ASTHMA

The word *asthma* is derived from the ancient Greek word for "panting." The currently understood basis of asthma is summarized in the National Heart, Lung, and Blood Institute's definition in the 1995 "Global Initiative for Asthma".

The first evidence-based asthma guidelines were published in 1991 by **The National Asthma Education and Prevention Program** (NAEPP), under the coordination of the **National Heart, Lung, and Blood Institute (NHLBI)** of the **National Institutes of Health (NIH)**. These guidelines were updated in 1997, 2002, and 2007.

The **Global Initiative for Asthma (GINA)** was established in 1993 in collaboration between the United States (the **National Heart, Lung and Blood Institute [NHLBI]**, the **National Institutes of Health [NIH]**) and the **World Health Organization (WHO)**.

Asthma is characterized by hyperreactive airways that respond to various stimuli by widespread inflammation and airway narrowing, which is often reversible either spontaneously or with treatment.

Aetiology

Asthma commonly begins in childhood but can occur at any age. There is often a predisposition to asthma if parents or close relatives are asthmatic or atopic. Environmental factors can trigger an attack of asthma, including allergens such as house dust mite; furred animals (dogs, cats, etc.); pollens and moulds and chemical irritants such as tobacco smoke, air pollution or inhaled chemicals. In addition, exercise (especially running in cold air), respiratory tract infections and
some foods and drinks can also be triggers.

Extrinsic Asthma (Allergic or Atopic Asthma)

When an asthmatic episode can clearly be linked to exposure to a specific allergen (antigen), the patient is said to have *extrinsic asthma* (also called allergic or atopic asthma). Common indoor allergens include house dust mites, furred animal dander (e.g., dogs, cats, and mice), cockroach allergen, fungi, molds, and yeast. Outdoor allergens include pollens, fungi, molds, and yeast. In addition, there are a number of occupational substances associated with asthma.

Intrinsic Asthma (Nonallergic or Nonatopic Asthma)

When an asthmatic episode cannot be directly linked to a specific antigen or extrinsic inciting factor, it is referred to as intrinsic asthma (also called nonallergic or nonatopic asthma). The etiologic factors responsible for intrinsic asthma are elusive. Individuals with intrinsic asthma are not hypersensitive or atopic to environmental antigens and have a normal serum IgE level. The onset of intrinsic asthma usually occurs after the age of 40 years, and typically there is no strong family history of allergy.

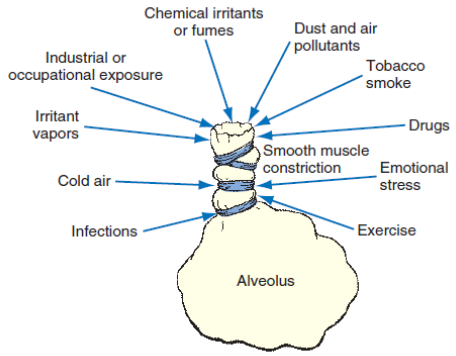


FIGURE 13-4 Some factors known to trigger intrinsic asthma (see

Immunologic mechanisms

1. When a susceptible individual is exposed to a certain antigen, lymphoid tissue cells form specific IgE (reaginic) antibodies. The IgE antibodies attach themselves to the surface of mast cells in the bronchial walls.
2. Reexposure or continued exposure to the same antigen creates an antigen-antibody reaction on the surface of the mast cell, which in turn causes the mast cell to degranulate and release chemical mediators such as histamine.

3. The release of these chemical mediators stimulates parasympathetic nerve endings in the bronchial airways, leading to reflex bronchoconstriction and mucous hypersecretion. Moreover, these chemical mediators increase the permeability of capillaries, which results in the dilation of blood vessels and tissue edema.

BOX 13-2 Risk Factors for Asthma

Host Factors

- Genes
  - That is, genes predisposing the patient to IgE-mediated allergic reaction; airway hyperresponsiveness; or a group of inflammatory mediators (e.g., cytokines, chemokines)
- Obesity
- Sex and gender

Environmental Factors

- Allergens
  - Indoor: Domestic mites, furred animals (dogs, cats, mice), cockroach allergen, fungi, molds, yeast
  - Outdoor: Pollens, fungi, molds, yeasts
- Infections (primarily viral)
- Occupational sensitizers and hobbies/leisure activity hazards
- Tobacco smoke
- Outdoor/Indoor air pollution
- Diet: especially in the case of food allergies

Pathophysiology

The mechanisms by which asthma occurs are complex and still under debate due to the variety of triggers for an attack of asthma. In asthma triggered by allergens, the antigen–antibody reaction causes the mast cells in the airways to degranulate, releasing substances, resulting in
contraction (bronchoconstriction), and increase capillary permeability and inflammation. Mucous glands may be hypertrophied, releasing thick sticky mucus that can lead to mucous plugging of small airways. Inflammatory cells (eosinophils) in the mucus may give the appearance of infection due to yellow colour.

- Smooth muscle constriction of bronchial airways (bronchospasm)
- Excessive production of thick, whitish bronchial secretions
- Mucous plugging
- Hyperinflation of alveoli (air trapping)
- In severe cases, atelectasis caused by mucous plugging
- Bronchial wall inflammation leading to fibrosis (in severe cases, caused by remodeling)

Anatomic Alterations of the Lungs

Asthma is described as a lung disorder characterized by
(1) reversible bronchial smooth muscle constriction,
(2) airway inflammation, and
(3) increased airway responsiveness to an assortment of stimuli.

During an asthma attack, the smooth muscles surrounding the small airways constrict. Over time the smooth muscle layers hypertrophy and can increase to three times their normal thickness.
The airway mucosa becomes infiltrated with **eosinophils** and other inflammatory cells, which in turn causes airway inflammation and mucosal edema. Microscopic crystals, called **Charcot-Leyden crystals**, are formed from the breakdown of eosinophils in patients with allergic asthma.
The goblet cells proliferate, and the bronchial mucous glands enlarge. The airways become filled with thick, whitish, tenacious mucus. Extensive mucous plugging and atelectasis may develop. As a result of smooth muscle constriction, bronchial mucosal edema, and excessive bronchial secretions, air trapping and alveolar hyperinflation develop. If chronic inflammation develops over time, these anatomic alterations become irreversible, resulting in loss of airway caliber. In addition, the cilia are often damaged, and the basement membrane of the mucosa may become thicker than normal (fibrosis). This whole process is referred to as "**remodeling**."

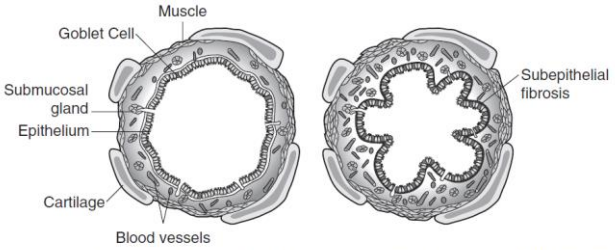


Figure 6-9 Airway remodeling in asthma is depicted through representations of the airway wall in health (*left*) and in asthma (*right*). In asthma there is goblet cell hyperplasia/hypertrophy, subepithelial fibrosis, increased vascularity, and smooth muscle hypertrophy and hyperplasia. Not shown is the increased collagen deposition throughout the airway wall, smooth muscle proliferation throughout the airway wall, degenerative changes in cartilage, and pericartilaginous fibrosis. (From Mason RJ, Murray JF, Broaddus VC, et al. Murray & Nadel's Textbook of Respiratory Medicine. ed 4. Philadelphia, 2005, Saunders.)

Clinical Features

Symptoms of asthma include recurrent episodes of wheezing, cough, breathlessness and chest tightness. Signs during an attack of asthma are wheeze on auscultation, obstructive pattern on spirometry, hyperinflation on chest X-ray, and use of accessory inspiratory muscles of breathing. Children with asthma often have atopic (allergic) features such as eczema, food allergies, hay fever or urticaria.

The most prominent symptoms of asthma are

- wheezing,
- chest tightness, and
- shortness of breath.

Diagnosis/ Investigations

A clinical diagnosis of asthma is based on episodic symptoms of wheeze, chest tightness, breathlessness and cough, especially if such symptoms occur after exposure to an allergen and there is a strong positive family history of asthma and atopy. Reversal of symptoms and improved spirometry (less obstruction) after the inhalation of a bronchodilator medication aids with diagnosis. There are various challenge tests to measure airway responsiveness, which help to establish a diagnosis of asthma, including inhaled methacholine or histamine, inhaled mannitol or an exercise challenge test.

Medical Management

Pharmacologic therapy can be divided into two classes of medications:

- Short-term "relievers" or "rescue "medications (inhaled medications that reverse acute bronchospasm)
- Long-term "controllers" (anti-inflammatory, long-acting bronchodilators that improve overall asthma control when taken regularly).

The most effective short-term "reliever" medications are the short-acting β-adrenergic agonists, such as albuterol (Proventil, Ventolin) or levalbuterol (Xopenex), or anticholinergics, such as ipratropium bromide (Atrovent) or tiotropium (Spiriva). They increase airway diameter by relaxing airway smooth muscle.

The most effective long-term controllers are the inhaled corticosteroids, which are believed to produce benefits through their anti-inflammatory action.

Nonpharmacologic therapy includes reduction of allergen exposure in the home and, in the United States, allergen immunotherapy (allergy shots). Prevention and prompt treatment of viral infections are very important as they can precipitate an asthma attack,

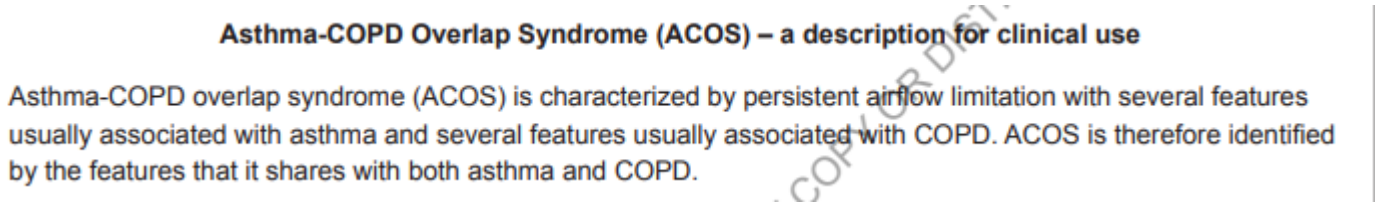
Implications for Physical Therapy Treatment

The most important treatment for asthma management involves the use of appropriate medications to reduce inflammation, stabilize airways, and relieve bronchospasm. Physical therapy interventions should not begin unless an appropriate regime of medication has been initiated. Individuals with exercise-induced asthma may require the use of bronchodilator medication 30 minutes prior to exercise. Following medical therapy, the following physical therapy interventions may be helpful:

- Secretion clearance techniques (Active cycle of breathing and gentle manual techniques, shaking and vibration work well with this population)
- Controlled breathing techniques
- Exercise and strength training

• Thoracic stretching exercises
• Postural reeducation to avoid round-shouldered postures

ASTHMA-COPD OVERLAP SYNDROME (ACOS)



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