Ministry of Health of the Republic of Belarus
Educational Institution
“Gomel State Medical University”

Department of Pathological Anatomy

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METHODICAL GUIDELINES

for conducting a practical class
in the academic discipline “Pathological Anatomy”
for students
of the 3rd year, Faculty of International Students (instruction in English),
enrolled in the specialty
7-07-0911-01 “General Medicine”

Topic: “Radiation injuries. Resuscitation pathology”

Duration: 3 hours

Approved at the meeting of the Department of Pathological Anatomy
(Minutes No. 1 dated 30.08.2025)

2025

EDUCATIONAL AND INSTRUCTIONAL GOALS, OBJECTIVES, AND MOTIVATION FOR MASTERING THE TOPIC

Educational Goal:

- To study the definition, classification, etiology, pathogenesis, morphology, clinical presentation, complications, and outcomes of radiation injuries and resuscitation pathology. To analyze radiation injuries: sources (therapeutic radiation, occupational exposure, nuclear accidents, atomic bombs), types (acute vs. chronic, local vs. total body). To examine the pathogenesis of radiation injury: direct DNA damage (double-strand breaks) and indirect damage via free radicals (radiolysis of water). To describe the effects of ionizing radiation on rapidly dividing cells (bone marrow, gastrointestinal epithelium, germ cells, skin, lymphoid tissue). To analyze acute radiation syndrome (ARS): prodromal phase (nausea, vomiting), latent phase, manifest illness. Three subsyndromes: hematopoietic syndrome (2-6 Gy – bone marrow suppression, pancytopenia, infection, bleeding), gastrointestinal syndrome (6-10 Gy – mucosal denudation, diarrhea, fluid loss, sepsis), neurovascular syndrome (>10 Gy – cerebral edema, hypotension, coma, death within hours to days). To examine local radiation injury: radiation dermatitis (erythema, desquamation, ulceration, fibrosis), radiation pneumonitis (diffuse alveolar damage, hyaline membranes, fibrosis), radiation enteritis (mucosal atrophy, fibrosis, strictures), radiation nephritis (glomerular and tubular injury, hypertension, renal failure). To discuss chronic radiation effects: carcinogenesis (leukemia, thyroid, breast, lung cancer), teratogenesis, genetic mutations. To examine resuscitation pathology: definition – morphological changes resulting from cardiopulmonary resuscitation (CPR) and intensive care interventions. To analyze complications of CPR: rib fractures (anterior, midclavicular line), sternal fractures, myocardial contusion, cardiac tamponade (from intracardiac injections or catheter injury), pneumothorax, hemothorax, liver and spleen lacerations (especially in children), gastric rupture (from bag-valve-mask ventilation), airway injuries (endotracheal tube complications – mucosal ulceration, tracheal stenosis). To discuss complications of mechanical ventilation: ventilator-associated lung injury (VALI) – barotrauma (pneumothorax, pneumomediastinum, subcutaneous emphysema), volutrauma, atelectotrauma, biotrauma; diffuse alveolar damage, organizing pneumonia. To examine complications of central venous catheterization: pneumothorax, hemothorax, arterial puncture, thrombosis, infection (catheter-related bloodstream infection). To discuss complications of defibrillation: skin burns, myocardial injury. To analyze post-resuscitation changes in the brain: hypoxic-ischemic encephalopathy (laminar necrosis, hippocampal and Purkinje cell loss), brain edema. To discuss the concept of brain death and its pathological correlates.

Instructional Goal:

- Within the framework of the educational process for this academic discipline, the student must acquire not only theoretical knowledge, practical skills and

abilities in the specialty but also develop their personal potential. They should cultivate qualities of responsibility and patriotism, be prepared to actively participate in the economic, socio-cultural, and public life of the country, recognize the social significance of their future professional activity, understand the norms of medical ethics and deontology, and learn to observe academic and labor discipline. In the course of studying the educational material, students should realize the importance of maintaining a healthy lifestyle and, as an example in the future, when performing professional duties, set an example for those around them and their patients.

Objectives: Upon completion of the practical session, the student must:

Know:

- The pathogenesis of radiation injury (direct and indirect DNA damage);
- The effects of radiation on rapidly dividing cells;
- The three subsyndromes of acute radiation syndrome (hematopoietic, gastrointestinal, neurovascular);
- The morphological changes in radiation pneumonitis, dermatitis, and enteritis;
- The complications of CPR (rib fractures, visceral lacerations, gastric rupture);
- The complications of mechanical ventilation (barotrauma, diffuse alveolar damage);
- The morphological features of hypoxic-ischemic encephalopathy.

Be able to:

- Identify radiation-induced changes in tissues (atrophy, fibrosis, atypical fibroblasts, vascular changes);
- Recognize rib fractures and visceral injuries as complications of CPR;
- Diagnose pathological processes and diseases based on the description of macroscopic and microscopic changes in organs and tissues of the body.

Master:

- Basic techniques for working with a microscope;
- Skills in clinical-anatomical analysis;
- The fundamentals of synthetic generalization of morphological diagnostic signs of diseases and their correct interpretation in cause-and-effect relationships.

Motivation for Mastering the Topic:

Radiation injuries are a concern not only in the context of nuclear accidents and occupational exposure but also in therapeutic radiation, which is used to treat millions of cancer patients worldwide. Understanding the pathological effects of radiation is essential for radiation oncologists, radiologists, pathologists, and emergency physicians. Resuscitation pathology is increasingly important as more patients receive CPR and intensive care interventions. Recognizing the complications of these life-saving procedures is critical for clinicians to improve patient safety, refine techniques, and understand the causes of morbidity and mortality in critically ill patients. This knowledge is also essential for forensic pathologists who must distinguish resuscitation-related injuries from inflicted trauma.

QUESTIONS

1. Acute radiation syndrome (ARS). Definition. Etiology. Pathogenesis. Morphology. Clinical manifestations. Complications and outcomes. Causes of death.
2. Chronic radiation syndrome (CRS). Definition. Etiology. Pathogenesis. Morphology. Clinical manifestations. Complications and outcomes. Causes of death.
3. Resuscitation pathology. Definition. Etiology. Classification. Pathogenesis. Morphology. Clinical manifestations. Complications and outcomes. Causes of death.

COURSE OF THE SESSION

Acute radiation syndrome (ARS), also known as radiation sickness, is a complex of clinical and pathological manifestations that develop after total or near-total body exposure to a high dose of ionizing radiation over a short time period. It reflects systemic injury primarily affecting rapidly dividing cells and tissues, particularly of the hematopoietic, gastrointestinal, and neurovascular systems. ARS occurs with absorbed doses typically **exceeding 1 Gray** (Gy), and its severity and progression depend on the dose, duration, and distribution of exposure.

Etiology.

The primary cause of ARS is exposure to **ionizing radiation**—either external (e.g., nuclear reactor accidents, radiological weapons, or therapeutic overdose) or internal (inhalation, ingestion, or wound contamination with radioactive materials). High-dose exposures are most commonly accidental or occupational, although intentional radiation use in terrorism is a potential scenario. Deterministic effects of radiation depend on surpassing specific threshold doses, with ARS occurring only when whole-body or substantial partial-body exposure exceeds those thresholds.

The pathogenesis of ARS involves direct DNA damage and indirect injury through the generation of reactive oxygen species (ROS), leading to widespread cellular dysfunction and apoptosis.

Tissues with high mitotic rates—such as **bone marrow**, **intestinal epithelium**, and **lymphoid organs**—are most vulnerable.

ARS typically progresses through four phases:

1) **Prodromal phase** – Nausea, vomiting, and fatigue appear within hours.

2) **Latent phase** – Apparent clinical improvement as underlying tissue damage progresses.

3) **Manifest illness phase** – Tissue-specific syndromes emerge depending on dose.

4) **Recovery or death** – Determined by the extent of organ damage and supportive care. Syndromes include hematopoietic (1–6 Gy), gastrointestinal (6–30 Gy), and neurovascular (>30 Gy) variants, each with distinct clinical timelines and outcomes.

Morphologic damage in ARS is most evident in highly proliferative tissues:

1) **Bone marrow**: Marked hypocellularity with depletion of myeloid, erythroid, and megakaryocytic precursors; severe lymphoid depletion in nodes and spleen.

2) **Gastrointestinal tract**: Necrosis of crypt epithelium, ulceration, and mucosal denudation, particularly in the small intestine.

3) **Skin**: Radiation dermatitis, with erythema, desquamation, ulceration, and fibrosis depending on dose.

4) **Brain and CNS**: In high-dose exposures, cerebral edema, endothelial injury, petechial hemorrhages, and demyelination may occur, though neural tissue is relatively radioresistant.

Histological features reflect **radiation-induced apoptosis**, **vascular injury**, and **impaired regeneration**.

Symptoms depend on the total absorbed dose and the tissue systems involved:

1) **Hematopoietic syndrome** (1–6 Gy): Onset within days to weeks; features include fatigue, fever, petechiae, anemia, and increased susceptibility to infection due to pancytopenia.

2) **Gastrointestinal syndrome** (6–30 Gy): Severe nausea, vomiting, diarrhea, electrolyte imbalance, and dehydration appear within hours to days. Bacterial translocation may lead to sepsis.

3) **Neurovascular syndrome** (>30 Gy): Rapid onset of confusion, ataxia, seizures, and coma within hours; almost universally fatal due to cerebral edema and vascular collapse.

Prodromal symptoms (nausea, vomiting, anorexia) may precede all syndromes and serve as early indicators of dose severity.

Complications and outcomes.

The primary complications include severe immunosuppression, hemorrhage, fluid and electrolyte disturbances, and multi-organ failure. Hematopoietic suppression predisposes to opportunistic infections and sepsis. Gastrointestinal injury leads to malabsorption and systemic endotoxemia. Survivors of sublethal doses may experience delayed complications, including infertility, chronic anemia, cataracts, and increased risk of malignancies (particularly leukemia and solid tumors). Long-term survivors also face potential fibrosis of irradiated organs and psychosocial impacts.

Causes of death.

Death in ARS results from the failure of one or more vital organ systems. In hematopoietic syndrome, death typically occurs due to infection or hemorrhage within weeks if untreated. In gastrointestinal syndrome, rapid dehydration, sepsis, and electrolyte imbalance lead to mortality within 7–14 days. Neurovascular syndrome causes death within hours to a few days due to catastrophic cerebral damage and cardiovascular collapse. Without aggressive medical and supportive care—including antibiotics, transfusions, and stem cell support—mortality rates are high.

Chronic radiation syndrome (CRS) is a progressive pathological condition resulting from **long-term, continuous or fractionated exposure to ionizing radiation** at relatively **low to moderate doses**, typically over weeks to years.

Unlike acute radiation syndrome (ARS), CRS develops insidiously and manifests with **cumulative**, **multisystem deterioration**—most notably involving the hematopoietic, nervous, endocrine, and cardiovascular systems.

CRS is considered a deterministic effect of radiation and has been most thoroughly described in occupational settings, particularly among nuclear industry workers and individuals exposed during early radiological accidents.

CRS: etiopathogenesis

CRS occurs as a result of chronic total-body or large-area exposure to ionizing radiation at doses **exceeding 0.1 Gy/year**, with a **cumulative dose typically above 1–3 Gy**. Etiologic factors include **occupational exposure** (e.g., radiologic technicians, nuclear reactor personnel, uranium miners), **environmental exposure**

(e.g., contaminated zones following nuclear incidents), and **therapeutic exposure** (e.g., radiation treatment with extensive field coverage). Poor radiological protection, equipment malfunction, or disregard for safety protocols are contributing factors. Genetic susceptibility may also modulate individual response to chronic radiation.

The **pathogenesis** of CRS is multifactorial and cumulative. Repeated or continuous radiation exposure results in persistent **low-level DNA damage**, **oxidative stress**, and **disruption of cellular homeostasis**. This leads to progressive injury of radiosensitive tissues, such as bone marrow, lymphoid organs, gonads, and vascular endothelium. The chronic nature of exposure allows for partial tissue repair, but eventual exhaustion of regenerative capacity and accumulation of genetic mutations contribute to dysfunction. Endocrine dysregulation, immune suppression, and neurodegeneration are key mechanisms in later stages.

Histological changes in CRS are subtle initially but become more pronounced over time.

1) **Hematopoietic system**: Progressive bone marrow hypocellularity with fatty replacement; lymphoid depletion in spleen and lymph nodes.

2) **Nervous system**: Neuronal loss, gliosis, and demyelination in the brain and spinal cord; cerebral atrophy may be evident on imaging.

3) **Endocrine glands**: Atrophy and fibrosis of the thyroid, adrenal, and gonadal tissues.

4) **Cardiovascular system**: Radiation-induced vasculitis, endothelial damage, and adventitial fibrosis contribute to accelerated atherosclerosis and microvascular ischemia.

5) **Skin and mucosa**: Atrophic changes, pigmentary disturbances, telangiectasia, and dermal fibrosis may develop in exposed regions.

CRS presents gradually and is often misdiagnosed in early stages. Common initial symptoms include **chronic fatigue**, **irritability**, **memory impairment**, **sleep disturbances**, and **decreased work capacity**. As the syndrome progresses, patients may exhibit **anemia**, **leukopenia**, and **thrombocytopenia**, leading to increased susceptibility to infections and bleeding. Neurologic signs such as **polyneuropathy**, cognitive decline, and motor dysfunction may emerge. Endocrine manifestations include **infertility**, **hypothyroidism**, and **adrenal insufficiency**. Cutaneous signs (dryness, hyperkeratosis) and cardiovascular symptoms (hypertension, ischemia) may also be observed in advanced cases.

CRS is a chronic, debilitating condition with progressive decline in functional capacity. **Complications** include immunodeficiency with recurrent infections, endocrinopathies, neurodegenerative changes, and chronic cardiovascular disease. A significant long-term consequence is radiation-induced carcinogenesis, with increased risk of leukemia, thyroid cancer, breast cancer, and solid tumors in various organs. Quality of life is significantly reduced due to chronic pain, fatigue, and neurologic impairment. While progression may stabilize in some individuals if exposure ceases, irreversible damage is common in moderate to severe cases.

Death in CRS typically results from one or more of the following:

1) **Bone marrow failure**, leading to hemorrhage or overwhelming infection.

2) **Secondary malignancies**, particularly hematologic cancers or radiation-induced sarcomas.

3) **Cardiovascular complications**, such as myocardial infarction or stroke secondary to radiation vasculopathy.

4) **Progressive neurologic decline**, contributing to debilitation and secondary complications such as aspiration or immobility-related infections.

With modern radioprotection standards, CRS is rare, but in historical cohorts it has been associated with significant morbidity and mortality over years or decades.

Resuscitation pathology is a specialized domain within forensic and anatomic pathology that examines the morphological changes resulting from cardiopulmonary resuscitation and subsequent intensive care interventions. This field critically distinguishes between lethal pathological changes attributable to the underlying disease process, reversible changes arising from agonal events, and iatrogenic injuries caused by medical attempts to restore circulation and ventilation. Understanding these findings is paramount for improving CPR techniques, managing medicolegal issues, and refining post-resuscitation care protocols.

Complications of cardiopulmonary resuscitation

The mechanical act of external chest compressions frequently produces skeletal injuries, with rib fractures being the most common complication and occurring in thirty to seventy percent of adults, with a notably higher incidence in elderly individuals with osteoporotic bone. Anterior rib fractures typically occur at the costochondral junction or mid-anterior arch due to focal pressure, while fractures along the midclavicular line commonly arise at the weakest point of the rib curvature and are often bilateral. Sternal fractures occur in approximately twenty percent of cases, usually presenting as transverse fractures of the sternal body, and the risk increases with compression depths exceeding six centimeters as well as with the use of mechanical CPR devices.

Beyond skeletal trauma, cardiothoracic soft tissue injuries are frequently encountered. Myocardial contusion is characterized grossly by epicardial or intramyocardial ecchymoses, most commonly involving the right ventricle, while histologically it manifests as contraction band necrosis, which is distinct from ischemic coagulative necrosis, along with myocyte disruption and intramyocardial hemorrhage. Cardiac tamponade, though rare, represents a lethal complication and may result either from intracardiac injections leaving a needle tract through the right ventricle leading to hemopericardium or from central line placement where a guidewire perforates the right atrium or ventricle, causing delayed tamponade. Pulmonary and pleural injuries include pneumothorax from rib fracture fragments lacerating the lung parenchyma or from barotrauma, as well as hemothorax typically due to laceration of intercostal vessels or the internal mammary artery secondary to fractured ribs.

Abdominal visceral injuries occur with increased frequency in children due to their more pliable thoracic cage and the subdiaphragmatic position of the liver and spleen. Liver lacerations represent the most common abdominal injury, typically

involving the right lobe from compression against the vertebral column, and can cause massive hemoperitoneum. Spleen lacerations are less common but remain clinically significant. Gastric rupture is a classic complication of bag-valve-mask ventilation, especially when air enters the stomach due to gastric insufflation from high peak airway pressures or esophageal intubation; the rupture is typically along the greater curvature, leading to chemical peritonitis and pneumoperitoneum.

Airway injuries related to endotracheal tube placement include mucosal ulceration from cuff overinflation or prolonged intubation, typically seen over the posterior glottis or at the cuff site, and these ulcers may progress to granulation tissue formation. Tracheal stenosis is a late complication occurring weeks to months after intubation, presenting as fibrotic narrowing at the cuff or stoma site, with histology showing collagen deposition, chronic inflammation, and cartilage destruction.

Complications of mechanical ventilation and ventilator-associated lung injury

Mechanical ventilation, while lifesaving, can paradoxically exacerbate or initiate lung injury through several mechanisms collectively termed ventilator-associated lung injury. Barotrauma results from injury caused by elevated airway pressure and manifests as pneumothorax from air entering the pleural space via a ruptured alveolus, pneumomediastinum from air dissecting along bronchovascular sheaths into the mediastinum, and subcutaneous emphysema from air dissecting from the mediastinum into the soft tissues of the neck, chest, and face. Volutrauma refers to injury from excessive tidal volume, causing overdistension of alveoli that leads to capillary leak, hyaline membrane formation, and surfactant dysfunction; grossly, the lungs appear heavy and edematous. Atelectotrauma is the injury resulting from repetitive opening and collapse of alveoli, generating shear stress on the alveolar epithelium that promotes inflammation and fibrosis. Biotrauma describes the inflammatory injury whereby mechanical stretch triggers the release of pro-inflammatory cytokines such as interleukin-six, interleukin-eight, and tumor necrosis factor-alpha into the bloodstream, potentially leading to multiorgan failure through the so-called two-hit model.

The histopathological correlates of ventilator-associated lung injury include diffuse alveolar damage, which is the hallmark of acute respiratory distress syndrome and severe ventilator-associated lung injury. In the exudative phase occurring during the first seven days, one finds hyaline membranes lining the alveolar ducts, interstitial and alveolar edema, and type two pneumocyte necrosis. In the proliferative phase occurring between days seven and twenty-one, there is organization of the exudates, type two pneumocyte hyperplasia, and fibroblast proliferation. Organizing pneumonia, also known as bronchiolitis obliterans organizing pneumonia, presents with granulation tissue plugs within alveolar ducts and bronchioles; this pattern is less severe than diffuse alveolar damage but can cause persistent hypoxemia.

Complications of central venous catheterization

Central venous catheterization is associated with a range of mechanical, infectious, and thrombotic complications. Among mechanical complications, pneumothorax is the most common, occurring in one to three percent of subclavian approaches and resulting from needle puncture of the lung apex. Hemothorax arises from laceration of the subclavian artery or vein, or from cardiac perforation. Arterial puncture involves accidental cannulation of the carotid or subclavian artery and carries the risk of hematoma, pseudoaneurysm, or stroke if air or thrombus embolizes.

Infectious complications are dominated by catheter-related bloodstream infection, typically caused by skin flora such as Staphylococcus epidermidis, Staphylococcus aureus, or Candida species. Pathologically, this manifests as thrombus formation on the catheter tip with bacterial biofilm, and septic emboli may be found in the lungs, spleen, or kidneys. Thrombotic complications include deep vein thrombosis of central veins, with formation of a fibrin sheath or mural thrombus that carries the risk of pulmonary embolism or superior vena cava syndrome.

Complications of defibrillation

Defibrillation produces characteristic skin burns resulting from electrical current causing thermal injury; these burns are typically circular and range from erythematous to full-thickness injuries at the pad or paddle sites, and the edge phenomenon, whereby higher current density occurs at the edges, may produce ring-shaped burns. Myocardial injury from defibrillation is histologically characterized by contraction band necrosis and coagulative necrosis, especially after multiple high-energy shocks exceeding three hundred sixty joules biphasic equivalent; although this injury can elevate cardiac biomarkers, it is often clinically overshadowed by the cardiac arrest itself.

Post-resuscitation changes in the brain

Following the return of spontaneous circulation, a secondary injury cascade occurs in the brain, leading to hypoxic-ischemic encephalopathy characterized by selective neuronal necrosis. Certain neuronal populations are exquisitely sensitive to hypoxia, including the hippocampal neurons in the CA1 sector, also known as Sommer's sector, the loss of which correlates with memory deficits. Purkinje cell loss in the cerebellum leads to ataxia and dysmetria. Laminar necrosis of the cerebral cortex involves necrosis of cortical layers three, five, and six, and grossly appears as a pseudolaminar band of pallor or yellow-brown discoloration. Histologically, one finds eosinophilic or red neurons, pyknosis, karyorrhexis, and microglial activation forming microglial nodules.

Brain edema following resuscitation occurs in two phases. Cytotoxic edema appears early and results from swelling of astrocytes and neurons due to failed sodium-potassium ATPase pumps; grossly it manifests as narrowed ventricles and sulci, while histologically one observes perineuronal and perivascular clearing that represents artifactual spaces. Vasogenic edema appears later and results from breakdown of the blood-brain barrier; grossly the brain is heavy and soft with flattened gyri.

The concept of brain death and its pathological correlates

Brain death is defined as the irreversible cessation of all functions of the entire brain, including the brainstem. Clinically, this is characterized by coma, absence of brainstem reflexes, and apnea. The pathological correlate of brain death, often termed the respirator brain, presents grossly as a globally swollen, soft, and autolytic brain that eventually undergoes liquefactive necrosis, whereby the parenchyma becomes a viscous, grey-pink paste, and the cerebellum and brainstem may herniate or fragment. Histologically, one observes coagulative necrosis of all cellular elements including neurons, glia, and endothelia, with loss of nuclear basophilia and a conspicuous absence of inflammation due to the lack of blood flow preventing leukocyte recruitment. Post-mortem angiography confirms the absence of cerebral blood flow as a confirmatory test in some jurisdictions. The medicolegal significance of brain death is profound, as brain death is equivalent to cardiopulmonary death by statute in most countries, and pathological examination after organ donation or at autopsy confirms the irreversibility of the damage.

INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

1. Pathomorphology : textbook / ed. by I.V. Sorokina, V.D. Markovskyi, D.I. Halata. - 2nd ed. - Kyiv : AUS Medicine Publishing, 2020. - 327 p., [10] col. inserts : tab.

2. Klatt, E. C. Robbins and Cotran Atlas of Pathology : [with Student Consult Online Access] / Edward C. Klatt. - third edition. - Philadelphia : Elsevier, 2015. - 587 p. : il.

3. Robbins Basic pathology / [ed. by] Vinay Kumar, Abul K. Abbas, Jon C. Aster. - 9th ed. - [Philadelphia] : Elsevier ; Saunders. - 910 p. : ill., scheme, tab.

ADDITIONAL LITERATURE

1. Sobotta. Atlas of human anatomy. [V. 1]. General anatomy and musculoskeletal system / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 400 p. : ill., col. ill.

2. Sobotta. Atlas of human anatomy. [V. 2]. Internal organs / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 259 p. : ill., col. ill.

3. Sobotta. Atlas of human anatomy. [V. 3]. Head, neck, and neuroanatomy / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 370 p. : ill., col. ill.

4. Weir & Abrahams' imaging atlas of human anatomy / ed.: J. D. Spratt [and oth.] ; consultant eds.: J. Weir, P. H. Abrahams. – 5th ed. - [S.l.] : Elsevier, 2017. - xvi, 263 p. : ill.

NORMATIVE LEGAL ACTS AND DOCUMENTS

1. Order of the Ministry of Health of the Republic of Belarus No. 111 of 06/01/1993 "On further improvement of the pathological service of the Republic of Belarus"

Access mode: http://patan.by/menyu/administrativnyie-proczeduryi.html - The date fccess: 20.03.2022.

ELECTRONIC DATABASES

1. Physician's consultant. Electronic Medical Library = Consultant of the doctor. Electronic medical library [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". – Access mode: http://www.rosmedlib.ru/ . – Access date: 03/15/2022.

2. Springer Link [Electronic resource] / Springer International Publishing AG. – Access mode: https:link.springer.com. – Date of access: 03/15/2022.