

there are few studies in the literature of the autonomic regulatory systems of the nasal membranes, the fact that most patients are seniors older than 50 years, who present with vasomotor rhinitis, may also suggest a gradual deterioration of this system. Surgical and nonsurgical trauma of the turbinates has been shown to affect their function.²

It is important to recognize this pattern of nasal hyperreactivity in patients without a positive ten-

dency for allergy, since the traditional methods of allergy management may fail to alleviate symptoms. As Dr Smith notes, pharmacologic and nonpharmacologic therapies may give adequate control for the symptoms and that in the future perhaps other methods of diagnosis will help elucidate the disorder.

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Vasomotor Rhinitis Should Not Be a Wastebasket Diagnosis

In his article Dr Smith discusses an important problem affecting millions of patients—chronic rhinitis. He does an excellent job of reviewing the most common forms of rhinitis, including allergic rhinitis, nonallergic rhinitis with eosinophilia syndrome (NARES), and vasomotor rhinitis. More exhaustive lists of the differential diagnosis may be found in standard texts and review articles.^{1,2}

The precise physiological mechanism present in vasomotor rhinitis is unknown; Dr Smith has described a possible mechanism for vasomotor rhinitis that involves dysregulation of the autonomic nervous system. This is not a new idea; references are quoted from 1950³ and 1951.⁴ Recent studies, including Dr Smith's own studies,⁵⁻⁸ are also suggestive of this mechanism. What we do not understand is how this exactly relates or does not relate to other rhinitides, such as allergic rhinitis, chronic infectious rhinosinusitis, NARES, rhinitis medicamentosa, or endocrine rhinitis (such as that caused by pregnancy or hypothyroidism). Other authors have postulated a continuum between allergic rhinitis and NARES and nonallergic rhinitis. In this case, diagnostic tests may be used to distinguish these entities—nasal cytological examination for eosinophils and evaluation for allergen-specific IgE by skin (in vivo) or serum (in vitro) testing for serum

eosinophils. This does not explain, however, why similar responses (ie, congestion and rhinorrhea) may be observed in multiple nasal disorders (such as allergic rhinitis, vasomotor rhinitis, and NARES). Merely noting that some types have eosinophils or IgE antibodies does not help to sufficiently delineate the pathophysiological mechanism. Eosinophils in the tissue may be found in several different conditions and may be recruited to the site by more than 1 mechanism; however, IgE-mediated allergic rhinitis is the most common. Dr Smith is correct in noting that most clinicians do not even go this far; rather than doing any testing (eg, testing for allergy, nasal cytology, or serum eosinophils), the diagnosis is often made by history review and therapeutic trial of allergy medications alone.

The lower and upper airways are continuous and have many similarities; for example, allergic inflammation, with both an early-phase and a late-phase immunologic reaction, occurs in both allergic rhinitis and allergic asthma using similar pathways and similar mediators of inflammation. There is also controversy about whether all asthma (reactive airway disease) is allergic. In the lung, testing, such as pulmonary function tests with methacholine challenge, can demonstrate clinical airway hyperreactivity. These tests can be used diagnostically to aid



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in the diagnosis of asthma, often along with a trial of antiasthmatic medications. In the nose, such a test is not used clinically in the otolaryngologist's office. To study these disorders, research studies have used challenges with methacholine, nitrous oxide, nonspecific irritants, capsaicin, histamine, allergens, and aspirin, with measurements by rhinomanometry, acoustic rhinometry, rhinostereometry, skin tests, nasal cytological tests, serum mediator levels, tests for serum eosinophils, complement activation, and nasal lavage or nasal peak flowmeters.⁴⁻²⁸ Standardization of a method that uses a defined challenge substance—with agreed upon amounts and measured by standardized methods—could help to better diagnose these patients. Reasonable candidates for practical measurement methods that could be easily adapted for routine

clinical office use include allergen, aspirin, or methacholine challenge, with acoustic rhinometry or nasal peak flow measurements.

Clinically, however, the picture is confounded by the observation that most patients with chronic rhinitis have a "mixed" type including both allergic-type symptoms (eg, sneezing in response to dust mite dander) and vasomotor symptoms (eg, nasal congestion with perfumes, smog, cleaning solutions, and drugs). This observation parallels the findings in the lung: patients exhibit both allergic and nonallergic inflammatory responses. In the lungs, infectious triggers (ie, viruses, bacteria, and fungi) are also common triggers of allergic inflammation (as they are in the nose). In the lungs, infection with certain viruses (eg, respiratory syncytial virus) in a genetically susceptible host may cause persistent reactive airway disease. It is now well established that reactive (lower) airway disease is a continuum from "no hyperreactivity" (as measured by negative methacholine challenge) to "hyperreactive reactive airway disease." This disease process may be triggered by viruses, allergens, or possibly pollution.

There is strong evidence to suggest that there is 1 airway (not a separate upper and lower respiratory tract) that produces similar, although not identical, response to stimuli. This raises another interesting thought—could we perhaps cause chronic rhinosinusitis in a genetically susceptible host with a virus such as respiratory syncytial virus?

On a practical basis, however, how does the careful clinician proceed with the differential diagnosis of rhinitis? Possible diagnostic tests for the workup of chronic rhinitis might include the following:

1. A careful history review checking for a history or family history of allergy, previous responses or lack of response to allergy medications, presence of hormonal changes (such as those caused by pregnancy or thyroid disease), use of drugs causing nasal congestion (eg, hormones and antihypertensive agents), as well

as a general assessment of health, including the immune system.

2. A careful head and neck examination for other causes of rhinitis and associated conditions such as purulent discharge, deviated septum, red eyes, turbinate hypertrophy, polyps, and tumors.
3. Diagnostic tests that may be indicated after the performance of steps 1 and 2 may include diagnostic nasal endoscopy, allergy testing, nasal cytology, acoustic rhinometry, nasal peak flows, or sinus computed tomographic scans.

I agree with Dr Smith that vasomotor rhinitis should not be a "wastebasket" diagnosis. All too often step 3 (diagnostic testing) is omitted. The practice of evidence-based medicine would be enhanced by more precise diagnosis, enabling the use of appropriately targeted therapies. Until then, vasomotor rhinitis may continue to be used as a wastebasket diagnosis.

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