Clinical functional anatomy of the pterygopalatine ganglion, cephalgia and related dysautonomias: A review

Seyed Ali Khonsary, Quanfeng Ma, Pablo Villablanca, Josh Emerson, Dennis Malkasian

Department of Neurosurgery, Skull Base Laboratory, Department of Radiological Sciences, UCLA School of Medicine, Los Angeles, CA 90036, USA

E-mail: *Seyed Ali Khonsary - akhon@ucla.edu; Quanfeng Ma - quanfeng.ma@gmail.com; Pablo Villablanca - pvillablanca@mednet.ucla.edu; Josh Emerson - jemerson@mednet.ucla.edu; Dennis Malkasian - dennis.malkasian@sbcglobal.net

*Corresponding author

Received: 14 February 13 Accepted: 21 October 13 Published: 20 November 13

This article may be cited as:
Khonsary SA, Ma Q, Villablanca P, Emerson J, Malkasian D. Clinical functional anatomy of the pterygopalatine ganglion, cephalgia and related dysautonomias: A review. Surg Neurol Int 2013;4:4522-8.
Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2013/4/7/422/121628

Copyright: © 2013 Khonsary SA. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract
The purpose of this article is to explain the anatomy of the pterygopalatine ganglion (PPG), its location in the pterygopalatine fossa (PPF) in the skull, and the relationship it has to the Vidian nerve terminal branches and the fifth cranial nerve. An overview of the neuro-anatomical/clinical correlations, a spectrum of pathologies affecting the seventh cranial nerve and some therapies both medical and surgical are noted. The focus is the pterygopalatine region with discussion of the proximal courses of the seventh and fifth cranial nerves and their pathological processes. The ganglion is used as an example of neuro-anatomical model for explaining cluster headaches (CH). Radiological correlation is included to clarify the location of the PPF and its clinical importance.

Key Words: Cluster headaches, facial paresis, greater superficial petrosal nerve, pterygopalatine fossa, pterygopalatine ganglion, radiosurgery, treatment of cluster headaches, seventh cranial nerve

INTRODUCTION
The goal of this paper is to underscore a clinical condition and correlate it with the known neuroanatomical elements. The so-called cluster headaches (CH) and the facial–trigeminal cranial nerve complex are appropriate models to illustrate such a relationship. Furthermore, it will familiarize the neurosurgeons who are involved in the surgical treatment of the CH with an in-depth neuroanatomy of this region.

HISTORICAL BACKGROUND
Sluder in 1908 described a constellation of symptoms that some clinicians now describe interchangeably to CH. He was the first to describe this type of headache as nasal

headache in 1908 due to sphenopalatine (Meckel’s) ganglion involvement, but later he called it sphenopalatine ganglion neuralgia. Some authors have referred to this disorder as Sluder Neuralgia. To be noted that, the sphenopalatine ganglion is the term used in animals, in humans it is called pterygopalatine ganglion (PPG), some authors use these terms interchangeably.

CH is a recurring pain that comes under a variety of names, for example, paroxysmal nocturnal cephalgia, histamine headache (Horton headache), cranial autonomic syndrome, etc.

More recently (2004) the International Headaches Society (ICHD-II) suggested a new term, the Trigeminal Autonomic Cephalgia (TACs) to explain a group of primary headaches, which are unilateral, of short duration in the trigeminal nerve distribution,
and associated with autonomic symptoms ipsilaterally. This group of cephalgias include: Paroxysmal hemierania, CH, short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT).\[^{[11,20]}\]

The question with this classification is that the trigeminal nerve has no autonomic function. It is the facial cranial nerve that renders parasympathetic autonomic secreto-motor function, but utilizes the trigeminal cranial nerve as an anatomical vehicle to reach their targets.

**CLINICAL MANIFESTATION**

CH is a severe pain, unilateral, localized in or around the eye, mostly in young males, clustering for a period of time recurring at specific time of night, often starting 1-2 hours after falling asleep or in the early morning, lasting for 6-12 weeks. Then the subject is free of symptoms for months or even years. In review of literature the character of pain is generally not addressed. However, trigger points and lancinating paroxysmal characteristic are absent, therefore it is different from trigeminal neuralgia. CH is associated with autonomic nervous system dysfunctional characteristics. Hyperparasympathetic release can be associated with hyperlacrimation, mucosal congestion, and rhinorrhea. Conjunctival vascular injection can also occur as part of the dysautonomia. Rarely, miosis (pupillary constriction) occurs, which is predominantly an oculomotor parasympathetic stimulating response. Ptosis can also occur, as a sign of inhibition of general somatic efferent function of the third cranial nerve. There is a mix of autonomic and nonautonomic dysfunctions. In short, Sluder’s CHs are of complex origin involving both, the sympathetic and the parasympathetic systems. It is a dysautonomia with a seventh cranial nerve parasympathetic propensity noteworthy of the detailed review presented herein.

**ANATOMY (NEUROANATOMY)**

**Pterygopalatine region**

The PPG is an inverted four-sided pyramid shaped space just posterior to the maxillary sinus. The PPG is located in the pterygopalatine fossa (PPF). The boundaries of the fossa are: Medially the vertical plate of the palatine bone, anteriorly is the posterior wall of the maxillary sinus, posteriorly the vertical portion of the common root of the pterygoid plates, and laterally the pterygomaxillary fissure [Figure 1]. The medial wall contains the sphenopalatine foramen, the posterior wall contains the opening of the pterygoid (Vidian) canal and foramen rotundum, and the antero-superior portion of the fossa meets the inferior orbital fissure [Figure 2]. The PPG is unique because of its parasympathetic seventh neuronal circuitry and its relationship to the maxillary branch (V2) of trigeminal nerve (V). The seventh cranial nerve uses the fifth cranial nerve as a pathway or structural vehicle as a “Freeway” for its postganglionic parasympathetic fibers.\[^{[7,12,21]}\]

**Anatomy of the autonomic fibers of the seventh cranial nerve**

The preganglionic parasympathetic neurons arise from the superior salivatory nucleus in the pons and via the nervus intermedius of facial nerve (VII) traversing but not synapsing at the geniculate ganglion of the facial nerve, forms the greater superficial petrosal nerve (GSPN). The GSPN continues over the internal carotid artery (ICA) at the distal carotid canal to enter the Vidian (Pterygoid) canal at the foramen lacerum, therefore entering the posterior medial aspect of the PPF. It synapses with the postganglionic neurons within the PPG. These fibers innervate the lacrimal gland, traveling with the zygomatic branch V2.

The preganglionic sympathetic neurons arise from the intermediate horn of spinal gray matter of spinal cord, at the first thoracic vertebrae (T1), ascending the cervical sympathetic trunk to the superior cervical ganglion to synapse with the postganglionic neurons. The postganglionic fibers follow along the internal carotid artery entering the skull as the deep petrosal nerve (DPN).

The sympathetic fibers traveling through the DPN join the GSPN at the proximal region of the canal and form the Vidian nerve, which traverses through the Vidian (Pterygoid) canal and reaches the PPF. These sympathetic post-ganglionic fibers traverse without synapsing in the PPF. They give innervation to the secretomotor elements of the lacrimal gland and nasal mucosa by traveling, as noted, with the zygomatic branch of fifth nerve.

The pure somatic sensory maxillary branch (V2) of the trigeminal nerve (V) exits the skull through the foramen rotondum and forms the infraorbital nerve with its branches. The maxillary nerve (V2) passes through the foramen rotondum and traverses superiorly in the PPF giving two to three branches to PPG named ganglionic branches (or pterygoid branches). The PPG inferiorly gives two major branches, the greater and the lesser palatine nerves, which innervate the bony palate of the buccal cavity, supplying the gum and its mucosa (the greater palatine nerve), and also the uvula, tonsils, and soft palate (the lesser palatine nerve), Figures 3 and 4.

The seventh nerve parasympathetic innervation increases the secretomotor function of nasal-palatal mucosa. The sympathetic innervation is inhibitory to the same elements. The secretomotor production is more watery-mucoid with parasympathetic stimulation, and more viscous-mucoid with sympathetic stimulation.
Several other investigators have studied different aspects of PPG and PPF. Rusu et al., in 2009 studying 20 human adult heads, found four morphological types of PPG: \cite{14}

- **Type A (10%)**: Partitioned PPG, the upper partition receiving the Vidian nerve.
- **Type B (55%)**: Single PPG, the upper part (base) receiving the Vidian nerve.
- **Type C (15%)**: Single, the Vidian nerve reaches the lower part (tip) of the ganglion.
- **Type D (20%)**: Partitioned, the lower partition receiving the Vidian nerve.

They proposed that these individual variations might be the reason of failures in ablation therapy. The same group, found two different paths concerning the sympathetic entry to the PPF. \cite{15} Apparently, postganglionic sympathetic projections use both the external carotid artery (via the maxillary artery neuronal plexus), and the ICA (via the Vidian nerve), routing to the PPG. These fibers pass through the PPG without synapsing, ending in nasal, oral, and antral region as described earlier.

Other investigators have used different approaches to study PPG and PPF. Alvernica et al. in 2007 studied cadaveric heads by using 1 mm thick slices of computed tomography (CT) and magnetic resonance (MR) images and applying software strategies. They concluded that there were clear and constant relationship between PPG and the Vidian canal, suggesting the Vidian canal as a landmark on coronal CT scan, to target the PPG with a Gamma Knife stereotactic radiosurgery for treatment of CH. \cite{3} Finally, Chen et al. in 2010 produced 0.6 mm thickness multislice spiral CT imaged in two adult cadaver heads embedded with gelatin and frozen. \cite{5} They sliced the heads with computerized milling machine with a thickness of 0.1 mm. Images were then taken by high resolution digital camera of these slices and compared with the images taken by multislice spiral CT. They...
concluded that both techniques have high consistency for displaying the PPF and its content. Figures 5-7 are three axial CT scans displaying the PPF.

TREATMENTS FOR CLUSTER HEADACHES

Medical treatment

The options for medical treatment during acute attack of CH are: Sumatriptan, ergotamine tartrate, analgesics, and oxygen inhalation of 100%. The prophylactic therapy options are valproic acid, calcium channel blockers, lithium, corticosteroids, and ergotamine, as few examples. Initially these episodes respond to medical therapy, later these become refractory to headache medications. About 10% of the patients with CH evolve to chronicity. Roughly 20% of this chronic CH is refractory to medical therapy, usually more than one year with no remission or remission lasting less than 2 weeks. These patients are candidate to surgical therapy. Interestingly, nitroglycerin, alcohol, and histamine could provoke this type of headache during the cluster period but not during the remission.[20]

Surgical and procedural treatment

Sluder in 1908 initiated the procedures for CH with cocaine moisture applicator cotton just posterior to the posterior tip of middle turbinate over the PPG. He also applied silver 2%, or formaldehyde 0.5% with variable results.

Later on in 1913, he reported injecting phenol–alcohol to the region of sphenopalatine (SPP) foramen.[19]

Multiple neurosurgical strategies were tried for this group of chronic refractory CH: Gasserian Ganglion injection, thermocoagulation of the gasserian ganglion and PPG, glycerol rhizotomy, microneurovascular decompression of trigeminal nerve, trigeminal nerve root sectioning, and stereotactic radiosurgery of PPG. Recently, deep brain stimulation (DBS) of posterior hypothalamus has been considered due to circadian nature of this disorder. This approach is at investigational level.

The following table is the summary list of some of those who contributed to the therapy of CH.

| Investigators | Year | Contribution |
|---------------|------|--------------|
| Sluder[18]    | 1908 | Applied cocaine |
| Sluder[19]    | 1913 | Injected/applied phenol-alcohol |
| Alajouanine[2] | 1933 | Percutaneous injection of cocaine |
| Gardner[8]    | 1947 | Resection of the GSPN |
| Brown[4]      | 1962 | Injected alcohol |
| Salar[16]     | 1987 | Thermocoagulation lesioning |
| Sanders[17]   | 1997 | Radiofrequency lesioning |
| Pollock[13]   | 1997 | Gamma knife surgery (used MRI or CT for localization) |
| De Salles[6]  | 2006 | Radiosurgery (used MRI, CT, and Skull X-ray for localization) |
| Kano[9]       | 2011 | Gamma knife surgery (North American Gamma Knife Consor) |

GSPN: Greater superficial petrosal nerve
MODERN SURGICAL MANAGEMENT OF CLUSTER HEADACHE

The exquisite visualization of the PPF with modern imaging and our understanding of the anatomy of this region allows for completely noninvasive approach to the treatment of CH.

De Salles et al., Kano et al., and few other investigators have pioneered new approaches for treatment of CH using stereotactic radiosurgery.

Kano et al. and De Salles et al. reported that radiosurgery provided 60% lasting pain reduction in patients with medical refractory CH.[6,9] In addition, the noninvasive technique is an advantage for the comfort of the patient.

Table 1: Lesions involving VII cranial nerve causing hypofunction

| Location of lesion | Symptoms                                                                 | Pathology                                                                                      |
|--------------------|-------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------|
| 1. Pons (brainstem) | Peripheral facial motor palsy (ipsilateral)                            | Superior salivary nucleus infarct Due to vascular thrombosis of one of the small vessels supplying the area near 4th ventricle at the level of the superior salivary nucleus involving AICA distribution |
|                    | Ipsilateral dry eye/corneal keratitis causing conjunctivitis, sicca like syndrome |                                                                                               |
|                    | Reduced salivary flow from submandibular gland                          |                                                                                               |
|                    | Dysacusia                                                               |                                                                                               |
|                    | Ipsilateral dryness (xerostomia) of paranasal sinuses, nasal cavity and oral palate |                                                                                               |
|                    | Motor, sensory, and possible coordination long track associated abnormalities |                                                                                               |
| 2. Brainstem to IAM (sub-arachnoid course) | Ipsilateral facial palsy | Ipsilateral site of damage along the facial nerve Pathway either at the CP angel, or in the petrous Bone, usually by tumors Types of tumors: VII, VIII Vest., VIII Coch. Neurolemmomas (schwanomas) Meningiomas/epidermoid-dermoid tumors Chondromas/chondrosarcomas of clivus Osteosarcoma, chordomas, lymphomas Metastatic tumors (invasive) Oral/pharyngeal carcinomas Usually unilateral multiple CN involvement of the V through XII |
|                    | Ipsilateral loss of tearing (dry eye)                                   |                                                                                               |
|                    | Loss of hearing/vestibular dysfunction (if VIII cranial nerve involved) |                                                                                               |
|                    | Decreased salivation                                                    |                                                                                               |
|                    | Altered taste sensation (Ipsil. Ant 2/3 of tongue)                      |                                                                                               |
|                    | Corneal hypesthesia (if V cranial nerve involved)                       |                                                                                               |
|                    | Ipsilateral conjunctivitis sicca-like symptoms                          |                                                                                               |
|                    | Hypesthesias of posterior portion of EAM and posterior pinna            |                                                                                               |
|                    | If brain stem is involved, long track and motor coordination abnormalities |                                                                                               |

Table 2: Lesions involving VII cranial nerve causing hypofunction

| Location of lesion | Symptoms                                                                 | Pathology                                                                                      |
|--------------------|-------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------|
| 3. Facial nerve in IAM, or facial canal or at the geniculate ganglion | Ipsilateral facial paresis/plegia | Idiopathic and viral inflammation within the facial canal Skull base FX |
|                    | Ipsilateral nasal cavity, paranasal sinuses and oral/palate mucosal dryness (xerostomia sicca like symtoms) |                                                                                               |
|                    | Hyperacusis (due to loss of the normal damping action of the stapedius muscle) |                                                                                               |
|                    | Numbness posterior 1/2 of EAM and posterior cutaneous region of pinna |                                                                                               |
|                    | Parotid ipsilateral usually spare Intact secreto-Motor from IX th. C.N. |                                                                                               |
| 4. Meckel's cavum: Trigeminal nerve neuroma compressing G.S.P.N. at petrosal apex and floor of meckel's cavum | Hypolacrimation (unilateral ipsilateral conjunctiva sicca like syndrome) | Tumors and possible bacterial/fungal abscess and osteomyelitis of petrosal region that can damage the trigeminal nerve at the upper CPA cistern, petrous apex, or Meckel’s cave |
|                    | Unilateral nasal/oral decreased mucus secretion form frust of xerostomia sicca |                                                                                               |
|                    | Possible ipsilateral homer’s syndrome 2 to deep petrosal involvement |                                                                                               |
|                    | Dysesthesias of the one or all three divisions of trigeminal nerve (V-1, V-2, and V-3 ) as well as weakness plus atrophy of the temporalis/masseter muscles (ipsilateral) |                                                                                               |
The use of Gamma Knife in treatment of CH is an ongoing process pending the final result of the North American Gamma Knife Consortium.

The 60% lasting reduction in CH is promising. In addition, with further refinement of the treatment parameters, and also the experience of the neurosurgeons involved in using the focused radiation of the PPG, with thin sections of PPG generated by CT/MRI scans, it is possible that stereotactic radiosurgery will become an important option for the neurosurgical treatment of the CH.

There are other causes of increased lacrimation that are not CH in origin. Apart from CH, PPG and its neuronal circuitry network lesions and inflammations of the surrounding anatomy play a major role in certain diseases of the eye. Hyperlacrimation, hypolacrimation, and inappropriate tearing could be due to lesions along the parasympathetic pathway from the pons up to lacrimal gland\[10\] (Tables 1-3). One example is the phenomenon of crocodile tears (Bogorad Syndrome) in which profuse and inappropriate tearing manifests when taste buds stimulation occurs and activates posttraumating and aberrant regenerated and misdirected facial parasympathetic fibers, erroneously re-routed to the lacrimal gland.\[10\]

Syndromes that cause unilateral or bilateral facial plegia of nonstructural or mechanical etiologies such as Melkerson–Rosenthal and Heerfordt’s Syndrome were not included in this discussion. Moreover, they bypass any involvement of the PPG.

**Table 3: Lesions involving VII cranial nerve causing hypofunction**

| Location of lesion                      | Symptoms                                                                 | Pathology                                      |
|----------------------------------------|--------------------------------------------------------------------------|------------------------------------------------|
| 5. Lesions of pterygopalatine fossa    | Ipsilateral hypolacrimation, conjunctiva sica like syndrome              | G.S.P.N. involvement by infection or tumor      |
|                                        | Ipsilateral dry nasal mucosa                                             | Vidian nerve involvement by infection or tumor  |
|                                        | Ipsilateral dry paranasal mucosa, xerostomia sica                       | Sphenopalatine ganglion                        |
|                                        | Ipsilateral dry oral palate mucosa, xerostomia sica like syndrome        | Nasal tumor, paranasal malignant adenocarcinomas |
| 6. Misdirection syndrome of VII\textsuperscript{th} parasympathetic system | Ipsilateral numbness of V2 distribution distribution causing              | Angiofibromas                                   |
|                                        | Numbness of infra-orbital distribution (check lower eyelid with sparing  | Neuromas/schwanomas                             |
|                                        | of the upper eyelid for sensory function)                               |                                                 |
|                                        | FREY syndrome:                                                           |                                                 |
|                                        | Parasympathetic post ganglionic fibers of VII\textsuperscript{th}        |                                                 |
|                                        | salivation misdirected in their regeneration to the skin resulting in  |                                                 |
|                                        | localized region of facial sweating when eating or drinking             |                                                 |
|                                        | Bogorad syndrome: Crocodile tears syndrome                              |                                                 |
|                                        | Tearing while eating or drinking (gustolacrimal reflex) due to           |                                                 |
|                                        | anomalous misdirected innervation of the lacrimal gland                 |                                                 |
|                                        |                                                                         |                                                 |
| **Table 4: Hyperlacrimation pathological etiologies**                     |                                                                          |                                                 |
| Types of disorders                     | Symptoms                                                                 | Pathology                                      |
| 7a. Pathological crying                | Excessive spells of crying (lack of voluntary control and of             | Damage to frontal lobes                        |
|                                        | corresponding mood (such as sadness))                                   | Damage to basal forebrain                      |
|                                        |                                                                         | Damage to thalamai                             |
|                                        |                                                                         | Damage to post. ventral hypothalamus (serotonergic dysfunction)          |
| 7b. Psychogenic crying                 | Increased tearing                                                        | Strong emotion (e.g., sadness)                 |
| 7c. Emotional incontinence             | Pathological crying and pathological laughing                           | Diverse neurologic and psychiatric findings    |
|                                        | Gelastic epilepsy                                                       | Hypothalamic hamartoma                         |
| 7d. Syndrome of pseudo bulbar palsy    | Emotional incontinence associated with dysphagia and dysarthria         | Cingulate gyrus cortical dysplasia             |
|                                        |                                                                         | Parkinsonism                                   |
|                                        |                                                                         | Various age-related dementias                  |
|                                        |                                                                         | Amyotrophic lateral sclerosis                  |
|                                        |                                                                         | Giant cell arteritis                           |
|                                        |                                                                         | Hypothalamic tumors                            |
CLASSIFICATION OF SEVENTH NERVE DYSFUNCTION

The authors classified the various causes of seventh cranial nerve dysfunction according to the location of the lesions, the associated symptoms and pathologies from the brainstem through the internal auditory meatus (IAM) and to the facial canal.

In addition, the lesions of PPF are summarized. The cases of misdirected syndromes of the parasympathetic system are discussed [Tables 1-3].

The etiologies causing hyperlacrimation are summarized according to possible causes [Table 4].

The Ramsay Hunt Syndrome caused by varicella zoster virus (VZV) affecting geniculate ganglion with facial palsy will be discussed separately in our future article.

CONCLUSION

The PPG and region are a cross road of sensory, sympathetic, and parasympathetic fibers that when dysfunctional can cause severe and variable symptoms involving the face. The most severe of these symptoms is severe and seasonal pain that impairs immensely the quality of life of patients suffering from these dysautonomias.

Better understanding of the anatomical correlation with radiological visualization of the region is important for procedural and surgical strategies now available to treat these diseases.

REFERENCES

1. Ahamed SH, Jones NS. What is Sluder’s Neuralgia. J Laryngol Otol 2003;117:437-43.
2. Alajouanine T, Thurel R. Les sympathopathies faciales. J Med Fr 1933;22:188-94.
3. Alvernia JE, Spornar DG, Olivero WC. A computed tomography scan and anatomical cadaveric study of the pterygopalatine ganglion for use in Gamma Knife treatment of cluster headache. J Neurosurg 2007;107:805-8.
4. Brown LA. Mythical sphenopalatine ganglion neuralgia. South Med J 1962;55:670-2.
5. Chen CC, Chen XX, Yang XD, Zheng JW, Li ZP, Huang F, et al. Comparative research of the thin transverse sectional anatomy and the multislice spiral CT on pterygopalatine fossa. Turk Neurosurg 2010;20:151-8.
6. De Salles AF, Gorgulho A, Golish SR, Medin PM, Malkadian D, Solberg T, et al. Technical and anatomical aspects of novalis stereotactic radiosurgery sphenopalatine ganglionectomy. Int J Radiat Oncol Biol Phys 2006;66 Suppl:553-7.
7. Drake RL, Vogel W, Mitchell AW. Gray’s Anatomy for students. London: Elsevier; Churchill Livingstone; 2005.
8. Gardiner WJ, Stowell A, Dulsinger R. Resection of the greater superficial petrosal nerve in the treatment of unilateral headache. J Neurosurg 1947;4:105-14.
9. Kano H, Kondziolka D, Mathieu D, Stafford SL, Flannery T, Niranjan A, et al. Stereotactic radiosurgery for intractable cluster headache: An initial report from the North American Gamma Knife Consortium. J Neurosurg 2011;114:1736-43.
10. Kawasaki A. Disorders of pupillary function, accomodation, and lacrimation. Walsh and Hoyt’s Clinical Neuro-ophthalmology, 6th ed, Vol. 1. Baltimore: Williams and Wilkins; 2005.
11. Nappi G, Moskowitz MA. Cluster headache and trigeminal autonomic cephalgies: General aspects. Handbook of Clinical Neurology 2011;97 (3rd series);387-8.
12. Netter F, editor. Atlas of human anatomy. Netherlands: Elsevier; 2011. p. 12.
13. Pollock BE, Kondziolka D. Stereotactic radiosurgical treatment of sphenopalatine neuralgia. J Neurosurg 1997;87:450-3.
14. Rusu MC, Pop F, Curca GC, Podoleanu L, Voinea LM. The pterygopalatine ganglion in humans: A morphologic study. Ann Anat 2009;191:196-202.
15. Rusu MC, Pop F. The anatomy of the sympathetic pathway through the Pterygopalatine fossa in humans. Ann Anat 2010;192:17-22.
16. Salar G, Ori C, Iob I, Fiore D. Percutaneous thermocoagulation for sphenopalatine ganglion neuralgia. Acta Neurochir (Wien) 1987;84:24-8.
17. Sanders M, Zuurmond WW. Efficacy of sphenopalatine ganglion blockade in 66 Patients suffering from cluster headache: A 12- to 70- month follow-up evaluation. J Neurosurg 1997;87:876-80.
18. Sluder G. The role of the sphenopalatine (or Meckle’s) ganglion in nasal headaches. New York Med J 1908;87:989-90.
19. Sluder G. Etiology, diagnosis, prognosis and treatment of sphenopalatine ganglion neuralgia. JAMA 1913;61:1201-6.
20. Waldenlind E, Sjostrand C. Pathophysiology of cluster headache and other trigeminal autonomic cephalgies. Handb Clin Neurol 2011;97:389-411.
21. Williams PL, Warwick R, Dyson M, Bannister LH. Gray’s Anatomy. London: Churchill Livingstone; 1989. p. 1098-107.