sciendo

DOI: 10.2478/rjr-2019-0016

Romanian Journal of Rhinology, Volume 9, No. 35, July - September 2019

LITERATURE REVIEW

Pain in the cervico-facial pathology: elements of etiopathogenesis, diagnosis and therapeutic principles

Bogdan Mihail Cobzeanu, Dragos Octavian Palade, Andrei Rosu, Patricia Sonia Vonica, Cristian Martu, Luminita Radulescu, Daniela Carmen Rusu, Luiza Maria Cobzeanu, Mihail Dan Cobzeanu

ENT Department, "Grigore T. Popa" University of Medicine and Pharmacy, Iasi, Romania

ABSTRACT

The authors conduct a review of the etiopathogenesis, the main diagnostic methods and the treatment principles in cervico-facial pain. Pain in otorhinolaryngology is a multifactorial symptom, based on the rich innervation and vascularisation of the cephalic extremity. Pain can be of central or peripheral origin, acute and chronic. With the diversification of methods for exploring pain-generating lesions, different treatment protocols could also be achieved.

KEYWORDS: cervico-facial pain, etiopathogenesis, diagnosis, therapeutic principles.

INTRODUCTION

Pain is an abnormal sensation, with an emotional component, which can be determined by different lesions of the parts of the human body perceived and analyzed by the central nervous system. Painful syndromes can be divided, according to the duration of evolution, in acute or chronic syndromes.

Acute pain, resulting from an accident or surgery, is a signal that informs the brain about the destruction of some tissues or about the presence of certain dangerous stimuli (mechanical, thermal, chemical).

Chronic pain is the presence of long-term pain (more than 3-6 months), as a consequence of secondary changes in the affected organism. In turn, it causes complex, physical and psychosocial changes, with consequences that are hard to bear by the patient. Chronic pain is a factor that can greatly influence the quality of life, all the more so as there is an increase in life expectancy worldwide[1-4].

In 1986, pain was classified by IASP (The International Association for the Study of Pain) according to the main anatomical regions, including cervico-facial pains[2,3]. This classification was updated in 1994 and 2004 in five distinct groups, each containing information about localization, causes, associated symptoms, characteristics (onset, duration, intensity), common evolution, complications, diagnosis criteria and treatment. Group I of pains comprises the head and face neuralgias, including idiopathic trigeminal neuralgia, post-herpetic neuralgia and glossopharyngeal neuralgia. Group II includes the craniofacial pain of musculoskeletal origin. Group III comprises pains of dental, sinus, nasal and otic origin. Group IV consists of the so-called "primary cephalalgias", including migraine, "cluster headache", tension or stress headache. Cranio-facial psychogenic pain is part of group V of the IASP classification of pain syndromes.

The classification of pain according to Ganex, from 1997, shows the existence of a nociceptive pain, somatic and visceral, and a non-nociceptive pain, neuropathic and psychogenic[1,2].

Corresponding author: Patricia Vonica
Address: ENT Department, "Grigore T. Popa" University of Medicine and Pharmacy, Iasi, Romania
e-mail: pvonica@yahoo.com;
Received for publication: February 28, 2019 / **Accepted:** April 3, 2019

ETIOPATHOGENESIS

Cervico-cranio-facial pain syndromes may be considered algias if the infectious or tumoral cause is excluded. Thus, two types of pain are described – craniofacial neuralgias and atypical cervico-facial algias.

Cranio-facial neuralgias, functional headaches, comprise both essential neuralgias at the level of the trigeminal nerve, facial nerve, glossopharyngeal nerve, Arnold occipital nerve, superior laryngeal nerve or vidian nerve, as well as symptomatic neuralgias generated by infectious and/or tumoral lesions especially in the regions of the trigeminal and glossopharyngeal nerve.

Cranio-facial algias in the form of cephalalgia may be due to vasomotor disorders of various etiology and localization.

The vascular cause, through angiospasm or of psychogenic nature, can be represented by: migrainous syndrome due to a period of vasoconstriction followed by vasodilation in the external carotid artery; Horton cephalalgia with localization on the superficial temporal branch of the external carotid artery; Sluder's syndrome or sphenopalatine ganglion syndrome; Charlin's syndrome or ciliary ganglion syndrome.

From a semiological point of view, cervico-facial algias are divided into posterior, anterior and anterolateral, median, functional pains or pains due to inflammation or tumors of the cervical region[3-6].

Atypical cervico-facial algias (neurovascular, causalgias, sympathalgias) are anterior exocranial cephalalgia and posterior exocranial cephalalgia. Anterior exocranial cephalalgia may be caused by lesions of the cranial calotte (skin infections, osteitis) or symptomatic algias determined by various otorhinolaryngological, ophthalmological, stomatological inflammatory or tumoral, traumatic conditions. Thus, in the head and neck region, pain syndromes can be due to rhinosinusal, otological, pharyngeal, laryngeal pathologies.

Posterior exocranial cephalalgia is found under three types of algias: myalgias due to prolonged contraction of the muscles of the neck, with pain at pressure; algias due to the cellulitis of the neck with pain on palpation; algias of the cervical spine by involvement of the cervical sympathetic system, which manifest through radiculalgias in arthroses, Pott disease, arthritis, medullary tumors or vascular-sympathetic syndromes (vertebrobasilar syndrome, Barré–Liéou syndrome).

In cervico-cranio-facial pain syndromes, it is essential to perform a thorough anamnesis and a multidisciplinary examination – ENT, ophthalmological, neurological and dental.

The most common acute pains in the ENT sphere are represented by acute external otitis and otitis media, rhinopharyngitis and angina, representing approximately 35% of the consultations in emergency departments[4-9].

The pain syndrome is constantly encountered in the neoplastic pathology of the head and neck, more rarely in the early stages, its incidence increasing up to 80% in the evolutionary and terminal stages. A complete evaluation allows to indicate the mechanism responsible for pain due to post-surgical or post-radiochemical nociceptive excess[5].

Another category of cervico-facial pain is represented by vascular algias of the face and the migrainous syndrome. Pain in vascular algias is often very intense, unilateral, usually involving the territory of a branch of the trigeminal nerve, with paroxysmal character and remission periods. Pain crises can be accompanied by vasomotor, ocular disorders, as well as phenomena such as nasal obstruction or facial redness[7-16].

A very important chapter of chronic cervico-facial pain is represented by cranial nerves disorders and the neurological pathology, the most notable being neuralgia of the trigeminal nerve (V), neuralgia of Arnold's nerve, neuralgia of the facial nerve (VII), neuralgia of the glossopharyngeal nerve (IX), neuralgia of the superior laryngeal nerve (X), neuralgia of the superficial cervical plexus. The trigeminal nerve (V) is the most important pain receptor at the level of the face, the viscerocranium and its associated cavities. The facial nerve (VII) through Wrisberg's intermediate nerve (VII B) ensures the sensitivity of the Ramsay-Hunt area, which includes the posterior part of the tympanic membrane, the posterior and inferior wall of the external auditory meatus and the concha. In this area there are also sensitive fibers that are part of the vagus nerve.

The glossopharyngeal nerve (IX) is a mixed nerve. It has a sensitive function for a part of the pharyngeal mucosa, the Eustachian tube, the middle ear, the tonsillar lodge, the posterior third and the base of the tongue.

The superficial cervical plexus ensures the sensory innervation of the skin of the cervical region (through the anterior cutaneous cervical nerve), of the auricle and mastoid (through the large auricular nerve and the small occipital nerve), of the parotid and retromandibular region (through the small occipital nerve) and of the supra-acromial and supraclavicular region (through the supraclavicular nerve). The vagus nerve (X) contributes to the cervico-facial sensory function

through the superior laryngeal nerve innervating the hypopharynx and the larynx[17-24].

Pain due to an excess of nociception is secondary to peripheral nociceptive stimulation. It is the most common mechanism in acute pain that occurs in a post-traumatic or post-operative context. These pains respond to the treatment of the cause and, generally, are ameliorated by analgesics of class I, such as paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs) or in severe situations by morphine derivatives.

Neuropathic pain originates in a central or peripheral nervous system injury. This pain can occur independently of any somatic lesion. Typically, the topography of pain includes a neurological territory – a trigeminal branch, a nervous terminal branch, a cervical root, etc. These pains hardly respond to classic analgesics, but are more sensitive to the treatment with antidepressants, antiepileptics, local anaesthetics or neurostimulation techniques.

Idiopathic pains are functional, unexplained pains that have a stereotypical picture. Due to these characteristics, diagnoses such as tension headache, glossodynia, myofascial pain can be formulated. The physiopathological mechanism is not known, and the clinical and paraclinical examination does not reveal pathological changes that may explain the symptoms.

Psychogenic pain is triggered by a psychological issue such as a life event, death or an underlying psychopathy (anxiety, anguish, depression). Often, the pain described is evocative for this type of pathology: lush, polymorphic, atypical and variable-over-time description.

Orofacial and cervical pain has different etiology. It can be caused by dental and temporomandibular joint disorders; facial bones disorders; rhinosinusal or pharyngeal disorders; salivary gland disorders (submaxillary, sublingual and parotid). Neurological disorders (trigeminal neuralgia, neuralgia of the glossopharyngeal nerve, of the superior laryngeal nerve, suboccipital or supraorbital neuralgias, etc.), vascular problems that can cause migraine, Horton's superficial temporal arteritis, reflex pain (for example, pain atypical to myocardial infarction with left mandibular reflection) are important causes in the etiopathogenesis of pain syndromes. Psychogenic disorders as well as neoplastic disorders in the ENT sphere are clinical entities potentially generating painful symptoms.

Pain due to otological causes can be generated by affections of the auricle and the external auditory meatus (for example, trauma, hematomas, external otitis, frost and burn injuries, foreign bodies, tumors) or middle ear disorders (acute and chronic suppurative otitis media, trauma with or without injury to the eardrum) [4,5].

Rhinosinusal pain is determined by the infecto-inflammatory or tumoral pathology of the nose or the paranasal sinuses. The etiology of rhinosinusal pain may include conditions such as acute catarrhal rhinosinusitis of viral etiology, acute bacterial suppurative rhinosinusitis (Group A Streptococci, Mycoplasma pneumoniae, Pseudomonas aeruginosa, Haemophilus influenzae) or fungal rhinosinusitis (Candidosis, Aspergillosis).

Local traumatic lesions, produced by contusions, fractures or the presence of intranasal foreign bodies, with the potential consecutive manifestations[6-9] (septal abscess, septal hematoma) are a cause of pain in the rhinosinusal region that needs to be mentioned. The existence of inflammatory lesions of the nasal pyramid (folliculitis, furunculosis, small fissures and crusted ulcers, Herpes simplex virus, chondritis and osteitis, Herpes Zoster of the trigeminal nerve) manifests by the occurrence of severe, intense painful crises.

DIAGNOSTIC

Diagnosis of orofacial and cervical pain should involve several stages.

A thorough anamnesis that includes the history of the illness and pain should provide detailed information about: the sudden or gradual nature of pain; pain duration and location; the progression of the painful sensation (slow or rapid evolution); the frequency of painful attacks and circadian rhythm; the severity of pain and its association with other vegetative symptoms (such as nausea, vomiting); the coexistence of cardiovascular disorders (myocardial infarction) or favouring factors such as administered medication, stress. Another very important thing is the way and the extent to which pain affects the quality of life.

The clinical examination of the patient should include both the specialized examinations, but also the interdisciplinary examinations of neurology, ophthalmology or rheumatology.

The paraclinical investigations that should be routinely performed in the case of a painful cranio-cervico-facial syndrome are radiological investigations (CT scan, angio-MRI to highlight endocranial or vascular disorders), laboratory tests, biopsy examinations, neurophysiological tests, drug tests (administration of carbamazepine in trigeminal neuralgia or triptans in case of suspicion of a migrainous syndrome)[5,10,11].

PRINCIPLES OF TREATMENT IN THE CERVICO-FACIAL PAIN SYNDROME

In case of cervico-facial pain, the therapeutic principles should take into account: the nature of pain (acute or chronic), the etiopathogenic mechanisms of pain, the induced or etiological psychoneurotic profile, as well as stress-related adjuvant factors.

The optimal pain therapy should include pharmacological treatment (such as anaesthetics, analgesics, co-analgesics that can potentiate the action of analgesics, para-analgesics), individual psychosocial counselling of the patient, non-invasive procedures (psychotherapy, acupuncture), surgical interventions or alternative methods of rehabilitation which seek to improve the patient's functional status.

Drug treatment

Nociceptive pain, associated with inflammation, responds to antipyretic analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), opioids.

Non-nociceptive pain, represented by the neuropathic pain, responds to treatment with antiepileptics, antipsychotics, and pain of psychogenic nature responds to treatment with antidepressants.

The WHO analgesic "ladder" provides the stepwise treatment depending on pain intensity. Pain is classified according to the intensity measured on a numerical or descriptive visual analogue scale (VAS) in mild pain (VAS 1-3), moderate (VAS 4-6) and severe (VAS 7-10). Each group resulting from this classification corresponds to a class of drugs that should be used to ensure the effective treatment of pain.

For mild pain, non-opioid analgesics should be used (NSAIDs and paracetamol). For moderate pain class II opioids will be used (codeine, dihydrocodeine, tramadol, dextropropoxyphene, pentazocine – but not all of these drugs are indicated in the treatment of pain). For severe pain, class III opioids (which include morphine, oxycodone, methadone, fentanyl, hydromorphone, buprenorphine, pethidine or heroin) are indicated.

Neoplastic pain is a special category. In the case of these pain syndromes three types of drugs can be used: pure agonists, represented by morphine and succinates; pure antagonists such as naloxone and naltrexone; or agonists – antagonists, this category comprising pentazocine-fortral.

If pain is semi-responsive to opioids, on each of the three steps of the WHO analgesic ladder, one can use, along with analgesics and co-analgesics, drugs that, as first intention, are not antalgic but which, added to an antalgic drug, potentiate its effect.

The analgesic "ladder" also provides how to combine analgesics. Thus, class II and III drugs will never combine because they have the same mechanism of action. The binding to opioid receptors makes it possible for class I drugs to combine with any drug of the other two classes. The transition from one class to the next is made if the patient's pain is not controlled using the analgesic of that class at the maximum dose. In class I – mild to moderate pain – Paracetamol, Ibuprofen, Piroxicam, etc. are used, but Algocalmin is no longer administered. In the cases unresponsive to these NSAIDs, weak opioids from the range of DH Codeine and Tramadol can be administered. In class II – intense to unbearable pain – strong opioids like Morphine or Fentanyl are directly used.

In chronic pain, the medullary, peridural neurostimulation can also be applied.

The neuropathic psychogenic component of pain responds to anticonvulsant and antidepressant therapy. Psychiatric treatment is useful in treating chronic pain.

Opioid analgesics are indicated in: cancerous or non-cancerous chronic pain; severe acute pain; preoperatively, to lower doses of postoperative analgesics; nociceptive pain (due to tissue destruction, postoperative pain) neuropathic pain (trigeminal neuralgia, post-thrombotic neuralgia), but with variable results[4,5,25-29].

Surgical therapy

Depending on the trigger cause, the surgical treatment of painful cervico-facial syndromes may consist of: cryosurgery, differential thermocoagulation of Gasser's ganglion or gangliolysis radicotomy. Classic surgery (the resection of the sphenopalatine ganglion in Sluder's syndrome, the resection of the internal maxillary artery in atypical neuralgias, the resection of the middle meningeal artery in rebel migrains, the resection of the superior maxillary nerve in the painful facial tic or facial neuralgias that occur after dental avulsions) or neurosurgical therapy (for endocranial and spine lesions) are alternative treatment methods, in case of failure of other therapies.

Adjuvant therapy

The adjuvant therapy of pain, such as balneophysiotherapy, kinesitherapy, hypnosis and antihypnosis, behavioural therapy, associeted or not with vitamin B-complex supplements, may be considered especially in cases where the medical or surgical treatment has not had the expected results or has had some contraindications.

Acupuncture can be used as an adjuvant method in trigeminal neuralgias[30].

Anesthetic blockages are used in vascular algias – novocaine periarterial infiltrations – and in spasmodic rhinopathy, rhinogenic headaches – intramucosal nasal blockage with 1% novocaine solutions.

Cognitive behavioural therapy, through specific methods of education, self-education and self-control through biofeedback, is done in order to reduce the background of anxiety and depression that often accompanies pain, especially chronic pain.

Pain therapy involves strategies that intervene in the processes of reception, transmission or projection of the painful sensitivity.

Complementary or alternative therapies of pain act on several levels to combat this suffering by using motion techniques (antalgic kinetotherapy), some physical factors and cognitive-behavioural psychological means.

If for pains of inflammatory origin the pharmacological arsenal is satisfactory, for neuropathic pains the results are only partially satisfactory despite the many pharmacological agents used.

It is paramount that treating pain does not rule out the resolution of the cause.

Psychological (sometimes psychiatric) behavioural cognitive therapy, when properly mastered, can greatly contribute to the correct management of pain[31-34].

CONCLUSIONS

Cervico-facial pain is part of a pain syndrome of diverse etiology, belonging to several specialties related to this anatomic region, the syndrome presenting an acute or chronic character that often results in differential diagnosis.

Knowing the etiopathogenic mechanisms of cervico-facial pain is useful in specifying the therapeutic protocol. The clinical and paraclinical diagnosis confirms both the type of lesion and the mechanism of producing painful manifestations.

The treatment proposed is individualized for each case, often requiring a multidisciplinary approach, with the application of therapeutic schemes according to the guidelines of National and International Algesiology Societies.

Conflict of interest: The authors have no conflict of interest.

Contribution of authors: All authors have equally contributed to this work.

REFERENCES

1. Bose CJ, Capobianco DJ, Cutrer FJ, Cutrer FJ, Dodick DW, Garza I, et al. Headache and other craniofacial pain. In: Bradley WG, Daroff RB, Fenichel GM, Jankovic J (eds). Neurology in clinical practice. Volume 2. Fifth Edition. Butterworth-Heinemann, Munich; 2008, p. 2011–62.
2. International Headache Society. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. 2nd Edition. Cephalalgia. 2004;24(Suppl 1):9–160.
3. Silberstein SD, Olesen J, Bousser MG, Diener HC, Dodick D, First M, et al. The international classification of headache disorders, 2nd Edition (ICHD-II)–revision of criteria for 8.2 Medication-overuse headache. Cephalalgia. 2005;25(6):460–5.
4. Enache R, Sarafoleanu D. Pain in otorhinolaryngology. Rom J Rhinol. 2013;3(10):103-16.
5. Tran Ba Huy P, Manach Y. Les urgencies en ORL. Societe Fracaise d'ORL et de Chirurgie de la Face et de Cou. Paris; 2002, p.431-49.
6. Cady RK, Schreiber CP. Sinus headache: a clinical conundrum. Otolaryngol Clin North Am. 2004;37(2):267–88.
7. Krams B, Echenne B, Leydet J, Rivier F, Roubertie A. Benign paroxysmal vertigo of childhood: long-term outcome. Cephalalgia. 2011;31(4):439–43. DOI: 10.1177/0333102410382797. Epub 2010 Sep 17.
8. Oshinsky ML. Headache pathogenesis. In: Silberstein SD. (ed) Neurology and clinical neuroscience. 1st Edition. Mosby, St Louis; 2007, p. 734-8.
9. Sarafoleanu C. Sindroamele algice de origine rino-sinusala. In: Sarafoleanu C. (sub redactia) Rinologie. Editura Medicala, Bucuresti; 2003, p.567-82.
10. Paulson EP, Graham SM. Neurologic diagnosis and treatment in patients with computed tomography and nasal endoscopy negative facial pain. Laryngoscope. 2004;114(11):1992–6.
11. Stein C, Clark JD, Oh U, Vasko MR, Wilcox GL, Overland AC, et al. Peripheral mechanism of pain and analgesia. Brain Res Rev. 2009;60(1):90-113. DOI: 10.1016/j.brainresrev.2008.12.017. Epub 2008 Dec 31.
12. Jarvis MF, Boyce-Rustay JM. Neuropathic pain: models and mechanisms. Curr Pharm Des. 2009;15(15):1711-6.
13. Lebovitz A. The psychological assessment of pain in patients with chronic pain. In: Wilson PR, Watson P, Haythornwaite J, Jensen TS, eds. Clinical Pain Management: Chronic Pain. 2nd Edition. London: Hodder and Stoughton Ltd; 2008, p. 122-31.
14. Dodick DW, Rozen TD, Goadsby PJ, Silberstein SD. Cluster headache. Cephalalgia. 2000;20(9):787–803.
15. Altura BM, Altura BT, Gebrewold A. Alcohol induced spasm of cerebral blood vessels: relation to cerebrovascular accidents and sudden death. Science. 1983;220(4594):331-3.
16. Ashina M. Tension-type headache. In: Silberstein SD. (ed) Neurology and clinical neuroscience. 1st Edition. Mosby; 2008, p. 757-61.
17. Devor M, Amir R, Rappaport ZH. Pathophysiology of trigeminal neuralgia: the ignition hypothesis. Clin J Pain. 2002;18(1):4-13.
18. Kondev L, Minster A. Headache and facial pain in children and adolescents. Otolaryngol Clin North Am. 2003;36(6):1153-70.
19. Baier B, Winkenwerder E, Dieterich M. "Vestibular migraine": effects of prophylactic therapy with various drugs. A retrospective study. J Neurol. 2009;256(3):436–42. DOI: 10.1007/s00415-009-0111-3. Epub 2009 Mar 6.
20. Tepper SJ, Rapoport A, Sheftell F. The pathophysiology of migraine. Neurologist. 2001;7(5):279-86.

21. Schreiber CP, Hutchinson S, Webster CJ, Ames M, Richardson MS, Powers C. Prevalence of migraine in patients with a history of self-reported or physician-diagnosed "sinus" headache. Arch Intern Med. 2004;164(16):1769–72.
22. Lempert T, Neuhauser H. Migrainous vertigo. Neurol Clin. 2005;23(3):715–30, vi.
23. Rozen TD. Trigeminal autonomic cephalalgias. Neurol Clin. 2009;27(2):537–56. DOI: 10.1016/j.ncl.2008.11.005.
24. Prasad S, Galetta S. Trigeminal neuralgia: historical notes and current concepts. Neurologist. 2009;15(2):87–94. DOI: 10.1097/NRL.0b013e3181775ac3.
25. Scher AI, Stewart WF, Lipton RB. Migraine and headache: a meta-analytic approach. In: Crombie IK, Croft PR, Linton SJ, Leresche L, Von Korff M. (eds) Epidemiology of pain. IASP Press, Seattle; 1999, p. 159–70.
26. Magis D, Schoenen J. Treatment of migraine: update on new therapies. Curr Opin Neurol. 2011;24(3):203–10. DOI: 10.1097/WCO.0b013e3283462c3f.
27. Kari E, DelGaudio JM. Treatment of sinus headache as migraine: the diagnostic utility of triptans. Laryngoscope. 2008;118(12):2235–9. DOI: 10.1097/MLG.0b013e318182f81d.
28. Thurel C, Serrie A, Cunin G, Tran Ba Huy P. Traitement des douleurs irreductibles des cancers de la sphere ORL. Rev Prat. 1989;4;294-8.
29. Cruccu G, Gronseth G, Alksne J, Argoff C, Brainin M, Burchiel K, Nurmikko T, et al. AAN-EFNS guidelines on trigeminal neuralgia management. Eur J Neurol. 2008;15(10):1013–28. DOI: 10.1111/j.1468-1331.2008.02185.x. Epub 2008 Aug 21.
30. Xie Z. 51 cases of occipital neuralgia treated with acupuncture. J Tradit Chin Med. 1992;12(3):180-1.
31. Gronseth G, Cruccu G, Alksne J, Argoff C, Brainin M, Burchiel K, et al. Practice parameter: the diagnostic evaluation and treatment of trigeminal neuralgia (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the European Federation of Neurological Societies. Neurology. 2008;71(15):1183–90. DOI: 10.1212/01.wnl.0000326598.83183.04. Epub 2008 Aug 20.
32. Katusic S, Williams DB, Beard CM, Bergstralh E, Kurland LT. Incidence and clinical features of glossopharyngeal neuralgia, Rochester, Minnesota, 1945- 1984 . Neuroepidemiology. 1991;10(5-6):266-75.
33. Braun ES, Schwedt JA, Swarm TJ, Hocking RA. Pain management in the head and neck patient. In: Flint PW, Haughey BH, Lund VJ, Niparko JK, Richardson MA, Robbins KT, et al (eds) Cummings Otolaryngology Head & Neck Surgery. Volume 1. 5th Edtion. Mosby/Elsevier; 2010, p. 239–49.
34. Graff-Radford SB, Newman A, Ananda A. Treatment options for glossopharyngeal neuralgia. Therapy. 2005;2(5):733-7. DOI: 10.1586/14750708.2.5.733.