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Pain is more widespread and referred to trigeminal areas in occipital neuralgia

A dor é mais difusa e referida para áreas do trigêmeo na neuralgia occipital

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RESUMO

Objetivo. Avaliar dor local e referida na neuralgia occipital. **Método**. Avaliação de prontuários de 32, 16 e 102 pacientes com neuralgia occipital, enxaqueca sem aura e enxaqueca tensional. Critérios para distúrbios craniomandibulares, bruxismo, dores de cabeça, questionários e exame clinico. **Resultados**. As áreas mais frequentes de dor local e referida na neuralgia occipital foram as regiões temporal, frontal, occipital, sub-occipital, retro-orbitária, cervical e vertex (78,1%). Áreas mais frequentes de dor local/referida na cefaleia tensional foram as regiões temporal e frontal bilateral em 65 pacientes (63,7%) e as regiões cervical, vertex, sub-occipital e parietal (21,6%). Áreas comuns de dor local e referida na cefaleia sem aura foramnas regiões temporal anterior direita, parietal e occipital, temporal anterior esquerda, parietal, frontal e cervical (62,5%). A mediana de zonas anatômicas com dor foram para neuralgia occipital 3,5; enxaqueca tensional 2,0; enxaqueca sem aura 2,0 e controles 0,5 (Kruskal-Wallis p<0,0001). **Conclusão**. A dor na neuralgia occipital foi mais difusa e mais locais anatômicos foram relatados. A dor na enxaqueca sem aura foi menos difusa que na neuralgia occipital e mais difusa que na enxaqueca tensional a qual foi mais localizada, bilateral nas regiões temporal e frontal.

Unitermos. Dor, Neuralgia, Cefaleia Comum, Cefaleia Tensional

Citação. Molina OF, Santos ZC, Simião BR, Torres SMP, Marçal RL, Penoni VB. A dor é mais difusa e referida para áreas do trigêmeo na neuralgia occipital.

ABSTRACT

Objetctive. Evaluate local and referred pain in occipital neuralgia. **Method**. Charts review of 32 occipital neuralgia, 16 migraine without aura and 102 tension-type headache individuals, respectively. Criteria for craniomandibular disorders, bruxism, headaches. Questionnaires to assess pain sites and descriptors for headaches. **Results**. Common areas of local/referred pain in occipital neuralgia were the temporal, frontal, occipital, sub-occipital, retro-orbital, cervical and vertex (78.1% cases). Common areas of referred pain in tension-type headache were located in the bilateral temporal and frontal areas (65=63.7%). Other areas of referred pain in tension-type headache were the cervical, vertex, sub-occipital and parietal areas (21.6%). Common areas of local and referred pain in migraine (62.5%) were located in the right anterior temporal area, right anterior temporal, parietal and occipital, left anterior temporal and parietal area, frontal and cervical areas. The median of painful anatomic zones were occipital neuralgia 3.5, tension-type headache 2.0, migraine 2.0; and Controls 0.5 (Kruskal-Wallis statistics with post-test p<0.0001). **Conclusions**. Pain in occipital neuralgia was reported in a more widespread anatomic area and in more anatomic zones as compared to migraine and tension-type headache. The latter was reported usually bilaterally in the frontal and temporal areas.

Keywords. Pain, Neuralgia, Migraine without Aura, Tension-Type Headache

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INTRODUCTION

Headache is one of the most common reasons for patients to visit physicians´ offices, specifically in primary care[1]. Occipital neuralgia (ON) is a rare cause of severe cranial headache, may be refractory to medical treatment and most patients are usually treated with analgesics and sedation[2]. The terms ON are used to describe the irritation of the greater occipital nerve (GON) and the signs and symptoms associated with it. ON is a very difficult headache to diagnose due to the variety of signs it presents with[3], and is also much more common than other cranial neuralgias, and occurs in the distribution of the second or third cervical dorsal roots. ON pain is described together with paresthesia in the occipital nerve distribution[4,5], as a paroxystic, lancinating pain located to the occipital skin area supplied by the GON, sometimes accompanied by dull, continuous pain[6].

The causes of ON include degenerative changes, congenital abnormalities, trauma, compression of the nerve and other factors[5]. Patients with a history of rheumatoid arthritis, degenerative changes and GON compression are more likely to develop this headache type[3]. There can be hypertonic cervical musculature, congenital abnormalities within the muscles or within the occipital nerve itself and a larger than normal occipital nerve can contribute to ON symptoms[5]. ON may occur secondary to acceleration-deceleration injuries, and to local or systemic diseases[4]. Clinically, ON may be caused by structural and/or functional disorders of the third occipital nerve and by cervical spine changes including spondylosis, arthritis of the upper cervical facets joints and thickening of the ligaments in the area corresponding to C1-C4[7].

Terms such as burning, aching and throbbing, have been used to describe ON, but dizziness, nausea and photophobia are also common symptoms[5]. Headache in ON is almost always described as unilateral or bilateral, starting at the posterior base of the occiput, and occasionally radiating behind the eye socket. Pain can also radiate to the lateral side of the scalp, may also be described as sudden, intermittent, paroxystic, and can be reproduced locally by compressing manually the GON or the adjacent region known as the "pain generator area"[5]. Pressing, shooting and sometimes lancinating are common descriptions in ON patients[8].

Anatomic and functional alterations in the C2 and C3 nerves which innervate the back of the head may cause ON. These nerves pierce the nuchal fascia and the base of the skull and thus, are prone to trauma from flexion, extension, injury and entrapment by spasm of the trapezius muscle[9]. One ON mechanism is related to increased muscle activity in the cervical region, or entrapment of the C2 root or dorsal root ganglion by paravertebral ligamentous structures[10]. GON compression and/or prolonged irritation by a nerve-vessel contact, may also be a mechanism in ON and some investigators[2,10] recommend GON decompression to alleviate intense pain.

ON may be associated with a number of symptoms in anatomic areas innervated by the trigeminal nerve (TN) and the responsible mechanism may be the convergence of sensory information from the occipital and TN in the upper cervical cord[11]. Referred pain in ON is believed to arise from sensory connections between TN main sensory nucleus and the substantia gelatinosa of the cervical spinal cord, which is continuous with the inferior end of TN spinal tract[12].

ON pain is described in the cervico-occipital junction and may be referred and/or radiate to unilateral or bilateral structures including the occiput, parietal, temporal, frontal and retro-orbital regions[8]. Regarding radiation pattern, ON is similar to migraine as pain is usually unilateral, but radiates to the frontal, orbital and periorbital regions[13]. ON pain usually originates in the sub-occipital area, with radiation to the skull, vertex and temporal regions and is described as peri or retro-orbital in some patients[12]. Even though ON signs and symptoms have been well described in the current literature[14], there is a paucity of data regarding the anatomic areas where ON pain is projected in craniomandibular disorder (CMDs) patients. Moreover, it has been difficult to establish which signs and symptoms should be treated by different specialists, and differentiate ON from other common headaches is a challenge for both the novice and the experienced specialist. Because it has been reported that there is a connection between the GON and the TN sub-nucleus caudalis, and that both major nerves innervate different anatomic areas, it is expected that ON produce local and referred pain in a widespread anatomic

area as compared to migraine without aura (MIG) and tension-type headache (TTH), thus, the goals of this investigation are the following: 1) Describe the patterns of local and referred pain in ON, MIG and TTH; 2) Test the hypothesis that pain in ON is more widespread and thus, involves more anatomic areas; 3) Describe the mechanisms of referred pain in ON.

METHOD

Sample

From a large sample of individuals presenting with CMDs, and bruxing behavior (BB) referred consecutively over a period of 10 years to UNIRG, University Center, Division of Orofacial Pain and Occlusion, for diagnosis and treatment, the charts of all those presenting ON (n=32), MIG (N=16), and TTH (n=102), were reviewed retrospectively so as to gather information about local and referred pain, by an expert in the field (OFM). Criteria to exclude CMDs + BB presenting with ON, TTH, or MIG from participating in this investigation were the presence of psychiatric disorders, severe neurologic disease (for instance, epilepsy, Parkinson disease), currently taking combinations of medications at the same time, for instance, muscle relaxants + anti-anxiety + antidepressant + analgesics which could masquerade the diagnosis of headache, and intellectual difficulties to respond properly to questionnaires. The investigation was approved by the Ethical Committee of The Dental School (003-2013).

Procedure

Patients were classified as presenting CMDs if they demonstrated at least three of the following signs, symptoms or behaviors[15]: A complaint of pain in the masticatory muscles and/or temporomandibular joints (TMJs), difficulties to perform normal jaw movements, tenderness to palpation of joint and muscles, joint noises, seeking active treatment for their complaint and presence of headaches usually of CMD origin. CMD individuals were classified as bruxers if they presented at least three signs or symptoms of a list published previously elsewhere[16]. The comprehensive protocol used in the current study contains a number of descriptors to describe and classify different pain types including those evaluated in the current study. Criteria to consider the presence of ON in CMD and BB individuals were the presence of symptoms described previously[5,13]. Briefly: Unilateral or bilateral, paroxystic, lancinating and severe pain described as burning, throbbing, shooting/jabbing, electric-shock like, continuous and/or intermittent, presenting with a tender, pain generator area in the sub-occipital region, pain reproduced with pressure over the pain generator spot, occurring in the occipital/sub-occipital area and usually radiating to the vertex, frontal, orbital and peri-orbital regions.

Inclusion criteria for MIG: Pain described as unilateral, severe, constant, and always throbbing or pulsating, pain increased by physical effort and a pulsating characteristic reported more frequently during severe migraine episodes[15]. Criteria for TTH were delineated in a previous investigation[15], briefly: Pain described as bilateral, occurring in the temporal, frontal and occasionally in the occipital/sub-occipital region, dull, constant, pressing, and constricting, presence of nausea more frequently than vomiting, mild/moderate and rarely as severe in intensity. Arbitrary criteria to consider the presence of "multiple painful sites" in ON, TTH and MIG were the presence of four or more painful sites based in the face, head and neck based on a comprehensive questionnaire used to record pain sites. Controls were those individuals referred to the same center in the same period of time without CMDs and BB characteristics, presenting with a specific complaint including pain but without CMDs characteristics. This group was used only for comparison purposes regarding number of painful sites.

Statistical Analysis

Statistical tests used in this study included unpaired t-test to evaluate statistical differences in age, Kruskal-Wallis ANOVA with post-test (Dunn) to evaluate medians in painful sites and Fisher´s Exact test to asses multiple pain sites when comparing ON and MIG, ON and TTH and TTH and MIG individuals. Significance was accepted if $p<0.05$.

RESULTS

More females were present in the ON, TTH, MIG, and control subgroups: 28=87.5%, 94=92.2%, 9=56.3%, and 21=70%, respectively. Mean ages in these four subgroups were about 38.0, 33.0, 37.0, and 36.6, respectively, but they were not different between groups (p=0.17, Table 1).

The most common anatomic areas with pain reported by ON patients were present in the temporal + frontal + occipital + sub-occipital areas unilateral or bilateral (n=9; 28.2%), temporal + frontal + occipital + retro-orbital areas (n=7; 21.8%), temporal + frontal unilateral or bilateral (n=4; 12.5%), frontal + occipital + sub-occipital unilateral or bilateral (n=3; 9.37%) and cervical + sub-occipital + vertex unilateral or bilateral (n=2; 6.25%; Table 2).

Most TTH patients reported pain in the temporal and frontal areas bilateral (n=65; 63.7%), temporal + frontal + cervical bilateral (n=8; 7.8%), temporal + frontal + vertex bilateral (n=6; 5.9%), temporal bilateral (n=6; 5.9%), temporal + frontal + sub-occipital bilateral (n=4; 3.9%) and temporal + parietal bilateral (n=4; 3.9%; Table 3).

Sixty two and half percent (10/16) of migraineurs reported their pain in the right anterior temporal (n=4; 25%), right anterior temporal + parietal + occipital (n=2; 12.5%), left anterior temporal + parietal (n=2; 12.5%), and right anterior temporal + frontal + parietal + cervical (n=2; 12.5%). The other 37.5% (6/16) patients reported their pain each one in a different anatomic location (Table 4).

The medians in painful sites in the subgroups ON, TTH, MIG, and control were about 3.5, 2.0, 2.0, and 0.5, respectively (p<0.0001). Regarding the reporting of multiple painful sites, their frequencies were about 43.7%, 12.7% , 12.5%, and 0% in the ON, TTH, MIG, and control subgroups, respectively: ON versus TTH (p<0.0006), ON versus MIG (p<0.05) and ON versus control (p<0.0001), TTH versus control (p<0.03; Table 5).

Table 1. *Demographic data in occipital neuralgia (ON), tension type--headache (TTH), migraine without aura (MIG) patients, and control individuals.*

	ON n=32	TTH n=102	MWA n=16	control n=30
	N (%)	N (%)	N (%)	N (%)
Females	28(87.5)	94(92.2)	9(56.3)	21(70)
Males	4(12.5)	8(7.8)	7(43.7)	9(30)
Mean Age	38.0	33.0	37.0	36.6
Standard Deviation	11.5	11.9	11.9	14.8
Range	18—75	14—61	18—61	17—68

Table 2. *Patterns of local and referred pain in Occipital Neuralgia (n=32).*

Anatomic region	N(%)
Temporal+Frontal+Occipital+suboccipital (unilateral or bilateral)	9(28.2)
Temporal+Frontal+Occipital+Retro-orbital	7(21.8)
Temporal+Frontal (unilateral or bilateral)	4(12.5)
Frontal+Occipital+Sub-occipital (unilateral or bilateral)	3(9.37)
Cervical+Sub-occipital+Vertex (unilateral or bilateral)	2(6.25)
Temporal+Frontal+Parietal (unilateral or bilateral)	1(3.12)
Temporal+Sub-occipital+Cervical+Vertex (unilateral or bilateral)	1(3.12)
Temporal+Occipital+Parietal+Vertex (unilateral or bilateral)	1(3.12)
Temporal+Frontal+Occipital+Sub-occipital+Teeth	1(3.12)
Sub-occipital (unilateral or bilateral)	1(3.12)
Sub-occipital+Occipital+Vertex+Teeth (unilateral or bilateral)	1(3.12)
Sub-Occipital+Vertex+Teeth (unilateral or bilateral)	1(3.12)

DISCUSSION

Frequency of specific signs and symptoms in ON as compared to TTH

In the current investigation, we found that nausea, vomiting, photophobia, a pulsating pain, a jabbing/shooting description, electric shock-like pain, numbness, more severe pain in multiple areas, a burning description, dizziness, blurred vision, tenderness, allodynia in a pain generator area, and nasal congestion/occlusion occurred much more frequently in ON patients as compared to TTH ones. Data in the current study also indicate that

Table 3. *Anatomic areas with pain in Tension-Type Headache (n=102).*

Anatomic region	N(%)
Temporal+Frontal bilateral	65(63.7)
Temporal+Frontal+cervical bilateral	8(7.8)
Temporal+Frontal+Vertex bilateral	6(5.9)
Temporal Bilateral	6(5.9)
Temporal+Frontal+Sub-occipital bilateral	4(3.9)
Temporal+Parietal Bilateral	4(3.9)
The whole head	3(2.9)
Frontal+Occipital Bilateral	3(2.9)
Temporal+Frontal+Occipital Bilateral	1(0.98)
Temporal+Cervical Bilateral	1(0.98)
Temporal+Parietal+Occipital+cervical	1(0.98)

both neuropathic and autonomic symptoms are more frequent and such characteristics better differentiate ON patients from those presenting TTH. The outcome in the current study concurs with those of previous investigations[16,17] indicating that ON is a true neuralgic/neuropathic disorder with paroxysmal episodes of shooting electric-like symptoms occurring in the distribution of the GON and/or lesser occipital nerves and suggesting that differential diagnosis is not difficult. Because ON is a true neuropathic pain, pharmacologic treatment for cervicogenic headache and ON includes medications for the prevention of neuropathic pain[18].

The outcome in the current study has additional support in another research[2], indicating that burning, throbbing, shooting and the presence of a tender/painful area in the sub-occipital region, are more typical of ON patients. Extreme localized tenderness is often found upon palpation over the occipital notches with reproduction of focal and radiating pain[17]. Although trigger points associated tenderness in the neck also occurs frequently in TTH patients, the pain generating area, is not observed in such patients. Even though ON may be described bilaterally anterior and posterior in the head, such a disorder occurs more commonly in one side of the head as compared to TTH, which is usually described bilaterally. A pain generator allodynic sub-occipital area is the landmark of ON and is rarely observed in TTH patients. Pressure over the occipital nerves may amplify the pain in ON patients, but there is usually no clear trigger[2]. Descriptions such as burning, severe, shooting, and the presence of a pain generating area in the posterior scalp[2,19], are frequently reported by ON patients and rarely by TTH cases. When patients use descriptors such as intermittent, jabbing or throbbing and the presence of a pain generating sub-occipital area is observed clinically, the presence of ON should be suspected6. All patients in the current study described ON as very intense, severe or unbearable and these descriptors were rarely used by TTH patients. ON pain is very severe[2] and TTH pain is usually described as mild or moderate[16,20].

Frequency of symptoms in ON as compared to MWA

In the current study, we found that the frequencies of nausea, a pulsating quality, electric shock-like, severe pain, nasal congestion and nasal occlusion were not different in ON and MWA patients. We found a frequency of 40.6% nasal congestion in occipital neuralgia (ON) patients and 25% in MWA patients. Thus, the results of this study are supported by one investigation reporting that nasal congestion is present in less than 45% of MWA patients[21]. Vomiting and photophobia were observed much more frequently in MWA patients as compared to ON ones. As a whole, it seems that there is no difference in autonomic symptoms when comparing both headache types. It may be that severe pain present in both disorders is associated with the predominance of autonomic symptoms and that severe pain stimulates certain subsets of neurons in the central nervous system which in turn enhances other anatomic areas responsible for the deve-

Table 4. *Anatomic painful areas in Migraine (n=16).*

Anatomic region	N(%)
Right anterior temporal	4(25)
Right anterior temporal+parietal+occipital	2(12.5)
Left anterior temporal + parietal	2(12.5)
Right anterior temporal + frontal+parietal+ cervical	2(12.5)
Left anterior temporal	1(6.25)
Left anterior temporal + orbital region	1(6.25)
Left anterior temporal + frontal	1(6.25)
Left anterior temporal + frontal + orbital	1(6.25)
Left anterior temporal + frontal + orbital	1(6.25)
Right anterior temporal + TMJ + Frontal	1(6.25)
Temporal+Parietal+Occipital+cervical	1(0.98)

Table 5. *Painful anatomic zones in occipital neuralgia (ON), tension type-headache (TTH), migraine without aura (MIG) patients, and control individuals.*

Disorder	ON	TTH	MIG	Control
N	32	102	16	30
Median	3.5±1.15	2.0±0.94	2.0±0.89	0.5*
Range	1-6	1-6	1-4	0-3
Multiple sites pain	14(43.7%)	13(12.7%)	2(12.5%)	0(0%)**

*Kruskal-Wallis statistics with Dunn´s post test p<0.0001; **Fisher´s exact test: ON + TTH (p<0.0006), ON+MIG (p<0.05), ON + Control (p<0.0001), TTH + MIG (p=1.000), TTH + Control (p<0.03), MIG + Control (p=0.11).

lopment of autonomic symptoms. The current data also indicate that jabbing pain, numbness, pain in multiple anatomic zones, burning, dizziness, blurred vision and the presence of a sub-occipital "pain generator area", are common characteristics in ON.

The outcome in the current study is in accordance with previous investigations[2,3,6,19] indicating that neuropathic features including a burning, throbbing and shooting quality, presence of a sub-occipital pain generator area, numbness, jabbing, dizziness and a positive response to local anesthetic blocks, are clinical characteristics of ON. It is noteworthy to mention that even though ON is typically a neuropathic pain, only 21.8% of ON patients in the current study described their pain as electric-shock like. It may be that pain quality in patients with such a disorder, is duration and/or intensity dependent. 34.4% of ON patients reported the presence of numbness and this outcome is in line with one case series study[3], reporting that tingling/paresthesia is not found in all ON patients[3].

Symptoms that differentiate ON from MWA

Based on the results of the current study, it is apparent that neuropathic rather than autonomic symptoms better differentiate ON from MWA. Descriptions of jabbing, electric-shock like, burning, dizziness and the presence of allodynia in the "pain generator area", were observed much more frequently in ON. These symptoms could be used by the novice and experienced clinician to establish a diagnosis and differentiate ON from MWA. This assumption and observations are echoed by other investigations[2,6,18,22] indicating that ON is a true neuropathic pain described in the sub-occipital, frontal, temporal, and orbital areas of the head.

MWA and ON are headache pains described as very intense, with the presence of many autonomic symptoms. However, pain in MWA is less severe, it occurs more frequently in the anterior/lateral region of the head, whereas pain in ON is always described as very intense occurring in both the anterior and posterior region of the head. Both ON and MWA present with many autonomic symptoms. However, there is a predominance of neuropathic symptoms in ON as compared to MWA. Thus, the presence of neuropathic symptoms should be used by the pain clinician to differentiate ON from MWA[8]. Even though throbbing and nausea are observed frequently in both ON and MWA, the presence of a burning, jabbing, intermittent, a shooting description and the "pain generator area" commonly observed in ON patients, should alert the astute clinician to differentiate one headache from the other[2].

The clinical relevance of different clinical presentations and probably different etiological factors in ON, MWA and TTH points to differential management strategies. ON is better treated with injections of bupivacaine 0.5% into the muscles of the occipital region along the nuchal line, thus blocking superficial occipital nerves which may bring immediate headache relief[23]. When anesthetic blockade and neurolytic procedures are used, a course of physical therapy and rehabilitation is recommended to enhance functional restoration and affect a longer lasting analgesic effect[24]. Other techniques including implantable electrodes for intractable ON[25], and medication frequently used for neuropathic disorders may also be of great value in ON patients. As for migraine, its etiology is not clearly understood, however, there is evidence that psychological factors play a major role, thus, counseling, psychotherapy and medication for anxiety, stress and depression are highly indicated[26]. MWA may also be aggravated by muscle tension and cervicogenic disorders, thus, electrical stimulation in the cervical region may be very helpful[11]. Abortive migraine medication such as triptans, beta-blockers, calcium channel blockers, NSAIDS, ergot derivatives and antidepressants are also commonly used as preventive treatment for migraine[12].

depression and even somatoform disorders are commonly observed in TTH patients[27], thus, psychological treatment, local anesthesia for trigger points, massage and electrical stimulation are also indicated and may be also helpful in the treatment of BB and CMDs. Because the frequencies of ON, TTH and MWA are very high among CMDs and BB individuals, the astute clinician should refer such patients for neurological assessment.

CONCLUSION

Based on the results of the current study and the review of the literature, there are reasons to believe that intensity, laterality of pain and the presence of many autonomic symptoms in MWA, better differentiate such a headache from TTH. On the other hand, because autonomic symptoms are predominant in both ON and MWA, they are not useful to establish a differential diagosis. It is apparent that neuropathic symptoms observed very frequently in ON, better differentiate this headache from MWA. The presence of a "pain generator area" in the sub-occipital area is observed only in ON patients. Because MWA may also present trigger points in the sub-occipital area, and tenderness rather than a "pain genera ntor area", is more frequently found, thus, the absence of a pain generator area in MWA may be used as a diagnostic tool to differentiate one headache from the other.

Limitations of this investigation: because the strength of a statistical test depends on sample size, and we evaluated only 16 MWA individuals , this relatively small sample may to a certain extent influence the results of the statistical tests herein used, and thus, limit the generalization of the results. Consequently, the results of this investigation should be examined with caution. Studies using larger samples of MWA patients should be carried out in order to replicate the results reported in the currentinvestigation.

REFERÊNCIAS

1.Ruiz-López R, Erdine S. Treatment of craniofacial pain with radiofrequency procedures. Pain Pract 2002;2:206-13. http://dx.doi.org/10.1046/j.1533-2500.2002.02025.x

2.Barna S, Hashmi M. Occipital neuralgia. Pain Manag Rounds 2004;1:1-6.

3.Yi X, Cook AJ, Hamill-Ruth RJ, Rowlingson JC. Cervicogenic headache in patients with presumed migraine. Missed diagnosis or misdiagnosis? Pain 2005;6:700-3.http://dx.doi.org/10.1016/j.jpain.2005.04.005

4.Ward JB. Greater occipital nerve block. Sem Neurol 2003;23:59-62.http://dx.doi.org/10.1055/s-2003-40752

5.Franzini A, Messina G, Leone M, Broggi G. Occipital nerve stimulation (ONS). Surgical technique and prevention of late electrode migration. Acta Neurochiru 2009;151:861-5. http://dx.doi.org/10.1007/s00701-009-0372-8

6.Sahai-Srivastava S, Zheng L. Occipital neuralgia with and without migraine: Difference in pain characteristics and risk factors. Headache 2011;51:124-8.http://dx.doi.org/10.1111/j.1526-4610.2010.01788.x

7.Ducic I, Hartman EC, Larson EE. Indications and outcomes for surgical treatment of patients with chronic migraine headaches caused by occipital neuralgia. Plast Reconst Surg 2009;123:1453-60. http://dx.doi.org/10.1097/PRS.0b013e3181a0720e

8.Weiss C, Meza N, Rojo A, Hernández J. Neuralgia occipital: Relato de caso y revisión de la literatura. Rev Memo Com 2009;3:8-16.

9.Evans RW. Diagnostic testing for headache. Med Clin North Am 2001;85:865-85.

10.Kapoor V, Rothfus WE, Grahovac SZ, Stephen Z, Kassam A. Horowitz MW. Refractory occipital neuralgia: Preoperative assessment with CT guided nerve block prior to dorsal cervical rhyzotomy. AJNR Amer J Neuroradiol 2003;24:2105-10.

11.Piovesan EJ, Di Stani F, Kowacs A, Mulinari RA, Radunz VH, Utiumi M, et al. Massaging over the greater occipital nerve reduces the intensity of migraine attacks. Arq Neuropsiquiatr 2007;65:599-604.http://dx.doi.org/10.1590/S0004-282X2007000400010

12.Mehta SA, Epstein JB, Greene C. Recognition and management of headache. JCDA 2006;72:835-9.

13.The International Classification of headache disorders: 2nd edition. Headache Classification Subcommittee of the International Headache Society. Cephalalgia 2004;24(Suppl 1):9-160.http://dx.doi.org/10.1111/j.1468-2982.2003.00824.x

14.Molina OF, Peixoto MG, Eid NL, Aquilino RN, Rank RC. Headache and bruxing behavior types in craniomandibular disorders (CMDs) patients. Rev Neurocienc 2011;19: 449-57.

15.Molina OF, dos Santos J, Nelson SJ, Nowlin T. A clinical study of signs and symptoms of CMDs in bruxers classified by the degree of severity. J Craniomand Pract 1999;17:268-79.

16.Skaribas I, Aló K. Ultrasound imaging and occipital nerve stimulation. Neuromodulation 2010;13:126-30. http://dx.doi.org/10.1111/j.1525-1403.2009.00254.x

17.Weiner RL, Reed KL. Peripheral stimulation for control of intractable occipital neuralgia. Neuromodulation 1999;2:217-21.http://dx.doi.org/10.1046/j.1525-1403.1999.00217.x

18.Yadla S, Ratliff SJ., Harrop JS. Whiplash: Diagnosis, treatment and associated injuries. Curr Rev Musculoskelet Med 2008;1:65-8. http://dx.doi.org/10.1007/s12178-007-9008-x

19.Rampello L, Vecchio I, Migliore M, Malaguarnera M, Malaguarnera G. The most frequent and surgical neuralgias: physipathology and clinical pictures. Acta Med Medit 2012;28:109-11.

20.Pielsticker A, Haag G, Zaudig M, Lautenbacher S. Impairment of pain inhibition in chronic tension-type headache. Pain 2005;118:215-23.http://dx.doi.org/10.1016/j.pain.2005.08.019

21.Nadkar MY, Desai SD, Itolikar MW. Migraine: Pitfalls in the diagnosis. JAPI;2010;58:10-3.

22.Gabrhelik T, Michálek P, Adamus M. Pulsed radiofrequency therapy versus greater occipital nerve block in the management of refractory cervicoge

nic headache: a pilot study. Prague Med Rep 2011;112:279-87.http://dx.doi.org/10.1016/S1754-3207(11)70669-1

23.Bano I, Chaudhary WA, Ashfaq M. Interventional pain management techniques can be helpful in headache management. Anaesth Pain Intens Care 2011;15:60-4.

24.Biondi DA. Cervicogenic headache: Diagnostic evaluation and treatment strategies. Pain Manag Rounds 2004;1:1-6.

25.Magown P, Garcia R, Beauprie I, Méndez IM. Occipital nerve stimulation for intractable occipital neuralgia: An open surgical technique. Clin Neurosurg 2009;56:119-24.

26.Waldie KE, Poulton R.Physical and psychological correlates of primary headache in young adulthood: A 26 year longitudinal study. J Neurol Neurosurg Psychiatry 2002;72:86-92.http://dx.doi.org/10.1136/jnnp.72.1.86

27.Tsuboi K. Tension-type headache. JMAJ 2002;45:202-6.