



Surgical Interventions for the Management and Treatment of Chronic Migraine in Clinical Nursing Practice

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Abstract

Background: Chronic migraine is a disabling neurological disorder characterized by frequent headache episodes and significant functional impairment. While pharmacological and behavioral therapies remain first-line treatments, a subset of patients continues to experience refractory symptoms that negatively impact quality of life.

Aim: This article aims to review and synthesize current evidence on surgical and procedural interventions for the management of chronic, drug-refractory migraine, with particular emphasis on their clinical relevance within nursing and interprofessional practice.

Methods: A narrative, evidence-based review was conducted using published clinical studies, anatomical and physiological research, and established diagnostic and therapeutic guidelines. The article examines migraine pathophysiology, indications and contraindications for surgery, and the effectiveness of interventions such as botulinum toxin injections, peripheral nerve decompression or neurectomy, rhinogenic surgery, and neuromodulation techniques.

Results: Surgical interventions targeting peripheral nerve compression sites demonstrate meaningful reductions in migraine frequency and intensity in carefully selected patients. Botulinum toxin serves both therapeutic and diagnostic roles by identifying trigger points and predicting surgical success. Peripheral neurolysis and neurectomy show sustained benefit in refractory cases, while neuromodulation—particularly occipital nerve stimulation—offers an alternative for patients unsuitable for decompression surgery. Complication rates are generally low when procedures are appropriately indicated and performed within multidisciplinary care models.

Conclusion: For patients with chronic migraine unresponsive to optimal medical therapy, targeted surgical and neuromodulatory interventions provide effective, individualized treatment options. Comprehensive evaluation, precise anatomical localization, and interprofessional collaboration are critical to achieving optimal outcomes.

Key Words: Chronic migraine, migraine surgery, peripheral neurolysis, botulinum toxin, neuromodulation, nursing practice

Introduction

Migraine is a complex primary neurological disorder marked by recurrent episodes of headache that vary in intensity, duration, and associated symptomatology. Patients frequently experience nausea, vomiting, sensitivity to light (photophobia) and sound (phonophobia), and occasionally vertigo, with some individuals presenting with prodromal or

postdromal aura symptoms such as visual disturbances, sensory changes, or speech difficulties [1]. The disorder is highly prevalent, affecting approximately 11.7% of the population in the United States, with a marked sex disparity: 17.1% of women and 5.6% of men are impacted [2]. Migraine can be classified as episodic, acute, or chronic, with chronic migraine representing the most debilitating form.

According to the International Headache Society's third edition of the classification of headache disorders, chronic migraine is defined by the occurrence of headache on fifteen or more days per month over a period exceeding three months, with at least eight days per month meeting migraine criteria [3]. Management of migraine traditionally begins with pharmacologic interventions. First-line medications include β -blockers, anticonvulsants, calcium channel blockers, tricyclic antidepressants, and nonsteroidal anti-inflammatory drugs (NSAIDs), which aim to reduce the frequency, duration, and severity of attacks. Additional therapies may incorporate abortive treatments, including triptans and ergot derivatives. Despite these interventions, a subset of patients remains refractory, experiencing persistent, frequent, or severe attacks that significantly impair quality of life and functional capacity. For these individuals, surgical interventions targeting specific peripheral nerve compression sites, trigger points, or vascular decompression may be considered. Such approaches are intended to alleviate nerve irritation, reduce inflammatory mediators, and prevent recurrent migraine episodes. Migraine surgery is therefore reserved for patients who demonstrate failure of optimized medical therapy, providing an adjunctive or definitive option for chronic migraine management when conventional pharmacologic measures are insufficient [3].

Anatomy and Physiology

Early conceptualizations of migraine pathogenesis emphasized cerebral vasodilation as the primary mechanism. This theory has been increasingly challenged by evidence demonstrating that agents known to increase cerebral blood flow, such as vasoactive intestinal peptide, do not consistently induce migraine attacks [4]. Likewise, pharmacologic agents that can precipitate migraine, including sildenafil, fail to produce enduring vascular alterations, further undermining the vascular hypothesis [5]. Contemporary research highlights the role of neuronal hyperexcitability within cortical and brainstem structures, which appears to contribute significantly to migraine susceptibility [6][7]. Cortical spreading depression, a propagating wave of neuronal and glial depolarization, is considered the underlying mechanism for the visual and sensory phenomena characteristic of migraine aura [8]. The trigeminal system plays a central role in the generation and maintenance of migraine pain. Afferent fibers from the trigeminal nerve project to the meninges, where they release proinflammatory neuropeptides, including substance P, calcitonin gene-related peptide, and neurokinin A. This release initiates and perpetuates a cycle of neurogenic inflammation that sensitizes pain pathways [9]. Clinical evidence increasingly supports a peripheral component in migraine pathophysiology. Interventions targeting peripheral nerves, such as

botulinum toxin injections and surgical neurolysis, have demonstrated efficacy in alleviating chronic migraine, suggesting that modulation of sensory input can significantly reduce symptom burden. Surgical strategies typically focus on decompression or neurolysis of sensory branches of the trigeminal and occipital nerves that innervate the face and occiput [10]. Key nerves implicated include the supraorbital, supratrochlear, zygomaticotemporal, auriculotemporal, and greater and lesser occipital nerves. In certain cases, migraine pain may be exacerbated or initiated by structural abnormalities in the nasal cavity, such as hypertrophic turbinates or a deviated septum, which mechanically stimulate branches of the trigeminal nerve. These findings collectively emphasize the complex interplay between central neuronal excitability and peripheral nerve contributions in the pathophysiology of migraine, underscoring the rationale for both pharmacologic and targeted surgical interventions.

Indications

Migraine surgery is primarily indicated in patients whose condition demonstrates resistance to conventional therapeutic strategies, including both pharmacologic and behavioral interventions. These patients typically exhibit persistent headache episodes despite adherence to guideline-directed medical therapy, which may include triptans, nonsteroidal anti-inflammatory drugs, preventive medications, and structured behavioral approaches such as cognitive-behavioral therapy and lifestyle modifications. The surgical approach is considered only after comprehensive evaluation confirms that conservative measures have failed to achieve adequate symptom control and quality-of-life improvement. Chronic migraine that remains disabling despite optimized therapy represents the principal indication for surgical intervention. Candidates often experience frequent, severe headache episodes that significantly impair occupational performance, social engagement, and daily functioning. Migraine surgery aims to address peripheral triggers or nerve compression that contributes to the perpetuation of pain. Identifying specific trigger sites through detailed clinical assessment, including physical examination and diagnostic nerve blocks, is essential to determine the appropriate surgical targets. Evidence supports that patients with well-defined trigger points experience meaningful symptomatic relief and reduction in headache frequency following surgical decompression or neurolysis. The decision to proceed with surgery requires multidisciplinary input, including confirmation of refractory status, documentation of headache characteristics, and careful consideration of potential risks and benefits. Surgical intervention is thus reserved for a subset of migraine patients for whom all noninvasive treatments have proven insufficient and for whom

targeted peripheral nerve decompression offers a plausible path to long-term relief and functional restoration.

Contraindications

Migraine surgery carries relative contraindications, among which the presence of a psychiatric disorder is particularly notable. Patients with active or poorly controlled psychiatric conditions, including major depressive disorder, anxiety disorders, somatoform disorders, or personality disorders, may exhibit altered pain perception, poor adherence to post-surgical care, or unrealistic expectations regarding outcomes. The presence of psychiatric comorbidities can confound the assessment of surgical efficacy and may exacerbate post-operative morbidity, reducing the likelihood of clinically meaningful improvement. While psychiatric conditions do not constitute absolute barriers to surgery, they necessitate careful evaluation and, when feasible, stabilization before surgical consideration. Preoperative psychiatric assessment facilitates identification of patients whose symptoms may respond better to psychological or medical interventions rather than invasive procedures. Additionally, ongoing psychiatric disorders may influence post-operative pain management, recovery trajectories, and overall patient satisfaction. The decision to defer surgery in such cases is informed by a risk-benefit analysis that weighs the potential for symptomatic relief against the possibility of poor outcomes due to underlying psychological vulnerability. Multidisciplinary collaboration involving neurology, psychiatry, and surgical teams ensures that patients are appropriately screened and optimized, minimizing the risk of post-operative complications and maximizing the likelihood of a favorable functional outcome.

Personnel

Patients being considered for migraine surgery should undergo comprehensive evaluation by a neurologist prior to any surgical intervention. The neurologist's role encompasses confirming the diagnosis of migraine, documenting headache characteristics, and assessing the degree of refractoriness to medical and behavioral treatments. Neurologists perform detailed histories and physical examinations to localize potential trigger sites and evaluate for comorbid conditions that may influence treatment outcomes. Coordination between neurology and surgical teams ensures that patients are appropriately selected and that indications for surgery are well justified. Preoperative neurological assessment may include diagnostic nerve blocks, imaging studies, and standardized migraine severity scales to quantify baseline functional impairment. Additionally, neurologists counsel patients regarding realistic expectations, procedural risks, and post-operative recovery, reinforcing the importance of

adherence to multidisciplinary management plans. Involving specialized nursing personnel, pain management specialists, and, when indicated, psychologists further enhances preoperative preparation, allowing for holistic evaluation and optimization. This structured, team-based approach reduces the risk of surgical failure, minimizes complications, and facilitates identification of patients most likely to derive sustained benefit from targeted peripheral nerve interventions.

Preparation

Accurate assessment of migraine severity is essential prior to surgical intervention, commonly achieved using validated instruments such as the Migraine Disability Assessment questionnaire (MIDAS) [11]. MIDAS scoring quantifies functional impairment across daily, occupational, and social domains. Scores of 0 to 5 correspond to grade I (minimal or no disability), 6 to 10 indicate grade II (mild disability), 11 to 20 represent grade III (moderate disability), and scores exceeding 21 correspond to grade IV (severe disability). Initial management of migraine encompasses both pharmacologic therapies, including abortive and preventive agents, and behavioral strategies such as cognitive-behavioral therapy, stress management, and lifestyle modifications [12][13]. Despite these measures, certain patients experience persistent, refractory headaches necessitating consideration of surgical options. The International Classification of Headache Disorders, third edition (beta version), defines refractory migraine as at least eight migraine attacks per month accompanied by fifteen or more headache days monthly, providing a standardized framework for identifying candidates for surgical intervention. For patients in whom rhinogenic headache is suspected, preoperative imaging with computed tomography of the nasal cavity and paranasal sinuses is recommended to identify structural abnormalities, including hypertrophic turbinates or septal deviations, that may contribute to trigeminal nerve irritation. Comprehensive preparation, including detailed clinical evaluation, severity scoring, imaging studies, and confirmation of refractoriness, ensures appropriate patient selection and optimizes surgical outcomes by targeting the peripheral mechanisms underlying chronic migraine.

Technique or Treatment

Botulinum toxin, a neurotoxic protein produced by the bacterium *Clostridium botulinum*, functions by inhibiting the release of acetylcholine at presynaptic neuromuscular junctions, resulting in localized muscle paralysis. Eight distinct subtypes of botulinum toxin have been identified, designated A through H, with type A being the most widely utilized and recognized for its safety and efficacy in therapeutic applications [14]. The historical origins of botulinum toxin as a medical treatment trace back to Justinus Kerner, who first investigated its physiological effects under the term "sausage

poison," conducting experiments on animals and on himself to explore its clinical potential [15]. Alan Scott subsequently pioneered the application of botulinum toxin in ophthalmology for the treatment of strabismus, marking a significant milestone in its transition from experimental toxin to controlled therapeutic agent [14]. Following this development, the clinical scope of botulinum toxin expanded to address various focal dystonias, including blepharospasm [16][17], cervical dystonia [18][19], oromandibular dystonia [20], laryngeal dystonia [21], hemifacial spasm [20][22], and focal hand dystonia [23]. Its utility further extended to the management of spasticity, focal hyperhidrosis [24], and the identification and treatment of migraine trigger points [25].

The therapeutic efficacy of botulinum toxin in migraine management is predicated on its ability to reduce peripheral nerve irritation by paralyzing muscles implicated in headache generation and by diminishing the release of proinflammatory neuropeptides [26]. Multiple double-blind, controlled trials have consistently demonstrated reductions of approximately 50% in both migraine frequency and intensity within three months of administration, with therapeutic effects lasting between three and four months [25][27][28]. Despite these encouraging results, certain studies have reported only marginal superiority of botulinum toxin compared to placebo, reflecting variability in clinical response among patient populations [29][30]. While botulinum toxin can serve as both an acute intervention and a prophylactic treatment, its long-term use is constrained by several factors, including the potential development of neutralizing antibodies leading to resistance, diminishing efficacy with repeated administration, and adverse outcomes such as excessive muscle paralysis, localized atrophy, and injection-site irritation, particularly when higher doses are employed [25][31]. Consequently, some experts advocate for its use primarily as a diagnostic or screening modality to identify suitable candidates for surgical neurolysis rather than as a definitive long-term therapy [32]. Administration of botulinum toxin requires careful localization to the pericranial musculature based on the distribution of headache pain. Standard dosing protocols involve diluting the total dose of 25 units in normal saline and distributing it across targeted muscles to optimize efficacy while minimizing adverse effects. For frontal headache pain, injections typically include 3 units per temporalis muscle (totaling 6 units), 2.5 units at four frontalis muscle sites (5 units unilaterally or 10 units bilaterally), 3 units into each corrugator muscle (totaling 6 units), and 3 units into the procerus muscle along the midline [25]. In patients with occipital headache predominance, doses ranging from 12.5 to 50 units may be administered bilaterally near the greater and lesser occipital nerves. Clinical

response is evaluated by the duration of pain relief; a sustained improvement lasting six to twelve weeks suggests that the patient may be an appropriate candidate for subsequent surgical interventions targeting the peripheral nerves implicated in headache generation.

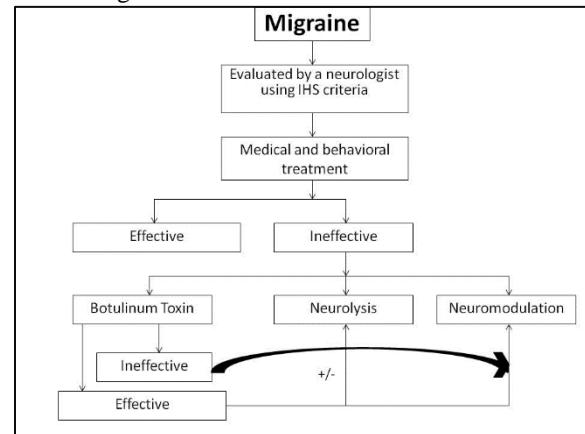


Fig. 1: Migraine Treatment algorithm.

Botulinum toxin thus serves a dual function in migraine management, providing symptomatic relief while facilitating the identification of precise surgical targets. Its role exemplifies the integration of pharmacologic and procedural strategies in contemporary headache therapy. Careful patient selection, precise dosing, and targeted injection techniques are critical to maximizing clinical benefit while minimizing the risks associated with neuromuscular modulation. Ongoing research continues to refine injection protocols, identify predictive markers of response, and evaluate long-term safety, ensuring that botulinum toxin remains a cornerstone in the multimodal management of chronic migraine and refractory headache disorders.

Peripheral Neurolysis/Neurectomy

The therapeutic application of peripheral neurolysis and neurectomy in migraine management emerged from an incidental clinical observation in which patients reported substantial relief from debilitating frontal headaches following cosmetic brow lift procedures. Guyuron and colleagues were the first to recognize that surgical manipulation in the region of the supraorbital and supratrochlear nerves could mitigate migraine symptoms [33]. Building upon these observations, a prospective study by the same group confirmed that targeted surgical interventions at peripheral nerve trigger sites could yield significant and sustained reductions in headache frequency and intensity [34]. The identification of appropriate candidates for surgery relies on rigorous preoperative evaluation, including confirmation of migraine trigger sites and demonstration of at least a 50% reduction in headache severity following localized botulinum toxin injections for a minimum of six weeks. In this context, botulinum toxin serves both as a diagnostic tool and as a temporary therapeutic measure, injected into the bilateral

frontal, temporal, and occipital regions, or specifically into the corrugator supercilii muscles at standardized doses of approximately 12.5 units per site [34][35]. Additionally, structural evaluation of the nasal cavity is indicated to detect hypertrophied turbinates or septal deviations that may impinge upon branches of the trigeminal nerve, particularly in cases of rhinogenic headache. Peripheral neurectomy has been demonstrated to provide sustained benefit in patients whose migraines are refractory to pharmacologic interventions [36].

Frontal migraine headaches are typically addressed through a trans-eyelid approach. After an upper eyelid incision, the orbital septum is retracted caudally, and the orbicularis oculi muscle is elevated cranially. The corrugator supercilii and depressor supercilii muscles are excised to relieve compressive forces on the supraorbital and supratrochlear nerves. Concurrently, associated blood vessels are ligated, and the supraorbital foramen is unroofed to provide complete nerve decompression. The resultant space is then filled with medial orbital fat to cushion the nerves and prevent postoperative adhesions, secured using 6-0 polyglactin 910 sutures. The skin incision is closed meticulously with 6-0 nylon sutures, and the procedure can be performed bilaterally to address symmetrical headache patterns [34]. Anterior temporal headaches are commonly mediated by the zygomaticotemporal nerve. Surgical management involves a 2 cm incision at a defined anatomical location approximately 16 mm lateral and 6 mm cranial to the lateral canthus. Dissection proceeds between the layers of the temporalis fascia to isolate and sever the nerve. The distal nerve segment is buried within the temporalis muscle to minimize neuroma formation. For patients with combined frontotemporal headache, dual incisions are utilized—one in the upper tarsal crease and another along the superior temporal line—permitting simultaneous decompression of frontal and temporal nerve branches. These procedures may also be executed endoscopically, allowing for minimal soft tissue disruption and enhanced postoperative recovery [34][35]. Posterior temporal headaches are frequently attributable to the auriculotemporal nerve. A targeted 2 cm incision at the base of the zygomatic arch allows for identification and isolation of the nerve along with associated superficial temporal vessels, which are ligated to prevent hemorrhage. The nerve is dissected over a 1–2 cm segment, severed, and embedded within the temporalis muscle to reduce the risk of neuroma formation, with layered closure of the incision ensuring structural integrity and optimal healing.

Occipital migraines are predominantly mediated by the greater and lesser occipital nerves. For diffuse occipital pain, a midline incision extending from the external occipital protuberance to the C2 spinous process facilitates exposure of the greater occipital nerve. The overlying trapezius fascia

is incised, and the muscle fibers are split longitudinally to access and free the nerve from the surrounding semispinalis capitis. Complete neurolysis involves transection of encasing muscle and fascia, with interposition of pedicled fat between the nerve and muscular structures to prevent postoperative entrapment. The incision is closed using 3-0 nylon sutures, and the procedure may be performed contralaterally as required [37]. Lateral occipital headaches implicate the lesser occipital nerve. Surgical access is achieved via a 2 cm incision approximately 2 cm medial to the mastoid process in the hairline. The thick temporal fascia is incised, the nerve and accompanying vessels are dissected, the artery is ligated, and the nerve is severed and embedded within the muscle. Closure is performed in layers with 3-0 nylon sutures. While occipital neurolysis often produces immediate symptomatic relief, recurrence rates remain high, prompting consideration of alternative interventions, including neuromodulatory techniques [37]. Rhinogenic headaches, resulting from mechanical irritation of trigeminal nerve branches by hypertrophied turbinates or a deviated septum, require a distinct surgical approach. Traditional migraine interventions, including botulinum toxin injections, are largely ineffective in this context because the pain is mediated through the maxillary nerve via the sphenopalatine ganglion. Surgical management involves turbinectomy or septoplasty to alleviate nerve compression, thereby addressing the underlying etiology of headache. Preoperative imaging and careful anatomical mapping are critical to ensuring complete resolution of compressive triggers and reducing the risk of postoperative recurrence or complications.

Peripheral neurolysis and neurectomy thus constitute a precise, anatomy-based intervention targeting specific migraine trigger sites. The surgical approach is highly individualized, relying on preoperative diagnostic modalities, including botulinum toxin response and structural imaging, to optimize outcomes. Each anatomical region—frontal, anterior temporal, posterior temporal, occipital, and rhinogenic—requires tailored operative techniques to isolate and decompress the implicated nerves while preserving adjacent vascular and muscular structures. Clinical outcomes have consistently demonstrated reductions in headache frequency and severity, particularly in patients with refractory migraine who have exhausted conservative pharmacologic and behavioral therapies. Nevertheless, considerations of potential recurrence, neuroma formation, and the necessity for adjunctive interventions such as neuromodulation highlight the importance of a multidisciplinary approach in patient selection, operative planning, and postoperative care. The evolution of these techniques underscores the integration of surgical precision with an understanding of migraine pathophysiology,

emphasizing the role of peripheral nerve modulation in the comprehensive management of chronic and drug-refractory headache disorders.

Neuromodulation for Migraine

Neuromodulation represents an advanced therapeutic option for patients with chronic migraine who fail to achieve adequate relief from conventional pharmacologic treatments or for whom peripheral neurolysis is either not feasible or has proven ineffective. This approach involves the targeted electrical stimulation of specific cranial or cervical nerves implicated in migraine pathophysiology, aiming to modulate nociceptive transmission and alter pain perception. The most commonly employed modality is occipital nerve stimulation, which entails the surgical placement of electrodes around the cervical dorsal nerves within the suboccipital region. Initially, these electrodes are connected to a temporary trial stimulator to evaluate the patient's responsiveness. Patients who report greater than 50% pain relief during this trial are then candidates for permanent implantation of a pulse generator, which maintains continuous neuromodulatory input [38]. The selection of candidates relies on careful clinical assessment to confirm the distribution of headache and the specific nerve involvement, as well as exclusion of conditions that may compromise device placement or function. Occipital nerve stimulation exerts its analgesic effects primarily through modulation of the trigeminocervical complex, a key integrative center that processes nociceptive input from both the upper cervical dorsal nerves and the trigeminal nerve. Stimulation of the occipital nerves is believed to preferentially activate large-diameter afferent fibers, which inhibit transmission of nociceptive signals from small, pain-conveying fibers in accordance with the gate control theory [44]. This mechanism allows for analgesia not only in the occipital region but also in areas innervated by the trigeminal nerve, providing broader headache relief. Clinical studies report that approximately 30% to 50% of patients undergoing occipital nerve stimulation achieve at least 50% reduction in headache intensity and frequency, highlighting its potential effectiveness in refractory cases [45][46][47]. Despite its efficacy, the procedure is associated with a high rate of lead migration or the need for readjustment, occurring in nearly half of implanted cases, although the technique is minimally invasive and well tolerated [47]. In addition to migraine, occipital nerve stimulation has demonstrated therapeutic benefit in cluster headaches, further supporting its role in the management of severe, primary headache disorders [48].

Other neuromodulatory techniques have also been explored, including sphenopalatine ganglion stimulation, supraorbital nerve stimulation, and vagus nerve stimulation [38][39][40][41][42]. These

approaches are designed to target specific peripheral or central components of headache circuitry, providing localized modulation of pain pathways. Initial studies suggest potential benefit, but evidence from large, randomized controlled trials remains limited, underscoring the need for further investigation to define efficacy, optimal patient selection, stimulation parameters, and long-term safety [43]. In addition to neuromodulation, nerve decompression at identified migraine trigger sites remains a complementary surgical strategy. By removing or displacing surrounding tissues, including muscles, fascia, and adjacent blood vessels, decompression reduces mechanical irritation of the involved nerves, thereby attenuating the frequency and severity of migraine attacks [49]. This approach is particularly relevant for patients with medical refractory, chronic migraine who exhibit clear correlation between specific peripheral nerve compression and headache episodes. Collectively, neuromodulation and targeted nerve decompression expand the therapeutic repertoire for refractory migraine, offering alternatives when conventional medical management is insufficient. The success of these interventions depends on precise anatomical localization, careful patient selection, and multidisciplinary management, integrating neurology, pain medicine, and surgical expertise. While neuromodulation provides a minimally invasive, adjustable option for altering nociceptive transmission, decompression addresses the underlying structural contributors to peripheral nerve irritation. Both strategies underscore the evolving paradigm in migraine management, emphasizing individualized, mechanism-based treatment approaches for patients with chronic and debilitating headache disorders.

Complications

Botulinum toxin administration, while generally safe, is associated with several procedure-specific adverse effects. Muscle atrophy is the most frequently reported complication, particularly in the temporalis muscle, with incidence rates reaching up to 23% [50]. This localized atrophy typically resolves over time but can produce temporary cosmetic or functional alterations in facial contour. Other notable complications depend on the anatomical site of injection. For example, injections into the frontalis muscle may result in transient eyelid ptosis, whereas cervical or pericervical injections can produce temporary neck muscle weakness. These effects are usually dose-dependent and reversible but require careful dosing and precise injection technique to minimize functional impairment and aesthetic concerns. Proper patient counseling regarding the potential for transient weakness or altered muscle tone is essential before initiating therapy. Peripheral neurolysis and neurectomy carry a distinct set of surgical risks. One of the primary complications is

neuroma formation at the site of nerve transection. This risk can be mitigated by burying the severed nerve end within surrounding muscular tissue, which reduces abnormal nerve regeneration and neuropathic pain [51]. Surgical procedures also carry a low but notable risk of infection and hemorrhage, which can be minimized by strict adherence to aseptic technique and meticulous hemostasis. Temporary sensory disturbances, including paresthesia and pruritus at the operative site, are also common. These symptoms generally resolve within weeks to months, but patients should be informed of the possibility of transient discomfort or altered sensation in the distribution of the affected nerve.

Turbinate surgery, including turbinectomy or septoplasty performed for rhinogenic headaches, carries complications typical of sinonasal procedures. Postoperative epistaxis is relatively common and often self-limited, whereas sinusitis may occur due to altered mucociliary clearance. Patients may also experience nasal dryness, crusting, or, in rare cases, deviation of the nasal septum if structural support is disrupted during surgery [50]. Close postoperative follow-up is required to monitor these outcomes and ensure prompt management if complications arise. Neuromodulation procedures are associated with device-related complications. Lead migration represents the most frequent technical issue and can compromise efficacy if electrodes are not adequately anchored. Infection, though uncommon, can usually be managed with antibiotic therapy but occasionally necessitates removal of the implant to prevent systemic or localized complications. Proper surgical technique, perioperative prophylaxis, and patient education regarding device care are essential to reduce the likelihood of these adverse events [50].

Clinical Significance

Migraine represents one of the most prevalent primary headache disorders, affecting millions worldwide and causing significant functional impairment. Accurate diagnosis relies on thorough clinical evaluation by a neurologist, who assesses headache characteristics including location, frequency, intensity, and associated symptoms such as photophobia, phonophobia, nausea, or aura. Quantitative measures, such as the Migraine Disability Assessment (MIDAS) questionnaire, provide objective assessment of functional impairment and help stratify the severity of the disorder. Initial management typically involves a combination of pharmacologic therapy—using abortive and preventive agents—and behavioral interventions, including lifestyle modification, stress management, and cognitive-behavioral strategies. Botulinum toxin has emerged as both a diagnostic and therapeutic tool, particularly in identifying peripheral nerve trigger sites that contribute to migraine pathophysiology. By targeting these specific areas, clinicians can determine candidates for more definitive interventions. In patients whose

headaches remain refractory to medical management, peripheral neurolysis or neurectomy has demonstrated considerable efficacy. These surgical procedures aim to decompress or remove peripheral nerves implicated in chronic migraine, resulting in sustained reduction of headache frequency and intensity. Clinical studies indicate that careful preoperative assessment, including evaluation of botulinum toxin responsiveness, enhances patient selection and optimizes surgical outcomes. The ability to identify and treat precise trigger sites underscores the importance of individualized, mechanism-based approaches in migraine management, particularly for patients with chronic, debilitating headaches unresponsive to conventional therapies. Understanding the clinical significance of migraine and the available interventions allows healthcare providers to deliver targeted, evidence-based care that addresses both symptom burden and functional impairment [50].

Enhancing Healthcare Team Outcomes

Effective migraine management requires the coordinated efforts of an interprofessional healthcare team. Neurologists serve as the primary evaluators, providing diagnostic accuracy, identifying trigger sites, and overseeing the overall treatment plan. Psychologists and psychiatrists contribute by addressing comorbid psychiatric conditions such as anxiety, depression, or stress-related disorders that can exacerbate migraine frequency or severity. Surgeons, including those specialized in peripheral neurolysis or neuromodulation, play a critical role in performing interventions for patients with drug-refractory migraines. Specialty-trained neurology nurses and other allied health professionals are essential in providing patient education, monitoring for complications, and supporting adherence to treatment protocols. Pharmacists contribute by selecting appropriate pharmacologic therapies, monitoring adverse effects, and optimizing medication regimens to minimize drug interactions and maximize efficacy. Successful surgical outcomes depend on appropriate patient selection, which requires thorough assessment and collaboration among all team members. Preoperative education provided by nurses ensures that patients understand procedural expectations, postoperative care, and symptom monitoring. Postoperative follow-up by nurses and allied health professionals facilitates early identification of complications, supports rehabilitation, and reinforces adherence to multimodal migraine management strategies. Integration of these diverse healthcare roles enhances communication, reduces errors, and ensures that patients receive comprehensive care addressing both neurological and psychosocial aspects of migraine. By leveraging interprofessional collaboration, healthcare teams can optimize functional outcomes, reduce headache burden, and improve overall quality

of life for patients with chronic or refractory migraine [50][51].

Nursing, Allied Health, and Interprofessional Team Interventions

Nursing, allied health, and interprofessional collaboration play a pivotal role in advancing migraine care, particularly in complex or refractory cases. Neurologists and neurosurgeons must maintain continuous collaboration to refine surgical interventions, including peripheral neurolysis, neurectomy, and neuromodulation, ensuring that operative techniques are optimized for efficacy and safety. Specialty-trained nurses provide critical support by conducting preoperative assessments, educating patients about procedural risks and expectations, and assisting with postoperative monitoring to identify complications early. Allied health professionals, such as occupational and physical therapists, may contribute to lifestyle and ergonomic modifications that reduce headache triggers, while psychologists and psychiatrists address behavioral and mental health factors influencing migraine frequency and severity. Pharmacists play an integral role in medication management, ensuring appropriate dosing, monitoring adverse effects, and adjusting regimens to achieve optimal symptom control. Interprofessional interventions also include ongoing outcome evaluation, data collection, and participation in research aimed at establishing evidence-based best practices. Collaborative care ensures that treatment is individualized, comprehensive, and continuously refined based on patient response and emerging clinical evidence [52]. Through structured communication, shared decision-making, and coordinated care plans, interprofessional teams enhance patient adherence, minimize procedural risks, and improve overall clinical outcomes. The integration of nursing, allied health, and medical expertise facilitates a patient-centered approach that addresses the multifactorial nature of migraine, balancing pharmacologic, behavioral, and procedural strategies to achieve sustained relief and functional improvement. Such coordinated interventions underscore the importance of a system-level approach to migraine management, promoting both immediate symptom control and long-term quality-of-life enhancement for patients with chronic headache disorders [52].

Conclusion:

Chronic migraine remains a significant clinical challenge due to its high prevalence, complex pathophysiology, and variable response to conventional medical therapies. This review highlights that surgical and procedural interventions play an important role in the management of patients with medically refractory migraines. Techniques such as botulinum toxin therapy, peripheral nerve decompression, neurectomy, rhinogenic surgery, and

neuromodulation address the peripheral contributors to migraine pain and offer substantial symptom relief in appropriately selected individuals. Successful outcomes depend on accurate diagnosis, identification of migraine trigger sites, and thorough preoperative assessment using standardized tools such as the MIDAS questionnaire and diagnostic nerve blocks. Nursing and interprofessional team involvement is essential in patient education, perioperative preparation, postoperative monitoring, and long-term follow-up. When integrated within a multidisciplinary care framework, surgical interventions can significantly reduce migraine burden, improve functional capacity, and enhance quality of life for patients with chronic, treatment-resistant migraine.

References:

1. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalgia : an international journal of headache*. 2013 Jul;33(9):629-808. doi: 10.1177/0333102413485658.
2. Lipton RB, Bigal ME, Diamond M, Freitag F, Reed ML, Stewart WF, AMPP Advisory Group. Migraine prevalence, disease burden, and the need for preventive therapy. *Neurology*. 2007 Jan 30;68(5):343-9.
3. Martelletti P, Katsarava Z, Lampl C, Magis D, Bendtsen L, Negro A, Russell MB, Mitsikostas DD, Jensen RH. Refractory chronic migraine: a consensus statement on clinical definition from the European Headache Federation. *The journal of headache and pain*. 2014 Aug 28;15(1):47. doi: 10.1186/1129-2377-15-47.
4. Rahmann A, Wienecke T, Hansen JM, Fahrenkrug J, Olesen J, Ashina M. Vasoactive intestinal peptide causes marked cephalic vasodilation, but does not induce migraine. *Cephalgia : an international journal of headache*. 2008 Mar;28(3):226-36. doi: 10.1111/j.1468-2982.2007.01497.x.
5. Kruuse C, Thomsen LL, Birk S, Olesen J. Migraine can be induced by sildenafil without changes in middle cerebral artery diameter. *Brain : a journal of neurology*. 2003 Jan;126(Pt 1):241-7
6. Welch KM, D'Andrea G, Tepley N, Barkley G, Ramadan NM. The concept of migraine as a state of central neuronal hyperexcitability. *Neurologic clinics*. 1990 Nov;8(4):817-28
7. Welch KM, Nagesh V, Aurora SK, Gelman N. Periaqueductal gray matter dysfunction in migraine: cause or the burden of illness? *Headache*. 2001 Jul-Aug;41(7):629-37
8. Parsons AA. Cortical spreading depression: its role in migraine pathogenesis and possible

therapeutic intervention strategies. Current pain and headache reports. 2004 Oct;8(5):410-6

9. Fusco M, D'Andrea G, Miccichè F, Stecca A, Bernardini D, Cananzi AL. Neurogenic inflammation in primary headaches. *Neurological sciences : official journal of the Italian Neurological Society and of the Italian Society of Clinical Neurophysiology*. 2003 May;24 Suppl 2():S61-4
10. Faizo E, Fallata A, Mirza I, Koshak AK, Bucklain YT, Alharbi R, Tasji A, Tasji T, Kabbarah A. The Efficacy of Trigger Site Surgery in the Elimination of Chronic Migraine Headache: An Update in the Rate of Success and Failure. *Cureus*. 2024 Feb;16(2):e54504. doi: 10.7759/cureus.54504.
11. Stewart WF, Lipton RB, Kolodner KB, Sawyer J, Lee C, Liberman JN. Validity of the Migraine Disability Assessment (MIDAS) score in comparison to a diary-based measure in a population sample of migraine sufferers. *Pain*. 2000 Oct;88(1):41-52. doi: 10.1016/S0304-3959(00)00305-5.
12. Ramadan NM, Schultz LL, Gilkey SJ. Migraine prophylactic drugs: proof of efficacy, utilization and cost. *Cephalgia : an international journal of headache*. 1997 Apr;17(2):73-80
13. Hepp Z, Dodick DW, Varon SF, Gillard P, Hansen RN, Devine EB. Adherence to oral migraine-preventive medications among patients with chronic migraine. *Cephalgia : an international journal of headache*. 2015 May;35(6):478-88. doi: 10.1177/0333102414547138.
14. Scott AB. Botulinum toxin injection of eye muscles to correct strabismus. *Transactions of the American Ophthalmological Society*. 1981;79():734-70
15. Erbguth FJ, Naumann M. Historical aspects of botulinum toxin: Justinus Kerner (1786-1862) and the "sausage poison". *Neurology*. 1999 Nov 10;53(8):1850-3
16. Ababneh OH, Cetinkaya A, Kulwin DR. Long-term efficacy and safety of botulinum toxin A injections to treat blepharospasm and hemifacial spasm. *Clinical & experimental ophthalmology*. 2014 Apr;42(3):254-61. doi: 10.1111/ceo.12165.
17. Truong D, Comella C, Fernandez HH, Ondo WG, Dysport Benign Essential Blepharospasm Study Group. Efficacy and safety of purified botulinum toxin type A (Dysport) for the treatment of benign essential blepharospasm: a randomized, placebo-controlled, phase II trial. *Parkinsonism & related disorders*. 2008;14(5):407-14. doi: 10.1016/j.parkreldis.2007.11.003.
18. Tsui JK, Eisen A, Stoessl AJ, Calne S, Calne DB. Double-blind study of botulinum toxin in spasmody torticollis. *Lancet (London, England)*. 1986 Aug 2;2(8501):245-7
19. Greene P, Kang U, Fahn S, Brin M, Moskowitz C, Flaster E. Double-blind, placebo-controlled trial of botulinum toxin injections for the treatment of spasmody torticollis. *Neurology*. 1990 Aug;40(8):1213-8
20. Jankovic J, Schwartz K, Donovan DT. Botulinum toxin treatment of cranial-cervical dystonia, spasmody dysphonia, other focal dystonias and hemifacial spasm. *Journal of neurology, neurosurgery, and psychiatry*. 1990 Aug;53(8):633-9
21. Brin MF, Blitzer A, Fahn S, Gould W, Lovelace RE. Adductor laryngeal dystonia (spastic dysphonia): treatment with local injections of botulinum toxin (Botox). *Movement disorders : official journal of the Movement Disorder Society*. 1989;4(4):287-96
22. Brin MF, Fahn S, Moskowitz C, Friedman A, Shale HM, Greene PE, Blitzer A, List T, Lange D, Lovelace RE. Localized injections of botulinum toxin for the treatment of focal dystonia and hemifacial spasm. *Movement disorders : official journal of the Movement Disorder Society*. 1987;2(4):237-54
23. Lungu C, Karp BI, Alter K, Zolbrod R, Hallett M. Long-term follow-up of botulinum toxin therapy for focal hand dystonia: outcome at 10 years or more. *Movement disorders : official journal of the Movement Disorder Society*. 2011 Mar;26(4):750-3. doi: 10.1002/mds.23504.
24. Schnider P, Binder M, Auff E, Kittler H, Berger T, Wolff K. Double-blind trial of botulinum A toxin for the treatment of focal hyperhidrosis of the palms. *The British journal of dermatology*. 1997 Apr;136(4):548-52
25. Silberstein S, Mathew N, Saper J, Jenkins S. Botulinum toxin type A as a migraine preventive treatment. For the BOTOX Migraine Clinical Research Group. *Headache*. 2000 Jun;40(6):445-50
26. Durham PL, Cady R, Cady R. Regulation of calcitonin gene-related peptide secretion from trigeminal nerve cells by botulinum toxin type A: implications for migraine therapy. *Headache*. 2004 Jan;44(1):35-42; discussion 42-3
27. Binder WJ, Brin MF, Blitzer A, Schoenrock LD, Pogoda JM. Botulinum toxin type A (BOTOX) for treatment of migraine headaches: an open-label study. *Otolaryngology--head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery*. 2000 Dec;123(6):669-76
28. Freitag FG, Diamond S, Diamond M, Urban G. Botulinum Toxin Type A in the treatment of chronic migraine without medication overuse. *Headache*. 2008 Feb;48(2):201-9
29. Evers S, Vollmer-Haase J, Schwaag S, Rahmann A, Husstedt IW, Frese A. Botulinum toxin A in the prophylactic treatment of migraine--a randomized, double-blind, placebo-controlled study. *Cephalgia : an international journal of headache*. 2004 Oct;24(10):838-43

30. Aurora SK, Gawel M, Brandes JL, Pokta S, Vandenburg AM, BOTOX North American Episodic Migraine Study Group. Botulinum toxin type a prophylactic treatment of episodic migraine: a randomized, double-blind, placebo-controlled exploratory study. *Headache*. 2007 Apr;47(4):486-99.

31. Coté TR, Mohan AK, Polder JA, Walton MK, Braun MM. Botulinum toxin type A injections: adverse events reported to the US Food and Drug Administration in therapeutic and cosmetic cases. *Journal of the American Academy of Dermatology*. 2005 Sep;53(3):407-15.

32. Janis JE, Dhanik A, Howard JH. Validation of the peripheral trigger point theory of migraine headaches: single-surgeon experience using botulinum toxin and surgical decompression. *Plastic and reconstructive surgery*. 2011 Jul;128(1):123-131. doi: 10.1097/PRS.0b013e3182173d64.

33. Guyuron B, Varghai A, Michelow BJ, Thomas T, Davis J. Corrugator supercilii muscle resection and migraine headaches. *Plastic and reconstructive surgery*. 2000 Aug;106(2):429-34; discussion 435-7.

34. Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. *Plastic and reconstructive surgery*. 2002 Jun;109(7):2183-9.

35. Jose A, Nagori SA, Roychoudhury A. Surgical Management of Migraine Headache. *The Journal of craniofacial surgery*. 2018 Mar;29(2):e106-e108. doi: 10.1097/SCS.0000000000004078.

36. Bajaj J, Doddamani R, Chandra SP, Ratre S, Parihar V, Yadav Y, Sharma D. Comparison of Peripheral Neurectomy vs. Medical Treatment for Migraine: A Randomized Controlled Trial. *Neurology India*. 2021 Mar-Apr;69(Supplement):S110-S115. doi: 10.4103/0028-3886.315973.

37. Bovim G, Fredriksen TA, Stolt-Nielsen A, Sjaastad O. Neurolysis of the greater occipital nerve in cervicogenic headache. A follow up study. *Headache*. 1992 Apr;32(4):175-9.

38. Khan S, Schoenen J, Ashina M. Sphenopalatine ganglion neuromodulation in migraine: what is the rationale? *Cephalalgia : an international journal of headache*. 2014 Apr;34(5):382-91. doi: 10.1177/033102413512032.

39. Reed KL, Black SB, Banta CJ 2nd, Will KR. Combined occipital and supraorbital neurostimulation for the treatment of chronic migraine headaches: initial experience. *Cephalalgia : an international journal of headache*. 2010 Mar;30(3):260-71. doi: 10.1111/j.1468-2982.2009.01996.x.

40. Schoenen J, Vandervissem B, Jeangette S, Herroelen L, Vandenhende M, Gérard P, Magis D. Migraine prevention with a supraorbital transcutaneous stimulator: a randomized controlled trial. *Neurology*. 2013 Feb;80(8):697-704. doi: 10.1212/WNL.0b013e3182825055.

41. Mauskop A. Vagus nerve stimulation relieves chronic refractory migraine and cluster headaches. *Cephalalgia : an international journal of headache*. 2005 Feb;25(2):82-6.

42. Hord ED, Evans MS, Mueed S, Adamolekun B, Naritoku DK. The effect of vagus nerve stimulation on migraines. *The journal of pain*. 2003 Nov;4(9):530-4.

43. Cocores AN, Smirnoff L, Greco G, Herrera R, Monteith TS. Update on Neuromodulation for Migraine and Other Primary Headache Disorders: Recent Advances and New Indications. *Current pain and headache reports*. 2025 Feb 15;29(1):47. doi: 10.1007/s11916-024-01314-7.

44. Maxey BS, Pruitt JW, Deville A, Montgomery C, Kaye AD, Urits I. Occipital Nerve Stimulation: An Alternative Treatment of Chronic Migraine. *Current pain and headache reports*. 2022 Apr;26(4):337-346. doi: 10.1007/s11916-022-01026-w.

45. Popeney CA, Aló KM. Peripheral neurostimulation for the treatment of chronic, disabling transformed migraine. *Headache*. 2003 Apr;43(4):369-75.

46. Saper JR, Dodick DW, Silberstein SD, McCarville S, Sun M, Goadsby PJ, ONSTIM Investigators. Occipital nerve stimulation for the treatment of intractable chronic migraine headache: ONSTIM feasibility study. *Cephalalgia : an international journal of headache*. 2011 Feb;31(3):271-85. doi: 10.1177/033102410381142.

47. Silberstein SD, Dodick DW, Saper J, Huh B, Slavin KV, Sharan A, Reed K, Narouze S, Mogilner A, Goldstein J, Trentman T, Vaisman J, Ordia J, Weber P, Deer T, Levy R, Diaz RL, Washburn SN, Mekhail N. Safety and efficacy of peripheral nerve stimulation of the occipital nerves for the management of chronic migraine: results from a randomized, multicenter, double-blinded, controlled study. *Cephalalgia : an international journal of headache*. 2012 Dec;32(16):1165-79. doi: 10.1177/033102412462642.

48. Mueller OM, Gaul C, Katsarava Z, Diener HC, Sure U, Gasser T. Occipital nerve stimulation for the treatment of chronic cluster headache - lessons learned from 18 months experience. *Central European neurosurgery*. 2011 May;72(2):84-9. doi: 10.1055/s-0030-1270476.

49. Chen G, You H, Juha H, Lou B, Zhong Y, Lian X, Peng Z, Xu T, Yuan L, Woralux P, Hugo AB, Jianliang C. Trigger areas nerve decompression for refractory chronic migraine. *Clinical neurology and neurosurgery*. 2021 Jul;206():106699. doi: 10.1016/j.clineuro.2021.106699.

50. Guyuron B, Kriegler JS, Davis J, Amini SB. Comprehensive surgical treatment of migraine headaches. *Plastic and reconstructive surgery*. 2005 Jan;115(1):1-9.

51. Totonchi A, Guyuron B, Ansari H. Surgical Options for Migraine: An Overview. *Neurology India*. 2021 Mar-Apr;69(Supplement):S105-S109. doi: 10.4103/0028-3886.315999.

52. Alrahbeni T, Mahal A, Alkhouri A, Alotaibi HF, Rajagopal V, Behera A, Al-Mugheed K, Khatib MN, Gaidhane S, Zahiruddin QS, Shabil M, Bushi G, Rustagi S, Kukreti N, Satapathy P, Mohapatra RK, Dziedzic A, Padhi BK. Surgical interventions for intractable migraine: a systematic review and meta-analysis. *International journal of surgery (London, England)*. 2024 Oct 1;110(10):6306-6313. doi: 10.1097/JJS.0000000000001480