

Case Report

Neuromodulation of the Great Auricular Nerve for Persistent Post-Traumatic Headache

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Headache is the most frequent reason for referral to an outpatient neurology and pain physician practice, with post-traumatic headache (PTH) accounting for approximately 4% of all symptomatic headaches. Headache following trauma has been reported for centuries. In this unique case report we will discuss the clinical course and successful headache treatment of a 57-year-old man diagnosed with PTHs. He suffered from chronic, intractable headaches resistant to multidisciplinary medical management for 4 years.

A trial of electrical neuromodulation of the C2-C3 branches within the great auricular nerve (GAN) distribution was proposed as a potential long-term treatment for his chronic, intractable headaches after having several prior headache attacks successfully aborted with ultrasound-guided GAN blocks.

Six months after permanent peripheral neurostimulator implantation, the patient reported a greater than 90% reduction in headache frequency, and was able to wean off all his previous prophylactic and abortive headache medications, with the exception of over-the-counter ibuprofen as needed.

Subcutaneous electrode application over the branches of C2-C3—namely greater, lesser, and the least occipital nerves—for the treatment of chronic, intractable headache is not a new concept within pain medicine literature. However, subcutaneous electrode application, specifically over the GAN, is unique. The following case report chronicles the novel application of ultrasound-guided peripheral nerve stimulation of the GAN as an effective and safe long-term treatment for chronic, intractable primary headache. The positive outcome chronicled in this case presentation suggests that peripheral nerve stimulation of the GAN should be considered for highly select cases. To our knowledge, this is the first such case report describing GAN as a target for the management of PTH in the literature.

Key words: Post-traumatic headache, great auricular nerve, great auricular neuralgia, headache, neurostimulation, electrical neuromodulation

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Headache is the most frequent reason for referral to an outpatient neurology practice, with post-traumatic headache (PTH) accounting for approximately 4% of all symptomatic headaches (1).

The immediate effects of head injury are complex and can include cell injury or death; neurovascular dis-

ruption; disturbances of ionic and neurotransmitter homeostasis; and electrical, chemical, and energetic dysfunction. The nature of the injury, whether more focal or more diffuse, influences the pattern of these processes (2).

Headache following head injuries has been reported for centuries. From the eighteenth century on-

wards, numerous hypotheses for post concussive symptoms were put forth, including "the hypothesis of brain commotion," "the hypothesis of circulatory failure," "the hypothesis of acute compressive anemia," and the hypothesis of "molecular vibration and spinal concussion." Although the proposed mechanisms for the generation and perpetuation of PTH have evolved over the years, they remain the subject of considerable debate (3). A new trauma-induced headache could develop as an add-on to an existing primary headache form or may bring out the primary headache for the first time in patients with genetic predisposition due to family history of primary headache disorders.

In 1988, the International Headache Society Classification Committee, First Edition, provided the first formal definitions for PTH by providing operational criteria for the diagnosis of acute and chronic PTH. Furthermore, they subdivided acute and chronic "headaches associated with head trauma" into 2 subforms related to mild head injury and moderate to severe head injury. In the revised International Headache Society Classification of Headache Disorders, 3rd edition (ICHD-3 beta), there are no specific headache features known to distinguish the subtypes of "headache attributed to trauma or injury to the head and/or neck from other headache types"; most often these resemble tension-type headache or migraine. Consequently their diagnosis is largely dependent on the close temporal relation between the trauma or injury and headache onset. PTH has many subtypes including acute PTH attributed to moderate or severe head injury, acute PTH attributed to mild head injury, chronic PTH attributed to moderate or severe head injury, chronic PTH attributed to mild head injury, acute headache attributed to whiplash injury, chronic headache attributed to whiplash injury, headache attributed to traumatic epidural or subdural hematoma, acute or chronic headache attributed to other head or neck trauma, and acute and chronic post craniotomy headache. The diagnostic criteria of ICHD-3 beta for all subtypes require that headache must be reported to have developed within 7 days of trauma or injury or within 7 days after regaining consciousness and/or the ability to sense and report pain when these have been lost following trauma or injury. When a new headache occurs for the first time in close temporal relationship to a known trauma, it is coded as a PTH. When a pre-existing headache (ie, migraine) is made worse in close temporal relation to a head trauma, the diagnosis should be pre-existing migraine headache exacerbated by headache attributable to head injury.

The majority of PTH patients will report resolution of their complaints within a few months from the time of the initial injury. Multiple studies have documented recovery rates; however, the methodology of those studies varied greatly, providing for inconsistent results. The percentage of patients with headaches at one month varies from 31.3% to 90%, at 3 months from 47% to 78%, and at one year from 8.4% to 35%. Almost 25% of patients will report refractory headaches at 4 years (4).

Exciting basic science research has uncovered an important connection between the trigeminocervical complex and the manifestation of primary headache syndromes, such as migraine and cluster headaches. For instance, direct coupling between meningeal afferents and cervical afferents in the spinal dorsal horn has been recently described in detail. Moreover, mapping of the trigeminocervical complex in cats revealed that nociceptive afferents reside in the caudal region of the trigeminal nucleus caudalis and extend into the dorsal horns of the C1 and C2 cervical segments without extending significantly to the C3 level. These neurons were easily accessible and could be activated by both electrical and mechanical stimuli. As a result, it is plausible that the trigeminocervical complex may serve as an important therapeutic target for the treatment of headache syndromes. Currently, a commonly employed treatment for headache disorders involves occipital nerve injection with a local anesthetic and corticosteroids (5).

The great auricular nerve (GAN) is a purely sensory nerve and is the largest of the ascending branches of superficial branches of the cervical plexus. The GAN arises from the second and third cervical nerves (C2, C3), winds around the posterior border of the sternocleidomastoid, and after perforating the deep fascia, ascends upon that muscle beneath the platysma to the parotid gland, where it divides into an anterior and a posterior branch. The branches are distributed to the skin of the face over the parotid gland and the skin over the mastoid process, extending to the back of the auricle. Great auricular branches communicate with the lesser occipital nerve, the auricular branch of the vagus nerve, and the posterior auricular branch of the facial nerve (6).

Regardless of the length of the sternocleidomastoid, the GAN at its most superficial location was found to be at a consistent ratio of one-third the distance from either the mastoid process or the external auditory canal to the clavicular origin of the sternocleidomastoid. (7). In addition to the use of surface anatomy

(Fig. 1) and bony landmarks for guidance, the external jugular vein can also be used as a landmark for the location of the GAN, as the GAN is approximately 1 cm superior and lateral to the external jugular vein coursing in a trajectory parallel to the vein (Fig. 2).

Anatomically, the GAN is protected as it courses behind the sternocleidomastoid. Once it emerges onto the anterior surface of the muscle, it resides in a superficial plane, making it accessible for blind injection, yet also vulnerable to traumatic, or even iatrogenic injury.

High definition ultrasonography has revolutionized the visualization of the GAN and other surrounding soft-tissues, such as the spinal accessory nerve, greatly facilitating the performance of several targeted interventional pain medicine procedures. For example, a percutaneous GAN peripheral nerve stimulator catheter can be implanted by utilizing ultrasound guidance. Likewise, among posterior cervical triangle structures, ultrasound guidance greatly enhances identification of the spinal accessory nerve (Fig. 3), which due to its relatively long and superficial course in the posterior triangle of the neck is also vulnerable to iatrogenic injury. An important advantage of utilizing ultrasound guidance for percutaneous nerve stimulator implantation, whether at the GAN or the spinal accessory nerve, is the presence of fewer complications—particularly iatrogenic complications—associated with open surgical permanent implantation of neurostimulation devices.



Fig. 1. On surface anatomy, GAN emerges onto the anterior surface, approximately at one-third the distance from either the mastoid process or the external auditory canal to the clavicular origin of the sternocleidomastoid. Regardless of the neck length, the picture shows the patient at the end of stimulator trial insertion procedure.

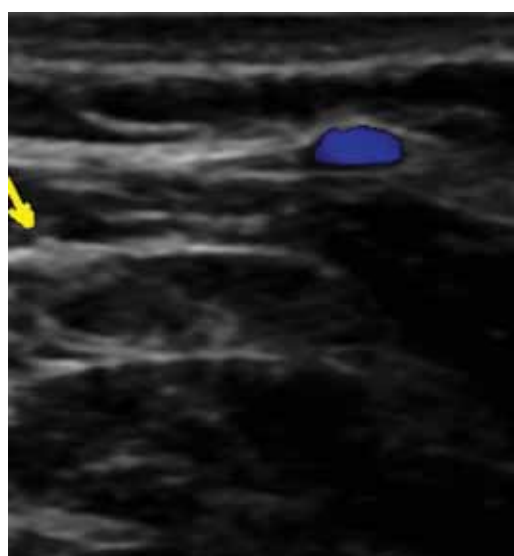


Fig. 2. Ultrasound picture; cross section of GAN in approximately 1 cm lateral to jugular vein.

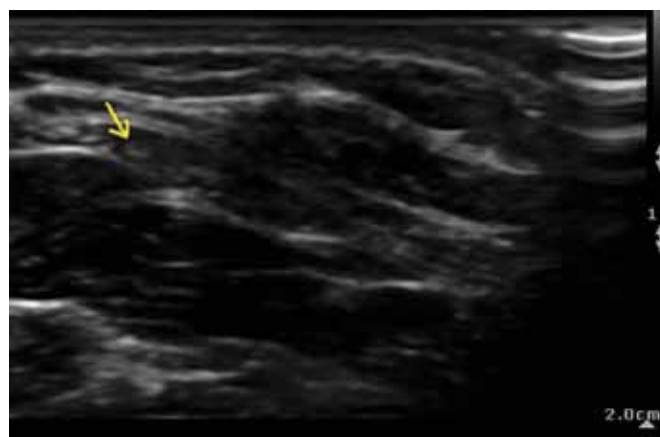


Fig. 3. Ultrasound depiction of accessory cranial nerve on the posterior cervical triangle.

Case Presentation

This is a 57-year-old man with a more than 4-year history of chronic headaches and facial pain secondary to a head injury. He was accidentally hit with a pipe to his forehead then fell backwards and developed loss of consciousness for more than 20 minutes. He was taken to a local emergency room where his left occipital scalp wound was repaired and evaluated with a brain computed tomography (CT) scan. The brain CT scan was reported as normal. He was discharged from the emergency room after 8 hours of close observation. He describes that his headaches started the next day after injury. He has a history of multiple ER visits due to exacerbations of headache.

He has been tried on several preventative medications. He had no significant benefit from a long list of medications including opioids. He has minimized caffeine and over-the-counter medications to address the concern for medication overuse rebound headaches. Currently he is taking naprosyn, depakote, celexa, and melatonin. He recently started using a TENS unit and reported some benefit.

Detailed evaluation under neurologist supervision during past 4 years, including brain CT scanning, brain magnetic resonance imaging (MRI), and electroencephalogram, was reported as normal. The patient has had

a detailed neuro psychologist evaluation which showed normal cognition and executive function except for unreliable attention which may be secondary to his head injury or his current medications. He was referred to a pain psychologist and he is actively participating in the teaching sessions for biofeedback, breathing, and relaxation therapy.

Due to persistent headaches, he referred to the Center of Pain Medicine. He reported that his headaches are constant, throbbing, start on the left fronto parietal side, and spread to the entire head. He denies having nausea, vomiting, aura, and photophobia, but he may have phonophobia and visual blurring during the worst pain. Pain intensity ranged from a low of 5 to a high of 10 on the 10-point pain numeric rating scale (NRS), whereas his average daily headache was reported as a 4 to 6 on the NRS.

As the patient's severe headaches persisted unabated, we proposed trialing a GAN block, to which the patient agreed. Under ultrasound guidance, a left-sided GAN block was performed using an injectate of 3 mL of 0.25% bupivacaine. The patient reported near complete pain relief 10 minutes after the block with a total absence of headache lasting 8 hours.

In light of the positive GAN block results, we discussed GAN neurostimulator implantation as a potential option for long-term treatment. The patient agreed to undergo 7 days of a GAN stimulator trial, and the procedure was done utilizing ultrasound guidance and percutaneous leads (Fig. 4). The procedure was tolerated well by the patient without any complications. We did telephone follow-ups on a daily basis during the 7 days of trial. He consistently reported 100% daily headache pain relief. He was able to recognize one headache episode on day 4 and he reported he experienced extreme reduction (90%) on severity. He did not use any medication during the 7 days of the trial.

At six-month follow-up post GAN permanent neurostimulator implantation, the patient reported significant, sustained pain alleviation, with daily baseline pain scores averaging one to 2 on the NRS scale. Furthermore, he reported not using any abortive medication for the past 6 months, even though we suggested over-the-counter analgesic medication use. Overall, he was extremely satisfied with the positive results.

Discussion

Acute PTH following head trauma is frequent and often resolves within a few weeks, but for a smaller proportion of patients the headache becomes persistent and often severely disabling. Paradoxically, head-

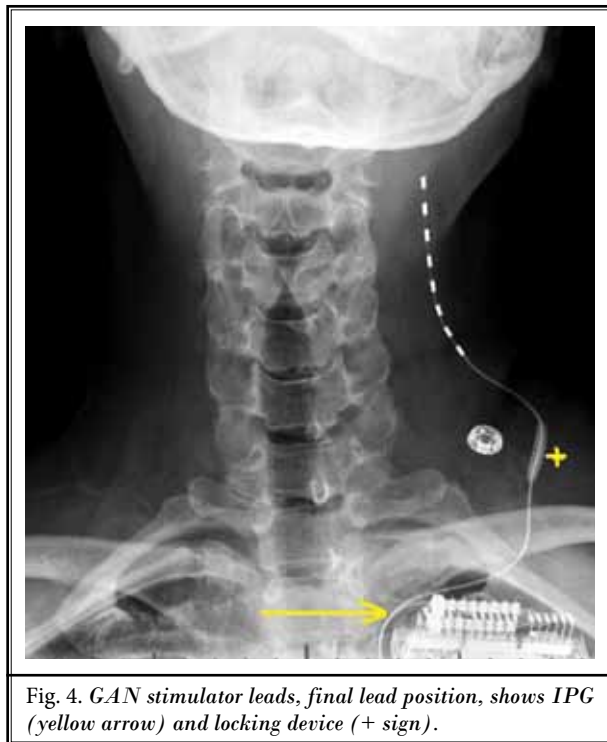


Fig. 4. GAN stimulator leads, final lead position, shows IPG (yellow arrow) and locking device (+ sign).

ache prevalence and duration is greater in those with mild head injury compared with those with more severe trauma (8).

Persistent PTH is often difficult to treat, particularly since significant symptomatic overlap amongst primary headache syndromes makes establishment of an accurate diagnosis challenging. As such, an integrated, interdisciplinary approach is of the highest priority for this patient group. With the implementation of multidisciplinary and multimodal approaches, only a small minority of chronic PTH patients remain refractory to treatment. This select group of PTH patients may potentially be appropriate candidates for electrical neuromodulation treatment.

For over a decade the use of neuromodulation for occipital neuralgia, as well as other headache syndromes, has become a widespread successful therapy. Although fewer case reports discuss neuromodulation for patients with cervicogenic and C2-mediated headaches, evidence in support of this use also exists. The most commonly accepted mechanism of action for this treatment is believed to involve stimulation of the distal branches of C2 and C3 that convergence with the trigeminal system, possibly inhibiting central nociceptive processing. It is well known in the medical literature that electrical neuromodulation of the great occipital nerve is beneficial in headache management (9,10).

Since favorable pain alleviation results have been seen with neurostimulation of the occipital nerve, we considered neurostimulation of the GAN—which is also composed of branches from C2 and C3—to see if similar pain alleviation effects would be produced.

In our case report we were able to demonstrate excellent pain relief not only on the ipsilateral side, but also on the contralateral side. This advantageous finding correlates with the underlying convergent synaptic connections between the trigeminocervical neurons and both ipsilateral and contralateral afferents. Collectively, these findings lend support for the convergent nature of trigeminocervical synaptic input, while also demonstrating that neuromodulation actively inhibits nociceptive input both ipsilateral and contralateral (11).

To the best of our knowledge, there have been no publications to date concerning the application of peripheral nerve stimulation over the GAN for PTH. This unique application of neuromodulation is an adaptation of 2 currently accepted chronic headache treat-

ments—greater occipital nerve blocks and occipital nerve neuromodulation—and its efficacy is believed to be derived from the same mechanism of action. On the whole, neuromodulation harbors several inherent advantageous qualities: it is nondestructive, minimally invasive, and usually fully reversible.

Determining which PTH patients are appropriate candidates for neuromodulation poses a unique challenge.

The positive outcome chronicled in our case presentation suggests that peripheral nerve stimulation of the GAN should be considered as a potentially viable and safe therapeutic option for persistent, refractory PTH.

From our experience, we recommend ultrasound guidance be used to perform GAN neurostimulator, to decrease the surgical complication risk of GAN damage, and also to avoid accessory nerve injury.

In recent years, neuromodulation has experienced a renaissance as a treatment option for a variety of chronic pain conditions. GAN neuromodulation offers a unique opportunity to better understand and reduce the disability of a proportion of patients with medically intractable, primary headache disorders. This case report potentially opens yet another treatment option in the armamentarium of interventional pain medicine practitioners against chronic, primary headache.

CONCLUSION

Ideal PTH candidates for electrical neuromodulation are those patients who fall under the category of medically “intractable headache,” defined as headache that is uncontrollable, unmanageable, and/or refractory to multimodal treatment.

Satisfactory pain relief after a GAN block may serve an important prognostic role in identifying appropriate GAN neuromodulation candidates.

A thorough work-up consisting of a complete history and physical examination and brain imaging studies is crucial to rule out other diagnoses and to correctly classify each headache presentation. Establishment of an International Headache Society diagnosis is desirable before consideration of any device-based therapy.

GAN electrical neuromodulation can be extremely beneficial for highly select head-injured individuals, suffering from intractable PTH with disabling headaches despite aggressive and comprehensive treatment.

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