

Chapter 45: Nonallergic Rhinitis and Infection

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To systematize thinking about the "stuffy nose" (nasal congestion or nasal dyspnea), it is helpful to list the various etiologic factors under four major categories as in the list below, provided one recognizes that some overlap exists between categories and that several coexisting factors may be operative in one patient.

1. Structural disorders
 - a. Deformities: external, internal, congenital malformations, injuries.
 - b. Neoplasms and masses.
 - c. Foreign bodies.
2. Inflammatory disorders
 - a. Rhinitis/sinusitis: bacterial, viral, fungal.
 - b. Nasal and sinus polyposis.
 - c. Ozena, atrophic rhinitis.
 - d. Immunologic disease: sarcoidosis, Wegener's granulomatosis, polyarteritis nodosa, midline granuloma.
3. Allergic rhinitis.
4. Vasomotor rhinitis (see box).

Allergists tend to classify nasal conditions as either allergic or nonallergic, the distinction being made on whether or not the patient's skin tests are positive for whatever antigens are applied (Zeiger, 1989). "Nonallergic rhinitis" by such classifications encompasses a rather broad conglomerate of disorders, some of which (those listed in the structural category) are not at all inflammatory, which makes the suffix "-itis" not appropriate. It would be rather silly to list nasal septal deformity as a type of nonallergic rhinitis.

Infections, however, are inflammatory conditions, and they will be discussed at the end of this chapter, but only briefly, since sinusitis is the subject of more detailed expositions elsewhere in this book.

In this chapter the term *vasomotor rhinitis* will be applied to a group of nasal conditions that are not structural, not infectious or suppurative, not autoimmune, and not allergic in the traditional sense (that is, not IgE mediated or positive for skin tests) (see box). Technically, bacterial or viral infections and even allergies cause a vasomotor reaction in the nasal membranes, but traditional use of the term *vasomotor rhinitis* excludes such entities. Some authors even prefer to use *vasomotor rhinitis* to indicate only conditions of unknown etiology (Georgitis, 1989). All classifications are arbitrary by nature. We prefer to use the term in its physiologic sense.

Vasomotor Rhinitis Physiology

Vaso refers to blood vessels; *motor* to forces; and *rhinitis* to inflammatory conditions of the nose. A brief review of nasal vascular anatomy helps one to understand this important and complex condition.

Box: Vasomotor rhinitis

1. Drug induced
 - a. Antihypertensives
 - b. Nose drop/spray abuse
 - c. Cocaine
 - d. Birth control pills
2. Pregnancy and "premenstrual colds"
3. Hypothyroidism
4. Emotional causes
5. Temperature mediated
6. Irritative rhinitis
7. End-stage vascular atony of chronic allergic or inflammatory rhinitis
8. Recumbency rhinitis
9. Paradoxical nasal obstruction and nasal cycle
10. Rhinitis of no airflow (laryngectomy, choanal atresia, adenoid hyperplasia)
11. Compensatory hypertrophic rhinitis
12. Eosinophilic and basophilic nonallergic rhinitis
13. Other systemic disorders: superior vena cava syndrome
14. Idiopathic rhinitis.

The internal part of the nose receives arterial supply from the anterior and posterior ethmoid arteries, the sphenopalatine arteries, and the greater palatine arteries. The venous drainage is less specific. In general, the venous plexuses about the head - chiefly the orbital, cavernous, and pterygoid - receive the blood from the nasal fossa and shunt it to the jugular veins in the neck. Since there are no valves in this venous system, the pressure and flow dynamics vary with the posture of the individual.

The minute arterioles course in parallel rows in a posteroanterior direction. In contrast to the usual sequential arrangement of arteriole-capillary-venule, venous sinusoids or lakes are located between the capillaries and venules. Thus capillary blood enters these sinusoids before it passes into the venules. The sinusoids are surrounded by fine fibrils of smooth muscle, giving them the power of vasoconstriction and vasodilatation. When they fill and distend, the tissue engorges to such a degree that it is looked on as erectile tissue (Fig. 45-1). This is most striking in the inferior turbinates (Fig. 45-2) and is less so in the nasal septum and other turbinate membranes.

The smooth muscle fibers of the arterioles and venous sinusoids are innervated by the autonomic nervous system (Ritter, 1970). Parasympathetic stimulation results in vasodilation, which engorges the sinusoids with blood and increases congestion and mucus production. Sympathetic stimulation results in vasoconstriction, which squeezes blood out of the nasal membranes, thereby increasing nasal patency and decreasing mucus production.

Causes of Vasomotor Rhinitis

Drug-induced rhinitis

Certain drugs, especially those that affect autonomic vascular control, may affect the vascular channels of the nose.

Antihypertensives

Sympathetic blocking agents, such as reserpine, guanethidine, hydralazine, methyldopa, propranolol, and other beta blockers, may produce a stuffy nose as a side effect. This comes from depletion of norepinephrine stores, resulting in unopposed parasympathetic vasodilation (Zeiger, 1989). Reserpine is the most troublesome in this regard, affecting 8% of users. If this is a problem, the patient's physician can usually substitute another antihypertensive agent that causes less nasal congestion.

Nose drop/spray abuse

Decongestants are sympathomimetics that are employed for their vasoconstrictive action. However, when used topically, their vasoconstriction is so intense that a semiischemic state occurs, during which time products of metabolism accumulate that are strong vasodilators. A rebound vasodilation occurs, creating congestion again. The more frequent and prolonged the use of the topical vasoconstrictors, the more profound the rebound until a loss of vascular tone ensues. The nasal congestion is often profound (Fig. 45-3). For this reason, decongestant nose drops and nose sprays should not be used for more than 3 consecutive days.

An old term for this condition is *rhinitis medicamentosa*, which is rather awkward and even comical sounding; a simpler, more descriptive term would be *rebound rhinitis*.

The treatment of rebound rhinitis requires immediate and total cessation of the nose drop/spray compulsion. Simultaneously, the physician must address the initiating condition that led the patient to start the habit. If it was an allergy, a short course of a corticosteroid might be very helpful. Likewise, infections and structural abnormalities may need attention.

Infants are particularly susceptible to rebound rhinitis and may develop it after just a few days of nose drop usage. Since infants are obligate nasal breathers, a hazardous condition ensues. Fortunately, their response to withdrawal of the drops is prompt and total. Adults, on the other hand, take several days to improve even under the best of circumstances. If the habit has been

well established for months or years, the nasal vasculature may have suffered such permanent changes as to require surgical therapy to the turbinates.

Cocaine

Because cocaine is also a vasoconstrictor, it is theoretically possible that a rebound rhinitis could occur from intranasal "recreational use" (Schwartz et al, 1989). As a practical matter, however, most abusers do not use it constantly. Further, "street cocaine" generally contains adulterants whose deleterious effects overshadow the rebound rhinitis that might result from cocaine. Common adulterants include lactose, mannitol, lidocaine, caffeine, salicylamide, heroin, camphor, talc, borax, and a variety of contaminating bacteria (Fairbanks and Fairbanks, 1983).

These irritants cause crusting, nasal picking, and atrophic rhinitis; when the vasoconstrictive effect of cocaine is superimposed, the septal cartilage suffers from a lack of adequate blood supply, which in turn may lead to nasal septal perforation.

Birth control pills

Some women complain of nasal congestion when they are taking anovulatory drugs (Zeiger, 1989). This condition is caused by the vasoactive effect of estrogens, as described next.

Pregnancy and "premenstrual colds"

Most women note some degree of nasal congestion during pregnancy, and for a few it becomes quite disabling (Malony, 1986; Schatz et al, 1987; Zeiger, 1989). This progressively worsens throughout pregnancy in a direct relationship with the endogenous estrogen levels, which rise dramatically during the later months. Estrogens cause vascular engorgement not only in the uterus, but also in the nose. For the same reason, some women note nasal congestion in the immediate premenstrual period, which they may mistakenly call a "cold".

Many obstetricians prefer that their pregnant patients suffer the nasal congestion rather than take drugs for it. Nevertheless, countless such patients take it on themselves to use over-the-counter antihistamines and decongestants. Many of these drugs have been widely used for many decades without proven harmful effects on the fetus, but actual experimentation in human subjects is lacking. Therefore one cannot state that safety for use in pregnancy has been officially established. Thus the physician can offer such bits of advice, and the patient is left to make her own choice in the matter.

In general, the older antihistamine preparations such as tripeleminamine (Pyribenzamine) and chlorpheniramine (ChlorTrimeton) are preferred over newer ones for patients whose condition is aggravated by a coincidental allergic rhinitis (Schatz et al, 1987; Zeiger, 1989). Topical application of corticosteroids (that is, beclomethasone) is preferred to oral administration (Gluckman, 1983). Orally taken decongestants such as pseudoephedrine (Sudafed, Novafed) enjoy a long safety record and may be useful for nonallergic patients, except for those who are at risk

for hypertension. Elevation of the head of the bed may also be somewhat helpful. Pregnant patients who develop the nose spray habit risk a major problem with rebound rhinitis.

Extremely disabled patients may require cryosurgical treatment of the turbinates, which can be done in the office with the patient under topical and local anesthesia. The temporary effect of cryosurgery is acceptable in this instance, since pregnancy is a self-limited condition.

Hypothyroidism

In 2% to 3% of patients with vasomotor rhinitis the diagnosis is hypothyroidism (Settipani, 1987). The generally hypoactive sympathetic status leads to predominance of parasympathetic activity in the nose, with vasodilation. After the endocrine abnormality is stabilized, the degree of residual change in nasal vasculature can be assessed and managed as below.

Emotional causes

Life situations that produce anxiety, hostility, guilt, or feelings of frustration and resentment can disturb the autonomic vascular balance (Zeiger, 1989). They may cause nasal congestion and discharge by themselves, but more often they exacerbate existing nasal disease. This etiology should be considered a diagnosis of exclusion. The nasal congestion that accompanies a migraine headache is a specific example of how dysfunction in the carotid arterial system affects the nose, and it explains why so many patients with vascular headaches are certain in their own minds that they have sinus headaches.

Even if stress and anxiety cannot be eliminated, treatment directed against the nasal congestion is usually appreciated.

Temperature-mediated rhinitis

Environmental temperature exerts a vasomotor-mediated effect on nasal airway patency. In general, heat elicits vasodilation, and cool air causes vasoconstriction in much the same way that skin wastes or conserves heat. Curiously, however, a person whose feet are exposed to cold is likely to suffer nasal congestion (Proctor, 1977). This commonly observed phenomenon once led a public official to complain about the British House of Commons, which was so poorly heated in the winter, that it gave the members "cold feet and stuffy heads" - the worst possible combination of effects for a legislator!

These observations recall some of the old remedies for a cold: bundle up warmly with feet in a tub of hot water, a hot water bottle on the head, and a cold breeze blowing into the room through an open window. That might be good advice but only in moderation, because prolonged exposure to cold (for example, sitting in a room at near-freezing temperatures for 60 minutes) evokes an increase in nasal airway resistance, particularly in persons with allergic rhinitis (Zeiger, 1989).

Irritative rhinitis

Acute or chronic exposure to irritating dust (especially woodworking dust), gases (that is, formaldehyde from particle-board construction materials), chemicals (that is, chlorophenol wood preservative, chromic acid fumes, and vanadium), cosmetic preparations delivered by aerosol, and other air pollutants (especially sulfur dioxide) elicits a vasomotor reaction that is not truly allergic, but one that creates nasal congestion nevertheless. The most pervasive of these is tobacco smoke, which is inhaled in much higher concentrations than any other environmental pollutant. Treatment of inhaled irritants is avoidance or air filtration. Filter masks are useful for dust at home or at work. (See also discussion of cocaine above.)

End-stage vascular atony of chronic allergic or inflammatory rhinitis

Every physician who has cared for nasal disorders can recall frustrating cases in which appropriate and vigorous therapy for proven allergic rhinitis has failed to relieve nasal congestion, even though other allergy symptoms were relieved. This is because prolonged and profound parasympathetic stimulation of the nasal vascular system may lead to permanent loss of vascular tone. It is seen not only with chronic allergies, but also with chronic sinusitis. Richardson (1948) reminded his audiences of this erectile nature of nasal vasculature by calling this condition *nasal turbinate priapism*. It can best be explained to patients as analogous to varicose veins.

Recumbency rhinitis

Recumbency rhinitis is a nonspecific complaint of persons who experience any form of vasomotor nasal congestion, whether acute or chronic. The hypotonic vascular bed of the nose responds to the dependent position by filling with blood in much the same way that varicose veins become engorged when the extremities are placed in a dependent position. Some investigators regard this phenomenon as a nasal reflex neurally mediated by pressure receptors in the skin.

When a person with rhinitis sleeps on his side, the dependent side becomes congested and the upper side more patent. This also explains why the person whose deviated septum obstructs the right side, for example, usually sleeps with his left side uppermost; otherwise, if he should position his more open side down, it would become congested, and then neither side would be functional.

Paradoxic nasal obstruction and nasal cycle

Eighty percent of adults experience a cyclic congestion and decongestion of the turbinates that alternates from one side to the other (Kern and Arbour, 1976). The mean duration of the cycle is 2.5 hours, but wide variability occurs. This is an automatic nervous system-mediated vasomotor phenomenon. Since it is normal and physiologic, most persons are unaware of it. However, when it is superimposed on some other cause for nasal obstruction, it can be troublesome. This is especially notable in patients with one-sided nasal obstruction from long-

standing deformity of the nasal septum.

With a fixed nasal obstruction, the abnormal side maintains a constant degree of resistance, and the patient may have lost awareness of it. However, the opposite or normal side has a variable resistance because of the continued fluctuations of the nasal cycle, which creates more awareness. Consequently, when the normal side is in the decongested phase of the cycle, the total nasal resistance may be within normal limits, whereas then the normal side is in the congested (turbinate engorgement) phase, the total nasal resistance may exceed the tolerable level, and the patient complains of nasal obstruction on the more normal side; hence the term *paradoxical nasal obstruction*.

Nonairflow rhinitis

Laryngectomy/tracheostomy rhinitis

When the nose has, by some structural abnormality, become excluded from the reciprocating flow of air with its cyclic variations in temperature and humidity and its effects on movement of mucus, a vasomotor reaction occurs. The vascular bed loses its tone, and the turbinates become boggy, swollen, and violaceous. This is a familiar picture to anyone who has examined the nose in a postoperative laryngectomy patient.

Choanal atresia rhinitis

The choanal atresia patient demonstrates an appearance of the nasal membranes similar to that in the laryngectomy patient, but additionally clear nasal mucus accumulates, which cannot be expelled posteriorly by the natural forces of airflow and ciliary action.

Adenoid rhinitis

To a variable extent vasomotor changes may also occur in a child whose nasopharynx is largely occluded by adenoidal hypertrophy (Ritter, 1970). Whether or not infection is active, the phenomenon of no airflow creates a vasomotor rhinitis with boggy, swollen turbinates and accumulations of clear, watery secretions.

Unfortunately, adenoid rhinitis may masquerade as an "allergic nose", which can lead an unwary physician and his patient on a costly, elaborate, and frustrating detour into allergy shots, dietary restrictions, and environmental prohibitions. The embarrassment of the physician is minor compared with the hostility of the parents when they insist on an adenoidectomy and find that it cures the "allergy".

Compensatory hypertrophic rhinitis

Anatomists have long been aware that when a nasal septum is deformed toward one side, the excess space created in the opposite nasal cavity becomes occupied by overgrowth of one or

more of the nasal turbinates (Fig. 45-4). It is possible this occurs to protect the more patent side from excess nasal airflow with its drying and cooling effects.

When the middle turbinate participates in this event, it may become curled and redundant in the middle meatus, or an ectopic sinus cell may develop (20% of adults have such a cell (Fig. 45-5)). The inferior turbinate is most commonly involved. The turbinate becomes thicker and more spongy (see Fig. 45-2) and arches further medially into the airway. Also, the mucosa hypertrophies, and a deep vascular bed develops with an exaggerated expansile capacity.

These changes are not spontaneously reversible, and they should be corrected in conjunction with any nasal septal surgery that is performed. Otherwise, the patient will complain that the septal surgery only partially relieved obstruction on one side of the nose, although significant obstruction persists bilaterally.

Eosinophilic and basophilic nonallergic rhinitis

Eosinophilic nonallergic rhinitis and *basophilic nonallergic rhinitis* are terms applied to clinical conditions of unknown etiology with symptoms suggestive of allergic rhinitis but in which immunoglobulin E (IgE) tests are normal and skin tests to allergens appropriate for the geographic area are negative (Georgitis, 1989). Cytology of nasal mucosal smears determines how the condition is named. The eosinophilic group has been termed *NARES* (nonallergic rhinitis with eosinophilia syndrome).

NARES patients suffer repetitive sneezing attacks, profuse rhinorrhea, and itching in the nose or eyes. They are exquisitely sensitive to environmental stimuli such as smoke, chemical odors, perfume, and changes in posture or weather (temperature and barometric pressure). Attacks can occur anytime of day but are usually worse on arising in the morning. There is no seasonal pattern, and congestion is not generally a complaint.

Neither antihistamines nor decongestants provide significant relief of symptoms, but nasal steroids usually produce a dramatic improvement, and eosinophilia diminishes. Response to steroids suggest to some investigators that these conditions should be caused by unrecognized allergens.

Other systemic disorders

Superior vena cava syndrome (periorbital erythema / edema, nasal stuffiness, headache, and progressive facial swelling) may so mimic allergy symptoms that patients are referred for allergy workups (Settipani, 1987). All of the head and neck venous vasculature is distended, including that of the nasal mucosa. Usually a chest x-ray film will reveal a mass (97% chance of malignancy) encroaching on the vena cava in the superior mediastinum.

Horner's syndrome (unilateral hyperemia, swelling, hypersecretion, miosis, and nasal obstruction) derives from interference with sympathetic innervation. It suggests neoplasm in the

neck or a stellate ganglion block.

Cirrhosis and uremia also produce some degree of nasal congestion.

Idiopathic rhinitis

Unfortunately, some patients with either nasal congestion or rhinorrhea defy diagnosis despite thorough investigation. Empiric therapeutic trials are then offered with topical ipratropium, atropine, or cromolyn and with nonspecific measures discussed below.

Diagnosis

History taking requires an awareness not only about nasal disorders per se, but also about systemic disorders with nasal manifestations, as noted in the previous list and box (also see Chapter 40).

Vasomotor rhinitis is often a diagnosis of exclusion, and necessarily so because other causes of nasal obstruction are far more prevalent. Allergic rhinitis is the most common cause of chronic nasal congestion; the common cold predominates as the most frequent cause of acute complaints. The patient's history is vital. A thorough examination of the nasal passages (including the nasopharynx) before and after application of a topical nasal decongestant may reveal the diagnosis.

When nasal examination shows only boggy, swollen membranes of the inferior turbinates (as in Fig. 45-3), it is a nonspecific finding. The nasal membrane congestion of allergy and infection is a pathophysiologic response that is indistinguishable from the vasomotor phenomenon of pregnancy, of nose spray abuse, or of hypothyroidism. Various authors from time to time have implied that certain colorations of the mucosa suggest certain specific disorders. In practice, however, such color variations are usually too subtle to be of much help.

The nature of the secretions is more helpful: yellow pus suggests bacterial infection; bloody/crusty secretions and ulcerations suggest bacterial infections, neoplasm, or granulomatous disease; clear secretions suggest either allergy or viral infection; and nasal smears with large numbers of eosinophils suggest allergy - as opposed to neutrophils which suggest infection.

The response of the nasal membranes to topical (nose spray) vasoconstriction helps to differentiate a vasomotor rhinitis (which should show a considerable decongestive response) from structural deformities, neoplasms, polyps, sarcoidosis, or the bony turbinate overgrowth of compensatory hypertrophy - all of which should show a limited response. Additionally, many patients with long-established nose spray abuse will demonstrate little vasoconstrictive response to the nose spray used during the examination.

Sarcoidosis (Fig. 45-6), granulomatous disorders, polyps, and tumors are identified by tissue biopsy. Sinus x-ray films are useful not only to detect sinus infections, but also to

document structural abnormalities of the septum and turbinates (see Figs. 45-4 and 45-5). Hypothyroidism is detected with thyroid function tests.

Sometimes a presumptive diagnosis is best verified or rejected by the response to empiric therapy, such as administration of corticosteroids. The more dramatic the response, the more likely that allergy or infection is the primary etiologic factor. Sarcoidosis may respond somewhat, but the various types of vasomotor rhinitis will respond only to the extent that allergy or inflammation is a coexisting element.

Patients who show little response to corticosteroids, either by mouth or by topical spray, will likely find allergy shots a disappointing mode of therapy, even if skin tests are quite positive for inhalant allergies. Golding-Wood (1961) has reminded us that some have extended the use of the term "allergy" to embrace a variety of hyper reactions irrespective of antigen and antibody. It may reasonably be objected that such devices imperil verbal precision and do not serve to clarify concepts.

Nonsurgical Management

Wherever specific therapeutic suggestions could be made for specific disorders, they were mentioned in the preceding paragraphs. The following self-help measures are nonspecific and apply to most cases of chronic long-standing nasal congestion.

1. Sleep with head of the bed tilted upward 30 degrees. This is accomplished not with pillows, but rather with a few bricks, blocks, or books placed under each bedpost at the head of the bed.

2. Establish a regular and vigorous exercise program that might help to reestablish vasomotor tone and control. Vigorous exercise is the body's most efficient homeostatic control to reduce nasal congestion (Proctor, 1977).

3. Avoid known irritating inhalants, especially tobacco smoke.

4. Try oral decongestants such as pseudoephedrine (Sudafed, Novafed), phenylephrine, and phenylpropanolamine (Entex). Since these drugs are sympathomimetics, caution is exercised for their use in patients with hypertension, cardiac arrhythmias, or glaucoma. Oral antihistamines, like corticosteroids, are helpful to the extent that allergy plays a role in the condition.

5. Sleep and work in a cool-air (but not cold) environment, keeping the body (especially the feet and head) warm (Proctor, 1977).

Well-established cases of vasomotor rhinitis are more or less refractory to medical management and ultimately respond best to surgery of the nasal turbinates.

Surgical Management

Middle turbinates do not often compromise the airway unless there is a coexistent nasal septal deformity or an ectopic ethmoid cell has grown into the turbinate. In either instance, surgery may be required.

Inferior turbinates are more often at fault. They have been subjected to a wide variety of treatments by surgeons throughout the history of rhinology. Following is a partial list of various turbinate treatment methods with brief comments on the limitations of each.

1. *Inferior turbinate injection with corticosteroids* is used primarily for seasonal allergy treatment and for weaning the rebound rhinitis patient off nose sprays. This method of treatment provides temporary relief of symptoms. The technique requires a nonforceful injection of small particle-size preparations such as triamcinolone acetonide or diacetate (Kenalog, Aristocort) or prednisolone tebutate (Hydeltra-TBA) into multiple sites along the turbinate with a small-bore needle. The medication is injected only after a topical vasoconstrictor has been applied so as to limit the chance of a direct intravascular injection, which can embolize into orbital vessels and (rarely) create visual loss (Mabry, 1981).

2. *Inferior turbinate injection with caustic agents* has been largely abandoned because of intensely painful reactions. This method provides only very brief relief, if any.

3. *Turbinate displacement ("out-fracture")* is easy and simple but fails to remove thickened membrane and bone. "Green-stick" effect occurs unless the fracture is created from underneath the turbinate at the apex of the meatus where turbinate bone inserts into the lateral nasal wall.

4. *Electrical or chemical surface cautery* creates damage to surface rather than to the vascular bed where it is needed. It gives only very temporary relief, if any.

5. *Electrical submucosal cautery* creates an intensive reaction that can lead to bone sequestration. More commonly, it fails to remove thickened bone (such as in Fig. 45-2) that occupies much of the airway. This gives only partial or temporary relief.

6. *Cryosurgery of the turbinates* (Ozenberger, 1970) causes submucosal destruction at the cost of surface destruction. Also, this method fails to remove thickened bone and gives only partial, temporary relief. Septal perforations may occur.

7. *Total turbinectomy* results in a loss of functioning nasal membranes needed to warm and moisturize air. Atrophic rhinitis may result.

8. In *partial turbinectomy* (that is, of the anterior end or the inferior margin), results and complications depend on the amount that is resected.

9. *Submucous resection* of the turbinate bone (Figs. 45-2, 45-7, and 45-8) affords the removal of disordered tissue (for example, thickened, spongy, space-occupying bone and hypervascular submucosa) while sparing the surrounding normal physiologically functioning tissue and produces consistent, long-lasting, and predictably favorable results (Raphael et al, 1991).

Because of bleeding that occurs with turbinate surgery, most of these operations are performed in an operating room and nasal packing may be required postoperatively.

Infectious Rhinosinusitis

The most common cause of nonallergic rhinitis is infection in the upper respiratory tract. The nose is continually exposed to a myriad of infectious organisms: viruses, bacteria, and fungi. Several endogenous factors protect the nasal mucosa from infection. The submucosal glands produce a thick layer of tenacious mucus that entraps microorganisms. Cilia of the mucosal epithelium then propel the mucus and the entrapped organisms into the nasopharynx where they are swallowed. This prevents penetration of these microorganisms into the nasal epithelium.

Additionally, the nasal glands produce a series of enzymes, antibodies, and other naturally protective proteins that inactivate or kill many viruses, bacteria, and fungi. The antimicrobial properties of glandular secretions have not been well described in the literature but may represent the single most important function of nasal secretions (Raphael et al, 1991).

Viral infections account for about six "colds" per year in young children. The frequency diminishes throughout life, so that older adults suffer only two or three "colds" per year. In the USA the common cold is most prevalent between the months of September and March. During a typical cold season, at least three-fourths of all families will experience a "cold" in at least one family member.

The common cold can be produced by any of more than 200 different species of viruses within the following groups: picornavirus (including rhinovirus, ECHO virus, Cocksackie virus), adenovirus, reovirus, orthomyxovirus, paramyxovirus, and coronavirus. Of these, the rhinovirus group produces the greatest proportion of colds. Such viruses are spread through person-to-person contact with secretions from the nose, mouth, or eyes. A person may inhale virus-laden droplets from another person's sneeze, or more commonly, a person may become infected by touching a hand or object (for example, a door handle, telephone, or handkerchief) that has been contaminated by someone with a cold. The person then touches his own eye, nose, or mouth, and the virus has been spread.

The nasal examination of a cold sufferer reveals diffusely swollen, erythematous mucosa with a watery mucous discharge, a picture not easily distinguished from an acute allergic attack, except by cytology. A common cold lasts about a week, but in 5% to 10% of sufferers it can persist up to 3 weeks. Viruses impair ciliary activity of the nasal mucosa, which makes the sufferer more susceptible to secondary bacterial infections of the respiratory tract. Bacterial

sinusitis follows a "cold" in about 0.5% to 10% of cases.

There is no specific therapy for the common cold. Symptoms are somewhat alleviated with the self-help remedies suggested for vasomotor rhinitis. Rest, increased fluid intake, and air humidification seem to be helpful, as are a variety of over-the-counter medications selected to treat specific individuals symptoms.

The influenza viruses produce a more debilitating illness than do the rhinoviruses. Fever may be more pronounced (and may last up to a week); there may be necrosis of the ciliated epithelial layer of the mucosa; systemic symptoms such as lethargy, fatigue, and myalgias are usually troublesome; and secondary bacterial infections are more common.

Viruses A, B, or C of the orthomyxovirus group cause influenza. Unlike for the common cold, immunization is available for influenza, although the selection of viruses used in the annual influenza vaccine (in advance of the influenza outbreak) still remains an art rather than a pure science. Since there is significant mortality associated with influenza (over 10,000 deaths per year), the vaccine is recommended for patients who are elderly, immunosuppressed, or debilitated with pulmonary, cardiac, or other systemic diseases.

Summary

The nasal vasomotor reaction creates nasal congestion because of dilation of the highly vascular bed of the nasal mucous membranes, especially those of the inferior turbinates. This may aggravate the nasal obstruction of a patient with a structural deformity. Vasomotor reaction is also the physiologic mechanism by which allergies and infections of the nose cause nasal congestion, or it may be caused by a variety of unrelated nasal or systemic disorders that give rise to vasomotor rhinitis (see box, p. 775).

When examination reveals a specific diagnosis, the treatment should be specific. However, a variety of nonspecific remedies (as detailed above) can also be helpful where a specific etiology is not apparent.