

## Case Report



## High Cervical Epidural Neurostimulation for Post-Traumatic Headache Management

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Headache following head injuries has been reported for centuries. The majority of post-traumatic headache (PTH) patients will report resolution of their complaints within a few months from the time of the initial injury. PTHs can contribute to disability, lost productivity, and health care costs.

In this article we discuss a 40-year-old male with a history of motor vehicle accident and basal skull fracture. The patient had no headache history prior to the accident. He presented with more than 3 years persistent daily headache. The patient described constant throbbing and stabbing quality headaches predominantly on the left hemicranium with constant facial pain. He denies having aura, nausea, or vomiting, but reported occasional neck tightness.

An extensive workup was carried out under the direction of the patient's primary neurologist. Secondary to persistent intractable pain, the patient was referred to the pain clinic for further evaluation. As his headaches were resistant to all trialed strategies, we decided to turn our therapeutic focus toward electrical neuromodulation along with continuing multimodal medications and multidisciplinary approach. During 7 days of high cervical dorsal column electrical nerve stimulation trial, he reported almost 90% pain reduction and significant improvement on his quality of life. On 12 months follow-up after he underwent a permanent implant of high cervical dorsal column electrical nerve stimulation, he reported the same level of pain reduction along with 100% satisfaction rate.

To the best of our knowledge, there have been no publications to date concerning the application of high cervical nerve stimulation for PTH.

**Key words:** Post-traumatic headache, migraine headache, cluster headache, electrical neurostimulation, neuromodulation, trigeminocervical complex, high cervical spinal cord stimulation

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**H**eadache 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 disruption; 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. Because of the poor correlation between the old and the modern headache types and classifications, until the end of the eighteenth century the history of headache is dealt with as a whole, largely regardless of headache category. There are

significant documents with original translations of the ancient Egyptian and Greek and the medieval Arabian doctors about symptoms, suspected mechanisms, and remedies for headache management.

From the eighteenth century onwards, 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 sub forms 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 (i.e., 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 (4).

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 (5).

Exciting basic science research has uncovered an important connection between the trigeminocervical complex and the manifestation of headache syndromes. 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 (6).

### Case Report

A 40-year-old right-handed white male referred to the center of pain medicine. He has history of motor vehicle accident that happened 3 years ago in which he sustained a left temporal linear skull base fracture. He does not exactly remember the mechanism of the accident, but reportedly he drove off the road and hit a tree. According to the hospital records, he experienced loss of consciousness less than 30 minutes and also had a short duration of left-sided facial palsy after the accident which has improved over time. The patient had no headache history prior to the accident.

He presented with persistent headache predominantly on the left side occipital, temporal, and frontal, and persistent left-sided facial pain ever since after the accident.

The patient described headaches as constant throbbing and stabbing quality with constant left side facial pain. He has mild to moderate pain on the right side with constant dull pain and occasional exacerbation. He denies having aura, nausea, vomiting, diplopia, hearing loss, or gait and balance problems, but reported occa-

sional neck tightness. In addition he occasionally thinks the left ear is on fire but never observed ear redness. Barometric pressure changes cause the pain in his ear to be much worse.

Noted exacerbating factors were bright lights, loud sounds, and activities that required strenuous physical tension and sustained mental concentration, such as multitasking. The sole consistent relieving factor reported was sleep.

He reported that he never experienced pain-free periods during past 3 years. He rated his pain intensity as ranging from a low of 6 to a high of 10 on the 10-point pain numeric rating scale (NRS).

An extensive workup was carried out under the direction of the patient's primary neurologist. Doppler solography of the extra- and intracranial vessels, x-rays of the cervical spine, brain and cervical magnetic resonance imaging (MRI), and electroencephalogram showed normal findings.

Secondary to persistent intractable pain, the patient was referred to the pain clinic for further evaluation. After obtaining a detailed history, performing a thorough physical examination—which was without any focal neurologic findings—and reviewing all prior imaging studies and tests performed, the findings were strongly suggestive of a diagnosis of PTH.

He has been tried on multiple medications through his neurologist in the past, including migraine headache prophylaxis, amitriptyline, gabapentin, pregabalin, and Topamax. He has also been on significant doses of opioids medications, with no significant improvement in his symptoms. Currently, he is on oxcarbazepine and Cymbalta. He thinks he has about a 10% – 20% improvement on daily headaches with his current medications.

He is actively utilizing biofeedback and coping and cognitive behavioral therapy techniques on a daily basis.

He experienced occipital nerve block, sphenopalatine ganglion block, and Botulinum Toxin injections in the past along with trigger-point injections on his neck with no significant improvement.

On physical examination he has diminished hearing in the left ear. He has mild left side naso-labial fold flattening. Muscle strength is rated normal throughout the upper and lower extremities. His balance and gait are normal.

As his headaches were resistant to all prior trials we decided to turn our therapeutic focus toward electrical neuromodulation along with continuing multimodal medications (chemical neuromodulation) and a multidisciplinary approach.

We decided to proceed with a high cervical electrical stimulation. Implantation of the spinal cord stimulation (SCS) electrode was performed after he demonstrated 90% improvement of his pain during 7 days of a percutaneous spinal cord stimulator trial. It followed standard procedures for high cervical epidural neurostimulation. A 45-cm, 8 contacts MRI compatible percutaneous electrode (Medtronic, Minneapolis, MN, USA) was used. The electrode was directed cranially and slowly pushed to the upper cervical region. The final position was reached when a further cranial movement was stopped by the C1 lamina (Fig. 1).

The optimum clinical goal was to cover the baseline daily pain. The clinical goal was achieved along with a significant decrease in attack frequency and intensity.

At follow-up 3, 6, 9, and 12 months after implantation of the device, the patient reported significant, sustained pain alleviation, with daily baseline pain scores averaging one to 2 on the NRS scale. He is using moderate doses of oxcarbazepine and Cymbalta. In fact, he also reported not requiring any abortive medication for the past 9 months, as he had not experienced any further headache episodes. He reported 100% facial pain improvement and no further ear pain with 90% headache improvement. Overall, he was extremely satisfied with the positive results.

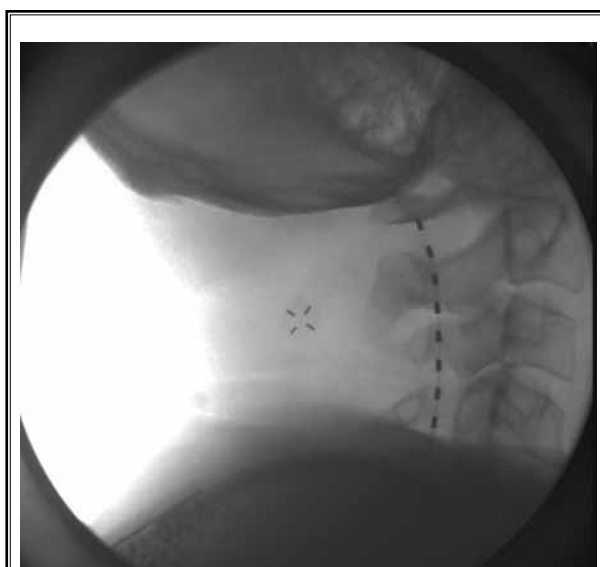


Fig. 1. Lateral cervical X-ray shows the final lead position.

Currently, the patient is constantly using the electrical stimulator on a daily basis with the stimulation parameters of 0.4V, 120 HZ, 120 msec.

**Discussion**

There is enough epidemiological evidence to suggest that PTH exists. 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, headache prevalence and duration is greater in those with mild head injury compared with those with more severe trauma (7).

Despite the advances in modern science in general, and headache medicine in particular, PTH has remained enigmatic for centuries. Given the high prevalence of PTH, and the disproportionately high degree of disability associated with PTH, the paucity of research in this field and the absence of treatment guidelines are disappointing for patients and their treating physician alike.

Unlike some of the primary headache disorders (migraine and cluster), the biological substrate of PTH is less well understood and functional imaging correlates have yet to be uncovered. The potential contributing factors from medication (analgesic) overuse, psychological factors (i.e., adjustment disorder, posttraumatic stress disorder), and litigation-related factors further muddy the waters. Consequently, the credibility of the PTH sufferer is, unfortunately, frequently called into question (8). Large voids remain in our understanding of the pathophysiology and multitude of clinical presentations of PTH.

In order to effectively study and manage headache disorders, diagnosis is essential. In both research and clinical arenas, separating secondary causes from primary headache disorders is a crucial first step, followed by further specificity within these broader categories.

At this time, it is unclear how PTH can best be treated. It is unknown whether typical headache treatments work as effectively for PTH as for primary headaches. There is little research and no evidence-based guidelines on which to base treatment as PTH is typically an exclusion criteria for studies examining headache treatments in the general population.

A multidisciplinary approach is critical in the treatment of PTH because of the multitude of associated symptoms and presentations. Before implementing a specific treatment plan, a treating physician must perform a comprehensive evaluation of each individual patient, taking into account their history and injury type in addition to exam and complaints.

Other options in the treatment of PTH include trigger point injections, occipital nerve blocks, botulinum toxin injections, and cervical medial branch blocks.

Non-pharmacological treatment methods also include physical therapy and manipulation, biofeedback and relaxation therapy, transcutaneous nerve stimulators, and behavioral therapies. Vestibular rehabilitation aids in the treatment of balance dysfunction due to migraine, cervicogenic dizziness, and other peripheral vestibular disorders.

A lack of proper diagnosis or lack of knowledge regarding headache after PTH by health care providers may also lead to inadequate treatment of PTH.

The use of a peripheral nerve stimulator for headaches was introduced even before the gate control theory was published. In 1966, Shelden implanted electrodes around the mandibular nerve for the treatment of trigeminal neuralgia, obtaining temporary relief. In 1967, Wall and Sweet inserted the electrode into their own infraorbital foramen and reported pain relief with electrical stimulation. Weiner and Reed placed electrodes subcutaneously near the occipital nerve on 12 patients, who showed good to excellent relief lasting up to 6 years (9) (Table 1).

Table 1. *Electrical nerve stimulation targets for craniofacial pain management (studies in human and animal models).*

High Cervical Dorsal Column Epidural Spinal Cord (trigeminal nucleus caudalis, trigemino-cervical convergence )
Greater and Lesser Occipital Nerve
Great Auricular Nerve
Supraorbital Nerve
Infraorbital Nerve
Sphenopalatine ( Pterygopalatine) Ganglia
Trigeminal Ganglia
Trigeminal Branches: Maxillary Nerve/Mandibular Nerve

For more than a decade, the use of neuromodulation for occipital neuralgia, as well as other primary headache syndromes, has become a widespread successful therapy. 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. As favorable pain alleviation results have been seen with neurostimulation of the occipital nerve, we considered neurostimulation of the high cervical dorsal column—which is also composed of the trigeminal nucleus caudalis and extends into the dorsal horns of the C1 and C2 cervical segments with some extension to the C3 level—to see if similar pain alleviation effects would be produced. In our case report, we were able to demonstrate excellent pain relief. This advantageous finding correlates with the underlying convergent synaptic connections between the trigeminocervical neurons.

Collectively, these findings lend support for the convergent nature of trigeminocervical synaptic input while also demonstrating that neuromodulation actively inhibits nociceptive input (10).

In recent years, neuromodulation has experienced a renaissance as a treatment option for a variety of chronic pain conditions. Dorsal SCS is an approved technique for chronic pain management that can be easily performed and does not carry great risk. Cervical

SCS, in particular, has been described as efficacious in complex regional pain syndrome, brachial plexus injury, Raynaud syndrome, and neck and upper limb pain after cervical spine surgery.

High cervical neuromodulation offers a unique opportunity to better understand and reduce the disability of a proportion of patients with medically intractable PTH.

To the best of our knowledge, there have been no publications to date concerning the application of high cervical nerve stimulation for PTH.

## CONCLUSION

PTH is an important public health issue. Head injuries are common, headache is the most common sequelae of head injury, and PTH can be particularly disabling. PTH can contribute to disability, lost productivity, health care costs, and decreased quality of life.

PTH suffers from a lack of clinical trial research, lack of proven treatments, lack of guidelines, and lack of coordinated care or “ownership” of the problem. Persistent PTH needs combined multimodality treatment strategies including non-pharmacological and pharmacological treatment strategies. Electrical neuromodulation appears to be extremely beneficial for highly selected head-injured individuals, suffering from intractable PTH with disabling headaches despite aggressive and comprehensive treatment.

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