Prior Authorization Review
Panel MCO Policy Submission

A separate copy of this form must accompany each policy submitted for review. Policies submitted without this form will not be considered for review.

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*All revisions to the policy must be highlighted using track changes throughout the document. Please provide any clarifying information for the policy below:

CPB 374 Trigeminal Neuralgia: Treatments

This CPB has been revised to state that the following are considered experimental and investigational for the treatment of trigeminal neuralgia: (i) intra-arterial modulation (e.g., intra-arterial lidocaine) of the trigeminal nerve ganglion, (ii) percutaneous ozone injection, and (iii) radial extracorporeal shock wave therapy.

Name of Authorized Individual (Please type or print): Dr. Bernard Lewin, M.D.

Signature of Authorized Individual: [Signature]

www.aetnabetterhealth.com/pennsylvania Revised 04/12/2018
Trigeminal Neuralgia: Treatments

Policy

Aetna considers the following surgical procedures for the treatment of trigeminal neuralgia medically necessary when the condition has persisted for at least 6 months despite conservative treatment with pharmacotherapies (carbamazepine, phenytoin, and baclofen) or the member is unable to tolerate the side effects of the medications.

- Balloon compression
- Gamma knife
- Microvascular decompression
- Percutaneous glycerol rhizotomy (or injections)∗
- Percutaneous radiofrequency rhizolysis/rhizotomy∗

*Please see amendment for Pennsylvania Medicaid at the end of this CPB.

Policy History

Last Review
04/12/2018
Effective: 03/09/2000
Next Review: 04/11/2019

Definitions

Additional Information

Clinical Policy Bulletin Notes
Aetna considers surgery for trigeminal neuralgia not meeting the afore-mentioned criteria experimental and investigational.

Aetna considers trigeminal nerve blocks medically necessary for refractory cases of trigeminal neuralgia.

Aetna considers the following interventions experimental and investigational for the treatment of trigeminal neuralgia because their effectiveness has not been established (not an all-inclusive list):

[hyperlink to document page]
- Adipose-derived stem cells
- Botulinum toxin
- Deep brain stimulation
- Electrical stimulation of the occipital nerve or trigeminal nerve
- Intra-arterial modulation (e.g., intra-arterial lidocaine) of the trigeminal nerve ganglion
- Intravenous lidocaine (alone or in combination with magnesium)
- Intravenous magnesium (alone or in combination with lidocaine)
- Motor cortex stimulation (CPB 0755 - Motor Cortex Stimulation (see ../700_799/0755.html)).
- Percutaneous ozone injection
- Percutaneous neuroablation
- Peripheral/subcutaneous nerve field stimulation
- Pulsed radiofrequency (see CPB 0735 - Pulsed Radiofrequency ( ../700_799/0735.html)).
- Radial extracorporeal shock wave therapy
- Spheno-palatine ganglion stimulation
- Spinal cord stimulation
- Topical lidocaine (e.g., lidocaine medicated plaster)
- Transection of the auriculo-temporal nerve, zygomatico-temporal nerve, and/or other branches of the mandibular nerve or trigeminal nerve
- Trigeminal tractotomy
Note: These peripheral procedures refer to techniques that target portions of the trigeminal nerve distal to the Gasserian ganglion or techniques that target the Gasserian ganglion itself.

See also CPB 0707 - Headaches: Invasive Procedures
(../700_799/0707.html)

Background

Trigeminal neuralgia (TN), also known as tic douloureux, is a neuropathic pain syndrome characterized by paroxysmal, triggered, trigeminally distributed pain. It affects more women than men (3:1 ratio), and is more common in the elderly. The anti-epileptic drug carbamazepine (Tegretol) is the drug of choice for the management of TN. Tegretol, at a dose of 600 to 1,200 mg per day, has been demonstrated to inhibit or shorten the duration of attacks. Phenytoin (Dilantin), is also effective in treating TN, but less so than carbamazepine. For patients who can not tolerate carbamazepine because of its adverse side effects (poor liver function, confusion, ataxia, drowsiness, and allergic responses), baclofen and other anti-convulsant drugs such as clonazepam (Klonopin) may be useful. If pharmacotherapy fails, surgical intervention may be necessary.

Surgical treatment can be divided into 2 categories: (i) percutaneous, and (ii) open. The former approaches include radiofrequency rhizolysis, glycerol injection, and balloon
compression techniques. The principal open approach is microvascular decompression, which entails posterior fossa craniotomy and has a small incidence of serious neurological morbidity. In general, elderly or medically debilitated patients, patients with multiple sclerosis, or individuals who have failed to attain pain relief from the open approach are encouraged to use the percutaneous approaches, while the open approach is recommended for younger and healthier subjects.

The gamma knife, one type of stereotactic radiosurgery, is also used as a means of treating patients with TN. It was developed in the 1950's by Larsson and Leksell, and is primarily used as a non-invasive alternative treatment for certain types of brain lesions such as intra-cranial arteriovenous malformations and brain tumors. When the gamma knife is used to treat TN, the beams are focused on the root of the trigeminal nerve. The stereotactic coordinates of the target site are often determined by computed tomography and/or magnetic resonance imaging. Patients undergoing gamma knife radiosurgery are usually given local anesthesia with a mild sedative.

Pulsed radiofrequency (PRF) treatment entails the application of short bursts of RF energy to nervous tissue. It is a minimally destructive procedure that may serve as an alternative to traditional RF heat lesion. In a review on PRF treatment, Gallagher (2006) stated that "we should cautiously prescribe this promising intervention following clinical algorithms that are based upon the best clinical evidence available. However, it is critically important to avoid the
mistake of creating a "carte blanche" environment for those practitioners who would abuse the privilege and opportunity presented by this new technology, besmirching our credibility and ultimately impeding the opportunity to use this treatment to the benefit of the public. Ultimately, evidence, not reimbursement, should determine whether PRF finds a place in our clinical toolbox”.

Cahana et al (2006) stated that PRF, a non- or minimally-neurodestructive technique, has been used as alternative to radiofrequency heat lesions. Clinical advantages and mechanisms of PRF remain unclear. These investigators reviewed current clinical and laboratory data on PRF. The final analysis yielded 58 reports on the clinical use of PRF in different applications: 33 full publications and 25 abstracts. They also retrieved 6 basic science reports, 5 full publications, and 1 abstract. The authors concluded that the accumulation of these data showed that the use of PRF generates an increasing interest of pain physicians for the management of a variety of pain syndromes. Although the mechanism of action has not been completely elucidated, laboratory reports suggested a genuine neurobiological phenomenon altering the pain signaling, which some researchers have described as neuromodulatory. No side effects related to PRF were reported to date. They stated that further research in the clinical and biological effects of this technique is justified.

In the only controlled clinical trial of PRF for TN published to date, Erdine et al (2007) concluded that PRF is not an effective treatment for TN.
Erdine et al (2007) reported on a prospective, randomized, double-blinded study to compare PRF to conventional radiofrequency (CRF) in the treatment of idiopathic TN. A total of 40 patients with idiopathic TN were randomly assigned to PRF or CRF. Visual Analog Scale (VAS) scores decreased significantly (p < 0.001) and Patient Satisfaction Scale (PSS) scores improved significantly (p < 0.001) after the procedure in subjects assigned to CRF. The VAS score decreased in only 2 of 20 patients from the PRF group and pain recurred 3 months after the procedure. The investigators reported that, at the end of 3 months, they performed CRF in patients assigned to PRF, because all patients in this group still had intractable pain.

Cheshire (2007) stated that many treatments have been developed for TN. Anti-epileptic drugs are superior to traditional analgesics with carbamazepine being the drug of first choice. Additional drugs for which there is evidence of efficacy include oxcarbazepine, baclofen, gabapentin, lamotrigine and phenytoin. However, many patients eventually experience tachyphylaxis or may not tolerate effective doses. Surgical interventions include microvascular decompression; balloon compression; RF thermocoagulation or glycerol rhizotomies; and subcutaneous alcohol branch blockade. Stereotactic gamma knife radiosurgery is a further option. Motor cortex stimulation and transcranial magnetic stimulation, although having shown initial promise for trigeminal neuropathic pain, seem to be ineffective for classical TN.
The Quality Standards Subcommittee of the American Academy of Neurology and the European Federation of Neurological Societies' practice parameter on the diagnostic evaluation and treatment of TN (Gronseth et al, 2008) stated that for patients with TN refractory to medical therapy, Gasserian ganglion percutaneous techniques, gamma knife, and microvascular decompression may be considered.

Adler et al (2009) stated that although stereotactic radiosurgery is an established procedure for treating TN, the likelihood of a prompt and durable complete response is not assured. Moreover, the incidence of facial numbness remains a challenge. To address these limitations, a new, more anatomic radiosurgical procedure was developed that uses the CyberKnife (Accuray, Inc., Sunnyvale, CA) to lesion an elongated segment of the retro-Gasserian cisternal portion of the trigeminal sensory root. Because the initial experience with this approach resulted in an unacceptably high incidence of facial numbness, a gradual dose and volume de-escalation was performed over several years. In this single-institution prospective study, these researchers assessed clinical outcomes in a group of TN patients who underwent lesioning with seemingly optimized non-isocentric radiosurgical parameters. A total of 46 patients with intractable idiopathic TN were treated between January 2005 and June 2007. Eligible patients were either poor surgical candidates or had failed previous microvascular decompression or destructive procedures. During a single radiosurgical session, a 6-mm segment of the
affected nerve was treated with a mean marginal prescription dose of 58.3 Gy and a mean maximal dose of 73.5 Gy. Monthly neurosurgical follow-up was performed until the patient became pain-free. Longer-term follow-up was performed both in the clinic and over the telephone. Outcomes were graded as excellent (pain-free and off medication), good (greater than 90% improvement while still on medication), fair (50 to 90% improvement), or poor (no change or worse). Facial numbness was assessed using the Barrow Neurological Institute Facial Numbness Scale score. Symptoms disappeared completely in 39 patients (85%) after a mean latency of 5.2 weeks. In most of these patients, pain relief began within the first week. Trigeminal neuralgia recurred in a single patient after a pain-free interval of 7 months; all symptoms abated after a second radiosurgical procedure. Four additional patients underwent a repeat rhizotomy after failing to respond adequately to the first operation. After a mean follow-up period of 14.7 months, patient-reported outcomes were excellent in 33 patients (72%), good in 11 patients (24%), and poor/no improvement in 2 patients (4%). Significant ipsilateral facial numbness (Grade III on the Barrow Neurological Institute Scale) was reported in 7 patients (15%). The authors concluded that optimized non-isocentric CyberKnife parameters for TN treatment resulted in high rates of pain relief and a more acceptable incidence of facial numbness than reported previously. Moreover, they stated that longer follow-up periods will be needed to establish whether or not the durability of
symptom relief after lesioning an elongated segment of the trigeminal root is superior to isocentric radiosurgical rhizotomy.

Fariselli et al (2009) reported the safety and effectiveness and safety of CyberKnife robotic radiosurgery as a first-line treatment against pharmacologically refractory TN. These investigators treated 33 patients with the frameless CyberKnife system as a monotherapy. The retro-Gasserian portion of the trigeminal nerve (a length of 4 mm, 2 to 3 mm anterior to the root entry zone) was targeted. Doses of 55 to 75 Gy were prescribed to the 100 % isodose line, according to a dose escalation protocol. Patients were evaluated for the level of pain control, time to pain relief, hypesthesia, and time to pain recurrence. The median age was 74 years. All but 2 patients (94 %) achieved a successful treatment outcome. The follow-up period was 9 to 37 months (mean of 23 months). The Barrow Neurological Institute Pain Intensity Scale (BPS) score before radiosurgery was III in 2 patients (6 %), IV in 8 patients (24 %), and V in 23 patients (70 %). The time to pain relief was 1 to 180 days (median of 30 days). No facial numbness was observed. Only 1 patient developed a transitory dysesthesia of the tongue. After treatment, the BPS score was I, II, or III in 31 patients (97 %). Pain recurred in 33 % (11 patients) at a mean of 9 months (range of 1 to 43 months). Three patients with recurrences had low pain control by medication (BPS score, IV), and 1 patient (BPS score, V) needed a RF lesioning (BPS score, I at 12 months). The authors concluded that CyberKnife radiosurgery for TN allows pain relief at safe doses and is suggested for pharmacologically
refractory TN. It should be noted that the rate of pain recurrence is rather high in this study (33%).

Kouzounias et al (2010) compared percutaneous balloon compression (PBC) and percutaneous retrogasserian glycerol rhizotomy (PRGR) for the treatment of TN in terms of effectiveness, complications, and technical aspects. A total of 66 consecutive PBC procedures were performed in 45 patients between January 2004 and December 2008, and 120 PRGR attempts were performed in 101 patients between January 2006 and December 2008. The PRGR procedures were not completed due to technical reasons in 19 cases. Five patients in the PBC group and 9 patients in the PRGR group were lost to follow-up and were excluded from the study. The medical records as well as the intra-operative fluoroscopic images from the remaining cases were retrospectively examined, and the follow-up was completed with telephone contact, when necessary. The 2 groups were compared in terms of initial effect, duration of effect, and rates of complications as well as severity and type of complications. The rates for immediate pain relief were 87% for patients treated with glycerol injection and 85% for patients treated with balloon compression. The Kaplan-Meier plots for the 2 treatment modalities were similar. The 50% recurrence time was 21 months for the balloon procedure and 16 months for the glycerol procedure. When the groups were broken down by the "previous operations" criterion, the 50% recurrence time was 24 months for the Glycerol First Procedure Group, 6 months for the Balloon First Procedure Group, 8 months for the Glycerol Previous
Procedures Group, and 21 months for the Balloon Previous Procedures Group. The rates of complications (excluding numbness) were 11 % for PRGR and 23 % for PBC, and this difference was statistically significant (chi-square test, p = 0.04). The authors concluded that both PRGR and PBC are effective techniques for the treatment of TN, with PRGR presenting some advantages in terms of milder and fewer complications and allowing lighter anesthesia without compromise of analgesia. For these reasons the authors consider PRGR as the first option for the treatment of TN in patients who are not suitable candidates or are not willing to undergo microvascular decompression, while PBC is reserved for patients in whom the effect of PRGR has proven to be short or difficult to repeat due to cisternal fibrosis.

A Cochrane review on “Neurosurgical interventions for the treatment of classical trigeminal neuralgia” (Zakrzewska and Akram, 2011) noted that surgical treatments for refractory trigeminal neuralgia are divided into 2 main categories: (i) ablative (destructive) and (ii) non-ablative. These treatments can be done at 3 different sites: (i) peripherally, (ii) at the Gasserian ganglion level, and (iii) within the posterior fossa of the skull. The authors concluded that “There is very low quality evidence for the effectiveness of most neurosurgical procedures for trigeminal neuralgia because of the poor quality of the trials. All procedures produced variable pain relief, but many resulted in sensory side effects .... Well-designed studies are urgently needed”.
An UpToDate review on "Trigeminal neuralgia" (Bajwa et al, 2013) states that "Peripheral neurectomy can be performed on the branches of the trigeminal nerve, which are the supraorbital, infraorbital, alveolar, and lingual nerves. Neurectomy is accomplished by incision, alcohol injection, radiofrequency lesioning, or cryotherapy. Cryotherapy involves freezing of the nerve using special probes, in theory to selectively destroy the pain fibers. The AAN/EFNS practice parameter noted that the evidence regarding peripheral techniques for the treatment of TN is either negative or inconclusive".

Collet et al (2013) stated that the etiology of TN remains unclear and several theories have been proposed. Many medical and surgical methods have been applied with only partial effectiveness and several side effects. New hypotheses and therapeutic methods are urgently needed. Using evidence presented in a literature review and in the authors’ case report, these researchers hypothesized that pain resulting from TN can be caused by demyelinating lesions in the trigger zone; and that these lesions can be repaired through the injection of fat containing adipose-derived stem cells.

Parmar et al (2013) stated that pharmacotherapy still remains the first line therapy for the management of TN. However, often the patients become refractory to the pharmacotherapy and need surgical interventions. There is a wide array of surgical treatment modalities available for TN. These investigators evaluated the various surgical modalities by employing a comparative analysis with respect to patient
selection, success rate, complications and cost-effectiveness. For the evaluation, a critical review of literature was done with pre-defined search terms to obtain the details of individual procedures, which were then compared, under similar parameters. The results suggested that microvascular decompression seem to be the most effective treatment in terms of patient satisfaction and long term cost-effectiveness. However, if patient factors do not permit, then the peripheral procedures may be employed as a substitute, though they have higher recurrence rate and complications and have relatively lower long-term cost effectiveness. The authors noted that newer modalities like stereotactic radiosurgery and botulinum toxin have promising results and further refinement in these procedures will provide additional options for the patients suffering from TN.

Hu and colleagues (2013) systematically reviewed the therapeutic safety and effectiveness of botulinum toxin type A (BTX-A) in the treatment of TN. PubMed, EMBASE, Cochrane Library Clinical Trials and Web of Science from January 1966 to March 2013 were searched with the terms of "botulinum toxin" and "trigeminal neuralgia", and references of related articles were traced. Data on the safety and effectiveness of BTX-A in this disorder were extracted and analyzed by at least 2 reviewers. Data for individual studies were reported, and pooled data were analyzed if appropriate. A total of 5 prospective studies and 1 double-blind, randomized, placebo-controlled study were identified. Response was achieved in approximately 70 to 100% of patients, and the mean pain intensity and frequency were reduced.
by approximately 60 to 100% at 4 weeks after treatment in most studies. Major adverse events were not reported. Available studies showed BTX-A may be effective in treatment of TN. However, the authors concluded that well-designed randomized, controlled, double-blinded trial is still lacking. They stated that future BTX-A treatment studies on optimal dose, duration of the therapeutic effectiveness, common adverse events, and the time and indications for repeat injection would be promising.

Fontaine et al (2014) stated that although most patients suffering from TN respond to medical or surgical treatment, non-responders remain in very severe painful condition. These researchers described for the first time a case of severe refractory classical TN treated successfully (follow-up 1 year) by chronic bilateral occipital nerve stimulation (ONS), because other classic medical and surgical options failed or could not be performed. The authors concluded that this single case suggested that ONS might be offered to TN patients who are refractory both to standard drugs and interventions, with a favorable risk/benefit ratio, although its long-term effectiveness remains unknown. This preliminary finding needs to be validated by well-designed studies.

Tamburin et al (2014) reported on 2 patients with classical TN in whom conventional drugs for TN were not tolerated. In these patients, treatment with 5% lidocaine medicated plaster (LMP) resulted in reduction of pain intensity and the number of pain paroxysms. Lidocaine medicated plaster is known to block the sodium
channels on peripheral nerves and may cause a selective and partial block of Aδ and C fibers. According to the TN ignition hypothesis, blockage of peripheral afferents by LMP may reduce pain paroxysms. The effect of LMP may outlast the pharmacokinetics of the drug by reducing pain amplification mechanisms in the central nervous system; LMP has limited or no systemic side effects. The authors concluded that LMP may be an effective and well-tolerated treatment option for TN in those patients who do not tolerate or who refuse other therapies. They stated that future randomized controlled studies should better address this issue.

Stavropoulou et al (2014) stated that intravenous lidocaine infusions are gaining acceptance in a variety of pain-management settings. These researchers stated that future clinical research should focus on identifying the effective dosage of lidocaine alone, magnesium alone, and lidocaine-magnesium combination to be used in the treatment of TN.

An UpToDate review on “Trigeminal neuralgia” (Bajwa et al, 2015) states that “Several other drugs have shown some evidence of efficacy for TN in small, generally lower-quality controlled trials .... Topical lidocaine given by intra-oral application was more effective than placebo for pain reduction in a two-week, randomized cross-over trial of 24 subjects with TN whose pain was most severe pain in the mouth. However, blinding may have been compromised because of the bitter taste or numbness perceived by some patients when treated with lidocaine .... There is only limited evidence to support treatment alternatives for
patients with TN who are refractory to first-line medical therapy. Intravenous infusion of phenytoin, fosphenytoin or lidocaine may provide analgesia while oral medications are titrated. Nevertheless, there are no randomized controlled trials comparing monotherapy with combination therapy for TN. Moreover, magnesium is not mentioned as a therapeutic option.

Tuleasca et al (2014) examined the safety and effectiveness of repeat Gamma Knife surgery (GKS) for recurrent TN. Using the prospective database of TN patients treated with GKS in Timone University Hospital (Marseille, France), data were analyzed for 737 patients undergoing GKS for TN Type 1 from July 1992 to November 2010. Among the 497 patients with initial pain cessation, 34.4% (157/456 with greater than or equal to 1-year follow-up) experienced at least 1 recurrence. Thirteen patients (1.8%) were considered for a second GKS, proposed only if the patients had good and prolonged initial pain cessation after the first GKS, with no other treatment alternative at the moment of recurrence. As for the first GKS, a single 4-mm isocenter was positioned in the cisternal portion of the trigeminal nerve at a median distance of 7.6 mm (range of 4 to 14 mm) anterior to the emergence of the nerve (retrogasserian target). A median maximum dose of 90 Gy (range of 70 to 90 Gy) was delivered. Data for 9 patients with at least 1-year follow-up were analyzed. A systematic review of literature was also performed, and results are compared with those of the Marseille study. The median time to re-treatment in the Marseille study was 72 months (range of 12 to 125 months) and in the literature
it was 17 months (range of 3 to 146 months). In the Marseille study, the median follow-up period was 33.9 months (range of 12 to 96 months), and 8 of 9 patients (88.9 %) had initial pain cessation with a median of 6.5 days (range of 1 to 180 days). The actuarial rate for new hypesthesia was 33.3 % at 6 months and 50 % at 1 year, which remained stable for 7 years. The actuarial probabilities of maintaining pain relief without medication at 6 months and 1 year were 100 % and 75 %, respectively, and remained stable for 7 years. The systematic review analyzed 20 peer-reviewed studies reporting outcomes for repeat GKS for recurrent TN, with a total of 626 patients. Both the selection of the cases for re-treatment and the way of reporting outcomes vary widely among studies, with a median rate for initial pain cessation of 88 % (range of 60 % to 100 %) and for new hypesthesia of 33 % (range of 11 % to 80 %). The authors concluded that results from the Marseille study raised the question of surgical alternatives after failed GKS for TN. The rates of initial pain cessation and recurrence seem comparable to, or even better than, those of the first GKS, according to different studies, but toxicity is much higher, both in the Marseille study and in the published data. Neither the Marseille study data, nor the literature data, answered the 3 cardinal questions regarding repeat radiosurgery in recurrent TN: which patients to re-treat, which target is optimal, and which dose to use.

Tempel et al (2015) reviewed pain outcomes and complications in TN patients who required 3 radiosurgical procedures for recurrent or persistent pain. These researchers performed a retrospective review of all patients who
underwent 3 Gamma Knife radiosurgery (GKRS) procedures for TN at 4 participating centers of the North American Gamma Knife Consortium from 1995 to 2012 was performed. The Barrow Neurological Institute (BNI) pain score was used to evaluate pain outcomes. A total of 17 patients were identified; 7 were males and 10 were females. The mean age at the time of last GKRS was 79.6 years (range of 51.2 to 95.6 years). The TN was Type I in 16 patients and Type II in 1 patient. No patient suffered from multiple sclerosis. Eight patients (47.1%) reported initial complete pain relief (BNI Score I) following their third GKRS and 8 others (47.1%) experienced at least partial relief (BNI Scores II-IIIb). The average time to initial response was 2.9 months following the third GKRS. Although 3 patients (17.6%) developed new facial sensory dysfunction following primary GKRS and 2 patients (11.8%) experienced new or worsening sensory disturbance following the second GKRS, no patient sustained additional sensory disturbances after the third procedure. At a mean follow-up of 22.9 months following the third GKRS, 6 patients (35.3%) reported continued Score I complete pain relief, while 7 others (41.2%) reported pain improvement (BNI Scores II-IIIb). Four patients (23.5%) suffered recurrent TN following the third procedure at a mean interval of 19.1 months. The authors concluded that a third GKRS resulted in pain reduction with a low risk of additional complications in most patients with medically refractory and recurrent, intractable TN. In patients unsuitable for other microsurgical or percutaneous strategies, especially those
receiving long-term oral anti-coagulation or anti-platelet agents, GKRS repeated for a third time was a satisfactory, low risk option.

**Combination of Pharmacotherapy and Lidocaine Block:**

In a pilot study, Stani et al (2015) evaluated the therapeutic effect of combination of pharmacotherapy and lidocaine block in the treatment of classical TN (CTN). A total of 13 patients with CTN managed with pharmacotherapy were recruited and assigned either to no additional treatment (Group I) or to additional analgesic block (Group II). The primary end-point was the reduction in the frequency of pain episodes in a month assessed at 30 and 90 days. Comparisons of measurements of pain, general health and depression scales were secondary end-points. The results from the follow-up visits at 30 and 90 days showed the Group II to have larger reduction in the frequency of pain and exhibited a bigger improvement in the scores of the pain, general health and depression scales. The authors concluded that the findings of this study suggested a clinical benefit of the combination of pharmacotherapy and lidocaine block. These preliminary findings need to be validated in well-designed studies.

**Peripheral/Subcutaneous Trigeminal Nerve Field Stimulation:**

Klein et al (2016) stated that peripheral nerve field stimulation (PNFS) is a promising modality for treatment of intractable facial pain.
However, evidence is sparse. These researchers presented their experience with this technique in a small patient cohort. Records of 10 patients (5 men, 5 women) with intractable facial pain who underwent implantation of one or several subcutaneous electrodes for trigeminal nerve field stimulation were retrospectively analyzed. Patients' data, including pain location, etiology, duration, previous treatments, long-term effects and complications, were evaluated. Four patients suffered from recurrent classical TN, 1 had classical TN and was medically unfit for microvascular decompression. Two patients suffered from trigeminal neuropathy attributed to multiple sclerosis, 1 from post-herpetic neuropathy, 1 from trigeminal neuropathy following radiation therapy and 1 from persistent idiopathic facial pain. Average patient age was 74.2 years (range of 57 to 87), and average symptom duration was 10.6 years (range of 2 to 17). Eight patients proceeded to implantation after successful trial. Average follow-up after implantation was 11.3 months (range of 5 to 28). Using the VAS, average pain intensity was 9.3 (range of 7 to 10) pre-operatively and 0.75 (range of 0 to 3) post-operatively; 6 patients reported absence of pain with stimulation; 2 had only slight constant pain without attacks. The authors concluded that PNFS may be an effective treatment for refractory facial pain and yields high patient satisfaction. These preliminary findings need to be validated by well-designed studies.

William et al (2016) retrospectively reviewed their early experience with stimulation involving the trigeminal and sphenopalatine ganglion (SPG) stimulation for trigeminal neuropathic pain.
(TNP), anesthesia dolorosa, and persistent idiopathic facial pain (PIFP) between 2010 and 2014 to evaluate the feasibility of implanting at these ganglionic sites. A total of 7 patients received either trigeminal and/or SPG stimulation with or without peripheral nerve stimulation, having failed multiple alternative modalities of treatment. The treatments were tailored on the physical location of pain to ensure regional coverage with the stimulation. Fluoroscopy or frameless stereotaxy was utilized to place the SPG and/or trigeminal ganglion stimulator. All patients were initially trialed before implantation. Trial leads implanted in the pterygopalatine fossa near the SPG were implanted via transpterygoid (lateral-medial, infra-zygomatic) approach. Trial leads were implanted in the trigeminal ganglion via percutaneous Hartel approach, all of which resulted in masseter contraction. Patients who developed clinically significant pain improvement underwent implantation. The trigeminal ganglion stimulation permanent implants involved placing a grid electrode over Meckel's cave via sub-temporal craniotomy, which offered a greater ability to stimulate subdivisions of the trigeminal nerve, without muscular (V3) side effects; 2 of the 7 overall patients did not respond well to the trial and were not implanted; 5 patients reported pain relief with up to 24-month follow-up. Several of the SPG stimulation patients had pain relief without any paresthesias. There were no electrode migrations or post-surgical complications. The authors concluded that refractory facial pain may respond positively to ganglionic forms of stimulation. It appeared safe and durable to implant electrodes in the
pterygopalatine fossa via a lateral transpterygoid approach. Also, implantation of an electrode grid overlying Meckel's cave appeared to be a feasible alternative to the Hartel approach. They stated that further investigation is needed to evaluate the usefulness of these approaches for various facial pain conditions.

Maniam et al (2016) stated that craniofacial pain, including TN, TNP, and PIFP, is difficult to treat and can have severe implications for suffering in patients afflicted with these conditions. In recent years, clinicians have moved beyond treating solely with pharmacological therapies, which are generally not very effective, and focused on new interventional pain procedures. These procedures have evolved as technology has advanced, and thus far, early results have demonstrated effectiveness in small patient cohorts with a variety of craniofacial pain states. Some of the most promising interventional pain procedures include deep brain stimulation, high-frequency spinal cord stimulation, PNFS, and SPG stimulation. These researchers focused on a better understanding of craniofacial pain and emerging interventional pain therapies. With the advent of newer miniature wireless devices and less invasive implantation techniques, this should allow for more widespread use of neurostimulation as a therapeutic modality for treating craniofacial pain. The authors concluded that larger studies would aid in best practice strategies vis-à-vis traditional pharmacological therapies and emerging interventional pain techniques.
Jakobs and colleagues (2016) stated that neurosurgical pain management of drug-resistant TN is highly challenging. Microvascular decompression is a first-line neurosurgical approach for classical TN with neurovascular conflict, but can show clinical relapse despite proper decompression. Second-line destructive techniques like RF thermos-coagulation have become reluctantly used due to their potential for irreversible side effects. Subcutaneous PNFS (sPNFS) is a minimally invasive neuro-modulatory technique which has been shown to be effective for chronic localized pain conditions. Reports on sPNFS for the treatment of trigeminal pain are still sparse and primarily focused on pain intensity as outcome measure. Detailed data on the impact of sPNFS on attack frequency are currently not available. Patients were classified according to the International Headache Society classification (ICHD-3-beta); 3 patients had classical TN without (n = 3) and another 3 TN with concomitant persistent facial pain (n = 3); 2 patients suffered from post-herpetic trigeminal neuropathy (n = 2). All 8 patients underwent a trial stimulation of at least 7 days with subcutaneous leads in the affected trigeminal area connected to an external neurostimulator. Of those, 6 patients received permanent implantation of a neurostimulator. During the follow-up (6 to 29 months, mean of 15.2), VAS-scores, attack frequencies, oral drug intake, complications and side effects were documented; 7 of 8 patients responded to sPNFS (i.e., greater than or equal to 50 % pain reduction) during the test trial. The pain intensity (according to VAS) was reduced by 83 ± 16 % (mean ± SD) and the number of attacks decreased by 73 ± 26 % (mean ± SD); 5 of 6
patients were able to reduce or stop pain medication; 1 patient developed device infection; 2 patients developed stimulation-related side effects that could be resolved by reprogramming. The authors concluded that treatment by sPNFS is a beneficial option for patients with refractory trigeminal pain. Moreover, they stated that prospective randomized trials are needed to systematically evaluate the safety and effectiveness of this low-invasive neurosurgical technique.

**Trigeminal Tractotomy:**

Cetas et al (2008) stated that non-malignant pain has been treated in the past century with ablative, or more appropriately, destructive procedures. Although individual outcomes for these procedures have previously been described in the literature, to the authors' knowledge this was the first comprehensive and systematic review on this topic. A US National Library of Medicine PubMed search was conducted for the following ablative procedures: cingulotomy, cordotomy, DREZ (also input as dorsal root entry zone), ganglionectomy, mesencephalotomy, myelotomy, neurotomy, rhizotomy, sympathectomy, thalamotomy, and tractotomy. Articles related to pain resulting from malignancy and those not in peer-reviewed journals were excluded. In reviewing pertinent articles, focus was placed on patient number, outcome, and follow-up. A total of 146 articles was included in the review. The large majority of studies (131) constituted Class III evidence; 11 Class I and 4 Class II studies were found, of which nearly all (13 of 15) evaluated radiofrequency rhizotomies for different pain
origins, including lumbar facet syndrome, cervical facet pain, and type I or typical trigeminal neuralgia. Overall, support for ablative procedures for non-malignant pain is derived almost entirely from Class III evidence; despite a long history of use in neurosurgery, the evidence supporting destructive procedures for benign pain conditions remains limited. The authors concluded that newly designed prospective standardized studies are needed to define surgical indications and outcomes for these procedures, to provide more systematic review, and to advance the field.

Kahilogullari et al (2010) stated that TN pain secondary to bee sting has not been reported previously in the literature. These investigators reported the case of a 52-year old male patient with right-sided trigeminal neuropathic pain that began a month earlier, following a honeybee sting to the right forehead. The patient was successfully treated by computed tomography (CT)-guided percutaneous trigeminal tractotomy. The present report showed that a honeybee sting may result in trigeminal neuropathic pain and CT-guided percutaneous trigeminal tractotomy is effective in the treatment of such cases.

Jiang et al (2010) examined the feasibility of surface tractotomy of trigeminal nerve sensory root (STS) for the treatment of TN. A total of 7 patients with TN were operated on using the STS; 6 patients were followed-up for 4.8 to 9.8 years. In addition, these researchers performed micro-anatomical studies using paraffin embedding and hematoxylin-eosin staining technique on the trigeminal nerve root (TNR)
obtained from 30 cadavers. Clinically, patients' symptoms (e.g., face ache) disappeared after the surface nerve fiber bundles of trigeminal nerve sensory root (TNSR) were severed. Only 1 patient died of brainstem bleeding on post-operative day 18. Histological examination: The common type of sensory root fibers were arranged parallel for 3 to 6 mm at its exit of brainstem, and then the glial myelin transformed to Schwann cells. The axon bifurcated from outer layer to middle region, and gradually formed the tiny nerve fiber bundles in the surface layer and the giant nerve fiber bundles in the center of the root. The authors concluded that TN can be cured by STS without impairing nerve functions. They stated that this new approach is an effective, advanced surgical technique for TN treatment.

Ibrahim et al (2015) presented a novel technique, ventral pontine trigeminal tractotomy via retrosigmoid craniectomy, as an adjunct treatment in TN when there is no significant neurovascular compression. These researchers presented a non-randomized, retrospective comparison between 50 patients who lacked clear or impressive arterial neurovascular compression of the trigeminal nerve as judged by pre-operative magnetic resonance imaging and intra-operative observations. These patients had intractable TN unresponsive to previous treatment. Trigeminal tractotomy was performed either alone or in conjunction with microvascular decompression. Stereotactic neuro-navigation was used during surgery to localize the descending tract via a ventral pontine approach for descending tractotomy; follow-up was a mean of 44 months. At first
follow-up, 80% of patients experienced complete relief of their pain, and 18% had partial relief. At the most recent follow-up, 74% of patients were considered a successful outcome; only 1 (2%) patient had no relief after trigeminal tractotomy. Of those with multiple sclerosis-related TN, 87.5% experienced successful relief of pain at their latest follow-up. The authors concluded that while patient selection is a significant challenge, this procedure represents an option for patients with TN who have absent or equivocal neurovascular compression, multiple sclerosis-related TN, or recurrent TN.

Furthermore, an UpToDate review on “Trigeminal neuralgia” (Bajwa et al, 2016) does not mention trigeminal tractotomy as a therapeutic option.

The current evidence supporting the use of trigeminal tractotomy for the treatment of TN is mainly based on case-series studies. Well-designed studies are need to validate the effectiveness of this approach.

Intra-Arterial Modulation of the Trigeminal Nerve Ganglion:

Qureshi and colleagues (2018) stated that nerves and nerve ganglions are supplied by segmental arteries and the vasa nervorum, but the intra-arterial route has not been used for diagnostic or therapeutic purposes. These investigators presented the results of intra-arterial delivery of medication for modulating trigeminal nerve ganglion function in patients with refractory TN. They administered intra-arterial lidocaine in
doses up to 50 mg in the middle meningeal artery territory adjacent to the arterial branch that supplies the trigeminal nerve ganglion. These researchers performed electrophysiologic monitoring to serially assess the latency and amplitude of R1 and R2 responses in the blink reflex before and concurrent with each incremental dose of lidocaine. Clinical outcome assessment included a 10-point numeric rating, 4-point severity grading, and the pain-free time interval pre- and post-treatment. Intra-arterial lidocaine was administered to 3 patients with TN (35-year old woman, 57-year old man, and 34-year old woman). In all patients, there was a latency prolongation and amplitude reduction of R1 or R2 responses or both that was evident after 5 to 10 mg of lidocaine administration; a more pronounced effect was seen with increasing doses. The 2nd and 3rd patients reported improvement in pain severity on all scales with pain-free intervals of 5 and 3 days, respectively. There was improvement in facial hyperalgesia in all 3 patients in all dermatomes. All 3 patients' symptoms had returned to baseline severity 1 month later. The authors found that modulation of trigeminal nerve activity via the intra-arterial route is possible based on consistent intra-procedural electrophysiologic suppression and short-term clinical improvement in patients with refractory TN. Moreover, they stated that this procedure needs more investigation and should only be considered for patients with therapy-refractory TN, due to the risk profile of the procedure and the short duration of pain relief.

The authors also acknowledged that the lack of electrophysiologic data limited their ability to
define the duration of R1 and R2 response alterations after lidocaine administration. They stated that further studies would be necessary to determine if intra-arterial injection of longer acting anesthetic agents or chemotherapeutic agents in the middle meningeal artery has any value in either the diagnosis or treatment of TN.

Percutaneous Ozone Injection:

In a retrospective study, An and colleagues (2018) evaluated the therapeutic effect of CT-guided percutaneous ozone injection for the treatment of refractory TN. A total of 29 patients with a diagnosis of refractory TN were enrolled. All patients were treated with a percutaneous ozone injection and 1 patient was excluded. There were 21 patients with classical TN (group A) and 7 patients with painful trigeminal neuropathy caused by post-herpetic neuralgia (group B). The percutaneous injection was an oxygen-ozone mixture at an ozone concentration of 30 μg/ml into the Gasserian ganglion performed under CT guidance. The number of procedures performed varied from 1 to as many as 16. Outcomes were evaluated using VAS pain scores. The combined VAS scores were 7.11 ± 1.23 pre-treatment, 2.86 ± 1.69 post-treatment (p < 0.05) and 3.25 ± 2.01 after 6-month follow-up (p < 0.05). In group A, the VAS scores were 7.10 ± 1.04 pre-treatment and 2.90 ± 1.84 post-treatment (p < 0.05). In group B, the VAS scores were 7.14 ± 1.77 pre-treatment and 2.71 ± 1.25 post-treatment (p < 0.05). After 6-months follow-up, the VAS score was 3.38 ± 2.18 in group A and 2.86 ± 1.46 in group B, a decrease compared to pre-treatment (p < 0.05); VAS of Group A and B showed no difference not
only in pre-treatment, but also in post-treatment and follow-up. The authors concluded that percutaneous ozone injection is a safe and effective treatment for patients with refractory TN.

The authors stated that this study had several drawbacks. First, this study was retrospective in design. Out of 28 charts reviewed, clinical records were incomplete with data missing in 1 case (3.57 %) during therapy and follow-up period. Second, a large-scale longitudinal prospective study would confirm the validity of these findings. Third, follow-up time was limited to 6 months and was relatively short. Moreover, researchers could not control for other treatments such as transcutaneous electrical nerve stimulation (TENS) and physical therapy (PT) during follow-up. These treatments might have influenced the results of this study. Finally, contrast medium injected at the site of the procedure might have provided further validation of correct needle placement, but it may also have interfered with the therapy itself.

Radial Extracorporeal Shock Wave Therapy:

Zhang and co-workers (2017) reported on the case of a patient with primary TN who exhibited pain relief without medication after radial extracorporeal shock wave therapy (ESWT). The 52-year old woman had a 3-year history of primary TN, involving the right maxillary division (V2) and the mandibular division (V3). She became refractory to carbamazepine and exhibited hepatic dysfunction. She hence received 3,000 to 6,000 impulses of craniofacial radial ESWT to the region centered on the
surface projection of the trigeminal ganglion and pain areas at 10 Hz; the intensity ranged from 1.4 to 4.5 bars twice-weekly for 8 weeks. At baseline, and 1, 2, and 5 months after treatment, the Barrow Neurological Institute scores were IV, IIIa, II, and II, and the VAS were 8, 3, 1, and 1, respectively. No complications or adverse effects were observed. The hepatic function returned to normal after the discontinuation of carbamazepine. The authors concluded that this case report demonstrated the feasibility of radial EWT for primary TN without complications or adverse effects with careful regulation of the therapy intensity. These findings need to be validated by well-designed studies.

CPT Codes / HCPCS Codes / ICD-10 Codes

Information in the [brackets] below has been added for clarification purposes. Codes requiring a 7th character are represented by "+":

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>61450</td>
<td>Craniectomy, subtemporal, for section, compression, or decompression of sensory root of gasserian ganglion</td>
</tr>
<tr>
<td>61458</td>
<td>Craniectomy, suboccipital; for exploration or decompression of cranial nerves</td>
</tr>
<tr>
<td>61460</td>
<td>for section of one or more cranial nerves</td>
</tr>
<tr>
<td>61796</td>
<td>Stereotactic radiosurgery (particle beam, gamma ray, or linear accelerator); 1 simple cranial lesion</td>
</tr>
<tr>
<td>+ 61797</td>
<td>each additional cranial lesion, simple (List separately in addition to code for primary procedure)</td>
</tr>
<tr>
<td>Code</td>
<td>Code Description</td>
</tr>
<tr>
<td>--------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>61798</td>
<td>1 complex cranial lesion</td>
</tr>
<tr>
<td>+ 61799</td>
<td>each additional cranial lesion, complex (List separately in addition to code for primary procedure)</td>
</tr>
<tr>
<td>64400</td>
<td>Injection, anesthetic agent; trigeminal nerve, any division or branch</td>
</tr>
<tr>
<td>64600</td>
<td>Destruction by neurolytic agent; trigeminal nerve; supraorbital, infraorbital, mental, or inferior alveolar branch</td>
</tr>
<tr>
<td>64605</td>
<td>second and third division branches at foramen ovale</td>
</tr>
<tr>
<td>64610</td>
<td>second and third division branches at foramen ovale under radiologic monitoring</td>
</tr>
<tr>
<td>64612</td>
<td>Chemodenervation of muscle(s); muscle(s) innervated by facial nerve, unilateral (eg., for blepharospasm, hemifacial spasm)</td>
</tr>
<tr>
<td>64716</td>
<td>Neuroplasty and/or transposition; cranial nerve(specify)</td>
</tr>
<tr>
<td>64732</td>
<td>Transection or avulsion of; supraorbital nerve</td>
</tr>
<tr>
<td>64734</td>
<td>Transection or avulsion of; infraorbital nerve</td>
</tr>
</tbody>
</table>

CPT codes not covered for indications listed in the CPB:

Trigeminal Tractotomy:

No specific code

20926 | Tissue grafts, other (eg, paratenon, fat, dermis) |
<p>| 61850 | Twist drill or burr hole(s) for implantation of neurostimulator electrodes, cortical |</p>
<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>61860</td>
<td>Craniectomy or craniotomy for implantation of neurostimulator electrodes, cerebral, cortical</td>
</tr>
<tr>
<td>61885</td>
<td>Insertion or replacement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to a single electrode array</td>
</tr>
<tr>
<td>61886</td>
<td>with connection to two or more electrode arrays</td>
</tr>
<tr>
<td>64640</td>
<td>Destruction by neurolytic agent; other peripheral nerve or branch</td>
</tr>
<tr>
<td>64553</td>
<td>Percutaneous Implantation of neuromuscular electrode; cranial nerve</td>
</tr>
<tr>
<td>95961</td>
<td>Functional cortical and subcortical mapping by stimulation and/or recording of electrodes on brain surface, or of depth electrodes, to provoke seizures or identify vital brain structures; initial hour of attendance by a physician or other qualified health care professional</td>
</tr>
<tr>
<td>+ 95962</td>
<td>each additional hour of attendance by a physician or other qualified health care professional (List separately in addition to code for primary procedure)</td>
</tr>
<tr>
<td>Code</td>
<td>Code Description</td>
</tr>
<tr>
<td>--------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>95970</td>
<td>Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude and 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, autonomic nerve, neuromuscular) neurostimulator pulse generator/transmitter, without reprogramming</td>
</tr>
</tbody>
</table>

**Other CPT codes related to the CPB:**

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>96365-96368</td>
<td>Intravenous infusion, for therapy, prophylaxis, or diagnosis (specify substance or drug)</td>
</tr>
</tbody>
</table>

**HCPCS codes covered if selection criteria are met:**

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>G0173</td>
<td>Linear accelerator based stereotactic radiosurgery, complete course of therapy in one session</td>
</tr>
<tr>
<td>G0251</td>
<td>Linear accelerator based stereotactic radiosurgery, delivery including collimator changes and custom plugging, fractionated treatment, all lesions, per session, maximum 5 sessions per course of treatment</td>
</tr>
<tr>
<td>G0339</td>
<td>Image guided robotic linear accelerator-based stereotactic radiosurgery, complete course of therapy in one session, or first session of fractionated treatment</td>
</tr>
<tr>
<td>Code</td>
<td>Code Description</td>
</tr>
<tr>
<td>--------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>G0340</td>
<td>Image guided robotic linear accelerator-based stereotactic radiosurgery, delivery including collimator changes and custom plugging, fractionated treatment, all lesions, per session, second through fifth sessions, maximum 5 sessions per course of treatment</td>
</tr>
</tbody>
</table>

HCPCS codes not covered for indications listed in the CPB:

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1767</td>
<td>Generator, neurostimulator (implantable), nonrechargeable</td>
</tr>
<tr>
<td>C1770</td>
<td>Imaging coil, magnetic resonance (insertable)</td>
</tr>
<tr>
<td>C1778</td>
<td>Lead, neurostimulator (implantable)</td>
</tr>
<tr>
<td>C1787</td>
<td>Patient programmer, neurostimulator</td>
</tr>
<tr>
<td>C1816</td>
<td>Receiver and/or transmitter, neurostimulator (implantable)</td>
</tr>
<tr>
<td>C1820</td>
<td>Generator, neurostimulator (implantable), non high-frequency with rechargeable battery and charging system</td>
</tr>
<tr>
<td>C1883</td>
<td>Adaptor/extension, pacing lead or neurostimulator lead (implantable)</td>
</tr>
<tr>
<td>C1897</td>
<td>Lead, neurostimulator test kit (implantable)</td>
</tr>
<tr>
<td>E0745</td>
<td>Neuromuscular stimulator, electronic shock unit</td>
</tr>
<tr>
<td>J0585</td>
<td>Injection, onabotulinumtoxinA, 1 unit</td>
</tr>
<tr>
<td>J0586</td>
<td>Injection, abobotulinumtoxinA, 5 units</td>
</tr>
<tr>
<td>J0587</td>
<td>Injection, rimabotulinumtoxinB, 100 units</td>
</tr>
<tr>
<td>J0588</td>
<td>Injection, incobotulinumtoxinA, 1 unit</td>
</tr>
<tr>
<td>J2001</td>
<td>Injection, lidocaine HCl for intravenous infusion, 10mg</td>
</tr>
<tr>
<td>J3475</td>
<td>Injection, magnesium sulfate, per 500 mg</td>
</tr>
<tr>
<td>Code</td>
<td>Code Description</td>
</tr>
<tr>
<td>--------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>L8680</td>
<td>Implantable neurostimulator electrode, each</td>
</tr>
<tr>
<td>L8681</td>
<td>Patient programmer (external) for use with implantable programmable neurostimulator pulse generator, replacement only</td>
</tr>
<tr>
<td>L8682</td>
<td>Implantable neurostimulator radiofrequency receiver</td>
</tr>
<tr>
<td>L8683</td>
<td>Radiofrequency transmitter (external) for use with implantable neurostimulator radiofrequency receiver</td>
</tr>
<tr>
<td>L8685</td>
<td>Implantable neurostimulator pulse generator, single array, rechargeable, includes extension</td>
</tr>
<tr>
<td>L8686</td>
<td>Implantable neurostimulator pulse generator, single array, non-rechargeable, includes extension</td>
</tr>
<tr>
<td>L8687</td>
<td>Implantable neurostimulator pulse generator, dual array, rechargeable, includes extension</td>
</tr>
<tr>
<td>L8688</td>
<td>Implantable neurostimulator pulse generator, dual array, non-rechargeable, includes extension</td>
</tr>
<tr>
<td>L8689</td>
<td>External recharging system for battery (internal) for use with implantable neurostimulator, replacement only</td>
</tr>
<tr>
<td>L8695</td>
<td>External recharging system for battery (external) for use with implantable neurostimulator, replacement only</td>
</tr>
</tbody>
</table>

Other HCPCS codes related to the CPB:

<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>J0475</td>
<td>Injection baclofen, 10 mg</td>
</tr>
<tr>
<td>J1165</td>
<td>Injection, phenytoin sodium, per 50 mg</td>
</tr>
</tbody>
</table>

ICD-10 codes covered if selection criteria are met:
<table>
<thead>
<tr>
<th>Code</th>
<th>Code Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>G50.0</td>
<td>Trigeminal neuralgia</td>
</tr>
</tbody>
</table>

The above policy is based on the following references:


41. IRSA. Stereotactic radiosurgery for patients with intractable typical trigeminal neuralgia.
who have failed medical management.
Radiosurgery Practice Guideline Report No.


47. Bajwa ZH, Ho CC, Khan SA. Trigeminal neuralgia. Last reviewed February 2013. UpToDate Inc. Waltham, MA.


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in trigeminal neuralgia: A systematic review.

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Salvage treatment of trigeminal neuralgia by occipital nerve stimulation. Cephalalgia.

52. Tamburin S, Schweiger V, Magrinelli F, et al.
Effect of 5% lidocaine medicated plaster on pain intensity and paroxysms in classical trigeminal neuralgia. Ann Pharmacother.

53. Stavropoulou E, Zis P, Vadalouca A. The use of intravenous lidocaine in trigeminal neuralgia pain relief and palliative care unit.

2014;121 Suppl:210-221.


56. Bajwa CH, Ho CC, Khan SA. Trigeminal neuralgia. UpToDate Inc., Waltham, MA. Last reviewed February 2015.


67. Bajwa CH, Ho CC, Khan SA. Trigeminal neuralgia. UpToDate [online serial]. Waltham, MA: UpToDate; reviewed February 2016.


There are no amendments for Medicaid.