

Neurovascular Compression of the Greater Occipital Nerve: Implications for Migraine Headaches

Jeffrey E. Janis, M.D.
Daniel A. Hatef, M.D.
Edward M. Reece, M.D.,
M.S.
Paul D. McCluskey, M.D.
Timothy A. Schaub, M.D.
Bahman Guyuron, M.D.

*Dallas and Houston, Texas; and
Cleveland, Ohio*

Background: Surgical release of the greater occipital nerve has been demonstrated to be clinically effective in eliminating or reducing chronic migraine symptoms. However, migraine symptoms in some patients continue after this procedure. It was theorized that a different relationship between the greater occipital nerve and occipital artery may exist in these patients that may be contributing to these outcomes. A cadaveric investigation was performed in an effort to further delineate the occipital artery–greater occipital nerve relationship.

Methods: Fifty sides of 25 fresh cadaveric posterior necks and scalps were dissected. The greater occipital nerve was identified within the subcutaneous tissue and its relationship with the occipital artery was delineated. A topographic map of the intersection of the two structures was created.

Results: The greater occipital nerve and occipital artery have an intimate relationship, and crossed each other in 27 hemiheads (54.0 percent). The relationship between these structures when they crossed varied from a single intersection to a helical intertwining.

Conclusions: The greater occipital nerve and occipital artery have an anatomical intersection 54 percent of the time. There are two morphologic types of relationships between the structures: a single intersection point and a helical intertwining. Vascular pulsation may cause irritation of the nerve and is a possible explanation for migraine headaches that have the occipital region as a trigger point. Future imaging studies and clinical investigation is necessary to further examine the link between anatomy and clinical presentation. (*Plast. Reconstr. Surg.* 126: 1996, 2010.)

Recent clinical and anatomical investigation has expounded on the concept of peripherally triggered migraine headaches caused by entrapment, compression, or irritation of the sensory nerves of the head and neck. One of the regions that has been focused on is the occipital region,^{1,2} where muscular and fascial entrapments of the greater, lesser, and third occipital nerves have been identified and investigated.^{1,2} Recent work demonstrates that the greater occipital nerve has multiple sites of potential compression along

its path, much like the nerves of the upper extremity, which have been well characterized.³

Clinically, it has been demonstrated that injection of botulinum toxin type A into the investing musculature surrounding these nerves can provide relief from migraines in some patients and is used as a diagnostic tool to find out which patients might benefit from endoscopic or open surgical release.⁴ Surgical release of these nerves produces a significant benefit in many patients. Release of the greater occipital nerve has been demonstrated to give total relief of chronic migraine symptoms in 62 percent of patients who undergo surgery.⁵ A double-blind placebo study comparing “sham surgery” to surgical decom-

From the Department of Plastic Surgery of University of Texas Southwestern Medical Center, Baylor College of Medicine, and the Department of Plastic Surgery, Case Western Reserve University School of Medicine.

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pression showed that the former had complete migraine relief in only 3 percent of patients, compared with 57 percent of patients in the group that underwent actual decompression.⁶ In an effort to improve on the clinical efficacy of this procedure, more thorough and complete releases of the sensory nerves in this region have been investigated and undertaken, yet this still leaves a small number of patients who do not benefit from this treatment.⁷

Many migraine patients describe their symptoms as being pulsatile in nature.^{8,9} It is accepted that there may be a vascular component to many headaches, and one of the major theories of migraine proposes that extracranial arterial dilation is responsible for migraine headache symptoms.^{10–12} Widely used pharmacologic treatments, such as the serotonin receptor agonists, are known to induce vasoconstriction,^{12–14} and are specifically designed to treat this phenomenon. It is also therefore possible that the pulsatile nature of their symptoms is the result of a vascular irritation to the nerve in question Tables 1 and 2.

Other headaches in the occipital region have already been linked to nerve-artery relationships. For instance, in 2007, Shimizu et al. reported on their anatomical investigation demonstrating that the greater occipital nerve and occipital artery frequently cross paths.¹⁵ They theorized that a close relationship between the two structures might be one of the causes of occipital neuralgia. In that article, they pointed out that trigeminal neuralgia has been shown to be caused in some cases by compression of the nerve root by an artery, and that decompression has been clinically efficacious.

Combining these previous authors' findings with the understanding of migraine patients' symptoms, it was realized that the relationship between the greater occipital nerve and the occipital artery required further investigation. The frequency of this relationship, its morphologic nature, and its anatomical location were all questions to benefit from focused study. An investigation was carried out through fresh tissue dissection to advance the understanding of this intricate anatomy.

Table 1. Results for the Greater Occipital Nerve–Occipital Artery Relationship

Relationship	No.
Hemiheads with no greater occipital nerve–occipital artery relationship	23
Hemiheads with single intersection relationship	8
Hemiheads with helical intertwining relationship	19

Table 2. Topographic Location of Greater Occipital Nerve–Occipital Artery Relationship

Relationship	Location
Mean location of single intersection	30.27 mm lateral to the midline (x); 10.67 mm caudal to a line through the protuberance (y)
Mean location of caudal extent/beginning of helical intertwining	25.34 mm lateral to the midline (x); 24.91 mm caudal to a line through the protuberance (y)
Mean location of cranial extent/end of helical intertwining	42.09 mm lateral to the midline (x); 0.97 mm caudal to a line through the protuberance (y)

METHODS

Twenty-five fresh cadaver heads were obtained from the Willed Body Program at the University of Texas Southwestern Medical Center in Dallas, Texas. All heads used were from donors between the ages of 42 and 86. All bodies were tested for human immunodeficiency virus and other communicable diseases before commencement of dissection. Heads were disarticulated at a low point on the neck (C7 to T1) to maximize the length of the posterior neck for dissection. After shaving, the heads were placed prone and stabilized in a Mayfield neurosurgical headrest. A horizontal line through the occipital protuberance was accurately marked out using an indelible surgical marker. Another line vertically through the midline was also drawn. A methylene blue–tipped, 16-gauge needle was passed through the skin to mark the subcutaneous tissue along these lines to allow accurate measurements within the deeper layers. A no. 10 blade was used to cut down through the skin and subcutaneous tissue, and flaps were raised at this level to expose the galea and trapezius. The greater occipital nerve was located in this plane along with the occipital artery. The occipital artery was dissected proximally to a point more lateral, deep to the sternocleidomastoid. Here, it was ligated and cannulated with a 24-gauge butterfly catheter (0.7-mm diameter) (BD Insyte; Becton Dickinson S.A., Madrid, Spain). Lead oxide stained with red dye was injected carefully into the arteries. The relationship between the occipital artery and the greater occipital nerve was assessed, measured in length, measured from the previously noted anatomical landmarks, and photographed. All data were collated in a Microsoft Excel database (Microsoft Corp., Redmond, Wash.), and means were calculated for the distances from the horizontal line through the protuberance and from the midline, and for the length and mor-

phology of the relationships between the artery and nerve.

RESULTS

A total of 25 fresh heads were dissected bilaterally, for a total of 50 hemihead dissections. Eight of these heads were from female donors, and 17 were from male donors. The mean donor age was 61 years. The greater occipital nerve was found in all specimens. A relationship between the greater occipital nerve and the occipital artery was found in 27 of 50 hemiheads (54.0 percent). This intersection was found either superficial to the trapezius and deep to the galea, or deep to the trapezius. Typically, this intertwining relationship was found in the “trapezial tunnel” area just caudal to the occipital protuberance; however, it was sometimes seen as far anterior as the lateral aspect of the skull, over the occipitalis (Fig. 1).

In heads where an intersection between the artery and nerve was found, there were two types of relationships: a single point of intersection (Fig. 2) and a helical intertwining (Figs. 3 and 4). Nineteen of the 27 intersecting structures were of the helical type (70.4 percent) and eight were of the single-intersection type (29.6 percent). When there was a helical intertwining, this relationship

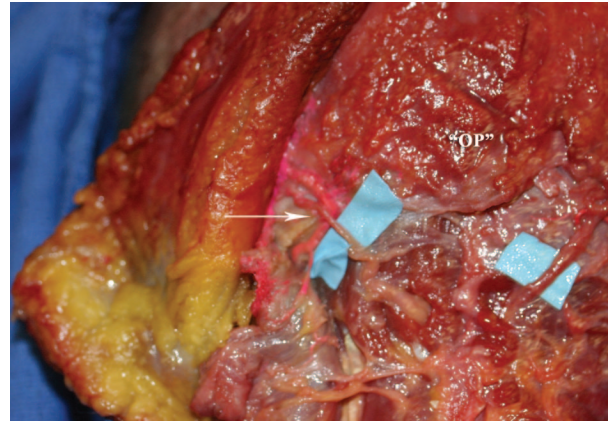


Fig. 2. Image of the single-point-of-intersection type of relationship. Arrow and blue glove cutout demonstrate the area of intersection. OP, occipital protuberance.

was typically a few twists long, with the mean length of interaction being 37.6 ± 14.5 mm. When there was a single point of intersection, the nerve was always superficial to the artery.

The mean location of the artery-nerve relationship when there was a single point of intersection was 30.27 ± 6.83 mm lateral to the midline and 10.67 ± 8.25 mm caudal to the horizontal line through the occipital protuberance (Fig. 5). The

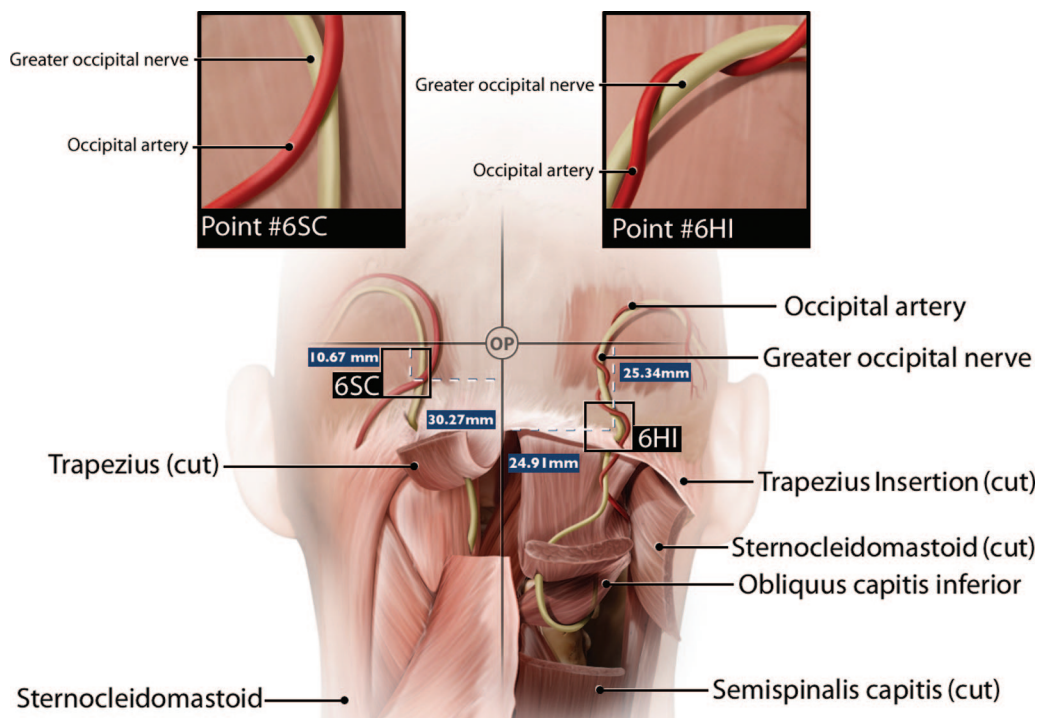


Fig. 1. Illustration of the occipital region, demonstrating more superficial dissection down to the trapezius on the left and deep to the trapezius on the right. (Above, left) The single-cross type of relationship can be seen. (Above, right) The helical intertwining relationship can be seen.

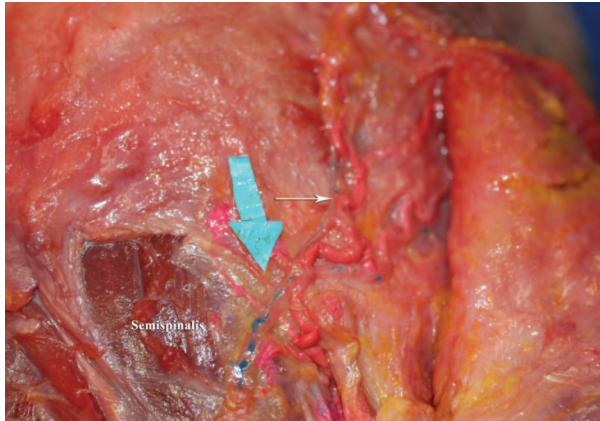


Fig. 3. Image of the helical intertwining type of relationship. Note that there are a number of twists of the artery around the nerve, just below the level of the occipital protuberance caudally (*blue glove cutout*) and then again more cephalad (*white arrow*). A number of small arterial branches off of the occipital artery surround the greater occipital nerve.

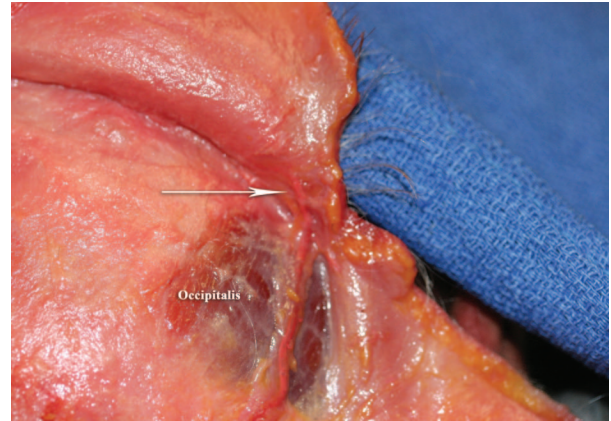


Fig. 5. Image of the anterior extent of the nerve-artery relationship. The occipitalis is labeled. The *arrow* indicates the area where the artery moves into a more superficial plane in the subcutaneous fat; the relationship between the artery and nerve has ended by this point.

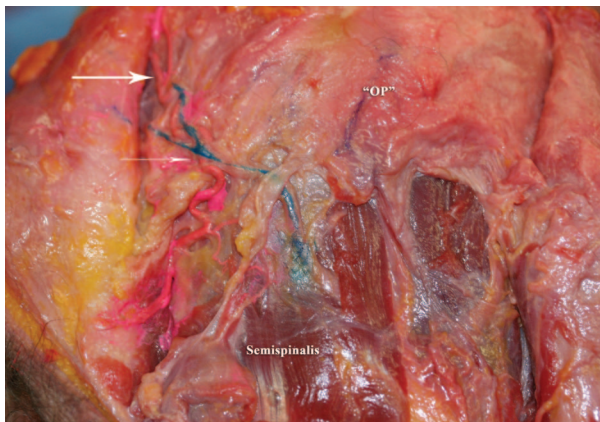


Fig. 4. Image of the helical intertwining type of relationship. Note the occipital protuberance (OP) and semispinalis. The *small arrow* indicates the area where the helical relationship begins, and the *large arrow* indicates the area where the helical relationship between the artery and nerve ends.

mean location of the caudalmost aspect of the artery-nerve relationship when there was a helical intertwining was 25.34 ± 12.16 mm from the midline and 24.91 ± 12.87 mm caudal to the horizontal line through the occipital protuberance; the mean location of the cranialmost aspect of the artery-nerve relationship in this group was 42.09 ± 25.61 mm from the midline and 0.97 ± 8.34 mm caudal to the horizontal line through the occipital protuberance (Fig. 5).

DISCUSSION

An estimated 17.6 percent of women and 5.7 percent of men experience at least one migraine

headache per year.^{16,17} Chronic migraines with and without aura are associated with very intense, pulsatile pain and can be of such severity that they limit patients from regular activities of daily living.¹⁸ Patients with a long history of migraines experience a great amount of anxiety between attacks with the expectation of the next impending attack.^{19,20} Because it is a chronic disease that affects adults during their prime income-producing years, there are staggering indirect costs associated with migraines.^{21,22} Although pharmacologic interventions are popular generally and clinically effective migraine cures, they only serve to reduce their severity and frequency. Therefore, a permanent surgical solution would be an optimal option for patients, physicians, and society as a whole.

Recent clinical reports estimate that approximately 38 percent of patients who undergo surgical decompression of the greater occipital nerve have residual symptoms of varying severity after the operation.⁵ Because of the great success that these decompressions have achieved in many but not all, patients, this suggests the possibility of the surgery being anatomically “incomplete.” This notion has led to further investigations into the anatomy of the lesser and third occipital nerves² and the anatomy of multiple potential compression points along the length of the greater occipital nerve.³ With the understanding that many neurologists strongly subscribe to vascular dilatation as being a primary cause of migraine headaches, it was theorized that the occipital trigger area “nonresponders” to surgery may be suffering from

occipital migraines because of some sort of relationship between the occipital artery with the greater occipital nerve, where the artery causes irritation to the nerve through its intimate association in some patients. This relationship may also be responsible for the pulsatile character of these headaches.

This study shows that there is a relationship between the artery and the nerve in 54.0 percent of specimens. Two types of relationships were found—some of these were singular discrete intersections, whereas others were more interrelated, taking the form of an intertwining helix. It is quite interesting to note that 38.0 percent of all specimens had a helical artery-nerve relationship—the same incidence of clinical nonresponders to surgery.⁵

These findings are in agreement with previous data from other authors. The artery-nerve relationship was also noted in the study by Shimizu et al. However, their findings differed in that they found this relationship to be present in every head, and that the relationship was only of the short intersection variety. They also found this relationship to always be in the subcutaneous tissues, superficial to the trapezius. We found this relationship to exist in this plane when the intersection was more lateral and cranial. When the relationship began at a more inferior point, this intersection seemed to be either deep to the trapezius or deep to the fascia overlying the trapezius yet superficial to the muscle. These authors also noted that the nerve was always superficial to the artery, which we found as well. The authors did note that the intersection occurred around the nuchal line, where the posterior extensors of the neck insert. This corresponds to our findings as well.

It is interesting to see that the specialty of plastic surgery has now come full circle with regard to its intellectual musings concerning the genesis of migraines. Guyuron's previous articles introduced a completely new paradigm in migraine treatment. It was postulated that migraines were perhaps not being caused by a central, vascular phenomenon, but that they were, in fact, incited by a peripheral mechanical phenomenon instead. Clearly, this is the case with some patients, as they respond so well to chemical and/or surgical decompression. Both neurologists and plastic surgeons have become more sophisticated in their understanding of migraine pathophysiology. Recent advances in neurologists' grasp of the central arterial vasodilation theory because of work in experimental animal models²³ have demonstrated

that there is a cascade that is caused not by intracerebral vasodilation but by intracerebral vasoconstriction.¹¹ It has become more apparent that there is a cascade whereby disturbance of brainstem ion channels leads to an ebb in cerebral perfusion.²⁴ This vasoconstriction leads to a release of prodilatory neuropeptides from peripheral nerves, inducing dilation of the extracerebral arterial system.²⁵ It may be that this vascular dilatation is affecting not only the trigeminovascular system but also the occipital artery, thus causing a pulsatile irritation to nerves that are very closely intertwined with the artery, specifically, the greater occipital nerve. Because the occipital artery may receive its parasympathetic innervation from the mandibular branch of the trigeminal nerve at its origin from the external carotid artery, increased activity within the trigeminovascular system may directly affect blood flow throughout the entire occipital artery. It has also been demonstrated that there is shared function in cranial nociception between the trigeminal nucleus and the C2 segment²⁶; increased activity in one should likely induce increased activity in the other. Further research in the field of neurology should yield more in-depth theories and better understanding.

The exciting results from this anatomical investigation will spur further clinical work looking into the relationship between the greater occipital nerve and the occipital artery in migraine patients with an occipital region trigger. Ligation of the occipital artery proximal and distal to its intertwining or intersection with the greater occipital nerve is more facile with the use of the topographic relationships uncovered in this study. Thirty-eight percent of occipital trigger point patients did not achieve complete resolution of migraine symptoms with surgical release; 38 percent of the specimens in this investigation had a helical intertwining relationship between the greater occipital nerve and the occipital artery. It will be interesting to see whether addressing the artery-nerve relationship surgically will bring the number of nonresponders closer to zero.

Jeffrey E. Janis, M.D.

Department of Plastic Surgery
University of Texas Southwestern Medical Center
1801 Inwood Road
Dallas, Texas 75390-9132
jeffrey.janis@utsouthwestern.edu

REFERENCES

1. 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.

2. 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.
3. 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.
4. Guyuron B, Kriegler JS, Davis J, Amini SB. Comprehensive surgical treatment of surgical migraines. *Plast Reconstr Surg.* 2005;115:1–9.
5. 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.
6. 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.
7. Janis JE. Unpublished data.
8. Eross E, Dodick D, Eross M. The sinus, allergy, and migraine study (SAMS). *Headache* 2007;47:213–224.
9. Saper JR. Diagnosis and symptomatic treatment of migraine. *Headache* 1997;37:S1–S14.
10. Kastrup A, Thomas C, Hartmann C, Schabet M. Cerebral blood flow and CO₂ reactivity in interictal migraineurs: A transcranial Doppler study. *Headache* 1998;38:608–613.
11. Goadsby PJ, Lipton RB, Ferrari MD. Migraine: Current understanding and treatment. *N Engl J Med.* 2002;346:257–270.
12. Maassenvandenbrink A, Chan KY. Neurovascular pharmacology of migraine. *Eur J Pharmacol.* 2008;585:313–319.
13. Villalon CM, Centurion D, Valdivia LF, de Vries P, Saxena PR. Migraine: Pathophysiology, pharmacology, treatment and future trends. *Curr Vasc Pharmacol.* 2003;1:71–84.
14. Silva SA, Marques FB, Fontes-Ribeiro CA. Characterization of the human basilar artery contractile response to 5-HT and triptans. *Fundam Clin Pharmacol.* 2007;21:265–272.
15. Shimizu S, Oka H, Osawa S, et al. Can proximity of the occipital artery to the greater occipital nerve act as a cause of idiopathic greater occipital neuralgia? An anatomical and histological evaluation of the artery-nerve relationship. *Plast Reconstr Surg.* 2007;119:2029–2034; discussion 2035–2036.
16. Stewart WF, Lipton RB, Celentano DD, Reed ML. Prevalence of migraine headache in the United States: Relation to age, income, race, and other sociodemographic factors. *JAMA.* 1992;267:64–69.
17. Tepper SJ. A pivotal moment in 50 years of headache history: The first American migraine study. *Headache* 2008;48:730–731; discussion 732.
18. Saunders K, Merikangas K, Low NC, Von Korff M, Kessler RC. Impact of comorbidity on headache-related disability. *Neurology* 2008;70:538–547.
19. Dahlof CG, Dimenas E. Migraine patients experience poorer subjective well-being/quality of life even between attacks. *Cephalalgia* 1995;15:31–36.
20. Freitag FG. The cycle of migraine: Patients' quality of life during and between migraine attacks. *Clin Ther.* 2007;29: 939–949.
21. Lipton RB, Stewart WF, Von Korff M. The burden of migraine: A review of cost to society. *Pharmacoeconomics* 1994; 6:215–221.
22. Solomon GD, Price KL. Burden of migraine: A review of its socioeconomic impact. *Pharmacoeconomics* 1997;11:1–10.
23. Yamamura H, Malick A, Chamberlin NL, Burstein R. Cardiovascular and neuronal responses to head stimulation reflect central sensitization and cutaneous allodynia in a rat model of migraine. *J Neurophysiol.* 1999;81:479–493.
24. Silberstein SD. Migraine pathophysiology and its clinical implications. *Cephalalgia* 2004;24:2–7.
25. Edvinsson L. Blockade of CGRP receptors in the intracranial vasculature: A new target in the treatment of headache. *Cephalalgia* 2004;24:611–622.
26. Bartsch T, Goadsby PJ. Stimulation of the greater occipital nerve induces increased central excitability of dural afferent input. *Brain* 2002;125:1496–1509.



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