

Historical, Pathophysiological, and Therapeutic Aspects of Vidian Neurectomy

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Abstract Vidian neurectomy yields dramatic relief of nasal hypersecretion in patients with allergic rhinitis. Clinical studies conducted on vidian neurectomized nasal mucosa have shown that nasal hypersecretion observed after challenging the nasal mucosa with antigen is caused by reflexively induced activation of the parasympathetic center secondary to stimulation of the sensory nerve terminals in the nasal mucosa by histamine. On the contrary, nasal mucosal swelling is caused mostly by the direct effects of chemical mediators on the nasal vasculature, although vascular reflex mediated by the noncholinergic parasympathetic nerve may be partially involved in the onset of nasal mucosal swelling after antigen challenge. Considering the long-term side effects of inhibition of lacrimation and possible partial recurrence of hyperreactive nasal symptoms observed after vidian neurectomy, less invasive endoscopic posterior nasal neurectomy is considered the treatment of choice for patients with allergic rhinitis who require surgical intervention.

Keywords Vidian neurectomy · Allergic rhinitis · Nasal hypersecretion · Nasal mucosal swelling · Pathophysiology · Nasal reflex

Introduction

Vidian neurectomy was introduced by Golding-Wood [1] in 1961 to relieve severe nasal hypersecretion in vasomotor

rhinitis. Because the clinical effect was dramatic for all symptoms of vasomotor rhinitis, including sneezing, various modified approaches were published by many authors from the 1960s to the 1980s. During this time, it was difficult to differentiate allergic rhinitis from vasomotor rhinitis, and the indications for vidian neurectomy were gradually expanded to include allergic rhinitis. Vidian neurectomized nasal mucosa in which the parasympathetic efferent pathway was cut was valuable in analyzing the role of glandular and vascular reflex in the pathophysiology of allergic rhinitis. In this article, following remarks on the history and anatomy of vidian neurectomy, pathophysiological data obtained from vidian neurectomized nasal mucosa in patients with allergic rhinitis are presented as part of a discussion of the pathophysiology involved in the development of hyperreactive nasal symptoms observed on challenging the nasal mucosa with antigen.

Historical Overview of Vidian Neurectomy

The vidian nerve consists of the presynaptic parasympathetic superficial petrosal nerve, which derives from the superior salivary nucleus; the postsynaptic sympathetic greater deep petrosal nerve, which derives from the glossopharyngeal nerve; and the pericarotid sympathetic nerve. On passing through the pterygoid canal, the vidian nerve emerges to join the sphenopalatine ganglion. The parasympathetic fibers synapse with cell bodies of the ganglion, and the sympathetic component of the vidian nerve passes through the ganglion. Pure sensory fibers from the maxillary nerve also pass through the ganglion. All these fibers form the posterior nasal nerve are relayed to the nasal mucosa through the sphenopalatine foramen. Although parasympathetic innervation of the nasal mucosa and the lacrimal glands is supplied mostly by the

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parasympathetic component of the vidian nerve, sympathetic innervation derives not only from the vidian nerve but also from the periarterial nerve plexuses of the maxillary and ophthalmic arteries.

In 1959, Malcomson [2] reported electrical stimulation of the vidian nerve in cats caused profuse nasal secretion along with nasal mucosal swelling and sneezing. Since the 1950s, vasomotor rhinitis has been thought to result from an imbalance in the autonomic input, particularly from overstimulation of the parasympathetic nervous system of the nasal mucosa. In 1961 and 1962, Golding-Wood [1, 3] introduced transantral vidian neurectomy to relieve profuse rhinorrhea in vasomotor rhinitis. He approached the vidian nerve through Caldwell-Luc's procedure—removing the posterior and some of the medial wall of the antrum to expose the pterygopalatine fossa. In dissecting through the pterygopalatine fossa, the opening of the vidian canal, the vidian foramen, was found to be located at the medial cranial portion of the maxillary surface of the sphenoid. The site of the vidian neurectomy was within the pterygoid canal, aiming predominantly for parasympathetic resection of the nasal autonomic supply.

In 1970 and 1972, Golding-Wood [4] reported long-term results of vidian neurectomy in 185 patients with severe vasomotor rhinitis. Vidian neurectomy yielded complete relief of excessive rhinorrhea in 94% of the patients throughout the 5- to 15-year follow-up. Complete relief was seen in 57% of atopic patients with hyperreactive nasal symptoms. During the 1970s and 1980s, various modified approaches were published by many authors, namely the transantral subperiosteal approach [5], the transpalatal approach [6], the transnasal approach through the septum [7], the transnasal approach through the middle meatus [8], the microscopic transnasal approach [9, 10], and the endoscopic transnasal approach [11–14]. Many publications in the 1970s claimed similar success rates for vidian neurectomy in the treatment of vasomotor rhinitis. However, Krant et al. [15] reported long-term recurrence rates of hyperreactive nasal symptoms as high as 71% during 1- to 6-year follow-up [15]. During the 1970s and 1980s, indications for vidian neurectomy were expanded until this surgical intervention was performed for allergic rhinitis [5, 7, 16, 17].

Short-Term and Long-Term Results of Vidian Neurectomy for Allergic Rhinitis

In 1979, Konno and Togawa [16] reported the changes in nasal allergic symptoms after 1 year of follow-up and also 3–7 years after vidian neurectomy in 28 patients with severe perennial allergic rhinitis who were confirmed to be allergic to house dust and mites [16].

Vidian neurectomy was performed using the transantral approach according to the Golding-Wood method. Because of the nasal obstruction that remained after vidian neurectomy, submucosal resection of the nasal septum and/or submucosal inferior turbinectomy was combined in most cases. However, for the purpose of evaluating the effect of vidian neurectomy alone, only vidian neurectomy was performed in nine cases. Patients were observed for 1 year, and a secondary operation was performed later for the remaining nasal obstruction. At 1 year after vidian neurectomy alone in nine patients, abundant rhinorrhea disappeared completely in eight patients (88.9%) and markedly improved in one patient (11.1%). On the other hand, nasal obstruction disappeared in two patients (22.2%), markedly improved in one patient (11.1%), and moderately improved in five patients (55.6%). No effect was observed in one patient (11.1%). Sneezing improved markedly but did not disappear completely in four patients (44.4%), improved moderately in four patients (44.4%), and no effect was observed in one patient (11.1%).

At 3–7 years after vidian neurectomy combined with submucosal resection of the septum and/or submucosal inferior turbinectomy in 28 patients, rhinorrhea remained relieved completely in 12 patients (42.9%), markedly improved in nine patients (32.1%), moderately improved in five patients (17.9%), and recurred completely to preoperative level in two patients (7.1%). The time of recurrence of rhinorrhea was within 1–3 years after vidian neurectomy.

The precision and efficacy of neurectomy were evaluated by Schirmer's I and II method. Immediately after the operation, lacrimation stopped in all patients. However, 1 year after the operation, lacrimation of more than 10 mm was observed by Schirmer's I method in about 50% of patients. The same tendency was observed by Schirmer's II method, which suggested that reinnervation occurred relatively early after the operation.

As for nasal obstruction, it remained relieved or markedly improved in 24 patients (85.8%), moderately improved in three patients (10.7%), and recurred completely in one patient (3.6%). Sneezing remained relieved or markedly improved in 15 patients (53.6%), moderately improved in eight patients (28.6%), and recurred to preoperative level in five patients (17.9%). Patients were asked to express their degree of postoperative satisfaction as very satisfied (25%), satisfied (36%), comparatively satisfied (25%), or dissatisfied (14%). The most frequent postoperative complaints were abnormal lacrimation observed in four patients and paresthesia of the cheek due to antrostomy. Three patients reported at least one of the following complaints: dry sensation in the nose, dry sensation in the eye, or rhinorrhea on consumption of hot food.

Pathophysiology Involved in the Development of Hyperreactive Nasal Symptoms in Allergic Rhinitis

Nasal Hypersecretion Observed on Antigen Challenge

Effects of Vidian Neurectomy on the Amount of Nasal Secretion Induced by Antigen Challenge

Abundant rhinorrhea observed on challenging the nasal mucosa with antigen in patients with allergic rhinitis may be induced in one of two ways. The first possibility is via stimulation of the sensory endings by histamine released from mast cells, which causes the reflexive activation of the parasympathetic center and results in release of acetylcholine from the parasympathetic terminal at the nasal glands. The other possibility is via the direct effects of various chemical mediators on the nasal glands or on the nasal vasculature, resulting in glandular activation or vascular leakage. Clinically, vidian neurectomy in individuals with allergic rhinitis can produce dramatic relief of hyperreactive nasal symptoms, particularly hyperrhinorrhea, although regeneration of the nerve may cause recurrence of symptoms in 1–3 years.

We evaluated the effects of unilateral vidian neurectomy on nasal secretion induced by the ipsilateral antigen challenge in seven patients with severe perennial allergic rhinitis due to house dust and mites. The operation was performed only unilaterally, and the surgeon paid special attention not to injure the sphenopalatine ganglion and the sensory fibers from the maxillary nerve. The contralateral nasal cavity was used as a control. Nasal provocations with antigen, histamine, and acetylcholine were performed 2 weeks before vidian neurectomy and again 3–5 weeks after neurectomy. Nasal provocation with antigen that lasted for 10 min was carried out by placing an antigen disc (house dust, 2–5 µg protein nitrogen, 7–12 µg total nitrogen per disc; mite 2.5 µg mite total nitrogen per disc; Torii Pharmaceutical Co., Tokyo, Japan) moistened with a measured amount of physiologic saline on the medial surface of the inferior turbinate mucosa. Nasal provocation with histamine and acetylcholine was performed by applying a filter paper strip soaked with various doses of histamine hydrochloride or acetylcholine chloride on the medial surface of the inferior turbinate; the amount of nasal secretion was measured for 10 min using a suction apparatus specially made to collect and measure the nasal secretion [18].

When the nasal mucosa was stimulated unilaterally with an antigen disc before vidian neurectomy, we could observe nasal secretion from the bilateral nasal cavities, with significant correlation between the amount of ipsilateral and contralateral nasal secretion and also between the frequency of sneezing and the amount of ipsilateral nasal secretion [19]. Nasal secretion from the ipsilateral nasal

cavity induced by antigen provocation on the unilateral nasal mucosa was inhibited almost completely following unilateral vidian neurectomy. However, nasal secretion from the contralateral nasal cavity was not affected by vidian neurectomy. Nasal secretion from the contralateral nasal cavity following unilateral antigen challenge on the vidian neurectomized nasal cavity was completely blocked by topical anesthesia of the vidian neurectomized nasal mucosa using 10% cocaine (Table 1). The same results were obtained when the nasal mucosa was stimulated with 0.02 mL of 0.1% and 0.5% histamine chloride [18].

Effect of Vidian Neurectomy on the Amount of Nasal Secretion Induced by Various Amounts of Histamine and Acetylcholine

The nasal mucosa was stimulated using various amounts of histamine hydrochloride and acetylcholine chloride before and after vidian neurectomy. Nasal stimulation was performed on different but consecutive days. Before vidian neurectomy, 1.17 ± 0.32 mL/10 min of nasal secretion was obtained with 0.02 mg of histamine; the amount of nasal secretion increased according to the amount of histamine applied until the dose of histamine reached 0.2 mg. At that dose, the amount of nasal secretion reached 1.53 ± 0.41 mL/10 min. No obvious increase in the amount of nasal secretion was observed by using higher doses of histamine. Histamine applied to the nasal mucosa before vidian neurectomy was considered to be diluted rapidly and washed out. By contrast, after vidian neurectomy, the amount of nasal secretion obtained was only 0.04 ± 0.03 mL/10 min to 0.08 ± 0.03 mL/10 min, even when the dose of histamine applied was increased from 0.02 mg to 0.2 mg. However, in doses greater than 1 mg of histamine, the amount of nasal secretion increased according to the dose. Nasal secretion was 0.18 ± 0.07 mL/10 min by provocation with 1 mg of histamine, 0.26 ± 0.1 mL/10 min by provocation with 2.5 mg of histamine, and 0.38 ± 0.19 mL/10 min by provocation with 5 mg of histamine (Table 2) [20]. The maximum dose that could be applied to the nasal mucosa after vidian neurectomy was 5 mg of histamine. At this dose, patients frequently demonstrated flushing of the face or the whole body. It seemed that histamine applied to the nasal mucosa after vidian neurectomy was absorbed into the mucosa to a considerable degree without being washed out by the nasal secretions that would normally be formed as a result of neural reflexes.

Effect of Vidian Neurectomy on Albumin Values in Nasal Secretion Induced by Nasal Challenge with Antigen or Histamine

Albumin indicates the degree of plasma leakage into nasal secretions. It is not synthesized by the nasal glands.

Table 1 The effects of unilateral vidian neurectomy on the amount of nasal secretion from the ipsilateral and contralateral nasal cavities induced by unilateral antigen challenge on the vidian neurectomized nasal mucosa in patients with perennial allergic rhinitis ($n=7$)

		Before vidian neurectomy	After vidian neurectomy	
		Antigen challenge	Antigen challenge	Antigen challenge after topical anesthesia using 10% cocaine
Amount of nasal secretion, mL/10 min	Vidian neurectomized nasal cavity	1.593±0.71	0.03±0.02 ^a	0.02±0.01
	Contralateral nasal cavity	1.17±0.52	1.08±0.64 ^b	0.02±0.02 ^c

^a $P<0.0001$ versus before vidian neurectomy^b Not significant versus before vidian neurectomy^c $P<0.0001$ versus before topical anesthesia using 10% cocaine

Albumin was measured after challenging the nasal mucosa with four antigen discs in five patients with house dust and mite allergic rhinitis. Concentrations were 171 ± 64 mg/dL before vidian neurectomy and 4917 ± 618 mg/dL after neurectomy. Plasma albumin values determined simultaneously were 5009 ± 511 mg/dL. Four pieces of antigen disc were used because a sufficient amount of nasal secretion could not be obtained by challenging the nasal mucosa with one antigen disc after neurectomy. Albumin values of nasal secretion obtained by nasal challenge with 0.1 mL of 5% histamine (5 mg) were 592 ± 98 mg/dL before vidian neurectomy and 4938 ± 463 mg/dL after neurectomy. Albumin values in the nasal secretion induced by the use of four antigen discs and by 5 mg of histamine hydrochloride before vidian neurectomy were only 4.4% and 12% of the plasma albumin value, respectively. However, albumin values in the minimal amount of nasal secretion obtained by nasal challenge with four antigen discs and also by 5 mg of histamine hydrochloride after neurectomy were equivalent to plasma albumin values [20]. Therefore, nasal secretions obtained after vidian neurectomy were a result of the direct effects of chemical mediators on the nasal vasculature and leakage of plasma. Histamine could provoke plasma leakage, but a fairly large dose (5 mg) was required. However, the total quantity of histamine contained in the nasal mucosa of eight patients with house dust and mite allergy was only 7.1 ± 3.5 µg/g (net weight) measured by modified Shore's method, and the total amount of histamine in nasal secretions obtained after a 10-min allergen disc provocation in 35 patients was only

53.5 ± 57.6 ng [21]. As a result, nasal hypersecretion observed in allergic rhinitis is considered to be caused by histamine-induced stimulation of the sensory endings distributed in the nasal epithelium, with excessive sensory input to the trigeminal sensory center and reflexive activation of the parasympathetic center. The hyperreactivity of the allergic nasal mucosa may significantly augment the sensory input to the trigeminal sensory center. The direct effects of histamine and the other chemical mediators, which are known to have a much higher potency to induce leakage of plasma than histamine, are clinically insignificant for causing hyperrhinorrhea in allergic rhinitis. This concept was supported by intramuscular injection of atropine sulfate, 1 mg/50-kg body weight, followed by unilateral antigen challenge in 13 patients with perennial allergic rhinitis. Ipsilateral and contralateral nasal secretion were almost completely blocked [21].

Participation of Vascular Reflexes in the Nasal Mucosal Swelling Induced by Antigen Challenge

Nasal mucosal swelling induced by unilateral antigen challenge was evaluated by changes in nasal airway resistance (NAR) using anterior rhinomanometry. Antigen challenge caused statistically significant nasal mucosal swelling in the ipsilateral nasal cavity before and after vidian neurectomy [21], which did not reduce the degree of nasal mucosal swelling. Nasal mucosal swelling induced by antigen challenge is largely due to the direct effects of

Table 2 Amount of nasal secretion induced by various doses of histamine hydrochloride applied on the vidian neurectomized nasal mucosa before and after vidian neurectomy in patients with perennial allergic rhinitis ($n=7$)

		Dose of histamine, mg					
		0.02	0.1	0.2	1	2.5	5
Amount of nasal secretion, mL/10 min	Before vidian neurectomy	1.17±0.32	1.34±0.38	1.53±0.41	1.58±0.53	1.5±0.6	1.57±0.49
	After vidian neurectomy	0.04±0.03	0.05±0.03	0.08±0.03	0.18±0.07	0.26±0.1	0.38±0.19

chemical mediators on the nasal vasculature, which cause dilatation of capacitance vessels and plasma leakage. However, rhinomanometry shows large daily physiologic variations of NAR before and after vidian neurectomy, making it difficult to exclude the participation of a vascular reflex in nasal mucosal swelling.

As mentioned previously, sensory stimulation of the nasal mucosa by histamine in patients with allergic rhinitis causes apparent excitation of the parasympathetic center, resulting in marked nasal secretion. Electrical stimulation of the vidian nerve in sympathectomized animals is known to cause atropine-resistant nasal mucosal swelling [22–25]. Electrical stimulation of the superior salivatory nucleus—the parasympathetic center—in cats increased nasal mucosal blood flow and NAR and enhanced plasma extravasation [26]. The nitric oxide synthase (NOS) inhibitor N-nitro-L-arginine methyl ester HCl significantly blocked the plasma leakage, increase in nasal mucosal blood flow, and increase in NAR induced by stimulation of the superior salivatory nucleus [27]. Human nasal vasculature is densely innervated with NOS-positive nerves that originate from NOS-positive sphenopalatine ganglion cells [28].

We studied the extent to which a centrally mediated vascular reflex contributes to nasal mucosal swelling during the immediate-phase reaction after unilateral nasal antigen challenge. Nasal volume measured by acoustic rhinometry and nasal blood flow measured by laser Doppler flowmetry were compared between the contralateral and ipsilateral nasal cavities in 20 patients with perennial allergic rhinitis [19]. Ipsilateral and contralateral nasal mucosal swelling occurred in 17 and 13 patients, respectively. Incidence of contralateral nasal mucosal swelling was significantly higher compared with that after control disc challenge ($P<0.001$). Unilateral antigen challenge caused bilateral nasal mucosal swelling in ten patients that lasted for more than 30 min in the ipsilateral nasal cavity, compared with only 15 min in the contralateral nasal cavity. The decrease in nasal volume in the contralateral nasal cavity was 45.3% of the ipsilateral side. Nasal blood flow was significantly increased bilaterally in 14 patients after unilateral antigen challenge ($P<0.0001$). This indicates that a centrally mediated parasympathetic vascular reflex was partially involved in the onset of nasal mucosal swelling after antigen challenge in allergic rhinitis. However, nasal mucosal swelling that persists beyond 15 min is caused by the direct effects of chemical mediators on the nasal vasculature.

Role of Hyperreactivity of the Nasal Mucosa in Development of Hyperreactive Nasal Symptoms

Figure 1 shows the mode of participation of neural reflexes in the development of hyperreactive nasal symptoms in

allergic rhinitis. Hyperreactivity of the afferent pathway of the reflex plays an essential role in augmenting the sensory input to the trigeminal sensory center. Hyperreactivity is intimately related to allergic inflammation, especially eosinophilic inflammation in the nasal mucosa caused by repeated antigen challenge [29–31]. Enhanced reactivity of the nasal mucosa observed in patients with Japanese cedar pollinosis during pollen season decreased to the level of normal control participants 4 months after pollen season [29]. The degree of nasal reactivity in the afferent pathway of the reflex can be evaluated via frequency of sneezing using histamine test.

In contrast, hyperreactivity of the efferent parasympathetic pathway is difficult to evaluate because enhanced nasal secretion after histamine or acetylcholine nasal provocation may be a result of sensory reflex-mediated activation of the parasympathetic center. This was tested in seven perennial allergic rhinitis patients via unilateral nasal challenge with 0.1 mL of 50% acetylcholine chloride (50 mg) before and after unilateral vidian neurectomy. Responses were compared with those of age- and sex-matched healthy control participants ($n=28$). Albumin concentrations induced by 50 mg of acetylcholine increased significantly from 57.3 ± 19.4 mg/dL before vidian neurectomy to 234.5 ± 59.1 mg/dL after vidian neurectomy. The volume of nasal secretion induced by 50% acetylcholine is derived mostly from nasal glands. The flux of nasal secretion was 1.74 ± 0.72 mL/10 min before vidian neurectomy but decreased to 1.11 ± 0.25 mL/10 min after neurectomy. The significant reduction ($P<0.05$) indicates the participation of neural reflexes after acetylcholine provocation. Acetylcholine (50 mg) induced significantly less nasal secretion in healthy control participants (0.45 ± 0.3 mL/10 min [$P<0.01$]) than in allergic rhinitis patients after vidian neurectomy [20]. This may result from postoperative, denervation-related hyperresponsiveness of the nasal glands to acetylcholine or from hyperplasia of the nasal glands due to remodeling. Efferent sympathetic reflexes were not altered because no differences were observed between perennial allergic rhinitis patients and healthy control participants in plasma norepinephrine or NAR induced by cognitive stimulation using mental arithmetic [32]. There was also no difference between these groups in the magnitude of sympathetically mediated nasal vasoconstriction, which was measured as increases in nasal volume observed upon dipping the feet in ice-cold water. However, the nasal vasoconstrictor response to cold water immersion was significantly depressed in patients with vasomotor rhinitis (idiopathic rhinitis) [33]. Participation of topical reflexes, including the axon reflex and ganglionic reflex, shown in Fig. 1 is clinically negligible as far as nasal hypersecretion is concerned, although it is difficult to deny their participation at the onset of nasal mucosal swelling induced by antigen challenge [34, 35].

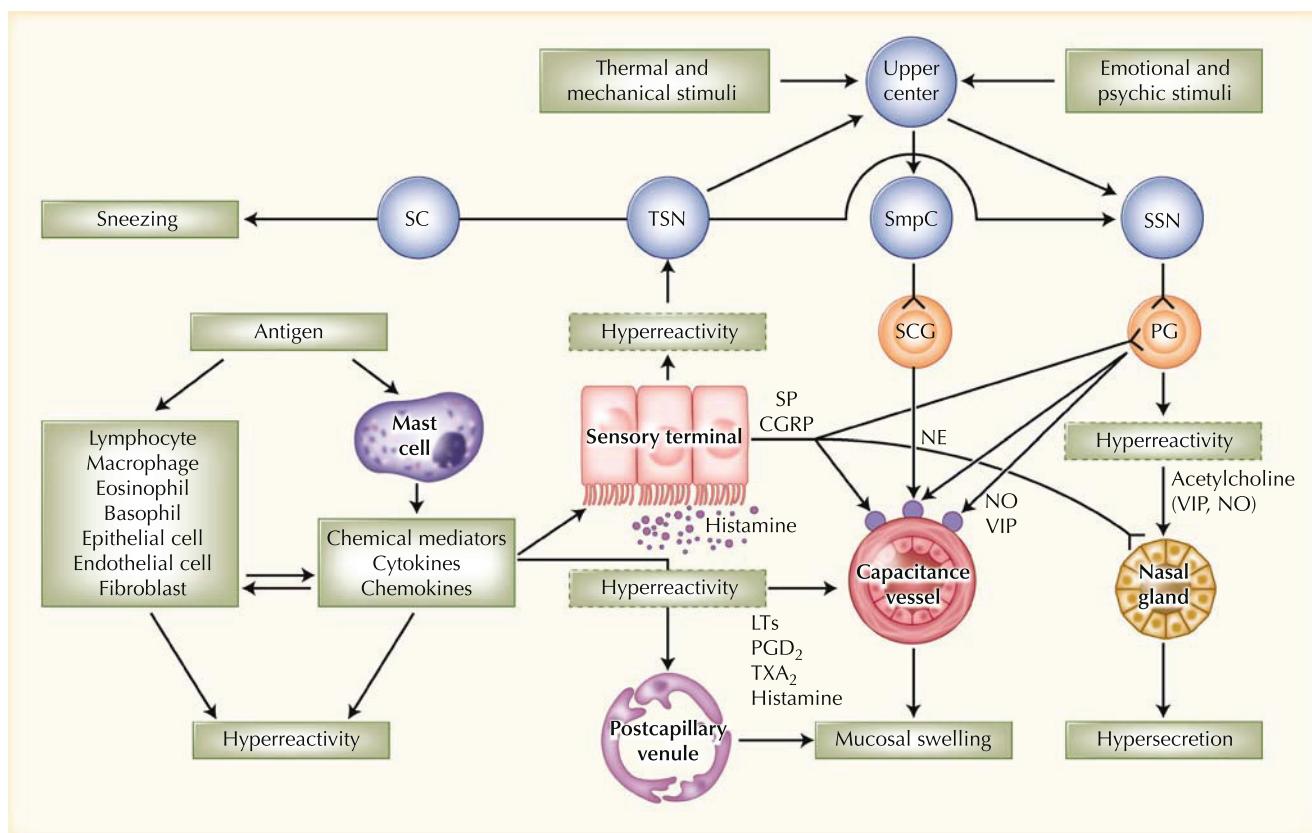


Fig. 1 Diagram illustrating the participation of the neural reflex in the development of hyperreactive nasal symptoms in allergic rhinitis. Hyperreactivity, which characterizes the allergic nasal mucosa, plays an essential role in augmenting the sensory input into the trigeminal sensory center. The effect of vidian neurectomy is caused not only by interrupting the efferent pathway of the parasympathetic reflex but also by lowering hyperreactivity of the afferent pathway induced by injury of the maxillary

sensory fibers during the surgical procedure. *CGRP*—calcitonin gene-related peptide; *LT*—peptide leukotriene; *NE*—norepinephrine; *NO*—nitrous oxide; *PG*—pterygopalatine ganglion; *PGD₂*—prostaglandin D₂; *SC*—sneezing center; *SCG*—superior cervical ganglion; *SmpC*—sympathetic center; *SP*—substance P; *SSN*—superior salivary nucleus; *TSN*—trigeminal sensory nucleus; *TXA₂*—thromboxane A₂; *VIP*—vasoactive intestinal polypeptide

Therapeutic Aspects of Vidian Neurectomy

Judging from the results of these pathophysiologic studies, vidian neurectomy for allergic rhinitis is very effective at relieving nasal hypersecretion. However, regeneration of the parasympathetic nerve fibers may cause recurrence during follow-up periods. Vidian neurectomy is only slightly effective at inhibiting nasal mucosal swelling induced by antigen challenge. Most patients who have severe nasal obstruction refractory to medical treatment still require submucosal inferior turbinectomy or partial resection of the inferior turbinate mucosa. Improvements in sneezing obtained by vidian neurectomy are likely due to damage of the sensory fibers during dissection through the pterygopalatine fossa or sphenopalatine foramen, depending on the approach to the vidian canal. It remains difficult to understand the effect of petrosal neurectomy on sneezing reported by Golding-Wood [1]. More definite effects on sneezing may be expected via coagulation and cutting of the posterior nasal nerve, which is composed of vidian nerve and sensory nerve fibers at the

sphenopalatine foramen. The most frequent long-term side effect of endoscopic transnasal vidian neurectomy is inhibition of lacrimation, which requires the use of tear substitutes. Because of these long-term complications and possible recurrence of hyperreactive nasal symptoms, together with the recent advent of potent topical steroids and second-generation H₁ antagonists, we abandoned the original vidian neurectomy in 1990. For those allergic rhinitis patients who have severe hyperreactive nasal symptoms refractory to medical treatment, Kawamura et al. [36] reported in 2000 on the clinical efficacy of posterior nasal neurectomy combined with submucosal inferior turbinectomy in which neural bundles of the posterior nasal nerve were selectively cut or cauterized at the sphenopalatine foramen using an endoscopic nasal approach, leaving the secretory nerve fibers to the lacrimal glands intact. Ogawa et al. [37•] reported on the long-term clinical effects of this surgical intervention and claimed superb clinical effectiveness that was accompanied by decreases in local inflammatory cell infiltration and the related cytokine production. Indication for surgery for

allergic rhinitis should be considered based on the balance between long-term efficacy and postoperative morbidity. Although this surgery may have the same or higher long-term recurrence rates compared with the original vidian neurectomy, it may offer less postoperative morbidity.

Conclusions

Nasal hypersecretion in allergic rhinitis is caused by reflexive excitation of the parasympathetic center induced via stimulation of the trigeminal nerve by histamine. Vidian neurectomy, which cuts the efferent pathway of the parasympathetic reflex, is very effective at relieving nasal hypersecretion in allergic rhinitis. Hyperreactivity, which characterizes the nasal mucosa in patients with allergic rhinitis, plays an essential role by augmenting the sensory input to the trigeminal center. Injury of the sensory nerve fibers during vidian neurectomy decreases hyperreactivity of the afferent pathway of the reflex, which is effective at inhibiting sneezing and nasal hypersecretion. Nasal mucosal swelling in allergic rhinitis is caused largely by the direct effects of chemical mediators on the nasal vasculature, with only a small contribution by centrally mediated vascular reflexes. Patients who have severe hyperreactive nasal symptoms, including nasal obstruction refractory to medical treatment, may benefit from posterior nasal neurectomy combined with submucosal inferior turbinectomy instead of vidian neurectomy in order to avoid inhibition of lacrimation, the most frequent side effect of vidian neurectomy.

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