# BRONCHIAL ASTHMA

Asthma is a common chronic inflammatory disorder of the airways characterized by reversible airflow limitation, airway hyper responsiveness and inflammation of the bronchi.

**Etiology:** Asthma is an enormously complex condition that may present at any age. Many factors can contribute to the development of asthma, understanding these are keys to effective management.

1. **Atopy:** Atopy is a genetic predisposition to IgE-mediated allergen sensitivity. Atopic individuals are predisposed to the following three conditions: Allergic asthma, Atopic dermatitis, Allergic rhinitis
2. **Hygiene hypothesis:** The hygiene hypothesis was first developed from epidemiological data showing increased autoimmune and allergic disease in western countries. It postulates that reduced exposure to infectious pathogens at a young age predisposes individuals to such diseases. In simple terms, this environment is thought to encourage Th2 predominant response - one that produces IgE.
3. **Aspirin-induced asthma**: A small subset of patients with asthma is affected by sensitivity to aspirin. Ingestion is capable of triggering an attack. These patients exhibit Samter's triad: Asthma, Aspirin sensitivity, Nasal polyps, Occupational asthma

Around 15% of cases of asthma in adults are related to occupational exposure. Asthma may be induced or exacerbated by such exposure. Hundreds of sensitisers have been identified. They may be categorized as:

1. **High molecular weight:** These are compounds that trigger an IgE-dependent response. The effects are immediate or soon after exposure. e.g. flour, latex.
2. **Low molecular weight:** These compounds develop a complex immune response after repeated and long-term exposure. e.g. isocyanates, wood dusts. Peak expiratory flow diaries during periods of work and holiday are key to the diagnosis of occupational asthma.
3. **Exercise-induced asthma:** In this variant asthma is triggered by strenuous physical activity. The etiology is complex but exposure to cold air and environmental pollutants contributes.

**Pathophysiology** Asthma is the result of aberrant airway inflammation and bronchospasm. Both contribute to an increase in airway resistance.

**Early phase:** Inhalation of allergens results in an immediate (type 1) hypersensitivity reaction in the airways. Sensitization occurs during the allergen exposure causing the release of IgE antibodies from plasma cells. IgE bind to high affinity receptors on mast cells.

Subsequent exposure to antigens cause mast cell degranulation and histamine release. These mediators cause smooth muscle contraction and bronchoconstriction whilst inflammation contributes to airway obstruction.

**Late phase:** The initial early phase reaction may be followed by a late response hours later. During the late phase, recruitment of a variety of inflammatory cells (e.g. PMN cells, T-cells) occurs. The late response is more complex than the early response involving multiple additional processes. Beta-agonists do not cause complete reversal of the late response.

**Chronicity:** In response to persistent chronic inflammation, the airways lay down fibrous tissue. Over time airway remodelling occurs and manifests as fixed airway obstruction - i.e. airway narrowing that is irreversible.

**Clinical features:** There may be little in the way of signs and symptoms between attacks. Cough, shortness of breath and an expiratory wheeze are typical.

*Symptoms:*

- Cough (may be worse at night)
- Dyspnea
- Chest tightness
- Signs
- Expiratory wheeze
- Prolonged expiratory phase
- Tachypnea
- Harrison's sulcus: a groove at the inferior border of the rib cage that may be seen inchildren with chronic severe asthma. Also seen in rickets.
- Severe acute attack
- Severe asthma requires rapid recognition and treatment. Wheeze may not be present andrespiratory effort is frequently depressed.
- Signs of hypoxia and altered mental status may be present.