

Nonallergic Rhinitis

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KEYWORDS

- Nonallergic rhinitis • Vasomotor rhinitis • Chronic rhinitis
- Irritant rhinitis • Idiopathic rhinitis

Key Points

- A patient with rhinitis symptoms should be fully evaluated from a rhinologic standpoint before nasal surgery is undertaken.
- If diagnosed with rhinitis, the patient should be aware that his or her complaints may not be improved with surgery alone; maintenance medical therapy may be required postoperatively.
- Although a common reason for nonallergic rhinitis (NAR), vasomotor rhinitis is a diagnosis of exclusion.
- Systemic diseases and local factors can contribute to NAR and should be recognized by the facial plastic surgeon caring for her or his patients.
- Patient education and awareness of symptom triggers are key factors in the management of NAR.
- Although there are few medications specifically for NAR, it can usually be reasonably managed with a combination of medical therapy and avoidance measures.
- Allergic rhinitis (AR) and NAR may coexist in the same patient. The facial plastic surgeon should be ready to address these entities.

An estimated 17 to 19 million Americans are affected by NAR.^{1,2} Women seem to be more affected by rhinitis, with 70% of women aged 50 to 64 experiencing some form of rhinitis during a 1-year period.^{3,4} Chronic rhinitis symptoms often interfere with school and/or work performance, and a lack of productivity is worsened by the need for frequent doctor visits. In a recent survey of rhinitis patients, 25% noted restricting their choice of occupation or residence to reduce their symptoms.⁵ In addition, medications—although usually helpful—may elicit undesirable side effects such as drowsiness, epistaxis, palpitations, and nasal dryness, which compound the overall impact of NAR.⁶

NAR and AR have similar presentations, manifestations, treatments, and impacts on school and work performance. Thus, statistics for AR can be used to infer the economic impact of NAR.

In many instances, AR and NAR are often indistinguishable and co-exist.^{1,2} Twenty to 40 million Americans are affected by AR, and the direct costs for doctor visits and medication expenses are at least \$1.9 billion annually. The cost of lost productivity approaches \$3.8 billion annually.⁷

Of the patients who present to the otolaryngologist's office, 50% are diagnosed with a form of NAR, and the rest are diagnosed with AR.^{1,8} In a survey of 975 patients visiting allergists' offices for chronic rhinitis, the National Classification Task Force found that 43% were diagnosed with pure AR, 23% with NAR, and 34% with mixed AR and NAR. Thus, 57% of chronic rhinitis patients have some component of NAR.⁹

Despite the health care burden of NAR, defining it is difficult. Symptom presentation is nonspecific, and patients may present in a variety of ways. Historically, vasomotor rhinitis was the term used

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to describe chronic, NAR; however, motor nerve or vascular dysfunction has not been well-described.^{8,10} Other terms used include perennial rhinitis, idiopathic rhinitis, perennial NAR, and nonallergic, noninfectious perennial rhinitis.^{2,8,11,12} Nonallergic rhinitis is the term used in this article to describe the various forms of the symptom complex of nasal congestion, obstruction, intermittent rhinorrhea as well as occasional itching and sneezing unrelated to an identifiable allergen sensitivity by skin or serum testing.

In many cases of NAR, treatment is often indiscriminate, with varied responses seen among patients. The results are often unsatisfactory and frustrating for both the physician and the patient. Because there have been no unifying criteria for this broad class of problems, further investigation and work-up are generally abandoned by many clinicians, especially since there are no clear diagnostic tests. This article will review a uniform way to describe nonallergic rhinitis in its various forms. The insights into its pathophysiology is briefly reviewed. A classification scheme for the different forms is provided. This is followed by descriptions of the diagnosis, evaluation and management of NAR.

INSIGHTS INTO THE PATHOPHYSIOLOGY OF NAR

Attempting to understand the pathophysiology of NAR requires an appreciation of nasal function. The upper and lower respiratory tracts are lined with pseudostratified ciliated columnar epithelium, which contains goblet cells, ciliated cells, and basal cells. This lining serves in the important regulatory functions for the nose, including the filtration and humidification of inspired air, temperature regulation, olfaction, and preparation of the inspired air for the lower airways.

The mucosa produces mucus secretions that provide lubrication. Additionally, these secretions contain lysozyme, glycoproteins, lactoferrin, and secretory immunoglobulin A, providing protection for the airway. The cilia propel the mucous blanket and their trapped contents toward the natural sinus ostia and toward the nasopharynx.¹³

The mucosa has a rich vascular supply with abundant venous sinusoids. The nasal cycle is comprised of a normal physiologic phenomenon during which time the nasal mucosa will alternately engorge and decongest. This is regulated by the autonomic nervous system, which controls the vasculature as well as glandular secretions.^{8,13}

Sensation primarily originates from the trigeminal nerve. Afferent ethmoidal nerves provide sensory innervation to the epithelium, vessels,

and glands. Unspecified afferent sensory nerves called C-fibers react to pain and changes in temperature and osmolality; they are the most relevant type of sensory fibers in NAR. These sensory fibers are stimulated by inflammatory mediators such as histamine and bradykinin and are involved in centrally mediated reflexes. Once stimulated, C-fibers depolarize, leading to increased vascular permeability and submucosal gland release. There is also acute stimulation of nasal mucosal endothelial cells and epithelial cells, with the resultant sensation of itching and/or burning, and the production of mucoid rhinorrhea.⁸

The sympathetic portion of the autonomic nervous system comprises 50% of the efferent nasal reflex arc, which causes vasoconstriction of the nasal vasculature. The parasympathetic nerves form the other half of the efferent nasal reflex arc, with relaxation of the blood vessels and stimulation of the serous glands of the nasal mucosa. Notably, unilateral stimulation of the efferent reflex arc leads to a bilateral response.^{8,10,13}

In essence, a derangement of any component of the nasal mucosa may lead to the symptoms of NAR. The nonspecific and variable symptoms of NAR are confounding; this compounds the difficult task of identifying the exact pathophysiologic source. Various possible etiologies for NAR have been investigated:

1. Inflammation
2. Hyperreactivity of the parasympathetic limb of the autonomic nervous system and/or hyporeactivity of the sympathetic arm
3. Glandular hyper-reactivity
4. Hypersensitivity of the sensory input via C-fibers.

Recent research on patients with NAR has begun to evaluate the role of mucosal immunoglobulin E (IgE) and inflammatory cell production.¹⁴ A subgroup of patients with NAR has been found to demonstrate an increased local production of IgE upon nasal provocation testing.¹⁵ A separate study indicates that a subgroup of NAR patients, who by definition are not systemically atopic, may display a local allergic disease pathway.¹⁶ This group could possibly be those classically described as possessing NAR with eosinophilia syndrome, or NARES.

In the case of potential autonomic problems, hyper-responsiveness of the afferent sensory limb leads to an exaggerated efferent response with resultant oversecretion of mucus and increased nasal congestion due to capillary plasma exudation. The same symptoms are seen with normal

afferent input and a hyper-reactive efferent arc. Less commonly, an intrinsic epithelial problem or a problem with central nervous system regulation can be the source of disordered responsiveness. Unfortunately, given the complex interaction of sinonasal mucosal regulation, it has been difficult to develop an accurate study model.^{10,17}

The inhalation of irritants is linked to the stimulation of the C-fibers, afferent sensory nerves in nasal mucosa, which innervate the epithelium, vessels, and glands. This results in the activation of the parasympathetic nerves and subsequent sneezing, rhinorrhea, and congestion. This reaction may be induced by histamine and possibly lead to an immunomodulatory response and local cellular events.¹⁸ Glandular hypersecretion can also occur independent of sensory nerve stimulation, as has been shown in methacholine challenge tests.^{12,19} Such findings indicate increased mucosal sensitivity and reactivity in patients with NAR.

Nasal provocation testing using various stimulants has been performed in attempts to characterize NAR. In addition to methacholine, histamine, cold dry air, and capsaicin have all been studied. However, no one study model has provided definitive information regarding the physiologic mechanisms involved in NAR. In many of the studies, the stimulant evaluated induced nasal symptoms greater than seen in controls, but symptom response often overlapped with those patients diagnosed AR. Unfortunately, studies exist with contrary results. Additionally, comparison between studies is difficult, as study methods have not been uniform. Furthermore, provocation studies have shown that upon stimulation, multiple reactions may occur simultaneously (eg, release of inflammatory mediators as well as an exaggerated autonomic response). As a result, there are many examples of how to study the many facets of the pathophysiology of NAR, but there is currently no specific model.^{8,12,19–26}

TYPES OF NAR

NARES

NARES was first described in 1981.²⁷ Patients were described as having perennial nasal complaints, rhinorrhea, epiphora, sneezing, and pruritis typical for AR. Cytologic examination of nasal secretions showed marked eosinophilia. However, the patients lacked immunologic reaction to common inhalant allergens. Most of the patients denied a specific trigger, although some reported reactions to weather changes, odors, or chemical irritants. Other studies demonstrated that mast cells, IgE-positive cells, and eosinophils are increased in the nasal mucosa of both AR

and NAR patients, possibly as a consequence of localized IgE-mediated reactions. In these patients, epithelial damage was seen in the presence of high local eosinophil counts. Nasal neural dysfunction has also been described to contribute to the symptomatology in NARES-type patients.^{14,28}

Hormone-Related Rhinitis

Rhinitis during pregnancy is a probably one of the best known forms of NAR. Rhinorrhea and congestion are prominent, and a review of the patient's history frequently elicits a prior history of chronic rhinitis, either allergic or nonallergic.²⁹ The etiology of this condition is considered to be due in part to direct effects on the nasal mucosa from changes in estrogen, progesterone, prolactin, and placental growth hormone levels.^{30,31} Vascular changes and physiologic expansion of circulating blood volume may also contribute to increased nasal vascular pooling and progesterone-induced vascular smooth muscle relaxation.^{1,30}

Hormones may act directly on the nasal mucosa, causing mucus gland hyper-reactivity and increased rhinorrhea.¹ As such, nasal symptoms may be associated with systemic conditions that result from hormone imbalance such as hypothyroidism and acromegaly.⁸ Rhinitis may arise as a result of changing blood hormone concentrations during puberty. Fluctuating serum hormone levels during menstruation and perimenopause are also associated with nasal symptoms.³²

Medication-Associated Rhinitis

Rhinitis is a common adverse effect of the chronic use of decongestant medications. Obtaining a patient history about medication overuse of topical nasal decongestants and cocaine abuse is critical. Frequent or prolonged use can result in rebound congestion that induces dependency on medication use for relief of nasal airway mucosal obstruction.

Rhinitis is an adverse effect of a myriad of other medications. Medications that affect the vasculature for the treatment of hypertension and cardiac conditions can also affect the nasal mucosa. The suspected patient should be queried about the use of beta-blockers, angiotensin-converting enzyme inhibitors, alpha-blockers, and vasodilators. As mentioned earlier, hormones are associated with nasal symptoms, so a history of the use of hormone replacement and oral contraceptives should also be sought. Psychotropic agents are also associated with congestion (eg, thioridazine, amitriptyline, perphenazine). The use of selective serotonin reuptake inhibitors may be

associated with rhinitis-type adverse effects as well. Nonsteroidal anti-inflammatory medications are well known for their potential association with rhinosinusitis, sinonasal polyps, and asthma.^{8,10}

Irritant Rhinitis

The production of profuse, mucoid rhinorrhea is known to be stimulated by cold, dry air.^{22,33} Facial pressure and headaches as well as nasal symptoms can be brought on by altitude shifts in some patients such as pilots and flight attendants. Alterations in barometric pressure and temperature can be other inciting causes of this form of NAR.

Gustatory rhinitis occurs when the ingestion of foods leads to mucoid or watery rhinorrhea that lasts for as long as the food is ingested.^{1,34} Hot, spicy foods are a common culprit, but sometimes just the act of eating can lead to runny nose. Other nasal symptoms are often absent; however, sweating and epiphora may accompany the reaction. The likely pathophysiologic mechanism is the stimulation of afferent sensory nerves with resultant activation of the parasympathetic nerves supplying the nasal mucosal and sweat glands.³⁴

Tobacco smoke, perfumes, floral fragrances, and cleaning chemicals produce annoyance reactions and are known triggers in patients with a heightened sense of olfaction. Also, some foods and alcohol are triggers for many patients. Patients are often sensitive to multiple irritants that are part of their environment. Air pollution and ozone levels are discussed daily in susceptible areas and times of the year.¹

Occupational Forms of Rhinitis

The occupational form of rhinitis is a special form of irritant rhinitis that represents a complex form of inhalant-induced rhinitis. Various types of chemical exposure have been classified by Baraniuk and Kaliner and consist of immunologic, annoyance, irritational, and corrosive forms.³⁵ Airway reactions can occur with suprathreshold exposure to chemicals and fumes in the workplace. Oftentimes, reactions are a result of exposure to respiratory inhalant irritants beyond threshold levels. Paint fumes, formaldehyde, oxides of nitrogen, and toluene are also examples of this problem.

Although the pathophysiology is not well understood, there is support that many forms of irritant rhinitis are mediated by neurogenic mechanisms, particularly those that are related to chemical exposures. Neurogenic inflammation is considered a pathway model in chemical sensitivity syndromes, including some forms of chronic

rhinitis and asthma. As discussed earlier, the proposed mechanism is the stimulation of irritant receptors on sensory nerves (ie, C-fibers), which induces neuropeptide mediator release. The mediator release then produces vasodilation and edema associated with inflammation independent of immune-mediated inflammation.^{36,37}

Unfortunately, along with typical NAR symptoms, victims of occupational rhinitis may also develop nasal mucosal hyper-reactivity, and problems with an impaired sense of smell, nosebleeds, nasal crusting, and reduced mucociliary function.³⁸ Nasal and oral mucosal contact with high concentrations of soluble chemical gases can cause inflammation, burns and ulcerations, as well as skin and eye reactions. Examples of such corrosive reactions include those associated with exposures to ammonium, chloride, hydrochloric acid, vinyl chloride, organophosphates, and acrylamide.¹

The prevalence of occupational rhinitis is estimated to be 5% to 15%. Nonallergic forms should be differentiated from occupationally induced allergic or immunologic rhinitis.³⁹ Immunologic aeroallergens associated with rhinitis in the workplace include animal proteins, wheat, latex, pyrethrum in the insecticide and garden industries, acid anhydrides in the adhesive industry, and toluene in auto body spray paints. As such, a detailed workplace history is essential.

Idiopathic

A diagnosis of idiopathic or vasomotor rhinitis is given when other identifiable causes of NAR have been excluded. Unfortunately, it is the most common form of NAR. Speculated as an entity more than 50 years ago, vasomotor rhinitis was historically considered to be due to an imbalance of the autonomic nerve supply to the nasal mucosa.^{2,4,11} Studies since then have demonstrated a possible relationship between vasomotor rhinitis and autonomic nervous system dysfunction. Hypoactivity of the sympathetic nervous system relative to the parasympathetic nervous system results in the diversity of local and systemic symptoms that may be seen in vasomotor rhinitis and autonomic dysfunction.^{40,41}

Associated Local and Systemic Problems

Local factors affecting the nose can result in nasal symptoms. Anatomic findings such as the presence of a septal deviation or nasal valve collapse affect nasal airflow and may give a sense of congestion. Hypertrophic turbinates may be a result of chronic rhinitis but also cause physical alterations in the nasal airway. The presence of

a septal perforation alters the natural lamellar airflow of the nasal cavities and may be perceived as congestion or obstruction by the patient.

The nasal symptoms that result from obstruction from inflammatory disease such as polyps or neoplasms are sometimes diagnosed as rhinitis or sinus problems before the correct diagnosis is made. Rhinitis may be the first sign of inflammatory sinonasal disease or infection. Systemic problems such as autoimmune disorders or vasculitides such as Sjogren syndrome, lupus, sarcoidosis, Wegener syndrome, and Churg-strauss syndrome are associated with chronic rhinitis. Congenital problems associated with ciliary dysmotility must also be kept in mind. Cystic fibrosis is also part of the differential diagnosis in chronic rhinitis.¹⁰

HISTORY AND DIAGNOSIS OF NAR

A thorough history of sinonasal complaints often provides the practitioner with the suspicions for the diagnosis of NAR. In addition to acquiring a complete patient history, the practitioner should include extensive questioning with regard to the pattern and timing of symptoms, exacerbating and relieving factors, and environmental triggers. A history indicative of NAR in a healthy patient includes age of onset in mid-30s, a lack of seasonal symptom exacerbation, and negative family history for allergies. A history of symptom exacerbation around perfumes or strong odors lends further support.

However, the practitioner should be aware that many patients may suffer with both AR and NAR. The presence of one form of rhinitis may compound the coexistent condition, resulting in severe and often persistent symptoms. Also, multiple systemic conditions may contribute or result in NAR as mentioned previously. With these possibilities in mind, the practitioner obtaining the history must also pay attention to the patient's past medical history and medication history.

A comprehensive head and neck examination includes the findings as seen on nasal endoscopy. The mucosa is usually boggy and edematous with clear mucoid secretions. Mucosal injection and lymphoid hyperplasia involving the tonsils, adenoids, and base of the tongue may be seen to varying degrees. Areas of blanched mucosa surrounding prominent vessels have been reported in chemical exposures.⁴² The presence of inflammation and purulent discharge from the middle meatus and sphenoethmoid recess indicate active infection. Polyps are an indication of chronic inflammation and obstruction. Atrophy

of the mucosa is seen in cases of aging, prior surgery, and drug abuse. Anatomic obstruction can be diagnosed with the presences of septal deviation, hypertrophic turbinates, and choanal stenosis or atresia. Nasal valve collapse is often overlooked when patients complain of nasal obstruction. The presence of a septal perforation with crusting and epistaxis can be associated with similar symptoms. Examination of the nasal cavity and nasopharynx also helps identify adenoid hypertrophy or unusual mucosal lesions suggestive of sarcoidosis or Wegener syndrome.

Testing for inhalant allergies should be part of the diagnostic work-up in NAR. This can be accomplished via skin or serum testing for specific IgE antibodies to local allergens. Information on cell types and the presence of inflammation is obtained by performing cytologic examination of nasal secretions. Microscopic examination revealing a high power field of 5 to 25 eosinophils in the absence of atopy is compatible with a diagnosis of NARES.²⁷

As mentioned earlier, some forms of chronic rhinitis have also been associated with smell impairment.⁴³ As such, validated smell testing can be a useful addition in the diagnostic work-up and management of patients with NAR. When available, measurements with acoustic rhinometry or rhinomanometry can provide information regarding nasal patency and responses to provocation testing.^{44,45}

Imaging with plain radiographs or computed tomography (CT) scanning is not particularly useful in uncomplicated rhinitis, especially after initial nasal endoscopy is performed. However, CT scans may be useful for evaluating sinonasal inflammatory disease, anatomic obstruction such as the presence of a concha bullosa or polyps, skull base defects, and cerebrospinal fluid rhinorrhea. Magnetic resonance imaging would be useful when a neoplasm is suspected.

TREATMENT OF NAR

Avoidance

Patient education is the most important factor in the treatment of NAR. Frequently, patients cannot immediately recall the specific trigger that incites their symptoms, but a thorough and accurate history can often elucidate the cause of an individual's problem and provides information for the patient. With this knowledge, the patient can take an active role in his or her health care and control his or her environment. Avoidance of inciting factors such as perfumes, tobacco smoke, cleaning supplies, and certain foods or wines can usually be accomplished without great difficulty.

If a change in environment is not feasible, occupational exposures can be eliminated by donning masks and protective coverings. The associated medications can be discontinued or changed. Unfortunately, in many circumstances, exposure to inciting triggers cannot be avoided; no substitute exists for an essential medication, or avoidance measures are not enough to control symptoms. In these cases, medical therapy can be used to supplement the control the symptoms of NAR. The clinician must remember that coexistent AR may complicate the diagnosis and treatment of NAR.

MEDICAL THERAPY FOR NAR

Topical Nasal Steroids

Topical nasal steroids decrease neutrophil and eosinophil chemotaxis, reduce mast cell and basophil mediator release, and result in decreased mucosal edema and local inflammation.⁴⁶ Although the pathophysiology of NAR is incompletely understood, topical nasal steroids are widely used for the treatment of NAR and are noted to be effective.

Optimal medication administration requires propelling the medications into the nasal cavity. Approximately 20% of intranasal steroid preparations are absorbed into the nasal mucosa; the most of the remaining 80% are swallowed and undergo first-pass hepatic metabolism in the portal circulation. Pointing the spray nozzle upward and away from the nasal septum is recommended. Topical steroids are generally tolerated quite well, and adverse effects are infrequently encountered. The most commonly reported adverse effects include anterior septal irritation, nasal dryness and crusts, epistaxis, throat irritation, and headaches. Systemic adverse effects are quite rare. Since the effectiveness of nasal steroids requires contact with the nasal mucosa, anatomic obstruction as well as obstruction from polyps and turbinate hypertrophy may prevent topical steroids from reaching their destination.

Given the findings of eosinophils and inflammation on nasal cytology, NARES is likely to respond well to topical nasal steroids.¹ Fluticasone propionate, beclomethasone, and budesonide are currently the only topical steroids preparations with a US Food and Drug Administration (FDA) indication for treating NAR.⁴⁷ Also, budesonide is currently the only topical nasal steroid that has a pregnancy category of B, whereas all others are category C.⁴⁸ It is recommended that these medications are used on a daily basis for maximum benefit, and a trial should be given for a minimum 6 weeks.

Antihistamines

Azelastine is a topical nasal antihistamine that is approved for use in both AR and NAR. Just like oral antihistamines, azelastine is an H1-receptor antagonist. In addition, studies show that it inhibits the synthesis of leukotrienes, kinins, and cytokines. It also inhibits the expression of intercellular adhesion molecules and the generation of superoxide free radicals. These anti-inflammatory effects, which are unrelated to H1-receptor antagonism, allow azelastine to provide relief in NARES and vasomotor rhinitis. This is felt by the patient as the relief of obstruction and decreased nasal symptoms, mucosal edema, and decreased amounts of rhinorrhea.^{49,50} This medication is generally well tolerated with a low discontinuation rate of 2.3%.⁵¹ Azelastine is a reasonable consideration in those patients with mixed AR and NAR.

Oral antihistamines tend to have a limited role in treating NAR; however, they may be beneficial for patients with NAR who also have sneezing as a component of their symptom complex.⁵² Adverse effects for both oral and nasal antihistamines are related to their anticholinergic properties.⁵³

Anticholinergic Nasal Spray

This class of medications is useful in the management of bothersome rhinorrhea as can be seen in NAR. The nasal preparation, ipratropium bromide, acts locally to block parasympathetic input to the nasal mucosal glands. The 0.03% strength is approved by the FDA for treating AR and NAR. Initially, 2 sprays are given 3 times a day. Symptom control should become apparent within 1 week. When symptoms abate, the dosing can be lowered by 1 spray at a time. One spray 2 times daily is the lowest recommended maintenance dose. It can be used in children as young as 6 years of age. For pregnant women, it carries a class B label. The use of intranasal ipratropium may be associated with local side effects such as nasal dryness and epistaxis. These problems are usually tolerable and their incidence decreased with continued use of the medication or dosage adjustment.^{54,55}

Nasal Saline

Frequent use of nasal saline spray mists throughout the day and the daily use of nasal saline irrigations are useful in managing the symptoms of NAR. This therapy serves to cleanse the nose of irritants, improve mucociliary clearance, and aid in the management of mucoid secretions.

THE ROLE OF SURGERY IN NAR

As mentioned previously, septal deviation, hypertrophic turbinates, sinonasal polyps, and other forms of local anatomic obstruction may contribute to chronic rhinitis and are relative indications for surgical intervention due to nasal obstruction.

Surgery is considered in other cases of rhinitis recalcitrant to medical therapy to address specific symptoms in NAR rhinitis and allow topical medications to reach the mucosa. Little debate exists about the consideration of surgery in chronic sinonasal inflammatory disease that is resistant to medical therapy. Tissue conservation and mucosal-sparing techniques are used wherever possible to retain physiologic function and reduce the risk of chronic problems related with surgery, including persistent disease.

In addition to highly vascular mucosa, the membranes of the inferior turbinates contain venous sinusoids surrounded by smooth muscle fibers under autonomic control; there is also a rich supply of mucous and serous glands.^{13,56} Hypertrophy that is unresponsive to medical therapy can be addressed in a variety of ways: direct steroid injection; submucosal electrocautery; laser ablation; submucosal resection, including the use of the microdebrider; lateral displacement or outfracture; partial turbinectomy; and radiofrequency ablation. The goals are to use conservative approaches to minimize interference with turbinate function and overall nasal health. Various techniques are also available when contemplating a septoplasty, including submucous resection and the endoscopic-assisted treatment of the septal deviation.

Vidian neurotomy is performed to address persistent rhinorrhea resistant to conservative measures. Historically, various approaches have been used: transantral, transpalatal, transethmoid, and transnasal.⁵⁷ Current trends favor endoscopic transnasal approaches. This approach has a lower morbidity rate as compared with other methods.^{58,59} Complications associated with the procedure include decreased lacrimation, mucosal engorgement when supine, dysesthesia, and recurrence of initial symptoms presumably from re-innervation.

SUMMARY

It is easy to wield a diagnosis of vasomotor rhinitis on those patients who lack an immunologic response to inhalant allergens and suffer with chronic nasal symptoms. However, NAR can be the result from a myriad of medical issues (eg,

Box 1

Summary of nonallergic rhinitis

History

- Medical problems
 - Coexistent sinonasal problems
 - Systemic problems with sinonasal extension
- Medications
- Family history
- Environmental triggers
 - Situations
 - Timing
 - Exacerbating/relieving factors

Physical examination

- Full head and neck examination
- Nasal endoscopy
 - Mucosal changes
 - Anatomic findings
 - Presence of a mass lesion

Diagnostic tests

- Allergy testing
- Olfactory function testing
- Cytologic examination of nasal secretions
- CT scan if concern for sinus involvement
- Acoustic rhinometry

Forms of rhinitis

- NARES
- Hormone-related
- Medication-induced
- Irritant
 - Occupational

- Idiopathic

Treatment

- Avoidance
- Medication trials
 - Topical nasal steroids
 - Topical nasal antihistamines
 - Adjunctive medications
 - Nasal anticholinergic sprays
 - Oral antihistamines
- Surgery
 - Anatomic obstruction
 - Turbinate reduction for persistent vascular engorgement
 - Vidian neurectomy

medication usage, hormonal imbalances, systemic medical problems) as well as occupational exposures. Since the facial plastic surgeon often takes a thorough history before performing surgery, she or he will have already gathered much of the information that can point to the diagnosis of NAR. Nasal endoscopy is an important of the examination in evaluating the status of the nasal mucosa and in the recognition of additional problem such as sinonasal polyps or septal deviation.

Patient education is a key factor in managing NAR. Avoidance measures and medical therapy will often take care of the majority of symptomatology and mucosal disease seen in NAR. Sometimes a multidisciplinary approach is required to address the related systemic medical problems. The facial plastic surgeon is aware of the many adjunctive surgical procedures that can be performed at the time of cosmetic or nasal reconstructive surgery. Taking an organized, stepwise approach in addressing the sinonasal symptoms and the underlying problems related to NAR before surgery will aid in positive postoperative outcomes and patient satisfaction (Box 1).

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