The Anatomy of the Greater Occipital Nerve: Part II. Compression Point Topography

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Background: Advances in the understanding of migraine trigger points have pointed to entrapment of peripheral nerves in the head and neck as a cause of this debilitating condition. An anatomical study was undertaken to develop a greater understanding of the potential entrapment sites along the course of this nerve.
Methods: The posterior neck and scalp of 25 fresh cadaveric heads were dissected. The greater occipital nerve was identified within the subcutaneous tissue above the trapezius and traced both proximal and distal. Its fascial, muscular, and vascular investments were located and accurately measured relative to established bony landmarks.
Results: Dissection of the greater occipital nerve revealed six major compression points along its course. The deepest (most proximal) point was between the semispinalis and the obliquus capitis inferior, near the spinous process. The second point was at its entrance into the semispinalis. The previously described "intermediate" point was at the nerve's exit from the semispinalis. A fourth point was located at the entrance of the nerve into the trapezius muscle. The fifth point of compression is where the nerve exits the trapezius fascia insertion into the nuchal line. The occipital artery often crosses the nerve, and this frequently occurs in this distal region of the trapezius fascia, which is the final point.
Conclusions: There are six compression points along the greater occipital nerve. These can be located using the data from this study, serving as a guide for surgeons interested in treating patients with migraine headaches originating in these areas. Long-term relief from migraine headaches has been demonstrated clinically by using both noninvasive and surgical decompression of these points. (*Plast. Reconstr. Surg.* 126: 1563, 2010.)

Migraine headache is a debilitating condition that affect many patients—a recent meta-analysis demonstrated that the 1-year prevalence of migraine headaches in European patients is between 14 and 35 percent in women and between 6 and 15 percent in men; also, it was seen that an estimated 12 to 28 percent of all people suffer from migraines at least once in their lifetime.[1] Migraine attacks can be of such severity that all activity is prevented.[2] Patients' lives are negatively impacted not only by the pain of acute attacks but also between episodes, as there is great anxiety over expected agony.[3,4]

A multitude of treatments have been explored, primarily focusing on different pharmacologic agents. Recently, more interventional methods have been shown to be efficacious in the amelioration of migraine symptoms. In 1998, the first report of the use of botulinum toxin type A for

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the treatment of tension headaches appeared in the headache literature.[5] Neurologists began to explore the use of botulinum toxin in patients with migraine headaches, with some success.[6,7]

At around the same time, it was demonstrated that corrugator resection during aesthetic rejuvenation of the brow led to an improvement in migraine symptoms in almost 80 percent of patients with a history of migraines.[8] In a prospective follow-up study, over 55 percent of migraine patients experienced complete resolution following corrugator removal, with partial improvement in another 28 percent. This exciting finding led to the notion of peripheral nerve compression as a potential cause of migraine headaches. Entrapment of the supraorbital nerve by the corrugator muscles was felt to be a possible trigger of migraine headaches originating in this region[9]; other sites were examined, and it was found that there are at least four separate peripheral trigger sites: frontal,[8–11] temporal,[11,12] occipital,[11,13,14] and nasal.[11] Clinical experience with open and endoscopic surgical release of nerves in these regions continues to demonstrate benefit for migraine patients. Because of the cost and the side effects of prophylactic and abortive migraine medications, a potentially permanent surgical treatment is clearly an advantageous alternative.

In the occipital region, 62 percent of occipital trigger region patients were demonstrated to have total relief of migraine symptoms after open release of greater occipital nerve entrapment at its entry into the semispinalis. Although this is an encouraging result bringing benefit to this large group of patients, it still leaves over one-third of patients in whom this was inadequate.[14] The three senior authors (J.E.J., I.D., and B.G.) have noticed other entrapment sites of the greater occipital nerve during open surgical release, and it was felt that there could be more sites of compression along the course of the nerve. To more fully elucidate these anatomical details, a cadaveric study was undertaken.

METHODS

Twenty-five fresh cadaveric heads were obtained from the Willed Body Program at the University of Texas Southwestern Medical Center in Dallas, Texas. Specimens used were between the ages of 42 and 86, and all bodies were tested for human immunodeficiency virus and other communicable diseases before commencement of dissection.

The heads were placed in the prone position in a Mayfield neurosurgical headrest and shaved. The heads were marked with an indelible surgical marker along a vertical line through the midline and a horizontal line through the occipital protuberance, similar to the technique demonstrated in the original Part I study on the greater occipital nerve reported in 2004.[13] A methylene blue–tipped 16-gauge needle was passed through the skin to mark the subcutaneous tissue along these lines and at the occipital protuberance to allow accurate measurements within the deeper layers. Using these lines as incision markings, a no. 10 blade was used to cut down through the skin and subcutaneous tissue. Flaps were raised at this level, exposing the galea and trapezius. The greater occipital nerve was located and its surrounding fascial inscriptions were found, measured, photographed, and noted. Bilateral trapezius flaps were then elevated from the midline to expose the greater occipital nerve and the deeper splenius and semispinalis muscles. Great care was taken not to disrupt any portion of the greater occipital nerve or its surrounding musculofascial or vascular interfaces. When the originally described point of compression was located as the nerve emerged from the semispinalis, the nerve was followed down through this muscle, through the splenius, and through the semispinalis collis muscles to its exit from deep to the obliquus capitis inferior muscle. It was also followed distally to the nuchal line. All points of

Table 1. Anatomical Locations of the Compression Points of the Greater Occipital Nerve as It Courses from Deep to Superficial in the Occipital Region

Compression Point	Description	Lateral Distance from the Midline (mm)	Distance from the Horizontal Line through the Occipital Protuberance (mm)
1	Through the fascial band between the obliquus capitis and the semispinalis	20.13	77.38
2	Entry into the semispinalis	17.46	59.71
3	Exit from the semispinalis	15.52	34.52
4	Entrance into the trapezius	24	21
5	Through the trapezius insertion	37.07	4.36
6	Occipital artery	30.27 (variable)	10.67 (variable)

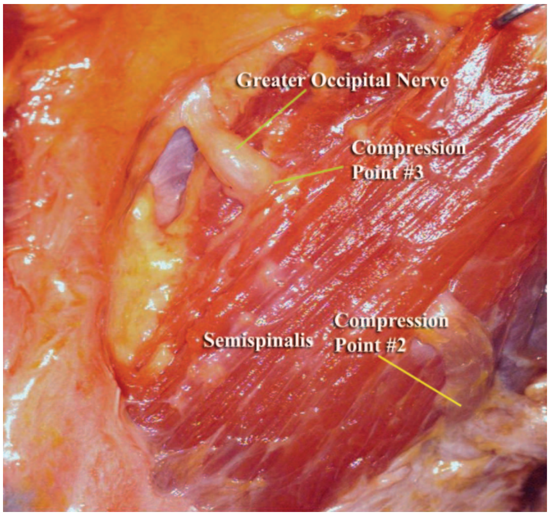


Fig. 1. Compression point 3, the originally described compression point where the greater occipital nerve exits the semispinalis muscle. Compression point 2, where the nerve is moving from deep to superficial, entering the semispinalis and/or the fascia surrounding the muscle, is also demonstrated.

Video 1. Supplemental Digital Content 1 demonstrates open decompression of the greater occipital nerve, ***http://links.lww.com/PRS/A208***. In this video, the surgeon is decompressing point 3 by using a right angle to release the nerve as it runs through the semispinalis by resecting a medial rectangular portion of the muscle. A lateral wedge of semispinalis muscle will also be released as part of the decompression of this point to fully release the nerve from the surrounding musculature.

compression of the nerve were found, measured, photographed, and noted. All data were entered into a Microsoft Excel database (Microsoft, Inc., Redmond, Wash.), and means were calculated for the distances from the horizontal line through the protuberance and from the midline (Table 1).

RESULTS

Fifty cranial halves (25 cadaveric heads) were dissected in total. Seventeen heads were from male donors and eight were from female donors. The mean age of the cadaveric donors was 61 years. The greater occipital nerve was found in all specimens.

Compression Points

The first point to note is the point that has been described previously,[13] where the nerve

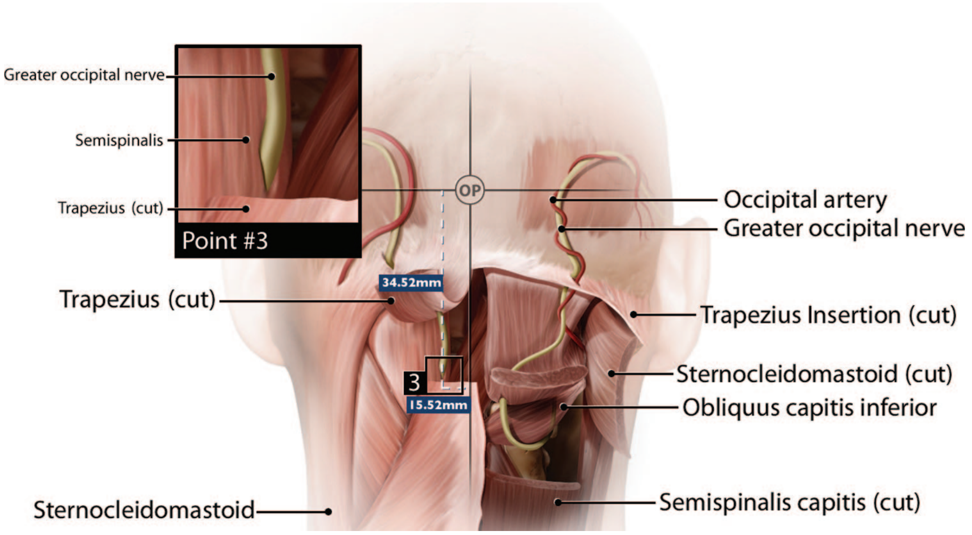


Fig. 2. Schematic illustration depicting compression point 3, where the greater occipital nerve exits from the semispinalis muscle.

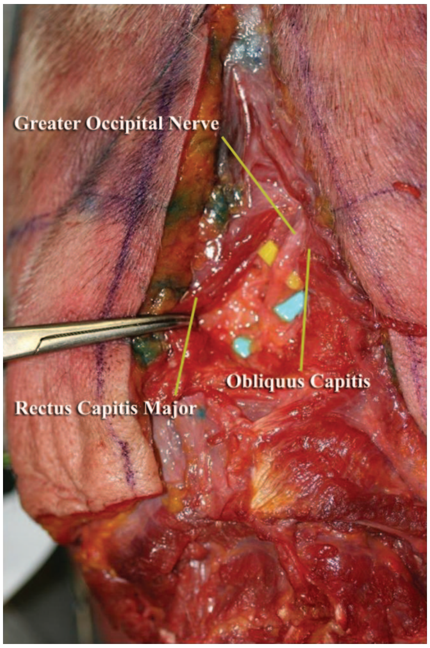
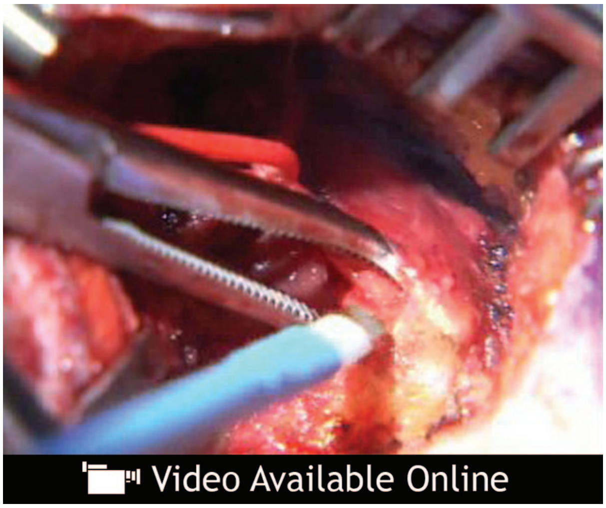


Fig. 3. Compression point 1, where the greater occipital nerve exits from the deep fascia to wrap around the obliquus capitis.



Video 2. Supplemental Digital Content 2 demonstrates open decompression of the greater occipital nerve, ***http://links.lww.com/PRS/A209***. The surgeon is dissecting deep to the semispinalis, opening up the fascia between this muscle and the obliquus capitis inferior, which is point 1. As the portion of semispinalis overlying the nerve is also subsequently excised, these maneuvers serve to decompress points 1 through 3.

emerges from the semispinalis capitis at approximately 30 mm inferior to the occipital protuberance and 15 mm lateral to the midline (point 3) (Figs. 1 and 2) (**see Video, Supplemental Digital Content 1,** which demonstrates open decompression of the greater occipital nerve, ***http://links.lww.com/PRS/A208***). This is labeled the "intermediate" point, as we have now found points both proximal and distal to this, and from our current work it is located at an average of 34.52 mm inferior to the line drawn through the protuberance and 15.52 mm lateral to the midline. There are five other potential sites that have been elucidated in this study, two of which are deeper than this and three that are more superficial. The deepest (most proximal along the course of the greater occipital nerve) potential compression point exists within a tight fascia that exists surrounding the belly of the obliquus capitis inferior muscle near the spinous process: this is located at an average of 77.38 mm inferior

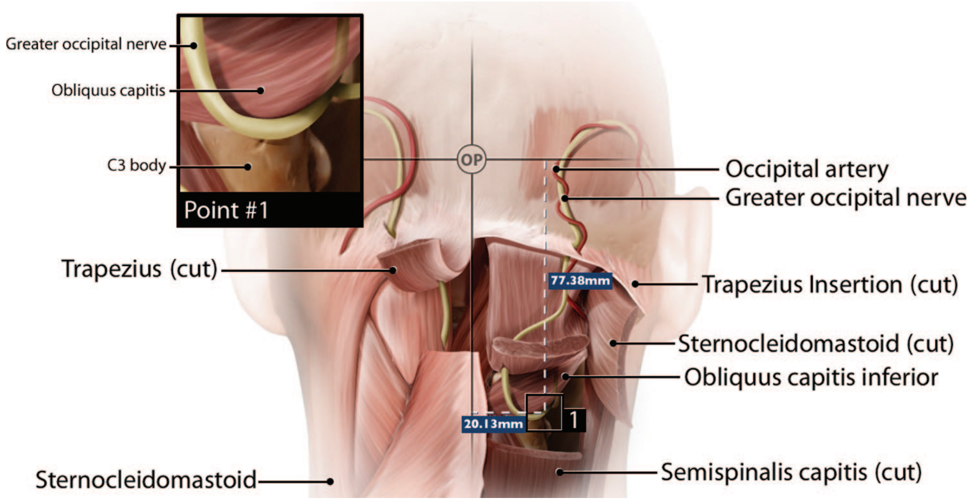


Fig. 4. Schematic illustration depicting compression point 1, where the nerve exits from deep to the obliquus capitis, wrapping around as it moves cranially and superficially.

to the occipital protuberance and 20.13 mm from the midline (Figs. 3 and 4) (**see Video, Supplemental Digital Content 2,** which demonstrates open decompression of the greater occipital nerve, ***http://links.lww.com/PRS/A209***). This is point 1. More cranial and superficial to this deepest point exists point 2, an area where the nerve can be impinged at its entrance into the deep fascia underlying the semispinalis, or the muscle itself, which is located approximately at 59.71 mm inferior to the line through the protuberance and 17.46 mm from the midline (Fig. 5). Finally, there are three more cranial and more superficial locations (relative to the originally described point 3) where the nerve can be impinged. Point 4 is at its entrance into trapezius, which is located at 21 mm inferior to the horizontal line through the occipital protuberance and 24 mm from the midline (Figs. 6 and 7). The nerve then travels farther cranial and lateral overlying the trapezius, and then pierces the tendinous insertion of the trapezius into the nuchal line, which is point 5, the most distal musculofascial compression point. This is located at an average of 4.36 mm from the line drawn through the protuberance and 37.07 mm from the midline (Figs. 8 and 9) (**see Video, Supplemental Digital Content 3,** which demonstrates the operator releasing the nerve as it runs through a fascial band within the trapezial tunnel, point 5, ***http://links.lww.com/PRS/A210***).

The greater occipital nerve was also seen to run across the occipital artery in many specimens, and the two often intertwined (Figs. 10 and 11) (**see Video, Supplemental Digital Content 4,** which demonstrates, the occipital artery being coagulated using bipolar cautery, in an effort to stop flow through the artery at the point at which it could be aggravating the occipital nerve; this is point 6, ***http://links.lww.com/PRS/A211***). This constitutes the sixth point of potential compression, although it is vascular rather than musculofascial. It is located variably. Sometimes, it is located deep to the trapezius, caudal to the horizontal line through the occipital protuberance; in other specimens, it has been noted distal and cranial to this line, approaching the ear. The detailed results of the investigation into the relationship between these structures will be reported separately.

DISCUSSION

The greater occipital nerve is the medial branch of the second cervical dorsal ramus. It has a well-described course from its origin as it ascends between the obliquus capitis inferior and the semispinalis through the extensor muscles of the cervical region along through the superficial subcutaneous areolar tissue, which lies above the occipital galea. This is the first body of work that focuses on the potential points of compression along the path of this nerve. Six points of compression were found in this study. There were five nonvascular points where muscle or fascia potentially entrap the greater occipital nerve; and there was one vascular point, where the occipital artery can cross, compress, or intertwine the nerve. The deepest point (point 1) was just past the origin of

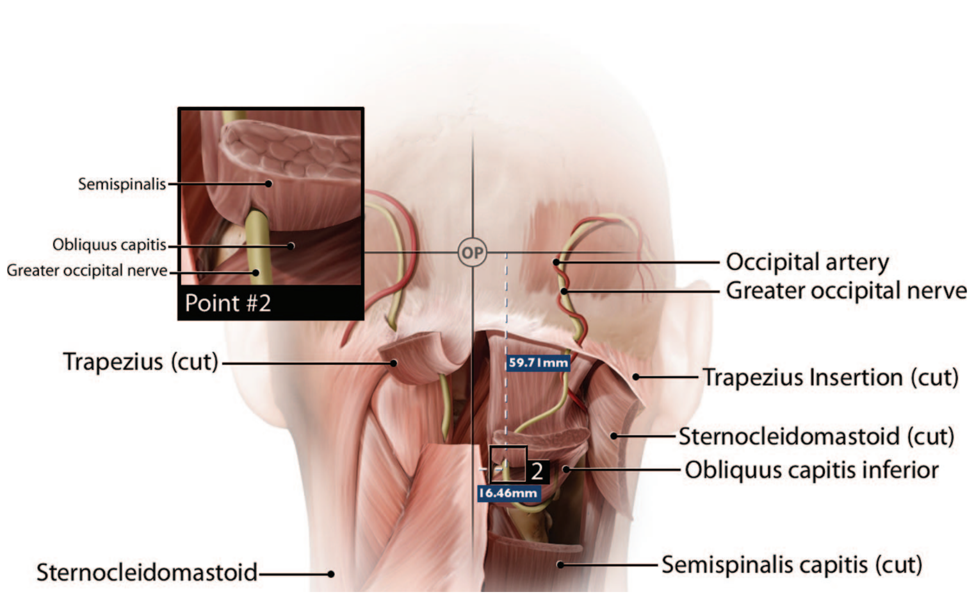


Fig. 5. Schematic illustration depicting compression point 2, the entrance of the nerve into the semispinalis muscle.

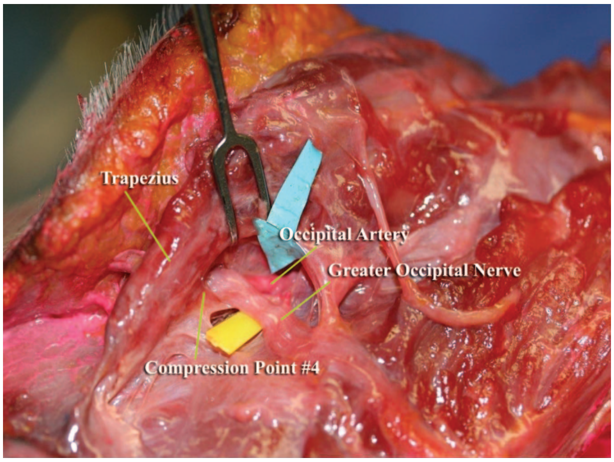


Fig. 6. Compression point 4, where the nerve enters the substance of the trapezius muscle.

the nerve as it passes through a tight fascial band sandwiched between the obliquus capitis inferior and the semispinalis. As the nerve enters the semispinalis muscle, it goes through another point of tight inscription (point 2). A tight point of compression is also noted as the nerve exits the semispinalis, a point that has been noted previously (point 3).[13] As the nerve ascends into the trapezius muscle, it encounters another area of entrapment on entering this muscle (point 4). A fifth point was found more superficial, cranial, and lateral, as the nerve traverses the trapezius insertion at the nuchal line—the roof of the so-called trapezial tunnel. This has been noted as a region rich in lymphatic channels; also, the occipital artery often crosses the nerve in this region, representing the sixth point of potential compression. This neurovascular relationship is seen in variable positions. Sometimes, it is between points 4 and 5. It was noted deep to the trapezius muscle but more often overlying the trapezius muscle; it is also sometimes seen in the subcutaneous tissues of the occiput, just distal to point 5, within the trapezial tunnel.[15,16] When there was a helical intertwining of the artery and nerve, this relationship could extend nearly to the ear.

The findings in this study correlate well with recent reports of the greater occipital nerve anatomy. The first part of this study, published in 2004,[13] reported that the nerve emergence from the semispinalis was at approximately 1.5 cm lateral to the midline and 3 cm inferior to the occipital protuberance. A recent report from the group of one of the senior authors (I.D.) on the clinically relevant intraoperative anatomy of this nerve demonstrated the same results: that point 3 is 14.9 mm lateral to the midline and 30.2 mm inferior to the protuberance.[15] In that report, the authors also noted intraoperative findings of the greater occipital nerve: in patients with increased symptoms of chronic headache, the nerve lacked a fascicular pattern, capillary flow, and seemed to have a yellow-brown appearance.

Entrapment of a peripheral nerve causing a regional pain syndrome is not a new concept. Greater occipital neuralgia is a well-described but little known syndrome that is even discussed as a clinical corollary in *Gray's Anatomy*.[17] This is a

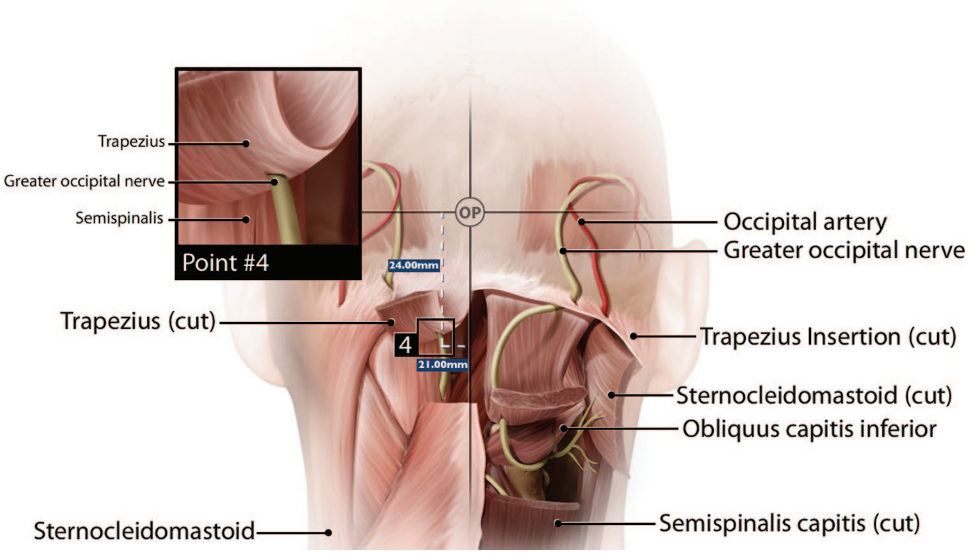


Fig. 7. Schematic illustration depicting compression point 4, where the nerve enters the trapezius.

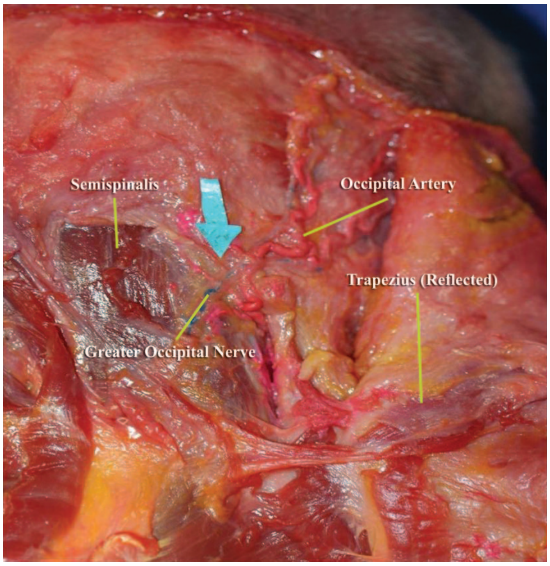


Fig. 8. Compression point 5, where the nerve travels through the trapezius insertion.

Video 3. Supplemental Digital Content 3 shows the operator releasing the nerve as it runs through a fascial band within the trapezial tunnel (point 5), ***http://links.lww.com/PRS/A210***. As the surgeon releases the fascial band of the insertion of the trapezius into the nuchal line, a greater occipital nerve/occipital artery intersection is found.

chronic headache that occurs within the cutaneous sensory distribution of the greater occipital nerve, and is often misdiagnosed as migraine headache.[18–20] Its underlying cause is a neuritis secondary to entrapment within the neck muscles and/or the atlantoepistrophic ligament. Local block of the greater occipital nerve is both diagnostic of and a treatment for this syndrome.[21] It must be noted, though, that occipital neuralgia is a separate disorder from migraines secondary to an occipital trigger. The former is a continuous chronic headache, whereas the latter is episodic.

Furthermore, evidence of morbidity from peripheral nerve compression may be gleaned from the literature on compartment syndrome. In this highly morbid scenario, the tissue pressure falls below perfusion pressure, and major nerves within the involved fascial compartment are tightly compressed and deprived of vital perfusion, albeit in an acute setting.[22,23] Acute intracompartmental pressures of 30 mmHg are thought to be elevated

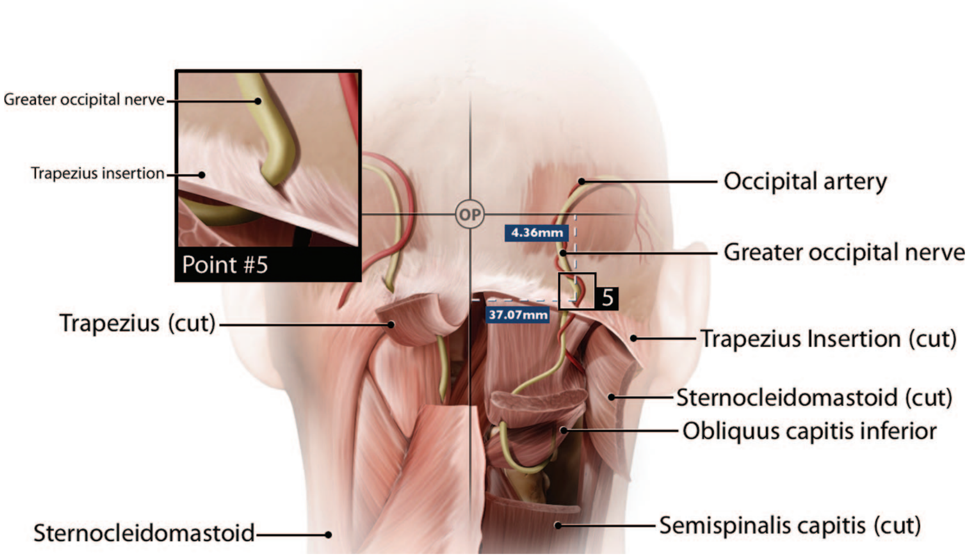


Fig. 9. Schematic illustration depicting compression point 5, where the nerve enters the trapezius insertion.

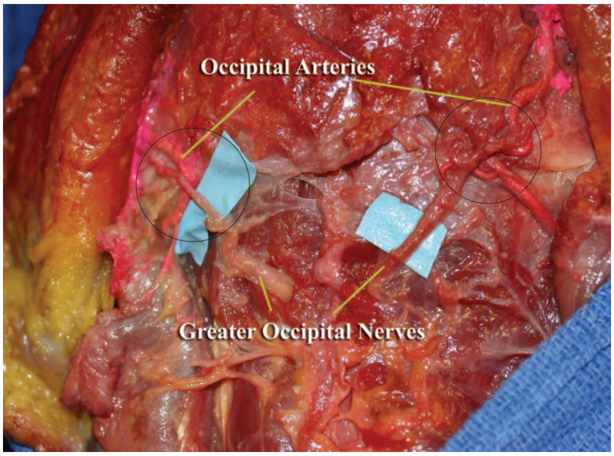


Fig. 10. Compression point 6, showing "single cross"–type relationships between the greater occipital nerve and the occipital artery bilaterally.

Video 4. Supplemental Digital Content 4 shows the occipital artery being coagulated using bipolar cautery, in an effort to stop flow through the artery at the point at which it could be aggravating the occipital nerve; this is point 6, ***http://links.lww.com/PRS/A211***.

to the point where the blood supply to the structures within the compartment is put in jeopardy.[24] Basic science studies have shown that mechanical loading on peripheral nerves leads to adverse effects in neuronal protein synthesis and transport at pressures as low as 20 mmHg.[25]

Yet another example of peripheral nerve compression is carpal tunnel syndrome. The current understanding of carpal tunnel syndrome is that the median nerve is chronically inflamed secondary to anoxic injury secondary to compression within the carpal tunnel.[26] Symptoms of chronic pain, weakness, and paresthesia are exacerbated as intraneural and extraneural pressure rise in a cycle of pressure, irritation, and inflammation.[27] Surgical release of the carpal ligament allows the pressure surrounding the nerve to be alleviated, correcting the situation.[28]

Further extending the current clinical logic with the field of peripheral nerve surgery, it is

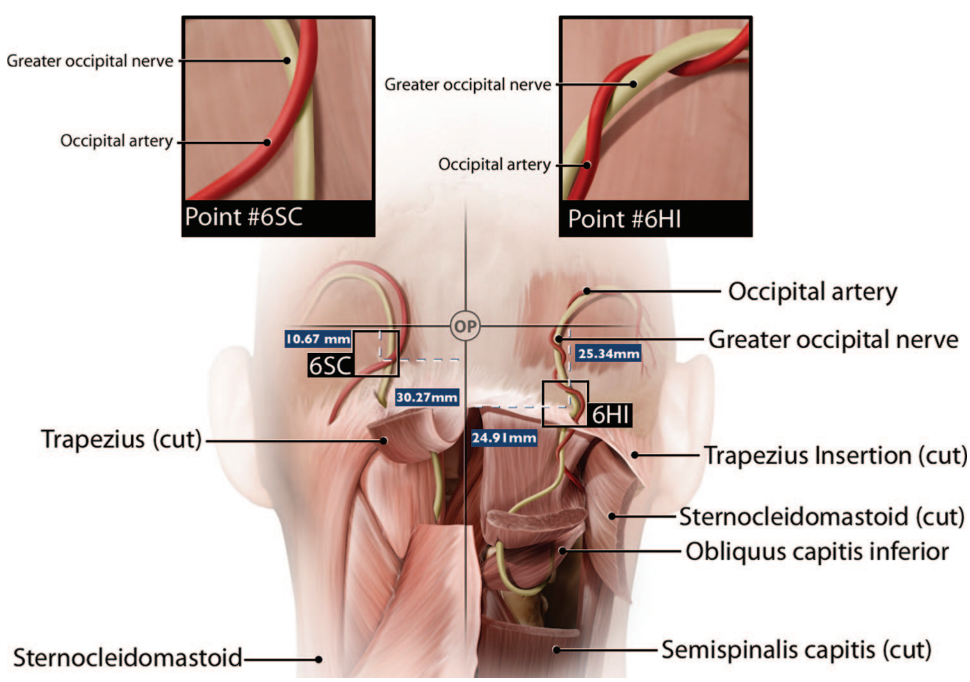


Fig. 11. Schematic illustration depicting compression point 6; different types of greater occipital nerve–occipital artery relationships are shown. *SC*, single cross; *HI*, helical intertwining.

noted that *multiple* sites of compression along a nerve is also not a new concept. The three major nerves of the upper extremity all have multiple sites where they can be compressed, and appropriate treatment of patients suffering from compression neuropathies often involves addressing more than just one site.[29,30] The ulnar nerve and the radial nerve both have six potential sites of compression; the median nerve has five.[29–36] That there would be multiple sites of compression along the path of another peripheral nerve that arises from branches of the spinal rami is intellectually consistent. Just as effective treatment of upper extremity compression syndromes often requires the surgeon to address more than one site, the same may be true for chronic migraines that have their cause in greater occipital nerve compression.

Clinically, the senior authors (J.E.J., I.D., and B.G.) have performed more extensive release of the greater occipital nerve for patients in whom liberation of this nerve is indicated. More extensive decompression of this nerve across multiple potential sources of entrapment has led to an average of almost 99 percent improvement in reported headache symptoms and an average of 81.5 migraine headache–free days immediately postoperatively.[37] These lateral points of compression can be accessed through either the vertical midline incision, used by two of the senior authors (J.E.J. and B.G.), or through bilateral transverse incisions well within the hairline, used by another of the senior authors (I.D.).

Migraine is a chronic disorder that primarily affects adults during the prime earning years of their life.[38] Secondarily, there is a large indirect cost associated with migraines attributable to the decreased productivity resulting from the debilitating nature of the attacks.[39] A permanent surgical solution to chronic migraine headaches would obviously be a welcome addition to the treatment of migraine headaches. Because of recent advances in the understanding of the anatomy of the peripheral nerves in the head and neck, and innovative clinical applications of this understanding, surgical release of nerve entrapment sites has become a clinically efficacious reality. This study aids in the surgeon's understanding of the anatomy of the greater occipital nerve, and more extensive release of this nerve is currently being performed. Furthermore, prospective clinical studies are indicated to assess the efficacy of more extensive releases of this nerve, which are currently underway at three different academic institutions (University of Texas Southwestern Medical Center, Georgetown University Medical Center, and Case Western Reserve University School of Medicine).

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REFERENCES

1. Stovner LJ, Zwart JA, Hagen K, Terwindt GM, Pascual J. Epidemiology of headache in Europe. *Eur J Neurol.* 2006;13:333–345.
2. Cady R, Dodick DW. Diagnosis and treatment of migraine (Reply). *Mayo Clin Proc.* 2002;77:876–877.
3. Dahlöf CG, Dimenäs E. Migraine patients experience poorer subjective well-being/quality of life even between attacks. *Cephalalgia* 1995;15:31–36.
4. Freitag FG. The cycle of migraine: Patients' quality of life during and between migraine attacks. *Clin Ther.* 2007;29:939–949.
5. Wheeler AH. Botulinum toxin A, adjunctive therapy for refractory headaches associated with pericranial muscle tension. *Headache* 1998;38:468–471.
6. Silberstein S, Mathew N, Saper J, Jenkins S. Botulinum toxin type A as a migraine preventive treatment. *Headache* 2000;40:445–450.
7. Göbel H, Heinze A, Heinze-Kuhn K, Jost WH. Evidence-based medicine: Botulinum toxin A in migraine and tension-type headache. *J Neurol.* 2001;248(Suppl 1):34–38.
8. Guyuron B, Varghai A, Michelow BJ, Thomas T, Davis J. Corrugator supercilii muscle resection and migraine headaches. *Plast Reconstr Surg.* 2000;106:429–434; discussion 435–437.
9. Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. *Plast Reconstr Surg.* 2002;109:2183–2189.
10. Behmand RA, Tucker T, Guyuron B. Single-site botulinum toxin type A injection for elimination of migraine trigger points. *Headache* 2003;43:1085–1089.
11. Guyuron B, Kriegler JS, Davis J, Amini SB. Comprehensive surgical treatment of surgical migraines. *Plast Reconstr Surg.* 2005;115:1–9.
12. Totonchi A, Pashmini N, Guyuron B. The zygomaticotemporal branch of the trigeminal nerve: An anatomical study. *Plast Reconstr Surg.* 2005;115:273–277.
13. Mosser SW, Guyuron B, Janis JE, Rohrich RJ. The anatomy of the greater occipital nerve: Implications for the etiology of migraine headaches. *Plast Reconstr Surg.* 2004;113:693–697; discussion 698–700.
14. Dash KS, Janis JE, Guyuron B. The lesser and third occipital nerves and migraine headaches. *Plast Reconstr Surg.* 2005;115:1752–1758; discussion 1759–1760.
15. 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.
16. 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.
17. Standring S, Ellis H, Healy JC, Johnson D, Williams A, eds. *Gray's Anatomy.* 39th ed. London: Churchill Livingstone; 2005:783.
18. Anthony M. Headache and the greater occipital nerve. *Clin Neurol Neurosurg.* 1992;94:297–301.

19. Chouret EE. The greater occipital neuralgia headache. *Headache* 1967;7:33–34.
20. Knox DL, Mustonen E. Greater occipital neuralgia: An ocular pain syndrome with multiple etiologies. *Trans Sect Ophthalmol Am Acad Ophthalmol Otolaryngol.* 1975;79:OP513–OP519.
21. Ward JB. Greater occipital nerve block. *Semin Neurol.* 2003;23:59–62.
22. Fasten FA III, Winquist RA, Krugmire RB Jr. Diagnosis and management of compartmental syndromes. *J Bone Joint Surg (Am.)* 1980;62:286–291.
23. An G, West MA. Abdominal compartment syndrome: A concise clinical review. *Crit Care Med.* 2008;36:1304–1310.
24. McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures: The pressure threshold for decompression. *J Bone Joint Surg (Br.)* 1996;78:99–104.
25. Rempel D, Dahlin L, Lundborg G. Pathophysiology of nerve compression syndromes: Response of peripheral nerves to loading. *J Bone Joint Surg (Am.)* 1999;81:1600–1610.
26. Hayes CW. Compression neuropathies in the upper limb. In: Aston SJ, Beasley RW, Thorne CHM, eds. *Grabb and Smith's Plastic Surgery.* 5th ed. Philadelphia: Lippincott-Raven; 1997:917.
27. Lundborg G, Dahlin LB. The pathophysiology of nerve compression. *Hand Clin.* 1992;8:215–227.
28. Hankins CL, Brown MG, Lopez RA, Lee AK, Dang J, Harper RD. A 12-year experience using the Brown two-portal endoscopic procedure of transverse carpal ligament release in 14,722 patients: Defining a new paradigm in the treatment of carpal tunnel syndrome. *Plast Reconstr Surg.* 2007;120:1911–1921.
29. Eversmann WW Jr. Entrapment and compression neuropathies. In: Green DP, Hotchkiss RN, Pederson WC, eds. *Green's Operative Hand Surgery.* 4th ed. Philadelphia: Churchill Livingstone; 1998:1404.
30. Eversmann WW Jr. Compression and entrapment neuropathies of the upper extremity. *J Hand Surg (Am.)* 1983;8:759–766.
31. Posner MA. Compressive ulnar neuropathies at the elbow: I. Etiology and diagnosis. *J Am Acad Orthop Surg.* 1998;6:282–288.
32. De Jesus R, Dellon AL. Historic origin of the "Arcade of Struthers". *J Hand Surg (Am.)* 2003;28:528–531.
33. al-Qattan MM. Anatomical classification of sites of compression of the palmar cutaneous branch of the median nerve. *J Hand Surg (Br.)* 1997;22:48–49.
34. Wertsch JJ, Melvin J. Median nerve anatomy and entrapment syndromes: A review. *Arch Phys Med Rehabil.* 1982;63:623–627.
35. Konjengbam M, Elangbam J. Radial nerve in the radial tunnel: Anatomic sites of entrapment neuropathy. *Clin Anat.* 2004;17:21–25.
36. Kleinert JM, Mehta S. Radial nerve entrapment. *Orthop Clin North Am.* 1996;27:305–315.
37. Janis JE. Unpublished data.
38. Lipton RB, Stewart WF, Von Korff M. The burden of migraine: A review of cost to society. *Pharmacoeconomics* 1994;6:215–221.
39. Solomon GD, Price KL. Burden of migraine: A review of its socioeconomic impact. *Pharmacoeconomics* 1997;11:1–10.