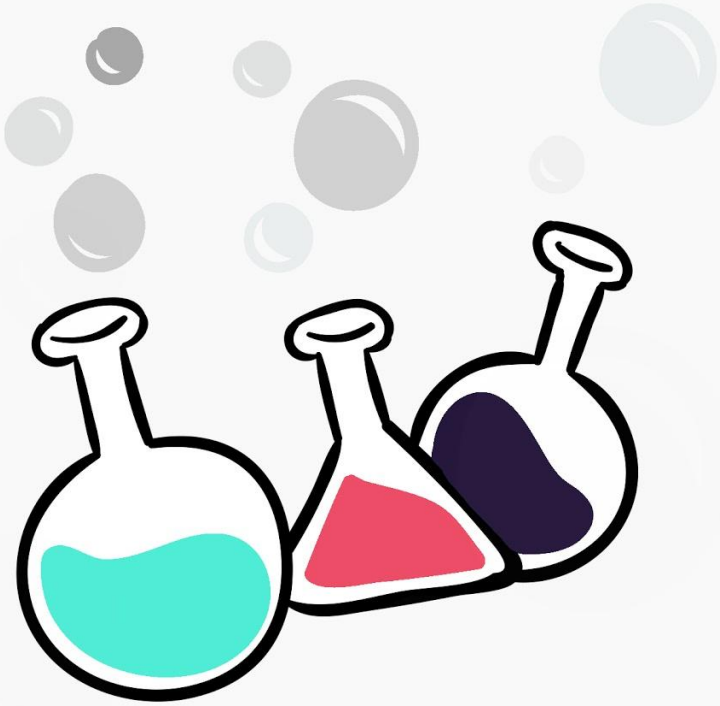


Heavy metals

Objectives:

- ✓ Not given.

Done by:

- ✓ Shahad Alenezi

Revised by:

- ✓ Abdullah Aljunaydil and Ahmed Al Yahya

[Important | Notes | Extra | Not mentioned by the doctor]

nake sure you read the take home massage

:Iron (Fe)

- Essential for the function of hemoglobin, myoglobin, many cytochromes, and many catalytic enzymes.
- Can be extremely toxic when levels are elevated after an overdose or from accumulation in disease state.
- **Causes of Iron toxicity:**
  - **Children:** overdose (multivitamins, or ingestion of potent adult preparation, such as prenatal vitamins.)
    - Highly toxic to the children younger than age 6
  - **Adult:** suicide attempts.

اللي ابيكم تعرفونه انه بالأطفال غالبًا يكون بسبب اخذهم جرعات زائدة ، أما الكبار فغالبا تكون محاولات انتحار

- **Normal serum iron levels:** **50 to 150** micg/dL Similar to creatinine level.

:Pharmacology

- **What happens to iron after ingestion?**
  - Normally, 10% of ingested iron is absorbed form intestine and subsequently bound to "transferrin", using only 15-35% of iron-binding capacity of transferrin.
  - Then it goes to the liver and stored as "Ferritin".
- **So what happens if the person ingests high amounts of iron?**
  - When iron levels rise following a significant iron overdose → transferrin becomes **saturated** → excess iron circulates as **free (unbound) iron in the serum which is toxic**.

لو اسألكم وش اللي يسبب كل المشاكل؟ الفري ايرون!!!!!!

- **The total iron-binding capacity (TIBC):** a crude measure of the ability of serum proteins—including transferrin—to bind iron, ranges from 300 to 400 micg/dL.
  - It is higher than the serum iron level because of a low degree of saturation.

- **Elemental iron:**
  - In the assessment of the severity of an iron exposure, it is IMPORTANT to refer to the amount of **ELEMENTAL IRON** ingested because toxicity of iron compound depends on the amount of elemental iron.
  - Manufactories are also required to list the amount of elemental iron per tablets.
  - **Ingestion of:**
    - **<20 mg/kg:** No symptoms. Observed **without** further therapy.
      no symptoms.
    - **20 to 60 mg/kg**: **Mild** to **moderate** symptoms.
    - **> 60 mg/kg:** May lead to **severe** morbidity.

COMPOUND	PERCENTAGE OF ELEMENTAL IRON
Ferrous sulfate	20
Ferrous fumarate	33
Ferrous gluconate	12
Ferric pyrophosphate	30
Ferrocholinate	14
Ferroglycine sulfate	16
Ferrous sulfate, dried	33
Ferrous carbonate, anhydrous	38
Carbonyl iron	100

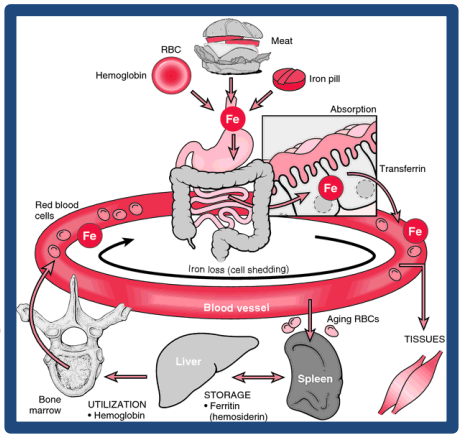
ELEMENTAL IRON (MG/KG)	PEAK SERUM IRON (µG/DL)	TOXICITY
<20	50–150	None
20–40	150–300	Mild
40–60	300–500	Moderate
>60	>500	Severe

✓ **How to calculate toxic dose?** جت بالاختبار تقريبا قبل سنة الى سنتين
  - 60 mg\kg.
  - Calculate the toxic dose If 20 kg?
    - (60*20) = 1200 mg = 1.2 g.

✓ The doses of elemental iron associated with **50% mortality (LD50)** is reported to be 200 to 250 mg/kg**.**
  - What is "LD50"? it is the dose of iron that may induce 50% mortality in a population.

:Pathophysiology

- **Iron has t wo distinct toxic effect**:
  - It causes injury to the **gastrointestinal mucosa**.
  - It impairs **cellular metabolism**, primarily of the heart, liver, and central nervous system (CNS).
- **Unbound (free) iron** moves into cells and localizes near the mitochondrial cristae → resulting in **uncoupling of oxidative phosphorylation** → **impairment of adenosine triphosphate (ATP) synthesis**. Most of the enzymes will be affected (they depend on ATP) → tissue necrosis.
  - Oxidative phosphorylation is the metabolic pathway in which cells use enzymes to oxidize nutrients, thereby releasing energy which is used to produce adenosine triphosphate (ATP).



- **Effects:**
  - **Cell membranes** are injured by free radical-mediated lipid peroxidation.
    - Iron excess is believed to generate oxidative stress, understood as an increase in the steady state concentration of oxygen radical intermediates
  - Iron **increases capillary permeability** and induces both arteriolar and veno-dilation.
  - **Myocardial toxicity** decreases cardiac output.
  - Hydration of the iron molecule creates an excess of unbuffered protons, worsening **metabolic acidosis.**
  - This multitude of effects, combined with severe gastrointestinal fluid losses, can lead to the development of **shock**, **cardiovascular collapse**, and **death**.

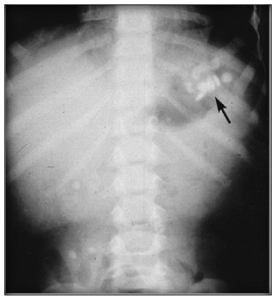
:Clinical features

Five stages. IMPORTANT (you can find a simple diagram in the summary)

Phase I:	○ **Stomach phase** reflects the corrosive effects of iron on the gut. (Vomiting, cramps, bleeding, hematemesis) ○ Vomiting occurs within 80 minutes of ingestion in more than 90%of symptomatic cases. Diarrhea, which can be bloody, follows. ○ Abscence of these symptoms→ غالبا ماراح يكون فيه توكسستي، وبهذي الحالة مانبلغ الأهل لأن يمكن الشخص يبحث عن الاهتمام :)
Phase II:	○ **Silent phase** الهدوء الذي يسبق العاصفة represents an apparent (but not complete) recovery that lasts less than 24 hours but can extend up to 2 days. ○ Most patients recover after this point.
Phase III:	○ **Characterized by:** the recurrence of **GI symptoms**, **severe lethargy or coma**, **anion gap metabolic acidosis**, **leukocytosis**, **coagulopathy**, **renal failure**, and **cardiovascular collapse**. ○ Serum iron levels may have fallen to normal during this phase due to distribution into the tissues. ○ Metabolic lactic acidosis derangements due to iron poisoning include hypoglycemia, leukocytosis, and severe from hypo-perfusion and interference with cellular respiration. ○ Early coagulation defects are probably related to direct effects of iron on vitamin K–dependent clotting factors. ○ Later, coagulationhepatic failure .
Phase IV:	○ **Characterized by fulminant hepatic failure**, occurs 2 to 5 days after ingestion. ✓ Liver is affected. Liver transplant is usually needed in this phase. (Iron "Fries" the liver). ○ This is relatively **rare**, appears to be dose related, and is usually **fatal**.
Phase V:	○ **Represents:** the consequences of healing the injured gastrointestinal mucosa. ○ **It is characterized by**: pyloric or proximal bowel scarring, which is sometimes associated with obstruction.

:Diagnostic stratigies

- The presence of **gastrointestinal symptoms** suggests a potentially serious ingestion, whereas their absence is reassuring & it'll be very unlikely to be caused by iron toxicity.
- Serum iron level: 3 to 5 hours after ingestion, is the most useful laboratory test to evaluate the potential severity of an iron overdose.
- 350 to 500 micg/dL with moderate toxicity, and greater than 500 micg/dL with potentially severe toxicity.
- **Iron tablets are Radiopaque on abdominal radiograph**.
  - Absence of radiopacity doesn't exclude iron toxicity. ليش؟ لأن يمكن صارلها ابزوربشن



:Management

❖ **Gastric Emptying**: NOT USED ANYMORE

- Iron does not bind to **activated charcoal**.
- Neither **gastric lavage** nor **ipecac** effectively removes large numbers of pills.
- Iron tablets clump together as their outer coatings dissolve.
- **Gastrotomy** has been performed to remove iron from the stomach.

❖ **Whole-Bowel Irrigation:** NOT USED ANYMORE - ما قرأ الكلام

- Polyethylene glycol electrolyte lavage solution (PEGELS) (CoLyte, NuLytely, or GoLYTELY) is routinely recommended.
- It is contraindicated in the presence of bowel obstruction, perforation, or ileus

- Hemodialysis and hemoperfusion are ineffective in removing iron due to its large volume of distribution.
- Exchange transfusions have been recommended for severely symptomatic patients with serum iron levels exceeding 1000 micg/dL.

❖ **Deferoxamine: DRUG OF CHOICE - IMPORTANT** "IT COMES IN THE EXAMS EVERY YEAR."

- **Mechanism: Deferoxamine chelates** iron to form the water-soluble compound **ferrioxamine**, also limits the entrance of iron into the cell.
- Pregnancy is not a contraindication to deferoxamine.
- The presence of ferrioxamine turns the urine into **"vin rose?"** color, which reflects the excretion of chelated iron

:Disposition

- The asymptomatic and less than 20 mg/kg of elemental iron can be observed without further therapy.
- If a patient remains asymptomatic for 6 hours of observation, discharge is recommended.

:Lead (pb)

- Lead poisoning is a disease of industrialization and has become the most common environmental toxicologic problem in the united states.
- **How?**
  - Most exposures are from: **ingestion** of contained substances (acute) or i**nhalation** of lead dust (chronic).
    - Less often: direct skin contact with organic lead compounds or from retained bullets in or near joints.

الأشخاص يكونون مشهورين بهالحالة لأن الرصاص ماينشال كله وتبقى منه اجزاء فلمن يروحون للمطار يعانون

- **Sources:** Lead-based paint, Curtain weights, Buckshot, Fishing weights, Food or beverages stored or prepared in lead-soldered cans, Lead-glazed pottery, and Lead crystal decanters.
- Children typically present to the emergency department: (Dr skipped this point)
  - Following an ingestion of lead
  - Symptomatic with a possible exposure history
  - Referred for management of an elevated BLL.
- Lead toxicity in adults most often results from inhalational exposure in the workplace.
- In dr slides (He skipped it):
  - Battery manufacture, radiator repair, bridge and ship construction or demolition, soldering or welding, cable or tin can production, stained glass manufacture, lead-glazed or crystal pottery making, glass production, firing range operation, and lead-based paint abatement.
  - Hobbies at risk include making glazed pottery, target shooting at indoor firing ranges, soldering lead, smelting lead in the preparation of buckshot and fishing sinkers, repairing cars or boats, and remodeling homes.

In dr slides (He skipped it):

- There is no known biologic need for lead.
- Its absorption is highest in malnourished children (approximately 40%) and in pregnant women.
- Although 90 to 95% of lead is stored in cortical bone and teeth, it is also found in the brain, liver, and kidneys.
- Approximately 75% of the absorbed lead is eliminated by the kidneys, with the remainder absorbed through the skin, hair, sweat, nails, and gastrointestinal tract.

:Pathophysiology

- Lead binds to **sulfhydryl groups**: interferes with critical enzymatic reactions.
- **Its toxic effects are most prominent in:**

Hematopoietic.	• **Anemia**: normochromic or hypochromic. (1st clue in lead toxicity - Caused by bone marrow inhibition) • The severity of the anemia correlates directly with the Blood Lead Level (BLL).
Neurologic.	• **High levels of lead >40 micg/Dl will cause Demyelination** and degeneration of motor axons result in peripheral neuropathies. • Lead toxicity also causes **neuropsychiatric disorders** and hypertension. • **Wrist drop and foot drop** are characteristic of adult lead poisoning • In children, LOW (IQ) scores, hyperactivity, decreased attention span, **overaggressive behavior**, learning disabilities, criminal behavior, and subclinical sensorineural hearing loss.
Renal systems	• Lead nephropathy is fibrosis in the proximal tubules, with relative sparing of the glomeruli. • Hyperuricemic gout ("saturnine gout") can result from increased reuptake of uric acid by the tubular cells.

- ✓ Adults and children with acute toxicity may present with lead encephalopathy.
- ✓ Lead poisoning has also been correlated with hypertension.

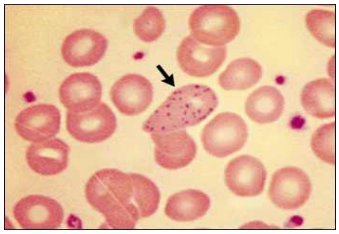
:Clinical features

Not mentioned by the doctor: اقروه على السريع اعادة للكلام اللي فوق

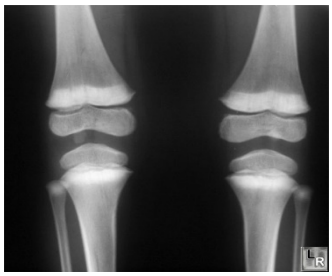
- Acute exposure to lead can result in symptomatic poisoning.
- "Lead colic" is characterized by cramping abdominal pain with nausea, vomiting, constipation, and, occasionally, diarrhea.
- Fatigue, anemia, peripheral neuropathy, renal impairment, and hepatic and CNS dysfunction.
- The CNS toxicity may manifest as mild headache or personality changes to full-blown encephalopathy with coma, convulsions, and papilledema.
- Permanent neurologic and behavioral sequelae may occur.
- Although capillary lead levels correlate well with BLLs, the most informative biomarker is a BLL.
- The Centers for Disease Control and Prevention has defined a chronic BLL of greater than 10 micg/dL as toxic for a child.
- Acute exposure can result in levels up to 100 micg/dL.

:Diagnostic stratigies

- blood cell count, serum glucose, blood urea nitrogen, creatinine, electrolyte levels, and urinalysis.
- A peripheral smear may show **basophilic stippling.**
- Markers of hepatic injury may be elevated following acute exposure.



- In cases of altered mental status, seizures, or coma, a CT of the head will show **cerebral edema** associated with **acute lead encephalopathy**
- In children : **"lead bands"** or **"lead lines"** that are characteristic of chronic exposures.



❖ Acute Lead Encephalopathy

- Standard measures to control cerebral edema, including intubation and neurosurgical consultation for invasive monitoring of ICP are indicated.

❖ whole-bowel irrigation

1. Severe poisoning
2. Radiopacities

:Management

❖ Chelation Therapy

- Any patient with a BLL greater than 70 micg/dL, or with signs suggestive of encephalopathy, will require admission for parenteral chelation therapy.
    - Chelation is a type of bonding of ions and molecules to metal ions. It involves the formation or presence of two or more separate coordinate bonds between a polydentate (multiple bonded) ligand and a single central atom.

الدكتور ماشرح التريتمنت بس قال ان تهمه اسماء الـ4 مع اختصاراتها، اقروه تحسبا لأي طاريْ وقال ماراح اسأل عن الدوز

❖ **Dimercaprol:**

- For these seriously poisoned patients, dimercaprol (or British anti lewisite [BAL]) should be the first chelator given.
- Since dimercaprol is diluted in peanut oil, it is contraindicated in patients allergic to peanuts.

❖ **ethylenediaminetetraacetic acid (CaNa2EDTA):**

- Dimercaprol is followed by calcium disodium ethylenediaminetetraacetic acid (CaNa2EDTA), a highly effective lead chelator.
- The dosage of CaNa2EDTA for patients with
- Acute lead encephalopathy is 75 mg/kg/day or 1500 mg/m2/day given IV or IM in two to four divided doses, with a maximum daily dose of 1 g in children and 2 g in adults.
- CaNa2EDTA should be given only with adequate urine flow or with hemodialysis in renal failure.
- The need for parenteral chelation therapy in asymptomatic or minimally symptomatic children is guided by the BLL.
- A BLL of more than 69 micg/dL mandates hospitalization and parenteral chelation therapy.
- For less seriously poisoned patients, the dosage of CaNa2EDTA is 50 mg/kg/day or 1000 mg/m2/day, given in two to four divided doses for up to 5 days.

❖ **2,3-dimercaptosuccinic acid :DMSA**

- For Serum lead levels of 45 to 69 micg/dL in patients without vomiting or CNS symptoms can be managed in the outpatient setting.
- The initial dose of DMSA is 10 mg/kg every 8 hours for 5 days, then 10 mg/kg every 12 hours for 14 days.

❖ **Oral d-penicillamine**:

- Should be used only in patients who do not tolerate DMSA.
- The usual oral dose of d-penicillamine is 25 mg/kg every 6 hours for 5 days.
- d-Penicillamine is less efficacious than DMSA and has more adverse reactions.
- Penicillin allergy is a contraindication to the use of d-penicillamine.

:Management

About Chelation Therapy: الدكتور ما قراهم

⇨ A BLL between 20 and 44 micg/dL in a patient who is asymptomatic or minimally symptomatic requires a more aggressive medical and environmental evaluation.

⇨ No need for chelation for children with a BLL lower than 45 micg/dL.

⇨ Children with lead levels of 10 to 19 micg/dL need

- Family counseling
- careful follow-up
- frequent screening of BLLs

⇨ The treatment of adults with chronic poisoning is less aggressive than for children.

⇨ If gastrointestinal symptoms or CNS problems are present, hospitalization with parenteral chelation therapy is indicated.

⇨ In the asymptomatic adult or the adult with only mild clinical problems, the only intervention needed is cessation of exposure.

:Disposition

➢ Patients who have ingested a single lead foreign body (e.g., fishing sinker) will usually pass it harmlessly.

➢ If the foreign body remains in the gastrointestinal tract after 2 weeks, removal should be considered to prevent lead toxicity.

➢ Patients who are significantly symptomatic after an acute lead exposure and children with a BLL of 69 micg/dL or greater require hospitalization and chelation therapy.

➢ Patients discharged home on oral chelation therapy should not return to a contaminated environment.

Arensic (As) الزرنيخ

- **What is Arsenic (As)?**
  - A **tasteless**, **odorless** substance that looks like sugar, has an infamous history as an agent of **homicide**.
  - Epidemic poisoning. بالمسلسلات المصرية كانوا يحطونه للحماة الميب مضبوطة
- **Found in:**
  - Smelters and electric power plants that burn arsenic-rich coal.
  - It has also been found as a contaminant in herbal remedies and drugs such as opium.
  - **Contaminated drinking water in underdeveloped countries.**
- **Uses?**
  - It is used in industry as a wood preservative and in the production of glass and microcircuits.
  - Inorganic arsenicals are also used in rodenticides, fungicides, insecticides, paint, and tanning agents and as defoliants in the cotton industry.
  - Arsenic is still used for medicinal purposes in the treatment of trypanosomiasis, amebiasis, and leukemia.

:Pharmacology

- Arsenic has **no** metabolic or biologic function.
- Of the two inorganic forms, trivalent **arsenite (As3+)** is highly lipid soluble and is 5 to 10 times more toxic than the pentavalent arsenate (As5+) form.
  - The more toxic lipophilic trivalent arsenite form has a lower gastrointestinal absorption but is well absorbed by the skin.
- Absorbed arsenic is bound by hemoglobin, leukocytes, and plasma proteins.
- It is cleared from the intravascular compartment within 24 hours and concentrates in the liver, kidneys, spleen, lungs, and gastrointestinal tract.
- Arsenic crosses the placenta and can also accumulate in the fetus.
- Its affinity for **sulfhydryl groups** in keratin makes arsenic detectable in the hair, skin, and nails.
- Arsine (AsH3), a colorless and almost odorless gas, is extremely toxic.
- It is immediately lethal at 250 ppm.
- The excretion of arsenic and its metabolites occurs mainly through the kidneys**.**

:Pathophysiology

- Arsenic binds avidly to **sulfhydryl groups**, inhibiting critical enzymes such as **lactate dehydrogenase** and **glyceraldehyde-3-phosphate dehydrogenase**, a critical step in glycolysis.
- It disrupts oxidative phosphorylation by replacing phosphorus in the formation of phosphate bonds (**arsenolysis**).
- Arsine causes massive hemolysis, the exact mechanism for that is poorly understood.

:Clinical features

- Acute exposure to **arsine gas** is characterized by **severe hemolysis** that is associated with renal tubular injury. غازه كان يستخدم بالحرب العالمية الثانية
- **Gastrointestinal symptoms are common (**Gastroenteritis**)**, and **CNS and liver dysfunction** can occur.
  - **GIT:** nausea, vomiting, abdominal pain, and diarrhea
    - Initial manifestations of acute exposure to arsenic salts.
    - Hematemesis and Hematochezia.
    - Within 30 to 60 minutes of exposure, patients complain of a metallic or garlicky taste.
  - Encephalopathy with seizures and coma, respiratory failure associated with ARDS and dysrhythmias associated with cardiac conduction disturbances.
- **Less common complications**: hepatitis, rhabdomyolysis, **hemolytic anemia**, renal failure, **unilateral facial nerve palsy**, pancreatitis, pericarditis, pleuritis ,and fetal demise.
- **Severe poisoning:** cardiovascular collapse and death

Gastrointestinal
Violent gastroenteritis
Hematemesis/hematochezia
Jaundice
Pancreatitis
Dysphagia
Hepatomegaly

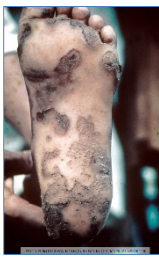
Cardiovascular
Third spacing with shock
Sinus/ventricular tachycardia
Prolonged QT interval, ST depression, T wave inversion
Torsades de pointes
Pericarditis

Respiratory
Respiratory failure
Adult respiratory distress syndrome
Pulmonary edema
Pneumonia

Renal
Proteinuria
Hematuria
Oliguria
Renal failure

Neurologic
Headache
Drowsiness
Delirium
Coma
Encephalopathy
Seizures

- The mortality rate is 25 to 30%.
- **Exchange transfusions** and **plasma exchange** have been used to remove arsine, which is tightly bound to the erythrocytes.
  - ➢ You remove 500 ang give 500 (Give simultaneously to avoid hypotension)
- Urinary alkalinization can be used to decrease renal deposition of hemoglobin.
- **Weeks to Months later on :**
  - ✓ Characteristic **lines in the nails (Mees' lines),** sensorimotor neuropathy.
  - ✓ **hyperkeratosis** of the palms and soles.



:Diagnosis

- **Normal arsenic levels are**:
  - ✓ Blood: 5 micg/L
  - ✓ Urine: (BEST to Dx) less than 50 micg/day in a (24-hour urine)
- Any urine level above 100 micg/day or 50 micg/L **necessitates treatment**.

:Managment

- The initial management should address life-threatening conditions with supportive management of shock, dysrhythmias, and seizures.
- **No Activated charcoal, does not adsorb arsenic**.
- **Hemodialysis** removes arsenic in the setting of acute renal failure.
- **Exchange transfusions** or **plasma exchange** should be considered very early after an arsine exposure.
- With a known history of exposure in asymptomatic patient, chelation should start as early as possible without waiting for laboratory confirmation of the arsenic levels.
- Intramuscular **dimercaprol** is the preferred chelator inpatients who are critically ill.
  DMSA is a water-soluble analogue of dimercaprol that can be given orally.

:Mercury (Hg)

- Mercury is a silver white metal.
- the only metal that **is liquid at room temperature**.
- It has a long history of medicinal uses.
- **Significant poisoning at home:**
  - Sphygmomanometer mercury spilled then was aerosolized by vacuuming
  - Mercury was heated on the kitchen stove to extract gold from ore.
- Various other sources of mercury have also been implicated in intoxication.
  - Because of many industrial uses that include the manufacture of fluorescent lights, batteries, polyvinyl chloride, and latex paint, mercury is a common pollutant of air and water.

  ➢ Found in fishes, and lights

Elemental
Spill from mercury-containing devices
Gastrointestinal exposure from ruptured Cantor or Miller-Abbott tube
Inhalational exposure in the workplace/home
Deliberate injection or ingestion
Accidental ingestion

Salts
Accidental disk battery ingestion
Deliberate ingestion
Laxative abuse

Organic
Oral/dermal exposure to mercurochrome or thimerosal
Repeated injections of drugs containing thimerosal as a preservative
Exposure from occupational or agricultural accidents
Water/soil pollution
Consumption of contaminated seafood
Exposure to paint containing mercury

:Diagnosis

- The most familiar form of mercury is elemental or metallic mercury, also known as "**quicksilver**."
- **A common route:** inhalation of volatilized vapor.
  - After inhalation, 74% of the metallic mercury is retained in the lungs. This can result in severe **pneumonitis** and ARDS.
  - Aspiration of elemental mercury results in primary pulmonary toxicity, in addition to CNS and renal toxicities.
- **Elemental mercury** is not absorbed by the gastrointestinal tract, so ingestion does not normally lead to systemic toxicity unless it becomes trapped in diverticulae.
- Mercury is absorbed through the skin at 1% of the rate of inhaled mercury and is not a concern.
- **Inorganic mercury:**
  - Have two different forms: Hg1+ (mercurous) and Hg2+ (mercuric).
  - Ingestion of either salt leads to significant gastrointestinal and renal toxicity.
- **The organic mercury:** severe hemorrhagic colitis.
  - The major route of exposure to this type of mercury is through ingestion, but these compounds are also readily absorbed through the skin.
  - These organic forms classically result in delayed neurotoxicity with prominent ataxia, tremor, dysarthria, and tunnel vision.

:Pathophysiology

- Mercury binds **sulfhydryl groups**.
- **Effects:**
  - **Nephrotoxicity:** direct damage and an immune reaction in the kidney.
  - **The skin changes:** immune reaction.
  - Mercury increases catecholamine level resulting in hypertension and tachycardia.
  - Atrophy of the cerebellum.

:Clinical features

- Inhalation of elemental mercury onset of shortness of breath, fever, and chills that progresses to **pneumonitis** and respiratory distress.
- Aspiration of liquid metallic mercury during medical procedures > rapid onset of tracheobronchial hemorrhage
- Acute ingestion of inorganic salts typically causes a corrosive gastroenteritis with third spacing and hemorrhage.
- Patients complain of a metallic taste in the mouth and may have a grayish discoloration of the mucous membranes.
- Massive fluid loss results in shock and acute tubular necrosis.

:Diagnosis

- Normal blood mercury levels are considered to be
  Blood: less than 10 micg/L
- Blood level more than 35 micg/L needs Rx.

:Management

- Initial management in the acutely poisoned patient should be aggressive support and decontamination.
- Gastric lavage with protein-containing solutions (e.g., milk and egg whites) may be beneficial in the decontamination of the gastrointestinal tract following ingestion of mercury salts.
- Charcoal adsorbs very little and is not recommended
- Ingested metallic mercury is generally harmless unless its passage is impaired by entrapment in a diverticulum or the appendix.
- For acute inhalational exposures patient should be removed from the source
- Supportive management provided.
- There is no role for prophylactic antibiotics or steroids.
- Suction and postural drainage are indicated in cases of acute aspiration of metallic mercury.
- Self-injection of metallic mercury often requires surgical debridement of infiltrated tissue

Chelation therapy:

- **BAL** is used for clinically significant acute **in**organic mercury intoxication.
  - Because it increases brain mercury levels in patients with methylmercury poisoning, BAL is contraindicated for patients poisoned with organic mercury compounds.
- **DMSA:** used for both acute and chronic mercury poisoning and may be the best chelator for methylmercury.
- **D-Penicillamine** is also used.

It should be administered only after thorough gastrointestinal decontamination because mercury absorption from the intestinal lumen is enhanced by the penicillamines.

[TAKE HOME MESSAGE!!]

- ✓ YOU HAVE TO MEMORIZE IRON BY HEART "VERY VERY IMPORTANT".
- ✓ Free iron is what kills the patient.
- ✓ The toxic dose of iron is 60 mg\kg.
- ✓ LD50? Reported to be 200-250
- ✓ It is IMPORTANT to refer to the amount of **ELEMENTAL IRON** ingested.
- ✓ Sulfhydryl groups = LEAD , ARESENIC , MERCURY.
- ✓ IMPAIRMENT OF ATP SYNTHESIS = Iron.
- ✓ ANTIDOTES ARE **VERY IMPORTANT**.
  - Iron antidote = ######### Deferoxamine.
  - Lead = Dimercaprol (BAL)– $CaNa_2EDTA$ – DMSA – D-penicillamine.
  - Arsenic = Dimercaprol (BAL) – DMSA.
  - Mercury = BAL (Dimercaprol) - DMSA - D-penicillamine
- ✓ IRON TOXCITY Phases are IMPORTAT → See the table below.
- ✓ Lead = WRIST DROP & FOOT DROP – anemia – demyelination , basophilic stippling – lead bands.
- ✓ Arsenic = Hemolytic anemia , unilateral fascial palsy – Mee's line in the nails , hyperkeratosis.
- ✓ **Iron tablets are Radiopaque on abdominal radiograph**.

TYPE OF MERCURY/ROUTE OF EXPOSURE	SIGNS/SYMPTOMS
Inhalation of metallic mercury	Hypoxemia Respiratory distress, ARDS Dyspnea, chest tightness Fever, chills Burning in mouth and throat Nausea, vomiting Bloody diarrhea Renal tubular necrosis
Aspiration of metallic mercury	Aspiration pneumonitis ARDS
Subacute/chronic inhalation of metallic mercury	Metal fume fever Neuropsychiatric symptoms Renal dysfunction Skin changes
Ingestion of inorganic mercury salts	Severe hemorrhagic gastroenteritis, shock, hypovolemia, third spacing Acute tubular necrosis in 24 hr, with albuminuria and hematuria
Subacute/chronic inhalation of inorganic mercury	Neurasthenia, erethism, acrodynia
Organomercury exposure (methyl-, diethyl-)	Delayed neurologic problems (ataxia, tremor, dysarthria), visual field constriction, hearing loss, spasticity, hyper-reflexia

ARDS, acute respiratory distress syndrome.

Stages of Iron Toxicity

Stage	Clinical Effect	Time Frame
Stage I	• GI Irritation	30 min - 6 hrs
Stage II Latent	• Recovery from GI symptoms	6 hrs - 24 hrs
Stage III Shock and metabolic acidosis	• Metabolic acidosis (anion gap) • Dehydration • Lactic acidosis	6 hrs - 72 hrs
Stage IV Hepatotoxicity/Hepatic necrosis	• Fulminant hepatic failure	12 hrs - 96 hrs
Stage V Bowel obstruction	• GI mucosa healing leads to scarring	2 wks to 8 wks

MCQs

❖ **Which one of the following agents and antidotes are correctly paired?**

A. Anti-cholinergic overdose – Flumazenil.
B. Aspirin overdose – N-acetyl cysteine (NAC).
C. Iron overdose – Deferoxamine
D. Paracetamol overdose – Physostigmine.

❖ **Which one of the following has the highest elemental iron composition?**

A. Ferrous sulfate.
B. Ferrocholinate.
C. Carbonyl iron.
D. Ferrous gluconate.

❖ **Which one of the following is antidote for arsenic poisoning?**

A. Nalmefene.
B. Glucagon.
C. D-penicillamine
D. Hydroxocobalamin.

❖ **Which one of the following is the antidote for Lead poisoning?**

A. DMSA.
B. Dantrolene.
C. digiband.
D. Deferoxamine.

Answers : 1) : C , 2) : C , 3) : C