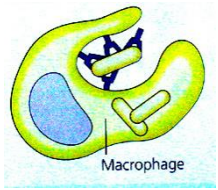
OCCUPATIONAL PNEUMOLOGY

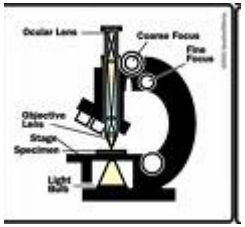
S I L I C O S I S

- Etiology: parenchymal lung disease caused by inhalation of crystalline silica from rocks and sand
- Exposure: mining, tunnelling, stone cutting, foundry work (abrasive sandblasting), ceramic industry,
- textil industry (sanding to make old looking jeans) in unsatisfactory conditions



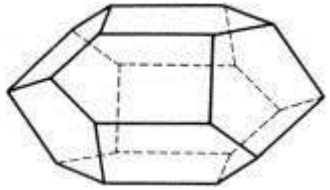
Pathogenesis:

- toxic interaction between silica crystals and alveolar macrophages :
- - macrophage release MFF(macrophage fibrogenic factor), cytokines Il-1, IL-6
- proliferation of fibroblasts
- permanent synthesis of collagen
- silicotic nodule –microscopic unit



Quartz – almost pure silica (SiO_2)

Hard mineral composed of silica and found in many different rocks (granit). Quartz be different color according to the size and purity of the crystals. Crystals of total quartz are colorless, and transparent.



Silica is present in

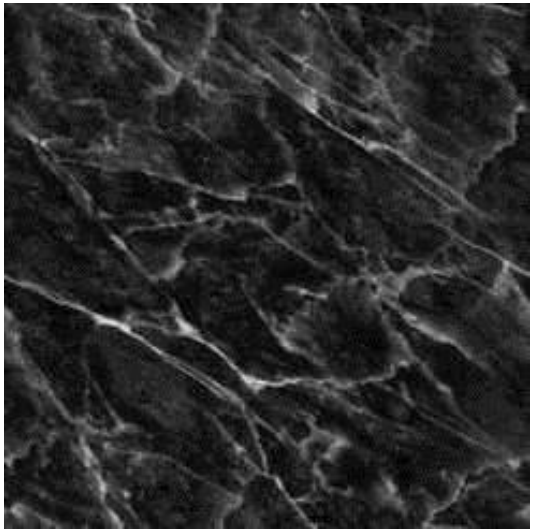
- **granit**

sandstone

Not fibrogenic calcium containing minerals

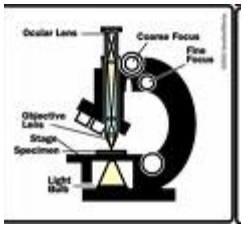
- Limestone

Marbre



silicotic nodule

onion-like structure



silicotic nodule :
silica crystals in macrophages in the middle

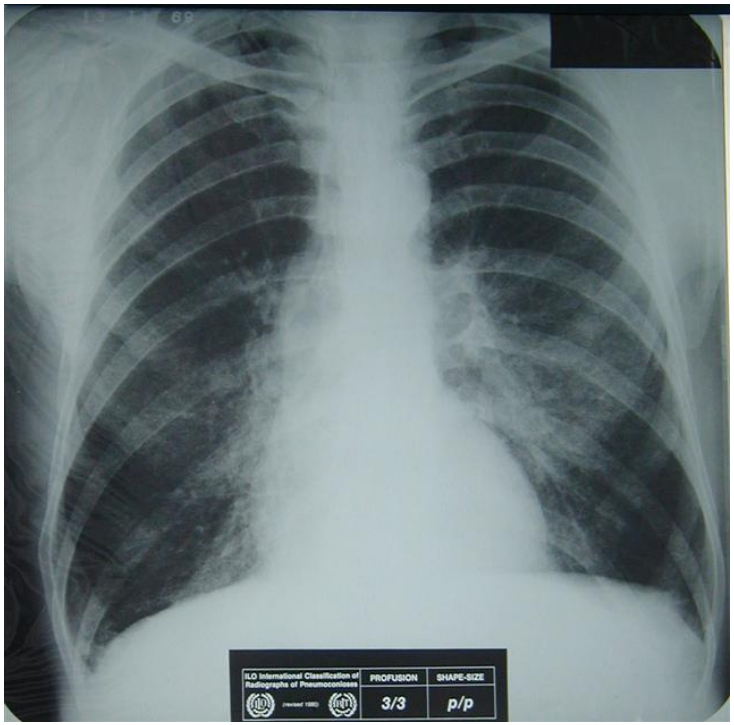
acellular core (swirls of collagen, hyalinisation)

cellular capsule (macrophages, fibroblasts)

1. Simple silicosis

nodules of diameter less than 1 cm

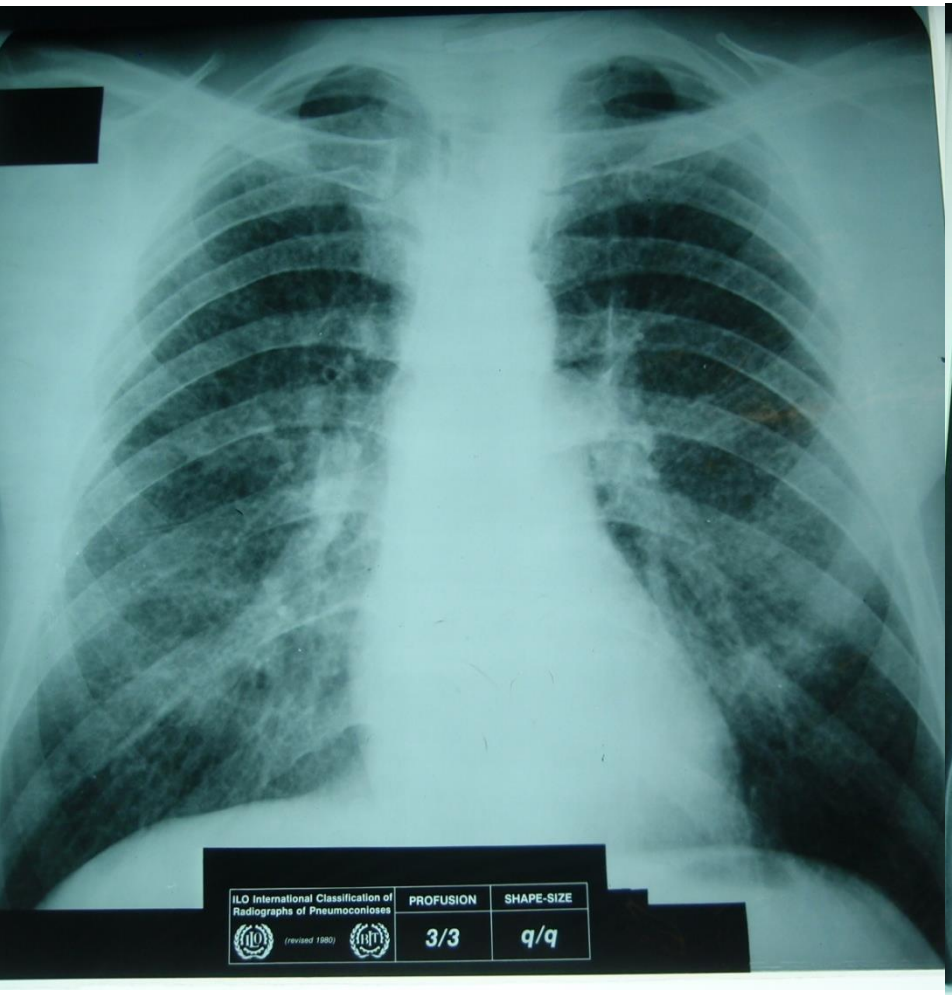
- Usually asymptomatic – to diagnose - preventive examinations, follow-up examinations
- rounded opacities
- **ILO classification**
- **Size:**
- **p - diameter up to 1.5 mm**
- **q - diameter up to 3 mm**
- **r - diameter up to 10 mm**
- generalized nodulation
- **Profusion (density) 1-3**



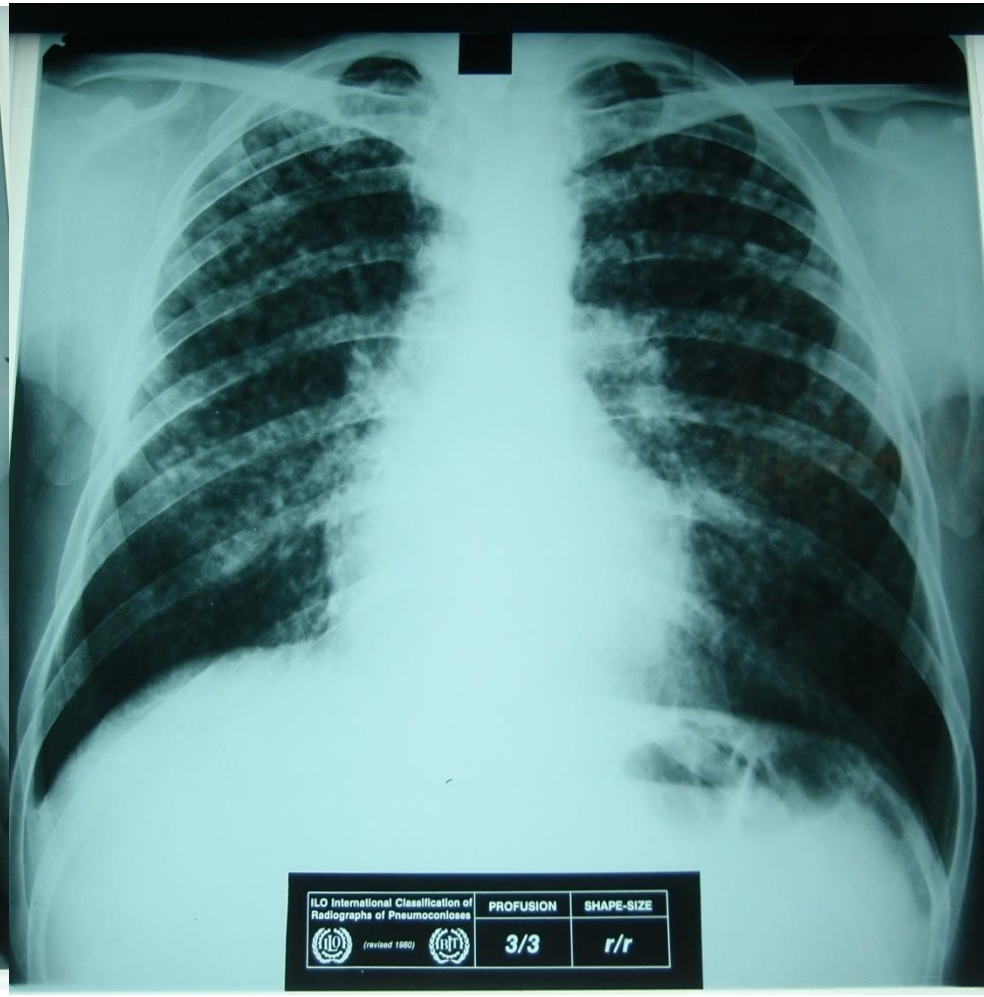
Simple silicosis p3

Simple silicosis with typical signs

q3

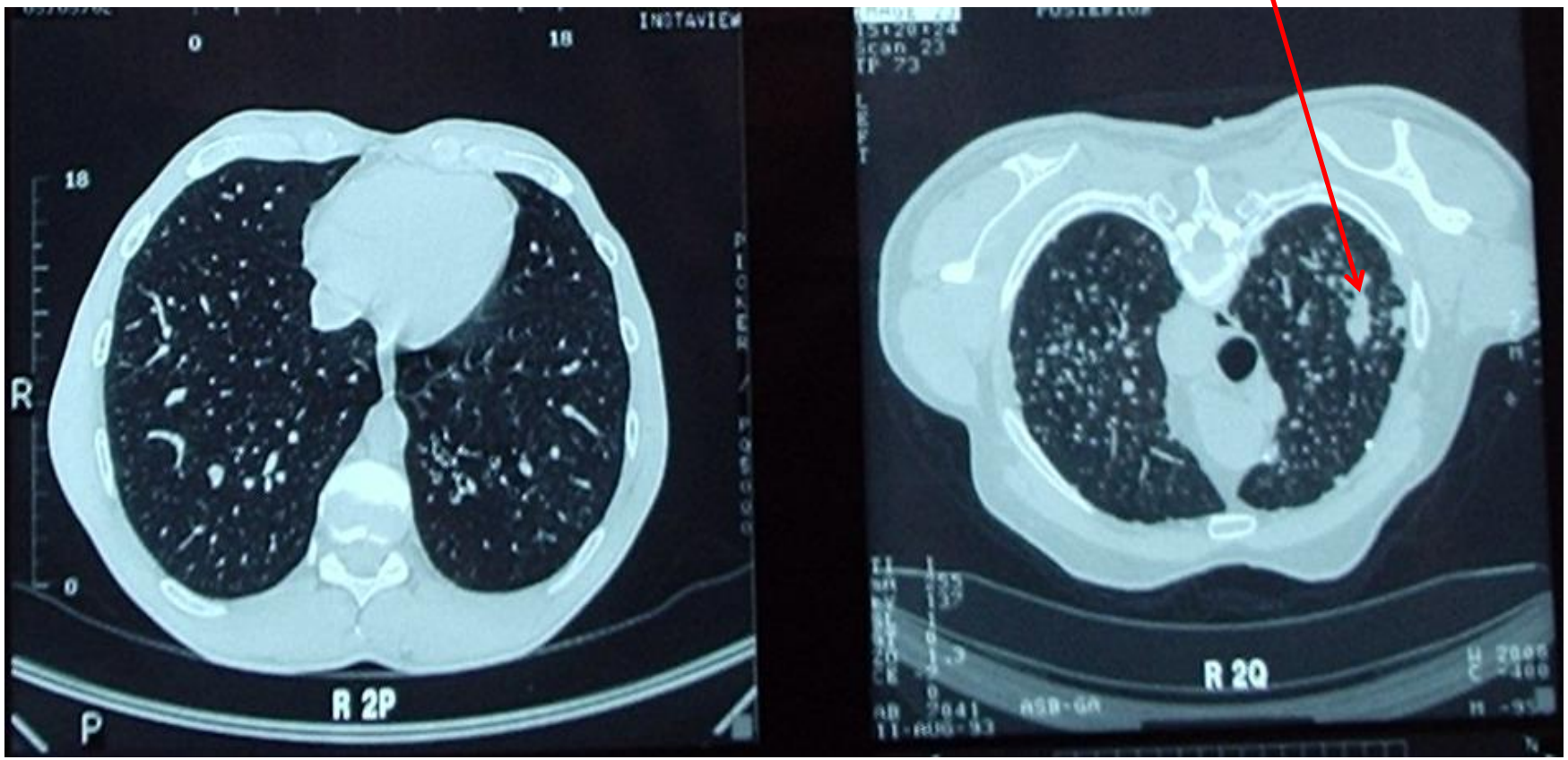


r3



Pneumoconiosis p2, A q2

HRCT according to ILO



p2

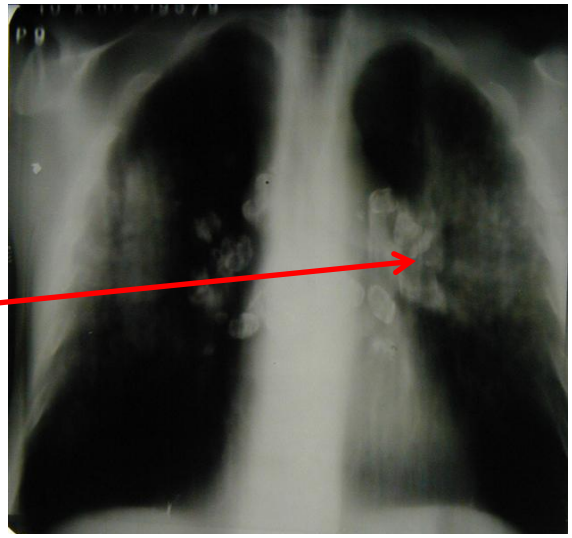
A q2

2. Complicated silicosis

= **nodules of diameter more than 1 cm**

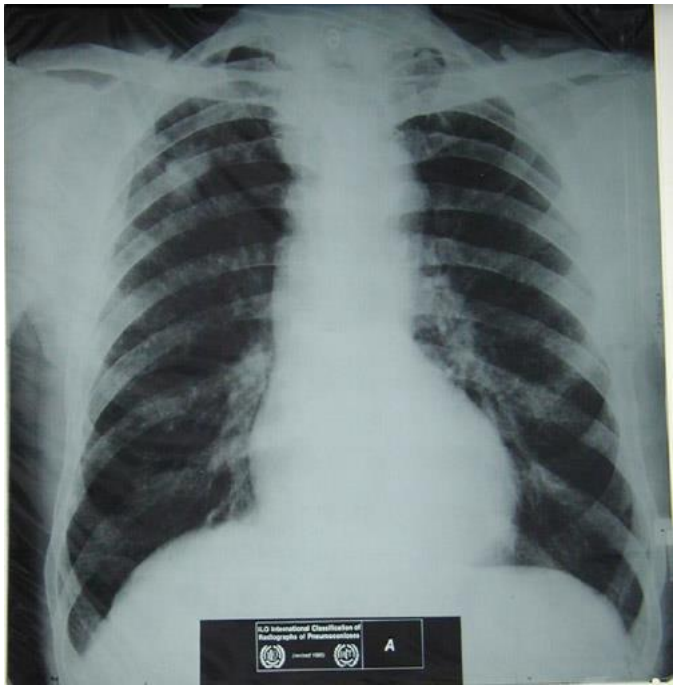
- **progressive massive fibrosis (ILO classif. A, B, C)**
- coalescence of nodules
- compression of lung structures
- cavitations
- compensatory emphysema
- enlarged hilar lymph nodes
- **eggshell calcifications**
- (calcium precipitation at the surface of hilar nodes)

Egg shells

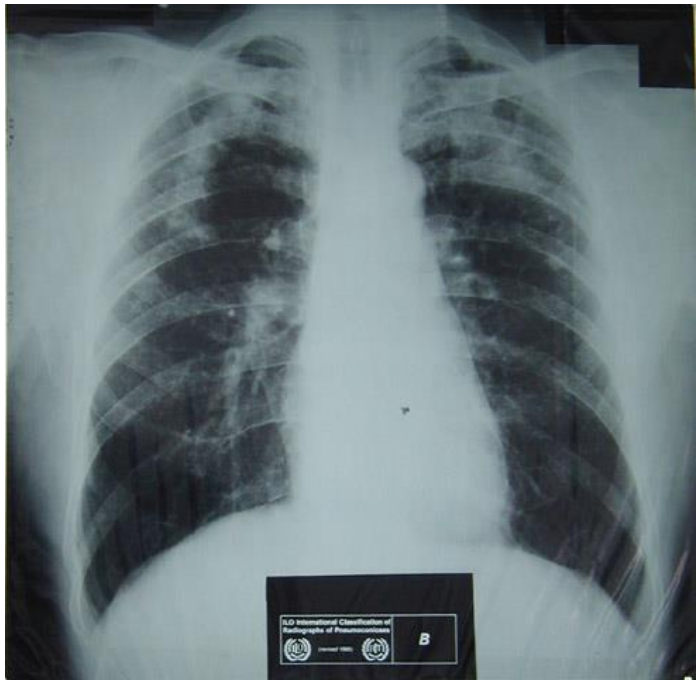


Complicated pneumoconiosis –A, B

- A – diameter
- up to 5 cm

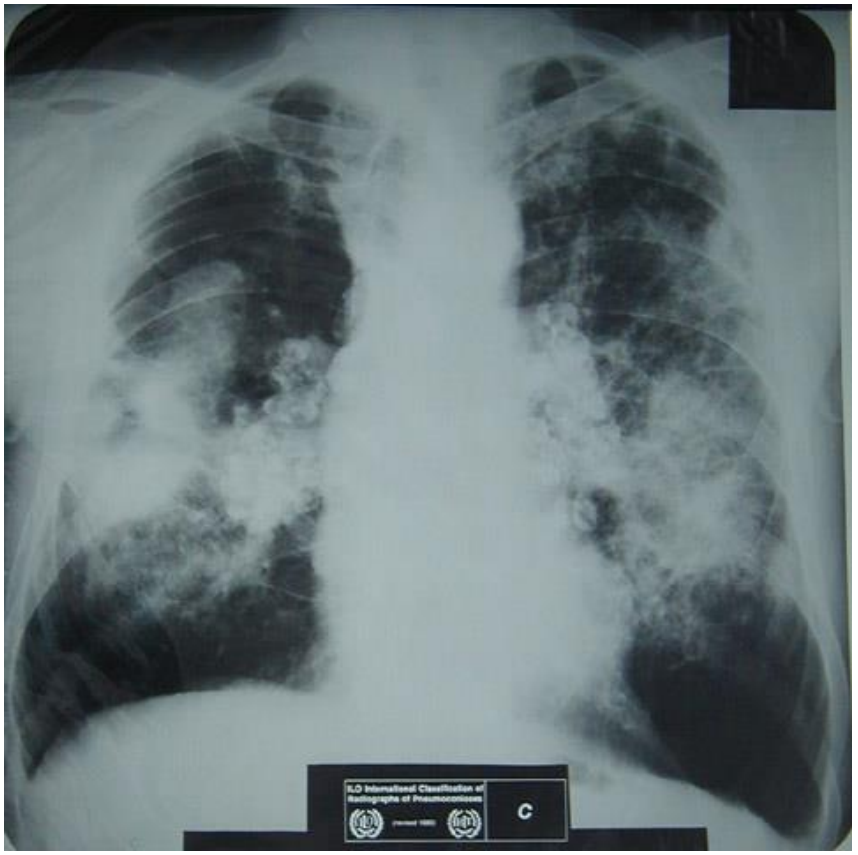


- B – 5 cm up to equivalent of right upper lung field



Complicated pneumoconiosis - C

- C – more than equivalent of right upper lung field



Clinical findings

- Dyspnoea on exertion, later dyspnoea at rest, cough
- Auscultation: SILENT - no typical sounds
- DIAGNOSIS:
- CHEST RADIOGRAM, EXPOSURE confirmed
- SPIROMETRY - lung functions - combination of obstruction + restriction
- COMPLICATIONS: chronic bronchitis, emphysema, cor pulmonale (right heart failure), tbc, lung cancer (SiO_2 human carcinogen IARC class 1)
- Dif.dg.: sarcoidosis, tbc

Silicosis

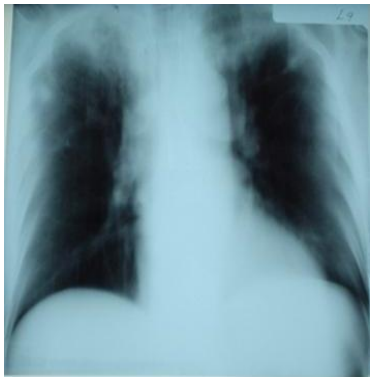
concentrator of oxygen

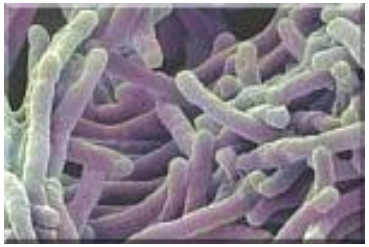
- Treatment**:** no specific treatment available,
- symptomatic, oxygen as a prophylaxix of cor pulmonale due to hypoxia (home concentrator of oxygen), anti tbc; lung transplant
- Prognosis: X-ray stable or PROGRESSIVE
- (never reduction of the changes) depending on quantity of silica exposure, and individual factors
- Prevention:

proper ventilation, wetting of the rock during mining, replacement of sand with less noxious substances where possible

3. Silicotuberculosis

- **infection with Mycobacterium tuberculosis (Mtb)**
- **common in patients with silicosis**
- **fever, night sweating, hemoptysis**
- **fatigue**
- **but frequently asymptomatic**
- **screening by Mtb in sputum**





Bacillus Koch BK

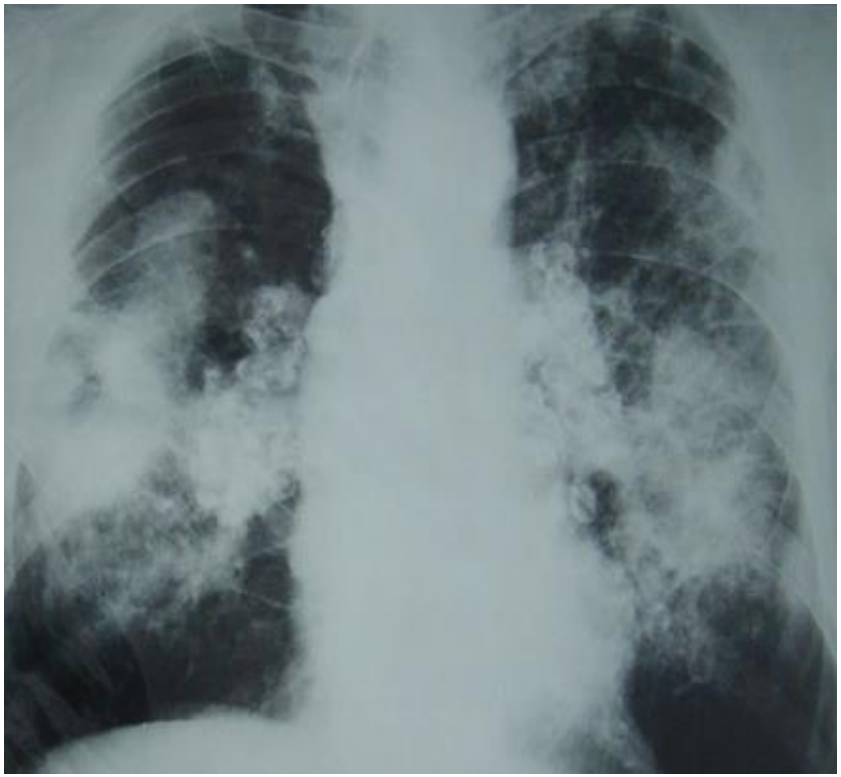
4. Silicosis with lung cancer

- with typical signs

of silicosis

according to ILO

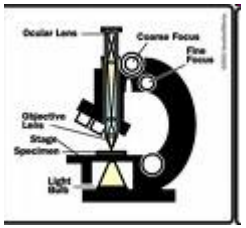
- (including smokers)



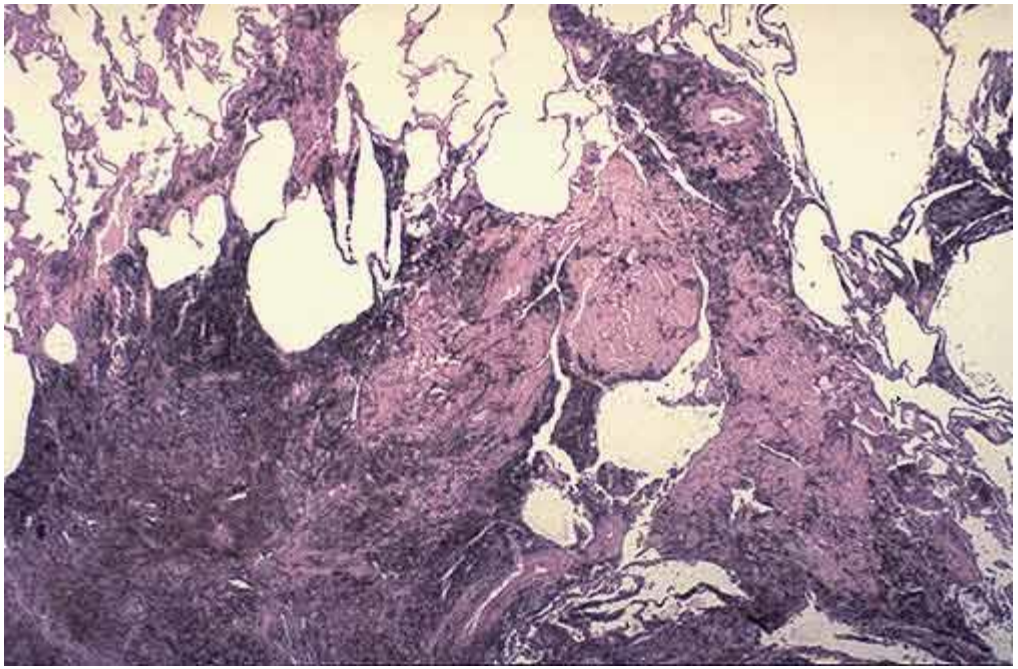
COAL WORKER´S PNEUMOCONIOSIS

- **Etiology**: parenchymal lung disease caused by coal dust
- Coal dust is less fibrogenic than silica,
- The exposure is usually mixed with silica.
- Black coal only
- Deep mines only
- **Caplan´s syndrome**
- Pneumoconiosis + rheumatoid arthritis

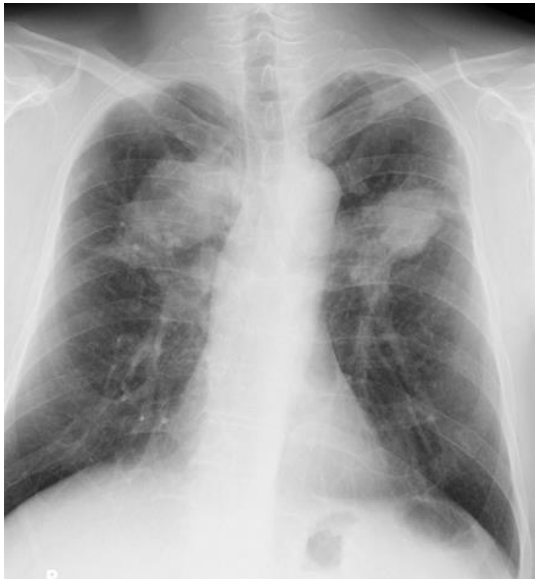
Coal nodule



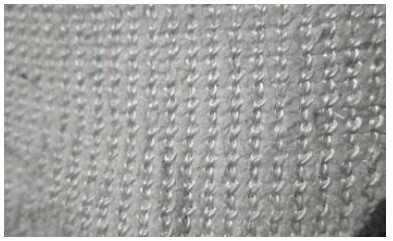
- accumulation of coal dust in the lungs. Macrophages are filled with coal dust, coal nodules contain collagen



COAL WORKER´S PNEUMOCONIOSIS

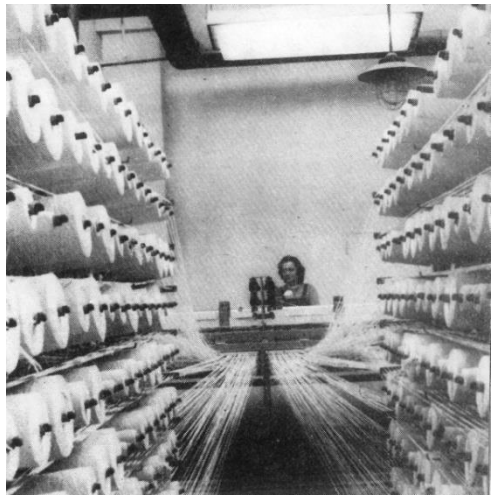


- Clinical findings very similar to silicosis
- Difference in history, hygienic evaluation of dust exposure
- Course, therapy, prognosis do not differ
- from silicosis



ASBESTOS

- Minerals - silicates of fibrous nature and potential to be woven, durable and resistant to heat and chemicals
- Exposure: mining, manufacture of insulation, friction materials for brake linings, asbestos+ cement (=eternit) or textile manufacture
- Products: Pipe warming, fire-protection and insulating material, fire-protection gloves, fire protection uniforms
- In the EU no more allowed (only demolition works)



Chrysotile and crocidolite

CHRYSOTILE – white asbestos - Serpentine group, long, thin fibers. More easily woven into cloth than other types of asbestos. Breaks down and disappears from the tissue after years

CROCIDOLITE – blue asbestos – amphibole group, probably more dangerous – persists for decades in the tissues

Dangerous is size of fibers less than 3 μm x longer than 5 μm (thin and long), *but frequently also short fibres are found in the authopsy of patients.*

EXPOSURE TO ASBESTOS
Eternite production

5 -7 % of asbestos in cement

- Wet production

Dry tubes sized
with grinders – dusty work

ASBESTOS INDUCED DISEASES

1. Asbestosis - parenchymal lung disease

occurs **only after large amount of fibres inhaled**, this disease will disappear in few decades.

2. Asbestos-induced pleural disorders

3. Asbestos-induced mesothelioma

4. Asbestos-induced lung cancer

5. Asbestos-induced larynx cancer

6. Asbestos-induced ovarian cancer

even small exposure represents a risk, an increase is expected due to the long latency of the diseases

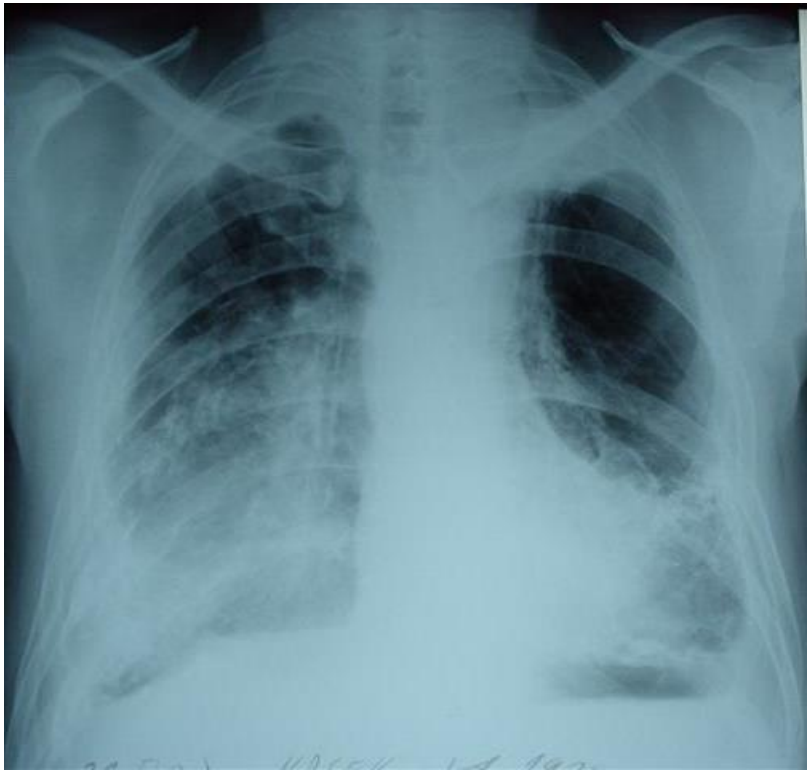
1. ASBESTOSIS

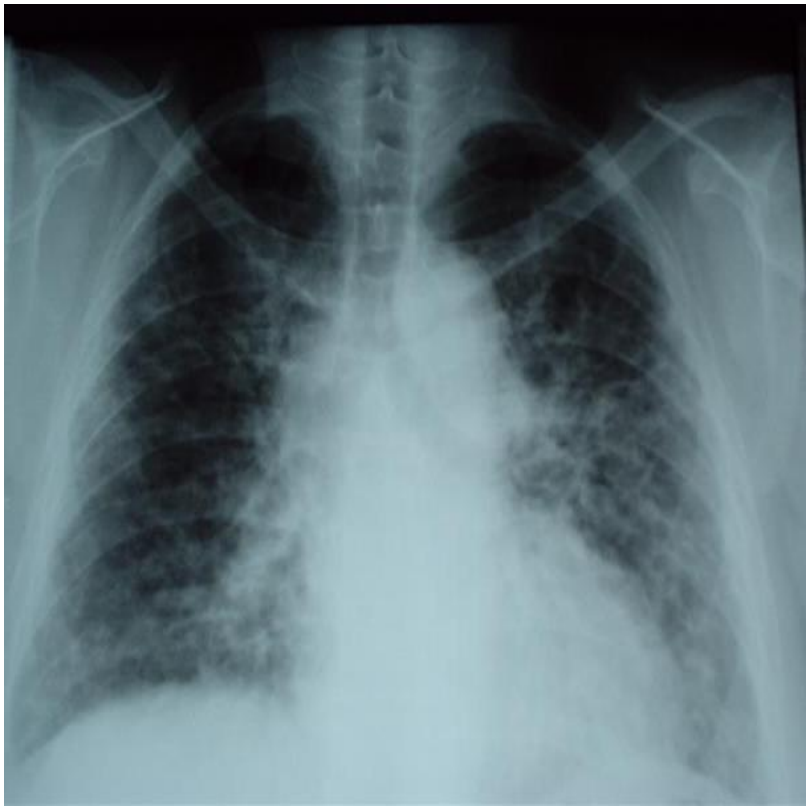
» macrophage + asbestos fibre

- Etiology: lung inflammation and fibrosis due to activation of alveolar macrophages.
- Because asbestos fibres cannot be completely engulfed by macrophages, they persist in the lungs for years.
- Oxidative stress, cytokines, inflammation, **growth factors** (TNF, IL-1, PDGF, IFG) **–proliferation of pneumocytes and fibroblasts – overproduction of collagen**
- Pathology: **diffuse interstitial lung fibrosis**

  in **lung bases** and in subpleural locations

Asbestosis





Clinical findings

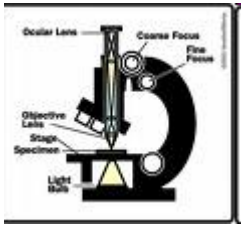
- dyspnoea on exertion
- non-productive cough,
- bibasilar crepitation („velcro rales“)

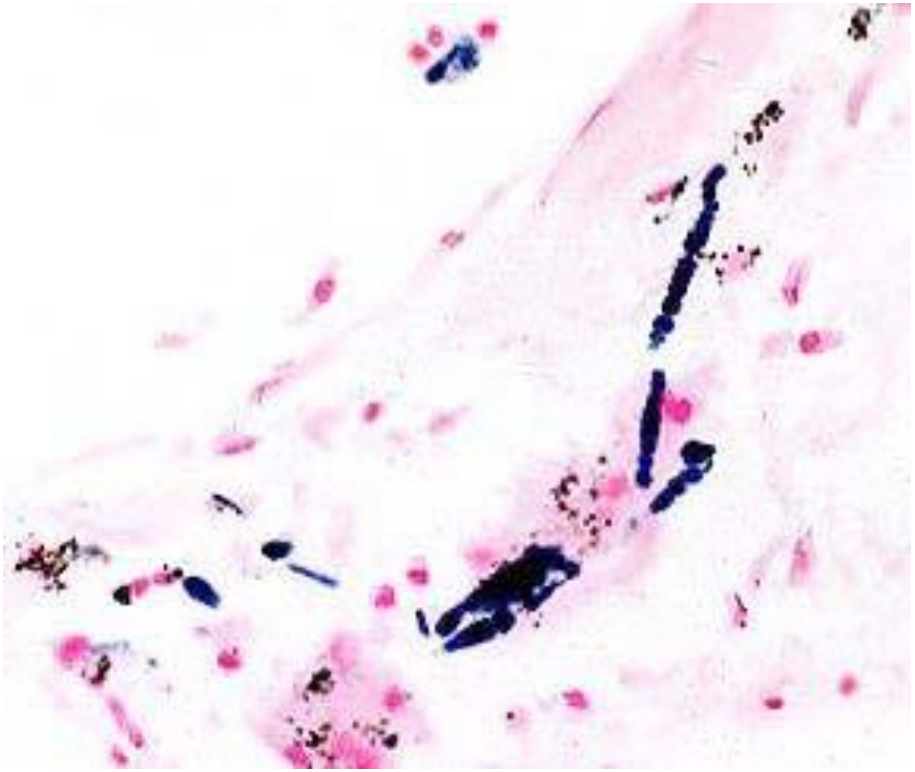
Diagnosis of asbestosis

- **Chest radiograph** (HRCT scans more sensitive) - symmetric irregular opacities in the lower parts of the lungs (ILO classif. s,t,u), relative sparing of the upper lobes, honeycomb lung,
- frequent pleural opacities
- **Spirometry :** symmetric reduction in lung volumes (FVC, TLC) – restrictive damage,
- reduction of the diffusing capacity for CO (DLCO)- transfer factor due to the thickening of the interstitium.

Asbestos bodies

(„beds on a file“)



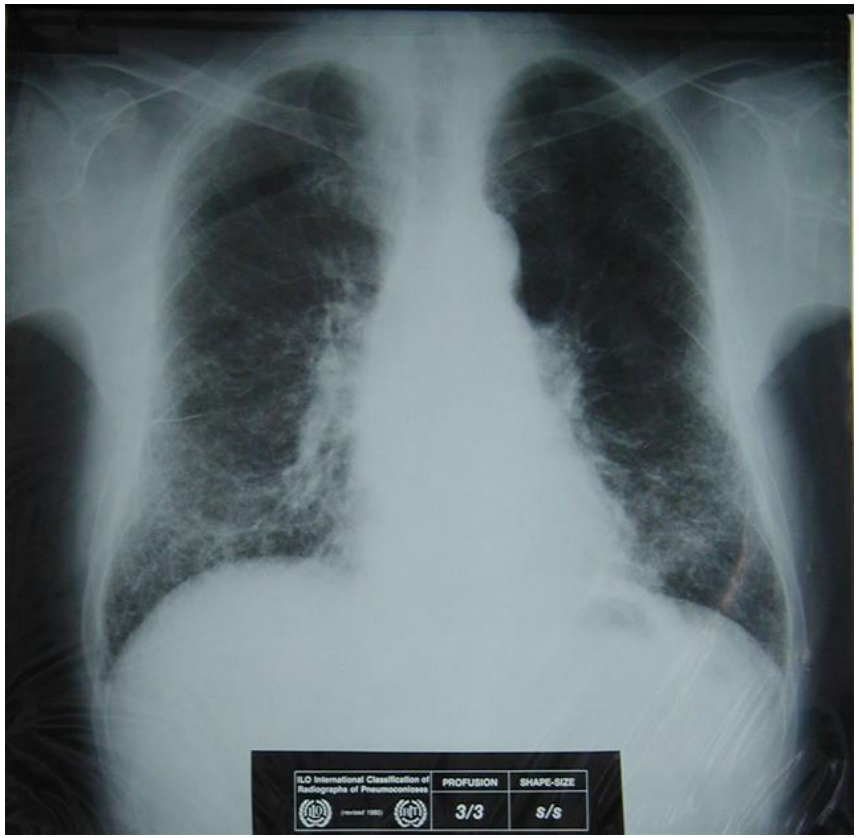


Asbestos fibres coated with hemosiderin

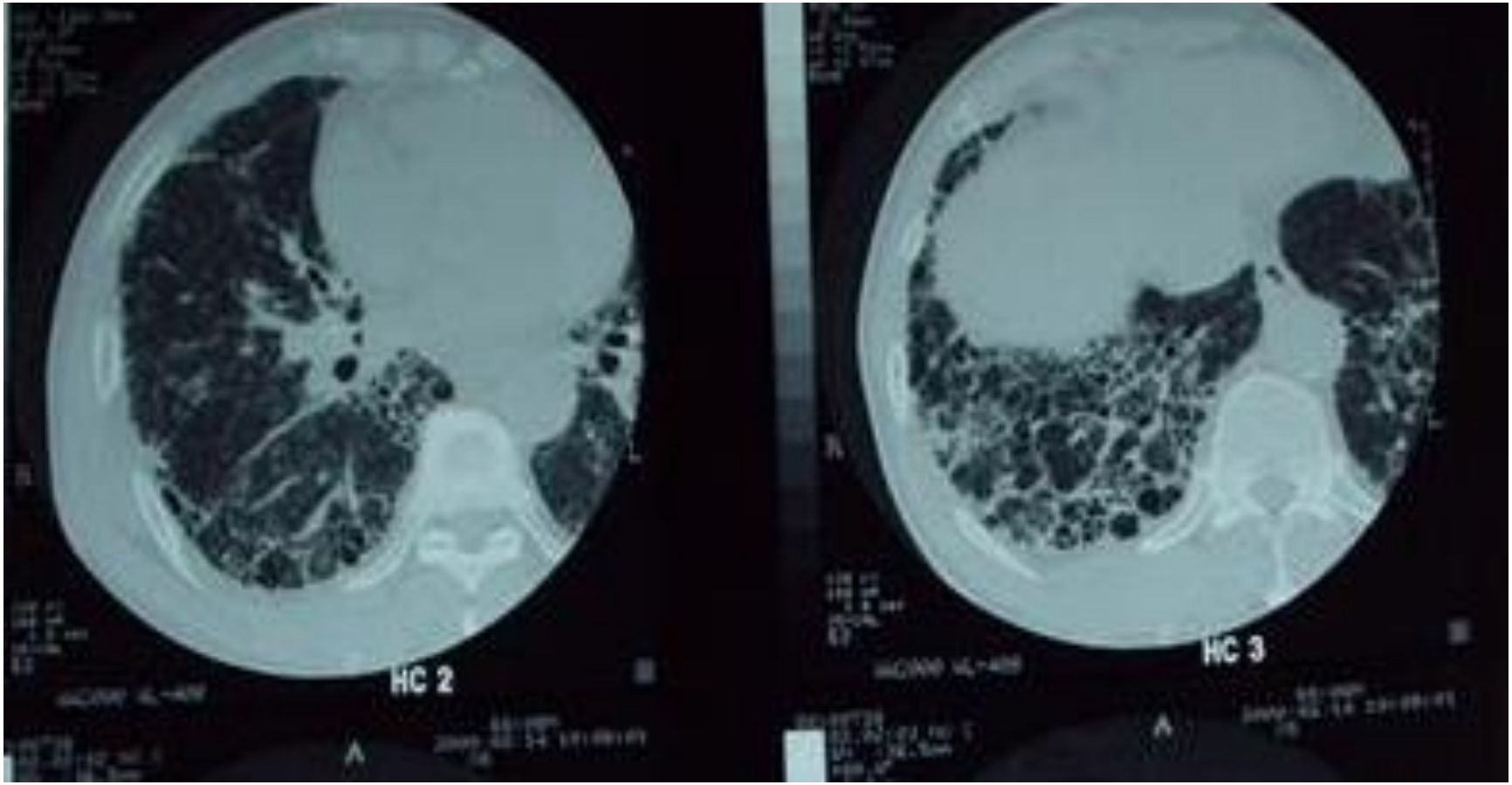
ILO classification

- irregular opacities
- width:

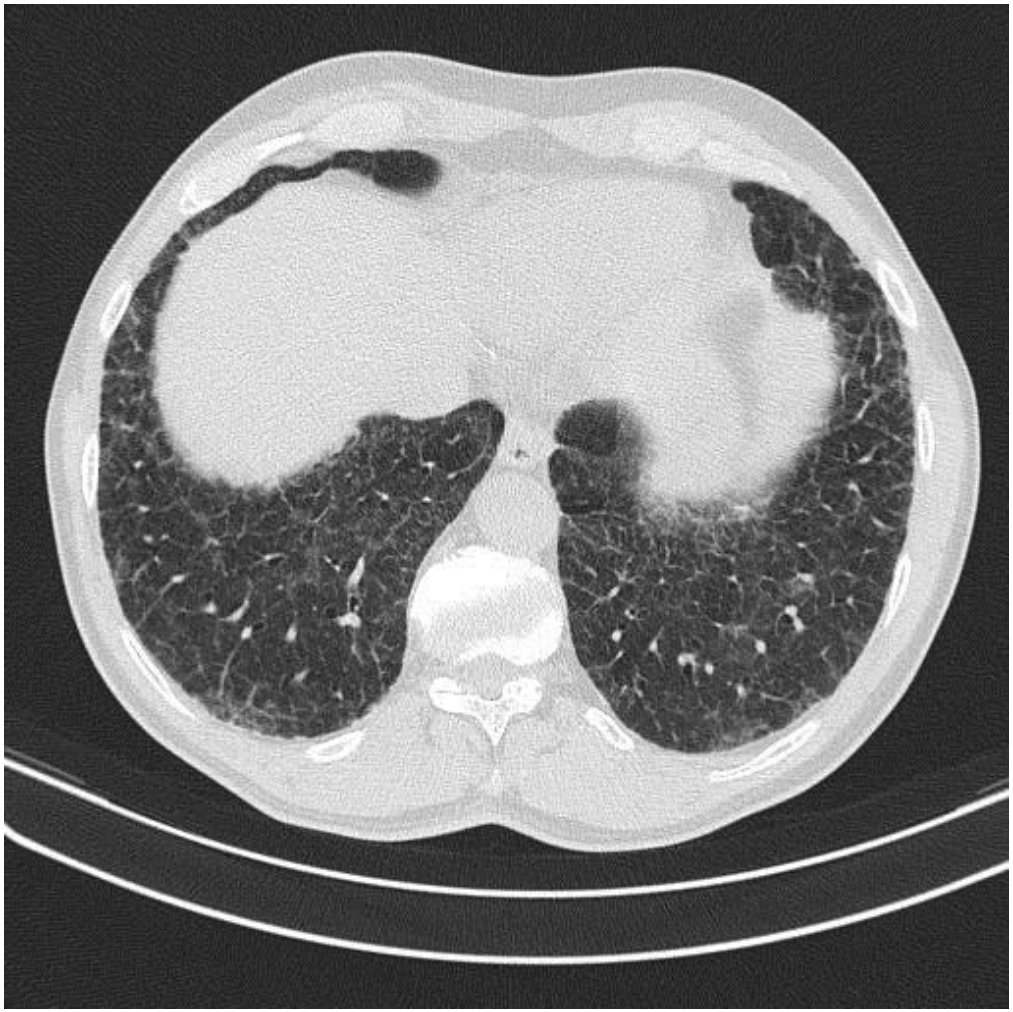
- s1 – s3 up to 1,5 mm
- t1 – t3   up to 3 mm
- u1 – u3 more mm



Asbestosis - ILO HRCT



Asbestosis

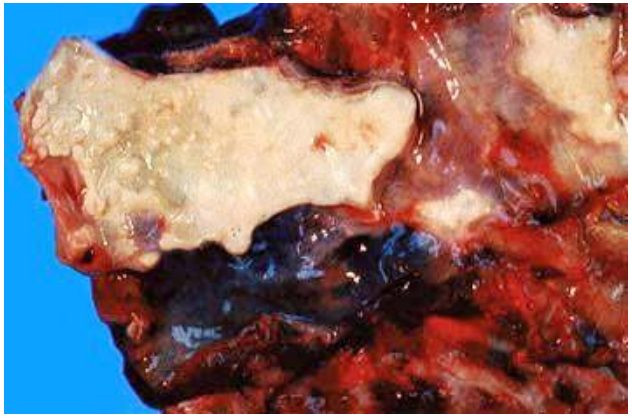


concentrator of oxygen

Management

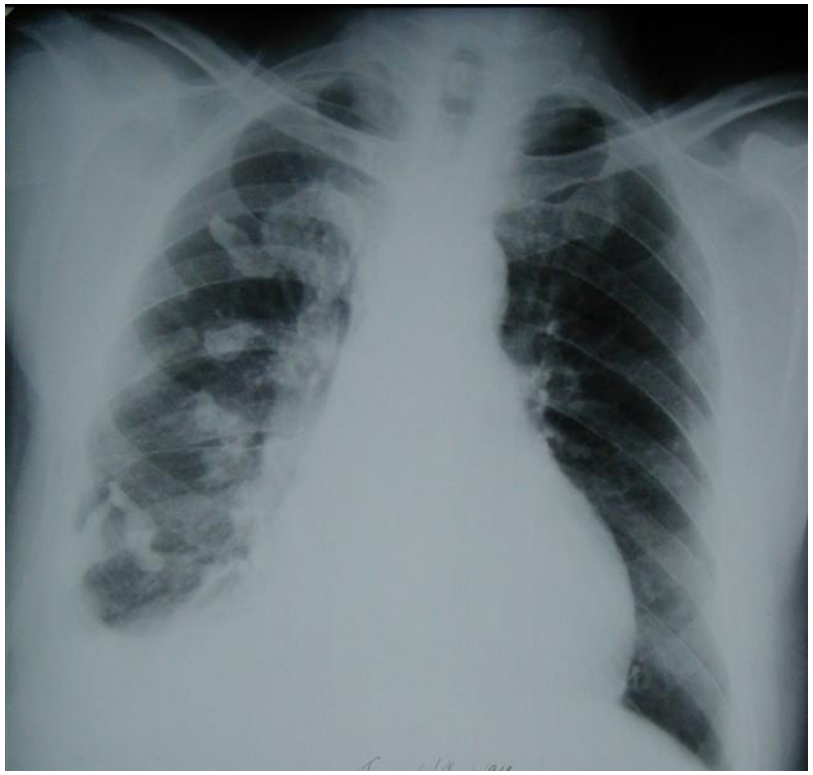
- Complications: respiratory failure, cor pulmonale
- Prognosis: depending on the exposure – the more fibres inhaled – the worse.
- Progression years after the end of exposure is frequent, regression never occurs
- Treatment**:** symptomatic, oxygen as a prophylaxis of cor pulmonale due to hypoxia (home concentrator of oxygen)
- Follow-up examinations needed, gradual compensation

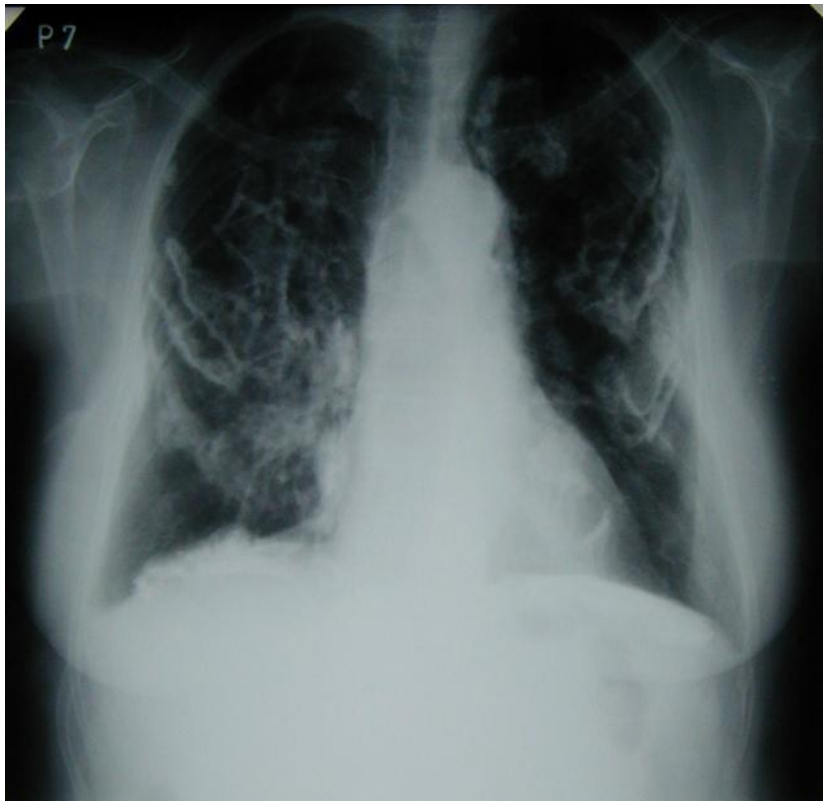
2. ASBESTOS - INDUCED PLEURAL DISORDERS

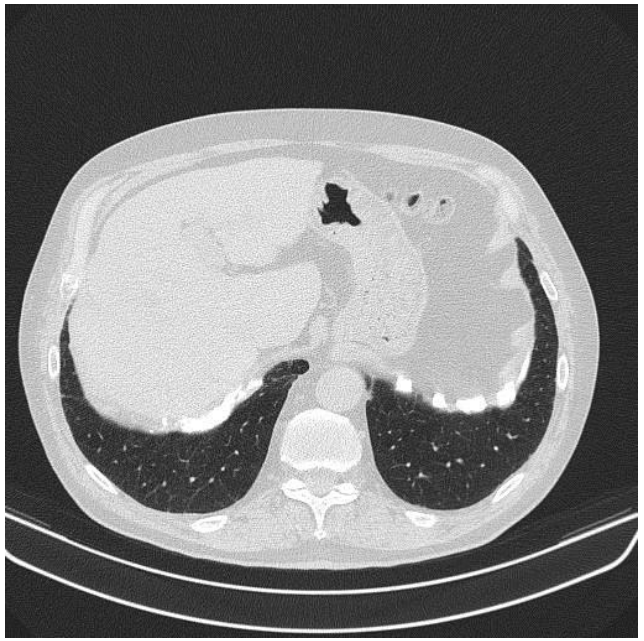


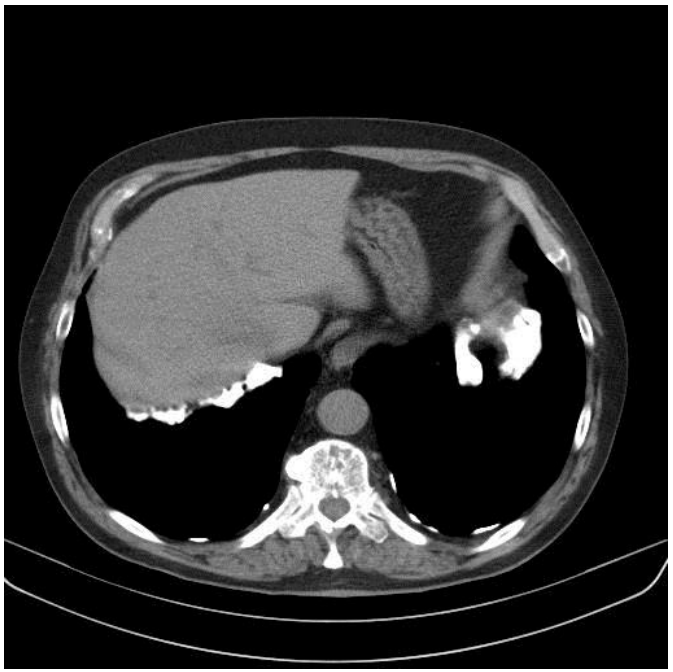
- Pleural thickening, focal pleural plaques, sometimes pleural and diaphragmatic calcifications
- Significance - marker of exposure of asbestos, precancerosis (dif. dg. mesothelioma !)
- Mostly asymptomatic, rarely dyspnoea – impaired movement of the lung, fixation to the chest wall: decrease in TLC and FVC

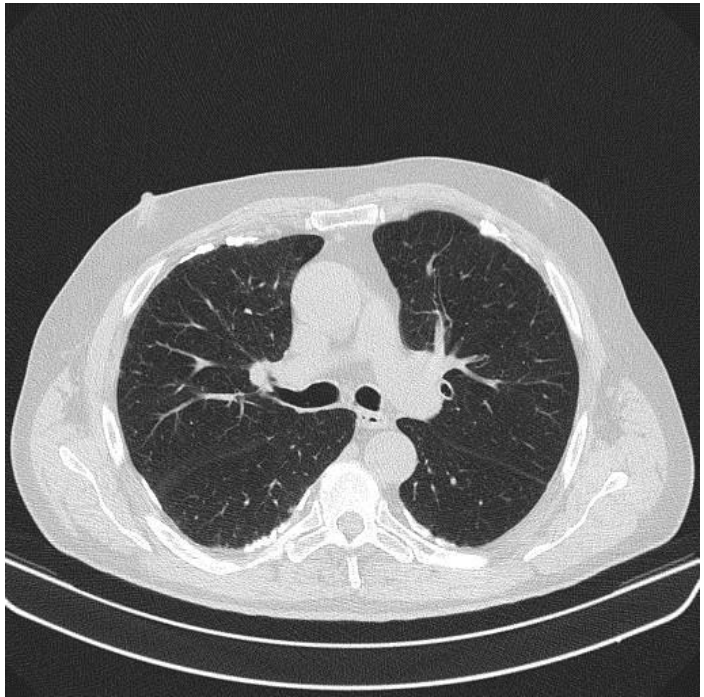
Pleural plaques and pleural thickenings





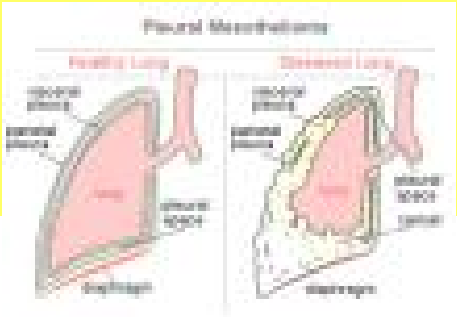




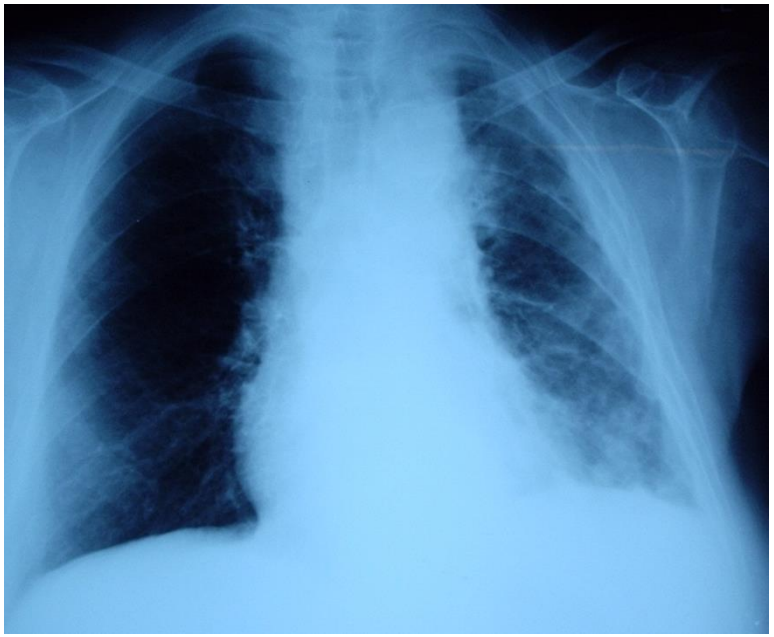


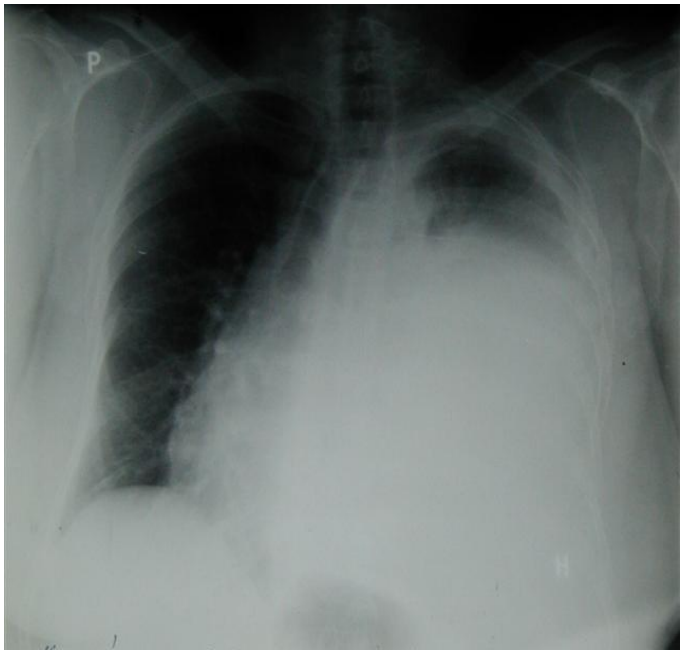
Calcificated
pleural plaques (smaller)
pleural thickenings (several cm)

3. MESOTHELIOMA



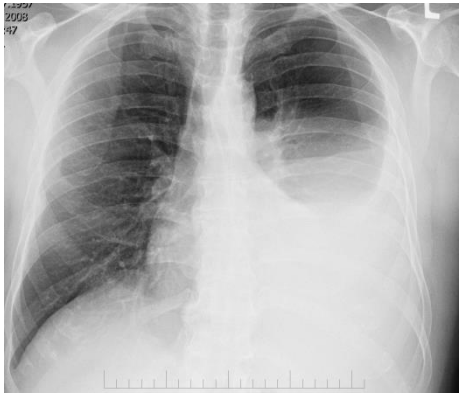
- **Malignant tumour of pleura** - diffuse pleural thickening, progressively encases the lung. May extend into the chest wall or the other lung.

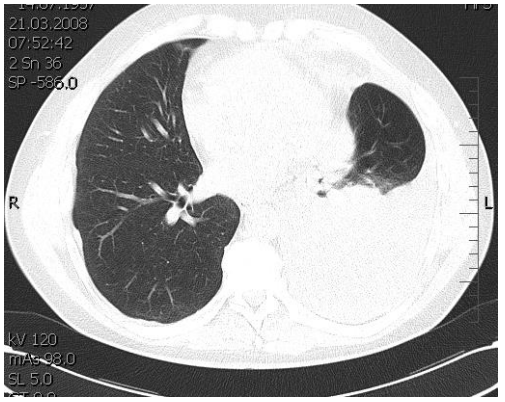




MESOTHELIOMA

- **Malignant tumour of pleura** - diffuse pleural thickening, progressively encases the lung. May extend into the chest wall or the other lung.
- Theory: „one fibre can cause mesotheolioma“ -
- 75 % cases of mesothelioma have a history of significant exposure to asbestos
- 95 % cases some exposure to asbestos





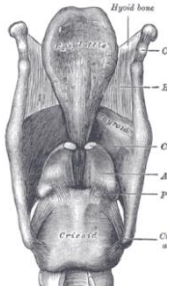
MESOTHELIOMA

- Latency period: 30 – 50 - more years
- Prognosis: 6-12 months

- Therapy: cytostatics
- Surgery - decortication of pleura

- Marker glycoprotein mesothelin in blood or exsudate (not completely specific and predictive)

4. LUNG CANCER
5. LARYNGEAL CANCER
6. OVARIAN CANCER



For lung cancer the effect of asbestos and cigarette smoke is **synergistic**

non-smoker exposed to asbestos **5-fold** increase in risk

smoker has 20-fold increase of probability

smoker exposed to asbestos **50-90-fold increase**

(all histological types)

Latency period : 15 - 50 years

To acknowledge lung and larynx cancer due to asbestos:

signs of asbestosis or hyalinosis are needed

compensation is therefore possible also in smokers

CHRONIC OBSTRUCTIVE BRONCHOPULMONARY DISEASE in black coal miners

In Czech Republic:

- COPD higher stage
- High dust cumulative dose
- Until 2 years after the end of exposure