MINISTRY OF HEALTH OF THE REPUBLIC OF BELARUS
INSTITUTION OF EDUCATION
"GOMEL STATE MEDICAL UNIVERSITY"

Department of Pathological Anatomy

FORENSIC MEDICAL EXPERTISE OF POISONING. FORENSIC EXPERTISE OF DEATH FROM MECHANICAL ASPFIXIA.

Educational-methodical recommendation
for 5th year students of medical and FIS faculties
in the discipline "Forensic medicine"

Author:
assistant Shpak V.V.

Gomel, 2022

FORENSIC MEDICAL EXPERTISE OF POISONING. FORENSIC EXPERTISE OF DEATH FROM MECHANICAL ASPFIXIA.

(total lesson time - 6 academic hours).

RELEVANCE OF THE TOPIC

Materials of topics are a necessary prerequisite for understanding the patterns of development and formation of the relationship and interdependence of pathophysiological and pathomorphological changes as a result of poisoning, morphological features of mechanical asphyxia. It is also necessary in the future professional activity of a doctor, for clinical diagnosis and analysis of the sources of diagnostic errors in clinical practice.

THE OBJECTIVES OF THE LESSON

To study the basic methods of determining poisoning with various substances. To form a concept about the poison and the changes caused by it in the body, the conditions of the course of poisoning. To master the classification of mechanical asphyxia, general asphyxia signs and diagnosis of death from mechanical asphyxia. To study the basic principles of conducting a forensic medical examination. To get acquainted with the peculiarities of the forensic medical examination of the corpse at the place of its discovery (in case of suspicion of poisoning), the procedure for seizing and sending the cadaveric material for forensic histological and forensic chemical research.

TASKS

1. Give a forensic classification of poisoning.
2. Show the basic principles of forensic diagnostics of poisoning.
3. To be able to distinguish between the principles and possibilities of examination in case of suspected poisoning.
4. Be able to explain the rules for the production of forensic biological, forensic histological, medico-forensic and forensic chemical examinations.
5. To be able to answer the questions resolved in the examination of poisoning.
6. Give a definition of the concepts "asphyxia", "mechanical asphyxia", "compression asphyxia".
7. Be able to distinguish between normative documents governing the production of expert examinations of material evidence.

KEY LEARNING QUESTIONS

1. The concept of poisons. Forensic classification of poisons.
2. Poisoning, their origin. The dependence of the course of poisoning on the properties of poisons, the conditions for their introduction and action, as well as the individual characteristics of the human body and the environment. Addiction to poisons. The course of the poisoning.
3. Drug addiction and substance abuse.
4. Features of the inspection of the scene of the suspected death from poisoning.
5. Forensic recognition of poisoning.
6. Questions to be resolved in the examination of poisoning.
7. The main stages of the forensic medical examination of poisoning.
8. Expert assessment of the results of forensic chemical analysis.

9. Characteristics of fatal poisoning: acids and alkalis; ethylene glycol, dichloroethane and other technical fluids; organophosphorus and other pesticides; poisons of general functional action; medicines. Poisoning with ethyl, methyl alcohol and alcohol-containing liquids.
10. The concept of hypoxia and mechanical asphyxia. Types and stages of mechanical asphyxia.
11. Strangulation asphyxia: hanging, strangulation with a noose, strangulation by hands.
12. Loops and their types, options for location on the neck.
13. Genesis of death when the neck is squeezed by a loop
14. Strangulation groove when hanging and strangled by a loop. Determination of the lifetime of the strangulation groove.
15. Forensic diagnosis of strangulation by hands.
16. Compression, obstructive asphyxia.
17. Drowning, its types, forensic diagnostics, laboratory research methods. Death in water, definition of the concept, main causes. Damage to corpses taken out of the water. Determination of the duration and signs of the stay of a corpse in the water.
18. Death in a confined space with a lack of oxygen.

MATERIALS FOR CONTROL OF THE TOPIC ASSEMBLY

Terminology

Poison balance - the ratio of the processes of absorption and excretion of poison from the body.
Dose - the amount of poison, expressed in certain weight or volume units.
Indifferent dose - the minimum amount of poison that does not cause any disturbances in the life of the body.
The dose is toxic - the minimum amount of a substance incompatible with the normal functioning of the body, i.e. causing painful changes.
Lethal dose- the minimum amount of a substance incompatible with life, i.e. causing the death of a person.
Zone of toxic action - the range from indifferent to lethal dose.
Concentration - the degree of dilution of the substance in various media (in the blood or in an organ susceptible to the action of this poison).
Material cumulation - the accumulation of poison in the body during its repeated receptions at short intervals.
Functional cumulation - the accumulation (summation) of not the poison itself, but the effect of its action.
Poisoning (intoxication) - a pathological state of the body that develops when a poisonous substance enters it, and is characterized by functional and morphological disorders.
Forensic Toxicology - the science of poisonous substances and the poisoning caused by them.
Poison - This is a substance that, when introduced into the body from outside and in small quantities, is capable, under certain conditions, of causing chemical or physicochemical changes leading to illness or death.
Destructive poisons - a group of toxic substances, the toxic effect of which is associated with a violation of the morphological structure of organs and tissues.
Poisons are caustic - Poisonous substances with a predominantly pronounced effect in the area of primary contact with tissues.
Concentration poisons - poisons, the strength of which is not associated with time, but is mainly due to their concentration.

Resorptive poisons - Poisonous substances, the toxic effect of which is manifested only after absorption.
Functional poisons - a group of toxic substances, the toxic effect of which is not accompanied by morphological changes in organs and tissues.
Chronoconcentration poisons - poisons, the intensity of the toxic effect of which depends both on their concentration and on the time during which they acted.
Asphyxia - an acute pathological process that occurs due to a lack of oxygen in the blood and tissues (hypoxia and hypoxemia) and the accumulation of carbon dioxide (hypercapnia) and is characterized by a severe symptom complex of disorders from the central nervous system, CVS and respiratory organs.
Asphyxia aspiration - a type of mechanical asphyxia, characterized by complete or partial closure of the airways by liquid and loose substances.
Compression asphyxia - a kind of mechanical asphyxia, characterized by compression of the chest and / or abdomen with massive blunt objects.
Mechanical asphyxia - violation of external respiration under the influence of mechanical factors.
Obstructive asphyxia - a type of mechanical asphyxia, characterized by complete or partial closure of the airways by soft and hard foreign objects.
Positional asphyxia - mechanical asphyxia as a result of giving the body a posture that impedes external breathing.
Strangulation asphyxia - a type of mechanical asphyxia, characterized by compression of the neck organocomplex under the influence of body gravity or external force.
Strangulation furrow - abrasion of the skin of varying depth, resulting from dynamic contact with the loop material and representing its negative trace.
Hypoxia - oxygen starvation of organs and tissues until the complete cessation of oxygen supply to the body.
Diatom plankton - unicellular microscopic organisms, ranging in size from 4 to 2000 microns, inhabiting various water bodies and having an organosilicon structure.
Lymphogemia - retrograde throwing of erythrocytes into the lymphatic thoracic duct.
Maceration - softening, loosening and swelling of the skin due to its impregnation with liquid.
Hanging - compression of the organs of the neck by a loop under the influence of the gravity of the whole body or its parts, sometimes between objects or by pressing.
Paltauf spots- vague hemorrhages with indistinct contours of light red color, up to 1-1.5 cm in diameter under the pulmonary pleura. They are a sign of lifetime drowning in fresh water.
Tardier spots- clearly delimited multiple small-point (2-3 mm in diameter), intense dark red, hemorrhages under the interlobar and diaphragmatic pleura and on the posterior surface of the heart under the epicardium, formed as a result of capillary rupture with increased pressure in the pulmonary circulation. They are a sign of rapid death, often found in various types of mechanical asphyxia.

FORENSIC MEDICAL EXPERTISE OF POISONING.

Forensic Toxicology- the science of poisonous substances and the poisoning caused by them. She studies the chemical and physical properties of poisons, their effect on the body and develops methods for the quantitative and qualitative determination of poisons in the environment and the body.

Poison - This is a substance that, when introduced into the body from outside and in small quantities, is capable, under certain conditions, of causing chemical or physicochemical changes leading to illness or death.

The subject of forensic toxicology studies is poisoning for the purpose of murder, suicide or poisoning as a result of an accident.

Poisoning or intoxication - a pathological state of the body that develops when a poisonous substance enters it, and is characterized by functional and morphological disorders.

Conditions for the action of poisons on the body

1. **Dose of the injected substance...**

Dose is the amount of poison expressed in specific weight or volume units. It must be incompatible with the normal functioning of the body. This is the main condition for the action of the poison. The severity of morphofunctional changes in the overwhelming majority of cases is in direct proportion to the dose of the administered substance.

Disruption of normal life is understood as various manifestations of the toxic effect - from minor functional disorders to death. For all poisons, there are indifferent, therapeutic, toxic, lethal and toxic-lethal doses.

Indifferent dose - the minimum amount of poison that does not cause any disturbances in the life of the body.

Therapeutic dose - the amount of poison that causes a healing effect.

Toxic dose - the minimum amount of a substance incompatible with the normal functioning of the body, i.e. causing painful changes.

Lethal dose- the minimum amount of a substance incompatible with life, i.e. causing the death of a person.

For some poisons it is very difficult to distinguish between poisonous doses and lethal ones; in these cases, the definition applies: toxic-lethal doses.

The size of the so-called zone of toxic action is of great importance, i.e. range from indifferent to lethal dose. The larger it is, the safer the substance, and vice versa.

2. The concentration of the poison in the body.

Concentration- the degree of dilution of the substance in various media (in the blood or in an organ susceptible to the action of this poison). The higher the concentration of the toxic substance, the more pronounced the toxic effect associated with it. The concentration of the poison largely depends on the routes of its introduction into the body.

In the development of poisoning, a very large role belongs to the ratio of the processes of absorption and removal of poison from the body, i.e. balance of poison**... So if the poison is absorbed slowly and excreted from the body quickly, then even the lethal concentration in the blood may not be enough to cause a fatal outcome. If the amount of isolated and neutralized poison is less than that received over the same period of time, conditions are created for its accumulation. The accumulation of poison in the body during repeated administrations at short intervals is called material cumulation. As a result, there is a summation of inactive amounts of poison to a certain toxic concentration. In cases where there is an accumulation (summation) not of the poison itself, but of the effect of its action, there is a functional cumulation or cumulation of action.**

The amount of poison that has entered the body in one way or another does not remain constant in it, not only due to elimination, but also as a result of biotransformation - various kinds of chemical transformations: neutralization, oxidation, reduction, deamination, etc. Thus, the poisonous substance changes in that or to another degree and is excreted from the body in

the form of secondary metabolites, the toxic effect of which may be more pronounced than the effect of the poison itself.

Along with the concentration of the poison, the strength of the toxic effect is largely determined by the time factor of its exposure.

In this regard, poisons are divided into two groups. One group includes poisons, the intensity of the toxic effect of which depends both on their concentration and on the time during which they acted. Such poisons are called chronoconcentration; these include substances that have an irritating effect on the metabolism, especially enzymes.

The second group includes poisons, the strength of which is not associated with time, but is mainly due to their concentration. These are the so-called concentration poisons. These include most of the substances acting on the nervous system.

3. Chemical and physical properties of the poisonous substance.

The chemical structure of toxic substances is extremely diverse. Noteworthy is the fact that substances are very similar in structure, can have different toxic effects (cocaine and morphine, choline and neurin). Poisons, however, are completely different in chemical structure, can cause similar clinical symptoms of poisoning.

The main physical characteristics that determine the effectiveness of the action of poisons are solubility, state of aggregation of poisonous substances and combination with other substances.

***Solubility*... The poisonous substance must be soluble in water or lipids, or in the process of metabolism, form toxic compounds that are soluble in these media. The so-called distribution coefficient is important, i.e. the ratio of the solubility of a substance in fats to its solubility in water.**

The predominantly water-soluble compounds can dissolve either in the entire body fluid or only in the extracellular fluid. They are able to easily pass through cell membranes, changing the osmotic balance.

Many substances, poorly soluble in water and well soluble in lipids, have a pronounced effect on the central nervous system, which, as you know, consists mainly of phospholipids.

A poison that does not have the ability to dissolve in the environments of the body is not capable of causing poisoning.

State of aggregation. Poisonous substances are in solid, liquid and gaseous state; the degree and speed of absorption depend on one or another state of the poison. Solids taken internally, in crushed or even powdered form, act only after their dissolution, which leads to a delayed development of intoxication. Substances introduced inside in liquid form are absorbed much faster, because no time is wasted on their preliminary dissolution in liquid media of the body, and they immediately enter into reactions of interaction with the biochemical components of the tissues of the human body.

Gaseous and vaporous substances are especially dangerous due to their rapid penetration through the pulmonary alveoli.

Combination with other substances. All toxic substances in relation to each other can be either synergists or antagonists. If the effect of one substance is added to the effect of another, one speaks of a cumulative effect. This happens when two synergistic substances have the same point of application in the body. An example of synergy is the simultaneous ingestion of ether and alcohol. When a combination of two or more substances gives a significantly greater toxic effect than each of them separately, they speak of potentiating their action. This phenomenon is

observed when substances of different chemical groups with different points of application act on the body in one direction.

Antagonism can be chemical or physiological... In the first case, poisons neutralize each other or create low-toxic compounds (for example, hyposulfite converts cyanide groups into thiocyan compounds). In the case of physiological antagonism, poisons, without entering into a chemical reaction with each other, have the opposite toxic effect on the body (for example, strychnine excites those parts of the nervous system that are paralyzed by chloral hydrate). Antidote therapy methods are based on the principle of antagonism.

4. Ways of introducing the poison into the body.

Depending on the route of administration, the doses of the same poison are different for the manifestation of its toxic, therapeutic or lethal effect. So the therapeutic doses when the poison is introduced through the mouth can be lethal when it enters the bloodstream. Moreover, the amount of poison introduced into the body and remaining in it may be different. This, first of all, is observed when the poison is introduced through the mouth, when, together with the vomit, part of it, sometimes very significant, is expelled from the body.

In forensic practice, the most common way of introducing poisons into the body is oral. The rate of absorption of poisons from the gastrointestinal tract depends on a number of factors. These are the solubility in water and lipids (the latter are already absorbed in the oral cavity), the degree of filling the stomach with food masses, the reaction of the environment, the motility of the stomach and intestines, convection (mixing), the functional state of the gastric and intestinal epithelium, the state of blood circulation, and the barrier function of the liver. Most poisons reach their maximum concentration in the blood after the oral route of administration in 2-3 hours.

***Inhalation*the route of introduction of toxic substances in forensic medical practice is not common; nevertheless, it has some expert value. Cases of poisoning are known when cocaine is injected into the nasal cavity and from the application of anesthetics to the mucous membrane of the larynx and trachea.**

The mucous membrane of the respiratory tract and especially the huge surface of the pulmonary alveoli (60–90 sq. M.), The abundant network of lymphatic vessels in the lung tissue ensure rapid absorption of the poison into the blood. The action of poisons entering the body through the respiratory tract is usually more pronounced than when absorbed through the gastrointestinal mucosa, because when they enter the blood from the lungs, they bypass the liver.

Poisons enter the body by inhalation in the form of vapors, aerosols, in the form of the smallest dust-like particles and droplets in the air.

The speed and degree of entry of dusty poisons through the respiratory tract largely depends on the size of the particles suspended in the air. The finer the particles, the deeper they penetrate and the faster they are absorbed. The alveoli are most often reached by particles with a diameter of no more than 5 microns. Larger ones are retained in the bronchioles and bronchi. The mechanism of resorption of poisons from the alveoli can be different and depends on the physical state of the poison. Gaseous substances enter the bloodstream by ordinary diffusion. The absorption rate depends on the solubility, concentration, and the amount of pulmonary ventilation.

Transcutaneous (transcutaneous) routepenetration of toxic substances. The ability to penetrate through intact skin is possessed by poisons that are readily soluble in lipids (technical liquids), and also have a local irritant effect. The degree of absorption increases with skin

hyperemia and increased sweating. Different areas of the skin have different capacities for the absorption of toxic substances. Faster and easier absorption occurs in those areas that have a thinner epidermis (axillary and groin areas, anterior surface of the forearm). The intensity of the penetration of poisons through the skin increases when it is damaged. Poisons very quickly penetrate into the body when they get on the mucous membrane of the eyes, mouth, nasopharynx.

Parenteral route of administration. Distinguish between intravenous, subcutaneous and intramuscular as the most common. Poisons introduced by these routes very quickly reach their toxic or lethal concentration and act mainly unchanged, because firstly, they enter the bloodstream, bypassing the liver, and secondly, they simply do not have time to undergo any significant changes due to the rapid development of a toxic effect or death.

In forensic practice, there are cases of the introduction of toxic substances through the mucous membranes of the vagina or rectum, from where they are quickly transferred with the bloodstream into the systemic circulation, mainly bypassing the liver and, therefore, not being rendered harmless in it.

Along with the resorptive effect, poisons can also have a local and reflex effect.

The local action of the poison is understood as changes in the place of primary contact with tissues. It can be irritating, necrotizing, astringent, anesthetic. With various types of local action of poisons, certain groups of extero - and interoreceptors are always involved, which causes corresponding reflex reactions from the nervous system.

5. General condition of the body.

The strength of the toxic effect of any poisonous substance also depends on the individual characteristics of the human body. From this point of view, in each case, the severity of the clinical symptoms of poisoning is different. The main factors on which the severity of intoxication depends are the following: gender, age, body weight, the presence of concomitant diseases, the nature of the diet, the level of metabolic processes, and a number of others.

For example, the elderly and children, women during pregnancy, lactation, menstruation are more sensitive to the action of poisons.

One of the most important factors determining the body's sensitivity to poisons is the state of nutrition. Emaciated people tend to succumb to harmful effects more quickly than people with a normal diet.

Physical stress also affects the body's sensitivity to poisons. During physical work, pulmonary ventilation increases, which contributes to a faster and deeper penetration of volatile toxic substances. In addition, the volume of blood flowing through the lungs increases, which leads to a rapid increase in the concentration of poison to critical levels. Poisons that cause hypoxia are stronger during exercise. Against the background of fatigue, the effect of poisons is manifested more intensely.

The toxic effects of poisons are influenced by general health. With long-term debilitating diseases (heart, liver, kidneys), this effect is very strong.

It is known that there are pronounced individual reactions of the body to one or another poison and the existence of a sharply reduced sensitivity, when the body easily tolerates toxic and even lethal doses. Such addiction, usually most often occurs among drug addicts, when, due to adaptation to the dose used, the desired state of euphoria does not occur, which makes them constantly increase the amount of the drug used.

6. The influence of the external environment.

Environmental factors have a significant effect on the degree of exposure to poisons on the body: temperature, humidity, illumination, atmospheric pressure, radiant energy, etc. The toxicity of toxic substances increases with increasing ambient temperature. This, first of all, concerns work with volatile and easily evaporating substances, the concentration of which in the air increases, and the simultaneous increase in respiration contributes to the increased intake of poisons into the body.

Anoxemia, which develops under the influence of low barometric pressure, can also change the sensitivity to toxic substances. The influence of UV radiation on the body's sensitivity to poisons affecting calcium metabolism was noted. For example, areas of the skin exposed to carbolineum, the active principle of which is anthracene oil, become very sensitive to sunlight, which is manifested by dermatitis, intense pigmentation, the skin acquires a brown and greenish tint.

Forensic classification of poisons

There are various classifications of poisoning and poisonous substances.

Poisons are classified according to the following principles: pathophysiological (depending on the mechanism of changes occurring in the body), hygienic (according to the quantitative scale of the danger of poisons), pathochemical (according to the mechanism of interaction with enzymes), etc.

In forensic medicine, a classification of poisoning is adopted, based on the pathophysiological effect of poisons. According to this, the following groups of poisons are distinguished:

Corrosive poisons.

This group includes substances (caustic alkalis, acids, vapors of ammonia and iodine, phenol, etc.) with a pronounced effect in the area of primary contact with tissues. Local action is the main and decisive factor in the symptom complex of poisoning.

The action of these poisons, of course, is not limited only to local action, being absorbed, they also have a general resorptive effect on the entire body.

Resorptive poisons.

The toxic effect of this group of substances appears only after absorption. All resorptive poisons, in turn, are subdivided into:

- *Destructive poisons* (cause significant morphological changes in internal organs: liver, kidneys, heart, etc.).
- *Blood Poisons* (various in a toxicodynamic respect substances that cause changes in the biochemical composition and properties of blood).
- *Functional poisons* (cause mainly functional lesions, without significant violations of the morphology of the organ).

General plan of forensic medical examination of a corpse in case of poisoning

1. Study of all materials provided to the expert, especially medical documentation (medical history, outpatient card, ambulance documents, certificates, prescriptions), as well as the protocol of inspection of the scene.

2. Identification during external and internal research of morphological changes that can be observed in case of poisoning with certain poisons or groups of poisons.

3. Taking material for forensic chemical and forensic histological studies.

Poisoning with caustic poisons

Acute poisoning with caustic poisons develops, as a rule, when they are ingested orally in the body as a result of an accident or for suicidal purposes. The most typical representatives of this group of substances are acids and alkalis.

Acid poisoning

Acids- these are hydrogen compounds, the molecules of which in an aqueous solution split off hydrogen protons (dissociate), which can be replaced by metals with the formation of salts. The toxic effect is due to the action of free H-ions, with an increase in the number of which, the severity of the toxic effect increases. In this regard, acids have an irritating, cauterizing, necrotizing, melting effect. Hydrogen ions take away water from tissues, cause protein denaturation with the formation of water-insoluble acid albuminates, lead to coagulation (dry) necrosis. Under the influence of H-ions, hemoglobin breaks down with the formation of its derivatives (hematoporphyrin, methemoglobin, acidic hematin), as a result of which the damaged tissues acquire a dark brown or black-brown color. The degree of tissue necrosis when exposed to acids is in direct proportion to the concentration, degree of dissociation and duration of contact of acid with tissues, sensitivity and reactivity of the body. In places of contact with acid, irritation first occurs, then inflammation, then burns and, finally, tissue destruction. The dead tissues form dense scabs surrounded by areas of inflammation, which prevent the deeper penetration of hydrogen ions into the tissues.

Morphological picturein case of poisoning with various acids, it has similar features: burns of the skin around the mouth, nose, in the chin area, on the neck in the form of streaks, on the hands, on the front surface of clothing. In addition, burns of the mucous membrane of the oral cavity, esophagus, stomach, duodenum 12, laryngeal edema in combination with signs of rapid death (plethora of internal organs, liquid blood in the vessels, etc.) are determined.

If death did not occur immediately, then signs of bronchopneumonia (multiple pneumonic foci), purulent-fibrinous peritonitis, mediastinitis, nephrosonephritis, severe dystrophic changes in internal organs join the above morphological picture: the heart is flabby, dull; liver - flabby, grayish-red, with a brown tinge; the kidneys are flabby, the boundaries of the layers are indistinguishable, the cortical layer is bulging; the brain tissue is flabby, edematous, anemic; the adrenal cortex is gray; thick dark brown blood in the lumen of the vessels.

Differential diagnosis of poisoning with various acids is the easier, the shorter the period from the moment of taking the poison to the onset of death, and it begins already with a sectional study.

Most often in forensic practice, acetic acid poisoning occurs, due to its easy availability in the required quantities. Poisoning with other acids is only of an episodic nature.

Acetic acid- weak monobasic organic acid of the aliphatic series. It occurs in the form of anhydrous crystals, which are formed at a temperature of +16.6 degrees and resemble ice ("glacial" acetic acid), as well as in the form of vinegar essence (70-80% solution) or table vinegar (3-8% solution). The lethal dose of the first is 12-15 g, essences - 20-30 ml, vinegar - about 200 ml. The local action of acetic acid is less pronounced than that of inorganic acids, which is explained by a weak degree of dissociation and a small concentration of hydrogen ions, which are quickly bound by blood and tissue proteins. However, acting as a whole molecule, it is rapidly absorbed and has a pronounced resorptive effect. Moreover, the degree of this action is in direct proportion to the depth of damage to the gastrointestinal tract wall. Violation of the integrity of the blood and lymph vessels during a burn leads to a slowdown in

the absorption of acid. Therefore, a stronger resorptive effect is observed in case of poisoning with diluted acetic acid. In addition, it is highly volatile, so it easily penetrates the respiratory tract. When vapors are inhaled, there is a sharp cough, shortness of breath, chest pains, rhinitis, laryngitis, tracheobronchitis. Death occurs either from respiratory failure against the background of developed hemorrhagic pneumonia, or from asphyxia as a result of laryngeal edema and spasm of the glottis. shortness of breath, chest pain, rhinitis, laryngitis, tracheobronchitis. Death occurs either from respiratory failure against the background of developed hemorrhagic pneumonia, or from asphyxia as a result of laryngeal edema and spasm of the glottis. shortness of breath, chest pain, rhinitis, laryngitis, tracheobronchitis. Death occurs either from respiratory failure against the background of developed hemorrhagic pneumonia, or from asphyxia as a result of laryngeal edema and spasm of the glottis.

When taken orally, there are burns of the skin around the mouth of a grayish color, in fresh cases having a loosened, softened appearance. The mucous membrane of the gastrointestinal tract in a state of pronounced coagulative necrosis in the form of dull gray-brown films, which in the first hours of poisoning are very strongly associated with the underlying tissues. In the subacute course of poisoning, areas of necrosis are edematous, swollen, easily rejected in the form of a cast of an organ with slight pressure on them. A burn of the stomach gives a picture of corrosive gastritis, more pronounced in the area of the cardia and pylorus. Sometimes the poison affects the entire thickness of the stomach wall, causing gangrenous-hemorrhagic gastritis with perforation. Acute multiple ulcers give profuse bleeding either in the first hours after taking the poison or in 1-2 weeks by the time the necrotic epithelium is rejected.

The resorptive effect is manifested by massive intravascular hemolysis of erythrocytes, which is accompanied by the release of hemoglobin into the plasma and the development of hemoglobinemia and hemoglobinuria. Initially, hemoglobin permeates the walls of the renal tubules, after about 6 to 8 hours, hemoglobin masses are found in the lumen of the tubules and loops of Henle, and by the end of the first day - lumpy hemocylinders. Small hemoglobin components also appear in the cytoplasm of the renal epithelium. The presence of hemoglobin in the kidneys is confirmed by a positive Perls reaction (to iron). As a result of obturation and impregnation of the renal tubules with hemoglobin, necrotic changes in the epithelium (hemoglobinuric nephrosis) occur and acute renal failure develops, which is the immediate cause of death. Intravital hemolysis promotes massive breakdown of hemoglobin in the liver and spleen. In the latter, intense hemosiderosis develops a few hours after poisoning. The breakdown of hemoglobin in the liver leads to the formation of a large amount of verdoglobin, biliverdin, bilirubin, which stain the skin, mucous membranes, and sclera yellow.

Hemolysis leads to a change in the rheological properties of blood and the development of thrombosis.

In a sectional study on the first day after death, a number of characteristic signs are noted that make it possible to make a diagnosis already at the sectional table:

- Yellowness of the skin and sclera;
- Burns of the mucous membrane of the gastrointestinal tract in the form of gray-brown films from layers of necrotic epithelium;
- At a later date, CO is rejected by the layers under light pressure;
- Gangrenous-hemorrhagic gastritis;
- Hemorrhagic pneumonia;
- The specific smell of vinegar;

- Hemoglobinuric or pigmentary nephrosis (the kidneys are sharply enlarged in size and mass, saturated dark red, with a poorly distinguishable border of the cortical and medullary substance in the section);
- Hemosiderosis;
- Signs of thrombosis.

Nitric acid- strong monobasic inorganic acid. In its pure form, it is colorless, but usually colored yellow due to partial decomposition on exposure to light to form nitrogen dioxide. It affects tissues not only with hydrogen ions, but also with anions, contributing to the decomposition of protein. As a result, xanthoproteic acid (nitro compound of tryptophan of a bright yellow color) is formed, imparting a yellow color to all affected tissues. This yellow coloration can be seen on the lips and around the mouth, in the mouth, esophagus, stomach and even intestines. However, this color is formed only when exposed to concentrated nitric acid (at least 30%). Otherwise, only a dark brown scab can be observed due to the formation of hematin, as in the case of poisoning with other corrosive acids. An additional diagnostic feature is the suffocating odor of nitrogen oxides from the cavities and tissues of the corpse. A lethal dose of about 5-10 ml.

Sulphuric acid- an energetic oxidizing agent, a powerful dehydrating agent that leads to carbonization of organic matter. Therefore, often, the scab formed during poisoning with sulfuric acid has a coal-black color. The wall of the stomach is sharply thickened, gray-black in color, with a pronounced pattern of blood vessels. The presence of multiple perforation holes, which have uneven edges, is often noted, sometimes the entire stomach is destroyed. Posthumous perforations are also common. Intravital perforations are characterized by reactive changes on the part of the peritoneum. Postmortem diffusion of acid through the stomach wall is possible, even without perforation. In this case, there are seals of adjacent tissues and organs up to the muscles of the back. Tissues and organs become grayish, muscles brown. Otherwise, the morphological picture does not fundamentally differ from that in case of nitric acid poisoning. In the lumen of the vessels, tarry blood and crumbling blood coagulation of red-black color. A lethal dose of 5-10 ml.

Alkali poisoning

Alkalisare strong water-soluble bases, the molecules of which are donors of hydroxyl ions, which are responsible for their toxic effect. It is manifested by saponification of fats and hydrolysis of proteins with the formation of alkaline albuminates, which are readily soluble in water. As a result, tissue swelling, melting and liquefaction occurs. Due to the dissolving action, alkalis penetrate deeply into tissues, forming a thick layer of moist colliquation necrosis. The boundaries of necrosis go far beyond the direct action of alkali, therefore, there is no demarcation zone along the periphery of the damaged tissues.

Alkalis have the ability to dissolve a wide variety of tissues, including skin, nails, hair. A feature of tissues exposed to alkali is their grayish color, softening, swelling, jelly-like appearance. The scab that occurs under the action of alkali is initially soft, whitish-grayish in color, but soon, after the destruction of blood vessels, it becomes greenish-brown, as a result of the formation of alkaline hematin.

The blood, leaving the damaged vessels, does not coagulate and acquires a greenish-brown color.

The resorptive effect of alkalis is manifested by the development of uncompensated alkalosis, which is expressed in a deep metabolic disorder and a weakening of cardiac activity up to collapse.

*Morphological picture*with fatal oral alkali poisoning, it is quite typical: a deep chemical burn of the skin in the area of the mouth and nose, neck in the form of streaks, as well as along the digestive tract (without a clear border with intact tissues). There is a sharp swelling of the mucous membrane in the form of grayish softening of a gelatinous consistency. In this case, the thickness of the submucosa (in the esophagus and stomach) is 5-6 times the thickness of the mucous membrane. You can find a grayish softening of adjacent tissues due to diffusion of alkali through the stomach wall. Microscopic examination of the stomach reveals a pronounced vasospasm; the epithelium is spread out almost everywhere and cut off. Characterized by swelling of the larynx and signs of rapid death.

However, it is possible to reliably diagnose caustic alkali poisoning only with the help of a forensic chemical study.

Poisoning with destructive poisons

Mercury poisoning

Mercury- liquid metal, found in nature in its natural state or in the form of a compound with sulfur - cinnabar, from which mercury is usually extracted. Although mercury boils at a temperature of 357 degrees, it easily evaporates at room temperature, and with an increase in the area of evaporation, the latter increases significantly. This is observed, for example, when a mercury-containing device is broken and, being spilled, it turns out to be finely crushed.

In toxicological terms, the degree of toxicity of mercury compounds depends primarily on their solubility. Thus, when ingested orally, metallic mercury does not cause poisoning due to its low solubility. This is due to the fact that metallic mercury is in a molecular state and therefore is inactive. However, in a finely dispersed state or in the form of vapors, it is easily absorbed, especially through the surface of the alveoli, and causes severe poisoning. The toxic effect of vaporous mercury is explained by its atomic state, in which it is most active.

Active mercury ions interact with sulfhydryl, amino and carboxyl groups of tissue proteins. By inactivating them, mercury changes the configuration and properties of protein molecules, inhibits their enzymatic, hormonal and immunological activity, disrupts the processes of oxidative phosphorylation, glycolysis, and mineral metabolism. The mercury albuminates formed during the interaction of mercury with proteins are capable of accumulating in the cell for a long time, causing the gradual disintegration of protoplasm.

The most toxic inorganic mercury compounds are mercury nitrate and mercury chloride (mercuric chloride).

Sectional picture

Morphological changes at the injection sites are very variable - from simple hyperemia and swelling of the mucous membrane through all transitional forms to coagulation necrosis in the form of a white or gray dense scab. In the stomach there is a sharp plethora, focal necrosis of the mucous membrane, hemorrhagic edema of the submucosal layer. In general, the gastric mucosa is grayish, dense, resembling shagreen skin. The histological picture can be different in different parts of the same stomach.

If death occurred a few days after the poison entered the body, then the main morphological changes are observed in the kidneys and the large intestine.

An important sign of mercury poisoning is the sublimate kidney. At death in the first days, the kidneys look reddish, full-blooded, swollen (large red sublimate kidney). Later, as a result of vasospasm, the kidneys become pale gray or yellowish, flabby consistency (large white sublimate kidney). Macroscopically, the kidney bark swells, looks gray, as if scalded. When the capsule is cut, the kidney appears to be exfoliated. By the end of the first week, the kidneys decrease in volume, shrink (small pale sublimate kidney) due to the development of necrobotic and necrotic changes (sublimate necrosis) followed by petrification (due to impaired calcium metabolism due to concomitant sublimate colitis).

In the large intestine, changes resemble those of dysentery ("sublimate dysentery"). They are mainly localized in the transverse colon. The intestines are swollen, the walls are thickened. The intestinal folds are edematous, the mucous membrane is full-blooded with multiple foci of hemorrhage and areas of necrosis of various depths, inflammatory infiltration of the mucous and submucous layers.

In a forensic chemical study, in addition to taking into account the absolute amount of mercury in organs, it is extremely important to know its content in the liver and kidneys per 100 g of an organ (poisoning is indicated by its content in the liver and kidneys exceeding 1 mg per 100 g of an organ). In addition, in normal conditions, the relative content of mercury in the kidneys exceeds that in the liver; in case of poisoning, this ratio changes the other way round. Mercury is able to accumulate in bones and remain in them for a long time even after death. In a corpse, it can be found even tens of years after burial.

Poisoning by arsenic and its compounds

Due to insolubility in water, pure arsenic does not cause poisoning and therefore has no toxicological significance, however, its compounds are rather strong poisons. Poisoning with arsenous anhydride (white arsenic) is most dangerous, poisoning with medications containing arsenic - salvarsan, novarsenol, miarsenol occurs.

High toxicity, lack of odor and taste have determined in the past the frequent use of this poison, both for the purpose of murder and suicide.

Arsenic compounds are classified as capillary toxic poisons. The paresis and paralysis of the capillaries that occur during poisoning lead to the development of vascular collapse. Due to this, blood pressure drops, hyperemia of the abdominal organs with pronounced congestion occurs, which leads to the sweating of a large amount of fluid into the lumen of the small and large intestines and the appearance of a liquid flocculent cholera-like stool with an admixture of blood and mucus.

The mechanism of the toxic action of arsenic compounds is associated with their ability to form stable compounds with sulfhydryl (dithiol) groups of enzyme proteins (cyclic arsenides). These include, for example, pyruvate kinase, an enzyme involved in the oxidation of pyruvic acid. As a result, all reactions of aerobic glycolysis stop at the stage of PVC formation, which leads to significant disorders of both carbohydrate and energy and lipid metabolism associated with it.

The toxic dose when taken orally (in terms of pure arsenic) is 0.01 g, the lethal dose of the most toxic compound, arsenous anhydride, is 0.1 - 0.2 g.

Sectional picture

The most vivid and full-fledged morphological picture develops in the gastrointestinal form of poisoning. Already with an external examination, a well-pronounced rigor mortis and signs of exicosis are revealed: dry, flabby, shiny skin, its loss of turgor and a decrease in

elasticity. In the case of a rapid onset of death, the blood in the vessels is light red, with a prolonged course of poisoning - hypervenous, with elastic fibrin convolutions. The loops of the small intestine, due to the loss of fluid as a result of sweating it into the lumen, look lethargic, stretched, collapsed. The leaf of the visceral peritoneum is light pink in color, sticky to the touch, due to sweating and fibrin deposition. In the lumen of the stomach, a liquid cloudy flocculent content with an admixture of bile or thick viscous mucus, firmly fixed to the walls, is found. In the depths of the folds of the mucous membrane, grains of poison in the form of octahedral crystals can be found, which should be carefully removed for forensic chemical research. The gastric mucosa is usually red in color, which is caused by multiple hemorrhages and paretic vasodilation, swollen due to edema and round cell infiltration, at the tops of the folds is often covered with grayish overlays. In some areas, foci of necrosis and ulceration are visualized, it is here, first of all, that you need to look for particles of poison. The contents of the small intestine are liquid, cloudy, with whitish flakes, or have a mushy or liquid consistency (depending on the length of the dying period). The mucous membrane of the small intestine is sharply edematous, swollen, in places with hemorrhages and areas of superficial necrosis. Solitary follicles and Peyer's patches are infiltrated or ulcerated. Due to the tenesmatic nature of the stool, the colon is contracted and contains only mucus. On rotten corpses in the lumen of the intestines, sometimes a yellow sediment is found, formed as a result of the interaction of hydrogen sulfide with arsenic compounds and consisting of arsenic sulphide.

Changes in other organs are unstable. There are signs of fatty degeneration in the heart, liver and kidneys. The substance of the brain is edematous, hyperemia, punctate hemorrhages, pericellular and perivascular edema are detected.

In the paralytic form, morphological changes are minimal or absent at all due to the rapid onset of death. Pathological changes are reduced by G.O. to swelling of the brain and its membranes. In addition, microscopic examination determines multiple small-point hemorrhages in the spinal cord (along the periphery of the central canal, in the walls of the fourth ventricle), vacuolization of the cytoplasm and the phenomenon of karyocytolysis in Betz cells, lumpy disintegration of myelin, swelling and fragmentation of axial cylinders in the pathways of the brain and spinal brain.

To confirm the diagnosis in all cases, a forensic chemical study is necessary. In addition to the standard kit for general chemical analysis, it is necessary to take samples of hair, bones and nails. In these tissues, arsenic compounds are able to accumulate and be detected many years after poisoning.

Poisoning with functional poisons

Cyanide poisoning

Hydrocyanic acid and its salts (potassium, sodium, mercury cyanides) are found in pure form only in laboratories.

At present, cyanide poisoning is extremely rare and is mainly due to the excessive consumption of fruits and seeds of some plants (apricots, cherries, plums, pears, apples, elderberries, and especially bitter almonds). The peculiarity of the seeds of these plants is that they contain*amygdalin glycoside*containing easily mobilized components of hydrocyanic acid. As a result, when this glycoside interacts with hydrochloric acid of gastric juice, hydrocyanic acid is formed. In addition, alcoholic beverages prepared from the fruits of the above plants can

also be a source of hydrocyanic acid, which is indifferent to ethyl alcohol, but absorbed faster in its presence.

With the penetration of active cyano groups into tissues, cytochrome oxidase A3 (terminal enzyme of the respiratory chain) is stabilized in a stable trivalent state of iron. As a result, the transfer of electrons from NAD H and FAD H to the final acceptor, oxygen, stops. Thus, cellular respiration is blocked, as well as the processes of utilization and restoration of oxygen. In this case, on the one hand, tissue hypoxia occurs without anoxemia (moreover, the blood is hyperoxygenated due to an excess of oxygen unclaimed by tissues and an increase in the affinity of hemoglobin for oxygen due to hypoxic changes in tissues).

It is interesting to note that myoglobin is able not only to neutralize freely dissociated cyano groups, but also to take them away from cytochrome oxidase, restoring tissue respiration. In this case, cyanmyoglobin is formed, which slowly dissociates in the blood, and therefore is not dangerous for the body.

Sectional picture

An autopsy of those killed from cyanide poisoning reveals the following morphological manifestations: pinkish color of the skin, cadaveric spots, mucous membranes, internal organs, from which (especially from the brain) the smell of bitter almonds emanates. The blood in the vessels is liquid, it may have a scarlet-red color with a cherry tint. The red color is due to the formation, primarily of oxyhemoglobin. The cherry hue is associated with the formation of cyanmethemoglobin. In some cases, there is a loosening and swelling of the gastric mucosa and its staining in cherry color. Swelling and loosening is associated with the alkali-like action of potassium cyanide, and staining is usually formed posthumously due to imbibition of the gastric mucosa with cyanide hematin.

Tests for the presence of hydrocyanic acid in internal organs are of historical and approximate importance. For these purposes, it is better to take crushed brain tissue, which is treated with a 1% solution of silver nitrate and 10% solution of tartaric acid (Neureiter's test).

Microscopic examination can reveal plethora and slight pulmonary edema, dystrophic changes in the nerve cells of the brain, chromatolysis, vacuolization, mainly of the motor neurons of the anterior horns of the spinal cord.

For the final verification of the diagnosis, the data of the circumstances of the case, clinical symptoms, the results of morphological and forensic chemical studies are used (it should be borne in mind that poisonous cyanide compounds are volatile and easily decompose during decay).

Blood poisoning

Poisoning with methemoglobin-forming poisons

Currently, many substances are known that can form methemoglobin: berthollet's salt, aniline, nitrobenzene, nitroglycerin, hydroquinone, and a number of other substances.

The main point of application of the action of these poisons is heme - the structural iron-containing element of hemoglobin. Unlike oxyhemoglobin, which contains ferrous iron, in methemoglobin it is in a stable trivalent state; while oxygen is combined with iron as part of the OH hydroxyl group. In view of this, hemoglobin loses its ability to carry out the function of transporting oxygen in the body, as a result of which acute oxygen starvation of tissues and organs develops. The lethal concentration of methemoglobin in the blood is about 70-80%.

In significant concentrations, methemoglobin reduces the osmotic resistance of erythrocytes, which entails their hemolysis. In addition, methemoglobin-forming poisons are capable of prolonged accumulation in the liver and adipose tissue, which often leads to a repeated wave of methemoglobin formation as a result of the release of the poison from the depot into the blood.

Sectional picture

The autopsy picture is specific enough to suspect methemoglobin-forming poisoning. Already upon external examination, attention is drawn to the grayish-brown, smoky (slate-gray) color of cadaveric spots. A similar color of cadaveric spots is due to the translucence of blood through the skin, which has a chocolate or coffee color, a viscous consistency. Soft tissues, internal organs have a similar grayish-brown color. The liver is enlarged, microscopic examination reveals signs of protein and fatty degeneration. The kidneys are full-blooded, flabby, on a cut with indistinguishable layers boundaries, the renal tubules are infiltrated with hemoglobin casts (hemoglobinuric nephrosis).

In case of poisoning with nitrobenzene from the cavities and from the organs of the corpse, the smell of almonds is felt, in case of poisoning with aniline - aniline. In case of poisoning with hydroquinone, the urine left in an unclosed bottle becomes greenish after a while.

Spectroscopic examination of cadaveric blood for methemoglobinemia indicates the presence of a characteristic band in the red-orange part of the spectrum.

The quantitative determination of methemoglobin is carried out by spectrophotometric or gas chromatographic methods.

Carbon monoxide poisoning

Carbon monoxide- colorless and odorless gas, slightly lighter than air. In its pure form, it can be obtained only in the laboratory, where poisoning with it is very rare. Most often, carbon monoxide is found in a mixture with other gases (carbon monoxide, light and water).

In terms of the frequency of occurrence of poisoning with carbon monoxide, they are second only to poisoning with alcohol and its surrogates.

Pathogenesis

Until now, all the mechanisms of the effect of carbon monoxide on the body have not been deciphered. The main, but not the only, pathogenetic mechanism is the close interaction of carbon monoxide with hemoglobin and the formation of a strong and very stable compound - carboxyhemoglobin. Possessing an extremely low degree of dissociation and reactivity, it completely blocks heme, thereby "turning off" the oxygen transport function of the blood. The result is the development of hemic hypoxia.

It should be borne in mind that the same process with the formation of carboxyhemoglobin can also occur posthumously if the tissues of the body are exposed to prolonged exposure to high concentrations of carbon monoxide (in the fire site). As you know, a similar method of restoring the original color of organs and tissues taken from corpses is used in the manufacture of museum preparations.

An alternative mechanism of the toxic effect of CO is an inhibitory effect on tissue enzymes, which include iron. Thus, the cytotoxic effect of carbon monoxide is realized, which is expressed in the suppression of tissue respiration (tissue or histotoxic hypoxia), primarily in the neurons of the respiratory and vasomotor centers.

Sectional picture

Autopsy reveals quite characteristic signs of carbon monoxide poisoning in cases of a more or less prolonged period of dying. These include: bright red cadaveric spots, as well as sometimes pink color of the skin outside the zone of distribution of cadaveric spots, on the higher parts of the body; bright red liquid blood in the vessels; in this regard, the muscles have a carmine-red color, the internal organs (lungs, liver, kidneys, brain) in the cut with a pinkish or reddish tint. The severity of these signs is in direct proportion to the concentration of carboxyhemoglobin in the blood. In cases of rapid death (with a fulminant form of poisoning, rapid removal of the victim from an area of high carbon monoxide content,

There is plethora and edema of internal organs, hemorrhages under the mucous and serous membranes, as well as in the membranes and substance of the brain. Often in young subjects, focal hemorrhages are found in lymphoid formations - tonsils, Peyer's patches and solitary intestinal follicles, lymph nodes of the chest cavity.

In protracted cases, symmetrical, nested foci of softening in the region of the subcortical nuclei, arising at the site of former hemorrhages, are determined in the brain tissue.

As an express diagnosis, you can use preliminary tests for the presence of carboxyhemoglobin in cadaveric blood. When added to blood containing carboxyhemoglobin, 33% solution of sodium or potassium hydroxide (Hoppe-Seiler test) or formalin (Liebman test), its color does not change. This is easily explained by the fact that carboxyhemoglobin, being a very strong non-dissociating compound, is unable to react with any substances due to the complete blockade of hemoglobin. A control study of obviously unchanged blood shows a dark brown staining. The Hoppe-Seiler test can give questionable results with the blood of newborns, because it contains a significant amount of fetal hemoglobin, which is more resistant to alkali.

It is also possible to use a preliminary spectroscopic sample directly at the section table The essence of the test is as follows: in two test tubes with cold water add a few drops of the test blood (test tube No. 1) and the same amount of obviously unchanged blood (test tube No. 2). After mixing, the contents of both tubes are examined with a spectroscope. In the test tube with the blood under study, in the presence of carboxyhemoglobin in it, in the yellow-green part of the spectrum, two dark absorption bands are determined, almost identical to the absorption bands of oxyhemoglobin. To differentiate them, several crystals of sodium hydrosulfite are introduced into each of the test tubes. In the presence of carboxyhemoglobin in the blood, the absorption spectrum does not change,

However, the tests described above are currently not of practical, but of historical interest. The quantitative determination of the content of carboxyhemoglobin is carried out by a forensic chemical study of blood taken from deep vessels and cavities of the heart, by the method of spectral analysis. The lethal concentration of carboxyhemoglobin is 60-80%.

If it is impossible to draw blood (in cases of severe burning of the corpse), at least 1 kg of muscle is removed for research. Carboxyhemoglobin is detected in the blood of a corpse long after death (2-3 months); it can also be found in dried blood traces of various ages.

Ethyl alcohol poisoning

Acute fatal alcohol intoxication accounts for about 60% of all cases of poisoning. But to date, no clear morphological markers of alcohol intoxication have been developed, which in no way diminishes the significance and urgency of the problem. There is a constant search for new, more specific morphological criteria for acute alcohol poisoning.

One of the tasks of an expert in a forensic medical examination of a corpse is to decide whether alcohol intoxication is the cause of death or only a contributing factor. This task is

solved in each specific case on the basis of a comprehensive analysis of preliminary information about the circumstances of the case, autopsy data and the results of laboratory tests. Moreover, the level of exogenous alcoholism, i.e. the concentration of alcohol in the blood at the time of the study is one of the main criteria that motivate expert conclusions about alcohol poisoning as a cause of death.

Sectional picture.

The sectional picture is not specific. On external examination of the corpse, a rich bluish-purple color of cadaveric spots, their diffuse nature, puffiness and blueness of the skin of the face, neck, and upper body are usually noted. Often, small ecchymosis is detected in the skin of the face, neck, conjunctiva of the eyelids, the vessels of the sclera are injected, the eyeballs are bulging (exophthalmos). Internal examination determines liquid dark blood in the vessels with overflow of the superior vena cava system, venous plethora of internal organs, plethora and edema of the vascular plexuses of the ventricles of the brain, edema of the membranes and brain matter, pulmonary edema, uneven blood filling of the heart muscle. Due to the burning effect of alcohol on the mucous membranes, there is a hyperemia of the mucous membrane of the larynx, trachea, stomach, proximal small intestine with simultaneous discoloration of the contents of the duodenum, edema of the gallbladder bed. In addition, the presence of Tardier spots, hemorrhages in the gastric mucosa, pancreas, kidney and adrenal tissue, bladder overflow are noted. These signs are not always found, they may be absent altogether, depending on the characteristics of thanatogenesis in each case.

Previously, the smell of alcohol from the contents of the stomach, lungs, and brain was considered a valuable diagnostic criterion. This sign is important only when deciding whether a person took alcohol during his lifetime or not. When comparing the results of a forensic chemical study with the intensity of the odor, it was found that in some cases fatal alcoholism is not accompanied by an odor from the internal organs, and vice versa, subtoxic doses of alcohol can produce a rather noticeable "aroma".

Histological examination reveals a violation of the permeability of the walls of vessels of all calibers, which is expressed in the loosening of the vascular wall, swelling, desquamation of endothelial cells, plasma impregnation of the walls of the arteries. Perivascular diapedesic hemorrhages are noted around the vessels. Similar changes are found in most internal organs: the brain, liver, pancreas, kidneys, adrenal glands, heart muscle.

Along with the above signs, more characteristic microscopic changes are determined, indicating the effect of alcohol on tissues. These include the myolysis of individual cardiomyocytes, caused by the destabilization of lysosomal membranes and the release of proteolytic enzymes into the cytoplasm, followed by the destruction of all subcellular structures. The foci of myolysis of cardiomyocytes are distinguished by the absence of cross striation, homogeneity and, in some cases, very fine, as it were, dust-like granularity. This symptom, as a rule, does not depend on the concentration of alcohol in the blood and does not occur in injuries and diseases of the cardiovascular system, therefore, it can be used as a differential diagnostic to establish the cause of death of persons who have died while intoxicated.

Intracellular edema of cardiomyocytes, edema of the myocardial stroma, enlarged polygonal hepatocytes can be considered additional morphological criteria of OAO. They have an enlightened cytoplasm and clear contours.

These morphological signs indicate that the immediate cause of death in OAO in most cases is acute heart failure, which is the final stage of the resulting alcoholic (brain) coma

resulting from this poisoning. However, the pathogenesis of AHF is not the same. In some cases, AHF is a consequence of damage to the centers of the medulla oblongata. In other cases, the hypoglycemic state that develops under the influence of toxic doses of ethanol, and in some cases, hypoglycemic coma, matters.

It follows from the above that all morphological characters have only approximate value and are not specific for JSC, which requires a correct assessment of them in the complex.

Forensic chemical research

The limited and nonspecific nature of the sectional signs of the open joint stock company make the forensic chemical examination of the blood and urine of the corpse especially relevant. According to the "Rules for a forensic medical examination of a corpse," the blood and urine of a corpse are subject to mandatory forensic chemical examination in all cases of violent and non-violent death. The exception is the deaths of adults who have been in the hospital for a long time and young children. Blood to determine the degree of alcoholism is recommended to be taken from peripheral vessels (femoral or brachial vein) or from the sinuses of the dura mater of a corpse, opened within 2 days after death. You cannot take blood for research, scooping it out of the opened body cavities or squeezing it out of the internal organs. It is advisable to collect blood and urine with sterile glass pipettes into clean sterile vials (from under penicillin). Failure to comply with the conditions of sterility may entail the introduction of microorganisms into the samples that can cause decomposition or neoplasm of alcohol. The vials are filled to the top, tightly closed with rubber stoppers, wrapped with parchment paper, provided with an appropriate label, on which I indicate the date of collection of the material, name and surname. corpse and expert, the number of the conclusion and without delay sent to the forensic chemical department. Preservation of blood and urine samples is not permitted except in special cases. If long-term transportation at high temperatures is necessary, blood and urine samples for gas chromatographic analysis are preserved with quinosol (3-5 drops of solution per 10 ml of blood).

In many cases, the onset of death is preceded by the development of a powerful stress reaction, which in some cases is accompanied by disorders of carbohydrate metabolism with symptoms of hyperglycemia and glucosuria. The latter circumstance contributes to the launch of alcoholic fermentation reactions, which in turn casts doubt on the reliability of the results of the forensic chemical analysis of blood and urine. A similar situation occurs in cases where a person suffered from diabetes during his lifetime.

According to a number of researchers, the formation of ethyl alcohol in the blood and urine occurs constantly both in the corpse and in the samples taken from the corpse, stored both at room temperature and in a refrigerator.

However, in the early stages of the postmortem period in the organs and tissues of the corpse at room temperature, the formation of ethanol occurs only in insignificant quantities. And only in the presence of a specific alcohol-forming microflora (yeast fungal cultures) in the corpse in its internal organs and tissues, during the same period of the postmortem period, the intensive formation of ethyl alcohol occurs up to a concentration of about 6 ppm. However, it should be noted that cases of the formation of alcohol in a corpse in high concentrations are rare, because this requires a combination of conditions: the presence of a specific microflora, an unchanged corpse, a relatively high ambient temperature.

Consequently, a high degree of alcoholism, revealed during the forensic chemical examination of a corpse stored at relatively low ambient temperatures, is in vivo.

The results of a forensic chemical study of the blood and urine of decayed corpses are of relative importance. In such cases, to check the possibility of alcohol neoplasm in the corpse, it is necessary to conduct a bacteriological examination of the internal organs. The most intense neoplasm of ethyl alcohol occurs in the vessels of the chest cavity and in the blood from the cavities of the heart, the least - in the blood from the vessels of the extremities.

Considering the above, it should be noted that sometimes it is more advisable to take for examination neither blood and urine, but samples of the vitreous body of the eye and cerebrospinal fluid, because the concentration of ethanol in them in the postmortem period, as well as during storage of the samples, practically does not change. The vitreous body is removed with a syringe, making a puncture with a needle in the corner of the eye, CSF - puncturing the spinal canal.

In some cases, it makes sense to send for the study of blood clots from the area of hemorrhage (intracranial, from the area of fractures). The concentration of alcohol in them corresponds to that which took place during life, at the time of the formation of hemorrhage.

If it is impossible to get the right amount of blood, for example, when the body is crushed or dismembered, muscle tissue and a kidney can be taken for alcohol testing. Sometimes the stomach contents are additionally withdrawn (if there is no content, then mucus in the amount of several milliliters), which in some cases can contribute to the establishment of the stage of alcohol intoxication (resorption or elimination), the time elapsed since the last alcohol intake until death, the amount of alcohol consumed ...

If death occurs in the resorption stage, the concentration of ethyl alcohol in the blood, as a rule, is higher than in the urine, and in the urine it is higher than in the cerebrospinal fluid. At the same time, a greater amount of alcohol is found in the contents of the stomach. These data indicate that no more than 1.5 hours elapsed from the moment of taking alcoholic beverages to the onset of death.

Moreover, the high concentration of ethanol in the gastric contents, its low level in the blood and trace amounts in the urine and cerebrospinal fluid indicate that death occurred a few minutes after ingestion of alcohol.

In cases of death in the elimination stage, the amount of alcohol in the urine and cerebrospinal fluid will be greater than in the blood.

To assess the severity of alcohol intoxication, the following indicative table is proposed:

Table 1. THE SEVERITY OF ALCOHOL INTOXICATION

Less than 0.3‰	No influence of alcohol
0.3 - 1.5 ‰	Light intoxication
1.5 - 2.5 ‰	Medium intoxication
2.5 - 3.0 ‰	Severe intoxication
3.0 - 5.0 ‰	Heavy intoxication, death may occur
5.0 and more	Fatal poisoning

Mushroom poisoning

Mushroom Poisoning - Mycetism is generally a true food poisoning.

Stitching- they differ from edible morels by a cellular structure in the cut and contain gellic acid, which has a hemolytic and hepatotropic effect. The amount of this acid is variable and increases in dry years. When boiled for 10 minutes, the poison turns into a decoction, and the mushrooms become edible. When dried, gelic acid is oxidized by atmospheric oxygen and

inactivated. Death occurs in 1-5 days. Mortality reaches 50%. Poisoning at first resembles a clinic of dysentery, and then infectious hepatitis. On external examination, an icteric coloration of the skin, visible mucous membranes and sclera is noticeable. Autopsy revealed multiple hemorrhages under the serous membranes of internal organs, spleen and liver of lemon-yellow color. Fatty degeneration of the liver, kidneys and myocardium.

Death cap(false champignon). The main role in the origin of poisoning is played by amanitotoxin, which is heat and enzyme stable. These mushrooms also contain amanitohemolysin, phalloidin, □ - and □ -amanitin, which are destroyed by the temperature of 70C and by the action of gastric juice.

Mortality reaches 90% and depends on the amount of mushrooms eaten (one specimen is enough to poison several people) and the age of the victim.

Pathomorphological changes are reduced to mild rigor mortis, jaundice, multiple hemorrhages in the mucous and serous membranes of internal organs, fatty degeneration of the kidneys, liver, myocardium, and skeletal muscles. From the gastrointestinal tract, the phenomenon of acute gastroenteritis.

Fly agaric(red and panther). The active principles are several toxins: muscarine, muscaridin (mycoatropin) and pilztoxin. A lethal dose is 4-6 mushroom bodies. Poisoning must be differentiated from FOS intoxication. The clinic of poisoning is manifested by profuse watery stools, hyperhidrosis, hypersalivation, lacrimation, visual impairment due to miosis, hallucinatory delirium, rampage, convulsions, coma, and death may occur. An autopsy reveals changes characteristic of gastroenteritis and signs of a quick death.

Forensic diagnostics of poisoning by poisonous mushrooms is based on the circumstances of the incident, vital signs of intoxication, autopsy results and data from botanical research of vomit, wash water, and stomach contents of the intestines.

Features of a forensic medical examination of a corpse at the place of its discovery (incident) in case of suspected poisoning

If there is a suspicion of poisoning with gaseous substances, a forensic medical expert (expert doctor), prior to examining the scene of the incident, must inform the investigator of the need to call a specialist from the sanitary-epidemiological station to take air samples. Inspection of the scene is carried out after taking air samples for research and subsequent ventilation of the room.

Examining a corpse at the place of its discovery (incident) in case of suspicion of poisoning, the state forensic medical expert (expert doctor) is obliged to draw the attention of the investigator and note the following features:

- Traces of exposure to poison on the skin and clothing (burns, vomit

on and near the corpse). Acceptance of caustic poisons through the mouth is accompanied by a chemical burn of the border of the lips, the mucous membrane of the vestibule and the oral cavity. When caustic poisons hit the clothes, its fabric is destroyed, forming defects of various shapes, or tissue remnants that are easily destroyed when touched. Typically, such damage is found on the front of the garment or sleeves.

- Remains of chemicals (powders, tablets, liquids and empty bottles, ampoules, drug convolutions). When examining the clothes of a corpse, one should look for traces or remains of toxic substances in the form of powders, wet or dried spots of various colors, etc. in the pockets you can find packages of medicinal substances, individual powders or tablets. In addition, recipes, labels, notes with the names of medicinal substances that could be used in case of poisoning can be found.

- The color of the skin and visible mucous membranes of the corpse, the severity of rigor mortis. The yellow color of the skin is characteristic for poisoning with phosphorus preparations, red (bright pink) for carbon monoxide, pronounced rigor mortis is characteristic for poisoning with convulsive poisons, weak - under the action of hemolytic poisons, narcotic substances, etc.
- The presence of traces of injections, the smell coming from the mouth of the corpse. The injection site looks like a punctured puncture wound, sometimes covered with a crust. Their exact location, number, presence and color of bruising around are described.
- The presence of a syringe, injection needle, etc. A forensic medical expert (expert doctor), assists the investigator in the seizure of vomit, food remains, medicines, medicine containers, etc. for laboratory examination.
- When vomit, food remains, medicines are found, their quantity, consistency, color, smell, and the presence of inclusions are described.
- If food poisoning is suspected, food remains in plates, pots and other utensils are examined and described. All these items are subject to confiscation and direction for forensic chemical research. Dishes and other items with their contents are hermetically sealed, empty dishes are also seized in order to find the remains of a poisonous substance on its walls. On the walls and at the bottom of the discovered dishes, there may be a crystalline or other plaque formed after the contents have dried out.
- Vomit, feces, are collected in clean glassware.
- Remains of dry matter found on clothing are collected by tapping the fabric of clothing over a sheet of clean paper or clean glassware.

Seizure and direction of cadaveric material for forensic chemical research in case of suspicion of poisoning with an unknown poison

1.1. For the purpose of detecting and quantifying toxic substances for forensic chemical research, various internal organs, blood and urine are withdrawn and directed, taking into account the nature of the alleged poison and the routes of its introduction into the body, distribution, routes and rate of excretion, the duration of the course of intoxication and therapeutic measures. They also send vomit, the first portions of wash water, remains of medicinal and chemical substances, food, drinks and other objects.

If poisoning is suspected, at least 2 kg of internal organs are sent from the corpse of an adult to a forensic chemical study. With a prolonged course of poisoning, as well as during resuscitation measures, the amount of material sent should be increased to 2.5-3 kg.

1.2. Organs should not be washed with water and contaminated with chemicals or mechanical impurities. The organs are placed in glassware (dry wide-mouth jars). The use of metal or ceramic utensils is prohibited.

1.2.1. The internal ones are removed after the imposition of double ligatures on the esophagus, stomach, intestines (at a distance of 1 m in different parts) to prevent mechanical movement of their contents.

1.2.2. The expert must ensure that the poison is not removed from the corpse and does not get into it from the outside. Therefore, thoroughly wash the breakout table, tools and gloves before opening, and do not use water or other liquids during opening.

1.3. Banks should be washed with a solution of mustard or soda, rinsed thoroughly with tap and then distilled water and dried in an oven.

1.4. In case of suspicion of poisoning with an unknown poison, as well as in case of combined poisoning, it is necessary to withdraw:

- in jar No. 1 - stomach with contents (with the imposition of ligatures at the entrance and exit)
- in jar No. 2 - one meter each of the small and large intestines with contents from the most altered sections (with the imposition of double ligatures)
- in jar No. 3 - at least 1/3 of the most full-blooded areas of the lung
- in jar No. 4 - one kidney
- in jar No. 5 - 300 gr. brain
- in jar No. 6 - at least 300 ml of blood
- in jar number 7 - all the urine
- in jar number 8 - liver with gall bladder

The contents of the cans are not canned. But if you suspect poisoning with cardiac glycosides and other digitalis derivatives, as well as alkaloids, pesticides, antidepressants, the contents of the cans are preserved with ethyl alcohol (96%), and the level of the preservative in the can should be 1 cm above the organ. For control, 300 ml of pure alcohol is sent for research in a separate jar.

If you suspect the injection of poison through the vagina or uterus, it is necessary to additionally take the uterus and vagina into separate banks, if you suspect subcutaneous or intramuscular injection of poison - a site of skin and muscles from the area of the intended injection.

If you suspect poisoning with acids and alkalis, the pharynx, trachea and esophagus are additionally removed; organochlorine compounds - gland; compounds of mercury and arsenic - hair and nails; the joints of the lead and the waist are flat bones.

When examining an exhumed corpse, six soil samples of 500 g each are sent for examination: from above, from below, from the sides, from the head and foot ends, as well as fragments of the coffin upholstery, clothing, bedding, coffin board 20 by 20 cm and all metal objects and decorations.

FORENSIC-MEDICAL EXAMINATION
DEATH FROM MECHANICAL ASFIXIA

Asphyxia - an acute pathological process that occurs due to a lack of oxygen in the blood and tissues and the accumulation of carbon dioxide and is characterized by a severe symptom complex of disorders on the part of the central nervous system, cardiovascular system and respiratory organs.

As follows from the definition, hypoxia is the cornerstone of the asphyxia state.

Oxygen starvation of organs and tissues until the complete cessation of oxygen supply to the body is called hypoxia....

As a result of the development of hypoxia, a large amount of intermediate metabolic products accumulates in the blood. Products of incomplete oxidation cause a sharp suppression of the function of cells, the ability of which to utilize oxygen is further reduced. Thus, the vicious circle is closed.

Classification of asphyxia

Following the principle of classification of death, there are:

– Violent asphyxiation

– Nonviolent (pathological) asphyxia

Nonviolent asphyxiationdevelops with various kinds of diseases. This also includes newborn asphyxia.

Violent asphyxiationcan arise under the influence of a number of reasons. For example, from mechanical obstacles to breathing, in case of poisoning, in case of electric shock, etc.

A type of violent asphyxia is mechanical asphyxia, which is defined as a violation of external respiration under the influence of mechanical factors.

Depending on the method of obstruction of breathing, mechanical asphyxia is divided:

1. Mechanical asphyxiation from compression

a) Strangulation asphyxia
— hanging
— strangulation by loops
— strangulation by hands
— strangulation by other objects or other parts of the body
b) Compression asphyxiation
— chest compression
— compression of the abdomen
— compression of the chest and abdomen with hard and loose objects

2. Mechanical asphyxia from airway closure

a) Obstructive asphyxia (covering the openings of the mouth and nose with soft bodies and objects)
b) Aspiration asphyxia (aspiration of bulk substances, liquids, foreign bodies)
c) Drowning (aspiration, spastic, reflex and mixed)

3. Positional asphyxia

Periods of development of mechanical asphyxia.

The intravital course of asphyxia has a certain pattern. There are 2 periods in the lifetime of asphyxia.

one. The pre-asphyxiation period is from the moment the oxygen supply to the body stops to its critical decrease in the blood (from 10-20 seconds to 1-2 minutes).

During this period, no signs of asphyxia are observed; carbon dioxide accumulates in the body. During this period, compensatory-adaptive reactions develop in the body (increased frequency and deepening of respiratory movements, tachycardia), the reflex of oxygen conservation or the phenomenon of centralization of blood flow turns on.

This mechanism is triggered by a decrease in the partial pressure of oxygen in the blood and the stimulation of vascular chemoreceptors by oxygen-depleted blood. The result of these events is an increase in perfusion and gas exchange in the lungs, an increase in blood flow in the brain, heart with a simultaneous contraction of the vessels of the extremities and the abdominal cavity.

2. Asphyxiation proper: This period is conventionally subdivided into stages, successively passing one into another.

1. Stage of inspiratory dyspnea... In this stage, which lasts about 1 minute, the body tries to compensate for the lack of oxygen with enhanced inhalation movements. These increased breaths are caused by carbon dioxide irritation of the inspiratory neurons of the respiratory

center. Due to deep breaths, the chest expands sharply, which leads to a significant, compared to normal, decrease in negative pressure in the pleural cavities.

The pronounced negative pressure in the pleural cavities makes it difficult for blood to pass into the left half of the heart and further into the arterial system. At the same time, the right heart is overflowing with blood together with the superior and inferior vena cava. the pumping force of the heart is insufficient to push blood through the lungs.

Due to the overflow of blood in the venous system, especially the superior vena cava, due to the absence of a developed valve apparatus in it, cyanosis and slight swelling of the soft tissues of the face and neck develop. As a result of a decrease in the flow of blood into the systemic circulation, blood pressure falls and venous pressure rises.

The lack of oxygen primarily affects the activity of the central nervous system. As a result of a critical decrease in blood oxygen, a prohibitively protective inhibition of the central nervous system develops, which can manifest itself in a disturbance of consciousness by the type of stunning.

2. Stage of expiratory dyspnea characterized by the predominance of expiratory movements. The beginning of this stage is associated with the involvement of the respiratory center in the process of excitation of expiratory neurons. This is a kind of desire of the body to get rid of excess carbon dioxide.

In the stage of expiratory dyspnea, the chest decreases sharply in volume, the pressure in the pleural cavities increases, which leads to a decrease in the suction capacity of the chest, due to the lack of adequate expansion. This promotes at first the expulsion of blood from the lungs into the left heart and then into the systemic circulation. As a result, blood pressure rises and venous pressure decreases for a while.

The lack of oxygen in the blood entails a change in the chemistry of muscle tissue and leads to the appearance of clonic-tonic seizures, turning into opisthotonus (in those who took alcohol before death, the severity of seizures is much less). Deepening of brain hypoxia with the spread of the inhibition process to the subcortical structures entails an involuntary eruption of feces and urine. This phenomenon is explained by the simultaneous development of paralysis of the corresponding sphincters, increased intra-abdominal pressure and increased peristaltic contractions of the intestine, due to the development of seizures.

Often, in the stage of expiratory dyspnea, involuntary ejaculation is observed, accompanied by orgasmic sensations. This fact can be easily explained if we remember that the center of sexual arousal is located in the immediate anatomical proximity to the respiratory center.

This circumstance contributes to the irradiation of excitation from the neurons of the respiratory center to the center of sexual arousal, which entails a corresponding reaction. In women, there may be a discharge of Kristeller's mucous plug from the cervical canal.

During the period of seizures, as a result of the involuntary collision of certain parts of the body with surrounding objects, additional damage may occur, which can be mistaken for traces of struggle and self-defense.

3. Stage of short-term respiratory arrest...Further deepening of hypoxia leads to overwork and depletion of the respiratory center. As a result, the rhythmic activity of the respiratory center stops, and respiratory movements stop for a short period of time. Blood pressure decreases even more, heart rate decreases, reflexes fade away, pupils dilate.

The duration of this stage is approximately 30-40 seconds.

4. Stage of terminal breathing...It is caused by the excitation of the respiratory parts of

the spinal cord. Breathing is restored, but respiratory movements are erratic and are characterized by rare deep convulsive sighs with the involvement of accessory muscles and often with a wide opening of the mouth. Exhalation is passive. Inhales are accompanied by synchronous bursts of blood pressure, which by the end of the stage decreases to a critical level.

The duration of the stage is 1.5 - 2 minutes.

5. Final cessation of breathing... The heart still works for some time (for several minutes), then stops and clinical death occurs.

By the end of the intravital course of asphyxia, functional changes become organic, with a pronounced pathomorphological picture.

Signs of asphyxiation on the corpse.

If asphyxia went through all its classical stages during life, then all general asphyxia signs will be expressed sharply and fully. If a perversion of classical asphyxia is observed, the signs of asphyxia are less pronounced, and sometimes they may be absent altogether.

General asthma signs can be divided into external and internal**...**

External signs:

- *Minor hemorrhages in the connective membrane of the eyes (subconjunctival ecchymosis*), arising from rupture of blood vessels as a result of a sharp increase in intracapillary pressure. With prolonged asphyxia, hemorrhages are formed in the skin of the eyelids, face, neck and upper chest. This is a rather significant sign of asphyxia, but not permanent: sometimes there are many ecchymosis, sometimes they are not, sometimes they are single.
- *Cyanosis of the face and some of its swelling*observed in the first minutes and often remains after death. However, if the corpse lay face up for several hours, then the blood flows downward, and the cyanosis disappears. On the contrary, if the corpse was lying face down, then the face takes on a blue-purple coloration, similar to cyanosis, even if it was pale at the time of death (cadaveric spot!).
- *Spilled, profuse dark purple cadaveric spots*... This symptom is due to the liquid state of the blood, as well as in vivo darkening of the blood, due to the accumulation of carbon dioxide. This sign cannot be considered a reliable sign of asphyxia, because it is observed in any other kind of rapid death.

This actually exhaust the external signs of asphyxia, which are of any significance. The rest of the signs are either inconsistent, or nonspecific, or practically they cannot be observed, for example, a slower cooling of a corpse.

- *Involuntary urination, excretion of feces, semen*...

It is far from always observed: they do not exist if the rectum and bladder are empty during the development of asphyxia, moreover, the poured urine can dry out and its traces are difficult to notice. In addition, these phenomena are observed in other types of death.

Traces of sperm in the circumference of the penis on male corpses, squeezing out a drop of semen from the external opening of the urethra is caused by ejaculation during asphyxiation.

In addition, the more rapid development of muscular rigor mortis and early decay should be attributed to the external signs of asphyxia.

Internal signs:

- *Liquid state of blood*very characteristic of asphyxia, but almost always occurs in other types of rapid death. This can be explained in terms of the enzyme theory. The amount of fibrinogen required for the normal clotting process is determined by the presence of fibrinogenase in the blood, which is produced in the lungs. Fibrinogenase is most active with a

rapid onset of death, in connection with which there is an accelerated destruction of fibrinogen and the blood does not clot.

- *Overflow of blood in the right heart* associated with difficulty in the outflow of blood from the pulmonary circulation and primary respiratory arrest with continued work of the heart.

- *Venous congestion of internal organs*... It is often observed with other types of death and not always with asphyxia. It is easily explained by the retention of blood in the right heart, as a result of which the outflow of blood from the internal organs is hindered.
- *Tardier spots*- clearly delimited multiple small-point (2-3 mm in diameter), intense dark red, hemorrhages. Localized mainly under the interlobar and diaphragmatic pleura and on the posterior surface of the heart under the epicardium. In their origin, play a role:

1. Increased intracapillary pressure due to stagnation in a small circle and increased intravenous pressure.
2. Increased permeability of the capillary walls due to hypoxic disorders.
3. Negative pressure in the pleural cavities, which contributes to the expansion of the vessels of the microvasculature.

These conditions are created in the stages of inspiratory dyspnea and terminal breathing.

Alveolar emphysema of the lungs characterized by a sharp swelling of the alveolar structures of the lungs due to sharp fluctuations in intra-alveolar pressure in the stages of inspiratory and expiratory dyspnea.

- *Spleen anemia (sign of Sabinsky*). It is explained by the vasoconstrictor effect of asphyxial blood. The symptom is very inconsistent, because the spleen is very susceptible to various influences and can be enlarged and full-blooded.

Each of these signs is not specific for asphyxia, because they happen with a quick death, but in their totality and in combination with particular signs, they testify to it.

Hanging.

Hanging - compression of the organs of the neck by a loop under the influence of the gravity of the whole body or its parts, sometimes between objects or by pressing.

Distinguish between complete (free) and incomplete (non-free) hanging.

When fully suspended, the body hangs freely in a noose without a fulcrum.

Incomplete hanging can occur while standing, kneeling, sitting, lying. There are cases when neck compression was observed in a fork in a tree, between fence boards. The role of a squeezing object can also be played by the back of a chair, the crossbar of a table or stool with the appropriate position of the head, the weight of which is sufficient for death.

The hanging tool is a noosethat tightens the neck.

According to the device, the hinges can be sliding and fixed. The latter do not tighten around the neck and, in turn, are divided into open and closed. Closed loops are tied near the neck, open loops are a ring into which the head freely passes.

Sliding loops (noose loops) tighten tightly around the neck, squeezing it.

The hanging position of the loop may vary. As a rule, when hanging, it has an obliquely ascending direction towards the node.

Distinguish:

– ***Typical position***loops when the knot is located at the back of the neck or back of the head. The loop of the loop in front is located in the area of the thyroid cartilage or above it

(between it and the hyoid bone). Thus, the loop has an oblique ascending direction from front to back, from bottom to top.

– ***Atypical position...*** In turn, it is divided into anterior (the node is located in the chin area) and lateral (the node is located on the right or left on the lateral surface of the neck).

Genesis of death by hanging.

In the genesis of death by hanging, compression of the neurovascular bundle of the neck, including the carotid artery, the internal jugular vein and the vagus nerve, plays a significant role.

With a light pressure, in cases of incomplete hanging of the body, the loop completely squeezes only the internal jugular veins, through which the bulk of blood flows from the cerebral sinuses, while the carotid and vertebral arteries are not completely compressed. Therefore, blood flow to the brain, while the heart is working, continues, while the outflow is impaired, which leads to a sharp increase in intracranial pressure.

With a stronger compression of the neck, in cases of complete hovering, the carotid, and even the vertebral arteries are compressed (for clamping the latter, an effort equivalent to 15-20 kg is needed), which causes instant acute anemia of the brain (because there is no inflow or outflow).

Irritation of the vagus nerve due to stretching of its trunks and compression of the upper laryngeal branches causes a slowdown in the work of the heart, and sometimes its complete stop. With a sick heart, this alone can lead to death. A similar effect causes irritation of the carotid sinus zone.

It is assumed that the stretching of the spine along the longitudinal axis and compression of the medulla oblongata by the odontoid process of the 2nd cervical vertebra play a role in the onset of death at full hanging.

In a certain number of cases of asphyxia in the genesis of death, a factor such as aspiration of food masses from the stomach becomes important (when the loop node is located in front or on the side of the neck, the esophagus remains patent and vomiting caused by irritation of the vagus nerve can also lead to asphyxia).

Due to the complete cessation of conscious movements due to edema of the cerebellar basin, and a rapid loss of consciousness, self-help is impossible and the person who hanged himself cannot free himself from the tightened loop.

Pathomorphology of hanging.

The main sign of neck compression when hanging is a strangulation groove - a superficial injury to the skin of the neck, which is a negative loop trace, morphologically representing an abrasion of the skin of the neck.

The strangulation groove is a grooved depression with a bottom, walls, upper and lower edges (ridges). Sometimes, in the presence of several compression elements, intermediate rollers are also observed.

Signs of a strangulation groove when hanging:

1. Located in the upper third of the neck (usually between the thyroid cartilage and the hyoid bone).
2. Has an oblique ascending direction towards the node.
3. Unevenly expressed.
4. An open groove (between its ends, an interval of intact skin is determined, corresponding to the place of loose contact with the loop material).
5. Sometimes when separating a skin flap in the projection of the SB, on its inner surface, the

so-called "internal" or "silver" strangulation groove (Casper's sign) is determined, which is a whitish silvery strip, which can be present even in the absence of an external SB.
6. The upper edge of the furrow is undermined, the lower edge is beveled.

The strangulation furrow should be well studied and described according to the plan:

1. Location of the furrow (can be at different levels of the neck: in the upper, middle or lower third, above or below the thyroid cartilage).

In addition, it is necessary to indicate the exact location of the strangulation groove on all surfaces of the neck. In this case, the location of the groove relative to the anatomical landmarks should be indicated. On the front surface of the neck - relative to the upper edge of the thyroid cartilage, on the lateral surfaces - it is necessary to indicate the distance from the upper edge of the strangulation groove to the angle of the lower jaw and to the apex of the mastoid process. On the back surface - the distance from the upper edge of the strangulation groove to the border of hair growth or the middle of the external occipital protuberance is indicated.

2. Direction... It can be horizontal or oblique.

3. The presence and severity of the rollers... Always between the individual elements (depressions) of the strangulation groove, rollers are formed from pinching the skin, narrow or wide, according to the width of the gap between the whorls.

4. The width of the groove on each surface of the neck. It depends on the width of the buttonhole. It should be remembered that the width of the furrow does not always correspond to the true width of the loop. This applies primarily to soft hinges. For example, a rope or towel loop can have different thicknesses, which is reflected in the width of the loop.

5. The depth of the furrow on each surface of the neck. Typically dependent on loop thickness and gravity. The narrower the loop, the greater the body weight and the duration of the hovering, the more favorable the conditions for drying and compaction, the deeper and more pronounced the strangulation groove. Wide soft loops form wide, pale grooves, sometimes poorly visible, or even invisible to the eye.

The depth of the strangulation groove on the corpse does not always correspond to the depth of the groove on the neck during hanging. In the latter case, the furrow is, of course, deeper, and after removing the loop, the skin on the corpse is somewhat leveled.

6. The bottom of the furrow... It is described on each surface of the neck with an indication of its density and color.

The density of the furrow may not differ from the density of the surrounding skin (soft loops), or, conversely, it may have a parchment density due to sloughing of the surface layers of the epidermis, followed by drying (rigid and semi-rigid loops). In the first case, the furrow has a pale pinkish-yellowish color, in the latter it is yellow-brown or even dark brown.

7. Presence of hemorrhages, abrasions, fibers or microparticles of the loop material along the strangulation groove. To detect particles of the loop material, examine the strangulation groove using an operating microscope or magnifying glass. If necessary, microparticles are removed with adhesive tape, applying it to the furrow. The need for this arises in the case when the loop was removed before the arrival of the investigator or expert and it is necessary to answer the question of what the loop was made of. In this case, you should also carefully examine the palmar surfaces of the corpse's hands, on which fibers and microparticles of the loop material can also be found.

In addition, during external examination of the corpse, it is necessary to measure the neck

circumference at the level of the strangulation groove (to determine the degree of compression of the neck by the loop) and the length of the body with the right arm extended upward (to confirm or refute the possibility of self-hanging).

After studying and describing the strangulation groove, it is photographed with a scale on different surfaces of the neck.

Signs of a lifetime of hanging.

One of the main questions in the study of a corpse removed from the loop is to establish the intravital or postmortem origin of the strangulation groove.

The presence of a groove in itself does not mean that death occurred from hanging, for a corpse may have been hanged, and a typical strangulation groove may form on its neck.

Signs of a lifetime of hanging include:

1. *Brown shade* strangulation furrow, indicating the presence of intravital sedimentation.
2. *Hemorrhages along the strangulation groove*or in rollers of strangulated skin. For this, the skin is removed from the SB area. Moreover, this must be done before opening, tk. an incision in the skin leads to the leakage of blood from the vessels and saturation of it with the skin, SFA, muscles and creates a picture of intravital hemorrhages. The skin for examination should be taken from an area outside the cadaveric spot. In this case, the removed piece of skin must include the upper and lower ridges of the groove and be limited from above and below to unchanged skin. The edges of the furrow should be marked, for this a piece of skin should look like a trapezoid, the smaller base of which corresponds to the upper edge, and the larger one corresponds to the lower edge of the furrow.

Next, the Bocarius test is performed... The PFA is carefully separated from the taken piece of skin, a piece of skin is placed between two glass slides, slightly squeezing it, and examined in transmitted light. At the same time, attention is paid to the presence of dilated small vessels and hemorrhages at the edges of the groove and the absence of these changes in the bottom area.

A similar examination of the removed skin flap can be performed using a stereoscopic microscope. In this case, the difference in the blood filling of the vessels of the upper and lower ridges is clearly visible. Venous plethora is noted in the upper ridge and arterial in the lower ridge.

3. *Hemorrhages in the subcutaneous fat and muscles in the projection of the strangulation groove.*
4. *Fuchsin triad - when stained by Lee's method, the following colors are stained: 1. flattened epidermis. 2. papillary dermis. 3. muscle fibers.*
5. *Hemorrhage into the thickness of the tip of the tongue when it bites during convulsions.*
6. *Hemorrhages in the subcutaneous muscle of the neck and legs of the sternocleidomastoid muscles in the places of their attachment to the clavicle and sternum (Walcher sign).*
7. *Hemorrhages in the anterolateral parts of the intervertebral discs of the lumbar and lower thoracic spine (Simon's sign).*
8. *Sprains and hemorrhages in the ligamentous apparatus of the spinal column, mainly of the cervical spine.*
9. *Hemorrhages in the Schlemm canal (it is located in the iris-corneal corner, in the area of the cornea-sclera transition).*
10. *Hemorrhages in the carotid zone (Musset sign).*

11. Hemorrhages in retrobulbar tissue.

12. Hemorrhages in the posterior wall of the pharynx and retropharyngeal tissue (Bruardel sign).

13. Hemorrhages in the adventitia of the carotid arteries (Martin's sign).

14. Hemorrhages in the lymph nodes located above the level of strangulation (chin, submandibular). The lymph nodes themselves are enlarged, rather dense, their vessels are injected.

15. Bleeding from the nose and external auditory canals.

*16. Tears of the intima of the carotid arteries (sign of Amyuss).*They are formed as a result of overstretching, have a linear shape and are located transversely, most often on the posterior wall of the carotid arteries below the level of strangulation. The frequency of detection of this symptom is low, which may be explained by the fact that intimal tears are poorly visible to the naked eye, because are small, and there are no hemorrhages at the sites of tears. Therefore, sometimes staining with neutral dyes can be used to visualize intimal tears when hanging. To do this, ink or black gouache is applied to the inner surface of the opened carotid artery. In this case, the particles of the coloring matter penetrate into the depth of the tears of the intima. Remove excess paint with water. Then the intima is examined with the naked eye or using a stereomicroscope.

17. Fractures of the cartilage of the larynx or hyoid bone with hemorrhage into soft tissues in the areas of fractures.

18. Anisocoria with strong, predominantly unilateral compression of the neck with a loop.

19. The signs of a lifetime of hanging should undoubtedly include all signs of a quick death. (see above).

However, it should be noted that if the body of a person who died from any other cause is immediately hanged (during the period of clinical death), then all the above signs will be expressed as well as in cases of intravital hanging. Therefore, it is always very important to compare the data of the external and internal examination of the corpse with the circumstances of the case and the results of the inspection of the scene.

Sometimes you have to decide whether the body was hanging or not. Such a need arises in cases where it is necessary to carry out a differential diagnosis between hanging and strangling with a noose or to reconstruct the circumstances of an event that took place.

A number of signs, revealed during external and internal examination of a corpse, allow to answer this question.

Signs of body hanging.

1. Sign of Amyuss.

2. Oblique ascending direction of the strangulation furrow...

3. Cadaveric spots are located in the lower parts of the body (an indirect sign), especially pronounced on the forearms, hands, legs and feet (like gloves and stockings). This sign is observed when the body was in an upright position for at least 8 hours during the formation of cadaveric spots, i.e. in the stages of hypostasis and stasis. When the body is removed from the loop within 24 hours after the onset of death, cadaveric spots change their original localization and appear in a new place.

If the body was suspended a day after death, in the imbibition stage, the formation of cadaveric spots on the distal extremities does not occur, they retain their original position (on the back of the body, if the corpse was in a horizontal position before the suspension).

5. Hypostases in the serous membrane of the loops of the ileum, blind, sigmoid colon... The presence or absence of this sign also depends on the period of stay of the corpse in the loop, the position of the body at the time of death (indirect sign).

6. Hemorrhages in the anterolateral parts of the intervertebral discs of the lumbar and lower thoracic spine (Simon's sign).

7. Sprains and hemorrhages in the ligamentous apparatus of the spinal column, mainly of the cervical spine.

8. Ruptures of the intervertebral discs of the cervical spine (due to a jerk - a quick transition from the existing point of support to free hanging and large body weight of the victim).

9. Hemorrhages in the carotid zone.

Strangling with a loop.

Strangulation with loops - compression of the organs of the neck by a loop, which is tightened under the influence of an external force (often by someone else's hands, occasionally with the help of any objects (twists), parts of moving mechanisms and machines), and not by the weight of its own body

The mechanism of action of the loop on the neck when strangling is the same as when hanging. However, damage to the soft tissues and organs of the neck under the strangulation groove is more common. This is due to the sometimes inadequate use of brute force, gusty repeated strong movements, which are usually intensified at the time of convulsions, tk. the latter are regarded as resistance.

Therefore, strangulation by a loop is often accompanied by fractures of the thyroid cartilage, which is accompanied by irritation of the superior laryngeal branches of the vagus nerve and death due to reflex cardiac arrest.

Differential diagnosis of death as a result of hanging and strangulation by a loop is made on the basis of an analysis of the morphological features of the strangulation groove. When strangling with a loop, the strangulation groove is usually located at the level of the middle or even lower third of the neck, in the projection of the lower edge of the thyroid cartilage, or even lower - at the level of the cricoid cartilage. The direction of the furrow is horizontal or close to it. Regardless of the location of the knot, the pressure of the loop is the same throughout the entire length, so the groove on all surfaces of the neck will be evenly pronounced. In addition, the groove in most cases is closed, except when soft objects (collar of clothing, scarf, beard) fall under the loop.

When strangled by a loop, there are no signs of body hanging. Due to the uniform compression of the neck organocomplex during strangulation with a loop, the phenomenon of anisocoria is absent.

Questions resolved by the forensic medical examination when hanging and manually strangling the noose:

1. How the loop was tightened - by gravity or by hand.

2. Was a noose put on the neck of the deceased after death.

3. How the loop was tied and applied.

4. What should be the properties of the loop, judging by the properties of the strangulation groove.

5. Are there abrasions, bruises, or other injuries indicative of struggle and self-defense before death?

6. If this is a hanging, then could it have been carried out by another person.

7. Is death the result of an accident, and how it could have happened.

Strangulation by hands.

When crushed by hands, the neck is squeezed with one or two hands. Sometimes compression of the neck is carried out by other parts of the body (knee, foot, forearm, any object).

The following options for squeezing the neck with the hands are possible: right, left, two. These options can be when the neck is squeezed from the front, back, right, left.

When squeezed by hands, the neck is subjected to static and dynamic loads.

Along with the compression of the vessels and nerve trunks of the neck, there is a decrease in the lumen of the trachea, sometimes complete closure of the glottis when pressing on the larynx from the sides.

From the squeezing of the hands on the neck, various injuries occur:

1. *Rounded spotted bruising*on the skin of the anterolateral surfaces of the neck from the pressure of the fingertips. Here the following options are possible: one bruise on the right, 3-4 - on the left - strangulation was performed with the right hand; one bruise on the left, 3-4 on the right - strangulation was performed with the left hand.

Multiple bruises on the right and left indicate that the killer acted with two hands. If the child's neck was compressed, the bruising may be localized on the back of the neck. an adult's hand completely wraps around a child's neck.

2. *Multiple abrasions, linear and crescent shape* 1 to 2 cm long from the action of the nails.

However, these injuries are characteristic of compression of the motionless neck, when a person does not offer resistance due to loss of consciousness, severe alcohol intoxication, deep sleep, or other helpless state. Most often, the victim resists, and the above injuries have the form of multiple abrasions and bruises of an irregular oblong shape, located in different directions and layering on top of each other. This is due to the fact that with repeated grasping of the neck, during the fight, the places of pressure change and new injuries arise.

As a rule, most of the hemorrhages are found not on the skin, but in the PZhK and intermuscular tissue, muscles in the projection of the bruises, in the root of the tongue, and in the lobe of the thyroid gland.

Abrasions and bruises can also be located on the skin of the face - around the nose and mouth. This happens when the mouth is closed in order to drown out the cry or to close all the airways.

3. *Fractures of the cartilage of the larynx and hyoid bone are much more common,* since the hand presses directly on them with great force. Therefore, when strangulated by hands, reflex cardiac arrest is possible due to irritation of the zones of the carotid sinuses or the vagus nerve.

When the neck is compressed, the latter experiences a dynamic impact not only from the front, but also from the sides. As a result, the horns of the hyoid bone are compressed towards each other. The horns of the bone are bent inward and fractures are formed in the area of synchondrosis, i.e. at the junction of the horns with the body of the hyoid bone.

In cases where the hands are squeezed through soft objects, sometimes any damage to the skin at all, and it is not possible to detect any damage in the soft tissues of the neck.

Self-suppression with the help of hands is practically not possible, since weakness and impairment of consciousness develop very quickly, as a result of which the compression of the neck stops already at the very beginning of the self-suppression attempt.

General asthma signs when strangled by hands are less pronounced than when hanging or strangling with a loop, because strangulation by hands is not as long as when the loop is applied.

Questions resolved by forensic medical examination when strangulation by hands:

1. What is the mechanism of neck compression.

2. Whether the compression was done with one hand, and which (right, left) or two.

3. Whether the pressure was short-term or long-term, single or repeated.

4. Was there a struggle and self-defense before death.

5. Are there any signs by which it is possible to establish the features of the crushed hand (length and shape of nails, their defects).

Mechanisms and pathomorphology of damage to the hyoid bone, cartilage of the larynx and trachea with compression of the neck.

Hyoid bone injury

Currently, three mechanisms of damage to the hyoid bone (PC) are known:

1. Tension of the lateral thyroid-hyoid ligaments between the PC and the thyroid cartilage (PC fragments deviate downward).

2. Pressing the PC to the spine (deviation of the fragments outward and somewhat upward).

3. Compression of the PC from both sides (deviation of the fragments inward).

When hanging, injuries to the PC arise from pressing the horns to the spine and from the tension of the lateral ligaments.

The first mechanism is characteristic of the atypical location of the loop. In this case, due to the divergence of large horns, fractures occur in the area of articulation with the body or in the distal third.

Injuries arising from the tension of the lateral thyroid-hyoid ligaments are more common in the typical location of the loop, when the neck is compressed at the level of the thyroid-hyoid membrane. At the same time, due to the indentation of the loop between the thyroid cartilage and the PC, the distance between them and the degree of tension of the lateral thyroid-hyoid ligaments increase. The fracture line runs along the border of the articulation, and the fragment deviates downward and somewhat inward.

When the emphasis on the spine is combined with the tension of the ligaments, the deflection of the fragment occurs in the lower-outer direction.

When the loop is strangled, due to its almost horizontal effect on the PC, the mechanism of maximum pressing it to the anterior surface of the spinal column is realized. The resulting fragments of large horns in this case are directed outward and somewhat downward or upward, depending on the direction of the vector of the force of the loop. In this case, signs of compression are found on the outer surface of the articulation area, and the tension zone - on the inner one.

In the case of strangulation by hands, the injury of the PC from its compression on both sides predominates, as a result of which the fragments are directed inward. The compression zone of the bone substance is localized on the inner surface of the articulation, and the stretch zone on the outer surface. It should be noted that in every third case, compression of the PC in the frontal plane is combined with pressing it to the spine.

Thus, summarizing the above, we can draw the following conclusions:

1. The large horns of the PC are normally directed backward, somewhat outward and upward.

2. Directly to the great horns from below is attached the thyroid-hyoid membrane, laterally passing into strong lateral ligaments.

3. Deviation of the PC horns downward is possible only when the lateral thyroid-hyoid ligaments are pulled.

4. Deviation of the PC horns upward and outward - due to the pressing of the PC to the spine.

5. The inward displacement of the horns is due to the compression of the PC from the sides.

Consequently, in the direction of deflection of fragments of the horns of the PC in one direction or another, it is possible to establish the mechanism of PC trauma and the direction of the loop action.

Damage to the thyroid cartilage.

Damage to the upper horns and plates is more common. With typical compression of the neck organo-complex (especially in the ascending direction), there is some overturning of the thyroid gland with simultaneous tension of the thyroid-hyoid ligaments, thyroid-hyoid membrane and pressing the upper horns to the anterior surface of the spine. In this case, the fragments of the horns deviate anteriorly and somewhat inward, subject to the mobility of the cartilaginous plates and only anteriorly, if the ossified plates are connected motionlessly.

Atypical compression is characterized by lateral displacement of the thyroid gland with unilateral tension of the ligaments and membrane and more frequent damage to the superior horn on the side of the predominant action of the loop. Depending on the level of compression, the fragment is deflected either inward or outward.

Compression of the neck at the level of the thyroid gland is also accompanied by damage to its plates along the midline or in their medial part on the right or left. When compressed on both sides, the signs of stretching are localized on the outer surface of the plates, the compression zone - on the inner one. Due to pressing and resting on the spine, the fracture line of the plates is characterized by the presence of signs of stretching on the inner surface and a compression zone on the outer surface of the plates.

Damage to the cricoid cartilage.

The impact on the cricoid cartilage is possible only in the anteroposterior or anterolateral directions. In this case, local damage occurs in the form of local cracks. Indirect damage is possible on the lateral surface of the arc or plate on the right or left with signs of stretching on the outer surface and signs of compression on the inner.

Damage to the trachea.

Damage to the hyaline half-rings of the trachea is possible when exposed to the sagittal plane (emphasis on the spine). They look like vertical cracks, are more often indirect and are located on the outer surfaces to the right or left. This kind of damage is characteristic of the 1st, 2nd, and 3rd half rings, not covered by the sternum.

Signs of compression on both surfaces of the fracture zone testify to the repeated and multidirectional impact on the neck; in the stretch zone with repeated exposure to the neck, signs characteristic of the compression zone appear.

Compression asphyxia.

This type of mechanical asphyxiation is the result of compression of the chest, abdomen

or chest and abdomen at the same time, by any heavy blunt objects, for example, a concrete wall, a car.

Genesis of death...It is important to consider the strength, area and direction of the compression. With compression in the anteroposterior direction, asphyxia occurs faster. For death to occur, compression of one half of the chest, or only the abdomen, is sufficient. Isolated compression of only one chest leads to a sharp restriction of respiratory excursions and the onset of death with symptoms of slow asphyxia for 15-20 minutes. In this case, the movement of one diaphragm compensates for the immobility of the chest for some time, but cannot provide sufficient expansion of the lungs. Simultaneous compression of the abdomen leads to a restriction of the movement of the diaphragm and a more rapid onset of death (after 6-8 minutes). In the latter case, the general signs will be less pronounced than with prolonged dying. With isolated compression of the abdomen, even for 1 hour,

Compression of the chest and abdomen leads not only to a restriction or complete cessation of respiratory movements, but also to a sharp disruption of blood circulation in the lungs and brain. The severity of signs of asphyxia death depends on the strength and duration of the compression.

At the same time, along with general asphyxial signs, there are also specific ones:

1. Ecchymotic mask (Olivier-Danger trait). The skin of the face, neck, upper body is sharply cyanotic (sometimes with a cast-iron tint) with many small and large hemorrhages in the skin and in the connective membranes of the eyes. The eyeballs protrude from the orbits, the neck veins are full of blood. Hemorrhages are also found in the thickness of the root of the tongue, in the lymph nodes of the upper body, thyroid and salivary glands, in the muscles of the neck, chest and abdomen.

The mechanism for the development of these changes is associated with the fact that the jugular and unnamed veins do not have valves, and a sharp increase in pressure in them leads to rupture of the terminal sections of these veins. The valve apparatus of the axillary and inferior vena cava is a barrier, so cyanosis and hemorrhages do not extend to the upper extremities and lower body.

2. *Carmine pulmonary edema (sign of Lakassan).* The lungs are enlarged, edematous, moderately airy and have a bright red (carmine) color from the surface and on the cut due to a sharp overflow of blood vessels with arterial blood.

The appearance of this symptom is explained by the retention of oxygenated blood in the lungs due to the restriction of respiratory movements, which normally promote the expulsion of blood from the lungs into the pulmonary veins and further into the left atrium. Edema of the lung tissue is explained by the stagnation of a large amount of blood, an increase in intracapillary pressure as a result and transudation of plasma into the interstitial space.

This symptom is not common, and there is no carmine coloration of the lungs with simultaneous compression of the chest and abdomen. At the same time, in cases of compression of only the chest, this sign is pronounced, which is associated with a prolonged onset of death.

To identify "carmine edema", the color of blood from the lungs is compared with the color of blood in other organs, for example, the liver, where the blood has a dark bluish, almost black color.

3. *The sign "dominoes".* In the area of direct pressure, blood is mechanically squeezed out of the vessels, and it looks pale in comparison with the area outside the pressure zone, which has a cyanotic color, due to the accumulation of displaced blood.

4. *Prints of a pattern of fabrics of clothes and accessories* (buttons, buttons, buckles) on

the victim's body.

5. Damage to soft tissues on the body surfaces opposite to compression.

Forced compression of the chest and abdomen can be accompanied by rupture of the alveoli and the release of air under the visceral pleura with the formation of bullous emphysema (with slow compression, air is forced out naturally - through the bronchi and trachea).

The diagnosis of "compression asphyxia" is valid only in cases when the examination of the corpse did not reveal any traumatic injuries incompatible with life, which in themselves can cause death (spinal fractures, multiple rib fractures, ruptures of internal organs and large blood vessels) ...

Closing the openings of the mouth and nose.

Closing the openings of the mouth and nose is usually done with some soft objects (handkerchief, sheet, pillow, palm), since it is impossible to close the respiratory openings with a hard object due to the fact that this is prevented by the relief of the face (protruding nose) and the mutually perpendicular arrangement of the respiratory openings.

As a result of such obturation, a typical picture of mechanical asphyxia develops without reflex and vascular influences and changes.

This type of obstructive asphyxia is very difficult to diagnose, because often does not have any external signs (if the victim did not resist), there are only signs indicating rapid death (sharp plethora of internal organs, liquid dark blood, multiple hemorrhages: subpleural, subconjunctival).

In this case, the expert cannot substantiate the conclusion that death occurred precisely from mechanical asphyxia, and in particular, due to obturation of the openings of the nose and mouth with soft objects.

In this regard, information about the circumstances of the case, data from the examination of the corpse at the place of its discovery, is of particular importance, allowing the expert to initially suspect asphyxiation from closing the mouth and nose openings with any soft object or hand and purposefully conduct a forensic medical examination of the corpse.

If the openings of the nose and mouth were closed with hands, and during the strangulation the victim resisted, then on the skin of the face in the circumference of the mouth and nose (on the cheeks, chin, in children in the area of the ears), you can find small linear and semilunar abrasions from the action of the nails, and rounded bruising from the effects of the nail phalanges of the fingers.

If the external respiratory openings were closed with a soft object, then there will be no damage to the skin. However, in both cases, on the inner surface of the lips, as well as on the mucous membrane of the cheeks, teeth imprints, bruises or ruptures of the mucous membrane surrounded by bruises are usually found, especially in the projection of the canines, arising from strong pressing of the mucous membrane to the lips. Sometimes teeth fractures, flattening of the cartilage of the nose can be observed.

An equally important circumstance that testifies in favor of the violence that took place is the traces of struggle and self-defense (disorder in clothing and the environment, abrasions and bruises on various parts of the body).

Internal examination in the cavity of the nose, mouth and respiratory tract can be found parts of the objects that have closed the holes - fluff from a pillow, thread, hairs, particles of cotton wool, etc.

There are known cases of suicide by tying the face with soft objects (towels, scarves, sometimes with the application of cotton wool on the nose and mouth).

Aspiration of bulk substances and liquids.

Closure of the airway lumen can occur as a result of aspiration of bulk substances: sand, cement, flour, cereals, etc. Bulk substances penetrate deeply into the respiratory tract, as far as the particle size and caliber of the respiratory tract allow. In addition, they are found in the mouth, esophagus, stomach and intestines. bulk solids are not only inhaled but also swallowed.

The detection of particles of sand, grain, flour, etc. in the middle, small bronchi and even in the alveoli in combination with generalized bronchospasm, which can be established by histological examination, is of absolute evidence value for intravital aspiration. They can get into the trachea, into the cavity of the nose and mouth after death.

Usually, when the airways are closed by loose bodies, asphyxia proceeds much more slowly than in other cases, because bulk substances, especially large-bulk ones, contain a lot of air, and if the loose layer is not very thick, then weak breathing is possible, and death occurs after a few hours or even later. Repeatedly people, covered with earth, were removed after a few hours, albeit unconscious, but alive. In some cases, the aspiration of loose substances is combined with compression asphyxia, then death occurs much faster.

Aspiration of food masses into the respiratory tract is observed in persons who were unconscious as a result of open vomiting, during the production of resuscitation measures, with improper performance of artificial respiration, chest compressions.

The detection of food masses in the respiratory tract does not in itself indicate death from aspiration asphyxia. Food masses can enter the respiratory tract during the agonal period or even with rotting of the corpse. The detection of particles of gastric contents not only in the trachea and large bronchi, but also in small bronchi, bronchioles and alveoli can serve as an absolute proof of the lifetime of aspiration.

Macroscopically, the lungs are swollen, bumpy from the surface, particles of food masses are released from the small bronchi and alveoli on cuts with pressure.

Histological examination of lung tissue reveals a pronounced generalized bronchospasm, undigested muscle fibers, starch particles, plant cells in the lumen of small bronchi and alveoli. As a rule, for research, 3 pieces are taken from each lobe of the lung: root, central, peripheral.

Death from blood aspiration occurs in persons who are unconscious with TBI, damage to the soft tissues of the face, cut or stab-cut injuries of the neck. In this case, histological examination of lung tissue is important to verify the diagnosis. At the same time, a number of changes characteristic of this type of death are revealed. The bronchi and bronchioles are dilated, full of blood, a small amount of blood is also determined in the alveoli, in the mass of erythrocytes one can find fibrin threads, single leukocytes. Generalized bronchospasm is noted.

When assessing the microscopic picture, it should be borne in mind that the presence of blood in the alveoli is not necessarily associated with its aspiration, but may be the result of hemorrhages in the pulmonary parenchyma, due to a variety of reasons. Therefore, in diagnostics, a thorough examination of the lumen of the bronchi at various levels is of particular importance. In addition, blood can be found in the sinus of the sphenoid bone, where it enters as a result of a sharp increase in pressure in the nasal cavity.

Closure of the airway with a foreign body.

This variant of mechanical asphyxia has recently been encountered very often in persons under the influence of alcohol of various depths. In such cases, there is a closure of the airways with a large piece of poorly chewed food (a piece of meat, bread, lard, etc.), due to a decrease in the reflex sensitivity of the mucous membrane of the nasopharynx and oropharynx, as well as a violation of the act of swallowing due to the development of inhibitory processes in the bulbar

sections brain stem. Therefore, death occurs unexpectedly and quickly during a meal in a dining room, restaurant, or at home.

In children, a wide variety of small objects can get into the respiratory tract that the child takes into the mouth (buttons, beads, beans, nuts, pills, chips, and much more).

Diagnosis of this type of mechanical asphyxia is not difficult and is based on the detection of a foreign body in the larynx, trachea, bronchi, which tightly or partially obstructs their lumen, in combination with signs of rapidly occurring death. In this case, the opening of the lumen of the larynx or trachea is performed on the spot, before removing the organocomplex. A foreign body, its size, localization in the airways, and the degree of closure of the lumen must be described in detail. The condition of the mucous membrane of the respiratory tract is described, in particular, edema, congestion, damage. If death did not occur immediately, all signs of inflammation are revealed at the place of fixation of the foreign body. With a long stay of a foreign body in the trachea or bronchus, bedsores are formed.

Drowning and death in water.

Drowning is a process that is characterized by a combination of pathophysiological reactions and the penetration of water or other fluids into the respiratory tract and lungs.

Traditionally, drowning is understood as the fact that the body is completely immersed in water. However, in real life, drowning is not required at all; it is enough to dive only the head or even the face. Therefore, cases of drowning in small streams and ponds, barrels, baths and even puddles are quite possible. Such cases are considered as special cases of obstructive asphyxia (the cause of death is the aspiration of fluid and the closure of the airways by it).

Currently, there are four main types of drowning, which differ from each other, both in thanatogenesis and in morphological manifestations.

Drowning types:

1. *Aspiration type* (true drowning).
2. *Reflex* (syncope) type.
3. *Spastic* (asfix) type.
4. *Mixed type* (a combination of aspiration type with asphyxia, aspiration and reflex, asphyxia and reflex is possible).

Aspiration type.

True (aspiration) drowning is understood as its kind when water penetrates deeply and fills the airways and alveoli in huge quantities, sometimes reaching the volume of circulating blood. The volume of inspired water depends on its temperature (warm water is inspired in greater quantities), on the intensity of respiratory movements, vital capacity of the lungs, reflex sensitivity of the upper respiratory tract.

Diagnosis of true drowning.

1.*Permanent pinkish white fine bubble foam*, resembling cotton wool, at the openings of the mouth and nose, as well as in the lumen of the respiratory tract (sign of Krushevsky S.V.). The mechanism of its formation is explained by the mixing of mucus, surfactant, water and air during intense respiratory movements. Because the basis of the foam is mucus, it is characterized by sufficient elasticity and does not burst when touched. When pressing on the chest, its synchronous mobility is noted. The presence of foam is noted in the first 2-3 days after death. Microscopic examination of foam bubbles can often reveal foreign inclusions: sand, small algae, etc.

The detection of foam at the openings of the nose, mouth and in the airways is a valuable indicator of active respiratory movements during the drowning process....

*2.Acute lung distension (acute alveolar emphysema)*is a very valuable and evidence-based sign of the lifetime of drowning. The mechanism of formation of emphysema is simple and banal: water, like a piston, presses with great force on the air in the alveoli and bronchi. This is accompanied by a sharp and outrageous increase in intrapulmonary pressure, which leads to rupture of the alveoli and the penetration of water and air under the visceral pleura.

As a result, the lungs significantly increase in size and volume, completely fill the pleural cavities and press on the chest from the inside, as a result of which transverse grooves-imprints from the indentation of the ribs are visible on the posterolateral surfaces of the lungs.

Emphysema is clearly visible when the lungs lie freely in the pleural cavities. In cases of the presence of multiple adhesions that prevent the expansion of the lungs, their acute swelling is poorly expressed. From the surface, the lungs have a "marble" appearance: pink, gray, red areas alternate; the surface of the incisions also has a motley appearance with areas of atelectasis, plethora, hemorrhages. In many cases, a large amount of frothy bloody fluid drains from the surface of the lungs.

3. Spots of Rasskazov - Lukomsky (A. Paltauf)- vague hemorrhages with indistinct contours of light red color, up to 1-1.5 cm in diameter under the pulmonary pleura (they are not formed in sea water). In essence, these are modified Tardier spots. Their pallor and vagueness is explained by the dilution of the blood with water penetrating through the ruptured alveolar capillaries, followed by hemodilution and hemolysis. The spots of Rasskazov-Lukomsky disappear after the corpse has been in the water for more than 2 weeks.

4.Presence of drowning fluid in the sphenoid sinus(Sign of Sveshnikov V.A.) This sign is more characteristic of the spastic type of drowning, in which spasm of the glottis occurs due to the reflex effects of water on the laryngeal mucosa. In this case, water, having limited access to the respiratory tract, under pressure penetrates through the pear-shaped openings into the cavity of the sphenoid sinus, and through the pharyngeal openings of the Eustachian tubes into the tympanic cavity.

5.Hemorrhage in tympanic membranes, mastoid cells, mastoid caves, into the middle ear cavity. Hemorrhages look like free accumulations of blood or abundantly saturate the mucous membrane, which in this case is edematous, full-blooded, dark red (K. Ulrich's sign). The mechanism of their formation is associated with an increase in pressure in the nasopharynx, which, in combination with pronounced hypoxia, leads to an increase in the permeability of the vascular walls and the formation of these hemorrhages. Occurs in spastic drowning.

6 lymphogemia - the throwing of erythrocytes into the lymphatic thoracic duct. Laryngospasm leads to venous congestion in the vena cava system and venous hypertension, resulting in a retrograde flow of blood into the thoracic duct. A quantitative assessment of lymphogemia is performed using a Goryaev counting chamber with microscopy of the contents of the lymphatic duct. It is observed with an asphyxia type of drowning.

7. Fluid of the drowning environment in the abdominal and chest cavity (Moreau sign).

The symptom has a diagnostic value only during the first few hours, with the further stay of the body in water, water enters the pleural and abdominal cavity due to passive diffusion.

8 the presence of a large amount of liquid drowning environment(often mixed with sand, silt, algae) in the stomach and small intestine (Fegeerlund's sign). This circumstance is explained by the fact that drowning water is not only inspired, but also swallowed in large quantities.

9 Overflow of the left ventricle of the heart hemolyzed blood (Casper's sign). Mechanism: the drowning medium enters the bloodstream through the torn capillaries of the interalveolar septa and enters the left heart through the pulmonary veins. It was found that the freezing point of blood in the left and right halves of the heart will be different, which is determined by the method of cryoscopy.

10 the presence of diatom plankton in the blood and internal organs... Plankton are the smallest organisms of plant (phytoplankton) and animal (zooplankton) origin that live in the water of rivers, lakes, seas and other bodies of water. Each reservoir is characterized by certain types of plankton, which have specific differences. For the diagnosis of drowning, phytoplankton is of greatest importance, in particular diatoms, which have a silicon shell that can withstand exposure to high temperatures, strong acids and alkalis. Diatoms up to 200 microns in size, together with water, easily penetrate into the systemic circulation and with the blood flow are carried throughout the body, lingering in the parenchymal organs and red bone marrow. The probability of plankton penetration through the gastrointestinal tract mucosa and from the air is negligible. The discovery of diatoms in parenchymal organs and red bone marrow is objective evidence of drowning death. The presence of plankton only in the lungs indicates only the presence of the corpse in water, the amount of plankton in the internal organs depends on the duration of the dying period: the longer the agony, the more plankton. The size of the plankton particles is determined by the degree of rupture of the pulmonary alveoli. The absence of diatoms in the blood and in the internal organs of a corpse removed from the water does not give the right to completely refute the fact of drowning. Plankton may be absent in cases of obliteration of the pleural cavities, in cardiac arrest at the very beginning of drowning, in the absence of diatoms in the reservoir, or during drowning during the diatom minimum. The size of plankton particles is determined by the degree of rupture of the pulmonary alveoli. The absence of diatoms in the blood and in the internal organs of a corpse removed from the water does not give the right to completely refute the fact of drowning. Plankton may be absent in cases of obliteration of the pleural cavities, in cardiac arrest at the very beginning of drowning, in the absence of diatoms in the reservoir, or during drowning during the diatom minimum. The size of the plankton particles is determined by the degree of rupture of the pulmonary alveoli. The absence of diatoms in the blood and in the internal organs of a corpse removed from the water does not give the right to completely refute the fact of drowning. Plankton may be absent in cases of obliteration of the pleural cavities, in cardiac arrest at the very beginning of drowning, in the absence of diatoms in the reservoir, or during drowning during the diatom minimum.

Currently, blood is taken from the right side of the heart for research on diatom plankton.

In case of putrefactive changes in the corpse, the entire femur or humerus is also removed for examination.

In addition, water samples should be taken from the water body where the drowning occurred. Water is taken from the surface layer of water 10-15 cm deep, from the middle layers and at the bottom at the place of drowning, and if it is unknown, then at the place where the corpse was found.

The method for detecting plankton is rather complicated and boils down to the following. After grinding, the parenchymal organ or bone marrow is placed in a flask and poured with perhydrol, boiled in concentrated sulfuric acid, then treated in nitric acid. At the last stage, a small amount of perhydrol is added again to clarify. After these manipulations, all organic matter leaves, and only inorganic components remain, including the silicon shells of diatoms.

After repeated centrifugation, smears are prepared from the resulting sediment and examined under a microscope.

A fairly valuable method for diagnosing drowning is a crystal-optical study based on the detection of quartz-containing minerals (pseudoplankton) in the internal organs and blood of a corpse. Crystal-optical examination of ashed sections of internal organs in the polarized light of a microscope reveals quartz-containing mineral particles ranging in size from 2 to 15 microns in an amount from 25 to 100 in 10 fields of view. The amount of mineral particles depends on the degree of turbidity of the water and the length of the drowning period.

Signs of the corpse being in the water.

When examining corpses removed from water, the question often arises whether death occurred in water (from drowning or other reasons) or whether a corpse has already fallen into the water.

Therefore, one should distinguish between signs of drowning and signs of a corpse being in the water. The latter are expressed the more sharply, the more time the corpse was in the water, and are found both on the corpses of persons who died as a result of drowning, and on the corpses of persons who died from other causes and then fell into the water.

The presence of a corpse in water can be judged by the following features:

1. The presence of wet clothes, hair (of course, this feature has practically no evidentiary value, since a corpse outside any room could repeatedly fall under the rain).

2. Traces of silt, sand on clothes, especially in her folds, hair, natural anatomical holes.

*3. Sharp pallor of the skin*due to vasospasm of the skin. At the same time, there is a contraction of the muscles that lift the hair, followed by rigor mortis and the appearance of the so-called "goose bumps". These signs can be observed both when drowning and when a corpse falls into the water shortly after death.

4. Maceration of the skin... Maceration is the softening, loosening and swelling of the skin due to soaking it with liquid. As a result of these processes, the skin of the palms, soles, knees, elbows thickens, wrinkles, becomes pearly pale. Maceration of the skin of the palms is called by the household term "washerwoman's hands", which are formed after 2-3 days of the body's stay in water. After 2-3 weeks, the epidermis completely loses its connection with the dermis and is easily detached along with the nails (“death glove”). After separation of the epidermis, the brush looks pink smooth and is called a "sleek hand".

The rate of development of maceration depends primarily on the temperature of the water; in colder water, it occurs more slowly, in warm water - faster. The maceration process is accelerated in running water. Clothing, hand gloves and footwear retard the development of maceration.

5. Posthumous baldness". With a prolonged stay in water, the hair on the head loses its connection with the macerated skin and, starting from 10-20 days, is easily pulled out, after 20 days it separates spontaneously, and after 30-40 days

the head may become completely bald.

6. Pink coloration of cadaveric spots (due to loosening of the epidermis and facilitated diffusion of oxygen into the skin).

7. Algae fouling of a corpse... On average, after a week, you can find islets of algae in the form of a cannon, by the 11th day the islets merge into a thin layer and after 18-20 days they cover the body with a continuous cover. But after three weeks they completely disappear and after 8-10 days they appear again.

8. Damage to the body by aquatic inhabitants (fish, crayfish), which eat, first of all, the eyelids, nostrils, face.

Positional asphyxia.

Asphyxia due to a position of the body that prevents normal breathing.

For example, positional asphyxia as a result of crucifixion occurs as a result of overstretching of the chest under the weight of its own body and, accordingly, the development of expiratory respiratory failure.

To diagnose positional asphyxia, in addition to sectional data, it is necessary to take into account the circumstances of death, body position, etc. factors.

LITERATURE

MAIN LITERATURE

1. Forensic medicine: textbook / under total. ed. V. N. Kryukova. - 2nd ed., Rev. and add. - Moscow: Norma: INFRA-M, 2015 .-- 431 p. : tab., cx., ph.

ADDITIONAL LITERATURE

1. Meter, VN Rules of a forensic medical examination of a corpse and drawing up an expert opinion: study guide. manual for stud. 4 and 5 courses of all fac. honey. universities, trainee doctors, honey. forensic experts / V. N. Meter; V. N. Meter; UO "GomGMU", Dept. pathological anatomy. - Gomel: GomGMU, 2016 .-- 20 p.

2. Objects of research of biological origin in the system of investigative actions [Electronic resource] / E. A. Bazikyan, V. V. Kuchin, P. O. Romodanovskiy, E. Kh. Barinov - M.: GEOTAR-Media, 2014. - 104 s. - Access mode:**http://www.rosmedlib.ru/book/ISBN9785970428825.html**... - Date of access: 22.05.2017.

3. Forensic medicine and forensic medical examination [Electronic resource] / ed. Yu.I. Pigolkina - M.: GEOTAR-Media, 2014 .-- 728 p. : ill. - Access mode:**http://www.rosmedlib.ru/book/ISBN9785970428207.html**... - Date of access: 22.05.2017.

4. Shpak, V.V. Forensic examination of material evidence of biological origin: study guide. manual for 5th year students of all fac. honey. universities, trainee forensic physicians / V.V.Shpak; UO "GomGMU", Dept. pathological anatomy. - Gomel: GomGMU, 2017. - 31 p. : ill.

5. Shpak, VV Forensic medical examination of transport injury: textbook method. manual for students 4 and 5 courses of all fac. honey. universities, trainees of forensic medical. / V.V.Shpak; UO "GomGMU", Dept. pathological anatomy with a course of forensic medicine. - Gomel: GomGMU, 2015 .-- 35 p. : ill., ph.

ELECTRONIC DATABASES

1. Physician consultant. Electronic Medical Library = Consultant of the doctor. Electronic medical library [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". - Access mode: http://www.rosmedlib.ru/. - Date of access: 09.11.2017.

2. Student advisor. Electronic library of a medical university = Student consultant. Electronic library of medical high school [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". - Access mode: http://www.studmedlib.ru. - Access date: 09/05/2017

3. Scientific electronic library eLIBRARY.RU = Scientific electronic library eLIBRARY.RU [Electronic resource]. - Access mode: https://elibrary.ru/. - Date of access: 09.05.2017.

4. Oxford Medicine Online [Electronic resource] / Oxford University Press. - Access mode: www.oxfordmedicine.com. - Date of access: 05/09/2017.

5. Springer Link [Electronic resource] / Springer International Publishing AG. - Access mode: https: link.springer.com. - Date of access: 05/09/2017.