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Nasal disorders and sinusitis

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Nose- and sinus-related medical complaints are most common in a primary care setting. The vast majority of office ear, nose, and throat (ENT) problems are benign in nature and have a self-limiting course. However, an obstructed nose, with or without rhinorrhea, may significantly affect lifestyle by compromising comfortable breathing, disrupting normal sleep patterns, interfering with daily activities, and persuading the patient to consult his or her physician for relief. In addition, some patients are prone to frequent, prolonged, and complicated upper respiratory episodes or suffer the symptoms year round. Management of even these difficult patients can be very rewarding for both patient and caregiver because in most cases adequate treatment leads to complete resolution of symptoms, as opposed to other chronic debilitating conditions that compromise a patient’s lifestyle in spite of the physician’s best efforts.

According to the Health Interview Survey, more than 122 million respiratory infections that restrict activity to some degree occur yearly in the United States.1 The actual number of respiratory episodes per person per year varies by age and population studied. Adults average three episodes per year. Young children average six per year. Most sources report that only up to 0.5% of upper respiratory tract infections (URTI) are complicated by a sinus disease.11,17 Nevertheless, it translates to as many as 30 million people per year treated for sinus infection in the United States. These data make sinusitis the leading and probably the most over-diagnosed condition for a number of reasons.

The diagnosis of sinusitis in most cases is based on a clinical impres-
sion only. The lack of a simple diagnostic test similar to a throat culture creates the tendency to label many common colds as sinusitis.\textsuperscript{19,30,31} We assume that a patient who felt sick enough to be evaluated by a physician for URTI deserves a somewhat less common diagnosis than the common cold, for example, sinusitis. Television commercials also promote a sinus brainwash campaign with the frightening pictures of sinus headaches and postnasal drip.

Once the diagnosis of sinusitis has been entertained, the gold standard of care, a full course of antimicrobial agents, is safely offered. Although today it is safer to over-diagnose and over-treat than to be on the opposite side, without a profound understanding of the modern pathophysiology of sinus disease, a stereotyped, purely antimicrobial approach often will fail or achieve a temporary sterilization of the sinuses at best.\textsuperscript{4,5,20,51}

The purpose of this article is to identify the pathophysiologic basis of rhinosinus disease; to introduce the concept that a turbulent course of infections often is caused by a variable degree of mechanical obstruction of the nasal passages and ostia; to identify which pertinent historical data should be obtained; and to emphasize the importance of an adequate focused physical exam, the ancillary tests, and treatment strategies.

The ultimate goal of this article is to enhance the autonomy and diagnostic accuracy of primary care physicians in managing nose and sinus related problems. The author reviews additional therapeutic options and discusses nasal hygiene and the prevention of sinus disease. The indications for referrals to an ENT specialist also are mentioned.

**HIGHLIGHTS OF APPLIED ANATOMY AND PATHOPHYSIOLOGY**

**Nose**

The nose is the most prominent structure on the face; it contributes to the harmony of the facial expression and projects personality to the external world. Someone who does not like the way his or her nose looks may express dissatisfaction with the way the nose works instead. Any deviated nasal structure from the tip of the nose to choanal openings may seriously compromise comfortable nasal breathing.

Grossly, the architecture of the nose resembles a triangular pyramid. Inside, this triangle is split down the middle by a septum; the palate is the floor. The narrow apex is the cribriform plate. The lateral wall has a complex anatomy and a great clinical relevance. Shell-shaped bony projections (inferior, middle and superior turbinates) arise from its surface overlying inferior, middle, and superior meatus respectively. (Fig. 1) The nasolacrimal canal opens into the inferior meatus. The ostia of frontal, maxillary and anterior ethmoid sinuses have their opening in the middle meatus, although the posterior ethmoid cells and sphenoid sinuses drain into the superior meatus. (Fig. 2) The passageways of the nose normally run 10 to 12 cm deep, from the tip of the nose to the posterior wall of the
nasopharynx; they essentially are horizontal parallels to the plane of the palate in spite of the vertical-oblique orientation of the external nose. It is important to provide the patient with this information if satisfactory results from self-administered topical agents are to be obtained. The individual nasal passages measure 3 to 6 mm in diameter, but the patency is remarkably influenced by various factors, including atmospheric conditions such as temperature and relative humidity, hormonal and emotional status, and medications. In addition, there are periods of increased resistance caused by unilateral engorgement of the mucosa, with reciprocal vasoconstriction on the contralateral side called “nasal cycles.” These alternating periods normally vary in time from 20 minutes to 3 hours, so that the total nasal resistance to airflow is constant. The musculature shaping the aperture of the external nares also may affect the airflow by widening and narrowing the external nares. Pinch the tip of the nose lightly to experience how dramatically a flail nasal tip may affect respiration.

The limen nasi or internal nasal valve is formed by the union of lateral and alar cartilages. Located between the nasal vestibule and nasal cavity, the narrowest area of the nasal airway, any degenerative, post-traumatic or postsurgical changes of this fine structure may compromise greatly the ability to breathe with the nose. The nasal valve (Fig. 3) can be seen easily in children by gently elevating the tip of the nose with some illumination; however, in adults, larger vestibules covered by vibrissae makes the nasal valve slightly more difficult to visualize.

Progressively, the lining of the internal nose becomes columnar ciliated respiratory epithelium from stratified squamous epithelium. The complex architectural structure of the internal nose presents a 100 to 200 cm² surface area to the inspired air. The mucosa and the thick, submucosal
Figure 2. Lateral wall of the right nasal fossa with removed parts of the turbinates to show the openings into the paranasal sinuses. (From Callander CL: Surgical Anatomy (ed. 2). Philadelphia, W.B. Saunders, 1941, p 79)

layer of the turbinates contain a large number of goblet cells and seromucous glands respectively; together they produce a mucus blanket covering the surface of nasal passages. The estimated amount of this mucus easily can be up to 1 L in 24 hours.

Ciliary action at a frequency of 700 beats per minute with the transit time of 6 mm/min conveys the mucus (also known as postnasal drip) down the pharynx, where it is swallowed and digested by the stomach. In physiologic conditions, this journey takes 10 to 15 minutes.

The nose has a very generous blood supply, which arrives through the external carotid artery via the maxillary and sphenopalatine branch, and through the internal carotid artery via the ophthalmic artery and its ethmoid branches. At the anterior portion of the septum, under the thin layer of the mucosa, there is a dense capillary network, Kiesselbach’s plexus, which is the most-common site of nosebleeds. The vasculature network of the turbinates closely resembles the cavernous erectile tissue.

In response to the sympathetic and parasympathetic stimuli, the size of the turbinates and, conversely, the size of the nasal passages may vary remarkably. Parasympathetic stimuli through sphenopalatine ganglion cause vasodilatation and increased secretion from the seromucous glands and goblet cells, leading to congestion and rhinorrhea. However, the sympathetic fibers cause vasoconstriction and dryness of the nasal mucosa that are similar to the pharmacologic effects of decongestants and anticholinergics. All of the above-mentioned mechanisms enable the nose to regulate autonomously the amount of inhaled and exhaled air, to purify it from the atmospheric pollution, and to offer a tight control of temperature and humidity levels of exchanged gases.

A relatively limited island of the olfactory mucosa covers the posterior part of the roof of the nasal cavity from the superior turbinate to the opposite septum. Although the olfactory function has lost some importance in the modern era, it still can save lives by promptly detecting noxious gas, smoke, or rotten food. The sense of smell allows enjoyment of
Figure 3. Nasal valve. A = Point of physiological narrowing and increased resistance to nasal air flow.

the natural and more-prevalent synthetic odors surrounding us, and every new episode of the common cold reminds us how precious and delicate this sense is. Providing a patient with some basic data in nasal physiology helps dissipate excessive concerns and can reassure or appropriately alert patients in some instances.\textsuperscript{23,26,27,30}

Paranasal Sinuses

Continuing with the nose there are four paranasal sinuses on each side of the nasal cavity: The maxillary antra, frontal, sphenoid sinuses, and ethmoidal cells, which vary in size and number. Their pathophysiology is directly related to the nasal passages, of which they represent the embryonic extension, share same respiratory lining, and communicate through tiny (1 to 4 mm) ostia.

Our primary focus is the osteomeatal unit (OMU) (Fig. 4), a 1.5- to 2.0-cm-long area located at the level of the middle meatus hidden beneath the middle turbinate of each of the nasal passages. The inferior boundary of the OMU is a bony ridge, uncinate process. Superiorly there is a prominent ethmoid air cell, bulla ethmoidalis and between the two there is a semicircular groove, the hiatus semilunaris, which anterolaterally extends into infundibulum. The latter is the area conjoining the drainage pathways from the frontal, maxillary and anterior ethmoid sinuses into the OMU.

At the posterior aspect of the superior turbinate, there is another area of relative interest, the spheno-ethmoidal recess. This is a root of drainage for the sphenoid and posterior ethmoid sinuses. The mucus then drips down to choanae and the nasopharynx.

Clearance of the sinuses occurs through mucociliary transport (Fig. 5), propelling the mucus against gravity. The fact that ostia of the maxillary sinuses are located high up in the sinus wall is more than just a curiosity, it is a major reason why humans are prone to sinus disorders. The plausible explanation is that paranasal sinuses are vestigial structures,
and the drainage pattern is appropriate for a quadrupedal animal, feeding with the head down. By changing to the upright position, the phylogenetic evolution brought many advantages to the human species, but also disadvantages like frequent low back pain and familiar sinus disease. Any significant anatomical aberration of the lateral wall structures (e.g., an enlarged middle turbinate, septal spur, polyps), or inflammatory swelling of the mucosa (in response to an allergen, infectious agent or noxious physical stimuli) will obstruct tiny sinus ostia and create an insurmountable barrier on the way of mucociliary transport. The inflammation itself increases the production and viscosity of the mucus, while stagnation, low oxygen tension, and lower pH promote the overgrowth of the bacteria that manifests a clinical picture of bacterial sinusitis.

Today’s physiologic relevance of the paranasal sinuses is not well understood, but the following reasonable assumptions can be made.\textsuperscript{1,23,26,27}

Paranasal sinuses
- Reduce the weight of the skull;
- Buffer the impact in case of head injury;
- Contribute to the thermal stability around the brain by amplifying the capacity of the nasal passages in air exchange; and
- Add to the resonance and personal timbre of the voice.

**EVALUATING NASAL OBSTRUCTION**

**Step by Step**

It would be nice to have a patient who provides a focused, pertinent history of experienced symptoms (onset, duration, severity, relation to
Figure 5. Paranasal sinuses with their route of drainage. A = frontal; B = ethmoidal; C = maxillary.

seasons, what triggers them, what relieves them). More often, however, patients come with diagnostic statements like "I have my sinuses," "My allergies are acting up," or "My sinus headache is killing me."

The first, useful distinction in a patient complaining of nasal obstruction is between a physiological variant and disease. Everyone occasionally may have nasal stuffiness or increased nasal discharge in response to an environmental or emotional factor. Also it is not that unusual to have a patient who is annoyed with the aesthetic aspect of her or his nose come with respiratory complaints instead. Some anxious individuals might just be obsessed with their alleged insufficient nose-breathing. More commonly, tension, vascular, or neuralgia headaches are blamed on sinusitis.

In most instances, however, some recent change in nasal function has occurred because prolonged, complete, nasal obstruction results in adjusted, obligatory mouth-breathing. Eventually, it can cause a new bronchopulmonary problem for which medical attention is sought. Finding out that the cause of the respiratory problem resides in the obstructed nose, and then correcting it, provides an excellent service to this particular patient.
The next step is to try to differentiate the cause of the nasal obstruction between infectious and noninfectious, and most commonly, allergic cause. Both the allergic and infectious components of rhinitis may coexist in the same patient.

The presence of constitutional signs and symptoms such as fever, chills, myalgia, arthralgia, lymphadenopathy, productive cough, sore throat, and earache point to infectious causes. Further, lack of a history of allergies or a seasonal trigger, absent sneezing with rhino-pharyngeal ocular pruritus (a cardinal symptom of allergy) may be a sign that there is a significant nonallergic component. If allergies are suspected in a symptomatic patient, the skin test and radioallergosorbent test should be considered early in evaluation.\textsuperscript{23, 26, 36}

**Common Cold**

Acute infectious rhinitis (Coryza) is common enough to be called a common cold. It is a viral illness usually caused by 1 of 110 serotypes of the rhinoviruses, although other agents like coronavirus, parainfluenza, respiratory syncytial virus, and adenovirus might be implicated. Locally in the upper airway, the pathogens trigger an acute inflammatory reaction with the release of vasoactive mediators and increased parasympathetic stimuli that produce congestion and rhinorrhea. The constitutional symptoms probably are secondary to acute viremia. The spread occurs by droplets and close contacts during colder seasons. Children are more susceptible and more symptomatic. The typical ENT presentation initially consists of a dry, burning, scratchy sensation in the nose and throat lasting about a day followed by a catarrhal phase (profuse, watery rhinorrhea, hyperemic, edematous, covered by a transudate nasal mucosa). The catarrhal phase usually lasts for 2 to 3 days. During the following postcatarrhal phase, before the symptoms start to improve, the secretions actually get thicker, adding to the severity of nasal obstruction. A dry and then progressively more productive cough may be associated with it. This mucopurulent phase lasts at least several days before the secretions clear again. This phase might be erroneously interpreted as a bacterial complication of the paranasal sinuses.\textsuperscript{3, 36, 52}

**Considering Sinusitis**

Whether and to what extent during the course of a rhinitis do the sinuses become involved is the next most important question to ask. Any source of prolonged nasal obstruction may lead to bacterial overgrowth of the paranasal sinuses. The data from extensive imaging studies have confirmed that in virtue of the common histologic origins and anatomical contiguity of the nasal and paranasal mucosa, the mucosal thickening or even diffuse opacification of the sinuses is a common finding.
matory changes on CT scan may show up as early as 48 hours of onset of viral URTI. But the viral rhinosinusitis at this stage has little to do with a bacterial sinus infection. The sinus changes seen on imaging studies either are not predictive for a bacterial overgrowth or persist weeks after complete resolution of symptoms. The important message is that the information collected from a patient and his or her body language speaks more distinctly than the data obtained through expensive high-tech equipment.

Any nasal obstruction, including anatomical abnormalities and uncontrolled allergies, may lead to a sinusitis initially characterized by a sterile inflammation with potential tendency to develop a bacterial overgrowth. The clinical correlation and knowledge of a natural course of infection is critical in diagnostic work up and successful treatment.

Acute bacterial sinusitis is a consideration in a susceptible individual with a documented cold beyond the expected 8 to 10 days or a prolonged nasal obstruction. Often the conditions deteriorate after an apparent earlier improvement throughout the course of the same respiratory episode.

Specifically the following symptoms must be present for a patient to be diagnosed with sinusitis:

- Nasal obstruction.
- Mucopurulent nasal drainage that is predominantly anterior with the involvement of frontomaxillary sinuses, and posterior if ethmoidal cells or occasionally sphenoidal sinuses are affected.
- Craniofacial discomfort described as interorbital heaviness with or without a frank periorbital/facial edema, dull facial pain often referred to the upper maxilla, and occasional throbbing pain in the case of sinuses completely obstructed by pus (Characteristically, sinus headaches are reproduced with morning activity; they exacerbate with head movements and gradually subside by afternoon. It is very unlikely for a bacterial sinus infection to present with an isolated headache or dry cough.)
- Impaired sense of smell.
- Cough present during the daytime that is worse at night.
- New-onset fever.
- Fetor oris.

It is critical to obtain basic information such as a history of over-the-counter medications and nasal preparations, exposure to first- and second-hand tobacco smoke, exposure to chemical and physical irritants at home and at work, allergic diathesis, tolerance to physical activity, and hobbies. For example, water sport activities, especially scuba diving, may lead to a barotrauma-induced rhinosinusitis. A clue to chronic rhinosinusitis can be found by a history of significant trauma or surgery to the nose, dental procedures, especially teeth extraction, which may lead to odontogenic sinusitis. Finding out how well preserved the sense of smell is will help to realize the severity of nasal obstruction.

Sinusitis can be categorized based on duration. With acute sinusitis, symptoms last up to 4 weeks. With subacute sinusitis, symptoms last from
4 to 8 weeks and with chronic sinusitis, symptoms last more than 8 weeks nonresponsive to therapy.

Maxillary sinusitis is most common, followed by ethmoidal and then frontal sinusitis. Isolated sphenoidal sinusitis is rare. More likely, ethmoidal and sphenoidal sinuses are involved in the picture of a pansinusitis.

Although the species of micro-organisms isolated from bacterial sinusitis are the same over the years, the insurgence of organisms resistant to commonly-used antibiotics represents an actual problem and may become a true emergency in the near future. The top micro-organisms isolated from acute sinusitis are *Streptococcus pneumoniae* (30%) and *Haemophilus influenzae* (20%). *Moraxella catarrhalis* occurs in up to 20% of sinusitis in children but is less frequent in adults. Occasional micro-organisms include *Streptococcus pyogenes* (in both children and adults), *Staphylococcus aureus* (from adults), *Pseudomonas aeruginosa* (from immunocompromised hosts), anaerobes (6%), and sterile cultures (15%).

The predominant bacterial pathogens in chronic sinusitis include anaerobic pathogens such as the *bacteroides* species, *peptostreptococcus* organisms, *S aureus*, and occasionally gram-negatives, often presenting as mixed flora.

The resistance to antibiotics through the β-lactamase production occurs in virtually 100% of *M catarrhalis* strains and 25% to 50% of *H influenzae*. The β-lactam resistance of *S pneumoniae* develops through structural changes in penicillin-binding protein and ranges from 10% to 50% in different geographic locations and populations studied.

Certainly not every patient presents with a classical, full-blown sinusitis; a judicious and perceptive clinician with good approximation can decide which patient to start on an antimicrobial agent. The indiscriminate use of the antibiotics might not prevent the complications from occurring although they can cause different side effects and immunologic manifestations. More importantly, antibiotics cultivate the resistant bacterial flora. Over the last year, health professionals have become more aware of this dangerous phenomenon. Some guidelines in prescription of antibiotics may be useful to limit over-prescription, and to protect physicians of being accused of under-treatment.

Complications of bacterial sinusitis are rare. They may limit to soft-tissue swelling around the involved sinus secondary to venous flow obstruction, without the invasion of infectious contents. The orbital complications anterior to orbital septum are the most common ones. The incidence of severe complications such as intraorbital abscess or intracranial or bony spread of infections is decreasing, but when they occur, they can lead to loss of vision, neurologic deficit, and a mortality rate of 20%. Therefore, physicians treating sinus infections should have a high level of suspicion for early signs of more serious complications. Lack of response to outpatient medical therapy with exacerbation of symptoms (inflammatory swelling of the orbit or forehead or development of new neurologic signs) should mandate an immediate admission for intravenous therapy and specialist consultation for possible drainage of the infected foci.
Classification of the Less-Common Causes of Nasal Obstruction

Less common causes of rhinosinusitis disease include

1. Nonallergic rhinitis with eosinophilia (NARES) is a condition that clinically behaves like a perennial allergic rhinitis with a nasal smear heavily loaded by eosinophils, but either skin testing or RAST results are negative. A nonidentifiable allergen or pathogenetic mechanism other than IgE-mediated sensitivity is probably implicated. Avoidance strategies and conventional anti-immune therapy cannot be used in these patients.

2. Vasomotor rhinitis, or perennial nonallergic rhinitis (NAR) without eosinophilia, is an idiopathic nasal hyper-reactivity with exaggerated parasympathetic response to a non-well-identified trigger or to physical stimuli such as mild irritants or a change in environmental temperature, that usually does not affect most people. Although it is important to recognize the existence of NARES and NAR without eosinophilia, these conditions are relatively infrequent and have a questionable relevance in a primary care practice. Exclusion of perennial allergic rhinitis, chronic infection, and other types of rhinitis should be considered before making the diagnosis. NARES respond better to the allergy stabilizing and anti-inflammatory agents than the vasomotor rhinitis, which can be challenging to control.

3. Nasal polyps are benign proliferative lesions arising from chronically inflamed mucosa of paranasal sinuses or nasal cavity. They are more frequent in asthmatic individuals and are highly correlated with eosinophilia. Nevertheless, about 75% of people with nasal polyps do not have documented allergies. Polyps appear as smooth, pale, mobile, nontender, often multiple masses. They may cause a significant degree of nasal obstruction often compromising the sense of smell. The majority of patients respond to topical steroids. More severe nasal polyposis is controlled by a combination of medical and surgical therapy, but frequent recurrence of the polyps is a major concern in spite of adequate treatment.

4. Gustatory rhinitis is a nasal secretory reaction to exquisitely spicy foods and is a common phenomenon. Food-induced allergic manifestations limited to the nasal symptoms are very rare, however. Although this phenomenon may be encountered in the pediatric population, children usually do outgrow it.

5. Rhinitis medicamentosa is rebound congestion (vasodilatation with interstitial edema and hypersecretion caused by a prolonged use of pharmacological agents). Some authors limit this definition to side effects from topical decongestants only. Indeed, the majority of people using topical decongestants develop rebound congestion just after several days of application. This situation progressively is getting worse because the vasocon-
stricting effect of the decongestant shortens, while more prominent rebound congestion develops earlier, driving the patient to use the medication more and more often. Once a patient is counseled about the nature of the problem, the management is successful by switching to a short course of oral decongestants. After a few weeks of topical steroid agents the condition reverts back to normal. Rhinitis caused by sniffed cocaine has the same pathogenesis. In advanced stages, mucosa may present as atrophic or crusty with occasional septal perforations. Some authors extend the definition of rhinitis medicamentosa to the oral decongestants and any other systemic drugs capable of causing nasal congestion as a side effect. These include antihypertensive agents such as hydralazine, prazosin, methyldopa, guanethidine, propranolol, nadolol; tranquilizers such as thioridazine, or alprazolam; and oral contraceptives.

6. Fungal sinusitis in the past was limited to the complications of uncontrolled diabetes. This invasive disease is caused by the *mucor* species. With the increased number of immunocompromised patients, invasive aspergillosis became more frequent. Clinically, these conditions may present with necrosis of the nasal structures involving palate and cribriform plate, and systemic symptoms usually are present. Treatment includes surgical débridement, parenteral antifungal medications, and control of the underlying conditions. A local fungal infection limited to a one-sinus cavity also may occur. It presents as mycetoma (fungal ball) and is caused by *aspergillus* and *candida* species. Unfortunately, it does not respond well to medical treatment and therefore requires surgical excision and establishment of drainage.

7. Allergic fungal sinusitis is a recently recognized entity. Again it is secondary to *aspergillus* infection. Patients typically present with history of a chronic nasal obstruction and demonstrate hypertrophic polyloid nasal mucosa. Local histology and skin testing usually confirm both fungal and allergic components.

8. Several systemic conditions known to have implications in nasal obstruction include cystic fibrosis, Wegener's granulomatosis, immotile cilia syndrome, and the aspirin triad (aspirin intolerance, asthma, and nasal polyps). Therefore, recalcitrant rhinosinus disease that is unresponsive to medical management deserves further investigation.

9. Rhinitis of pregnancy is recognized as a separate entity. The nasal congestion is the result of increased estrogen production; however, on careful questioning, the majority of women had symptoms preceding the pregnancy. What they are experiencing is caused by the common causes of rhinitis.

10. Hypothyroidism-related rhinitis is mentioned in the literature, but its incidence and clinical relevance is not clear.

11. Atrophic rhinitis or ozena is a diffuse atrophy of nasal mucosa, secondary to chronic mucopurulent or crusty rhinopathy with characteristically foul odor. This condition rarely is encountered
in the western world today. The cause of atrophic rhinitis is probably multifactorial, including nutritional and endocrine factors; *Klebsiella ozaened* overgrowth also has been implicated. Some success with ciprofloxacin has been documented in addition to aggressive hygienic measures.\(^\text{38}\)

12. Other conditions that can mimic rhinitis include:

- Adenoid hypertrophy and foreign bodies (in children)
- CSF rhinorrhea (usually a complication of head trauma)
- Unilateral chronic nasal obstruction in adults, especially if associated with epistaxis, should raise a red flag of a possible neoplasm in the nasopharynx, nasal cavity, or paranasal sinus. Therefore a physician comfortable with performing and interpreting a nasopharyngoscopic exam or an ENT specialist should evaluate suspicious conditions lasting over a month in spite of the best medical management.\(^\text{10, 26, 36, 52}\)

**FOCUSED EXAM AND DIAGNOSTIC CONSIDERATIONS**

The exam starts with observing the patient's external nose, facial expression, and comfort level of nose breathing. For example, broad nasal arch, retracted lip, dull facial expression and obligatory mouth breather suggest adenoid hypertrophy in a child. An exam reveals enlarged tonsils and neck lymph nodes, arched palate, teeth malposition, and dull tympanic membranes.

The signs of allergic rhinitis, such as allergic shinners (secondary to the venous outflow obstruction from the periorbital region), semilunar creases in the lower eyelid (Dennie's lines), allergic salute with or without a permanent crease above the tip of the nose, and facial grimacing like rubbing the upper lip against the tip of the nose can be appreciated by an attentive observer.

The indication of difficult nose breathing can be a very narrow nasal bridge, drooping or flail tip of the nose, or structural deformities suggestive of previous nasal fractures. A rhinoscopic exam helps to further investigate signs of obstruction. A light source (preferably headlight) with or without magnification, nasal speculum, or halogen otoscope with a large ear speculum can be used. Keeping eyes and mind open assists in detecting interesting findings. The first landmark to be identified by anterior rhinoscopy is the nasal valve (see Fig. 3), where the vestibular cutaneous and the mucosal lining merge, shaping the narrowest point of the nasal cavity. The inferior turbinate, middle turbinate, anterior aspect of the middle meatus (Fig. 6), septal integrity, and degree of septal deviation should be visualized next, followed by the assessment of patency of the nasal passages, degree of hypertrophy, deformity, or asymmetry of the structures. Attention should be paid to the color and thickness of the mucosal lining, septal integrity, and deviation. The origin of the nasal discharge should be searched for and its quantity, quality (watery, exudative, frankly purulent, crusty), and smell should be evaluated. The differentiation between middle turbinate and polyps sometimes might be difficult.
Figure 6. Magnified anterior rhinoscopy. A = middle meatus; B = middle turbinate; C = nasal septum; D = inferior turbinate.

The middle turbinate may appear with pseudopolypoid hypertrophic lining of the mucosa, but it is a single structure of firm consistency, relatively immobile, and sensitive to touch. The color of the middle turbinate also is more like the rest of the nasal mucosa and not as ashy and transparent as a polyp.

Acute inflammatory changes commonly seen by anterior rhinoscopy are hyperemia and edema of the nasal mucosa covered by exudate. In response to allergens, characteristic signs include pale, bluish, bulging, watery mucosa. Visualization of purulent discharge draining down the middle meatus and facial tenderness on palpation over one or more sinuses with or without facial and periorbital edema is very suggestive of a sinus involvement. By applying a Q-tip, the patency of the nasal passage down to the nasopharynx (notice: not “up” to the nasopharynx) can be assessed, and suspicious structures, which may be a turbinate, a polyp, thick mucus, or any other sort of growth can be checked. This method can provide immediate, magic relief by gently squeezing against the edematous turbinates with a Q-tip soaked in decongestant. A gross functional assessment can be obtained by

- Asking the patient to breathe in and out separately with each side of the nose.
- Watching for mist formation while the patient breathes over some polished surface.
- Listening with the stethoscope for nasal airflow.
- Watching the patient blow his or her nose, checking for the effort, appearance and smell of the discharge. Energetic nose blowers are unlikely to develop sinusitis.

A thorough physical exam gives confidence to the examiner in diagnosis and choice of treatment and tends to reassure an apprehensive patient. Palpation with percussion over the sinuses and transillumination may add more information for somebody who is familiar with these techniques and uses them routinely. A normal transillumination is good evidence against fronto-maxillary sinusitis. Flexible Nasopharyngolaryngoscopy (FNPL) is
an excellent tool for examining the nasopharynx for adenoids and tumors, eustachian tube landmarks, function and, with growing experience, visualizing the middle and superior meatus.

Radiographs, including occipitomental (Waters), posteroanterior (Caldwell), and lateral views, can confirm the clinical impression by presence of opacification, air-fluid levels, and mucoperiosteal thickening of the affected sinuses. The CT coronal sections of the sinuses can give anatomic details about the obstructive pattern of the osteomeatal unit, and axial views can complete the information regarding sphenoidobital recess. Either radiographs or CT scans usually are done in case of treatment failure or recurrence of the disease. Taking the pictures in an acute setting may show just mucosal thickness or generalized opacity, obscuring the actual site of the obstruction.

A-mode sinus sonography is more popular in Europe than in the United States. It is used in evaluation of frontal and maxillary sinuses with the advantage of not exposing the patient to radiation, but does require the physician to develop interpretative skills. The general information about presence of fluid is well-correlated with other imaging techniques.

Nasal cytology with prevalent eosinophils versus neutrophils can confirm the clinical impression of an allergic component, although the reversed picture can suggest an infectious process.

Nasal cultures have a low specificity. They correlate with sinus aspirate only in 60% of the cases. In a patient with clinically diagnosed sinusitis who is still symptomatic after 4 weeks of adequate treatment, the antral puncture should be considered next. It has both diagnostic and therapeutic value of aspiration and lavage. ENT consultation might be warranted.13, 23, 26, 29, 48

Treatment of Nasal Obstruction and Sinusitis

The pharmacologic agent able to completely relieve common cold symptoms has yet to be developed. Nevertheless, the topical and systemic decongestants available on the market can be quite helpful to control symptoms such as rhinorrhea. The nonsteroidal anti-inflammatory agents may alleviate the constitutional symptoms of the common cold. Therefore, the oral preparations in combination with these two are the most reasonable choice to help the patient deal with an upper respiratory episode. New topical formulations such as active antiviral agents, vaccines, nasal mucosa protective agents, and antiseptics suitable for nasal mucosa currently are being studied.24

The ideal topical agent besides the primary pharmacologic properties should be gentle enough with the nasal mucosa and its function. The drawback of topical nasal decongestants include slowing down the ciliary motility and mucociliary clearance, an ischemic effect on the nasal mucosa, and the rebound congestion effect that may occur just after 3 days of use. The role of preservatives such as benzalkonium chloride in the mucosal swelling also should be considered.16 Currently the long-acting
topical adrenergic decongestants available on the market such as oxymetazoline probably are the better choice.ª ²

The new topical agent recently approved for the common cold is the anticholinergic Ipratropium bromide 0.06%, which is marketed in 0.03% strength for vasomotor rhinitis as well.ª

Sodium cromoglycate by inhalation or intranasally has proven in one study to be an effective treatment for the symptoms of URTI.¹

The usefulness of megadoses (from 1 to 6 g/d) of vitamin C is still a subject for debate.²¹

Zinc gluconate lozenges that recently became available on the market with or without vitamin C are advertised as capable to reduce significantly the duration of the common cold.¹⁰

If a diagnosis of bacterial sinusitis is entertained, the choice of first-line antibiotic therapy should reflect the prevalence of β-lactamase producing organisms. Amoxicillin is adequate where the presence of these strains is low but should be combined with clavulanic acid wherever the prevalence of the β-lactamase is substantial. Alternative choices include oral cephalosporins such as cefuroxime axetil, cefprozil, loracarbef, cepodoxime proxetil, or other cephalosporins. In case of an allergy to penicillin, Trimethoprim/sulfamethoxazole and Clarithromycin are the other options. The length of treatment varies with different clinicians; it has an empiric basis not supported by strong evidence that one regimen is better than the other. The more general consensus is to recommend 10 to 15 days of treatment, but if the clinical response is slow, the continuation of therapy for a week after the resolution of symptoms can be a reasonable choice. Probably the longer we treat the more we see the natural course of the infection, that is, the spontaneous resolution regardless of what is done. Chronic sinusitis should be treated for 3 to 4 weeks with anaerobic coverage.⁵,⁶,¹⁸,⁴²

Thinking of rhinosinusitis as a closed-space purulent infection, 500 mg every 8 hours of amoxicillin with or without clavulanic acid is unlikely to address the problem alone. Relieving the obstruction and establishing drainage is at least as important as attempting to sterilize the paranasal sinuses. Without minimizing the importance of antimicrobial treatment of bacterial sinusitis, they are not the only answer to the approach to sinus disease. Edema of the mucosa, increased amount and viscosity of secretions, and insult to ciliary activity all lead to the obstruction of the tiny ostia and creates favorable media for the bacterial overgrowth. Therefore, we propose an alternative term for an efficient management of sinus disease called nasal hygiene, including adequate body hydration, environmental humidification, identification and possibly avoidance of exposure to causative agents, allergic triggers, smoke, and abrupt change in temperature.

The patient should be encouraged to periodically blow his or her nose (each nostril separately). It is surprising how many people are not able to do it. The physician should teach a moderate intensity Valsalva maneuver (air is forced from the nasal cavity into the sinus pockets) and should instruct the patient not to squeeze his or her nose but rather to blow it loudly. In the case of a little child, gentle suction and blowing the nose
achieves an early awareness of nasal respiration. Parents can teach the child how to use his or her nose by simulating sniff/snuff for him. However, lack of nursing, a smoky or dusty dry environment, careless day care, or laziness of the caregiver leads to the stagnation of secretions, paralyzing the cilia, and causing chronic ear and sinus problems.

Use of steam as a decongestant and irrigation with homemade saline solution (a pinch of baking soda and few drops of iodine solution, or commercially prepared saline mixed half and half with Alkalol [aromatic alkaline solution]) are very beneficial and harmless. A forceful pulsating system such as Water Pik will irrigate the cavities under certain pressure. The benefits include the cleansing action, improvement in mucociliary flow, and restoration of the optimal 7.0 pH.

Topical decongestants such as phenylephrine and oxymetazoline can be used for a short period of time because of the early rebound effect and development of rhinitis medicamentosa with chronic use.

Nasal steroid sprays are indicated in sinusitis or whenever an allergic component is suspected. Assuming that the patient is using them correctly, they are very effective but do have a relatively slow onset of action, and patients should be educated about this. They differ in formulation (dry aerosols versus aqueous spray) age limits, and dosing regimen (see box).

| Steroid           | Dosage |
|-------------------|--------|
| Beclomethasone    | Twice a day from age 12, aqueous/aerosol |
| Budesonide        | Every day from age 6, only aerosol       |
| Flunisolide       | Twice a day from age 6 only aqueous     |
| Fluticasone       | Every day from age 12, only aqueous      |
| Mometasone        | Every day from age 12, only aqueous      |
| Triamcinolone     | Every day from age 6, only aqueous       |

If the discharge is watery, aerosolized spray is more suitable. The aqueous solution is reserved for a thick nasal discharge and stuffiness. Apart from personal preferences, all the agents available on the market work very well. The major reason for failure of treatment with topical steroids is because of the inability to get to the site of inflammation through the obstruction. The general principle regarding the way you use medicine is at least as important as what medicine you choose still applies.

Topical agents work better when a patient understands nasal anatomy, applies the medicament properly, and uses positioning techniques that favor sinus drainage and instillation of topical agents. It is a good idea to educate the patient at the first visit, and then review again the correct technique of application at the next encounter.

The antihistaminic spray azelastine may control itching, sneezing, and watery rhinorrhea especially in someone who has contraindications for the steroids and intolerance to oral antihistaminic agents. The symptom's relief is expected after 1 to 2 hours; therefore it can be used as needed.
The mast cell stabilizer, cromolyn sodium, is used as preventive agent in allergic rhinitis, but it is a short-acting agent requiring 3 to 6 applications daily.

Oral decongestants such as phenylpropanolamine and pseudoephedrine, with or without a mucolytic agent such as guaifenesin, can be added. Based on the severity of allergic manifestations, oral antihistamines and oral steroids can play an important role, but their use in presumed acute infectious sinusitis remains controversial.\textsuperscript{18,22,26,38,47}

The anticholinergic nasal spray version of ipratropium bromide comes in 0.03% strength indicated for vasomotor rhinitis and 0.06% strength for the common cold.

**Surgical Option**

It is fairly easy to convince a patient with nasal obstruction to undergo surgery, especially if more than one medical attempt was unsuccessful. It is imperative therefore to recognize the cause of nasal obstruction, to give a thorough trial of medical therapy, and to allow enough time for medications such as topical steroids to become effective. In case of treatment failure, the physician should reevaluate the findings preferably with endoscopy and imaging techniques in an attempt to determine whether there are surgically correctable defects before scheduling surgery.

Patients need to understand that surgery is not always successful, even without complications. The creation of surgically enlarged passages leads to an excessive loss of resistance to the airflow, compromises the ability of air exchange, and does not improve subjectively the nasal breathing. The preservation of the turbinates also is essential to the humidification and conditioning of the inhaled air. In patients with allergic diathesis the surgical outcomes are not satisfactory, or offer only temporary relief at best. Therefore, allergy investigations should precede surgical procedure. There is no doubt that in properly selected patients, functional endoscopic sinus surgery (FESS) can be an important problem-solving procedure. Ideally, referral should be given to patients with chronic, frequently recurrent or complicated rhinosinusitis, who fail the maximal medical management, and in whom a surgically correctable obstruction of the osteomeatal unit or other anatomic defect has been diagnosed.

**CONCLUSION**

The primary care physician needs to be familiar with the pathophysiology of the upper airway, able to obtain pertinent historical data, perform a thorough ENT exam, and use a step-by-step approach to determine the cause of nasal obstruction. Modern imaging techniques should not replace clinical judgment. Antibiotic therapy must be reserved for patients with a clinical picture consistent with bacterial infection. A consideration of allergic cause should be given early in the management. Surgery should
address chronic or complicated cases that did not respond to the maximal medical management.

In order to achieve the best results, encourage the patient to be an active partner in the treatment, putting a major emphasis on nasal hygiene.

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