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HEADACHE CURRENTS

Narrative review of peripheral nerve blocks for the management of headache

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Abstract

Objective: To provide an overview of the current available literature on peripheral nerve blocks for the management of migraine and other headache disorders in adults.

Background: Peripheral nerve blocks have been commonly performed in the headache practice for migraine, cluster headache, occipital neuralgia, and other headache disorders, despite a paucity of evidence supporting their use historically. In the past decade, there has been an effort to explore the efficacy and safety of peripheral nerve blocks for the management of headache, with the greatest interest centered around greater occipital blocks.

Design: We performed a search in PubMed using key words including "occipital nerve blocks," "peripheral nerve blocks," "occipital nerve," "migraine," "cluster headache," and "neuralgia." We reviewed the randomized controlled trials (RCTs), observational studies, and case series, and summarized the anatomy, techniques, and the evidence for the use of peripheral nerve blocks in different headache disorders, with particular focus on available RCTs. Case reports were included for a detail review of adverse events.

Results: Of 12 RCTs examining the use of greater occipital nerve blocks for migraine, all but one demonstrate efficacy with reduction in headache frequency, intensity, and/or duration compared to placebo. Studies have not demonstrated a difference in clinical outcomes with the use of corticosteroids for nerve blocks compared to blocks with local anesthetic in the treatment of migraine. There are two RCTs supporting the use of greater occipital blockade for cluster headache, both showing benefit of suboccipitally injected corticosteroid. One RCT suggests benefit of greater occipital nerve blocks for cervicogenic headache. Observational studies and case series/reports show that greater occipital nerve block may be effective in prolonged migraine aura, status migrainosus, post-dural puncture headache, and occipital neuralgia. Overall, peripheral nerve blocks are well tolerated. Serious side effects are rare but have been reported, including acute cerebellar syndrome and infection.

Conclusions: Peripheral nerve blocks, especially occipital nerve blocks, are a viable treatment option for migraine and may be helpful in cluster headache as a transitional therapy or rescue therapy. Additional prospective studies are needed to investigate

Abbreviations: AHS, American Headache Society; ATN, auricular temporal nerve; CM, chronic migraine; EM, episodic migraine; GON, greater occipital nerve; LAST, local anesthetic systemic toxicity; LON, lesser occipital nerve; RCT, randomized controlled trial; SON, supraorbital nerve; STN, supratrochlear nerve; TMJ, temporomandibular joint.

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the efficacy and safety of occipital nerve blocks for long-term migraine prevention, as well as for other headache disorders, such as occipital neuralgia.

KEYWORDS
cluster, injection, migraine, occipital neuralgia, peripheral nerve block

INTRODUCTION

Peripheral nerve blocks have been used for decades in the treatment of cranial neuralgias and multiple headache disorders, including migraine,[1–4] cluster headache,[5–8] and cervicogenic headache.[9–11]

Nerve blocks are generally well tolerated and may provide rapid pain relief that can last up to days or weeks.[12] Technique varies but nerve blocks usually involve administration of local anesthetic with or without corticosteroid. When nerve blocks are used for headache disorders, the greater occipital nerve (GON) is the most common target. Some centers will block the lesser occipital nerve (LON) together with the GON.[13] The supraorbital (SON), supratrochlear (STN), and auriculotemporal (ATN) nerves are also common nerve targets. Local anesthetics inhibit conduction in the sensory nerve fibers within mixed nerves, but headache relief often far outlasts the duration of action of local anesthesia.[12]

The precise mechanism underlying prolonged headache relief following nerve blocks is unknown, but may involve central pain modulation.[14–16] The upper cervical nerve roots are anatomically and functionally connected to trigeminal pathways,[14,17] with convergence in the trigeminal cervical complex.[18] Recent studies suggest Lamina I in the dorsal horn may play a significant role in the trigeminocervical integration, with upper cervical Lamina I projection neurons and local-circuit neurons receiving diverse input from trigeminal and cervical C-fibers and A-delta fibers.[19,20] Interestingly, anesthetic blockade of the GON, from the posterior division of C2, has been demonstrated by electrophysiology and functional imaging to reduce activation of trigeminal nociceptors,[16,21] though changes in trigeminal function do not always translate into direct clinical benefit.[22]

The literature for peripheral nerve blocks varies widely in trial design, technique of blockade, duration of follow-up, and primary outcomes studied. Until recently, there were only a handful of randomized controlled trials (RCTs) to support the use of nerve blocks for any headache disorder.[2,23] However, in the last decade, there has been a concentrated effort to expand our understanding of the efficacy of nerve blocks for head and face pain, with the greatest efforts devoted to GON blocks. This review starts with an overview of the relevant nerve anatomy and technique for nerve blocks, then highlights the accruing evidence for the use of GON blocks in adult patients with migraine, cluster headache, and additional select headache disorders, with special attention to RCTs. Finally, we discuss pertinent topics of interest, including the current understanding of the need for the use of corticosteroids in nerve blocks.

NERVE BLOCK ANATOMY AND TECHNIQUE

To block a terminal branch of the trigeminal or cervical nerves, it is necessary to understand the anatomy of the nerve origin and associated landmarks. Prior to a nerve block, the target area should be cleaned with an alcohol swab, chlorhexidine, or a similar cleaning solution before injecting. Many nerves run alongside arteries, and to avoid arterial injection, negative aspiration is recommended. Within 5–15 min after a successful block, the patient should have reduced sensation to pin and light touch in the dermatome of the nerve being blocked, even distal to the site of the injection.

The most common local anesthetics used in RCTs include short-acting lidocaine (1%, 2%, occasionally 5%) and long-acting bupivacaine (0.25% or 0.5%), with open-label studies less commonly using medium-acting mepivacaine (2%) or long-acting ropivacaine (0.5% or 1%) or levobupivacaine (0.25%).[24,25] Some studies describe using mixtures of lidocaine and bupivacaine, and when lidocaine and bupivacaine are used together, the recommended ratio ranges from 1:1 to 1:3 (lidocaine: bupivacaine).[23] Per manufacturer guidance, lidocaine and bupivacaine dosing in a given session should be limited to a maximum of 300 and 175 mg, respectively.[26,27] There is little experience with the use of bupivacaine without epinephrine at doses above 175 mg.[24]

Greater occipital nerve block

By far, the GON block is the most well studied block for the treatment of headaches and pericranial neuralgia. The GON arises from the posterior division of C2 and gives sensation to the medial portion of the posterior scalp (see Figure 1A).[28] There are numerous techniques described on the approach to blocking this nerve.[1,29–33] During the procedure, patients are typically sitting upright or leaning forward. However a lateral decubitus[28] or prone position have also been described.[34,35] To find the location of the GON, one of several methods may be used. The method most used in RCTs involves palpating the occipital protuberance and mastoid process. The target area for the GON block is approximately one third of the distance between these two points, starting from the occipital protuberance (see Figure 1B).[1,3,36,37] Another method uses distance from the occipital protuberance, typically 1.5–2 cm lateral and 2–3 cm inferior.[4,30,38–40] Clinicians can also palpate for the occipital artery pulse about 2 cm lateral to the occipital protuberance. The occipital nerve is often, but not always, just medial to the artery.[23] After the identification of the probable occipital nerve location using one of these or other methods, it is common to palpate for an area of maximal tenderness,

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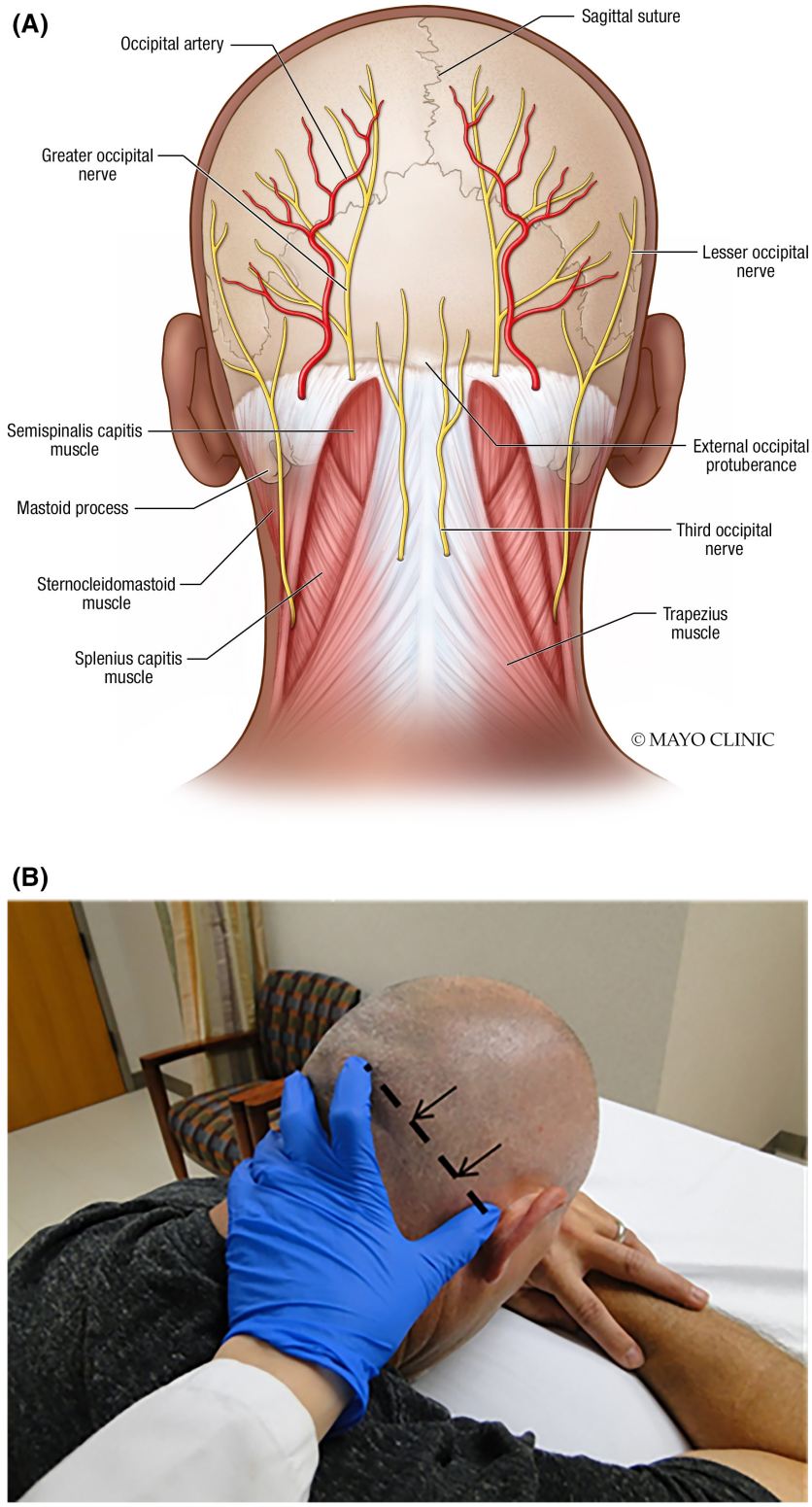

FIGURE 1 (A) Anatomy of greater and lesser occipital nerves. (B) Landmarks for greater and lesser occipital nerve blocks. Figure 1a modified from Harbell et al.[92] Used by permission from Mayo Foundation for Medical Education and Research, all rights reserved. Figure 1b participant gave written permission for use. Used by permission from CE Robertson, all rights reserved. [Color figure can be viewed at wileyonlinelibrary.com]

as this may increase the accuracy of the block.[12] According to the procedural group consensus guideline for the American Headache Society (AHS), it is recommended that providers use a 25-, 27-, or 30-gauge needle and a 3 or 5 ml syringe. The AHS guideline suggests inserting the needle to a depth of 3–4 mm,[23] but another described technique is to insert the needle perpendicular to the skin to a point where it gently contacts the periosteum, then pulling back slightly before injecting.[28,38,39,41,42] After negative aspiration to verify that

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the needle is not intravascular, about 1.5–3 ml solution is injected either in a single injection or a fan-like distribution.[23]

Lesser occipital nerve block

The LON arises from the ventral rami of C2–C3, ascending along the posterior border of the sternocleidomastoid to supply sensation to the lateral posterior scalp and sometimes part of the pinna. Though the LON block is included in most guidelines and reviews on nerve blocks for headache disorders,[23,28,43] there is minimal evidence to support its use in the management of headaches. In clinical practice, blockade of the LON may be combined with the blockade of the GON, typically with similar technique and injectate. The LON is found approximately two thirds of the way along the distance from the occipital protuberance to the mastoid (see Figure 1A), palpating for maximal tenderness in the region.[23]

Supraorbital and supratrochlear blocks

The SON and STN nerves are terminal branches of the frontal nerve, the largest branch of the ophthalmic division of the trigeminal nerve (see Figure 2). The SON exits through the supraorbital foramen, supplying sensation to the skin of the upper eyelid, forehead, and anterior scalp, sometimes as far back as the lambdoid suture. Patients are typically placed in a supine position for blockade of these two nerves, and the block is typically with anesthetic alone (avoiding corticosteroids out of concern for cosmetic adverse effects).[23] The SON can be localized by palpating for the SON foramen/notch, within the eyebrow, along the mid-pupillary line. Using a 1 or 3 ml syringe and a 30-gauge needle, the needle is introduced at the superior margin of the orbit, just slightly above the brow line to a depth of about 3–4 mm and after negative aspiration, 0.5–1 ml of anesthetic is injected. Care should be taken to avoid insertion of the needle into the SON foramen.

The STN runs in the superior orbit above the trochlea, exiting through the frontal notch, medial to the SON to supply sensation to the medial upper eyelid and forehead. Using a 1 or 3 ml syringe and a 30-gauge needle, 0.5–1 ml of anesthetic is injected at the supero-medial orbit, about one fingerbreadth lateral to the procerus (along the medial border of corrugator), to a depth of about 3–4 mm.[23,28]

Auriculotemporal nerve block

Like the LON block, the ATN block is often included in peripheral nerve block guidelines for headaches, without significant evidence of benefit. The ATN is a terminal branch of the mandibular division of the trigeminal nerve, emerging from behind the temporomandibular joint (TMJ) to course onto the face (see Figure 3). It supplies sensation to the skin over the posterior temple, tragus,

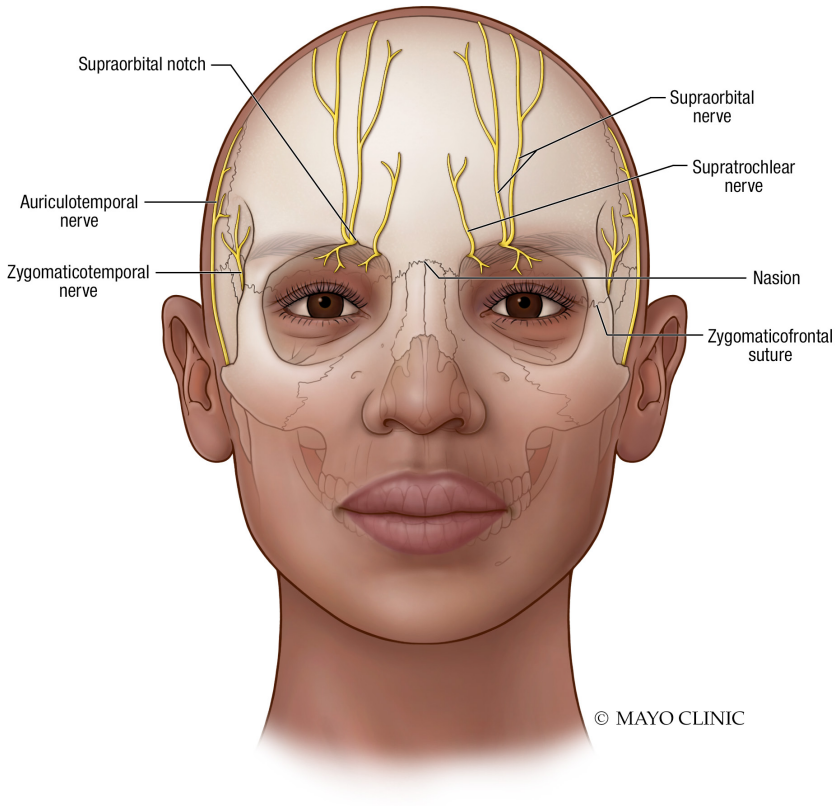

FIGURE 2 Anatomy of supraorbital nerves. Modified from Harbell et al.[92] Used by permission from Mayo Foundation for Medical Education and Research, all rights reserved. [Color figure can be viewed at wileyonlinelibrary.com]

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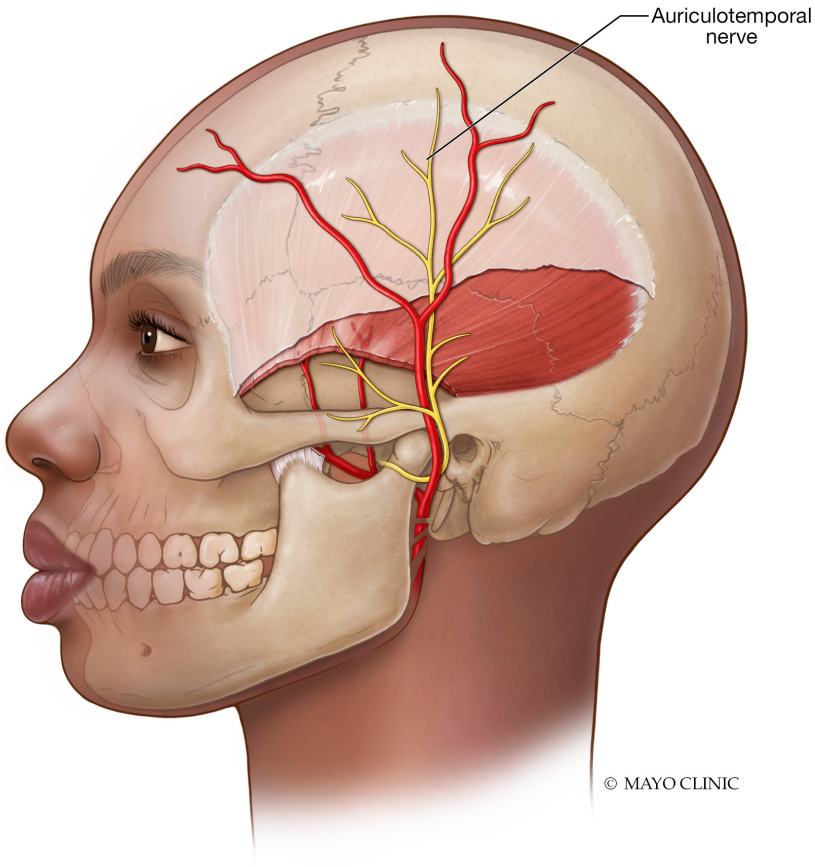

FIGURE 3 Anatomy of auriculotemporal nerves Modified from Harbell et al.[92] Used by permission from Mayo Foundation for Medical Education and Research, all rights reserved. [Color figure can be viewed at wileyonlinelibrary.com]

TMJ, and part of the pinna and ear canal. To localize the ATN for injection, the temporal artery can be located by palpation for pulse in front of the tragus, and the needle inserted for injection 2 mm anterior to this location. Using a 30-gauge needle, 0.5–1 ml is typically injected at this proximal portion of the nerve, at a depth of 4–6 mm. Additional injections of 0.25 ml may be given at the temporal fossa, where the distal branches are located.[23] The ATN is located close to the superficial temporal artery, so negative aspiration prior to injection is recommended to avoid inadvertent intraarterial injection. Because of the proximity of the ATN to the facial nerve, there is also risk of transient facial paresis with this injection, with reported incidence as high as 8.6% in one study, using 5 ml of anesthetic.[44] To minimize complications such as facial paresis with the ATN block, lower volumes of injectate and ultrasound guidance may be used.[39]

INDICATIONS FOR NERVE BLOCKS

Migraine

There are now 12 RCTs that examine the evidence for GON blocks in migraine, discussed in greater detail in Table 1. Of the trials that compared GON blocks to a placebo for the treatment of migraine, all but one of these[3] showed at least some benefit of GON blocks, most often in headache severity.[4,29,30,36,38,39,45–47] In the trial with no significant benefit, there is speculation that the placebo group (0.25 ml 1% lidocaine) may have produced a therapeutic effect, making the difference between groups difficult to detect.[25]

In patients who receive a GON block during a migraine attack, migraine-related pain, allodynia, and photophobia may be reduced within 5 min,[48] with randomized studies showing improved headache severity at 20 min,[37] 30 min,[47] and 120 min[38] after GON blocks. The reported duration of effect varies between studies and among individual patients, but many studies report some level of response for days to weeks. Ashkenazi et al.[37] reported a mean duration of headache freedom of 2.7 ± 3.8 days in patients with CM receiving a GON block and 12 trigger point injections with lidocaine and 1.0 ± 1.1 day in patients receiving lidocaine with triamcinolone (no significant different between these groups, $p = 0.67$). In their prospective cohort, Afridi et al. reported patients with migraine had a mean duration of pain freedom of 9 days (median 6 days), with a mean partial response (more than 30% headache reduction in severity or frequency) duration of 61 days (median 30 days).[12] Interestingly, there was a mean latency of response of 2 days in their trial.[12] Cuadrado et al. followed patients with chronic migraine (CM) for 1 week after a single round of bilateral GON blocks and found that patients receiving bupivacaine had a greater reduction in moderate to severe headache days than

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TABLE 1 Randomized controlled trials of peripheral nerve blocks

	Number patients (completed)	Nerves blocked/ frequency	Drugs/groups	Follow-up/efficacy
Migraine				
Double-blind RCT (Malekian et al. 2021)[46] **Migraine**	55 EM	Bilateral GON Once	**Active A (*n* = 10):** 20 mg triamcinolone + saline **Active B (*n* = 16):** 2% lidocaine + saline **Active C (*n* = 13):** 20 mg triamcinolone +2% lidocaine **Placebo group (*n* = 16):** Saline	**Follow-up:** Assessed at 1 week, 2 weeks, and 4 weeks Headache severity ($p < 0.001$) and duration ($p = 0.001$) decreased significantly compared to baseline in all 4 groups Headache frequency per month decreased compared to baseline in Groups B (−5.81, $p = 0.002$) and C (−5.69, $p = 0.019$). No difference between groups in headache severity or duration at any of the time points ($p > 0.05$)
Double-blind RCT (Hokenek et al. 2021)[38] **Migraine**	128 EM	Bilateral GON and bilateral SON injections Once	**Active A (*n* = 30):** Bilateral GON: 1% lidocaine Bilateral SON: saline **Active B (*n* = 28):** Bilateral GON: saline Bilateral SON: 1% lidocaine **Active C (*n* = 43):** Bilateral GON: 1% lidocaine Bilateral SON: 1% lidocaine **Placebo group (*n* = 27):** Bilateral GON: saline Bilateral SON: saline	**Follow-up:** 120 min after injection Compared to placebo (−9.9), Active groups A, B, and C had greater decreased VAS scores (scale 1–100) with respect to baseline (−54.1, −42, −59.3 respectively; all $p < 0.001$). Active A and C had greater change in VAS compared to baseline than Active B, by VAS score ($p = 0.001$, $p < 0.001$ respectively) and 5-point verbal Likert scoring ($p = 0.001$, $p < 0.001$ respectively). No difference between Active A and Active C groups in terms of decreased VAS with respect to baseline ($p > 0.05$)
Single-blind RCT (Ozer et al. 2019)[39] **Migraine**	43 EM 28 CM	Bilateral GON + bilateral SON Weekly × 3 weeks	**Active group (*n* = 43):** 2% lidocaine + saline **Placebo group (*n* = 28):** Saline	**Follow-up:** 2 months after the 3 sets of injections Active group had greater decreased VAS score (1–10 scale), (from 8.3 to 5.5) with respect to baseline than the placebo group (from 8.2 to 7.4); ($p < 0.001$). Decrease in headache frequency with respect to baseline was not significantly different between groups. Response, defined as reduction of at least 50% in the number of headache days, was significantly higher in active group (65.1%) than placebo group (28.6%) ($p = 0.003$)
Double-blind RCT (for GON block portion) (Korucu et al. 2018)[47] **Migraine**	60 (EM vs. CM not specified)	Bilateral or unilateral GON, depending on headache location Once	**Active A (*n* = 20):** GON block with bupivacaine + saline **Active B (*n* = 20):** IV dexketoprofen + metoclopramide **Placebo (*n* = 20):** GON block with saline	**Follow-up**: 5, 15, 30, and 45 min after injection. Median decrease in pain score (scale 1–10) at each time point with respect to baseline was greater in Active A group than in Active B and placebo groups, but no significant difference among groups. At the 30- and 45-min time point, there was a significant difference in the pain score reduction between the Active A group (from 9 at baseline to 3 at 30 min and 1 at 45 min) and the placebo group (from 8 at baseline to 4.5 at 30 min and 3 at 45 min); ($p = 0.012$ for 30 min; $p = 0.016$ for 45 min)

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TABLE 1 (Continued)

	Number patients (completed)	Nerves blocked/ frequency	Drugs/groups	Follow-up/efficacy
Single-blind RCT (Friedman et al. 2018)[106] **Migraine**	28 (EM vs. CM not specified)	Bilateral GON Once	**Active group (*n* = 13):** Bilateral GON block with 6 ml 0.5% bupivacaine **Sham group (*n* = 15):** Bilateral intradermal scalp injection with 1 ml 0.5% bupivacaine Both groups refractory to 10 mg of IV metoclopramide at least 1 h prior to injection	**Follow-up:** 30 min and 48 h after injection Headache freedom at 30 min seen in 4 of 13 patients in active group and 0 patients in sham group ($p = 0.035$). Sustained headache relief (no return to moderate–severe) at 48 h seen in 3 of 13 patients in active group and 0 patients in sham group ($p = 0.087$)
Double-blind RCT (Gul et al. 2017)[29] **Migraine**	44 CM	GON block once per week × 4 weeks	**Active group (*n* = 22):** 0.5% bupivacaine + saline **Placebo group (*n* = 22):** Saline	**Follow-up:** monthly × 3 months Active group had significant decrease in headache days/month compared to baseline at all 3 follow-ups (from 21.1 days pretreatment to 11 days 1st month, 11.9 days 2nd month, 12.5 days 3rd month) ($p < 0.001$ for all). Active group had significant decrease in VAS score (1–10 scale) compared to baseline at all 3 follow-ups (from 9 at pretreatment to 5.7 at 1st month [$p < 0.001$], 6.2 at 2nd month [$p < 0.001$], 6.3 at 3rd month [$p < 0.01$]). Active group had significantly decreased headache days and VAS compared to placebo in 2nd and 3rd months only
Double-blind RCT (Cuadrado et al. 2017)[4] **Migraine**	36 CM	Bilateral GON Once	**Active group (*n* = 18):** 0.5% bupivacaine **Placebo group (*n* = 18):** Saline	**Follow-up:** 1 week after injection Active group had greater reduction of moderate-to-severe headache days compared to placebo group (−2 vs. -0.4; $p = 0.027$). Active group also had greater reduction of any headache days compared to placebo group (−1.5 vs. -0.1; $p = 0.04$)
Double-blind RCT (Palamar et al. 2015)[45] **Migraine**	23 CM	GON (ultrasound-guided) Once	**Active group (*n* = 11):** 0.5% bupivacaine **Placebo group (*n* = 12):** Saline	**Follow-up:** 1 month after injection Active group had significant decrease in monthly average pain intensity (VAS score, 0–10 scale) on injected side (from 3.93 to 1.55) ($p = 0.003$); placebo group did not. No significant difference in monthly average pain intensity on the non-injected side in either group
Double-blind RCT (Dilli et al. 2015)[3] **Migraine**	63 (CM vs. EM not specified)	Bilateral or unilateral GON, depending on headache location Once	**Active group (*n* = 33):** 0.5% bupivacaine + methylprednisolone **Placebo group (*n* = 30):** Saline +0.25 ml 1% lidocaine	**Follow-up:** 28 days after injection 30% of patients in both groups had ≥50% reduction in frequency of moderate or severe headache days compared to baseline. No significant difference in migraine frequency and duration between groups

(Continues)

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TABLE 1 (Continued)

	Number patients (completed)	Nerves blocked/ frequency	Drugs/groups	Follow-up/efficacy
Double-blind RCT followed by unblinded treatment period (Inan et al. 2015)[30] **Migraine**	72 CM	GON Weekly × 4 weeks (double-blind) Monthly for 2 months (unblinded)	*4-week double-blind RCT portion*: **Active group (*n* = 39):** 0.5% bupivacaine + saline **Placebo group (*n* = 33):** Saline *2-month unblinded portion*: **Both groups:** 0.5% bupivacaine	**Follow-up:** Weekly follow-up × 4 weeks, then monthly for 2 more months *4-week double-blind RCT portion*: Active group had decreased number of headache days (from 18.1 at baseline to 8.8 at 4 weeks; $p < 0.001$) compared to placebo group (from 16.9 at baseline to 13.2 at 4 weeks; $p = 0.035$) ($p = 0.004$ between groups). Active group had greater change in VAS score (from 8.4 at baseline to 5.3 at 4 weeks; $p < 0.001$) compared with placebo group (from 8.1 at baseline to 6.7 at 4 weeks; $p = 0.002$); ($p = 0.004$, between groups). *2-month unblinded portion*: Response similar between both groups at 3rd month
Double-blind RCT (Kashipazha et al. 2014)[36] **Migraine**	48 (CM vs. EM not specified)	Bilateral GON Once	**Active group (*n* = 24):** 2% lidocaine + triamcinolone (also, propranolol 20 mg BID) **Placebo (no steroid) group (*n* = 24):** 2% lidocaine + saline (also, propranolol 20 mg BID)	**Follow-up:** 2, 4, and 8 weeks after injection Significant decrease in pain severity (on 11-point scale) in both the active group (from 7.46 at baseline to 3.50 at 2 weeks, 4.29 at 4 weeks, and 5.46 at 8 weeks; $p < 0.001$ for all) and the placebo group (from 7.29 at baseline to 3.5 at 2 weeks, 4.71 at 4 weeks, and 5.29 at 8 weeks; $p < 0.001$ for all). Significant decrease ($p < 0.001$) in pain frequency (unit details unclear) in active group (from 11.71 at baseline to 5.5 at 2 weeks, 6.71 at 4 weeks, and 8.38 at 8 weeks; $p < 0.001$ for all) and placebo group (from 11.36 at baseline to 6.50 at 2 weeks, 7.58 at 4 weeks, and 9.42 at 8 weeks; $p < 0.001$ for all). No significant differences in pain severity or frequency between the two groups
Single-blind randomized comparative study (Ashkenazi et al. 2008)[37] **Migraine**	37 CM	Bilateral GON block + 12 trigger point injections	**Active group (*n* = 19):** 2% lidocaine +0.5% bupivacaine + triamcinolone in each GON and 12 trigger points **Placebo (no steroid) group (*n* = 18):** 2% lidocaine +0.5% bupivacaine + saline in each GON and 12 trigger points	**Follow-up:** 20 min after injection Active group: Mean headache severity was reduced by 3.1 points ($p < 0.01$); mean neck pain severity was reduced by 1.7 points ($p < 0.01$). Placebo group: mean headache severity was reduced by 3.2 points ($p < 0.01$); mean neck pain severity was reduced by 1.5 points ($p < 0.01$). There was no significant difference in response between active and placebo at 20 min. Mean duration of headache freedom was not different between groups (1.0 days for active vs. 2.7 days for placebo; $p = 0.67$)

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TABLE 1 (Continued)

	Number patients (completed)	Nerves blocked/ frequency	Drugs/groups	Follow-up/efficacy
Cluster headache				
Double-blind RCT (Leroux et al. 2011)[55] **Cluster headache**	28 Episodic cluster 15 Chronic cluster	Ipsilateral GON (suboccipital) 3 injections-48–72 h apart	**Active group** ($n = 21$): Cortivazol + saline (also, verapamil 240–720 mg for EC and previous prophylaxis for CC) **Placebo group** ($n = 22$): Saline (also, verapamil 240–720 mg for EC and previous prophylaxis for CC)	**Follow-up:** 2–4 days after last injection; first 15 days after injection All patients had >2 attacks / day prior to injections *On days 2–4 after 3rd injection*: More patients in the active group (20/21) had ≤2 attacks per day compared to the placebo group (12/22); $p = 0.012$ *In first 15 days after injection*: Active group had significantly fewer cluster attacks than placebo (10.6 vs. 30.3 attacks; $p = 0.004$)
Double-blind RCT (Ambrosini 2005)[8] **Cluster headache**	16 Episodic cluster 7 Chronic cluster	Ipsilateral GON (suboccipital) Once	**Active group** ($n = 13$): Mixture of long and rapid-acting betamethasone +2% lidocaine **Placebo group** ($n = 10$): Saline +2% lidocaine	**Follow-up:** 1 and 4 weeks after injection *Week 1*: Significantly more patients in the active group (11 patients) were attack free compared to placebo (0 patients); $p < 0.001$ *Week 4*: 8 patients in active group were still attack-free; 0 patients in placebo group became attack-free; $p = 0.0026$ 5 patients in active group remained attack-free for 4–26 months
Secondary headache				
Double-blind RCT (Naja et al. 2006)[9] **Cervicogenic headache**	47	GON + LON ± facial nerve block depending on location of headache (nerve stimulator guided)	**Active group** ($n = 24$): GON + LON ± facial nerve block Active injectate contained the following per 10 ml: 3 ml 2% lidocaine, 3 ml 2% lidocaine with epinephrine, 2.5 ml 0.5% bupivacaine, 0.5 ml fentanyl (25 μg), and 1 ml clonidine (150 μg) **Placebo group** ($n = 23$): GON + LON ± facial nerve block: saline	**Follow-up:** 2 weeks after injections Frequency of headache was significantly lower in active group (5.50) than placebo group (7.04); $p = 0.026$. Active group had reduced VAS (1–10) and Total Pain Index (TPI) scores by approximately 50% of baseline values ($p < 0.001$)

Abbreviations: BID, twice daily; CM, cluster migraine; EM, episodic migraine; GON, greater occipital nerve; LON, lesser occipital nerve; RCT, randomized controlled trial; VAS, visual analog scale.

those that received saline (−2 vs. −0.4; $p = 0.027$).[4] Palamar et al. followed 32 patients with CM for 1 month after unilateral ultrasound-guided GON injections of 1.5 ml of either 0.5% bupivacaine or saline. Both groups showed improvement in severity through week two, but the treatment group had reduced monthly pain intensity compared to placebo, on the injected side ($p = 0.003$).[45] Ruiz Pinero et al. followed 60 patients with migraine (43 with CM and 17 with episodic migraine [EM]) after blocks of GON, SON, or both GON + SON unilaterally or bilaterally (nerves chosen by tenderness to palpation) and reported at 2 weeks, 38% of their patients had a complete response and 40% reported a partial response (reduced frequency or intensity by 50%).[49] A few cases did report sustained response at 3 months.[49] In an open-label prospective study of 150 patients getting unilateral or bilateral GON blocks suboccipitally for CM, 81 patients (54%) had ≥50% reduction in functionally incapacitating migraine days per month compared to baseline, at 1 month follow-up.[50]

Given the reported benefit from a single set of GON blocks, some trials have investigated potential benefit for repeat injections. Inan et al.[30] looked at weekly GON blocks for 4 weeks and found that patients with CM receiving bupivacaine had greater reduction in headache frequency ($p = 0.004$) and severity ($p = 0.004$) compared to those receiving saline. Gul et al. looked at 44 patients with CM treated with GON blockade weekly (bupivacaine vs. saline) for 4 weeks, with follow up for 3 months.[29] Both bupivacaine and placebo had some improvement in headache frequency and severity the first month, but the bupivacaine group continued reduction in

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the second and third month compared to baseline.[29] Ozer et al. performed a combination of GON + SON blocks weekly for 3 weeks on patients with migraine (43 with EM and 28 with CM) and followed them for 2 months.[39] Patients with EM receiving lidocaine had significantly reduced headache frequency and severity compared to those receiving saline, whereas patients with CM receiving lidocaine noted a reduction in pain severity but no difference in frequency compared to placebo.[39] The results of one open label study suggest that in patients who fail GON block, there may be benefit in repeating a block with additional trigeminal and cervical branches, such as a combination of SON, STN, GON, LON, and ATN.[51]

In addition to helping with migraine-related pain, GON blocks may be helpful for migraine aura. There have been several case reports of patients who received GON blocks during an active migraine aura, including one with prolonged hemiplegic migraine aura and another with brainstem aura who experienced improvement in both the headache and neurologic symptoms within 5–15 min after GON block, with resolution of aura in 1 h.[52,53] Baron et al. report a similar case of migraine with brainstem aura and left>right suboccipital tenderness who noted dramatic improvement of headache, photophobia, phonophobia, nausea, visual changes, imbalance, and speech difficulties within several minutes of a left GON block.[54] Cuadrado et al. treated 22 auras lasting longer than 2 h with bilateral GON blocks and described complete response without recurrence in 11 cases, complete response with recurrence within 24 h in 2 cases, and greater than 50% improvement in 6 cases.[33]

Based on the available literature, recent narrative reviews concluded that GON blocks can be an effective acute and short-term prevention option for patients with migraine.[2,25] GON blockade may be also considered for use as rescue therapy in patients with status migrainosus or to abort prolonged migraine aura.[33,52] Repeat GON blocks may be helpful, but additional studies are needed to prove benefit over single injections and to investigate the ideal frequency of injections.

Cluster headache

Ambrosini et al. performed a small RCT of GON blocks in episodic and chronic cluster headache and found 11 of 13 patients (85%) became attack free within the first week of their block (0.5 ml 2% lidocaine with 2 ml long-acting and rapid-acting betamethasone) while none in the placebo group (0.5 ml 2% lidocaine with 2 ml saline) were pain free ($p < 0.001$); 8 were still attack free for 4 weeks.[8] Leroux et al. gave three rounds of suboccipital corticosteroid (cortivazol 3.75 mg) injections 48–72 h apart to patients with episodic and chronic cluster headache, and found patients with cortivazol had fewer attacks than controls in the first 15 days ($p = 0.004$).[55] Multiple open label studies and series have shown benefit of GON block with anesthetic, corticosteroid, or a combination.[5–7,22,56,57]

Response duration varies between studies and patient populations, with patients with episodic cluster headache tending to have longer responses, possibly because they end their cluster cycle.[5] In a prospective open label study of 83 patients with chronic cluster headache who underwent unilateral greater occipital nerve blockade with 2% lidocaine and methylprednisolone, 35 patients had a complete response (defined as pain freedom for at least 7 days) and 12 patients had a partial response (defined as a 50% or greater reduction in headache frequency or severity for at least 7 days). Median duration of response was 21 days.[7] In a series looking at long-term use of GON blocks for 10 patients with chronic cluster headache, repeat injections of 9 ml of 1% lidocaine with 40 mg triamcinolone ipsilateral to the pain provided complete pain freedom in 9 patients for an average of 10.3 weeks (range 1.5–44 weeks).[56] These studies support the use of suboccipital corticosteroid injections as an option for transitional or rescue therapy for patients with cluster headache.[58]

Secondary headache disorders and neuralgias

Within the secondary headache disorders, GON blockade has been studied most frequently in cervicogenic headache and post-dural puncture headache. Case reports and small series in support of use in post-traumatic headache (headache attributed to traumatic injury to the head), medicine overuse headache, and neuralgias. Most data remain observational, with very few RCTs available.

Cervicogenic headache

Procedures for cervicogenic headache often target the presumed cervicogenic sources of pain (e.g. upper cervical medial branch blocks/ablation) or attempt to interrupt the upper cervical nerve signal (e.g. blockade of the C2-3 dorsal rami).[59,60] While preliminary evidence suggests GON blockade may also be helpful for cervicogenic headache, it may be that patients do better with either proximal GON blocks, recurrent blocks, or the combination of GON blockade with blockade of other cervical terminal branches.

Inan et al. reported that a series of three weekly GON blocks (lidocaine for first block, bupivacaine for second and third blocks) had comparable efficacy to C2/C3 nerve blocks in cervicogenic headache.[61] In contrast, Lauretti et al.[62] reported that a fluoroscopically guided suboccipital unilateral GON block provided significantly longer duration of reduced pain severity compared to a classic GON block (24 weeks vs. 2 weeks; $p < 0.01$). Kissoon et al. looked at a mixed population of cervicogenic headache and occipital neuralgia and found an ultrasound-guided C2-level GON block resulted in greater reduction in intensity at 30 min ($p = 0.007$) and 4 weeks ($p = 0.013$) compared to a distal GON block.[35] Naja et al. randomized 47 patients to nerve stimulator–guided GON blockade of GON/LON with either lidocaine/bupivacaine/fentanyl/clonidine or placebo (saline).[9] If patients described pain radiating into the orbit, they also received a facial nerve block ($n = 16$ in each group). At 2-week follow-up, the patients receiving the anesthetic mixture had reduced

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visual analog scale (VAS) score and reduced analgesic consumption compared to those receiving placebo ($p < 0.001$). Anthony studied the use of combination GON and LON blocks in 180 patients with cervicogenic headache. Using a nerve stimulator to locate the exiting origin of nerves, patients were given 3–4 ml 1% lidocaine followed by 160 mg methylprednisolone into the region of the GON and LON. One hundred sixty-nine (90.6%) patients achieved significant pain relief ranging from 10–77 days (mean 23.5 days).[10]

Post-dural puncture headache

Available evidence suggests GON blockade may provide some amount of headache improvement in select patients with post-dural puncture headache (PDPH), and this procedure might be a minimally invasive alternative for patients with PDPH when conservative management is ineffective, and for patients who either decline or are unable to undergo an epidural blood patch.

Naja et al. randomized PDPH patients to either nerve stimulator guided GON/LON blockade with anesthetic mixture (lidocaine 2%, epinephrine, bupivacaine 0.5%, fentanyl, clonidine) or conservative management (hydration, bed rest, oral analgesics).[63] Sixteen of the 24 (68%) patients randomized to the block group had improvement in their headache after one to two rounds of blocks, with the other patients requiring a third or fourth block. Patients had a lower pain severity in the first 6 days, and were able to leave the hospital earlier ($p < 0.001$) with less sick leave ($p < 0.001$).[63] Of 18 patients with PDPH who received bilateral GON block with dexamethasone and 1% lidocaine, 12 had resolution of their headache without recurrence of headache at their 1-week follow-up, while 6 went on to get an epidural blood patch.[64] Of 21 patients with PDPH who did not derive benefit from conservative management for 48 h, ultrasound-guided bilateral GON blocks were helpful at providing pain relief within 24 h for the 12 patients with moderate (VAS score 4–6) pain. Patients with more severe VAS scores did not have significant improvement.[65]

Occipital neuralgia

While response to a nerve block is part of the International Classification of Headache Disorders 3rd edition diagnostic criteria for occipital neuralgia,[66] there is limited evidence to support the use of occipital nerve blocks for treatment of this condition. Series that describe benefit of GON blockade for occipital neuralgia frequently involve a mixed cohort of patients including cervicogenic headache, migraine, or other craniofacial pain with occipital tenderness and pain.[35,67–72] A few studies suggest that in patients with occipital neuralgia, guided proximal GON blockade may provide additional benefit over the GON block at the nuchal line.[35,73] A retrospective review examined the response of 33 patients with severe refractory occipital neuralgia to computed tomography-guided GON blocks given at the first bend of GON between the inferior obliquus capitis and semispinalis capitis muscles, and found that 32 of 37 GON blocks resulted in >50% pain reduction with a mean relief duration of 9.15 months (range 3–24 months).[74] Of note, case series have described benefit of blockade of specific trigeminal branches in other localized neuralgias, such as supraorbital, supratrochlear, and auriculotemporal nerves.[75–77]

Other secondary headaches

Case series suggest GON blocks may be helpful for post-traumatic headache.[78–80] Case reports also describe benefit of GON block for spontaneous intracranial hypotension,[81] and secondary short-lasting neuralgiform headache with conjunctival injection and tearing syndrome following whiplash.[82] In patients with medication-overuse headache due to triptans, Karadas et al. found three weekly GON blocks improved the number of headache days and severity of headache when combined with abrupt withdrawal of triptans.[83]

LESSONS LEARNED FROM THE LITERATURE

Does the addition of corticosteroids provide additional benefit?

Available studies comparing nerve blocks using a combination of corticosteroid with anesthetic to nerve blocks using anesthetic alone for the treatment of migraine have not shown any significant difference in clinical outcome. Two single blind studies compared anesthetic plus corticosteroid to anesthetic alone and found the addition of corticosteroid provided no additional benefit.[36,37] Chowdhury et al. demonstrated that adding 80 mg methylprednisolone to the first of three monthly GON blocks provided no additional benefit over lidocaine alone.[84] Dilli et al. showed that a GON block with corticosteroid and local anesthetic did not reduce the frequency of migraine days compared to placebo using 0.25 ml of 1% lidocaine.[3] In a randomized double-blind placebo-controlled trial in EM, Malekian et al. compared patients randomized to GON blocks with either triamcinolone 20 mg, 2% lidocaine, triamcinolone 20 mg plus 2% lidocaine, or saline. They found no injection to be superior to placebo at 1-, 2-, or 4-week follow-up. Notably, Malekian et al. stopped recruiting patients into the groups receiving triamcinolone after three patients developed cutaneous atrophy with alopecia over the injection site.[46] Cutaneous atrophy, hypopigmentation, and alopecia are known potential adverse effect of corticosteroids, and may be more common in particulate corticosteroids such as methylprednisolone and triamcinolone.[12,85,86] Triamcinolone has particle sizes ranging from 15–60 μm and when diluted with bupivacaine, may coalesce into large (>100 μm) aggregates, creating clouding of the solution.[87] Nonparticulate corticosteroids, such as dexamethasone, have not been included in the randomized migraine trials, likely in part because the effects of dexamethasone are thought to be more transient, as it is rapidly absorbed.[87] Notably, local corticosteroid injections can have systemic side effects as well, with transient

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hyperglycemia in diabetes,[88] sleep disturbances,[5] oral candidiasis,[5] and iatrogenic Cushing's syndrome with repeated injections.[89] A systematic review assessing the addition of corticosteroid to local anesthetic blocks in chronic non-cancer pain injections showed small benefit with a potential for harm.[90] Based on the available evidence, a 2022 practice guideline addressing percutaneous interventional strategies for migraine prevention provides a weak recommendation against the use of corticosteroids.[2]

In contrast to migraine, studies suggest benefit of corticosteroid in GON blockade for cluster headache, including one RCT that showed superior efficacy of combining anesthetic with corticosteroid versus saline[8] and two studies with positive results using corticosteroids alone.[55,57] Though the evidence seems more supportive of the use of corticosteroids in patients with cluster headache, these patients are not immune to adverse events. In one series of cluster headache patients, 9.8% of patients developed cutaneous atrophy and alopecia after their GON block.[57] Rozen reported a patient who developed unilateral avascular necrosis of the hip after 30 high-volume suboccipital blocks for chronic cluster headache.[56]

Does the presence of medication overuse affect the block?

Tobin and Flitman conducted a chart review of patients at their clinic and found that patients overusing pain medication were three times more likely to report no benefit to occipital nerve blocks than non-overusers, and this failure rate was greater in patients with migraine than in patients with occipital neuralgia.[67] In contrast, Afridi et al., Ruiz Pinero et al., Dilli et al., and Weibelt et al. did not find medication overuse to influence the likelihood of treatment response.[3,12,49,50] It is not clear why the retrospective study by Tobin and Flitman found dramatically different results, but it may be related to a difference in either the nerve block technique, the refractoriness of their patient population, or the types of medicines being overused (e.g., barbiturates, opioids, triptans). The available data suggests that patients overusing medications may still have a response to nerve blocks.

When to use ultrasound guidance?

Anatomic variation in location and trajectory of foramen, nerves, and nearby vascular structures have led some to question the utility and safety of landmark-based injections.[45,68,91] Apparent success of landmark-based nerve blocks despite this anatomic variation may be explained by the significant spread of injectate from the injection sites, as demonstrated in one study using methylene dye in cadaveric scalp injections.[92] The complication rates for landmark-based injections has been generally low in patients with normal anatomy. A recent narrative review raised concern about the increased cost of ultrasound guidance[1] and future studies are needed to assess the overall impact of health-care cost for headache nerve blocks both with and without the use of ultrasound. The greater precision with ultrasound guidance may be helpful when performing blocks with smaller volumes,[68] as well as in patients with altered anatomy, difficult-to-palpate occipital artery pulses, or implanted hardware.[35,93]

A distinct advantage of ultrasound guidance is the potential for proximal blocking of the occipital nerve at the level of C2. Several studies have reported outcomes with ultrasound-guided proximal injections in migraine.[35,40,70,94] Flamer et al. compared outcomes between proximal and distal ultrasound-guided GON nerve blocks in a CM cohort and found that while both techniques had similar short-term efficacy, there was a greater likelihood of sustained pain relief at 1 and 3 months with the more proximal block.[95] Karaoglan and Inan[40] looked at patients with EM or CM in their retrospective review and found that 1 month of weekly ultrasound-guided GON blocks performed distally or proximally at C2 both provided significant improvement in migraine frequency and severity at the first and third months, compared to baseline ($p < 0.001$; frequency of injections confirmed by personal communication, M. Karaoglan, March 19, 2022). In both treatment groups, headache severity, frequency, and analgesic use were improved compared to baseline, but the blocks at the C2 level were associated with a greater reduction in severe monthly migraine attacks compared to distal blocks. Adverse events were higher in the C2-level injections ($p = 0.006$), but all adverse events were transient, with a subset of patients in the C2 group noted to have transient dizziness or cerebellar symptoms such as ataxia (25%), while a number of patients treated with distal blocks had transient worsening of headache (12.9%).[40]

In a study looking at a mixed population of occipital neuralgia and cervicogenic headache, ultrasound-guided proximal GON blockade had a significantly higher rate of complete dermatomal anesthesia than the distal landmark-based block (76.9% vs. 30.8%, $p < 0.05$).[73] In another study examining patients with occipital neuralgia or cervicogenic headache, proximal GON blockade at the C2 level with ultrasound guidance resulted in a greater reduction in intensity at 30 min ($p = 0.007$) and 4 weeks ($p = 0.013$) than the more traditional landmark-based distal block with sham ultrasound.[35]

Adverse effects of nerve blocks?

Adverse events are reported to occur in about 5%–31% of patients receiving GON blocks.[25,50,96] Most adverse events are transient and mild, such as post-injection dizziness, local site swelling, worsening headache, prolonged head numbness, elevated blood pressure, and pain.[50,96,97] Presyncope–syncope has been listed as an adverse event in several studies,[12,31,49,50] and some recommend additional caution in the elderly and patients prone to syncope.[97] Higher concentration anesthetic (lidocaine 5%) had a trend of higher adverse events in one retrospective series.[97]

While the risk of local anesthetic systemic toxicity (LAST) is considered low (estimated incidence of 0.27 episodes per 1000 nerve blocks[98]), it is worth mentioning. LAST may present as central nervous system (CNS) toxicity including seizures, as well as cardiac toxicity, including arrhythmia, cardiac conduction defects, and cardiac arrest.

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The risk of LAST may be reduced with restriction of drug dosage, with injections of less than 5 ml per aliquot, and with sterile aspiration.[98] Ropivacaine has been demonstrated to have decreased CNS and cardiac toxicity compared to bupivacaine;[99] however, the literature to support the use of ropivacaine in occipital nerve blocks is limited.

Though serious adverse events are uncommon, a few case reports underscore the importance of proper injection technique. Five patients have been reported to develop an acute cerebellar syndrome after GON blocks.[40,100,101] One patient with a Chiari malformation and possible excessive anterolateral neck flexion during positioning was reported to have needle penetration and injection of local anesthetic and non-steroidal anti-inflammatory drugs into the cerebellar tonsils.[100] The other four were receiving injections at the C2 level and described unilateral dysmetria, dysdiadochokinesia, and ataxic gait for about 4–6 h after their procedure.[40,101] Skull base abnormalities, such as a previous posterior fossa craniotomy, have been associated with post-injection loss of consciousness[102] and coma,[103] and are considered a contraindication for landmark-based occipital nerve blocks.[23] Finally, one patient was reported to have developed a focal abscess and occipital osteomyelitis in the location of two recently performed GON blocks containing both anesthetic and corticosteroid.[104] Subcutaneous abscesses are a known risk for peripheral nerve blocks,[105] emphasizing the need for aseptic technique.

CONCLUSION

There is growing evidence for the use of GON blockade for the acute treatment and short-term prevention of migraine and this should be considered a viable treatment option. There is some evidence that GON blockade provides benefit in cluster headache and may also have a beneficial role in other headache disorders such as cervicogenic headache, postdural puncture headache, and occipital neuralgia. Corticosteroids should be used with caution and there is weak evidence against the use of corticosteroids with occipital nerve blocks in migraine. However, corticosteroids are generally used for the treatment of cluster headache. Patients should be counseled appropriately about corticosteroid related side effects, especially when particulate corticosteroids are used.

The duration of therapeutic effect from nerve blocks varies, but may be days to weeks, with a potential for partial response for months. Guided proximal GON blocks at the level of C2 may provide a longer benefit than distal injections. The use of ultrasound guidance and consideration of additional blockade of the LON or trigeminal branches depend on patient factors such as the headache indication, prior treatment response, and the presence of altered anatomy. There is a need for additional trials to investigate the role of GON blocks for other primary and secondary headache disorders, most specifically for occipital neuralgia.

AUTHOR CONTRIBUTIONS

Study concept and design: Jennifer I. Stern, Chia-Chun Chiang, Narayan R. Kissoon, Carrie E. Robertson. *Drafting of the manuscript*: Jennifer I. Stern, Carrie E. Robertson. *Revising it for intellectual content*: Chia-Chun Chiang, Narayan R. Kissoon, Carrie E. Robertson. *Final approval of the completed manuscript*: Jennifer I. Stern, Chia-Chun Chiang, Narayan R. Kissoon, Carrie E. Robertson.

CONFLICTS OF INTEREST

Dr. Stern is a shareholder in Emmyon, Inc., which has no conflict of interest with any topic in this article. Dr. Chiang has no conflicts of interest. Dr. Kissoon serves on the editorial board for *Pain Medicine*. Dr. Kissoon reports grant from Nevro Corporation and royalties from UpToDate that are outside the submitted work. Dr. Kissoon is on the scientific advisory board for Bright Minds Biosciences, LTD. Dr. Robertson has served on the advisory board for Lundbeck, Biohaven, Amgen, Eli Lilly, Impel pharmaceuticals; has received research support from Lundbeck and Teva; and has received honoraria as author for UpToDate and Continuum.

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