PRINSIP DIAGNOSIS PENYAKIT PARU KERJA dan PNEUMOCONIOSIS

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FK UMM

OUTLINE:

- Definisi Paru Kerja
- Etiologi/Kausa/ faktor pencetus
- Langkah Diagnosis
- Tata laksana
- Definisi Pneumoconiosis
- Epidemiologi
- Patogenesis
- Tipe Pneumoconiosis
- Silicosis
- Asbestosis
- Anthracosis
- Pneumoconiosis tipe lain
- Preventive Measurement

DEFINISI PENYAKIT PARU KERJA

Penyakit atau kerusakan paru disebabkan oleh debu/ gas/ asap berbahaya yang terhirup oleh pekerja ditempat pekerjaan.

Etiologi / Kausa/ Faktor pencetus

1. Golongan fisika: suhu ekstrem, bising, pencahayaan,vibrasi, radiasi pengion dan non pengion, tekanan udara.
2. Golongan kimia: semua bahan kimia: debu, uap, uap logam, gas, larutan,kabut, partikel nano dll.
3. Golongan biologi: bakteri, virus, jamur, bioaerosol dll.

Etiologi / Kausa/ Faktor pencetus

4. Golongan ergonomi: angkat angkut berat, posisi kerja janggal, posisi kerja statis, gerak repetif, penerangan, *Visual Display Terminal (VDT),* dll.
5. Golongan psikososial: beban kerja kualitatif dan kuantitatif, organisasi kerja, kerja monoton, hubungan interpersonal,kerja *shift,* lokasi kerja. Lingkungan kerja yang mengakibatkan stres dapat merupakan pencetus asma

Prinsip Diagnosis

3 prinsip yang harus diperhatikan:

1. Hubungan antara pajanan yang spesifik dengan penyakit
2. Frek kejadian penyakit pada populasi pekerja lebih tinggi daripada di masyarakat
3. Penyakit dapat dicegah dengan tindakan promosi kesehatan dan pencegahan penyakit

7 Langkah Diagnosis Penyakit Paru akibat Kerja

1. Menegakkan Dx klinis penyakit paru:
   A. Anamnesis
      a. Riwayat pekerjaan
      b. Keluhan penyakit
      c. Riwayat penyakit
      d. Riwayat kebiasaan
   B. Pemeriksaan fisis: general dan lokal paru
   C. Pemeriksaan penunjang: rutin dan khusus

7 Langkah Diagnosis Penyakit Paru akibat Kerja

2. Menentukan pajanan yang dialami pekerja di tempat kerja:
   - Kronologis semua pekerjaan tenaga kerja
   - Lamanya pekerjaan
   - Bahan yang diproduksi
   - Bahan baku yg digunakan
   - Pemakaian APD
   - Pola waktu terjadinya gejala
   - Informasi tenaga kerja lain
   - Material safety data sheet

7 Langkah Diagnosis Penyakit Paru akibat Kerja

3. Menentukan hubungan antara pajanan dan Dx klinis

   Harus ada bukti ilmiah dalam kepustakaan yg mendukung bahwa pajanan menyebabkan penyakit paru.

7 Langkah Diagnosis Penyakit Paru akibat Kerja

4. Menentukan besar pajanan:
   A. Kualitatif
      - Observasi cara, proses, lingkungan kerja dengan memperhitungkan lama kerja
      - Pemakaian APD secara benar dan konsisten
   B. Kuantitatif
      - Data pengukuran lingkungan kerja
      - Data monitoring biologis

7 Langkah Diagnosis Penyakit Paru akibat Kerja

5. Menentukan faktor individu yg berperan
   a. Jenis kelamin
   b. Usia
   c. Kebiasaan
   d. Riwayat penyakit keluarga (genetik)
   e. Riwayat atopi
   f. Riwayat penyerta

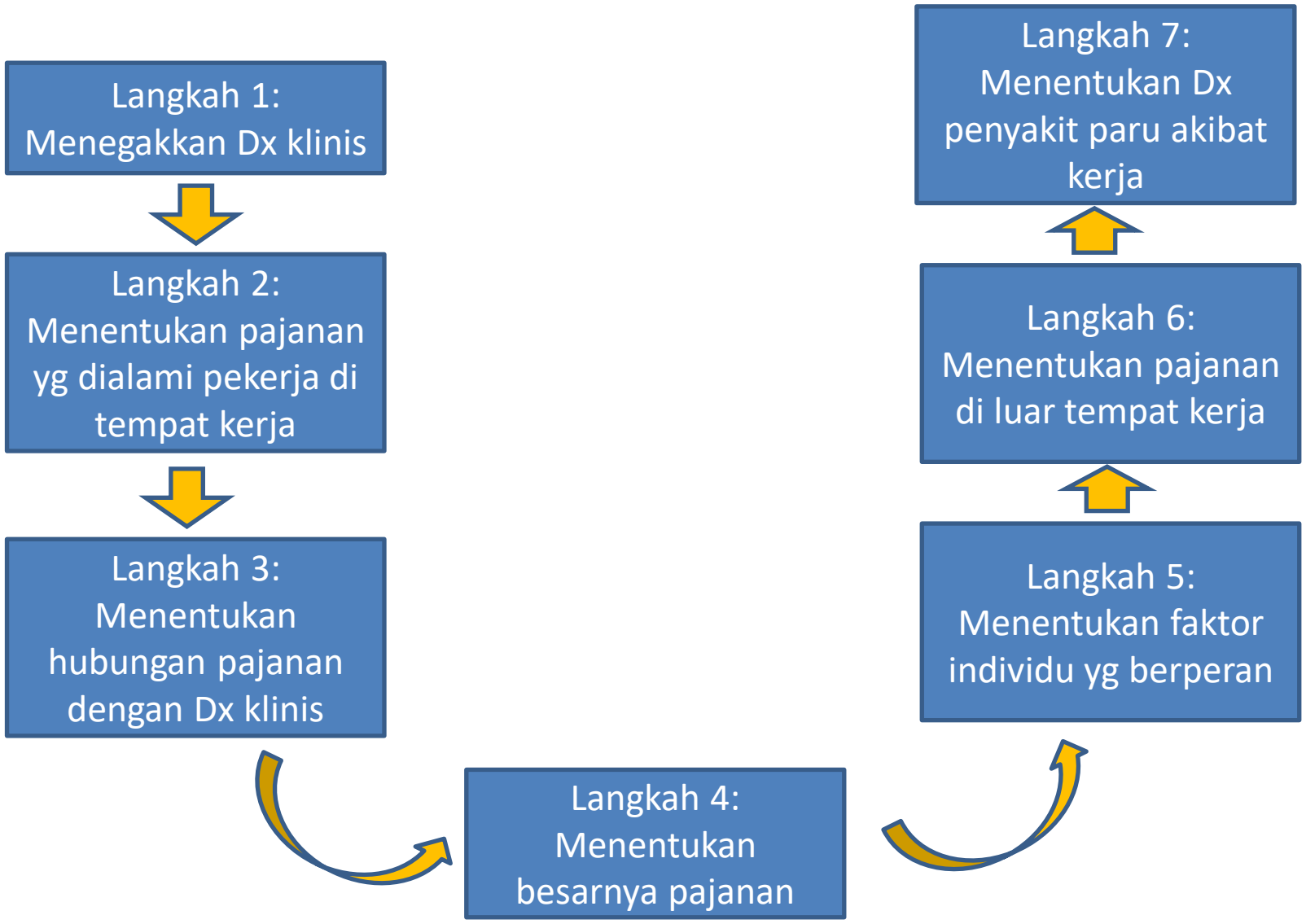
7 Langkah Diagnosis Penyakit Paru akibat Kerja

6. Menentukan pajanan di luar tempat kerja

   Informasi kegiatan di luar tempat kerja: hobi; pekerjaan rumah; pekerjaan sampingan

7. Menentukan diagnosis penyakit paru akibat kerja

7 Langkah Diagnosis Penyakit Paru akibat Kerja



Tata laksana

a. Pencegahan penyakit akibat kerja
b. Penemuan dini penyakit akibat kerja
c. Penentuan kelaikan kerja
d. Penentuan kembali bekerja
e. Penentuan kecacatan paru

Pneumoconiosis

Definitions

- The term *pneumoconiosis* derives its meaning from the Greek words: *pneuma* = air and *konis* = dust
- The International Labour Organization defines pneumoconiosis as "the accumulation of dust in the lungs and the tissue reactions to its presence".
- Not included in the definition of pneumoconiosis are conditions such as asthma, chronic obstructive pulmonary disease (COPD), and hypersensitivity pneumonitis, in which there is no requirement for dust to accumulate in the lungs in the long term.

Definitions

- Pneumoconiosis can be defined as the non-neoplastic reaction of lungs to inhaled minerals or organic dust and the resultant alteration in their structure excluding asthma, bronchitis and emphysema.

Textbook of Pulmonary Medicine , D Behera

Epidemiology

Globlal Burden of Disease Study 2013:

- Pnemoconiosis causes of death: 260.000
- (1990: 251.000 death)
- Silicosis : 46.000 death
- Asbestosis : 24.000 death
- Coal Worker Pneumoconiosis : 25.000 death

Lancet 385: 117 -71

Pathogenesis

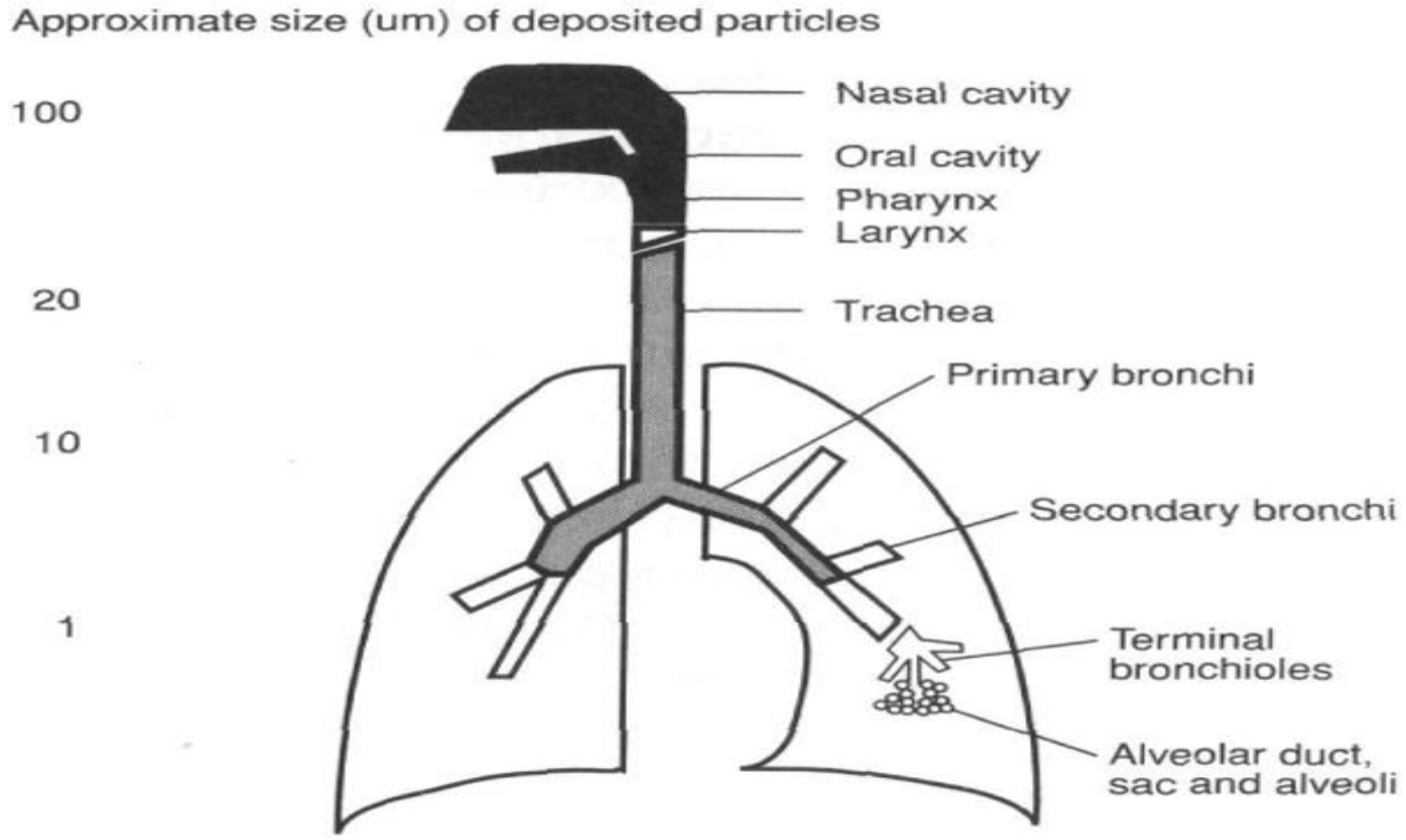
- Clinical pneumoconiosis to develop, 3 essential factors are required:
  - Exposure to specific substance: coal, appear relatively inert and may accumulate in considerable amounts with minimal tissue response; while silica and asbestos, have potent biologic effects.
  - Particles of appropriate size to be retained in lung (1-5μm)
  - Exposure for a sufficient length of time (usually around 10 years)

Pathogenesis

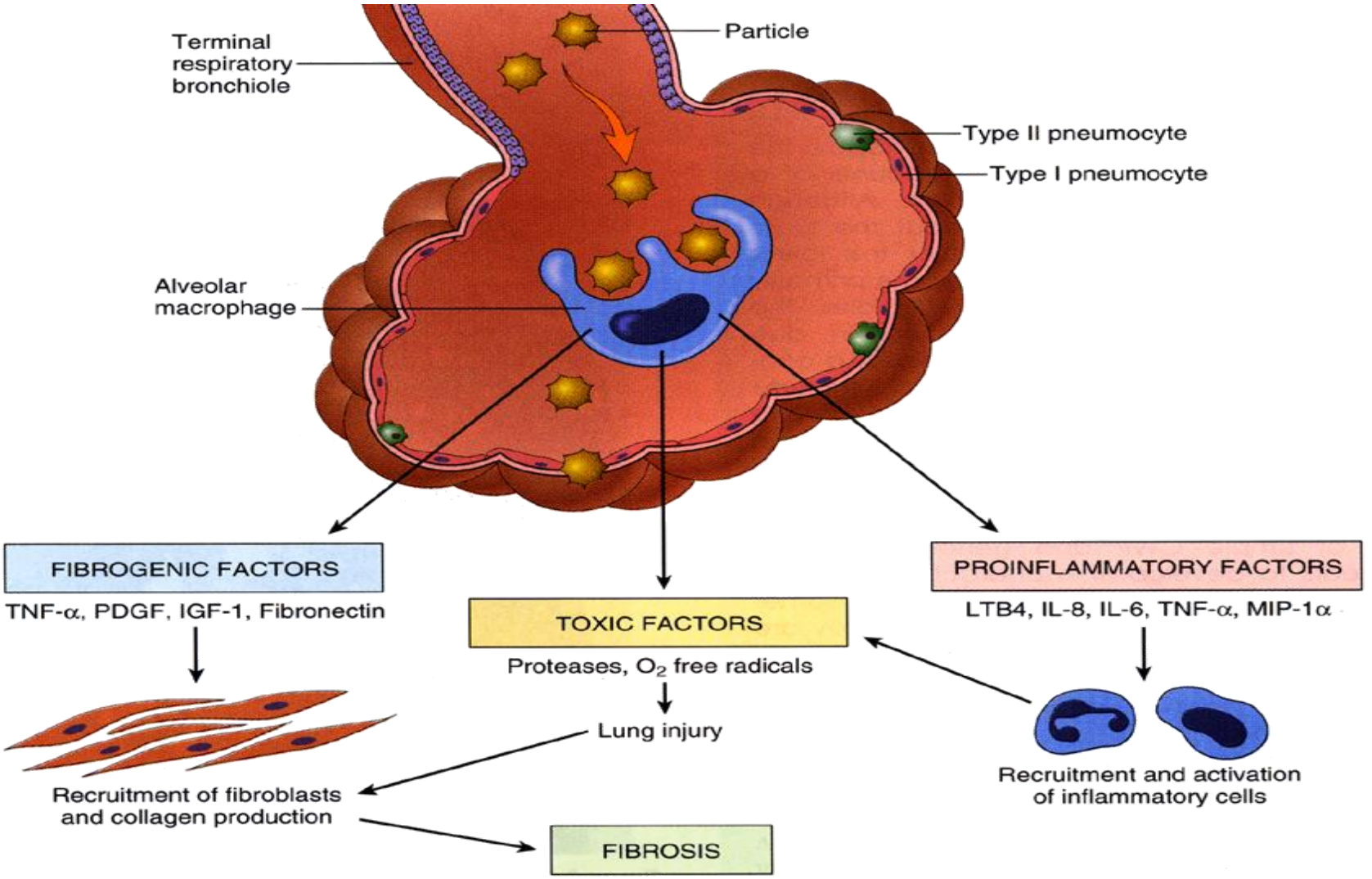
Dust is classified by size into following categories:

- Inhalable Dust: is the one which enters the body, but is trapped in the nose, throat, and upper respiratory tract. Particle size is usually 6-25µm.
- Respirable Dust: particles that are small enough to penetrate the nose and upper respiratory system beyond the body's natural clearance mechanisms of cilia and mucous and are more likely to be retained in the lungs. Particle size is usually 1-5µm.
- Particles of <1 µm are exhaled out.

Pathogenesis



Pathogenesis



Pathogenesis

- Dust inhalation
- Escape removal by defence apparatus
- Particles penetrate epithelium → direct injury
- Fibrosis
- Engulfment by alveolar & interstitial macrophages → lymphatics → lymph node (modify immune response)

Types

- Silicosis – from silica dust
- Asbestosis – from asbestos dust
- Coal workers pneumoconiosis (anthracosis) – from coal dust
- Byssinosis – from cotton dust
- Bagassosis – from sugarcane dust
- Farmer's lung - from hay dust or mold spores or other agricultural products.
- Berylliosis – from beryllium

Types

- Siderosis – from iron oxide
- Tanosis – from tin oxide
- Talcosis – from talc (hydrated magnesium silicate)
- Bauxite fibrosis – from bauxite dust
- Mixed dust pneumoconiosis – from a mixture of dusts
- Hard metal pneumoconiosis – from certain metals like cobalt
- In addition, others dust such as aluminum, barium, antimony, graphite, kaolin and mica can also cause pneumoconiosis.

Types

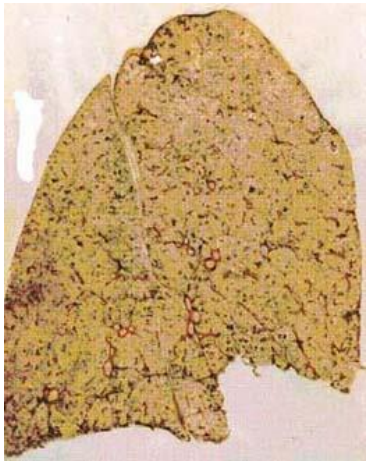
- Pneumoconiosis is usually divided into three groups:
  - Major pneumoconiosis
  - Minor pneumoconiosis

“ Fibrotic Pneumoconiosis”

  - Benign pneumoconiosis

Types

- Major Pneumoconiosis: Inhalation of some dusts results in "major fibrosis" of the lungs, which results in interference of lung architecture or lung function tests.
- Examples are:
  - Silica → silicosis
  - Asbestos → asbestosis
  - Coal → anthracosis



Healthy lung

Silicotic lung

Types

- Minor Pneumoconiosis: Inhalation of some dusts results in "minor fibrosis" of the lungs
- There is minimal fibrosis of the lungs without interference of lung architecture or lung function tests.
- These dusts include:
  - Mica pneumoconiosis
  - Koalin (china clay) pneumoconiosis

Types

- Benign Pneumoconiosis: There isn't any reaction in the lungs, but dust deposition casts a shadow in x-ray of the lung. There is no fibrosis and no disturbance of lung functions.
- It can result from the inhalation of:
  - Iron dust→ siderosis
  - Tin dust→ stannosis
  - Calcium dust→ chalcosis
- They are characterized by the presence of small rounded dense opacities on a chest film due to perivascular collections of dusts.
- The deposits in the lung disappear when exposure is discontinued.

TABLE 15–6 Lung Diseases Caused by Air Pollutants

Agent	Disease	Exposure
Mineral Dusts		
Coal dust	Anthracosis Macules Progressive massive fibrosis Caplan syndrome	Coal mining (particularly hard coal)
Silica	Silicosis Caplan syndrome	Foundry work, sandblasting, hardrock mining, stone cutting, others
Asbestos	Asbestosis Pleural plaques Caplan syndrome Mesothelioma Carcinoma of the lung, larynx, stomach, colon	Mining, milling, and fabrication; installation and removal of insulation
Beryllium	Acute berylliosis Beryllium granulomatosis Bronchogenic carcinoma (?)	Mining, fabrication
Iron oxide	Siderosis	Welding
Barium sulfate	Baritosis	Mining
Tin oxide	Stannosis	Mining
Organic Dusts That Induce Hypersensitivity Pneumonitis		
Moldy hay	Farmer's lung	Farming
Bagasse	Bagassosis	Manufacturing wallboard, paper
Bird droppings	Bird-breeder's lung	Bird handling
Organic Dusts That Induce Asthma		
Cotton, flax, hemp	Byssinosis	Textile manufacturing
Red cedar dust	Asthma	Lumbering, carpentry
Chemical Fumes and Vapors		
Nitrous oxide, sulfur dioxide, ammonia, benzene, insecticides	Bronchitis, asthma Pulmonary edema ARDS* Mucosal injury Fulminant poisoning	Occupational and accidental exposure

SILICOSIS

Silicosis

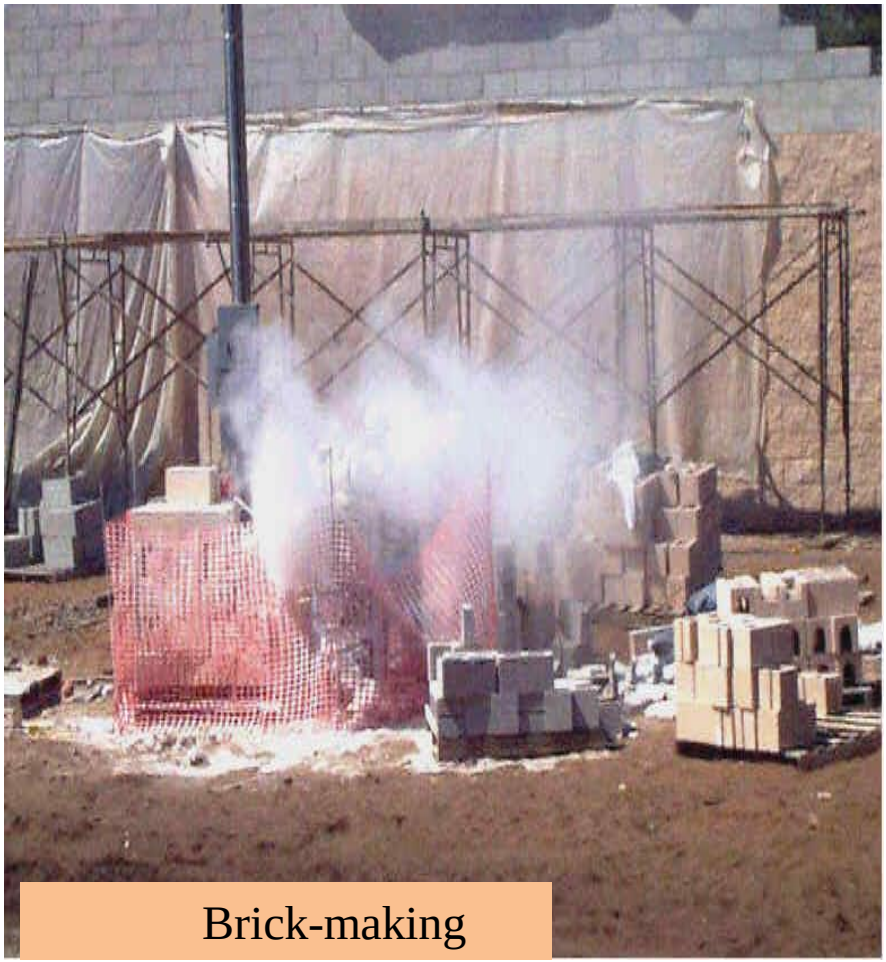
- Develops with repeated and usually long-term exposure to crystalline silica (silica dust)
- The silica dust causes irritation and inflammation of the airways and lung tissue.
- Scar tissue forms when the inflammation heals, resulting in fibrosis that gradually overtakes healthy lung tissue.
- The fibrosis continues extending through the lungs even after exposure ends.

Silicosis

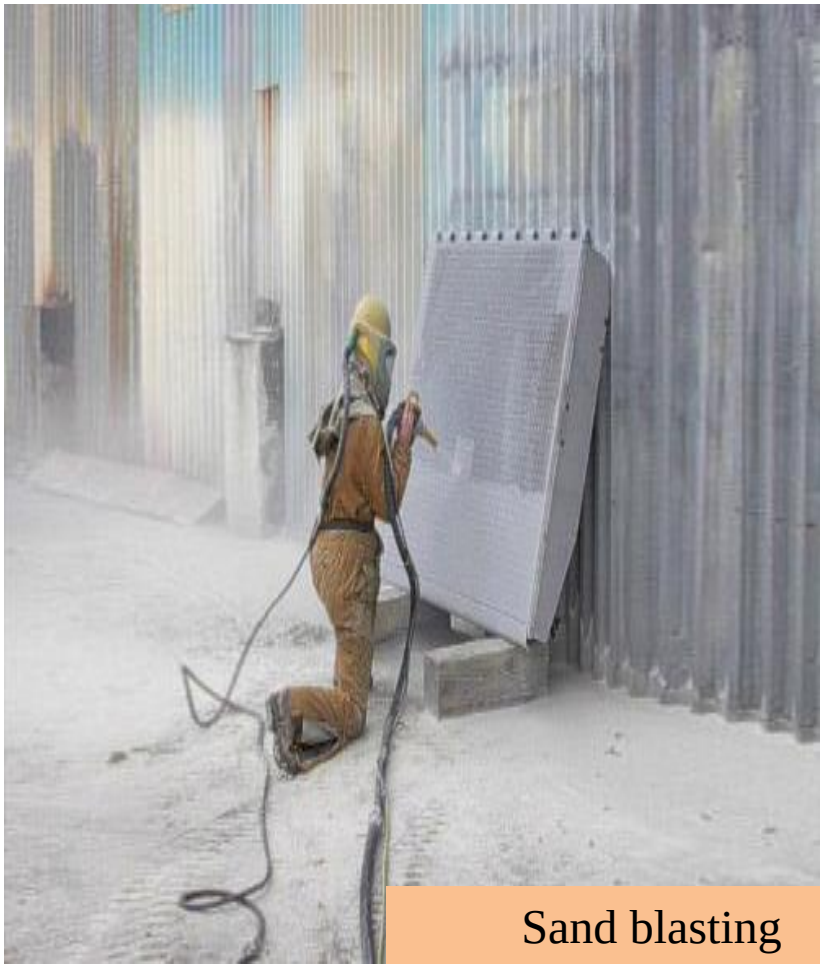
Occupations with exposure to silica dust

- Mining
- Tunnelling
- Quarrying
- Sandblasting
- Ceramics
- Brick-making
- Silica flour manufacture
- Slate Pencil Industry
- Agate Industry
- Quartz Grinding

Silicosis



Brick-making



Sand blasting

Silicosis

- Three forms of silicosis:
  - Acute silicosis: occurs with exposure to fine dust with high quartz content; very heavy exposure for months, shows symptoms within weeks to months of exposure,
  - Accelerated silicosis: shows rapidly progressive symptoms after 5 to 10 years of high exposure to fine dust of high silica content.
  - Chronic silicosis: the most common form, results from long-term exposure (10 to 20 years or longer) to dust containing less than 30% silica content.

Silicosis

- Clinical features:
  - Chronic cough
  - Dyspnea (shortness of breath) that worsens with exertion.
  - Fatigue
  - Loss of appetite
  - Chest pains
  - Acute silicosis patients may also have fever and experience rapid, unintended weight loss.
- **Silicotuberculosis:**
  - Pulmonary tuberculosis occurs in about 25% of patients with acute or classic silicosis
- **"Eggshell"** calcification, when present, is strongly suggestive of silicosis

- On histopathology the hallmark of silicosis is the silicotic nodule

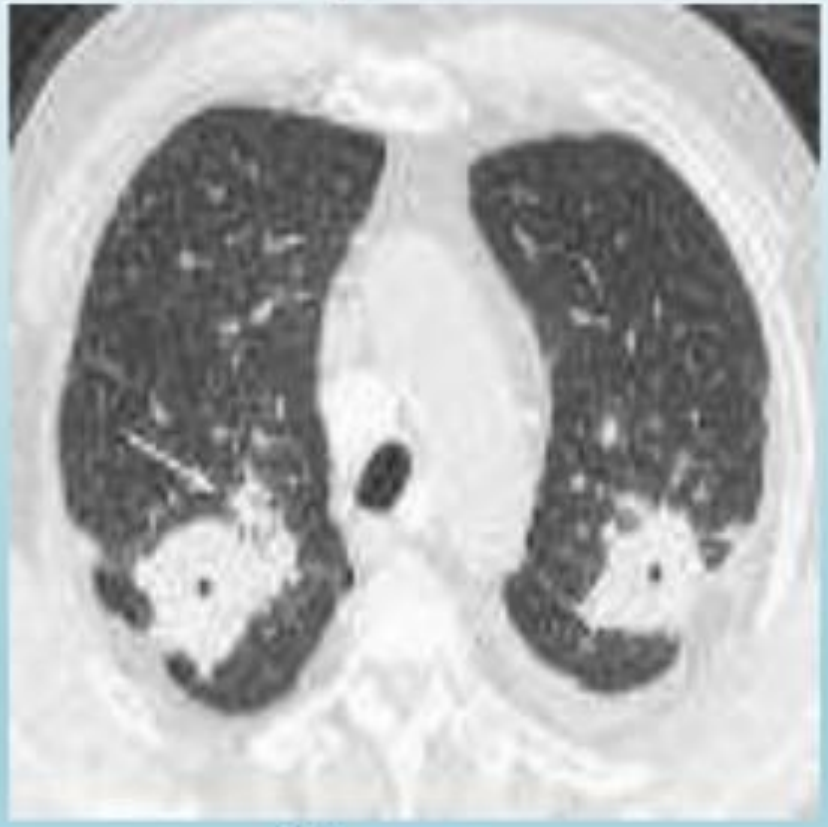
Simple Silicosis

normal chest x-ray

simple silicosis

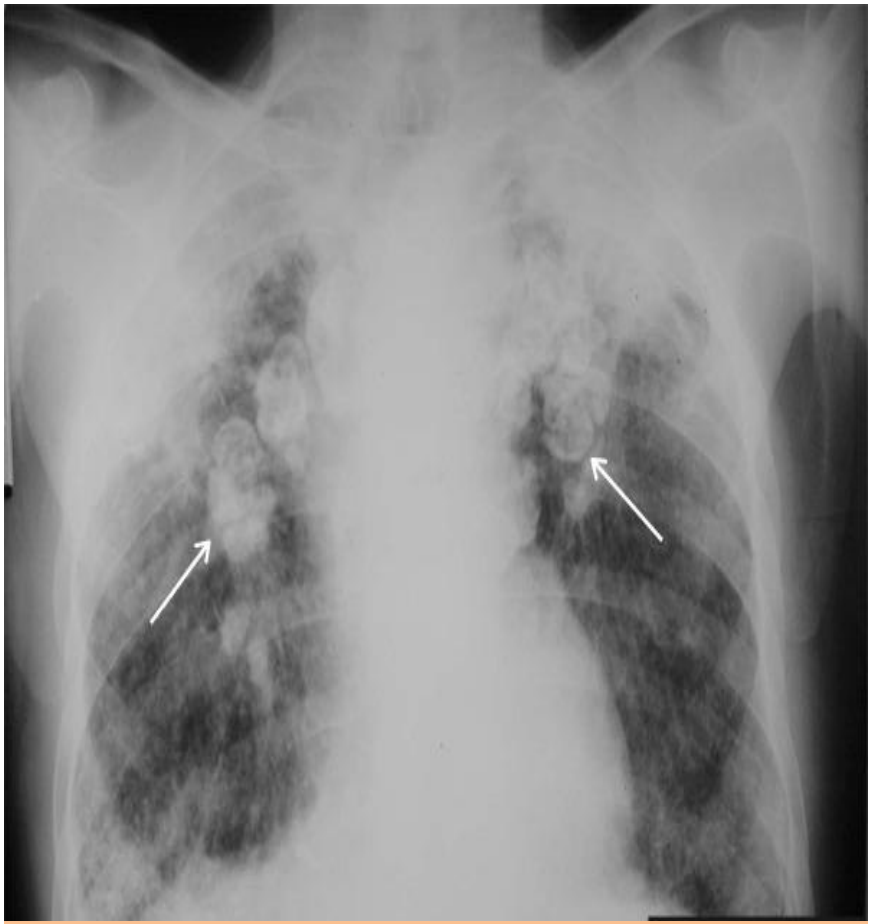
Accelerated Silicosis (PMF)

chest x-ray

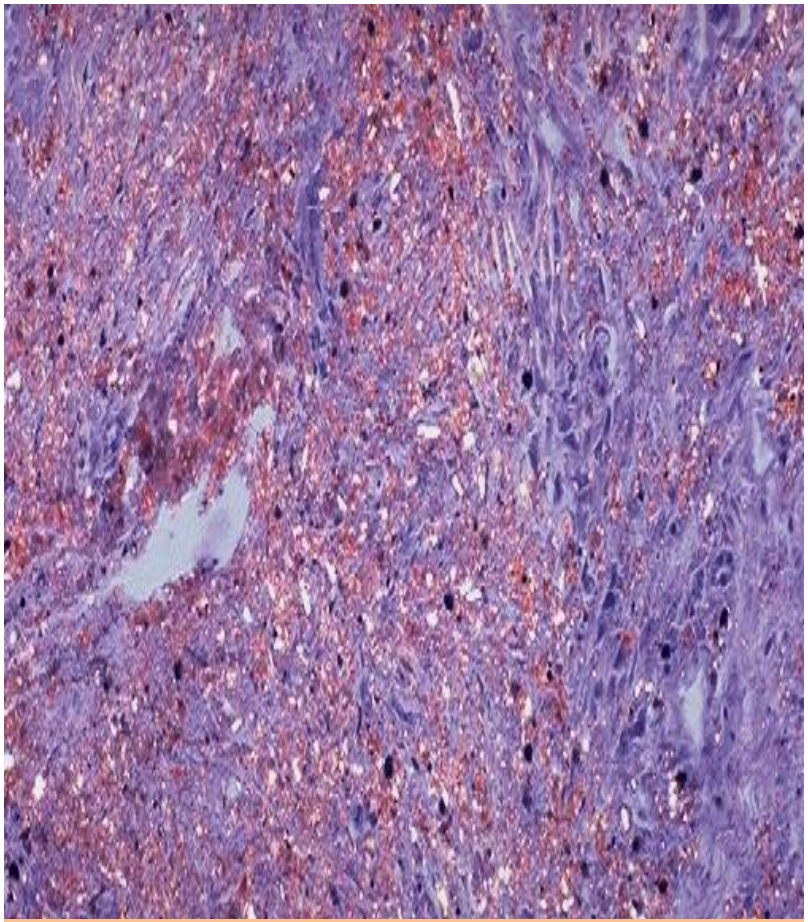


CT scan

Silicosis



Chest radiography showing Eggshell calcification



Polarized light microscopy showing Crystals of silica

Silicosis

- Treatment:
  - There is no specific treatment for the silicosis,
  - There is no known method of intervention to prevent the condition's progression.
  - Silica exposure has to be stopped to prevent further damage to the lungs,
  - Smokers should quit smoking.
  - Tuberculosis positive patients need to be put on anti-tuberculosis treatment
  - The course of progression often extends over decades even after cessation of exposure.
  - Prevention remains the most effective therapeutic approach.

ASBESTOSIS

ASBESTOSIS

- Asbestosis is diffuse interstitial pulmonary fibrosis that occurs secondary to the inhalation of asbestos fibers.
- It is considered separately from other asbestos-related diseases, such as benign pleural effusion and plaques, malignant mesothelioma, and bronchogenic carcinoma.
- Asbestos is classified into two groups: serpentine and amphibole.

ASBESTOSIS

Serpentine (93% of commercial use)	Amphibole (7% of commercial use)
Chrysolite	Actinolite, Amosite, Anthophyllite, Crocidolite, Richterite, Tremolite

Forms of asbestos

Serpentine

- Curly,more used in industry e.g.chrysotile.
- less pathogenic
- Breaks into fragments
- Fibrogenic
- Impacts in upper airways & removed by mucociliary apparatus & more soluble-leached out
- Not associated with mesothelioma

Amphibole

- straight & stiff
- e.g. crocidolite
- more pathogenic
- Resists breaking into fragments
- Fibrogenic
- Align in airstream & go deep ,penetrate epithelium,enter interstitium
- <0.5 µm thick,>8 µm long more fibrogenic
- Associated with mesothelioma

ASBESTOSIS

- Significant occupational exposure to asbestos occurs mainly in
  - Asbestos cement factories
  - Asbestos textile industry a

Asbestos cement factories

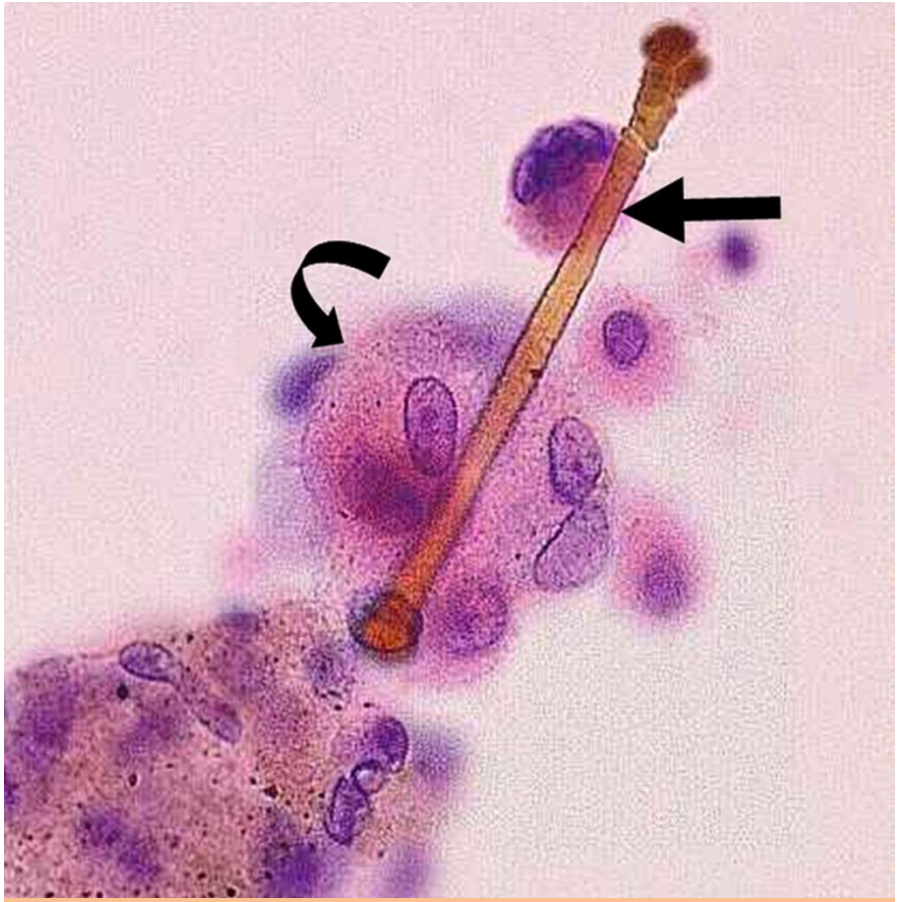
Asbestos textile industry

Asbestos mining

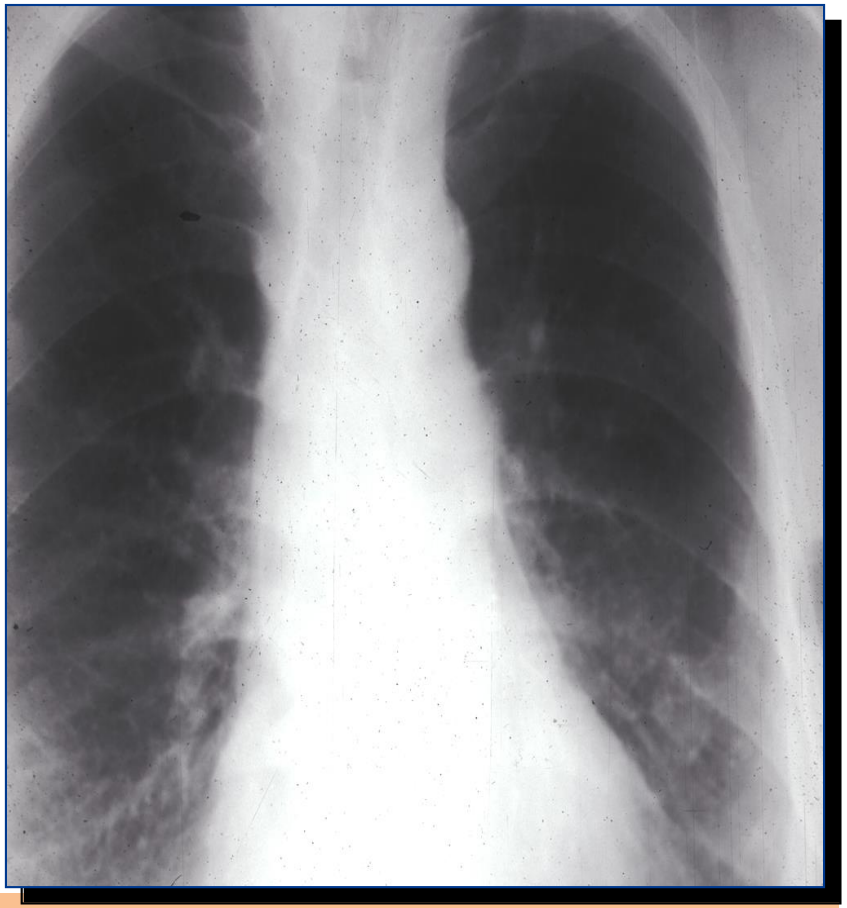
ASBESTOSIS

- Symptoms
  - Average latency period is 20-30 years
  - Dyspnoea
  - Cough
  - Chest pain
  - In advanced cases, clubbing of fingers
- At histopathologic analysis, asbestos bodies, which may consist of a single asbestos fiber surrounded by a segmented protein-iron coat, can be identified in intraalveolar macrophages.

ASBESTOSIS



Translucent asbestos fiber (straight arrow) surrounded by a protein-iron coat and an alveolar macrophage (curved arrow)



Chest x-ray showing Small, irregular oval opacities Interstitial fibrosis and “Shaggy heart sign”

Diagnosis of Asbestosis

Chest X- Ray :

Interstitial pneumoscelerosis

Diagnostic Particularities:

a) In sputum - asbestos bodies
b) In skin - asbestos Warts
(containing asbestos)

PLEURAL PLAQUE OF ASBESTOSIS

FIGURE 1. A chest radiograph reveals bilateral, rounded, calcified pleural plaques.

FIGURE 2. Pleural plaques seen on CT

ASBESTOSIS

- Treatment Strategy:
  - Stopping additional exposure
  - Careful monitoring to facilitate early diagnosis
  - Smoking cessation
  - Regular influenza and pneumococcal vaccines
  - Disability assessment
  - Pulmonary rehabilitation as needed
  - Aggressive treatment of respiratory infections
  - Health education to patient

ANTHRACOSIS

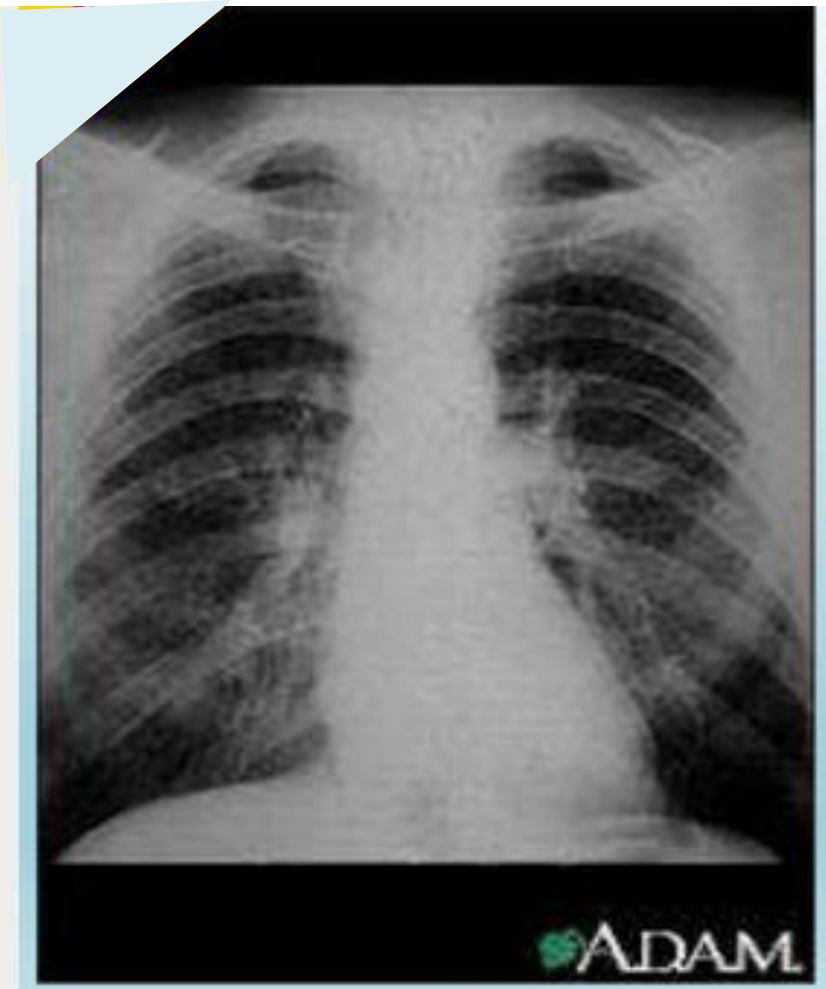
Anthracosis

- **Anthracosis / Coal Worker's Pneumoconiosis (CWP) / Black lung disease**:
  - Accumulation of coal dust in the lungs and the tissue's reaction to its presence.
  - Associated with coal mining industry
  - Takes one or two decades to cause symptoms
  - The disease is divided into 2 categories:
    - ***Simple CWP*** and
    - ***Complicated CWP*** or

      Progressive Massive Fibrosis (PMF).

Anthracosis

- ***Simple Coal Worker's Pneumoconiosis***:
  - The presence of radiological opacities < 1cm in diameter.
  - Benign disease if no complications.
  - Common symptoms: cough, expectoration (black in colour) and dyspnea.
  - Slight decrease in FVC and FEV1/FVC



Simple CWP

Minute opacities are diffusely scatterred throughout both lung fields, providing a crude measure of excessive exposure.
Early pneumoconiosis is essentially a focal disorder and may produce little physiologycal disorders

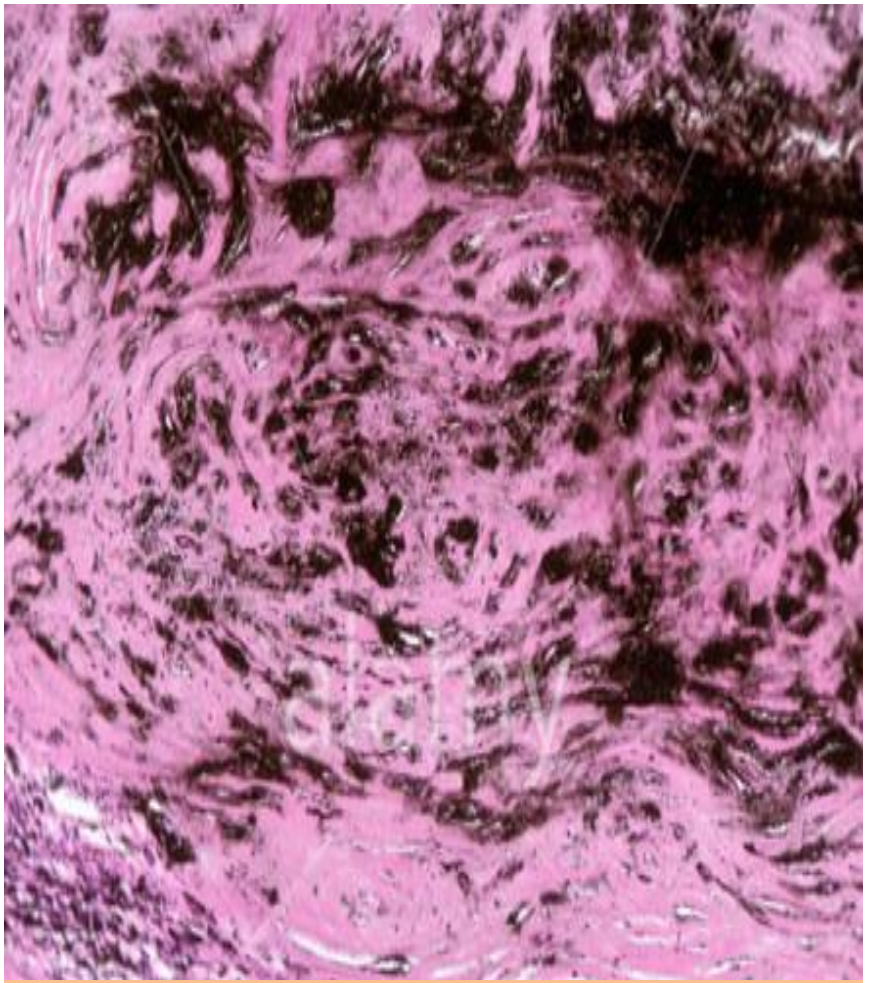
Anthracosis

- ***Complicated Coal Worker's Pneumoconiosis***
  - Large opacity of ≥ 1cm in diameter is observed in the CXR
  - Pathologically it is characterized by large masses of black colored fibrous tissue.
  - Symptoms are similar but more severe
  - Recurrent pulmonary infection
  - The large lesions may cavitate as a result of ischemic necrosis or infection (TB).
  - PFT (Pulmonary function test) reveals decreased FVC, FEV1/FVC and increased residual volume.

These pictures show **complicated coal workers pneumoconiosis**. There are diffuse, small, light areas (more than 1 cm) in all areas on both sides of the lungs. There are large light areas which run together with poorly defined borders in the upper areas on both sides of the lungs.

Anthracosis

Cut section of lungs in anthracosis



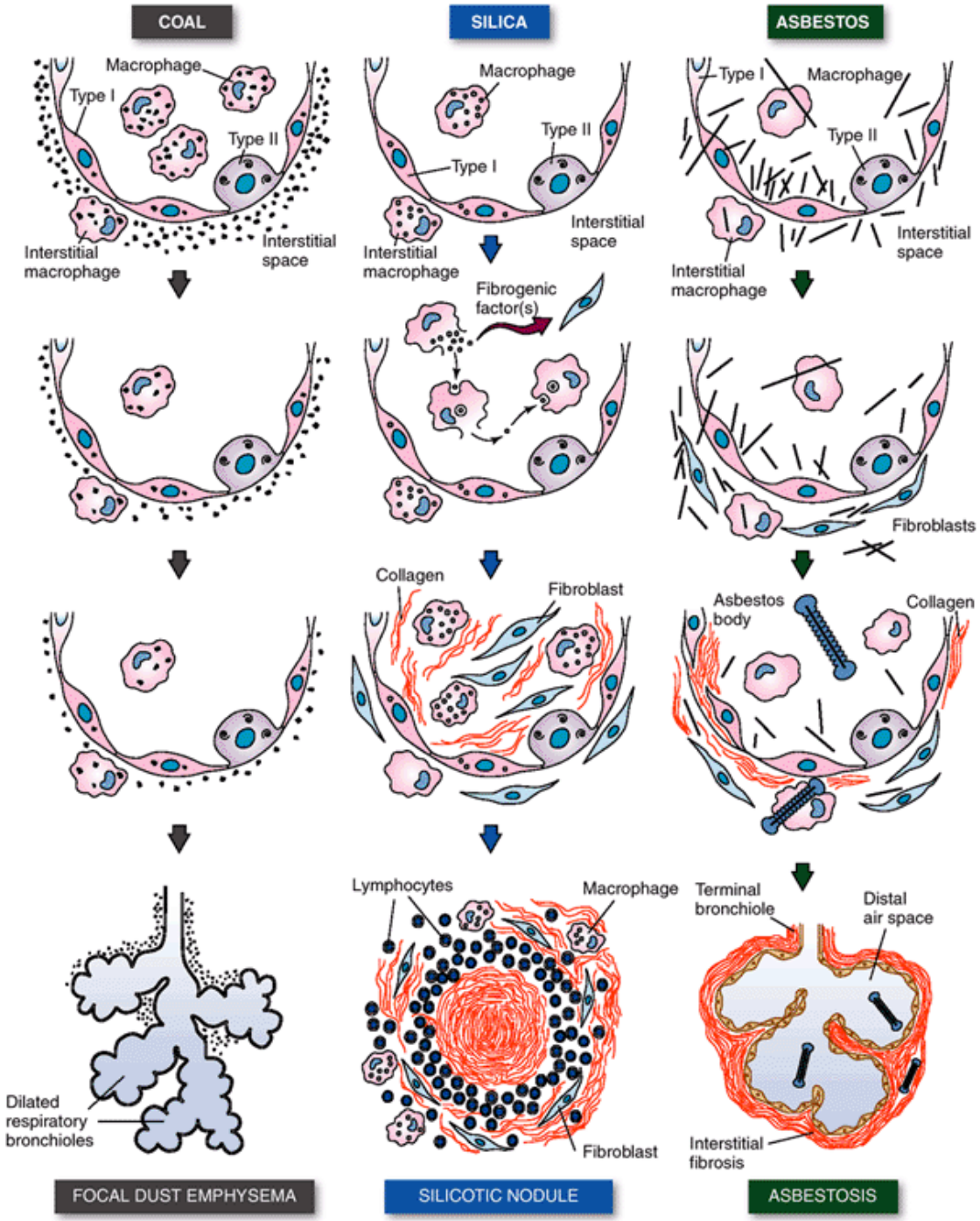
On histopathological examination

If coal worker's pneumoconiosis occurs with rheumatoid arthritis it is called Caplan syndrome.

Caplan's syndrome (or **Caplan's disease**) is a combination of rheumatoid arthritis and pneumoconiosis that manifests as intrapulmonary nodules, which appear homogenous and well-defined on chest X-ray

Caplan's syndrome presents with

- Cough, shortness of breath
- features of rheumatoid arthritis (painful joints and morning stiffness)
- Examination should reveal tender, swollen MCP joints and rheumatoid nodules
- Auscultation of the chest may reveal diffuse rales that do not disappear on coughing or taking a deep breath.


COAL
SILICA
ASBESTOS
Macrophage
Type I
Type II
Interstitial macrophage
Interstitial space
Fibrogenic factor(s)
Fibroblasts
Collagen
Fibroblast
Asbestos body
Lymphocytes
Terminal bronchiole
Distal air space
Dilated respiratory bronchioles
Interstitial fibrosis
FOCAL DUST EMPHYSEMA
SILICOTIC NODULE
ASBESTOSIS

OTHER TYPES OF OCCUPATIONAL LUNG DISEASE

Other types of occupational lung disease

Byssinosis

- Byssinosis is a narrowing of the airways caused by inhaling cotton, flax, or hemp particles.
- The substance or substances in the material that cause the disease are not known, but it is believed that the protein component rather than the cellulose or mineral constituents is responsible

Other types of occupational lung disease

Hypersensitivity Pneumonitis

- Hypersensitivity Pneumonitis (also referred to as "extrinsic allergic alveolitis") is an immunologic-induced, non-IgE mediated inflammatory pulmonary disease. It affects primarily the interstitium, alveoli, and terminal airways, and is caused by prolonged, repeated inhalation of organic dusts or certain chemicals (Farmer's lung, Bagassosis

Other types of occupational lung disease

Occupational Asthma

- Reversible airflow obstruction caused by workplace exposures
- With latency period (sensitization)
- Without latency period (irritant)
- Causes: a broad group of vegetable, animal products, chemicals, metals-referred to as "asthmagens"

PREVENTIVE MEASURE

Preventive measures

- Preventive measures:
  - Medical measures
  - Engineering measures
  - Other measures

Preventive measures

- ***Medical measures***:
  - Pre-placement examination
  - Periodical examination
  - Medical and health care services
  - Notification
  - Maintenance and analysis of records
  - Health education and counselling
  - Practicing good personal hygiene

Preventive measures

- Practicing good personal hygiene:
  - Washing hands and face before eating, drinking, going to the toilet, smoking.
  - Do not eat, drink, smoke, or apply cosmetics in areas where silica is being used.
  - Wear protective clothes and respiratory protection (Respirators must fit tightly.)
  - Before leaving work, shower and change into clean clothes. Leave dusty clothes at work.

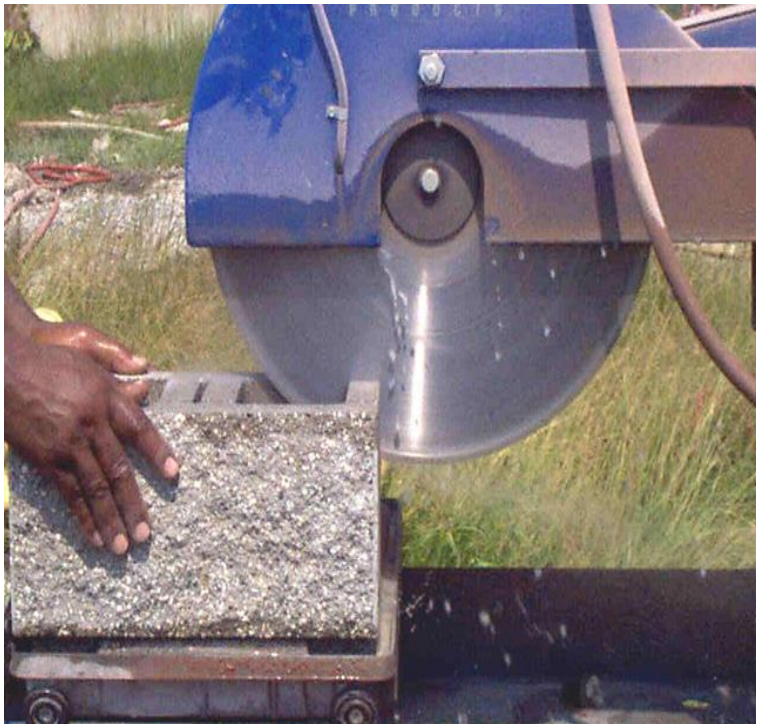
Preventive measures

- ***Engineering measures***
  - Design of building
  - Conduct air monitoring to measure the workers' exposure to crystalline silica.
  - Minimize exposures by controlling the creation of airborne particles, for example, use wet drilling, local exhaust ventilation.
  - Personal Protective Equipments: Provide workers with protective clothes, respiratory protection, and facilities for washing (showers) and changing.
  - Enclosure / isolation
  - Environmental monitoring

Preventive measures

Prohibit Dry Cutting

Promote wet Cutting



Preventive measures

Personal Protective Equipments

Tyvek suit

Respirator

Gloves

Goggles

Boots

Preventive measures

Fume extractor system

Labeling of products

Prevention of occupational lung diseases

Ventilation and exhaust systems

Preventive measures

- ***Other measures***:
  - Legal measures: Measures to minimize dust emissions and exposure to dust.
  - Law compliance mechanisms, including effective workplace inspection systems
  - Cooperation between management and workers and their representatives
  - A mechanism for the collection and analysis of data on occupational diseases
  - Collaboration with social security schemes covering occupational injuries and diseases

Preventive measures

- ***Other measures***:
  - Training of health professionals in occupational diseases as majority of medical practitioners lack training in occupational health and consequently lack the skills to diagnose and prevent occupational diseases.


THANK YOU