

Treatment of Bronchial Asthma

- **Definition of Asthma:** Chronic inflammatory disorder with intermittent narrowing of the airways and it is characterized by wide variations, over short periods of time, in the resistance to flow in the intrapulmonary airways.
- **Factors in the Treatment Strategy:**
 - Asthma is a chronic condition.
 - The goal of therapy is normal function.
 - Asthma is heterogeneous in terms of:
 - Cause or trigger mechanism.
 - Extent of bronchoconstriction.
 - Degree of inflammation.
 - The course is unpredictable.
 - Therapy must be individualized.
- **Risk of Not Treating Asthma:**
 - Poor or no control of the patient's asthma.
 - Accelerated decline in the function of the patient's lungs.
 - Increased number of attacks of asthma.
 - Poorer response to therapy if started late.
 - Increased mortality from asthma.
- **Goals of Therapy in Asthma:**
 - Minimal symptoms even during sleep.
 - No, or infrequent, acute episodes.
 - No ED visits or missed days in school or work.
 - Rare need for beta-agonist inhaler therapy.
 - No limitation of activities – even sports.
 - Peak flow rate variability less than 20%.
 - No or minimal adverse effects from drugs.
- **Pathogenesis:**
 - Early Asthmatic Response:
 - Allergens provoke IgE production.
 - The tendency to produce IgE is genetically determined.
 - Re-exposure to the allergen causes antigen-antibody interaction on the surface of the mast cells leading to: Release of stored mediators, synthesis of other mediators and activation of neural pathways.
(This response is prevented by bronchodilators).

○ Late Asthmatic Response:

- 4-5 hours later.
- More sustained phase of bronchoconstriction.
- Influx of inflammatory cells and an increase in bronchial responsiveness.
- The mediators here are cytokines produced by TH2 lymphocytes, especially interleukins: 5, 9, and 13.
- These mediators (produced in the early and late phases) will stimulate IgE production by B lymphocytes, and directly stimulate mucus production.
(This response is prevented by corticosteroids).

➤ **Asthma Triggers:**

- Exercise / cold air.
- Cigarette smoke.
- Stress / anxiety situations.
- Animal dander's (cats, dogs etc..).
- Allergens (grass, trees, molds, cockroach).
- Pollutants (sulfur dioxide, ozone, etc...).
- Fumes/toxic substances.
- Medications (ASA, NSAID's, others).

➤ **Diagnosis of Asthma – Subjective:**

- **Cough** - usually in spasms and to the point of vomiting – nighttime worse than daytime. Cough may follow exposure to cold air, exercise, URI (common cold), or exposure to an allergen.
- **Dyspnea** > cough or wheezing > sputum.
- Past history of **bronchiolitis** as a child.
- **Family history** of asthma is common.

➤ **Diagnosis of Asthma – Objective:**

- Diminished peak expiratory flow rate (PEFR).
- Reduced mean and forced expiratory flow rate (FEFR).
- Reversibility with Bronchodilators
- Heightened response to Methacholine Test.
- Increase in expired Nitric Oxide.
- Increase in Inflammatory mediators and their metabolic products in body fluids.

➤ **Myths and Misconceptions:**

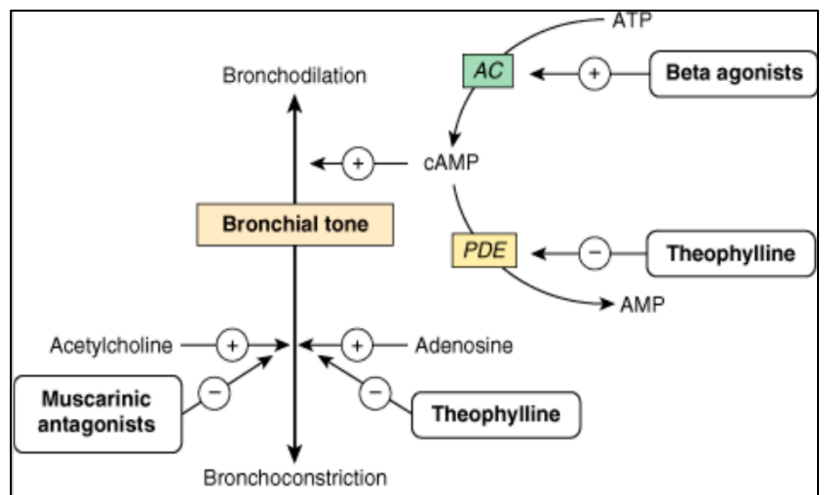
- Patient and physician “Steroid-o-phobia”.
- Asthma is an emotional illness.
- Asthma is an acute disease.
- Asthma medications are addictive.

- Asthma medications become ineffective if they are used regularly.
- Asthma is not a fatal illness / It does not kill.
- **Overview of the changing therapy of asthma by decade:**
 - 1960's: Aminophylline, Epinephrine, Ephedrine.
 - 1970's: Beta-agonists, Theophyllines, Beclomethasone, Cromolyn, Ipratropium.
 - 1980's: Beta-agonists, Inhaled Corticosteroids, Cromolyn, Ipratropium.
 - 1990's: Inhaled Corticosteroids, Beta-agonists, Theophylline, Leukotriene Inhibitors.
 - 2000's: Corticosteroids + LABA, LTRAs (Leukotriene receptor antagonists), Theophylline, Cromolyn, Ipratropium, Tiotropium.
 - 2010's: Prevention, including gene therapy.

➤ **Relievers / Controllers:**

- Quick relief medications:
 - Inhaled Short acting Beta-2 Agonists.
 - Inhaled Anticholinergics.
 - Systemic Corticosteroids.
- Long-term control medications:
 - Topical (inhaled) Corticosteroids.
 - Inhaled Cromolyn Na and Nedocromil.
 - Oral Methylxanthines (Theophyllines).
 - Inhaled Long-acting Beta-2 Agonists (LABA).
 - Oral Leukotriene modifiers (LTRA).

Step-wise approach to asthma therapy					OCS
			LABA	LABA	
		LABA	ICS High dose	ICS High dose	
	ICS Low dose	ICS Low dose			
Short-acting β_2 -agonist as required for symptom relief					
Mild intermittent	Mild persistent	Moderate persistent	Severe persistent	Very severe persistent	



➤ The following figures summarize what was previously mentioned, check them:

