

Medical Policy



Title: Surgical Deactivation of Headache Trigger Sites

Professional / Institutional
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Populations	Interventions	Comparators	Outcomes
Individuals: • With migraine headaches	Interventions of interest are: • Surgical deactivation of headache trigger sites	Comparators of interest are: • Optimal medical management • Botulinum toxin	Relevant outcomes include: • Symptoms • Change in disease status • Quality of life • Treatment-related morbidity
Individuals: • With non-migraine headaches	Interventions of interest are: • Surgical deactivation of headache trigger sites	Comparators of interest are: • Optimal medical management	Relevant outcomes include: • Symptoms • Change in disease status • Quality of life • Treatment-related morbidity

DESCRIPTION

Migraine is a common headache disorder that is treated using various medications, which can be taken at the onset of an attack and/or for migraine prophylaxis. Other treatments include behavioral treatments and botulinum toxin injections. Surgical deactivation of trigger sites is another proposed treatment. Surgical deactivation is based on the theory that migraine headaches arise due to inflammation of the trigeminal nerve branches in the head and neck and that specific trigger sites can be identified in individual patients. Surgical deactivation has also been proposed for other types of headaches (eg, tension headaches).

OBJECTIVE

The objective of this evidence review is to evaluate whether surgical deactivation of trigger sites as a treatment for migraine headache or non-migraine headache improves net health outcomes.

BACKGROUND**Migraine Headache**

Migraine is a common headache disorder with a prevalence in the United States of approximately 18% in women and 6% in men.¹ According to the International Headache Society (2018), migraine headache is a recurrent disorder with attacks lasting 4 to 72 hours.² Typical features of migraine headaches include unilateral location, pulsating quality, moderate or severe intensity, and associated symptoms such as nausea, photophobia, and/or phonophobia.

Treatment

A variety of medications are used to treat acute migraine episodes. These include medications taken at the onset of an attack to abort the attack (eg, triptans, ergotamines, and certain calcitonin gene-related peptide [CGRP] receptor antagonists), and medications to treat the pain and other symptoms of migraines once they are established (eg, non-opioid analgesics, antiemetics). Prophylactic medication therapy (eg, certain antidepressants, beta-blockers, and anti-seizure medications) may be appropriate for people with migraines that occur more than 2 days per week. Onabotulinumtoxin A and several CGRP receptor antagonists have also been approved by the U.S. Food and Drug Administration (FDA) as prophylactic treatments for episodic and/or chronic migraines. In addition to medication, behavioral treatments such as relaxation and cognitive therapy are used to manage migraine headache.

Surgical Deactivation

Surgical deactivation of trigger sites is another proposed treatment of migraine headache. The procedure was developed by a plastic surgeon (Bahman Guyuron, MD), following observations that some patients who had cosmetic forehead lifts reported improvement or elimination of migraine symptoms postsurgery.^{3,4} The procedure is based on the theory that migraine headaches arise due to inflammation of trigeminal nerve branches in the head and neck caused by irritation of the surrounding musculature, bony foramen, and perhaps fascia bands. Accordingly, surgical treatment of migraines involves removing the relevant nerve sections, muscles, fascia, and/or vessels. The treatment is also based on the theory there are specific migraine trigger sites and that these sites can be located in individual patients. In studies conducted by Guyuron's research group, clinical evaluation and diagnostic injections of botulinum

toxin have been used to locate trigger sites. The specific surgical procedure varies according to the patient's migraine trigger site. The surgical procedures are performed under general anesthesia in an ambulatory care setting and take an average of 1 hour.

Surgical procedures have been developed at 4 trigger sites: frontal, temporal, rhinogenic, and occipital. Frontal headaches are believed to be activated by irritation of the supratrochlear and suborbital nerves by glabellar muscles or vessels. The surgical procedure involves the removal of the glabellar muscles encasing these nerves. Fat from the upper eyelid is used to fill the defect in the muscles and shield the nerve. Temporal headaches may be activated by inflammation of the zygomatico-temporal branch of the trigeminal nerve by the temporalis muscles or vessels adjacent to the nerve. To treat migraines located at this trigger site, a segment ($\gg 2.5$ cm) of the zygomatico-temporal branch of the trigeminal nerve is removed endoscopically. Rhinogenic headaches may involve intranasal abnormalities (eg, deviated septum), which may irritate the end branches of the trigeminal nerve. Surgical treatment includes septoplasty and turbinectomy. Finally, occipital headaches may be triggered by irritation of the occipital nerve caused by the semispinalis capitis muscle or the occipital artery. Surgery consists of removal of a segment of the semispinalis capitis muscle medial to the greater occipital nerve approximately 1 cm wide and 2.5 cm long, followed by insertion of a subcutaneous flap between the nerve and the muscle to avoid nerve impingement.

Non-Migraine Headache

It has been proposed that other types of headaches (eg, tension headaches) may also be triggered by irritation of the trigeminal nerve.

Treatment

Although the mechanism of action is less well established for headaches other than migraine, it is possible that surgical treatment of trigger sites may also be beneficial for some non-migraine headaches.

REGULATORY STATUS

Surgical deactivation of headache triggers is a surgical procedure and, as such, is not subject to regulation by the FDA.

POLICY

Surgical deactivation of trigger sites is considered **experimental / investigational** for the treatment of migraine and non-migraine headache.

POLICY GUIDELINES

International Headache Society (IHS) Classification II criteria (3rd edition, 2018) are listed in Table PG1.

Table PG1. International Headache Society Classification Criteria for Migraines

Classification Criteria
Migraine without aura
Description
Recurrent headache disorder characterized by attacks lasting 4-72 hours.
Diagnostic criteria
A. At least five attacks fulfilling criteria B-D
B. Headache attacks lasting 4-72 hours (untreated or successfully treated)
C. At least two of the following four characteristics: <ol style="list-style-type: none"> 1. unilateral location 2. pulsating quality 3. moderate or severe pain intensity 4. aggravation by or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
D. During headache, at least one of the following: <ol style="list-style-type: none"> 1. nausea and/or vomiting 2. photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis
Migraine with aura
Description
Recurrent attacks, lasting minutes, of unilateral fully reversible visual, sensory or other central nervous system symptoms that usually develop gradually and are usually followed by headache and associated migraine symptoms.
Diagnostic criteria
A. At least two attacks fulfilling criteria B and C
B. One or more of the following fully reversible aura symptoms: <ol style="list-style-type: none"> 1. visual 2. sensory 3. speech and/or language 4. motor 5. brainstem 6. retinal
C. At least three of the following six characteristics: <ol style="list-style-type: none"> 1. at least one aura symptom spreads gradually over ≥ 5 minutes, 2. two or more aura symptoms occur in succession 3. each individual aura symptom lasts 5-60 minutes 4. at least one aura symptom is unilateral 5. at least one aura symptom is positive 6. the aura is accompanied, or followed within 60 minutes, by headache
D. Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded.

Adapted from Headache Classification Committee of the International Headache Society 2018 (available at <http://www.ihs-headache.org/ichd-guidelines>).
ICHD-3: International Classification of Headache Disorders, 3rd edition.

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RATIONALE

The evidence review was created using searches of the PubMed database. The most recent literature update was performed through January 2, 2026.

Evidence reviews assess the clinical evidence to determine whether the use of technology improves the net health outcome. Broadly defined, health outcomes are the 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 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 technology, 2 domains are examined: the relevance, and quality and credibility. To be relevant, studies must represent 1 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. Randomized controlled trials 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.

MIGRAINE AND NON-MIGRAINE HEADACHES

Clinical Context and Therapy Purpose

The purpose of surgical deactivation as a treatment for migraine or non-migraine headache is to provide a treatment option that is an alternative to or an improvement on existing therapies. The following PICO was used to select literature to inform this review.

Populations

The relevant population of interest is individuals with migraine or non-migraine headache refractory to medical therapy.

Interventions

The therapy being considered is surgical deactivation for the treatment of migraine or non-migraine headache. The specific surgical procedure varies according to the patient's migraine

trigger site. Surgical procedures have been developed at 4 trigger sites: frontal, temporal, rhinogenic, and occipital.

Comparators

The following practices are currently being used to treat migraine and non-migraine headache: a variety of medications are used to treat acute migraine episodes. These include medications taken at the onset of an attack to abort the attack (eg, triptans, ergotamines, and certain calcitonin gene-related peptide [CGRP] receptor antagonists), and medications to treat the pain and other symptoms of migraines once they are established (eg, non-opioid analgesics, antiemetics). Prophylactic medication therapy (eg, certain antidepressants, beta-blockers, and anti-seizure medications) may be appropriate for people with migraines that occur more than 2 days per week. Onabotulinumtoxin A and several CGRP receptor antagonists have also been approved by the U.S. Food and Drug Administration (FDA) as prophylactic treatments for episodic and/or chronic migraines. In addition to medication, behavioral treatments such as relaxation and cognitive therapy are used to manage migraine headache.

Outcomes

The general outcomes of interest are migraine intensity and frequency, the effect of the migraines or treatment on quality of life as measured by instruments such as the 12-Item Short Form Health Survey (additional examples described in Table 1), hospitalizations due to migraine, and adverse effects of the treatment. Migraine severity and frequency are measured over 6 to 12 months.

Table 1. Self-Reported Outcome Measures

Outcome Measure	Abbreviation	Description
Monthly Migraine Days	MMD	The average number of days that there is onset or continuation of a migraine headache. Outcomes are typically reported as a decrease in MMD.
50% Decrease in MMD	50% MMD	The proportion of people who achieve a decrease of 50% in MMD. Also frequently reported are 75% and 100% decrease in MMD.
Migraine Disability Assessment ⁵ ,	MIDAS	Report on the number of days that a headache has impacted function at home, work, or school.
Headache Impact Test ⁶ ,	HIT-6	Six item measure of the impact of headache on social, role, and cognitive function and psychological distress.
Migraine Specific Quality of Life Questionnaire ⁷ ,	MSQL	Migraine specific quality of life questionnaire.

Study Selection Criteria

Methodologically credible studies were selected using the following principles:

- a. To assess efficacy outcomes, comparative controlled prospective trials were sought, with a preference for RCTs;

- b. In the absence of such trials, comparative observational studies were sought, with a preference for prospective studies.
- c. To assess long-term outcomes and adverse events, single-arm studies that capture longer periods of follow-up and/or larger populations were sought.
- d. Studies with duplicative or overlapping populations were excluded.

REVIEW OF EVIDENCE

MIGRAINE HEADACHE

Randomized Clinical Trials

The initial RCT assessing surgical deactivation of migraine trigger sites was published by Guyuron et al (2005); this unblinded trial did not include a sham control.⁸ Eligibility included a diagnosis of migraine headache using the International Classification of Headache Disorders II (ICHD-II) criteria. Patients were assigned to the treatment group (n=100) or to the control group (n=25) in a 4:1 allocation. Active treatment patients received up to 3 injections of botulinum toxin type A (Botox), 1 at each of their most common trigger sites, to identify a predominant site of headache trigger and potential response to treatment. To be considered candidates for surgery, patients had to have at least a 50% reduction in symptoms for 4 weeks after a botulinum toxin type A injection. Patients in the control group received saline injections instead of botulinum toxin and were ineligible for surgery; for the remainder of the treatment period, the patients received usual care. For patients in the intervention group, surgery varied by trigger site. For example, for patients with a predominantly frontal trigger migraine headache, the glabellar muscle group was removed to relieve compression of the supraorbital and supratrochlear nerves; for those with a temporal migraine headache, 3 cm of the zygomatico-temporal branch of the trigeminal nerve was removed; patients with both temporal and frontal migraine headaches underwent both procedures. Among treatment group, 91 responded to botulinum toxin type A injection and underwent surgery and 89 (89%) of 100 completed the 12-month follow-up. There was a differential dropout in the 2 groups: 19 (76%) of 25 patients in the control group were evaluated at 12 months. A total of 17 (14%) of 125 randomized patients were excluded from the analysis. In a per-protocol analysis at 12 months, 82 (92%) of 89 patients in the treatment group and 3 (16%) of 19 in the control group experienced significant improvement, defined as at least a 50% reduction in baseline migraine frequency, intensity, or duration. The difference between groups was statistically significant ($p < 0.001$). Thirty-one (35%) patients in the treatment group and none in the control group reported complete elimination of migraines. Most adverse events following surgery were minor and transient. The most commonly reported events were temporary nasal dryness (n=12) and rhinorrhea (n=11). Seven patients experienced intense scalp itching that lasted a mean of 6 months. Five-year outcomes for patients in the treatment group were reported by Guyuron et al. (2011).⁹ Follow-up data were available for 79 patients (87% of those who underwent surgery, 79% of those randomized to the treatment group). Outcomes were reported for 69 patients. The other 10 had received additional migraine headache surgery and were excluded from the analysis. At 5 years, 20 (29%) of 69 reported complete elimination of migraine headache, 41 (59%) reported a significant decrease in symptoms, and 8 (12%) reported no significant change. All measured variables improved significantly at 5 years compared with baseline. For example, mean headache frequency per month decreased from 10.9 to 4.0 ($p < 0.001$). Long-term data were not reported for the control group. Limitations of the 2005 RCT included lack of blinding, lack of a sham control, and randomization before determining eligibility for surgery. In addition, there was a potential cointervention bias: the surgery group

but not the sham group received botulinum toxin injections, which might have had a therapeutic effect. Moreover, about 14% of patients were excluded from the analysis, which could have biased results. Furthermore, findings were not reported separately by surgical procedure. In terms of long-term follow-up, 5-year data were reported only for the treatment group.

Guyuron et al (2009) published a double-blind, sham-controlled trial evaluating surgical deactivation of migraine trigger sites in 76 patients.³ Eligibility criteria included a diagnosis of migraine headache according to ICHD-II criteria¹⁰, and headaches triggered from a single or predominant site, as determined by a headache diary and physical examination. Participants were then given an injection of botulinum toxin type A (Botox) at the prominent site from which migraine pain started. Patients who had a positive response to botulinum toxin type A (ie, at least a 50% decrease in headache symptoms) and in whom headaches recurred after the effect of the botulinum toxin had disappeared were eligible for randomization. The methodology differed in this trial from that of the 2005 RCT (previously described), which randomized patients before receiving diagnostic botulinum toxin type A injections. In addition, Liu et al (2012), (Guyuron coauthored this study), further investigated the method of botulinum toxin injections to select patients for deactivation surgery and found that outcomes were similar in migraine surgery patients who did and did not undergo diagnostic Botox injections.⁴ The Liu et al. (2012) analysis raises questions about the need for the complex patient selection process used in the published RCTs. In the 2009 RCT, participants were stratified by the predominant site from which headaches were triggered, frontal, temporal, or occipital, and were randomized 2:1 to active or to sham surgery. A total of 317 participants were screened for inclusion; 130 received botulinum toxin type A injections and, based on responses to the injections, 76 were considered eligible for randomization. In each of the 3 active treatment groups, surgery consisted of exposure and removal of nerves and/or muscles. For patients in the sham group, surgery was limited to exposing the nerves and/or muscles; the integrity of the structures was left intact. The procedures differed according to the predominant headache trigger site and were similar to procedures used in the Guyuron et al. (2005) trial. Briefly, patients in the frontal active surgery group underwent removal of the glabellar muscles encasing the supraorbital and supratrochlear nerves. Patients in the temporal active surgery group underwent removal of a segment of the zygomatico-temporal branch of the trigeminal nerve. In the occipital surgery group, a segment of the semispinalis capitis muscle medial to the greater occipital nerve was removed. Patients kept headache diaries and were seen at 3, 6, 9, and 12 months post-surgery. Seventy-five of 76 patients (49 in the active treatment group, 26 in the sham group) completed the 1-year follow-up. There were 29 patients in the frontal group (19 active treatment, 10 sham), 28 in the temporal group (19 active treatment, 9 sham), and 18 in the occipital group (11 active treatment, 7 sham). Patients remained blinded to their group assignment through 12 months, at which time patients in the sham surgery group were offered the surgical procedure. Key results are displayed in Table 2. Note that, for the frequency, intensity, and duration variables, there were no statistically significant differences by trigger site, so overall results are displayed. Results for the same outcomes from the Guyuron et al. (2005) RCT are also summarized in Table 2. In addition to the between-group differences, there were statistically significant improvements in headache frequency, intensity, and duration from baseline to 12 months within the active surgery group and significant improvements in headache frequency and intensity within the sham surgery group. The improvement in outcomes within the sham group in the 2009 RCT was greater than those seen after usual care in the 2005 RCT, suggesting there might have been a substantial placebo effect associated with the surgery to deactivate trigger sites. No adverse events were reported in the sham surgery group. All patients in the active treatment group reported some

degree of paresthesia immediately after surgery. One patient experienced numbness 12 months after surgery. The most common adverse event in the active treatment group was temporal hollowing in 10 (53%) of 19 patients in the surgery group. Advantages of the 2009 study included a sham control group and blinded comparison of outcomes in the 2 groups through 12 months post-surgery. Study limitations included small numbers of patients in each subgroup and a lack of reporting patients' use of other migraine treatments (eg, botulinum toxin type A, medications) during the 12-month follow-up. In addition, patient selection involved a long multicomponent selection process, which may be impractical on a widespread basis.

Table 2. Summary of Outcomes for the Guyuron Trials

Outcome Measures	Guyuron et al (2009) ³			Guyuron et al (2005) ⁸		
	Active Surgery (n=49)	Sham Surgery (n=26)	p ^b	Active Surgery (n=89)	Usual Care (n=19)	p ^b
Completely eliminated headaches	28/49 (57.1)	1/26 (3.8)	<.001	31/89 (35)	0/19 (0)	<.001
Significant improvement ^a	41/49 (84)	15/26 (58)	.005	82/89 (92)	3/19 (16)	<.001
Mean headache frequency, mo Baseline (SD) 12 months (SD) ^c	9.9 (6.0) -7.4 (5.8)	9.5 (4.4) -3.5 (5.4)	.005	10.9 (0.8) 3.8 (0.4)	9.9 (1.7) 10.2 (1.7)	<.001
Mean headache intensity (1 to 10 VAS) Baseline (SD) 12 months (SD) ^c	6.2 (1.7) -3.0 (3.5)	5.5 (1.4) -1.3 (2.9)	.03	8.6 (0.13) 4.0 (0.3)	8.8 (0.24) 7.0 (0.3)	<.001
Mean headache duration Baseline (SD) 12 months (SD) ^c	0.5 (0.6) -0.3 (0.5)	1.7 (5.6) -0.9 (4.5)	.43	1.4 (0.14) 0.4 (0.05)	1.3 (0.25) 1.0 (0.2)	.007

Values are n/N (%) unless otherwise noted.

SD: standard deviation; VAS: visual analog scale.

^a Significant improvement defined as at least a 50% reduction in migraine frequency, intensity, or duration versus baseline.

^b Between-group p values.

^c In the 2009 study, results are reported as change from baseline.

A 2014 review article critically evaluated the RCTs on surgical deactivation of migraine trigger sites and raised a number of important concerns.¹¹ The authors of the sham-controlled trial did not mention patients' use of other headache treatments. Postoperative use of medications could have resulted in a reduction in headache frequency; these cases would have been counted as a surgical success in the study. In the sham-controlled trial, baseline headache frequency was 9.9 migraines per month in the intervention group and 9.5 migraines per month in the control group and, therefore, the reduction of a small number of migraine episodes per month (which might not be clinically significant) could be considered a surgical success based on the author's criterion of a 50% decrease in frequency. Use of the terminology "migraine headaches per month" does not provide information on the number of days per month with migraine headaches or the number of non-migraine headaches per month. Patients in the sham group might have guessed their group assignment because of retained movement of the corrugator supercilii, depressor supercilii, and procerus muscles. This could have biased their responses to subjective outcome

questions. Botulinum toxin type A (Botox) injection is a nonspecific screening tool and can lead to false- positives when used to select patients for migraine surgery because the injections into the peripheral nerves may also modulate pain at central targets.

Omrarifard et al (2016) published an RCT comparing surgical deactivation of migraine trigger sites with medical treatment in 50 patients from a single center in Iran.¹² The trial did not include a sham control and patients were not blinded to treatment group. Patients met ICHD diagnostic criteria for migraine headache and were asked about their most common migraine trigger sites. All patients received injections of botulinum toxin into the frontal, temporal, and occipital trigger sites in a stepwise manner, with the most common site injected first. Investigators did not state how they evaluated patients' responses to botulinum toxin or how patient responses to botulinum toxin affected their eligibility to participate in the trial. Patients in the medical treatment group (n=25) were prescribed propranolol (80 mg daily) and amitriptyline (100 mg daily). Patients assigned to the surgery group (n=25) underwent decompression surgery in 1 or any combination of 4 trigger sites (frontal, temporal, septum, and/or occipital) surgeons identified as relevant to their pattern of headaches. Surgical procedures were similar to those used in the Guyuron et al (2005, 2009) RCTs except that a septal surgery option was added. Trial findings are summarized in Table 3. All 12-month outcomes were significantly better in the surgery group than in the medical treatment group. No adverse events were reported. Interpreting trial findings were influenced by the lack of patient blinding, which raises concerns about subjective and patient-reported outcome measures. Results could have been influenced by the placebo effect. Moreover, it is not clear how patient outcomes data were collected (trialists did not mention patient diaries). Furthermore, surgeries differed by patient trigger sites, which makes it difficult to evaluate any particular surgical procedure.

Table 3. Summary of Outcomes for the Omranifard Trial

Outcome Measures	Surgery (n=25)	Medical Treatment (n=25)	p ^b
Completely eliminated headaches, n/N (%)	9/25 (36)	1/25 (4)	<.001
Success rate, n/N (%) ^a	19/25 (76)	10/25 (40)	<.001
Mean headache frequency, mo			<.001
Baseline (SD)	15.9 (3.3)	15.2 (3.1)	
12 months (SD)	6.4 (2.3)	10.5 (2.2)	
Mean headache intensity (1 to 10 VAS)			.001
Baseline (SD)	8.3 (0.3)	8.4 (0.3)	
12 months (SD)	4.1 (0.2)	6.0 (0.2)	
Mean headache duration, d			<.001
Baseline (SD)	1.1 (0.5)	1.0 (0.4)	
12 months (SD)	0.5 (0.3)	0.8 (0.3)	

Adapted from Omranifard et al (2016).¹²

SD: standard deviation; VAS: visual analog scale.

^a Success was defined as at least a 50% reduction in the migraine index score at 12 months versus baseline.

^b Between-group p values.

Section Summary: Migraine Headache

Three RCTs have evaluated surgical deactivation of headache trigger sites. One RCT was double-blind and sham-controlled and the other 2 did not use a sham control or blinded patients. All 3 reported statistically significantly better outcomes at 12 months in patients who received decompression surgery for migraine headache than the control intervention. However, the trials were subject to methodologic limitations (eg, variability in surgical procedures, the potential use of cointerventions, issues related to patient selection, outcome validation and measurement). In addition, in 2 trials patients were unblinded and findings subject to the placebo effect. Furthermore, all 3 were single-center and 2 were conducted by the same research group headed by the inventor of the procedure. Additional multicenter and sham-controlled randomized studies are needed.

Non-Migraine Headache

No studies were identified that have evaluated surgical deactivation of trigger sites as a treatment of non-migraine headache.

SUPPLEMENTAL INFORMATION

The purpose of the following information is to provide reference material. Inclusion does not imply endorsement or alignment with the evidence review conclusions.

Practice Guidelines and Position Statements

Guidelines or position statements will be considered for inclusion in 'Supplemental Information' if they were issued by, or jointly by, a US professional society, an international society with US representation, or National Institute for Health and Care Excellence (NICE). Priority will be given to guidelines that are informed by a systematic review, include strength of evidence ratings, and include a description of management of conflict of interest.

American Headache Society

The American Headache Society (2013) approved a list of 5 items that provide low value in headache medicine.¹³ This list was produced as part of the American Board of Internal Medicine Foundation's Choosing Wisely initiative. One of the 5 recommendations was: "Don't recommend surgical deactivation of migraine trigger points outside of a clinical trial." The 2013 document stated that the value of this procedure is still a research question and that large, multicenter randomized controlled trials with long-term follow-up are needed to provide accurate information on its benefits and harms.

U.S. Preventive Services Task Force Recommendations

Not applicable.

Ongoing and Unpublished Clinical Trials

As of January 2026, no ongoing or unpublished trials were identified that might influence this review.

CODING

The following codes for treatment and procedures applicable to this policy are included below for informational purposes. This may not be a comprehensive list of procedure codes applicable to this policy.

Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

The code(s) listed below are medically necessary ONLY if the procedure is performed according to the "Policy" section of this document.

There is no specific CPT code for this procedure but it might be reported using one of the following codes:

CPT/HCPCS	
15824	Rhytidectomy; forehead
15826	Rhytidectomy; glabellar frown lines
30130	Excision inferior turbinate, partial or complete, any method
30140	Submucous resection inferior turbinate, partial or complete, any method
30520	Septoplasty or submucous resection, with or without cartilage scoring, contouring or replacement with graft
64716	Neuroplasty and/or transposition; cranial nerve (specify)
64722	Decompression; unspecified nerve(s) (specify)
64771	Transection or avulsion of other cranial nerve, extradural
64772	Transection or avulsion of other spinal nerve, extradural
67900	Repair of brow ptosis (supraciliary, mid-forehead or coronal approach)

REVISIONS	
12-11-2014	Policy added to the bcbsks.com web site.
10-13-2015	Updated Description section.
	Updated Rationale section.
	Updated References section.
04-13-2016	Updated Description section.
	In Policy section: <ul style="list-style-type: none"> ▪ Removed "headache" to read "Surgical deactivation of trigger sites is considered experimental / investigational for the treatment of migraine and non-migraine headache."
	Updated Rationale section.
	Updated References section.
03-29-2017	Updated Description section.
	In Policy section: <ul style="list-style-type: none"> ▪ Under Policy Guidelines, updated International Headache Society classification criteria.
	Updated Rationale section.
	Updated References section.
03-28-2018	Updated Description section.

REVISIONS	
	In Policy section: <ul style="list-style-type: none"> ▪ Updated Policy Guidelines.
	Updated Rationale section.
	Updated References section.
03-27-2019	Updated Description section.
	Updated Rationale section.
01-01-2020	In Coding section: Added CPT Code: 15769
05-21-2021	Updated Description section
	In Policy guidelines: <ul style="list-style-type: none"> • Updated the International Headache Society Classification Criteria for Migraines from 2013 to 2018.
	Updated Rationale section
	In Coding section: <ul style="list-style-type: none"> • Removed CPT codes 15769 and 20926 • Added CPT code 64772
04-08-2022	Updated Description Section
	Updated Rationale Section
	Updated References Section
03-28-2023	Updated Description Section
	Updated Rationale Section
	Updated Coding Section <ul style="list-style-type: none"> ▪ Removed ICD-10 Diagnoses box
	Updated References Section
03-26-2024	Updated Description Section
	Updated Rationale Section
	Updated References Section
03-27-2025	Updated Description Section
	Updated Rationale Section
	Updated References Section
04-14-2026	Updated Description Section
	Updated Rationale Section
	Updated References Section

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2. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia.* Jan 2018; 38(1): 1-211. PMID 29368949
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