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Classification of recommendations and evidence

**Category of evidence**

- **Ia.** Evidence from meta-analysis of randomized controlled trials
- **Ib.** Evidence from at least 1 randomized controlled trial
- **IIa.** Evidence from at least 1 controlled study without randomization
- **IIb.** Evidence from at least 1 other type of quasi-experimental study
- **III.** Evidence from nonexperimental descriptive studies, such as comparative studies
- **IV.** Evidence from expert committee reports or opinions or clinical experience of respected authorities, or both
- **LB** Evidence from laboratory-based studies.
- **NR** Not rated.

**Strength of Recommendation**

- **A** Directly based on category I evidence
- **B** Directly based on category II evidence or extrapolated recommendation from category I evidence
- **C** Directly based on category III evidence or extrapolated recommendation from category I or II evidence
- **D** Directly based on category IV evidence or extrapolated recommendation from category I, II, or III evidence

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**PREFACE [SUMMARY STATEMENTS 8, 9, 13]**

Rhinitis, as defined for the purposes of this document, is characterized by 1 or more of the following nasal symptoms: congestion, rhinorrhea (anterior and posterior), sneezing, and itching. Rhinitis is usually associated with inflammation, but some forms of rhinitis such as vasomotor rhinitis or atrophic rhinitis are not predominantly inflammatory.

Rhinitis is a significant cause of widespread morbidity, medical treatment costs, reduced work productivity, and lost school days. Although sometimes mistakenly viewed as a trivial disease, symptoms of allergic and nonallergic rhinitis may significantly affect a patient’s quality of life and can be associated with conditions such as fatigue, headache, cognitive impairment, and sleep disturbance. Appropriate management of rhinitis may be an important component in effective management of coexisting or complicating respiratory conditions, such as asthma, sinusitis, and sleep apnea. The financial burden to society for allergic rhinitis is substantial. The total direct ($7.3 billion) and indirect costs ($4.28 billion, including loss of productivity) estimated in the United States for 2002 were $11.58 billion.1

Allergic rhinitis affects between 10% and 30% of all adults and as many as 40% of children.2,3-6 In most studies, the ratio of allergic to pure nonallergic rhinitis is 3:1.2 Preliminary data suggest that 44% to 87% of patients with rhinitis may have mixed rhinitis, a combination of allergic and nonallergic rhinitis.2,6 Worldwide, the prevalence of allergic rhinitis continues to increase.

The objective of “Diagnosis and Management of Rhinitis: An Updated Practice Parameter” is to improve the care of patients by providing the practicing physician with an evidence-based approach by reviewing data in the medical literature and
incorporating this evidence into development of this guideline. While giving an overview of all categories of rhinitis, the parameter will focus on the diagnosis and treatment of allergic rhinitis. Using the 1998 practice parameter on “Diagnosis and Management of Rhinitis: Complete Guidelines” as the basis, the working draft of this updated rhinitis practice parameter was prepared by a work group chaired by Mark S. Dykewicz, MD, and was revised and edited by the Joint Task Force on Practice Parameters under the leadership of Dana V. Wallace, MD. Preparation of this draft included a review of the recent medical literature using a variety of search engines such as PubMed. Published clinical studies were rated by category of evidence and used to establish the strength of the recommendations, as defined in the preamble to this parameter. The parameter was then reviewed by experts on rhinitis selected by the sponsoring organizations of the American Academy of Allergy, Asthma & Immunology and the American College of Allergy, Asthma, and Immunology. Based on this process, this parameter represents an evidence-based document.

Components and organization of this parameter

The “Diagnosis and Management of Rhinitis: An Updated Practice Parameter” contains an annotated algorithm that presents the major decision points for the appropriate evaluation and treatment of patients with suspected rhinitis. This is followed by a collation of Summary Statements, which represent key points in the evaluation and management of this condition. Tables that provide clinically useful information in a concise format precede the body of the practice parameter, which provides a referenced narrative discussion of each Summary Statement. The graded references and figures complete the document. The Executive Summary emphasizes the key updates since the 1998 rhinitis parameter (Box).

To obtain the maximum value from this practice parameter in the most time-efficient manner, the clinician should review the Executive Summary, annotated algorithm, Summary Statements, and tables because these are created to provide the key information. The text and graded references provide the foundation on which the Joint Task Force formulated and graded the Summary Statements.

ABBREVIATIONS

ACE: Angiotensin-converting enzyme
AERD: Aspirin-exacerbated respiratory disease
ARIA: Allergic Rhinitis and its Impact on Asthma
BHR: Bronchial hyperresponsiveness
CF: Cystic fibrosis
CNS: Central nervous system
CSF: Cerebral spinal fluid
CT: Computed tomography
cysLT: Cysteinyl leukotriene
ECO: Eosinophilic cationic protein
FDA: US Food and Drug Administration
HEPA: High-efficiency particulate air
IOC: International Olympic Committee
IOP: Intraocular pressure
LT: Leukotriene
LTRA: Leukotriene receptor antagonist
MRI: Magnetic resonance imaging
NARES: Nonallergic rhinitis with eosinophilia syndrome
NSAID: Nonsteroidal anti-inflammatory drug
OA: Occupational asthma
OME: Otitis media with effusion
OSAS: Obstructive sleep apnea syndrome
OTC: Over-the-counter
PCD: Primary ciliary dyskinesia
PRN: When necessary (from Latin pro re nata)
QOL: Quality of life
RFVTR: Radiofrequency volumetric tissue reduction
RQLQ: Rhinoconjunctivitis Quality of Life Questionnaire
RUDS: Reactive upper-airways dysfunction syndrome
SIT: Specific immunotherapy
SPT: Skin prick test
USOC: US Olympic Committee

COLLABORATION OF SUMMARY STATEMENTS

Definition and classification of rhinitis

1. Rhinitis is characterized by 1 or more of the following symptoms: nasal congestion, rhinorrhea (anterior and posterior), sneezing, and itching. D

Differential diagnosis of rhinitis and associated conditions

2. Rhinitis should be classified by etiology as allergic or nonallergic and differentiated from conditions that mimic symptoms of rhinitis. C

Key updates

The following is a list of key updates discussed in this document:

- Pharmacologic products introduced since publication of the 1998 “Diagnosis and Management of Rhinitis: Complete Guidelines”
- More defined positioning of agents (eg, leukotriene receptor antagonists) in management based on more recent evidence
- Introduction of episodic as a term to describe rhinitis elicited by sporadic exposures to inhalant allergens, and implications for treatment
- Use of certain agents—that is, intranasal corticosteroids—on an as-needed basis
- Emphasis on recognizing comorbidities of allergic rhinitis (AR), such as asthma, sinusitis, and obstructive sleep apnea, and conducting appropriate studies, such as pulmonary function testing and sleep apnea studies
- Evidence on using combination therapy, specifically leukotriene receptor antagonists, with antihistamines
- Need to consider the benefits versus recently raised safety concerns about oral decongestants before their use in children below age 6 years
- Recommendation of considering second-generation antihistamines as safe agents for use during pregnancy
- Use of intranasal corticosteroids for symptoms of allergic conjunctivitis associated with rhinitis
- Consideration of using a Rhinitis Action Plan
- Emerging diagnostic and surgical procedures, such as acoustic rhinometry and radiofrequency volumetric tissue reduction
3. Symptoms of allergic rhinitis may occur only during specific seasons, may be perennial without seasonal exacerbation, may be perennial with seasonal exacerbations, or may occur episodically after specific aeroallergen exposures. C

4. **Episodic allergic rhinitis** is a new rhinitis category that denotes allergic nasal symptoms elicited by sporadic exposures to inhalant aeroallergens. D

5. The severity of allergic rhinitis ranges from mild and intermittent to seriously debilitating. D

6. Although there is no generally accepted method of grading the severity of rhinitis, the clinician may want to consider a graphic rating scale. D

7. Mixed rhinitis (combined allergic and nonallergic rhinitis) is noted in approximately 44% to 87% of patients with allergic rhinitis and is more common than either pure allergic rhinitis or nonallergic rhinitis. C

**Burden and epidemiology of rhinitis**

8. Allergic rhinitis affects 30 to 60 million people in the United States annually, including 10% to 30% of adults and as many as 40% of children. C

9. Risk factors for allergic rhinitis include (1) family history of atopy, (2) serum IgE >100 IU/mL before age 6 years, (3) higher socioeconomic class, and (4) presence of a positive allergy skin prick test (SPT). C

10. The influence of early childhood exposure to infections, animals, and secondary tobacco smoke on the development of atopy and allergic rhinitis is still unknown. C

11. Aeroallergen sensitization may occur within the first 2 years of life. C

12. The cost of treating allergic rhinitis and indirect costs related to loss of workplace productivity resulting from the disease are substantial. Rhinitis is also a significant cause of lost work and school days. C

**Allergic rhinitis**

**Pathogenesis**

13. The symptoms of allergic rhinitis result from a complex allergen-driven mucosal inflammation caused by interplay between resident and infiltrating inflammatory cells and a number of vasoactive and proinflammatory mediators, including cytokines. Sensory nerve activation, plasma leakage, and congestion of venous sinusoids also contribute. C

14. Allergic rhinitis may be characterized by early-phase and late-phase responses. Each type of response is characterized by sneezing, congestion, and rhinorrhea, but congestion predominates in the late phase. C

**Seasonal and perennial allergic rhinitis**

15. Seasonal allergic rhinitis is caused by an IgE-mediated reaction to seasonal aeroallergens. The length of seasonal exposure to these allergens is dependent on geographic location and climatic conditions. C

16. Perennial allergic rhinitis is caused by an IgE-mediated reaction to perennial environmental aeroallergens. These may include dust mites, molds, animal allergens, or certain occupational allergens, as well as pollen in areas where pollen is prevalent perennially. C

**Associated allergic conjunctivitis**

17. Allergic rhinitis is often accompanied by symptoms of allergic conjunctivitis. C

18. Many treatments used for allergic rhinitis can benefit associated symptoms of allergic conjunctivitis, and a variety of topical ophthalmic agents is useful for specific treatment of associated ocular symptoms. A

19. Intranasal corticosteroids, oral antihistamines, and intranasal antihistamines have similar effectiveness in relieving ocular eye symptoms associated with rhinitis. A

**Nonallergic rhinitis syndromes**

20. Nonallergic rhinitis is characterized by periodic or perennial symptoms of rhinitis that are not a result of IgE-dependent events. Examples of nonallergic rhinitis are infectious rhinitis, vasomotor rhinitis, and the nonallergic rhinitis with eosinophilia syndrome (NARES). C

**Vasomotor rhinitis**

21. Vasomotor rhinitis (idiopathic rhinitis) accounts for a heterogeneous group of patients with chronic nasal symptoms that are not immunologic or infectious in origin and is usually not associated with nasal eosinophilia. D

**Rhinitis from foods and alcohol**

22. Rhinitis may occur after ingestion of foods or alcoholic products. This may be a result of vagally mediated mechanisms, nasal vasodilation, food allergy, and/or other undefined mechanisms. Food allergy is a rare cause of rhinitis without associated gastrointestinal, dermatologic, or systemic manifestations. B

**Infectious rhinitis**

23. Infectious rhinitis and rhinosinusitis may be acute or chronic. Acute infectious rhinitis is usually a result of 1 of a large number of viruses, but secondary bacterial infection with sinus involvement may be a complication. Symptoms of acute infectious rhinosinusitis include nasal congestion, mucopurulent nasal discharge, pain and pressure, headache, olfactory disturbance, postnasal drainage, and cough. C

24. Viral infections account for as many as 98% of acute infectious rhinitis and the majority of rhinitis symptoms in the young child. Routine nasopharyngeal cultures when bacterial infections are suspected do not add diagnostic value. C

**NARES**

25. NARES is characterized by nasal eosinophils in patients who have perennial symptoms and occasionally reduced sense of smell. These patients often lack evidence of allergic disease as demonstrated by absence of positive skin tests and/or specific IgE antibodies in the serum. C

**Occupational rhinitis**

26. Occupational rhinitis is rhinitis arising in response to airborne substances in the workplace, which may be mediated by allergic or nonallergic factors, such as laboratory animal...
antigen, grain, wood dusts, chemicals, and irritants. It often coexists with occupational asthma (OA). C

Hormonal rhinitis
27. Causes of hormonal rhinitis include pregnancy and menstrual cycle–related rhinitis. Pregnancy rhinitis, when present, is associated with significant nasal congestion, starts after the second month of pregnancy, and usually disappears within 2 weeks after delivery. C

Drug-induced rhinitis
28. Drug-induced rhinitis may be caused by a number of medications, including angiotensin-converting enzyme (ACE) inhibitors, phosphodiesterase-5–selective inhibitors, phenolamine, ß-receptor antagonists, aspirin, and other nonsteroidal anti-inflammatory drugs (NSAIDs). Rhinitis medicamentosa is a syndrome of rebound nasal congestion that follows the overuse of intranasal ß-adrenergic decongestants or cocaine. C

Atrophic rhinitis
29. Treatment of primary and secondary atrophic rhinitis involves reducing crusting and alleviating the foul odor by continuous nasal hygiene, such as nasal lavage and crust debridement, and the use of topical and/or systemic antibiotics when purulent secretions or an acute infection is present. C

Conditions that mimic rhinitis
Nasal polyps
30. Nasal polyps may occur in conjunction with chronic rhinitis or sinusitis and may contribute significantly to the patient’s symptoms. Nasal polyps should always be considered in the differential diagnosis of patients who present with invariant nasal congestion and/or anosmia and its sequelae. Allergy as a cause of nasal polyps has not been established, but nasal polyps may occur in conjunction with allergic rhinitis. C

Anatomic abnormalities
31. Signs and symptoms suggestive of rhinitis can be produced by other conditions, including nasal septal deviation, tumors, and hypertrophy of the nasal turbinates. C
32. In infants and young children, nasal congestion or obstruction can result from structural problems, such as cleft palate and adenoidal hypertrophy, or functional problems, such as laryngopharyngeal reflux. D

Cerebral spinal fluid rhinorrhea
33. Refractory clear rhinorrhea may be a result of cerebral spinal fluid (CSF) leak, which is often caused by trauma or recent surgery. B

Ciliary dysfunction
34. Ciliary dysfunction can be primary (primary ciliary dyskinesia; PCD) or secondary (eg, viral infection) and may contribute to recurrent rhinitis and sinus infections. C

Evaluation and diagnostic studies
History
35. An effective evaluation of the patient with rhinitis often includes the following: a determination of the pattern, chronicity, and seasonality of nasal and related symptoms (or lack thereof); response to medications; presence of coexisting conditions; occupational exposure; and a detailed environmental history and identification of precipitating factors. D
36. Evaluation of rhinitis therapy should include assessment of quality of life (QOL). C

Physical examination
37. A physical examination of all organ systems potentially affected by allergies with emphasis on the upper respiratory tract should be performed in patients with a history of rhinitis. The nasal examination supports but does not definitely establish the diagnosis of rhinitis. D

Testing for specific IgE antibody
Skin testing
38. Determination of specific IgE, preferably by skin testing, is indicated to provide evidence of an allergic basis for the patient’s symptoms, to confirm or exclude suspected causes of the patient’s symptoms, or to assess the sensitivity to a specific allergen for avoidance measures and/or allergen immunotherapy. B
39. Skin tests are the preferred tests for the diagnosis of IgE-mediated sensitivity. The number of skin tests and the allergens selected for skin testing should be determined on the basis of the patient’s age, history, environment, and living situation, such as area of the country, occupation, and activities. D

In vitro assays for specific IgE
40. The precise sensitivity of specific IgE immunoassays compared with skin prick/puncture tests is approximately 70% to 75%. Immunoassays have similar sensitivity to skin tests in identifying those patients with nasal symptoms elicited after natural or controlled allergen challenge tests. C
41. Interpretation of specific IgE immunoassays may be confounded by variables such as potency of allergens bound to solid support systems, cross-reactive proteins and glycoepitopes, specific IgG antibodies in the test serum, and high total IgE. D

Special diagnostic techniques
42. In selected cases, special techniques such as fiber optic nasal endoscopy and/or rhinomanometry may be useful in evaluating patients presenting with rhinitis symptoms. These tests may require special expertise for performance and interpretation. Patients with nasal disease require appropriate examination for associated diseases, such as sinusitis and otitis media. B
43. Nasal smears for eosinophils are not necessary for routine use in diagnosing allergic rhinitis when the diagnosis is clearly supported by the history, physical examination, and specific IgE diagnostic studies but may be a useful adjunct when the diagnosis of allergic rhinitis is in question. C
44. Although the saccharin test for mucociliary clearance has been relied on as a clinical screening test, it cannot be relied on for a definitive diagnosis of mucociliary dysfunction. C
45. Nasal biopsy may be indicated when determining whether a lesion is neoplastic or granulomatous or if there is an abnormality in the ultrastructure of cilia. C
46. The measurement of total IgE and IgG subclasses for the diagnosis of allergic rhinitis has limited value and should not be routinely performed. C
47. The presence of β-2-transferrin in the nasal secretions is a sensitive method of confirming cerebral spinal fluid rhinorrhea. B

Special testing considerations in children
48. In children with rhinitis, the use of immune studies, sweat test, sinus computed tomography (CT), and nasal endoscopy may be indicated when they are suspected to have comorbid conditions such as immune deficiency, cystic fibrosis (CF), and chronic sinusitis. C

Testing for comorbid conditions
49. A formal evaluation for obstructive sleep apnea may be considered in children and adults presenting with chronic rhinitis and other risk factors associated with sleep-disordered breathing. C
50. Pulmonary function tests should be considered in patients with rhinitis to assess the possibility that asthma might be present. D

Tests without diagnostic validity
51. There is no evidence that the following procedures have diagnostic validity for allergic rhinitis: cytotoxic tests, provocation-neutralization, electrodermal testing, applied kinesiology, iridology, and hair analysis. B (see “Allergy Diagnostic Testing: An Updated Practice Parameter”)

Management of rhinitis
Environmental control measures
52. The most common allergic triggers for rhinitis include pollen, fungi, dust mites, furry animals, and insect emanations. B
53. The types of pollen responsible for rhinitis symptoms vary widely with locale, climate, and introduced plantings. B
54. Highly pollen-allergic individuals should limit exposure to the outdoors when high pollen counts are present. B
55. Fungi are ubiquitous organisms, many of which produce clinically important allergens. B
56. Reduction of indoor fungal exposure involves removal of moisture sources, replacement of contamination materials, and the use of dilute bleach solutions on nonporous surfaces. D
57. Clinically effective dust mite avoidance requires a combination of humidity control, dust mite covers for bedding, high-efficiency particulate air (HEPA) vacuuming of carpeting, and the use of acaricides. B

58. Avoidance is the most effective way to manage animal sensitivity. D
59. Cockroaches are a significant cause of nasal allergy, particularly in inner-city populations. C
60. The best treatment for rhinitis triggered by irritants, such as tobacco smoke and formaldehyde, is avoidance. B

Pharmacologic therapy
Oral antihistamines
61. Second-generation antihistamines are generally preferred over first-generation antihistamines for the treatment of allergic rhinitis. First-generation antihistamines have significant potential to cause sedation, performance impairment, and anticholinergic effects. Although occasionally advantageous (eg, sleep induction when taken at bedtime or a reduction in rhinorrhea), these properties are usually undesirable and are potentially dangerous. Second-generation antihistamines have less or no tendency to cause these effects. B
62. Before prescribing or recommending a first-generation antihistamine, the physician should ensure that the patient understands both the potential for adverse effects and the availability of alternative antihistamines with a lower likelihood of adverse effects. D
63. There are important differences among the second-generation antihistamines in regard to their sedative properties: fexofenadine, loratadine, and desloratadine do not cause sedation at recommended doses; loratadine and desloratadine may cause sedation at doses exceeding the recommended dose; cetirizine and intranasal azelastine may cause sedation at recommended doses. A
64. Among the newer, non-sedating antihistamines, no single agent has been conclusively found to achieve superior overall response rates. C

Intranasal antihistamines
65. Intranasal antihistamines may be considered for use as first-line treatment for allergic and nonallergic rhinitis. A
66. Intranasal antihistamines are efficacious and equal to or superior to oral second-generation antihistamines for treatment of seasonal allergic rhinitis. A
67. Because systemic absorption occurs, currently available intranasal antihistamines have been associated with sedation and can inhibit skin test reactions. A
68. Intranasal antihistamines have been associated with a clinically significant effect on nasal congestion. A
69. Intranasal antihistamines are generally less effective than intranasal corticosteroids for treatment of allergic rhinitis. A

Oral and topical decongestants
70. Oral decongestants, such as pseudoephedrine and phenylephrine, are α-adrenergic agonists that can reduce nasal congestion but can result in side effects such as insomnia, irritability, and palpitations. A
71. Oral and topical decongestants agents should be used with caution in older adults and young children, and in patients
of any age who have a history of cardiac arrhythmia, angina pectoris, cerebrovascular disease, hypertension, bladder neck obstruction, glaucoma, or hyperthyroidism. C

72. Topical decongestants can be considered for short-term and possibly for intermittent or episodic therapy of nasal congestion, but are inappropriate for regular daily use because of the risk for the development of rhinitis medicamentosa. C

Over-the-counter cough and cold medications for young children
73. The efficacy of cold and cough medications for symptomatic treatment of upper respiratory tract infections has not been established for children younger than 6 years. Because of the potential toxicity of these medications, the use of these over-the-counter (OTC) drugs generally should be avoided in all children below 6 years of age. A

Intranasal corticosteroids
74. Intranasal corticosteroids are the most effective medication class for controlling symptoms of allergic rhinitis. A
75. In most studies, intranasal corticosteroids have been shown to be more effective than the combined use of an antihistamine and leukotriene (LT) antagonist in the treatment of seasonal allergic rhinitis. A
76. Intranasal corticosteroids may provide significant relief of symptoms of seasonal allergic rhinitis when used not only on a regular basis but also on an as-needed basis. B However, as-needed use may not be as effective as continuous use of intranasal corticosteroids. D
77. When comparing the available intranasal corticosteroids, the overall clinical response does not appear to vary significantly between products irrespective of the differences in topical potency, lipid solubility, and binding affinity. C
78. Intranasal corticosteroids may be useful in the treatment of some forms of nonallergic rhinitis. A
79. Intranasal corticosteroids when given in recommended doses are not generally associated with clinically significant systemic side effects. A
80. Although local side effects are typically minimal with the use of intranasal corticosteroids, nasal irritation and bleeding may occur. Nasal septal perforation is rarely reported. B

Oral corticosteroids
81. A short course (5-7 days) of oral corticosteroids may be appropriate for the treatment of very severe or intractable nasal symptoms or to treat significant nasal polyposis. However, single administration of parenteral corticosteroids is discouraged and recurrent administration of parenteral corticosteroids is contraindicated because of greater potential for long-term corticosteroid side effects. D

Intranasal anticholinergics
82. Intranasal anticholinergics may effectively reduce rhinorrhea but have no effect on other nasal symptoms. Although side effects are minimal, dryness of the nasal membranes may occur. A
83. Intranasal anticholinergics may effectively reduce rhinorrhea but have no effect on other nasal symptoms. Although side effects are minimal, dryness of the nasal membranes may occur. A
84. The concomitant use of ipratropium bromide nasal spray and an intranasal corticosteroid is more effective than administration of either drug alone in the treatment of rhinorrhea without any increased incidence of adverse events. A

Oral anti-LT agents
85. Oral anti-LT agents alone, or in combination with antihistamines, have proven to be useful in the treatment of allergic rhinitis. A

Omalizumab
86. Omalizumab has demonstrated efficacy in AR; however, it has US Food and Drug Administration (FDA) approval for use only in allergic asthma. A

Nasal saline
87. There is evidence that topical saline is beneficial in the treatment of the symptoms of chronic rhinorrhea and rhinosinusitis when used as a sole modality or for adjunctive treatment. A

Allergen immunotherapy
88. Allergen immunotherapy is effective for the treatment of allergic rhinitis. A
89. Allergen immunotherapy should be considered for patients with allergic rhinitis who have demonstrable evidence of specific IgE antibodies to clinically relevant allergens, and its use depends on the degree to which symptoms can be reduced by avoidance and medication, the amount and type of medication required to control symptoms, and the adverse effects of medications. A
90. Allergen immunotherapy may prevent the development of new allergen sensitizations and reduce the risk for the future development of asthma in patients with allergic rhinitis. B

Surgery
91. Although there is no surgical treatment for allergic rhinitis, surgery may be indicated in the management of comorbid conditions, such as nasal obstruction from severe nasal septal deviation or inferior turbinate hypertrophy, adenoidal hypertrophy, or refractory sinusitis and complications thereof. C

Management decisions
92. Management and monitoring of rhinitis should be individualized and based on the spectrum, duration, and severity of symptoms; physical examination findings; comorbidities; age of the patient; and patient preferences using both step-up and step-down approaches. C
93. Effective allergic rhinitis management requires the development of a physician/patient/family partnership, avoidance of environmental triggers, and the appropriate use of prescribed therapeutic interventions. C

Education of patient and caretakers
94. Education is a key element in promoting adherence and optimizing treatment outcomes in allergic rhinitis. D

Major comorbid conditions
95. Patients with allergic rhinitis are at increased risk for the development of asthma. A
96. Treatment of allergic rhinitis may improve asthma control in patients with coexisting allergic rhinitis and asthma. B
97. There is no established cause-and-effect relationship of rhinitis with recurrent otitis media and otitis media with effusion (OME). C

Special considerations
Pregnancy
98. When selecting medications for treating rhinitis in pregnancy, the clinician might consider the FDA risk categories that are based largely on animal data and limited human studies. However, it is also beneficial to review human cohort and case-control studies as well as birth registry data before reaching a decision. C
99. The most critical time for concern about potential congenital malformation because of medication use is the first trimester, when organogenesis is occurring. D
100. A sufficient amount of human observational data has now been accumulated to demonstrate safety for second-generation as well as first-generation antihistamines. C
101. Oral decongestants should be avoided during the first trimester. Topical decongestants when used on a short-term basis may have a better safety profile than oral agents for first trimester use. C
102. Sodium cromolyn is a safe treatment for allergic rhinitis during pregnancy. C
103. Montelukast is a safe treatment for allergic rhinitis during pregnancy. C
104. Intranasal corticosteroids may be used in the treatment of nasal symptoms during pregnancy because of their safety and efficacy profile. C
105. Immunotherapy for allergic rhinitis may be continued during pregnancy but without dose escalation. C

Elderly patients
106. Rhinitis in the elderly may be caused by types of rhinitis common in other age groups but may also be influenced by age-related physiologic changes such as cholinergic hyperactivity, anatomic changes, and medications taken for other medical conditions. C

Athletes
107. Athletic performance can be affected by rhinorrhea and chronic or rebound nasal congestion. Rhinitis medication for the competitive athlete must be a US Olympic Committee (USOC) and/or International Olympic Committee (IOC)–approved product and should be one that does not adversely affect performance. C

Consultation with an allergist/immunologist
108. Allergist/immunologist care improves patient outcomes; however, consultation/referral services are often underused. C
109. Consultation with an allergist/immunologist should be considered for patients with rhinitis who have inadequately controlled symptoms, a reduced QOL and/or ability to function, adverse reactions to medications, a desire to identify the allergens to which they are sensitized and to receive advice on environmental control, or comorbid conditions such as asthma and recurrent sinusitis, or when allergen immunotherapy is a consideration. C

EXECUTIVE SUMMARY
Definition of rhinitis [Summary Statement 1]
Rhinitis is characterized by 1 or more of the following symptoms: nasal congestion, rhinorrhea (anterior and posterior), sneezing, and itching. Rhinitis is usually associated with inflammation, but some forms of rhinitis such as vasomotor rhinitis or atrophic rhinitis are not predominantly inflammatory. Rhinitis frequently is accompanied by symptoms involving the eyes, ears, and throat.

Classification and differential diagnosis of rhinitis and associated conditions [Summary Statements 2-7]
Rhinitis is classified as allergic or nonallergic, but not all types of rhinitis can be easily separated into one of these categories. For example, occupational rhinitis has been classified separately from allergic and nonallergic because it may have components of both allergic and nonallergic rhinitis. Conditions that mimic symptoms of rhinitis include nasal polyps, cerebrospinal fluid rhinorrhea, ciliary dyskinesia syndrome, and structural/mechanical factors, such as deviated septum and pharyngonasal reflux (Tables I and II).
There is no generally accepted method of grading rhinitis severity. In an attempt to do this, an international working group (Allergic Rhinitis and its Impact on Asthma [ARIA]) has proposed a classification for allergic rhinitis that placed patients into 1 of 4 categories: (1) mild intermittent, (2) mild persistent, (3) moderate/severe intermittent, and (4) moderate/severe persistent. This classification system discarded the terms seasonal and perennial, emphasizing that an allergen (eg, grass pollen) that occurs seasonally in one region may be detected throughout the year in another geographical area. The ARIA definition of mild rhinitis may be a useful comparative reference point for other severity grading schemes; this states that none of the following items is present: sleep disturbance; impairment of daily activities, leisure, and/or sport; impairment of school or work; and symptoms present but not troublesome. This updated parameter supports the concept that more severe rhinitis is defined as more symptoms or interference with QOL, because data show that it may not be
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In this document, the Joint Task Force retains and uses the terms seasonal and perennial in classifying patients with allergic rhinitis. These traditional descriptive terms are clinically useful and allow for accurate categorization of the vast majority of patients as having seasonal, perennial, or perennial allergic rhinitis with seasonal exacerbations. It has become well recognized that the traditional seasonal/perennial and ARIA schemes define different patient populations. This parameter introduces the term episodic allergic rhinitis, denoting allergic nasal symptoms elicited by sporadic exposures to inhalant aeroallergens that are not usually encountered in the patient’s indoor or outdoor environment, such as while visiting a farm where there is exposure to horses or while visiting a home with pets when a patient has no pet exposure in their own home or work environments.

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**S10** Wallace et al J ALLERGY CLIN IMMUNOL AUGUST 2008

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### Allergic rhinitis: Risk factors and presentation [Summary Statements 8-17]

**Risk factors for allergic rhinitis**

Risk factors for allergic rhinitis include (1) family history of atopy, (2) serum IgE >100 IU/mL before age 6 years, (3) higher socioeconomic class, and (4) the presence of a positive allergy SPT. The influence of early childhood exposure to infections...
(the hygiene hypothesis), animals, and secondary tobacco smoke on the development of atopy and allergic rhinitis is still unknown.17-24

Presentation of allergic rhinitis

In childhood, allergic rhinitis is more frequent in boys, but in adults, it is more frequent in women. Children with a bilateral family history of atopy may develop symptoms more frequently and at a younger age than those with a unilateral family history.6,25 Aeroallergen sensitization rarely begins before 6 months of age26 but may start between 6 months and 2 years of life.27 Infants born to atopic families are sensitized to pollen aeroallergens more frequently than to indoor aeroallergens in the first year of life.27 Seasonal allergic rhinitis symptoms generally do not develop until 2 to 7 years of age.28-30 Food ingestion rarely causes allergic rhinitis in infants, children, or adults unless there are associated gastrointestinal, dermatologic, or systemic manifestations. The prevalence of seasonal allergic rhinitis is higher in children and adolescents, whereas perennial allergic rhinitis has a higher prevalence in adults.31

Early-phase and late-phase responses in allergic rhinitis

As in patients with asthma, early-phase and late-phase responses may be seen in allergic rhinitis. Both the early-phase and late-phase responses in allergic rhinitis are characterized by symptoms of sneezing, rhinorrhea, and nasal congestion. However, nasal congestion is predominantly a late-phase response. Mediators released from eosinophils during the late phase contribute to tissue damage.32,53 Pretreatment with glucocorticoids effectively reduces eosinophils and the release of cytokines during the late-phase response.34-36

When allergen challenges are given repeatedly, the amount of allergen required to induce an immediate response decreases. This priming effect is thought to be a result of the release of inflammatory mediators from effector cells during ongoing, prolonged allergen exposure and repeated late-phase responses. Consequently, at the end of a pollen season, symptoms may decline at a slower rate than the pollen count. Therefore, it is important to know the full spectrum of aeroallergens to which the patient responds as well as seasonal variations in symptoms. Initiating anti-inflammatory therapy before pollen season or before any repetitive aeroallergen exposure, as indicated, will modify the late-phase response that is associated with the priming effect.

Allergic conjunctivitis [Summary Statements 17-19]

Oral antihistamines, intranasal antihistamines, oral anti-LT agents, intranasal corticosteroids, and allergen immunotherapy are treatments for allergic rhinitis that have been reported to relieve associated ocular allergy symptoms in controlled trials.37-52 In systematic reviews of randomized controlled studies, intranasal...
corticosteroids compared with oral antihistamines \(^{53,54}\) and intra-nasal corticosteroids compared with intranasal antihistamines \(^{46}\) were not significantly different in relieving eye symptoms.

Use of cold compresses and irrigation with saline solution or artificial tears has been advocated to relieve mild symptoms of allergic conjunctivitis. Topical ophthalmic agents are indicated for specific treatment of itching or symptoms of allergic conjunctivitis. Vasoconstrictors are indicated for relief of ocular redness, although they do not reduce the allergic response. Prolonged use of ocular decongestants may lead to rebound hyperemia, which is often referred to as “conjunctivitis medicamentosa.” \(^{55}\) Use limited to 10 days does not appear to induce this condition. \(^{56}\) The combination of an ocular antihistamine and a vasoconstrictor works better than either agent alone. \(^{57}\) Mast cell stabilizers, also approved for vernal and atopic keratoconjunctivitis, have a slow onset of action and may require several days of treatment before optimal symptom relief is achieved, \(^{58}\) making them more suitable for prophylactic or longer-term treatment of chronic ocular allergies than for acute symptom relief. Topical NSAIDs reduce prostaglandin production involved in mediating ocular allergy. \(^{59}\) Multiple-action agents possess both antihistamine and mast cell stabilizer activities, generally have onset of action within 30 minutes, and are suitable for acute and long-term treatment of allergic conjunctivitis symptoms. Ocular corticosteroids should be reserved for more severe symptoms of allergic conjunctivitis considering that the ocular side effects from their use can threaten vision because of the increased risk of cataract formation, elevated intraocular pressure (IOP), and secondary infections. The modified steroid loteprednol is indicated for the temporary relief of symptoms and signs of seasonal allergic conjunctivitis and has a greatly reduced risk of causing increased IOP compared with many other ocular corticosteroids. \(^{60-64}\)

**Vasomotor rhinitis [Summary Statements 20-22]**

Vasomotor rhinitis, a type of nonallergic rhinitis, may be episodic or perennial. \(^{7}\) The exact pathophysiology of vasomotor rhinitis has never been established, and for this reason, it is often classified as “idiopathic” rhinitis. \(^{7}\) When rhinorrhea is the predominant symptom, there appears to be enhanced cholinergic glandular secretory activity based on the fact that atropine-like agents effectively reduce secretions. \(^{65}\) Gustatory rhinitis (rhinitis symptoms associated with eating) is a form of nonallergic rhinitis felt to be vagally mediated that may respond to intranasal anticholinergic agents. \(^{66}\) Patients with predominant nasal congestion may have nociceptive neurons that have heightened sensitivity to stimuli such as temperature change, airborne irritants, foods (especially hot and spicy foods), alcoholic beverages, cold dry air, and exercise. \(^{67,70}\)

Temperature change has also been noted to increase symptoms and the inflammatory nasal response in patients with allergic rhinitis. \(^{71}\)

**Acute infectious rhinitis [Summary Statements 23, 24]**

Acute viral upper respiratory infections are the most common predisposing factor for bacterial sinusitis, accounting for 90% to 98% of all episodes of sinusitis in children and adults. \(^{72-76}\) In uncomplicated cases of viral rhinitis, a 7-day to 10-day observation period for spontaneous resolution of symptoms is recommended before prescribing antibiotics. \(^{77}\) Acute bacterial rhinosinusitis is usually associated with a viral upper respiratory infection and is characterized by symptoms persisting beyond the usual 7-day to 10-day duration of a viral infection. Careful consideration of the need for antimicrobial use is increasingly important because antibiotic use has been causally related to the development of bacterial drug resistance. \(^{78-82}\) Furthermore, the administration of antimicrobials increases the carriage of antimicrobial-resistant strains of certain bacterial pathogens, such as *Streptococcus pneumonia*, especially in children. \(^{78,79,83,84}\) Although it is generally believed that atopic children experience more episodes of acute otitis media and acute sinusitis compared with nonallergic children, this has not been firmly established. \(^{77,85,86}\) Differentiating allergic rhinitis from infectious rhinosinusitis or adenoiditis may be difficult, especially in children, because the symptoms overlap and even purulent nasal drainage may be present in noninfectious rhinitis.

**Acute and chronic sinusitis [Summary Statements 23, 24]**

Distinguishing noninfectious perennial rhinitis from acute and chronic sinusitis can be difficult because many symptoms, such as mucosal erythema, increased pharyngeal secretions, olfactory disturbance, cough, nasal congestion, and headache, are found in both types of rhinitis. Although nasal cytology may be useful in differentiating infectious from noninfectious nasal and/or sinus disease, the clinical value, particularly for the diagnosis of allergic rhinitis, is limited by low specificity and sensitivity. \(^{87-94}\) For example, neutrophils may be present not only in acute and chronic sinusitis but also in conjunction with eosinophils in patients with allergic rhinitis who also have an acute infection process. \(^{95}\) Cultures of the nasopharynx without visualization in children with rhinitis is of no value because pathogenic bacteria, as part of the normal flora, have been recovered in as many as 92% of asymptomatic healthy children. \(^{93}\) In adults, endoscopically directed middle meatus cultures have given promising results in diagnosing acute bacterial sinusitis. \(^{94-97}\)

**Nonallergic rhinitis with eosinophilia syndrome [Summary Statement 25]**

Patients with NARES have paroxysmal exacerbations of symptoms, including sneezing, profuse watery rhinorrhea, nasal pruritus, nasal congestion, and occasional anosmia. These patients are typically middle-age. The prevalence in the general population is unknown. NARES is characterized by large numbers (inconsistently defined as >5% to >20%) of eosinophils on nasal smear. \(^{98-102}\) The etiology is unknown. In some patients, NARES may precede the development of nasal polyposis and aspirin sensitivity. \(^{103}\) Patients with NARES are at increased risk for the development of obstructive sleep apnea. \(^{104}\)

**Occupational rhinitis [Summary Statement 26]**

Occupational rhinitis may be triggered by allergic factors, such as laboratory animal antigen and psyllium, \(^{105,106}\) or irritant factors, such as chemicals, grain dust, and ozone. \(^{107-109}\) Allergic occupational rhinitis frequently coexists with OA. \(^{110}\) Irritant exposures elicit neutrophilic inflammation in the nasal mucosa, \(^{107-109}\) whereas allergic exposures are associated with eosinophils, basophils, eosinophilic cationic protein (ECP), and tryptase in the nasal lavage. \(^{111-113}\) However, immunologic mechanisms may also be important in the response to chemical sensitizers, such as acid anhydrides, where both neutrophils and eosinophils are increased in nasal lavage fluid of workers with serum specific IgE to the relevant anhydride-human serum albumin (HSA) antigen. \(^{114}\) The prevalence of occupational rhinitis is essentially 100% among workers with OA who are sensitized to high-molecular-weight proteins, whereas only 50% of those with OA caused by chemicals have been identified with
work-related rhinitis. Atopy and intensity of exposure are risk factors for developing occupational rhinitis. An asymptomatic latency period of exposure lasting weeks to years often precedes work-related symptoms. Symptoms are temporally related to exposure at work and often improve away from the workplace. The diagnostic validity of nasal allergen challenge for occupational allergens has not been evaluated.

Optimal management is avoidance of the occupational trigger, but avoidance of nonoccupational allergens that contribute to the nasal symptoms is also recommended. Chronic pharmacologic therapy as used for allergic and nonallergic rhinitis can be instituted. In general, there is insufficient evidence to support the efficacy of immunotherapy for IgE-dependent occupational rhinitis, and it is inappropriate to use immunotherapy to treat occupational rhinitis caused by low-molecular-weight chemical allergens.

### Pregnancy and Menstrual Cycle Rhinitis [Summary Statement 27]

Symptoms of rhinitis during pregnancy and at the time of patients’ menstrual cycles have long been considered to be hormonally induced. The most common causes of nasal symptoms during pregnancy are allergic rhinitis, sinusitis, rhinitis medicamentosa, and vasomotor rhinitis. Symptoms of allergic rhinitis increase in 1/3 of pregnant patients, perhaps attributed to nasal vascular pooling caused by vascular dilatation and increased blood volume. A type of rhinitis unique to the pregnant patient is “vasomotor rhinitis of pregnancy” or “pregnancy rhinitis.” Pregnancy rhinitis had been defined as rhinitis without an infectious, allergic, or medication-related cause that starts before the last 6 weeks of pregnancy (corresponding to 34 weeks gestation), persists until delivery, and resolves completely within 2 weeks after delivery. When pregnancy rhinitis causes snoring, it may even be a factor in the development of pre-eclampsia. Although it is assumed that hormonal changes contribute to this condition, there is no convincing evidence. There may be an association of nasal congestion with ovulation and the rise in serum estrogen during the normal menstrual cycle in some women.

### Drug-induced Rhinitis [Summary Statement 28]

Drug-induced rhinitis may be caused by ACE inhibitors, α-receptor antagonists used in the treatment of benign prostatic hyperplasia, and phosphodiesterase-5 selective inhibitors used for treatment of erectile dysfunction. There is no direct evidence that the current combined oral contraceptive pills cause nasal symptoms. Aspirin and other NSAIDs may produce rhinorrhea as an isolated symptom or as part of aspirin-exacerbated respiratory disease (AERD), formerly termed “Samter’s triad.”

### Rhinitis Medicamentosa [Summary Statement 28]

Rhinitis medicamentosa may develop after the repetitive and prolonged use of topical α-adrenergic nasal decongestant sprays such as oxymetazoline and phenylephrine. The recreational use of cocaine may result in a rhinitis medicamentosa-like state. Benzalkonium chloride in vasoconstrictor spray products, when used for 30 days or more, may augment local pathologic effects. Patients may develop rebound congestion, tachyphylaxis, reduced mucociliary clearance because of loss of ciliated epithelial cells, and on rare occasions, nasal septal perforation. The pathophysiology of this condition is not understood. Treatment of rhinitis medicamentosa consists of suspending the use of topical decongestants and administering intranasal corticosteroids to control symptoms while allowing the rebound effects of the nasal decongestant spray to resolve. At times, a short course of oral corticosteroids may be needed to control the patients’ symptoms while the effects of the nasal decongestant spray dissipate. Once the rhinitis medicamentosa is treated, the patient should be evaluated for an underlying condition, such as allergic rhinitis.

### Atrophic Rhinitis [Summary Statement 29]

Primary (idiopathic) atrophic rhinitis is a chronic condition characterized by progressive atrophy of the nasal mucosa, nasal crusting, nasal dryness (caused by atrophy of glandular cells), and fetor. The nasal cavities appear abnormally wide on examination, and there is absence of identifiable turbinates on sinus CT, referred to as the “empty nose syndrome.” Secondary atrophic rhinitis is most commonly a result of chronic sinusitis or excessive surgery to the nasal turbinates. Although saline irrigation is the mainstay of treatment, topical or systemic antibiotics are indicated with the appearance of purulent nasal secretions.

### Conditions that Mimic Rhinitis

Conditions that mimic rhinitis must be considered in the differential diagnosis of nasal symptoms.

### Nasal Polyps [Summary Statement 30]

Nasal polyps may coexist with allergic rhinitis; however, allergy as a cause of nasal polyps has not been established. Nasal polyps have a prevalence of 2% to 4% and usually occur after age 40 years. Although previous studies showed a 2:1 male to female prevalence of nasal polyps, a recent large population study showed no sex preference. AERD, previously referred to as the aspirin triad, consists of nasal polyps, acetylsalicylic acid intolerance, and asthma, and is recognized in 13% to 40% of patients with nasal polyps. Eosinophils are a consistent finding in nasal polyp tissue. When nasal polyps are associated with asthma, there is hypersecretion of cysteinyl LTs (cys-LTs). Oral steroids may be required in severe nasal polyposis to reduce polyp size, improve airflow, and allow for effective topical medication delivery. A short course of oral steroids followed by maintenance use of intranasal corticosteroid administered twice daily should follow. One study demonstrated that after surgery, reoccurrence rates and rescue medication requirements were the same in patients treated cooperatively with montelukast or beclomethasone. In some adult patients with nasal polyps and AERD, aspirin desensitization followed by long-term daily aspirin treatment has successively reduced the need for removal of nasal polyps and systemic corticosteroids.

### Anatomic Abnormalities and Cerebral Spinal Fluid Rhinorrhea [Summary Statements 31-33]

Nasal septal deviation and turbinate hypertrophy may lead to postnasal drip or nasal obstruction. Unilateral obstruction, especially when associated with bleeding, hyposmia or anosmia, pain, and otalgia, should alert one to the possibility of a tumor. Clear rhinorrhea, even in the absence of trauma or recent surgery, may rarely be a result of a CSF leak. Nasal symptoms, particularly congestion, may be noted in infants and children with pharyngosonal reflux resulting from prematurity, neuromuscular disease, or cleft palate. In infants and children, the most common acquired anatomic cause of nasal obstruction is adenoidal hypertrophy.
Ciliary dysfunction syndromes [Summary Statement 34]

Ciliary dysfunction syndromes cause ineffective mucociliary clearance and include (1) PCD162 (also known as immotile-cilia syndrome), a rare genetic disorder, and (2) secondary ciliary dysfunction,163,164 a more common condition caused by acute or chronic infections, multiple sinus surgeries, or irritant rhinitis. In PCD, the clinical history may include recurrent sinusitis, otitis, rhinitis, chronic cough, nasal polyposis, atypical asthma that is unresponsive to therapy, and bronchiectasis.165 Approximately 50% of subjects with PCD are affected by situs inversus (Kartagener syndrome). Whereas screening diagnostic tests for mucociliary clearance use saccharine166 or Teflon (DuPont) tagged particles, definitive diagnosis requires biopsy and examination by electron microscopy.167-169 After an infection, resolution of secondary ciliary dysfunction and cytopathic epithelial damage may require weeks.163,164,170-172 An adverse effect of tobacco smoke on mucociliary clearance in the upper airways in healthy smokers has not been established.173,174

Evaluation and diagnostic studies in patients with rhinitis

History [Summary Statements 35, 36]

A thorough allergic history remains the best diagnostic tool available. The history will include the patient’s chief concerns and symptoms and often includes the pattern, chronicity, seasonality, and triggers of nasal and related symptoms, family history, current medications, response to previous treatment modalities, presence of coexisting conditions, occupational exposure, and a detailed environmental history. Questions relating symptoms to pollen and animal exposure have been shown to have positive predictive value for diagnosing allergic rhinitis.175 In addition to upper respiratory symptoms, it is important to determine the effect of rhinitis on QOL, including symptoms of fatigue, sleep disturbances, learning and attention problems, and absenteeism and presenteeism (present but with impaired function) at work and/or school.176-179

The psychological ramifications of untreated allergic rhinitis can lead to low self-esteem, shyness, depression, and anxiety.180 Recent findings that the sexual QOL is affected by seasonal allergic rhinitis and that appropriate treatment improves the patient’s sexual functioning emphasizes that allergic rhinitis is an underappreciated disease with systemic effects.181 As evidence of the disparities between patients’ and physicians’ perspectives of allergic rhinitis, the symptom severity and the reduced work, home, and social functioning, as indicators of QOL, are often underrecognized and inadequately treated by the patient’s physician.182

The effect of rhinitis on QOL has been measured using both generic and disease-specific questionnaires (Tables III and IV). Using generic QOL questionnaires, it has been shown that adults with moderate to severe perennial rhinitis and moderate to severe asthma have equal functional impairment.183,184 On the other hand, disease-specific QOL questionnaires, including those specific for rhinitis, describe disease-associated problems more accurately and seem to be more responsive to measuring the change with therapeutic interventions. Although both the generic and disease-specific QOL questionnaires are often used in research trials, their sensitivity and precision for use with individual patients have not been determined.185-190

Physical examination [Summary Statement 37]

The physical examination (Table V) of all organ systems potentially affected by allergies should be performed in all patients with a history of rhinitis. Emphasis should be on the upper respiratory tract, but the examiner should carefully look for accompanying otitis191 or eustachian tube dysfunction,192 chronic sinusitis, nasal polyps, conjunctivitis, asthma,193 and atopic dermatitis. If the patient is asymptomatic or mildly symptomatic at the time of the physical examination, there may be minimal or no findings even with a history suggestive of rhinitis.

The nasal and oropharyngeal examination may be accomplished with a nasal speculum with appropriate lighting, otoscope...
TABLE IV. Representative rhinitis QOL questionnaires

<table>
<thead>
<tr>
<th>Questionnaire</th>
<th>Web site for information</th>
<th>Cost of noncommercial use</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhinitis QOL adult</td>
<td><a href="http://www.qoltech.co.uk/Rhinoconj.htm#rqlq">http://www.qoltech.co.uk/Rhinoconj.htm#rqlq</a></td>
<td>Free request: <a href="mailto:adultrqlqpack@qoltech.co.uk">adultrqlqpack@qoltech.co.uk</a></td>
<td>643-645</td>
</tr>
<tr>
<td>Standardized Rhinoconjunctivitis Quality of Life Questionnaire</td>
<td><a href="http://www.qoltech.co.uk/Rhinoconj.htm#rqlqs">http://www.qoltech.co.uk/Rhinoconj.htm#rqlqs</a></td>
<td>Free request: <a href="mailto:adultrqlqpack@qoltech.co.uk">adultrqlqpack@qoltech.co.uk</a></td>
<td>646</td>
</tr>
<tr>
<td>Mini Rhinoconjunctivitis Quality of Life Questionnaire</td>
<td><a href="http://www.qoltech.co.uk/Rhinoconj.htm#rqlq">http://www.qoltech.co.uk/Rhinoconj.htm#rqlq</a></td>
<td>Free request: <a href="mailto:adultrqlqpack@qoltech.co.uk">adultrqlqpack@qoltech.co.uk</a></td>
<td>647</td>
</tr>
<tr>
<td>Nocturnal Rhinoconjunctivitis Quality of Life Questionnaire</td>
<td><a href="http://www.qoltech.co.uk/Rhinoconj.htm#noct">http://www.qoltech.co.uk/Rhinoconj.htm#noct</a></td>
<td>Free request: <a href="mailto:adultrqlqpack@qoltech.co.uk">adultrqlqpack@qoltech.co.uk</a></td>
<td>648</td>
</tr>
<tr>
<td>Rhinitis Quality of Life Questionnaire</td>
<td><a href="http://www.qoltech.co.uk/Rhinoconj.htm#rqlq">http://www.qoltech.co.uk/Rhinoconj.htm#rqlq</a></td>
<td>Free request: <a href="mailto:adultrqlqpack@qoltech.co.uk">adultrqlqpack@qoltech.co.uk</a></td>
<td>649</td>
</tr>
<tr>
<td>Rhinitis Symptom Utility Index</td>
<td></td>
<td></td>
<td>650</td>
</tr>
<tr>
<td>Rhinitis QOL pediatric and adolescent</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paediatric Rhinoconjunctivitis Quality of Life Questionnaire</td>
<td><a href="http://www.qoltech.co.uk/PaedRhinoconj.htm#rqlq">http://www.qoltech.co.uk/PaedRhinoconj.htm#rqlq</a></td>
<td>Free, request: <a href="mailto:paedrqlqpack@qoltech.co.uk">paedrqlqpack@qoltech.co.uk</a></td>
<td>651</td>
</tr>
<tr>
<td>Adolescent Rhinoconjunctivitis Quality of Life Questionnaire</td>
<td><a href="http://www.qoltech.co.uk/PaedRhinoconj.htm#arqlq">http://www.qoltech.co.uk/PaedRhinoconj.htm#arqlq</a></td>
<td>Free, request: <a href="mailto:paedrqlqpack@qoltech.co.uk">paedrqlqpack@qoltech.co.uk</a></td>
<td>652</td>
</tr>
</tbody>
</table>

Questionnaires have been validated for research groups but not for an individual.

TABLE V. Physical examination of patient presenting with symptoms compatible with rhinitis

**Vital signs** including weight and height should be recorded in all patients.

**General observations:** facial pallor, elongated facies, preferred mouth breathing, and any evidence of systemic disease.

**Eyes:** Excessive lacrimation, erythema and swelling of the bulbar and/or palpebral conjunctiva, cobblestoning of the tarsal conjunctiva, swelling or dermatitis of outer eyelids, Dennie-Morgan lines, or venous stasis below the lower eyelids (“allergic shiners”).

**Nose:** Reduced patency of nasal valve; alar collapse; transverse external crease; external deformity such as saddle nose; sepal deviation or perforation, spurs, ulcers, perforation, prominent vessels, or excoriation; nasal turbinate hypertrophy, edema, pallor or erythema, and crusting; discharge (amount, color, consistency), and nasal polyps. The presence of tumors or foreign bodies should be noted.

**Ears:** Tympanic membrane dullness, erythema, retraction, perforation, reduced or increased mobility, and air-fluid levels.

**Oropharynx:** Halitosis, dental malocclusion, high arched palate, tonsillar or adenoidal hypertrophy. Observe for malocclusion or high arched palate associated with chronic mouth breathing, tonsillar hypertrophy, cobblestoning of the oropharyngeal wall, pharyngeal postnasal discharge, temporomandibular joint pain or clicking with occlusion, furrowing, coating, or ulceration of tongue or buccal mucosa.

**Neck:** Lymphadenopathy, thyroid enlargement, or tenderness.

**Chest:** Signs of asthma. Chest wall deformity or tenderness, abnormal percussion, egophony, audible wheezing, or abnormal or diminished sounds by auscultation.

**Abdomen:** Tenderness, distension, masses, or enlargement of liver or spleen.

**Skin:** Rashes, especially eczematous or urticarial (distribution and description), or dermatographism.

**Other organ systems** when history or general observation indicate these should be included.

Note: This list is not intended to be totally inclusive. Elements of the examination that will assist in the differential diagnosis of rhinitis or that may indicate complications of treatment are included. Documentation of presence or absence of these elements should be considered.

with nasal adapter, indirect mirror, and/or rigid or flexible nasopharyngoscope, based on the expertise of the examiner and/or the assessment needs. If after applying a topical decongestant there is a reduction of turbinate mucosa edema, this may assist in delineating mucosal versus bony hypertrophy and in differentiating severely edematous mucosa from nasal polyps. A pneumatic otoscope is used to assess tympanic membrane mobility. At times, an impedance tympanometer is also needed to assess the tympanic membrane mobility and the presence or absence of fluid, especially in children.

Many typical allergic findings are supportive of but not specific for allergic rhinitis. Mucosal appearance may not distinguish between allergic and nonallergic noninfectious rhinitis or even infectious rhinitis, because hyperemia, for example, may be present with all 3. Likewise, classic “allergic shiners” are reported in 38% of nonatopic individuals.

**Testing for specific IgE [Summary Statements 38-41]**

Determination of specific IgE, preferably by skin testing, is indicated to (1) provide evidence of an allergic basis for the patient’s symptoms, (2) confirm suspected causes of the patient’s symptoms, or (3) assess the sensitivity to a specific allergen for avoidance measures and/or allergen immunotherapy. The number of skin tests and the allergens selected for skin testing should be determined on the basis of the patient’s age, history, and environment and living situation, such as area of the country, occupation, and activities. The precise sensitivity of specific IgE immunoassays compared with skin prick/puncture tests can vary with the technique used, from less than 50% to greater than 90%, with the average 70% to 75%. Similar sensitivity has been reported when these immunoassays are compared with symptoms induced after natural or controlled challenge—that is, nasal provocation challenge. The simplicity, ease, and rapidity of performance, low cost, and high sensitivity make skin testing preferable to in vitro testing for determining the presence of specific IgE antibodies in patients with rhinitis. However, specific IgE immunoassays may be preferable to skin testing in certain clinical situations, such as extensive skin disease, skin test suppressive therapy (for example, antihistamines) that cannot be discontinued, or uncooperative patients, or when the history suggests an unusually high risk of...
anaphylaxis from skin testing. Positive results of testing for specific IgE antibody to allergens must be correlated with history and physical findings to assess their clinical significance. A positive immediate hypersensitivity skin test in the absence of symptoms has been shown to be a significant risk factor for the later development of seasonal allergic rhinitis.

Special diagnostic techniques [Summary Statement 42]

Special diagnostic techniques may be useful in selected cases. Fiber optic nasal endoscopy may be especially useful when symptoms or physical findings are atypical, complications or other conditions are suspected, or symptoms do not respond adequately to therapy. Although CT and magnetic resonance imaging (MRI) are not indicated in the evaluation of patients with uncomplicated rhinitis, they may be useful with suspected complications or comorbidities such as nasal polyposis and/or concomitant sinusitis. MRI provides better imaging of soft tissue than CT, but it is less suited to imaging the bony anatomy. Standard radiographs are generally not indicated because of the availability of preferred procedures, as noted. When available, dynamic video rhinoscopy is more accurate at assessing adenoidal hypertrophy than a lateral nasopharyngeal radiograph.

Rhinomanometry and acoustic rhinometry [Summary Statement 42]

Rhinomanometry, a technique that measures functional obstruction to airflow in the upper airway, may be used to obtain objective assessment of nasal congestion, and may be particularly helpful in occupational rhinitis, or in assessing response to therapeutic interventions; (2) to assess the severity of anatomical abnormalities; or (3) to assist in the evaluation of patients with obstructive sleep apnea. Acoustic rhinometry reflects acoustic signals from structures in the nasal cavity, thereby producing an image that represents variations in the cross-sectional dimensions of the nasal cavity. This produces an approximate nasal cavity volume and identifies the distance to the minimal cross-sectional area from the nares. Measurement by acoustic rhinometry has been validated by comparison to CT and MRI. Using this comparison, there is high correlation for the anterior 2/3 of the nasal cavity, but the posterior nasal cavity shows more variance. Clinically, acoustic rhinometry may be of value in monitoring therapy and adherence to medical therapy as well as nasal pharyngeal surgical outcome. Although nasal congestion does not interfere with acoustic rhinometry, profuse nasal secretions may lead to measurement inaccuracy. Acoustic rhinometry is rapid, safe, and noninvasive; requires minimal patient training and cooperation; and may obviate the need of CT and MRI in some situations, such as when septoplasty and turbinoplasty are considered, as well as for postoperative evaluation.

Acoustic rhinometry and rhinomanometry have similar reproducibility and compare favorably in challenge studies, but measure nasal obstruction differently and are therefore best viewed as complementary.

Nasal provocation testing [Summary Statement 42]

Nasal allergen challenge may be used for confirmation of sensitivity to an allergen. A single allergen dose may be used to measure nasal reactivity, whereas incremental doses of allergens can be used to assess sensitivity. The clinical utility of measuring nasal sensitivity/hyperresponsiveness to histamine and methacholine is limited because of a considerable overlap in the response of patients with allergic and nonallergic rhinitis.

Nasal cytology [Summary Statement 43]

Nasal smears for eosinophils are not recommended for routine use in diagnosing allergic rhinitis, but a positive nasal smear (>10% eosinophils) may prompt nasal or conjunctival challenge when there remains a high index of suspicion of allergy in a history-positive, skin test-negative patient. A negative allergen challenge in a patient with >5% eosinophils on nasal smear would support a diagnosis of NARES. If nasal smears are obtained, nasal secretions from both nostrils should be studied. A prominence of neutrophils on nasal smear suggests an infectious process, with nasal neutrophils usually higher in bacterial than viral infections. However, the presence of neutrophils on nasal smear is not diagnostic because as many as 79% of asymptomatic school children have neutrophils in their nasal secretions.

Saccharin test and cilia biopsy [Summary Statements 44, 45]

The saccharin test for nasal mucociliary clearance can be performed in the office but has limited utility as a screening test for ciliary dyskinesia. It cannot be relied on for a definitive diagnosis of primary nasal ciliary dyskinesia but may be useful in diagnosing and following the resolution of secondary nasal ciliary dysfunction. For a definitive diagnosis of primary nasal ciliary dyskinesia, a brush biopsy is obtained from the inferior concha and examined by electron microscopy. Combining electron microscopy with computer-based image processing algorithms can improve the visualization of ultrastructural defects.

Additional laboratory testing [Summary Statements 46-48]

Laboratory studies that may be indicated in some patients with rhinitis include immune function studies and sweat test and/or genetic typing for CF. Total IgE, including cord blood samples, and specific IgG4 subclasses have limited clinical benefit and should not be routinely performed in patients with rhinitis. The presence of β-2-transferrin in the nasal secretions is a sensitive method of confirming CSF rhinorrhea.

Sleep apnea study [Summary Statement 49]

Atopy has been associated with habitual snoring in infants. In children, the presence of rhinitis is a strong predictor of habitual snoring. Children who are African American, have upper respiratory disease, and have a family history of sleep apnea are at increased risk for sleep-disordered breathing. Thus, formal evaluation for obstructive sleep apnea syndrome (OSAS) may be considered in children as well as adults presenting with chronic rhinitis and other risk factors associated with sleep-disordered breathing.

In snoring adults with rhinitis and sleep apnea symptoms, increased nasal airway resistance has been associated with apnea and hypopnea. Intranasal corticosteroids reduce nasal airway resistance and apnea-hypopnea frequency in patients with OSAS and rhinitis and may be of benefit in the treatment of some patients with OSAS.

Studies based on the link between the upper and lower airway (pulmonary function test) [Summary Statement 50]

Rhinitis and asthma are linked by common epidemiologic, physiologic, and pathologic mechanisms, as well as common comorbidities and therapeutic approaches. Therefore, it has been suggested that patients with persistent allergic rhinitis be evaluated for asthma. Because allergic rhinitis frequently
coexists with asthma and patients may not recognize symptoms of asthma, pulmonary function tests should be considered in patients with rhinitis. The presence of asthma may not be apparent because such patients (1) may have difficulty in recognizing their symptoms, (2) may not have consistent symptoms throughout the day, (3) may have a physical examination of the respiratory system that appears normal, and (4) may present with symptoms that are atypical (see Major Comorbid Conditions, Summary Statements 95-97).

**Diagnostic tests with no validity [Summary Statement 51]**

There is no evidence that the following procedures have diagnostic validity for allergic rhinitis: cytotoxic tests, provocation-neutralization, electrodermal testing, applied kinesiology, iridology, and hair analysis (see “Allergy Diagnostic Testing: An Updated Practice Parameter”

**Management of rhinitis**

**Environmental control measures for allergens [Summary Statement 52]**

The success of environmental control measures for rhinitis should be judged by clinical improvement, such as reduction in symptoms and medication scores, and not by a decrease in allergen concentration. Individual host sensitivity to an aeroallergen influences the intensity of symptoms; for example, the pollen counts that causes symptoms may vary on the basis of an individual’s degree of sensitivity and may be different for different pollens. Studies have not been consistently able to demonstrate symptom and/or medication reduction with any of the commonly used environmental control measures in patients with rhinitis.

**Pollens [Summary Statements 53, 54]**

Patients with allergic rhinitis caused by pollens may be exposed to allergen from (1) nonpollen plant fragments, (2) allergenic bioaerosols without intact pollen grains, and (3) even high pollen concentrations of insect-pollinated plants. Pollen counts are bioaerosols without intact pollen grains, and (3) even high pollen allergen concentrations that causes symptoms may vary on the basis of an individual’s degree of sensitivity and may be different for different pollens. Studies have not been consistently able to demonstrate symptom and/or medication reduction with any of the commonly used environmental control measures in patients with rhinitis.

**Fungi [Summary Statements 55, 56]**

Hydrophilic fungi, such as *Fusarium* and *Phoma*, are most abundant during rainy weather, whereas *Alternaria* and *Cladosporium* have elevated levels during dry, windy weather. When involved in plant-disturbing activity, such as gardening and lawn mowing, facemasks can reduce exposure to fungi. The first step in reduction of indoor fungal exposure consists of eliminating the source of moisture, such as water intrusion, cold surfaces, and elevated humidity. As a second step, dilute bleach solution with detergent denatures fungal allergens and may prevent regrowth with application to nonporous surfaces, whereas porous surfaces must be removed and/or replaced.

**Dust mites and cockroaches [Summary Statements 57, 59]**

Dust mite exposure can be reduced through measures that kill the mites or degrade and/or prevent their fecal pellets from becoming airborne. This may include HEPA air filtration and vacuum cleaning with a HEPA filter, low humidity, and protection on pillows and mattresses, and the use of acaricides. The patient should be encouraged to use multiple interventions because an isolated intervention, such as use of dust mite–impermeable bedding, is unlikely to offer clinical benefit. On the other hand, regular dusting and duct cleaning have not been shown to offer significant benefit. Some of these measures are also helpful for animal and insect allergen reduction, but none are as effective as removing the animal and/or insects. Cockroach allergen, a significant cause of nasal allergy in urban populations, is most abundant in the kitchen area. Environmental control of cockroach allergen involves an integrated pest management with the combination of family education—for example, emphasis on food debris removal and sealing of all sources of food and repetitive home cleaning; the use of newer gel or bait pesticides, such as odorless and colorless hydramethylnon and abamectin; and structural elimination of harborage. As with animal dander, it may take more than 6 months of aggressive pest management control to remove residual cockroach allergen.

**Animals [Summary Statement 58]**

Cat and dog allergens have been shown to produce symptoms in sensitized individuals when there is contamination of animal-free homes and schools with passive transport, such as on clothing. After cat removal from the home, an average of 20 weeks is required before the allergen concentration reaches levels found in the animal-free home. Confining a cat to an uncarpeted room (other than bedroom) with HEPA filtration may reduce by 90% airborne allergen dissemination to the remainder of the house.

**Irritants [Summary Statement 60]**

Irritants reported to cause nasal symptoms include tobacco smoke, microbiologically derived volatile organic compounds from bacteria and fungi, formaldehyde, chlorine, and perfume. The symptoms of rhinitis are directly related to the duration of exposure and usually resolve when the irritant is removed. Hyperresponsiveness to irritant triggers such as chlorine is enhanced among patients with seasonal allergic rhinitis during the season when they have symptoms. Formaldehyde, a recognized nasal and ocular irritant, produces symptoms only at concentrations well above those that produce a detectable odor.

**Pharmacologic therapy**

The selection of pharmacotherapy for a patient depends on multiple factors, including the type of rhinitis present (eg, allergic, nonallergic, mixed, episodic), most prominent symptoms, severity, and patient age (Summary Statements 92, 93). Principal medication options are summarized in Table VI. The following sections provide a more expansive discussion of medication options.

**Second-generation oral antihistamines [Summary Statements 61-64]**

Second-generation antihistamines are generally preferred over first-generation antihistamines for treatment of allergic rhinitis because they have less tendency to cause sedation, performance impairment, and/or anticholinergic adverse effects. First-generation antihistamines may produce performance impairment.
### TABLE VI. Principal medication options for rhinitis (see indicated Summary Statement [SS] discussion for supporting data) (listed in alphabetical order)

<table>
<thead>
<tr>
<th>Monotherapy</th>
<th>Therapeutic considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oral agents</strong></td>
<td></td>
</tr>
<tr>
<td>Antihistamines, oral (H1 receptor antagonists; SS 61-64)</td>
<td>Continuous use most effective for SAR and PAR, but appropriate for PRN use in episodic AR because of relatively rapid onset of action.</td>
</tr>
<tr>
<td></td>
<td>Less effective for nasal congestion than for other nasal symptoms.</td>
</tr>
<tr>
<td></td>
<td>Other options, in general, are better choices for more severe AR.</td>
</tr>
<tr>
<td></td>
<td>Less effective for AR than INS (SS 74), with similar effectiveness to INS for associated ocular symptoms (SS 19).</td>
</tr>
<tr>
<td></td>
<td>Because generally ineffective for nonallergic rhinitis, other choices are typically better for mixed rhinitis.</td>
</tr>
<tr>
<td></td>
<td>To avoid sedation (often subjectively unperceived), performance impairment, anticholinergic effects of first-generation antihistamines, second-generation agents generally preferred (SS 61).</td>
</tr>
<tr>
<td></td>
<td>Of second-generation agents, fexofenadine, loratadine, desloratadine without sedation at recommended doses (SS 63).</td>
</tr>
<tr>
<td>Corticosteroids, oral (SS 81)</td>
<td>A short course (5-7 days) of oral corticosteroids may be appropriate for very severe nasal symptoms.</td>
</tr>
<tr>
<td></td>
<td>Preferred to single or recurrent administration of intramuscular corticosteroids, which should be discouraged (SS 81).</td>
</tr>
<tr>
<td>Decongestants, oral (SS 70-72)</td>
<td>Pseudoephedrine reduces nasal congestion (SS 70).</td>
</tr>
<tr>
<td></td>
<td>Side effects include insomnia, irritability, palpitations, hypertension.</td>
</tr>
<tr>
<td>Leukotriene receptor antagonists (SS 85)</td>
<td>Montelukast approved for SAR and PAR.</td>
</tr>
<tr>
<td></td>
<td>No significant difference in efficacy between LTRA and oral antihistamines (with loratadine as usual comparator; SS 85).</td>
</tr>
<tr>
<td></td>
<td>Approved for both rhinitis and asthma; may be considered in patients who have both conditions (SS 85).</td>
</tr>
<tr>
<td></td>
<td>Side effects minimal.</td>
</tr>
<tr>
<td><strong>Intranasal agents</strong></td>
<td></td>
</tr>
<tr>
<td>Intranasal antihistamines (SS 65-69)</td>
<td>Effective for SAR and PAR (SS 65).</td>
</tr>
<tr>
<td></td>
<td>Have clinically significant rapid onset of action, making them appropriate for PRN use in episodic AR (SS 65-69).</td>
</tr>
<tr>
<td></td>
<td>Effectiveness for AR equal or superior to oral second-generation antihistamines (SS 64), with clinically significant effect on nasal congestion (SS 68).</td>
</tr>
<tr>
<td></td>
<td>Less effective than INS (SS 69) for nasal symptoms.</td>
</tr>
<tr>
<td></td>
<td>Appropriate choice for mixed rhinitis, because also approved for vasomotor rhinitis.</td>
</tr>
<tr>
<td></td>
<td>Side effects with intranasal azelastine: bitter taste, somnolence (SS 69).</td>
</tr>
<tr>
<td>Intranasal anticholinergic (ipratropium; SS 83)</td>
<td>Reduces rhinorhoea but not other symptoms of SAR and PAR.</td>
</tr>
<tr>
<td></td>
<td>Appropriate for episodic rhinitis because of rapid onset of action.</td>
</tr>
<tr>
<td></td>
<td>Side effects minimal, but dryness of nasal membranes may occur.</td>
</tr>
<tr>
<td>Intranasal corticosteroids (SS 74-80)</td>
<td>Most effective monotherapy for SAR and PAR (SS 74).</td>
</tr>
<tr>
<td></td>
<td>Effective for all symptoms of SAR and PAR, including nasal congestion.</td>
</tr>
<tr>
<td></td>
<td>PRN use (eg., &gt;50% days use) effective for SAR (SS 76).</td>
</tr>
<tr>
<td></td>
<td>May consider for episodic AR.</td>
</tr>
<tr>
<td></td>
<td>Usual onset of action is less rapid than oral or intranasal antihistamines, usually occurs within 12 hours, and may start as early as 3 to 4 hours in some patients.</td>
</tr>
<tr>
<td></td>
<td>More effective than combination of oral antihistamine and LTRA for SAR and PAR (SS 75).</td>
</tr>
<tr>
<td></td>
<td>Similar effectiveness to oral antihistamines for associated ocular symptoms of AR.</td>
</tr>
<tr>
<td></td>
<td>Appropriate choice for mixed rhinitis, because agents in class also effective for some nonallergic rhinitis.</td>
</tr>
<tr>
<td></td>
<td>Without significant systemic side effects in adults.</td>
</tr>
<tr>
<td></td>
<td>Growth suppression in children with PAR has not been demonstrated when used at recommended doses.</td>
</tr>
<tr>
<td></td>
<td>Local side effects minimal, but nasal irritation and bleeding occur, and nasal septal perforation rarely reported (SS 80).</td>
</tr>
<tr>
<td>Intranasal cromolyn (SS 82)</td>
<td>For maintenance treatment of AR, onset of action within 4 to 7 days, full benefit may take weeks.</td>
</tr>
<tr>
<td></td>
<td>For episodic rhinitis, administration just before allergen exposure protects for 4 to 8 hours against allergic response (SS 82).</td>
</tr>
<tr>
<td></td>
<td>Less effective than nasal corticosteroids, inadequate data for comparison to leukotriene antagonists and antihistamines (SS 82).</td>
</tr>
</tbody>
</table>
### TABLE VI. (Continued)

**Allergic rhinitis: Seasonal allergic rhinitis and perennial allergic rhinitis**

<table>
<thead>
<tr>
<th>Monotherapy</th>
<th>Therapeutic considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intranasal decongestants (SS 71,72)</td>
<td>Minimal side effects (SS 82) For short-term and possibly for episodic therapy of nasal congestion, but inappropriate for daily use because of the risk for rhinitis medicamentosa May assist in intranasal delivery of other agents when significant nasal mucosal edema present</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Combination therapy</th>
<th>Therapeutic considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antihistamine, oral with decongestant, oral (SS 63)</td>
<td>More effective relief of nasal congestion than antihistamines alone</td>
</tr>
<tr>
<td>Antihistamine, oral with LTRA, oral (SS 85)</td>
<td>May be more effective than monotherapy with antihistamine or LTRA Less effective than INS An alternative treatment for patients unresponsive to or not compliant with INS</td>
</tr>
<tr>
<td>Antihistamine, oral with intranasal antihistamine (SS 65-69)</td>
<td>Combination may be considered, although controlled studies of additive benefit lacking</td>
</tr>
<tr>
<td>Antihistamine, oral with intranasal corticosteroids (SS 74-77)</td>
<td>Combination may be considered, although supporting studies limited and many studies unsupportive of additive benefit of adding an antihistamine to an intranasal steroid</td>
</tr>
<tr>
<td>Intranasal anticholinergic with intranasal corticosteroid (SS 84)</td>
<td>Concomitant use of ipratropium bromide nasal spray and an intranasal corticosteroid is more effective for rhinorrhea than administration of either drug alone</td>
</tr>
<tr>
<td>Intranasal antihistamine with intranasal corticosteroid (SS 65-69)</td>
<td>Combination may be considered based on limited data Inadequate data about optimal interval between administration of the 2 sprays For mixed rhinitis, there may be significant added benefit to the combination of an intranasal antihistamine with an intranasal corticosteroid</td>
</tr>
<tr>
<td>LTRA, oral with intranasal corticosteroid (SS 85)</td>
<td>Subjective additive relief in limited studies, data inadequate</td>
</tr>
</tbody>
</table>

**Nonallergic (idiopathic) rhinitis**

<table>
<thead>
<tr>
<th>Monotherapy</th>
<th>Therapeutic considerations (for side effects, see AR table)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral agents</td>
<td>Generally ineffective for nonallergic rhinitis</td>
</tr>
<tr>
<td>Antihistamines, oral (H1 receptor antagonists; SS 61, 62)</td>
<td>Pseudoephedrine reduces nasal congestion (SS 70, 71)</td>
</tr>
<tr>
<td>Decongestants, oral (SS 70, 71)</td>
<td></td>
</tr>
<tr>
<td>Intranasal agents</td>
<td>Effective for vasomotor rhinitis</td>
</tr>
<tr>
<td>Intranasal antihistamines (SS 65-69)</td>
<td>Effective for rhinorrhea of nonallergic rhinitis syndromes</td>
</tr>
<tr>
<td>Intranasal anticholinergic (ipratropium; SS 83)</td>
<td>Special role for preventing rhinorrhea of gustatory rhinitis</td>
</tr>
<tr>
<td>Intranasal corticosteroids (SS 78)</td>
<td>Effective for some forms of nonallergic rhinitis, including vasomotor rhinitis and NARES</td>
</tr>
</tbody>
</table>

| Combination therapy | Inadequate data to provide firm recommendations in nonallergic rhinitis |

**IR, Allergic rhinitis; INS, intranasal corticosteroids; PAR, perennial allergic rhinitis; SAR, seasonal allergic rhinitis.**

in school and driving that can exist without subjective awareness of sedation, and the use of first-generation antihistamines has been associated with increased automobile and occupational accidents. Individual variation exists with respect to development of sedative effects with first-generation antihistamines. Concomitant use of other central nervous system (CNS)—active substances, such as alcohol and sedatives, may further enhance performance impairment from these antihistamines. In part because of prolonged plasma half-life and metabolites (Table VII), these undesirable and potentially dangerous side effects cannot be eliminated by administration of first-generation antihistamines only at bedtime. Anticholinergic effects include dryness of mouth and eyes, constipation, inhibition of micturition, and an increased risk for provocation of narrow-angle glaucoma. Increased sensitivity and a greater incidence of pre-existing comorbid conditions, such as prostatic hypertrophy, elevated IOP, and cognitive impairment, place older adults in a high-risk category for the side effects of first-generation antihistamines. The anticholinergic effects of the first-generation antihistamines may explain the reported better control of rhinorrhea compared with the second-generation antihistamines. The overall efficacy of first-generation antihistamines compared with second generation for the management of allergic rhinitis symptoms has not been adequately studied. Second-generation antihistamines differ in their onset of action, sedation properties, skin test suppression, and dosing guidelines (Table VIII). No single agent has been conclusively shown to have superior efficacy. Exceeding the recommended dosage may result in increased sedation with many of these products that do not produce sedation at recommended doses. Although antihistamines can be used on an intermittent basis, such as for episodic allergic rhinitis, it has been shown that continuous treatment for seasonal or perennial allergic rhinitis is more effective, primarily because of unavoidable, ongoing allergen exposure.

Intranasal antihistamines [Summary Statements 65-69] Intranasal antihistamines have demonstrated efficacy that is equal to or superior to oral second-generation antihistamines in the treatment of seasonal allergic rhinitis. They are also effective and have been associated with a clinically significant effect on nasal congestion for nonallergic rhinitis but...
<table>
<thead>
<tr>
<th>Generic drug</th>
<th>Trade name example</th>
<th>Metabolites if significant (T 1/2 in hours of product or metabolite)</th>
<th>Tmax hours (metabolite)</th>
<th>Skin test suppression mean (max) days</th>
<th>% Sedation/ somnolence or CNS impairment (control)</th>
<th>Dosage forms</th>
<th>Age limit</th>
<th>Adult dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second generation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acrivastinet</td>
<td>Semprex-D</td>
<td>(1.4-3.1)</td>
<td>1.16 (55^{53}) 1.4 (55^{53})</td>
<td>–3, T 1/2 = 1.7 h</td>
<td>8 (55^{55}) 12 (6)</td>
<td>8 mg</td>
<td>12 y</td>
<td>8 mg qid</td>
</tr>
<tr>
<td>Azelastine hydrogen chloride</td>
<td>Astelin nasal</td>
<td>Desmethylazelastine (22)</td>
<td>2.56 (55^{56}) 258 (341)</td>
<td>(2^{556,657})</td>
<td>11.5 (5.4)</td>
<td>137 mcg/spray</td>
<td>5 y</td>
<td>2 sp/nostril bid</td>
</tr>
<tr>
<td>Cetirizinet</td>
<td>Zyrtec</td>
<td>None (7-11)</td>
<td>1.0 ± 0.5</td>
<td>36</td>
<td>14 (10)</td>
<td>5, 10 mg 5 mg/5 mL</td>
<td>5 mg 2.5 mg/ 5 mL</td>
<td>5 mg q d</td>
</tr>
<tr>
<td>Desloratadinet</td>
<td>Clarinex</td>
<td>3 Hydroxy desloratadine (7.8 ± 4.2)</td>
<td>3.17 (4.76)</td>
<td>21-31 h</td>
<td>2.1 (1.8)</td>
<td>5 mg 2.5 mg/5 mL</td>
<td>6 mo</td>
<td>5 mg q d</td>
</tr>
<tr>
<td>Fexofenadinet</td>
<td>Allegra</td>
<td>None (14.4-14.6)</td>
<td>2.66</td>
<td>0.5</td>
<td>1.3 (0.9)</td>
<td>30, 60, 180 mg 30 mg/5 mL</td>
<td>2 y</td>
<td>180 mg q d</td>
</tr>
<tr>
<td>First generation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brompheniramine</td>
<td>Dimetapp</td>
<td>24.9 ± 9.3</td>
<td>4</td>
<td>&gt;2</td>
<td>24 (5)</td>
<td>12 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chlorpheniramine</td>
<td>Chlor-Trimeton</td>
<td>Mono and didesmethyl chlorpheniramine (27.9 ± 8.7)</td>
<td>2-6</td>
<td>2.8</td>
<td>45%</td>
<td>4, 8, 12 mg 2 mg/5 mL</td>
<td>2 y</td>
<td>4 mg qid</td>
</tr>
<tr>
<td>Clemastinet</td>
<td>Tavist</td>
<td>(21.3 ± 11.6)</td>
<td>4.77 ± 2.26</td>
<td>3</td>
<td>14 (1.5)</td>
<td>1.34, 2.68 mg .67 mg/5 mL</td>
<td>6 y</td>
<td>1.34 mg bid to tid</td>
</tr>
<tr>
<td>Cyproheptadinet</td>
<td>Perciactin</td>
<td>(16)</td>
<td>4</td>
<td>97 11(167)</td>
<td>8-50</td>
<td>4 mg 2 mg/ 5 mL</td>
<td>2 y</td>
<td>4 mg tid</td>
</tr>
<tr>
<td>Diphenhydraminet</td>
<td>Benadryl</td>
<td>Nordiphenhydramine (9.2 ± 2.5)</td>
<td>2.67 (67^{53}) 1.7 + 1.0</td>
<td>26</td>
<td>50%</td>
<td>25, 50 12.5 mg/mL</td>
<td>2 y</td>
<td>25-50 mg qid</td>
</tr>
<tr>
<td>Hydroxyzinet</td>
<td>Atarax</td>
<td>(20 ± 4.1)</td>
<td>2.1 ± 0.48</td>
<td>56</td>
<td>80%</td>
<td>10, 25, 50 100 mg 10 mg/5 mL</td>
<td>All ages</td>
<td>25 mg qid</td>
</tr>
<tr>
<td>Promethazine</td>
<td>Phenergan</td>
<td>Promethazine sulfoxide &amp; N-desmethy promethazine (9-16)</td>
<td>4.4675</td>
<td>36</td>
<td>60-73676</td>
<td>12.5, 25, 50 mg 6.25 mg/5 mL</td>
<td>2 y</td>
<td>25 mg qid</td>
</tr>
<tr>
<td>Triprolidinet</td>
<td>Actifed</td>
<td>(3.2)</td>
<td>2.677</td>
<td>3(654,660,661,664</td>
<td>10% to 25%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

T 1/2, Half life; \(5\), available with decongestant; \(6\), package insert; qid, 4 times a day; q d, every day; bid, 2 times a day; tid, 3 times a day.

*Onset of action, not Tmax.
are generally less effective than intranasal corticosteroids for treatment of allergic rhinitis.\textsuperscript{46} Combination therapy with intranasal corticosteroids may provide added benefit.\textsuperscript{339} The only intranasal antihistamines currently available in the United States, azelastine,\textsuperscript{332-338} and olopatadine,\textsuperscript{340} have a rapid onset of action.\textsuperscript{341} Bitter taste has been reported with both preparations, and sedation may occur (Table VII).

**Oral decongestants [Summary Statements 70, 71]**

Oral decongestants, such as pseudoephedrine, are effective at relieving nasal congestion in patients with allergic and nonallergic rhinitis but can result in side effects such as insomnia, loss of appetite, irritability, and palpitations.\textsuperscript{343} The efficacy of an oral decongestant in combination with an antihistamine in the management of allergic rhinitis has not been adequately documented to increase the efficacy of either drug alone.\textsuperscript{344}

Pseudoephedrine is a key ingredient used in making methamphetamine. In an effort to reduce illicit production of methamphetamine, restrictions have been placed on the sale of pseudoephedrine in the United States.\textsuperscript{345} This has promoted substitution of phenylephrine for pseudoephedrine in many OTC cold and cough remedies. However, phenylephrine, which appears to be less effective than pseudoephedrine, is extensively metabolized in the gut.\textsuperscript{346,347} In fact, the efficacy of phenylephrine as an oral decongestant has not been well established.\textsuperscript{345,348,349}

Elevation of blood pressure after taking an oral decongestant is very rarely noted in normotensive patients and only occasionally in patients with controlled hypertension. However, because of variation in patient response, patients receiving oral decongestants should be followed for changes in blood pressure. Concomitant use of caffeine and stimulants, such as medications used for management in attention-deficit/hyperactivity disorder, may be associated with an increase in adverse events.\textsuperscript{350} Oral decongestants should be used with caution in patients with rhinitis with certain conditions, such as cerebrovascular or cardiovascular disease, hyperthyroidism, closed-angle glaucoma, and bladder neck obstruction.

Oral decongestants, when used in appropriate doses, are usually very well tolerated in children over 6 years of age. However, use in infants and young children has been associated with agitation, psychosis, ataxia, hallucinations, and even death.\textsuperscript{351-353} (see Summary Statement 73). At times, even at recommended doses these agents may cause increased stimulatory effects resulting in tachyarrhythmias, insomnia, and hyperactivity, especially when combined with other stimulant medications, such as stimulants used in attention deficit hyperactivity disorder management.\textsuperscript{354} Therefore, the risks and benefits must be carefully considered before using oral decongestants in children below age 6 years.

**Topical decongestants [Summary Statements 71, 72]**

Topical decongestants such as phenylephrine or imidazoline derivatives—for example, oxymetazoline and xylometazoline—cause nasal vasoconstriction and decreased nasal edema but have no effect on antigen-provoked nasal response.\textsuperscript{355} Xylometazoline was found to have superior efficacy for nasal decongestion compared with intranasal corticosteroids in a 28-day study.\textsuperscript{356} However, topical decongestants are not recommended for continuous use because of the potential development of rhinitis medicamentosa.\textsuperscript{357} Furthermore, they have no effect on itching, sneezing, or nasal secretion. The development of rhinitis medicamentosa is highly variable; it may develop within 3 days of use\textsuperscript{357} or fail to develop after 6 weeks of daily use.\textsuperscript{358,360} Topical decongestants can be associated with local stinging or burning, sneezing, and dryness of the nose and throat. Intermittent use of topical decongestants is frequently prescribed; however, the efficacy and safety of this approach have not been formally studied.

**OTC cough and cold medications in young children [Summary Statement 73]**

Controlled trials have shown that antihistamine-decongestant combination products are not effective for symptoms of upper respiratory tract infections in young children.\textsuperscript{361-365} Furthermore, there has been increasing concern over the safety of OTC cough and cold medications in children. An Adverse Event Reporting System review\textsuperscript{366} showed that between 1969 and September 2006, there were 54 fatalities associated with 3 reviewed decongestants and 69 fatalities associated with 3 antihistamines found in OTC and prescription preparations. Drug overdose and toxicity were common events reported in these cases.

Currently cough and cold OTC preparations indicate the user should “consult a physician” for dosing recommendations below age 2 years for decongestants and below age 6 years for antihistamines. In mid-October 2007, the FDA’s Nonprescription Drugs and Pediatric Advisory Committees recommended that the OTC medications used to treat cough and cold no longer be used for children below 6 years of age.\textsuperscript{366} The FDA has yet to respond to these recommendations. In contrast, second-generation antihistamines

### TABLE VIII. Intranasal corticosteroid sprays

<table>
<thead>
<tr>
<th>Spray trade name</th>
<th>Generic drug</th>
<th>Type</th>
<th>mcg/spray</th>
<th>Adult dose</th>
<th>Usual child dose</th>
<th>Age limit (y)</th>
<th>Pregnancy/nursing risk category</th>
<th>Alcohol BKC propylene glycol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beconase AQ</td>
<td>Beclomethasone, monohydrate</td>
<td>Pump 200 spray</td>
<td>42</td>
<td>1-2 spray nos bid</td>
<td>1-2 sp/nos bid</td>
<td>6</td>
<td>C</td>
<td>Alcohol BKC</td>
</tr>
<tr>
<td>Flonase</td>
<td>Fluticasone propionate</td>
<td>Pump 120 spray</td>
<td>50</td>
<td>2 spray nos q d</td>
<td>1-2 sp/nos q d</td>
<td>4</td>
<td>C</td>
<td>Alcohol BKC</td>
</tr>
<tr>
<td>Nasonex</td>
<td>Mometasone</td>
<td>Pump 120 spray</td>
<td>50</td>
<td>2 spray nos q d</td>
<td>1 sp/nos q d</td>
<td>2</td>
<td>C</td>
<td>Alcohol BKC</td>
</tr>
<tr>
<td>Rhinocort AQ</td>
<td>Budesonide</td>
<td>Pump 120 spray</td>
<td>50</td>
<td>2 spray nos q d</td>
<td>1 sp/nos q d</td>
<td>2</td>
<td>C</td>
<td>Alcohol BKC</td>
</tr>
<tr>
<td>Veramyst</td>
<td>Fluticasone furoate</td>
<td>Pump 120 spray</td>
<td>50</td>
<td>2 spray nos q d</td>
<td>1 sp/nos q d</td>
<td>2</td>
<td>C</td>
<td>Alcohol BKC</td>
</tr>
<tr>
<td>Omnaris</td>
<td>Ciclesonide</td>
<td>Pump 120 spray</td>
<td>50</td>
<td>2 spray nos q d</td>
<td>NA</td>
<td>12</td>
<td>C</td>
<td>Alcohol BKC</td>
</tr>
</tbody>
</table>

* nos, Nostril; bid, 2 times a day; q d, every day; tid, 3 times a day; NA, not applicable; BKC, benzalkonium chloride.
such as cetirizine, desloratadine, fexofenadine, levocetirizine, and loratadine, when used in young children, have been shown to be well tolerated and to have a very good safety profile. 367–374

**Intranasal corticosteroids [Summary Statements 74–80]**

Intranasal corticosteroids are the most effective medications for treating allergic rhinitis. In most studies, intranasal corticosteroids are more effective than the combined use of an antihistamine and a LT antagonist. 375–379 The clinical response does not appear to vary significantly between intranasal corticosteroids that are currently available (Table VIII). 53,380–382 The onset of therapeutic effect of intranasal corticosteroid occurs between 3 and 12 hours. 383–385 The as-needed dosing (which equated to 55% to 62% of days) of an intranasal corticosteroid (fluticasone propionate) has been shown to be effective in the treatment of seasonal allergic rhinitis, 380,386,387 but may not be as efficacious as continuous use. In 1 study, PRN use of an intranasal corticosteroid (fluticasone propionate) was superior to PRN use of an oral antihistamine (loratadine) for seasonal allergic rhinitis. 380 Intranasal corticosteroids are also effective in the treatment of nonallergic rhinitis, especially NARES 388–390 and vasomotor rhinitis. 389,391,392 Intranasal corticosteroids may also benefit ocular allergy symptoms associated with allergic rhinitis (see Summary Statement 19).

Intranasal corticosteroids when given in recommended doses are not generally associated with clinically significant systemic side effects. Studies in both children and adults have failed to demonstrate any consistent, clinically relevant effect from intranasal corticosteroids on the hypothalamic-pituitary-adrenal (HPA) axis, 393,401 ocular pressure or cataract formation, 393,402–404 or bone density. 405–407 In children, growth effect may be a better indicator of systemic effect than HPA axis suppression. The transient effect on growth suppression that has been demonstrated in children after administration of intranasal corticosteroids is dependent on the specific intranasal corticosteroid, and the dose administered, technique used for measuring growth, time of administration, and concomitant use of oral or inhaled corticosteroid. Studies with intranasal fluticasone propionate, mometason furoate, and budesonide have shown no effect on growth at recommended doses compared with placebo 408–410 and reference values (at as much as 2 times the recommended doses). 400 Growth suppression from intranasal corticosteroids has been reported only with long-term use of beclomethasone dipropionate that exceeded recommended doses 409 or administration to toddlers. 411

Local side effects of intranasal corticosteroids such as nasal irritation, bleeding, and nasal septal perforation 412,413 are rare and can be avoided with proper administration technique. The patient should be periodically examined to assure that these side effects are not present. Preparations containing propylene glycol and benzalkonium chloride may result in local irritation or ciliary dysfunction, respectively 414,415 (Table VIII).

**Systemic corticosteroids [Summary Statement 81]**

Oral corticosteroids, prescribed for a few days, may be required for the treatment of very severe intractable rhinitis or nasal polyposis. 416,417 The use of parenteral and intraturbinate injections of corticosteroids is discouraged. 418–423

**Intranasal cromolyn sodium [Summary Statement 82]**

Nasal cromolyn sodium, an inhibitor of mast cell degranulation, is effective in the prevention of symptoms and in the treatment of other types of rhinitis. 424–431 It has a strong safety profile and has a reported onset of action of 4 to 7 days for seasonal or perennial rhinitis. Nasal cromolyn is effective in the treatment of episodic allergic rhinitis—for example, before anticipated allergen exposure, in which case there appears to be a more rapid onset of action. 332–343 Although cromolyn sodium is less effective than intranasal corticosteroids, it has never been adequately studied to determine its effectiveness in comparison with antihistamines or LT antagonists. 435

**Intranasal anticholinergics [Summary Statements 83, 84]**

The nasal anticholinergic ipratropium bromide is effective in reducing rhinorrhea caused by allergic rhinitis, nonallergic rhinitis (including cold-induced rhinitis 336 and gustatory rhinitis 366), and the common cold. 347,348 Ipratropium bromide is only approved (down to the age of 5 years) for the treatment of rhinorrhea, although 1 pediatric study showed modest benefit for controlling nasal congestion. 348 Ipratropium bromide has no adverse effect on physiologic nasal functions (eg, sense of smell, ciliary beat frequency, or mucociliary clearance) and has a low incidence of adverse events, in particular epistaxis and nasal dryness. 449 Concomitant use of ipratropium bromide and intranasal corticosteroid or antihistamines has an additive effect in controlling rhinorrhea. 390,442

**LT receptor antagonists [Summary Statement 85]**

LT receptor antagonists (LTRAs) are effective in the treatment of seasonal and perennial allergic rhinitis. 450–453 There is no significant difference in efficacy between LTRA and antihistamines (with loratadine as the usual comparator), and their concomitant use may be additive. 46,42,377 However, not all studies with the concomitant administration of an antihistamine and a LTRA have shown an additive effect. Although the concomitant administration of a LTRA and an antihistamine can have an additive effect, in general this approach is less efficacious than administering intranasal corticosteroids as monotherapy. 48,375,377,379 However, such combination therapy may provide an alternative treatment for patients who are unresponsive to or not compliant with intranasal corticosteroids.

Montelukast has an excellent safety profile and has been approved down to 6 months of age. As many as 40% of patients with allergic rhinitis have coexisting asthma. Because montelukast has been improved for both rhinitis and asthma, it may be considered in such patients. 454–456 The combination of montelukast and a second-generation antihistamine may protect against seasonal decrease in lung function in patients with allergic rhinitis. 457

**Omalizumab [Summary Statement 86]**

Omalizumab has been shown to have the potential for improvement in nasal and ocular symptoms as well as QOL in 1 study of patients with both seasonal and perennial allergic rhinitis. 458 However, superiority to currently approved rhinitis treatments has not been shown. In addition to the limited data of omalizumab on symptoms of rhinitis, the high cost of omalizumab treatment precludes its use for rhinitis without concomitant asthma to perennial allergens.

**Saline [Summary Statement 87]**

Although less effective than intranasal corticosteroids and no more effective than other active agents for rhinitis, isotonic and hypertonic saline solutions, used as either single or adjunctive agents, are of modest benefit for reducing symptoms and
improving the QOL in patients with allergic rhinitis and rhinosinusitis. Various mechanisms, such as improvement in mucus clearance; enhanced ciliary beat activity; removal of antigen, biofilm, or inflammatory mediators; and a protective effect on sinonasal mucosa, have been proposed but not confirmed to explain the reported symptom improvement. The use of topical saline is associated with minimal side effects, such as burning, irritation, and nausea; has low cost; and has overall good patient acceptance. The preferred method of delivery, the volume, the concentration—that is, the ratio of isotonic to hypertonic saline—and the dose frequency have not been established.

Allergen immunotherapy [Summary Statements 88-90]
Allergen immunotherapy is effective for therapy for allergic rhinitis and can potentially modify the disease. Unlike pharmacotherapy, the clinical benefits may be sustained years after discontinuation of treatment (see allergen immunotherapy practice parameter for more details). Allergen immunotherapy for allergic rhinitis may prevent the development of new allergen sensitization and reduce the risk for the future development of asthma in some patients. Immunotherapy has been associated with significant improvement in rhinitis symptom and medication scores and QOL measures as well as objective parameters such as nasal provocation challenge. Immunotherapy is usually no more costly than pharmacotherapy over the projected course of treatment.

Allergen immunotherapy should be considered for patients who have symptoms of allergic rhinitis after natural exposure to allergens and who demonstrate specific IgE antibodies to relevant allergens. There is no specific upper or lower age limitation for allergen immunotherapy. Other factors that justify consideration of immunotherapy include (1) severity and duration of symptoms, (2) responsiveness to other forms of therapy, (3) unacceptable adverse effects of medications, (4) the patient’s desire to avoid long-term pharmacotherapy, (5) reduction of the risk of future asthma, and (6) the presence of comorbid conditions, such as sinusitis or asthma. Contraindications include, for example, severe, uncontrolled asthma and significant or unstable cardiovascular disease. There should be a cautious attitude in regard to the concomitant use of β-adrenergic blocking agents and allergen immunotherapy because β-adrenergic blocking agents might make allergen immunotherapy–related systemic reactions more difficult to treat (see allergen immunotherapy practice parameter).

Clinical improvement can usually be noted after reaching the patient’s maintenance dose. Lack of improvement after 1 year of maintenance treatment should prompt a review of the patient’s immunotherapy program and possible discontinuation of immunotherapy. If allergen immunotherapy is effective, treatment may be continued for 3 years or longer. If discontinuation of effective inhalant allergen immunotherapy is considered, there are no specific tests or clinical markers currently available that will distinguish between patients who will or will not remain in long-term clinical remission. Thus, the decision to continue or stop immunotherapy must be individualized. Patients may experience local swelling at the injection site of subcutaneous immunotherapy and, on rare occasions, an anaphylactic reaction to allergen immunotherapy (refer to “Allergen Immunotherapy: A Practice Parameter Second Update” for further information regarding allergen immunotherapy).

Surgery [Summary Statement 91]
Surgery may be indicated for the management of structural/mechanical problems or comorbid conditions of allergic rhinitis, such as nasal polyps and adenoidal hypertrophy. The most common surgical procedures include (1) septoplasty, (2) reduction of inferior turbinate hypertrophy, (3) adenoidectomy, (4) functional endoscopic sinus surgery, and (5) nasal polypectomy. The reduction of nasal obstruction after surgery not only improves nasal airflow but also allows for better delivery of topical medications.

At times, the nasal congestion of rhinitis may be confused with obstruction created by structural or mechanical problems. A disturbance of normal airflow resistance and turbulent flow pattern creates the perception of nasal obstruction, regardless of the actual size of the air passage. Anterior septal deviation, with or without nasal valve collapse, and anterior inferior turbinate hypertrophy are the major structural components resulting in the symptom of nasal obstruction. Whenever there is septal deviation, typically there is compensatory turbinate hypertrophy on the opposite side. Septoplasty is infrequently performed in children because it may have a negative effect on nasal growth, particularly of the nasal dorsum.

Septoplasty, which involves reshaping, repositioning, or recontouring the cartilage, has a high reported success rate and is preferred over submucosal resection, a procedure that involves more extensive resection of cartilage and bone. Turbinate hypertrophy reduction surgery may be performed in conjunction with or separate from septoplasty, depending on the surgical assessment. The various surgical procedures available (eg, bipolar cautery or radiofrequency ablation) alleviate the mucosal hypertrophy, the bony hypertrophy (eg, submucosal resection), or a combination of bony and mucosal hypertrophy (eg, powdered turbinoplasty or laser turbinectomy). If the patient with rhinitis and coexisting turbinate hypertrophy has been unresponsive to medical therapy, a surgical evaluation can be considered.

In children, the indications for adenoidectomy are sleep apnea caused by adenotonsillar hypertrophy, chronic adenoiditis, and...
chronic sinusitis. For OME, an adenoidectomy is usually recommended after the first set of tympanostomy tubes extrudes, efusion returns, and a second set of tympanostomy tubes are being considered. An adenoidectomy may also be considered for adenoiditis, postnasal obstruction, or chronic sinusitis. Recent clinical studies recommend a trial of intranasal corticosteroids for adenoidal hypertrophy before surgical intervention.

Management decisions [Summary Statements 92, 93]

Management decisions must be individualized and guided by (1) age; (2) frequency, severity, and spectrum of symptoms (eg, predominant congestion versus rhinorrhea); (3) allergen exposure pattern; and (4) comorbidities. Response to previous treatment, patient and family preferences, compliance with therapy, and cost are additional factors that enter management decisions for the patient with rhinitis. Rhinitis medication management frequently will require consideration of a step-up approach, if therapy is inadequate, or a step-down approach, if symptom relief is achieved or maximized with other approaches, such as avoidance measures. Medications may be required only on an intermittent or short-term basis for the treatment of episodic rhinitis. The patient and physician should agree on what therapeutic approach can realistically be instituted. These therapy decisions can be committed to a Rhinitis Action Plan developed jointly with the patient and family (see Fig 5 for sample).

When a patient is compliant with the prescribed medication and yet is not responding to treatment, substitution of another class of medication can be considered. Adding another medication to the patient’s treatment regimen will not always improve the patient’s symptoms to a degree that outweighs the cost of this approach.

Appropriate follow-up for patients with rhinitis increases therapeutic success, improves compliance, and identifies complications from rhinitis or its treatment. During each follow-up patient visit, the treatment plan should be reviewed and possibly modified on the basis of physician and patient assessment of how effectively the treatment regimen is, judging from symptom control and improvement in QOL. In large part this will relate to the patient’s compliance with the agreed-on therapeutic interventions.

Education of patients and family members or other patient advocates [Summary Statement 94]

Education is a key element in promoting adherence and optimizing treatment outcomes in allergic rhinitis. Education for the patient and family members or other patient advocates begins at the initial encounter and continues at following visits. The education program should emphasize the chronicity of rhinitis as a disease, the realistic outcome of therapy, an understanding of how to implement appropriate environmental changes, appropriate methods of medication administration, medication benefits and possible side effects, the comorbidity of other allergic diseases, and the effect that disease control can make in overall improvement in QOL.

Although it is recognized that education is important for rhinitis, the best delivery method, frequency, and educational setting have yet to be determined. One-on-one allergy treatment educational sessions about rhinitis treatment may not be any more effective than a handout. Reduced use of medication, reduced office visits, or improvement in QOL has not been consistently shown when educational programs are implemented for rhinitis. Whatever rhinitis educational delivery method is selected, it is important to review the content of the educational material.

Major comorbid conditions

Asthma [Summary Statements 95, 96]

Patients with allergic rhinitis are at increased risk of developing asthma. Patients with allergic rhinitis without asthma, especially those sensitized to dust mites, often have nonspecific bronchial hyperresponsiveness (BHR), and many patients with seasonal allergic rhinitis experience a seasonal increase in BHR. Conversely, subsegmental bronchial allergen challenge in patients with allergic rhinitis has been shown to result in both bronchial and nasal inflammatory responses. It has been suggested that in patients with moderate to severe allergic rhinitis, especially those with longstanding rhinitis and sensitization to dust mites, a reduced forced expiratory flow at 25% to 75% of forced vital capacity may be a marker of early bronchial pathology. Treatment with intranasal corticosteroids has been shown to prevent the seasonal increase in BHR experienced by patients with allergic rhinitis, to reduce existing BHR, and to improve pulmonary function tests. Allergen immunotherapy for allergic rhinitis may reduce the development of asthma in children and possibly in adults. Treatment of allergic rhinitis with intranasal corticosteroids and certain second-generation antihistamines may improve asthma control when both diseases coexist.

Recurrent OME [Summary Statement 97]

Recurrent otitis media and OME are frequently associated with allergic rhinitis. Eustachian tube dysfunction remains the most common etiology for otitis media. Mediators released after allergen exposure result in nasal allergic inflammation and contribute to the dysfunction of the eustachian tube by producing eustachian tube edema and inflammation. Under natural circumstances, the middle ear is not exposed to allergens. However, measurements of elevated ECP, IL-5, and IgA within the middle ear during chronic OME support a localized inflammatory process. Similar cytokine and cellular profiles have been noted concurrently in the middle ear and nasopharynx of atopic patients. These findings suggest that the ear may be part of the unified airway in atopic patients.

Special considerations

Pregnancy [Summary Statements 98–105]

When selecting medications for the pregnant patient, the FDA pregnancy risk categories (Table X) should be considered. However, these are based largely on animal studies with limited human data. Therefore, human cohort and case-control studies as well as birth registry data should be reviewed before making a medication selection. Concern about the potential for congenital malformation because of medication use occurs primarily during the first trimester, when organogenesis is occurring.

First-generation antihistamines have previously been recommended as first-choice agents because of their observed safety and longevity of use. However, in general, their sedative and impaired performance characteristics make them less desirable choices than second-generation antihistamines. The accumulated safety data during pregnancy on the second-generation antihistamines are comparable to those of the first-generation antihistamines (Tables XI and XII). Although there are no reports of increased congenital malformations with the use of fexofenadine during pregnancy and animal studies are negative for
teratogenicity, no epidemiologic studies in human pregnancy have been published.594 Currently, there are also limited data on desloratadine, azelastine, and levocetirizine. Although diphenhydramine is frequently used during pregnancy and has good overall safety data, administration of diphenhydramine has been associated with the development of cleft palate.595-599 Hydroxyzine should be used cautiously during the first trimester based on animal data.594

Oral decongestants should be avoided, if possible, during the first trimester because of conflicting reports of an association of phenylephrine and pseudoephedrine with congenital malformations such as gastroschisis and small intestinal atresia.594,600

Hydroxyzine

<table>
<thead>
<tr>
<th>Antihistamines</th>
<th>Nasal Corticosteroids</th>
<th>Oral Decongestants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allegra (fexofenadine)</td>
<td>D mg tab</td>
<td>Syrup</td>
</tr>
<tr>
<td>Claritin (loratadine)</td>
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</tr>
<tr>
<td>Clarinex (desloratadine)</td>
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<tr>
<td>Xyzal (levocetirizine)</td>
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<td>Zyrtec (cetirizine)</td>
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</tr>
<tr>
<td>Astelin sp/nostril</td>
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<td>Syrup</td>
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<tr>
<td>Combinations</td>
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<tr>
<td>Oral decongestants</td>
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<td>Syrup</td>
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<tr>
<td>NasalCrom (cromolyn)</td>
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<tr>
<td>Parnel</td>
<td>phenylephrine</td>
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<th>Rhinitis Steps</th>
<th>What to do</th>
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<td>Step 1: Episodic</td>
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<td>Decongestant Naq Oral Nasal</td>
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<td>Eye Drops</td>
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<td>dose(s)</td>
</tr>
<tr>
<td>Atrovent</td>
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</tr>
<tr>
<td>Atrovent</td>
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</tbody>
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**FIG 5. Sample rhinitis action plan.**
TABLE X. FDA pregnancy risk categories

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<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Adequate studies in pregnant women have not demonstrated a risk to the fetus in the first trimester of pregnancy, and there is no evidence of risk in later trimesters.</td>
</tr>
<tr>
<td>B</td>
<td>Animal studies have not demonstrated a risk to the fetus, but there are no adequate studies in pregnant women.</td>
</tr>
<tr>
<td>OR</td>
<td>Animal studies have shown an adverse effect, but adequate studies in pregnant women have not demonstrated a risk to the fetus in the first trimester of pregnancy, and there is no evidence of risk in later trimesters.</td>
</tr>
<tr>
<td>C</td>
<td>Animal studies have shown an adverse effect on the fetus, but there are no adequate studies in human beings/animals benefits from the use of the drug in pregnant women may be acceptable despite its potential risks.</td>
</tr>
<tr>
<td>OR</td>
<td>There are no animal reproduction studies and no adequate studies in human beings.</td>
</tr>
<tr>
<td>D</td>
<td>There is evidence of human fetal risks, but the potential benefits from the use of the drug in pregnant women may be acceptable despite its potential risks.</td>
</tr>
<tr>
<td>X</td>
<td>Studies in animals or human beings demonstrate fetal abnormalities, or adverse reaction reports indicate evidence of fetal risk. The risk of use in a pregnant woman clearly outweighs any possible benefit.</td>
</tr>
</tbody>
</table>

Allergen immunotherapy for allergic rhinitis may be continued during pregnancy if it is effective and not causing significant reactions.614,615 The immunotherapy doses that the patient receives when she becomes pregnant should not be increased and should be adjusted appropriately during pregnancy if necessary to minimize the chance of inducing a systemic reaction. The benefit/risk considerations do not generally favor starting immunotherapy during pregnancy.50

Elderly patients [Summary Statement 106]

Rhinitis in the elderly may be influenced by age-related physiologic changes (eg, cholinergic hyperactivity), anatomic changes, and/or medications taken for other medical conditions. Many of the pathological changes in connective tissue and vasculature associated with aging may predispose to rhinitis symptoms.616,617 These changes can result in dryness of the mucus membranes and increased nasal congestion in some elderly patients. Intranasal corticosteroids may be safely used for treatment of allergic rhinitis in the elderly because they do not cause any clinical or histological atrophic changes in the nasal mucosa.618

Athletes [Summary Statement 107]

Athletes with rhinitis can have their performance affected by rhinorrhea and nasal congestion. Endurance athletes, such as long-distance runners or triathletes, may experience rebound nasal congestion after the initial vasoconstriction that naturally occurs with exercise.619 Prescription of medication for the competitive athlete should be based on 2 important principles180: (1) no medication given to the athlete should be on any list of doping products and should be approved for use by the USOC (www.wada-ama.org) and IOC (1-800-233-0393)550; and (2) no medication should adversely affect the athlete’s performance.620 Intranasal corticosteroids and topical decongestants are approved by the USOC, but all oral decongestants are banned. Although antihistamines are approved for use by the USOC, some are banned by the IOC.

Consultation with an allergist/immunologist [Summary Statements 108, 109]

Studies have shown that consultation with an allergist/immunologist improves patient outcomes, including QOL, compliance, and satisfaction.621-624 by providing education on rhinitis and allergen avoidance. Consultation with an allergist/immunologist should be considered when any of the following are present:

1. The patient has had prolonged manifestations of rhinitis.
2. The patient has complications of rhinitis, such as otitis media, sinusitis, and/or nasal polypsis.
3. The patient has a comorbid condition, such as asthma.
4. The patient has required systemic corticosteroids for the treatment of rhinitis.
5. The patient’s symptoms or medication side effects interfere with his/her ability to function, such as causing sleep disturbance or impairing school/work performance.
6. The patient’s symptoms significantly decrease QOL, such as a decrease in comfort and well being, sleep disturbance, anosmia, or ageusia.
7. Treatment with medications for rhinitis is ineffective or produces adverse events.
8. The patient has been diagnosed with rhinitis medicamentosa.
9. Allergic/environmental triggers causing the patient’s rhinitis symptoms need further identification and clarification.
10. There is a need for more complete education.

risks of such malformations have been reported to be increased by combining a decongestant with acetaminophen or salicylates.600,601 The safety of intranasal decongestants during pregnancy has not been studied.

Sodium cromolyn and montelukast are both Pregnancy Category B drugs. Sodium cromolyn is a safe treatment for allergic rhinitis in pregnancy with the previously discussed clinical limitations.599,602-604 Montelukast has reassuring animal reproductive studies and unpublished human safety data. A published observational study including 9 patients on LTRAs (specific agent not identified) demonstrated no adverse events.605 This drug could be considered if there has been a favorable prepregnancy response.

Intranasal corticosteroids may be used during pregnancy because of their safety and efficacy profile. Clinical and epidemiologic studies on safety in human beings are limited. Although most intranasal corticosteroids are given an FDA Pregnancy Category C rating, gestational risk has not been confirmed in observational human data.603 A recent meta-analysis concluded that the use of intranasal corticosteroids during pregnancy does not increase the risk of major malformations, preterm delivery, low birth weight, and pregnancy-induced hypertension.606 The reported safety data on all intranasal corticosteroids have been reassuring, but beclomethasone,11,602,607-609 budesonide (Pregnancy Category B),603,610 and fluticasone propionate611-613 have more accumulated data than triamcinolone,612,613 mometasone, and flunisolide.611 Because no substantial difference in efficacy and safety has been shown among the available intranasal corticosteroids, it would be reasonable to continue any of the intranasal corticosteroids that have adequately controlled the patient’s symptoms before pregnancy.609,614 If intranasal corticosteroids are started during pregnancy, intranasal budesonide, which is Pregnancy Category B, largely on the basis of extensive human safety data, may be preferred.603,610 As with all medication use in pregnancy, intranasal corticosteroids should be administered at the lowest effective dose.

Studies have shown that consultation with an allergist/immunologist improves patient outcomes, including QOL, compliance, and satisfaction.621-624 by providing education on rhinitis and allergen avoidance. Consultation with an allergist/immunologist should be considered when any of the following are present:

1. The patient has had prolonged manifestations of rhinitis.
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3. The patient has a comorbid condition, such as asthma.
4. The patient has required systemic corticosteroids for the treatment of rhinitis.
5. The patient’s symptoms or medication side effects interfere with his/her ability to function, such as causing sleep disturbance or impairing school/work performance.
6. The patient’s symptoms significantly decrease QOL, such as a decrease in comfort and well being, sleep disturbance, anosmia, or ageusia.
7. Treatment with medications for rhinitis is ineffective or produces adverse events.
8. The patient has been diagnosed with rhinitis medicamentosa.
9. Allergic/environmental triggers causing the patient’s rhinitis symptoms need further identification and clarification.
10. There is a need for more complete education.
11. The patient has required multiple and/or costly medications over a prolonged period.

12. Allergy immunotherapy is a treatment consideration. Consultation with an allergist/immunologist may be indicated in other situations when there is agreement between the patient and the referring physician that such an approach is in the patient’s best interests.

**ANNOTATIONS**

**Box 1: Patient presents with symptoms of rhinitis (Fig 6)**

Patients with rhinitis can present with symptoms of rhinorrhea, nasal congestion, sneezing, nasal pruritus, postnasal drainage, and/or associated ocular symptoms. These symptoms can occur with both allergic and nonallergic rhinitis. Symptoms of allergic rhinitis may occur only during specific seasons, may be perennial without seasonal exacerbations, may be perennial with seasonal exacerbations, or may occur episodically after specific aeroallergen exposures. Conjunctival symptoms frequently occur in conjunction with allergic rhinitis. Rhinitis symptoms often worsen during complications, such as otitis media and sinusitis, and frequently coexist with symptoms of other comorbid conditions, such as wheezing, cough, and chest tightness caused by asthma. Patients may be initially evaluated either by a generalist, such as a primary care physician, or by a specialist, such as an allergist/immunologist.

The history should include (1) the nature of the presenting symptoms, such as rhinorrhea, nasal congestion, sneezing, and

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**TABLE XI. Antihistamines in pregnancy first trimester H1 first-generation antihistamines**

<table>
<thead>
<tr>
<th>H1 antihistamine study drug (FDA pregnancy category)</th>
<th>Reference</th>
<th>Study type</th>
<th>Study group (n)</th>
<th>Control group (n)</th>
<th>Specific H1%</th>
<th>RR (CI)</th>
<th>All H1%</th>
<th>Control%</th>
<th>Congenital malformations</th>
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</thead>
<tbody>
<tr>
<td>Chlorpheniramine (B)</td>
<td>682</td>
<td>Prospective</td>
<td>23</td>
<td>929</td>
<td>Major 0%</td>
<td>NA</td>
<td>4%</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>Chlorpheniramine (B)</td>
<td>598</td>
<td>Collaborative project</td>
<td>1070</td>
<td>49,212</td>
<td>8.4%</td>
<td>1.2 (.98, 1.46)</td>
<td>6.4%</td>
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<tr>
<td>Chlorpheniramine (B)</td>
<td>597</td>
<td>Cohort retrospective</td>
<td>257</td>
<td>6252</td>
<td>1.56%</td>
<td>.96 (.36, 2.6)</td>
<td>1.6%</td>
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<tr>
<td>Brompheniramine (C)</td>
<td>598</td>
<td>Collaborative project</td>
<td>65</td>
<td>50,217</td>
<td>5.4%</td>
<td>2.34 (1.31, 4.17)</td>
<td>6.4%</td>
<td></td>
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<tr>
<td>Brompheniramine (C)</td>
<td>597</td>
<td>Cohort retrospective</td>
<td>172</td>
<td>6337</td>
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<td>1.6%</td>
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<tr>
<td>Brompheniramine (C)</td>
<td>683</td>
<td>Meta-analysis</td>
<td>34</td>
<td>34</td>
<td>2.9%</td>
<td>.5 (.98, 1.26)</td>
<td>1.6%</td>
<td></td>
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<tr>
<td>Triprolidine (C)</td>
<td>597</td>
<td>Cohort retrospective</td>
<td>244</td>
<td>6265</td>
<td>1.2%</td>
<td>.76 (2.2, 2.36)</td>
<td>1.6%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triprolidine (C)</td>
<td>684</td>
<td>Cohort retrospective</td>
<td>384</td>
<td>6452</td>
<td>1.56%</td>
<td>1.36 (6.31)</td>
<td>1.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clemastine (B)</td>
<td>685</td>
<td>Birth registry</td>
<td>1239</td>
<td>16,967</td>
<td>3.17%</td>
<td>.98 (.72, 1.33)</td>
<td>3.45%</td>
<td>3.24%</td>
<td></td>
</tr>
<tr>
<td>Hydroxyzine (C)</td>
<td>598</td>
<td>Collaborative project</td>
<td>50</td>
<td>50,232</td>
<td>10%</td>
<td>1.57 (.68, 3.62)</td>
<td>6.4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triprolidine (C)</td>
<td>686</td>
<td>Prospective double blind</td>
<td>74</td>
<td>34</td>
<td>1.35%</td>
<td>1.4 (.06, 33.51)</td>
<td>0%</td>
<td></td>
<td></td>
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<tr>
<td>Triprolidine (C)</td>
<td>687</td>
<td>Prospective</td>
<td>43</td>
<td>44</td>
<td>13.6%</td>
<td>3.07 (.66, 14.38)</td>
<td>4.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triprolidine (C)</td>
<td>682</td>
<td>Prospective</td>
<td>20</td>
<td>929</td>
<td>5%</td>
<td>1.67</td>
<td>4%</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>Diphenhydramine (C)</td>
<td>598</td>
<td>Collaborative project</td>
<td>595</td>
<td>49,687</td>
<td>8%</td>
<td>1.25 (.95, 1.64)</td>
<td>6.9%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diphenhydramine (C)</td>
<td>595</td>
<td>Drug registry, partly prospective</td>
<td>599</td>
<td>599</td>
<td>3.3%</td>
<td>1.56% (1.25, 1.94)</td>
<td>1.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diphenhydramine (C)</td>
<td>684</td>
<td>Cohort retrospective</td>
<td>361</td>
<td>6476</td>
<td>.27%</td>
<td>.23 (.03, 1.63)</td>
<td>1.2%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diphenhydramine (C)</td>
<td>597</td>
<td>Cohort retrospective</td>
<td>270</td>
<td>6239</td>
<td>1.5%</td>
<td>.92 (.34, 2.47)</td>
<td>1.6%</td>
<td></td>
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</tr>
</tbody>
</table>

**TABLE XII. Antihistamines in pregnancy first trimester H1 second-generation antihistamines live birth data**

<table>
<thead>
<tr>
<th>H1 antihistamine study drug (FDA pregnancy category)</th>
<th>Reference</th>
<th>Study type</th>
<th>Study group (n)</th>
<th>Control group (n)</th>
<th>Specific H1%</th>
<th>RR (CI)</th>
<th>All H1%</th>
<th>Control%</th>
<th>Congenital malformations Cardiac</th>
<th>Spontaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cetirizine (B)</td>
<td>685</td>
<td>Birth registry</td>
<td>917</td>
<td>403,545</td>
<td>3.95% (NS)</td>
<td>1.22 (.89, 1.69)</td>
<td>3.45%</td>
<td>3.16%</td>
<td>Major 0%</td>
<td>1.15 (.17, 7.73)</td>
</tr>
<tr>
<td>Cetirizine (B)</td>
<td>687</td>
<td>Prospective</td>
<td>33</td>
<td>38</td>
<td>Major 0%</td>
<td>1.15 (.17, 7.73)</td>
<td>Major 0%</td>
<td>1%</td>
<td>.4%</td>
<td>18% (NS)</td>
</tr>
<tr>
<td>Loratadine (B)</td>
<td>685</td>
<td>Birth registry</td>
<td>1769</td>
<td>408,545</td>
<td>3.4% (NS)</td>
<td>1.05 (.38, 1.34)</td>
<td>3.45%</td>
<td>3.16%</td>
<td>Minor 6%</td>
<td>.57 (.06, 5.39)</td>
</tr>
<tr>
<td>Loratadine (B)</td>
<td>688</td>
<td>Prospective</td>
<td>140</td>
<td>149</td>
<td>3.5% (NS)</td>
<td>.93 (.48, 1.79)</td>
<td>4%</td>
<td>13% (NS)</td>
<td>8%</td>
<td></td>
</tr>
<tr>
<td>Loratadine (B)</td>
<td>682</td>
<td>Prospective</td>
<td>175</td>
<td>844</td>
<td>Major 2.3%</td>
<td>.77 (.27, 2.19)</td>
<td>4%</td>
<td>11% (NS)</td>
<td>7.2%</td>
<td></td>
</tr>
<tr>
<td>Terfenadine (C)*</td>
<td>685</td>
<td>Birth registry</td>
<td>1162</td>
<td>408,545</td>
<td>3.22% (NS)</td>
<td>.98 (.72, 1.35)</td>
<td>3.45%</td>
<td>3.16%</td>
<td>Minor 5%</td>
<td>.57 (.06, 5.39)</td>
</tr>
<tr>
<td>Terfenadine (C)*</td>
<td>689</td>
<td>Prospective multicenter</td>
<td>118</td>
<td>118</td>
<td>Major 0%</td>
<td>.57 (.06, 5.39)</td>
<td>2%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**NS,** Not significant; **RR,** relative risk.

*/No longer available.

†The active metabolite of terfenadine.

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11. The patient has required multiple and/or costly medications over a prolonged period.

12. Allergy immunotherapy is a treatment consideration.

Consultation with an allergist/immunologist may be indicated in other situations when there is agreement between the patient and the referring physician that such an approach is in the patient’s best interests.
associated ocular symptoms; (2) length of symptomatology; (3) the current and past medications used for treatment of rhinitis, including duration, effectiveness, and any associated adverse events; (4) current or past medications taken for other medical conditions and the relationship, if any, with rhinitis symptoms; (5) the degree to which the patient’s rhinitis symptoms interfere with the patient’s QOL; (6) seasonality of nasal and related symptoms (or lack thereof); (7) occupational exposure; (8) a detailed environmental history; (9) identification of precipitating factors; (10) the presence of other medical conditions; (11) presence of symptoms consistent with complications, such as sinusitis or otitis media, or comorbid conditions, such as asthma; (12) family history of allergic rhinitis, asthma, or atopic dermatitis; and (13) personal or family history of chronic sinus problems or infections, as well as diagnoses that may represent allergic symptoms, such as recurrent bronchitis.

When reviewing the allergic history in children, one may inquire about sniffing, snorting, clearing of the throat, chronic gaping mouth, halitosis, cough, dark circles under the eyes, and eye rubbing. The parents may describe the child as having a poor appetite, learning or attention problems, sleep disturbances, malaise, irritability, and a general sense of not feeling well.

The physical examination should focus on examination of the nose but may include evaluation of the ears, eyes, throat, and lungs. Examination of the nose should focus on the appearance of the nasal mucous membranes, the patency of the nasal passages, unilateral or bilaterality of findings, causes for anatomical nasal obstruction, and the quality and quantity of the nasal discharge.
**Box 2: Is history and examination suggestive of allergic rhinitis?**

A diagnosis of allergic rhinitis can be confirmed only on the basis of a history of symptoms after exposure to known allergens, which correlates with specific IgE testing. Nonetheless, the history and physical examination alone is often suggestive of either allergic rhinitis or nonallergic rhinitis. Symptoms of pruritus and sneezing are much more common in allergic than nonallergic rhinitis. Seasonal exacerbations are also suggestive of allergic rhinitis. Patients with allergic rhinitis tend to develop the onset of symptoms earlier in life, typically before the age of 20 years, than those with nonallergic rhinitis. In contrast, isolated postnasal drainage is less likely to be a result of allergic rhinitis. Patients with vasomotor rhinitis may have symptoms triggered by strong odors such as perfume or tobacco smoke. A history of isolated rhinorrhea associated with eating is suggestive of gustatory rhinitis. Patients with chronic and frequent use of topical decongestant sprays may have rhinitis medicamentosa. Symptoms that are primarily unilateral suggest a structural problem, such as a nasal polyp, foreign body, septal deformity, or rarely a tumor. Hyposmia or anosmia are often associated with nasal polyposis but may also occur in other forms of rhinitis.

Many typical allergic findings are supportive of but not specific to allergic rhinitis. Mucosal appearance may not distinguish between allergic and nonallergic rhinitis, because nonallergic rhinitis may also present with mucosal pallor, edema, or hyperemia. However, the physical examination can help identify nasal polyps, foreign bodies, or other structural abnormalities.

**Box 3: Therapeutic trial for allergic rhinitis symptoms**

Initial treatment of nonsevere rhinitis may include single-agent or combination pharmacologic therapy and avoidance measures.

Oral antihistamines are generally effective in reducing rhinorrhea, sneezing, and itching associated with allergic rhinitis but have little objective effect on nasal congestion. These agents may reduce symptoms of allergic conjunctivitis, which are often associated with allergic rhinitis. Antihistamines have a limited role in treating nonallergic rhinitis syndromes. Although antihistamines can be used on an intermittent basis, such as for episodic allergic rhinitis, it has been shown that continuous treatment for seasonal or perennial allergic rhinitis is more effective, primarily because of unavoidable, ongoing allergen exposure. First-generation antihistamines have significant potential to cause sedation, performance impairment (that may not be subjectively perceived by patients), and/or anticholinergic effects (such as dry mouth and urinary retention). Consequently, second-generation antihistamines, which are associated with less risk or no risk for these side effects, are generally preferred over first-generation antihistamines for the treatment of allergic rhinitis. Intranasal antihistamines may be useful alternatives to oral antihistamines but may cause sedation in some patients and/or may be perceived to have a bitter taste.

Oral anti-LT agents, alone or in combination with antihistamines, have proven to be useful in the treatment of allergic rhinitis. There is no significant difference in efficacy between LTRA and antihistamines (with loratadine as the usual comparator), and their concomitant use may be additive, but not all studies with the concomitant administration of an antihistamine and a LTRA have shown an additive effect. Although the concomitant administration of a LTRA and an antihistamine can have an additive effect, in general, this approach is less efficacious than administering intranasal corticosteroids.

Oral decongestants, such as pseudoephedrine or phenylephrine, help reduce symptoms of nasal congestion in both allergic and nonallergic rhinitis and are beneficial for use in combination with antihistamines. However, they can cause insomnia, loss of appetite, irritability, and palpitations. Elevation of blood pressure after taking an oral decongestant is very rarely noted in normotensive patients and only occasionally in patients with controlled hypertension. However, based on interindividual variation in response, hypertensive patients should be monitored.

Topical decongestants are appropriate to use on a short-term basis for nasal congestion associated with acute bacterial or viral infections, exacerbations of allergic rhinitis, and eustachian tube dysfunction. Intermittent use of topical decongestants may be considered, but efficacy and safety of this approach have not been formally studied. With regular daily use, some patients may develop rhinitis medicamentosa in 3 days, whereas others may not have evidence of rebound congestion after 4 to 6 weeks of use. Given this variability, it would be prudent to instruct patients of the risk of rhinitis medicamentosa when intranasal decongestants are used more than 3 days.

Intranasal corticosteroids are typically the most effective medication class for controlling sneezing, itching, rhinorrhea, and nasal congestion, the 4 major symptoms of allergic rhinitis. They are particularly useful for treatment of more severe allergic rhinitis and may be useful in some forms of nonallergic rhinitis. Intranasal corticosteroids when given in recommended doses are not generally associated with clinically significant systemic side effects. Although local side effects are minimal, if the patient is carefully instructed in the use of this class of drugs, nasal irritation and bleeding may occur. Patients should be instructed to direct sprays away from the nasal septum. The nasal septum should be periodically examined to assure that there are no mucosal erosions. Although nasal septal perforations are rarely caused by intranasal corticosteroids, mucosal erosions may suggest an increased risk for their subsequent development. In children, intranasal corticosteroids should be used at the lowest effective dose. Intranasal corticosteroids may be considered for initial treatment without a previous trial of antihistamines and/or oral decongestants, and they should always be considered before initiating treatment with systemic corticosteroids for the treatment of rhinitis.

A short course (5-7 days) of oral corticosteroids may be appropriate for the treatment of very severe or intractable rhinitis or nasal polyposis. However, single administration of parenteral corticosteroids is discouraged, and recurrent administration of parenteral corticosteroids is contraindicated because of greater potential for long-term corticosteroid side effects.

Nasal cromolyn is less effective than intranasal corticosteroids. It can reduce symptoms of allergic rhinitis in some patients and is most likely to be effective if initiated before symptoms become severe. For maximum efficacy, nasal cromolyn should be administered 4 times a day.

Intranasal anticholinergics may effectively reduce rhinorrhea but have minimal effects on nasal congestion or other nasal symptoms. The combination of intranasal anticholinergics with either antihistamines or intranasal corticosteroids may provide...
increased efficacy over either drug alone without any increased adverse effects.

Empiric avoidance of suspected inciting factors, such as allergens, irritants, and medications, should be implemented, if possible, even in early treatment of rhinitis. In the management of severe seasonal allergic rhinitis, patients should be advised to follow avoidance measures such as staying inside air-conditioned buildings, whenever possible, with windows and doors closed.

**Box 4: Therapeutic trial for nonallergic rhinitis symptoms**

Many of the medications used to treat allergic rhinitis are also used in the management of nonallergic rhinitis. Intranasal corticosteroids and intranasal antihistamines may relieve both congestion and rhinorrhea associated with vasomotor rhinitis. Intranasal anticholinergics are useful in nonallergic rhinitis with predominant rhinorrhea (eg, gustatory rhinitis). Nonsedating oral antihistamines have not been shown to be effective in nonallergic rhinitis. Oral and intranasal decongestants may be considered in patients with nonallergic rhinitis and nasal congestion with similar precautions as discussed. Avoiding aggravating irritants may be helpful, particularly in patients suspected to have vasomotor rhinitis. For patients with rhinitis medicamentosa, discontinuation of nasal decongestant sprays and treatment with either intranasal or systemic corticosteroids may be necessary. Finally, patients suspected of infectious rhinitis should be treated with supportive measures to relieve ostiomeatal obstruction and judicious use of antibiotics for suspected bacterial sinusitis.

**Box 5: Does the patient respond?**

In assessing response to therapy, a variety of parameters should be evaluated.

These include nasal symptoms (eg, congestion, itching, and rhinorrhea), physical signs of rhinitis (eg, edema of nasal turbinates), and QOL (eg, affect, ability to sleep, and ability to function effectively at work or school or while driving). In patients who have concomitant conditions that may be aggravated by rhinitis (eg, asthma), an assessment of concomitant conditions should also be made because improved control of rhinitis may be associated with improvement of these conditions. Patients who do not have a good response to treatment should be referred to an allergist/immunologist.

**Box 6: Further follow-up, meets consultation criteria?**

If the initial treatment of rhinitis is successful, there is still a need for patient follow-up to assure that there is continued control of symptoms, maintenance of improved QOL, lack of impairment of performance at work or school and in other activities, and absence of medication side effects. Consultation with an allergist/immunologist is appropriate when these conditions are not met. Characteristics that should lead to consideration of consultation with an allergist/immunologist include the following:

1. The patient has had prolonged manifestations of rhinitis.
2. The patient has complications of rhinitis, such as otitis media, sinusitis, and/or nasal polyps.
3. The patient has a comorbid condition, such as asthma and chronic sinusitis.
4. The patient has required a systemic corticosteroid for the treatment of rhinitis.

5. The patient’s symptoms or medication side effects interfere with ability to function, such as causing sleep disturbance or impairing school/work performance.
6. The patient’s symptoms significantly decrease QOL, such as a decrease in comfort and well being, sleep disturbance, anosmia, or ageusia.
7. Treatment with medications for rhinitis is ineffective or produces adverse events.
8. The patient has been diagnosed with rhinitis medicamentosa.
9. Allergic/environmental triggers causing the patient’s rhinitis symptoms need further identification and clarification.
10. There is a need for more complete education.
11. The patient has required multiple and/or costly medications over a prolonged period.
12. Specific allergy immunotherapy is a treatment consideration.

Consultation with an allergist/immunologist may be indicated in other situations when there is agreement between the patient and the referring physician that such an approach is in the patient’s best interests.

**Box 7: Consultation with an allergist/immunologist**

An assessment of rhinitis by a rhinitis specialist requires a detailed history and appropriate physical examination. The history should include all of the components outlined in Box 1 but in more depth. The physical examination should assess the upper airway (nose, oropharynx) and lungs. In addition, rhinoscopy or examination by rigid or flexible rhinolaryngoscopy (endoscope) allows for better visualization of the middle meatus, the posterior septum, the sinus ostia, the nasopharynx, and presence of nasal polyps. Immediate hypersensitivity skin tests or *in vitro* tests for specific IgE to confirm an underlying allergic basis for the patient’s symptoms may be necessary. Nasal cytology may be of value. Rarely, other tests may be indicated such as β-transferrin in nasal secretions (for suspected CNS fluid leakage) or tests of nasal ciliary function. Specific tests may also be necessary for coexisting conditions such as asthma (eg, pulmonary function), nasal polyps (eg, rhinoscopy), or sinusitis (eg, CT scan).

A thorough evaluation is the key component to the development of a long-term management plan. Management may include education regarding environmental avoidance and medication compliance, institution of environmental control measures, changes in medication, and allergen immunotherapy.

**Box 8: Does patient have an allergic basis for rhinitis?**

A diagnosis of allergic rhinitis depends on the history of nasal symptoms after exposure to suspected allergens, which are confirmed with positive skin or *in vitro* tests for specific IgE. Determination of specific IgE, preferably by skin testing, is indicated to provide evidence of an allergic basis for the patient’s symptoms, to confirm suspected causes of the patient’s symptoms, or to assess the sensitivity to a specific allergen for avoidance measures and/or allergen immunotherapy. The precise sensitivity of specific IgE immunoassays compared with skin prick/puncture tests is approximately 70% to 75%. Skin tests are the preferred tests for the diagnosis of IgE-mediated sensitivity. The number of skin tests and the allergens selected for skin testing should be determined on the basis of the patient’s age,
Box 9: Management of allergic rhinitis

Effective management of allergic rhinitis may require combinations of medications, aggressive avoidance measures, management of coexisting conditions, and/or allergen immunotherapy. Avoidance of triggers of rhinitis, such as allergens, irritants, medications, and occupational factors, is fundamental to the successful management of allergic rhinitis. After triggers are identified, the patient or representative should be educated about avoidance. If it is possible to anticipate the onset of symptoms associated with seasonal exposure to pollen or sporadic exposure to other triggers, early administration of medications (eg, before exposure or the development of symptoms) may lessen the impact of such exposures (see Box 3 annotation for a discussion of appropriate medications).

A short course of oral corticosteroids may be appropriate for the treatment of intractable nasal symptoms (see Box 3 annotation) or severe nasal polyposis. The chronic use of oral or parenteral corticosteroids is inappropriate in allergic rhinitis.

Allergen immunotherapy is effective for treatment of allergic rhinitis and allergic rhinoconjunctivitis. Effective immunotherapy has been associated with significant improvement in symptom and medication scores and QOL measures as well as objective parameters such as nasal provocation challenge, immunologic changes in cell markers, and cytokine profiles. Allergen immunotherapy should be considered for patients with allergic rhinitis who have demonstrable evidence of specific IgE antibodies to clinically relevant allergens. The decision to begin allergen immunotherapy depends on the degree to which symptoms can be reduced by avoidance and medication, the amount and type of medication required to control symptoms, and the adverse effects of medications. The severity and duration of symptoms as well as the impact of the patient’s symptoms on QOL should also be considered in assessing the need for specific allergen immunotherapy.

Box 10: Management of nonallergic rhinitis

Nonallergic rhinitis is characterized by sporadic or persistent perennial symptoms of rhinitis that do not result from IgE-mediated immunopathologic events. Examples of nonallergic rhinitis are infectious rhinitis, hormonal rhinitis, vasomotor rhinitis (including gustatory rhinitis), NARES, certain types of occupational rhinitis, and drug-induced rhinitis.

The signs and symptoms suggestive of rhinitis can be produced by anatomic conditions including nasal septal deviation, tumors, adenoidal hypertrophy, and hypertrophy of the nasal turbinates. Examination of the nose should include evaluation of the nasal passageways, secretions, turbinates, septum, and determination of whether nasal polyps are present. In selected cases, fiber optic nasal endoscopy and/or rhinomanometry may be useful. Nasal cytology may aid in differentiating allergic rhinitis and NARES from other forms of rhinitis.

The primary treatments for nonallergic rhinitis syndromes may vary and include (1) avoidance of aggravating irritants that may precipitate symptoms, (2) intranasal corticosteroids, (3) decongestants and exercise to relieve congestion, (4) anticholinergics to relieve rhinorrhea, (5) intranasal corticosteroids and intranasal antihistamines to relieve both congestion and rhinorrhea associated with vasomotor rhinitis, (6) institution of intranasal corticosteroids and discontinuation of nasal decongestant sprays in rhinitis medicamentosa, and (7) antibiotics and supportive measures to relieve osteomeatal complex obstruction in bacterial rhinosinusitis.

Box 11: Cooperative follow-up

Cooperative follow-up for allergic rhinitis patients includes the patient, family, and health care providers (ie, the primary care physician, allergist/immunologist, and possibly otorharyngologist). Goals include the reduction of symptoms and improvement in the patient’s QOL and ability to function. These goals require cooperative management of exacerbations and complications by optimal use of environmental avoidance measures and medications, and in appropriate patients, use of immunotherapy.

Tapering of medications should always be considered to lessen the risk of adverse reactions. Side effects of medications must be carefully looked for during follow-up of patients. Maximizing compliance with medications and environmental controls can be challenging for the patient and physician, especially if the patient is very young or elderly.

Periodic assessment of the patient’s QOL is essential. This includes evaluation of time lost from work or other activities, sleep quality, smell and taste, fatigue level, and general well-being.

Patient education is a basic part of the follow-up plan for patients with allergic rhinitis. At each visit, it is important to review preventative measures (eg, environmental controls), medication use, and immunotherapy status with the patient. In addition, the presence of comorbid conditions such as sinusitis, asthma, and otitis media should be ascertained.

Effective follow-up requires awareness of the patient’s goals, needs, and concerns. Allergen immunotherapy may be appropriate for patients with allergic rhinitis, especially if the patient is not responding to other therapeutic approaches and symptoms are interfering with the patient’s ability to function. Follow-up also requires effective interaction between all health care providers as well as interaction with the patient and often the patient’s family.

Although there is no surgical treatment for rhinitis, surgery may be indicated in the management of comorbid conditions, such as nasal obstruction from severe nasal septal deviation or inferior turbinate hypertrophy, adenoidal hypertrophy, or refractory sinusitis and complications thereof. Other reasons for referral to an
otolaryngologist include the evaluation of ostiomeatal obstruction, nasal polyp surgery, biopsy of nasal tumors, or other surgical requirements.

**SUMMARY STATEMENTS WITH DISCUSSION**

**Definition**

1. Rhinitis is characterized by 1 or more of the following symptoms: nasal congestion, rhinorrhea (anterior and posterior), sneezing, and itching. D

Although the term rhinitis connotes inflammation, and the majority of rhinitides are associated with inflammation, some forms of rhinitis such as vasomotor rhinitis or atrophic rhinitis may not be associated with inflammation of the nasal mucosa. Rhinitis frequently is accompanied by symptoms involving the eyes, ears, and throat.

**Differential diagnosis of rhinitis**

2. Rhinitis should be classified by etiology as allergic or nonallergic and differentiated from conditions that mimic symptoms of rhinitis. C

Rhinitis is classified as allergic or nonallergic, but not all types of rhinitis can be easily separated into 1 of these categories. For example, occupational rhinitis has been classified separately from allergic and nonallergic because it may have components of both allergic and nonallergic rhinitis. Conditions that mimic symptoms of rhinitis include nasal polyps, cerebrospinal fluid rhinorrhea, ciliary dyskinesia syndrome, and structural/mechanical factors, such as deviated septum and pharyngonasal reflux (see Tables I and II in the Executive Summary).

**Classification of allergic rhinitis**

3. Symptoms of allergic rhinitis may occur only during specific seasons, may be perennial without seasonal exacerbation, may be perennial with seasonal exacerbations, or may occur episodically after specific allergen exposures. C

4. **Episodic** allergic rhinitis is a new rhinitis category that denotes allergic nasal symptoms elicited by sporadic exposures to inhalant allergens. D

5. The severity of allergic rhinitis ranges from mild and intermittent to seriously debilitating. D

6. Although there is no generally accepted method of grading the severity of rhinitis, the clinician may want to consider a graphic rating scale. D

Recently, an international working group released ARIA recommendations and proposed a revised classification for allergic rhinitis that categorized all patients as either intermittent (<4 days per week or <4 weeks) or persistent (>4 days per week and >4 weeks) and classified severity as mild or moderate-severe. 3,10 This system proposes 4 classes, which include (1) mild intermittent, (2) mild persistent, (3) moderate/severe intermittent, and (4) moderate/severe persistent. 10 This classification system discarded the terms seasonal and perennial, on the basis of several rationales including the observation that an allergen (eg, grass pollen) that occurs seasonally in one region may be detected throughout the year in another geographical area. Demoly et al 690 reported that 44% of patients traditionally classified as having seasonal rhinitis had persistent rhinitis according to the ARIA classification and that 44% with perennial allergic rhinitis were reclassified as intermittent. Thus, the traditional seasonal/perennial and ARIA schemes define different patient populations.

Treatment guidelines based on the ARIA guidelines have not been adequately studied. 12 However, the ARIA definition of mild rhinitis may be a useful comparative reference point for other severity grading schemes; this states that none of the following items is present: sleep disturbance; impairment of daily activities, leisure and/or sport; impairment of school or work; and symptoms present but not troublesome. 13 This updated parameter supports the concept that more severe rhinitis is defined as more symptoms or interference with QOL, because data show that it may not be possible to separate patients into moderate and severe categories. 12 A nonvalidated modified 7-point visual analog (graphic rating) scale for grading severity of nasal and nonnasal symptoms of allergic rhinitis and the effects of this disorder on the QOL has been developed and published by the Joint Task Force on Practice Parameters and is included, with minor modification, in Figs 1-4. 13

In this document, the Joint Task Force retains and uses the terms seasonal and perennial in classifying patients with allergic rhinitis. These traditional descriptive terms are clinically useful and allow for accurate categorization of the vast majority of patients as having seasonal, perennial, or perennial allergic rhinitis with seasonal exacerbations. In addition to seasonal and perennial, episodic is used in this practice parameter to denote allergic nasal symptoms elicited by sporadic exposures to inhalant allergens that are not usually encountered in the patient’s indoor or outdoor environment. Although the terms seasonal, perennial, and episodic are clinically useful, therapeutic decisions should also be guided by frequency, duration, and severity of symptoms, and by current and previous responsiveness to medications. 13

**Allergic rhinitis**

7. Mixed rhinitis (combined allergic and nonallergic rhinitis) is noted in approximately 44% to 87% of patients with allergic rhinitis and is more common than either pure allergic rhinitis or nonallergic rhinitis. C

8. Allergic rhinitis affects 30 to 60 million people in the United States annually, including 10% to 30% of adults and as many as 40% of children. C

9. Risk factors for allergic rhinitis include (1) family history of atopy, (2) serum IgE >100 IU/mL before age 6 years, (3) higher socioeconomic class, and (4) presence of a positive allergen SPT. C

10. The influence of early childhood exposure to infections, animals, and secondary tobacco smoke on the development of atopy and allergic rhinitis is still unknown. C

11. Aeroallergen sensitization may occur within the first 2 years of life. C

12. The cost of treating allergic rhinitis and indirect costs related to loss of workplace productivity resulting from the disease are substantial. Rhinitis is also a significant cause of lost work and school days. C

Rhinitis is reported to be a very frequent disease, although data regarding the true prevalence of rhinitis are difficult to interpret. Most population surveys rely on physician-diagnosed rhinitis for their data, and this may give rise to a much lower reporting of
rhinitis. Some population studies have been performed with questionnaires administered to the subjects, followed in many cases by telephone interviews, to try to make a specific diagnosis of rhinitis. It has been estimated that 25% to 33% of cases of rhinitis are a result of nonallergic rhinitis, and that 44% to 87% of allergic rhinitis has an element of nonallergic rhinitis, referred to as mixed rhinitis. Several studies may reflect a more accurate prevalence of rhinitis but probably still underreport this disease.

The prevalence of allergic rhinitis in various epidemiologic studies ranges from 3% to 19%. According to the Centers for Disease Control and Prevention, 23.7 million cases of allergic rhinitis were reported in 1996, including 15.9 million cases among persons age 45 years or younger.

Seasonal allergic rhinitis is apparently becoming more common. One study showed that the prevalence of hay fever increased from 4% to 8% in the 10 years from 1971 to 1981. In another study, atopic skin test reactivity increased from 39% to 50% during an 8-year period of evaluation. However, a recent study of Swiss children 5 to 7 years old conducted during the last decade suggests that the increasing prevalence of allergic rhinitis may have plateaued in some countries.

Studies suggest that seasonal allergic rhinitis (hay fever) is found in approximately 10% to 20% of the population. However, 1 study of physician-diagnosed allergic rhinitis showed a prevalence of 42% in 6-year-old children. Overall, allergic rhinitis affects 30 to 60 million individuals in the United States annually.

In childhood, boys with allergic rhinitis outnumber girls, but the sex ratio becomes approximately equal in adults and may even favor women. Allergic rhinitis develops before age 20 years in 80% of cases. Studies have shown that the frequency of allergic rhinitis increases with age until adulthood and that positive immediate hypersensitivity skin tests are significant risk factors for the development of new symptoms of seasonal allergic rhinitis. There is a greater chance of a child developing allergic rhinitis if both parents have a history of atopy than if only 1 parent is atopic. Children in families with a bilateral family history of allergy generally develop symptoms before puberty; those with a unilateral family history tend to develop their symptoms later in life or not at all.

There tends to be an increased prevalence of allergic rhinitis in nonwhites, in some polluted urban areas, and in individuals with a family history of allergy. Allergic rhinitis is more likely in first-born children. Studies in children in the first years of life have shown that the risk of rhinitis was higher in those youngsters with early introduction of foods or formula, higher serum IgE levels (100 IU/mL before age 6 years), and parental allergic disorders.

Over the past few years, several studies supporting the hygiene hypothesis have suggested that early exposure to viral and bacterial infections, such as day care attendance or more siblings, may reduce the incidence of atopic disease by redirecting the immune system away from the allergic Th2 pattern to the Th1 pattern. One early explanation proposed that the increased incidence of atopy as explained by the hygiene hypothesis is a result of the reduced production of IL-12 and IFNs by cells of the innate immune system that are normally stimulated by bacterial products via their Toll-like receptors. More recently, the role of reduced activity of regulatory T cells has been emphasized. It is now felt that early infections reinforce the physiological mechanisms of natural dominant tolerance by expanding natural regulatory T cells. It also appears that the proinflammatory ligands of Toll-like receptors on the natural regulatory T cells play a major role in their activation and expansion. However, some recent studies refute the hygiene hypothesis, demonstrating that increased infections in early life increase allergic disease in childhood and do not contribute to any reduction of atopic disease in adults. Animal exposure in early infancy is likewise controversial because some studies have demonstrated that cat exposure in early infancy may reduce atopy and asthma, whereas others have shown either no effect or increased allergic disease. Environmental risk factors for rhinitis in early infancy include environmental smoke exposure as well as allergens. Exposure to >20 cigarettes per day has been shown to be associated with an increased risk of developing allergic rhinitis at age 1 year. The effect of tobacco smoke on allergic sensitization at age 1 year and the frequency of upper respiratory infections and ear infections in the young child remain controversial. It has been suggested that the month of birth increases the risk of pollen and dust mite sensitization especially in childhood, but not all studies agree.

A critical period appears to exist early in infancy in which the genetically programmed individual is at greatest risk of sensitization on exposure to food and aeroallergens. In infancy, food allergies cause primarily gastrointestinal symptoms and atopic dermatitis and rarely induce nasal symptoms. Infants born to atopic families are sensitized to pollen aeroallergens more frequently than to indoor aeroallergens in the first year of life. Although perennial allergic rhinitis (eg, dust mite and animal dander) may be present at a very early age, seasonal allergic rhinitis typically does not develop until the child is 2 to 7 years of age, because 2 seasons of exposure are generally required for sensitization. The prevalence of seasonal allergic rhinitis is higher in children and adolescents, whereas perennial allergic rhinitis is higher in adults.

The financial impact on society is tremendous. The severity of allergic rhinitis ranges from mild to seriously debilitating. The direct cost of treating allergic rhinitis and the indirect cost related to loss of workplace productivity resulting from the disease are substantial. The estimated cost of allergic rhinitis based on direct and indirect costs is $2.7 billion for the year 1995, exclusive of costs for associated medical problems such as sinusitis and asthma. The cost to society has continued to increase. Based on pharmacy and medical care expenditure data, the estimated direct medical cost of allergic rhinitis was $7.3 billion for the year 2002, which was primarily incurred by costs of prescriptions and outpatient clinic visits. The total direct cost ($7.3 billion) and indirect cost ($4.28 billion) estimates for allergic rhinitis have been estimated to be $11.58 billion for 2002. This figure included the higher indirect costs associated with increased loss of productivity, which in turn was related to extensive OTC first-generation antihistamine use. Such treatment can cause drowsiness and impair cognitive and motor function.

Rhinitis is also a significant cause of lost school attendance, resulting in more than 2 million absent school days in the United States annually. In children, there is evidence that symptoms of allergic rhinitis can impair cognitive functioning, which can be further impaired by the use of first-generation antihistamines.
between resident and infiltrating inflammatory cells and a number of vasoactive and proinflammatory mediators, including cytokines. Sensory nerve activation, plasma leakage, and congestion of venous sinusoids also contribute.

The nasal mucosa is designed to humidify and clean inspired air. The actions of epithelium, vessels, glands, and nerves are carefully orchestrated to perform these functions.710 Dysfunction of any of these structures may contribute to the symptoms of allergic and nonallergic rhinitis.

14. Allergic rhinitis may be characterized by early-phase and late-phase responses. Each type of response is characterized by sneezing, congestion, and rhinorrhea, but congestion predominates in the late phase.

Atopic subjects inherit the tendency to produce specific IgE antibodies and T\(\text{H}2\)-directed immune responses. Intermittent or continuous exposure to low concentrations of indoor or outdoor aeroallergens over time may result in sensitization, a process initiated by processing of allergens by dendritic cells expressing CD1a and CD11c,711 in the nasal epithelium mucosa and subsequent presentation of allergenic peptides by MHC II molecules to T-cell receptors on resting CD4\(^+\) cells in regional lymph nodes. With appropriate costimulatory signals, allergen-stimulated resting T cells proliferate into T\(\text{H}2\)-biased cells that produce IL-3, IL-4, IL-13, IL-5, GM-CSF, and other cytokines. T\(\text{H}2\) cytokines promote B-cell isotype switching and allergen specific IgE production by plasma cells, mast cell proliferation and infiltration of airway mucosa, and eosinophilic infiltration into the nasal mucosa and nasal epithelium.

Early or immediate allergic response

With continued allergen exposure, increasing numbers of IgE-bound mast cells recognize the mucosally deposited allergen and degranulate.710 Mast cell products include preformed mediators such as histamine, tryptase, chymase, kininogenase, heparin, and other enzymes.712 Newly formed mediators including prostaglandin D\(_2\),713 and the cysLTs (LTC\(_4\), LTD\(_4\), and LTE\(_4\)) are produced by mast cells, eosinophils, basophils, and macrophages and bind to specific receptors in the nose.714 These mediators produce edema, watery rhinorrhea, and mucosal hypertrophy; stimulate glands to exocytose their mucoglycoconjugates and antimicrobial substances; and dilate arteriole-venule anastomoses to cause sinusoidal filling and occlusion of nasal air passages. The cysLTs also play an active role in recruitment of inflammatory cells.714 Sensory nerves are stimulated that convey the sensations of nasal itch and congestion and initiate reflexic symptoms such as sneezing paroxysms. Within minutes of allergen exposure, there is release of mast cell mediators and induction of the response. This is known as the early or immediate allergic response. Although most subjects experience sneezing and copious rhinorrhea after allergen exposure, some subjects have sensations of nasal congestion as their predominant symptom. Neuropeptide expression (eg, substance P) has been demonstrated in mucosal nerve fibers of patients with seasonal allergic rhinitis, although the exact roles of sensory neural mediators in the pathogenesis of symptoms of allergic rhinitis are uncertain.715

Late-phase response

The mast cells mediators, including cytokines, are thought to play active roles in generating the late-phase response, which is initiated 4 to 8 hours after allergen exposure. LTC\(_4\) increases both during the early-phase and late-phase nasal responses to allergen.716 In a study evaluating kinetics of mediators and cytokines in nasal secretions after allergen challenge, histamine was increased in nasal secretions during the early-phase and late-phase nasal responses, and IL-1\(\beta\) and IL-4 were significantly elevated during the late-phase response.717 In another study that examined nasal mucosal late responses after a single nasal grass allergen exposure, T\(\text{H}2\) cytokines including IL-5 and IL-13 were expressed in association with increased numbers of eosinophils.718

Local endothelial expression of intercellular adhesion molecule 1, E-selectin, and vascular adhesion molecule 1 have been correlated with increased nasal mucosal eosinophils at 24 hours after nasal allergen provocation, indicating that adhesion molecules are upregulated and facilitate transmigration of activated eosinophils into the nasal mucosa.719 Chemoattractants, such as IL-5 and eotaxin for eosinophils, as well as chemokines IL-8 and monocyte chemoattractant protein 1 have been detected in nasal secretions during the late-phase response and are thought to enhance infiltration of the superficial lamina propria of the mucosa with neutrophils, eosinophils, and, at later time points, T lymphocytes and macrophages.720 Eosinophil products such as major basic protein, ECP, hypohalides, LTs, and others are thought to damage the epithelium and other cells, resulting in an inflammatory response that promotes the tissue damage of chronic allergic reactions.32 Pretreatment with glucocorticoids is effective at reducing eosinophils and the release of cytokines (eg, IL-4, IL-5, and IL-13) during the late-phase response.

**Priming response**

When allergen challenges are given repeatedly, the amount of allergen required to induce an immediate response decreases.719-721 This priming effect is thought to be a result of the influx of inflammatory cells during ongoing, prolonged allergen exposure and repeated late-phase responses. The priming effect demonstrates the importance of knowing the full spectrum of aeroallergens to which a patient responds and seasonal variations in allergic symptoms, and provides the rationale to consider initiating effective anti-inflammatory therapies before the pollen season or before other chronic or repetitive allergen exposures.

**Seasonal and perennial allergic rhinitis**

15. Seasonal allergic rhinitis is caused by an IgE-mediated reaction to seasonal aeroallergens. The length of seasonal exposure to these allergens is dependent on geographic location and climatic conditions.

16. Perennial allergic rhinitis is caused by an IgE-mediated reaction to perennial environmental aeroallergens. These may include dust mites, molds, animal allergens, or certain occupational allergens, as well as pollen in areas where pollen is prevalent perennially.

Symptoms of allergic rhinitis may include paroxysms of sneezing, nasal pruritus and congestion, clear rhinorrhea, and palatal itching. The conjunctiva,716 eustachian tubes, middle ear, and paranasal sinuses may also be involved. Allergic rhinitis is associated with ear fullness and popping, itchy throat, and pressure over the cheeks and forehead. Malaise, weakness, and fatigue may also be present. Allergic rhinitis often begins during childhood and may coincide with or precede development of allergic asthma.722 A positive family history of
atopy is associated with development of allergic rhinitis in childhood. When not all the typical rhinitis symptoms are expressed, the diagnosis is more difficult to make. Nasal airflow obstruction, a major symptom of seasonal or perennial allergic rhinitis, is associated with nasal eosinophilic inflammation. Distinct temporal patterns of symptom production may aid diagnosis. Symptoms of rhinitis that occur whenever the patient is exposed to a furry pet suggest IgE mediated sensitivity to that pet. Patients who are acutely sensitive to animal proteins may develop symptoms of rhinitis and asthma when entering a house or laboratory even though the animal is no longer present. Children who own pets can passively transfer cat allergen on their clothing into schools and may contribute to high levels of ambient cat allergen in classrooms. Seasonal and perennial forms of allergic rhinitis often coexist in the same individual. Symptoms may be chronic and persistent, and patients may present with secondary complaints of mouth-breathing, snoring, or symptoms of sinusitis. Severe allergic rhinitis has been associated with diminished QOL, disordered sleep (in as many as 76% of patients), and impairment in work performance. Seasonal allergic rhinitis symptoms typically appear during a defined season in which Aeroallergens are abundant in the outdoor air. The length of seasonal exposure to these allergens is dependent on geographic location and climatic conditions. Familiarity with the pollinating season of the major trees, grasses, and weeds of the locale makes the syndrome easier to diagnose. Certain outdoor mold spores also display seasonal variation, with highest levels in the summer and fall months. Tree (eg, birch, oak, maple, and mountain cedar), grass (eg, timothy and Bermuda), and weed (eg, ragweed) pollens and fungi (eg, Alternaria, Aspergillus, and Cladosporium) are common seasonal allergens. Hyperresponsiveness to irritant triggers such as chlorine is enhanced among patients with seasonal allergic rhinitis. In studies of seasonal allergic rhinitis, a correlation between the daily pollen count and overall daily symptom score and medication score has been found. Nasal sensitivity to seasonal pollen allergens increases as the pollen season progresses because of the priming phenomenon. As a consequence of priming, at the end of the pollen season, nasal symptoms may decline more slowly than the pollen counts. Individual host sensitivity to an aerallergen may influence the intensity of symptoms. The levels of pollen counts that cause symptoms may vary with an individual’s degree of sensitivity and with different pollens. Indoor allergens responsible for perennial allergic rhinitis are present in the environment throughout the year.

Both research and clinical experience support the concept that allergic rhinitis and allergic conjunctivitis may exist in rare patients with negative skin tests and/or in vitro tests for specific IgE. A patient with a complicating history of symptoms after exposure to an allergen can have a positive nasal challenge with that allergen despite negative skin tests and/or in vitro tests for specific IgE antibody. Studies have shown that patients with allergic rhinitis symptoms after exposure to house dust have been found to have local inflammation, nasal IgE production, and a positive response to a nasal allergen provocation test with Dermatophagoides pteronyssinus, despite having negative skin tests and specific IgE to D pteronyssinus. Further research is needed to determine what allergens are capable of producing this type of reaction, the prevalence of this condition, the mechanism responsible for local allergic antibodies, and the optimal treatment for these patients.

### Allergic conjunctivitis

17. Allergic rhinitis is often accompanied by symptoms of allergic conjunctivitis. C

18. Many treatments used for allergic rhinitis can benefit associated symptoms of allergic conjunctivitis, and a variety of topical ophthalmic agents is useful for specific treatment of associated ocular symptoms. Intranasal corticosteroids, oral antihistamines, and intranasal antihistamines have similar effectiveness in relieving ocular symptoms associated with rhinitis. Allergic rhinitis is often accompanied by allergic conjunctivitis (a disease complex sometimes referred to as allergic rhinoconjunctivitis) that produces conjunctival injection and chemosis associated with symptoms of itchy eyes and tearing. Estimates of the prevalence and severity of conjunctival symptoms associated with allergic rhinitis vary depending on the aeroallergen, geographic region, and other factors. In seasonal allergic rhinitis study, allergic conjunctivitis symptoms were reported in more than 75% of patients. Sensitivity to pollens is more frequently associated with rhinoconjunctivitis, whereas sensitivity to house dust mites (D pteronyssinus, Dermatophagoides farinae) is reported to cause less ocular symptoms.

The Joint Task Force is developing a complete Parameter on Diagnosis and Treatment of Allergic Conjunctivitis that will provide more comprehensive discussion than the more limited statements on allergic conjunctivitis in this Rhinitis Parameter. A complete review of the differential diagnosis of conjunctivitis is beyond the scope of this document. Ocular allergy may include seasonal and perennial allergic conjunctivitis discussed here, but also 2 vision-threatening disorders, atopic keratoconjunctivitis (associated with eczematous lesions of the lids and skin) most commonly seen in older adult patients, and vernal keratoconjunctivitis (chronic inflammation of palpebral conjunctiva), seen most commonly seen in the pediatric and adolescent age groups with a male predominance. In seasonal and perennial allergic conjunctivitis associated with allergic rhinitis, both eyes are typically affected, and itching is usually a prominent symptom.

Oral antihistamines, intranasal antihistamines, oral anti-LT agents, intranasal corticosteroids, and allergen immunotherapy are treatments for allergic rhinitis that have been reported to relieve associated ocular symptoms in controlled trials. In systematic reviews of randomized controlled studies, intranasal corticosteroids compared with oral antihistamines and intranasal corticosteroids compared with intranasal antihistamines were not significantly different in relieving eye symptoms. In placebo-controlled studies of adults, fluticasone furoate nasal spray has been demonstrated to reduce significantly ocular symptoms associated with seasonal allergic rhinitis.

Use of cold compresses and irrigation with saline solution or artificial tears has been advocated to relieve mild symptoms of allergic conjunctivitis. A variety of topical ophthalmic agents are indicated for specific treatment of itching or symptoms of allergic conjunctivitis. These include medications listed in Table XIII and can be summarized by the following categories.
Ocular corticosteroids should be reserved for more severe symptoms. Multiple-action agents possess both antihistamine and mast cell stabilizer activities, generally have onset of action within 30 minutes, and are suitable for acute and longer-term treatment of allergic conjunctivitis symptoms.

Ocular corticosteroids should be reserved for more severe symptoms of allergic conjunctivitis in consideration that ocular side effects from their use can be vision-threatening, and include cataract formation, elevated IOP, and secondary infections. The modified steroid loteprednol is indicated for the temporary relief of ocular redness, although they do not appear to induce this.60

Oral antihistamines are generally less effective in relieving ocular allergy symptoms than topical ophthalmic agents and have slower onset of action.750-752 Although comparative efficacy trials of topical ophthalmic agents in real-life settings are generally lacking, studies performed in environmental challenge chambers or using acute ocular allergen challenges have generally demonstrated that dual action ophthalmic agents are more effective in preventing or treating ocular itching than other ocular agents.75,76 Oral agents have also been associated with excessive drying of the tear film.

Nonallergic rhinitis

20. Nonallergic rhinitis is characterized by periodic or perennial symptoms of rhinitis that are not a result of IgE-dependent events. Examples of nonallergic rhinitis are infectious rhinitis, vasomotor rhinitis, and NARES.

Vasomotor rhinitis

21. Vasomotor rhinitis (idiopathic rhinitis) accounts for a heterogeneous group of patients with chronic nasal symptoms that are not immunologic or infectious in origin and is usually not associated with nasal eosinophilia.

Vasomotor rhinitis is unrelated to allergy, infection, structural lesions, systemic disease, or drug abuse. Although the term vasomotor implies increased neural efferent traffic to the blood vessels supplying the nasal mucosa, this has never been proven. Subjects with predominant rhinorrhea (sometimes referred to as cholinergic rhinitis) appear to have enhanced cholinergic glandular secretory activity because atropine effectively reduces their secretion.66 Subjects with predominantly nasal congestion and blockage may have nociceptive neurons that have heightened sensitivity to innocuous stimuli.

The term vasomotor rhinitis has been used loosely to describe the condition of patients with perennial rhinitis whose symptoms are intensified by changes in temperature or relative humidity, alcohol, and odors such as bleach, perfume, or solvents. Other triggers include tobacco smoke, dusts, automotive emission fumes, and nonspecific irritant stimuli such as chlorine.67 Cold dry air and exercise may also trigger symptoms.68,69,757 The symptoms are variable, consisting mainly of nasal obstruction and increased secretion. Sneezing and pruritus are less common. Although the term vasomotor implies increased neural efferent traffic to the blood vessels supplying the nasal mucosa, this has never been proven. A lack of change in nasal compliance after administration of nasal oxymetazoline compared with normal subjects lends support to the hypothesis that vasomotor rhinitis may be attributable to autonomic dysfunction.758

Rhinitis from food ingestion

22. Rhinitis may occur after ingestion of foods or alcoholic products. This may be a result of vagally mediated mechanisms, nasal vasodilation, food allergy, and/or other undefined mechanisms. Food allergy is a rare cause of rhinitis without associated gastrointestinal, dermatologic, or systemic manifestations.

Foods can provoke rhinitis symptoms by a variety of different mechanisms.759,760 Ingested food allergens rarely produce

<table>
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<tr>
<th>TABLE XIII. Topical ophthalmic preparations for ocular allergy symptoms</th>
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<tr>
<td><strong>Classification</strong></td>
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<tr>
<td>-------------------------</td>
</tr>
<tr>
<td>Antihistamine</td>
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<tr>
<td>Combination antihistamine/ vasoconstrictor</td>
</tr>
<tr>
<td>Mast cell stabilizer</td>
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<td>Mast cell stabilizer</td>
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<td>Mast cell stabilizer</td>
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<td>NSAID</td>
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<tr>
<td>Dual action agent</td>
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<tr>
<td>(antihistamine and mast cell stabilizer)</td>
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<tr>
<td>(antihistamine and mast cell stabilizer)</td>
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<tr>
<td>Corticosteroid</td>
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- Vasoconstrictors are available in OTC preparations and are indicated for relief of ocular redness, although they do not reduce the allergic response. Prolonged use of ocular decongestants may lead to rebound hyperemia or conjunctivitis medicamentosa, although use limited to 10 days does not appear to induce this.
- Antihistamines (H1-receptor antagonists) are available in OTC and prescription ophthalmic preparations and are sometimes combined with a topical vasoconstrictor for acute relief of ocular allergy symptoms. The combination of an antihistamine and a vasoconstrictor works better than either agent alone.
- Mast cell stabilizers have a slow onset of action and may require several days of treatment before optimal symptom relief is achieved, making them more suitable for prophylactic or longer-term treatment of chronic ocular allergies than for acute symptom relief. They are also approved for chronic ocular allergy conditions involving corneal defects including vernal keratoconjunctivitis and atopic keratoconjunctivitis.
- Topical NSAIDs reduce prostaglandin production involved in mediating ocular allergy. Ketorolac is indicated for temporary relief of ocular itching caused by seasonal allergic conjunctivitis.

Oral antihistamines are generally less effective in relieving ocular allergy symptoms than topical ophthalmic agents and have slower onset of action. Although comparative efficacy trials of topical ophthalmic agents in real-life settings are generally lacking, studies performed in environmental challenge chambers or using acute ocular allergen challenges have generally demonstrated that dual action ophthalmic agents are more effective in preventing or treating ocular itching than other ocular agents. Oral agents have also been associated with excessive drying of the tear film.

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Foods can provoke rhinitis symptoms by a variety of different mechanisms. Ingested food allergens rarely produce...
isolated IgE-mediated rhinitis without involvement of other organ systems. Urticarial rash, facial or lip swelling, or bronchospasm strongly suggest an IgE-mediated reaction.\textsuperscript{66} In a large group of children undergoing double-blind, placebo-controlled food challenges, nasal symptoms developed in 70% of the positive challenges.\textsuperscript{762,763} In that study, the most common food allergens confirmed in respiratory tract symptoms included egg, cow’s milk, peanut, soy, fish, shellfish, and tree nuts. In contrast, rhinitis may occasionally be reported in unusual food allergens—for example, 2 of 43 patients reporting rhinitis with kiwi allergy.\textsuperscript{764} In another descriptive study that did not include double-blind, placebo-controlled food challenges, rhinitis or conjunctivitis accounted for 5.7% of the total symptoms reported.\textsuperscript{765} In adults, food skin tests may be appropriate in occasional cases if a careful history suggests food-related rhinitis symptoms, particularly if rhinitis symptoms are associated with other systemic symptoms. Although a variety of opinions have been expressed in the literature,\textsuperscript{25,765-769} there are few or no credible data available to justify routine performance of food skin tests in the evaluation of rhinitis in adults. In the evaluation of rhinitis in children, in whom the history may be more difficult to interpret and food allergy is more common, there is greater justification to consider performance of limited food skin testing. Beer, wine, and other alcoholic drinks may produce symptoms by nasal vasodilation. Alcohol-induced hypersensitivity symptoms are also more prevalent in persons with allergic rhinitis and asthma.\textsuperscript{70} The syndrome of watery rhinorrhea occurring immediately after ingestion of foods, particularly hot and spicy foods, has been termed gustatory rhinitis and is vagally mediated.\textsuperscript{56}

Infectious rhinitis

23. Infectious rhinitis and rhinosinusitis may be acute or chronic. Acute infectious rhinitis is usually a result of 1 of a large number of viruses, but secondary bacterial infection with sinus involvement may be a complication. Symptoms of acute infectious rhinosinusitis include nasal congestion, mucopurulent nasal discharge, pain and pressure, headache, olfactory disturbance, postnasal drainage, and cough.\textsuperscript{C}

Acute rhinitis is usually associated with a viral upper respiratory infection and frequently presents with rhinorrhea, nasal obstruction, and fever. Initially, viral rhinitis is characterized by clear, watery rhinorrhea that is accompanied by sneezing and nasal obstruction. Edema of the nasal mucosa produces occlusion of the sinus ostia with resulting facial pain or of the eustachian tube with resulting ear fullness. The nasal drainage may become cellular and cloudy due to the presence of organisms, white blood cells, and desquamated epithelium. Responsible viruses include rhinoviruses, respiratory syncytial virus, parainfluenza, influenza, and adenoviruses. Unless there is bacterial superinfection (<2% of the time),\textsuperscript{75,76} the condition is self-limiting and usually resolves within 7 to 10 days. Acute bacterial rhinosinusitis may occur de novo or may follow viral rhinitis. Nasal obstruction, cloudy drainage, vestibular crusting, and facial pain occur. Not all patients report fever. Bacteria frequently recovered from nasal or sinus cultures include Streptococcus pneumoniae, Moraxella catarrhalis, and Haemophilus influenzae.\textsuperscript{71}

Prominent symptoms reported by patients with chronic rhinosinusitis include nasal congestion, sinus congestion, nasal discharge, headache, fatigue, and change in olfaction.\textsuperscript{772} Although nearly 50% of patients diagnosed with chronic rhinosinusitis exhibit no growth on culture, in sinus puncture studies the most frequently isolated organisms are H influenzae, Staphylococcus aureus, and Pseudomonas aeruginosa.\textsuperscript{773} In patients with immunodeficiency, HIV positivity, or AIDS, mycobacterial, fungal, and other opportunistic organisms may be involved.

The symptoms of allergic rhinitis are frequently confused with infectious rhinitis when patients complain of a constant cold. Purulent nasal drainage may be present in either infectious or noninfectious rhinitis. Symptoms persisting longer than 2 weeks should prompt a search for causes other than infection. Foreign body rhinitis should be considered in the differential diagnosis, especially in children. Symptoms may be acute or chronic, unilateral or bilateral, and the nasal discharge may be bloodstained or foul smelling.

Exacerbations of rhinitis symptoms with predominant clear rhinorrhea in patients with a known history of allergic rhinitis may prove to be a diagnostic challenge. The distinction between active infection and allergy should be made. When the history or physical examination is not diagnostic, a nasal smear may be obtained to aid in differentiation. Early in rhinovirus infections, there is an increase in vascular permeability that is likely a result of bradykinin. Later, there may be an increase in glandular secretion, particularly of locally synthesized secretory IgA.\textsuperscript{774} Neutrophil infiltrates may be present in rhinoviral and other viral rhinitis syndromes.

Physical examination findings in both acute and chronic sinusitis may include sinus tenderness on palpation, mucosal erythema, purulent nasal secretions, increased pharyngeal secretions, and peribulbar edema. Furthermore, because these symptoms tend to overlap with those of perennial rhinitis, there is a frequent need to perform imaging studies to assist in the differential diagnosis. Nasal cytology may be useful, but the clinical value is limited by low specificity and sensitivity. Although the absence of neutrophils argues against infection, neutrophils may be present in both acute and chronic sinusitis and may be noted alongside eosinophils in allergic rhinitis during acute sinusitis (see “The Diagnosis and Management of Sinusitis: A Practice Parameter Update”\textsuperscript{775} for more detail).

Allergy, mucociliary disturbance, and immune deficiency may predispose certain individuals to the development of more frequent acute\textsuperscript{775} or chronic infections. Mucociliary abnormalities may be congenital (eg, PCD, Young syndrome, CF) or secondary to infection.\textsuperscript{776,777}

Infectious rhinitis in children

24. Viral infections account for as many as 98% of acute infectious rhinitis and the majority of rhinitis symptoms in the young child. Routine nasopharyngeal cultures when bacterial infections are suspected do not add diagnostic value.\textsuperscript{C}

Viral rhinitis, starting in the neonatal period, averages about 3 to 8 episodes per year in children and accounts for the majority of infectious rhinitis.\textsuperscript{777} The progression from viral rhinitis to secondary bacterial rhinitis occurs in approximately 10% of children and adults.\textsuperscript{72,74} These bacterial infections may progress to acute sinusitis and otitis media.\textsuperscript{779} Although it is generally accepted that atopic-prone infants and young children compared with their lower-risk cohorts appear to experience more episodes of otitis media and sinusitis,\textsuperscript{77} not all research supports this conclusion.\textsuperscript{85,86} Primary bacterial rhinitis, although uncommon, may occur in the newborn because of congenital syphilis with...
characteristic rhinorrhea followed by ulceration. Primary localized bacterial rhinitis may also occur during β-hemolytic streptococcal infections, particularly when scarlet fever is present (50% prevalence). Secondary bacterial rhinitis with or without sinusitis occurs more frequently in children with antibody, complement, and leukocyte deficiency disorders: hyper-IgE syndrome; structural defects (eg, cleft palate and osteopetrosis); and CF. In CF, S aureus and P aeruginosa are important pathogens in infectious rhinitis. Children with normal immunity may also develop secondary bacterial rhinitis with S aureus infection manifesting as impetigo of the anterior nares with characteristic crusting and secondary bacterial rhinitis with or without sinusitis.783 Patients commonly lack evidence of allergic disease as determined by skin testing and/or determinations of patients with NARES.783 Patients commonly lack evidence of allergic disease as demonstrated by absence of positive skin tests and/or specific IgE antibodies in the serum. C

Differentiating allergic rhinitis from infectious rhinosinusitis or adenoiditis may be difficult especially in children, because the symptoms overlap and even purulent nasal drainage may be present in noninfectious rhinosinusitis. Careful consideration of the need for antimicrobial use is increasingly important because antibiotic use has been causally related to the development of bacterial drug resistance.78-82 Furthermore, the administration of antimicrobials increases the carriage of antimicrobial-resistant strains of certain bacterial pathogens, such as S pneumoniae, especially in children.78,79,83,84

NARES

25. NARES is characterized by nasal eosinophils in patients who have perennial symptoms and occasionally reduced sense of smell. These patients often lack evidence of allergic disease as demonstrated by absence of positive skin tests and/or specific IgE antibodies in the serum. C

In NARES, individuals experience perennial symptoms of sneezing paroxysms, profuse watery rhinorrhea, nasal pruritus, and occasional loss of smell.101,780 Patients with NARES are at risk for development of obstructive sleep apnea.104 They are typically middle-age and have a characteristic perennial course but with paroxysmal episodes. NARES occurs extremely infrequently in childhood and probably accounts for less than 2% of children with nasal eosinophilia.781 The prevalence of this syndrome in the general population is unknown.

The etiology of the syndrome is obscure but may be an early stage of nasal polyposis and aspirin sensitivity.103 NARES is characterized by large numbers (inconsistently defined as >5% to >20%) of eosinophils on nasal smear.99-100,102,782 Similar to findings in patients with allergic rhinitis, mast cells with bound IgE and elevated tryptase have been detected in nasal mucosal biopsies of patients with NARES.782 Patients commonly lack evidence of allergic disease as determined by skin testing and/or determination of in vitro aerallergen specific IgE assays.

Occupational rhinitis

26. Occupational rhinitis is rhinitis arising in response to airborne substances in the workplace, which may be mediated by allergic or nonallergic factors, such as laboratory animal antigen, grain, wood dusts, chemicals, and irritants. It often coexists with OA. C

Occupational rhinitis may be defined as inflammation of the nasal mucosa resulting in nasal symptoms caused by exposures in the workplace. The concept of “the united airway” is likely applicable to occupational rhinitis in which the respiratory mucosa forms a continuum from the nose to the lower airways,784 whereby nasal inflammatory responses triggered by exposure to occupational sensitizers are associated with parallel inflammatory responses in the lower airways.112 Occupational rhinitis may be caused by direct effects of respiratory irritants or via immunologic mechanisms. Irritant exposures encountered in the workplace to agents such as grain dust constituents (eg, endotoxin), flour dust, fuel oil ash, and ozone elicit neutrophilic inflammation of the nasal mucosa.107-109 Alternatively, IgE-mediated sensitization and rhinoconjunctival symptoms may result from occupational exposure to protein allergens including flour, laboratory animals (rats, mice, guinea pigs, and so forth), animal products, coffee beans, natural rubber latex, storage mites, mold spores, pollen, psyllium, enzymes, and many other substances.105,106 In workers with IgE-dependent sensitization to proteins (eg, flour, laboratory animals, and natural rubber latex), eosinophils, basophils, ECP, and tryptase are significantly increased in nasal lavage after nasal allergen challenge.111-113 Some chemicals such as acid anhydrides, platinum salts, and chloramine may cause IgE-mediated occupational rhinitis.114,785 After nasal challenge with hexahydrophthalic anhydride, an acid anhydride, increased eosinophils and neutrophils have been identified in nasal lavage fluid.114 Immunologic mechanisms may be important for other chemical sensitizers (eg, glutaraldehyde and disocyanates) that cause occupational rhinitis and OA even though specific IgE is not detected consistently.766,767

The incidence of occupational rhinitis attributable to specific substances may depend entirely on the nature of industrial exposures encountered in a given geographical region. For example, the relative risk of occupational rhinitis in Finland, which has many agricultural industries, was highest among furriers, bakers, and livestock breeders.788 The prevalence of occupational rhinitis is essentially 100% among workers with OA who are sensitized to high-molecular-weight protein allergens, whereas only 50% of those with OA caused by chemicals have been identified with work-related rhinitis.510 The prevalence of work-related rhinoconjunctival symptoms is frequently reported by laboratory animal handlers (24%), although concomitant SPT reactivity to laboratory animal allergens is demonstrated in only a minority (9.6%) of symptomatic workers,106,789 suggesting that nonallergic factors may also be important. Airborne exposure to endotoxin is commonly detected in animal housing facilities and has been considered as a potential cause of occupational rhinitis, although current evidence is lacking to support effects in animal workers.789 Occupational rhinitis may precede or accompany the development of OA. Atopy and intensity of exposure are risk factors for developing occupational rhinitis.511

Symptoms may occur acutely at work after intermittent exposure or more chronically at work after continuous exposure. Occupational rhinitis should be suspected in patients with nasal symptoms, which are temporally related to exposure at work and often improve away from the workplace. An asymptomatic latency period of exposure lasting weeks to years often precedes
work-related symptoms caused by occupational respiratory sensitizers. Skin prick testing may confirm sensitization if appropriate and if suitable reagents are available. Occupational rhinitis has been evaluated with nasal allergen challenge methods that measure prechallenge and postchallenge symptoms scores, nasal lavage cells, and mediators as well as nasal airflow; however, their diagnostic validity has not been evaluated.

Irritant-induced rhinitis in an occupational setting, referred to as reactive upper-airways dysfunction syndrome (RUDS), is a chronic rhinitis syndrome triggered acutely by high-level exposure to irritants. Chronic RUDS has been reported among fire fighters exposed to complex mixtures of airborne pollutants during the World Trade Center disaster. Exposed workers present acutely with nasal burning, hypersecretion of mucus, and nasal congestion. Because there is insufficient information regarding the natural history and diagnosis of RUDS, the condition is poorly defined, and further study is required.

Optimal management of occupational rhinitis is avoidance of the occupational trigger by modifying the workplace, using filtering masks, or removing the patient from the adverse exposure. Pharmacologic therapy as discussed in earlier sections can be instituted, recognizing that chronic use of medication will probably be required. Strategies to prevent or reduce symptoms may include the daily use of intranasal corticosteroids or the administration of antihistamines and/or intranasal cromolyn immediately before allergen exposure. It is also important to institute avoidance measures for nonoccupational (and occupational) allergens that may contribute to rhinitis symptoms. Specific immunotherapy (SIT) is a possible treatment option for IgE-dependent occupational rhinitis to occupational protein allergen. Immunotherapy could be considered when 1 or a few allergens have been linked clinically to disease, avoidance of the triggering allergens is impossible, a commercial allergen extract is available, and efficacy and safety have been demonstrated to the treatment allergens. For example, SIT with pollen extracts may benefit outdoor workers with seasonal allergic rhinitis, and SIT with standardized cat allergen extract may decrease occupational symptoms among sensitized animal workers.

Trials of subcutaneous SIT have been conducted in workers with natural rubber latex allergy but failed clearly to demonstrate acceptable safety and/or efficacy. Immunotherapy is not appropriate to treat occupational rhinitis caused by low-molecular-weight chemical antigens.

Hormonal-induced rhinitis

27. Causes of hormonal rhinitis include pregnancy and menstrual cycle–related rhinitis. Pregnancy rhinitis, when present, is associated with significant nasal congestion, starts after the second month of pregnancy, and usually disappears within 2 weeks after delivery.

Rhinitis symptoms are common during pregnancy. The most common causes of nasal symptoms during pregnancy are allergic rhinitis, sinusitis, rhinitis medicamentosus, and vasomotor rhinitis of pregnancy. Allergic rhinitis worsens in approximately 1/3 of pregnant patients. Sinusitis has been reported to be 6 times more common in pregnant than nonpregnant women. Nasal vascular pooling caused by vasodilation and increased blood volume may account for worsening allergic rhinitis and increased sinusitis during pregnancy. The development of a type of rhinitis unique to the pregnant patient is referred to as vasomotor rhinitis of pregnancy or pregnancy rhinitis. It has been suggested that pregnancy rhinitis be defined as rhinitis without an infectious, allergic, or medication-related cause that starts before the last 6 weeks of pregnancy, persists until delivery, and resolves completely within 2 weeks after delivery. It has been suggested that when pregnancy rhinitis causes snoring, it may even be a factor in the development of pre-eclampsia. Elevated progesterone, estrogen, prolactin, vasoactive intestinal peptide, and/or placental growth hormone levels during pregnancy have been associated with a number of secondary phenomena. They include nasal mucosal swelling caused by vascular pooling of blood and vascular leaking of plasma into the stroma as well as the increase in glandular secretion and nasal vascular smooth muscle relaxation. However, there is no convincing evidence that any of these hormones contribute directly to pregnancy rhinitis. Pregnancy rhinitis may respond in milder cases to exercise, head of bed elevation, nasal alar dilatation, and saline rinses. Although there is no research on the safety of short-term topical decongestants combined with intranasal corticosteroids in pregnancy, these have been suggested for management of pregnancy rhinitis when the measures discussed are not effective.

During the menstrual cycle, nasal congestion has been shown to concur with ovulation and rise in serum estrogens, although additional evidence supporting this relationship is lacking.

Drug-induced rhinitis

28. Drug-induced rhinitis may be caused by a number of medications, including ACE inhibitors, phosphodiesterase–5–selective inhibitors, phospholamine, α-receptor antagonists, aspirin, and other NSAIDs. Rhinitis medicamentosus is a syndrome of rebound nasal congestion that follows the overuse of intranasal α-adrenergic decongestants or cocaine.

Medications may induce symptoms of nasal congestion and/or rhinorrhea. In the past, antihypertensive medications (eg, reserpine and guanethidine) were frequently incriminated, but they are no longer commonly used. Currently, ACE inhibitors and β-blockers occasionally elicit rhinitis symptoms. Rhinitis symptoms are often caused by α-receptor antagonists used in treatment of benign prostatic hypertrophy (eg, prazosin, terazosin). Phosphodiesterase–5–selective inhibitors used for treatment of erectile dysfunction can cause nasal congestion.

Phentolamine mesylate, an α-1 and α-2–selective adrenergic receptor antagonist, has been reported to cause rhinitis symptoms in 7.7% of patients treated for erectile dysfunction. Although oral contraceptives have long been implicated as causes of nasal symptoms, a recent study found no nasal physiologic effects on female patients receiving a modern combined oral contraceptive pill. Aspirin and NSAIDs may produce rhinorrhea as an isolated symptom or as part of a symptom complex of rhinosinusitis, nasal polyposis, and asthma. Prolonged usage of α-adrenergic decongestants may lead to a hypertrophy of the nasal mucosa termed rhinitis medicamentosus. The repetitive and prolonged use of topical α-adrenergic nasal decongestant sprays may induce rebound nasal congestion on withdrawal. These agents include OTC products containing oxymetazoline or phenylephrine. Benzalkonium chloride in vasoconstrictor spray products may augment local pathologic effects. Also, patients may develop tachyphylaxis because of the need
for more frequent doses to provide adequate decongestion. The nasal mucosa is often beefy red, appears inflamed, and shows areas of punctate bleeding and scant mucus. There is loss of ciliated epithelial cells leading to reduced mucociliary clearance. Nasal septal perforation is a rare complication. Similar consequences may occur with prolonged use of other vasoconstrictor agents such as cocaine.

**Atrophic rhinitis**

29. Treatment of primary and secondary atrophic rhinitis involves reducing crusting and alleviating the foul odor by continuous nasal hygiene, such as nasal lavage and crust debridement, and the use of topical and/or systemic antibiotics when purulent secretions or an acute infection is present.

Primary (idiopathic) atrophic rhinitis, more prevalent in developing countries with warm climates, is a chronic condition characterized by progressive atrophy of the nasal mucosa, nasal crusting, nasal dryness (caused by atrophy of glandular cells), and fetor. The nasal cavities appear abnormally wide on examination, and squamous metaplasia, atrophy of glandular cells, and loss of pseudostratified epithelium have been detected in nasal biopsies. The sinus CT shows the characteristic resorption of underlying bone and the absence of identifiable turbinates, a finding referred to as the “empty nose syndrome.” The dryness and reduction of nasal mucosal tissue with the resultant resistance to airflow are, paradoxically, perceived by patients as severe nasal congestion. The etiology of primary atrophic rhinitis has not yet been established. Klebsiella ozaenae and other bacteria including *S. aureus*, *Proteus mirabilis*, and *Escherichia coli* may be causative, although it is also plausible that these secondarily infect previously damaged nasal mucosa. A genetic association has also been suggested but needs further confirmation.

Primary atrophic rhinitis should be separated from secondary atrophic rhinitis, which develops as a direct result of other primary conditions, such as chronic granulomatous disorders, chronic sinusitis, excessive surgery to the nasal turbinates, trauma, and irradiation. Although secondary atrophic rhinitis may be less severe and progressive, treatment of primary and secondary atrophic rhinitis is aimed at reducing crusting and alleviating the foul odor. There are no controlled trials evaluating therapies for atrophic rhinitis. Although even the published observational data are limited, the mainstay of treatment is continuous nasal hygiene—for instance, intranasal irrigations with saline or sodium bicarbonate solution, and periodic debridement of the crusts, if necessary. As used for recalcitrant rhinitis and sinusitis, adding antibiotics such as mupirocin to the lavage solution has been suggested for purulent secretions. Systemic antibiotics are indicated when an acute infection is present.

**Nasal polyps**

30. Nasal polyps may occur in conjunction with chronic rhinitis or sinusitis and may contribute significantly to the patient’s symptoms. Nasal polyps should always be considered in the differential diagnosis of patients who present with invariant nasal congestion and/or anosmia and its sequelae. Allergy as a cause of nasal polyps has not been established, but nasal polyps may occur in conjunction with allergic rhinitis.

Nasal polyps are an inflammatory condition of the nasal and sinus mucosa and usually presents as persistent nasal obstruction. Nasal polyps have a prevalence of 2% to 4% in the general as well as the allergic population and usually occur after age 40 years. Although previous studies showed a 2:1 male to female prevalence of nasal polyps, a recent large population study showed no sex preference. Frequently reported symptoms are rhinorrhea (39%), nasal congestion (31%), and anosmia (29%). Chronic nasal polypsis has been associated with reduced QOL and greater risk of sleep disturbances.

Nasal polyps may occur in as many as 50% of children with CF, in immotile cilia syndrome, and in 7% to 15% of adults with asthma. Nasal polyps coupled with AERD are rarely noted before the adolescent years. The profile of inflammatory mediators (eg, myeloperoxidase and IL-8) and prominent numbers of neutrophils found in nasal polyps of patients with CF differ from increased eosinophils and associated mediators (eg, IL-5, eotaxin, ECP) detected in adults with non-CF polyps, suggesting that these are different disorders.

AERD is recognized in 13% of patients with nasal polypsis. In adult patients without CF, the pathogenesis of nasal polypsis is uncertain. Infiltrates of eosinophils, T cells, plasma cells, and mast cells are consistent findings in nasal polyp tissue and may explain why corticosteroids are therapeutically effective. When associated with asthma, patients with nasal polypsis hyperexcrete urinary 

Reduced apoptosis of eosinophils in nasal polyp tissue has been suggested to play a pathogenetic role. Reduced apoptosis of eosinophils in nasal polyps has been demonstrated, which could enhance tissue inflammation and growth of nasal polyps. Allergy does not appear to predispose to polyp formation. Between 10% and 15% of patients with allergic rhinitis also have nasal polyps. The noses and sinuses of patients with chronic sinusitis and nasal polyps are frequently colonized with fungi (principally *Aspergillus* and *Penicillium*). Allergic fungal sinusitis is a distinct pathologic entity defined by specific IgE for *Aspergillus*, *Bipolaris*, or other mold antigens; chronic sinusitis with nasal polypsis; radiographic sinus opacification and eosinophilic mucin material containing eosinophilic debris; and fungal hyphae in tissue resected from the sinuses. Proinflammatory mediators including eosinophilic major basic protein and neutrophil elastase have been identified in allergic mucin. The efficacy of local or systemic antifungal therapy in treating allergic fungal sinusitis has not been established.

The treatment and control of nasal polyps is challenging. Sinus disease and nasal polyps are more difficult to control in patients with asthma and AERD. Intranasal corticosteroids are effective in improving sense of smell and reducing nasal congestion, and effects are optimized with twice-daily versus once-daily dosing. For severe nasal polypsis, a short course of oral prednisone is effective in reducing symptoms and polyp size and improving nasal flow. The beneficial effects are then maintained by subsequent administration of maintenance intranasal corticosteroids. Subjective improvement in nasal polyp symptoms has been observed in patients administered the LT modifiers montelukast, zafirlukast, and zileuton as add-on therapy to intranasal corticosteroids. After sphenoethmoidectomies, recurrence rates and rescue medication requirements in patients treated with montelukast were equivalent to those observed in patients receiving postoperative nasal beclomethasone. In recent years, functional endoscopic sinus surgery has been used extensively for treating rhinosinusitis associated with nasal polypsis. Patients with AERD and nasal polyps have worse outcomes with functional endoscopic sinus surgery
than aspirin-tolerant patients.\textsuperscript{817} In patients with nasal polyposis and AERD, aspirin desensitization followed by long-term daily aspirin treatment may be considered. Long-term studies of patients with AERD suggest that maintenance aspirin therapy may reduce nasal symptoms, frequency of sinus infections, requirement for nasal polypectomies, and need for systemic corticosteroids.\textsuperscript{155,156} The numbers of nasal mucosal leukocytes expressing cysLT receptors (cysLT1), which are increased in nasal mucosa of patients with AERD, decrease after aspirin desensitization.\textsuperscript{157}

Other conditions that may be confused with rhinitis
Anatomic abnormalities

31. Signs and symptoms suggestive of rhinitis can be produced by other conditions, including nasal septal deviation, tumors, and hypertrophy of the nasal turbinates. C

32. In infants and young children, nasal congestion or obstruction can result from structural problems, such as cleft palate and adenoidal hypertrophy, or functional problems, such as laryngopharyngeal reflux. D

Nasal obstruction may be caused by congenital or acquired anatomic abnormalities, which may mimic symptoms of rhinitis. Reduced airflow through the nasal passages in infants may be a result of congenital choanal atresia. Nasal septal deviation and nasal turbinate or adenoidal hypertrophy may block flow of nasal secretions, leading to rhinorrhea or postnasal drip, as well as causing nasal blockage.

Although comparatively rare, both benign and malignant tumors may cause rhinitis symptoms.\textsuperscript{158,159} Lesions generally cause unilateral occlusion of the nasal airway. Rapidly growing nasal malignancies may cause nasal obstruction early in the disease. Lesions arising in the maxillary sinus present with nasal symptoms in the late stages of the disease, usually after the tumor has penetrated the medial wall of the antrum. These tumors may present with bleeding, hyposmia or anosmia, pain, and/or otalgia. Prolonged occupational exposure to chemicals such as nickel and chrome have been associated with carcinoma.\textsuperscript{818}

Systemic immunologic and nonimmunologic diseases may affect the nose. These include Wegener granulomatosis, sarcoidosis, relapsing polychondritis, and midline granuloma. Patients with uremia develop thinning of the nasal epithelium.\textsuperscript{821} At times, the systemic symptoms may be absent or undetected when patients present with nasal complaints. Infections such as tuberculosis, syphilis, leprosy, sporotrichosis, blastomycosis, histoplasmosis, and coccidiomycosis also may cause granulomatous nasal lesions. These are usually ulcerative, and crust formation may lead to nasal obstruction or bleeding. Rhinoscleroma is a rare chronic infectious granulomatous disease caused by \textit{Klebsiella rhinoscleromatis} that presents as a polypoid mass with symptoms of epistaxis and nasal obstruction.\textsuperscript{822} Rhinoscleroma is endemic in tropical and subtropical regions of America, Asia, and Africa.

Complete or partial nasal obstruction in the infant below 2 to 6 months of age can lead to fatal airway obstruction, because many neonates are obligate nasal breathers. In the newborn, the nasal passages may contribute to 50% of the total airway resistance.\textsuperscript{93,161} Thus, any minor increase of congestion, such as a URI, can create near total obstruction. Although food allergy, such as, milk, is often considered to contribute to nasal symptoms including congestion, 1 large prospective study demonstrated that only .3% of food hypersensitivity in children and adolescents is associated with rhinitis symptoms.\textsuperscript{707} Cinematoradiographic findings have implicated a causal relationship of nasal obstruction and sudden infant death syndrome.\textsuperscript{823}

The most common acquired anatomic cause of nasal obstruction in infants and children is adenoidal hypertrophy. Enlarged adenoids commonly result in mouth breathing, nasal speech, and snoring. The main indication for adenoidectomy, a common outpatient surgical procedure in children, is sleep apnea caused by adenontonsillar hypertrophy, chronic adenoiditis, and chronic sinusitis. Nasal symptoms, such as congestion, are also common in infants and children with pharyngonasal reflux resulting from prematurity, neuromuscular disease, dysautonomia, velopharyngeal incoordination, or cleft palate.\textsuperscript{161} This is a result of inflammation and narrowing of the posterior choanae because of acid inflammation.\textsuperscript{161} When gastroesophageal reflux involves the upper esophagus, larynx, and/or pharyngeal area, it is often referred to as \textit{laryngopharyngeal reflux}. Infants with laryngopharyngeal reflux experience frequent choking, apneic spells, recurrent pneumonia (because of concomitant gastroesophageal reflux and/or tracheal aspiration), and aspiration of formula leading to secondary chemical/infectious rhinitis. Although diagnosis of laryngopharyngeal reflux can usually be made with nasopharyngoscopy, milk scintigraphy or a pH probe study may be required in select cases.\textsuperscript{161} Thickened feedings, positioning upright after feeding, and the use of histamine-2 receptor antagonists or proton pump inhibitors have become the mainstay of treatment.\textsuperscript{161,824} Of some concern is a recent pediatric prospective study showing that therapy with gastric acidity inhibitors, both protein pump inhibitors and H2 blockers, may increase the risk of acute gastroenteritis and community acquired pneumonia,\textsuperscript{825} a finding that has also been described in adults.

CSF rhinorrhea

33. Refractory clear rhinorrhea may be a result of CSF leak, which is often caused by trauma or recent surgery. B

Cerebral spinal fluid rhinorrhea should be differentiated from the rhinorrhea found in patients with chronic rhinitis. Refractory clear rhinorrhea may be a result of CSF leak even in the absence of trauma or recent surgery, although these remain the most common causes of CSF leak.\textsuperscript{160} Benign intracranial hypertension or pseudotumor cerebri, which typically presents in middle-age women with chronic headaches, has been implicated as a cause of spontaneous, nontraumatic CSF rhinorrhea.\textsuperscript{826,827} Although detection of glucose historically has been used as an indication for its presence,\textsuperscript{828} \(\beta\)-2-transferrin protein is a more sensitive and specific indicator because it is found in cerebral spinal fluid and inner ear perilymph, but not in blood, nasal, or ear secretions.\textsuperscript{255,256}

Ciliary dysfunction syndromes

34. Ciliary dysfunction can be primary (PCD) or secondary (eg, viral infection) and may contribute to recurrent rhinitis and sinus infections. C

Defective ciliary function in the airway may be described as ciliary immotility (no movement), ciliary dyskinesia (abnormal movement), or ciliary aplasia (absence of cilia). Screening diagnostic techniques for upper airway disease include measures of mucociliary clearance with saccharin or Teflon particles tagged with the short-lived isotope technetium 99m. An absence of mucociliary clearance is a sign of immotility, dysmotility, or aplasia that may be congenital or acquired.
Primary ciliary dyskinesia, also known as immotile-cilia syndrome, is a rare genetic defect (1/20,000 to 1/60,000) caused by a genetically, functionally, and ultrastructurally diverse or heterogeneous disease involving a defect in ciliary function. Defective epithelial ciliary clearance of secretions from the upper airway compartments including eustachian tubes and sinuses as well as lower airways produces chronic inflammatory injury to these areas. Approximately 50% of subjects with PCD are affected by situs inversus (Kartagener syndrome). The majority have an autosomal-recessive form of PCD, but autosomal-dominant and X-linked cases have been reported.

The clinical history is usually that of respiratory disease in the newborn or recurrent upper and lower respiratory disease including recurrent sinusitis, otitis, otosalpingitis, rhinitis, chronic cough, and nasal polyposis. It may also be associated with difficult-to-control asthma and/or cylindrical or saccular bronchiectasis demonstrated on a chest radiograph or chest CT. Additional clinical findings include situs inversus, agenesis of the frontal sinuses, hydrocephalus, heterotaxy, and infertility. Spirometry reveals mild to moderate obstruction with a positive response to a bronchodilator. Any of these clinical findings in combination with a positive family history of diagnosed or probable PCD warrant a full investigation.

Secondary ciliary defects are certainly more common than PCD. Natural as well as experimentally induced viral upper respiratory infections have been associated with prolonged mucociliary clearance as measured by radiolabeled resin beads or dyed saccharin. This effect is maximum at 3 days, persists up to 11 days, and is found in a higher proportion of patients with versus without allergy. Acute viral infections may also cause cytopathic epithelial damage that may take a number of weeks to resolve. Tobacco smoking of 1 or more cigarettes in an in vitro frog palate model results in a reduction of mucus clearance and disruption and defoliation of the ciliated epithelium. Although it is often assumed that similar effects occur in human beings, 1 study using saccharin clearance time demonstrated normal mucociliary clearance in healthy smokers, although the average clearance time was longer than in nonsmokers. Likewise, there was no significant difference in mean nasal ciliary beat frequency when comparing healthy smokers with nonsmokers. It has been suggested that tobacco smoking leads to the dysfunction of the normal metachronal waves that drive mucus over nonciliated areas and that prolonged, sustained exposure to cigarettes may produce loss of ciliated epithelium secondary to activation of matrix metalloproteinases. However, even this ciliary dysfunction shows at least partial recovery within 30 minutes of cigarette smoke avoidance in pure air.

Evaluation of rhinitis

History

35. An effective evaluation of the patient with rhinitis often includes the following: a determination of the pattern, chronicity, and seasonality of nasal and related symptoms (or lack thereof), response to medications, presence of coexisting conditions, occupational exposure, and a detailed environmental history and identification of precipitating factors.

An appropriate general medical history and a thorough allergic history are usually the best diagnostic tools available. If a structured allergy history questionnaire is used, the physician should review this with the patient during the initial evaluation. When obtaining an allergic history, it is important to ask about chief concerns and symptoms, including the patient’s perception of what is causing the allergic symptoms or the patient’s self-diagnosis (although this may be misleading) as well as directed questions relating to nasal symptoms. For example, questions relating to symptoms to exposure to pollen and animals may have positive predictive value for diagnosing allergic rhinitis. Each complaint or symptom should be separately evaluated for date of onset including (1) related or resolved symptoms from infancy or childhood; (2) frequency (eg, continual or episodic); (3) characteristics (eg, color and tenacity of nasal secretions); (4) pattern (eg, seasonal, perennial, or a combination); (5) severity, both past and present; (6) triggers that precipitate or worsen the complaint, including allergens, irritants, hormonal influences, exercise, eating, medications, and weather changes; (7) timing after exposure to trigger (eg, immediate or delayed onset); (8) association with geographical and environmental (eg, home vs work vs day care) location or relationship with a particular activity or event (eg, dusting or raking leaves); (9) previous medical evaluation and treatment results, including specific pharmacologic success or failure; and (10) severity (an estimate of effect of the allergic symptoms on QOL, including work or school performance and sleep quality). When a likely allergen is identified by history, a directed question regarding willingness to modify the exposure, such as house pet or occupational allergen, can be asked of the patient and family/care givers. In addition, preferences for the treatment of allergic symptom control including delivery method (eg, oral or nasal) of pharmacologic therapy or a long-term treatment approach with allergy immunotherapy may be explored with the patient and/or others involved in this decision.

Each patient concern or symptom should be evaluated and documented. This could include (1) nasal congestion, sneezing, and rhinorrhea; (2) throat symptoms of soreness, dryness, and PND; (3) cough; (4) ocular redness, tearing, and itching; (5) voice changes; (6) sinus pain or pressure; (8) ear pain, blockage, or reduced hearing; and (9) itching of nose, ears, or throat. Hyposmia and anosmia are most often associated with severe obstructive upper airway disease, frequently caused by the presence of nasal polypos. It may be helpful to question the patient about symptoms of fatigue, irritability, poor quality sleep, absenteism and presenteeism at work and/or school, and general QOL problems during their symptomatic periods.

When reviewing the allergic history in children, one may inquire about sniffing, snorting, clearing of the throat, chronic gaping mouth, halitosis, cough, dark circles under the eyes, and eye rubbing. The parents may describe the child as having a poor appetite, learning or attention problems, sleep disturbances, malaise, irritability, and a general sense of not feeling well. When allergic symptoms are recurrent but nonseasonal, the presenting complaint may be recurrent URI, because the parent cannot differentiate a URI in a young child attending day care from allergic rhinitis. Further complicating this differentiation is the increased responsiveness of the nasal mucosa to allergens and irritants after a viral URI.

The history for rhinitis generally includes the family history of allergic rhinitis, asthma, atopic dermatitis, chronic sinus problems or infections, diagnoses that may represent allergic symptoms such as recurrent bronchitis, and all major nonallergic medical diagnoses. Although a positive family history makes it more likely that...
the patient’s nasal symptoms are a result of allergic rhinitis, a negative family history by no means rules out the diagnosis of allergic rhinitis. Important components of the initial interview include the past medical history, previous trauma or surgery to the nose and sinuses, and established allergic and nonallergic medical diagnoses. Response to previous therapeutic interventions, such as pharmacotherapy or surgery, should be discussed. Documentation of all current medications or herbal preparations, physician-prescribed or OTC, is recommended. An environmental survey should be conducted exploring potential sources for indoor and outdoor allergens in the patient’s home and the homes of close relatives, friends, and caretakers, as well as school and work settings. Questions relating to the indoor environment will usually include items such as the presence of pets and insects, carpet, curtains, and upholstered furniture; age and composition of mattress, pillows, and bed coverings; and the cleaning methods in use. One should inquire about the air conditioning and heating sources and their customary use patterns. It is also important to determine the presence of active or passive tobacco smoke exposure.

36. Evaluation of rhinitis therapy should include assessment of QOL. C

At any age, allergic rhinitis can compromise QOL. In adults, this often manifests as sleep disorders, impairment at work, limitations of activities, or impairment of social functioning. Recent findings that the sexual QOL is affected by seasonal allergic rhinitis and that appropriate treatment brings the patient’s sexual functioning back toward normal emphasizes that allergic rhinitis is an underappreciated disease with systemic effects. As evidence of the disparities between patients’ and physicians’ perspectives of allergic rhinitis, the symptom severity and the reduced work, home, and social functioning, as indicators of QOL, are often underrecognized and inadequately treated by the patient’s physician. Understanding the impact of allergic rhinitis on the patient’s QOL represents the cornerstone of therapy.

There are definitely some significant consequences of untreated allergic rhinitis in children. Unrecognized or uncontrolled allergic rhinitis can lead to structural complications and permanent disfigurement such as increased facial length, a high arched palate, class II dental malocclusions, and retrognathic maxilla and mandible. Allergic rhinitis that is poorly controlled can result in poor sleep, school absenteeism, learning impairment, inability to integrate with peers, anxiety, and family dysfunction. After effective treatment of perennial allergic rhinitis, improvement in school attendance, school work concentration, and sleep can be demonstrated. Furthermore, chronic nasal congestion and secondary sleep apnea and disordered sleep can lead to systemic symptoms of headache, fatigue, irritability, poor growth, and reduced QOL. The psychological ramifications of untreated allergic rhinitis can lead to low self-esteem, shyness, depression, anxiety, and fearfulness.

The effect of rhinitis on QOL has been measured using both generic and disease-specific questionnaires (Tables III and IV). The advantage of using a generic questionnaire is that the burden of rhinitis can be compared with other diseases, such as asthma. In fact, in adults, moderate-to-severe perennial rhinitis and moderate-to-severe asthma have equal functional impairment. On the other hand, disease-specific QOL questionnaires describe disease-associated problems more accurately and seem to be more responsive to measuring the change with therapeutic interventions. Adult generic questionnaires include the Sickness Impact Profile, the Duke Health Profile, the Nottingham Health Profile, and the Medical Outcome Survey Short Form 36 (Table III). The Short Form 36 has been used to evaluate the effects of a nonsedating H1-antihistamine on QOL. Generic QOL questionnaires in children, such as the Child Health Questionnaire–Parental Form 50, Pediatric Quality of Life Inventory, and SF-10 (Table III), are used at times for comparing different allergic diseases. Disease-specific rhinitis QOL questionnaires have been adapted to different age groups. For example, when studying rhinitis in children the Juniper Pediatric Rhinoconjunctivitis Quality of Life Questionnaire (RQLQ) can be used for ages 6 to 12 years and Juniper Adolescent RQLQ for ages 12 to 20 years (Table IV). Although both the generic and disease-specific QOL questionnaires are often used in research trials for the evaluation of a study group, their sensitivity and precision for use with individual patients has been questioned, especially because QOL and clinical measurements may not be fully interchangeable and distortion, masking, or faking can occur. For example, there are studies documenting a clear clinical improvement after antihistamines or bronchodilators, but these are not accompanied by detectable changes in the health-related quality of life. In contrast, in other studies, the clinical evaluation did not discriminate between 2 different treatments, whereas a difference was noted with QOL assessment. Use of generic QOL measurements in individual patients when studied in other areas of medicine has not led to changes in practice style and has not improved patients’ health outcomes. While we await the development of a tool that will be valid and useful for following the individual patient, the clinician may find the current QOL questionnaires of benefit in select clinical settings and in clinical trials. The use of a modified visual analog (graphic rating) scale for assessing the severity of allergic rhinitis has been recommended for the clinician when assessing the individual patient for nasal and nonnasal symptom severity, global nasal and nonnasal severity, and QOL assessment of rhinitis severity (see Figs 1-4). We are also starting to see the development of rhinitis instruments, such as the Multiattribute Rhinoitis Symptom Utility index, to assist in comparing the cost effectiveness of various medical treatments of rhinitis.

Physical examination

37. A physical examination of all organ systems potentially affected by allergies with emphasis on the upper respiratory tract should be performed in patients with a history of rhinitis. The nasal examination supports but does not definitely establish the diagnosis of rhinitis. D

The elements of the physical examination of the patient with rhinitis with emphasis on the nasal passages are described in Table V. The examiner should carefully look for any signs of accompanying otitis, eustachian tube dysfunction, acute or chronic sinusitis, allergic conjunctivitis, asthma, and atopic dermatitis in addition to findings compatible with rhinitis. In children, findings of dental malocclusion, a high-arched palate, and upper lip elevation may suggest early-onset and/or longstanding disease. On the other hand, if the patient is asymptomatic at the time of the physical examination, there may be minimal or no findings even with a history suggestive of rhinitis.
The nasal examination should note the patency of the nasal valve and any alar collapse. If there is improvement in breathing when performing the Cottle maneuver—pulling the patient’s cheek laterally to open the nasal valve angle—this may suggest nasal valve pathology. A deviated/deformed nose may suggest previous trauma, whereas a saddle nose deformity may indicate previous trauma, previous surgery, cocaine abuse, or an inflammatory process.

The nasal and nasopharyngeal examination is accomplished with a nasal speculum with appropriate lighting, otoscope with nasal adapter, indirect mirror, and/or rigid or flexible nasopharyngoscope, based on the expertise of the examiner and the assessment needs. The anterior rhinoscopy examination will reveal any caudal septal deformity or inferior turbinate hypertrophy. If there is significant caudal septal deflection, the inferior turbinate on the side opposite the deviation is often enlarged. If after applying a topical decongestant there is a reduction of turbinate mucosal edema, this may assist in delineating mucosal versus bony hypertrophy.

The use of the nasopharyngoscope allows for better visualization of the middle meatus, the posterior septum, the sinus ostia, posterior choanae, the nasopharynx, and the presence of nasal polyps. The presence of mucopurulent material in this region is suggestive of sinusitis. The use of a mirror or the nasopharyngoscope is necessary to complete a posterior rhinoscopy examination. A pneumatic otoscope is used to assess tympanic membrane mobility. At times, the impedance tympanometer is also needed to assess the tympanic membrane mobility and the presence or absence of fluid, especially in children.

Many typical allergic findings are supportive but not specific to allergic rhinitis. Mucosal appearance may not distinguish between allergic and nonallergic rhinitis, because nonallergic rhinitis may also present with mucosal pallor, edema, or hyperemia. The mucosa is usually reddened in acute infections and with overuse of topical decongestant sprays. Occasionally, the mucosa can be hyperemic with allergic rhinitis. Dennie-Morgan lines, often noted in patients with atopic dermatitis, are symmetrical, prominent folds extending from the medial aspect of the lower lid. These are usually present at birth, or appear shortly thereafter, and persist for life. Dennie-Morgan lines (noted in 60% to 80% of patients) are similar to the folds seen in patients with Down syndrome. They may have an ethnic variation and are characterized but not exclusively present in patients with allergy. Allergic shiners, asymmetrical, symmetrical, blue-grey discolorations of the periorbital skin, are most apparent below the orbit and are attributed to venous stasis. They are reported to occur in as many as 60% of atopic patients and in 38% of nonatopic individuals. Their presence is usually associated with nasal congestion. These do tend to fade with increasing age and are often found in atopic family members.

The quantity and quality of nasal secretions should be noted. With allergic rhinitis, there may be watery mucus on the epithelial surface or on the floor of the nasal passage. With abnormal mucociliary clearance or total nasal obstruction, thick secretions can be seen pooling in the floor of the nose. Unlike the nasal turbinates, with which they are often confused, polyps appear glistening, mobile, and opaque and are insensitive to touch. Nasal polyps may be differentiated from severely edematous mucosa by applying a small amount of topical vasoconstrictor such as phenylephrine to the mucosa and re-examining the mucosa 5 to 10 minutes later. Nasal polyps will not shrink in size after topical vasoconstrictor has been applied, unlike edematous mucosa. Crusting on an inflamed mucosa may suggest atrophic rhinitis or a systemic disease such as sarcoidosis. The presence of a septal perforation should raise the possibility of intranasal narcotic abuse, adverse effects of other topical preparations, previous surgery, or systemic granulomatous diseases.

### Testing for specific IgE antibody

**Skin testing**

38. Determination of specific IgE, preferably by skin testing, is indicated to provide evidence of an allergic basis for the patient’s symptoms, to confirm or exclude suspected causes of the patient’s symptoms, or to assess the sensitivity to a specific allergen for avoidance measures and/or allergen immunotherapy.

39. Skin tests are the preferred tests for the diagnosis of IgE-mediated sensitivity. The number of skin tests and the allergens selected for skin testing should be determined on the basis of the patient’s age, history, environment, and living situation, such as area of the country, occupation, and activities.

**In vitro assays for specific IgE**

40. The precise sensitivity of specific IgE immunoassays compared with skin prick/puncture tests is approximately 70% to 75%. Immunoassays have similar sensitivity to skin tests in identifying those patients with nasal symptoms elicited after natural or controlled allergen challenge tests.

41. Interpretation of specific IgE immunoassays may be confounded by variables such as potency of allergens bound to solid support systems, cross-reactive proteins and glycoepitopes, specific IgG antibodies in the test serum, and high total IgE.
In vitro specific-IgE tests (eg, RAST) are an alternative to skin testing. The precise sensitivity of specific IgE immunoassays compared with skin prick/puncture tests has been reported to range from less than 50% to greater than 90%, with the average being around 70% to 75% for most studies. Skin tests are therefore the preferred tests for the diagnosis of IgE-mediated sensitivity. Interpretation of specific IgE tests may be confounded by variables such as potency of allergens bound to solid support systems, cross-reactive proteins and glycoepitopes, specific IgG antibodies in the test serum, and high total IgE (see “Allergy Diagnostic Testing: An Updated Practice Parameter” for more detail).

Specific IgE immunoassays may be preferable to skin testing under special clinical conditions such as widespread skin disease (ie, severe eczema or dermatographism), skin test suppressive therapy in use, an uncooperative patient, or a history suggesting an unusually high risk of anaphylaxis from skin testing.

Special diagnostic techniques

42. In selected cases, special techniques such as fiber optic nasal endoscopy and/or rhinomanometry may be useful in evaluating patients presenting with rhinitis symptoms. These tests may require special expertise for performance and interpretation. Patients with nasal disease require appropriate examination for associated diseases, such as sinusitis and otitis media.

Although history and routine physical examination are usually sufficient for a diagnosis of allergic rhinitis, special techniques may be useful in selected patients. Furthermore, patients with nonallergic upper airway pathology may initially report symptoms suggestive of rhinitis.

Upper airway endoscopy

Endoscopy may be especially useful for the evaluation of rhinitis when symptoms or physical findings are atypical, complications are noted, therapeutic response is suboptimal, or conditions other than rhinitis are suspected. Without endoscopy, it is not possible to view many of the important recessed structures of the upper airway. Either the rigid or a flexible fiber optic endoscope can be used for this examination.

Upper airway endoscopy (rhinolaryngoscopy) is the most useful diagnostic procedure in an evaluation for anatomic factors causing upper airway symptoms. Endoscopy provides a clear view of the nasal cavity and allows detailed examination of the middle meatus, superior meatus, sphenethmoidal recess, posterior choanae, and nasopharynx, as well as the structures of the oropharynx and larynx. The procedure is usually performed in the office after decongestion and topical anesthesia. Analysis of videotaped fiber optic upper airway endoscopy has also been used as a research technique to measure cross-sectional area of the nasal cavity.

Imaging techniques

The primary goals of radiologic imaging of the upper airway are to provide an accurate reproduction of the regional anatomy and to establish the presence and extent of anatomic disease. This information may assist in planning medical therapy and provide an anatomic guide to facilitate subsequent surgical treatment.

Radiologic imaging techniques, such as plain films, CT, and MRI, have no use in the evaluation of patients with uncomplicated rhinitis. However, imaging may be merited when rhinitis with complications or comorbidities such as nasal polyposis and concomitant sinusitis are present.

CT and MRI

Computerized tomographic scanning and MRI using coronal sections for imaging of sinuses frequently identify turbinate congestion, concha bullosa, nasal polyps, and septal deviation as causes of nasal airway obstruction. Although CT and MRI have been used to validate acoustic rhinometry as a diagnostic technique, they are expensive and may not correlate well with functional obstruction when used for this purpose.

High-resolution CT can demonstrate disease that is not shown on routine x-ray films. It can also delineate pathologic variations and demonstrate anatomic structures inaccessible by physical examination or endoscopy. Because of its superb contrast resolution, CT is an excellent method for examining the complex anatomy of the upper airway, particularly the ostiomeatal complex. The capability of CT to display bone, soft tissue, and air facilitates accurate definition of regional anatomy of the nose and paranasal sinuses. The main indications for the CT are chronic sinusitis not responding to appropriate medical therapy, acute recurrent sinusitis, abnormal diagnostic nasal endoscopic examination, and persistent facial pain.

Magnetic resonance imaging provides better imaging of soft tissue than CT, but it is less suited to imaging the bony anatomy of this region. MRI is useful in the evaluation of upper airway malignancies.

Aerodynamic methods for estimation of nasal airway obstruction

Resistance to airflow through the nose (or conductance, the inverse of resistance) may be measured by rhinomanometry. Rhinomanometry objectively measures functional obstruction to airflow in the upper airway, although the technique has not been fully standardized. Subjective perception of nasal stuffiness may correlate only loosely with measured nasal airway resistance, and rhinomanometry may be used in the assessment of the severity of symptoms. In addition, rhinomanometry may provide objective information on the results of therapeutic interventions. The objective information obtained from rhinomanometry may be particularly important when it is suspected that occupational exposure results in nasal symptoms, including nasal congestion. Rhinomanometry is not a substitute for careful endoscopy of the nose because significant pathology in the nose can occur with nasal airway resistance values in the normal range. Rhinomanometry may be used to assess the severity of anatomical abnormalities that are causing airway obstruction in the nose, including nasal valve abnormalities, septal deviation, and polyposis. This application requires measurements before and after a potent intranasal decongestant. Other indications for rhinomanometry include the evaluation of patients with obstructive sleep apnea.

Acoustic rhinometry

Acoustic rhinometry depends on reflection of acoustic signals from structures in the nasal cavity. It is currently not a technique used in the routine evaluation of patients with rhinitis. It produces an image that represents variations in the cross-sectional dimensions of the nasal cavity and closely approximates nasal cavity volume and minimal cross-sectional area. It also allows identification of the distance of the minimal cross-sectional area of the nasal cavity from the nares. Changes in nasal geometry measured by acoustic rhinometry during histamine challenge testing have been documented, and the results of parallel...
44. Although the saccharin test for mucociliary clearance has been performed on a severely congested nose because it does not require nasal flow. Measurements by acoustic rhinometry have been validated by comparison with CT and MRI. Although there is high correlation for the anterior 2/3 of the nasal cavity, the posterior nasal cavity shows more variability. Compared with CT and MRI, acoustic rhinometry is more rapid, may be performed at the bedside, and, unlike CT, does not expose the patient to radiation. Acoustic rhinometry and rhinomanometry have similar reproducibility and compare favorably in challenge studies, but measure different changes and are best viewed as complementary. Clinically, acoustic rhinometry is used to help diagnose rhinitis, evaluate nasal pharyngeal surgical outcome, and monitor response and adherence to medical therapy such as intranasal corticosteroids. It is a logical choice for the objective measurement of area and volume in diseases of the nose. A number of factors lead to measurement variation when the procedure is used. Common reasons for measurement inaccuracy are an air leak between the nosepiece and the nose and the presence of nasal secretions. Studies have shown that patient perception of nasal obstruction does not correlate with actual compromised nasal airflow.

Nasal provocation testing

Identification of sensitivity of the nose to a particular allergen usually can be based on a history of symptoms of allergic rhinitis provoked by exposure to the allergen and confirmed by skin testing. Nasal provocation testing with allergen is unnecessary unless criteria that are more stringent are needed to incriminate the suspected allergen. For example, nasal provocation testing with allergen may be required for confirmation of sensitivity to allergens in the workplace. Testing of sensitivity to allergens requires that responses to incremental doses of allergens be assessed. Single-dose allergen provocation measures nasal reactivity to allergens, not sensitivity. Because nasal reactions to instillation of placebo materials may occur, response to diluent must be measured before provocation with allergens.

Nasal sensitivity/hyperresponsiveness to histamine and methacholine has been found in allergic rhinitis and vasomotor rhinitis. Although this measurement may be a marker for these diseases, the clinical utility of nasal provocation testing with histamine or methacholine may be limited; there is a considerable overlap between patients with and without allergy in their sensitivity to these agents. However, these provocation tests may be useful during clinical trials to determine the efficacy of drugs and allergen immunotherapy in reducing nasal irritability.

Nasal cytology, ciliary functional studies, and biopsy

43. Nasal smears for eosinophils are not necessary for routine use in diagnosing allergic rhinitis when the diagnosis is clearly supported by the history, physical examination, and specific IgE diagnostic studies but may be a useful adjunct when the diagnosis of allergic rhinitis is in question.

44. Although the saccharin test for mucociliary clearance has been relied on as a clinical screening test, it cannot be relied on for a definitive diagnosis of mucociliary dysfunction.

45. Nasal biopsy may be indicated when determining whether a lesion is neoplastic or granulomatous or if there is an abnormality in the ultrastructure of cilia.

Nasal smears for eosinophils are usually considered elevated when 10% of cells are eosinophils. The presence of eosinophils has been associated with loss of epithelial integrity in patients with both allergic and nonallergic rhinitis. When taking nasal smears, both nostrils should be sampled because the findings from 1 nostril are not reliably representative of the total cell distribution in both nostrils. Samples collected by blowing mucus into transparent wrap contain less cellular material than when a cytolysis probe, or ultrasonic nebulization of hypertonic saline are used, but are adequate for detecting eosinophils and neutrophils. Once collected, the nasal secretions are transferred to a slide, fixed, and then treated with Hansel stain, which highlights eosinophil granular contents. Although nasal smears are generally adequate for assessment of nasal eosinophilia, there is some evidence that nasal biopsy for eosinophils is more accurate. Many studies have shown a high correlation of nasal eosinophilia and allergic rhinitis; however, it is questionable how much this benefit the clinician in making the diagnosis of allergic rhinitis. In a study of adolescents and adults, adding nasal smears for eosinophils to an expert’s clinical evaluation in establishing a diagnosis of allergic rhinitis contributed very little to the final diagnosis and was considered clinically irrelevant. When eosinophils in nasal smears are present, there is only a 71% correlation with SPTs and a 69% correlation with nasal challenge tests, suggesting that routine use of the nasal smear may not be beneficial. In clinical trials, nasal eosinophils have been used to evaluate anti-inflammatory effects of intranasal corticosteroids and may be associated with improved nasal symptom scores. As many as 4.7% of patients with a history very suggestive of allergy will have negative prick tests but a positive nasal challenge and a positive nasal smear for eosinophils, and may have nasal specific IgE, supporting the diagnosis of allergic rhinitis. Under special circumstances, when faced with a convincing history, negative SPT, and elevated nasal eosinophils, the clinician may wish to conduct a nasal or conjunctival challenge test. On the other hand, as many as 6% of patients with a similar history suggestive of allergy will have negative prick skin test and nasal challenge but will have nasal eosinophilia, establishing the diagnosis of nonallergic rhinitis with eosinophilia. When both SPTs and nasal smears for eosinophils are negative in patients with rhinitis, a less favorable response to medical therapy can be anticipated.

Additional research is needed to establish whether nasal smears for eosinophils are useful for predicting the onset, course, and progression of allergic disease. In 1 pediatric study, nasal eosinophilia was found to predict prolonged or subsequent allergic rhinitis symptoms. In addition, the nasal eosinophil count has been shown to correlate with the severity of perennial allergic rhinitis in children. Recognizing that many patients with allergic rhinitis have increased airway reactivity and will subsequently develop asthma, the nasal smear for eosinophils may help predict the progression of disease; in adult patients with allergic rhinitis as the only allergic diagnosis, it has been shown that the number of nasal eosinophils correlates with the methacholine response. The absence of eosinophils and the presence of large numbers of polymorphonuclear neutrophils especially when intracellular bacteria are noted suggest an infectious rhinitis or sinusitis.
basis of sinus aspirates, the number of polymorphonuclear cells varies with the infecting organism, because *H. influenzae* is associated with significantly fewer leukocytes than *Streptococcus pyogenes.* A viral infection is usually associated with fewer polymorphonuclear cells than a bacterial process. However, the mere presence of neutrophilia cannot be diagnostic of an infectious process because as many as 79% of school children and 97% of infants have neutrophils in their nasal secretions.

The saccharin test (with saccharine or a substitute dye marker) may be used by the clinician as a screening test for primary or secondary ciliary dysfunction. The test requires a cooperative subject but is quite noninvasive. The saccharin test involves placement of a 1-mm to 2-mm particle of saccharin on the inferior nasal turbinate 1 cm from the anterior border. The subject then sits with the head bent forward and the test is completed when either the patient tastes the saccharin or the clinician visualizes the presence of the dye marker in the posterior pharynx. If the time is beyond 1 hour or the subject is unable to taste the saccharin or detect the dye, mucociliary clearance is considered impaired. When an abnormal study is obtained, additional studies are required before a firm diagnosis can be established, because the saccharin test has too many false-positives and false-negatives.

Nasal biopsies are used to determine if a suspicious lesion is neoplastic or granulomatous and to evaluate suspected ciliary dysfunction. Biopsies to determine ciliary structure and function can be obtained endoscopically from bronchi or the nasopharynx and by curette or brush from the inferior concha. The cilia can be viewed by video or examined in cross-section by electron microscopy for specific defects or usual ultrastructure. Combining electron microscopy with computer-based image processing algorithms can improve the visualization of ultrastructural defects. Depending on the results of the nasal biopsy evaluation of ciliary function, a tracheal biopsy may be required for confirmation of ciliary dyskinesia. Electrophoresis, genetic analysis using a dynein gene probe, and decreased exhaled nitric oxide are additional diagnostic procedures under investigation for the evaluation of ciliary function.

**Additional laboratory testing**

46. The measurement of total IgE and IgG subclasses for the diagnosis of allergic rhinitis has limited value and should not be routinely performed. C

47. The presence of β-2-transferrin in the nasal secretions is a sensitive method of confirming cerebral spinal fluid rhinorrhea. B

Measurement of total IgE in cord blood or in children has been proposed as means of predicting the risk of allergic disease; however, recent studies have found that cord blood is not a reliable predictor for atopic disease. The total serum IgE has low sensitivity (43.9%) as well as low positive and negative predictive values when evaluating a patient for allergic rhinitis and therefore is of limited clinical benefit.

In recent research, the presence of specific immunoglobulin responses of the IgG subclasses has been suggested to be a risk factor for allergic disease. Furthermore, measurement of nonspecific and specific IgG4 and/or of other subclasses has been advocated as a diagnostic test for clinical allergy. In general, scientific evidence supporting its use has been controversial and inconclusive. The routine measurement of IgG4 should not be part of the diagnosis evaluation of patients with allergic nasal disease.

**Special testing considerations in children**

48. In children with rhinitis, the use of immune studies, sweat test, sinus CT, and nasal endoscopy may be indicated when they are suspected to have comorbid conditions such as immune deficiency, CF, and chronic sinustis. C

In children with rhinitis, select tests that may be indicated on an individual basis include quantitative immunoglobulins, complement studies, ciliary functional and morphologic studies (as described in Summary Statements 44, 45), and the sweat test and/or genetic typing for CF when disorders such as immunodeficiency, ciliary dyskinesia, and CF are suspected (see “Allergy Diagnostic Testing: An Updated Practice Parameter” and “Practice Parameter for the Diagnosis and Management of Primary Immunodeficiency”).

There is ongoing controversy on the usefulness of plain sinus radiography in children for the diagnosis of acute bacterial sinusitis. Although the diagnosis of acute bacterial sinusitis in children can usually be made by clinical assessment, plain radiography may be considered for confirmation in children 6 years and older with persistent symptoms and for all children (regardless of age) with severe or worsening symptoms. In children, as in adults, CT scans of the sinuses are more sensitive than plain radiography and should be considered for potential complications of acute bacterial sinusitis, such as orbital or intracranial complications, for patients who fail to improve with appropriate medical therapy, or for the diagnosis of chronic sinusitis. A lateral nasopharyngeal radiograph may help to exclude adenoid hypertrophy in children with mouth breathing, snoring, sleep apneic episodes, and nasal obstruction. When available, dynamic video rhinoscopy is more accurate at assessing adenoid hypertrophy and percent airway occlusion than lateral neck radiography. Overnight polysomnography may be necessary to confirm the diagnosis of OSAS, noted in 1% to 3% of children, before surgical removal of enlarged adenoids and tonsils.

**Testing for comorbid conditions**

49. A formal evaluation for obstructive sleep apnea may be considered in children and adults presenting with chronic rhinitis and other risk factors associated with sleep-disordered breathing.

Atopy has been associated with habitual snoring in infants. In children, the presence of rhinitis is a strong predictor of habitual snoring. Children who are African American, have upper respiratory disease, and have a family history of sleep apnea are at enhanced risk for sleep-disordered breathing. Thus, formal evaluation for OSAS may be considered in children presenting with chronic rhinitis and other risk factors associated with sleep-disordered breathing.

In snoring adults with rhinitis and sleep apneic symptoms, increased nasal airway resistance has been associated with apnea and hypopnea. Obstructive sleep apnea episodes determined by polysomnography were more frequent in patients with ragweed allergy during symptomatic periods when nasal resistance was increased than during asymptomatic periods. Intranasal corticosteroids reduce nasal airway resistance and apnea-hypopnea frequency in patients with OSAS and rhinitis and may be of benefit in the management of some patients with OSAS.

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50. Pulmonary function tests should be considered in patients with rhinitis to assess the possibility that asthma might be present. D

Rhinitis and asthma are linked by common epidemiologic, physiologic, and pathologic mechanisms, as well as common comorbidities and therapeutic approaches, leading to the concept of “one airway, one disease.”262-265 This concept has been popularized by a publication of the ARIA workshop group, where it is stated in the introduction, “…patients with persistent allergic rhinitis should therefore be evaluated for asthma and patients with asthma should be evaluated for rhinitis.”11 Thus, pulmonary function tests should be considered in patients with rhinitis to assess the possibility that asthma might be present. The presence of asthma may not be apparent because such patients (1) may have difficulty in recognizing their symptoms, (2) may have variable symptoms throughout the day, (3) may have a physical examination of the respiratory system that appears normal, and (4) may present with symptoms that are atypical. Furthermore, the physical examination of the lower respiratory systems may be normal during the medical evaluation of rhinitis.

Unproven tests
51. There is no evidence that the following procedures have diagnostic validity for allergic rhinitis: cytotoxic tests, provocation-neutralization, electrodermal testing, applied kinesiology, iridology, and hair analysis. B (see “Allergy Diagnostic Testing: An Updated Practice Parameter”9)

Management of rhinitis
Environmental control measures
Environmental triggers for rhinitis can be divided into 2 general categories: allergens and irritants. Ideally, the management of rhinitis includes identification of these triggers when possible and implementation of avoidance measures when practical.

52. The most common allergic triggers for rhinitis include pollen, fungi, dust mites, furry animals, and insect emanations. B

Allergens are substances that trigger rhinitis through an IgE-dependent mechanism. The most common allergic triggers for rhinitis include pollens, fungi, dust mites, furry animals, and insect emanations. The ideal way for patients to manage allergic rhinitis is with complete avoidance of all relevant allergens. Because this generally is not possible, patients should be counseled to reduce their exposure to as many relevant allergens as is practical. This may improve their ability to tolerate exposure to unavoidable aeroallergens. With the development of sensitive immunochemical techniques, direct measurement of indoor allergen concentrations to confirm that exposure reduction has occurred is now possible.293 Even so, the effectiveness of environmental control measures should be judged primarily by clinical parameters such as reduction in patient symptoms and medication scores.266

53. The types of pollen responsible for rhinitis symptoms vary widely with locale, climate, and introduced plantings. B

54. Highly pollen-allergic individuals should limit exposure to the outdoors when high pollen counts are present. B

Pollens that trigger allergic rhinitis principally comes from plants that are wind-pollinated (anemophilous). This includes many trees, grasses, and weeds, although exposure to pollen from insect-pollinated (entomophilous) plants may produce symptoms if sufficient concentrations are encountered.872 Allergens are quickly eluted from pollen grains on contact with ocular or respiratory mucosa. In addition, similar allergens may be found on fragments derived from other portions of the plant. Pollen allergens, forming an allergenic bio aerosol, can be detected in the outdoor environment even when intact pollen grains are not present.267-269

The types of pollen responsible for symptoms vary widely with locale, climate, and introduced plantings.873,874 In temperate regions of North America, tree pollen generally predominates in early to mid-spring, grasses in late spring and early summer, and weeds from late summer until early fall.575 The dose of pollen allergen that is able to elicit symptoms depends on the degree of allergic sensitization and on nasal mucosal inflammation that already is present, often referred to as “priming.”721,876,877

Indoor pollen exposure increases when windows are open or attic fans are in use during pollen season. Therefore, to reduce indoor exposure, windows and doors should be kept closed. If air conditioning is used to keep the home or vehicle comfortable, any outdoor vents should remain closed.878 It may be helpful to take a shower or bath following outdoor activity, thereby reducing indoor pollen contamination. Because pets also can be vectors for pollen intrusion, it may be beneficial to wash furry animals after they have been outdoors. Although it is not practical to remain indoors all of the time, it is helpful to limit outdoor exposure during periods with high pollen counts.879 For example, because ragweed pollen concentrations tend to peak at noon or in the early afternoon, it may help to plan outdoor activities for the early morning or late evening.880 Highly sensitive patients whose symptoms are triggered by very low pollen levels may need to limit their outdoor activities.277 Having such individuals wear a facemask during outdoor activities should be considered.277 Activities involving extended time outdoors, such as camping trips, may need to be avoided during certain pollen seasons. Because pollen counts tend to be higher on sunny, windy days with low humidity, it may help to limit outdoor activities when those weather conditions are present. In contrast, outdoor activities may be well tolerated after a gentle, sustained rain when pollen counts tend to be low. Because the interplay of different weather factors (eg, wind, temperature, rain, humidity) is complex, it is not reliably possible to predict levels of outdoor aeroallergens on the basis of the influence of a single weather factor.276-272

55. Fungi are ubiquitous organisms, many of which produce clinically important allergens. B

56. Reduction of indoor fungal exposure involves removal of moisture sources, replacement of contamination materials, and the use of dilute bleach solutions on nonporous surfaces. D

Fungi are ubiquitous organisms, many of which produce clinically important allergens.881-886 They exist in great numbers outdoors but also may contaminate indoor environments. Most fungal allergens are encountered through inhalation of spores, although fragments of hyphae also may be important. Fungi are present in the air throughout the year except during periods of snow cover. In the Northern United States, spore concentrations tend to increase in late spring and again during late summer, whereas in the southern United States, they tend to remain elevated year-round. Most fungi are found in soil and tend to release spores...
when the earth is disturbed (e.g., during plowing, excavation, and so forth). Outdoor fungi grow on both viable and decaying vegetation and are particularly abundant during harvesting activities in agricultural regions.

Because there is a temporal association between exposure to spores and development of allergic symptoms in sensitive individuals, it is important for patients to be aware of conditions that are associated with elevated spore concentrations. Spore concentrations are strongly influenced by local conditions such as moisture, temperature, wind, rain, and humidity. Some fungi require moist conditions for spore release, leading to elevated levels of these spores during rainy weather and with dew formation at night. Such hydrophilic fungi include *Fusarium*, *Phoma*, most ascospores, and basidiospores (mushrooms). Other common allergenic fungi, such as *Alternaria* and *Cladosporium*, are more abundant during dry, windy weather. Rain or high humidity can induce spore release, particularly when the rainy period ends. Thunderstorms have even been associated with spore plumes.

Avoidance of outdoor fungi may require the patient to remain indoors as much as is practical. As with pollen avoidance, air conditioning units, if necessary for comfort, should be used with the outdoor vents closed. Because air conditioning units at home, at work, and in automobiles may become contaminated with fungi, they should be inspected regularly and maintained according to the manufacturer’s instructions. Other situations associated with increased fungus exposure include plant-disturbing activities such as mowing, threshing, or raking leaves and proximity to compost, silage, or dry soil. Use of face masks may be helpful for reducing exposure during such outdoor activities.

Many factors influence growth of indoor fungi, including the age and construction of the residence; the presence of a basement or crawl space; the type of heating, ventilation, and air conditioning system; and the use of humidifiers. Fungi require oxygen, a source of carbohydrate, and moisture for optimal growth. Moisture becomes available to fungi through entry into the building by intrusion from outside, leakage from pipes, or condensation onto cold surfaces with temperatures that are below the dew point of air. Therefore, environments most likely to become contaminated with fungi include homes with elevated humidity (above 50%), basements with water intrusion, and cold surfaces. Other locations often providing favorable conditions for fungal growth include window moldings, sinks, shower stalls, nonrefrigerated vegetable storage areas, and garbage pails.

Reduction of fungal exposure consists of eliminating the source of moisture. Fungicides may kill or at least retard fungal growth. Products containing a dilute bleach solution with a detergent have been shown to denature fungal allergens and are preferred. Homes constructed with a crawl space should have a plastic vapor barrier over exposed soil, and foundation vents should be kept open to provide ventilation. Spores are often present in carpets, bedding, and upholstered furniture and are reduced by the same measures used for dust mite avoidance. Carpets, bedding, and upholstered furnishings therefore should be avoided in damp areas or in locations that tend to flood. A dehumidifier should be used and standing water removed as quickly as possible in such locations.

57. Clinically effective dust mite avoidance requires a combination of humidity control, dust mite covers for bedding, HEPA vacuuming of carpeting, and the use of acaricides. B

A major source of allergen in house dust is the fecal residue of dust mites belonging to the genus *Dermatophagoides*. A principal food source of dust mites consists of exfoliated human skin cells. Consequently, mites are most abundant in locations where skin cells are shed such as bedding, fabric covered furniture, soft toys, and carpeting. In addition to the availability of this food source, the other major factors influencing mite growth are temperature and humidity. To reproduce, dust mites generally require a relative humidity of 50% or greater. Recent changes in home construction and housecleaning methods have created environments conducive to dust mite proliferation. These include enhanced energy efficiency in buildings leading to reduced ventilation and increased humidity, wall-to-wall carpeting, furnished basements, and use of water for laundry that is not hot enough to kill mites.

To reduce mite allergen exposure, humidity should be maintained between 35% and 50%, reservoirs in which they reside should be minimized, and barriers should be created between the mites and the building’s occupants. Humidity can be reduced with air conditioning or a dehumidifier and can easily be measured with an inexpensive hygrometer. Common reservoirs to be avoided include upholstered furniture, carpeting, bedding, and stuffed toys. Dust mite fecal pellets easily become airborne when their reservoir is disturbed, although they rapidly settle once the disturbance stops. Ordinary vacuuming and dusting therefore have little effect on mite allergen concentrations because the mites are not removed and the pellets easily pass through low-efficiency vacuum bags, becoming widely dispersed throughout the room. Carpeting therefore is best removed from the bedroom and replaced with smooth finish wood, tile, or vinyl flooring. If this is impractical, one may consider treating carpets with an acaricide (benzyl benzoate) that kills mites. Carpets installed over a concrete slab will inevitably become contaminated with both mites and fungi because of condensation and is best avoided if possible. If carpeting is present, vacuum cleaners with HEPA filtration or central vacuums that remove the air to a distant location should be used. Ideally, housecleaning should be performed when the allergic person is not at home, although patients who do their own cleaning may benefit from wearing a face mask.

To create an effective barrier to exposure, mattresses, box springs, and pillows in the patient’s bedroom should be encased in zippered, allergen-proof encasings. Vinyl encasings are effective, although cloth encasings with semipermeable plastic backing are more comfortable and durable. If a mattress is old, replacement should be considered, but even new hypoallergenic mattresses and pillows should be encased because mite colonization occurs within weeks. Unfortunately, when impermeable
bedding is used as an isolated intervention, it is unlikely to offer clinical benefit. Bedding should be washed in hot water (greater than 130°F) at least every 2 weeks to remove mite allergen and to kill mite ova, although lower temperatures will remove the mite allergen itself. Quilts and comforters should be avoided or covered with an allergen-proof duvet.

Because elimination of mites in upholstered furniture is extremely difficult, plastic, leather or wood furniture is recommended. When upholstered furniture cannot be avoided, a 3% tannic acid solution may be used to denature mite and other allergens on these furnishings. Because this does not kill the mites, the allergen reaccumulates over time, necessitating repeated treatments. Stuffed toys that cannot be washed can be placed in plastic bags and frozen to kill dust mites.

There is increasing evidence that HEPA air filtration is effective for reducing dust mite exposure. On the other hand, duct cleaning has not been demonstrated to be of significant benefit.

Avoidance is the most effective way to manage animal sensitivity. D

Because of the popularity of indoor pets, allergens from cats, dogs, and other domestic animals are important triggers of allergic rhinitis. All warm-blooded animals, including birds, potentially are capable of sensitizing susceptible patients with allergy. Animal allergens are a significant occupational hazard for workers exposed to mice, rats, guinea pigs, and so forth. Farm workers may develop sensitivities to farm animals. In inner city areas, rodent urine may be an important source of animal allergen. Although furs processed for use in clothing are no longer allergenic, feather products retain significant allergenicity. Because allergen-bearing particles of animal origin are generally quite small and low-density, they remain suspended in air for extended periods and disseminate widely in homes and other facilities. Symptoms of allergic rhinoconjunctivitis may occur within minutes of entering a contaminated area.

The major antigen in cat allergen, Fel d 1, is found on cat skin/dander and in saliva and urine. Cat albumin is also allergenic but is a less frequent cause of sensitivity than Fel d 1. Fel d 1 and albumin are common to all breeds of cats. Cat allergen has been identified in homes and other locations where cats were never present and occasionally may reach concentrations found in homes where cats are kept. This is presumed to be passive contamination from cat allergen borne on clothing. Such contamination may be an unsuspected cause of symptoms in sensitive individuals. Allergy to dogs appears to be less frequent than cat allergy. The major dog allergen, Can f 1, is found in dog skin/dander and saliva and is present in varying amounts in all breeds tested. Many dog-sensitive patients claim to respond differently to various breeds of dogs or even specific dogs of a single breed. Like cat allergen, Can f 1 has been found in rooms in which dogs were never present, suggesting passive transport on clothing. Concentrations may be sufficient to elicit symptoms in sensitized patients. Avoidance is the most effective way to manage animal sensitivity. Patients and their families should be advised to consider removing an animal to reduce exposure. A trial removal of a pet for a few days or even weeks may be of little value or, worse, misleading, because cat allergen can be detected an average of 20 weeks (and in some cases much longer) before reaching concentrations found in homes without cats. Steam cleaning of carpets and upholstered furniture after removal of the animal seems to have little advantage over routine vacuuming with a HEPA filter vacuum system. It also helps to wash the bedding with soap and water. If the patient and/or family decide not to remove the pet, confining the animal to an uncarpeted room (other than the bedroom) containing a HEPA or electrostatic air purifier may reduce airborne allergen in the remainder of the home by 90%. In general, measures used to reduce exposure to dust mite work to some extent for cat allergen as well. Some but not all studies have demonstrated reduced airborne cat allergen by washing the animal on a weekly basis. Frequent bathing of dogs (at least twice a week) similarly has been found to be effective for reducing dog allergen exposure. Litter boxes should be placed in an area separated from the air supply to the rest of the home to avoid dispersal of allergen. If not removed, caged pets (birds, rodents, guinea pigs, and so forth) also should be kept in an uncared for area of the home and remote from the patient’s bedroom.

59. Cockroaches are a significant cause of nasal allergy, particularly in inner-city populations. C

Allergic rhinitis and asthma have been reported after exposure to debris of numerous insects including cockroaches, crickets, caddis flies, houseflies, midges, spider mites, mosquitoes, ladybugs, and moths. In endemic areas, such as West Virginia, ladybug is a major allergen causing rhinoconjunctivitis at a prevalence rate of as high as 8%. Ladybug skin test sensitization is comparable in frequency and age distribution with cat and cockroach in endemic areas.

Because of their prevalence and indoor living habits, cockroaches are a significant cause of respiratory allergy, especially in inner-city populations. As many as 60% of dust-sensitive patients from urban areas react to cockroach allergens. The major cockroach allergens, Bla g I and Bla g II, are found on the insect’s body and its feces. Cockroach allergen is most abundant in kitchen floor dust and may reach high levels in poorly maintained homes and apartments. Cockroach elimination requires careful sanitation such as not allowing food to stand open or remain on unwashed dishes, promptly wiping up food spills, and storing garbage in tightly closed containers. Use of roach traps, such as odorless and colorless gel baits containing hydramethylnon or abamectin, has been advocated because these permit removal of the allergen-containing bodies of the insects. If the infestation is heavy, repeated applications of insecticide by a professional exterminator or changing homes may be required.

60. The best treatment for rhinitis triggered by irritants, such as tobacco smoke and formaldehyde, is avoidance. B

An irritant is defined by the Occupational Health and Safety Administration as “a noncorrosive chemical which causes a reversible inflammatory effect on living tissue by chemical action at the site of contact.” A more general definition is that an irritant is a substance that, on immediate, prolonged, or repeated contact with normal living tissue, will induce a local inflammatory reaction. The amount of inflammation associated with irritants depends on their degree of irritation, the duration of exposure, and the sensitivity of the target organ. The effect of irritants is temporary. Pepper spray, for example, is a severe eye and nose irritant, although it causes no lasting effects. People with asthma tend to be more sensitive to the irritant effects of airborne substances such as perfumes, ozone, and smoke than those with normal lung function. It should be noted that the amount of exposure to a substance...
that is capable of causing an irritant reaction often is orders of magnitude less than the amount causing organ toxicity.

Because many substances in buildings are volatile and potentially irritating, it often is difficult to determine the source of a particular inflammatory reaction. To determine whether an irritant is responsible for a symptom, it is necessary to demonstrate that the substance is present in the environment, that exposure is sufficient in magnitude and duration to trigger the observed reaction in the affected individual, and that other substances that could account for the symptoms are not present.

Fungi produce a number of potentially irritating substances. These include microbially derived volatile organic compounds (MVOCs), glucans that are related to endotoxins, and ergosterols. However, the full spectrum of bacterial and fungal irritants has not been fully enumerated. As with other irritants, the health effects of exposure to these substances are directly related to the amount and duration of exposure. Thus, buildings with good ventilation are considered healthier than those in which airborne, potentially respirable irritants can accumulate.

Rhinitis has been attributed to irritants such as tobacco smoke, formaldehyde, perfume and other strong odors, and even newspaper ink. Environmental tobacco smoke is a significant irritant as well as a potentially toxic substance. Because rhinitis symptoms that occur in response to tobacco smoke exposure do not involve IgE, avoidance of passive tobacco smoke is the best treatment. Formaldehyde is known to cause stinging and burning of the eyes and nose, lacrimation, and decreased nasal mucus flow. This appears also to be an irritant effect because even prolonged, high-level formaldehyde exposure rarely results in development of IgE to formaldehyde-protein conjugates, and this does not correlate with clinical symptoms. Because respiratory symptoms generally occur at concentrations well above those at which the odor of formaldehyde is detectable, it is unlikely that formaldehyde would be an unsuspected cause of rhinitis. Some patients with rhinitis claim that exposure to perfume and newsprint can elicit symptoms. The mechanism for this is uncertain but is likely to be an irritant reaction also.

**Pharmacologic therapy**

The selection of pharmacotherapy for a patient depends on multiple factors including the type of rhinitis present (eg, allergic, nonallergic, mixed, episodic), most prominent symptoms, severity, and patient age (Summary Statements 92, 93). Principal medication options are summarized in Table VI. The following sections of the parameter provide detailed discussion of medication options.

**Oral antihistamines**

61. Second-generation antihistamines are generally preferred over first-generation antihistamines for the treatment of allergic rhinitis. First-generation antihistamines have significant potential to cause sedation, performance impairment, and anticholinergic effects. Although occasionally advantageous (eg, sleep induction when taken at bedtime or a reduction in rhinorrhea), these properties are usually undesirable and are potentially dangerous. Second-generation antihistamines have less or no tendency to cause these effects. B

62. Before prescribing or recommending a first-generation antihistamine, the physician should ensure that the patient understands both the potential for adverse effects and the availability of alternative antihistamines with a lower likelihood of adverse effects. D

First-generation antihistamines such as diphenhydramine, hydroxyzine, and clemastine are associated with sedative effects—drowsiness and/or performance impairment—in many patients. Interindividual variation exists with respect to development of sedative effects with either single-dose or regular use of these agents. Although patients may deny sedation with first-generation antihistamines, performance impairment can exist without subjective awareness of drowsiness. Although there are conflicting data, first-generation antihistamines have also been associated with impaired learning and school performance in children, as well as driving impairment and fatal automobile accidents in adolescents and adults. A large epidemiologic study found that drivers responsible for fatal automobile accidents were 1.5 times more likely to be taking first-generation antihistamines than drivers killed but not responsible for accidents. Workers taking first-generation antihistamines may exhibit impaired work performance and productivity; they are more likely to be involved in occupational accidents. Concomitant use of other CNS-active substances, such as alcohol, sedatives, hypnotics, or antidepressant medication, may further enhance performance impairment from antihistamines. A recent report found that impaired driving performance associated with hydroxyzine worsened with cellular phone use. Paradoxical CNS stimulation may also occur with use of first-generation antihistamines, particularly in children.

In a strategy intended to reduce costs of antihistamine therapy while avoiding daytime drowsiness and performance impairment, administration of a non-sedating second-generation antihistamine (that would otherwise be dosed twice daily) only once daily in the morning, followed by a first-generation (and less costly) antihistamine in the evening, has been advocated. However, first-generation antihistamines dosed only at bedtime can be associated with significant daytime drowsiness, decreased alertness, and performance impairment. In part this is because antihistamines and their metabolites have prolonged plasma half-lives, and their end-organ effects persist longer than plasma levels of the parent compound (Table VII). Consequently, an AM/PM dosing regimen, combining a second-generation agent in the AM with a first-generation agent in the PM, is not a preferred strategy for avoiding daytime drowsiness and performance impairment from antihistamine treatment in the management of allergic rhinitis.

Anticholinergic effects can also occur with first-generation antihistamines, including dryness of mouth and eyes, constipation, inhibition of micturition, and an increased risk for provocation of narrow angle glaucoma. Anticholinergic effects may also be desirable in some patients (eg, those with persistent rhinorrhea despite a second-generation antihistamine and an intranasal corticosteroid). However, a topical anticholinergic agent approved for allergic rhinitis without the potential for sedation or performance impairment would generally be preferred over a systemic agent with anticholinergic properties.

Older adults are more sensitive to the psychomotor impairment promoted by first-generation antihistamines, and are at increased risk for complications such as fractures and subdural hematomas caused by falls, and are more susceptible to adverse anticholinergic effects. Because of concomitant comorbid conditions (eg, increased IOP, benign prostatic hypertrophy, preexisting cognitive
impairment, and so forth) that may increase the risk associated with regular or even intermittent use, extra caution should be used when considering the use of first-generation antihistamines in older adults. In comparative studies, the second-generation antihistamines have been associated with less or no tendency for impairment of performance, drowsiness (Table VII), or anticholinergic effects.\textsuperscript{307-311} For this reason, the second-generation antihistamines are generally preferred for the treatment of allergic rhinitis. First-generation antihistamines may be prescribed at bedtime when a soporific effect is desired (with the caveat noted that performance impairment can exist the next morning without subjective awareness of drowsiness), and/or it is viewed as advantageous to administer an antihistamine with anticholinergic properties.

There are important differences among the second-generation antihistamines in regards to their sedative properties: fexofenadine, loratadine, and desloratadine do not cause sedation at recommended doses; loratadine and desloratadine may cause sedation at doses exceeding the recommended dose; cetirizine and intranasal azelastine may cause sedation at recommended doses.\textsuperscript{63} Among the newer, non-sedating antihistamines, no single agent has been conclusively found to achieve superior overall response rates.\textsuperscript{C}

The absence of sedative properties among the second-generation agents is not uniform. In multiple studies, the use of fexofenadine, loratadine, and desloratadine when used at recommended doses for the treatment of allergic rhinitis has not been associated with sedative properties compared with placebo.\textsuperscript{307,309} Even at higher than FDA-approved doses, fexofenadine has no sedative properties when used for the treatment of allergic rhinitis.\textsuperscript{308,913} Loratadine and desloratadine have sedative properties when dosed at higher than recommended doses,\textsuperscript{323,328} or at recommended doses in certain individuals. Patients with low body mass for whom a standard dose (based on age) is prescribed may conceivably reach an elevated dosage level (on a milligram per kilogram basis), and thereby develop drowsiness and/or performance impairment.\textsuperscript{307,309} Use of cetirizine or intranasal azelastine has been associated with sedative properties compared with placebo.\textsuperscript{309} However, in many but not all cases, the effect tends to be milder than that observed with first-generation antihistamines.\textsuperscript{307} Nonetheless, patients given these drugs for allergic rhinitis should be cautioned regarding this risk. Cetirizine 10 mg may be associated with mild drowsiness (13.7% for patients \( \geq \) age 12 years) compared with placebo (6.3%)\textsuperscript{329} but without performance impairment. Development of drowsiness without performance impairment has been observed with both cetirizine 10 mg (the standard dose)\textsuperscript{314} and 20 mg.\textsuperscript{330} However, in other studies, the 10 mg or higher dose of cetirizine, was associated with performance impairment.\textsuperscript{307}

Among the newer, non-sedating antihistamines, no single agent has been conclusively shown to have superior efficacy.\textsuperscript{326,327} A recent meta-analysis found ebastine (an agent not available in the United States) superior to loratadine for the decrease in mean rhinitis symptom scores in seasonal allergic rhinitis.\textsuperscript{915} Several studies have found cetirizine to be superior to loratadine, although in 1 study, the differences were not statistically significant.\textsuperscript{915} In a study of patients with seasonal allergic rhinitis who remained symptomatic after treatment with fexofenadine, azelastine significantly improved total nasal symptom score.\textsuperscript{335}

The availability of second-generation antihistamines has substantially improved the therapeutic utility of antihistamines, because patients such as older adults who otherwise would avoid antihistamine therapy as a result of sedation or anticholinergic effects can be given antihistamine medications that are favorable for allergic rhinitis management from a risk/benefit standpoint.\textsuperscript{A}

**Intranasal antihistamines**

65. Intranasal antihistamines may be considered for use as first-line treatment for allergic and nonallergic rhinitis.\textsuperscript{A}

66. Intranasal antihistamines are efficacious and equal to or superior to oral second-generation antihistamines for treatment of seasonal allergic rhinitis.\textsuperscript{A}

67. Because systemic absorption occurs, currently available intranasal antihistamines have been associated with sedation and can inhibit skin test reactions.\textsuperscript{A}

68. Intranasal antihistamines have been associated with a clinically significant effect on nasal congestion.\textsuperscript{A}

69. Intranasal antihistamines are generally less effective than intranasal corticosteroids for treatment of allergic rhinitis.\textsuperscript{A}

Azelastine and olopatadine are the only intranasal antihistamines currently available in the United States, are approved for the treatment of seasonal allergic rhinitis, and have been shown to improve congestion, rhinorrhea, sneezing, and nasal pruritus.\textsuperscript{334,335} These agents may be considered as a first-line treatment for allergic rhinitis or as part of combination therapy with intranasal corticosteroids\textsuperscript{339} or oral antihistamines. Intranasal azelastine has been demonstrated to be efficacious for nonallergic rhinitis.\textsuperscript{916} Several studies have demonstrated that their efficacy for seasonal allergic rhinitis is superior\textsuperscript{313-315} to placebo, or equal to\textsuperscript{312} oral second-generation antihistamines. A systematic review of 9 randomized controlled studies comparing intranasal antihistamines with intranasal corticosteroids\textsuperscript{46} concluded that intranasal corticosteroids are more effective for controlling symptoms of perennial allergic rhinitis and seasonal allergic rhinitis. For mixed rhinitis, there may be significant benefit to the combination of an intranasal antihistamine with an intranasal corticosteroid.

Asterol (azelastine hydrochloride; Meda Pharmaceuticals, Somerset, NJ)\textsuperscript{32-338} is formulated as a 0.1% aqueous solution and Patanase (olopatadine hydrochloride; Alcon Laboratories, Fort Worth, Tex) is formulated as a 0.6% aqueous solution, both in a metered spray delivery device. Recommended dosing is 2 sprays in each nostril twice daily for patients \( \geq 12 \) years of age. Clinically significant onset of action of nasal azelastine has been reported at 15 minutes.\textsuperscript{341} The onset of action of nasal olopatadine has been reported at 30 minutes after dosing in an environmental challenge unit. However, head-to-head comparisons of azelastine and olopatadine have not been performed. In clinical trials of nasal azelastine, 19.7% of patients complain of bitter taste, and 11.5% report somnolence.\textsuperscript{342} In clinical trials of nasal olopatadine, 12.8% of patients complain of bitter taste, and 0.9% report somnolence.\textsuperscript{917} In contrast with oral second-generation antihistamines, intranasal azelastine and olopatadine have been associated with clinically significant reduction in nasal congestion.\textsuperscript{336-338,340} Because intranasal antihistamines are absorbed via the gastrointestinal tract, they can suppress skin test response for at least 48 hours in the case of azelastine.\textsuperscript{657}

**Oral and topical decongestants**

70. Oral decongestants, such as pseudoephedrine and phenylephrine, are \( \alpha \)-adrenergic agonists that can reduce nasal
congestion but can result in side effects such as insomnia, irritability, and palpitations. A

71. Oral and topical decongestants should be used with caution in older adults and young children, and in patients of any age who have a history of cardiac arrhythmia, angina pectoris, cerebrovascular disease, hypertension, bladder neck obstruction, glaucoma, or hyperthyroidism. C

Oral α-adrenergic agents relieve nasal congestion by acting as vasoconstrictors. These drugs may be useful in the management of allergic rhinitis and nonallergic rhinitis, including relief of nasal congestion caused by upper respiratory infections. The efficacy of an oral decongestant in combination with an antihistamine in the management of allergic rhinitis has not been adequately documented to increase the efficacy of either drug alone.344

Adverse effects from oral α-adrenergic agents may include elevated blood pressure, palpitations, loss of appetite, irritability, tremor, and sleep disturbance.345 Concomitant use of caffeine, which at one time was prescribed by physicians as a decongestant,350 may be associated with adverse effects that are additive.

Elevation of blood pressure after taking an oral decongestant is generally observed in hypertensive, but not normotensive, individuals. The effect of these agents on blood pressure was examined in 2 meta-analyses of phenylpropanolamine and pseudoephedrine. The meta-analysis of phenylpropanolamine use examined 33 trials reporting 48 treatment arms with 2165 patients938 and found that exposure to phenylpropanolamine increased systolic blood pressure 5.5 mmHg (95% CI, 3.1-8.0) and diastolic blood pressure 4.1 mmHg (95% CI, 2.2-6.0), with no effect on heart rate. Patients with controlled hypertension were not at greater risk of blood pressure elevation. Eighteen studies included at least 1 treated subject with blood pressure elevation ≥140/90 mmHg, an increase in systolic blood pressure ≥15 mmHg, or an increase in diastolic blood pressure ≥10 mmHg.

A meta-analysis that assessed risk for cardiovascular effects with pseudoephedrine919 found that use of this agent was associated with a small increase in systolic blood pressure (0.99 mmHg; 95% CI, 0.08-1.90) and heart rate (2.83 beats/min; 95% CI, 2.0-3.6), with no effect on diastolic blood pressure (0.63 mmHg; 95% CI, −0.10 to 1.35). Oral decongestants are generally well tolerated by most patients with hypertension. However, based on interindividual variation in response, hypertensive patients should be monitored.

Pseudoephedrine is a key ingredient in making methamphetamine. For this reason, in an effort to reduce illicit production of methamphetamine, restrictions have been placed on the sale of pseudoephedrine in the United States345 such that pseudoephedrine and pseudoephedrine-containing preparations have been taken off drugstore shelves and are maintained behind the counter.345 Phenylephrine remains a nonrestricted decongestant, but is inappropriate for regular daily use because of the risk for the development of rhinitis medicamentosa. C

Topically applied sympathomimetic decongestant α-adrenergic agonists, are catecholamines, such as phenylephrine, or imidazole agents, such as oxymetazoline or xylometazoline. These medications cause nasal vasoconstriction and decreased nasal edema but have no effect on antigen provoked nasal response.350 α-Adrenergic vasoconstrictors reduce nasal obstruction but do not affect itching, sneezing, or nasal secretion. Intranasal decongestants were associated with superior efficacy for nasal decongestion compared with intranasal corticosteroids in a 28-day study.356 However, topical decongestants are not recommended for long-term treatment because of the concerns of the development of rhinitis medicamentosa.

Although generally well tolerated, topical decongestants may cause local stinging or burning, sneezing, and dryness of the nose and throat. Delivery technique should follow the same general recommendations that apply to intranasal corticosteroids (see Summary Statement 80). Intranasal decongestants usually do not cause systemic sympathomimetic symptoms; however, a variety of cerebrovascular adverse events have been reported, including anterior ischemic optic neuropathy,920 stroke,921 branch retinal artery occlusion,922 and “thunderclap” vascular headache.923,924 Caution for use of decongestants during the first trimester is recommended because fetal heart rate changes with administration during pregnancy have been reported. Topical vasoconstrictors should be used with care below age 1 year because of the narrow margin between the therapeutic and toxic dose, which increases the risk for cardiovascular and CNS side effects.11

Topical decongestants are appropriate to use on a short-term basis for nasal congestion associated with acute bacterial or viral infections, exacerbations of allergic rhinitis, and eustachian tube dysfunction. Intermittent use of topical decongestants may be considered, but efficacy and safety of this approach have not been formally studied. Regular use of topical decongestants can lead to rebound nasal congestion with rhinitis medicamentosa.129 Unfortunately, few prospective studies have critically examined rhinitis medicamentosa. Furthermore, its pathophysiology is not fully understood. Topical decongestants cause vasoconstriction, reduce nasal secretion of mucus, and inhibit nasal ciliary action. Initial relief of nasal congestion can be prompt and dramatic; however, rebound congestion may follow as the vasoconstrictive...
Intranasal corticosteroids
74. Intranasal corticosteroids are the most effective medication class in controlling symptoms of allergic rhinitis. A
75. In most studies, intranasal corticosteroids were shown to be more effective than the combined use of an antihistamine and LT antagonist in the treatment of seasonal allergic rhinitis. A
76. Intranasal corticosteroids may provide significant relief of symptoms of seasonal allergic rhinitis when used not only on a regular basis but also when used on an as needed basis. B
77. When comparing the available intranasal corticosteroids, the overall clinical response does not appear to vary significantly between products irrespective of the differences in topical potency, lipid solubility, and binding affinity. C

The main mechanism by which corticosteroids relieve the symptoms of allergic rhinitis is through their anti-inflammatory activity, although it is possible that they may exert an effect through other mechanisms. The concept of delivering corticosteroids locally to the nasal airways was developed to minimize potential side effects of using systemic corticosteroids. Intranasal corticosteroids are available in various formulations (Table VIII). When comparing the available intranasal corticosteroids, the overall clinical response does not appear to vary significantly between products irrespective of the differences in topical potency, lipid solubility, and binding affinity.

Intranasal corticosteroids are effective in controlling the 4 major symptoms of allergic rhinitis: sneezing, itching, rhinorrhea, and nasal congestion. In clinical studies, intranasal corticosteroids have been shown to be more effective than nasal cromolyn sodium or LTRAs. In most studies, intranasal corticosteroids were shown to be more effective than the combined use of an antihistamine and LT antagonist in the treatment of seasonal allergic rhinitis. However, for patients who are unresponsive to or noncompliant with intranasal corticosteroids, combination therapy using an antihistamine in combination with an anti-LT or decongestant may provide a viable alternative. In 2 systematic reviews of randomized controlled studies, intranasal corticosteroids were significantly more effective than oral and intranasal antihistamines in relieving symptoms of sneezing, nasal congestion, discharge, and itching, and were not significantly different for the relief of eye symptoms. However, in 1 study included in these reviews, a nasal antihistamine was more efficacious than intranasal corticosteroids. Although the addition of an oral antihistamine to an intranasal corticosteroid generally has not demonstrated greater clinical benefit than intranasal corticosteroid monotherapy in controlled trials, in well controlled study of seasonal allergic rhinitis, the addition of cetirizine to intranasal fluticasone propionate led to greater relief of pruritus. In another study, the combination of fluticasone propionate and loratadine was superior to fluticasone propionate alone for some patient-rated symptoms. Likewise, 1 study found that at least 50% of patients need to take both intranasal corticosteroids and oral antihistamines to control symptoms of seasonal allergic rhinitis adequately. A study comparing the effectiveness and safety of intranasal corticosteroids and anticholinergic agents has shown that an intranasal corticosteroid is more effective than an anticholinergic agent for all nasal symptoms except rhinorrhea.
Clinical studies have also shown that intranasal fluticasone propionate can provide significant relief of the symptoms of seasonal allergic rhinitis compared with placebo when used on an as-needed basis (which equated to 55% to 62% usage). However, this may not be as effective as continuous use. A well controlled trial of intranasal fluticasone propionate compared with loratadine when used on an as-needed basis for seasonal allergic rhinitis demonstrated significantly better scores for the fluticasone-treated patients in activity, sleep, practical, and overall domains. The onset of therapeutic effect of intranasal corticosteroids seems to occur within 12 hours and as early as 3 to 4 hours in some patients for nasal symptoms. Because a patent nasal airway is necessary for optimal intranasal delivery of intranasal corticosteroids, use of a nasal decongestant spray may be necessary for several days when intranasal corticosteroids are introduced.

78. Intranasal corticosteroids may be useful in the treatment of some forms of nonallergic rhinitis. A

The effectiveness of intranasal corticosteroids has been shown in studies that have involved a large number of patients with nonallergic rhinitis, especially those with NARES. Intranasal corticosteroids have also been shown to be effective in the treatment of vasomotor rhinitis. Intranasal corticosteroids have also been shown to be effective in the treatment of vasomotor rhinitis.

79. Intranasal corticosteroids when given in recommended doses are not generally associated with clinically significant systemic side effects. A

It is unusual for adult patients to develop systemic corticosteroid side effects after administration of intranasal corticosteroids in recommended doses. In children, an effect of intranasal corticosteroids on growth has been demonstrated, as discussed below, although an effect on the HPA axis has not been demonstrated and no reduction in bone density or other systemic effects have been reported.

HPA AXIS

Studies of corticosteroid preparations at recommended and moderate doses given once daily demonstrate minimal systemic corticosteroid effects on the HPA axis, as assessed by morning cortisol concentrations, cosyntropin stimulation, and 24-hour urinary-free cortisol excretion. Studies in children have shown no clinically significant effect of intranasal corticosteroids on the HPA axis. However, the effect of intranasal corticosteroids on growth, recognizing the variability in individual patient response, may be a better indicator of systemic effects in children, and can occur without an effect on the HPA axis. Growth suppression is both a sensitive and relatively specific indicator of excessive corticosteroid effect, compared with measures of basal HPA function that are highly sensitive but have limited value as a predictor of a clinically significant effect. Therefore, there may be a disparity between the effect of intranasal corticosteroids on the HPA axis assessment as an indication of systemic effect and their transient effect on growth in children. The transient effect on growth is dependent on the specific intranasal corticosteroid product, dose, techniques for measuring growth, time of administration, and use of concomitant oral or inhaled corticosteroids.

OCULAR EFFECTS

There have been reports of a possible association between the development of posterior subcapsular cataracts and the use of intranasal or inhaled corticosteroids in older patients, but this association has not been confirmed by other studies with intranasal corticosteroids or studies of intranasal corticosteroids. Concomitant use of systemic corticosteroids in some patients receiving intranasal corticosteroids confounds interpretation of these studies. Studies of intranasal corticosteroids in prospective studies of 24 weeks of treatment have not demonstrated the development of lenticular changes consistent with posterior subcapsular cataracts. On the basis of available studies, patients receiving standard doses of intranasal corticosteroids are not at increased risk for the development of glaucoma. As with all potential side effects of intranasal corticosteroids, individual patient variability may allow for the development of ocular effects from intranasal corticosteroids, especially in older patients.

BONE

Studies and a review of the literature point toward a negative relationship between total cumulative inhaled corticosteroids and bone marrow density in children and adults with asthma. However, there are limited data examining the effect, and, in particular, the effect after long-term administration of intranasal corticosteroids on bone marrow density. Short-term administration of budesonide, triamcinolone, and mometasone at a dose of 200 mcg/d resulted in no suppression of plasma osteocalcin levels. However, other studies have shown reduced bone mineral density after use of inhaled corticosteroids.

GROWTH

In children, concerns about possible adverse effects on growth raise special considerations. Growth suppression, assessed by stadiometer height measurement, was detected in children with perennial allergic rhinitis treated with intranasal beclomethasone dipropionate for 1 year at twice the recommended dose. Similar studies with intranasal fluticasone propionate, mometasone furoate, and budesonide show no effects on growth compared with placebo at recommended doses and reference values (as much as 2 times recommended doses), except in toddlers.

80. Although local side effects are typically minimal with the use of intranasal corticosteroids, nasal irritation and bleeding may occur. Nasal septal perforation is rarely reported.

The most common side effects associated with the use of intranasal corticosteroids are a result of local irritation. Burning or stinging is most often associated with the use of propylene glycol-containing solutions.

Nasal bleeding has been seen with intranasal corticosteroids, usually as blown blood-tinged secretions. Nasal septal perforation has rarely been reported with long-term use of intranasal corticosteroids. Patients should direct the spray away from the septum to prevent repetitive direct application to the septum. The nasal septum should be periodically examined to assure that there are no mucosal erosions present because these may precede the development of nasal septal perforations.

Nasal biopsies in patients with perennial allergic rhinitis show no evidence of atrophy or other tissue change after 1 to 5 years of therapy. Evaluation of the histologic and macroscopic appearance of the nasal mucosa after administration of intranasal corticosteroids has shown no deleterious pathological changes from that after placebo or antihistamines. In vitro and some in vivo studies have shown that benzalkonium chloride alone, and in 1 clinical study, a corticosteroid nasal spray containing benzalkonium chloride, can promote ciliary stasis and reduce mucociliary transport.
**Oral corticosteroids**

81. A short course (5-7 days) of oral corticosteroids may be appropriate for the treatment of very severe or intractable nasal symptoms or to treat significant nasal polyposis. However, single administration of parenteral corticosteroids is discouraged and recurrent administration of parenteral corticosteroids is contraindicated because of greater potential for long-term corticosteroid side effects. D

Oral corticosteroids should not be administered as therapy for chronic rhinitis, except for rare patients with severe intractable nasal symptoms who are unresponsive to other modalities of treatment. In such patients, especially those with polyposis, a short course of short acting oral corticosteroids, such as prednisone or methylprednisolone, may be appropriate.416,417 Because of the variability of patient response, as reflected in data from studies addressing adrenal response to oral corticosteroids, the potential for adrenal suppression should be considered in any patient who receives oral corticosteroids.

Parenteral corticosteroid administration is not recommended because of the greater potential for long-term corticosteroid side effects, in particular prolonged adrenal suppression as well as local muscle atrophy and fat necrosis.418-420 Recurrent parenteral corticosteroid administration in the treatment of rhinitis is contraindicated.

Intratrubinate injection of corticosteroids is sometimes used by otolaryngologists for the treatment of inferior turbinate hypertrophy. Side effects are usually minor, but permanent vision loss because of cavernous vein thrombosis has been reported in 0.006% of patients.421-423 Nasal and oral corticosteroids are safer alternatives.

**Intranasal cromolyn**

82. Intranasal cromolyn sodium is effective in some patients for prevention and treatment of allergic rhinitis and is associated with minimal side effects. It is less effective in most patients than corticosteroids and has not been adequately studied in comparison with LT antagonists and antihistamines. A

A 4% pump spray solution of cromolyn sodium, United States Pharmacopeia, is available for topical intranasal treatment of seasonal and perennial allergic rhinitis. The main benefit is a strong safety profile. When used to treat symptoms of seasonal allergic rhinitis, cromolyn should be started as early in an allergy season as possible. An effect is normally noted within 4 to 7 days of initiation. However, severe or perennial cases may require 2 weeks or more for maximum effect. Patients who are highly symptomatic may require the addition of an antihistamine-decongestant combination during the first few days of cromolyn treatment. Because a patent nasal airway is a prerequisite, a decongestant may be necessary for a few days. Thereafter, the treatment is continued at whatever maintenance dose is effective for the remainder of the expected season or period of exposure.

Cromolyn sodium has been shown to inhibit the degranulation of sensitized mast cells, thereby preventing the release of mediators of the allergic response and of inflammation. Thus, it prevents the allergic event rather than alleviates symptoms once the reaction has begun.424-429 Nasal cromolyn is effective in the treatment of episodic rhinitis, such as before anticipated allergen exposure, where there appears to be a more rapid onset of action.432-434 The protective effect of cromolyn against nasal antigen challenge persists for 4 to 8 hours after insufflation,493 making it an ideal preventative treatment to consider with predictable exposures such as veterinarians.444

In controlled treatment studies, cromolyn was superior to placebo. A randomized, double-blind, placebo-controlled study in children 2 to 5 years of age demonstrated that cromolyn sodium provided relief of symptoms of allergic rhinitis.445 In addition, the effectiveness of cromolyn sodium in allergic rhinitis was demonstrated among self-selected patients in a nonprescription setting.946 However, cromolyn was generally less effective than intranasal corticosteroids and has not been adequately studied in comparison with LT antagonists and antihistamines.535

Side effects are usually mild and local, including sneezing and nasal stinging or burning. Nasal septal perforations and nasal crusting have not been reported with the use of nasal cromolyn sodium. Because of its excellent safety profile, including a lack of significant drug interaction, cromolyn should be considered in very young children and pregnancy.902,905 Patient selection is critical, and published review articles describe its limited role in treating and preventing allergic rhinitis symptoms.947

There is no evidence that intranasal cromolyn will benefit patients with (1) vasomotor rhinitis, (2) NARES, or (3) nasal polyposis.430,431

**Intranasal anticholinergics**

83. Intranasal anticholinergics may effectively reduce rhinorrhea but have no effect on other nasal symptoms. Although side effects are minimal, dryness of the nasal membranes may occur. A

84. The concomitant use of ipratropium bromide nasal spray and an intranasal corticosteroid is more effective than administration of either drug alone in the treatment of rhinorrhea without any increased incidence of adverse events. A

Increased cholinergic hyperreactivity has been documented in patients without and with allergy as well as in patients with recent upper respiratory tract infections.710,948-950 A significant proportion of histamine-induced and antigen-induced secretion appears to be cholinergically mediated.951,952 In addition to increased glandular secretion, parasympathetic stimulation causes some vasodilation, particularly sinusoidal engorgement, which may contribute to nasal congestion. Ipratropium bromide and glycopyrrolate are quaternary structured ammonium muscarinic receptor antagonists that are poorly absorbed across biological membranes. Ipratropium bromide is poorly absorbed into the systemic circulation from the nasal mucosa.953

Ipratropium bromide has been the most extensively studied intranasal anticholinergic agent. Ipratropium bromide exerts its effect locally on the nasal mucosa, resulting in a reduction of systemic anticholinergic effects (eg, neurologic, ophthalmic, cardiovascular, and gastrointestinal effects) that are seen with tertiary anticholinergic amines. Controlled clinical trials have demonstrated that a quaternary agent such as intranasal ipratropium bromide does not alter physiologic nasal functions (eg, sense of smell, ciliary beat frequency, mucociliary clearance, or the air conditioning capacity of the nose).959 Atrovent (ipratropium bromide; Boehringer-Ingelheim, Ridgefield, Conn) nasal spray 0.03% has been approved for use in patients 6 years of age and older on the basis of its effectiveness in treating rhinorrhea caused by perennial allergic and nonallergic rhinitis in adults and children.437-442 The 0.06% concentration has been approved for patients 5 years of age and older for rhinorrhea associated with the
common cold. Ipratropium bromide is approved only for the treatment of rhinorrhea, although 1 pediatric study showed modest benefit for controlling nasal congestion.\(^{448}\) It has been shown that the concomitant use of ipratropium bromide nasal spray and antihistamines may provide increased efficacy over either drug alone without any increase in adverse events.\(^{442}\) The combined use of ipratropium bromide nasal spray 0.03% and an intranasal corticosteroid is also more effective than administration of either drug alone in the treatment of rhinorrhea without any increased incidence of adverse events.\(^{390}\) The effectiveness and safety of ipratropium bromide nasal spray 0.03% have also been demonstrated in cold-induced rhinitis (eg, skiers),\(^{436}\) and it is useful in reducing rhinorrhea associated with eating (gustatory rhinitis).\(^{66}\) Ipratropium bromide nasal spray 0.06% is effective for rhinorrhea produced by the common cold, in part because of parasympathetic stimulation.\(^{443-447}\)

The most frequently reported adverse events in studies evaluating ipratropium bromide nasal spray 0.03% (as reported in the product information) were mild transient episodes of epistaxis (9%) compared with 5% after use of saline vehicle and nasal dryness (5%) compared with 1% after use of saline vehicle. In addition, the safety of the 0.06% concentration has been demonstrated in children with upper respiratory infections.\(^{954}\)

**Oral anti-LT agents**

85. Oral anti-LT agents alone or in combination with antihistamines have proven to be useful in the treatment of allergic rhinitis. A

Leukotriene receptor antagonist produce statistically significant improvement in nasal symptoms and standardized rhinoconjunctivitis QOL scores compared with placebo in a number of studies for seasonal allergic rhinitis,\(^{450-452}\) and for perennial allergic rhinitis as well.\(^{453}\) The onset of action occurs by the second day of daily treatment.\(^{46}\) There is no significant difference in efficacy between LTRA and antihistamines (with loratadine as the usual comparator).\(^{40,42,377}\) Likewise, compared with pseudoephedrine, montelukast shows similar reduction in all symptoms of allergic rhinitis except the symptom of nasal congestion, for which pseudoephedrine is more effective.\(^{955}\) Unlike antihistamines, LTRA do not significantly suppress skin tests.\(^{956,957}\) LTRA are less effective than intranasal corticosteroids.\(^{375,958}\) The combination of an antihistamine and LTRAs is superior to either therapy when given alone. Intranasal corticosteroids are either equal to \(^{378,379}\) or superior\(^{376,377}\) to the combination of an antihistamine and an LTRA. These differences may in part be a result of which antihistamine is used in the combination therapy. Combination therapy with an antihistamine and an anti-LT agent or decongestant may provide alternative treatment for patients who are unresponsive to or not compliant with intranasal corticosteroids, or for whom intranasal corticosteroids are contraindicated.

Montelukast is asafe and effective treatment for the management of allergic rhinitis in children. It is approved for perennial allergic rhinitis in children as young as 6 months and for seasonal allergic rhinitis in children as young as 2 years. Other LTRAs may also be efficacious but have not been adequately studied. Combination of montelukast and a second-generation antihistamine may show added benefit for allergic rhinitis and provide better protection against seasonal decrease in lung function.\(^{457}\) Recognizing that as many as 40% of patients with allergic rhinitis have coexisting asthma, montelukast may be considered when treatment can benefit the combined upper and lower airway.\(^{454-456}\) In children with mild persistent asthma and coexisting allergic rhinitis, montelukast has been recommended for monotherapy.\(^{354}\) The use of LTRA for combined upper and lower airway allergic diseases as either a monotherapy or combined therapy is particularly attractive when treating a child whose parents are steroid-phobic.

**Omalizumab**

86. Omalizumab has demonstrated efficacy in AR; however, it has FDA approval for use only in allergic asthma. A

Although not approved for the use in allergic rhinitis, omalizumab has demonstrated efficacy in this illness. Humanized mAb (omalizumab) has demonstrated efficacy in attenuating bronchial responses to inhaled Aeroallergen challenges,\(^{959}\) and in allergic asthma,\(^{960,961}\) through a reduction of circulating IgE. Patients with seasonal and perennial allergic rhinitis had significant reduction of both nasal and ocular symptoms and improved QOL\(^{458}\) after the use of omalizumab. Omalizumab, however, has not demonstrated superiority to currently approved treatments for rhinitis. Thus, when one considers the cost of this treatment, it precludes its use for the treatment of allergic rhinitis in the absence of asthma. Unlike conventional allergen immunotherapy, which may improve the long-term course of allergic rhinitis even after it is discontinued, there is no evidence that omalizumab improves the natural course of allergic rhinitis after its discontinuation.

**Saline**

87. There is evidence that topical saline is beneficial in the treatment of the symptoms of chronic rhinorrhea and rhinosinusitis when used as a sole modality or for adjunctive treatment. A

Topical saline is commonly used as a treatment for rhinitis and rhinosinusitis in both children and adults. Although less effective than intranasal corticosteroids and no more effective than other active agents for rhinitis, isotonic and hypertonic saline solutions, used as either single or adjunctive agents, are of modest benefit for reducing symptoms and improving the QOL in patients with allergic rhinitis and rhinosinusitis.\(^{459}\) In one 4-week study, the use of saline as a wetting agent for perennial rhinitis demonstrated reduced sneezing and nasal stuffiness, reduced nasal blockage (measured by peak flow), and a reduction in eosinophils (nasal biopsy).\(^{962}\) Overall, there is no difference in symptom or radiologic scores when comparing isotonic with hypertonic saline.\(^{462,463}\) Various mechanisms, such as improvement in mucus clearance; enhanced ciliary beat activity; removal of antigen, biofilm, or inflammatory mediators; and a protective role on sinonasal mucosa, have been proposed but not confirmed to explain the reported symptom improvement. Although it has been shown that hypertonic saline solutions improve mucociliary clearance,\(^{462,468}\) this may not be the explanation for the clinical improvement obtained from saline irrigation.

The use of topical saline is associated with minimal side effects such as burning, irritation, and nausea; has low cost; and has overall good patient acceptance.\(^{460,461}\) The preferred method of delivery—nose spray, bottle, pump, irrigation, or nebulizer; the volume; and the dose frequency have not been established. Frequently used homemade formulas for isotonic and hypertonic saline are listed in Table IX.
Allergen immunotherapy

88. Allergen immunotherapy is effective for the treatment of allergic rhinitis. A

89. Allergen immunotherapy should be considered for patients with allergic rhinitis who have demonstrable evidence of specific IgE antibodies to clinically relevant allergens, and its use depends on the degree to which symptoms can be reduced by avoidance and medication, the amount and type of medication required to control symptoms, and the adverse effects of medications. (A)

90. Allergen immunotherapy may prevent the development of new allergen sensitizations and reduce the risk for the future development of asthma in patients with allergic rhinitis. B

Multiple randomized, prospective, single-blind or double-blind, placebo-controlled studies demonstrate effectiveness of specific allergen immunotherapy in the treatment of allergic rhinitis. 51,464,365 Allergen immunotherapy is the only treatment intervention that has been shown to modify the natural history of allergic rhinitis. 50,468 Unlike pharmacotherapy, the clinical benefits may be sustained years after discontinuation of treatment. 466,467 Allergen immunotherapy for allergic rhinitis may prevent the development of new allergen sensitizations and reduce the risk for the future development of asthma in patients with allergic rhinitis. 527-481 The expected response to allergen immunotherapy is allergen-specific and depends on proper identification and selection of allergens on the basis of the patient’s history, exposure, and diagnostic test results. Allergen immunotherapy is effective in both adults and children. 471,479,482-485 Its efficacy is confirmed for the treatment of inhalant allergy caused by pollen, 466,486-492 fungi, 493-497 animal allergens, 498-505 dust mite, 506-517 and cockroach. 518

Allergen immunotherapy should be considered for patients who have symptoms of allergic rhinitis or rhinoconjunctivitis after natural exposure to allergens and who demonstrate specific IgE antibodies to relevant allergens. The severity, lack of response to or side effects from other interventions, and duration of symptoms should all be considered when assessing the need for specific allergen immunotherapy. Likewise, the patient’s desire to avoid long-term pharmacotherapy and to seek treatment that can potentially modify allergic disease are additional factors to be considered when reaching a decision to initiate allergen immunotherapy. Coexisting medical conditions, such as asthma and sinusitis, should also be considered in evaluation of a patient who may be a candidate for allergen immunotherapy. Patients with moderate or severe allergic asthma and allergic rhinitis should be managed with a combined aggressive regimen of allergen avoidance and pharmacotherapy, but these patients may also benefit from allergen immunotherapy providing their asthma is stable when the allergen immunotherapy injection is administered. 50,468 Immunotherapy is usually not more costly than pharmacotherapy over the projected course of treatment. 519,520

The risks of allergen immunotherapy include common local reactions, swelling and induration at the injection site, and in rare instances, life-threatening and fatal reactions. 963 The estimated allergen immunotherapy fatality rate was 1 per 2.5 million injections (average of 3.4 deaths per year) according to a recent AAAAI survey of physician members. 964 Similar to 2 previous surveys of AAAAI physician members. 965,966 Identified risk factors for anaphylaxis after allergen immunotherapy include symptomatic asthma, injections administered from a new vial, β-blockers, a high degree of skin test reactivity, and injections given during times of symptom exacerbations. 967

Contraindications for allergen immunotherapy include patients with medical conditions that would reduce their ability to survive allergen immunotherapy systemic allergic reactions or the resultant treatment. 56 Examples include severe asthma uncontrolled by pharmacotherapy and significant cardiovascular disease.

Clinical improvement can be demonstrated very shortly after the patient reaches a maintenance dose. 494,503,968,969 If clinical improvement is not apparent after 1 year of maintenance therapy, possible reasons for lack of efficacy should be evaluated, and discontinuation of treatment should be considered if none are found. If allergen immunotherapy is effective, treatment may be continued for longer than 3 years depending on the patient’s ongoing response to treatment. Currently there are no specific tests or clinical markers that will distinguish between patients who will relapse and those who will remain in long-term clinical remission after discontinuing effective inhalant allergen immunotherapy, and the decision to continue or stop immunotherapy must be individualized (refer to “Allergen Immunotherapy: A Practice Parameter Second Update” 58 for further information regarding allergen immunotherapy).

Surgical approaches for comorbid conditions

91. Although there is no surgical treatment for allergic rhinitis, surgery may be indicated in the management of comorbid conditions, such as nasal obstruction from severe nasal septal deviation or inferior turbinate hypertrophy, adenoidal hypertrophy, or refractory sinusitis and complications thereof. C

A variety of anatomical variants can lead to persistent nasal obstruction that may amplify the congestion and turbinate hypertrophy secondary to allergic inflammation. Surgery may play a beneficial role in the management of conditions associated with rhinitis—for example, mechanical nasal obstruction caused by anatomical variants such as septal deviation or concha bullosa, 75 refractory sinusitis with or without nasal polyposis, 524 and inferior turbinate hypertrophy, mucosal or bony, refractory to maximal medical treatment. 5 Surgery to reduce nasal obstruction may improve the nasal airflow and allows for more effective delivery of topical medications.

Patients with rhinitis who develop acute bacterial sinusitis will usually require antibiotics. However, even with appropriate treatment, a small percentage of patients will develop complications such as periorbital edema, meningitis, brain abscess, cavernous sinus thrombosis, or subperiosteal abscess with the risk of permanent vision loss or even death. 77,521-523 These patients may require surgical intervention. Patients with chronic sinusitis with or without nasal polyps may also require surgical intervention (see “The Diagnosis and Management of Sinusitis: A Practice Parameter Update”). 77

The nasal airway creates more than half of the total respiratory resistance to the lungs. Within the nose the internal nasal valve, the narrowest portion found in the anterior nose, is responsible for more than 2/3 of the airflow resistance produced by the nose. 970 As air passes through the nasal vault in a laminar flow pattern, there is an increase in its speed and pressure. Expanding into the nasal valve cavity, a turbulent flow pattern is created as the air is exposed to a large surface area for conditioning. Any time the turbulent airflow pattern is disturbed, it is perceived as nasal
obstruction, whether the passage is either too narrow or too wide. The septal valve involves the space between the anterior tip of the inferior turbinate and the septum and is the area most commonly associated with the subjective perception of obstruction. A small anterior deviation of the septum is much more significant that a larger posterior deviation. Anterior septal deviation, with or without nasal valve collapse, and anterior inferior turbinate hypertrophy are thus the major structural components resulting in the symptom of nasal obstruction.

Correction of nasal septal deviation is one of the most common surgical procedures completed. The nasal septum is off-center in approximately 80% of the general population and appears deviated by CT in as many as 56%. It has been estimated that in patients with nasal obstruction, a clinically significant deviated nasal septum is present in 26%. Trauma, intrauterine, during birth, or postnataally, is the most common etiology of a deviated septum, and the degree of trauma required for clinically significant deviation is inversely related to the patient’s age. Furthermore, the obstruction becomes more pronounced over time with cartilaginous overgrowth on the dominant side. The type of deviation varies, with the most common classifications septal tilt (40%), C-shaped anteroposterior deviation (32%), and S-shaped anteroposterior (9%). Typically there is also unilateral compensatory turbinate hypertrophy on the side opposite the deviation, which may even be bilateral with an S-shaped deviation. The surgical procedures for correction of a deviated septum usually are submucosal resection and septoplasty. Submucosal resection involves more extensive resection of cartilage and bone, is less tissue-sparing, and has a higher incidence of septal perforation complications. Septoplasty, currently the preferred procedure, reshapes, repositions, or recontours the cartilage, with as many as 77% of patients achieving subjective improvement. The exact techniques, such as scoring, morselization, or removal of cartilage, with manual or powdered instrumentation, and/or the use of cartilage grafts will depend on the type and severity of the septal deviation. Endoscopic septoplasty is replacing traditional septoplasty in many clinical settings. Inferior turbinate reduction surgery, as described below, is often performed concurrently with septoplasty, although some studies fail to show any long-term benefit. Because long-term results from septoplasty are not always satisfactory—for example, there may be recurrence of deviation or a disturbed nasal cycle—the surgeon must make a careful preoperative assessment and attempt to differentiate between physiological and pathological septal deviation and consider all factors that may be contributing to nasal obstruction.

Allergic rhinitis and nonallergic rhinitis cause swelling of the nasal mucosa, most notably of the inferior turbinates. It has been estimated that as much as 20% of the population has chronic nasal obstruction caused by turbinate hypertrophy. Medical treatment may not be successful in shrinking the nasal mucosa and alleviating the symptoms of chronic nasal obstruction. At times, unrelated nasal surgery, such as cosmetic rhinoplasty, may inadvertently lead to increased nasal obstruction by reducing the nasal valve or changing the airflow pattern. To select the most appropriate surgical procedure for long-term outcome, the surgeon must assess the contribution of turbinate mucosal hypertrophy versus the position and degree of bony hypertrophy. The degree of shrinkage with a topical decongestant may assist in delineating mucosal from bony hypertrophy and predict success of certain procedures, such as radiofrequency volumetric tissue reduction (RFVTR).

Multiple surgical procedures on the inferior turbinate have been described, and all are considered to have some beneficial effects. The goal of these techniques is to reduce the size of the inferior turbinate outright, or to diminish its ability to swell and block the nasal passages. The various surgical procedures address the mucosal hypertrophy, the bony hypertrophy, or a combination of bony and mucosal hypertrophy. Mucosal hypertrophy reduction focuses either on the surface mucosa (eg, electrocautery and laser vaporization) or intramurally (eg, bipolar cautery or radiofrequency ablation), with intentional submucosal tissue injury resulting in tissue loss and subsequent scarring thereby leading to a reduced bulk of the inferior turbinate, while preserving the surface mucosa. Bony hypertrophy is addressed with submucosal resection, which tends to spare submucosa and mucosa. In contrast, partial turbinectomy and turbinoplasty procedures remove bone, submucosa, and mucosa. Lateral outfracture, a procedure of repositioning the turbinate laterally by fracturing the turbinate bone, does not reduce either mucosal or bony hypertrophy and has reduced surgical complications but may give only temporary results.

When bony hypertrophy is present, the surgeon has several techniques from which to choose. Turbinectomy involves fracturing the turbinate bone and then snipping off the bone, submucosa, and mucosa. Turbinoplasty involves fracture, followed by mucosal incision and removal of a wedge of conchal bone with attached inferior and lateral soft tissue. The posterior turbinate tip is also excised. The mucosal flap is then used to form a neoturbinate. Compared with partial turbinectomy, turbinoplasty spares more mucosal surface and has less chance of bleeding and postoperative crusting. Submucosal resection preserves the most mucosa but is more technically difficult and does not address the posterior inferior turbinate. Powered microdebrider-assisted inferior turbinoplasty, a relatively new procedure, can be conducted in the office setting under local anesthesia. After a small incision in the anterior inferior turbinate tip, the powered blade/suction device is introduced, and the bone and submucosa are crushed and removed by suction, thereby preserving the turbinate mucosa. It is associated with no significant bleeding or crusting. It is felt to be superior to both submucosal cautereion and submucosal resection. Laser turbinectomy may use the carbon dioxide, neodymium-doped yttrium aluminum garnet, or diode lasers. The tissue is vaporized in areas, leaving islands of intact mucosa. This can be performed under local anesthesia, minimal bleeding is noted, and there is no need for packing. Postoperative crusting may be noted. Any of these procedures may offer a beneficial effect of symptom improvement and increased nasal airflow in patients with allergic rhinitis and coexisting turbinate hypertrophy that has been unresponsive to medical therapy.

Electrocautery can be either linear mucosal or submucosal using a unipolar or bipolar electrode inducing fibrosis and wound contracture with resultant volume reduction. Surgical bleeding is minimal; however, mucosal edema and crusting are usually noted for 1 week postoperatively. Cryosurgery results in the formation of intracellular ice crystals causing cell membrane destruction, blood vessel thrombosis, tissue ischemia, and resultant tissue destruction. This procedure can also be completed in the clinic setting under local anesthesia and has minimal bleeding but has...
prolonged healing over 6 weeks as the necrotic tissue sloughs and may not have long-term benefit. Argon plasma coagulation uses high-frequency electrocoagulation without tissue contact because the electric current is conducted via ionized argon gas. This produces tissue desiccation but preserves the mucosa and has shown promising 1-year results. 533

Radiofrequency ablation (RFVTR), a relatively new technique conducted under local anesthesia, creates ionic agitation in the tissue, inducing submucosal necrosis and fibrosis and reduced blood flow to the turbinate. Resultant wound contraction causes volume reduction of the inferior turbinate without damage to the overlying mucosa and preserves mucciliary clearance. 524 There is minimal bleeding and postoperative crusting, there is no requirement for packing, and repeat surgery may be completed if necessary. 534 In 1 small prospective study of patients with allergy not responding to medical treatment, RFVTR reduced nasal obstruction for as long as 6 months. 524,535

Adenoidectomy in children (average age, 7 years 976) continues to be 1 of the 10 most frequently performed surgical procedures, with more than 196,000 adenoidectomies performed annually in the United States. 976 In children, the indications for adenoidectomy are sleep apnea caused by adenotonsillar hypertrophy, chronic adenoiditis, and chronic sinusitis. 543 For OME, an adenoidectomy is usually recommended after the first set of tympanostomy tubes extrude, effusion returns, and a second set of tympanostomy tubes are being considered. An adenoidectomy may also be considered for coexisting adenoiditis, postnasal obstruction, or chronic sinusitis. 543 Recent clinical studies recommend a trial intranasal corticosteroids for adenoidal hypertrophy before surgical intervention. 544-547 Septoplasty is infrequently performed in children because it may have a negative effect on nasal growth, particularly of the nasal dorsum. 527

Important considerations in management

92. Management and monitoring of rhinitis should be individualized and based on the spectrum, duration, and severity of symptoms; physical examination findings; comorbidities; age of the patient; and patient preferences using both step-up and step-down approaches. C

93. Effective allergic rhinitis management requires the development of a physician/patient/family partnership, avoidance of environmental triggers, and the appropriate use of prescribed therapeutic interventions. C

The approach to rhinitis management must be individualized, considering such variable factors as the patient’s age as well as the frequency, severity, and spectrum of presenting symptoms, the degree of impairment of QOL, the specific allergens to which the individual is sensitized, the response to previous medications, the presence of comorbid conditions, and the costs. 545 An individualized approach begins with the history, which will reveal the pattern, seasonal or perennial; the frequency, severity, and spectrum of presenting symptoms; the response to and compliance with previous medications; indoor and/or outdoor allergen exposures; and the presence of comorbid conditions such as allergic conjunctivitis and asthma. Symptoms confined to a defined season allow the formulation of a prophylactic regimen consisting of the initiation of medication before the onset of that season. 977 In contrast, perennial symptoms may require daily and, frequently, year-round therapy. The more days per year that therapy is required, the more medication safety and ease of use become prime factors to consider in individualizing therapy. The approach to treatment may also need to be modified for individuals who have perennial symptoms with seasonal exacerbations. Episodic rhinitis may be approached by administering certain medications appropriate as prophylaxis before anticipated acute allergen exposure (eg, nasal cromolyn, oral or intranasal antihistamines) and/or medications suitable for as-needed use in response to symptoms or scheduled shorter-term use (Table VI). Although antihistamines can be used on an intermittent basis, such as for episodic allergic rhinitis, it has been shown that continuous treatment for seasonal or perennial allergic rhinitis is more effective, 331 primarily because of unavoidable, ongoing allergen exposure.

The physical examination will assist in assessing severity of disease as well as the presence of comorbid conditions. For example, the presence of a polyp in a patient may result in modification of the diagnostic tests requested—for example, CF testing in a child—as well as the therapy recommended—for example, oral or high dose intranasal corticosteroids and possibly surgical resection in the adult.

The age of the patient becomes important in developing the individual evaluation and treatment plan. The spectrum of allergens tested as well as the choice of a medical regimen must be modified by the age of the individual. Medical choices are most heavily influenced by the extremes of age as in the infant or young child and the elderly. After allergy testing, the physician should design environmental control measures to target the specific allergens identified for the patient. Environmental controls will frequently also need to target nonallergen, irritant triggers such as tobacco smoke, strong odors, and extremes in temperature and humidity. Individuals who respond poorly to environmental control measures and optimal medical management should be considered for allergen immunotherapy.

The treatment plan should be developed jointly with the patient and family. Ideally it will take into account not only the patient’s school or work schedule for medication administration, but also the patient’s medication preferences such as liquid versus pill versus spray; realistic goals for environmental modification; and a plan to encourage compliance, such as use of a planning calendar or check-off list. 11 For example, programs tailored for the school-age child highlighting the importance of pleasant taste and ease of use of medications for this age group have resulted in increased adherence to the prescribed medications. 550

Evidence-based guidelines for the treatment of rhinitis have recommended selection of appropriate medications on the basis of the severity and frequency of the patient’s symptoms. 175 In addition, the therapy of rhinitis should involve a step-up approach (when therapy is inadequate) or step-down approach (after symptoms relief is achieved or maximized). As indicated, selection of the pharmacologic agent for treatment (eg, intranasal corticosteroids or second-generation antihistamines) must be individualized on the basis of the patient’s age, symptoms, tolerability of route of administration, overall clinical condition, comorbidities, 11 and concomitant medication. This requires a careful benefit/risk assessment in each individual patient. 549 See Table VI for a summary of medication classes and their properties that lend themselves to different types of rhinitis and administration strategies.

Intranasal corticosteroids and second-generation antihistamines (with or without decongestants) have been shown to be safe and effective for most patients. LT antagonists and nasal
cromolyn may also be appropriate in some patients. In regard to nasal congestion, intranasal corticosteroids have been shown consistently to be effective, whereas oral second-genera-
tion antihistamines have not, often requiring the addition of an oral decongestant. Decongestant nasal sprays may significantly decrease nasal congestion and if used for short periods or intermit-
tently may not produce rebound nasal congestion. Because most patients have multiple symptoms, the use of a single medication designed primarily for a specific symptom, such as a decongestant for nasal congestion, is usually not necessary. One exception is the use of anticholinergic nasal sprays for patients who have rhinor-
hea without other nasal symptoms.

Whatever medication is first selected to treat a patient with rhinitis, addition or substitution of another class of medication should be considered if the first medication does not sufficiently control the patient’s symptoms. This may be particularly relevant if there is a need to control other nonnasal symptoms, such as ocular or lower respiratory symptoms, which may also require an additional therapeutic agent. The use of combination therapy for rhinitis, on the other hand, has not always been shown to provide a major therapeutic advantage that outweighs the cost of this approach. Administration of most med-
ifications for a period of 2 to 4 weeks is usually long enough to determine efficacy.

If patient compliance with a therapy was poor in the past or an adverse event was experienced, an alternative medical regimen should be designed with that in mind. Some patients and parents harbor fears of medication side effects, and these should be addressed on an individual basis through education to optimize medication adherence. Individuals vary in their ability to learn to use new devices, so the approach to education often needs to be tailored to the individual.

After initiation of therapy, appropriate follow-up for patients with rhinitis is recommended. This optimizes the likelihood that a patient will benefit from the broad array of therapeutic approaches available and that possible complications from rhinitis or its treatment are identified and addressed. At each follow-up patient visit, the physician should assess symptom control, QOL, and compliance, and evaluate whether current therapy should be maintained, stepped up, or stepped down. Patient and family education should be an integral part of each patient encounter.

Education of patients and family members or other patient advocates

94. Education is a key element in promoting adherence and op-
timizing treatment outcomes in allergic rhinitis. D

Education of the patient and family members or other patient advocates encompasses knowledge of and sensitivity to the cultural, socioeconomic, and demographic characteristics of the patient. To provide for optimal compliance, a trusting partnership of the physician and office staff with the patient and patient’s family is needed. Education for the patient and family members begins at the initial encounter and continues at ensuing visits. The education program should emphasize the chronicity of rhinitis as a disease; the realistic outcome of therapy; an understanding of how to implement appropriate environmental change; appropriate methods of medication administration, medication benefits, and possible side effects; the comorbidity of other allergic diseases, such as asthma, sinusitis, and otitis media; and the effect that disease control can make in overall improvement in QOL. In some studies, rhinitis education has been shown to result in en-
hanced compliance with rhinitis treatment and follow-up care, re-
duced concomitant asthma symptoms, and reduced use of short-
acting β-agonists. Physician-delivered educational programs have also resulted in a decrease in prescribed medications, an in-
crease in the implementation of preventative measures, and im-
provement in the patient-physician partnership. When allergy immunotherapy is recommended, an emphasis on education is needed to increase patients’ knowledge about immunotherapy be-
fore and during aeroallergen immunotherapy, to increase compli-
cance and safety, and to aid in the ultimate success of this therapeutic modality.

Although there is general agreement that education is impor-
tant, the best delivery method, frequency, and educational setting are still not established. The published research on success of rhinitis educational efforts is very limited, and what is published does not always demonstrate a positive result. Contrary to expectations, 1-on-1 allergy treatment educational sessions may not increase knowledge any more than a simple handout. In fact, it is difficult to demonstrate reduced use of medication, re-
duced office visits, or improvement in QOL when educational programs are implemented for rhinitis or asthma. In recent asthma surveys, for example, nonadherence (at times intentional) to daily controller therapy was related not to lack of information but to a desire for the patient to have active control over use of medica-
tions, often using medications on a PRN basis. When one reviews asthma educational programs, the newer, more innovative methods, such as videos, computer, and web-based programs, have not demonstrated any measurable advantage over in-office educational discussions, which may increase asthma knowledge and reduce symptoms but which do not show any reduction in acute care visits or rescue inhaler use.

Whatever rhinitis educational delivery method is selected, it is important to review the content of the material. Although a large number of commercially prepared brochures and leaflets are available on allergic rhinitis, these are of variable quality and are often outdated, may project a biased treatment perspective, usually have poor readability scores, may contain factual inaccuracies, and are written predominantly for adults.

Patient education is essential to provide the best care for the patient with rhinitis. Additional research to determine the best methods for education delivery is urgently needed to attain this goal.

Major comorbid conditions

95. Patients with allergic rhinitis are at increased risk for the de-
velopment of asthma. A
96. Treatment of allergic rhinitis may improve asthma control in
patients with coexisting allergic rhinitis and asthma. B
97. There is no established cause-and-effect relationship of rhi-
nitis with recurrent otitis media and OME. C

The upper and lower airways are closely related with respect to rhinitis and asthma. Although allergic rhinitis and asthma frequently coexist, patients presenting with allergic rhinitis are at an increased risk for the development of asthma. There is also evidence of interaction between the upper and lower air-
ways. Patients with allergic rhinitis without asthma, especially those sensitized to dust mites, often have nonspecific BHR; and many patients with seasonal allergic rhinitis experience a seasonal increase in BHR. Nasal allergen provocation has been shown to result in temporary increases in BHR.
airway adhesion molecules, and lower airway eosinophilic inflammation. Conversely, subsegmental bronchial allergen challenge in patients with allergic rhinitis has been shown to result in both bronchial and nasal inflammatory responses. It has been suggested that in patients with moderate to severe allergic rhinitis, especially those with longstanding rhinitis and sensitization to dust mites, a reduced forced expiratory flow at 25% to 75% of forced vital capacity may be a marker of early bronchial impairment.

There is clinical evidence that treatment of rhinitis can improve the status of coexisting asthma. Treatment with intranasal corticosteroids has been shown to prevent the seasonal increase in BHR experienced by patients with allergic rhinitis, to reduce existing BHR, to improve pulmonary function tests, to diminish asthma symptoms, and to reduce exhaled nitric oxide and hydrogen peroxide.

Treatment of allergic rhinitis with intranasal corticosteroids and certain second-generation antihistamines may improve asthma control when both diseases coexist.

Given the convincing relationship between allergic rhinitis and asthma and the beneficial effects of treating rhinitis in patients who have asthma, it is also imperative that physicians who treat patients with asthma also consider aggressive treatment of coexisting rhinitis.

Allergen specific subcutaneous immunotherapy has been associated with a reduction in nonspecific bronchial hyperresponsiveness in patients with perennial allergic rhinitis. Several controlled studies have also reported a reduction in the incidence of asthma in pediatric patients with allergic rhinitis treated with subcutaneous immunotherapy, and this effect appears to be sustained at least 2 years after discontinuing immunotherapy. One study reported a similar effect in adult patients. Likewise, sublingual immunotherapy for allergic rhinitis, although not yet FDA-approved, may reduce the development of asthma in children.

There has been ongoing discussion of the linkage of rhinitis, especially allergic rhinitis, and recurrent acute otitis media and OME, but there are no controlled studies to show a definite causal relationship. The American Academy of Pediatrics has not recommended any specific allergic rhinitis management for OME. Furthermore, the American Academy of Pediatrics concludes that the use of antihistamines and decongestants is ineffective for OME and are not recommended for treatment. Rhinitis and otitis are both common childhood diseases, making the casual association with viruses, bacteria, and allergens difficult to establish at times. Eustachian tube dysfunction remains the most common etiology for otitis media. However, the same allergic mediators released after allergen exposure resulting in nasal allergic inflammation may contribute to the dysfunction of the eustachian tube by contributing to eustachian tube edema and inflammation. Although under natural circumstances the middle ear is not exposed to allergens, measurements of elevated ECP, IL-5, and IgA within the middle ear support a localized inflammatory process during chronic OME. Similar cytokine and cellular profiles (Eosinophils, T lymphocytes, IL-4 mRNA, neutrophils, and IFN-γ mRNA) have been noted concurrently in the middle ear and adenoid tissue of atopics, thus suggesting that the ear may be part of the united airway. Prospective studies examining the effect of allergy immunotherapy or food elimination on the natural course of OME are lacking.

Special considerations

Treatment of rhinitis during pregnancy

98. When selecting medications for treating rhinitis in pregnancy, the clinician might consider the FDA risk categories that are based largely on animal data and limited human studies. However, it is also beneficial to review human cohort and case-control studies as well as birth registry data before reaching a decision.

The FDA pregnancy risk categories A, B, C, D, and X (Table X) were developed to guide the physician in choosing medications for which the benefit versus risk ratio can be weighed in an informed manner. Most medications fall into B or C categories, based predominantly on animal studies, because there are limited human studies available. It is therefore useful to consider, in addition to the FDA risk category, the exposed lives as reported in birth registry as well as case-control and cohort studies when comparing the available medications and developing a treatment plan. The following medication-related Summary Statements use this combined approach.

99. The most critical time for concern about potential congenital malformation because of medication use is the first trimester, when organogenesis is occurring.

100. A sufficient amount of human observational data has now been accumulated to demonstrate safety for second-generation antihistamines in general as well as first-generation antihistamines.

First-generation antihistamines, such as chlorpheniramine, have previously been recommended as first-choice agents because of their observed safety and longevity of use. However, their undesirable sedative qualities and possible effect on performance may make them less desirable choices. The safety of second-generation antihistamines used during the first trimester of pregnancy has now been confirmed through large birth registries, case-control studies, and cohort studies (Table XII). The available human data for first trimester use of antihistamines are summarized for first-generation and second-generation antihistamines in Tables XI and XII, respectively. The available safety data, efficacy, and patient preference will all influence the final drug selection. Both first-generation and second-generation antihistamines in general have excellent safety records and do not show a significant increase in congenital malformations when used during the first trimester. However, caution is still advised for a few antihistamines. Although diphenhydramine is often used by pregnant patients and recent studies have not detected any increased risk for congenital malformations, there is still some concern over a case-control study suggesting an association with cleft palate that has yet to be sufficiently refuted.

Hydroxyzine should be used cautiously during the first trimester based on animal data. Although there are no reports of increased congenital malformations with the use of fexofenadine during pregnancy and animal studies are negative for teratogenicity, no epidemiologic studies in human pregnancy have been published. Currently there are also limited data on desloratadine, azelastine, and levocetirizine. The only ophthalmic antihistamine for which epidemiologic studies have been conducted is pheniramine, and there was no reported increase in congenital malformations.

101. Oral decongestants should be avoided during the first trimester. Topical decongestants when used on a short-term basis may have a better safety profile than oral agents for first trimester use.
There have been conflicting reports of the association of phenylephrine and pseudoephedrine with increased congenital malformations such as gastroschisis and small intestinal atresia.594,600 The risks of such malformations were increased by combining a decongestant with acetaminophen or salicylates.600,601 Because of these findings, it is generally recommended that oral decongestants be avoided during the first trimester of pregnancy. Likewise, the data on the safety of topical intranasal decongestants during pregnancy have not been studied.

102. Sodium cromolyn is a safe treatment for allergic rhinitis during pregnancy. C

For allergic rhinitis during pregnancy, nasal sodium cromolyn, a Pregnancy Category B drug, may be considered for use in view of its topical application and reassuring gestational human and animal data.599,602-604 Unfortunately the need for frequent 4 times a day dosing and reduced relative efficacy compared with other agents limits its acceptance by patients.

103. Montelukast is a safe treatment for allergic rhinitis during pregnancy. (C)

Reassuring animal reproductive studies and unpublished human safety data have given montelukast a Pregnancy Category B classification. A published observational study including 9 patients on LTRAs (specific agent not identified) demonstrated no adverse events.605 Montelukast has been recommended for use in pregnancy for asthma management only when there has been a uniquely favorable prepregnancy response.614 The same guidelines would be reasonable for the use of montelukast for rhinitis in pregnancy management until additional information on efficacy and safety becomes available.

104. Intranasal corticosteroids may be used in the treatment of nasal symptoms during pregnancy because of their safety and efficacy profile. C

Clinical and epidemiologic studies on the safety of intranasal corticosteroids for rhinitis in pregnancy are limited. Although animal gestational studies have shown risk for all inhaled corticosteroids, this does not appear to apply directly to human beings based up observational data.603 Pharmacologic studies show a much lower systemic exposure after intranasal than (orally) inhaled corticosteroids. It is reasonable, therefore, to extrapolate the safety profile of inhaled corticosteroids to intranasal corticosteroids. A recent meta-analysis concluded that the use of orally inhaled corticosteroids during pregnancy does not increase the risks of major malformations, preterm delivery, low birth weight, and pregnancy-induced hypertension.606 Inhaled or intranasal corticosteroid use in pregnancy has demonstrated no convincing evidence of congenital defects using beclomethasone,113,602,607,608 budesonide,603,610 or fluticasone propionate.227,611 Reported safety data on triamcinolone,612,613 mometasone, and flunisolide611 are extremely limited. No substantial difference in efficacy and safety has been shown among the available intranasal corticosteroids. Thus it would be reasonable to continue any of the intranasal corticosteroids that have adequately controlled the patient’s symptoms before pregnancy.609,614 If intranasal corticosteroids are begun during pregnancy, intranasal budesonide, which is in Pregnancy Category B largely on the basis of extensive human safety data, may be preferred.503,610 The decision which intranasal corticosteroid to prescribe often requires a discussion of the benefits and risks with the patient. Intranasal corticosteroids may also be used to allow discontinuation of topical decongestants in patients with rhinitis medicamentosa. As with all medication use in pregnancy, intranasal corticosteroids should be tapered to the lowest effective dose.

105. Immunotherapy for allergic rhinitis may be continued during pregnancy but without dose escalation. C

Specific allergy immunotherapy for allergic rhinitis may be continued during pregnancy if it is providing benefit without causing systemic reactions.614,615 The immunotherapy doses that the patient receives when she becomes pregnant should not be increased and should be adjusted appropriately during pregnancy if necessary to minimize the chance of inducing a systemic reaction. However, benefit/risk considerations do not generally favor starting immunotherapy during pregnancy.50,615

Rhinitis in the elderly

106. Rhinitis in the elderly may be caused by types of rhinitis common in other age groups but may also be influenced by age-related physiologic changes such as cholinergic hyperactivity, anatomic changes, and medications taken for other medical conditions. C

As the US elderly population rapidly increases (41% growth rate for those over 65 years vs 11% for general population), treatment of the elderly with rhinitis will likewise become a major part of the rhinitis practice.991 Many of the pathologic changes in connective tissue and vasculature associated with aging may predispose to rhinitis complaints.516,617 These include atrophy of the collagen fibers and mucosal glands, loss of dermal elastic fibers, fragmentation and weakening of septal cartilage, and a reduced blood flow to nasal tissues.138,992,993 These changes can result in drying and increased nasal congestion in some elderly patients. Furthermore, these aging effects often magnify or complicate other causes of rhinitis, such as allergic causes. Nasal steroids, however, may be safely used for treatment of allergic rhinitis, because they do not cause any clinical or histologic atrophic changes in the nasal mucosa.618

Rhinitis in the elderly may also be a result of cholinergic hyperreactivity, associated with profuse watery rhinorrhea, which may be aggravated after eating (gustatory rhinitis), α-adrenergic hyperactivity (eg, congestion associated with therapy for hypertension or benign prostatic hypertrophy), or chronic sinusitis. The watery rhinorrhea syndrome frequently responds to intranasal ipratropium bromide.994 However, ipratropium bromide should be used with caution with pre-existing glaucoma or prostatic hypertrophy.

Elderly patients more commonly have more pronounced clear rhinorrhea from cholinergic hyperactivity associated with the aging process. Medications taken for unrelated medical problems may also cause or contribute to rhinitis in this age group. Selection of medications for rhinitis treatment should take into account that elderly patients may be more susceptible to adverse effects of some of these medications.

Rhinitis in the athlete

107. Athletic performance can be affected by rhinorrhea and chronic or rebound nasal congestion. Rhinitis medication for the competitive athlete must be a USOC and/or IOC–approved product and should be one that does not adversely affect performance. C
Rhinitis affects a high proportion of all athletes. In fact, the majority of all individuals, allergic and nonallergic, report nasal symptoms, especially rhinorrhea, with both outdoor (56%) and indoor (61%) exercise, but this rate is higher in patients with allergic rhinitis (72% and 70%, respectively).\(^6\) Furthermore this exercise-induced rhinitis adversely affects athletic performance in athletes with allergy (53%) and without allergy (28%).\(^6\) Among elite athletes, endurance athletes report a higher frequency of physician-diagnosed allergic rhinitis and use of antiallergic medications.\(^9\) Nasal congestion can contribute to sleep dysfunction, leading to daytime fatigue and decreased performance.\(^9\) In normal exercise situations, nasal vasoconstriction and decreased nasal resistance develop and persist for about 1 hour. Athletes, especially long-distance runners, cyclists, or triathletes, may experience a rebound nasal congestion after the initial improvement in nasal patency, which may affect peak performance.\(^6\)

Prescription of medication for the competitive athlete should be based on 2 important principles:\(^1\) no medication given to the athlete should be on any list of doping products and should be approved for use by the USOC and IOC,\(^5\) and no medication should adversely affect the athlete’s performance.\(^6\) The USOC generally observes the IOC list of banned and allowed drugs. Before a competitive athlete takes any medication prior to competition, it should be determined whether it is allowed (www.wada-ama.org). The USOC has a toll-free hotline (1-800-233-0393) to answer any questions a physician or athlete may have. Athletes and their physicians should be aware that all intranasal corticosteroids are allowed but that all decongestants are banned with the exception of topical (nasal or ophthalmologic) phenylephrine and imidazole preparations (ie, oxymetazoline and tetrahydrozoline).

Antihistamines are allowed by the USOC but may be banned by the international federation of certain sports. An adverse influence on physical performance may occur in the athlete with rhinitis treated with first-generation antihistamines, which may have undesirable sedative and anticholinergic effects. After consideration of these issues, the optimal therapy for the athlete with symptomatic allergic rhinitis consists of aggressive allergen avoidance frequently in combination with a second-generation H1-antihistamines and/or intranasal corticosteroids. Intranasal cromolyn may be useful 30 minutes before commencing a competition likely to be associated with high allergen exposure. Immunotherapy may provide help for those athletes with allergic rhinitis not responding adequately to avoidance and medication.

**Allergist/immunologist consultation and referral guidelines**

1. **Direct evidence**
   - Allergist/immunologist care for rhinitis is associated with improved QOL, compliance, and satisfaction with care.\(^6\)
2. **Diagnostic evidence**
   - Allergists/immunologists are highly trained to interpret the clinical history and allergy diagnostic test results in upper and lower airways conditions.\(^6\)
3. **Indirect evidence**
   - Avoidance: Allergists/immunologists have knowledge of aeroallergen exposures in the patient’s environment and have the expertise to provide avoidance education.\(^6\)
   - Immunotherapy: Allergy immunotherapy can be highly effective in controlling symptoms of rhinitis and may provide lasting benefit after immunotherapy is discontinued.\(^6\)

Patients with rhinitis under the care of primary care physicians often desire more education about their disease.\(^6\) Allergists/immunologists have familiarity with the wide variety of aeroallergens and have the expertise to provide avoidance education.\(^6\) They provide expertise in the interpretation of the clinical history and diagnostic studies pertaining to upper and lower airways conditions.\(^6\) Allergen immunotherapy, as offered by allergists/immunologists, effectively treats allergic rhinitis with clinical benefits that may be sustained for years after discontinuation of treatment.\(^5\)

It is recognized that whereas some patients may benefit from ongoing allergist/immunologist treatment, others may require only 1 or a few consultation visits, and/or cotreatment with the primary care physician with periodic follow-up care.

There are a variety of circumstances in which the special expertise and training of an allergist/immunologist may offer benefits to a patient with rhinitis. A detailed listing of reasons for consultation with an allergist/immunologist that may be provided as a guide for primary care physicians is detailed in Box 6.

The following outline provides the allergist/immunologist with a referral guideline and associated rationale and level of evidence that is based on recently published consultation and referral guidelines.\(^9\)

**Referral guideline**\(^8,11,30,46\)

1. Patients with rhinitis with prolonged and severe disease with a. Comorbid conditions i. Asthma ii. Recurrent sinusitis iii. Nasal polyps b. Symptoms interfere with i. QOL ii. Ability to function c. Medications are i. Ineffective ii. Associated with adverse reactions iii. Unacceptable for chronic use by patient choice, such as cost or concern with long-term side effects
2. Patients with allergic rhinitis, children, and possibly adults, being considered for allergy immunotherapy as a means of preventing the progression of allergic disease

**Referral rationale and evidence level**

1. **Direct evidence**
   - Allergist/immunologist care for rhinitis is associated with improved QOL, compliance, and satisfaction with care.\(^6\)
2. **Diagnostic evidence**
   - Allergists/immunologists are highly trained to interpret the clinical history and allergy diagnostic test results in upper and lower airways conditions.\(^6\)
3. **Indirect evidence**
   - Avoidance: Allergists/immunologists have knowledge of aeroallergen exposures in the patient’s environment and have the expertise to provide avoidance education.\(^6\)
   - Immunotherapy: Allergy immunotherapy can be highly effective in controlling symptoms of rhinitis and may provide lasting benefit after immunotherapy is discontinued.\(^6,36,7\)
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