Chronic Upper Airway Cough Syndrome Secondary to Rhinosinus Diseases (Previously Referred to as Postnasal Drip Syndrome)

ACCP Evidence-Based Clinical Practice Guidelines

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Objective: To review the literature on postnasal drip syndrome (PNDS)-induced cough and the various causes of PNDS. Hereafter, PNDS will be referred to as upper airway cough syndrome (UACS).

Methods: MEDLINE search (through May 2004) for studies published in the English language since 1980 on human subjects using the medical subject heading terms “cough,” “causes of cough,” “etiology of cough,” “postnasal drip,” “allergic rhinitis,” “vasomotor rhinitis,” and “chronic sinusitis.” Case series and prospective descriptive clinical trials were selected for review. Also, any references from these studies that were pertinent to the topic were obtained.

Results: In multiple prospective, descriptive studies of adults, PNDS due to a variety of upper respiratory conditions has been shown either singly or in combination with other conditions, to be the most common cause of chronic cough. The symptoms and signs of PNDS are nonspecific, and a definitive diagnosis of PND-induced cough cannot be made from the medical history and physical examination findings alone. Furthermore, the absence of any of the usual clinical findings does not rule out a response to treatment that is usually effective for PND-induced cough. The differential diagnosis of PNDS-induced cough includes allergic rhinitis, perennial nonallergic rhinitis, postinfectious rhinitis, bacterial sinusitis, allergic fungal sinusitis, rhinitis due to anatomic abnormalities, rhinitis due to physical or chemical irritants, occupational rhinitis, rhinitis medicamentosa, and rhinitis of pregnancy. Because of a high prevalence of upper respiratory symptoms associated with gastroesophageal reflux disease (GERD), GERD may occasionally mimic PNDS. A crucial unanswered question is whether the conditions listed above actually produce cough through a final common pathway of PND or whether, in fact, in some circumstances they cause irritation or inflammation of upper airway structures that directly stimulate cough receptors and produce cough independently of or in addition to any associated PND.

Conclusion: PNDS (ie, UACS) secondary to a variety of rhinosinus conditions is the most common cause of chronic cough. Because it is unclear whether the mechanisms of cough are the PND itself or the direct irritation or inflammation of the cough receptors located in the upper airway, the guideline committee has decided that, pending further data that address this difficult question, the committee unanimously recommends that the term upper airway cough syndrome be used in preference to postnasal drip syndrome when discussing cough associated with upper airway conditions.

(CHEST 2006; 129:63S–71S)

Key words: allergic rhinitis; bacterial sinusitis; empiric therapy; perennial nonallergic rhinitis; postinfectious rhinitis; postnasal drip syndrome; silent postnasal drip; throat clearing; upper airway cough syndrome

Abbreviations: A/D = antihistamine/decongestant; GERD = gastroesophageal reflux disease; NARES = nonallergic rhinitis with eosinophilia; PND = postnasal drip; PNDS = postnasal drip syndrome; UACS = upper airway cough syndrome
Postnasal drip (PND) is the drainage of secretions from the nose or paranasal sinuses into the pharynx. Clinically, the diagnosis of PND syndrome (PNDS) largely rests on the reporting of the patient of this sensation of having something drip down into the throat, nasal discharge, or frequent throat clearing. The presence on examination of the nasopharynges or oropharynges of mucoid or mucopurulent secretions, or cobblestoning of the mucosa also is suggestive. The problem, however, encountered when trying to diagnose PNDS is that there is no objective test for it and no way to quantify the amount of PND or to directly prove that it is causing cough. Therefore, because we are actually defining a syndrome and because no pathognomonic findings exist, the diagnosis of PNDS-induced cough is best determined by considering a combination of criteria, including symptoms, physical examination, radiographic findings, and, ultimately, the response to specific therapy. Because the improvement or resolution of cough in response to specific treatment is the pivotal factor in confirming the diagnosis of PNDS as a cause of cough, an empiric trial of therapy is both diagnostic and therapeutic. Further complicating the determination that PNDS is the etiologic factor in a patient’s chronic cough is the fact that it has been clearly demonstrated that in approximately 20% of patients with PNDS-induced cough the patients are unaware of either the presence of PND or its link to their cough. It is this lack of availability for objective testing coupled with the relatively nonspecific and sometimes insensitive findings from the medical history and physical examination that have led to the use of an empiric treatment approach for PNDS as a step in the diagnostic algorithm for chronic cough. Complicating the issue further is the crucial, unanswered question of whether the conditions listed above actually produce cough through the final common pathway of PND or whether, in fact, in some circumstances they cause irritation or inflammation of upper airway structures that directly stimulates cough receptors and produces cough independently of or in addition to any associated PND. Because this is a plausible explanation for some of the cough associated with these conditions, the cough guideline committee has decided that pending further data that address this difficult question, upper airway cough syndrome (UACS) is a term that is preferable to PNDS when discussing cough that is associated with these conditions. Consequently, from this point on, the term UACS will be used instead of PNDS.

Recommendation

1. In patients with chronic cough related to upper airway abnormalities, the committee considers the term UACS to be more accurate, and therefore it should be used instead of PNDS. Level of evidence, expert opinion; benefit, substantial; grade of recommendation, E/A

Prevalence

In multiple prospective, descriptive studies of adults, UACS due to a variety of upper respiratory conditions has been shown, either singly or in combination with other conditions, to be the most common cause of chronic cough. UACS has also been shown to be a principal cause of cough that is associated with the common cold. It follows, therefore, that, because the common cold is the most common condition afflicting mankind, UACS is the most common cause of acute cough as well.

Pathogenesis

Clinical studies have suggested that the pathogenesis of cough from UACS is due to the mechanical stimulation of the afferent limb of the cough reflex in the upper airway. One proposed mechanism is that cough receptors located in the hypopharynx or larynx are stimulated by secretions emanating from the nose and/or sinuses dripping down into these areas. There is, however, also some evidence that suggests that in patients with UACS-induced cough that the cough reflex in the upper airway is more sensitive than normal and that this increased sensitivity can be a contributing factor to the pathogenesis of cough. It may be that through some sort of direct physical or...
chemical irritation the afferent limb of the cough reflex is stimulated peripherally, and this could also lead to some sort of increased central reactivity. Cough from UACS could also conceivably be caused by aspirated secretions stimulating cough receptors in the lower respiratory tract; however, there are limited data to support this mechanism.

**Clinical Presentation**

The clinical presentation of patients with UACS, in addition to cough, commonly involves complaints (or at least an affirmative response to questioning) of a sensation of something draining into the throat, a need to clear the throat, a tickle in the throat, nasal congestion, or a nasal discharge. Patients sometimes complain of hoarseness. A medical history containing an upper respiratory illness (e.g., a cold) is often present. A history of wheeze is also common. Most patients with UACS cough will have symptoms or evidence of one or more of the following: drainage in posterior pharynx; throat clearing; nasal discharge; cobblestone appearance of the oropharyngeal mucosa; or mucus in the oropharynx. These clinical findings are relatively sensitive but are not specific. They are also found in many patients with cough due to other causes.³

A minority of patients with cough will have no upper respiratory signs or symptoms that are suggestive of PND, yet they will respond to therapy with first-generation antihistamine/decongestant (A/D) agents. The authors of one prospective study¹ interpreted this response to treatment as implying that silent PND caused the cough.

**Diagnosis**

A prospective study of chronic cough in adults⁸ demonstrated that a careful medial history including the character and timing of cough and the complications associated with cough is of little diagnostic value. This certainly is true regarding UACS cough. The symptoms and signs of UACS are nonspecific, and a definitive diagnosis cannot be made from the medical history and physical examination alone. Furthermore, the absence of any of the usual clinical findings does not rule out a response to treatment that is usually effective for UACS cough. Although this does not prove that silent PND is the cause of the cough, it at least suggests the possibility. Alternatively, it is possible that the first-generation antihistamines that have been used empirically for this purpose have a primary central antitussive effect, and therefore the apparent effectiveness of these agents in patients without findings for PNDS may be nonspecific. The fact that the resolution of cough in response to treatment with a first-generation A/D is gradual and usually takes place over a period of days to weeks argues against this interpretation. It is also possible that these agents have a direct peripheral effect on histamine levels that may stimulate cough receptors independently of any supposed PND. There is also, however, some evidence that the first-generation antihistamine diphenhydramine has some central cough-suppressing activity.⁹ Therefore, a central component cannot be entirely discounted. The key point is that it is a common pitfall in the diagnostic approach to chronic cough to fail to consider the possibility that “silent” UACS is a causative factor and to give an empiric trial of therapy for this diagnosis before looking for less common causes of chronic cough.

**Differential Diagnosis of UACS-Induced Cough**

The differential diagnosis includes allergic rhinitis, perennial nonallergic rhinitis, postinfectious rhinitis, bacterial sinusitis, allergic fungal sinusitis, rhinitis due to anatomic abnormalities, rhinitis due to physical or chemical irritants, occupational rhinitis, rhinitis medicamentosa, and rhinitis of pregnancy. Because gastroesophageal reflux disease (GERD) is frequently associated with upper respiratory symptoms, GERD may mimic a UACS-induced cough.

**Allergic Rhinitis**

Allergic rhinitis is an IgE-mediated hypersecretory state that is stimulated by specific antigens. Allergic rhinitis is extremely common; it is present in up to 20% of individuals¹⁰ and is doubtless a cause of UACS-induced cough. Allergic rhinitis can be seasonal, which often makes it easy to diagnose clinically, or perennial, a form more easily confused with other causes of chronic UACS. Not surprisingly, the allergens most commonly associated with seasonal allergic rhinitis are outdoor antigens such as grass and tree pollens, while indoor antigens such as dust mite and cockroach are more commonly associated with the perennial form. Features that are more common to allergic rhinitis than to other forms of rhinitis are sneezing and extranasal involvement, such as itching of the eyes and ears. Analogous to asthma, early-phase and late-phase nasal responses to allergens have been described, with congestion being the predominant symptom of the late phase. Allergic rhinitis is generally diagnosed when clinical suspicion leads to skin testing, which identifies pu-
tative specific antigens. However, the presence of skin reactivity to specific allergens is not proof that allergy is the etiology of the UACS. Allergy testing is likely to be positive for various pollens when there is a seasonal component to the UACS-induced cough (ie, seasonal allergic rhinitis). Allergy testing should be considered in cases of perennial rhinitis when an allergic basis is suspected but the history is less clear-cut compared to the repetitive onset of symptoms at the same time of year that is associated with seasonal allergic rhinitis due to tree or grass pollen. Testing for allergens such as animal danders, house dust mite (Dermatophagoides farinae and Dermatophagoides pteronyssinus), and indoor mold may be of particular value in these cases.

Perennial Nonallergic Rhinitis

Nonallergic perennial rhinitis can be divided into vasomotor rhinitis and nonallergic rhinitis with eosinophilia (NARES). Vasomotor rhinitis is characterized by excessive, thin, watery secretions, often in response to stimuli such as odors, changes in temperature or humidity, eating (called gustatory rhinitis), or alcohol ingestion. Patients typically describe the sudden unexpected onset of profuse rhinorrhea, or nasal congestion with or without the sensation of postnasal drip. It has been postulated that this is related to an autonomic imbalance. Increased cholinergic tone or sensitivity is suggested by the effectiveness of ipratropium bromide in controlling vasomotor symptoms. Nasal examination findings are nonspecific; watery rhinorrhea may be seen in patients whose symptoms are more secretory than congestive. Allergy testing is negative, and there is no relationship to infection, structural abnormalities, or systemic disease; it is in essence idiopathic and a diagnosis of exclusion.

NARES

NARES presents with nasal symptoms similar to those of vasomotor rhinitis, but in addition pruritus of nasal and ocular mucosae as well as excessive lacrimation are common. The diagnosis is based on the clinical syndrome coupled with the presence of eosinophils in nasal secretions in the absence of evidence for both allergy (skin testing) and asthma (methacholine challenge).

The diagnosis of perennial nonallergic rhinitis is to a significant extent a diagnosis of exclusion. Allergic rhinitis can be present year round (especially when the offending antigen is dust mites or some other persistent indoor allergen). Therefore, allergy evaluation should be considered even in cases of presumed perennial nonallergic rhinitis if the response to therapy is inadequate. Similarly, the possibility that the rhinitis is due to an environmental irritant, a nasal medication, or illicit drug abuse should be considered.

Postinfectious UACS

A history of a upper respiratory tract infection is the key to making the diagnosis of postinfectious UACS. This condition typically responds to a first-generation antihistamine along with a decongestant (eg, pseudoephedrine), just as the acute cough of the common cold does. This suggests that the chronic cough that develops following a viral respiratory tract infection may simply represent a continuation of the same process found during the acute infection. Mycoplasma, Chlamydia pneumoniae (TWAR), Chlamydia, and especially pertussis in its catarrhal stage may also result in cough of sufficient duration to fall into this category.

Bacterial Sinusitis

UACS-induced cough secondary to chronic sinusitis may or may not be associated with chronic excessive sputum production (ie, > 30 mL/d). In one study that specifically identified patients with excess sputum production (≥ 30 mL), the cause of the cough was UACS only approximately one third of the time (asthma, GERD, and bronchiectasis made up the majority of the causes). Therefore, it is important to recognize that chronic sinusitis may cause a productive cough, but also may be “clinically silent” in that the cough can be relatively or even completely nonproductive and none of the typical findings associated with acute sinusitis are likely to be present. Traditionally, the presence of sinus mucosal thickening, and in particular of opacification, and/or air-fluid levels in the presence of chronic cough has been considered to be predictive evidence for bacterial sinus infection and is the basis for antibiotic treatment. While this approach has been effective, there is evidence in the literature that mucosal thickening alone is not specific for bacterial infection. One study in particular demonstrated that < 8 mm of mucosal thickening was associated with a sterile nasal puncture in 100% of cases. A study of sinus changes in patients with chronic cough demonstrated that antibiotic therapy was needed for the resolution of cough in only 29% of cases in which the only abnormality was the presence of mucosal thickening. The most common etiologic agents for chronic bacterial sinusitis, based on specimens obtained during endoscopic surgery or needle aspiration, are Staphylococcus aureus, coagulase-negative staphylococci, anaerobic bacteria, Haemophilus influenzae, Moraxella catarrhalis, and a variety of Gram-negative bacillary organisms.
centage of anaerobes varies considerably from study to study being present in as little as \( \leq 5\% \) to \( > 50\% \). Four-view sinus radiographs may detect changes suggestive of the presence of chronic sinusitis, but this does not confirm that the chronic cough is UACS-induced, or, if it is, that the sinus disease is responsible. However, based on a favorable response to sinusitis therapy, in patients with both chronic cough and excess sputum production, routine sinus radiographs have been shown to have a positive predictive value of 81% and a negative predictive value of 95% for predicting that chronic sinusitis was responsible for the UACS-induced cough. In patients with chronic cough, the majority of whom did not have excess sputum production, the positive and negative predictive values were 57% and 100%, respectively.

Similar data do not exist for sinus CT scanning, but it would be expected that sinus CT scans would be at least as accurate as plain sinus films.

**Allergic Fungal Sinusitis**

Allergic fungal sinusitis is a rhinosinusal disease that is analogous to allergic bronchopulmonary mycosis in the lung. Diagnosis of this disease should be considered in atopic patients with chronic sinusitis with purulent expectoration or nasal drainage that is refractory to antibiotic treatment. Any one of a number of fungi can create an allergic and inflammatory response that can be symptomatic and destructive. The presentation may be gradual in onset or relatively abrupt. The key feature of the disease is a thick allergic fungal mucin that contains eosinophils and fungal elements, which may occlude the sinuses. Nasal crusting and nasal polyposis are also commonly encountered. The inflammatory response to fungal antigen (not fungal invasion) can cause bony destruction and ocular disorders. The total IgE level is commonly elevated, and the results of skin testing are generally positive for one of the dematia-cous fungi.

**Rhinitis Due to Anatomic Abnormalities**

Rhinitis due to anatomic abnormalities can be primary or secondary. A deviated nasal septum, enlarged turbinates, or a dysfunctional nasal valve can engender rhinitis. Polyps secondary to inflammatory nasal disease can become an anatomic obstruction that plays a role in the perpetuation of a rhinosinusitis syndrome (eg, obstructions leading to secondary bacterial sinusitis). Rare structural causes of rhinitis include neurofibroma, chemodectoma, squamous cell carcinoma, and inverting papilloma. Concha bullosa is a common abnormality that, in the majority of studies, does not appear to increase the incidence of symptomatic rhinosinusitis. There are some reports, however, of an increased incidence of chronic sinusitis in patients with concha bullosa, although a cause-effect relationship has not been established.

**Rhinitis Due to Physical or Chemical Irritants**

Rhinitis due to physical or chemical irritants is a category that includes multiple agents and possibly multiple mechanisms. Chronic exposure to the cold can lead to excessive nasal symptoms in individuals without any underlying nasal disorder. Various fragrances, cleaning agents, odors, smokes, fumes, and corrosive agents are all capable of causing rhinitis. When these agents are a cause of chronic rhinitis, most are encountered in an occupational context.

**Occupational Rhinitis**

Occupational rhinitis is a term that refers more to context than to pathophysiology, although it is conceptually useful. Occupational rhinitis is an episodic rhinitis that is related to the work environment. The features are sneezing, rhinorrhea, and nasal obstruction. Occupational rhinitis can be either allergic or nonallergic, and the work environment can be either the primary cause or an aggravating factor. The diagnosis should be considered when the patient’s symptoms clearly worsen following exposure to the work place. An analysis by an industrial engineer or hygienist may be required to identify the source.

**Rhinitis Medicamentosa**

Rhinitis medicamentosa is a term that is specific for the nasal congestion caused, paradoxically, by the persistent use of a drug, the immediate effect of which is the relief of nasal congestion via local vasoconstriction. The most common cause of rhinitis medicamentosa is the long-term use of topical \( \alpha \)-agonists that are specifically designated for the treatment of nasal congestion, although it can also be a side effect of nasal cocaine usage. Therefore, obtaining a careful medical history containing information on the inappropriate use of either legal nasal drug use (eg, nasal oxymetazoline hydrochloride) or illegal nasal drug use (eg, snorting cocaine) is critical to diagnosing rhinitis medicamentosa. As usage continues, the nasal mucosae become refractory; the duration of efficacy decreases, and rebound nasal edema and congestion occur. The effect may be mediated via a parasympathetic response to long-term \( \alpha \)-adrenergic stimulation and can only be terminated with the cessation of therapy with the topical vasoconstrictor. Other drugs such as \( \beta \)-blockers can cause nasal congestion or stuffiness.
**Rhinitis of Pregnancy**

There appears to be an increased incidence of nonspecific rhinitis seen during pregnancy. The key clue to this diagnosis is obviously its onset with pregnancy and its postpartum disappearance.

**Recommendation**

2. In patients with chronic cough, the diagnosis of UACS-induced cough should be determined by considering a combination of criteria, including symptoms, physical examination findings, radiographic findings, and, ultimately, the response to specific therapy. Because it is a syndrome, no pathognomonic findings exist. Level of evidence, low; benefit, substantial; grade of recommendation, B

**Treatment**

The treatment options for UACS-induced cough are somewhat dependent on the specific subcategory of disease that is present. In patients in whom the cause of the UACS-induced cough is apparent specific therapy that is directed at this condition should be instituted (see below). When a specific etiology of cough, however, is not apparent, empiric therapy for UACS should be applied using drugs that have been specifically studied and have been shown to be efficacious in the treatment of this syndrome before beginning an extensive diagnostic workup. The specifics for each category are discussed below, but in general treatment options can be classified into (1) avoidance, (2) treatment to block or reduce inflammation and secretions, (3) treatment of infection, and (4) correction of structural alterations.

**UACS Due to Allergic Rhinitis**

Although the avoidance of the offending antigens is always desirable, this is usually not completely possible. Nasal corticosteroids, antihistamines, and/or cromolyn are usually the initial drug choices for the treatment of UACS due to allergic rhinitis.10 Based on numerous controlled studies of allergic rhinitis,33–38 there is good reason to think that nasal corticosteroids, nasal cromolyn, nasal antihistamines, oral leukotriene inhibitors and oral antihistamines will be efficacious for the treatment of cough from allergic rhinitis. Nonsedating antihistamines are likely to be more effective in treating patients with this type of rhinitis than in patients with nonallergic rhinitis in whom the anticholinergic effect of the first-generation antihistamines appears to play a more important role.39 Treatment with A/D preparations is a time-tested and often effective approach to treating allergic rhinitis.57 The combination mitigates some of the effects of mast-cell degranulation via the antihistaminic effect, and causes vasoconstriction that limits the secretory response to inflammatory cytokines and may limit inflammatory cell access to areas of antigen deposition. (A central antitussive effect of antihistamines has also been suggested but appears to be minor.9) Leukotriene blockers also have been shown to decrease the symptoms of allergic rhinitis.38 It should also be noted that a metaanalysis40 has questioned whether the presumed increased sedation effect of first-generation antihistamines compared to that of the newer “nonsedating” antihistamines is substantiated by objective data or is clinically relevant.

Environmental controls to avoid the offending allergens are highly desirable, when feasible. Allergen desensitization (ie, allergen immunotherapy) may be of value over the long term, but not for immediate help. If the cough and other symptoms of allergic rhinitis are well controlled using environmental controls and intranasal therapy, allergen desensitization is not necessary.

**Vasomotor Rhinitis**

Due to its anticholinergic effect, the use of an older-generation antihistamine plus a decongestant is usually effective for treatment of this disorder. In addition, ipratropium bromide nasal spray may also be effective for this condition, but studies suggesting that it is helpful in treating the cough due to this condition have been limited to a few patients in a prospective study.3 In those few cases, therapy with ipratropium was found to be helpful when patients did not respond to the older-generation A/D preparation or was contraindicated, such as in a patient with glaucoma or symptomatic benign prostatic hypertrophy.

**Postviral Upper Respiratory Infection**

The older generation of A/D agent combinations have been shown to be consistently efficacious in one randomized, double-blind, placebo-controlled study of acute cough5 and in prospective descriptive studies of chronic cough.1–3 The combination of dexbrompheniramine maleate (6 mg bid) or azatadine maleate (1 mg bid) plus sustained-release pseudoephedrine sulfate (120 mg bid) were the treatments used in these studies. In contradistinction, newer generation, relatively nonsedating antihistamines, such as terfenadine in two studies41,42 and loratadine plus pseudoephedrine in one study,43 were found to be ineffective in treating acute cough associated with
the common cold. Based on these data, the older
generation of antihistamines should be used prefer-
entially in patients with UACS that is non-histamine-
mediated. The older-generation (ie, first-generation)
antihistamines probably work because of their anti-
cholinergic properties. In most patients, some im-
provement in cough will be seen within days to 2
weeks of the initiation of therapy.5

Severe side effects have usually not been a major
problem with the first-generation A/D preparations
in the context of treating cough; but in individual
patients they may cause difficulties requiring the
discontinuation of the therapy. In a randomized,
double-blind, placebo-controlled study5 assessing
the effect of a first-generation A/D combination
medication on cough associated with the common
cold, no patient dropped out of the study due to an
adverse occurrence from the drug. Only dry mouth
and transient dizziness were more common in the
drug group.2 Sedation is the primary side effect due
to the antihistamine, but a meta-analysis40 of the
available literature has questioned whether the se-
dation effect seen with first-generation antihista-
mines is really significantly greater than with the
newer nonsedating preparations. It is our opinion
that initiating therapy once a day at bedtime for a
few days before going to twice-daily therapy can
sometimes obviate this problem. Insomnia, difficulty
with urination (primarily in older men), jitteriness,
tachycardia or palpitations, worsening of hyperten-
sion, and increased intraocular pressures in patients
with glaucoma are all potential concerns with ther-
apy using the decongestant. Increased problems with
urination or increased intraocular pressures in glau-
coma can also occur with the use of an anticholin-
ergic medication.

Sinusitis

Although sinusitis is usually thought of as being
caused by bacterial infection, acute sinusitis (gener-
ally defined as being of no > 3 weeks duration) is
probably most often caused by acute viral rhinosi-
itus (ie, "the common cold").44 Furthermore, be-
cause it is clinically often difficult to distinguish
acute viral rhinosinusitis from acute bacterial sinus-
itis it may be reasonable to hold off on the use of
antibiotics and instead to first prescribe a first-
generation A/D preparation for 1 week.45,46 When
acute sinusitis does involve a bacterial infection, the
most common organisms are Streptococcus pneu-
omonae and H influenzae. Other organisms include
anaerobes, streptococcal species, M catarrhalis (es-
pecially in children), and S aureus.

Therapy for acute bacterial sinusitis includes anti-
biotics, intranasal corticosteroids to decrease inflam-
mation, and decongestants such as oxymetazoline
hydrochloride. While the use of intranasal cortico-
steroids has been shown to be helpful,47,48 there are
no prospective, randomized, double-blind studies
that have proven that either nasal or oral deconges-
tants are efficacious in either acute or chronic sinus-
itis. No one has investigated the efficacy of any of
these medicines on acute cough that is related to
acute sinusitis.

The treatment of chronic sinusitis is even less
clear-cut. The role of bacterial infection and the
importance of antibiotic therapy are controversial.
However, in four prospective descriptive studies1–3,13
the following initial treatment regimen has been
efficacious: a minimum of 3 weeks of treatment with
an antibiotic effective against H influenzae, mouth
anaerobes, and S pneumoniae; a minimum of 3
weeks of oral treatment with an older-generation
A/D twice per day; and 5 days of treatment with a
nasal decongestant twice per day. When cough dis-
appears with this therapy, intranasal corticosteroids
should be continued for 3 months. In patients with
documented chronic sinus infection that appears to
be refractory to medical therapy and in whom anat-
omeic obstruction is present that is thought to be
amenable to endoscopic sinus surgery, this option
should be considered.

Treatment for allergic fungal sinusitis has centered
on the surgical removal of the allergic fungal mucin
and the subsequent aeration/drainage of any in-
volved sinus.30 Unlike allergic bronchopulmonary
mycosis, steroid therapy is only suppressive. The role
of oral antifungal agents, however, though inade-
quately defined, may be of potential value and a
worthwhile option to consider prior to proceeding
with surgery.

Rhinitis Due to Physical or Chemical Irritants
(Including Occupational Exposure)

When environmental irritants are identified, the
avoidance of exposure, improved ventilation, filters,
and, in rare circumstances, the use of personal
protective devices (eg, dust/mist/fume masks with
high-efficiency particulate air filters for occupational
exposures) can be an effective part of therapy.

Rhinitis Medicamentosa

The key to therapy is the patient stopping or
weaning off the offending agent. Sometimes this can
be done one nostril at a time. The use of an A/D or
nasal corticosteroids seems reasonable, but there are
no significant data available on their efficacy.
Recommendations

3. In patients in whom the cause of the UACS-induced cough is apparent, specific therapy directed at this condition should be instituted. Level of evidence, low; benefit, substantial; grade of recommendation, B

4. For patients with chronic cough, an empiric trial of therapy for UACS should be administered because improvement or resolution of cough in response to specific treatment is the pivotal factor in confirming the diagnosis of UACS as a cause of cough. Level of evidence, low; benefit, substantial; grade of recommendation, B

5. A patient suspected of having UACS-induced cough who does not respond to empiric A/D therapy with a first-generation antihistamine should next undergo sinus imaging. Although chronic sinusitis may cause a productive cough, it may also be clinically silent, in that the cough can be relatively or even completely nonproductive and none of the typical findings associated with acute sinusitis may be present. Level of evidence, low; benefit, substantial; grade of recommendation, B

6. In patients for whom a specific etiology of chronic cough is not apparent, empiric therapy for UACS in the form of a first-generation A/D preparation should be prescribed before beginning an extensive diagnostic workup. Level of evidence, low; benefit, intermediate; grade of recommendation, C

Summary of Recommendations

1. In patients with chronic cough that is related to upper airway abnormalities, the committee considers the term UACS to be more accurate, and it should therefore be used instead of the term PNDS. Level of evidence, expert opinion; benefit, substantial; grade of recommendation, E/A

2. In patients with chronic cough, the diagnosis of UACS-induced cough should be determined by considering a combination of criteria, including symptoms, physical examination findings, radiographic findings, and, ultimately, the response to specific therapy. Because it is a syndrome, no pathognomonic findings exist. Level of evidence, low; benefit, substantial; grade of recommendation, B

3. In patients in whom the cause of the UACS-induced cough is apparent, specific therapy directed at this condition should be instituted. Level of evidence, low; benefit, substantial; grade of recommendation, B

4. For patients with chronic cough, an empiric trial of therapy for UACS should be administered because the improvement or resolution of cough in response to specific treatment is the pivotal factor in confirming the diagnosis of UACS as a cause of cough. Level of evidence, low; benefit, substantial; grade of recommendation, B

5. A patient suspected of having UACS-induced cough who does not respond to empiric A/D therapy with a first-generation antihistamine should next undergo sinus imaging. Although chronic sinusitis may cause a productive cough, it may also be clinically silent, in that the cough can be relatively or even completely nonproductive and none of the typical findings associated with acute sinusitis may be present. Level of evidence, low; benefit, substantial; grade of recommendation, B

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