Evidence for the unified airway

Diagnosis and investigations

Management of rhinitis

Case studies

The authors

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Background

Since the 1990s, our pathophysiological understanding of chronic airway conditions has progressed from simple models of obstruction and infection to a more comprehensive understanding of mucosal health, inflammation and the concept of a “unified airway”.

Recent clinical and pathophysiological research suggests that symptoms in one portion of the respiratory tract are a marker of diffuse airway inflammation, and may indicate the presence of concurrent disease elsewhere in the respiratory tract. Even when concurrent disease is not present, there is an increased likelihood it will develop subsequently over time.

Compartmentalisation of airway inflammation into anatomical areas would appear to be the exception rather than the rule. This unified airway concept represents a shift from the traditional focus on local factors such as focal infection. Patients with chronic airway conditions often initially respond to antimicrobial therapy, but an underlying inflammatory process continues, with recalcitrant symptoms or recurrence of localised infection.

Both epidemiological and pathophysiological data suggest the respiratory tract behaves as an integrated system. This includes the middle-ear mucosa, nose, paranasal sinuses, as well as the entire lower respiratory tract including the larynx. This interdependence has been the focus of clinical studies, which have explored the concept of a common inflammatory process and sought to explain the benefits of airway-wide treatment compared with localised interventions.

This phenomenon of concurrent upper and lower respiratory disease was noted as early as the 1920s, but little formal research was conducted until clinical observations in the 1980s. The model of a unified airway has its foundations in three important observations:

- Epidemiological evidence of a high prevalence of rhinitis and rhinosinusitis in people with asthma and, likewise, an increased prevalence of upper respiratory disease when the lower airway is affected.
- The underlying pathophysiology is common to all airway compartments, explaining the observed inter-relatedness of upper and lower airway disease.
- Treatment of one airway compartment results in improvement in a separate portion of the airway, or even the entire airway.

This article aims to:
- Update the reader on current clinical and research concepts of the unified airway.
- Explain the importance of treating both the upper and lower airway concurrently to achieve resolution of symptoms.
- Help the physician manage patients who develop airway-wide inflammatory changes and answer patients’ questions regarding causative relationships between the upper and lower airway.

Cont’d next page
**The unified airflow: evidence and implications**

Epidemiological evidence

Most patients with asthma have rhinitis (figure 1). Multiple studies have shown rhinitis to be present in 50-85% of subjects with asthma, with the differences between studies likely caused by differences in methodology. Self-reported symptoms may be an insensitive measure, considering that many patients with asthma may be more bothered by their asthma than rhinitis symptoms (figure 2). In a retrospective study of 1,245 subjects with asthma in the US Midwest, 52% were found to have allergic rhinitis, and 6% had non-allergic rhinitis. However, on prospective assessment with direct questioning and examination of patients with asthma:

- 100% of subjects who had allergic asthma induced by pollen had allergic rhinitis from pollen.
- 99% of subjects who had allergic asthma caused by animals had allergic rhinitis from animals.
- 95% of subjects who had allergic asthma caused by mites had allergic rhinitis from mites.

That the vast majority of patients with asthma also have rhinitis has therapeutic implications.

While the prevalence of rhinitis in patients with asthma is common, the converse association was thought to be less strong. However, recent evidence suggests that patients with rhinitis are also likely to have asthma. Large population studies have shown the incidence of cases of asthma in patients with rhinitis to be 19-38%. In two early surveys, the prevalence of rhinitis was found to be 21.3%, with symptoms in people with rhinitis was reported as 13% in a follow-up study of these individuals. These studies were based on questionnaire or video questionnaire diagnosis.

The frequency of diagnosis of asthma is much greater when other investigations are used. In a 23-year follow-up of college students using an initial skin-prick test and then questionnaires, the prevalence was 21.3%. When skin-prick testing, spirometry and methacholine challenge were used in a population of almost 3,000 Italian subjects, the prevalence of asthma was 67.2% in those with rhinitis.

In a prospective study using the Dutch rhinitis and asthma study, more than 8,000 patients with one or both of these disorders, rhinitis and asthma, were developed within the same year in 25% of patients. Asthma diagnosis rates were 75% in patients the two conditions developed within two years. Similarly, in a Finnish twin cohort of 11,000 patients there was a fourfold increased risk of developing asthma in those with rhinitis.

These correlations have been demonstrated in differing demographic and racial groups including both atopic and non-atopic individuals. Longitudinal studies document that rhinitis is almost always diagnosed before asthma. This trend, when rhinitis precedes the development of asthma, is often referred to as ‘the allergic march’.

The prevalence of atopic and allergic diseases has increased both in Australia and worldwide over recent decades. In Australia, asthma prevalence is 10.2%, or two million people, and plateauing over the past 5-10 years. The proposed explanation for the worldwide increase in allergic disease has been titled the ‘hygiene hypothesis’, which suggests a ‘cleaner environment’ (eg, less exposure to bacteria, use of vaccines, antibiotics etc.) predisposes to persistent allergic phenotype from childhood. This hypothesis is supported by a diverse range of studies and literature and is referred to as the ‘hygiene’ hypothesis. A comprehensive overview.

Anecdotally, there is a link between rhinitis, asthma and the development of chronic rhinosinusitis (CRS). While ‘cause and effect’ theories have been the focus of research, but the relationship remains unclear. There is good data to support higher incidence of allergic rhinitis in CRS and these patients also have worse CT findings along with higher IL-5 levels and more eosinophilic mucosa.

However, studies of rhinitis and allergy in CRS seem to indicate that CRS is an inflammatory condition resulting from interacting immunological, microbial and mucociliary factors (figure 3).

The pathological interaction of intrinsic mucosal inflammation, microbial flora and mucociliary function results in ongoing inflammatory changes.

Current topical therapies can affect all three of these processes:

1. The ability to substitute for loss of mucociliary clearance.
2. The ability to affect mast cell and eosinophil mediator release.
3. The ability to affect eosinophil and mast cell recruitment.

Rhinitis and asthma

Both conditions are chronic and recurrent diseases. The hallmark of rhinitis is sneezing, nasal congestion and rhinorrhea. The hallmark of asthma is episodic wheezing, chest tightness and coughing. The hallmark of allergic rhinitis is sneezing, nasal congestion and rhinorrhea. The hallmark of allergic asthma is wheezing, chest tightness and coughing. The hallmark of allergic rhinitis is sneezing, nasal congestion and rhinorrhea. The hallmark of allergic asthma is wheezing, chest tightness and coughing. The hallmark of allergic rhinitis is sneezing, nasal congestion and rhinorrhea. The hallmark of allergic asthma is wheezing, chest tightness and coughing.

**Figure 1: Proportions of clinically overt rhinitis and asthma. The severity of symptoms is measured by scoring the severity of cases as mild, moderate or severe.**

**Figure 2: Proportions of clinically overt rhinitis and asthma. The severity of symptoms is measured by scoring the severity of cases as mild, moderate or severe.**

**Figure 3: The triangle of pathological mechanisms causing the unregulated pro-inflammatory and pro-remodelling responses.**
Interleukin-5 (IL-5) is a cytokine and bone morphogenic protein (BMP) that is produced by T helper 2 (Th2) cells and plays a pivotal role in the pathogenesis of allergic asthma. IL-5 is a key mediator, and clinically, a selective IL-5 receptor antagonist has recently been approved for the treatment of severe asthma. IL-5 is involved in the disease process. It acts on the eosinophilic granulocyte and increases the production of eosinophils in the respiratory mucosa, thus contributing to the development of eosinophilic asthma. IL-5 also promotes the survival and differentiation of eosinophils in the airways, leading to increased eosinophil accumulation and inflammation in the airways. This cytokine release is associated with the clinical manifestations of asthma, such as airway hyperreactivity and increased eosinophil infiltration. The selective inhibition of IL-5 receptors provides a potential therapeutic target for the treatment of allergic asthma.

Skin-prick tests (SPTs) are the most commonly used methods in the diagnosis of allergic diseases. The SPT technique involves the placement of a small amount of allergen extract on the skin, usually on the forearms, and then puncturing the skin with a needle to allow the allergen to penetrate. The skin is then observed for a reaction, typically a red, itchy, or swelling response, which indicates the presence of an allergic reaction. SPTs are useful in identifying specific allergens and are commonly performed in conjunction with other diagnostic tests, such as total serum IgE levels or specific IgE antibody levels.

In the upper airway, nasal polyps are common in patients with chronic rhinosinusitis and asthma. Although popular in patients with asthma, nasal polyps are not benign neoplastic 'growths', in comparison with most colonic polyps. Although surgery is used to remove them, they are not easily removed. Swelling of the sinus mucosa due to inflammatory infiltrate.

Diagnostic and Investigations

There are multiple approaches to the diagnosis of allergic disease, and many of these approaches are based on the detection of specific IgE antibodies. Skin-prick tests (SPTs) are the most commonly used methods in the diagnosis of allergic diseases. The SPT technique involves the placement of a small amount of allergen extract on the skin, usually on the forearms, and then puncturing the skin with a needle to allow the allergen to penetrate. The skin is then observed for a reaction, typically a red, itchy, or swelling response, which indicates the presence of an allergic reaction. SPTs are useful in identifying specific allergens and are commonly performed in conjunction with other diagnostic tests, such as total serum IgE levels or specific IgE antibody levels.

Figure 5: Epicutaneous antigen testing or skin-prick testing.

Figure 6: Lower airway challenge.

This group of patients often presents a difficult clinical management problem, with poor control of upper and lower airway symptoms. Almost 70% of patients with asthma also have rhinitis. Nasal and bronchial symptoms, nasal and peripheral blood eosinophilia, and bronchial response to methacholine challenge were reduced in a double-blind, randomised placebo-controlled trial in patients with allergic rhinitis and bronchial asthma. Patients with allergic rhinitis and bronchial asthma who receive inhaled steroids have a better asthma control.

Figure 7: Upper airway challenge testing. A: The use of rhinometry and acoustic rhinometry is critical for effective nasal challenge testing. B: Lysine asparagine is administered into the nasal airway with an insufflator. C: The corresponding resistance and cross-sectional airway measurements, with symptom reporting, determine the response.

Figure 4: Nasal polyposis (A) are really chronic inflammatory changes, with sinus mucosa that results in slow distension and swelling of the sinus mucosa due to inflammatory infiltrate. They are not benign neoplastic 'growths', in comparison with most colonic polyps. Although surgery is used to remove them, they are not easily removed. Swelling of the sinus mucosa due to inflammatory infiltrate. This hypothesis. Scintigraphy does not demonstrate pulmonary aspiration after placement of radionuclide tracer in the maxillary sinus in rhinosinusitis. Although there is obvious anatomical continuity between the upper and lower airways, it is unlikely that direct transfer of inflammatory or infected secretions occurs.

Treatment-based evidence

Double-blind placebo-controlled trials have shown that rhinitis management with intranasal steroid improves asthma. A Cochrane review demonstrated a trend to overall benefit, even with heterogeneous studies. Much attention has been placed on managing sino-nasal disease to alter outcomes in patients with asthma. Nasal and bronchial symptoms, nasal and peripheral blood eosinophilia, and bronchial response to methacholine challenge were reduced in a double-blind, randomised placebo-controlled trial in patients with allergic rhinitis and bronchial asthma. Patients with allergic rhinitis and bronchial asthma who receive inhaled steroids have a better asthma control.
Management of rhinitis

Principles of management

There are four broad groups of therapeutic modalities — antigen avoidance, pharmacotherapy, surgery and immunotherapy. Too often these modalities are used sequentially when evidence suggests that a multimodal approach is best for most patients (table 1).

For example, a patient with rhinitis (without CRS) with persistent nasal congestion after trialling intranasal steroids should not be directed to antihistamines or further antigen avoidance. Immunotherapy is long term and not effective for nasal obstruction. However, an endoscopic turbinectomy will provide immediate relief from the obstruction. Understanding severity scales is useful to ascertain a reasonable starting point for patient treatment (tables 2 and 3). For example, in persistent asthma or rhinitis a topical steroid should always be used as first-line management.

Antigen avoidance

Allergic avoidance has a limited impact on real-life disease course. Although removal of people with allergic rhinitis to places such as high-altitude sanatoria can significantly reduce symptoms, this is of little practical benefit. Significant focus has been placed on altering the indoor environment to improve control. This is a multimillion dollar industry, with a variety of interventions. Allergic avoidance for allergic rhinitis can significantly improve control. This includes placing on altering the indoor environment to improve control. This is a multimillion dollar industry, with a variety of interventions. Allergic avoidance for allergic rhinitis can significantly improve control. This includes air filters commercially available, reducing indoor dust mite exposure and removal of pets from the home. For example, air filters are beneficial, but the evidence suggests that a multimodal approach is best for most patients (table 1).

Pharmacotherapy

Intranasal steroids (INS), antihistamines and nasal saline form the basis of pharmacotherapy. The early-phase response is primarily mediated by histamine, and acute symptoms dominated by sneezing and itching, with rhinorrhea and congestion also present but often more delayed in their presentation. The late-phase response generally occurs more than two hours from antigen exposure and is mediated by T-cell cytokines. These symptoms are more prolonged than those triggered by histamine alone. Nasal congestion and postnasal discharge are common, while rhinorrhea and pruritus are less frequent.

Antihistamines

Non-lipophilic second-generation oral antihistamines (eg, loratidine, cetirizine) are the most commonly available medications to manage the early symptoms. They do not cross the blood-brain barrier and have minimal sedative effects. Not only do these agents compete with histamine in binding to the H1 receptor, they also change the three-dimensional configuration of the receptor, decreasing its affinity for histamine. This deforming of the receptor has been termed reverse agonism. Although oral antihistamines are usually prescribed for itching and sneezing, they are less effective than topical steroids. However, when multi-end organ symptoms occur (pharynx, mouth, conjunctivae and upper or lower airway), they may be more convenient than topical corticosteroids delivered to multiple sites. The topical antihistamine azelastine is currently available for the treatment of allergic rhinitis and conjunctivitis, and an excellent solution for rapid short-term relief in intermittent disease, especially in socially awkward exacerbations.

Intranasal corticosteroids

Newer INS, mometasone furoate (Nasonex) and fluticasone furoate (Avasyn), generally have bioavailabilities less than 1% (reducing the risk of systemic side effects), and a greater affinity for the glucocorticoid receptor. Corticosteroids affect a large number of cellular and humoral mediators and influence both early and late phases of antigen exposure. Similar to correct inhaler technique, effective use of intranasal sprays is essential for optimal benefit (figure 8).

Tests for confirming IgE-mediated allergy

Skin tests

Intactaneous (intradermal)
Intradermal single-dilution test
Intradermal dilutional test

Epicutaneous (prick/puncture) tests:
• Multiple antigen
• Single antigen
End-organ challenge (provocation) tests
Nasal provocation
Bronchial provocation
Conjunctival provocation

Sero biological tests

Radiosensitive labelling (rarely used), eg, RAST
Enzyme-linked labelling, eg, ELISA

Table 1: Targeted strategies for symptoms (evidence levels)*

<table>
<thead>
<tr>
<th>Treatment strategies</th>
<th>Grade</th>
<th>Symptoms</th>
<th>Evidence level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal obstruction</td>
<td>Medical</td>
<td>Topical corticosteroids (1a)</td>
<td>Surgical</td>
</tr>
<tr>
<td>Rhinorrhea</td>
<td>Topical corticosteroids (1a)</td>
<td>Ipratropium (3)</td>
<td>Leukotriene inhibitors (5)</td>
</tr>
<tr>
<td>Sneezing</td>
<td>Chromones (1a)</td>
<td>Antihistamine (1a)</td>
<td>Intranasal capisole (4)</td>
</tr>
<tr>
<td>Postnasal discharge</td>
<td>Saline irrigation (2a)</td>
<td>Topical corticosteroids (higher dose) (5)</td>
<td>Adenoidectomy (4)</td>
</tr>
<tr>
<td>Sinusitis</td>
<td>Corticosteroids</td>
<td>Culture directed antimicrobial therapy (4)</td>
<td>Endoscopic septal surgery with postural topical therapies (1d)</td>
</tr>
</tbody>
</table>

*The benefit of interventions depends on specific symptoms. Most patients benefit from a multi-faceted concurrent therapeutic approach to symptom control.

Table 2: Severity grading for asthma

<table>
<thead>
<tr>
<th>Classification</th>
<th>Pathology</th>
<th>Criteria</th>
<th>Grading</th>
<th>Symptoms</th>
<th>Symptoms</th>
<th>PEF or FEV1 % of normal (variability post pre-bronchodilator)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergic</td>
<td>IF clinical history AND skin prick test (or IgE evaluation) supports antigen exposure induced symptoms</td>
<td></td>
<td></td>
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<tr>
<td>Non-allergic</td>
<td>A collective group of conditions that involve non-allergic mechanisms, including: vasomotor, intrinsic, neurogenic and occupational rhinitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration</td>
<td>Four or more days a week OR Fewer than four weeks' duration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent</td>
<td>Four or more days a week AND Four or more weeks' duration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td>Mild or moderate-severe Abnormal sleep or impairment of sport, leisure, work or trouble-free symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PEF = peak expiratory flow</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Classification and severity grading for rhinitis

<table>
<thead>
<tr>
<th>Grading</th>
<th>Symptoms</th>
<th>Symptoms</th>
<th>PEF or FEV1 % of normal (variability post pre-bronchodilator)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent</td>
<td>Less than once a week Asymptomatic and normal PEF between attacks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild persistent</td>
<td>More than once a week and less than once a day Attacks affect activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate persistent</td>
<td>Daily Attacks affect activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe persistent</td>
<td>Continuous Limitation of physical activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PEF = peak expiratory flow</td>
<td></td>
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</tbody>
</table>

Figure 8: Intranasal corticosteroid technique. Sniffing the steroid on initial application should prolong the patency of the nasal airway (C and D). A and B show the preoperative and postoperative appearance of the nasal airway, respectively. C and D show the postoperative appearance of the nasal airway with a turbinate reduction (right A and left B). Figure 9: Turbinectomy hypertrophy and surgical reduction. Severe preoperative hypertrophy of the inferior turbinate (A and B). Postoperative appearance of the nasal airway after turbinate reduction (C and D). Table 2 continued on next page.
**Case study**

COLLEEN, 56, has had upper airway problems since age 11, when recurrent epistaxes were treated with cautery. From age 17 she has had recurrent allergic rhinitis, treated with the older oral antihistamine, but the patient still has troublesome hayfever. Colleen found it difficult to manage her symptoms and was referred to a specialist for further evaluation.

After an episode of anaphylaxis after taking aspirin in 1987, she saw an allergist who found, on SPT, multiple allergens to which Colleen was sensitised, particularly to dust mite, cat dander, and grass pollen. Colleen was advised to avoid contact with these allergens, but her symptoms persisted.

**Conclusion**

Our current understanding of airway disease suggests that both upper and lower airway disease exist concurrently. It is likely that both are manifestations of a single inflammation process that extends along the respiratory tract and also involves a systemic inflammatory response.

Clinically this means that even if a patient presents with symptoms relating to one site, it is important to elicit symptoms and signs from both upper and lower airways with appropriate supportive investigations.

**Summary**

- **Compartmentalisation** of chronic inflammatory disease is an important part of the pathophysiology of the upper and lower airways.
- **Assistance of inflammatory processes**, as a causal factor in both upper and lower airway disease, is unlikely in a medically tractable condition.
- **Good evidence** suggests the same inflammatory process occurs in both the upper and lower airways.
- **Maxifaceted concurrent therapy** is key to obtaining rapid symptom control.
- **Surgery** such as turbinectomy reduction should be used as an adjunct in therapy, and not as a treatment of last resort.

**Multifaceted concurrent therapy** started early may alter the course of rhinitis and asthma, hence "allergic march."
from page 32

The Medihaler Eps prescribed at that time very useful in relieving his symptoms. She has since moved from a rural to an urban area and the frequency of attacks has decreased. However, she can get episodes of facial swelling and severe rhinitis with exposure to tobacco smoke, latex, strong perfumes and dust. Epinephrine did help but now it takes 1-2 days to get over an episode. A recent ENT consultation found no polyps and the cautery to septal nasal mucosa gave only temporary relief to the epistaxes.

Questions for the author
Would there be any advantage in repeating the skin tests with a view to immunotherapy? What of the newer treatments would be most suitable for relieving his symptoms?

If there is a convincing clinical history to accompany the patient’s report of rhinitis, would you test for any specific antigens? Could this help to narrow down the diagnosis and provide an obvious target for therapy?

1. Which THREE statements are correct?
   a) Epidemiological and pathophysiological data suggest that the upper and lower respiratory tract are really two separate compartments.
   b) Most patients with allergic asthma induced by pollens, animals or insects also have allergic rhinitis from those causes.
   c) The allergic march refers to the development of widespread airways disease as a result of the persistence of an allergic phenotype, and thus is not recommended for the short term and is not of long benefit.
   d) Topical azelastine is unlikely to provide an effective delivery mechanism.
   e) A nasal challenge test with an allergen will confirm the diagnosis of allergic rhinitis.

2. Which TWO statements are correct?
   a) Nasal challenge tests have a greater overlap with allergen provocation in CRS than with CRS.
   b) SPT is a rapid and convenient way of testing for allergen sensitisation.
   c) The ‘allergic march’ refers to the observation that patients with rhinitis are more likely to develop asthma.
   d) Subcutaneous immunotherapy early in the course of allergic disease may prevent the development of widespread airways disease.
   e) Nasal polyps have a neoplastic aetiology.

3. Which THREE statements are correct?
   a) Patients with CRS who have mixed inflammatory indices including eosinophils, nasal polyps, and IgE have a significantly poorer quality of life than patients with non-polypoid disease.
   b) Nasal polyps, by definition, have a homogeneous eosinophilic content.
   c) Both the lower and upper respiratory tracts may be affected by inhalants.
   d) When an allergic patient has sinus symptoms, biopsy of the nasal mucosa gives only temporary relief to the epistaxes.
   e) Frontal rhinosinusitis (FRS) patients have significant sinus symptoms and signs with exposure to tobacco smoke, latex, strong perfumes and dust.

How to Treat Quiz
Rhinitis and the unified airway — 2 July 2010

1. Which THREE statements are correct?
   a) Antigen placed onto the bronchial mucosa using a bronchoscope results in subsequent inflammation.
   b) Nasal stimulation with allergen produces an inflammatory bronchial response on biopsy.
   c) There are good correlations between asthma and severe eosinophil numbers in sputum from the lower airway and in the nasal lavage.
   d) Aspiration of infected or inflammatory sinonasal secretions is a likely explanation of the association between rhinosinusitis and asthma.
   e) Allergen challenge of the airway is a routine part of lung function tests.

2. Which THREE statements are correct?
   a) Nasal challenge tests have a greater overlap with results normal patients and those with rhinitis, compared with lower airway disease.
   b) Testing with intranasal larynx can be used to diagnose femoral triad (rhinitis with nasal polyps and asthma).
   c) Oral aspirin desensitisation in CRS may trigger asthma and cause GI side effects.
   d) Intranasal larynx aspirin desensitisation is inefficient in people with aspirin-related asthma.
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6. Which THREE statements are correct?
   a) Mast cells are the key effector cells in type 1 hypersensitivity and reside in the submucosal layer of the respiratory tract and the skin.
   b) SPT is a rapid and convenient way of accessing the mast cell population to detect specific IgE responses.
   c) Challenge tests for the lower airways use direct agents (histamine or methacholine) or indirect stimuli (exercise, hypertonic saline or mannitol).
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   e) Intranasal larynx aspirin desensitisation is inefficient in people with aspirin-related asthma.

8. Which TWO statements are correct?
   a) Antigen avoidance, pharmacotherapy, surgery and immunotherapy are best used sequentially.
   b) Antigen avoidance interventions have little impact on the disease course of CRS or asthma.
   c) Specialised pillows, mattress and quilts reduce dust mitre allergen exposure, and result in clinical benefit in adults.
   d) The early phase allergic response is primarily mediated by histamine and characterised by sneezing and itching.
   e) Intranasal larynx aspirin desensitisation is inefficient in people with aspirin-related asthma.

9. Which THREE statements are correct?
   a) In a neurologically intact patient the lower airways are protected by a strong cough reflex, unlike skin.
   b) The ‘allergic march’ refers to the observation that patients with rhinitis are more likely to develop asthma.
   c) There is no role for sinus surgery in the treatment of sinusitis.

10. Which THREE statements are correct?
   a) The ‘allergic march’ refers to the observation that patients with rhinitis are more likely to develop asthma.
   b) Oral antihistamines are more effective than topical corticosteroids for the treatment of itching and sneezing.
   c) Oral antihistamines may be more effective than topical corticosteroids delivered to multiple sites.

CDP QUIZ UPDATE
This RACGP requires that a brief GP evaluation form be completed with every quiz to obtain category 2 CPD or PDP points for the 2008-10 triennium. You can complete this online along with the quiz at www.australiandoctor.com.au. Because this is a requirement, we are no longer able to accept the quiz by post or fax. However, we have included the quiz questions here for those who like to prepare the answers before completing the quiz online.

参考文献

下一步
Rhinitis and the unified airway

INSTRUCTIONS
Complete this quiz online and fill in the GP evaluation form to earn 2 CPD or PDP points. We no longer accept quizzes by post or fax.

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

ONLINE ONLY

HOW TO TREAT
Rhinitis and the unified airway — 2 July 2010

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   b) Antigen avoidance interventions have little impact on the disease course of CRS or asthma.
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   d) The early phase allergic response is primarily mediated by histamine and characterised by sneezing and itching.

9. Which THREE statements are correct?
   a) In a neurologically intact patient the lower airways are protected by a strong cough reflex, unlike skin.
   b) The ‘allergic march’ refers to the observation that patients with rhinitis are more likely to develop asthma.
   c) There is no role for sinus surgery in the treatment of sinusitis.