Debunking myths in headache diagnosis for the pain practitioner

Hsiangkuo Yuan ,[1] Samantha Strutner,[2] Clara Yuh,[3] Suwarna Anand,[4] Gaurav Chauhan,[5] Anand Prem,[4] Shalini Shah,[6] Nathaniel M Schuster[7]

[1]Jefferson Headache Center, Department of Neurology, Thomas Jefferson University, Philadelphia, Pennsylvania, USA
[2]School of Medicine, University of California, Irvine, Irvine, California, USA
[3]Department of Physical Medicine & Rehabilitation, University of California, Irvine, Irvine, California, USA
[4]Department of Anesthesiology, University of Mississippi University Hospital, Jackson, Mississippi, USA
[5]Department of Anesthesiology, University of Pittsburgh, Pittsburgh, Pennsylvania, USA
[6]Department of Anesthesiology & Perioperative Care, University of California, Irvine, Orange, California, USA
[7]Headache and Pain Neurologist, Center for Pain Medicine, Department of Anesthesiology, University of California, San Diego, La Jolla, California, USA

Correspondence to
Dr Nathaniel M Schuster, Center for Pain Medicine, Department of Anesthesiology, University of California, San Diego, La Jolla, CA 92037, USA;
nmschuster@health.ucsd.edu

Received 26 June 2023
Accepted 18 September 2023
Published Online First 10 October 2023

To cite: Yuan H, Strutner S, Yuh C, *et al*. *Reg Anesth Pain Med* 2024;**49**:545–549.

ABSTRACT

With headache being one of the most common chief complaints, it is essential for pain practitioners to interpret and differentiate a variety of headache characteristics to accurately diagnose and treat specific headache disorders. Certain misconceptions often lead to misdiagnosis. This article presents and discusses six myths about several common headache disorders (migraine, tension-type headache, cluster headache, cervicogenic headache, sinus headache, and occipital neuralgia) often encountered in clinical practice. The discussion is based primarily on the International Classification of Headache Disorders, 3rd edition and the latest studies. Recognizing and understanding the intricacies behind key headache diagnoses will help providers devise appropriate plans to better care for their patients.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Pain doctors frequently encounter headaches in practice. The International Classification of Headache Disorders, 3rd edition offers a guide to identify different headache disorders. Misconceptions are common with several headache disorders.

WHAT THIS STUDY ADDS

⇒ Several common headache myths were discussed and debunked to inform pain doctors' approach to accurately diagnosing headache disorders.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ A better understanding of common headache disorders improves clinical diagnosis and management.

INTRODUCTION

Headache is a common presenting complaint to all healthcare providers. It ranks as the third leading cause of years lived with disability worldwide.[1 2] Based on the 3rd edition of the International Classification of Headache Disorder (ICHD-3), headache is classified into primary headaches (eg, migraine, tension-type headache, trigeminal autonomic cephalalgias), secondary headaches (eg, headache attributed to trauma or injury to the head and/or neck, etc), and neuropathies and facial pains and other headaches. However, most pain medicine providers have received only a few hours of headache education during their training, thus limiting their awareness of consensus guidelines.[3] As such, there are commonly held headache myths that result in misdiagnoses. The American Society of Regional Anesthesia and Pain Medicine's Headache and Facial Pain Special Interest Group convened a multidisciplinary group of neurologists, anesthesiologists, and physiatrists to identify and dispel a few of these pervasive myths based on our clinical experience.

Myth #1: if a headache is bilateral and/or squeezing, then it is not a migraine

Tension-type headache is the most common primary headache disorder,[1] with a lifetime prevalence of 30%–78%. It is usually bilateral, nonthrobbing, not associated with migrainous features, and not typically disabling.[4] While migraine is generally unilateral and throbbing, it can be bilateral (40%) and can be described as pressure-like (75%).[5] Furthermore, migraine can be 'imploding' in quality, described as bilateral tightness, stiffness, and vice-like pain with pressure squeezing in. In a prospective cross-sectional survey of 198 female patients with migraine, 31.6% of subjects described their migraine as imploding, and 10.2% described them as both imploding and exploding.[6] Furthermore, although triptans do not work for pure tension-type headache, triptans are effective in patients with migraine with attacks that phenotypically resemble tension-type headache.[7]

A common misunderstanding among patients and clinicians alike (reinforced by simplistic cartoons perpetuated on the internet) is that migraine and tension-type headache are differentiated by whether the headache is unilateral versus bilateral. However, this is an oversimplification. Based on the ICHD-3 criteria,[4] migraine and tension-type headache are differentiated based on the attack duration, headache characteristics, and associated symptoms (boxes 1 and 2). Patients with tension-type headache do not present with typical migrainous features; they cannot have nausea or vomiting and can have no more than one of photophobia or phonophobia.

The clinical distinction between tension-type headache and migraine is crucial for therapeutic reasons. There are many evidence-based treatments for migraine that are not indicated for tension-type headache, including triptans, calcitonin gene-related peptide antagonists, and chemodenervation with onabotulinumtoxinA for chronic migraine. If

Box 1 Diagnostic criteria for migraine without aura

A. At least five attacks fulfilling criteria B–D.
B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated).
C. Headache has at least two of the following four characteristics:
   1. Unilateral location.
   2. Pulsating quality.
   3. Moderate or severe pain intensity.
   4. Aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs).
D. During a headache at least one of the following:
   1. Nausea and/or vomiting.
   2. Photophobia and phonophobia.
E. Not better accounted for by another ICHD-3 diagnosis.

the migraine diagnosis is missed, patients will not receive these evidence-based treatments.[8] Furthermore, if these relatively expensive treatments are used when they are not indicated, it may impact the resources available for those who need them.

Conclusion: Migraine is distinguished from tension-type headache by the headache characteristics and the presence of migrainous features. Contrary to popular belief, migraine is bilateral in 40% of cases and 'imploding' in over 30% of cases.

Myth #2: men are more likely to get cluster headache than migraine

Clinicians often remember from their training that cluster headache (box 3) has a male predominance, while migraine has a female predominance. However, this does not mean that men are more likely to develop cluster headache than migraine, a misunderstanding that likely contributes to men being underdiagnosed and undertreated for migraine. In our clinical experience, this also results in encountering many men with migraine who have been misdiagnosed by prior physicians with cluster headache.

Replicating many prior studies, a recent epidemiology study by Schor *et al*,[9] found that men were more likely than women to have cluster headache between 10 and 50 years of age. In a meta-analysis of population-based studies, Fischera *et al* showed a lifetime cluster headache prevalence of 0.12% with an overall sex ratio of 4.3 (men to women).[10] However, cluster headache is about 100 times rarer than migraine, with migraine affecting an estimated 12% of the population and over 1 billion people worldwide in 2019.[1] Likely due to the influence of sex hormones, the

Box 2 Diagnostic criteria for tension-type headache

A. At least 10 episodes of headache fulfilling criteria B–D.
B. Lasting from 30 min to 7 days.
C. At least two of the following four characteristics:
   1. Bilateral location.
   2. Pressing or tightening (non-pulsating) quality.
   3. Mild or moderate intensity.
   4. Not aggravated by routine physical activity such as walking or climbing stairs.
D. Both of the following:
   1. No nausea or vomiting.
   2. No more than one of photophobia or phonophobia.
E. Not better accounted for by another ICHD-3 diagnosis.

Box 3 Diagnostic criteria for cluster headache

A. At least five attacks fulfilling criteria B–D.
B. Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15–180 min (when untreated).
C. Either or both of the following:
   1. At least one of the following symptoms or signs, ipsilateral to the headache:
      – Conjunctival injection and/or lacrimation.
      – Nasal congestion and/or rhinorrhoea.
      – Eyelid edema.
      – Forehead and facial sweating.
      – Miosis and/or ptosis.
   2. A sense of restlessness or agitation.
D. Occurring with a frequency between one every other day and eight per day.
E. Not better accounted for by another ICHD-3 diagnosis.

prevalence of migraine in women is higher than in men (19.0% vs 11.2%).[1] Putting this all together, due to the high prevalence of migraine and the much lower prevalence of cluster headache, existing data provide irrefutable evidence that men are more likely to suffer from migraine than cluster headache.[10 11]

Conclusion: Migraine affects over 1 billion people worldwide annually. Therefore, despite men being three times more likely to be affected by cluster headache than women, migraine is roughly 100 times more common among men than is cluster headache.

Myth #3: migraine with neck pain is equivalent to cervicogenic headache

It can be useful to divide patients with head and neck pain into three categories.

The first category is the population of patients with migraine or another primary headache disorder who only have neck pain before or during a headache attack and have physical exams and imaging that are not suggestive of cervical pathology. In these patients, neck pain is likely a symptom of the primary headache disorder due to central sensitization rather than a cervical pain generator.

The second category of patients is the population with no prior history of migraine or other primary headache disorder with cervical pathology on exam and/or imaging that is likely causing their headache and neck pain; these are the patients with pure cervicogenic headache. The likelihood of cervicogenic headache increases with a history of plausible inciting causes such as whiplash injuries as well as the absence of migrainous features. The age distribution of these patients likely skews older than the more well-defined age distribution of migraine.

The third category is the population of patients who straddle these first two cleanly defined groups, where cervical pain generators such as painful C2/3 facet joints might be causing a new-onset of migraine-type headache or worsening a pre-existing primary headache disorder, such as causing episodic migraine to become more frequent and turn into chronic migraine. In our experience, these patients often benefit from a combination of treatment with both migraine medications and percutaneous treatments for cervicogenic headache.

Based on the ICHD-3 criteria, neck pain is not part of the criteria for migraine, nor does it distinguish migraine from cervicogenic headache (box 4) or other headache disorders. If a pain practitioner encounters a patient with headache and neck pain with unrevealing imaging studies, without inciting cervical

Box 4 Diagnostic criteria for cervicogenic headache

A. Any headache fulfilling criterion C.
B. Clinical and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck, known to be able to cause headache.
C. Evidence of causation demonstrated by at least two of the following:
   1. Headache has developed in temporal relation to the onset of the cervical disorder or appearance of the lesion.
   2. Headache has significantly improved or resolved in parallel with improvement in or resolution of the cervical disorder or lesion.
   3. Cervical range of motion is reduced and headache is made significantly worse by provocative maneuvers.
   4. Headache is abolished following diagnostic blockade of a cervical structure or its nerve supply.
D. Not better accounted for by another ICHD-3 diagnosis.

cause, and/or who does not respond to cervical interventions, a migraine history should be taken (including assessing for presence of photophobia, phonophobia and nausea with headaches and whether the neck pain is only present at the beginning of or during a headache attack). If the patient meets ICHD-3 criteria for migraine, the clinician should consider the possibility that the patient's neck pain is a migraine symptom.

Pain doctors might not realize that in many patients with migraine, neck pain can occur as a migraine symptom, even in the absence of cervical pathology. Epidemiological and cross-sectional studies have found a high rate of cervical symptoms among patients with migraine, but these studies are unable to answer 'chicken and egg' questions of causation. Among patients with migraine, population-based studies have reported a neck pain prevalence in upwards of 75% of adult patients,[12] and provide evidence that neck pain is more frequently associated with migraine than is nausea. Al-Khazali *et al*, in a recent meta-analysis,[13] showed that 77% of patients with migraine in clinic-based studies reported neck pain, compared with only 23% of non-headache controls. The odds ratio (OR) of neck pain in patients with migraine was 11.5 (95% CI 5.8 to 22.4) compared with non-headache controls; the OR in chronic migraine was 2.04 (95% CI 1.3 to 3.3) compared with episodic migraine.[13] Anarte-Lazo *et al*, in another meta-analysis,[14] found a greater range of rotation reduction (by cervical flexion-rotation test) in cervicogenic headache than in migraine. Migraine also displayed a reduced cervical range of motion versus asymptomatic individuals.[14] However, the clinical validity of the cervical flexion-rotation test has been questioned.[15]

Pain practitioners should recognize that cervical symptoms are highly prevalent among patients with migraine, and that the pain practitioner's task is to use history, exam, imaging, clinical reasoning, and appropriate use of diagnostic injections and other procedures to tease apart which of the aforementioned categories to place a given patient into. Neck pain and migraine both involve activation of the trigeminocervical complex (TCC). While conflicting data exist, neck pain may result from migraine-related central sensitization, cervical pain generators, and local inflammation-related peripheral sensitization.[16 17] There is likely a bidirectional relationship between migraine and neck pain. Given the high prevalence of migraine (over 1 billion people worldwide), its heritability, and its typical onset in the first four decades of life, one of the common causes of neck pain is often likely migraine, especially in patients without cervical pathology. Conversely, neck pain can also reflect the underlying upper cervical pathology. True cervicogenic headache, with prevalence ranging from 0.4% to 4%, often benefits from cervical spine interventions, such as medial branch blocks and radiofrequency ablations.[18 19] In short, pain practitioners need to know that episodic neck pain may be a norm among people with migraine, and is likely more often an accompanying symptom of migraine than its cause.

Conclusion: Neck pain and reduced range of motion may present in both migraine and cervicogenic headache. Recognizing that such clinical nuance exists is critical in reaching the correct diagnosis and deciding subsequent treatment.

Myth #4: migraine is just a headache and is not serious

While migraine may not be deadly, its seriousness is due to the disability that it causes and the effects it has on the lives of patients and their families. Migraine is the second leading cause of years lived with disability for ages 15–49 worldwide.[1] Migraine's disability is beyond headache alone. People with migraine experience numerous non-headache symptoms, including nausea, vomiting, photophobia, and phonophobia. Some experience aura before or during an attack, characterized by reversible neurological symptoms, including visual changes, sensory, language, brainstem, or motor symptoms. They also often experience various prodromal (or premonitory) and post-dromal symptoms before and after the headache that contribute to migraine disability. These symptoms can impact the ability to carry out daily activities leading to reduced productivity and decreased quality of life. Underdiagnosis, social stigma, and lack of appropriate care further exacerbate the overall impact.[20 21]

Migraine has negative impacts not only on patients but also on their family members, activities, and relationships. The burden of migraine on the patient's family was greater among those with chronic migraine than among people with episodic migraine. Migraine has also been linked to an increased risk of depression, anxiety, stroke, and cardiovascular disease. Migraine with aura conveys an increased risk of ischemic stroke, hemorrhagic stroke, and cardiovascular disease.[22] While the correlation between the frequency of migraine attacks and the risk of stroke exists, there are no data indicating that migraine prophylaxis decreases the risk of a migrainous infarct.

Conclusion: Migraine is a debilitating disease with serious sequela for patients and their families. It is crucial for physicians and the public alike to understand that migraine is a leading cause of disability worldwide so that patients with migraine seek medical treatment and providers deliver the appropriate diagnosis and treatments. It is also important for policymakers to recognize that migraine is a disabling disease that is treatable and deserving of resources.

Myth #5: sinus headaches are caused by allergies

In an observational study, Schreiber *et al* found that 88% of patients with a 'sinus headache' history fulfill the International Headache Society migraine or migrainous criteria.[23] 'Sinus headache', which may feel like an infection in the sinuses, can often be migraine with nasal symptoms (eg, rhinorrhea). According to the American Academy of Otolaryngology—Head and Neck Surgery guidelines, rhinosinusitis is defined as symptomatic inflammation of the paranasal sinuses and nasal cavity that can be classified by duration as acute (<4 weeks) or chronic (>12 weeks).[24] Sinus inflammation, which may be due to allergy or infection, can cause pressure, pain, and discomfort in the face.

They often report sinus pressure, sinus pain, and nasal congestion. However, nasal obstruction or facial pain without purulent nasal drainage is not consistent with rhinosinusitis. In another observational study, Kari *et al* showed that 82% of patients with 'sinus headache' with negative findings on nasal endoscopy or sinus computer tomography had significant head pain reduction with triptan use.[25] The ICHD-3 defines diagnostic criteria for headache attributed to rhinosinusitis: any headache phenotype is allowed, but there must be clinical, nasal endoscopic, or imaging evidence of paranasal sinus infection/inflammation, as well as evidence of causation.[4] In rare instances, isolated sphenoid sinusitis can present with atypical and noncharacteristic headache, which is most commonly in the vertex but can be anywhere in the craniofacial region and may be associated with cranial nerve involvement. While the presentation of headaches secondary to sinus disease may overlap with primary headache disorders, it is important to evaluate for the possibility of migraine.

However, it is important not to oversimplify all sinus headache as migraine. Positive migraine histories apparently do not obviate the need for a thorough otolaryngologic evaluation, including nasal endoscopy or sinus computer tomography, especially in patients with persistent sinus pain. In the case where headache is associated with mucosal contact points, nasal surgery may be needed.

Conclusion: Rather than caused by allergy or rhinitis, nearly 90% of patients with 'sinus headache' meet migraine criteria.

Myth #6: response to occipital nerve block is specific for occipital neuralgia

The response to occipital nerve block (ONB) is required but insufficient to diagnose occipital neuralgia. Per ICHD-3, occipital neuralgia is defined by headache presentation and exam findings, along with the response to ONB (box 5). Many pain providers may conflate occipital headache with occipital neuralgia.[26] Occipital headache can be a symptom of many primary and secondary headache disorders, and the effect of an ONB is not limited to occipital headache. For example, for those with occipital headache who respond to ONB but have migrainous symptoms, the most likely diagnosis is migraine. That said, having a migraine diagnosis does not negate the possibility of concurrent occipital nerve compression or inflammation. The convergence of sensory input from both cervical and trigeminal fibers in the TCC provides the rationale for the use of ONBs in the treatment of various headache disorders, not just occipital neuralgia.[27] Typically, ONB leads to increased pressure pain thresholds in the trigeminal distribution, inhibiting central sensitization in the TCC.[28]

The majority of evidence for ONBs is for use in migraine. In a narrative review of peripheral nerve blocks for headache management, 11/12 randomized controlled trials (RCTs) showed ONBs reduced migraine intensity, frequency, and duration compared with placebo, with no difference with or without steroid use.[29] Two RCTs show the benefit of ONBs with steroids in cluster headache and another one in cervicogenic headache. In contrast, the evidence is limited to observational studies in occipital neuralgia, as well as in status migrainosus, prolonged aura, and post-dural puncture headache.

Conclusion: ONB has evidence of efficacy for treating migraine, CH, and cervicogenic headache, and as such, response to ONB is not diagnostic of occipital neuralgia. According to the ICHD-3, response to ONB is necessary but insufficient to diagnose occipital neuralgia.

Box 5 Diagnostic criteria for occipital neuralgia

A. Unilateral or bilateral pain in the distribution(s) of the greater, lesser and/or third occipital nerves and fulfilling criteria B–D.
B. Pain has at least two of the following three characteristics:
   1. Recurring in paroxysmal attacks lasting from a few seconds to minutes.
   2. Severe in intensity.
   3. Shooting, stabbing or sharp in quality.
C. Pain is associated with both of the following:
   1. Dysaesthesia and/or allodynia apparent during innocuous stimulation of the scalp and/or hair.
   2. Either or both of the following:
      – Tenderness over the affected nerve branches.
      – Trigger points at the emergence of the greater occipital nerve or in the distribution of C2.
D. Pain is eased temporarily by local anesthetic block of the affected nerve(s).
E. Not better accounted for by another ICHD-3 diagnosis.

CONCLUSION

These myths may lead pain practitioners and other healthcare providers astray in diagnosing patients presenting with different headache disorders. Recognizing these heuristics as myths that have been debunked by evidence will hopefully help providers correctly diagnose patients and improve the care they provide to patients with various headache disorders.

X Shalini Shah @ShaliniShahMD and Nathaniel M Schuster @NatSchusterMD

Contributors HY and SS contributed to conception and design. HY, SS, CY, SA, GC, AP, SS and NMS contributed to acquisition of data. HY, SS, CY, SA, GC, AP, SS and NMS contributed to drafting the article. HY, SS, CY, SA, GC, AP, SS and NMS contributed to revision for intellectual content. NMS contributed to final approval of completed article.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests Within the past 24 months, HY has received funding from NIH (R44NS115460); institutional support for serving as an investigator from Teva, Abbvie, Trillen, Thermaquil; consultant fees from Salvia, Pfizer, Cerenovus; and royalties from Cambridge University Press and MedLink. NS has received compensation from Eli Lilly & Co., Averitas, ShiraTronics, Syneos, Schedule 1 Therapeutics, and Vectura Fertin and research funding from Migraine Research Foundation, Novaremed, UCSD Academic Senate, UCSD Department of Anesthesiology RAG, and NIH CTSA Grant UL1TR000100. SS received consulting honorarium from Allergan.

Patient consent for publication Not applicable.

Ethics approval Not applicable.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement No data are available.

ORCID iD

Hsiangkuo Yuan http://orcid.org/0000-0002-4536-9974

REFERENCES

1 Gbd compare, Available: https://vizhub.healthdata.org/gbd-compare [Accessed 26 Mar 2023].
2 GBD 2017 Disease and Injury Incidence and Prevalence Collaborators. Disease injury incidence prevalence collaborators. global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990-2017: A systematic analysis for the global burden of disease study 2017. *Lancet* 2018;392:1789–858.
3 Minen MT, Yang J, Ashina S, *et al*. Survey of pain medicine specialists' familiarity with migraine management. *Pain Med* 2021;22:3030–40.
4 Headache classification committee of the International headache society (IHS) the International classification of headache disorders, 3RD edition. *Cephalalgia* 2018;38:1–211.

5 Kelman L. Pain characteristics of the acute migraine attack. *Headache* 2006;46:942–53.
6 Files JA, Schwedt TJ, Mayer AP, *et al*. Imploding and exploding migraine headaches: comparison of methods to diagnose pain Directionality. *Headache* 2014;54:1010–8.
7 Lipton RB, Stewart WF, Cady R, *et al*. Wolfe award. Sumatriptan for the range of headaches in migraine sufferers: results of the spectrum study. *Headache* 2000;40:783–91.
8 Cohen F, Yuan H, Silberstein SD. Calcitonin gene-related peptide (Cgrp)-Targeted Monoclonal antibodies and antagonists in migraine: Current evidence and rationale. *BioDrugs* 2022;36:341–58.
9 Schor LI, Pearson SM, Shapiro RE, *et al*. Cluster headache epidemiology including pediatric onset, sex, and Ichd criteria: results from the International cluster headache questionnaire. *Headache* 2021;61:1511–20.
10 Fischera M, Marziniak M, Gralow I, *et al*. The incidence and prevalence of cluster headache: A meta-analysis of population-based studies. *Cephalalgia* 2008;28:614–8.
11 Allais G, Chiarle G, Sinigaglia S, *et al*. Gender-related differences in migraine. *Neurol Sci* 2020;41(Suppl 2):429–36.
12 Calhoun AH, Ford S, Millen C, *et al*. The prevalence of neck pain in migraine. *Headache* 2010;50:1273–7.
13 Al-Khazali HM, Younis S, Al-Sayegh Z, *et al*. Prevalence of neck pain in migraine: A systematic review and meta-analysis. *Cephalalgia* 2022;42:663–73.
14 Anarte-Lazo E, Carvalho GF, Schwarz A, *et al*. Differentiating migraine, Cervicogenic headache and asymptomatic individuals based on physical examination findings: A systematic review and meta-analysis. *BMC Musculoskelet Disord* 2021;22:755.
15 Paquin J-P, Dumas J-P, Gérard T, *et al*. A perspective on the use of the Cervical flexion rotation test in the physical therapy management of Cervicogenic headaches. *Arch Physiother* 2022;12:26.
16 Al-Khazali HM, Krøll LS, Ashina H, *et al*. Neck pain and headache: pathophysiology, treatments and future directions. *Musculoskelet Sci Pract* 2023;66:102804.
17 Andersen S, Petersen MW, Svendsen AS, *et al*. Pressure pain thresholds assessed over Temporalis, masseter, and Frontalis muscles in healthy individuals, patients with tension-type headache, and those with migraine—a systematic review. *PAIN* 2015;156:1409–23.
18 Bogduk N, Govind J. Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment. *Lancet Neurol* 2009;8:959–68.
19 Hurley RW, Adams MCB, Barad M, *et al*. Consensus practice guidelines on interventions for Cervical spine (facet) joint pain from a Multispecialty International working group. *Pain Med* 2021;22:2443–524.
20 Buse DC, Armand CE, Charleston L 4th, *et al*. Barriers to care in episodic and chronic migraine: results from the chronic migraine epidemiology and outcomes study. *Headache* 2021;61:628–41.
21 Parikh SK, Kempner J, Young WB. Stigma and migraine: developing effective interventions. *Curr Pain Headache Rep* 2021;25:75.
22 Øie LR, Kurth T, Gulati S, *et al*. Migraine and risk of stroke. *J Neurol Neurosurg Psychiatry* 2020;91:593–604.
23 Schreiber CP, Hutchinson S, Webster CJ, *et al*. "Prevalence of migraine in patients with a history of self-reported or physician-diagnosed "sinus" headache". *Arch Intern Med* 2004;164:1769–72.
24 Rosenfeld RM, Piccirillo JF, Chandrasekhar SS, *et al*. Clinical practice guideline (update): adult sinusitis. *Otolaryngol Head Neck Surg* 2015;152(2 Suppl):S1–39.
25 Kari E, DelGaudio JM. Treatment of sinus headache as migraine: the diagnostic utility of Triptans. *Laryngoscope* 2008;118:2235–9.
26 Love SM, Hopkins BD, Migdal CW, *et al*. Occipital headache evaluation and rates of migraine assessment, diagnosis, and treatment in patients receiving greater occipital nerve blocks in an academic pain clinic. *Pain Med* 2022;23:1851–7.
27 Ashkenazi A, Levin M. Greater occipital nerve block for migraine and other headaches: is it useful? *Curr Pain Headache Rep* 2007;11:231–5.
28 Cuadrado ML, Aledo-Serrano Á, Navarro P, *et al*. Short-term effects of greater occipital nerve blocks in chronic migraine: A double-blind, randomised, placebo-controlled clinical trial. *Cephalalgia* 2017;37:864–72.
29 Stern JI, Chiang C-C, Kissoon NR, *et al*. Narrative review of peripheral nerve blocks for the management of headache. *Headache* 2022;62:1077–92.