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SPECIAL TOPIC
Peripheral Nerve

Secondary Trigger Point Deactivation Surgery for Nerve Compression Headaches: A Scoping Review

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Background: Primary trigger point deactivation surgery has been successful in reducing or eliminating nerve compression headaches between 79% and 90% of the time. The aim of this review article was to discuss the factors that contribute to index trigger point deactivation surgery failure, the importance of reevaluating trigger points following failure, and the options for secondary surgery.

Methods: A literature search was performed using a combination of keywords involving "chronic headache" and "nerve deactivation surgery," in databases until February 2023.

Results: Data of 1071 patients were evaluated and included (11 articles). The failure rate after index trigger point deactivation surgery occurs is approximately 12%, primarily due to incomplete primary trigger point deactivation. Secondary trigger points may not appear until the primary trigger is eliminated, which occurs in 17.8% of patients. Reevaluation of previously diagnosed trigger points as well as uncovered trigger points and additional preoperative testing is indicated to help determine candidacy for further surgical deactivation. To address scarring that could contribute to failure, corticosteroid injection, acellular dermal matrix, adipofascial fat, or expanded polytetrafluoroethylene sleeves have been described with beneficial effects. For neuroma management, regenerative peripheral nerve interface, targeted muscle reinnervation, a combination of both, relocation nerve grafting, or nerve capping have also been described. Neurectomy can be performed when patients prefer anesthesia and/or paresthesia over current pain symptoms.

Conclusion: Secondary trigger point deactivation surgery is indicated when there is suspicion of incomplete deactivation, internal scarring, neuroma, or newly-diagnosed trigger points. *(Plast Reconstr Surg Glob Open 2024; 12:e5620; doi: 10.1097/GOX.0000000000005620; Published online 23 February 2024.)*

INTRODUCTION

Migraine headaches affect over 10% of the world's population,[1] leading to a high prevalence of depression and anxiety. This has a significant impact on the quality of life and socioeconomic burden.[2] Chronic migraine headaches refractory to medical management often require a multidisciplinary approach that includes neurologists, psychiatrists, and surgeons (eg, plastic surgeons, otolaryngologists, or neurosurgeons) to determine whether trigger point deactivation may be indicated.[3,4] This procedure involves the surgical deactivation of trigger points that often involve sensory branches of trigeminal and occipital nerves.[5] Surgical deactivation can be performed at frontal, temporal, occipital, rhinogenic, and nummular sites,[6–8] with success rates ranging between 79% and 90%[9] and has demonstrated overall safety and minimal complications.[10–13] Guyuron and colleagues reported their 5-year postoperative outcomes and found that 29% (20 patients) reported complete elimination of headache, 59% (41 patients) noticed a significant improvement (>50% improvement in frequency, intensity and/or duration), and 12% (eight patients) experienced no significant change.[14] Surgical failure has been associated with specific factors such as younger age of symptom onset, intraoperative excessive bleeding, and operating on two or fewer surgical sites (proxy for incomplete diagnosis of all relevant trigger points).[15,16] Moreover, complete elimination of pain in all patients is not realistic.[17] The

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aim of this review article was to discuss the factors that contribute to primary surgical failure and the importance of reevaluating both preexisting and new trigger points. Furthermore, the options and adjuncts for secondary surgery are outlined.

METHODS

Search Strategy

A literature search was conducted using PubMed, Medline, Cochrane, Web of Science, and Google Scholar databases to identify relevant articles through February 2023. We used the following search strategy: "chronic headache" OR "migraine headache" OR "migraine" AND "surgery" OR "nerve deactivation" OR "nerve decompression" OR "secondary surgery" OR "failed" OR "secondary deactivation surgery." The search strategy was designed to focus on outcomes after trigger point deactivation surgery (ie, primary inclusion criterion). Articles describing surgical techniques were also included to provide additional perspective. Other inclusion criteria were articles (1) written in English and (2) that had available abstracts. Articles describing chronic headache studies in animal models were excluded. Titles and abstracts were screened to determine whether studies met criteria, and the relevant articles were selected for full-text review. Two authors (S.S. and T.M.S.) independently reviewed search results. Additional articles retrieved from references or snowballing were also included.

RESULTS

A total of 146 articles were screened on title and abstract. Of these, 89 were screened on full text. Data of 1071 patients were evaluated and included (11 papers). A breakdown of numbers of patients is provided per treatment for nerve deactivation surgery in the sections below.

Primary Nerve Deactivation Surgery Failure

Trigger point deactivation surgery for migraine headaches is considered successful when any parameter included within the migraine headache index is decreased by at least 50% after surgery, encompassing headache frequency, severity, and duration.[18] Another variable that has been used to track the headache burden is the frequency of the monthly migraine days.[19] A positive response to botulinum toxin injections or nerve blocks has been shown to be a positive predictor of successful surgery,[20–22] with the positive-predictive value of diagnostic peripheral nerve blocks being 0.89 (95% CI, 1–0.74) and the positive-predictive value of diagnostic botulinum toxin type A injections being 89.5%.[20,22] Nevertheless, a negative response to injections does not definitively preclude surgical treatment or predict failure of trigger point deactivation surgery.[20,21]

Trigger point deactivation can be performed at various established sites, including site I (frontal), site II (zygomaticotemporal), site III (rhinogenic), site IV (greater occipital), site V (auriculotemporal), site VI (lesser occipital), and site VII (nummular).[6,7] For the greater occipital nerve (GON), complete decompression of six compression points from proximal to distal is recommended and includes addressing the interaction with the occipital artery if present.[23–28] The most common reason for failure after trigger point deactivation surgery is incomplete primary trigger point deactivation.[29] Secondary trigger points may not appear until the primary trigger is eliminated,[24] which occurs in 17.8% of patients.[29] The etiology for this has been hypothesized to be the existence of a clinically dominant trigger point, which conceals the existence of lesser, yet impactful, trigger points. In these instances, only after the dominant point has been deactivated do these other secondary points become clinically evident to both the patient and surgeon.[30,31] Furthermore, anatomic variation of trigger points may exist as well as anatomical overlap with other nerves, clouding the ability to accurately locate the correct trigger point.[28,29,32–37] This variability makes it of paramount importance to correctly identify the primary trigger points and deactivate all active anatomic components at the time of the index surgery.[25,33] Finally, chronic migraine headache patients are found to have disrupted myelin sheaths compared with controls without such headaches, which may impact outcomes.[38] It is also postulated that diffuse dysfunction in pain pathways can occur (eg, hyperexcitable neurons, dysfunction in antinociceptive periaqueductal gray matter, and cortical spreading depression), implying that these patients are more susceptible to new trigger point activation.[39] Although it is often difficult to confirm whether the nerve compression is recurrent or persistent after initial surgery, the correct diagnosis is of the utmost importance (Table 1).

New pain can also be caused by neuroma formation or an iatrogenic nerve injury.[24] Transient sensation changes, lasting up to one year, are expected after surgery at all points and should be discussed with the patient before surgery to set expectations. Guiding the patient's expectations is key to patient satisfaction and plays an important role in surgical success. Furthermore, establishing a close collaboration between surgeons and headache physicians is imperative to provide the optimal care for patients with chronic headaches.[40]

Takeaways

Question: This study aimed to identify the factors contributing to index trigger point deactivation surgery failure, how to reevaluate these cases, and how to proceed with secondary surgery.

Findings: This scoping review suggests that the most common reason for failure after index trigger point deactivation surgery is incomplete primary trigger point deactivation. Reevaluation of previously diagnosed trigger points as well as uncovered trigger points and additional preoperative testing are indicated to help determine candidacy for further surgical deactivation.

Meaning: This study delves into surgical challenges in chronic migraine treatment, emphasizing the importance of addressing secondary trigger points and fostering collaboration among medical experts for effective patient care.

Table 1. Causes of Primary Nerve Deactivation Surgery Failure

Category of Pain	Symptoms	Causes
Temporary	Transient numbness	• Release of nerve branches during dissection. Expected after surgery and may last up to 1 year
Permanent	Numbness	• Nerve avulsion or transection (eg, in cases of excision of greater occipital nerve)
Persistent	Headaches are the same etiology	• Incorrect primary diagnosis • Incomplete release • Missed trigger point
Recurrent	Headaches are the same etiology or include new trigger points	• Postoperative scarring • Emergence of secondary trigger points after primary nerve deactivation surgery • History of cervicogenic headaches in case of occipital pain. Greater occipital nerve excision is suggested in these cases when primary decompression fails
New	New pain or numbness	• Neuroma formation • Secondary trigger points that become evident only after the dominant point has been deactivated • Iatrogenic nerve injury

Categories, symptoms, and causes are described to provide the correct diagnosis if chronic headache reoccurs after primary nerve deactivation surgery. Once the correct diagnosis is confirmed, further medical or surgical treatment can be provided, if indicated.

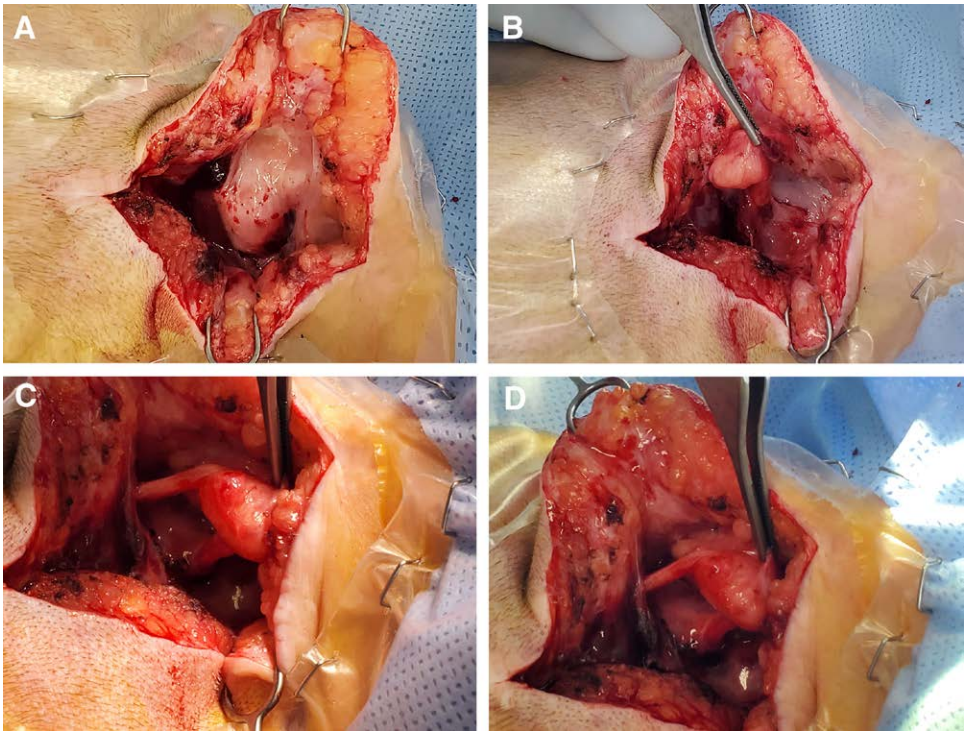

Fig. 1. Neuroma of the GON was identified after prior GON transection (A). Extensive scar tissue was encountered (B). Careful neurolysis was performed to isolate the neuroma prior to resection (C and D).

Neuroma Prevention and Treatment

Neuromas can occur after nerve transection and can cause severe neuropathic pain (Fig. 1). In major limb amputation patients, the nerve has traditionally been buried in soft tissue and bone after transection to avoid neuroma formation ("neurotization"). Newer techniques have been established in these patients addressing or preventing neuroma formation using targeted muscle reinnervation (TMR) and regenerative peripheral nerve interface (RPNI).[42–44] In TMR, the proximal nerve stump is connected to a muscle motor nerve branch to allow for directed axonal growth and gives the nerve a purpose.[42] RPNI is a surgical technique in which the proximal nerve stump is inserted into a denervated muscle wrap allowing for targeted axonal growth.[44,45] In nerve deactivation surgery for chronic headaches, muscle burial has remained the most common method to address the proximal nerve stump after nerve transection or neuroma excision thus far.[41] RPNI, TMR, a combination of RPNI and TMR, or relocation nerve grafting are increasingly explored in trigger point deactivation surgery-related neuroma management (Fig. 2).[41] In relocation nerve grafting, a long nerve autograft or allograft is used to connect the proximal nerve stump to a muscle remote from the zone of injury after nerve transection or reset neurectomy in case of diffuse nerve injury.[41] Due to the length of the nerve graft,

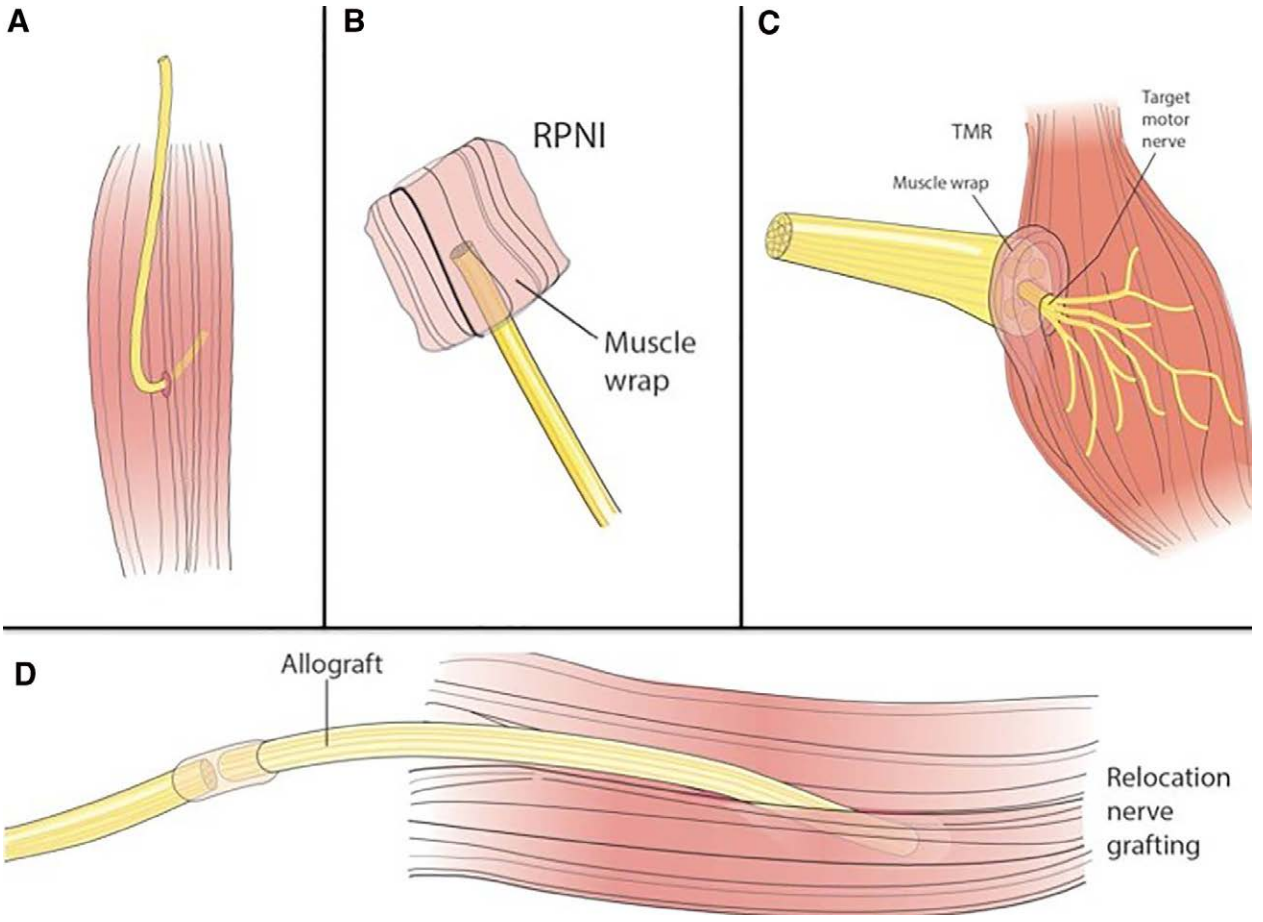

Fig. 2. Techniques for proximal nerve stump handling after nerve transection during nerve deactivation surgery. Several techniques have been illustrated including muscle burial (A), RPNI (B), TMR (C) techniques and relocation nerve grafting using an autograft or allograft (D). Reprinted with permission from *Plast Reconstr Surg Glob Open.* 2022;10:e4201.[41] Copyrighted, used, and reprinted with permission of Gferer and colleagues; all rights reserved.

few axons regenerate along the graft reaching the muscle.[46] It is of paramount importance to discuss known side effects and risks associated with proximal nerve division, including permanent numbness, transient tingling, paresthesia, potential chronic pain, and formation of painful neuromas. Gfrerer and colleagues found that RPNI and TMR are feasible options for GON/lesser occipital nerve transection patients.[41] Moreover, relocation nerve grafting with GON autograft relocation has been found beneficial in a patient with diffuse nerve injury requiring proximal nerve division.[41] Although these techniques are commonly used in limb amputation patients, it is only recently described in three cases for chronic headache patients and needs to be further explored to evaluate the efficacy.[41]

In certain regions, such as the supraorbital zone, little muscle mass might be available for muscle burial of the proximal nerve stump after resection. In these cases, neural tubes may be used for end-to-end coaptation of the proximal nerve stumps to prevent neuroma formation or postoperative scarring.[47] Ducic and colleagues reported the use of nerve tubes for end-to-end coaptation of the proximal stump of the supraorbital and supratrochlear nerves on the affected side for refractory posttraumatic or postoperative supraorbital neuralgia. Eighty percent (five patients) demonstrated an improvement of at least 50% of preoperative pain scores with a mean follow-up of 14 months.[47] In a more recent study, de Ru and colleagues also describe treatment of traumatic neuromas with consecutive transection of the causative nerve and capping of the proximal nerve stump (Neurocap, Polyganics BV, the Netherlands). Preoperative pain scores were improved in all three patients, with a follow-up of 7–24 months.[48] Basic science studies have also supported these findings and found that the use of a bioabsorbable nerve conduit in a rat sciatic nerve amputation model resulted in relief from neuroma-induced neuropathic pain and prevented perineural scar formation and neuroinflammation surrounding the proximal nerve stump. The ideal nerve conduit length was determined to be four times the diameter of the original nerve.[49]

Options after Failed Index Trigger Point Deactivation Surgery

Reevaluation of Trigger Points

The evaluation of patients after failed surgery begins with a thorough history of previous treatments, evaluation of the change in character and intensity, and reevaluation

of their trigger points. Established algorithms include identification using the acronym "PAINS": pain point (identifiable with one finger), appropriate symptoms (constellation), injectables improve pain (nerve block/botulinum toxin type A), neurologist confirmed diagnosis, and sketch matching.[7] The proper trigger point is identified by classic pain patterns, most effectively by using the patient's single index finger.[50] Each trigger point is associated with specific triggers that can be tested to confirm the diagnosis.[50–53] Additional testing for preoperative planning may include Doppler ultrasound to confirm vessel involvement.[8,54] Computed tomography scans of the face or paranasal sinuses may also be indicated in cases of primary or missed rhinogenic and frontal trigger points to evaluate the presence of turbinate hypertrophy and other intranasal pathologies, including deviated septum, the Haller cell, contact points, concha bullosa, and septal spurs.[55] An overview of the commonly used approaches for deactivation per trigger point is provided in Table 2, and these approaches are of importance for reevaluation when the primary surgery has failed.[7,9,29,56,57] When new trigger points are identified, deactivation is indicated.[30,31]

Postoperative Adhesions

Postoperative adhesions have been identified as one of the causes of persistent or recurrent headaches after complete primary nerve decompression due to excessive scar tissue around the nerve. Revision surgery in scarred tissue is known to be more challenging, requiring additional techniques to prevent recompression of the nerves.[56,58] To prevent or address this, the use of expanded polytetrafluoroethylene (ePTFE, Gore-Tex, W.L. Gore & Associates, Flagstaff, Ariz.) sleeves has been described. Vofo and colleagues described the use of 4-cm longitudinally cut sleeves to shield the released nerve along its length with a 100% success rate in five patients (follow-up between four and 18 months).[58] Another option to prevent rescarring is wrapping acellular dermal matrix (ADM, AlloDerm, Allergan, Inc.) around the GON and lesser occipital nerve at the time of primary surgery by placing the dermal side

Table 2. Common Chronic Headache Trigger Points

	Trigger Point	Compressed Nerve	Common Compression Points	Decompression Approach
I	Frontal and temporal (zygomaticotemporal) trigger point	Supraorbital and supratrochlear nerves	Frontal site: • Bony foramen orbitale • Tight fascia crossing a notch, by fibers of the corrugator supercilii muscles, depressor supercilii muscles, procerus muscles, or adjacent vessels Temporal site: • Entrance nerve in deep temporal fascia	• Open approach through the transpalpebral incision/upper eyelid blepharoplasty incision • Endoscopic approach
II	Isolated temporal trigger point	Zygomaticotemporal branch of trigeminal nerve	• Exit point nerve through zygomatic bone Compression by temporalis muscle, deep temporal fascia and superficial temporal artery	• Open approach through a temporal hairline incision • Endoscopic approach
III	Rhinogenic trigger point	Trigeminal nerves	• Compression is commonly caused by turbinate hypertrophy and other intranasal pathology, including deviated septum, Haller's cell, concha bullosa and septal spurs. • Location of compression point is dependent on cause	• Septoplasty through a Killian or hemitransfixion incision • Turbinate reduction; outfractured or reduced
IV	Occipital trigger point	Greater occipital nerve/third occipital nerve	• Musculofascial tissue surrounding the obliquus capitis inferior muscle • Epimysium underlying the semispinalis, or the muscle itself • Exit point from the semispinalis • The entrance to the trapezial tunnel • Insertion into the nuchal line • Interaction with the occipital artery if present	• Open approach through vertical occipital midline • Open approach through transverse occipital midline
V	Temporal trigger point	Auriculotemporal nerve	• The location of compression is variable • The site of maximum pain preoperatively is marked, and compression points are identified using a Doppler probe along the course of the nerve	• Open approach at point of maximal tenderness with positive Doppler
VI	Occipital trigger point	Lesser occipital nerve	• The location of compression is variable • Given anatomic variability, the site of maximum pain preoperatively is marked	• Open approach: vertical or transverse incision over site of maximum pain preoperatively given large anatomic variability
VII	Nummular trigger point		• Nummular triggers at sites that are not associated with classic trigger patterns are often associated with vessels • The site of maximum pain preoperatively is marked, and compression points are identified using a Doppler probe	• Open approach at point of maximal tenderness with positive Doppler

Compressed nerves, common compression points and nerve deactivation approaches are described per trigger point.

of the ADM against the nerve epineurium.[59] Alizadeh and colleagues evaluated 153 patients who underwent trigger point deactivation using this technique and demonstrated significant headache improvement (129 patients, 84.3%, up to 26 months follow-up).[59] Other options include intraoperative corticosteroid injections at point IV (ie, GON).[56] In a retrospective chart review of 476 patients who underwent point IV deactivation only, triamcinolone acetonide injection (282 patients) at the time of primary surgery was found to significantly decrease migraine headache index and migraine frequency compared with surgery alone.[56] It is believed that steroid injections aid in decreasing the local scarring around the nerve postoperatively.[56]

Patients with refractory chronic headache may also benefit from autologous fat grafting.[9,60] Adipose-derived mesenchymal stem cells, present in autologous fat, have been postulated to improve nerve regeneration by repairing myelin in patients with demyelination disorders.[38,61–63] A recent study by Guyuron and colleagues evaluated autologous fat injection after primary nerve deactivation failure.[9] Sixty-nine percent (20 patients) experienced successful improvement, and 41% (12 patients) experienced complete resolution (mean follow-up time of 29.4 months).[9] Another study found that autologous fat grafting significantly decreased neuropathic pain in up to 28 months of follow-up (14 patients), with improved quality of sleep in 50% of patients (seven patients).[64] Injection of autologous fat, harvested from the abdomen or lateral thigh, may be used as an adjunctive therapy with relatively minimal side effects or complications.[9,65] Fat injection is indicated in case of diffuse residual pain after initial surgery. If the symptoms are not completely eliminated after two rounds of fat injection, or if there is no symptom relief after the first round of fat injection, other procedures are indicated.[9]

Neurectomy

In patients with refractory pain after primary nerve deactivation, neurectomy may be an option in select patients. Neurectomy is occasionally performed as a last resort in revision nerve deactivation surgery, for patients in whom anesthesia and/or paresthesia is preferred over the patient's current symptom complex.[18,51,53,66] This procedure should only be performed in extenuating circumstances (eg, permanent nerve injury, loss of fascicular patterns, discoloration of the nerve, extensive scarring, or absence of the vasa vasorum).[41,67] When performed, 70.4% of patients (50 patients) achieved a 50% or greater reduction, and 41% of patients (29 patients) achieved a 90% or greater reduction in headache severity, respectively.[66] Diagnosis of cervicogenic headache was associated with failure of surgery in 15% of these cases.

Limitations

The use of TMR and RPNI for trigger point deactivation surgery-related neuroma management is relatively novel and includes studies with few patients only. Moreover, ePTFE sleeves to prevent adhesions have also been described in small numbers of patients. Further exploration in larger groups of patients with a longer follow-up is needed to evaluate the efficacy with higher levels of evidence. Another limitation is the fact that many of the studies addressed multiple trigger points, which limits our ability to assess the effectiveness of a certain treatment per trigger point.

CONCLUSIONS

The success rate of primary trigger point deactivation surgery for chronic headaches ranges between 79% and 90%, with proven overall safety and minimal complications. Although approximately only 12% of patients experience failure, the management of unsuccessful primary surgery is critical. Secondary trigger point deactivation surgery is often necessitated due to persistent or recurrent symptoms after initial surgery. Reevaluation of trigger points (at the initial site and/or other sites, if uncovered), their associated anatomical variations, and additional diagnostic testing may be indicated. To address or prevent postoperative scarring, corticosteroid injection, ADM, adipofascial fat, or ePTFE sleeves are proposed with demonstrated beneficial effects. For neuroma management, RPNI, TMR, a combination of both, or relocation nerve grafting are increasingly explored. Nerve tubes could be used for capping of the end-to-end coaptation of the proximal stump to prevent neuroma and perineural scar formation and improve preoperative pain when muscle burial is not possible. Neurectomy can be performed when there is significant intrinsic damage to the nerve or when patients would prefer numbness over the persistent pain symptoms. Finally, establishing a close collaboration between surgeons and headache physicians is imperative to provide the optimal care for these headache patients.

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DISCLOSURES

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