



Anatomic and Compression Topography of the Lesser Occipital Nerve

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Background: The surgical treatment of occipital headaches focuses on the greater, lesser, and third occipital nerves. The lesser occipital nerve (LON) is usually transected with relatively limited available information regarding the compression topography thereof and how such knowledge may impact surgical treatment.

Methods: Eight fresh frozen cadavers were dissected focusing on the LON in relation to 3 clinically relevant compression zones. The *x* axis was a line drawn through the occipital protuberance (OP) and the *y* axis, the posterior midline (PM). In addition, a prospectively collected cohort of 36 patients who underwent decompression of the LON is presented with their clinical results, including migraine headache index scores.

Results: The LON was found in compression zone 1, with a mean of 7.8 cm caudal to the OP and 6.3 cm lateral to the PM. The LON was found at the midpoint of compression zone 2, with an average of 5.5 cm caudal to the OP and 6.2 cm lateral to the PM. At compression zone 3, the medial-most LON branch was located approximately 1 cm caudal to the OP and 5.35 cm lateral to the PM, whereas the lateral-most branch was identified 1 cm caudal to the OP and 6.5 cm lateral to the PM. Of the 36 decompression patients analyzed, only 5 (14%) required neurectomy as the remainder achieved statistically significant improvements in migraine headache index scores postoperatively.

Conclusion: The knowledge of LON anatomy can aid in nerve dissection and preservation, thereby leading to successful outcomes without requiring neurectomy. (*Plast Reconstr Surg Glob Open* 2016;4:e639; doi: 10.1097/GOX.0000000000000654; Published online 17 March 2016.)

Chronic headaches represent a huge medical burden in the United States and worldwide. In the United States alone, it is estimated that

there are approximately 36 million people experiencing chronic migraines resulting in estimated annual costs of over \$15 billion to the medical system.^{1,2} Chronic headaches such as migraines were responsible for over 100 million days of bedrest in 1999 and yearly expenditures per patient of over \$11,000 in terms of both direct and indirect costs.^{3,4} More telling, in a study measuring health-related quality of life, Turner-Bowker et al⁵ were able to demonstrate that the health-related quality of life of migraineurs is similar to that of patients with congestive heart failure, diabetes, and hypertension. Such results underscore

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the impact, severity, and chronic nature of headaches such as migraines, despite their sometimes episodic characteristics. Pharmacologic therapy has traditionally been the mainstay for the treatment of chronic headaches, and yet, the results with some of the more well-known agents are rather underwhelming.⁶⁻⁸

In a number of studies, the surgical treatment of chronic headaches has been demonstrated to be effective to treat severe headaches that are refractory to medical management and to nonsurgical therapeutic options, such as radiofrequency ablation, physical therapy, and acupuncture.^{9,10} First described by Guyuron et al⁹ and later expounded upon by others, there are now 4 well-established zones where surgical intervention can be implemented: endonasal, frontal, temporal, and occipital.¹¹⁻¹⁷ Subsequent work has clearly shown that neuroanatomic knowledge of these regions can aid the headache surgeon in adequately decompressing the relevant nerves or in deciding when to perform a neurectomy and implantation.

Within the occipital region, the surgical targets are typically the greater occipital, lesser occipital (LON), and third occipital nerves. The first of these nerves is the largest and also happens to have the biggest area of sensory distribution within the posterior scalp. Not surprisingly, a number of authors have defined the pathway that the greater occipital nerve takes through the posterior nuchal musculature until it is located in the subcutaneous tissues within the posterior scalp.¹⁸ However, there are times when a patient with occipital headaches has involvement of the other “minor” occipital nerves. Interestingly, most articles that discuss the surgical management of chronic, occipital headaches suggest that the LON and third occipital nerve are commonly resected as a first-line approach.^{19,20} The presumption with this approach is that the sensory distribution of these nerves is small enough to be clinically irrelevant after neurectomy and/or that the risks of neurectomy are the same as those of decompression/neuroplasty. However, a transected nerve must be implanted deeply within a large muscle and carries the risk of neuroma formation if extruded.²¹⁻²³ In addition, after exposure and decompression/neurolysis, sometimes the nerve seems viable, hence allowing for a clinical judgment to be made regarding preservation of nerve integrity whenever possible to minimize the risks noted above and also to maintain optimal sensation to the relevant head and neck region(s). Therefore, the purpose of this study was to further define the compression topography of the LON and correlate these findings with a clinical series of patients who

underwent LON decompression/neuroplasty with or without surgical treatment of other nerves to elucidate whether the knowledge of this anatomy and decompression would yield results comparable with LON neurectomy and implantation.

MATERIALS AND METHODS

A series of 8 fresh frozen cadaver head dissections were performed specifically looking for the LON. The neuroanatomy of the LON was measured in relation to well-established occipital landmarks, such as the occipital protuberance (OP), the posterior midline (PM), and the nuchal line (NL), defined as the line connecting the external auditory meatuses. In addition, the nerve was carefully examined in relation to 3 potential compression zones noted by the senior author (Z.M.P.) in his prior clinical experience. The 3 zones of compression noted during prior LON procedures were as follows: zone 1—the emergence of the LON from behind/deep to the sternocleidomastoid muscle (SCM), zone 2—the ascent of the nerve cephalically along the posterior border of or posterior to the SCM, and zone 3—the crossing point of the LON at the NL. Fifteen LONs were identified in the 8 heads examined throughout the nerve course as noted above; 1 nerve on 1 side was not found on 1 specimen. Unfortunately, we did not have control over specimen preparation as they were kindly donated by the University Hospital of Lausanne (CHUV), and there was a small amount of variability in the craniocaudal level and obliquity of the cuts used. This small discrepancy or a simple avulsion during preparation could have accounted for the inability to find this nerve.

In addition, the senior author’s clinical experience with LON decompression/neuroplasty and neurectomy/implantation was also prospectively evaluated between 2011 and 2014. Forty patients (31 females, 9 males) who underwent decompression/neuroplasty of the LON ± surgical treatment of other relevant nerves were prospectively followed with regard to their headache symptoms. All patients had been previously seen and evaluated by a neurologist and had all failed pharmacologic management with at least several classes of medications and nonoperative modalities (eg, acupuncture, physical therapy, paraspinal injections). Patients were deemed candidates for surgical intervention based on a history consistent with nerve compression causing their headaches, a positive physical examination consisting, in part, of a positive Tinel’s sign over the relevant, suspected nerve compression site, and at least a 50% improvement in headache symptoms after focal injection of either Botulinum Toxin Type A (Botox,

Allergan Pharmaceuticals, Inc, Irvine, Calif.) or local anesthetic consisting of a lidocaine and marcaine mixture.^{17,24} Once optimal relief of symptoms was delineated, the appropriate nerves were selected for either decompression/neuroplasty or possible neurectomy/implantation.

Intraoperatively, the LON was accessed through an oblique incision parallel to the posterior border of the SCM about 4 cm in length; 3.5× loupe magnification and microneurosurgical techniques were used along with the needle-tip electrocautery through the subcutaneous tissues and the bipolar cautery exclusively deep to that layer. The incision was lengthened as needed for adequate decompression, with the longest incision length being 5.5 cm. The LON was dissected from deep to the SCM proximally and cephalic to the NL distally, and all compressive connective tissue/fascial bands, muscle insertions, and blood vessels were either cauterized or sharply divided. Each nerve treated during these procedures was released until it was ensconced in soft, subcutaneous tissues distally.

Scores of the frequency, severity, and duration of symptoms were collated to calculate a migraine headache index (MHI) value, a well-established metric used in similar studies.²⁵ The preoperative MHI scores were compared with postoperative MHI scores using a paired *t* test analysis, with *P* < 0.05 considered significant.

RESULTS

Fifteen LONs were identified in the 8 cadaver heads dissected. Overall measurements along with a graphical representation of their anatomic locations

are summarized in Figure 1. With regard to compression zone 1 (ie, emergence of the LON from deep to or behind the SCM), the LON was found an average of 7.8 cm caudal to the OP (range, 4.4–12 cm; SD ±1.84) and 6.3 cm lateral to the PM (range, 2.5–8.5 cm; SD ±1.37). Figure 2 demonstrates the cadaveric and in vivo representations of this potential compression zone. Because compression zone 2 (the cephalic ascent of the LON along or posterior to the SCM) can be several centimeters in length, the midpoint was measured in each of our cadavers. The location of the LON at the midpoint of this zone was measured on average to be 5.5 cm caudal to the OP (range, 3–7.5 cm; SD ±1.38) and 6.2 cm lateral to the PM (range, 4–8 cm; SD ±1.10). Figure 3 demonstrates the cadaveric and in vivo representations of this potential compression zone. All the identified LONs had branched in our cadaver specimens caudal to the NL (compression zone 3), with the first branch point occurring an average of 3.8 cm caudal to the OP and 5.9 cm lateral to the PM. At the NL itself, the medial-most and lateral-most branches were measured with regard to the abovementioned landmarks. The medial-most branch was located approximately 1 cm caudal to the OP and 5.35 cm lateral to the PM (range, 3–9 cm; SD ±1.10), whereas the lateral-most branch was identified 1 cm caudal to the OP and 6.5 cm lateral to the PM (range, 4–9.5 cm; SD ±1.40). Figure 4 demonstrates the cadaveric and in vivo representations of this potential compression zone. As noted by others, we found a consistent fascial band at the NL in each of our cadaver specimens and in all 40 of our patients.

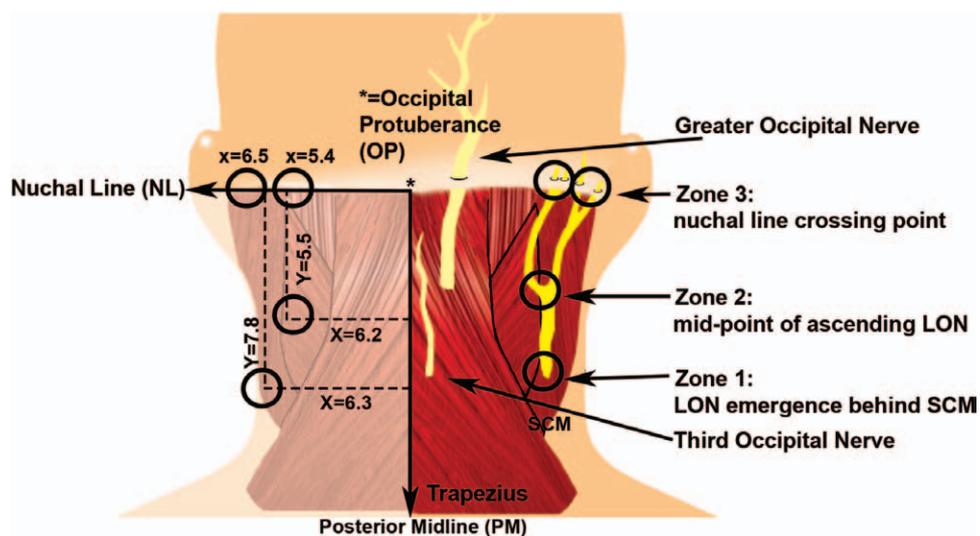


Fig. 1. A summary of the anatomic measurements of the LON from the cadaver portion of our study. Also included for reference are the general positions of the greater occipital nerve, the third occipital nerve, the trapezius muscle, and the SCM. All numbers represent measurements in centimeters.

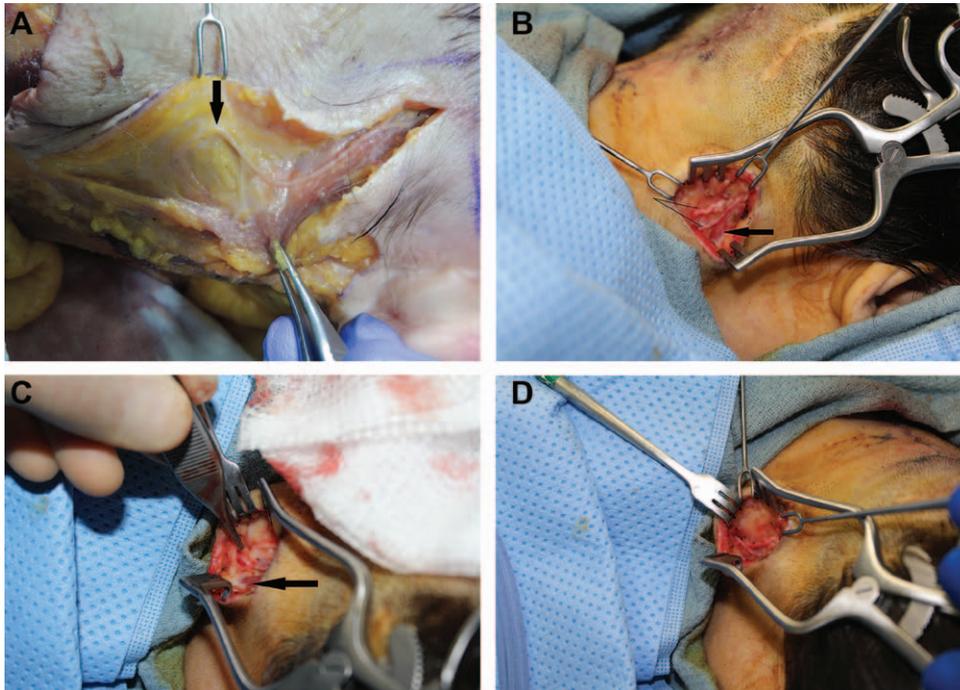


Fig. 2. A, The emergence of the LON in zone 1 from behind the SCM (arrow) in a cadaver specimen. The cadaver is lying prone with the auricular skin visible just to the right of the gloved hand. The SCM has been retracted laterally in the forceps revealing a small fascial tunnel through which the nerve emerges. B, The emergence of the LON from behind the SCM (arrow) in a patient. Note that the LON has already branched well below the nuchal line (thin black lines). C, A view from the head of the bed with the patient prone and looking caudally, thus demonstrating the emergence of the LON from behind the SCM (arrow) in the same patient as (B). The forceps point to a fascial band compressing and separating the 2 LON branches. D, Identical image to (C) in the same patient, but the compressive band/septum has been released. Note the increased amount of space surrounding the LON branches and the more visible presence of the vasa nervorum indicating increased blood flow to the released nerves.

Forty patients underwent surgical decompression/neuroplasty of the LON between 2011 and 2014. Eighteen patients had the LON addressed uni-

laterally, whereas 22 patients had the LONs treated bilaterally. Two patients were lost to follow-up and 2 patients were excluded as they were not at least

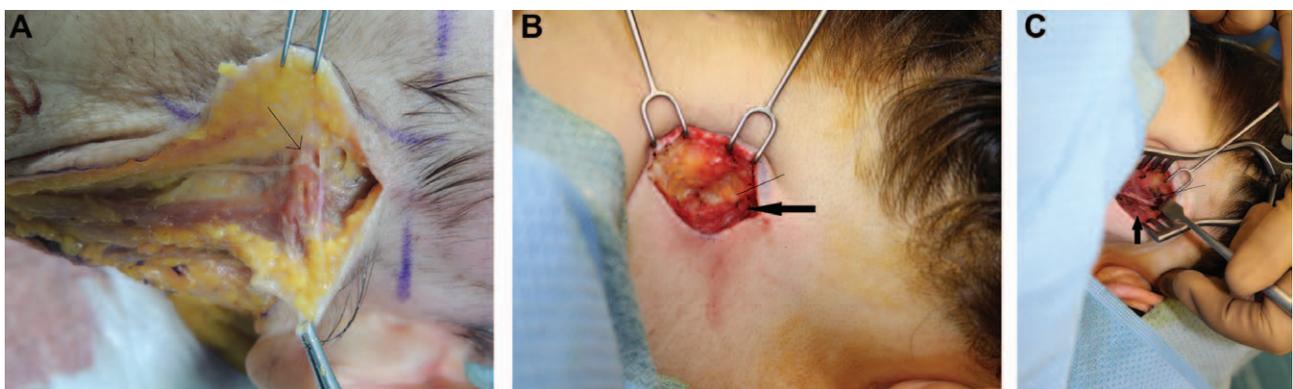


Fig. 3. A, A potential compression point within zone 2 as the LON ascends cranially toward the nuchal line in a cadaver specimen. Note the abnormal muscle insertion of the posterior SCM border (arrow) that would compress the LON as the neck is turned from side to side. B, Compression of the LON, which is barely visible under thick fascia (thin line) within zone 2. The posterior SCM border (arrow) is clearly seen. C, The same patient as (B), but now the LON (thin line) has been decompressed. The nerve is now clearly visible and note that unlike the patient in Figure 2, the LON has not yet branched. The posterior SCM border (arrow) is clearly seen retracted with the Senn, and the splenius muscle is seen deep to the nerve.



Fig. 4. A, Several potential compression points within zone 3 as the LON crosses the nuchal line in a cadaver specimen. Note the caudal LON branch point (arrow) over the blue background and additional branch points in the medial branch more cephalically (thin lines). B, Compression of the LON at the nuchal line in a patient. Note the LON (arrow) as it ascends cephalically and is compressed by a crossing fascial band (thin line). The posterior SCM border is clearly seen just deep to the skin hooks. C, Same patient as (B), but now the LON has been decompressed and is more clearly visible. Note the area of hyperemia within the nerve (thin line) corresponding to the former site of compression.

12 months following their procedure. Of the remaining 36 patients, there were 28 females and 8 males in this cohort, corresponding closely to the expected 3:1 incidence of chronic migraines in females versus males.²⁶ Average follow-up was 27.6 months. Average age at the time of the procedure was 41.3 years. Of note, 32 of these 36 patients underwent surgical treatment of one or more other nerves at the same time as the LON per the criteria noted above. Only 4 patients underwent surgical decompression and neuroplasty of the LON alone.

A successful postoperative outcome was defined as a 50% or greater reduction in the frequency, severity, and/or duration of headache symptoms at least 12 months postoperatively. The mean preoperative MHI was 145 and the mean postoperative MHI was 32 for a statistically significant improvement in overall symptomatology ($P < 0.0001$). Five patients did not obtain at least a 50% or more reduction in the parameters noted above, 4 of whom subsequently underwent resection of the involved LONs and 1 of whom is awaiting this procedure and is counted an unsuccessful outcome for the purposes of this study. Two of these neurectomy patients subsequently reported a greater than 50% reduction in overall symptomatology, whereas the other 2 reported no change from their preoperative values. All patients reported preserved LON sensation after decompression/neuroplasty, and no postoperative complications were reported.

DISCUSSION

Over the past several years, a number of studies have reported successful outcomes with surgical decompression or resection of specific occipital or trigeminal nerve branches in treating refractory headaches.^{9,10,14,19,27-33} However, in the clinical articles discussing the LON, it has been primarily re-

sected. Therefore, the aim of the current study was to demonstrate its anatomy in relation to previously established landmarks and to clinically relevant compression zones. Moreover, we sought to demonstrate that the knowledge of such anatomy can translate to successful clinical outcomes with LON preservation.

Lee et al³² recently published a study detailing several compression points for the LON although the compression sites noted were slightly different from the senior author's experience with this nerve. In that article, the authors delineated the anatomy of the LON noting the presence of an artery and its relation to the nerve, which was found in 50% of cases and a consistent fascial band at the NL. Although the anatomy of the LON has been previously described, we still find this procedure to be quite challenging. The senior author's clinical experience with decompression/neuroplasty of the LON suggests that there are additional potential compression zones. Therefore, we decided to perform an anatomic study concentrating on all potential compression points along the course of the LON in the occipital area. Moreover, we included a prospectively collected series of actual patient data on LON procedures to assess for similarities and outcomes.

The results in our cadaver specimens and in our patient cohort show that there seem to be 3 potential compression zones for the LON. Based on our cadaver measurements, the anatomy of the LON can be quite variable with respect to its location within these 3 compression zones as has been suggested by others.³⁴ That being said, compression zones 1 and 2 in this study are similar to those reported in prior work.³² However, we also found that the branching pattern of LON is highly variable, something which has not been previously noted. We therefore believe that to avoid missing any LON branches intraoperatively, the nerve needs to be dissected from zone 1 through zone 3 and measurements such as those de-

scribed here would facilitate this dissection. Unlike in the recent article by Lee et al,³² we did not find consistent vascular compression in this particular study although we do agree that there seems to be a constant fascial band present at the NL, which was seen in each of our cadavers and in all 40 of our patients. The LON was not found on 1 side of 1 cadaver head as noted. The most likely possibility for this finding is that during processing and preparation of the cadaver head, the LON was avulsed or removed based on the obliquity and level of transection.

Our patient cohort demonstrates that the knowledge of LON anatomy in the lateral neck can assist with adequate decompression/neuroplasty of said nerve and leads to successful clinical results with preservation of sensation. We report an 86% success rate with nerve preservation as only 5 of 36 patients (14%) did not achieve at least a 50% reduction in their headache symptoms. These results are comparable with those published with other nerve decompression procedures for chronic headaches.^{9,35} Of the 5 patients who did not respond to decompression, 1 is awaiting evaluation for neurectomy and implantation. All the remaining 4 patients ultimately underwent LON resection and muscle implantation in a second procedure and 2 of these eventually achieved greater than 50% reduction in their symptoms. In 1 of these responders, an additional LON branch was identified in the second procedure and could be considered to have had an incomplete release during the first procedure. In the other 3, a moderately inflamed and scarred LON was identified, dissected back to healthy nerve, and transected. It is unclear as to why 1 of these 3 patients eventually achieved a greater than 50% improvement in symptoms, whereas the other 2 did not, but one possibility is an inadequate proximal dissection leaving behind an inflamed proximal nerve end that is implanted in the local muscle. It is therefore possible to consider an even more proximal resection if additional nerve blocks are successful.

There are several limitations to this study. One is that the senior author and primary surgeon obtained both the preoperative and the postoperative data, which could have affected the patients' responses. A second limitation is the clinical sample size of 36 patients and 8 cadaver heads, which could both be larger. A third limitation and one final point to emphasize would be that the results presented herein are not to be interpreted as suggesting that neuroplasty/decompression is necessarily superior to neurectomy and implantation with respect to the LON as these 2 approaches were not directly compared against one another in a prospective fashion. Although the former approach certainly seems effec-

tive, it should be emphasized that only part of the improvement in 32 of the 36 patients can be attributed to the LON as other nerves were addressed at the same time. Indeed, isolated lesser occipital neuralgia is quite rare, and only 4 patients among the initial clinical cohort had their LONs treated alone. As noted previously, 1 of these patients was among the 2 lost to follow-up, and while she had reported complete relief at her last visit, she was shy for several months of a year after her operation and therefore was unable to be included in the data set. Two of these isolated LON patients were completely headache-free after decompression/neuroplasty and 1 ultimately required neurectomy before achieving a successful outcome. These results suggest that treatment of the LON with either method can have a clinically relevant impact on patient symptoms; however, with only 4 patients, meaningful statistical comparisons are not possible.

CONCLUSION

This study follows the LON through the occipital region/lateral neck and describes several compression zones with clinically relevant examples. We believe that LON compression is a clinically relevant component of the symptom complex in many patients experiencing occipital headaches and that the knowledge of LON neuroanatomy can improve diagnostic accuracy and clinical outcomes after surgery with nerve preservation.

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REFERENCES

1. Goldberg LD. The cost of migraine and its treatment. *Am J Manag Care*. 2005;11(2 Suppl):S62–S67.
2. Lipton RB, Silberstein SD. Episodic and chronic migraine headache: breaking down barriers to optimal treatment and prevention. *Headache* 2015;55 Suppl 2:103–122; quiz 123–106.
3. Hazard E, Munakata J, Bigal ME, et al. The burden of migraine in the United States: current and emerging perspectives on disease management and economic analysis. *Value Health*. 2009;12:55–64.
4. Hu XH, Markson LE, Lipton RB, et al. Burden of migraine in the United States: disability and economic costs. *Arch Intern Med*. 1999;159:813–818.
5. Turner-Bowker DM, Bayliss MS, Ware JE Jr, et al. Usefulness of the SF-8 Health Survey for comparing the impact of migraine and other conditions. *Qual Life Res*. 2003;12:1003–1012.
6. Aurora SK, Winner P, Freeman MC, et al. OnabotulinumtoxinA for treatment of chronic migraine: pooled analyses of the 56-week PREEMPT clinical program. *Headache*. 2011;51:1358–1373.

7. Dodick DW, Turkel CC, DeGryse RE, et al; PREEMPT Chronic Migraine Study Group. OnabotulinumtoxinA for treatment of chronic migraine: pooled results from the double-blind, randomized, placebo-controlled phases of the PREEMPT clinical program. *Headache*. 2010;50:921–936.
8. Ferrari MD, Roon KI, Lipton RB, et al. Oral triptans (serotonin 5-HT_{1B/1D} agonists) in acute migraine treatment: a meta-analysis of 53 trials. *Lancet*. 2001;358:1668–1675.
9. Guyuron B, Kriegler JS, Davis J, et al. Five-year outcome of surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2011;127:603–608.
10. Guyuron B, Reed D, Kriegler JS, et al. A placebo-controlled surgical trial of the treatment of migraine headaches. *Plast Reconstr Surg*. 2009;124:461–468.
11. Dirnberger F, Becker K. Surgical treatment of migraine headaches by corrugator muscle resection. *Plast Reconstr Surg*. 2004;114:652–657; discussion 658.
12. Guyuron B, Kriegler JS, Davis J, et al. Comprehensive surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2005;115:1–9.
13. Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2002;109:2183–2189.
14. Guyuron B, Varghai A, Michelow BJ, et al. Corrugator supercillii muscle resection and migraine headaches. *Plast Reconstr Surg*. 2000;106:429–434; discussion 435–427.
15. 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.
16. Janis JE, Hatef DA, Ducic I, et al. Anatomy of the auriculotemporal nerve: variations in its relationship to the superficial temporal artery and implications for the treatment of migraine headaches. *Plast Reconstr Surg*. 2010;125:1422–1428.
17. Mosser SW, Guyuron B, Janis JE, et al. The anatomy of the greater occipital nerve: implications for the etiology of migraine headaches. *Plast Reconstr Surg*. 2004;113:693–697; discussion 698.
18. Janis JE, Hatef DA, Ducic I, et al. The anatomy of the greater occipital nerve: part II. Compression point topography. *Plast Reconstr Surg*. 2010;126:1563–1572.
19. Ducic I, Moriarty M, Al-Attar A. Anatomical variations of the occipital nerves: implications for the treatment of chronic headaches. *Plast Reconstr Surg*. 2009;123:859–863; discussion 864.
20. Lee M, Lineberry K, Reed D, et al. The role of the third occipital nerve in surgical treatment of occipital migraine headaches. *J Plast Reconstr Aesthet Surg*. 2013;66:1335–1339.
21. Patrick J, Frank W, Theodora M, et al. The pedicled serratus anterior muscle wrap-around flap: a treatment option in the management of posttraumatic axillary neuroma and neuropathic pain. *Ann Plast Surg*. 2010;65:170–173.
22. Wolfort SF, Dellon AL. Treatment of recurrent neuroma of the interdigital nerve by implantation of the proximal nerve into muscle in the arch of the foot. *J Foot Ankle Surg*. 2001;40:404–410.
23. Dellon AL, Mackinnon SE. Treatment of the painful neuroma by neuroma resection and muscle implantation. *Plast Reconstr Surg*. 1986;77:427–438.
24. Guyuron B, Tucker T, Kriegler J. Botulinum toxin A and migraine surgery. *Plast Reconstr Surg*. 2003;112(5 Suppl):171S–173S; discussion 174S.
25. Kung TA, Guyuron B, Cederna PS. Migraine surgery: a plastic surgery solution for refractory migraine headache. *Plast Reconstr Surg*. 2011;127:181–189.
26. Lipton RB, Bigal ME, Diamond M, et al.; AMPP Advisory Group. Migraine prevalence, disease burden, and the need for preventive therapy. *Neurology*. 2007;68:343–349.
27. 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.
28. Janis JE, Ghavami A, Lemmon JA, et al. The anatomy of the corrugator supercillii muscle: part II. Supraorbital nerve branching patterns. *Plast Reconstr Surg*. 2008;121:233–240.
29. Janis JE, Hatef DA, Reece EM, et al. Neurovascular compression of the greater occipital nerve: implications for migraine headaches. *Plast Reconstr Surg*. 2010;126:1996–2001.
30. Janis JE, Hatef DA, Thakar H, et al. The zygomaticotemporal branch of the trigeminal nerve: Part II. Anatomical variations. *Plast Reconstr Surg*. 2010;126:435–442.
31. Kung TA, Pannucci CJ, Chamberlain JL, et al. Migraine surgery practice patterns and attitudes. *Plast Reconstr Surg*. 2012;129:623–628.
32. Lee M, Brown M, Chepla K, et al. An anatomical study of the lesser occipital nerve and its potential compression points: implications for surgical treatment of migraine headaches. *Plast Reconstr Surg*. 2013;132:1551–1556.
33. Poggi JT, Grizzell BE, Helmer SD. Confirmation of surgical decompression to relieve migraine headaches. *Plast Reconstr Surg*. 2008;122:115–122; discussion 123.
34. Dash KS, Janis JE, Guyuron B. The lesser and third occipital nerves and migraine headaches. *Plast Reconstr Surg*. 2005;115:1752–1758; discussion 1759.
35. Ducic I, Hartmann EC, Larson EE. Indications and outcomes for surgical treatment of patients with chronic migraine headaches caused by occipital neuralgia. *Plast Reconstr Surg*. 2009;123:1453–1461.