LEAD POISONING

CLASSIFICATION, ETIOLOGY, POSITIVE DIAGNOSIS, TREATMENT AND PROPHYLAXIS.

Professional lead poisoning (Saturnism)

• The use of human lead dates back over 2000 years. Lead poisoning has been known since antiquity, with Hippocrates being the first to masterfully describe Saturnine colic. From the first place in occupational morbidity held in the 19th century, poisoning has now reached the third place after pneumoconysis and occupational poisoning with organic solvents. This is due to the modification of technological processes that have substantially reduced the risk of intoxication, medical control in enterprises and improved means of diagnosis and treatment. In industry, poisoning can occur with metallic lead, its inorganic and organic compounds.

Definition of Lead:

• Lead is a white-gray metal with high density, making it a category of bivalent heavy metals. In the solid metal state, it is soft, malleable, easy to pull into thin sheets. It oxidizes rapidly on the air contact surface. It is soluble in nitric acid and organic acids, acidic foods, water containing nitrates and ammonium salts, but is very resistant to the action of sulfuric acid.

Distribution in the Lead environment:

•Lead is widespread in the environment. It can be found in:

- Sea water
- Aer
- Sol
- Plant
- Animal body
- Food
- The earth's crust
- Metal ores

Commercial and industrial applications where Lead is found:

Lead can be present in:

- Varnishes and paints
- Battery
- Pigments
- Insecticides
- Plastic and ceramic materials
- Medical equipment
- production of weapons
- Gasoline
- Welding industry.

Jobs at Risk of Lead Exposure:

- Extraction of lead-containing ores
- Melting Lead and Lead alloys
- Collecting and smelting old lead-containing materials
- Welding of Lead plates
- Industry that produces: batteries, graphics, chemicals

- Lead lining and repair of acid storage tanks
- ..... and food industry
- Making Lead pipes
- Lead alloy mowers
- Manufacture of lead and glass
- Preparation of Lead paints and their use
- Manufacture of explosives, fuels, plastics industry, etc.

Etiology

A. The main etiological factor: Lead.

- Lead is not found in its natural state, only in ores.

The most important lead ore is galena (lead sulfide), from which metallic lead is extracted.

- Metal lead:
- melts at 327ºC
- emits vapors at 450ºC

chemical combinations in which lead is found

Lead oxides:

- lead monoxide, PbO = litarga or masicot (yellow)
- lead tetraoxide, Pb3O4 = minium lead (red)

Inorganic Lead salts:

- lead carbonate, PbCO3 = waxed
- another type of lead carbonate , 2PbCO3 + Pb (OH) 2 + wax (white lead)
- lead chromate, PbCr2O3 (yellow)
- lead azide, Pb (N3) 2

Organic lead salts:

- lead tetraethyl and tetramethyl Pb (C2H5) 3
- lead stearate
- lead naphthenate

(secondary) : predisposing etiological factors

A. with regard to the human body:

- intense physical exertion
- overworked
- malnutrition
- alcoholism
- virus
- the appearance of metabolic acidosis

B. regarding the workplace:

- unventilated interior spaces
- broken ventilation
- non-compliance with individual hygiene
- non-compliance with labor protection rules
- not using specific personal protective equipment.

Exposure time probably until intoxication: from 2-3 months to 2-3 years (depending on the amount of lead absorbed and individual characteristics: the presence of favorable factors).

contamination:

1. Respiratory tract - the major tract of entry

, the deposition rate ranges from 30-85%, half of the exposed rate is found in the blood over 50

hours, the rest is deposited in the tissues or is eliminated

2. Digestive tract-
non-compliance with hygienic measures at work - unwashed hands, keeping food at work, smoking while working

3. tegumentary - only in the case of fat-soluble organic compounds - Lead stearate, naphthenate, tetraethyl and tetramethyl.

Metallic lead is not absorbed through the skin.

Mechanism of action:

• Lead enters the body through the respiratory tract in the form of vapors and powders and through the digestive tract, but absorption is limited to about 2% of the amount ingested.

• A large part is transformed in the digestive tract, into insoluble lead sulfide, black in color. Lead produces a caustic effect on the digestive tract.

• Lead is not absorbed through intact skin (exception - lead tetraethyl

• can penetrate the skin intact).

• Circulation takes place in the colloidal form of basic Pb triphosphate, bound to

• erythrocytes.

• Storage is done in bones in a non-toxic form, its metabolism being

• similar to that of calcium.

• Lead deposition in bone trabeculae is a mechanism of inactivation by its sequestration in a biological compartment with a low sensitivity to its toxic action.

PATHOGENESIS

Lead acts by enzymatic mechanisms, mainly by inhibiting the SH groups of the protein component of the enzyme. Lead inhibits heme synthesis by blocking enzymes that catalyze the transition from lacoproporphyrin to protoporphyrin. Through this mechanism, Pb intoxication achieves hypersideremic hypochromic anemia, called acrestic.

Excess Lead in the blood causes toxic phenomena through the following mechanisms:

• Direct aggression on the hematopoietic tissue followed by inhibition of hemoglobinosynthesis;

• Direct action on the erythrocyte membrane followed by weakening of the erythrocytes, reducing their lifespan and early intravascular hemolysis;

• Action on chromosomes followed by alteration of genetic material, which in young women causes

• infertility, abortions or stillbirths with birth defects;

• Direct nephrotoxic action as well as damage to the mitochondria of renal tubule cells, which is

• morphophysiopathological substrate of saturnine nephropathy;

• Neurotoxic action on the central and peripheral nervous system;

• Harmful action on the cardiovascular system.

• Excess lead ions in the peripheral blood inhibit at least three enzymes in the heme synthesis chain:

• Aminolevulinic acid dehydrase which presides over the conversion of aminolevulinic acid to porphobilinogen,

• Coproporphyrinogenase that allows the conversion of coproporphyrin III to protoporphyrinogen

• Hemsynthetase that promotes the integration of activated iron (Fe2 +) in the tetrapyrrole structure of protorphyrin IX to result in heme.

Effect of Lead on Renal Function:

From a clinical-functional point of view, two types of manifestation of nephrotoxicity of lead have been described:

• Acute nephrotoxicity - morphological and functional changes occur in the proximal renal

tubules

• As a consequence decreases the transtubular transport of ions with hyperaminoaciduria, glucosuria;

• These changes are the consequence of altered respiratory function of mitochondria and oxidative phosphorylation in proximal tubule cells;

• Chronic nephrotoxicity - the consequence of chronic acute nephropathy or the progressive development of chronic nephropathy

• With the advancement of nephropathy, tubular atrophy and interstitial fibrosis appear, then the decrease of the glomerular filtration rate and the increase of serum nitrogen are associated;

• In addition to tubular damage, the glomerular vessels also change;

• Lead produces effects on DNA and RNA metabolism by increasing urinary excretions of N-aminoisobuitric acid (ABA which is a product of thymidine degradation, a constituent of DNA and RNA).

Effect on erythropoiesis:

• Affects erythropoiesis

• Inhibits heme synthesis in the bone marrow erythroblast

• Lead blocks the activity of some of the enzymes in the heme biosynthesis chain (ALAdehydrase, coproporphyrinogen oxidase, hemsynthetase, PBG deaminase).

Consequences of enzymatic damage:

• Increased ALA levels in the blood and excreted in the urine

• Increased levels of coproporphyrinogen III in red blood cells and coproporphyrin in urine

• Increased protoporphyrin IX levels in red blood cells

• Increased porphobilinogen and uroporphyrin in the urine

• Increased total serum iron levels

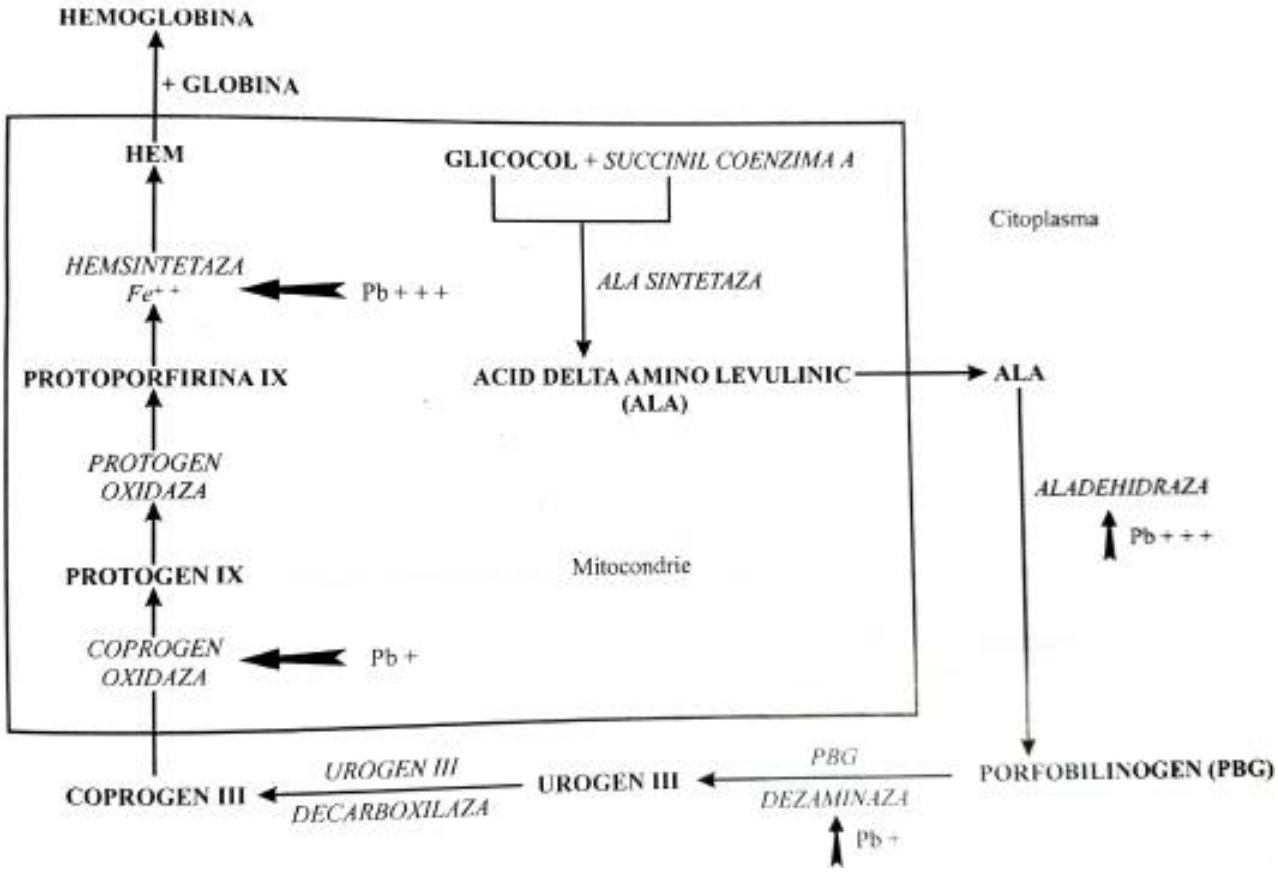

Fig.1 Heme biosynthesis and areas of lead attack. The and + arrows represent the location of the lead inhibitory action and the degree of inhibition.
(Scheme adapted from Granick cit.34 and M.Duc)

Effect of Lead on BP:

• Disorder of eicosanoid synthesis in renal tissue

• Increased thromboxane production (vasoconstriction) with decreased production of PG E and G, ketoprostaglandin F (vasodilation)

• Prostaglandin depletion increases sodium retention and intensifies the pressor response to angiotensin II and vasopressin - essential hypertension is installed

• Decreased urinary kallikrein

• Lead acts on renin-angiotensin-aldosterone with increasing plasma renin, angiotensin and conversion enzyme activity

• The relationship with BP depends on the dose. At low exposures, affected individuals may have elevated plasma renin activity. In severe exposures - its activity may be normal or even low

Effect on vitamin D metabolism:

• Physiological synthesis of vit. D requires a present hydrolysis in the kidneys which converts 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D which facilitates the absorption of Ca in the intestineLead produces an inhibitory effect on the hydroxylase which presides

• vit training. D

• Lead reduces the excretion of urinary uric acid with the consequent increase in the concentration of uric acid in the blood (the level of lead caused is higher in patients with gout who have nephropathy)

• Lead alters the receptivity of the smooth muscles of the vessels to vasoactive stimuli with the consequent increase in the response to alpha 2-stimulation of cardiac and vascular cholinergic and dopaminergic adrenoreceptors

• Lead can alter the Ca-related functions of vascular smooth muscle by lowering ATPase and stimulating the Na / Ca pump

Effects on reproduction and carcinogenesis:

• Reducing the number of births in the families of members who were exposed to the Lead action

• Increased number of premature births, premature rupture of the membrane, sperm changes (mobility, morphology, density)

• Improvement of sperm parameters has been shown experimentally with a decrease in the blood concentration of Lead and protoporphyrin zinc

• There is a risk of congenital cardiovascular abnormalities in newborns whose parents have been exposed to significant amounts of Lead.

• Lead increases the incidence of kidney tumors following the administration of substances

• Proven carcinogens (N-ethyl-N-hydroxyethylnitrosamine)

• This is due to the proliferation of renal tubular cells.

Effects on thyroid function and immune system:

• Lead causes decreased iodine uptake in the thyroid with a decrease in serum thyroxine below 60 mcg%

• On the other hand, hyperthyroidism can cause the mobilization of lead stored in the bones inducing clinical phenomena of intoxication

• Regarding the immune system, there was a decrease in the percentage of B lymphocytes and a decrease in the total number of lymphocytes, monocytes and granulocytes in workers exposed to Lead.

Acute intoxication:

• It is rare due to adequate technical and medical prevention measures.

• It is manifested by hypersalivation, nausea, vomiting, colic pain in the abdomen. Death occurs quickly.

• The major forms are saturnine colic and acute saturnine encephalopathy

• Saturnine colic occurs suddenly or in the background

• astheno-neurotic or dyspeptic manifestations

• (nausea, vomiting, anorexia)

• It is characterized by intense pain

• diffuse throughout the abdomen, or paraumbilical,

• with lumbar irradiation

• The pain gives way to deep abdominal pressure which differentiates saturnine colic from other abdominal colic

• The abdomen is normal or slightly excavated and has no muscle contracture

• On palpation - periumbilical, epigastric or colic sensitivity

• Constipation accompanies pain, is tenacious and does not yield to the usual symptomatic medication; very rarely diarrhea

• Advancing BP figures that return to normal with pain relief
• Oliguria

Acute saturnine encephalopathy:
• Prodromal period with intense headache, insomnia, agitation, tremor
• generalized, then
• Disorientation
• Stupefaction
• Visual disturbances
• Paresis of the cranial nerves
• Delirium with hallucinations and agitation
• Convulsions, coma and death may occur
• At the objective examination, tremor, osteotendinous hyperreflectivity are highlighted
• At the examination of the fundus - edema and papillary stasis, arterial spasm, retinal hemorrhages
• Blood Lead, Urinary Lead values are much higher

Chronic intoxication:
In industrial conditions it is common.
The main syndromes:
• Asteno-neurotic (headache, dizziness, asthenia, fatigue, insomnia, behavioral changes, daytime drowsiness, decreased memory, loss of appetite)
• Dyspeptic (epigastric embarrassment, constipation, bloating, early satiety, metallic taste, Burton lyser - coloring of gums and teeth in lilac color, width 1-2-3 mm)
• Impaired peripheral CNS (impaired visual acuity, behavioral disorders, paresis and paralysis of the radial nerve, weakness in the hands and fingers, tingling, polyarticular pain in the joints of the lower limbs)
• Anemic - reticulocytosis, basophilic granular erythrocytes in large quantities, oligochrome anemia.
• Cardinal symptom - Lead color - green-earth skin color

POSITIVE DIAGNOSIS:
In order to establish the clinical diagnosis, it is mandatory to establish the PROFESSIONAL ROUTE with the detailing of the professional risk factors evaluated for each job where the suspect in the occupational disease has activated and a series of paraclinical examinations.
Establishing professional lead exposure
• Subjective: - professional anamnesis.
• Objective: - determinations of lead in the workplace air, showing exceeding the maximum permissible limits of lead.
• official documents regarding professional seniority.
Clinical aspects : the presence of one, several, or all of the above-mentioned clinical syndromes.

Laboratory examinations:
a. Exposure indicators:
• Pb-S blood lead level, over 70 mg / 100 ml
• Pb-U leaden, over 150 mg / l
b. Biological effect indicators:
• ALA-U delta aminolevulinic acid in urine, over 20 mg / l
• CP-U coproporphyrins in urine, over 300 mg / l
• Low Hb - less than 11g% - in women, - less than 12 g% - in men

• Red blood cells with basophilic granules - HGB
• normal = less than 500 per 1 mil / h,
• HGB between 500x10 - 5000x10 indicates increased lead absorption
• safe lead poisoning = over 5000 to 1 mil / hm
• Reticulocytosis over 13
• Sideremia = over 140 mg / 100 ml

c. Lead elimination test caused by EDTAMIN (total elimination over 5 days, corrected for normal diuresis):
• normal = less than 2 mg Pb-U
• increased absorption = between 2-12 mg Pb-U
• lead poisoning = over 12 mg Pb-U

DIFFERENTIATED DIAGNOSIS:

A. Saturnian colic must be differentiated from:
• kidney colic
• hepatic colic
• bowel obstruction
• perforated gastric or duodenal ulcer
• acute appendicitis

B. Anemia from lead poisoning must be differentiated from:
• other types of anemia
• viral hepatitis
• other hemolytic anemia with jaundice

C. Saturnine encephalopathy:
• other encephalopathies: hepatic encephalopathy, hypertensive encephalopathy

TREATMENT:

1. Etiological:

a) Interruption of professional contact with lead
b) Gastric lavage with precipitating sodium or magnesium sulphate solution
c) Lead in the form of insoluble sulphate
d) EDTA chelating treatment (EDTAMIN) = (disodium monocalcium salt of ethylenediaminetetraacetic acid)

• 2 ampoules (2 g) slowly intravenously, 1 ampoule in the morning and 1 ampoule in the evening for 5 days, during which time the urine is collected and the total amount of lead excreted is determined over 5 days.
• break 3 or 5 days
• repeat the treatment (2 ampoules / day for 5 days) if the total amount of lead removed for 5 days exceeds 12 mg (12000 mcg) at the first discharge.
• Dimercaptosuccinic acid (DMSA) - mobilizes Pb from soft tissues, is used orally, 10-20 mg / kg divided into 4 doses, in 1 day with the daily increase of the dose by 3 mg / kg up to 30-40 mg per day 6th or 30 mg / kg for 5 days.
• Penicillamine - 1-1.5 gr / day under the control of lead.
• It is less effective than EDTA but the advantage is in oral use. Prolonged administration may cause aplastic anemia and neuropathy due to vitamin C deficiency.
• Patients with no encephalopathy are usually given Succimer 10 mg / cg orally every 8 hours for 5 days, followed by 10 mg / cg orally every 12 hours for 14 days.
• Patients with encephalopathy are treated with Unitiol at a dose of 75 mg i / m every 4 hours and 1000-1500 mg / m Na Ca Edetat i / v 1 time in 24 hours. The first dose of Na Ca Edetat should be given no earlier than 4 hours after the first introduction of Unitiol to prevent Pb from entering the

brain. Unitiol administration may be discontinued after a few doses depending on the Pb concentration and the severity of the symptoms. The combined treatment is administered for 5 days, after which the detoxification takes place for 3 days.

• Symptomatic: in saturnine colic, in addition to chelation treatment, to relieve pain - Plegomazine (prozim) or Chlorthalasin, 1-2 ampoules of 25 mg i / m.

• Pathogenic: broad-spectrum vitamin therapy (B1, B6, C). Concomitant use of vitamin C increases the effectiveness of EDTA treatment.

PROPHYLAXIS:

• Technical-organizational measures:

• Removal of lead from the technological process

• Automation of technological processes

• Isolation of the equipment and / or of the technological processes generating steam or lead dust, from the place where the worker works.

• Preventing the penetration of vapors and lead dust into the workplace air by: local ventilation, use of processes
humid, general ventilation.

• Preventing or reducing lead on workers by:

• reduction of physical effort;

• endowing and obliging the use of personal protective equipment; its maintenance in good condition;

• appropriate work regime (succession of work and rest periods), with appropriate rest conditions,

• realization and good maintenance of the social-sanitary annexes (locker rooms, showers, toilets);

• reduction of the working day, in certain cases (to 6 hours / shift);

• rational and protective power supply,

• effective training for labor protection and observance of technological discipline.

• Permissible concentration of lead and compounds (excluding PbS):

• average permissible concentration: 0.05 mg / m^3

• the maximum allowable concentration. 0.1 mg / ml

Medical measures

a. Recognition of the risk of Pb poisoning in the territory of the enterprise or of the urban or rural constituency.

b. The employment medical examination (according to H.G. 1025/2016), which consists of:

• blood count

• blood creatinine

• urinary uroporphyrins

• ECG

c. Periodic medical examination:

• half-yearly clinic

• annual creatinine

• aminolevulinic delta acid or free erythrocyte protoporphyrin annually

• annual lead

• annual blood count

• EMG

Health education.

• for the technical and administrative bodies: the implementation of the technical-organizational measures will be taken into account, recommended through a report of findings with recommendations, discussions within the board of directors of the enterprise, talks with the

employer, etc.

- for workers, special consideration will be given to:
- smoking cessation in the workplace
- prohibiting the consumption of food at work
- observance of individual hygiene before meals, smoking at workplaces where it is allowed.
- oral hygiene
- correct wearing of personal protective equipment
- observance of individual hygiene at the end of the exchange
- consuming protective food (milk) during the shift (do not take it home)
- reduction of alcohol consumption (pay special attention to brandy produced by private producers, which may contain lead)
- Knowledge of the first signs of illness
- compulsory presentation at the periodic medical examination

Question
FOR KNOWLEDGE VERIFICATION:

1. What are arenas?

• arenas are substances composed of carbon and hydrogen in the structure of which one or more benzene nuclei appear.
• are also called aromatic hydrocarbons, containing in their molecule one or more rings of 6 carbon atoms.
• when the arena molecule consists of a single ring they are called mononuclear arenas, and when the molecule comprises two or more cycles they are called polynuclear arenas.

2. What is the structure after Friedrich August Kekule?

• the 6 C atoms in the molecule are joined in a ring by 3 double bonds that alternate with 3 single bonds.
• As a gasoline additive, benzene increases its octane number and reduces detonation.
• Consequently, it often contained significant amounts of benzene before the 1950s, when lead tetraethyl was introduced as an antidetonator.
• In recent years, as a result of declining lead gasoline production, benzene has been reintroduced as an additive.
• In the USA, due to the negative effect on health and to reduce the risk of groundwater pollution with this substance, a maximum allowable emission of approximately 1% of benzene was imposed.

3. What are the high-importance chemical compounds obtained by replacing one or more benzene hydrogen atoms with other functional groups?

• Compounds obtained by substitution with an alkyl group:
• C6H5-CH3 toluene
• C6H5-CH2CH3 ethylbenzene
• xylene C6H4 (-CH3) 2
• mes6 ethylene C6H3 (-CH3) 3
• Compounds obtained by substitution with other groups:
• phenol C6H5-OH
• C6H5-NH2 aniline
• chlorobenzene C6H5-Cl
• nitrobenzene C6H5-NO2
• picric acid C6H2 (-OH) (-NO2) 3
• trinitrotoluene C6H2 (-CH3) (-NO2) 3
• C6H5-COOH benzoic acid
• salicylic acid C6H4 (-OH) (- COOH)
• acetylsalicylic acid C6H4 (-O-C (= O) -CH3) (- COOH)
• paracetamol C6H4 (-NH-C (= O) -CH3) -1 - (- OH) -4
• phenacetin C6H4 (-NH-C (= O) -CH3) (- O-CH2-CH3)
• Compounds with two or more benzene rings:
• mothballs
• anthracene
• indol
• Benzofuran.

4. What does benzene represent?

• The simplest combination of aromatic hydrocarbons is 2.7 times heavier than air.
• Colorless liquid

• Boiling point 80 C
• Easily soluble in water
• Very fat soluble.

5. What are the professions at high risk of exposure?
• Dry distillation of coal
• Oil refineries
• Synthetic chemical industry
• Pharmaceutical industry
• Dye industry
• Explosives industry
• Rubber industry (solvent)
• Solvent for paints, varnishes
• Plastic industry (solvent)
• Fat extraction
• Degreasing in metallurgy, leather, textile, footwear industry
• Printing houses
• Wood industry
• Manufacture and use of soldering solutions
• Leather processing (solvent for paints, gluing).

6. What are the ways of penetration into the body?
• Respiratory
• Digestive
• Through the skin.

7. Benzene is obtained from which compounds?
• Benzene is obtained from carbon-rich compounds that suffer from incomplete combustion.
• It is obtained naturally from volcanoes and forest fires, being present in many other combustion products including cigarette smoke.
• Until World War II, significant amounts of benzene were a by-product in the production of coke used in the iron industry.
• In the 1950s, the demand for benzene increased substantially, especially in the plastics industry, thus necessitating its much more productive extraction from oil.
• Currently, benzene is obtained mainly in the petrochemical industry, the production of the coal compound being very little used.

8. What are the four chemical processes in industrial benzene production?
• catalytic reforming
• hydrodalkylation of toluene
• disproportionation of toluene
• steam cracking.

9. What are the states where benzene came from?
• In 1996, it was 33 million tons, of which 7 million came from the United States
• 6.5 million from the European Union
• 4.2 from Japan
• 1.4 million from South Korea
• one million from China.

• Romania is a country that produces, consumes and exports benzene, the most important refineries where benzene is manufactured are Rafo Onești, Oltchim S.A. and Rompetrol.

10. What was benzene used for before the 1920s?

• benzene was frequently used as an industrial solvent, especially for degreasing metals but due to its high toxicity it was replaced with other solvents.
• Its main use is as an intermediate reagent for the synthesis of other chemical compounds.
• Benzene derivatives that are produced in significant quantities are styrene, used in the manufacture of polymers and plastics, phenol, from which resins and adhesives are prepared, cyclohexane, used for the preparation of nylon.
• Smaller amounts of benzene are used in the manufacture of tires, lubricants, dyes, detergents, medicines, explosives or pesticides.

11. In the 1980s, what were the main benzene compounds listed?

• ethylbenzene, in the process using 48% benzene
• cumen 18%
• 15% cyclohexane
• 7% nitrobenzene.
• As a gasoline additive, benzene increases its octane number and reduces detonation.
• Consequently, it often contained significant amounts of benzene before the 1950s, when lead tetraethyl was introduced as an anti-knock.
• In recent years, as a result of declining lead gasoline production, benzene has been reintroduced as an additive.
• In the United States, due to the negative effect on health and the reduction of the risk of groundwater pollution with this substance, a maximum permissible emission of approximately 1% benzene has been imposed.
• The same figure is found in European Union standards.

12. What is the role in the human body?

• in the blood it is transported by lipoproteins and accumulates in high-fat tissues, especially in the hematoforming marrow and CNS, adrenal glands.
• In the body undergoes metabolic changes and turns into phenols, pyrocatechins, hydroquinone (Nomyama) and degrades even to carbon dioxide. By opening the benzene nucleus, muconic acid is also produced, it can be condensed with cysteine, by acetylating its NH2 radical and 1-phenyl-mercapturic acid is formed.
• Phenols are conjugated with glucuronic acid and sulfates, appear in the urine as ether sulfates and glucuroconjugates.

13. How does the disposal take place?

• Through the respiratory tract (30-75%) on average 50% of the inhaled, through urine in the form of phenols and diphenols; pyrocatechol, hydroquinone and phenyl-mercapturic acid. Lim. fiz. sup. for total phenols: 130mg / l.

14. Explain the mechanism of action?

• Toxic to mitosis; inhibitory action on medullary cells, enzymatic disorders of mitosis, on young forms - on granulocyte, erythrocyte, platelet series.
• Causes disorders in the synthesis of corticosteroids, hypovitaminosis B2, C, B6, PP, toxic endothelial damage, enzymatic disorders: catalase, peroxidase, phosphatase; decreases the phagocytic reaction. It is hepatotoxic. It has a narcotic effect.

15. Symptoms of acute intoxication:
• begins with dizziness
• walking unsafe
• state of euphoria
• drowsiness
• headache
• vomit
• anesthesia
• areflexion
• vasomotor paralysis
• heart attack
• convulsions
• the death

16. What are the complications in acute intoxications?
• in the first phase, serious complications are cerebral and parenchymal hemorrhages, followed by neuropsychiatric disorders, toxic hepatitis, kidney damage and hematological changes.
• Prognosis - reserved, serious.
• The disease begins slowly, insidiously, in the first phase, in latent benzeneism, usually without characteristic, obvious symptoms. In the first phase of the disease there is macrocytosis, hyperchromia, (Manu P.) a transient leukocytosis, (Timar M.) a net hyperplasia of the reticulo-histiocytic system (Hilt, Manu) and chromosomal abnormalities (Manu, Popescu). In this phase a transient increase of the phagocytic reaction and a transient catalase hyperactivity (Dienes) is observed.

17. What is characteristic of chronic intoxication?
• The leukopenicizing action of benzene, alteration of the white series, erythrocytes and platelets, occur gradually, successively, simultaneously or in isolation.
• At this stage, astheno-vegetative symptoms are observed and anemia becomes more and more obvious.
• Bleeding occurs, hepatosplenomegaly is associated, signs of toxic hepatitis.
• In untreated cases there are intercurrent infections, superinfected ulcers. Acute or chronic leukemias have been described in chronic benzene poisoning.
• Toxic encephalopathies occurred only in very severe cases.

18. What is the prognosis of the disease?
• in all cases it is reserved
• serious
• the toxicant going in severe cases until the complete destruction of the bone marrow.

19. The diagnosis of this pathology?
• It is based on exposure, characteristic clinical and laboratory symptoms (leukopenia, myelocytes, promyelocytes) and phenols (over 130mg / l);
• decrease in urine ratio = inorganic sulfur / total sulfur below 0.8;
• hematological changes
• Roetter test 1 (delayed skin discoloration more than 10-15 min after intradermal injection of 2mg dichlorophenol-indophenol solution in 4.9 cm water, 0.1 ml)
• Rumpel-Leede test.

20. Differential diagnosis?
• organic solvents with narcotic action
• diseases of the hematopoietic system.

21. Indicate the treatment:
• in acute intoxication: ADRENALINE is CONTRAINDICATED,
• analeptics
• glucose infusions
• procainamide (in case of extrasystole)
• gluconic calcium
• corticosteroids
• antibiotics
• oxygen therapy
• combating hemorrhages.
• Chronic intoxication - glucose infusions
• C vitamin
• B vitamins
• sodium hyposulphite
• transfusions
• corticosteroids
• antibiotics
• bone marrow transplant
• symptomatic treatment.

22. What is prophylaxis?
• of early importance is the early diagnosis of the disease, in the phase of latent benzeneism.
• technical
• individually - in protective clothing, masks, shower, technical training.
• medical - urine examination (for haematuria), reactive test with dichlorophenol solution, alcohol test, alkaline leukocyte phosphatase (FAL), red blood cell count, hemoglobin, leukocyte count, leukocyte formula, reticulocytes, platelets, before and after medullary excitation, bleeding time, coagulation time, dysproteinemia tests, Rumpel-Leede test, Sulfate-index, urine phenols, myelogram (in the presence of quasi-specific changes in the peripheral blood), clot retraction.

23. Contraindications?
• Congenital or acquired haematological diseases, involving red series, leukocyte, platelet, hemorrhagic syndromes, liver diseases affecting the parenchyma, small and repeated bleeding (hemorrhoids, menometrorrhagia, etc.), operated stomach (resection), significant exposure to medullary toxins ionizing in the background (in the last 5 years).
• C.M.A. 50mg / m3.

24. What are benzene derivatives?
• Toluene (methylbenzene C6H5CH3)
• Xylene (dimethylbenzene) C6H4 (CH3) 2
• Styrene (vinylbenzene) C6H5CH = CH2
• Ethylbenzene C6H5C2H5
• Cumene (isopropylbenzene) C6H5CH (CH3) 2
• Naphthalene C10H8

25. Benzene nitride derivatives are:
• Nitrobenzene C6H5NO

26. Amine derivatives of benzene?
• Aniline (phenylamine, aminobenzene)
• Benzidine
27. Aliphatic halogenated hydrocarbons ...?
• Dichloroethane (ethylene chloride)
• Trichlorethylene (CHCl = CCl2)

28. Characteristics of Toluene (methylbenzene C6H5CH3):
• Higher volatility than benzene
• Professions: benzene benzene + aviation fuel.
• Mechanism of action: does not produce hematological changes.
• Role in the body: in the body it is converted into benzoic acid which is conjugated with glycochol and is eliminated as hippuric acid.
• Elimination: upper physiological limit of hippuric acid: 0.7g / urine of 24; by expired air 18-20%
• Acute intoxication: begins with euphoria, followed by drowsiness and narcosis.
• Chronic intoxication: it is characterized by astheno-vegetative symptoms and irritation of the respiratory tract and conjunctiva, toxic hepatitis, minor-moderate kidney damage.
• Diagnosis:
• symptoms
• exposure
• hippuric acid 1 g / l of urine
• Differential diagnosis: intoxications with other organic solvents.
• First aid and treatment: see benzene.

29. What is characteristic about xylene (dimethylbenzene) C6H4 (CH3) 2?
• Professions: see benzene.
• Role in the body: it turns into toluic acid.
• Mechanism of action: inhibits the function of the hematoforming marrow, effect on erythrocyte, platelet and leukocyte series.
• Acute intoxication: euphoria, drowsiness, anesthesia.
• Chronic intoxication: dermatitis, eczema, conjunctivitis, respiratory tract irritation, anemia, leukopenia, thrombocytopenia, dyspepsia, neuro-vegetative dysfunction.
• Diagnosis:
• symptoms
• exposure
• Treatment: see benzene.
• C.M.A. 100mg / m3.

30. Styrene (vinylbenzene) C6H5CH = CH2?
• Professions: chemical synthesis industry, monomer for polystyrene, solvent for polyesters, synthesis of synthetic rubber, manufacture of emulsifying substances.
• Penetration route: respiratory, gastrointestinal, through the skin.
• Accumulation: in the liver, kidneys, adrenal glands, small intestine and blood.
• Role in the body: turns into mandelic acid and benzoic acid.
• Elimination: through expired air and unchanged in the urine in the form of metabolites.

• Acute intoxication: with various symptoms: irritation of the conjunctiva, respiratory tract, tremor, balance disorders, dizziness, drowsiness, narcosis.
• Chronic intoxication: with conjunctivitis, dermatitis, depressive states, pseudo-neurosis with EEG changes, astheno-vegetative signs, dysmenorrhea.
• C.M.A .: 350 mg / m3.

31. Ethylbenzene C6H5C2H5, what does it represent?
• Occupations at risk of exposure: production of styrene, solvent for paints and varnishes, motor fuel in aviation.
• Role in the body: it turns into hippuric acid (70%), mandelic acid and phenaceturic acid.
• Elimination: through exhaled air and urine.
• Acute intoxication: begins with drowsiness, anesthesia, in severe cases causes pulmonary edema, pulmonary hemorrhage. Causes conjunctival irritation, tearing.
• Chronic intoxication: conjunctivitis, dermatitis.
• C.M.A .: 1400 mg / m3.

32. Cumen (isopropylbenzene) C6H5CH (CH3) 2?
• Professions: solvent for nitrocellulose, motor fuel, synthetic chemical industry.
• Role in the body: turns into phenylpropanol and phenylpropionic acid.
• Elimination: through unchanged exhaled air and in the form of phenols in the urine.
• Mechanism of action: has a higher narcotic effect than toluene and benzene.
• Accumulation: in the liver, CNS, endocrine glands.
• Acute intoxication: in high concentrations causes acute intoxication with narcosis that installs slowly and has a long duration. Liver damage is possible after acute intoxications.
• Chronic intoxication: can cause dermatitis, hepatitis, kidney damage.
• Since 2014 it has been included in the list of carcinogens.

33. Naphthalene C10H8, characterized?
• Water-insoluble, colorless crystals with a characteristic odor.
• Professions: it is used in the chemical industry, in wood preservation and as an insecticide against moths.
• Naphthalene is mainly used in the synthesis of thinners, dyes or adhesives in the plastics industry such as PVC, in the development of insecticides from the carbamate group, as well as in the manufacture of soaps.
• It has an irritating effect on the mucous membranes and skin. Inhalation of vapors or accidental consumption of crystals causes nausea, vomiting, tenesmus, diarrhea, chills, fever, tachycardia and hypotension, hematuria. In severe cases, coma and death. Kidney damage is possible, even causing uremia.
• Differential diagnosis: compared to intoxications with nitro- or aminoderivatives of benzene.
• Treatment: gastric lavage with paraffin oil, transfusions, in case of artificial renal-renal lesions.
• Contraindicated: foods containing fats, milk and castor oil in the first aid.
• C.M.A .: 40 mg / m3.

34. From the group Benzene Nitroderivatives - Nitrobenzene C6H5NO2, do you list the characteristics?
• It is an oily, yellowish liquid with an almond smell.
• Occupations at risk of exposure: intermediate in the synthesis of aniline and benzidine, cosmetic industry, constituent of boot cream, synthetic chemical industry. To a lesser extent it is used as a diluent in obtaining ointments, fuels, photographic films or explosives. In the past it was used as a flavoring in soaps, today it is forbidden to use it in the manufacture of cosmetics.
• Routes of entry: respiratory, through the skin.

• Role in the body: turns into phenols.
• Elimination: through phenols in the urine. Mechanism of action: methemoglobinizing, causes redox disorders, is toxic to the SN, hepatotoxic and nephropathic.
• Acute intoxication: cyanosis and dipnea. After high-dose exposure, circulatory failure, collapse.
• Chronic intoxication: the characteristic signs are cyanosis, anemia (methemoglobinemia) and toxic hepatitis with splenomegaly, kidney damage. It is also associated with gastrointestinal symptoms: nausea, vomiting, rarely causes toxic pneumonia. Dermatitis occurs frequently.
• Diagnosis: exposure, symptoms, is confirmed by the present methemoglobinemia and phenols in the urine.
• Prognosis: favorable.
• Treatment: in acute intoxication: oxygen, glucose infusions, vitamin C, methylene blue i.v., 1% nicotinic acid.
• C.M.A .: 6 mg / m3.

35. What is characteristic of the group Aminoderivatives of benzene Aniline (phenylamine, aminobenzene)?
• Oily liquid widely used in chemical synthesis industry, pharmaceutical industry, paint industry, varnishes. It oxidizes in the air and becomes closed.
• Route of penetration: skin, gastrointestinal and respiratory.
• Mechanism of action: it is a strong methemoglobinizer.
• Acute intoxications: latency 2-3 hours, there is a cyanosis, fatigue, dyspnea, so phenomena caused by methemoglobin.
• Prognosis: favorable.
• Treatment: see nitrobenzene.

36. Benzidine, list characteristics:
• Used in: organic synthesis; manufacture of paints, especially Congo red, detection of blood stains (Gregersen reaction), dye in microscopy, analytical reagent for Pb, Ce, Pt, W, hardening agent in the preparation of rubber, Used in chemical and medical laboratories, enters body, primarily through the skin.
• After prolonged exposure, it may cause bladder and uterine cancer. Naphthylamine - used in the manufacture of dyes, Aminophenol - used in the manufacture of rubber, Auramine - made of dimethylaniline and formaldehyde, used in the coloring of paper and as an antiseptic, have a similar carcinogenic effect.

37. Aliphatic halogenated hydrocarbons - Dichloroethane (ethylene chloride):
• Colorless, chloroform-like liquid.
• Professions: solvent for greases, resins, paraffins, cellulose, rubber, disinfectant, insecticide.
• Route of penetration: respiratory and through the skin.
• Role in the body: turns into glycol and oxalic acid.
• Elimination: through exhaled air and urine.
• Mechanism of action: irritant effect on the respiratory tract and nephrotoxic due to oxalic acid metabolites.
• Acute intoxication occurs in three phases:

• I- prenarcosis, nausea, vomiting, abdominal pain, anesthesia
• II- latency time- oligosymptomatic lasting several hours
• III- signs of toxic hepatitis, signs of toxic nephrosis with proteinuria, oliguria and rarely pulmonary edema.
• Chronic intoxication: is characterized by digestive disorders, polyneuritis, astheno-vegetative symptoms, in severe cases - toxic encephalopathy.
• Complications: anuria, uremia.
• Diagnosis: 1) exposure and 2) symptoms.
• Treatment: D.M.P., symptomatic, in case of artificial kidney-kidney lesions.
• Prophylaxis: C.M.A. 150mg / m3, periodic medical check-up.

38. Trichlorethylene (CHCl = CCl2)?
• Colorless liquid, with aromatic odor, insoluble in water. It evaporates at room temperature and decomposes under intense light, producing hydrochloric acid and phosphogen.
• It is non-flammable.
• It is used in countless industries, among which we can mention: the rubber industry, in the footwear industry, in the vulcanization process, in the cleaning of clothing, in the chemical industry.

39. What is the role in the body?
• It enters the body in the form of vapors, through the respiratory tract, accidentally through the digestive tract.
• By contact with the skin it has an irritating effect, transcutaneous absorption is also possible.
• ¾ of the inhaled amount is retained in the body, the rest is eliminated by respiration.
• In the body it turns into hydrochloric acid, followed by trichloroacetic acid and trichloroethanol.
• Both metabolites are slowly excreted in the urine.

40. What is the mechanism of action?
• Locally acts as an irritant on the skin and mucous membranes of the airways. By drying the skin leads to microtrauma and secondary infection.
• It has a narcotic and euphoric effect; in long-term exposed workers have been described neurological and psychological changes (Grandjean), is hepatotoxic and nephrotoxic.

41. What is characteristic of acute intoxication?
• It begins with phenomena of anesthesia, respectively prenacosis, rarely with euphoria, more frequently with nausea, vomiting, dizziness and symptoms of irritation of the upper respiratory tract.
• In the case of high doses it can lead to loss of consciousness: death can occur due to paralysis of the bulbar respiratory center or due to heart failure (ventricular fibrillation).

42. Explain what characterizes chronic intoxication?
• It is installed after a longer exposure with neurovegetative disorders: sleep disorders, headache, asthenia, irascibility, paresthesias, alcohol intolerance, decreased sexual potency; with lesions of the nervous system: materialized by gait disorders, altered reflexes, characteristic of TRIGEMENE NEURITIS, rarely optic neuritis, with signs of toxic encephalopathy.
• Prolonged exposure can lead to addiction; drug addiction leading to a series of serious morphofunctional changes. Ventricular fibrillation is thought to occur due to the hypersympathototonizing action of the toxicant. Euphoria in drug addicts can lead to serious accidents.

43. What is the diagnosis based on?

• It is based on occupational or accidental exposure, on characteristic clinical signs and is confirmed by the presence of toxic metabolites in urine, trichloroacetic acid above a limit of 200mg / l in urine.

44. What is the prognosis?

• It is generally favorable in acute intoxications if a quality first aid is ensured over time.

• Complications are related to damage to the trigeminal nerve. In chronic intoxications the prognosis is also good in the case of appropriate treatment.

45. What is the treatment tactic?

• In acute intoxications: with oxygen, artificial respiration, if necessary, corticosteroids, vitamin infusions. Adrenaline, noradrenaline, ephedrine are contraindicated.

• In chronic intoxications - treatment with corticosteroids, vitamins (B1, B6, infusions) and symptomatic treatment. Adequate diet in case of liver or kidney damage.

46. What is prophylaxis:

• It is recommended to seal the technological processes where the trichlorethylene evaporates, ensuring a local suction ventilation.

• Workers will be monitored during the regular check on toxic addiction.

47. The contraindications to this intoxication are:

• Obesity, alcoholism, lung disease, hypertension, gastric and duodenal ulcers, liver, skin, kidney, peripheral nerve and people who are sensitive to the toxic.

• C.M.A. 300mg / m3.

TESTS FOR KNOWLEDGE VERIFICATION:

1. C.M What are arenas?
[*] arenas are substances composed of carbon and hydrogen in the structure of which one or more benzene nuclei appear.
[*] are also called aromatic hydrocarbons, containing in their molecule one or more rings of 6 carbon atoms.
[*] when the arena molecule consists of a single ring they are called mononuclear arenas, and when the molecule comprises two or more cycles they are called polynuclear arenas.
[] arenas are substances composed of nitrogen and hydrogen.
[] no benzene nucleus appears in their structure.

2. C.M What is the chemical structure and what is the importance of benzene?
[*] The 6 C atoms in the molecule are joined in a ring by 3 double bonds that alternate with 3 single bonds.
[*] As a gasoline additive, benzene increases its octane number and reduces detonation.
[*] Consequently, it often contained significant amounts of benzene before the 1950s, when lead tetraethyl was introduced as an antidetonator.
[*] In recent years, as a result of declining lead gasoline production, benzene has been reintroduced as an additive.
[] In the USA, due to the negative effect on health and to reduce the risk of groundwater pollution with this substance, a maximum allowable emission of approximately 5% of benzene was imposed.

3. C.M What is the chemical structure and what is the importance of benzene?
[] the 5 C atoms in the molecule are joined in a ring by 3 double bonds that alternate with 3 single bonds.
[*] As a gasoline additive, benzene increases its octane number and reduces detonation.
[*] Consequently, it often contained significant amounts of benzene before the 1950s, when lead tetraethyl was introduced as an antidetonator.
[*] In recent years, as a result of declining lead gasoline production, benzene has been reintroduced as an additive.
[*] In the USA, due to the negative effect on health and to reduce the risk of groundwater pollution with this substance, a maximum permissible emission of approximately 1% of benzene was imposed.

4. C.M Chemical compounds obtained by replacing one or more benzene hydrogen atoms with other functional groups, namely compounds obtained by substitution with an alkyl group which are:
[*] toluene C6H5-CH3
[*] ethylbenzene C6H5-CH2CH3
[*] xylene C6H4 (-CH3) 2
[*] mesethylene C6H3 (-CH3) 3
[] salicylic acid C6H4 (-OH) (- COOH)

5. C.M What are the compounds obtained (replacement of one or more hydrogen atoms of benzene) by substitution with other groups:

[] phenol C8H5-ON
[*] C6H5-NH2 aniline
[*] chlorobenzene C6H5-Cl
[*] C6H5-NO2 nitrobenzene
[*] picric acid C6H2 (-OH) (-NO2) 3

6. C.M What are the compounds obtained by substituting one or more benzene hydrogen atoms:
[*] trinitrotoluene C6H2 (-CH3) (-NO2) 3
[*] benzoic acid C6H5-COOH
[*] salicylic acid C6H4 (-OH) (- COOH)
[*] acetylsalicylic acid C6H4 (-O-C (= O) -CH3) (- COOH)
[] xylene C6H4 (-CH3) 2

7. C.M Compounds obtained (replacement of one or more benzene hydrogen atoms) by substitution with other groups are:
[*] acetylsalicylic acid C6H4 (-O-C (= O) -CH3) (- COOH)
[*] paracetamol C6H4 (-NH-C (= O) -CH3) -1 - (- OH) -4
[*] phenacetin C6H4 (-NH-C (= O) -CH3) (- O-CH2-CH3)
[] phenol C6H5-OH
[] C6H5-NH2 aniline

8. C.M Compounds with two or more benzene rings are:
[*] mothballs
[*] anthracite
[*] indol
[*] benzofuran
[] aniline

9. C.S Compounds with two or more benzene rings are:
[*] benzofuran
[] trinitrotoluene C6H2 (-CH3) (-NO2) 3
[] benzoic acid C6H5-COOH
[] salicylic acid C6H4 (-OH) (- COOH)
[] acetylsalicylic acid C6H4 (-O-C (= O) -CH3) (- COOH)

10. C.S Benzene, what is specific?
[] The simplest combination of aromatic hydrocarbons is 6.7 times heavier than air.
[] Color liquid
[] Boiling point 50 C
[*] It is easily soluble in water
[] Very insoluble in fat

11. C.M Benzene, what is specific?
[*] The simplest combination of aromatic hydrocarbons is 2.7 times heavier than air.
[*] Colorless liquid
[*] Boiling point 80 C
[] Slightly insoluble in water
[*] Very soluble in fats

12. C.M What are the professions at high risk of exposure to benzene poisoning?
[*] Dry distillation of coal
[*] Oil refineries
[*] Synthetic chemical industry
[*] Pharmaceutical industry
[] Pastry

13. C.M What are the professions at high risk of exposure to benzene poisoning?
[*] Dye industry
[*] Explosives industry
[*] Rubber industry (solvent)
[*] Solvent for paints, varnishes
[ ] Cake shop

14. C.M What are the professions at high risk of exposure to benzene poisoning?
[*] Plastics industry (solvent)
[] Food
[ ] Construction
[*] Fat extraction
[*] Degreasing in metallurgy, leather, textile, footwear industry.

15. C.M What are the professions at high risk of exposure to benzene poisoning?
[*] Printing houses
[*] Wood industry
[*] Manufacture and use of soldering solutions
[*] Leather processing (solvent for paints, gluing).
[ ] Public Alimentation

16. C.M What are the pathways of benzene poisoning?
[*] Respiratory
[*] Digestive
[*] Through the skin.
[] Bloody
[] Contact

17. C.M Benzene is obtained from which compounds?
[*] Benzene is obtained from carbon-rich compounds that suffer from incomplete combustion.
[*] Obtained naturally from volcanoes and forest fires, being present in many other combustion products including cigarette smoke.
[*] Until World War II, significant quantities of benzene were a by-product in the production of coke used in the iron industry.
[*] In the 1950s, the demand for benzene increased substantially, especially in the plastics industry, thus necessitating its much more productive extraction from oil.
[] Benzene is obtained from carbon-rich compounds that undergo complete combustion.

18. C.S Benzene is obtained from which compounds?
[*] At present, benzene is mainly obtained in the petrochemical industry, as the production of the coal compound is very little used.
[] Benzene is obtained from carbon-rich compounds that undergo complete combustion.

[] It is obtained artificially from volcanoes and forest fires, being present in many other combustion products including cigarette smoke.
[] Until World War II, significant quantities of benzene were the main product in the production of coke used in the iron industry.
[] In the 1950s, the demand for benzene increased insignificantly, especially in the plastics industry.
19. C.M What are the four chemical processes in industrial benzene production?
[*] catalytic reforming
[*] hydrodesalkylation of toluene
[*] disproportionation of toluene
[*] steam cracking.
[] toluene proportion
20. C.M What are the states where benzene came from?
[*] In 1996, it was 33 million tonnes, of which 7 million came from the United States
[*] 6.5 million from the European Union
[*] 4.2 from Japan
[*] 1.4 million from South Korea
[] two million from China.
21. C.S What are the states where benzene came from?
[] 7.5 million from the European Union
[] 10.2 from Japan
[] 5.4 million from South Korea
[] Three million from China.
[*] Romania is a country that produces, consumes and exports benzene, the most important refineries where benzene is manufactured are Rafo Onești, Oltchim S.A. and Rompetrol.

22. C.M Before the 1920s, for what purpose was benzene used?
[*] Benzene was frequently used as an industrial solvent, mainly for degreasing metals but due to its high toxicity it was replaced with other solvents.
[*] Its main use is as an intermediate reagent for the synthesis of other chemical compounds.
[*] Benzene derivatives that are produced in significant quantities are styrene, used in the manufacture of polymers and plastics, phenol, from which resins and adhesives are prepared, cyclohexane, used for the preparation of nylon.
[*] Smaller quantities of benzene are used in the manufacture of tires, lubricants, dyes, detergents, medicines, explosives or pesticides.
[] Larger quantities of benzene are used in the manufacture of tires, lubricants, dyes, detergents, medicines, explosives or pesticides.
23. C.M In the 1980s, what were the main compounds obtained from benzene listed?
[] cumen 16%
[*] cyclohexane 15%
[*] 7% nitrobenzene.
[*] As a gasoline additive, benzene increases its octane number and reduces detonation.
[*] Consequently, it often contained significant amounts of benzene before the 1950s, when lead tetraethyl was introduced as an anti-knock agent.
24. C.M. In the 1980s, the main compounds obtained from benzene which were, listed:
[*] ethylbenzene, using 48% benzene in the process
[*] In recent years, as a result of declining lead gasoline production, benzene has been reintroduced as an additive.
[*] In the United States, due to the negative effect on health and the reduction of the risk of groundwater contamination with this substance, a maximum permissible emission of approximately 1% benzene was imposed.

[*] The same figure is found in European Union standards.
[] ethylbenzene, in the process using 4.9% benzene.

25. C.M The role of benzene in the human body?
[*] in the blood is transported by lipoproteins and accumulates in high-fat tissues, especially in the hematoforming marrow and CNS, adrenal glands.
[*] It undergoes metabolic transformations in the body and is transformed into phenols, pyrocatechins, hydroquinone (Nomyama) and degrades even to carbon dioxide. By opening the benzene nucleus, muconic acid is also produced, it can be condensed with cysteine, by acetylating its NH2 radical and 1-phenyl-mercapturic acid is formed.
[*] Phenols conjugate with glucuronic acid and sulfates, appear in the urine as ether sulfates and glucuroconjugates.
[] Phenols do not conjugate with glucuronic acid and sulfates, they appear in the urine as ether sulfates and glucuroconjugates.
[] in the blood is carried by lipoproteins and does not accumulate in high-fat tissues, especially in the hematoforming marrow and CNS, adrenal glands.
26. C.M How does benzene be eliminated from the human body?
[*] Through the respiratory tract (30-75%) on average 50% of the inhaled, through urine in the form of phenols and diphenols; pyrocatechol, hydroquinone and phenyl-mercapturic acid.
[*] Lim. fiz. sup. for total phenols: 130mg / l
[] Lim. fiz. sup. for total phenols: 120mg / l
[] Lim. fiz. sup. for total phenols: 170mg / l
[] Through the respiratory tract (30-95%) on average 40% of the inhaled, through urine in the form of phenols and diphenols; pyrocatechol, hydroquinone and phenyl-mercapturic acid.
27. C.M Explain the mechanism of action of benzene?
[*] Toxic to mitosis; inhibitory action on medullary cells, enzymatic disorders of mitosis, on young forms - on granulocyte, erythrocyte, platelet series.
[*] Causes disorders in the synthesis of corticosteroids, hypovitaminosis B2, C, B6, PP, toxic endothelial damage, enzymatic disorders: catalase, peroxidase, phosphatase; decreases the phagocytic reaction. It is hepatotoxic. It has a narcotic effect.
[] Toxic to mitosis; non-inhibitory action on medullary cells, enzymatic disorders of mitosis, on young forms - on granulocyte, erythrocyte, platelet series.
[] Causes insignificant disorders in the synthesis of corticosteroids, hypovitaminosis B12, B1, C, B6.
[*] Enzymatic disorders: catalase, peroxidase, phosphatase

28. C.M What are the symptoms of acute benzene poisoning?
[*] begins with dizziness
[*] went unsafe
[*] state of euphoria
[*] drowsiness
[] upper digestive hemorrhage
29. C.M What are the symptoms of acute benzene intoxication?
[*] headache
[] intestinal bleeding
[*] anesthesia
[*] areflexie
[*] vasomotor paralysis
30. C.M What are the symptoms of acute benzene poisoning?

[*] heart attack
[*] convulsions
[*] the death
[] gingival hemorrhages
[] tremor.
31. C.M What are the complications in acute benzene poisoning?
[*] In the first phase, serious complications are cerebral and parenchymal hemorrhages, followed by neuropsychiatric disorders, toxic hepatitis, kidney damage and haematological changes.
[] Prognosis - favorable.
[*] The disease begins slowly, insidiously, in the first phase, in latent benzeneism, usually without characteristic, obvious symptoms.
[*] In the first phase of the disease there is macrocytosis, hyperchromia, (Manu P.) a transient leukocytosis, (Timar M.) a net hyperplasia of the reticulo-histiocytic system (Hilt, Manu) and chromosomal abnormalities (Manu, Popescu).
[*] In this phase a transient increase of the phagocytic reaction and a transient catalase hyperactivity (Dienes) is observed.
32. C.M What is characteristic of chronic benzene poisoning?
[*] The leukopenicizing action of benzene, alteration of the white series, erythrocytes and platelets, occur gradually, successively, simultaneously or in isolation.
[*] At this stage, astheno-vegetative symptoms are observed and anemia becomes more and more obvious.
[*] Bleeding occurs, hepatosplenomegaly is associated, signs of toxic hepatitis.
[*] In untreated cases there are intercurrent infections, superinfected ulcers. Acute or chronic leukemias have been described in chronic benzene poisoning.
[] Toxic encephalopathies occurred only in mild cases.

33. C.M What is the prognosis of benzene poisoning?
[*] in all cases is reserved
[*] grav
[*] the toxicant going in severe cases until the complete destruction of the bone marrow.
[] favorable
[*] unfavorable
34. C.M What is the diagnosis of benzene poisoning?
[*] Based on exposure, characteristic clinical and laboratory symptoms (leukopenia, myelocytes, promyelocytes) and phenols (over 130mg / l);
[*] decrease in urine ratio = inorganic sulfur / total sulfur below 0.8;
[*] haematological changes
[*] Roetter sample 1 (delayed skin discoloration more than 10-15 min after intradermal injection of 2mg dichlorophenol-indophenol solution in 4.9 cm water, 0.1 ml)
Rumpel-Leede test
[] decrease in urine ratio = inorganic sulfur / total sulfur below 0.6;
35. C.M Differential diagnosis of benzene poisoning is made with:
[*] intoxications with organic solvents with narcotic action
[*] diseases of the hematopoietic system.
[] diseases of the musculoskeletal system
[] immune diseases
[] parasitic diseases
36. C.S In the treatment of acute benzene intoxication what is contraindicated to administer?
[*] adrenaline
[] analeptics

[] glucose infusions
[] procainamide (in case of extrasystole)
[] gluconic calcium.
37. C.M What includes the treatment of acute benzene poisoning:
[*] corticosteroids
[*] antibiotics
[*] oxygen therapy
[*] combating hemorrhages.
[] adrenaline
38. C.M Chronic benzene intoxication, treatment components:
[*] C vitamin
[*] B vitamins
[*] sodium hyposulphite
[*] transfusions
[] plasma
39. C.M Chronic benzene intoxication, treatment components:
[*] corticosteroids
[*] antibiotics
[*] bone marrow transplant
[] asymptomatic treatment
[*] glucose infusions
40. C.M What is the prophylaxis of benzene intoxication?
[*] of early importance is the early diagnosis of the disease, in the phase of latent benzeneism.
[*] technical
[*] individual- in protective clothing, masks, shower, technical training.
[*] medical - urine examination (for haematuria), reactive test with dichlorophenol solution, alcohol test, alkaline leukocyte phosphatase (FAL), red blood cell count, hemoglobin, leukocyte count, leukocyte formula, reticulocytes, platelets, before and after arousal medullary, bleeding time, coagulation time, dysproteinemia tests, Rumpel-Leede test, Sulfate-index, urine phenols, myelogram (in the presence of quasi-specific changes in peripheral blood), clot retractability.
[] secondary control.

41. C.M Contraindications to employment in relation to possible benzene poisoning?
[*] Congenital or acquired haematological diseases, affecting the red, leukocyte, platelet series, hemorrhagic syndromes, liver diseases affecting the parenchyma, small and repeated bleeding (hemorrhoids, menometrorrhagia, etc.)
[*] operated stomach (resected)
[*] significant exposure to other medullotropic toxins or ionizing radiation in the past (in the last 5 years).
[*] C.M.A. 50mg / m3.
[] C.M.A. 30 mg / m3.
42. C.M What are benzene derivatives?
[*] Toluene (methylbenzene C6H5CH3)
[*] Xylene (dimethylbenzene) C 6 H 4 (CH 3) 2
[*] Styrene (vinylbenzene) C6H5CH = CH2
[*] Ethylbenzene C6H5C2H5
[] Benzidine
43. C.M What are benzene derivatives?
[*] Cumene (isopropylbenzene) C6H5CH (CH3) 2
[*] Naphthalene C10H8

[] Aniline (phenylamine, aminobenzene)
[] Benzidine
[] Dichloroethane (ethylene chloride)
44. C.S Benzene nitroderivatives are:
[*] Nitrobenzene C6H5NO
[] Toluene (methylbenzene C6H5CH3)
[] Xylene (dimethylbenzene) C 6 H 4 (CH 3) 2
[] Styrene (vinylbenzene) C6H5CH = CH2
[] Ethylbenzene C6H5C2H5
45. C.M Aminoderivatives of benzene are:
[*] Aniline (phenylamine, aminobenzene)
[*] Benzidine
[] Ethylbenzene C6H5C2H5
[] Cumene (isopropylbenzene) C6H5CH (CH3) 2
[] Naphthalene C10H8
46. C.M Aliphatic halogenated hydrocarbons are:
[*] Dichloroethane (ethylene chloride)
[*] Trichlorethylene (CHCl = CCl2)
[] Toluene (methylbenzene C6H5CH3)
[] Xylene (dimethylbenzene) C 6 H 4 (CH 3) 2
[] Styrene (vinylbenzene) C6H5CH = CH2

47. C.M Characteristics of Toluene (methylbenzene C6H5CH3):
[*] Higher volatility than benzene
[*] Professions: benzene associated as aviation fuel.
[*] Mechanism of action: does not cause haematological changes.
[*] Role in the body: in the body it is converted into benzoic acid which is conjugated with glycochol and is eliminated as hippuric acid.
[] Elimination: upper physiological limit of hippuric acid: 0.6g / urine of 24; by exhaled air 15-16%.
48. C.M Characteristics of Toluene (methylbenzene C6H5CH3):
[*] Elimination: upper physiological limit of hippuric acid: 0.7g / urine of 24; by expired air 18-20%
[*] Acute intoxication: begins with euphoria, followed by drowsiness and narcosis.
[*] Chronic intoxication: characterized by astheno-vegetative symptoms and irritation of the respiratory tract and conjunctiva, toxic hepatitis, minor-moderate kidney damage.
[] Lower volatility than benzene
[] Professions: pastry.
49. C.M The diagnosis of toluene poisoning (methylbenzene C6H5CH3) is determined by the following components:
[*] symptoms
[*] exposure
[*] hippuric acid 1 g / l of urine
[] arterial hemorrhage
[] bone tumors
50. C.M What is characteristic of Xylene (dimethylbenzene) C6H4 (CH3) 2?
[*] Role in the body: it turns into toluic acid.
[*] Mechanism of action: inhibits the function of the hematoforming marrow, effect on erythrocyte, platelet and leukocyte series.
[*] Acute intoxication: euphoria, drowsiness, anesthesia.
[*] Chronic intoxication: dermatitis, eczema, conjunctivitis, respiratory tract irritation, anemia,

leukopenia, thrombocytopenia, dyspepsia, neuro-vegetative dysfunction.
[] Melena on display