Morphometric Evaluation of the Frontal Migraine Trigger Site

Jessica J. Hsu, M.D., Ph.D.
Ashley M. Stasiak, M.D.
Kavitha Ranganathan, M.D.
Eric E. Adelman, M.D.
Jeffrey E. Janis, M.D.
Paul S. Cederna, M.D.
Theodore A. Kung, M.D.

Ann Arbor, Mich.; Los Angeles, Calif.; and Columbus, Ohio

Background: Migraine headache has been attributed to specific craniofacial peripheral nerve trigger sites. Some have postulated that hypertrophy of the corrugator muscles causes compression of the supraorbital and supratrochlear nerves, resulting in migraine headache. This study uses morphometric evaluation to determine whether corrugator anatomy differs between patients with migraine headache and control subjects.

Methods: A retrospective review identified patients with and without migraine headache who had a recent computed tomographic scan. Morphometric evaluation of the corrugator supercilii muscles was performed in a randomized and blinded fashion on 63 migraine headache and 63 gender-matched control patients using a three-dimensional image-processing program. These images were analyzed to determine whether corrugator size differed between migraine and control patients.

Results: There was no difference in mean corrugator volume or thickness between migraine and control patients. The mean corrugator volume was 1.01 ± 0.26 cm^3 compared with 1.06 ± 0.27 cm^3 in control patients ($p = 0.258$), and the mean maximum thickness was 5.36 ± 0.86 mm in migraine patients compared with 5.50 ± 0.91 mm in controls ($p = 0.359$). Similarly, subgroup analysis of 38 patients with frontal migraine and 38 control subjects demonstrated no difference in corrugator size. Further subgroup analysis of nine patients with unilateral frontal migraine showed no difference in corrugator size between the symptomatic side compared with the contralateral side.

Conclusions: Muscle hypertrophy itself does not play a major role in triggering migraine headache. Instead, factors such as muscle hyperactivity or peripheral nerve sensitization may be more causative. (*Plast. Reconstr. Surg.* 141: 726e, 2018.)

Migraine headache is a common and debilitating condition that affects approximately 10 percent of the population worldwide, with approximately twice as many women affected as men.[1] Historically, migraine headache has been considered a medical diagnosis reflective of an intracerebral cascade of events, with pharmacologic intervention as the mainstay for first-line treatment.[2] A surgical treatment for medically refractory migraine headache was discovered when patients who originally underwent cosmetic forehead rejuvenation surgery subsequently had elimination or significant improvement of their migraine headache, a finding that has since been validated by multiple authors.[3–12] The prevailing theory was that resection of the corrugator supercilii muscles leads to decompression of the supraorbital and supratrochlear nerves. The resultant mitigation of migraine headache reinforced that, at least in a proportion of patients, pain may be mediated by a peripheral

From the Department of Surgery, Section of Plastic and Reconstructive Surgery, and the Department of Neurology, University of Michigan Health System; the Department of Surgery, Division of Plastic and Reconstructive Surgery, University of Southern California; and the Department of Plastic Surgery, The Ohio State University Wexner Medical Center.
Received for publication June 4, 2017; accepted November 15, 2017.
Presented at the 2016 Annual Meeting of the American Society for Peripheral Nerve, in Scottsdale, Arizona, January 15 through 16, 2017; and the 61st Annual Meeting of the Plastic Surgery Research Council, in New York, New York, May 19 through 22, 2016.

DOI: 10.1097/PRS.0000000000004296

Disclosure: *Dr. Janis is a consultant for LifeCell, Bard, and Daiichi Sankyo, has received prior honoraria from Pacira and KCI, and receives royalties from Thieme Publishing. The remaining authors have no financial interest to declare in relation to the content of this article. The other authors have no commercial associations or financial disclosures. No funding was received for this article.*

mechanism outside the brain secondary to muscle compression around these nerves.[3]

This mechanism for migraine headache has been further supported by the use of botulinum toxin as a neuromodulator or chemodenervation agent for both treatment and diagnostic purposes in patients with migraines refractory to medical management.[10,13–17] For patients with refractory migraine headache, surgical decompression of the nerves at identified migraine trigger sites reduces migraine frequency and improves symptomatology in more than 70 to 80 percent of pati ents.[8,9,12,17–19]

The anatomical pathways of these nerves have been carefully defined as they relate to possible points of compression, including muscle, bone, fascia, and vessel.[20–24] Given their close relationship, it has been postulated that frontal migraine headache may be triggered in part by corrugator supercilii hypertrophy in some patients.[25] This would be akin to other peripheral nerve compression syndromes that are associated with hypertrophy of adjacent muscles, such as quadrilateral space syndrome,[26,27] pronator teres syndrome,[28,29] and piriformis syndrome.[30]

The purpose of this study was to determine whether there are anatomical differences in corrugator size in patients with known migraine headache that may contribute to compression of the supratrochlear or supraorbital nerves. This investigation uses morphometric analysis to determine whether hypertrophy of the corrugator supercilii is associated with migraine headache.

PATIENTS AND METHODS

Study Design and Population

This study was performed with approval from the University of Michigan Institutional Review Board. To determine whether increased corrugator supercilii volume or thickness is associated with migraine headache, a retrospective review was conducted to identify patients with the diagnosis of migraine headache who were followed by a neurologist at the University of Michigan during a 10-year period (2002 to 2011) and had undergone computed tomographic study of the head or brain. Control patients consisted of patients who had computed tomographic scans of the head or brain but did not have a diagnosis of migraine headache. These were typically trauma patients who underwent head, brain, or maxillofacial scans as part of whole-body imaging for their trauma workup, but did not have any head or maxillofacial trauma. Patients were excluded from either group if they had age younger than 18 years; psychiatric hospitalization; psychiatric diagnosis other than depression or anxiety; history of trauma to the forehead, glabellar region, temporal region, nose, or skull base; previous rhinoplasty, forehead lift, or craniofacial surgery; any intracranial abnormality; temporal arteritis; or atypical facial pain. These attributes were identified by the study neurologist as potential confounders in migraine headache diagnosis or morphometric analysis. Demographic information including age; sex; date of initial evaluation by a neurologist; and medical comorbidities including sinusitis, history of sinus surgery, anxiety/depression, diabetes, and smoking were collected retrospectively from chart analysis. For migraine patients, information was also collected regarding headache sidedness, trigger site, and history of botulinum toxin injection.

Subgroup Analysis

Subgroup analysis was performed for migraine patients who specifically reported frontal trigger-site initiation of migraine headache. Because the study population reflects the known female predominance in migraine patients,[1] corrugator volume and thickness of this subgroup was compared to that of gender-matched control subjects. Additional subgroup analysis was performed for migraine patients with unilateral frontal migraine headache. In these patients, the mean corrugator volume and thickness values of the symptomatic side were compared with the respective mean values from the contralateral asymptomatic side, which served as the internal control.

Morphometric Analysis

Computed tomographic scan data (2- to 5-mm slice width) were imported into VitreaCore (Vital Images, Inc., Minnetonka, Minn.) for analysis. VitreaCore is a commercially available software program used for three-dimensional reformatting of radiographic images. It is a powerful tool previously used for accurate volumetric measurement of anatomical structures much smaller than the corrugator muscles, such as the vestibular aqueduct[31] and lumbar foramina.[32] Three-dimensional reconstruction was performed from axial, coronal, and sagittal images, which were then used to define the borders of the left and right corrugators for each patient in a blind and randomized fashion. The three-dimensional shape and volume of each corrugator muscle was determined by the VitreaCore program using multiobject segmentation (Fig. 1).

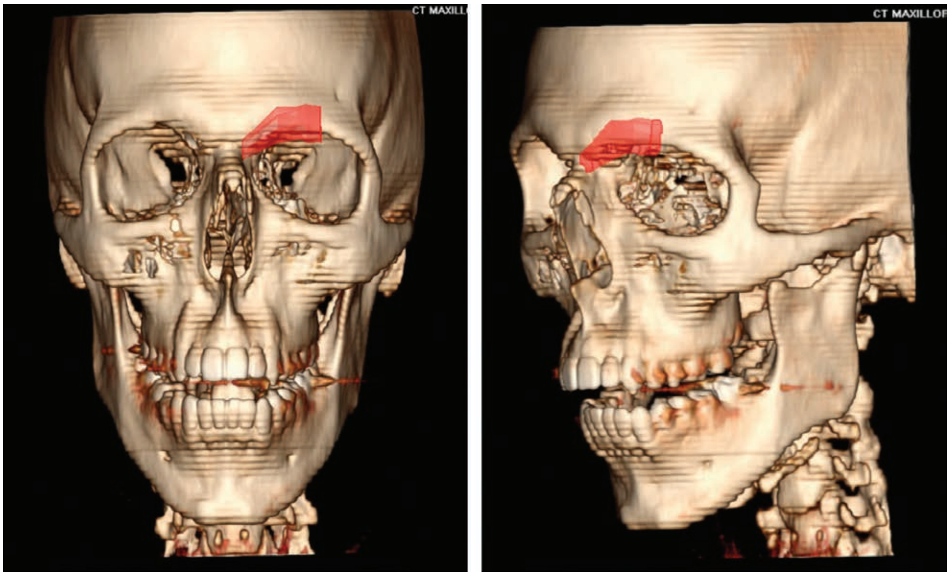

Fig. 1. Corrugator supercilii muscle defined for measurement using VitreaCore. The boundaries of the corrugator supercilii muscle were identified using axial, coronal, and sagittal computed tomographic images within VitreaCore for measurement of corrugator volume and thickness. This was performed for the left and right corrugator muscles for all migraine patients and control subjects.

Statistical Analysis

Prestudy power analysis demonstrated that 63 patients in each group would be more than adequate to detect a 15 percent difference in corrugator volume or thickness. Patient characteristics and measurements were compared between migraine and control groups using *t* tests for continuous variables and chi-square or Fisher's exact tests for categorical variables. Independent paired *t* test was used to compare the affected to the unaffected side in patients with unilateral migraine headache. For numerical variables, the data are presented as the mean with standard deviation. For categorical variables, the data are presented as percentages. Multivariate logistic regression was performed using collected patient characteristics as variables (i.e., diagnosis of depression and/or anxiety, history of sinusitis, diabetes, and tobacco use). History of sinus surgery could not be included in the regression analysis because of perfect separation, as no control subjects had a history of sinus surgery. Values of $p < 0.05$ were considered statistically significant. All analyses were performed using IBM SPSS Version 24 (IBM Corp., Armonk, N.Y.).

RESULTS

Summary of Demographic Data

Sixty-three patients with migraine and 63 gender-matched control patients were included in our study. Patient characteristics are listed in Table 1. Each study population included 46 women (73 percent) and 17 men (27 percent). The mean age of the migraine group was 40 ± 13 years and the mean age of the control group was 38 ± 20 years ($p = 0.407$).

Multivariate Analysis of Factors Associated with Migraine Headache

In addition to determining corrugator size, we also collected patient characteristic data and other variables relevant to the diagnosis and treatment of migraine headache or other peripheral nerve pathophysiology, including history of sinus disease and treatment, depression/anxiety, diabetes, and smoking status. Results from logistic regression analysis controlling for other covariates are shown in Table 2. After controlling for covariates, significantly higher odds of suffering from migraine headache were found with co-diagnosis of anxiety or depression (OR, 3.711; 95 percent CI, 1.536 to 8.968; $p = 0.0036$), history of sinusitis (OR, 15.436; 95 percent CI, 3.304 to 72.109; $p = 0.0005$), and nonsmoking status (OR, 5.917; 95 percent CI, 1.664 to 20.833; $p = 0.0060$). The association of migraine headache with diabetes was not statistically significant. These attributes were not considered potential confounders and were not used for subgroup analyses, as they are not expected to have any anatomical effect on corrugator size.

Corrugator Size in Migraine Patients

The corrugator supercilii volume and thickness were measured from computed tomographic

Table 1. Patient Characteristics of the Study Population

Variable	Migraine Group (%)	Nonmigraine Control Group (%)	p*
No. of patients	63	63	
Sex			
Female	46 (73)	46 (73)	
Male	17 (27)	17 (27)	1.000
Mean age ± SD, yr	40 ± 13	38 ± 20	0.407
Sinusitis	23 (37)	2 (3)	<0.0001
History of sinus surgery	14 (22)	0 (0)	<0.0001
Depression/anxiety	35 (56)	20 (32)	0.0071
Smoker	5 (8)	21 (33)	0.0004
Diabetes	8 (13)	2 (3)	0.0480

*Based on χ^2 test or Fisher's exact test. The t test was used for age.

Table 2. Association of Patient Factors with Migraine Headache

Variable	OR	95% CI	p
Sinusitis	15.436	3.304–72.109	0.0005
Depression/anxiety	3.711	1.536–8.968	0.0036
Smoking	0.169	0.048–0.601	0.0060
Diabetes	5.049	0.897–28.433	0.0663

Table 3. Corrugator Volume and Thickness in the Full Study Population

	Migraine Group	Nonmigraine Control Group	p*
No. of patients, total study population	63	63	
Mean corrugator volume ± SD, cm³	1.01 ± 0.26	1.06 ± 0.27	0.258
Mean corrugator thickness ± SD, mm	5.36 ± 0.86	5.50 ± 0.91	0.359

*Based on t test.

Table 4. Corrugator Volume and Thickness in Frontal Migraine Patients

Variable	Migraine Group (%)	Nonmigraine Control Group (%)	p*
No. of patients	38	38	
Sex			
Female	32 (84)	32 (84)	
Male	6 (16)	6 (16)	
Mean age ± SD, yr	41 ± 13	44 ± 21	0.517
Mean corrugator volume ± SD, cm³	1.00 ± 0.26	1.04 ± 0.24	0.539
Mean corrugator thickness ± SD, mm	5.33 ± 0.82	5.40 ± 0.83	0.709

*Based on t test.

scans of all migraine and control patients using the three-dimensional image processing software VitreaCore (Fig. 1). Mean corrugator volume in migraine patients was 1.01 ± 0.26 cm³ compared with 1.06 ± 0.27 cm³ in control patients ($p = 0.258$), and the mean maximum thickness was 5.36 ± 0.86 mm in migraine patients compared with 5.50 ± 0.91 mm in controls ($p = 0.359$). Results are summarized in Table 3.

Subgroup Analysis with Frontal Migraine

Within our study population, 39 patients reported migraine headaches specifically involving the frontal trigger site. One patient was excluded because of a history of botulinum toxin treatment. We compared the corrugator volume and thickness of these 38 frontal migraine patients to that of 38 gender-matched, control patients and found that there was no difference between the two groups (Table 4). The mean corrugator volume for migraine patients was 1.00 ± 0.26 cm³ compared with 1.04 ± 0.24 cm³ for control patients ($p = 0.539$). Similarly, the mean maximum corrugator thickness for migraine patients was 5.33 ± 0.82 mm compared with 5.40 ± 0.83 mm for control patients ($p = 0.709$).

Subgroup Analysis with Unilateral Frontal Migraine

Among migraine patients with a known frontal trigger, nine patients reported that their headaches originated from a specific side. In these patients, we measured the corrugator supercilii volume and maximum thickness from the symptomatic side and compared it to the contralateral side as a control. We found no difference in corrugator size between the affected and unaffected sides (Table 5). The mean corrugator volume of the symptomatic side was 1.01 ± 0.20 cm³ compared with 1.04 ± 0.17 cm³ on the contralateral side ($p = 0.674$). Similarly, the mean maximum corrugator thickness of the symptomatic side was 5.51 ± 0.66 mm compared with 5.44 ± 1.07 mm on the contralateral, nonsymptomatic side ($p = 0.764$).

DISCUSSION

Our study found no evidence of corrugator hypertrophy (i.e., increased maximum thickness or volume) in migraine patients compared to control subjects. Subgroup analysis of migraine patients with primarily frontal-site triggering compared to a gender-matched group of control subjects showed no difference in either corrugator volume or thickness. Furthermore, there was no difference in corrugator volume or thickness between the symptomatic side and the contralateral nonsymptomatic side in the nine patients with unilateral frontal migraine headache. Considered together, our study suggests that corrugator supercilii hypertrophy is not the root cause of

Table 5. Corrugator Volume and Thickness in Unilateral Migraine Patients

Variable	Migraine Side (%)	Contralateral Nonmigraine Side (%)	p*
No. of patients		9	
Sex			
Female	8 (89)		
Male	1 (11)		
Mean age ± SD, yr	45 ± 15		
Mean corrugator volume ± SD, cm^3	1.01 ± 0.20	1.04 ± 0.17	0.674
Mean corrugator thickness ± SD, mm	5.51 ± 0.66	5.44 ± 1.07	0.764

*Based on paired t test.

supraorbital or supratrochlear nerve compression that may contribute to migraine headache.

A peripheral nerve–mediated mechanism is supported by the preponderance of data using botulinum toxin as a diagnostic tool and for efficacious albeit temporary treatment of migraine headache.[10,13–17] Furthermore, examination of the terminal branches of the trigeminal nerve in patients with and without migraine headache has demonstrated both proteomic and electron microscopic abnormalities, suggesting a peripheral mechanism for migraine headache.[33] Finally, surgical decompression of peripheral nerves has been shown to be highly effective, with improvement and elimination rates of greater than 70 percent at multiple trigger sites, across multiple studies, and with consistent results with surgeons at different institutions.[3–12]

It is important to continue to elucidate the mechanism for migraine headache so that we can more accurately diagnose and optimize treatments for this disorder, particularly to identify those individuals that would benefit most from surgical intervention. One of the goals of this study was to use noninvasive means such as radiographic imaging to determine whether it was possible to discern any anatomical differences in migraine patients compared with control subjects. Anatomical studies have been conducted to further characterize the supraorbital and supratrochlear nerves and the bony structure where the supraorbital nerve exits the skeleton as a potential site of nerve compression.[20–24] In one study, Fallucco et al. found that, 27 percent of the time, the supraorbital nerve exits through a bony foramen; however, 83 percent of the time, the nerve exits through a notch. In 86 percent of supraorbital notches, there is a fascial band that, together with the bony notch, encircles the supraorbital nerve, rendering it vulnerable to compression.[22] The majority of these studies have been cadaveric and so the relationship between anatomical variants and migraine pathophysiology remains unknown.[22,24] We attempted to examine the bony structure along the supraorbital rim using our computed tomographic data and VitreaCore; however, it was not always possible to discern notch from foramen. For the nine patients who reported unilateral migraine headache, several individuals had the same supraorbital rim morphology bilaterally. The remaining two patients had a notch on the migraine side and a bony foramen on the contralateral nonmigraine side (unreported data). This finding could potentially be clinically significant, but a much larger study with adequate sample sizes would be required.

This study was limited by its retrospective design and reliance on accurate patient reporting, physician diagnosis, and documentation, not only of migraine characteristics, but also medical and surgical history. Our regression analysis suggests that migraine headache is associated with depression and anxiety and history of sinusitis. Although the confidence intervals are wide, this is consistent with previously described findings that associate migraine headache with psychiatric diagnoses and the initial misdiagnosis with sinus maladies before being diagnosed with migraine headache.[34]

The study was designed to be well-powered with 63 migraine patients and 63 gender-matched control subjects; however, subgroup analyses were performed on fewer patients. The overall study groups (n = 63 versus n = 63) were gender-matched and reflected the female predominance of migraine headache patients. Our study relied on the use of heterogeneous computed tomographic data from different types of scans performed for various purposes; however, the corrugator supercilii was easily identified and measured in every scan. The use of VitreaCore, although novel for the purpose of measuring small facial muscles, has been validated in its use to measure even smaller anatomical structures with high accuracy across multiple observers.[31,32]

Our findings suggest that there are other important considerations aside from corrugator hypertrophy that warrant further investigation. These include determining the clinical significance of variation in additional anatomical parameters such as the course of the supraorbital and supratrochlear nerves, and the underlying bony orbital anatomy. Another future aim is to explore the possibility that corrugator supercilii muscle hyperactivity is an independent migraine trigger in the absence of muscle hypertrophy. Furthermore, the particular shape and distribution of the muscle across the supraorbital and supratrochlear nerves may be important in trigger-site activation, as muscle topography was not investigated in our study. Finally, it is likely that there are multiple factors that contribute to migraine headache that may explain why we did not find evidence of corrugator hypertrophy in our migraine population. Future investigations will aim to use other imaging modalities to further characterize corrugator trigger-site anatomy and to include other methods of studying corrugator action, including electrophysiologic measurements to directly assess corrugator activity. The goal of future studies will be to further identify differences between migraine and control groups and to better tailor treatment options for migraine headache.

CONCLUSIONS

This study demonstrates that neither the thickness nor the volume of the corrugator supercilii muscle is greater in migraine patients compared to control subjects. Furthermore, the corrugator volume and thickness were similar between the symptomatic trigger side and the contralateral asymptomatic side in patients with unilateral migraine headache. These findings indicate that muscle hypertrophy does not significantly contribute to migraine headache and instead suggest that other mechanisms, such as abnormal muscle activity or nerve entrapment, may mediate migraine headache.

Theodore A. Kung, M.D.
University of Michigan Health System
1500 East Medical Center Drive
Ann Arbor, Mich. 48109
thekung@med.umich.edu

ACKNOWLEDGMENTS

The authors thank Eric Wizauer for time and assistance with VitreaCore and the University of Michigan Consulting for Statistics, Computing, and Analytics Research and Niki Matusko for guidance with statistical analysis.

REFERENCES

1. Woldeamanuel YW, Cowan RP. Migraine affects 1 in 10 people worldwide featuring recent rise: A systematic review and meta-analysis of community-based studies involving 6 million participants. *J Neurol Sci.* 2017;372:307–315.
2. Silberstein SD, Lipton RB. *21st Century Prevention and Management of Migraine Headaches.* Califon, NJ: Gardiner-Caldwell SynerMed; 2001.
3. Guyuron B, Varghai A, Michelow BJ, Thomas T, Davis J. Corrugator supercilii muscle resection and migraine headaches. *Plast Reconstr Surg Glob Open* 2000;106:429–434; discussion 435–437.
4. Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. *Plast Reconstr Surg.* 2002;109:2183–2189.
5. Dirnberger F, Becker K. Surgical treatment of migraine headaches by corrugator muscle resection. *Plast Reconstr Surg.* 2004;114:652–657; discussion 658–659.
6. Guyuron B, Kriegler JS, Davis J, Amini SB. Comprehensive surgical treatment of migraine headaches. *Plast Reconstr Surg.* 2005;115:1–9.
7. Poggi JT, Grizzell BE, Helmer SD. Confirmation of surgical decompression to relieve migraine headaches. *Plast Reconstr Surg.* 2008;122:115–122; discussion 123–124.
8. Guyuron B, Reed D, Kriegler JS, Davis J, Pashmini N, Amini S. A placebo-controlled surgical trial of the treatment of migraine headaches. *Plast Reconstr Surg.* 2009;124:461–468.
9. Guyuron B, Kriegler JS, Davis J, Amini SB. Five-year outcome of surgical treatment of migraine headaches. *Plast Reconstr Surg.* 2011;127:603–608.
10. Janis JE, Dhanik A, Howard JH. Validation of the peripheral trigger point theory of migraine headaches: Single-surgeon experience using botulinum toxin and surgical decompression. *Plast Reconstr Surg.* 2011;128:123–131.
11. Gfrerer L, Maman DY, Tessler O, Austen WG Jr. Nonendoscopic deactivation of nerve triggers in migraine headache patients: Surgical technique and outcomes. *Plast Reconstr Surg.* 2014;134:771–778.
12. Janis JE, Barker JC, Javadi C, Ducic I, Hagan R, Guyuron B. A review of current evidence in the surgical treatment of migraine headaches. *Plast Reconstr Surg Glob Open* 2014;134:131S–141S.
13. Aurora SK, Dodick DW, Turkel CC, et al.; PREEMPT 1 Chronic Migraine Study Group. OnabotulinumtoxinA for treatment of chronic migraine: Results from the double-blind, randomized, placebo-controlled phase of the PREEMPT 1 trial. *Cephalalgia* 2010;30:793–803.
14. Diener HC, Dodick DW, Aurora SK, et al.; PREEMPT 2 Chronic Migraine Study Group. OnabotulinumtoxinA for treatment of chronic migraine: Results from the double-blind, randomized, placebo-controlled phase of the PREEMPT 2 trial. *Cephalalgia* 2010;30:804–814.
15. Liu MT, Armijo BS, Guyuron B. A comparison of outcome of surgical treatment of migraine headaches using a constellation of symptoms versus botulinum toxin type A to identify the trigger sites. *Plast Reconstr Surg.* 2012;129:413–419.
16. Nahabet E, Janis JE, Guyuron B. Neurotoxins: Expanding uses of neuromodulators in medicine—Headache. *Plast Reconstr Surg.* 2015;136(Suppl):104S–110S.
17. Janis JE, Barker JC, Palettas M. A retrospective review of targeted peripheral nerve-directed onabotulinum toxin A injection for effective long-term therapy for migraine headache: Technique and outcomes. *Plast Reconstr Surg Glob Open* 2017;5:e1270.
18. Kung TA, Pannucci CJ, Chamberlain JL, Cederna PS. Migraine surgery practice patterns and attitudes. *Plast Reconstr Surg.* 2012;129:623–628.
19. Kurlander DE, Ascha M, Sattar A, Guyuron B. In-depth review of symptoms, triggers, and surgical deactivation of frontal migraine headaches (site I). *Plast Reconstr Surg.* 2016;138:681–688.
20. Janis JE, Ghavami A, Lemmon JA, Leedy JE, Guyuron B. Anatomy of the corrugator supercilii muscle: Part I. Corrugator topography. *Plast Reconstr Surg.* 2007;120:1647–1653.

21. Janis JE, Ghavami A, Lemmon JA, Leedy JE, Guyuron B. The anatomy of the corrugator supercilii muscle: Part II. Supraorbital nerve branching patterns. *Plast Reconstr Surg.* 2008;121:233–240.
22. Fallucco M, Janis JE, Hagan RR. The anatomical morphology of the supraorbital notch: Clinical relevance to the surgical treatment of migraine headaches. *Plast Reconstr Surg.* 2012;130:1227–1233.
23. Janis JE, Hatef DA, Hagan R, et al. Anatomy of the supratrochlear nerve: Implications for the surgical treatment of migraine headaches. *Plast Reconstr Surg.* 2013;131:743–750.
24. Hagan RR, Fallucco MA, Janis JE. Supraorbital rim syndrome: Definition, surgical treatment, and outcomes for frontal headache. *Plast Reconstr Surg Glob Open* 2016;4:e795.
25. Chepla KJ, Oh E, Guyuron B. Clinical outcomes following supraorbital foraminotomy for treatment of frontal migraine headache. *Plast Reconstr Surg.* 2012;129:656e–662e.
26. Francel TJ, Dellon AL, Campbell JN. Quadrilateral space syndrome: Diagnosis and operative decompression technique. *Plast Reconstr Surg.* 1991;87:911–916.
27. Paladini D, Dellantonio R, Cinti A, Angeleri F. Axillary neuropathy in volleyball players: Report of two cases and literature review. *J Neurol Neurosurg Psychiatry* 1996;60:345–347.
28. Rehak DC. Pronator syndrome. *Clin Sports Med.* 2001;20:531–540.
29. Andreisek G, Crook DW, Burg D, Marincek B, Weishaupt D. Peripheral neuropathies of the median, radial, and ulnar nerves: MR imaging features. *Radiographics* 2006;26:1267–1287.
30. Jankiewicz JJ, Hennrikus WL, Houkom JA. The appearance of the piriformis muscle syndrome in computed tomography and magnetic resonance imaging: A case report and review of the literature. *Clin Orthop Relat Res.* 1991;262:205–209.
31. Hwang M, Marovich R, Shin SS, Chi D, Branstetter BF IV. Optimizing CT for the evaluation of vestibular aqueduct enlargement: Inter-rater reproducibility and predictive value of reformatted CT measurements. *J Otol.* 2015;10:13–17.
32. Khiami F, Aziria SA, Ragot S, et al. Reliability and validity of a new measurement of lumbar foraminal volume using a computed tomography. *Surg Radiol Anat.* 2015;37:93–99.
33. Guyuron B, Yohannes E, Miller R, Chim H, Reed D, Chance MR. Electron microscopic and proteomic comparison of terminal branches of the trigeminal nerve in patients with and without migraine headaches. *Plast Reconstr Surg Glob Open* 2014;134:796e–805e.
34. Al-Hashel JY, Ahmed SF, Alroughani R, Goadsby PJ. Migraine misdiagnosis as a sinusitis, a delay that can last for many years. *J Headache Pain* 2013;14:97.