Treatment Alternatives for Migraine

Sphenopalatine Ganglion Blocks and Photobiomodulation

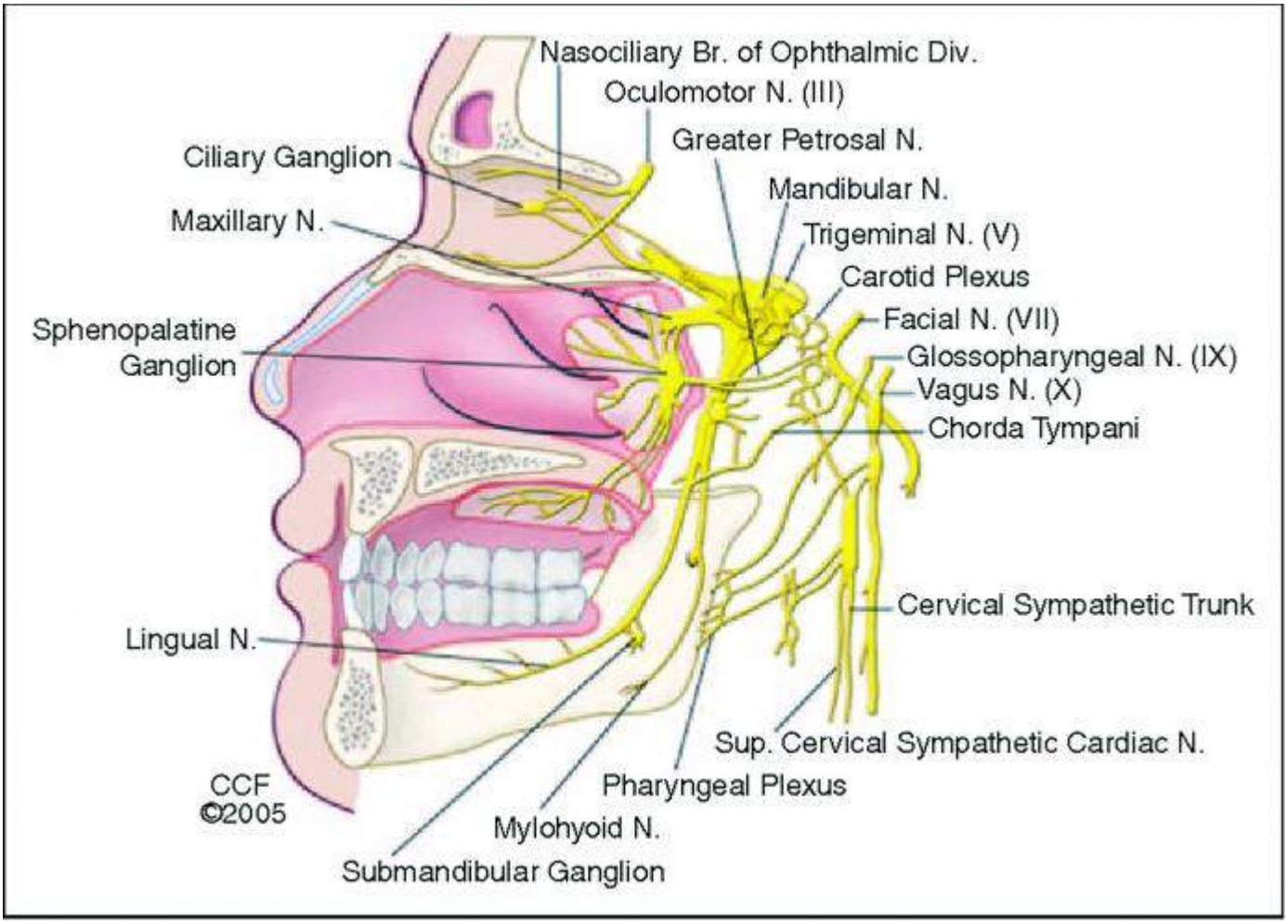
By Shelby J. Doades, ATC, MS (/author/31625/doades) , Timothy J. Demchak, PhD, ATC, LAT (/author/28938/demchak) and Christopher Carraway, DC, DIBCN, CLS (/author/31626/carraway)

Pages 38-41

The Path from Episodic to Chronic Migraine

Although episodic migraine and chronic migraine are common, they represent distinct types of headaches on the migraine pain spectrum.1 Factors involved in the transformation from episodic to chronic migraine include frequency of episodes, failure to optimize acute treatment, overuse of acute migraine medication, lower socioeconomic status, obesity, and being female.1,2 The most common technique for managing these headache conditions is pharmacologic, however, medication overuse is also the most common reason that episodic migraine may evolve into chronic migraine, often resulting in medicine overuse headache (MOH).

According to Lipton, et al,3 patients have reported that their acute treatment of episodic migraine was poorly managed as measured by the Migraine Treatment Optimization Questionnaire, with 6.8% of patients developing chronic migraine within one year compared to 1.9% of patients reporting optimized acute treatment. These results suggested the need for more effective acute treatment strategies to manage symptoms associated with episodic and chronic type migraine. In response, several studies have since shown a potential alternative treatment involving the sphenopalatine ganglion (https://www.practicalpainmanagement.com/treatments/interventional/injections/new-look-sphenopalatine-ganglion-blocks-chronic-migraine) (SPG) to be effective in reducing episodes of chronic migraine (see Figure 1).



(https://www.practicalpainmanagement.com/sites/default/files/imagecache/lightbox-large/images/2019/06/13/Figure%201.jpg)

Figure 1. The sphenopalatine ganglion (SPG). (Image courtesy of authors).

The SPG is the largest neurological ganglion outside the brain, located within the pterygopalatine fossa at the posterior attachment of the middle turbinate. This ganglion has sensory, parasympathetic, and sympathetic components that house the trigeminal nerve, branches of the palatine nerves, and various sympathetic and parasympathetic automatic branches all of which innervate the cranial cavities (eg, nose, mouth) as well as facial areas, and the nasal and pharyngeal glands.4 Based on the SPG's anatomy and physiology, it has become evident that many associated symptoms of chronic migraine may be managed by targeting the SPG using alternative methods that aim to decrease activity in this region.

Migraine may be related, at least in part, to a hyper-excited SPG. Stimulation of the SPG (https://www.practicalpainmanagement.com/resources/news-and-research/sphenopalatine-ganglion-stimulation-possible-tool-cluster-headaches) has been shown to induce a pathophysiological response seen in migraine attacks, including vasodilation of intra- and extra-cranial arteries, release of substance P and neurokinin A, as well as activation of meningeal nociceptors, which may be contributing to the pain.4

Treatment Alternatives

Neurological Blockage of the SPG

Alternative treatments targeting the SPG have been developed as a means of lessening the symptoms associated with migraine. One trialed approach is a neurological blockade at the SPG with bupivacaine using a nasal applicator5,6 and topical lidocaine applied with a deep nasal anesthetic applicator (DNAA).7 Cady, et al, published two studies utilizing the device to deliver bupivacaine to the mucosa of the SPG. The first, primarily a safety study, was designed to determine acute effects. The researchers reported the bupivacaine treatment group (n = 26) decreased from pre-treatment 3.18 ± 2.79 to post-treatment at 15 minutes 2.53 ± 2.61, 30 minutes 2.41 ± 2.61, and 24 hours 2.85 ± 2.74.5 The second study was designed to determine the long-term effects of bupivacaine by delivering a set of 12 treatments over a period of six weeks. Results demonstrated that the bupivacaine treatment group (n = 25) had a significant decrease in the number of headaches in a month from 23.15 ± 5.12 to 17.44 ± 9.08 compared to 24.75 ± 4.35 to 22.82 ± 5.36 in a sham group, which was administered saline. Additionally, the average pain scores reported by the subjects in the prior 24 hours decreased from pre-treatment of 4.92 ± 2.2 to 2.86 ± 2.62 at six months after the last bupivacaine treatment.6

Lee, et al, reported 59 out of 66 cases treated with 26% lidocaine applied with DNAA had an average decrease of 4.9 pain points and 4.2 points at 15 minutes and 60 minutes post-application, respectively.7 The treatment provided rapid relief of the headache pain and decreased activity of the SPG, thereby reducing the pain associated with the migraine.5-7

Similarly, an inhibitory dose of photobiomodulation (PBM) appears to have a similar efficacy in decreasing SPG activity and may reduce migraine pain and frequency by inhibiting nerve conduction of type C pain fibers.8,9

(https://www.practicalpainmanagement.com/sites/default/files/imagecache/lightbox-large/images/2019/06/13/82408819_L.jpg)

Moving away from pharmacologic methods to treating migraine. (Source: 123RF)

Photobiomodulation

Adopted by the North American Association of Photobiomodulation Therapy, photobiomodulation (https://www.practicalpainmanagement.com/pain/myofascial/photobiomodulation-treatment-fibromyalgia) refers to light therapy treatments that utilize non-ionizing light sources in the visible and infrared spectrum.8 PBM is a non-thermal process that involves endogenous chromophores which

elicit photophysical and photochemical events. These events theoretically lead to beneficial therapeutic outcomes, including the alleviation of pain or inflammation and immunomodulation, as well as the promotion of wound healing and tissue regeneration.8 More specifically, PBM emits photons of light that penetrate the skin and stimulate endogenous light receptors, which result in a physiological response. Low doses of PBM stimulate tissue healing and increase blood flow9 while higher doses tend to have an inhibitory effect, which may be used therapeutically to decrease pain.9

For example, low doses of light that are delivered to the tissue stimulate the cytochrome C oxidase (CCO) within the mitochondria, resulting in an increase in adenosine triphosphate (ATP) and a release of nitric oxide (NO) and reactive oxygen species (ROS).9 The ATP provides an increase in energy availability within the cells. When NO is released from CCO and from blood vessels, the result is an increase in ATP production and vasodilation. When ROS is in low concentrations, it activates the transcription factors, which lead to cell proliferation and growth.9

The authors trialed PBM on three patients (ages 42, 53, and 72) with a history of chronic migraine. Each patient had suffered from two to five migraine attacks per week for at least the prior 10 years (see Table I). Each was successfully treated using a PBM protocol to the SPG.

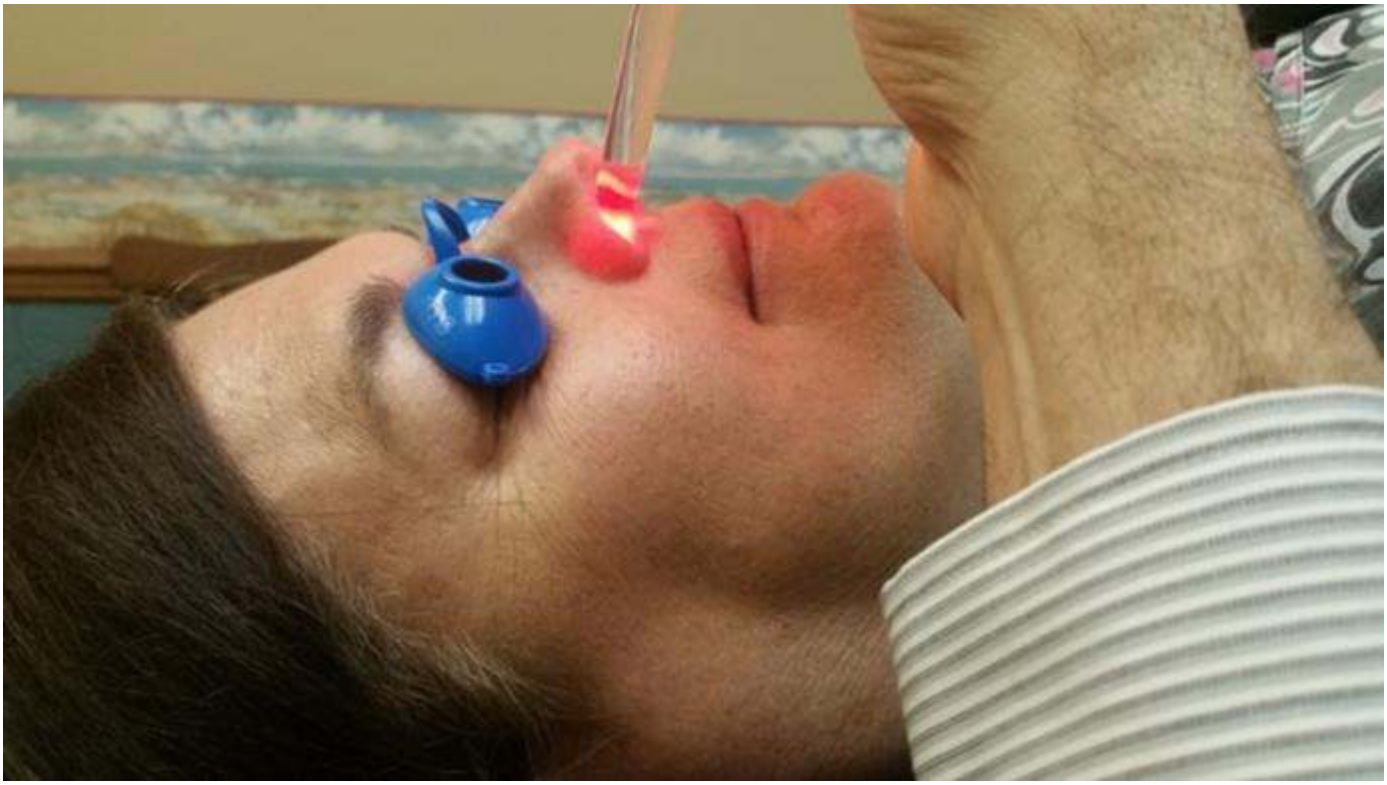
Table I: Mini Case Patient Demographics and Photobiomodulation (PBM) Treatment Protocols.

History	Patient 1	Patient 2	Patient 3
Age/Gender	42-year-old male	53-year-old female	72-year-old female
Migraine History	10 years following skull fracture	30 years since given birth to one of her children	59 years since first menarche
Migraine Frequency	2 per week	2 to 3 per week	3 to 5 per week
Pre-Rx Symptoms	Pain level 8 out of 10; vertigo	Pain level 8 out of 10; aura; mental dullness from acute medications	Pain level 8 out of 10
PBM Treatment Protocol	1 day per week, for 4 weeks (3 total)	2 days per week for 3 weeks; then 1 day per week for 4 weeks (10 total)	2 days per week for 4 weeks; then 1 day per week for 4 weeks (12 total)
Post-Treatment Symptoms	0 migraine 2 weeks post-Rx	0 migraine attacks during treatment and at 90-day follow-up	0 migraine attacks during treatment

(https://www.practicalpainmanagement.com/sites/default/files/imagecache/lightbox-large/images/2019/06/13/Table%201.jpg)

Initial reported pain levels ranged from 8 to 10 out of a 10-point pain scale. All three patients completed daily activities with difficulties due to frequent and painful symptoms. All patients had previously attempted pharmacological methods of treatment with little to no relief, or with additional side effects from MOH that hindered daily functioning.

Each PBM treatment consisted of applying a laser puncture utility probe attached to a PBM-transducer (Multi Radiance Medical) that delivered the photons to the SPG. The probe was placed just inside each nostril pointing toward the posterior nasal cavity where the SPG is located (see Figure 2). The treatment frequency and number of overall treatments were tailored to each patient's responsiveness.



(https://www.practicalpainmanagement.com/sites/default/files/imagecache/lightbox-large/images/2019/06/13/Figure%202.jpg)

Figure 2. Inhibitory photobiomodulation treatment to the sphenopalatine ganglion with probe (image courtesy of authors).

Each treatment lasted 180 seconds per nostril (23.9 joules, 0.0382 watts, 6.87J/chronic). The device characteristics were as follows: wavelength super pulsed laser 905 nm; infrared 875 nm; Red 670 nm; total power, 25W, SPL variable frequency: 1000 Hz and beam spot size 0.4. The patients were evaluated for migraine frequency and intensity both pre- and post-treatment, and throughout the duration of the treatment.

Patient 1: This 42-year old male developed a chronic migraine condition following a traumatic head injury, resulting in a skull fracture. His regimen encompassed three PBM treatments over a 4-week period. After the first treatment, the patient experienced no migraine for 2 weeks. His reported migraine pain decreased from 8 out of 10 to a 0 out of 10 after each treatment. After the full course, the patient reported no migraine for another 2 weeks and self-discharged from our care (follow-up was not possible).

Patient 2: A 53-year-old female was scheduled to receive a course of six PBM treatments over 21 days. The patient reported reduced frequency and intensity of migraine with aura after the first six treatments. This patient did not miss any work during the treatment period. It is worth noting that prior to starting the PBM treatments, Patient 2 had missed work due to pain intensity of her migraine and what she reported as mental dullness as a result of the medication used to control her migraine symptoms. Due to the decrease in frequency and intensity of her migraine attacks, PBM treatments were reduced to one per week for 4 weeks. Patient 2 was migraine-free at discharge after 8 weeks of total treatment. At 90-day follow-up, the patient reported that she had not experienced a post-treatment migraine.

Patient 3: A 72-year-old female underwent a course of eight PBM treatments over 4 weeks (two applications per week). Patient 3 reported that she was migraine free for 10 days after the month-long treatment was completed. She was prescribed a second round of treatments, which were then reduced to weekly for another 4 weeks. The patient reported being migraine-free during the continued treatment period. There was no 90-day follow-up for this patient.

Discussion and Steps Forward

Two of the main factors that may cause episodic migraine attacks to become chronic are medication overuse and improper care for acute attacks.1-3 As demonstrated in the three cases herein, PBM treatments to the SPG were shown to be effective in decreasing pain ratings from 8 out of 10 to 0 out of 10 after each treatment. If PBM could be used effectively to treat episodic migraine, patients may not overuse medications, which may ultimately prevent the transition of episodic to chronic migraine.

Overall, the PBM treatments described herein were deemed successful in treating chronic migraine (see Table I for details). Patient 1 started with two migraine attacks per week and decreased his episodes to zero attacks per week after a course of three treatments over 4 weeks. Patient 2 reduced her migraine frequency from two to three per week to zero after 10 treatments over 12 weeks. Patient 3, who had

previously experienced three to five migraine attacks per week for 59 years, reduced her attack frequency to zero migraine attacks per week after 12 treatments over 8 weeks. None of the patients reported any side effects and tolerated the treatments well.

Similar to neurologic blocks of the sphenopalatine ganglion, the response to PBM is bi-phasic, stimulatory or inhibitory, and dose dependent.9 There has been strong evidence supporting PBM inhibition of acute, chronic, and neurological pain.10 As noted, light may reduce the formation of inflammatory proteins associated with pain including prostaglandin, cox 2 mRNA, and TNF α.10 Additionally, PBM works to inhibit nerve conduction along the AΔ and C nerve fibers, which are the main nerve fiber types that conduct pain.10

It appears that the SPG and associated nerves are hyperactive during migraine attacks, as suggested by Khan, et al.4 An inhibitory dose of PBM seems to restore the SPG and associated nerves back to their normal physiological levels. A similar occurrence was reported by Cady, et al,5-6 and Lee, et al,7 after treating migraine (with bupivacaine and lidocaine, respectively) applied to the posterior nasal cavity directed at the SPG. There is some evidence that the SPG may also be associated with refractory chronic post-traumatic headaches.10 For example, Sussman, et al, successfully treated a post-concussion headache utilizing intranasal lidocaine application to the SPG.11

In this case presentation, use of PBM treatment reduced migraine frequency to zero episodes per week in patients with a 10-year or greater history of migraine for whom medication failed to manage symptoms effectively. Due to a decrease in pain and episode occurrence, all three patients were able to improve their daily function following completion of individualized PBM treatment regimen to block the SPG. With a growing demand for non-pharmacological treatments for migraine pain, photobiomodulation may be a noninvasive therapeutic option for chronic migraine. To demonstrate the efficacy of this treatment protocol, large randomized control trials should be completed to confirm validity and long-term effects.

View Sources (/pain/headache/treatment-alternatives-migraine#fieldset)

Last updated on: June 20, 2019

Continue Reading:

Tension-Type Headache: Evidence for Trigger Points

Oral and Maxillofacial Pain
TMJ
Tooth Pain
Rheumatologic and Myofascial Pain
Fibromyalgia
Inflammatory Arthritis
Lupus and Other Autoimmune Disorders
Lyme and Other Infectious Diseases
Osteoarthritis
Plantar Fasciitis
Spine Pain
Discogenic Pain
Facet Syndrome
Myofascial Back Pain
Radiculopathy
Trauma
Other Types of Pain
Abdominal and Pelvis Pain
Brain Injury
Ehlers-Danlos Syndrome
General Musculoskeletal Pain
Pain Co-morbidities
Pain Treatments
Addiction Medicine
Drug Monitoring/Screening
Medication-Assisted Treatment (MAT)
Opioid Use Disorder (OUD)
Complementary Treatments
Acupuncture
Biobehavioral
Homeopathy
Lasers
Magnets
Prolotherapy
Interventional Pain Management
Ablations
Injections
Pumps
Stimulators
Manipulation and Massage
Massage
Osteopathic
Pharmacological
Marijuana/Cannabis
Non-opioids/OTC
Opioids
Tapering
Psychological
Biofeedback

Cognitive-behavioral Therapy

Rehabilitation

Exercise

Physical Therapy

Resources

Clinical Practice GuidelinesDiagnostic TestsEthics and LegalClinical ResourcesHospice/Palliative CareNews and ResearchOpioid Prescribing and Monitoring - Second EditionOpioid Prescribing and Monitoring - First EditionPractice Management

Literature Review

Past Issues

FOR PATIENTS

Types of Pain

Acute Pain

Cancer Pain

Headache

Cluster Headache

Migraine

Post-trauma Headache

Tension Headache

Neuropathic Pain

Oral and Maxillofacial Pain

Rheumatologic and Myofascial Pain

Spine Pain

Other Types of Pain