



Occupational pulmonary diseases

Pneumoconioses



Pneumoconioses

- The effective exposure time is 2 – 35 years
 - (Czech and Slovak republic 8 – 20 years)

Non-collagenous

- Siderosis (dust of iron, Fe)
- Stanosis (dust of tin, Sn)
- Anthracosis (dust of anthracite coal – C content is between 92–98%)

Collagenous

- Silicosis (dust of silica – SiO_2)
- Coal worker's pneumoconiosis
- Asbestosis (dust of asbestos)
- Talcosis (dust of talc)
- Berylliosis (dust of beryllium and compounds)

Welder's lungs – inhalation of fumes during welding

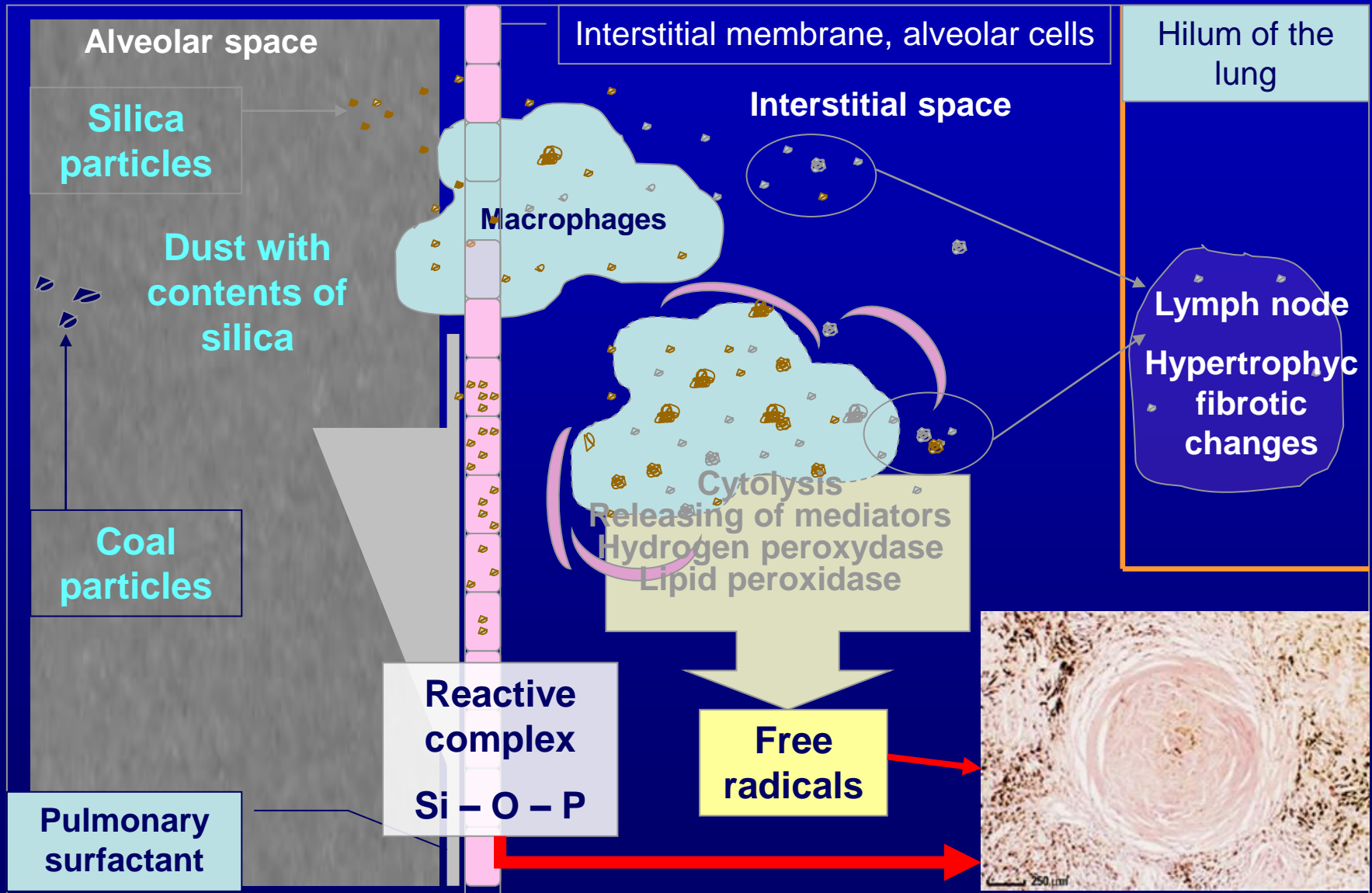
- Siderosis + other cations from welding fumes

Pneumoconioses

Examples of risk professions:

- Mineworkers – mineral mines, black coal mines
- Graphite mineworkers,
- Coal trimmers
- Tunnelers
- Foundry workers
- Glassblowers, ceramics workers
- Glasscutters, stonecutters
- Sculptors
- Construction workers – reparation of buildings
- Production of emery papers

Silicosis – ethiology and pathogenesis



Silicosis etiology and pathogenesis

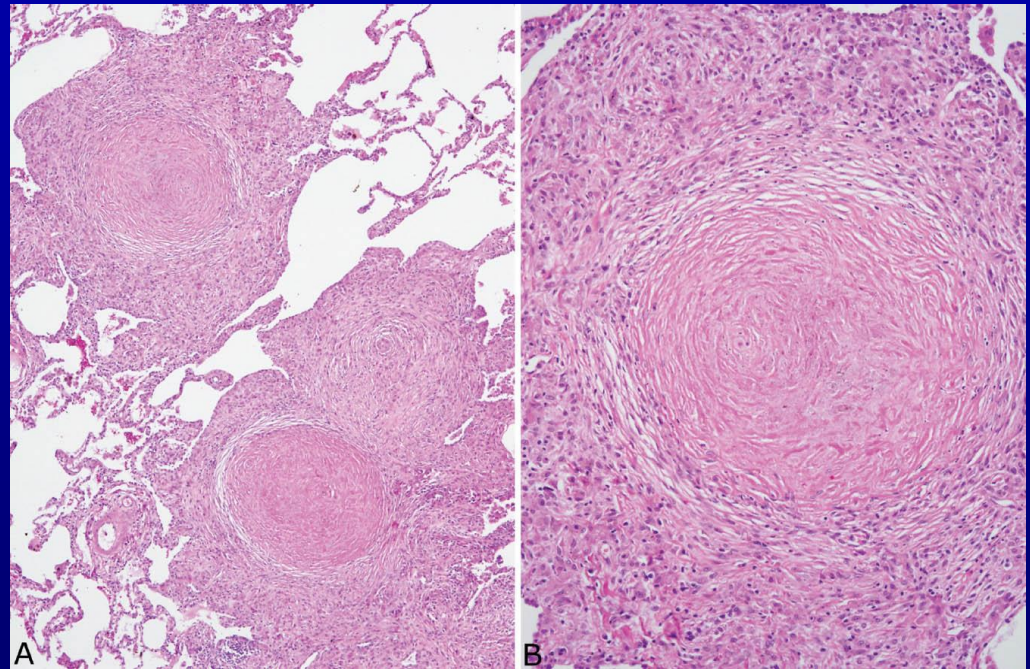
Parenchymal lung disease caused by inhalation of crystalline silica (SiO_2)

On presence of SiO_2 the pulmonary tissue react by
aseptic interstitial reaction with production of silicotic nodules

Acellular core – composed of
swirls of collagen

Cellular capsule – containing:

- Macrophages
- Fibroblasts
- Plasma cells



© <https://thoracickey.com/wp-content/uploads/2016/10/f0205-01.jpg>

Silicosis- forms and clinical findings

1. **Acute silicosis** – after massive exposure to high concentration free silica dust – rare occurrence
2. **Subacute silicosis** – after exposure 2 – 5 years to high concentration of silica
3. **Chronic silicosis** – usually after exposure 10 and more years, concentration of silica less than 30% – most often occurrence
 - Most patients don't have symptoms long time
 - Symptoms
 - Frequently **dry cough** (moist cough due to chronic bronchitis – is present at more than 50% patients and smokers)
 - **Dyspnea** – first light, severe if there is progressive massive fibrosis
 - **Chest pain** – sometime sharp – presence suggest complications

Silicosis – diagnostic

Physical examinations depends on development

- initially normal, later dyspnea, later signs cor pulmonale, cyanosis, partial later global respiratory insufficiency, wheezing, crackles ...

Spirometric examination – evaluation of ventilatory disturbances

Examination of blood gases – evaluation respiratory insufficiency

ECG – development of the cor pulmonale

Blood count – secondary polycythemia

FW – can be higher

Immunological examinations – higher value of IgG

Tuberculin test – once a year (recommendation of US authors)

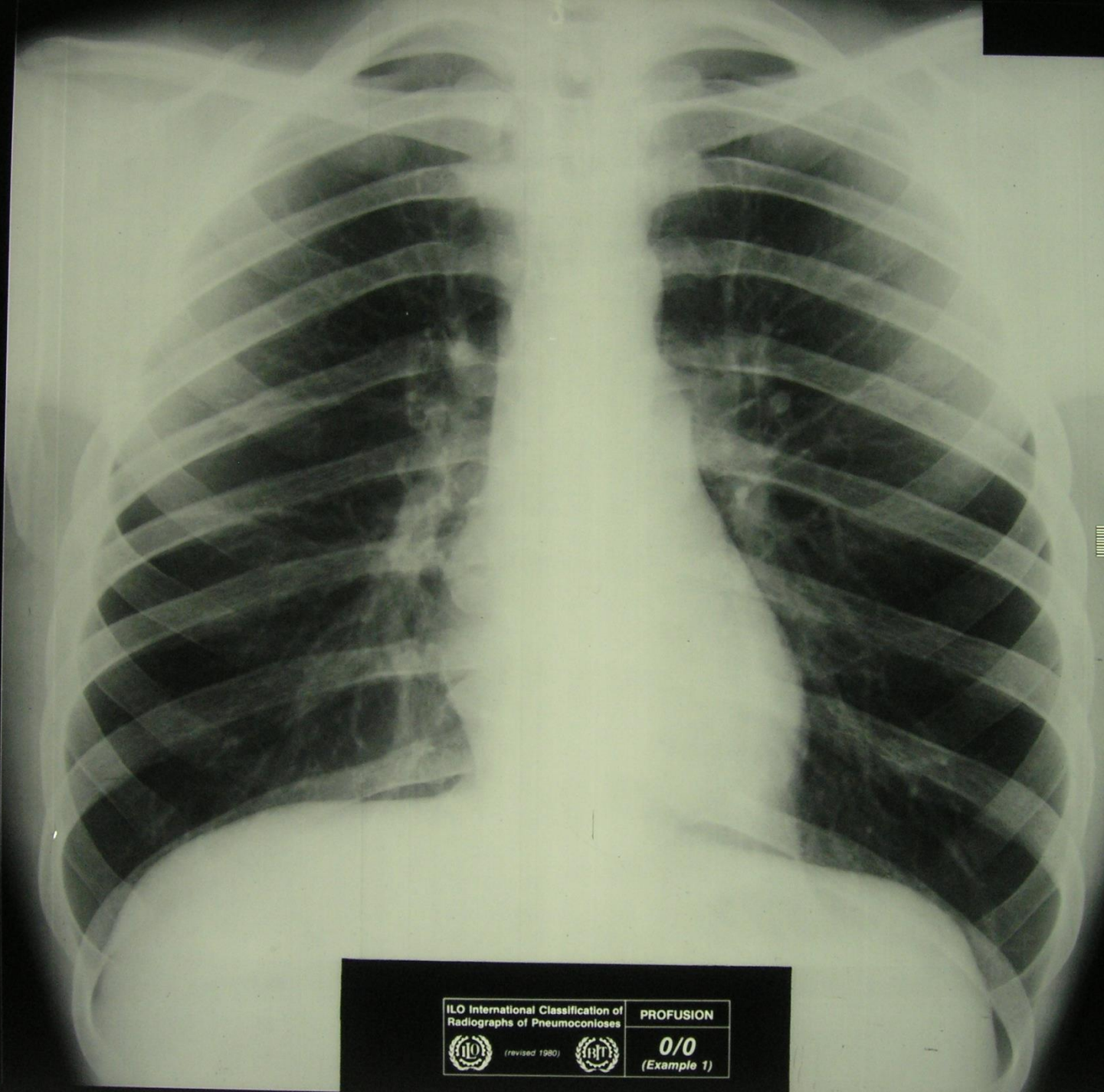
Bacterial examination and cultivation of sputum – when is suspicion on TBC

Silicosis – diagnostic

X ray findings – PA x-ray picture 35x35 cm

- Chronic simple silicosis - small round opacities in both lungs especially in upper lobes < 10 mm diameter
- Calcifications of silicotic nodes and hilar lymph nodes can occur = eggshell calcification
- Chronic complicated silicosis - conglomerate of shadows in perihilar regions (big – nodes more than 10 mm)
- Progressive massive fibrosis – is the end of complicated silicosis – presence of the big fibrotic nodules with the obliteration of bronchioles and blood vessels
- Perifocal end compensatory emphysema

HRCT, MR lungs



ILO International Classification of
Radiographs of Pneumoconioses

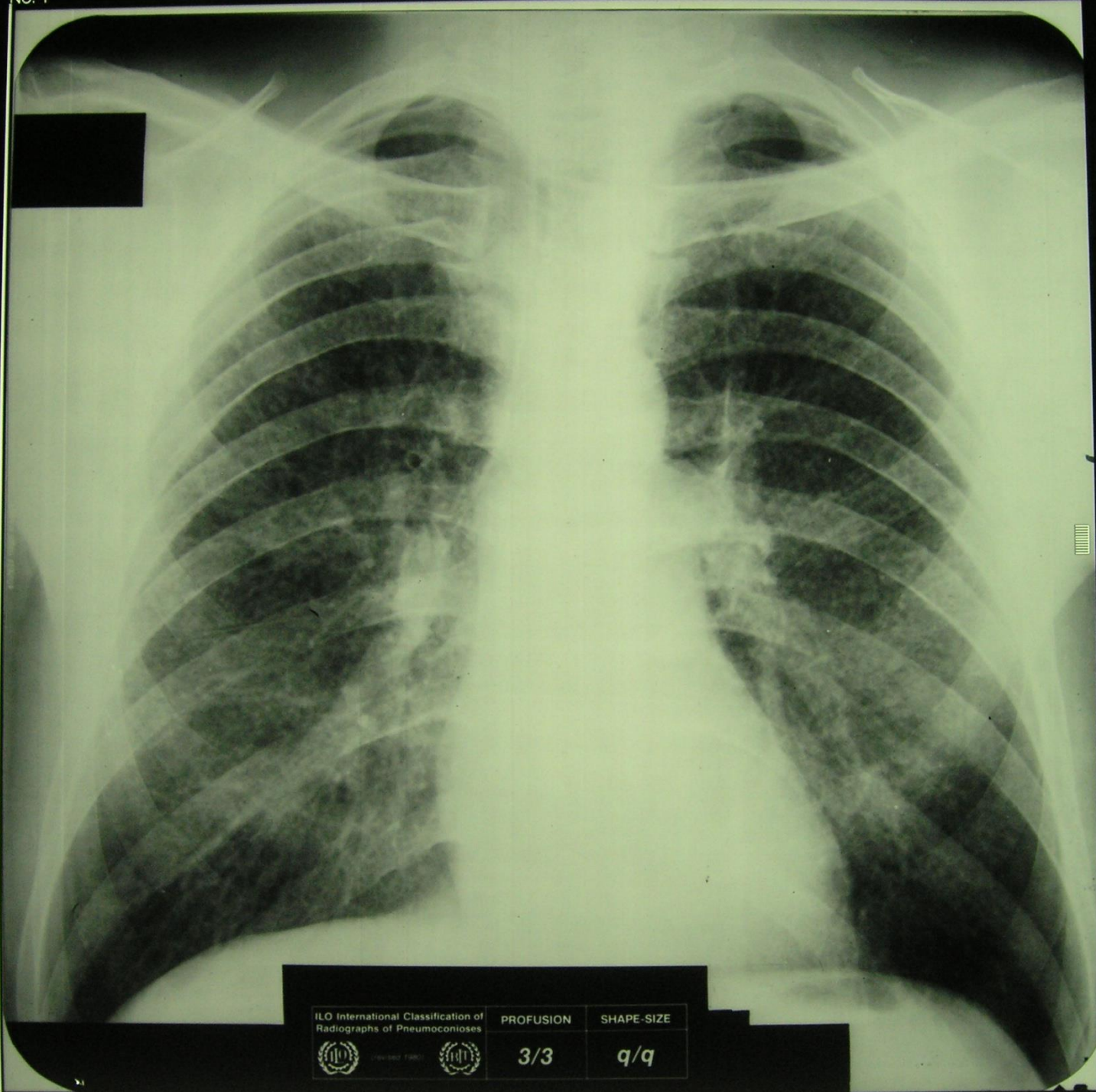


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(Example 1)



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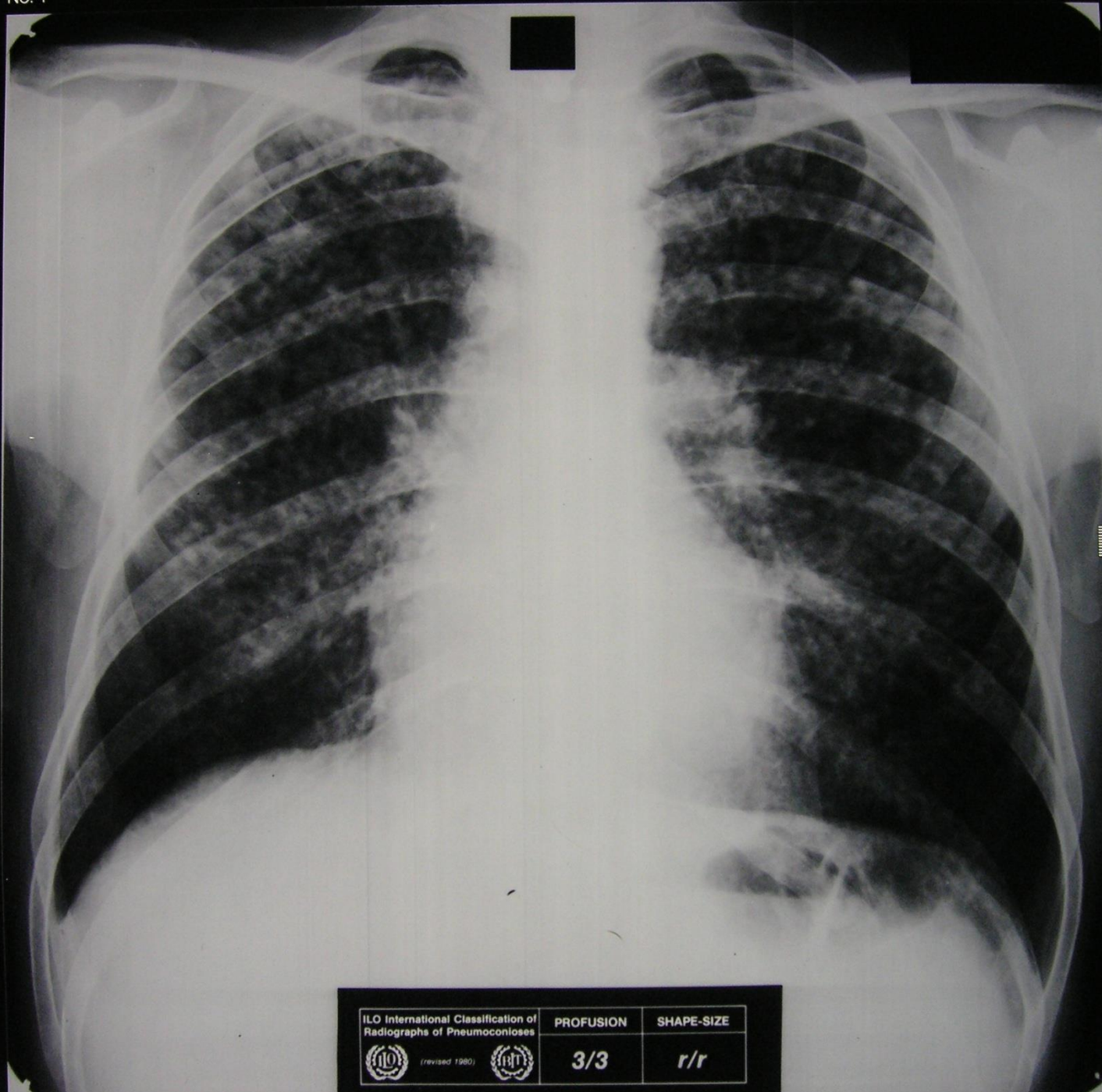


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Silicosis

Complications

- Lung tuberculosis (TB) – especially in big - nodes forms
- Spontaneous pneumothorax
- Caplan's syndrome – Silicosis + Rheumatoid arthritis
 - Fibrotic changes are developing faster + often TBC
- Destruction of blood vessels – cor pulmonale
- Respiratory failure

Treatment

- We don't have effective causal treatment
- Most effective is prevention
- We usually treat symptoms and complications

Coal worker's pneumoconiosis

Definition – parenchymal lung disease caused by inhalation of coal dust

Examples of risk works:

- Coal or graphite miners, workers in manufacture carbon electrodes

Etiology and pathogenesis

Coal dust is less fibrogenic than silica, development of disease depend on:

- Quality and type of coal (contents of silica in coal can different)
- Quantity inhaled coal dust
- Underground exposure (10 – 20 years), sensitivity of workers

Coal macule – small conglomerate macrophages which are filed with dust

- Distribution is within alveolar spaces and surrounding terminal bronchioles with response in nearby parenchyma

Coal nodules – contain irregularly stored collagen – most prominent is in upper lobes

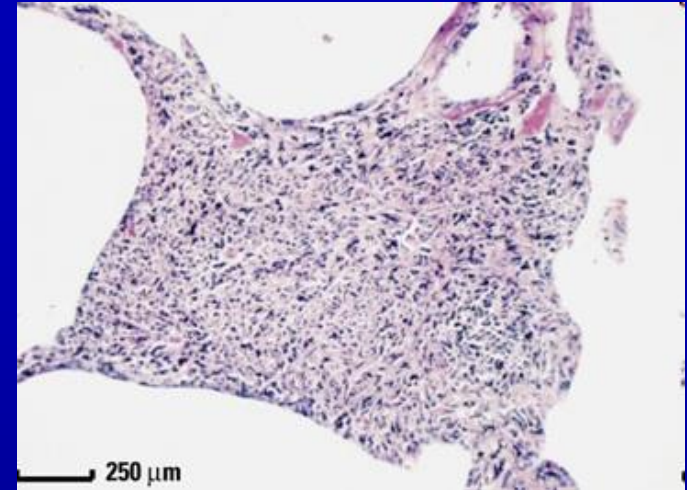
Coal worker's pneumoconiosis

Forms

Simple coal worker's pneumoconiosis

Complicated coal worker's pneumoconiosis

- Because of collagen content is less than by silicoses – in centre of biggest nodes can occur cavities filled by black liquid



©<http://www.ehponline.org/members/2000/suppl-4/675-684castranova/castranova-full.html>

Complications

Caplan's syndrome – occur more often than at silicosis

Biggest nodes has more expressive tendency to necrosis of collagen

Secondary mycobacterial infection is more often (TBC)

Coal worker's pneumoconiosis

Clinical picture:

- Patients often expectorate black sputum
- Simple fibrosis – physical examination is without expressive changes
- Progressive fibrosis
 - Dyspnea at hard work
 - Twinge pain on the chest
 - Cough – chronic bronchitis
 - Physical examination
 - hypersonorous percussion
 - weakened breathing
 - Decreasing of weight – massive progressive fibrosis
 - Respiratory failure

Coal worker's pneumoconiosis – diagnostics

Objectification of work condition – dustiness, content of silica in coal

X ray findings – PA x-ray picture 35x35 cm

- **Simple coal worker's pneumoconiosis** – small irregular opacities

Typical irregular opacities with diameter to 10 mm

- **Complicate coal worker's pneumoconiosis** – big nodes

More than 10 mm diameter

- Other x ray signs
 - Enlargement of lungs hiles
 - Calcification of nodes is rare (les than 5% of patients)
 - Big nodes are more often in upper and middle lugs fields



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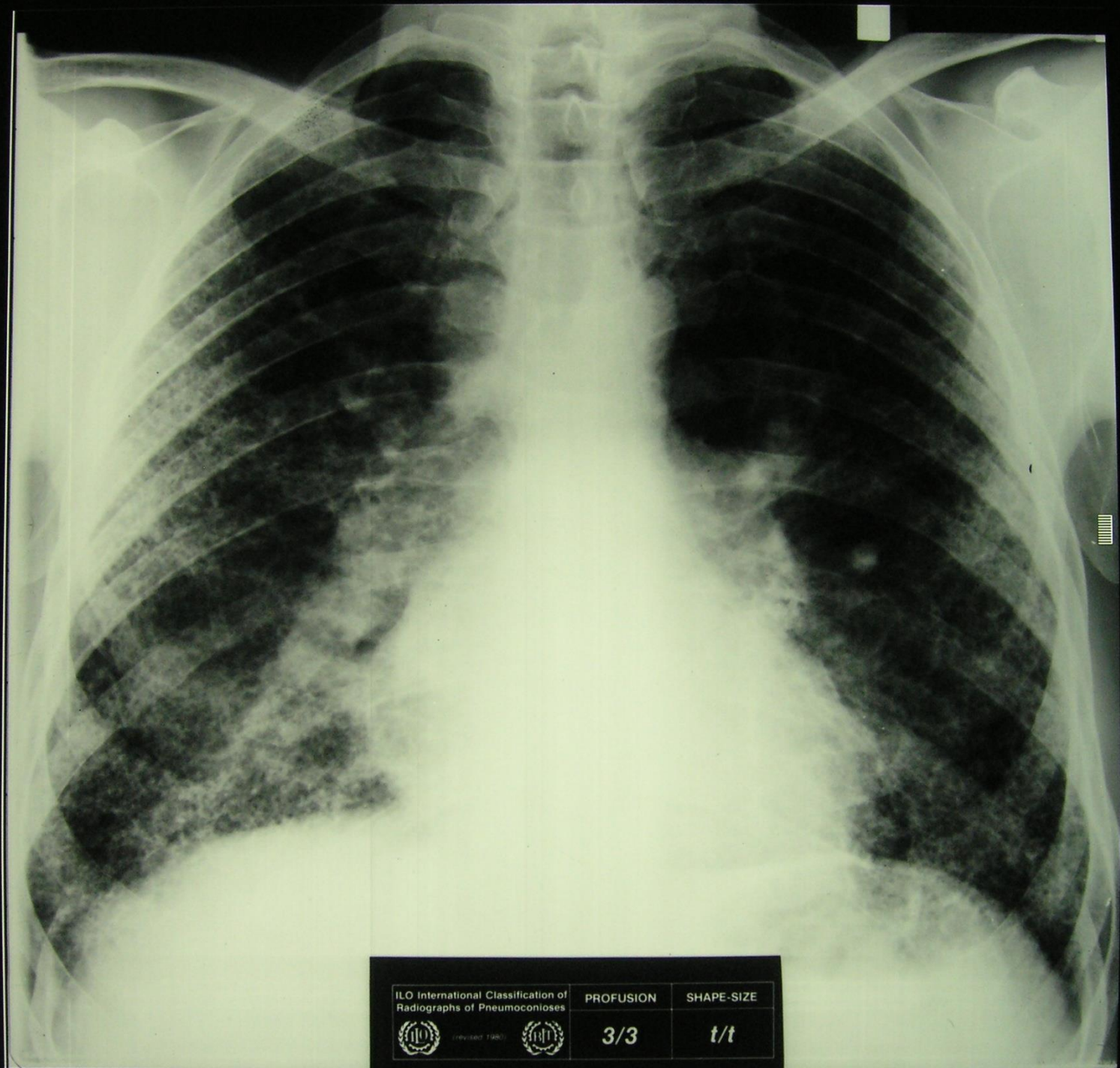


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Coal worker's pneumoconiosis – diagnostics

Diagnostics

- X ray
- Spirometric examination – evaluation of ventilatory disturbances
- Examination of blood gases – evaluation respiratory insufficiency
- ECG – development of the cor pulmonale
- Bacterial examination and cultivation of sputum – suspicion on TBC
- Immunological examinations – higher value of IgG, IgA

Treatment

- Causal treatment doesn't exist
- Most effective is prevention
- We usually treat symptoms and complications

Asbestos

- Exist in two forms of asbestos - **serpentine** and **amphiboles**.
- Properties are - resistance to cold and heat, acids and alkalis, good insulating properties against noise, are used into fabrics.
- The serpentine includes
 - **CHRYSTILE** (white asbestos) - deposits are in Canada, Russia, USA, South Africa.
- The amphibols includes
 - **CROCIDOLITE** (blue asbestos)
 - **AMUZITE** (brown asbestos)
 - **ANTHOPYLITE**, **ACTINOLITE** and **TREMOLITE**
 - The sites are located in South Africa, Zimbabwe, United States.



https://upload.wikimedia.org/wikipedia/commons/thumb/8/8a/Chrysotile_1.jpg/300px-Chrysotile_1.jpg



© <http://www.epa.gov/asbestos/index.html>

Asbestosis

Definition – disease caused by inhalation and retention a group of mineral silicates with content of silica and other minerals (Mg, Fe, Al, Ca, Na)

Asbestos is confirmed human carcinogen

Examples of risk:

Professional risk works

- Asbestos mining, production of asbestos materials, liquidation of asbestos materials and products
- Repairing of wall and ceiling covering by asbestos panels

Non professional risk

- Dust around of asbestos factories

Asbestosis – etiology and pathogenesis

Precise mechanism formation of the disease is unknown
– probably chronic irritation of lung tissue

Development of disease depend on:

- Form of asbestos (length 3 –30 μm , thickness of fibers)
 - Concentration in inhaled air
 - Length of exposure - after exposure 10 years
 - Individual sensitivity
-
- Asbestos cause lung inflammation and fibrosis as a result of activation alveolar macrophages
 - Asbestos fibers can persist in the lungs many years after exposition
 - Typical is long latency period between exposure and development of asbestosis

Asbestos induced disorders

Non malignant

- **Asbestos induced pleural disorders**
 - **Pleural plaques** – on most movable parts of parietal pleura, into plaques can be stored calcium, pleural hyalinosis
 - **Benign exsudative pleuritis** – formation of exudates with spontaneous resorption
 - **Rounded atelectase** – serious warning on risk of malignant tumor
- **Asbestosis diffuse parenchymal lung disease**
 - Most often form – fibrotic rebuilding of lung tissue
 - Maximum changes is in both upper lobes

Malignant

- **Malignant mesothelioma**
 - Very invasive and metastatic cancer
- **Lung cancer**
 - Incidence is to 25 years after first exposure

Asbestosis – clinical picture

- **Clinical manifestation** after middle or heavy exposure more than 10 yy.
- **Dyspnea** – most common symptom – develop early and progress is faster than at silicosis
 - Initially decrease tolerance for exercise
- **Nonproductive cough** (sometimes productive with small volume of sputum)
- **Crepitations** – are typical, stabile – without changes after cough
- **Cyanosis** – more often as in case of silicosis
- Developing of global respiratory insufficiency and cor pulmonale
- **Asbestos bodies** (asbestos fibers covered with haemosiderin)
 - Light microscopic examination of sputum or liquid after bronchoalveolar lavage

Asbestosis – diagnostics

Objectification of work condition

- Dustiness
- Content of asbestos fibres

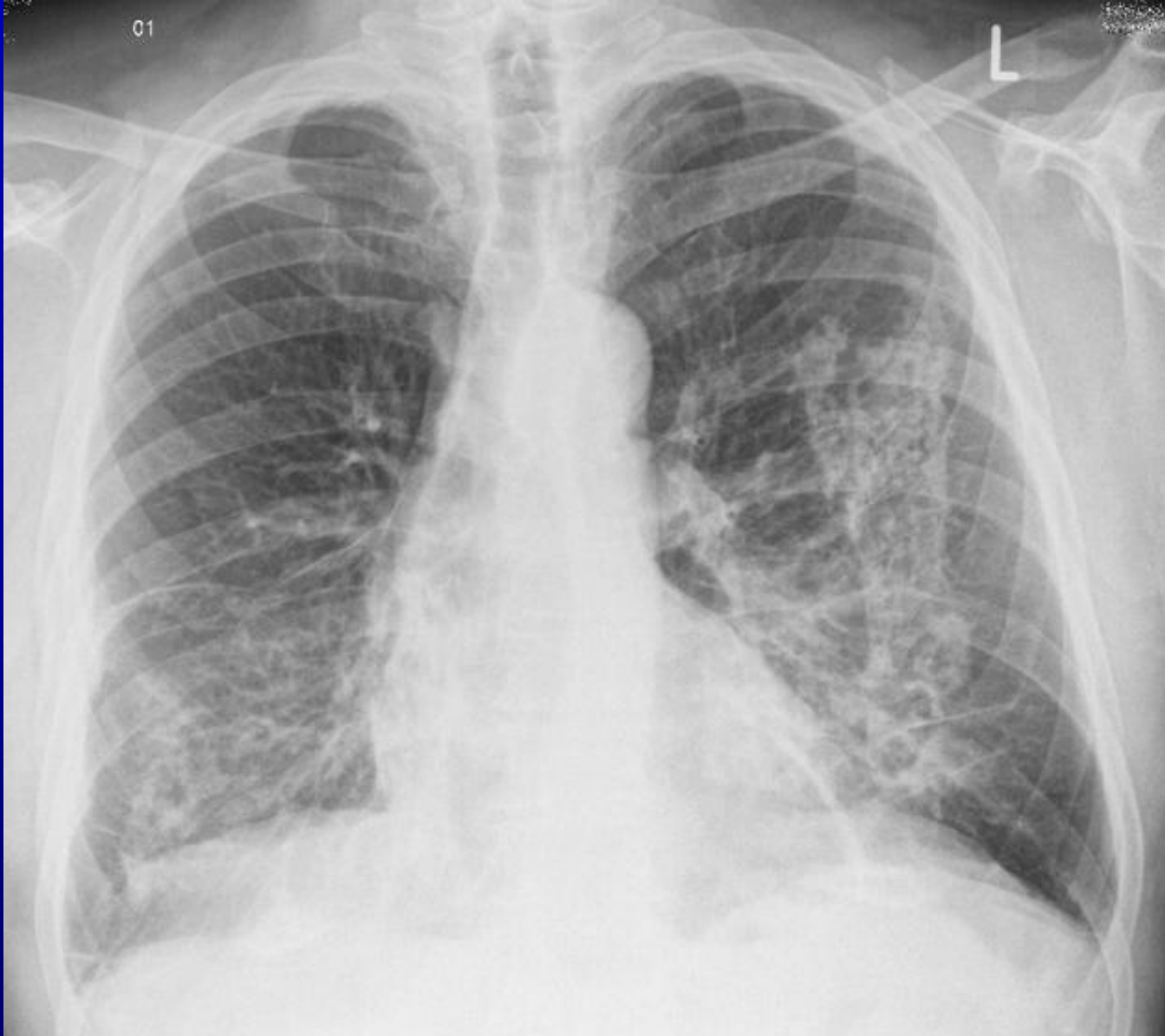
PA x-ray picture

- Symmetric, irregular, streaked opacities
- Pleural adhesions

Other diagnostics

- HRCT
- Spirometry
- ECG
- Blood gases

Treatment is only symptomatic.



Case courtesy of Dr Roberto Schubert, [Radiopaedia.org](https://radiopaedia.org/). From the case [rID: 17322](https://radiopaedia.org/cases/17322)

Asbestos induced lung cancer

- Manifestation of lung cancer is predominantly 15 – 20 years after first exposure
- Smokers exposed to asbestos have 20 – 50 times higher probability cancer formation than nonsmokers

Clinical picture

- Progressive dyspnea, pain on the chest
- Caught – repetitive haemoptysis and bronchopneumonias
- Total symptoms of malignity – lost of weight, fatigue, lassitude
- Repetitive pleural exudates

Diagnostics – x ray, HRCT, bronchoscopy, cytology of sputum, biopsy

- Histological types – squamous cell carcinoma, small cell carcinoma, adenocarcinoma

Treatment and prognosis are similar like other cancers.

Asbestos induced mesothelioma

- Tumor – originated from mesotelial cells lining the pleural space or peritoneum
- Mesothelioma is caused in 80% by asbestos exposure
 - Occurrence after massive exposure is 1 – 2 years
 - Typical occurrence is 20 – 25 years after exposure
 - Smoking doesn't have influence on incidence of mesothelioma
- Tumor can encroach to lungs tissue or chest wall
- Types of mesothelioma – pleural 70%, peritoneal 20%, pericardial and testicular 10% – death from respiratory failure occurs within months

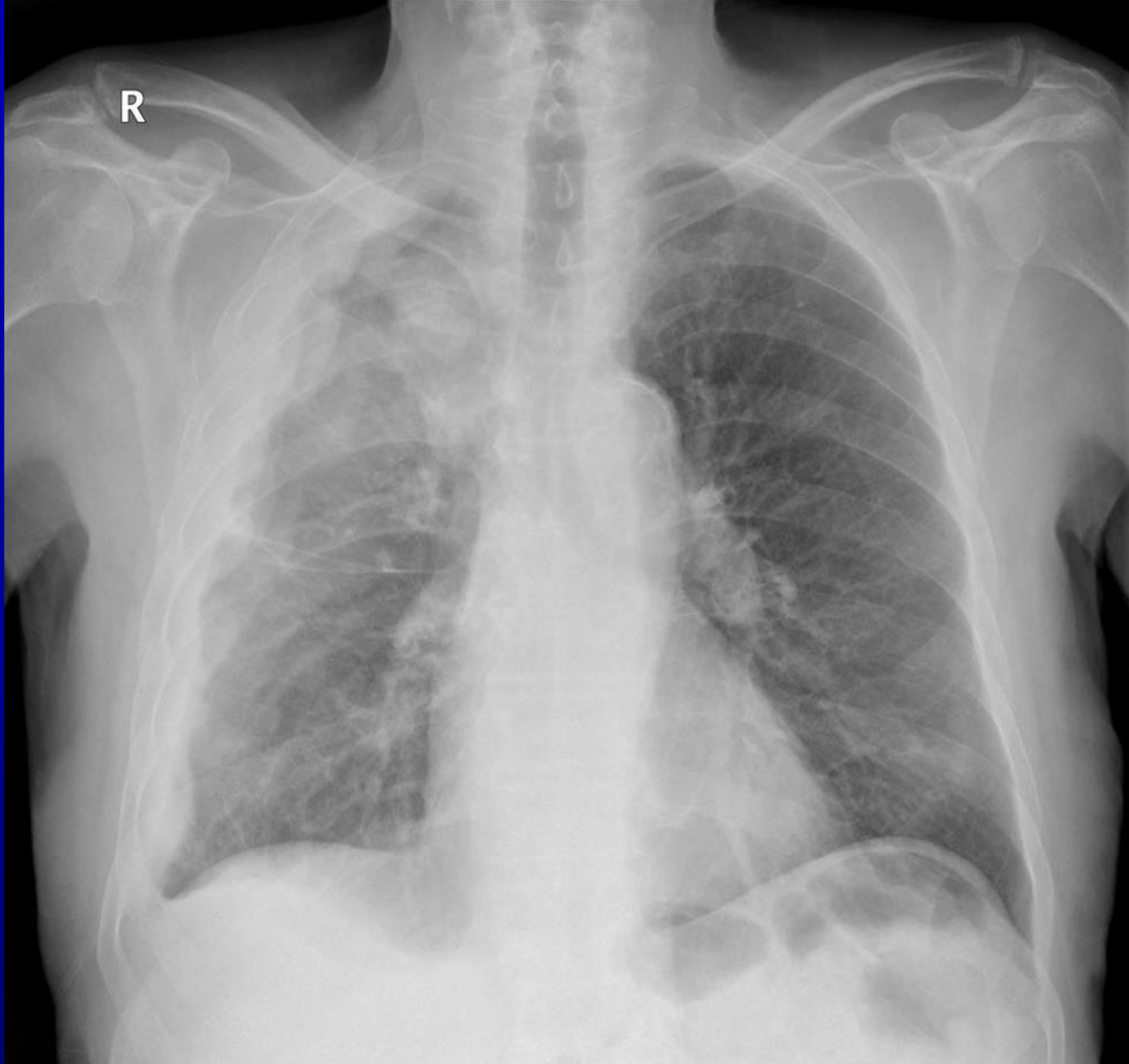
Asbestos induced mesothelioma

Symptoms of pleural mesothelioma

- Persistent dry cough, Dyspnea
- Persistent chest pain, or pain while breathing
- Exudative pleuritides – epithelial and mix forms of tumor
- Mesenchymal type – exudates are rarely, after short time make extrapulmonal metastases
- Total symptoms of malignity – lost of weight, cachectisation, fatigue, lassitude

Diagnostic – x-ray, HRCT, cytological examination, biopsy

Treatment – surgical resection,
radiotherapy, chemotherapy (unsatisfactory), analgesics



©Case courtesy of Dr Paul Leong, [Radiopaedia.org](https://radiopaedia.org/). From the case [ID: 26805](https://radiopaedia.org/cases/26805)