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Учреждение образования  
«Гомельский государственный медицинский университет»

Кафедра патологической физиологии  
Обсуждено на заседании кафедры  
Протокол №7 от 30.08.2017

**МЕТОДИЧЕСКАЯ РАЗРАБОТКА**  
Для проведения занятия со студентами  
3 курса ФПСЗС, обучающихся на английском языке  
по патологической физиологии

Тема: **Патогенное действие факторов окружающей среды на организм человека**

Theme: **Pathogenic effects of environmental factors on the human body**

Время 3 ак. часа

**Actuality of the theme.** The environment factors constantly affect a man. Inadequate nutrition, bad social conditions, toxins influences and other factors decrease organism resistance. Going in for sport, rational labour and rest routine guard of environment raise unspecific organism resistance. Some factors can be used in medicine for prophylaxy of the diseases. For example, hypothermia (hibernation) increases organism resistance to hypoxia and doctors use that at operation on heart and brain.

**Learning goals of the lesson:** to study mechanisms of action of environment pathogenic factors, etiology and pathogenesis of effects of ionizing radiation on the human body.

**Educational goals of the lesson:** formation of scientific outlook and theoretical basis of future specialists on the basis of fundamental knowledge and the latest achievements of pathological physiology.

**Objectives of the lesson:**

1. To know characteristics of environmental factors.
2. To study mechanisms of action of pathogenic factors on the organism, and consequences of their effects.
3. To know the mechanisms of cell damage by ionizing radiation, forms and stages of radiation sickness.
4. To know features of action of small doses of radiation.

**To repeat the following questions from related disciplines to ensure absolute mastery of the material:**

1. Physiological thermoregulation mechanisms (physiology discipline).
2. Types of ionizing radiation; characteristic of isotopes:  $^{131}\text{I}$ ,  $^{137}\text{Cs}$ ,  $^{90}\text{Sr}$  molecular mechanisms of action of ionizing radiation: indirect action, radiolysis of water, radiotoxins, direct action (medical physics discipline)
3. Structure of cell (histology, cytology, embryology disciplines).

**Control questions of the lesson:**

1. Actions of mechanical factors: general characteristics, pathogenesis and manifestations.
2. Actions of barometric pressure: general characteristics, pathogenesis and manifestations.
3. Actions of electric current: general characteristics, pathogenesis and manifestations.
4. Effects of high and low temperatures: general characteristics, pathogenesis and manifestations.
5. Mechanisms of action of ionizing radiation. Radiosensitivity of cells.
6. Pathogenic actions of chemical factors; exo- and endogenous intoxications.
7. Biological factors as causes of infectious diseases.
8. Psychogenic pathogenic factors. Iatrogenic diseases.

**Calculation of study time**

Total study time 3 ac.hours

№ п/п	Contents	Calculation of study time
1.	Introduction. Motivational characteristic of the theme	3 minutes
2.	Written control of students on the topic of the lesson	15 minutes
3.	Interviews with students about the topic of the lesson	60 minutes
4.	Self-managed student work	15 minutes
5.	Summing up the results of the lesson	5 minutes
6.	Decision of situational tasks	20 minutes
7.	Task for the next lesson	2 minutes

### Additional materials:

In the medical literature pathogenic environmental factors are called "devastating effect" (I.M. Sechenov), "extraordinary stimulus" (I.P. Pavlov), "stressors" (H. Selye), "extreme factors." Among them distinguish mechanical, physical, chemical, biological and social pathogenic factors.

The degree of pathogenicity of any environmental factors is relative and depends on the conditions of the body existence.

### DAMAGING EFFECT OF MECHANICAL FACTORS

Mechanical factors may have both local and general damaging effect on the body. The effect of their pathogenic action determined by the strength of this action (kg / cm<sup>2</sup>) (tension, compression) or in the form of kinetic energy of the mass moving at a certain speed (mV<sup>2</sup> / 2g) (kick, falling, bullet or other gunshot wound). Damaging effect of mechanical factors also depends on the state of reliability, strength or resistance of damaged structures.

**Stretching** – resistance to deformation and the ability to restore the initial state. Pathological processes and initial condition of the tissues effect on the elasticity of the tissues (eg, inflammation reduce elasticity and increase the risk of rupture, muscle at rest, more stretchable than a shrinking).

**Compression** – small in strength, but long-acting factors of compression can lead to soft tissue necrosis, atrophy (eg due to pressure of tumors).

As a result of prolonged pressure on the human body, soon after the liberation (decompression) arises – crush syndrome.

#### Crush syndrome

Crush syndrome develops as a result of crushing long limbs (usually lower) by ground, heavy objects, wreckage after earthquakes, **blockages** in the mines, etc., with duration of **compression more than 4 hours**. Prolonged (for 8-24 hours) stay in the affected one position (coma, poisoning, severe alcohol intoxication) can develop positional compression syndrome. In this case, own body weight press on the limbs.

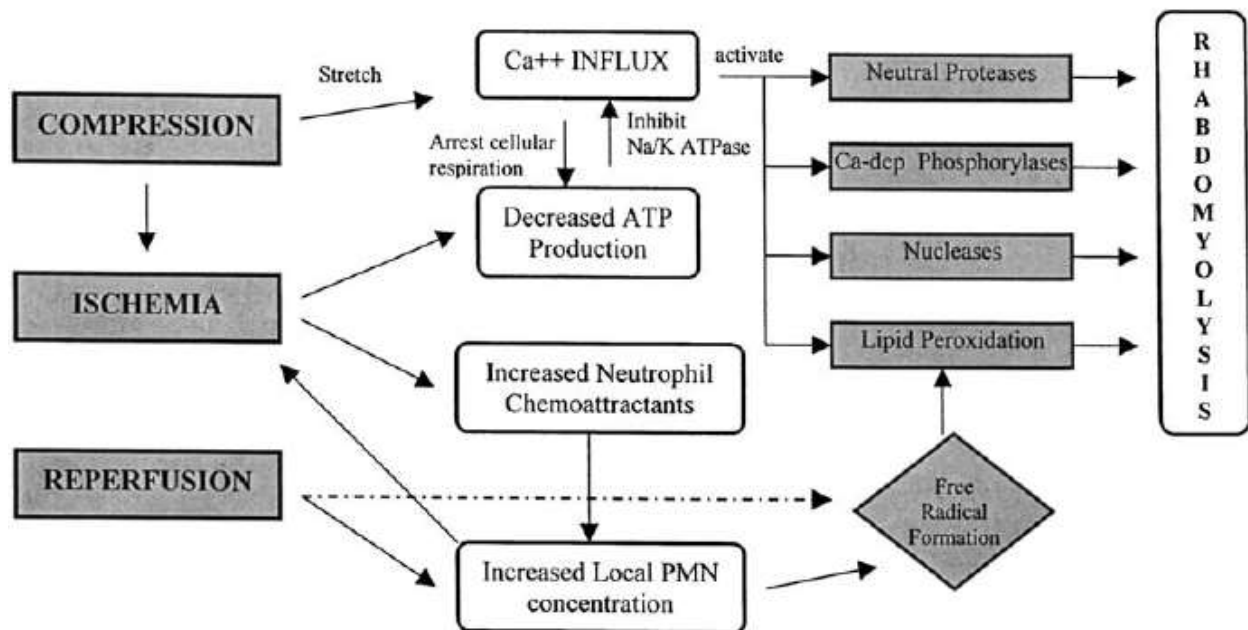
Three pathogenetic factors are important in the development of crush syndrome:

1. painful stimulus;
2. traumatic toxemia due to absorption of toxic products of tissues autolysis from the lesion;
3. plasma and blood loss (edema and hemorrhage in the area of crushed or long ischemic tissues).

Clinical severity depends on degree and duration of limb compression, volume and depth of the lesion, and combined damage to other organs and structures (traumatic brain injury, injury to the internal organs, bones, joints, blood vessels, nerves, etc.).

By severity there are four clinical forms of crush syndrome:

1. light (crush the individual segments less than 4 hours) – minor symptoms, the prognosis is favorable;
2. moderate (crush of both legs less than 6 hours) – there is a moderate impairment of renal function;
3. heavy (crush of both legs about 6 hours) – characterized by significant hemodynamic disorders. Possible death in the early and intermediate period;
4. extremely heavy (crush both legs more than 6 hours) – rapidly progressing clinical manifestations, patients die in the first or second day after the injury.



**Figure 1** – The pathogenesis of rhabdomyolysis (Malinoski D.J., 2004)

Three are 3 periods in the clinical picture:

**I period** (initial or early) – **hemodynamic disorders** (in 1-3 days after release from compression). After a few hours appear local changes in the compressed limb. It becomes pale, cyanotic fingers, rapidly increases swelling, the skin becomes woody density. Peripheral vascular pulsation is not defined. Progressively increase the symptoms of endogenous intoxication, plasma loss, hemoconcentration, creatinemia, proteinuria, cylindruria. Period is characterized by hemodynamic instability. General condition deteriorates with rise of local changes.

**II period** (intermediate) – **acute renal failure** (from 3-4 to 8-12 days). Swelling of the affected limb is enhanced by formation of bubbles with clear or hemorrhagic content, appear dense infiltrates with local and sometimes even total necrosis of the entire limb. Haemoconcentration replaced hemodilution, anemia increases sharply reduced urine output, up to anuria. In the blood, increases the amount of residual nitrogen, urea, creatinine, potassium, the picture of uremia. Body temperature increased. The patient's condition rapidly deteriorating, intensified weakness and confusion, there is a vomiting and thirst, jaundice of the sclera and skin. The fatality rate in this period is 35%.

**III period** (late period or local complications) begins in a 3-4 week. Normalized hemodynamic parameters, renal functions are restored. In uncomplicated cases, limb edema and pain end at the end of month. Perhaps the development of local (infection of open injuries) and general complications (generalization of infection with sepsis).

## PATHOGENIC ACTION OF ACCELERATION

In biology and medicine are the following types of acceleration:

1. Rectilinear acceleration – overload occurs when increasing or decreasing the speed without changing the direction.

In relation of vector to longitudinal axis of the body distinguish overload:

- longitudinal positive – from the head to the feet,
- longitudinal negative – from the feet to the head,
- positive transverse – from the chest to the back,
- negative transverse – from the back to the chest,
- positive sideward – right to left,
- negative sideward – left to right.

2. The radial and centripetal - occurs when changing the direction of the body motion, for example when the banking by plane, rotations of human in a centrifuge.

3. The angular acceleration - occurs when a non-uniform motion of a body in a circle, ie, by increasing or decreasing angular velocity.

Action of acceleration manifested by offset of soft tissue, organs in the direction of the inertial forces of deformation; unusual afferent impulses to the central nervous system, leading to the breakdown of its regulatory role. The leading place in the genesis of physiological changes during overload belongs to blood and tissue fluid.

In the action acceleration are distinguished:

- compensatory phase: increased frequency of heart rate, increased blood pressure, increased cardiac output and regional blood flow, increase lung ventilation, increased oxygen consumption, oxygen tension in the tissues of the brain, enhancing the function of several endocrine glands;
- decompensatory phase: the breakdown of compensation, bradycardia, arrhythmia, disorders of cardiac conduction, drop in blood pressure, impaired redox processes, disorders of the regulatory function of nervous and endocrine systems.

## EFFECTS ON THE BODY BAROMETRIC PRESSURE CHANGES

### Effect of reduced barometric pressure (hypobaria)

People found hypobaria while climbing in the mountains, on the ascent to altitude in leaky aircraft, spacecraft during accidents, in pressure chambers.

At an altitude 3000-4000 meters (corresponding barometric pressure 530- 460 mm Hg) is an expansion of gases and increase their pressure in enclosed and semi-cavities of the body, leads to irritation of the walls of the receptors, causing pain (especially eardrums and the mucous membranes of the middle and inner ear, maxillary and frontal sinuses).

At an altitude 9000 meters or more (which corresponds to 225 mmHg or below) occur symptoms of decompression. This is due to the transition of oxygen, and especially nitrogen dissolved in body fluids to a gaseous state. Formed bubbles of free gas (gas emboli), are spread by the vessels in different parts of the body, causing embolism. This in turn leads to the development of tissue ischemia. Especially dangerous are embolism of coronary artery and cerebrovascular disease.

High altitude (HA):

- High altitude: 1500 to 3500 m – high-altitude illness common with abrupt ascent to above 2500 m;
- Very high altitude: 3500 to 5500 m – most common range for severe high-altitude illness;
- Extreme altitude: 5500 to 8850 m – progressive deterioration of physiologic function eventually overcomes acclimatization.

### Acute mountain sickness (AMS)

Acute mountain sickness is caused by reduced air pressure and lower oxygen levels at high altitudes. Barometric pressure falls as altitude increases. As the barometric pressure decreases, the partial pressure of oxygen decreases proportionately. This condition is referred to as hypobaric hypoxia. It lead to:

- increased pulmonary ventilation;
- increasing the oxygen capacity of blood (ejection of RBCs from blood depot (spleen, liver), in prolonged state of hypoxia increased erythropoiesis);
- increase in the minute volume of circulating blood, acceleration of blood flow.
- at the tissue level – increased capillarity; increased myoglobin; improving the system of regulation of redox processes, and others.

Excess hypoxia stimulates the respiratory center as a result is hyperventilation with decreases in the blood CO<sub>2</sub>, it lead to respiratory alkalosis develops, disturbed regulation of respiration. During waking mind takes the signals to breath. During sleep, when the mind control is weakened, there is a Cheyne-Stokes periodic breathing (respiration stopped on 10-15 seconds (due to a lack of CO<sub>2</sub>), after which the

respiratory movements renewed, first increasing in depth (due to increased concentration of CO<sub>2</sub> during apnoe), then decreasing.

Hypocapnea shifts HbO<sub>2</sub> dissociation curve to left (increases alveolar O<sub>2</sub> uptake and inhibits release of O<sub>2</sub> to tissues) it leads to hypoxia deepening.

Risk factors of acute mountain sickness are rate of ascent, individual susceptibility, preexisting cardiopulmonary disease, physical exertion, obesity.

Symptoms of AMS occur in 6 - 36 H after ascent. There are headache, dizziness, fatigue, malaise, disturbed sleep, anorexia, nausea, vomiting, shortness of breath. In humans, the most sensitive and susceptible to hypoxia are the brain tissue and the tissue of pulmonary alveoli. It is the cause of development edematous processes in the brain and lung alveoli.

Hypoxic mechanisms of pulmonary and brain edema:

1. The increase of pressure in the blood vessels and capillaries due to their spasm, water retention in the body and blood stagnation in the venous system;
2. increasing the permeability of capillary wall leads to the exit of the liquid plasma components into the intercellular space;
3. increased permeability of the cell membrane (hypoxia violates selective permeability of cell membranes, resulting in begins equalization of ion concentration inside and outside the cell: the cell loses K<sup>+</sup> ions and overloaded by Na<sup>+</sup>, Ca<sup>2+</sup> ions);
4. decrease in plasma oncotic pressure of leads to blood concentration.

Manifestations of brain edema:

- change in mental status; e.g., confusion
- photophobia
- hallucinations
- ataxia (discoordination)
- coma
- can cause death from brain herniation

Manifestations of pulmonary edema are progressive: initial nonproductive cough, progressive dyspnea, tachypnea and tachycardia, production of pink, frothy sputum, due to weakness the patient tries to lie, but due to suffocation forced to sit; severe hypoxemia, patchy infiltrates on chest x-ray. It leads to progress of hypoxia, lethargy, coma and death.

### **Chronic mountain sickness (CMS, Monge's disease)**

Occurs in high altitude natives or long-term residents (>2500 m). May be primary CMS (acclimatized individuals) and secondary (individuals with conditions; e.g., obesity, neuromuscular disorders, chronic lung disease).

Chronic hypoxia leads to excessive erythropoiesis (erythrocytosis with Hct >58%), decreased partial pressure of O<sub>2</sub> in the air is associated with pulmonary hypertension, leading to cor pulmonale, reduced exercise tolerance, dyspnea, headache, anorexia, inability to concentrate, memory loss.

### **Explosive decompression**

Explosive decompression occurs usually at fast depressurization of the aircraft at high altitude (more than 16 km above sea level).

Pathogenesis:

1. per second there is decrease in ratio of end atmospheric pressure to the initial in more than 2 times;
2. partial pressure of oxygen in the inhaled air reduced to below 16 mm Hg, and in the alveoli – less than 6.5 mm Hg .;
3. barometric pressure is equalized with the sum of pressures of water vapor (47 mm Hg or more) and carbon dioxide (30 mmHg or more).

All this leads to the fact that the oxygen content in the tissue close to zero, and breathing switches from oxygen to nitrogen. Is attached multiple gas embolism of tissues and organs (rapid formation of gas bubbles, mainly nitrogen, due to a sharp decrease in its solubility in tissue and interstitial fluid). There is effect of "boiling" blood, intercellular and even intracellular fluids, leading to rupture of blood vessels, lungs and other organs.

On the background of excess afferentation with huge receptor field and mechanical limit excursions of the lungs, heart and blood vessels rapidly difficult and depressing respiration, heart work, blood return to the heart, reduced blood pressure and increased venous and cerebrospinal fluid pressure. Already within 1-2 minutes after the onset of explosive decompression the heart stops, develops collaptoid state, lost consciousness, convulsions and death occurs.

### **Effect of increased barometric pressure (hyperbaria)**

There are two main types of hyperbaric: natural and artificial.

Artificial hyperbaric carried out with different purposes occurs when the human or experimental animal stay in the pressure chamber (eg, hyperbaric oxygenation).

Natural hyperbaric –is a compression of the body when submerged under water (while diving to great depths, divers and caisson work). When submerged under water, for every 10 meters per person acts additional 1 atmosphere.

High hydrostatic pressure suppresses biochemical reactions occur with an increase in volume of the final products. However, the main pathogenic effect is associated with increased dissolved gases in body fluids (saturation). When submerged under water increases the amount of dissolved nitrogen. Especially active are saturated with nitrogen organs rich in fat (adipose tissue and nerve dissolved in 5 times more nitrogen than blood). At the same narcotic effect of nitrogen manifest at a barometric pressure more than 0.6 MPa. Oxygen at elevated pressure more 0.2-0.4 MPa becomes toxic.

#### **Decompression Sickness (Caisson disease)**

Decompression associated with the sudden decrease in pressures during underwater ascent, usually occurring during free or assisted dives.

There are three periods (stages):

1 period – dive (during the transition from normal to increased pressure). When submerged under water at a depth of 20-40 m compressed surface vessels, chest, lungs, increased blood circulation in the internal organs (including the lungs, heart, brain), accompanied by overstretching the walls of their blood vessels (up to rupture), impression of eardrums (up to rupture). There are possible displacement and compression of the internal organs, as well as lung tissue ruptures, the occurrence of air embolism and even death.

2 period – saturation (the period of constant high saturation of liquids and gases in tissues a result of increased their solubility). Risk of development of lung barotrauma and air embolism enhanced. Dissolved in plasma, tissues nitrogen (especially in nerve and fat) causes initially euphoria, then - anesthesia and finally - the toxic effect. Toxic effect of nitrogen and oxygen are manifested the development of headache, dizziness, disorders of the cardiovascular system (in the form of bradycardia, reduce of volume flow velocity), damage of the airway epithelium, their alveolar surfactant layer (up to pulmonary edema), intestinal mucosa, inhibition of erythropoiesis, the development and progression of metabolic acidosis, seizures, necrobiosis, necrosis and even death .

3 period – desaturation (during ascent or decompression, characterized by the formation and an increase in gas, especially nitrogen, bubbles in the extracellular and intracellular fluids). Develops when the body passing from high blood pressure to normal atmospheric pressure.

During infringement of ascent rules develops caisson disease.

Faster the diver ascends with depth more rapidly, in large quantities, and larger formed gas bubbles (especially nitrogen and helium), because it moves from the dissolved gaseous state. The gas accumulates in the form of bubbles in the blood, extracellular fluids, fat and nerve tissues.

#### **Manifestations**

- bends pain in large joints
- chokes cough, substernal pain
- marbling of skin; skinny bends – cutaneous, itchy rash
- lymphedema
- spinal cord: ascending paresthesia (tingling), ascending paralysis, loss of bowel and bladder control
- bleeding froth from mouth, nose

- headache, confusion, sensory and motor deficits
- in worst case convulsions, coma, death

## DAMAGING EFFECT OF SOUND AND NOISE

### Noise

Harmful to health boundary volume is 80 dB.

Distinguish specific and non-specific effect of noise on the human body.

**Specific effect** of noise – **dysfunction of the auditory analyzer**, due to prolonged spasm of sound-perceiving apparatus resulting in the disruption of metabolic processes and as a consequence is degenerative changes in the endings of vestibular-cochlear nerve cells and Corti organ.

**Non-specific effects of noise:**

- entering of **excitation in the cerebral cortex** of the brain. In the initial stages develops protective inhibition of the central nervous system with impaired mobility and balance of excitation and inhibition. In the future, there is a depletion of nerve cells and, consequently, irritability, emotional instability, memory loss, reduced attention and working capacity;
- **excitation of the hypothalamus**, which is realized by the type of **stress reaction**;
- entering of **excitation in the spinal cord** is switched on its **autonomic nervous system** centers, which causes a change in the functions of many organs.

### Ultrasound

Ultrasound – inaudible to the human ear elastic waves with a frequency above 20 kHz. Sound pressure in the ultrasonic wave can vary  $\pm 303,9$  kPa (3 bar).

The biological effect of ultrasound is due to:

- **mechanical effect**: negative pressure promotes the formation of microscopic cavities in the cells, followed by rapid slamming them, which is accompanied by intense hydraulic beats and ruptures – **cavitation**;
- **physico-chemical effect**: cavitation leads to depolarization and degradation of molecules, causing them to ionization that activates chemical reactions, normalizes and accelerates tissue metabolism;
- **heat effect** of ultrasound mainly due to the absorption of acoustic energy. When the ultrasound intensity  $4 \text{ W/cm}^2$ , and its exposed for 20 seconds the temperature of tissue at a depth of 2-5 cm is increased at  $5-60^\circ \text{C}$ .

A positive biological effect in tissues causes ultrasound of low (up to  $1.5 \text{ W/cm}^2$ ) and secondary ( $1.5-3 \text{ W/cm}^2$ ) intensity. High-intensity ultrasound ( $3-10 \text{ W/cm}^2$ ) has a damaging effect: violates the capillary blood flow, causing destructive changes in the cells, leading to local overheating of tissues.

The nervous system is most sensitive to ultrasound: selectively are affected peripheral nerves, broken transmission of nerve impulses in the synapses. As a result, there is vegetative polyneuritis and paresis, raises the threshold of excitability of the auditory, vestibular-cochlear and visual analyzers, sleep disorder, irritability, fatigue.

## DAMAGING EFFECT OF THERMAL ENERGY

### Action of high temperatures

Action of high temperatures can cause burns, burns disease and overheating.

### Burns injury

Burns (thermal) - local tissue damage as a result of fire, flammable liquids, steam, heated solids.

Mechanism of burns associated with the development of inflammatory reactions at the site of agent action and the thermal coagulation of proteins, leading to cell death and tissue necrosis.

The depth of **burn injury** is classified as follows:

**1<sup>st</sup> degree** – **Superficial** burns involving **only the epidermis**. The skin will be red and hypersensitive.

**2<sup>nd</sup> degree** – burns involving **the epidermis and part of the dermis**, location for collagen, elastic fibers and sweat glands and hair follicles. The skin is red, blistered and swollen, painful (due to damage of sensory nerves). These burns may heal without grafting.

**3<sup>rd</sup> degree**: 3a – partial or complete necrosis of malpighian (sprout) skin layer; 3b – complete necrosis of the **skin in the full thickness**. The sensory nerves are destroyed; therefore, all sensation to pinprick is lost in the affected areas.

**4<sup>th</sup> degree**– full thickness burns that destroy both layers of the **skin and underlying structures, including fascia, muscle and bone**.

The location of burn injury is a factor in determining the severity and outcome. Burn injury to the face, neck, hands, feet, perineum, respiratory system and crossing major joints are associated with complications, functional loss and handicap.

### **Burns disease**

Burns disease – functional disorders of internal organs and systems developed due to extensive **(more than 10-15% of the body surface) and deep burns**.

There are **four periods** of development burn disease:

1. **Burn shock** – in extensive and deep burns over 15% of the body surface (in children and elderly even smaller areas). In the first 12-36 hours, in a zone of burning **sharply increases capillary permeability**, which leads to significant **release of fluid** from the vessel into the tissue. At the site of injury evaporates large amounts of edematous fluid, **blood volume is decreased**. Leading pathogenetic factors: hypovolemia, pain stimulation and increase vascular permeability.

2. **General toxemia** –develops due to **autointoxication by decay products** of burns site tissue (denatured protein, biologically active amines, polypeptides, et al.) and the production of specific burns autoantibodies (in skin identified burns autoantigens specific for this type of injury).

3. **Septicotoxemia (secondary infections)**.

4. **Reconvalescence (recovery)**.

### **Overheating**

**Overheating (hyperthermia)** – a temporary increase in body temperature due to the **accumulation of excess heat** (with a loss of heat transfer processes and the effect of high environmental temperature).

**The causes** of overheating:

- **environmental factors**: high ambient temperature (ambient temperature about 33°C stops heat loss from the body surface due to convection and radiation of heat; at higher temperature, the heat loss is possible only due to the evaporation of sweat from the skin surface); high humidity (development of overheating even at 33-34 ° C due to cessation of sweating or evaporation of sweat); water deficit in the body and due to its loss with sweat;

- the presence of agents impeding implementation of the mechanisms of heat transfer of the body;

- **uncoupling of oxidation and phosphorylation** in the mitochondria.

Increase in body temperature accompanied by:

- sharp acceleration of respiratory movements (irritation of the respiratory center of the heated blood) develops heat dyspnea

- acceleration of heart rate and blood pressure;

- due to water loss through sweating occurs blood clots, disturbed electrolyte metabolism, increased erythrocyte hemolysis;

- damage to various tissues leads to the accumulation of decay toxic their;

- due to destruction of VII, VIII, X, and other plasma factors disrupted blood clotting.

**Overexertion of thermoregulation mechanisms leads to their depletion**, followed by **inhibition of central nervous system, respiratory depression, heart function and decreases in blood pressure and as a result – a deep hypoxia**.

## Heat stroke

**Heat stroke** – is acute hyperthermia with a rapid rise in body temperature or prolonged exposure to high environmental temperature.

Manifestations: core temperature exceeding 40°C, cessation of sweating, rapid pulse, rapid respiration, hypotension, CNS symptoms predominate: unsteady gait, confusion, combative behaviour, reduced consciousness, convulsion and coma.

Death in heat stroke occurs from paralysis of the respiratory center.

## Action of low temperatures

Effect of low temperatures can cause supercooling (hypothermia), frostbite and chronic lesions of the cold – cold neurovasculitis.

## Supercooling (hypothermia)

Primary hypothermia: Excessive exposure to cold, wind, snow, water, or altitude.

Secondary hypothermia: patient becomes cold in normal environmental temperature, secondary to underlying condition such as trauma, myxedema, sepsis. Elderly, alcoholic, and chronically ill have impaired heat generation owing to reduced lean body mass, impaired mobility, inadequate nutrition, reduced shivering; also, impaired vasoregulatory responses and ability to sense temp extremes. Medications that may alter central thermoregulation (dopaminergic tone) are phenothiazines, barbiturates, lithium,  $\alpha$ -blockers).

In the pathogenesis of hypothermia distinguish the following phase:

1. **Compensation.** Reaction intended to limit heat transfer: a reflex spasm of blood vessels, decreased sweating, slow breathing. Increase heat production: muscle tremors (chills), strengthening the processes of glycogenolysis in the liver and muscles, increased blood glucose, increased basal metabolic rate.
2. **Decompensation.** Develops under the long-term low temperatures. Reduced body temperature, muscle tremors stopped, reduced oxygen consumption and intensity of metabolic processes, dilate peripheral blood vessels. As a result, inhibition of functions of the cerebral cortex, subcortical and bulbar centers, reduces blood pressure, slows the heart rate, progressively weakens and reduces the incidence of respiratory movements. Gradual fading all vital functions. Death occurs from paralysis of the respiratory center.

Stages of hypothermia:

- Mild Hypothermia – individual response to cold varies. In general, body temperatures from 33° to 35°C constitute mild hypothermia. In this temperature range, the casualty is in an excitation (responsive) stage. The casualty will usually remain conscious, however, they may start to exercise poor judgment or have irrational behavior. The body's natural defense mechanism, shivering, will eventually diminish. The body will attempt to retain and generate heat by increasing heart rate, blood pressure, and cardiac output. The respiratory rate will increase, which, in the long run, only cools the body more by breathing in cold air and losing moisture through respirations.

- Moderate Hypothermia – moderate hypothermia occurs when the core temperature is between 30°-33°C. Cognitive abilities become more difficult and the patient becomes stuporous and does not respond to painful stimuli. Shivering is replaced by progressive muscular rigidity. In the initial excitation phase, heart rate, blood pressure, and cardiac output all rise. With decreasing temperatures, these all decline. The patient in this stage is at risk for lethal cardiac dysrhythmias.

- Severe Hypothermia – when the core temperature is below 30°C, the patient is in severe hypothermia. The casualty will be unconscious with no response to pain. Pupils dilated. Vital signs will be barely detectable or non-detectable. Without immediate and intensive treatment, this patient will die.

**Hibernation** – is artificial decrease in body temperature in medical practice under anesthesia achieved by physical effects used to reduce the body's need for oxygen and prevention of the temporary cerebral ischemia.

## Frostbite

The most susceptible body parts are those areas farthest from the body's core, such as the hands, fingers, feet, toes.

Depending upon wind velocity and air temperature, the exposure time necessary to make frostbite varies from a few minutes to several hours. The degree of cold injury, just like burn injuries, in many cases will not be known for at least 24 to 72 hours. There are four degrees of frostbite injury (identical to burn injuries) based on physical findings.

**1<sup>st</sup> degree – superficial injury.** The first signs - burning, tingling, followed by numbness of the affected area. Then there are itching and pain of varying severity, edema develops. After a few days may be a slight peeling. Full recovery occurs in 5 - 7 day after frostbite.

**2<sup>nd</sup> degree** – involves all the **epidermis and superficial dermis**. After warming intense and prolonged pain, itching, burning sensation. In the initial period there pallor, cooling, loss of sensitivity, formation of bubbles filled with clear content. Full restoration of the skin integrity occurs within 1-2 weeks, granulation and scars are not formed.

**3<sup>rd</sup> degree** – involves the **epidermis and dermis layers** and frozen skin is stiff with restricted movement. Long-term intense pain. Formed at the initial stage of bubbles filled with bloody contents, their bottom blue-purple, is not sensitive to stimuli. There is a destruction of all skin elements with the development of granulation tissue and scarring. Nails do not grow again or grow deformed. Rejection of dead tissue ends in 2-3 weeks, after which comes the scarring that lasts up to 1 month.

**4<sup>th</sup> degree** – frozen tissue involves **full thickness** through **dermis** with **muscle** and **bone** involvement. No bubbles develop at much edema, loss of sensitivity indicate the frostbite 4<sup>th</sup> degree.

## EFFECTS OF ULTRAVIOLET RADIATION

Ultraviolet (UV) irradiation comprises three distinct spectra: A (320-400 nm), B (290-320 nm), and C (200-290 nm).

UVA penetrates to the deeper dermis, formation of pigment by converting of tyrosine to melanin.

UVB penetrate the epidermis and are nearly fully absorbed in the upper dermis. UVB wavelengths have strong general stimulating effect, vitamin-anti-rachitic action (synthesis of cholecalciferol). Mechanism common stimulating and photochemical actions: UV radiation excites atoms, enhancing their reactivity, which leads to increased activity of chemical reactions in the cells, providing a stimulating effect on the metabolic and trophic processes. Ultimately, the enhanced growth and tissue regeneration, increases the body's resistance to the action of infectious and toxic agents, improves physical and mental performance.

UVC is absorbed by atmospheric ozone. Pronounced bactericidal action.

Pathogenic effect of excessive UV exposure:

- **skin lesions** it causes **photochemical burn**, with the development of erythema and Blistering skin reactions, fever, headache, common painful condition. Pathogenic effect is associated with activation of lipid peroxidation, resulting in damage to the membrane, disintegration of protein molecules, cell death;

- defeat of **conjunctiva** (photoelectric ophthalmia) manifested its redness and swelling, burning sensation and "sand" in the eyes, lacrimation, photophobia pronounced;

- can provoke an **aggravation of some chronic diseases** (rheumatism, stomach ulcer, tuberculosis, etc.);

- due to increased melanin formation and degradation of proteins increases the body's need for essential amino acids, vitamins, calcium and other salts;

- excessive UV radiation in the wavelength range area C can lead to inactivation of cholecalciferol - to its conversion into indifferent or even harmful substances;

- **prolonged excessive** UV exposure can promote the **formation of peroxy compounds** and **epoxy compounds having a mutagenic effect**, and induce occurrence of basal cell and squamous **cell carcinomas** of the skin, especially in people with fair skin;

- effects on the nervous system mediated through the irradiated skin capillaries in blood proteins and cholesterol. Excitation occurs in the autonomic centers of the hypothalamus and basal ganglia, increased body temperature, increased and then drop in blood pressure, drowsiness, collapse and death

from paralysis of the respiratory center.

### THE DAMAGING ACTION OF LASER RADIATION

Lasers – a device for obtaining narrow beam of monochromatic light energy of high intensity. It is successfully used for the treatment of numbers diseases (eye diseases, tumor, etc.). The action of laser radiation is measured in hundreds of thousand parts of second, so, despite the fairly deep penetration of laser beams into the body (20-25 mm), the sensation of pain does not arise. The greatest sensitivity to laser radiation has pigmented tissue.

**Effect** of laser radiation on the cell:

- **free radical mechanism**: the direct damaging effect of laser radiation on the cell associated with the excitation of atoms and ultimately to damage of protein molecules;
- **thermal effects** associated with absorption by tissue energy of infrared radiation and heat inactivation of protein;
- **cavitation effect** is due to a rapid rise in temperature to level at which evaporation of the liquid portion in cell. There is a "blast effect" (cavitation) due to the momentary formation of a microcavity with elevated pressure (up to tens or hundreds of atmospheres), and propagating from her shock wave, disruptive a tissue. This effect is at the heart of the laser scalpel;
- **biological** – inactivation of enzymes and changes in their specific activity.

### EFFECT OF ELECTRIC CURRENT ON THE BODY

Features of electric damaging effect:

- tissue damage throughout the path;
- irritating vast number of receptors;
- cause biological effects, chemical, mechanical, thermal damage.

Pathway of the current through the body:

- vertical pathway parallel to the axis of the body is the most dangerous. It involves all the vital organs; central nervous system, heart, respiratory muscles, in pregnant women the uterus and fetus;
- horizontal pathway from hand to hand: the heart, respiratory muscles and spinal cord;
- pathway through the lower part of body – local damage.

Table 2. Pathophysiology Effects of Different Intensities of Electrical Current

Current intensity	Probable effect
1 mA	Tingling sensation; almost not perceptible
16mA	Maximum current a person can grasp and "let go"
6-9 mA	"Let-go" current for an average adult
3-5 mA	"Let-go" current for an average child
16-20 mA	Tetany of skeletal muscles
20-50 mA	Paralysis of respiratory muscles; respiratory arrest
50-100 mA	Threshold for ventricular fibrillation
>2 A	Asystole
15-30 A	Common household circuit breakers
240 A	Maximal intensity of household current

#### ***Mechanism of action:***

Specific action – manifested by passing current through the body, resulting effects are caused by the redistribution of ions.

Nonspecific action – due to other forms of energy, which is converted into electricity outside the body (flame and electric arc radiation, light, ultraviolet, infrared radiation).

#### ***Specific action:***

• **Biological effect** is stimulation of skeletal and smooth muscle, glandular tissue, nerve receptors and conductors.

• **Electrochemical action** includes:

✓ electrolysis ;

✓ polarization of cell membranes ;

✓ accumulation in some parts of positively charged ions, occurrence of acid reaction and coagulation of proteins (coagulation necrosis), on other negatively charged ions are accumulated, there is alkaline reaction, swelling of colloids, there colliquative necrosis;

✓ movement of protein molecules;

✓ the accumulation of toxic products of electrolysis;

✓ dissolved gases pass in the gaseous state;

✓ plating of skin during contact of body with metals.

• **The thermal effect** due to the conversion of electrical energy into thermal energy, with a large amount of heat release in the tissues. Manifestations:

✓ «pearl beads" occur during melting of the bone with the release of calcium phosphate;

✓ «current signs" - areas of coagulated epidermis having a circular or oval shape, gray-white, solid consistency

✓ fringed roll elevation with retraction in the center;

✓ branching pattern of red color is due to paralysis of the blood vessels. Such changes are observed on the skin, if the temperature at the point of passage of the current does not exceed 120 ° C;

✓ burns are formed at higher temperature, the current passes through the tissue, it may damage the underlying tissue until carbonization.

• **Mechanical effect** due to significant thermal and mechanical energy high voltage currents. The combined action of heat and mechanical energy has an explosive effect.

### **Complications**

• postraumatic myositis with rhabdomyolysis

• acute/delayed-onset central and peripheral nervous system complications (very often)

• sepsis

• psychiatric complications

### **Immediate death may occur from:**

• Current-induced ventricular fibrillation

• Asystole

• Respiratory arrest secondary to:

– Paralysis of the central respiratory control system

– Paralysis of the respiratory muscles

## **THE DAMAGING EFFECTS OF IONIZING RADIATION**

### **Mechanisms of action of ionizing radiation**

Etiological factors of radiant disease are the various forms of ionizing radiation of high energy ( $\alpha$ -,  $\beta$ -,  $\gamma$ - rays, X-radiation, etc.). Their general property is an ability to penetrate in biological medium and ionize atoms and molecules.

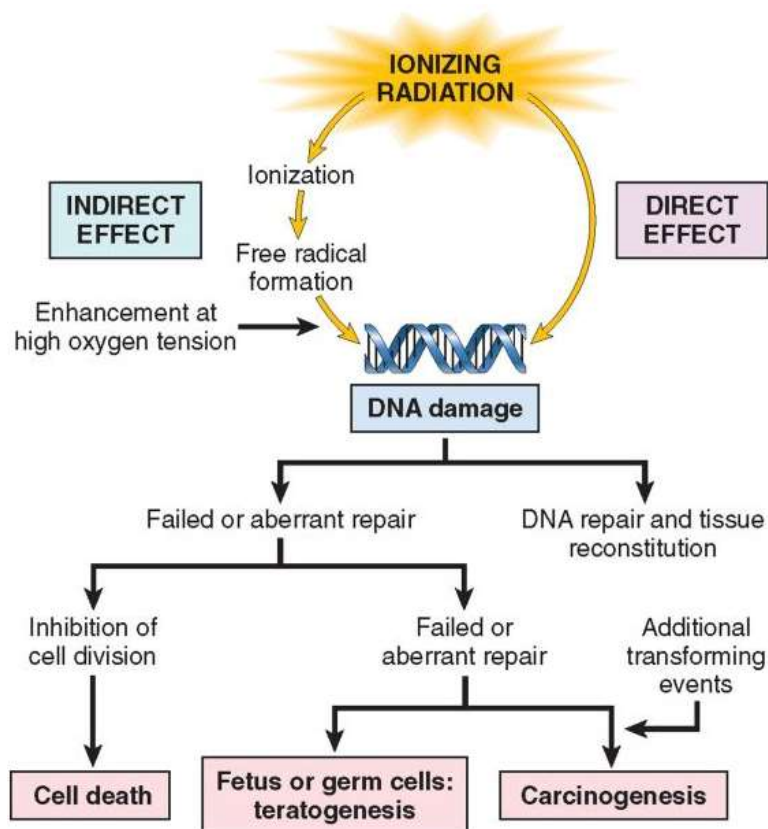
Intensity and duration of injury depends on form of radiation, dose, time factor and type of molecular or supramolecular target, which occurs on the pathway of radiation.

The external irradiation is such one, when a source of it is located out of organism. The internal (incorporated) irradiation is the one, when radioactive materials enter inside.

### **1. The primary action of IR**

**Direct effect** of radiant energy is a damage of the macromolecules of organism by radiation itself.

Eventually, intramolecular alterations happen. Any type of molecules may be a target - organic macromolecules as DNA, lipids, phospholipids, enzymes, proteins, vitamins, hemoprotein, etc.



**Figure 2** – Effects of ionising radiation (V. Kumar, 2010).

**Indirect effect** is a damage of macromolecules by the water radiolysis products. Ionization of water molecules is the most significant of all primary radiochemical transformations. The first products are the ionized water molecules  $\text{H}_2\text{O}^+$  and  $\text{H}_2\text{O}^-$ . Then free hydrogen and hydroxyl radicals are formed ( $\text{H}^*$ ,  $\text{OH}^*$ ), which initiate a chain of further reactions and new products are formed (peroxide of hydrogen  $\text{H}_2\text{O}_2^*$ , hydroperoxide  $\text{HO}_2^*$ , atomic oxygen  $\text{O}^*$  etc.). The water radiolysis products are very active biochemically and cause extensive nonenzymatic oxidation.

## 2. Action of IR on cells

Cellular reaction to the action of IR:

- temporary blocking of mitosis;
- complete suppression of mitosis;
- interphase cell death;

- mitotic cell death.

By radiosensitive include actively dividing and undifferentiated cells: hematopoietic bone marrow cells, germ cells of the testes, skin and intestinal epithelium. In spite of differentiation, high radiosensitivity has lymphocytes. Radioresistant cells include brain, muscles, liver, kidneys, cartilage, ligaments.

## 3. Action of IR at the organism level:

### Immediate consequences:

- Radiation sickness from external exposure
  - ✓ Acute radiation sickness
  - ✓ Chronic radiation sickness
- Radiation sickness from internal exposure
- Local action of IR (radiation burns, cataracts, necrosis)

**Long-term consequences:** may develop after 10-20 years or more after total body irradiation or local.

Distinguished **somatic consequences** (occur in irradiated organism):

- **non-tumor forms** – reduced life expectancy, hypoplastic state in hematopoietic tissue, mucous membranes of digestive system, respiratory tract, in the skin; sclerotic processes (cirrhosis, nephrosclerosis, arteriosclerosis, cataract beam), and dishormonal states (obesity, hypophysial cachexia, diabetes insipidus);
- the development of **tumors**, radiation leukemia.

**Genetic consequences** (as a result of damage to reproductive cells) may occur loss of zygote or embryo, the birth of individuals with inherited abnormalities or carrying mutant genes. "The genetic load" can be transmitted from generation to generation.

## Acute radiation sickness (ARS)

In total, single, uniform irradiation of the organism in a dose of 1 Gray.

There are 4 form of ARS:

1. Bone marrow form (1-10 Gy)
2. Intestinal form (10 to 20 Gy, the death at 7-10 days)
3. Toxemic form (20 -80 Gy, the death at 4-7 days)

4. Cerebral form (more than 80 Gy, the death at after 1-3 days, and during the actual exposure — "death under the beam")

**Bone marrow form**

Depending on the dose there are four degrees of severity:

- I - mild (1-2 Gy);
- II - medium (2-4 Gy);
- III - severe (4-6 Gy);
- IV - extremely severe (more than 6 Gy).

The disease is characterized by 4 phases:

1) **Primary acute phase reaction** occurs in the first minutes or hours after exposure. Duration of phase 1-3 days.

Manifestations:

- excitation, headache, general weakness.
- dyspeptic disorders (nausea, vomiting, loss of appetite).
- lability of the autonomic functions - fluctuations in blood pressure, heart rhythm.
- activation of the pituitary-adrenal system, enhanced secretion of adrenal hormones
- at doses of 8-10 Gy seen the development of shock-like state with a decrease in blood pressure, transient loss of consciousness, fever, diarrhea development
- Peripheral blood: leukocytosis with a left shift, absolute lymphopenia

2) **Phase of the imaginary clinical well-being** — including in the pathological process of defense mechanisms. Duration depends on the radiation dose and ranges from 10-15 days to 4-5 weeks. For very severe defeat this phase is absent.

Manifestations:

- patients feel satisfactory becomes visible clinically signs disappear
- in the gonads can atrophy, inhibition in early stages of spermatogenesis,
- in the small intestine and skin atrophic changes
- neurological symptoms quenched
- Peripheral blood: progress lymphopenia on the background of leucopenia, decreased reticulocyte count and platelets.
- In the bone marrow develops devastation (aplasia).

3) **Phase height of disease**: a sharp deterioration of health. Duration phase from several days to 2-3 weeks. When irradiated over 2.5 Gy possible death.

Manifestations:

- weakness, increased body temperature;
- appear bleeding and hemorrhage in the skin, mucous membranes, gastrointestinal tract, brain, heart and lungs
- reduced body weight
- hypoproteinemia, hypoalbuminemia, elevated levels of residual nitrogen and reduction of chlorides
- infection a result of decrease immunity
- Peripheral blood: leukopenia, thrombocytopenia, anemia, ESR increase
- Bone marrow: picture of devastation with initial signs of recovery.

4) **Recovery phase**: gradual normalization of disturbed functions. Duration 3-6 months, in severe cases, 1-3 years, can become chronic.

Manifestations:

- general condition significantly improved
- normal temperature
- disappear diarrhea and hemorrhagic manifestations

- after 2-5 months normalizes the function of sweat and sebaceous glands, hair growth renewed
- Peripheral blood: recovered blood counts and metabolism.

### **Intestinal form**

10–20 Gy, death at 7-10 days.

Manifestations: nausea, vomiting, bloody diarrhea, increased body t, there may be full pseudoileus and bloating. Develop hemorrhages and deep leukopenia with a complete lack of lymphocytes, the picture of sepsis. Death is a result of dehydration, accompanied by loss of electrolytes and protein, shock.

### **Toxemic form**

20 -80 Gy, death at 4-7 days.

Manifestations: hemodynamic instability in the intestine and liver, vascular paresis, tachycardia, hemorrhage, severe intoxication and meningeal symptoms, oliguria and hyperasotemia.

### **Cerebral form**

more than 80 Gy, death after 1-3 days, and during the actual exposure - "death under the ray."

Manifestations: convulsively paralytic syndrome, blood circulation, lymph circulation in the central nervous system, vascular tone and thermoregulation, digestive and urinary systems, a progressive decrease in blood pressure. Cause of death - cell death of the cerebral cortex, neurons of hypothalamic nuclei.

## **Chronic radiation sickness (CRS)**

CRS caused by prolonged exposure of the organism in small doses after a total dose of 0.7 - 1 Gy.

Forms of CRS:

1. Total external irradiation or diffuse distribution of isotopes causes a form with detailed clinical syndrome;
2. Clinical syndrome with lesion of certain organs and systems from internal and external irradiation.

### **Period of development:**

1. **Initial period** - unstable leukopenia, asthenia signs, vegetative-vascular instability.
2. **Extended period** - lack of physiological regeneration and functional changes in the nervous and cardiovascular systems.
3. **Recovery** period, the prevalence of reparative processes.

Features: the gradual development of a long wavy current.

### **Severity:**

- light (grade I): poorly expressed neuro-regulatory disorders of organs and systems, unstable moderate leukopenia and thrombocytopenia;
- moderate (grade II): add functional impairment of NS, cardio-vascular system and gastrointestinal tract, and progress leukopenia lymphopenia, thrombocytopenia, hypoplasia of bone marrow.
- severe (grade III): atrophic processes in the gastrointestinal mucosa, add infectious-septic complications, anemia, severe hypoplasia of hematopoiesis, hemorrhagic syndrome and circulatory disorders.

## **Radiation sickness from internal exposure**

**Radiation sickness from internal exposure** – an independent nosological form, a chronic disease caused by the gradual accumulation of radioactive elements are  $\alpha$ ,  $\beta$ ,  $\gamma$  emitters.

There are three main types of radionuclides distribution: skeletal, reticuloendothelial and diffuse.

By the skeletal type distributed mainly radionuclides of alkaline earth group elements (calcium, strontium, barium, radium) accumulated in the mineral part of the skeleton.

Reticuloendothelial type of distribution is typical for nuclides of rare earth elements – zinc, thorium, americium, transuranic elements.

The diffuse type distributed alkali elements – potassium, sodium, cesium, rubidium, hydrogen nuclides.

"Organotropic" radionuclides selectively accumulate in certain organs (such as isotopes of iodine concentrates in the thyroid gland, in the kidney – uranium, radioactive lead and beryllium).

Manifestations: syndromes of general and local lesions in areas dominating penetration of radioactive substances in the body, their removal and accumulation.

## PATHOGENIC EFFECTS ON THE BODY OF CHEMICAL FACTORS

Damaging effect on the body of chemical substances and compounds (poisons) found both in industry (industrial poisoning) and at home (household poisoning).

Arising under the influence of diverse chemical the biological effects can be classified as follows:

- local irritant (damaging) effects;
- total specific (toxic) effects (eg, the effect of botulinum toxin on nerve terminals in the muscles);
- general non-specific action (eg, the occurrence of hypoxia of many tissues, organs and systems with damaged lungs, heart, etc.);
- mutagenic effects;
- carcinogenic effect;
- teratogenic effects.

The main way of chemicals penetration are: through the skin barrier, through the respiratory tract, per os, parenteral.

Features of the toxic effect of chemicals determined by:

- species (structure);
- ability to form complexes;
- dose;
- duration of action;
- features of metabolism;
- place of absorption, accumulation and excretion;
- ability to provide local and / or systemic damaging effect, specific and / or non-specific;
- ability to provide local damage to cellular and tissue structures, or cause systemic changes.

## BIOLOGICAL FACTORS

### INFECTIOUS PROCESS

**Infectious process** – developed in the course of evolution typical pathological process that occurs in the interaction of microorganism with the macroorganism, under adverse conditions, external and / or internal environments.

The main types of infectious process:

- **bacteremia, viremia** – the presence in the blood of bacteria and / or viruses with no evidence of their reproduction;
- **sepsis** – severe generalized form of infectious process caused by the multiplication of microorganisms in the blood (sometimes in other body fluids);
- **septicopyemia** – infectious process characterized by the development of secondary purulent lesions in various organs and tissues in patients with sepsis;
- **mixed infection** – infectious process caused simultaneously by two or more agents;
- **reinfection** – the recurrence of infectious process caused by the same organism after the patient's recovery;
- **superinfection** – re-infection by the same pathogen until the period of recovery;
- **secondary infection** - infection process that develops against the backdrop of existing (primary) infectious disease, caused by other microorganisms.

Character and severity of infectious process depends on the following factors:

- basic properties of the infectious agent;
- pathogenicity and virulence;
- features of the interaction of microorganisms and macroorganism;

- tropism of microorganisms to certain tissues of macroorganism;
- initial state macroorganism (especially the immune system);
- condition of the environment.

## PSYCHOGENIC PATHOGENIC FACTORS IATROGENIC DISEASES

There are the following types of psychosomatic and somatopsychic disorders:

- **Psychosomatic disorders** – somatic disorders arising from impact of the complex of psychosocial factors (hypertension, coronary artery disease, gastric and duodenal ulcer, neurodermatitis);
- **Nosogenic disorders** - pathological reactions to stressful factors of the disease and its consequences
- **Iatrogenic disease**
- **Somatogenic disorders** – mental disorders that develop as a result of neurotoxic effects somatic disease
- **Mental disorders complicating somatic pathology** (eg, alcoholism, eating disorders)
- **Somatoform disorders** – mental disorders manifested by somatic complaints that are not objectively confirm the presence of somatic disease
- **Dissociative (conversion) disorders** of movement and sensation - mental disorders manifest violation of motor and sensory functions that mimic organic pathology and cannot be explained by structural damage to the nervous system

Common signs of psychosomatic disorders:

- chronic course
- significant role of mental stress in manifestation, development and course of the disease
- personal characteristics of the patient, determining emotional lability, difficulties in interpersonal relationships, and others
- lack effectiveness of traditional methods of somatic pathology treatment
- positive effect of pharmacotherapy and psychotherapy.

### **Iatrogenic diseases**

**Iatrogenesis** – (from the Greek iatros – doctor; genesis - origin) – is any unwanted or adverse effects of preventive, diagnostic and therapeutic interventions or procedures that lead to disturbances of body functions, controlling habitual activity, disability or death; complications of medical interventions, which developed as a result of the error and correct doctor's actions (according to ICD-10).

#### **Classification by iatrogenic A.P. Krasilnikov:**

1. **Psychogenic** «diseases of the word» – careless remarks and misunderstood of medical worker about the health, insight the patient's own medical history and special medical literature, listening to public lectures.
2. **Drugs**: pharmacological negative effects (for example, a hypoglycaemic shock after insulin injection); drug intoxication, including toxic, mutagenic, oncogenic, teratogenic, embryotoxic, immunosuppressive effect; drug allergies, drug intolerance of pseudoallergic nature; drug dependency; drug psychoses; incompatibility of simultaneously administered drugs; vaccination reactions and complications.
3. **Traumatic**: surgical manipulation and occasional medical injuries, burns (radiation, thermal, chemical), and consequences of injuries.
4. **Infectious**: intrahospital (hospital, nosocomial) infections, abscess formation with intramuscular injections, etc ..
5. **Mixed**

#### **Questions for self-control of knowledge:**

1. What is environment pathogenic factors?
2. What is mechanism of environment pathogenic factors action on the body?
3. What is pathogenic effect of physical factors?
4. What is feature of action of high and low temperatures on a organism?
5. What is mechanism of damaging effect of electric current?

6. What is mechanism of biological factors action on human body?
7. What are effects of environmental factors on body?
8. What are main ways of intake radionuclides into the body and types of their distribution in it?
9. What are direct and indirect effects of ionizing radiation?
10. What are features of lesions caused by incorporated radionuclides?
11. What are basic metabolic disorders in body that caused by ionizing radiation?
12. What is effect of psychogenic factors on body?
13. Describe clinical forms of acute radiation sickness.
14. What are principles of diseases prevention caused by action of external environmental factors?

**Tasks for self-managed student work:**

1. Caisson disease.
2. Mountain sickness.
3. Pathogenic effect of acceleration.
4. Acute radiation sickness.
5. Chronic radiation sickness.

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**Compiler:**

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