

OCCUPATIONAL HEALTH

11+12

Chemical Hazards



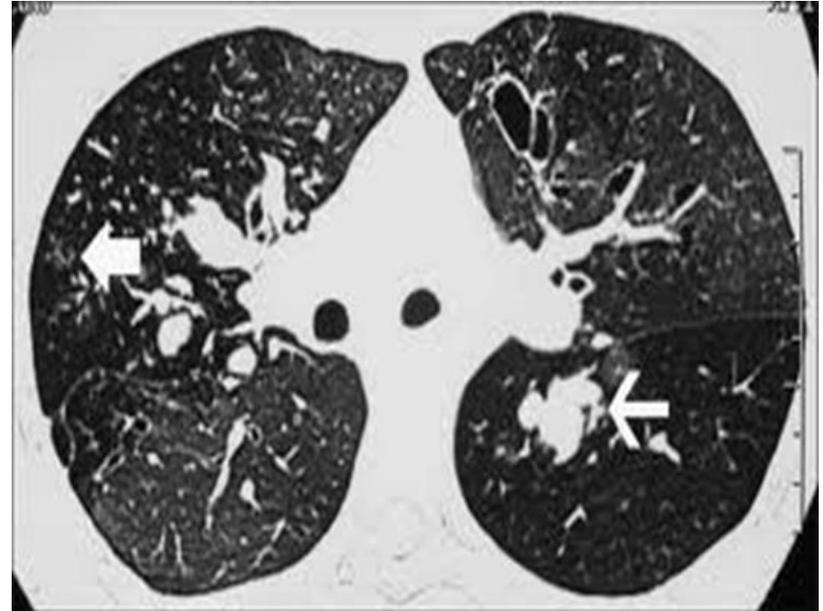
15TH MAY 2023

PROF. DR. WAQAR AL-KUBAISY

Pneumoconiosis

contents

- **Definitions**
- **Pathogenesis**
- **Types**
- **Individual diseases**
 - **Silicosis**
 - **Asbestosis**
 - **Anthracosis**
- **Preventive measures**

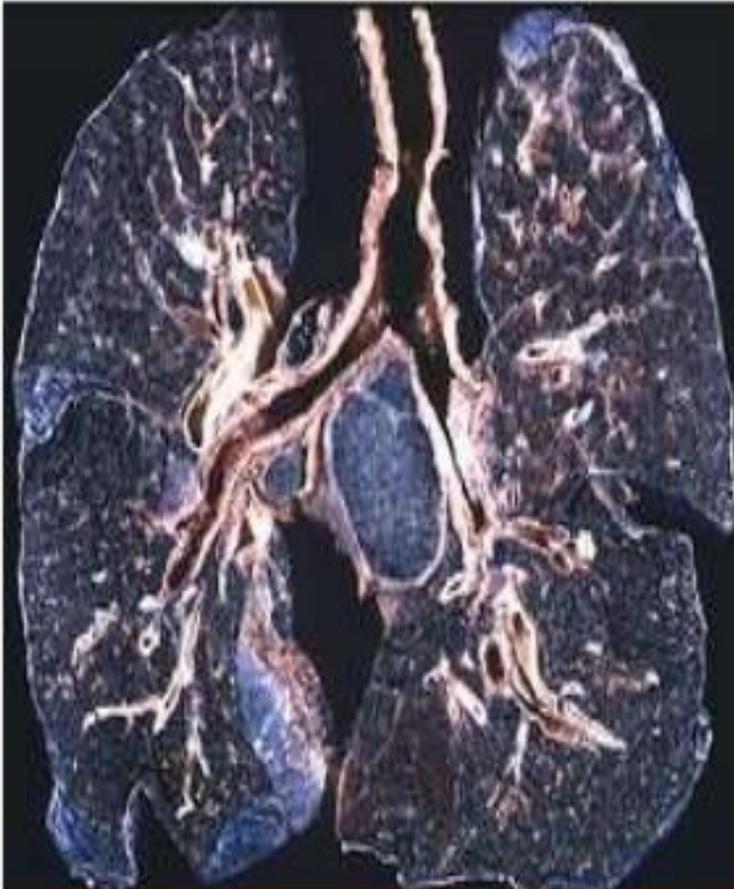


Anthracosis +Lead

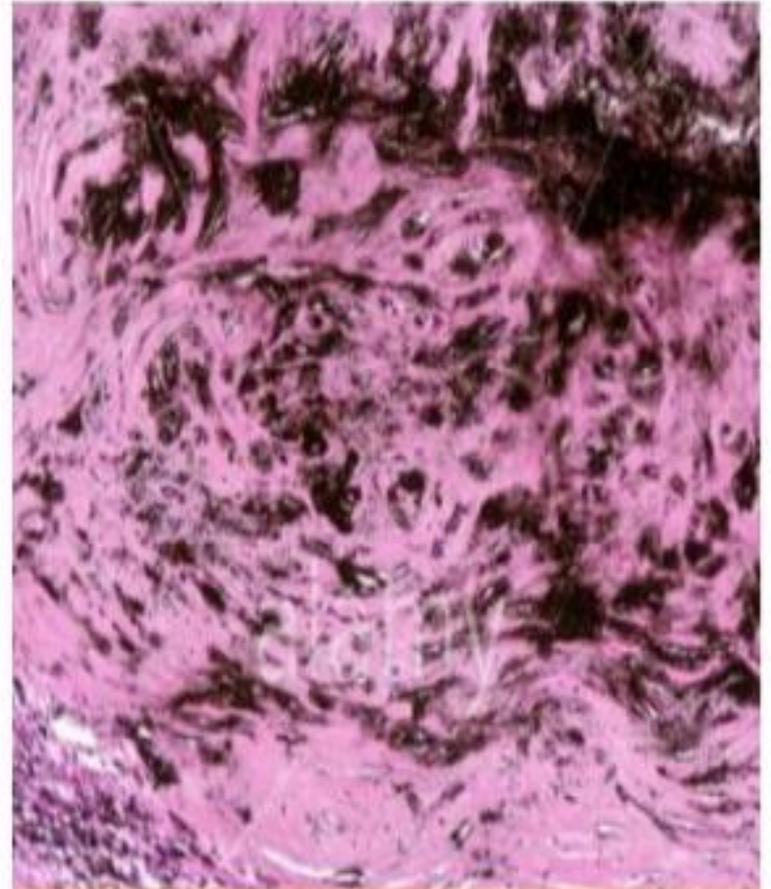
<https://www.amazon.com/Best-Sellers-Books-Biostatistics/zgbs/books/227277>

Cut section of lungs in anthracosis On histopathological examination

Anthracosis



Cut section of lungs in anthracosis



On histopathological examination

Anthracosis

Anthracosis

Coal Worker's Pneumoconiosis (CWP) / Black lung disease:

- **Accumulation of coal dust** in the lungs and the tissue's **reaction** to its presence.
- **Associated with coal mining industry**
- Takes **one or two decades** to cause symptoms
- **The disease is divided into 2 categories:**
 - I. **Simple CWP** and
 - II. **Complicated CWP** or **Progressive Massive Fibrosis (PMF)**.

I. Simple Coal Worker's Pneumoconiosis:

- the presence of **radiological opacities < 1cm** in diameter.
- **Benign disease** if no complications.
- **is associated with little respiratory impairment.**
- This phase may require **about 12 years** of **work exposure** for its development

❖ **Common symptoms:**

- cough,
- expectoration (black in colour) and
- dyspnea.
- – Slight decrease in FVC and FEV1/FVC??

❖ Once a background of simple pneumoconiosis has been attained in the coal worker, a **progressive massive fibrosis** may develop out of it without further exposure to it.

❖ From the epidemiology point of view

❖ the **risk of death** among coal miners has been nearly **twice** that of the general population .

II. Complicated Coal Worker's Pneumoconiosis or Progressive Massive Fibrosis (PMF).

- ❖ Is diagnosed when **large opacity** of **1cm or more** in diameter is observed in the CXR
 - ❑ Pathologically it is characterized by **large masses of black colour fibrous tissue.**
 - ❑ Symptoms are similar but **more severe**
- ❖ The large lesions **may cavitate** as a result of **ischemic necrosis or infection (T.B).**
- ❖ **Recurrent pulmonary infection**
- ❖ PFT (Pulmonary function test) reveals **decreased FVC, FEV1/FVC**

Cont. ...Complicated CWP

- The Second phase is characterised by
 - ❖ *progressive massive fibrosis* (PMF) this causes
 - ❖ severe respiratory **disability** and frequently results in
 - ❖ **premature death**

- Special type of PMF associated with **rheumatoid disease** (rheumatoid pneumoconiosis or **Caplan syndrome**) occur and is characterized by
 - typically smooth rounded nodule **1-5 cm in** diameter with concentric internal lamination and relatively little coal dust compared with other PMF lesions.
 - ❖ **Pulmonary function changes:** obstructive or mixed lesion.
 - decrease in FEV 1 and FEV1/FVC ratio

X-ray picture:

❖ Simple CWP

- frequently **mixed nodular and irregular** and
- **occasionally exclusively irregular opacities** was noted
- first in **upper and middle lung zone**.
- **irregular opacities** raises the possibility of previous exposure to asbestos.

❖ PMF appear radiologically as

- **nodular opacity 1cm or larger**
- usually **found posteriorly in upper lung zone**.

▪ D.D of small opacities in x-ray picture includes:

Miliary T.B and viral pneumonia

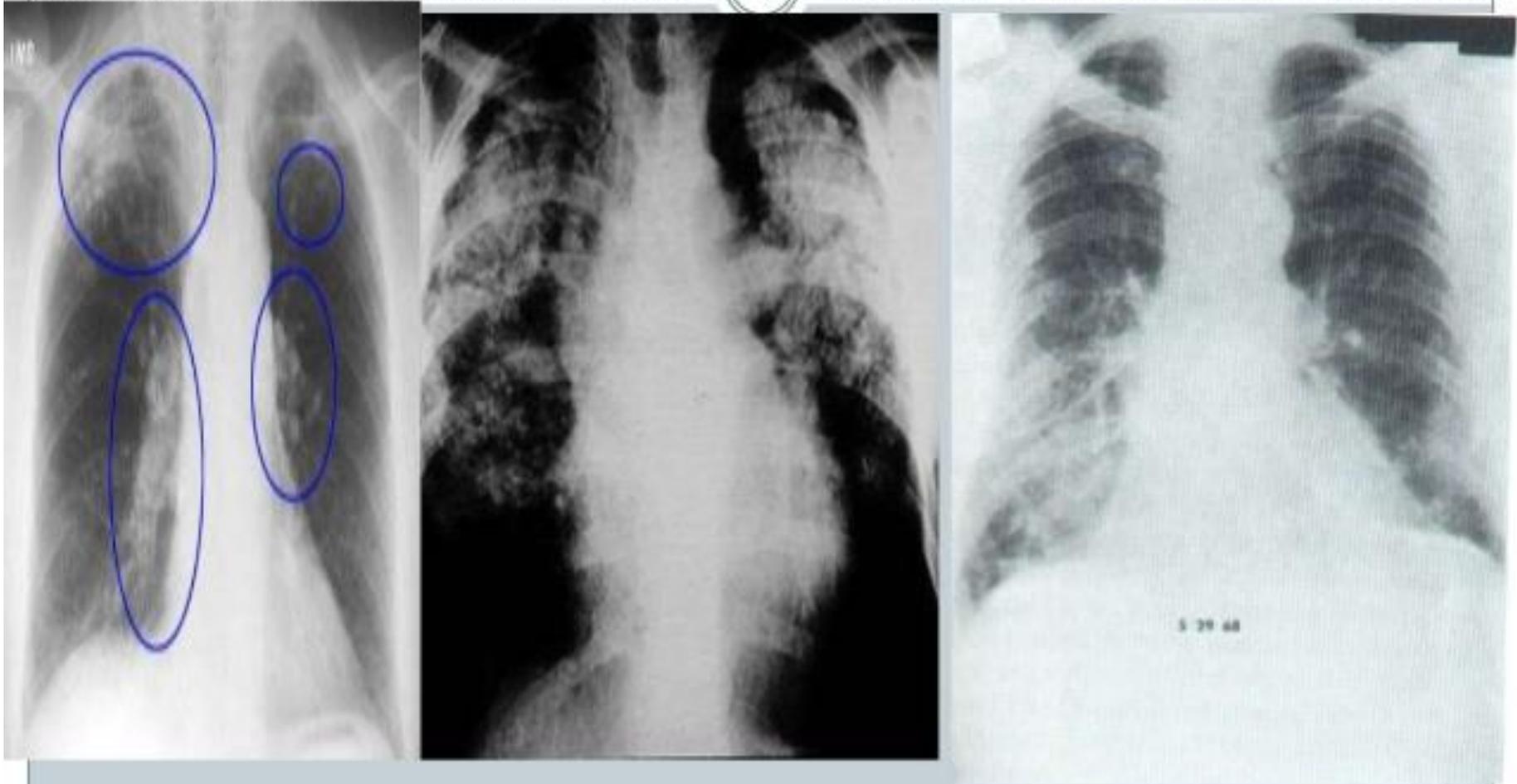
Other pneumoconiosis, metastatic carcinoma, chronic T.B

❖ PMF should be differentiated from malignancy,

Silicosis, Anthracosis & asbestosis

X-ray findings

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❖ Diagnosis and clinical assessment:

- History of present and past exposure .
- Look for previous chest X- ray and lung function tests.
- S. & S. including cough, sputum, dyspnea or cardiovascular symptoms

❖ Treatment and clinical care:

Symptomatic, for dyspnea , ch. bronchitis and congestive H.F

Caplan's syndrome (Caplan disease or (Rheumatoid pneumoconiosis)

is a **combination** of **rheumatoid arthritis (RA)** & **pneumoconiosis** manifests as intrapulmonary nodules,

which appear **homogenous** and **well-defined** on chest X ray

The nodules in the lung typically occur **bilaterally** and **peripherally**, on *a background of simple coal worker's pneumoconiosis*

There are usually **multiple nodules**, varying in size from **0.5 to 5.0 cm**.

The nodules typically **appear rapidly**, often in only a few weeks.

Nodules may grow, remain unchanged in size, **resolve**, or **disappear** and then **reappear**.

They **can cavitate**, calcify, or **develop air-fluid levels**

❖ **Caplan syndrome** occurs only in patients with **both RA and pneumoconiosis** related to mining dust (coal, asbestos, silica).

❖ . There is probably also a **genetic predisposition**, and

❖ smoking is thought to be an aggravating factor

Comparative features of different types

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Features	Silicosis	Asbestosis	Anthracosis
1. Agent/ dust	<ul style="list-style-type: none">•Silica free or silicon dioxide or silicic acid•Particles are 0.5 - 3 μ are most dangerous.	<ul style="list-style-type: none">•Asbestos fibres<ol style="list-style-type: none">1. Serpentine or chrysotile (safer)2. Amphibole<ol style="list-style-type: none">i. Crocidolite (blue)ii. Amosite (brown, safer)iii. Anthrophyllite (white)• 20-500μ in length and 0.5-50 μ in diameter	<ul style="list-style-type: none">• Coal dust

Comparative features of different types

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Features	Silicosis	Asbestosis	Anthracosis
Occupational exposure	Mining, pottery, ceramic, sand blasting, metal grinding, building & construction work, rock mining, iron & steel industry.	Manufacturers of Asbestos cement, fire proof textiles, roof tiling, brake lining & gaskets.	Coal miners, coal processors & coal handlers and those manufacturing carbon electrodes.
Incubation period	6 months to 6 years		12 years

Comparative features of different types

Features	Silicosis	Asbestosis	Anthracosis
Pathogenesis	<p>Fibrosis is initiated by silicic acid leading to nodular fibrosis, emphysema, and right heart failure. Pulmonary tuberculosis may intervene in 50% of cases. Fibrosis is nodular and in upper part of lung.</p>	<p>Asbestos fibers initiate fibrosis of pulmonary tissue, emphysema and its associated complications. Fibrosis is due to mechanical irritation, it is peri-bronchial, diffuse and basal in location</p>	<p>•Coal dust initiates diffuse and massive fibrosis</p> <ol style="list-style-type: none"> Simple pneumoconiosis with ventilatory impairment. Progressive massive fibrosis leading to emphysema and right heart failure
Clinico-Pathologic features	<p>Irritant cough, dyspnea on exertion & pain in chest. Dense nodular fibrosis 3-4 mm nodules. X-ray shows “snow-storm” appearance</p>	<p>Dyspnea out of proportion, clubbing, cyanosis, cardiac distress. Sputum shows “asbestos bodies”. X-ray shows ground glass appearance.</p>	<p>•From little ventilatory impairment to severe respiratory disability leading to pre-mat death.</p>

Control of pneumoconiosis

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- **Rigorous dust control measures**
 - × Substitution, enclosure, isolation, hydroblasting, good house keeping, personal protective measures
 - × Regular physical examination of workers.
- **Periodic examination of workers, biological monitoring (X-ray & Lung function)**
- **Personal protection**
 - × Masks, respirators with mechanical filters
- **Regulated exposure**
- **Health education**



**Thank
you**



Any questions?
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Chemical hazards

Occupational exposure to Toxic Metals

"heavy metals"

16th May 2023

LEAD POISONING

PROF DR. WAQAR AL – KUBAISY

Toxic metals,

- ✓ Toxic metals, including "heavy metals,"
- ✓ are **individual metals** and **metal compounds**
- ✓ that negatively affect people's health.
- ✓ very **small amounts** many of these metals, **are necessary to support life.**
- ✓ However, in larger amounts, they become **toxic.**
- ✓ They may **build up in biological systems** and become a **significant health hazard.**

Most hazardous:

Lead

Mercury

Arsenic

Cadmium

Beryllium

Hexa-valent

Chromium

Other toxic metals:

- **Aluminum**
- **Antimony**
- **Cobalt**
- **Copper**
- **Iron**
- **Manganese**
- **Molybdenum**
- **Nickel**
- **Selenium**
- **Silver**
- **Tin**
- **Vanadium**
- **Zinc**

LEAD POISONING

CONTENTS

- Source & Uses
- Body stores & Distribution
- Lead poisoning
- Clinical features
- Diagnosis
- Management
- Prevention



LEAD POISONING

Lead exposure:

- ❑ Lead over-exposure is one of the **most common overexposures** found in industry and
 - ❖ is a **leading cause of workplace illness**.
 - Therefore, **OSHA** (*The Occupational Safety and Health Administration*,
 - ❖ has **established the reduction of lead exposure** to be a **high strategic priority**.
 - ❖ OSHA's five year strategic plan a goal of
 - ❖ a **15% reduction** in the average severity of **lead exposure** or
 - ❖ **Employee blood lead levels** in selected industries & workplaces.
- ❑ **Lead poisoning** is also a **major potential public health risk**
 - ❑ In general populations,
 - ❖ Lead poisoning is **the leading environmentally induced illness**
 - ❖ in children.
 - **children under the age of six** are at greatest risk because
 - they are undergoing **rapid neurological and physical development**
 - ❑ lead may be **present in hazardous concentrations** in
 - ❖ **food, water, and air**.
 - ❖ **Sources** include paint, urban dust, and folk remedies.

- ❑ Lead dust or fumes are inhaled, or is ingested via contaminated hands, food, water, cigarettes or clothing
- ❖ Lead entering the respiratory and digestive systems is released to the blood and distributed throughout the body.
- ❖ More than 90% of the total body burden of lead is accumulated
- ❖ the bones, where it is stored.
- ❖ Lead in bones may be released into the blood,
- re-exposing organ systems long after the original exposure.

❑ Body Stores :

- ❖ Normal adults ingest about 0.2 to 0.3 mg of lead /day largely from food and beverages
- ❖ The body store of lead in the average adult population is
- about 150 to 400 mg and
- ✓ blood levels average about 25µg/100 ml.
- 70µg/100 ml blood is generally associated with clinical symptoms.

Mode Of Absorption :

❑ Lead poisoning (Plumbism) may occur in three ways:

(1) Inhalation:

- ❖ Most cases of industrial lead poisoning is due to inhalation of fumes and dust of lead or its compounds.

(2) Ingestion:

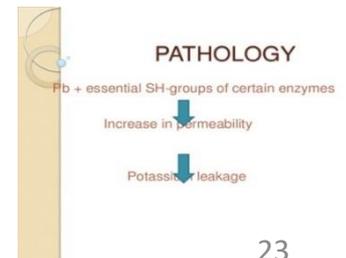
- ❖ Poisoning by ingestion is of less common occurrence.
- ❖ Small quantities of lead trapped in the upper respiratory tract may be ingested.
- ❖ Lead may also be ingested in food or drink through contaminated hands.

(3) Skin :

- ❖ Absorption through skin occurs only in respect of the organic compounds of lead, especially tetraethyl lead.
- Inorganic compounds are not absorbed through the skin

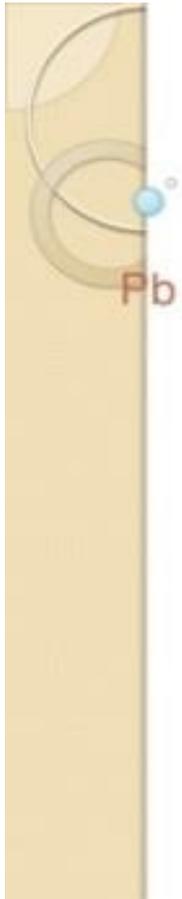
Distribution in the Body:

- ❑ 90% of the ingested lead is excreted in the faeces.
 - ❖ Lead absorbed from the gut enters the circulation, and
 - ❖ 95 % enters the erythrocytes.
 - ❖ It is then transported to the liver and kidneys and finally
 - ❖ transported to the bones where it is laid down with other minerals.
- ❑ Although bone lead is thought to be 'metabolically inactive',
 - ❖ it may be released to the soft tissues again under conditions of bone resorption.
 - ❖ Lead probably exerts its toxic action by combining with essential
 - ❖ SH-groups of certain enzymes, for example some of those
 - ❖ involved in prophyrin synthesis and carbohydrate metabolism.
- ❑ Lead has an effect on membrane permeability and
- ❑ potassium leakage has been demonstrated
- ❑ from erythrocytes exposed to lead



characteristic finding of lead poisoning, dense metaphyseal lines.





PATHOLOGY

Pb + essential SH-groups of certain enzymes



Increase in permeability



Potassium leakage

Lead absorption

Oral:

adults absorb 10%

children absorb 40-50%

increased absorption if low **Fe, Ca**

Inhalation (<1 μ m):

dust or lead fumes

absorb 50-70%

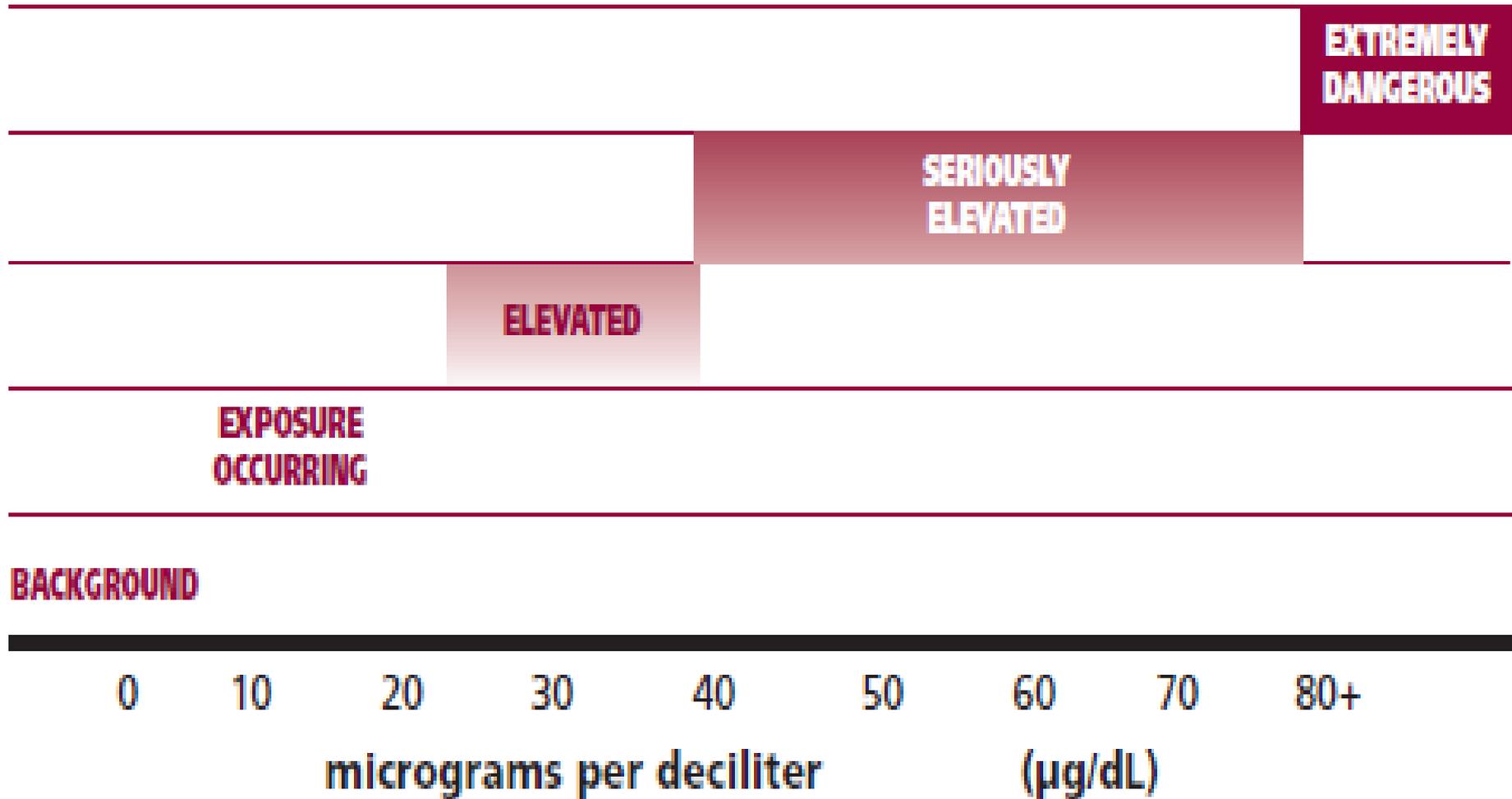
Skin:

little/no absorption

What Lead Levels Are Considered Elevated in Adults?

- At levels **above 80 μ g/dL**, serious permanent health damage may occur (**extremely dangerous**).
- **Between 40 and 80 μ g/dL**, serious health damage may be occurring, even if there are no symptoms (**seriously elevated**).
- **Between 25 and 40 μ g/dL**, regular exposure is occurring.
- There is some evidence of **potential physiologic problems (elevated)**.
- **Between 10 and 25 μ g/dL**, lead is **building up** in the body and some **exposure is occurring**.
- ❖ The typical level for U.S. adults is
- ❖ **less than 10 μ g/dL** (mean = 3 μ g/dL).

What Lead Levels Are Considered Elevated in Adults?



SOURCE & USES

- Lead(Pb) is a heavy metal

- **Occupational & Non-occupational sources**

- ❖ **Main source of environmental (non-occupational)source of Pb**

 - is Gasoline

 - Also through **drinking water** from **lead pipes**,

 - **chewing lead paints** on **toys** etc..

- ❖ More **industrial workers** are **exposed to lead** than to any other toxic metal.

- **Lead is used widely** in a variety of **industries**

 - because of **its properties**:

 - (1) low boiling point

 - (2) mixes with other metals easily to form alloys سبائك

 - (3) easily oxidised and

 - (4) anticorrosive.

LEAD POISONING(PLUMBISM)

All lead compounds are toxic

❖ MOST Dangerous

- lead arsenate,
- lead oxide and
- lead carbonate;

❖ the least toxic is lead sulphide

Industrial Uses :

Over 200 industries are counted where **lead is used**

- manufacture of storage batteries
- glass manufacture;
- ship building;
- printing and potteries;
- rubber industry and
- several others

Non-occupational Sources

- The greatest source of environmental (non-occupational) lead is **gasoline**.
- Thousands of tons of lead every year is exhausted from automobiles.
- Lead is one of the few trace metals *that is abundantly present in the environment*
- Lead exposure may also occur through **drinking water from lead pipes;**
- chewing lead paint on window sills or toys in the case of children.

Clinical Picture :

The clinical picture of lead poisoning or **plumbism** is different in the **inorganic and organic** lead exposures.

Clinical Features

Inorganic Pb exposure:-

- Abd. Colic
- Obstinate(rigid) constipation
- loss of appetite
- blue lines on gums
- stippling of red cells
- anaemia
- wrist drop foot drop

Organic Pb compounds:- (toxic **effect mainly on CNS**)

- Insomnia
- Headache
- Mental confusion
- Delirium etc..

Clinical picture

Acute lead poisoning

- ❖ (as short as days)
- loss of appetite,
- nausea,
- vomiting,
- stomach cramps,
- constipation,
- difficulty in sleeping,
- fatigue,
- moodiness,
- headache,
- joint or muscle aches,
- anemia, and
- decrease in sexuality.
- ❖ **Acute health poisoning** from uncontrolled occupational exposures has resulted in
- ❖ **fatalities**

- ❖ Long term (chronic):
- ❖ as long as several years result
- ❖ in **severe damage** to the
 - blood-forming,
 - nervous,
 - urinary, and
 - reproductive systems.
- ❖ The frequency and severity
- ❖ of clinical symptoms increases with the **concentration of lead in the blood**

Key lead-induced health effects.

Neurological Effects

- ◆ Peripheral neuropathy
- ◆ Fatigue/Irritability
- ◆ Impaired concentration
- ◆ Hearing loss
- ◆ Wrist/Foot drop
- ◆ Seizures
- ◆ Encephalopathy

Gastrointestinal Effects

- ◆ Nausea
- ◆ Dyspepsia
- ◆ Constipation
- ◆ Colic
- ◆ Lead line on gingival tissue

Reproductive Effects

- ◆ Miscarriages/Stillbirths
- ◆ Reduced sperm count & motility
- ◆ Abnormal sperm

Heme Synthesis

- ◆ Anemia
- ◆ Erythrocyte protoporphyrin elevation

Renal Effects

- ◆ Chronic nephropathy with proximal tubular damage
- ◆ Hypertension

Other

- ◆ Arthralgia
- ◆ Myalgia

Lead poisoning

Lead buildup in the body causes serious health problems

Symptoms

- Headaches
- Irritability
- Reduced sensations
- Aggressive behavior
- Difficulty sleeping

- Abdominal pain
- Poor appetite
- Constipation
- Anemia

Additional complications for children:

Lead is more harmful to children as it can affect developing nerves and brains

- ▶ Loss of developmental skills
- ▶ Behavior, attention problems
- ▶ Hearing loss
- ▶ Kidney damage
- ▶ Reduced IQ
- ▶ Slowed body growth

Source: MedlinePlus/Mayo Clinic

240809 AFP

DIAGNOSIS

Diagnosis of lead poisoning is based on :

- **History**
- **Clinical features** such as loss of appetite, intestinal colic, persistent headache, weakness, abdominal cramps and constipation, joint and muscular pains, blue line on gums, anaemia, etc.
- **Laboratory diagnosis**

a) **Coproporphyrin in urine(CPU)** Normal- $<150\mu\text{g/L}$

Measurement of CPU is a **useful screening test**.

In non-exposed persons, it is less than **150 microgram/litre**

b) **Amino levulinic acid in urine(ALAU)**

$>5\text{mg/L}$ it indicates clearly lead absorption.

c) **Pb in blood and urine:**

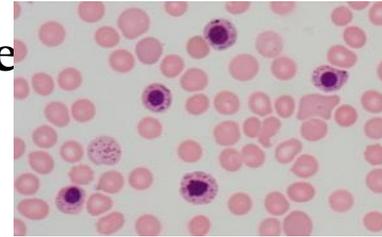
They provide **quantitative indicators** of exposure

In urine- $>0.8\text{mg/L}$ indicates **lead exposure** and **lead absorption**

In blood- $>70\mu\text{g}/100\text{ml}$ Pb **absorption** is associated with clinical symptoms

d) Basophilic stippling of RBC: Is a sensitive parameter of the haematological response.

Basophilic stippling, also known as **punctate basophilia**, is the presence of numerous **basophilic** granules that are dispersed through the **cytoplasm** of **erythrocytes** in a peripheral blood smear.



MANAGEMENT :

❖ The major objectives in management of lead poisoning are the

- i. **prevention** of further **absorption**,
- ii. **removal of lead** from **soft tissues** and
- iii. **prevention of recurrence**.

➤ Early recognition of cases will help in removing them from further exposure.

□ Treatment

- A saline purge will **remove unabsorbed** lead from the gut.
- Like Ca-EDTA, **it is a chelating** agent and works by **promoting lead excretion in urine**.
- Chelation therapy is an antidote for poisoning by mercury, arsenic, and lead.
- Chelating agents convert these metal ions into a chemically and biochemically inert form that can be excreted
- **Chelating agents** are used to reduce blood and tissue levels of **injurious** heavy metals. **Chelating agents** are generally **classified based upon the target heavy metal** – iron, copper, mercury and lead being the major targets

What are the Treatment Issues to Be Considered For Adults?

- ❑ When lead poisoning has been diagnosed, the **first course of action** is
- ❑ to **discontinue exposure**.

Whether discontinuation of exposure is **sufficient to treat** depends on:

- the blood lead **level**,
- **severity** of clinical symptoms,
- **biochemical** and hematologic **disturbances**,
- the nature and history of exposure

All of these factors must be considered in determining the necessity for chelating therapy.

❑ There is no exact blood lead concentration above which treatment with a chelating agent is always indicated.

❑ In most cases, however, when a blood lead level **rises to 80 µg/dL**, **chelation should be considered**, especially in the presence of more severe signs and symptoms

Treatment:

Chelating agents for lead poisoning:

1. EDTA - Sodium calcium edetate
2. DMSA – Dimercapto-succinic acid
3. BAL - Dimercaprol
4. Penicillamine - *no* longer recommended

EDTA -Sodium Calcium Edetate

IV for severe toxicity,
particularly encephalopathy

Well tolerated,

<1% nephrotoxicity

Therapeutic chelating agents have potentially **adverse side effects** and should be used cautiously and on an **individual basis**

Remember:

The exposure must first be discontinued before initiating chelation therapy.

A single course of chelation may **not sufficiently** reduce blood lead levels and **repeat courses** may be required among heavily exposed individuals.

DMSA - 2,3dimercaptosuccinic acid

- **Oral agent** of choice *for lead poisoning*
- Given as a 19 days course
- Well tolerated
- ❖ The main problem is foul taste and
- ❖ smell !!

PREVENTIVE MEASURES

The most effective way to **protect workers is to minimize their exposure through:**

- engineering controls,
- good work practices and training,
- the use of personal protective clothing and equipment, including **respirators**, where required.

Engineering controls include:

- 1) material substitution,
- 2) isolation,
- 3) process/equipment modification
- 4) local ventilation.



- a) **Substitution** Pb compounds should be substituted by **less toxic** materials
- b) **Isolation** All processes which give rise to harmful concentration of pb dust or fumes should **be enclosed and segregated**
- c) **Local exhaust ventilation** There should be **adequate local exhaust ventilation system** To remove dust & fumes



d) Personal protection By **approved respirators**

e) Good housekeeping is essential where lead dust is present.

Floors, benches, machines should be kept clean by **wet sweeping**

f) In working atmosphere **Pb conc.** Should be kept **<2mg per 10cu.m** of air which is usually the **permissible limit** or threshold value

g) **Periodic medical examination** of workers.

All workers must be given **periodical medical examination**.

Laboratory determination of

- urinary lead,
- blood lead,
- red cell count, haemoglobin estimation and
- coproporphyrin test of urine should be done periodically.
- Estimation of basophilic stippling may also be done

h) **Personal hygiene** (Hand washing) before eating is an important measure of personal hygiene.

There should be **adequate washing facilities** in industry.

Prohibition on **taking food** in work places is essential

i) **Health education Workers** should be educated on the risks involved and personal protection measures

WHO states that in the case of exposure to lead, it is **not only** the average **level of lead in the blood** that is important, but also the **number of subjects** whose blood level exceeds a certain value. e.g., **70µg/ml** or whose **ALA in the urine exceeds 10 mg/litre**)

How to reduce exposure?



- **Wash hands** and face before eating, drinking or smoking.
- **Eat, drink and smoke** only **in areas free of lead** dust and fumes.
- Store food and tobacco in clean areas.
- Wear a clean, **properly fitted respirator** in all areas that have lead dust or fumes.
- **Change into** different **clothes and shoes** before engaging in work with lead.
- **Keep street clothes** and shoes in a clean place.
- **Shower after working** with lead before going home.
- **Launder clothes** separately from other family members' clothes

I. Guidelines for the Control of Lead in the Workplace

- ❑ First, **test each** worker **before they begin** any work involving lead
- ❑ Then **test** that worker **every month**:
 - For the **first 3 months** of testing, and
 - **Whenever the previous blood lead level was greater than 25 µg/dL**

 - (If the previous blood lead level was at least **50 µg/dL**,
 - a **follow-up test within 2 weeks** and **medical removal is required**), **or**
 - **Whenever an increase of at least 10 µg/dL** from the previous test is observed

II. Voluntary Guidelines for the Control of Lead in the Workplace

☐ After the **first three months**, continue testing **every 2 months**:

➤ When the blood lead levels have **remained below 25 $\mu\text{g}/\text{dL}$**
for 3 months, and

➤ If an **increase less than 10 $\mu\text{g}/\text{dL}$** from the previous test is observed

☐ **Test every 6 months**:

➤ When the blood **lead levels remain below 25 $\mu\text{g}/\text{dL}$ for 6 mths**, &

➤ If **an increase less than 10 $\mu\text{g}/\text{dL}$ from the** previous test is observed

☐ **Results of each test should be provided to the worker.**

☐ **Tracking the test results** can help the employer and the worker
identify whether blood lead levels are

➤ dropping,

➤ remaining stable or

➤ increasing.

➤ The **employer should** also **review the test results for all workers to help identify jobs where problems may be occurring**

Thank you for attention

