MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty *Medicine*
Department *Surgery, Radiological Diagnostics, Radiation Medicine, Therapy and Oncology*

APPROVED BY
Vice-Rector for Scientific and Pedagogical Work
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METHODOLOGICAL RECOMMENDATION FOR PRACTICAL CLASSES OF THE ACADEMIC DISCIPLINE

Faculty, course Medical 6th year

Academic discipline Surgery

(name of the discipline)

PRACTICAL CLASSES

Practical class № 3

Topic: "Critical conditions in surgical patients: acute respiratory distress syndrome, abdominal cavity syndrome, collapse"

Approved:

At the meeting of the Department of Surgery, Radiation Diagnostics, Radiation Medicine, Therapy and Oncology of Odesa National Medical University

Odesa National Medical University

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PRACTICAL CLASSES

Practical class № 3

Topic of the practical class: 'CRITICAL CONDITIONS IN SURGICAL PATIENTS: ACUTE RESPIRATORY DISTRESS SYNDROME, COMA, ABDOMINAL CAVITY SYNDROME' - 4 HOURS.

1. Relevance of the topic. Critical conditions in surgical patients. Acute respiratory distress syndrome, coma, abdominal syndrome - an extremely important problem, since their development leads to a direct threat to the patient's life.

There is a need to determine critical conditions in surgical patients: acute respiratory distress syndrome, abdominal syndrome, collapse from the point of view of a post-syndrome approach, since this makes it possible to conduct diagnostics at the level of a general practitioner and provides for the application of the correct and timely algorithm of actions in these pathological conditions.

2. OBJECTIVES OF THE LESSON:

2.1. **GENERAL LEARNING OBJECTIVES OF THE LESSON**: To familiarize higher education students with critical conditions in surgical patients: acute respiratory distress syndrome, abdominal cavity syndrome, collapse in surgical practice, with the algorithm of diagnosis, differential diagnosis and treatment for these pathological conditions:

2.2. SPECIFIC OBJECTIVES OF THE LESSON:

1.1. To know the etiology and pathology of critical conditions in surgical patients: acute respiratory distress syndrome, coma, abdominal syndrome.

2.1. To know the morphological changes in organs and tissues in critical conditions of surgical patients: acute respiratory distress syndrome, coma, abdominal syndrome.

3.2. To master the clinical picture in critical conditions of surgical patients: acute respiratory distress syndrome, coma, abdominal syndrome.

4.2. To master laboratory methods of blood testing in critical conditions of surgical patients: acute respiratory distress syndrome, coma, abdominal syndrome.

5.2. To master the principles of treatment of critical conditions in surgical patients: acute respiratory distress syndrome, coma, abdominal syndrome.

6.3. To learn to conduct clinical examinations, take complaints; to take anamnesis;

7.3. To learn to conduct differential diagnostics of critical conditions in surgical patients: acute respiratory distress syndrome, coma, abdominal syndrome.

8.3. To be able to interpret laboratory and X-ray data;

9.3. To be able to determine an individual treatment regimen in a specific clinical situation in a patient;

10.3 To learn to perform laparocentesis;

11.3. To master methods of abdominal drainage.

2.2. EDUCATIONAL OBJECTIVES

1. To form a deontological idea when working with surgical patients in critical conditions: acute respiratory distress syndrome, coma, abdominal cavity syndrome.

2. To develop an understanding of the influence of environmental factors, risk factors in critical conditions of surgical patients: acute respiratory distress syndrome, coma, abdominal syndrome.

3. Using the materials of this topic, to develop a sense of responsibility for the timeliness and correctness of professional actions.

4. To form an idea of the basis of a psychotherapeutic approach to surgical patients in critical conditions: acute respiratory distress syndrome, coma, abdominal syndrome.

5. To master the ability to establish psychological contact with the patient and his relatives.

3. INTERDISCIPLINARY INTEGRATION

Discipline	To know	To be able to
Normal anatomy and pathomorphology	Know the anatomical structure of body regions, morphological changes in conditions related to the topic being studied.	Use the data to prepare for class.
Normal and pathological physiology	To know the normal function of organs and systems, haemodynamic changes, changes in metabolic processes in systems and organs related to the topic, changes in other organs and systems, immune shifts in the body, the main mechanisms of etiology and pathogenesis of the studied conditions.	Use the data to prepare for class.
Pharmacology	The main drugs used in the treatment of critical conditions in surgical patients are: acute respiratory distress, coma, abdominal cavity syndrome.	Be able to write out prescriptions, write a list of medical appointments.
Propedeutics of internal medicine, topics of modules 1, 2, 3, 4 in internal medicine	Methods of physical examination in critical conditions in surgical patients: acute respiratory distress syndrome, coma, abdominal cavity syndrome.Instrumental and laboratory methods of research	To conduct a systemic examination of the body with the isolation of the main symptom complex in critical conditions in surgical patients: acute respiratory distress, coma, abdominal syndrome. Interpret the data of laboratory and instrumental studies.

General surgery, topics of modules 1, 2, 3, 4: surgical diseases with paediatric surgery and oncology	Methods of physical examination in critical conditions in surgical patients: acute respiratory distress, coma, abdominal cavity syndrome.Instrumental and laboratory methods of research	To conduct a systemic examination of the body with the selection of the main symptom complex in critical conditions in surgical patients: acute respiratory distress, coma, abdominal syndrome. Interpret the data of laboratory and instrumental studies.
Paediatrics propaedeutics, topics of modules 1, 2, 3, 4 in paediatrics	Methods of physical examination in critical conditions in surgical patients: acute respiratory distress, coma, abdominal cavity syndrome. Instrumental and laboratory research methods	To conduct a systemic study of the body with the selection of the main symptom complex in critical conditions in surgical patients: acute respiratory distress, coma, abdominal syndrome. Interpret the data of laboratory and instrumental studies.

Topics of modules 1, 2, 3, 4 in obstetrics and gynaecology	Methods of physical examination in critical conditions in surgical patients: acute respiratory distress syndrome, coma, abdominal cavity syndrome.Instrumental and laboratory methods of research	To conduct a systemic examination of the body with the selection of the main symptom complex in critical conditions in surgical patients: acute respiratory distress syndrome, coma, abdominal cavity syndrome. Interpret the data of laboratory and instrumental studies.

5. 1. CONTENT OF THE LESSON: - 2 hours.

Acute respiratory distress syndrome (ARDS) is an extremely severe form of respiratory failure that develops in stages in response to lung damage by exogenous or endogenous factors and is characterised by progressive non-cardiogenic pulmonary edema due to damage to the alveolar-capillary membranes, dyspnoea and hypoxaemia resistant to oxygen therapy.

Prevalence. ARDS causes more than 150,000 cases of respiratory failure per year in the United States. In 50 cases, it leads to death, which puts it on a par with lung cancer (about 100,000 deaths per year) as one of the most common causes of death in pulmonology today. At the same time, the mortality rate from ARDS worldwide has not decreased for 25 years and ranges from 50 to 75%.

Etiology. Among a large number of factors that can cause acute lung injury/ARDS under certain conditions, the most clinically significant are

Table 1.

Clinical conditions associated with the development of ARDS

Direct damage to the lungs	Indirect lung damage
Aspiration of acidic gastric contents	Pakreonecrosis
Severe thoracic trauma, lung contusion	Peritonitis
Diffuse lung infection: bacterial and viral	Long-term traumatic operations
Toxic inhalation and burns	Polytrauma
Drowning (especially in chlorinated pool water)	Sepsis
	Massive transfusions
	Fat, amniotic and bacterial emboli
	Disseminated intravascular coagulation syndrome
	Hypovolaemic shock
	Extracorporeal circulation

Table 2.

Frequency of ARDS in some clinical conditions

Risk factors	Probability of ARDS, %	Mortality rate, %.	
		with ARDS	with ARDS
Sepsis	41	69	50
Massive transfusions	36	70	35
Lung contusions	22	49	12
Aspiration	22	48	21
Polytrauma	11	49	9
Other causes	26	62	19

Pathogenesis. The mechanisms of ARDS development remain unclear. Experimental studies have shown that ARDS development occurs in three phases. **In the first phase**, activated leukocytes and platelets accumulate in the capillaries, interstitium and air spaces of the lungs, releasing prostaglandins, toxic oxygen radicals, proteolytic enzymes and other biologically active substances that secondary damage cells, stimulate fibrosis, change bronchomotor tone and vascular reactivity. Damage to the endothelium of the pulmonary capillaries and alveolar epithelium leads to blood plasma seepage into the interstitial and alveolar space and, ultimately, to filling the alveoli with fluid, their atelectasis, which is also facilitated by a decrease in pulmonary surfactant activity. **The second phase** develops in 2-3 days and is characterised by interstitial and bronchoalveolar inflammation, proliferation of epithelial and interstitial cells. The third phase is characterised by rapid collagen accumulation, which leads to severe interstitial fibrosis within 2-3 weeks. Such pathological changes cause, first of all, a decrease in lung elasticity, pulmonary hypertension, a decrease in functional residual capacity, a violation of the ventilation-perfusion ratio and, as a result, severe hypoxia, which is poorly corrected by oxygen therapy.

Clinic and diagnostics.

Criteria for ARDS: acute onset, radiological: 2-sided lung infiltrates; pulmonary artery wedge pressure £ 18 mmHg or no clinical signs of chronic heart failure; RaO2/FiO2 £ 200 mmHg. necessarily reduced pulmonary compliance.

The most informative criterion for ARDS is severe hypoxaemia (PaO2/FiO2 £ 200 mmHg) with acute onset, an etiological factor (although not necessarily), absence of cardiogenic pulmonary edema as the primary cause (with PAP > 18 mmHg) and typical radiological changes in the lungs.

Table 3.

Diagnostic criteria for acute lung injury syndrome and acute respiratory distress syndrome

Diagnostic criterion	ALISA *	ARDS
Acute onset	+	+
Diffuse bilateral pulmonary infiltrates on X-ray	+	+
Absence of clinical signs of congestive heart failure (pulmonary capillary wedge pressure < 18 mm Hg)	+	+
Reduced blood oxygenation ratio (PaO2/Fi O2)	<300 mmHg	<200 mmHg

Note: * - acute lung injury syndrome.

Clinical manifestations: within 1-2 days after the onset of the etiological factor, severe pulmonary insufficiency develops with progressive symptoms of hypoxaemia, dyspnoea with the participation of auxiliary muscles in breathing, various rales are heard in the lungs, the RaO2/FiO2 decreases, and shunting in the small circle of blood circulation increases. Patients often require the use of a ventilator, although in severe cases the latter is not able to ensure normal gas exchange in the lungs.

On a lung radiograph, symptoms may be delayed compared to the clinic, but then stagnation in the lung vessels, bilateral infiltrates appear, and in the final stages of ARDS they are confluent with foci of pneumosclerosis. Computed tomography confirms the presence of diffuse pulmonary infiltrates, dis- and microathelectasis, and a decrease in the volume of functioning lung parenchyma.

For an integrated assessment of the severity of the patient's condition, as well as predicting the course and outcome of ARDS, it is advisable to use evaluation scales. The most convenient is the lung injury scale proposed by J.F.Murray et al. in 1988 (Table 4). Thus, with a score of more than 2.5, the predicted mortality rate is 58-59%, 0.25-2.5 - 40-41% and less than 0.25 - about
36,4%.

Examined indicators	Points
Chest radiography	
no alveolar consolidation	0
alveolar consolidation in one quadrant of the lungs	1
alveolar consolidation in two quadrants of the lungs	2
alveolar consolidation in three quadrants of the lungs	3
alveolar consolidation in four quadrants of the lungs	4
Blood oxygenation ratio (PaO2/Fi O2)	
≥300	0
225-299	1
175-224	2

100-174	3
<100	4
Lung compliance (ml/cm Hg) during mechanical ventilation	
≥80	0
60-79	1
40-59	2
20-39	3
<20	4
Positive end-expiratory pressure (cm Hg) during mechanical ventilation	
<6	0
6-8	1
9-11	2
12-14	3
≥15	4
Total points	
No lung damage	0
Acute lung damage	0,1-2,5
Acute respiratory distress syndrome	>2,5

Diagnostic programme

Visual examination, determination of the etiological factor, assessment of the general condition of the patient, auscultation and percussion of the lungs.

1. Obligatory measurement of heart rate, blood pressure, central venous catheterisation (CVC) (if possible, pulmonary artery wedge pressure).

2. Laboratory examination:

a. measurement of arterial blood gases, BUN and lactate;

b. complete blood count and urinalysis;

c. coagulogram;

d. biochemical blood test;

e. ECG.

3. Chest radiography.

4. Fibrobrochoscopy with bacteriological analysis of the contents of the lower of the lower respiratory tract.

Treatment programme

1: Treatment of the underlying disease (peritonitis, pancreatic necrosis, etc.) is in the foreground.

2. Oxygen therapy and respiratory support for the patient: A protective ventilatory strategy has been proven to be effective, including:

respiratory volume = 6 - 7 ml/kg and positive end-expiratory pressure = 6 - 10 cm Hg;

plateau pressure

< 35 cmHg. RaCO2 can be maintained at a level that does not affect haemodynamics and consciousness of the patient - safe hypercapnia.

3. Active kinetotherapy: it is imperative to turn the patient onto the stomach (hemodynamic instability, as well as severe traumatic brain injury, spinal fractures, pelvic bones may be contraindicated). There are no recommendations on the mode of turning yet, but it is important to start kinetotherapy from the first days of mechanical ventilation, turn the patient at least 2 times per day for 4-6 hours, provided the patient is well tolerated, the time spent on the stomach may be longer. If it is not possible to

turn the patient on the stomach, it is mandatory to turn the patient on the sides with a change of body position at least every 2 hours. The 'restorative manoeuvre' can be used, which consists in periodically inflating the lungs for 40 to 45 seconds by increasing positive end-expiratory pressure or respiratory volume.

If it is impossible to maintain blood oxygenation at a safe oxygen concentration ($FiO2 < 0.6$), an inverted ventilator mode with an increase in the inhalation/exhalation ratio > 0.5 is possible.

5. Indications for the use of *extracorporeal membrane oxygenation*: $RaO2 < 50$ mmHg or $SaO2 \leq 85\text{-}90\%$ with $FiO2 = 1$ and PTCV ≥ 10 cmHg.

Partial liquid lung ventilation (PLV) - carry out with a liquid that tolerates oxygen and hydrogen carbon (perfluorocarbon compounds).

6. Intratracheal administration of artificial surfactant.

7. Anti-inflammatory therapy: glucocorticoids are not indicated in the first stages of ARDS and worsen the results of treatment, only in late ARDS (proliferative stage) small doses (methylprednisolone 2-3 mg/kg per day) are indicated; non-steroidal anti-inflammatory drugs are not indicated.

8. After recovery from shock, restrictive intravenous infusion regimen (minimum intravenous fluids), maintain fluid balance through enteral (gastric or intestinal) nutrition, provide adequate enteral nutrition with omega-3 polyunsaturated fatty acids (fish oil).

9. Antioxidants and antihypoxants.

10. Diuretics are not indicated (unless there are signs of hypervolaemia).

11. Antibiotic therapy based on bacteriological analysis of the respiratory tract and sensitivity of the microflora to them.

12. Inhalation of beta2-adrenoagonists.

Duration of treatment in the intensive care unit:

From 10 days to 1 to 2 months.

Treatment quality criteria

Transfer of patients to independent breathing, normalisation of $RaO2/FiO2$ (> 300 mmHg), $RaCO2$,% shunt in the lungs (< 10%) and other indicators of external respiration.

Possible side effects and complications

1. A common complication is ventilator-associated pneumonia, the most severe complication is asphyxia and death.

2. **Recommendations for further medical care**

a. Prevention of pneumonia;

b. Prevention of other infectious complications;

c. prevention of chronic lung failure.

5.2. CONTENT OF THE CLASS: - 2 hours.

COMA

'Coma' is the Greek word for deep sleep. According to the classical definition, this term refers to the most significant degree of pathological inhibition of the central nervous system (CNS), characterised by profound loss of consciousness, lack of reflexes to external stimuli and disruption of the regulation of vital body functions.

However, due to the significance of this diagnosis for practice, the life-threatening nature of this condition and the need for early treatment, coma is diagnosed in practice even in the case of less severe CNS depression, if it is considered as a stage of its development. Therefore, it is more appropriate to define coma as:a state of cerebral insufficiency characterised by impaired CNS coordinating activity, separation of the body into separate, autonomously functioning systems that lose their ability to self-regulate and maintain homeostasis at the level of the whole organism; clinically, coma is manifested by unconsciousness, impaired motor, sensory and somatic functions, including vital ones.

MAIN CAUSES AND PATHOGENESIS

Comatose states develop as a result of various causes that can be divided into four groups: a) intracranial processes (vascular, inflammatory, volumetric, etc.)

b) hypoxia states in somatic pathology (respiratory hypoxia in case of respiratory system damage, circulatory hypoxia in case of circulatory disorders, haemic hypoxia in case of haemoglobin pathology):

1. disorders of tissue respiration (tissue hypoxia)

2. drop in oxygen tension in the inhaled air (hypoxic hypoxia);

c) metabolic disorders (primarily of endocrine origin)

d) intoxication (both exo- and endogenous).

Despite the diversity of coma etiologies, there is much in common in their pathogenesis, and the factors that are the primary causes of some types of coma act as pathogenetic mechanisms in others. The immediate mechanism of cerebral insufficiency is the disruption of the emergence, propagation and transmission of nerve impulses in brain cells due to depression of tissue respiration, metabolism and energy. This occurs due to a reduction in the delivery of oxygen and nutrients to the brain tissue (ischaemia, venous stasis, microcirculatory disorders, vascular stasis, perivascular edema), changes in acid-base and electrolyte balance, increased intracranial pressure, swelling and edema of the brain and meninges. The latter can lead to brain dislocation with mechanical damage to the tissue of vital centres. At any stage of coma, tissue hypoxia of varying severity develops. Acid-base disturbances are most often metabolic acidosis; with primary damage to the respiratory system, respiratory acidosis develops. Less commonly, for example, metabolic alkalosis occurs with repeated vomiting, and hyperventilation leads to respiratory alkalosis. A combination of various metabolic and respiratory changes is characteristic

Among electrolyte disorders, the most significant are changes in potassium concentration (both hypo- and hyperkalaemia) and hyponatraemia. The latter plays an important role in the growth of cerebral edema. Progressive metabolic disorders have a histotoxic effect. As the coma deepens, respiratory and subsequently circulatory disorders develop.

CLASSIFICATION

Depending on the causative factors, ‘primary’ and ‘secondary’ comas are distinguished (see Table 1).

In order to assess the prognosis and choose treatment tactics, it is very important to determine what led to the development of a coma: focal brain damage with a mass effect, brainstem damage, or diffuse cortical and brainstem damage. The

first two variants are characteristic of primary comas, and the latter is almost exclusively found in secondary comas.

Loss of consciousness - stunning - can have different depths, depending on which it is divided into

- obnibulation - blurring, clouding, ‘cloudiness of consciousness’, stunning
- somnolence - drowsiness
- soporas - forgetfulness, insensitivity, pathological hibernation, deep stunning
- coma - the most profound degree of cerebral insufficiency.

As a rule, the diagnosis of ‘precoma’ is made instead of the first three variants. However, there are no pathogenetically sound (which to some extent can be attributed to the classifications of coma depth) and clearly defined clinical distinctions between the four degrees of stunning, and therefore, regardless of the degree of loss of consciousness, the term coma is acceptable, the depth of which can be assessed using a simple but informative clinical scale.

Table 1.

CLASSIFICATION OF COMAS DEPENDING ON THE CAUSES OF THEIR OCCURRENCE

A. Primary	B. Coma as a result of secondary damage to the central nervous system	
cerebral coma (‘brain coma’)	a) endogenous factors	b) external factors
cerebrovascular (as a result of ischaemic or haemorrhagic stroke, subarachnoid haemorrhage); epileptic; intracranial volumetric processes (tumours, echinococcosis, abscesses); in case of infectious lesions of brain tissue or meninges; traumatic.	in case of internal organ dysfunction (uremic, hepatic, hypoxia due to respiratory or circulatory system damage); diseases of the endocrine system (diabetic, hypothyroid and thyrotoxic, hypocorticoid, etc;) in neoplasms (hypoglycaemia in hormonally active pancreatic tumours or massive hormonally inactive malignant tumours); in other therapeutic, surgical, infectious and other diseases (malaria, pernicious anaemia	in case of relative or absolute overdose of hypoglycaemic agents (hypoglycaemic); in case of starvation (alimentary dystrophy): in case of intoxication (alcohol, opiate, barbiturate, poisoning with tranquillisers, methanol, carbon monoxide, etc;) in case of overheating (hyperthermia or ‘heat stroke’); in case of hypothermia; in case of electrical trauma etc.

DIAGNOSTIC CRITERIA

The diagnosis of comas is based on the identification

- a certain degree of depression of consciousness
- decreased sensitivity to external stimuli up to its complete loss
- Specific signs of certain types of coma (Table 3 in the section 'Clinical picture').

Differential diagnosis is carried out with pseudocomatose states (isolation syndrome, psychogenic reactivity, abulic status, nonconvulsive epileptic status).

CLINICAL PICTURE

Clinical manifestations, the rate of coma development, and anamnesis data are usually quite specific for different coma variants (Table 3).

CLINICAL SYMPTOMS OF DIFFERENT TYPES OF COMA

In addition to specific signs, signs of depression of consciousness and weakening of reflexes (tendon, periosteal, cutaneous and cranial nerves) play a significant and sometimes leading role in the clinical picture of coma, which progresses to complete extinction as coma deepens.

These signs have already been discussed in Table 2. The youngest reflexes are the first to disappear, and the oldest reflexes are the last. In the absence of focal brain lesions, deepening of coma is accompanied by the appearance and subsequent loss of bilateral pathological signs (Babinski reflex), while focal lesions are characterised by their unilateral nature. Meningeal signs - occipital muscle flushing, Kernig and Brudzinski symptoms, characteristic of meningitis and meningoencephalitis - also appear in case of cerebral edema and irritation of the meninges. The progression of cerebral insufficiency with the decline of functions leads to various respiratory disorders with hypo- or hyperventilation and corresponding respiratory shifts in acid-base status. Gross haemodynamic disorders usually join in the terminal state.

TREATMENT OF COMA

The treatment of coma consists of differentiated therapy of individual comatose states and general, universal measures that do not depend on the causes, pathogenesis and clinical manifestations.

A. UNDIFFERENTIATED THERAPY OF COMA

When providing first aid to a comatose patient, several goals are pursued. Measures to achieve the main ones are carried out simultaneously:

1. **Immediate hospitalisation to the intensive care unit, and in case of traumatic brain injury or subarachnoid haemorrhage - to the neurosurgical unit.**

Despite mandatory hospitalisation, emergency therapy for insects in all cases should be started immediately.

2. **Restoration (or maintenance) of an adequate state of vital functions:**

a) respiration

- airway rehabilitation to restore airway patency, installation of an airway or tongue fixation, artificial lung ventilation using a mask or intubation tube, in some cases - tracheal or conicotomy; oxygen therapy (4-6 l/min through a nasal catheter or 60% through a mask, intubation tube); tracheal intubation in all cases should be

preceded by premedication with 0.1% atropine solution in a dose of 0.5-1.0 ml (except for cholinolytic drug poisoning);

b) blood circulation

- in case of arterial hypertension - lowering the blood pressure to a level that exceeds the usual values by at least 15-20 mm Hg, and, in the absence of anamnestic data, not lower than 150-160/80-90 mmHg by reducing intracranial pressure (see below); administration of 5-10 ml of 25% magnesium sulfate solution (IV bolus over 7-10 minutes or drip), bolus administration of 3-4 ml of 1% solution (6-8 ml of 0.5% solution) of dibazole, and in case of a slight increase in CT, bolus administration of 5-10 ml of 2.4% eufiline solution (over 3-5 minutes) is sufficient;

- control of arterial hypotension is carried out in three stages:

- slow intravenous injection of dexamethasone at a dose of 8-20 milligrams or mazipredone (prednisone) at a dose of 60-150 milligrams
- If ineffective, dextran 70 (polyglucin) in a dose of 50-100 ml IV jet, then IV drip in a volume of up to 400-500 ml; coma in the setting of intoxication, excitosis and haemoconcentration is an indication for infusion of 1000-2000 ml of 0.9% sodium chloride solution or 5% glucose solution
- if ineffective, drip administration of Dopamine at a dose of 5-15 μg/kg/min or norepinephrine
- In case of arrhythmias, restore an adequate heart rhythm (see the lesson on rhythm disturbances).

3. **Immobilisation of the cervical spine in case of any suspected injury.**

4. Providing the necessary conditions for treatment and monitoring.

The ‘rule of three catheters’ (catheterisation of the peripheral vein, bladder and gastric, preferably nasogastric, tube) is not so categorical in the management of coma at the pre-hospital stage:

- in a comatose state, medicines are administered only parenterally (there is a high risk of aspiration with oral administration) and preferably intravenously; it is mandatory to insert a catheter into a peripheral vein; infusions are carried out through it, and in case of stable haemodynamics and no need for detoxification, an indifferent solution is slowly drip injected, which ensures a constant possibility of administering medicines;

- bladder catheterisation should be carried out according to strict indications, since in pre-hospital care this manipulation is associated with the risk of septic complications, and it is difficult to ensure the required degree of fixation during transport;

- insertion of a gastric tube with a preserved vomiting reflex without prior intubation of the trachea and its sealing with an inflated cuff is risky in coma due to the possible development of aspiration of gastric contents (a potentially fatal complication, which is why the tube is installed).

5. **Diagnosis of carbohydrate metabolism disorders and ketoacidosis:**

- determination of glucose concentration in capillary blood using a visual test strip; glycaemic level allows to diagnose hypoglycaemia, hyperglycaemia and suspect hyperosmolar coma; at the same time, in patients with diabetes mellitus, accustomed to hyperglycaemia due to inadequate treatment, it is necessary to take into account the possibility of developing hypoglycaemic coma even with normal glucose levels;

- Determination of ketone bodies in urine using visual test strips; this manipulation is not feasible in case of anuria, and in case of ketonuria, it requires differential diagnosis of all conditions that can be manifested by ketoacidosis (not only hyperglycaemic ketoacidotic, but also starvation or nutritional deficiency, some poisoning).

6. **Differential diagnosis and control of hypoglycaemia, which is a pathogenetic link in a number of comatose states.**

Bolus injection of 40% glucose solution in the amount of 20.0-40.0; if the effect is obtained, but its severity is insufficient, the dose is increased (see below).

7. **Prevention of a potentially fatal complication - acute Wernicke's encephalopathy**.

This syndrome is the result of vitamin B1 deficiency, most pronounced in case of alcohol intoxication and prolonged fasting, and aggravated by large doses of glucose. In this regard, the administration of a 40% glucose solution in all cases, in the absence of intolerance, should be preceded by a bolus of 100 milligrams of thiamine (2 ml of vitamin B1 in the form of a 5% thiamine chloride solution).

8. Therapeutic and diagnostic use of antidotes:

a) opiate receptor antagonist

- the diagnostic administration of Naloxone should be treated with caution, since a positive reaction (albeit incomplete and short-lived) is possible in other types of coma, for example, in alcoholic coma

- indications for the administration of Naloxone are

- respiratory rate < 10 per minute
- pinpoint pupils
- suspicion of drug intoxication

- the initial dose of Naloxone (IV or endotracheal) can vary from 0.4-1.2 milligrams to 2 milligrams with possible additional administration after 20-30 minutes in case of repeated deterioration, but most often 0.4-0.8 milligrams is sufficient, which also provides the greatest safety; to prolong the effect, it is possible to combine IV administration with subcutaneous administration;

b) benzodiazepine receptor antagonist

- in case of poisoning or suspected poisoning with benzodiazepine drugs (Diazepam [Relanium, Seduxen], oxazepam [tazepam, nozepam], medazepam [Rudotel, mezapam], phenozepam), flumazenil (anexate) is indicated in a dose of 0.2 milligrams intravenously for 15 seconds, followed by 0.1 milligrams every minute up to a total dose of 1 milligram if necessary

- The danger of using flumazenil is the risk of developing convulsive syndrome in case of mixed poisoning with benzodiazepines and tricyclic antidepressants.

9. Combating intracranial hypertension, edema and swelling of the brain and meninges:

a) the most effective and universal method is mechanical ventilation in hyperventilation mode, which provides the required result within an hour, but due to the need for special equipment and a number of severe side effects, especially in the absence of adequate control, it can be used in the pre-hospital stage only on vital signs;

b) in the absence of high blood osmolarity (for example, in hyperglycaemia or hyperthermia), oxygen deprivation and in the absence of a threat of development or increase of bleeding (for example, in trauma, inability to exclude haemorrhagic stroke), dehydration is achieved by administration of an osmotic diuretic - Mannitol in the amount of 500 ml of 20% solution for 10-20 minutes (1-2 g/kg); to prevent further increase in intracranial pressure and increase in cerebral edema (ricochet syndrome), up to 40 milligrams of furosemide is administered after completion of the Mannitol infusion;
c) the traditional use of glucocorticoid hormones, which reduce vascular permeability and tissue edema around the brain lesion, based on their proven effect in cases of brain tumour; glucocorticoids with minimal concomitant mineralocorticoid activity and therefore not retaining sodium and water are used; dexamethasone (8 milligrams) is the most effective and safe;

d) limiting the administration of hypotonic solutions, as well as 5% glucose solution and 0.9% sodium chloride solution (no more than 1 l/m2/day), which does not apply to comas occurring against the background of haemoconcentration (hyperglycaemic, hyperthermic, hypocorticoid, alcoholic).

10. Neuroprotection and increasing the level of wakefulness:
- in case of prevalence of focal symptoms (especially in case of speech defect and other changes in higher cortical functions) over general brain symptoms (disturbance of consciousness not deeper than superficial soporific), piracetam is effective, activating metabolic processes and blood circulation in the brain, and has a protective effect in hypoxic and toxic lesions (drip infusion at a dose of 6-12 g);
in case of disturbances of consciousness up to the level of superficial coma, it is indicated
a) sublingual (or behind the cheek) administration of glycine in a dose of 1 g
b) intravenous administration of the antioxidant mexidol in a dose of 200 milligrams (6 ml of 0.5% solution) bolus over 5-7 minutes;
- in case of deep coma, intranasal administration of Semax in a dose of 3 milligrams (3 drops of 1% solution in each nasal passage)

11. Measures to stop the intake of toxin in the body in case of suspected poisoning:
a) gastric lavage through a probe with the introduction of a sorbent
- in case of poison ingestion by mouth
- when the poison is excreted by the gastric mucosa
- after intubation of the trachea and its sealing with an inflated cuff; b) washing the skin and mucous membranes with water
- when the poison enters through the integumentary tissues.

12. Symptomatic therapy:
a) normalisation of body temperature
- in case of hypothermia - warming the patient without using hot water bottles (burns are possible in the absence of consciousness) and intravenous administration of heated solutions
- in case of high hyperthermia - hypothermia by physical methods (cold compresses on the head and large vessels, rubbing with cold water or solutions of ethyl alcohol

and table vinegar in water) and pharmacological means (drugs from the group of analgesics-antipyretics);
b) seizure relief
- administration of Diazepam (Relianium) in a dose of 10 milligrams; c) vomiting relief
- administration of metoclopramide (Cerucal, Raglan) in a dose of 10 milligrams by IV or IM.

13. ECG recording is mandatory for all comas.

TREATMENT OF COMA

B. DIFFERENTIATED THERAPY OF CERTAIN COMA STATES

1. Hypoglycaemic coma.

Bolus administration of 40% glucose solution (with preliminary administration of 100 milligrams of thiamine) in a dose of 20-40-60 ml, but due to the threat of cerebral edema, not more than 120 ml; if further administration is necessary, infusion of glucose in a decreasing concentration of 20%-10%-5% with administration of dexamethasone in a dose of 4-8 milligrams to prevent cerebral edema and as a counterinsulin factor; when administering large doses of glucose and in the absence of contraindications, subcutaneous injection of up to 0.5-1 ml of 0.1% epinephrine solution is permissible; if the duration of coma is more than several hours, intravenous administration of up to 2500 milligrams of magnesium sulfate (10 ml of 25% solution) is indicated.

2. **Hyperglycaemic ketoacidotic and hyperosmolar coma nonketoacidotic coma.**

Infusion of 0.9% sodium chloride solution in a volume of 1 and 1.5 litres, respectively, for the first time. In case of hyperosmolar and prolonged ketoacidotic coma, heparin therapy is indicated - up to 10 thousand intravenous units.

3. Starvation (alimentary-dystrophic) coma.

Warming the patient (see above), infusion of 0.9% sodium chloride solution (with 40% glucose solution at the rate of warming the patient (see above), infusion of 0.9% sodium chloride solution (with the addition of 40% glucose solution at the rate of 60 ml per 500 ml of solution) at an initial rate of 200 ml per 10 minutes under the control of respiratory rate, heart rate, PEC and auscultatory lung sounds, fractional administration of vitamins - thiamine (100 milligrams), pyridoxine (100 milligrams), cyanocobalamin (up to 200 mcg), ascorbic acid (500 milligrams); hydrocortisone 125 milligrams; in case of hemodynamic ineffectiveness of adequate infusion therapy and signs of stagnation - pressor amines - dopamine, norepinephrine

4. Alcoholic coma.

For suppression of bronchorrhoea and as a premedication before tracheal intubation - bolus injection of 0.5-1 ml of 0.1% atropine solution. Within 4 hours after alcohol intake, the following is indicated

gastric lavage through a probe (after tracheal intubation) to clean lavage water (10-12 litres of water at room temperature) and administration of an enterosorbent, warming (see above), infusion of 0.1% solution of atropine. above), infusion of 0.9% sodium chloride solution at an initial rate of 200 ml per 10 minutes under the control of respiratory rate, heart rate, PEC and auscultatory lung sounds with possible further switching to Ringer's solution, bolus or drip administration of up to 120 ml of 40%

glucose solution, fractional administration of vitamins - thiamine (100 milligrams), pyridoxine (100 milligrams), cyanocobalamin (up to 200 mcg), ascorbic acid (500 milligrams); in case of haemodynamic ineffectiveness of adequate infusion therapy - pressor amines - dopamine, norepinephrine.

5. Opiate coma.

Administration of Naloxone (see above; if tracheal intubation is required, premedication with 0.5-1.0 ml of 0.1% atropine solution is mandatory.

6. Cerebro-vascular coma in stroke.

Since the differential diagnosis of ischaemic and haemorrhagic strokes is absolutely impossible at the pre-hospital stage of care, only undifferentiated treatment is performed here:

- haemodynamic disorders are corrected according to the usual recommendations for coma (see above);
- to improve brain perfusion - bolus slow injection of 7 ml of 2.4% eufilin solution (with a CT exceeding 120 mm Hg)
- in severe cases, to reduce capillary permeability, improve microcirculation and haemostasis - bolus administration of 250 milligrams of ethamsylate, to suppress proteolytic activity - drip administration of Aprotinin (Gordox, Contrical, Trasylol) at a dose of 300 thousand units (30 thousand atre)
- stroke is the main indication for the use of glycine, semax, mexidol, piracetam (see above).

7. Eclampsic coma.

Bolus injection of 3750 milligrams of magnesium sulfate for 15 minutes, if convulsive syndrome persists - Diazepam bolus 5 milligrams until it is relieved; drip injection of Ringer's solution at a rate of 125-150 ml/hour, dextran 40 (rheopolyglucin) 100 ml/hour.

8. Hyperthermic coma (heat stroke).

Cooling (see above), normalisation of external respiration (see above), infusion of 0.9% sodium chloride solution at an initial rate of 1-1.5 litres per hour, hydrocortisone up to 125 milligrams.

9. Hypocorticoid (adrenal) coma.

Bolus injection of 40% glucose and thiamine solution (see above), hydrocortisone 125 milligrams, infusion of 0.9% sodium chloride solution (with addition of 40% glucose solution at the rate of 60 ml per 500 ml of solution, taking into account the amount already bolus injected) at an initial rate of 1-1.5 litres per hour under control of respiratory rate, heart rate, PEEP and auscultatory lung sounds.

MEASURES THAT ARE UNACCEPTABLE IN COMATOSE STATES

In any comatose state, regardless of the depth of cerebral insufficiency, the use of CNS depressants (narcotic analgesics, neuroleptics, tranquillisers) is fraught with increased severity of the condition; the exception is coma with convulsive syndrome, in which Diazepam is indicated.

Coma is a contraindication to the use of drugs with a stimulating effect (psychostimulants, respiratory analeptics); the exception is the respiratory analeptic bemegrid, which is indicated as a specific antidote for barbiturate poisoning.

Nootropic drugs (piracetam) are contraindicated in case of disturbances of consciousness deeper than superficial soporas.

Insulin therapy is not allowed at the pre-hospital stage.

ALGORITHM OF MANAGEMENT OF PATIENTS IN A COMATOSE STATE

Respiratory disorders	Restoration of airway patency. Ventilation through a mask or intubation tube (after atropine premedication). Inhalation of oxygen.
Hemodynamic disorders	Lowering the blood pressure to a level that is 15-20 mm Hg higher than usual (magnesium sulfate, dibazole, or eufyilin. ncrease the CT to an acceptable level with glucocorticoids, infusion therapy or pressor amines. Restoration of an adequate heart rhythm.
Suspected trauma	Immobilisation of the cervical spine.
Diagnosis and management of hypoglycaemia	Bolus injection of 20-40 ml of 40% glucose solution with preliminary administration of 100 milligrams of thiamine.
Intoxication	Gastric lavage through a probe with the introduction of a sorbent. Washing of the skin and mucous membranes with water.
Suspected drug intoxication, pupillary dilatation, pupillary pressure < 10 in 1 min.	Intravenous or endotracheal administration of 0.4-0.8 milligrams of Naloxone.
Suspected intoxication with benzodiazepines	Bolus injection of 0.2 milligrams of flumazenil with a possible additional injection of 0.1 milligrams up to a total dose of 1 milligram.
Fighting intracranial hypertension and cerebral edema	Infusion of Mannitol (if there are no contraindications) 2 g/kg over 10-20 minutes, followed by 40 milligrams of furosemide. Administration of 8 milligrams of dexamethasone. Restriction of administration of hypotonic and isotonic solutions. Mechanical ventilation in hyperventilation mode (as a therapy of despair).
Neuroprotection and increased wakefulness	If focal symptoms predominate over generalised symptoms, piracetam 6-12 g IV. In case of disturbances of consciousness up to the level of superficial coma - glycine 1 g s/l or per cheek, mexidol 200-300 milligrams IV. At any level of impaired consciousness - Semax 3 milligrams intranasally.

INDICATIONS FOR HOSPITALISATION

A coma is an absolute indication for hospitalisation, which can only be refused if an agonal state is diagnosed.

COMMONLY ENCOUNTERED MISTAKES

The most common mistakes in the provision of pre-hospital care to comatose patients are due to

1. inadequate material support of ambulance crews to modern requirements and
2. insufficient familiarity of ambulance personnel with these requirements.

The most frequent mistakes at the pre-hospital stage in general and with insects in particular are related to the correction of arterial hypertension. As a rule, it is carried out by intramuscular(!) injection of magnesium sulfate, less often - dibazole, which is always combined with papaverine, which is not indicated in these cases; dangerous clofelin and pentamine are used, and often in combination with other antihypertensive drugs, which often leads to an excessive decrease in CT.

The most commonly used solution for infusion therapy is isotonic sodium chloride solution, and less commonly 5% glucose solution, which is at the expense of colloidal solutions.

Diagnostic administration of 40% glucose solution, which is mandatory in the care of comatose patients, is extremely rare; in no case was the administration of concentrated glucose preceded by thiamine.

Glycaemia and ketonuria are not measured at the pre-hospital stage due to the lack of possibility; flumazenil and mexidol, which are not included in the package, are not used. Only in a few cases is a catheter inserted into a peripheral vein, which does not allow for a serious attitude to ‘ongoing infusion therapy’. No atropine premedication is performed before tracheal intubation. Oxygen therapy was rarely performed.

The doses of a number of drugs are limited by their quantity and rarely exceeded 0.4 milligrams for naloxone and 2 g for piracetam. The latter was administered in patients with the most severe generalised brain symptoms, i.e. when it is contraindicated.

5. 3. ABDOMINAL SYNDROME. PERITONITIS-2 hours

Peritonitis is one of the most frequent, severe and dangerous complications of acute surgical diseases and traumatic injuries of the abdominal organs. This term refers to a special form of the body's response to the contact of a pathogenic agent, most often microbial, with the serous membrane covering the internal organs of the abdominal cavity and its walls, which is accompanied by severe dysfunction of vital organs.

Peritonitis is an inflammatory, often purulent-inflammatory lesion of both individual areas and the entire peritoneum, which occurs in phases and is manifested by severe intestinal paresis, endogenous intoxication, and water and electrolyte balance

disorders, resulting in systemic and regional circulatory disorders, pulmonary gas exchange, liver and kidney function.

In peacetime, acute peritonitis is the cause of 2/3 of deaths in surgical diseases of the abdominal cavity. In almost 85% of patients, peritonitis is caused by various acute diseases of the abdominal cavity, in 5-8% by abdominal trauma, and in 5-10% peritonitis is a postoperative complication.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF PERITONITIS

The diagnosis of peritonitis is based on general and local symptoms.

General symptoms include

- acute, sudden onset or intensification of pain on the background of pre-existing chronic inflammation;

- symptoms associated with the development of general tissue hypoxia and intoxication: pallor of the skin, cyanosis of the lips and nose, acrocyanosis, aggravation of facial features, possible jaundiced skin and sclerae;

- changes in the cardiovascular system: tachycardia, arrhythmia, decreased blood pressure and central venous pressure;

-respiratory failure: frequent shallow breathing, increased skin moisture, swelling of the saphenous veins, venous hyperaemia, restricted chest excursion;

- hepatic and renal failure: decreased diuresis, in severe cases, yellowing of the skin and sclerae;

- progressive dehydration (fluid loss with vomiting, urine, transudation of fluid into the abdominal cavity and intestinal lumen): dry mucous membranes, decreased salivation, thirst, oliguria, cramps, decreased skin turgor;

- signs of circulatory failure: dizziness, fainting, weak pulse, cold extremities, decreased body temperature.

Local symptoms of peritonitis:

- abdominal pain, most pronounced at the location of the source of the peritoneal inflammatory reaction;

- **positive Schotkin-Blumberg symptom** (sharp increase in abdominal pain when the palpating hand is quickly removed from the abdominal wall after pressure). To detect the symptom, during abdominal palpation, the hand is pressed on the anterior abdominal wall, noting at this moment more or less pronounced pain. When the palpating hand is quickly withdrawn, the abdominal pain increases sharply - the symptom is positive. If the nature of the pain does not change when the hand is removed, the symptom is negative. A positive Shotkin-Blumberg symptom is caused by peritoneal irritation and is the most prominent sign of peritonitis. The symptom may be local, for example, it is determined in the right hypochondrium in patients with acute appendicitis, or diffuse, for example, in case of perforation of a hollow organ and leakage of the gastrointestinal tract contents into the abdominal cavity; abdominal distension;

- tension of the abdominal wall muscles, especially pronounced in the area of localisation of the source of peritonitis;

- tympanitis on abdominal percussion;

- weakening or absence of intestinal peristalsis, in advanced cases, a ‘splashing noise’ during abdominal auscultation;

- delayed bowel movements and gas.

Local or widespread tension of the abdominal wall muscles, as well as a positive Shotkin-Blumberg symptom, should be considered the main signs of peritonitis.

The classical picture of peritonitis is not always observed. Some patients with various diseases of the abdominal cavity, chest, retroperitoneal space, etc. develop a clinical symptom complex that simulates the clinic of peritonitis, the so-called pseudo-abdominal syndrome (acute false abdomen). In such cases, diagnosis is difficult and often requires the use of additional examination methods.

FEATURES OF THE CLINICAL COURSE OF DISEASES ACCOMPANIED BY PSEUDO-ABDOMINAL SYNDROME

Diseases and pathological conditions that in some cases are accompanied by the development of pseudo-abdominal syndrome include

- *respiratory diseases* (pneumonia, pleurisy)

-*Diseases of the cardiovascular system* (angina pectoris, rheumatism, some forms of myocardial infarction, chronic intestinal circulatory disorders - the so-called angina abdominalis)

- *Urological diseases* (acute pyelitis, pyelonephritis, paranephritis, acute urinary retention, urolithiasis, cortical necrosis of the kidneys);

- *Diseases and injuries of the central and peripheral nervous system* (meningitis, encephalitis, tumours, subarachnoid haemorrhage, brain contusion, acute lumbosacral radiculitis, shingles, intercostal neuralgia, etc;)

- *Hemorrhagic diathesis, some infectious diseases (*influenza, food toxicity, 20

- *diabetes mellitus*;

- *chronic fistula intoxication, etc.*

During the examination of a patient with pseudo-abdominal syndrome, the most important thing is to establish the true disease, in which pseudo-abdominal syndrome is only one of its manifestations.

Pseudo-abdominal syndrome is characterised by abdominal pain of various locations. At the same time, pain may be observed in different parts of the chest, lumbar region, external genitalia, etc. For example, in case of lower lobe pleuropneumonia, some forms of myocardial infarction, acute gastritis, pain is localised in the epigastric region, in case of renal colic, as a rule, it involves the lumbar region, the ureteral projection zone on the anterior abdominal wall, external genitalia, and the inner thigh. At the same time, in case of acute enterocolitis, food toxicity infection, haemorrhagic diathesis, the pain usually spreads throughout the abdomen. In some diseases and syndromes (myocardial infarction, renal colic), the pain can be very intense, which can cause diagnostic errors.

Differential diagnosis of acute abdomen should be based on a thorough examination of the patient's complaints, medical and life history, objective, laboratory and instrumental studies. This approach will allow to make the correct diagnosis in a

timely manner and in the vast majority of patients to avoid diagnostic errors and unnecessary operations.

Gastritis, gastroenteritis. Gastritis most often occurs in the presence of eating disorders (overloading the stomach with a large amount of poor-quality food, including spoiled or poorly cooked food, alcohol abuse, and ingestion of food allergens). The development of gastritis is also caused by many drugs (drug-induced gastritis).

Clinically, acute gastritis is manifested by a feeling of pain and heaviness in the epigastric region, belching with a rotten egg smell, nausea, which is accompanied by profuse vomiting. The vomit contains pieces of poorly digested food. Later, during repeated vomiting, bile may be observed. There are no symptoms of peritoneal irritation.

In typical cases, when there is a history of eating disorders, it is not difficult to diagnose acute gastritis. Diagnostic methods: FEGDS, faecal bacterial culture to determine the microflora and its sensitivity to antibiotics.

There is nausea, sometimes vomiting, and fever (up to 37-38°C). The general condition of the patients is satisfactory. The abdomen remains soft, and some patients have mild muscle tension in the right hypochondrium, sometimes throughout the right side of the abdomen. The Shotkin-Blumberg symptom is rarely positive. When palpating the abdomen on the right, there is significant tenderness at the outer edge of the rectus abdominis muscle (1-3 cm below the navel - McFadden's zone of ‘maximum tenderness’). There are positive symptoms of Steinberg (tenderness along the root of the mesentery of the small intestine inward from the cecum), Klien (displacement of the pain point during palpation from the right hypochondrium to the left when the patient turns to the left side). Leukocytosis is noted in the blood, in some patients with a shift of the leukocyte formula to the left.

The clinic of mesoadenitis is sometimes so similar to other diseases (appendicitis, intussusception, diverticulitis, pyelonephritis, etc.) that it is almost impossible to make a correct diagnosis before surgery. This determines the surgical tactics. In case of complete confidence in the diagnosis and the absence of peritoneal phenomena, treatment can be conservative (bed rest, sparing diet, antibiotics, thermal physiotherapy procedures, antispasmodics, vitamins), which is carried out for 10-15 days.

If there are doubts about the diagnosis after observing the patient for 2-3 hours, in case of increasing clinical symptoms and peritoneal signs, urgent surgery is indicated. It is better to start it with a right-sided pararectal incision. In case of mesenteric lymphadenitis, exudate is removed from the abdominal cavity. Antibiotics and novocaine solution are injected into the mesentery of the small intestine. Only severely altered lymph nodes are removed, or the abscess cavity is opened and drained. One of the lymph nodes is necessarily taken for histological examination to clarify the diagnosis and differentiate it from tuberculous lymphadenitis.

Worm infection (ascariasis). The causative agent of ascariasis is a large nematode, the human roundworm. Intestinal ascariasis is characterised by dyspeptic symptoms. Often, especially in children, there is a violation of appetite, nausea,

salivation, sometimes there are abdominal pains that simulate acute appendicitis, as well as intestinal dysfunction (constipation or diarrhoea, or their alternation).

General weakness, fatigue, poor sleep, and dizziness are also noted. On the part of the blood, hypochromic anaemia may be mildly expressed, and in some cases, eosinophilia. Sometimes intestinal obstruction develops on the basis of ascariasis as a result of mechanical blockage of the intestinal lumen by hookworms or its spasm. In rare cases, intestinal breakthrough is observed with subsequent development of peritonitis.

The diagnosis is based on the detection of mature parasites or their eggs in the faeces.

Hemorrhagic capillary toxicosis (Schonlein-Henoch disease). The intestinal form of haemorrhagic capillary toxicosis can mimic the picture of acute appendicitis. However, when taking a detailed history, it can be established that patients with haemorrhagic capillary toxicosis have a history of skin haemorrhages, traces of which in most cases can be seen during a closer examination of the patient's skin. The abdominal pain is not localised but widespread, the body temperature is normal, and there is no hyperleukocytosis.

Having excluded the diagnosis of acute appendicitis, haemorrhagic capillary toxicity cannot exclude intestinal necrosis in a particular area. If abdominal pain increases, symptoms of peritoneal irritation, intestinal obstruction are observed, surgical intervention is indicated.

Urolithiasis. If renal colic occurs on the basis of urolithiasis, the behaviour of patients is very characteristic. If the stone is located in the pelvis or ureter, patients cannot find a place to sit due to severe pain and behave extremely restlessly. The pain is localised in the lumbar region, radiating to the thigh and genitals. If the stone is located in the lower ureter, painful and frequent urination, a positive Pasternacki's symptom, and haematuria occur.

Renal colic may be accompanied by nausea, vomiting, abdominal distention, and reflex tension of the anterior abdominal wall muscles. As a rule, the body temperature is normal, there are no changes in the blood.

To establish the diagnosis and differential diagnosis, ultrasound, chromocystoscopy, and X-ray examination of the urinary tract (selective pyelography) are used.

Endocrine diseases. Diabetes. Surgeons providing emergency care should remember that patients with diabetes mellitus in the stage of severe decompensation with acidosis and in a precomatose state may have pronounced abdominal manifestations that simulate acute abdomen syndrome.

Diabetic pseudoperitonitis occurs as a result of deep metabolic disorders, which lead to numerous minor subserosal hemorrhages in the stomach and intestines. Clinically manifested by abdominal pain, tension of the muscles of the anterior abdominal wall, restriction of respiratory mobility of the abdominal wall, tenderness on palpation. The pulse is frequent, the tongue is dry. In the blood - leukocytosis.

Intensive and rational antidiabetic therapy allows you to relieve abdominal manifestations within a few hours, as well as avoid unnecessary and extremely dangerous surgery in such a situation.

Thyrotoxicosis (toxic goiter). Regardless of the form (diffuse, nodular, mixed), goiter occurs with increased thyroid function. Excessive influx of thyroid hormones into the blood affects all organs and systems, primarily the nervous and cardiovascular. In some cases, patients with increased thyroid function are referred to the surgical department with a diagnosis of "acute appendicitis" or "acute cholecystitis". However, thyrotoxic crises that simulate the picture of an acute abdomen, despite their intensity, are functionally reversible. After surgery - subtotal strumectomy - pain in the abdominal area usually disappears.

Lung and pleural diseases. Pneumonia is an acute inflammation of the lungs, which affects both the entire lobe and its individual segments. The disease begins acutely, with chills that last 1-3 hours and are accompanied by a sharp rise in temperature (up to 39-40°C). Patients complain of a headache, then pain appears in the chest on the affected side, but it can also occur below the costal arch (in the case of localization of the pathological process on the right) - in the abdominal area, simulating acute appendicitis, acute cholecystitis, an attack of renal colic, etc. The diagnosis of pneumonia in typical cases is not difficult: acute onset, pain in the side, chills with high fever, herpetic rashes, "rusty sputum", crepitus and bronchial breathing during examination of the lungs, increased leukocytosis. A rather important role in the diagnosis belongs to X-ray examination of the lungs.

Pleurisy can be observed in almost all lung diseases, as well as in many diseases of other organs and systems. The main symptom of pleurisy is pain, which occurs when the parietal pleura is involved in the process and intensifies during coughing, talking, deep breathing.

Patients are concerned about cough, more often dry or with minor sputum production, body aches, increased fatigue, weakness. Auscultatively, pleural friction noise is heard.

In the case of supradiaphragmatic localization of pleurisy on the right, pain may be observed in the right half of the abdomen, simulating a subdiaphragmatic abscess, acute appendicitis or acute cholecystitis. The main diagnostic method is lung X-ray.

Myocardial infarction - an area of necrosis of the heart muscle caused by the cessation of blood flow or its receipt in an amount insufficient to cover the functional needs required at a certain moment.

The following clinical syndromes are characteristic of acute myocardial infarction: preinfarction state, pain syndrome, resorption-necrotic syndrome, acute heart failure, cardiogenic shock, cardiac arrhythmias, syndromes caused by dynamic disorders of cerebral circulation, abdominal syndrome.

In the presence of abdominal syndrome, acute myocardial infarction can give false symptoms of acute abdomen. Diagnostic difficulties in myocardial infarction are that there is no single constant sign or symptom in its clinical picture.

An important place in the diagnosis of myocardial infarction belongs to electrocardiographic examination. Based on the electrocardiogram, the localization of myocardial damage can be established.

Rheumatism is a common infectious-allergic disease with systemic inflammatory damage to the connective tissue, with a predominant localization in the cardiovascular system and frequent involvement in the process of other internal organs.

In the active phase of rheumatism, abdominal syndrome may be observed, in the presence of which there is severe paroxysmal abdominal pain with a dull tension of the muscles of the anterior abdominal wall. Flatulence, tenderness on palpation are noted. These symptoms often occur at the beginning of an active rheumatic process, which leads to an erroneous diagnosis of true abdominal pathology (acute appendicitis, acute cholecystitis, perforated gastric ulcer and duodenal ulcer, acute intestinal obstruction, etc.). Often, after a diagnostic error, unnecessary surgery is performed. Neutrophilic leukocytosis with a shift of the leukocyte formula to the left contributes to the erroneous diagnosis of acute appendicitis in rheumatism.

The pathogenesis of abdominal syndrome in rheumatism is associated with inflammatory changes in the peritoneum.

Quite important in the pathogenesis of changes occurring in the gastrointestinal tract is damage to the mucous membrane of the stomach and intestines. In case of damage to the vessels of the abdominal cavity, abdominal syndrome occurs, which is accompanied by dyspeptic phenomena. Asymptomatic or asymptomatic ulcers localized in the stomach or duodenum may also occur.

A well-collected anamnesis, the presence of damage to the valvular apparatus of the heart, the mild course of abdominal pathology, and a positive effect after antirheumatic therapy can help to establish the true cause of abdominal syndrome.

Typhoid fever is an acute infectious epidemic disease from the group of intestinal infections. In some cases, changes develop in the appendix, which can be so profound that they cause the clinical picture of acute appendicitis, which usually resolves after conservative treatment.

In some cases, as a result of the ulcerative-necrotic process of the appendix, a breakthrough may occur, which gives the clinical picture of acute peritonitis. In diagnosis, an important role is played by the anamnesis and the periodicity of the clinical course.

Nonspecific ulcerative colitis - widespread ulcerative lesion of the mucous membrane of the colon with periods of remissions and exacerbations.

Surgical complications: perforation of the colon, massive intestinal bleeding, acute toxic dilatation of the colon (toxic megacolon), lesions of the anorectal zone - stricture, fistulas, anal fissures. Complications of chronic nonspecific ulcerative colitis: stricture of the colon, cancerous degeneration, bleeding.

To establish the diagnosis of nonspecific ulcerative colitis, special research methods are used - sigmoidoscopy, irrigography, colonoscopy, if necessary - with biopsy.

Sigmoidoscopy in the acute stage of the disease reveals symptoms of a "weeping" mucosa, contact bleeding, continuous ulcerative surfaces with significant discharge of blood, pus, with excessive growth of granulations (pseudopolyps) in the late stage. Nonspecific ulcerative colitis must be differentiated from Crohn's disease, tuberculosis and intestinal cancer, in the presence of which intestinal perforation and the clinical picture of peritonitis may also occur.

Abdominal syndrome (abdominalgia), characterized by abdominal pain, is more common in childhood. The pain is of an unstable nature, without clear localization, sometimes vomiting occurs. There is tension of the muscles of the anterior abdominal wall, but the peritoneal symptom of Shchetkin-Blumberg is negative (not detected). In the pathogenesis of abdominal pain, irritation of the phrenic nerve and solar plexus, irradiation of pain from the pleura, pericardium, irritation of the peritoneum are of great importance. The outcome is favorable, but relapses are possible. It is necessary to treat the underlying disease.

Dysmenorrhea is a menstrual cycle disorder characterized by pain in the lower abdomen, sacrum, and lumbar region. In this case, a number of common symptoms are observed - dyspeptic disorders, palpitations, sleep disorders, skin rashes, etc.

With dysmenorrhea, before menstruation, many women feel general malaise, fatigue, irritability, and frequent mood swings are noted. Some women are unable to work and are forced to lie down for several days. Pain in the lower abdomen is cramp-like in nature, resembling colic, which gives a false acute abdomen syndrome.

Dysmenorrhea occurs mainly in adolescents, girls, and young women who have not given birth, but is sometimes observed in those who have given birth.

Polyarteritis nodosa is an independent nosological form of collagenous disease with systemic damage mainly of small and medium-sized arteries of the muscular type.

Polyarteritis nodosa is characterized by damage to the vessels of the mesentery, stomach, intestines, liver, pancreas and the development of abdominal syndromes of varying complexity, which give a false syndrome of acute abdomen. Abdominal manifestations in periarteritis nodosa are quite polymorphic not only in different patients, but also in the same patient at different stages of the disease. The variety of symptoms is due to the variability of the stages of the process.

Symptoms of damage to the vessels of the gastrointestinal tract are marked by abdominal pain of varying intensity, nausea, vomiting, lack of appetite. Sometimes there is aversion to food, intestinal activity is disturbed (constipation, diarrhea), tension of the muscles of the anterior abdominal wall, bloating, gastrointestinal bleeding, etc.

The first symptom is pain, which can be sharp, resembling intestinal colic, or dull, diffuse or localized. Sometimes it depends on the food intake and its nature. The pain is often combined with diffuse or local soreness, which increases with deep palpation, with abdominal distension and is accompanied by intestinal bleeding.

Thus, when conducting differential diagnostics, it should be remembered that all of the above diseases have specific clinical manifestations and with a thorough and attentive examination of the patient, one can, as a rule, avoid mistakes.

It should be remembered that any disease that causes clinical manifestations similar to peritonitis (pseudo-abdominal abdomen) almost never has all the combined symptoms characteristic of acute peritonitis, but has only some similarity in individual signs, of which the most common is abdominal pain.

However, in some situations, it is difficult for the doctor on duty to completely exclude or confirm peritonitis (extremely serious condition of the patient, concomitant pathology, advanced and senile age, etc.) and to make a decision on the need or refusal of emergency surgery. In this case, the doctor must use the entire arsenal of additional and instrumental diagnostic methods at his disposal. For diseases that cause a false acute abdomen, additional diagnostic methods are different (Table 5.3).

If, despite a complete examination, the doctor still has doubts about the diagnosis, it is necessary to resort to diagnostic laparoscopy (laparocentesis of the "balloon catheter" type) or laparotomy.

LABORATORY RESEARCH METHODS

Mandatory research methods:

- complete blood count, which includes the study of platelet count, hematocrit, blood viscosity;
- complete urine analysis;
- BCC and its components;
- determination of water-electrolyte and protein balance, acid-base blood status;
- determination of hemocoagulation and a number of other indicators (glucose, bilirubin, transaminase, amylase, urea, creatinine).

Instrumental research methods: - ECG;

- X-ray examination;
- ultrasound examination; - laparoscopy;
- laparocentesis;
- computed tomography.

Most often, the doctor on duty in an emergency situation, especially at night, does not have the opportunity to study a clinical blood test. Therefore, the most likely way is to study blood leukocytes. It has been established that the more severe the inflammatory process, the higher the number of leukocytes. However, it must be remembered that this phenomenon is not always natural, since even in the presence of purulent peritonitis, the level of leukocytes is often normal or slightly increased. Therefore, indicators of the degree of intoxication of the body are of particular interest in the diagnosis of peritonitis.

ETIOLOGY OF PERITONITIS

The main cause of peritonitis is infection.

1. Microbial (bacterial) peritonitis.

1.1. Nonspecific, caused by the microflora of the gastrointestinal tract. The most important strains of the following microorganisms are:

Aerobic and facultative gram (+) cocci: *Staphylococcus. Streptococcus. Enterococcus.*

Facultatively anaerobic gram (-) bacilli: bacteria of the family Enterobactericeae

Escherichia, Klebsiella, Proteus, Enterobacter, Citrobacter.

Aerobic non-fermenting gram (-) bacilli and coccobacilli: *Pseudomonas, Acinetobacter.Aerobic and facultatively anaerobic gram (+) bacilli: Lactobacillus.*

Anaerobic gram (-) bacteria: *Bacteroides. Fusobacterium. Veillonella.*

Anaerobic gram (+) cocci: *Peptostreptococcus. Peptococcus. Anaerobic gram (+) bacteria: Clostridium.*

1.2. Specific, caused by microflora unrelated to the gastrointestinal tract - *Neiseria gonorrhoeae,*

Streptococcus pneumoniae. Mycobacterium tuberculosis, etc.

2. Aseptic (abacterial, toxico-chemical) peritonitis:

- due to the impact on the peritoneum of aggressive agents of a non-infectious nature: blood, bile, gastric juice, chylous fluid, pancreatic juice, urine;
- aseptic necrosis of internal organs.

Special forms of peritonitis:

- carcinomatous (in advanced stages of abdominal tumors);
- parasitic;
- rheumatoid;
- granulomatous

PATHOGENESIS OF PERITONITIS

Peritonitis is inflammation of the peritoneum, which is a consequence of a complex local reaction of the body in response to damage to its tissues by various pathogenic stimuli - aggressive stimuli, which develops as a result of the interaction of the body with numerous pathogenic factors of the external and internal environment.

The sequence of changes in peritonitis has phases typical of a classical acute exudative-destructive inflammatory process: alterations (tissue damage with the release of inflammatory mediators), exudation (circulatory and microcirculatory disorders, migration of leukocytes from the vascular bed with their accumulation in the focus of inflammation), proliferation (reactions of connective tissue proliferation).

The features of the serous cover contribute to its rapid involvement in the inflammatory process, which, as a rule, is accompanied by a reaction of the peritoneal vascular apparatus in response to damage (the action of an irritant), which can be, in addition to the infectious agent, the contents of the abdominal cavity organs after their damage, and the effect of a mechanical damaging factor on the peritoneal cover. As a result, a spasm of the widespread capillary-vascular network (microcirculatory bed) occurs, which is then replaced by dilation of the vessels, thereby causing hyperemia and an exudative reaction. During exudation, fibrin deposits are formed on the parietal and visceral peritoneum, which adsorb toxins. A favorable development of events is associated with the activation of phagocytes and fibroblasts with subsequent

delimitation of the primary focus of inflammation in the abdominal cavity. If the creative activity of cellular elements is insufficient, the process ends with diffuse fibrinous-purulent peritonitis.

Based on the source and microbiological characteristics of the peritoneal exudate, peritonitis can be divided into three groups:

1. Peritonitis as a complication of diseases of the stomach, duodenum, bile ducts and pancreas. In these cases, the number of species of microorganisms involved in the infectious process is small, and they are represented by aerobic and facultative anaerobic microflora. Anaerobic bacteria appear only in the presence of paralytic intestinal obstruction.

2. Peritonitis as a complication of diseases of the small intestine, in the presence of which the contents of the abdominal cavity contain not only coccal microflora and enterobacteria, but in 50-60%

- anaerobes.

3. Peritonitis associated with the pathology of the large intestine, in the presence of which there is always a synergism of enterobacteria and bacteroids.

In the pathogenesis of peritonitis, the main role belongs to intoxication. The microbial factor in peritonitis is a trigger mechanism that leads to the development of complex, often irreversible disorders of the internal environment of the body. The penetration of microorganisms and their waste products through the peritoneum into the lymphatic and bloodstream marks the beginning of general damage to the body, i.e. intoxication.

The main pathogenetic links of endotoxicosis in peritonitis are:

- entry into the bloodstream from the infected abdominal cavity of toxins of bacterial origin;
- "generalization" of endogenous intoxication as a result of the pathological influence of microbial toxins, biologically active substances, the appearance of toxic products of impaired metabolism and autoantigenic toxic substances as a result of these influences;
- additional entry into the blood of toxic products from the intestine during the development of paralytic intestinal obstruction;
- gross violation of metabolic processes;
- functional and morphological damage to the organs of natural detoxification with

the development of multiple organ failure and toxic encephalopathy.

The development of intoxication syndrome leads to the formation of alterative inflammation in all organs and tissues, no system remains uninterested, although the manifestations of alteration in different systems are expressed differently.

If the infection in the focus is not suppressed and toxemia is not stopped, the alteration passes into an irreversible form: dystrophy, amyloidosis, tissue atrophy.

The regularity of this process and the severity of tissue damage in the alteration of internal organs are the basis for the classification of the severity of the intoxication syndrome.

At the 1st degree of intoxication, edema and swelling of the interstitium of parenchymal organs are observed without damage to the functioning tissue. A number of metabolic disorders occur in the tissues, caused by the production of certain biologically active substances - mediators of inflammation. These include, first of all, biogenic amines, eicosanoids, platelet activation factor, pro-inflammatory cytokines - interleukins 1,6,8, tumor necrosis alpha factor, as well as a whole group of incompletely studied chemotactic factors.

The main producers of pro-inflammatory mediators at the initial stage are endothelial cells. In the process of activation of endothelial cells, an important role is played by lipopolysaccharide (LPS) of the cell wall of gram (-) flora that colonizes the human intestine. It is believed that LPS is a key agonist of the synthesis of pro-inflammatory cytokines in peritonitis, triggering a cascade of pathophysiological reactions responsible for the development of multi-organ complications. Damage to cells and tissues by inflammatory mediators leads to disruption of their vital activity.

Almost all authors are unanimous in assessing the leading role of neutrophil granulocytes as inducers of pro-inflammatory and catabolic processes in the peritoneum. In particular, the important role of oxygen metabolism products and lysosomal enzymes in the initiation of initial inflammatory manifestations in the peritoneum is noted. Proteolytic and antitrypsin activity of the blood increases by more than 30%, and only by 8-12% is there an increase in blood toxicity. An important role is played by biogenic amines - histamine, serotonin, the main sources of which are mast cells, basophilic and neutrophil leukocytes, platelets. Biogenic amines cause dilation of microcirculatory vessels, increased capillary permeability, edema, increased mucus secretion. There is evidence that the degree of histamineemia correlates with the severity of peritonitis.

The condition of the patients is satisfactory, of moderate severity, consciousness is preserved, in some cases euphoria or depression of the psyche is observed. This indicates deeper metabolic disorders in the cerebral cortex due to the progression of the pathological process in the primary focus. Blood pressure is usually within normal limits, tachycardia corresponds to body temperature. A decrease in minute blood flow is detected within 10-15% of the norm. Gas exchange is not changed, since the shunts in the lungs open up to 10%, ventilation is within normal limits or there is hyperventilation with compensated respiratory alkalosis due to increased breathing. Enteral insufficiency, which is hypoxic in nature, predominates, which is manifested by intestinal paresis, impaired resorptive and barrier function of the small intestine, and disorders of cavity and parietal digestion. Changes in the liver are due to edema of the intercellular space and swelling of hepatocytes. This is determined by functional changes in the form of increased transaminases, bilirubin, changes in sediment samples. Diuresis is preserved, functional changes in urination are noted with preserved parenchymal apparatus. Biochemical blood tests do not reveal products of incomplete metabolism at this stage.

In the second degree of intoxication, there is a progression of disorders of the microcirculatory system and DIC syndrome, which is also a consequence of

hyperproduction of pro-inflammatory mediators in conditions of a diffuse inflammatory process in the peritoneum. Deep toxic lesions of the functioning tissue and internal organs occur, both with toxins of microorganisms and metabolic products. Changes in the nervous system increase in the form of degeneration of nerve ganglion cells of varying degrees. Blood flow decreases by 35-40% of normal, metabolic processes decrease significantly with inhibition of the cerebral cortex, as a result of which a state of stupor or stupor develops. There is an accelerated breakdown (catabolism) of protein and accumulation in the blood above the critical level of various amino acids up to 50% and polynucleoproteins up to 42%.

The functioning of the cardiovascular system deteriorates, toxic carditis develops, manifested by hypotension, unstable hemodynamics, a decrease in cardiac output and stroke volume within 25% of normal, total blood flow velocity and circulatory efficiency coefficient. Subcompensation of cardiac activity develops. Under these conditions, a compensatory decrease in peripheral vascular resistance is noted, which leads to the opening of arteriovenous shunts. In the lungs, peripheral blood flow and microcirculation decrease to 50% of normal, interstitial edema develops, gas exchange is disturbed, and decompensated metabolic acidosis is formed. The influence of exogenous and endogenous factors, biologically active substances of a protein nature, acidosis, hypoxia, dehydration and loss of a large amount of protein with exudate and digestive juices have an extremely adverse effect on the course of metabolic processes and the functional activity of the liver and kidneys. Enzymatic detoxification mechanisms are disrupted.

Blood flow in the liver decreases to 45% of normal, hepatocyte damage is noted with impaired metabolic processes. Hemodynamic disorders are observed in the form of portal congestion with reduced arterialization and liver hypoxia. Hypoxia is followed by a violation of the protein-forming function of the organ: first, the level of protein decreases sharply, and then its synthesis and resynthesis are disrupted. The deaminating and urea-forming functions of the liver deteriorate. The content of ammonium and glycol, products of incomplete metabolism: acetone, acetaldehyde, methyl isocyanide, ethanol, ethanenitrile, increases in the blood. Renal blood flow decreases, and, consequently, diuresis, acidosis develops. The specific gravity of urine increases, the number of cylinders, nitrogenous wastes in the blood increases. Intestinal paresis progresses, the barrier function of the small intestine is disrupted. The result of universal enteral insufficiency is the second "wave" of pathogenetic mechanisms: deep disorders of protein metabolism and water-electrolyte balance, which capture not only the interstitial, but also the cellular sector, translocation of anaerobic microorganisms from the lower parts of the intestine, replacement of cavity and parietal digestion with its symbiont forms (with the participation of microbial proteolytic enzymes), which lead to the formation of toxic polypeptides, as well as the release of lipopolysaccharide complex (LPS) and other bacterial endotoxins. This stage of intoxication is characterized by the development of lymphadenitis as a result of the fact that microorganisms and toxins are carried from the abdominal cavity by the flow of lymph

through the lymphatic vessels to the first-order lymph nodes and cause an inflammatory process in them.

In the III degree of intoxication, as a result of the progression of the inflammatory process in all organs and tissues, deep organic changes are formed in the form of atrophy or dystrophy with damage to the functioning tissue.

Blood flow in the brain progressively decreases (more than 55% of the norm), developing degeneration of nerve ganglia cells of varying degrees. Gross metabolic disorders with a predominance of catabolic tendencies are accompanied by coma, disruption of central regulation of respiration and blood circulation. The ventricles of the heart are dilated, there is swelling of heart muscle cells, the formation of carditis is noted. Systemic hemodynamics is unstable, pronounced hypotension with a tendency to collapse. Central hemodynamics is reduced by more than 25% of the norm with signs of right and left ventricular failure.

Increasing the output of water and protein into the extravascular space, which often reaches critical values, the osmotic pressure of the blood decreases, enzymatic systems are blocked in the tissues and metabolites accumulate. Blood flow and microcirculation in the lungs are sharply reduced (by more than 60%), shunts are opened by 40%. A typical clinical picture of adult respiratory distress syndrome develops.

Blood oxygen tension falls, acidosis progressively increases. Arterial blood oxygen saturation reaches 68% (normal 92% and more), and venous - 39% (normal 63%). Oxygen starvation of tissues increases against the background of progressive anemia. Its development is due to intravascular destruction of erythrocytes (hemolysis), decreased production of erythropoietin by the kidneys, insufficient production of erythrocytes as a result of toxic damage to hematopoietic cells of the bone marrow. The development of amyloidosis is noted in the liver. The glycogen reserve is depleted, the assimilation (use) of monosaccharides by the body and glycogen synthesis are disrupted. The level of hydrocortisone in the blood increases, there is an increased excretion of nitrogen, phosphates and calcium. At the same time, protein catabolism and the formation of glycogen from amino acids increase, while insulin production is inhibited. The hypoglycemia that initially occurred is replaced by hyperglycemia. Dehydration increases, the concentration of sodium and chlorine in the blood decreases with a simultaneous increase in its level in the intracellular sector (erythrocytes).

Amyloidosis or various forms of dystrophy develop in the kidneys with impaired metabolic processes: hepatorenal syndrome develops.

Lymph nodes are significantly affected with changes in their structure. Microorganisms penetrate the thoracic lymphatic duct and blood. The lymphatic system is the main connecting link in the transport of microbes from the abdominal cavity to the blood. Toxins are a source of significant pathological impulses in the vegetative mesenteric ganglia, leading to the development of a persistent pathological reaction - acute insufficiency of the motor, secretory and absorption functions of the small intestine. Deep disorders of intermediate metabolism develop. Intermediate metabolic products accumulate against the background of increasing blood toxicity.

Deep suppression of the immunosecretory system of the small intestine in combination with its pronounced microbial contamination play a key role in significantly increasing the permeability of the intestinal barrier for enteral microflora and massive bacterial invasion into the internal environment of the body and, first of all, into the portal tract. The increase in small intestinal intoxication increases the insufficiency of the liver barrier function, which is accompanied by the "breakthrough" of infectious agents into the systemic bloodstream and the development of endotoxic shock. Progressive heart and respiratory failure increase the disturbances of blood flow and metabolism in the brain, forming an irreversible process, and due to the neurotoxic block, it is not possible to break this vicious circle.

Thus, in the case of the development of intoxication syndrome, all organs and systems are involved in the pathological process. **At stage I,** reversible alteration is formed in parenchymal organs with preservation of basic functions. **At stage II,** degenerative changes occur in them with damage to functioning tissue. **At stage III**, deep dystrophic and atrophic processes occur in these organs with severe functional disorders, which ultimately determines a fatal outcome.

The natural mechanisms that develop in peritonitis include:

- the reaction of the CNS, sympathetic and neuroendocrine systems, which regulate hemodynamic and metabolic changes in the body in response to inflammation, trauma, and the presence of non-viable tissues;

- natural non-specific immune reactions, such as chemotaxis, opsonization, phagocytosis, complement activation, immunoglobulins, production of histamine, serotonin, prostaglandins, etc.;

- inflammatory mediators produced by cells (cytokines), discovered relatively recently thanks to the successes of molecular biology and molecular genetics.

Monocytes, macrophages, neutrophil leukocytes, lymphocytes, endothelial cells, fibroblasts participate in the production of cytokines. Cytokines produced by leukocytes are called interleukins (IL), because, on the one hand, they are produced by leukocytes, and on the other hand, leukocytes are target cells for IL and cytokines. Currently, more than 20 cytokines are known, 15 of which belong to IL.

Cytokines are relatively large protein molecules with a molecular weight of 10,000 to 45,000. They are similar in chemical structure, but have different functional properties. The most important role in the production of cytokines belongs to activated macrophages, both those that circulate freely in the blood and peritoneal fluid, and fixed (resident) macrophages, which are found in the liver, spleen, lungs and other organs. In the capillaries of the liver, i.e. in the sinusoids, along with endothelial cells, a significant place is occupied by Kupffer cells. They play the role of resident macrophages. Kupffer cells make up more than 70% of all macrophages in the body. They play a major role in the removal of microorganisms, endotoxins, protein breakdown products, and xenogenic substances.

The cytokine system includes 5 large classes, united by their dominant effect on other cells:

- interleukins: pro-inflammatory (IL 1, 6, 8, 12) and anti-inflammatory (IL4, 10, 11, 13, ILa, etc.);

- tumor necrosis factor (TNF);

- lymphocyte growth and differentiation factors;

- factors that stimulate the growth of macrophage and granulocyte colonies;

- factors that cause the growth of mesenchymal cells.

- Most reactions during inflammation are mediated by cytokines.

IL-1, for example, activates T and B lymphocytes, stimulates the formation of early phase inflammatory proteins, the production of anti-inflammatory mediators (IL-6, IL-8, TNF), platelet aggregation factor. It also increases the procoagulant activity of the endothelium and the adhesiveness of molecules, and causes an increase in body temperature.

IL-2 stimulates the production of interferon, increases the proliferation and cytotoxic properties of T lymphocytes.

IL-6 activates T and B lymphocytes and leukocytes, promotes an increase in leukocytosis, an increase in temperature, and the synthesis of early phase inflammatory proteins.

TNF stimulates the secretion of IL-1, IL-6, the excretion of prostaglandins, enhances the activation of neutrophils, eosinophils, monocytes. Activates complement and coagulation, increases molecular adhesion and vascular permeability, promotes the development of hypoxia, increases body temperature.

Factors that stimulate the growth of colonies of macrophages and granulocytes stimulate the growth of neutrophils, macrophages, eosinophils, interferon-production of TNF, IL-1, IL-6.

The above information shows that pro-inflammatory cytokines really determine almost all the changes that occur in the process of inflammation.

The production of cytokines depends on the state of the organism. In the body, their secretion is extremely small, designed to ensure the interaction between the cells that produce them and other mediators of inflammation. But it increases sharply during inflammation due to the activation of the cells that produce them. Activation of cells that produce cytokines occurs under the influence of endotoxins, microorganisms, hypoxia and reperfusion of tissues, significant trauma, the presence of non-viable tissues, shock, which is inevitably accompanied by hypoxia. A fundamental role in the regulation of acute inflammation through the activation of the cytokine cascade and the production of other pro-inflammatory mediators is played by protein molecules known as "nuclear factor kappa B". This factor activates the production of cytokines and other inflammatory mediators, plays an important role in the development of systemic inflammatory response syndrome and multiple organ dysfunction syndrome. For therapeutic purposes, it is possible to use agents to reduce the activity of this factor. This can lead to a decrease in the production of inflammatory mediators, reduce tissue damage, and reduce the risk of organ dysfunction. Natural killer cells play an important role in protecting the body from infection. They are produced by the bone marrow and are a subpopulation of large granular lymphocytes that, unlike T-killers, are capable of

lysing bacteria and target cells without prior sensitization. These cells, together with macrophages, perform a surveillance and sanitation function.

The mechanisms of the immune system, which function normally, prevent the uncontrolled release of cytokines and other mediators of inflammation, ensuring an adequate response of the body to inflammation. At the very beginning of inflammation, pro- and anti-inflammatory ILs appear simultaneously in the blood. Under such conditions, they functionally create a balance that determines the favorable course of the inflammatory process, the delimitation of the focus of inflammation (damage).

Excessive activation of cells that produce cytokines can lead to excessive release of ILs and other mediators of inflammation. In this regard, the body's reaction (response) to inflammation becomes systemic, a syndrome of systemic response (reaction) to inflammation occurs. This is a signal of the danger of developing complications, a violation of the function of the immune system that controls the production of cytokines and the severity of the body's reaction to inflammation.

Further uncontrolled release of cytokines by overly activated macrophages and other cytokine-producing cells leads to serious consequences. Cytokines, together with other mediators of inflammation, transform from a factor of the body's immune defense into a factor of aggression. The systemic response syndrome (reaction) to inflammation in these conditions will progress continuously, the patient's condition will worsen, and in the absence of adequate therapy it may develop into sepsis, septic shock, and multiple organ failure.

Increased production of N0 under the influence of high levels of endotoxins and cytokines enhances the autodestructive, damaging effect of the latter, contributing to the development of a severe systemic reaction of the body to inflammation. At the same time, N0 under certain conditions can reduce the production of cytokines, have an anti-inflammatory effect. Inhibition of the activity of NO synthetase can reduce the production and concentration of N0 in the blood, reduce its damaging effect, increase the tone of venous vessels, and increase blood pressure.

Of great importance in the development of inflammation are free oxygen radicals 02, N0, 0N00, produced by activated polymorphonuclear leukocytes together with cytokines and other inflammatory mediators. The most active radical is nitric oxide (N0). It is synthesized not only by leukocytes, but also by the endothelium of blood vessels. The small size of this particle, the absence of an electric charge, and lipophilicity allow it to easily penetrate membranes, participate in many reactions, and change the functional properties of some protein molecules. Optimal levels of NO in the blood are necessary to maintain normal venous tone and vascular wall permeability, adequate tissue perfusion, and cell protection from damage. NO protects the vascular endothelium (especially the liver) from the damaging effects of endotoxins and TNF, inhibits excessive macrophage activation, relaxes muscle cells in the vascular walls, participates in the regulation of vascular tone and vascular wall permeability, sphincter relaxation, and the destruction of bacteria.

Excessive production of N0 under the influence of cytokines has a damaging effect on tissues, as it contributes to a decrease in venous tone and peripheral resistance,

the development of hypotension, blood deposition, the development of edema, septic shock, the occurrence of multiple organ dysfunction, which often ends with irreversible multiple organ failure. In this regard, the effect of N0 can be both a damaging and protective factor for tissues.

The role of the endothelium in the development of inflammation and the edematous reaction to it is difficult to overestimate. The endothelium is a central link in the development, course and outcome of inflammation, the type of reaction of the body in response to it. Endothelial cells not only produce NO, endothelium, platelet-activating factor, but also serve as a link between parenchymal organ cells and platelets circulating in the bloodstream, macrophages, neutrophils, cytokines, their soluble receptors, NO and other inflammatory mediators. The endothelium of the microcirculatory bed subtly responds to changes in the concentration of these mediators in the blood and to their content outside the vascular bed. Endothelial cells are at the center of all reactions that develop during inflammation. It is this cell that, after stimulation by cytokines, acquires the ability to "direct" leukocytes to the site of damage.

In normal immune homeostasis, the beneficial effect of inflammatory mediators prevails over their damaging effect. The reaction of the body's vital systems to inflammation is moderate, adequate in nature, without signs of a systemic reaction, without organ dysfunction. With massive bacterial aggression, severe trauma (in particular, surgical), the presence of foci of necrosis, non-viable tissues, and acute pancreatitis, hyperactivation of macrophages, neutrophils, and other cells occurs. In this regard, the production and content of cytokines in the blood and the cells that produce them sharply increase, and the balance between pro- and anti-inflammatory cytokines and other mediators is disturbed. As a result, the immune system is damaged: it ceases to control the secretion of cytokines and other inflammatory mediators. The damaging effect of inflammatory mediators begins to prevail over the protective one. Disorganization of the immune system function, loss of control over the production of cytokines and other inflammatory mediators lead to the fact that pro-and anti-inflammatory cytokines and other inflammatory mediators (N0,02, prostaglandin E2), instead of limiting the inflammatory process, begin to have a damaging, destructive effect on tissues not only in the focus of infection, but also in other organs.

Cytokines circulating in the blood continuously activate macrophages, leukocytes and other cytokine-producing cells: their uncontrolled production occurs. As a result, the surface of the endothelium acquires increased thrombogenicity and adhesiveness, microthrombosis occurs, microcirculation is disturbed, massive vasodilation occurs, venous congestion, a sharp increase in vascular wall permeability, tissue hypoxia. Edema and hypovolemia develop, blood supply to vital organs is disrupted, their dysfunction occurs, which under certain conditions can develop into irreversible multiple organ failure, sepsis and septic shock.

Thus, the pathological process can develop continuously: from systemic inflammatory response syndrome to sepsis, multiple organ failure and septic shock. Recognition of the continuity of the pathological process allows for early recognition

of the danger of sepsis and the implementation of necessary therapeutic measures before complications develop.

THE MOST COMMON CAUSES OF PERITONITIS

The most common sources of peritonitis are:

- **appendix (30-65%)**: appendicitis - perforated, phlegmonous, gangrenous;
- **stomach and duodenum (7-14%)**: perforated ulcer, perforation of gastric cancer, gastric phlegmon, foreign bodies, etc.;
- **female genital organs (3-12%):** salpingoophoritis, endometritis, pyosalpinx, ruptured ovarian cysts, gonorrhea, tuberculosis;
- **intestines (3-5%)**: acute intestinal obstruction, hernia, thrombosis of mesenteric vessels, perforation of typhoid ulcers, perforation of ulcers in nonspecific ulcerative colitis, tuberculosis, granulomatous colitis (Crohn's disease), diverticulitis;
- **gallbladder (10-12%)**: cholecystitis - gangrenous, perforative, phlegmonous, prolapsed biliary peritonitis without perforation of the gallbladder;
- **pancreas (1%):** pancreatitis, pancreatic necrosis;
- **postoperative peritonitis** accounts for 5-10% of all diseases.
- **peritonitis, which is rare**, occurs with liver and spleen abscesses, suppuration of chylous ascites, breakthrough of paranephritis, pleurisy, some urological diseases, etc.

In some cases, the root cause of peritonitis cannot be established even after autopsy.

Such peritonitis is called cryptogenic.

PERITONITIS CLASSIFICATION

According to the clinical course**, acute and chronic peritonitis are distinguished.** The latter in the vast majority of cases is of a specific nature: tuberculous, parasitic, etc. In practice, surgeons most often have to deal with acute peritonitis.

It is customary to distinguish primary, secondary and tertiary peritonitis. **Primary peritonitis** is quite rare, occurring in approximately 1% of all cases. In primary peritonitis, widespread peritoneal inflammation is caused by haematogenous, lymphogenous or other translocation of pathogens **from** an extraperitoneal site. ***Primary peritonitis*** is divided ***into tuberculous and spontaneous in children and adults.*** *Tuberculous peritonitis* develops as a result of haematogenous peritoneal infection in specific intestinal lesions, as well as tuberculous nephritis and in women with tuberculous salpingitis. *Spontaneous peritonitis in children* (neonatal period and 4-5 years of age) is usually caused by systemic diseases (lupus erythematosus) or nephrotoxic syndrome. *Spontaneous peritonitis in adults* often occurs in patients with liver cirrhosis, chronic renal failure after ascites drainage, and in the case of prolonged peritoneal dialysis. The same form includes peritonitis that develops in women as a result of bacterial transmigration to the abdominal cavity from the vagina through the fallopian tubes.

Secondary peritonitis is the most common form of abdominal surgical infection, occurring in 80-90% of cases. The types of secondary peritonitis include

- peritonitis caused by perforation and destructive diseases of the abdominal cavity;

- post-traumatic peritonitis due to closed trauma or penetrating wounds of the abdomen;
- postoperative peritonitis.

Tertiary peritonitis is an inflammation of the peritoneum, sometimes referred to as ‘peritonitis without a source of infection’, ‘delayed peritonitis’. It usually develops in severe, weakened patients who have undergone several operations on the abdominal organs. The course of such peritonitis is not manifested by pronounced peritoneal symptoms, and is marked by an erased clinical picture. Moderate signs of severe sepsis (multiorgan dysfunction and refractory endotoxemia) are characteristic. Tertiary peritonitis develops as a result of severe secondary immunodeficiency, in the presence of which significant changes in the immune system are expressed to the maximum.

In practical surgery, acute peritonitis is most often encountered as a manifestation of an inflammatory process in the abdominal cavity.

According to the prevalence of the process, there are

- *local;*
- *widespread:*
- *diffuse* (extends beyond the area of inflammation and covers adjacent areas);
-*spreading* (covers a significant part or the entire abdominal cavity).

By the nature of the exudate:

- *serous*
- *fibrinous*
- *purulent;*
- *fibrinous-purulent;*
- *putrefactive*
- *biliary;*
- *dry.*

By the causes of occurrence:

- *traumatic*
- *contusion (after closed organ damage);*
- *perforation;*
- *postoperative;*
- *by duration;*
- *hepatogenic;*
- *cryptogenic;*
- *aseptic.*

PREDICTION OF ACUTE PERITONITIS SEVERITY

Given the significant differences in the treatment outcomes of different forms of acute peritonitis, **timely determination of the severity of the disease** is an essential stage of the diagnostic and tactical algorithm **.** In this case, the most important is the early detection of severe peritonitis, the results of treatment of which are largely determined by the timing of its onset.

The accumulated clinical experience shows that in many patients, the clinical course of peritonitis depends not only on the degree of peritoneal damage (which, of course, remains the main one), but also on many other factors related to age,

concomitant diseases, immune status, etc. In this regard, the severity of endotoxemia is assessed using a scoring system.

The scoring system is based on a numerical assessment of clinical, physiological, laboratory and biochemical parameters. The presence of clinical symptoms or deviation of a clinical or biochemical parameter from the norm is determined by a score, and the values relating to one patient are summed up in a common scale. The resulting number provides an explanation of the accuracy of the diagnosis or a certain degree of severity of the disease.

APACHE II scale is recognised as the gold standard and is widely used for quality assessment, intensive care management and reasoning in patients with trauma, septic shock and peritonitis (Chapter 18).

To predict the outcome of purulent peritonitis, an index has been developed (Table 5.4), called **the Mannheim peritoneal index (MPI).** PIM provides three degrees of severity of peritonitis:

-***Grade -1:*** the sum of points is 12-20, the predicted mortality is 0%;
-***Grade 2 :*** score 21-29, predicted mortality - up to 29%;
- ***Grade C:*** score of 30-47, predicted mortality - 100%

Table 5.4. Mannheim's peritonitis index

RISK FACTORS	**RISK ASSESSMENT (points)**
Age over 50 years old	**5**
Female sex	**5**
Organ failure	**7**
Malignant tumour	**4**
The duration of peritonitis is more than 24 hours.	**4**
The colon is the source of peritonitis	**4**
Diffuse spread of peritonitis	**6**
Exudate (only one answer):	
- clear	**0**
- muddy-purulent;	**6**
- faecal-purulent	**12**
Sum of positive answers:	**47 (max)**

PERITONITIS STAGES

Stage I is the initial stage. It lasts from several hours to a day or more. At this stage, the inflammatory process in the abdominal cavity is just beginning to develop; local peritonitis turns into generalised peritonitis. The effusion is serous or serous-fibrinous.

If peritonitis begins to develop due to organ perforation, its clinical picture consists of symptoms characteristic of a perforated ulcer, gallbladder perforation, intestinal perforation, perforated appendicitis, etc. The common symptoms of this initial phase of peritonitis due to perforation are more or less sudden sharp abdominal

pain accompanied by a picture of shock (sharply expressed, for example, in case of a perforated ulcer, less sharply expressed in case of perforated appendicitis, etc.) Peritonitis, which complicates inflammatory diseases of the abdominal cavity, does not have such an abrupt onset: there is no catastrophe, but there is a more or less rapid progression of the local process.

In the first period of peritonitis, patients always complain of pain, the intensity and radiation of which depends on the cause of the peritonitis. Pain may be absent only in the rarest cases of lightning or transient septic peritonitis. In addition to pain, reflex nausea and vomiting are almost always present.

Usually, from the very beginning, the patient looks like a person who is suffering severely, covered in cold sweat, lying in a forced position (often on his back with his legs pulled up to his stomach), unable to breathe deeply, but in absolute consciousness. The mood may be anxious, depressed, and speech is normal. The body temperature may be normal, but is often elevated. The pulse is frequent and of low filling, does not correspond to the temperature. Blood pressure during this period is often slightly lowered. The tongue is covered with a white coating, dry, but the mucous membrane of the cheeks is still moist. The abdominal wall does not participate in the act of breathing (only the intercostal spaces are retracted during inhalation), sometimes its stiffness can be visually determined.

The abdomen should be palpated gently, starting with superficial palpation of the least painful area, trying to determine the protective muscle tension. The clinical significance of this symptom is invaluable. G. Mondor (1937) believed that ‘in all pathology it is difficult to find a more correct, more accurate, more useful and more life-saving symptom’. It is ‘the supersign of all abdominal disasters’. As peritonitis progresses, the severity of this symptom decreases due to increasing intoxication and abdominal wall distention. Painfulness during deep palpation, the Shchotkin-Blumberg symptom, expressed in varying degrees, is detected from the very beginning of peritonitis.

During auscultation in the first hours of the disease, increased intestinal noise can be noted, then peristalsis becomes more sluggish, unstable, and the abdomen begins to swell.

Stage II is toxic. It occurs in 24-72 hours from the onset of the disease (sometimes earlier). its duration is 2-3 days (may be less). It is characterised by a pronounced inflammation process. There is fibrin and pus in the effusion, phagocytosis is weakened, and blood circulation in the intestinal loops is impaired.

The patient's condition becomes severe. He is worried about weakness and thirst. The patient continues to vomit, and in the end, it becomes regurgitation. The vomit is dark, brown and has an unpleasant odour (‘faecal vomiting’). The skin is moist, the face is pale, aggravated, and the eyes are sunken. There is cyanosis of the tip of the nose, earlobes, and lips. The extremities become cold; the nails are blue.

Breathing is frequent, shallow, sometimes intermittent, arrhythmic. Blood pressure is low, pulse pressure is reduced. There is a pulse rate of -120-140 beats/min, does not correspond to the temperature, soft, sometimes barely perceptible, then fuller, heart sounds are deaf.

The tongue is dry, covered with a dark coating that is difficult to remove. The mucous membrane of the cheeks is also dry. Dry mouth prevents the patient from speaking. The abdomen is distended, moderately tense and moderately painful to palpation, with a pronounced Shotkin-Blumberg symptom. Percussion of the abdomen reveals a uniform high tympanite, and in the gentle areas - a dull percussion sound, which changes its level when the patient turns, indicating the accumulation of fluid (exudate).

Auscultation reveals a sharp weakening, more often a complete absence of intestinal noise. Sometimes a 'falling drop noise' is heard. Gas does not pass, there is no stool. The urine becomes dark, there is little of it (less than 25 ml per hour). Urination may be painful. Examination through the rectum is painful.

Patients during this period usually remain conscious, although sometimes agitation and delirium may occur. Patients are often depressed.

Stage III is irreversible. It occurs 3 days or more after the onset of the disease, sometimes later, and lasts 3-5 days. The patient's condition is extremely serious. His appearance corresponds to the description of Hippocrates. Consciousness is confused, sometimes euphoria is observed. The skin is pale and jaundiced, cyanotic. Abdominal pain is almost absent. Breathing is shallow, arrhythmic, frequent, barely perceptible pulse, low blood pressure. The patient lies motionless, then tosses and turns, shudders, 'catches flies', eyes become dull. The abdomen is distended, its palpation is slightly painful, and auscultation is 'dead silence'

The transition of peritonitis from one stage to another occurs gradually, with no clear boundaries between the stages. In lightning-fast septic forms of peritonitis (peritoneal sepsis), it is impossible to distinguish the phases.

PROGNOSIS. Mortality rate in severe forms of purulent peritonitis is 25-30%, and in case of multiorgan failure - 80-90%.

In terms of prognosis, surgeons have returned to the positions formulated by S.I. Spasokukotsky in 1926: 'In case of peritonitis, surgery in the first hours gives up to 90% recovery, on the first day - 50%, after the third day - only 10%'.

BASIC PRINCIPLES OF PERITONITIS TREATMENT

Basic principles of peritonitis treatment:

- preoperative preparation;
- ***surgical methods of treatment;***
- ***postoperative treatment.***

Preoperative preparation. Along with general hygiene measures, gastric emptying with a probe and bladder catheterisation to control hourly diuresis, preoperative preparation in the presence of widespread acute peritonitis includes three main tasks. The first of them is solved comprehensively - **elimination of tissue dehydration, hypovolaemia and electrolyte disturbances.** This is achieved by infusion of isotonic polyionic solutions at the rate of 30-50 ml per 1 kg of body weight in the hemodilution mode. Infusion therapy is completed with the administration of protein and colloidal drugs.

The average duration of preoperative preparation is 2-4 hours. The total volume and qualitative composition of infusion therapy is determined by

- fluid deficit;
- volume of plasma and interstitial fluid;

- cardiac output per minute;
- total peripheral vascular resistance;
- stroke volume of the heart;
- in the most severe cases, electrolyte disturbances.

The volume of infusion therapy is also determined by the duration of the disease, taking into account the severity and condition of patients (APACHE II, SAPS, MODS scales):

- with a score of <10 (SAPS), the total volume of infusion before surgery is 20-35 ml/kg or 1.5-2 litres over 2 hours (haemodynamic disorders are not pronounced, dehydration does not exceed 10% of body weight);

- if the sum of points is > 10 (SAPS), the infusion volume is increased to 25-50 ml/kg or 3-4 litres over 2-3 hours (severe haemodynamic and water metabolism disorders: fluid loss of more than 10% of body weight).

The second task of preoperative preparation is to **medically correct disorders caused by endogenous intoxication and background diseases,** if indicated.

The third, extremely important task is to **ensure early (cooperative) initiation of adequate antibiotic therapy.** As is well known, surgery is associated with the inevitable mechanical destruction of the preserved biological barriers that separate the foci of inflammatory destruction and intestinal microbiocenoses. Hence the need to proactively create a therapeutic concentration of antibiotics in tissues not yet affected by the infectious process. Antibiotics for peritonitis are administered before surgery and continue in the postoperative period. To achieve a quick and maximum effect, antibiotics are administered intravenously. Antibacterial therapy regimens should include drugs that affect all clinically significant strains of microorganisms.

In moderate and medium peritonitis, preference should be given to 3rd generation cephalosporins : Cefobid, Medoceph (cefoperazone) - 1 g intravenously after 12 hours, Fortum (ceftazidime) - 1-2 g intravenously after 8-12 hours, Rocephin, Medaxon (ceftriaxone) - 1-2 g intravenously after 8-12 hours or ***semi-synthetic penicillins*** resistant to penicillinase: Unazin (ampicillin + sulbactam) - 3 g intravenously over 6 hours or ***fluoroquinolones:*** Tavanic (levofloxacin) - 0.5 g after 12 hours in combination ***with nitro-imidazole derivatives:*** Ornidazole - 0.5 g intravenously after 12 hours.

In severe forms of peritonitis, treatment should begin with carbapenems: Meronem (meropenem) - 1 g after 8 hours, Tienam (imepenem-celastatin) - **1 g** after 6 hours, **or *fluoroquinolones:*** Avelox (moxifloxacin) - 0.4 g intravenously after 24 hours, Gatifloxacin - 0.4 g intravenously after 24 hours, **or *4th generation cephalosporins:*** Maxipim (cefepime) - 2 g after 12 hours in combination with ***lincosamides:*** Dalacin C (clindamycin) - 0.6 g intravenously after 6 hours or ***nitroimidazole derivatives:*** Ornidazole - 0.5 g intravenously over 12 hours. It should be remembered that prolonged use of antibiotics should be combined with the administration of antifungal drugs - Diflucan, Fluconazole.

The choice of a specific combination of drugs and their regimen is differentiated depending on the assessment of the functional status of the body, which is determined by the score on one of the **SAPS, SOFA** or **APACHE II** scales (see sections 18 and 19).

Surgical treatment. The choice of anaesthetic method depends on the patient's condition. The most suitable type of anaesthesia is general anaesthesia.

In case of diffuse or general peritonitis, the operation is performed from the midline access in an open way. Recently, video-laparoscopic and laparoscopically assisted operations have been used for peritonitis.

The main stages of the operation:

- inspection of the abdominal cavity;
- elimination of the source of peritonitis;
- sanitation of the abdominal cavity;
- intestinal intubation;
- drainage of the abdominal cavity.

Abdominal examination includes:

- assessment of the nature of the exudate, its quantity and distribution;
- aspiration of exudate, bacterial culture;
- assessment of the condition of internal organs;
- clarification of the source of peritonitis: first of all, the cecum, gallbladder, stomach and duodenum, pancreas, hernia outlet, small and large intestine are examined;
- if there is blood in the abdominal cavity, the examination should begin with an examination of the parenchymal and pelvic organs to identify the source of bleeding and stop it.

Elimination of the peritonitis source. One of the main tasks of the operation is to eliminate the source of peritonitis. In these cases, the extent of surgical intervention depends on the cause of peritonitis. If it is impossible or inexpedient to completely eliminate the source of infection, it is drained and separated from the abdominal cavity with antiseptic tampons.

In the presence of widespread peritonitis, special attention is paid to determining the indications for resection of the abdominal hollow organs and the adequate choice of its volume.

If, due to suspicion of small intestine nonviability, the expected volume of its resection is close to significant (up to 1/2 of the total length of the intestine) or sub-total (up to 2/3 of the total length of the intestine), then, by the agreed decision of the operating surgeon and anaesthetist, it is acceptable to leave the intestine in the abdominal cavity with further decision on the volume of resection during relaxation in 6-8 hours. Elimination of the cause of circulatory disturbance in the intestine (dissection of the occluding ring, adhesions, etc.), as well as targeted infusion and drug therapy, help restore blood flow in the still viable part of the intestine and more clearly delineate the area of necrosis. The rationale for such tactics is the severe functional consequences of extensive resections of the small intestine, which are expressed in the malabsorption syndrome.

In the postoperative period, these patients should be in the intensive care unit. If total intravenous anaesthesia with mechanical ventilation was used as an anaesthetic procedure, then before programmed relaparostomy, corrective therapy is performed against the background of prolonged mechanical ventilation, without taking the patient out of the state of medication sleep. This reduces the traumatic nature of anaesthetic support and normalises the function of external respiration and gas exchange, which is extremely important for this patient population.

In conditions of widespread peritonitis, the risk of failure of intestinal anastomoses after resection increases. Therefore, **in the case of significant**

inflammatory changes in the ileal wall, anastomosis can be postponed until the peritonitis is eliminated. The ends of the crossed intestine are brought out side by side on the abdominal wall through a separate incision in the form of complete fistulas. Such a tactic cannot be recommended as optimal if the resection area is located near the ligament of Treitz. In this case, the risk of anastomotic failure competes with the risk of an artificially created high small bowel fistula with its dangerous functional consequences, especially for a critically ill patient. Therefore, the dilemma is often resolved in favour of anastomosis.

The question of the primary anastomosis after resection of the right half of the colon in the setting of widespread peritonitis is decided individually depending on the severity of peritoneal inflammation and the timing of its development. Resection of the left half of the colon in case of peritonitis should be completed with the imposition of a single-barrel unnatural anus with occlusion of the peripheral segment of the intestine, such as Hartmann's operation. An important element of such an intervention is devulsion of the external sphincter of the anus to decompress the disconnected colon and prevent suture failure at its sutured end.

Sanitation of the abdominal cavity. After removing the source of peritonitis, the abdominal cavity is sanitised with a large amount of antiseptic solutions. If possible, loose fibrin films are removed. Attempts to remove tightly fixed fibrin films are inappropriate and even dangerous.

The decompression tube can be inserted during open surgery or endoscopic examination.

According to the method of insertion of the decompression probe, there are

- closed integumentary route of probe insertion (through the nose);
- open through gastrostomy, appendicostomy, cecostomy.

According to the level of decompression probe insertion, there are

- proximal (antegrade) intubation and decompression;
- distal (retrograde) anal-intestinal intubation and decompression.

Intubation is used for decompression, fractional or permanent intestinal lavage, and intestinal portal haemodilution (simultaneous injection of glucose-electrolyte mixture into the umbilical vein and oxygenated glucose-electrolyte mixture with antibiotics, enterosorbents, and enzymes into the intestinal lumen).

Intestinal intubation. The next stage of intervention is intubation of the intestinal tube. Indications for it include widespread peritonitis, subcompensated and decompensated intestinal obstruction, and severe adhesions in the abdominal cavity. **The most gentle and effective method of decompression of the small intestine** is **nasogastrointestinal drainage with a Miller-Ebbott probe.** Of particular importance is the drainage of the initial part of the small intestine within 50-70 cm. At the same time, a separate probe channel that ends in the stomach is necessary for complete drainage of the stomach and prevention of regurgitation.

Drainage of the abdominal cavity. Drains are placed and fixed in a position that ensures the most adequate outflow of the contents:

- the upper abdominal cavity is drained through the counterpart in the subcostal area;

- the lower abdominal cavity is drained through a counterpart in the hypochondrium.

The location of drains in the abdominal cavity depends on the prevalence of ***peritonitis:***

- local peritonitis in the right hypochondrium - paired drains to the right hypochondrium and pelvis through the contraperitoneum in the right hypochondrium;

- diffuse peritonitis in the lower abdominal cavity - paired drains in both iliac zones;

- local peritonitis in the right hypochondrium (e.g., due to acute cholecystitis) - paired drains through the contraperitoneum located on the right immediately below the rib arch along the mid-axillary line. The drains are placed under the liver;

- Diffuse peritonitis involving the right half of the abdominal cavity (for example, in acute cholecystitis or perforated gastroduodenal ulcer - effusion in the right flank and pelvis) - paired drains through the counterparts located on the right immediately below the rib arch along the mid-axillary line and in the right hypochondrium;

- Diffuse peritonitis involving the upper abdominal cavity - twin drains through the counterparts located on the right and left immediately below the rib arch along the mid-axillary line;

- spilled peritonitis - drainage of the abdominal cavity according to A. A. Shalimov (Fig. 5.1): ***right*** hypochondrium - one drainage into the subhepatic space, the second - into the right subdiaphragmatic space; left ***hypochondrium*** - one drainage into the left subdiaphragmatic space; ***right*** hypochondrium- *paired* drains into the small pelvis; ***left hypochondrium*** - one drainage up the flank. In case of retroperitoneal phlegmon, preference is given to its extraperitoneal opening and drainage.

For the treatment of severe forms of spilled purulent and faecal peritonitis, programmed laparostomy and video laparoscopic rehabilitation are indicated. Some surgeons use ***planned or programmed relaxation****tomies* for this purpose. presence of temporarily joined surgical wound edges.

Indications for laparostomy or programmed relaparotomy:

- any stage of diffuse peritonitis with multiorgan failure;

- any stage of spilled peritonitis with massive faecal contamination of the abdominal cavity;

- anaerobic peritonitis;

- enteritis into a purulent wound in case of widespread peritonitis;

- Multiple abdominal abscesses with pyogenic capsules or thick fibrin deposits intimately adherent to the serous membrane of organs and not removed during abdominal lavage;

- the source of peritonitis **has not been** eliminated;

- failure of anastomotic sutures with peritonitis;

- a high probability of anastomotic sutures failure against the background of spilled peritonitis.

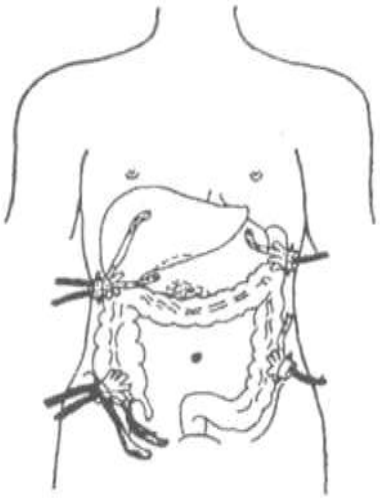

Fig. 1. Drainage of the abdominal cavity in case of spilled peritonitis.

The technique of laparostomy:

- the abdominal wall wound is left open;
- After the abdominal cavity is sanitised, the large omentum is fixed in the peritoneum;
- internal organs are isolated from the external environment with a sterile perforated plastic film;
- the film is fixed to the peritoneum or aponeurosis along the perimeter of the laparotomy wound with nylon sutures;
- loose wipes with antiseptic are placed on the abdominal wall wound.

The fundamental difference between programmed laparotomy and laparostomy is that during a programmed laparotomy, after completing all the necessary stages during the first operation, only the skin wound is sutured with separate sutures.

Some surgeons use special zip ties that are fixed to the laparotomy wound. **During laparostomy, the skin wound is not sutured but left open.** *Eventeration is prevented by a plastic tape sutured along the edge of the aponeurosis.*

The advantages of laparostomy over programmed relaparotomy are that in the case of an open method of treatment, it is possible to constantly monitor the condition of the abdominal cavity and lavage it. Moreover, laparostomy, unlike programmed relaparotomy, does not increase the pressure in the abdominal cavity, which helps to improve microcirculation in the intestinal wall and early recovery of its motor function, and also contributes to better aeration of the abdominal cavity, which is important in the presence of anaerobic microflora.

Programmed postoperative video laparoscopic rehabilitation (VLR).

Indications:

-duration of the disease not more than 12 hours;
- peritonitis caused by pathology of the colon;
- presence of massive dense fibrin deposits on the intestinal loops and peritoneum;
- significant visceral and visceral-parietal fusions;
- fibrinous-purulent nature of peritoneal exudate;
- severe inflammatory reaction of the parietal and visceral peritoneum.

Programmed postoperative video-laparoscopic rehabilitation (VLR).

Indications:

-duration of the disease not more than 12 hours;

- peritonitis caused by colon pathology;

- presence of massive dense fibrin deposits on the intestinal loops and peritoneum;

- significant visceral and visceral-parietal fusions;

- fibrinous-purulent nature of peritoneal exudate;

- severe inflammatory reaction of the parietal and visceral peritoneum.

Treatment in the postoperative period. Patients in whom the operation is completed by suturing a plastic film into the wound, require a staged lavage of the abdominal cavity. Peridural anaesthesia is the optimal method of anaesthetic manipulation during programmed relaparostomy. It is advisable to start staged lavage no earlier than 48 hours after the first operation (laparostomy). The total number of lavages can be from 2 to 6 or more. Repeated lavage of the abdominal cavity is usually performed every 48 hours until the peritonitis is completely resolved. Only then is the abdominal cavity finally closed and the laparotomy wound sutured. The procedure of the operation is as follows:

- After removing gauze napkins, plastic film or removing sutures and fixation devices, the contents of the abdominal cavity are aspirated;

- examine the anastomotic sutures, if necessary, strengthen them;

- the abdominal cavity is washed with antiseptics, and loose fibrin films are removed;

- if necessary, reinsert the plastic film, loosely place antiseptic wipes on top (laparostomy) or apply pharmaceutical sutures to the wound (programmed laparotomy).

Contraindications to the use of staged lavage are the predogonal and agonal state of patients.

Video-laparoscopic revision and rehabilitation of the abdominal cavity is performed 18-24 hours after surgery in the operating room under general anaesthesia. Prior to the intervention, up to 1.5-2 litres of solution can be forced into the abdominal cavity through the drains with an exposure time of 1 hour. The injected solution consists of 300 ml of 0.25% novocaine solution, 1000 ml of Ringer's solution, 100 ml of dioxidin. With an increase in the volume of the injected fluid for sanitation, the ratio of its components is proportionally increased. After that, glove-tube drains are removed, usually placed at four points. First, a 10-mm trocar is inserted through one of the counterperforations (mainly in the left hypochondrium) under finger control, and a laparoscope is passed through it. Then, nylon sutures are applied to the wounds of the counterparts, including the wound through which the laparoscope was inserted, to ensure the tightness of the abdominal cavity during the application of carboxyperitoneum.

The intra-abdominal pressure should not be lower than 10-12 mm Hg. After video-laparoscopic revision, trocar tubes are additionally inserted through other

counterperforations, and, if necessary, between the laparotomy sutures. It is optimal to perform adhesiolysis simultaneously with two or three instruments.

Subsequently, the adhesions are separated, organs are isolated from the adhesions, fibrin films are removed, and the condition of the sutures is assessed. After that, the abdominal cavity is lavaged with subsequent aspiration of the solution. Ringer's chlorhexidine solution, saline, etc. can be used as a washing solution. To increase the effectiveness of the antimicrobial effect of intraoperative decontamination, electrochemically activated (ECA) solutions are used, for example, 0.9% ECA sodium chloride solution.

Complex treatment of acute disseminated peritonitis in the postoperative period should include

- correction of haemodynamics;
- correction of water and electrolyte balance and metabolic disorders;
- meeting the energy and plastic needs of the body;
- ensuring normal gas exchange and eliminating microcirculatory disorders;
- antibacterial therapy;
- detoxification therapy, including active detoxification methods: plasmapheresis, lymph and haemosorption, ultra sound therapy;
- Increasing the body's natural resistance;
- elimination of functional intestinal insufficiency;
- parenteral and enteral nutrition;
- symptomatic therapy.

The main tasks of infusion therapy are to correct haemodynamic and metabolic disorders of water-electrolyte, protein, carbohydrate, and fat metabolism, metabolic shifts in the BUN, and parenteral nutrition.

When correcting hemodynamic disorders in patients with peritonitis, it is crucial to eliminate fluid deficiency in the body, as most patients have general dehydration.

At the same time, it is necessary to take into account fluid loss. During the first 3 days, patients with peritonitis lose an average of 200 to 550 ml of gastric contents daily, 800-1200 ml with respiration, 50 to 200 ml from the abdominal cavity through drains per day, and about 500 ml for each degree of temperature above 37°C. Thus, on average, in the postoperative period, a patient with peritonitis loses up to 3000 ml of fluid (30-45 ml/kg) per day.

To restore these losses and the BCC deficit associated with the transfer of fluid to the ‘third space’, infusion therapy is performed in the volume of 50-80 ml/kg of body weight, depending on the volume of pathological losses. Controlled haemodilution is performed with crystalloids and colloids in a ratio of 2:1 or 1:1. This volume of infusion therapy helps to eliminate dehydration, oliguria, arterial and venous hypotension, and restore intestinal motility.

Due to the development of isotonic dehydration in patients with peritonitis, infusion therapy should begin with transfusion of isotonic sodium chloride solutions, balanced saline solutions - Ringer's solution, Ringer-Locke, lactosol. Given that oncological pressure is reduced in peritonitis, infusion therapy should include colloidal

solutions such as Stabilol, Helofusin, 5% albumin, which ensures fluid retention in the vascular bed and prevents the development of edema. The maximum daily dose is 20 ml/kg of body weight. For the same purpose, a 5% sodium chloride solution in a dose of 3-4 ml/kg can be used. For haemodilution, improvement of microcirculation, prevention of coagulation and thrombosis, Refortan, Rheosorbilact, Rheopo-ligucin are administered, the optimal daily dose of which is 5-15 ml/kg.

Normalisation of water and electrolyte disorders. The daily water requirement for adults is 40 ml/kg. It has been proven that preserving the volume of extracellular fluid is more important for the body than maintaining its chemical composition, which is achieved by reducing the loss of Na+ in the urine and gastrointestinal contents.

Along with fluid losses into the 'third space', patients with peritonitis develop electrolyte metabolism disorders in the postoperative period: the concentration of K, Ca, Mg in plasma and cells decreases, resulting in general dehydration, intracellular acidosis and extracellular alkalosis (Na+ movement into the cell and its replacement by K+1H+, which are released into the extracellular space).

The deficiency of any electrolyte can be calculated using the universal formula:

Electrolyte deficiency (mmol/l) = (K1 - K2) x M x 0.2, where

K1 - the normal content of anions or cations in the plasma; K2 - the content of anions or cations in the patient's plasma;

M - body weight in kg.

0.2 - coefficient of calculation of electrolyte in extracellular fluid. Correction of electrolyte disturbances is carried out by the introduction of balanced polyionic solutions: isotonic sodium chloride solution, Ringer's solution, disodium, trisodium, lactasol, etc.

Correction of disturbances of the COS in the postoperative period is carried out taking into account the etiopathogenetic factors of the disease, which are important in the selection of appropriate methods of intensive care. The main measures for the correction of metabolic acidosis are the treatment of hypovolaemia, dyshydria, elimination of haemodynamic disorders, external respiration, and electrolyte balance.

Prevention of acute renal failure is carried out as follows: infusion therapy in the volume of 20-25 ml/kg at a rate of 80-100 drops/min, followed by 10 ml of 2.4% Eufilin and 0.5 mg/kg of Lasix under the control of diuresis, which should be at least 10 ml/kg or 60-80 ml/h. In order to improve renal blood flow, it is advisable to use a 4% dopamine solution - 2.5 ml intravenously drip per 400 ml of saline. Correction of microcirculatory disorders, elimination of BCC deficiency, normalisation of water and electrolyte balance are the main components of acute renal failure prevention.

Criteria for the effectiveness of infusion therapy:

-Heart rate <100 bpm.

- BP sys. > or = 100 mmHg
- BP > or = 80 mmHg.
- CVP > or = 50 mmHg.
- Diuresis - 30-40 ml/hour.

Antibacterial therapy. Antibiotic therapy begins immediately before surgery and continues in the early postoperative period. In most patients, the duration of antimicrobial therapy should be no more than 7 days. However, the optimal duration of antibiotic use may be based on intraoperative data obtained during the primary surgery. The remaining clinical manifestations of infection at the end of the recommended period of antibiotic therapy may be due to an unliquidated focus of infection. In such cases, additional diagnostic methods should be used rather than continuing the course of antibiotic therapy. If the infection cannot be controlled, the use of prolonged courses of antibiotic therapy may be considered reasonable. The criteria for the adequacy of antibiotic therapy are positive dynamics of the underlying disease, normalisation of body temperature, leukocyte and neutrophil count, stabilisation of haemodynamics, and absence of respiratory distress.

To prevent **acute ulcers and erosions of the digestive tract mucosa**, drugs that suppress gastric secretion are used (see Section 12.9).

Active methods of detoxification (plasmapheresis, lymph- and haemosorption, haemofiltration, haemodiafiltration), as well as **immunocorrective therapy** (pentoglobulin, roncoleukin, etc.) are important components of postoperative intensive care.

Restoration of the motor function of the gastrointestinal tract. In the postoperative period, it is necessary to establish constant aspiration of gastrointestinal contents through a probe with enterosorption with enterosgel, 1 tablespoon per 100 ml of water or Belosorb, 4 tablets 5 times a day. In the presence of decompensated intestinal obstruction, it is necessary to lavage the intestine with enterosorbents or sorbilact in the first hours after surgery.

To stimulate intestinal motility and eliminate the effects of paralytic intestinal obstruction, blockade of the entero-enteric inhibitory reflex at various levels is used: Ubretide 0.5 ml intramuscularly, and then 0.1 ml intramuscularly every 2 hours, peridural anaesthesia.

The main task of **further parenteral nutrition** is to provide the patient with the necessary energy and plastic material to prevent the breakdown of tissue protein and create conditions for the synthesis of new protein. Protein losses after major surgical interventions reach 50-70 g/day and increase by 30-50 g/day with the administration of glucocorticoids. To prevent protein breakdown, carbohydrates are administered, the nitrogen-preserving effect of which has long been known. The following rules are followed for parenteral nutrition:

1.Glucose is administered at a rate not exceeding the rate of its utilisation in the body, i.e. not more than 0.5 g/kg/h.

2. Mixtures of amino acids (Infesol, Aminosol, Aminoplasm-E, Aminoplasm-he-pa) are administered simultaneously with substances that release enough energy for their assimilation: 1 g of administered nitrogen should provide 800 kJ of energy.

C. Water-soluble vitamins (ascorbic acid, thiamine chloride) are administered daily in doses 2 times higher than the normal daily requirements; in case of prolonged parenteral nutrition, fat-soluble vitamins should also be administered.

4. Trace elements are replenished by plasma transfusion, iron - with its preparations, phosphorus requirements (30-60 mmol/day is normal) are replenished with potassium dihydro-orthophosphate solution.

Parenteral nutrition must be combined with early enteral nutrition, which contributes to faster recovery of intestinal motility, reduction of fluid loss and absorption of toxic substances, and translocation of microorganisms.

PLAN AND ORGANISATIONAL STRUCTURE OF THE CLASS.

(applies to each of the 2-hour components of a 6-hour class)

№	The main stages of the lesson, their functions and content	Learning objectives in terms of learning levels	Learning and control tools	Materials for methodological support of class visibility, control of students' knowledge	Duration (in minutes or in % of the total class time)
1	2	3	4	5	6
1. 1.1. 1.2.	Preparatory stage: Checking the attendance of higher education students and their readiness for the class Name of the topic and its motivational characteristics			Attendance and progress log	25-30 2-3 3-5
1.2.1. 1.2.2. 1.2.3.	The relevance of the topic in theoretical and practical terms. The importance of the topic for mastering the following sections of surgery, as well as for mastering the necessary practical skills and abilities. . The significance of the material for the perception of the material in other departments, as well as in relation to cross-cutting programmes and interdepartmental programmes. The importance of the topic for			Methodological development on the topic of the lesson	

	the formation of clinical thinking in the future doctor				
1.3.	Preparation of higher education students for the task in the classroom			Sets of situational tasks, test tasks, guidelines for performing practical skills, phonendoscopes.	4-6
1.3.1.	Setting the overall goal of the lesson				1-2
1.3.2.	Setting specific objectives for the lesson				
1.3.2.1	Knowledge to be acquired by the student				
1.3.2.2	Practical skills and abilities to be acquired by the student				
1.4.	Checking the initial knowledge of higher education students.			Sets of situational tasks, test tasks, guidelines for performing practical skills. Computer.	12-17
1.5.	Correction of the initial knowledge of higher education students by the teacher, as well as individual recommendations for eliminating deficiencies				4-6

2.	The main stage of the lesson:			Methodological developments and recommendations, tables, case studies.	43-46
2.1	Acquaintance of higher education students with the task for independent work with an indication of the method of implementation (possibly with a creative search of the teacher to improve the level of training of higher education students).				4-6
2.2	Independent work of higher education students				

3. 3.1. 3.2. 3.3.	The final stage of the lesson: Checking the level of knowledge of higher education students on the topic of the lesson and their motivational assessment. Form of control - solving situational tasks, written test control. Checking the practical skills of higher education students and their motivational characteristics. Indication of the topic of the next lesson with the teacher's comments and recommendation of literature for self-study.			Sets of situational tasks, test tasks, methods for performing practical skills.	15-20 9-12 4-6

6. Materials for activating higher education students during the teaching of the topic.

6.1. Topic: Acute respiratory distress syndrome.

ISSUES OF THEORETICAL TRAINING

1. Definition of acute respiratory distress syndrome?
2. Etiological factors of ARDS?
3. Pathogenesis of ARDS?
4. Diagnostic criteria for acute lung injury syndrome?
5. Diagnostic criteria for ARDS?
6. Basic principles of treatment of ARDS?

6.2. **Topic: 'COMA'**

CLINICAL SITUATIONAL TASKS

TASK 1.

The patient is 76 years old. The call to the ambulance team is due to the fact that 'the patient has stopped talking'. The previous morning, repeated vomiting was noted against the background of a moderate increase in blood pressure (up to 180/95 with the usual 150-160/85-90 mm Hg), in connection with which his wife gave him 1 tablet of Adelphan and 0.75 milligrams of clofelin). Since then, he became increasingly lethargic, slurred speech, and later stopped answering questions. The line team, which arrived at 14.5 hours and 5 minutes after a call received at 13.28 hours, recorded a CT scan of 160/90 mm Hg. The patient was found to be in a state of retardation, monosyllabic inadequate answers to simple questions, dysarthria, facial asymmetry, and left-sided hemiparesis. For the last 2 years, after a femoral neck fracture, the patient has not been getting out of bed. Suffers from arterial hypertension for at least 15 years. He is not treated regularly, takes adelphane and clofelin when the CT scan is elevated. The relatives could only mention pneumonia as a past illness. Diagnosis: 'Acute

cerebrovascular accident'. Treatment was carried out: 10 ml of 2.4% eufilin solution IV. Given the immobility of the patient and the need for constant care after the injury, it was decided to refrain from hospitalisation.

When saying goodbye, the patient's wife asked the team whether to continue giving him mannitol prescribed by the endocrinologist, who warned that this drug should always be taken without stopping. A closer questioning revealed that the patient had been suffering from diabetes for 8 years and had been taking mannitol 3(!) times a day for the past 2 years. After vomiting the previous morning, he refuses to eat, but continues to take mannitol. Having received this information, the paramedic administered a 40 ml IV glucose solution, against which all generalised and focal symptoms disappeared. The patient is adequate, orientated, asks to eat. He was left at home.

What are some common mistakes made by healthcare professionals?

This example demonstrates a number of common mistakes:

1. It was not taken into account that hypoglycaemia can mimic many different pathological conditions, including in the form of a mask of acute cerebrovascular accident.
2. Despite the presence of relatives, the medical history was not taken thoroughly enough; there were no active attempts to find out whether the patient had diabetes mellitus and was taking any hypoglycaemic drugs.
3. The ambulance crew was going to leave the patient in a coma at the scene.
4. No attempt was made to administer a concentrated glucose solution for therapeutic and diagnostic purposes.
5. Thiamine was not administered before the administration of concentrated glucose.
6. The patient was not hospitalised after the first hypoglycaemia in his life.

TASK 2.

Patient M-v, 50 years old. The call to the ambulance team is due to burning pains in the lower third of the sternum and epigastrium, which have been disturbing for the last 3 days. The line team noted: the patient is hypernourished, inhibited, answers questions with difficulty after a long pause. Heart rate - 90 in 1 minute, rhythm is correct, MAP - 150/100 mm Hg with usually normal values. On the ECG: sinus rhythm, horizontal position of the EOS, moderate signs of left ventricular hypertrophy, changes in the T wave in the form of its smoothed or biphasic in most leads. The diagnosis was made: 'IHD: Progressive angina'. She was treated with isoket 2 doses (without effect), 2 ml of 50% analgin solution and 1 ml of 1% dimedrol solution by mouth, followed by 1 ml of 2% promedolol solution and 2 ml of 0.25% droperidol solution. She was admitted to the therapeutic intensive care unit.

During admission: The patient tries to answer questions (makes wordless sounds), his voice is quiet, which was initially regarded as a medication-induced sleep. However, there was a sharp dryness of the skin and mucous membranes, decreased skin and eyeball turgor. Tachypnea up to 24 in 1 min, despite the recent administration of promedol. Faint smell of acetone from the mouth. HR 100 in 1 min, MAP 110/80 mm Hg. There are no signs of stasis in the small or large circulation. The liver protrudes 2 cm from under the rib edge, densely elastic consistency. ECG: sinus tachycardia, horizontal position of the EOS, diffuse myocardial changes of the hypertrophied left ventricle.

The relatives who accompanied the patient managed to find out that for six months he had been experiencing severe thirst (he got up many times at night to drink water) and increased appetite, despite which he had lost about 20 kg during this time

The examination revealed hyperglycaemia (blood glucose concentration - 35 mmol/l) and ketonuria (acetone 4+ in a small amount of urine obtained through a catheter).

Infusion therapy, insulin therapy (10 units each under glycaemic control), oxygen therapy, cerebral edema control, and parenteral nutrition were performed. The patient's condition rapidly deteriorated: within 30 minutes, the stunning reached the level of coma, cerebral edema increased, against which, despite satisfactory glycaemic compensation, the patient died on the 5th day of hospitalisation.

What were the mistakes in the provision of pre-hospital care in this case?

Mistakes in the provision of pre-hospital care in this case were the following:

1. Despite the obvious clinical picture of stunning, the question of a possible developing coma was not considered, although there can be no changes in consciousness in 'coronary heart disease and progressive angina'.
2. The anamnesis was not collected thoroughly enough, which deprived the ambulance doctor of the most important and very characteristic information.
3. The patient was not examined thoroughly enough, which did not allow to detect such typical symptoms as dry skin and mucous membranes, decreased skin and eyeball turgor, tachypnea, acetone smell (although the latter is very subjective).
4. Most likely, the ambulance doctor was unaware that oesophagitis often develops as a result of excitosis in the setting of hyperglycaemia, which is manifested by burning behind the sternum.
5. Despite the obvious signs of stunning, the patient was administered drugs contraindicated in coma - dimedrol, droperidol and promedol, which led to a rapid progression of impaired consciousness and increased brain edema, from which the patient could not be brought out of.

TASK 3.

Patient E-ko, 84 years old, single, no medical history. The ambulance crew was called by neighbours due to the fact that for 2 days the patient does not get out of bed and does not answer questions. The line team, which arrived at 12.12 a.m. following a call received at 11.50 a.m., recorded: unconsciousness, unresponsive to external stimuli, anisocoria, paralysis of the right and upward gaze, facial asymmetry, right-sided hemiplegia, noisy, rattling breathing, respiratory rate - 28 per 1 min, heart rate - 72 per 1 min, CT - 235/140 mm Hg: 'Acute cerebrovascular accident'. Treatment was performed: 10 ml of 25% solution of

magnesium sulphate and 1 ml of 0.01% clofelin solution IV, 10 ml of 2.4% eufilin solution. The patient is left at home.

What mistakes were made in this case?

In this case, the following mistakes were made:

1. No attempt was made to diagnose the administration of a concentrated glucose solution.
2. Only eufiline was used out of all the possible treatments.
3. The CT was lowered without appropriate control by the administration of uncontrolled clofelin with unpredictable effects.

4. The patient is not hospitalised, which deprives her of any chance of a favourable outcome.

TEST CONTROL QUESTIONS

1.Which of the following symptoms is characteristic of acute respiratory distress syndrome

a) Rapidly progressive respiratory failure
b) Mild and painful dysphagia
c) Sharp increase in blood pressure
d) Mood instability

2. Which of the following factors is the main one in the pathogenesis of acute respiratory distress syndrome?

a) Release of inflammatory mediators and increased permeability of alveolar capillaries
b) Disturbance of electrolyte balance
c) Hypoglycaemia
d) Hypocalcaemia

3**.Which of the following symptoms is most characteristic of abdominal cavity syndrome?**

a) Abrupt increase in abdominal volume
b) High body temperature
c) Constant headache
d) Nausea without accompanying symptoms

4. Which of the following methods is the most diagnostically important for the determination of abdominal syndrome?

a) Ultrasound examination of the abdomen
b) Chest radiography
c) ECG
d) Blood glucose test

5.Which of the following is the main cause of collapse in surgical patients?

a) Loss of blood volume
b) High ambient temperature
c) Low electrolyte levels
d) Hormonal imbalance

6. Which of the following symptoms is characteristic of collapse?

a) Low blood pressure and heart palpitations
b) High platelet count
c) Rapid breathing without changes in blood pressure
d) Decrease in body temperature

7.Which of the following treatment strategies is the first priority in collapse?

a) Restoration of circulating blood volume by intravenous administration
b) fluids
c) Lowering the body temperature
d) Long-term observation without active treatment
e) Administration of antiviral drugs

8. What are the main methods in the treatment of acute respiratory distress syndrome?

a) Ventilation of the lungs with positive end-expiratory pressure (PEEP) and
b) adequate oxygenation

c) Long-term use of antibiotics without correction of the underlying pathology
d) Use of anticoagulants and corticosteroids
e) Prophylactic administration of analgesics

9. What is the main symptom of acute respiratory distress syndrome?

a) Acute respiratory failure
b) Weight gain
c) Headache
d) Increased body temperature

10. What treatment method is most often used for patients with acute respiratory distress syndrome?

a) Artificial ventilation of the lungs
b) Oxygen therapy via a mask
c) Prescribing antibiotics
d) Taking bronchodilators

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