Advances in Respiratory Care: COPD, Acute Bronchitis, and URI

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Disclosure

• No real or potential conflict of interest to disclose
• No off-label, experimental or investigational use of drugs or devices will be presented.

Objectives

• Having completed the learning activities, the participant will be able to:
  – Identify the pathophysiology and clinical presentation of URI and chronic bronchitis.
  – Describe a plan of pharmacologic intervention for the person with acute bronchitis or viral URI.

Objectives (continued)

• Having completed the learning activities, the participant will be able to: (cont.)
  – Develop a plan of pharmacologic intervention for long-term therapy as well as COPD exacerbation using the GOLD COPD Guidelines.

Global Initiative for Chronic Obstructive Lung Disease

National Heart, Lung, and Blood Institute
NIH
World Health Organization
www.goldcopd.org

COPD Defined

• COPD is a preventable and treatable disease with some significant extra pulmonary effects that can contribute to its severity in individual patients.
• Its pulmonary component is characterized by airflow limitation that is not fully reversible.
Per goldcopd.org

- The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases.

Per goldcopd.org

(continued)

- The diagnosis should be considered in any patient with progressive dyspnea, chronic cough, or sputum production and/or history of exposure to risk factors (tobacco smoking, pollution [outdoor, indoor, or occupational]).

Assessment of COPD

<table>
<thead>
<tr>
<th>Degree of airflow limitation</th>
<th>Spirometry is required for diagnosis. When possible, use age-related values to avoid over-diagnosis in elders. FEV₁:FVC &lt;0.70 post-bronchodilator confirms persistent airflow limitation/COPD Classification of severity determined by FEV₁</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha₁-antitrypsin deficiency screening</td>
<td>Perform when COPD develops in patients of Caucasian descent under 45 years of age or with a strong family history of COPD.</td>
</tr>
</tbody>
</table>

Alpha₁-antitrypsin Deficiency Screening:

- In presence of
  - COPD
  - Emphysema, chronic bronchitis
  - Bronchiectasis
  - Asthma that is incompletely reversible after aggressive treatment
  - Chronic liver disease
  - Unexplained liver disease in children
  - Source: https://www.alpha1.org/Newly-Diagnosed/Learning-about-Alpha-1/Testing-for-Alpha-1

Alpha₁-antitrypsin Deficiency Screening:

- Panniculitis
  - Inflammation of panniculus, layer of fatty and fibrous tissue just beneath skin’s outer layers
  - Source: http://www.alpha1.org/Newly-Diagnosed/Learning-about-Alpha-1/Panniculitis#sthash.iw5G5C.dpuf

Classification of Severity of Airflow Limitation in COPD

Based on Post-bronchodilator FEV₁

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
<th>FEV₁/FVC %</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GOLD 1</strong></td>
<td>Mild</td>
<td>≥80%</td>
</tr>
<tr>
<td><strong>GOLD 2</strong></td>
<td>Moderate</td>
<td>50% ≤ FEV₁ &lt;80%</td>
</tr>
<tr>
<td><strong>GOLD 3</strong></td>
<td>Severe</td>
<td>30% ≤ FEV₁ &lt;50%</td>
</tr>
<tr>
<td><strong>GOLD 4</strong></td>
<td>Very severe</td>
<td>FEV₁ &lt;30%</td>
</tr>
</tbody>
</table>

Source: Global Initiative for Chronic Obstructive Lung Disease, Pocket Guide to COPD Diagnosis, Management and Prevention
Medications Used in the Treatment of COPD:
What are the therapeutic goals of each medication class in the treatment of COPD?
Medications mentioned represent examples of the given drug class, not a comprehensive list of all options. Many of these medications are used for the same purpose in asthma.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Therapeutic goal</th>
</tr>
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<tbody>
<tr>
<td>Short-acting beta2-agonist (SABA) (albuterol), short-acting anticholinergic/muscarinic antagonist (SAMA) (ipratropium bromide)</td>
<td>Relief of bronchospasm</td>
</tr>
<tr>
<td>Long-acting beta2-agonist (LABA) (salmeterol)</td>
<td>Protracted duration bronchodilation</td>
</tr>
<tr>
<td>Long-acting anticholinergic/muscarinic antagonist (LAMA) (tiotropium bromide)</td>
<td>Protracted duration bronchodilation, Minimizes risk of COPD exacerbation</td>
</tr>
<tr>
<td>Inhaled corticosteroid (ICS)</td>
<td>Antiinflammatory, Minimizes risk of COPD exacerbation</td>
</tr>
<tr>
<td>Theophylline</td>
<td>Bronchodilator</td>
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<td>PDE-4 inhibitor (roflumilast)</td>
<td>Minimizes risk of COPD exacerbation</td>
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</table>

PK Comparisons: SABA vs. LABA

Is there a PD difference?

- SABA (albuterol)
  - Time to clinical effect = ½ h
  - $T_{1/2} = 4$ h
- LABA (salmeterol)
  - Time to clinical effect = 1–2 h
  - $T_{1/2} = 8$ h

True or false?
The boxed warning attached to the LABA in the treatment of asthma does not extend to LABA using in COPD.

TRUE
**Medication Therapeutic goal**

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**Muscarinic Antagonist/Anticholinergics**

- **Examples**
  - Ipratropium bromide (Atrovent®)
  - SAMA- Short-acting muscarinic antagonist
    - With albuterol (Combivent® Respimat)
  - MDI
  - Tiotropium bromide (Spiriva®)
    - LAMA-Long-acting muscarinic antagonist
    - DPI

**Muscarinic Antagonist/Anticholinergics (continued)**

- **Mechanism of action**
  - Affinity to muscarinic receptors
  - Inhibits M3-receptors at smooth muscle leading to bronchodilation
  - Different mechanism of bronchodilation when compared to beta₂-agonist
- **Well-established role in COPD, emerging role in asthma**

**Tachyphylaxis/Tolerance**

No evidence of tachyphylaxis, tolerance, reduced clinical effect with inhaled anticholinergic/antimuscarinic therapy

**With Long-acting Bronchodilator Use**

- **If adding LABA**
  - Advise patient to discontinue use of timed (by-the-clock) SABA use.
- **If adding tiotropium bromide**
  - Advise patient to discontinue use of ipratropium bromide.
Inhaled Corticosteroids (ICS) Examples

- Budesonide
  - Pulmicort®
- Fluticasone
  - Flovent®
- Mometasone
  - Asmanex®

Inhaled Corticosteroids with LABA

- Advair Diskus® = 1 puff BID
  - Fluticasone (Flovent®) with salmeterol
    - 100 mcg/50 mcg
    - 250 mcg/50 mcg
      - Recommended COPD dose
    - 500 mcg/50 mcg
- Advair HFA® = 2 puff BID
  - Fluticasone (Flovent®) with salmeterol
    - 45 mcg/21 mcg
    - 115 mcg/21 mcg
    - 230 mcg/21 mcg

Inhaled Corticosteroids with LABA (continued)

- Symbicort® = 2 puff BID
  - Budesonide (Pulmicort®) with formoterol
    - 80 mcg/4.5 mcg
    - 160 mcg/4.5 mcg
      - Recommended dose for COPD

Inhaled Corticosteroids with LABA

- Breo Ellipta®
  - Maintenance treatment of COPD
    - 1 inhalation of Breo Ellipta® 100/25 once daily
  - Maintenance treatment in asthma
    - 1 inhalation of Breo Ellipta® 100/25 or Breo Ellipta® 200/25 once daily

Medication Therapeutic goal

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Theophylline vs. Caffeine: Commonalities and Differences

Theophylline

- Substrate
  - CYP 1A2
    - Levels influenced by amount of tobacco use
- Pharmacogenomics implications
  - Documented influences dependent on

Caffeine

- Substrate
  - CYP 1A2
    - Levels influenced by amount of tobacco use
- Pharmacogenomics implications
  - Documented influences dependent on
### Medication Therapeutic goal

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### Roflumilast (Daliresp®)

- **Adverse effects**
  - Diarrhea=9.5%
  - Weight decrease=7.5%
  - Nausea=4.7%
  - Most common reason to discontinue
  - Headache=4.7%
  - Insomnia=2.4%

**Pharmacologic Therapy for Stable COPD**

Medications mentioned represent examples of the given drug class, not a comprehensive list of all options. "Risk" refers to risk of COPD exacerbation or other untoward event.

### First-line Therapy at Each Stage of COPD

**In patients with FEV1/FVC <0.70:**

- **GOLD 1:**
  - Mild
  - FEV1 ≥80% predicted

- **GOLD 2:**
  - Moderate
  - 50% ≤ FEV1 <80% predicted

- **GOLD 3:**
  - Severe
  - 30% ≤ FEV1 <50% predicted

- **GOLD 4:**
  - Very Severe
  - FEV1 <30% predicted

**GOLD 1st-line Recommendations for Pharmacologic Therapy**

**GOLD 1–2, ≤1 Exacerbation/year**

- Patient Group A (Low risk/less symptoms):
  - SAMA or SABA

- Patient Group B (Low risk/more symptoms):
  - LAMA or LABA

**GOLD 3–4, ≥2 Exacerbations/year**

- Patient Group C (High risk/less symptoms):
  - (ICS + LABA) or LAMA

- Patient Group D (High risk/more symptoms):
  - (ICS + LABA) or LAMA

**SAMA:** Short-acting muscarinic antagonist (e.g., ipratropium [Atrovent®])

**SABA:** Short-acting beta2-agonist (e.g., albuterol [Ventolin® HFA, Proventil® HFA])

**PRN:** As needed

**ICS:** Inhaled corticosteroid (e.g., fluticasone, budesonide)


See supplement for complete guidelines on COPD therapy.
Exacerbation: Definition, Evaluation, and Treatment

• An exacerbation of COPD is an event in the natural course of the disease characterized by a change in the patient’s baseline dyspnea, cough, and/or sputum beyond day-to-day variability sufficient to warrant a change in management.

Treatment of COPD Exacerbation

If baseline FEV$_1$ < 50% of predicted (severe, very severe)

Add a systemic corticosteroid such as prednisone 40 mg/d for 5–10 days. Recent study supports shorter (5-day) course equally effective with fewer adverse effects than longer (10-day) course. Consider adding inhaled corticosteroid if not currently using.

Antimicrobial Therapy in COPD Exacerbation

Likely indicated in the presence of 3 cardinal symptoms: increased dyspnea, increased sputum volume, and increased sputum purulence, though evidence varies.

Smoking cessation is associated with COPD exacerbation reduction and reduction in rate of loss of lung function.

Antimicrobial Therapy in COPD Flare

• Aside from bacterial infection, tobacco use, air pollution, and viruses common contributing factors to COPD flare.

Antimicrobial Therapy

Inhaling corticosteroids if not currently using.

Consider adding long-acting bronchodilator (LABA, [salmeterol], LAMA [tiotropium bromide]) if patient currently not using one.
Advances in Respiratory Care: COPD, Acute Bronchitis, and URI

**Antimicrobial Therapy in COPD Flare (continued)**

- Causative bacterial pathogens (30–50%) include select Gram-negative (*Haemophilus influenzae*, *Haemophilus parainfluenzae*, *Moraxella catarrhalis*) and Gram-positive (*Streptococcus pneumoniae*) pathogens.
  - Less common pathogens include atypical pathogens, other Gram-positive and -negative organisms.

- If prescribed, consider using the following agents:
  - Respiratory fluoroquinolone
  - Macrolide
  - Beta-lactam

  - More severe COPD exacerbation/acute exacerbation of chronic bronchitis
  - Role of antimicrobial therapy debated even for severe disease. If prescribed, consider spectrum of antimicrobial activity and benefit vs. risk ratio with each product including drug interactions.
  - Consider severity of COPD and comorbidities in decision-making process.

- Mild to moderate COPD exacerbation/acute exacerbation of chronic bronchitis
  - Antimicrobial therapy usually not indicated. If prescribed, consider spectrum of antimicrobial activity with each product.

- More severe COPD exacerbation/acute exacerbation of chronic bronchitis
  - Role of antimicrobial therapy debated even for severe disease. If prescribed, consider spectrum of antimicrobial activity and benefit vs. risk ratio with each product including drug interactions.
  - Consider severity of COPD and comorbidities in decision-making process.

**Antimicrobial Therapy in COPD Flare (continued)**

- Consider chest x-ray only with fever and/or low SaO₂ to help rule out concomitant pneumonia.

- True or false?
  - According to the most recent update of GOLDCOPD guidelines, the use of a daily dose of azithromycin to minimize COPD exacerbation risk does not have a favorable benefit vs. risk ratio and is not recommended.

- Use one of the following agents:
  - Beta-lactam
    - Amoxicillin-clavulanate
    - Cephalosporin (cefdinir, cefpodoxime, others)
  - Macrolide
    - Azithromycin
    - Clarithromycin
  - Respiratory fluoroquinolone
    - Moxi-, levofloxacin

  - Less common pathogens include atypical pathogens, other Gram-positive and -negative organisms.
Stepwise Approach for Managing Asthma in Patients Age ≥ 12 Years

**Intermittent Asthma**

Consult with asthma specialist if Step 1 care is required. Consider consultation at Step 3.

**Step 1**
- Patient Adherence: Yes
- Control: Not controlled
- Asthma severity: Intermittent
- Use: SABA PRN

**Step 2**
- Preferred: Low-dose ICS
- Alternative: Cromolyn, LTRA, Nedocromil*, Theophylline

**Step 3**
- Preferred: Low-dose ICS + LABA
- OR
- Medium-dose ICS
- Alternative: Low-dose ICS + either LTRA, Theophylline, or Zileuton **

**Step 4**
- Preferred: Medium-dose ICS + LABA
- Alternative: Medium-dose ICS + either LTRA, Theophylline, or Zileuton **

**Step 5**
- Preferred: High-dose ICS + LABA
- AND
- Consider Omalizumab for Patients Who Have Allergies

**Step 6**
- Preferred: High-dose ICS + LABA + Oral Corticosteroid
- AND
- Consider Omalizumab for Patients Who Have Allergies

Estimated Comparative Daily Dosages for ICS in Patients Aged ≥ 12 Years

<table>
<thead>
<tr>
<th>Low Daily Dose</th>
<th>Medium Daily Dose</th>
<th>High Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beclomethasone HFA 40 or 80 mcg/puff</td>
<td>&gt;240–480 mcg</td>
<td>&gt;480 mcg</td>
</tr>
<tr>
<td>Budesonide DPI 200 mcg/inhalation</td>
<td>&gt;600–1200 mcg</td>
<td>&gt;1200 mcg</td>
</tr>
<tr>
<td>Fluticasone 250 mcg/puff</td>
<td>1000–2000 mcg</td>
<td>&gt;2000 mcg</td>
</tr>
<tr>
<td>Fluticasone HFA 80 mcg/puff</td>
<td>320 mcg</td>
<td>640–1200 mcg</td>
</tr>
<tr>
<td>Fluticasone DPI 44, 110, or 220 mcg/puff</td>
<td>264–440 mcg</td>
<td>&gt;440 mcg</td>
</tr>
<tr>
<td>Fluticasone DPI 50, 100, or 250 mcg/puff</td>
<td>300–500 mcg</td>
<td>&gt;500 mcg</td>
</tr>
<tr>
<td>Mometasone DPI 200 mcg/puff</td>
<td>400 mcg</td>
<td>&gt;400 mcg</td>
</tr>
</tbody>
</table>


LAMA Use in Asthma

- Approved for use in asthma
  - Tiotropium bromide (Spiriva®, Respimat®)
  - Once-daily maintenance treatment for patients with asthma age ≥ 12 years who remain symptomatic on ICS or ICS/LABA
- Not for the relief of acute bronchospasm

LAMA Use in Asthma vs. COPD

- In asthma age ≥12 years and older
  - 2 inhalations of Spiriva® Respimat® 1.25 mcg once-daily
- In COPD
  - 2 inhalations of Spiriva® Respimat® 2.5 mcg once-daily
- Likely need 4–8 weeks of use prior to full clinical effect

Tiotropium in Asthma Poorly Controlled with Standard Combination Therapy

Source: Huib A.M. Kerstjens, M.D., Michael Engel, M.D., Ronald Dahl, M.D., Pierluigi Paggiaro, M.D., Ekkehard Beck, M.D., Mark Vandewalker, M.D., Ralf Sigmund, Dipl.Math., Wolfgang Seibold, M.D., Petra Moroni-Zentgraf, M.D., and Eric D. Bateman, M.D.

N Engl J Med
Volume 367(13):1198-1207
September 27, 2012

Study Overview

- In two trials, the addition of tiotropium to the treatment of patients whose asthma was not controlled by inhaled glucocorticoids and long-acting beta-agonists led to a modest improvement in lung function and a decrease in severe asthma exacerbations over 48 weeks.
Advances in Respiratory Care: COPD, Acute Bronchitis, and URI

### Lung Function and Severe Exacerbations

![Graph](https://newenglandjournalofmedicine.org/doi/fig/full/10.1056/NEJMoa1208606)


**Conclusion**

- In patients with poorly controlled asthma despite the use of inhaled glucocorticoids and LABAs, the addition of tiotropium significantly increased the time to the first severe exacerbation and provided modest sustained bronchodilation.


### Antihypertensive Medication Use in Person with COPD

- In COPD, increased risk of ACEI-induced cough?
- Cardioselective beta blocker therapy be safe to use in COPD?
- Additional potential benefits of beta blocker therapy?

### Renin-angiotensin Cascade: What Works Where?

![Diagram](https://newenglandjournalofmedicine.org/doi/fig/full/10.1056/NEJMoa1208606)

**ACEI-induced Cough**

- The mechanism of ACE inhibitor-induced cough remains unresolved, but likely involves the protussive mediators bradykinin and substance P, agents that are degraded by ACE and therefore accumulate in the upper respiratory tract or lung when the enzyme is inhibited, and prostaglandins, the production of which may be stimulated by bradykinin.


### Effect of Beta Blockers in Treatment of Chronic Obstructive Pulmonary Disease: A Retrospective Cohort Study

- Conclusions- β blockers may reduce mortality and COPD exacerbations when added to established inhaled stepwise therapy for COPD, independently of overt cardiovascular disease and cardiac drugs, and without adverse effects on pulmonary function.

  - Source: [BMJ 2011 May 10;342:d2549. doi: 10.1136/bmj.d2549](http://bmj.com/content/342/bmj.d2549)
Beta Blocker Therapy: Examples

- Non cardioselective
  - B1-, B2-blockade
    - Propranolol
    - Nadolol
    - Pindolol
    - Sotalol
    - Carvedilol
  - Also alpha1-blockade

- Cardioselective
  - B1-receptor selective
    - Metoprolol
    - Bisoprolol
    - Betaxolol
    - Atenolol

Considering Route and Method of Administration for Medications in Asthma and COPD

Albuterol Nebulizer vs. MDI

- Typical nebulized albuterol dose=2.5 mg with 12% deposition=300 mcg
- Typical MDI albuterol dose=180 mcg with 20% deposition=36 mcg


Benefits vs. Drawbacks of Nebulized Medications in Asthma/COPD

- Potential benefits
  - Delivery with lower lung volumes
  - Potential to deliver larger medication doses (i.e., nebulized albuterol during COPD flare)
  - Does not require breath holding, coordinated breath as with many MDI, DPI

Benefits vs. Drawbacks of Nebulized Medications in Asthma/COPD (continued)

- Drawback
  - Need for specialized device
  - Possible medication overuse
  - Potential for limited mobility
  - “Attached to the machine”

Non-nebulized Medications

- MDI (metered dose inhaler)
  - Active ingredients dissolved or suspended in propellant, solvents, or combination in compact pressurized aerosol dispensers
  - Requires patient activation of device, inhalation and breath-holding in combination
Non-nebulized Medications (continued)

- **DPI** (dry powder inhaler)
  - Dry powder inhaled on activation by inhalation driven by patient inspiration alone or with power assistance

True or false?

- The diagnosis of acute bronchitis is usually limited to those without chronic airway disease (e.g., asthma or COPD).
  - **TRUE**

Cough associated with acute bronchitis can typically last up to:

A. 1 week.
B. 2 weeks.
C. 3 weeks.
D. 3 months.

Which of the following is the most common pathogen implicated in acute bronchitis?
A. *S. pneumoniae*
B. *H. influenzae*
C. *M. pneumoniae*
D. Respiratory virus

### Acute Bronchitis: Likely Causative Pathogens

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<thead>
<tr>
<th>Organism</th>
<th>%</th>
<th>Comment</th>
</tr>
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<tbody>
<tr>
<td>Respiratory tract viruses</td>
<td>90</td>
<td>Consider using anticholinergic bronchodilator, such as ipratropium bromide (Atrovent®), inhaled beta₂-agonist, such as albuterol, or short course of oral corticosteroid (for example, prednisone 40 mg PO daily dose for 3–5 days) with protracted, problematic cough</td>
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<tr>
<td>Bacterial pathogens, such as <em>M. pneumoniae, C. pneumoniae, B. pertussis</em></td>
<td>10</td>
<td>Consider use of macrolide or tetracycline form such as doxycycline when antimicrobial therapy indicated.</td>
</tr>
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Conclusion

Thank you for your time and attention.

Margaret A. Fitzgerald, DNP, FNP-BC, NP-C, FAANP, CSP, FAAN, DCC, FNAP

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End of Presentation
• Images/Illustrations: Unless otherwise noted, all images/illustrations are from open sources, such as the CDC or Wikipedia or property of FHEA or author.
• All websites listed active at the time of publication.
First-line Therapy at Each Stage of COPD

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**GOLD 1st-line Recommendations for Pharmacologic Therapy**

GOLD 1–2, ≤1 Exacerbation/year  
GOLD 3–4, ≥2 Exacerbations/year

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SAMA: Short-acting muscarinic antagonist (e.g., ipratropium [Atrovent®]) PRN  
SABA: Short-acting beta₂-agonist (e.g., albuterol [Ventolin® HFA, Proventil® HFA]) PRN  
LAMA: Long-acting muscarinic antagonist (e.g., tiotropium [Spiriva®])  
LABA: Long-acting beta₂-agonist (e.g., salmeterol [Serevent®])  
ICS: Inhaled corticosteroid (e.g., fluticasone, budesonide)


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Stepwise Approach for Managing Asthma in Patients Age ≥12 Years

<table>
<thead>
<tr>
<th>Intermittent Asthma</th>
<th>Persistent Asthma: Daily Medication</th>
</tr>
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<tbody>
<tr>
<td>Consult with asthma specialist if Step 4 care or higher is required. Consider consultation at Step 3.</td>
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</table>

**Step 1**  
Preferred:  
SABA PRN

**Step 2**  
Preferred:  
Low-dose ICS

Alternative:  
Cromolyn, LTRA, Nedocromil*, or Theophylline

**Step 3**  
Preferred:  
Low-dose ICS + LABA

OR  
Medium-dose ICS

Alternative:  
Low-dose ICS + either LTRA, Theophylline, or Zileuton**

**Step 4**  
Preferred:  
Medium-dose ICS + LABA

OR  
High-dose ICS + LABA AND Consider Omalizumab for Patients Who Have Allergies

**Step 5**  
Preferred:  
High-dose ICS + LABA + Oral Corticosteroid AND Consider Omalizumab for Patients Who Have Allergies

**Step 6**  
Preferred:  
High-dose ICS + LABA + Oral Corticosteroid AND Consider Omalizumab for Patients Who Have Allergies

Steps 2-4: Consider subcutaneous allergen immunotherapy for patients who have allergic asthma

Quick-relief medication for all patients

• SABA as needed for symptoms. Intensity of treatment depends on severity of symptoms. Up to 3 treatments at 20-minute intervals as needed. Short course of systemic oral corticosteroids may be needed

• Use of SABA > 2 days a week for symptom relief (not prevention of EIB) generally indicates inadequate control and the need to step up treatment

*Not available
**Seldom used

Assess Control

Step Up if Needed (first, check adherence, environmental control, and comorbid conditions)

Step Down if Possible (and asthma is well controlled at least 3 months)

Intermittent  
Persistent  
Assessment

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