

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

Department of Pathological Anatomy

Author:

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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: “Pathology of pregnancy”

Duration: 3 hours

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

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 pregnancy-related pathology. To analyze placental pathology: normal placenta morphology, placental abnormalities (circumvallate placenta, battledore placenta, velamentous insertion of cord, placenta accreta/increta/percreta – abnormal implantation). To examine gestational trophoblastic disease: hydatidiform mole (complete mole – 46,XX androgenetic, no fetal tissue, marked trophoblastic proliferation, hydropic villi; partial mole – triploid, fetal tissue present, focal hydropic change), invasive mole (myometrial invasion), choriocarcinoma (malignant trophoblastic tumor, no chorionic villi, biphasic pattern of cytotrophoblast and syncytiotrophoblast, hemorrhage, necrosis, high hCG, metastatic potential), placental site trophoblastic tumor (PSTT). To analyze hypertensive disorders of pregnancy: preeclampsia (hypertension, proteinuria, edema after 20 weeks), eclampsia (preeclampsia with seizures), HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets). Pathological findings: acute atherosclerosis (fibrinoid necrosis of decidual arterioles), placental infarction, glomerular endotheliosis (kidney). To examine obstetric hemorrhage: abruptio placentae (premature separation of placenta – retroplacental hematoma, Couvelaire uterus), placenta previa (placenta overlies cervical os). To analyze infections in pregnancy: chorioamnionitis (inflammation of membranes, funisitis – inflammation of umbilical cord), congenital infections (TORCH – see Topic 31). To examine complications of labor and delivery: uterine rupture, amniotic fluid embolism (sudden dyspnea, hypotension, coagulopathy – squamous cells, lanugo, mucin in pulmonary vessels), retained products of conception. To discuss postpartum pathology: postpartum hemorrhage, puerperal sepsis (endometritis, parametritis, pelvic thrombophlebitis). To analyze miscarriage (spontaneous abortion): causes (chromosomal abnormalities, anatomical, endocrine, immunological, infectious), types (threatened, inevitable, incomplete, complete, missed, septic). To discuss ectopic pregnancy (most common in fallopian tube – tubal pregnancy; presentation: abdominal pain, vaginal bleeding, rupture, hemoperitoneum, shock). To examine gestational diabetes mellitus (GDM) and its effects on the fetus (macrosomia, neonatal hypoglycemia, stillbirth).

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 morphology of hydatidiform mole (complete vs. partial) and choriocarcinoma;
- The pathology of preeclampsia and eclampsia (acute atherosclerosis, glomerular endotheliosis);
- The causes and morphological findings in abruptio placentae and placenta previa;
- The pathology of amniotic fluid embolism;
- The morphology of ectopic pregnancy and its complications;
- The classification and causes of spontaneous abortion.

Be able to:

- Identify hydropic villi and trophoblastic proliferation in hydatidiform mole;
- Recognize the biphasic pattern of choriocarcinoma;
- Diagnose pathological processes and diseases based on the description of macroscopic and microscopic changes in organs and tissues of the body;
- Distinguish complete from partial mole based on histological features.

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:

Pregnancy-related pathology encompasses a range of conditions that can threaten the life of both mother and fetus. Hypertensive disorders of pregnancy (preeclampsia, eclampsia) are leading causes of maternal mortality worldwide. Gestational trophoblastic disease, particularly choriocarcinoma, is a highly curable malignancy but requires prompt diagnosis and treatment. Obstetric emergencies such as abruptio placentae, placenta previa, amniotic fluid embolism, and ectopic pregnancy rupture require rapid recognition and intervention. Understanding the pathological basis of these conditions is essential for obstetricians, pathologists, emergency physicians, and maternal-fetal medicine specialists.

QUESTIONS

1. Ectopic pregnancy. Definition. Etiology. Pathogenesis. Morphology. Clinical manifestations. Complications and outcomes.

2. Hydatidiform mole (molar pregnancy). Definition. Etiology. Pathogenesis. Morphology. Clinical manifestations. Complications and outcomes.
3. Hypertensive disorders of pregnancy (gestational nephropathy, preeclampsia, eclampsia). Definition. Etiology. Pathogenesis. Morphology (placental and renal changes). Clinical manifestations. Complications and outcomes.
4. Placental pathology. Definition. Etiology. Pathogenesis.
5. Placental insufficiency: primary vs. secondary, acute vs. chronic.
6. Placental inflammation: morphology based on infection route, effects on the fetus. Clinical manifestations.

COURSE OF THE SESSION

Ectopic pregnancy is a potentially life-threatening condition in which a fertilized egg implants outside the uterus, typically in the fallopian tube. The main etiology of ectopic pregnancy is **damage to or obstruction of the fallopian tubes**, which can be caused by previous **pelvic infections**, **pelvic surgery**, or **endometriosis**. In addition, certain risk factors increase the likelihood of ectopic pregnancy, including advanced maternal age, smoking, and use of assisted reproductive technologies. Women who have undergone **tubal sterilization** or have a **history of ectopic pregnancy** are also at higher risk.

The epidemiology of ectopic pregnancy varies by region and population, with reported incidence rates ranging from **1% to 2% of all pregnancies** in developed countries. Ectopic pregnancy is more common in women aged 35 to 44 years and in women with a history of infertility. It is also more prevalent in certain populations, such as women with sexually transmitted infections or who have had pelvic inflammatory disease.

The most common symptom is **abdominal pain**, which may be localized or diffuse and can vary in severity. The pain is often described as **sharp** or **stabbing** and may be accompanied by **vaginal bleeding** or spotting. In some cases, the pain may radiate to the shoulder or neck due to irritation of the phrenic nerve.

Other clinical manifestations of ectopic pregnancy may include nausea, vomiting, dizziness, and weakness. Women may also experience symptoms of **shock**, such as rapid heartbeat, low blood pressure, and cold, clammy skin.

Prompt diagnosis and management are critical to prevent potential complications such as **rupture of the fallopian tube and hemorrhage**.

In early ectopic pregnancies, **histopathologic examination** reveals the presence of **chorionic villi** and **syncytiotrophoblasts** in the tubal wall or other ectopic sites.

As the pregnancy progresses, trophoblast invasion into the surrounding tissue can cause **hemorrhage and necrosis**, leading to the formation of a **gestational sac**. The gestational sac may contain embryonic tissue, but this is not always present.

In cases of **tubal rupture or hemorrhage**, histopathologic examination may reveal evidence of **acute inflammation** and **tissue necrosis**, as well as hemorrhage into the surrounding tissue. Chronic inflammation and fibrosis may also be present in cases of recurrent ectopic pregnancy or long-standing ectopic pregnancy that is not promptly diagnosed and managed.

A hydatidiform mole, also known as a **molar pregnancy**, is a rare type of pregnancy where the fertilized egg develops into a **nonviable mass of abnormal placental tissue**. Molar pregnancies are caused by an imbalance in genetic material in the pregnancy. This usually occurs when an egg that contains no genetic information is fertilized by a sperm (a **complete molar pregnancy**), or when a normal egg is fertilized by two sperm (a **partial molar pregnancy**), resulting in triploid (3 sets of chromosomes) genetic material.

Hydatidiform mole is a relatively rare condition, occurring in approximately **1 in every 1,000 to 2,000 pregnancies worldwide**. Risk factors for hydatidiform mole include a history of previous molar pregnancy, a history of miscarriage, and maternal age over 35.

Hydatidiform mole is more common in **Southeast Asia, Central and South America, and the Middle East**. The incidence of hydatidiform mole may also be increased in populations with a high prevalence of consanguineous marriages or other genetic disorders, and in women who have undergone assisted reproductive technologies such as **in vitro fertilization (IVF)**.

The clinical presentation of hydatidiform mole can vary, but most women experience **vaginal bleeding** and cramping **in early pregnancy**. Other symptoms may include nausea, vomiting, and **hyperemesis gravidarum** (severe nausea and vomiting during pregnancy).

On physical examination, the uterus may be larger than expected for the gestational age, and there may be a palpable mass within the uterus. In cases where there is significant bleeding or fluid accumulation within the uterus, abdominal distention and discomfort may be present.

Ultrasound is the primary diagnostic tool for identifying a hydatidiform mole. On ultrasound, a hydatidiform mole appears as a **grape-like cluster of cystic structures** within the uterus, with **no visible fetal parts**. The absence of a fetal heartbeat is also a key finding on ultrasound. Blood tests may also be performed to measure levels of pregnancy hormones such as **human chorionic gonadotropin (hCG)**, which are typically elevated.

Morphologically on **gross examination**, a hydatidiform mole appears as a mass of grape-like vesicles within the uterine cavity.

Microscopically, these vesicles are lined by **proliferating trophoblastic cells** that form delicate branching villi. The villi are typically smaller and more crowded than those in a normal pregnancy, and may show hyperplasia or atypia. In some cases, there may be diffuse or focal necrosis of the villi, leading to the formation of hyalinized material within the vesicles.

In a **partial mole**, there may be some evidence of fetal tissue, such as a small fetal sac or recognizable fetal parts. However, the fetal tissue is typically abnormal and may show growth restriction or anomalies. In both complete and partial moles, there is an increased risk of developing **gestational trophoblastic neoplasia**.

Hypertensive disorders of pregnancy (HDP) encompass a group of conditions characterized by elevated blood pressure arising during gestation.

These include **gestational hypertension, preeclampsia, eclampsia, and chronic hypertension with superimposed preeclampsia**.

Preeclampsia is a multisystem disorder that typically develops after 20 weeks of gestation, characterized by new-onset hypertension ($\geq 140/90$ mmHg) and proteinuria (≥ 300 mg/day), or in its absence, other signs of end-organ dysfunction.

Eclampsia refers to the occurrence of tonic-clonic seizures in a woman with preeclampsia, in the absence of other neurologic causes.

Gestational nephropathy, often used synonymously with preeclampsia, highlights the renal involvement central to disease progression.

Etiology.

The exact cause of preeclampsia and related hypertensive disorders remains incompletely understood, though several risk factors are well recognized. These include nulliparity, advanced maternal age, multiple gestation, obesity, preexisting hypertension, diabetes mellitus, and autoimmune disorders such as antiphospholipid syndrome. Genetic predisposition and immunological maladaptation at the maternal-fetal interface also contribute. An abnormal maternal response to trophoblast invasion of the spiral arteries is considered a central initiating event.

Pathogenesis.

Preeclampsia is conceptualized as a two-stage disorder. The **first stage involves abnormal placentation**: defective trophoblastic invasion and failure of spiral artery remodeling lead to inadequate uteroplacental perfusion. The **second stage entails the release of antiangiogenic factors** (e.g., soluble fms-like tyrosine kinase-1, sFlt-1) and inflammatory cytokines into the maternal circulation, resulting in widespread endothelial dysfunction, vasospasm, increased vascular permeability, and activation of the coagulation cascade. These changes underlie the clinical features of hypertension, proteinuria, edema, and multiorgan involvement.

Morphology: placental changes.

The placenta in preeclampsia typically shows signs of **hypoperfusion** and **ischemic damage**. Grossly, it is often smaller than normal and may exhibit **areas of infarction**, **retroplacental hemorrhages**, and **increased fibrin deposition**. Microscopically, features include accelerated villous maturation, distal villous hypoplasia, and increased syncytial knotting. Uteroplacental arteries demonstrate fibrinoid necrosis, thrombosis, and lack of physiologic transformation, reflecting persistent muscular and elastic layers instead of low-resistance vessels.

Morphology: renal changes.

The hallmark renal lesion in preeclampsia is **glomerular endotheliosis**, characterized by swelling of glomerular endothelial cells, narrowing or occlusion of capillary lumina, and subendothelial proteinaceous deposits. This noninflammatory lesion impairs glomerular filtration, resulting in **proteinuria**. Tubular changes are less prominent but may include ischemic injury in severe cases. The renal pathology closely correlates with clinical severity and may persist postpartum in some cases.

Clinical manifestations.

Preeclampsia often begins with nonspecific symptoms such as headache, visual disturbances, and epigastric or right upper quadrant pain. **Edema**, particularly of the face and hands, may be prominent. **Hypertension** and **proteinuria** are diagnostic hallmarks, though other signs such as thrombocytopenia, elevated liver enzymes, and renal insufficiency indicate severe disease. Eclampsia is heralded by **seizures**, which may be preceded by prodromal symptoms or occur suddenly. The clinical spectrum ranges from mild hypertension to life-threatening multiorgan failure.

Maternal complications include abruptio placentae, acute renal failure, pulmonary edema, stroke, HELLP syndrome (Hemolysis, Elevated Liver enzymes,

Low Platelets), and DIC. Eclampsia carries a significant risk of intracerebral hemorrhage and death. The risk of long-term cardiovascular disease, including chronic hypertension and ischemic heart disease, is increased in women with a history of preeclampsia. Recurrent preeclampsia in subsequent pregnancies is also common.

Fetal complications stem primarily from placental insufficiency. These include intrauterine growth restriction (IUGR), oligohydramnios, prematurity, and increased perinatal mortality. Placental abruption may cause fetal hypoxia or death. Neonates born to mothers with severe preeclampsia or eclampsia are at elevated risk for respiratory distress, intracranial hemorrhage, and long-term neurodevelopmental impairments.

Outcomes and prognosis.

Delivery remains the definitive treatment for preeclampsia and eclampsia. In mild cases, expectant management with close monitoring may be appropriate before term. Severe preeclampsia typically requires hospitalization, magnesium sulfate for seizure prophylaxis, and antihypertensive therapy. Maternal and fetal outcomes have improved with early diagnosis and multidisciplinary care, but significant morbidity and mortality persist, particularly in resource-limited settings. Postpartum resolution is expected within weeks, but long-term follow-up is essential due to increased lifetime cardiovascular risk.

Congenital malformations of the placenta

Congenital anomalies of the placenta, amnion, and umbilical cord are structural abnormalities present from early embryogenesis, often discovered during prenatal imaging, delivery, or histological examination.

These malformations can interfere with normal fetal development and pregnancy outcomes. While some are incidental findings with minimal clinical impact, others may cause significant complications including fetal growth restriction, intrauterine hypoxia, preterm birth, or stillbirth.

Key examples include **placenta succenturiata**, **placenta membranacea**, **velamentous cord insertion**, **single umbilical artery**, **amniotic bands**, and **circumvallate placenta**.

The **etiologic factors** underlying these anomalies are multifactorial, including genetic predispositions, teratogenic exposures, vascular accidents, and abnormal implantation dynamics. Maternal conditions such as diabetes, uterine malformations, or multiple gestation can also predispose to abnormal placental development. Chromosomal anomalies in the fetus, such as trisomies, are frequently associated with umbilical cord and amniotic malformations. Environmental insults during early gestation (e.g., infections, hypoxia, or trauma) may disrupt morphogenesis of the fetal membranes and supporting structures.

Most congenital placental and cord malformations originate from **disruptions during early implantation or differentiation of the chorionic villi and extraembryonic mesoderm**. Abnormal trophoblastic invasion, altered vascularization, or improper folding and fusion of the amnion and chorion can result in structural anomalies. For instance, velamentous insertion arises from failure of

the umbilical cord to insert centrally, leading to exposed vessels traversing the membranes. In cases such as amniotic band syndrome, early rupture of the amnion allows fibrous strands to entangle fetal parts, restricting development or causing amputations.

Congenital *placental malformations* include accessory lobes (placenta succenturiata), bilobed placenta, and circumvallate placenta. In **placenta succenturiata**, one or more accessory lobes are connected by vessels that are prone to rupture. **Circumvallate placenta** features a thickened, elevated margin due to inward folding of the membranes, often associated with perivillous fibrin deposition and infarctions. **Placenta membranacea** involves persistence of the chorion over a wide area, leading to diffuse villous development over the fetal membranes. Placental anomalies may be accompanied by irregular villous branching or avascular villi in microscopic sections.

Amniotic malformations include amniotic bands and absence of the amniotic sac. **Amniotic band** syndrome is characterized by fibrous strands arising from amnion rupture that constrict or amputate fetal limbs and digits. **Umbilical cord anomalies** include abnormal length (too short or long), coiling defects, true or false knots, and vessel anomalies such as single umbilical artery. Velamentous cord insertion, where vessels traverse the membranes without Wharton's jelly, increases the risk of vessel rupture. Marginal or furcate insertions also predispose to circulatory compromise. Microscopically, umbilical cord anomalies may show hypoplastic vessels, thrombi, or fibrosis.

Many of these malformations are asymptomatic and identified only incidentally. However, in clinically significant cases, manifestations may include **abnormal fetal heart rate patterns**, intrauterine growth restriction (**IUGR**), polyhydramnios or oligohydramnios, vaginal bleeding, or preterm labor. Velamentous insertion can cause fetal distress or bleeding during labor if unprotected vessels rupture (vasa previa). Amniotic band syndrome typically presents with fetal deformities, such as limb constrictions, clefts, or amputations, often detectable by prenatal ultrasonography.

Maternal complications are generally secondary to poor placental attachment or hemorrhagic events. Accessory lobes or succenturiate placenta increase the risk of retained placental tissue and postpartum hemorrhage. Circumvallate placenta may be associated with preterm delivery, abruption, or miscarriage. Fetal complications include IUGR, fetal hypoxia, congenital anomalies, and stillbirth. True knots and cord entanglements may result in intermittent or sustained compromise of fetal circulation. Single umbilical artery is often associated with other congenital malformations, particularly of the cardiovascular and renal systems.

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Placental pathology encompasses a broad spectrum of structural, inflammatory, vascular, and degenerative changes affecting the placenta during pregnancy.

These abnormalities may **compromise its essential functions** of nutrient exchange, hormonal support, and immunologic protection.

Placental pathology is a major contributor to adverse pregnancy outcomes, including **fetal growth restriction, preterm birth, and stillbirth.**

Examination of the placenta provides crucial insight into the pathophysiology of various maternal and fetal conditions and serves as a bridge between prenatal events and postnatal outcomes.

The causes of placental pathology are diverse and include maternal, fetal, and environmental factors. **Maternal contributors** include preeclampsia, diabetes, thrombophilias, infections, and chronic hypertension. **Fetal factors** include chromosomal abnormalities and intrauterine infections. **Environmental influences**—such as smoking, drug use, and uterine anomalies—can also disrupt placental development. In many cases, the etiology is multifactorial, with abnormal implantation or vascular adaptation at the uterine-placental interface playing a central role.

Placental pathology often begins with **abnormal trophoblastic invasion** and failure of the physiologic transformation of spiral arteries, leading to **inadequate uteroplacental perfusion.** This hypoperfusion results in oxidative stress, villous injury, and compensatory or maladaptive changes such as infarction, fibrosis, or increased syncytial knotting. Inflammatory processes may be triggered by **ascending or hematogenous infections,** while thrombotic or ischemic injury arises from maternal vascular disease or **immunologic dysfunction.** The balance between maternal-fetal tolerance and immune activation is a key determinant of placental health.

Placental insufficiency refers to a state in which the placenta fails to meet the metabolic demands of the growing fetus. **Primary insufficiency** results from intrinsic defects in placental development, such as abnormal trophoblast differentiation, deficient villous branching, or genetic anomalies of the chorionic villi. It is often seen in early-onset growth restriction. **Secondary insufficiency** occurs when a previously normal placenta becomes compromised due to maternal

or fetal conditions, including hypertension, diabetes, or intrauterine infection. Secondary insufficiency is more common and typically develops later in pregnancy.

Chronic placental insufficiency is characterized by progressive reduction in perfusion and exchange capacity, leading to intrauterine growth restriction (IUGR), oligohydramnios, and fetal hypoxia. Morphologic changes include villous infarctions, distal villous hypoplasia, increased syncytial knots, and avascular villi. In contrast, **acute placental insufficiency** presents suddenly and may result from events such as placental abruption, massive intervillous thrombosis, or umbilical cord accident. These events can cause abrupt cessation of oxygen delivery, resulting in acute fetal distress or intrauterine death.

Infectious placental inflammation (placentitis) varies by the route of microbial entry. **Ascending infections** from the lower genital tract typically cause chorioamnionitis and funisitis. Histologically, this is seen as acute neutrophilic infiltrates in the chorion, amnion, and umbilical cord. **Hematogenous infections**, such as those caused by TORCH pathogens (e.g., *Toxoplasma gondii*, *Rubella*, *Cytomegalovirus*, *Herpes simplex*), affect the villous core, causing chronic villitis, intervillitis, or necrosis. Viral inclusions, plasma cell infiltrates, and calcifications may be observed. Chronic placental inflammation is often patchy and may reflect maternal-fetal immune dysregulation in the absence of identifiable organisms.

Placental inflammation—particularly of infectious origin—can result in vertical transmission and cause **fetal sepsis, congenital malformations, preterm labor, and stillbirth**. *Acute chorioamnionitis* is strongly associated with preterm premature rupture of membranes (PPROM) and preterm delivery. *Chronic villitis*, even when non-infectious (villitis of unknown etiology, VUE), is linked to IUGR and recurrent pregnancy loss. Intervillitis can impair nutrient exchange and has been associated with fetal demise. Inflammatory cytokines and microbial products can also affect fetal brain development, contributing to long-term neurologic sequelae.

Clinical manifestations.

Placental pathology often manifests indirectly through maternal or fetal complications. Clinical signs include decreased fetal movements, abnormal fetal heart rate patterns, IUGR, preeclampsia, and antepartum hemorrhage.

Infections may present with maternal fever, uterine tenderness, or foul-smelling amniotic fluid.

Ultrasound findings suggestive of placental pathology include abnormal placental thickness, echogenic foci, or reduced blood flow on Doppler studies.

Definitive diagnosis is often made postnatally through histological examination of the placenta, which remains critical in explaining adverse outcomes and guiding future pregnancy management.

INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

1. Pathomorphology : textbook / ed. by I.V. Sorokina, V.D. Markovskiy, 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.