Heavy Metal Poisoning

By

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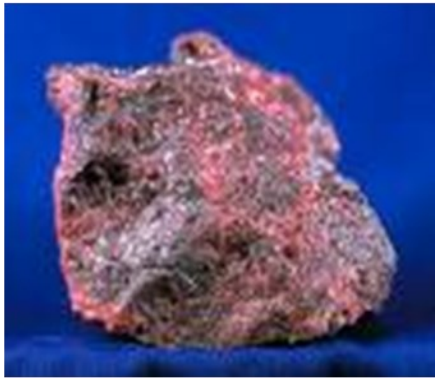
Chemical Hazards I

Arsenic

Lead



Mercury

Emilia Zainal

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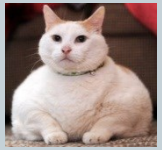
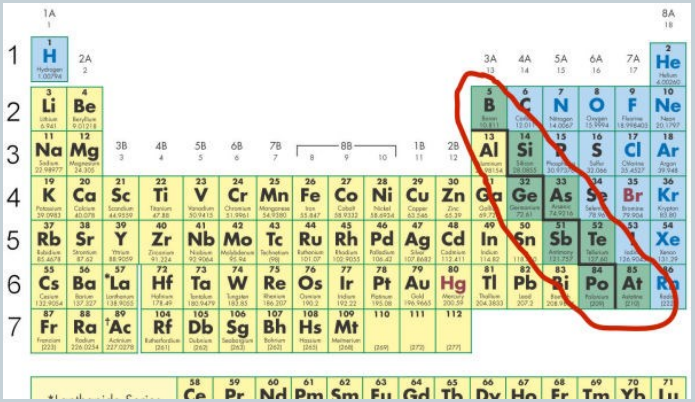
UPM

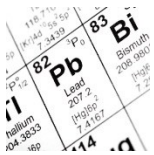
Definition

- ‘Metals’ originally included only gold, silver, copper, iron, lead, and tin.
  - Dense, malleable (able to be hammered or pressed permanently out of shape without breaking or cracking), lustrous
  - Conduct heat and electricity, cations

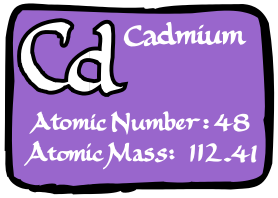


- Many other elements since added to the list with some of these characteristics
- ‘Metalloids’ are elements with features intermediate between metals and non-metals
- Example: Arsenic - near or in hazardous waste sites and areas with high levels naturally occurring in soil, rocks, and water





Heavy metal



- Heavy metal are chemicals elements with a specific gravity that is at least 5 times the specific gravity of water
  - **Arsenic 5.7; cadmium 8.65; lead 11.34; mercury 13.54**
- A metal having an atomic weight greater than Na, a density greater than 5 g/cm^3
- Physical properties
  - **High reflectivity, electrical and thermal conductivity, strength**
  - **Easily traced and measured and fate determined**
- Some notion of toxicity
- Usually includes lead, cadmium and mercury

Metals in workplace

- Metals are extensively used in industrial operation thus resulting in a high risk of exposure to workers and environment
  - Welding
  - Grinding
  - Soldering
  - Painting
  - Smelting
  - Storage battery
  - Recycling
- Industries with high potential of lead exposures include construction work, most smelter operations, radiator repair shops, and firing ranges.
- Cadmium is found in industrial workplaces, particularly where any ore is being processed or smelted.
- Common sources of mercury exposure include mining, production, and transportation of mercury, oil and gas industry as well as mining and refining of gold and silver ores.

Recycling industry

Shipbreaking industry

- Mercury is a naturally occurring trace element in fossil fuels
- It is predominantly present in the metallic form but may be present in the form of inorganic salts and organic species.

Schedule 2-USECHH 2000

- METALS/Chemicals for which medical surveillance must be performed
  1. Arsenic and any of its compound
  2. Beryllium
  3. Cadmium
  4. Chromium
  5. Lead
  6. Manganese
  7. Mercury

Notifiable Occupational Poisoning and Disease

- 3rd schedule OSH (Notification of Accident, Dangerous Occurrence, Occupational Poisoning and Occupational Disease) Regulations (7) 2004
- Poisoning by Cd, Ar, Pb, Hg, Mn, Phosphorus, antimony, chromium, nickel, beryllium
- Column 2 in NADOPOD – the use of handling, or exposure to fumes, dust, vapour
  - FUMES
  - **Solid aerosols generated by the condensation of vapours or gases from combustion or other high temperature processes + Usually very small and spherical**

Compensable occupational disease (5th schedule SOCSO act 1969)

- Poisoning by
  - Lead or compound of lead
  - Arsenic
  - Mercury
  - Beryllium
  - Cadmium
  - Antimony
  - Nickel
  - Chromium

Guidelines on Mercury Management in Oil and Gas 2011 by DOSH

- **Chapter 2 - Mercury & Its Effects**
- **Chapter 3 - Mercury Health Risk Management**
- **Chapter 4 - Workplace Exposure Monitoring and Measurement**
- **Chapter 5 – Health Surveillance for Mercury Exposure – BEI from ACGIH**
- **Chapter 6 – Controlling Mercury Risks**
- **Chapter 7 – Mercury Decontamination**
- **Chapter 8 – Mercury Waste Management**
- **Chapter 9 - Mercury Emergency Response Chapter 10 – Personal Protective Equipment (PPE) Chapter 11 – Record Keeping**

(a) (b)

Figure 5 Example of mercury passive sampler (a) SKC 520 series and (b) Assay Technology X593

Biological monitoring and biological effect monitoring refers to laboratory and/or clinical testing of selected body fluids and/or tissues to evaluate the amount of the hazards present in the workers' bodies or the presence of early damage to target organs due to such exposures.

The specific types of biological monitoring for mercury exposure are as follows:

Table 11 Biological Exposure Determinants for Mercury[16]

Determinant	Biological Exposure Indices (BEI)
Total inorganic mercury in urine	35 µg/g creatinine
Total inorganic mercury in blood	15 µg/L

Understanding Metal Toxicity

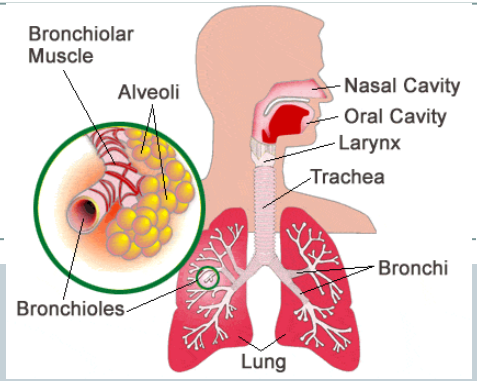
Fundamental concepts of : -

1. Classification of Metal
2. Absorption, storage and excretion of metal
3. Mode of action of metal toxicity

Classification of Metal

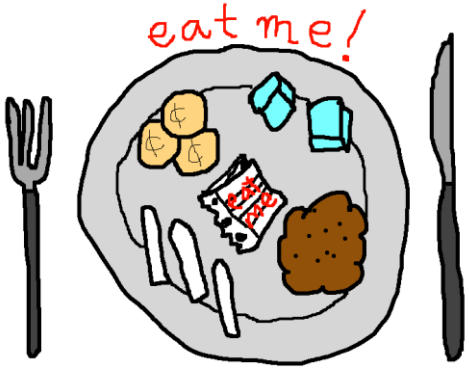
- Based upon physical properties
  - High reflectivity and metallic cluster
  - High electrical conductivity
  - High Thermal conductivity
  - Strength and Ductility - **characterized by the material's ability to be stretched**
- Base upon biological perspective
  - Solubility
  - Oxidation state
- Heavy metal ⇔ Toxic metals

Absorption

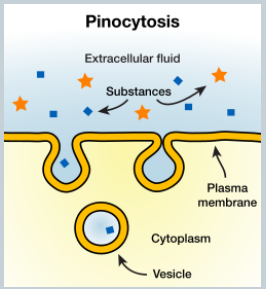


- Respiratory Absorption
  - Metal may be inhaled as vapor or aerosol (fume or dust particulate)
    - Fume or vapor of some metals & compound are readily absorbed in from alveolar space (cadmium, mercury, tetraethyl lead)
  - Large particles trapped in upper respiratory tract, cleared by mucociliary transport to pharynx and swallowed (equivalent to oral exposure)
    - Small particles may reach alveolar/gas exchange. Water soluble metal aerosols are rapidly absorbed from alveoli into the blood

Absorption



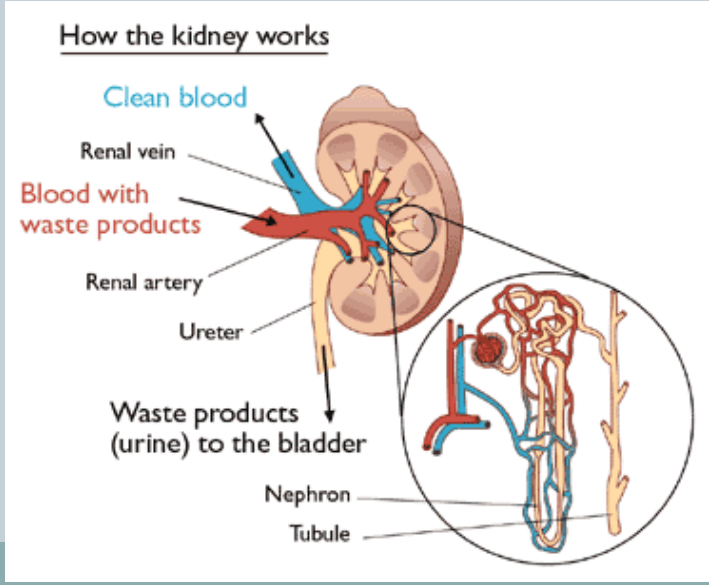
- Gastrointestinal Absorption
  - Metal may introduce into GI tract through food, water, mucociliary clearance
  - Metal are absorbed into the cells lining the intestinal tract by:
    - Passive or facilitated diffusion
    - Specific transport process
    - Pinocytosis



  - Depends on many factors
    - Solubility of metal in fluids of the intestinal tract
    - Chemical forms of metal (lipid soluble methyl mercury is completely absorbed compare to inorganic mercury – poorly absorbed)
    - Presence and composition of other materials in GI tract
    - Composition for absorption sites between similar metals (zinc & cadmium or calcium & lead)
    - Physiological state of the person exposed (Vitamin D enhance the absorption of lead)

Excretion

- Kidney - Important route of excretion
  - Metals in blood plasma are bound to plasma proteins and amino acids
  - Metals bound to low molecular weight proteins and amino acids are filtered in glomerulous into fluid of the renal tubule
  - Some metals (Cd & Zn) are effectively resorbed by tubular epithelia before they reach the urinary bladder where very little resorption occur



Excretion

- Enterohepatic Circulation
  - Absorbed metal may also excreted into intestinal tract in bile, pancreatic secretion or saliva
- Minor Pathways
  - Hair (Hg, Zn, Cu and As)
  - Nails
  - Saliva
  - Perspiration
  - Exhaled air
  - Lactation
  - Exfoliation of skin

Acute Toxicity of Metal

Organs and tissue affected are those involved in the absorption and elimination

- Result of the accumulation of high, critical concentrations of metal that at these sites with little opportunity to detoxify, eliminated or adapted to metal
- Tx of acute metal intoxication is design to:
  - Enhance the elimination of the metal through neutralization
  - Prevent irrriversible damage to organs and tissue
  - Treat the symptoms of acute toxicity

Chronic Toxicity

- Duration of initial exposure to the onset of signs and symptoms months to years
  - Diagnosis of chronic metal intoxication is more difficult than acute intoxication
- Diagnosis – presence of excessive metals in blood and urine
- Organ system not involve in absorption or elimination of metal such as hematopoetic or immune system may be affected

Mechanism of intoxication

- There is often little correlation between the sensitivity of organ or tissue to the toxic effects of metal and concentration in that tissue
  - **95% percent of the body burden of lead in adults are found in calcified tissue (bone and teeth); however toxicity is manifest primarily in the nervous systems, renal systems and hematopoetic systems**

Lead

Lead is a chemical element with symbol Pb (from the Latin *plumbum*) and atomic number 82. It is a heavy metal that is denser than most common materials. Lead is soft and malleable, and has a relatively low melting point. When freshly cut, lead is bluish-white; it tarnishes to a dull gray color when exposed to air. Lead has the highest atomic number of any stable element and concludes three major decay chains of heavier elements.

These properties, combined with its relative abundance and low cost, resulted in its extensive use in construction, plumbing, batteries, bullets and shot, weights, solders, pewters, fusible alloys, white paints, leaded gasoline, and radiation shielding.

The ancient Romans **used lead** to make water pipes, some of which are still in **use** today. ... Most of the **lead used** today is **used** in the production on **lead**-acid storage batteries, such as the batteries found in automobiles. Several **lead** alloys are widely **used**.

Lead

- Types of lead
  - Inorganic – PbO_2
  - Organic – Tetraethyl lead, tetrametyl lead, not water soluble lead
- Sources of exposure
  - Mining/Smelting (melting, baking, cooking, burning, and producing)
  - Cutting and welding lead-painted structure
  - Manufacture/Recycling of lead storage batteries
  - Production of lead based paints
- Routes of exposure
  - Respiratory tract
    - Dominant pathway – 50% absorbed
    - Particle size of lead dust <5 micron
    - Soluble
- Absorption
  - Inorganic lead is poorly absorbed from GI tract
  - Pregnant woman is 50%, normal adult

Lead poisoning
Absorption

- Skin:
  - little/no absorption
- Inhalation (<1µm):
  - dust or lead fumes
  - absorb 50-70%
- **Oral**:
  - adults absorb 10%
  - children absorb 40-50%
  - increased absorption if low Fe, Ca

Lead

Transport and storage

- Pb is transported to all organs and tissue of body by blood
- 95% of Pb in blood is associated with the erythrocytes and remain with plasma protein
- Lead accumulates in bone throughout life
- 90% of body burden of lead is found in bone and most remaining 10% in kidneys and liver
- Biological half-life of lead bone is 10-20 years, while half life of lead in soft tissues is several months

- Organ systems
  - GI
  - Hematopoetic
  - Nervous & neuromuscular
  - Renal and cardiovascular
  - Reproductive system – low sperm count, abortions, stillbirths, low sperm motility, premature baby
- Signs and symptoms include
  - Muscle weakness, anemia, Insomnia, loss of memory, headache, paralysis of extensor muscles of the wrist

Lead poisoning
Storage & Distribution

1. Rapid turnover soft tissue pool:
   - $T_{1/2}$ 30-40 days; blood, liver, kidney, CNS
2. Slow turnover skeletal pool:
   - $T_{1/2}$ 10-20 years; 75% - 90% in skeletal pool
   - Chronic exposure results in a steady state distribution between bone and blood

Excretion: Renal (90%) and biliary (10%)

- Maximum excretion is ~ 3.5µg/kg/day
- If intake > 3.5 µg/kg/day accumulation will occur

Lead

- Correlation between blood lead levels and clinical effects
  - < 40 ug/dl – usually none
  - 40 – 80 ug/dl – mild symptoms
  - > 80 ug/dl – severe manifestation such as convulsions
- Lead Regulation, 1984 under FMA
  - Action level
  - Airborne concentration of 75 ug/m3 of air averaged over 8-hour period
- PEL in airbone
  - 150 ug/m3

Lead poisoning

Sources

Occupational

- Lead smelters
- Painter/decorators
- Battery manufacturers
- Stain-glass workers
- Jewellery makers
- Bronze workers etc…

Environmental

- paint (walls, furniture, toys)
- water
- food
- air (petrol, industry), dust/soil

Other

- traditional remedies (Ayruvedic)
- surma & kohl cosmetics
- lead shot
- lead glazed ceramics
- foreign body ingestion

  e.g. curtain/fishing weight, snooker chalk

Lead

- Exposure monitoring
  - Full shift personal samples, at least 1 sample per work area
  - If below action level no further assessment needed unless there has been a change in production, process, control or personnel
  - If at or above action level, should repeat every 6 months
  - If at/above PEL, repeat every 3 months
- Medical surveillance
  - For all workers exposed above action level for more than 30 days per year
- Biological monitoring
  - At least every 6 months for exposed workers
  - Every 3-months if blood Pb 40-60 ug/100 gm blood
  - Monthly if 60-80, during removal period for female worker of child bearing capacity

Diagnosis of Lead Poisoning

- Blood lead is the best test (normal <100µg/l)
- Other bloods
  - FBC (film), U&E, LFT, Ca, Vit D, Ferritin
- Radiology
  - AXR ?lead in gut
  - Long bone XR in children
- Other tests much less reliable
  - Urine lead - variable, more useful for organic lead
  - RBC Zn protoporphyrin, Urine coproporphyrin, δALA

Lead toxicity

- The fall of the Roman empire was due to the fact they used lead for pipes to carry water and for drinking goblets and utensils.
- Sources - used in the past in medicines (sugar of lead), insecticides, pesticides, gasoline (tetraethyl lead), batteries, paints, manufacturing, automobile exhaust.
- Lead poisoning is probably the most important chronic environmental illness affecting modern children.
- The organ of greatest concern is the developing brain which last well into early adulthood.

Lead toxicity

- Mortality today is rare.
- Mounting evidence suggests that lead poisoning in children produces long term problems with learning, intelligence and earning power.
- Adults with lead poisoning have problems with depression, aggressive behavior and antisocial behavior.
- Males with lead poisoning have lower sperm counts, women have an increase in miscarriages and smaller babies

Lead Toxicity, cont.

Clinical features, plumbism:

Acute intoxication: not common

- Colic(misdiagnosis Acute Appendicitis)
- metallic taste to mouth
- vomiting, diarrhea or constipation
- increased thirst
- hemolysis, hemoglobinuria
- oliguria
- paresis and paresthesias

Lead Toxicity, cont.

Chronic lead intoxication - much more common

- Burtonian line - dark gray bluish black line on the gingival margin ($H_2S + Pb = PbS$)
- Basophilic stippling (clumping of RNA)
- Anemia
- Colic, diarrhea, vomiting
- Skeletal muscle weakness
- Increase uric acid in blood
- Headache, confusion, insomnia
- Lead palsy (wrist drop and foot drop)

Lead Toxicity, cont.

- Consider lead poisoning whenever a small child presents with peculiar symptoms that do not match any one particular disease entity.
- Especially:
  - irritability
  - sleeplessness
  - poor appetite
  - headaches
  - if parents use folk remedies or their parents work in a lead-related occupation

Lead Toxicity, cont.

<u>Patient management:</u>

- Acute intoxication - induce vomiting, give cathartics, give proteins to delay absorption (milk, egg whites), chelating agents
- Chronic toxicity - give chelating agents

Management of Lead Poisoning

- IDENTIFY & REMOVE from SOURCE
- Treat coexisting iron (& calcium) deficiency
- *Consider* the use of chelation therapy
  - Good data for benefit with blood lead >450µg/l (*children*)

Chelating agents for lead poisoning

1. EDTA - Sodium calcium edetate

2. DMSA - Dimercaptosuccinic acid

3. BAL - Dimercaprol

    - IM for severe toxicity only, particularly encephalopathy

4. Penicillamine - *no* longer recommended

EDTA and DMSA

- EDTA - Sodium Calcium Edetate
  - IV for severe toxicity, particularly encephalopathy
  - Well tolerated, <1% nephrotoxicity
- DMSA - 2,3dimercaptosuccinic acid
  - The oral agent of choice for lead poisoning
  - Given as a 19 day course
  - Well tolerated
  - The main problem is foul taste and smell !!

Lead – preventive measures

- Improvement of work process – elimination, substitution, enclosure, engineering control
- Work-place hygiene
- Appropriate PPE
- Appropriate signages
- Prevent childhood lead poisoning

Engineering control equipment

- Local exhaust ventilation system
- Water spray to control dust or
- Airborne chemical removal and containment equipment
- Maintenance requirements
  - During operational conditions
  - Monthly inspection
  - Annual examination and testing
  - Record keeping

Lead

- Control measures USCHH Regulation 15(2), (3)
- Safe work systems and practices
  - Documented
  - Implemented
  - Reviewed if
    - Changes to
      - Process
      - Equipment
      - Materials and control measures
- PPE Part V, USCHH regulation 16
  - Impracticable application of control measures a to g
  - As an interim measure
  - Control measures are not adequate

Test used to identify lead poisoning

- Blood lead
- Heme metabolism
  - Pb inhibits delta-amino-levulinic acid dehydratase (enzymes invovle in synthesis of porphyrins and heme)
  - Inhibition of the enzymes result in accumulation of the substrate aminolevulinic acid (ALA) in blood or urine
- Nerve conduction velocity
  - Lead decreases the velocity at which nerve impulse is conducted along the arm
- Ca EDTA mobilization test – estimate body burden of lead

Susceptibility of Nervous System to effects of toxins

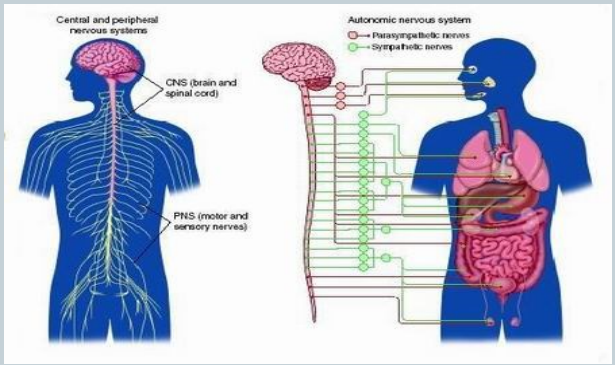
- Large surface area of nervous system – will increase exposure to toxins
- High lipid content (myelin) – accumulate and retain lipophilic toxins
- Neuron is sensitive to

shortage of O2

- Electrochemical transmission

at the synapse – toxins disrupts synaptic function

- Nerve cells killed by toxins cannot regenerate

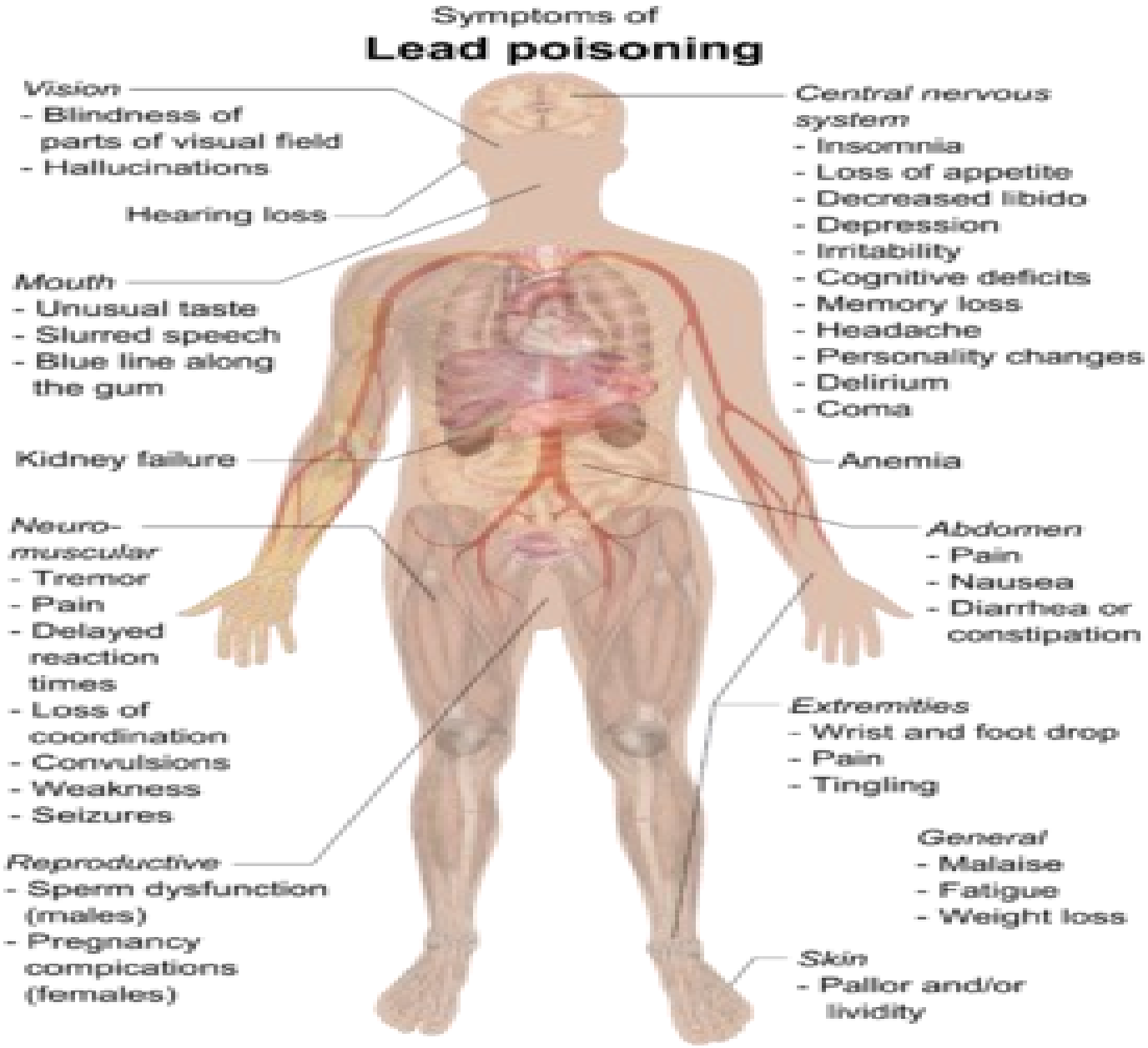


Investigation tools for neurological toxicity

WHO Neurobehavioural Core Test Battery

Detect subtle, mild neurological changes in early stage of intoxication

TEST	FUNCTIONAL DOMAIN
Simple reaction time	Attention/domain
Digit span	Auditory memory
Santa Ana dexterity test	Manual dexterity
Digit symbol	Perceptual-motor speed
Benton visual reaction	Visual perception
Aiming	Motor steadiness


Symptoms of
Lead poisoning
Vision
- Blindness of parts of visual field
- Hallucinations
Hearing loss
Mouth
- Unusual taste
- Slurred speech
- Blue line along the gum
Kidney failure
Neuro-muscular
- Tremor
- Pain
- Delayed reaction times
- Loss of coordination
- Convulsions
- Weakness
- Seizures
Reproductive
- Sperm dysfunction (males)
- Pregnancy compications (females)
Central nervous system
- Insomnia
- Loss of appetite
- Decreased libido
- Depression
- Irritability
- Cognitive deficits
- Memory loss
- Headache
- Personality changes
- Delirium
- Coma
Anemia
Abdomen
- Pain
- Nausea
- Diarrhea or constipation
Extremities
- Wrist and foot drop
- Pain
- Tingling
General
- Malaise
- Fatigue
- Weight loss
Skin
- Pallor and/or lividity

preventive measures

1. Improvement of work process – elimination, substitution, enclosure, engineering control
2. Work-place hygiene
3. Appropriate PPE
4. Prevent childhood lead poisoning
5. Engineering control equipment
    - Local exhaust ventilation system
    - Water spray to control dust or
    - Airborne chemical removal and containment equipment
    - Maintenance requirements
    - During operational conditions
    - Monthly inspection
    - Annual examination and testing
    - Record keeping

Cadmium

Sources

- By-product from smelting of lead & zinc ores
- Solders containing cadmium
- Welding
- Food & smoking

Absorption, Storage, Elimination

- Poorly absorbed from GI
- Inhaled cadmium is absorbed more efficiently (10 – 50 %) depends on size and solubility
- Absorb cadmium is bound to plasma proteins and transported to liver and accumulated in kidney
- Biological half life –20 years
- Renal Tubular damage occurs when the Cd concentration reaches or exceeds 200 ug/g wet weight in the kidney vortex

Cadmium

Toxic effects

- Mechanism
  - Displacing or replacing zinc from the many (over 200) enzymes requiring zinc as a catalytic or structural component
- Acute exposure to Cd fumes
  - Cough, chest pain, irritation to upper Resp. tract, respiratory damage
  - Death
- Chronic
  - Liver damage, anaemia, tetratogenic effects, renal tubular necrosis
- Facts – “Itai-Itai” is Japanese for “ouch-ouch” – refers to bone pain related to calcium loss

Cadmium

Diagnosis of intoxication

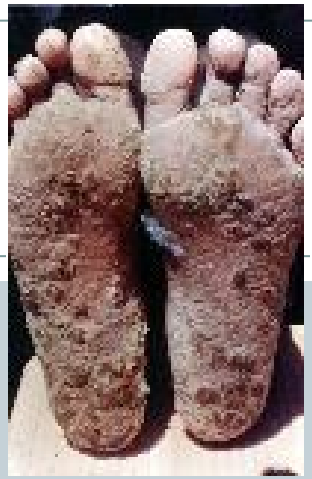
- History of exposure
- Increase urinary cadmium (blood cadmium)
- Reduce pulmonary function
- Impaired renal tubular function (proteinuria)

Arsenic

- Sources
  - Arsenic containing mineral ores
  - Industrial processes
    - Semiconductor manufacturing (gallium arsenide)
    - Fossil fuels
    - Wood treated with arsenic preservatives
    - Metallurgy
    - Smelting (copper, zinc, lead) and refining of metals and ores
    - Glass manufacturing
- Commercial products
  - Wood preservatives
  - Pesticides
  - Herbicides
  - Fungicides

Arsenic

Palmer Keratosis



- Toxicokinetics
  - $T_{1/2}$ of inorganic arsenic in the blood is 10 hrs and of organic arsenic is around 30 hours
  - 2-4 weeks after the exposure ceases, most of the remaining arsenic in the body is found in keratin-rich tissues (nails, hair, skin)
- Inorganic arsenic is converted to organic arsenic (biomethylation to monomethyl arsonic- MMA or DMA) in the liver
- This may represent a process of detoxification
- Renally excreted (30-50% of inorganic arsenic is excreted in about 3 days)
- Both forms are excreted depend on the acuteness of the exposure and dose

Manifestations of acute arsenic poisoning

Bodily system affected	Symptoms or signs	Time of onset
Systemic	Thirst Hypovolemia, Hypotension	Minutes Minutes to hours
Gastrointestinal	Garlic or metallic taste Burning mucosa Nausea and vomiting Diarrhea Abdominal pain Hematemesis Hematochezia, melena Rice-water stools	Immediate Immediate Minutes Minutes to hours Minutes to hours Minutes to hours Hours Hours
Hematopoietic system (formation of blood or blood cells in the body)	Hemolysis Hematuria Lymphopenia Pancytopenia	Minutes to hours Minutes to hours Several weeks Several weeks
Pulmonary (primarily in inhalational exposures)	Cough Dyspnea Chest Pain Pulmonary edema	Immediate Minutes to hours Minutes to hours Minutes to hours
Liver	Jaundice Fatty degeneration Central necrosis	Days Days Days
Kidneys	Proteinuria Hematuria Acute renal failure	Hours to days Hours to days Hours to days

Biological Monitoring

- Urinary arsenic measurement
  - Spot sample (mcg/L)
  - Timed urine collection (mcg/24 hours)
- Normal values
  - Spot urine= ~10 mcg/L (10-150 mcg/L)
  - 24 hours urine collection=<25 mcg/24 hours
  - Whole blood= < 1mcg/L (usually is elevated in acute intoxication)

MERCURY

Mercury is a chemical element with symbol Hg and atomic number 80. It is commonly known as quicksilver and was formerly named hydrargyrum (/haɪˈdrɑːrdʒərəm/).[4] A heavy, silvery d-block element, mercury is the only metallic element that is liquid at standard conditions for temperature and pressure

MERCURY

Mercury is used
in thermometers, barometers, manometers, sphygmomanometers, float valves, mercury switches, mercury relays, fluorescent lamps and other devices,
in amalgam for dental restoration

Table 1. Properties and Uses Of Mercury

PROPERTIES USES

1. Liquid metal ----------Barometers, blood pressure cuffs
2. Expands/contracts with temperature -Thermometers
3. Conducts electricity---------- ------Switches, fluorescent bulbs, electrolytic production of chlorine
4. Amalgamates with other metals ------- Dental fillings, gold purification
5. Kills bacteria and fungi---------- ------Disinfectants, preservatives

MERCURY

- Was used as “cure” for almost every a
  past
- Incident of methyl mercury
  - Minimata Bay 1953 – 1960
  - Methylmercury - The highly toxic compound bioaccumulated in fish and shellfish when eaten by the people living around the bay, gave rise to Minamata disease
  - On grain in Iraq 1971 – 1972
- Metabolism – Three form
  - Elemental – Hg^0
  - Inorganic : Hg^+ and Hg^{2+}
  - Organic

Table 4Types of Inorganic and Organic Mercury

Elemental		Inorganic Mercury		Organic Mercury	
Elemental Mercury	Hg^0	Mercury sulphide	HgS	Ethyl Mercury	$C_2H_5Hg^+$
		Mercury sulfate	$HgSO_4$	Methyl Mercury	$(CH_3Hg)^+$
		Mercury Oxide	HgO	Diethyl Mercury	$(C_2H_5)_2Hg$
		Mercury selenide	HgSe	Dimethyl Mercury	$(CH_3)_2Hg$
		Mercury Iodide	HgI_2	Dibutyl Mercury	$(n\text{-}C_4H_9)_2Hg$
		Mercury chloride	$HgCl_2$	Dipropyl Mercury	$(n\text{-}C_3H_7)_2Hg$
		Ionic Mercury	Hg^{+2}, Hg^{+1}	Di-isopropyl Mercury	$(iso\text{-}C_3H_7)_2Hg$

MERCURY

Widespread mercury poisoning occurred in rural Iraq in 1971–1972, when grain treated with a methylmercury-based fungicide that was intended for planting only was used by the rural population to make bread, causing at least 6530 cases of mercury poisoning and at least 459 deaths (see Basra poison grain disaster)

MERCURY

- Absorption
  - Hg^0 via respiratory tract (80% retained)
  - Hg^+ and Hg^{2+} about 7% retained
  - Organic Hg about 70% retained
- Distribution and Metabolism
  - Oxidation finally to Hg^{2+}
  - Affinity for kidney
- Excretion (half life 70 days for organic, 35-90 days for elemental)
  - Mainly via urine
  - Organic Hg mainly faecal
- Cross placenta

Symptoms of chronic and acute toxicity of inorganic mercury

Inorganic mercury intoxication

Acute	Chronic
Nausea Headache Diarrhea Abnormal pain Metallic taste	Ataxia – lack of muscle coordination Dysarthria – motor speech disorder Dysphagia – difficulty in swalloing Impaired vision Loss if coordination Hearing Taste & smell

Biological Effects

- Central Nervous System
  - Neuropsychiatric by Hg^0
    - Tremor, insomnia, emotional instability (erethism), depression
  - Sensorimotor for organic Hg
    - Tremor, loss of senses, incoordination, paralysis
  - Mechanism
    - Disrupts metabolism and causes degeneration of neurons
- Kidney
  - Mainly inorganic – tubular damage
- Others
  - Stomatitis
  - Gingivitis
  - Excessive salivation

Common symptoms of mercury poisoning include peripheral neuropathy, presenting as paresthesia or itching, burning, pain, or even a sensation that resembles small insects crawling on or under the skin (formication); skin discoloration (pink cheeks, fingertips and toes); swelling; and desquamation (shedding or peeling of skin).
Mercury irreversibly inhibits selenium-dependent enzymes (see below) and may also inactivate *S*-adenosyl-methionine, which is necessary for catecholamine catabolism by catechol-*O*-methyl transferase. Due to the body's inability to degrade catecholamines (e.g. epinephrine), a person suffering from mercury poisoning may experience profuse sweating, tachycardia (persistently faster-than-normal heart beat), increased salivation, and hypertension (high blood pressure).

Affected children may show red cheeks, nose and lips, loss of hair, teeth, and nails, transient rashes, hypotonia (muscle weakness), and increased sensitivity to light.
Other symptoms may include kidney dysfunction (e.g. Fanconi syndrome) is a syndrome of inadequate reabsorption in the proximal renal tubules[1] of the kidney.
or neuropsychiatric symptoms such as emotional lability, memory impairment, or insomnia.
Thus, the clinical presentation may resemble pheochromocytoma or Kawasaki disease. Desquamation (skin peeling) can occur with severe mercury poisoning acquired by handling elemental mercury.

Other symptoms may include kidney dysfunction (e.g. **Fanconi syndrome**) is a syndrome of inadequate reabsorption in the proximal renal tubules[1] of the kidney. **Fanconi syndrome** or **Fanconi's syndrome** is a syndrome of inadequate reabsorption in the proximal renal tubules[1] of the kidney. The syndrome can be caused by various underlying congenital or acquired diseases, by toxicity (for example, from toxic heavy metals), or by adverse drug reactions. It results in various small molecules of metabolism being passed into the urine instead of being reabsorbed from the tubular fluid(for example, glucose, amino acids, uric acid, phosphate, and bicarbonate). Fanconi syndrome affects the proximal tubules, namely, the proximal convoluted tubule (PCT), which is the first part of the tubule to process fluid after it is filtered through the glomerulus, and the proximal straight tubule (pars recta), which leads to the descending limb of the loop of Henle. The loss of phosphate results in the bone diseases rickets and osteomalacia

CHROMIUM

- Uses
  - Alloy with iron (stainless steel), cobalt, nickle
  - Chrome pigment
  - Tanning leather
  - Wood preservative
  - Anticorrosive in cooling system, boiler, oil drilling mud
  - Cement

Health Effects CHROMIUM

- Acute
  - Acute renal tubular necrosis
- Chronic
  - Skin allergic
  - Chrome ulceration & perforation of nasal septum
  - Skin ulceration
  - Cancer of respiratory tract (genotoxic mechanism)

Summary: Target-organ toxicity

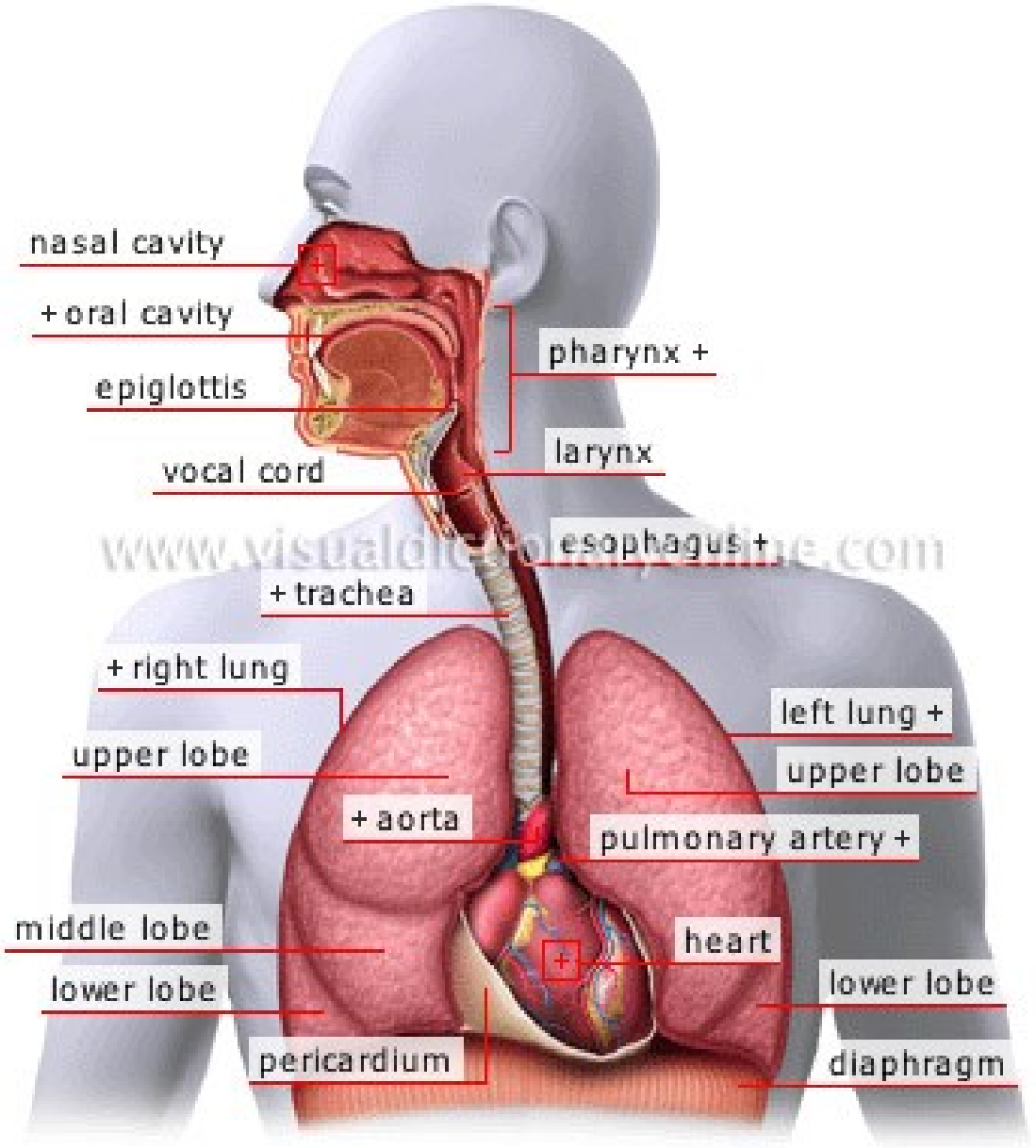
Metal	Kidney	Nerve	Liver	Gut	lung	Blood	bone	repro	Skin	Heart
Arsenic		+	+	+	+	+		+	+	+
Cadmium	+	+		+	+		+			+
Chromium			+		+				+	
Lead	+	+		+		+		+	+	
Mercury	+	+		+	+			+		
Nickel		+			+				+	

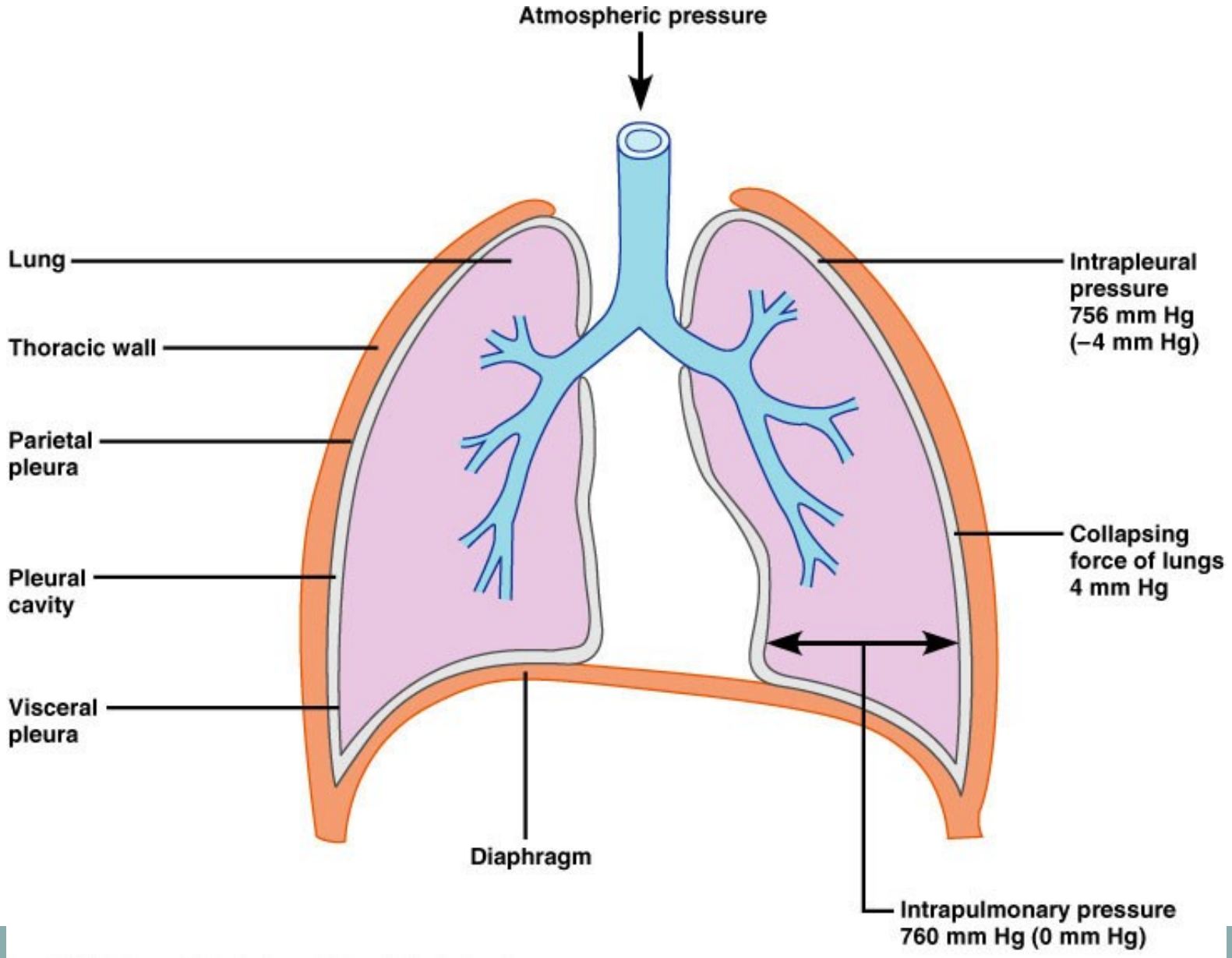
Suggested readings

- 1. Effects of nickel and beryllium
- 2. Permissible Exposure Limit – ceiling limit or 8-hour TWA or maximum exposure limits for these chemicals
- 3. Management and control of exposure to other chemicals beside lead

- Thank you for your attention.

Occupational lung diseases


nasal cavity
+ oral cavity
epiglottis
vocal cord
pharynx +
larynx
esophagus +
+ trachea
+ right lung
upper lobe
+ aorta
middle lobe
lower lobe
pericardium
left lung +
upper lobe
pulmonary artery +
heart
lower lobe
diaphragm


Atmospheric pressure
Lung
Thoracic wall
Parietal pleura
Pleural cavity
Visceral pleura
Diaphragm
Intrapleural pressure 756 mm Hg (–4 mm Hg)
Collapsing force of lungs 4 mm Hg
Intrapulmonary pressure 760 mm Hg (0 mm Hg)

Classification of Chemical Hazards

1. Dusts
2. Fumes
3. Mists
4. Vapors
5. Gases

Classification of Chemical Hazards

- Inorganic Chemicals
- Toxic gases
- Particle
- Organic

Inorganic Chemical

- Lead
- Mercury

Organic chemical

- Organic chemicals are carbon containing compounds
- DDT

Toxic gases

Simple asphyxiants	Nitrogen, CO2, methane
Chemical asphyxiants	CO, H2S
Irritant : U.R.T L.R.T	Ammonia, SO2 Oxides of nitrogen, phosgenen (NOx)

Particles

1. Coal dust
2. Silica dust
3. Asbestos
4. Cotton dust

The occupational lung diseases are caused by the inhalation of dusts, fumes, gases or vapors. Those which are caused by dusts are the most important. Pulmonary disease may also result from the inhalation of bacteria such as B. anthracis .

Four broad categories of occupational lung disease can be identified :-

1. **Obstructive airways disease**
2. **Granulomatous disease**
3. **Chemical pneumonitis**
4. **Pneumoconiosis**
    - **Fibrotic**
    - **Nonfibrotic**

Obstructive airways disease

Many of the dusts which are encountered in the industry are capable of producing asthmatic symptoms. Two types of asthma can be differentiated:-

A. Immediate type

B. Late or intermission type

Immediate asthma

This is reaginic (type 1) asthma occurring predominantly in atopic subjects. It arises in susceptible subjects during the handling of wool, furs, or feathers.

Late or intermission asthma

This is non-reaginic type of asthma, the symptoms of which appear sometime after the exposure to the precipitating allergens has begun. Intermission asthma may be caused by cotton, gums used in printing industry, by a variety of wood dusts and by formalin vapor.

Asthma

- Characterized by dyspnea, wheezing, and chest tightness
- Active inflammation of the airways precedes bronchospasms
- Airway inflammation is an immune response caused by release of IL-4 and IL-5, which stimulate IgE and recruit inflammatory cells
- Airways thickened with inflammatory exudates magnify the effect of bronchospasms

There are two types of lung diseases:-

A. Obstructive

- **Decrease FEV1**
- **Decrease FEV1/FVC Ratio**

B. Restrictive

- **Decrease FVC**
- **Normal FEV1/FVC Ratio**

Chronic Obstructive Pulmonary Disease (COPD)

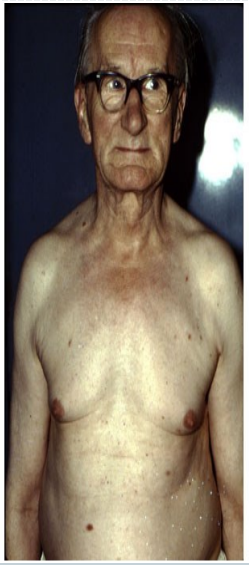
- Exemplified by chronic bronchitis and obstructive emphysema
- Patients have a history of:
  - Smoking
  - Dyspnea, where labored breathing occurs and gets progressively worse
  - Coughing and frequent pulmonary infections
- COPD victims develop respiratory failure accompanied by hypoxemia, carbon dioxide retention, and respiratory acidosis

Chronic Obstructive Pulmonary Disease

- progressive airflow limitations
  caused by an abnormal inflammatory reaction
  to the chronic inhalation
  of particles

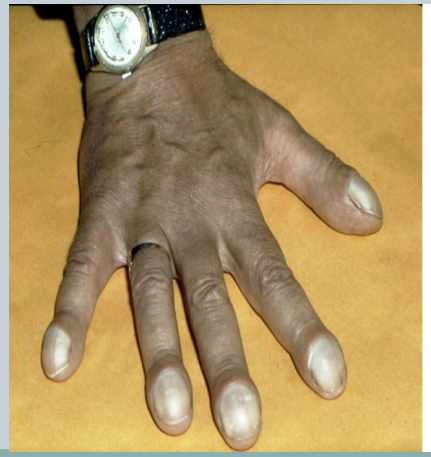
- chronic bronchitis and emphysema

- Signs of COPD are consequences of
  the anatomical changes caused by the disease:
  - barrel chest
  - pursed-lip breathing
  - productive cough
  - cyanosis.



Module #2

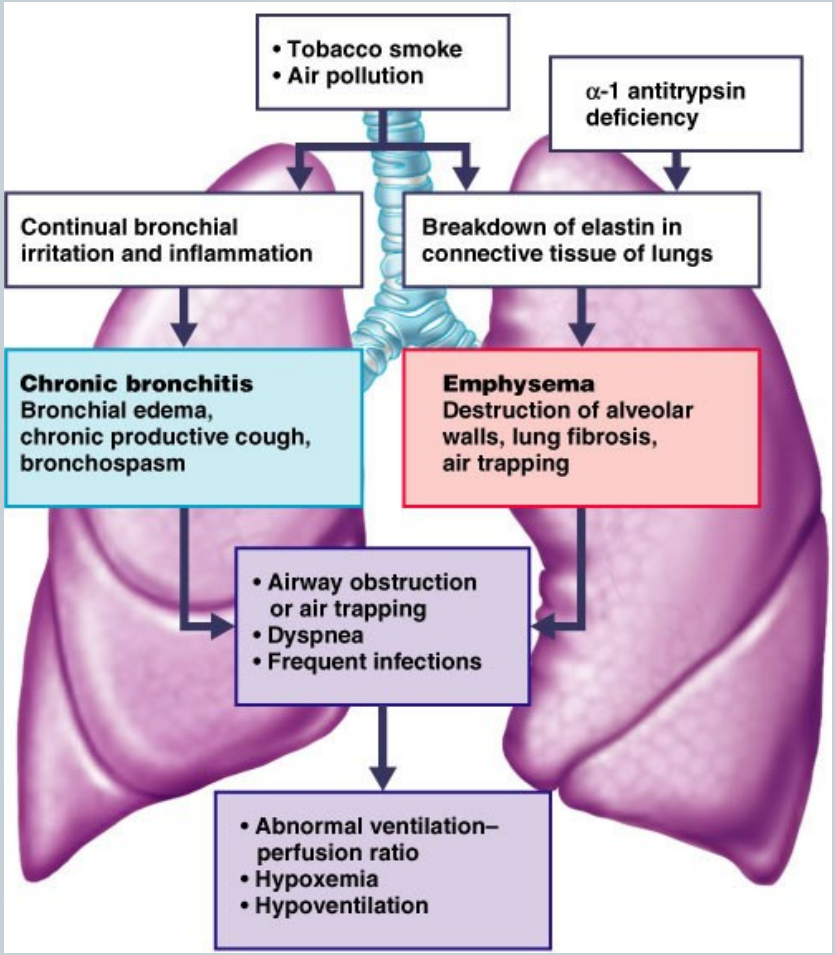
Barrel Chest



Module #2

Digital Clubbing

Pathogenesis of COPD



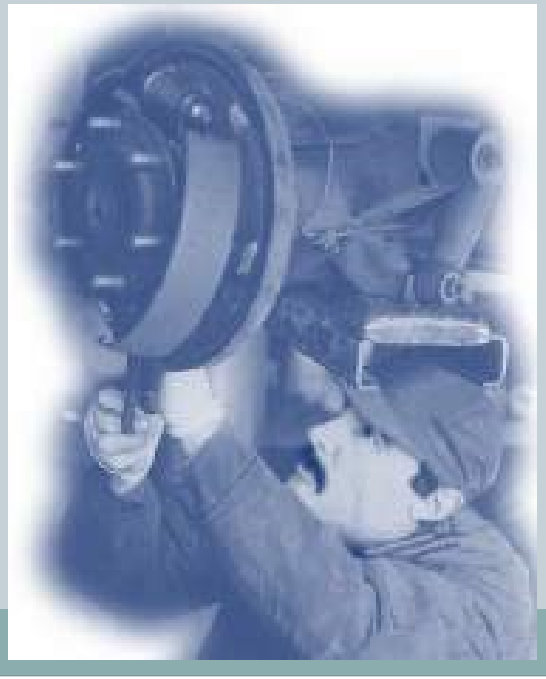
Respiratory functions in occupational lung diseases:-
Use simple portable Respirometer
•Measurement of Forced expiratory volume (FEV), usually the volume expelled in
one second (FEV1)
•The forced vital capacity (FVC)
•The vital capacity (VC)
There are three board categories of impairment in lung functions :-
1. Airway obstruction
Asthma, bronchitis, and emphysema produce some narrowing of the airway with a resultant obstruction to air follow, predominantly during Expiration
In asthma –decrease FEV1
VC is larger than FVC
2. Reduced compliance
Diffuse interstitial fibrosis—the lung are unable to expand fully and there is a resultant decrease in VC and decrease FVC
Asbestoses is the most important occupational disease and the VC is an excellent screening test for lung damage in asbestos workers.
3. Impairment of gas exchange.

Asbestos

- Properties of asbestos
- Uses of asbestos
- Health hazards of asbestos
- Activities resulting in potential asbestos exposure
- Asbestos regulations

General Overview

- 1.3 million workers exposed in the U.S.
- Construction Industry
  - renovation, demolition heaviest exposures



- **General Industry**
  - **manufacture of asbestos products**
  - **automotive brake and clutch repair**
  - **housekeeping, custodial**

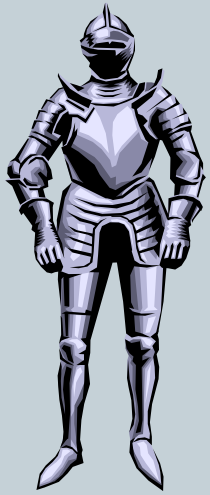
Properties of Asbestos

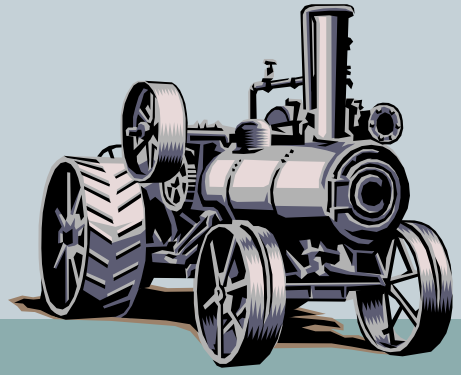
- Naturally occurring fibrous minerals
- Good tensile strength
- Flexibility
- Heat resistant
- Electrical resistance
- Good insulation
- Chemical resistant

Uses of Asbestos

Asbestos has been used for centuries

- Egyptians; Greeks & Romans
  wrapping pharaohs; lamp wicks, cloth
- Middle Ages
  insulating armor
- Industrial Revolution
  insulating boilers, steam pipes, turbines





- **Twentieth century –**
  World War II + next 30 years
  insulating; fireproofing; sound-proofing; decorating; strengthening

Uses of Asbestos

Asbestos insulated pipe in utility space

Asbestos "CAB" siding

- Thermal system insulation
- Surfacing materials
- Reinforcement of materials
- Fireproofing
- Acoustic and decorative plaster
- Textiles

Uses of Asbestos

- Friction materials (brakes, clutches, etc.)
- Asphalt and vinyl felts
- Papers and adhesives
- Flooring and roofing materials
- Filters, sealants, caulk, and gaskets

Sprayed-on fireproofing material

Vinyl flooring

Some Asbestos-Containing Materials

(This list does not include every product/material that may contain asbestos. It is intended as a general guide to show which types of materials may contain asbestos.)

- **Cement Pipes**
- **Cement Wallboard**
- **Cement Siding**
- **Asphalt Floor Tile**
- **Vinyl Floor Tile**
- **Vinyl Sheet Flooring**
- **Flooring Backing**
- **Construction Mastics**
- **(floor tile, carpet, ceiling tile, etc.)**
- **Acoustical Plaster**
- **Decorative Plaster**
- **Textured Paints/Coatings**
- **Ceiling Tiles and Lay-in Panels**
- **Spray-Applied Insulation**
- **Blown-in Insulation**
- **Fireproofing Materials**
- **Taping Compounds (thermal)**
- **Packing Materials (for wall/floor penetrations)**
- **High Temperature Gaskets**
- **Laboratory Hoods/Table Tops**
- **Laboratory Gloves**
- **Fire Blankets**
- **Fire Curtains**

Some Asbestos-Containing Materials

(Continued)

- Elevator Equipment Panels
- Elevator Brake Shoes
- HVAC Duct Insulation
- Boiler Insulation
- Breaching Insulation
- Ductwork Flexible Fabric Connections
- Cooling Towers
- Pipe Insulation (corrugated air-cell, block, etc.)
- Heating and Electrical Ducts
- Electrical Panel Partitions
- Electrical Cloth
- Electric Wiring Insulation
- Chalkboards
- Roofing Shingles
- Roofing Felt
- Base Flashing
- Thermal Paper Products
- Fire Doors
- Caulking/Putties
- Adhesives
- Wallboard
- Joint Compounds
- Vinyl Wall Coverings
- Spackling Compounds

“ACM” and “PACM”

Asbestos Containing Material

Any material containing more than 1% asbestos by weight.

Presumed Asbestos Containing Material

Installed prior to 1981

- **Surfacing materials**
- **Thermal System Insulation**
- **Flooring**

Must be handled as ACM unless proved otherwise

Many uses of asbestos have been banned under EPA and Consumer

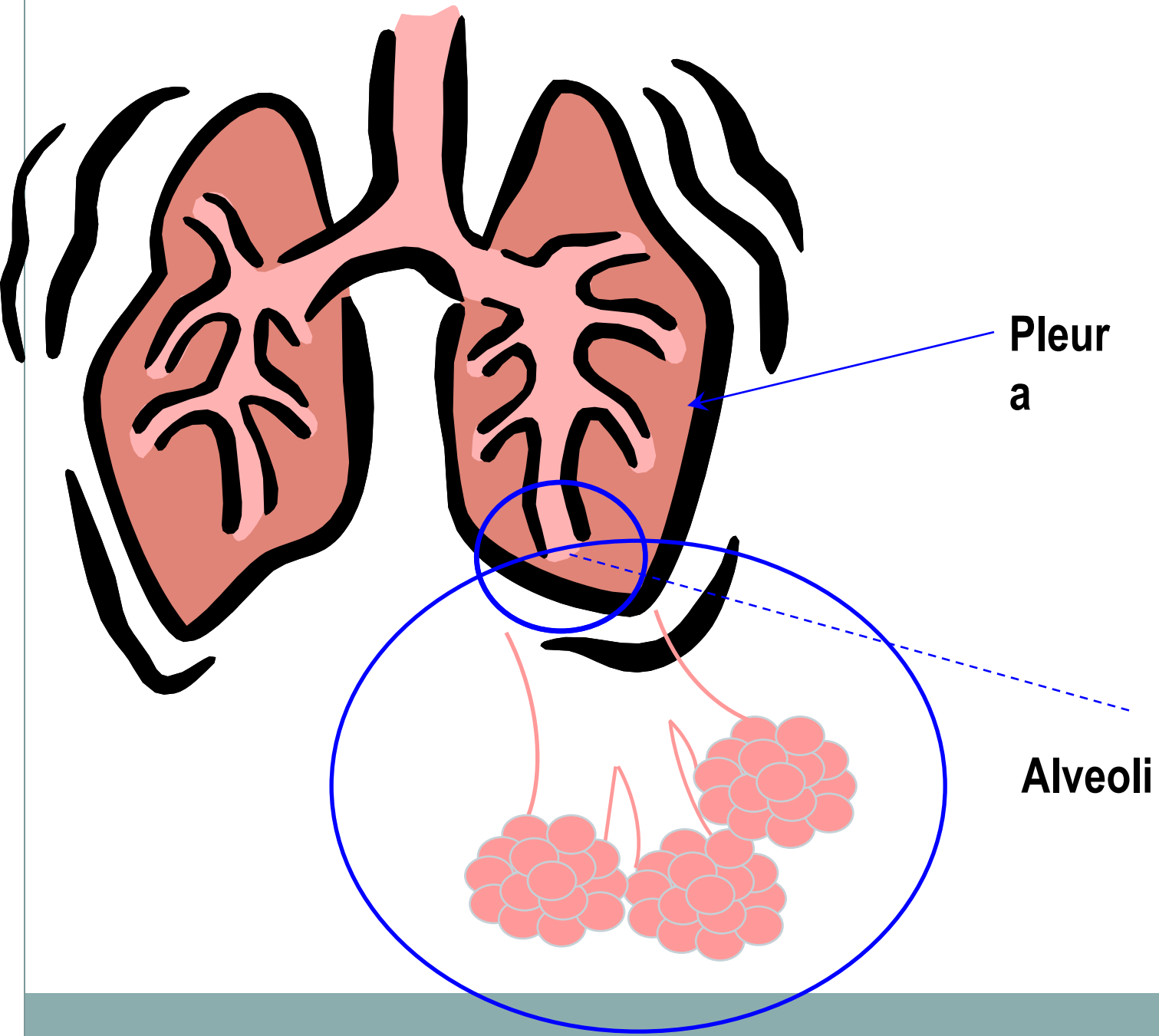
Product Safety Commission regulations. However, some materials where asbestos

fibers are generally well bound in the materials were not banned.

Previously installed products still pose a hazard to workers. Asbestos fibers can be released during repair work, demolition, and renovation of older buildings and structures containing ACM.

Asbestos is an Inhalation Hazard

- Airborne asbestos fibers inhaled deep into the lung can cause damage.
- Breathable fibers are deposited in the alveoli, the ending small air sacs in the lungs.
- Body's defense mechanisms cannot break down the fibers.
- Fibers cause damage to respiratory system.
- Fibers may also travel to the pleura, the membrane lining the lungs.


Pleur
a
Alveoli

Asbestos Exposure: Causes and Risks

- **Asbestos exposure may occur in the workplace, home, or community. Mined and used commercially since the 1800s, asbestos has been used in many products, including: car brake shoes and clutch pads; building materials, including ceiling and floor tiles; paints, coatings, and adhesives; plastics; vermiculite-containing garden products; and some talc-containing crayons. Due to federal regulations and health concerns, asbestos is much less widely used than it was just a few decades ago.**
- **Most cases of asbestos poisoning occur in asbestos workers; however, there is some evidence that family members of workers heavily exposed to asbestos face an increased risk of developing mesothelioma, possibly due to the exposure of asbestos fibers brought into the home on the clothing, skin, and hair. Cases of mesothelioma have also been seen in people living close to asbestos mines.**
- **Another group at risk of developing asbestos-related disease is workers involved in the Sept. 11, 2001, attack on the World Trade Center in New York City. Asbestos was used in the construction of the North Tower, and hundreds of tons of asbestos were released into the atmosphere during the attack.**

Asbestos-related Diseases

Asbestos can cause disabling respiratory disease, cancer, and eventually death.

- Asbestosis
- Mesothelioma
- Lung Cancer
- Other cancers

- Usually symptoms take 15 to 30 years or more to develop.
- Health effects from asbestos exposure may continue to progress even after exposure is stopped.

Asbestosis is a serious chronic, progressive disease that can eventually lead to disability or death in people exposed to high amounts of asbestos over a long period.

Asbestos fibers cause the lung tissues to scar; when the scarring spreads, it becomes harder and harder to breathe. Symptoms include shortness of breath, a dry crackling sound in the lungs while inhaling, coughing, and chest pain. This condition is permanent and there is no effective treatment.

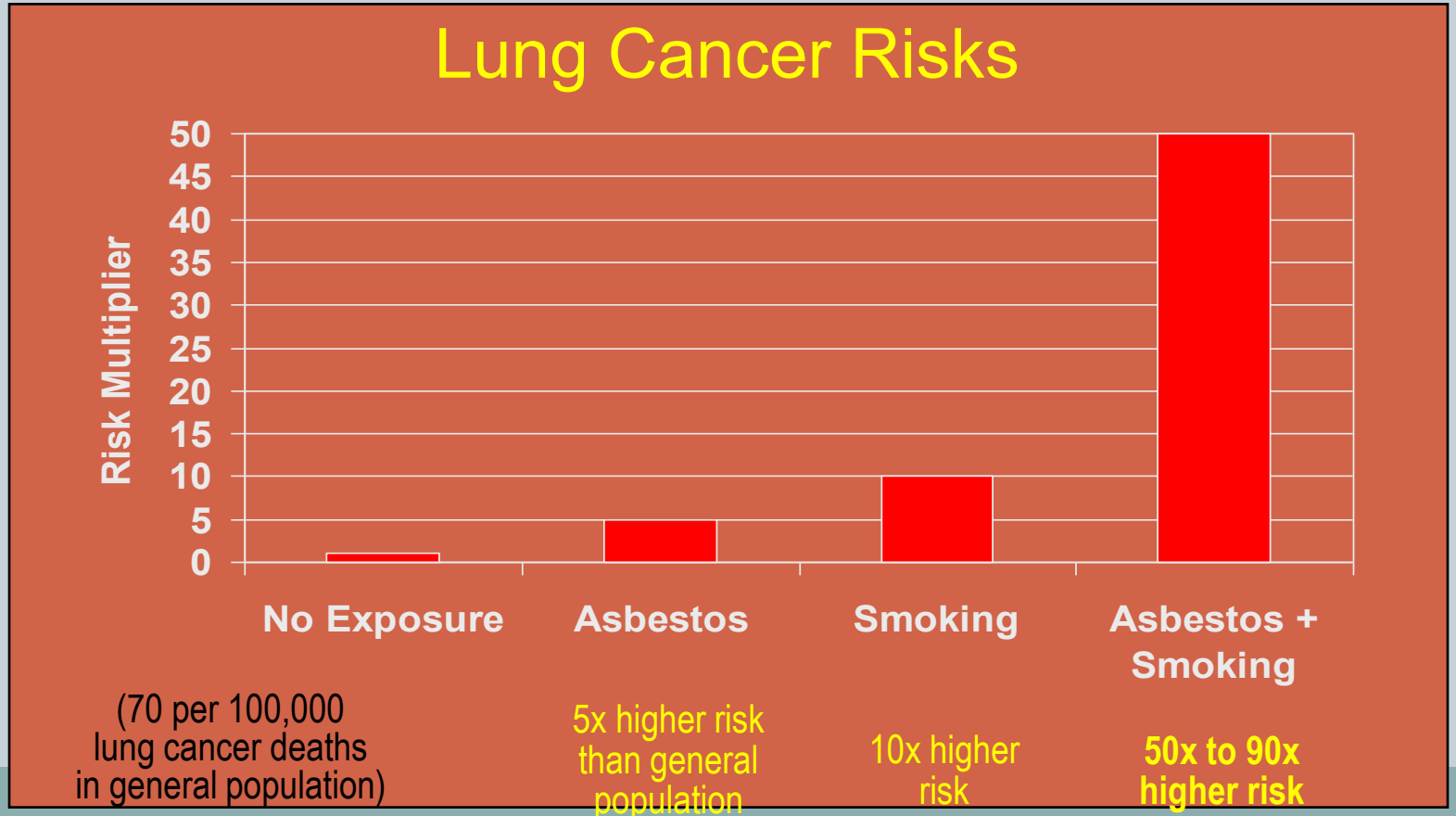
Mesothelioma Example

Mesothelioma is a rare form of cancer of the pleura, the thin membrane lining the lungs. About 200 cases are diagnosed each year in the U.S. Virtually all cases are linked with asbestos exposure.

The cancer is very invasive and spreads quickly, eventually crushing the lungs so that the patient cannot breathe. It is painful and always fatal. It can be caused by very low exposure and is not directly related to dose. May take 30-40 years to develop.

Lung Cancer

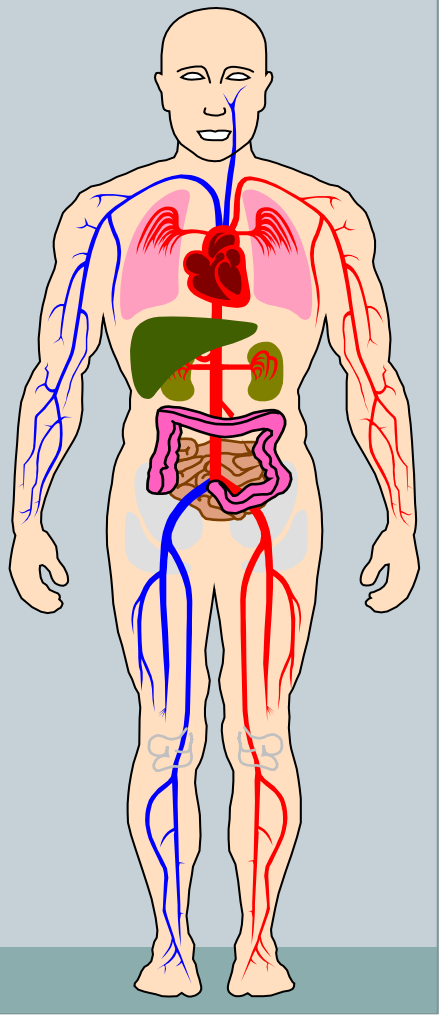
Lung cancer causes the largest number of deaths from asbestos exposure. The risk greatly increases in workers who smoke.



Other Cancers

Evidence suggests that ingesting asbestos can cause cancers in the:

- Esophagus
- larynx
- oral cavity
- stomach
- colon
- Kidney



Fibers can enter the mouth and be swallowed. Poor hygiene, leaving food/drinks out in contaminated areas, and carelessness can result in the ingestion of asbestos.

Asbestos-related Diseases

The potential for asbestos–related disease depends on:

- **Amount of fibers inhaled**
- **Length of exposure**
- **Whether exposed worker smokes**
- **Age**

Don’t smoke! An asbestos worker is at much greater risk of developing lung cancer if he/she smokes.

“King of Cool” - Steve McQueen

- **Died of Mesothelioma in 1980 at the Age of 50**
- **Exposed to Asbestos from Work/Hobbies**
  - **Brake Pads on Cars**
  - **Construction Work**
  - **Ship Work**

How do asbestos fibers get in the air?

Photo of friable asbestos

Asbestos is most hazardous when it is "FRIABLE".

•Friable: can be easily crumbled or crushed by hand, releasing fibers into the air

•Very small fibers stay in the air for long periods

•Damaged or deteriorated ACM increases friability

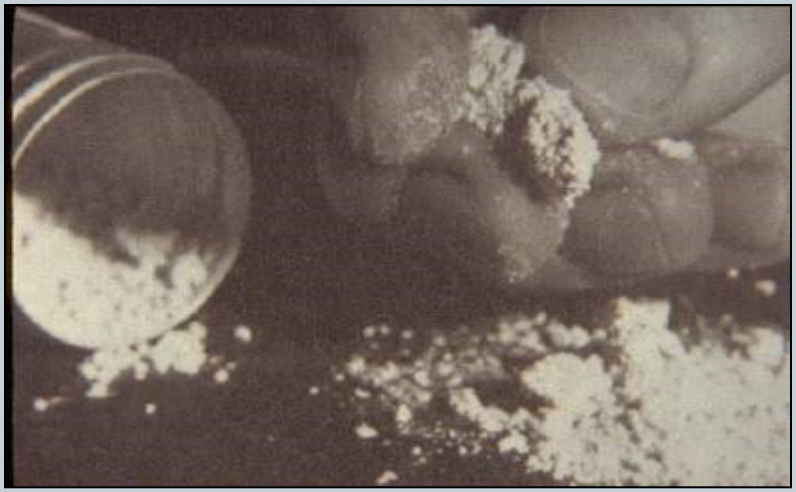


Photo of friable asbestos

Non-friable ACM (floor and ceiling tiles, siding, fire doors, etc.) won't release fibers unless disturbed or damaged in some way

How do asbestos fibers get in the air?

Activities and situations that can result in workers inhaling asbestos fibers:

- **Mechanical action on ACM (cutting, sawing, grinding, sanding, drilling, buffing)**
- **Disturbing/breaking ceiling tiles**
- **Removing/replacing insulation**
- **Disturbing sprayed-on asbestos**
- **Damaged/deteriorated ACM**
- **Asbestos abatement project**
- **Un-surveyed construction projects on older buildings**

Water damage, deterioration

Water damage, deterioration

General Safety and Health Requirements

You must implement the following measures to minimize employee exposure to asbestos:

- Hazard assessment; exposure evaluation
- **Communication of hazards**
- **Exposure and medical monitoring, notification**
- **Medical surveillance, exposure records**
- **Exposure Control Program**
  - **written program required if TWA_8 and/or STEL is exceeded**

Permissible Exposure Limits (PEL)

- **0.1 fibers per cubic centimeter of air (0.1 f/cc) 8-hour time weighted average (TWA_8)**
- **1.0 f/cc 30-minute short-term exposure limit (STEL)**

Exposure Control

Asbestos exposure must be controlled by one or more of the following engineering and work practices:

- **Local exhaust ventilation with HEPA filter system**
- **HEPA-filtered vacuums**
- **Enclosure, isolation**
- **Wet methods**
- **Prompt disposal**
- **Housekeeping**

HEPA filter = high efficiency particulate air filter HEPA vacuum units

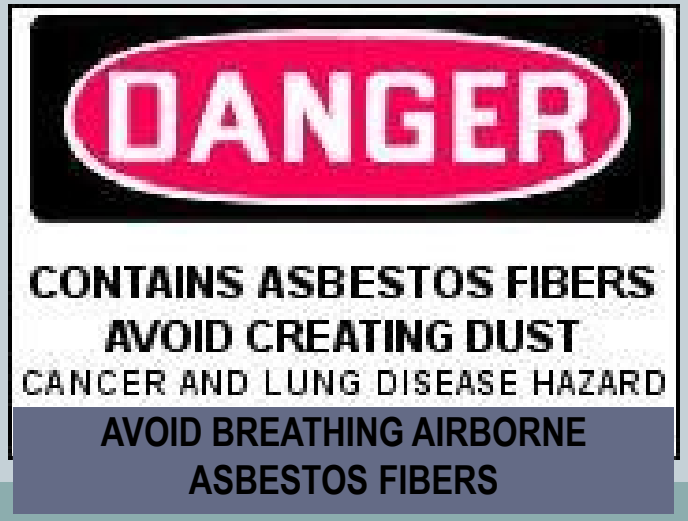
Exposure Control

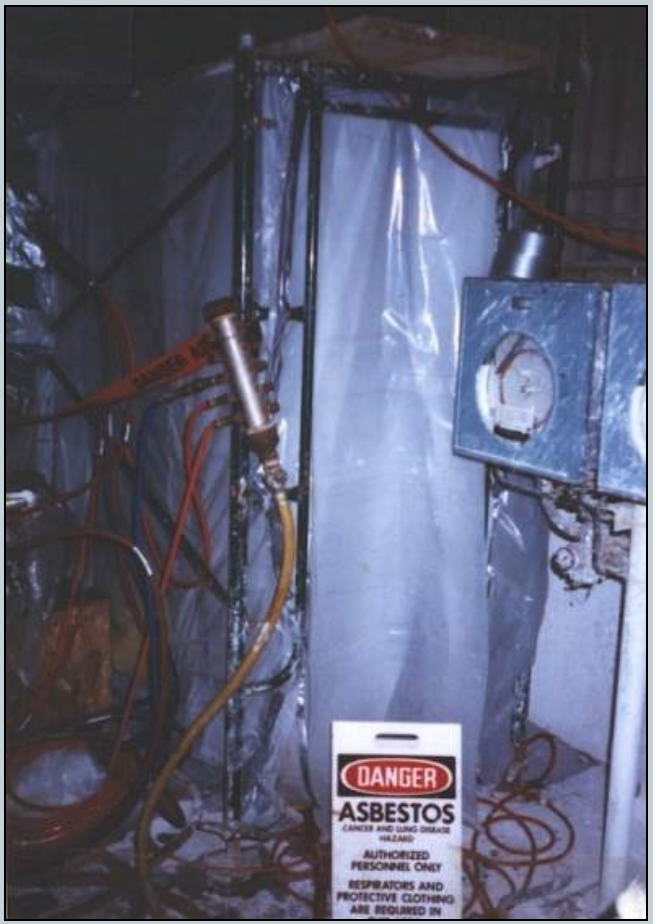
Decontamination shower

- **Worker training**
  - "Asbestos Awareness"
  - Initially and annually
  - Work practices, safety procedures
  - Program elements
- **Worker protection**
  - PPE: respirator, gloves, head and foot protection, coveralls
  - Hygiene facilities: decontamination, change room

Communication of Hazards

- **Warning Signs**
  - **regulated areas**
  - **visible before entering**
- **Warning Labels**
  - **attached to all products and their containers**





Entrance to regulated area

Building/Facility Owner

Responsibilities

- Determine presence, location, and quantity of ACM/PACM.
- Inform employers, employees, and others who may be impacted.
- Permit only certified individuals to perform work that may release asbestos fibers into the air.
- Submit “Notice of Asbestos Abatement Project,” which has to be received by Nevada OSHA enforcement section 10 days prior to engaging in an asbestos abatement project.

Construction/Maintenance Activities

The regulations cover any activity releasing or likely to release asbestos fibers into the air:

- **construction** **maintenance, repair**
- **Renovation** **demolition**
- **Remodeling** **removal, disposal**

Covers work done in:

- **buildings**
- **structures**
- **mechanical piping equipment and systems**
- **ships**
- **other facilities**

Construction/Maintenance Activities

Asbestos Training Certification:

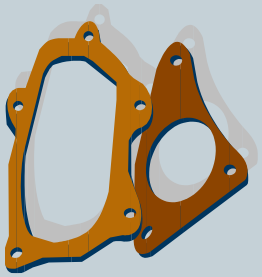
- **required when asbestos exposures are above PELs**
- **required for removal or encapsulation of any materials containing one percent asbestos and more by volume**
- **covers contractors, supervisors, workers (depending on type/size of work)**
- **additional training (depending on type/size of work)**

Asbestos abatement area contained and enclosed

Specific requirements for other work

- Automotive brake and clutch inspection, disassembly, repair, and assembly operations

- Roofing, flooring, siding and gaskets



• Custodial/Light maintenance

Asbestos

Is the condition of diffuse interstitial fibrosis which is typically distributed subpleurally over the lower half of the lungs. The pulmonary pleura may be thickened.

Bronchiectasis, emphysema, fibrosing alveolitis .

Inhalation of asbestos may give rise to four separate conditions :-

1. Asbestos is by which is meant fibrosis of the lungs with or without pleural fibrosis.

2. Hayaline pleural plaque formation.

3. Malignant mesothelioma.

4. Bronchial carcinoma.

Symptoms

1. Dyspnea

At first is moderate, the breathlessness increase in severity and patient find difficulty in taking deep breath because of the reduced compliance of the lung.

2. Cough

Physical signs

1. **Impairment of chest expansion**
2. **Persistent crepitations**
3. **finger clubbing**

Radiography

1. **Abnormal radiographic signs in asbestosis are more common in lower zones**
2. **Diffuse interstitial fibrosis with fine punctuate mottling.**
3. **A cystic or honey-comb appearance.**
4. **The costo-phrenic angles become obliterated and the cardiac outline becomes blurred.**
5. **Pleural thickening.**
6. **Pleural plaques, irregular shadows of patchy density.**

Clinical grades of Byssinosis

C½ --Occasional tightness of the chest on the first day of the working week.

C1—Tightness of the chest and/or difficulty in breathing on each first day only of the working week.

C2—Tight of the chest and/or difficulty in breathing on the first and other days of the working week.

C3—Grade C2 symptoms accompanied by evidence of permanent respiratory disability from reduced ventilatory capacity.

Signs and symptoms of silicosis

Divided into three arbitrary stages which merge into one another :-

1. First stage

Some degree of dsypnoea on exertion which gradually becomes more marked, may have unproductive cough.

2. Second stage

Is marked by dsypnoea which impairs the patients ability to work.

3. Third stage

The patient is totally incapacitated.

In the second stage—the clinical signs include some dullness to percussion, scattered rhonchi and bronchial breathing

In the third stage—signs of right heart failure will be noted.

Pathology

The principle lesion is a nodule composed of connective tissue. The nodules tend to predominate in the upper zones and may coalesce to form confluent masses.

The pleura are often adherent and thickened and the lungs may have a gritty feel. Some degree of bronchiectasis.

Radiography

Discrete rounded nodules, calcification in the periphery of the hillar lymph nodes, so-called egg-shell calcification.

Silicosis: Medical Aspects

Silica and Silicosis - definitions

- Silica is silicon dioxide, the oxide of silicon, chemical formula SiO_2.
- SiO_2 is the most abundant mineral on earth; comprises large part of granite, sandstone and slate.
- Silicosis is lung disease caused by inhalation of fine silica dust; the dust causes inflammation and then scarring of the lungs. Scarring shows up on chest x-ray.
- Silicosis is one type of pneumoconiosis, the medical term for lung scarring from inhaled dust. Pneumoconiosis can also occur from inhaled asbestos (asbestosis), coal (coal workers' pneumonconiosis), beryllium (berylliosis), and other respirable dusts.
- There is no effective treatment for any pneumoconiosis, including silicosis

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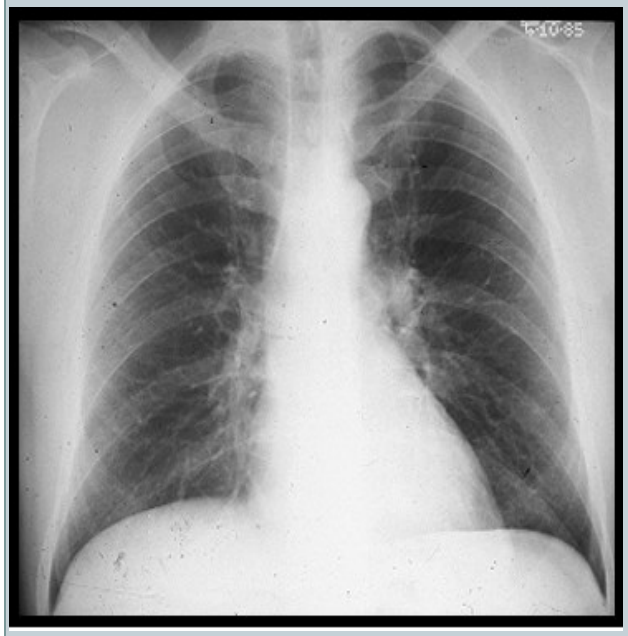
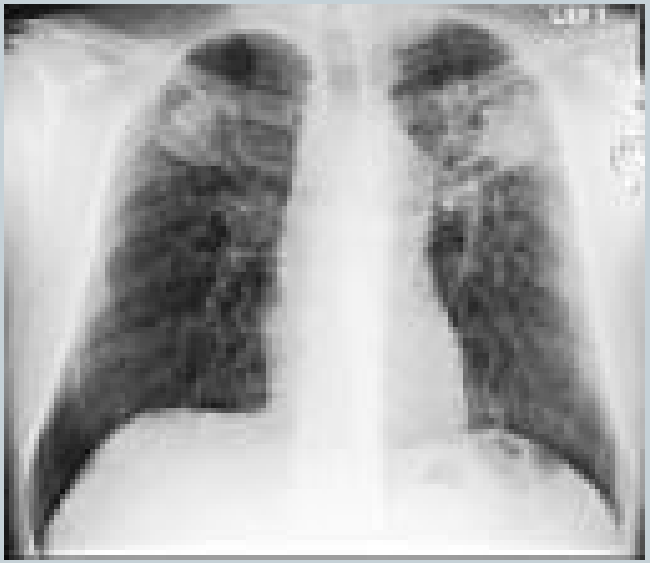
Chest x-rays – silicosis

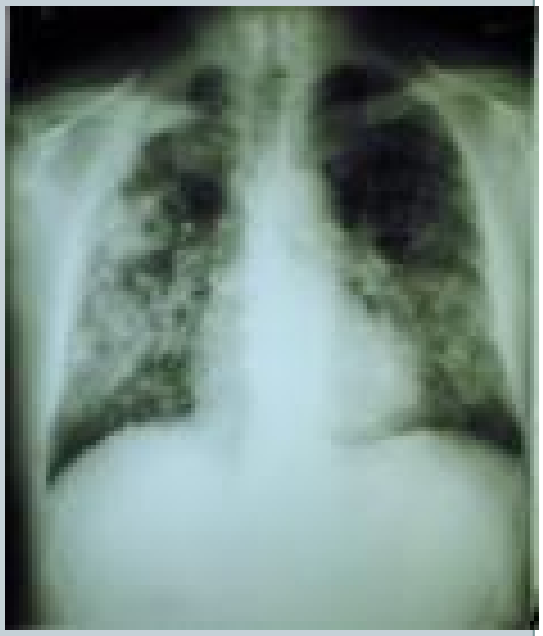
normal x-ray



silicosis (upper lobes)



silicosis -- diffuse



Diagnosis of Silicosis

- Abnormal chest X-ray (or chest CT scan) consistent with silicosis
- History of *significant* exposure to silica dust
- Medical evaluation to exclude other possible causes of abnormal chest x-ray
- *Pulmonary function tests are helpful to gauge severity of impairment, but NOT for diagnosis.*
- *Lung biopsy rarely indicated* (since no effective treatment, biopsy is done only when other diagnoses are being considered)

Silica Dust

- Silica is a common, naturally occurring crystal. Found in most rock beds, it forms a fine dust during mining, quarrying, and tunneling. Silica is a principal component of sand, so glass workers and sand-blasters can also receive heavy exposure.

Sand

- Beach sand, desert sand, golf bunker sand -- not harmful with ordinary exposure.
- Silicosis requires intense &/or prolonged exposure to very fine airborne sand particles.

Silica Dust Exposure – Risk Factors

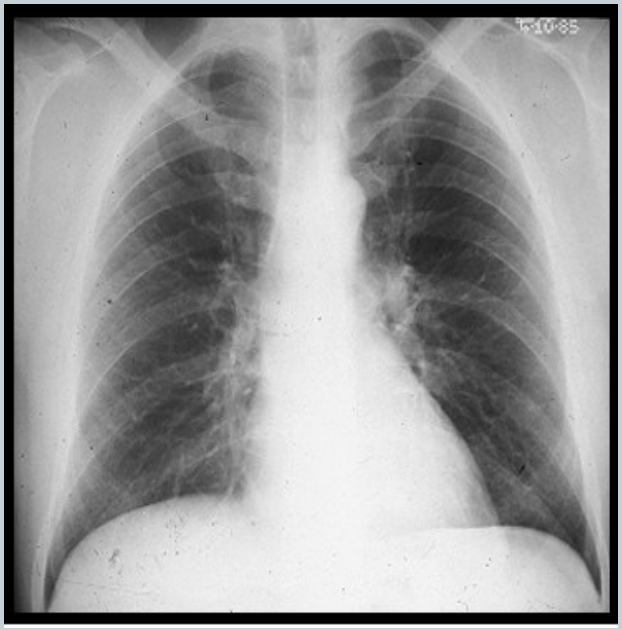
- Any work that exposes you to silica dust:
  - mining
  - stone cutting
  - quarrying
  - road and building construction
  - work with abrasives
  - glass manufacturing
  - sand blasting
  - Also, some *hobbies* can involve exposure to silica (sculptor, glass blower)

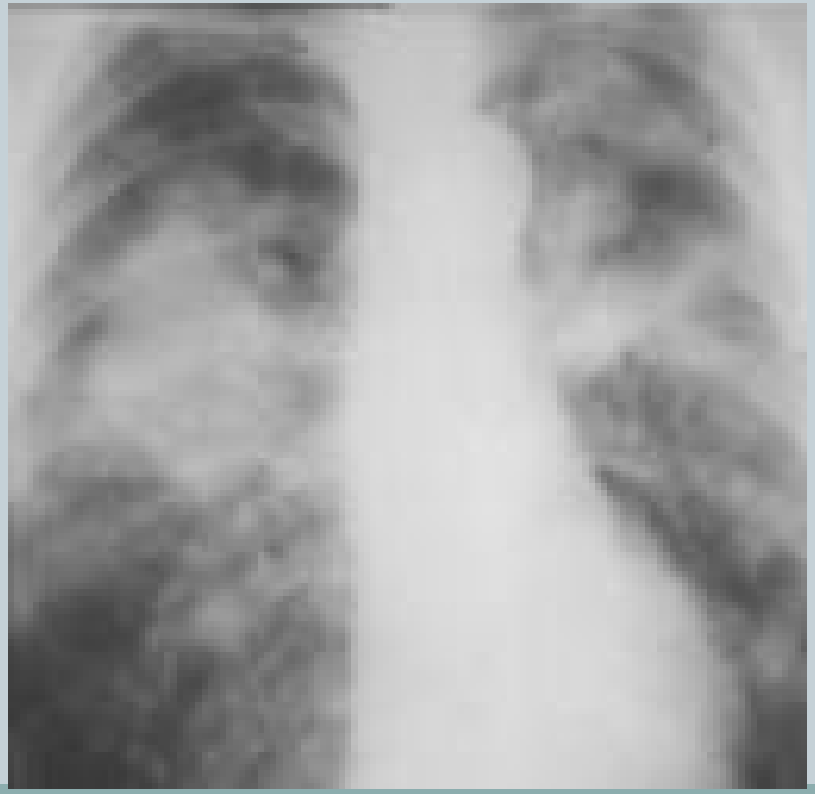
Silicosis - Coal Mining

Coal Worker's Pneumoconiosis

- CWP is indistinguishable from Silicosis

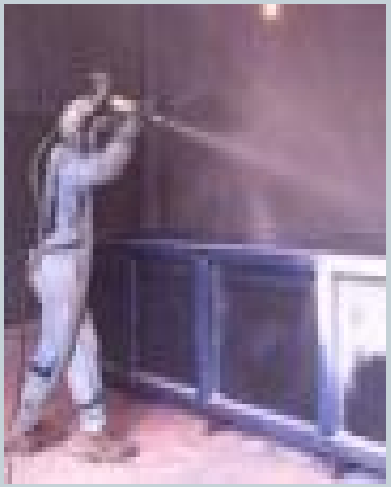
Normal chest x-ray





Silicosis - Sandblasting

- Compressed air at high pressure is used to blow *fine sand or other abrasive material* through a hardened spray nozzle. The abrasive particles quickly eat away whatever they are directed at, leaving a clean, matte surface.

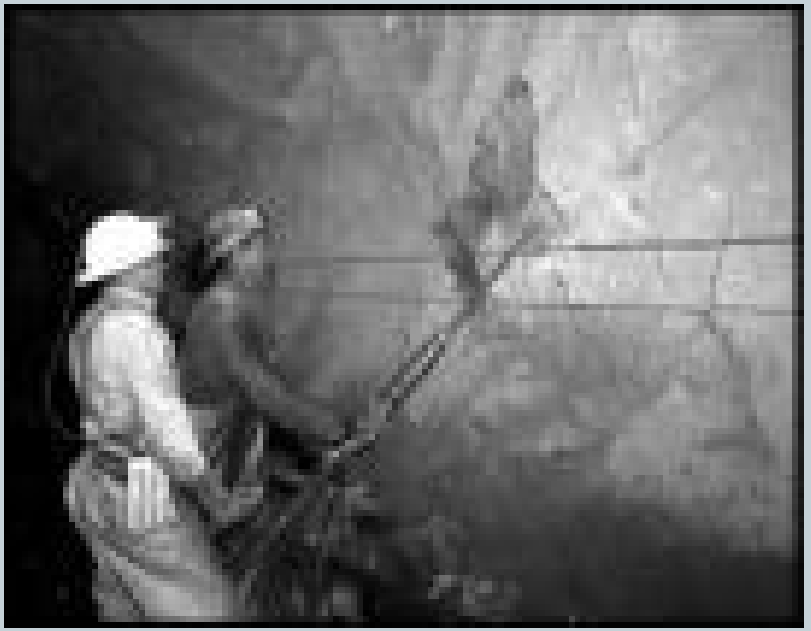


Silicosis – Foundry work

Silicosis - Stone cutting

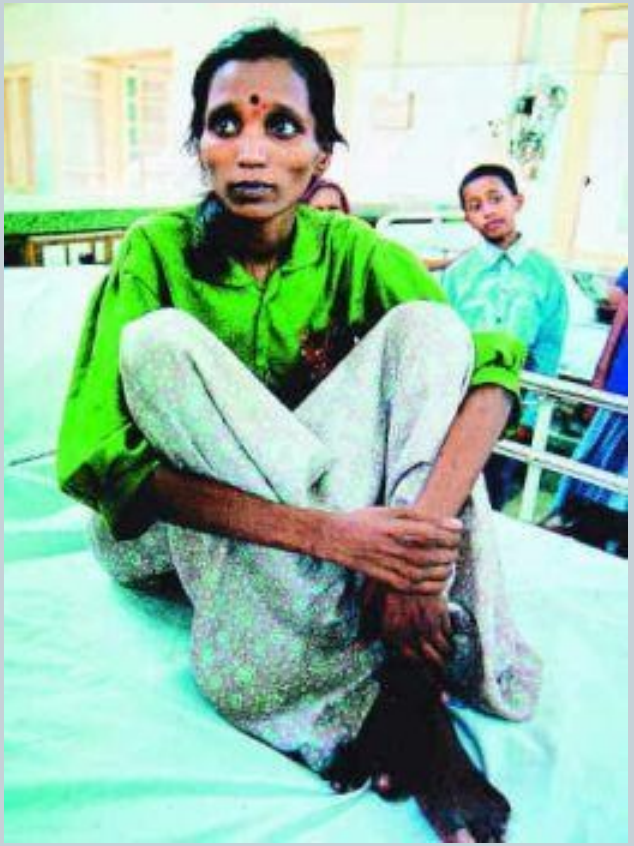
Silicosis - Tunnel construction

Worst single incidence of silicosis in U.S. –
Hawk's Nest Tunnel, Gauley Bridge, W. Va., 1930-1931

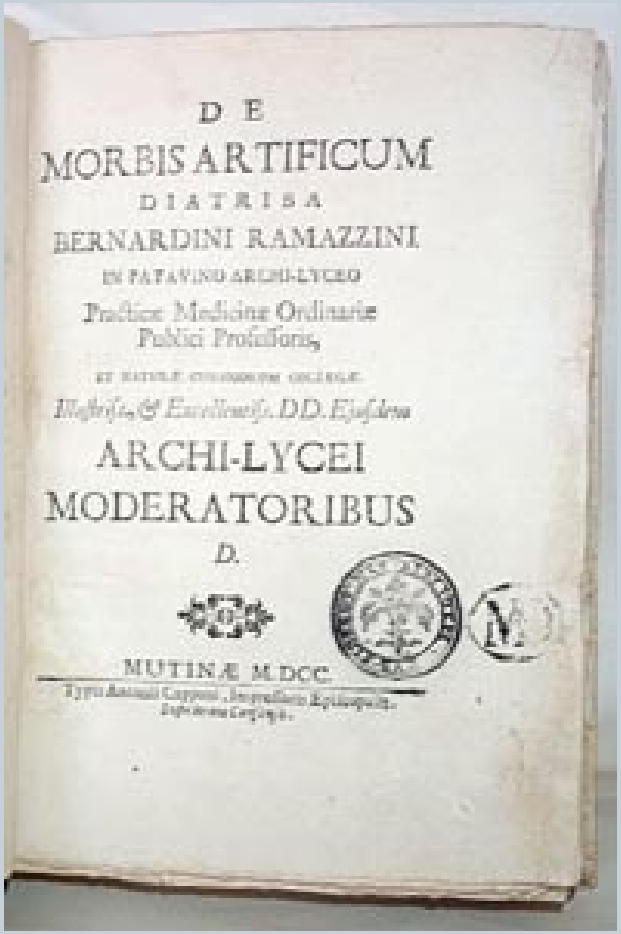


Silicosis – Glass Factory Workers

Sumathi, 19, admitted to Government Hospital, Pondicherry, India, suffers from severe silicosis. She worked in the sand plant (where silica is sieved) of a glass-container manufacturing plant.



Silicosis – history


DE
MORBIS ARTIFICUM
DIATRIBA
BERNARDINI RAMAZZINI
PATAVINO ARCHI-LYCEO
Practicæ Medicinæ Ordinariæ
Publici Professoris,
Illustris, & Excellentiss. DD. Ejusdem
ARCHI-LYCEI
MODERATORIBUS
D.
MUTINÆ M.DCC.

Full description by Bernardino Ramazzini (1633-1714) in early 18th century. "…when the bodies of such workers are dissected, they have been found to be stuffed with small stones." *Diseases of Workers (De Morbis Artificum Diatriba, 1713).*

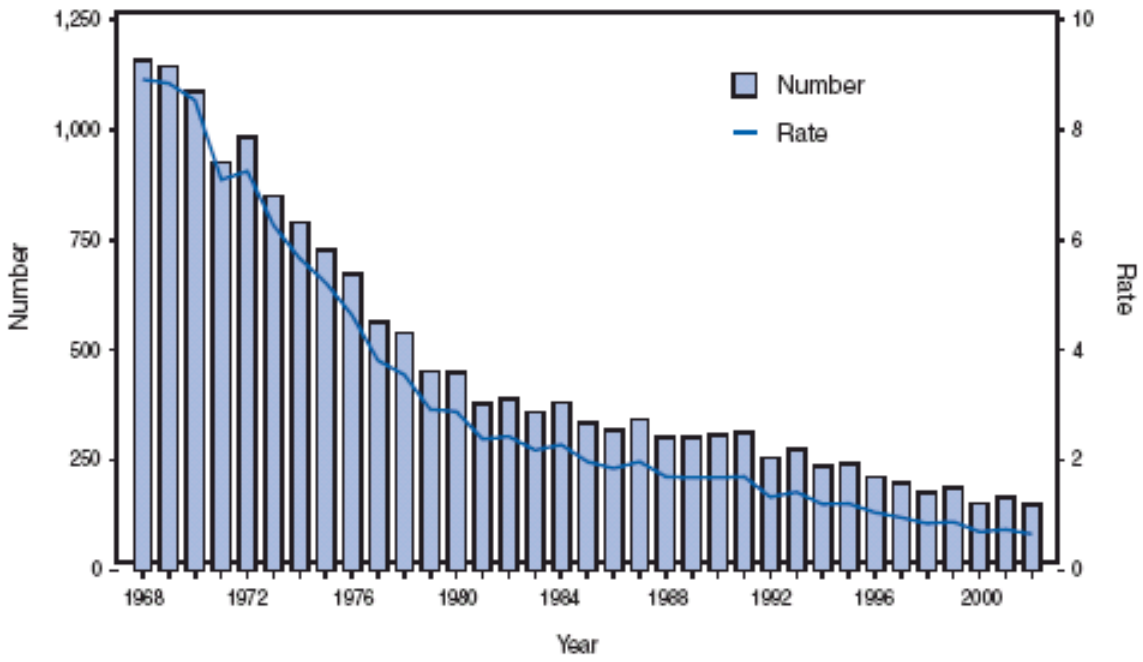
Silicosis - history

- First U.S. description in 19th century.
- Term silicosis introduced in 1870, from Latin *silex*, or flint.
- Prevalence increased markedly with introduction of mechanized mining.
- Came to national attention 1930-1931 with construction of Hawk's Nest Tunnel in Gauley Bridge, West Virginia. Called "the worst industrial accident in U.S. history." At least 764 tunnel workers died from silicosis. Hawk's Nest disaster led to Congressional hearings in 1936, and new laws protecting workers in many states.
- Prevalence of silicosis has greatly declined in recent decades because of effective industrial hygiene measures.

Silicosis deaths - declining

1,157 (1968) 148 (2002)

FIGURE 1. Number of silicosis deaths and age-adjusted mortality rate*, by year — National Occupational Respiratory Mortality System, United States, 1968–2002



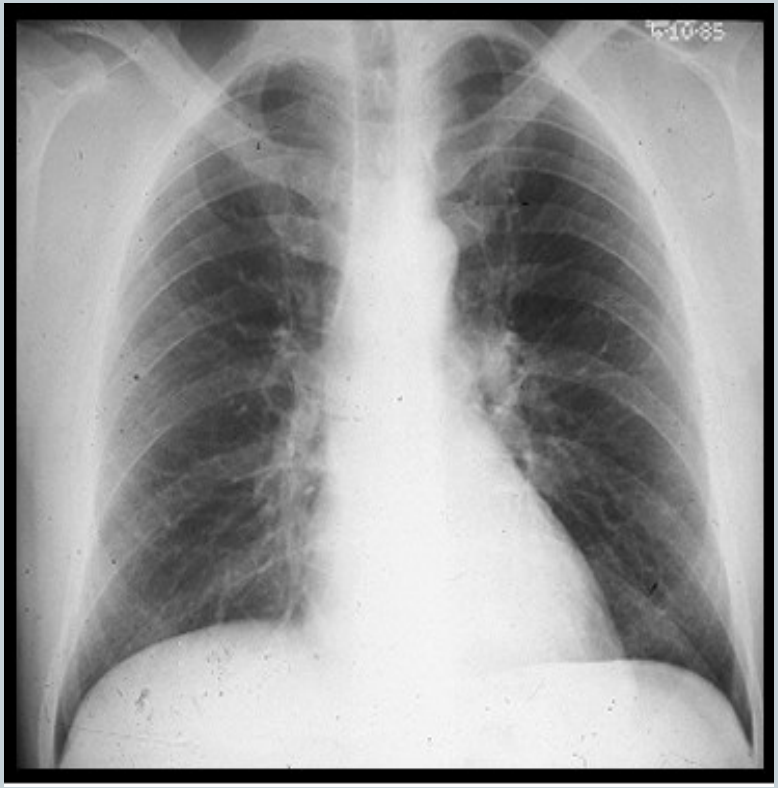
* Per million persons aged ≥15 years.

Three 'types' of silicosis

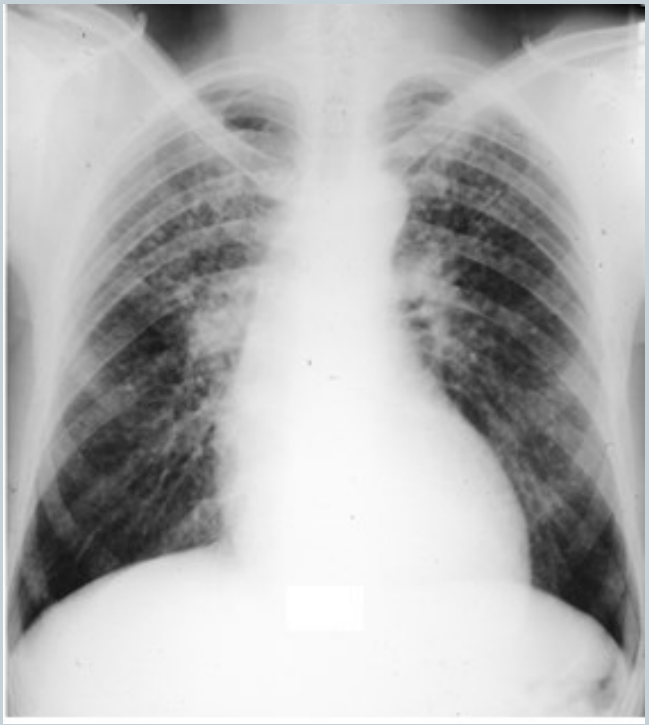
- **Simple chronic silicosis** From long-term exposure (10-20 years) to low amounts of silica dust. Nodules of chronic inflammation and scarring, provoked by the silica dust, form in the lungs and chest lymph nodes. Patients often asymptomatic, seen for other reasons.
- **Accelerated silicosis** (= PMF, progressive massive fibrosis) Occurs after exposure to larger amounts of silica over a shorter period of time (5-10 years). Inflammation, scarring, and symptoms progress faster in accelerated silicosis than in simple silicosis. Patients have symptoms, especially shortness of breath.
- **Acute silicosis** From short-term exposure to very large amounts of silica dust. The lungs become very inflamed, causing severe shortness of breath and low blood oxygen level. Killed hundreds of workers during Hawk's Nest Tunnel construction early 1930s.

Simple Silicosis

normal chest x-ray

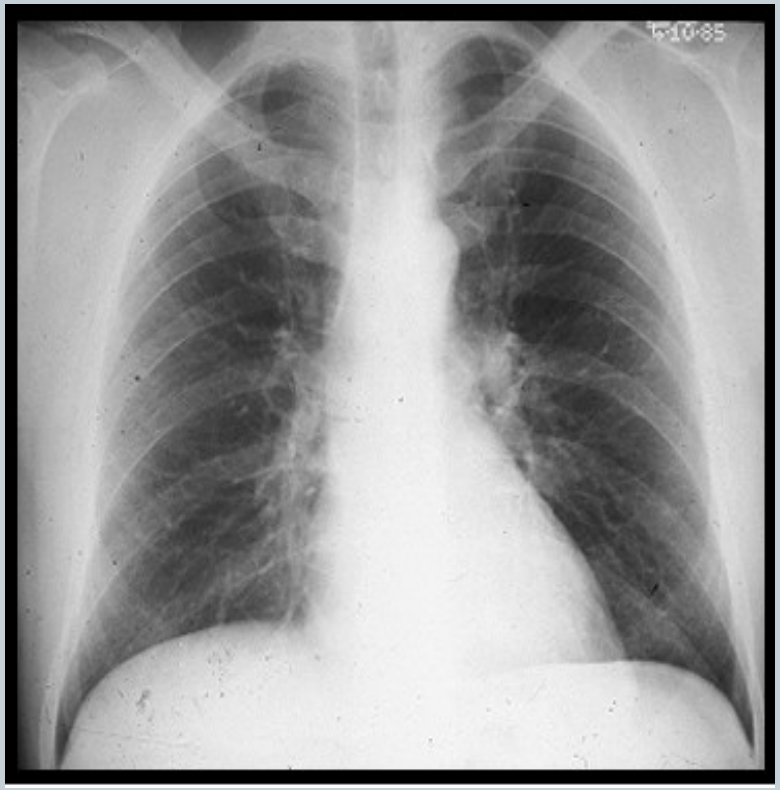


simple silicosis

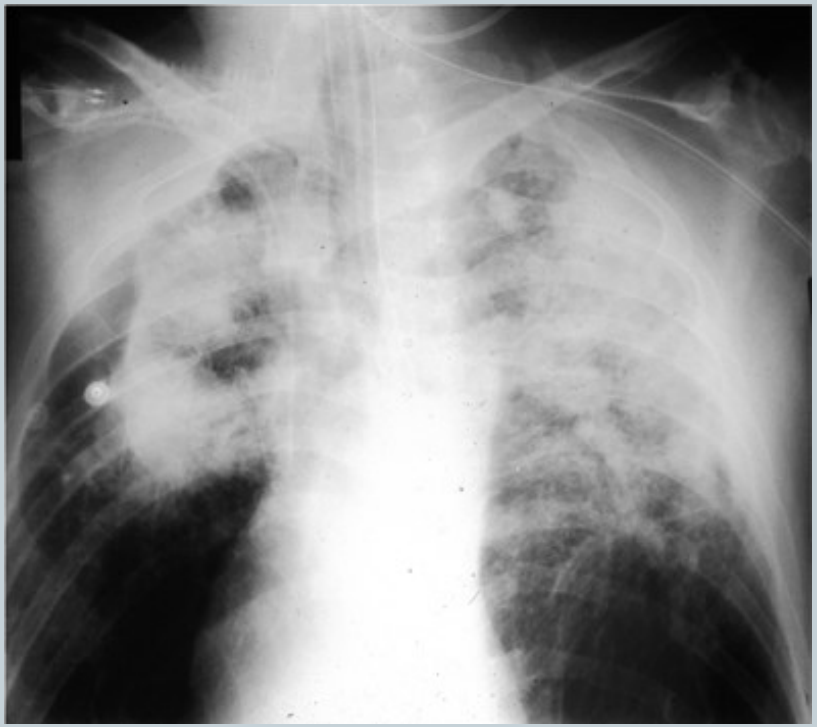


Accelerated Silicosis (= Progressive Massive Fibrosis)

normal chest x-ray

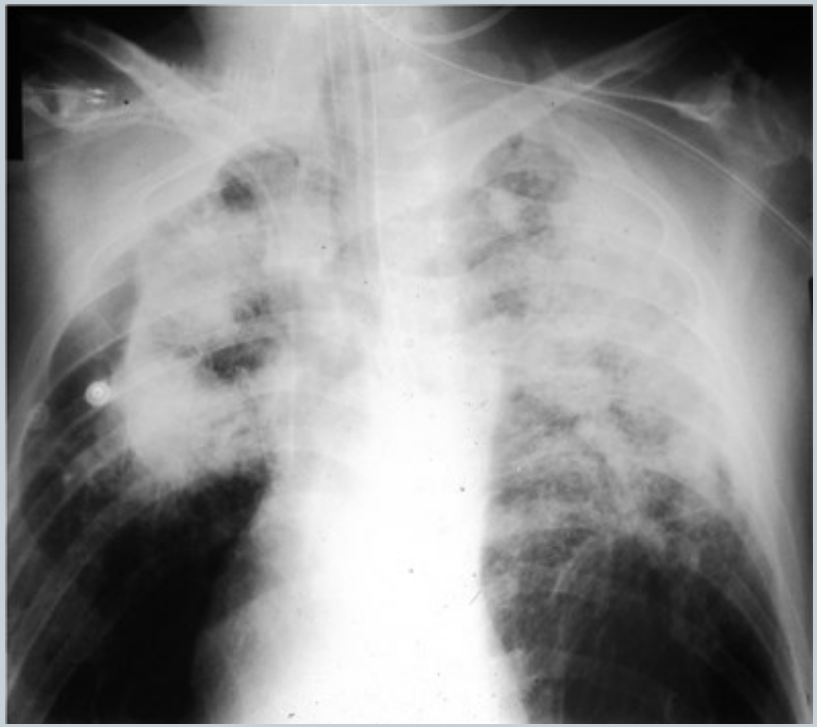


PMF

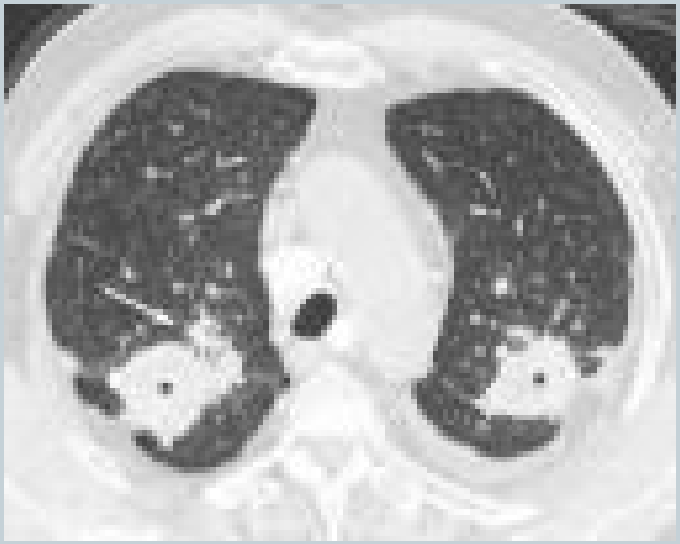


Accelerated Silicosis (PMF)

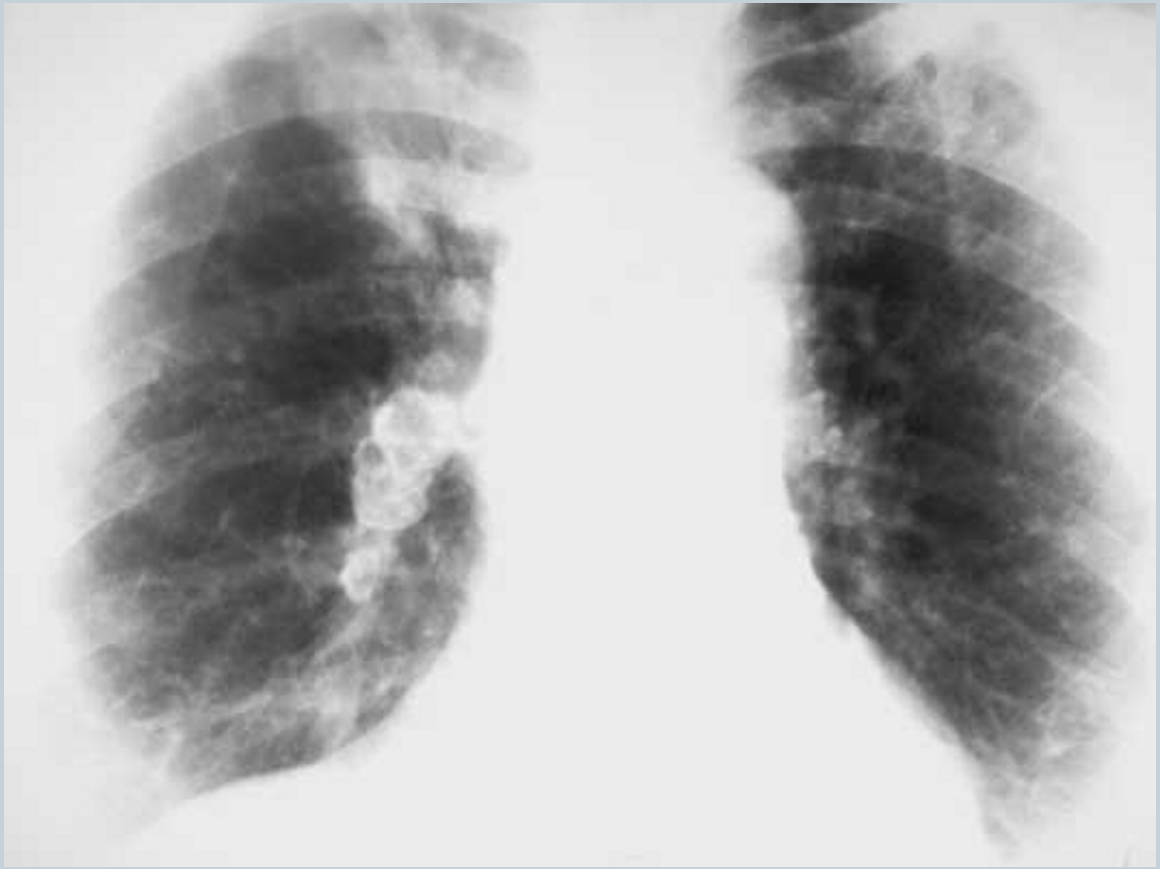
chest x-ray



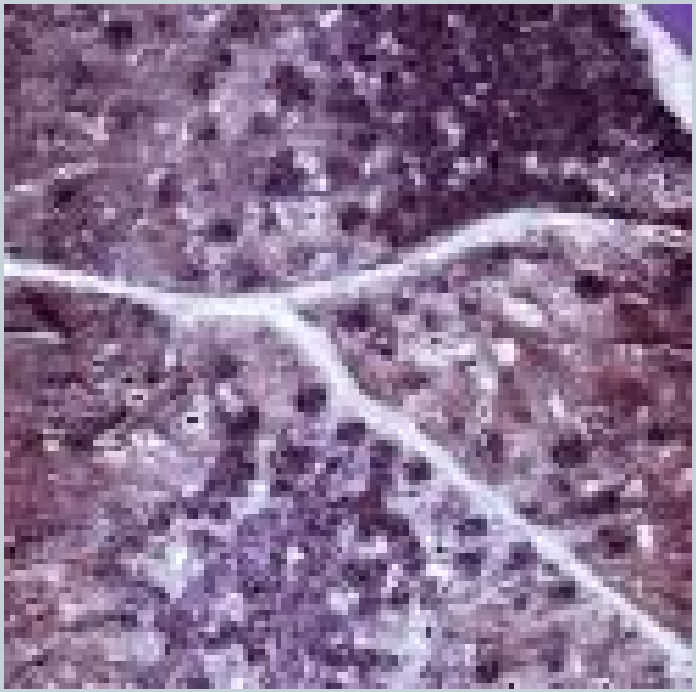
CT scan



Eggshell calcification – almost exclusively silicosis



Lung pathology – autopsy specimens



Silicosis – associated risks

- Having silicosis increases risk of contracting tuberculosis & lung cancer.
- Degree of increased risk is highly variable; depends on several OTHER factors, including immune system & exposure history (for TB), and amount of lung scarring, age & smoking history (for cancer).
- Silicosis also strongly associated with scleroderma and rheumatoid arthritis.
- Other associations less well established: lupus, systemic vasculitis, end-stage kidney disease.

Diagnosis of silicosis - summary

- Abnormal chest X-ray or chest CT scan
- History of *significant* exposure to silica dust
- Medical evaluation to rule out other causes of abnormal x-ray
- Pulmonary function tests
- Lung biopsy rarely used

Silicosis can be mis-diagnosed as something else

- Silicosis can mimic:
  - Sarcoidosis (benign inflammation of unknown cause)
  - Idiopathic pulmonary fibrosis (lung scarring of unknown cause)
  - Lung cancer
  - Several other lung conditions (chronic infection, collagen-vascular disease, etc.)

Can usually make right diagnosis with detailed history (occupational & medical) or, rarely, a lung biopsy.

Silicosis first diagnosed as Sarcoidosis

- **March 2000 – 32 yo male presented with cough and abnormal chest x-ray. Bronchoscopic lung biopsy read as "suggestive of sarcoidosis in proper clinical setting." At the time he was meter reader for local utility. Had prior history of foundry work, but no workers' comp claim filed (sarcoidosis is not occupational illness). He was treated with prednisone for cough and progressive shortness of breath.**
- **Seen by new lung specialist Feb 2005. Found to have worsening chest x-ray; also ?eggshell calcification. History noted of foundry work 1987-1993, with intense exposure to silica ("sand blaster"). Occupational history strongly suggested silicosis, *not* sarcoidosis.**
- **New chest CT scan ordered. It confirmed eggshell calcification and other abnormalities much more consistent with silicosis.**
- **Presumptive diagnosis changed to silicosis, and workers' comp claim filed. BWC accepted new diagnosis.**
- **He continues to work, albeit with severe pulmonary impairment.**

Who should make the diagnosis of silicosis?

- Treating doctors? *Yes, in some cases, but not practical for disease screening*
- Plaintiff-attorney-hired physicians? *Never, considering the asbestos and silicosis-MDL experience*
- Objective physicians not beholden to either plaintiff or defense interests? *Yes, especially for disease screening*

Multidistrict Litigation (MDL) Decision Criticizes Thousands of Silicosis Claims

- **Despite the marked decline in silicosis, in recent years plaintiff attorneys have filed thousands of claims for this disease. In order to ascertain the validity of the diagnoses, they were consolidated in a single Texas federal court, under U.S. District Judge Janis Jack. In June 2005, Judge Jack issued a 249-page decision, stating ". . . that truth matters in a courtroom no less than in a doctor's office."**

- **Judge Jack found that the vast majority of approximately 10,000 silicosis claims consolidated in multidistrict litigation "were essentially manufactured on an assembly line" run by plaintiffs' lawyers, screening companies and doctors.**

- **Her decision sharply criticized the plaintiffs' diagnoses, granted a motion for sanctions against a plaintiff law firm and concluded that most of the MDL cases should be remanded to state court for further proceedings.**

Multidistrict Litigation (MDL) Decision Criticizes Thousands of Silicosis Claims (cont.)

- Of >8000 cases of silicosis manufactured by MDL plaintiff attorneys whose medical records were examined, not a SINGLE one was ever diagnosed by the claimants' own treating physicians.

- Judge Jack's conclusion that the **MDL** cases "were driven by neither health nor justice" but instead **"were manufactured for money**" has become a watershed moment not only in the silica litigation nationwide, but is likely to affect other areas of mass tort litigation based upon a similar model.

Dallas Ft Worth Star-Telegram, February 17, 2005

"Judge calls diagnoses methods frightening"

++++++++++++++++++++

Mobile Register, March 13, 2005

Doctor's testimony ignites legal storm

Fortune, June 13, 2005

Diagnosing for Dollars

****

Client Alert, July 14, 2005

2005 Multidistrict Litigation Order Criticizes Thousands of Silicosis Claims

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Wall Street Journal, August 12, 2005

Silicosis Scandal

****

New York Times, October 9, 2005

The Tort Wars, at a Turning Point

Plaintiff-attorney-manufactured process for silicosis is *same as for asbestosis cases*

- "The significance of Judge Jack's order goes far beyond the silicosis cases…Given that asbestosis cases used the same techniques to recruit plaintiffs and used the same medical screeners, [I'm] confident that if the same level of discovery were permitted with respect to asbestosis claims, the same kind of evidence of fraud on a massive scale would be uncovered."

  -- Professor Lester Brickman, Cardozo Law School

Plaintiff attorneys may set up the manufacturing process, but bogus diagnoses still require the complicity of physicians

<u>In vast majority of asbestos claims, diagnoses are:</u>

- Medically unfounded; diagnostic methods violate sound medical principles (relevant medical history ignored or omitted; no differential diagnosis offered; serious diagnoses assumed from just a single chest x-ray)

<u>In some cases, diagnoses are:</u>

- Outright fraud (X-ray interpretations provided without looking at x-rays; pulmonary function data fudged)

Will silicosis be different?

Mass bogus diagnoses will always be possible as long as:

- There are physicians willing to make diagnoses for money.

AND

- The courts can't/won't distinguish legitimate diagnoses from manufactured ones.

AND

- Organized, academic medicine (AMA, ATS, ACCP) remains silent about these diagnostic scams.

Silicosis Screening

Without a fair and objective diagnostic process *from the beginning*:

- Abuses will occur.
- Physicians and their bogus diagnoses will continue to be bought.
- Organized/academic medicine will continue its hurtful silence.
- Plaintiff attorneys will shop venues until they find judge(s) that allow junk science into evidence.
- More companies will be bankrupted.
- Compensation for seriously-injured workers will be delayed.

Silicosis Screening

- It is unconscionable that workers with real occupational disease are delayed just compensation because bogus diagnoses -- manufactured simply to make money -- clog the system.

- This has happened repeatedly to asbestos-inured workers: delayed compensation because tens of thousands manufactured asbestos diagnoses have overwhelmed the courts.

To avoid repeating the asbestos diagnosis scam, absolutely essential that silicosis screening process be fair & objective

1) Screening process should be agreed to by both plaintiff and defense interests, and its methodology published in advance (listing all parties who will be involved). Any revisions should also be published as they occur.

2) Chest x-rays in any screening process must be interpreted in a BLINDED fashion, i.e., origin of x-rays must be unknown to interpreting radiologists.

To avoid repeating the asbestos diagnosis scam, absolutely essential that silicosis screening process be fair & objective (cont.)

3) Radiologists must NOT be paid by one side alone, but from a common fund, and irrespective of their findings.

4) The entire diagnostic process must be made transparent. For example, an audit of every physician's readings, positive and negative, should be made available at all legal proceedings involving that physician's reports.

Bogus diagnoses

- Fool me once, shame on you.
- Fool me twice, shame on me.

Don’t let it happen again!

Thank you