HYPOXIA

HYPOXIA IS A TYPICAL PATHOLOGICAL PROCESS THAT OCCURS AS A RESULT OF INSUFFICIENT SUPPLY OF OXYGEN TO TISSUES AND/OR WHEN ITS UTILIZATION IS DISRUPTED BY TISSUES DURING BIOLOGICAL OXIDATION, WHICH LEADS TO A DISORDER OF ENERGY SUPPLY OF FUNCTIONS AND PLASTIC PROCESSES IN THE BODY.

CLASSIFICATION OF HYPOXIA

EXOGENOUS HYPOXIA

- NORMOBARIC (IN BASEMENTS, MINES ACCUMULATES CARBON DIOXIDE)
- HYPOBARIC (IN THE MOUNTAINS);

ENDOGENOUS HYPOXIA

- LOCAL
  - ❖tissue (inflammation, tumor);
  - ❖hemodynamic (inflammation);
  - ❖mixed
- GENERAL
  - ❖respiratory,
  - ❖cardiovascular,
  - ❖tissue, hemic,
  - ❖mixed

HEMIC HYPOXIA

THE REASON IS A DECREASE IN THE OXYGEN CAPACITY OF THE BLOOD AND, CONSEQUENTLY, ITS OXYGEN - TRANSPORTING FUNCTION DUE TO:

- SEVERE ANEMIA, ACCOMPANIED BY A DECREASE IN HB CONTENT.
- VIOLATIONS OF THE TRANSPORT PROPERTIES OF HB (HEMOGLOBINOPATHY). IT IS CAUSED BY CHANGES IN ITS ABILITY TO OXYGENATE IN THE CAPILLARIES OF THE ALVEOLI AND DEOXYGENATE IN THE CAPILLARIES OF TISSUES. THESE CHANGES MAY BE INHERITED OR ACQUIRED.
  - HEREDITARY HEMOGLOBINOPATHIES ARE CAUSED BY MUTATIONS IN THE GENES ENCODING THE AMINO ACID COMPOSITION OF GLOBINS.
  - ACQUIRED HEMOGLOBINOPATHIES ARE MOST OFTEN THE RESULT OF EXPOSURE TO NORMAL HB WITH CARBON MONOXIDE, BENZENE, OR NITRATES.

TISSUE HYPOXIA

OCCURS AS A RESULT OF THE FOLLOWING REASONS:

- INHIBITION OF BIOLOGICAL OXIDATION BY VARIOUS INHIBITORS (CYANIDES – CYTOCHROME OXIDASE; ALCOHOL, DRUGS – DEHYDRASES),
- VIOLATIONS OF THE SYNTHESIS OF RESPIRATORY ENZYMES (DEFICIENCY OF VITAMINS – THIAMINE, RIBOFLAVIN, PANTOTHENIC ACID, NICOTINAMIDE),
- DAMAGE TO CELL MEMBRANE STRUCTURES (IONIZING RADIATION, OVERHEATING, INTOXICATION),
- WITH A PRONOUNCED SEPARATION OF OXIDATION AND PHOSPHORYLATION (DINITROPHENOL, THYROXINE).

PATHOGENESIS OF HYPOXIA

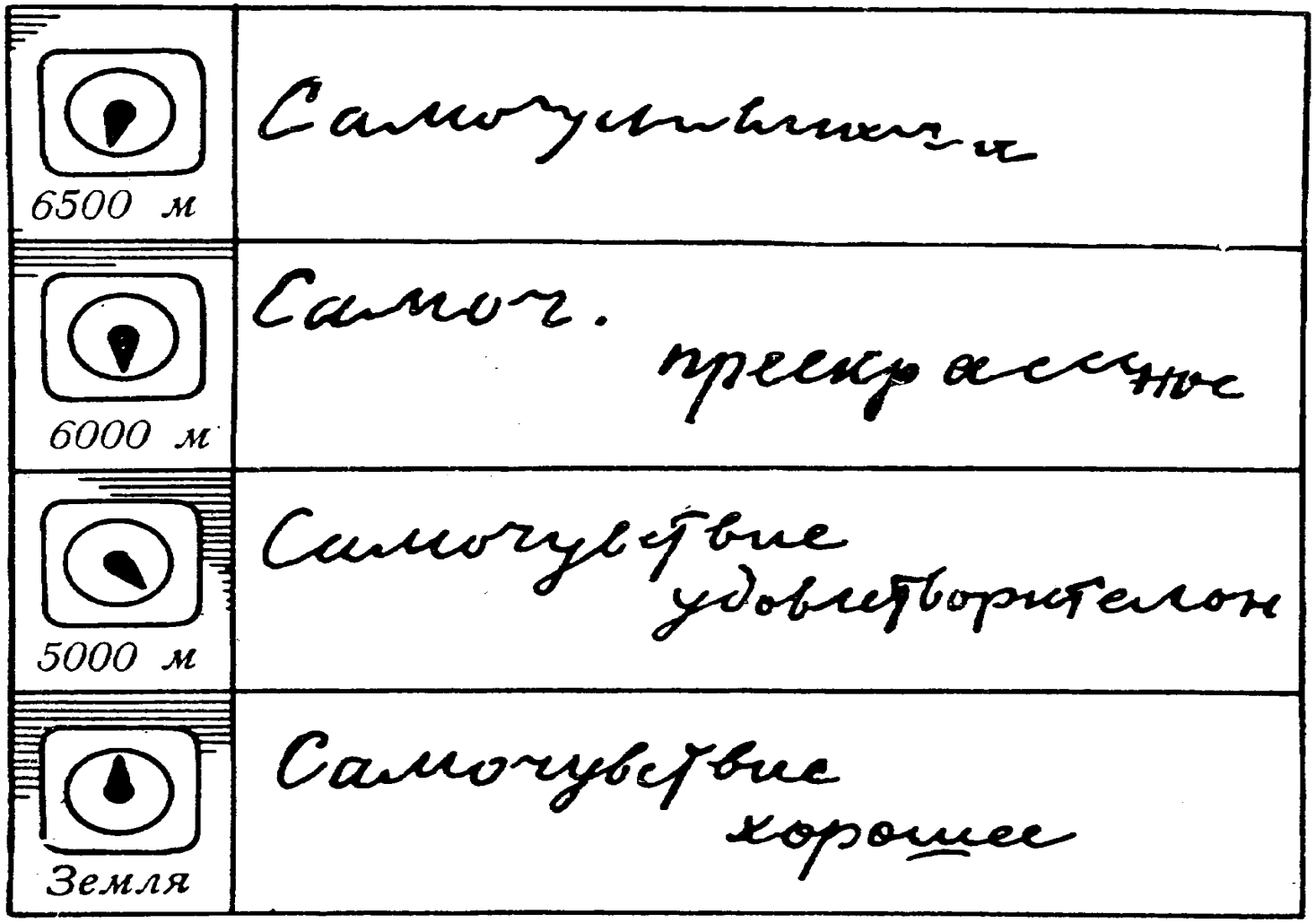
THE MAIN LINKS IN THE PATHOGENESIS OF HYPOXIA INCLUDE: ARTERIAL HYPOXEMIA, HYPOCAPNIA, GAS ALKALOSIS AND HYPOTENSION.

ARTERIAL HYPOXEMIA - THE INITIAL AND MAIN LINK OF EXOGENOUS HYPOXIA. HYPOXEMIA LEADS TO A DECREASE IN OXYGEN SUPPLY TO THE TISSUES, WHICH REDUCES THE INTENSITY OF BIOLOGICAL OXIDATION.

DECREASE IN BLOOD CARBON DIOXIDE (HYPOCAPNIA) OCCURS AS A RESULT OF COMPENSATORY HYPERVENTILATION OF THE LUNGS (DUE TO HYPOXEMIA).

GAS ALKALOSIS IS THE RESULT OF HYPOCAPNIA.

REDUCED SYSTEMIC BLOOD PRESSURE (ARTERIAL HYPOTENSION), COMBINED WITH TISSUE HYPOPERFUSION, IS LARGELY A CONSEQUENCE OF HYPOCAPNIA. A MARKED DECREASE IN CARBON DIOXIDE IS A SIGNAL TO NARROW THE LUMEN OF THE ARTERIOLES OF THE BRAIN AND HEART.



Изменение почерка на высоте (по А.А.Лавникову)

PROTECTIVE-ADAPTIVE REACTIONS BY LOWERING THE PARTIAL PRESSURE OF OXYGEN

1. The increase of lungs ventilation
2. Increase in the respiratory surface of the lungs
3. The increase in minute volume of blood
4. Increase of a tone of vessels and acceleration of blood flow
5. Polycythemia redistribution and activation of erythropoiesis
6. The increased dissociation of oxyhemoglobin
7. The increase of absorption of oxygen by the tissues
8. Activation of anaerobic processes

ASPHYXIA – A PATHOLOGICAL PROCESS WHEN THE BLOOD DOES NOT RECEIVE OXYGEN AND CARBON DIOXIDE IS NOT REMOVED.

CAUSES THAT LEAD TO ACUTE ASPHYXIA: PULMONARY AND EXTRAPULMONARY

PULMONARY CAUSES:

- COMPRESSION OF THE AIRWAYS FROM THE OUTSIDE IS OBSERVED DURING STRANGULATION (HANGING, STRANGLING WITH A NOOSE OR HANDS), COMPRESSION OF THE TRACHEA, NECK INJURIES, ETC.
- OBSTRUCTIVE BREATHING DISORDERS ARE MOST OFTEN CAUSED BY TONGUE ENTRAPMENT, BLOCKAGE OF THE TRACHEA AND BRONCHI BY FOREIGN BODIES, INTRALUMINAL TUMORS, INGESTION OF FOOD, VOMIT, WATER DURING DROWNING, AND BLOOD DURING PULMONARY BLEEDING.
- ACUTE STENOSIS OF THE RESPIRATORY TRACT CAN DEVELOP WITH TRACHEOBRONCHITIS, AN ASTHMATIC ATTACK, ALLERGIC EDEMA OR BURN OF THE LARYNX, EDEMA OF THE VOCAL CORDS. ALSO AMONG THE PULMONARY CAUSES OF ASPHYXIA INCLUDE GAS EXCHANGE DISORDERS CAUSED BY ACUTE PNEUMONIA, MASSIVE EXUDATIVE PLEURISY, TOTAL PNEUMOTHORAX OR HEMOTHORAX, ATELECTASIS OR PULMONARY EDEMA.

EXTRAPULMONARY CAUSES:

- CONDITIONS ARE THOSE THAT LEAD TO DAMAGE TO THE RESPIRATORY CENTER: INTOXICATION, TRAUMATIC BRAIN INJURIES, STROKES, OVERDOSE OF DRUGS AND NARCOTIC DRUGS (FOR EXAMPLE, MORPHINE, BARBITURATES).
- PARALYSIS OF THE RESPIRATORY MUSCLES, AS A CAUSE OF ASPHYXIA, CAN DEVELOP AGAINST THE BACKGROUND OF INFECTIOUS DISEASES (BOTULISM, POLIO, TETANUS), POISONING WITH CURARE-LIKE DRUGS, SPINAL CORD INJURY, MYASTHENIA GRAVIS, ETC.
- VIOLATIONS OF OXYGEN TRANSPORT TO THE TISSUES OCCUR WITH MASSIVE BLEEDING, CIRCULATORY DISORDERS, CARBON MONOXIDE POISONING, AND METHEMOGLOBIN-FORMING AGENTS.TRAUMATIC ASPHYXIA IS BASED ON COMPRESSION OR DAMAGE TO THE CHEST, WHICH MAKES IT DIFFICULT TO BREATHE.
- ASPHYXIA CAUSED BY INSUFFICIENT OXYGEN CONTENT IN THE INHALED AIR CAN DEVELOP DURING PROLONGED STAY IN POORLY VENTILATED MINES AND WELLS, WITH ALTITUDE SICKNESS, WHEN THE OXYGEN SUPPLY TO LIMITED CLOSED SYSTEMS IS DISRUPTED (FOR EXAMPLE, IN DIVERS).
- FETOPLACENTAL INSUFFICIENCY, INTRACRANIAL BIRTH INJURIES, AND AMNIOTIC FLUID ASPIRATION MOST OFTEN LEAD TO ASPHYXIA OF NEWBORNS.

STAGES OF ACUTE ASPHYXIA

1. STRUGGLE WITH HYPOXIA. THE REACTION IS DIRECTED TO THE SUPPLY OF ADDITIONAL OXYGEN. MECHANISM – IRRITATION OF THE RESPIRATORY CENTER. THERE IS ALSO AN INCREASE IN THE ACTIVITY OF THE SYMPATHETIC NERVOUS SYSTEM – TACHYCARDIA, INCREASED HEART RATE, INCREASED BLOOD PRESSURE.
2. STRUGGLE WITH HYPERCAPNIA. THE REACTION IS AIMED AT REMOVING EXCESS CARBON DIOXIDE. MECHANISM – EXTREME INHIBITION OF THE RESPIRATORY CENTER. THERE IS ALSO AN INCREASE IN THE ACTIVITY OF THE PARASYMPATHETIC NERVOUS SYSTEM – BRADYCARDIA, LOWERING BLOOD PRESSURE.
3. PARALYSIS OF THE RESPIRATORY CENTER AND RESPIRATORY ARREST.
4. AGONAL RESPIRATORY MOVEMENTS – BY ACTIVATING THE SPINAL RESPIRATORY CENTERS.

QUESTIONS FOR SELF-CONTROL OF KNOWLEDGE

1. GIVE THE DEFINITION OF HYPOXIA.
2. NAME THE MAIN TYPES OF HYPOXIA.
3. WHAT IS THE MECHANISM OF POLYCYTHEMIA IN ACUTE AND CHRONIC HYPOXIA?
4. WHAT TYPE OF HYPOXIA OCCURS IN A TEST ANIMAL WITH MASSIVE BLOODLETTING?
5. WHAT TYPE OF HYPOXIA DEVELOPS WHEN CYANIDES ARE INTRODUCED INTO THE BODY?
6. WHAT IS THE DIFFERENCE BETWEEN ASPHYXIA FROM HYPOXIA?
7. WHAT IS THE PATHOLOGICAL PROCESS THAT DEVELOPS IN AN ANIMAL WHEN IT IS PRIMED WITH CO?
8. NAME THE MECHANISM OF TOXIC ACTION OF CO.
9. WHAT IS THE ETIOLOGY AND PATHOGENESIS OF NEONATAL ASPHYXIA?
10. WHAT DETERMINES THE DURATION OF CLINICAL DEATH?

INDEPENDENT WORK OF STUDENTS IN THE CLASSROOM

EXPERIMENT 1. OBJECTIVE: TO STUDY THE PATHOGENIC EFFECT OF LARGE AMOUNTS OF CARBON DIOXIDE ON THE BODY.

METHODOLOGY: PUT THE MOUSE IN THE JAR, ENTER CARBON DIOXIDE INTO THE JAR FROM THE BALE MACHINE. OBSERVE CHANGES THAT OCCUR IN THE ANIMAL UNDER THE INFLUENCE OF CO_2 (BEHAVIOR, RESPIRATORY RATE, COLOR OF THE EYES AND VISIBLE SKIN).

EXPERIMENT 2. OBJECTIVE: TO STUDY THE EFFECT OF CARBON MONOXIDE ON THE BODY.

METHOD: THE MOUSE IS PLACED IN A JAR. THEN CO IS INTRODUCED FROM THE APPARATUS (CARBON MONOXIDE IS OBTAINED BY ADDING A FEW DROPS OF FORMIC ACID TO CONCENTRATED SULFURIC ACID). TAKE INTO ACCOUNT THE EFFECT ON THE MOUSE CO. NOTE CHANGES IN THE ANIMAL'S BEHAVIOR, CHANGES IN RESPIRATORY RATE, EYE COLOR, AND VISIBLE SKIN.

TASKS

1. A PATIENT WITH COMPLAINTS OF SEVERE PAIN IN THE LOWER ABDOMEN WAS TAKEN TO THE SURGICAL DEPARTMENT. UPON ADMISSION, IT WAS REVEALED: PALE SKIN, BLOOD PRESSURE – 70/40 MM HG, PULSE 120 BEATS PER MINUTE, WEAK FILLING, DEEP BREATHING WITH A FREQUENCY OF 22 PER MINUTE. THE PATIENT HAD AN URGENT LAPAROTOMY: BLEEDING FROM A RUPTURED FALLOPIAN TUBE WAS DETECTED AGAINST THE BACKGROUND OF AN ECTOPIC PREGNANCY. AFTER STOPPING THE BLEEDING, 1 LITER OF BLOOD WAS REMOVED FROM THE ABDOMINAL CAVITY. WHAT TYPES OF HYPOXIA DID THE PATIENT DEVELOP? EXPLAIN THE MECHANISMS OF THEIR DEVELOPMENT.

2. DURING A FIRE WAS DISCOVERED THE DECEASED MAN WITH THE BRIGHT PINK COLOUR OF THE SKIN. WHAT IS THE CAUSE OF DEATH, AND WHAT HYPOXIA IS IT ASSOCIATED WITH? WHAT OTHER TYPES OF HYPOXIA ARE POSSIBLE IN THIS CASE?

3. FROM THE PRODUCTION OF ANILINE DYES, AN EMPLOYEE WAS TAKEN TO THE HEALTH CENTER OF THE ENTERPRISE WITH COMPLAINTS OF NAUSEA, VOMITING, SEVERE HEADACHE, TINNITUS, FLICKERING OF FLIES BEFORE HER EYES, WEAKNESS, DROWSINESS. THE PATIENT HAS PRONOUNCED CYANOSIS OF THE SKIN AND MUCOUS MEMBRANES. THE BLOOD TEST REVEALED A LARGE AMOUNT OF METHEMOGLOBIN. WHAT FORM OF HYPOXIA DID THE PATIENT DEVELOP? WHAT ARE THE MECHANISMS OF ITS DEVELOPMENT?

4. GEOLOGIST IN THE EXPEDITION ARRIVED IN THE HIGHLANDS. ON THE SECOND DAY OF HIS STAY AT AN ALTITUDE OF 3000 METERS, THE GEOLOGIST'S CONDITION SIGNIFICANTLY WORSENED: HEADACHE, SHORTNESS OF BREATH, GENERAL WEAKNESS, AND SLEEP DISORDERS OCCURRED. WHAT FORM OF HYPOXIA DID THE PATIENT DEVELOP? WHAT ARE THE MECHANISMS OF ITS DEVELOPMENT?5. THE LITERATURE DESCRIBES AN AMAZING CASE. A FOUR-YEAR-OLD BOY FELL THROUGH THE ICE IN THE WINTER AND WAS IN ICY WATER FOR 20 MINUTES. WHEN THE CHILD WAS REMOVED FROM THE WATER, THERE WERE NO SIGNS OF LIFE, THE BODY TEMPERATURE DROPPED TO 27OC. HOWEVER, AFTER 1.5 HOURS OF RESUSCITATION, THE BOY WAS REVIVED. WHAT IS THE CAUSE OF CLINICAL DEATH? WHAT CONDITIONS PREVENTED IRREVERSIBLE CHANGES IN THE CEREBRAL CORTEX?