0.003)	<i>p_G</i> = 0.005
Frontal	6.3 ± 6.7 (<0.001)	1.5 ± 3.3 (0.18)	<i>p_L</i> = 0.17
Temporal	7.8 ± 74.8 (<0.001)	4.1 ± 6.8 (0.11)	
Occipital	8.7 ± 6.1 (<0.001)	5.7 ± 5.6 (0.04)	
Intensity (visual analogue scale, 1–10)	3.0 ± 3.5 (<0.001)	1.3 ± 2.9 (0.03)	<i>p_G</i> = 0.03
Frontal	2.5 ± 3.5 (0.005)	2.1 ± 3.1 (0.51)	<i>p_L</i> = 0.34
Temporal	2.4 ± 3.8 (0.001)	0.46 ± 2.7 (0.17)	
Occipital	4.2 ± 3.4 (<0.001)	1.3 ± 3.2 (0.45)	
Duration, days	0.30 ± 0.46 (<0.001)	0.87 ± 4.5 (0.34)	<i>p_G</i> = 0.43
Frontal	0.24 ± 0.36 (0.01)	-0.18 ± 0.94 (0.57)	<i>p_L</i> = 0.13
Temporal	0.21 ± 0.47 (0.07)	0.10 ± 0.33 (0.40)	
Occipital	0.54 ± 0.55 (0.009)	3.37 ± 7.7 (0.34)	
Migraine headache index	21.6 ± 29.6 (<0.001)	9.7 ± 23.9 (0.05)	<i>p_G</i> = 0.07
Frontal	15.4 ± 19.1 (0.003)	12.2 ± 15.4 (0.03)	<i>p_L</i> = 0.29
Temporal	18.9 ± 21.8 (0.001)	7.8 ± 36.5 (0.54)	
Occipital	37.1 ± 48.4 (0.03)	8.5 ± 15.1 (0.18)	
MIDAS	1.5 ± 1.5 (<0.001)	0.77 ± 1.3 (0.007)	<i>p_G</i> = 0.05
Frontal	1.3 ± 1.5 (0.001)	0.2 ± 1.0 (0.56)	<i>p_L</i> = 0.32
Temporal	1.6 ± 1.6 (<0.001)	1.3 ± 1.2 (0.01)	
Occipital	1.5 ± 1.5 (0.01)	0.86 ± 1.7 (0.22)	
MSQEM	36.0 ± 45.8 (<0.001)	10.8 ± 39.0 (0.17)	<i>p_G</i> = 0.02
Frontal	24.0 ± 41.9 (0.02)	0.4 ± 29.6 (0.97)	<i>p_L</i> = 0.12
Temporal	36.5 ± 44.6 (0.002)	16.6 ± 52.1 (0.37)	
Occipital	56.0 ± 51.0 (0.005)	18.1 ± 33.2 (0.20)	
MSQPRE	18.7 ± 22.0 (<0.001)	-13.1 ± 22.1 (0.006)	<i>p_G</i> = 0.29
Frontal	-18.8 ± 19.7 (<0.001)	-16.0 ± 30.7 (0.13)	<i>p_L</i> = 0.85
Temporal	-15.3 ± 21.6 (0.006)	-14.4 ± 11.6 (0.006)	
Occipital	-24.5 ± 26.9 (0.013)	-7.1 ± 19.8 (0.39)	
MSQRES	-27.8 ± 23.3 (<0.001)	-13.3 ± 20.9 (0.003)	<i>p_G</i> = 0.01
Frontal	25.7 ± 23.2 (<0.001)	-11.8 ± 29.6 (0.24)	<i>p_L</i> = 0.83
Temporal	-29.1 ± 22.5 (<0.001)	-16.3 ± 17.3 (0.02)	
Occipital	-29.2 ± 26.9 (0.005)	-11.4 ± 9.1 (0.02)	
SFPH	-4.9 ± 8.7 (0.003)	-2.1 ± 8.7 (0.23)	<i>p_G</i> = 0.20
Frontal	5.9 ± 6.9 (0.002)	1.5 ± 7.0 (0.51)	<i>p_L</i> = 0.87
Temporal	-5.4 ± 11.4 (0.056)	-0.89 ± 8.4 (0.76)	
Occipital	-2.1 ± 5.6 (0.24)	-8.7 ± 8.6 (0.4)	

p_G, comparison between actual and sham surgery (*p* value computed from two-sample *t* test and verified by Wilcoxon rank test); *p_L*, comparisons among the trigger sites; MH, migraine headaches; MIDAS, Migraine Disability Assessment Score; MSQEM, Migraine-Specific Quality of Life, emotional; MSQPRE, Migraine-Specific Quality of Life, preventive; MSQRES, Migraine-Specific Quality of Life, restrictive; SFPH, Medical Outcomes Study 36-Item Short Form Health Survey, physical.

*The *p* values inside parentheses adjacent to each measure represent the comparison between baseline and 12 months using paired analysis. Continuous data are represented as mean ± SD.

tures over 12 months was significantly improved from the baseline in the actual surgery group (*p* < 0.01 for all).

Logistic Regression Analysis

To identify the factors affecting the significant improvements defined above, we used multivariable logistic regression modeling and set “significant improvement” as the dependent variable. All baseline data, including migraine measures, gender, trigger sites, and types of surgery, were used as independent variables. The only significant factor affecting improvement was actual surgery (*p* = 0.016), with an adjusted odds ratio of 3.97.

Adverse Events

The complications and the frequencies with which they occurred are listed in Table 3. All pa-

Table 3. Adverse Events

Nature of Adverse Event	No. (%)	Group
Numbness 1 yr postoperatively	1 (5)	T
Temporal hollowing	10 (53)	T
Temporary intense itching	2 (11)	F
Uneven brow movement	1 (5)	F
Temporary hair loss or thinning	1 (5)	T
Residual CSC muscle function	1 (5)	F
Neck stiffness 1 yr postoperatively	1 (9)	O

F, frontal (*n* = 19); T, temporal (*n* = 19); O, occipital (*n* = 11); CSC, corrugator supercilii.

tients reported some degree of paresthesia in the immediate postoperative period. Only one patient experienced persistent forehead numbness after 1 year. No neuromas were observed. A slight degree of temple hollowing was noted in 10 of 19 patients

in actual surgery group T. Temporary intense pruritus was reported by two patients. Slight asymmetric eyebrow movement was noted in one patient and temporary hair loss or thinning was seen in one patient. One patient noted residual function of the corrugator supercilii, presumably as a result of incomplete resection or regeneration of the muscle. This was associated with corresponding residual migraine headaches. One patient reported some neck stiffness 1 year postoperatively in actual surgery group O. No adverse events were observed in the sham surgery group.

DISCUSSION

The high incidence of improvement of symptoms in the sham surgery group is intriguing. Some of this may be attributable to the placebo effect and is similar to what other studies have found.¹⁸ However, it is also possible that the incision and the undermining of the tissues may have, to some extent, altered neurosensory function, at least temporarily. In addition, it is possible that some of these patients exaggerated their preoperative symptoms to increase their chance of selection. Nevertheless, when the surgical treatment group as a whole was analyzed and when each surgery site was assessed separately, the surgical treatment group had a statistically significant improvement. More importantly, whereas only 3.8 percent of patients in the placebo group reported complete elimination of symptoms, 57.1 percent in the actual surgery group experienced complete elimination of migraine headaches.

The pathophysiologic mechanism underlying migraine headaches is poorly understood. A number of hypotheses have emerged regarding the neural events mediating the initiation of migraine headaches. Some have postulated that cortical neuronal hyperexcitability is the culprit.^{19,20} Others have suggested that cortical spreading depression, the basis of auras, is the cause.²¹ Others feel that peripheral and central activation and sensitization of the trigeminal system culminates in migraine headaches.^{22,23} Lastly, abnormal modulation of brain nociceptive systems because of dysfunctional periaqueductal gray matter and alteration of its facilitatory or inhibitory pain-processing functions may trigger migraine headaches.¹⁹ Of these hypotheses, the one that is supported by sufficient scientific evidence and that has the most relevance to our findings is peripheral activation of the trigeminal nerve, with subsequent peripheral and central sensitization.

Migraine headaches are also postulated to be caused by dilatation of the meningeal vasculature

innervated by the trigeminal nerve and activation of perivascular sensory fibers supplying the dura mater following an episode of cortical spreading depression and meningeal inflammation.^{24–31} Vasodilatation is the consequence of meningeal nociceptor-induced release of calcitonin gene-related peptide, substance P, and neurokinin A, which are found in the cell bodies of trigeminal neurons.^{32–36} Studies have shown that peripheral inflammation leads to increased excitability of central neurons (central sensitization) by means of the release of neuropeptides, resulting in amplification of sensory inputs, including exaggerated responses to stimuli that are normally innocuous.^{37–39} What precipitates the initial release of these peptides is unknown.

Burstein et al. demonstrated that sensitization of nociceptors results in increased spontaneous neuronal discharges, with subsequent increased receptiveness to both painful and nonpainful stimuli.⁴⁰ Often, the receptive fields are expanded and patients feel pain over a greater portion of the dermatome, clinically recognized as cutaneous allodynia. A number of studies have shown that stimulation of muscle afferents increases the excitability of central neurons, and muscle afferents appear to be more effective at inducing changes in central neuron responsivity than cutaneous afferents.^{41–43} Previous anatomical studies have shown that the three trigger sites investigated in the current study contain sensory nerves that traversed the muscle to reach the skin, providing a setting for mechanical stimulation and irritation of the trigeminal and greater occipital nerves.^{44–46} Based on those findings, the research team designed a randomized comprehensive study that included surgical treatment of four trigger sites, three of which were deactivated using the techniques described here.¹⁰ Of the 89 patients, 82 reported at least 50 percent reduction in the frequency, intensity, and duration of migraine headaches, supported by the response to validated questionnaires. Although this was a randomized study, it did not include a sham surgery group.

In a recent report, Jakubowski et al. categorized migraine headaches as exploding or imploding.⁴⁷ Imploding headaches, which have a greater likelihood of responding to Botox, may be prompted by stimulation of the superficial branches of the trigeminal nerve, which pass through the cervicofacial muscle. Exploding migraine headaches may originate in the deeper branches of the trigeminal nerve within the lining of the turbinates, septum, and sinuses and consequently are impervious to the effects of Botox. Therefore, for

patients with exploding migraine headaches, elimination of friction between the deviated septum and enlarged turbinates or concha bullosa may provide lasting improvement.⁴⁸⁻⁵⁰

CONCLUSIONS

In the current study, one reason why not every patient observed complete elimination is that the operation was performed on a single predominant trigger site, which was not necessarily the *only* trigger site. Although the pain originating from the dominant trigger site was eliminated, migraine headaches originating from another trigger site and extending to the surgery target area were not stopped. However, these migraine headaches were less severe, less frequent, and more easily controlled with traditional abortive therapy after surgery. Lastly, when the failed cases were analyzed, it appeared that the predominant migraine headache trigger site was incorrectly assigned in some instances.

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