

Original Article

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A New Technique Employing Direct Tactile Pressure on the Common Carotid Artery to Relieve Acute Episode Attack of Migraine Headache: A Single-Arm Interventional Study

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Abstract

Introduction: Migraine, is a common neurological disorder, and its pathology and acute treatment has not been determined so far. In the contemporary literature, there is no remedy that can abort an acute episode of migraine.

Objective: We aimed to evaluate the effect of transient pressure on the middle part of common carotid artery for terminating an acute attack of migraine headache.

Methods: It is an interventional study without a control limb, performed on patients within age range of 18-45 years. Patients with established migraine headache based on International Classification of Headache Disorders (ICHD) guidelines, who had no atheromatous plaque in their common carotid arteries were included. Pain intensity was evaluated by Universal Pain Assessment Tools (UPAT). In safe position, applying vital signs monitoring and ipsilateral of headache, a gentle pressure was applied on the common carotid artery, lateral to the cricothyroid membrane and medial border of the Sterno-Cleido-Mastoid (SCM) muscle, by using both index and middle fingers, till the headache was relieved, following which the pressure was maintained for a period of 15 seconds. Then the middle finger was maintained at its position and the index finger slid caudally with the same pressure as far a distance of four centimeters and the pressure withdrawn slowly. After 2 minutes, patients were asked to report any change in headache which was recorded.

Results: Totally, 215 patients entered this study. The mean of pain score before and after using the technique regarding to UPAT, was 6.28 ± 1.34 and 0.4 ± 0.64 , respectively; Also, the pain decreases equal 5.88 score was significant, special by according sex ($p < 0.001$). No side effect was seen.

Conclusions: It seems that pressure on the common carotid artery and extending the pressure caudally, helped a rapid, safe and significant reduction in pain score for patients with acute attack of migraine headache.

Key words: Carotid Artery, Common; Headache; Home Remedy; Migraine Disorders; Pain Management

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INTRODUCTION

Migraine, is a common neurological disorder, and its pathology and acute treatment has not been determined so far. According to migraine vascular theory, cranial arteries dilation in the scalp, dura, and pia activates trigeminal pain fibers and trigger the subsequent headache. Based on this theory, vasoconstrictors such as ergotamine and triptans were developed as abortive therapies for migraine. But during chemically induced migraine headache, vascular changes observed by Imaging studies and the controversy over the vascular theory with conflicting results has been left unresolved (1). The pathogenesis of migraine pain had undergone vigorous debate. Patients with migraine frequently

referred to outpatient clinics, are usually on oral or parenteral medication such as triptans for acute treatment of episodic attacks. These drugs specifically seem to counteract the dilation of some arteries and arterioles, including external carotid artery branches, and they are classified as vasoconstrictors (2-5). Also they are treated with botulinum toxoids, acupuncture, traditional medicine and other methods (6-8). Newly publications refer to a method and apparatus, by selectively applying a predetermined electrical stimulus of the vagus nerve by implantable devices, which can alleviate migraine symptoms, but it is not only costly, but invasive as well and the results

not decisively fruitful (9, 10). So far, in the contemporary literature, there is no remedy that can abort an acute episode of migraine. Compression of the extracranial arteries in some migraine patients during the headache transiently decreased the symptoms (11). However, amelioration of pain with this procedure only occurs during the period of compression itself, but in this study, we evaluated a new technique by compression effect on common carotid artery in relieving migraine attack headache, to become considerably less in intensity.

Methods

Study design and setting

This was a single-arm interventional study performed from September 2019 until March 2020 in Tehran, Iran. Written informed consent from the patients and approval form the ethical committee of Tehran University of Medical Sciences were obtained (code: IR.TUMS.IKHC.REC.1397.182). The authors accepted Declaration of Helsinki principles. The protocol of the study has been registered in www.irct.ir and a specific code has been assign to it (Registry code: IRCT20150116020676N4).

Participants and Intervention

The patients were evaluated by consensus sampling. Patients aged 18-45 years who had an established migraine headache and referred to our clinic through an advertisement announced by our pain outpatient departments were recruited patients with established migraine based on International Classification of Headache Disorders (ICHD) guidelines (12), in whom color doppler sonography of the common carotid arteries depicted no narrowing of the common carotid arteries or any atheromatous plaque were included. Patients with histories of syncopal attacks, sensitivity of carotid sinus, cardiac dysrhythmia, history of any neurologic disease, patients with another non migraine headache symptoms and basilar migraine were excluded.

The patients continued their medications as prescribed by their physicians. All patients had Doppler echo of the carotid arteries to rule out any narrowing of the carotid arteries or any atheromatous plaque in the arteries. The pain score at the time of admission was not considered owing to the fact that the intervention was extremely short, in the event of failure in the reduction of pain or else an increase in the intensity of pain, the intervention was aborted and patient allowed to take his medications as before. After having obtained an informed consent, and having



Figure 1: Pressure applied on the anterior border of SCM, at the crico-thyroid cartilage level.



Figure 2: Index finger slid 4 centimeters caudally while the middle finger maintaining the same pressure as before.

evaluated the pain intensity according to the Universal Pain Assessment Tools (UPAT), the patient was seated on a chair and conventional monitors (pulse oximetry, non-invasive blood pressure, electrocardiography) applied, and resuscitative trolley made available. Then depending on the site of the headache, the landmark was identified by palpating the cricothyroid membrane by the finger of person performing the intervention (Figure 1). Then the finger was slid laterally till it reached the medial border of the Sterno- Cleido -Mastoid muscle (SCM), a point coinciding with the carotid sinus (13). Then using both index and middle fingers, a gentle pressure was applied on the middle part of the common carotid artery. After having applied the pressure, the patient was asked whether the pulsatile headache got terminated or not. If the

patient's headache was not relieved, the index and the middle fingers, were moved so as to impinge the common carotid artery between them and the pressure applied again. If the headache had been terminated, the tactile finger pressure was maintained for a period of 15 seconds. Then the middle finger was maintained at its position and the index finger slid caudally with the same pressure as far a distance of four centimeters (Figure 2) and pressure withdrawn slowly. After a lapse of 2 minutes, the patient was asked to report any change in headache which was recorded. Then the patient was asked to lie down in a bed and vital signs monitored for a period of 1-hour following which the patient was discharged.

Data analysis

All the data were analyzed using SPSS VER 18. Frequency and percentage are used for demonstrating qualitative data and mean and SD

for quantitative data. Paired sample T test was used for evaluation of pain mean difference before and after intervention. A P-value less than 0.05 was considered as significant.

RESULTS

Totally, 215 patients entered this study. Demographic factors and clinical signs of patients with migraine headache history by sex are being depicted in (Table 1). The mean age of patients was 32.3 ± 6.3 and 155 patients (72.1%) were female and 60 patients (27.9%) were male. Of all participants, 94.9% of patients had photophobia, 87.0% had nausea, and 82.8% had phonophobia. All of the patients had one of the clinical signs (such as nausea, vomiting, photophobia, phonophobia and aura) and 56.7% had three positive signs simultaneously (Figure 3). The distribution of clinical signs did not have any significant difference

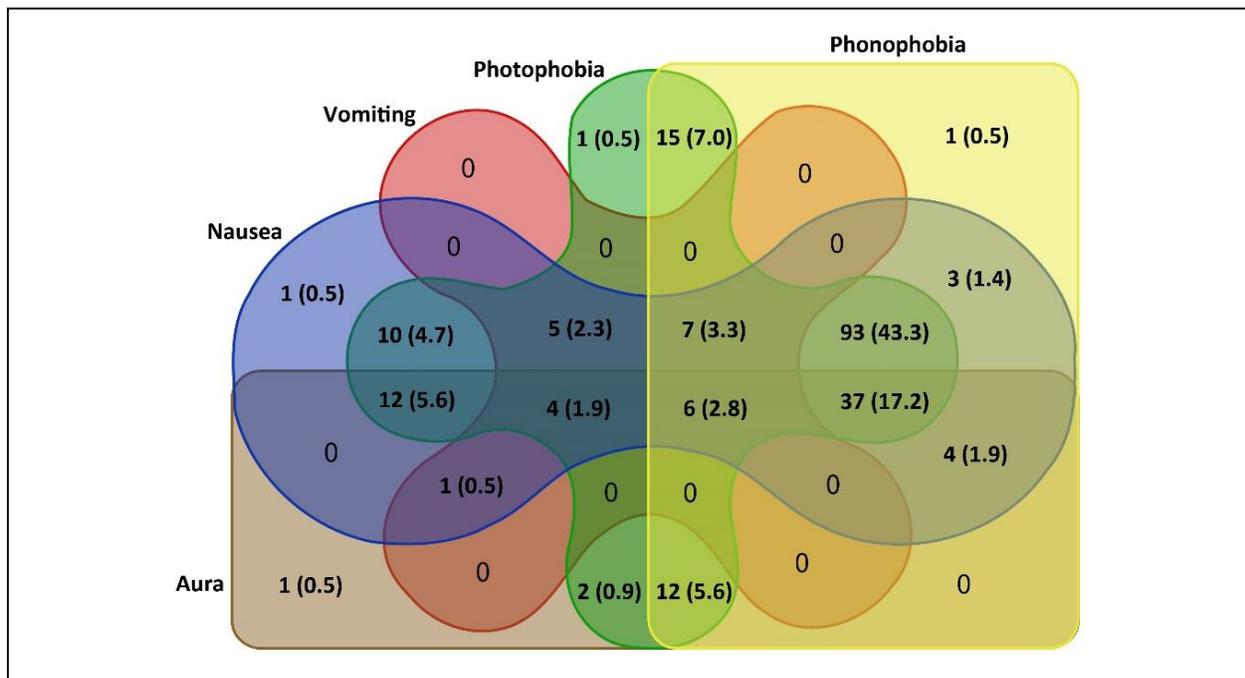
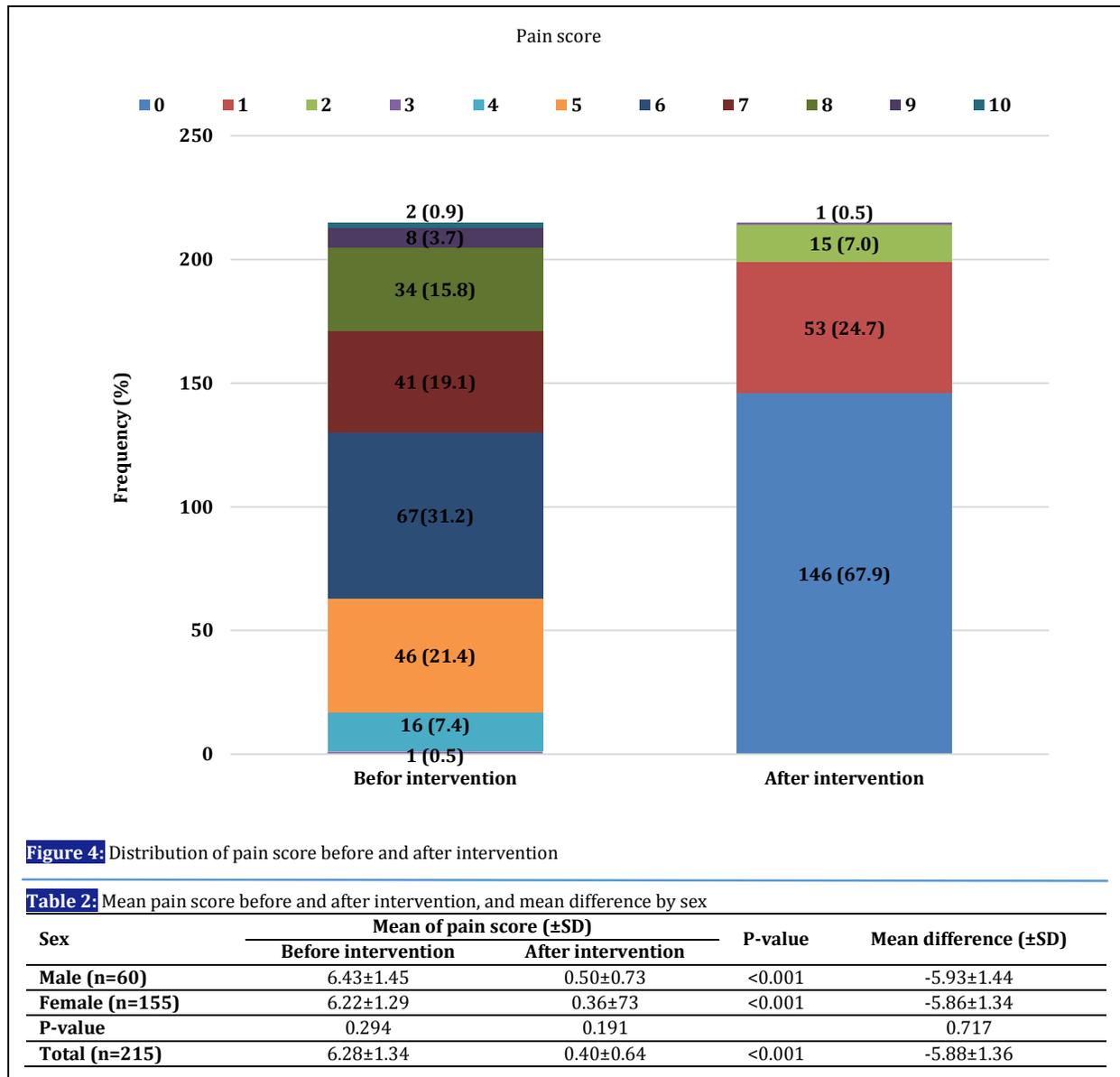


Figure 3: The venn diagram of clinical signs' frequency (%) of migraine patients

Table 1: Demographic factors and clinical signs of patients with migraine headache history by sex

Variable	Total patients (n=215)	Sex		P-value
		Male (n=60)	Female (n=155)	
Age (mean±SD), year	32.3±6.3	31.9±5.9	32.4±6.5	0.563
Duration of Migraine (mean±SD), year	11.9±4.7	11.9±4.9	11.8±4.6	0.895
Aura, (%)	79 (36.7)	23 (38.3)	56 (36.1)	0.764
Photophobia, (%)	204 (94.9)	57 (95.0)	147 (94.8)	1.0
Phono phobia, (%)	178 (82.8)	48 (80.0)	130 (83.9)	0.500
Nausea, (%)	187 (87.0)	53 (88.3)	134 (86.5)	0.713
Vomiting, (%)	186 (86.5)	9 (15.0)	20 (12.9)	0.686
Triptan, (%)	26 (12.1)	8 (13.3)	18 (11.6)	0.729
Boutax, (%)	13 (6.0)	5 (8.3)	8 (5.2)	0.359



according to gender distribution ($p > 0.05$). All patients received nonsteroidal anti-inflammatory drugs (NSAIDs) treatment, 12.1% had history of receiving triptans and 13 patients (6.0%) had received Boutax injections. The mean of migraine history was 11.9 ± 4.7 year which was not statically significant among patients of both sexes ($p = 0.895$). The pain score before intervention was ≥ 3 according to UPAT score, and more than 85% of patients had scores between 5 to 8. Pain score after treatment in more than 90% of patients was ≤ 3 (between 0-10) (Figure 4). Pain score mean before treatment was 6.28 ± 1.34 and 0.40 ± 0.64 at and after the technique, respectively. The mean of pain decreased to a score of almost 5.88 ($p < 0.001$) (Table 2) which was statically significant. This,

decrease was significant according sex ($p < 0.001$) (Table 2). The general linear model (GLM) analysis showed that age ($p = 0.712$), duration of migraine ($p = 0.162$) and sex ($p = 0.155$), did not have effects covariate on outcome. There was also no significant relation reported between pain difference score with age ($r = -0.08$, $p = 0.267$), and duration of migraine ($r = -0.09$, $p = 0.181$).

DISCUSSION

In our study, we evaluated 215 patients who were being treated for migraine headache and fulfilled the ICDH criteria for migraine headache. The results of this maneuver showed a significant reduction in the UPAT pain score of migraine headache in less than one minute.

Headache is considered as a major health issue with a prevalence of active headache disorders in the general population and also in both sexes, its prevalence peaks between 25 to 55 years of age and has significant direct and indirect costs and in the workplace, it can be increased in burnout due to active headaches disorders (14-17). Many medications and methods have been advocated, but as the pathogenesis of migraine is not clear and debatable, treatment options remain uncertain. Other studies have shown, that prior to the onset of headache, vasoconstriction and increased resistance to blood flow occurs. They concluded that the extra cranial arteries are a major source of pain in migraine (18, 19). These studies also observed tenderness of the dilated arteries, which suggested that some migraine pain may emanate from the arterial walls (20, 21). Some studies provided a rationale for the use of drugs that relieve migraine pain by reversing vasodilation in the extra cranial vascular system, in particular, ergotamine tartrate (22-24). Management of acute migraine by pharmacological approach has not changed much since 2012. The mainstays of acute migraine therapy presently are non-steroidal anti-inflammatory drugs (NSAIDs), triptans, antiemetics, and etc. (25, 26). Different type of non-oral and oral preparations of triptans and NSAIDs such as (nasal sprays, intravenous/intramuscular injection, transdermal patches, and oral powder formulations), might improve the action efficacy, by more rapid onset than that of existing medications (27). Acute treatment response is most effective when these medications are administered, within 15 minutes of the onset of pain (28). Since these drugs take a few minutes to show their effect, which indeed might be a long period for patients with headache. Also these drugs have side effects such as nausea, vomiting, tachycardia and palpitation, uncontrolled hypertension, hemiplegic migraine and vascular disease (29-35). Other studies revealed that the role of the extra cranial terminal branches of the external carotid artery, provided convincing evidence of their involvement in the progression of migraine pain and it led to the propose that the pain of migraine originated in the distended extra cerebral arteries (32). These studies showed that the change in pulsations of branches of the external carotid arteries (occipital and superficial temporal) had a direct relation with the intensity of migraine. Factors such as ergotamine tartrate can decrease the amplitude of pulsations and intensity of headache and with this effect amplitude of pulsations of the temporal and occipital arteries

decreased to almost 50%. When the amplitude of pulsations rapidly decreased, dimension of pain intensity rapidly changed, too and this effect, was seen as vice versa. In a study, compression of extracranial artery during migraine headache reduced or eliminated the pain. Some researches had noted that the terminal branches of carotid artery, might be a source of migraine pain (11). Another technique by vagus nerve stimulation (VNS) was successful in regarding the pain (36). In another study at first treated attack, VNS could affect pain in four out of 19 (21%) (37). Similar reduction of pain using VNS. was reported in another study (38). A pressure on the carotid artery for the relief of headache has been of some help, but its etiology is somehow not understood. Some hypothesis exists for such a relief:

- Hypothalamus, an important autonomic center in the brain could be involved in migraine headache as the symptoms of the headache tally with the alteration in autonomic nervous system. Activation of neurons in the locus coeruleus (LC) is believed to mediate many of the effects of vagus nerve stimulation (VNS) in the central nervous system (CNS). LC is the largest noradrenergic nucleus in the brain; Despite LC, some other neural activity was seen due to VNS (39). Structures related to both sympathetic and parasympathetic processing, such as the LC, also exhibited better hypothalamic connectivity. The LC could be involved in both normal and abnormal pain modulation during migraine because of its supposed involvement in the inhibition of nociceptive reflexes and the firing mode of thalamic and prefrontal neurons in response to noxious stimuli raise the possibility that during migraine some involvements in normal or abnormal pain modulation may happen. Factors that have been mentioned in favor of this maneuver could be an excitatory or inhibitory effect on the messages which have an effect on the neuronal discharge over the LC, which has been mentioned as a cause of migraine (40, 41).
- Some studies have mentioned dilation of the extra cranial vessels as a cause of migraine. A pressure on the carotid artery causes a reduction of flow in these arteries and thus terminates the episode of migraine. But it is not known as to why the symptoms do not return after withdrawal of the pressure.
- Moreover, the presence of the stellate ganglion and its role in sympathetic mediated pain has not been fully elucidated.
- The internal and external jugular vessels lying in

the vicinity could also be playing some role in the genesis of migraine headache.

Our maneuver, the tactile pressure, could be of value in patients who do not use any medications or else pregnant patients who pose some limitations in taking drugs. Likewise, it could also be helpful for pilots, drivers or people who do exhaustive works and had to remain fully alert during skillful assignments. This technique can be easily accomplished by the patient himself or his/her companion.

Limitations

A possible limitation of this study was that a control group was not included. Having debated over this issue, it was decided that a control loop could not be included because it was literally impossible as no intervention as a control group could have given us instantaneous reward as is evident in our intervention group thus defeating the very purpose of including a control group.

REFERENCES

1. Levy D, Burstein R. The vascular theory of migraine: leave it or love it? *Ann Neurol*. 2011;69(4):600-1.
2. Tfelt-Hansen PC, Koehler PJ. History of the use of ergotamine and dihydroergotamine in migraine from 1906 and onward. *Cephalalgia*. 2008;28(8):877-86.
3. Silberstein SD, Hargreaves RJ. The History and Pharmacology of Ergotamine and Dihydroergotamine. In: *Drug Treatment of Migraine and Other Headaches 2000* (Vol. 17, pp. 52-65). Karger Publishers.
4. Tfelt-Hansen P, Saxena PR, Dahlöf C, Pascual J, Lainez M, Henry P, et al. Ergotamine in the acute treatment of migraine: a review and European consensus. *Brain*. 2000;123(1):9-18.
5. Diamond S, Bigal ME, Silberstein S, Loder E, Reed M, Lipton RB. Patterns of Diagnosis and Acute and Preventive Treatment for Migraine in the United States: Results from the American Migraine Prevalence and Prevention Study. *Headache*. 2007;47(3):355-63.
6. Binder WJ, Brin MF, Blitzer A, Schoenrock LD, Pogoda JM. Botulinum toxin type A (BOTOX) for treatment of migraine headaches: an open-label study. *Otolaryngol Head Neck Surg*. 2000;123(6):669-76.
7. Diener HC, Kronfeld K, Boewing G, Lungenhausen M, Maier C, Molsberger A, et al. Migraine Study Group. Efficacy of acupuncture for the prophylaxis of migraine: a multicentre randomised controlled clinical trial. *Lancet Neurol*. 2006;5(4):310-6.
8. Pfaffenrath V, Diener HC, Fischer M, Friede M, Henneicke-von Zepelin HH. The efficacy and safety of Tanacetum parthenium (feverfew) in migraine prophylaxis—a double-blind, multicentre, randomized placebo-controlled dose-response study. *Cephalalgia*. 2002;22(7):523-32.
9. Kufe TM, Pintea B, Muhammad S, Zaremba S, Roeske S, Simon BJ, et al. Cervical non-invasive vagus nerve stimulation (nVNS) for preventive and acute treatment of episodic and chronic migraine and migraine-associated sleep disturbance: preliminary findings from a prospective observational cohort study. *J Headache Pain*. 2015;16(1):101.
10. Grazi L, Egeo G, Calhoun AH, McClure CK, Liebler E, Barbanti P. Non-invasive Vagus Nerve Stimulation (nVNS) as mini-prophylaxis for menstrual/menstrually related migraine: an open-label study. *J Headache Pain*. 2016;17(1):91.
11. Shevel E. The extracranial vascular theory of migraine—a great story confirmed by the facts. *Headache*. 2011;51(3):409-17.

CONCLUSIONS

Direct Tactile pressure on the common carotid artery and extending the pressure caudally helped in relieving the migraine headache but we can only make a cautious conclusion in this regard as a control loop was lacking.

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AUTHORS' CONTRIBUTION

All the authors met the standards of authorship based on the recommendations of the International Committee of Medical Journal Editors.

CONFLICT OF INTEREST

None declared.

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12. Olesen J. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, Abstracts. *Cephalalgia*. 2018;38(1):1-211.
13. Pasquier M, Clair M, Pruvot E, Hugli O and Carron P-N. Carotid Sinus Massage. *N Engl J Med*. 2017;377(15):e21.
14. Stovner LJ, Hagen K, Jensen R, Katsarava Z, Lipton RB, Scher AI, et al. The global burden of headache: a documentation of headache prevalence and disability worldwide. *Cephalalgia*. 2007;27(3):193-210.
15. Lipton RB, Stewart WF, Diamond S, Diamond ML, Reed M. Prevalence and burden of migraine in the United States: data from the American Migraine Study II. *J Headache Pain*. 2001;41(7):646-57.
16. Edmeads J, Mackell JA. The economic impact of migraine: an analysis of direct and indirect costs. *Headache*. 2002;42(6):501-9.
17. Kernick D. An introduction to the basic principles of health economics for those involved in the development and delivery of headache care. *Cephalalgia*. 2005;25(9):709-14.
18. Bigal ME, Kurth T, Santanello N, Buse D, Golden W, Robbins M, et al. Migraine and cardiovascular disease: a population-based study. *Neurology*. 2010;74(8):628-35.
19. De Hoon JN, Willigers JM, Troost J, Struijker-Boudier HA, Van Bortel LM. Cranial and peripheral interictal vascular changes in migraine patients. *Cephalalgia*. 2003;23(2):96-104.
20. Rocca MA, Ceccarelli A, Falini A, Colombo B, Tortorella P, Bernasconi L, et al. Brain gray matter changes in migraine patients with T2-visible lesions: a 3-T MRI study. *Stroke*. 2006;37(7):1765-70.
21. Spierings EL. Mechanism of migraine and action of antimigraine medications. *Med Clin*. 2001;85(4):943-58.
22. Bigal ME, Tepper SJ. Ergotamine and dihydroergotamine: a review. *Curr Pain Headache Rep*. 2003;7(1):55-62.
23. Amin FM, Asghar MS, Hougaard A, Hansen AE, Larsen VA, de Koning PJ, et al. Magnetic resonance angiography of intracranial and extracranial arteries in patients with spontaneous migraine without aura: a cross-sectional study. *Lancet Neurol*. 2013;12(5):454-61.
24. Burstein R, Blake P, Schain A, Perry C. Extracranial origin of headache. *Curr Opin Neurol*. 2017;30(3):263-71.
25. Marmura MJ, Silberstein SD and Schwedt TJ. The acute treatment of migraine in adults: The American Headache Society evidence assessment of migraine pharmacotherapies. *J Headache Pain* 2015;55(1):3-20.
26. Worthington I, Pringsheim T, Gawel MJ, Gladstone J, Cooper P, Dilli E, et al. Canadian Headache Society Guideline: acute drug therapy for migraine headache. *Can J Neurol Sci*. 2013;40(S3):S1-3.
27. Diener H-C, Charles A, Goadsby PJ, Holle D. New therapeutic approaches for the prevention and treatment of migraine. *Lancet Neurol*. 2015;14(10):1010-22.
28. Sabovchik AY, Oros M. Migraine and tension cephalalgia. *Int J Neurol Neurother*. 2018;5(99):61-7.
29. Ferrari MD, Roon KI, Lipton RB, Goadsby PJ. Oral triptans (serotonin 5-HT_{1B/1D} agonists) in acute migraine treatment: a meta-analysis of 53 trials. *Lancet*. 2001;358(9294):1668-75.
30. Gallagher R, Kunkel R. Migraine patient concerns affecting compliance: results from the NHF survey. *Headache*. 2003;43:36-43.
31. Jamieson DG. The safety of triptans in the treatment of patients with migraine. *Am J Med Sci*. 2002;112(2):135-40.
32. Shevel E. The role of the external carotid vasculature in migraine. *Migraine Disorders Research Trends* New York: Nova Science Publishers. 2007:165-82.
33. Drummond PD. Motion sickness and migraine: optokinetic stimulation increases scalp tenderness, pain sensitivity in the fingers and photophobia. *Cephalalgia*. 2002;22(2):117-24.
34. Hmaidan Y, Cianchetti C. Effectiveness of a prolonged compression of scalp arteries on migraine attacks. *J Neurol*. 2006;253(6):811-2.
35. Iversen HK. Human migraine models. *Cephalalgia*. 2001;21(7):781-5.

36. Schröer FJ, inventor; Schroer Frederikus Johannes, assignee. Pressure application device and method for ameliorating migraine headache. United States patent US 6,638,295. 2003 Oct 28.
37. Goadsby P, Grosberg B, Mauskop A, Cady R, Simmons K. Effect of noninvasive vagus nerve stimulation on acute migraine: an open-label pilot study. *Cephalalgia*. 2014;34(12):986-93.
38. Hord ED, Evans MS, Mueed S, Adamolekun B, Naritoku DK. The effect of vagus nerve stimulation on migraines. *J Pain*. 2003;4(9):530-4.
39. Hulsey DR, Riley JR, Loerwald KW, Rennaker RL, Kilgard MP, Hays SA. Parametric characterization of neural activity in the locus coeruleus in response to vagus nerve stimulation. *Exp Neurol*. 2017;289:21-30.
40. Pertovaara A, Almeida A. Descending inhibitory systems. In *Handbook of clinical neurology* 2006 Jan 1 (Vol. 81, pp. 179-192). Elsevier.
41. Moulton EA, Becerra L, Johnson A, Burstein R, Borsook D. Altered hypothalamic functional connectivity with autonomic circuits and the locus coeruleus in migraine. *PloSone*. 2014;9(4):e95508.