

**COLD**

**COAD**

**Chronic Obstructive Pulmonary  
Disease  
COPD**

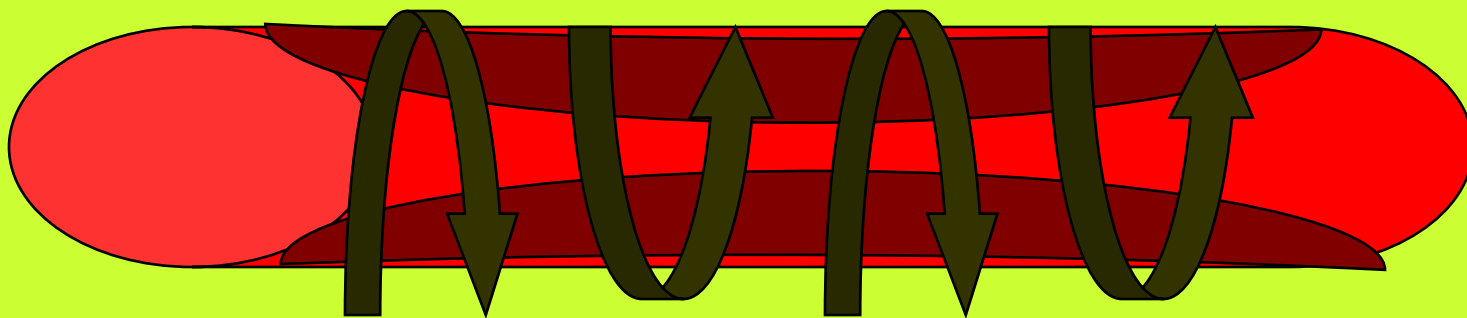


**CRAP**

**COAP**

# Asthma

- **Acute/chronic bronchospastic inflammatory airway disease**



The National Asthma Education and Prevention Program released the Report of the Second Expert Panel on the Guidelines for the Diagnosis and Management of Asthma. The report, based on a comprehensive review of the scientific evidence accumulated during the past 6 years, updates the first asthma clinical practice guidelines released in 1991.

The new report continues to advocate a step-wise approach to pharmacological therapy. But based on the enhanced understanding of inflammation and its contribution to abnormalities in lung function, it is emphatic that persistent asthma should be controlled with daily anti-inflammatory medications. Medications are now categorized into two general classes: long-term control medications, used to achieve and maintain control of persistent asthma, and quick-relief medications, used to treat acute symptoms and attacks.

The report also establishes more relevant classifications for asthma severity and links its recommendations for the amount and frequency of medication to the severity of each patient's asthma. The new classifications are mild intermittent, mild persistent, moderate persistent, and severe persistent.

The new report confirms the close relationship between allergy and asthma in most asthma patients and the importance of reducing exposures to indoor and outdoor allergens. But it expands on this by establishing that allergy testing should be used to identify perennial indoor allergens for certain asthma patients and by including recommendations for controlling other factors that can increase asthma symptoms, such as aspirin/NSAIDs and respiratory infections.

The report also makes the point that asthma onset may possibly be prevented by reducing exposures to allergens and tobacco smoke. Stating that undertreatment of asthma in young children is a problem, it also includes a new section on asthma in infants and young children.

The new report establishes clear criteria and mechanisms for an asthma diagnosis and strongly recommends that spirometry be used in an initial diagnostic work-up. It also includes new recommendations for use of spirometry and peak flow monitoring in diagnosing and monitoring asthma.

As in the 1991 Expert Panel Report, education for an active partnership with patients is advocated as the cornerstone of asthma management. The new report calls for starting education at the time of asthma diagnosis, integrating it into every step of clinical asthma care, and tailoring it specifically to the needs of each patient, with sensitivity to cultural beliefs and practices. Patient outcomes should be measured in terms of the patients' perceptions of improvement, especially quality of life and the ability to live a normal, active life.

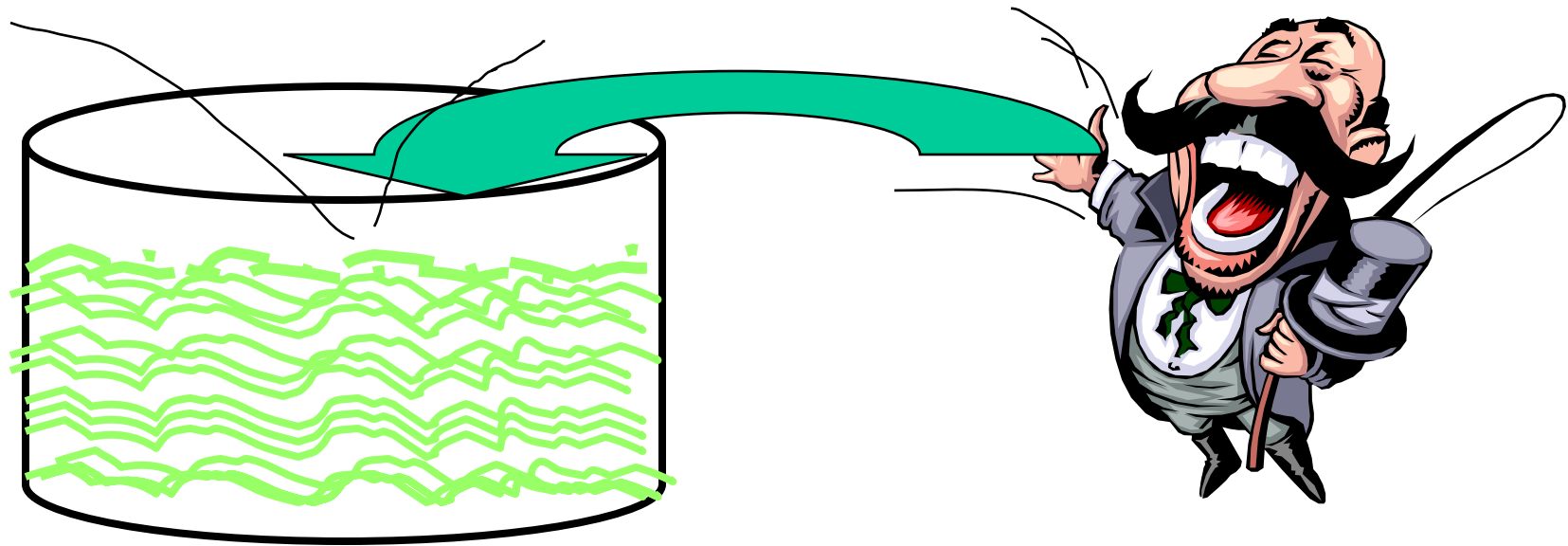
The report will be converted into a concise practical guide for physicians and widely distributed. The full Expert Panel Report is available online at <http://www.nhlbi.nih.gov/nhlbi/lung/asthma/prof/asthgdln.htm>

# Asthma

- ABGs to watch:
  - Initially PaO<sub>2</sub> normal, low, PaCO<sub>2</sub> below normal
  - Worsens, PaO<sub>2</sub> below normal
  - Intubate, PaCO<sub>2</sub> starts to climb

# Chronic Bronchitis

- Chronic Bronchitis is defined by symptoms: a history of a productive cough for at least 3 months a year for 2 consecutive years.



# Chronic Bronchitis(cont)

## Pathophysiology

- 1)there is hypertrophy of the bronchial glands and an increase in the number of goblet cells lining the respiratory tract mucosa, which typically is chronically inflamed.

# Chronic Bronchitis(cont)

## Oxygenation

- 1)These patients usually have a widened A-a gradient while breathing room air.
- 2)hypoxemia worsens with the extent of bronchial obstruction.
- 3)Chronic hypoxemia may lead to secondary polycythemia.

(cont)

# Chronic Bronchitis (cont)

## Oxygenation (cont)

- 4) The combination of elevated HGB concentration and hypoxemia may result in cyanosis.

# Chronic Bronchitis (cont)

## Acid/Base

- 1) Advanced chronic bronchitis is often accompanied by hypercapnia.
- 2) The elevated Pa CO<sub>2</sub> is compensated by renal retention and production of bicarbonate making the pH usually in the normal range.

# Chronic Bronchitis (cont)

## Cardiac Effects

- 1) Chronic hypoxemia also favors development of cor pulmonale.
- 2) V/Q mismatching causes vasoconstriction of pulmonary arterioles in hypoxic lung units.
- 3) This increased pressure raises afterload on the right ventricle, causing it to become enlarged.

(cont)

# Chronic Bronchitis (cont)

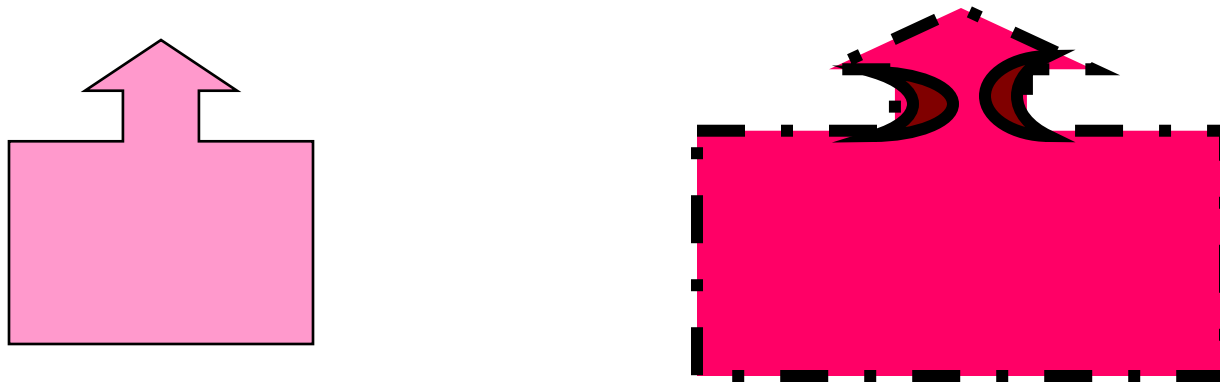
## Cardiac Effects (cont)

- 4) Radiographic evidence of an enlarged right ventricle and pulmonary arteries is consistent with chronic bronchitis.
- 5) Compromise of the right ventricle is accompanied by distension of neck veins, an enlarged liver, and peripheral edema.

# Emphysema

## Definition

Emphysema is defined anatomically as a destructive process of the lung parenchyma leading to permanent enlargement of the distal airspaces.



# Emphysema (cont)

## Pathophysiology

1) Can be one or a combination of the following:

a) CLE is centrilobular, which mainly involves the respiratory bronchioles.

CLE is predominantly in smokers.

# Emphysema (cont)

PLE or panlobular which can involve the entire terminal respiratory unit.

PLE can occur in non-smokers.

# Emphysema (cont)

## Pathophysiology (cont)

3) As CLE progresses, it becomes increasingly difficult to distinguish it from PLE, at which point it may be labeled mixed or end-stage emphysema.

4) In either case, bullae or airspaces greater than 1 cm in size may develop.

(cont)

# Emphysema (cont)

## Pathophysiology (cont)

- 5) May also be due to alpha-antitrypsin deficiency. This is a genetic defect:
- a) alpha 1-antitrypsin is an anti-protease which blocks the effects of the proteases.
  - b) the proteases are enzymes that digest proteins.

(cont)

# Emphysema

pathophysiology (cont)

5c) elastin and collagen are the major protein components of lung tissue.

d) so, with the release of elastase, elastin is destroyed.

e) without an anti-protease to stop the action of the protease (elastin-eating enzyme) there is an imbalance and more lung tissue is destroyed than intended.

# Emphysema

## Pathophysiology (cont)

- 6) Airway obstruction in any form of emphysema is due to destruction of elastic tissues that normally maintain small airway patency.
- 7) This results in loss of elastic recoil (compliance increases).
- 8) TLC will increase due to this increased distensibility.

# Emphysema (cont)

## Oxygenation (cont)

- 1) Diffusion is impaired due to decrease in surface area available for gas transfer.
- 2) The loss of alveolar surface tends to match destruction of the capillary bed, so that ventilation and perfusion mismatching is minimal.

(cont)

# Emphysema

## Oxygenation (cont)

- 3) Radiographic exam shows that there are flattened diaphragms and increased retrosternal airspace (lung hyper inflation).
- 4) Radiographic exam may also show bullae near the apices.

# COPD Continuum

