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## Discussion

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# The Anatomy of the Greater Occipital Nerve: Implications for the Etiology of Migraine Headaches

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This work addresses a topic that I believe to be the most interesting and potentially revolutionary concept engaging plastic surgeons in the past 10 years. Specifically, it relates the anatomy of the greater occipital nerve to external landmarks, and it reviews the apparent role of that nerve as an etiology of a clinical problem that affects approximately 28 million people in the United States: migraine headache. As an anatomical study, it is well designed and cogently presented. As a therapeutic construct, it expands upon the senior author's working hypothesis that "the underlying etiology of migraine symptomatology may be peripherally rather than centrally mediated." The basis of this argument that chronic compression by skeletal muscle surrounding a sensory nerve can serve as a trigger point for migraine is the observation by many different sources that botulinum toxin A chemoblockade is often effective in eliminating migraine pain for several months. This is a startling concept, quite counter to the traditional views of migraine as a complex phenomenon reflective of the intracranial interplay of neurotransmitters, external stimuli, genetics, and emotional factors. As such, it has been met with skepticism by many neurologists and other pain management specialists.

Before considering the theoretical implications of this article, it may be useful to reiterate its anatomical goal: describing the course of the greater occipital nerve "so that points of chemodenervation using external landmarks can be used to relieve migraine symptoms." The authors found, in dissections of cadavers with no known history of migraine, that this nerve reliably courses through the semispinalis

muscle but otherwise traverses fascial planes. No other muscle typically invests the nerve. They locate the specific point at which the greater occipital nerve can be expected to be coursing through the semispinalis muscle, finding the target zone of the botulinum toxin A block to be 3 cm below the external occipital protuberance and 1.5 cm lateral to the midline. These measurements are similar to those of Bovim et al.,<sup>1</sup> but the additional information regarding the distribution of these measurements is reassuring. If the pharmacologic effect of botulinum toxin A is limited to neuromuscular blockade, it is obviously critical to know where muscle and nerve are intimately related, i.e., this is the place, and the only place. As a final observation of the experimental model, it should be noted that this study describes the course of the greater occipital nerve in a "normal" population. It is possible that some or all migraine patients experience headache because their anatomical relationships are not normal. This is not a criticism of this work, because an anatomical study of head pain patients would present significant difficulties, and in any case, a "normal" baseline is necessary for significant abnormalities to be appreciated.

Botulinum toxin A blockade for headache serves two purposes. First, it offers some significant chance of long-term pain relief, usually lasting 2 to 4 months. This is generally very similar to the duration of the effect of botulinum toxin A on muscle. A second value of botulinum toxin A chemoblockade of the semispinalis muscle or the muscles of the brow is diagnostic; relief suggests that muscle is playing an active role in causing pain. This is di-

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rectly analogous to the use of local anesthetic nerve blockade in other clinical situations, such as meralgia paresthetica, to diagnose nerve irritation or compression. If the only effect of botulinum toxin A is its interference with acetylcholine release at the neuromuscular junction, weakening or paralyzing the injected muscle, this logic is valid. Unfortunately, if one prefers straightforward solutions, many neurologists hypothesize that botulinum toxin A also causes “central” nervous system effects that, while poorly elaborated, somehow cause pain relief. I do not find the evidence for the existence of these other effects to be compelling. It is difficult to imagine a local anesthetic-type response or a morphine-like effect lasting for several months.

As a final point, the authors focus on the relationship of the greater occipital nerve to the semispinalis muscle but do not discuss other sites of potential compression of this nerve. Until recently, my experience with exploration and decompression of the greater occipital nerve for pain was limited to the more superficial course of the nerve after its exit from the semispinalis muscle. In a large series of patients treated during the last 30 years and reported somewhat anecdotally in this *Journal* a few years ago,<sup>2</sup> I relied on local anesthetic block responses as my primary indication for decompression of the nerve at its exit point from the trapezius fascia. At that level, I often encounter significant lymphadenopathy (Fig. 1) or an anomalous relationship of the occipital artery to the nerve; both of these often appear to be definite sources of nerve compression.

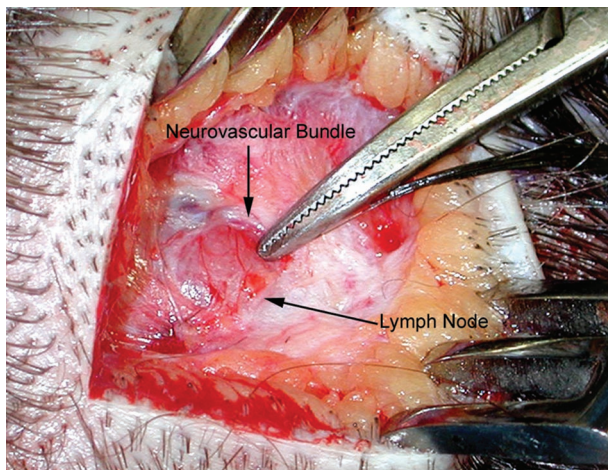


FIG. 1. Greater occipital nerve exposed at the exit point from trapezius fascia, with the perineural lymph node causing apparent compression.

I believe that this is analogous to a herniated spinal disc causing nerve root pain or to pain resulting from compression of the median nerve in the carpal canal. These anesthetic blocks often contain steroids as well and are placed in a horizontal band along the nuchal line. This is approximately 2 to 3 cm more cephalic than the botulinum toxin A blocks under discussion here. There would appear to be little logic in using botulinum toxin A at that level. There is simply no muscle surrounding the greater occipital nerve as it exits the trapezius fascia and branches beneath the occipital scalp. Thus, I believe that there is diagnostic value to both botulinum toxin A and local anesthetic/steroid blocks in evaluating headache. The former is appropriate proximally, at the point identified by these authors. This is the only site of potential muscle compression. More distally, local anesthetic is the logical agent to determine whether nerve compression is resulting from a structural abnormality at a more superficial level.

In summary, I share the belief of these authors that headache, whether classified as migraine or one of the many other diagnostic types of head pain, often arises from irritation or compression of peripheral sensory nerves of the face or occipital region. Is it possible that this major health problem, which often results in significant disability and is refractory to the efforts of pharmaceutical companies and other sources of treatment, is frequently a simple mechanical phenomenon? The ongoing work of Dr. Guyuron and his coauthors, as reported in two previous articles and further refined by this current study, is compelling. It is appropriate to doubt that a simple explanation for a disorder long believed to be highly complex and multifactorial could be valid in this age of sophisticated science. In that regard, the recently appreciated relationship of peptic ulcer disease to chronic infection by *Helicobacter pylori* should be recalled. In that illness, factors of emotion and personality type, in addition to other systemic and often mysterious causes, were considered to be of primary importance for many years. Finally recognizing that a “simple” chronic infection is a major cause of most peptic ulcer disease was enlightening and appropriately humbling. The work of Dr. Guyuron and his colleagues will ultimately be confirmed or refuted by studies at other centers; these are in

progress. With these, the appropriate role of plastic surgeons in the treatment of head pain will be defined. Stay tuned.

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## REFERENCES

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