Ministry of Health of Belarus
Gomel State Medical University

Department of Orthopedic, Trauma and military field surgery
with the course of Anesthesiology and Critical Care Medicine

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SUBJECT: "Intensive care of diseases accompanied by acute respiratory failure"

Educational and methodical development for students
4th year medical faculty

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Methodical development is designed for self-study. It provides:

1. Background.
2. The purpose of training (skills and knowledge).
3. Basic sections.
4. Suggested Reading.
5. Questions for self-preparation.
6. Topics UIRS.
7. Teaching tools for organization of independent work of students
8. Training Material
9. Self-study.
10. Clinical problems and test control.

Relevance of the topic

Acute respiratory failure (ARF) - one of the most serious complications occurring in critical care practice. Later, her recognition and inadequate treatment can lead to death. ARF occurs in many situations: in violation of the respiratory center (drug overdose, poisoning, sleeping, coma, stroke), in violation of the airway (aspiration of foreign bodies, the secrets of the oral cavity, including the stomach and the mass of blood in wounds facial skeleton, the delay of bronchial secretions, in violation of cough in patients in a coma or injury of the chest with limited respiratory movements due pleural effusion, high standing of the diaphragm or chest pain syndromes and the upper floor belly, with functional impairment of respiratory muscles (paralysis or spasms) , with severe violations of electrolyte balance, especially deficiency of K + and acid-base status, in some infectious lesions and poisoning, in connection with changes in parenchymal lung tissue (alveolar liquid filling, pulmonary edema, embolism, traumatic injury). Given the multiplicity of causes of ARF, methods of diagnosis, prevention and treatment should own physician of any specialty.

Purpose of the lesson

To teach students the general principles pathogenetically substantiated intensive treatment of acute respiratory failure and features of its conduct at the most common condition.

The student should know:

- etiology, pathogenesis, types of acute respiratory failure
- clinical and diagnostic of acute respiratory failure
- ODN treatment guidelines
- methods of mechanical ventilation (ALV), complications of its holding
- methods of airway management
- methods of oxygen therapy
- features of intensive care at SGL asthmatic status, aspiration syndrome, respiratory distress syndrome, acute respiratory failure due to massive pneumonia, atelectasis, mechanical obstruction of the trachea and bronchi, subglottic edema

The student should be able to:

- be able to diagnose the extent of ODN
- be able to assist in the pre-hospital mechanical asphyxia
- to dosage and controlled oxygen therapy

• possess technique of spontaneous breathing with resistance exhalation
• Identify indications for assisted ventilation (VIVL) and ventilation (ALV)
• mechanical ventilation manual respirator
• determine the sequence of therapeutic interventions in relieving asthma status, acute respiratory failure due to massive pneumonia, atelectasis, mechanical obstruction of the trachea and bronchi, aspiration syndrome, respiratory distress syndrome
Sections studied before and needed for the session
• basic pharmacology of drugs
• Human Physiology
• physiopathology
• Internal Medicine
• surgical disease

Recommended Reading

Textbooks on pharmacology, normal and abnormal physiology, surgery, internal medicine for medical students.
Suggested Reading on lessons

Main Reading

1. Radiology for Anaesthesia and Intensive Care, Second Edition (Cambridge Medicine) Richard Hopkins, Carol Peden, Sanjay Gandhi
2. Handbook of Drugs in Intensive Care: An A - Z Guide, Fourth Edition.Henry Paw, Rob Shulman
3.Morgan Jr.., Edward J. Clinical Anesthesiology / G. Edward Morgan, Jr., Michael S. Magid / / M., 2015. - V.1, № 3. - p3-375.
4. Kattsung, Bertram G. Basic and Clinical Pharmacology / Bertram G.Kattsung / / 2017. - P.5-577.
5. Marini, John J. critical care medicine / John J. Marini, Arthur P. Wheeler. - M., 2012. - 992s.
6. Hams, AN Internal Medicine: Prakt. Guide: V3t. T3.Kn.2. / AN Hams. 2017. - 480p.
7. Intensive Care Medicine: Annual Update 2010/Jean-Louis Vincent
8. Recent Advances in Anaesthesia and Intensive Care 022 (Recent Advances)/A. P. Adams, J. N. Cashman, R. M. Grounds (Editors), 2000. - 464.
9. Chest Physiotherapy in the Intensive Care Unit, 2nd Edition/Colin F. MacKenzie, P. Cristina Imle, Nancy Ciesla
10.Handbook of Drugs in Intensive Care: An A - Z Guide 3rd Edition/Henry G. W. Paw, Gilbert R. Park
11.Online Resources

Questions for self-Questions on basic knowledge

1. Arterial hypoxemia
2. Pathological variability ventilation-perfusion relationships and mixed venous shunting blood to the lungs as a cause of arterial hypoxemia
3. Obstructive and restrictive disorders of alveolar ventilation
4. Inadequate resparitory ventilation the cause of arterial hypoxemia
5. Respiratory acidosis and alkalosis

6. Pathogenetic principles of mechanical ventilation
7. Neurogenic mechanisms of the pathogenesis of asthma
8. Pathogenesis of arterial hypoxemia and disorders of acid-base status in status asthmaticus

Questions about the topic studied

1. The concept of ARF. Types of ARF.
2. Monitoring of respiratory function (pulse oximetry, blood gases, monitoring).
3. Ventilation (mechanical ventilation). The main differences between mechanical ventilation and spontaneous breathing. The negative effects of mechanical ventilation. Complications of mechanical ventilation, prevention and treatment.
4. Methods of mechanical ventilation. Absolute and relative indications and methods of implementation. Equipment for ventilation, classification and function of respirators.
5. Prolonged mechanical ventilation. Auxiliary ventilation. Selecting the pulmonary ventilation, depending on the underlying pathology in surgical and medical patients. Features mechanical ventilation with positive end-expiratory pressure, the use of high-frequency ventilator injection.
6. Indications and technique of oxygen therapy, heliotherapy, percutaneous cannulation of the trachea and bronchi, therapeutic bronchoscopy.
7. Indications for tracheostomy and cricotomy. Complications. Traheostomirovannymi care patients.
8. Hyperbaric oxygen (HBO). The mechanism of action of HBO on the body. Indications and contraindications to HBO in resuscitation.
9. Resuscitation and IT in SGL, developed with massive pneumonia, atelectasis, asthmatic status, aspiration syndrome, bronchopulmonary and laryngospasm, subglottic edema.
10. Respiratory distress syndrome of adults.

Topics UIRS

1. Extracorporeal membrane oxygenation of blood
2. Especially Intensive Care hospital pneumonia

Teaching tools for organization of independent work of students

1. Computer database.
2. Objectives, test control.
3. Thematic sick.
4. Patient records.
5. Bank jobs for self-study.

Training Material

Acute respiratory failure (ARF) - sharply growing disparity between gas exchange (respiratory) metabolic needs of the body.

The efficiency of gas exchange measured by blood gas, which is determined by invasive, measures the partial pressure (tension) of oxygen (PaO2) and carbon dioxide (PaCO2) in arterial and mixed venous blood. Their regular measurement provides the most complete picture of the ventilation and oxygenation. It is particularly important to carry out these measurements at any abrupt changes of respiration.

Adequate ventilation is convenient to control the voltage of CO2 in arterial blood. Ventilation is reduced by reducing the amount of alveolar ventilation in general, or by increasing the proportion of physiological dead space. Alveolar ventilation in general may decline initially (depression of the respiratory center, severe airway obstruction, etc.), or again at fatigue of respiratory apparatus at high resistance.

Oxygenation measured by oxygen tension in arterial blood. Oxygenation falls at 1) reducing the fraction of oxygen in the inspired mixture below normal, 2) reducing the total alveolar ventilation (although artificial increase in the proportion of oxygen in the inspired mixture can compensate for this), 3) reducing alveolocapillary diffusion membrane, and 4) increasing the proportion of the shunt.
Arterial blood gas composition characterizes the efficiency of light as a gas exchange device, and gas composition of mixed venous blood entering the lungs, the indicator of the metabolic processes in the body.
The gas composition of arterial and mixed venous blood (mm Hg.)
Arterial blood mixed venous blood
PaO_2 90-110 37-42
$PaCO_2$ 34-46 40-52
Then the alveolar-arterial difference (PAO2-PaO2 normal is 9-15mm Hg) may serve as a criterion of inefficiency through the pulmonary gas exchange membrane, and arterio-and alveolovenous difference reflects the efficiency of gas exchange tissue.
Significant signs of NAM is to reduce the PaO2 below 60 mm Hg and an increase in PaCO2 above 50 mm Hg and a decrease in pH to 7.2 or lower when breathing air and at atmospheric pressure.
RF severity depending on hypoxia
AVD PaO2 mmHg. SpO2%
Moderate 60-90
Heavy 40-75
Hypoxic coma 30-60
Hypoxic death 20-35

SGL is not necessarily seen significant changes of blood gas composition. Some time on the "normal" gas composition is supported by the hard work of respiratory system, especially the respiratory muscles. Therefore, it is crucial to begin intensive respiratory therapy without waiting exhaustion of compensatory mechanisms external ventilation, exhaustion of respiratory muscles.
Pulse oximetry
Pulse oximetry is particularly useful in cases where you need to frequently monitor the oxygenation: no contraindications to pulse oximetry.
At the core are the principles of pulse oximetry and plethysmography. It is designed for non-invasive measurement of arterial oxygen saturation. The sensor consists of a light source (two light-emissive diode) and light detector (photodiode). Sensor placed on the finger or toe, on the earlobe - that is, where possible transillumination (translucence through) perfused tissues. Oximetry is based on the fact that oxyhemoglobin and deoxyhemoglobin differ in their ability to absorb the rays of the red and infrared spectrum (Lambert-Beer law). Oxyhemoglobin (NO2) strongly absorbs infrared light (wavelength 990 nm), whereas deoxyhemoglobin intensively absorbs red light (wavelength 660 nm), so that deoxygenated blood is attached to the skin and mucous membranes bluish color (cyanosis). Therefore, based on the change in oximetry absorption of light pulses in the arteries. The ratio of absorption of red and infrared absorption wave is analyzed microprocessor, resulting in a pulsating flow is calculated saturation of arterial blood oxygen - SpO2 (S - from the English. Saturation - saturation (saturation), and p - from the English. Pulse - pulse). Ripple artery is identified by plethysmography, which takes into account the light absorption of non-pulsed flow of venous blood and tissues and make proper corrections.

Pulse oximetry, in addition to oxygen saturation, assess tissue perfusion (pulse amplitude) and measures the heart rate. As is normal oxygen saturation is about 100% (in adults 96-98%), the more commonly cases deviation from this figure indicates a serious disease. Depending on the individual curve of oxyhemoglobin dissotiation SpO2 90% may correspond PaO_2 <65 mm Hg. Art. These data are comparable to those of physical research. Cyanosis occurs when the concentration of desoxyhemoglobin>5g/l, which corresponds to SpO2 <80%.
Classification ARF associated with the pathology of external ventilation (pulmonary causes of ARF)

1. Central ARF is associated with depression, agitation, discoordination of the respiratory center.
• Inhibition of the respiratory center. Cause: cerebral blood flow, the effect of drugs (opioids, hypnotics, sedatives), severe traumatic brain injury, neural infections, brain tumors, damage to the brain stem.
• Overexcitation respiratory center. Causes: severe traumatic brain injury, cerebral edema, damage to the hypothalamus, neuroinfection.
• incoordination of the respiratory center. Causes: diabetic coma, endogenous intoxication.
2. Neuromuscular ARF associated with impaired transmission of nerve impulses from the respiratory center to the respiratory muscles or with the pathology of respiratory muscles.
• Pathology of the pulse nervously conducting system. Causes: trauma and spinal cord diseases, and abducens (traumatic intersection, tumors, ischemia, infectious inflammation, amyotrophic lateral sclerosis, demyelisation, polio, neuropathy, damage n. Phrenicus innervating the diaphragm).
• Pathology of the pulse at the neuromuscular synapse. Causes: Myasthenia gravis (an autoimmune synaptic mediator of damage), intoxication, toxic damage synapses and neurotransmitters (botulism, tetanus poisoning FOC) or a depression medication (muscle relaxants).
• Pathology of respiratory muscle contractility. Reasons: nonspecific myopathy, cachexia, collagen, myodystrophy, electrolyte disturbances (especially hypokalemia, hypomagnesemia).
Central and neuromuscular ARF is usually attributed to the so-called ventilation respiratory failure. This emphasizes the violation of the mechanical process of external ventilation. ARF of ventilation - a direct indication for emergency ventilation even until the reasons ARF and early specific treatment (if we exclude tension pneumothorax).
3. Torakodiafragmal ARF is associated with the violation of the integrity of the skeleton of the chest, diaphragm injury, acute respiratory mixture distribution with pressure or collapsing by lung, pain and high standing of the dome diaphragm.
• Violation of the integrity and mobility of thoracic cage. Reasons: multiple fractures of the ribs, sternum, traumatic rupture of the diaphragm.
• Compression and / or collapse lung tissue. Reason: open or tension pneumothorax, hydrothorax, hemothorax. When tension pneumothorax chest tube drainage before starting mechanical ventilation is impossible, since it may worsen due to compression of mediastinal forced admission breathing gas in the pleural cavity on the side of destruction, and further compression of the lungs and heart.
• High standing dome of the diaphragm. Causes of obesity, intestinal paresis, ascites.
• Restricting the mobility of the chest associated with the pain factor. Causes: broken ribs, thoracic surgery or abdominal cavity, etc. dramatically reduced the amplitude excursions of the chest, there is insufficient alveolar ventilation, atelektasing, hypoxemia, impaired release of

CO2.
Torakodiafragmal ARF may be accompanied by severe disorders once the mechanics of external ventilation. In this case, the ARF is initially vent character, immediately followed by hypercarbia and hypoxemia. In another situation, (hydro-, hemo-, pneumothorax, flatulence) elimination of CO2 is maintained by compensatory mechanisms normocarbia maintained that there should not be misleading when assessing the gravity torakodiafragmal ARF.
4. Obstructive ARF. Associated with acute airway at some level. One of the most common and dangerous types of ARF.
• retraction of the tongue, larynx block gastric contents, foreign body in the throat, bronchi, bruising, swelling, and so on;
• Traumatic obstruction of the upper respiratory tract;
• Inflammatory swelling of the vocal cords, subglottic laryngitis, congestion sputum inflammatory secret in violation of the drainage function of the bronchi;
• Acute bronchospasm bronhoreya, swelling of the mucous membrane of the large bronchi in asthma or COPD exacerbation;
• Early expiratory closure of small airways.
• Fine endotracheal (tracheostomy) tube or occlusion
Regardless of the cause of obstructive pathology causes a sharp increase in airway resistance. High airway resistance increases the work of breathing, his energy and oxygen price, compensatory mechanisms are exhausted, there is a dangerous hypoxemia, joined by hypercapnia.
5. Restrictive ARF. Associated with severe acute violation of elasticity of lung tissue, atelektazirovaniem, blockade alveolocapillary membrane.
• polysegmentary pneumonia, fibrotic processes nonobturating atelectasis;
• ARDS, Mendelson's syndrome;
• Cardiogenic and noncardiogenic pulmonary edema;
• Heavy gestoses
At the heart of the deterioration elasticity are: inflammation in the lung tissue, alveolar collapse, interstitial edema. In this case, mostly broken ventilation, reduced ventilation-perfusion ratio (V / Q <0,8). Develops desoxygenesing shunting blood from right to left - the most characteristic syndrome, typical restrictive ARF. Developing hypoxemia resistant to oxygen therapy. Also particularly restrictive ARF is a marked increase in work of breathing to overcome the high elastic resistance "hard" light.
6. Perfusion ARF. Associated with the restriction of blood flow in the pulmonary artery branches and increased physiological dead space.
• Thromboembolism branches of the pulmonary embolism (PE);
• Severe hypovolemia (blood loss, dehydration).
There is a sharp decrease in perfused lung zones in relation to the ventilated (ventilation-perfusion ratio V / Q> 1), increased physiological dead space, reducing the area of real gas exchange. Progressing hypoxemia and hypoxia, tachypnea not compensate them. When pulmonary embolism detected hemodynamic and right ventricular failure.
Torakodiafragmal, obstructive, restrictive and perfusion ARF in the literature often referred to as parenchymatous or gas exchange (hypoxemic) ARF. When parenchymal ARF at the forefront progressive hypoxemia, often resistant to oxygen therapy. PaCO2 levels can long remain in the normal range due to compensatory mechanisms external ventilation.
The main causes of ARF extrapulmonary

1. Breathing a gas mixture with low oxygen content. O_2 concentration in the inspired mixture <17% for the untrained organism leads to the clinical manifestation of ARF. A typical example - rise to a height of> 3 km without prior adaptation and an additional source of oxygen.
2. Severe acute anemia (Hb <65-70 g / l). Hypoxia hemic origin, although SaO_2 indicator may remain in the normal range. Characterized by the decrease PvO_2 <30-32 mm Hg and SvO2 <65% due to increased oxygen extraction ratio fabrics.
3. Hemodynamic abnormalities. Low blood pressure (systolic blood pressure <80-85 mmHg) from any cause leads to a decrease in O2 delivery and tissue hypoxia, although PaO2 and SaO2 may remain in the normal range. For tissue hypoxia circulatory origin also characterized by the lower RvO2 <30-32 mm Hg and SvO2 <65% due to increased oxygen extraction ratio fabrics.
4. Carbon monoxide poisoning. In this case, the hemoglobin becomes stable compound with CO to form carboxyhemoglobin (HbCO). When carbon monoxide poisoning dramatically reduced PaO2, SaO2 but may remain in the normal range because of the red color HbCO.
5. Cyanide poisoning. Cyanide causes blockade of tissue enzyme cytochrome oxidase, involved in the transport of molecular oxygen in the tissue (internal) respiration. Hypoxia tissuetoxyc genesis with attendant phenomena ARF, although PaO2 and SaO2 remained within normal limits.
6. Metabolic ARF. Associated with overproduction of tissue CO2 (hyperthermia, sepsis, active catabolism, hyperthyroidism, necrotizing pancreatitis, peritonitis, burns) with a concomitant increase in oxygen demand. Characterized by the decrease PaO2 and RvO2.
The clinical picture of SGL
• Tachypnea (> 25 per minute), shortness of breath;
• bradypnea (<10 min), sleep apnea;
• Depression or loss of consciousness (if PaO2 less than 30 mm Hg)
• Cyanosis (when PaO2 below 60 mm Hg and SaO2 less than 90%);
• Participation of the auxiliary respiratory muscles in the act of inspiration or expiration (abdominal muscles, torso, chest, muscles, neck, face);
• thoracoabdominal asynchrony;
• Tachycardia, bradycardia;
• The progressive weakening of breathing on auscultation ("dumb light"), and 2-way spread rales (pulmonary edema);
• Progressive hypercapnia;
• Progressive hypoxemia (PaO 2 declines, SaO2, RvO2, SvO2;
• Anemia, carboxygemoglobinemia (at hemic ARF);
• The X-ray - a massive infiltration, pneumothorax, hydrothorax).
Diagnosis of ARF:
• physical examination, medical history;
• Biochemical blood (protein metabolism, electrolytes, urea, creatinine, bilirubin);
• A study of blood gas composition;
• Bronchoscopy (if necessary);
• X-rays of the chest;
• Computed tomography (magnetic resonance imaging);
• A study of ECG, echocardiography with Doppler;
• If indicated - consultation surgery, urology, ENT, tuberculosis, spirometry;The simplest assessment tests SGL:
• The restrictive disorders (lung compliance) decreased tidal volume ventilation (le

s than 5 ml / kg) and vital capacity (less than 15 ml / kg), slightly shortened breath and its relation to the total time of the respiratory cycle to be smaller 0.43;
• In obstructive disorders because of increased airway resistance reduces the dynamic (speed) performance;
• If the alveolocapillary diffusion occurs against hypoxemia normo-or hypocapnia, and arbitrary hyperventilation does not decrease but increases hypoxemia, as judged by the degree of cyanosis, or according to oximetry;
• With a large alveolar shunt hypoxemia little downwards despite inhaled O2.
Treatment.
I. Emergency:
a) restoration of the airway:
• tri-Safar is straightening the head of the mandible and the nomination of the opening of the mouth.
• The introduction of the oral and nasal airways can support cross at the throat, as prevents the tongue. Atraumatic introduction orofaringial duct helps application spatula, bent at an angle, and the language the patient lying on his back, push forward up and put duct in the oropharynx. To prevent possible laryngospasm and gagging to be done to the duct does not touch the back wall of the pharynx and epiglottis, and the grease duct ointment containing anesthetic.
• intubation performed through the mouth (orotraheal) or nose (nazotraheal).
• Conikotomia (crikotyreotomia) - can play an important role in emergency airway management. Unbend his head under the blade enclose roller. To find Cricothyreoid bunch, do not hesitate to place the index finger of the convexity of the thyroid cartilage and slide the neck down. Bulge begins to decrease, and then glide your finger in a small depression and then go up. It is this deepening is Cricothyreoid ligament, and the next lift a finger mark the beginning of the cricoid cartilage. Make a cross-section of skin 1-1.5 cm long entry 2 finger incision so that the tip of the nail bone rested against the membrane is inserted through the opening in the trachea tracheostomy cannula. In primitive conditions, instead of a scalpel can use a pen-knife, and instead of a pipe cylinder pen, a piece of rubber tubing, etc. The main thing - do not get lost! Do not be afraid bleeding, do not be afraid to make an infection - the death of a lot worse blood infection.
• Tracheotomy (tracheostomy) - is used to eliminate the obstruction at the level of the larynx and above many days during mechanical ventilation, and to prevent aspiration of the toilet respiratory tract, with bulbar disorders with impaired swallowing and cough. If possible, it is better to perform a tracheostomy on the background of endotracheal intubation. At 2-3 tracheal rings do transverse skin incision with dissection of superficial muscles of the neck to the deep fascia. Soft tissue above the deep fascia stupidly shifted up to the level of cricoid cartilage and down to the level of 4 rings of the trachea. Deep fascia dissected longitudinally with blunt and sharp outcrop 2 and 3 rings, stop bleeding and, if necessary, the intersection of thyroid isthmus between the two clamps. If you can shift up or down the isthmus, there is no need to cross. Be careful of damaging blood vessels and nerves, taking them apart. Dissect the tracheal rings better Bjork: in front of the trachea cut out lingulate flap tip directed cranially. The flap is folded down, and its top sewn into the bottom corner of the wound. Convenience of this method lies in the fact that the change of the cannula is easier because trachea is fixed in the wound. If there is no need for a tracheostomy, the flap is sutured to the old place, which reliably seals the trachea. Tracheostomy requires careful care: frequent antiseptic, moisturizing, and warm inhaled air disinfection, removal of phlegm.

• Rehabilitation of the tracheobronchial tree (LDP). Indications: cough chaotic motion patient sputum visible through the transparent wall of endotracheal tube, the emergence of large bubbling rale auscultation over the area of the trachea, increasing the peak pressure at a flow ventilation.

Bronchoscopy may be used for diagnostic and therapeutic purposes. Indications for therapeutic bronchoscopy: excess mucus or sekretoproduktsiya airway, very viscous and thick sputum, aspiration syndrome, the need to remove mucous (purulent) "blocks", the removal of foreign bodies, auscultatory respiratory depression on one side of the lung after endotracheal prevent incorrect state tube and pneumatic and hydrothorax. The procedure is performed under local or general anesthesia. In either case shall premedication (atropine, promedol) 30-40 minutes prior to the study. The procedure is performed after preoxygenation with 100% oxygen for 2-3 minutes. For general anesthesia is normally used thiopental sodium, exercise relaxation and mechanical ventilation. With the introduction of a bronchoscope anesthetized area of the carina or spray lubrication anesthetic. With the deterioration of the patient manipulation immediately cease. Bronchoscopic lavage with sanitation: after the introduction of a bronchoscope suction bronchial contents and take the secret for bacteriological examination. Then turn to the right and left bronchi through a catheter pour 5-10 ml of warm saline solution, which can add a 4% solution of bicarbonate, enzymes. Then pour the solution through the catheter suction. Completely suck pour the solution is not possible, therefore, be appropriate to the patient position for postural drainage. The solution was poured into each bronchus 4-8 times, until the washings are clean. Before each infusion make a break for at least 2-3 minutes, during which produce full ventilation.

• Anti-inflammatory, anti-inflammatory, antispasmodic therapy

b) inhalation of oxygen

c) ventilation (mechanical ventilation).

II. The planned combination therapy SGL: after removing the threat to life can be established physiological mechanisms and nosological form of the disease, determine the tactics and means of therapy.

III. Symptomatic therapy: is carried out to prevent the deepening of ODN by secondary functional disorders.

• correction of metabolic disorders, acid-base status (CBS), the water and electrolyte balance (EBV);

• detoxification

• methods that improve sputum production and improve its properties;

• antihypoxants.

Oxygen therapy.

Types of oxygen therapy:

• inhalation (lung)

• noningalation

• oxygination hyperbaric (HBO) - a separate form of oxygen therapy, combining features of Inhaled neingalyatsionnyh ways.

Indications:

• All types of hypoxia than tissuetoxyc (violation of tissue redox enzymes prevents waste delivered O2), clinical signs - cyanosis, tachypnea, metabolic acidosis, decreased PaO 2 of 70 mm Hg. Art. and less, SaO2 <80%;

• Voltage compensatory reactions to the fall in pO2 in the surrounding gas environment (eg, low barometric pressure at high altitudes);

• poisoning, especially carbon monoxide;
• patients with cardiac and respiratory failure in order to restore the therapeutic action of some drugs is reduced in hypoxia (cardiotonic action of cardiac glycosides, diuretic effect of diuretics);
• Improve liver and kidney function in patients with lesions of these bodies;
• enhance the effect of treatment of malignancy.
Indications for topical application of oxygen:
• local hypoxia;
• local trophic disorders against vascular lesions;
• subacute inflammatory processes;
• wounds infected with anaerobic flora.
Absolute contraindications for oxygen is not present, but the choice of method and technique of her must meet the individual patient (age, the nature of the pathological process.)
Inhaled oxygen includes all routes of administration of oxygen to the lungs by inhalation. Inhalation O_2 conducted at a concentration of 30 to 100%. Oxygen is inhaled with oxygen via nasal cannula apparatus, face mask, endotracheal tube, tracheostomy cannula. In children and adults rarely use oxygen tents, tents.
Nasal cannula mounted in both nasal passage to a depth of 1 cm and a band-aid fix. Efficiency increase FiO_2 using nasal oxygen supply depends on the strength of the oxygen flow, strength and duration of the inspiratory breath patients, the incidence of his breath. Power flow to 8 l / min is not comfortable for the patient and cause damage to the mucous membrane of the nasopharynx.
Face masks used for oxygen, can be simple (without exhalation valve and the tank), or more complex, provides for the tank, which provides a partial return inspired gas mixture. This leads to a greater increase the flow of oxygen in the inspired air (FiO2). Using a simple mask gives increase FiO2 to 3.5-5% for each liter of oxygen per minute at a flow rate of 6-10 l / min. The presence of the tank can further increase the efficiency of oxygen therapy, but is fraught with the advent of hyperoxia its possible toxic manifestations in the form of free radicals that damage the alveolar epithelium and pulmonary capillary endothelium. These lesions become significant after a few hours of breathing 100% oxygen. Rational way for prolonged oxygen therapy - the minimal concentration that provides the lower margin of the oxygen parameters. The optimum concentration of O2 in the breathing mixture must be the minimum concentration that provides a lower margin of PaO_2 (about 75 mm Hg and SrO2 90%), rather than the more normal or overweight.
If inhalation of oxygen is in the tent, a tent or a nasooral (facial) mask, ie gas passes through the mouth, nose and nasopharynx, the extra moisture it is not necessary since it adequately hydrated in the airways. If oxygen is fed through nasal catheters or endotracheal tube or tracheostomy cannula, and dehydration patient - it takes a special moisturizing breathing gas. To do this, it is advisable to use the aerosol inhaler, creating a gas mixture slurry droplets of water (about 1 mm), the evaporation of which saturates airway gas with water vapor up to 100%. Transmission of oxygen through a container of water is less effective because large bubbles of oxygen does not have time to get enough water vapor. Inhalation of helium-oxygen mixture is designed to reduce aerodynamic drag, ie to improve the airway with subglottic stenosis, bronchiolitis, status, etc. In bronhoastmatic status Helium improves transport of O_2 in the mixture to which it is applied to the alveolar membrane. Reducing air resistance, helium mixture reduces the work of respiratory muscles, spent less O_2. Most often helium-oxygen mixture is used in a concentration of 70%: 30%. For inhalation use as modes and devices, as for oxygen therapy. For dosage of helium can

be used anesthesia machines with dosimeters nitrous oxide dosimeter performance multiplying by 3.4 (this value is obtained by dividing the square root of the density of the two gases). Complications of inhaled oxygen and prevention:

• respiratory failure or significant hypoventilation with hypercapnia - occurs in patients with reduced sensitivity of the respiratory center to increase the concentration of CO2 in the blood. In these cases, the breath driven from carotid chemoreceptors hypoxemia, which wound up in CT. To prevent this complication is recommended for conditions with the presence or threat of depression of the respiratory center (especially if respiratory arrhythmia) begin CT 25% oxygen mixture and gradually increase the concentration of oxygen in it to 60% on the use of means of pathogenetic therapy of central respiratory disorders;

• denitrogenation (leaching of nitrogen from the body) - edema and hyperemia of the mucous membranes in the various cavities (frontal sinuses, etc.), light absorption microatelektaesis;

• oxygen intoxication - develops during prolonged inhalation of a mixture with a high concentration of oxygen or pure oxygen. Excess oxygen interferes with the normal chain of biological oxidation, cutting them and leaving a large amount of free radicals, is irritating to the tissues. Major manifestations of oxygen toxicity are signs of respiratory and central nervous system. Initially, the patients' dry mouth, dry cough, burning sensation in the chest, chest pain, damaged ciliated epithelium, disrupted drainage function of bronchi, increasing their resistance to gas flow. In the lung collapses surfactant increases the surface tension of the alveoli, are developing micro-and then makroatelektaesis, pneumonitis. Decreased vital capacity and reduced diffuse lung capacity increases uneven ventilation and blood flow. Spasm of peripheral vessels, acroparesthesia. CNS disease is most often seen convulsions and impaired thermoregulation, are also possible mental disorders, sometimes develops a coma. Nonigalation oxygen combines all extrapulmonary routes of administration of oxygen - enteral, intravascular (including using a membrane oxygenator), subcutaneous, intracavitary, intra-articular, subconjunctival, skin (general and local oxygen baths). Enteric oxygenation, ie introduction of oxygen into the digestive tract through the probe, carried out with a dosimeter or pick mode of administration on the number of oxygen bubbles passing through the bank machine Bobrova in 1 min. Sometimes using a so-called enteric without gastric tube oxygenation - swallowing sick oxygen in the form of a special foam or mousse. The effectiveness of this method of KT, which was used for the treatment of abortion pregnant, gastritis, liver failure, prevention of aging, etc., not confirmed. Extracorporeal membrane oxygenation (ECMO) - carried out using a membrane oxygenator, which consists of two parallel layers of thin polymer film, which takes place between the blood and the outside surrounding the film 100% O2. Through the micropores of the film is free of O2 and CO2, but the plasma and blood cells are delayed. Blood movement provides pump blood back from one vessel and return it in another. Through a membrane oxygenator is only part of the BCC, which allows you to use it for a few days or even weeks. ECMO is a traumatic deficiencies of blood cells, the inability to remove damaged cells from the blood (as occurs in the lungs), oxygenizing only part of the BCC. It should be noted that the diffusion capacity "of the membrane lung" for O_2 and CO_2 is approaching capacity alveolocapillary membrane. ECMO is indicated in patients with PaO_2 of 50 mm Hg., When the patient is on the ventilator in the mode of PEEP (5 cm H_2O) To the inhalation of 100% O_2. Oksigination hyperbaric (HO) - a separate form of oxygen therapy, combining features of Inhaled nonigalation ways and is essentially self-treatment. The method is based on the therapeutic application of O2 pressure greater than one atmosphere absolute. HBOT sessions conducted in hyperbaric chambers. As a result of inhalation of O_2 at high pressure to the

tension in body fluids increases, which leads to increased infusion of O_2 to the cells. Normally, the oxygen capacity of the blood of 20.3%, of which about 0.3% of the oxygen dissolved in the plasma (0.3 ml to 1 liter and 15 ml in 5 liters). O_2 saturation of Hb by inhalation air - 96-97%. Full saturation of Hb occurs at O_2 in the breathing mix to 35%. A further increase in pO2 will have no effect on the oxygen capacity of Hb, but will result in a linear increase in plasma levels of dissolved O_2. For each additional atmosphere dissolves in the blood pressure of 2.3 O2. Therefore, when breathing O_2 at 3 atmospheres pressure in the blood plasma of a solution of 6% O_2, which corresponds to the normal O_2 consumption at rest - his arteriovenous difference for O_2. In this case, the oxygen capacity of the blood is quite sufficient to sustain life (the phenomenon of "life without blood"). Method is shown in hypoxic conditions, intractable inhalation oxygen therapy. Special indications for HBO treatment are anaerobic infections, gas embolism and poisoning hemic poisons. HBO has a positive effect in all cases of shock, when there is hypoxia, connected with the violation of the rheological properties of blood and microcirculation. This group may include all the critical (terminal) state. HBO is effective in all types of hypoxia: hypoxic, circulatory, hemic and gistotoxical, ie when the mismatch between demand cells in G2 and its supply to it. The rapidity of clinical effect of oxygen deficiency no one method can be compared with HBO. When exposed to therapeutic regimens HBO slows and deepens breathing, decreased tachycardia, normal blood pressure, decreased cardiac output and organ blood flow, increased peripheral vascular resistance. The toxic effect of O2 on the cell due to the inhibition of certain respiratory enzymes. Acute poisoning is affected CNS (convulsions), autonomic nervous system (such as nausea, dizziness, blurred vision, paresthesia). Upon the termination of the session all the complications of HBO rapidly disappearing, and the effects are not observed.

Contraindications to HBO:

• epilepsy,
• presence of cavities in the lungs,
• severe hypertension,
• violation of the Eustachian tube patency,
• Drain bilateral pneumonia, pneumothorax, acute respiratory infections,
• claustrophobic
• Hypersensitivity to the O2.

Ventilation (ALV)

Not every patient with ARF requires immediate transfer to the ventilator. The decision to initiate mechanical ventilation is made based on clinical symptoms, laboratory data and instrumental examination.

Clinical indications for mechanical ventilation:

• Apnea or bradypnea (<8 per minute), tachypnea> 35 per minute;
• Hypoxic depression of consciousness;
• Shallow breathing on auscultation spread zones "dumb light" in patients with severe restrictive or obstructive diseases (asthma status);
• Excessive work of breathing, and depletion of accessory muscles;
• The progressive cyanosis and moisture of the skin;
• Coma in violation of swallowing and cough reflex;
• Repeated convulsions, requiring the introduction of high doses of muscle relaxants or sedatives;
• Progressive tachycardia hypoxic genesis;

• Progressive alveolar edema;
• cardiac arrest.

Laboratory and instrumental indications for mechanical ventilation:

• Progressive hypoxemia refractory to oxygen;
• PaO_2 <60 mm Hg (<65 mm Hg with an oxygen flow of 5 l / min);
• SaO_2 <90%;
• $PaCO_2$> 55 mm Hg (COPD patients> 65 mm Hg);
• VC <15 ml / kg

Methods of mechanical ventilation

1. Ventilation without the use of tools (breathing techniques "mouth to mouth" and "mouth-to-nose") - are used to the sudden apnea from any cause, until breathing is restored or not be applied other methods of mechanical ventilation. Despite the fact that in the exhaled air contains only 15-17% 2-4% CO2 O_2 ie expiratory methods are highly effective in maintaining the necessary gas exchange for tens of minutes or even hours. The first portion of the injected air (about 150 ml) comes from an anatomic space resuscitation, ie is the ambient air. In addition, imperfect gas composition of air blown by an increased volume. Expiratory ventilation methods can be applied using a face mask, duct intubation and tracheostomy tube.

2. IVL manual respirator. The most common two types of respirators hand - self-expanding bags "AMBU") and corrugated bellows. A significant advantage of manual respirator patient is blowing air, convenience and ease of use, the ability to enrich the mixture was injected O2, small size and weight. For blowing air into the lungs using muscle power source. Self-expanding bags (ARA-1, "Ambu", etc) have three functions:

• Bag, cracking down after compression (inhalation) due to the elastic properties of its own;
• irrevocable valve with a connector for a mask or endotracheal tube, allowing for passive exhalation to the atmosphere;
• inlet valve fitting to enrich breathing gas O2.

Tidal volume 0.5-1L, the frequency of 12-15 per minute. When applying to the input fitting respirator 100% O2 in the amount of 0.5-1 l O2 blown into the mixture reaches 80-90%.

3. Mechanical ventilation - ventilation with a breathing apparatus.Ventilators provide positive airway pressure, periodically creating a pressure gradient between their breathing system and alveoli, resulting in an active forced inhalation, exhalation is passive. Automatic respirators differ in the type of drive (working on the energy of the compressed gas or electricity), for switching phases of the respiratory cycle (pressocircle, taimecircle, frequency and mixed), the characterization of inhalation (constant flow generators, generators, constant pressure), may also be a microprocessor-controlled providing different ways of switching characteristics and inspiration. The devices can be portable and stationary. The method of mechanical ventilation Performed through an endotracheal tube ventilation, tracheostomy, stream (without intubation and tracheostomy) and through a face mask. For the prevention of obstructive disorders are used all methods toilet airways including artificial cough in different body positions.

In respirators can adjust the phase of inhalation and exhalation, pauses, changes in tidal volume, pressure, frequency and speed gaseflow. Mode is selected on the minute ventilation and tidal volume, respiratory rate, peak value and the pressure curve of inhalation and exhalation, the duration and the phase relations breath, an exhalation and pause.

There are two basic methods of organization of control of the ventilator:

• Ventilation to control the volume (Volume Controlled Ventilation - VCV) is designed to provide the given values of tidal volume and minute ventilation. Create airway pressure is

derived factor and limited dangerously high pressures. Application: temporary postoperative mechanical ventilation, mechanical ventilation of short duration in obstructive pathology ventilator on relatively intact lung (central and neuromuscular ODN), or the apparatus has to make provision for only the volume ventilator.

• Ventilation to control the pressure (Pressure Controlled Ventilation - PCV) first determine the amount of pressure in the airways. Tidal volume is not directly specified, his account when any pressure and control the means of measurement. PCV is the most effective method of mechanical ventilation in restrictive disease.

By the degree of participation of the patient / ventilator may include the following options ventilation:

1. Forced ventilation (Controlled Mechanical Ventilation - CMV). The mode in which a fixed amount of gas mixture at a given frequency comes from a respirator in the respiratory tract. When you try to separate inhalation respirator does not start. Forced ventilation is required for the complete inability of the patient to spontaneous breathing (drug poisoning, severe brain damage, the use of muscle relaxants). For patients with preserved spontaneous breathing mode is very inconvenient.

2. Auxiliary ventilation (VIVL) (Assisted Mechanical Ventilation - AMV) defining principle VIVL - with inadequate ventilation of the patient mask takes part of the work of breathing. It has the options:

• Add the missing volume of air (gas mixture) at low tidal volume;
• breaths (5-8 insufflations in minutes) on the background of a lack of spontaneous ventilation;
• autoreguliruemaya ALV - artificial injection included on the discharge generated by an attempt to breath a patient.

The auxiliary mode used for the treatment of ARF in the spontaneous breathing mode also include a positive end-expiratory pressure (PEEP, PEEP) and the mode of continuous positive airway pressure (CPAP, CPAP). At first maintained normal breath, but by the end of the output in the lungs creates a positive pressure, and the second - the same breath as the first, but as you breathe into your lungs fed special air backup, maintaining positive pressure. The main thing in these modes that mean intrapulmonary pressure is above normal. This increases lung compliance due to reduced pulmonary blood volume prevents expiratory airway closure, atelectasis and straighten increases functional residual capacity, decreased alveolar shunt, improved ventilation-perfusion ratio, decreasing the content of extravascular fluid in the lungs, reduces the penetration of water into the alveoli, improving gas exchange. Testimony to PEEP / CPAP is respiratory distress syndrome, pulmonary edema, pneumonia, atelectasis, exacerbation of chronic nonspecific lung disease, prolonged immobilization in bed.

Jet ventilation can be performed without sealing the airway. This method of intensive therapy based on SGL air intake of a directed jet of gas. After an injection needle into the tube bronchoscopy, endotracheal tube or directly into the respiratory tract is periodically thin stream of O2, which sucked the air entering the lungs - they inhale. When the injection of oxygen jet breaks, passive exhalation occurs under the elastic recoil of the lungs. Duration and tidal volume depends on the duration of the injection pressure and flow. Jet velocity and the presence of a plateau at the end of the entrance are of great importance for the uniform distribution of intrapulmonary gas, especially in obstructive mechanisms NAM. Jet ventilation in critical care can be done through a needle introduced into the trachea under the pressure of the jet at least 3 atm. Interruption of the jet can be done by manual or automatic. Most often jet ventilation is an aid, provides for various invasive treatment Nam, for example, lung lavage, bronchoscopy,

aspiration from the respiratory tract. Other manipulations in which unattainable tightness, also serve indication for jet ventilation. Effect of mechanical ventilation on the function of the body. When mechanical ventilation normalized blood gases, respiratory and metabolic acidosis respiratory origin. General and functional capacity of the lung ventilation increases due to distention of atelectasis, improve drainage of phlegm, reduce swelling and blood filling the lungs. Improves metabolism, normal heart rate and blood pressure, liver, kidneys and central nervous system. On the improvement of the functions of organs and systems, and clinical evidence and the results of functional and biochemical studies. However, prolonged or too saline mode ventilation can be observed and adverse functional consequences of mechanical ventilation.

With hardware method ventilator (manual and automatic), the following complications: Complications auxiliary techniques:

• fracture of the cervical vertebrae (fracture of the odontoid process of the 2nd cervical vertebra) in coarse hyperextension of the head
• mucosal injury in the exercise ventilation ducts through the naso-or oropharyngeal airway
• reflex reactions (provocation laryngospasm, vomiting, aspiration) with the introduction of air
• complications of intubation

Complications main mode:

• pulmonary barotrauma leading to tension pneumothorax
• Disconnect hoses and connectors
• airway obstruction
• pneumonia and atelectasis
• impaired gas exchange (respiratory alkalosis - is the result of hyperventilation, with hypoventilation - hypoxia and respiratory acidosis)
• hemodynamic disturbances due to impaired venous and lymphatic return in the chest cavity, reduce cardiac output
• swelling and dehydration - the result of decreased urine output, due to stimulation of antidiuretic hormone, activation of the renin-angiotensin system.

During prolonged mechanical ventilation can worsen lung compliance due atelektasing different bands of light in connection with violation of respiratory drainage and ventilation-perfusion ratio, degradation of surfactant (especially when large amounts of ventilation) and interstitial edema. Total dehydration or lack of moisture in the ventilator breathing mixtures is dangerous, as damaged mucociliary mechanism of bronchial drainage because of insufficient moisture mucosa. For the prevention of obstructive disorders used the toilet all the ways of the airways. Prevention of infection is easily accomplished by disinfecting the breathing system, the use of antibacterial filters and antimicrobial therapy. Infusion therapy with mechanical ventilation is important because without sufficient hydration of tissues violated the rheological properties of sputum. As with mechanical ventilation in the tissues may be retained water and interstitial edema occur, it may also stimulate diuresis. During mechanical ventilation requires careful monitoring of hemodynamic parameters, if necessary use inotropic support.

So, now is the most active ventilation by critical care ODN, but the harmony between its activity and efficiency is achieved with a combination of mechanical ventilation with other treatments

Features intensive care ARF at different pathology. Intensive management of status asthmaticus. Status asthmaticus is defined as a condition complicating bronchial asthma and characterized by an increase in intensity and frequency of asthma attacks against resistance to standard therapy,

inflammation and swelling of the bronchial mucosa in violation of their drainage function and accumulation of thick mucus.
In the body, the patient developed the following pathological changes:
• violation of the drainage function of the bronchi;
• inflammation and swelling of the bronchial mucosa;
• hypovolemia, blood clots;
• hypoxia and hypercapnia;
• sub-metabolic acidosis or decompensated

Treatment. Oxygen therapy. Conducted inhalation of humidified oxygen by nasal catheter or through a mask at a rate of 1-2 l / min. According to the testimony of oxygen flow rate may be increased to 5.3 l / min., Which corresponds to 30-40% of its concentration in the inspired air. Further increase of the concentration in the inspired air is impractical because giperoksigenatsiya can cause depression of the respiratory center.

Medicines.
Corticosteroids. The effect of corticosteroid therapy appears to treat acute airway inflammation and increased sensitivity to beta-adrenergic agents. The more severe asthma status, the greater the indication for immediate treatment with corticosteroids. Attention is drawn to the need for the original high-dose intravenous corticosteroids. Prednisolone, methylprednisolone, the initial dose - 2 mg / kg, maintenance - 0.5-1 mg / kg every 6 hours / in; or equivalent doses of other drugs. If therapy is not effective, the dose increases. Status asthmaticus docked less quantity per dose as the duration of treatment. Not be used to attack the uncropped low dose of hormones. After removing the patient from the asthmatic condition of corticosteroids gradually reduced - by about 25% in each subsequent day. Intravenous administration of an oral substitute. Aminophylline (2.4% solution) - contains 80% theophylline and 20% ethylenediamine, inhibits phosphodiesterase, which contributes to the accumulation of cAMP and removal of bronchospasm. Assigned to the initial dose of 5-6 mg / kg (loading dose) and weight of the patient is injected slowly / drip for 20 minutes. With the rapid introduction of the drug may cause hypotension. "Load" doses administered only if, within the last 24 hours, preparations containing theophylline, not used or used only in subtherapeutic doses. Subsequent appointment of aminophylline is based 1mg/1kg/1chas to clinical improvement. Keep in mind that the highest daily dose of aminophylline is 2r. Aminophylline overdose include nausea, vomiting, diarrhea, tachycardia, tachyarrhythmia, drowsiness, agitation, and seizures. The use of aminophylline in the treatment of status asthmaticus is also due to its positive effect on the beta-adrenergic receptors and indirect effects on the impaired energy cells.
Inhaled beta2-agonist therapy. Some patients with inhalation therapy is contraindicated sympathomimetics. It is known that the development of asthma status promotes the uncontrolled use of inhaled sympathomimetics. Observed in this and the well known syndrome of "rebound" is characterized by a progressive worsening asthma attacks because of the accumulated metabolites bronchoconstrictor sympathomimetics. Without denying the whole, this type of

therapy, it should be emphasized that the connection of inhaled beta2-agonists appropriate conduct at the reduced sensitivity to them. For this purpose, more appropriate drugs acting selectively on the beta 2-adrenergic receptors, such alupent (ortsiprenalin, astmopent) at an initial dose of 0.75 mg.

Intravenous therapy beta2-agonists. This type of treatment has significant limitations, is contraindicated in cases of heart (coronarocardiosclerosis, myocardial infarction), severe tachycardia, tachyphylaxis and in old age. Complications - arrhythmias and acute myocardial infarction - are the result of increased demand for oxygen, dissatisfied with status asthmaticus. Intravenous administration of beta-agonists may be used only if the therapy were not an option. Ensure accurate dosage difficult, this is done using infusion pumps. The rate of introduction alupenta 0.1 mcg / kg per minute until tachycardia (heart rate 130 per minute, or a few more). Need to increase the supply of oxygen, cardiomonitoring conduct surveillance and control the content of gases in the blood. Adrenaline stimulates alpha1-, beta1-and beta2-adrenergic receptors, increases and decreases bronchial airway resistance. Perhaps the use of "testing dose" of adrenaline in the early treatment of status asthmaticus maximum of 0.3-0.4 mg subcutaneously.
Intravenous administration of epinephrine at the rate of 0.1-1 mcg / kg / min continuous infusion is indicated in the form of anaphylactic asthma status.

Infusion therapy. This method is the most important component of the treatment of status asthmaticus. Treatment aims to fill the gap and eliminate fluid hypovolemia. The total volume of fluid therapy from 3 to 5 liters per day. By adding a hydration solution containing sufficient amount of free water (mainly of glucose), and hypo-and isotonic electrolyte solution containing sodium and chlorine. Keep in mind that the treatment with corticosteroids increases the body's need for potassium. To correct hypovolemia sometimes use dextrans, mainly rheological action (reopolyglukin) HES. Drugs that affect blood clotting, are shown in status asthmaticus for thromboembolic complications and improve the properties of blood. Typically used heparin solution of 2.5 thousand units per 0.5 liters of intravenous fluid, low molecular weight heparins. For the correction of metabolic acidosis (pH below 7.25) appropriate use of small doses of bicarbonate, which helps to improve the drainage function of the bronchi. We can not allow passage of metabolic acidosis in alkalosis. Indicators of adequate therapy - cessation of craving, wet tongue, restoring normal urine output, improve evacuation of phlegm, lower hematocrit to 0.30-0.40. They should not be infused HPC solutions at more than 12 cm of water column
In the treatment of severe asthma attack that is resistant to conventional treatment, can be used ftorotanom anesthesia. Antibiotics - their purpose in asthma status is shown in the presence of a bronchial infection recommended drugs with respect to the lower body sensitizing activity: erythromycin, azithromycin (sumamed) and other macrolides. VIVL shows the progression of hypercapnia and hypoxemia on a background of oxygen therapy, and in patients with severe respiratory muscle fatigue. She performed with the help of facial or nasal masks stored in the patient's spontaneous breathing mode of PEEP / CPAP. Indications for transfer of patients with

status asthmaticus on mechanical ventilation have to be very strict, as this procedure often causes complications. Indications for mechanical ventilation: the progression of asthma, despite intensive therapy (auscultation increases the area of "silent" areas, sometimes absent breath sounds in both lungs, heart rate increased significantly (up to 160 per minute), PaCO2> 60 mm Hg, and RaO2 <50 mm Hg), the progression of symptoms indicating the functional disorders of CNS, increasing fatigue and exhaustion, coma. Establish a sufficiently large tidal volume (TV), with a relatively small respiratory rate (RR - 8-16 in min.). Since expiratory prolonged status asthmaticus, regulate the ratio of phases inhalation / exhalation within 1:2-1:4, while observing for chest and VU meters and indicators. Necessary to verify the effectiveness of not only inspiration, but also the exhalation. Ventilator shall be performed with PEEP normoventilyatsii under constant blood gas and CBS. Intensive management of nosocomial pneumonia. Hospital (nosocomial) pneumonia - pneumonia that occurred after 48 hours of hospital. In patients on mechanical ventilation (ventilator-associated pneumonia (VAP)) - a type of hospital-acquired pneumonia.

The basis of treatment for the treatment of acute respiratory failure is the dynamic monitoring of respiratory parameters, blood gas and acid-base status. These data should be compared with the parameters of oxygen transport function of the cardiovascular system and other organs. Includes:
1. Diagnostic tests (clarification nosological diagnosis, assessment of severity, identification of agent);
2. Emergency start adequate antibiotic therapy (ABT) - after taking the material for analysis. Choosing ABT conducted taking into account the most likely causative agent, the weight of the patient, duration of hospitalization and / or mechanical ventilation to pneumonia earlier ABT, pharmacokinetics and pharmacodynamics of antibiotics, the range of their activity; microbiological evaluation of biological substrates. If the pathogen is known, the causal treatment is carried out in case of an unspecified agent conducted empirical ABT. Empirical therapy in the general pathogens most often Strep.pneumonii, St. aureus (MR), Enterobakter H. influenza. When mild current, with no prior ATB antibiotics 1st line are cefotaxime or ceftriaxone tsefaperazon / sulbactam (sitizon), ciprofloxacin, ofloxacin, pefloxacin, levofloxacin (at the risk of asiratsii added clindamycin or metronidazole), an antibiotic of the 2nd series - cefepime. In severe antibiotics 1st line are cefepime, ciprofloxacin, levofloxacin, antibiotics 2nd row - imipinem, Meronem. In resuscitation agents are most often P. aeroginosa, Enterobakter, Acenetobacter. At moderate flow antibiotics 1st row are: ceftazidime (at the risk of aspiration add amikacin), cefepime, tsefaperazon / sulbactam, ciprofloxacin, levofloxacin, antibiotics 2nd row - imipenem, Meron (at the risk of MR-selection of strains - vancomycin or linezolid) . In severe antibiotics 1st line are imipinem, Meronem, cefepime, antibiotics 2nd row - imipinem, Meron (at the risk of MR-selection of strains - vancomycin or linezolid, adjusting for selected flora). With the ineffectiveness of previous antibiotic therapy or confirmation of fungal infections: amphotericin B or Diflucan.
Severe community-acquired pneumonia: ceftriaxone (intramuscular or intravenous injection of 1.0 - 2.0 g 1-2 p / day.) Or cefotaxime (w / m or / in the 1.0 - 2.0 g 2 p / day.) in combination

with azithromycin (inside 0.25 -0.5 g 1 p / day.), levofloxacin (w / w to 0.5 g 1 p / day.) imepenem or meropenem (1.0 g intravenously 8 hours), ciprofloxacin (intravenous 0.4 - 0.6 g in 12 hours), in the future - possibly on the basis of bacteriological control.

3. Additional activities (pathogenetic and symptomatic treatment, prevention and treatment of sepsis and / or organ dysfunction) Bronchodilator and mucolytic therapy is done when indicated. To ensure adequate ventilation and oxygenation of the blood.Depending on the degree of hypoxemia:

• inhalation of oxygen

• when FiO2 more or equal to 0.5 is not correcting hypoxia - ventilation with constant high pressure (CPAP / CPAP) by face mask

• endotracheal intubation and mechanical ventilation, if these measures are not effective. Ventilator pneumonia preferably carried out with the help of modern machines, provides a mode to control the pressure to maintain optimal lung compliance. In the application of mechanical ventilation have to use antibacterial filters, carry adequate hydration breathing gas. Infusion therapy. Adequate fluid balance with maintaining tissue perfusion; correction water-electrolyte disorders. Immunemodulation shown in immunecompromised patients, and sepsis. Use immune replacement therapy - intravenous immunoglobulin.

General activities:

• Frequent changes in body position, elevated position of the head and chest;

• Physical therapy to the chest;

• Frequent deep breaths and cough.

In circumstances in which violated the act of swallowing and consciousness intragastric gavage completely balanced diet provides the energy needs of the body and the plastic at high energy. Intensive therapy with aspiration syndrome. The classical description of exudative pneumonitis aspiration of acid was Mendelssohn in 1946, however, the prevention, diagnosis and treatment of the syndrome is still valid. The reason - the extremely high mortality rate (40-50% or more). Predisposing factors: regurgitation or aspiration of gastric contents is possible with impaired consciousness (anesthesia, intoxication, the effect of sedatives, coma). Promotes high aspiration and intra intragastric pressure, such as increasing the volume of the stomach (birth, ileus), toning the abdominal wall during the introductory and basic anesthesia. To regurgitation and aspiration predisposes weakness gastroesophageal sphincter in patients suffering from long-term gastric ulcer and duodenal ulcer with high acidity and other diseases of the digestive tract. Etiology and pathogenesis: aspiration of gastric contents is

• chemical burn of the airways and alveoli from exposure to HCl (actually Mendelson's syndrome);
• airway obstruction vomit.

In some cases, dominated the first type of violation, in others - the second. With massive aspiration of gastric contents rapidly evolving asphyxial syndrome. In this case, the principal value is a factor of mechanical obstruction of the trachea, bronchi and bronchioles. If the pH of the aspirated fluid is low, except possibly obstruction chemical damage. For the emergence of a true syndrome Mendelssohn enough inhalation of 20-30ml of gastric juice, which has a low pH. Chemical burns of the mucous membranes of the respiratory tract accompanied by damage to the epithelium of the trachea, bronchi, bronchioles, alveolar walls and pulmonary capillary endothelium. The extent of damaging action is directly related to the acidity and the amount of aspirated gastric juice. The result is an acid burn plasma extravasation of blood into the pulmonary interstitium and in the alveolar cavity, resulting in the development of pulmonary edema and respiratory distress syndrome. Rapidly increasing edema mucosal and submucosal layers creates a bronchial obstruction, manifested total bronhiolospasm and hypoxemic respiratory failure.

Destructive changes in the lungs not only occur under the influence of gastric juice with pH 2.5 - 5, 0, and also in contact with bile, gastric enzymes and other fluids. Aspiration of infected oropharyngeal content also leads to pulmonita, which mainly affects the lower lobes of the lungs. This leads to the formation of necrotic cavities, abscesses and empyema. The same result is observed in the aspiration of small pieces of food, obturating bronchial lumen and causing the formation of atelectasis. The clinical picture is characterized by the acute onset (after aspiration or after 2-12h), a growing concern the patient, clear signs of respiratory disorders - laryngospasm or bronchospasm, dyspnea expiratory by type asthmatic condition. Typical is the following triad of symptoms: tachycardia, tachypnea, cyanosis. Aspiration often accompanied by a decrease in blood pressure and other reflex disorders of the cardiovascular system. Cyanosis does not disappear even with oxygen 100% concentration. Over the entire surface of the lungs auscultated whistling, and in the lower divisions - crepitusing wheezing. With the progression of respiratory disorders PaO2 decreased to 35-45 mm Hg, increased pulmonary vascular resistance and pulmonary artery pressure. Decreased lung compliance and increased air resistance of the airways. Further changes in the lungs are the type of respiratory distress syndrome (ARDS). Fairly typical X-ray picture - it looks like a "shock lung": there are sites reduce air - "snow storm", diffuse lung tissue shadowing. Sometimes the syndrome occurs more favorably. Possible aspiration fluid having a moderately acidic or neutral reaction. In cases where the aspiration was small, it is limited to some areas of the lung damage with clinical pneumonia. Treatment: the most important task of intensive care - to ensure adequate ventilation and oxygenation of the blood. If you suspect aregurgitation:

• stop the flow of gastric contents into the oropharynx: the patient must quickly make a draining and lower the head end, apply the method Sellick

• immediately aspirate the contents of the mouth,

• intubation, the presence of the inflatable cuff on the tube protects the airway from re-entering them vomit

• aspiration of the trachea and bronchi.

• If there is food debris, gastric or intestinal contents - remedial bronchoscopy with lavage. It is desirable to preserve cough shock, in which the emptying of the bronchi more efficiently. Required vibratory chest. In the absence of effective spontaneous breathing ventilator shown 100% oxygen concentration. The monitoring of respiratory and cardiovascular systems. Under the control PaO2 reduce FiO2 to 50%. If this restored adequate spontaneous respiration, extubated. Any failure of the patient to maintain adequate gas exchange is carried out prolonged mechanical ventilation. The preferred mode of ventilation on pressure. In the application of mechanical ventilation have to use antibacterial filters. To combat bronchospasm and shock intravenous steroids (eg, dexamethasone or tseleston (4-8mg)), aminophylline (10 ml of 2.4% solution), epinephrine (0.3 ml of 0.1% solution), antihistamines (diphenhydramine - 30 mg or suprastin - 20-40 mg). The shortfall in blood volume, correction of fluid and electrolyte disorders. Spend infusion of isotonic electrolyte and colloid solutions, fresh frozen plasma (400 ml) and albumin (100 mL of 20% solution), 20% glucose solution (100-200 ml). Improvement of the rheological properties of blood, prevention of DIC (heparin, low molecular weight heparin). With low blood pressure: dopamine - 10-15 mg / kg per minute.
Starting antibiotic therapy should include agents that are effective against gram-negative and anaerobic flora:
• Cephalosporins II-III generation (cefoxitin / m or / of 2.0 g every 6 hours or cefotaxime / m or in / on 3.0 g 8-12 hours) in combination with aminoglycosides (amikotsin in / m or / in 0.5 grams of 8-12 hours). IV-generation cephalosporin (cefepime in / on 1.0 -2.0 g in 12 hours) in combination with aminoglycosides

• fluoroquinolones in combination with macrolides (eg, ciprofloxacin in / on 0.2-0.4 g every 12 hours, and erythromycin in / on 1.0 g every 6 hours) or with aminoglycosides

• carbapenems as monotherapy or in combination with aminoglycosides
• Additionally, drugs active against anaerobic flora (metronidazole or clindamycin)
• When methicillin-resistant staphylococcus flora-glycopeptides (vancomycin)

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epinephrine (0.3 ml of 0.1% solution), antihistamines (diphenhydramine - 30 mg or suprastin - 20-40 mg). The shortfall in blood volume, correction of fluid and electrolyte disorders. Spend infusion of isotonic electrolyte and colloid solutions, fresh frozen plasma (400 ml) and albumin (100 mL of 20% solution), 20% glucose solution (100-200 ml). Improvement of the rheological properties of blood, prevention of DIC (heparin, low molecular weight heparin). With low blood pressure: dopamine - 10-15 mg / kg per minute. Starting antibiotic therapy should include agents that are effective against gram-negative and anaerobic flora:

• Cephalosporins II-III generation (cefoxitin / m or / of 2.0 g every 6 hours or cefotaxime / m or in / on 3.0 g 8-12 hours) in combination with aminoglycosides (amikotsin in / m or / in 0.5 grams of 8-12 hours). IV-generation cephalosporin (cefepime in / on 1.0 -2.0 g in 12 hours) in combination with aminoglycosides

• fluoroquinolones in combination with macrolides (eg, ciprofloxacin in / on 0.2-0.4 g every 12 hours, and erythromycin in / on 1.0 g every 6 hours) or with aminoglycosides

• carbapenems as monotherapy or in combination with aminoglycosides
• Additionally, drugs active against anaerobic flora (metronidazole or clindamycin)
• When methicillin-resistant staphylococcus flora-glycopeptides (vancomycin)

Respiratory distress syndrome (ARDS). Respiratory distress syndrome (ARDS) (synonyms: shock lung, hyaline membrane disease of adults) is a severe form of noncardiogenic pulmonary edema and pulmonary manifestations of a systemic inflammatory response syndrome. ARDS can occur in acute lung injury interfaced to various causes. The main factors of ARDS:

• Pathophysiology. Is a key part of the pathogenesis of damage alveolocapillary membrane. Whatever the reason for the reaction of the lungs rather stereotyped. This response includes the release of a large number of mediators, complement activation, coagulation, fibrinolysis and kinin cascade. First released by tumor necrosis factor, interleukins 1 and 6, a factor that activates platelets, and various prostaglandins and leukotrienes. The subsequent activation of neutrophils and macrophages in the lung parenchyma exposes the pulmonary effects of free radicals and proteases. The released mediators increase the permeability of lung capillaries, alter pulmonary vascular reactivity, inhibiting an important mechanism - hypoxic pulmonary vasoconstriction. Intensively destroyed alveolocytes I and II. Due to increased permeability of the membrane or destruction a alveolocapillary amount of extravascular lung water increased. As the leakage of albumin in the interstitial space of the protective effect of plasma oncotic pressure becomes insolvent. Opposes the normal force vanishes, and even a reduced hydrostatic pressure in the pulmonary capillaries, leading to extravasation of fluid into the tissue of the lungs. Accumulation of fluid in the alveoli, along with the violation of Education surfactant leads to the collapse of the alveoli, mikroatelectasing. Hyaline membranes begin to form the second day of the acute onset of the disease, and their presence is the most prominent morphological feature of exudative phase of 4-5 hours. These eosinophilic, hyaline (transparent) "membrane" composed of plasma

proteins, remnants of cytoplasm and nuclei with exfoliated epithelial cells. Exudative phase of ARDS can be resolved quickly or be delayed for an indefinite period of time, for it is often followed by the phase of fibrosis (fibrosing alveolitis) with complete cessation of gas exchange, which leads to irreversible scarring of the lungs.

Clinic and diagnostics. The main diagnostic criteria for ARDS, adopted at the American-European Consensus Conference on ARDS in 1994, include the following:

• acute onset;

• bilateral infiltrates on frontal chest radiograph;

• Violation of blood oxygenation in the lungs - PaO2/FiO2 <300 mm Hg. Art. for acute pulmonary damage (LFCC) and <200 mm Hg. Art. for ARDS;

• No left ventricular failure (pulmonary capillary wedge pressure <18 mm Hg. Art. leftatriumal hypertension or not). The main clinical signs of respiratory distress syndrome of adults demonstrate increasing dyspnea and hypoxemia with increased pressure on the breath, progressive infiltration of the lungs, measured by X-ray, and arterial hypotension. They are identified in 12-24 hours after exposure to the etiologic factor. More rarely, it is developing pulmonary complications in the early hours (aspiration of stomach acid), but at least - after 2-3 days after exposure to damaging factors for lung patients. ARDS may divide into four stages:

Stage 1. The patient's condition is often regarded as moderate. Often there are euphoria, reduced ability for critical evaluation of the condition due to cerebral hypoxia. Pale skin, sometimes with a touch of gray and earthy. Auscultation - hard breathing, wheezing, dry scattered in small numbers. Tachycardia. Blood pressure is usually adequate BCC, the introduction of vasopressor drugs and relatively often tends to increase. In the analysis of blood gases revealed progressive arterial hypoxemia refractory to oxygen therapy. According to X-ray of the chest, lung injury if not, the 1st stage ARDS is characterized by low-intensity spotty darkened diameter of 0.3 cm with indistinct outlines the periphery of light amid a moderate decline in lung tissue transparency and a significant increase in lung markings.

Stage 2. The patient's condition is regarded as serious. Patients drowsy or agitated. Against the background of the inspiratory apnea in some patients is determined by cyanosis of the skin, mucous membranes. Auscultated respiratory depression, and low back parts of the lungs - a few moist rales. Blood pressure in most cases tends to increase, there is a persistent tachycardia. If there is no attendant, hemodynamically significant, disorders of the heart, the 2nd stage is characterized by the reaction of hyperdynamic circulation with high cardiac output. In this case, there is a significant change in pulmonary hemodynamics - increase in pulmonary artery pressure and pulmonary capillaries, which leads to increased work of the right ventricle. Some patients appear pastoznost lower extremities. Reduced oxygen tension in the arterial blood against the remaining hypocapnia. X-ray picture of the 2nd stage of ARDS is characterized by increased

small focal spot around the lung field with a predominance in the periphery to reduce the transparency of the background of lung tissue. These changes are more pronounced than in the 1st stage.

The third stage. In the third stage of ARDS condition of the victims, as a rule, is assessed as extremely serious. Notes further confusion, accompanied by a massive agitation, delirium. Patients in monosyllables, with an effort to answer questions, the mouth is always open. Prominent part of the nose in breathing, lack of coordination of movements appear intercostal muscle twitching trachea, breathing begins to participate supporting musculature, increased inspiratory dyspnea. Tachypnea reaches 40 or more breaths per minute. Auscultation - hard breathing, the vast area of the bronchial. Over the entire surface of the light scattered dry rales are heard, and in the posterior-lower divisions - wet. Even if it contains 80% oxygen in the inspired gas mixture ($FiO_2 = 0,8$) arterial hypoxemia is below 55 mm Hg. Art. Blood pressure and central hemodynamics vary widely. This is often due to the actions of intensive therapy. There are signs of peripheral circulatory disorders: cold snap and pale cyanosis of the skin, particularly the fingertips of limbs, ears and nose. Frequent swelling of the feet and hands against decrease in protein content in the plasma. There are increasing signs or kidney and liver failure, metabolic disorders is increasing. Radiographic signs third stage ARDS characterized by further decreasing the transparency of light, mainly in the previously dilated vessels. Mid focal get a lot of shadows in the form of flakes of various sizes.

4th stage. At this stage of the condition of patients characterized as extremely serious. Patients are on mechanical ventilation. Confused consciousness, coma progresses. Marked hypotension and tachycardia limit. Cardiac output was sharply reduced and maintained by continuous infusion of inotropic agents. Patients with increasing signs of peripheral circulatory failure and multiple organ: liver-kidney failure, protein metabolism, fluid and electrolyte balance and other breathing can clearly hear only peredneverhnih departments. From the lower corner or from the middle of the shoulder blade and barely breathing down to listen, but a lot of mixed wet and dry rales. The partial oxygen pressure in the arterial blood, despite the increase in the concentration of inspired oxygen, is critical (50 mm Hg. Cent.), $PaCO_2$ is more dependent on the mode of mechanical ventilation. When X-ray there is a progressive darkening of the lung fields as a result of alveolar edema. Against excessive density of lung tissue, or "Blizzard" is very difficult to detect signs of pneumonia and lung abscess formation complicating usually late stage ARDS. Sometimes visible areas of enlightenment due to the development of fibrosis. Laboratory and functional methods.

• reduced almost all lung volume: respiratory, functional residual capacity, vital capacity (VC), etc. In stage 3 VC dramatically decreases and reaches 10-15% of predicted
• reduced distensibility of lung tissue - a measure of elastic recoil, which is denoted in the physiology of the letter C (English «compliance» - stretch). On lung compliance is influenced by such factors as the amount of lung tissue, the amount of blood in the pulmonary circulation, the

amount of extravascular fluid in the lungs, as well as inflammation and fibrosis. When ARDS C <50ml/sm wg (N adults 50-150ml/sm waters. Cent.)

• Decrease in SpO2 (SpO2 = 90-94% is regarded as a moderate arterial hypoxemia)
• PaO 2 of 50 mm Hg. Art., with FiO2 = 60% (PaO2/FiO2 <250)

• Ret low CO2 (carbon dioxide tension in the exhaled air) and very high PaCO2. Indicate severe ARDS is increasing PaCO2 greater than 50 mm Hg. Art.

Treatment should be comprehensive and should include:
1. treatment of the underlying disease process that caused ARDS

2. maintain oxygenation and ventilation (respiratory support, rehabilitation respiratory tract)
3. an adequate fluid resuscitation

4. treatment of the effects of tissue hypoxia (multiple organ dysfunction / failure)

5. prevention and treatment of nosocomial infections

Respiratory support. Oxygen is shown in the first hours of the disease. The best indicator of its effectiveness are the results of the dynamic studies SrO2 and PaO2. At all stages of the treatment is important to prevent the toxic effect of oxygen. Using the lowest concentration of oxygen, try to maintain SaO2 above 90%. In mild cases, you can use the CPAP via a face mask. But most patients require intubation and respiratory support with the ventilator. Early intensive application of PEEP / CPAP can reduce the FIO2 to nontoxic levels. However, one can avoid high peak airway pressure because alveolar overdistension may exacerbate lung injury. Sometimes in severe refractory hypoxemia (including with hypercapnia) use mechanical ventilation to control the pressure and reverse ratio inhale / exhale, high frequency ventilation, extracorporeal membrane oxygenation. Additional methods. In severe ARDS can have a beneficial effect inhalation of nitric oxide, which leads to a decrease in pulmonary artery pressure and reduces intrapulmonary shunting due to a selective increase in the perfusion of ventilated areas of the lung. Successfully used in the aerosol mixtures of synthetic and natural surfactants. Steroids in most clinics in the world is used for severe ARDS when other therapy within the first 2 weeks is not effective (not to use with sepsis). Can prednisolone at a dose of 2 mg / kg / day for 1-2 weeks. Infusion-transfusion therapy - the most important and integral part of intensive treatment ARDS. One of the major pathogenetic factors ARDS is a permanent impairment of microcirculation, followed by cell and organ dysfunction. Consequence of hypovolemia and low perfusion pressure is decreased blood flow and worsening tissue hypoxia. Infusion therapy is aimed at correcting water, electrolyte and acid-base, as well as the energy and plastic balance. Selecting media for infusion therapy is largely determined by the nature of the primary disease process, disorders of homeostasis. For adequate O2 transport to the tissues to maintain sufficient levels of hemoglobin (Hb not <100 g / L). Of plasma-solutions preferred solutions, hydroxyethyl starch (HES), as along with the normalization of plasma oncotic

pressure, HES molecules can close the pores in the walls of the capillaries and reduce the damage associated with the high permeability of the endothelium. We can not allow a large blood clots, not maintaining hematocrit> 0.4. Anticoagulants improves not only pulmonary tissue blood flow, but the quality of the blood flowing to the lungs. Recommended to assign direct anticoagulants: heparin, low molecular weight heparins.
For conducting fluid therapy should be closely monitored with appropriate hemodynamic monitoring, as ARDS at extremely high probability of hydration, alveolar edema, progression of lung and heart failure. If necessary use inotropic support, is the first choice drug dopamine, adrenaline and norepinefrine. The complex treatment of patients with severe multiorgan dysfunction is imperative to include nutritional support. The choice of method of nutritional support depends on the degree of nutrient deficiency and functional state of the gastrointestinal tract.

Self-study
Task number one
Examine the patient with respiratory pathology (ARDS, asthma):
• Perform physical examination (blood pressure, heart rate, heart rate, respiratory rate and depth, tenderness);
• Identify the respiratory patient parameters;
• Select the method of treatment and further tactics of the patient, give reasons for your choice.

Task number two
Analyze the history of a patient with severe pneumonia, is in the intensive care unit and intensive care. To do this:
• analyze the amount of emergency assistance;
• appreciate the severity of respiratory failure in a patient based on the diaries of follow-up;
• evaluate the proposed method of treatment of respiratory failure, if necessary, make the corrections and justify them.

IX. Clinical problems
Objective number one
During the meal, the patient 60 years suddenly there was a pain in the throat, worse when swallowing, the feeling of a foreign body in the throat, difficulty in swallowing food, salivation, coughing, shortness of breath and sharp.
Explain the reason for the patient develop a condition that is necessary to make the diagnosis? What care should a patient have?
Objective number two
5 years old girl taken to the emergency room at Children's Hospital ambulance crews. From history we found out that two days ago, there were signs of SARS. Today at 2:00 am sharp child started to choke, there was a barking cough. In the study of 130 notes per minute heart rate, t

body 37,6 ˚ C.
Explain the reason for the patient develop a condition with which diseases should be a differential diagnosis.
What kind of medical care to provide pre-hospital patient?

Test control:
1. Normal levels of O2 and decreased arteriovenous difference observed in:
a) Violation of the airway.
b) hemorrhagic shock.
c) Cyanide poisoning.
d) Sickle-cell disease.
2. For which of the following has the highest hypoxia arteriovenous oxygen difference:
a) hypoxic.
b) stagnant.
c) anemia.
d) Gistotoksichesky.
3. Cyanosis can be seen when the saturation of arterial blood falls below 02:
a) 60%
b) 70%
a) 80%
d) 90%
4. The most serious consequence of long-term course of asthma is:
a) Extension of the bronchi.
b) Sealing walls smallest bronchi.
a) Lots of atelectasis.
d) filling the large bronchial mucus and pus.
5. Asthma attack is accompanied by:
a) 2-fold reduction in forced expiratory volume.
b) an increase in residual volume.
c) increase the frictional resistance.
d) increase the elasticity of the lungs.
6. Patients are able to threatening asthma attack symptoms are:
a) Obstruction of the bronchioles.
b) Cyanosis.
c) Increasing pCO2.
d) a pH below 7.4.
7. Bronchial spasm during anesthesia appears:
a) the duration of the forced exhalation.
b) the duration of a breath.
c) Weakened inhalation and exhalation weakened.

d) a short breath and prolonged exhalation.
8. What reasons can lead to the development of bronchospasm:
a) Neurogenic.
b) Chemical.
c) Mechanical.
d) pulmonary embolism.
9. The surest sign of oxygen deficiency are:
a) Hypertension.
b) Tachycardia.
c) Hypertension.
g) In-depth breath.
10. The following examples lead to a decrease in the oxygen content in the blood, except for:
a) Alveolar O2 concentration of 16%.
b) CO poisoning.
c) continuously for 60 s aspiration of the tracheobronchial tree.
d) The transition to air breathing after prolonged anesthesia N2O: O2.

Answers:
Objective number one
Diagnosis: Foreign body throat. Diagnosis can be made on the basis of medical history, physical examination of the pharynx, palpation, radiography.
Emergency care: to try to remove the foreign body with your finger, use a technique Heymliha, failing with a deterioration of the patient is necessary Konik-or tracheostomy.
Removing small foreign body in the throat should be made ENT office (desk). After contact with a small foreign body in the bronchi - bronchoscopic lavage with sanitation

Objective number two
Diagnosis: obstructive laryngotracheobronchitis. (Diffferentsirovat with an attack of asthma, retropharyngeal abscess).
Treatment:
• Foot baths with mustard (at body temperature no higher than 37,5 ± C);
• Mustard (up to 3-4 times a day);
• Warm alkaline water (milk with sodium bicarbonate or mineral water).
• Alkaline (2 teaspoons of sodium bicarbonate in 1 liter of water) and steam inhalations every 3 hours
The appearance of a soft cough demonstrates the effectiveness of the procedure.
Medications:
• Prednisolone - 1-5 mg / kg per day or hydrocortisone - 35 mg / kg, depending on the severity.
• pipolfen (Promethazine) 0,008-0,01 g reception children up to 6 years and 0,012-0,015 g for children older than 6 years 2-4 times a day, or intramuscular 0.5-1 ml of 2.5% solution;

• diphenhydramine 1 mL of 1% solution intramuscularly to children aged 3-9 years or 2% 0.5 ml suprastina children 5-6 years;

Test control:

1. at 6. a, b, c, d
2. b 7. a, d
3. at 8. a, b, c, d
4. b 9. b
5. a, b, c 10. and