Asthma kills about 5,000 Americans per year, representing a steady increase over the past several decades, according to a 1997 estimate of the National Heart, Lung & Blood Institute (NHLBI). Studies of the cause and prevention of death from asthma indicate that up to 90% of these deaths are preventable. Most asthma deaths occur outside the hospital and are precipitated by inadequate assessment of the severity of airway obstruction by the patient or physician, coupled with inadequate therapy. Allergic rhinitis, while it seldom results in death, can cause serious complications to the eustachian tubes, the nose, and the sinuses. Patients with allergic rhinitis also have a threefold increase in the risk of asthma. Similarly, an allergic component can be demonstrated in about 50% of asthmatic patients.

Both asthma and allergic rhinitis are inflammatory disorders of the airways. Allergic rhinitis can be seasonal or perennial and centers in the nose; asthma affects the lungs. Because both conditions are related from a pathophysiological standpoint, their pharmacotherapy also has the same rationale: To control the inflammatory processes that create the symptoms.

This lesson is intended to define asthma and allergic rhinitis as inflammatory diseases for which anti-inflammatory medications are the cornerstones of control. For asthma, therapeutic agents offer either quick relief or long-term control; however, drugs with anti-inflammatory activity are preferred for long-term asthma control.

Quick relief can be obtained...
through short-acting beta agonists. Regular use of beta agonists does not worsen asthma; their use can help to assess the appropriateness of long-term therapy.

In allergic rhinitis, intranasal steroids are the most potent medications that can inhibit the inflammatory response.

Epidemiology
About 5% of the American population—or 15 million persons—has asthma. Asthma is the most common chronic disease of children, affecting one in every seven. Asthma can be triggered by a wide range of agents and events, from aspirin to anxiety (Table 1). Allergic rhinitis is one of the most common medical disorders found in humans, affecting one in five Americans, and it ranks as the sixth most prevalent chronic condition in the United States.

Pathophysiology of asthma
The airways can balk at various physical, chemical, and pharmacological stimuli as they try to remove the offending substance by closing the route needed by oxygen to get to the blood. This is called hyperresponsiveness. Besides blocking the entrance of air, hyperresponsiveness can also result in damage to the lining of the airways: Remodeling, subepithelial fibrosis, and collagen disposition have recently been implicated as complications of hyperresponsiveness. When asthmatic lungs are examined at autopsy, they are observed as hyperinflated because of air trapping from widespread mucus plugging. The histologic examination is characterized by (1) marked hypertrophy and hyperplasia of the airway smooth muscle, (2) increased airway wall thickness with an exudative inflammatory reaction, epithelial desquamation, and edema, and (3) mucus gland hypertrophy and mucus hypersecretion.

Inflammation of the airways results in the release of mediators that cause symptoms. Mast cells, found throughout the respiratory tract, release mediators when they are challenged with an allergen. These mediators include:
- Histamine that causes vascular and mucosal permeability
- Eosinophil and neutrophil chemotactic factors that cause an influx of inflammatory cells
- Leukotrienes that cause mucus secretion and vascular permeability
- Prostaglandins that help constrict the bronchial muscles
- Platelet-activating factors that cause airway permeability, mucus secretion, and further vascular permeability.

Pathophysiology of allergic rhinitis
Allergic reactions in the nose are mediated by similar antigen-antibody responses that interact with specific IgE molecules bound to nasal mast cells and basophils. In patients with allergic rhinitis, these cells increase both in number and in reactivity. Thus, when airborne allergens enter the nose, they push the lymphocytes into action to produce antigen-specific IgE, and when the person is reexposed to the allergen, the IgE bound to the mast cells triggers a release of inflammatory mediators. These mediators include histamine to cause the pruritus and vascular permeability, leukotrienes to contract the smooth muscle and cause mucus secretion, and thromboxanes to cause the smooth muscle to spasm. Symptomatology of allergic rhinitis includes rhinorrhea; conjunctivitis; sneezing; nasal congestion; and itchy eyes, nose, ears, or palate.

Rationale for asthma therapy
Asthma used to be viewed as a disease in which lung function was normal between flare-ups. For this reason, therapy was minimized or excluded completely between problem times. Now we know that airway inflammation, constant and insidious, is the pivotal
feature of asthma, and the degree of inflammation influences bronchial hyperresponsiveness to the factors that trigger asthma. This shift in our understanding of asthma has resulted in a treatment strategy shift with an emphasis on chronic therapy for long-term exacerbations.

The first step in designing the patient’s step therapy treatment plan is to assess the patient’s disease severity. This is accomplished through evaluation of symptoms and medical history, current treatment and response, clinical examination, and, when possible, objective measurements of lung function to establish a baseline. Medication plans should address and accommodate variability in disease severity and take into account a patient’s circumstances, including economic status.

Because asthma has a wide range of symptoms that can occur daily, or intervals that span months or years, a more important factor is severity of symptoms. The National Asthma Education & Prevention Program (NAEPP), sponsored by the NHLBI, has provided a means to classify asthma (Table 2).

### Long-term maintenance therapy

**Anti-inflammatory agents.** These agents include inhaled and oral corticosteroids. Perhaps due to their effects on multiple inflammatory processes, corticosteroids are the most potent and consistently effective long-term control medication. Clinically, inhaled steroids are the initial drugs of choice and include beclomethasone dipropionate, triamcinolone acetonide, fluticasone, budesonide, and flunisolide. These medications reduce symptom severity, improve peak flow measurements and other measures of lung function, prevent exacerbations, and possibly prevent lung remodeling. For severe persistent asthma that cannot be controlled with inhaled corticosteroids, oral preparations are used, such as prednisone. Cromolyn sodium and nedocromil sodium have similar anti-inflammatory properties; they appear to modulate mast-cell mediator release and eosinophil recruitment. Both reduce asthma symptoms and improve lung function.

**Leukotriene antagonists and formation inhibitors.** The newer drugs include montelukast and zafirlukast, which block the action of leukotrienes, and zileuton, which prevents leukotriene formation. They have been shown to improve lung function, improve symptoms, and decrease the need for quick-relief medications. A report that was presented at the annual meeting of the American Academy of Allergy, Asthma & Immunology (AAAAI) stated that montelukast, previously recommended for use in asthmatics over age six years, had a safety profile similar to that of placebo in study subjects aged two to five years. Another report showed that montelukast was preferred to inhaled beclomethasone in asthmatic children aged six to 11 years. This gives parents and children another alternative for long-term asthma control without the use of an inhaler, a drug delivery system that is not ideal for all children.

**Long-acting beta, agonists.** One of the theories behind asthma is that the disease represents a beta2 receptor blockade that results in bronchial hyperreactivity. Inhaled salmeterol or oral sustained-released albuterol relaxes airway smooth muscle by stimulating beta2 receptors to prevent bronchoconstriction. Long-acting beta2 agonists have a duration of 12 hours. These drugs are not used for acute exacerbations but rather as an adjunct to anti-inflammatory medications, particularly for control of nocturnal asthma symptoms.

**Methylxanthines.** Sustained-release theophylline is the...
principally used long-acting methylxanthine, and it is used primarily as an adjuvant therapy, particularly for controlling nighttime symptoms. Theophylline has a complex pharmacokinetic and adverse-event profile that is important to understand if the drug is to be safely and effectively employed. For example, patients in the one- to nine-year-old age range can have a theophylline clearance rate that varies by two- to threefold. Thus, no patient should be treated with theophylline without routine serum monitoring. Also important to note, theophylline has not been shown to add to the efficacy of aerosolized beta₂ agonists in acute severe exacerbations of asthma and thus is no longer recommended. However, in the outpatient setting, theophylline, if taken regularly, can reduce asthma symptoms, reduce the amount of steroid needed in steroid-dependent asthmatics, and reduce the symptoms of nocturnal asthma if taken as a sustained-release preparation.

**Quick-relief therapy**

**Inhaled short-acting beta₂ agonists.** Short-acting beta₂ agonists produce a treatment effect within five to 15 minutes and are thus the treatment of choice for acute asthma symptoms. The aerosol administration of these agents provides a greater degree of protection against provocations that induce bronchospasm, such as exercise and allergen challenges, than does systemic administration. A controversy about these agents began when one report noted a worsening of asthma from regular use of short-acting beta₂ agonists. Subsequent double-blind, placebo-controlled trials in large numbers of patients have not confirmed this claim. However, regular use of these agents offers little benefit over “as needed” use, and it is therefore not recommended.

**Inhaled anticholinergics.** Ipratropium bromide is a derivative of atropine, without its adverse reactions. This drug may be used with inhaled beta₂ agonists in severe asthma and is manufactured as a combination therapy with beta₂ agonists. The role of these agents in asthma is limited because they are not as potent as beta₂ agonists and they attenuate but do not block allergen- or exercise-induced asthma in a dose-dependent fashion. Ipratropium bromide is indicated only as adjunctive therapy in acute severe asthma not completely responsive to beta₂ agonists alone.

**Oral corticosteroids.** Although oral corticosteroids are not quick-relief medications, they are often administered in short “bursts” together with inhaled beta₂ agonists for acute exacerbations. Burst oral corticosteroids (60 mg of prednisone for four days) can be self-administered by patients who experience more severe asthma attacks in the home. The patient should be instructed to fill the prescription and keep the medication in a place that is readily accessible in case of an asthma attack.

*Rationale for allergy therapy*

The therapeutic goal for people with allergic rhinitis is to minimize or prevent symptoms. Ideally, this goal should be reached without adverse medication events or a balance of tolerable adverse effects with an amelioration of symptoms. Once the causative allergens and specific symptoms are identified, management of allergic rhinitis consists of allergen avoidance, pharmacotherapy for symptom prevention and/or treatment, and specific immunotherapy.

Immunotherapy is a slow, gradual process of injecting the offending antigen, in increasing doses, in the hopes of building immunity to the antigen. Such therapy is expensive and possibly life-threatening, such as when anaphylactic reactions to the antigen occur. Allergic rhinitis can be treated effectively with a variety of nasal sprays that have anti-inflammatory, antihistaminic, anticholinergic, or mast-cell stabilizing properties. They can be used in combination with each other, with oral decongestants or antihistamines, as needed or recommended.

**Anti-inflammatory sprays**

These sprays are actually corticosteroids that are used intranasally for their local effect. They are the most potent agents available to inhibit the inflammatory response associated with allergic rhinitis.

**Triamcinolone acetonide.** Triamcinolone acetonide is a synthetic glucocorticoid that has been formulated as both an aerosol and an aqueous metered-dose pump spray for nasal inhalation in the treatment of allergic rhinitis. Nasally administered triamcinolone acetonide is not significantly absorbed into the systemic circulation and does not suppress hypothalamic-pituitary-adrenal (HPA) axis function at therapeutic dosages. Clinical trials with either formulation have shown that once-daily triamcinolone acetonide 110 to 220 mcg reduces symptoms of allergic rhinitis within the first day of administration. Once symptoms are under control, the dosage of aqueous triamcinolone acetonide may be reduced from 220 mcg to 110 mcg/day without loss of effect.

**Flunisolide.** This medication can take from three days to three weeks to show an effect. A few years ago, the formulation of this medication was changed because of complaints of nasal burning and stinging. In fact, the
new formulation produced significantly fewer adverse effects than the original formulation \( (P = 0.006) \) with subsequent improved patient compliance and symptom control. Once the patient obtains the desired clinical effect on this medication, instruct him or her to reduce the maintenance dose to the smallest amount necessary to control symptoms.

Dexamethasone sodium phosphate. One of the original corticosteroid nasal products, dexamethasone has been associated with iatrogenic Cushing’s syndrome, a rare but present danger among those who use the medication. One review study reports five such cases, although four of them occurred in countries where this medication is available over the

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Classification of asthma severity: Clinical features prior to treatment</th>
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<tbody>
<tr>
<td><strong>STEP 1</strong></td>
<td>Mild intermittent</td>
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| Symptoms | • Symptoms \( \leq 2 \) times a week  
• Asymptomatic and normal PEF between exacerbations  
• Brief exacerbations (from a few hours to a few days); intensity may vary  
• Nocturnal awakenings \( \leq 2 \) times a month |
| Lung function* | FEV1/PEF \( \geq 80\% 
Predicted PEF variability \( < 20\% 
|
| **STEP 2** | Mild persistent |
| Symptoms | • Symptoms > two times a week but < one time a day  
• Exacerbations may affect activity  
• Nocturnal awakenings > two times a month |
| Lung function* | FEV1/PEF \( \geq 80\% 
Predicted PEF variability \( 20\%-30\% 
|
| **STEP 3** | Moderate persistent |
| Symptoms | • Daily symptoms  
• Daily use of inhaled short-acting beta\(_2\) agonist  
• Exacerbations affect activity  
• Exacerbations \( \geq 2 \) times a week; may last days  
• Nocturnal awakenings > one time a week |
| Lung function* | FEV1/PEF \( >60\% - <80\% 
Predicted PEF variability \( < 30\% 
|
| **STEP 4** | Severe persistent |
| Symptoms | • Continual symptoms  
• Limited physical activity  
• Frequent exacerbations  
• Nocturnal awakenings frequent |
| Lung function* | FEV1/PEF \( \leq 60\% 
Predicted PEF variability \( > 30\% 
|

FEV1: Forced expiratory volume in one second; PEF: Peak expiratory flow

*The presence of one of the features of severity is sufficient to place a patient in that category. An individual should be assigned to the most severe grade in which any feature occurs. The characteristics noted in this figure are general and may overlap because asthma is highly variable. Furthermore, an individual’s classification may change over time. Patients at any level of severity can have mild, moderate, or severe exacerbations. Some patients with intermittent asthma experience severe and life-threatening exacerbations separated by long periods of normal lung function and no symptoms.

Source: NHLBI, 1997
counter. Whereas the newer intranasal steroids are not absorbed as readily through the nasal and gastrointestinal mucosa and are inactivated in the liver, dexamethasone has a high systemic bioavailability. Treatment consists of the discontinuation of the intranasal steroid preparation and tapering doses of prednisone to cover the secondary adrenal insufficiency until the axis recovers.

**Fluticasone propionate.** Intranasal fluticasone propionate has shown clinical efficacy similar to that of other intranasal corticosteroids, including beclomethasone administered at up to a twofold higher dosage than fluticasone, budesonide, flunisolide, and triamcinolone acetonide. It provides greater relief from nasal symptoms, including nasal blockage, than antihistamine agents and intranasal cromolyn. Its efficacy in the treatment of seasonal allergic rhinitis and perennial allergic and nonallergic rhinitis has been demonstrated in large, well-controlled studies in which the drug maintained adequate control of symptoms when administered in a once-daily dose of 200 mcg.

**Budesonide.** One study compared the use of budesonide with that of fluticasone propionate in patients with allergic rhinitis. Budesonide decreased combined symptoms to a significantly greater extent than did fluticasone (P=.03); both treatments significantly decreased mean combined nasal-symptom scores compared with placebo. Of the three nasal symptoms assessed—nasal blockage, runny nose, sneezing—nasal blockage was decreased to a significantly (P<.01) greater degree with budesonide compared with fluticasone.

**Beclomethasone dipropionate.** Beclomethasone has long been used effectively for allergic rhinitis. However, a recent study shows that the drug may work better when combined with the anticholinergic ipratropium bromide. The researchers concluded that ipratropium bromide nasal spray 0.03% alone should be considered in patients for whom rhinorrhea is the primary symptom. Its use in combination with a nasal steroid should be considered in patients where rhinorrhea is one of the predominant symptoms or in patients with rhinorrhea not fully responsive to other therapy. One note to pass along to patients: The therapeutic effect of beclomethasone may decrease when the canister is cold. Leave it at room temperature before using.

**Mometasone furoate.** Mometasone is the only nasal steroid currently indicated for prophylaxis of seasonal allergic rhinitis. In a study that started four weeks prior to the start of ragweed season, mometasone furoate nasal spray was compared with beclomethasone dipropionate in 330 patients with allergic rhinitis for a period of eight weeks. Both drugs worked the same in terms of offering as many “minimum symptom” days as compared with placebo. The main advantage of mometasone was that it can be taken once daily, compared with twice-daily beclomethasone.

**Antihistamines**

Azelastine is a second-generation histamine H₁ receptor antagonist that has shown clinical efficacy in relieving the symptoms of allergic rhinitis when administered intranasally. Improvement has been seen in both early- and late-phase symptoms of rhinitis through a combination of antihistaminic, antiallergic, and anti-inflammatory mechanisms. Symptom improvements are evident as early as 30 minutes after intranasal administration of azelastine and are apparent for up to 12 hours in patients with allergic rhinitis. Overall, twice-daily intranasal azelastine offers an effective and well-tolerated alternative to other antihistamine agents currently recommended for the symptomatic relief of mild to severe allergic rhinitis in adults and children over age 12.

**Anticholinergics**

Ipratropium bromide is an anticholinergic drug that has antisecretory properties. Applied locally, it inhibits secretions from the serous and seromucous glands lining the nasal mucosa. It is most often used to treat patients with emphysema or chronic obstructive pulmonary disease (COPD), but it can also be used as a nasal inhalant to treat both allergic rhinitis and rhinorrhea secondary to viral infections. The drug is used effectively with perennial cases due to its low side-effect profile and is typically dosed as two sprays in each nostril two to three times daily.

**Mast-cell stabilizers**

Cromolyn sodium, the only inhaled mast-cell stabilizer currently available for treatment of allergic rhinitis, works by inhibiting the effects of inhaled antigens. Cromolyn is best used to prevent allergy symptoms in those who typically develop the symptoms.

**Alternative therapies**

A comprehensive database of natural medicines lists handfuls of herbs that people use to treat asthma, including alfalfa, colt’s foot, digitalis, feverfew, ginkgo, marijuana, and onion. Yet, not one of these has been rated as “likely effective” or “effective” for asthma or allergy. One study looked at people with asthma who self-treated their condition with coffee, black tea, and over-the-counter medications, such as epinephrine and ephedrine. The researchers noted that self-treatment with these substances was associated with an increased risk of hospitalization, not because of something these substances did but because their use delayed the utilization of more efficacious treatments.
Case studies
Case 1: Mark
Mark, a 45-year old male in otherwise sound health, called the pharmacy and asked for a refill of his albuterol. He had obtained a refill the week before, but because he was having more breathing difficulties, the canister was almost empty and he required another.

Mark recently started a job doing interior house painting and had bought a treadmill to lose weight. His new exercise plan has made his leg muscles sore, a symptom for which he takes an over-the-counter remedy. His current symptoms include breathlessness, chest tightness, and wheezing.

Mark's current medications
- Albuterol aerosol: Inhalation one to two puffs every four to six hours as needed for bronchospasm
- Acetaminophen 325mg: Patient takes three to four tablets two or three times a day for soreness from exercise

Strategy for Mark
Mark is using his bronchodilator in an effort to get air into his lungs. The degree of bronchial restriction must be assessed. Using a peak flow meter is the best way to measure airflow. If Mark does not use such a device, he should be instructed to contact his physician immediately, and a decision should be made as to whether he would benefit from the use of oral corticosteroids.

In this case, Mark used his peak flow meter to measure his peak expiratory flow (PEF). His value was 60% of his personal best, suggesting moderate exacerbation. In this case, he should be instructed to use his albuterol as such: two to four puffs at three 20-minute intervals. If after one hour his PEF increases to 80%, this is deemed a good response.

The problem with Mark’s therapy is the lack of an inhaled corticosteroid. Because asthma is intrinsically an inflammation of the airways, the steroid is essential in keeping the inflammation under control. The pharmacist has the opportunity to contact the physician to make this recommendation for Mark, while relating the patient’s recent breathing exacerbation.

Fumes from house paint can cause airway hyper-responsiveness. Mark needs to ventilate the rooms in which he is working. And while running on his treadmill did not create an exercise-induced breathing problem in Mark’s case, the acetaminophen he takes for his sore leg muscles—especially at the high dose of over 1,000 mg several times a day—can contribute to the problem, particularly if he is sensitive to aspirin. In fact, cross-sensitivity has been reported among aspirin, acetaminophen, and the nonsteroidal anti-inflammatory drugs (NSAIDs).

Case 2: Valerie
Valerie, a 13-year-old female in junior high school, has had asthma since the age of five. Her mother accompanied her to the pharmacy to get information on a medication that she heard might help Valerie with her asthma—salmeterol xinafoate, inhaled once every 12 hours. She said that Valerie is nervous about being in junior high.

Valerie’s current medication
- Nedocromil aerosol: Inhale two puffs four times a day

Strategy for Valerie
Valerie’s life is changing, and, as she grows, she will naturally assume more responsibility for her life. Controlling her asthma on her own is an important step. The first step in Valerie’s treatment should be an assessment of medication compliance. This is best done by seeing the adolescent initially without parents and to discuss peak flow monitoring and inhaler technique and to see how much she knows about asthma.

From a medication standpoint, Valerie may have outgrown her anti-inflammatory agent, nedocromil. According to the 1997 asthma guidelines, children with mild or moderate persistent asthma usually benefit from nedocromil or cromolyn, and these anti-inflammatory therapies have no known long-term systemic effects. However, for children with severe persistent asthma, and for many children with moderate persistent asthma, like Valerie, these two agents do not provide adequate control; thus, inhaled corticosteroids are necessary for long-term therapy. For Valerie, a useful corticosteroid would be beclomethasone dipropionate taken at the minimum dose in the medium-dose range: one to two inhalations of the 42-mcg-per-puff concentration, three or four times daily depending on the response. A short-acting bronchodilator, such as albuterol, can be used for quick relief as needed for symptoms of breathlessness. Salmeterol xinafoate has a longer onset of action; it lasts up to 12 hours and could be added to Valerie’s therapeutic regimen later if she had trouble sleeping or wanted to avoid taking medication to school.

Case 3: Will
Will is an 80-year-old widowed male who lives with his sister. He has had asthma for most of his life. He suffered through the bouts and battles of his disease, which included many hospitalizations. He had a stroke last year but survived with his movement and speech faculties intact. His sister arrived home a few weeks ago with six canisters of generic epinephrine aerosol she had purchased at the dollar store. She said the epinephrine would help him breathe better. In the past two weeks, he has been having problems breathing, especially at night. He also has been experiencing heart palpitations, stomach upset, and difficulty urinating. His physician just gave him a prescription for zafirlukast, 20 mg twice daily.
Will’s current medications
• Warfarin 5 mg: Take one tablet daily for anticoagulation.
• Epinephrine solution: Patient inhales as needed for bronchospasm.
• Theophylline elixir 80 mg per 15 ml: Take one tablespoon three times a day.
• Metoprolol 25 mg: Take one tablet twice daily for hypertension.
• Cimetidine 200 mg: Patient takes one to two tablets every four hours for stomach upset.
• Guarana 500 mg: Patient takes two to three tablets a day for “energy.”

Strategy for Will
The pharmacist who brown-bagged Will’s medications found some interesting evidence that could explain some of his problems.

Theophylline: While this methylxanthine can provide long-term control and prevention of asthma symptoms, dose-related acute toxicities can occur, especially in the elderly. Theophylline clearance is reduced in the elderly, causing increased drug-blood levels. Age is a risk factor for theophylline overdose: A 75-year-old has a 16-fold greater risk of death from theophylline overdose than does a 25-year-old. Will has the initial symptoms of theophylline overdose: stomach problems, insomnia, palpitations. What is causing it?

Metoprolol, cimetidine: The beta-blocker and the H₂ histamine antagonist both increase theophylline levels. In Will’s case, there may be a Catch-22 effect with the cimetidine: the more stomach upset he gets from theophylline toxicity, the more cimetidine he takes, making theophylline clearance even slower.

Epinephrine: This drug gives adequate bronchodilation for just one to three hours versus four to six hours for albuterol. Epinephrine can also exacerbate underlying heart conditions, a bad mix for this poststroke patient.

Guarana is an herbal remedy used as a stimulant and tonic. Will assumed that because it was a “natural” product, the substance could do no harm. While the herb has never been scrutinized via controlled clinical studies, information exists to show that it contains theophylline, theobromine, and caffeine, all of which are likely contributing to the theophylline toxicity. Also, the compound inhibits blood platelet aggregation, important since he is on warfarin.

Zafirlukast is an important drug in asthma therapy because it blocks the effects of leukotriene. In terms of drug interactions, the drug is not benign. Zafirlukast can interact with warfarin. This patient’s prothrombin time would need to be closely monitored if he took this drug.

The pharmacist should contact this patient’s physician. At the very least, a theophylline blood concentration should be measured, because theophylline toxicity can be fatal. The patient would be better controlled off theophylline, a problematic drug for someone Will’s age and one that is interacting with the other drugs he is taking. The bronchodilator epinephrine should be replaced with albuterol, which gives better rescue coverage. The patient should be counseled to discontinue the cimetidine and guarana.

Case 4: Jack
Jack is 12 years old and suffers from allergic rhinitis caused by tree pollens in the spring, grass pollens in the summer, and weed pollens in the fall. He has been taking beclomethasone dipropionate for over three years, and the medication does tend to help. However, his father read that inhaled anti-inflammatory medications can result in stunted growth if used for long periods during a child’s developmental years.

Jack’s current medications
• Beclomethasone dipropionate: one oral inhalation three times a day

Strategy for Jack
Long-term use of systemic corticosteroids is inadvisable due to the risk of serious adverse effects, including slowed or interrupted growth. Fortunately, systemic side effects are minimized when used intranasally. The majority of currently available intranasal steroids have low systemic availability via this administration route. Jack is on an inhaled steroid rather than a systemic corticosteroid, but to allay his father’s concern, Jack may benefit from switching his inhaled steroid to one that is delivered intranasally.

Conclusions
Both asthma and allergic rhinitis have inflammatory components that determine the severity of symptoms. Therefore, pharmacotherapy should be focused on anti-inflammatory measures. For asthma, this includes use of an inhaled corticosteroid for long-term control, supplemented by a bronchodilator for intermittent breathing exacerbations. Patients who rely solely on a bronchodilator may experience exacerbations of their condition that can result in increased morbidity and mortality.

Allergic rhinitis can be controlled with a variety of antihistamines and decongestants to decrease symptomatology. However, inhaled nasal steroids, with their predominantly local effect, can often delay or eliminate symptoms completely if taken before symptoms begin. The pharmacist, with a knowledge database that can sort through and eliminate ineffective or competitive medications, can contribute greatly to making the patient’s pharmacotherapy more effective.

References are available upon request.
1. Which one of the following is true about the incidence of asthma and allergic rhinitis?
   a. Deaths from asthma are up.
   b. Deaths from allergic rhinitis are up.
   c. Deaths from both asthma and allergic rhinitis are up.
   d. Neither condition contributes significantly to mortality.

2. Which one of the following is true about asthma and allergies?
   a. They are both autoimmune diseases.
   b. They both can be controlled with bronchodilators.
   c. They are both inflammatory diseases.
   d. Collectively, whites suffer from them more than Hispanics.

3. Which one of the following apparently does not trigger an asthma attack?
   a. ACE inhibitor
   b. Laughter
   c. NSAIDs
   d. Ozone

4. Asthmatic lungs, at autopsy, show:
   a. Little histologic damage
   b. Decreased airway-wall thickness
   c. A dearth of mucus secretion
   d. Epithelial desquamation

5. In allergic rhinitis, which mediator causes mucus secretion?
   a. Prostaglandins
   b. Leukotrienes
   c. Histamine
   d. Platelet-activating factors

6. In asthma therapy, which group is the most potent for long-term control?
   a. Leukotriene antagonists
   b. Corticosteroids
   c. Long-acting beta_2 agonists
   d. Short-acting beta_2 agonists

7. Which one of the following is not a leukotriene antagonist?
   a. Albuterol
   b. Zileuton
   c. Montelukast
   d. Zafirlukast

8. What symptoms are characteristic of moderate persistent Step 3 asthma?
   a. Nighttime symptoms more than twice a month
   b. Nighttime symptoms more than twice a week
   c. Nighttime symptoms more than once a week
   d. Rare nocturnal symptoms

9. Which is not true about theophylline?
   a. Theophylline can be helpful for nocturnal symptoms.
   b. Theophylline has a complex pharmacokinetic profile that warrants serum monitoring.
   c. The clearance rate of theophylline can vary widely, even among patients in the same age group.
   d. Immediate-release theophylline adds to the efficacy of beta_2 agonists

10. What is true about inhaled, short-acting beta_2 agonists?
    a. They are recommended for use on a regular basis.
    b. They produce a treatment effect within 20 to 30 minutes after administration.
    c. They are the treatment of choice for acute asthma symptoms.
    d. Double-blind, placebo-controlled trials have confirmed their ability to worsen asthma.

11. Which one of the following is not part of the management of allergic rhinitis?
    a. Xenotransplantation
    b. Immunotherapy
    c. Avoidance of allergens
    d. Pharmacotherapy

12. Triamcinolone acetonide:
    a. Is a short-acting beta_2 agonist
    b. Can reduce symptoms of allergic rhinitis within the first day of administration
    c. Has been formulated as both an aerosol and a transdermal patch
    d. Suppresses the HPA axis function at therapeutic doses

13. When counseling patients about beclomethasone dipropionate:
    a. Tell them to gently warm the canister in a microwave oven
    b. Tell them to keep the canister in the refrigerator at all times
    c. Tell them the therapeutic effect of the drug is enhanced in cold weather
    d. Tell them to leave the canister at room temperature before using

14. Which of the following is true about azelastine?
    a. It is a potent new oral anti-inflammatory
    b. It is a potent new oral antihistamine
    c. It can improve allergic rhinitis in the early stages only
    d. It is administered intranasally

15. Ipratropium bromide:
    a. Is an anticholinergic drug that has antisecretory properties when taken orally
    b. Is most often used to treat patients with emphysema and COPD
    c. Has a problematic side-effect profile
    d. Has potent mast-cell stabilizing properties

16. Which of the following herbal remedies has been proven “likely effective” in treating asthma?
    a. Onion
    b. Feverfew
    c. Alfalfa
    d. None of the above

17. John is a 25-year-old male who has been taking only albuterol aerosol for his asthma symptoms. Lately, his symptoms have worsened. What could be the problem?
    a. His albuterol dose could be too low.
    b. He is not inhaling the albuterol deeply enough.
    c. He lacks an anti-inflammatory drug, which is the cornerstone of asthma therapy.
    d. He probably is allergic to his cat.

18. A teenager with asthma can expect that:
    a. The disease will resolve itself over time without medications
    b. Medications will be more costly than hospitalization
    c. The medications that once controlled symptoms may change over time
    d. Life’s travails will not significantly impact asthma
19. Mildred, an elderly patient with cardiac problems, has been increasing her theophylline dose because her breathing symptoms are worsening. What can be said about this?
   a. Theophylline clearance is reduced in the elderly, increasing the chances of adverse events.
   b. Age is not a risk factor for theophylline overdose.
   c. Encourage her to increase her dose as needed until symptoms are resolved.
   d. Encourage her to supplement her therapy with OTC epinephrine.

20. What is true about the relationship between intranasal steroids and growth in children?
   a. Long-term use of systemic corticosteroids is recommended for control of allergic rhinitis.
   b. Systemic side effects are minimized when these drugs are used intranasally.
   c. Because of their extremely high system availability, the intranasal steroids can cause significant growth suppression.
   d. When metabolized, intranasal steroids bypass the liver.

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