Objectives

Upon completion of this chapter, you will be able to

- Define key terms relative to bronchodilator pharmacotherapy
- Describe the neurological control of bronchial smooth muscle, including the sympathetic and parasympathetic nerves, their chemical mediators, and how bronchodilation is achieved
Objectives

- Differentiate bronchospasm and bronchoconstriction
- Describe three pharmacologic methods for bronchodilation (sympathomimetic, anticholinergic, and methylxanthine) and the mode of action of each
- State the indications, contraindications, adverse reactions, onset of action, and dosage range for each bronchodilator
Objectives

- Recommend appropriate bronchodilator therapy for various patient situations, including drug, dosage, frequency, and route of delivery
- Describe appropriate techniques for monitoring the patient’s response to bronchodilator therapy
Airway Anatomy and Physiology

- Conducting airways are made up of three layers
  - **Mucosa**—Contains ciliated cells that move the mucus toward the pharynx to keep the lumen of the airway clear.
  - **Submucosa**—Contains bronchial glands, smooth muscle, capillary network, and elastic tissue.
  - **Adventitia**—A sheath of connective tissue that surrounds and supports the airways.
Airway Anatomy and Physiology

- Large airways begin with the trachea and then divide to form bronchi.
- Between the submucosa and the adventitia layers are incomplete rings of cartilage that provide support to prevent the airways from collapsing.
- Below the subsegmental bronchi, the bronchi become bronchioles.
Airway Anatomy and Physiology

- **Bronchioles**—Small bronchi or small airways, 1 to 2 mm in diameter, that lack supporting cartilage.
Figure 5–1: Cross-Sectional Anatomy of Bronchus, Bronchiole, and Alveolus
Bronchoconstriction

- Decrease or narrowing in the diameter of the airways.
- Occurs due to swelling, mucus obstruction, or spasm of the smooth muscle in the airway.
Three Factors Contributing to Bronchoconstriction

- **Bronchospasm**—Spasm or contraction of the smooth muscle in the bronchial wall. Airway diameter is reduced, causing a reduction in airflow and increased work of breathing.
Three Factors Contributing to Bronchoconstriction

- **Edema**—Occurs when insult or injury to the mucous membranes causes dilation of the blood vessels and accumulation of fluid in the tissues. The swollen tissue reduces the diameter of the lumen of the airway, and breathing becomes more difficult.
Three Factors Contributing to Bronchoconstriction

- **Secretions**—Reduce airway diameter when contained within the airway. Can result from impairment of the normal mucociliary clearance mechanism of the lungs.
Figure 5–2: (a) Normal Bronchi, (b) Bronchospasm, (c) Mucosal Edema, and (d) Mucus Narrowing the Airway
Sympathetic Nervous System

- Dominates the body’s reaction to stressful circumstances.
- Fight-or-flight response.
- Stimulates the heart, increases cardiac output and blood pressure, dilates pupils, increases metabolism, and enhances alertness.
Sympathetic Nervous System

- Relaxes smooth muscle to dilate the airways and lower airway resistance, helping to increase ventilation.
- The increased rate and depth of breathing cause an increase in ventilation.
Parasympathetic Nervous System

- Dominates the body’s maintenance functions.
- Decreases heart rate and blood pressure and increases bronchoconstriction and mucus secretion.
- Increases salivation and mucus secretion, increases blood flow to the gut, and increases peristalsis and urination.
Figure 5–3: Balance Between the Sympathetic and Parasympathetic Nervous Systems that Maintains Normal Bronchial Smooth Muscle Tone
Figure 5–4: Stimulated Parasympathetic Nerve Causing Bronchospasm
Figure 5–5: Impulse Transmission in the Sympathetic and Parasympathetic Nervous Systems
Sympathetic Nervous System

Receptors

- Three types of receptors
  - Alpha \((\alpha)\)
  - Beta_1 \((\beta_1)\)
  - Beta_2 \((\beta_2)\)

- Each receptor type is distributed to different parts of the body and, when stimulated, produces different effects.
α Receptors

- Found in the arteries and veins throughout the body, including the vessels in the lungs.
- Distributed evenly throughout the large and small airways.
- Stimulation results in vasoconstriction.
**β₁ Receptors**

- Found mainly in the heart, where they increase both the rate and force of contraction when stimulated.
- Many cardiac agents stimulate these receptors.
$\beta_2$ Receptors

- Found in the bronchiolar smooth muscle of the lung, the uterus, and skeletal muscle blood vessels.
- Found throughout the tracheal bronchial tree, especially in the small airways.
- When stimulated, they relax the smooth muscle in the lungs, causing bronchodilation.
**β₂ Receptors**

- In the uterus, stimulation of beta2 receptors can stop contractions of premature labor.
<table>
<thead>
<tr>
<th>Receptor</th>
<th>Location</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha</td>
<td>Arteries and veins</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td>Beta₁</td>
<td>Heart</td>
<td>Increase in rate and force of contraction</td>
</tr>
<tr>
<td>Beta₂</td>
<td>Lungs, skeletal muscle, and uterus</td>
<td>Smooth muscle relaxation</td>
</tr>
</tbody>
</table>
Sympathetic Nervous System in the Lung

- Arteries and submucosal glands of the lung are innervated by sympathetic nerve fibers.
- The bronchial smooth muscle lacks nerve fibers and responds to circulating hormones or medications.
- $\alpha$ and $\beta_2$ receptors of the bronchial smooth muscle are stimulated by the circulating epinephrine and NE.
<table>
<thead>
<tr>
<th>Effects</th>
<th>Sympathomimetic</th>
<th>Parasympathomimetic</th>
<th>Sympatholytic</th>
<th>Parasympatholytic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchial smooth muscle</td>
<td>Relaxes</td>
<td>Relaxes</td>
<td>Constricts</td>
<td>Constricts</td>
</tr>
<tr>
<td>Mucus secretion</td>
<td></td>
<td>Decreases</td>
<td></td>
<td>Increases</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Increases</td>
<td>Increases</td>
<td>Decreases</td>
<td>Decreases</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Increases</td>
<td>Increases</td>
<td>Decreases</td>
<td>Decreases</td>
</tr>
<tr>
<td>Pupils</td>
<td>Dilate</td>
<td>Dilate</td>
<td>Constrict</td>
<td>Constrict</td>
</tr>
<tr>
<td>Blood flow to skeletal muscles</td>
<td>Increases</td>
<td>Increases</td>
<td>Decreases</td>
<td>Decreases</td>
</tr>
<tr>
<td>Salivation</td>
<td>Decreases</td>
<td>Decreases</td>
<td>Increases</td>
<td>Increases</td>
</tr>
<tr>
<td>Digestion</td>
<td>Decreases</td>
<td>Decreases</td>
<td>Increases</td>
<td>Increases</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>Increases</td>
<td>Increases</td>
<td>Decreases</td>
<td>Decreases</td>
</tr>
<tr>
<td>Insulin</td>
<td>Decreases</td>
<td>Decreases</td>
<td>Increases</td>
<td>Increases</td>
</tr>
</tbody>
</table>
Types of Bronchodilators

- Divided into three categories based on the mechanism of their action
  - Sympathomimetics (beta-adrenergic)
  - Anticholinergics (parasympatholytic)
  - MethylXanthines
Bronchodilators

- Sympathomimetics cause bronchodilation by increasing cyclic AMP, a substance that causes bronchial smooth muscle relaxation.
- Anticholinergics block the bronchoconstricting effects of the parasympathetic system by decreasing cyclic GMP, a substance that causes bronchial smooth muscle constriction.
Both sympathomimetics and anticholinergics are administered as inhaled aerosols.

Methylxanthines act as weak bronchodilators and are taken orally or intravenously.
Sympathomimetic (Adrenergic) Bronchodilators

- Have three potential effects, depending on the receptor that is stimulated
  - Alpha stimulation results in vasoconstriction, reducing blood flow and decreasing swelling. In the upper airway, decongestion can result.
  - $\beta_1$ stimulation results in increased heart rate and force of contraction.
Sympathomimetic (Adrenergic) Bronchodilators

- Have three potential effects, depending on the receptor that is stimulated
  - $\beta_2$ stimulation results in bronchial smooth muscle relaxation, inhibition of inflammatory response, and increased mucociliary clearance.
Mechanism of Action

- When a $\beta_2$ agonist binds to the $\beta_2$ receptor, it activates special stimulatory proteins that activate the membrane-bound enzyme adenyl cyclase.
- Adenyl cyclase increases synthesis of cyclic adenosine monophosphate (cAMP).
- cAMP causes relaxation of smooth muscle by inactivating an enzyme that initiates the interaction of actin and myosin.
Mechanism of Action

- cAMP decreases the amount of intracellular calcium, causing relaxation because calcium is needed for contraction.
- cAMP has anti-inflammatory properties related to its ability to inhibit mast cell chemical mediator release.
Mechanism of Action

- cAMP can be broken down by the enzyme phosphodiesterase. Then cAMP becomes AMP, which is inactive and does not produce bronchodilation.
Figure 5–6: Sympathetic Stimulation Favoring Bronchodilation

*Note:* That anything that enhances cAMP (sympathomimetics) or anything that decreases or lightens cAMP (anticholinergics) tips the scales toward bronchodilation.
The Earliest β Agonists: Catecholamines

- The basic catecholamine molecule consists of a catechol nucleus and an amine side chain.
- The nucleus is made up of a benzene ring and two hydroxyl groups.
- Researchers have found that making changes to the structure of the benzene ring could make drugs more resistant to degradation by enzymes.
The Earliest $\beta$ Agonists: Catecholamines

- Changes to the benzene ring have resulted in three chemical classes of sympathomimetics
  - Catecholamine
  - Resorcinols
  - Saligenins
Figure 5–7: Basic Catecholamine Molecule
**Figure 5–8: Catecholamine, Resorcinol, and Saligenin Chemical Modifications**

*Note:* Increasing length of amine side chain results in more 2-specificity.
Basic Catecholamine Structure Animation

Click the screenshot to view an animation showing basic catecholamine structure.

Back to Directory
Resorcinol Modification Animation

Click the screenshot to view an animation showing a resorcinol modification.

Back to Directory
Click the screenshot to view an animation showing a saligenin modification.
Chemical History of Beta Drugs

Click the screenshot to view an animation showing a chemical history of beta drugs.
Catecholamines

- Oldest group of inhaled bronchodilators.
- Sympathetic bronchodilators.
- Basic structure consists of a benzene ring and an amine chain.
- Rapid onset, though have a short duration, therefore requiring frequent dosages.
Catecholamines

- Deactivated by COMT and MAO.
- Cannot be given orally because they are degraded by the GI tract.
- Drugs in this class are no longer used as inhaled bronchodilators.
Catecholamines

- **Examples**
  - Epinephrine (IV for cardiac effect).
  - Norepinephrine (for cardiac effect).
  - Racemic epinephrine (treats upper airway swelling—weak bronchodilator).
  - Dopamine (for cardiac effect).
  - Isoproterenol (for cardiac effect).
  - Isoetharine (no longer used).
  - Bitolterol (no longer used).
Racemic Epinephrine (Vaponefrin, Micronefrin)

- Synthetic form of epinephrine that has both $\alpha$ and $\beta$ effects.
- Used as a topical vasoconstrictor for treatment of airway edema associated with croup and laryngeal edema.
Resorcinols

- Drugs are resistant to the breakdown by COMT.
- Longer acting than catecholamines.
- Can be taken orally because they resist the action of enzymes in the GI tract and liver.
- First maintenance drugs for treatment of reactive airway disease.
Metaproterenol
(Alupent, Metaprel)

- Can be administered as aerosol solution, tablet, and syrup. (MDI form removed from market)
- Onset of action is 5 to 15 minutes.
- Duration time is 4 to 6 hours.
- More $\beta_2$ specific than catecholamines.
- Cardiac side effects because of its structural similarity to isoproterenol.
Terbutaline (Brethine, Bricanyl)

- $\beta_2$ specific and longer acting than catecholamines.
- Few cardiac side effects.
- Onset of action is 5 to 15 minutes.
- Duration time is 4 to 6 hours.
- No longer available as an MDI but can be administered subcu to stop premature labor.
Saligenins

- Most recently developed.
- Most widely prescribed type of bronchodilator.
- Most $\beta_2$ specific.
- Similar to resorcinols in that they have a rapid onset of action and duration of 4 to 6 hours.
Albuterol (Proventil, Ventolin)

- Frequently administered bronchodilator.
- Very few side effects.
- Long side chain.
- Onset of action is 15 minutes.
- Reaches its peak effect in 30 to 60 minutes and has a duration of 4 to 6 hours.
Albuterol (Proventil, Ventolin)

- Available in a syrup for children, and a oral tablets, extended-release tablets, nebulizer solution, MDI, and DPI.
Figure 5–7: Ventolin® HFA metered-dose inhaler
Piributerol (Maxair)

- Same side chain as albuterol.
- Available orally, as a syrup for children, and as an MDI with a breath-actuated inhaler device.
- Less potent by weight than albuterol.
- Similar to metaproterenol in efficacy and toxicity.
- Similar side effects of other $\beta_2$ agonists.
Levalbuterol (Xopenex)

- R-isomer saligenin.
- More potent dilator than albuterol, with fewer side effects (some controversy on this).
- Metabolizes very slowly.
- Onset of action is 15 minutes after administration.
Levalbuterol (Xopenex)

- Reaches peak effect in 30 to 60 minutes.
- Lasts 3 to 8 hours.
Salmeterol (Serevent)

- **Lipophilic**—Salmeterol diffuses into the cell’s bilayer phospholipid membrane and enters the receptor from a lateral approach instead of the \( \beta \) agonist directly approaching from the extracellular space.
- Provides 12-hour duration of action.
- Ideal for maintenance use.
- Not recommended for use as a rescue treatment.
Salmeterol (Serevent)

- Slow onset.
- Albuterol should be given in conjunction with Serevent.
Figure 5–7: Serevent Diskus®
Copyright GlaxoSmithKline. Used with permission.
Formoterol (Foradil)

- Both a fast- and long-acting $\beta_2$ agonist.
- Used as a maintenance drug in treatment of asthma in adults and children 5 years or older.
- Available as DPI.
Formoterol (Foradil)

- Even though it has a rapid onset, it is not recommended as a rescue drug for acute asthma because repeated administration of long-acting drugs increases toxicity risks.
Side Effects of $\beta$ Agonists

- Most side effects are associated with epinephrine and isoproterenol.
- New $\beta_2$-selective drugs have fewer side effects.
- Most common side effect is muscle tremor due to stimulation of $\beta_2$ receptors in the skeletal muscle.
- Increased heart rate and vasodilation.
Side Effects of $\beta$ Agonists

- **Tolerance**—Decreased response to a drug that occurs with long-term use.
- **Tachyphlaxis**—Decreased response to a drug that occurs shortly after administration.
- **Down regulation**—A decrease in the number of $\beta_2$ receptors.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Route</th>
<th>Dose</th>
<th>Frequency</th>
<th>Onset (min)</th>
<th>Peak (min)</th>
<th>Duration (hr)</th>
<th>Receptor</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Catecholamines</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Neb</td>
<td>0.25–0.5 ml</td>
<td>q4–q6 hr</td>
<td>3–5</td>
<td>5–20</td>
<td>1–3</td>
<td>α, β</td>
</tr>
<tr>
<td>Adrenalin®</td>
<td>Neb</td>
<td>0.25–0.5 ml</td>
<td>q4–q6 hr</td>
<td>3–5</td>
<td>5–20</td>
<td>1–3</td>
<td>α, β</td>
</tr>
<tr>
<td>Racemic epinephrine</td>
<td>Neb</td>
<td>0.25–0.5 ml</td>
<td>q3–q6 hr</td>
<td>3–5</td>
<td>5–20</td>
<td>0.5–2</td>
<td>α, β</td>
</tr>
<tr>
<td><strong>Resorcinols</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metaproterenol</td>
<td>Neb</td>
<td>0.2–0.3 ml</td>
<td>t.i.d.–q.i.d.</td>
<td>1–5</td>
<td>60</td>
<td>6</td>
<td>β₂ &gt; β₁</td>
</tr>
<tr>
<td><strong>Saligenins</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Albuterol</td>
<td>HFA</td>
<td>2 puffs</td>
<td>t.i.d.–q.i.d.</td>
<td>15</td>
<td>30–60</td>
<td>5–8</td>
<td>β₂ &gt; β₁</td>
</tr>
<tr>
<td>Ventolin®</td>
<td>HFA</td>
<td>2 puffs</td>
<td>t.i.d.–q.i.d.</td>
<td>15</td>
<td>30–60</td>
<td>5–8</td>
<td>β₂ &gt; β₁</td>
</tr>
<tr>
<td>Levalbuterol</td>
<td>Neb</td>
<td>0.25–0.5 ml</td>
<td>t.i.d.–q.i.d.</td>
<td>15</td>
<td>30–60</td>
<td>5–8</td>
<td>β₂ &gt; β₁</td>
</tr>
<tr>
<td>Xopenex®</td>
<td>Neb</td>
<td>0.31–3.78 mg</td>
<td>t.i.d.</td>
<td>15</td>
<td>30–60</td>
<td>3–8</td>
<td>β₂ &gt; β₁</td>
</tr>
<tr>
<td>Formoterol</td>
<td>DPI</td>
<td>1 capsule</td>
<td>q12 hr</td>
<td>3–4</td>
<td>30–60</td>
<td>12</td>
<td>β₂</td>
</tr>
</tbody>
</table>
Classifying by Duration of Action

- Bronchodilators are classified as either short-acting or long-acting.
  - Short acting beta agonists (SABA’s).
  - Long-Acting Beta Agonists (LABA’s).
Classifying by Duration of Action

- Rescue therapy drugs—rapid-acting medications that provide rapid relief of symptoms—the SABA’s.
- Maintenance therapy drugs—Provide long-term control of symptoms. (LABA’s)
Patient and Family Education: New Warning Label for LABA’s

- Ongoing FDA studies show an increased risk of exacerbations of asthma resulting in hospitalizations and deaths with patients using LABA’s.
Patient and Family Education: New Warning Label for LABA’s

- While LABA’s are felt clinically appropriate for the asthmatic patient, they should only be used with an asthma control medication such as an inhaled steroid. Combination products containing a LABA and an inhaled steroid are encouraged.
Figure 5–11: Parasympathetic Stimulation Favoring Bronchoconstriction
Anticholinergic (Parasympatholytic) Bronchodilators

- Class of bronchodilators.
- Can block the effects of the parasympathetic system and therefore cause bronchodilation (e.g., atropine, ipratropium bromide [Atrovent]).
- Ipratropium bromide is an atropine derivative with fewer side effects and is a commonly used anticholinergic bronchodilator.
Figure 5–12: Diagram of Parasympathetic Neurotransmission Blockage (parasympatholytic) Leading to Bronchodilation
Atropine Sulfate

- Prototypical parasympathetic.
- Relaxes airway smooth muscle.
- Not commonly used as a bronchodilator due to having many side effects.
- Side effects include restlessness, irritability, and fatigue with small doses; patients may experience hallucinations and disorientation with higher doses.
Atropine Sulfate

- Inhibits mucus production and reduces mucociliary clearance.
- Primarily used as a cardiac drug.
Ipratropium Bromide (Atrovent)

- Available as a nebulization solution, in MDI from, and as a nasal spray pump.
- Can be given in conjunction with Albuterol in a single MDI canister to form the drug Combivent.
- Onset of bronchodilation begins in minutes.
Ipratropium Bromide (Atrovent)

- Peak effect in 1 to 2 hours.
- Little or no effect on mucociliary clearance.
- Decreases hypersecretion of mucus in the nose.
- Has no effect on heart rate, blood pressure, or the GI tract.
Figure 5–12: Atrovent® MDI

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Tiotropium Bromide (Spiriva)

- Long-acting anticholinergic agent indicated for long-term, once-daily maintenance treatment of COPD.
- Not indicated as rescue drug.
- Available in DPI.
<table>
<thead>
<tr>
<th>Drug and Trade Names</th>
<th>Dosage</th>
<th>Frequency</th>
<th>Onset (min)</th>
<th>Peak (hr)</th>
<th>Duration (hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipratropium bromide</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atrovent® Solution for Nebulization</td>
<td>500 mcg/unit dose</td>
<td>t.i.d., q.i.d.</td>
<td>1–5</td>
<td>0.25</td>
<td>4–8</td>
</tr>
<tr>
<td>Atrovent® MDI</td>
<td>2 puffs</td>
<td>q.i.d.</td>
<td>15</td>
<td>1–2</td>
<td>4–6</td>
</tr>
<tr>
<td>Ipratropium bromide and albuterol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combivent® MDI</td>
<td>2 puffs</td>
<td>q.i.d.</td>
<td>15</td>
<td>2</td>
<td>6–8</td>
</tr>
<tr>
<td>Duoneb®</td>
<td>3 ml</td>
<td>q.i.d.</td>
<td>5</td>
<td>0.8</td>
<td>4–5</td>
</tr>
<tr>
<td>Tiotropium bromide</td>
<td>1 inhalation/day</td>
<td>Once daily</td>
<td>30</td>
<td>3</td>
<td>&gt;24</td>
</tr>
<tr>
<td>Spiriva®</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Monitoring Outcomes of Bronchodilator Therapy

- Patients should be assessed before, during, and after treatment in order to get an accurate assessment of the effectiveness of the medication.
- When delivering the treatment, one could obtain baseline data such as heart rate, respiratory rate, and breath sounds.
Monitoring Outcomes of Bronchodilator Therapy

- The practitioner should ask the patient about his or her perceived level of dyspnea.
- The practitioner should observe the patient’s breathing pattern and accessory muscle use.
Monitoring Outcomes of Bronchodilator Therapy

- Measurement of effort-dependent peak expiratory flow rate (PEFR) and forced expiratory volume in 1 second (FEV$_1$) could be performed before and after the treatment.
- Measurements allow the practitioner to determine posttreatment improvement.
Click the screenshot to view a video on the topic of peak flow use.

Back to Directory
Signs of Positive Bronchodilator Response

- Improved appearance.
- Decreased dyspnea.
- Decreased use of accessory muscles.
- Improved vital signs.
- Increased sputum production.
Signs of Positive Bronchodilator Response

- Decreased wheezing or increased intensity of breath sounds.
- Increased FEV\textsubscript{1} or PEFR.
- Improved oxygenation.
Signs of Negative Bronchodilator Response

- Increased heart rate can occur.
- HR greater than 20% of the baseline heart rate or 20 beats/minute is significant.
- If increased HR occurs
  - Stop the treatment.
  - Monitor the patient until the effects subside.
  - Notify the physician.
  - Document the incident in the patient’s chart.
Signs of Negative Bronchodilator Response

- Changes in the route of administration or smaller doses of the medication may help reduce unwanted side effects of the treatment.
MethylXanthines

- **Examples**
  - Theophylline (found in tea leaves)
  - Aminophylline

- **Other well-known examples** (both are found in cocoa)
  - Theobromine
  - Caffeine

- Administered orally or intravenously.
Traditionally, methylxanthines were thought to produce bronchodilation by inhibiting phosphodiesterase, which deactivates cAMP.
Methylxanthines’ Mechanism of Action

- Although this is still thought to be true, at dosages used in humans, methylxanthines are poor inhibitors of phosphodiesterase.
- Bottom line is they are considered a weak bronchodilator.
Nonbronchodilating Effects of Theophylline

- Increases the strength and endurance of muscle contraction, especially the diaphragm.
- Prevents respiratory failure by increasing the patient’s ventilatory drive.
- Metabolizes at different rates in different people.
Nonbronchodilating Effects of Theophylline

- Serum drug levels should be monitored because methylxanthines react with other medications
- Side effects can include
  - **CNS**—Dizziness, headache, restlessness
  - **Cardiovascular**—Palpitations, arrhythmias, tachycardia
  - **GI**—Nausea, vomiting, diarrhea
### Table 5-6  Common Brand Names for Theophylline

<table>
<thead>
<tr>
<th>Brand Name</th>
<th>Brand Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elixirphyllin®</td>
<td>Theo-Dur®</td>
</tr>
<tr>
<td>Slo-Phylllin®</td>
<td>Theobid®</td>
</tr>
<tr>
<td>Slo-Bid®</td>
<td>Respbid®</td>
</tr>
<tr>
<td>Theolair®</td>
<td>Sustaire®</td>
</tr>
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</table>