Ministry of Health of Belarus
Gomel State Medical University

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

Head of the department, MD, Doctor of medical sciences, Julia Cherniakova

Topic on the Intensive care in the early postoperative period

Educational and methodical development

for practical training teachers for 4th year students of medical faculty

Author: MD, PhD, Lyzikova TV

Gomel 2020

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

I. Relevance of the topic
II. Purpose of the lesson
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I. Relevance of the topic

An integral part of a physician anesthesiologist-resuscitator is proper management of the patient in the postoperative period. Knowledge of physiology and understanding of the mechanisms that occur in the body in response to operational stress and anesthetics are needed. Basic knowledge of fluid and electrolyte balance, acid-base metabolism, and methods of nutritional support allows the doctor to provide effective assistance to patients in critical condition.

II. Purpose of the lesson

Mastering the principles of diagnosis and intensive therapy of fluid and electrolyte imbalance (FEI), disturbances of the acid-base balance (ABB), nutritional support in critically ill patients in the postoperative period.

III. Tasks

The student should know:

• the definition of "the postoperative period", characteristics of its phases;
• key areas of the postoperative intensive care;
• normal levels of ABB, types of buffer systems of the body, mechanisms of compensation for disturbances of ABB;
• the causes of disorders of ABB, their clinic, diagnosis and correction;
• the definition of "fluid and electrolyte balance ", causes and clinical features of fluid and electrolyte imbalance (FEI);
• algorithm of laboratory diagnosis, treatment principles of essential FEI;
• the definition of "nutritional support", types of nutritional support;
• indications and contraindications for enteral nutrition;
• indications and contraindications for parenteral nutrition, basic preparations for parenteral nutrition, parenteral nutrition complications and their treatment.

The student should be able to:

• navigate in the physiology of the postoperative period;

• collect history and conduct a survey of patients with surgical pathology;
• diagnose various types of FEI on clinical and laboratory data;
• construct a differential diagnosis of various types of FEI;
• assess the nutritional (nutrient) status and estimate the energy needs of the patient;
• identify the indications and contraindications for the nutritional support of the patient.

IV. Sections studied before and needed for the session

Normal and pathological physiology, pharmacology, surgery, internal medicine, endocrinology.

V. Recommended Reading

Textbooks on the normal and abnormal physiology, pharmacology, surgery, internal medicine and endocrinology for medical students.

Suggested Reading on lessons

Main Reading

1. Dale, OA Anaesthesia and Intensive Care / O. Valley [and others] Ed. Ed. OA Valley - M., Medicine - 2008. - 574 p.
2. Lecture material.

Further Reading

1. Bachmann, AL Formula. / AL Bachman. Per. English. - Moscow - St. Petersburg: "Bean" - "Nevsky Dialect" 2001. - 192 p.
2. Usenko, LV Theoretical framework and practical foundations of nutrition support in hospital critical states. / Edited by prof. L.V.Usenko and prof. LA Maltsev. - Dnepropetrovsk, Art Press, 2008. – 352 p.
3. Marini, John J. Critical care medicine / John J. Marini, Arthur P. Wheeler. - M., 2002. - 992 p.
4. Sumin, SA Emergency conditions. / SA Sumin. - M., 2006. – 799 p.

VI. Questions for self-study

Questions on basic knowledge

1. Methods for monitoring postoperative patients;
2. Clinical pharmacology of analgesics and pain management;
3. Acute circulatory disorders, the means for fluid therapy, blood products;
4. Total body water, water sections of the body, the regulation of water-electrolyte balance;
5. Need for nutrients and energy in a healthy person, and changes in metabolism in critical conditions.

Questions on this topic:

1. The phases of the postoperative period. The value of pain in the mechanism of development of functional disorders in the postoperative period;
2. The main directions of postoperative intensive care;
3. The main forms of disorders of ABB and their clinical manifestations;
4. The principles of the correction of disturbances of ABB;
5. Clinical signs, diagnosis, treatment of FEI;
6. Nutritional support: methods, indications, contraindications;

7. Definition of metabolic needs. Monitoring during nutritional support;
8. Options for enteral feeding, types of enteral mixtures. Complications of enteral feeding and their prevention;
9. Parenteral nutrition (PN): a statement of the problem (providing energy exchange, plastic functions). Types of PN (full, partial). Composition, control of PN.

Topics of educational and research work of students.

1. Enteral nutrition in burns;
2. Parenteral nutrition in patients with renal insufficiency;
3. Parenteral nutrition in liver failure;
4. Modern methods of pain relief in the postoperative period.

Teaching tools for organization of independent work of students

1. Computer database;
2. Tasks, test control;
3. Thematic patients;
4. Patient records and other documentation;
5. Safety instructions, aseptic and antiseptic;
6. Bank of tasks for self-study.

VII. Training Material

Postoperative period - a period after the operation until the patient's recovery. There are the early and the late postoperative period. The early period starts from the end of the operation and continues until discharge from the hospital. It is the most important period.

There are 4 phases of the postoperative period:

1. catabolic;
2. transition;
3. anabolic;
4. weight gain.

The catabolic phase - takes 3 to 7 days. It is characterized by the activation of the sympathoadrenal system, increased synthesis of catecholamines, glucocorticoids, aldosterone, ACTH, angiotensin and renin. These cause a disturbance of microcirculation, hypoxia and the development of metabolic acidosis, the leakage of fluid from the bloodstream into the interstitial space, reduction of blood volume. As a result - the deterioration of the rheological properties of blood and excessive blood clotting. Also characterized by an increased protein breakdown and hyperglycemia due to the increased formation of glucose from glycogen and enhanced gluconeogenesis. Clinical features of the catabolic phase: in the first days bother pain in the wound, weakness, drowsiness, loss of appetite, thirst, body temperature of 37-38 C, increased heart rate to 20-30% of the basic level, a moderate increase in blood pressure, shortness of breathing with a decrease in its depth. VC is reduced by 30-50%. A mild leukocytosis is determined in the blood.

The transition phase begins in 4-6 days, is characterized by a gradual decrease in the activity of the sympathoadrenal system and catabolism. Consumption of energy and plastic

materials gradually decreases and begins the synthesis of protein, glycogen and fat. Clinical features: the disappearance of pain, normalization of body temperature and skin color, appearance of appetite, respiratory rate and heart rate closer to the basic level, there is peristalsis.

The anabolic phase is characterized by increased protein synthesis, glycogen, fat that were consumed during the operation and in the catabolic phase. Anabolic phase is a period of recovery of the cardiovascular, respiratory, excretory and digestive system. It lasts from 2 to 5 weeks, the duration depends on the initial condition of the patient, the severity of the catabolic phase.

The phase of weight gain continues to recovery and rehabilitation. Within 3-6 months, finally completed reparative organization, maturation of connective tissue and scar formation.

The role of pain in the development of functional disturbances in the postoperative period

Pain syndrome is a generalized response to the pain of the body and is characterized by activation of metabolic processes, the tension of the endocrine, cardiovascular and respiratory systems to the level of stress, which increases the risk of complications and mortality in the postoperative period. Systemic effects of pain:

Cardiovascular system: the rise of blood pressure, tachycardia, an increase in total peripheral vascular resistance (SVR), and myocardial oxygen demand;

Respiratory system: reducing the depth of breathing, decrease in tidal volume (TV), the occurrence of ineffective cough and risk of atelectasis;

GI: an increase in sphincter tone, a decrease of intestinal motility;

Urinary system: an increase in sphincter tone (urinary retention);

Coagulation system: increased platelet aggregation, inhibition of fibrinolysis;

Immunity: the development of lymphopenia, inhibition of the reticuloendothelial system, as a result - increased risk of infectious complications;

Endocrine organs: the increase in the concentration of catabolic hormones, reduction of concentration of anabolic hormones (insulin, testosterone), the increase of concentration of cortisol, renin, aldosterone, angiotensin and antidiuretic hormone (sodium retention, water and a secondary increase in extracellular space);

CNS: the emergence of anxiety, sleep disorders, depression (in continuous pain).

The main directions of postoperative intensive care

1. Assessment of consciousness and actions to restore it;
2. Assessment of lung function. Indications for extubation or prolonged mechanical ventilation. Prophylaxis and therapy of acute respiratory failure;
3. Stabilization and maintenance of the heart and circulatory system. Fighting hypovolemia;
4. Monitoring and timely diagnosis of complications requiring further surgery performance;
5. Control and correction of FEI, disturbances of ABB and metabolism;

6. Restoring passage through the gastrointestinal tract, early enteral and (or) parenteral nutrition.
7. Conduction of etiologic and pathogenetic therapy (rational antibiotic therapy, glucocorticoids, preventing thromboembolic complications, etc.).

Acid-base balance

The human body constantly produces acid as a by-product of metabolism. But herewith it maintains the pH within narrow limits required for the normal enzyme activity and the millions of chemical reactions. Normal blood pH is 7.35 - 7.45, it is supported by buffer systems and excretory function of the kidneys and lungs.

Pulmonary pH normalization mechanism - change of ventilation and, accordingly, change in allocation of carbon dioxide in the form of CO2 + H2O.

Renal pH normalization mechanism:
1. reabsorption and excretion of bicarbonate - HCO3-
2. formation of bicarbonate;
3. excretion of H + as titratable acid and ammonium ions.

Renal compensation mechanism is slower than respiratory, develops in a few days after changing pH.

Buffers

Buffer is a substance that resists changes in pH by absorbing or releasing H + by adding to it an acid or a base. Any buffer system of the body is composed of two parts - a weak acid and salt of a weak acid of a strong base.

Basic buffer system:
1. bicarbonate buffer [H2CO3/HCO3-] - up to 53% of the buffer systems;
2. hemoglobin buffer [HbH/Hb-] - 35% of the total;
3. protein buffer [HPr/Pr-] - 7% of the total;
4. phosphate buffer [H2PO4-/HPO4-] - 5% of the total number of buffer systems.

Response time for compensation mechanisms of ABB disturbances is different:
1. Extracellular buffering carried out using bicarbonate buffer develops in 10-15 minutes;
2. Intracellular buffering performed mainly by hemoglobin buffer develops over 2-4 hours;
3. Respiratory compensation develops over 3-5 hours (to a maximum of 12-24 hours);
4. Renal compensation takes effect within 6-8 hours (to a maximum of 5-7 days).

Key indicators of ABB and blood gases

pH - indicator of acidity, measured in units;

PaCO2 (partial arterial CO2 pressure) - the partial pressure of carbon dioxide in arterial blood, measured in mm Hg;

PaO2 (partial arterial oxygen pressure) - the partial pressure of oxygen in arterial blood is determined in mm Hg;

AB (actual bicarbonate) - true (actual) plasma bicarbonate, calculated at the real (true) PaCO2 and true blood oxygen saturation, measured in mmol/L;

SB (standard bicarbonate) - Standard bicarbonate reflects the concentration of HCO3- in the blood sample, equilibrated at 37 º C with a standard gas mixture at PaO2 = 100 mm Hg and PaCO2 = 40 mm Hg, measured in mmol/L;

BE (base excess) - the calculated deficit or base excess, i.e., the amount of a strong base or acid required to return the pH to normal in PaCO2 = 40 mm Hg and a temperature of 37 º C. A positive value indicates a deficit BE acids and the loss of hydrogen ions. Negative BE says about the relative abundance of acids and growth of hydrogen ions measured in mmol/L;

BB (base buffers) - the concentration of buffer bases, i.e., the amount of bicarbonate ions and anions of proteins measured in mmol/L;

Table. The normal value of the main indicators of ABB in the arterial blood

Indicator	Value
pH	7.35-7.45
$PaCO_2$	35-45 mm Hg
PaO_2	80-110 mm Hg
AB	19-25 mmol/L
SB	20-27 mmol/L
BE	± 2-3 mmol/L
BB	40-60 mmol/L

Arterial blood gas analysis can be done very quickly, the information is as follows:
- PaO2 shows oxygenation;
- PaCO2 shows ventilation;
- SB or BE displays perfusion.

ABB disturbances occur with violations of ventilation, renal dysfunction, and in excessive acid "overload", which the body cannot cope.

ABB disturbances:

1. respiratory and metabolic acidosis;
2. respiratory and metabolic alkalosis;
3. compensated acidosis / alkalosis (pH in the normal range);
4. decompensated acidosis / alkalosis (pH outside the normal modes).

1. Respiratory acidosis

Acute respiratory acidosis is the most dangerous disturbance of ABB, developed in connection with acute respiratory failure. It is characterized by a sharp initial accumulation of CO2 in the body due to the decrease in alveolar ventilation. There is no renal compensation by excretion of non-volatile “fixed” acids. PaCO2 increases, combined with a decreased pH, BE level remains constant (PaCO2> 45 mmHg, BE ± 2 mmol / L, pH <7.35). Changes in the other

parameters are associated with the characteristics of ABB shifts of the buffer systems in the blood. Buffer base remains constant. The accumulation of CO2 leads to an increase in cerebral blood flow, raised intracranial pressure and coma. Cardiovascular disorders are progressing much faster when combined with acidosis, hypoxia.

Treatment:

- improving of patency of the tracheobronchial tree;
- breathing support by external methods (mechanical ventilation);
- treatment of the underlying disease that caused acute respiratory acidosis.

Chronic respiratory acidosis develops long enough to enable renal compensation mechanism. Increased blood pCO2 accompanied by a moderate decrease in pH. Simultaneously, the base excess and HCO3- are increasing (pH <7.35, PaCO2> 45 mmHg, BE> +3 mmol/L). H^+ и Cl^- are excreted from the body. NH4Cl with the properties of a strong acid is release with urine. Compensatory metabolic alkalosis obvious. Respiratory disorders can progress despite renal compensation. Chronic respiratory acidosis does not pose a direct threat to the patient's life.

Treatment:

- Require treatment of the underlying disease.

2. Respiratory alkalosis

Acute respiratory alkalosis is characterized by primary acute loss of CO2 due to excessive alveolar ventilation. This is due to hyperventilation during mechanical ventilation or stimulation of the respiratory center and carotid cells induced by hypoxemia or metabolic disorders. There is an increase of pH due to a decrease of PaCO2, BE is not changed (pH <7.45, PaCO2 <35 mmHg, BE ± 2-3 mmol/l). There is dilatation of the vessels of the lungs and muscles and spasm of the vessels of the brain. Cerebral blood flow and intracranial pressure decrease. Possible violations of the regulation of respiration and brain disorders such as paresthesia, muscle twitching, seizures.

The treatment of the underlying disease (trauma, swelling of the brain) or condition (hypoxia) causing respiratory alkalosis *is required.*

Chronic respiratory alkalosis develops over a period of time sufficient to compensate at the expense of the kidneys. Urinary excretion of $HCO3^-$ increases and the secretion of non-volatile acids decreases. In the plasma of the blood increases the base deficit, pH is in the normal range or slightly elevated (PaCO2 <35 mmHg, BE <-3 mmol/L, SB <20 mmol/L, AB <19 mmol/L).

Treatment. There is need to address the root causing respiratory stimulation.

3. Metabolic acidosis

ABB indicators change as follows: pH <7.35, PaCO2 normal or <35 mmHg, BE <3 mmol / L, AB <19 mmol/L; SB <20 mmol/l.

The causes of metabolic acidosis:

- Lactic acidosis type A (due to anaerobic metabolism of tissue): Any condition that causes tissue hypoperfusion;
- Lactic acidosis type B (due to liver dysfunction): reduction of lactate metabolism in hepatic failure;
- Ketoacidosis: insulin deficiency and starvation;
- Renal failure (↑uric acid and SO_4^{2-});
- The massive rhabdomyolysis (↑H +, organic anions from damaged cells);
- Loss bicarbonate (diarrhea, the presence of intestinal and biliary fistula, diseases of the digestive tract).

Overall, the most common cause of metabolic acidosis in clinic is tissue hypoperfusion. Oxygen and optimization of volume status are important components of treatment as well as treatment of the underlying disease.

5. Metabolic alkalosis

ABB indicators change as follows: pH <7.45, PaCO2 normal or> 45 mmHg, BE> 3 mmol/L, AB> 25 mmol/L; SB> 27 mmol/L.

The least-studied disturbance of acid-base balance. There are 2 types of metabolic alkalosis: responding to the introduction of saline solution (chloride-dependent) and not reacting to the introduction of saline solution (stable). Chloride-dependent metabolic alkalosis occurs much more frequently. It develops due to reduction of the body water (eg, vomiting, use of diuretics). Profuse vomiting or nasogastric tube lead to the loss of hydrochloric acid. Metabolic alkalosis is often associated with hypokalemia due to secondary hyperaldosteronism amid falling blood volume.

Treatment. Metabolic alkalosis should not be fully corrected until removing of the main pathology. The method of choice for chloride-dependent metabolic alkalosis - infusion of NaCl and reimbursement of potassium deficiency. When alkalosis caused by a primary surplus of mineralocorticoids antagonists of aldosterone (spironolactone) give a good effect. When arterial blood pH is more than 7.60 there is need for infusion of acids (ascorbic acid 5 - 10 g / day) or hemodialysis. Metabolic alkalosis is much easier to prevent than to cure.

Algorithm to interpret arterial blood gases

1. Estimate pH 2. Estimate PaCO2 and standard bicarbonate (or BE) - this will help determine the nature of the offense - a metabolic or respiratory, to evaluate the adequacy of any

compensation. In metabolic acidosis we would expect a low PaCO2, in respiratory acidosis - high HCO3-, BE.

Fluid and electrolyte imbalance

Water makes up about 60% of body weight in adults and 75-80% in children (with age the water content decreases).

The total amount of water is divided into: *intracellular water* of 40% of body weight, and *extracellular water* is about 20% of body weight. Extracellular volume of water is distributed between the interstitial water, plasma, lymph and transcellular water. The movement of water through the cell membrane depends on the difference in osmotic pressure of the intra- and extracellular fluid. This value is referred to as the osmolarity and osmolality, and calculated as mg/dL or mmol/kg.

Osmolarity (osmolality) - the number of osmotically active substances in 1 liter (or 1 kg), plasma osmolality is 285-310 mg/dL. The osmotic pressure of the plasma is made by Na and Cl, intracellular osmolality depends on the presence of potassium and anions. Osmotically active substances are sodium, chloride, contained in plasma glucose, urea, mannitol, glycerol, sorbitol, etc. Water flows freely through the cell membrane and is always moving in the direction of the medium with greater osmolarity. The normal intracellular osmolality, interstitial fluid and plasma is the same. In practice, osmolarity is understood as plasma osmolarity.

Fluid and electrolyte imbalance (FEI) is called violation of water exchange. There are two main groups of FEI - *dehydration and overhydration*. Depending on the plasma osmolality each group consists of three types of FEI: *hyperosmotic, isosmotic, hyposmotic.*

Diagnosis of FEI:
1. History (vomiting and diarrhea);
2. The patient's complaints and clinical data: thirst, loss of turgor of tissues and skin, appearance of tongue (additional lines in the dehydration), the tone of the eyeballs, body weight (if it has changed for short periods of time), blood pressure and heart rate (tachycardia - an early sign of decreased blood volume, blood pressure is reduced with a significant water deficit), filling of the external jugular veins, edema, rales in the lung, assessment of the blood volume.

Dehydration

Hyperosmotic dehydration - water loss exceeds the loss of electrolytes, increased [Na] in plasma and osmolarity.

Reasons: lack of fluid intake or insufficient fluid replacement, significant fluid loss.

Clinic: cellular dehydration (thirst, nervous system disorders, pyrexia, fatigue, apathy) and extracellular dehydration (reduction of blood volume, manifested by reduced blood pressure, central venous pressure, increased heart rate, blood clots, oliguria, loss of turgor, dry mucous membranes).

Treatment:
- introduction of hypotonic and isotonic electrolyte solutions using the formula: the amount of required 5% solution of glucose (L) = (Na plasma - 142) × body weight (kg) × 0,2 / 142
- correction of hypokalemia;

Hyposmotic dehydration – develops mainly due to the loss of electrolytes, when plasma osmolality decreases. Cells are fed up with water.

The reasons: the loss of salts; polyuric phase of acute renal failure; diuretics and laxatives;

Clinic: signs of extracellular dehydration and cellular hydration (swelling of the brain, convulsions, coma, vomiting).

Treatment:
- infusion of isotonic solutions to restore the osmolarity of the extracellular space;
- infusion of hypertonic solutions;
- introduction of hypotonic solutions is contraindicated! (Danger of cell hydration).

Isosmotic dehydration - characterized by shortage of water and electrolytes, there is a steady fluid deficit in all environments.

Causes: hemorrhage, acute intestinal obstruction, burns, gastrointestinal fistula.

Clinic: cellular dehydration (apathy, weakness) and extracellular dehydration.

Treatment:
- isotonic electrolyte solutions;
- introduction of colloids;
- anti-shock therapy when indicated.

Hyperhydration

Isosmotic hyperhydration - is characterized by an excess of water. Osmolarity of plasma is normal.

Causes: heart failure, impaired renal function, preeclampsia, inadequate infusion.

Clinic: an increase of blood volume, CVP, coma, cerebral edema, ascites, pulmonary edema, edema of the lower extremities.

Treatment: the underlying disease, stimulation of diuresis, the compensation of protein deficiency.

Hyperosmotic hyperhydration - is characterized by an excess of water and electrolytes. Cells are dehydrated.

Causes: enteral administration of hypertonic solutions, parenteral administration of hypertonic solutions in violation of renal excretory function.

Clinic: extracellular fluid overload (pulmonary edema, heart failure, edema) and cellular dehydration (thirst, neurological and psychiatric disorders - delirium, coma).

Treatment: the restriction of salt and fluid; saluretics; hemodialysis.

Hyposmotic hyperhydration - (water poisoning) is characterized by an excess of water on the background of huposmotic syndrome.

Causes: excessive introduction of hypotonic solutions; increased activity of antidiuretic hormone (ADH).

Clinic: extracellular fluid overload (pulmonary edema, heart failure, edema); cellular hydration (neurological and psychiatric disorders, apathy, lethargy, convulsions, nausea, vomiting).

Treatment: stimulation of diuresis, limitation of water intake, infusion of hypertonic glucose solutions with insulin.

Imbalance of basic electrolytes

Na: Norm - 136-144 mmol/L, is extracellular electrolyte, the volume of extracellular fluid depends mainly on its content.

Hypernatremia (plasma sodium above 145 mmol/L) is accompanied by plasma hyperosmolarity and outflow of fluid from cells.

The reasons:
- Excessive intake of sodium;
- Increased catabolism;
- Loss of hypotonic fluid (hyperventilation, sweating, etc.);
- Renal dysfunction.

Clinic: caused mainly by cellular dehydration (thirst, anxiety, depression, coma, circulatory problems).

Diagnosis: hyperosmolarity of plasma, plasma sodium above 145 mmol/L.

Treatment:
- Restriction of salt;
- Infusion of isotonic solutions of glucose with insulin;
- Stimulation of diuresis if there is hypertonic fluid overload.

Hyponatremia (sodium less than 135 mmol/L)

The reasons:
- Loss of hypertonic fluid (vomiting, fistula, diarrhea, sequestration);
- Insufficient intake of sodium.

Clinic: cellular hydration.

Diagnosis: lower osmolarity of plasma, plasma sodium levels below 135 mmol/L.

Treatment:
- Infusion of hypertonic sodium solutions;
- When hyposmotic hyperhydration water restriction, stimulation of diuresis.

Potassium - is the major cation in intracellular fluid. The normal plasma levels are 3.3 - 5.5 mmol/L.

Hypokalemia - plasma levels below 3.3 mmol/L.

Causes: Inadequate intake / excessive loss of potassium, diuretic therapy, prolonged elevation of mineralocorticoid levels.

Clinic:
- CNS - apathy, psychosis, irritability;
- CVS - hypotension, cardiac arrest in systole, ventricular fibrillation;
- GIT - intestinal paresis, vomiting, flatulence, anorexia;
- Weakness, fatigue, paralysis of the respiratory muscles;

Diagnosis: clinical signs and plasma potassium levels below 3.3 mmol/L.

Treatment:
- The introduction of potassium solutions with glucose and insulin;
- Foods rich in potassium;

Hyperkalemia - plasma levels above 5.5 mmol/L.

Causes: renal failure, major trauma, burns, crush syndrome, hemolysis of blood.

Clinic:
- CNS - weakness, stupor, delirium;
- CVS - lowering blood pressure, ventricular fibrillation, cardiac arrest in diastole;

- Gastrointestinal - vomiting, diarrhea;
- Paralysis of the respiratory muscles, reduced tendon reflexes, muscle twitching, "pins and needles".

Diagnosis: clinical signs and plasma potassium above 5.5 mmol/L.

Treatment:
- Removal of potassium from the body (diuretics, laxatives, hemodialysis, hemosorption);
- Transfusion of glucose with insulin (for the transfer of potassium into the cell);
- The introduction of calcium (to neutralize the toxic effect of potassium on the heart).

Calcium - plasma levels of 2.2-2.6 mmol/L. Ca is involved in neuromuscular excitability, in the process of coagulation of blood, etc.

Hypocalcemia - Ca in plasma is less than 2.2 mmol/L.

Causes: hypoparathyroidism, vitamin D deficiency, acute pancreatitis, renal failure, rhabdomyolysis, the collapse of the tumor.

Clinic:
- Increased irritability of the nervous system (tetany, tonic seizures, laryngospasm);
- Motor and secretory dysfunction of the digestive tract;
- Tachyarrhythmia;
- Weakness, dizziness, headache, mental disorder.

Diagnosis: clinical features + reduction of Ca in the plasma + history (neck surgery - hypoparathyroidism develops either immediately after surgery, or for several years).

Treatment:
- Foods rich in calcium;
- Vitamin D;
- 10% solution of calcium gluconate or chloride 10-20 ml
- Parathyroidin, parathyreocrine at a dose of 50-100 units/day.

Hypercalcemia - Ca content in the plasma above 2.6 mmol/L.

Causes: primary hyperparathyroidism, malignancy, increased breakdown of bone tissue, increasing the flow of calcium and vitamin D.

Clinic:
- Gastrointestinal disturbances (nausea, vomiting, constipation, atony of organs, anorexia);
- Neurological symptoms (confusion, weakness, stupor, memory lapses, muscle hypotonia);
- Violations of CVS (hypertension).

Diagnosis: clinical + Ca increase in plasma.

Treatment:
- Treatment of the underlying disease;
- Infusion of glucose solution;
- Corticosteroid therapy;
- Hemodialysis.

Magnesium (0.7-1.2 mmol/L) - is an intracellular cation, is involved in the metabolism, stimulates fibrinolysis, inhibits neuromuscular stimulation.

Hypomagnesemia - magnesium content of less than 0.7 mmol/L.

Causes: Inadequate intake / enhanced renal excretion of magnesium results from osmotic diuresis, pancreatitis, acute intestinal obstruction, alcohol withdrawal.

Clinic:
- Neuromuscular disorders (anxiety, depression, nervousness, tremor, hyperreflexia, lethargy, seizures);
- Violations of CVS (hypotension, tachycardia, angina).

Diagnosis: Clinical and laboratory data.

Treatment:
- Treatment of the underlying disease;
- 25% solution of magnesium sulfate at a dose of 20-40 ml (contraindication is ARF).

Hypermagnesemia - magnesium content of more than 1.2 mmol/L.

Causes: Increased intake of magnesium (taking antacid, laxative, treatment of pre-eclampsia magnesium therapy), renal insufficiency.

Clinic: neuromuscular disorders (muscle weakness, hyporeflexia, paralysis, respiratory failure); violations CVS (hypotension, bradycardia).

Diagnosis: Clinical and laboratory data.

Treatment:
- Cessation of drug administration containing magnesium;
- The introduction of 10% solution of calcium gluconate at a dose of 10-20 ml;
- The use of neostigmine at a dose of 0.5 to 1 mg.

The formulas for calculating the deficit of electrolytes:

Deficiency of sodium (mmol) = (desired level of sodium - the current level of sodium in the blood serum) × 0,6 × body weight (kg)

Deficiency of potassium (mmol) = (desired level of potassium mmol/L - the current level of potassium in the blood serum) × 0,3 × body weight (kg)

Chloride deficit (mmol) = (desired level of chlorides mmol/L - the current level of chlorides) × 0,45 × body weight (kg)

Deficit of free water (with hypertonic dehydration): 4 ml/kg per mmol of sodium per liter of more than 145 mg/dL

Nutritional support

Malnutrition in patients leads to longer hospitalization period of rehabilitation and mortality. Nutritional support involves the introduction of nutrients needed for the body through the gastrointestinal tract (preferred) or directly into the blood.

I. Methods of nutritional support:

- Enteral tube feeding;
- Oral enteral nutrition;
- Mixed enteral-parenteral nutrition;
- Total parenteral nutrition.

II. Indications for nutritional support:

- Body mass index <18;
- Prolonged mechanical ventilation for more than 24 hours
- Disorders of swallowing and chewing that do not allow adequate nutrition
- Post-operative or pathologic (including chemical burn) injury of the esophagus, stomach, small and large intestine, pancreas, which does not allow to eat ordinary food;
- Hypoproteinemia less than 55 g/L or hypoalbuminemia less than 28 g/L;
- Availability of burn wounds or infected more than 15% of the total body surface area.

III. Nutritional support is not carried out in the following cases

- Refractory shock syndrome;
- Severe uncurable hypoxia;
- Sever uncorrected hypovolemia.

IV. Definition of metabolic requirements:

Stage 1 (the starting therapy):
Energy requirement of 35 kcal/kg or 2200-2500 kcal/day;
Need for protein - 1.5 g/kg/day or 80-100 g/day;

Stage 2 - calculation of the true energy requirements for nitrogen excretion in the urine:
The need for protein (g) = (urinary nitrogen excretion (g) + 4 g (extrarenal loss of protein) + 2-4

g of anabolic processes) × 6,25
The demand for energy (kcal/day) = (protein needs (g): 6,25) × 130

V. Monitoring during nutritional support:

Indicators	Multiplicity of measuring
Temperature	daily
Pulse	daily
Blood pressure	daily
Respiration rate	daily
Blood:	
Glucose	daily
Hemoglobin	daily
Hematocrit	daily
Leukocytes	daily
Platelets	daily
Prothrombin ratio	daily
ABG	daily
Potassium, sodium, chloride, calcium,	daily
magnesium	daily
Creatinine, urea	1 every 2-3 days
Albumin	daily
AST, ALT, bilirubin	1 every 2-3 days
Daily urinary urea	3 times a week
Osmolarity	

VI. Enteral nutrition

Currently, there are two main types of enteral nutrition: enteral nutrition (*tube feeding*) - introduction of enteral probe mixtures or stoma, and sip feeding - *oral* enteral diet through a straw sip.

The concept of oral enteral mixtures is to use an enteral mixture in those situations when the nasogastric tube is not necessary and the patient can self-supply through the mouth, however, the need for protein and energy substrates are high. In this case, administration of lactose free diet by mouth is possible, starting from the second day after the operation.

When you are unable to feed through the mouth use another option as tube feeding.

Enteral mixtures

All enteral mixtures vary in caloric density, osmolality, lactose, number of pharmaconutrients.

Quality standard for modern enteral mixture:

- Adequate caloric density (not less than 1 kcal/ml);
- Lactose-free or low-lactose;
- Osmolarity is not more than 340 mOsm/L;
- The mixture should not cause dangerous stimulation of intestinal motility;
- The place of production of the nutrient mixture is clearly indicated.

Classification of modern enteral mixtures:

1. Standard lactose-free isocaloric or hypercaloric mixtures:
- Dry powder mixtures: Nutrikomp Standard, Nutrizon, Nutrien Standard
- Ready-to-eat liquid mixtures: Nutrizon Standard, Nutrikomp, Liquid Standard, Nutrizon Energy.

2. Organ-specific and specialized mixtures:
- For patients with diabetes (Diabetes Nutrikomp, Glyutserna etc.)
- For patients with renal insufficiency (Nutrikomp Rena, Nutrien nephritis)
- For patients with diseases of the gastrointestinal tract and / or dysbiosis (Nutrikomp Fiber)
- For patients with respiratory failure (Nutrien Pulmo)
- For patients with hepatic impairment (Nutrien Hepa)

3. Semi-element diets (Nutrilon Pepto TSC, Alfaro, Peptamen, etc.)

4. Enteral composition for oral administration (Nutridrink, Nutrikomp Liquidity Standard and energy, Nutrikomp Diabetes, Nutrikomp Rena, Nutrikomp Fiber)

5. Modules to enrich the diet of natural products (Nutrikomp Protein module, power module Nutrikomp, MCT module, etc.)

Several authors single out a group hypercaloric diets, however, from our point of view, such a division is not appropriate because of any dry enteric mixture can be prepared as hypercaloric mixture at a concentration of 1.5 kcal/mL (1500 kcal/liter). This is accompanied by the growth of the osmolarity of the solution and significantly increases the risk of osmotic diarrhea.

Complications of enteral feeding and their prevention

1. Mechanical:
- tube twisting
- graze wound of the oropharynx/esophagus mucosa;
- tracheoesophageal fistula: a very rare in patients on mechanical ventilation;
- aspiration of gastric contents.

2. Gastrointestinal:
- nausea, vomiting, constipation, diarrhea.

3. Metabolic:
- hyperglycemia, disorders of ABB, FEI

VII. Parenteral nutrition

By parenteral nutrition understand the mode of administration of nutrients needed by the body directly into the bloodstream, bypassing the gastrointestinal tract. Parenteral nutrition can be added when it is used in conjunction with enteral feeding, and total when all the nutrients are introduced intravenously.

Indications:

- Inflammatory bowel disease (Crohn's disease, ulcerative colitis);
- Severe catabolism when only enteral nutrition is not enough (sepsis, burns, cancer, polytrauma).
- Preoperative patients;
- Postoperative patients - if you cannot feed through the digestive tract (intestinal obstruction, pancreatic, high intestinal fistula, etc.).

Contraindications:

- individual intolerance of components of food;
- refractory shock syndrome;
- hyperhydration;

The main components of parenteral nutrition

- donators of plastic material for protein synthesis: crystalline solutions of amino acids
- donators of energy: carbohydrates, fat emulsions

I. Donators of plastic material

The current standard is to use only the solutions of crystalline amino acids. Protein hydrolysates (casein hydrolysate, Infuzamin) currently excluded from clinical parenteral nutrition.

Standard solutions of crystalline amino acids:

- Aminoplasmal E 10%, 15% (20 amino acids in solution);
- Aminosteril KE 10% (14 amino acids);
- Vamin 18 (18 amino acids).

Specialized by age and pathology solutions of crystalline amino acids:

- Aminoplasmal Hepa 10% (20 amino acids), Aminosteril Hepa 5% and 8% (15 amino acids);
- Aminosteril-Jade (9 amino acids), Neframin (8 amino acids);
- AMINO Infant 6% (17 amino acids).

Solutions with a low concentration of amino acids:

- Aminoplasmal E 5% (20 amino acids);
- Infezol 4% (14 amino acids);
- Aminosol KE 5% (14 amino acids);
- Aminosol 600 5% (14 amino acids).

II. Donators of energy

Fat emulsions

Fat emulsions are the best source of energy - the energy density of 1 g is 9.3 kcal. There are various fat emulsions in the form of 10 and 20% solutions with calorie 1 kcal/ml and 2 kcal/ml.

- Lipofundin MCT / LCT 10 and 20% (MCT / LCT-emulsion)
- Lipovenoz 10 and 20% (LST-emulsion)
- Intralipid 10 and 20% (LST-emulsion)

Solutions of glucose 10%, 20%, 25%, 30%, 40%.

Carbohydrates are the most conventional energy sources in the practice of parenteral nutrition, the energy density of 1 gram glucose - 4 kcal. Currently, concentrated solutions of glucose are the most commonly used.

Additional ingredients of parenteral nutrition

Vitamin and trace element complexes for parenteral nutrition

• Soluvit (water-soluble vitamins)
• Vitalipid (fat soluble vitamins)
• Trakutil (trace)
• Addamel (trace)
• Tsernevit (vitamins)

Rules of the parenteral nutrition

1. Donator of energy (carbohydrates and / or lipids) should be introduced in parallel with the donators of plastic material (amino acids), preferably through a Y-shaped adapter.

2. The rate of infusion of fat emulsions is: 10% - up to 100 ml per hour, 20% - no more than 50 ml per hour.

3. Hyperosmolar solutions should be administered in a central vein

4. Infusion systems for total parenteral nutrition should be changed every 24 hours

5. Inclusion of glucose concentrates to the mixture during total parenteral nutrition

Complications of parenteral nutrition

1. Infectious:
- phlebitis and thrombosis of the central and peripheral veins;
- catheter infection (angiogenic sepsis).

2. Metabolic:
- hyper-hypoglycemia, hyper-hypokalemia, natremia, chloremia, fosfatemia etc.;
- disorders of acid-base status: hyperchloremic acidosis, etc.

VIII. Self-study

Task number one

Estimate daily energy requirement for the patient P. weighing 75 kg for 2 days after resection, if nitrogen excretion in the urine is 3 g

Task number two

Inspect septic patients to determine if there is any disorders of the ABB, the availability of compensation. To do this:

- inspect the patient, assess monitoring values;
- estimate laboratory data and ABG

IX. Clinical problems

Objective number one

The patient was 42 years old. For enteral nutrition was used hyperosmolar lactose milk mixture. On the 2nd day appeared stomach pain, stool 8-10 times / day, fatigue, hypotension.
What are the likely causes of the developed condition? What measures should be taken?

Objective number two

The patient after subtotal resection of the thyroid gland in the postoperative period developed seizures. What is the most likely diagnosis? Your tactics?

Test control:

1. Early postoperative period, it is ...

a) the period from the end of the operation to discharge the patient from the hospital;
b) the period after discharge from the hospital until they are cured;
c) the period from the end of the operation to complete recovery;
d) the first day after surgery.

2. The phases of the postoperative period:

a) catabolic;
b) transition;
c) anabolic;
d) recovery.

3. In patients with significant burn injury energy needs:

a) increase;
b) decrease;
c) do not change.

4. Place the buffer systems in descending order of importance:

a) bicarbonate buffer;
b) phosphate buffer;
c) hemoglobin buffer;
d) the protein buffer.

5. What percentage of body weight is water, an adult?

a) 60%
b) 65%
a) 70%
d) 80%

6. What percentage of body weight is intracellular water in an adult?
a) 20%
b) 30%
a) 40%
d) 60%

7. Osmolarity of plasma depends on its content:
a) sodium;
b) chloride ions;
a) glucose;
g) urea, mannitol.

8. What ABB disturbances should be expected in patients admitted to the hospital in a coma of unknown etiology?
a) respiratory acidosis;
b) metabolic acidosis;
c) metabolic alkalosis;
d) respiratory alkalosis.

9. Select the methods of nutritional support of the following:
a) the introduction of fat emulsion in the central vein;
b) the introduction of enteral mixture through jejunostoma;
c) the introduction of crystalloids into a peripheral vein;
d) the introduction of enteral mixture via nasogastric tube.

10. Indications for nutritional support are:
a) a body mass index <18;
b) disorders of swallowing and chewing, not adequately fed;
c) hypoproteinemia less than 55 g/L;
d) severe dehydration.

Answers:

Objective number one

Probable cause: Osmotic diarrhea and / or lactose intolerance.

Treatment: iso-osmolar (osmolality less than 340 mOsm L) or lactose-free mixture. With no effect - add to antibiotic regimen of vancomycin or metronidazole.

Objective number two

Diagnosis: Hypocalcemia.
Treatment: The introduction of calcium supplements (calcium gluconate, calcium chloride).

Test control:

1 - a;
2 - a, b, c;
3 - a;
4- a, c, d, b;
5 - a;
6 - c;
7 - a, b, c, d;
8 - a;
9 - a, b, e;
10 - a, b, c.