

# Role of Sphenopalatine Ganglion Neuroablation in the Management of Cluster Headache

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**Abstract** Cluster headache is a primary neurovascular headache. It is a strictly unilateral head pain that is associated with cranial autonomic symptoms and usually follows circadian and circannual patterns. Chronic cluster headache, which accounts for about 10% to 15% of patients with cluster headache, lacks the circadian pattern and is often resistant to pharmacological management. The sphenopalatine ganglion (SPG), located in the pterygopalatine fossa, is involved in the pathophysiology of cluster headache and has been a target for blocks and other surgical approaches. Percutaneous radiofrequency ablation of the SPG was shown to have encouraging results in those patients with intractable cluster headaches.

**Keywords** Cluster headache · Sphenopalatine ganglion · Radiofrequency ablation

## Introduction

There are two forms of cluster headache, episodic and chronic. Episodic cluster headache occurs over periods from 7 days to 1 year separated by pain-free periods lasting at least 1 month. Chronic cluster headache occurs over the interval of more than 1 year without remission or with remissions lasting less than 1 month [1] (Tables 1 and 2).

Chronic cluster headache accounts for about 10% of patients with cluster headache, and it usually lacks the circadian pattern typical of the episodic cluster. Chronic

cluster headache patients are often resistant to pharmacological management, as they are usually more prone to tachyphylaxis and eventual loss of response [2, 3].

## Pathophysiology of Cluster Headache

Neuroimaging studies have identified the hypothalamus as the generator in cluster headaches. Cluster headache involves activation of the parasympathetic outflow from the superior salivary nucleus of the facial nerve, predominantly through the sphenopalatine ganglion (SPG) [2]. The SPG is a large extracranial structure that has rich autonomic innervation (both sympathetic and parasympathetic), which explains the autonomic features associated with cluster headache.

## Sphenopalatine Ganglion Neuroanatomy

Why the SPG?

The SPG has rich parasympathetic (preganglionic axons and postganglionic cell bodies and axons) and sympathetic (postganglionic axons) components. The parasympathetic preganglionic cell bodies projecting to the SPG originate in the superior salivatory nucleus (SSN) of the facial nerve in the pons.

The efferent fibers of the SSN travel in the nervus intermedius and divide at the geniculate ganglion to become the greater petrosal nerve and chorda tympani nerve. The first-order parasympathetic neurons in the greater petrosal nerve are joined by the postganglionic sympathetic fibers from the deep petrosal nerve, forming the nerve to the pterygoid canal (vidian nerve). The

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**Table 1** ICHD-II diagnostic criteria for cluster headache

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A. At least five attacks fulfilling B through D
B. Severe or very severe unilateral orbital, supraorbital, and/or temporal pain lasting 15–180 min if untreated
C. Headache is accompanied by at least one of the following:
1. Ipsilateral conjunctival injection and/or lacrimation
2. Ipsilateral nasal congestion and/or rhinorrhea
3. Ipsilateral eyelid edema
4. Ipsilateral forehead and facial sweating
5. Ipsilateral miosis and/or ptosis
6. A sense of restlessness or agitation
D. Attacks have a frequency from one every other day to eight per day
E. Not attributed to another disorder

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ICHD—International Classification of Headache Disorders.

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preganglionic parasympathetic neurons then synapse with the second-order parasympathetic neuronal cell bodies located in the SPG.

The postganglionic parasympathetic fibers then run with branches of the maxillary nerve ( $V_2$ ) to reach their targets. Therefore, the only cell bodies located within the SPG are those of the second-order postganglionic parasympathetic neurons, which may explain the clinical observation that patients after radiofrequency ablation (RFA) of the SPG usually notice improvement of the autonomic parasympathetic symptoms either earlier or even without improvement of the headache pain.

The sympathetic cell bodies projecting to the SPG originate in the upper thoracic spinal cord (T1–T2). The preganglionic sympathetic neurons then synapse in the cervical sympathetic ganglia, mainly the superior cervical ganglion. The postganglionic second-order sympathetic neurons form the carotid sympathetic plexus and reach the pterygoid canal through the deep petrosal nerve where it joins the first-order parasympathetic neurons in the greater petrosal nerve, forming the nerve to the pterygoid canal (vidian nerve). Postganglionic sympathetic fibers pass through the SPG without synapsing and innervate mainly blood vessels.

**Table 2** ICHD-II diagnostic criteria for episodic and chronic cluster headache**Episodic cluster headache**

- A. All fulfilling criteria A through E in Table 1
- B. At least two cluster periods lasting from 7 days to 365 days and separated by pain-free remissions of > 1 month

**Chronic cluster headache**

- A. All fulfilling criteria A through E in Table 1
- B. Attacks recur over > 1 year without remission periods or with remission periods lasting < 1 month

ICHD—International Classification of Headache Disorders.

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**Sphenopalatine Ganglion Anatomy**

The SPG is located in the pterygopalatine fossa, which is a small, upside-down pyramidal space 2 cm high and 1 cm wide. The pterygopalatine fossa is located behind the posterior wall of the maxillary sinus and is bordered posteriorly by the medial plate of the pterygoid process, superiorly by the sphenoid sinus, medially by the perpendicular plate of the palatine bone, and laterally it communicates with the infratemporal fossa [4, 5]. Superolaterally lies the foramen rotundum with the exiting maxillary nerve, and inferomedially there is the vidian nerve (greater petrosal and deep petrosal nerves) within the pterygoid canal. The pterygopalatine fossa contains the internal maxillary artery and its branches, the maxillary nerve, and the SPG and its afferent and efferent branches. The SPG is located posterior to the middle turbinate and is few millimeters deep to the lateral nasal mucosa. It is suspended from the maxillary nerve by the pterygopalatine nerves, inferiorly it is connected to the greater and lesser palatine nerves, and posteriorly it is connected to the vidian nerve. Efferent branches of the SPG form the posterior lateral nasal and pharyngeal nerves (Fig. 1).

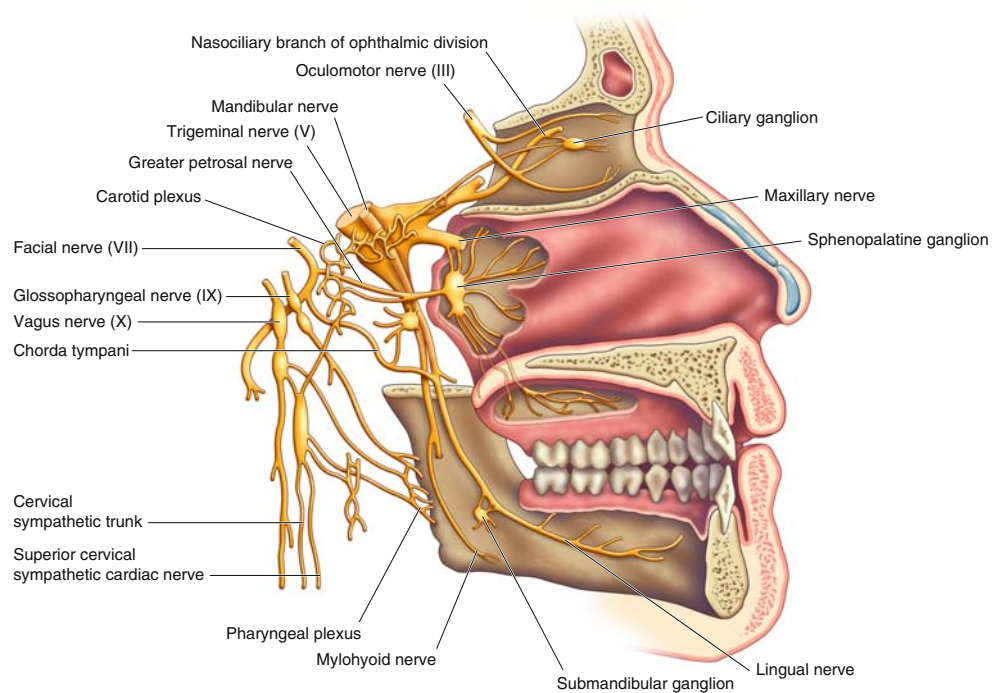
**Radiofrequency Ablation Technique**

The SPG can be blocked by intranasal topical application of local anesthetic, greater palatine foramen approach, or using the infrazygomatic approach. Neuroablation techniques are only feasible via the infrazygomatic approach, whereas the other common approaches are used mainly for diagnostic blocks [6].

The procedure is performed via the infrazygomatic approach and under fluoroscopic guidance. The needle can be advanced either anterior to the mandible or through the coronoid notch.

With the patient in the supine position and the head inside the C-arm, a lateral view is obtained and either the C-arm or the head of the patient is rotated until both pterygoid plates are superimposed on each other to better visualize the pterygopalatine fossa. The skin entry site overlying the fossa is marked just inferior to the zygomatic arch either anterior to the mandible or through the coronoid notch [7••]. A 22-gauge, 10-cm, blunt RFA needle with a 2-mm or 5-mm active tip with a slight bend at the tip is used. The needle is first introduced in the lateral view and advanced medially and superiorly toward the pterygopalatine fossa using real-time fluoroscopy. Once in a proper direction, an anteroposterior view is obtained, and the tip of the needle is advanced to be just lateral to the nasal wall. If the lateral pterygoid plate is encountered, the needle should be walked off the bone anteriorly and cephalad to slip into the fossa (the curved tip will help to guide the needle). Sensory

**Fig. 1** Anatomy of the sphenopalatine ganglion. (From The Cleveland Clinic Center for Medical Art & Photography; with permission)



stimulation is obtained with 50 Hz to look for deep paresthesias behind the root of the nose at <0.5 V (Table 3). Once proper stimulation is achieved and prior to lesioning, 0.1 mL to 0.2 mL of contrast agent is injected under real-time fluoroscopy to rule out intravascular spread. Then, 0.5 mL of lidocaine 2% is injected and two radiofrequency lesions are carried out at 80°C for 60 s each. After lesioning, 0.5 mL of bupivacaine 0.5% and 5 mg of triamcinolone are injected with the aim to prevent post-procedure neuritis.

### Efficacy of Sphenopalatine Ganglion Radiofrequency Ablation

In a retrospective analysis of patients with refractory cluster headache treated by radiofrequency lesion of the SPG, 56 patients with episodic cluster headache and ten patients with chronic cluster headache were followed over a period

of 12–70 months [5]. In the episodic cluster headache group, 60.7% experienced complete pain relief, whereas only three of ten patients with chronic cluster headache had the same result. The above report showed that RFA of the SPG may improve episodic cluster headache but not chronic cluster headache. Recently, however, Narouze and colleagues [7••] reported favorable outcome after intractable chronic cluster headache as well. They reported significant improvement in both mean attack intensity and mean attack frequency for up to 18 months in 15 patients. Of these patients, 20% (3/15) reported no change or increase in the headache intensity and/or frequency during the first few postprocedure weeks before noticing improvements in their headache pattern. However, 46.7% of the patients (7/15) reported change in the headache pattern with return to the episodic form of cluster headache at a mean follow-up period of 18 months. Three patients remained headache-free and off medications for the duration of the follow-up (18–24 months).

**Table 3** Possible scenarios of stimulation before attempting radiofrequency thermocoagulation of the sphenopalatine ganglion

Location of paresthesia	Nerves stimulated	Location of needle tip	Action needed
Upper teeth and gums	Maxillary branches	Superolateral	Redirect the needle, caudally and medially
Hard palate	Greater and lesser palatine nerves	Anterior, lateral, caudal	Redirect the needle, posteromedially and cephalad
Root of the nose	SPG efferents; posterior lateral nasal nerves	Correct needle placement	None

SPG—sphenopalatine ganglion

(Adapted from Narouze [8])

Two patients reported complete relief of their usual unilateral headache symptoms, and instead they developed episodic cluster headache on the contralateral side [7••].

## Complications of Radiofrequency Ablation

### How to Avoid?

Epistaxis is more frequent after the traditional intranasal topical application of local anesthetic; however, it can occur with this infrazygomatic approach if the needle is advanced too far medially through the lateral nasal wall. Intravascular injection and hematoma formation can occur after maxillary artery injury, which lies within the pterygopalatine fossa. Cheek hematoma is the most common complication, and infection is always a possibility especially if the oral or nasal mucosa was accidentally penetrated [8]. Further, reflex bradycardia was reported during radiofrequency lesioning, which could be explained by the rich parasympathetic connections to the SPG [9].

Radiofrequency lesioning of the SPG can result in permanent or, more commonly, temporary hypesthesia or dysesthesia in the palate, maxilla, or posterior pharynx [4–7••]. Dryness of the eye as a result of interruption of the parasympathetic supply is also common; however, it is usually only temporary. We have also noticed temporary diplopia, which is more common after local anesthetic injections, and can be explained by the spread of the injectate from the pterygopalatine fossa to the inferior orbital fissure containing the abducent nerve.

Pulsed radiofrequency would seem to be safer; however, there are only few case reports of its application in conjunction with SPG [10, 11].

A thorough understanding of the anatomy allows the clinician to predict correct needle placement during RFA according to the result of the stimulation and hence can reduce the incidence of complications (Table 3) [8].

## Conclusions

The role of the SPG in cluster headache makes it an attractive target for RFA. SPG RFA is a minimally invasive

modality of treatment that is effective in the management of intractable cluster headache. Additional studies are warranted to determine its safety and effectiveness compared with other surgical approaches.

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