



BlueCross BlueShield
of Alabama

Name of Policy:

Treatment of Cervicogenic Headache and Occipital Neuralgia

Policy #: 314
Category: Surgery

Latest Review Date: October 2018
Policy Grade: B

Background/Definitions:

As a general rule, benefits are payable under Blue Cross and Blue Shield of Alabama health plans only in cases of medical necessity and only if services or supplies are not investigational, provided the customer group contracts have such coverage.

The following Association Technology Evaluation Criteria must be met for a service/supply to be considered for coverage:

- 1. The technology must have final approval from the appropriate government regulatory bodies;*
- 2. The scientific evidence must permit conclusions concerning the effect of the technology on health outcomes;*
- 3. The technology must improve the net health outcome;*
- 4. The technology must be as beneficial as any established alternatives;*
- 5. The improvement must be attainable outside the investigational setting.*

Medical Necessity means that health care services (e.g., procedures, treatments, supplies, devices, equipment, facilities or drugs) that a physician, exercising prudent clinical judgment, would provide to a patient for the purpose of preventing, evaluating, diagnosing or treating an illness, injury or disease or its symptoms, and that are:

- 1. In accordance with generally accepted standards of medical practice; and*
- 2. Clinically appropriate in terms of type, frequency, extent, site and duration and considered effective for the patient's illness, injury or disease; and*
- 3. Not primarily for the convenience of the patient, physician or other health care provider; and*
- 4. Not more costly than an alternative service or sequence of services at least as likely to produce equivalent therapeutic or diagnostic results as to the diagnosis or treatment of that patient's illness, injury or disease.*

Description of Procedure or Service:

Headache

Headaches are common neurologic disorders and are among the top reasons that patients seek medical care. Headaches affect approximately 50% of the general population in a given year and over 90% of people have a lifetime history of headache. Several treatments or procedures for headache (e.g., chronic migraine, tension-type headache, chronic cluster or cervicogenic headache) and occipital neuralgia have been introduced with varying levels of success. The consensus on standard of care does not exist due to the variability in patient selection and clinical outcomes.

The International Headache Society (IHS), through expert consensus, has created a headache classification system to help diagnose and classify headaches. The IHS criteria are regarded as the gold standard for diagnosis of all types of headaches. The first edition was published in 1988, the second edition was published in 2004 and the third edition was released in early 2018.

The third edition classifies headaches into 3 major groups:

- The first group, the primary headaches, includes migraine, tension-type headache, cluster headaches, other trigeminal autonomic cephalgias, and other primary headaches;
- The second group, the secondary headaches, includes headaches attributed to head and/or neck trauma, cranial or cervical vascular disorder, non-vascular intracranial disorder, a substance or its withdrawal, infection, disorder of homeostasis, disorder of cranial, cervical or facial structures, or psychiatric disorder; and
- The third group includes cranial neuralgias, central or primary facial pain, and other headache disorders.

Primary Headache Disorders

Migraines

Migraines are the second-most common headache disorder with 1-year prevalence of migraine in the United States of approximately 12% and can be categorized by headache frequency.

Migraines are characterized by severe pain on one or both sides of the head, an upset stomach, and, at times, disturbed vision.

According to the Third Edition of the International Headache Classification (ICHD-3), migraine without aura (previously known as common migraine) is defined as at least 5 attacks per month meeting other diagnostic criteria. Chronic migraine is defined as attacks on at least 15 days per month for more than 3 months, with features of migraine on at least 8 days per month.

A variety of medications are used to treat acute migraine episodes. They include medications taken at the onset of an attack to abort the attack (triptans, ergotamines), and medications to treat the pain and other symptoms of migraines once they are established (nonsteroidal anti-inflammatory drugs, narcotic analgesics, antiemetic's). Prophylactic medication therapy may be appropriate for people with migraines that occur more than 2 days per week. In addition to medication, behavioral treatments such as relaxation and cognitive therapy are used in the management of migraine headache. Moreover, botulinum toxin type A injections are a U.S. Food and Drug Administration (FDA) –approved treatment for chronic migraine.

Tension-Type Headaches (TTH)

Tension-type headaches (TTH) have a prevalence range from 30% to 78%. They are diagnosed when patients report at least 2 of the following characteristics: bilateral headache location, non-pulsating pain of mild to moderate intensity and headache not aggravated by physical activity. Tension-type headaches tend to be mild in nature and are frequently self-diagnosed and self-treated.

There are three types of TTH: Infrequent episodic, frequent episodic and chronic. Infrequent episodic headache is identified in patients who experience headache episodes less than 1 day per month. Frequent episodic TTH is classified as patients whom experience headache episodes 1 to 14 days per month and chronic TTH is diagnosed in patients who experience headache episodes for 15 days or more per month. Treatment for episodic TTH consists of simple analgesics, simple analgesics with caffeine, parenteral treatments, triptans, and muscle relaxants. Chronic TTH treatment consists of tricyclic antidepressants, behavioral therapies and physical therapy.

Cluster Headaches

Cluster headaches are less common than tension or migraine headaches, with an estimated prevalence of 0.1% of the population. Cluster headaches are characterized by severe unilateral orbital, supraorbital and/or temporal pain that also includes other symptoms in the eye and/or nose on the same side such as rhinorrhea, eyelid edema, or drooping. Cluster headaches occur in a series lasting for weeks to months. Approximately 10-15% have cluster, periods separated by remission periods which usually lasts for months or years. Due to the severity of pain associated with cluster headaches, patients may seek emergency treatment.

Severe acute cluster headaches may be treated with abortive therapy including breathing 100% oxygen and triptan medications. Other medications used to treat cluster headaches include steroids, calcium channel blockers and nerve pain medications.

Occipital Neuralgia

Occipital neuralgia is a specific type of headache that is located on one side of the upper neck, back of the head, and behind the ears, and sometimes extending to the scalp, forehead, and behind the eyes. The pain, which may be piercing, throbbing, or electric-shock-like, follows the course of the greater and lesser occipital nerves. Occipital neuralgia is believed to occur due to pressure or irritation to the occipital nerves, which may result from injury, entrapment by tight muscles, or inflammation.

Occipital neuralgia results in posterior occipital headaches when pressure occurs on the greater and/or lesser occipital nerves. It may be classified as intermittent (e.g., paroxysmal) or continuous, with an acute or chronic nature. Paroxysmal occipital neuralgia is pain that occurs only in the distribution of the greater occipital nerve. The attacks are unilateral, with sudden and severe pain prescribed as sharp, twisting or lancing. The attacks may occur spontaneously but can be provoked by specific maneuvers applied to the back of the scalp or neck regions.

Acute continuous occipital neuralgia attacks can last for many hours, with duration of up to 2 weeks before remission. This type is not usually associated with radiating facial symptoms. In chronic continuous occipital neuralgia, the attacks are accompanied by localized muscle spasms.

The pain is described as steady, sharp or aching, with referred pain into facial areas, especially above and behind the orbit. Unilateral pain is more common, but it can also be bilateral. Scalp tenderness is common. Pain may be increased or be provoked with postures that occur in reading or sleeping positions or with hyperextension or rotation of the head to the involved side. Physical findings include pain with palpation of the occipital nerves. Occasionally, there is hyperesthesia or allodynia in the distribution of the occipital nerve. Local muscle spasm is frequently found with palpable trigger points and taut bands. Cervical range of motion may be restricted, and neurological exams are typically normal. An anesthetic block given at the site of maximal tenderness or at the site of the occipital groove confirms the diagnosis of occipital neuralgia if there is pain relief. Treatment may include massage, rest, muscle relaxants, nerve blocks, or injection of steroids directly into the affected area.

The IHS considers the diagnostic criteria for occipital neuralgia as follows:

- Paroxysmal, stabbing pain, with or without persistent aching between paroxysms, in the distribution of the greater, lesser, and/or third occipital nerves.
- Tenderness over the affected nerve.
- Pain eased temporarily by local anesthetic block of the nerve.

Secondary Headache Disorder

Cervicogenic Headache

Cervicogenic headache is a headache that is secondary to a disorder of the cervical spine. The pain may be referred from facet joints, intervertebral discs, or soft tissue. The pain is constant and may be aggravated by movements of the neck or pressure to certain areas on the neck. The first three cervical spinal nerves can refer pain to the head. The C1 suboccipital nerve innervates the atlanto-occipital joint; the C2 spinal nerve and the C3 dorsal ramus have close proximity to and innervate the C2-C3 facet joint. The C2-3 facet joint is the most frequent source of a cervicogenic headache. A diagnosis of a cervicogenic headache may be confirmed by an anesthetic block of the lateral atlanto-axial joint, the C2-3 facet joint, or the C3-4 facet joint.

Cervicogenic headache and occipital neuralgia are syndromes whose diagnosis and treatment have been reported as controversial in the medical literature due to lack of expert consensus regarding their etiology and treatment. The terminology refers to specific types of headache thought to arise from impingement or entrapment of the occipital nerves and/or the upper spinal vertebrae. Compression and injury of the occipital nerves within the muscles of the neck and compression of the second and third cervical nerve roots are generally thought to be responsible for the symptoms including unilateral and occasionally bilateral head, neck, and arm pain. The convergence of the afferents of the upper 3 cervical spinal nerves is thought to be responsible for this head pain that arises from the neck.

Generally accepted causes of head pain originating in the neck include:

- Developmental abnormalities, tumors, ankylosing spondylitis, rheumatoid arthritis, and osteomyelitis.

Controversial causes include:

- Cervical disc herniation, degenerative disc disease, and whiplash injuries.

The prevalence of cervicogenic headache in the general population is about 2.2% and is 4 times more prevalent in women. The clinical features of cervicogenic headache may mimic those associated with primary headache disorders, such as tension-type headache, migraine, or hemicrania continua, so it may be difficult to distinguish among headache types. Cervicogenic headache is characterized by continuous, unilateral head pain radiating from the occipital areas to the frontal area, with associated neck pain and ipsilateral shoulder or arm pain. The headache is non-throbbing and of moderate intensity. It is described as a dull, boring, dragging pain that can fluctuate in intensity. The headache may last from a few hours to several days and, in some cases, for several weeks. The pain is exacerbated by neck movements and is usually caused by neck trauma. Associated symptoms, such as nausea, photophobia, phonophobia, dizziness, blurred vision, and dysphagia may be present, but are generally not pronounced.

The IHS considers the diagnostic criteria for cervicogenic headache as follows:

- Pain referred from a source in the neck and perceived in one or more regions of the head and/or face.
- Clinical, laboratory and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck, generally accepted as a valid cause of headache.
- Evidence that the pain can be attributed to the neck disorder or lesion, based on either clinical signs that implicate a source of pain in the neck or abolition of headache following diagnostic nerve block.
- Pain resolving within three months after successful treatment of causative disorder or lesion.

Sjaastad et al identified another type of CGH with bilateral head and neck pain, aggravated by neck positions and specific occupations such as: hair-dressing, carpentry, and truck/tractor driving. The neck pain precedes or co-exists with the headache, and is aggravated by specific neck movements or sustained postures.

In 2011, Vincent et al described several factors to differentiate CGHs, including:

- Unilateral pain with a facet 'lock' irradiating from the back of the head
- Evidence of cervical dysfunction presenting during manual examination
- May occur with trigger point palpation in the head or neck
- Aggravated by sustained neck positions
- Normal imaging

Because the diagnosis of CGH is relatively new, its particular etiology remains unclear. Treatment of cervicogenic headache (CGH) includes nerve blocks, physical therapy, and exercise.

Policy:

The following treatments for **chronic headaches, including cervicogenic headache, occipital neuralgia, and migraine do not meet** Blue Cross and Blue Shield of Alabama's medical criteria for coverage and are considered **investigational**:

- Discectomy and spinal fusion
- Dorsal column stimulation
- Electrical stimulation of occipital nerve
- Ganglionectomy
- Implantable infusion pumps (refer to Medical Policy #442 for additional information)
- Injection of anesthetic
- Nerve root decompression
- Neurectomy
- Neurolysis of the great occipital nerve with or without section of the inferior oblique muscle
- Occipital nerve neurolysis
- Pulsed radiofrequency
- Radiofrequency denervation of cervical facet joints
- Radiofrequency ablation of the planum nuchale
- Radiofrequency ablation of peripheral nerves
- Rhizotomy
- Sphenopalatine Ganglion block
- Surgical release of the lesser occipital nerve within the trapezius

The safety and effectiveness of these treatments for these indications have not been established.

Blue Cross and Blue Shield of Alabama does not approve or deny procedures, services, testing, or equipment for our members. Our decisions concern coverage only. The decision of whether or not to have a certain test, treatment or procedure is one made between the physician and his/her patient. Blue Cross and Blue Shield of Alabama administers benefits based on the member's contract and corporate medical policies. Physicians should always exercise their best medical judgment in providing the care they feel is most appropriate for their patients. Needed care should not be delayed or refused because of a coverage determination.

Key Points:

The most recent literature update was performed through July 9, 2018.

Evidence reviews assess the clinical evidence to determine whether the use of a technology improves the net health outcome. Broadly defined, health outcomes are length of life, quality of life, and ability to function including benefits and harms. Every clinical condition has specific outcomes that are important to patients and to managing the course of that condition. Validated outcome measures are necessary to ascertain whether a condition improves or worsens; and

whether the magnitude of that change is clinically significant. The net health outcome is a balance of benefits and harms.

To assess whether the evidence is sufficient to draw conclusions about the net health outcome of a technology, 2 domains are examined: the relevance and the quality and credibility. To be relevant, studies must represent one or more intended clinical use of the technology in the intended population and compare an effective and appropriate alternative at a comparable intensity. For some conditions, the alternative will be supportive care or surveillance. The quality and credibility of the evidence depend on study design and conduct, minimizing bias and confounding that can generate incorrect findings. The randomized controlled trial (RCT) is preferred to assess efficacy; however, in some circumstances, nonrandomized studies may be adequate. RCTs are rarely large enough or long enough to capture less common adverse events and long-term effects. Other types of studies can be used for these purposes and to assess generalizability to broader clinical populations and settings of clinical practice.

Treatment Modalities for Headache and Occipital Neuralgia

Local Injection Therapy

One commonly used diagnostic procedure for pain relief is the use of local injected anesthetics, with or without a corticosteroid, to block the affected nerves. These injections have been used as therapeutic treatment measures for pain relief, although the duration of pain relief varies from hours to months. However, the scientific evidence regarding injection therapy or percutaneous nerve block for occipital neuralgia and cervicogenic headache has been limited.

Freund et al (2000) reported on a randomized controlled study to evaluate therapy with botulinum toxin A (BTX-A) as a treatment for cervicogenic headache. There were 26 chronic headache patients: one half received botulinum toxin A injections and one half received saline injections. The results showed that at 4 weeks, the patients who received BTX-A had significantly lower pain scores than they had prior to treatment. However, patients who received the placebo injection also had lower pain scores. There was no statistically significant difference between the two groups. The results suggest a substantial placebo effect or a nonspecific effect of injection.

Bogduk (2004) reported that a response to diagnostic blockade of cervical structures or nerves is an appropriate diagnostic criterion that establishes headaches arise in the neck. He reported that many, but not all, patients can be temporarily relieved of pain by blocking the greater occipital nerve, the C-2 spinal nerve, or the C2-3 zygapophysial joint. The author noted that the available studies have not used controlled blocks to establish the validity of the response, and the studies do not provide conclusive evidence of a cervical source of pain for cervicogenic headache.

Naja et al (2006) reported on a randomized controlled trial that evaluated the effectiveness of nerve stimulator guided occipital nerve blockade to treat cervicogenic headache. There were 50 patients who were randomly divided into 2 groups of 25 patients each. All patients in both groups received greater and lesser occipital blocks, but only 16 patients in each group received facial nerve blockade in association with the occipital blocks. The control group received injections of an equivalent volume of normal saline. Pain was assessed using the visual analog scale (VAS) and the total pain index (TPI). There were 47 patients at follow-up. The results

showed the anesthetic block was effective in reducing the VAS and TPI by approximately 50% from baseline values. Analgesic consumption, duration of headache and its frequency, nausea, vomiting, photophobia, phonophobia, decreased appetite, and limitations in functional activities were significantly less in block group compared to control group. The nerve stimulator-guided occipital nerve blockade significantly relieved cervicogenic headache and associated symptoms at 2 weeks following injection. The authors reported the limitations of this study were the short duration of follow-up and the difficulty in blinding when numbness resulted in patients who received the anesthetic block.

Naja et al (2006) also reported on a follow up study of these patients. They reported that 41/47 patients (87%) required more than one injection to achieve 6-month pain-relief period. For every 3 years of headache history, the outcomes demonstrated that a patient needed one additional injection to the basic injection.

Kapural et al (2007) reported on a retrospective trial of 6 patients with severe occipital neuralgia who had conservative and interventional therapies, including oral antidepressants, membrane stabilizers, opioids, and traditional occipital nerve blocks without traditional relief. The group then underwent occipital nerve blocks using botulinum toxin type A. In 5/6 patients, there were significant decreases in pain visual analog scale (VAS) scores and improvement in pain disability index (PDI) at 4 weeks follow-up. The duration of pain relief increased as compared to diagnostic 0.5% bupivacaine block (median 16 weeks vs. median 2 weeks). Following block resolution, the average pain scores and PDI returned to similar levels as before the botulinum toxin block. This was a small study group.

Sphenopalatine Ganglion Block

Sphenopalatine ganglion (SPG) nerve blocks are a proposed treatment option for chronic migraines and some severe non-migraine headaches. The SPG is a group of nerve cells that is located behind the bony structures of the nose. The nerve bundle is linked to the trigeminal nerve, the primary nerve involved in headache disorders. The SPG has both autonomic nerves, which in this case are associated with functions such as tearing and nasal congestion, and sensory nerves, associated with pain perception. SPG nerve blocks involve topical application of local anesthetic to mucosa overlying the SPG. The rationale for using SPG blocks to treat headaches is that local anesthetics in low concentrations could block the sensory fibers and thereby reduce pain while maintaining autonomic function. Several catheters approved by the Food and Drug Administration are available for the SPG blocking procedure.

The currently proposed procedure for SPG nerve blockade is to insert a catheter intranasally that is attached to a syringe carrying local anesthetic (e.g., lidocaine or bupivacaine). Once the catheter is in place, the local anesthetic is applied to the posterior wall of the nasal cavity and reaches the SPG. Some form of SPG blocking procedure has been used for many years. Originally, SPG blocks were done by inserting a cotton-tipped applicator dabbed with local anesthetic into the nose; this technique may be less accurate and effective than the currently proposed procedure. Another variation is to insert a needle into the cheek and inject local anesthetic but this no longer appears to be used since it is more invasive and can be painful. Neurostimulation of the SGB and SGB blockade with radiofrequency lesioning have been used outside of the United States but these treatments are not FDA-cleared or approved.

Three catheter devices are currently commercially available in the United States for performing SPG blocks. The catheters have somewhat different designs but all are attached to syringes that contain local anesthetic. The catheters are inserted intranasally and once in place, the local anesthetic is applied through the catheter. With 2 of the 3 commercially available catheters, the SpenoCath® or Allevio™, patients are positioned on their back with their nose pointed vertically and their head turned to the side. With the Tx360® device, patients remain seated. Pulsed radiofrequency modulation is also being used to treat migraines.

The company marketing the Tx360® device is proposing its use in the context of a protocol called the MiRx™ protocol. This 2-part protocol includes a medical component for immediate pain relief and a physical component to reduce headache recurrences. The medical component involves clinical evaluation and, if the patient is considered eligible, an SPG block procedure. The physical component can include any of a number of approaches such as physical therapy, ergonomic modifications, massage and dietary recommendations.

The optimal number and frequency of SPG treatments is unclear. Information from the American Migraine Foundation states that the procedure can be repeated as often as needed to control pain. An RCT described a course of treatment for migraines consisting of SPG blocks twice a week for 6 weeks (total of 12 treatments).

Chronic Migraine Treatment with SPG Block

The published literature on SGB blocks to treat chronic migraine consists of 1 double-blind placebo-controlled RCT and a small case report with 3 patients.

Findings of the RCT were published in two 2015 publications by Cady et al. The first publication reported on the primary outcome measure and key secondary outcomes, and the subsequent publication reported on supplemental secondary outcomes and longer term follow-up. The trial included patients who met International Classification of Headache Disorders (ICHD-2) diagnostic criteria for chronic migraine headache and had CM for at least 3 months. Patients could use concomitant headache medication, but needed to agree not to make changes in medication use during the study period. Following an initial 28-day baseline period to confirm the diagnosis of CM, patients were randomized 2:1 to receive treatment with 0.5% bupivacaine or saline (placebo) applied using the Tx360® device. Patients received a series of 12 treatments, 2 treatments a week for 6 weeks. The primary outcome was change in pain severity, measured by a 0 to 10 numeric rating scale (NRS). Pain severity was assessed 15 minutes, 30 minutes and 24 hours after each treatment. Key secondary outcome measures were the Patient's Global Impression of Change (PGIC), the Headache Impact Test (HIT-6) questionnaire and patient satisfaction with treatment. In addition, patients kept headache diaries throughout the study.

Forty-one patients met eligibility criteria and had CM diagnoses confirmed during the baseline period. These patients were randomized to receive application of bupivacaine (n=27) or placebo (n=13). One patient in the placebo group withdrew consent, and 3 patients were excluded from analysis due to protocol violations, leaving 38 patients in the final dataset. This included 26 in the bupivacaine group and 12 in the placebo group. Mean baseline scores on the NRS were 4.8 in the bupivacaine group and 4.5 in the placebo group. When pooling findings for all treatments, patients in the bupivacaine group reported a significantly greater reduction in the NRS than the

placebo group at 15 minutes, 30 minutes, and 24 hours after treatment. An analysis also found significantly lower PGIC scores in the bupivacaine than saline groups at 30 minutes and 24 hours posttreatment. No statistically significant differences between group differences were found in HIT-6 scores or in average acute medication use. Only 1 serious adverse event (SAE) was reported and it was not treatment-related.

Another 2015 publication by Cady et al on this study reported on 1- and 6- month follow-up results and on supplemental secondary end points. To control for multiple comparisons, the cutoff for statistical significance for the supplemental secondary end points was p less than 0.01. There were not statistically significant differences between groups in the reported supplementary secondary outcomes. These outcomes include the number of headache days per month, the mean pain score and quality of life measures. A post hoc power analysis revealed that the study was underpowered to detect significant differences in secondary outcomes. Some results were suggestive of a possible long-term effect (e.g. the bupivacaine group had a lower, albeit nonsignificant number of headache days in the month posttreatment than the placebo group (17 vs 23)). However, a study with a larger sample size would be needed to confirm whether or not 1- or 6-month results are significantly better after bupivacaine versus placebo treatment.

Severe Acute Headache Treated with SPG Block in an Emergency Setting

The published literature on SGB blocks to treat severe acute headache consists of 1 double-blind placebo-controlled RCT. The study included patients between the ages of 18 and 65 who presented to the emergency department with a frontal-based crescendo-onset headache and a negative neurological examination. The study focused on frontal-based headaches because these were considered most likely to respond to SPG blocks. Headaches were not classified into specific types but patients with sudden-onset headache were excluded. Ninety-three patients met eligibility criteria and were randomized 1:1 to receive treatment with bupivacaine 0.5% ($n=45$) or a saline placebo ($n=48$) applied using the Tx360® device. The intervention consisted of 1 treatment session. The primary outcome was a 50% absolute pain reduction on a 100-mm visual analog scale (VAS) 15 minutes post-treatment. Four patients, 2 in each group, withdrew before receiving the intervention and 2 were deemed ineligible after randomization. Thus, 41 patients in the bupivacaine group and 46 in the placebo group were included in the primary analysis.

For the primary outcome, 20 (49%) patients in the bupivacaine group and 19 (41%) patients in the placebo group had at least a 50% reduction in the mean VAS score. The difference between groups was not statistically significant (difference, 7.5%; 95% CI, -13% to 27%). Secondary outcomes including at least a 19mm reduction in VAS, percent of patients who were headache-free 15 minutes post-intervention and percent of patients who were nausea-free 15 minutes post-intervention, also did not differ significantly between groups. Seventy-six (88%) patients were available for follow-up after 24 hours. The percent of patients headache free at 24 hours was significantly higher in the bupivacaine group ($n=26$ [72%]) than the placebo group ($n=19$ [48%]; difference, 25%; 95% CI, 2.6 to 44%). No SAEs were reported in either group. The authors stated that, in retrospect, outcome assessment at 1 hour after treatment would have been useful since headache relief at 1 hour, but not at 24 hours, is clinically relevant for ED headache patients.

Cluster Headache treated with SPG Block

No RCTs or non-randomized controlled studies were identified that evaluated intranasal SPG blocks for treating cluster headache. Two case series in patients with chronic drug-resistant cluster headache (CH) were published by a research group in Milan, Italy. Both studies used a needle (20-gauge in 1 study and 18-gauge in the other) under endoscopic control to inject a mixture of local anesthetics and steroid as close as possible to the SPG. The mixture consisted of triamcinolone acetonide (40mg), 1% bupivacaine (4mL) and 2% mepivacaine with 1/100,000 adrenaline (2mL). The earlier study, published in 2006 by Felisati et al included 21 patients who received between 2 and 4 total treatment sessions, provided 1 week apart. Including 1 patient in whom the treatment could not be applied, 9 (45%) experienced no efficacy, 3 (15%) experienced a partial benefit and 8 (40%) experienced a complete temporary benefit. In the 8 patients who had complete disappearance of attacks, the benefit lasted between 2-4 weeks in 3 patients, 3-6 months in 3 patients and 12-24 months in 2 patients. Four patients (19%) experienced treatment-related complications which consisted of 1 case of marked nasal epistaxis 3 days after the procedure and 3 cases of temporary diplopia.

In 2010, Pipolo reported on 15 patients who received 3 treatments a mean of 3 days apart. Eight of the 15 patients (53%) experienced complete remission of CH symptoms. Three of these (20%) continued to be in remission at last follow-up (mean: 18 months). One patient (7%) experienced partial benefit and 6 (40%) reported either no benefit or a benefit for less than 2 weeks. Three patients (20%) experienced complications including 2 cases of severe epistaxis and 1 reduced buccal opening that resolved after 5 months.

Peripheral Nerve Electrical Stimulation

Another proposed treatment method for chronic intractable headaches is the use of peripheral nerve electrical stimulation, either by the percutaneous route or by an implantable electrical stimulator. Once the electrodes are in place, they are turned on to administer a weak electrical current to the nerve. The patient experiences this as a pleasant tingling sensation. By stimulating non-painful sensory pathway, the electrical current tricks the brain into turning off (or significantly attenuating) the painful signals. In this manner, pain relief occurs.

Surgical Treatment for Cervicogenic Headache or Occipital Neuralgia

Jansen (2000) reported the results of 3 different surgical treatments in 102 patients with cervicogenic headache that had been non-responsive to physical or drug therapy. A group of 38 patients were treated with C2 ganglionectomy, and 64 patients with demonstrable spinal structural abnormalities were treated with dorsal or ventral spinal decompression and fusion. About 80% of surgically treated patients were relieved of pain. About 15% of patients had 60-80% relief of pain and about 6% of patients had no relief of pain. The mean duration of pain relief varied: 5 months for dorsal decompression, 14 months for ventral decompression, and 44 months for C2 ganglionectomy. About 80% of the surgically treated patients were relieved of pain or improved during a long period of follow up. The recurrence of degenerative alterations with new irritation from pain-conducting structures is thought to be responsible for the recurrence of headache. Further surgical approaches for the treatment of patients with the recurrence of pain are discussed.

Kapoor et al (2003) reported on a retrospective study of 17 patients with occipital neuralgia who underwent CT fluoroscopy-guided C2 or C3 nerve root blocks and had positive results. All 17 patients then underwent unilateral (n = 16) or bilateral (n = 1) intradural dorsal rhizotomies. Immediately after surgery, all patients had complete relief from pain. Patients were followed a mean of 20 months. At follow up, 11 patients (64.7%) had complete relief of symptoms; two (11.8%) had partial relief; and four (23.5%) had no relief. There were seven of eight (87.5%) patients without prior surgery who had complete relief of symptoms and four of nine (44.4%) patients with a history of prior surgery who had relief. Eight of 16 (50%) patients felt they were more active and functional after surgery, and 25% felt they were either unchanged or less functional than before surgery. There was a trend toward better response to rhizotomy in patients without prior head or neck surgery. The study was limited by its size and lack of control group.

Gille et al (2004) reported on a retrospective study of 10 patients who had surgery for greater occipital neuralgia, which consisted of neurolysis of the greater occipital nerve and section of the inferior oblique muscle. The average age of the patients was 62 years and the average follow up was 37 months. The results showed anatomic anomalies in 3 patients (i.e., hypertrophy of venous plexus around C2, nerve penetration of the inferior oblique muscle, and degenerative C1-C2 osteoarthritis). The mean VAS score was 80/100 before surgery and 20/100 at the last follow-up. The consumption of analgesics decreased and 7/10 patients was satisfied with the operation.

Stovner et al (2004) reported on a randomized, controlled study of 12 patients with unilateral cervicogenic headache. The patients were randomized to receive radiofrequency neurotomy of facet joints C2-C6 (n = 6) or to sham treatment (n = 6). The patients were followed for 2 years. The results showed the treated group had some improvement at 3 months, but later there were no marked differences between the groups. The authors concluded that the procedure is probably not beneficial in cervicogenic headache.

Denton et al (2017) discussed extracranial radiosurgery for the treatment of occipital neuralgia in those patients that are not surgical candidates. A dedicated phantom study was conducted to determine the optimum imaging studies, fusion matrices, and treatment planning parameters to target the C2 dorsal root ganglion which forms the occipital nerve. The conditions created from the phantom were applied to a patient with medically and surgically refractory occipital neuralgia. A dose of 80 Gy in one fraction was prescribed to the C2 occipital dorsal root ganglion. The phantom study resulted in a treatment achieved with an average translational magnitude of correction of 1.35 mm with an acceptable tolerance of 0.5 mm and an average rotational magnitude of correction of 0.4° with an acceptable tolerance of 1.0°. Dedicated quality assurance of the treatment planning and delivery is necessary for safe and accurate SRS to the cervical spine dorsal root ganglion. With additional prospective study, linear accelerator-based frameless radiosurgery can provide an accurate, noninvasive alternative for treating occipital neuralgia where an invasive procedure is contraindicated.

Radiofrequency Ablation for Occipital Neuralgia and Cervicogenic Headache

Clinical Context and Therapy Purpose

The purpose of RFA in patients who have occipital neuralgia or a cervicogenic headache is to provide a treatment option that is an alternative to or an improvement on existing therapies.

The question addressed in this evidence review is: Does the use of RFA improve the net health outcome in patients with occipital neuralgia or a cervicogenic headache?

The following PICOTS were used to select literature to inform this review.

Patients

The relevant population of interest is patients with occipital neuralgia or a cervicogenic headache.

Interventions

The therapy being considered is RFA. RFA involves the percutaneous insertion of a catheter that is directed toward the nerve of interest. RFA can be used to ablate the nerve by thermal lesioning.

Comparators

The following therapy is currently being used to make decisions about treating occipital neuralgia or a cervicogenic headache: conservative management.

Outcomes

The most clinically relevant outcome measures for pain treatments are measures of pain severity and functional limitations. Pain is most commonly measured with a VAS or RNS. Quantifiable pre- and posttreatment measures of functional status are also used, such as SF-12 and SF-36.

Timing

The time for follow-up is within days to determine the procedural success and months to years to evaluate durability.

Setting

RFA would be administered in an outpatient setting, typically pain clinics.

Radiofrequency Ablation (RFA)

Radiofrequency ablation of nerves has been proposed as a treatment for several different types of pain. It has been used to treat a number of clinical pain syndromes such as trigeminal neuralgia, cervical and lumbar pain, and headache syndromes. Radiofrequency procedures have been reported to have a high number of complications compared with other ablative neurosurgical procedures. Pulsed radiofrequency uses a pulsed time cycle that delivers short burst of RF energy to nervous tissue. Pulsed radiofrequency is performed under fluoroscopic guidance and is purported to be a less painful alternative to conventional radiofrequency therapy.

Systematic Reviews

A systematic review by Ducic et al (2014) did not identify any RCTs assessing RFA for chronic occipital neuralgia. Twelve reviewers identified 3 case series (total N=131 patients) on pulsed RF treatment. Success rates in these series ranged from 51% to 100%, with an overall success rate of 55%. Follow-up ranged from 3 to 10 months.

Grandhi et al (2018) conducted a systematic review of RFA for the treatment of a cervicogenic headache.¹¹ Ten studies met selection criteria, including 3 RCTs, 3 prospective studies, and 4 retrospective studies. There were no high-quality RCTs. Two of the RCTs evaluated RFA of the facet joints and failed to find a benefit of RFA. The third RCT compared RFA with steroid injection of the greater occipital nerve, finding no difference between the groups in the short term, but a longer duration of pain control in the RFA group.

Section Summary: RFA for Occipital Neuralgia and Cervicogenic Headache

No RCTs of RFA for chronic occipital neuralgia have been identified. A systematic review identified 3 RCTs of RFA for a cervicogenic headache, none of which were high quality. Pain is a subjective, patient-reported measure that is particularly susceptible to placebo effect. Trials with sham or active controls are needed to evaluate the efficacy of this treatment.

Pulsed Radiofrequency

Published evidence on the effectiveness of pulsed radiofrequency in the treatment of patients with headaches and occipital neuralgia is limited and based on retrospective, case series studies.

There is limited information available on the use of pulsed radiofrequency modulation for migraine headaches. Pulsed radiofrequency modulation for migraine via the stellate ganglion, located in front of the junction between C7 vertebral body and the transverse process, has also been used as a technique for relief. Incorrectly placed injections of local anesthetics may lead to loss of consciousness, seizures, paralysis, cardiac arrest, hoarseness, shortness of breath, sensation of an obstacle in the throat, and death.

Bayer et al (2005) evaluated the effectiveness of spheno-palatine ganglion pulsed radiofrequency (SPG-PRF) treatment in patients suffering from chronic head and face pain. A total of 30 patients were observed from 4 to 52 months after PRF treatment. The primary outcome measures were reduction in oral medication use (including opioids), time to next treatment modality for presenting symptoms, duration of pain relief, and the presence of residual symptoms. Secondary outcome measures included the evaluation of adverse effects and complications. All data were derived from patient charts, phone conversations, and clinical follow-up visits. A total of 14 % of respondents reported no pain relief, 21 % had complete pain relief, and 65 % of the patients reported mild-to-moderate pain relief from SPG-PRF treatment. A total of 65 % of the respondents reported mild-to-moderate reduction in oral opioids. None of the patients developed significant infection, bleeding, hematoma formation, dysesthesia, or numbness of palate, maxilla, or posterior pharynx. The authors concluded that these findings suggested that a prospective, randomized, controlled study to confirm the safety and effectiveness of PRF treatment for chronic head and face pain is justified.

Neurostimulation

Weiner et al (1999) reported on a study of 13 patients who underwent percutaneous peripheral nerve electrical stimulation for medically refractory occipital neuralgia. Thirteen patients underwent 17 implant procedures for medically refractory occipital neuralgia. A subcutaneous electrode placed transversely at the level of C1 across the base of the occipital nerve trunk produced paresthesia and pain relief covering the regions of occipital nerve pain. The results showed that 12 of 13 patients reported good to excellent response with > 50% pain control at follow-up of 1½ to 6 years. Subcutaneous peripheral neurostimulation in the region of one or more occipital nerves represents a new application of PNS neuroaugmentation for control of refractory occipital nerve mediated pain. The long-term efficacy of occipital nerve stimulation requires further study.

Ahmed et al (2000) conducted a crossover study in 30 patients with longstanding headaches of 3 types: tension, migraine, and posttraumatic injury. Two-week courses of active and sham PENS were compared. Outcomes were assessed at the completion of each treatment. Active PENS achieved better outcomes than sham PENS in terms of VAS pain, physical activity, and quality of sleep. Results did not vary by headache type. The investigators stated that the study was single-blinded but gave no details about blinding methods or whether withdrawals occurred. The report did not offer long-term outcomes data.

This single study does not establish the effectiveness of PENS for treatment of chronic headache.

Kapural et al (2005) reported on a case series of 6 patients with severe occipital neuralgia who underwent occipital nerve electrical stimulation lead implantation using a modified midline approach. These patients had previously been treated with oral antidepressants, membrane stabilizers, opioids, occipital nerve blocks, and radiofrequency ablations. Significant decreases in pain visual analog scale (VAS) scores and drastic improvement in functional capacity were observed during the occipital stimulation trial and during the 3 month follow-up after implantation. The mean VAS score changed from 8.66 ± 1.0 to 2.5 ± 1.3 . The pain disability index improved from 49.8 ± 15.9 to 14.0 ± 7.4 . These findings need to be validated by randomized controlled trials.

Slavin et al (2006) analyzed the records of 14 patients with chronic, intractable occipital neuralgia treated with peripheral nerve stimulation (PNS). Overall, 23 occipital nerves were stimulated in 14 patients. There were 17 trials in 10 patients considered successful, and those patients had permanent internalization of the stimulator. At the time of the last follow-up exam (mean 22 months), seven patients (70%) with implanted PNS had adequate pain control. There were two patients who had their systems explanted due to loss of stimulation effect or significant improvement of pain, and one patient had part of the hardware removed because of infection. The authors concluded that chronic peripheral nerve stimulation may be a safe and relatively effective method for long-term treatment of chronic pain syndrome in patients with medically intractable occipital neuralgia. This was a small study (only 14 patients). The authors stated that this study had a large variation between patients in regard to the etiology of their occipital neuralgia; therefore, they were unable to find any correlation between etiology of occipital neuralgia and the outcome of stimulation.

Schwedt et al (2007) reported on a retrospective analysis of 15 patients with medically intractable headache who underwent implantation of an occipital nerve stimulator. There were 15 patients (12 female, 3 male), (mean age 39 years), who had chronic migraine (n = 8), chronic cluster (n = 3), hemicrania continua (n = 2), or post-traumatic (n = 2) headache. Patients underwent either bilateral (n = 8) or unilateral (n = 7) lead placement. Patients were measured after 5-42 months. The results showed that all 6 headache measures improved significantly from baseline, including headache frequency per 90 days, headache severity, MIDAS disability, HIT-6 scores, BDI-II, and subjective pain. Most patients (60%) required lead revision within one year. The authors noted that safety and efficacy results from prospective, randomized, sham-controlled studies in patients with medically refractory headache are needed.

Boston Scientific Corporation is sponsoring the PRISM (Precision Implantable Stimulator for Migraine) clinical trial in which the Precision implantable stimulator is used to treat 179 migraine headache patients, at up to 15 sites in the U.S. Originating in 2006 and ending in 2016, this trial assessed the safety and efficacy of occipital nerve stimulation as a treatment for refractory migraine headaches.

The implantable pulse generator (IPG) will deliver electrical impulses to the occipital nerves located just under the skin at the back of the neck. The Precision neurostimulation system is approved by the U.S. Food and Drug Administration (FDA) for spinal cord stimulation to treat intractable chronic pain of the trunk and limbs. The use of the Precision neurostimulation system for treatment of refractory migraine headache is considered investigational and limited by Federal Law to investigational use only in the U.S.

Other Therapy Treatments

The use of oral medications is not effective for some patients, so other treatments have been proposed, such as local injections of anesthetics and/or steroids and epidural steroid injections. Other treatments for cervicogenic headache and occipital neuralgia that have been investigated include radiofrequency ablation of the planum nuchale, rhizotomy, ganglionectomy, nerve root decompression, discectomy and spinal fusion. These are generally performed under local or general anesthesia.

Summary of Evidence

Sphenopalatine Ganglion Block

For individuals who have chronic migraine who receive sphenopalatine ganglion blocks, the evidence includes 1 RCT and a case report. Relevant outcomes are symptoms, functional outcomes, quality of life, and treatment-related morbidity. The RCT was double-blind and placebo controlled, and provided a course of 12 SPG blocks over 6 weeks. It found significantly greater short-term (up to 24 hours) benefits of active treatment versus placebo. There were not significant longer-term effects (i.e., 1 and 6 months after a course of 12 treatments). The study was underpowered to detect longer term efficacy. Additional adequately powered RCTs demonstrating efficacy are needed. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals who have severe acute headache treated in an emergency setting who receive sphenopalatine ganglion blocks, the evidence includes 1 RCT. Relevant outcomes are symptoms,

functional outcomes, quality of life, and treatment-related morbidity. The RCT was double-blind and placebo controlled, and provided a single SPG block. There was not a statistically significant difference between active treatment and placebo in the primary outcome, pain reduction 15 minutes post-intervention. The study did not collect pain data again while patients were in the emergency department (e.g., at 1 hour after treatment). At 24 hours after treatment, significantly more patients were headache-free in the active treatment versus placebo group. However, there is insufficient evidence that SPG blocks are an effective treatment in the emergency setting. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals who have cluster headache who receive sphenopalatine ganglion blocks, the evidence includes case series. Relevant outcomes are symptoms, functional outcomes, quality of life, and treatment-related morbidity. Two small case series were available; the approach to intranasal SPG blocks differed from the intervention currently available in the United States. It is not clear how the safety or efficacy of the procedure used in the case series differs from an intranasal SPG block applying local anesthetics and using an FDA cleared device. In the series, 40-50% of patients experienced complete symptom relief for a variable length of time and about 20% had treatment-related complications. Additional studies, preferably RCTs are needed to evaluate SPG blocks for treating cluster headaches. The evidence is insufficient to determine the effects of the technology on health outcomes.

Occipital Neuralgia or Cervicogenic Headache

For individuals who have occipital neuralgia or cervicogenic headache who receive RFA of peripheral nerves, the evidence includes systematic reviews. Relevant outcomes are symptoms, functional outcomes, and quality of life. No RCTs of RFA for chronic occipital neuralgia have been identified. Three RCTs of RFA for a cervicogenic headache have been published, none of which were high quality. Pain is a subjective, patient-reported measure that is particularly susceptible to placebo effect. Randomized trials with sham or active-controls are needed to evaluate the efficacy of this treatment. The evidence is insufficient to determine the effects of the technology on health outcomes.

Key Words:

Cervicogenic headache, occipital neuralgia, migraine headache, Sphenopalatine Ganglion (SPG), Sphenopalatine Ganglion Block, Tx360[®], MiRx[™] protocol, SpenoCath[®], Allevio[™], Radiofrequency Ablation (RFA) sphenopalatine ganglion pulsed radiofrequency (SPG-PRF), Pulsed Radiofrequency Ablation (PRFA)

Approved by Governing Bodies:

The Tx360[®] Nasal Applicator (Tian Medical), the Allevio[™] SPG Nerve Block Catheter (JET Medical), and the SpenoCath[®] (Dolor Technologies) are considered class I devices by the U.S. Food and Drug Administration (FDA) and are exempt from 510(k) requirements. This classification does not require submission of clinical data regarding efficacy but only notification of FDA prior to marketing. These 3 devices are all used to apply numbing medication intranasally.

U.S. Preventative Services Task Force Recommendations

Not applicable.

Benefit Application:

Coverage is subject to member's specific benefits. Group specific policy will supersede this policy when applicable.

ITS: Home Policy provisions apply

FEP contracts: Special benefit consideration may apply. Refer to member's benefit plan. FEP does not consider investigational if FDA approved and will be reviewed for medical necessity.

Current Coding:

CPT Codes:

There is no specific CPT for sphenopalatine ganglion block. Use the unlisted procedure codes 30999 or 64999.

The use of code 64505 would be inappropriate as the procedure is for a spray application.

30999	Unlisted procedure, nose
63020	Laminotomy (hemilaminectomy), with decompression of nerve root(s), including partial facetectomy, foraminotomy and/or excision of herniated intervertebral disc, including open and endoscopically-assisted approaches; one interspace, cervical
63035	Laminotomy (hemilaminectomy), with decompression of nerve root(s), including partial facetectomy, foraminotomy and/or excision of herniated intervertebral disc; including open and endoscopically-assisted approaches; each additional interspace, cervical or lumbar (list separately in addition to code for primary procedure)
63040	Laminotomy (hemilaminectomy), with decompression of nerve root(s), including partial facetectomy, foraminotomy and/or excision of herniated intervertebral disc, reexploration, single interspace; cervical
63043	Laminotomy (hemilaminectomy), with decompression of nerve root(s), including partial facetectomy, foraminotomy and/or excision of herniated intervertebral disc, reexploration, single interspace; each additional cervical interspace (list separately in addition to code for primary procedure)
63045	Laminectomy, facetectomy and foraminotomy (unilateral or bilateral with decompression of spinal cord, cauda equine and/or nerve root(s), (e.g., spinal or lateral recess stenosis)), single vertebral segment; cervical

- 63048** Laminectomy, facetectomy and foraminotomy (unilateral or bilateral with decompression of spinal cord, cauda equina and/or nerve root(s), (e.g., spinal or lateral recess stenosis)), single vertebral segment; each additional segment, cervical, thoracic, or lumbar (list separately in addition to code for primary procedure)
- 63050** Laminoplasty, cervical, with decompression of the spinal cord, two or more vertebral segments;
- 63075** Discectomy, anterior, with decompression of spinal cord and/or nerve root(s), including osteophyctomy; cervical, single interspace
- 63076** Discectomy, anterior, with decompression of spinal cord and/or nerve root(s), including osteophyctomy; cervical, each additional interspace (list separately in addition to code for primary procedure)
- 63081** Vertebral corpectomy (vertebral body resection), partial or complete, anterior approach with decompression of spinal cord and/or nerve root(s); cervical, single segment
- 63082** Vertebral corpectomy (vertebral body resection), partial or complete, anterior approach with decompression of spinal cord and/or nerve root(s); cervical, each additional segment (list separately in addition to code for primary procedure)
- 63185** Laminectomy with rhizotomy; one or two segments
- 63190** Laminectomy with rhizotomy; more than two segments
- 63650** Percutaneous implantation of neurostimulator electrode array, epidural
- 63655** Laminectomy for implantation of neurostimulator electrodes, plate/paddle, epidural
- 63661** Removal of spinal neurostimulator electrode percutaneous array(s), including fluoroscopy, when performed
- 63662** Removal of spinal neurostimulator electrode plate/paddle(s) placed via laminotomy or laminectomy, including fluoroscopy, when performed
- 63663** Revision including replacement, when performed, of spinal neurostimulator electrode percutaneous array(s), including fluoroscopy, when performed
- 63664** Revision including replacement, when performed, of spinal neurostimulator electrode plate/paddle(s) placed via laminotomy or laminectomy, including fluoroscopy, when performed
- 63685** Insertion or replacement of spinal neurostimulator pulse generator or receiver, direct or inductive coupling
- 63688** Revision or removal of implanted spinal neurostimulator pulse generator or receiver
- 64405** Injection, anesthetic agent; greater occipital nerve
- 64450** Injection, anesthetic agent; other peripheral nerve or branch
- 64555** Percutaneous implantation of neurostimulator electrodes array; peripheral nerve (excludes sacral nerve)

- 64600** Destruction by neurolytic agent, trigeminal nerve; supraorbital, intraorbital, mental, or inferior alveolar branch
- 64612** Chemo-denervation of muscle(s); muscle(s) innervated by facial nerve, unilateral (e.g., for blepharospasm, hemifacial spasm)
- 64615** Chemodenervation of muscle(s); muscle(s) innervated by facial, trigeminal, cervical spinal and accessory nerves, bilateral (e.g., for chronic migraine)
- 64616** Chemodenervation of muscle(s); neck muscle(s), excluding muscles of the larynx, unilateral (e.g., for cervical dystonia, spasmodic torticollis)
- 64633** Destruction by neurolytic agent, paravertebral facet joint nerve(s), with imaging guidance (fluoroscopy or ct); cervical or thoracic, single facet joint
- 64634** Destruction by neurolytic agent, paravertebral facet joint nerve(s), with imaging guidance (fluoroscopy or CT); cervical or thoracic, each additional facet joint (List separately in addition to code for primary procedure)
- 64640** Destruction by neurolytic agent; other peripheral nerve or branch
- 64716** Neuroplasty and/or transposition; cranial nerve (specify)
- 64722** Decompression; unspecified nerve(s) (specify)
- 64727** Internal neurolysis, requiring use of operating microscope (List separately in addition to code for neuroplasty) (Neuroplasty includes external neurolysis)
- 64744** Transection or avulsion of; greater occipital nerve
- 64802** Sympathectomy, cervical
- 64804** Sympathectomy, cervicothoracic
- 64999** Unlisted procedure, nervous system
- 95970** Electronic analysis of implanted neurostimulator pulse generator system (e.g. rate, pulse amplitude, pulse duration, configuration of wave form, battery status, electrode selectability, output modulation, cycling, impedance and patient compliance measurements); simple or complex brain, spinal cord, or peripheral (i.e. cranial nerve, peripheral nerve, sacral nerve, neuromuscular) neurostimulator pulse generator/transmitter, without programming
- 95971** Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude, pulse duration, configuration of wave form, battery status, electrode selectability, output modulation, cycling, impedance and patient compliance measurements); simple spinal cord, or peripheral (i.e., peripheral nerve, autonomic nerve, neuromuscular) neurostimulator pulse generator/transmitter, with intraoperative or subsequent programming

HCPCS:

- E0745** Neuromuscular stimulator, electronic shock unit
- J0585** Injection, OnabotulinumtoxinA, one unit
- J0587** Injection, RimabotulinumtoxinB, 100 units

L8679	Implantable neurostimulator, pulse generator, any type
L8680	Implantable neurostimulator electrode (with any number of contact points), each
L8681	Patient programmer (external) for use with implantable programmable neurostimulator pulse generator, replacement only
L8682	Implantable neurostimulator radiofrequency receiver
L8683	Radiofrequency transmitter (external) for use with implantable neurostimulator radiofrequency receiver
L8684	Radiofrequency transmitter (external) for use with implantable sacral root neurostimulator receiver for bowel and bladder management, replacement
L8685	Implantable neurostimulator pulse generator, single array, rechargeable, includes extension
L8686	Implantable neurostimulator pulse generator, single array, non-rechargeable, includes extension
L8687	Implantable neurostimulator pulse generator, dual array, rechargeable, includes extension
L8688	Implantable neurostimulator pulse generator, dual array, non-rechargeable, includes extension
L8689	External recharging system for battery (internal) for use with implantable neurostimulator, replacement only
L8695	External recharging system for battery (internal) for use with implantable neurostimulator, replacement only

References:

1. Ahmed HE, White PF, Craig WF, et al. Use of percutaneous electrical nerve stimulation (PENS) in the short-term management of headache. *Headache*. Apr 2000; 40(4):311-315.
2. American Migraine Foundation. Sphenopalatine Ganglion Blocks in Headache Disorders. 2016; //americanmigrainefoundation.org/understanding-migraine/sphenopalatine-ganglion-blocks-in-headache-disorders/. Accessed April 14, 2017.
3. Barna S, et al. Occipital neuralgia. *Pain Management Rounds* 1004; 1(7).
4. Bayer E, Racz GB, Miles D, Heavner J. Sphenopalatine ganglion pulsed radiofrequency in treatment in 30 patients suffering from chronic face and head pain. *Pain Pract* 2005; 5(3):223-7.
5. Biondi DM, Bajwa ZH, et al. Cervicogenic headache. Up-to-Date, August 2007. www.uptodate.com.
6. Biondi DM. Cervicogenic headache: A review of diagnostic and treatment strategies. *Journal of American Osteopathic Association*, April 2005; 105(4 Suppl 2): 16S-22S.
7. Biondi DM. Cervicogenic headache: Diagnostic evaluation and treatment strategies. *Current Pain Headache Rep*, August 2001; 5(4): 361-368.
8. Blue Cross Blue Shield Association (BCBSA) Technology Evaluation Center (TEC). Botulinum toxin for treatment of primary chronic headache disorders. TEC Assessment Program, December 2004; 19(10).
9. Bogduk N. Cervicogenic headache: Anatomic basis and pathophysiologic mechanisms. *Current Pain Headache Rep*, August 2001; 5(4): 382-386.

10. Bogduk N. The anatomical basis for cervicogenic headache. *Journal of Manipulative Physiology Therapeutics*, January 1992; 15(1): 67-70.
11. Bogduk N. The neck and headaches. *Neurology Clinics*, February 2004; 22(1): 151-171.
12. Bovin G, et al. Cervicogenic headache: Anesthetic blockades of cervical nerves (C2-C5) and facet joint (C2/C3). *Pain*, June 1992; 49(3): 315-320.
13. Bovin G, et al. Neurolysis of the greater occipital nerve in cervicogenic headache. A follow-up study. *Headache*, April 1992; 32(4): 175-179.
14. Byrd D, Mackey S. Pulsed radiofrequency for chronic pain. *Current Pain and Headache Reports* 2008; 12:37-41.
15. Cady R, et al. A double-blind, placebo-controlled study of repetitive transnasal sphenopalatine ganglion blockade with Tx360® as acute treatment for chronic migraine. *Headache*. January 2015, 55:101-116.
16. Cady RK, Saper J, Dexter K, et al. Long-term efficacy of a double-blind, placebo-controlled, randomized study for repetitive sphenopalatine blockade with bupivacaine vs. saline with the Tx360 device for treatment of chronic migraine. *Headache*. Apr 2015; 55(4):529-542.
17. Candido KD, Massey ST, Sauer R, et al. A novel revision to the classical transnasal topical sphenopalatine ganglion block for the treatment of headache and facial pain. *Pain Physician*. Nov-Dec 2013; 16(6):E769-778.
18. Chavin JM. Cranial neuralgias and headaches associated with cranial vascular disorders. *Otolaryngology Clinics of North America*, December 2003; 36(6): 1079-1093.
19. Chou LH, et al. Cervicogenic headache. *Pain Physician*, April 2002; 5(2): 215-255.
20. Comprehensive Pain Management of North Florida. Interventional pain management. <http://www.cpmnf.com/Procedure.asp>. Accessed November 18, 2008.
21. Denton, et al. Practical considerations of linear accelerator-based frameless extracranial radiosurgery for treatment of occipital neuralgia for nonsurgical candidates. *J Appl Clin Med Phys*. 2017 May 18.
22. Dodick, et al. Botulinum neurotoxin for the treatment of migraine and other primary headache disorders. *Clinical Dermatology*, Jan-Feb 2004; 22(1): 76-81.
23. Drottning M, et al. Cervicogenic headache (CEH) six years after whiplash injury. *Functional Neurology*, July-Sept 2007; 22(3): 145-149.
24. Ducic I, Felder JM, 3rd, Fantus SA. A systematic review of peripheral nerve interventional treatments for chronic headaches. *Ann Plast Surg*. Apr 2014;72(4):439-445.
25. Ehni G, et al. Occipital neuralgia and the C1-2 arthrosis syndrome. *Journal of Neurosurgery*, November 1984; 61(5): 961-965.
26. Evans RW. Post-traumatic headaches. *Neurology Clinics*, February 2004; 22(1): 237-249.
27. Evers S. Botulinum toxin and the management of chronic headaches. *Current Opinions Otolaryngology Head and Neck Surgery*, June 2004; 12(3): 197-203.
28. Evers S. Status on the use of botulinum toxin for headache disorders. *Current Opinion Neurology*, June 2006; 19(3): 310-315.
29. Felisati G, Arnone F, Lozza P, et al. Sphenopalatine endoscopic ganglion block: a revision of a traditional technique for cluster headache. *Laryngoscope*. Aug 2006; 116(8):1447-1450.
30. Freund BJ, et al. Treatment of chronic cervical-associated headache with botulinum toxin A: A pilot study. *Headache*, March 2000; 40(3): 231-236.

31. Freund BJ. Use of botulinum toxin in chronic whiplash-associated disorder. *Clinical Journal Pain*, November 2002; 18 (6 Suppl): S163-S168.
32. Gille D, et al. Surgical treatment of greater occipital neuralgia by neurolysis of the greater occipital nerve and sectioning of the inferior oblique muscle. *Spine*, April 2004; 29(7): 828-832.
33. Grandhi RK, Kaye AD, Abd-Elsayed A. Systematic review of radiofrequency ablation and pulsed radiofrequency for management of cervicogenic headaches. *Curr Pain Headache Rep*. Feb 23 2018; 22(3):18.
34. Haldeman S. Cervicogenic headaches: A critical review. *Spine Journal*, January 2001; 1(1): 31-46.
35. Haspelslagh SR, et al. Randomised controlled trial of cervical radiofrequency lesions as a treatment for cervicogenic headache. *BMC Anesthesiology*, February 2006; 16: 6(1).
36. Headache Classification Subcommittee of the International Headache Society (IHS). The international classification of headache disorders, 2nd edition. *Cephalgia* 2004; 24: 115-116, 126-129.
37. Higa S. [Migraine and nerve block]. *Nippon Rinsho* 2001; 59(9): 1717-1721.
38. Inan N, et al. Cervicogenic headache: Pathophysiology, diagnostic criteria, and treatment. *Agri*, October 2005; 17(4): 23-30.
39. Inan N, et al. C2/C3 nerve blocks and greater occipital nerve block in cervicogenic headache treatment. *Functional Neurology*, July-Sept 2001; 16(3): 239-243.
40. International Association for the Study of Pain (IASP). *Epidemiology of Headache*. 2011; www.iasp-pain.org/files/Content/ContentFolders/GlobalYearAgainstPain2/HeadacheFactSheets/1-Epidemiology.pdf. Accessed April 10, 2017.
41. The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia*. Jul 2013; 33(9):629-808.
42. The MiRx™ Protocol www.mirxprotocol.com/. Accessed April 14, 2017.
43. Jansen J, et al. Cervicogenic headache: Long-term prognosis after neck surgery. *Acta Neurology Scandinavia*, March 2007; 115(3): 185-191.
44. Jansen J. Surgical treatment of non-responsive cervicogenic headache. *Clinical Experience Rheumatology*, March-April 2000; 18(2 Suppl 19); S67-S70.
45. Jensen S. Neck related causes of headache. *Australian Family Physician*, August 2005; 34(8): 635-639.
46. Johnstone CS, et al. Occipital nerve stimulation for the treatment of occipital neuralgia-8 case studies. *Pain Medicine*, September 2006; 7(5): 467.
47. Jull GA, et al. Predictors of responsiveness to physiotherapy management of cervicogenic headache. *Cephalgia*, February 2005; 25(2): 101-108.
48. Kapoor V, et al. Refractory occipital neuralgia: Preoperative assessment with CT-guided nerve block prior to dorsal cervical rhizotomy. *AJNR*, November-December 2003; 24(10): 2105-2110.
49. Kapural L, et al. Botulinum toxin occipital nerve block for the treatment of severe occipital neuralgia: A case series. *Pain Practice*, December 2007; 7(4): 337-340.
50. Kapural L, et al. Occipital nerve electrical stimulation via the midline approach and subcutaneous surgical leads for treatment of severe occipital neuralgia: A pilot study. *Anesthesia Analgesia*, July 2005; 101(1): 171-174.

51. Kastler B, Michalakis D, et al. [Stellate ganglion radiofrequency neurolysis under CT guidance. Preliminary study]. JBR-BTR 2001; 84(5): 191-194.
52. Mariana de Silva, Jr H, et al. Cervicogenic headache. Current Pain Headache Rep, August 2006; 10(4): 306-311.
53. Martelletti P, et al. Cervicogenic headache: Practical approaches to therapy. CNS Drugs 2004; 18(12): 793-805.
54. Mellick GA, et al. Regional head and face pain relief following lower cervical intramuscular anesthetic injection. Headache, November-December 2003; 43(10): 1109-1111.
55. Naja ZM, et al. Occipital nerve blockade for cervicogenic headache: A double-blind randomized controlled clinical trial. Pain Practice, June 2006; 6(2): 89-95.
56. Naja ZM, et al. Repetitive occipital nerve blockade for cervicogenic headache: Expanded case report of 47 adults. Pain Practice, December 2006; 6(4): 278-284.
57. Nilsson N. The prevalence of cervicogenic headache in a random population sample of 20–59 year olds. Spine (Phila Pa 1976), 1995. 20(17): p. 1884–8.
58. Numa W, et al. Classification of headache and facial pain. Otolaryngology Clinics of North America, December 2003; 36(6): 1055-1062.
59. Padberg M, et al. Treatment of chronic tension-type headache with botulinum toxin: A double-blind, placebo-controlled clinical trial. Cephalgia, August 2004; 24(8): 675-680.
60. Pipolo C, Bussone G, Leone M, et al. Sphenopalatine endoscopic ganglion block in cluster headache: a reevaluation of the procedure after 5 years. Neurol Sci. Jun 2010; 31 Suppl 1:S197-199.
61. Sjaastad et al. Cervicogenic Headaches: An Evidence Led Approach to Clinical Management. Physical Therapy, September 2011; 6 (4): 254.
62. Schaffer JT, Hunter BR, Ball KM, et al. Noninvasive sphenopalatine ganglion block for acute headache in the emergency department: a randomized placebo-controlled trial. Ann Emerg Med. May 2015; 65(5):503-510.
63. Schwedt TJ, et al. Occipital nerve stimulation for chronic headache – long-term safety and efficacy. Cephalgia, February 2007; 27(2): 153-157.
64. Sharma RR, et al. Current status of peripheral neurectomy for occipital neuralgia. Neurosurgery Quarterly, December 2005; 15(4): 232-238.
65. Siccoli MM, et al. Facial pain: Clinical differential diagnosis. Lancet Neurology 2006; 5: 257-267.
66. Silverman, SB. Cervicogenic headache: Interventional, anesthetic, and ablative treatment. Current Pain Headache Rep, August 2002; 6(4): 308-314.
67. Singh A, Soares WE. Management strategies for acute headache in the emergency department. Emerg Med Pract. Jun 2012; 14(6):1-23; quiz 23-24.
68. Sjaastad O, et al. Prevalence of cervicogenic headache: Vaga study of headache epidemiology. Acta Neurology Scandinavia, November 2007.
69. Slavin KV, et al. Peripheral neurostimulation for treatment of intractable occipital neuralgia. Neurosurgery, January 2006; 58(1): 112-119.
70. Slavin KV, et al. Trigeminal and occipital peripheral nerve stimulation for craniofacial pain: A single-institution experience and review of the literature. Neurosurgery Focus 2006; 21(6).
71. Staats PS and Patel Nilesh. Pain management in the head and neck patient. Cummings Otolaryngology Head and Neck Surgery, 4th edition, Chapter 17. Published by Elsevier.

72. Stovner LJ, et al. Radiofrequency denervation of facet joints C2-C6 in cervicogenic headache: A randomized, double-blind, sham-controlled study. *Cephalgia*, October 2004; 24(10): 821-830.
73. Taylor F, Swanson J, Dashe J. Tension-type headache in adults: Pathophysiology, clinical features, and diagnosis. Up To Date September 2018. www.uptodate.com/contents/tension-type-headache-in-adults-pathophysiology-clinical-features-and-diagnosis?search=tension%20type%20headache%20symptoms&source=search_result&selectedTitle=1~103&usage_type=default&display_rank=1#references. Accessed October 17, 2018.
74. The International Classification of Headache Disorders: 2nd edition. *Cephalalgia*. 2004; 24 Suppl 1:9-160.
75. van Suijlekom JA, et al. Cervicogenic headache: Techniques of diagnostic nerve blocks. *Clinical Exp Rheumatology*, March-April 2000; 18(2 Suppl 19): S39-S44.
76. van Suijlekom HA, et al. Radiofrequency cervical zygapophyseal joint neurotomy for cervicogenic headache: A prospective study of 15 patients. *Functional Neurology*, October-December 1998; 13(4): 297-303.
77. Voigt CL, Murphy MO. Occipital nerve blocks in the treatment of headaches: safety and efficacy. *J Emerg Med*. 2015 Jan; 48(1):115-29.
78. Weiner RL. Occipital neurostimulation for treatment of intractable headache syndromes. *Acta Neurochir Suppl* 2007; 97(Pt1): 129-133.
79. Weiner RL, et al. Peripheral neurostimulation for control of intractable occipital neuralgia. *Neuromodulation*, July 1999; 2(3): 217-229.
80. Yi X, et al. Cervicogenic headache in patients with presumed migraine: Missed diagnosis or misdiagnosis? *The Journal of Pain*, October 2005; 6(10): 700-703.

Policy History:

Medical Policy Group, January 2008 (3)

Medical Policy Administration Committee, February 2008

Available for comment February 9-March 24, 2008

Medical Policy Group, August 2008 (2)

Medical Policy Administration Committee, August 2008

Available for comment, August 13-September 26, 2008

Medical Policy Group, November 2008 (1)

Medical Policy Administration Committee, December 2008

Available for comment February 17-April 2, 2009

Medical Policy Group, August 2010 (3)

Medical Policy Administration Committee, September 2010

Available for comment September 4-October 18, 2010

Medical Policy Group, October 2010

Medical Policy Group, March 2011 (3)

Medical Policy Group, December 2011 (3): 2012 Coding Updates – Added 63030, 64633 and 64634, changed verbiage in 64555, 95970 and 95971 and deleted 64626 and 64627.

Medical Policy Group, December 2012 (3): 2013 Coding Update: Verbiage change 64612 and addition of 64615

Medical Policy Group, June 2013 **(3)**: added bullet for clarification of implantable infusion pumps as injections as non-covered treatment

Medical Policy Group, December 2013 **(1)**: 2014 Coding Update: added new code 64616, effective 01/01/14; moved deleted code 64613 to previous coding, effective 01/01/14

Medical Policy Group, May 2014 **(5)**: 2014 Coding Update: Deleted code L8680 effective July 1, 2014.

Medical Policy Group, June 2014 **(5)**: Quarterly 2014 Coding Update: Code L8680 did not delete added back to policy under current codes.

Medical Policy Group, April 2015 **(5)**: Under policy statement removed effective for dates of service on or after October 15, 2010 from Botulinum toxin and just left to refer to MP 074. No change in policy statement.

Medical Policy Group, July 2015 **(6)**: Updates to Key Points and References; no change in policy statement.

Medical Policy Group, June 2016 **(6)**: Updates to Policy Statement, Key Points, Key Words Coding and References.

Medical Policy Administration Committee, July 2016

Available for comment June 28 through August 11, 2016

Medical Policy Group, November 2016 **(6)**: Updated coding, added L8679 Implantable neurostimulator, pulse generator, any type.

Medical Policy Group, May 2017 **(6)**: Updates to Description, Key Points, Key Words and Governing Bodies, and References. Included updated information regarding Sphenopalatine Ganglion Block.

Medical Policy Group, October 2017 **(6)**: Removed Botox from policy statement, Botox is a Physician Administered Drug Policy.

Medical Policy Group, December 2017 **(6)**: Removed coding “63030 - Laminotomy (hemilaminectomy), with decompression of nerve root(s), including partial facetectomy, foraminotomy and/or excision of herniated intervertebral disc; one interspace, lumbar”.

Medical Policy Group, October 2018 **(3)**: Policy statement now includes: “radiofrequency ablation of peripheral nerves”, Added code 64722 to current coding. Updates to Description, Key Points, References and Key Words: added Radiofrequency Ablation (RFA) Sphenopalatine Ganglion Pulsed Radiofrequency (SPG-PRF), Pulsed Radiofrequency Ablation (PRFA).

This medical policy is not an authorization, certification, explanation of benefits, or a contract. Eligibility and benefits are determined on a case-by-case basis according to the terms of the member's plan in effect as of the date services are rendered. All medical policies are based on (i) research of current medical literature and (ii) review of common medical practices in the treatment and diagnosis of disease as of the date hereof. Physicians and other providers are solely responsible for all aspects of medical care and treatment, including the type, quality, and levels of care and treatment.

This policy is intended to be used for adjudication of claims (including pre-admission certification, pre-determinations, and pre-procedure review) in Blue Cross and Blue Shield's administration of plan contracts.