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

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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

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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.[18] 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.[18,19] Some authors have referred to this disorder as Sluder Neuralgia.[1] 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 hemicrania, 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 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 (pupilllary 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 pteygoid 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 nervous intermedus 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 secreto-motor production is more watery-mucoid with parasympathetic stimulation, and more viscous-mucoid with sympathetic stimulation.

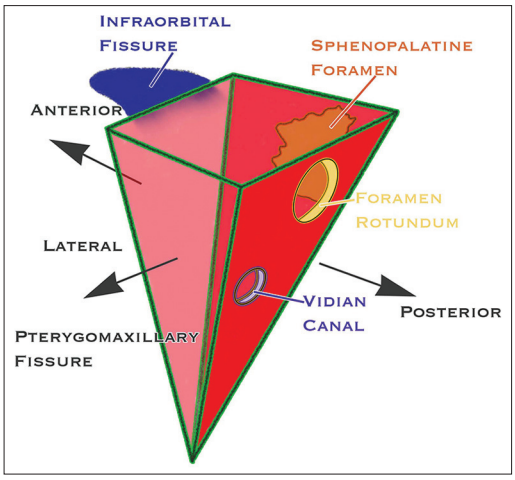

Figure 1: Schematic drawing of left sided view (lateral to medial) of the pterygopalatine fossa

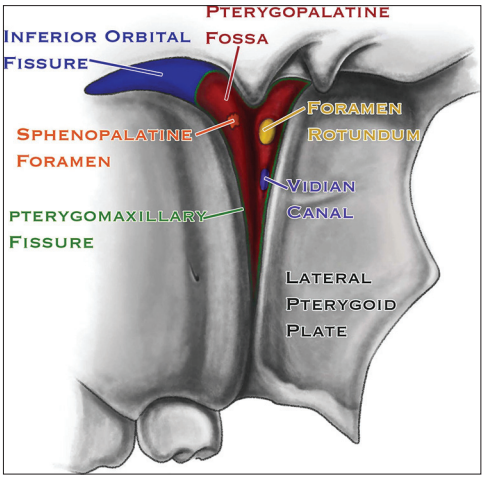

Figure 2: Left sided view of pterygopalatine fossa through pterygomaxillary fissure

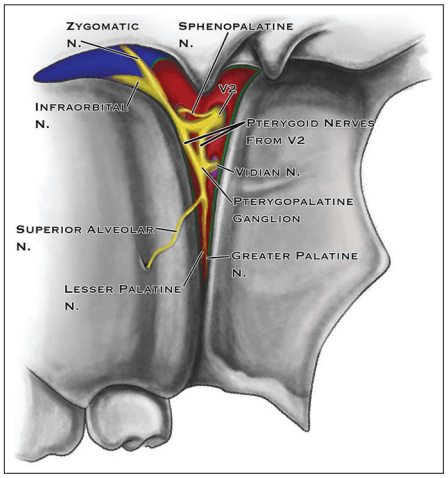

Figure 3: Left sided view of pterygopalatine fossa and its neural components through pterygomaxillary fissure (the vascular elements are not illustrated)

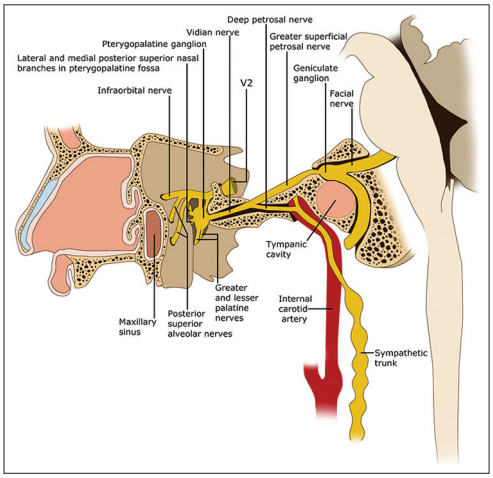

Figure 4: Detailed neuronal network of pterygopalatine ganglion and the relationship to the skull base and internal carotid artery

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:[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.[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. Alvernia *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.[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.[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.

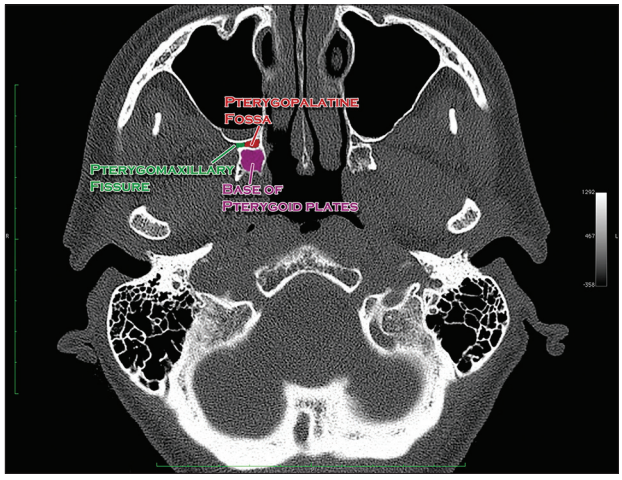

Figure 5: CT scan (bone window) axial section through the lower portion of the pterygopalatine fossa

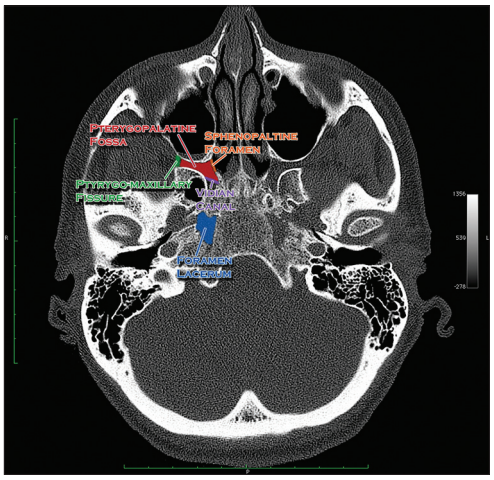

Figure 6: CT scan (bone window) axial section through the medial portion of the pterygopalatine fossa

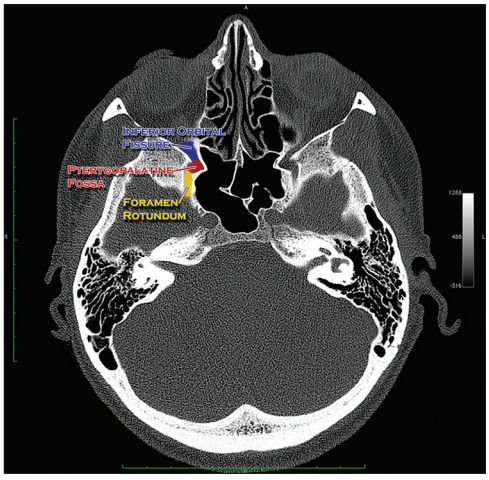

Figure 7: CT scan (bone window) axial section through the uppermost portion of the pterygopalatine fossa

TREATMENTS FOR CLUSTER HEADACHES

Medical treatment

The options for medical treatment during acute attack of CH are: Sumatriptan, ergotamine tartarate, 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.[18] 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 alcohol injection, thermocoagulation of the gasserian ganglion and PPG, glycerol rhizotomy, microvascular 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 salivery nucleus infarct
	Ipsilateral dry eye/corneal keratitis causing conjunctivitis, sicca like syndrome	Due to vascular thrombosis of one of the small vessels supplying the area near 4th ventricle at the level of the superior salivery nucleus involving AICA distribution
	Reduced salivery 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	Intramedullary neoplasms and vascular lesions
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
	Ipsilateral loss of tearing (dry eye)	Bone, usually by tumors
	Loss of hearing/vestibular dysfunction (if VIII cranial nerve involved)	Types of tumors: VII, VIII Vest., VIII Coch. Neurolemmomas (schwanomas)
	Decreased salivation	Meningiomas/epidermoid-dermoid tumors
	Altered taste sensation (Ipsil. Ant 2/3 of tongue)	Chondromas/chondrosarcomas of clivus
	Corneal hypesthesis (if V cranial nerve involved)	Osteosarcoma, chordomas, lymphomas
	Ipsilateral conjuctivitis sicca-like symptoms	Metastatic tumors (invasive)
	Hypesthesia of posterior portion of EAM and posterior pinna	Oral/pharyngeal carcinomas
	If brain stem is involved, long track and motor coordination abnormalities	Usually unilateral multiple CN involvement of the V through XII

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
	Ipsilateral nasal cavity, paranasal sinuses and oral/palate mucosal dryness (xerostomia sicca like symtoms)	Skull base FX
	Hyperacusis (due to loss of the normal damping action of the stapedius muscle)	Infection of geniculate ganglion (e.g., syphilis, herpes zoster)
	Numbness posterior 1/2 of EAM and posterior cutaneous region of pinna	Otitis media/bacterial petrositis of temporal bone e.g., pseudomonas (malignant infection)
	Parotid ipsilateral usually spare Intact secreto-Motor from IX th. C.N.	Staph aureus Rarely tumors: Metastatic or choleastoma (post-infection, advanced)
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 horner'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 posttraumatizing 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 Melkersson–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	
	Ipsilateral numbness of V2 distribution distribution causing	Nasal tumor, paranasal malignant adenocarcinomas
	Numbness of infra-orbital distribution (check lower eyelid with sparing of the upper eyelid for sensory function)	Angiofibromas Neuromas/schwanomas
6. Misdirection syndrome of VIIth parasympathetic system	FREY syndrome:	
	Parasympathetic post ganglionic fibers of VIIth salivation misdirected in their regeneration to the skin resulting in localized region of facial sweating when eating or drinking	G.S.P.N. and/or chorda tympani to the submandibular ganglion is/are misdirected and incorrectly innervate the facial skin
	Bogorad syndrome: Crocodile tears syndrome	
	Tearing while eating or drinking (gustolacrimal reflex) due to anomalous misdirected innervation of the lacrimal gland	Regeneration of post-ganglionic fibers of parasympathetic PPG toward the lacrimal gland after damage to the PPG

Table 4: Hyperlacrimation pathological etiologies

Types of disorders	Symptoms	Pathology
7a. Pathological crying	Excessive spells of crying (lack of voluntary control and of corresponding mood (such as sadness))	Damage to frontal lobes Damage to basal forebrain Damage to thalami 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 Cingulate gyrus cortical dysplasia
7d. Syndrome of pseudo bulbar palsy	Emotional incontinence associated with dysphagia and dysarthria	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.

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