Chief Complaint: Nasal Congestion

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Nasal obstruction is the subjective perception and objective state of insufficient airflow through the nose. Nasal congestion, conversely, describes a state of not just inadequate airflow or obstructive phenomena but also pressure- and mucus-related states to the patient. Nasal receptors belonging to the transient receptor potential (TRP) protein family mediate the sense of nasal patency via the trigeminal nerve. The transient receptor potential melastatin-8 (TRPM8) responds to temperatures around 8°C to 22°C, and is stimulated by menthol and other cooling agents. The radiant effects of airflow create heat loss to activate these receptors and humans perceive this as nasal patency rather than the direct detection of airflow. The thermovascular state of the mucosa, in conditions such as rhinitis, influence TRPM8 activation. Nasal endoscopy can show signs of rhinitis and should be considered an essential part of the workup of nasal congestion. Efforts to relieve nasal congestion need to manage the mucosal state and surgery needs to ensures that the nasal cavity mucosa is exposed to the cooling effects of airflow rather than simply creating a passage to the nasopharynx. **© 2024 American Academy of Allergy, Asthma & Immunology** **(J Allergy Clin Immunol Pract 2024;12:1462-71)**

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INTRODUCTION

Nasal congestion is the dominant symptom of many diseases of the upper airway . This review aims to define the terminology, prevalence, and clinical relevance for patients. The anatomy of the inner nose, anatomical structures, and physiological mechanisms involved in nasal patency along with differential diagnosis, diagnostic workout, and therapeutic options are described.

TERMINOLOGY AND PREVALENCE

Nasal obstruction is the subjective perception and objective state of insufficient airflow through the nose. "Nasal congestion," conversely, is a term used to describe nasal turbinate mucosal swelling, caused by dilation of the capacitance vessels in the turbinate tissue and a key component of sinonasal pathology such as rhinitis.[1] However, the term "congestion" is often used to describe both subjective perceptions in mucosal pathology, nasal obstruction, and objective nasal airway measurements, such as nasal airway resistance or nasal airflow.[2] Although nasal congestion can result in nasal obstruction, not all nasal obstruction is caused by congestion. Similarly, the sense of nasal congestion is not always associated with nasal obstruction or airflow restriction.

Nasal obstruction is one of the most common complaints in rhinology practice.[3] It is estimated that nasal obstruction can affect at least 30% of the general population.[4] The 2 most common causes are inflammatory disease and nasal obstruction due to anatomical abnormalities. Most studies of nasal obstruction have been conducted in patients with inflammatory disease, and common conditions are allergic rhinitis (AR), nonallergic rhinitis (NAR), and rhinosinusitis, with AR the most common condition. The prevalence of AR varies across studies, ranging from 10% to 40%.[5] Up to 85% of patients with AR report nasal obstruction, and it is the most problematic symptom in 50% to

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Abbreviations used

AR- Allergic rhinitis
CT- Computed tomography
IgE- Immunoglobulin E
NAR- Nonallergic rhinitis
NHR- Nasal hyperreactivity
NOSE- Nasal Obstruction and Septoplasty Effectiveness
NPIF- Nasal peak inspiratory flow
sIgE- specific immunoglobulin E
SNOT-22- Sino-Nasal Outcomes Test 22
TRP- Transient receptor potential
TRPM8- Transient receptor potential melastatin-8

78% of cases. The incidence of nasal obstruction observed in chronic rhinosinusitis is 66% to 70% of patients.[6-8]

In Australia, the health utility values, a measure of preference-based health-related quality of life used in cost-utility analyses, were studied in patients with nasal airway obstruction. The results show similar health utility values to those in individuals with chronic diseases in the Australian population, including chronic obstructive pulmonary disease, diabetes mellitus, and renal disease requiring dialysis.[9] In 2007, approximately 13 million outpatient visits for the assessment of nasal congestion took place in the United States.[10] The monetary cost of nasal obstruction is significant—approximately 30 years ago, an estimated $5 billion was spent for medical management annually, and another $60 million was spent on surgical intervention.[11,12]

WHEN PATIENTS USE THE TERM "Nasal CONGESTION," IT IS NOT ALWAYS THE SAME AS THE MEDICAL TERM

Symptom descriptors by patients may differ from those used by the medical professional. For a clinician, nasal congestion is the perception of swelling of nasal blood vessels that expand to restrict and sometimes completely obstruct the airflow through 1 or both nasal passages. Nasal obstruction associated with nasal congestion can be distinguished from anatomical obstruction by application of a topical nasal decongestant spray. However, to patients, owing to popular media and commercial marketing replete with references to congestion, it is ambiguous as to what disorder of "nasal congestion" actually is. Advertisements for the common cold, flu, and allergy medication claim to "relieve congestion" via everything from topical and systemic vasoconstrictors through to expectorants, mucolytics, antihistamines, corticosteroids, and herbal preparations. Given the wide range of pharmacokinetic effects of these medications, the symptoms generated are a conglomerate of obstructive, pressure, mucus, and other symptoms.

Importantly, pain and pressure, which are commonly associated with acute upper respiratory events, are perceived as "congestion" and, thus, many conditions that affect facial pressure and pain are perceived as "nasal congestion."

McCoul et al[13] investigated the words and phrases that patients and physicians associate with "nasal congestion," including blockage of the nose; postnasal drip; difficulty breathing; stuffy nose; cold; facial pressure or pain; mucus or phlegm in the throat; cough; clogged ears; mucus or phlegm in the nose; throat clearing; headache; wheezing; runny nose; heaviness in the head; and mucus or phlegm in the chest. The study authors considered 4 categories: obstructive symptoms, pressure-related symptoms, mucus-related symptoms, or other symptoms.[13]

Patients, compared with physicians, more frequently defined congestion as pressure-related symptoms (difference 38.8%; 95% CI 7.5%−70.1%), mucus-related symptoms (difference 51.2%; 95% CI, 22.6%−79.9%), and other symptoms (difference 49.4%; 95% CI 13.7%−85.2%). In addition, 32% of patients associated "headache" with nasal congestion compared with 0% of otolaryngologists and trainees. Other notable disparities included "pressure in face" with 50% compared with 16.1% of clinicians and "postnasal" drip with 38% of patients associating with "nasal congestion" compared with 3.5% of clinicians.[13] All clinicians agreed that obstruction is essential to defining congestion, but a significant proportion of non-otolaryngologists also included terms related to pressure or mucus. For the term "nasal congestion" in this review, we focus on the patient-doctor−agreed terms of "blockage of nose," "stuffy nose," and "difficulty breathing" and synonymous with "nasal obstruction."

THE ANATOMICAL STRUCTURES AND PHYSIOLOGICAL MECHANISMS INVOLVED IN NASAL CONGESTION

A complex vascular structure in the nasal cavity serves to modify the nasal cavity morphology[14] and maintain normal nasal air conditioning. The vascular complex is prominent in the septum (and septal swell body) and inferior and middle turbinate. The complex represents an arterial and venous anastomosis consisting of precapillary resistance vessels, capillaries, venous sinusoids, and venules. The venous sinusoids are interposed between capillaries and venules and act as capacitance vessels.[15-17] The blood flow of the anastomoses is regulated by smooth muscle surrounding the endothelial layer, enabling the resistance vessel and venous sinusoids capacitance vessel to control blood volume according to the state of congestion/decongestion.[18,19] A change in the congestion/decongestion states is largely responsible for nasal airflow resistance.[17] The vascular tone in the capacitance and resistance vessels is influenced by sympathetic innervation. Cholinergic parasympathetic fibers are found around seromucous glands, primarily affecting glandular secretions.

The autonomic nervous system controls the vascular tone and level of congestion. The adrenergic sympathetic pathway stimulation induces vasoconstriction of the arteriovenous anastomoses and collapse of the venous sinusoid capacitance vessel, resulting in nasal airspace volume expansion and perception of nasal patency. Adrenergic receptors are present on the anastomoses and are the target of topical and systemic vasoconstricting decongestants.[20] Conversely, sympathetic tone loss generates an increase in nasal resistance and in the sensation of nasal congestion, as found in patients with cervical sympathectomy and Horner syndrome. Inhibition of sympathetic tone by cardiovascular drugs and medications to manage prostate hypertrophy (α-receptor antagonists) will often result in nasal congestion.

Regulation of this autonomic nervous system plays an important role in the normal physiological nasal cycle. The nasal cycle is a spontaneous phenomenon of cyclic unilateral nasal mucosa congestion due to an asymmetrical venous sinusoid engorgement that alternates from one nasal passage to the other over a period of time.[21] The nasal cycle is presented in

approximately 70% to 90% of adults,[22] but some studies reported a true periodicity exists only in 21% to 39% of the population.[23,24] The nasal cycle periodicity ranges from 25 minutes to 8 hours. During waking hours, the average interval is between 1.5 and 4 hours.[25] In the normal population, the cycle generally goes unnoticed, with unchanged total airflow and resistance, but in patients with nasal pathologies, such as anatomical obstruction or sinonasal inflammation, this alternating obstruction can be detected.

The physiological mechanism of the nasal cycle is still unknown but may be related to fluctuations in autonomic nervous systems. The sympathetic stimulation on one side promotes vasoconstriction, whereas parasympathetic function causes vasodilation and congestion on the contralateral side. Evidence confirms that the nasal cycle is centrally controlled and persists even after total laryngectomy, when nasal airflow ceases.[26] The nasal cycle is affected by changes in blood pressure rate, blood glucose level, age, or positional changes.[25] The purpose of the nasal cycle is thought to be an evolutionary adaptation that allows for optimal sharing of humidification duties, moisturizing, and cleaning of nasal mucosa.[22]

THE HUMAN PERCEPTION OF NASAL BREATHING IS NOT ONE OF DIRECT AIRFLOW DETECTION

Studies have shown that most nasal obstruction surgery is successful in improving nasal airflow.[27] However, despite an improvement in nasal airflow and resistance, objective measurements often poorly correlate with the subjective improvement of nasal airflow.[28,29] This discrepancy explains the report of surgical failure rates being as high as 28% to 33%.[30-32] Contemporary evidence suggests that the primary mechanism of nasal airflow sensation is not airflow resistance but rather mucosal surface cooling, from evaporative radiant airflow, with activation of the trigeminal nerve.

The primary pathophysiological mechanism of the perception of nasal breathing is trigeminal cool thermoreceptor activation. The current theory of nasal sensation was developed based on the understanding of the effect of menthol. It was shown that menthol vapor improves the subjective sensation of nasal airflow without altering nasal resistance.[33-35] The sensation of nasal patency is derived from a cooling of the nasal lining, which is detected by cool thermoreceptors.[36] Cooler nasal lining temperatures (within the receptor activation range) are correlated with the greater subjective perception of nasal breathing.[37-39] The combination of evaporative heat loss and conductive heat loss drives the cooling of nasal mucosa, and this change in temperature or temperature gradient provides nasal patency perception.[40]

The specific receptors stimulated by cold temperature were identified on trigeminal nerve endings,[41] and these cold receptors respond to chemical compounds such as menthol.[42] Cold receptors belong to the transient receptor potential (TRP) protein family. The general role of the TRP protein family is thermosensation.[43] The thermoreceptor transient receptor potential melastatin-8 (TRPM8) is responsible for the cooling signal in nasal perception. The TRPM8 responds to temperatures around 8°C to 22°C, menthol and other cooling agents, such as icilin, eucalyptol, WS-3, lysophosphatidylinositol, lysophosphatidyl choline, and lysophosphatidyl serine. The TRPM8 is predominantly expressed in a primary afferent sensory neuron within the trigeminal ganglia found in the nasal epithelium, mucous glands, and vessels.[44-47]

When high-speed air moves through the nostril and induces evaporation of water from the epithelial lining fluid, the cooling signal is sensed and activated by TRPM8 receptors, causing depolarization of neurons that connect to the brainstem respiratory center, and the cool message is interpreted as a patent nose.[2,48] A normal nasal-cooling effect requires airflow to mucosal surface contact and a normal mucosal vascular condition. Areas of structural abnormalities result in impaired evaporate heat loss, due to diversion of airflow, and a higher local temperature from mucosal inflammatory disease contributing to ineffective nasal cooling activation.[49,50]

A cool stimulus to the nasal mucosa activates primary trigeminal sensory neurons to synapse in the spinal trigeminal nucleus, then secondary neurons cross the midline and ascend via trigeminospinothalamic tracts onto the thalamus and brainstem. The brainstem reticular formation could trigger arousal and cerebral cortex activity, as demonstrated on electroencephalogram and functional magnetic resonance imaging.[51,52] The specific cortical activation areas include somatosensory cortex regions of the rostral insula, which involve sensory and emotional processing; the anterior cingulate cortex area, which relates to decision making;, the insula cortex, and the precentral gyrus of the frontal lobe, which is the motor cortex.[2,48,53] The involvement of the limbic system or emotional processing area highlights the impact of cognitive function and emotional control on nasal perception. Feelings of dyspnea, shortness of breath, air hunger, and inability to breathe are common to anxiety disorders. Such symptoms overlap with those presenting to otolaryngologists with nasal pathology. Anxiety disorders include generalized anxiety disorder, social anxiety disorder, panic disorder, separation anxiety disorder. and phobias.[54] The underlying etiology of conditions, such as empty nose syndrome, may lie more in this emotional processing connection than in aberrations of local anatomy.[55]

WHAT ARE THE DIFFERENTIAL DIAGNOSES OF NASAL CONGESTION?

Longstanding noninfectious rhinitis is broadly classified to be of either allergic or nonallergic etiology but many other conditions may be associated with nasal congestion (Box 1). Allergic rhinitis is an immunoglobulin E (IgE)—mediated event after a sensitized individual is exposed to an offending allergen.[56] Allergic rhinitis is the most common form of rhinitis[57] diagnosed by proving the presence of allergen-specific immunoglobulin E (sIgE) toward aeroallergens. The term "nonallergic" rhinitis is often avoided because several studies have demonstrated positive reactions toward allergens among those with negative systemic allergy tests.[58-61] Some patients thought to have NAR have actually been found to have local AR.[60,61] Furthermore, those with local AR have been found to have similar inflammatory profiles as AR,[60,61] and they respond well to immunotherapy.[62]

Some patients with NAR have an eosinophilic form (nonallergic rhinitis with eosinophilia) that is responsive to intranasal corticosteroids. The NAR with eosinophilia is discussed in more detail in another article in this issue.[63] In contrast, in patients with vasomotor rhinitis or true nasal hyperreactivity (NHR), symptoms are triggered by environmental stimuli such as

Box 1: Common conditions that lead to nasal congestion

- Structural eitiologies: nasal valve dysfunction, septum deformity, tissue remodelling (polypoid change)
- Inflammatory eitiologies: IgE-mediated allergy, type 2 inflammation, occupational delayed-type hypersenstivity
- Neurogenic eitiologies: NHR
- Psychogenic eitiologies: somatic symptom disorder, hyperventilation, anxiety

fragrances, allergens, and changes in temperature. These factors cross the epithelial barrier and are detected by the trigeminal nerve terminals. The NHR is associated with alterations of the 2 divisions of nasal innervation: afferent (somatosensory systems) and efferent (sympathetic or parasympathetic motor systems).[64] Nerve terminals transduce stimuli through multiple molecular mechanisms, such as the activation of TRP cation channels from pain/nociceptive nerves, which leads to nasal sensations such as itch. In addition, this triggers antidromic afferent nerve and efferent reflex mechanisms leading to mucus secretion and vasodilation, ultimately producing rhinorrhea, sneezing, and nasal obstruction.[64]

WHAT DIAGNOSTIC WORKUP IS USEFUL?

Clinical history should focus on the nature or description of nasal congestion including dynamic physiological changes. There is a hydrostatic force of 8 mmHg when positioning from erect to supine.[65] This is exacerbated with lying in the lateral decubitus position and exaggerated in disease in which vascular dilation already exists. This is referred to as "postural congestion." The perception of posutral congestion alongside any "cycling" congestion (ie, from side to side either in positioning or with nasal cycle) and a positive response to decongestant sprays (sympathomimetis or α-adrenergic agonists) are affirmative of the vascular turbinate—based pathophysiological process occuring in the nose. These are Ray rules and often used to help distugish from patients with a heavy mental health, anxiety, or hyperventialation contribution to their sense of nasal obstruction.[66] Other features of clinical history may help with identifying an underlying eitiology (Box 2).

Box 2: Clinical history relevant for assessment of nasal congestion

- Associated allergic/respiratory symptoms: skin (dermatitis/rash/urticaria), conjunctival (itch, redness, periorbital edema), lung (cough, bronchospasm/wheeze, shortness of breath)
- Associated secondary disorders: mouth breathing, snoring, sleep dysfunction, orthodontic abnormalities, fatigue, exercise/fitness impairment, dyspnea, anxiety, hyperventilation
- Exposure risks: smoking, vaping, pollution, intranasal drug use, occupational, and hobbies
- Familial: atopy

WHAT CLINICAL EXAMINATION SHOULD BE PERFORMED?

Nasal endoscopy

Local examination with nasal endoscopy is an essential tool in evaluation of nasal congestion. Nasal endoscopy is a readily available, minimally invasive, and highly useful diagnostic and therapeutic tool in the clinician's armamentarium.[67] It provides a significantly better view of posterior nasal structures than anterior rhinoscopy and has the benefit of being videorecordable to facilitate objective comparisons over time (Figure 1). A strong argument can be made for routine nasal endoscopy in the examination of patients' nose and sinuses.[68] Nasal endoscopy can also be used to assess response to therapies including immunotherapy (Figure 2) and turbinate reduction surgery (Figure 3).

Within this review, middle turbinate (diffuse/polypoid) edema was defined as (middle) turbinate contact with the lateral wall by 2 studies.[69,70] Hamizan et al[71] defined diffuse and polypoid edema of the middle turbinate separately, "Diffuse edema was defined as a translucent, jelly-like mucosal surface occupying the entire leading edge of the middle turbinate mucosa without any intervening normal mucosa. Polypoid edema was defined as a grapelike, translucent protrusion hanging beyond the leading edge of the middle turbinate mucosa" (Figure 4).

Some endoscopic findings do not have a widely accepted (or well-defined) way of being reported. This can lead to endoscopic features being incorrectly reported as present or absent. For example, "watery secretions" was an umbrella term used by this review to describe "rhinorrhea"[72] and "watery discharge."[73] Anecdotally, watery discharge seen in patients with inhalant allergy has been referred to as having a spider-webbed appearance (Figure 1).

Allergy testing

Allergy (sIgE) can be detected by skin testing or *in vitro* serum testing. Clinicians are quick to interpret epicutaneous and serum tests to detect sIgE as the definitive assessment of allergy, but they represent sites distant from the nose (skin and serum). As mentioned previously, evidence has shown that in some patients, sIgE can be locally synthesized in the nose after allergen exposure in sufficient quantities to cause local disease but such IgE may escape systemic detection.[74] Local allergic rhinitis is discussed in another article in this theme issue.[75]

WHAT OBJECTIVE METHODS ARE AVAILABLE FOR MEASURING THE NASAL PATENCY?

A test of nasal congestion should be easy to administer and interpret, and should be cost effective. Some options include nasal peak inspiratory flow (NPIF), rhinomanometry, and acoustic rhinometry.[76] It is important to note that recent exercise, alcohol, and some medications may affect the results of these tests.[77]

Nasal peak inspiratory flow

The NPIF can be measured using a flowmeter mask. Inspiration is recorded with the mouth closed using maximal effort (Figure 5). Values above 90 to 120 L/min are considered normal.[78] The NPIF has been investigated as a tool to measure nasal congestion.[79] In a study of 88 patients with nasal congestion, the accuracy of NPIF change in predicting nasal obstruction that can respond to decongestion was noted to be 75% sensitive and 61% specific for a

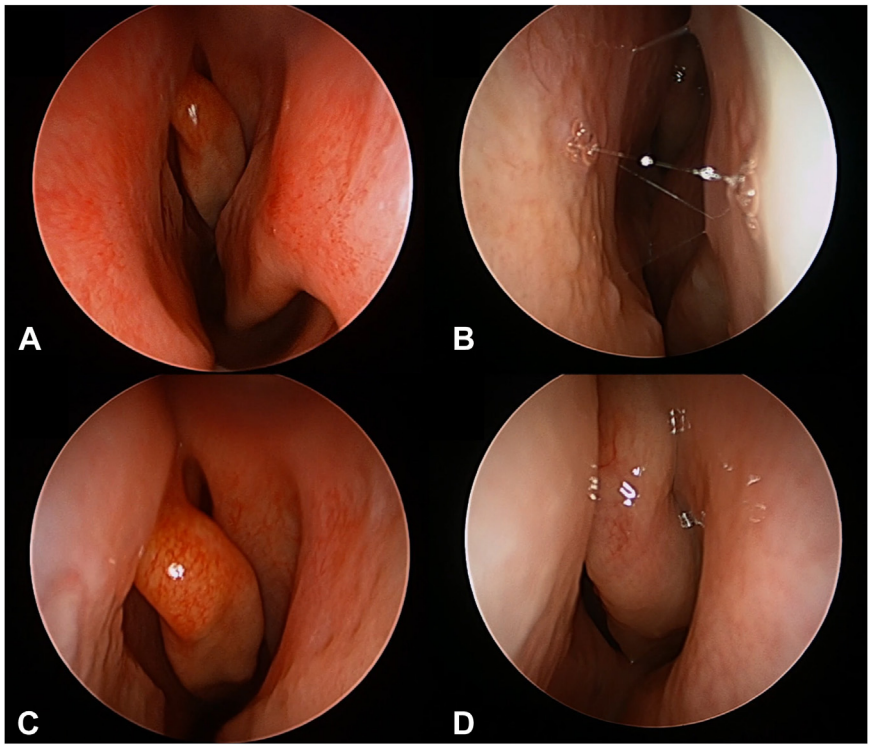

FIGURE 1. (**A** and **C**) The endoscopic image of a left nasal cavity in a normal patient compared with (**B** and **D**) an AR patient. (**B**) There is cobble-stoning of the mucosa and sticky spider-web secretions. (**D**) When examining the left middle meatus of the allergic patient, both middle turbinate edema and "contact" with the lateral wall with occlusion of the middle meatal space are observed.

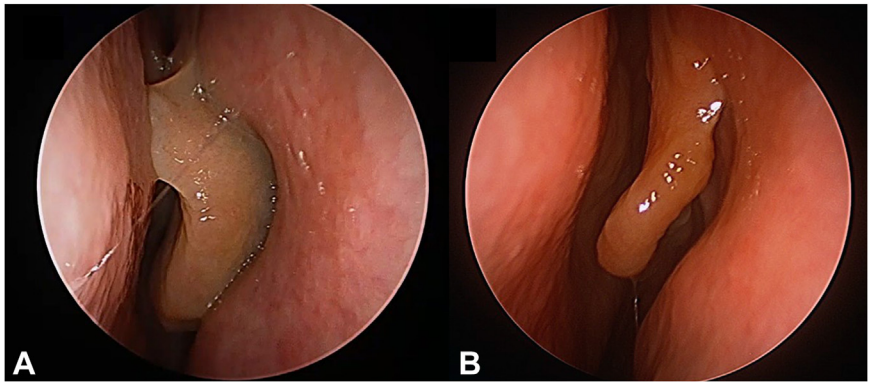

FIGURE 2. (**A**) The most robust sign of inhalant allergy is middle turbinate edema and polypoid change. Airflow through the middle part of the nasal cavity can sometimes be completely occluded by edema and tissue remodelling. (**B**) The same patient, examined after 18 months of allergen immunotherapy, shows a large proportion of the tissue remodelling resolved and airflow across these nasal structures restored.

minimum of 20 L/min improvement in NPIF. The change in NPIF following decongestion was noted to be more appropriate for accurate assessment of nasal congestion, instead of pre- or post-decongestion measurements.[79]

Rhinomanometry

Rhinomanometry can also be helpful to distinguish decongestable obstruction from anatomical obstruction.[80] It is a test of functional breathing and measures airflow during normal, physiological breaths. In this assessment, the patient wears a face mask and pressure can be measured both anteriorly and posteriorly.[77] Collapsibility of the lateral nasal wall may limit this technique owing to turbulence and alterations of expected flow-pressure curves.[28] Rhinomanometry remains the most reliable measure of "nasal congestion" (Figure 6) and is sensitive to changes in the airway from treatment (Figure 7). However, it is not commonly used in routine clinical care.

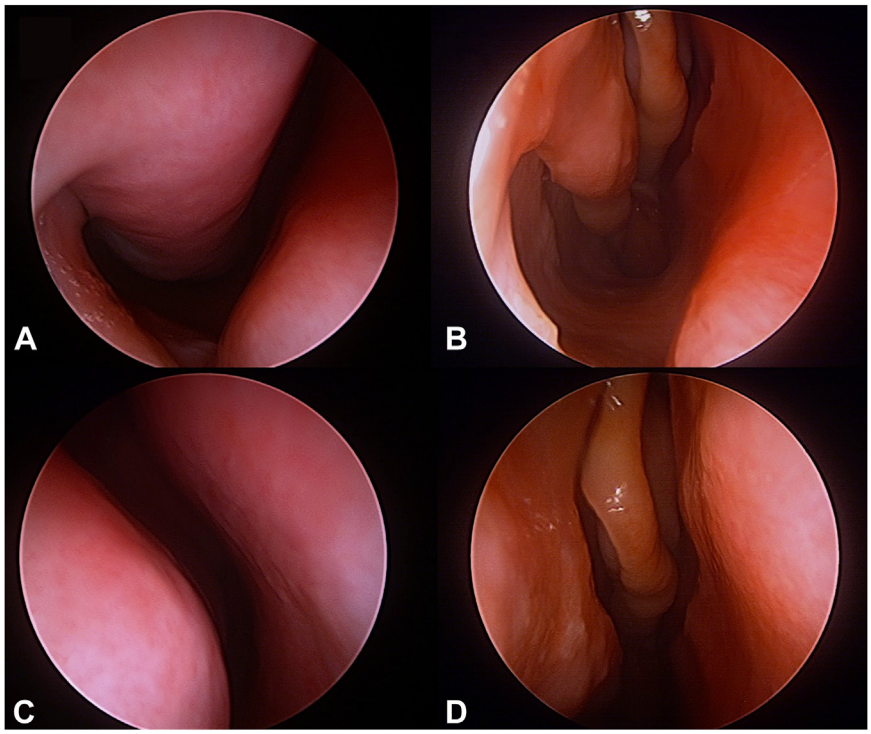

FIGURE 3. (**A** and **C**) The turbinate hypertrophy changes in a 14-year-old male with severe AR after 6 months of allergen immunotherapy. Although other allergic symptoms had improved, the patient had both nasal obstruction and high nasal airway resistance. (**A**) The head of the inferior turbinate is enlarged with loss of the inferior meatal space and (**C**) the septal swell body makes the upper nasal cavity narrow. (**B**) After turbinate reduction, the lower nasal airway is restored, and (**D**) with swell body reduction, the upper nasal airway is likely to benefit from radiant cooling of airflow.

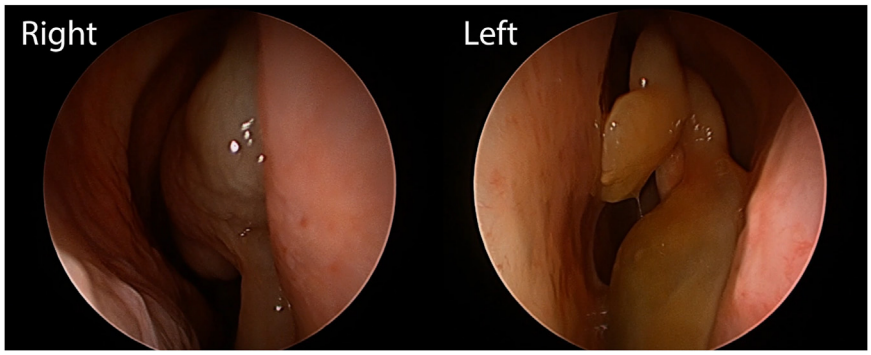

FIGURE 4. (**Right**) In allergic rhinitis, the middle turbinate edema can progress to tissue remodelling that resembles nasal polyps but is just an advanced form of allergic change. (**Left**) The patient's paranasal sinuses are normal here.

Acoustic rhinometry

Acoustic rhinometry is very limited and gives a measure of nasal cross-sectional area only.[77] For this method, sound wave reflections are recorded and analyzed both before and after decongestion. This method identifies anatomical boundaries and the cross-sectional area of the nasal cavity.[28]

The correlation between objective findings from these various techniques and subjective nasal patency is controversial.[28] The topic of nasal congestion and objective measures is ongoing but the gold standard of rhinomanometry remains the best tool.

WHAT ARE EMPIRIC APPROACHES TO INITIAL TREATMENT?

Initial assessment and investigations should ensure that the nasal congestion is limited to the "nasal cavity/passage" disorder and not part of broader paranasal sinus pathology. Single-agent corticosteroid nasal spray or combinations (with azelastine/olapatindine) are commonly utilized and are over-the-counter medications in many countries. A trial or query about the symptom response from decongestant sprays should be sought. Topical capsaicin desensitization has shown efficacy for patients

FIGURE 5. NPIF is measured in the seated position in stable respiratory and environmental condition. The mask is fitted over the face without compressing the nose, and the best of 3 maximal inspiratory efforts are taken as the value in liters per minute. There needs to be less than 10% variability between efforts to ensure consistency of technique.

with NHR[81] or where there is a mix of allergic and hyperreactivity symptoms.[82]

Nonpharmacological and nonsurgical options for the treatment of nasal congestion include acoustic vibration. Incorporation of acoustic vibration is based on reports demonstrating that human humming results in up to a 15-fold increase in exhaled nasal nitric oxide, a molecule known to stimulate mucociliary movement.[83] Medical devices developed to simultaneously apply acoustic vibration and oscillating positive expiratory pressure to the nasal cavity to treat nasal congestion will benefit some patients.[84]

WHAT SURGICAL OPTIONS ARE USED TO MANAGE NASAL CONGESTION?

Surgical modificaitons of the nasal airway are well established to provide relief of nasal obstruction and level 1 evidence exists for septal surgery,[85,86] level 1a for turbinate reduction,[87-89] and level 3a for nasal valve surgery.[90] However, optimal surgical treatment of nasal obstruction relies upon an accurate assessment of nasal anatomy with particular attention paid to structural aspects that may be addressed surgically. Typically, structural causes of nasal obstruction do not respond to medical treatments and tend to have fewer dynamic changes to body position, seasonal/allergic exposures, nonallergic irritants, and other inflammatory triggers. In the absence of chronic rhinosinusitis, clinical evaluation of nasal anatomy in patients with nasal obstruction generally focuses upon 3 sites: (1) nasal septum; (2) inferior turbinates; and (3) nasal valve. The nasal septum is the bone and cartilage that divides the nasal passages into right and left sides. Septal deviations are reported to occur in up to 80% of the population. These deviations vary widely and can include septal spurs or ridges and broad deflections. These deviations can occur anywhere along the septum from the caudal septum/columella posteriorly to the choanae, as well as inferiorly along the nasal floor up to the skull base. This wide variability does make it difficult to reliably quantify the severity of septal deformities and the impact upon sensation of nasal patency. The inferior turbinates are attached to the lateral nasal wall and occupy the inferior portion of the nasal cavity. They consist of bone covered by vascular soft tissue and mucosa. Given the significant soft tissue component, the turbinates are the primary structures that swell with viral, AR, or NAR. For this reason, nasal obstruction is typically treated medically to minimize mucosal swelling and hypertrophy; however, surgery is reasonable when conservative medical therapy fails. Finally, the nasal valve is an often-underappreciated site of nasal obstruction. It consists of the internal nasal valve made up of the nasal septum, upper lateral cartilage, and head of the inferior turbinate. The external nasal valve is made up of the medial and lateral crura of the lower lateral cartilage, caudal septum, and premaxilla. Nasal valve dysfunction can lead to dynamic or static obstruction, depending upon the nature of anatomical deformity. The surgical treatment of nasal obstruction consists of a wide variety of surgical techniques for each anatomical site, thus, this review article focuses upon the highest levels of evidence to provide a broad overview rather than delving into nuances of specific techniques.

A number of septoplasty techniques can be performed depending upon the site of deviation and surgeon preference. As part of the validation process for the Nasal Obstruction and Septoplasty Effectiveness (NOSE) instrument, Stewart et al[91] evaluated 59 patients undergoing septoplasty. There was no correlation between surgeon grading of deviation severity and patient rating of symptom severity or outcomes after septoplasty.[91] Kim et al[92] and Allessandri-Bonetti et al[86] have performed meta-analyses of septoplasty methods and both demonstrated significant improvements in patient-reported nasal obstruction, postnasal drip, and hyposmia. More recently, Van Egmond et al[93] conducted a trial of 203 patients randomized to septoplasty or nonsurgical management. The mean Glasgow Health Status Inventory, NOSE scale, Sino-Nasal Outcomes Test 22 (SNOT-22), and NPIF all favored septoplasty with benefits maintained to 24 months.[85] Once again, deviation severity did not impact outcomes and subsequent economic evaluation[93] demonstrated cost effectiveness.

Turbinate surgery can be done using a wide spectrum of methods including complete or partial resection, submucosal resection of bone and/or mucosa with variable instrumentation, outfracture, or submucosal and transmural ablation with cautery, laser, and radiofrequency energy. The largest meta-analysis of 62 studies[89] demonstrated at least 50% improvement in subjective nasal obstruction up to 5 years with no difference between AR and NAR and benefits in objective measures including nasal airflow and resistance.

Nasal valve surgery aims to correct lateral nasal wall collapse. This surgery is also called functional rhinoplasty and can include both open and endonasal approaches with placement of spreader grafts, alar battens, suspension sutures, and other methods. The largest review of nasal valve surgery by Goudakos et al[90] included 53 studies of 2,785 patients. They examined a variety of objective and subjective outcome metrics with improvement rates ranging from 61% to 100%. Similarly, Zhao et al's review[94] demonstrated that NOSE scores improved by 70% in overall cohort with maintained NOSE benefits of 68% at 12 months. This meta-analysis also looked at NPIF and nasal resistance, showing improvements in both objective metrics.

Clinicians should be aware of newer procedures that are available to patients. A number of less-invasive options for nasal valve dysfunction have been reported. Bioabsorbable implants (Latera) that can be placed in the clinic have been developed to

FIGURE 6. (**A**) Active anterior rhinomanometry is performed by placing a pressure transducer in the contralateral side, fitted and sealed without distortion of the opposite nostril/nasal valve. (**B**) The mask is placed with a seal and without compression of the nose. (**C**) The patient breathes through both inspiratory and expiratory phases with flow-pressure readings recorded via flow within 10% variability. The final values are recorded at 150 Pa and as an airflow resistance at Pa/cm^3/s. The left and right nostrils are recorded separately and used to calculate a total airway (1/R(total) = 1/r(left) + 1/R(right)).

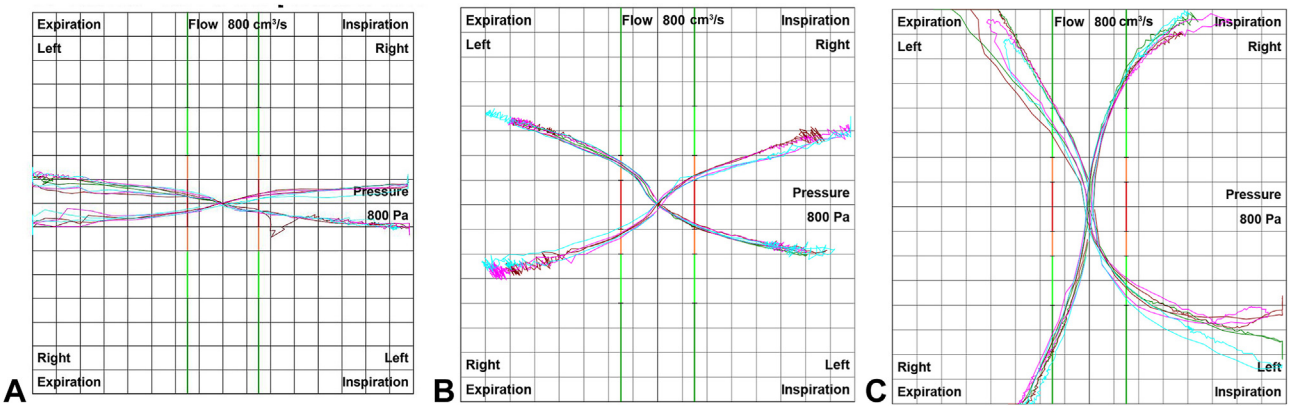

FIGURE 7. The rhinomanometry flow curves for the 14-year-old AR patient seen in Figure 3. (**A**) The baseline untreated nasal airway resistance was 2.07 Pa/cm^3/s. (**B**) After 6 months of allergen immunotherapy the nasal airway resistance drops to 0.75 Pa/cm^3/s but as the upper limit of normal is 0.25 Pa/cm^3/s, the nasal airway resistance is still high and the patient complains of "nasal congestion" unchanged despite clear improvement in other allergic phenomena. (**C**) The postsurgery NAR is now 0.17 Pa/cm^3/s with normalization of the airway and resolution of patient symptoms.

address dynamic collapse of the nasal valve. A systemic review and meta-analysis of this procedure with 396 patients found a mean 43-point improvement on the NOSE questionnaire at 12 months.[95] A second option for nasal valve collapse has been reported using a radiofrequency device with the goal of ablating soft tissue and stiffening cartilage in the lateral nasal wall. A systematic review and meta-analysis of this procedure included 218 patients and found a 46-point improvement in NOSE scores at 3 months[96] with some studies showing benefit to 24 months.[97] The same device can also be used to ablate soft tissue overlying the septal swell body and reduced NOSE scores by 45 points with objective evidence of CT reduction in mucosal thickness.[98] Whereas this method addresses soft tissue overlying the septum, it does not correct deviations of bone or cartilage. Finally, recent methods have been described to ablate the posterior nasal nerve. Although these techniques were originally designed to treat rhinorrhea that is common in AR and NAR, they have also shown benefit in nasal obstruction. The 2 most commonly reported methods are use of cryotherapy and radiofrequency ablation.[99] Both techniques can be performed in the office and have been shown to have benefit in over 80% of patients with follow-up to 2 years.

Nasal obstruction is a common problem and health care providers should be aware that the impact of this condition extends well beyond the nasal cavity. A systematic review of 103 studies of various rhinological disorders[100] found abnormal subjective sleep measures for rhinitis and septal deviation and objective apnea-plus-hypopnea index with mild obstructive sleep apnea across studies. Treatment of these rhinological conditions significantly improved subjective sleep quality across several outcome instruments.

CONCLUSION

"Nasal congestion" is an ambiguous term that needs to be carefully described by the patient if the physician is to understand the pathophysiological nature of the underlying condition. The sense of nasal congestion is not always associated with nasal obstruction or airflow restriction. Because the human perception of breathing is not the direct sensation of airflow, efforts to create

more nasal airway space are not always the path to symptom relief. After screening for functional or conversion disorders, controlling the underlying mucosal pathology and ensuring adequate distribution of airflow over the nasal mucosal surface provide the best possible strategy.

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