REVIEW ARTICLE

The Pterygopalatine Ganglion and its Role in Various Pain Syndromes: From Anatomy to Clinical Practice

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■ **Abstract:** The postsynaptic fibers of the pterygopalatine or sphenopalatine ganglion (PPG or SPG) supply the lacrimal and nasal glands. The PPG appears to play an important role in various pain syndromes including headaches, trigeminal and sphenopalatine neuralgia, atypical facial pain, muscle pain, vasomotor rhinitis, eye disorders, and herpes infection. Clinical trials have shown that these pain disorders can be managed effectively with sphenopalatine ganglion blockade (SPGB). In addition, regional anesthesia of the distribution area of the SPG sensory fibers for nasal and dental surgery can be provided by SPGB via a transnasal, transoral, or lateral infratemporal approach. To arouse the interest of the modern-day clinicians in the use of the SPGB, the advantages, disadvantages, and modifications of the available methods for blockade are discussed.■

Key Words: pterygopalatine ganglion, sphenopalatine neuralgia, craniofacial pain syndrome, sphenopalatine ganglion block

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INTRODUCTION

Broad morphological and functional knowledge of the head anatomy is essential for neurology, neurosurgery, and maxillofacial surgery practice. Nevertheless, there are areas and structures that still have not been described or depicted sufficiently, owing to their small volume and complicated access. The pterygopalatine ganglion (PPG), also known as sphenopalatine ganglion (SPG), Meckel's or sphenomaxillary ganglion, is located in the cranial section of the autonomic nervous system and bears unique characteristics favorable for the treatment of many painful syndromes involving the face and head.[1] SPG is located near important neuroanatomic structures for pain perception. Its proximity to multiple sensory facial and trigeminal branches

suggests that SPG may be involved in persistent idiopathic facial pain (PIFP)[2] and unilateral headaches.[3] A few motor nerves accompany the SPG sensory trunks. An irritation of the SPG motor root may

- produce face and neck neuralgias by its connection with the facial nerve (FN), lesser occipital and cutaneous cervical nerves
- account for disturbances in the eye and mandible region by its connections with the ciliary and otic ganglions and a variety of visceral symptoms by its connection with the vagus nerve
- cause reflex otalgia by its connection with the tympanic plexus

In addition, there may be motor phenomena of the soft palate related to the involvement of the motor fibers to the levator palate and azygos uvulae muscles. SPG has also been identified as the first relay station of the autonomic fibers after emerging from the pons, suggesting that it may be used therapeutically in autonomic imbalance situations. SPG may play a critical role as a vasodilator to protect the brain against ischemia in stroke or ischemia of migraine with aura.[4] The SPG as a major source for postganglionic parasympathetic fibers to the vascular beds of the cerebral hemispheres is involved in tone regulation of the cerebral vessels.[5] SPG's communication with the superior cervical ganglion (SCG) (*through the internal carotid plexus*) and the intracranial portion of the internal carotid artery (*through its fibers from SPG*) is related to edema, pain, dilatation, and low-grade encephalitis.[6,7] SPG's superficial location in the pharynx may explain its sensitivity to odors, chemicals, and air particles. SPG's connection to the vagus nerve and the innervation of the saliva producing glands predicts many of the digestive symptoms observed in dysfunctional states.[8] Overall, the SPG is important for intraocular pressure balance and cerebral vasodilatation associated with vascular originated headaches.[1] Recent research has highlighted the important role of the SPG in cerebrovascular autonomic physiology, in pathophysiology of cluster and migraine headaches, and in conditions of stroke and cerebral vasospasm.[9]

ANATOMY OF THE PTERYGOPALATINE GANGLION (PPG OR SPG)

The SPG, as a parasympathetic ganglion is a nervous mass of nervous tissues found in the course of the greater petrosal nerve (GPN) located deeply in the pterygopalatine fossa (PPF). It is a small triangular or heart-shaped structure located close to the sphenopalatine foramen (SPF) posterior to the middle turbinate and maxillary sinus, anterior to the medial plate of the pterygoid process, and inferior to the sphenoid sinus and maxillary nerve (MN). The SPG is morphologically configured in the last trimester of fetal life, and during this period, its structure is established to ensure the nervous signal transmission.[10] The SPG, a complex neural center with multiple connections to trigeminal, facial, and sympathetic systems, consists of somatosensory, sympathetic, and parasympathetic fibers and receives a sensory, motor, and sympathetic root. The SPG sensory root is derived from two pterygopalatine branches of the MN; their fibers pass directly into the palatine nerves, and a few enter the SPG constituting its sensory root. The sensory distribution to the nose, throat, and sinuses gives characteristic indications in these regions.[11] The SPG motor root probably derived from the nervus intermedius (*glossopalatinus or nerve of Wrisberg*) through the greater superficial petrosal nerve (GSPN) and may consist of sympathetic efferent (*preganglionic*) fibers from the medulla. SPG motor fibers form synapses with neurons whose postganglionic axons are distributed with the deep branches of the trigeminal nerve (TN) to the mucous membrane of the nose, soft palate, tonsils, uvula, roof of the mouth, upper lip, and gums and to the upper part of the pharynx.[12] The SPG autonomic innervation is more complex. The SPG sympathetic root is derived from the internal carotid plexus through the deep petrosal nerve (DPN). The GSPN and the DPN join to form the nerve of the pterygoid canal (*vidian nerve*) before entering the SPG. The SPG parasympathetic root has its preganglionic origin in the superior salivary nucleus. The preganglionic fibers pass through nervus intermedius, FN, geniculate ganglion, and greater petrosal nerve reaching the ganglion via nerve of pterygoid canal. The GSPN carries taste along with the presynaptic parasympathetic fibers. The taste fibers pass through the SPG to the soft palate, while the parasympathetic fibers synapse in the SPG and postsynaptic fibers supply the lacrimal gland, mucosa of the palate, nasopharynx, and nasal cavity.[13]

The DPN, a branch of the internal carotid plexus (*the continuation of the cervical sympathetic trunk in the cranium*), carries postganglionic sympathetic fibers from the SCG. The postganglionic sympathetic nerve fibers pass through the SPG without synapsing and join branches of the MN, which distributes to the nasal

cavity, palate, and upper pharynx. The GSPN and DPN (join at the foramen lacerum to form the vidian nerve) pass forward, through the pterygoid canal, with the corresponding artery, and joined by a small ascending sphenoidal branch from the otic ganglion. Finally, vidian nerve enters the PPF and joins the posterior angle of the SPG[14] (Figure 1).

NERVE SUPPLY FROM THE PTERYGOPALATINE GANGLION

The nerves of the SPG are predominantly composed of MN sensory fibers. Several delicate orbital branches of the pterygopalatine nerves enter the orbit via the inferior orbital fissure to supply the orbital periosteum and muscle, and the mucous membrane of the posterior ethmoid and sphenoid sinuses. The three palatine nerves, the one greater (*anterior palatine nerve*) (GPN) and two lesser (*middle and posterior*) palatine nerves (LPN), exit the SPG inferior surface to descend behind the perpendicular plate of the palatine bone. The anterior or GPN descends through the pterygopalatine canal (PPC) accompanied by the descending palatine artery. Emerging onto the oral palatal surface at the greater palatine

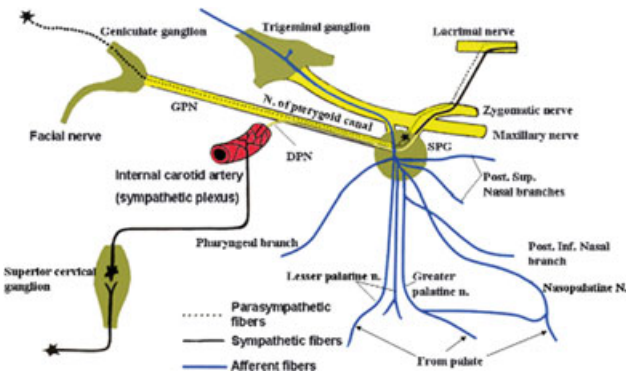

Figure 1. Parasympathetic preganglionic axons derived from the facial nerve leave the main trunk of the nerve and course as the greater superficial petrosal nerve. Sympathetic postganglionic axons derived from cell bodies in the superior cervical ganglion (SCG) course on the ICA surface and enter the skull with the artery at the carotid canal. The sympathetic axons course through the adjacent sphenopalatine ganglion (SPG) without synapsing and are distributed with the nerve and arterial branches emanating from it, to the nasal and pharyngeal mucosa. The parasympathetic fibers entering the SPG synapse there and distribute to glands in the same mucous membranes. Some of the postganglionic axons pass to the lacrimal gland by way of first the maxillary nerve, then 1 of its branches, the zygomatic nerve, and finally by way of 1 of the branches of the ophthalmic division of the trigeminal nerve, the lacrimal nerve. Sources: Adapted from Basic Human Anatomy - O Rahilly, Muller, Carpenter and Swenson (on line darmouth.edu).

foramen (GPF), it passes anteriorly, medial to the alveolar bone process giving branches to mucosa and glands of the hard palate, as far as the incisor teeth. The GPN communicates in the incisive canal region with a branch of the nasopalatine nerve. Posterior inferior lateral nasal branches of the GPN leave the nerve in the PPC and enter the nasal cavity to supply the middle and inferior meatuses and the inferior concha. Several posterior superior lateral nasal branches of the PPG pass through the SPF into the posterior part of the nasal cavity and distribute to the superior and middle nasal conchae, the posterior ethmoidal air cells, and the posterior part of the septum. The middle and posterior or LPN penetrate the hard palate via the lesser palatine foramina (LPF) to supply the soft palate, uvula, and tonsils. In addition, taste receptors of the palate are innervated by special visceral afferent fibers of the FN that are carried by the GPN and LPN, back through the PPG and the greater petrosal nerve to the geniculate ganglion. The nasopalatine nerve (the larger of the posterior nasal branches) passes across the roof of the nasal cavity to the septum and runs inferiorly and anteriorly on the inclined edge of the vomer, supplying the septum. At the incisive canal, the two nasopalatine nerves descend to the palate, communicate with the terminals of the anterior palatine nerves, and consequently participate in the innervation of the upper central incisors. Finally, the pharyngeal nerve leaves the SPG posterior part and enters the pharynx via the palatovaginal canal with a pharyngeal branch of the maxillary artery (MA) to reach the sphenoid sinus and nasopharynx behind the auditory tube[15](Figure 2).

SPHENOPALATINE GANGLION BLOCKADE (SPGB)

Sphenopalatine ganglion blockade has been achieved with a variety of methods, for various pain disorders, when more conservative treatments have failed. In 1908, Sluder proposed that a high-grade inflammatory reaction in the posterior ethmoid and sphenoid sinuses may be involved in certain cases of unilateral facial pain associated with lacrimation, rhinorrhea, and mucosal congestion. These symptoms are associated with the extension of inflammation or the transmission of toxins into the SPG.[16] Sluder reported that patients who had refused surgery for an active ethmo-sphenoid inflammation later developed sphenopalatine ganglion neuralgia (SPGN). He also claimed success in relieving the symptoms of facial neuralgia, asthma,[17] earache,[18] and lower-half headache[19] using ablation. Ruskin described

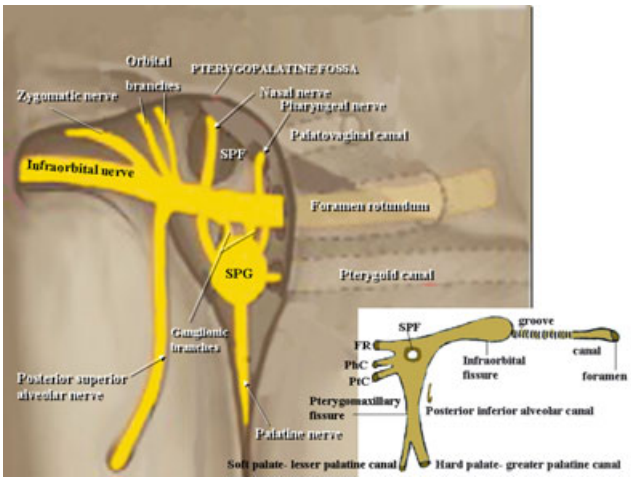

Figure 2. The sphenopalatine ganglion and its connections—communications of the PPF. SPF, sphenopalatine foramen; FR, foramen rotundum; PhC, pharyngeal canal; PtC, pterygoid canal; PPF, pterygopalatine fossa.
Source: Adapted from Google pictures database (95 1252 pair.-com).

the technique with cocaine for the treatment of chronic muscle spasm,[20] arthritis,[21] and polyarthritis,[22] and Ruskin A.P. used pentocaine for a variety of other conditions.[23] Barre[24] described a SPGB method using a dropper to instill local anesthetic into the nose, but much of the anesthetic was absorbed throughout the nasopharynx without reaching the SPG. Hardebo and Elner[25] reported that SPGB can be used as an abortive agent for cluster headache (CH). Although SPGB was historically employed for various pain syndromes, many of these reports are anecdotal and remain controversial.[6,26–28]

INDICATIONS FOR SPGB

Diagnostic and therapeutic head and neck blocks can also assist with the diagnosis and management of many chronic pain conditions (*pain of musculoskeletal, vascular, and neurogenic origin*).[29] Currently accepted indications for the SPGB include: SPG and trigeminal neuralgia (TGN), PIFP (*previously referred to as atypical facial pain*), acute migraine, acute and chronic CH, herpes zoster involving the ophthalmic nerve, and a variety of other facial neuralgias[12,29–31]

Sphenopalatine ganglion neuralgia or Sluder's neuralgia (SPGN) is a type of facial neuralgia, defined as a symptom complex consisting of neuralgic, motor, sensory, and gustatory manifestations.[32] SPGN could be a trigeminal autonomic cephalalgia (TAC), described as unilateral facial pain in the orbital region that sometimes spreads retro-orbitally toward the zygoma and extends posteriorly to the mastoid and occiput.[33] SPGN refers to intermittent episodes of vasomotor hyperactivity causing conjunctival injection, lacrimation, serous nasal discharge and unilateral nasal mucosal inflammation, and sensory disturbances of the palate and oropharynx with distorted gustatory sensations. Occasionally, SPGN is a bilateral constant pain with exacerbations or a pain that stopped and reappeared cyclically or with stabbing sharpness. The pain is typically associated with anesthesia of soft palate, pharynx, tonsils and nose, and hyperesthesia along the distribution of the *TN*. The parasympathetic signs are ipsilateral lacrimation, conjunctival injections, nasal obstruction, rhinorrhea, serious nasal discharge, or mucosal congestion[34,35] (Table 1).

Trigeminal neuralgia, also called "tic douloureux," is a peculiarly painful paroxysmic disorder that undergoes spontaneous remissions and recurrences. The pain, subserved by large fibers, can be triggered from outside the trigeminal territory and by other factors than mechanical stimuli. There are strong autonomic influences of the pain, and there is a cutaneous vasoconstriction in the trigeminal territory in which it

Table 1. Sphenopalatine ganglion blockade (SPGB) in the Treatment of Sluder's Neuralgia

Authors	Patients	Technique	Efficacy
Salar et al.[35]	7	Percutaneous radiofrequency thermocoagulation (RFTC)	1/10 needed repeat lesioning 2/10 needed additional RFTC
Pollock and Kondziolka[36]	1	Stereotactic radiosurgery targeted at the SPG—repeated radiosurgery 17 months later for partial pain recurrence	2 years pain-free (without nasal discharge or eye irritation)
Puig and Driscoll[37]	8	13 occasions of SPG (88% phenol) intranasal	90% decrease in head and face pain (for an average of 9.5 months). The recurrent pain was less severe, less frequent and of shorter duration
Olszewska-Ziaber et al.[38]	3	4% xylocaine intranasally and intranasal phenolization	Total relief of pain
Karas et al.[33]	1	SPG gamma knife radiosurgical ablation	Total relief of pain

occurs.[39] TGN manifests as severe disabling facial pain unilaterally either episodic or chronic and is most common in older people than 50 years of age and in women.[40,41] Pain paroxysms can be precipitated by nonpainful stimuli (touch, movement, wind exposure, chewing, brushing teeth, shaving, and swallowing) in 1 or more branches of the 5th cranial nerve.[13,41] The pain is of much shorter duration than that of CH, but sometimes, it can be associated with CH giving rise to a "cluster tic." Atypical TGN encompasses various facial pain syndromes including those arising from dental or otorhinolaryngological disorders. Paroxysmal hemicrania (PH) belongs to TACs with severe strictly unilateral pain attacks localized to orbital, supraorbital, and/or temporal sites accompanied by 1 or more ipsilateral autonomic features.[40,42] SPGB could be considered a reasonable alternative in drug-resistant PH cases where indomethacin is contraindicated. Several treatment options are available (Table 2).

Cluster headache (CH) is relatively uncommon and the most painful form of recurring primary neurovascular headache. CH has been described with a variety of names, including histamine cephalgia (*Horton headache*) and paroxysmal nocturnal cephalgia.[49] CH is a severe, strictly unilateral head pain (*ocular, frontal, and temporal*) in the territory of the TN distribution, associated with autonomic manifestations homolateral to the pain and usually follows circadian and circannual patterns. The autonomic manifestations seem to be both parasympathetic (*lacrimation and rhinorrhea*) and sympathetic (*ptosis and miosis*). The periodicity of the attacks and seasonal recurrence of the cluster periods suggest the involvement of a biologic clock within hypothalamus.[50] CH is characterized by a constant unilateral orbital location and tends to recur nightly at the same time and last for 6 to 12 weeks, followed by complete freedom from headache for months or even years. The associated autonomic phenomena of a blocked nostril, rhinorrhea, injected conjunctivae, lacrimation, ptosis, miosis, and flushing of the cheek are frequently present. This headache typically responds at least initially to medication. The pathophysiology of CH involves the activation of parasympathetic nerve structures located within the SPG, which explains many of the associated symptoms (*conjunctival injection, lacrimation, nasal congestion, rhinorrhea, forehead and facial sweating, miosis, ptosis, or eyelid edema*).[51] The prominent cranial autonomic symptoms may be due to central disinhibition of the trigeminovascular autonomic reflex by the hypothalamus, possibly through direct hypothalamic-trigeminal connections.[42] Communication between the SPG and the V_2 TN has also been described as a mechanism responsible for CH.[52]

The International Headache Society (HIS) divided CH into groups according to they include: CH periodicity undetermined; episodic; and chronic CH (*unremitting from onset or evolved from episodic*) (*Headache Classification Subcommittee*).[53]About 90% of people with CH have the episodic form, but 10% have chronic CH (*the attacks occur for more than 1 year without remission or with remissions lasting < 1 month*).[42] Attacks occur in CH which follows a chronobiological pattern that appears to coincide with the seasonal change in the length of day. During a cluster period, headaches occur 2 to 3 times daily for

Table 2. Sphenopalatine ganglion blockade (SPGB) in the Treatment of Trigeminal Neuralgia

Authors	Patients	Technique	Efficacy
Manahan et al.[43]	1	10 treatments (bupivacaine 0.5%)	Pain-free for 30 months
Spacek et al.[44]	39	17 patients (carbamazepine and apuncture therapy) (*group A*) 11 patients (carbamazepine local ganglionic opiod analgesia (GLOA) and apuncture therapy (*group B*) 11 patients carbamazepine and GLOA without apuncture (*group C*) GLOA was carried out with 0.045 mg buprenorphine at the superior cervical ganglion or at the SPG	8 patients of group A, 5 of group B, and 2 of group C remained pain-free Most patients with no improvement were from group C
Saberski et al.[45]	1	11 times repeated therapy (transnasal approach)	Bradycardia was resolved with successful alleviation of pain
Grégoire et al.[46]	1	3 separate CT-guided injections	Pain-free
Kanai et al.[47]	25	2 sprays (0.2 mL) of either lidocaine 8% or saline placebo in the affected nostril using a metered-dose spray.	Intranasal lidocaine 8% spray decreased symptoms. Pain was described as moderate or better by 23 patients of the lidocaine spray and 1 of the placebo group.
Zarembinski and Graff-Radford[48]	26	SPGB fluoroscopically on 12 patients that responded to stellate ganglion blocks and 14 additional patients	20 of 26 patients responding to SPGB (76.9%)

45 minutes to 1 hour. The SPG seems to play a very important role in pathology. It is hypothesized that SPGB relieves headaches because it breaks the vicious autonomic cycle of pain within the facial and cranial area[54,55] (Table 3).

Migraine headache is an intensely unilateral, periorbital severe pain. The perception of migraine headache is formed when nociceptive signals originating in the meninges are conveyed to the somatosensory cortex through the trigeminal ganglion, medullary dorsal horn, and thalamus. Different migraine triggers activate a wide variety of brain areas that impinge on parasympathetic neurons innervating the meninges. Migraine triggers either activate or originate in a number of brain areas whose projections converge on the superior salivatory nucleus. The superior salivatory nucleus, in turn, activates postganglionic parasympathetic neurons in the SPG, resulting in vasodilation and local release of inflammatory molecules that activate meningeal nociceptors. Typically, migraine headache is a unilateral pain beginning most commonly in frontotemporal area that may involve whole hemicranium, alternating sides between or during an attack. It is associated with depression, stress, hunger, fatigue, sleepiness, anorexia, nausea, vomiting, photophobia, and phonophobia. Redness and swelling of the mucous membrane of the nose and conjunctival injection may also occur with migraine[70,71] (Table 3).

In recent years, SPG neurolysis after successful blockade has been utilized in such cases with varying success.[34,51,57,58,72] SPG neurolysis is indicated for PIFP,[2] previously known as atypical facial pain. PIFP is defined as a constant, deep, and pulling or crushing pain in the head or face, present all or most of the day, which may become more intense during exercise. Pain is most often localized to the maxilla and may extend to the region of the eyes, nose, cheek, and temple.[73] Patients who have PIFP syndromes (*after trauma, surgery, or on an idiopathic basis*) are more difficult to treat than those with typical TGN.[58] The pathophysiologic mechanism remains unknown, and the cause is likely to be multifactorial.[57] Remick et al.[74] reported a 68% incidence of psychiatric disorders in patients with PIFP, but PIFP cannot be related to a psychiatric disorder. If symptoms make it impossible to determine the pain origin or if they do not guide the clinicians toward any other facial pain syndrome, then the diagnosis is a PIFP.[75] PIFP may have a sympathetic component, which makes neurolysis of the SPG ideal as the postganglionic sympathetic nerves pass through the ganglion. Pulsed radiofrequency (PRF) treatment of the SPG can be considered with a maximal temperature of 42°C for 1 or for more times during a period of 120 seconds.[2]

Head, Neck, Shoulders Pain, and Complex Regional Pain Syndromes[6]

Because the C2, C3, and C4 cervical roots are connected to the SCG and through the DPN to the SPG, the SPGB is able to relieve pain not only from the head, face, neck, and upper back but also diminishes the symptoms from the head and face, which are attributed to the upper cervical spine. The first successful SPGB for myofascial pain originating in the trapezius and sternocleidomastoid muscles was reported by Ruskin.[76] Since then, other authors have asserted potential applicability for SPGB in the treatment of myofascial pain and fibromyalgia[77,78] while others question the therapeutic applicability of SPGB for myofascial pain (Table 4).

Head and Neck Advanced Malignancies

Pain resulting from head and neck cancer can be severe and difficult to manage. In certain patients, SPGB can be effectively self-administered at home to manage chronic pain.[81] Prasanna and Murthy[82] have been used SPGB as an adjunct therapy for immediate relief from pain in the ear arising from the floor of the mouth and extensive tongue lesions because of malignancy (Table 5). Recently, SPGB has been applied for the treatment of musculoskeletal pain, especially that of the neck and back.[28,31]Although various studies with SPGB for management of low back pain have used cocaine,[21] pentocaine,[27] and 4% topical xylocaine[28] with some success, these conditions are not yet considered among the primary indications.

TECHNIQUES FOR SPHENOPALATINE GANGLION BLOCKADE (SPGB)

The techniques for SPGB range from superficial to highly invasive. The drugs frequently used are local anesthetics (*4% cocaine, 2% to 4% lidocaine, or 0.5% bupivacaine*), depot steroids, or 6% phenol. To prolong the SPGB, radiofrequency thermocoagulation (RFT) can be employed. PRF has gained interest as a method. Because it is delivered in pulses, followed by a silent period, allows adequate time for dissipation of heat and energy, resulting in less damage to surround-

Table 3. Sphenopalatine ganglion blockade (SPGB) in the Treatment of Cluster Headache (CH) and Facial Pain

Authors	Patients	Syndrome	Technique	Efficacy
Devoghel[54]	120	CH	Alcohol infiltration	Relief of pain > 85%
Onofrio and Campbell[56]	26	Chronic CH	Posterior fossa trigeminal rhizotomy or percutaneous radiofrequency trigeminal ganglion rhizolysis	54% excellent relief of pain, 15% fair to good relief of pain, 31% poor relief
Cepero et al.[57]	12	Facial pain	SPG neurectomy	High incidence of pain recurrence
Stechison and Brogan[58]	4	Post-traumatic PIFP	Transfacial transpterygomaxillary access to foramen rotundum, SPG, and maxillary nerve	3 patients (complete pain relief) 1 patient free of pain for 12 months 1 patient free of pain for 5 months
Taha and Tew[59]	7	Chronic CH	Percutaneous stereotactic radiofrequency rhizotomy	Long pain relief
Sanders and Zuurmond[51]	66	Episodic CH (56 patients) (ECH) Chronic CH (10 patients) (CCH)	Radiofrequency lesioning in the SPG (infrazygomatic approach)	Complete relief of pain in 34 patients of ECH (60.7%) and in 3 patients of CCH (30%) and no relief in 8 patients of ECH (14.3%) and in 4 of CCH (40%)
Maizels[4]	1	Recurrent headache (migraine with aura)	Intranasal lidocaine 4% during the aura phase	The headache following the aura was prevented and remained successful on all but 2 occasions over 1.5 years
Costa et al.[60]	15	Episodic CH (6 patients) Chronic CH (9 patients)	10% solution of cocaine hydrochloride or 10% lidocaine or saline under anteriorrhinoscopy	All patients responded promptly to both anesthetic agents, with complete cessation of induced pain. In the case of saline application, pain severity increased
Krasuki et al.[61]	15	Severe headache head trauma (7 patients) dental work (2 patients) herpes zoster (2 patients) fibromyalgia (1 patient) pharyngitis (1 patient) migraine (1 patient) unkown reason (1 patient)	SPGB with sterile cotton sticks, soaked in 4% lidocaine, inserted through the patient's nose to the back of nasopharynx for 10 minutes, daily for a week	In 12 of 15 patients, the pain was reduced
Yarnitsky et al.[62]		Migraine	SPGB by applying lidocaine intranasally	Mean pain score was 7.5 of 10 during untreated migraine and 3.5 of 10 after the SPGB. Cutaneous allodynia remained despite the pain relief
Shah and Racz[63]	1	Post-traumatic headache	SPG pulsed-mode radiofrequency lesioning	Pain relief more than 17 months
Bayer et al.[64]	46	Chronic face and head pain (various aetiologies)	SPG pulsed-mode radiofrequency lesioning (procedure was repeated in 20% of the patients)	65% mild-to-moderate pain relief 14% no pain relief and 21% complete pain relief
De Salles et al.[65]	5	Classic unilateral CH (1 patient) neuropathic pain (2 patients) bilateral migrainous neuralgia (1 patient) bilateral PIFP (1 patient)	Radiosurgery of the SPG with a single maximal dose of 90 Gy with a 5- or 7.5 mm circular collimator	Only the patient with CH experienced lasting pain relief
Felisati et al.[66]	21	Chronic drug-resistant CH	Endoscopic SPG via the lateral nasal wall	Treatment was inapplicable a cause nasal stenosis (1 patient) complete disappearance of the attacks (8 patients) a partial benefit (3 patients) no response (9 patients)
Yang and Oraee[67]	1	Chronic, left-sided CH	Topical anesthesia to the nasal mucosa, followed by a needle insertion guided by its tailored plastic cover-sheath for injecting blocking agents	Pain-free for 1 week, 60% pain reduction with less frequent episodes and easily controlled with abortive medicine for 3 weeks
Tepper et al.[68]	11	Intractable migraine	SPGB using the infrazygomatic transcoronoid approach under fluoroscopy guidance	2 patients—pain-free 3 patients—pain reduction 5 patients—no response 1 patient—was not stimulated
Narouze et al.[69]	15	Chronic CH	SPGB via infrazygomatic approach under fluoroscopic guidance	3 patients—no response 7 patients—pain reduction 3 patients—pain-free 2 patients—complete relief of pain

Table 4. Sphenopalatine ganglion blockade (SPGB) in the Treatment of Myofascial Pain (Fibromyalgia and Pain from the Area of Head, Neck, and Shoulders)

Authors	Patients	Technique	Efficacy
Scudds et al.[79]	61	Insertion of 2 soaking pledgets (lidocaine 4% or sterile water) under direct vision, 6 times over a 3-week period	21 patients (35%) decrease in pain (> 30% of their baseline value). Of these 21, 10 had received lid and 11 placebo ⇒ 4% lid is no better than placebo in the treatment of chronic muscle pain
Ferrante et al.[6]	23	4% lidocaine, then TPI with 1% lidocaine, and finally SPGB with saline placebo or SPGB with saline placebo, then TPI with 1% lidocaine, and finally SPGB with 4% lidocaine	SPGB with 4% lidocaine is no more efficacious than placebo and less efficacious than administration of standard trigger point injections (TPI)
Quevedo et al.[80]	2	One culturette swab dipped in 4% tetracaine, in each nare, is advanced gently under the inferior turbinate until it reaches the area of the branch of the SPG. After 10 to 15 minutes, the culturettes are removed	Both patients experienced a 50% reduction in pain level

Table 5. Sphenopalatine ganglion blockade (SPGB) in the Treatment of Other Pain Syndromes

Authors	Patients	Syndrome	Technique	Efficacy
Slade et al.[83]	13	Tear secretion	Topical anesthesia (proparacaine 0.5%, followed by lidocaine 2%)	Tear secretion was reduced substantially or stopped completely after SPGB
Hennerberg et al.[84]	17	Nicotine addiction	Intranasal application of bupivacaine, cocaine, or saline	Significantly fewer symptoms of physical discomfort in the anesthetic treatment groups
Prasanna and Murthy[82]	30	Chronic vasomotor rhinitis	Bilateral SPGB	29 patients—no recurrence of symptoms (follow-up 12 to 20 months) 1 patient—symptom-free (for 8 months)
Robiony et al.[85]	12	Skeletal transverse discrepancy of the maxilla	Transcutaneous truncal anesthesia of the maxillary nerve in association with transmucosal anesthesia of the SPG (lidocaine-prilocaine cream)	The maxillary anesthesia facilitated the operations and reduced the amount of postoperative pain
Hilinsky et al.[86]	1	Post-traumatic pseudo-cerebrospinal fluid rhinorrhea	Restore the normal autonomic homeostasis in the nasal cavity either with topical medicines or with surgical procedures aimed at disruption of parasympathetic preganglionic fibers proximal to or at the SPG	
Varghese et al.[87]	22	Head & neck cancer pain	Nasal endoscopically guided neurolytic SPGB with 6% phenol	17 patients had good immediate relief, 1 patient had partial relief, and 4 patients reported inadequate relief
Prasanna[5]	1	Hyperhydrosis	2 SPGB at weekly intervals	50% reduction in the sweating with the first block, and 90% improvement with the second block. In a follow-up of 12 months, there was no recurrence.
Windsor and Jahnke[30]	1	Eye pain due to herpes keratitis		The patient experienced a month of relief before the pain returned

ing structures.[88,89] More recently, electromagnetic field-pulsed radiofrequency lesioning (EMF) has been used. The current hypothesis on the mechanism of EMF action is that the nerve membrane has a capacitor function and EMF creates a high electric field, which punches holes in the capacitor, thus blocking transmission of stimuli through A-delta and C-fibers.[90]

The Intranasal Approach

The intranasal application of topical anesthetic is the simplest and most tolerable technique. Cocaine or lidocaine is placed on the nasopharyngeal mucosa posterior to the middle turbinate, using a cotton-tipped applicator. The zygomatic arch is taken as a reference, because it is parallel to middle turbinate. However, the topical anesthetic diffusion to the SPG is unpredictable, and the SPGB is not durable.[67] Nose abnormalities can make this route difficult, uncertain, and sometimes dangerous.[11] Several modifications of the traditional intranasal technique have been described. Mingi[91] modified the technique by using an intratracheal cannula, instead of a cotton applicator, for better patient tolerance. Varghese et al.[92] combined both the

principles of external approach for neurolytic and surface SPGB for pain owing to advanced head and neck cancer. Windsor and Jahnke[30] attempted to control the medication amount reaching the posterior nasopharynx as well as provide better needle guidance.[67] The increased danger to the nasal mucosa during needle insertion led to the development of the transnasal endoscopic technique, where the needle is inserted under direct vision, using rigid sinuscope. Recently, Felisati et al.[66] described an endoscopic SPGB technique that approaches the PPF via the lateral nasal wall. Overall, intranasal topical application is not invasive and should be attempted before submitting patients to more invasive surgical approaches. Once a patient experiences good pain relief, repeat blocks are performed before finally deciding on SPG radiofrequency lesioning (RF). A RTFC or EMF procedure can be performed for prolonged analgesia. The use of phenol for neurolysis via this approach is not recommended because of the possibility of inadvertent spread of the phenol into surrounding tissues.[13]

The Transoral Approach (Greater Palatine Foramen Approach)

Injection through the pterygomaxillary fissure has been commonly used by dentists for tooth extractions and routine periapical surgery in the anterior maxillary region. The intraoral route is the most direct, difficult, uncertain, and occasionally impossible to reach the SPG[11] and exhibits the greatest complications. Via this approach, a curved dental needle passes through the GPF at the posterior portion of the hard palate just medial to the gum line opposite the third molar tooth to reach the superior aspect of the PPF[67] where the main trunk of the maxillary division of the TN lays.[93] Paresthesia may be induced because the MN is just cephalad to the SPG. The SPGB via this route is extremely painful for the patient because the needle passes through the canal that contains a nerve trunk, artery, and vein and the anesthetic solution is deposited into a space that has very little compliance.[94] Because of the high vascularity of the region, an intraorbital hematoma may result from injecting local anesthetic in the terminal branches of the MA,[77] which follow the distribution of the terminal branches of the MN. Infiltration of the PPF through the GPF approach may result in infraorbital nerve injury and anesthesia or injury of the orbital nerves.[93] Another significant disadvantage may be the difficulty in correctly identifying the GPF, as the needle may not stop automatically on the sphenoid bone and pass beyond the sphenoid buttress to anesthetize other structures.[67] Because the GPC and hard palate form an angle of approximately 60°,[95] it has been suggested to bend the needle 45° to facilitate the passage of the needle through the GPC and to prevent it from penetrating too far into the PPF. There is no data on standard radiofrequency, EMF lesioning, or phenol injection of the SPG via this approach.[90]

The Infrazygomatic Arch (Lateral Infratemporal) Approach

The infrazygomatic approach is necessary to perform the majority of neurolysis techniques. The infrazygomatic approach has been commonly performed blindly, without C-arm fluoroscopic guidance, but it is highly recommended using fluoroscopy. The approach is carried out by using C-arm fluoroscopic guidance and a direct lateral approach to place a cannula into the superior portion of the PPF without requiring local anesthetic to diffuse across mucous and bony membranes and does not rely on secondary spread via the nerves of the GPC.[3,67]

A transfacial transpterygomaxillary technique in the region of foramen rotundum can be used to block the MN and the SPG. For this, a spinal needle is inserted at the intersection site of a vertical line extending along the lateral orbital wall and a horizontal line tangential to the lateral aspect of the inferior surface of the zygomatic process of the maxilla. The procedure is guided by CT scan to confirm the final needle position or to guide fine adjustments in position. The used CT scan helps avoid inadvertent passage of the needle through the inferior orbital fissure into the intraorbital contents or even the globe. This technique is indicated for PIFP in the MN distribution. The advantage is that allows the needle to be oriented more closely to the axis of the foramen rotundum and the MN.[58]

COMPLICATIONS

The intimate relationship between the SPG and traversing second-division trigeminal branches explains the majority of potential complications following SPG lesioning. SPGB is not a benign procedure, and infection is possible if proper aseptic technique is not followed. Nasal epistaxis can occur if the practitioner is not careful when placing the cotton-tipped applicators

into the nasal passageway or if too much pressure is applied from the needle to the lateral nasal wall.[13] Epistaxis occurs more often during winter months, when forced air heating may cause drying the nasal mucosa. Complications associated with any invasive craniocervical procedure, include hemorrhage, failure to relieve pain, worsening of pain, nontarget nerve injury, blindness, paralysis, stroke, seizures, and death, although the risk of devastating complications in experienced hands is remote.[3] The use of chemical, surgical, cryolysis, and thermocoagulation neurolysis methods may not be the ideal strategy to treat noncancer pain, because of the possibility of damaging vital tissue.[92]

In contrast to the neurolytic procedures, pulsed radiofrequency lesioning (PRF) provides long-term pain relief without destroying neural tissue and has been incorporated into existing percutaneous techniques for craniofacial pain syndromes.[96] In PRF, the impact of electric field on the neuronal cells is well accepted; the high-density electrical field generated around the RF electrode tip is hypothesized to induce changes in the nerve cells responsible for an altered pain signal transmission. In PRF where the electrical current is delivered in pulses, the heat generated is desipated during the silent periods, suggesting that local heat induction may not play a significant role. On the other hand, PRF was found to induce a significant transient inhibition of evoked excitatory transmission, and PRF activity appears to be selective for small diameter (*C and Aδ*) axons that play a primary role in nociception.[88] Radiofrequency lesioning (RF) of the SPG is performed for 70 to 90 milliseconds at 67 to 80°C. The mechanism of action of RF has not been completely resolved. There is some evidence that a heating effect induced by the current may be involved, at least, in a part.[97] However, there is also growing evidence suggesting that the high-density electrical field generated around the electrode may alter neuronal activity and subsequently alter pain transmission. PF can result in permanent or more commonly temporary hypoesthesia, dysesthesia, or differentiation pain in the palate, maxilla, or posterior pharynx.[98,99] Hematoma formation can occur after injury of the MA large venous plexus, which lies within the PPF following intravascular injection.[100] (Figure 3). Cheek hematoma and infection are always a possibility especially if the oral or nasal mucosa was accidentally penetrated. Attention must be paid to the total dose of local anesthetic used if toxic effects are to be avoided.[101]

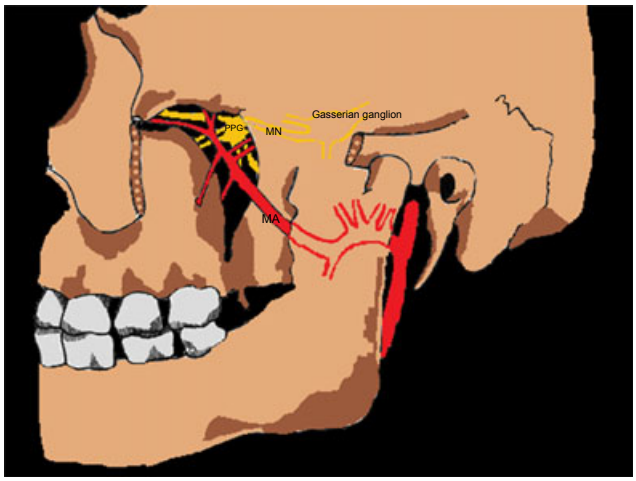

Figure 3. Anatomical lateral view of the pterygopalatine fossa (PPF), which houses the maxillary artery (MA), the sphenopalatine ganglion, and the MN. The (PPG) lies deeply in the fossa. The sphenopalatine artery (SPA) is the end artery of the MA located within the PPF and passes through the sphenopalatine foramen on lateral nasal wall. Nasal bleeding from this artery is potentially life threatening and may urgently require endonasal endoscopic occlusion.[100]
Source: Adapted from Google Database (on line dar-mouth.edu).

Complications associated with SPGB or neurolysis include injury to the MN and MA, perforation of the nasal wall and orbit, bradycardia, hemodynamic instability, toxicity, nasal bleeding, facial hematoma and numbness of the upper teeth, hard palate, or pharynx. Intranasal sensory changes are rarely noticed. These complications may be minimized if sharp needles are replaced with curved-blunt needles and standard neurolysis is replaced with pulsed radiofrequency lesioning.[63] Recently, it has been noted in some patients, reflex bradycardia (*referred to as the "Konen reflex"*) has been observed during radiofrequency treatment of the SPG. In these cases, the lesioning was halted and the bradycardia resolved.[13,102]

SUMMARY

Sphenopalatine ganglion blockade has been performed for the past 90 years. The role of the SPG in the pathogenesis of pain still remains debatable. Although the effectiveness and duration of SPGB can vary, usually depending on the patient, SPGB can be rewarding, safe, and lasting intervention in the treatment of a wide range of environmental sensitivity disorders and illnesses. A thorough understanding of the anatomy allows clinicians to predict correct needle placement and may reduce the incidence of complications. The

search for the precise technique continues, and the currently available evidence should be completed with well-designed trials.

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