# OCCUPATIONAL RESPIRATORY DISEASES

By: Dr Chavoshi



## The respiratory tract is often the site of injury from occupational exposures

 The respiratory tract has a limited number of ways to respond to injury

♦ Acute : rhinosinusitis, laryngitis, ...

Chronic : asthma, parenchymal fibrosis, ...

#### **Evaluation of Patients**

Detailed history (occupational & environmental exposures, hobbies)
Thorough physical exam (not specific, insensitive)
Appropriate imaging studies
Pulmonary function tests (spirometry, bronchoprovocative tests)

#### **Types of Diseases**

Toxic inhalation injury
Occupational asthma
Hypersensitivity pneumonitis
Inhalation fever
Pneumoconioses
Chronic bronchitis
Pleural disorders
Lung cancer

#### **Toxic Inhalation Injury**

Short term exposure to high concentrations of noxious gases, fumes, mists
Severity & site of injury depends on:

Water solubility
Concentration
Duration of exposure
Ventilation of victim



#### Treatment

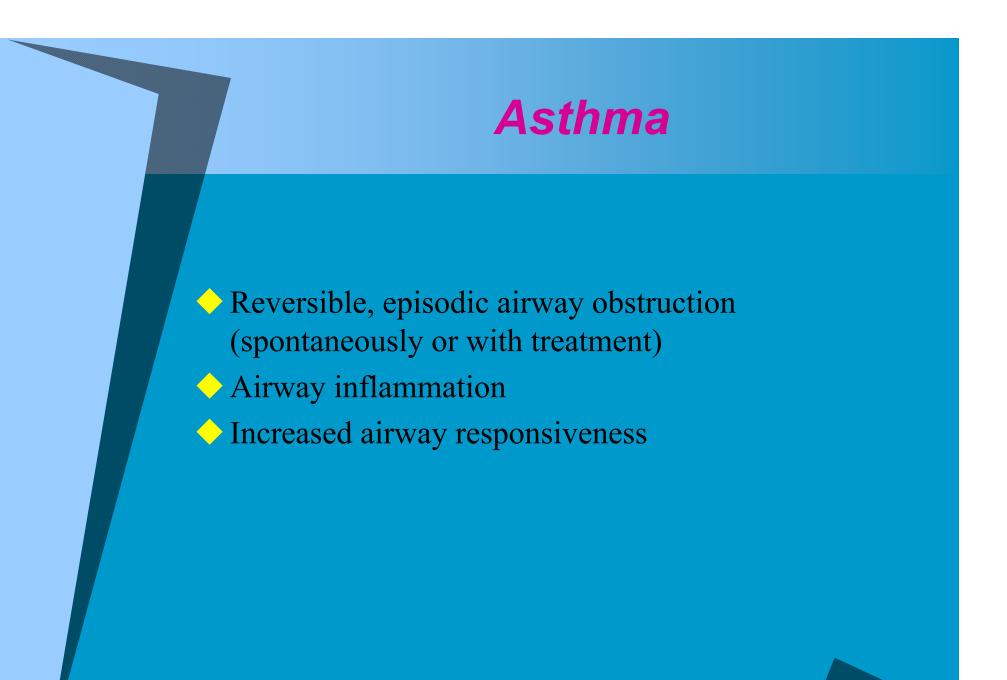
Decontamination

 $\diamond$  O2

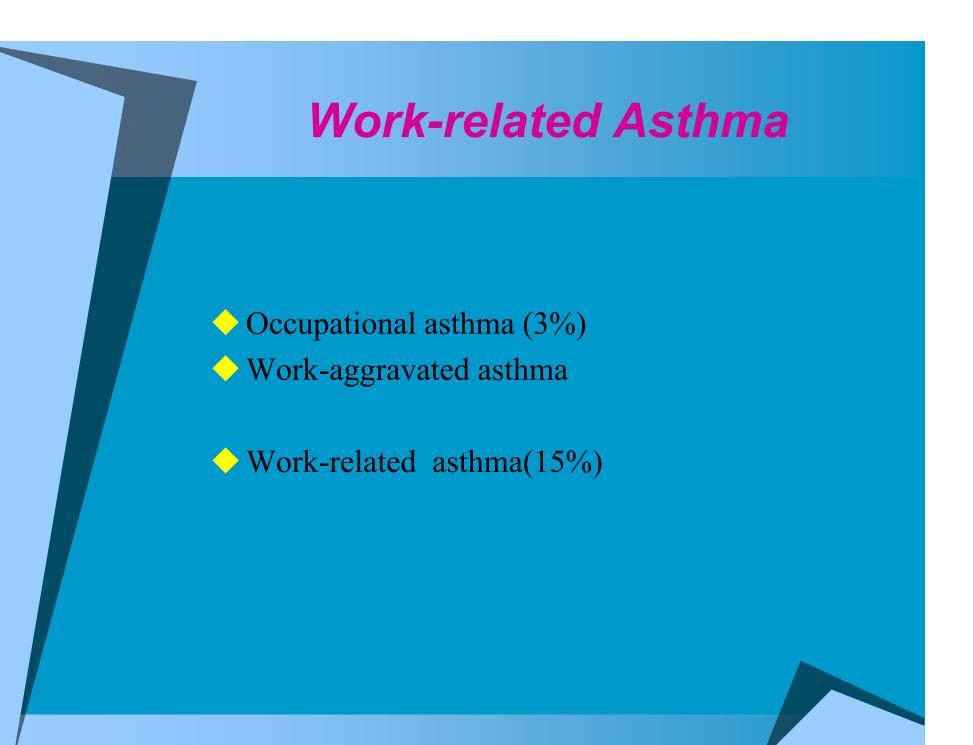
Bronchodilator

♦ Drainage

 No steroids unless the patient is not recovering promptly



 Occupational asthma is the most common occupational respiratory disorder



#### **Occupational Asthma**

#### Sensitizer-induced

- long term exposure
- specific responsiveness to an etiologic agent
- Irritant-induced (RADS)
  - a single intense exposure
  - persistent non-specific hyperresponsiveness

 Non-specific hyper-responsiveness is a very important characteristic of allergic & non-allergic induced asthma

#### **Sensitizing Agents**

#### **Non-sensitizing Agents**

Anticholinesterase effect

 e.g. organophosphates

 Endotoxin effect

 e.g. cotton dust

 Airway inflammation

 e.g. acids, ammonia, chlorine
 Airway irritation

 e.g. dusts, fumes, mists, vapors, cold



#### Sensitizer-induced Asthma

Early response (HMW) (15-30 min)
Late response (LMW) (4-8 hours later)
Dual response (HMW)

#### Diagnosis

Confirming the diagnosis of asthma

 intermittent respiratory symptoms
 evidence of reversible airway
 obstruction

#### **2** Relationship between asthma & the work environment

- symptoms occur only at work
- symptoms improve on weekends or vacations
- symptoms occur regularly after work shift

- symptoms progressively increase over the course of the work week

- symptoms improve after a change in the work environment



 At least one of the symptoms (wheezing, shortness of breath, cough, chest tightness) should occur while the worker is at or within 4-8 hours of leaving the workplace

Note : With persistent exposure symptoms become chronic & lose relationship to work

## **Spirometry**

The most reliable method
May be normal between attacks
After bronchodilator : 12% increase in FEV1 is significant (hyper- responsiveness)
Across work shift : 10% fall in FEV1 is suggestive



◆ PEFR is the best way
 ◆ 4 times/day + symptoms + medications
 ◆ ≥ 20% diurnal variability is considered asthmatic response

#### Prevention

In all work places where cases are diagnosed
Removal from exposure
Engineering controls
Medical surveillance
Education
Smoking cessation program



## Hypersensitivity Pneumonitis Extrinsic Allergic Alveolitis

 Immunologically-mediated inflammatory disease of lung parenchyma

 Induced by inhalation of organic dusts containing the etiologic agents

#### **Causing Agents**

 Bacteria (Thermoactinomycetes in moldy hay, grain, sugar cane,...)

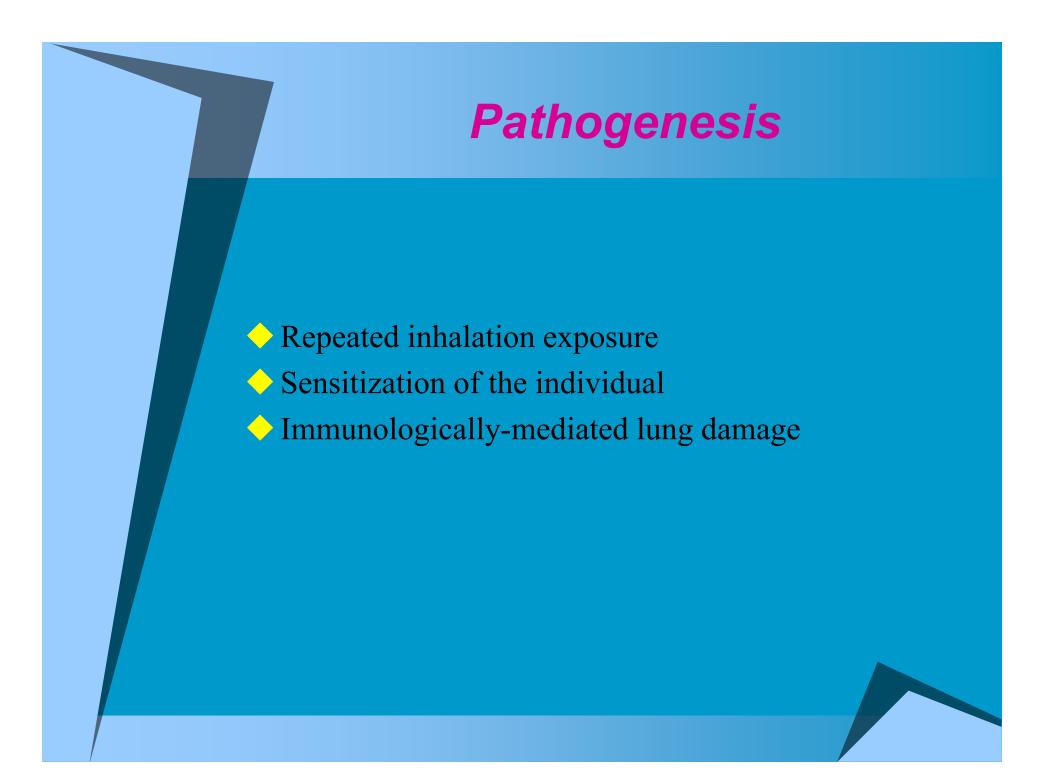
Fungi (Aspergillus in moldy malt, cheese,...)

Amoeba (Acanthamoeba in contaminated water)

 Animal proteins (bird feathers, urine, droppings,...)

 Chemicals (toluene diisocyanate, TMA, ... in polyurethane foams, epoxy resins,...)







Few exposed people develop it
Complete resolution in early removal
Progressive interstitial fibrosis in continued exposure



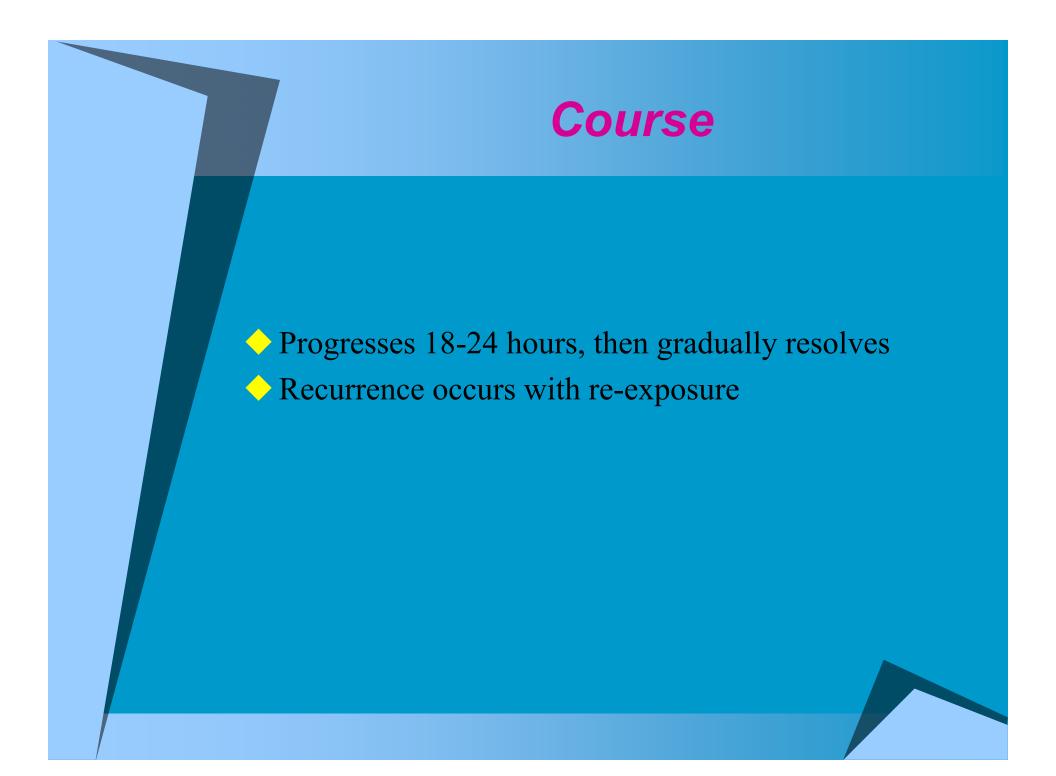
#### Acute HP

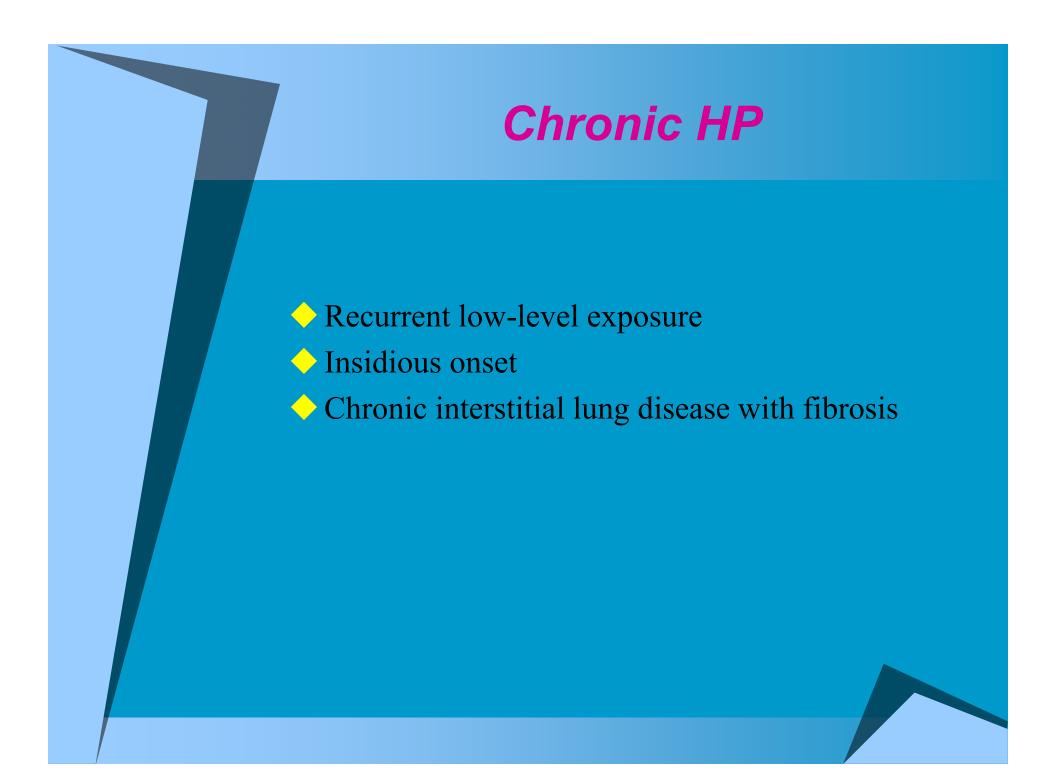
More common
4-6 hours of an intense exposure
Chills & fever
Malaise & myalgia
Dyspnea & cough
Headache
Bibasilar inspiratory crackles



#### LAB

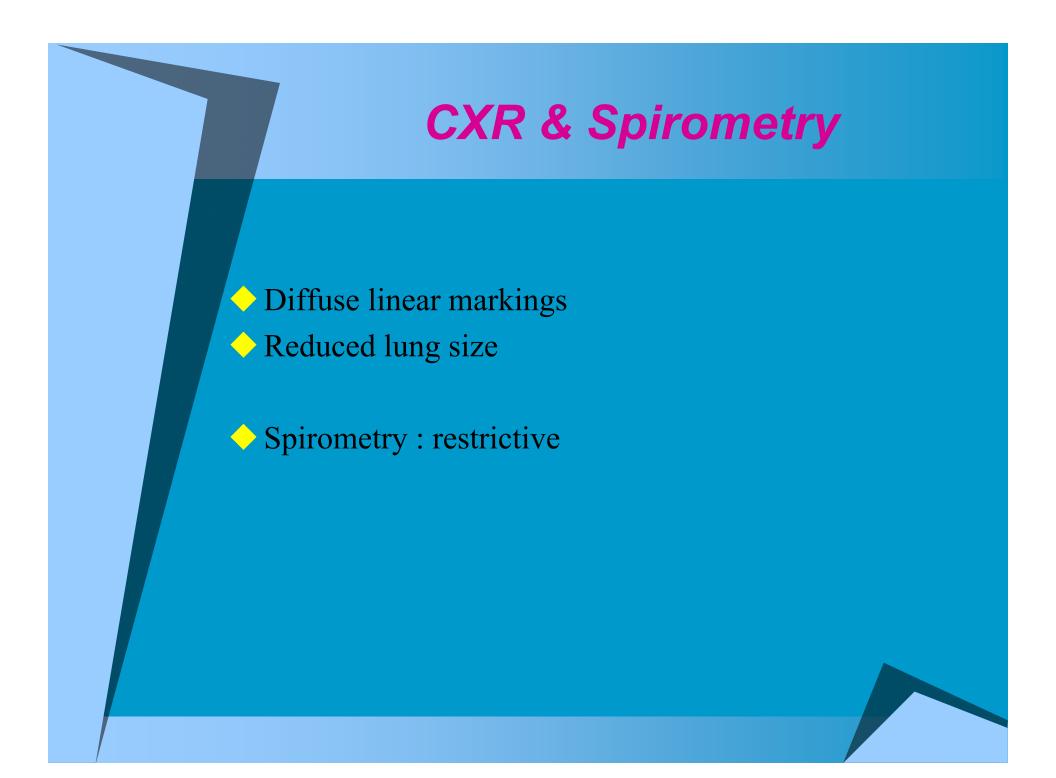
- Diff. : Leukocytosis (increased neutrophils, decreased lymphocytes)
- ♦ ABG : hypoxemia
- ♦ CXR : bilateral reticulonodular pattern
- ♦ Spirometry : restrictive





### Symptoms & Signs

Dyspnea & cough
Excessive fatigue
Weight loss
Cyanosis & clubbing
Inspiratory crackles



#### Treatment

Avoidance of exposure
 Respiratory protective equipment
 Corticosteroids (Prednisone 1mg/kg/day – 1 month) – follow with CXR & PFT
 Beta-agonists
 Oxygen

#### **Inhalation Fever**

- Exposure to polymer fumes, metal fumes, organic dusts (grain, cotton,...)
- ↔ High attack rate
- Most common cause : zinc oxide
- Chills, fever, myalgia, headache, malaise, cough, chest discomfort
- ♦ CXR, PFT, ABG : normal
- CBC : leukocytosis
- Treatment : symptomatic
- Complete resolution in 1-2 days
- No evidence for steroid therapy

# **Pneumoconioses** ♦ Silicosis ♦ Asbestosis Coal workers' pneumoconiosis

#### Pneumoconiosis

Literally means dust in lungs
Not all dusts cause disease
ILO : accumulation of dust in lungs and the tissue reaction to its presence

#### **Benign Pneumoconiosis**

Inert dusts contain < 1% quartz</li>
Do not disrupt alveolar architecture or give rise to collagenous fibrosis
No symptoms or functional abnormalities
May be cleared form lungs over a period of time with avoidance of exposure
e.g. iron, tin, antimony, barium

#### **Collagenous Pneumoconiosis**

 Dusts that stimulate a response in lungs and lead to irreversible fibrosis and structural alterations

↔ e.g. silicosis, asbestosis

#### **Chronic Bronchitis**

Minerals (coal, silica, cement,...)
Metals (vanadium, welding fumes,...)
Organic dusts (cotton, grain,...)
Smoke (tobacco, fire,...)
Esp. in smokers
Increased incidence among coworkers



#### **Pleural Disorders**

Benign pleural effusions
Pleural plaques
Mesothelioma
The primary cause is asbestos
Not always accompanied by asbestosis



## Lung Cancer

♦ Arsenic

- Chloromethyl ethers
- Chromium (hexavalent)
- ETS (synergism)
- ↔ Mustard gas
- Nickel
- ♦ PAH
- 🔶 Radon
- 🔶 Silica
- ↔ Beryllium

