

# Chapter 10

## Surgery for Migraine: An Evidence-Based Review

Ibrahim Khansa and Jeffrey E. Janis

### Introduction

Migraine headaches are believed to result from irritation of the trigeminal nerve, leading to dural inflammation [1]. This is mediated by factors such as calcitonin gene-related peptide, substance P, and neurokinin A [1–3]. According to the peripheral theory of migraine headaches, peripheral noxious stimuli irritate branches of the trigeminal nerve, or cervical nerves and trigger the central events [4]. Patients suffering from migraine headaches often have point tenderness precisely overlying a trigger point at which a sensory nerve is compressed by muscle, fascia, artery, or bone [5].

The peripheral theory of migraine headaches is validated by the efficacy of both botulinum toxin (which temporarily weakens muscles that compress sensory nerves involved in migraine pathogenesis), and surgical trigger point decompression. The latter has been proven to be efficacious by numerous studies [6–12], including a prospective randomized trial using sham surgery as a placebo control [10].

In this chapter, the diagnostic methods commonly used in the localization of the trigger site or sites responsible for the migraine headaches are delineated. The anatomy of the compression points of the frontal, temporal, occipital, nasal and atypical trigger sites are then described. This is followed by a description of the different procedures to decompress those trigger points, and a summary of the published clinical outcomes of surgical decompression.

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## Trigger Site Localization

The surgical treatment of migraine headaches is indicated for patients with chronic migraines diagnosed by a neurologist, refractory to medical treatment. Once chronic migraine headaches are diagnosed, the accurate identification of the peripheral trigger site(s) involved is essential to obtaining successful outcomes with surgical decompression.

The first step in trigger site localization is a detailed history. This helps with migraine localization in two ways. First, most patients will, when asked, be able to pinpoint with one finger the location where their migraine headaches begin [13]. Second, an accurate history of the nature and timing of the headaches, as well as their exacerbating, ameliorating, and associated factors, may elucidate a pattern that fits known constellations of symptoms. For example, patients with headaches originating from a nasal trigger site often have headaches that originate behind the eyes, typically in the morning, exacerbated by menstrual periods and alleviated by nasal decongestants [14].

The second step is physical examination. Digital pressure over a suspected trigger site may reveal tenderness. An intranasal examination may reveal mucosal contact points leading to migraine headaches. Finally, handheld Doppler examination over a suspected auriculotemporal, lesser occipital or atypical trigger site may reveal an arterial signal, indicating arterial compression of a sensory nerve branch as a likely source of migraines, or even potential arteritis.

Patients who present with an active migraine headache offer an opportunity for the use of diagnostic nerve blocks: a small amount of local anesthetic is injected precisely into the suspected trigger site. Resolution of the migraine headache is diagnostic of the trigger site. However, an ineffective nerve block does not always rule out a peripheral trigger site as the trigeminal tree may already be inflamed from a long-standing headache by the time the block is performed.

Patients who present without an active headache, and with suspected muscular compression of a nerve, are good candidates for diagnostic botulinum toxin A injection. The toxin is injected into the corrugator supercilii muscle (in the case of a suspected frontal trigger site), the temporalis muscle (in the case of a suspected zygomaticotemporal trigger site), or the semispinalis capitis muscle (in the case of a suspected central occipital trigger site). Improvement in migraine headache intensity, frequency, and duration over the following weeks to months helps identify the injected trigger site as contributory to the headaches.

Finally, in patients with a suspected rhinogenic trigger site, computed tomography imaging may reveal mucosal contact points, septal deviation/spurs, turbinate hypertrophy, and/or concha bullosa.

## Frontal Trigger Site

The frontal migraine trigger site includes the supraorbital (SON) and supratrochlear (STN) nerves.

### *Compression Points*

The first potential compression point of the SON is the foramen or notch through which it exits the orbit. A notch is present 83% of the time, and located 25 mm lateral to the midline [15]. A foramen is present 27% of the time, and located 31 mm lateral to the midline [16–18]. In 10% of patients, both a foramen and a notch are present [19].

The SON then divides into a superficial and deep branch. In 78% of individuals, one or both of these branches travel through the corrugator supercilii muscle (CSM) [20, 21], which extends from 3 mm lateral to the midline to 85% of the distance to the lateral orbital rim, with its apex 33 mm above the nasion at the level of the lateral limbus [20]. In 40% of individuals, the branches off the deep branch of the SON alone travels through the CSM. In 34% of individuals, branches off both the superficial and deep branches of the SON travel through the muscle. In 4% of individuals, only branches off the superficial branch travel through the muscle, and in 22% of individuals, no branches of the SON travel through the muscle [21]. The SON fibers may then be compressed more superiorly by the interdigitations of the horizontal CSM fibers with vertical frontalis muscle fibers [21].

The first potential compression point of the STN is the supratrochlear notch (present in 72% of individuals) or foramen (28% of individuals) [22]. This compression point is located 16 to 23 mm lateral to the midline [6]. When a foramen is present, it is located 4 mm cranial to the superior orbital rim [22]. The STN then divides into two branches after exiting the orbit. In 84% of individuals, both branches travel through the CSM (18.8 to 19.6 mm lateral to the midline), exiting it 15 mm cranial to the superior orbital rim [22]. In 4% of individuals, only one branch of the STN travels through the CSM. In 12% of individuals, no branches of the STN travel through the CSM.

As the STN travels further superiorly, it may also be compressed by the interdigitations between the horizontal CSM fibers and the vertical frontalis muscle fibers [21].

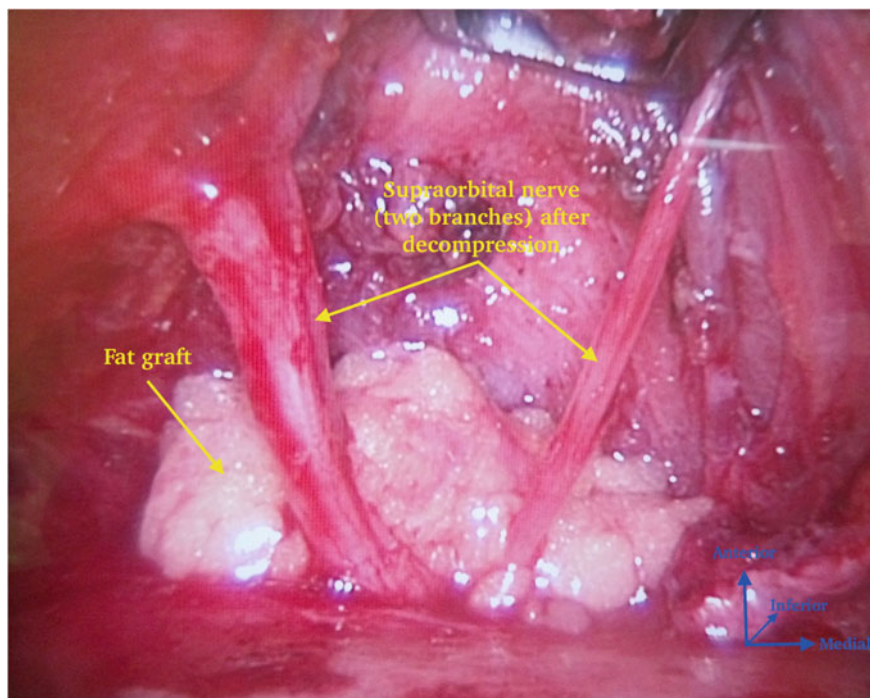
## *Surgical Decompression*

There are three described approaches to decompress the frontal trigger site. For symmetry purposes, these procedures are usually performed bilaterally.

In a transpalpebral approach, a standard upper blepharoplasty incision is used. Dissection is continued through the orbicularis oculi muscle, to reach the plane just superficial to the orbital septum. Dissection is then carried superiorly in this plane until the superior orbital rim is reached. The SON and STN are identified as they exit the orbit through notches or foramina. Osteotomies can be performed to turn any foramina into wide-mouthed notches. The supraorbital and supratrochlear arteries and veins may need ablation if they abut or compress the nerves. Limited intraconal dissection of both nerves should also be performed in order to make sure that no bony compression remains [23]. The corrugator supercilii and depressor supercilii and procerus muscles are then excised using bipolar electrocautery. A fat graft is usually used to replace the volume of those excised muscles.

In an endoscopic approach, the brow is accessed through four to six endoscopic ports, which are sagittally oriented and located behind the hairline. One incision is marked centrally, two paramedian incisions are marked approximately 7 cm lateral to the midline on each side (medial to the temporal crest), and two temporal incisions are marked approximately 10 cm lateral to the midline on each side (lateral to the temporal crest, approximately 3 cm lateral to the paramedian incisions). The central and paramedian incisions are carried to the subperiosteal plane, and the temporal incisions are carried to the deep temporal fascia. A periosteal elevator is then inserted into each temporal incision to release the temporal fusion line. Subperiosteal dissection is performed from cranial to caudal toward the superior orbital rims bilaterally. About 1 cm above the rim, the periosteum is incised, revealing the corrugator muscle. The supraorbital and supratrochlear artery and vein are ablated. The SON and STN are carefully dissected, and the CSM is excised completely in piecemeal fashion with an endoscopic grasper (Fig. 10.1). If a foramen is present, it may be addressed through a separate upper eyelid stab incision with a 2 mm osteotome, with direct endoscopic visualization.

In the direct brow approach, an incision is made along the medial two-thirds of the upper border of the brow. Dissection is carried through the orbicularis oculi muscle, then cranially in the plane deep to the orbicularis oculi muscle and superficial to the orbital septum. The remainder of this procedure is similar to the transpalpebral approach described above.



**Fig. 10.1** Endoscopic visualization of the supraorbital nerve (two branches) after decompression. A fat graft is used to buttress the nerve branches and to replace the volume of the excised corrugator, procerus and depressor supercilii muscles

### *Outcomes of Decompression*

Multiple studies have examined the clinical outcomes of surgical decompression of the frontal trigger site [7–12]. Complete migraine elimination has been reported in 35% [12] to 64% [9], and elimination or significant improvement in 79.5% [8] to 99% [9].

### **Temporal Trigger Site**

The temporal compression point includes the zygomaticotemporal (ZTN) and auriculotemporal (AT) nerves. In 13–40% of individuals, the ZTN and AT have interconnecting branches [24, 25].

## ***Compression Points***

The first potential compression point of the ZTN is the foramen through which it exits the orbit and enters the temporal fossa [26], located 6.7 mm lateral to the lateral orbital rim, and 7.9 mm cranial to the nasion.

The second potential compression point of the ZTN occurs as it travels through the temporalis muscle. This occurs in 50% of individuals [24]. In the remaining 50%, the ZTN courses between the temporal periosteum and the temporalis muscle before piercing the deep temporal fascia 16.9 mm lateral and 6.5 mm cranial to the lateral palpebral commissure [25]. The sentinel vein is a reliable landmark for locating the ZTN: The ZTN is usually found approximately 1 cm lateral and 0.5 mm caudal to the sentinel vein [27].

The first two compression points of the AT are both fascial bands, which occur 13.1 mm anterior and 5 mm cranial to the anterosuperior external auditory meatus [28] (present in 100%), and 11.9 mm anterior and 17.2 mm cranial to the anterosuperior external auditory meatus (present in 85%).

In 80% of individuals, the AT intersects with the superficial temporal artery [29], which constitutes the third potential compression point. The intersection is simple in 81.2% of cases (19.2 mm anterior and 39.5 mm superior to the anterosuperior external auditory meatus) and helical in 18.8% (between 20.0 mm and 24.7 mm anterior, and 53.7 mm and 62.7 mm cranial to the anterosuperior external auditory meatus) [27, 30].

## ***Surgical Decompression***

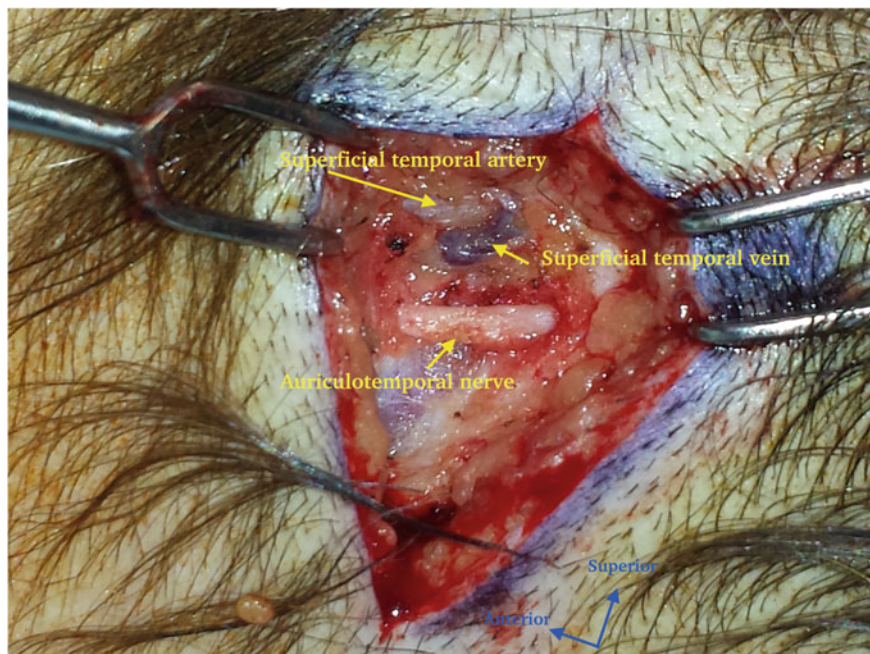
There are three described approaches to the decompression of the ZTN.

In an endoscopic approach, when combined with the SON and STN decompressions previously described, the same four to six sagittally oriented incisions are marked behind the hairline. The ZTN can be accessed without additional incisions in this fashion. If addressed in isolation, however, the central incision(s) are not created. Instead the paramedian and temporal incisions will suffice. The temporal access incision is used to dissect along the superficial surface of the deep temporal fascia, from cranial to caudal, until the superficial temporal fat pad is visualized about 2 cm cranial to the zygomatic arch. The superficial layer of the deep temporal fascia is incised, and that layer and the underlying superficial temporal fat pad are elevated, continuing caudal dissection along the deep layer of the deep temporal fascia, to the lateral orbital rim. The sentinel vein is identified and controlled (if necessary). The ZTN can be identified about 1 cm lateral and 0.5 mm caudal to the sentinel vein [26]. The ZTN is separated from its associated artery in order to control them individually. The zygomaticotemporal artery is ablated with cautery. The nerve can either be decompressed (by widening its entry and exit point into the deep temporal fascia/temporalis muscle) [31], or avulsed by applying traction to it, allowing its proximal end to retract into the temporalis muscle.

In a transpalpebral approach, described by Austen and Gfrerer [32], a standard upper blepharoplasty incision is made, and dissection is performed in a preseptal plane to the lateral orbital rim. The plane just superficial to the deep temporal fascia is dissected. This allows identification of the sentinel vein and ZTN just lateral to the lateral orbital rim. The nerve is then decompressed or avulsed, as described above.

In a modified Gillies approach, described by Peled [33], a single, larger 3.5 cm incision is made 5–7 mm posterior to the temporal hairline, and taken to the deep temporal fascia. The plane just superficial to the deep temporal fascia is dissected under direct visualization toward the lateral canthus, until both the sentinel vein, and the ZTN and its associated artery are visualized. The nerve is then decompressed or avulsed, as described above.

Decompression of the AT is usually performed via a direct approach. The patient is asked to use an indelible marker to place a mark over the point of maximal tenderness during a migraine attack. A handheld Doppler is used to confirm the presence of an arterial signal at that point [34], indicating an anterior branch of the superficial temporal artery. A small incision is made directly over the mark. This will allow identification of the main trunk or a branch of the AT. Its relationship to the superficial temporal artery and vein is assessed, and the nerve is decompressed by ablating any neighboring or crossing branches of the artery and vein (Fig. 10.2).



**Fig. 10.2** Visualization of the auriculotemporal nerve, in close proximity to branches of the superficial temporal artery and vein

## ***Outcomes of Decompression***

Multiple studies have examined the clinical outcomes of the decompression of the ZTN [9, 10, 12, 30, 35]. Complete migraine elimination has been reported in 52.6% [12] to 63% [9] of patients, and significant improvement or elimination has been reported in 85% [34] to 100% [10].

## **Occipital Trigger Site**

The central occipital trigger site includes the greater occipital (GON) and third occipital (TON) nerves, and the lateral occipital trigger site consists of the lesser occipital nerve (LON).

## ***Compression Points***

The first compression point of the GON occurs as it interacts with fascial bands between the obliquus capitis muscle and the semispinalis capitis muscle. This is located 77.4 mm caudal and 20.1 mm lateral to the external occipital protuberance (EOP) [36]. The second compression point, present in 90% of individuals [37], occurs when the GON enters the deep surface of the semispinalis capitis muscle. This occurs 59.7 mm caudal and 17.5 mm lateral to the EOP [35]. The third compression point occurs as the GON exits the superficial surface of the semispinalis muscle, 34.5 mm caudal and 15.5 mm lateral to the EOP [35, 38, 39]. The fourth compression point occurs as the GON enters the trapezius tunnel 21 mm caudal and 24 mm lateral to the EOP [35]. The fifth compression point occurs as the GON pierces the trapezius tendon 4.4 mm caudal and 37.1 mm lateral to the EOP [35]. The sixth potential compression point of the GON, present in 54% of individuals, is the occipital artery. When this is a simple intersection, it is 10.7 mm caudal and 30.3 mm lateral to the EOP. When it is a helical intertwining, it is 24.9 to 1 mm caudal, and 25.3 to 42.1 mm lateral to the EOP [35].

The potential compression point of the TON occurs as it emerges from the superficial aspect of the semispinalis capitis muscle. This is located 61 mm caudal to the inferior external auditory canal, and 13 mm lateral to the posterior midline [40].

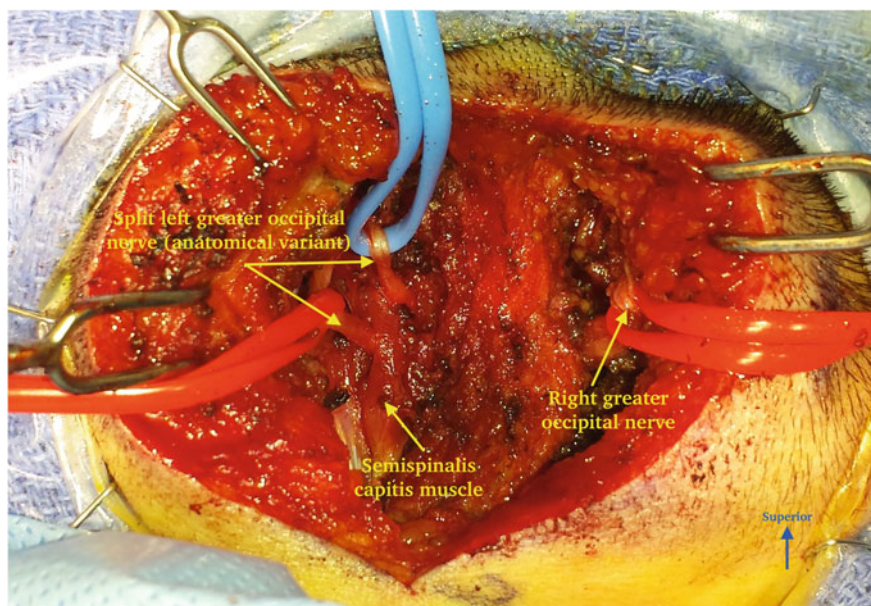
The first potential compression point of the LON occurs as it emerges from the sternocleidomastoid (SCM). This is located along the posterior border of the SCM in 86.7%, and through the SCM in 13.3% [39]. This compression point is located 53.2 mm caudal to the inferior external auditory canals, and 61–69 mm lateral to the posterior midline [39, 41]. The second compression point of the LON is its intersection with the occipital artery, present in 55% of individuals 20 mm caudal to



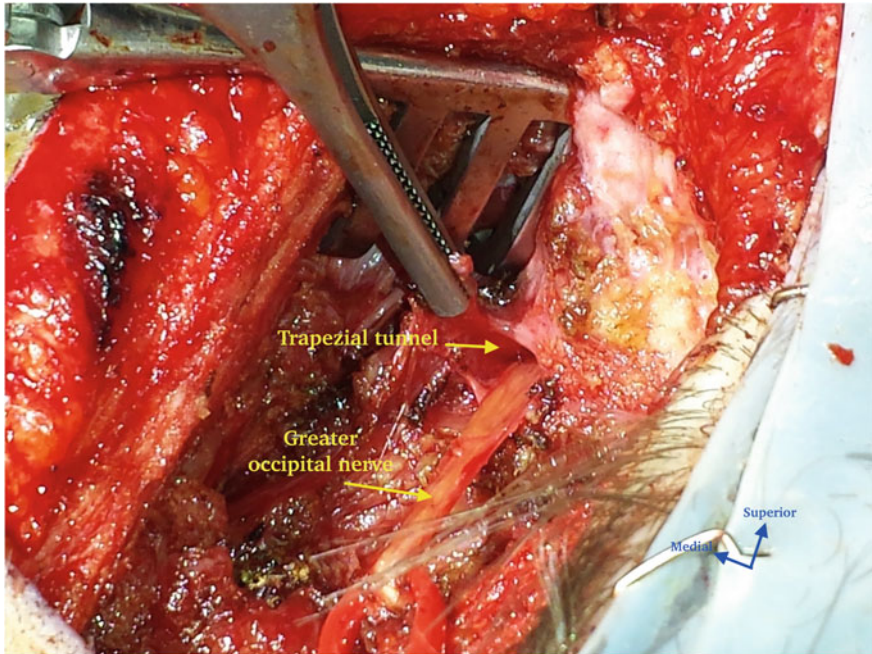
the anterosuperior external auditory canals, and 51 mm lateral to the posterior midline [40]. This is a simple crossing in 82% of cases, and a helical intertwining in 18%. The third compression point of the LON is a fascial band, present in 20% of individuals, located 13.1 mm inferior to the anterosuperior external auditory canals, and 47 mm lateral to the posterior midline [40].

### ***Surgical Decompression of the Central Occipital Site (GON and TON)***

A 4 cm vertical incision is made in the posterior midline, within the hairline. Dissection is made directly to the median raphe of the trapezius muscle. The skin and subcutaneous tissue are then elevated off the trapezius muscle bilaterally for 1 cm. The trapezius fascia and trapezius muscle are then incised 1 cm lateral to the midline, revealing the underlying vertically oriented fibers of the semispinalis muscle. The TON and GON are then both identified in the plane between the semispinalis capitis and trapezius muscles (Fig. 10.3). The TON is usually avulsed, allowing its proximal end to retract into muscle. The GON is decompressed from inferior to superior, starting with the release of the obliquus capitis muscle and its associated fascial bands. Subsequently, a rectangular portion of the semispinalis capitis muscle medial to the GON, and a triangular portion lateral to the GON, are



**Fig. 10.3** Bilateral greater occipital nerves, with an anatomical variant split nerve on the left



**Fig. 10.4** The greater occipital nerve as it enters the trapezial tunnel

resected in order to relieve compression of the GON by this muscle. The trapezial tunnel and the nuchal fascia surrounding the GON are then lysed (Fig. 10.4), and if an intersection between the nerve and the occipital artery is present, the artery is ablated using cautery. A three-sided flap of fat is then developed and transposed under the nerve to the median raphe.

### ***Surgical Decompression of the Lesser Occipital Site (LON)***

The patient is asked to use an indelible marker during a migraine attack to indicate the site of maximal tenderness in the posterior lateral neck region. A handheld Doppler is used to verify the presence of an arterial signal at that site.

There are two potential approaches to the LON. If the GON and/or TON are being decompressed concurrently, the LON can be accessed through the midline incision, although this can be more difficult. Dissection is performed in a lateral direction to the point previously marked by the patient. The LON can usually be identified in the subcutaneous tissue. The nerve is traced proximally and distally. Proximally, if it pierces the sternocleidomastoid (SCM), a segment of surrounding SCM may be resected. Distally, if it intersects with a branch of the occipital artery, that branch is dissected free from the LON and ligated using bipolar electrocautery.

If the LON is being decompressed in isolation (the senior author's preferred approach), or if the trigger site is too lateral to be accessed through the midline incision, a small incision is made directly over the mark indicated by the patient, and the nerve is decompressed as described above.

### *Outcomes of Decompression*

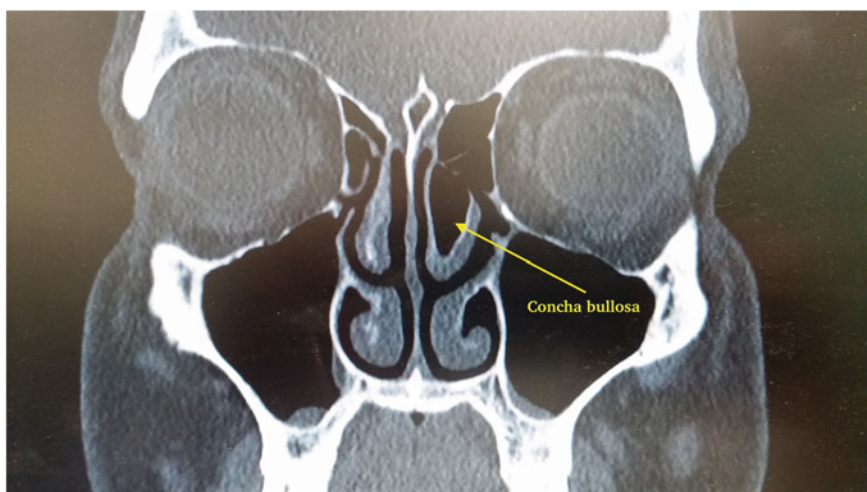
Multiple studies have examined the clinical outcomes of GON decompression [9, 10, 12, 41]. Complete migraine elimination has been reported in 43.4% [42] to 62% [9] of patients. Significant improvement or complete elimination has been reported in 80.5% [41] to 100% [9].

There are few clinical studies reporting outcomes of LON and TON release, and most patients in those studies underwent concomitant greater occipital nerve release [43].

### **Nasal Trigger Site**

#### *Compression Points*

The nasal trigger site is due to intranasal mucosal contact points, which cause irritation of branches of the trigeminal nerve [44]. These are usually between the septum and the superior or middle turbinate [45]. A pneumatized turbinate (concha bullosa) may also lead to a contact point with the septum (Fig. 10.5) [46].



**Fig. 10.5** Pneumatized left middle turbinate (concha bullosa)

## ***Surgical Decompression***

Depending on the specific inciting contact point, decompression of the nasal trigger site may consist of septoplasty, turbinate reduction, or both.

The septum is accessed through a modified Killian incision 1 cm posterior to the caudal border of the septum, followed by subperichondrial dissection of the quadrangular cartilage to the perpendicular plate of the ethmoid. A cartilaginous L-strut with 10 mm caudal and anterior limbs is preserved, and the remaining cartilage is resected. Remnants of the perpendicular plate are then removed.

The turbinate is accessed through a small mucosal incision, through which submucosal dissection is performed. A microdebrider is used to reduce the size of the turbinate head, and outfracture of the turbinate is performed with a Vienna speculum. A similar approach is used for the treatment of concha bullosa, medializing the middle turbinate after decompression.

## ***Outcomes of Decompression***

Multiple studies have examined the clinical outcomes of nasal trigger site decompression [9, 10, 12, 43, 47]. Complete migraine elimination has been reported in 34% [9] to 62.5% [12] of patients. Significant improvement or elimination has been reported in 65% [48] to 100% [12] of patients.

## **Atypical Trigger Sites**

### ***Compression Points***

Other trigger sites may be present, but are quite variable between individuals and have not been fully studied with cadaver dissections. These trigger sites often consist of a small nerve branch compressed by a blood vessel. They are best identified by asking the patient to point with one fingertip at the site of maximal tenderness, where the headache begins. They can be confirmed with an arterial Doppler signal over the point indicated by the patient.

## ***Surgical Decompression***

Surgical decompression of an atypical trigger site is similar to that described for the auriculotemporal nerve. A small incision is made over the point indicated by the patient and the Doppler signal, and the compressed nerve is identified. Vascular

structures neighboring or crossing the nerve are then ablated with cautery, and any fascial bands are released.

## Summary

Numerous cadaver and clinical studies have delineated the precise location of the compression points involved in the pathogenesis of migraine headaches. Accurate diagnosis of the trigger site or sites responsible for the migraine headache, coupled with complete surgical decompression of those sites, can achieve migraine headache resolution or significant improvement in the vast majority of patients with refractory, chronic migraine headaches.

**Disclosures** Dr. Janis is a consultant for LifeCell, has received prior honoraria from Pacira and Bard, and receives royalties from CRC Press. Dr. Khansa has no relevant financial disclosures

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