REPUBLIC OF UZBEKISTAN
MINISTRY OF HIGHER EDUCATION, SCIENCE AND INNOVATION

SAMARKAND STATE UNIVERSITY OF VETERINARY MEDICINE, ANIMAL HUSBANDRY AND BIOTECHNOLOGY

" EPIZOOTOLOGY AND INFECTIOUS DISEASES "
DEPARTMENT

"IAPPROVE"
Vice-rector for educational affairs, d.of a.s.
professor _____ A. A. Elmurodov
“ ___ ” _____ 2022 years

" EPIZOOTOLOGY AND INFECTIOUS DISEASES "
ON SCIENCE

EDUCATIONAL METHODOLOGY COMPLEX

Field of knowledge: 400000 - Agriculture and water management

of study : 440000-Veterinary

Course of Study: 5440100-Veterinary Medicine
(by type of activity)

Samarkand - 2022

The educational and methodological complex of the subject was developed in accordance with the approved curriculum, working curriculum, curriculum and working curriculum .

Developers:

Salimov X.S. - " Epizootology and infectious diseases "
professor of the department , doctor of veterinary sciences

Kambarov A.A. - " Epizootology and infectious diseases "
associate professor of the department , candidate of veterinary sciences

Allazov A.S. - " Epizootology and infectious diseases "
department, assistant

Klichov O.I. - " Epizootology and infectious diseases "
department, assistant

" EPIZOOTOLOGY AND INFECTIOUS DISEASES "
SCIENCE

CURRICULUM COMPLEX :

The working curriculum of the subject " Epizootology and Infectious Diseases " department was discussed at the meeting No. __ on "__" _____, 2022 and was recommended for discussion at the faculty council.

Head of the department, candidate of veterinary sciences _____ Z.E.Ruziyev

The working curriculum of the subject was discussed and recommended for use at the Council of the "Veterinary Prevention and Treatment" faculty (2022 "__" _____ No. __ - protocol number).

Chairman of the faculty council, professor _____ X.B.Niyazov

Agreed:
Head of the educational and methodological department,
associate professor _____R.F.Ruzikulov

TABLE OF CONTENTS

Science curriculum

Working curriculum of science

The main educational materials of science :

SUBJECT: General epizootology . Infection and infectious diseases

Lecture	**General epizootology . Infection and infectious diseases**
Lecture teaching technology	
Study time: 2 hours	of students: 90 people
The form of training	Enter. Visual lecture
Lecture plan The subject of epizootology. Tasks of the science of epizootology The science of epizootology, its history. Science of epizootology, relationship with other sciences Importance of epizootology in agriculture .	
The purpose of the training session: To inform students about the subject of study giving gifts	
Pedagogical tasks: The subject of epizootology. Tasks of the science of epizootology The science of epizootology, its history. Science of epizootology, relationship with other sciences Importance of epizootology in agriculture .	*Results of educational activities:*
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. The subject of epizootology. Tasks of the science of epizootology The science of epizootology, its history. Science of epizootology, relationship with other sciences Importance of epizootology in agriculture . *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: Epizootic process, epizootic, biological, geographical and socio-economic factors, immunoreactivity, infection, protein, reservoir, zoonosis , zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive path, horizontal path, vertical road, sporadic, panzootic, pandemic, anthropurgic.

A social literature

1. Salimov X.S, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov X.S, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4. Mirziyoyev Sh.M. Development strategy of new Uzbekistan. Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www. fvat@.academy.uzsci.net

Communicable diseases are fundamentally different from non-communicable diseases. These diseases:

first, it appears only when a disease-causing microorganism or virus enters the body.

Secondly, the pathogen is characterized by its transfer from a sick animal to a healthy one. This allows the disease to continue to spread and cause mass destruction. As a result, the infectious disease spreads over a large area, and the epizootic process continues continuously, causing great economic damage to the national economy.

Thirdly, an antibody is formed against the pathogen that has entered the body

EPISOOTOLOGY *(gr.* ***Epi - surface, outside + zoon - animal , logos - doctrine*** *) is a special field of veterinary science, which studies the laws of epizootic processes (the causes and conditions of the origin, spread and extinction of infectious diseases) and on this basis Sha is a science that determines ways to prevent and fight against diseases.*

EPIZOOTIC PROCESS *(lat.* ***processus*** **-** shift) - the mechanism and factors of the transmission of an infectious agent from a sick animal to a healthy animal, as

well as overt and hidden as a result of the interaction between the agent and susceptible animals emergence, spread and disappearance of infections.

The term "epizootology" consists of 2 concepts: "epizootia" and logos. **Epizootic** *(gr.* ***epi*** — surface, ***zoon*** - animal) is the average level of the intensity (severity) of the epizootic process, the spread of the infection beyond the boundaries of one unhealthy place and across regions, even republics and countries.

Epizootology performs 2 tasks:

-first, he studies the cause of the disease, its non-uniformity, spread, fading and disappearance of the infection (the essence of the epizootic process), the influence of the external environment on the intensity of the epizootic process, in a word, the epizootic process ;

- secondly, it develops and improves the methods of disease prevention and elimination, that is, it eliminates the epizootic process due to the active participation of human influence in the laws of its development.

Epizootology consists of 2 parts: general epizootology and special epizootology.

General epizootology *studies the laws of occurrence, development and control of epizootic process. These laws are determined on the basis of the analysis of the results obtained during the fight against an infectious disease in a certain historical period, epizootological experiments.*

Concepts of infection and immunity, epizootic and infectious processes, classification of the evolution of infectious diseases and the basic principles of their prevention and control are the subject of general epizootology.

Privaty epizootology studies the cause (causing agent) of an infectious disease, its specific epizootological features, pathogenesis, clinical symptoms, diagnoses the disease, develops special and non-specific measures to combat it, and determines the ways of its prevention .

The emergence of new diagnostic methods makes it possible to detect previously unknown diseases. M: Highly pathogenic influenza, leukemia, SPID, encephalopathy, hantavirus pneumonia, human atypical pneumonia, and several other new diseases became known in the next 20-30 years. Currently, there are more than 300 infectious diseases among domestic and wild animals, fish and bees. In addition, dozens of diseases are transmitted from animals to humans.

The history of the development of epizootology

People had very primitive concepts about epizootics in BC. Later, people learned that diseases can be passed from one person to another. It is becoming known that after the sick people are cured, they will not get sick again. It is suspected that there is a disease-causing agent.

The Greek physician Hippocrates (460-377 BC) hypothesized that the epidemic was caused by an agent transmitted by living contact. In Rome, Lucretius (96-55 BC) wrote down the clinical symptoms of diseases such as **rabies, smallpox, swine scurvy, and cattle peripneumonia .**

In 1546, the Italian doctor Girolomo **Fracastro** wrote about **protein, sheep pox and horse mange** in his book "Contagion and contagious diseases" . However, the causative agents of contagious diseases were not identified until Louis Pasteur. In 1861, a new era in epizootology began after L. Pasteur discovered that **rot and decay are caused by microbes.**

L. Pasteur created a vaccine against rabies, anthrax and pasteurellosis **by weakening** the pathogen .

R. Koch in 1861 identified the causative agents of tuberculosis and cholera by growing the causative agents in artificial **nutrient media .**

Daven in France in 1849 and Brauel in Russia in 1856 were the first to see the causative agent of anthrax.

I. Mechnikov (1845-1916) **immunity** creates the **phagocytic theory**

P. Ehrlich (1854-1915) **immunity** creates **humoral** theory Both were awarded the Nobel Prize in 1908.

D. **Ivanovsky** (1892) proved that tobacco mosaic disease is caused by a virus.

I. **_ S. Andriyevsky** (1759-1809) wrote the 1st book on infectious diseases, and he infected himself in **1787 with the blood of an anthrax animal.**

1st textbook of epizootology by the Medical Surgeon. Academy professor PI Lukin (1790-1838) wrote.

VI **Vsevolodov** (prof.) 1846. wrote the 1st book on animal infectious diseases.

In the middle of the 19th century **II Ravich** (1822-1875) wrote the 1st epizootology

LS **Senkovsky** (1822-1887) created a vaccine against anthrax

O. **Kalning** - vet doctor allergist. diagnosis created **mallein** for horse manka

SN Vyshelessky (1874-1958) was a mass allergist of horses. diagnosis and they managed to lose **the manka by doing RSK** . He does great work on tuberculosis, anthrax, protein, cattle peripneumonia. In Belarus, he conducts research on the etiology of tuberculosis, mange, brucellosis, rabies, and infectious encephalomyelitis of horses in Alma-Ata. During the years 1934-1958, he headed the department of epizootology at MVA. In 1935, he published a book for students on the 1st **century epizootology**

MS Gannushkin - **general** publishes a book for **epizootology students.**

PP Vishnevsky, MK Yuskoves, II Lukashov, Poddubsky - tuberculosis.

E.S. Orlov, K. Shumilov - Brussels

Ya.I. Kolyakov, IV Poddubsky, AM Laktionov - YuAN of horses

FA Terentev, SG Kolesov - anthrax

MD Polikovsky, NV Likhachev - sheep diseases, anaerobic diseases

Ya.R. Kovalenko - necrobacteriosis, emkar, anaerobic diseases

S.Ya Lyubashenko has done extensive research on leptospirosis.

In Uzbekistan - Sh.T. Rasulov, Parmonov MP (Trichophytosis), AKSitdigov, IDBurlusky, AM Akhmedov, GA Kudryavsev (colibacteriosis, salmonellosis), AA Volkova, FD Lukashenko (peripneumonia of goats), XS Salimov (leukemia, rabies, anthrax, protein, blackworm, bradzot and enterotoxemia) , NM Mamatov conducted research on (rabies) diseases and created special preventive and diagnostic tools

against the aforementioned diseases and achieved a sharp reduction of these diseases in our country.

Erelationship of epizootology with other sciences

Based on the modern tariff of epizootology, we are convinced that this science is a very complex and multidisciplinary problem, and it is necessary to use molecular biological, veterinary and socio-economic methods in the study of its many aspects. M: molecular biology, biochemistry, biophysics and immunochemistry, the laws of the origin of infection, the formation of immunity in it; Cytomorphology and pathomorphology study the changes in cells, tissues and organs in the body, while microbiology and virology determine the infectious process in the body. And epizootology studies the epizootic process in that population - group or herd.

Epizootology is related to epidemiology because it deals with animal diseases that can be transmitted to humans.

Infectious disease agents are often spread to other animals with the help of insects (flies, moles, ticks), which means that it requires studying the biology of insects. Therefore, parasitology depends on bn.

Epizootology deals with the clinical diagnosis, pathogenesis, pathomorphology, and treatment of tuberculosis. So it is related to clinical diagnostics, therapy, pathological anatomy, physiology, pharmacology.

It is also related to Animal Hygiene, Nutrition Sciences.

It is related to nosogeography, because it is very important to study the influence of natural factors in the spread and origin of the disease (soil, water, wind and landslides, floods).

Statistics is also related to Economics. The economic structure is very important.

Epizootological methods

EPIZOOTOLOGICAL METHODS - methods of studying the laws of epizootic processes. One of the main of these methods is the method of epizootological investigation, which includes a system of comparative-geographic description, epizootological investigation and experimental methods.

EPIZOOTOLOGICAL INSPECTION - inspections conducted to determine the epizootological situation in the farm, address, district, region and republic. It is important to make a diagnosis of the disease on the basis of ET, to determine the source of the infectious agent, the ways of its spread.

EPIZOOTOLOGICAL EXPERIMENT — experiments conducted in the laboratory and livestock in order to study the epizootological process in detail from all sides, to check the effectiveness of various preventive and therapeutic means.

EPISOTOLOGICAL ANALYSIS *(gr.* ***analysis*** **—** study of the behavior and character of the epizootic process in a certain place (farm, population center, district, region, republic) and time (month, year) using specific methods.

Statistical methods are used for epizootological analysis. Epizootological statistics turn an absolute indicator into a relative indicator when evaluating the same phenomenon. Relative indicators are intensive and extensive. **The intensive indicator** includes: morbidity rate, death rate, death rate and fatality rate (lethalnost).

INCIDENCE is an indicator characterizing the number of animals suffering from a disease, the ratio of infected animals to all animals susceptible to this disease. This indicator is calculated for 100, 1000, 10000, 100000 animals. For example, if 15 out of 1,000 people prone to this disease are sick, it is 1.5 percent.

MORTALITY RATE is an indicator of the severity of infectious diseases, expressed as a percentage of the number of dead animals to the number of susceptible animals. The mortality rate can be calculated per 10,000 susceptible animals.

MORTALITY RATE is the ratio of the number of dead cattle to the number of cattle infected with this disease expressed as a percentage. The degree of lethality characterizes the severity of an epidemic of an infectious disease under certain conditions.

An extensive indicator is the geographical distribution of the disease, the prevalence of the disease among animals grouped by age. The level of spread of an infectious disease is determined by statistical comparison of the number of unhealthy points in a farm, district, region for a certain period of time (m: 1, 3, 5 years).

Epizootologists use bacteriological, virological, allergic, serological and epizootological experimental methods to diagnose an infectious disease.

Importance of epizootology in agriculture

The science of epizootology has achieved great success.

Rinderpest in 1928, peripneumonia in 1930, horse sickness in 1940, rabies, encephalomyelitis, epizootic lymphangitis were completely eliminated between 1945-1950, that is, they did not return. If in 1948 the rate of infectious diseases was **22% of the total number of diseases, in 1968 it was -7%, in 1971 -** up to 3.6%, and now it is around **0.3-07%** .

Infectious diseases cause huge losses to livestock. For example: in 2001, the country of England suffered a total loss of 11 billion pounds from an epizootic of cattle protein disease.

The main task of epizootologists is prevention of infectious diseases. Because some diseases spread as epizootics, while some spread as panzootics. M: protein occurs annually in 55-60 countries of the world. By preventing infectious diseases, epizootologists not only bring great economic benefits to the national economy, but also protect people from zooanthroponosis (anthrax, rabies, brucellosis, tuberculosis, etc.).

The 2nd special task is to detect an infectious disease, take measures to prevent it from spreading, and carry out measures to cure the farm and settlement of this disease.

PANZOOTIA *(gr.* ***pan*** **-** all **+** ***zoon*** **-** animal) is the most intense high level of the epizootic process, an epizootic of animal diseases that covers several countries and continents. For example, protein, rinderpest, influenza.

ANTI-EPIZOOTIA MEASURES - planned measures aimed at detection, prevention and complete elimination of infectious diseases. These measures consist of methods and tools affecting all parts of the epizootic chain, which are aimed at neutralizing disease agents, eliminating the factors of transmission from one animal to another, and increasing the general and specific resistance of susceptible animals present there. will be directed.

Control questions

1. The subject of epizootology.
2. Tasks of the science of epizootology
3. The science of epizootology, its history.
4. Science of epizootology, relationship with other sciences
5. Importance of epizootology in agriculture .

Infection and its forms. Importance of microorganisms and viruses in the formation of infection and pathogenicity characteristics. Importance of macroorganism and environmental factors in the formation of infection. Clinical forms of infectious diseases .

INFECTION (***infectio*** — contagion, contagion) is a complex biological process, a state of damage resulting from the interaction of an organism with a disease-causing virus or microbes. As a result of the increase in the pathogen entering the body, complex pathological and protective adaptation reactions are observed in response to the pathogen. These reactions are in the form of biochemical, morphological and functional changes in the body, as well as an immunological response, aimed at maintaining the stability of the internal environment of the body (homeostasis). The interaction between the pathogen and the organism is manifested in 3 forms. The most visible form is an infectious disease with obvious clinical signs. This condition causes an infectious disease with certain clinical symptoms. Type 2 is a latent infection without clinical signs, in which the virus or bacteria are transmitted. Type 3 is also called immunizing subinfection. In this case, the causative agent entering the body ensures the formation of antibodies, but the causative agent itself is destroyed in the body, the organism does not become a source of the causative agent of the disease (m.: anthrax, anthrax).

INFECTIOUS PROCESS – dynamics of micro- and macroorganism interaction reactions, that is, a set of processes observed in an organism infected with a pathogen. The latent period, the stages of the beginning, development, and disappearance of symptoms of CK constitute the infectious process.

INFECTIOUS DISEASE is an infectious disease caused by viruses and microorganisms that have evolved to parasitize the animal body. Usually, this disease is characterized by passing from one animal to another, progressive development, special reactions of the macroorganism against viruses and microbes (allergies and antibodies) and the formation of immunity after recovery from the disease.

THE ROLE OF THE MICROORGANISM IN INFECTION AND ITS PATHOGENIC EFFECT - The study of the essence of the infectious process showed

that it is carried out on the basis of the laws of symbiotic living of micro- and macroorganisms. This symbiotic life takes place in different ways: *mutualism*, *commensalism* and *parasitism.*

Mutualism is benefits for both (moderate microorganisms) Some are antagonistic to each other M: lactic-yeast microorganisms stop the development of putrefactive microorganisms, others break down cellulose based on beneficial enzymatic activity (in the large stomach of ruminants), and others produce vitamins. In return, meyeric microorganisms living in the stomach and other cavities of animals serve as the main factor that ensures the body's resistance and natural protection.

In commensalism, 1 symbiont lives at the expense of another (skin-dwelling bacteria, actinomycetes, fungi, staphylo- and streptococci, in the intestine: coli, salm., putrefaction microbes and enterococci, Pasteurella, pneumococci, mycoplasmas, streptococci in the upper respiratory tract). But many of these listed microbes have the ability to cause disease when the body's resistance decreases, and as a result of endoinfection, especially young animals die.

parasitism , the causative agent not only lives in the organism, but also destroys it.

CHARACTERISTICS OF INFECTION:

Alimentary infection *(lat.* ***alimentarus*** **-** food) is the entry of a pathogen into the body through the mouth.

Mixed infection is a disease that occurs when two or more pathogens enter the body (various viruses and bacteria).

Associated infection (***associatio*** **-** united) is an infection caused by the combination of various viruses and microorganisms entering the animal's body. In this case, synergism can be observed, that is, the disease-causing properties of one type of microbes are enhanced by the second type of microbes. For example, staphylococci increase the causative capacity of the microbe. But in some cases, microbial antagonism can be observed. See Intercurrent disease. Parainfluenza or rotavirus +coli+pasteur.

Aerogenic infection *(Gr.* ***ayeg*** **-** air, ***genes*** **-** formation) is an infection caused by the entry of pathogens into the body through the air.

Bacterial infection is an infection caused by bacteria.

Unmarked or (Latent) infection — *(lat.* ***latenta, ae, f —*** hidden) — an infection without clinical manifestations. It is determined by immunological reactions, bacteriological, virological and pathomorphological examinations. Animals with such hidden infections are a very dangerous source of disease. As a result of a hidden infection, immunity can arise in the body.

Wound infection is an infection caused by the introduction of certain pathogenic microbes into wounds, especially deep wounds. This situation is more evident in the case of arthritis.

Fungal infection is an infection caused by pathogenic fungi.

Purulent infection is an infection of pus-forming microorganisms.

Simple infection, monoinfection - an infection caused by a virus or a type of microorganism.

Regional infection *(lat.* ***regie*** **—** a certain area, place) — after entering the body, some pathogens (tuberculosis — tuberculosis, brucellosis, tularemia, etc.) first settle in the lymph nodes via lymphatic channels and become the primary infection site will appear. As a result, the lymph nodes are injured.

Respiratory infection *(lit.* ***respiratory*** **-** nafa0Crqali) is an infection transmitted through the air.

A slow-moving infection is a slow-developing infectious disease (leukemia, visna ve medi, scrapie, adenomatosis, etc.) with a very long latent period after the causative agent enters the body.

Secondary infection *(lat.* ***secundarus*** **-** secondary) is a secondary infection added to the primary (main) infection. This complicates the passage of the first infection. For example, Pasteurella and Salmonella bacteria aggravate the transmission of plague in pigs. Usually, secondary infection is associated with microorganisms belonging to the group of more conditional pathogens. They live on the skin and mucous membranes, increase their activity and become pathogenic only when the body's resistance to diseases decreases.

Spontaneous infection *(lat.* ***spontaneus*** **-** by itself) is an infectious disease that occurs spontaneously under natural conditions.

Artificial infection is an infection caused by artificial introduction of pathogens.

Disseminated infection is an infection that occurs as a result of microorganisms breaking through the protective barriers in the animal's body and spreading throughout the body.

Droplet infection is an infection caused by the causative agent entering the animal's respiratory tract by mixing with mucus and liquid particles released from the sick animal.

Transmissible infection *(lat.* ***transmissibilis*** **-** transmitted) is an infectious disease spread by blood-sucking arthropods, rodents and other virus and microbe carriers.

Dust infection is an infection caused by inhalation of infected dust particles.

Exogenous infection *(lat.* ***yexo*** — besides, ***genes*** — birth, formation) — a disease caused by pathogenic viruses and microbes that entered the animal organism from the external environment.

Endogenous infection *(lat.* **e** ***ndon*** **-** inside, ***genes*** **-** formation) is a disease that occurs due to the increase of microbes present in the body as a result of a decrease in the general resistance of the animal organism.

Conditions for the appearance of an infectious disease:

- the causative agent is pathogenic and virulent and it falls on a susceptible animal;
- the level of protection of the macroorganism from the disease is low;
- the role of the external environment in the introduction of the pathogen into the body - aerogenous infections increase during cold and wet weather, and toxic-infections during food increase in summer. Cold and very hot also reduce the body's resistance.

<u>Infectious disease agents:</u>

Pathogenic bacteria, viruses, fungi, mycoplasmas, chlamydia, rickettsiae.

PATHOGENICITY — the ability of microorganisms and viruses to cause disease. P. is a set of complex disease-causing properties of microorganisms and viruses that have arisen in the process of adaptation to free eating in the organism of plants, animals, people, living beings in general. P. is **a specific sign of a certain type** of microbe, that is, each microbe causes only a specific infectious disease. However, different strains of this type of microbe can have different pathogenicity. P. determines the virulence of microorganisms and viruses. Although P. is a genetically fixed sign of microorganisms, it can be changed under the influence of physical, chemical and biological factors. Manifestation of P. depends on the susceptibility of the organism to that microbe and environmental factors affecting its resistance.

VIRULENCE *(lat.* ***virulentus*** — toxicity) — degree of pathogenicity of a specific microbe or virus strain. V. depends on the endurance of the animal and the conditions of its damage. V. It also depends on the properties of germs and viruses to cause disease: their infectivity, ability to overcome protective means, how fast they multiply in the macroorganism and produce toxic substances. The main virulence factors of bacteria are exo- and endotoxins, toxic antigenic components (for example, S-antigen of microbes that cannot be stained with Gram), aggresins, capsule formation, their enzymes (lecithinase, phosphatase, hyaluronidase, fibrinolisin, streptokinase, DNase, decarboxylase, urease). V. factors of viruses are nucleic acids. V. is determined in experimental animals. Their age, type, live weight must be the same. V. can be weakened under the influence of physical and chemical factors. The virulence of the virus can be reduced by growing it in chicken embryos, a more resistant animal organism, artificially cultured cells.

Gate of infection. M: YuAN, Ephemeral fever - viruses enter the body through the skin with insects, protection of animals from insects - k. prevents Anthrax, protein, tuber. oral, aerogenous and dermal.

The effect of the pathogen on the body: *aggresin* , *exotoxin*, *endotoxin* - after the death of the microbe, it acts together with the mb.

Exotoxin - a layer, botulism - an antitoxin against it organic i.ch., they are inactivated at thermolabile-60 ^{0} .

Endotoxin is thermostable. M: Cholera. Against it, the organism bacterio-lysin, agglutinin, precipitatein, opsonin i.ch.

A *gressins* are not poisonous, but **reduce the body's defenses.**
They are emkar, anthrax, pastellez, tuber. It will be.

Infectious diseases have a latent period.

Passing infectious diseases.

They are *acute, acute, subacute, chronic* can **pass** .

They are ***typical*** and ***atypical*** can pass.

The shape of ***the*** disease **must be *distinguished*** , ***the period of time*** with, the form is characterized by **the location of the pathological process** .

Propensity is the opposite of immunity, that is, the appearance of reactivity. It can be high or low.

The role of macroorganism and external environmental factors in the emergence of an infectious disease - the emergence, development and fate of an infection depends not only on the virulence and quantity of microorganisms and viruses that enter the organism, but also on the natural resistance of the organism against these pathogens. . Therefore, it is necessary to focus not only on pathogens, but also on strengthening the defenses of the macroorganism (increasing natural resistance) and external environmental factors that contribute to the development of the disease.

Before talking about immunity, we need to understand what is general and immunological reactivity. Reactivity is the ability of an organism to respond to external environmental influences by changing life processes. This ability is very clearly manifested in animals with a highly developed and perfect nervous system. If the reactivity is normal, it is easy for the animal to get used to the effects of the external environment, but if there is a change in the reactivity - asthma.

An infectious disease is also a process that occurs as a result of the interaction between a microorganism and a macroorganism. If the reactivity is normal, after the microorganism enters the macroorganism, the body can quickly use its immunological mechanisms against this microorganism, that is, if phagocytosing cells fight against it, and if antibody-producing plasmocytes perform their function, the disease will manifest itself. doesn't. In the opposite case, the body's adaptation mechanisms to the microorganism do not work, the microbes multiply in the body and the symptoms of the disease appear, in some diseases: anthrax, anthrax, the animal dies.

Immunological reactivity is the ability of the body to use immunological defenses against infectious agents, that is, the ability to respond specifically to the antigen that causes the disease - to ensure complete protection of the body from the negative effects of the microorganism.

The ability to protect the body and provide immunity against pathogens depends on many general and specific factors. The reactivity based on non-specific physiological defenses of the body is included in **the general immunological reactivity** . It depends on the animal's type, individual characteristics, age, physiological state of the organism, the nutritional value of the given feed, and the effects of various peculiarities of the external environment. This reactivity actually indicates the animal's natural susceptibility to disease, or vice versa, its resistance.

On the other hand, the state of immunological reactivity and the reaction of the animal also depends on the effect of biological agents and antigens. The variable immunological reactivity of such an organism to a specific stimulus or antigen is called **specific immunological reactivity** .

Specific immunological reactivity is common is a part of immunological reactivity and plays an important role in the formation of immunity in the body.

IMMUNITY *(lat.* ***immunitas, atis, f —*** resistance, immunity) is the protection of the organism from infectious and non-infectious substances (antigens) that have

genetic information foreign to it, that is, the state of immunity, the ability to fight against diseases. Level I depends on the physiological defenses that keep the body's internal environment always in balance.

Information about immunity arose from the observation of epizootics and epidemics. It is known from ancient times that some people and animals do not get sick even in the conditions of any epizootics and epidemics, and those who recover from the disease do not get sick again.

In the early stages of the study of immunity - in 1887, ru0Climi II Mechnikov discovered the phagocytic theory, connecting immunity with phagocytes. Then P. Ehrlich created the humoral theory of immunity in 1901. Later, based on the successful experiments of IP Pavlov (1938), G. Selye (1942), F. Bernet (1959), PF Zdrodovsky (1961), RV Petrov (1976) and others, instead of the previous theories of immunity based on various factors, a whole organism information on immunological reactivity was obtained and it was proved that all immunological events in the body are subject to general physiological laws.

Immunity arises not only when recovering from a disease, but also to dead or living microorganisms, some of their parts, poisons, and various proteins and haptens. Immediately notices all antigenic substances foreign to the body.

ANTIGEN DRIFT is a gradual, small change of some viruses in the process of evolutionary development. This process is manifested by a change in the antigen of the envelope of viruses. AD reduces the effectiveness of veterinary interventions.

ANTIGEN CEILING is a structural change in the antigenic properties of viruses. This causes the disease to spread very widely. The main cause of AS is mutation in viruses and bacteria.

ANTIGENS *(gr.* ***anti*** **↑,** ***genes*** — formation) — high-molecular organic substances (viruses, microbes and their products) of a protein nature, which have genetic information foreign to the organism and, when exposed to it, produce specific antibodies (antibodies) or other immunological processes (immunological memory, tolerance). A. is very complex and may consist of several active parts. A. has the ability to combine with antibodies in the body or in a test tube, in any container.

TYPES OF ANTIGENS:

Heterogeneous antigens are common antigens with antigenic determinants specific to different species.

Metabolite antigens are protein substances (metabolites) formed during the life of bacteria.

Perfect antigens are those that cause the process of formation of antibodies (immune bodies) in the body. Protein substances that increase the sensitivity of lymphocytes (sensitizing), embody the properties of foreignness to the body.

Imperfect antigens, haptens are non-protein substances. Examples are small molecule substances, lipids, ribonuclease, insulin and similar substances that bind to antibodies, but cannot independently form antibodies in the body. If large molecular substances are added to such substances, they become antigens and have the ability to form antibodies. See G aptens.

Protective antigens *(lat.* ***protectivus*** **-** protective) are antigens of viruses or bacteria that produce antibodies against a particular disease in the body. This feature is present in the capsule antigen of pneumostreptococci. Mucoprotein of streptococci and alphaprotein of staphylococci also have protective properties.

Transplant antigens are antigens that indicate the genetic similarity or foreignness of the cells and tissues of two organisms.

GROUP OF ANTIGENS is a type of antigen specific to individuals of a group or species. For example, based on this indicator, people are divided into blood groups, and bacteria are divided into serological types.

ANTIGENICITY is an indicator that expresses the ability to form antibodies against itself and combine with it when a foreign substance (antigen) enters the body. A. It is characteristic of proteins with a molecular weight of more than 10,000 D and biopolymers with scattered polypeptide chains and artificial polypeptides with a tertiary structure containing many disulfide bonds with L-amino acids. Polypeptides containing tyrosine and lysine amino acids are highly antigenic.

ANTIGEN ACTIVE PART (epitope) - an amino acid antigen is the location of the spatially active part of protein residues. Forming a bonding area on the surface of the antigen, it adheres to the special center of a special antibody (immune body) or serves as an adhesive group for it.

ANTIGENIC CHANGE — changes in the antigenic properties of bacteria and viruses under certain conditions, in which certain antigenic features disappear or new features appear.

ANTIBODIES - immunoglobulins that appear against them in the blood and tissues when antigens enter the body - immune cells. A. has the property of combining with antigens in the organism or container. A. after the antigen enters the body, it is formed in the plasma cell of the lymphoid tissue. A. is specific only to the corresponding antigen, that is, it has the property of joining (reacting) only with the antigen sent for its own formation. Immunological diagnosis of infectious diseases is based on antigen-antibody binding reaction. Determining the presence of A. in the blood serum of a sick organism with a special antigen of a virus or bacteria means that the animal is infected with this virus or bacteria. A. It is divided into 5 classes: A, M, Ye, D and J immunoglobulins. A. mainly neutralizes exo- and endotoxins of bacteria and viruses, snake, black worm venom and other antigens. See Immunoglobulins.

TYPES OF ANTIBODIES:

Avid antibodies are antibodies that are formed upon second contact with the same antigen.

Homologous antibodies are specific antibodies against the same antigen.

Humoral antibodies are antibodies in blood serum.

Monoclonal antibodies) are highly specific antibodies that act selectively depending on the chemical composition and location of antigen molecules.

Neutralizing antibodies are antibodies that bind to an antigen and neutralize an infectious agent.

Labeled antibodies are antibodies conjugated with a self-emitting substance.

Normal antibodies are normal antibodies-isoagglutinins that are formed in the blood without the introduction of antigen.

Surrounding antibodies are incomplete, monovalent immunoglobulins, which differ from normal antibodies by having only one active center in the molecule. These antibodies can only be detected by a special Coombs reaction.

All pathogens entering the body cause 2 types of reactions: a) non-specific, related to general immunological reactivity; b) special, related to the special immunological reactivity of the organism. Manifestation of all non-specific reactions in the body (hematological, histological, cytological, biochemical, etc.) is associated with 3 types of special immunological conditions in the emergence of immunity:

a) slow type of extreme sensitivity;

b) immediate type of extreme sensitivity;

c) tolerance (reactivity).

A slow type of hypersensitivity is formed due to specific changes in immunocompetent cells without the appearance of serum antibodies against the pathogen. This condition is determined as a result of an allergic test. In this case, instead of immunity, there is a very sensitivity (sensitization) to this stimulus. Tbs.

The immediate type of hypersensitivity is in the blood It is formed by the formation of special antibodies and their reaction with special antigens. The appearance of antibodies in the blood indicates that immunological changes have occurred in the body, and in many cases strong immunity is formed.

In the case of tolerance (reactivity), the ability of the body to form antibodies against this pathogen is lost, but the ability to form antibodies against other pathogens is preserved. The state of tolerance (reactivity) often appears in young animals, when it is exposed to the pathogen in the neonatal or prenatal periods, or it can be formed when a large amount of antigen is administered. In this case, instead of immunity, tolerance appears. Therefore, immunity does not always occur when a vaccine or stimulus is injected into the body. In some cases, susceptibility may increase due to increased sensitivity or tolerance of the organism to this stimulus. Therefore, it is necessary to determine the special immunological state of the organism.

There are many types and mechanisms of immunity. Most of them are non-specific, so they are equally effective against all microbes. On the contrary, the characteristics of the immunity manifested in the process of the formation of immunity are directed only against that particular microbe or its serovariant.

Any incoming pathogen is faced with non-specific protective means of the organism and they prevent the entry, development and reproduction of the microbe. And special protective means are aimed at neutralizing this microbe.

Immune factors: depending on the time of appearance:

a) *constant;*

b) after entering the microbe.

Depending on the nature of exposure:

a) non-specific;

b) special.

the n omaxsu0Cmils that are constantly affecting :

1. *Protective properties of skin and mucous membranes;*
2. *Meierian microflora protective properties;*
3. *Physiological factors: t-ra, urinary excretion, metabolism, spores do not multiply in an oxygen environment. Sugar in the womb and xz*
4. *Phagocytosis and barrier service of lymphoid system ;*
5. *Humoral factors (lysozyme, complement , meyer antibodies, etc.);*
6. *Genotypic and phenotypic reactivity in tissue and cells.*

the microbe, it affects n omaxsu0Cmils :

1. *Inflammation;*
2. *C-reactive protein;*
3. *Interferon.*

After entering the microbe, the special effects are :

1. *Special macrophages;*
2. *Plasmacytic cells;*
3. *Lymphoid cells;*
4. *Antibodies.*

The protective properties of the skin and mucous membranes depend on their integrity. It also has bactericidal properties. Mucous membranes contain lysozyme, which affects all mbs, unicellular organisms, except viruses.

Protective properties of Meierian microflora - Meierian microflora in all mucous membranes, skin and cavities is a strong natural protective factor. (m: lactic yeast destroys putrefactive microbes, escherichia streptococci - has an antagonistic effect - consumes the nutrient medium, changes RN, self-antibiotic i.ch. and x-zo).

Physiological factors - If t-ra rises, mb does not increase, under the influence of t-ra and the excretion of the pathogen in the urine through the kidneys rids the body of the pathogens.

Metabolites protect against disease in the living organism. M: if there is sugar in the uterus of a cow, it becomes difficult to pass brucellosis and abortion occurs. Because the tissues are well supplied with O_2, anaerobes do not grow.

Inflammation - if mb passes through the barrier, there will certainly be inflammation in that place. Inflammation is a means of protection - adaptation, where t-ra increases, rn changes, ***phagocytosis*** begins. Inflammation and phagocytosis capture the antigen at the point of entry and do not destroy it. Phagocytosis is carried out by micro- and macrophages. If the microbe dies under the influence of phagocytosis and undergoes lysis - complete phagocytosis, if it does not die - m: tbs, brus., Pasteurella is called incomplete phagocytosis. Viruses are phagocytosed by pinocytosis. ***Factors increasing phagocytosis*** : antibodies, calcium, cholesterol, histamine (released from inflammation).

Factors weakening phagocytosis : avitaminosis, acetylcholine, corticosteroids. Phagocytosis is of secondary importance in viral diseases. It is the 1st degree in bacterial diseases.

Immunological protection service of the lymphoid-macrophage (immune) system

Lymph nodes, spleen, bone marrow and thymus (sac of Fabrizio in birds) and lymphoid tissues in the alimentary tract, lungs and other organs (tonsils, Peyer's patches and solitary follicles) are non-specific protective-barrier and immunologically competent. performs the service. Lymphoid tissues, which make up 1% of the animal body, prevent the entry of microbes, phagocytose them, slow type of hypersensitivity, immediate type of hypersensitivity, i.e. produce antibodies. Therefore, the lymphoid-macrophage (immune) system is a morphological system that responds to immunogenesis in the body.

Cells performing immunological services in the body are mainly micro- and macrophages, lymphocytes and plasma cells.

Microphages (neutrophils and eosinophils) and macrophages (mononuclear phagocytes-monocytes) in the bone marrow perform the same function in immunity - phagocytosis, even though they are morphologically different. Phagocytosis is the 1st stage of immunity, the beginning.

Microphages dissolve the captured antigen and turn it into an elemental substance. ***Macrophages*** transform the bacterial antigen into an immunologically active form and provide information about the antigen to lymphocytes, accelerating the transformation process in them.

Lymphocyte origin, morphological and are divided into 2 large groups according to their functional characteristics: **1.** ***T-lymphocytes*** (tumus-dependent) are morphologically small lymphocytes, and under the influence of an antigen, they become 2 types - ***immune lymphocytes*** and ***immunological memory lymphocytes.*** *Immune lymphocytes are similar to* ***T-lymphocytes*** , but have a cytopathic effect and *do not destroy antigen without antibodies* . Immunological memory lymphocytes are similar to ***T-lymphocytes*** , but the information about the antigen is stored in its core genetic apparatus.

T-lymphocytes are in cellular immunity, in the slow type of hypersensitivity and is involved in autoimmune diseases.

2. ***Independent of Tumus V - lymphocytes*** are morphologically small lymphocytes. During the immunological response, they transform into plasma cells that produce antibodies and lymphocytes that store immunological memory through the immunoblast stage as a result of transformation. When the body meets the previous antigen again, *plasma cells* arises from lymphocytes that store immunological memory .

Plasma cells are very different from lymphocytes in terms of their structure and function. Depending on their maturation, they are divided into *plasmablasts, immature and mature lymphocytes* . These cells are highly specialized glandular (secretory) cells, and their main service is the synthesis and secretion of immunoglobulins (antibodies).

Macrophages, T- and V-lymphocytes participate together in immunogenesis.

Protective properties of humoral factors and antibodies in immunity

Substances that inhibit the growth of microbes and viruses are present in all body fluids. Tissue suspensions, blood serum, and secretions from glands specifically inhibit the growth of bacteria. It is known that non-specific protective factors in the body are lysozyme, complement and meyer antibodies. After micro-organisms and viruses enter the body or a vaccine is administered, special protective antibodies against them appear. First (1-3 days) antibodies begin to appear slowly (*inductive phase*), later (7-10 days) *in the productive phase* , rapid and maximum release begins and then slows down again. After several months, the titer of antibodies against this antigen in the blood serum decreases greatly or cannot be detected at all. However, as we mentioned above, immunological memory lymphocytes: T- and V-lymphocytes remain. If the previous antigen enters the body again, the antibody is formed quickly (1-2 days) and in a larger amount than the first time. Immunological memory is of great importance in determining the timing of subsequent vaccinations.

Genotypic and phenotypic reactivity of cells . The origin of the disease or the immunological condition in a susceptible animal depends on the genetic resistance (***genotypic reactivity) of the cell in which the pathogen reproduces in the organism (m: coli).*** Selection of the animal taking into account its genetic resistance to this pathogen is a promising way to prevent this disease (m: leukemia).

In a disease-prone animal species, the reactivity of cells changes throughout ontogeny and immune formation without being constant. This condition is especially noticeable in viral diseases. In the early stages of immunogenesis, a non-specific reactivity appears in the body against this virus due to the production of a low-molecular protein - an inhibitor (which interferes with the reproduction of the virus). After recovery from the disease, *special reactivity (**phenotypic reactivity)** tissue immunity in those cells* appeared.

Specific features of immunity against viruses.

Immunity against viruses is similar to immunity against bacterial diseases, but since virus reproduction takes place in cells and its metabolites are associated with metabolites of damaged and dead cells, *immunity* also has its own characteristics.

Innate immunity against the virus does not get sick - due to the lack of receptors in *the* cells where virus reproduction should take place, viruses do not enter the cell, the adsorption stage does not take place.

2. Non-specific immune factors - *inhibitors* are more important in *immunity against viruses than in immunity against bacterial diseases, they* prevent *adsorption - entry of viruses into cells.* They are present in all fluids and serve as antibodies, but are nonspecific.

3. We have already mentioned that the body's urinary system and t-ra play an important role in immunity against viruses.

4. *Interference* phenomenon - loss of reproduction of 2 viruses of one virus (m: herpes-smallpox; flu-encephalomyelitis; protein - smallpox;). Interference occurs not only with a live, but also with an inactivated virus vaccine. In studying the phenomenon of *interference , Isaac and Lindeman (1957)* discovered a very powerful

non-specific protective agent - *interferon* . **Interferon** does not affect virus adsorption, viropexy, deproteinization, NK release, virus release from the cell. It affects the virus only through the sensitive cell and **prevents the reproduction of the virus.**

Antibodies formed *in immunity* against the virus affect only *virions (outside the cell), not the intracellular virus.* It should be noted here that not all types of anti-virus antibodies are protective against the virus, only *virus-neutralizing antibodies* have protective properties, they affect the outer corpuscular antigen *of the virion and cause it to be adsorbed, i.e. to the cell.* prevents its entry and neutralizes its toxic effect. *Virus-neutralizing antibodies* also activate phagocytosis. As a result, the viral cells that are phagocytosed by macrophages are neutralized in the macrophage cytoplasm together with the virus toxins.

Antibody structural classes: IgM, IgA, IgG, IgD, IgE.

Types of antibodies: aggl., presip, complement. compound., hemaggl. - is important only in serological reactions.

6. *In immunity against the virus* ***phagocytosis*** plays an important role, although not as much as in bacterial diseases. Even if there is no viru0C in the phagocytosed viral cells, they are neutralized together with their toxins in the macrophage cytoplasm. Microphage reaction against virus antigen is not observed at all, we talked about macrophage reaction.

7. *Local secretory antibodies* play an important role in immunity against viruses . In pneumoenteritis, it was determined that the state of resistance does not depend on the titer of antibodies in blood serum, but on special secretory antibodies released from the mucous membranes of these organs. **IgA** antibodies are found only in respiratory and digestive organs, not in saliva, tears, nasal and bronchial fluids, bile, colostrum, intestines, conjunctiva, urinary system. found in mucous membranes. This **secretory IgA is** structurally different from immunoglobulin A in blood serum. It was found that this IgA has an additional antigenic determinant and its ***molecular mass is larger*** . It meets the virus on the mucous membrane and prevents the virus from entering the body, mainly acting **as a barrier** .

NATURAL RESISTANCE (resistance) *(Lat.* ***resistentia*** **-** resistance) - resistance of the organism to infections due to its biological characteristics or due to vaccination. TR depends on the non-specific protective forces of the body: complement, lysozyme, properdin, bactericidal activity of the blood, the lymphoid macrophage system. TR will be absolute and relative. For example, infectious diseases specific to animal species affect only a certain species of animal. Cattle are naturally resistant to equine infectious anemia.

Types of immunity:

Depending on the origin: **HEREDITARY** - *congenital, type and natural* and **INCREASED** and divided into *antibacterial, antiviral and anti-toxic immunity* .

Immunity *(lat.* ***Immunitas,*** resistance, non-infectiousness) — protection of the organism from infectious and non-infectious substances (antigens) that have genetic information foreign to it, that is, the state of non-infectiousness, the ability to fight

against diseases. Level I depends on the physiological defenses that keep the body's internal environment always in balance.

Hereditary immunity is such an immunity that is genetically specific to this type of animal and is passed on to the next generation (horses are resistant to protein, cattle are resistant to mange.

Increased (active) immunity - the formation of immunity after recovering from an illness or receiving a vaccine. Immunity the duration is different in the body.

IMMUNITY (antibacterial type) - resistance to bacterial diseases, caused by recovery from illness and vaccination, is formed in the unity of the body's general (humoral substances, phagocytosis) and special protective means (antibodies).

IMMUNITY (antitoxin immunity) — resistance to toxin-producing pathogens, mainly occurs as a result of administration of anatoxin, antitoxin to the body. M: in the layer.

IMMUNITY (humoral type) - endurance due to the ability of special immunoglobulins (antibodies) in blood serum to neutralize microorganisms and viruses.

IMMUNITY (post-infection) is the emergence of a strong resistance against a certain infectious disease after recovery.

IMMUNITY (non-sterile type) - resistance to a certain disease that occurs as a result of recovery from illness, in which the body is not completely free from the causative agent (brus, tuberculosis, yurt, leukemia).

IMMUNITY (sterile type) - resistance that occurs after being completely cleansed from the causative agent of the disease after recovery.

IMMUNITY (natural variety) - natural resistance, resistance to some infectious diseases. M:, cattle do not suffer from anemia.

IMMUNITY (transplantation type) is an immunological process formed against tissues and organs introduced into the body. At such a time, the cellular part of the immune system is actively involved, and this process is slowed down, in the form of hypersensitivity.

Phagocytic immunity - immunity based on special phagocytes.

Cells immunity - resistance based on tissue protection.

Passive immunity - ready antibodies induced immunity. It takes 15-20 days. Colastral im-t.

Antibodies - immunoglobulins - immune cells that appear against them in the blood and tissues when antigens enter the body. A. has the property of combining with antigens in the organism or container.

Antigens *(gr.* ***anti*** **↑,** ***genes*** — formation) — any high-molecular organic substances (viruses, microbes and their products) of a protein nature, which have genetic information foreign to the organism, and when exposed to it, produce specific antibodies (antibodies) or other immunological processes (immunological memory, tolerance).

Type of antibodies:

1. Antitoxins - neutralizes toxins
2. Virus neutralizing antibodies

3. Agglutinins
4. Precipitins
5. Complement binding antibodies
6. Bacteriolysins, cytolysins and hemolysins are antibodies that prepare bacteria for lysis (dissolving) and kill them.
7. Opsonins, tropins - antibodies that prepare bacteria for phagocytosis
8. Properdins have a bactericidal and virus neutralizing effect and work under the effect of complement.
9. Leukins, erythrins - have a bactericidal effect.

Immunoglobulins

1. Immunoglobulin A
2. Immunoglobulin M
3. Immunoglobulin D
4. Immunoglobulin Ye
5. Immunoglobulin G

Cell culture

1). In 1949, the cultivation of cells in artificial conditions was achieved, and in 1952, ***Enders, Weller and Robbins Nobel*** received ***the award*** .
2). Huang and Enders (1943) - developed a method for SPD virus detection.
3). Dulbecca and Fogt (1952) obtained 1-layered cells by treatment with trypsin.

Blood elements

1). In 70 years, 1.5 kg of bone marrow - km, 275 kg of lymphocytes, 460 kg of erythrocytes
5400 kg of granulocytes and 40 kg of platelets are produced.
2). Erythrocytes are formed in 2-3 days, granulocytes in 3-8 days.
3). Erythrocytes live 100-120 days.

Control questions

1. Infection and its forms
2. Infection and its types
3. Explain the infectious process
4. Describe an infectious disease
5. Explain mutualism, commensalism, parasitism
6. Gate of infection
7. Immunological reactivity
8. Types of immunity
9. Describe anaphylaxis and allergy
10. Antigens and their immunogenicity

SUBJECT: IMMUNITY AND IMMUNOLOGICAL REACTIVITY . EPISOTOIC PROCESS AND ITS MOTIVATING FORCES .

Lecture	**Epizootic process and its driving forces**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
The form of training	Enter. Visual lecture
Lecture plan Epizootic process The mechanism of transmission of the pathogen from one animal to another Susceptible animals are the driving forces of the epizootic process	Development laws of the epizootic process and stages of epizootics Periodicity and seasonality of epizootics. Epizootic outbreaks and natural outbreaks .
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Epizootic process The mechanism of transmission of the pathogen from one animal to another Susceptible animals are the driving forces of the epizootic process Development laws of the epizootic process and stages of epizootics Periodicity and seasonality of epizootics. Epizootic outbreaks and natural outbreaks ..	*Results of educational activities:* Students: Epizootic process The mechanism of transmission of the pathogen from one animal to another Susceptible animals are the driving forces of the epizootic process Development laws of the epizootic process and stages of epizootics Periodicity and seasonality of epizootics. Epizootic outbreaks and natural outbreaks
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Epizootic process The mechanism of transmission of the pathogen from one animal to another Susceptible animals are the driving forces of the epizootic process Development laws of the epizootic process and stages of epizootics Periodicity and seasonality of epizootics. Epizootic outbreaks and natural outbreaks *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: Epizootic process, epizootic, biological, geographical and socio-economic factors, immunoreactivity, infection, protein, reservoir, zoonosis , zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive path, horizontal path, vertical road, sporadic, panzootic, pandemic, anthropurgic.

A social literature

1. Salimov X.S, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov X.S, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4. Mirziyoyev Sh.M. Development strategy of new Uzbekistan. Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www. fvat@.academy.uzsci.net

The main characteristic of infectious diseases is the participation of a special pathogen in the emergence of an epizootic chain, the danger of a sick animal to a healthy animal. Therefore, the introduction of a special pathogen into the body and continuous contact of a sick animal with a healthy organism is the main responsible reason for the emergence and spread of all infectious diseases.

EPIZOOTIC PROCESS *(lat.* ***processus*** **-** progress) - the source of the disease, the mechanism and factors of the pathogen's transmission from a sick animal to a healthy animal, as well as the interaction between animals susceptible to the disease as a result of the appearance, spread and disappearance of obvious and hidden infections. In a word, **the entry of a pathogen into the body, causing a disease, and the release of the pathogen into the external environment.**

For the emergence and passing of an infectious disease-epizootic process, there are definitely 3 main links, i.e. 3 parts **of the epizootic chain : 1) the source of the disease** ; 2) **transfer of the causative agent** from one animal to another **ways,**

factors ; 3) **prone animals** must participate. If none of them is involved, the epizootic process, that is, the disease does not occur and does not spread. Determining the role of each link in epizootics is of great theoretical and practical importance. M. and V. adapted to live in the organism during the evolutionary development. Only some - *leptospira live* in low-protein water, and *anthrasis, clostridia, and tetanus* live in soil (organic matter). Fungi - causative agents of fusariotoxicosis, stachyobotritoxicosis, botulism - Clostr. Botulinum lives in silage, hay, grain.

The source of the pathogen. The epizootic process is based on biological parasitism, which can be considered as a process of interaction between a susceptible animal population and a specific pathogen under certain environmental conditions. As a result of this interaction, the causative agent of an infectious disease has adapted to parasitism in a susceptible animal organism during evolutionary development and has become its natural **host** . Therefore, when studying the spread of infectious diseases, it is not necessary to consider a microorganism without a natural means of survival, because it cannot live long without a host.

In most infectious diseases, the causative agent must be present in the body in order to be stable in nature and ensure the continuity of the epizootic process, but a small number of certain microorganisms: **Leptopira** in protein **water, causative agent of Saramas, listeria, clostridia, anthracis - in the soil** , **salmonella** living **on milk** , adapted to reproduction. Foods made from plants (hay, straw and hay) are a source of life for some pathogenic bacteria and fungi.

The spread of the causative agent to the environment is different in different stages of the disease, less in the latent period, and more and faster in the advanced period.

Clinically healthy animals are considered to be the source of the causative agent of the **disease** . The causative **agents** are different in different diseases. In **chronic** transitory diseases: br., tbs, leptosp., Yuan, **lasts for a long time in salmonella.** Factors that reduce the body's resistance: cold, heat, wind, hunger, dehydration, poisoning, **latent** or **subclinical x-rays** can aggravate the **current disease.**

Although the carriers of the pathogen do not play an important role in spreading it quickly in relation to the sick, because they are not detected in time by veterinarians, they lead to the stationary storage of the infectious disease in a healthy area, the emergence of an epizootic focus.

Reservoir of an infectious agent is a storage of pathogenic agents in certain natural conditions. M: occurrence of leptospira in water bodies and rodents, anthrax causative agent in places where carcasses are buried.

ZOONOSES are a group of infectious diseases specific only to animals. For example, infectious rhinotracheitis, distemper, swine fever, and other diseases.

ZOOANTHROPONOSES - a group of infectious diseases common to animals and humans (diseases such as tuberculosis, anthrax, brucellosis, rabies).

ANTHROPONOSES - specific only to humans. M: hemorrhagic fever.

The mechanism of the transmission of the pathogen from one animal to another animal - the ways of the transmission of the infectious disease agent - is one of the main links, which consists of 3 stages: 1) transmission of the pathogen exit to the outside environment; 2) storage in the external environment; 3) entry into a new susceptible organism. This situation ensures the continuity of the epizootic process. Each pathogen has its own special mechanism of transmission to another organism.

According to the parasitism of pathogens in the body: ***monotropic*** - only in one tissue or organs m: rabies virus in nerve cells, paratuberculosis causative agent - in intestines, black causative agent - in muscles rich in glycogen, or **polytropic-pantropic** - parasitic in several tissues and organs: protein, plague viruses and x-zo. The location, reproduction and exit of the organism and transfer to another organism of each pathogen are unique and specific. In the mechanism of transmission of the causative agent to another healthy organism, it is necessary to pay attention ***to the stages of separation and entry of the causative agent into the body.*** The release of the pathogen from the body **is a physiological process** (through exhalation, saliva, urine, feces) or during ***a pathological process*** (cough, runny nose and eyes, diarrhea, vomiting, abortion and blood-sucking bloodsucking insects). Due to the large number of microorganisms and viruses, they are located in 4 anatomic-physiological systems of the body: digestion, breathing, blood circulation and the covering covering the body from the outside. That is why pathogens are transferred to other organisms in 4 ways: **oral, respiratory, contact and transmissive** .

One or several factors may be involved in the transmission mechanism of the pathogen. All these serve as ***ways for*** the pathogen to pass from one animal to a healthy animal . In epizootology, 4 ways of pathogens are scientifically based: **contact, air, water and food and transmissible**

1. Way of contact - direct or indirect contact of healthy and sick animals. Here, the skin, visible and eye, breathing, digestive and urinary systems mucous membranes serve as the gateway to infection. Direct contact with the bite of a rabid animal, during fertilization of an animal ((campylobacteriosis, YuRT, brucellosis), contact of organisms with each other in smallpox, protein and trichophytosis. It is worth noting here that the above-mentioned diseases are only contact or It is necessary to take into account that it passes not only by the way, but also by other ways.

2. Airway (respiratory) - the pathogen passes through the air in the form of liquid or solid particles to a healthy organism. In infectious diseases of the respiratory organs (tuberculosis, pasteurellosis, and other resp. diseases), when coughing, wheezing, and sneezing, the causative mucus can travel up to 10 m. The causative agents of protein, Newcastle, smallpox, tuberculosis, anthrax also pass through the dust.

3. Through food and water - **alimentary infection** . When giving various wastes from slaughtering - swine fever, anthrax, Auyeski; through milk and skimmed milk - tuberculosis, brucellosis, proteinosis, salmonellosis; through water - leptospirosis, escherichia, salmonellosis and x-zo;

Where there is no veterinary control - **raw materials -** skin, wool, horn, hoof, bone; **if you die manure, soil** ; in some cases, when rodent **droppings** fall into feed - leptospirosis, Auyeski, listeriosis, tularemia); **soil** infection in diseases - anthrax, anthrax, ringworm, bradzot, enterotoxemia, etc.; **manure is the basis of the fight against epizootics!**

4. Transmissive path! This path is also 2 different. **Obligate-transmissive** way - in which the causative agent passes only transmissively, cannot be transferred in any other way (equine YUE and Plague viruses) **Facultative-transmissive** - in which the causative agent passes by other ways is also transmitted by - anthrax Yuan, swine fever and x-zo.

Horizontal way - in which the pathogen spreads from the organism to the external environment. **Vertical way** - in which the causative agent passes through the egg, placenta and milk without leaving the organism (leukemia, sarcoma); through placenta and milk - YURT, swine fever, Auyeski. Through eggs - all poultry diseases.

Susceptible animals are the driving forces of the epizootic process - For the development of an epizootic process, it is not enough to have a disease-causing source and the factors of the pathogen's transfer from one animal to 2, there must also be **susceptible animals for it.** An animal that has not previously been infected or not vaccinated is 100% susceptible to some diseases (rabies, swine fever). However, all animals susceptible to salm., colibact., Auyeski, and mute diseases are not susceptible. Therefore, the contagion index is different (100 how many of the animals in contact with the pathogen become ill).

Herd immunological structure - herd immunity (group susceptibility)

The immunological structure of the herd depends on many factors and conditions, they can be divided into 2 groups:

Special and non-special.

the natural resistance of the animal organism **:** breed, age, gender, physiological state, feeding, zoog. placement, use, stress and suffering from other diseases.

Recovery from illness, vaccination for special immunity. In some cases, tolerance or allergy can be called instead of immunity.

The combination of the 2 factors mentioned above creates herd immunity.

Laws of development of the epizootic process and stages of epizootics - The driving forces of the epizootic process are characterized by a complex interrelationship. An animal infected with a pathogen pollutes the external environment with the pathogen, creates conditions for the transmission mechanism of the pathogen, and increases the sources of the disease, and new sources appear. As a result, all animals are infected with the pathogen and some of them die, and population immunity is formed in the rest, which leads to the cessation of the epizootic process due to the negative impact on the transmission mechanism of the pathogen.

STAGES OF EPIZOOTIA — consecutive development stages of epizootics based on a certain law. There are several (6) stages in the development of an

epizootic: inter-epizootic, pre-epizootic, development, escalation, extinction and post-epizootic stages.

Inter-epizootic phase – (silent phase) 2 epizootic outbreaks – e. a certain time between waves. This disease is characterized by the observation of the disease in the population by chance and ensures the continuous continuation of the epizootic process, the disease does not multiply and spread widely. In the herd, there are more animals carrying pathogens and animals with latent infection. Most animals remain immune, but the number of susceptible animals is also increasing day by day.

In the pre-epizootic stage , conditions for epizootics are created, the number of immune animals decreases sharply, newborns do not have it at all, and as a result, **the number of sick people** increases. Pathogens are released a lot, as a result, the number of sick people increases.

E **development of pizootic In this stage,** the disease is widespread. They show characteristic clinical signs of the disease. At this stage, the number of immune animals that have recovered from the disease also increases.

E **pizootic promotion stage** i. **The number of patients** increases dramatically over a period of time. The disease begins to be chronic at the same time as acute. Recovered and immune animals in the herd

Extinction of epizootics As a result of the increase of recovered and immune animals **in** the herd, the phase of epizootic extinction begins. The mechanism of transmission of the pathogen is derailed, the disease is asymptomatic or atypical, and the chronic course increases.

The stage after the epizootic begins . The next period is the inter-epizootic stage, during which epizootic foci and morbidity are greatly reduced

Periodicity and seasonality of epizootics.

PERIODICITY OF EPIZOOTICS - recurrence of the intensity of the epizootic process in the same place after several years. Examples of diseases characterized by the periodicity of epizootics are rabies, anthrax and other diseases.

Intensity of manifestation of the epizootic process - speed

There are the following concepts that represent the degree of spread of the disease.

SPORADIC DISEASE *(gr.* ***sporadieos*** **-** scattered) - occurrence of infectious diseases by chance rarely, occasionally, in some cases (botulism, measles, rabies, anthrax, ringworm, actinomycosis).

EPIZOOTIA is the average level of the intensity (severity) of the epizootic process, the spread of infection beyond the boundaries of one unhealthy place and spread throughout the region, even the republic and countries (pasteurellosis, salmonellosis, brucellosis, tuberculosis, swine fever, etc.) lat.).

PANZOOTIYA *(gr.* ***Pan-*** everyone **+** ***zoon*** **-** animal) **-** the most intense high level of the epizootic process is an epizootic of animal diseases that covers several countries and continents. For example, protein, parr. influenza, rinderpest.

Factors affecting the passing of the epizootic process

The role of the source of the disease and the mechanism of transmission of the pathogen in the epizootic process

The driving forces of the epizootic process or the main links of the epizootic chain are *the source of the disease, the mechanism of transmission of the pathogen, and susceptible animals.*

The effect of animal resistance on the course of epizootics . In animals with high resistance, in herds that have recovered from the disease, in vaccinated herds, the disease passes easily. Anthrax is also chronic in some animals, especially pigs.

Effect of natural climatic conditions on epizootics

In Africa, Rift Valley fever, Nairobi k, OAO', ChAO' nodular dermatitis of cattle, in America - viral encephalitis of horses, Venezuelan encephalitis, in Europe - viral encephalitis of sheep (Born k), viral encephalitis of pigs (Teshen k.). But some CAOs moved from Africa to Europe - Spain, France, Italy, Portugal, Russia, and even Cuba.

EPISOTOTIC OUTBREAKS AND NATURAL OUTBREAKS

EPIZOOTIC FOCUS - buildings, pastures and livestock farms where a highly dangerous infectious disease has been recorded in animals is considered a disease spreading center, i.e., the location of the source of the disease - a certain area. The causative agent passes from the epizootic focus to a healthy animal. Its activity varies.

An anthropurgical outbreak is a natural anthropurgical epizootic outbreak that appears anew under the influence of human activities. For example, as a result of development of new lands, epizootic outbreaks of carnivore plague, trichenellosis, and anthrax occur.

Primary, New outbreak — a new or recent epizootic outbreak. In such foci, the number of newly infected animals increases day by day, and for this reason, the risk of spreading the disease increases. This situation is more common in diseases such as swine fever.

Stationary outbreak - in stationary or stable epizootic outbreaks of a certain disease after a certain period of time under the influence of certain conditions (the presence of germ-carrying animals, rodents and the presence of the causative agent in the external environment) resistance, etc.) to appear several times. For example: an anthrax outbreak.

Fading hearth — disease control results in the recovery of diseased animals and reduces or stops the occurrence of new diseased animals. This reduces the risk of spreading the disease.

Control questions

1. Explain the epizootic process .
2. The mechanism of transmission of the causative agent from one animal to another .
3. Suspicious animals are the driving forces of the epizootic process .

4. Development laws of the epizootic process and stages of epizootics .
5. Periodicity and seasonality of epizootics.
6. Epizootic foci and natural foci.

SUBJECT: MEASURES AGAINST EPISODE . VETERINARY SANITATION

Lecture	**Comprehensive control measures against epizootics i**
Lecture teaching technology	
Study time: 2 hours	of students: 90 students
The form of training	Enter. Visual lecture
Lecture plan Epizootic control principles and main tasks. P rophylactic and health measures. Preventing the emergence and spread of infectious diseases Sanitation measures and elimination of infectious diseases Disinfection and its types	P rophylactic and health measures. Preventing the emergence and spread of infectious diseases Sanitation measures and elimination of infectious diseases
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Epizootic control principles and main tasks. P rophylactic and health measures. Preventing the emergence and spread of infectious diseases Sanitation measures and elimination of infectious diseases Disinfection and its types	*Results of educational activities:* Epizootic control principles and main tasks. P rophylactic and health measures. Preventing the emergence and spread of infectious diseases Sanitation measures and elimination of infectious diseases Disinfection and its types
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture

Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Epizootic control principles and main tasks. P rophylactic and health measures. Preventing the emergence and spread of infectious diseases Sanitation measures and elimination of infectious diseases Disinfection and its types *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov X.S, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov X.S, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4. Mirziyoyev Sh.M. Development strategy of new Uzbekistan. Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www. fvat@.academy.uzsci.net .

Epizootic control principles and main tasks. Epizootic control works - this are scientifically based systems of preventive and health measures. Its main task is to protect people from zooanthroponous diseases and to create a stable healthy state in terms of infectious diseases in order to increase their productivity without allowing the death of animals.

In practice, the fight against epizootics is carried out in 3 interrelated directions:

1. Protection of animals from foreign infectious diseases in healthy regions and settlements of regions and districts for all infectious diseases, preventive measures and measures to prevent the spread of diseases in all regions of the Republic.

2. To carry out activities of rehabilitation and elimination of disease in unhealthy farms and settlements regarding infectious diseases.

3. To protect people from diseases common to humans and animals.

Regardless of the tasks and directions of the events, the fight against epizootics must be based on the following principles:

- approach to the fight against infectious diseases from the state point of view;
- carry out calculation of infectious diseases;
- the direction of disease prevention is the main criterion;
- conduct the event according to plan and complex;
- in the prevention and elimination of this infectious disease

to identify the main link of the epizootic chain.

The fight against infectious diseases is strictly defined in the Veterinary Regulation of the Veterinary Act. The law clearly defines the rights and duties of veterinary specialists, animal owners, heads of farms, organizations and enterprises in the fight against infectious diseases and their prevention. In addition to the law, there are "Manuals" for the control of each infectious disease, in which the implementation of all required expressions is mandatory for veterinary specialists, animal owners, managers of farms, organizations and enterprises.

Preventing the emergence and spread of infectious diseases - is the 2nd main principle and task of veterinary service, because prevention of disease is easier than fighting against it. That is why in the fight against infectious diseases, the main focus is on its prevention. Infectious disease control plans are a directive plan, its implementation is mandatory, material and technical resources are allocated to it by the state.

If the fight against infectious diseases starts on time, the results will be high. Therefore, it is very important to report the disease on time. According to the regulations, animal owners, heads of farms, organizations and enterprises must inform a veterinary specialist about the disease in a timely manner. If the animal dies suddenly, if there is more head discharge in poultry, the veterinarian immediately ***informs the higher organization about it.*** must do. It is necessary to immediately begin to determine *the diagnosis* and take measures to prevent the spread of the disease. It is necessary to inform ***the Minister*** about anthrax, protein, ulat within 24 hours . Every infectious disease situation must be taken into account. Analysis of the report of an infectious disease during a certain period allows to predict its occurrence and spread.

The principle of the comprehensiveness of measures to combat epizootics is the combination of measures against the three driving forces of the epizootic process, i.e. 1st - isolation and neutralization of the causative source of the disease, 2nd - the transfer of the causative agent to another animal. disconnection, elimination and 3rd – to increase the general and special resistance of animals. These measures are the fight against epizootics
must be reflected in the plan.

General and special prevention - *prevention of infectious diseases* is a system of state measures to prevent the occurrence and spread of disease in healthy farms and throughout the country. Preventive measures carried out at the country level consist of the following:

a) protection of the territorial border of the country against the introduction of infectious agents of animal diseases from foreign countries;

b) carrying out veterinary-sanitary control during collection of animals, driving, transportation of animal products by road, rail, air transport;

c) conducting strict veterinary-sanitary control at markets, exhibitions and other places of stock gathering;

g) carrying out veterinary-sanitary control in meat plants, poultry houses and enterprises engaged in transportation, storage, processing of animal raw materials (milk, meat, skin, wool, etc.);

d) protection of livestock farms from the entry of pathogens of infectious diseases from unhealthy places and organization of preventive measures in farms and settlements;

e) animal insurance and campaigning on infectious diseases.

General prevention **-** It is always and everywhere veterinary-sanitary and organizational measures aimed at prevention of animals from infectious diseases. This group includes:

- controlling the implementation of relevant restrictions and laws and regulations in mixing animals, transporting them and their products, and organizing herds, herds and farms;
- preventive quarantine of animals arriving in the country or farm;
- selection of animals for genetic resistance;
- feeding with nutritious feed, placing them in a zoohygienic enclosure; to follow the principle of "all busy - all busy" in the use of buildings;
- control the health of animals on a planned basis, isolate and treat the sick in time;
- regular cleaning and disinfection of barns, used inventory, territory;
- timely cleaning of barns, their disinfection and disposal of dead and various biological wastes;
- regular deratization, disinfection and disinsection;
- maintaining high sanitary conditions of pastures, watering places and cattle roads;
- organization of livestock farms and farms in the form of a closed enterprise, care of calves within the farm, transportation of carcasses and cows, etc.;
- providing farm workers and servants with special clothes, footwear, personal protective equipment;
- Livestock farms must meet veterinary and sanitary requirements.

general preventive measures against epizootics is only the prevention of sick people is not limited to doing, if an infectious disease appears, measures are immediately taken against its spread. Therefore, *against the above-mentioned epizootics preventive measures* regardless of whether or not there will be an infectious disease should be conducted regularly and everywhere.

Special prevention is a special event aimed at a specific infectious disease.

Special prevention includes:

- special diagnostic examination (m: tuberculinization, serol. bruce);

- isolation of the patient, mandatory quarantine and observation of the patient;
- use of therapeutic drugs;
- ***conducting immunoprophylaxis*** based on a plan against a certain infectious disease in a healthy farm with various special preparations (vaccine, hyperimmune blood serum, immunoglobulins) . It is *preventive* , if it is vaccinated after the appearance of the disease in the farm - mandatory called vaccination.

Immunoprophylaxis methods and special means - Vaccination of animals is the 3rd stage of the epizootic chain in the anti-epizootic system - a special measure aimed at susceptible animals. This monovalent vaccine is specific to one disease, and polyvalent vaccines are made from the strains of the disease, which gives immunity against only those diseases.

Types of immunization: ***active*** *and* ***passive***

A vaccine is an antigen preparation prepared from a microbe or its product isolated during life, and when it is injected into the body, it creates immunity against an infectious disease.

According to the preparation method: it will be ***alive*** and ***deactivated*** .

Deposited inactivated vaccines - various adsorbents: saponin, aluminum hydroxide-hydroxyl, mineral oil and others are added.

Chemical vaccines are prepared from a certain active soluble antigen (polysaccharide, polypeptide, lipid) of bacteria.

Anatoxins - mb toxins lost their toxinogenicity and retained their antigenicity under the influence of formalin or heat. *Antitoxin anatoxin.* ***Associated with –*** against several patients.

Routes of delivery : subcutaneous, intermembrane, intradermal, aerosol, mouth.

Passive immunization. *Simultaneous vaccination* - ***active*** *and* ***passive*** at the same time . This reduces the active im-t.

Organization of vaccination - when choosing the method of immunization, epizootic status is taken into account and they must be healthy. It is necessary to use a separate needle for each animal. It is necessary to fix the animal. You should not vaccinate a sick, thin, weak, dark cow, animal on the day of birth, they can be isolated and vaccinated with hyperimmune blood serum first, after 10-12 days. Vaccination should be carried out under the supervision of a doctor. After the vaccination is completed, a document is written and signed by the doctor and the participants.

Basic veterinary requirements for enterprises engaged in livestock breeding or its products of various categories -

a). ***Protection of state borders from animal infectious disease agents;***

There are veterinary checkpoints on all border roads, airports, railway stations of the republic. There are more than 90 of them, and all species of animals, poultry,

bees, fish and their products (skins, wool, horns, hooves) coming from all neighboring countries undergo strict veterinary control. will be under control, during that period they will be checked for brucellosis, tuberculosis, leukemia, campylobacteriosis and other necessary diseases, sick ones will be sent back or they will be slaughtered here in agreement with the experts of that state, without payment. If they are healthy, they are kept separately without adding them to local goods. If dangerous diseases are found near the borders of the neighboring country, a 30 km wide area of bush pasture will be left, where animals will not be grazed.

b). Veterinary-sanitary control of all animals and their products transported in transport;

Transported from a healthy area for infectious diseases only. Only the meat is transported from the unsanitary area to the plant. Transported animals do not have any contact with local livestock.

c). At the place where animals are gathered, market, exhibition, artificial escape points veterinary-sanitary control;

g). Veterinary-sanitary control in places where animals are slaughtered, where their products are processed, stored and sold;

d). Veterinary-sanitary control in places where animal raw materials are collected, processed, stored and disposed of (warehouses, bases of organizations that collect raw materials, leather tanning and wool cleaning, and meat and bone meal production plants);

It is not allowed to leave animal raw materials from an unhealthy point due to an infectious disease. The raw material of anthrax, scurvy, rabies, mange and other very dangerous diseases is burned and destroyed in that place. In the spread of the disease, it is important not to lose the corpse in time.

eat). Decontamination and disposal of manure

In highly dangerous diseases (anthrax, rinderpest, black fever, African swine fever, bradzot, African horse sickness, mange, epizootic lymphangitis, avian-Newcastle (false plague), influenza and myxamatosis of rabbits) manure is burned

Preventive measures in livestock farms

Prevention of infectious diseases in healthy farms is based on general and special measures, which are not to introduce pathogens of infectious diseases from outside the farm, to prevent the spread of the disease, to increase the general and special resistance of farm animals. directed. The reliability of these measures depends on the farm's isolation from other livestock objects (closedness), on the care of animals with nutritious feed, and on keeping them in accordance with zoohygienic requirements.

In order to protect the farm from infection that may enter from outside, it is necessary to observe the following minimum rules:

a) fill the farm only with animals brought from the farm that are healthy in terms of infectious diseases;

b) keep the animal brought to the farm under preventive control for 30 days in a separate place and during that period perform all necessary diagnostic tests for infectious diseases and other veterinary requirements;

c) collect feed only from the area that is healthy in terms of infectious disease

g) use of all post-slaughter, biological and food wastes after thermal treatment, regardless of their source;

d) prevent direct and indirect contact of healthy and unhealthy farm animals;

e) keep pastures, watering places, roads where animals are driven in order;

j) not to allow strangers to enter livestock farms and buildings;

z) reliable protection of the farm from all kinds of insects, rodents and wild animals;

i) routine veterinary control of clinical and movement of animals belonging to the population;

k) regular monitoring of the epizootic condition of settlements, farm and district areas and paying attention to wild fauna;

l) regular control of the sanitary conditions in livestock products and fodder collection, processing and storage enterprises;

m) propaganda of veterinary knowledge among the population and cattle breeders in various forms (newspaper, radio, television).

In healthy farms, general and special preventive measures are carried out in 3 interrelated directions:

1. Selection-genetic direction.
2. Increase the general immunoreactivity and resistance of animals.
3. Special immunoprophylaxis.

Increasing the general immunoreactivity and resistance of animals is currently the main direction, which is based on the veterinary-sanitary culture of animal husbandry and consists of the following:

- feeding animals with premixes with curative-prophylactic effect based on the requirements of complete and rational hygiene in return for the presence of a suitable feed base;

- performing sanitary-technical operations on the farm: timely collection of manure, corpses, and biological waste and work directed towards cleanliness, including disinfection, disinsection of buildings and the farm area, in a word, veterinary- maintaining sanitary condition based on zoohygienic requirements;

- establishment of veterinary control on the basis of a regular plan for dispensation of animal health, ensuring the immunological structure of the herd, sanitary quality of feed and zoohygienic parameters of buildings at the required level;

- achieving a high indicator of the amount of products obtained from animals;

- availability of quarantine farm, isolators, veterinary facilities, special disinfecting equipment, medicines, special clothing and other personal protective equipment for the operation of highly qualified veterinary specialists in the farm;

- achieving regular propaganda of veterinary knowledge among livestock breeders and people.

Special immunoprophylaxis is currently the most widely used, especially in farms that are not reliably protected from foreign pathogens, in farms and settlements that are not fully isolated from other farms, and in cases of infectious diseases. is a very useful event in a dangerous area,

In order to prevent infectious diseases in accordance with the Veterinary Regulations, livestock farms, managers of livestock enterprises, farmers and animal owners are responsible for:

a) achieve protection of livestock farms and their animals from infection that may enter from outside; bringing in and taking out new animals to the farm without the permission of a veterinary specialist, mixing their groups together; unsupervised keeping of a new animal in the farm area and in the settlement and giving feed that has not been checked for veterinary sanitary quality; to organize a veterinary facility on the farm and prevent outsiders from entering it; compliance with the rules for keeping animals in preventive quarantine;

b) to achieve a high level of veterinary sanitary conditions in livestock farms, pastures, drinking places, buildings where animals, their products and feed are kept, and rooms where livestock breeders are undressed and fed; timely removal and disposal of animal carcasses, manure and other waste, keeping the buildings and the farm area tidy, disinfection, disinsection and deratization according to the recommendation of a veterinary specialist;

c) strict observance of veterinary-sanitary and zoohygienic requirements in the construction of animal husbandry buildings, feed storage warehouses, veterinary facilities, aviaries and buildings for the processing of livestock products;

g) to observe the veterinary-sanitary regulations in the accommodation, feeding and use of animals, to organize all measures necessary for the diagnosis, treatment and elimination of the disease in the event of an outbreak; compliance with the rules of separation and quarantine of sick animals;

d) to create the necessary conditions for seeing animals, diagnostic examination, vaccination and treatment, as well as conducting veterinary activities at the request of a veterinary specialist; immediately notify the veterinary specialist about the accidentally dead animal and transfer the pathogen to another farm until it arrives. Take measures not to spread to the population;

Activities to educate the public about animal diseases and to protect people from zoonotic diseases .

Epizootological activities will be effective if cattle breeders and the entire population are introduced to the knowledge about the origin and spread of the infectious disease and the possibility of it being transmitted to humans.

During the resolution of epizootic problems in farm conditions, the veterinarian should take measures to protect people from diseases common to animals and humans (br, tbs, rabies, anthrax, leptospirosis, dermatomycosis, etc.). For this:

- providing information to animal keepers on regular infectious and highly dangerous diseases;

-providing livestock workers with special protective equipment (gown, cap, mask, rubber boots, gloves, goggles, goggles, space suit, etc.), hand wash, soap, disinfectants, towels;

- to provide workers on the farm with a kitchen where they eat, a rest room, a toilet and a room where ideological work is carried out;

- provision of equipment for the decontamination of animal and its by-products contaminated with pathogens: (pasteurizer, disposal and boiling pot. Immersion, disinfectant, etc.).

Sanitation measures and elimination of infectious diseases

In the fight against infectious diseases, complex measures are aimed at the 3 driving forces of the epizootic process: 1) isolation and neutralization of the source of the disease; 2) learning the mechanism of transmission of the pathogen from one animal to another; 3) increase the general and special resistance of susceptible animals to the disease. These measures must be reflected in the plans for the fight against infectious diseases.

1. Measures aimed at eliminating the causative source of the disease *- diagnostics: epizootological, clinical, patholog-anatomical, allergic, serological, bacteriological, vrusological, biotesting.*

Epizootics are eliminated by isolating and neutralizing the causative source, that is, healthy animals are protected from causative agents. Quarantine measures prevent pathogens from entering healthy farms.

Organization of mass inspection of animals - ***definitely sick, animals suspected of disease and animals suspected of damage.***

2. Measures against the transfer of the pathogen from one animal to another *- are directed against contact, alimentary, respiratory and transmissive ways.*

3. ***Activities that increase the resistance of animals***

Disinfection and its types:

- preventive;
- daily;
- mandatory
-final

Disinfection methods and tools:

-physical-mechanical, light, drying, high temperature, iron, boiling water, steam, fire, gamma ray, ultrasound;

- chemical alkalis, acids, chlorine substances, potassium permanganate, phenol, formalin, hydrogen peroxide;

-biological-biothermal;

Control questions

1. Epizootic control principles and main tasks.
2. P rophylactic and health measures.
3. Preventing the emergence and spread of infectious diseases
4. Health measures and elimination of infectious diseases .

Disinfection (fr. des - elimination and Latin infectio - infection) is a method of disinfecting and cleaning objects of the external environment aimed at eliminating pathogens . Disinfection has a destructive effect on pathogens, completely eliminates the mechanism of transmission of the pathogen, which is considered the second driving force of the epizootic process, in a word, allows the epizootic process to

occur. does not eat Therefore, disinfection is one of the most necessary measures in the complex of measures to combat epizootics in the prevention, recovery and eradication of any infectious disease.

the term disinfection (cleansing objects from pathogens) from *disinfection* (decontamination) of pathogens and their life products - toxins, and *sterilization* - from the removal of pathogenic and non-pathogenic (saprophytic), conditionally pathogenic microorganisms and viruses. necessary.

disinsection along with disinfection in the system of combating epizootics - lot. insectum (insect) and *deratization* - Latin. rattus (rat), that is, requires the use of measures aimed at the elimination of insects and rodents that transmit the causative agents of many infectious diseases. The importance of each measure comes from the epizootological characteristics of a specific infectious disease, and the choice of how to affect the pathogen depends on the specificity of the mechanism of transmission of the pathogen, the way of spread and factors. For example, in transmissible diseases - disinfection and disinsection, and in the case of certain zoonotic diseases (tularemia, listeriosis, leptospirosis, etc.), disinfection, disinsection and deratization measures are required.

Disinfection, disinsection and deratization measures are of primary importance in the fight against epizootics, as they eliminate both the source of the disease and the transmission mechanism, the spread of the disease. Therefore, it is necessary to hold these events not only in livestock farms, but also in meat processing plants, poultry farms, milk and leather factories, and other enterprises that collect, transport and process livestock products.

Disinfection types and objects. Disinfection is divided into preventive, mandatory, current and final.

Prophylactic disinfection is carried out in healthy livestock farms to prevent infectious diseases, that is, to prevent pathogens from entering farms and the spread of pathogenic microorganisms and viruses among animals. Such disinfection dramatically reduces the number of pathogens in the farm, prevents its reproduction in the external environment. This event eliminates not only pathogens, but also opportunistic pathogens, mixed infections of viruses and bacteria, as well as ubiquitous pathogens. In small livestock farms, livestock *products* preventive disinfection in processing plants 2 times a year, in a fattening farm every time after animals go to meat, maternity hospital, calf house at least 1 time a month, preventively when the cages are empty of calves before placing a new calf and in chicken houses before and after the slaughter of animals.Prophylactic disinfection is required after mass anti-epizootic events (vaccination, tuberculinization, blood sampling, etc.), as well as after the gathering of animals at markets, exhibitions.

In large poultry factories and farms, it is necessary to organize a sanitary day once a month, to carry out current repair work in the building, to clean it from garbage, uneaten feed and preventive disinfection of all objects in it.

In sheep farms, preventive disinfection is carried out after mechanical cleaning after driving the sheep to the pasture in the spring and before entering them into the fold. In the fall, it is advisable to carry out disinfection together with disinsection.

Mandatory disinfection is carried out when an infectious disease appears in the farm. Compulsory disinfection is current and final, and is used to eliminate the primary focus of pathogenic microorganisms and prevent their accumulation in livestock farms (poultry) infected with infectious diseases.

Current disinfection is used at all stages of the period of health of the farm or farm to reduce the level of contamination of external objects with microorganisms and viruses and to reduce the risk of re-infection of animals in the farm. Based on the characteristics of each infectious disease, it is carried out from the moment the disease appears in the farm, regularly after each disease case is detected. Current disinfection is an activity aimed at eliminating pathogens (viruses, bacteria) released from sick, disease-carrying animals in the external environment. All inventory used for animal care, isolators where sick and suspected animals are kept are disinfected daily. This event will continue until the disease is completely eliminated from the animals on this farm.

Current disinfection periodicity and the list of objects to be disinfected, the nature of the disease, the epizootic situation of that disease, the specification of production technology, the natural and climatic conditions of the place where the disease occurred, and other features, as well as the current practices in the fight against this or that disease It is determined taking into account the requirements of the application.

Final disinfection is carried out before quarantine or restriction from the farm (farm, point, specific area), that is, after sanitization. The final disinfection consists of two consecutive operations: deep mechanical cleaning and disinfection. The purpose of the final disinfection is to completely eliminate pathogens in the external environment as a result of the thorough disinfection of floor holes, wall cracks, and places that have not been affected by disinfectants after the current disinfection. is a loss. All buildings, environment, vehicles, inventory, clothes and shoes, farm equipment contaminated with manure, animal excreta are disinfected. Disinfection is one of the mandatory measures for obtaining quarantine and restrictions imposed on highly infectious patients.

Disinfection methods and tools

Due to the fact that the objects to be disinfected are different, different disinfection methods and means are used to eliminate pathogens from them. 3 methods of disinfection: physical, chemical and biological are used to disinfect various objects.

Physical disinfection method. This method is the cleaning of external environmental objects from pathogens using physical means: mechanical cleaning, radiant energy, drying, high temperature, high frequency current and ultrasound.

Mechanical cleaning is the removal of the causative agent from the place where the animal is, together with manure, dust, leftover feed, bedding, to a place where there is no animal. The same process is carried out during ventilation of the building, air purification with the help of ventilators, air and water filtration. When cleaning the building, it can be cleaned with a shovel, broom or water under strong

pressure. It is possible to use 1-2% hot (35-40 ^{0} C) caustic soda instead of water. Whitening outdoor objects with lime, painting them with paint or washing them with soap and keeping the farm tidy is also a type of mechanical cleaning.

Use of radiant energy. In Uzbekistan, free sunlight inactivates most non-spore-forming bacteria and viruses in the outdoor environment. Especially direct and scattered sunlight has a destructive effect on the triggers. A mercury lamp is used as an artificial light source.

BUV-15, BUV-30, BUV-30-P with a wavelength of 254-257 nm, a power of 15-30 W, and a power of 30-60 W for the disinfection of buildings, especially calf houses BUV-60-P and wall-mounted N-60 and ceiling-mounted P-60 lamps are used.

Drying also has a negative effect on the life activity of the instigators. In a non-aqueous environment, the pH changes, and their rate of reproduction decreases sharply. Vegetative bacteria lose their pathogenic properties in such an environment. Once they enter the body, they cannot harm it. Drying is used for decontamination of leather, wool and wetlands.

Effect of high temperature. Boiling, hot steam, dry heat, and flame incineration also completely inactivate triggers. They die when the proteins in the protoplasm of the bacterial cell coagulate under the influence of *hot steam* and unbearable dry heat. Cotton fiber tissues, glassware, metal tools are neutralized in drying cabinets (under the influence of dry heat). *Ironing is used* to disinfect gowns, masks, special clothes, bandages . Inactivation of pathogens by *boiling* is a method used every day in veterinary practice. It kills vegetative (15-30 minutes) and spore forms (45-120 minutes) of bacteria and all types of viruses.

Steam (*autoclave*) *is* a method of disinfection and sterilization that reliably kills pathogens. It is a device that uses high-temperature water vapor (115-120 ^{0} C) under a pressure of 1.5-2 atm. *Fire flame - soot* is usually used to burn feed residues, bedding, manure, dead animal carcasses contaminated with irritants. In addition, the flame is used to disinfect the place where the dead animal is lying (soil), inventory used in animal care, metal containers, dog and rabbit cages, laboratory tools and equipment, the animal pathological-tomic dissecting table, and metal carts carrying the corpse. It should be noted here that the device connected to 2 120 kg gas (methane) cylinders and the device for blowing air into the flame (ventilator) are installed on the tractor and the metal furnace for cremation is placed on the 2-wheeled trailer. The organization of a mobile crematorium is an urgent problem . *Because it is difficult to economically justify the establishment of a stationary crematorium in every farm, livestock farm, but* it is necessary from an epizootological and economic point of view to make one *mobile crematorium* on the district level, which can be moved anywhere with the help of a tractor. This device can be used in any farm as a crematorium and flame disinfection.

Chemical disinfection method. The chemical method of disinfection is the use of various chemical agents, often in the form of water solution, less solid, dispersable, gas and aerosol. Chemical means are most often used in veterinary practice for disinfection of livestock premises.

Aqueous solution of disinfectants (soaking method) is mostly used using

various sprayers. Later, spraying with very small droplets (diameter 0.1-0.5 mm) is started. In this case, the consumption of the disinfectant is slightly reduced. In this way, disinfectant consumption (0.2-0.5 l/m ^{2}) is 4-10 times less and uniformly covers the entire surface of the object. Therefore, this method is epizootologically and economically effective.

One of the most advanced ways of chemical disinfection is spraying a water solution in the form of an aerosol. This method is used for disinfection of indoor calf houses, pig houses, and poultry buildings, and in some cases simultaneous disinfection. Disinfection in the aerosol form allows to reduce the amount of chemicals by 3-5 times. The effectiveness of this method depends on hermetically closing the room. It is desirable that the room temperature is not lower than +15 ^{0}C and the humidity is around 60-95. TAN, PVAN, AG-UD-2 devices are used to spray the aqueous disinfectant solution in aerosol form. The treatment room is closed for a certain time. The duration of this depends on the concentration of the disinfectant used, the bactericidal effect and the sensitivity of the pathogen to it. After a certain period of time, the room is opened and well ventilated, the drinking devices and troughs are washed with clean water.

In disinfection, in addition to the bactericidal effect of disinfectants, protection against corrosion of metals is also taken into account. M: chlorinated lime, sodium hypochlorite, acids, caustic sodium activates the corrosion of metals, especially zinc-treated tools. In contrast, formaldehyde does not harm metal.

Alkalis for disinfection (caustic soda, caustic soda), acids (phenol - carbolic acid), formaldehyde, active chlorine preservatives (chlorine lime, chloramine B, slaked lime), phenols, salts of heavy metals and other substances are used.

Alkalis. *Unrefined caustic soda is used* for disinfection . A 3-4 percent concentration of the drug is used to eliminate protein, swine plague, parainfluenza-3, influenza viruses from the environment. The solution is applied hot (60-80 ^{0}C) after a three-hour break. A 10 percent hot solution is disinfected against anthrax with the addition of a 10 percent solution of table salt. In tuberculosis and fungal infection, a mixture of 3% caustic sodium and 3% formaldehyde solution is used in a 1:1 ratio.

When using caustic soda, it is necessary to strictly observe the safety of the technique and be extremely careful. If it gets on the skin, the drug can cause deep burns. It can cause poisoning, vomiting, bleeding, severe pain, and difficulty urinating in the body. It is necessary to wear protective glasses to protect the eyes. Weak organic acids, for example, a 1-2 percent solution of boric acid, act as an antidote. In addition to caustic sodium, caustic potassium, lime (SaO), slaked lime (Sa(ON) $_2$), calcium soda (Na $_2$CO $_3$), ash are used in disinfection.

Acids. Relative to alkalis less used. In practice, when treating skins against anthrax, *hydrochloric acid is mainly* used for disinfection. It is also used in the preparation of monochlorinated iodine. Sulfuric acid in its pure form is used less frequently in disinfection. It is used in preparation of sulfate-corbol mixture. *The sulfate-corbol mixture* is used more often in disinfection.

From organic acids , *lactic, formic, vinegar, oxalic* (oxalic) acids are used in aerosol form for disinfection of poultry houses, places where skin and wool are kept.

Dezoxon (a mixture of perhydrol and acetic acid) is also used in disinfection .

Chlorine-preserving disinfectants. Chlorine-preserving disinfectants include chlorinated lime, hypochlorites, chloramine, monochlorinated iodine, and others. They are very strong oxidizers, like potassium permanganate and perhydrol. They have a destructive effect on organic substances (proteins) of microorganisms and viruses as oxidants. Chlorine gas reacts with the wet (water) part of the catalysts and turns into hydrochloric acid. Free *oxygen* scavengers formed as a result of the reaction it oxidizes organic substances and has a destructive effect. Chlorine renders the protoplasm of the microorganism cell in an inert state and has a strong bactericidal effect, but its effect depends on the presence of moisture in the pathogen. They are used in the disinfection of drinking water, wagons and closed livestock, poultry houses. *The advantages of chlorine* are that it is cheap, easy to transport in a balloon, it has a strong bactericidal effect, it is not difficult to prepare a specific concentration for disinfecting water and building surfaces. However, it does not corrode fabrics, paint, metals, and paints and quickly poisons animals.

Chlorinated water is used for disinfection of livestock buildings, floors and walls, as well as drinking water.

Chlorinated lime. Chlorine lime consists of a brownish-white hygroscopic powder with a pungent odor. It has a good disinfectant and deodorizing effect against microorganisms and viruses. 2-25 percent chlorine solutions are used for disinfection. 10 percent sodium chloride (table salt) is added to the solution to increase its anti-microbial and anti-viral properties. The solution is prepared in a wooden barrel. When disinfected with a solution, it irritates the eyes and respiratory tract. Therefore, animals should be removed from the building during disinfection. Due to the strong effectiveness of the drug, it is impossible to disinfect threading and metal objects. Chlorine lime and other chemicals containing chlorine are used to eliminate infectious diseases such as plague, tuberculosis, brucellosis, protein, campylobacteriosis, salmonellosis, pasteurellosis, Aujeski's disease, listeriosis from the environment.

Chlorine lime should be stored in a wooden container with a plastered mouth. Do not store in a bag as it may overheat and explode. Chlorine lime cannot be stored together with explosive and flammable materials.

Chloramine B. Chloramine B is a white, yellowish crystalline powder with a faint odor of chlorine. Easily soluble in water. For disinfection, 2-10 % solution is used in any object . Chloramine B contains 30% active chlorine.

Slaked lime. Slaked lime is a white, porous powder that hardly dissolves in water. Slaked lime is obtained by adding water to unslaked lime in a ratio of 1:1. It has disinfectant, deodorizing and anti-parasitic properties. 20 percent slaked lime is whitewashed three times at 2-hour intervals for disinfecting and whitewashing walls, ceilings, mangers, barns, manure bins, cages, beams, benches and other equipment. Drug consumption: 1 per 1 m^2 liter.

Calcium hypochloride is a yellowish powder with a chlorine smell, which is produced by passing chlorine through slaked lime. The amount of active chlorine in this drug is 80-90%. It dissolves well in water. The solution has very strong oxidizing

properties. It has 2.2 times more bactericidal effect compared to slaked lime. 5% and 10% solutions against bacilli are used for disinfection against bacteria.

Iodine monochlorine is used in the treatment of animals infected with trichophytia by applying it to their skin and in the disinfection of livestock and poultry houses, especially against fungi.

Phenols. They are rarely used in practice because they are less effective against spore-forming microorganisms.

Phenol (carbolic acid) consists of a hygroscopic crystal with a specific smell. Crystals are soluble in water, alcohol and oil. When exposed to air and light, the crystals develop a pink color. Phenol has anti-microbial and anti-viral, insecticidal and parasitic properties. Phenol has a strong effect on the skin of animals, inflammation and pain appear in the mucous membrane, then painless and deep, dry gangrene occurs. Phenol poisoning is very bad, the function of the central nervous system is disturbed, breathing, blood circulation, body temperature drop is felt. Death occurs when breathing stops. Cats are especially susceptible to phenol.

Livestock buildings, running water, animal care items are disinfected with a 3-5 percent solution of phenol. Phenol and its preparations (cresol, creosote, creolin, etc.) cannot be used in buildings where dairy cows and animals for slaughter are kept, because the unpleasant smell remains in milk and meat for a long time.

Cresol is less toxic than phenol, but has a stronger bactericidal effect. It does not dissolve well in water, therefore its mixture with other disinfectants (*sulphate-carbol-cresol mixture*) is more commonly used in practice.

Formalin is is a 35-40% aqueous solution of formaldehyde, a clear, colorless liquid with a pungent pungent odor, highly toxic to cells. A certain amount of formaldehyde (not formalin) is taken for disinfection. Formaldehyde has the ability to form a very strong bond with protein and bond with water vapor.

Formaldehyde. As a result of the application of formaldehyde in aqueous solution or gas form, it has a bactericidal, sporicidal and viricidal effect on microorganisms and viruses. The basis of its destructive effect on pathogens lies in the property of formaldehyde to form a strong bond with protein. As a result of such an effect, the protein undergoes denaturation. It is one of the universal disinfectants that are used in livestock buildings and have a fatal effect on all types of pathogens in different concentrations. It can also be used to disinfect precious metals. If you add alkali - caustic sodium to it, the effect will increase even more. A 2-4 percent solution is used as a disinfectant against protein, swine fever, saramas, Auyeski's disease, pasteurellosis, salmonellosis, chicken pullorosis, sheep pox, as well as tuberculosis, dermatomycosis and other infectious diseases.

During disinfection, the temperature in the building should be around 25-30 degrees, and the humidity should be 95-100 percent. Solution consumption is 100-200 ml per 1m ^{3} and lasts 10-24 hours. 3 percent formaldehyde and 3 percent caustic sodium solutions are used for disinfection of farm fences.

In addition to formalin, paraform, lysoform, thiazone, metaphor, fospar and other preparations of formaldehyde are used for disinfection.

, *glutaraldehyde* , a light yellow liquid with its own odor, is used. For

preventive disinfection, it is used in the form of a 0.3 percent solution at the rate of 1 liter per 1 m ^{3} · A 0.5 percent solution of glutaraldehyde at the rate of 0.5 liters per 1m 3 is used against the causative agents of saramas, swine fever, campylobacteriosis, pasteurellosis, listeriosis, brucellosis, protein and other infectious diseases. In tuberculosis, a 1% solution at the rate of 1 liter per 1m2 is used for 4 hours, in anthrax, a ^{2} % solution at the rate of 1.5 liters per 1m2 is used for 3 hours, and in the case of ringworm and aspergillosis, a ^{4} % solution at the rate of ^{1} liter per 1m2 is used for 24 hours.

Glac and *Glac S preparations* of glutaraldehyde are also used for disinfection in infectious diseases.

Before cleaning and disinfecting the livestock and poultry houses, animals and poultry are removed, equipment that can be damaged by water and disinfectant solutions is removed or covered with a polyethylene film, and if necessary, manure, rodents and other residues are collected. is used. Full mechanical cleaning means that the color of the surface of the material is clearly visible and large parts of manure, feed and other mechanical waste are not visible even in hard-to-reach places. For disinfection, disinfectants approved by the veterinary department, having a certificate of the manufacturing plant, and their compliance with state, industry standards or technical conditions, are used.

Buildings, equipment, inventories and other objects are treated by thoroughly wetting their surface with a solution of chemical disinfectants. An aerosol obtained from disinfectant solutions is also used in the disinfection of closed buildings. Individual objects are disinfected using thermal, gas, steam, air, radiation, paraformalin and other methods of disinfection.

Depending on the condition of the object and its level of cleaning, disinfectant solutions are prepared at the rate of 0.3-0.5 liters per m ^{2} for one time soaking.

According to the instructions of the veterinarian responsible for disinfection, the consumption of solutions can be increased in justified cases. When determining the consumption of the areas that need to be wetted with disinfection solutions, the area of the floor, wall, ceiling, partitions, external and internal surfaces of the equipment in livestock buildings and other objects is taken into account.

When treating the surface of the building with a disinfection solution, the following procedure is followed: first, starting from the exit point at the end of the building, the floor, curtains, equipment, walls are evenly wetted, then the ceiling, the floor in the passage is disinfected.

Animal care items and inventory used in this building are disinfected at the same time. If freshly slaked lime is used for disinfection, then the walls, partitions between the workbenches, ceilings and other objects that need to be whitewashed are treated, then the floor, manger, building and equipment are treated with another disinfection solution. After spraying disinfection solutions, the building is closed for 3-12 hours.

After disinfection, the building is ventilated until the smell of the drug completely disappears, milking drugs, mangers are cleaned of waste.

Before disinfection, the equipment taken out is wiped with rags moistened with disinfectants. After that, the equipment is put in place. The concentration of working solutions of disinfectants is determined based on the purpose of disinfection (prophylactic or mandatory) and the tolerance of pathogens to the effects of chemical disinfectants.

Concentration of disinfectants (formalin, paraformaldehyde, chlorinated lime, calcium hypochloride, glutaraldehyde, lysol, phenosmolin, sodium phenolate solution, caustic sodium, monochlorinated iodine, and calcium soda) used according to Caustic sodium, calcium soda and cresol solutions are used while boiling. A suspension of freshly slaked lime and calcium soda is used only for preventive disinfection.

In paratuberculosis and tuberculosis, caustic sodium or cresol, formalin or paraform contains 3 percent alkali and 3 percent formaldehyde, and in mycoses, it is used as an alkaline solution of formaldehyde in the proportion of 1 percent and 3 percent, respectively.

In poultry aspergillosis, all disinfectants except dezonon are used after soaking with 0.5 percent OP-7 solution or 0.3 percent OP-10 solution at the rate of 1 liter per meter ^{2} area, or they are added to the disinfection solution. 4 percent is used for washing horses with chlorinated lime and calcium hypochloride.

3 percent glutaraldehyde and cresol (formaldehyde-free) are used to disinfect vehicles after transporting animals with tuberculosis. Solution consumption - 0.5 liters per ^{m2} · Waiting time - 1 hour. In case of anthrax in fur-bearing animals, a hydrogen peroxide solution of 7% with 0.2% of lactic acid and the same amount of OP-7 detergent are used for disinfection. Treatment is carried out twice every 1 hour.

When fur-bearing animals and dogs are rabid, metal cages are burned in compliance with fire safety. When animals are infected with streptococcosis and colibacteriosis, the premises occupied by them are treated with a 2 percent solution of chloramine or desmol.

According to the tolerance of the main infectious agents of animals and poultry to chemical disinfectants, they are divided into groups such as *moderate, resistant, highly resistant* and *extremely resistant* .

causative agents of cambric diseases includes leukemia, brucellosis, leptospirosis, Aujeski's disease, pasteurellosis, salmonellosis, trichomonosis, campylobacteriosis, trinaposomosis, toxoplasmosis, infectious rhinotracheitis, parainfluenza-3 and viral diarrhea, contagious pleuropneumonia of sheep and goats, malignant tumor, infectious diseases of pigs atrophic rhinitis, dysentery, transmissible gastroenteritis, balantidiosis, hemophilic polyserositis, hemophilic pleuropneumonia, saramas, equine rhinopneumonia, poultry pullorosis, mycoplasmosis, myxomatosis of rabbits, diarrheal diseases of young calves caused by conditionally pathogenic microflora (proteus, klebsiella, morganella, etc.) .

resistant pathogens includes adenovirus infection, protein, rabies, smallpox, tularemia, ornithosis, diplococcosis, staphylococcus, streptococcus, plague, necrobacteriosis, aspergillosis, candidomycosis, trichophytosis, microsporia, other mycoses of animals and birds, chlamydia, rickettsiosis.)) infectious hepatitis, viral

hemorrhagic diseases of rabbits.

highly resistant to the effects of chemical disinfectants .

highly resistant group includes anthrax, lamb anaerobic dysentery, anaerobic enterotoxemia, bradzot, malignant tumor, blackworm and other spore infections.

Preparations for disinfecting buildings when animals and poultry are staying

For this purpose at the present time a hydrogen fairy (perhydrol), sodium hypochlorite, glutar aldehyde and iodtriethylene glycol are used.

Pergidrol solution used for disinfection is prepared from 30% pergidrol. To stabilize the effect of 3% hydrogen peroxide, 0.5% lactic or acetic acid is added to it. Poultry houses can be disinfected by sprinkling small drops with it. Its consumption is 100-200 ml/m ^{2} .

Sodium hypochlorite is prepared by dissolving 200 grams of calcium soda and chlorinated lime (25% active chlorine) in 1 liter of water. The finished solution is left to stand for 24 hours (stirred 5 times for 5 hours). Active chlorine content in diluted sodium hypochlorite It will be 5-6%. By diluting it with water, a working solution containing 1.5-2% active chlorine is prepared. It does not inactivate for 1 month in closed containers.

Chlorine-turpentine aerosol. This compound is prepared by mixing chlorinated lime and turpentine in a ratio of 4:1. As a result of the exothermic reaction, the smell of chlorine and turpentine is emitted into the air of the building. In 5-6 places of the building there is this solution in the tank and the air is disinfected all day long. After the solution is placed, the fan is working in the room.

Glutaraldehyde in the form of a 0.2% solution is effective in most bacterial and viral diseases.

Iodotriethyleneglycol is a black-red oily liquid with a special iodine smell. To prepare 1 liter of preparation, add 160 g of potassium iodine and 915 ml of triethylene glycol to 300 g of fine crystalline iodine powder and mix until completely dissolved. If there is a hermetically sealed container and a dark place, it can be prepared before disinfection. Aerosol of this drug has a good effect on infectious laryngotracheitis, infectious bronchitis and escherichia in unhealthy poultry farms. If healthy birds are treated with an aerosol of this drug, it cleans the air of the building and the respiratory tract of the bird from pathogenic and conditionally pathogenic microflora.

Manure can contain many pathogens of infectious diseases from infected animals, so it is necessary to collect and disinfect it in time.

The dung of animals infected with and suspected of anthrax, scurvy, mange, infectious anemia, rabies, bradzot, rinderpest is first disinfected and then burned.

Manure should be biothermally neutralized in cases of protein, swine plague and saramas, salmonellosis, tuberculosis, leptospirosis.

Biological method of disinfection. Biothermal disinfection of manure occurs due to the high temperature generated by thermophilic microorganisms that multiply in it. Disinfection is carried out in manure pits. A 3-meter-wide and 0.5-meter-deep

pit is dug in the farm area to prepare a manure pile. The bottom of the drum is covered with a layer of clay 15 cm thick. Then a layer of straw or clean, uninfected manure is placed in a thickness of 35-40 cm. Then, at a height of 2-2.5 meters, at an arbitrary length, the slope of the sides is 70 ^{0} C, and the manure to be neutralized is placed. Then the manure is sifted. During manure application, unsterilized manure or 10 cm thick straw is placed, followed by 10 cm thick soil. Manure is stored for 1 month in hot days and 2 months in cold days of the year for biological disinfection.

Checking the quality of disinfection. Prophylactic and mandatory disinfection in salmonellosis, saramas, plague, brucellosis, bird flu diseases and the quality of current disinfection in protein disease *, distribution and absence of Escherichia coli* in the outdoor facility , tuberculosis, protein (final disinfection), sheep and fowl pox , leptospirosis and viral hepatitis of ducks, determined by *the presence and absence of staphylococci* .

Bacteriological examination is carried out as follows. 2-3 hours after disinfection, 10-20 samples are taken from the floor, walls, corners, troughs (a 10x10 cm area is thoroughly wiped with a sterile swab moistened with a neutralizing disinfectant and placed in a glass container). Each swab is individually placed in a glass container containing a neutralizing disinfectant solution. The concentration of the neutralizing disinfectant solution is 10 times lower than the concentration of the disinfectant. Hyposulfite is used to neutralize chlorinated lime, acetic acid is used for alkaline solutions, and alcohol is used for formalin.

Samples are tested in laboratories on the same day. Swabs are thoroughly squeezed, the liquid is centrifuged 2 times (the liquid is diluted with sterile water in the 2nd), then the sediment is poured off and the sediment is inoculated into selective nutrient media. Disinfection is effective if *there is no growth of bacteria* in all nutrient media during preventive and final disinfection , or *if there is no growth in less than 90% of nutrient media* during current disinfection .

Mechanization of disinfection works. As a result of mechanization of disinfection work, it is possible to disinfect a very large area and the efficiency increases.

^{2} with 1 hose (mounted on a car), LSD-2 intended for veterinary-sanitary and economic activities, capacity 100 l/min, mounted on a trailer.

The veterinary disinfection machine (VDM) is intended for the complex performance of veterinary and sanitary measures. The following activities are carried out using this machine:

a) disinfection and disinsection of livestock buildings and pastures with hot and cold disinfectants and disinfectant solutions; b) cleaning livestock buildings, washing their floors; c) bathing animals with water or weak disinfectants; g) hermetically closing the premises, disinfection and disinsection with appropriate drug aerosols. The VDM device is installed on a UAZ-69 car, a heating boiler for 320 l of liquid, 2 metal containers for 40 liters of mother liquid and 40 liters of aerosol liquid, a special pump and equipment for heating the car engine air and 2 consists of a drum to which a rubber hose is connected.

In addition, electric and gasoline spray pumps can be used for disinfection.

Control questions

1. Disinfection types and objects.
2. Disinfection methods and tools.
3. Chemical disinfection method.
4. Physical disinfection method.
5. Checking the quality of disinfection.
6. Mechanization of disinfection works.

SUBJECT: ANTHRAL DISEASE

Lecture	**Anthrax disease**
Lecture teaching technology	
Study time: 2 hours	of students: 90 people
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Illness, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: Anthrax, acute, severe, infectious disease, farm animals, wild animals, disease, septicemia and pizootic, microbe, pathogen, antibody, immune factors, L. Pasteur, vaccine, epizootic outbreak, specific prevention , epizootic, potential foci, Bacillus anthracis, Oxygen medium, spore form, vegetative, pearlescent, meat-peptone gelatin medium, alimentary and transmissible.

Main textbooks and training used
list of manuals

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used
list of applications

A social literature

1. Salimov X.S, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov X.S, Kambarov A.A "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www. fvat@.academy.uzsci.net

Anthrax is an acute, severe infectious disease that occurs in farm animals and wild animals. The disease occurs in septicemia and is also observed in humans.

In Uzbekistan, the epizootology of the disease, its geography, the state of epizootic foci and specific prevention problems are studied in detail by BS Sayidkulov, G. Mengliyev.

Economic damage . If the epizootic situation is taken into account in time and the animals are not vaccinated, the disease will spread quickly and widely. If sick

animals are not immediately identified and treated, they will die. Quarantine is declared on the farm where the disease is registered, which in turn requires spending a lot of additional funds. As a result, the economy will suffer huge economic losses. People get sick too.

Spread. Anthrax occurs in many countries . Including the Republic of Uzbekistan a q an ch a regions are also potential hotbeds.

If errors and omissions are made in taking special measures, the disease will certainly reappear. There are anthrax furnaces in several districts of Namangan, Kashkadarya, Tashkent, Bukhara and Samarkand regions. In particular, they are found every year in Bostonliq, Ohangaron, Urgut, Jomboy, Gijduvan, Kitab, Namangan districts. Failure to use special vaccines against the disease in accordance with the epizootic situation will lead to its re-emergence in the old foci of the disease. Such a situation was repeatedly noted in Namangan district of Namangan region.

Trigger. The causative agent of the disease, Bacillus anthracis, appears as a stationary rod, single or double, and long-short filaments. It forms a capsule in the body of a dead animal and in a protein environment. Spores are formed when the temperature is 12-42 ^{0} C in an oxygen environment. Some strains are always found in the spore state even in artificial environments. Some are rarely in spore form. In addition to these, asporogenous sporiasis is also found.

Endurance. The anthrax pathogen in the vegetative state is not very resistant to various effects of the external environment. Spores can survive in the environment for several decades. In the carcass of an unopened animal, the causative agent of anthrax dies in 1-2 days. It also dies quickly in gastric juice, but in the spore state it can live comfortably in this environment. Capsule and vegetative pathogens die quickly when boiled, and spores die in 10-20 minutes. When meat and skin are salted, the spores are preserved both in curing and curing .

durability of the spore depends on the conditions of its occurrence . It has been proven that the spore formed at 18-20°C is more resistant to the external environment, and less resistant at 35-38 °C . Among the substances used for disinfection , 3 % creolin solution for 48 hours, 10-20% chlorine lime, 10% sodium alkali, 4% formaldehyde, etc. have a good effect on bacteria and spores .

Cultivation. Bacilla grows very well in an artificial environment. Good growth is observed in MP A at 35-37 ^{0} C, and in MPB at 32-33 ^{0} C. It looks like cotton in the sediment when it grows on the b u lo n in a test tube. S. of the causative agent of anthrax and M forms are present, which are mainly evident when horse serum is used for artificial medium. For diagnosis , it is very useful to grow MP A in an environment that has been enriched with pe nisi lin q . In this case, the microbe is rounded and has a spherical shape, and the phenomenon of "pearl bunch " appears . Meat- pep tone grows in a gelatinous environment in a state reminiscent of an upside - down tree .

Epizootology. Anthrax infects sheep, cattle, deer, wild animals, and people. It is rare in pigs. In some cases, the susceptibility varies between species and breeds. For example, Algerian sheep are more resistant to disease than European sheep. Sometimes lambs and calves are more resistant to the disease than older cattle.

Disease-causing source is sick animals. They, in turn, spread the causative agent to the external environment and affect the ecology. People can catch the disease quickly during tanning, working in tanneries, wool receiving points, skin growing, butchers butchering animals, when products from sick animals are consumed undercooked, etc. The pathogen is released into the environment through saliva, urine, feces and milk. In this way, the external environment (soil, grass, water, etc.) becomes a factor that spreads the pathogen.

The spread of the disease can also be caused by burrowing rodents, water and wind erosion, floods, floods and other natural disasters.

Pathogenesis. The mechanism of development of the disease is as follows: when the bacillus enters the tissues of the body through the injured skin and mucous membrane, it releases aggresin and exotoxin substances, affecting the local defense ability. It then goes to the lymph nodes and blood. Septicemia occurs in the body. The acid-base balance changes and the blood becomes non-coagulable. If the animal is dehydrated, it will die quickly. If the germ gets through the skin, the disease takes place in the form of carbuncle.

Course and clinical signs . Anthrax disease in sheep can be manifested by subtle and visible signs. Depending on the location of the pathological process, skin, intestine, lung and carbuncle forms are distinguished. latent period of illness lasts for 1-3 days and passes in severe, acute and semi-acute state. In most cases , anthrax in goats is a severe disease . _ They gnash their teeth , walk around , do all sorts of strange things . One of the patients with the disease dies a few minutes after the symptoms appear . _ _ If the disease is prolonged , it becomes difficult to work and breathe. Mucous membranes penetrate into the skin . When sick sheep and goats are in the pasture, they stay behind the father and breathe hard. When sick sheep and goats walk around , their body temperature rises to 40.5-42.5"C , and their pulse beats 80-100 times per minute . They shiver , rub their bodies , and The sick animal is in great pain , has diarrhea , bloody diarrhea, blood in the urine, and may vomit. The sick animal it is difficult to breathe , he dies of asphyxiation in 2-3 days .

Semi - acute course . The above signs are also observed during the semi- acute period, but they are not very characteristic and are a little longer. It is also possible that the situation will ease for a certain period of time. But then it relapsed and the patient died . In arid zones, the disease sometimes occurs in intestinal form . Goats suddenly start to jump , then stop suddenly , open their mouths , breathe , and walk around. Symptoms of hemorrhagic pneumonia in the form of lungs on the day of illness develop quickly . In case of carbuncle , in the evening , carbuncles appear on the skin .

Cattle anthrax. The disease is also severe in cattle, acute semi- hepatic and acute . In severe cases , animals die without any clinical signs of the disease. If the disease lasts for a while , the animal becomes restless and afraid of everything , the body temperature is 40-42°C, the blood vessels begin to fast, anxiety increases . He dies with his head on his hip. A blood-mixed liquid comes from the natural pores. A sick animal sometimes dies suddenly , sometimes in a few hours.

A sharp passage. The body temperature rises to 41-42°C, and there is a chill . Heartburn, indigestion , loss of appetite. The cows did not give milk . If there is a sore throat, the child will throw up. Q i moves and t ' stops . The stomach rests, it becomes stiff, and then the diarrhea starts. It dies after 2-3 days.

Semi-acute course. The above signs are visible , but not clearly visible . The disease can last 8-10 days . Acute and chronic episodes are rare.

Swine anthrax. In this case , the body temperature rises to 40-41°C and rises in 1-3 days , then stops . The jaw , throat, and ears are very swollen. Death occurs as a result of suffocation due to swelling and constriction of the throat . Along with angina, lymph nodes are also injured . A sick pig can't sneeze and breathe, it becomes very difficult to swallow food, and it stops passing. In this case, clinical signs may not appear. The pig looks healthy and is often diagnosed at slaughter. Diarrhea may occur when passed in the form of a bowel. Se pt dies quickly in case of delay. This condition is very rare.

Pathologist o anatomical changes. If he dies, he will die very swollen . Fluid mixed with blood flows from natural pores . In summer , it is especially difficult to die , it swells up, it does not harden, blood does not coagulate, and it remains dark . The blood vessels in the subcutaneous tissues are filled with blood, the muscles resemble a red brick color and become pale. As a result , a large amount of serous-hemorrhagic fluid flows in the abdominal cavity . Spleen is enlarged several times , it is full of blood, the papules are loosened, and when cut, black or coffee-colored thickened liquid flows .

can be slightly enlarged . The heart is filled with blood , becomes dark in color, undergoes severe hyperemia. A pointless blood clot is visible , hyperemia is observed in the intestines, and there is a mixed mass of blood inside . Blood flows to the small intestine and duodenum . Colon is less injured . Ulcers and necrotic changes may occur in Peyer's nodes . As recognized in our legislation, if the diagnosis is not clear, it is not possible to open the carcass of an animal that died from anthrax . If it is cut , the place must be disinfected according to the requirements (the ground is dug up to a depth of 40 cm and mixed with a 20% solution of chlorinated lime in the ratio of 1:3) . All waste is incinerated. The surroundings are disinfected.

Diagnosis. Diagnosis of anthrax is based on the epizootology of anthrax , clinical signs , pathologoanatomical changes , and the results of laboratory tests . The dead animal is tight in two places It is cut , cut, and sent to the laboratory for examination . From the pigs, a piece of the injured lymph node or swollen part of the neck is removed. In case of anthrax , the sample sent to the laboratory is placed in a special container with a tightly closed mouth, allowing it to pass the test . Upon arrival at the laboratory , the sample is examined under a microscope , culture is separated and identification is made (determined). A smear (smear) from the blood or other samples of that dog is prepared . After it is dried and fixed , it is stained with Gram 's method , and for capsules, with Rebi 's and Mixin's methods , and an anthrax cell is found . For bacteriological examination , the sample is grown on MP A , MPB media . After growth, it is examined under a microscope , cultured and identified . For this purpose , "K" VIEV VKI "Gram-MVA", "Bacteriophage " and phages give a

good result, which accelerates the killing . Skins are examined by RP or Askol reaction .

Differential diagnosis . It is necessary to be able to distinguish between blackworm, pasteurellosis and parasitic diseases of the blood. Cattle are mainly infected with emkar from 3 months to 4 years old. In case of anthrax, animals of all ages and all types are affected, and the result of bacteriological examination is taken into account. In the case of blackheads, there is a well-defined swelling in the fleshy parts of the body. In pyeterellosis, there is an inflamed tumor in the subcutaneous tissue, which is injured, and the causative agent is Pasteurella. In cases of parasitic diseases of the blood , the parasite is observed on blood smear .

Treatment. Sick animals are immediately transferred to an isolator and treated . Hyperthyroidism is used to treat anthrax . _ _ _ _ _ _ It is injected under the skin for prophylaxis and treatment. 15-20 ml for prophylaxis in horses , cattle, camels , 100-200 ml for treatment purposes, 15-20 ml and 100-200 ml respectively in cattle , sheep - 8-10 ml and 50-100 ml are used in goats and pigs . It is also sent into a vein. Passive immunity lasts 14-15 days . Hyperimmune blood cells give better results if combined with antibiotics (pe n icillin, biomi s in , streptomi n , ekmo n o vo silli n) . Serum 3 7^{0} is heated up to

In order not to physically torture the animal, it is first tested by injecting 0.5-1 ml of serum. Penicillin is administered 3 times at a dose of 500.0 thousand TB per 100 kg of weight, it has been proven that intravenous administration of 1 g of terromycin in a 10% solution for three days gives a good result. Streptomycin and tetracycline are injected intramuscularly 4 times a day. When the disease occurs in the form of a carbuncle or a throat tumor, the injection of a 3-5% solution of carbolic acid around the pathological process gives a good result. Globulin is also recommended for treatment.

Immunity. 1. There is a liquid vaccine made from 55 strains of anthrax. It is 20-25 million in 1 ml. vaccinad i r with live spores . The vaccine is administered subcutaneously for prophylactic and mandatory vaccination

Immunity appears after 10 days. Only veterinarians and raccoons are required to vaccinate against anthrax .

Getting ahead . Comprehensive measures against cancer - measures will be done .

- All animals susceptible to the disease are vaccinated according to the schedule.
- If the disease seems to be out, special measures are taken.
- All animals slaughtered for meat are subjected to veterinary inspection , meat and internal organs are checked . k ' will be cut.
- Potential epizootic outbreaks are mapped , It is necessary to obtain a ruikha tg a. It is difficult to take action and it is necessary to be able to distinguish between kha and fli zones . Vaccinate with a veterinarian _ _ _ sanitary measures are implemented.
- Around the place where they used to live it is necessary to turn it around , put special signs and keep them under control. Mall t o' planned places,

market and The places where the exhibition is organized are under control . It is strictly forbidden to slaughter cattle without medical supervision . In dangerous zones , goods are being taken out to pasture _ _ _ _ _ _ and e mlan a d i while returning .

Goods imported from abroad are kept in preventive quarantine for 30 days, and then they are completely vaccinated . The act is registered , the vaccination document is issued and the 14 days are taken under control. Vaccinated cattle are allowed to be slaughtered after 14 days e wished Quarantine is announced in the farm if the disease is likely to break out .

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.

6. Immunity .
7. Treatment and prevention.

SUBJECT: PROTEIN DISEASE
Aphthae epizooticae

Lecture	**Protein disease**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis.	*Results of educational activities:* Students: Pathogenicity and resistance. Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity.

Immunity. Treatment and prevention..	Treatment and prevention.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes

	described using visual materials.	down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.net

Protein - (Yashur - Russian, Epizootic aphthae - English) is an acute, rapidly spreading infectious viral disease of ungulates (cattle, buffalo, sheep, goat, pig, camel). and wild (deer and reindeer group, wild boars, arkar, white-tailed animals) get sick and cause great economic damage to the national economy. Although this disease is very rare, people, especially people who are in contact with a sick animal : veterinarians and paramedics, milkmaids , dairy farmers, and workers working with the disease virus may be infected, according to ASKorotich, AAVasilchenko, AISobko et al., (1974) ATKravchenko et al., (1966) and others. However, humans are rarely infected with this disease.On average, 1-1.5 million people in contact with infected animals are infected with 1 person out of 200,000.

If we look at the historical data, D. Fracastoro gave information about this animal disease in Italy in 1546. In 1898, scientists Leffler and Frosh first discovered protein disease caused by a filterable virus. Valle and Carre about the fact that there are several types of viruses He gave information in 1922.

Tuberculosis differs sharply from other infectious diseases in its rate of spread . _ _ _ q _ First of all , this disease can spread very quickly and cover not only one or two countries , but also 1-2 regions . In recent years (2000-2001), this disease has spread to Great Britain , France , Germany , Uruguay, Argentina , Mauritania , Turkey , Iran , Afghanistan , Korea , It was recorded in countries belonging to several continents , such as Kazakhstan , Kyrgyzstan , and Tajikistan . _ _ _ There are 7 types of viruses that cause this disease : A , O, S, Sat-1, Sat-2, Sat-3, Os - 1 and 80 . q in options are available. The disease is caused by a type or variant of the causative virus An animal can get sick in different ways and variants . In 1996-1997, a severe tuberculosis epidemic occurred in Iran , and later in 1998 , in Armenia . The disease-causing virus belongs to the " A " type , but its immunobiological It differs from variants A_1 -A 32 $_{with}$ the properties of x Because of this , he was named "Armanist on 98 " . Thirdly , the infection of different types of cloven- hoofed animals with this virus , especially wild animals that are difficult for experts to carry out control measures against this disease, is caused by the causative agent in nature . ensures safety. And finally , fourthly , the disease- causing virus is resistant to external environmental conditions compared to other viruses . Therefore, it is very difficult to effectively fight against this disease , and for this reason, not only veterinary workers,

but all authorities , businesses and organizations are required to prevent the disease from spreading to us . Bars, public gatherings , property owners need to actively participate .

But the fact that our country is located in a very dangerous area, that is, the presence of this disease more often in the foreign countries bordering us (Kazakhstan, Kyrgyzstan, Iran, Afghanistan, Tajikistan), always trades with those countries. Due to the high level of contact with sales and other ways, the risk of protein disease entering and spreading in our area remains.

The causative agent of protein disease. The causative virus belongs to the family of picornaviridae, the genus rhinovirus, and the size of the virion is 20-25 nm. There are 7 types and about 80 variants of the virus according to its antigenic characteristics, its types A, O and C are found in all regions of the world, and types SAT-1, SAT-2 and SAT-3 are mainly in the African continent and Middle Eastern countries. , and Asia-1 type is found mainly in Asia, Middle and Middle East and European countries.

In our regions, in the past, the A, O and Asian types of the virus caused disease. Therefore, in Uzbekistan, vaccines prepared from some variants of Aso san virus types A, O and Asia-1 are used for vaccination.

Each type of the virus creates a specific immunity, so each type and variant differs from each other in its immunobiological characteristics.

The virus is resistant to ether and chloroform, 1 percent phenol, 75 percent ethyl alcohol cannot inactivate it. In summer, it loses its viru0C properties on the surface of hay for 11 days at 20°C, 21 hours at 37°C, 7 hours at 43°C, 70 days in winter months, 37 days in autumn months. The protein virus is preserved in the skin of a salted animal for 50 days at 15°C, 342 days at 4°C, 40-50 days in manure, and 5-6 months in winter. The virus is very sensitive to the RN of the environment , and if its index is below 6 , it is immediately inactivated. It keeps its activity in cool areas of mountain pastures until the next grazing seasons , in non - flowing water up to 103 days in cold seasons, 21 days in summer , and 49 days in autumn . The virus can remain active on the skin of an animal for up to 50 days, on clothes for 100 days, and indoors for 70 days . Tuberculosis virus in milk immediately loses its activity at 65°C, 30 minutes at 70 ° C, and 15 minutes at 80-100 ° C . In meat , the virus quickly loses its activity under the influence of lactic acid , but in salted and smoked meat , the virus can be stored for up to 50 days . The virus is inactivated in a solution of 2 percent formalin and 1-2 percent caustic soda for 10-30 minutes .

Epizootic information about the disease. Cattle and pigs are the most prone to protein disease. Sheep and goats and wild ungulates are somewhat less susceptible. The age of animals also affects its susceptibility. young animals, especially calves, lambs and piglets, are very susceptible to the disease, in which the disease is severe and often ends in death.

The source of the protein pathogen is animals that are ill, recovered from illness, and are undergoing a latent period of illness. Infected and recovered animals release large amounts of the virus into the environment through their saliva, milk, urine, feces, and respiratory tract.

The occurrence and development of the disease. The virus first breathes in, and after 18 hours after entering the body through the mucous membranes of the digestive organs, it begins to multiply in the mucous membranes of the throat and larynx, in the sublingual and head lymph nodes, and in the tonsils . From the primary location, it passes through the lymph into the blood, then it multiplies under optimal conditions in lymphoid and myeloid tissues and forms disease foci. During this period, only the body temperature of animals increases by 1-2°C. then soon secondary blisters appear in hairless areas of the skin (nostrils, nostrils, udder, sometimes under the horn), between the hooves, on the mucous membranes of the mouth. These events are observed mainly in 2-3 days. Since the virus is myotrope (myo-meat, trop - conditions for good development), it settles in the heart and body tissues and causes various dystrophic and degenerative changes there.

Course of the disease and clinical signs. Manifestation of clinical symptoms of the disease depends on the individual sensitivity of the animal to the virus, its physiological state and the degree of virulence (pathogenicity) of the virus. Symptoms of the disease are well manifested in young cattle, and in calves, lambs and piglets, the course is often atypical. In young calves, the disease is fatal, often ending in death.

The "latent period" of the disease is from 36 hours to 7 days, sometimes up to 21 days. In general, the disease is very acute.

Since the clinical signs of the disease are different in different animals, we will focus on its specific course in different types of animals. The first clinical sign in cattle is death and an increase in body temperature of 41 C^0 and above. The sick animal becomes weak and loses its appetite, it does not eat and does not chew, the pulse accelerates, on the 2nd day of the disease, the mucous membranes in the oral cavity are reddened, and the outer and inner lips, lung, toothless part of the lower jaw, and the tongue are abundant. A number of blisters appear.

If treatment measures are not carried out in time, if they are fed poorly and living conditions are poor, the complications of the disease will be strong in animals . In such conditions, ulcers in the oral cavity fester and neurotic processes intensify. The hooves on the legs fall off, the purulent wounds expand and the tendons and joints become infected. Ulcers in the udder deepen and suppurate under the influence of secondary infections, and the cows become unfit for milking. In some cattle, especially cattle that have not been vaccinated against the protein at all, the disease is very severe. In them, heart failure is observed, they become weak, the activity of the circulatory system also goes out of control, and the molluscs suddenly die in 7-14 days of the disease. Mortality can be 70–100 percent. This condition is more common in young calves. In them, protein disease is manifested by high temperature and gastroenteritis, and bleeding in the heart in the case of endocarditis. Sometimes sick calves can have a complication of bronchopneumonia. The disease lasts 8-10 days when it is mild, and up to 25 days when it is fatal. Usually sick calves die from heart paralysis due to myocarditis within 12-30 hours . In them, the disease passes without blisters. Tuberculosis is a disease of old age Up to 40% of animals can die if it passes in a fatal form in the forests .

In humans, the disease is very mild, and lesions are observed on their hands and mouth. It can be found mainly in veterinary specialists dealing with protein disease of animals, cattlemen and milkmen.

The latent period of protein disease in guinea pigs lasts from 12 hours to 4 days. A large number of aphthae appear on the mucous membranes of the mouth and feet, remain from food and movement. Rabbits and white mice are also affected by protein disease.

Pathologoanatomical changes. The most characteristic of pathological anatomical changes is the observation of aphthae of various sizes on the mucous membranes of the oral cavity, tongue, tongue, and palate. Similar changes occur in the udder and hooves of cattle. At this point, it should be noted that if the protein disease is severe, if the diseased animal is kept in dirty buildings instead of in good and clean places, various secondary micro-organisms enter the wounds in the mouth and legs, and in some cases, they develop purulent inflammation. may turn into phlegmon, cattle's hooves may fall off. Ulcers at the site of aphthae in the udder are complicated by secondary microbial action and turn into purulent mastitis. There are also changes in the lymph nodes, where the lymph draining from the above-mentioned organs collects. They are usually large, mushy and hearty or freshly roasted. Sometimes , aphthae and erosions are observed on the mucous membranes of the large abdomen and flat abdomen.

Diagnosis of the disease. Diagnosis of protein disease is based on clinical signs, epizootological data, pathomorphological and laboratory examination results.

The effectiveness of laboratory testing depends on many factors, the main of which is the specificity of the method used and the level of sensitivity of antigens and antibodies in immunochemical reactions. It depends on the activity of immunodiagnostics used in the reaction. With the help of serological reactions, the clinical-epizootological and pathanatomical diagnosis of the disease is confirmed, the type or variant of the virus, its epizootic significance, the genetic affinity of the epizootic strain to the vaccine virus strain used, and depending on the results of the examination, healthy susceptible animals in the dangerous area are determined. vaccinated with a vaccine prepared from the relevant virus types and variants.

Differentiate protein disease from other similar diseases . _ _ _ _ Even if the diagnosis is made on the basis of clinical and epizootological data, it is necessary to carry out serological and virological tests. Vesicular stomatitis also affects the oral cavity and hooves. In order to distinguish between these diseases, it is necessary to infect horses or donkeys with the pathological material of the diseased animal. Adult white mice weighing 16-20 grams are susceptible to vesicular stomatitis, and young, non-lactating mice are susceptible to protein.

Simple vesicular stomatitis caused by eating spoiled coarse grass, firstly, it is not contagious, and secondly, the leg does not get infected and the body temperature does not rise. Vesicular disease of pigs does not affect other species of animals. Cattle pox only occurs in the udder. Aphtha and erosions between the hooves are not observed in patients with viral diarrhea, infectious rhinotracheitis, plague.

Treatment. Sick animals are isolated in a special room, and cattle are fed with quality fodder, kept in clean, dry, well-ventilated barns. Wounds in the mouth, hooves and teats of cattle are frequently washed with 1-1.5% copper sulfate, 1:250 potassium permanganate and 1% trypoflavin solutions, and antibiotic emulsion is applied.

tissue hydrolysates , which increase the body's resistance, are sent to treat sick animals with special methods .

Immunoglobulin preparations, blood serum of animals vaccinated against protein and recovered from protein disease , immunolactone q are used for special treatment of sick animals .

Measures to prevent and combat protein disease In our country, the fight against protein disease is carried out with the help of a comprehensive system of measures. First of all, it should be noted that all the regions of our Republic border with foreign countries: Kazakhstan, Kyrgyzstan, Tajikistan, Turkmenistan, Afghanistan, and so on. Each region is divided into 2 areas: general and dangerous (buffer to the border 30 km distance) divided into regions. Because the fight against protein disease carried out in dangerous border areas is more serious than in general areas.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention .

TOPIC: TUBERCULOSIS DISEASE
Tuberculosis .

Lecture	**Tuberculosis disease**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov X.S, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov X.S, Kambarov A.A "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.net

Tuberculosis is a chronic infectious disease that occurs in farm animals, wild animals, fur animals and poultry. Special nodules - tubercles appear in the internal organs and tissues of infected animals. Tuberculosis is a common disease among people.

Economic damage. Farms affected by tuberculosis suffer a lot, because infected cattle are removed from the farm within 15 days and sent for meat, which in turn leads to a decrease in the number of hooves and a decrease in production. Quarantine is established in the farm where the disease is registered. It is necessary to spend additional funds to carry out its activities. As a result of the decrease in the standard level of livestock products, the farm receives a reduced amount from meat and milk combines. Each infected cow causes an average loss of 18-20 thousand soums to the farm. In addition, people are also infected with tuberculosis, and the diseased animal pollutes the environment with tuberculosis bacilli.

Trigger. Tuberculosis is caused by a bacterium called Musobacterium, discovered by Robert Koch in 1882. Mycobacterium is thin, straight, sometimes with a bent tip, length is 0.8 to 3-5 μm, width is 0.2 to 0.5 μm. The bacterium often changes its shape, that is, it has the property of polymorphism. When the microorganism is grown in an artificial environment for a long time, it also appears in a circular form. Does not form spores and capsules. The tuberculosis microorganism differs from other microbes in that it is resistant to alkali, acid and alcohol, antimorphine. This microbe reproduces by simple division.

According to the current classification, there are three groups of this type of microorganisms.

1. Pathogenic mycobacteria (tuberculosis, tubercle , and moss) .
2. Atypical mycobacteria (photochromogenic , scotochromogenic, petrochromogenic and fast - growing) .
3. Acid-resistant saprophytes.

Tuberculosis grows well in an artificial environment with glycerin added . For the cultivation of tuberculosis microorganism in laboratory conditions , artificial mediums such as Petrinya , Lübenau , Leuvenshte , Dubo water medium are recommended .

Microorganism S il - Nielsen It is carefully thought out and seen under a microscope . Its essence consists of the following :

1. The smear (smear) prepared from the affected part of the body is dried in the air.
2. is fixed in methyl alcohol for 5 minutes do n adi .
3. Lubrication with phenol, Sil fuchsin dye oil 1-3 during the minute b thought .
4. The affected face is washed with water .
5. Ointment is 5% sulfuric acid solution or 15 % nitrate solution with the solution, it is discolored within 2-5 seconds .
6. is washed with water .
7. Methyl k is added for 0.5-1 minutes .
8. After washing with water, pat dry with a towel .

Painted smear in the immersion system of the microscope k ' rilad i . Tuberculosis bacilli appear red, other microorganisms appear bluish, and the base appears cloudy.

There are three types of microorganisms that cause tuberculosis:

1. The human microorganism is a type of tuberculosis (M. Humonus) causes tuberculosis in humans .
2. (M. Vovinus) causes tuberculosis disease in cattle .
3. Poultry microorganism (M . avium) in p arra n fields tuberculosis causes illness .

Endurance. Tuberculosis causative agents belong to group 1 bacteria that are resistant to environmental conditions and can maintain their disease-causing properties for a long time. For example, it can live up to 10 months in running water, and up to 12 months in swampy water sources.

Atypical mycobacteria. Mycobacteria belonging to this type are divided into the following groups according to the Rinon scheme.

1. Photochromogens. These grow quickly, giving off a yellow color. Resistant to antibiotics. Dsngiz does not cause disease in pigs and rabbits. The lungs and kidneys of mice are damaged.
2. Spirochromogens, 22 ^{0} It grows yellow-red in C. Does not grow at 45° C . It is resistant to antibiotics and causes disease in laboratory animals .
3. Nephtokhr o moge nl ar. Grows in normal environment , only causes disease in mice , lymphadenitis in pigs.
4. Rapid growth v th group, 22 ^{0} C It grows at 3 k u n . Color there are those who grow by giving . This group of microbes is for land found in the peat used (not in other environments).
5. Ki slo ta g a species belonging to the resistant group. They grow at room temperature and are resistant to laboratory animals .

When it gets into the animal's body, it causes an allergic reaction .

Epizootology. From farm animals, tuberculosis is more common in cattle, pigs, goats and poultry, less common in goats, dogs and geese, and very rare in sheep, goats and cats. Diseased cattle are a source of disease. In tuberculosis, as in other diseases, three main factors play a major role: the source of the disease, the mechanism of transmission of the disease to a healthy animal, and susceptibility to the disease. Disruption of this triangular chain link prevents the origin of the epizootic process. In addition to these primary factors, there are also secondary factors such as natural climate and socio-economic conditions that affect the origin of the disease.

Exciting sources. Animals infected with tuberculosis in cattle are the source of the disease. It should be noted that sick pigs, sheep, goats, horses, dogs and cats sometimes act as a source of disease for the above-mentioned species. Tuberculosis bacillus grows stronger in the body of a sick animal and spreads to the external environment through urine, manure, milk, semen. The dangerous part of the disease is that, before its clinical symptoms are manifested, the tuberculosis bacillus begins to be released into the environment during its latent period, since it is chronic and long-lasting. When sick animals cough, a large amount of mycobacteria fall into the external environment with mucus and liquid, contaminating walls, mangers, fences and various objects.

SA Traubayev and V. Ye. According to Shurevskii, 1 ml of sputum contains 50,000 mycobacteria, and in 66% of sick animals, mycobacteria are released into the environment through manure. According to NN Daronin and SI Muratov, when the disease was artificially induced, the release of the tuberculosis bacillus through manure was observed after 38 days.

Pathogenesis. Tuberculosis bacilli enter the body through food or air and spread to the lungs and other organs. The place where the tubercle bacillus landed becomes inflamed, and the process of tissue proliferation and exudation continues. As a result, multinucleated giant and epithelioid cells are collected. Accumulated fibrins

between the tissues become clotted and avascular tubercular nodes appear. Then the knots are wrapped with a capsule.

The nodules that fall into the capsule die due to the lack of nutrients and the toxins released by the microorganism. It is a dry mass, reminiscent of a more yellowish color. If the first tuberculosis nodules are found in the lungs and intestines, this is called the first effect. From this place, the microorganism spreads through the lymph to the lymph nodes and can infect the entire regional lymphatic system . Mycobacteria get into the blood and cause a short-term mycobacterial condition . Often cases of transition in a gradual or gradually hyperalized form are observed . Infected animals develop large tubercular nodules in their lungs . _ _ _ When the mucous membranes and other parts of the internal organs are damaged , coral cysts of many sizes appear . Severe (generalized) damage to the lungs and other organs leads to rapid growth of animals and a sharp decrease in productivity . As a result, the sick animal will soon lose weight and die.

In recent years, tuberculosis is often observed in our conditions.

Klinik characters. The latent period of tuberculosis lasts 2-6 weeks . In the origin of the disease and its course , cattle feeding , storage conditions and the virulence of the tubercle bacillus are of great importance. After infection , it may take months , sometimes years , for clinical symptoms to appear . Tuberculosis is mostly chronic. Therefore , it is very difficult to diagnose it based on its clinical signs . In most cases, the temperature does not change. Sick animal asta - seki n begins to lose weight, and a decrease in appetite is not felt. The elastic state of the skin remains .

Patologoanatomical change shl ar. The appearance of special nodes and tubercles (tuberculosis) in various organs and tissues is one of the characteristic signs of the disease . These knots are the size of a millet grain to a hen's egg and much smaller . Tuberculosis and nodules resemble a dry fibrous mass inside . Tuberculosis - related changes in cattle are most evident in the lungs and lymph nodes . The lymph nodes become enlarged, hard, and a small mass is thrown in the center . When mucous membranes are damaged (pearl- like), a large number of hard shiny nodules similar to forest fire appear on its surface . According to the information provided by P. I. Kakuricheva (1950), in infected cattle, lymph nodes in the chest are 100%, lungs are 90%, spleen is 5% , spleen is 3%, intestine is 1% . z will be sawn. RV Tuzo and a (1974) determined that 88% of the lymph nodes around the intestine were infected with tuberculosis in pigs during the year, and 5-36 % of other lymph nodes were affected . Even in the summer, there are changes in the lymph nodes . Tuberculosis changes in poultry occur in 70 % of the liver, 90% in the spleen, bones and intestines .

Diagnosis. In order to make a diagnosis of tuberculosis, its epizootology, clinical signs and course, pathologo-anatomical changes are studied, and surgical, serological and laboratory testing methods are used. Tuberculosis in the animal is detected mainly by the allergic test method. Allergens in sheep are used to detect tuberculosis:

1. For mammals al tt uberk u lin.
2. Dry cleaned tuberc u li n (PP D), for su t emi z flourers.

3. Dry cleaned tuberc u li n (PPD), p arrandas three n .
4. A complex allergen (KAM) prepared from an atypical mycobacterium is used in simultaneous allergy testing together with a special type of allergen. In the box containing the drug there is also its instruction (nastavleniye).

Veterinary sanitary measures. In farms where the disease has been recorded , it will be announced . Sale and purchase of goods is prohibited. It is illegal to exchange or mix a group of goods . Diseased cattle are immediately separated and delivered to meat within 15 days, they are marked with a "T" sign. Sick animals n i one if it is not possible to transfer , a temporary isolator can be organized. It is necessary that the insulator and the terinary - fully meet the requirements of the sa n itary. It is impossible to breed and escape from sick animals.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention.

SUBJECT: BRUTCELLOSIS DISEASE
Brucellosis

Lecture	**Brucellosis disease**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.

Treatment and prevention.	
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis,

anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov X.S, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov X.S, Kambarov A.A "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.net

Brucellosis is a chronic infectious disease that is characterized by miscarriage, endometritis, placental abruption, orchitis, and fever in recurrent cases.

Economic damage . Brucellosis is very damaging. Sick cattle are not treated. Calves throw a child. When the disease is registered, quarantine is announced (requires spending a lot of money). Diseased animals are immediately sent to the warehouse. You have to spend a lot of money for diagnostic testing and prevention. The most dangerous thing is that the disease has medical and sanitary importance and is contagious to people.

Q exciter. The causative agent of brucellosis is a microorganism belonging to the Vrusella group. Disease in cattle Vr.abortus; in sheep and goats Vr. melitensis in pigs Br. suis; Vr in rats. neotomae in epididymitis of rams Vr. ovis; q changes . Very dangerous for people.

There are three biotypes of the causative agent in sheep and goats, nine biotypes in cattle, and one biotype of fleas in pigs, including deer. Some biotypes are distinguished by biochemical and antigenic characteristics. The shape does not differ much from each other. It is 0.3-1.5 μm, inactive, does not form spores. Grows in a normal environment (pN 6.6-7.4), develops well at 36-38°C. The microorganism grows very well in agar prepared from liver, meat-liver medium, 10% whey, potato medium.

Endurance . Brucella can live from a few minutes to 2-3 hours under the direct sunlight. It lives in the sunlight for about a week. Sskin can live 37 days in dry land, 100 days in rotten soil, 2-3 months in water, 80 days in salted meat, 42 days in brine. It does not lose its virulence up to 160 days at a negative temperature. It is stored in frozen pathological material for up to 1.5 years. Brucella dies quickly in a boiled environment. Its preservation in the external environment depends on physical, chemical and biological factors. For disinfection, 1% chlorine lime, 10-20% lime solution, 3% lysol, 3-5% carbolic acid, 2% alkali, and 1-2% formalin give good results.

Epizootology. In natural conditions, brucella enters a healthy body through food, water and food, eyes, nose, mouth and genitals. When a cow sheds a baby, it releases a large amount of irritants into the environment. In this case, the causative agent falls from the placenta and fetal shell to the hay, soil, manger, and equipment, and additional factors appear that allow the disease to spread. A large amount of Brucella is released from the genitals of the animal until about 15 days after giving birth (Fig. 9). It is also excreted through urine, feces and milk. It is extremely dangerous for an abandoned child not to be neutralized . The disease is rarely transmitted in pasture conditions. It mainly occurs when cattle are kept in one place.

Pathogenesis. The causative agent of brucellosis enters the animal body through the mucous membrane or skin. Then it dies under the influence of the body's resistance, or if it becomes stronger, it passes to the internal organs and a generalized state appears. During this period, the body temperature increases by 1-2°C. After 3-4 weeks, the pathogen moves to the udder and lymph nodes. Brucella rapidly multiplies and develops in the uterus. In the process of development, the pathogen releases a toxin. As a result, a necrotic inflammatory process occurs. As a result, the connection

between the endometrium and the chorion is broken, the fetus dies and is expelled as a foreign object. Cattle give birth in 5-8 months of gestation. If the disease is contracted later, the calf is born as a cripple and dies within 1-2 weeks. A second miscarriage in Brussels is very rare. In bulls, Brucella causes a pathological process in the genitals, the genitals swell and become inflamed.

Pathologoanatomical changes. In many cases, the head, legs, and body of a child who throws a mole are swollen. A serous-hemorrhagic infiltrate is noticeable under the skin. The umbilical cord is also swollen, thickened , red fluid with fibrin accumulates in the chest and abdomen. In the internal organs, there is spotting of blood, lymph nodes, liver and spleen are enlarged. Small necrotic foci are visible in the liver. When examining cows that have given birth, purulent catarrhal endometritis and necrosis of cotyledons and suppuration are revealed. Lymph nodes around the udder are severely injured, and in some cases, gran u lema nodes appear. In males, purulent necrotic orchitis and epididymitis occur.

Diagnosis . To diagnose brucellosis, the epizootological situation is studied. Depending on the clinical symptoms of the disease, bacteriological, serological and allergic tests are used. And the child of the mole will undergo a pathologo-anatomical examination. The contact of cattle in pasture conditions, the beginning of calving, the lack of water sources, the recording of disease among people should serve as a signal.

accurate diagnosis, the following tests are carried out .

Serological examination. Serological testing is based on the search for antibodies in the blood serum using a specific antigen. For this purpose, RA - agglutination reaction, Roz-Bengal reaction, RSK (complement binding reaction), RDSK, and milk is checked using the milk ring reaction.

Allergic examination . Allergens are used in this. VIEV allergen - brucellin gives good results. 0.5 ml of it is injected into the left eyelid (subcutaneously) in sheep and goats . The injections are injected into the skin on the outer part of the upper eyelid . A positive result is evaluated by the appearance of a hard swelling under the lid, and in pigs by redness and swelling.

Treatment and immunity . Infected cattle are not treated and are sent to meat. Vaccination against the disease is carried out using vaccines in sheep.

Strain 19. Prepared from Brucella obortus species, Dry live vaccine. A sterile physiological solution or distilled water is used to dissolve the vaccine. After dissolving, it should be used within 4-5 hours. The vaccine is used strictly according to the instructions. Vaccinated animals are strictly accounted for and cannot be lost or replaced.

REV 1 is used to vaccinate sheep and goats. It is also recommended for epididymitis in horses . The vaccine is made from a weak virulent strain of Brucella melitensis . It is dissolved in a special solution or sterile physiological solution before use. After 30 minutes, 2 ml is injected under the skin. After 3 weeks, immunity appears.

Strain 82. The vaccine prepared from it is used to vaccinate cattle. It is prepared from weak and glutinogenic obortus.

A vaccination scheme with Strains 19 and 82 was developed at the Scientific Research Institute of Brucellosis and Tuberculosis in the past . It gives good results in production. Small-dose vaccination is now being carried out in our country.

Prevention. Br u cellosis control measures are as follows:

Protecting the farm from brucellosis.

unhealthy x cells healthy.

Proper organization of vaccination work.

Protecting people from disease.

To carry out these tasks, the following tasks must be solved:

of brucellosis , isolation of diseased cattle and transfer to meat.

As the cattle keep getting sick, they are completely replaced by healthy ones.

To prevent the disease, a number of organizational and sanitary-disinfection works are carried out.

Making an unhealthy economy healthy. If brucellosis is detected, the farm is immediately quarantined. A calendar work plan for the health of the farm is drawn up and approved. Vaccination is carried out with or without vaccination with the permission of the Veterinary Department.

In accordance with the requirements of the quarantine, the following are prohibited:

Import and export of goods.

Grouping of animals without the permission of a veterinarian.

Milk extraction, distribution for kindergartens, schools, and selling on the market. It is necessary to pasteurize it at 70°C for 30 minutes in such a dairy farm, and the milk coming out of the dairy farm must be transported in special containers.

Protecting people from brucellosis. In case of outbreak of brucellosis among farm animals, the following measures are taken to protect people from this disease :

All employees working on a farm with unhealthy livestock are under the supervision of a medical institution and must strictly follow the rules of personal hygiene.

Only persons vaccinated against brucellosis are allowed to work on sheep and goat farms. All employees of the farm are provided with special clothes. Hand wash, towel, soap, medicine in each livestock building it is necessary to ensure that there are boxes. Livestock workers must undergo a special medical examination.

Epididymitis disease of rams. Epididymitis is a disease characterized by various degrees of proliferative inflammation in the gland and a decrease in reproductive function . Acute and chronic epididymitis . In the disease, the body temperature rises to 41-42°C, exudative inflammation occurs in the gland . The scrotum becomes tense, hot and reddened . As a result of work, hair becomes stiff and droopy. A large amount of exudate oozes out. It becomes difficult for him to walk and he limps. After some time , the temperature drops, the swelling of the scrotum returns. The testicle atrophies. Sperm secretion decreases, the density changes , and it becomes yellow.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention.

Topic: RABIES
Lyssa, Rabies

Lecture	**Rabies and the fight against it**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.net

Rabies is an acute infectious disease caused by a neurotropic virus. Passes with severe damage to the central nervous system.

Economic damage. Diseased people die and animals are destroyed. A large amount of vaccine is required for immunization. The sad thing is that there is a lot of disease these days.

Trigger . The causative agent of rabies is a neurotropic filterable virus. It is the head in the body of a sick animal There is a large amount of t op la n adi in the brain . Spinal cord, salivary glands and saliva are abundant. At the beginning of the disease, the virus was also found in the blood. In very rare cases, the virus can also be found in the spleen, kidney, mammary glands , pancreas, and eye glands .

Babesh-Negri bodies appear in neurons of the brain, which are found only in animals infected with the rabies virus. The body contains viral protein, RNA. Formed virions are not found. Therefore, some scientists assume that this body is a "grave" of viruses.

Ch i relaxation. The virus is resistant to high temperatures. 5-10 minutes at 60°C, At 70°C, it dies immediately. Low temperature preserves. Frozen brain can be stored for up to 2 years. Lyophilization can last 3-4 years. Ultraviolet rays kill in 5-10 minutes.

Epizootology. All types of wild and domestic animals, as well as humans, can be infected with rabies . Cold-blooded people do not get sick. Among the wild animals , mainly carnivores (tulki , wolves , chiyab -ori) are often infected, while the disease also occurs frequently in rodents and bats . From pets, dogs, especially dogs and cats are very sick . In poultry, the disease can be induced naturally , it is very rare in nature.

Pathogenesis. The virus enters the body, settles in the nerve fibers and travels through the nerve trunk towards the head and spinal cord. It can also go by the lymph-hematogenous route. Salivary saluronylase is of great importance in the transmission of the virus to living tissue. When the virus reaches the brain, it injures nerve tissue, multiplies and develops, passes through the peripheral nerve to the head and spreads through saliva. It affects the nervous tissues and tickles them, then the reflex excitation increases, aggression and nervousness appear. Then degeneration begins in the nerve tissue, leading to paralysis. Respiratory muscles are paralyzed and patients die as a result of asphyxiation.

Course and clinical signs. The latent period depends on the place of the bite, the nature of the wound, the number and virulence of the virus, a short period of 7-10 days, and it can last for 3-8 weeks. Sometimes it can last 5-6-12 months . Le kin is very rare. The disease is often progressive , and the clinical symptoms are different . In dogs , the reaction occurs in typical cases .

Dogs rabies. Mostly in severe and mild (paralytic) form , therefore , abortion _ _ _ v a iti appears in p ic state . The severe form is in three stages (pro dromal , acute) . and paralysis) o' tadi.

In **the prodromal stage** , the condition of the sick animal is different . Extends to 4 days in 12 hours. The dog is silent , pulls itself to a dark place and does not come even if the owner calls. Sometimes he is very busy and likes to go home with his wife . He licks his mouth and hands (at this point there is a virus in the saliva, it is very dangerous). This dog later becomes very restless , it runs from side to side , stutters and is afraid of any noise . _ _ Appetite is lost , and when he eats his usual meals , he scratches the ground with his hunger and starts biting the soil , his own garbage , excrement , wood or iron . It is possible to eat the licks too . Sometimes , due to strong urination , he can bite and pull out that place . As a result of the paralysis of the throat muscles, it becomes difficult to swallow or swallow , salivation , and the patient may vomit . His voice changes and he sings more . _

Click on the button . _ Discomfort turns into aggression . _ _ The symptoms of fear remain . The dog bites the chain link or chain . When he gets his prey , he runs away for a very long distance (several tens of kilometers per day) . It is thrown on people and animals . In this case , they come quietly without barking and bite suddenly . A lot of saliva flows from the mouth because the throat and tongue are paralyzed . Because of this, the sound is hissing. The lower jaw drops. The eyes

squint, one pupil becomes smaller, and the other becomes larger . Disturbance lasts 3-4 days, then the second stage begins.

Fal a jlik stage. The dog loses weight, there is aphonia (the sound remains). Paralysis of the throat, tongue, and jaw is accompanied by paralysis of the back . This condition also spreads to the bladder and hindgut. Then the front legs are paralyzed. The dog is dragging its back. It dies after 8-10 days .

Peaceful (paralytic) form In 10-15% of dogs, anxiety is not always noticeable. It is often difficult to breathe due to paralysis of the lower jaw. Drooling a lot. It reminds me of having a bone stuck in my throat. Paralysis worsens and dies after 2-4 days.

At ip shk kschi sh . Rarely, continuous b ' lad i . By giving a hemorrhagic gas s troe nte rit r o' , the sick dog will lose a lot of weight . Sometimes abortion can happen. Cats have the same clinical symptoms as dogs , and they die within 3-4 days . Dogs are very aggressive towards people .

Cattle rabies . The disease occurs mainly in acute and paralytic cases . _ Hadeb runs restlessly , pulls on the rope , grunts louder , throws at people , starts to swim , his eyes blink, his pupils dilate, he becomes red with a strong bloodshot . , drools a lot, sweats profusely and the bite itches. It is also possible to work in that place . _ Sexual intercourse in some cases it may increase . Digestion decreases and sometimes stops . He tries to defecate, but he does not have a bowel movement. A violent state alternates with a calm state. As in dogs , paralysis is observed . Sick animal dies after 3-6 days . In calves, it passes in a specific state. Symptoms of the disease are not characteristic , gastric atony occurs. The throat becomes paralyzed and the sick calf loses weight. It dies after 4-5 days . If the disease is contracted as a result of contact with meat, there will be no aggressiveness . It just gets annoying and gets worse . The body temperature rises , he sleeps without eating anything and without drinking water , he becomes lethargic and defecates . Then he begins to tremble and falls, the head i or throws it. It moves as if swimming with its limbs and dies after 1-3 days .

Q think _ – goats rabies. The cooling phase does not last very long . A state of aggression is observed. When symptoms of the disease appear, after 2 days paralysis begins, and after 3-6 days the diseased animal dies. It is acute in sheep . In horses, it passes as well as in cattle , and is often severe.

Bullet rabies . Swine rabies is mainly acute and severe. Inside the fence, he hits himself in all directions, makes noise , scatters the bed in all directions, digs the ground, or grinds on wooden objects . Aggressiveness increases . _ _ Rabid pigs are also known to their children . Paralysis begins and dies in 2-3 days .

P arran d alar box beat . Very rare. Very agitated and wheezing q aq agl said. Aggressiveness is observed . Poultry quince n _ v a is also thrown to people. Then depression, eventually paralysis, and death in 2-3 days.

P atologaana to mic change l ar. When he dies , his wealth will be greatly reduced . In many cases, due to the itching of the skin part of the body , the scar becomes visible . The wool in the lower part becomes contaminated with saliva and hardens . Inflammation is observed in respiratory and gastrointestinal diseases . It

will be the same accident . In the stomachs of meat eaters, fat , pieces of fat and other things can be found . Blood clots in the mucous membranes are visible . The membranes of the head and brain are swollen, there is a point -like hemorrhage everywhere , the blood vessels are dilated. goes

Diagnosis. When making a diagnosis of rabies, its epizootology , clinical signs and pathology are taken into account . The head of a dead animal is sent to the laboratory . From the smear obtained from the brain , B -Abesch-Negri corpuscles are found. If this does not work, the bios will be updated. A 10 % brain suspension was administered to mice and rabbits (subdural _ or intracerebral) is sent and observed for a month . Neutralization , RSK, RP, RN reactions are considered . It is the most effective fluorescent method . _ _ _ _ _

Differential diagnosis . _ It is necessary to distinguish between Auyeski's disease. It passes acutely , strong itching occurs , aggressive condition is not observed, lower limb paralysis does not occur. It is different from plague in dogs . Only meat and animals are sick . This disease is chronic, highly contagious, it can be contagious , aggressive condition is not observed. In horses, en se falomi is similar to ye lit. There is no aggressiveness in this, the mucous membranes are really smooth . Some of the diseased animals can also die .

Currently used Shelko vo - 51 vaccine is a good vaccine for immunity . Now there are also vaccines that are given once in a day . All vaccines are administered strictly as directed . _ _ _

Getting ahead . He loves dogs and cats . _ Wild carnivores in the dangerous zone are shot and killed, dogs and cats, black cattle are killed according to a plan . Dogs that have bitten people or animals are kept in isolation for 10 days. If there are no clinical signs during this period , it indicates that there was no rabies virus in the person's saliva on the day of the dog bite . A sick animal killed and burned. For disinfection, 10% alkali, 4% formaldehyde are used, and manure , bedding, food residues are burned. If a sick person is registered, the farm is considered unhealthy . The restriction is lifted after 60 days after the slaughter of sick animals .

Human rabies . The latent period can last from 2 weeks to several months. The stage of the disease passes like animals . The patient looks helpless , convulses and becomes restless. There is pain and itching in the bitten area . Swallowing becomes difficult, the noise increases perceptually . Hydrophobia occurs. Throat muscle spasms and gets stronger at the sight of water . Fear and anxiety are at their peak. The state of Gallus i nasia is manifested. A violent attack begins. Drooling a lot. The face, eyes , tongue and legs become paralyzed and go limp and die within 4-7 days. It is a dog to prevent n day calamity it is necessary to thoroughly wash the bitten area with 1:1000 saline or $KMnO_4$ and immediately contact a medical institution.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .

3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention.

Topic: Leptospirosis disease

Lecture	**Leptospirosis disease**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2021. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.net

Leptospirosis is mainly an acute infectious disease that affects a number of animal species and humans. The disease is manifested by anemia, jaundice, hemoglobinuria, vomiting, hemorrhagic diathesis, necrosis of the skin and mucous membranes, atony in the intestines and stomach.

Economic damage . Leptospirosis causes a lot of damage . Mortality among infected cattle is very high, and many young cattle give birth. Those who are born are stillborn or unable to live and die within 2-3 days . This condition is especially common in children . It is very rare to give a lot . Diagnostic testing, vaccination, treatment, quarantine measures require a lot of money .

Q ' z g' shooters. The disease is caused by leptospira, that is, L. romona, L. Grippotyphosa, L., Tarassovi, L. Hebdomadis, L. batavia, LCanicola . (Figures 10, 11).

To date, about 90 pathogenic leptospira have been isolated from the animal world and humans, and they belong to the 13th serological group. Leptospira are morphologically not very different from each other, they are found in semi-circular, spiral, S, X, 8 forms. This group is characterized by different actions, some of them also have filtering properties. Leptospira are grown in a thermostat at 28-30°C for 5-20 days, mainly using Lyubashenko, Terskikh, Fervort-Wolfa, VGNKI, Korthof, Fletcher media.

2 - picture leptospira darkened objectified appearance.

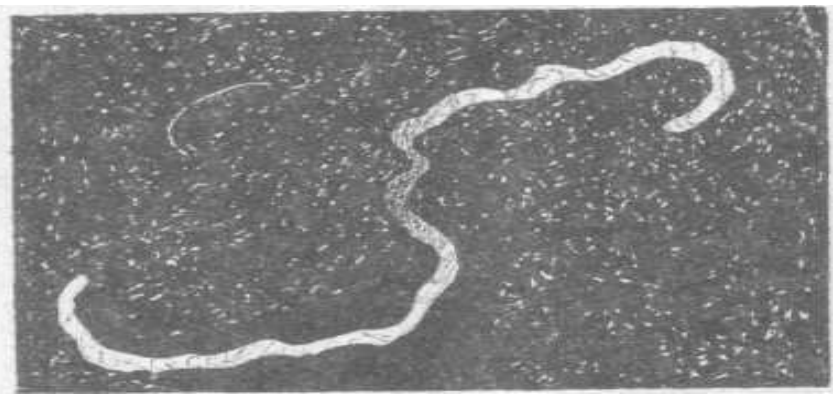


Figure 3, Electronogram of Leptospira.

Endurance. In our conditions, it dies within an hour under direct sunlight, within 30 minutes when heated to 56°C, and within 4 hours at 20°C. Leptospira can live even when pond water freezes in winter.

For disinfection, 2% chlorine lime solution, 2% alkaline solutions, 3% sulfate-carbol mixture, 5% disinfectant phenol solution, 2% formaldehyde are recommended. Disinfection is carried out every time after isolation of sick animals.

Epizootology. In natural conditions, foxes, cattle, sheep, goats and pigs get sick. In some cases, it is observed in dogs, cats, deer, cats, donkeys, chickens and ducks.

Leptospirosis occurs in animals of all ages, but mainly in young animals. Cattle brought from healthy farms immediately get sick in an unhealthy farm, and the disease is severe in them. In localized jaidari cattle, the disease is less common. Young dogs, rabbits, woodpeckers, pigeons and frogs are susceptible to artificial infection. In natural conditions, the source of the disease is mainly rodents. They can spread pathogens for life. According to some data, foxes can spread pathogens for 514 days, cattle for 120 days, sheep and goats for 180 days, yearlings for 210 days, and dogs for 300-700 days.

Pathogenesis . Leptospira naturally enter the body through mucous membranes of the stomach and intestinal tract, conjunctiva and injured skin, and after 12 hours accumulate in the liver in large quantities. It stays in this place until the fever rises. In very rare cases, it can also be found in kidney, heart muscle, and mesenteric lymph nodes before fever. Leptospira accumulate in the blood in large numbers during fever. As soon as jaundice appears, leptospires gradually leave the blood and begin to accumulate in the kidney. In turn, by this period, lysine and

agglutinins appear. In a dead animal, leptospira accumulate in large numbers mainly in the kidneys, liver and other organs.

Course and clinical signs . The latent period of the disease varies in different animals and can last from 2 days to 3 weeks. Clinical signs in lsptospirosis are different. Depending on the course, acute, acute, semi-acute, chronic and latent forms are distinguished. In cattle, sheep, goats, goats, deer, it happens as follows:

Very sharp passage . A person suffering from an illness can be put on the spot . Hadeb becomes restless.

He breathes and his heart beats faster. The number of erythrocytes drops to 1-3 million. Hemoglobin decreases by 12-15%. There are different degrees of fatigue . A sick animal urinates often, and the urine is cloudy and dark in color. Diarrhea is observed, and the body temperature rises mainly at the beginning of the disease (40-41.5 °C) . A mole trembles before death. The disease passes very quickly and the animal dies within 12-48 hours.

A sharp passage . In this case, the body temperature rises to 40.5-41°C and can last 7-8 days. A sick animal becomes restless, has diarrhea and becomes weak. The milk of dairy cows decreases sharply. Later, atony (constipation) is observed in the gastrointestinal tract, and the stomach becomes bloated . Jaundice occurs, and the mole urinates with blood. Body temperature is normal, sometimes it can drop. In some cases, conjunctivitis and tears flow. He breathes and his heart beats faster. Necrosis occurs in the head, neck , udder, genitals, and mucous membranes of the mouth. Sometimes there are cases where three parts of the penis, tail, udder teat, and outer lips of the genital organ are cut off . Necrosis can cover most of the skin on the outside of the body. Appetite and cravings stop. Stomach - intestinal peristalsis stops and constipation stops. The strait moles give birth. Hemolysis occurs in the blood, leukocytosis is observed, and bilirubin increases. Death occurs in 50-70 percent of cases if not properly treated in time .

Semi-acute course. The above-mentioned condition does not develop well and goes unnoticed. In most cases, sick animals recover. Fever continues in a relapsing state. Sometimes it's cold and cows give birth. Hemolysis may not be obvious. If the disease lasts up to three weeks and treatment is not intensified, death is observed in 20-40 percent of cases. So the lost goods will gradually return to their original state.

Chronic course . Fever returns periodically and lasts 4-5 days. Recurring 3-4 times, the disease lasts 4-5 months. In rare cases, jaundice and hemoglobipuria occur. Appetite disappears, strong atony and constipation are observed. In some cases, even if the appetite is maintained, the sick animal continues to lose weight. She may not give milk. A child sheds, a young animal grows up. In relapse, hemolysis occurs and leukocytosis is observed.

become unfit for the farm and are slaughtered. Some cattle die from severe emaciation .

Atypical behavior. Clinical signs are the same as in the acute stage and are not obvious. A sick animal does not eat anything and becomes restless. Reversible fever, milk depletion, weak hemoglobinuria, constipation and diarrhea develop rapidly.

Sometimes strait moles give birth. The disease lasts 5-10 days and becomes chronic. Death is not observed.

Y pig leptospirosis. When the disease becomes acute, the patient suddenly stops eating anything, the body temperature rises to 39.5-40.5°C, severe jaundice occurs, the muscles of the stomach become very tense. .

Gastrointestinal activity slows down and causes colic. Urine is coffee colored. The disease lasts from 12 hours to 2-3 days and ends with 100% death.

A sharp passage . Appetite decreases, temperature rises to 39.2-39.5°C, the body becomes pale and sweaty. Sometimes the appetite suddenly stops, and the body temperature rises to 40-45.5°C. Nausea increases . During this period, the body temperature drops, it becomes difficult to walk. Sagrin and o or musk ul lar are painful. Ich ket alternates with constipation, mild colic. It becomes difficult to urinate and becomes yellow, dark-yellow, yellow-brown and dark-gray in color. Breathing and heart work becomes difficult. There are ulcers on the mucous membranes of the oral cavity . The fur of the body falls and the skin grows.

Little bitches give birth. There is a strong hemolysis in the blood, and leukocytosis peaks. The disease lasts 5-18 days and 40-60 percent of animals die.

Semi-acute course. Above clinical signs are observed, but not clearly visible and last longer. The fever keeps coming back. Often hypothermia will be (36.5-38.5° C). Sick mol lose weight, the disease lasts up to a month. Mortality is 10-15 percent.

Chronic pain. Most often, the disease occurs in acute and semi-acute years. Despite maintaining appetite and good nutrition, the animal is pale and lethargic, gets tired quickly at work, body temperature periodically rises and falls for up to 2-3 months. Sometimes it rises to 39-40°C. Mucous membranes become anemic and more yellowish. Hemolysis is observed in the blood. Death occurs in 8-10 percent of cases.

Atypical course. The disease begins with a slight rise in body temperature . The animal gets tired quickly, sweats a lot, and has a lot of fat . Mucous membranes become bloodless and slightly yellow. Hemolysis in the blood is weak and the disease lasts 10-15 days. Mortality is 10-15 percent.

Canine leptospirosis. Body temperature rises to 40-410 C and drops to 36° before death · Strong depression and tremors. Pain is felt in the neck muscles. It 's a strong feeling of anger and bloody diarrhea is observed. Severe stomatitis develops . _ The disease lasts up to 10 days. Death occurs in 50 percent of cases.

Pathologoanatomical changes. Necrosis occurs on the skin of a dead animal. Mucous membranes are yellow; Ulcers appear in the cavity of the eye . In most cases, yellowness, serous i n filtration is observed. Under the skin, mucous membranes and blood clots occur . The kidney enlarges, becomes palpable when urinating, and changes characteristic of nephritis occur. Blood clots occur in the bladder and lymph nodes are enlarged. Degenerative changes occur in the liver, and leptospires settle between the tissues.

Diagnosis . It is not difficult to make an acute diagnosis with clear signs, laboratory methods are used to make a final diagnosis. Blood serum is determined by microagglutination reaction using typical leptospira strains. Agglutinin and lysis

appear from the 3rd to 8th day of the disease, and its titer rises to 1:1000 on the 12th to 17th day. This should definitely be taken into account. After the animals recover, the titer is maintained for a long time. The RMA reaction gives a good and accurate result.

To obtain a Leptospira culture, it is planted in Lyubashenko and Terskikh media. Patmaterial (n amunas) should be taken during high fever. Urine should be taken for examination at the end of the disease. A piece of liver and kidney is taken for bacteriological examination. Urine is examined under a microscope to determine the presence of Leptospira .

Differential diag n oz. It is necessary to distinguish the disease from brucellosis, campylobacteriosis , piroplasmidoses.

Treatment. A special blood serum used against polyvalent leptospirosis is used in this. 10-120 ml is injected under the skin. Depending on the age and type, a half dose is injected into the vein. Special blood serum gives good benefits if it is administered in the early stages of the disease.

to this , 10-15000 units of streptomycin are used for 1 kg of body weight 2 times a day for 4 days . This stops leptospira from being released from the kidney. Antibiotics are recommended for constipation: Glauber and i nglyz salts 200-500.0 for adults; 400.0-800.0 for cattle; 40.0-100.0 for sheep and goats; 25.0-50.0 for ч ' chchka; 10.0-25.0 is given to dogs and cats .

2.0-3.0 for sheep and cattle to improve heart function; sheep-goats, pigs 0.5-2.0; 0.1-0.3 doses of caffeine are administered to dogs and foxes. To improve blood composition, 50-5000 ml of 40% glucose solution is injected into the vein.

the prevention and treatment of the disease, if leptospirosis is recorded in the first Farm, it is immediately declared unhealthy and a plan for the health of the farm is drawn up. From now on, all work will be carried out in cooperation with SES employees. After the ban is announced, the following are prohibited:

a) release of breeding stock;
b) selling young animals to workers;
c) import and export of unvaccinated cattle;
g) organizing groups without the permission of a veterinarian;
d) forced slaughter and distribution of meat;
e) use of open water sources ;
j) introduction of sows to the place where previously infected cattle were ;
z) introducing cattle to the pasture where sick cattle graze for up to a week.

It is necessary to improve the sanitary condition of livestock farms and buildings.

must be slaughtered in a special place in accordance with the rules of veterinary hygiene .

Sick animals are isolated and treated, and healthy ones are vaccinated. The restriction is lifted after 30 days after the farm is cured.

Human leptospirosis (Vasiliev-Weil's disease). The body convulses, the body temperature rises to 39-40°C, and the person becomes confused, sometimes the dizziness increases. Temperature repeatedly pops up and down. He has a severe

headache, his body aches and he groans . After 7-8 days, yellowing occurs. The liver is enlarged and painful, and nephritis is always observed.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention .

Subject: PASTERELLOSIS DISEASE
Pasteurellosis

Lecture	**Pasteurellosis disease**
Lecture teaching technology	
Study time: 2 hours	of students: 90 people
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	*Results of educational activities:* Students: Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.

Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.
3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.
4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.
5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".
6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.net

Pasteurellosis (Pasteurellosis) is a hemorrhagic septicemia infectious disease of mammals and birds, acute septicemia, semi-acute and chronic, more signs of lung damage.

Economic damage. The economic damage from pasteurellosis is huge. Mortality reaches 70-75 percent, sick animals lose weight. The milk of dairy cows decreases sharply. It is necessary to spend a large amount of money on treatment and prevention .

non -spore-forming bacteria, in pairs, rarely arranged in chains. Bacteria in the tissue of diseased animals are small (0.3-1.2X0.5 μm), round in shape and are well stained by the Romanovsky-Giemza method. Pasteurella grows well at 37°C in facultative aerobic, normal aquatic nutrient media. Serum-supplemented media should be used when reseeding a freshly isolated culture . It forms 3 forms in MPA: smooth (S), rough (R), mucoid (Me). Fermentative properties are weak.

Endurance . Pasteurella die quickly in natural conditions. Lives in manure, blood, cold water up to 2-3 weeks, in carcasses up to 4 months, in frozen chicken meat up to 1 year. A fallen bird dies in a few minutes under the influence of sunlight, in 5-10 minutes at 70-90°C. Disinfectants of the usual concentration act quickly.

Economic damage . The economic damage from pasteurellosis is huge. Mortality reaches 70-75%, sick animals lose weight. The milk of dairy cows decreases sharply. It is necessary to spend a lot of money on treatment and prevention.

Epizootology. All domestic and wild mammals and poultry are susceptible to pasteurellosis. This disease also occurs in humans. Among chickens and cattle, the disease is usually epizootic . Horses and carnivorous animals are somewhat resistant to pasteurellosis. Acute hemorrhagic septicemia type of the disease is caused by R. Mi n ossha type V in older cattle . In African conditions , R. Multocida type E occurs in young cattle, R. Multocida type V in goats, and R. Multocida type A in poultry . In the sporadic enzootic pneumonia type of pasteurellosis, it is caused by R. Multocida type A and R. Multocida in whites, and R. Multocida types A and D and R. Multocida in pigs. Sick and sick and recovered animals - Pasteurella carriers are the source of disease. Portability may continue for one year . Long-term carriage of the pathogen by healthy animals is typical for pasteurellosis. The entry of disease-carrying animals into unsanitary farms is the main way in which the disease spreads. Epizootic and stable epizootic focus of Pasteurella summer is its epizootic peculiarity . Cattle and sheep of all ages are affected by Pasteurellosis , but young animals are more susceptible. Most of the roosters are infected, and the death rate is 2 times higher than that of cattle. Pasteurellosis is more common among cattle in tropical countries and the death rate reaches 70-100%. In temperate zones, pasteurellosis is observed in autumn and spring (incidence 1-53%).

Pathogenesis . Under natural conditions, Pasteurella enters the body through the respiratory and alimentary routes, rarely through the skin. Pasteurella multiply at the place of entry, get into the blood and lymph and cause septicemia, death occurs within 12-36 hours. Due to the production of toxic substances, Pasteurella suppresses phagocytosis and damages the capillaries. As a result , edema and hemorrhagic diathesis develops in the subcutaneous and intermuscular tissues . _ _ _ _ _ _ _ quick thinking . Asterella with weak virulence in animals that are resistant to lambswool and the organism se p tisemia does not develop. They have a disease semi - sharp and It is chronic, it is located more in the lungs , and there is a croupous or serous catarrhal fever . p gives. In very acute and acute cases , croupous pneumopia develops , and pulmonary edema and hyperemia appear .

Course and clinical signs . Incubation lasts from a few hours to 2-3 days . Pasteurella can be acute, acute, semi - acute and mild in all animals . In cattle and goats , the body temperature rises suddenly to 41-42 ° C and the general septic condition occurs . Animals die within a few hours due to heart failure, pulmonary edema, and bloody diarrhea . They can die without any clinical signs .

When pasteurellosis becomes acute, general weakness, anorexia, and hyperthermia occur in the animal, and the temperature rises to 40°C and higher. The tip of the nose is dry and cold. Chewing and milking stop, defecation slows down, then becomes liquid, sometimes fibrin fragments and blood are mixed. Sometimes the nose bleeds, acute conjunctivitis occurs, and the sick animal urinates blood. They develop septicemia, death occurs in 1-2 days as a result of heart failure.

Swelling, breast and intestinal forms of pasteurellosis are distinguished depending on the manifestation of clinical symptoms when the disease is prolonged. In the swelling form of the disease, rapidly growing, painful, hot, non-creasing swellings are formed in the subcutaneous tissues of the jaw, neck, abdomen, and legs. When the tongue and neck are swollen, it becomes difficult to breathe, stretchy saliva is released, the visible mucous membranes are bruised, and there is a lot of blood. In some animals, the disease passes with agitation (pasteurellous meningoencephalitis in calves). Symptoms of croupous (fibrinous) pneumonia for the chest form are characteristic: weakness, anorexia, atony, difficulty in breathing, dry painful cough and discharge of mucous foamy fluid from the nose. At the end of the disease, bloody diarrhea occurs. A sick animal dies in 5-8 days.

pasteurellosis in pigs is acute and acute, fever occurs, body temperature rises (41° C and above), pharyngitis, difficulty breathing, heart failure, swelling of the jaw and neck. Animals die from asphyxiation in 1-2 days. Fibrinous pleuropneumonia, cough and mucoid purulent rhinitis develop when the disease is prolonged. The disease ends with death in 5-8 days. In chronic cases, symptoms of pneumonia, slight weight loss, and sometimes swelling of the joints are observed.

In fur-bearing animals, when the disease is acute, there is sudden weakness, anorexia, slow and wobbly walking, and an increase in body temperature to 42 °C. Symptoms of hemorrhagic gastroenteritis develop in foxes. In black cousins, swelling occurs in the head area, subcutaneous tissue, and hind legs are paralyzed. The disease lasts from 12 hours to 2-3 days.

Pathologoanatomical changes. These changes depend on the duration and form of the disease. In acute and late acute cases, hemorrhagic diathesis is seen in dead animals (hemorrhage and inflammation in organs, mucous and serous membranes), liver and kidney are damaged, spleen is slightly swollen, lymph nodes are swollen , dark-red in color, serous fibrinous infiltrates are seen in various parts of the body in the subcutaneous tissues, especially in the edematous form of the disease. Pulmonary edema is a characteristic change in the initial stage of croupous pneumonia. Fibrinous-hemorrhagic inflammation is seen in the gastrointestinal tract in the intestinal form. In semi-acute and chronic cases, dead animals are emaciated and bloodless, the pre-bronchial lymph nodes are enlarged, reddened, and contain a lot of blood. Foci of necrosis are visible in the lungs. The spleen is slightly enlarged,

there are small necrosis foci in the liver and kidneys. Pathologoanatomical changes in birds are similar to those of mammals and mainly depend on the course of the disease.

Diagnosis . Pasteurellosis is diagnosed based on the results of epizootological data, clinical signs and pathologoanatomical changes, bacteriological examination (extraction of a pure culture of virulent Pasteurella for white mice). Spleen, liver, kidney fragments, damaged lung sections, lymph nodes and tubular bone are sent to the laboratory. These litters should be taken 3-5 hours after the animal's death and before treatment. Small animals are sent whole. In the summer months, the feather material is preserved in a solution of 40% glycerin in water.

Differential diagnosis. Anthrax, piroplasmidosis, and rabies in older cattle, and staphylococcal and streptococcal infection in young animals, salmonellosis, colibacteriosis, and respiratory viral infections (parainfluenza-3, infectious rhinotracheitis), epizootic bronchopneumonia , in chickens from plague, scurvy and salmonellosis, in sheep from anthrax, piroplasmidosis, clostridiosis and streptococcal infections , and in chickens from Newcastle disease, spirochetosis, mycoplasmosis and infectious should be able to distinguish from laryngotracheitis.

Treatment . Sick animals are transferred to warm, dry buildings, provided with nutritious food. Antibiotics, tetracycline series and s u lfa n ilamide drugs are used according to the instructions.

Anti-pasteurellosis serum is useful if it is used at the beginning of the disease, when the disease is acute, when the first clinical symptoms appear. The serum is administered intramuscularly or intravenously in twice the prophylactic dose. The effectiveness of treatment increases if the serum is added together with prolonged-acting antibiotics, sulfonamide drugs. The course of treatment depends on the condition of the animal. Poultry infected with pasteurellosis are not treated.

Immunity . Animals that have recovered from Pasteurella disease have immunity for 6-12 months. For special prevention, N. Nikiforova's precipitated formal vaccine, AzNIVI's semi-liquid formal aluminum hydroxide vaccine, polyvalent vaccine against pasteurellosis and diplococci, vaccinated vaccines are recommended. It is necessary to use them strictly following the rules, taking into account all epizootic conditions .

Vaccines are forcibly used for preventive purposes, in unhealthy farms and in dangerous places. Animals are vaccinated twice with precipitated, semi-liquid and concentrated vaccines. Immunity appears 7-10 days after the 2nd vaccination and lasts up to 6 months. The vaccine is given once together with the vaccine. Immunity lasts for one year. Dry, live vaccines are used to prevent pasteurellosis in poultry. French (Pasterovsky) avirulent and weak avirulent strains (K and AV Krasnodar NIVS) and inactivated vaccines are used . Live vaccines are used to vaccinate chickens and waterfowl from unhealthy farms and dangerous zones.

Prevention . To prevent pasteurellosis, it is necessary to take measures to prevent the introduction of the pathogen into healthy farms by sick animals and pasteurella carriers and feed. The main attention should be paid to the observance of general veterinary-sanitary rules and keeping animals in normal zoohygiene

conditions and feeding them on the basis of a balanced ration. All animals should be vaccinated against pasteurellosis throughout the year if the disease has been recorded in the farm before. It is necessary to fill such farms only with vaccinated animals.

Control questions

6. Pathogenicity and susceptibility .
7. Epizootology .
8. Pathogenesis, clinical symptoms .
9. Pathologoanatomical changes .
10. Diagnosis and differential diagnosis.

SUBJECT: SMALLPOX
Variola

Lecture	**Smallpox _**
Lecture teaching technology	
Study time: 2 hours	of students: 90 people
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	*Results of educational activities:* Students: Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.

Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.
3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.
4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.
5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".
6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.net

Smallpox is an acute infectious disease caused by a virus. Special papulosis-pustulosis exanthemas appear on the skin and mucous membranes.

Economic damage . The economy suffers a lot from the disease. Mortality can reach up to 50 percent. In addition, quarantine measures require a lot of money. In pig farming, piglets die around 70-80 percent. If cows are infected with smallpox, their milk will decrease dramatically.

Trigger. The causative agent of smallpox is a virus belonging to the poxvirus group and has epitheliotropic properties. A special body appears in the disease. In

living tissue culture, it grows and develops in the chorioallantoic membrane of the chicken embryo. Although smallpox viruses are very similar and close to each other in terms of morphology, some of them are completely distant from their ancestors from the immunobiological point of view. For example: sheep pox virus is independent and causes disease only in sheep. Goat pox virus is also dangerous only for goats. Cattlepox virus can cause disease in humans, monkeys, pigs and horses. The same situation is characteristic of the virus of chickens and other poultry .

Endurance year . The virus is resistant to high temperatures , it dies in 20 minutes at 55°C. The virus is preserved in smallpox crustoses that have fallen from the body of a sick animal . The herb stays alive in tons for up to 6 months. A sheep that gets sick in pasture conditions can live for up to 2 months. 2% formalin, 3% alkali and 2-3% solutions of carbolic acid are recommended for de - infection. Antibiotics do not affect the virus .

Epizootology. Sheep and goats, pigs and cattle, goats, chickens and poultry are infected with smallpox.

Sheep pox . Sick sheep are considered to be the source of the disease . The virus is transmitted to the environment through the mucous fluid from the nose, mainly from dried smallpox spores . The disease is transmitted through direct contact with caerogen oil , through wounded skin and mucous membranes , sometimes through food . In very rare cases, it can also be transmitted in the womb. In the case of disease, n sheep are transferred to a healthy farm and spreads as a result of q ' growth. In the spread of the disease, other types of animals , people, and vehicles can also be a virus-carrying factor.

Smallpox epizootics occur at any time of the year, but the disease is most severe in winter. Rainy days are also the cause of the fatal course of the disease. Sheep with fine wool are seriously ill. The disease is also severe in lambs and many die. This is especially evident when forage is weighed.

Cattle pox. It is often observed in people, especially young children, when vaccinations are carried out. The disease spreads rapidly among dairy cows after they are kept in one place on farms . Especially, the first broods are more infected , and the weaned bees are less infected , and the disease is transmitted behind the udder .

The warm ones smallpox The disease spreads in an epizootic state and is rare. In this too , people or cattle play a certain positive role . In some cases, the opposite can happen .

Swine pox. In many cases, it coincides with the time when cattle are sick . The disease is transmitted through cows or by inoculating pigs. Sometimes pigs get sick even when people are vaccinated against smallpox . Sometimes it is transmitted by an immunologically independent herpes virus . The virus enters the organism through air , skin or mucous membrane wounds . Young pigs often get sick . In the spread of the disease , pigs are also very important.

Pathogenesis. After entering the organism, the virus appears in blood (erythrocytes), parenchymatous organs and spleen after 3-4 days . A state of viremia is observed for 2-3 days.

The virus is transferred to the skin, mucous membrane, lung epithelium and cornea of sheep with blood. In these places, the virus grows and multiplies and causes exanthemic changes characteristic of smallpox. When smallpox passes in a typical state, a step-by-step pathological process occurs. After the virus enters the blood, it spreads throughout the body, fever rises, rhinitis and conjunctivitis begin, after 1-2 days, the body temperature drops, the virus disappears from the blood and settles on the skin and mucous membranes. Due to this, small red spots (roseola) appear on the skin and mucous membranes. After 1-2 days, in the place of roseola, there are nodular swellings enriched with a reddish garden. The lesions are called papules. Papules appear mainly due to the strong proliferation of the virus in the tissues. After 2-3 days, reddish-yellow serous fluid accumulates inside the papules and forms a blister, which is called a vesicle. It occurs mainly due to the lysis of degenerated tissues. After 5-6 days, as a result of the accumulation of pus-producing microorganisms in the vesicles, the process of suppuration begins, which is called **a pustule .** By this time, the condition of the sick animal worsens , the body temperature rises. After 3-4 days, the pust starts to dry , and in its place , brown spots appear , this condition is called crustosis.

Passing and kl ini k signs. Smallpox is acute and the latent period lasts 4-10 days.

Sick sheep are sick and have a high body temperature . Mucous membranes and eyelids swell. Mucous-pus mixed fluid flows from the nasal cavity. After 2-4 days , roseolas appear on the head, udder and udder, on the skin of the genitals, sometimes on the crotch. In some cases, chicken pox is very severe . Confluent and hemorrhagic conditions appear. In the form of enlarged or swollen papules , they grow together , take up the floor space and accumulate pus . The body temperature rises, and sick sheep can die of sepsis. Hemorrhagic (black) pox occurs as a result of blood clots in the inner and inner spaces of the papule . Bleeding and blood-tinged diarrhea can occur . When smallpox is complicated, pneumonia, gastroenteritis and purulent arthritis are observed. An eye injury can lead to blindness . If the disease coincides with the period of lambing, the child will start throwing.

In cattle, the disease begins with slight weakness and an increase in body temperature. Then several roseolas appear in the udder . The right- hand side shows the step-by-step development mentioned above . The udder is damaged, passes into the parenchyma and becomes mastitis. Papules and pustules on the skin of bulls _ _ _ changes happen. The papules and pustules burst , and the result of blood clots is formed .

Calves have skin lesions and papules on their lips and beaks. It is relatively easy.

Smallpox- like changes occur in the mucous membrane of the oral cavity in the form of pustulosis-stomatitis. In some cases, conjunctivitis occurs, roseola, vesicles and pustules appear on the nose, skin, around the hooves, lips, teeth and tongue. As a result of the bursting of bubbles, pocketed erosions are visible. A lot of saliva flows, the lymph nodes under the throat are swollen.

In pigs, the body temperature rises, they become weak, conjunctivitis is observed. Smallpox rashes appear on the abdomen, ears, and udders. The injured area itches. Older pigs get sick easily and recover quickly. In young pigs, the disease is severe and 60-80 percent die. The mucous membranes of the mouth and nose are also damaged, and pneumonia develops. A complicated course is observed and salmonellosis can be added. In some cases, smallpox occurs.

Goats sometimes have open pox, the same symptoms as sheep. In case of complications, pneumonia, mastitis, vomiting are observed. Most of the time it is mild.

Camels have a fever, the mucous membranes of the nose and mouth are red, and mucous fluid flows. The lips and nose are slightly swollen. The lymph nodes of the bone are enlarged . Later, smallpox rashes appear on the mucous membranes of the nose, lips and mouth. The most characteristic rashes are clearly visible on the skin and hairless areas of the body . Rashes also occur around the genitals and on the skin . Older camels will recover from the disease in 40-50 days, and in camels, it may be difficult and death may occur . Sometimes old camels have diarrhea .

Pathologoanatomical changes. Smallpox rashes are visible on the skin of cattle that died from smallpox. On examination, mucous membranes are inflamed, and sometimes erosion is observed. A state of hepatization has occurred in the lungs, the stomach is inflamed, the mucous membranes are slightly swollen, pustules can be found.

Diagnosis . Diagnosis is made by looking at the typical changes characteristic of smallpox (clinical method). The epizootology of the disease is studied. Pathologoanatomical examination is carried out. Sheep brought from a healthy farm are bioscreened.

Differential diagnosis . It is necessary to be able to distinguish it from eczema with pustulosis . Smallpox is non-contagious, and smallpox is contagious, with a distinctive rash. In smallpox, a Paschen corpuscle appears (visible under a microscope) . Pust u ll yz dermatitis goes from acute to chronic . Often the temperature does not rise, the lip is severely injured.

Cattle should be separated from the protein. Aphtha appears in the white, and it is located between the hooves and the udder . In suspected cases, the disease is detected by biochemistry.

Treatment. Sick animals should be provided with good food , but it is necessary to protect them from rain, snow and wind . In the case of complications, zinc and 2% alysium oil are used for symptomatic treatment . 2% streptocid, iodine - glycerins gives a good result. The injured area is thoroughly washed with a 1 :3000 solution of potassium permanganate . Propolis oil diluted in 20-30% petroleum jelly is very useful.

Prevention. The main task is to take measures to prevent the disease from occurring . The purchase of goods is carried out at the expense of the healthy economy, and preventive vaccinations are carried out according to the plan . Imported goods are kept in preventive quarantine for 30 days . Quarantine is declared on the farm when smallpox is recorded. Sick sheep are isolated and treated, and

healthy flocks are vaccinated. Mandatory vaccinations are carried out in farms deemed dangerous . According to the requirements of the quarantine, all the roads were blocked , and the movement of goods was prohibited . Entry and exit of foreigners to the farm is prohibited . It is important to change the places of Moll a r it will be done.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention .

Topic: Iron disease

Lecture	**Iron disease**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
Form of training	Enter. Visual lecture
Lecture plan About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Education	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Training conditions	Specially equipped lecture hall.
Monitoring and evaluation	Oral examination. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Educator*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results. 1.2. Planning.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick answers:* Planning objects. About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1. Listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. The attention of the students who participate in the lecture is focused on the main issues, active students are encouraged. 3.2. The house is manufactured and evaluated.	3.1. He listens and clarifies. 3.2. Can be written.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.net .

Temiratki is an infectious disease that occurs in farm animals (cattle, sheep, goats, camels, deer, fur animals, dogs and cats, etc.) and humans. The appearance of wounds (spots of various shapes) on the skin is the main symptom of the disease.

Historical information . The disease has been known for a long time and is often found in different parts of the world.

Due to the planned use of the LTF-130 vaccine during the former USSR, iron deficiency was greatly reduced in many places. To date, vaccination of susceptible animals has been disrupted due to the lack of vaccine supply. This in turn led to the spread of the disease. It has become difficult to find a farm where iron is not found.

Economic damage . The growth of diseased cattle slows down. The fur of fur animals is damaged. Wool production in sheep is reduced. Treatment takes a long time, requires a lot of work and money. The worst part is that the disease is

transmitted to people. During the course of the disease, the weight decreases to 1.2-2.5 kg. Sick animals, in turn, spread the fungus , affect and destroy the ecology .

Epizootology. Mostly young animals are affected and it occurs in all seasons of the year, more often in winter and early spring and late autumn. The same seasonality is observed in neighboring countries (Russia, Ukraine, etc.). By this time , factors such as lack of fodder, wind blowing, increasing humidity, slowing down of movement on the farm accelerate the spread of disease . This situation is very bad for the civilization of the countries . That 's why the sanitary condition of iron is very common in low-income farms and is recorded throughout the year. Eat and live _ Lack of response to therapy reduces the animal's endurance and increases its susceptibility .

A sick animal is considered the source of the disease . LX Sarkisov, LI Moskov, VP Koroleva (1956) noted that trichophytia occurs in cattle throughout the year, mainly in winter, early spring, less often in late autumn, and very rarely in summer. Sh. T. Ras u lov (1970) also in Uzbekistan, trichophytosis of cattle escalates in the winter months, mainly affecting calves under one year of age . F. Zalikhanova, Ye. Marininlar (1972) notes the increase of iron in autumn-winter months in Kabardino-Bolgor AR and Vologda region. If we analyze the literature on iron pox published in 1960-1970 , it is noted that the disease starts mainly in calves in August-September and escalates in winter months.

Yilqilar temiratkisi . In the former Union, on the eve of the 1930s, it was noted that the production of annuals was widespread. The disease was observed throughout the year. For example, at the hippodromes, it is found in any season of the year, because the yearlings come here brought throughout the year. And in horse farms, it depends on their breeding . SV Petrovich and VV Adsryushin write that they meet in almost any age , but mostly in winter . They proved that the sex, breed and color of the yearlings are not important in the spread of the disease. In natural conditions, chickens get sick from the age of 2 months. The disease was especially widespread among them in the fall , and peaked when previously infected cattle and horses were brought into the buildings.

Camel factory. S. Khamiyev (1987) showed that the spread of the disease is caused by contact of camels with sick camels and their emaciation. This is especially evident when the herds are raised in the place where the sick camels were kept. Flocks get sick in October , when they are 5-6 months old. In some cases, the disease occurs in 2-3-year-old camels. We witnessed a case of a 3-year-old camel in the village of "Mingb u Loq" with severe trichophytosis.

M. Azimov (1961) noted the disease in zebu and said that it occurs mainly in children from one month to 1 year.

Among nutria and other fur-bearing animals, iron spreads very quickly. In a short period of time, the disease spreads up to 70-80% and causes great economic damage. The main reason for this is that they are kept indoors in cages (RFR) and in our conditions they do not spread as much as they are kept outdoors in large wire mesh cages.

The disease is also widespread among rabbits, and in some cases it reaches 86-93 percent. The disease occurs in spring and summer months and is mainly observed in young rabbits. According to some data, it is more common in the winter months (L. Nikiforov).

Rodents are also infected with iron, and they can sometimes become a source of pathogens for farm animals.

Triggers . The causative agents of the disease are fungi (Trichophyton verrcosome, T. Eqvinum, T. Mentagrophytes, T, Sarcisovi, M. Eqvinum, M. Canis), which can live in buildings and barns for several years. , fences, doors, wood and the edges of containers can easily maintain their virulence for 2-3 years. When the material taken from a sick animal is examined under a microscope, arthrospores are seen in the form of semi-glossy spherical balls arranged in rows or irregularly around the affected wool fiber.

The causative agents of the disease mainly grow well on wort-agar, MPGA with 2% glucose with meat-peptone and Saburo agar, after 3-4 weeks fluffy fluffy colonies appear. Depending on the type of fungus and animal, they grow as leathery, brown, yellowish fluff when cultivated. When separated, microconidia and chlamydospores differ depending on their condition.

Pathogenesis . On the basis of pathogenesis, the fungus - the causative agent affects the skin and its mucous membrane. The pathological process begins with inflammation of the follicles of leukocyte infiltrations. In the place where the fungus has fallen, the upper layer of the skin turns red, a papule appears, and then it turns into a blister. The fungus develops in the stratum corneum of the skin and secretes proteolytic and keratolytic enzymes. This, in turn, affects the mucous membrane and dissolves it. As a result, the mycelium of the fungus penetrates into the follicle of the double fiber. The place where the fungus entered is inflamed and exudate is released. As a result of the violation of the natural condition of the skin and the epidermis layer, pimples appear. Mucous substances harden as a result of dust and other impurities falling on the skin, forming a hard coating. Wool fiber dries and begins to break. Secondary substances produced by toxins and inflammation irritate the skin. In superficial mycoses, the pathological process occurs in the epidermal layer of the skin, and the wool and its follicles are damaged. In case of deep mycoses, the damage goes to all layers of the skin and covers the secretion glands. Dropping of pus-forming microbes. As a result, a suppurating area is formed. The integrity of the skin is of great importance in the pathogenesis of tinea. Due to the injury, the skin loosens and clots. As a result, the resistance of the skin tissue decreases, and the fungus enters the body and settles faster. In addition, trauma disrupts the barrier between blood and tissue. Fungal elements spread from the wound site to the blood and lymph throughout the body. This in turn causes leukocytosis and monocytosis.

Clinical signs. The latent period of the disease lasts 1-4 weeks, and its appearance depends on the organism of the animal, the virulence of the fungus , the place where it fell, and the season of the year . The disease occurs in superficial, deep, foliar and atypical cases . In cattle, iron spots appear in circular, circular forms on the upper part of the skin . These spots can be found on the head, neck , shoulders,

abdomen, and legs. Its shape and size are different. At first, there are hard, small bumps on the skin, which later take the form of slightly swollen spots . Its surface is completely covered with la r and forms a scaly surface. It has a yellowish, greenish-blue color. Wool fibers break. In deep mycoses, the injured area is strongly inflamed and exudate is formed. When the injured area is pressed, pus begins to glisten from the cracks. This situation is observed in many farms with low economic status, which do not meet sanitary requirements.

In horses, the disease occurs mainly with the appearance of round spots on the skin of the body. The wound can be found in any part of the body, covered with bluish scales, and the surface is scaly. In deep mycoses, a hard crusty surface is formed. It is accompanied by suppuration in microsporia and deep mycoses. It is also found in the vesicular state in horses. In iron, the injured area swells a little, loosens and pus accumulates.

In camels, iron spots are often located on the head, covered with thin scales, and are circular or circular in shape. The injury gradually spreads to other parts of the body. In damaged areas, wool loses its softness and shine, begins to break. In the case of blisters, the injured area itches, and in severe cases, the blister may die. We observed such a situation with VIEV employees in the village of "Mingbulok". Temiratki is also found in reindeer, and the head part is mainly damaged (E. Beradaev, L. Ivanova, 1971). The fur on the injured area begins to shrivel, break and shed. In working deer, hind legs are often injured, and they are very prone to antlers. They also have superficial and deep leukemias. In zebu cattle, the disease occurs in the form of superficial and deep mycosis, iron spots start from the head and then spread everywhere.

In nutria, there is hyperemia, the skin thickens, and mucus is released. Then the exudate is separated and thickened skin appears.

In rabbits, the head and muzzle are often affected, and after 10-15 days, it spreads to other places. Broadness becomes a state of generalization. In some cases, small iron spots merge, become larger and occupy a large part of the body.

Diagnosis. The diagnosis of the disease is made using a complex examination . Epizootological information, clinical signs of the disease and the results of mycological examination are taken into account.

Epizootology . The disease is recorded throughout the year, but peaks in the winter months and decreases in the summer. In the origin of the disease, factors such as feeding animals, keeping them in dense and moist, semi-dark places are of great importance. In the clinical case , the clinical symptoms of the disease are taken into account. The results of mycological examination are decisive .

Pathological material and its classification . Pathological material is taken from the injured area of a diseased animal. Q-10 pieces of damaged wool fiber are taken and placed on a glass with black paper underneath. Then the product is placed in a window, dripped with 10-15% alkali solution and slightly heated. Checking under a microscope, attention is paid to the location of arthrospores. Plants are grown in the previously mentioned media and growth is monitored .

Immunity . _ It was proved 20 years ago that cattle infected with iron pox develop immunity. First TF-130, and then LTF-130 vaccine in practice confirmed this opinion and m u lokhas . Academician AH Sarkisov founded and led the great discovery in this field at ViEV. After the above-mentioned vaccine, "mentavak" - for fur animals, "trikhovio" - for sheep, "kamelvak" - for camels, SP-1 - for yearlings were developed and introduced in the same organization. .

And unlike a. The LTF-130 vaccine is used for the prevention and treatment of bovine tuberculosis . The vaccine is released in a dry state and has 10, 20, 40 doses. A special liquid is added to dissolve it. If such a solution is not given, sterile physiological solution is used, the prophylactic dose is 5 ml for 1-4 months old animals, 6 ml for 4-8 months old animals, 10 ml for animals over 8 months old. The vaccine is given twice with an interval of 10-14 days. Immunity lasts for 1 year, after which the sick person will not be infected.

Treatment. For treatment, the LTF-130 vaccine is given in the same scheme as above, but the dose is increased by 2 times. If the animal is seriously ill and does not recover quickly, it is possible to administer the vaccine a third time in a therapeutic dose.

In the summer months, a mixture of 7% alkali and formalin prepared in petroleum jelly gives good results. It is applied every 5-6 days.

The taste of the mixture of "Yam" oil is high. If available, a mixture of 1.5% lye can also be used.

Prevention. In healthy farms, all calves are given 2 prophylactic vaccinations at 10-14 day intervals after reaching the age of 1 month.

All veterinary and sanitary measures are carried out on time.

Sick animals are isolated and treated according to the instructions.

- Cattle breeders must strictly follow the rules of personal hygiene . A mixture of alkali with formalin (2 l of alkali, 5 l of formalin and 100 l of water) is the most effective for disinfection. When the disease is registered, restriction will be announced.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis .
6.

Topic: CATTLE DISEASE _
Carbunculus emphysematicus

Lecture	**Cattle distemper**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
Form of training	Enter. Visual lecture

Lecture plan About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Education	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Training conditions	Specially equipped lecture hall.
Monitoring and evaluation	Oral examination. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Educator*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results. 1.2. Planning.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick answers:* Planning objects. About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1. Listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes

		down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Can be written.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.ne

Scabies is an acute infectious disease that is not directly transmitted and occurs in cattle and sheep. In fleshy areas of the body, a creaking (crepitation), well-defined swelling appears, the animals become limp and quickly die.

Economic damage. If it is not treated in time, the diseased animals will die. A lot of money is spent on quarantine and treatment. A herd of cattle from 3 months to 4 years old is fully vaccinated according to the plan. For this event, in turn, a large amount of vaccine is produced or purchased. If the disease is registered, sheep and cattle must be vaccinated.

Trigger. Cl. Chauvoei is a stick with a curved tip. It is well stained with alkaline-apilin dye and Gram method. Spores can be painted in any way. The pathogen is strictly anaerobic, the most favorable medium for cultivation is Kit -T arossi and Hottinger broth.

The microorganism emits an odor when applied, similar to the odor of rancid oil during growth. The microorganism is strictly anaerobic.

Endurance. Clostridium spore is very resistant. 100°C will die in 5-10 minutes. It can live up to 2 hours at 80° C. It can be stored for several years in dried meat, and up to 6 months in rotten material. According to Kallam (1952), clostridium can be stored in sea water for up to 6 months. The pathogen can remain viable for a long time in manure, burial, and uncontrolled litter. (Prevost, 1977). To maintain its virulence, it is periodically tested by infecting calves and guinea pigs.

Epizootology. Cattle from 3 months to 4 years old are highly susceptible to the disease. Adult animals gradually become immune throughout their lives, but this is not absolute. Young animals are protected from disease due to passive immunity. In the body, this situation occurs due to colostrum and milk in general. If special methods of disease prevention are violated in the farm, animals of all ages will get sick. Purebred cattle are more susceptible than others. The degree of susceptibility is evident when cattle are brought from a healthy farm to an unhealthy one, and the disease is very severe and destructive.

Pathogenic. According to the conclusion of the majority of scientists who studied the disease, transmission occurs mainly through the oral cavity. In natural transmission, spores enter the animal's body mainly with food and water. Later, it

passes through the injured or scratched area of the mucous membranes and reaches deep tissues. Here, in turn, it grows and develops. Academician Ya. Kovalenko placed the importance of injuries in the origin of Karason in one of the main places. In the development of the disease in young animals, injuries that occur during teething and replacement are the main causes. During growth and development, the microorganism secretes toxins and aggresins, which in turn protect them from phagocytosis. The worm that hatched from the eggs of the sona got into the body. during its development, it causes various injuries and causes the occurrence of black disease. The importance of muscle injury in the transmission and occurrence of the disease is greater than that of the skin part of the body. This is the condition that completes the manifestation of the disease. Fat cattle are often infected with Karason, and the disease is very rare among lean cattle. The main reason for this is the presence of a large amount of glycogen in the muscles. Glycogen, in turn, is the best source of nutrients for C lostridium shavoye. The causative agent can be isolated from the body of a freshly dead animal. Because bacteremia is observed in the body before death.

Clinical symptoms. The latent period of the disease lasts 4-5 days, is acute and usually ends in death. The disease begins with a sudden rise in body temperature (40-42°C). The diseased animal is lame. In the most fleshy parts of the body (thighs, thighs, etc.), a strictly limited, hot and hard swelling appears. In infected cattle, the clinical signs of the disease develop quickly and are clearly manifested within 8-10 hours. If the swollen area is pressed, a crepitation sound is heard. This happens as a result of the formation of gas bubbles in the injured area. In some cases, the injury also occurs in the throat, diaphragm, jaw, and chewing muscles, where the muscles are less developed.

Pathologoanatomical changes. The characteristic changes of the disease are the manifestation of a red inflammation of the mucous membrane and a limited hemorrhagic necrotic myositis in the connective tissue between the subcutaneous muscles. The injury occurs mainly in the thigh, neck, shoulder, chest, abdomen, rarely in the throat and diaphragm, tongue and myocardium. The affected area is dark-red, sometimes very dark. When cut, it can be felt that there are air bubbles and a dry and porous structure.

If the wound is cut deeper, it is sometimes revealed that the inside is bluish-yellow in color. When the regional lymph nodes are cut, the fluid is separated and becomes cystic. The chest and abdominal cavity, around the heart sac are filled with a reddish thick liquid.

Sometimes the mucous membranes are covered with a soft thin layer of fibrin, in some cases the spleen is slightly enlarged and softened, protein and fatty dystrophy is visible. The base of the capsule is drier, gas bubbles, walnut-sized necrotic foci appear. The kidney softens, the bark is reddish-yellow, and air bubbles are observed. There are no visible changes in the gastrointestinal system, sometimes inflammation can be observed in the jejunum and small intestines. The heart expands a little, the epicardial base is filled with blood, and granular dystrophy is observed. The lungs are swollen and swollen **.** All the above-mentioned changes are not always noticeable. In

some cases, the change to the disease can occur only at the site of the injury. During the histological examination, it is determined that the muscle fibers have undergone necrosis, fibrin is formed, and leukocytic reactions are present. Among the muscle fibers, germ cells, hemorrhages and air bubbles are noticeable.

Diagnosis. Black epizootological observations (age of the animal, breed, season, etc.), clinical signs (squealing swelling, lameness, high body temperature, injury in the fleshy area, etc.), pathologoanatomical changes, bacteriological, determined according to the results of biological tests. A piece of injured flesh, exudate or wound is taken from the site and sent to the laboratory for examination. Testing is carried out as follows: finding the pathogen by observing it under a microscope, planting it in artificial environments and infecting laboratory animals.

Microscopic examination. An ointment is prepared in a clean object bottle, degreased from the meat taken from the injured area. It is stained by Gram or Muromsev method. When examined, polymorphic spore-shaped, round and pear-shaped microorganisms are observed. They are surrounded by spores, and spores do not accept dye.

Differential diagnosis. First of all, it is necessary to distinguish from anthrax. In anthrax, blood does not coagulate, swelling rarely occurs, mainly reminiscent of a carbuncle , **and** does not vibrate. The causative agent is aerobic and is surrounded by a capsule in the body and spores in the external environment. The causative agent of anthrax is strictly anaerobic and does not have a capsule. It is more difficult to distinguish from a dangerous tumor, so it is advisable to use laboratory methods. Guinea pigs die within 18-48 hours when infected. In malignant edema, there are lesions on the skin and mucous membranes of the mole. In cows, this disease occurs when childbirth is difficult, placental abruption, inflammation of the uterus, and after abortion.

Treatment. Since the disease is acute, treatment is not always beneficial. At the beginning of the disease, the use of hyperimmune blood serum according to the instructions gives a good result. Chloro-tetracycline is administered intramuscularly at 8-5 mg per 1 kg of weight once a day for 4-5 days. A single injection of dibiomycin suspension in a 40% glycerine solution in the amount of 40,000 TB per 1 kg of live weight gives a good benefit.

Until the general condition changes, it is advisable to inject 5-7 thousand TB of ampicillin per 1 kg of weight dissolved in 0.5% novocaine every 6 hours.

It is recommended to use Bicillin-3 in the amount of 10 thousand TB for cattle, 15 thousand TB for calves, and 15-20 thousand TB for sheep and goats. If necessary, after 10-15 days, the course of treatment is returned again. 2% hydrogen peroxide, 3-5% carbolic acid, 3-5% lysol or phenol, and 0.1% potassium permanganate solutions are injected into the area of the itchy swelling and around it. But these drugs are not always useful, so it is advisable to detect the disease in time and start treatment immediately.

Immunity. There is no complete and reasonable information on natural immunity in cattle and sheep. The tendency decreases with age. The formal vaccine discovered by SN Muromsev has been used for many years and has given good

results. FI Kogan and AI Kolesov developed concentrated formal hydroxide vaccine and put it into practice. This vaccine is still used, and 2 ml is injected into the muscle of the calf. Immunity appears 14 days after vaccination and lasts up to 6 months. Successful experiments are also being carried out on receiving associated vaccines. They are recommended for production. Any vaccine should be administered strictly following the instructions.

Prevention. This event will be conducted as follows:

- susceptible animals are vaccinated according to schedule. If the disease is suspected:
- timely diagnosis;
- declaring a quarantine and eliminating the epizootic center;
- it is necessary to carry out sanation (resin) activities in the equipment, territory and barns to eliminate the carcasses of cattle. An unhealthy farm is recorded in the epizootic journal and a mark is placed on the card.

Veterinary measures: swamps in pastures are drained and the surroundings of water sources are regulated. Cattle houses and slaughterhouses are disinfected. The surroundings of the biothermal well and burial sites are cleaned. When carrying out large-scale soil and reclamation works, strict control is necessary. Quarantine will be announced if the patient is about to leave. Carcasses are burned and destroyed.

Vaccination is completed 14 days before going to pasture, if the pasture period exceeds 6 months, it is revaccinated. Vaccinated with live vaccine 7 days before and once. Quarantine will be canceled after 14 days after the farm is cured **.**

10% alkali solution, 4% formaldehyde, 10% iodine (1)-chloride, 2% glutaraldehyde, etc. are used for disinfection. In case of death, the cut and lying place is first burned, then it is mixed with a 5% solution of chlorinated lime at a ratio of 10 l/m ^{3} by digging 25 cm.

Sheep bleating . The latent period lasts up to 24 hours, sometimes 2-3 days. Diseased areas begin to blister like a wound infection. The swelling area is clearly demarcated, hard, hot, pasty and painless. Squealing may not always be present. The skin at the site of injury dries up, loses its elasticity, its color is dark-red, sometimes black. Throat, tongue, genitals are also affected. The sheep become weak and lag behind the herd, foaming at the mouth, bloated stomach and gnashing of teeth. The disease lasts 6-20 hours and usually ends in death.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7 . Treatment and prevention.

Topic: CAMPYLOBACTERIOSIS DISEASE

Lecture	**Campylobacteriosis disease**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
Form of training	Enter. Visual lecture
Lecture plan About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Education	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Training conditions	Specially equipped lecture hall.
Monitoring and evaluation	Oral examination. Quick survey.

The lecture technological map		
Lecture stages	*Activity content*	
	Education giver	*Educator*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	2.1. Listens, expresses his understanding, thinks, answers.

	2.2. The main theoretical parts of the lecture are described using visual materials.	2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It ends the lecture and students' attention is focused on the main issues, active students are encouraged. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Can be written.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.ne

Campylobacteriosis (lat. — Campylobacteriosis, Vibriosis genitalis enzootica bovis/ovis; visual. — Vibriosis, Vibrio fetus infection of cattle/sheep; Russian-vibriosis) is an infectious disease that occurs mostly in cattle and sheep, sexually transmitted It is characterized by infection of the genitals, frequent estrus in female animals, infertility, mass abortion, retained placenta, and the birth of a non-viable fetus.

Historical information. The causative agent was first identified in 1909 by McFadian and Stockman (England) in the uterus, placenta, and discarded fetal tissues of sheep and cattle in 1913. In 1919, Smith and Taylor named the pathogen Vibrio fetus. According to the current modern classification of bacteria, it belongs to the family Spirillaceae and the genus Campylobacter. That's why at present this disease is called campylobacteriosis, not vibriosis. Currently, campylobacteriosis is spread among animals in all countries of the world. It was registered in the former Union in 1929 by V. Yakimov in cattle. Among the sheep was written in 1929. The service of EV Kozlovsky, PA Trilenko, NN Mikhailov and MA Luchko in the study of the disease is great.

Economic damage. The economic damage is as follows: 20-40% of infertility, miscarriages, treatment and prevention measures cost a lot of extra money. Breeding of breeding cattle and the economic activity of stations where breeding bulls are kept will be completely derailed. Because it is forbidden to breed young cattle for sale, and to take seeds for escape.

Trigger. The causative agent of the disease, Sampyllobacter fetus, is a polymorphic microorganism, a bacterium resembling a short curved comma or the shape of the Latin letter S. Less commonly, there are also spiral forms with 2-5 turns. Length 0.5-5, width 0.2-0.8 μm. Campylobacter is motile, has no spores and capsule. gram-negative (may be gram-positive in old cultures), good in all aniline stains will be painted. Under the microscope, these bacteria can be seen at one or both ends.

Campylobacter grows only in nutrient environments with reduced oxygen (10-20% of oxygen is replaced by carbon dioxide in a desiccator). From semi-liquid and solid media for growing them: 0.15-0.20% meat-peptone liver agar (semi-liquid), 2-3% meat-peptone liver agar (solid), vaseline fat-free Kitt-Tarotsi medium, Marten's

agar, etc. nutrients are used. Cattle, sheep, rabbit blood or horse blood serum is added to make the feed medium nutritious. No turbidity or gas formation is ever observed in the feed. Bacteria grow on solid media for 72-96 hours. It grows in 4 different forms: S (smooth), M (mucoid), ragged and smooth glass colonies.

There are 5 species of Campylobacter in Spirillaceae family, mainly one species is S. fetus subsp. fetus (V. fetus veneralis) is a causative agent of disease in cattle. This type of bacteria is an obligate parasite, passes through the genitals and induces abortion in cows, leaving them sterile. Campylobacter can be seen and isolated in cow vaginal mucus, semen, bull genital sac, placenta and fetal tissues. The causative agent also passes through the alimentary tract.

Campylobacter is also pathogenic for pigs, goats, chickens and humans, 7-15-day-old chicken embryos, guinea pigs, rabbits, guinea pigs and white mice. This property depends on the endotoxin released from it and the activity of mucinase enzyme in it.

Exciter durability. Campylobacter is not a very resistant bacterium to external environmental influences. 10-20 days in manure, soil, water and hay at 6-20°C; it lives actively in the stomach, liver, fetus, cotyledon for 20-50 days. It dies in 3 hours in the dried mass. It is stored in an unopened fetus at 20-25°C for 10-20 days. It can live only 3-4 days at a temperature above 25°C. Very sensitive to hot temperatures and antibiotics. Infection of animals with other microorganisms (esherichia, proteus, pyogenes, etc.) increases the viability of these bacteria. Campylobacter-ar survives in frozen tissue for up to 5-6 months. In liquid nitrogen (-196°C), the seed is kept active for a long time.

Epizootological data. In natural conditions, the disease occurs in cattle and sheep regardless of breed. Campylobacteriosis occurs at any time of the year, but is more common in the autumn and winter months during mating. The age of the animal it doesn't really matter. Cattle of both sexes are infected, and the causative agent is released in large quantities into the environment when the cow gives birth.

a lifetime , in the sac of the genital organ (preputium), in the testicles and in the seminal tract, and the semen ', it is released with the mucous fluid of the sac and the juice of the prostate gland and is transmitted to cows during sexual intercourse.

The bodies of cows and females infected with campylobacter are also very dangerous in spreading the disease, because for 3-10 months, they are in contact with the mucus, urine and milk, especially during childbirth, with the fetus, its water, placenta. releases the stimulant in large quantities. The pathogen is mainly transmitted from an infected bull or bull to a bull during the natural shedding of cows and females in heat. If the semen is obtained from a bull infected with campylobacteriosis, the cow and female carcasses can be transferred even if artificially inseminated. Even if seed contaminated with Campylobacter has been treated with antibiotics, it has been shown to transmit the pathogen to artificially bred cows. 40-90% of natural escapes and 30-70% of artificial escapes are infected with pathogens. Campylobacter has been found to be transmitted to the cow's vagina if it is on the bed. The importance of non-sterilized obstetric gloves in the transmission of the pathogen needs to be emphasized. Juvenile females, even calves, can be infected

with campylobacteriosis. This confirms that the pathogen is transferred to another organism by alimentary route, in addition to contact.

The source of the pathogen in sheep is a mother sheep that has given birth to a diseased fetus. They carry bacteria for 1-1.5 years. Sheep become infected when they drink feed and water contaminated with the pathogen. Rams from unhealthy flocks have campylobacter in their galls and livers, but transmission to ewes during lambing has not been proven. Also, the transmission of this pathogen from cattle to sheep and from sheep to cattle has not been proven.

Usually, the reason for the detection of the disease in a healthy flock is the introduction of a sheep carrying the bacteria into the flock without examination. Pigs, birds, dogs and foxes that eat the discarded fetus can serve as carriers and agents of transmission to other animals.

In a new, unhealthy furnace, cows are repeatedly calved, and the service period is prolonged. If the disease is acute, 20-55% of cows, 60-64% of female bodies remain barren. Abortion is observed in strait cows. This condition is usually observed for one season, then the process calms down. Animals develop immunity against this disease. In the herd, infection with this pathogen can be up to 0.5-2%, but the disease occurs sporadically.

Pathogenesis. If a female animal is artificially inseminated with a sick bull or infected seed, campylobacter appears in the genitals and uterus after 2-3 days. From this moment, the pathogen can be isolated from the uterus. After 10-15 days, the pathogen reaches the ovary. It is separated from the uterus by 3-6 months. As a result of the increase of the stimulus in this organ, inflammation begins, becomes evident on the 10-12th day and lasts for 4-5 months. In some cases, the causative agent can be isolated from the fallopian tube and bladder. The inflammation increases, and the inner part of the uterus reaches the tissue layers. As a result, the recovery of the uterus becomes very difficult, and metritis can last up to 8-9 months. Due to this, it becomes difficult for the egg and sperm to join in the inflamed uterus of the escaped female animal.

In some cases, the fertilized fetus does not develop well and dies in up to 50% of cases at the initial stage of miscarriage, as a result of which an abortion is observed. Because bacteria multiply in the stomach, liver, back and brain of the fetus, inflame them and cause toxicosis. After an abortion, a cow usually remains barren for 3-6 months. The runaway mole will be placed again and again. This is caused by the release of toxins as a result of the increase of the stimulus in the uterus and the violation of the pH-environment there. In bulls, the causative agent is located in the preputial cavity, urethra and prostate gland. In such cases, quality deterioration of the seed is manifested.

The causative agent in sheep is C. fetus subsp. intestinalis enters the bloodstream from the intestine after alimentary entry and settles in the causative uterus after a short period of bacteremia. In the case of strangulation, the pathogen passes to the fetus and kills it, resulting in an abortion.

Course and clinical signs. In sick cows, the main clinical sign is inflammation of the vagina, abortion (often in 4-7 months of gestation), retained placenta,

inflammation of the uterus. This situation leads to increased barrenness of cows and females on the farm. In some cases, due to campylobacteriosis in cows, in the 2nd month of goiter, the fetus may die without development and reabsorption. This condition is usually not noticed by farmers and experts. This can be known by the cow coming to the tune again after a long time. Due to this disease, the placenta is retained almost all the time in a cow that has given birth, inflammation is observed in the vagina and uterus. Some cows may give birth to a non-viable calf during the disease, but it will get sick in 2-4 days and die in 5-7 days. Usually, 6-15 days after cows are infected with campylobacter, the mucous membranes of the vagina redden and swell, rashes appear, thickened mucus flows from the vagina, fever is observed. They raise their tails and bend their backs, then pus is visible in the mucus. After 15-20 days, pea-sized hemorrhages are observed on the mucous membranes of the vagina.

In female animals, the disease can occur in two ways. In some cases, cattle are not fertilized. The cow or the body comes to the tune again and again, this situation continues for 5-6 months. Sometimes, estrus cows give birth in 4-5 months of estrus. It is necessary to run sick animals 4-7 times. The rhythm of the sexual cycle is disturbed, and the period of sexual peace (diestrus) lasts for 90 days. Embryo death is observed in 50% of cattle that have been burned several times.

Inflammation of the genital organ begins a week after infection. Later, the disease becomes chronic. Vomiting is one of the main clinical signs of the disease. According to most scientists, this condition is observed during the first four months of menopause. Animals that have given birth can then give birth normally, sometimes there is a recurrence.

Disease-specific changes are not clearly visible in male animals. Only the foreskin is slightly reddened, and mucus is released from it for 2-3 days. These symptoms disappear quickly, but remain a campylobacter carrier for life.

The main clinical sign of the disease in sheep is the observation of mass littering in the 2nd half of the strait. In some sheep, 3-5 days before abortion, there is a lack of appetite, lethargy, redness, swelling of the vagina and discharge of mucus from it. If the disease manifests itself during lambing, 10-70% of ewes may abort.

Pathologoanatomical changes. Adults do not die. Basically, the fetus thrown by the mole is dissected. The skin, subcutaneous tissue and muscles will be swollen. Hemorrhages are observed in the chest and abdominal cavity and parenchymal organs. It is noticeable that the blood vessels are filled with blood. In some cases, a reddish liquid accumulates in the chest and abdomen and contains fibrin. The fetus is sometimes embalmed, and necrosis is observed in the liver. Around the placenta, a yellow liquid mucous coating is visible.

Diagnosis. The diagnosis of campylobacteriosis is based on clinical symptoms, epizootological data and, of course, isolation of campylobacter as a result of laboratory examination. Observation of abortion in sheep and cows, repeated coming to the rut, infertility is the only reason to suspect this disease.

The fetus or its head, stomach, liver and lungs, placenta or its part are sent for laboratory bacteriological diagnosis. If this is not possible, mucus fluid from the

cervix after abortion, mucus from the bull's preputial sac, prostate gland juice or semen are sent for examination. Pathological material should be delivered to the laboratory very quickly, cold and on ice. Especially in the summer, it can break down quickly. Material is also obtained by the swab method, and this method gives good results in serological testing (AR, KBR, KUBR).

Differential diagnosis. In cattle, campylobacteriosis should be differentiated from brucellosis, trichomoniasis, and in sheep, salmonellosis and chlamydiosis. When the blood serum is tested with a special antigen (AR, KBR, KUBR), it is determined that there are antibodies against brucellosis. In brucellosis, in the first months of gestation, abortion does not occur, heifers abort. Brucella is isolated from the pathological material. In trichomonosis, trichomonad is secreted, and the child's discharge corresponds to 2-3 months of thrush. In leptospirosis, leptospira is isolated, yellowness, high temperature (42°C). Hemoglobulinuria and skin necrosis are prominent. In addition to vomiting, cases of nervous disorders are observed in listeriosis.

In sheep, it should be distinguished from salmonellosis and chlamydial abortion. Salmonellosis not only causes abortion, but also affects the gastrointestinal system, lungs and joints in lambs and rams. Abortion in chlamydia occurs 2-3 weeks before the onset of lambing, and the results of bacteriological and serological examination clarify all suspicions.

Treatment. Many drugs have been recommended for the treatment of campylobacteriosis. Since the trigger is located inside the foreskin, drugs are not always effective. Sick bulls are treated 2 times in 4 days with an interval of 5-6 days. In the first course, after treating the foreskin with antiseptic drugs, streptomycin and penicillin of 1 million units dissolved in 50-60 ml of vegetable or fish oil, and streptomycin and penicillin at the rate of 4 thousand units/kg twice a day in 0.5% novocaine are injected into the muscle . The 2nd course of treatment is carried out as follows. In the second course, oxytetracycline at the rate of 5,000 units/kg twice a day, and 5% furazolidone emulsion is injected into the foreskin. 1 month after the treatment, the testicles of the bull and the mucous membrane of the foreskin are bacteriologically examined 3 times with an interval of 10 days. If the test results are negative, the bull is considered cured.

Streptomycin in 0.5% novocaine is injected into the uterus of sick cows and heifers every day for 4 days at the rate of 4,000 units/kg. 1:5000 furatsilin or 1:1000 rivanol solutions are used to wash the skin. In the treatment of calves, 0.15 g of bigumal is given with milk 2 times on the first day, and 1 time during the next 4 days, and at the same time, 20% norsulfasol solution is injected intramuscularly for 3 days in a dose of 2 ml.

Streptomycin, penicillin, bicillin-3 and tetracycline are used to treat sick sheep. One of the above-mentioned antibiotics at the rate of 2 million units dissolved in sterile 20 ml of vegetable oil and streptomycin or oxytetracycline at the rate of 4 thousand b/kg twice a day for 4 days are injected into the uterus.

Immunity. Cattle have weak immunity. After the sheep recover from the disease, they almost never get sick. Therefore, a vaccine against campylobacteriosis

of sheep was created (MALuchko, 1974). It is used in unhealthy farms. Basically, in September, 1 ml is injected under the tail, immunity appears after 15 days and lasts up to 12 months.

Prevention and countermeasures. A new animal brought to a healthy farm or farm must be taken from a healthy farm that is being checked for campylobacteriosis, during a 30-day preventive quarantine period before entering the farm, bulls must be bacteriologically tested for this disease 3 times with an interval of 10 days, only healthy it is required to add cattle to the herd. This disease can be prevented by feeding all existing cattle on the farm with nutritious feed, monitoring the clinical condition, keeping the farm tidy, timely collection of every dropped fetus, regular disinfection, artificial escape of cow and female bodies. helps to get

If campylobacteriosis among cattle is detected in the farm, the farm is declared *unhealthy and a plan of health measures is developed.* The number and groups of animals, the number of sick people, and the number of those who gave birth are calculated. All calves in unhealthy farms are kept in isolation. Clinically ill cattle and sheep are treated as indicated above. Miscarriages are separated, fetuses, placentas, beds are burned, buildings and playgrounds are disinfected. 2% alkali, 2% chlorine lime solution, 5% creolin solution, etc. are recommended for disinfection in unhealthy farms.

Cow and female bodies are artificially removed and after 10-12 hours streptomycin and penicillin of 100,000 units dissolved in 10-20 ml of sterile physiological solution at body temperature are injected into the uterus to prevent disease. 250-300 ml of bigumal dissolved in water at a ratio of 1:1000 is given to cows and heifers every day for 5 days in order to prevent castration of cows. A. Golikov recommends administering bicillin-5 or dibiomycin in a dose of 25-30 thousand units/kg per animal. If no campylobacter has been isolated as a result of bacteriological examination within 12 months after the detection of the last diseased animal, and after all sanitation measures and final disinfection have been carried out, the farm is declared ***healthy*** **.** After that, the bulls are bacteriologically examined every quarter for 1 year.

If a disease is detected among sheep, all aborted animals are isolated and treated, and the flock is moved to another pasture. Pasture infected with the pathogen can be used after 1.5-2 months in the summer. In order to prevent abortion, chlortetracycline or 0.5 g of bigumal is administered to sheep at the rate of 5-8 mg/kg per head with soft feed every day for 10-12 days 1.5-2 months before giving birth. Drink every day for 3 days. Rams on a 4-day course: streptomycin and penicillin in 0.5% novocaine 4 thousand/kg are injected intramuscularly 2 times a day. If there is no abortion from this disease for 2 years, the herd is considered healthy.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .

5. Diagnosis and differential diagnosis.
6. Immunity .
7 . Treatment and prevention .

Topic: INFECTIOUS RHINOTRACHEIT DISEASE

Lecture	**Infectious Rhinotracheitis**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
Form of training	Enter. Visual lecture
Lecture plan About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Education	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Training conditions	Specially equipped lecture hall.
Monitoring and evaluation	Oral examination. Quick survey.

The lecture technological map		
Lecture stages	*Activity content*	
	Education giver	*Educator*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1. Listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It ends the lecture and students' attention is focused on the main issues, active students are encouraged. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Can be written.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.ne

Infectious rhinotracheitis (lat. - Rhinotracheitis infectiosa bovum; visual. - Infectious bovine rhinotracheites; Russian - infectious rhinotracheitis) is an acute infectious contagious disease characterized by fever, upper respiratory tract, eye, genital inflammation characterized by miscarriage and abortion and damage to the central nervous system.

Historical information. The disease was first reported in the United States in 1950 fattening and dairying are found in cattle farms and are called by different names. Acute upper respiratory tract infection, infectious rhinotracheitis, conjunctivitis. Its genital form is called infectious pustular vulvovaginitis. 1958 J. Gilespe et al. found that these diseases are caused by a single pathogen. Since 1960, it has been established that there are several forms of it, conjunctivitis, abortion, meningoencephalitis, fever, and respiratory syndromes in calves. Later, detailed information about infectious rhinotracheitis (IRT) appeared in a number of countries with well-developed cattle breeding (Canada, New Zealand, Australia, Hungary,

England, Germany, Italy, Bulgaria, etc.). Ru0Climari: NN Kryukov in 1970 Tambov, Yu. In 1971, Fomina, Z. Zudilina, and A. Maiboroda identified this disease among purebred cattle in several fattening farms in the Perm region, created methods for its clinical signs, pathologoanatomical changes, and serological methods of diagnosing it, and identified the causative agent. isolated and propagated the virus in cell culture and succeeded in preparing a vaccine against infectious rhinotracheitis.

In Uzbekistan, including Samarkand, Jizzakh, Syrdarya, Tashkent regions, IXSalimov, Z. Shopulatova, G. Uzakova identified this disease, and it was noted that it came mainly with bred cattle imported from European regions.

Trigger. Infectious rhinotracheitis virus (bovine herpesvirus BHV I) belongs to the genus and genus of DNA-storing herpesvirus, the diameter of the virion is 120-140 nm. It reproduces in cell culture of calf organs, showing SPT in cow fetuses. The virus is infected with bull semen or fetus kidney cell culture is the most susceptible. The tropism of the virus on the epithelial cells of the respiratory and genital mucous membranes of calves is strong. Therefore, in 5-6 days in animals infected with the virus, the titer of the virus is in the nasal mucus and in the vaginal mucus in the form of the genitals. will be very high.

Virus-neutralizing (VN), complement-binding (KB), special antibodies that form a precipitate and agglutinate are detected in the blood serum of an animal infected with the virus or recovered from the disease.

Exciter endurance. The virus is kept active for up to 9 months at low temperature (-60-70 ^{0} C) and weak alkaline environment (RN 6-9). At a temperature of 56 ^{0} C, it is deactivated in 20 minutes, at 37 ^{0} C in 4-10 days, at 22 ^{0} C in 50 days. The virus is very sensitive to ether and chloroform. Disinfectants (1-2% caustic soda, formalin, phenol) inactivate the virus in 10 minutes.

Epizootological data. Only cattle are susceptible to the disease, regardless of age and breed. It is more severe in cattle, especially meat breeds, which are fed more in the natural state. Pigs and sheep can be infected under experimental conditions.

The source of the disease is the sick and recovered virus carriers. The virus carrier period *is 6-19 months.* The virus is excreted through the nose, eyes, secretions from the genitals, milk, urine, feces and semen. When the disease is acute, the virus is released from the nose, genitals and eyes. From the nose from the first day to the 11th day, from the genitals from the 1st to the 6th day, from the eye 5 days after infection. The virus is mostly released from the nose (N. Kryukov, 1971).

Meat-breed animals with genital warts are very dangerous, and the virus is released from the genitals into the environment after 2-3 months. Illness when artificially infected, it was observed that the virus was isolated from the genital organ for up to a year (Straub, 1967). At the same time, the spread of hidden viruses by bulls is also *extremely dangerous* . In many cases, these factors are the source of rhinotracheitis. Air, feed, water, seeds, inventory, vehicles, birds, insects, and people serve as a virus transmission factor. In natural conditions, the virus enters the body mainly through contact (through the genitals during mating), inhalation (nose), eyes and mouth, through the mucous membranes of the digestive tract (alimentary) and transmissible

(insects). The disease begins 10-15 days after the contact of a healthy animal with a sick cattle or an animal carrying the virus.

The disease occurs more often in large livestock complexes, when buildings are filled with calves with different immunity, when they are crowded, hot or cold conditions, when they are fed with insufficient feed, in unfavorable microclimate conditions. The number of susceptible animals in the herd is important in the spread of the disease. Because the more susceptible animals there are, the easier it is for the virus to pass, its virulence increases, and it leads to the development of an epizootic process and a severe course of the disease. That is why IRT suffers when new cattle are brought to large fattening complexes and they are mixed with the cattle there.

In calves with reduced colostral immunity, the course of the disease is characterized by the appearance of non-specific symptoms of the organs of the respiratory and digestive systems. In healthy farms where the disease is recorded, the disease spreads quickly, within 2-3 weeks almost all cattle are infected. Then the disease is latent. The disease occurs in all seasons, but in winter, spring and autumn, it is noted more due to the influence of external environmental factors, violations of various rules and requirements for keeping and feeding cattle. Morbidity in IRT is 30-100%, mortality is 1-15%, if the disease is detected for the first time or is affected by secondary infections, the mortality can be even higher (on average 18%).

Pathogenesis. At the point of entry into the organism (respiratory tract, genitals), the virus enters the epithelium of the mucous membranes, multiplies and has a cytopathic effect on them, that is, it kills them and first small, then large necrotic foci appear. The pathological process passes from the nose to the trachea. Inflammation passes from the nose through the tear duct to the eye, forming conjunctivitis. The virus attaches to leukocytes and enters the blood, causing viremia. Fever and depression are observed in the body. If the virus crosses the placental or hematoencephalic barrier, inflammation is observed in the brain or uterus. Therefore, the death of the fetus and its exit (abortion) are observed in strait cows. Antibodies against this virus are detected in the blood serum of an aborted cow, and the virus can be isolated from the fetus or cotyledons. During the period of viremia, the virus can be isolated from the blood, but with the appearance of antibodies that neutralize the virus in the blood, this condition is not manifested.

The course of the disease depends more on conditionally pathogenic microorganisms and viruses. Secondary and mixed infections (PG-3, RS, adenovirus, streptococci, staphylococci, etc.) aggravate IRT disease. If secondary infection occurs, metritis, purulent metritis, bronchopneumonia are observed in the body.

Course, clinical signs and forms. The latent period is 2-4 days. It depends on the age of the animal, its resistance, the place of introduction of the pathogen, its quantity and its virulence. Depending on the route of damage, more respiratory or genital symptoms occur. The disease is more acute. In them, fever suddenly rises to 41-42 ^{0} C, depression, anorexia, rapid weight loss are observed, milk in cows decreases. In general, the disease takes several forms: *respiratory* (rhinotracheitis), *enteritis* , *genital* (vesicular rash), *conjunctival* and *meningoencephalitic* forms. It should be noted here that these forms do not appear separately, and often, depending

on the age of the sick animal, its resistance, the way the pathogen entered the body, and its virulence, from one to four of these forms are present in one animal. can meet

In calves , during *the respiratory* form, a serous-mucous, later serous-fibrinous, sometimes bloody fluid is released from the nose, and they have general depression, lethargy, anorexia, fever up to 41.5 - 42.1 ^{0} C. Nasal glass reddens. It is called *a red nose* . The nose, throat, larynx will be swollen. Because they breathe quickly, they breathe through their mouths. The nasal cavity and the mucous membranes of the eyes are strongly inflamed. After 1-2 days, mucous fluid flows from the nose. Later, secondary infection is added to it and pus mixes. They have a lot of drooling and coughing. In some cases, foci of necrosis and fibrin coatings appear on the mucous membrane of the nose. If these coverings are scraped off, a wound will be visible beneath them. Such coatings are also seen in the upper respiratory tract. Later, they thicken and constrict the airways. As a result, it becomes difficult to breathe. When the secondary microflora falls, the course of the disease is prolonged and the animal dies. Conjunctivitis, arthritis, lameness are observed in some animals. Calves have inflammation in the digestive system, diarrhea. A foamy liquid flows from the mouth of a sick calf. Body temperature is maintained at a high level for 3-4 or more days. If a secondary infection is added, pneumonia and a rise in temperature are observed. As a result, the calf dies (IXSalimov, 1994). Mortality in the respiratory form is up to 10%.

Abortion is observed in heifers and cows at 6-8 months of gestation. They are usually infected with the respiratory form 3-4 weeks before the abortion. Abortion is observed within 10 days after the death of the fetus. This happens 9-105 days after the animal is infected with the virus. Metritis is observed after abortion, and milk is sharply reduced.

Enteric form of the disease in calves with severe diarrhea. Faeces are liquid, in some cases with mucus, smelly when used. This form is always observed to go along with the respiratory form.

Conjunctival in the form, the mucous membranes of the eyes are reddened, before them, serous mucus, and then pus mixed tears flow. The body temperature of calves rises to 41.2 - 42.1 ^{0} C. A white coating (white) develops on the corneas and completely covers the eye, and the calf's eyes protrude from their sockets and become squinted, resulting in blindness in one eye, sometimes in both eyes. remains.

Genital form occurs when the virus enters through the genitals . In this case, pustular vulvovaginitis and bolonoposthitis are observed in bulls. 2-4 days after being infected with the virus, depression, anorexia, decreased milk production, reddening and swelling of the mucous membranes of the vagina are observed in animals. Then blisters filled with fluid appear on the mucous membranes. Later, the pustules coalesce and form a slurry mass mixed with pus and blood, and the presence of ulcers is noticeable at the base. Mucous purulent exudate accumulates in the penis. Vaginal mucosa and vulva swell. The animal becomes restless and urinates frequently, and this situation is repeated. A similar change is found in the genital organ of bulls and its sac. Thus, the disease lasts from 3 - 8 weeks to several months. A sick animal can recover. Abortion is observed 4-5 weeks after infection. The virus infects the fetus

and kills it, and abortion occurs during the 6-8 month period of straitjacket. In bulls, the genital sac is inflamed. If there is no secondary infection, the animal will recover from the disease in 10-14 days. It should be noted here that IRT in cows is more often pustular vulvovaginitis, and in heifers, abortion is observed.

The form of meningoencephalitis in most cases occurs in calves aged 4-6 months. In calves, signs of encephalitis, ataxia are observed - circling movement, muscle twitching, frothy saliva flowing from the mouth. The condition of sick calves worsens, they lose their appetite, their body temperature rises to 40.5-41.5 ^{0} C, they stumble and turn around when walking, fall and start kicking their hind legs, throw their head back or to the side. Such calves die within 5 days. If they become comatose, they lie down with their legs stretched out and die within 1-2 days (IXSalimov, 1994).

This disease is more latent in cattle and older bodies, and they serve as carriers and spreaders of the virus in 50-60% of cases. On average, antibodies against this virus were detected in 56.9% of cows (IX Salimov, 1994). The duration of the disease is not the same in the farm. In one case, 80-90% of animals become infected within 2-3 weeks, in the second case, only some groups of animals become infected within a few weeks.

Pathologoanatomical changes. Pathologoanatomic changes depend on the form of this disease. If it passes in respiratory form, the presence of mucous purulent and fibrinous exudate is visible in the mucous membranes of the nose, larynx, and larynx of the dead body. It is determined that the nasal cavity is blocked due to the solidification of the mucous-purulent mass flowing from the nose. Mucous membranes are swollen, areas of necrosis, wounds and hemorrhages are observed. Regional lymph nodes are reddened, filled with blood, in some cases hemorrhages are observed. Catarrhal, purulent catarrhal pneumonia is detected in the case of aggravated disease. Conjunctivitis and keratitis are visible in the eyes. Spleen is slightly enlarged, with hyperplasia and hemorrhages under the skin. Hemorrhage is also observed in the epicardium and endocardium. The cerebral cortex is filled with blood vessels. When the disease subsides, pathologoanatomical changes are mainly limited to acute inflammation of the mucous membranes of the respiratory organs.

In the case of genital infection, there are ulcers on the mucous membranes of the genitals, and when it is aggravated, endometritis is observed. The aborted fetus is swollen, there are foci of necrosis in its liver, tissues around the kidney are covered with hemorrhagic exudate.

Diagnosis. The diagnosis of IRT disease is based on clinical signs, pathologoanatomical changes, epizootological data and, of course, the results of laboratory examination.

For virological examination, mucus is taken from the nostrils, eyes, and vagina of sick cattle with a swab. Pieces of the nasal wall, larynx, lungs, liver, spleen, brain, submandibular, mediastinal lymph nodes, placenta and parenchymal organs of the fetus are sent from the dead animals within 2 hours. In summer, it is better to send in 40% glycerine, with ice. Definitive diagnosis requires pure isolation of the virus in

cell culture from a sample taken from a sick animal and its identification by virus neutralization reaction (VN).

For retrospective diagnosis, blood serum is sent at the beginning of the disease and after 2-3 weeks. An increase of at least 4 times the antibody titer in the serum of IDR, NR and BGAR reactions in the 2nd examination is the basis for making a diagnosis of IRT. Enzyme immunoassay (IFT) and the simplest, easy to apply and high specificity IDR are also used for virus antigen detection.

Microscopic observation of Cowdry's inclusions in injured tissues of sick animals is the main sign in diagnosing IRT (Z. Zudulina, 1970).

Differential diagnosis. IRT should be differentiated from catarrhal fever of poor quality, parainfluenza-3, viral diarrhea, adenovirus and chlamydial infections. Differentiation from these infections is carried out on the basis of laboratory tests, because they can also be accompanied by IRT.

Treatment. Sick animals are isolated in a warm, dry room and fed with nutritious food. For special treatment, the blood serum of a convalescent animal (titre higher than 1:32) is injected subcutaneously or between the muscles in 2-3 places at a dose of 2 ml/kg. To prevent secondary infection, antibiotic and sulfonamide drugs and general strengthening and symptomatic drugs are used. Nitrofuran, sulfanilamide preparations and antibiotic ointments are used in the genital form.

IXSalimov (1994) divided 64 calves into 4 groups of 16 for the treatment of pneumo-enteritis (IRT, VD, PG-Z, bacterial mixed infections), the first group of calves was treated with 1% Baytril (5 ml orally), the 2nd group 1% when treated with iodinol (40 ml orally and 40 ml intravenously), 3rd group calves with furozolidon (200 ml orally) and 4th group calves with antibiotics (ampicillin 500 thousand/B and streptomycin 1 million/B) Li wrote that Baytril, Iodinol and Ethonium suspension were 100% effective with furozolidon, and the effectiveness of antibiotics was 81.25%. Calves in the first group recovered in 4-5 days, calves in the second group in 5-6 days, and calves in the third group in 5-7 days. During this period, 3 calves from the fourth group died. He informed that the rest were cured by continuing treatment.

Immunity. Animals that have recovered from the disease develop active immunity. They form VN, KB and precipitating antibodies in blood serum, but they do not protect animals from the disease. Animals in the respiratory form have stronger immunity than in the genital form. Secretory immunoglobulin A and interferon protect against IRT disease.

In areas with IRT, colostral antibodies are present in calves for 2-5 months, making it difficult for them to develop active immunity.

For active immunity, there is a live dry vaccine made from the TK-A VIEV strain. It is administered subcutaneously to large animals 1 time 2 ml, to young calves 2 times with an interval of 14-20 days, the first time 1 ml and the second time 2 ml. Immunity appears after a week and lasts for a year. It is recommended to use bivak dry-associated culture vaccine (against IRT and parainfluenza-3 diseases), it protects against two diseases. Calves are vaccinated with it twice before the age of 3 months. For the first time, 1 ml is injected into both nostrils. The second time, after 14 days, 2 ml is injected under the skin. After 3 months, 1 ml is injected into the nostrils, and

after 14 days, 3 ml is injected under the skin. Immunity appears after 2 weeks and lasts for 6 months.

Prevention. Prevention of respiratory diseases is based on veterinary-sanitary and zootechnical activities. Organization of storage of livestock in summer pastures, feeding with nutritious feed, housing animals in the building according to zoohygienic standards, protection of calves from poisonous gases, timely cleaning of the building and pastures from manure, disinfection in them according to a regular schedule , carrying out disinsection measures, not allowing other animals and strangers to the farm, helps to prevent disease. Cattle should be brought only from healthy farms for this disease. Stables are filled with cattle of the same age, and strict adherence to the principle of "busy" and "free" is always required. "Closed" farming should remain the main rule. These measures allow to protect the farm from diseases, to increase the resistance of the organism and to neutralize external environmental agents.

If this disease has been detected in the farm before, it is necessary to introduce only vaccinated animals to the farm. Cattle brought to the farm will be under preventive control in another place for 1 month. Only healthy and IRT-vaccinated cattle should be introduced to the farm.

Preventive measures against IRT depend on the direction of livestock farming: in the direction of dairy farming - bulls should be checked for IRT during the quarantine period. It is not possible to run free with a bull. It is necessary to establish an artificial escape. Vaccines are used for special prevention, and hyperimmune and convalescent blood sera are used in the same amount as for treatment.

Countermeasures. If this disease is detected among cattle in a farm or farm by clinical, pathomorphological and serological methods, the farm is declared unhealthy and *restricted under the Veterinary Regulation* . In the unsanitary point, all containment measures are strictly followed and measures are taken to prevent the spread of the disease. According to the restriction requirements, animals, their products, strangers, and vehicles are prohibited from entering and leaving the farm, mixing goods on the farm, and transferring them from one place to another farm or barn. Cattle are examined by clinical, serological, pathomorphological methods. It is forbidden to remove equipment, implements and hay from the farm. Special caretakers are appointed to take care of sick animals. All cattle are clinically examined, thermometry is conducted, sick and suspected cattle are separated and treated, and healthy ones are subject to compulsory vaccination. The current disinfection is carried out until the restriction is removed. Patients are recommended hyperimmune blood serum, convalescent serum and immunoglobulins. *The restriction* from the farm is taken 30 days after the end of the disease, after the final disinfection. The release of animals for breeding purposes and the use of bull semen from this farm is allowed 2 months after receiving the restriction.

Bringing cattle to breeding stations and breeding farms is due to epizootic healthy farming. They are in preventive quarantine for 60 days. Bulls are checked every month. Those found to be sick are slaughtered.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7 . Treatment and prevention.

SUBJECT: Bradzot's disease

Lecture	**B radzot disease of sheep**
Study hours: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*

1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He hears, clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.ne

Bradzot is an infectious disease characterized by hemorrhagic inflammation of the jejunum and duodenum, tissue changes in the parenchymatous organs, and gas accumulation in the gastrointestinal tract.

Spread. Bradzot is found in many countries (Australia, Greece, Turkey, Germany, etc.). Based on the information of K. Lamikhova, M. Farzaliyev, A. Volkova, there is a problem. In particular, it is often noted in our Uzbekistan.

Economic damage. Sheep infected with the disease die, and a lot of money is spent on preventive measures.

Trigger. Most Sl. sepficum and Sl. Oedemmafiens is isolated. In many cases Sl. Also found in Didas. But this microorganism often increases the effectiveness of both previous microbes. Sl. septicum is strictly anaerobic, growing at 37°C. Grows in Kitt-Tarossi medium and becomes turbid and gasses in 16-24 hours, after 48 hours the broth becomes stagnant and it settles. Spores are rarely found in the body, they are often found in carcasses. Grows in Seissler's medium, showing the zone of hemolysis. The microbe releases gas and ferments in the following environments (glucose, maltose, fructose, etc.). Does not decompose mannitol with glycerin. It rarely breaks down sucrose. It is from this feature that Sl. septicum and Sl. used in distinction from chavoei. Because the latter always ferments sucrose.

Sl. septicum secretes a very strong toxin, which is especially evident in Marten's broth. The toxin has four different components:

- Alpha toxin causes necrotic and hemolytic factor (hemolysis of sheep erythrocytes).
- Beta toxin-oxidative enzyme-deoxyribonuclease, causes rapid hemolysis.
- Gamma toxin-enzyme hyaluronidase.
- Delta toxin is oxygen-soluble and lyses erythrocytes.

Sl. septicum contains O and N antigens, which in turn produce agglutinin, precipitin, and hemagglutinins.

Sl. oedemmatiens is a bacilli polymorphic anaerobe. When growing in harsh environments, it forms a grainy surface and has uneven edges. Grows in a Kitt-Tarossi medium, forming a gas. Highly pathogenic for laboratory animals.

Cl. In addition to didas bradzot, it causes infectious necrotizing hepatitis. Gangrene can occur in humans.

Endurance. In sporadic cases, the microbe is highly resistant. It can live in the soil. It dies in 50-60 minutes when boiled.

Epizootology. In natural conditions, sheep can get sick regardless of breed and gender. In most cases, children older than 2 years are affected. Bradzot occurs at any time of the year. According to our observation, in the spring, in the pasture, the bushes are ripe, in the summer months, and in the fall. In Scandinavian countries, there is information that it started in September and lasted until March.

Pathogenesis. The resistance of the sheep's organism is very important in the mechanism of disease development. As a result of the effect of various factors (cold, heat, heating) on the body, the resistance decreases, the secretory and motility of the stomach and intestines are disturbed. As a result, favorable conditions are created for the pathogen to live and develop. It, in turn, releases a very strong poison and poisons the macroorganism.

In general, the study of the pathogenesis of bradzot requires more research.

Clinical signs. Bradzot is lightning fast and sharp. A healthy sheep suddenly dies at night when it moves at lightning speed. Sometimes one or two sheep fall behind the flock. He shivers for 10-15 minutes, his eyes turn red, his stomach swells and he dies. Before death, foamy liquid flows from his mouth. Body temperature may rise slightly. Even when it is acute, the body temperature rises a little, he becomes weak, he does not eat anything. He breathes and the cardiovascular activity accelerates. A frothy mucous fluid flows from the nose and mouth. Sometimes there is bloody diarrhea, frequent urination, the stomach is swollen, and the sick sheep chatters its teeth. In some cases, he gets restless, taps his leg, spins in one place, moves his legs as if he is swimming while lying down. It trembles from time to time. Usually restlessness alternates with general weakness. A sick sheep dies within 10-14 hours due to general weakness and severe panting. If the disease is prolonged, it can last 3-5 days.

Pathologoanotomic changes . The carcass of a sheep that dies of Bradzot becomes very swollen within 2-5 hours. The swelling is so strong that in some cases the skin may even crack, and the color will turn blue. It grows very quickly. A putrid

smell emanates from the corpse, and a reddish liquid flows from the natural pores. Air-mixed serous hemorrhagic infiltrate accumulates in the head, neck, chest and other places. The wool is easily plucked. The mucous membranes of the mouth, nose and eyes become blue. Peripheral blood vessels do not clot. Dotted bleeding is observed in the trachea and bronchi. Red fluid accumulates in the chest and abdomen. The lungs are swollen and puffy. Hemorrhage is prominent in the epicardium and endocardium. The anterior compartments of the stomach are full. Mucous membranes of the ileum and duodenum are swollen, severe bleeding is observed. The liver is cystic, and there are foci of necrosis.

Diagnosis. When making a diagnosis of Bradzot, its epizootology (sheep are infected, gender and age are not important, epizootic can occur in any season), clinical symptoms (severe, acute, in some cases restlessness, bloody diarrhea, tremors are observed), patho-anatomical changes (the spleen swells quickly, the skin may crack, putrid smell), the results of laboratory examination methods are taken into account. Parenchymatous organs (necrotic area of the liver), injured area of the liver, swollen tissues, duodenum, etc. are sent to the laboratory.

Bacteriological examination . Kitt-Tarossi medium from the sent samples, planted in MPBs and MPAs. Planted test tubes are placed in a thermostat with a temperature of 37-38°. For an anaerobic environment, a microaerostat or a desiccator is used. Sl. septicum grows rapidly and releases gas. Sl. Oedemmatiens grows slightly turbid and rarely produces gas. In a solid environment, the first forms a zone of hemolysis, later the center grows darker, the surface is rough, and the edges appear cut.

Biological method. An infusion is made from the injured organ in MPB, the mixture is injected into 2 guinea pigs at 0.5-1.0. If there is bradzot, the guinea pigs will die in 16-48 hours. They have septicemia. There is air in the intestines. Red fluid accumulates in the chest and abdomen. Observation is carried out for 8 days and all members are examined. If 1 guinea pig dies, histopathology is confirmed, and a culture is obtained, the diagnosis is confirmed.

Differential diagnosis. Bradzot should be distinguished from anthrax, infectious enterotoxemia, pasteurellosis, emkar, pyraplasmosis and aconite poisoning.

- In anthrax, the spleen enlarges and when cut, a black oily mass comes out. All kinds of animals get sick, and only in the summer months. An anthrax bacillus is found on a blood smear taken from the ear. In Karason, FI Kagan and LV Kirillov (1976) recommend infecting rabbits with the disease. The rabbit is resistant to emcar, susceptible to bradzot. Emkar's causative agent ferments sucrose, Sl. septicum ferments salicin.

- Kidney softens in infectious enterotoxemia. In Bradzot, the jejunum and duodenum are severely damaged. The problem is mainly solved by the conclusion of the bacteriological method.

- Paterellosis affects internal and respiratory organs. Of course, it is necessary to carry out a bacteriological examination.

- In case of aconite poisoning, poison grass in the pasture is checked.

- In piroplasmosis, a parasite is found in a blood smear.

Treatment. Due to its severe and rapid course, bradzot treatment is not beneficial. If the disease is protracted, antibiotics biomycin, synthomycin, terramycin 0.5-1.0 per 1 kg of weight for large sheep, 0.2 for lambs, 0.5-0.75 per head per day with biovetin feed is given. In this case, the main means of treatment are polyvalent hyperimmune blood serum and anatoxins, which are used according to the instructions.

Immunity. The vaccine was first developed and put into use by academician AA Volkova. F. Kagan, A. Kolesovlar created a concentrated polyvalent aluminum hydroxide vaccine. It is used against bradzot, enterotoxemia, severe swelling and dysentery of lambs. To prevent the disease, lambs are vaccinated from 3 months of age.

Prevention. It is necessary to raise the sanitary conditions of pastures and water sources to the level of veterinary-sanitary requirements. Any place where bradzot is released is under strict control. According to the plan, sheep are vaccinated, taking into account the epizootic situation. Restrictions are announced in the places where the disease occurs. It is strictly forbidden to bring and sell sheep to the farm. Shearing is stopped and sheep driving is not allowed. Mowing from unhealthy pastures is stopped. From which flock a bradzot emerges, its place or pasture is immediately changed. Sick sheep are separated and healthy ones are vaccinated. Healthy sheep are returned from the pasture and transferred to confinement. Coarse hay and minerals are added to the diet immediately . Sheep sheds where sick sheep are kept are disinfected with 3% active chlorine solution of chlorinated lime , 5% formaldehyde or alkalis, 5% formaldehyde , 10% chlorine (1)-iodide . Slaughtering sick sheep for meat, removing skin from carcasses, shearing wool, sucking milk and using it for consumption is strictly prohibited . Carcasses are collected only in specially adapted transport vehicles , the place is disinfected . The carcasses of the affected sheep, wastes , carcasses are burned without removing the skin . Autopsy is possible only for diagnosis and is carried out in specially designated areas. The ban will be lifted two weeks after the last sick leave .

Control questions

1. Pathogenicity and toxicity .
2.Pathogenesis, clinical symptoms .
3.Pathologoanatomical changes .

Topic: INFECTIOUS RHINOTRACHEIT DISEASE OF HERBS

Lecture	**Infectious rhinotracheitis disease of horses**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90

The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.

2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.
3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.
4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.ne

Rhinopneumonia (lat. - Rhinopneumoniae equorum; visual. - Equine virus abortion; Russian - viral abortion kobil) is an acute contagious infectious disease characterized by fever, conjunctivitis, catarrhal inflammation of the mucous membranes of the respiratory tract and is characterized by abortion in the last months of estrus.

Historical information. Until the middle of the 19th century, all fever and diseases of the respiratory organs of horses were called influenza. In the 30s-50s of the 20th century, infectious diseases of horses were distinguished from each other, and they were called separately: rhinopneumonia, influenza, viral catarrh of the upper respiratory tract, contagious pleuropneumonia and influenza. In 1933, US scientists Dimock and Edward Ct observed mass abortions in bees in addition to disease of the respiratory organs, and in 1936 they proved that this disease has a viral etiology. In 1941, Manninger and Chantosh experimentally induced upper respiratory tract injury and abortion in strait bees in Hungary. This disease was recorded in 1951 in France, in 1954 in Italy, Poland, and Yugoslavia. Later in 1957, Doll et al. proved that the isolated virus is the causative agent of rhinopneumonia.

Rhinopneumonia was registered in 1954 in Tunisia, in 1963 in Canada, in 1966 in Japan. The disease occurs in almost all countries. This disease is widespread among horses in many European countries, South Asia, Africa and both subcontinents of America. Serological tests have shown that the majority of horses in the world have antibodies against this disease.

Trigger. It is a DNA-storage virus belonging to the 1st generation of Herpesviruses and the Herpesoviridae family. The diameter of the virion is 100-150

nm. There are 2 subspecies of this virus. The subtype 1 strain was isolated mainly from North and South American horses, and the subtype 2 strain was isolated from horses of the European and Asian continents. Hemagglutination of erythrocytes of viruses, endothelio- and epitheliotropic. Hemagglutinating, precipitating, complement-binding and virus-neutralizing antibodies are detected in the blood serum of a horse that has recovered from the disease. The virus develops in a cellular environment, in chicken embryos and in laboratory animals.

Exciter durability. The virus is resistant to external influences. Infected organs are kept active for 457 days at -18 ^{0} C. In a dark room at 20-27 ^{0} C, the virus does not lose its activity for 14 days, in horse hair for 42 days. In the tissues of the aborted fetus, the virus is inactivated in 6-7 days at 4 ^{0} C, but in 10-20 minutes at 55-56 ^{0} C. Ether, chloroform, 0.5% formaldehyde kills the virus in 10-15 minutes. It is resistant to acidic environment.

Epizootological data. In natural conditions, horses, mules and donkeys are prone to rhinopneumonia regardless of breed, age and sex. Purebred horses, mares under one year old are especially susceptible to this disease. Cattle, sheep, goats, pigs and humans are not infected at all. Laboratory animals are prone to guinea pigs and porcupines.

The origin and spread of the disease is made possible by uncontrolled transfer of horses from one farm and stable to another, not feeding them with nutritious feed, not keeping them in accordance with zoohygienic requirements, and breeding with close relatives.

The source of the pathogen is a sick animal, which secretes the virus in large quantities through its nasal fluids, genitals, and the discarded fetus and its fluids. When the disease is in the respiratory form, the virus contaminates objects of the external environment with coughing, when the horse coughs, sneezes, and infects healthy horses through airborne droplets and contact. In addition, the virus is transmitted through food. The virus isolated from a sick horse also contaminates water, bedding, and stable equipment and serves as a carrier of the pathogen through which the pathogen is transmitted. Throat beetles excrete the virus in their urine before abortion. The lice, in which the disease is *latent* , serve as *a dangerous virus carrier* for a long time . Stallions also act as carriers of the virus and transmit the pathogen to mares during natural mating. This condition can *last* for months or even years in stallions .

Diseases usually come to healthy farms and herds with virus-carrying or diseased horses imported from elsewhere. The transmission of the virus to the herd of healthy horses, especially thoroughbred mares, causes the disease to spread widely. Diseased bees serve as a source of transmission of the virus for a long time. The disease occurs in the form of enzootic disease, usually 40-60%, in some cases up to 90% of gypsy moths abort. Abortion increases when young ewes are kept with a flock of sick ewes. Respiratory rhinopneumonia is more common in winter and autumn.

Pathogenesis. The virus enters the body through the intestines or respiratory organs, settles in the mucous membranes of the respiratory tract and throat, where it

multiplies and causes catarrhal inflammation. From these places, the virus enters the blood through the lymph and spreads to all organs, and *viremia* occurs. The virus, together with leukocytes, easily passes through the uterine and placental barriers and enters the fetus. When the virus enters the fetus, it multiplies in all its organs and tissues without any resistance and leads to the development of sepsis in the fetus. As a result, degenerative and necrotic processes occur in all organs of the fetus, so hemorrhages are observed in all its organs (capillaries, brain), including parenchymal organs (liver, spleen), and as a result, the fetus dies. This ends in abortion or stillbirth.

During histological examination, *viral inclusions* appear in the nuclei of necrotic tissue cells in the endothelium of the liver, biliary tract, bronchi, and endothelium of blood vessels.

Course, clinical signs and forms . The latent period of the disease is 2-10 days. Depending on the manifestation in the organs of the body, 4 forms are distinguished: *respiratory, abortion, genital* and *nervous forms.* In horses that are infected for the first time, the disease is *acute* , and then it can turn into a *chronic* form. The onset of the disease is manifested in *the respiratory form - catarrhal inflammation of the upper respiratory tract and conjunctiva. A fever of 40* ^{0} C and above is observed for 2-3 days at a time . Usually, the disease passes in a relatively mild and *abortive form* . Abortion is observed 100-150 days after being infected with the virus. Abortion occurs more often in 8-11 months of gestation, less frequently in 5-7 months. Abortion occurs *suddenly , the fetus falls wrapped in a veil.* Usually, placental abruption and other postpartum complications are not observed. In some cases, a bee that aborts *in the form of a nerve can die as a result of paralysis.* In other forms of the disease, the uterus quickly returns to its normal state after an abortion, and after 1 month, the uterus may return to normal. Sometimes, if the ewes are infected with the virus in the last stage of estrus, the ewes will be born alive, but it will become incapacitated and die within 1-3 days.

Horses that are not fed with nutritious feed, lacking minerals and vitamins, get sick, and the disease is severe in them. The mucous membranes of their upper respiratory tract are reddened, the conjunctiva of the eyes turns yellow, the mucous membranes of the nose, throat, and larynx are inflamed and swollen. Head and neck regional lymph nodes are enlarged. Swelling of the throat and larynx makes it difficult to breathe, wheezing and wheezing. Serous-mucous fluid from the nose becomes purulent due to secondary infection. In some cases, pneumonia, inflammation of the mucous membranes of the gastrointestinal tract are observed.

If the disease is very severe, the central nervous system and the heart function will fail. Serous-fibrinous inflammation in joints, orchitis in stallions can be detected. Leukopenia is usually detected in rhinopneumonia, and leukocytosis when aggravated by secondary infection. Horses usually recover in 1-3 weeks unless complications occur. Biyas can also have *a genital form* . In this case, the mucous membrane of the vagina turns red, rashes appear, which turn into white spots.

Pathologoanatomical changes. Symptoms characteristic *of septicemia* in this disease : mucous membranes of the upper respiratory tract, redness, swelling, inflammation of the conjunctiva, degenerative changes in the parenchymatous organs

- kidneys, liver, spleen, heart, lymph nodes and their edema, mucus and hemorrhages are noticeable in the serous membranes. Small rashes appear on the mucous membranes of the nose and throat, and in some cases, the larynx. Shiny amber-yellow or red-yellow liquid collects in serous spaces. 50 ml of fluid accumulates in the pericardium, 1-2 liters in the chest cavity. Under the epicardium, endocardium and splenic capsule, spotty, striated hemorrhage is observed in the renal capsule. The lungs are swollen, there will be hemorrhagic spots.

In the mucous membranes of the stomach and small intestine, ilviral layers are visible. Peyer's rash and solitary follicle in the small intestine have erosions and deep ulcers. In addition to hyperemia, swelling in the lungs, catarrhal inflammation and peripneumonia are observed in rare cases. The liver is swollen and darkens. Dotted hemorrhages and necrosis are observed in the mucous membranes of the stomach and intestines. When 7-11-month-old gilts abort, *the fetus does not undergo autolysis* . When the histological sample prepared from the liver, spleen and lungs of the fetus during an abortion is examined, *the inclusions of Caudrivirus* in the nucleus of the cells are visible under the microscope, which is of great diagnostic value.

Diagnosis. The final diagnosis of the disease is made on the basis of clinical signs (abortion), epizootological data, pathologoanatomical changes and, of course, virological and serological tests. The observation of abortion among strait bees may be a reason to suspect rhinopneumonia. The final diagnosis is made only on the basis of *virological*, *serological* and *histological examination* of pathological paterials taken from sick horses and aborted fetuses . In the laboratory, the nasal lavage of the sick horse, the material scraped from the nasal mucosa, lung, liver, spleen tissues, the discarded fetus, the stomach, liver, lung, spleen, brain, heart and kidney were cut into the laboratory. the received fragments are sent. The virus is isolated from the pathological material sent in the laboratory and it is identified. NR, GATR and KBR reactions are performed with blood serum of sick horses, and antibodies formed against rhinopneumonia virus are determined. The immunofluorescent reaction is of great importance in the diagnosis of the disease and in the identification of the virus.

Differential diagnosis . This disease should be distinguished from influenza, abortion with salmonellosis, food poisoning. Flu symptoms are: fever, worsening of the patient's condition, conjunctivitis, catarrhal inflammation of the upper respiratory tract. Clinical symptoms of rhinopneumonia are almost invisible. Abortion with salmonellosis occurs at 4-8 months, and with rhinopneumonia, abortion occurs suddenly at 7-11 months, and it is born wrapped in membranes. Spots of necrosis, hyperemia are observed in the placenta. In abortion with salmonellosis, the placenta is retained and metritis develops as a complication. There are no complications after an abortion in rhinopneumonia. During bacteriological examination, salmonellosis grows in nutrient media in abortion with salmonellosis. Food poisoning is determined by examining feed samples in the laboratory.

Treatment . A special treatment method for the disease has not been developed. If the disease becomes complicated, it is treated symptomatically. Antibiotics and sulfanilamide drugs are used to prevent secondary infection.

Immunity. Cows that have recovered from the disease have short-term immunity, so within 1 year they may have a recurrence of the disease. Colostral immunity lasts only 2-3 months. Infected older sows also have a very short immune system and may experience abortions once they are engorged. However, in aborted bitches, the immune system is long-lasting, and when they are spayed, they give birth to healthy pups. Therefore, compared to the respiratory form, *immunity* lasts longer in the abortive form of the disease. To date, it has not been determined which high titer of antibodies in sick horses is a sign of recovery from the disease.

Prevention and countermeasures. Horses purchased for breeding or sports should be taken from countries, farms and farms that are healthy for infectious diseases, checked for mange, rhinopneumonia, influenza and other infectious diseases before arrival, and 1 month preventive after arrival. During the quarantine period, it is necessary to carry out clinical and laboratory tests again. Horses are also not taken from a farm where an abortion has been observed in the last 2 months. In order to ensure the resistance of horses, it is necessary to feed them with nutritious food, rich in vitamins and minerals, and keep them according to zoohygienic requirements.

If rhinopneumonia is detected clinically, pathologically, anatomically, or virologically, the farm, farm or settlement is declared unhealthy and *restricted under the Veterinary Regulations* . In the unsanitary point, measures are taken to carry out all health measures and prevent the spread of the disease. Entry and exit of new horses to the farm, mixing of horses with other groups is prohibited.

After the disease is detected on the farm, the horses at the point are kept without being released to pasture and are clinically examined. Sick animals are separated and treated symptomatically, the remaining healthy horses are left in pens and vaccinated according to the Instructions for the use of the vaccine. 1-3-month-old fawns are vaccinated, and revaccination is required after 6-7 months. Young mares and stallions should be revaccinated 3-4 months after the first vaccination. Stables are disinfected weekly with 3% chlorinated lime, 3-5% caustic sodium, 10% formaldehyde, monochlorinated iodine, and biothermal disinfection of manure for 3 months.

The restriction from the unhealthy point is taken 2 months after the last patient was treated, after the final disinfection, if there are no pus passing through the 2nd half of the strait.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention

Topic: MUMITY OF HORSES

Lecture	Dumb disease of horses
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.ne

Mute (Lat. - Adenitis equorum; English - Strangles Adenitis equorum; Russian - mit) is an acute contagious infectious disease of horses, characterized by fever, purulent-catarrhal inflammation of the mucous membranes of the nasal cavity and throat. , is characterized by inflammation of submandibular lymph nodes turning into an abscess.

Historical information . For the first time, the information about this disease was recorded by researcher Zolleizel in France in 1664. By the 19th century, researchers Viborg and Erdely experimentally proved that the disease of muteness is infectious. Schütz, Iensen, Zand in 1888 proved that the causative agent of muteness is streptococcus and isolated it from a sick horse.

Later, a group of researchers suggested that the causative agent of muteness is a virus, and streptococcus is a secondary infection. However, I. Ya. Sadovsky and others. (1930) proved the causative agent of muteness to be streptococcal, disproving the doctrine of viral etiology. This disease occurs among horses in almost all

countries of the world. In the middle of the 20th century, 80.8% of horse diseases in the regions of MHD were dumb.

The causative agent is Streptococcus equi (silent streptococcus) - a non-moving, non-spore-forming microorganism of round or oval shape. In a smear made of pus, the cocci appear in the form of long thin curved threads. In smears made from cultures grown in nutrient media, the threads and chains formed by them are shorter. In sick horses, mute streptococci is observed only in smears prepared from abscesses. Nasal fluids from sick horses also contain other microorganisms, especially mixed with purulent microorganisms. Mute causative agents belong to the C group according to the type of antigen, that is, the strong hemolytic group. Streptococci are gram-positive, stain with all aniline dyes, and at 37°C, growth increases if horse serum is added to the culture medium. In pure young cultures, the capsule and blood agar form v - hemolysis. Unlike saprophytic streptococci, the causative agents of muteness are weak in their biochemical activity: they do not curdle milk, cannot break down lactose, sorbitol, and mannose. Mute streptococci secrete hematotoxin, leukotoxin, aggresin when grown in broth. These organisms grow in facultatively aerobic, artificial nutrient media, but grow best when supplemented with 10-20% horse serum or 5-10% blood.

Exciter durability. Mute streptococci are relatively resistant to environmental influences. In stables with poly soil, it is stored at a depth of 10-15 cm for up to 9 months, in dried pus - for a year, it is also resistant to disinfectants. It is kept active for 4 weeks in manure, 22 days in hay, hay, horse wool. It is inactivated in 6-8 days by sunlight, 1 hour at 70-75°C, boiling kills it immediately. 5% carbolic acid, 3% creolin, 2% formalin kill in 10-15 minutes.

Epizootological data. Mutism is an equine disease that is more common in horses. Horses get sick more often from 6 months to 5 years. Foals aged 1-2 months and horses older than 5 years are relatively rarely infected. In some epizootic cases, there are reports that even 15-20-year-old horses are infected with dumbness. Adult horses are less likely to be infected with the infection in the form of an immunizing subinfection. The immunity of young foals is related to colostral immunity. In the experiment, young mares brought from a healthy farm with this disease can be infected by rubbing the mucoid culture or abscess fluid on the nasal mucosa or by adding them to the feed. When administered subcutaneously, local suppuration occurs, when administered intravenously, *septicemia* occurs.

The source of the pathogen is *sick* horses, which secrete the pathogen with pus from the nose, sometimes mute streptococci is also found in the nasal mucus of a healthy horse. Animals that have recovered from the disease also serve as *carriers of the bacteria* for more than a year . For this reason, the disease of muteness, which is not brought from outside, can be recorded in the farm itself.

Transmission of the disease occurs mainly through alimentary and airborne routes. Factors that transmit the pathogen are fodder contaminated with it - hay, water, mangers, pasture hay, stable equipment. In less cases, contact: when dodging, sucking, touching each other, and other ways of transmitting the pathogen. The rapid spread of the disease is also caused by cold temperature, close keeping of horses, and

deficiencies in feeding. Mute often occurs sporadically, in some cases in the form of enzootic, epizootic. The disease can enter from other farms or appear on the farm itself. In the Arkhangelsk region of Russia, in the autumn season, among 3500 4-8-year-old horses, 300 of the 4-8-year-old horses were diagnosed with deafness. The rapid spread of the disease was caused by the fact that the horses were kept tightly in the stable in the rainy and cold weather. In this case, the disease is spread by air-droplet, because in this disease, horses sneeze. In the regions of Siberia, Kazakhstan, and the North Caucasus, deafness was also observed in the summer. In this case, the weather became cold and the horses drank ice-cold water, causing their resistance to drop. In case of muteness, the incidence is up to 5.67%, and the mortality is from 1.9% to 5.5%.

Pathogenesis. When dumb streptococci get on the nasal mucosa, they go from it to the submandibular lymph node through the lymphatic system, multiply there, and due to the irritant and the toxins released from them, the mucous membranes of the nasal cavity and throat are first serous, then purulent inflammations appear. As a result, it becomes very painful and difficult to drink water, especially to eat coarse hay. Inflamed submandibular lymph nodes become enlarged, hard, painless and warm. After 4-6 days, they soften, fester (abscess) and burst. Then the inflamed swelling is quickly absorbed, the pain disappears, the ruptured abscess is closed with healthy tissues, and the animal completely recovers from the muteness. Usually, if the mute is *typical* , the infectious process ends as mentioned above.

If the resistance of the horse is strong, if the virulence of the pathogen is low, the abscess will not form. Phagocytosis, antibodies, and all other defense systems are activated to destroy the mute streptococci. The disease will heal quickly. This condition is observed only if the disease occurs in an abortive form. However, in mares with low resistance, *a complicated metastatic form of dumbness* develops. In this case, the causative agent of the disease causes purulent inflammation of the deep lymph nodes of the back of the throat, ears, and neck through the lymphatic system. The causative agent enters the blood and causes a metastatic abscess process in the lungs, liver, kidneys and other organs. The transfer of bacterial toxins and decayed tissue from abscesses into the blood causes metabolic disorders in the body, as a result of which the body temperature rises, leukocytosis and heart failure are observed. Mute streptococci spreads throughout the body, if it spreads through the lymphatic system, all lymph nodes become inflamed, if it spreads through the blood, it causes *bacteremia* in the body due to the appearance of metastatic abscesses in the internal organs . *septicemia* occurs and the animal dies. Complicated form of muteness will definitely end in death.

Course, clinical signs and forms. The latent period of the disease is from 1-2 to 15 days, on average 4-8 days. Dumb is sharp. Depending on the development and clinical manifestations of the pathological process, *typical, abortive, complicated (metastatic)* and *genital forms of the disease* are distinguished.

The typical form usually begins with an increase in body temperature of 40-41°C. The animal is sad, the appetite decreases. At the beginning of the disease, the mucous membranes of the nasal cavity and throat become inflamed, red, and swollen,

which makes swallowing difficult. When drinking water, it comes back out of the nostrils. On palpation of the throat, submandibular lymph nodes are enlarged, local temperature is high, hard and painful. Rhinitis is observed in the nose, from which liquid, mucus, pus flows. The breath cooks and it accelerates. If the inflammation has spread to the larynx, a cough appears and it becomes difficult to breathe, wheezing is observed. Swelling covers the ear and lower jaw. The horse stretches its neck and stands motionless. After 4-6 days, the skin of the place where the submandibular lymph node is located softens, a special sound (fluctuation) is heard when palpated, and the abscess bursts and releases creamy pus. After the abscess bursts, the animal's body temperature normalizes. After the discharge of pus has finished, the place where the abscess has ruptured is gradually filled with healthy tissue over time. The disease lasts 15-25 days and recovers. Urinary excretion decreases in sick animals, protein appears in the urine. During the period of abscess formation, the amount of indican in the urine increases. During the recovery period, polyuria is observed for several days. ECHT in the blood is accelerated, leukocytosis is observed (18-25 thousand/μl), the disease lasts 2-3 weeks.

A genital form of the disease can appear when the bats are infected with mute streptococci during the breeding process . In this form, catarrhal-purulent inflammation, purulent mastitis in some cases is observed in the mucous membranes of the vagina, regional lymph nodes. The disease begins with the appearance of menstrual symptoms, redness and swelling of the mucous membranes of the vagina, followed by purulent vaginitis. In this form, acute catarrhal-purulent inflammation is visible in the head of the genital organ and in the urethra.

the abortive form , the mucous membranes of the nose become inflamed, a slight enlargement of the submandibular lymph nodes occurs. A sick animal discharges mucous, purulent fluid from the nasal cavity, it becomes difficult to swallow, and the throat is painful when palpated from the outside. In sick horses, fever up to 39-39.5 ^{0}C, slowing down of reactions, loss of appetite is observed, which lasts for 5-7 days and they recover.

In the complicated (metastatic) form , in addition to catarrhal purulent inflammation of the mucous membranes of the nasal cavity and throat, submandibular lymph nodes, the pathological process also includes purulent inflammation of the back of the throat, in front of the ears, and deep lymph nodes of the neck. Metastatic abscesses are detected in the lungs, liver, kidneys and other organs due to the transfer of the causative agent to the blood and spread throughout the body. Abscess is often observed in pre-ear lymph nodes, shoulder, knee, chest, and abdominal lymph nodes. In most cases, mucous membranes and regional lymphatic glands are inflamed. This condition, in turn, causes difficulty in swallowing and breathing. There is drooling from the mouth, pain when swallowing and signs of choking. If the abscess bursts, pus will come out of the mouth. If the abscess is in the parotid gland, the pus flows into the throat and enters the lungs, causing pneumonia, sometimes pus comes out of the jugular vein pit. If the abscess in the throat and larynx gets into the larynx, it causes *aspiration pneumonia* . It becomes difficult to breathe in and out. Inflammation of the deep lymph nodes in the neck

causes the pathological process to move to the pleura and pleuropneumonia. The most common complication of deafness is purulent inflammation of the lymph nodes behind the throat and in front of the ear. These lymph nodes become enlarged, pus forms in them, the horse stretches its neck and stops moving its head.

Inflammation of the mesenteric lymph nodes causes bowel dysfunction, diarrhea, constipation, colic, fever, and weight loss. If the abdominal abscess ruptures, peritonitis occurs and the animal dies. A characteristic symptom of this form is constant fever in the body. In complicated (metastatic) deafness, leukocytosis in blood increases to 20-25 thousand/μl, erythrocytes up to 2 million/μl, hemoglobin by 20-30%, ECHT - up to 75 mm/h. Complicated mute usually ends in death.

Pathologoanatomical changes. A catarrhal - purulent inflammation is observed in the nasal cavity, mucous membranes of the throat, and submandibular lymph nodes of a dead horse when it is typical of a dead horse. In the complicated (metastatic) form, purulent foci are visible in all lymph nodes and internal parenchymatous organs. There is hyperemia in the mucous membranes of the throat, hyperemia and hemorrhage in the mucous membranes of the small and large intestines. Bronchial, mediastinal, mesenteric lymph nodes are inflamed, enlarged, and bleeding. Purulent foci are observed in lungs, liver, spleen, kidneys. Several liters of serous-fibrinous, yellowish-reddish liquid accumulates in the chest cavity. If a horse has died of mute bronchopneumonia, the lungs will look like a sheep's butt and be hard.

Diagnosis. When the disease is typical, a diagnosis is made based on clinical symptoms, epizootological data, pathologoanatomical changes and, of course, laboratory tests. It is difficult to diagnose the disease if the clinical symptoms are only in the respiratory tract. In such cases, a bacteriological examination will be carried out. Dumb streptococci differ from purulent streptococci by the following symptoms:

- up to 250 microorganisms form a chain in a culture of mute streptococci or a smear made from pus, and in a bacillus with pus there are only 10-15 b points (segments) in the chain;

- In 5% blood agar, streptococci of mute produces v-hemolysis, the colony becomes round b, smooth, slimy. V-hemolysis also occurs in the culture of bacilli, but the colonies are in the form of drops and do not join each other, another difference is that the colonies are dry;

- Mute streptococci form a capsule on 5% blood agar. Purulent bacilli do not form a capsule. If these data are not sufficient , biochemical tests are performed.

Differential diagnosis. It is necessary to distinguish the disease of muteness from the diseases of the flu and the flu. It can be distinguished from manga disease by performing maleinization and, if necessary, applying KBR. Influenza spreads quickly and does not have purulent inflammations.

Treatment. Sick animals are separated in a separate bright, clean room. It is advisable to store in the open during hot weather. Feeds with easily digestible soft hay, boiled or steamed roots and fruits are given. If swallowing is difficult, a slurry is recommended. Antibiotics and sulfonamides are used in general treatment. In special

treatment, an antiviral drug prepared from mute streptococci is used. Antiviral is injected under the skin of the neck of a sick animal in the amount of 50-100 ml. It is advisable to send it to several places. If the effect of the antivirus is not good, the antivirus will be sent again after a day. Antiviral drug can be used in the treatment of abscesses, washing. In case of hyperplasia of the lymph nodes under the jaw, in front of the ear, the antiviral drug is also injected under the skin of that area. In patients with metastasis, in addition to antibiotics, 33% alcohol is added to 20-30% glucose and 1% norsulfazol in the form of a complex solution. The first time, the amount of alcohol is 150-200 ml, and the following days it is increased from 50 ml.

In order for abscesses to be absorbed faster or faster, fur is cleaned and degreased, and sulfur-mercury ointment is applied. The ointment is applied in a linear pattern. After applying the ointment, a warm bandage is applied. Mature abscesses are treated surgically. The cut abscess is washed with a 1:1000 solution of potassium permanganate or other disinfectants, then wiped with an alcohol iodine solution or washed with the 2nd fraction of 20% ASD. Later, if the regeneration is good, the wound is not washed, but it is necessary to clean the wound with a dry tampon. 3-4 g of calomel and 10 g of bisalol are given per day for gastrointestinal injuries. Caffeine or camphor oil is injected to stimulate the heart. A liniment made of 1 part of turpentine + 2 parts of camphor alcohol is rubbed into the swelling. A tracheotomy is used for respiratory distress. Water supply to sick animals is reduced. Add 8-10 ml of hydrochloric acid to a bucket of water or add 15-20 g of calcium chloride.

AP Alatarsev, taking into account the anaphylactic nature of the disease, recommended adding adrenaline with calcium chloride. The duration of treatment is 5-7 days. Every day, 50 ml of 5% calcium chloride, 10 ml of adrenaline 1:1000 solution are injected into the vein every day.

Immunity. Animals that recover from the disease develop strong immunity, which lasts a lifetime. Even in uninfected horses, the susceptibility decreases after 5 years, which is evidence of the presence of an immunizing subinfection.

Prevention and countermeasures. Mutism has not been recorded in Uzbekistan. However, this disease exists in some countries of the European continent, so there is a risk of introducing the disease. The way to protect vulnerable animals is to strictly control the dangerous border area, the horses and their products crossing the border, and if there is suspicion, it is necessary to check new arriving horses during the preventive quarantine period.

In nature, the main reservoir of the disease is horses, carriers of bacteria in latent form. Horses purchased for breeding or sports must be taken from countries, farms and farms that are healthy in terms of infectious diseases, checked for infectious anemia, mange, rhinopneumonia and other infectious diseases in the territory of that country before bringing them, and after bringing them 1 During the monthly preventive quarantine, it is necessary to carry out a clinical and laboratory examination of the mute. In order to ensure the resistance of horses, it is necessary to feed them with nutritious food rich in vitamins and minerals, and to keep them in accordance with the hygienic requirements.

If this disease is detected clinically, pathologoanatomically, bacteriologically among horses, within the framework of the Veterinary Regulation, the farm, farm or settlement is declared unhealthy and *restrictions* are imposed. In the unsanitary point, measures are taken to carry out all health measures and prevent the spread of the disease. Entry and exit of new horses to the farm, mixing of horses with other groups is prohibited.

Horses in the herd are clinically examined. Sick animals are separated and treated. Clinically healthy horses are left in the barn and given coarse hay and soft feed. The cage is disinfected weekly with 3% chlorinated lime, 3-5% caustic soda, 10% formaldehyde, monochlorinated iodine. If a disease occurs when the horses are fed in the pasture, the sick are isolated and treated, and the remaining horses are given 50-100 ml of antiviral. If the antivirus is ineffective the first time, it will be sent again after a day.

Horses are tied or kept in a closed place. Horses at the point are kept without being let out to pasture and are subjected to daily thermometry and clinical examination. The sick building is cleaned daily and disinfected every 7 days with 4% caustic sodium, the manure is biothermally disinfected for 3 months. *The restriction* from the unhealthy point is taken 15 days after the last patient has been treated, and the final disinfection is carried out.

Control questions

1.Pathogenicity and susceptibility .
2.Epizootology .
3.Pathogenesis, clinical symptoms .
4.Pathologoanatomical changes .
5.Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention .

Topic: CINFLUENZA DISEASE OF DOGS

Lecture	**Swine flu disease**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.

Treatment and prevention..	
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main

	lecture are described using visual materials.	points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.ne

Swine flu (Latin - Influenza suis; English - Swine influenza, Swine (Hog, Pig) flu; Russian-gripp sviney) - is an acute, highly contagious infectious disease characterized by depression, fever, weakness, It is characterized by catarrhal inflammation of the mucous membranes of the respiratory organs, conjunctiva of the eyes and, in severe cases, lung damage.

Historical information - the disease was first identified in 1918 in the United States during the human influenza pandemic. During this period, influenza spread widely among people in the world and more than 20 million people died. The flu virus first started in Spain and then spread around the world, hence the name "Spanish". A subsequent disease recurrence was attributed to Haemophilus suis in pigs. Only in 1931, Shop and Lewis proved that the causative agent of the disease is Influenzavirus A suis and that human and swine influenza viruses are closely related. Swine flu has been registered in almost all countries of the world. It spreads as an epizootic.

Trigger. Influenzavirus A suis, an RNA-storing orthomix virus, is a virus belonging to the genus influenza virus A. Its size is 70-120 nm. The virus has hemagglutination properties. The classification of the influenza virus is based on the arrangement of hemagglutinin and neuraminidase types in it in a certain order. The virus is genetically related to A-type, the causative agent of human and avian influenza. The virulence of this virus is determined by the gram-negative, polymorphic, low-virulence hemophilic bacterium Bact. It is more pronounced when combined with Haemophilis suis. Among the laboratory animals, the white mouse, rat, and mouse are susceptible to the virus. It can be grown in cell cultures made from chicken embryos and piglet kidneys and lungs. It is determined by neutralization of antibodies formed against it in the body of pigs infected with the virus, cessation of hemagglutination, complement binding reactions. Influenza virus can be detected in the larynx, bronchial exudates, lung regional lymph nodes, and nasal fluids when symptoms of the disease appear.

The origin of the swine flu virus is believed to have been transmitted to pigs from a sick soldier returning to the United States from Europe during the 1918-1919 human influenza pandemic. In fact, the N1N1 and less common N3N2 antigenic structures, which are more common in the swine flu virus, are also observed in

human and avian influenza. Based on the above, it can be said that the human, swine and bird flu viruses were genetically combined to **form a recombinant - hybrid virus** . This is why this "swine" flu virus attracts more attention than the common flu virus in humans, and the effectiveness of traditional flu drugs is lower. To date, 4 serotypes of influenza virus have been identified in pigs: H1N1, H1N2, H3N2 and H3N1.

Exciter durability. Resistant to external environmental factors, the virus is inactivated in 20 minutes at 60 ^{0} C, in 6 days at 18-22 ^{0} C. It retains its activity for 4 years under vacuum conditions and freeze-drying. Solutions such as 2-3% chloramine, formalin, phenol, caustic sodium inactivate the virus in 5-10 minutes.

Epizootological data. Pigs of all breeds and ages can get influenza, but young suckling piglets 2-8 weeks old are more susceptible. The disease is more common in cool and cold weather. Keeping pigs in unsanitary conditions, drafts in the building, humidity, rainy weather, close storage, keeping pigs of different groups together contribute to the emergence of the disease.

The source of the pathogen are *diseased* and *virus-carrying* pigs that shed viruses with nasal fluids when they cough and sneeze. The virus is mainly transmitted through the air (aerogeneous) from one pig to another. The spread of the disease from one farm to another occurs through sick, recovered or subclinically sick pigs. *Virus carriage* persists *for several months* in recovered pigs . Factors that spread the disease include forage, bedding, offal, manure and water contaminated with the virus. The presence of herd immunity among pigs is of great importance in the occurrence of the disease. This influenza virus is harbored by the eggs of metastrongylid helminths in the lungs and earthworms, which are considered its intermediate hosts. Pigs that eat earthworms also pick up flu viruses, causing healthy pigs to get the flu. That's why *it's raining Worms are the reservoir* of this disease . White mice, rats and gerbils are susceptible to influenza virus when experimentally infected. Influenza can spread rapidly as an epizootic, especially in damp, unsanitary conditions, tightly kept pigsties, and when pigs are undernourished, the disease rate is up to 100%.

In recent years, swine flu has been registered in countries with developed pig breeding, especially in the regions of countries belonging to different continents, such as Mexico, USA, Canada, Israel, Australia, Brazil, Guatemala, Spain. The swine flu virus can be transmitted from pig to pig, pig to human, human to pig, and human to human through the air (coughing, sneezing). Therefore, the spread of the swine flu virus is easier.

Pathogenesis . The virus enters mainly through the respiratory tract, settles and multiplies in the epithelial cells of mucous membranes, causing degenerative and destructive changes there and creating conditions for the development of secondary infection (cocci, bacteria). Haemophilic bacteria are more common in swine flu. Under the influence of the virus, the permeability of capillaries increases, as a result, the entry of toxic substances and pathogens becomes easier. In a pig infected with a virus, the body temperature rises, antibodies against the virus begin to form in the body, and phagocytosis increases. In pigs with low resistance, on the contrary, secondary infections develop, inflammation begins in the lungs, which turns into

chronic bronchopneumonia. If the activity of secondary infection is not eliminated, the pathological process deepens, worsens and the pig dies.

Course and clinical signs . The incubation period of the disease is 1-2 days, sometimes 6-7 days. The disease can be more *acute* , *semi-acute* , *typical* and *atypical* . The disease *is typical, acute* , begins with an increase in body temperature to 41 $^{0-42}$ 0 C, the animal is depressed, loses appetite, conjunctivitis, runny nose, cough appears , moves with difficulty. The mucous membranes of the eyes are swollen and reddened, serous catarrhal exudate flows from the corner of the eye. It becomes difficult to breathe due to the swelling of the mucous membranes of the airways, wheezing sounds when breathing. Nostrils, bronchi are blocked or narrowed by hardened exudate. A sick pig is in a state of depression. He digs the bed and lies down, his sensitivity to surroundings decreases, he has difficulty standing, and cough symptoms appear. Catarrhal exudate from the nose hardens and forms a crust in the beak. In the typical course of the disease, symptoms of lung inflammation appear. He sits like a dog to ease his breathing. The skin of the ears, tail and hooves turns blue due to a decrease in heart activity. Some patients have diarrhea and constipation. Knees, shoulder blades, lymph nodes swell.

The most susceptible 2-8-week-old piglets have influenza in the form of acute catarrhal or catarrhal-purulent pneumonia. In piglets, breathing becomes faster, wheezing is heard in the lungs, and coughing intensifies. Death is often observed in sick piglets of this age. In large pigs, when the flu is acute, the disease usually lasts 7-10 days. In pigs with high resistance, the disease is mild and can recover in 4-6 days. In older pigs, the output usually does not exceed 2-4%, but in suckling piglets it can be 60-70%. Because the disease is severe in suckling pigs and young pigs separated from their mothers. They have inflammation of the lungs. In addition, they develop pleurisy, pericarditis, and dermatitis. Sick pigs lose weight.

the flu is *semi-acute* , it lasts for several weeks, usually in pigs bronchopneumonia, and in some cases pleurisy is observed. Such pigs die in 15-30 days. Those who recover are small and emaciated. Sometimes a relapse is observed in pigs. They have more dermatitis and eczema compared to the acute one. In salmonellosis and pasteurellosis unhealthy farms, piglets die from secondary infection when the flu is subacute.

When the disease is atypical , the clinical symptoms are unknown, coughing, runny nose, rapid breathing, loss of appetite. The lungs are not inflamed, the temperature does not rise. If properly treated, well fed and well kept, sick pigs will recover in 3-6 days.

Pathologoanatomical changes . The disease is typical, when it is acute, upper respiratory organs are inflamed, mucous membranes are inflamed, there is bloody foamy mucous fluid in the nasal cavity, larynx and bronchi, the bronchi are filled with mucous plug and the lungs are swollen. will be Hemorrhages are visible in the mucous membranes. Serous-catarrhal pneumonia is observed in the upper and middle part of the lung, sometimes this change completely occupies the left or right part of the lung. In rare cases, pleurisy is detected. A clear, sometimes slightly cloudy, red fluid may accumulate in the abdomen, chest cavity, and under the pericardium. The

inflamed tissue is harder, pale red or dark red in color, shiny. The mediastinal and mesenteric lymph nodes in the chest and abdominal cavities are swollen, filled with blood and enlarged, and in some cases, point hemorrhages are observed. Hemorrhages are visible in mucous and serous membranes. In the semi-acute stage, the main changes are in the form of croupous - necrotic or purulent inflammation in the lungs. Fibrinous pleurisy and pericarditis are often detected. Catarrhal inflammation is usually observed in the mucous membranes of the gastrointestinal tract.

Diagnosis. The season (autumn-winter-spring), the rate of infection and spread, inflammation of the respiratory organs are characteristic symptoms of influenza. However, in pigs, parainfluenza, chlamydial pneumonia, adenovirus infection, and mycoplasmosis pneumonia also have the same clinical symptoms as influenza. Therefore, the diagnosis based on clinical signs, epizootological data and pathologoanatomical changes is the initial diagnosis. A final diagnosis of influenza is made based on laboratory tests. Nasal fluid, a piece of the mucous membrane of the nose, larynx, lung and lymph node are sent to the laboratory. Blood serum is sent with a referral letter at the beginning and end of the disease to determine the increase in the titer of anti-influenza antibodies in the body. Using immunofluorescence and hemagglutination reactions, the virus or antibodies against it are detected in mucous membranes.

Differential diagnosis. Influenza should be distinguished from plague, pasteurellosis, salmonellosis. Season is not important in plague, death rate is high. Diphtheritic inflammations (cecum, colon) are observed in internal organs, stomach and intestines. In pasteurellosis, fibrinous inflammation is observed in the lungs and intestines. Pasteurella is detected during bacteriological examination. Piglets under 2 months of age are mainly infected with salmonellosis. Characteristic necrotic, diphtheritic and bumpy changes occur in the mucous membranes of the large intestines. Bacteriological examination isolates salmonella.

Hemagglutination inhibition reaction (HATR), KBR, immunofluorescence reaction, IFT and PCR are used to diagnose influenza.

Treatment . Special treatments have not been created. Antibiotics, sulfanilamide and nitrofuran drugs are used to prevent secondary bacterial infections. First of all, patients are immediately separated into a separate dry, bright, clean, ventilated room and maintained with complete blood nutrients. The bedding is changed frequently. Dietary food, jelly, liquid porridge, aromatic porridge are provided.

Immunity. The formation of virus-neutralizing, agglutinin and complement-binding antibodies was detected in the blood of animals recovered from the disease. However, their importance in the course of the disease, the period and level of preservation in the body have not been well studied.

Prevention and countermeasures. To date, this disease has not been recorded among pigs in the territory of our country. To prevent the swine flu virus from entering the territory of our country, it is bought from abroad pigs should be taken only from a farm, region and country healthy for this disease, and imported pigs

should be under veterinary-sanitary control. Keeping pig farms in a closed type, allowing people to enter through a sanitary conduit at the entrance, organizing a dezobarrier at the entrance to the farm, organizing an entry through dezogilams at the entrance to each building, preventing the entry of strangers and all kinds of animals, among the measures for the prevention of this disease. is one. Placement of pigs based on veterinary and sanitary requirements, care, attention to protein balance in feeding, feeding rich in trace elements and vitamins, protection from heat and stress, quick cleaning of the pigsty from manure, regular disinfection, in particular, disinfection of the transport that entered the farm allows to keep the farm healthy from this disease. Since the cooling of the weather is one of the main factors in the origin of the disease, the pigsties should be warm, bright, clean, and the ration should be full of value. There should be no drafts in the pigsty, and the bedding should be dry. Newly brought pigs are kept in preventive quarantine for 1 month and undergo daily veterinary examination.

Pigs and their products coming through the border customs, regardless of the means of transportation (airplane, train, car), must undergo a thorough veterinary-sanitary inspection, the place of origin of the product must be checked for this disease. it is necessary to have a document confirming the authenticity and verify that the product is not coming through a third country; pork and meat products from unhealthy areas should not be allowed to enter the country through customs, even if they are alone or with tourists.

If the disease breaks out, it should be prevented from spreading as an epizootic. It is necessary to bring the sanitary conditions and microclimate conditions of the piggery to the level of zoohygienic standard. Sick animals are isolated and treated. Current disinfection is carried out with 20% freshly slaked lime, 2-3% caustic soda, 1-2% chloramine.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention .

Topic: C SARAMAS DISEASE OF DOGS

Lecture	**Swine fever**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture

Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	*Results of educational activities:* Students: About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens,

	Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.
3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.
4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.
5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".
6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.ne

Saramas disease of pigs

Trigger. Eryseplotrix insidiosa, the causative agent of saramas, is a bacterium with a straight or slightly twisted form, sometimes also found in a filamentous form, which is mainly manifested in the presence of verrucous endocarditis in the heart and in stale broth cultures. . It is non-motile, has no spores and capsule, is stained with standard aniline stains and Gram's method. Grows in aerobic and anaerobic environments. Bacteria isolated from different sources differ in their antigenic status. A. Pokhil, V. Tilgalar note the presence of more "A" type, less "V" type, less often "N" type in the strains isolated from sick pigs, the virulence of the "V" type strains is low. , the immunogenicity is stronger, so it is used in the preparation of hyperimmune blood serum and vaccine against this type of sarcoid. This type is sometimes found in the back of healthy pigs, causing a subclinical course of the disease. Precipitation and hemagglutination reactions are used to determine the "V" strain.

Endurance. The bacterium is resistant to many environmental factors, especially the process of decay. Bacillus saramas was isolated 280 days after the carcass of a pig that died from the disease was buried. Even in organs left in the open air, the rod is kept for a long time. It can live comfortably in river water at 18-20 °C for up to two months, in hydrogen water for 3 months, in urine for 5-6 months, in manure for 3 months, and in soil for up to 3.5 months. Pickling and smoking do not kill the bacteria. Direct sunlight kills in a few hours. 100°C kills bacteria in seconds. 10% solution of chlorinated lime, 2% solution of alkali and 20% solution of freshly prepared lime and other substances are recommended for disinfection, resistant to the effects of substances used for disinfection.

Epizootology. Saramas bacteria are widespread in nature. It is pathogenic not only for pigs, but also for other species of animals and humans. R. Koch and Löfler isolated this bacterium in mice and called the disease murine septicemia. Later, it was found that the causative agents of murine septicemia, swine sera and human erysipeloid diseases belong to the same species. N. Olsuvrev, V. Svetkov, T. Dunayev proved that many types of rodents are susceptible to bacillus saramas, and it acts as a carrier and a focal epizootic. Water rats and mice (crots) perform the same

function. Rodents are so widespread in nature that they not only retain the bacillus saramas in their bodies, but are also a source of the pathogen for pigs. Sheep from domestic animals, mainly lambs from a few weeks to 4-8 months old, are infected. They are chronic polyarthritis, and they lose weight and wear. According to the observations of N. Rozanov, verrucous endocarditis is observed, sometimes acute. Body temperature rises, weakness, hemorrhagic enteritis and bronchopneumonia are observed. In laboratory animals, mice and pigeons are highly susceptible to saramas. According to the observations of P. Stepaykin and others, the causative agent of seramas was found in the state of secondary infection in chickens, cattle, horses, dogs and deer. In some cases, saramas is also observed in wild animals and birds in the zoo. According to V. Karchevyokyi, it spread through meat and fish products given together with rodents in the zoo. This type of disease has been found in deer, gazelles, kangaroos, wild boars, seals, turkeys, peacocks and quails. The reason for the spread of erysipeloid among fishermen is that some fish species are carriers of the microbe (V. Stefansky, A. Grinfeld, etc.). Saramas bacillus was also isolated from ticks (N. Olsuvrev, Ya. Golota). The most dangerous source of the causative agent is acutely infected and recovered pigs, as well as pigs with chronic disease. In some cases, the bacterium is stored in the solitary follicle of the back and intestine of healthy pigs. This condition can cause latent disease in pigs. If the requirements for feeding and care are violated, the latent disease can turn into an acute condition. White mice and rats are also a source of the pathogen. V. Kotov proved that there is a direct connection between water rats and pigs. Wellman and I. Toletyak state that mosquitoes carry the causative agent. Yes. Golat and V. Petrov consider this disease to belong to the group of natural foci, taking into account the fact that the saramas mite is widespread in nature among rats, insects and ticks. Feed, water, equipment and unsanitized waste from slaughterhouses are among the factors that lead to the spread of disease. The pathogen is also spread from farm soil (land), summer camps, and pastures. The disease of 3-12-month-old pigs is one of the main epizootological features. But in our conditions, saramas can also be found in winter, because the winter comes mildly, and pigs are released into the pastures. Lactating pigs are resistant because they have passive immunity. The pig and fattening group that is put for repair is considered to be very susceptible. Adult pigs are resistant to the disease as a result of vaccination and due to immunizing subinfection. Susceptibility to the disease depends to a certain extent on the conditions of keeping and feeding pigs. The lack of protein, mineral substances, vitamins in the ration has a negative effect on the animal's body and increases the susceptibility to disease. On the other hand, in fattening, due to the fact that carbohydrates are given a lot, it does not spread. Rapid changes in temperature, excess humidity, and frequent overheating of the body affect the degree of susceptibility. Saramas often spread in the summer, when humidity increases. Violation of the means of ventilation of pig houses, carrying pigs in transport for a long time accelerates the disease.

Pathogenesis. The bacterium enters the pig's body through the alimentary route, and when the integrity of the skin is broken, it passes through the skin. The latent period of the disease can last up to 10 days. The bacterium that enters the body

does not immediately pass to the blood and other internal organs. It first falls on the fringe and solitary follicle. In this place, it releases toxins and affects the body with it (sensitization). This, in turn, contributes to the emergence of a septic process. At a certain stage, the bacterium overcomes the body's defenses and enters the lymphatic system, then the blood and spreads throughout the body. As the infection increases and the toxin increases, tissue metabolism is disrupted, strong functional changes occur, and dystrophic and necrobiotic changes occur in parenchymatous organs. This condition is especially aggravated in the cardiovascular system. As a result of blood stagnation in the heart muscle and its capillaries, swelling occurs and blood clots form. Tissue elements also undergo proliferation in the reticuloendothelial system.

Course and clinical signs. Depending on various factors (age, degree of obesity, nature of virulence) Sarams can be very acute, acute, semi-acute and chronic, as well as in the form of skin and ass.

Very acute course. This condition occurs mainly in 7-10 month old pigs fed on cuttings. It spreads rapidly when the ventilation equipment of the pig houses is not working properly or when the pigs are transported for a long time in transport. Such a transition lasts for several hours, and the body temperature rises. Hyech does not eat anything, is very relaxed, a nervous breakdown occurs, and the sick pig dies.

Acute or septic condition. In this case, the body temperature rises to 42°C and does not fall until the end of the disease. After a few hours, the appetite is suppressed, and general weakness and malaise occur. A sick pig is bedridden. When he walks, his legs keep hitting each other. Conjunctivitis is observed. Stomach-intestinal damage becomes inflamed and constipated. Sometimes he vomits. The condition of a sick pig will worsen. Blood vessels beat up to 100 times per minute. Breathing becomes difficult as a result of heart failure and lung swelling. Bluish spots appear on the skin under the jaw, chest, neck and abdomen. The disease lasts 2-3 days. In most cases, a sick pig dies.

Donkey (krapivnisa). The overall change is slower and lighter. The first sign of this phenomenon is an increase in body temperature of 41 °C. Appetite is stifled, heartburn and gnawing. 1-2 days after the body temperature rises, eczematous swellings appear on the shoulders, flanks, and neck. They are colorless at first, then reddish and gradually turn reddish-blue. This condition occurs as a result of inflammation passing under the skin. Tumors are rectangular, rhombic, and square in shape, ranging from 1X2 to 3X4 cm and larger. Sometimes these tumors become very large by joining together. With the appearance of changes on the skin, the condition of the sick animal will be slightly relieved. He may have a fever and lose his appetite. In obese pigs, the disease worsens, turns into septicemia and ends in death. Deep and large dermatitis can become necrotic, and the disease can turn into a chronic form.

Passing in the form of skin lasts 10-12 days.

Chronic pain. Verrucose. passes with endocarditis, arthritis and necrosis of the skin. This condition often begins after an acute episode or at the end of a chronic illness. A sick pig gets up with a meltdown, its temperature and appetite do not change much. When Saramas chronically sneezes, the condition of a sick pig periodically worsens. The temperature rises and it becomes difficult to breathe.

Bluish spots appear in the ears and abdomen. As the disease progresses, the patient loses his appetite, begins to lose weight, and becomes sluggish. He breathes heavily, his heart beats faster, and his lungs swell. Serous-fibrinous polyarthritis develops in the joints. Joint injury disrupts coordination of walking and makes it difficult. In some cases, joints can be deformed and broken. The disease causes abortion in piglets. As the necrosis increases, the skin cracks and rises, causing pus to ooze. Death occurs as a result of heart failure.

Immunity. Vaccinated from the age of 2 months with a stored, live, liquid vaccine against distemper (not possible before and after birth up to 1 month). Piglets are vaccinated twice in 12-14 days with an interval of 14 days after weaning. The vaccine is injected subcutaneously in the inner side of the thigh or in the neck for the first time 0.3 ml and for the second time 0.5 ml. Immunity appears after 7-10 days and lasts up to 6 months. In some cases, if there is a reaction, it is treated with anti-serum serum and penicillin.

$_{2\ \text{strain}}$ against Saramas . For preventive purposes, all pigs from 2.5 months of age and sows are vaccinated 15-20 days before farrowing. The vaccine is liquid, 0.5 ml is injected intramuscularly in the thigh or neck of 2-4 month old pigs. The first revaccination is carried out in a dose of 1 ml after 25-35 days, and the second after 4-5 months in a dose of 1 ml. Pigs sent in bulk for transport are vaccinated by sending 1 ml 20-30 days before shipment. Immunity lasts 4-5 months.

Pathologoanatomical changes. In a septic state, dark-red spots are visible on the skin of the chest, abdomen, chest, ears and feet of the carcass. Due to swelling in the lungs, foamy fluid mixed with blood flows from the nose. The cyst becomes swollen and swollen, and when cut, a mucous liquid flows. The blood is dark-red and poorly coagulated. There was a serous fluid in the chest and abdomen, and fibrins were deposited. The large and small veins are full of blood, the lungs are swollen, and there is foamy fluid in the bronchi and tracheas. The blood vessels of the heart muscle are full of blood and there is a point of bleeding. Gastrointestinal inflammation and sometimes bleeding. The liver becomes enlarged and cystic. The kidney is enlarged, and point-like bleeding is observed at the base of the capsule. Verrucous endocarditis occurs in the heart. This in turn leads to embolism and infarction (in the lungs, kidneys and spleen). And in arthritis of the joints, the joint swells, thick serous fluid flows. If the process is severe and deep, it leads to caries of the bone tissue and deformation of the joint.

Diagnosis. The diagnosis is based on the epizootology of the disease, clinical symptoms, pathologoanatomical changes and the results of laboratory examination methods. The whole carcass or its heart, liver, spleen, kidney and tubular bone are sent to the laboratory. Laboratory tests include microscopic examination of the smear, inoculation into artificial media, and infection of laboratory animals.

Examination under a microscope. A smear is prepared from the sent organs and stained by the Gram method. In chronic cases, a smear is made from the heart valve. In the first case, the trigger is rod-shaped, and in the second, it appears as a thread.

Bacteriological examination . From the sent samples, it is cultivated in MPA, MPB and Hottinger broth. Incubation lasts one day in a thermostat at 36-37°C.

Biological method . The sent sample is crushed in physiological solution and then filtered (1:10). 0.1-0.2 ml of this liquid is injected subcutaneously into 2 mice. Mice die after 2-4 days, observation lasts up to a week.

Differential diagnosis. Swine fever is directly transmitted and often ends in death. Pigs of all ages are affected, regardless of the season. In the end, the disease is complicated by salmonellosis and pasteurellosis. In this case, an autopsy shows a button in the colon and pneumonia in the lungs.

Pasteurellosis. Sometimes it occurs independently, and sometimes in the case of secondary infection. Acute and chronic course passes with a sign of croupous pleuropneumonia. Bacteriological examination gives a good result.

Listeriosis. It has a limited distribution, mainly in suckling pigs. Meningoencephalitis is caused by listeria.

Treatment. Hyperimmune blood serum prepared against Saramas is the most effective. It is injected intramuscularly at the rate of 1-1.5 ml per 1 kg of weight. If there is no change after 8-10 hours, the serum is re-injected. Penicillin works well. 2000-3000 GB per 1 kg of weight is sent between the muscles. It is even more beneficial when administered with hyperimmune serum.

Ekmonovocillin is administered 2-3 times a day at the rate of 4000-6000 TB per 1 kg of weight.

Eat. T. Trishkina recommends 5-8 mg of erythromycin per 1 kg of weight. Injecting this with serum has been shown to speed recovery in sick pigs.

Prevention. Control of rodents and various insects is one of the common measures. Deratization and disinfection are carried out according to the plan. It is necessary to raise a herd of healthy pigs. The veterinary sanitary condition of piggeries and summer camps will be raised to the required level. Scheduled vaccination of pigs is carried out on time in strict compliance with the exhibition. All pigs are clinically examined and thermometry is performed when disease is detected. Sick pigs are treated in the above way. In an unhealthy economy, restrictions are announced. After the last sick pig recovers, the restriction on the farm is lifted after 14 days. Forced slaughter is carried out in a special place, the meat is boiled and then allowed to eat.

Human sera can be found in tanneries, butchers, and fishers. The disease is transmitted to people through scratches and wounds. The injured area becomes swollen, painful, and redder in color. Sometimes the joints swell, the temperature rises, the body aches, the lymph nodes swell and give pain. The illness lasts 10-20 days and then recovers.

$_4$ in a ratio of 1:3000 is recommended for treatment . Ixtiol oil, antibiotics and ultraviolet rays are used.

An acute, highly contagious viral infectious disease, usually in cold climates, appears unexpectedly, is characterized by fever, malaise, and lung inflammation.

Historical information - the disease was discovered in the USA in 1918, in that year more than 20 million people died in the flu, which was widespread among

people in the world. The pathogen was invented by Shope and Lewis in 1931. The disease is registered in almost all countries of the world.

Economic damage - the disease causes a large economic damage, death is 10-60%, it is calculated by the costs of treatment and control measures.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention.

Topic: SALMONELLOSIS OF YOUNG ANIMALS DISEASE.

Lecture	**Young animals s with almonellosis _**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
Form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	*Results of educational activities:* Students: Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
Education	Lecture. Brainstorming.
Organizational form of education	Mass, collective.

Educational tools	Text. Table . Video projector. Computer.
Training conditions	Specially equipped lecture hall.
Monitoring and evaluation	Oral examination. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Educator*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1. Listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He hears, clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic .

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.
3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.
4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.
5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".
6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _
2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.ne

Salmonellosis (paratyphoid) is an infectious disease of young animals, which is characterized by a rise in temperature, impaired gastrointestinal activity , and diarrhea.

Historical information. Microorganisms belonging to this group were identified by Salmon and Smith in 1885. In 1934, the International Society of Microbiologists found it necessary to give their name to the disease and its causative agent . In the former USSR, in 1929, P. Tavelsky identified the sheep parade fin. Almonellosis of various species of animals was studied by R. Sion, P. Andreyev, P.

Solomkin, K. Svetkov, I. Poddubsky, N. Mikhin, S. Vishelessky and others during 1934-1956 . And in Uzbekistan, A. Ahmedov, A. Siddikov, I. Bur lusky contributed to the study of the disease .

Economic damage. Salmonellosis causes huge economic damage. On average, 2-10 percent of calves get sick, and 20-30 percent of them die. In addition, there will be a compulsory slaughter. It is necessary to spend a lot of money on the treatment of sick animals .

Trigger. The causative agent of the disease belongs to the genus Salmonella , and in calves it is S enteritids dublin, sometimes S tupli murium, in pigs S cholerae suis, in lambs S abortu0Cvis , and in goats S abortus equivi. Found in a polymorphic state , it is well dyed with aniline dyes. Grows well in MPA and broth. Salmonella does not produce indole , releases hydrogen sulfide, does not curdle milk. The varnish is pure and without change in sucrose, glucose mannitol, gas and acid appear in maltose. Monoreceptor O and N sera are used for serological differentiation . White mice are very susceptible to salmonella. Den guinea pigs are less prone. The microorganism releases a strong poison (endotoxin) and affects laboratory animals in larger doses, and when administered intravenously, it also affects winter livestock. If the toxin of Salmonella affects a person and gets into food, severe poisoning occurs.

Endurance. Salmonella is very resistant to temperature. 70-75°C for 15-20 minutes, at minus temperature for 3-4 months, and in manure and water for a month. For disinfection, 3% alkali, 20% chlorine lime solution, 5% xylon naphtha are used.

Epizootology. Calves often get sick from one week of age, and it occurs at the age of two months and older . The disease is transmitted mainly by alimentary route, it can also be transmitted during the period of growth in the mother's womb. When the bacterium passes through a susceptible body, its virulence increases, and even healthy calves do not get sick. Salmonella nellosis is most often transmitted through milk and milk contaminated with the causative agent. The udder is infected with salmonella even when there is very little milk . When cows are kept in dirty areas , the disease can be transmitted when their calves suckle from udders contaminated with salmonella, or during milking. This situation is especially evident in the summer months. Bacteria-carrying animals pose a great danger in the epizoology of the disease , because they carry out pathogenic micro-organisms with their feces. If such animals enter a healthy farm, they will spread disease. Salmonella can easily live in the intestines of adult animals. A sick animal is the main source of disease spread. The feces of a sick animal is a source of a lot of bacteria, and urine and other excreta also infect the environment with salmonella. In an unsanitary farm, the clothes of experts and farmers, various dishes and other equipment, if they are not kept in accordance with sanitary requirements, are strongly contaminated with bacteria. Bacteria can easily live in dry organic environments . Therefore, if dried feces, nasal and oral fluids stick to clothes, dishes, barn walls, and mangers, they become a long-term source of salmonellosis. Salmonellosis can occur in all seasons of the year when good winter conditions are not created . But the susceptibility of young animals to the disease increases in winter and early spring . Because by this time, a number of

factors occur that lower the body's resistance. Salmonellosis in pigs is usually sporadic and is transmitted mainly in the womb. After birth, through the umbilical cord, or through milk during breastfeeding, through a bacterium-contaminated hook, infections are considered secondary, and often older puppies catch the disease during breastfeeding . It is an enzootic disease.

Pathogenesis. Once Salmonella enters the mouth, it travels to the gut. It releases endotoxin in the intestine and causes inflammation. If the body's resistance is reduced, it passes to the lymphatic apparatus on the intestinal wall, then enters the lymphatic and blood system. Thus, when the microorganism enters the body through the intestinal walls, septicemia occurs and similar clinical symptoms appear. Salmonella toxins play a major role in the pathogenesis of the disease. A sick organism is damaged not only by bacteria, but also by the poison it secretes. As a result of the effect of the poison on the body , an exudative process occurs, and strong hemorrhagic changes are observed in the mucous membranes . The areas of necrosis in the liver, spleen and kidneys are noticeable. Toxins that enter the blood affect the entire body through the central nervous system.

Course and clinical signs. Salmonellosis is acute and chronic in calves.

A sharp transition. Body temperature rises (40-410 C) . Heart activity becomes heavy (the artery beats 110-150 times per minute). Breathing is 60-80 per minute. From the first day, serous conjunctivitis occurs , many tears flow. Calves' reaction to the external environment decreases, they often lie with their heads on their sides . It does not stand by its own will. Appetite is unstable, sometimes he drinks milk, in some cases he does not drink it. After 2-3 days, diarrhea begins. Mucous substance, air bubbles are mixed in stool, it has a very unpleasant smell, and then there is diarrhea mixed with blood. When the disease is severe, the kidney is injured, and the sick animal urinates often, which causes pain. The total amount of urine is reduced, it is mixed with protein and epithelial tissue . In severe cases, the temperature rises very high. The sick calf lies down, does not react to the external environment , and dies in 5-10 days. When the disease becomes milder, the diarrhea stops, the temperature drops, and the disease becomes chronic. In this case, the gastrointestinal injury is relieved , and the respiratory organs are injured. A mixture of mucus and pus flows from the nose. At first, it is a dry, slow cough, and then it gets worse. The process usually starts with bronchitis and eventually turns into pneumonia. In some cases, the joints become swollen and painful, the area becomes hot, and it becomes difficult to stand and walk.

The disease is acute, semi-acute and chronic in pigs . It is acute in young pigs, and chronic in older ones .

A sharp transition. A septic condition appears, the body temperature rises to 41-42 °C. Breathing heavily, he presses his head between the beds, does not let his mother nurse him , and sleeps a lot. Strong diarrhea. In some cases, the stomach hurts, and the pig takes a sitting position. The eye becomes severely inflamed, conjunctivitis occurs, and the eye becomes pus-filled and unable to open it. Heart activity is disturbed, blue spots appear on the skin of the abdomen, ears, and under

the jaw . If it is not treated in time, the condition worsens due to severe diarrhea and pigs die within 5-7 days . Mortality is 50-80 percent.

Both acute and chronic course. A septic process occurs that is not actually manifested. From time to time, the body temperature rises, the heart beats faster, and breathing becomes difficult. With the onset of diarrhea , the above symptoms decrease and appetite is suppressed. Intermittent diarrhea alternates. In some cases, enteritis continues until the animal dies. Sick pigs lose weight, the mucous membranes become anemic, the skin is wrinkled and covered with dirty scales . Sometimes pneumonia occurs and mucous discharge from the nose. Piglets stop growing.

Pigs are infected with the disease in their mother's womb. When they are born, they do not try to suckle, it is very difficult . Breathing becomes difficult, body temperature rises to 40 °C. After 1-2 doses, diarrhea begins, and then the slave dies. Arthritis occurs in slightly older dogs. The joints are swollen and painful. It becomes white and difficult to walk. Diarrhea is rare in older dogs , but severe diarrhea can be fatal. When it's mild, the wounds can heal very slowly. As a result of stunted growth and prolonged arthritis, the legs become deformed, which in turn renders sick dogs useless .

Treatment. After clinical examination and thermometry , it is recommended to divide the calves into the following groups : 1) healthy; 2) a disease is suspected; 3) clearly infected; 4) healed calves. Groups should have their own equipment and trainers. Nutritious and high-quality nutrition should be established. Levomycetin, synthomycin, tribrissen are recommended for treatment . Antibiotics with sulfonamides (norsulfazol, disulfan, etazol, sulfa din, sulfademyzin) give good results when complications of pneumonia are observed.

Nitrofuran series furazolidone, furasin, furazolins have a high value. Antitoxic serums used against hyperimmune salmonellosis are very beneficial. It is recommended to give Sintomycin 3 times a day with milk . The first time is 0.04 per 1 kg of weight, and the second and third time is 0.02-0.03. In order to prevent recurrence , after the healing of the wounds, syntomycin administration is continued for another 2 days, and terromycin and biomycin are continued for 3 days . They are given from 0.02 per 1 kg of weight. In addition to these, it is possible to inject penicillin into the muscle . Always work to determine the effectiveness of microorganisms in relation to drugs, increasing the useful coefficient of washing. This task is assigned to laboratories.

Emlash S c h e m e

Age of sheep	Vaccine dose (ml)	
	First	Second
From 20 days to 3 months	1-2	2-3
From 3 months to 1 year	1.5-2	2-3
First birth	3-4	4-5
From 3 years old	4-5	5-6

Immunity. Animals that have recovered from the disease develop immunity. The following vaccines are available for immunization .

gan formal kvass vaccine used against salmonellosis . In unhealthy farms, two months before calving, 10-15 ml is sent every 8-10 days. Calves are vaccinated twice with an interval of 3-5 days at the age of 1-2 days. After 1.5-2 months, revaccination is done .

Sheep are vaccinated against salmonellosis with a formal thiomersal vaccine (scheme).

using the dried live vaccine prepared from the TS-177 strain used in swine paratyphoid, 1 ml/mln to the state of the germ cell. is dissolved in physiological solution. The vaccine is injected subcutaneously 2 times starting from 2 weeks of age . 0.3 ml in 2 weeks, 0.8 ml after 1 month. 0.5 ml at 1-4 months, 1 ml after 4 months. Apart from these, there is also a vaccine for waterfowl . Currently, the vaccine in the associated state is also used. Any vaccine recommended for use in practice is accompanied by instructions for use and must be strictly followed.

Prevention. The fight against salmonellosis is carried out throughout the entire strait, starting from the day the cattle are released. During this period , special attention is given to care and diet along with nutritious and recommended food . It is necessary to carry out these measures with care, especially in the third half of the strait . To increase the resistance of newborn calves , it is recommended to give acidophilin, acidophilic broth culture (ABK) and propion-acidophilic broth culture (PABK). If the disease appears , it is immediately treated with the methods and drugs mentioned above . Newborn animals are vaccinated with the above-mentioned vaccines.

In salmonellosis, sick animals are the most dangerous source of the disease. Therefore, it is necessary to diagnose them in time, isolate them and carry out current disinfection. 2% active chlorine, 20% chlorine lime solution, 5% chlorine (I) -iodide, 2% formalin are recommended for disinfection .

Bacteria-carrying animals are strictly considered and bacteriological and serological examination is carried out.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention

Topic: COLIBACTERIOSIS OF ANIMALS DISEASE.

Lecture	**Young animals colibacteriosis ill _**
Lecture teaching technology	
Study time: 2 hours	Number of students: 90
Form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention..	*Results of educational activities:* Students: Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
Education	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Training conditions	Specially equipped lecture hall.
Monitoring and evaluation	Oral examination. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Educator*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.

2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1. Listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He hears, clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.
3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.
4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.ne

Colibacteriosis . Acute infectious diseases of young animals . It is found mainly in animals from 1 to 8 days old . Symptoms of the disease: enteritis, sepsis and weakness.

Economic damage. Since the disease starts on the first day in young animals and is acute, the loss is high. In some cases, 10-20 percent mortality is observed. In farms where diagnosis is not made in time , treatment and preventive measures are not properly organized, death does not stop and the farm suffers great economic losses.

Trigger. Eat the disease. The main representative of microorganisms belonging to the Soli group is Escherichia Coli . Several groups of enteropathogenic escherichia are found in calves: 08, 09, 015, 026, 041, 055, 078, 0101, 0115, etc.

Endurance. Escherichia coli can live for months in the dried protein environment in animal excrement, mucous substances, and blood. It is not very resistant to heating, it dies immediately at 75-80°C. In our conditions, summers die quickly. At the same time, it lives in the body of a healthy animal and is released into the environment.

Epizootology. All types of young animals are susceptible to colibacteriosis. Newborn animals are sick on the first day. Colimastitis and coliendometritis were sometimes observed in older animals. The causative agent of the disease is widespread in nature , and it can be isolated not only from a sick animal, but also from the gastrointestinal tract of a healthy animal . Colibacteriosis is strictly an enzootic disease. The main source of its pathogen is sick and recovered animals. Adult animals also spread enteropathogenic escherichia. The disease is mainly

transmitted through the alimentary route, there are clear data on the transmission of the disease in the womb of calves and lambs. A sick animal isolates Escherichia coli through all wastes released into the environment and infects a number of objects in the environment. In some cases, the disease can be transmitted through unsanitary oral milk. Contamination of pacifiers and various dishes with microorganisms is important in the origin of the disease . Farm workers play a positive role in the spread of colibacteriosis in unhealthy farms . Not wearing special clothes , not washing and ironing them on time are also factors that cause the spread of the disease . The influence of economic conditions on the origin of colibacteriosis is great. The keeping of young animals in dirty, dark, old buildings , lack of balanced feeding of strait animals with the necessary food , lack of sanitary issues are among the factors that accelerate infectious diseases of young animals , including colibacteriosis .

Pathogenesis. The mucous membrane of the intestine of a healthy young animal can completely prevent the passage of microorganisms . If a young animal is born prematurely, its defenses are greatly reduced, and the pathogenic microorganism that enters it can pass through the mucous membrane .

causes inflammation . The inflammatory process, in turn, allows microorganisms to penetrate deeper . At the same time, intestinal motility increases. It is a reflective protective device that is formed to repel the tickling agent . If the protective device fails, the bacilli enter the lymph nodes and blood, and a septic process occurs . Severe diarrhea occurs and the body becomes dehydrated. Bacteria increased in the blood develops and spreads to the whole body, affecting the central nervous system through its toxin, disrupting its function. As a result, a comatose state appears.

Course and clinical signs. The latent period of the disease lasts from several hours to a day, and clinical signs are basically the same in all young animals. The disease begins with a sudden increase in body temperature , heart rate and breathing speed up. The sick person lies on the bed, the nose is dry, and the mucous membranes of the eyes are swollen. After 1-2 days, enteritis is added to the septic condition. It goes like water, it is foamy, mixed with air bubbles, white-bluish in color, and has a bitter smell. Clotting occurs when undigested milk passes through the bowels . Diarrhea is observed in a mixed state of mucous fluid and blood . As a result, the back legs and thighs become dirty, and pain is felt when the abdomen is touched . As soon as the diarrhea stops, the temperature drops. Work will disappear. With increased diarrhea, the sick animal loses weight and lies down. Throwing his neck to his side , he reaches for his waist. The eyes sink and the wool loses its natural luster. Sticky sweat hardens on the skin and emits a foul odor. The pulse slows down, breathing becomes shallow. As the disease worsens, the noted symptoms reach their peak. A comatose state occurs. If the disease is prolonged in the joints, the joints may become inflamed and swollen. In these cases , it becomes acute, enteritis increases, and weight loss occurs quickly. If left untreated, the slaves will die.

Pathologoanatomical changes. The body of the dead animal is emaciated, the area around the hind excrement hole and the hind legs are contaminated with feces.

Mucous membranes are strongly hemorrhagic . There is a cheesy mass in the stomach, curdled serum has accumulated, the mucous membrane of the stomach is red and bloody, and there is a liquid, slimy mixture of food residues in the small intestine . The mucous membranes of the small and large intestines are swollen and covered with mucous fluid, hyperemia and hemorrhage occur, lymph nodes are enlarged and reddened, the spleen has not changed much, the kidney and liver are anemic, and blood is poured into the capsules.

Diagnosis. Diagnosis is made based on epizootological data, taking into account clinical signs and pathologoanatomical changes . All these indicators are confirmed by the results of bacteriological tests. To which serological group it belongs will be determined by checking with serum . Microbiological examination is carried out as follows: on the 1st day, the organs and the mucous membrane of the small intestine are taken and planted in artificial media. It is checked by sliding. On day 2, colonies are cultured on Endo, Levin, MPB and Simmons media. On the 3rd day, antigen is prepared from culture media and serologic group is determined. White mice are infected. On the 4th day, biosine is determined. After that, sensitivity to antibiotics is studied.

Differential diagnosis. Clinical manifestations of colibacteriosis are very similar to salmonellosis, dyspepsia. Therefore , not only clinical signs, but also its epizootic and pathologo-anatomical changes, as well as bacteriological examination methods, are addressed. Unlike salmonellosis, colibacteriosis starts from day one. If you get sick on the 7-8th day, the temperature starts to rise, enteritis is observed, colibacteriosis is suspected. In case of salmonellosis, the disease usually starts on the 7-10th day and can last for several months. In case of coli bacteriosis, septicemia develops strongly, there is non-stop diarrhea, the sick animal is completely weakened and lies down. In salmonellosis, clinical symptoms develop more slowly, but there is always a fever in the body. The absence of strong changes in the spleen and the absence of necrotic foci in the liver indicate pathologoanatomical changes characteristic of colibacteriosis. Blood culture is isolated in acute salmonellosis . In chronic condition, respiratory organs and joints are injured.

Toxic dyspepsia. This also starts from the first day , there is non-stop diarrhea, the sick animal lies down without medicine, but the body temperature does not rise. Absence of bleeding in serous linings and unchanged spleen is characteristic of dyspepsia. In case of coli bacteriosis, the entire body, if it dies in winter, and pieces of tubular bone and parenchymatous organs, if it dies in summer , are sent to the laboratory in 30% glycerin solution, escherichia is isolated from the samples and its serotypes are determined. For this, type-specific serums are used .

Treatment. When the disease is accurately diagnosed in time , treatment begins with diet. Instead of colostrum, physiological solution or bitter brewed black tea is given cold. It is even more useful to mix chicken eggs in 1 l of the liquids mentioned above . In addition to being nutritious, it is also rich in lysozyme. Before using antibiotics , it is necessary to determine the sensitivity of the isolate to them. Only then we can choose drugs that affect the microorganism and the treatment will be effective. Synthomycin is used for treatment . 40 mg for the first time, then 20 mg

every 4-6 hours. Biomycin, terromycin , tetracycline 15-20 mg 2-3 times, colimycin 15-20 mg, and polymyxin 4 mg are recommended. It is better to give antibiotics with milk. In some cases , when doctors get hold of a newer antibiotic, they immediately start using it against gastrointestinal disease . This is definitely wrong. First, the sensitivity of the microorganism to the antibiotic should be studied in the laboratory, and then it should be used. If this is not done, the treatment will not help.

Caffeine, camphors are used to support cardiovascular activity. If antibiotics cannot be found, some sulfanilamide drugs - sulfazol, sulsimid, disulfan, phtolazol - can be used. Administration of glucose-saline solutions under the skin or in the abdominal cavity helps to maintain the process of water-salt exchange. The solution recommended by Professor IG Sharabrin (1 l distilled water, 8.5 sodium chloride, 13.0 sodium bicarbonate, 0.3 calcium chloride, 0.5 potassium chloride, 50.0 glucose, 0.2 caffeine, 500 thousand TB penicillin) is also beneficial in enteritis . For calves, 0.5-1 l is administered intravenously, and subcutaneously for pigs, pigs and lambs. A deep enema cleans the intestines. In addition, there are various preparations recommended by a number of scientific testing institutes and laboratories . If they pass the control and arrive with a blind eye, it is necessary to support. Serum and bacteriophages prepared in biofabrics and biosex , in case of colibacteriosis, serum is injected under the skin, and phages are ingested. The treatment effect is very high.

Immunity. The composition of colostrum is known to doctors, so there is no need to dwell on it. Polyvalent GOA formal thimersol vaccine is used 1.5-2 months before calving in cows and calves in unhealthy farms . It is injected intramuscularly twice with an interval of 14 days. It is used in a dose of 10-15 ml for cows, 3-5 ml for sheep. Protektan drug, recommended by VIEV, is given orally 30 times before breast milk for 2 days, 10-15 ml with breast milk 5 times, and then 10 ml orally. In the conditions of Uzbekistan, the use of the vaccine prepared at UzNIVI is also beneficial .

Prevention. Gastrointestinal diseases of young animals occur mainly in farms with poor sanitary conditions and insufficiently balanced feed . Therefore, the main issue here is to increase the resistance of the animal organism along with the elimination of these factors. This event should start from the period of the animal's strait. It is necessary to provide balanced, nutritious feed and organize rations for strait animals . During this period , it is strictly forbidden to give nutritious products such as silage and fodder . It is desirable to organize a diet rich in vitamin nutrients, micro- and macroelements. Giving young animals ABK, PABK, ochko zon juice is one of the factors that increase the body's resistance. The sanitary condition of farms and current disinfection are the main factors in the prevention of gastrointestinal diseases in young animals.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .

4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention .

Topic: BIRD FLU DISEASE

Lecture	**Avian influenza _**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	*Results of educational activities:* Students: Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.ne

Influenza (Latin - Grippus avium; English - Infuenza; Russian - grippp) is a contagious viral disease of birds, characterized by septicemia, inflammation of respiratory and digestive organs.

Historical information. The disease was first described by Perronchito (1880) in Italy under the name "exudative typhus" of poultry. The disease later went by many names, including the "European plague" or "classical plague." Chentani discovered that the virus is a natural disease in 1901, and in 1955 this virus was called Influenza virus A, after that, from 1971, the disease was *found in poultry.* called *flu* .

Nowadays, *influenza* is rarely seen as a *classic plague , as influenza is caused by more low* - pathogenic strains. Scientists such as M.Tartakovsky, VN Syurin, NGOsidze conducted research on this disease in Russia.

The causative agent is an RNA virus belonging to the family Orthomyxoviridae, influenza genus Influenza virus A, type Hav-1. The size of the

virion is 80-120 nm. All known influenza viruses are divided into 15 subspecies by hemagglutinin (N) and 7 subspecies by neurominidase divided into antigenic groups. This viru0Cdam, swine and equine influenza viruses are genetically close to type A. Virus serogroups N_5 and N_7 are highly pathogenic for poultry. Virions develop and multiply well in 9-12-day-old chicken embryos and in cell culture. The virus has the property of hemagglutination of erythrocytes of several species of birds, mammals and humans. Virus-neutralizing, hemagglutinating and complement-binding antibodies are formed in the blood serum of recovered birds.

Exciter endurance. The virus is not resistant to high temperature, at 65-70 ^{0}C for 2-5 minutes, at low temperature, on the contrary, it retains its activity for a long time. For example, at 4 ^{0} C, the virus retains its infectious and hemagglutination properties for several weeks. The virus is kept active in a lyophilized state for 2 years at 4 ^{0} C, at - 70 ^{0} C for more than 5 years, and in frozen meat for more than 300 days. Disinfectants (2-3% caustic soda, formalin, 4% phenol, 5% hydrochloric acid) inactivate the virus in 5 minutes.

Epizootological data. Influenza is common among many species of domestic poultry and wild birds. A_1 virus isolated from chickens, pigeons and turkeys pathogenic for mouse, rabbit, guinea pig. Human and equine influenza virus *neuraminadase was detected* in virus strains isolated from poultry . This means that the gene pool of viruses isolated from birds contains the genes of other types of influenza viruses.

that the human influenza virus (A_2) can be found in domestic and wild birds, and vice versa, the avian virus can be found in humans. Wild and domestic poultry can simultaneously contain many types of animal (pig, horse) and human influenza viruses. The farm comes with virus, inventory, non-disinfected meat and egg trays. First of all, chicks, chickens with reduced resistance get sick with flu, especially when they are fed with poor feed, in a bad microclimate, when they are crowded together.

After the first passage, the virus can cause disease even in well-sanitary farms. Within 30-40 days, all susceptible birds are infected. In farm conditions, poultry are mainly affected by air and water. The virus is mainly inhaled through the respiratory and oral routes, and when it enters the mucous membranes of the digestive organs, it causes influenza. *The source* of the disease is *the sick* and recovered *virus carriers* (2 months). The virus is excreted from the body of sick and virus-carrying birds with all secretions, excreta and eggs.

Rodents, cats, wild birds, feed, water, inventory, and transport serve as a spreading factor of the virus. In natural conditions, the virus enters the body of birds mainly through respiratory organs and digestive mucous membranes. Once the virions enter the body, they spread throughout the body through the lymphatic system and blood. In flu, morbidity is 80-100%, mortality is 10-90%. It depends on the virulence of the virus, the resistance of the birds, their storage and feeding conditions. In this disease, 40-60% egg production is reduced, chickens can recover after 2 months. In most cases, influenza occurs as a result of reduced immunity due to Newcastle disease, infectious bronchitis, infectious laryngotracheitis and other

diseases. Seasons, age, sex, breed, and constitution of birds play almost no role in the occurrence of the disease.

Pathogenesis. Influenza has *respiratory* and *systemic forms* . At the point of entry into the body (respiratory organs), the virus multiplies in the epithelium of the mucous membranes. After 4-12 hours, the virus adheres to blood erythrocytes and spreads throughout the body and multiplies in parenchymal organs. As a result of the multiplication of the virus, toxins are formed, which poison the body and kill the birds. This usually happens when the flu *is acute* . If the disease is semi-acute or chronic, it lasts 10-25 days, and the outcome of the disease depends on the body's resistance. All virulent strains cause systemic disease. Hypoplasia of the lymph nodes, lymphopenia in the blood, and a decrease in the functions of the body's defenses are observed. This condition leads to viremia, and as a result of hemodynamic disturbances, hemorrhagic diathesis, exudative conditions are manifested.

Blood begins to leak from the walls of blood vessels, hyperemia and hemorrhages occur. Heart and body muscles undergo dystrophic, degenerative changes, and their functional activity is lost. As a result of intoxication, dystrophic, degenerative changes are also observed in the tissues of the spleen, liver, kidneys, lungs, gastrointestinal tract and brain.

Course, clinical signs and forms. The incubation period of the disease lasts 1-3 days, sometimes longer. The disease is severe in non-immune birds, which is almost 100% in young birds. The disease is *acute*, *semi-acute* and *chronic* . At the beginning of the disease, the feathers are ruffled, the eggs drop sharply, the sick bird is sad, it hangs its head, closes its eyes and stands on its feet, there is a decrease in appetite. The mucous membranes are reddened and swollen, stretchy mucous fluid flows from the half-open nostrils, the nostrils are clogged with hardened exudate. Some chickens have swollen ears, crowns and ears are black-brown due to intoxication and blood dynamics. Wheezing is observed during breathing, acceleration of its and pulses, increase of body temperature to 43 - 44 ^{0}C and drop to 30 ^{0}C before death are detected. The disease ends with death in 3-4 days. When influenza is caused by the A_1 virus serotype, mortality is usually 100%. If the disease is semi-acute or chronic, only 5-20% of birds die.

Due to changes in the nervous system, the legs and wings, neck are paralyzed. It hatches, becomes weak, closes its eyes, and sags. Rhinitis, conjunctivitis, keratitis appear, sticky mucous liquid flows from the nose. When the disease is in severe, acute form, shivering occurs, and the bird dies in 1-2 days.

In addition to respiratory symptoms, diarrhea is observed in them, the stool is gray-green liquid. They have ataxia, twitching, circling movement, clonic twitching of neck and wing muscles before death convulsions.

Pathologoanatomical changes. Pathologoanatomical changes are different depending on the duration of the disease. The crown, earrings turn blue and swell, mucous fluid flows from the beak, bursitis appears, and joint deformation is formed. Inflammation and hemorrhagic diathesis (hemorrhages) in the mucous membranes of the nose, larynx, and larynx, and swelling under the skin of the throat, larynx, chest,

and legs are observed. When air accumulates in the gills and the food is not digested, it becomes old and stagnant, and an unpleasant smell comes out when it is used. Under the skin, in the lungs, heart and other parenchymal organs, in the mucous membranes, gross or punctate hemorrhages are detected. Rhinitis, pharyngitis, conjunctivitis are observed in 45% of cases, bleeding in the stomach and intestines is observed in 60%. Blood fills the lungs and swells a little, and pneumonia foci appear. In all cases, gastroenteritis, peritonitis, pericarditis, bronchitis, aerosacculitis, swelling in lungs, blood filling of internal organs, bruises of muscles are observed. Hemorrhagic meningitis, diffuse hemorrhages are observed in the brain. In histology, necrobiotic foci are observed in all sections of the brain.

Diagnosis. Clinical, epizootological data (presence of recently purchased poultry or eggs, a sharp decrease in egg production in chickens, disinfection of cages, when was the last time poultry was slaughtered for meat, proximity of the farm to a neighboring farm, access to it by wild birds, transport to the farm , the entry of strangers, the clinical condition of existing birds around the hearth, mass disease, their number, the number of dead, the rate of spread, whether the birds in the farm and the surrounding area are vaccinated against this disease) and the diagnosis based on pathologoanatomical changes is the initial diagnosis .

The final diagnosis is based on laboratory tests. To do this, in the acute stage of the disease, pieces of lungs, heart, liver, brain, and other organs are sent to the laboratory quickly (within 1-2 hours) or in 50% glycerol in a thermal suitcase with a referral letter, put ice around it. and sent to the laboratory. After injecting the material into the allantois cavity of 9-12-day-old chicken embryos, after 48 hours, the allantois liquid is checked for the presence of virus in GAR, NR, GATR. For biotesting, a suspension prepared from sterile pathological material is injected into the muscle of a 2-4-month-old chick in the amount of 0.5-1 ml. After 3-5 days, the chicks get sick and die, if the virus is weak, the blood of the chick is checked for antibody titer in GATR, PR, KBR on the 5th and 15th days. In one of these reactions, a 4-fold increase in the antibody titer in the second blood serum compared to the first blood serum indicates influenza. Virus antigen is detected in the pathological material by the IFT method. Antibodies against this disease, IFT or IDR, are detected in the blood serum of poultry. Using PCR, the type of virus antigen is determined in the material.

Differential diagnosis. It is necessary to distinguish the septic form of the disease from Newcastle disease, infectious bronchitis (IB), infectious laryngotracheitis (ILT), pasteurellosis, mycoplasmosis and other respiratory diseases. In all cases, special laboratory tests (virological, bacteriological, serological, biotest) make it possible to distinguish this disease.

Treatment. A special treatment method has not been created, so that the pathogen does not spread, the birds are immediately killed and destroyed (burned) in a mandatory bloodless method.

Immunity. Immunity lasts for 6 months in chickens that have recovered from the flu. Liquid and dry inactivated GOA embryo vaccines against type A_1 virus are used for special prevention . Vaccines are used for prophylactic purposes or for 45-day-old chicks in areas with risk of this disease. After 14-21 days, immunity is

formed and it is preserved for 6 months. His titer is checked at GATR. If the titer in GATR is not less than 1:10 in 80% of birds, immunity is considered sufficient.

Prevention and countermeasures. It is necessary to achieve compliance with zoohygienic standards and zooveterinary requirements when placing birds according to their age. A building where poultry is not kept must be cleaned, disinfected 3 times and sanitized.

Regarding this disease, in a healthy (incubating egg-producing) farm, regular disinfection of transport and cages, veterinary-sanitary rules are required. Poultry are vaccinated prophylactically with influenza vaccine. It is necessary to take measures not to bring pathogens to the farm and not to contaminate the farm with viruses, not to allow other animals to walk on the farm. There is a need to educate the public about influenza.

Eggs for incubation are taken from a flu-free farm, and chicks are kept in a separate building. At the age of 45 days, they are vaccinated with an inactivated vaccine. Feathers from conditionally healthy birds are dried at 85-90 ^{0} C for 15 minutes or disinfected with 3% formaldehyde at 45-50 ^{0} C and then dried. The method of keeping the farm always clean is the least controversial and the most effective, proven by many years of experience. This event protects against several pathogens at the same time. Separating groups of birds from each other (contact) also helps prevent disease. Carrying out regular disinfection, disinsection and deratization activities, keeping poultry at the level of zoohygienic standards and feeding them with vitamin feeds will increase their natural resistance and help prevent this disease. Vehicles entering the farm must be disinfected. All kinds of wild birds should not be allowed to fly into feed stores and warehouses. After the hatching of each batch of chicks, it is necessary to carry out disinfection and study the epizootic status of the surrounding farms regarding this disease. It is required not to allow strangers to enter the farm, not to mix poultry inside the farm without the permission of a veterinarian, not to remove poultry, eggs and other poultry products, as well as tools, equipment, and manure from it.

Laboratory diagnosis of influenza on a farm or settlement among poultry upon detection, the poultry farm or settlement is *quarantined by the decision of the district (city) mayor based on the certificate of the chief veterinarian of the district* .

According to quarantine requirements, sick birds are destroyed and those suspected of disease are slaughtered and thoroughly disinfected. If pathologo-anatomical changes are observed among the slaughtered birds (peritonitis, hemorrhages in the chest cavity, bruising of the muscles), the slaughtered carcasses and waste are lost. If there are no pathologoanatomical changes, the internal organs are removed, and the carcass is boiled and eaten. Eggs of sick chickens are destroyed by boiling.

Healthy birds in the risk area are vaccinated. Poultry are kept in a closed building, their waste is burned. Unaffiliated persons (even family members or relatives) and motor vehicles are not allowed on the farm. Low-value items are burned, the rest are disinfected with 1.5% caustic sodium, creolin, formaldehyde. Markets close.

The chief veterinarian of the district appoints an epizootologist to be responsible for the organization of all measures and elimination of the disease in the center of the disease, and gives written information to the governor about the formation of a special commission for the prevention of the spread of the disease and its elimination. It also informs the neighboring districts and higher veterinary organizations and the employees of the district sanitary-epidemiological department about the outbreak of *influenza* . Poultry is kept in a closed building and their products are not released. The building where sick birds are kept is cleaned of garbage, washed and regularly disinfected. Dead birds are immediately cremated.

Wild birds and animals (dogs, cats) are not allowed to enter the farm. Hot (70-80 ^{0} C) 3% caustic sodium (exposure - 3 hours), 2% formaldehyde, 3-5% active chlorine lime, 1% acetic acid are used for disinfection (aerosol disinfection 15 -20 ml/m ^{3}).

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention .

Topic: PARRANDA N'HIGH DISEASE

Lecture	**Newcastle disease of poultry**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance . _ _ _ Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	

Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	*Results of educational activities:* Students: Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.
Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students.	3.1. He listens and clarifies. 3.2. Writes.

	3.2. Homework is assigned and graded.	

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.
2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.
2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.
3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.
4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.
5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".
6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz
3. www.sea@mail.net21
4. www.veterinariy.actavis
5. www.fvat@.academy.uzsci.ne

Newcastle disease is a highly contagious, acute infectious disease caused by a virus, characterized by septicemia, damage to the central nervous system, gastrointestinal and respiratory organs.

Economic damage. When the disease is acute, when the period of immunization is violated, young chicks die up to 100% when it is first recorded. Mother hens also die around 70-100 percent. In addition, a large amount of vaccine is required to carry out vaccination work. Quarantine measures also require a lot of money. All this causes huge economic damage to poultry farms.

Trigger. Paramyxovirus, polymorphic virions-filamentous, rod-shaped, racket-shaped virus. It agglutinates erythrocytes of humans, cattle, pigs and horses. It is very resistant to external environmental conditions, can be preserved at low temperatures. Frozen chicken can live up to 6 months, and up to a year at 20°C. It dies in 30 minutes at 65-70°C, and in one second at 100°C. It can survive for months at an ambient temperature of 18-20 ° C . Chicken feathers can be kept alive for 18 days, and eggs can live for years in the refrigerator. The virus develops well in 9-10-day-old chicken embryos.

Epizootology. Chickens are the first to be susceptible to the disease of Nyukasla. Turkeys also get sick. But this type of bird does not always show the disease in a typical state. In natural conditions, all birds, regardless of age and breed, get sick. The disease occurs at any time of the year. Sparrows, parrots, mallards are more susceptible to artificial diseases. Viruses enter the body in different ways. The main ones are the respiratory and alimentary tract. When the virus enters the body , it passes through the mucous membranes into the blood, and then causes disease. The disease is mainly transmitted through imported poultry, feathers or eggs from unsanitary farms. Sometimes it can be transmitted by virus carriers on the farm itself, as well as through facilities that isolate the virus. Sick poultry is the main source of the pathogen. The disease is transmitted through direct contact and infected objects. Infected eggs are extremely dangerous for healthy birds around them during disease outbreaks. Feed products stored in unhealthy farms are also factors that cause the spread of disease.

The disease becomes an epizootic and causes a huge loss to the farm where it was first recorded. Within 2-5 days, up to 70-100 percent of birds are infected. If it is recorded in private farms, it will pass from one village to another village or yard, and the chickens will be "exterminated".

Pathogenesis. When the virus enters the body, septicemia occurs. It spreads to organs and releases toxins in the process of development. This, in turn, affects the wall of blood vessels and causes an inflammatory-necrotic process. Blood begins to seep out, hyperemia and hemorrhage occur. Heart muscles undergo dystrophic-

degenerative changes, and its activity is disturbed. In addition, damage to the spleen, liver, kidney, gastrointestinal tract and brain is observed.

Course and clinical signs. The latent period of the disease can last 1-5 days, sometimes a week. Manifestation of clinical signs in cancer mainly depends on the presence of immunity in the body. In non-immune birds, the disease is severe. This condition is especially strong in young chicks. They become completely exhausted, it becomes difficult to breathe. When he breathes, he opens his mouth and stretches his neck, making a unique sound. After the onset of the disease, the loss in the first 4-5 days is strong and can reach 80-100 percent. After 8-10 days, it will decrease. Nervous system disorders are observed, tremors occur in the head, wings and legs are paralyzed. The neck is bent back, sometimes bent to the chest (pictures 22-23). Older chickens also have the same condition as above, and those who have not been immunized are very sick. The number of eggs decreases sharply. He gets extremely weak, closes his eyes, squats down, and sleeps. Therefore, in some villages, this disease is called "chonkaymish". Does not seek food. As if yawning, nervousness and diarrhea. In non-immunized birds, cases of diarrhea and difficulty breathing are observed. The inside is greenish, mixed with blood. Wings and legs are paralyzed, death rate is 90-100%.

Pathologoanatomical changes. In acute cases, blood can be poured into the mucous membranes without actual pathologoanatomical changes. In acute cases, the crown becomes bluish, and the skin hemorrhages are visible. There will be severe bleeding in the mucous membranes, swelling of the subcutaneous tissue of the head, neck, and chest. When air accumulates in the gills and the feed is not digested, it wears out and stops, and an unpleasant smell comes out when used. Heavy bleeding is observed at the border of the muscle and the stomach. In the lymphoid follicle of the intestine, blood is also poured under the epicardium. The spleen becomes slightly smaller. The lungs are filled with blood, slightly swollen, and foci of pneumonia appear. Cerebral blood vessels swell, and blood flow to the brain is observed.

Diagnosis. Epizootology, clinical signs and pathologoanatomical changes of the disease are taken into account to make a diagnosis of Nyukasla disease. In addition, the virus is isolated and identified in laboratory conditions. This process is performed with special hyperimmune blood serum using RTGA. To isolate the virus, a 10% suspension is prepared from the spleen or brain of sick or dead birds. It is rotated in a centrifuge that rotates 1500 times per minute and is kept for one hour. Then, adding 1000 TB of penicillin and 1000 mg of streptomycin to the suspension, 9-10- day-old embryos are infected. After 72 hours, the presence of hemagglutinin with RGA, the level of immunity with RTGA is determined using the RTGA method with a diagnostic antigen (any vaccine used against Newcastle disease is possible). After 5-6 months after immunization, the high titer of the antibody in the blood serum indicates the presence of the field strain of the virus in the body.

Differential diagnosis. It is necessary to distinguish between infectious bronchitis and laryngotracheitis. One of the diagnostic signs in infectious bronchitis is the detection of virus-neutralizing antibodies. RN is put for this. Chickens that have recovered from bronchitis do not become infected with Newcastle disease. In some

cases, Newcastle disease can be accompanied by bronchitis. To distinguish laryngotracheitis, attention is paid to clinical signs, its virus is isolated and identified. It is differentiated from pasteurellosis by microbiological examination methods.

Immunity. The following vaccines are used to prevent the disease.

- Dry virus vaccine prepared from strain V1 (B-prim). Healthy farms are vaccinated with this vaccine at 15-20, 45-60, 140-150 days, then once every 4 months. One ampoule is dissolved in 50 ml of distilled water and used by intranasal and aerosol methods. Two drops are instilled into the nose with a pipette.

- Dry virus vaccine prepared from "La Sota" strain. The vaccine is administered intranasally in healthy and unhealthy farms. In healthy farms, vaccination is carried out at 15-20, 45-60, 140-150 days, in unhealthy farms at 10-15, 35-40, 120-140 days, then every 6 months. When administering intranazol, one ampoule of vaccine (500 nazol doses) is dissolved in 50 ml of distilled water. Water is drunk before instilling 2 drops of vaccine into the nose. Chickens can be given water 1.5 hours after vaccination.

When drinking with water, 500 nazol doses of the vaccine are dissolved in 1 l of distilled water and 5 ml are given to each chick for 2 days in the morning. It is forbidden to give food and water 6 hours before vaccination. After immunization, it is allowed to give food and water after 1-1.5 hours. Immunity appears after 7-8 days.

- Dry virus vaccine prepared from strain "N". The vaccine is live and is released in an ampoule. It is applied to adult clinically healthy chickens. 1 ml of vaccine (0.5 ml virus mass) is dissolved in 500 ml of sterile physiological solution. The solution is injected between the muscles of the chest area in the amount of 1 ml. Immunity appears after 48 hours and lasts for a year.

Prevention. Poultry farms are allowed to bring only poultry and eggs from healthy farms. It is necessary to disinfect vehicles entering poultry farms. All kinds of wild birds should not be allowed to fly into feed stores and warehouses. Disinfection is carried out when each batch of chicks is hatched. The epizootic status of Newcastle disease in the surrounding farms is monitored and studied. Vaccination must be carried out according to the plan. When the disease appears, the diagnosis is made immediately and quarantine is announced. It is strictly prohibited to open and sell chicks, give poultry to other farms, and release eggs under quarantine conditions. All clinically sick birds are destroyed.

Veterinary specialists in poultry factories, state, collective and other farms and state veterinary institutions must perform the following tasks:

- implementation of special veterinary measures (vaccination for disease prevention, diagnostic tests) in poultry farms, settlements and regularly checking the condition of poultry;

- To control the emergence of immunity in vaccinated poultry in farms where vaccination was carried out for the prevention of measles.

When there is a suspicion of the occurrence of Nyukasla disease, heads of farms (poultry factories, state, collective farms, enterprises and veterinary specialists or owners of poultry in private farms) should perform the following tasks:

- not to allow outsiders to enter the farm, not to mix poultry inside the farm, not to remove poultry, eggs and other poultry products, as well as tools, equipment, manure from it;

- farm (population) about the occurrence of the disease point), immediately inform the veterinary specialist of the institution and the chief veterinarian of the district.

The veterinary specialists of the farms and state veterinary institutions take measures to immediately detect the disease and send the blood serum obtained from newly dead birds (3-5 heads) and sick birds (10-20 heads) to the veterinary laboratory for examination.

The diagnosis of Nyukasla disease is made on the basis of epizootological data, clinical signs, pathologo-anatomical changes and laboratory examination results.

After receiving information about the outbreak of the disease, the chief veterinarian of the district:

- immediately go to an unhealthy farm, settlement, eliminate the epizootological focus and identify the source of infection;

- in necessary cases appoints a veterinarian (epizootologist) responsible for the organization and removal of all measures in the place where there is an outbreak of the disease;

- within one day, he sends materials to the district authorities that a quarantine has been announced in an unhealthy farm (settlement), and that a special commission has been formed for the prevention and elimination of the spread of the disease;

- at the same time, the veterinarians of the neighboring districts and higher veterinary organizations are informed about the occurrence of Newcastle disease.

If the disease occurs in cities, individual streets, quarters, or the entire city are quarantined. The following are prohibited in farms and settlements quarantined for Newcastle disease:

- Removal of poultry resistant to Nyukasla disease from buildings;

- access of outsiders to poultry farms;

- sale and export of poultry and products. The following are conducted in unhealthy farms (poultry factories, farms) with regard to the disease of Newcastle disease:

- if a disease occurs in young birds, all sick and healthy chicks are killed by a bloodless method, lost or sent to slaughter. The remaining clinically healthy birds are slaughtered for meat or vaccinated against the disease. These birds are kept in a separate place and slaughtered for meat 2 weeks before the quarantine is lifted;

- eggs obtained during the quarantine period are boiled for at least 10 minutes and used for feed inside the unhealthy farm.

Unhealthy poultry farms and factories are subject to mechanical cleaning and disinfection. Manure and bedding are biothermally neutralized.

Quarantine is canceled 30 days after the destruction of the last sick bird in the unhealthy farm, the settlement, and after full sanitization of farm premises and buildings. Slaughter of poultry for meat is carried out with the permission of the veterinary department and, if deemed necessary, is carried out in special places. All

measures to combat the disease and eliminate it are carried out according to the manual.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.
6. Immunity .
7. Treatment and prevention

Topic: DISEASES OF DOGS AND FUR ANIMALS

Lecture	**Plague of dogs and fur animals**
Lecture teaching technology	
Study time: 2 hours	of students: 90 person
The form of training	Enter. Visual lecture
Lecture plan Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	About the disease, causes of origin Clinical signs. Pathogenesis, diagnosis, comparative diagnosis.
The purpose of the training session: to give students the correct ideas about the subject of study	
Pedagogical tasks: Disease causative agent and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.	*Results of educational activities:* Students: Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.
Educational methods	Lecture. Brainstorming.
Organizational form of education	Mass, collective.
Educational tools	Text. Table . Video projector. Computer.
Educational conditions	Specially equipped lecture hall.

Monitoring and evaluation	Verbal request. Quick survey.

Technological map of the lecture		
Lecture stages	*Activity content*	
	Educator	*Learner*
1. Introduction to the lecture (10 minutes)	1.1. Subject. Expected results from it. 1.2. Announcing the plan.	1.1. He hears and writes. 1.2. Asks questions, clarifies.
2. Main part (60 minutes)	*2.1. Quick Q&A:* Planning objects. Pathogenicity and resistance. Epizootology Pathogenesis, course and clinical symptoms Pathologoanatomical changes Diagnosis and differential diagnosis. Immunity. Treatment and prevention.. *2.2. The main theoretical parts of the lecture are described using visual materials.*	2.1 . He listens, expresses his understanding, thinks, answers. 2.2. He listens, discusses the content of the tables and writes down the main points.
3. Conclusion (10 minutes)	3.1. It concludes the lecture and focuses students' attention on the main issues, encouraging active students. 3.2. Homework is assigned and graded.	3.1. He listens and clarifies. 3.2. Writes.

Key words: epizootic process, epizootic, prevention, sanitation, disinfection, immunoreactivity, infection, protein, reservoir, zoonosis, zooanthroponosis, anthroponosis, susceptible organism, alimentary infection, transmissive route, horizontal route, vertical route , sporadic, panzootic, pandemic.

Main textbooks and training used list of applications

A social literature

1. Salimov XS, and others "Epizootology and infectious diseases" textbook 2022. F. Nasimov publishing house.

2. Salimov XS, Kambarov AA "Epizootology" textbook, 2016. F. Nasimov publishing house.

Additional literature

1. Mirziyoyev Sh.M. Study and dissemination of his speech at the 75th session of the United Nations General Assembly. Study guide. Tashkent, "Spirituality" NMIU, 2021. - 280 pages.

2. Mirziyoyev Sh.M. Let's live freely and prosperously in the new Uzbekistan. Tashkent, "Tasvir" publishing house, 2021. - 52 pages.

3. Mirziyoyev Sh.M. Humanity, goodness and creativity are the foundations of our national idea. Tashkent, "Tasvir" publishing house, 2021. - 36 pages.

4 . Mirziyoyev Sh.M. New development strategy of Uzbekistan . Tashkent, "Uzbekistan " publishing house, 2022. - 416 pages.

5. Decree of the President of the Republic of Uzbekistan dated March 28, 2019 No. PF-5696 "On measures to radically improve the state management system in the field of veterinary medicine and animal husbandry".

6. Resolution PQ-187 of the President of the Republic of Uzbekistan dated March 31, 2022 "On the fundamental improvement of the personnel training system in the field of veterinary medicine and animal husbandry" .

Foreign literature

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

Websites :

1 . www.Ziyo.net.uz. _

2. www.vetjurnal.uz

3. www.sea@mail.net21

4. www.veterinariy.actavis

5. www.fvat@.academy.uzsci.ne

Plague of carnivorous animals (Lat. - Pestis carnivorum; English - Canine distemper; Russian-chuma plotoyadnix, bolezn Carre; Uzb. - Carre's disease) is an acute infectious contagious disease, characterized by fever, inflammation of the mucous membranes of the digestive and respiratory organs, pneumonia, conjunctivitis, redness of the skin and damage to the central nervous system.

Historical information. This disease has been known since the domestication of dogs (10,000 BC). Aristotle described it as angina. In 1905, Fr. Scientist Carré determined. However, Dunkin and Laidlaw proved in 1926 that this virus causes disease in dogs.

In the former Union in 1932, I. Mirolyubov recorded plague in dogs. VA

Pankov in 1938 identified plague in blackbirds and foxes. NV Syurin, VA Pankov and S. Ya. In 1953-1957, the Lyuboshenkos publish the results of research on this disease in the press. Currently, plague of carnivores is found in many countries of the world, including Uzbekistan.

Trigger. RNA virus, size 115-160 nm. Belongs to the family *Paramyxoviridae* and genus Morbillivirus. It is *antigenically* and *immunobiologically* close to the human *measles* virus and has a one-way *antigenic relationship with the rinderpest virus* . A dog infected with human *measles virus will not be infected with plague, or a dog infected with plague will have measles antibodies in its serum.* In addition, the serum of plague-infected cattle neutralizes the canine plague virus.

Exciter endurance. Resistant to external influences. In nasal fluid and dog feces, the virus loses its activity in 7-11 days. In the external environment, it is kept active for 7-11 days, in the blood for 4-14 days, in the spleen, for up to 2 months. In the internal organs of infected animals, it can survive up to 6 months at minus 20 ^{o} C.

The virus can be kept active for several months in a dried or frozen state. It lasts a long time in the cold. At 60 ^{0} C in 30 minutes, at 100 ^{0} C it dies immediately. Resistant to disinfectants. 1% lysol for 30 min., 2% caustic sodium for 60 min. and 0.1-0.5% formalin, phenol and sunlight inactivate in a few hours.

Economic damage. Fur farms will suffer huge economic losses from the plague, because fur is only sold in foreign currency on the world market. Mortality is 10-50 percent. In special dog breeding and breeding farms, the death rate is 60-90 percent. Domestic dogs bought for several thousand soums will die 100% if they are not vaccinated. In addition, a lot of money is spent on quarantine measures.

Epizootological data. All carnivorous animals are susceptible to the virus: dog, wolf, black wolf, hyena, fox, northern fox, enot, skunk cousin, black cousin, marten, white marten, beaver, badger. Among them, the most susceptible are enot, sassy cousin and relatively more resistant marten. Young animals are more susceptible, 12-month-old dogs, 5-month-old fur animals are 2-5 times more infected than adults. From laboratory animals: a field mouse, a cat dies from an acute disease in 10-14 days.

The extent of the spread of plague depends on many factors, especially immunity - the vaccination of dogs against this disease. The disease is sure to spread in places where dogs are often present. Plague mainly affects carnivores under the age of one year. Young lactating animals are less likely to get sick, because they receive colostral immunity through mother's milk. Young carnivores are prone to disease if they are born to unvaccinated or unvaccinated mothers. Colostral immunity reaches two weeks, then young lactating animals need to be vaccinated. Susceptibility to the disease and its course depends on a number of factors. If feeding and feeding are not fully balanced, if the living conditions are not at the required level, the resistance of the carnivorous organism will decrease. This allows the disease to occur. As a result, the disease worsens. Frequent weather changes, windy, rainy-snowy days in fur farms lead to an increase in non-infectious catarrhal diseases among animals. This, in turn, increases susceptibility to plague. The spread of this disease accelerates when animals are left to escape in fur farms and the veterinary-

sanitary conditions are out of track.

The source of the disease is mainly sick, recovered and *virus-carrying* animals. The virus is usually released for 10-51 days with the tears, nasal fluid, breath, excrement of animals and other body excreta.

In pathological materials (blood, spleen, marrow, pleural and abdominal fluids), the virus is kept at a high titer for a long time. Recovered dogs shed the virus for 3 months, otters for 9 months, and other fur animals for 3-5 months. Damage occurs mainly by *respiratory* and *alimentary routes.* The virus can spread up to 12 meters through the air.

Materials used to enrich the dog, special clothes of caretakers, food, poultry, arthropods, and rodents can serve as factors that transmit the virus.

In nature , *the reservoir of the virus* is wild animals (raccoons) and wild dogs. The virus comes to dog kennels mainly with dogs brought from unhealthy places and with infected dogs or objects infected with the virus. Rats and mice, blood-sucking insects and birds play an important role in the transmission of infection. The disease can spread epizootically or sporadically in all seasons of the year. In fur farms, 70-90% of young fur animals can die from plague.

Secondary infections, helminthiasis lower the immunobiological status of the body, increase its susceptibility to plague, aggravate the course of the disease.

Manifestation of the epizootic process in carnivore plague is not the same, it depends on many factors, it depends on the age, species of the animal, susceptibility and resistance to the virus, virulence of the virus, natural-geographical and economic conditions. However, *the index of contagion* in this disease is high enough (70-100%).

Pathogenesis. The virus enters the body through the mucous membranes and enters the blood, which causes an increase in temperature, weakness, inflammation of the respiratory system, eyes, stomach, intestinal mucous membranes. The virus is inhaled on the first day and enters mononuclear lymphoid cells in the digestive organs, and remains in mononuclear leukocytes for 2-3 days. Within 6 days, the virus spreads throughout the body. When the level of immunity in the body is low, the virus enters all epithelial cells and multiplies. During this period, pathological changes are observed in parenchymatous organs, head and spinal cord. If the organism is resistant, antibodies are intensively secreted and the virus does not spread throughout the organism, but only in the epidermis and nervous system. The antibody titer is usually very high for 9-12 days, and the same condition lasts for 60-70 days.

In the pathogenesis of the plague, secondary infections: salmonella, escherichia, pasterella, toxoplasma and cocci are of primary importance.

B *ordotella bronchiseptica* has been the etiological agent of plague for 50 years counted. Plague can be accompanied by infectious hepatitis or adenovirus infection in dogs, and Aleutian disease in black dogs. Mixed infections, helminths, avitaminosis reduce immunity, make the disease more complicated and increase mortality.

Clinical signs and forms of the course. The latent period of the disease lasts 14-21 days and more in dogs, 9-30 days in fur animals, and in some cases 90 days. Depending on the manifestation of clinical signs, *pulmonary, intestinal, nervous, skin*

and *mixed forms* are distinguished. In what condition the disease manifests itself depends on the reactivity of the organism and the virulence of the pathogen. Therefore, a virus belonging to the same type can manifest a disease with several different clinical conditions (from fever to nervous system disorders). Therefore, it is difficult to make a diagnosis in some cases.

In dogs, the disease begins with an increase in body temperature of 39.5-40 ^{o}C, and this condition is maintained for a long time. In 1.5-month-old dogs, it may show an atypical appearance and may not have a fever.

According to the development of clinical signs of the disease, *lung, intestinal, skin, nervous and mixed forms* are observed. It depends on the reactivity. In the dog, lethargy, tremors, loss of appetite, and inflammation of the mucous membranes of the eyes are observed. Symptoms of rhinitis, tracheitis, and bronchitis appear as a result of the multiplication of the disease virus in the lungs. He breathes faster, up to 80 times per minute. A rise in temperature indicates catarrhal inflammation in the lungs. As a result of the multiplication of the virus in the intestine, vomiting, constipation, and constipation are observed, and then it alternates with diarrhea, mucus mixed with blood is separated in the feces, and the smell is noticeable when used. As a result of the reproduction of the virus on the skin, pustular rashes are observed in hairless areas - the abdomen and inner thighs. Then the nodules burst and a bluish substance is released from them. These nodules are quickly replaced by a wet wound followed by a dry crust. Although the proliferation of the virus in nerve cells is not significant, it plays an important role in the diagnosis of the disease.

As a result of weakness, timidity and increased sensitivity in sick animals, there is a sudden fainting and, in the last stages, tension in certain groups of muscles. Disruption of movement function and *paralysis* in some animals will escalate. Most often, plague passes in a mixed form, and when the disease is acute, dogs die in a few days.

The course of the disease can be *acute, semi-acute, chronic* and *abortive* . *When* the disease is *acute , the temperature* rises to 41-42 ^{0} C, loss of appetite, a state of coma is observed, and the dog dies in 2-3 days. This form is rare.

Semi-sharp ***the temperature*** stays at 41-42 ^{0} C for 1-2 days, in some cases 1-2 weeks . Then the body temperature can rise and fall. During the period of fever, lethargy, depression, muscle spasms, tremors, fear, dryness of the nasal mucosa and loss of appetite are observed in the dog. Young dogs may not have a fever. After 2-3 days, serous-mucous, then purulent liquid flows from the nose. Then it dries and closes the nostrils. The dog coughs, he kicks, sneezes and scratches his nose with his foot. His breathing is fast, heavy and wheezing. Moist wheezing is heard in the lungs. Hardened foci are revealed when the lungs are percussed. The pulse accelerates and is arrhythmic *(pulmonary form of the disease).* Serous, mucous and purulent tears flow from the eyes, they harden and clog the eyelashes. The conjunctiva is reddened, swollen, afraid of light. In some cases, conjunctivitis turns into keratitis and ulcers.

In some dogs, along with inflammation in the respiratory tract, constipation, vomiting, mucous, bloody diarrhea are observed due to catarrhal inflammation in the digestive tract *(intestinal form).*

Rashes with small red spots appear on the skin of the mouth, nose, lips, around the ears and on the skin of the abdomen, the inside of the legs, in the groin area. Later, blisters appear in their place, the size of a mush, and the largest ones are the size of a dime coin, their surface is shiny, and there is yellow liquid pus inside. Later, these blisters burst, dry and become covered with brown crusts. Exudative eczema also occurs around the ear (*skin form).* In some dogs, where the joints bend, the upper part of the skin undergoes severe hyperkeratosis (corneal coating).

nervousness turns into nervousness, muscle tension is observed, movement coordination is disturbed. *Epileptic seizures,* later in the legs, bladder and rectum and *paralysis* of the facial nerve is observed (XXVI - picture) .

Chronic pain more often seen in the form of nerves. In this case, muscle spasms, premature paralysis and paralysis, blindness and deafness, inability to speak, and long-term, in some cases, permanent scarring of the eye. The disease can last from 21-28 days to several months. Death - 50%. It can be **85%** or more in nerve form .

Abortive form - gets better after 1-2 days of discomfort.

In foxes, the skin form is not observed at all, the nerve form increases at the end of the epizootic (Fig. XXVII) . The skin of the paws, eyelids, nose, lips and ears swells in blackheads, it cracks and forms a crust. If the disease is prolonged, i.e. chronic, dermatitis reaches the neck and back.

Pathologoanatomical changes. Pathologoanatomical changes depend on the forms of this disease. Catarrhal or purulent inflammations in the upper respiratory organs, red-gray or red-brown hardened atelectasis foci in the lungs; hemorrhages under the epicardium, pericardium and kidney capsule and in the bladder; hemorrhages, erosion and ulcers are observed on the mucous membranes of the digestive organs. Similar changes are detected in the duodenum and rectum. Lymph nodes in the chest and abdomen are enlarged, watery when cut. The cerebral cortex is full of blood vessels.

Diagnosis. Many symptoms of the disease make it difficult to diagnose animals in time. Diagnosis is based on epizootological, clinical and pathologoanatomical data and *laboratory tests* . The following 6 symptoms are taken into account when making a diagnosis based on clinical signs: diarrhea, inflammation of the respiratory tract, mucous membranes of the eyes and nose, hyperkeratosis on the palm, damage to the central nervous system and the duration of the disease for at least 3 weeks. Plague is suspected if **4 of** these **6** symptoms are present.

The following 5 symptoms are of great importance in the first onset of the disease: cough, photophobia, increased temperature in the absence of appetite, normal temperature in the case of good appetite, and nervous symptoms. Plague is suspected if 2 of these 5 symptoms are present. In a complicated situation, 30-45-day-old dog, fox cubs or skunk-cousin, black-cousin are infected with pathological material (blood taken during fever or suspension of parenchymatous organs) and a biotest is performed.

KBR, IDR, NR are used for diagnosis. To identify the virus, NR is applied - 5% special serum is added to the cell culture infected with the virus and it is known by the result of the neutralization reaction.

Differential diagnosis. Canine plague should be distinguished from: leptospirosis, infectious hepatitis, infectious encephalomyelitis of foxes, rabies, Aueski, pasteurellosis and salmonellosis. Death in leptospirosis occurs in 2-3 days. Infectious hepatitis occurs mainly in young puppies, in which stationary and enlarged liver is observed, encephalomyelitis in foxes. Paralysis of the throat, lower jaw, and legs is observed in rabies. In Aueski, the animal dies on the day it becomes ill. Salmonellosis occurs in puppies of 1-2 months and is acute and keratitis is not observed. In all cases, the final diagnosis is based on laboratory tests.

Treatment. Polyvalent hyperimmune blood serum or gamma-globulin used in human measles is used for treatment. Broad-spectrum antibiotics, sulfonamides, and nitrofurans are used against secondary and mixed bacterial infections. Symptomatic and stimulating drugs are used. Hyperimmune blood serum or gamma-globulin drug is injected intramuscularly based on the "Instructions" for their use. Interferons also have very useful therapeutic effects. It is recommended to use three groups of drugs at the same time: antibacterial (against secondary infectious agents), symptomatic (to reduce fever, restore heart function, as well as suppositories), stimulating (group V vitamins, cocarboxylase, ascorbic acid, etc.). From antibacterial drugs, penicillin-10000 TB intramuscularly 3-4 times a day, streptomycin - 1 kg/10-20000 TB, sulfadimezin, sulfadimethoxine, norsulfazol 0.5-1.0 3-4 times a day give good results. Phthalazol, levomycetin and enteroseptol 0.25-0.5 g/kg are given 3-4 times a day against gastrointestinal disturbances. 2-3 ml of 20% caffeine, 0.5-1.5 ml of 20% camphor are injected under the skin. 5 ml of calcium gluconate intravenously, 1 ml of 1% diphenhydramine is recommended. 1 ml of 0.05% Proserin is injected under the skin for 10 days against paralysis.

For conjunctivitis, the eye is washed 2-3 times a day with boric acid solution. If the plague passes with the appearance of exanthema on the skin, the skin is treated with 5% iodine tincture or sprinkled with drying powders (bismuth, zinc oxide and talc). If the plague occurs in the nervous form, it is necessary to use complex powder preparations (0.05 luminal, 0.25 glutamic acid, 0.03 diphenin, 0.05 spasmolitin, 0.015 caffeine). It is taken as a powder 2-3 times a day for 15-30 days. If the muscles are paralyzed, massage is recommended, physiotherapy is used, and strychnine 0.001 is injected under the skin. To maintain the normal activity of the central nervous system, 1 ml of cerebrolysin is injected intramuscularly once a day for 30 days. Diet is one of the main treatments. During this period, it is good to boil minced meat, liver, cheese, and rice. It is necessary to have B group vitamins in dog food. Rooms should always be ventilated. It is recommended to turn sick animals in the open air from time to time. For disinfection, 2% caustic soda, 5% active chlorine lime solution, 5% lysol, 2% chloramine are recommended for disinfection.

Immunity. Dogs who recover from the disease develop lifelong immunity. In vaccinated dogs, immunity is formed in 10-21 days and lasts for 1 year. Young

puppies born from vaccinated dogs are resistant to distemper virus for 28 days due to colostral immunity.

To protect carnivores from plague virus, 3 dried virus vaccines are used: 668-KF, EPM and VAKCHUM. When the dogs are one month before giving birth, the puppies are vaccinated from 2 months.

Prevention and countermeasures. General prevention consists of the following: it is strictly forbidden to bring dogs and fur animals from an epizootic unhealthy farm. It is allowed to take only from healthy kennels and farms for this disease. After they are brought, they are kept in preventive quarantine for 30 days. Dogs brought to the exhibition are first vaccinated against plague and then allowed. General veterinary-sanitary measures, animal care at the level of zoohygienic standards increase the effectiveness of active immunization. Only healthy animals are vaccinated. Vaccines are used based on the "Instructions" for their use.

If the plague is confirmed by the laboratory, the kennel, settlement or farm is *quarantined by the decision of the governor based on the certificate of the chief veterinary inspector of the district* . Sick animals are immediately transferred to a warm and dry isolator and given dietetic food. Equipment and cages in contact with the patient are disinfected with 2% formalin.

The temperature of conditionally healthy animals is measured and healthy ones are vaccinated against plague. Dezogylams, placed before entering the voles, are soaked with a 2% alkali solution. Dishes are washed with disinfectant solutions. The entry of strangers is strictly prohibited, and special clothes are disinfected. During the period of illness, weighing, stamping and other activities will not be held. Manure is biothermally neutralized. The skin of a dead fur animal is dried in an isolator, for 3 days at 25-33 ^{0} C dried, then stored at room temperature for 10 days, and the body is burned.

After 30 days of the last recovered or dead animal, the final disinfection is carried out, and the kennel, settlement or farm is *quarantined by the decision of the governor based on the report of the chief veterinary inspector of the district* . It is allowed to release a dog from this point 1.5 months after quarantine, and a fur animal after 6 months.

Control questions

1. Pathogenicity and susceptibility .
2. Epizootology .
3. Pathogenesis, clinical symptoms .
4. Pathologoanatomical changes .
5. Diagnosis and differential diagnosis.

A FINANCIAL STUDY MATERIALS FOR COURSES

Exercise: Rules of procedure for working with infectious diseases and infected animals

Materials and equipment:

1. Isolator, sick animal, tables, posters, overhead projector.

2. Disinfectant solutions, special headgear, gown, cap, protective glasses, rubber boots, sterile gauze bandage for mouth and nose.

3. Methodical manual, equipment of workplace in field conditions, vivarium using distribution indicators.

4. Equipping the veterinary workplace in laboratory conditions.

5. In laboratory conditions - equipment, laboratory table, flask, distilled water, spirit bottle, Pasteur pipettes, bacteriological hook, cotton, microscope, various dyes, 3% phenol solution, ethyl alcohol, pencil, immersion oil, object window and other devices.

6. Necessary tools and equipment for equipping the workplace in field conditions: collapsible laboratory table, syringe, disinfectant solution, gauze, sterilizer, scissors, tweezers, scalpel, animal fixation items, vaseline, alcohol, cotton, 3% phenol solution, ethyl alcohol, pen, blood collection needles, syringe, dog muzzle clamps, test tubes, hand wash, towel, soap and other supplies.

Basic concepts:

Infectious diseases characteristic of animals and humans (zooanthroponoses). The World Health Organization (WHO) lists more than 150 zoonotic diseases in total. In particular, there are about 30 types of diseases in Uzbekistan: actinomycosis, aspergillosis, alveococcosis, rabies, brucellosis, influenza, leptospirosis, leishmaniasis, listeriosis, microsporia, Newcastle disease, ornithosis, salmonellosis, anthrax, trichophytosis, toxoplasmosis, trichinellosis, tuberculosis, tularemia, large horned animals found cysticercosis, saramas, protein.

2. Highly infectious diseases transmitted from animals to humans, ways of transmission.

3. Special clothing, place, tools and equipment when working with an infectious disease animal and pathological material.

4. Diagnosis of animals dying of infectious disease, differential diagnosis.

epizootic centers, unhealthy and endangered farms, points, laboratories are as follows:

To prevent the spread of infection and to educate the population about the ways of its transmission.

Veterinary workers can get infected with zooanthroponosis diseases during their activities, activities, laboratory, hunting, animal treatment processes.

There are 2 ways of transmission of diseases from sick animals to humans

1. By direct contact:

- during clinical examination and treatment
-by dissecting animal carcasses and sending fur material

- during vaccination and diagnostic examination
- when working with a sample in the laboratory
- during processing of animal raw materials (hide, fur, wool, hooves, etc.) in enterprises
- when consuming livestock products that have not been neutralized from infection

2. Through indirect contact:

- through external environmental factors (air, special clothing, vegetables, water, etc.), transmissible, blood-sucking insects (mites, wasps, wasps), rodents.

Through the ways of exiting the pathogen from the diseased animal body to the external environment:

-excreted through milk, urine, sputum, animal waste, epidermal layer, fluid released from wounds, genitals, eyes, fluids flowing from natural pores, sweat fluid, feces.

Zooanthroponoses and ways of their transmission to humans

- Anthrax - when looking after a sick animal, when opening a carcass, when sorting skins.
- Tuberculosis - as a result of consumption of unboiled milk of animals (cows) infected with tuberculosis.
- Brucellosis - when caring for a sick animal and providing obstetric care during childbirth.
- Rabies - when bitten by rabid animals.
- Leptospirosis - during the care of a sick animal, obstetrical assistance to it during childbirth, when eating food, as a result of damage by rodents.
- Protein - when consuming unboiled milk and compared to sick animals.
- Listeriosis - when taking care of sick animals and eating raw milk, when opening the carcass.
- Manka - when looking after sick animals, when eating raw milk, when opening a dead body.
- Camel plague - when eating the meat and milk of a sick camel, processing its wool.
- Ornithosis - looking after a sick bird.
- Temiratki - in treating and caring for a sick animal.

Measures to ensure the health of people caring for and treating a sick animal :

1. People caring for a sick animal, veterinarians must learn the special instructions.
2. When working with animals infected with brucellosis, tuberculosis - they must be vaccinated against this disease.
3. It is forbidden to involve pregnant women and minors in the care of sick animals.
4. If there is a wound on the skin of the hand or face, it must be treated with a 5% iodine solution.

Measures to ensure the health of veterinarians:

1. Pass the briefing before starting work
2. If there is a wound on the skin of the hand or face (open areas), it must be treated with a 5% iodine solution.

3. A special top must be worn.
4. It is forbidden to touch the face, mouth, nose, eyes and other parts of the body with hands, to smoke, to eat food, to talk with assistants.
5. In order to prevent the spread of infection, all equipment, tools, glassware are checked for integrity and proper operation.
6. When working with pathological material, the liquids extracted from them are taken in Pasteur pipettes, the containers are hermetically sealed. Must be sterile.

Review questions

1. What does epizootology teach?
2. Rules for working with a sick animal.
3. Prepare a draft decision for quarantine .

Basic textbooks and study guides

1. Salimov XS, Kambarov AA , Salimov I. X. , "Epizootology and Infectious Diseases " Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise : Separation of sick animals, isolators, structure of isolators and requirements for them

Before examining animals infected with zooanthroponoses, their epizootological data are collected, based on their clinical signs, pathological-anatomical changes, and the results of laboratory tests.

For example, in case of anthrax and several similar diseases, in the process of epizootological investigation, it is necessary to remember that all types of farm animals or many wild animals are susceptible, except for humans.

About insulators - isolation means separation. Isolation of sick cattle is one of the most important measures in the fight against epizootics and its elimination. Diseased and suspected disease cattle are separated from the general herd. Regardless of the form of the infectious disease, the diseased animal is the main source of transmission of the infectious agent. Separation of such diseased cattle from healthy cattle means loss of the source of infection, severing the most important link of the epizootic chain. This is the main requirement for fighting infectious diseases. Care of a sick animal is performed by specially trained people. They are provided with a special clothing head. Clothes - heads are stored in special cabinets in the isolator and periodically disinfected. It is necessary to pay attention to the spread of the disease during manure cleaning, haymaking, and water supply. And shoes are disinfected in dezomats built at the threshold and in front of the door.

Sick animals are kept in special rooms, and sometimes with t oda. Animals with the same diagnosis are often kept together.
All work related to a sick animal is carried out in special clothing - head.

The initial acquaintance of the student is registration of a sick animal, study of documents of a sick animal, collection of anamnesis and epizootological data.

Separation and storage of sick animals is carried out in special places (isolator).

Isolators are built on the basis of a special project, 500 meters from residential areas, poultry farms, 300 meters from roads and railways, 200 meters from livestock and fur breeding farms, at least 50-150 meters from regional and local roads. must be. - any farm barns are built on the farm area to accommodate 3-5% of cattle. The isolator should be surrounded by a deep ditch, walls or fences. Insulation should be h=2.4 meters, for horses h=2.7m. Isolators should be cleaned daily, and kept disinfected by neutralizing cattle litter and various wastes.

Equip with the necessary drugs: we use biological and chemical treatments t/ drugs that affect the correct pathogen. It should be enriched with hyperimmunized serum, convalescent blood, gammaglobulins, bacteriophages, sulfonamides, nitrofuran drugs, premixes, etc.

Measures to ensure the health of cattle breeders.

1. Timely and quality implementation of effective general and special preventive measures against zooanthroponosis diseases on the farm.
2. It is necessary to keep a sanitary record for workers on the farm, undergo medical examination of new workers, all other workers must also undergo medical examination according to the plan and learn the instructions through special interviews and lectures.
3. Zoohygiene, sanitary conditions must be at the required level in animal husbandry buildings, rest and recreation rooms must be organized.
4. It is necessary to have hot water, cold water, hand washers, soap, towel, medicine cabinet, bandage, cotton, gauze, etc. in farms.
5. Each worker must keep his work place, tools, and special clothes clean on the farm.
6. In the process of milking, milkers are not allowed to touch other objects, their own aft stalls (coughing, cigarettes, smoking, eating...).

At the end of the work, the following actions should be performed.

1. Wash your hands thoroughly first with soap and then with a disinfectant solution.
2. Tools, tables, all other equipment, special heads used in the work are disinfected.
3. Residual pathological materials are destroyed by incineration in a muffle furnace.
4. In the course of work, if samples of infection fall on the skin, membranes (eyes, nose, mouth...), it is necessary to consult a veterinarian.

Review questions

1. Infectious and epizootic processes driving forces ?
2. Zooanthroponoses and their ways of transmission to humans?
3. Measures to ensure the health of livestock farmers?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA , Salimov I. X. , "Epizootology and Infectious Diseases " Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Principles of diagnosing infectious diseases .

of obtaining the complex (confirmed by the veterinary laboratories of the district, region, republic) is considered legally correct. Based on these, the following are included in the comprehensive examination of infectious diseases. (epizootological, clinical, pathologoanatomical, bacteriological, virological, immunological, allergic, serological, etc.). Figure 1

When diagnosing infectious diseases, epizootological criteria are necessarily taken into account, the importance of which is that every sick animal is a source of an infectious disease agent. The transmission of a desired pathogen from a source to another animal is called an epizootic.

Disinfection - that is, it consists of the concept of infection, which is the French word des - to lose, infectio - the Latin word.

Disinfection is a method of decontamination of external environment buildings, its effect is aimed at killing pathogenic microorganisms in these places.

Disinfections carried out by us will destroy the 2nd link of the epizootic chain, i.e. the transmission mechanism of the causative agent.

The causative agent of the disease is transmitted from infected animals to healthy animals through infected buildings and living organisms. (arthropods, mites, rodents, etc.).

Disinfections are divided into prevention, mandatory, current and final.

In the implementation of modern livestock **preventive disinfection, the following:**

1). **Before placing animals in the building** - disinfection carried out before placing animals in the building, after the completion of livestock constructions.

2). **Technological disinfection** - depends on the size of the farm and the technological direction of livestock production. It includes special large livestock associations that produce animal products on an industrial basis.

Mandatory - when infectious diseases spread among animals in the farm.

Current - when symptoms of the disease appear continuously in the farm. Current disinfection is carried out taking into account the timely destruction of the specific infectious agent and the state of microbial transport in an unhealthy household.

The final is the disinfection that is carried out after sanitizing the farm, before taking the demarcation from the farm.

In veterinary medicine, we deal with disinfection by 3 methods - a) physical, c) chemical, c) biological.

Deratization - fight against rodents. There are currently more than 2,500 species of rodents on Earth. They join 30 families. Their dangerous side is that, according to modern science, they spread more than 200 infectious and invasive diseases. Also for rodents a) mechanical - traps, daviloks, etc.

c) chemical - simple and effective zoocoumarins, its sodium salt, penocoumarin, bactocoumarin, etc. c) biological - we fight with cats, puppies, natural wild animals, foxes, martens, birds.

Disinsection is the fight against insects. Many insects are carriers of infectious diseases. The loss of insects is of great epizootological significance. Because they can be a natural source of some diseases. Biting and blood-sucking insects not only disturb cattle, but also affect their productivity. Physical, chemical and mechanical methods are used to control insects. The effectiveness of disinsection is high if the agronomist and entomologist work together on the farm.

Importance of veterinary sanitation. Zooanthroponous diseases are very dangerous for animals and humans. Therefore, measures against epizootics are effective only if they are carried out with the active participation of the population, especially livestock workers.

When organizing practical work against epizootics, it is necessary to protect people from some infectious diseases. (tuberculosis, brucellosis, anthrax, rabies, listeriosis, skin diseases...) Therefore, to the following

1). Regularly conduct explanatory work for Molbokar employees;

2). Timely supply of livestock workers with special clothes;

3). Creating conditions for personal hygiene. (special recreation room, rest area, toilet...).

4). Provision of special disinfection equipment (pasteurizers, vacuum cleaners, cooking pots...)

5). There should be chambers for disinfecting clothes.

interviews among the population , organizing consultation points; The development of special booklets on veterinary and infectious diseases should be the focus of every manager and practicing veterinarian.

Review questions

1. Preventive and anti-epizootic measures?
2. What is the fight against epizootics?
3. Disinfection ?
4. Disinsection ?
5. Organization of deratization activities. ?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA , Salimov I. X. , "Epizootology and Infectious Diseases " Textbook. Tashkent , 2021 .

2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.

3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.

4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

EXERCISE : The importance of disinfection, deratization and disinsection in the fight against infectious diseases .

1. Disinfectants, their preparation.
2. Types of disinfection
3. Mechanization of disinfection.
4. Evaluation of the quality of disinfection.
5. Completion of the lesson, questions - answers. Assessment of mastery, knowledge and skills by rating.

The main task of veterinary and sanitation is to prevent animals from infectious diseases. For veterinary hygiene: water, fodder, air hygiene, vet for livestock. sanitary or zoohygienic requirements, requirements for livestock products or their vet. number ex. includes Epizootology includes methods of disinfection, deratization, disinsection and disposal of dead animal carcasses.

Disinfection means getting rid of infection. Depending on the purpose of disinfection, it is divided into preventive, current, mandatory and final types according to the plan. Depending on the type of disinfectant, it is divided into chemical, physical, biological... methods.

1. Chemical disinfection - destruction of the pathogen with the help of chemical means.

- alkaline disinfectants NaOH, KOH, Ca (OH) $_2$ NH_4OH ...
- mechanism of action of microorganisms.
- acid disinfectants HCL, $HCLO_3$, H_3HPo_4, H_2S, H_2Co_3 ....
- chlorine disinfectants
- phenolic disinfectants
- heavy metal salt disinfectants
- formalin disinfectants

1. Chlorine lime - granular white powder. It is considered one of the most effective disinfectants, widely used in veterinary medicine and medicine. Its effectiveness is determined depending on the amount of active chlorine in the chlorinated lime. The amount of active chlorine is measured in %. The amount of active chlorine in technical chlorine lime is 38-40%. When stored in the open , chlorinated lime reacts with moisture and Co $_2$, resulting in a semi-dark, liquid mass.

Active chlorine in standard chlorine lime must not be less than 25%, if the amount of active chlorine is less than 15%, it is unsuitable for disinfection. Before chlorine liming, the content of active chlorine is determined. For this, cylinders of 100, 250, 500, 1000 ml divided into scales are prepared. 10, 25, 50, 100 ml pipettes are prepared. 250, 500 ml Erlenmeyer flasks are also available. 2% KJ solution 0.1n or 0.01n hydrochloric acid, 0.01n sodium thiosulfate, 1% starch solution.

Take 1 g of samples from different parts of the barrel containing chlorinated lime, mix well, add a little distilled water and mix in a mortar. It is placed in a measuring cylinder and distilled water is added to the last mark. After mixing, 0.1 volume of the liquid in the cylinder is poured into an Erlenmeyer flask, so 10 ml of

2% potassium iodide solution is added and 10-15 drops of hydrochloric acid solution are added. A separate pipette is used for each solution. Mixtures are shaken. The solution will be orange. Then titrate with 0.1 or 0.01 N sodium thiosulphate solution until pale yellow, then add 1 ml of 1% starch solution until the solution turns blue until the blue color disappears. is titrated. The amount of consumed sodium thiosulfate is equal to the equivalent of added iodine, and the amount of active chlorine is determined accordingly.

It is calculated according to the following formula.

X = ac x 0.0355 x 100/0.1

X is the amount of active chlorine in %

A is the amount of spent sodium thiosulfate

K - the coefficient of sodium thiosulfate solution is 0.00355 gram equivalent of 0.1 n HCL.

100 is the coefficient

0.1 - a bowl on the scales.

For example, 1 g of chlorinated lime powder is added to 100 ml of distilled water. For titration, 10 ml was taken or 0.1 part was taken. 6.6 ml of 0.1 N sodium thiosulfate is used for titration. The correction factor of sodium sulfate is 1.1.

Then X = 6.6x1.1 x 00355 x 100/0.1 = 25.8%

So active chlorine in tested chlorinated lime is equal to 25.8%. There are other ways. Slaked lime is prepared from NaOH, KOH, CaO. Phenol - serno carbol mixture, creolin, naphthalizol, xylonaft - 5 are used. In this case, H_2So_4 is poured over carbolic acid little by little. (the reverse is not possible). It is kept for 3-5 days, after which 3-5-10% solutions are prepared and used.

Formalin contains 40% formaldehyde (standard), 8-20% methyl alcohol, formic acid, and similar compounds. Formalin is stored in a tightly closed container at ^{90}C. The amount of formaldehyde in formalin also determines its effectiveness. For example, to prepare a 3% solution from 36% formalin, X =3 . 100/36 = 8.333 l. so 8,333 l of formalin (36%) is dissolved in 91,667 l of water.

An alkaline solution of formaldehyde is 3% formaldehyde in 3% alkali. For this, 50 l of 6% NaOH solution is prepared, then formalin is added in the required amount, or 8.333 l is prepared according to the calculation given above, and then water is added to 100 l.

Aerosol disinfectants are aerosolized in the VDM-2 machine, ADA unit. In this case, liquid is used to create smoke and fog.

Bacteriological control is carried out to determine the quality of disinfection work. The concentration of 0.1% sodium thiosulfate to neutralize chlorine lime, 0.01% acetic acid to neutralize alkalis, and 1-2% NH_4OH to neutralize formalin must be 10 times lower than that of the disinfectant.

The swabs and samples sent to the laboratory are cultured in nutrient media and the result is determined based on the colonies.

The corpses of sick animals are destroyed in special crematories, in muffle furnaces or in specially prepared places, or they are sent in special vehicles to special waste plants.

The causative agent of tuberculosis is kept in the body for 17 months, the causative agent of pasteurellosis is kept for 4 months, the causative agent of swine fever is kept for 9 months. Decontamination, disinsection, and deratization of manure are carried out.

Preparation of chemical disinfectants used in veterinary practice.

1. Chlorinated lime solution - technical chlorinated lime contains 30-38% of active chlorine. In the composition of standard chlorinated lime, chlorinated lime should not be less than 25%, if it is less than 15%, it is considered unsuitable for use. In the laboratory exercise, the determination of the amount of active chlorine in chlorinated lime is studied. (Titration method)
2. NaOH -Sodium hydroxide in liquid form - 42% sodium NaOH crystals are extracted in the form of 92-95% NaOH. Here too, 2-3-5% solutions of NaOH are determined by shaking method.
3. $(OH)_2$ – calcium hydroxide solution is prepared from quicklime SAO. It absorbs and burns the skin and tissues. A 20% freshly slaked lime solution is prepared.
4. In veterinary practice, in phenolic compounds, cernocarbol (sulfur) mixture, creolin, naphthalizol, xylonaft - 5 solutions are used.
5. Formalin - 40% formaldehyde in the standard condition is removed in technical formalin 8-20% methyl alcohol.
6. Formaldehyde alkali solution 3% formaldehyde + 3% NaOH solution is often used.
7. Aerosol disinfection is also used in veterinary practice.
8. Disinfection of carcasses, excrement, manure of sick animals: crematoria, muffle furnaces, biothermal decontamination, incineration in special places, etc.

List of references

1. Antonov V. YA., Blinov PN "Laboratornye issledovanie v veterinirii". Moscow - 1971, str. 5-30.
2. Urban. VP "Praktikum po e pizootologii ii nf.bolezney".Uch.pos . L. 1987
3. MPParmanov et al. "Epizootology" 2007.

EXERCISE : Disinfection of farm buildings, areas where there are piles of soft fodder and coarse hay.

The purpose of the lesson: to master the methods of destroying insects in farms. Study of insects that transmit infectious diseases and their biogenesis.

Supply of laboratory training: collection of insects, insecticide preparations, tools, equipment.

Students from this laboratory lesson are as follows;

1. What infections are spread by insects, ways of spreading.
2. What epizootological features are characteristic for transmissible infections.

3. In transmissible infections, which indicator shows the intensity level of the epizootic process.

Disinsection is a complex set of measures aimed at destroying arthropods that harm animals and spread infectious diseases. Disinsection is mainly studied more deeply than the science of parasitology. The science of epizootology deals with the elimination of insects that spread infections.

Mites - pasture mites are found in the north. I. persulcatus is common in forested areas.

I. risinis is found in h/. Dermacentor pistus is found in hayfields, D. marginatis is found in mountainous and desert areas. All ticks play an important role in the spread of infectious diseases.

The role of argas, gamoz, krasnotelkovkh mites in the spread of infectious diseases has been sufficiently studied.

Disinfection methods used in different conditions: mechanical, physical, biological, chemical disinfestation methods are used to destroy arthropod harmful insects.

The mechanical method is more often used to destroy flies and mosquitoes. The method could not eliminate 100% of insects. Therefore, other combinations are used. Mechanical cleaning of livestock and breeding grounds, use of adhesive paper, etc.

Physical method - disinsection is mainly used to destroy mites and ticks. Steam, boiling water, dry steam, flames are used for this purpose.

Insecticides are used in the biological method of disinfestation. In this, birds, some insects, microbes, viruses, fungi are used. M. Birds eat flies, mosquitoes, ducks, beetles and fly larvae.

Chemical methods of disinsection are performed using chemical insecticides. Insecticidal preparations are divided into types with contact, enteric, fumigant and repellent effects depending on the nature of action.

1. Contact insecticides kill insects by entering through their body. (issued in the form of dust, solution, aerosol ...)
2. Insecticidal drugs that act on the intestines, they enter insects with food. (calcium, sodium arsenite, arsenic, boron, etc.).
3. Fumigant insecticide enters the body of insects through the respiratory system. (in the case of gas, steam, vapor).
4. Repellent insecticide - does not kill insects. "drives", threatens. (dimethylphthalate, diethyltoluamide, etc.).

Pure 15-20% glycerin, vaseline emulsion, 10-20% alcohol solution are used, harmless to humans.

Diethyltoluamide (DET) is a pale yellow oily liquid. Has an insecticidal effect. Stronger than dimethyl phthalate. Pure or 50% alcohol solution is used.

Among the repellents, a solution of benzimin, syodrin, karbofos, polychlorpenin and other insecticides is often used. They are also used as an emulsion in water.

If the solution is prepared from liquid concentrates, first the concentrate itself is thoroughly mixed, then the emulsion is prepared in it. It is widely used in the form of powder and dust. They are used with special pollinators. Large areas are sprayed with tractors and machines, sometimes by aviation. Insecticides are also used in the form of smoke.

Chlorophos aerosol 0.05g/m $^{3 \text{ to houseflies}}$. Sevin will be happy. When insecticides are used, people should not be harmed and food products should not be contaminated. Special headgear, mask, goggles, gloves, space suit, etc. it is absolutely impossible to increase the concentration of insecticide preparations. Livestocks are cleaned mechanically before applying insecticide preparations. Mangers, water containers, equipment are washed with an alkaline solution after disinfection.

Preparation of working solutions, emulsions from concentrated preparations.

t/r	**Amount of active substance in drugs**	**The amount of ftm for the preparation of 1l working solution is calculated by the amount of insecticide in g**				
		0.5%	**1%**	**2%**	**3%**	**5%**
1	5	100,	200	400	600	1000
2	7	71.4	142.9	285.7	428.6	714.3
3	10	50	100	200	300	500
4	12	41.7	83.3	166.7	250	416.7
5	15	33.3	66.7	133.3	200	333.3
6	20	25	50.0	100	150	250
7	25	20	40	80	120	200
8	30	16.7	33.3	66.7	100	166.7
9	40	12.5	25	50	75	125
10	50	10	20	40	60	100
11	60	8.3	16.7	33.3	50	83
12	70	7.7	15.4	30.8	46.2	77

livestock farms is carried out in a complex way. Complex disinsection works consist of preventive measures. Preventive disinsection works, full compliance with

general and special veterinary sanitary rules, the farm should be clean, there should be no dirty places where flies, mosquitoes, and insects breed. Manures should be biothermally neutralized.

Istribite measures (elimination of eggs and larvae of insects) 0.1% trichlorophos-3 emulsion, 5% li-polychlorpenin emulsion, 10% naphthalizol, creolin emulsions are used 3-5 l per 1 m^2 · It is held once in 10 days. Eggs of flies and mosquitoes develop quickly in pig and cattle manure. Chlorine lime powder is sprinkled on the manure. YSHH livestock farms are treated with 0.5-1% chlorophos, 3% PCP emulsion. Calves, piggeries, stables are treated once in 3-5 days with 0.5% trichlormetaphos-3, 1% chlorophos, 2% chlorpene, 0.1% pyrethrin in animal pasture conditions.

Deratization is a set of actions aimed at destroying rodents. Rodents are the spreader, reservoir, and source of infection.

During the laboratory lesson, students should master the following questions.

- What rodents live on the farm.
- The role of rodents in the epizootology of infectious diseases.
- Economic damage caused by rodents.

Rodents give birth 4-8 times in 1 year. 1. Mice at 1-2 months, rats at 3-4 months.

0.5 or 1 ha of land 2 m wide diagonally 2500-5000 m, how many in (nest) are there ? We will count again the next day.

M: Suppose 350 nests are found on 0.5 ha, 250 of which contain rats. So there are 700 nests in 1 ha, 500 rats. All nests are covered with sand, earth, paper, and those that are opened the next day are counted.

1 per mouse. 200-1500 g of grain, 100-150 g of bone meal, 500-3000 g of liquid.

Rat 2000-3000, 1000-3000 g Raticides: Crcid, zinc phosphide, zoocoumarin, larinat, monofluorine and others.

Control questions:

1. Send a referral letter.
2. Importance of comprehensive epizootological investigation.

List of references

1. Antonov V. YA., Blinov PN "Laboratornye issledovanie v veterinirii". Moscow - 1971, str. 5-30.
2. Urban. VP "Praktikum po e pizootologii ii nf.bolezney".Uch.pos . L. 1987
3. MPParmanov et al. "Epizootology" 2007.

EXERCISE : Anthrax diagnosis and countermeasures.

Plan:

1. K honey prevention, detection and documentation of inspection results.

2. X organization of treatment and implementation of countermeasures in cold sores.

Materials and equipment.

1. Microscope.
2. Dyes according to Gramm's method.
3. Test sample (taken from positive and negative skins)
4. Precipitating anthrax serum.
5. Anthrax antigen.
6. Ready-made ointment in the form of a capsule (must be placed under a microscope for control, to show to students).

Anthrax - in Latin Febris carbunculosa, in English Anthrax, in German Milzbrand, in French Fievre, in Russian Sibirskaya yazva - an acute, septic, abortive, carbunculosis, intestinal, lung and localangina severe infectious disease is characterized by septicemia, severe intoxication and the formation of carbuncles.

People, animals and birds are infected with this disease. Humans suffer mainly from skin anthrax, localized forms in pigs and foodborne infections in fur animals.

When anthrax is in its classic form, the disease can be mistakenly diagnosed based on clinical symptoms.

But in sporadic cases, extreme vigilance is required when animals are suspected of having the disease. Therefore, it is necessary to carry out a comprehensive examination when diagnosing anthrax.

Epizootological, clinical, pathologo-anatomical and laboratory examinations are carried out.

The causative agent - B acillusan th racis - belongs to the genus Bacillus, family Bacillaceae, rod-shaped, large (1-1.3x3.0-10.0), non-motile, gram-positive, single or double, short-long It is a filamentous, spore- and capsule-forming aerobic bacterium.

epizootological examination method. The source of anthrax disease can be soil, water bodies, places where animal carcasses are buried, places where animal raw materials are processed, places where skins are accepted, factories where meat and bone meal are prepared. At the time of inspection, Bac in that territory. Is there an anthrax outbreak? - no? It counts. Were animals fed (pasture) in that hearth or not? The burial place of dead animals with anthrax, the surrounding water (ponds, ponds, etc.), secretions and excreta from infected animals infect the ecological environment. Blood-sucking arthropods should also be taken into account during epizootological investigation, as the disease can also be spread by transmissible means.

An infected animal develops weakness and fever.

If the animal is dead, if anthrax is suspected, such carcasses should be examined without opening them: the carcass does not harden, it swells strongly, we see various pasty swellings in the subcutaneous tissue, foamy from natural pores fluid mixed with blood flows, visible mucous membranes are stained blue, blood is shed. But these changes are not always the same. The final diagnosis is made by the method of laboratory examination. For this, a swab from the animal's ear is sent to the laboratory. Or it is sent from the lower side of the ear by tying it well from both sides. In the laboratory, the smear is stained according to Gramm's method and viewed under a microscope. If bacilli are found in the smear in the form of a chain in the form of a capsule, it is necessary to start anti-anthrax measures without waiting for its result. The

serological method is checked by the RP reaction, for this, 10 x 10 cm of animal skin is cut, placed in an autoclave, then crushed and placed in a 1:10 carbolic physiological solution in 50 mm jars.

The jar is kept at room temperature for 16-20 hours. During this time, the anthrax antigen extract is formed. Then it is filtered through asbestos paper through a funnel into a Florinsky test tube, cleaned. 0.25-0.3 ml of the precipitated serum of anthrax is poured into a special test tube, and the same amount of the purified solution is poured over it. In pouring, the reaction is carried out by slowly pouring the liquid down the wall of the test tube. The result of the reaction is determined in 15 minutes. If a yellowish-white ring is formed at the point where both liquids are joined, it is considered a positive reaction. If there is no ring, the reaction is negative. These reactions are placed in areas where the skin receives. It is necessary to distinguish the disease from pasteurellosis, bradzot, black fever, gas swelling.

Anthrax is a very dangerous infectious disease due to its acute course. In order to prevent, healthy animals are vaccinated with STI, GNKI, Strain-55 vaccines.

Animals diagnosed with the disease while alive are quickly isolated. Animals should be protected from arthropods, mainly bloodsuckers. Treatment of sick animals is carried out in a special or symptomatic way. The sooner the treatment is started, the more effective the result.

In a special method of treatment, hyperimmune serum or immune globulin prepared against anthrax is injected. Serum in large quantities: 100-200 ml for large animals, 30-60 ml for sheep and pigs, mainly through a vein. And at the same time, antibiotics (terramycin, oxytetracycline...) are administered.

Medicines affecting the cardiovascular system are administered for the purpose of symptomatic treatment. A cold compress is placed on the animal's head or cold water can be poured. If there are carbuncles, swellings, a hot phenolic compress is applied. A 3-5% carbolic acid solution is injected around the swelling, rubbing camphor alcohol on the skin is also effective.

Review questions

1. Methods of diagnosing anthrax.
2. Create an action plan against epizootics .
3. Sending pathological material to the laboratory in Kuydtrgi.
4. Prepare a draft decision for quarantine .

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: detection, prevention and countermeasures of AUTOIMMUNE diseases .

Tuberculosis is often an acute, rapidly spreading infectious disease. Therefore, accurate diagnosis of white tuberculosis and timely implementation of measures against it will have a good effect.

Diagnosis can be difficult in a previously healthy household because tuberculosis can be a mixed infection. In this case, we need to carry out a deep epizootological examination in making a diagnosis. Focusing on the economic relations of the economy, transport, people and other vehicles serve as mechanical transmitters of tuberculosis.

Tuberculosis - Latin Aphtae epizooticae, Foot-and-Moythdisease in English, Maul-und-klaunseuche in German, Fievre aphteuse in French, Yashur in Russian - extremely contogious, fever, mucous membrane of the mouth, udder and is characterized by the appearance of canker sores between the hooves. Both ungulates and humans are infected with the disease. Horses do not get tuberculosis, they are susceptible to vesicular stomatitis. Cattle are infected everywhere. They have weakness, fever, up to 41 ^{o}C in young animals. After the period of generalization, aphthae appear in the oral cavity, on the teats of the udder, in some hairless areas, between the hooves, and then vesicles.

The causative agent is Aphtae viridae, a filterable RNA virus belonging to the genus Aphtovirus, family Picornaviridae, and has a size of 20-25 nm. It is divided into 7 serological types depending on its antigenic properties: O, A, S, Sat-1, Sat-2, Sat-3 and Asia-1. Each type has several variants: O-13, A-32, S-5, 7 in Sat-1, 3 in Sat-2, 4 in Sat-3 and 2 in Asia-1. Protein virus is highly resistant to lysol, toluene, ether and chloroform, which kill a number of bacteria and viruses.

Epizootological data. When analyzing epizootological data, it is necessary to take into account all protein-unhealthy points in the country, because the protein virus enters the healthy region from the epizootic center thousands of km away. Humans get the disease when feeding animals, eating their cooked milk and products made from this milk. The virus enters the body through the mucous membrane of the oral cavity, sometimes through the respiratory tract and damaged skin.

Pigs are more susceptible to the disease, and sheep, goats and wild boars are less susceptible to the disease. The disease is more severe in young animals than in adults. The source of the disease is sick animals.

In the epizootic analysis, it is necessary to take into account how the disease was diagnosed in the previously unhealthy farm, and how it entered the farm. If vesicular stomatitis is widespread in the farm, it should be differentiated. After the appearance of a tooth on the upper jaw in pigs, the appearance of an exanthema in the body, the temperature in the body returns to its normal state.

After aphthae burst and ulcers appear in the oral cavity, foamy saliva begins to flow. In **COWS INFECTED WITH tuberculosis,** the ability to give milk decreases, and the quality of milk also changes.

TUBERCULOSIS in calves is very severe, in a diseased form, animals die within 12-30 hours after swallowing with high temperature **.** In sheep, similar to cattle, fever is expected, vesicles in the oral cavity, then aphthae appear, but saliva

does not flow. Ulcers appear between the hooves and lameness. For laboratory examination (sampling is carried out as indicated in the Book of Veterinary Regulations, Volume 1, page 190).

White tuberculosis is taken from the vesicles in the epithelial wall for laboratory examination. For one examination, 5-10 g of epithelial aphtha is placed in a clean container, on top of which there is 10 times more glycerin with a 50% pure chemical solution (RN-7, 47.6). (10 g of epithelial afta is put in 100 ml of 50% glycerin solution). For inspection, only a clean sample, unlit, ripe blisters are cut with clean scissors. It is taken from the tongue from cattle, from the toothless or hoof gap of the upper jaw from sheep, from the nasal window from pigs. The sample is taken from 2-3 animals at a time. The obtained sample is taken in a container with a tight closure and covered with cotton, then a cork and a lid. The obtained sample is used to determine the type of virus. Then the clinical course of Tuberculosis in various animals is shown through specially prepared tables, slides, pictures.

treatment of animals infected with TUBERCULOSIS .

TUBERCULOSIS DEPENDS ON the factors of their proper feeding and storage. Keeping sick animals in a quiet position, because in order not to strain their cardiovascular system, the place where they stay should be clean and always have fresh air. The bedding of the animals is mixed. In tuberculosis, the animal is severely dehydrated, so it must drink water voluntarily. In the case of gastroenteritis in young cattle, the ratio of manganese-acidic potassium solution (1:1000) is given once every time in the volume of 500 ml. Sick animals are given round, fast-digesting food.

For example: Round blue grasses, flour slurry, good silage during winter, round hay are given. It is necessary to rinse the oral cavity with clean water with 2% acetic acid or use a solution of manganese oxide potassium 1:1000, furacilin 1:5000. If the mucous layer of the oral cavity is strongly inflamed, it is necessary to apply an ointment (anesthetic 2.5 g: novocaine 2.5 g, copper cup 5 g: fish oil 20 g, petroleum jelly 70 g) . Such an ointment accelerates the recovery of erosion, relieves pain, and the animal's nutrition improves.

The hooves are cleaned every 1-2 days and rubbed with a mixture of "degot" and fish oil. Such animals are driven through a debarrier containing diogot soaked in wood shavings or 5% formalin solution. If the hooves are slightly injured, wash them with soapy water, 1% copper sulphate, 2% chloramine solution. Then it is sprinkled with a mixture of manganese acid and potassium, boric acid (aka), or a 10-20% aqueous solution of formalin is applied. Dyogot, fish oil mixture can also be applied. If there are deep wounds on the hoof, iodine solution is applied. If there is dead tissue in the hoof, it is cleaned and blocked with 0.5% novocaine solution and penicillin.

If the animal has sepsis due to white tuberculosis, it is recommended to inject 0.5 ml of 0.5% novocaine solution per 1 kg of live weight. It is necessary to milk the udder of an infected cow with clean milk, wash it with soapy water before milking, and then wipe it with a clean towel.

If there are animals with severe tuberculosis, if there is a change in the vascular part of the heart, a mixture is recommended (10 ml of valerian tincture, 15 ml of

landish tincture, 6 g of potassium bromide in 400 ml of distilled water). If among the sick animals there are those who do not drink water, 20-30 liters of flour slurry is given through a probe or a rubber bottle. Very weak animals are given 100-200 g of honey or 200-400 g of sugar added to skimmed milk.

Serum or convalescent blood or immunolactone preparation against Tuberculosis is used (see Vol. 1 VK p. 628) written in the "Veterinary Laws" book.

Establish prevention and elimination measures against tuberculosis .

clear IT Due to the fact that it is an infectious disease that spreads rapidly and widely, it is necessary to conduct an event on the basis of the manual adopted in 1987 .

It is only necessary to establish strict, rapid disease elimination measures as indicated in the manual. The method of events is as follows:

Preventing the spread of white tuberculosis in healthy households and farms and carrying out vaccination against this type, organization of zoosanitary.

Timely finding the source of the disease in the infected farm and eliminating it completely, so that it does not become the source of the disease.

Explain to the students about TB elimination measures and quarantine project in unsanitary farm based on complete Tuberculosis manual. It is necessary to describe the work carried out during quarantine, the final disinfection, the draft of the decision to be drawn up for quarantine (it is necessary to write a document explaining the form of the document to be drawn up).

It is also necessary to provide full information about the activities that will be carried out after the quarantine, i.e. restrictions.

Review questions

1. Initial diagnosis in protein disease.
2. Sending patmaterial in case of protein deficiency to the laboratory.
3. Measures to prevent and eliminate protein disease

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: T uberculosis detection, prevention and control measures. Materials and equipment

Complex methods of diagnosis

Epizootological method. To study the epizootic situation of tuberculosis, to determine all the unhealthy points, the time of rehabilitation, the structure of the herd, where and when the breeding bulls were brought, and the economic relationship of the farm with other unhealthy farms. It is necessary to determine the epidemiological situation regarding the health of animals and poultry of private households in settlements, as well as the health of people who serve animals.

Clinical method. It allows you to identify visible signs of the disease. Clinical signs include weight loss, coughing, rapid breathing, occasional fever, enlargement of superficial lymph nodes, diarrhea, liquefaction of milk, purulent fibrin fragments, and atrophy of the pectoral muscle of birds, despite good nutrition. signs are grounds for suspicion of tuberculosis. These signs are not always observed and cannot be the basis for making a diagnosis. Therefore, it is necessary to make a diagnosis using complex methods.

Basic concepts:

ALLERGIC METHOD .

Allergic testing is performed to detect animal and poultry tuberculosis. Purified PPD for mammals (protein, pouioified derivative, tuberculin and PPD tuberculin used for poultry).

In the allergic examination of animals and poultry, the allergic method is recognized, and in horses it is determined by the drip method.

The technique of intradermal injection .

Before injecting tuberculin into the skin, the thickness of the skin is measured with a caliper , the wool is clipped and treated with 70% ethyl alcohol. Tuberculin is applied to large horned animals, buffaloes, deer in the middle of the neck, and in bulls under the tail, in the stomach of camels, in sheep, goats, dogs and monkeys in the inner side of the rump or in the armpit, in blackbirds - intrapalpebral or It is sent to the top of the eye, the eyelid, on one side of the earring in birds.

Tuberculin is administered to mammals (except monkeys and baboons) 0.2 ml, to monkeys, baboons and birds - 0.1 ml using special small needles and syringes. After the administration of tuberculin, the reaction to it is evaluated after 72 hours when examining large horned animals, buffaloes, camels and deer, after 48 hours when examining sheep, goats, pigs, dogs, cats, and fur animals. , and when checking birds, it is evaluated after 30-36 hours. Skin thickness is measured using a calliper to evaluate the reaction. Large horned animals, buffaloes, camels and deer, if the skin thickness is 3 mm and more, other animals, if the skin of the tuberculin injection site is swollen in poultry, if the eyelid is swollen in blackbirds, the result is considered positive.

Eye drop technique

The method of instillation of tuberculin into the eyes is carried out 2 times with an interval of 5-6 days. Allergen is instilled with an eye pipette in 3-5 drops on the mucous membrane of the lower eyelid with closed eyelids. This method cannot be used if the animal's eye is damaged.

are taken into account every three hours after the first instillation of the allergen into the eye, i.e. after 3, 6, 9, 12 and 24 hours, and after 3, 6, 9 and 12 hours after the second instillation. In the case of a positive reaction, the mucous membranes of the eyes turn red, and pus begins to flow from the inner corner of the eyeball.

Pathologoanatomical method

When making a diagnosis of tuberculosis, the organs in the tune are seen. Lymph nodes, lungs, liver, spleen, mammary gland, pleura, abdominal intestine and other organs and tissues of mammals, liver, spleen, intestines, tendons of birds are examined.

Tuberculosis-like changes are characterized by the formation of nodules (tubercles) in the above-mentioned organs and tissues. They are a hard, gray or dark, gray curd mass, partially or completely covered by a capsule made of calcified connective tissue. The liver, kidney, spleen and mammary glands are damaged when the tuberculosis process spreads throughout the body.

When the serous layers (pleura, pericardium) are damaged, a pearly appearance occurs, in which pea-sized nodules appear in the serous layers, shining gray or pink, reminiscent of pearls.

Laboratory method .

Serological and bacteriological examination is carried out. Complement fixation reaction (CRT) is used in serological examination of large horned animals. The reaction is made with complex tuberculosis antigen (CTA) and is carried out at the diagnostic slaughterhouse of animals. Bacteriological tests are carried out if they are not visible or unclear, and to determine the type of mycobacteria.

Bacteriological examination uses the method of bacterioscopy (smears are prepared and stained by the Sil-Nilson method) to isolate the pure product (nutritional media are planted in Leventein-Gelberga and Petriani media with eggs and a bioassay is carried out).

A bioassay is used to determine which type the pathogen belongs to. Guinea pigs, rabbits and chickens are used for biotesting.

Bioassay method to determine the type of tuberculosis mycobacteria in sera of laboratory animals.

Animal type	Method of transmission	A type of mycobacteria	
	Bulls	People	poultry
To the veins of rabbits	Generalized	In the liver	

	Insulation	Furnace	Tuberculosis sepsis
	Tuberculosis	Tuberculosis	
Sea pigs	Under the skin	Generalized	Generation
	Tuberculosis		Tuberculosis
Birds	To the muscle	Generalized	Tuberculosis

Differential diagnosis: When diagnosing tuberculosis, it is necessary to differentiate from contagious pleuromoniasis (PVL) and pasteurellosis (breast form). Scheme of differential diagnosis of tuberculosis.

Diseases with damage to the lungs.

	PVL (poval. vospol. legkix) gross inflammation of the lungs
Course: tuberculosis is chronic	Movement: Lightning fast, sharp, semi-acute and chronic
Superficial lymph nodes are enlarged and swollen	In the upper lymph nodes there is no difference
On examination, in the lungs, in the lymphatic layers	On examination, the lungs are marble nodules, serous, in chronic cases
Sometimes in the tissue of the udder and in other organs and tissues, nodules with a curdled necrotic mass. The result of tuberculinization when testing for tuberculosis Tuberculosis	The result of a negative reaction when examining the sequestrants for tuberculosis is contagious pleuropneumonia (PVA).

Measures to prevent and eliminate tuberculosis .

1. General rules

Tuberculosis is a chronic disease of agriculture, wild animals and poultry. It is characterized by the formation of tubercles in various injured organs and tissues.

Depending on the epizootic status of farms, farms are divided into healthy and unhealthy farms. Such a farm, herd and farm are considered healthy if no sick animals are found during the tuberculosis inspection. If a sick animal is found as a result of the inspection, such a farm, herd and household is considered unhealthy.

The degree of unhealthiness of the farm herd is set depending on the nature of the course of the infection and the spread of the disease among the animals. If the disease is found in 25% of the total number of animals within 1-2 months, tuberculosis is considered to be rare.

Treatment of tuberculosis in unhealthy areas is carried out in the following way:

a) Animals are systematically checked for allergies. Diseased animals from the herd that have a positive reaction to tuberculin are sent for meat.

b) If the disease is detected for the first time in healthy regions, regardless of the level of spread, or when the disease spreads widely, and in order to get rid of swine and poultry tuberculosis, the unhealthy herd and farm animals are slaughtered for meat. is

established, the farm is freed from cattle, sanitary measures are carried out, and the farm is restored by buying healthy cattle from healthy farms. This method is also used in cases where it is not possible to rehabilitate herds, farms and farms using the method specified in point " A ".

2. Tuberculosis prevention (prevention)

(General prevention).

Managers of a healthy household (on a farm) for the absence of tuberculosis in animals. According to the charter, it is obliged to:

It is prohibited to bring animals from an unhealthy farm to a healthy farm (farm). The health of newly brought animals must be confirmed by a certificate issued by the veterinary service.

Animals newly brought to the farm are kept in preventive quarantine for 30 days. During the quarantine, animals are tested against tuberculosis with an allergic method. They are introduced in the general herd with the permission of a veterinarian or paramedics, who are determined to be healthy against tuberculosis.

Animals should not be allowed to mix with animals from an unhealthy farm (farm) due to tuberculosis, and people who care for animals from an unhealthy farm should not be allowed to enter the farm.

It is necessary to ensure the implementation of veterinary-sanitary and zoohygiene norms and work such as feeding and keeping (farm packing, sanitation transfer point, dezobarrier, disinfection carpet mechanical cleaning, preventive disinfection, disinsection, deratization, improvement of animal feeding and keeping).

(Special prevention).

In order to timely determine whether animals are infected with tuberculosis in healthy farms and settlements, preventive and diagnostic tests are carried out, i.e. testing by injecting tuberculin into the skin.

Cows and breeding bulls on healthy farms belonging to unhealthy farms 2 times a year, heifers (mares) before and after insemination, young calves from 2 months 1 time a year, sows and males and 2 times in 1 year, an allergy test against tuberculosis is performed.

Animals are examined by injecting tuberculin between the skin from the age of 2 months. Cows (foes), buffaloes, camels are tested by the allergic test method against tuberculosis regardless of the period of estrus, mother goats, sows, sows, sows, donkeys 1-2 months after giving birth, deer (moral) from November until February, the mother hens are checked before laying eggs.

3. Rehabilitating sick cattle farms from tuberculosis .

The choice of methods of health treatment of unhealthy farms, herds and households in relation to tuberculosis depends on the course and spread of the disease (see point 1.4.)

If tuberculosis is detected once among cattle in healthy districts, regions and republics, the farm is declared unhealthy and quarantine is announced, the number of unhealthy animals is completely replaced by healthy animals. . All animals in the herd (form) together with their calves are sent to slaughter within 30 days. After the herd of unhealthy animals is taken to the slaughterhouse, sanitation works are carried

out on the farm (disinfection, mechanical cleaning, sanitary repair works, deratization, final disinfection) and the quarantine is canceled, and the farm is filled with healthy animals. is filled.

Unhealthy birds are sent for meat, after the final veterinary sanitary measures are carried out, quarantine is carried out, the farm (shack) is considered healthy.

Review questions

1. Preliminary diagnosis of tuberculosis.
2. Tell how to perform the intradermal injection method.
3. Measures to prevent and eliminate tuberculosis

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: B rucellosis detection, prevention and control measures .

Purpose: To master preventive measures against brucellosis in a healthy farm

Basic concepts:

Brucellosis is a chronic zoonotic disease. The causative agent of the disease consists of species belonging to the genus *Brucella :*

1. *Br. melitensis* - type of sheep and goats. It is very dangerous for humans
2. *Br. aborlus bovis* - bulls type
3. *Br. suis* – pigs type
4. *Br. ovis* - causes infectious epididymitis in rams.
5. *Br. neotomae* - rats type
6. *Br. canis* is a type of dog

Brucella are small, polymorphous, non-spore-forming gram (-) bacteria. Nutrient media with added glucose, glycerin for growing them. D-nutrient medium, ploskirev agar is used. Differences in the growth and SO_2 production of Brucella in nutrient media supplemented with dyes .

TABLE 1

The causative agent of the disease	Fuchsin 1:25000	Thionin 1:25000	SO_2 formation
Br. Melitensis	+	+	-
Br. aborlus bovis	+	-	+
Br. suis	-	+	+

Diagnosis of brucellosis is based on epizootological data, clinical symptoms, allergic and laboratory examination methods. Blood is tested using serological reactions.

Complex diagnostic methods of brucellosis

1. Epizootological method (incidence of the disease, spread of the disease, transmission)
2. Clinical method (throwing, bursitis, orchitis, abscess in horses and pigs)
3. Pathanatomic method (abscesses in the liver, spleen, kidneys, blood in the fetus, necrotic nodules in pus...)
4. Allergic method (brucellin is injected under the skin palpebrally).
5. Laboratory test method:

a) Bacteriological method. Gram negative, stained by the Kozlovsky method. Grown on Ploskirev agar.
b) Serological method (RBP, RA, RSK. Milk ring reaction).
g) Biological test method (in guinea pigs, white mice) is carried out.

Differential diagnosis. Brucellosis should be distinguished from leptospirosis, campylobacteriosis, and trichomoniasis.

Table

	Inspection methods				
	Epizootology	Clinical	Allergic _ _	Bacteriological	Serological
1. Brucellosis	Sheep, goats, cattle, pigs, camels, deer are infected	Abortion, bursitis arthritis, orchitis abscess	K	Gr. Kozlovsky's method produces typical colonies	RPB, RA, KBR, reaction with milk
2. Campylobacteriosis	Cattle and sheep were infected	Metritis, vaginitis abortion			Gr. Action RA chan
3. Trichomoniasis	Mammals	Abortion (at 2-3 months), nodules are formed on the mucous membranes of the vagina (trichomonas vaginalis).			Polyforms 8-25 μm long are grown in Petrovsky nutrient medium

1. Measures to prevent brucellosis .

NEWLY imported animals are tested for brucellosis by serological methods, sheep and goats by allergic method, and rams by serological method for infectious epididymitis. If some animals in the herd show a positive result, if the diagnosis of

brucellosis is confirmed in them, all animals in this herd will be slaughtered. Black cattle with a negative result can be used in an unhealthy farm.

It is prohibited to keep animals bought for meat in dairy, breeding, and duck breeding farms and owned by farm employees.

It is forbidden to keep, feed and water sick animals in the same place as animals from unhealthy farms and healthy animals.

Animals are removed from the farm and sold only after 12 months and a year later. Animals vaccinated with brucellosis vaccine are not sold outside the country for breeding purposes.

Brucellosis-free farms must have a special health certificate issued by state veterinary inspectors.

All animals are checked for brucellosis.

A veterinary sanitary procedure is established for keeping animals.

If animals on the farm are suspected of brucellosis, farm managers , farm and animal owners should immediately inform veterinary specialist managers, farm and animal owners should quickly inform veterinary specialists, separate sick and suspected animals, and stop producing milk and milk products . and undertake not to give them to animals without neutralizing them.

The farm veterinarian examines unhealthy animals, sends pathological material to the laboratory for diagnosis and starts quarantine.

2. Preventive measures against B RUCELLOSIS.

If brucellosis is detected in X , QUARANTINE IS ANNOUNCED.

2.1. During the quarantine, farm managers should establish quarantine posts on the borders of the farm.

2.2. It is forbidden to enter and leave the quarantined farm, transfer animals from one group to another place, hold animal exhibitions, markets, take calves from sick animals, sell sick animals to private farms.

2.3. Animals with clinical signs that aborted and showed a positive result in the serological test are separated and given to meat for 15 days. It is forbidden to slaughter such animals on the farm.

2.4. Pastures where sick animals were raised can be used for healthy animals after 2 months in summer and 3 months in winter. Hay prepared in these pastures is fed to unhealthy animals after 2 months of storage.

2.5. It is forbidden to milk sheep and goats from unhealthy farms, to process non-disinfected skins, to make cheese and all products from their milk.

2.6. Wool from sheep and goats should be disinfected with bromomethyl alcohol.

2.7. Carcasses of dead animals, discarded fetuses are immediately burned (utilized).

2.8. The milk of animals with clinical symptoms is neutralized by adding 5% formaldehyde, creolin and rendered unfit for consumption.

2.9. When serologically testing for brucellosis, milk from animals with a positive result is boiled or turned into butter.

2.10. Milk obtained from healthy animals in unhealthy farms is pasteurized at 70 °C for 30 minutes at 85-90 °C or milk is boiled.

2.11. Animal hygiene and disinfection, disinsection, and deratization must be carried out in an unhealthy farm. 20% freshly slaked lime, DP-2 drug, 2% caustic sodium, 2% formaldehyde, 0.5% glutaraldehyde solutions are used for disinfection.

2.12. The manure and litter of animals suspected of having a disease are left in the fodder, or they are neutralized using biological, chemical-physical methods. (Biothermic method).

3. Making healthy farms healthy remedial measures.

3.1. When brucellosis is registered for the first time in a healthy region, republics, the rehabilitation of unhealthy farms is based on the complete (complete) replacement of animals in the herd with healthy animals. Cattle from neighboring farms and private farms are tested twice every 15 days by serological method. Vaccination is not carried out. In the farms where isolated sick animals are found, the above-mentioned measures are taken.

3.2. Remediation carried out in farms where the disease is widespread is based on the use of vaccines against brucellosis. Vaccination is carried out according to the instructions of the General Directorate of Veterinary Medicine of the Republic and the order of the Regional Veterinary Department.

3.3. In a herd with chronic brucellosis, sick animals identified by diagnostic tests are sent for meat within 15 days. After that, the animals in the herd are systematically checked every 15-30 days using the serological method until two consecutive negative reactions are obtained in the herd. After 2 negative serological results for the group, 6 months are left for veterinary control.

During this period, this herd of animals will be tested twice by serological method. If the test shows a negative result, if there are no abortions due to brucellosis in the animals, the animals in the herd are considered healthy. If during the control test (within 6 months) positive results are found, they are removed from the herd and sent for meat, and the remaining animals are subjected to systematic serological testing, or all animals in the herd, together with calves, are slaughtered for meat. If the farm is not cured by this method within two years, it will be replaced with animals from the unhealthy herd. Milk from animals on the farm being treated is sterilized (boiled or pasteurized, or made into butter).

3.4. In a farm where more than 25% of the total number of animals are sick, health care is carried out by the method of transferring all sick animals to meat. Animals with clinical signs are immediately transferred to meat, bulls are 8-year-old cows, and calves are delivered to meat for 3 months. In order to prevent abortion, all cows are vaccinated with a vaccine prepared from the 19th strain.

3.5. All the animals in the herd that are found to be sick in heifer breeding and dairy complexes are put to meat for 12-30 days, and the rest for 6 months. Before

quarantine, the livestock of this and neighboring farms, personal livestock are all checked by serological method (RA, RSK).

Review questions

1. Initial diagnosis in brucellosis.
2. List the causative agents of brucellosis.
3. Measures to prevent and eliminate brucellosis

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: diagnosis , prevention, and countermeasures of DIARRHEAL disease

Learning elements : K honey prevention, detection and documentation of inspection results.

X organization of treatment and implementation of countermeasures in cold sores.

Appendix 1

Brainstorming method

1. What do you know about rabies ? 2. The causative agent? 3. What is the economic damage in case of rabies . 4. What restraint measures will be taken.

Materials and equipment.

1. Microscope.
2. Test sample (taken from positive and negative skins)
3. Rabies antigen.
4. Ready-made ointment in the form of a capsule (must stand under a microscope for control, to show to students).

Purpose: To master rabies prevention and control measures.

Rabies - Latin name Lyssa, English - Rabies, Russian - beshenstva, all warm-blooded animals, birds and people are infected with the disease.

Rabies is an acute viral disease characterized by severe damage to the central nervous system.

All warm-blooded domestic and wild animals are susceptible to rabies, and the disease ends in death.

The causative agent is a round arrow-shaped neurotropic filterable virus belonging to the genus of RNA lussaviruses, belonging to the rhabdavirus family, the size of the virion is 100-150 μm.

The virus is very sensitive to lipid solvents (laundry soap), ether, chloroform, acetone and 70% ethyl alcohol, iodine and ammonium preparations. It is resistant to cold, it is stored in frozen brain for years, it is quickly inactivated at high temperature. It is quickly inactivated in 1-2% lysol solution, alkali, 2-3% formalin in 10 minutes at 60 o ^{C} , instantly at 100 o C.

Diagnosing the disease. The epizootology of the disease is studied, clinical signs and pathological-anatomical changes are taken into account.

Epizootological information. The following should be taken into account during epizootological investigations.

- When was the last time rabies was recorded in this area:
- What species of wild animals live and their distribution level.
- Cases of animals attacking each other and people:
- Abundance or scarcity of rodents:
- The number of dogs and cats in this area and their keeping conditions, their vaccination percentage:
- Whether or not there are garbage dumps around settlements.

Clinical signs **.** The disease is characterized by an acute course, and the incubation period is often 3-6 weeks.

Prodromal in the form of a disease that passes with symptoms of aggression. The stages of excitation and paralysis are different.

The silent or paralytic form often occurs when dogs are infected by foxes.

The atypical form of rabies in cattle lasts for a short time, mainly with symptoms of tympany, paralysis and paralysis.

Pathologoanatomical changes. The body of dead animals is emaciated, bitten areas are observed. Internal organs are filled with blood, non-nutritive foreign objects can be found in the stomach.

Basic concepts

Laboratory diagnosis of rabies. Laboratory diagnosis of the disease is important. Saliva of living animals is examined using fluorescent antibody method.

The heads or brains of dead animals are sent. In complex laboratory tests, the histological method of finding the inclusion bodies of "Babesh-Negri" from the ointment prepared from the ammon horn, the immunofluorescence method, the precipitation reaction in Agar Gel, the biotesting methods are used in young white mice.

If a positive result is obtained in any of these methods, the diagosis is considered established.

Differential diagnosis.

The disease must be differentiated from Aujeski's disease. Auyeski has an epileptic form in young piglets, and influenza (flu) symptoms in older pigs. In other species of animals, a strong reduction is observed. Sometimes he bites off small parts. Aggression is not observed and Babesh-Negri corpuscles are not found. Based on laboratory tests .

Treatment. There are no specific treatments for rabies. A person suspected of having rabies is admitted to an infectious disease hospital, immediately diagnosed with rabies, and mandatory anti-rabies gamma-globulin is administered. If the animal is found to be rabid, it is immediately killed and burned in a special furnace.

Disease prevention measures. To prevent rabies, farms, institutions, organizations and citizens should take measures.

- Owners of dogs, cats, and fur animals must comply with the rules for keeping them and carry out diagnostic examinations and preventive vaccinations within the time limits set by local authorities and veterinary institutions;
- Notify veterinary and medical staff when farm animals are bitten by dogs and wild animals or when rabies is suspected in animals;
- To prevent the entry of unvaccinated dogs, cats and wild animals into flocks, herds and ewes, measures are developed:
- All stray dogs, regardless of breed, must be captured or shot;
- Dogs and cats that have bitten people should be immediately brought to the veterinary hospital, undergo a veterinary examination and be under supervision for 10 days;
- Dogs being transported to other regions, countries and republics must have a veterinary certificate and the presence of a sign stating that they have been vaccinated against rabies;
- Creation of rabies immune zones. To do this, vaccinate all existing dogs in particularly unhealthy areas;

The following vaccines are available against rabies.

1. Dry inactivated ethanol vaccine (VGNKI). With this vaccine, dogs are vaccinated with a dose of 2 ml and cats with a dose of 1 ml. Immunity appears in 14-30 days and lasts for more than 6 months. After re-vaccination, immunity extends up to 2 years.
2. Dry culture vaccine prepared from "Shelkovo-51" strain. There is a special solvent RKAV (rastvoritel antirabichesky). For the purpose of prevention, previously unvaccinated animals are vaccinated 2 times in 3 weeks, and previously vaccinated animals are vaccinated 1 time.
   Immunity appears in 14-28 days and lasts for 2 years.
   Compulsory vaccination is mainly carried out in expensive animals. Vaccinated animals should be vaccinated without delay in 3 days. Large animals are vaccinated once for 3 days and in a dose on 16 days.
3. A cultured, lyophilized virus vaccine prepared from the TS-80 strain. The vaccine is dissolved in a physiological solution and animals are vaccinated

once intramuscularly starting from 3 months of age, the immunity lasts up to 1 year.

Rabisin vaccine produced in the USA is used worldwide.

Measures TO ELIMINATE DIARRHEA :

Farms, settlements, pastures and districts with rabies disease are declared unhealthy according to the decision of the authorities with the recommendation of the chief veterinarian. The chief veterinarian of the district prepares a plan of activities for the eradication of the disease and the authority approves it. According to this plan, veterinary health authorities and other organizations will carry out the following activities at unhealthy points.

- Identification of people, conditions of keeping dogs and cats, and animals suspected of having the disease in the unsanitary point of rabies;
- All infected dogs, cats and wild animals (except those biting humans and animals) are killed and burned;
- Wide awareness of disease prevention measures is carried out among the population;
- Administration of vaccinations and sera to farm animals, fur animals and dogs suspected of being affected;
- Vaccination of sick and suspected disease animals is prohibited;
- Consumption of milk of clinical animals from unhealthy farms after pasteurization at 80-85 ^{o} C or boiling for 5 minutes;
- The last time the disease was registered is taken into account when determining rabies-free districts. An area is considered unhealthy if less than 2 years have passed since the last rabies registration.

Review questions:

1. What methods are used to determine rabies?
2. Describe the laboratory test?
3. Do we use regular vaccinations for rabies?
4. Measures to prevent rabies?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Diagnosis, prevention and countermeasures of Pasterelly 's disease .

The goal: To master the methods of disease detection and to learn how to formulate anti-disease measures using guidelines.
Materials and equipment:

Provision of training equipment: slides, pictures, tables, instructions and biological drugs.

training is in the classroom and stationary of the department.

Pasteurellosis (Lat., English - Pasteurellosis; Russian - hemorragicheskaya septicemia) is a contagious infectious disease of mammals and birds, acute hemorrhagic septicemia, croupous pneumonia, pleurisy and watery swelling in many parts of the body. , semi-acute and chronic - characterized by purulent necrotic pneumonia in the lungs, kerato-conjunctivitis, arthritis, mastitis and hemorrhagic enteritis.

Driver. Pasteurella multocida is a small, gram-negative, non-motile, non-spore-forming bacterium, which often resides singly, in pairs, and in some cases in chains. All are dyed with aniline dyes. Pasteurella are small, ovoid in the tissues of diseased animals (0.3-1.25 x 0.25-0.5 μm). They are stained with bipolar methyl zinc or Gimza Romanovsky dye. Forms a capsule in fresh culture.

grows as a facultative aerobic, usually liquid, solid nutrient medium at 37 ^{0} C. As bacteria grow, the broth becomes cloudy. Colonies are observed in GPA in 3 forms: smooth S, rough R and mucoid M. Fermentative properties are weak.

There are 4 immunological serogroups of P. multocida based on capsule antigens: A, V, D and E. Their pathogenicity depends on the type of animal. M: **D** strain in all species of animals; **V, E strains** - more in cattle; Strains of type **A cause more disease in poultry and less in pigs and cattle.** P. haemolytica species causes enzootic pneumonia in calves.

There is a correlation between the virulence of Pasteurella and the production of a capsule and toxin (endotoxin with lipopolysaccharide). Epizootic strains of Pasteurella are highly virulent for white mice.

Exciter endurance. Pasteurella die quickly in natural conditions. Lives in manure, blood, cold water up to 2-3 weeks, in dead bodies up to 4 months, in frozen chicken meat up to 1 year. It dies in a few minutes under direct sunlight, in 5-10 minutes at 70-90°C. Disinfectants at usual concentrations quickly inactivate Pasteurella.

Epizootological data. All types of domestic and wild mammals and poultry are susceptible to pasteurellosis. This disease also occurs in humans. Among chickens and rabbits, the disease usually appears as an epizootic. Lesser epizootics are observed in other species of animals. Horses and carnivorous animals are more resistant to pasteurellosis.

of hemorrhagic septicemia in large young cattle and wild ruminants is caused only by **the V** strain of *P. multocida , in Africa by* **the E** strain, and in poultry by **the A strain.**

Sporadic disease, semi-acute and chronic enzootic pneumonia is caused by *P. multocida* strain **A and** *P. haemolytica in calves* , and strains **A, D** and *P. haemolytica in pigs* .

Pasteurella is mainly inhaled and enters the body through the alimentary route, through the mouth, nose, eyes, digestive mucous membranes.

The source of the disease is the sick and recovered Pasteurella carriers. Pasterella *shelf life* is 1 year. It is typical for pasteurellosis that healthy animals carry the pathogen for a long time. Many researchers conclude that a case of pasteurellosis on a farm is caused by a newly introduced Pasteurella-carrying animal.

Cattle and sheep are affected at all ages, but young ones are more susceptible. Buffaloes are more sensitive than cattle and die 2 times more often.

A characteristic feature of the disease is its *enzootic nature* and *the appearance of a stationary epizootic focus* is considered *to do* .

In tropical countries, epizootics spread during the rainy season and mortality is high (70-100%). In average climatic conditions, the disease is observed in autumn and spring (1-53%). The diseased animal releases the pathogen with the liquid from the nose, exhaled breath, saliva, and feces.

A building, air, hay, bedding and inventory contaminated with pasteurella from a sick animal serve as a factor that spreads the disease.

In addition to the factors mentioned above, ticks in poultry - *Dermanisus galini, Argas persicus* are important because the causative agent lives in ticks for more than 60 days. Rats and mice also serve as disease spreaders in unhealthy poultry farms. In the spread of the disease, the mixing of the animal group, close keeping, veterinary and sanitary conditions on the farm play an important role.

Morbidity, mortality depends on the virulence of Pasteurella, the immunological state of the herd, the keeping and feeding of animals, and the timely implementation of health measures.

Changing the place of animals without taking into account the health of the farm in terms of pasteurellosis, dense keeping, violation of veterinary-sanitary rules, use of non-detoxified waste products play an important role in the spread of this disease.

Pathogenesis . Under natural conditions, Pasteurella enters the body through the respiratory and alimentary routes, rarely through the skin. Pasteurella multiply at the point of entry, enter the blood and lymph and cause septicemia, death occurs within 12-36 hours. Due to septicemia and toxic substances produced by Pasteurella, the activity of phagocytosis decreases sharply, it is not full, toxic substances destroy capillaries, edema and hemorrhagic diathesis are observed in a large area under the skin. The higher the virulence of the pathogen, the faster septicemia occurs. Septicemia does not develop in animals resistant to the pathogen and when less virulent Pasteurella enter the body. In them, the disease is semi-acute and chronic, the causative agent is located more in the lungs, and croupous or serous catarrhal inflammation occurs. In acute and acute cases, croupous pneumopia does not have time to develop, so swelling and hyperemia appear in the lungs.

Course, clinical signs and forms. The latent period lasts from several hours to 2-3 days. It depends on the age of the animal, resistance, amount and virulence of the pathogen. The disease is *very acute* in cattle and buffaloes . They suddenly have a fever up to 41-42 ^{0} C, depression, heart failure or watery edema in the lungs in a few hours, and in some cases die without clinical symptoms.

The disease *is acute* later, they have general depression, lethargy, anorexia (not eating), fever up to 41 $^{0\ C.}$ Nasal glass is cold and dry, mastication and feeding stops, bowel movements and defecation slow down at the beginning of the disease, feces become liquid, sometimes blood, even blood from the nose, conjunctivitis and blood in urine are observed. Animals die in 1-2 days with septicemia, heart defects.

The disease is *semi-acute* In addition to fever, local changes are in 3 *forms* **:** *edema (watery swelling), chest and intestine* ***takes place*** *in forms* .

edema , there is a rapidly growing, painful, hotter fluid accumulation under the skin of the lower jaw, neck, abdomen, and legs. If this phenomenon occurs in the tongue and throat, wheezing and breathing becomes difficult, water flows from the nose and sticky saliva from the mouth; there are many hemorrhages in the mucous membrane.

chest - croup - fibrinous pneumonia, depression, anorexia, atony, breathing difficulty and acceleration, painful cough, serous, vesicular discharge from the nose are observed. At the end of the disease, bloody diarrhea is observed and death occurs in 5-8 days.

the intestinal form, the main symptoms are characterized by severe damage to the digestive system, pneumonia symptoms are not well manifested, but developing anemia and general weakness are observed.

the disease is *chronic* , symptoms in the respiratory and digestive organs are weaker than in the intestinal form, but diarrhea leads the animal to cachexia.

Acute septicemia is rarely observed *in sheep* . Fever, depression , watery swelling under the skin of the jaw, neck, abdomen and pleuropneumonia are observed. A sick sheep will die in 2-5 days.

In pigs If it is very acute and *acute* , the fever rises to 41 ^{0} C, pharyngitis, heavy breathing, heart failure, swelling under the jaw, neck, and under the skin is observed and in 1-2 days dies of asphyxiation. If the disease lasts for a long time, pleuropneumonia with fibrin, rapid breathing, cough and purulent rhinitis are observed, and death occurs in 5-8 days. Symptoms of *chronic* late-pneumonia: weakness, constantly developing cachexia, in some cases arthritis - joint swelling is observed. *Semi-sharp,* when *chronic* long persistent fibrinous pneumonia, keratitis, serous rhinitis, arthritis and cachexia are observed.

In poultry, it is very acute at the beginning of the epizootic . They suddenly fall, flap their wings and die without any clinical signs. Often the disease is *acute* . They are loxas, their wings are down, their feathers are ruffled, their heads are under their wings or bent back. Body temperature rises to 44 ^{0} C and above, anorexia and severe thirst are observed. A bubbly mucous fluid flows from the nose and snout. Then there is diarrhea, in some cases bloody diarrhea. In some birds, the crown becomes swollen and hard. Later, abscess and necrosis are formed. When

pasteurellosis becomes chronic, symptoms of rhinitis, sinusitis, and exudate accumulation around the nostrils and eyes appear. The crown and earring are blue, it becomes difficult to breathe and it dies wheezing and struggling. *Semi- sharp,* if it is *chronic* , anemia, cachexia, arthritis and ear swelling will occur (Fig. VIII).

In rabbits in *acute* cases, the body temperature rises, malaise, anorexia, flu, sneezing, and in some cases diarrhea are observed, and they become weak and die in 1-2 days. In stable healthy farms, pasteurellosis is chronic and symptoms of rhinitis and conjunctivitis appear. Rarely, diarrhea, fibrinous-purulent pneumonia, and abscesses under the skin are observed.

In fur-bearing animals, when the disease is *acute* , there is sudden weakness, anorexia, slow and shaky walking, and an increase in body temperature to 42°C. Symptoms of hemorrhagic gastroenteritis develop *in foxes* . *In black cousins* , swelling occurs in the head area, subcutaneous tissue, and hind legs are paralyzed. The disease lasts from 12 hours to 2-3 days.

Pathologoanatomical changes. These changes depend on the duration and form of the disease. In acute and late acute cases, hemorrhagic diathesis is seen in dead animals (hemorrhage and inflammation in organs, mucous and serous membranes), liver and kidney are damaged, spleen is slightly swollen, lymph nodes are swollen , dark-red, serous-fibrinous infiltrates are visible in the subcutaneous tissues, especially in the edematous form of the disease, in different parts of the body. Pulmonary edema is a change to the initial stage of croupous pneumonia. Fibrinous-hemorrhagic inflammation is seen in the gastrointestinal tract in the intestinal form. In semi-acute and chronic cases, dead animals are emaciated and bloodless, the pre-bronchial lymph nodes are enlarged, reddened, and contain a lot of blood. Foci of necrosis are visible in the lungs. The spleen is slightly enlarged, there are small necrosis foci in the liver and kidneys. Pathologoanatomical changes in birds are similar to those of mammals and mainly depend on the course of the disease.

Diagnosis . Pasteurellosis is diagnosed on the basis of clinical signs, epizootological data, pathologoanatomical changes and, of course, the results of bacteriological examination (bacterioscopy and isolation of Pasteurella culture). Spleen, liver, kidney fragments, damaged lung fragments, lymph nodes and tubular bone are sent to the laboratory. These feather materials should be taken after the death of the animal, no later than 3-5 hours and without treatment. Small animals are sent whole. In the summer months, the feather material is preserved in a solution of 40% glycerin in water and sent to the laboratory in a cold state by a letter of reference.

Differential diagnosis. Anthrax, piroplasmidosis, and rabies in adult cattle; in young animals from staphylococcal and streptococcal infection, salmonellosis, colibacteriosis and respiratory infections (parainfluenza-3, infectious rhinotracheitis), enzootic bronchopneumonia; from plague, distemper and salmonellosis in pigs; from anthrax, piroplasmidosis, clostridiosis and streptococcal infections in sheep; and in chickens it should be distinguished from Newcastle disease, spirochetosis, mycoplasmosis and infectious laryngotracheitis. The results of a complex laboratory examination form the basis of the final diagnosis.

Treatment . Sick animals are separated in warm, dry buildings, provided with nutritious feed. Modern antibiotics are used after determining the sensitivity of the pathogen to them, and drugs with sulfonamide and nitrofuran are used.

Hyperimmune blood serum against pasteurellosis is useful if it is used at the beginning of the disease when the disease is acute, when the first clinical symptoms appear (XSSalimov, BA Elmurodov, SA Abdalimov). Serum is administered subcutaneously or intravenously in two prophylactic doses (1.5-2 ml/kg). If the serum is added together with antibiotics, sulfanilamide preparations, the treatment effect increases. The course of treatment depends on the condition of the animal. Poultry infected with pasteurellosis are not treated.

Immunity . Animals that have recovered from pasteurellosis have immunity for 6-12 months. Formal *vaccine* for vaccination of cattle, sheep and pigs for active immunity (N. Nikiforov), GOA formal vaccine VITI (BA Elmurodov, SA Abdalimov), radiovaccine against polyvalent pasteurellosis, salmonellosis, colibacteriosis (VITI - RUBulkhanov, IV Ryasnyansky) against pasteurellosis and diplococcosis formalvaccine against GOA (VITI - BA Elmurodov) and emulsin vaccines for vaccination of pigs are used based on the Instructions for their use. Immunity lasts for one year.

Dry live and inactivated vaccines prepared from avirulent pasteurella imported from France and weakened in Russia are used to vaccinate poultry in dangerous areas. Immunity appears in 5 days and lasts for 6 months. 4 days after vaccination, sulfadimycin or norsulfazole is given to the birds.

For passive immunity, hyperimmune blood serum is used only for prevention. Used for newly imported goods.

Vaccines are forcibly used for preventive purposes, in unhealthy farms and in dangerous areas. Dry, live vaccines are used to prevent pasteurellosis in poultry. Live vaccines are used to vaccinate chickens and waterfowl from unhealthy farms and dangerous areas. Immunity is formed after 5 days and lasts for 4-6 months. Emulsin vaccine is used in dangerous and stagnant unhealthy farms. On the 4th day after vaccination, sulfadimyzin or norsulfazole is added to the grain of all poultry for 3-4 days. Immunity appears on the 8th day and lasts for 6 months in chickens and 7 months in waterfowl, after which the 2nd time is vaccinated without the use of sulfonamides.

For passive immunity, hyperimmune blood serum against pasteurellosis for cattle, buffaloes, sheep and pigs is administered to large animals for preventive purposes before transportation, and to young animals (calves, piglets, lambs) on the first day of arrival at livestock complexes.

Prevention . Only buying animals, poultry and feed from healthy farms for pasteurellosis, keeping animals in pastures in the summer, feeding them with nutritious feed, and not letting other animals and strangers into the farm will make it possible to prevent this disease. If this disease was previously detected in the farm, all animals should be vaccinated against pasteurellosis throughout the year, and only vaccinated animals should be admitted to the farm. Arriving animals must be under preventive control for 1 month.

Countermeasures. Pasteurellosis among cattle, pigs and sheep-goats in the farm (farm, herd, herd) in the case of laboratory detection, on the basis of the certificate of the chief veterinarian of the district (city), by the decision of the authorities, this area will be declared unhealthy for this disease and a *restriction* will be imposed on it. All animals are clinically examined, sick and suspected animals are separated and treated, and the rest are vaccinated against pasteurellosis. Current disinfection is carried out when each sick animal is detected and every 10 days until the restriction is lifted. Dead animals are cremated. After all animals are vaccinated and final disinfection is carried out, *the restriction* is lifted after 14 days.

In poultry farms, even if pasteurellosis is detected in a laboratory method, restriction is established in the above-mentioned manner. Sick and suspected disease birds are killed and cremated. Sometimes all the chickens in an unhealthy coop are killed. Eggs are disinfected in formaldehyde vapor. Suspected infected birds are separated, healthy ones are vaccinated. When the disease is widespread, antibiotics and sulfonamides are urgently given to healthy birds before vaccination. After all the sick birds are lost, the premises are cleaned, disinsection, deratization and final disinfection are carried out, and its quality is bacteriologically checked, the *restriction* is canceled.

Strict containment measures are also carried out in rabbit farms that are unhealthy for pasteurellosis. Sick rabbits are killed, buildings and cages are disinfected. All healthy rabbits are injected intramuscularly with 20 ml of terramycin per 1 kg of live weight or biomycin 2 times with an interval of 8-10 hours, and after 24 hours, rabbits older than 45 days are given GOA formalvaccine against pasteurellosis with an interval of 7 days. is vaccinated twice. When all sick rabbits are lost in the farm, the premises are cleaned, disinsection, deratization and final disinfection are carried out, and its quality is checked bacteriologically, and after 14 days after the last disease was detected, the *restriction* is canceled.

If pasteurellosis is detected in fur farms by laboratory method, animals are provided with high-quality feed and for the purpose of prevention and treatment, antibiotics and special hyperimmune blood serum against this disease are used according to the Instructions for its use. If cockroaches and nutria (water rats) are infected, healthy animals are vaccinated with emulsin vaccine against pasteurellosis.

Review questions

1. Methods of determining the disease ?
2. Differential diagnosis of the disease ?
3. Disease control measures ?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA , Salimov I. X. , "Epizootology and Infectious Diseases " Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Necrobacteriosis detection and control measures

The goal: To master the methods of disease detection and to learn how to formulate anti-disease measures using guidelines.
Materials and equipment:

Provision of training equipment: slides, pictures, tables, instructions and biological drugs.

training is in the classroom and stationary of the department.

• Necrobacteriosis - Necrobacteriosis is an infectious disease of domestic and wild animals, characterized by the appearance of necrotic lesions on the skin, mucous membranes, and internal organs.

• The causative agent is Bact . necrophorum does not form spores and capsules. It is stained by strict anaerobic, Sil fuchsin, Lefler, Muromsev methods. It occurs in filamentous cocci and small rod-shaped forms. Kitt – Grows well on Tarossi and serum blood agar.

• Durability – can survive in frozen environments for up to 3 weeks. It is stored in manure for 40-50 days and in urine for 15 days. It lives in the soil for up to 2 months.

Basic concepts:

Complex methods of diagnosing the disease:

1. *Epizootological method* . In this, attention is paid to the previous epizootological condition of the farm, keeping, feeding and use of animals. Wounds are the gateway to necrobacteriosis. The causative agent of this disease lives in soils with a lot of moisture. When animals are raised in marshy pastures, the coagulation of the skin of the legs and hooves creates conditions for the disease. Dry tussock pastures, rocky mountain and foothill pastures are good factors for the spread of the disease.

2. *Clinical method.* It is characterized by damage to the mucous membranes of the legs, head, face, oral cavity, genital organs, purulent necrosis and a peculiar smell.

3. *Laboratory method.* To confirm the diagnosis, a sample is taken, a smear is prepared and examined under a microscope. Smears are painted in the usual way. Diagnosis is made by looking at long, thread-like, gram-negative rods under a microscope. Artificial nutrient media are cultivated and bioassays are performed on laboratory animals, white mice and rabbits, to determine the virulence of the pathogen.

Differential diagnosis

Necrobacteriosis disease should be distinguished from hoof rot in sheep, protein, smallpox, streptococcal, polyatritis, plague in cattle, viral diarrhea, poor-quality catarrhal malaria, scurvy and dermatitis.

Treatment of sick animals

Sick animals are treated individually or collectively. The treatment is carried out in a specially equipped place with a dry floor, protected from wind, rain and sunlight.

The damaged area is cleaned of dead tissues and treated with antiseptic solutions, potassium permanganate, hydrogen peroxide, copper sulfate, and sulfanilamide preparations or antibiotics tetracycline or penicillin are sprinkled. Treatment with dibiomycin gives good results. Dibiomycin 15% oil solution to the affected area. At the same time, dibiomycin suspension in the form of 30% glycerin and 0.5-1% novocaine solution is used for general treatment. This suspension is injected into the thigh muscle at 20,000 TB per 1 kg of live weight. The drug is sent a second time after 6-8 days. Dibiomycin can be given orally, 6-5 g to deer, 3-4 g to 6-month-old calves. When the mucous membranes of the mouth are damaged, 3% hydrogen peroxide or copper sulfate, iodine solution, 3% carbolic acid solution are used. When the skin of the lips is disinfected, iodine glycerin and zinc ointment are applied.

PREVENTIVE MEASURES FOR NECROBACTERIOSIS

It is necessary to increase the natural resistance of fat animals to the disease and carry out comprehensive measures to neutralize the causative agent of the disease in the external environment. Timely detection of the disease, isolation and treatment of sick animals . In order to prevent necrobacteriosis, it is necessary to carry out measures to drain watery, swampy pastures, perform a veterinary examination and clean the hooves of animals every two months, and twice a year to clean the hooves of small horned animals by 10% It is necessary to treat with formalin solution or 5% paraform solution. Do not water the animals from ponds and puddles, trim the wool around the udders of lambs and wash their udders with soap in hot water. It is cleaned with 35% bornic acid, 12% lysol or KmnO4 solution: the navel of newborn lambs is treated with iodine solution, if there is a sign of lameness, the animals should be isolated and treated. If necrobacteriosis is detected in animals in farms (farms, departments, herds) or settlements, it is considered unhealthy and restrictions are imposed. All animals undergo clinical examination, sick animals are isolated and treated. The rest of the animals in the unhealthy herd (healthy or conditionally healthy) are transferred to unused pastures, or kept in buildings with dry floors and drains. Conditionally healthy animals are examined every month in order to detect, isolate and treat them during illness. The milk of these animals is boiled and consumed, the milk from sick animals is discarded. Animals that died from necrobacteriosis are skinned and burned or sent to a slaughterhouse. If it is agreed to slaughter sick animals, they will be slaughtered in sanitary aviaries. These animals are transported in specially equipped vehicles. The restriction is removed from the farm (population, department, horses) one month after the final disinfection, when the

last sick animals recover or die. For necrobacteriosis, special vaccines for sheep and cattle have been prepared and are being used with great success. Until now, live vaccines have also been recommended for cattle and sheep.

Review questions

1. Diagnosis of necrobacteriosis and treatment of sick animals?
2. Differential diagnosis of necrobacteriosis?
3. Measures for necrobacteriosis?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA , Salimov I. X. , "Epizootology and Infectious Diseases " Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Leptospirosis diagnosis, prevention and countermeasures .

PLAN:

1. To prevent, identify and document the results of inspections .
2.X organization of treatment and implementation of countermeasures.

Purpose: To learn how to identify the disease and develop anti-disease measures using guidelines. The place of the training is in the study room and stationary of the department. Complex diagnostic methods are used to detect leptospirosis.

Basic concepts, diagnosis

1. *Epizootic method* . It should take into account the previous epizootological status of the farm, the incidence of human disease, the natural focus of the disease, the presence of rodents, the seasonality of the disease, and natural geographical factors.
2. *CLINICAL method* . Clinical signs of the disease include yellowing of mucous membranes, necrosis of the skin, especially in the muzzle and ear tips, blood in the urine, lack of appetite, short-term diarrhea and constipation, and atony in the gastrointestinal tract. such signs are of great importance in identifying the disease.
3. *Pathanatomical method* . Pathanatomical changes include yellowing of the entire mucous membranes and subcutaneous tissue, the presence of blood clots in the serous and mucous membranes, enlargement of the kidney, dark brown color, hemorrhages in it, solidification of food masses in the large abdomen, especially in the colon. , liquefaction and poor coagulation of blood are among the changes in this disease.

4. *LABORATORY method.* The bodies of discarded fetuses, livers and blood from sick animals are sent to the laboratory for examination. It is recommended to send blood for bacteriological examination after the 8th day of the disease. It is also recommended to send urine from pigs because the disease is latent in pigs.

DIFFERENTIAL diagnosis .

The disease should be distinguished from similar diseases, i.e., piroplasmidoses, brucellosis, campylobacteriosis, catarrhal fever, etc.

D drive. Treatment of the disease is effective in such cases, that is, the treatment should be carried out in the initial period of the disease, and in addition, special immune globulins, hyperimmune serums, antibiotics should be used, or symptomatic methods of treatment should be used. Leptospirosis hyperimmune blood serum for cattle 50-120 ml, for calves 20-40 ml sheep-goats, for pigs 5-70 ml, subcutaneously. Steptomycin is administered intramuscularly at 10-12000 IU per 1 kg of weight. In cattle, streptomycin and polyomycin are used in the usual doses. For pigs, tetracycline, tetramycin, biomycin are added 1 kg per 1 ton of feed. Biovetin, biovetin 40, biovetin 80 is added half kg to one ton of feed. This process takes 14 days. Uramycin is injected at 4-10 ml of gram per 1 kg of weight. Glauber's salt is recommended for an upset stomach. Large animals up to 500 grams. Small goods up to 100 grams. Caffeine is injected under the skin. Large animals up to 5 grams, small animals up to 2 grams. Oral cavities are treated with a 1:1000 solution of potassium permanganate. For necroses of the skin, ixtiol oil and baby vaseline are recommended.

Measures to prevent and eliminate HONEYDEW .

Measures to prevent and eliminate leptospirosis should consist of two parts. The first part is aimed at ending the complications of the disease in livestock farms by eliminating the source of the disease, preventing the disease from appearing anew. The second part is aimed at full rehabilitation of the farm.

1. *PIEZOOTOLOGICAL method* . Many domestic and wild animals are susceptible to this disease, in natural conditions, sheep and rodents are more affected, and listeriosis appears among them in the form of an epizootic. In most cases, this disease is more common in winter and spring seasons. This disease is characterized by the stability and naturalness of epizootic foci. The incidence rate among sheep is 0.5-5%, in some cases it increases to 20%.

2. *CLINICAL method* . The animal's body temperature rises (40-41C) . the general condition worsens, signs of damage to the central nervous system appear, confusion, confusion, and depression alternate with agitation. In this case, the animal moves forward or in a circle without stopping, falls, trembles and has seizures. Sheep throw their neck back and swim in the water, then paralysis occurs and the animal dies. Sometimes miscarriage, retention of the placenta, and endometritis are observed in the second half of the esophagus.

3. *ANATOMICAL method* . Although pathanatomical changes are not characteristic of the disease, swelling of the brain, filling of blood vessels, hemorrhages, catarrhal inflammation of the intestinal-stomach mucosa, heart and

parenchymatous organs, hemorrhages, enlargement of the spleen, and necrotic points in the liver, spleen, and lymph glands. such changes should not be overlooked.

4. *LABORATORY method.* For bacteriological examination, the body of a freshly dead animal or the material obtained from it (brain, liver, spleen) is sent to the laboratory. The material is cultured in brain and liver broth with glucose glycerin, agar (1% glucose, 2% glycerin) and placed in a thermostat at 35-37 g C ^{o}, after 3-4 days listeria formation appears. Listeriosis is stained positive by the Gram method.

Biotest . A 25% suspension is prepared from the pad material using a sterile physiological solution, and 0.05-0.2 ml is injected into white mice, 0.5-1.5 ml into the guinea pig and rabbit into the brain (intracerebral) or intraperitoneally into the abdominal cavity. After 3-10 days, laboratory animals die.

Differential _ diagnosis : Diagnosis in putting _ rabies , Auyeski 's disease , senurosis , brucellosis Campylobacteriosis differentiate need _

Review questions

1. Tell the diagnosis of leptospirosis.
2. Differential diagnosis of leptospirosis.
3. Measures to prevent and eliminate leptospirosis.

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Diagnosis , prevention and treatment of T -emirate disease combat measures . _

Temiratki is an infectious disease that occurs in farm animals (cattle, sheep-goats, camels, goats, fur-bearing animals, dogs and cats, etc.) and humans. The appearance of wounds (spots of various shapes) on the skin is the main symptom of the disease.

Triggers . The causative agents of the disease are fungi (Trichophyton verrcosome, T. Eqvinum, T. Mentagrophytes, T, Sarcisovi, M. Eqvinum, M. Canis), which can live in buildings and barns for several years. , fences, doors, wood and the edges of containers can easily maintain their virulence for 2-3 years. When the material taken from a sick animal is examined under a microscope, arthrospores are seen in the form of semi-glossy spherical balls arranged in rows or irregularly around the affected wool fiber.

The causative agents of the disease mainly grow well on wort-agar, MPGA with 2% glucose with meat-peptone and Saburo agar, after 3-4 weeks fluffy fluffy colonies appear. Depending on the type of fungus and animal, they grow as leathery,

brown, yellowish fluff when cultivated. When separated, microconidia and chlamydospores differ depending on their condition.

Diagnosis. The disease is diagnosed by a complex examination method. Epizootological information, clinical signs of the disease and the results of mycological examination are taken into account.

Pathological material and its classification . Pathological material is taken from the injured area of a diseased animal. Q-10 pieces of damaged wool fiber are taken and placed on a glass with black paper underneath. Then the product is placed in a window, dripped with 10-15% alkali solution and slightly heated. Checking under a microscope, attention is paid to the location of arthrospores. Plants are grown in the previously mentioned media and growth is monitored .

Treatment. For treatment, the LTF-130 vaccine is given in the same scheme as above, but the dose is increased by 2 times. If the animal is seriously ill and does not recover quickly, it is possible to administer the vaccine a third time in a therapeutic dose.

In the summer months, a mixture of 7% alkali and formalin prepared in petroleum jelly gives good results. It is applied every 5-6 days.

The taste of the mixture of "Yam" oil is high. If available, a mixture of 1.5% lye can also be used.

Prevention. In healthy farms, all calves are given 2 prophylactic vaccinations at 10-14 day intervals after reaching the age of 1 month.

All veterinary and sanitary measures are carried out on time.

Sick animals are isolated and treated according to the instructions.

- Cattle breeders must strictly follow the rules of personal hygiene .

A mixture of alkali with formalin (2 l of alkali, 5 l of formalin and 100 l of water) is the most effective for disinfection. When the disease is registered, restriction will be announced.

Basic textbooks and study guides

1. Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
2. Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
3. MP Parmanov et al., "Epizootology" training manual. T, 2007.

Exercise: Diagnosis , prevention and countermeasures of the disease of cattle .

Learning elements : Identifying the disease, treatment, prevention of the disease, implementation of countermeasures and studying the results of investigations.

Basic concepts on the topic : Agricultural and laboratory animals, vaccine, guidelines, developments.

Appendix 1

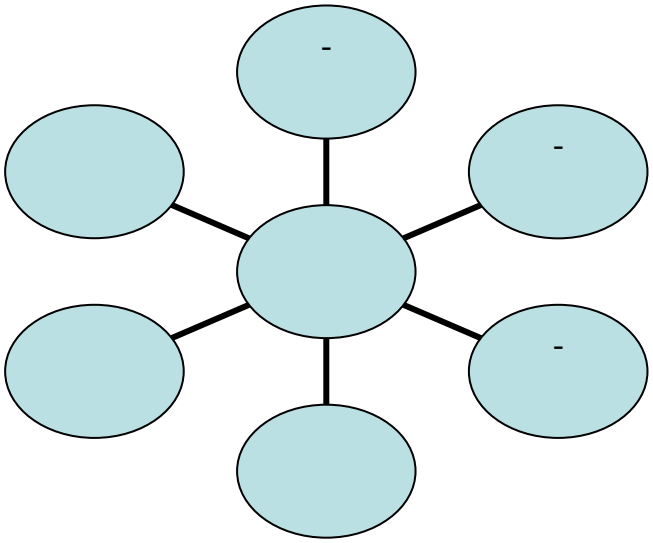
Brainstorming method

1. What kind of animals are affected by the disease.
2. Who identified the pathogen.

3. How resistant is the causative agent in the case of tuberculosis.
4. What is the degree of susceptibility to disease in animals.

Appendix 2

Cluster method



Appendix 3

" WORKING IN SMALL GROUPS" .

Working in small groups ensures students' activeness in the lesson, gives everyone the right to participate in the discussion, gives the opportunity to learn from each other in the audience , and teaches to value the opinion of others.

Basic concepts

BLACKLEG - (Gangraena emphysematosa, Blackleg, emphysematous carbuncle, emkar). It is an infectious disease of ruminant animals (cattle, sheep, goats, deer, deer), characterized by fever and crepitation in the muscular and fleshy parts of the body, with a well-defined swelling. limping, characterized by rapid death.

Cl. Chauvoei - a straight or slightly curved (2-8 μm), single or double, spore-forming, mobile rod.

It is well stained by the Gram method with alkaline-aniline dye. Spores can be painted in any way. The causative agent is strictly anaerobic.

The microorganism emits an odor when applied, similar to the odor of rancid oil during growth.

Pathogens are found in soil, manure, mud, and intestines of cattle, sheep, and horses.

Gram+ in young cultures, Gram - in old cultures, and in anaerobic conditions in the soil, it forms a spore larger than the thickness of the microbe, located in its center or at its tip. Does not form a capsule.

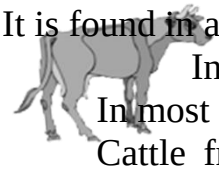
Pathogens are found in soil, manure, mud, and intestines of cattle, sheep, and horses.

Durability - Clostridium spores are very resistant. It will die in 5-10 minutes at 100°C. It can live up to 2 hours at 80°C. It can be stored for several years in dried meat, and up to 6 months in rotten material. Clostridium can be stored in sea water for up to 6 months. The pathogen can remain viable for a long time in manure, burial, and uncontrolled litter. In order to maintain its virulence, the calf and

is carried out by infecting the organism of guinea pigs .

Karason with cattle and sheep breeding of the world

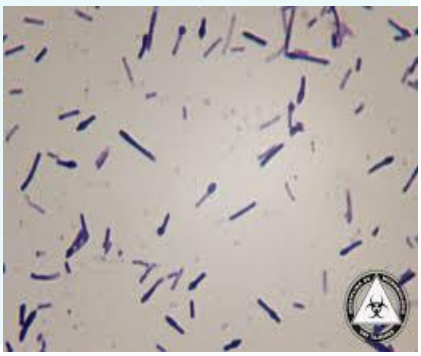
It is found in all countries where it is practiced.



In the Republic of Uzbekistan, blackbirds are also common.

In most cases, it is observed in sporadic, sometimes enzootic distribution.

Cattle from 3 months to 4 years old are highly susceptible to the disease. Adults develop immunity gradually throughout their lives, but it is not absolute.



Purebred cattle are more susceptible than others. The degree of susceptibility is evident when cattle are brought from a healthy farm to an unhealthy one, and the disease is very severe and destructive. Death - up to 80%.

In all seasons, but more common in autumn. Because in the fall, the grass dries up and they scratch the mucous membranes.

According to the conclusion of most scientists involved in the study of the disease, transmission occurs mainly through the oral cavity (peroral). In natural transmission, spores enter the animal body mainly with food and water. Later, it passes through the injured or scratched area of the mucous membranes and reaches deep tissues.

Fat cattle are often infected with scurvy, and the disease is very rare among lean cattle. The main reason for this is the presence of a large amount of glycogen in the muscles. Glycogen, in turn, is the best source of nutrients for Clostridium shavoye. The causative agent can be isolated from the body of a freshly dead animal. Because bacteremia is observed in the body before death.

Clinical signs. The latent period of the disease lasts 4-5 days, is acute and usually ends in death. The disease begins with a

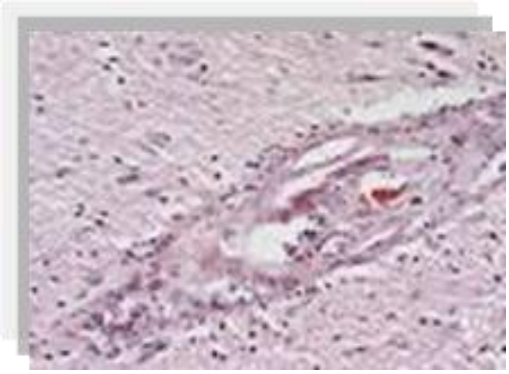
sudden rise in body temperature (40-42°C). Diseased cattle become lame. In the most fleshy parts of the body (thighs, thighs, etc.), a strictly limited, hot and hard swelling appears.

The sick animal becomes extremely weak, does not eat anything, lies down, it becomes difficult to stand up, and carefully presses on the side of the lame leg. Respiratory and cardiovascular systems are disturbed. The sick animal will die in 1-2 days due to hypothermia, as the blood vessels slow down. If the disease is septicemic, 3-4 month old calves develop a general fever and die within 5-10 hours.

Certain changes also occur in the blood composition of a sick animal. According to Ya.R. Kovalenko, the amount of erythrocytes will decrease significantly.

Pathologoanatomical changes. Manifestations of reddened mucous inflammation and limited hemorrhagic necrotic myositis in the connective tissue between the subcutaneous muscles are characteristic changes of the disease.

The injury occurs mainly in the thigh, neck, shoulder, chest, abdomen, rarely in the throat and diaphragm, tongue and myocardium.



When cut, it can be felt that there are air bubbles and a dry and porous structure.

Sometimes the mucous membranes are covered with a soft thin layer of fibrin, in some cases the spleen is slightly enlarged and softened, protein and fatty dystrophy is visible.

Among the muscle fibers, germ cells, hemorrhages and air bubbles are noticeable.

Laboratory tests . The fluid taken from the wound site, muscle, spleen fragments, blood from the heart are sent to the laboratory by registered letter within 2-3 hours

after the death of the animal.

Bacterioscopic and bacteriological tests are carried out in the laboratory. Bioassays are conducted on guinea pigs. They die after 18-48 hours. Individual or combined triggers are seen in the liver smear.

It is necessary to distinguish the disease from anthrax and malignant tumor.

In anthrax, there is no crackling gas.

Malignant swelling occurs after an injury. In all cases, laboratory tests give an accurate diagnosis.

Treatment - Since the disease is acute, treatment is not always beneficial. At the beginning of the disease, the use of hyperimmune blood serum according to the instructions (5-8 mg per 1 kg of weight + chlortetracycline once a day for 4-5 days) gives a good result. Administer ampicillin dissolved in 0.5% novocaine m/o until the general condition changes. Sending Bicillin 10 TB to cattle, 15 TB to calves, 15-20 thousand TB to sheep and goats.

2% hydrogen peroxide, 3-5% carbolic acid, 3-5% lysol or phenol, and 0.1% potassium permanganate solutions are injected into the area of the itchy swelling and around it. But these drugs are not always useful, so it is advisable to detect the disease in time and start treatment immediately.

Immunity - Immunity appears 14 days after vaccination and lasts up to 6 months. The formal vaccine discovered by SN Muromsev has been used for many years and has given good results. FI Kogan and AI Kolesov developed and put into practice the GOA formal vaccine . This vaccine is still used, and 2 ml is injected into the muscle of the calf.

Prevention. This event is carried out as follows: do not drink water from mud, do not graze in wet pastures, do not give ground food, it is necessary to take measures to protect them from mechanical injuries.

- susceptible animals are vaccinated according to schedule. If the disease is suspected:
- timely diagnosis;
- loss of epizootic focus;
- it is necessary to dispose of the bodies of dead animals, carry out remedial measures in the equipment, territory and barns. An unhealthy farm is recorded in the epizootic journal and a mark is placed on the card.

Veterinary measures: swamps in pastures are drained and the surroundings of water sources are regulated. Cattle houses and slaughterhouses are disinfected. The surroundings of the biothermal well and burial sites are cleaned. When carrying out

large-scale soil and reclamation works, strict control is necessary. Quarantine will be announced if the patient is about to leave.

Educational task.

Handouts based on the BBB method

No	Concept	I know	I want to know	I knew
1.				
2.				
3.				
4.				
5.				
6.				
7.				

Test question answers on the topic.

1. What animal diseases are black?
   A. Ruminants
   V. Odd ungulates
   S. Pair of ungulates
   D. Cattle

2. What are the clinical symptoms?
   A. Fever and swelling of the muscular and fibrous parts of the body.
   V. The temperature k is transferred.
   S. Animals lose their appetite.
   D. Clinical signs do not appear.

3. Who discovered the causative agent and in what year?
   A. Bollinger in 1875, Fezer in 1876.
   V. Bollinger in 1888.
   S. 1857 Cogan.
   D. 1876 R. Koch

4. How is the stimulus according to the degree of endurance?
   A. Unbearable.
   V. Durable.
   S. Extremely resistant
   D. Moderately resistant.

5. Cattle from 3 months to 4 years old are highly susceptible to the disease.
   A. Aujeski's disease.
   V. Rabies.
   S. Karason's disease.
   D. Leptospirosis.

6. What are the pathological anatomical changes in the disease?
   A. Mucous- red inflammation in the connective tissue between the subcutaneous muscles and a case of limited hemorrhagic necrotic myositis.

V. The injury is mainly in the hip, b game, shoulder, chest, stomach, in rare cases in the throat and diaphragm legs, tongue and myocardium.

S. The injury is mainly in the hip, b game, shoulder, chest, stomach, in rare cases in the throat and diaphragm, changes in the tongue and myocardium.

D. The above answers are wrong.

7. How long does immunity appear after vaccination?

A. 21 days after vaccination

V. 10 days after vaccination

S. 14 days after vaccination

D. 7 days after vaccination .

8. What measures are taken when the disease occurs?

A. Limit q is calculated.

V. Quarantine is established.

S. Susceptible animals are vaccinated according to schedule.

D. sanitation is carried out in livestock farms.

Name of the vaccine	**Epizootic condition**		**Amount (ml)**	**Shipping method**	**Number of submissions**	**Sending interval**	**The duration of the onset of**	**Duration of immunity**	**Storage period**
	unhealthy	**Dangerous econo**							
Concentrated GOA formal vaccine against scabies of cattle and sheep	+	-	2.0	m/o	2 times for 1 year old	-	14	6	12

Review questions:

1. Tell us about your understanding of the disease?
2. Describe the causative agent of measles?
3. Clinical signs of the disease, pathogenesis?
4. How is the disease treated?
5. Organization of veterinary sanitary measures ?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

EXERCISE : Diagnosis, prevention and control measures of cattle campylobacteriosis

Campylobacteriosis (lat. — Campylobacteriosis, Vibriosis genitalis enzoo-tica bovis/ovis; visual. — Vibriosis, Vibrio fetus infection of cattle/sheep; Russian-vibriosis) is an infectious disease that occurs mostly in cattle and sheep, sexually transmitted It is characterized by infection of the genitals, frequent estrus in female animals, infertility, mass abortion, retained placenta, and the birth of a non-viable fetus.

Trigger. The causative agent of the disease, Sampyllobacter fetus, is a polymorphic microorganism, a bacterium resembling a short curved comma or the shape of the Latin letter S. Less commonly, there are also spiral forms with 2-5 turns. Length 0.5-5, width 0.2-0.8 μm. Campylobacter is motile, has no spores and capsule. gram-negative (may be gram-positive in old cultures), good in all aniline stains will be painted. Under the microscope, these bacteria can be seen at one or both ends.

Campylobacter grows only in nutrient environments with reduced oxygen (10-20% of oxygen is replaced by carbon dioxide in a desiccator). From semi-liquid and solid media for growing them: 0.15-0.20% meat-peptone liver agar (semi-liquid), 2-3% meat-peptone liver agar (solid), vaseline fat-free Kitt-Tarotsi medium, Marten's agar, etc. nutrients are used. Cattle, sheep, rabbit blood or horse blood serum is added to make the feed medium nutritious. No turbidity or gas formation is ever observed in the feed. Bacteria grow on solid media for 72-96 hours. It grows in 4 different forms: S (smooth), M (mucoid), ragged and smooth glass colonies.

There are 5 species of Campylobacter in Spirillaceae family, mainly one species is S. fetus subsp. fetus (V. fetus veneralis) is a causative agent of disease in cattle. This type of bacteria is an obligate parasite, passes through the genitals and induces abortion in cows, leaving them sterile. Campylobacter can be seen and isolated in cow vaginal mucus, semen, bull genital sac, placenta and fetal tissues. The causative agent also passes through the alimentary tract.

Campylobacter is also pathogenic for pigs, goats, chickens and humans, 7-15-day-old chicken embryos, guinea pigs, rabbits, guinea pigs and white mice. This property depends on the endotoxin released from it and the activity of mucinase enzyme in it.

Exciter endurance. Campylobacter is not a very resistant bacterium to external environmental influences. 10-20 days in manure, soil, water and hay at 6-20°C; it lives actively in the stomach, liver, fetus, cotyledon for 20-50 days. It dies in 3 hours in the dried mass. It is stored in an unopened fetus at 20-25°C for 10-20 days. It can live only 3-4 days at a temperature above 25°C. Very sensitive to hot temperatures and antibiotics. Infection of animals with other microorganisms (esherichia, proteus, pyogenes, etc.) increases the viability of these bacteria. Campylobacter-ar survives in frozen tissue for up to 5-6 months. In liquid nitrogen (-196°C), the seed is kept active for a long time.

Epizootological data. In natural conditions, the disease occurs in cattle and sheep regardless of breed. Campylobacteriosis occurs at any time of the year, but is

more common in the autumn and winter months during mating. The age of the animal it doesn't really matter. Cattle of both sexes are infected, and the causative agent is released in large quantities into the environment when the cow gives birth.

a lifetime , in the sac of the genital organ (preputium), in the testicles and in the seminal tract, and the semen ', it is released with the mucous fluid of the sac and the juice of the prostate gland and is transmitted to cows during sexual intercourse.

The bodies of cows and females infected with campylobacter are also very dangerous in spreading the disease, because for 3-10 months, they are in contact with the mucus, urine and milk, especially during childbirth, with the fetus, its water, placenta. releases the stimulant in large quantities. The pathogen is mainly transmitted from an infected bull or bull to a bull during the natural shedding of cows and females in heat. If the semen is obtained from a bull infected with campylobacteriosis, the cow and female carcasses can be transferred even if artificially inseminated. Even if seed contaminated with Campylobacter has been treated with antibiotics, it has been shown to transmit the pathogen to artificially bred cows. 40-90% of natural escapes and 30-70% of artificial escapes are infected with pathogens. Campylobacter has been found to be transmitted to the cow's vagina if it is on the bed. The importance of non-sterilized obstetric gloves in the transmission of the pathogen needs to be emphasized. Juvenile females, even calves, can be infected with campylobacteriosis. This confirms that the pathogen is transferred to another organism by alimentary route, in addition to contact.

The source of the pathogen in sheep is a mother sheep that has given birth to a diseased fetus. They carry bacteria for 1-1.5 years. Sheep become infected when they drink feed and water contaminated with the pathogen. Rams from unhealthy flocks have campylobacter in their galls and livers, but transmission to ewes during lambing has not been proven. Also, the transmission of this pathogen from cattle to sheep and from sheep to cattle has not been proven.

Usually, the reason for the detection of the disease in a healthy flock is the introduction of a sheep carrying the bacteria into the flock without examination. Pigs, birds, dogs and foxes that eat the discarded fetus can serve as carriers and agents of transmission to other animals.

In a new, unhealthy furnace, cows are repeatedly calved, and the service period is prolonged. If the disease is acute, 20-55% of cows, 60-64% of female bodies remain barren. Abortion is observed in strait cows. This condition is usually observed for one season, then the process calms down. Animals develop immunity against this disease. In the herd, infection with this pathogen can be up to 0.5-2%, but the disease occurs sporadically.

Pathogenesis. If a female animal is artificially inseminated with a sick bull or infected seed, campylobacter appears in the genitals and uterus after 2-3 days. From this moment, the pathogen can be isolated from the uterus. After 10-15 days, the pathogen reaches the ovary. It is separated from the uterus by 3-6 months. As a result of the increase of the stimulus in this organ, inflammation begins, becomes evident on the 10-12th day and lasts for 4-5 months. In some cases, the causative agent can be isolated from the fallopian tube and bladder. The inflammation increases, and the

inner part of the uterus reaches the tissue layers. As a result, the recovery of the uterus becomes very difficult, and metritis can last up to 8-9 months. Due to this, it becomes difficult for the egg and sperm to join in the inflamed uterus of the escaped female animal.

In some cases, the fertilized fetus does not develop well and dies in up to 50% of cases at the initial stage of miscarriage, as a result of which an abortion is observed. Because bacteria multiply in the stomach, liver, back and brain of the fetus, inflame them and cause toxicosis. After an abortion, a cow usually remains barren for 3-6 months. The runaway mole will be placed again and again. This is caused by the release of toxins as a result of the increase of the stimulus in the uterus and the violation of the pH-environment there. In bulls, the causative agent is located in the preputial cavity, urethra and prostate gland. In such cases, quality deterioration of the seed is manifested.

The causative agent in sheep is C. fetus subsp. intestinalis enters the bloodstream from the intestine after alimentary entry and settles in the causative uterus after a short period of bacteremia. In the case of strangulation, the pathogen passes to the fetus and kills it, resulting in an abortion.

Course and clinical signs. In sick cows, the main clinical sign is inflammation of the vagina, abortion (often in 4-7 months of gestation), retained placenta, inflammation of the uterus. This situation leads to increased barrenness of cows and females on the farm. In some cases, due to campylobacteriosis in cows, in the 2nd month of goiter, the fetus may die without development and reabsorption. This condition is usually not noticed by farmers and experts. This can be known by the cow coming to the tune again after a long time. Due to this disease, the placenta is retained almost all the time in a cow that has given birth, inflammation is observed in the vagina and uterus. Some cows may give birth to a non-viable calf during the disease, but it will get sick in 2-4 days and die in 5-7 days. Usually, 6-15 days after cows are infected with campylobacter, the mucous membranes of the vagina redden and swell, rashes appear, thickened mucus flows from the vagina, fever is observed. They raise their tails and bend their backs, then pus is visible in the mucus. After 15-20 days, pea-sized hemorrhages are observed on the mucous membranes of the vagina.

In female animals, the disease can occur in two ways. In some cases, cattle are not fertilized. The cow or the body comes to the tune again and again, this situation continues for 5-6 months. Sometimes, estrus cows give birth in 4-5 months of estrus. It is necessary to run sick animals 4-7 times. The rhythm of the sexual cycle is disturbed, and the period of sexual peace (diestrus) lasts for 90 days. Embryo death is observed in 50% of cattle that have been burned several times.

Inflammation of the genital organ begins a week after infection. Later, the disease becomes chronic. Vomiting is one of the main clinical signs of the disease. According to most scientists, this condition is observed during the first four months of menopause. Animals that have given birth can then give birth normally, sometimes there is a recurrence.

Disease-specific changes are not clearly visible in male animals. Only the foreskin is slightly reddened, and mucus is released from it for 2-3 days. These symptoms disappear quickly, but remain a campylobacter carrier for life.

The main clinical sign of the disease in sheep is the observation of mass littering in the 2nd half of the strait. In some sheep, 3-5 days before abortion, there is a lack of appetite, lethargy, redness, swelling of the vagina and discharge of mucus from it. If the disease manifests itself during lambing, 10-70% of ewes may abort.

Pathologoanatomical changes. Adults do not die. Basically, the fetus thrown by the mole is dissected. The skin, subcutaneous tissue and muscles will be swollen. Hemorrhages are observed in the chest and abdominal cavity and parenchymal organs. It is noticeable that the blood vessels are filled with blood. In some cases, a reddish liquid accumulates in the chest and abdomen and contains fibrin. The fetus is sometimes embalmed, and necrosis is observed in the liver. Around the placenta, a yellow liquid mucous coating is visible.

Diagnosis. The diagnosis of campylobacteriosis is based on clinical symptoms, epizootological data and, of course, isolation of campylobacter as a result of laboratory examination. Observation of abortion in sheep and cows, repeated coming to the rut, infertility is the only reason to suspect this disease.

The fetus or its head, stomach, liver and lungs, placenta or its part are sent for laboratory bacteriological diagnosis. If this is not possible, mucus fluid from the cervix after abortion, mucus from the bull's preputial sac, prostate gland juice or semen are sent for examination. Pathological material should be delivered to the laboratory very quickly, cold and on ice. Especially in the summer, it can break down quickly. Material is also obtained by the swab method, and this method gives good results in serological testing (AR, KBR, KUBR).

Differential diagnosis. In cattle, campylobacteriosis should be differentiated from brucellosis, trichomoniasis, and in sheep, salmonellosis and chlamydiosis. When the blood serum is tested with a special antigen (AR, KBR, KUBR), it is determined that there are antibodies against brucellosis. In brucellosis, in the first months of gestation, abortion does not occur, heifers abort. Brucella is isolated from the pathological material. In trichomonosis, trichomonad is secreted, and the child's discharge corresponds to 2-3 months of thrush. In leptospirosis, leptospira is isolated, yellowness, high temperature (42°C). Hemoglobulinuria and skin necrosis are prominent. In addition to vomiting, cases of nervous disorders are observed in listeriosis.

In sheep, it should be distinguished from salmonellosis and chlamydial abortion. Salmonellosis not only causes abortion, but also affects the gastrointestinal system, lungs and joints in lambs and rams. Abortion in chlamydia occurs 2-3 weeks before the onset of lambing, and the results of bacteriological and serological examination clarify all suspicions.

Treatment. Many drugs have been recommended for the treatment of campylobacteriosis. Since the trigger is located inside the foreskin, drugs are not always effective. Sick bulls are treated 2 times in 4 days with an interval of 5-6 days. In the first course, after treating the foreskin with antiseptic drugs, streptomycin and

penicillin of 1 million units dissolved in 50-60 ml of vegetable or fish oil, and streptomycin and penicillin at the rate of 4 thousand units/kg twice a day in 0.5% novocaine are injected into the muscle . The 2nd course of treatment is carried out as follows. In the second course, oxytetracycline at the rate of 5,000 units/kg twice a day, and 5% furazolidone emulsion is injected into the foreskin. 1 month after the treatment, the testicles of the bull and the mucous membrane of the foreskin are bacteriologically examined 3 times with an interval of 10 days. If the test results are negative, the bull is considered cured.

Streptomycin in 0.5% novocaine is injected into the uterus of sick cows and heifers every day for 4 days at the rate of 4,000 units/kg. 1:5000 furatsilin or 1:1000 rivanol solutions are used to wash the skin. In the treatment of calves, 0.15 g of bigumal is given with milk 2 times on the first day, and 1 time during the next 4 days, and at the same time, 20% norsulfasol solution is injected intramuscularly for 3 days in a dose of 2 ml.

Streptomycin, penicillin, bicillin-3 and tetracycline are used to treat sick sheep. One of the above-mentioned antibiotics at the rate of 2 million units dissolved in sterile 20 ml of vegetable oil and streptomycin or oxytetracycline at the rate of 4 thousand b/kg twice a day for 4 days are injected into the uterus.

Immunity. Cattle have weak immunity. After the sheep recover from the disease, they almost never get sick. Therefore, a vaccine against campylobacteriosis of sheep was created (MALuchko, 1974). It is used in unhealthy farms. Basically, in September, 1 ml is injected under the tail, immunity appears after 15 days and lasts up to 12 months.

Prevention and countermeasures. A new animal brought to a healthy farm or farm must be taken from a healthy farm that is being checked for campylobacteriosis, during a 30-day preventive quarantine period before entering the farm, bulls must be bacteriologically tested for this disease 3 times with an interval of 10 days, only healthy it is required to add cattle to the herd. This disease can be prevented by feeding all existing cattle on the farm with nutritious feed, monitoring the clinical condition, keeping the farm tidy, timely collection of every dropped fetus, regular disinfection, artificial escape of cow and female bodies. helps to get

If campylobacteriosis among cattle is detected in the farm, the farm is declared *unhealthy and a plan of health measures is developed.* The number and groups of animals, the number of sick people, and the number of those who gave birth are calculated. All calves in unhealthy farms are kept in isolation. Clinically ill cattle and sheep are treated as indicated above. Miscarriages are separated, fetuses, placentas, beds are burned, buildings and playgrounds are disinfected. 2% alkali, 2% chlorine lime solution, 5% creolin solution, etc. are recommended for disinfection in unhealthy farms.

Cow and female bodies are artificially removed and after 10-12 hours streptomycin and penicillin of 100,000 units dissolved in 10-20 ml of sterile physiological solution at body temperature are injected into the uterus to prevent disease. 250-300 ml of bigumal dissolved in water at a ratio of 1:1000 is given to cows and heifers every day for 5 days in order to prevent castration of cows. A.

Golikov recommends administering bicillin-5 or dibiomycin in a dose of 25-30 thousand units/kg per animal. If no campylobacter has been isolated as a result of bacteriological examination within 12 months after the detection of the last diseased animal, and after all sanitation measures and final disinfection have been carried out, the farm is declared ***healthy*** **.** After that, the bulls are bacteriologically examined every quarter for 1 year.

If a disease is detected among sheep, all aborted animals are isolated and treated, and the flock is moved to another pasture. Pasture infected with the pathogen can be used after 1.5-2 months in the summer. In order to prevent abortion, chlortetracycline or 0.5 g of bigumal is administered to sheep at the rate of 5-8 mg/kg per head with soft feed every day for 10-12 days 1.5-2 months before giving birth. Drink every day for 3 days. Rams on a 4-day course: streptomycin and penicillin in 0.5% novocaine 4 thousand/kg are injected intramuscularly 2 times a day. If there is no abortion from this disease for 2 years, the herd is considered healthy.

Review questions:

1. Tell us about your understanding of the disease?
2. Describe the causative agent of campylobacteriosis?
3. Clinical signs of the disease, pathogenesis?
4. How is the disease treated?
5. Organization of veterinary sanitary measures ?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

EXERCISE : Diagnosis, prevention and countermeasures of brazot disease of sheep .

B radzot disease y is an infectious disease, with hemorrhagic inflammation of the jejunum and duodenum, tissue changes in the parenchymatous organs, and gas accumulation in the gastrointestinal tract.

Trigger. Most Sl. sepficum and Sl. Oedemmafiens is isolated. In many cases Sl. Also found in Didas. But this microorganism often increases the effectiveness of both previous microbes. Sl. septicum is strictly anaerobic, growing at 37°C. Grows in Kitt-Tarossi medium and becomes turbid and gasses in 16-24 hours, after 48 hours the broth becomes stagnant and it settles. Spores are rarely found in the body, they are often found in carcasses. Grows in Seissler's medium, showing the zone of hemolysis. The microbe releases gas and ferments in the following environments (glucose, maltose, fructose, etc.). Does not decompose mannitol with glycerin. It

rarely breaks down sucrose. It is from this feature that Sl. septicum and Sl. used in distinction from chavoei. Because the latter always ferments sucrose.

Sl. septicum secretes a very strong toxin, which is especially evident in Marten's broth. The toxin has four different components:

- Alpha toxin causes necrotic and hemolytic factor (hemolysis of sheep erythrocytes).
- Beta toxin-oxidative enzyme-deoxyribonuclease, causes rapid hemolysis.
- Gamma toxin-enzyme hyaluronidase.
- Delta toxin is oxygen-soluble and lyses erythrocytes.

Sl. septicum contains O and N antigens, which in turn produce agglutinin, precipitin, and hemagglutinins.

Sl. oedemmatiens is a bacilli polymorphic anaerobe. When growing in harsh environments, it forms a grainy surface and has uneven edges. Grows in a Kitt-Tarossi medium, forming a gas. Highly pathogenic for laboratory animals.

Cl. In addition to didas bradzot, it causes infectious necrotizing hepatitis. Gangrene can occur in humans.

Diagnosis. When making a diagnosis of Bradzot, its epizootology (sheep are infected, gender and age are not important, epizootic can occur in any season), clinical symptoms (severe, acute, in some cases restlessness, bloody diarrhea, tremors are observed), patho-anatomical changes (the spleen swells quickly, the skin may crack, putrid smell), the results of laboratory examination methods are taken into account. Parenchymatous organs (necrotic area of the liver), injured area of the liver, swollen tissues, duodenum, etc. are sent to the laboratory.

Bacteriological examination . Kitt-Tarossi medium from the sent samples, planted in MPBs and MPAs. Planted test tubes are placed in a thermostat with a temperature of 37-38°. For an anaerobic environment, a microaerostat or a desiccator is used. Sl. septicum grows rapidly and releases gas. Sl. Oedemmatiens grows slightly turbid and rarely produces gas. In a solid environment, the first forms a zone of hemolysis, later the center grows darker, the surface is rough, and the edges appear cut.

Biological method. An infusion is made from the injured organ in MPB, the mixture is injected into 2 guinea pigs at 0.5-1.0. If there is bradzot, the guinea pigs will die in 16-48 hours. They have septicemia. There is air in the intestines. Red fluid accumulates in the chest and abdomen. Observation is carried out for 8 days and all members are examined. If 1 guinea pig dies, histopathology is confirmed, and a culture is obtained, the diagnosis is confirmed.

Treatment. Due to its severe and rapid course, bradzot treatment is not beneficial. If the disease is protracted, antibiotics biomycin, synthomycin, terramycin 0.5-1.0 per 1 kg of weight for large sheep, 0.2 for lambs, 0.5-0.75 per head per day with biovetin feed is given. In this case, the main means of treatment are polyvalent hyperimmune blood serum and anatoxins, which are used according to the instructions.

Prevention. It is necessary to raise the sanitary conditions of pastures and water sources to the level of veterinary-sanitary requirements. Any place where

bradzot is released is under strict control. Sheep are vaccinated according to the plan, taking into account the epizootic situation.

Restrictions will be announced in outbreak areas. It is strictly forbidden to bring and sell sheep to the farm. Shearing is stopped and sheep driving is not allowed. Mowing from unhealthy pastures is stopped. From which flock a bradzot emerges, its place or pasture is immediately changed. Sick sheep are separated and healthy ones are vaccinated. Healthy sheep are returned from the pasture and transferred to confinement. Coarse hay and minerals are added to the diet immediately . Sheep sheds where sick sheep are kept are disinfected with 3% active chlorine solution of chlorinated lime , 5% formaldehyde or alkalis, 5% formaldehyde , 10% chlorine (1)-iodide . Slaughtering sick sheep for meat, removing skin from carcasses, shearing wool, sucking milk and using it for consumption is strictly prohibited . Carcasses are collected only in specially adapted transport vehicles , the place is disinfected . The carcasses of the affected sheep, wastes , carcasses are burned without removing the skin . Autopsy is possible only for diagnosis and is carried out in specially designated areas. The ban will be lifted two weeks after the last sick leave .

Review questions:

1. Tell us about your understanding of the disease?
2. Describe the causative agent of Bradzot's disease?
3. Clinical signs of the disease, pathogenesis?
4. Disease prevention measures ?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

EXERCISE : Diagnosis, prevention and countermeasures of enterotoxemia of sheep .

Enterotoxemia (Lot. - Enterotoxemia infectiosa anaerobica; visual - Struck, Pulpy kidney disease; Russian - infectious enterotoxemia) - "renal failure" is an acute severe infectious disease of sheep, characterized by hemorrhagic enteritis, nervousness, severe kidney damage and poisoning by toxins released by the pathogen. characterized by

Trigger. The causative agent is anaerobic, belongs to the genus Clostridia, and in sheep Cl. Perfringens D, S, and less often type A, and in other animals types A, V, E, and G' cause the disease. Each of them causes a specific disease. Cl. These 6 types of Perfringens have similar morphological, cultural, and biochemical characteristics,

but differ in pathogenicity, pathogenicity, and toxin secretion. Toxins in 15 different combinations were isolated from the causative filtrate.

Cl. Perfringens is gram-positive in young culture, gram-negative in old, non-motile (4-8x1-1.5μm, size) slightly bent rods with coiled ends, coccoidal and filamentous forms. In the external environment, the bacterium forms a spore in the center or near the tip. It forms a capsule in the body and in the nutrient medium. Kitt-Tarotsi produces gas and turbid broth when grown in glucose medium. 3 types of colonies grow on agar medium: S-smooth, Rgadir and M-smooth forms. Cl. Perfringens type C alpha and betatoxins, Cl. Perfringens type D secretes alpha and epsilon toxins. These toxins are separated from the pathogen in the form of inactive prototoxins, prototoxins are activated under the influence of trypsin, pancreatin and other enzymes in the body.

In the territories of Australia, New Zealand, USA, Canada, France, Peru, Angola, the disease is Sl Perfrigens type D, and in some cases type C, have been found to trigger. Some literature suggests that type A is the cause in the United States and France. In Greece, Cyprus, Bulgaria Sl. Types C, D of Perfrigens, type D in Iran, type C in Turkmenistan, type D in Kazakhstan, type C in the northern regions of Russia, and type D in Dagestan, Kyrgyzstan, and in some farms, both types cause disease. it provokes.

Exciter endurance. Spore forms of clostridia are highly resistant to physical and chemical effects. It remains active in soil and water for 16-20 months, and on wool and skin for more than 2 years. It is kept active in the soil for 16-20 months at 35-40°C, and up to 40 months at 15-20°C. It can live in water for 20 months. It is stored for up to 3 days in dried form, up to 2 years in leather and wool at 10-20 °C. When dried, it dies in 1-2 days. Boiling kills the pathogen in 15-20 minutes. 3-5% active chlorine lime, 5% hot caustic soda, 15% hot sulfuric-corbolic acid mixture, 5-10% formaldehyde solutions inactivate the pathogen in 10-15 minutes, and therefore in disinfection is used. And the pathogen in the vegetative state is resistant to the effects of the external environment.

Epizootological data. In a natural state, sheep are susceptible regardless of breed and age. Cattle, goats, horses, poultry, pigs, camels and wild animals are less susceptible. Guinea pigs, rabbits, pigeons and white mice are prone to laboratory animals. People get sick too. Sheep of all ages are affected, more sows, lambs and young 8-10 month old sheep. Sheep with little movement in the flock, fat breeds and fast growing ones get sick faster. Cl. The disease caused by Perfringens type D is observed in all ages of sheep: in spring - lambs, in autumn - in older sheep.

Cl. The disease caused by Perfringens S species is mainly observed in large sheep. The disease occurs more in spring, less in autumn and winter.

The causative source of the disease, sick and recovered clostridia are carriers. A sick animal pollutes the environment with its excrement, especially soil, pastures and water. The causative alimentary tract enters through the mucous membranes of the digestive system through food and water. Mass disease among lambs is observed in spring and late spring, early summer in rainy years. In natural conditions, animals are infected when they feed on pastures, mainly when they eat soil feed (grass, hay)

contaminated with the pathogen, or drink water. Disruption of the secretory and motor functions of digestive organs enables the disease to occur. This includes, in particular, a rapid change in the quality of feed, the sudden exit of sheep standing in the barn to the pasture or frost and dew, snow on the grass or eating frozen grass, grazing after rain, mineral and caused by a lack of protein.

The disease occurs in many countries of the world, including Australia, New Zealand, the USA, Canada, Argentina, Peru, Angola, Italy, France, Cyprus, Bulgaria, Hungary, Turkey, Iran, the Siberian regions of the Russian Federation, Transcaucasia, Kazakhstan and registered in the Republics of Turkmenistan, Kyrgyzstan, Uzbekistan.

The disease occurs among sheep of different ages. In some cases, it has been reported among sheep fed on fat, especially when the diet contains a lot of concentrates or when the pasture is very rich in grass. In Dagestan, all older sheep from 8-10 months of age are infected. According to K. Urgeev (1985), the disease is recorded in 52 percent of mother sheep, 21 percent of lambs and 27 percent of mixed age sheep. Ewes usually get sick in the last months of the estrus. When 640 sheep died of enterotoxemia were dissected, 489 of them were pregnant, and 276 of them had 2 or more fetuses. In some regions, the incidence of enterotoxemia in lambs was higher, and in the regions around Western Siberia and Baikal, the incidence of lambs was also higher. Their age is 1.5-2 months, and they are often found when they are fed in the same place with soft feed. This situation was not observed in sheep raised on pasture. In unhealthy farms and flocks, the disease is acute, sheep of all ages are affected. In most cases, the causative agent of enterotoxemia can be isolated from the organism of healthy sheep. This disease mainly affects sheep, but it can also be found in cattle, goats, horses, pigs, and camels. Guinea pigs, cats, and white mice are prone to laboratory animals. Rabbits and rats are not infected. Factors that disrupt the secretory and motor function of the stomach are of great importance in the origin of the disease. This is especially the case when they are raised by hand or in one place and then suddenly put on pasture. In our conditions, the disease occurs mainly in early spring, when new foliage begins to grow. The sheep, which have suffered from winter, are very open, thrown to the new green, greedily and eat a lot. In this case, the stomach of the sheep, which is not used to green, swells while resting, and gas accumulates. As a result, an anaerobic environment is created in the gastrointestinal tract, and clostridia develop and multiply. This is especially evident in early spring, when young greens are covered with dew or frost and have not yet evaporated. Enterotoxemia is a *non-infectious* disease.

Pathogenesis. In nature wide after spreading clostridia enter the animal body, in exchange for the violation of the secretory and movement activities of the organs of the gastrointestinal system, in anaerobic conditions, because the stomach is full of undigested food, it is considered a favorable condition for the existence of the causative agent of enterotoxemia. multiply in the organ and release prototoxin from themselves. Then, under the influence of proteolytic enzymes in the intestine, the prototoxin becomes epsilon-toxin, which is absorbed into the blood, lyses erythrocytes, injures the mucous membranes of the intestines, epithelial cells, kidney

parenchyma, liver and central nervous system, and poisons the entire animal organism. Increases the permeability of the endothelium of blood vessels and capillaries, as a result of which hemodynamics is disturbed. These pathological processes derail metabolism, especially carbohydrate metabolism in the liver, kidneys, and brain. The heart, kidneys, liver and central nervous system fail to perform their normal functions and death occurs. Conditionally pathogenic bacteria in the organs of the digestive system can contribute to such a severe course of the pathological process.

Course and clinical signs . The latent period of the disease depends on the virulence of the causative agent of enterotoxemia, the resistance of the animal and the state of the organs of the digestive system. It is 2 - 6 hours when artificially infected. The disease can be *very acute* , *acute, semi-acute* and *chronic* . Many scientists distinguish between comatose and hemorrhagic forms, which depends on the nature of the toxin released from the trigger.

the disease is *very acute* , they suddenly die in 2-3 hours without clinical signs. Such a transition is observed in young and fat sheep. Dead sheep are seen in the morning in the barns and pastures. Affected sheep lag behind without grazing and become a little weak. They have depression, a normal or slight increase in body temperature, the pulse is weak and accelerated. From their nose and mouth, foamy serous, blood-mixed mucus flows, frequent urination is observed. In some cases, bloody diarrhea and cramping are observed. The gait of sick sheep is reminiscent of the swaying posture of a cradle rocker, they crawl and fall. While lying down, he swims with his legs, trembles, grinds his teeth, his eyes are swollen, his mucous membranes are red. Eventually they die.

the disease is *acute* , they have general depression, lethargy, anorexia, fever up to 41 ^{0} C, bloody mucous diarrhea, ataxia, paralysis of the legs. Symptoms of central nervous system disease are observed in sheep, they walk forward, fall and fall again, swim with their legs, lie unconscious. His head falls back as a result of the contraction of his muscles. A foamy mucous substance flows from the mouth. Anemia in the mucous membranes, blood in the urine. They urinate involuntarily. Gastrointestinal activity slows down, the patient quickly weakens and dies after 2-3 days.

semi-acute course of the disease occurs in young and older sheep, in which anorexia, diarrhea, and thirst are observed. This form may be a continuation of the acute course. In this form, food digestion is disturbed, appetite decreases, diarrhea and weight loss quickly. During diarrhea, stools are liquid mucus, foul-smelling and bloody, mixed with mucus or blood. The mucous membranes of the eyelid are yellowish without blood. The animal loses weight, hair falls in some places. The disease lasts 10-12 days. Abortion is observed in the straits.

Chronic shedding is more common in lean sheep. Sick sheep become weak, do not eat anything, the mucous membranes bleed, the sheep becomes silent and sleepy. The lambs lose appetite, become lethargic, shiver, have colic, have diarrhea, and are nervous. Sick sheep usually lose a lot of weight. Some of the sick sheep may sometimes recover.

Cl. In sheep infected with Perfringens type C, *a hemorrhagic condition* in the intestine and parenchymal organs is observed, which is called *hemorrhagic enterotoxemia* . Cl. In sheep infected with perfringens D (epsilon - toxin) type, toxic condition is often observed and it is called infectious enterotoxemia. In this case, the disease can be very acute, acute and chronic, and the state *of glucosuria* is manifested.

Pathologoanatomical changes. A dead body is opened only for the purpose of making a diagnosis. A dead body rots quickly, and the smell is noticeable if it is handled acutely. In woolless places a dark brown spot is visible. The dead body immediately fills with air within 2-5 hours and begins to grow. The fur is easily pulled off and the skin has large blue spots. A thick foam mixed with blood flows from the mouth and nose, hemorrhagic swelling and bleeding are observed when the skin is rubbed. Mucous reddish fluid accumulates in the chest and abdomen. Hemorrhage is observed in the epicardium. Hemorrhage and inflammation are visible in the mucous membrane of the large abdomen. The lungs are swollen and cystic, the bladder is full of blood-mixed urine, the kidney is cystic, and there is blood under the capsule. The kidney becomes a shapeless mass, a pulpy mass filled with a very loose sac. In older sheep, this condition may not be evident. When lymph nodes are cut, mucous fluid flows and small foci of necrosis are found.

Cl. In sheep infected with Perfringens type C - hemorrhagic inflammation in the small intestines and softening of the kidney, Cl. In sheep infected with type D perfringens, only the kidney parenchyma changes into a shapeless mushy mass. This condition is observed especially in lambs.

Diagnosis. The diagnosis of enterotoxemia is based on epizootological data, clinical signs, pathologoanatomical changes, and, of course, the results of laboratory tests. A dead body can be opened in a special place only for diagnosis. In laboratory examination, pathogens and their toxins are isolated from parenchymatous organs and small intestines and identified using special blood serum.

At the same time, the presence of a toxin (stimulating product) is detected in the small intestine. The body of a dead sheep, parenchymatous organs, or a piece of the small intestine taken in a position where both sides are tied are sent to the laboratory. The examination is carried out by finding the presence of toxins in the intestine and the trigger. For the first method, a 1:1 or 1:2 ratio physiological suspension is prepared from a piece of intestine, which is strained and filtered. To determine the type of toxin, 1 ml of filtrate is taken in 5 test tubes and 1 ml of antitoxin serum is added. In the first test tube there is serum "A", in the second "S", in the third "D", in the fourth "E", in the fifth there is 1 ml of physiological solution. This mixture is injected 0.5 ml into the stomach of 2 white mice or 0.2 ml into the skin of guinea pigs or rabbits. Rabbits get necrosis, guinea pigs die.

For bacteriological examination, a smear is prepared from the mass of the small intestine or parenchymatous organs, stained by the gram method, and from parechymatous organs, bone marrow is cultured in Kitt-Tarotsi, GPB, GPA, and Kitt-Tarotsi media from the intestine and grown at 37-38 °C. will be erased. Cl. One of the important diagnostic signs of perfringens type D is the presence of sugar in the

urine (*glucosuria)* .

Differential diagnosis. It is necessary to distinguish enterotoxemia from bradzot, anthrax, pasteurellosis, listeriosis and food poisoning, piroplasmosis. In Bradzot, hemorrhagic inflammation, ulcerative colitis, necrotic cells are observed in the duodenum, liver, and there is no toxin in the intestines, and the kidneys are not empty. In case of anthrax, the spleen enlarges, the pulp becomes soft, and when cut, the pulp becomes similar to degt. When pasteurellosis is semi-acute and chronic, a septic process and pneumonia are observed. In listeriosis - there is no toxin in the intestine. When food poisoning, the disease is observed in many animals. In piroplasmosis, the parasite is seen in erythrocytes. In all cases, laboratory methods - bacteriological, serological and toxicological tests are the basis for making a final diagnosis.

Treatment. Enterotoxemia is difficult to treat because it is acute and acute, and in chronic cases, bivalent hyperimmune blood serum is administered (antibiotics are also added). Some experts have obtained good results by injecting 2-2.5 mg/kg of biomycin intramuscularly 4-5 times. Sintomycin was given to large sheep at 0.5-1 mg/kg and to lambs at 0.2 mg/kg. K. Riskulov (1983) recommends prolonged antibiotics for the treatment and prevention of the disease. It states that it is highly desirable to add it with bivalent blood serum. When 0.25-1 kg of cormogrizin, 0.5-1 kg of biovit, 1-1.5 kg of baxicillin were mixed with 10 Czuka of antibiotics, lambs were protected from disease (the disease decreased by 4.2 times). Intramuscular injection of dibiomycin and tetracycline works well.

Immunity. For active immunity, the concentrated GOA *formalvaccine* (F. Kagan, AP Kolesova) is used to vaccinate sheep and goats against polyvalent bradzot, enterotoxemia, malignant tumor and dysentery. The vaccine is administered 2 times with an interval of 12-14 days and preventive vaccination with an interval of 20-30 days. Immunity appears in 12-14 days and lasts for 6 months. Polyanatoxin is also used against sheep clostridiosis (LV Kirillov, FI Kagan). If this drug is vaccinated 2 times with an interval of 30-45 days, the immunity lasts for 10 months. If the anatoxin vaccine against Cl. Perfringens type D (A. Volkova) is vaccinated subcutaneously 2 times with an interval of 12-28 days, immunity is maintained for 4-5 months. It is also possible to use the method of complex vaccination of sheep against bradzot, enterotoxemia, anthrax and smallpox.

Prevention and countermeasures. For prevention, it is necessary to control the veterinary-sanitary condition of the cows, pastures and drinking places. Factors contributing to the development of the disease are eliminated. Sheep should not be driven to pasture in early spring when grasses are emerging. At this time, it is necessary to give dry hay. If this is not possible, the sheep can be put out to pasture after the frost or dew has lifted. All areas of enterotoxemia should be considered and sheep should be vaccinated in the spring, 30-45 days before being released to pasture.

If this disease is detected clinically, pathologoanatomically, bacteriologically among sheep, the herd or farm is declared unhealthy and a *restriction* is placed on it within the framework of the Veterinary Regulations. At the unhealthy point, all

containment measures are taken to prevent the spread of the disease. Entry and exit of new sheep to the farm, shearing, and mixing of sheep with other groups are prohibited.

After the disease is detected in the herd, it is transferred to another pasture, not driven to distant places. Sheep in the flock are clinically examined. Diseased and suspected animals are separated and kept in a pen and treated with special, hyperimmune blood serum, symptomatic and antibiotics. Clinically healthy sheep are left in the pen, vaccinated, and given coarse hay and mineral salts. The cage is disinfected weekly with 3% chlorinated lime, 3-5% caustic soda, 10% formaldehyde, monochlorinated iodine. Dead bodies are burned without removing the skin and fur. Sick sheep are not slaughtered for meat, wool and milk are not taken. *The restriction* from the farm is taken after 20 days after the end of the outbreak and recovery, after all measures and final disinfection have been carried out.

Review questions:

1. Tell us about your understanding of the disease?
2. Describe the causative agent of enterotoxemia?
3. Clinical signs of the disease, pathogenesis?
4. Disease prevention measures?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Topic: Diagnosis, prevention and countermeasures of rhinopneumonia disease of horses.

Rhinopneumonia (lat. - Rhinopneumoniae equorum; visual. - Equine virus abortion; Russian - viral abortion kobil) is an acute contagious infectious disease characterized by fever, conjunctivitis, catarrhal inflammation of the mucous membranes of the respiratory tract and is characterized by abortion in the last months of estrus.

Trigger. It is a DNA-storage virus belonging to the 1st generation of Herpesviruses and the Herpesoviridae family. The diameter of the virion is 100-150 nm. There are 2 subspecies of this virus. The subtype 1 strain was isolated mainly from North and South American horses, and the subtype 2 strain was isolated from horses of the European and Asian continents. Hemagglutination of erythrocytes of viruses, endothelio- and epitheliotropic. Hemagglutinating, precipitating, complement-binding and virus-neutralizing antibodies are detected in the blood serum of a horse that has recovered from the disease. The virus develops in a cellular environment, in chicken embryos and in laboratory animals.

Exciter endurance. The virus is resistant to external influences. Infected organs are kept active for 457 days at -18 ^{0} C. In a dark room at 20-27 ^{0} C, the virus does not lose its activity for 14 days, in horse hair for 42 days. In the tissues of the aborted fetus, the virus is inactivated in 6-7 days at 4 ^{0} C, but in 10-20 minutes at 55-56 ^{0} C. Ether, chloroform, 0.5% formaldehyde kills the virus in 10-15 minutes. It is resistant to acidic environment.

Epizootological data. In natural conditions, horses, mules and donkeys are prone to rhinopneumonia regardless of breed, age and sex. Purebred horses, mares under one year old are especially susceptible to this disease. Cattle, sheep, goats, pigs and humans are not infected at all. Laboratory animals are prone to guinea pigs and porcupines.

The origin and spread of the disease is made possible by uncontrolled transfer of horses from one farm and stable to another, not feeding them with nutritious feed, not keeping them in accordance with zoohygienic requirements, and breeding with close relatives.

The source of the pathogen is a sick animal, which secretes the virus in large quantities through its nasal fluids, genitals, and the discarded fetus and its fluids. When the disease is in the respiratory form, the virus contaminates objects of the external environment with coughing, when the horse coughs, sneezes, and infects healthy horses through airborne droplets and contact. In addition, the virus is transmitted through food. The virus isolated from a sick horse also contaminates water, bedding, and stable equipment and serves as a carrier of the pathogen through which the pathogen is transmitted. Throat beetles excrete the virus in their urine before abortion. The lice, in which the disease is *latent* , serve as *a dangerous virus carrier* for a long time . Stallions also act as carriers of the virus and transmit the pathogen to mares during natural mating. This condition can *last* for months or even years in stallions .

Diseases usually come to healthy farms and herds with virus-carrying or diseased horses imported from elsewhere. The transmission of the virus to the herd of healthy horses, especially thoroughbred mares, causes the disease to spread widely. Diseased bees serve as a source of transmission of the virus for a long time. The disease occurs in the form of enzootic disease, usually 40-60%, in some cases up to 90% of gypsy moths abort. Abortion increases when young ewes are kept with a flock of sick ewes. Respiratory rhinopneumonia is more common in winter and autumn.

Pathogenesis. The virus enters the body through the intestines or respiratory organs, settles in the mucous membranes of the respiratory tract and throat, where it multiplies and causes catarrhal inflammation. From these places, the virus enters the blood through the lymph and spreads to all organs, and *viremia* occurs. The virus, together with leukocytes, easily passes through the uterine and placental barriers and enters the fetus. When the virus enters the fetus, it multiplies in all its organs and tissues without any resistance and leads to the development of sepsis in the fetus. As a result, degenerative and necrotic processes occur in all organs of the fetus, so

hemorrhages are observed in all its organs (capillaries, brain), including parenchymal organs (liver, spleen), and as a result, the fetus dies. This ends in abortion or stillbirth.

During histological examination, *viral inclusions* appear in the nuclei of necrotic tissue cells in the endothelium of the liver, biliary tract, bronchi, and endothelium of blood vessels.

Course, clinical signs and forms . The latent period of the disease is 2-10 days. Depending on the manifestation in the organs of the body, 4 forms are distinguished: *respiratory, abortion, genital* and *nervous forms.* In horses that are infected for the first time, the disease is *acute* , and then it can turn into a *chronic* form. The onset of the disease is manifested in *the respiratory form - catarrhal inflammation of the upper respiratory tract and conjunctiva. A fever of 40* ^{0} C and above is observed for 2-3 days at a time . Usually, the disease passes in a relatively mild and *abortive form* . Abortion is observed 100-150 days after being infected with the virus. Abortion occurs more often in 8-11 months of gestation, less frequently in 5-7 months. Abortion occurs *suddenly , the fetus falls wrapped in a veil.* Usually, placental abruption and other postpartum complications are not observed. In some cases, a bee that aborts *in the form of a nerve can die as a result of paralysis.* In other forms of the disease, the uterus quickly returns to its normal state after an abortion, and after 1 month, the uterus may return to normal. Sometimes, if the ewes are infected with the virus in the last stage of estrus, the ewes will be born alive, but it will become incapacitated and die within 1-3 days.

Horses that are not fed with nutritious feed, lacking minerals and vitamins, get sick, and the disease is severe in them. The mucous membranes of their upper respiratory tract are reddened, the conjunctiva of the eyes turns yellow, the mucous membranes of the nose, throat, and larynx are inflamed and swollen. Head and neck regional lymph nodes are enlarged. Swelling of the throat and larynx makes it difficult to breathe, wheezing and wheezing. Serous-mucous fluid from the nose becomes purulent due to secondary infection. In some cases, pneumonia, inflammation of the mucous membranes of the gastrointestinal tract are observed.

If the disease is very severe, the central nervous system and the heart function will fail. Serous-fibrinous inflammation in joints, orchitis in stallions can be detected. Leukopenia is usually detected in rhinopneumonia, and leukocytosis when aggravated by secondary infection. Horses usually recover in 1-3 weeks unless complications occur. Biyas can also have *a genital form* . In this case, the mucous membrane of the vagina turns red, rashes appear, which turn into white spots.

Pathologoanatomical changes. Symptoms characteristic *of septicemia* in this disease : mucous membranes of the upper respiratory tract, redness, swelling, inflammation of the conjunctiva, degenerative changes in the parenchymatous organs - kidneys, liver, spleen, heart, lymph nodes and their edema, mucus and hemorrhages are noticeable in the serous membranes. Small rashes appear on the mucous membranes of the nose and throat, and in some cases, the larynx. Shiny amber-yellow or red-yellow liquid collects in serous spaces. 50 ml of fluid accumulates in the pericardium, 1-2 liters in the chest cavity. Under the epicardium, endocardium and

splenic capsule, spotty, striated hemorrhage is observed in the renal capsule. The lungs are swollen, there will be hemorrhagic spots.

In the mucous membranes of the stomach and small intestine, ilviral layers are visible. Peyer's rash and solitary follicle in the small intestine have erosions and deep ulcers. In addition to hyperemia, swelling in the lungs, catarrhal inflammation and peripneumonia are observed in rare cases. The liver is swollen and darkens. Dotted hemorrhages and necrosis are observed in the mucous membranes of the stomach and intestines. When 7-11-month-old gilts abort, *the fetus does not undergo autolysis* . When examining a histological preparation made from the liver, spleen and lungs of an aborted fetus, under a microscope, inside the nucleus of the cells, *Cowdry viral inclusions* are visible, its diagnostic value is great.

Diagnosis. The final diagnosis of the disease is made on the basis of clinical signs (abortion), epizootological data, pathologoanatomical changes and, of course, virological and serological tests. The observation of abortion among strait bees may be a reason to suspect rhinopneumonia. The final diagnosis is made only on the basis of *virological, serological* and *histological examination* of pathological paterials taken from sick horses and aborted fetuses . In the laboratory, the nasal lavage of the sick horse, the material scraped from the nasal mucosa, lung, liver, spleen tissues, the discarded fetus, the stomach, liver, lung, spleen, brain, heart and kidney were cut into the laboratory. the received fragments are sent. The virus is isolated from the pathological material sent in the laboratory and it is identified. NR, GATR and KBR reactions are performed with blood serum of sick horses, and antibodies formed against rhinopneumonia virus are determined. The immunofluorescent reaction is of great importance in the diagnosis of the disease and in the identification of the virus.

Differential diagnosis . This disease should be distinguished from influenza, abortion with salmonellosis, food poisoning. Flu symptoms are: fever, worsening of the patient's condition, conjunctivitis, catarrhal inflammation of the upper respiratory tract. Clinical symptoms of rhinopneumonia are almost invisible. Abortion with salmonellosis occurs at 4-8 months, and with rhinopneumonia, abortion occurs suddenly at 7-11 months, and it is born wrapped in membranes. Spots of necrosis, hyperemia are observed in the placenta. In abortion with salmonellosis, the placenta is retained and metritis develops as a complication. There are no complications after an abortion in rhinopneumonia. During bacteriological examination, salmonellosis grows in nutrient media in abortion with salmonellosis. Food poisoning is determined by examining feed samples in the laboratory.

Treatment . A special treatment method for the disease has not been developed. If the disease becomes complicated, it is treated symptomatically. Antibiotics and sulfanilamide drugs are used to prevent secondary infection.

Immunity. Cows that have recovered from the disease have short-term immunity, so within 1 year they may have a recurrence of the disease. Colostral immunity lasts only 2-3 months. Infected older sows also have a very short immune system and may experience abortions once they are engorged. However, in aborted bitches, the immune system is long-lasting, and when they are spayed, they give birth to healthy pups. Therefore, compared to the respiratory form, *immunity* lasts longer in

the abortive form of the disease. To date, it has not been determined which high titer of antibodies in sick horses is a sign of recovery from the disease.

Prevention and countermeasures. Horses purchased for breeding or sports should be taken from countries, farms and farms that are healthy for infectious diseases, checked for mange, rhinopneumonia, influenza and other infectious diseases before arrival, and 1 month preventive after arrival. During the quarantine period, it is necessary to carry out clinical and laboratory tests again. Horses are also not taken from a farm where an abortion has been observed in the last 2 months. In order to ensure the resistance of horses, it is necessary to feed them with nutritious food, rich in vitamins and minerals, and keep them according to zoohygienic requirements.

Rhinopneumonia if clinical, pathologo-anatomical, virological findings are found among horses, the herd, farm or settlement is declared unhealthy and a *restriction* is imposed within the scope of the Veterinary Regulation. In the unsanitary point, measures are taken to carry out all health measures and prevent the spread of the disease. Entry and exit of new horses to the farm, mixing of horses with other groups is prohibited.

After the disease is detected on the farm, the horses at the point are kept without being released to pasture and are clinically examined. Sick animals are separated and treated symptomatically, the remaining healthy horses are left in pens and vaccinated according to the Instructions for the use of the vaccine. 1-3-month-old fawns are vaccinated, and revaccination is required after 6-7 months. Young mares and stallions should be revaccinated 3-4 months after the first vaccination. Stables are disinfected weekly with 3% chlorinated lime, 3-5% caustic sodium, 10% formaldehyde, monochlorinated iodine, and biothermal disinfection of manure for 3 months.

The restriction from the unhealthy point is taken 2 months after the last patient was treated, after the final disinfection, if there are no pus passing through the 2nd half of the strait.

Review questions:

1. Tell us about your understanding of the disease?
2. Describe the causative agent of enterotoxemia?
3. Clinical signs of the disease, pathogenesis?
4. Disease prevention measures ?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Topic: Diagnosis, prevention, and countermeasures of horse dumbness .

Mute (Lat. - Adenitis equorum; English - Strangles Adenitis equorum; Russian - mit) is an acute contagious infectious disease of horses, characterized by fever, purulent-catarrhal inflammation of the mucous membranes of the nasal cavity and throat. , is characterized by inflammation of submandibular lymph nodes turning into an abscess.

The causative agent is Streptococcus equi (silent streptococcus) - a non-moving, non-spore-forming microorganism of round or oval shape. In a smear made of pus, the cocci appear in the form of long thin curved threads. In smears made from cultures grown in nutrient media, the threads and chains formed by them are shorter. In sick horses, mute streptococci is observed only in smears prepared from abscesses. Nasal fluids from sick horses also contain other microorganisms, especially mixed with purulent microorganisms. Mute causative agents belong to the C group according to the type of antigen, that is, the strong hemolytic group. Streptococci are gram-positive, stain with all aniline dyes, and at 37°C, growth increases if horse serum is added to the culture medium. In pure young cultures, the capsule and blood agar form v - hemolysis. Unlike saprophytic streptococci, the causative agents of muteness are weak in their biochemical activity: they do not curdle milk, cannot break down lactose, sorbitol, and mannose. Mute streptococci secrete hematotoxin, leukotoxin, aggresin when grown in broth. These organisms grow in facultatively aerobic, artificial nutrient media, but grow best when supplemented with 10-20% horse serum or 5-10% blood.

Exciter endurance. Mute streptococci are relatively resistant to environmental influences. In stables with poly soil, it is stored at a depth of 10-15 cm for up to 9 months, in dried pus - for a year, it is also resistant to disinfectants. It is kept active for 4 weeks in manure, 22 days in hay, hay, horse wool. It is inactivated in 6-8 days by sunlight, 1 hour at 70-75°C, boiling kills it immediately. 5% carbolic acid, 3% creolin, 2% formalin kill in 10-15 minutes.

Epizootological data. Mutism is an equine disease that is more common in horses. Horses get sick more often from 6 months to 5 years. Foals aged 1-2 months and horses older than 5 years are relatively rarely infected. In some epizootic cases, there are reports that even 15-20-year-old horses are infected with dumbness. Adult horses are less likely to be infected with the infection in the form of an immunizing subinfection. The immunity of young foals is related to colostral immunity. In the experiment, young mares brought from a healthy farm with this disease can be infected by rubbing the mucoid culture or abscess fluid on the nasal mucosa or by adding them to the feed. When administered subcutaneously, local suppuration occurs, when administered intravenously, *septicemia* occurs.

The source of the pathogen is *sick* horses, which secrete the pathogen with pus from the nose, sometimes mute streptococci is also found in the nasal mucus of a healthy horse. Animals that have recovered from the disease also serve as *carriers of*

the bacteria for more than a year . For this reason, the disease of muteness, which is not brought from outside, can be recorded in the farm itself.

Transmission of the disease occurs mainly through alimentary and airborne routes. Factors that transmit the pathogen are fodder contaminated with it - hay, water, mangers, pasture hay, stable equipment. In less cases, contact: when dodging, sucking, touching each other, and other ways of transmitting the pathogen. The rapid spread of the disease is also caused by cold temperature, close keeping of horses, and deficiencies in feeding. Mute often occurs sporadically, in some cases in the form of enzootic, epizootic. The disease can enter from other farms or appear on the farm itself. In the Arkhangelsk region of Russia, in the autumn season, among 3500 4-8-year-old horses, 300 of the 4-8-year-old horses were diagnosed with deafness. The rapid spread of the disease was caused by the fact that the horses were kept tightly in the stable in the rainy and cold weather. In this case, the disease is spread by air-droplet, because in this disease, horses sneeze. In the regions of Siberia, Kazakhstan, and the North Caucasus, deafness was also observed in the summer. In this case, the weather became cold and the horses drank ice-cold water, causing their resistance to drop. In case of muteness, the incidence is up to 5.67%, and the mortality is from 1.9% to 5.5%.

Pathogenesis. When dumb streptococci get on the nasal mucosa, they go from it to the submandibular lymph node through the lymphatic system, multiply there, and due to the irritant and the toxins released from them, the mucous membranes of the nasal cavity and throat are first serous, then purulent inflammations appear. As a result, it becomes very painful and difficult to drink water, especially to eat coarse hay. Inflamed submandibular lymph nodes become enlarged, hard, painless and warm. After 4-6 days, they soften, fester (abscess) and burst. Then the inflamed swelling is quickly absorbed, the pain disappears, the ruptured abscess is closed with healthy tissues, and the animal completely recovers from the muteness. Usually, if the mute is *typical* , the infectious process ends as mentioned above.

If the resistance of the horse is strong, if the virulence of the pathogen is low, the abscess will not form. Phagocytosis, antibodies, and all other defense systems are activated to destroy the mute streptococci. The disease will heal quickly. This condition is observed only if the disease occurs in an abortive form. However, in mares with low resistance, *a complicated metastatic form of dumbness* develops. In this case, the causative agent of the disease causes purulent inflammation of the deep lymph nodes of the back of the throat, ears, and neck through the lymphatic system. The causative agent enters the blood and causes a metastatic abscess process in the lungs, liver, kidneys and other organs. The transfer of bacterial toxins and decayed tissue from abscesses into the blood causes metabolic disorders in the body, as a result of which the body temperature rises, leukocytosis and heart failure are observed. Mute streptococci spreads throughout the body, if it spreads through the lymphatic system, all lymph nodes become inflamed, if it spreads through the blood, it causes *bacteremia* in the body due to the appearance of metastatic abscesses in the internal organs . *septicemia* occurs and the animal dies. Complicated form of muteness will definitely end in death.

Course, clinical signs and forms. The latent period of the disease is from 1-2 to 15 days, on average 4-8 days. Dumb is sharp. Depending on the development and clinical manifestations of the pathological process, *typical, abortive, complicated (metastatic)* and *genital forms of the disease* are distinguished.

The typical form usually begins with an increase in body temperature of 40-41°C. The animal is sad, the appetite decreases. At the beginning of the disease, the mucous membranes of the nasal cavity and throat become inflamed, red, and swollen, which makes swallowing difficult. When drinking water, it comes back out of the nostrils. On palpation of the throat, submandibular lymph nodes are enlarged, local temperature is high, hard and painful. Rhinitis is observed in the nose, from which liquid, mucus, pus flows. The breath cooks and it accelerates. If the inflammation has spread to the larynx, a cough appears and it becomes difficult to breathe, wheezing is observed. Swelling covers the ear and lower jaw. The horse stretches its neck and stands motionless. After 4-6 days, the skin of the place where the submandibular lymph node is located softens, a special sound (fluctuation) is heard when palpated, and the abscess bursts and releases creamy pus. After the abscess bursts, the animal's body temperature normalizes. After the discharge of pus has finished, the place where the abscess has ruptured is gradually filled with healthy tissue over time. The disease lasts 15-25 days and recovers. Urinary excretion decreases in sick animals, protein appears in the urine. During the period of abscess formation, the amount of indican in the urine increases. During the recovery period, polyuria is observed for several days. ECHT in the blood is accelerated, leukocytosis is observed (18-25 thousand/μl), the disease lasts 2-3 weeks.

A genital form of the disease can appear when the bats are infected with mute streptococci during the breeding process . In this form, catarrhal-purulent inflammation, purulent mastitis in some cases is observed in the mucous membranes of the vagina, regional lymph nodes. The disease begins with the appearance of menstrual symptoms, redness and swelling of the mucous membranes of the vagina, followed by purulent vaginitis. In this form, acute catarrhal-purulent inflammation is visible in the head of the genital organ and in the urethra.

the abortive form , the mucous membranes of the nose become inflamed, a slight enlargement of the submandibular lymph nodes occurs. A sick animal discharges mucous, purulent fluid from the nasal cavity, it becomes difficult to swallow, and the throat is painful when palpated from the outside. In sick horses, fever up to 39-39.5 ^{0}C, slowing down of reactions, loss of appetite is observed, which lasts for 5-7 days and they recover.

In the complicated (metastatic) form , in addition to catarrhal purulent inflammation of the mucous membranes of the nasal cavity and throat, submandibular lymph nodes, the pathological process also includes purulent inflammation of the back of the throat, in front of the ears, and deep lymph nodes of the neck. Metastatic abscesses are detected in the lungs, liver, kidneys and other organs due to the transfer of the causative agent to the blood and spread throughout the body. Abscess is often observed in pre-ear lymph nodes, shoulder, knee, chest, and abdominal lymph nodes. In most cases, mucous membranes and regional

lymphatic glands are inflamed. This condition, in turn, causes difficulty in swallowing and breathing. There is drooling from the mouth, pain when swallowing and signs of choking. If the abscess bursts, pus will come out of the mouth. If the abscess is in the parotid gland, the pus flows into the throat and enters the lungs, causing pneumonia, sometimes pus comes out of the jugular vein pit. If the abscess in the throat and larynx gets into the larynx, it causes *aspiration pneumonia* . It becomes difficult to breathe in and out. Inflammation of the deep lymph nodes in the neck causes the pathological process to move to the pleura and pleuropneumonia. The most common complication of deafness is purulent inflammation of the lymph nodes behind the throat and in front of the ear. These lymph nodes become enlarged, pus forms in them, the horse stretches its neck and stops moving its head.

Inflammation of the mesenteric lymph nodes causes bowel dysfunction, diarrhea, constipation, colic, fever, and weight loss. If the abdominal abscess ruptures, peritonitis occurs and the animal dies. A characteristic symptom of this form is constant fever in the body. In complicated (metastatic) deafness, leukocytosis in blood increases to 20-25 thousand/μl, erythrocytes up to 2 million/μl, hemoglobin by 20-30%, ECHT - up to 75 mm/h. Complicated mute usually ends in death.

Pathologoanatomical changes. A catarrhal - purulent inflammation is observed in the nasal cavity, mucous membranes of the throat, and submandibular lymph nodes of a dead horse when it is typical of a dead horse. In the complicated (metastatic) form, purulent foci are visible in all lymph nodes and internal parenchymatous organs. There is hyperemia in the mucous membranes of the throat, hyperemia and hemorrhage in the mucous membranes of the small and large intestines. Bronchial, mediastinal, mesenteric lymph nodes are inflamed, enlarged, and bleeding. Purulent foci are observed in lungs, liver, spleen, kidneys. Several liters of serous-fibrinous, yellowish-reddish liquid accumulates in the chest cavity. If a horse has died of mute bronchopneumonia, the lungs will look like a sheep's butt and be hard.

Diagnosis. When the disease is typical, a diagnosis is made based on clinical symptoms, epizootological data, pathologoanatomical changes and, of course, laboratory tests. It is difficult to diagnose the disease if the clinical symptoms are only in the respiratory tract. In such cases, a bacteriological examination will be carried out. Dumb streptococci differ from purulent streptococci by the following symptoms:

- up to 250 microorganisms form a chain in a culture of mute streptococci or a smear made from pus, and in a bacillus with pus there are only 10-15 b points (segments) in the chain;

- In 5% blood agar, streptococci of mute produces v-hemolysis, the colony becomes round b, smooth, slimy. V-hemolysis also occurs in the culture of bacilli, but the colonies are in the form of drops and do not join each other, another difference is that the colonies are dry;

- Mute streptococci form a capsule on 5% blood agar. Purulent bacilli do not form a capsule. If these data are not sufficient , biochemical tests are performed.

Differential diagnosis. It is necessary to distinguish the disease of muteness from the diseases of the flu and the flu. It can be distinguished from manga disease by performing maleinization and, if necessary, applying KBR. Influenza spreads quickly and does not have purulent inflammations.

Treatment. Sick animals are separated in a separate bright, clean room. It is advisable to store in the open during hot weather. Feeds with easily digestible soft hay, boiled or steamed roots and fruits are given. If swallowing is difficult, a slurry is recommended. Antibiotics and sulfonamides are used in general treatment. In special treatment, an antiviral drug prepared from mute streptococci is used. Antiviral is injected under the skin of the neck of a sick animal in the amount of 50-100 ml. It is advisable to send it to several places. If the effect of the antivirus is not good, the antivirus will be sent again after a day. Antiviral drug can be used in the treatment of abscesses, washing. In case of hyperplasia of the lymph nodes under the jaw, in front of the ear, the antiviral drug is also injected under the skin of that area. In patients with metastasis, in addition to antibiotics, 33% alcohol is added to 20-30% glucose and 1% norsulfazol in the form of a complex solution. The first time, the amount of alcohol is 150-200 ml, and the following days it is increased from 50 ml.

In order for abscesses to be absorbed faster or faster, fur is cleaned and degreased, and sulfur-mercury ointment is applied. The ointment is applied in a linear pattern. After applying the ointment, a warm bandage is applied. Mature abscesses are treated surgically. The cut abscess is washed with a 1:1000 solution of potassium permanganate or other disinfectants, then wiped with an alcohol iodine solution or washed with the 2nd fraction of 20% ASD. Later, if the regeneration is good, the wound is not washed, but it is necessary to clean the wound with a dry tampon. 3-4 g of calomel and 10 g of bisalol are given per day for gastrointestinal injuries. Caffeine or camphor oil is injected to stimulate the heart. A liniment made of 1 part of turpentine + 2 parts of camphor alcohol is rubbed into the swelling. A tracheotomy is used for respiratory distress. Water supply to sick animals is reduced. Add 8-10 ml of hydrochloric acid to a bucket of water or add 15-20 g of calcium chloride.

AP Alatarsev, taking into account the anaphylactic nature of the disease, recommended adding adrenaline with calcium chloride. The duration of treatment is 5-7 days. Every day, 50 ml of 5% calcium chloride, 10 ml of adrenaline 1:1000 solution are injected into the vein every day.

Immunity. Animals that recover from the disease develop strong immunity, which lasts a lifetime. Even in uninfected horses, the susceptibility decreases after 5 years, which is evidence of the presence of an immunizing subinfection.

Prevention and countermeasures. Mutism has not been recorded in Uzbekistan. However, this disease exists in some countries of the European continent, so there is a risk of introducing the disease. The way to protect vulnerable animals is to strictly control the dangerous border area, the horses and their products crossing the border, and if there is suspicion, it is necessary to check new arriving horses during the preventive quarantine period.

In nature, the main reservoir of the disease is horses, carriers of bacteria in latent form. Horses purchased for breeding or sports must be taken from countries,

farms and farms that are healthy in terms of infectious diseases, checked for infectious anemia, mange, rhinopneumonia and other infectious diseases in the territory of that country before bringing them, and after bringing them 1 During the monthly preventive quarantine, it is necessary to carry out a clinical and laboratory examination of the mute. In order to ensure the resistance of horses, it is necessary to feed them with nutritious food rich in vitamins and minerals, and to keep them in accordance with the hygienic requirements.

If this disease is detected clinically, pathologoanatomically, bacteriologically among horses, within the framework of the Veterinary Regulation, the farm, farm or settlement is declared unhealthy and *restrictions* are imposed. In the unsanitary point, measures are taken to carry out all health measures and prevent the spread of the disease. Entry and exit of new horses to the farm, mixing of horses with other groups is prohibited.

Horses in the herd are clinically examined. Sick animals are separated and treated. Clinically healthy horses are left in the barn and given coarse hay and soft feed. The cage is disinfected weekly with 3% chlorinated lime, 3-5% caustic soda, 10% formaldehyde, monochlorinated iodine. If a disease occurs when the horses are fed in the pasture, the sick are isolated and treated, and the remaining horses are given 50-100 ml of antiviral. If the antivirus is ineffective the first time, it will be sent again after a day.

Horses are tied or kept in a closed place. Horses at the point are kept without being let out to pasture and are subjected to daily thermometry and clinical examination. The sick building is cleaned daily and disinfected every 7 days with 4% caustic sodium, the manure is biothermally disinfected for 3 months. *The restriction* from the unhealthy point is taken 15 days after the last patient has been treated, and the final disinfection is carried out.

Exercise: Diagnosis, prevention and countermeasures of swine flu .

Purpose: To acquaint students with swine flu diagnosis, differential diagnosis, immunity, general and special treatment, preventive means.

Material and equipment:

Smear, microscope, color tables, standard strain, pat.material, paints for smear smear, standard stamp smear, immersion oil, special anatoxins, slide.

Basic concepts.

Diagnosis, differential diagnosis

1. Immunity
2. General and special treatment

3. Quarantine measures
4. Farm health, preventive means

Swine flu (Latin - Influenza suis; English - Swine influenza, Swine (Hog, Pig) flu; Russian-gripp sviney) - is an acute, highly contagious infectious disease characterized by depression, fever, weakness, It is characterized by catarrhal inflammation of the mucous membranes of the respiratory organs, conjunctiva of the eyes and, in severe cases, lung damage.

Trigger. Influenzavirus A suis, an RNA-storing orthomix virus, is a virus belonging to the genus influenza virus A. Its size is 70-120 nm. The virus has hemagglutination properties. The classification of the influenza virus is based on the arrangement of hemagglutinin and neuraminidase types in it in a certain order. The virus is genetically related to A-type, the causative agent of human and avian influenza. The virulence of this virus is determined by the gram-negative, polymorphic, low-virulence hemophilic bacterium Bact. It is more pronounced when combined with Haemophilis suis. Among the laboratory animals, the white mouse, rat, and mouse are susceptible to the virus. It can be grown in cell cultures made from chicken embryos and piglet kidneys and lungs. It is determined by neutralization of antibodies formed against it in the body of pigs infected with the virus, cessation of hemagglutination, complement binding reactions. Influenza virus can be detected in the larynx, bronchial exudates, lung regional lymph nodes, and nasal fluids when symptoms of the disease appear.

The origin of the swine flu virus is believed to have been transmitted to pigs from a sick soldier returning to the United States from Europe during the 1918-1919 human influenza pandemic. In fact, the N1N1 and less common N3N2 antigenic structures, which are more common in the swine flu virus, are also observed in human and avian influenza. Based on the above, it can be said that the human, swine and bird flu viruses were genetically combined to **form a recombinant - hybrid virus** . This is why this "swine" flu virus attracts more attention than the common flu virus in humans, and the effectiveness of traditional flu drugs is lower. To date, 4 serotypes of influenza virus have been identified in pigs: H1N1, H1N2, H3N2 and H3N1.

Exciter endurance. Resistant to external environmental factors, the virus is inactivated in 20 minutes at 60 ^{0} C, in 6 days at 18-22 ^{0} C. It retains its activity for 4 years under vacuum conditions and freeze-drying. Solutions such as 2-3% chloramine, formalin, phenol, caustic sodium inactivate the virus in 5-10 minutes.

Pathogenesis . The virus enters mainly through the respiratory tract, settles and multiplies in the epithelial cells of mucous membranes, causing degenerative and destructive changes there and creating conditions for the development of secondary infection (cocci, bacteria). Haemophilic bacteria are more common in swine flu. Under the influence of the virus, the permeability of capillaries increases, as a result, the entry of toxic substances and pathogens becomes easier. In a pig infected with a virus, the body temperature rises, antibodies against the virus begin to form in the body, and phagocytosis increases. In pigs with low resistance, on the contrary,

secondary infections develop, inflammation begins in the lungs, which turns into chronic bronchopneumonia. If the activity of secondary infection is not eliminated, the pathological process deepens, worsens and the pig dies.

Course and clinical signs . The incubation period of the disease is 1-2 days, sometimes 6-7 days. The disease can be more *acute* , *semi-acute* , *typical* and *atypical* . The disease *is typical, acute* , begins with an increase in body temperature to 41 $^{0-42}$ 0 C, the animal is depressed, loses appetite, conjunctivitis, runny nose, cough appears , moves with difficulty. The mucous membranes of the eyes are swollen and reddened, serous catarrhal exudate flows from the corner of the eye. It becomes difficult to breathe due to the swelling of the mucous membranes of the airways, wheezing sounds when breathing. Nostrils, bronchi are blocked or narrowed by hardened exudate. A sick pig is in a state of depression. He digs the bed and lies down, his sensitivity to surroundings decreases, he has difficulty standing, and cough symptoms appear. Catarrhal exudate from the nose hardens and forms a crust in the beak. In the typical course of the disease, symptoms of lung inflammation appear. He sits like a dog to ease his breathing. The skin of the ears, tail and hooves turns blue due to a decrease in heart activity. Some patients have diarrhea and constipation. Knees, shoulder blades, lymph nodes swell.

The most susceptible 2-8-week-old piglets have influenza in the form of acute catarrhal or catarrhal-purulent pneumonia. In piglets, breathing becomes faster, wheezing is heard in the lungs, and coughing intensifies. Death is often observed in sick piglets of this age. In large pigs, when the flu is acute, the disease usually lasts 7-10 days. In pigs with high resistance, the disease is mild and can recover in 4-6 days. In older pigs, the output usually does not exceed 2-4%, but in suckling piglets it can be 60-70%. Because the disease is severe in suckling pigs and young pigs separated from their mothers. They have inflammation of the lungs. In addition, they develop pleurisy, pericarditis, and dermatitis. Sick pigs lose weight.

the flu is *semi-acute* , it lasts for several weeks, usually in pigs bronchopneumonia, and in some cases pleurisy is observed. Such pigs die in 15-30 days. Those who recover are small and emaciated. Sometimes a relapse is observed in pigs. They have more dermatitis and eczema compared to the acute one. In salmonellosis and pasteurellosis unhealthy farms, piglets die from secondary infection when the flu is subacute.

When the disease is atypical , the clinical symptoms are unknown, coughing, runny nose, rapid breathing, loss of appetite. The lungs are not inflamed, the temperature does not rise. If properly treated, well fed and well kept, sick pigs will recover in 3-6 days.

Pathologoanatomical changes . The disease is typical, when it is acute, upper respiratory organs are inflamed, mucous membranes are inflamed, there is bloody foamy mucous fluid in the nasal cavity, larynx and bronchi, the bronchi are filled with mucous plug and the lungs are swollen. will be Hemorrhages are visible in the mucous membranes. Serous-catarrhal pneumonia is observed in the upper and middle part of the lung, sometimes this change completely occupies the left or right part of the lung. In rare cases, pleurisy is detected. A clear, sometimes slightly cloudy, red

fluid may accumulate in the abdomen, chest cavity, and under the pericardium. The inflamed tissue is harder, pale red or dark red in color, shiny. The mediastinal and mesenteric lymph nodes in the chest and abdominal cavities are swollen, filled with blood and enlarged, and in some cases, point hemorrhages are observed. Hemorrhages are visible in mucous and serous membranes. In the semi-acute stage, the main changes are in the form of croupous - necrotic or purulent inflammation in the lungs. Fibrinous pleurisy and pericarditis are often detected. Catarrhal inflammation is usually observed in the mucous membranes of the gastrointestinal tract.

Diagnosis. The season (autumn-winter-spring), the rate of infection and spread, inflammation of the respiratory organs are characteristic symptoms of influenza. However, in pigs, parainfluenza, chlamydial pneumonia, adenovirus infection, and mycoplasmosis pneumonia also have the same clinical symptoms as influenza. Therefore, the diagnosis based on clinical signs, epizootological data and pathologoanatomical changes is the initial diagnosis. A final diagnosis of influenza is made based on laboratory tests. Nasal fluid, a piece of the mucous membrane of the nose, larynx, lung and lymph node are sent to the laboratory. Blood serum is sent with a referral letter at the beginning and end of the disease to determine the increase in the titer of anti-influenza antibodies in the body. Using immunofluorescence and hemagglutination reactions, the virus or antibodies against it are detected in mucous membranes.

Differential diagnosis. Influenza should be distinguished from plague, pasteurellosis, salmonellosis. Season is not important in plague, death rate is high. Diphtheritic inflammations (cecum, colon) are observed in internal organs, stomach and intestines. In pasteurellosis, fibrinous inflammation is observed in the lungs and intestines. Pasteurella is detected during bacteriological examination. Piglets under 2 months of age are mainly infected with salmonellosis. Characteristic necrotic, diphtheritic and bumpy changes occur in the mucous membranes of the large intestines. Bacteriological examination isolates salmonella.

Hemagglutination inhibition reaction (HATR), KBR, immunofluorescence reaction, IFT and PCR are used to diagnose influenza.

Treatment . Special treatments have not been created. Antibiotics, sulfanilamide and nitrofuran drugs are used to prevent secondary bacterial infections. First of all, patients are immediately separated into a separate dry, bright, clean, ventilated room and maintained with complete blood nutrients. The bedding is changed frequently. Dietary food, jelly, liquid porridge, aromatic porridge are provided.

Immunity. The formation of virus-neutralizing, agglutinin and complement-binding antibodies was detected in the blood of animals recovered from the disease. However, their importance in the course of the disease, the period and level of preservation in the body have not been well studied.

Prevention and countermeasures. To date, this disease has not been recorded among pigs in the territory of our country. To prevent the swine flu virus from entering the territory of our country, it is bought from abroad pigs should be taken

only from a farm, region and country healthy for this disease, and imported pigs should be under veterinary-sanitary control. Keeping pig farms in a closed type, allowing people to enter through a sanitary conduit at the entrance, organizing a dezobarrier at the entrance to the farm, organizing an entry through dezogilams at the entrance to each building, preventing the entry of strangers and all kinds of animals, among the measures for the prevention of this disease. is one. Placement of pigs based on veterinary and sanitary requirements, care, attention to protein balance in feeding, feeding rich in trace elements and vitamins, protection from heat and stress, quick cleaning of the pigsty from manure, regular disinfection, in particular, disinfection of the transport that entered the farm allows to keep the farm healthy from this disease. Since the cooling of the weather is one of the main factors in the origin of the disease, the pigsties should be warm, bright, clean, and the ration should be full of value. There should be no drafts in the pigsty, and the bedding should be dry. Newly brought pigs are kept in preventive quarantine for 1 month and undergo daily veterinary examination.

Pigs and their products coming through the border customs, regardless of the means of transportation (airplane, train, car), must undergo a thorough veterinary-sanitary inspection, the place of origin of the product must be checked for this disease. it is necessary to have a document confirming the authenticity and verify that the product is not coming through a third country; pork and meat products from unhealthy areas should not be allowed to enter the country through customs, even if they are alone or with tourists.

If the disease breaks out, it should be prevented from spreading as an epizootic. It is necessary to bring the sanitary conditions and microclimate conditions of the piggery to the level of zoohygienic standard. Sick animals are isolated and treated. Current disinfection is carried out with 20% freshly slaked lime, 2-3% caustic soda, 1-2% chloramine.

Exercise: Salmonellosis diagnosis, prevention and countermeasures

Salmonellosis (paratyphoid) is an infectious disease of young animals, which is characterized by a rise in temperature, impaired gastrointestinal activity , and diarrhea.

Trigger. The causative agent of the disease belongs to the genus Salmonella , and in calves it is S enteritids dublin, sometimes S tupli murium, in pigs S cholerae suis, in lambs S abortu0Cvis , and in goats S abortus equivi. Found in a polymorphic state , it is well dyed with aniline dyes. Grows well in MPA and broth. Salmonella does not produce indole , releases hydrogen sulfide, does not curdle milk. The varnish is pure and without change in sucrose, glucose mannitol, gas and acid appear in maltose. Monoreceptor O and N sera are used for serological differentiation . White mice are very susceptible to salmonella. Den guinea pigs are less prone. The microorganism releases a strong poison (endotoxin) and affects laboratory animals in larger doses, and when administered intravenously, it also

affects winter livestock. If the toxin of Salmonella affects a person and gets into food, severe poisoning occurs.

Treatment. After clinical examination and thermometry , it is recommended to divide the calves into the following groups : 1) healthy; 2) a disease is suspected; 3) clearly infected; 4) healed calves. Groups should have their own equipment and trainers. Nutritious and high-quality nutrition should be established. Levomycetin, synthomycin, tribrissen are recommended for treatment . Antibiotics with sulfonamides (norsulfazol, disulfan, etazol, sulfa din, sulfademyzin) give good results when complications of pneumonia are observed.

Nitrofuran series furazolidone, furasin, furazolins have a high value. Antitoxic serums used against hyperimmune salmonellosis are very beneficial. It is recommended to give Sintomycin 3 times a day with milk . The first time is 0.04 per 1 kg of weight, and the second and third time is 0.02-0.03. In order to prevent recurrence , after the healing of the wounds, syntomycin administration is continued for another 2 days, and terromycin and biomycin are continued for 3 days . They are given from 0.02 per 1 kg of weight. In addition to these, it is possible to inject penicillin into the muscle . Always work to determine the effectiveness of microorganisms in relation to drugs, increasing the useful coefficient of washing. This task is assigned to laboratories.

E mla sh s c h e m e

Age of sheep	Vaccine dose (ml)	
	First	Second
From 20 days to 3 months	1-2	2-3
From 3 months to 1 year	1.5-2	2-3
First birth	3-4	4-5
From 3 years old	4-5	5-6

Prevention. The fight against salmonellosis is carried out throughout the entire strait, starting from the day the cattle are released. During this period , special attention is given to care and diet along with nutritious and recommended food . It is necessary to carry out these measures with care, especially in the third half of the strait . To increase the resistance of newborn calves , it is recommended to give acidophilin, acidophilic broth culture (ABK) and propion-acidophilic broth culture (PABK). If the disease appears , it is immediately treated with the methods and drugs mentioned above . Newborn animals are vaccinated with the above-mentioned vaccines.

In salmonellosis, sick animals are the most dangerous source of the disease. Therefore, it is necessary to diagnose them in time, isolate them and carry out current disinfection. 2% active chlorine, 20% chlorine lime solution, 5% chlorine (I) -iodide, 2% formalin are recommended for disinfection .

Bacteria-carrying animals are strictly considered and bacteriological and serological examination is carried out.

Control questions

1. Epizootology of the disease?
2. Pathogenesis of the disease?
3. Measures to prevent disease from entering the country?
4. When a disease is suspected, should measures be taken to prevent its spread?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Diagnosis, prevention and countermeasures of colibacteriosis .

Colibacteriosis is an acute infectious disease of young animals . It is found mainly in animals from 1 to 8 days old . Symptoms of the disease: enteritis, sepsis and weakness.

Trigger. The disease is caused by Escherichia Coli, the main representative of microorganisms belonging to the E. Soli group . Several groups of enteropathogenic escherichia are found in calves: 08, 09, 015, 026, 041, 055, 078, 0101, 0115, etc.

Diagnosis. Diagnosis is made based on epizootological data, taking into account clinical signs and pathologoanatomical changes . All these indicators are confirmed by the results of bacteriological tests. To which serological group it belongs will be determined by checking with serum . Microbiological examination is carried out as follows: on the 1st day, the organs and the mucous membrane of the small intestine are taken and planted in artificial media. It is checked by sliding. On day 2, colonies are cultured on Endo, Levin, MPB and Simmons media. On the 3rd day, antigen is prepared from culture media and serologic group is determined. White mice are infected. On the 4th day, biosine is determined. After that, sensitivity to antibiotics is studied.

Treatment. When the disease is accurately diagnosed in time , treatment begins with diet. Instead of colostrum, physiological solution or bitter brewed black tea is given cold. It is even more useful to mix chicken eggs in 1 l of the liquids mentioned above . In addition to being nutritious, it is also rich in lysozyme. Before using antibiotics , it is necessary to determine the sensitivity of the isolate to them. Only then we can choose drugs that affect the microorganism and the treatment will be effective. Synthomycin is used for treatment . 40 mg for the first time, then 20 mg every 4-6 hours. Biomycin, terromycin , tetracycline 15-20 mg 2-3 times, colimycin 15-20 mg, and polymyxin 4 mg are recommended. It is better to give antibiotics with milk. In some cases , when doctors get hold of a newer antibiotic, they immediately start using it against gastrointestinal disease . This is definitely wrong. First, the

sensitivity of the microorganism to the antibiotic should be studied in the laboratory, and then it should be used. If this is not done, the treatment will not help.

Caffeine, camphors are used to support cardiovascular activity. If antibiotics cannot be found, some sulfanilamide drugs - sulfazol, sulsimid, disulfan, phtolazol - can be used. Administration of glucose-saline solutions under the skin or in the abdominal cavity helps to maintain the process of water-salt exchange. The solution recommended by Professor IG Sharabrin (1 l distilled water, 8.5 sodium chloride, 13.0 sodium bicarbonate, 0.3 calcium chloride, 0.5 potassium chloride, 50.0 glucose, 0.2 caffeine, 500 thousand TB penicillin) is also beneficial in enteritis . For calves, 0.5-1 l is administered intravenously, and subcutaneously for pigs, pigs and lambs. A deep enema cleans the intestines. In addition, there are various preparations recommended by a number of scientific testing institutes and laboratories . If they pass the control and arrive with a blind eye, it is necessary to support. Serum and bacteriophages prepared in biofabrics and biosex , in case of colibacteriosis, serum is injected under the skin, and phages are ingested. The treatment effect is very high.

Prevention. Gastrointestinal diseases of young animals occur mainly in farms with poor sanitary conditions and insufficiently balanced feed . Therefore, the main issue here is to increase the resistance of the animal organism along with the elimination of these factors. This event should start from the period of the animal's strait. It is necessary to provide balanced, nutritious feed and organize rations for strait animals . During this period , it is strictly forbidden to give nutritious products such as silage and fodder . It is desirable to organize a diet rich in vitamin nutrients, micro- and macroelements. Giving young animals ABK, PABK, ochko zon juice is one of the factors that increase the body's resistance. The sanitary condition of farms and current disinfection are the main factors in the prevention of gastrointestinal diseases in young animals.

Control questions

1. Epizootology of the disease?
2. Pathogenesis of the disease?
3. Measures to prevent disease from entering the country?
4. When a disease is suspected, should measures be taken to prevent its spread?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Diagnosis, prevention and countermeasures of poultry disease N ' .

Newcastle disease is a highly contagious, acute infectious disease caused by a virus, characterized by septicemia, damage to the central nervous system, gastrointestinal and respiratory organs.

Trigger. Paramyxovirus, polymorphic virions-filamentous, rod-shaped, racket-shaped virus. It agglutinates erythrocytes of humans, cattle, pigs and horses. It is very resistant to external environmental conditions, can be preserved at low temperatures. Frozen chicken can live up to 6 months, and up to a year at 20°C. It dies in 30 minutes at 65-70°C, and in one second at 100°C. It can survive for months at an ambient temperature of 18-20 °C. Chicken feathers can be kept alive for 18 days, and eggs can live for years in the refrigerator. The virus develops well in 9-10-day-old chicken embryos.

Diagnosis. Epizootology, clinical signs and pathologoanatomical changes of the disease are taken into account to make a diagnosis of Nyukasla disease. In addition, the virus is isolated and identified in laboratory conditions. This process is performed with special hyperimmune blood serum using RTGA. To isolate the virus, a 10% suspension is prepared from the spleen or brain of sick or dead birds. It is rotated in a centrifuge that rotates 1500 times per minute and is kept for one hour. Then, adding 1000 TB of penicillin and 1000 mg of streptomycin to the suspension, 9-10- day-old embryos are infected. After 72 hours, the presence of hemagglutinin with RGA, the level of immunity with RTGA is determined using the RTGA method with a diagnostic antigen (any vaccine used against Newcastle disease is possible). After 5-6 months after immunization, the high titer of the antibody in the blood serum indicates the presence of the field strain of the virus in the body.

Prevention. Poultry farms are allowed to bring only poultry and eggs from healthy farms. It is necessary to disinfect vehicles entering poultry farms. All kinds of wild birds should not be allowed to fly into feed stores and warehouses. Disinfection is carried out when each batch of chicks is hatched. The epizootic status of Newcastle disease in the surrounding farms is monitored and studied. Vaccination must be carried out according to the plan. When the disease appears, the diagnosis is made immediately and quarantine is announced. It is strictly prohibited to open and sell chicks, give poultry to other farms, and release eggs under quarantine conditions. All clinically sick birds are destroyed.

Veterinary specialists in poultry factories, state, collective and other farms and state veterinary institutions must perform the following tasks:

- implementation of special veterinary measures (vaccination for disease prevention, diagnostic tests) in poultry farms, settlements and regularly checking the condition of poultry;
- To control the emergence of immunity in vaccinated poultry in farms where vaccination was carried out for the prevention of measles.

When there is a suspicion of the occurrence of Nyukasla disease, heads of farms (poultry factories, state, collective farms, enterprises and veterinary specialists or owners of poultry in private farms) should perform the following tasks:

- not to allow outsiders to enter the farm, not to mix poultry inside the farm, not to remove poultry, eggs and other poultry products, as well as tools, equipment, manure from it;

immediately inform the veterinary specialist of the farm (settlement), the institution and the chief veterinarian of the district about the occurrence of the disease .

The veterinary specialists of the farms and state veterinary institutions take measures to immediately detect the disease and send the blood serum obtained from newly dead birds (3-5 heads) and sick birds (10-20 heads) to the veterinary laboratory for examination.

The diagnosis of Nyukasla disease is made on the basis of epizootological data, clinical signs, pathologo-anatomical changes and laboratory examination results.

After receiving information about the outbreak of the disease, the chief veterinarian of the district:

- immediately go to an unhealthy farm, settlement, eliminate the epizootological focus and identify the source of infection;

- in necessary cases appoints a veterinarian (epizootologist) responsible for the organization and removal of all measures in the place where there is an outbreak of the disease;

- within one day, he sends materials to the district authorities that a quarantine has been announced in an unhealthy farm (settlement), and that a special commission has been formed for the prevention and elimination of the spread of the disease;

- at the same time, the veterinarians of the neighboring districts and higher veterinary organizations are informed about the occurrence of Newcastle disease.

If the disease occurs in cities, individual streets, quarters, or the entire city are quarantined. The following are prohibited in farms and settlements quarantined for Newcastle disease:

- Removal of poultry resistant to Nyukasla disease from buildings;

- access of outsiders to poultry farms;

- sale and export of poultry and products. The following are conducted in unhealthy farms (poultry factories, farms) with regard to the disease of Newcastle disease:

- if a disease occurs in young birds, all sick and healthy chicks are killed by a bloodless method, lost or sent to slaughter. The remaining clinically healthy birds are slaughtered for meat or vaccinated against the disease. These birds are kept in a separate place and slaughtered for meat 2 weeks before the quarantine is lifted;

- eggs obtained during the quarantine period are boiled for at least 10 minutes and used for feed inside the unhealthy farm.

Unhealthy poultry farms and factories are subject to mechanical cleaning and disinfection. Manure and bedding are biothermally neutralized.

Quarantine is canceled 30 days after the destruction of the last sick bird in the unhealthy farm, the settlement, and after full sanitization of farm premises and buildings. Slaughter of poultry for meat is carried out with the permission of the veterinary department and, if deemed necessary, is carried out in special places. All

measures to combat the disease and eliminate it are carried out according to the manual.

Control questions

1. Epizootology of the disease?
2. Pathogenesis of the disease?
3. Measures to prevent disease from entering the country?
4. When a disease is suspected, should measures be taken to prevent its spread?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Diagnosis, prevention and countermeasures of influenza disease of birds .

Avian influenza is a highly contagious, acute viral disease that affects all domestic and wild poultry and migratory birds. The disease is invisible in the form of clinical symptoms such as septicemia, inflammation of the central and peripheral nervous system, gastrointestinal tract, respiratory organs, eyes, joints, crown and earrings.

Trigger. Paramyxovirus, polymorphic virons - filamentous, rod-shaped, racket-shaped viruses, pass through all filters, virus size is 60-80 nm. It often resembles virions that cause influenza in humans. Virions can form cultures in 9-12-day-old chicken embryos, and in 12-14-day-old duck embryos. Repeating the passage several times in embryos ensures an increase in the level of virulence of virions. Virions agglutinate erythrocytes of domestic fowl, erythrocytes of monkeys, guinea pigs, horses, and cattle. The recovered birds have antibodies in their blood serum that inhibit the hemagglutination reaction and neutralize the virions. Virions are very resistant to low temperature (freezing), and resistant to high temperature +100 C kills in 2-3 minutes. PG - disease often occurs as a secondary infection.

Pathologoanatomical changes. The crown, earrings turn blue and swell, mucous fluid flows from the muzzle, bursitis appears, joint deformation is formed, hemorrhages are observed in the mucous membranes. Dystrophic changes characteristic of rhinitis, conjunctivitis, keratitis appear. There will be swelling and hemorrhages in the mucous membranes, subcutaneous tissue, neck and breasts. When air accumulates in the stomach and food is not digested, it wears out and stops, and an unpleasant smell comes out when it is used. Heavy bleeding is observed at the border of the muscular and glandular stomach. Intestinal lymphoid follicles, under the epicardium, blood is also poured. The spleen is slightly atrophied. The lungs are

filled with blood and become swollen, and foci of pneumonia appear. Cerebral blood vessels swell, and blood flow to the brain is observed.

Diagnosis. Epizootological data, clinical signs, pathanatomical changes, laboratory diagnostic tests, virological tests, serological tests, the data obtained from all of them are fully studied and a diagnosis is made, if necessary, a bioassay is conducted. In this case, the 8-10-day-old chicken embryo is infected with the chorionallantois membrane. The results of RTGA, RA, RMA are also taken into account.

Differential diagnosis. It is necessary to distinguish YuPPG disease from parotid, viral hepatitis, pasteurellosis, aerocystitis.

- young birds are infected with paratyphoid, mainly gastrointestinal diseases, the main difference is that bacteriological examination of paratyphoid can clearly resolve the issue of endo and bactoagar culture.
- viral hepatitis also occurs mostly in young chickens, almost all pathologo-anatomical changes are observed in the liver. If necessary, a virological examination is carried out.
- pasteurellosis is mainly in young birds, and in this case, pasteurellas are also found by microbiological examination.
- aerocystitis is a non-infectious disease, the result of microbiological examination, micro-macroclimate indicators, ration and care conditions can easily differentiate this disease.

Treatment. Radical treatment, effective treatment has not been developed. In addition, when the issue is approached from an ethical point of view, diseased birds are destroyed by burning because of the possibility of infection to humans.

Treatment – IKKravesa (1960) applied iodine to infected birds in a ratio of 1:1000 as drinking water, a solution of biovetin in water at the rate of 15–20 mg per chicken 2 times a day. The treatment is repeated at daily intervals.

Immunity and Immunization – A live and dead vaccine against PG disease has been developed. Transvarial immunity was studied one month after vaccination.

Control questions

1. Epizootology of the disease?
2. Pathogenesis of the disease?
3. Measures to prevent disease from entering the country?
4. When a disease is suspected, should measures be taken to prevent its spread?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Exercise: Plague of carnivores and fur-bearing animals .

Plague of carnivores and fur-bearing animals - is an acute, highly contagious infectious disease, clinically characterized by skin rash, inflammation of mucous membranes, fever, pneumonia, and damage to the nervous system.

Historical information - A. Krayevsky registered the first plague from Russia in 1882. The causative agent of the disease was identified by Carré in 1905, in 1912. Defined by I. Engein in 1932 in the former USSR. I. Mirolyubov registers plague in dogs. In 1938, VAPankov writes about the plague of mink and foxes. NVSyurin, A.Pantov and S.Ya. Lyubashenko published a number of scientific works in 1953-1957. Currently, the plague of carnivores is registered in many countries of the world, including our Uzbekistan. It is especially widespread in densely populated areas. In our country, mainly purebred dogs often get sick with this disease, and the disease is more common when they are under one year old. Epizootology of the disease, clinical symptoms, diagnosis and treatment, prof. MP Parmanov and G. Yaroshenko conducted scientific and practical work.

The causative agent is the causative agent of the disease, a filterable virus that stores RNA and belongs to the group of paramyxoviruses. The virus is immunobiologically identical and antigenically related to human measles virus and rinderpest virus. When dogs are infected with the measles virus, they become resistant to their distemper virus. The opposite is also observed, that is, in dogs infected with plague, antibodies to the measles virus appear. According to VGSlugin (1984), serum prepared for rinderpest neutralizes the virus of canine distemper.

Resilience of the virus - the virus of carnivore plague is quite resistant to environmental factors. At minus temperature, the virus retains its properties for up to 5 years. The virus can live in the organs of dead animals for up to six months at 20 ^{0} C. It can remain virulent in the blood for one month and in mucous fluids from the nose for up to two months. Boiling temperature kills the virus immediately, and when heated at 60 ^{0} C it dies in 30 minutes, 1% lysol kills in 30 minutes from ultraviolet light, 2% alkali solution inactivates it and kills it. Effect of antibiotics on the virus does not

Economic damage - due to pestilence, industrialized fur farms suffer huge economic damage. 10-50% mortality is observed. As we know, every fur is sold in the world market only with foreign currency. In special dog breeding and breeding farms, the death rate is 60-90%. Purebred dogs bought for households for several thousand soums will die 100% if they are not vaccinated. In addition, large expenses are spent on quarantine measures. -cost required.

Epizootology of the disease - the following animals are prone to plague of carnivores. Dogs, foxes, mink, sable, sable, badger, jackal, wolf, cat and jackal. Carnivores are mostly susceptible to plague under one year of age. Lactating young animals are less likely to get sick. The reason for this is the transfer of immunity through mother's milk. Young carnivores get sick quickly if they are born from unvaccinated or unrecovered foals. Colostral immunity lasts up to two weeks. vaccination is necessary. Susceptibility to the disease and its progression depend on a number of factors. If the feeding and feeding is not a complete balanced diet, the conditions of failure to implement the living conditions at the required level will

lower the resistance of the body of carnivores of this species. As a result, the disease accelerates and worsens.

Pathogenesis - when the virus enters the body through mucous membranes, it develops in the lymphoid tissues. Then, it enters the blood and lymph and spreads throughout the body. As a result, fever rises, inflammation occurs in the eyes, respiratory organs, and the gastrointestinal system. At the same time, inflammatory and degenerative changes occur in the liver, kidneys, brain and spinal cord, as well as in the skin. Injury to the brain and spinal cord affects the work of the moving organs. Antibodies against the virus appear in the blood after 10 days. occurs and persists for up to two months. In the pathogenesis of the disease, the causative agents of secondary infection: salmonella, escherichia, pastrella, toxoplasma, cocci, etc. play a positive role. The disease can occur simultaneously with hepatitis and parvovirus. .Secondary and mixed infections, helminthiasis change the immunobiological characteristics of the organism, increase its susceptibility to plague, aggravate the course, increase death.

Clinical signs - the latent period of the disease lasts up to one month. Depending on the manifestation of clinical symptoms, it is manifested in lung, intestine, nerve, skin and mixed cases. The manifestation of the plague depends on the reactivity of the organism and the virulence of the pathogen. Therefore, a virus belonging to the same type can manifest several different clinical conditions. From fever to nervous system disorders. due to this, it becomes difficult to make a diagnosis in some cases. The virus has a strongly developed tropism feature.

In dogs, the disease occurs in acute and chronic cases. Usually, the disease begins with an increase in fever of 1.3 ^{0} C, and in 1.5-month-old dogs, it manifests in an atypical form and does not have a temperature.

Acute course - in the acute course, clinical signs are evident. The disease begins with an increase in fever (39.7-41 C). After a couple of days, the temperature will drop a little. If pneumonia begins, the fever rises again. At the onset of the disease, the animal's behavior changes, its activity decreases, it trembles, as if it is afraid of something or has a strong stutter. Often, the danger of the upper respiratory organs begins, dogs bite their nose with their claws. Mucous fluid flows from the nose (exudate). When breathing, it is felt that the nose is blocked. The cough will be short at first, then stronger and expectorant. ***Semi-acute course*** - in this case, the fever rises and lasts for a day or two. He becomes depressed, weak, has hiccups and panics, avoids light, suppresses his appetite, and has a dry nose. His nose is stuffy and it becomes difficult for him to breathe. Breathing becomes faster and harder. If the lungs are auscultated, a wheezing condition is observed. Arrhythmia is observed as the pulse accelerates. The eye becomes inflamed, dry, and the lids stick together. There will be conjunctivitis and keratitis. At the same time, an acute inflammation of the stomach and intestine occurs, which causes constipation at first, and then diarrhea.

Chuma exanthema - small red spotty rashes appear on the skin of the nose, lips, ears and abdomen, as well as in the groin area. Later on. In the place of these spots, blisters, the size of a penny, appear. The surface is shiny, and yellow liquid pus

appears inside. Later, these blisters burst and dry, and brown crusts appear. Eczema with exudate is also found around the ear holes. Some dogs have strong hyperkeratosis (corny coating) on the top of the skin where the joints fold.

Nervous form - most often plague (chuma) occurs in the form of nerve, (nerve). Nervous breakdown begins with short-term excitement, and sometimes there is also an aggressive state (aggressiveness). The whole body sometimes and some muscles fly and tremble. Walking coordination of dogs is disturbed. From time to time he has seizures and it turns into paresis. Often the back leg becomes paralyzed and cannot walk. Paralysis also occurs in the bladder, rectal sphincter, and superficial nerves of the head. Certain changes in the composition of the blood also occur in patients. Leukocytosis is observed, erythrocytes and hemoglobin increase. When the disease attacks, anemia is observed. The duration of the plague varies. In mild cases, recovery may take a week, in severe cases, it may take months. Usually, after two or three weeks, it is observed in a state of nervous breakdown, and when they recover from the disease, certain conditions such as flying muscles, paresis, paralysis, blindness, deafness, and loss of sense of smell remain. Mortality in dogs is around 50-85%.

Pathanatomical changes - if death does not occur acutely, or at least during the acute phase, death is very emaciated. Pus has hardened around the eyes and nose, the pupil is enlarged, as if the eye is protruding, small erosions and ulcers appear around the lips and nose. Vesicular and pustulosis dermatitis is noticeable on the skin of the abdomen. Pustules and vesicles and lymphoid infiltration occur in the skin epidemic. The respiratory tract is inflamed and clogged with catarrhal and purulent exudate. Blue spots are visible in the lungs. Internal lymph nodes in the chest and abdomen are swollen and, when cut, ooze mucous fluid, bluish-red color. Encephalomyelitis without purulence is observed in the head and spinal cord, the vessels are filled with blood, slightly swollen, and appear in a bleeding state.

Diagnosis - its epizootology, clinical signs, pathanatomical changes are taken into account to make a diagnosis of plague. Virological examination is carried out and laboratory methods are used. It is one of the epizootological data, taking into account his age (up to 1 year), it is noted that vaccinations were carried out on the pure breed. The clinical symptoms are based on injuries of the respiratory organs, inflammation of the mucous membranes of the eyes and nose, hyperkeratosis of the skin, injuries of the nervous system.

Among the most characteristic panatomic signs, changes such as bleeding in the bladder, duodenum, and rectum are taken into account. In laboratory practice, a bioprobe is placed. It is recommended to use RSK, RDP immunoenzyme reaction, passive hemagglutininase (RPGA) RZGAs. It is necessary to distinguish plague from the following diseases.

Differential diagnosis should be distinguished from leptospirosis. Leptospirosis manifests itself in two forms. In the case of hemorrhagic and jaundice, the hemorrhagic condition develops rapidly and dies in two or three days. When jaundice occurs, mucous membranes become very yellow. The main separation method is bacteriological and serological examination. In this, leptospira are found

and identified. In addition, hyperimmune serum and streptomycin are of good benefit in leptospirosis.

Infectious hepatitis mainly affects young dogs under 1 year of age. On examination, the liver in hepatitis is greatly enlarged and strongly yellow. And the passage of the plague in the nervous form is separated from the fever. Foam flows in Chuma. And in case of fever, there is a strong excitement, the feeling increases, the appetite increases, he eats and swallows things that he cannot eat. When the lower jaw is paralyzed, when it is split open, all kinds of inedible objects come out.

Babesh-negri corpuscle is found by historesection. In Aujeski's disease, severe itching and scratching are observed along with nervous disorders.

Parvavirus enteritis can start suddenly and add to it. The temperature is high, RGA, RTGA reactions are performed with pig erythrocytes.

Bacteriological examination solves the problem in salmonellosis, and in pyraplasmosis the pathogen is found from peripheral blood.

Treatment - hyperimmune blood serum and gamma globulins are used to treat plague. Gamma-globulin, which is used in humans against measles at the beginning of the disease, also gives good results. 3 ml of the drug is injected intramuscularly. The use of interferons is extremely beneficial. It is recommended to use three groups of medicines at once or in parts. Antibacterial (against secondary infectious agents), symptomatic (to reduce fever, to restore heart function) for stimulation ("V" group vitamins, cocarboxylase, ascorbic acid, etc.) from antibacterial drugs, penicillin 10 thousand IU subcutaneously or intramuscularly three to four times a day; Ecmonovacillin 10-15 M.YeD per 1 kg of weight between the muscles once a day; streptomycin 10-20 thousand IU per 1 kg of weight; administration of sulfadimycin, sulfadimitoxin, norsulfasol 0.5-1.0 3-4 times a day leads to a good result.

Phthalozol, levomycetin, enteroseptol and cerucal are given 0.25-05 g per kg of body weight 3-4 times a day against gastrointestinal disturbances. For the purpose of symptomatic treatment, drugs of the most common spectrum are used: 0.2-0.5 g of acetylsalicylic acid is taken; 24 ml of 50% analgin solution is injected into a vein; 2-3 ml of 50% analgin solution is injected subcutaneously and 24 ml is injected into a vein; 2-3 ml, 20% camphor oil, 05.-1.5 ml are injected under the skin. 1.5 ml calcium gluconate, 1% diphenhydramine 1 ml intramuscularly. For paresis, 0.005% prozerin 1 ml is injected subcutaneously for 10 days.

2% alkali, 5% solution of chlorine lime, 5% Lysols are used for disinfection. At home, 2% chloramine is recommended. A specially heated insulator is used in fur farms.

Prevention and Loss – general prevention consists of:

It is strictly prohibited to bring dogs and fur animals from an epizootic unhealthy farm. Permission is granted only to healthy farms. After delivery, they are kept in preventive care for 30 days. All meat animals participating in exhibitions or shows are first vaccinated and then allowed. In general, veterinary sanitary measures and zootechnical parameters increase the level of usefulness of active immunization. Only healthy people are vaccinated. It is not possible to vaccinate during hot, cold and rainy weather.

In order to prevent the disease from spreading, the official leaders and specialists are obliged to carry out the following veterinary and sanitary measures

- foreigners are not allowed to enter farms or kennels;

-Dogs and other herbivores are not allowed to enter.

- In the 2nd week before the holiday, all inventory, tools and equipment are disinfected (2% alkali, 5% chlorine lime, 5% lysol, 2% chloramine).

- Dezobarer, dezokovriks are treated with 2% alkali.

-Dogs and rodents are brought from epizootic healthy zones and farms.

- Carnivorous and fur-bearing animals for sale are vaccinated 15-30 days before sale, regardless of the period of vaccination. In accordance with the quarantine requirements, carnivores and fur-bearing animals are not allowed to enter or leave the farm.

- The sick are treated, then the sick are vaccinated.

-G right waste is biometrically disinfected.

-Skin and fur removal of dead and slaughtered animals is done only in isolators.

- skins taken from patients and suspected patients are dried at 25-33 ^{0} C for 3 days. During the 10th day, it is kept at 18-20 ^{0} C. Veterinary certificate is required for transportation in all vehicles.

Control questions

1. Epizootology of the disease?
2. Pathogenesis of the disease?
3. Measures to prevent disease from entering the country?
4. When a disease is suspected, should measures be taken to prevent its spread?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

STUDY MATERIALS FOR LABORATORY EXERCISES

Laboratory training : Anthrax diagnosis.

The purpose of the lesson: to learn the rules of obtaining and sending the material to the laboratory for testing for anthrax. Mastering examination of patmaterial by microscopic, serological (PR) methods. Differentiation of the pathogen from saprophytic bacilli.

Bacillus anthracis **is** the causative agent of anthrax *(* Belonging to the genus Bacillus). It is an acute infectious disease manifested by the appearance of intoxication, fever, septicemia, carbuncles in most agricultural and wild animals, as well as in humans. The latent period of the disease lasts 1-3 days and passes in a severe, acute and semi-acute state. A few minutes after the appearance of the first convulsive symptoms of the disease, it dies. In an acute attack, the body temperature rises to 41-42 ^{0} C and shivers. It dies after 2-3 days. The above signs are also observed during the semi-acute period, but they are not very characteristic and are a little longer. It is also possible that the situation will ease for a certain period of time. But then it happens again and the sick animal dies. The disease can last 8-10 days. Atypical and chronic course is rare. Pigs are in the state of anthrax angina, the body temperature rises to 40-41 ^{0} C and lasts for 1-3 days and then drops. Death occurs as a result of suffocation due to swelling and constriction of the throat. If it dies, it will be very swollen. A liquid mixed with blood flows from the natural pores. The blood vessels in the subcutaneous tissues are filled with blood, the muscles resemble a red brick color and become pale. A large amount of serous-hemorrhagic fluid accumulates in the chest and abdomen. The spleen enlarges several times, is full of blood, the pulp is loosened, and when cut, a blackish or coffee-colored thickened liquid flows. When anthrax is suspected, it is forbidden to open the corpse.

Padmaterial. The base of the ear on the lying side (below) of the corpse is tied from both sides, cut, and the cut sides are burned with a heated spatula. The cut ear is wrapped in gauze soaked in 3% boric acid, wrapped in cellophane, wrapped again with parchment paper, and placed in a hermetically sealed container (carton, metal box). The site for blood sampling is disinfected, blood is taken, and the site is burned on fire or with a heated spatula. From pigs - throat lymph nodes and pieces of swollen connective tissue are taken. If anthrax is suspected during the rupture, the rupture is stopped and a part of the spleen is taken for examination.

1. Microscopy. Smears made from patmaterial are stained with Gram and capsules by Mixin or Romanovsky Gimza methods. The causative agent is gram-positive, rods, short chains, or pairs, singly. The sides of the stick facing each other are cut straight, and the exposed sides are curved like a moon. Often, in smears made from feather material obtained from pigs, the shape of the trigger can change: the rods are short, thick, bent or granular, and the middle or both ends are swollen. Forms a capsule (in an animal organism or special environment), forms spores (in culture and external environment). In culture smears, *B. anthracis* forms long chains of rods. Motionless. An immediate response is given regarding the approximate result of the microscopic examinations, and it is noted that other examinations are in progress.

2. Bacteriology. Planting GPB from patmaterial on GPA (pH 7.2-7.6) and growing in a thermostat at 37 ° C for 18-24 hours. *B. anthracis* is aerobic. GPA forms smooth, slightly dull, gray, wide (*R -shaped) colonies.* The center of the colonies is darkened, the edges are curled, like curly hair. Colonies look like "jellyfish heads" or "lion's tracks" when viewed under a microscope.

GPB remains clear and a soft cottony sediment forms at the bottom. When the test tube is tapped, the precipitate breaks up into small pieces or rises like a cloud. Sometimes the culture grows diffusely (slight turbidity), producing moiré waves when tapped.

In suspicious cases, in order to distinguish the causative agent of anthrax from saprophytic bacilli, its motility, hemolytic properties are determined, fluorescent microscopy, phagotyping, "Coral" test are conducted, and laboratory animals are infected. The causative agent of anthrax is mobile (it grows in the form of an overturned spruce in GPJ, after 3-5 days, the gelatin dissolves and a funnel-like shape appears), does not cause hemolysis in blood agar, forms a capsule in the body, sensitive to penicillin - "Coral" test is positive (1 ml medium contains 0.5; 0.05 TB of penicillin culture is planted in GPA, grown in a thermostat at 37-38 ° C for 3 hours, the causative cell becomes coral). Fluorescence microscopy is performed using fluorescent anthrax serum "OKVC". With a positive result, the cell contour gives radiation to four or three pluses.

Phagotyping: the flowing drop method ("Gamma - MVA" or "K" VIEV) is performed with anthrax bacteriophages in test tubes or in the micro method. The test culture is inoculated evenly onto GPA slants in 6 test tubes. 15 minutes is placed in a thermostat at 37 ° C. Then one drop of phage agar 10 mmis added to 4 test tubes leaving 8 from the edges and placed on a tripod. At 37 ° C, phaseolysis appears after 6-8 hours, after 12-18 hours it becomes more pronounced - that is, the culture does not grow in the flow paths of the drop, the culture grows around it as usual. gives the shape of a "border".

3. The biotest must be performed on the day of delivery of the patmaterial. 0.1-0.2 ml of the base of the tail to 2 white mice, or 0.5-1 ml of the suspension of the abdominal part to guinea pigs. Animals are observed for 10 days. The dead animal is dissected, and thoroughly bacteriologically examined.

Serological testing (PR)

If the ear is anesthetized, an additional PR is also placed. If the material is contaminated and not suitable for bacteriological examination, it is limited to placing PR only.

The final diagnosis is made:

- if the causative agent of anthrax is isolated from the litter, at least one of the infected animals dies, and the culture is isolated from it.

- if the culture does not grow in the nutrient media planted with the litter material, but even one of the animals biotested with this material dies, and the causative culture is isolated from its organs.

- if the luminescent microscopy results are positive and capsular bacilli are found in smears made from cotton material.

- from the same (old) material, when the PR result is positive.

Differential diagnosis . It is necessary to be able to distinguish between blackworm, pasteurellosis and parasitic diseases of the blood. Cattle are mainly infected with emkar from 3 months to 4 years old. In case of anthrax, animals of all ages and all types are affected, and the result of bacteriological examination is taken into account. In the case of blackheads, there is a well-defined swelling in the fleshy parts of the body. In pyeterellosis, there is an inflamed tumor in the subcutaneous tissue, which is injured, and the causative agent is Pasteurella. In parasitic diseases of the blood, the parasite is observed in the blood smear.

Treatment. Sick animals are immediately transferred to an isolator and treated. Hyperimmune blood serum is used to treat anthrax. It is injected under the skin for prevention and treatment. 15-20 ml of this drug for prevention in horses, cattle, camels, 100-200 ml for treatment, 15-20 ml and 100-200 ml in cattle, deer, 8-10 ml in sheep, goats and pigs and It is used in the amount of 50-100 ml. It is also sent into a vein. Passive immunity lasts 14-15 days. Hyperimmune blood serum gives better benefits if it is added with antibiotics (penicillin, biomycin, streptomycin, ekmonovocillin). The whey is heated to 37^{0} .

In order not to physically torture the animal, it is first tested by injecting 0.5-1 ml of serum. 100 kgPenicillin is injected 3 times at a dose of 500,000 TB per body weight, intravenous 1 gterromycin in a 10% solution for three days has been proven to give good results. Streptomycin and tetracycline are injected intramuscularly 4 times a day. When the disease occurs in the form of a carbuncle or a throat tumor, the injection of a 3-5% solution of carbolic acid around the pathological process gives a good result. Globulin is also recommended for treatment.

Immunity. 1. There is a liquid vaccine made from 55 strains of anthrax. It is 20-25 million in 1 ml. is a live spore vaccine. The vaccine is administered subcutaneously for preventive and mandatory vaccination.

Young animals are not allowed to be vaccinated before 3 months of age. In sheep and goats, 0.5 ml is injected into the neck, chest or inner side of the thigh. 1.0 ml is injected into the neck of horses, cattle, deer, camels, fur animals, behind the ears or inside the thighs of pigs. Immunity appears after 10 days and lasts up to eighteen months.

Dry spore strain 55 vaccine is dissolved in sterile saline or distilled water. It is only sent under the skin

Control questions

1. Explain the rules for receiving patmaterial.
2. Methods of laboratory examination of anthrax.
3. Characteristics of *B. anthracis*

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .

2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory exercise : Determination of protein disease

Complement fixation reaction (CBR) is one of the traditional serological reactions used in the diagnosis of many viral diseases. The name of the reaction, which consists of two separate steps, indicates to some extent the nature of the work performed. In the first stage, antigen and antibody are involved (one of them is known in advance), and a pre-titrated certain amount of complement is needed. Due to the matching of the antigen and antibody complex, the complement is attached, which, in the second stage, is shown by the indicator (mixture of ram erythrocytes and antiserum-hemolysin).

If complement binds due to combination of antigen and antibody, then erythrocytes are not lysed (positive KBR). In negative KBR, unbound complement participates in hemolysis of erythrocytes. (Fig. 71).

Stopping hemolysis is positive - KBR (+)

negative KBR - hemolysis (-)

CBR is often used in practice for diagnosis, detection and identification of viruses, detection and titration of antibodies in blood serum.

The main components of KBR are antigens (known and sought), antibodies (known antisera or tested serum), complement, hemolytic serum and ram erythrocytes; an isotonic solution of sodium chloride with a pH of 7.2-7.4 or different buffer solutions are used instead of a diluent.

Antigens and sera are anticomplementary in nature, and can also adsorb complement, that is, stop hemolysis and show the result of the reaction incorrectly. To get rid of anticomplementarity, antigens are purified by various methods, ie, acetone, freon, ether, chloroform, and the like, depending on the type of tissue used for the antigen and virus. Sera are purified from anti-complementarity by heating and complement treatment by other methods.

are prepared from the organs of infected animals, from the allantois or amniotic fluid of chicken fetuses infected with the virus, and from the liquid medium of virus-infected cell cultures . Antigen preparation for KBR in viral infections differs in many ways from that of bacterial infections. This is related to a number of specific features of the virus.

First , in order to separate the viral antigen from inside the cell, in most cases, it is necessary to destroy the infectious material and undergo additional processing during the separation of the antigen.

Secondly , the greater thermolability of virus antigens compared to bacteria. In most viruses, the complement-fixing antigen is associated with infectious particles, and their destruction is parallel to the loss of infectivity. Also, the material for obtaining antigen should be taken within one to two hours after the death of the animal, if possible while it is alive.

Preservation of virus-containing material with various disinfectants does not give positive results in most cases and often leads to destruction of viral antigens.

Thirdly , fixation of complement will not be uniform due to different ratio of antigen+antibody concentration. An antigen+antibody complex is formed only when they are equal in number; When the number of antibodies is high, the fixation of complement decreases suddenly, the active complex of antigen + antibody binds to the antibody, because the active surface of complement is very small. This situation is also observed when the number of antigens is more. Because the reduction of complement fixation is very fast. Prior titration of antigen and antibody is required for optimal binding of complement.

Fourthly , the small size of the virus fragment that enters the antigen+antibody complex is very small, so the area of complement fixation is small. As a result of increasing the size of the antigen+antibody complex by prolonging the complement fixation period (18 hours at 4 ° C), the sensitivity of the reaction increases, but its specificity decreases; Also, fixation of non-specific antigens increases due to long-term fixation.

Fifth , high procomplementary activity of viral antigen. In order to prevent the fixation of non-specific complement, it is necessary to completely purify the viral antigen from tissue enzymes. A major obstacle in the diagnosis of human and animal viral diseases using KBR is the uneven accumulation of viral antigens at different stages of the disease. KBR is used to determine the type and variants of the protein virus, to check the production and strain of the protein virus, and in scientific research. Protein-pair is a high, contagious, acute disease of ungulates, characterized by a rise in temperature, vesicular inflammation of the mucous membranes of the oral cavity, the place where the hoof joins the skin (venchik) and the udder. , in young animals, it is accompanied by injuries of the myocardium and skeletal muscles.

Protein disease is registered in most countries of the world. The latent period lasts 1-3 days, in some cases it lasts up to 7-10 days. Visible symptoms of this disease are vesicular inflammation of the mucous membranes of the mouth and skin.

In cattle and pigs, protein disease is acute, but in older animals it is harmless (treatable). First of all, there is a deterioration of appetite, increased salivation (Fig. 72), and an increase in body temperature (40.5-41.5 ° C). After 2-3 days, aphthae appear on the inner surface of the lip, on the tongue. (73 pictures). Some animals develop aphthae between the hooves and on the udder. After a day, aphthae burst and erosion appears in its place. (76 pictures). After 2-3 weeks, the erosions will end and the animals will recover. In pigs, sheep and goats, lesions are observed mostly on the legs and in some cases on the mucous membrane of the mouth. (Fig. 77). In most cases, the udder is injured. In young animals, protein disease is worse (mortality 80% and higher), without aphthae, hemorrhagic inflammation of the intestine and degenerative changes in the heart muscles (tiger-like heart) and similar changes also found in skeletal muscles.

The virus belongs to the Picornaviridae family and the aphtovirus genus. It stores RNA and has a supercapsid shell. The virions consist of small particles in the shape of an icosahedron. Protein virus is resistant to environmental factors. Virulence

can be preserved for 67 days in aphthous walls, 39 days in liquid feces, and 103 days in running water. The best disinfectant is a hot solution of 2 or 3% sodium bicarbonate and 1% formaldehyde.

50% of recovered large horned animals shed the virus for 8 months, some even up to 2 years.

Even ungulates are sensitive to the protein virus under natural conditions. In sick animals, the virus can be isolated during the latent period of the disease (from milk, semen, saliva). A high amount of the virus is stored in the epithelium and vesicle fluid. Excreta and secretions of sick animals are contagious for more than 10 days. Virus transmission is observed for a long time after recovery from illness

The virus is cultured from natural hosts and laboratory animals: newborn mice and rabbits, guinea pigs, 60-day-old hamsters,

Determining the type of protein disease is done in the laboratory.

The diagnosis of protein disease is based on epizootological data only on the damage and high contagiousness of ungulates, clinical signs (vesicular lesions on the mucous membranes of the mouth, skin of the legs and udder), pathologoanatomical changes (injury of the intestine and heart muscle, death of young animals) laboratory based on the results of investigations.

Sampling: For laboratory tests, the wall and contents of the aphthae of 2-3 sick animals, the mucous membrane of the tongue (from a large horned animal, the muzzle of a pig, the skin fold and between the hooves, Y.sh.h., m.sh.h., cho' of camels and camels) 5 gis taken at least.

Blood is taken from the carcasses of all types of small animals - lymph nodes of the head, pancreas or heart muscle, when the temperature of the animal rises without a plague. Esophagus-pharyngeal mucus is taken using a special probe to check for virus transmission. It is necessary to prevent the spread of the virus in the material taken from an unhealthy farm to the external environment and to protect the employees who work with the infectious material.

For this: a) the veterinary worker on the farm must have sufficient skills in obtaining material from a sick animal; b) for obtaining material - tweezers, scissors, napkins, thick-walled flocs, leucoplaster, rubber stopper, sterile mixture of 50% glycerin in isotonic sodium chloride solution, thermos with cooling mixture, 2% - NaOH or 1% solution of acetic or lactic acid, special from clothes there should be robes, overalls, a coat or cap, rubber boots, gloves, etc.

After all the necessary things are placed in the container and go to the unhealthy farm, they are put on before entering the room where the sick animals are;

c) after taking the material from the sick animal, the instruments, mask, gloves are immersed in a disinfectant solution, and the outer part of the vial and thermos is treated with a disinfectant solution.

In the place where people and things are inspected (sanpropusknik), all clothes are removed and showered. The protein virus lives in the nasal cavity of a person for up to 7 days. Therefore, contact with healthy ungulates should be avoided as much as possible for 7 days after visiting an infected farm. A material sample without signs of deterioration is frozen after being placed in screw-cap or tightly closed vials, if there

are no conditions for freezing, a preservative solution is poured (50% sterile glycerin solution in an isotonic solution of NaCl). A label is affixed to the vial, indicating the type of animal, the name of the material, its quantity, the time of receipt, and the sender's address. The vials are placed in a metal airtight container, sealed, and placed in a thermos with ice and sealed again. A letter of referral is attached to the material and the following is indicated in it: the time of receiving the material, reporting on the epizootic situation in the farm regarding protein disease, and signed by the veterinary officer. The material is sent through a special veterinary officer. In the laboratory, a separate room (with a box and a box room) is allocated for working with the protein virus, where there must be equipment necessary for performing diagnostic work (preparation of material, placing KBR, biological experiments, etc.) . When working in boxing, special clothes and shoes are completely changed, rubber gloves and a face mask are worn. Dishes and tools are boiled, special clothing is placed in a container and autoclaved, tables, floors, walls are treated with a disinfectant solution, and then irradiated with UBN.

The material brought to the laboratory and its consumption are counted with an accuracy of 0.1 mg. The material brought to the laboratory is kept in a sealed refrigerator closed with a key until it is checked and during its use. After the work is completed, a document is drawn up about the destruction of the material left over from the check or the mortars where the biological experiment was carried out.

Testing for protein disease in the laboratory includes: finding and identifying the antigen of the protein virus (determining which type and variant it belongs to);

detection and titration of anti-viral antibodies in the blood of convalescent animals using radial immunodiffusion reaction (RIDR) and indirect immunofluorescence reaction (BIFR).

Detection and identification of protein virus antigen using KBR.

Components of the reaction: a) an epizootic strain of the tested viral antigen obtained from an infected animal;

b) serum of a hyperimmunized guinea pig with a variant and species-specific strain of the protein virus (produced in a biofactory);

c) antigens in the control are a type and variant specific strain of the protein virus (produced in a biofactory);

d) compliment - dried fresh normal guinea pig serum;

e) hemolysin developed in a biofactory;

f) 0.85% solution of pure table salt in distilled water of 2% mixture of ram erythrocytes in physiological solution;

g) to other viruses causing vesicular damage, specific sera and antigens. collection.

KBR can be placed in different sizes:

if the total volume is 1 ml - 0.2 ml of each component is taken; In the case of 0.5 ml - 0.1 ml of each component or in the micro method - if the total volume is 0.125 ml - each component is taken in a volume of 0.025 ml.

Preparation of protein antigen. The aphthae wall taken from a sick animal is cleaned from the preservative liquid using a physiological solution with a pH of 7.4-

7.6, dried with filter paper, weighed on a scale, after grinding, it is placed in a porcelain mortar with a small sterile glass. mix well until a mass of the type is formed. 2 ml of physiological solution with a pH of 7.4-7.6 is added to twice the mass of the aphtha . 1 gThe resulting 33% suspension is left (extracted) at room temperature for 2 hours, 20^0Cfrozen at -10,- for 5-18 hours. After melting, 3-5 thousand ail/min. Centrifuge for 15-30 minutes. The liquid on the sediment 58^0Cwill lose its activity for 40 minutes, if the liquid is in small quantities, it will be reactivated for 10-15 minutes. It is centrifuged at 3,000 rpm and then used as an antigen for KBR.

Preparation of hemolysin. Hemolysin is titrated when a new series is obtained. Titration is carried out according to the generally used method. Hemolysin produced by biofactories is sometimes preserved 1:1 with glycerol, so when preparing a basic (1:1000) dilution of hemolysin, 0.2 ml of hemolysin and 9.8 ml of saline (1:100) are used, **then** It is prepared by diluting it 1:1000: 9 ml of physiological solution is added to 1 ml (1:10) of hemolysin. From the main (1:1000) solution, the next ones are prepared based on the scheme shown (Table 18). After obtaining the required dilution of hemolysin, the titration is carried out according to the table in scheme N19. For hemolysin titration, it is necessary: 1) control of spontaneous hemolysinization of erythrocytes (0.5 ml of 2% - erythrocyte suspension + 2 ml of physiological solution); 2) hemolysin control (0.5 ml of 1:1000 diluted hemolysin + 0.5 ml of 2% suspension of erythrocytes + 1.5 ml of physiological solution) to exclude hemolysis of erythrocytes without complement; 3) control of complement to exclude hemolysis of erythrocytes without the participation of hemolysin (0.5 ml of 1:20 diluted complement + 0.5 ml of 2% erythrocyte suspension + 1.5 ml of physiological solution); 4) control of the hemolytic system (0.5 ml of 1:1000 diluted hemolysin + 0.5 ml of 2% - erythrocyte suspension + 0.5 ml of 1:20 diluted complement + 1.0 ml of physiological solution). As a result of titration, the most recent titer of hemolysin is determined. So, in the presence of complement, the ability to cause minimal hemolysis in erythrocytes.

In the given scheme, the dilution number of hemolysin is 1:3000.

Table 18. Scheme of preparation of diluted hemolysin

Components, ml	**Dilution of hemolysin**							
	1:1500	**1:2000**	**1:3000**	**1:4000**	**1:5000**	**1:6000**	**1:7000**	**1:8000**
Hemolysin 1:1000	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Physiological solution	0.5	1.0	2.0	3.0	4.0	5.0	6.0	7.0

Table 19I. Hemolysin titration scheme.

Components, ml	**Dilution of hemolysin**							
	1:1000	**1:1500**	**1:2000**	**1:3000**	**1:4000**	**1:5000**	**1:6000**	**1:7000**
Hemolysin	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5

Physiological solution (sera instead of antigen)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
1:20 diluted complement	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
2% erythrocyte suspension	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
37 °C	It is kept in a water bath for 10 minutes							
Titration result		-	-	-	++	+++	++++	++++

In the first three controls, hemolysis should stop completely, and in the fourth control, complete hemolysis. For the main experiment, the last titer is obtained from a 4-fold concentration of hemolysin (working dilution);

For example, the latest titer of hemolysin is 1:3000, its working dilution is 1:750.

If it is preserved with glycerol (1:1), then 2:750 is taken to prepare the working dilution, or 0.2 ml of whole hemolysin and 74.8 ml of physiological solution are taken.

Preparation of hemolytic system (hemosystem).

A working dilution of hemolysin is mixed with an equal amount of 2% ram erythrocytes. Before using the hemosystem, erythrocytes are kept in a thermostat for 30 minutes to sensitize them.

Preparation of 2% suspension of ram erythrocytes.

98 ml of physiological solution is added to the sediment of 2 ml of washed erythrocytes.

Complement titration

On the day of the main experiment, complement is titrated according to the indicated scheme in the hemolytic system (Table 20).

The complement titer is called complement hemolysis until the minimum amount of complement in erythrocytes is full. Complements with a titer of not less than 2.5% are used to determine the type of protein disease.

Table 20. Complement titration scheme

Components, ml	**Good complement retention, %**								
	0.5	**1.0**	**1.5**	**2.0**	**2.5**	**3.0**	**3.5**	**4.0**	**4.5**
1:20 diluted complement	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45
0.5 ml of insufficient physiological solution	0.45	0.4	0.35	0.3	0.25	0.2	0.15	0.1	0.05
Hemolytic	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0

system									
Physiological solution (instead of antigen and serum)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
37-38 °C	kept in a water bath for 15 minutes								
Titration result	++++	+++	++	+	-	-	-		

1% excess complement, or 2 conditional units of its titer in the hemosystem, is taken to put the basic experiment of KBR.

For example, the titer of complement in the hemosystem is 2%, 3% is taken for the main experiment, 97 ml of physiological solution is added to 3 ml of complete complement to prepare a working volume.

The preparation of diluted complement, determination of its activity and calculation of the working amount require great attention when preparing for KBR.

The working amount of the correctly selected complement is calculated from the conditions of the normal course of the reaction and ensures the accuracy of the result.

An excess of complement causes a specific antigen or antibody to be partially detected or completely lost. Hemolysis of the reaction stops due to the lack of specific antigen and antibody in the absence of complement.

Zardobar. When determining the type of protein virus, the working titer of the positive type-specific protein serum is used. So, if the latest serum titer is listed on the label as 1:40, the working titer is 1:20.

Antigens. Species-specific protein antigens (similar to those produced by specific serum in biofactories) are used in double titers.

In testing antigen reactions, whole (33% mixture) is used diluted 1:2, 1:4, 1:8.

If there is a risk of entry of another type of protein disease in the border zones of our country (Sat-1, Sat-2, Sat-3 and Asia-1), the serum and antigens of the protein virus found in another country are added to the main experiment for type identification and control.

Putting the basic experiment to determine the type of protein disease

In conjunction with the basic experiment, specific protein antigens and sera are controlled according to the scheme. The components are placed in the following order (see table 21 for the main experimental scheme):

l) a specific serum in a working titer of 0.2 ml for each serum - a series of test tubes vertically;

2) specific antigens in working titer from 0.2 ml, seven rows horizontally, one row for each antigen;

3) 0.2 ml of the antigen being tested for each dilution, a series of test tubes, horizontally;

4) physiological solution from 0.2 ml, on the last horizontal line (serum control) in place of antigen and in the last vertical line (control of antigens) in place of serum;

5) 0.2 ml of working diluted complement - to all test tubes of the main experiment.

Test tubes are gently shaken and placed in a water bath at 37-38 ^{o} C for 20 minutes;

6) 0.4 ml of hemolytic system is poured into all test tubes. After the test tubes are shaken again, they are placed in a 37-38 ^{o} C water bath for 30 minutes.

After 5-10 minutes in a water bath, the results of the reaction are calculated, and after 10-12 hours, the latest result is obtained.

The degree of cessation of hemolysis is evaluated by crosses:

(++++) - 100% stop hemolysis; (+++) - 75% stop hemolysis; (++) - 50% stop hemolysis; (+) - 25% stop hemolysis; (-) - complete hemolysis.

If the tested antigen is homologous to the specific antibody, hemolysis stops and the reaction is considered positive; if there is no hemological antigen, complete hemolysis is observed and the reaction is considered negative.

Table 21. Scheme of the main experiment in determining the type of protein virus

Antigens	**Working titer of antigens**	**Serums**							**Control of antigens**
		Atitr 1:30	**0 Titre 1:40**	**C Titre 1:20**	**Asia-1 Titre 1:30**	**Sat-1 Titre 1:30**	**Sat-2 Titre 1:20**	**Sat-3 Titr 1:30**	
		Working titer of sera							
		1:15	**1:20**	**1:10**	**1:15**	**1:15**	**1:10**	**1:15**	
A, titer 1:4	1:2	++++	-	-	-	-	-	-	-
0, titer 1:6	1:3	-	++++	-	-	-	-	-	-
C, titer 1:8	1:4	-	-	++++	-	-	-	-	
Asia-1, titer 1:8	1:4	-	-	-	++++	-	-	-	-
Sat-1, titer 1:8	1:4	-	-	-	-	++++	-	-	-
Sat-2, titer 1:6	1:3	-	-	-	-	-	++++-	-	-
Sat-3, titer 1:4	1:2	-	-	-	-	-	-	++++	-
	S	++++	-	-	-	-	-	-	-
Trying-gan antigen	1:2	+++	-	-	-	-	-	-	
	1:4	++	-	-	-	-	-	-	-
	1:8	-	-	-	-	-	-	-	-
Serums-control of to do		-	-	-	-	-	-	-	-

Note: All antigens and sera are active and type-specific in terms of results. The antigen being tested belongs to the A-type. Of necessity, after determining the type of protein virus in production, then its variant is determined. For this purpose, KBR is

placed in the same way, and serum and antigens of the appropriate identified variant are used.

However, the most recent titer of the variant sera, the antigen double titer, is used.

If the tested antigen reacts positively with the serum even at high dilution, it means that it belongs to that variant. (Table 22).

Table 22. Estimated result of determining which variant the epizootic strain of the protein virus belongs to in KBR.

Antigens in variate	*Dilution of antigens*	*Sera of the latest titer of the variant*				*Antigen-to control*
		A_{ST}	A_{7}	A_{20}	A_{22}	
A_{ST}, titer 1:4	*1:2*	++++	-	-	-	-
A7, titer 1:8	*1:4*	-	++++	-	-	-
A_{2}o, titer 1:6	*1:3*	-	-	++++	-	-
A22, titer 1:8	*1:4*	-	-	-	++++	-
Test antigen (epizootic)	*S*	++++	++++	++++	++++	—
	1:2	++++	++++	++++	++++	—
	1:4	+++	++++	++++	++++	—
	1:8	++	+	+	++++	—
	1:16 a.m	+	±	±	+++++	—
	1:32	-	-	-	+++	—
Serum control		-	-	-	-	—

Summary. The test strain belongs to variant A_{22}.

If the amount of viral material sent from the farm is insufficient for KBR, it is propagated in cultured cells or in 3-6 day old lactating mice or adult guinea pigs. The tested suspension is injected under the skin of mice, 0.1-0.2 ml into the shoulder area, 0.2-0.5 ml into the skin of guinea pigs and into the pads of the two hind legs. Animals are observed for 5-7 days.

After the mice die, an antigen for KBR is prepared from its body. As a result, canker sores appear on the legs of guinea pigs; those afta walls and its contents are used for KBR. If necessary, 2-3 "blind" passages are conducted.

In the third passage, without degeneration of the cells or death of the mice, the protein virus antigen in the suspensions obtained from them is not detected in KBR, and the tested suspension sample is considered negative.

Retrospective diagnosis of protein disease

Blood serum obtained from animals infected with protein disease or suspected of other vesicular diseases is needed as the test material for detection of antibody to protein virus. Blood serum is collected after the 7th day after symptoms of vesicular disease appear in animals. 5-10 samples from adult animals of each group are sent for examination. In case of inconclusive results in the first test, the same animals are bled again after 7-10 days. Serum is preserved by the general method (penicillin and streptomycin 500 units/ml) or frozen at -20 $^{o\ C.}$ At least 5 ml of serum is taken from each animal for testing and sent in a thermos with ice.

Serum is examined in laboratories using radial immunodiffusion reaction (RIDR) and indirect immunofluorescence (BIFR) reactions.

RIDR. The essence of the reaction is that as a result of joining the agar gel with virus antigens and antibodies, a specific precipitation line is formed. RIDR is species specific.

, an equal volume of serum to be tested diluted (to 50-55 $^{o\ C}$) diluted 1:5, 1:10, 1:20 to 1:320 in dissolved agar (with 2%) is mixed and 4 ml is added to the test glass. is poured.

4-diameter well is made in the solidified agar 7,7 mm, and the wells are filled with standard antigens of the species. Then the product is placed in a humid chamber with a window temperature of 37 $^{o\ C.}$ The results of the reaction are taken into account after 6-7 hours and 18 hours.

In a positive reaction, an opalescent precipitation ring is formed in relation to the homologous pathogen that causes the disease around the pit where the antigen is injected.

After the animals recover from the disease, the antibody titer is more than 1:160.

BIFR. This reaction is based on antibody-specific fluorescence (antigen+antibody complex) in the blood serum of recovered animals, and no fluorescence is observed in the serum complex of vaccinated animals.

The procedure for putting the reaction is as follows.

VNK-21, SHB, ChHB cultured cells infected with any type of protein virus are injected with test serum diluted 1:10 and 1:20, incubated for 30 minutes in a humid chamber at 37 o C, ^{unbound} antibody is washed off, air-dried labeled with rhodamine stained with a working dilution of fluorescent antiserum. Then it is incubated at 37 ^{o}C for 30 minutes in a moist chamber, dried, observed under a fluorescent microscope (objective x 40, eyepiece x 4 or 5).

In a positive reaction, the cytoplasm of the cell emits green or light green light. The reaction is monitored with appropriate controls. If even one specific light emission is found in 5-10 sera sent from the farm, the diagnostic result is considered positive. Thus, the serum being tested is titrated to determine the level of antibody found in it. For this, the test serum is diluted from 1:40 to 1:1280. Pre-infected preparations were treated with each diluted as indicated above.

To determine the titer of antibodies produced after infection, dilution is considered after a positive result in BIFR.

Luminescence of preparations treated with sera diluted 1:10, 1:20, 1:40, protein obtained during acute illness, i.e. 7 days after illness, 1:80 diluted and above, serum obtained from a convalescent animal means that

A protocol is drawn up on the results of the investigation in protein disease, and it describes the day of the investigation, the name of the farm, the type of material, brief epizootological information, the name of the components used in the investigation, and the control method.

Assignment.

1. To introduce students to the methods of diagnosis of protein disease in the laboratory.

2. Antigen preparation for KBR from pathological material (a piece of calf skin) taken from an animal suspected of protein disease.

3. Put the main experiment of KBR to determine which type of protein virus it belongs to.

4. Calculation and completion of reaction results.

Material supply:

The examined pathological material (a piece of skin from the skin in a vial with 50% glycerin solution); standard type antigens (A, 0, Ctypes); standard (A, 0, Ctype) sera; hemolysin; complement; 2% mixture of ram erythrocytes; isotonic solution - 0.85% NaCl solution; tripods; test tubes; 1,2 and 5 ml pipettes; water bath; subject tables; a container with a disinfectant solution (2% - NaOH solution); sterile mortar made of porcelain; crushed sterile glass; cuvettes; Petri dish; 10x10 filter paper; tweezers; scissors.

Review questions.

l. What are the main characteristics of the protein virus do you know?

2. Describe the rules of working with the material that preserves the protein virus.

3. What are the epizootological features and symptoms of the disease caused by the protein virus?

4. What methods do you know of making a diagnosis of protein disease in the laboratory?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory training : Tuberculosis to identify the disease

The purpose of the training: to master the rules of obtaining litter material, placing it and sending it to the laboratory, methods of processing (preparation) of litter material for bacteriological examination. To study the methods of inspection of the given patmaterial.

Content of Das: **Tuberculosis (tuberculosis)** is a chronic infectious disease of domestic and wild animals, including poultry and humans, characterized by the formation of specific nodules (tubercles) in various organs and tissues. The trigger was discovered in 1882 by R. Koch. Currently, 5 types of tuberculosis causative agents are known.

1. In humans - *Mycobacterium tuberculosis*
2 **.** In cattle - *M. bovis*
3. In poultry - *M. avium*
4. In mice- *M. microt (murium)*
5. In cold-blooded animals - *M. poycilothermorum.*

farm animals *and humans - Mycobacterium tuberculosis, M. bovis , M. avium* species are important. Tuberculosis is mainly chronic. In order to identify the sick animal in time, it is tested with tuberculin in an allergic way.

Pathological material: Samples of nasal discharge, sputum, tracheal mucus, dung, and urine are taken *from sick animals . Fragments of damaged* organs, bronchial, throat, upper udder, prescapular lymph nodes are taken from the dead bird. The body of the dead bird is sent. It is necessary to observe the rules of asepsis, personal prevention, and equipment safety when taking patmaterial. The sample is sent to the laboratory as soon as it is received. If this is not possible, it is sent in 30-40% glycerin or frozen.

1 . **Microscopy:** The causative agent belongs to the group of acid-alcohol-alkali-resistant bacteria. Its shell contains steoric acids and waxy substances. These substances do not transfer water, dyes, acid and alkali solutions into the cell. That is why tuberculosis bacteria hardly accept the paint. It is specially stained by Sil-Nielsen method.

1. Special filter paper is applied to the fixed smear, Sil fuchsi with carbol is poured over it. The alcohol is heated in the flame of the lamp until steam is formed and it stands on the bridge for 5-7 minutes.
2. Remove the filter paper and pour a 3-5% solution of sulfuric acid on it. 5-7 seconds.
3. Wash well with water.
4. An additional Leffler is stained with methylene blue for 4-5 minutes.
5. The smear is washed with water and dried on filter paper.

Under the microscope, acid-resistant bacteria are red, and acid-resistant ones are blue. Gram-positive, rod-shaped bacteria with a length of 1.5-5 μm and a diameter of 0.3-0.6 μm can be seen in the Gram-stained smear. *M. tuberculosis* - a thin, slightly curved rod, *M. bovis* - short, thick: *M. avium* **is** small compared to others, polymorphous. They are placed one by one, in groups. It is inactive, does not form spores and capsules. A long stringy form is also found in the smear made from culture.

2 . **Bacteriology:** First, pat material is treated in one of Gon or Alikayev methods.

Gon's method : The material is crushed in a sterile mortar and mixed with a solution of 10-12% sulfuric acid in water in a ratio of 1:4. Exposure (effect of acid) should not exceed 20-30 minutes. Smears are prepared from the sediment, planted in a nutrient medium. For bioassay, the sediment is washed 1-2 times with sterile physiological solution.

Alikayev's method : It is often used when the bedding material is new and less contaminated. The material is crushed in a sterile mortar to a size of 0.5 cm^3, and a solution of 10-8-6% sulfuric acid in water is poured over it. It takes 10-20 minutes. The exposure time and concentration of the acid depends on the degree of contamination of the material. After 10-20 minutes, the acid is drained, instead of it, a physiological solution is poured and it stands for 8 minutes. Then the physiological solution is poured, the material is crushed in a mortar, a suspension is prepared in the physiological solution, 5-6 test tubes are planted in the nutrient medium.

Treated litter is planted in selective nutrient media: egg-starch, clay-glycerin-broth - mediums that inhibit the growth of foreign microorganisms. Petranyani, Levenstein-Jensen, Gelberg media are often used. GPB and GPA with glycerin are also used.

The causative agent of tuberculosis is aerobic, grows slowly (2-4 weeks or more). When grown in glycerin broth for a long time (6-8 weeks), the toxic substance **tuberculin** accumulates. It is used to detect tuberculosis. In a liquid environment, the pathogen forms a growing film after 10-30 days.

M.tuberculosis - thick film, *M. bovis* - black reticulated tumor veil, *M.avium* - on the 7-10th day, it forms a thin, delicate, more fluid, strongly wrinkled membrane on the 21st day. In dense nutrient media, microcolonies appear, which are barely visible at first, and then grow in size. 1-2 colonies are small or large, shiny or opaque, smooth or wide from the surface of the nutrient medium, or the colonies merge and form a single fluid layer with the surface. Bacteriological examination period - 2 months. See the seedlings every 4-5 days and record the results.

3. Biotest . 1 ml is injected under the skin of guinea pigs, 2 ml into the ear vein of rabbits, 1-2 ml into the vein under the wing of chickens. The observation period is 3 months.

Biotesting animals should be tested for tuberculosis in advance with tuberculin by allergic method. Only those with negative results are used for bioassay. The dead animal is split open, swabs are prepared from the characteristic tubercles, and planted in a nutrient medium.

Differentiation of triggers (typing).

M. bovis **culture** causes generalized tuberculosis in guinea pigs and rabbits.

M.tuberculosis - generalized in guinea pigs, and local process in the lungs in rabbits.

M.avium - causes a septic process in rabbits, kills. Sometimes a local process appears. In guinea pigs, only a local process (abscess at the place where the culture is sent) occurs.

3 tests are performed to distinguish low virulence culture from acid-resistant saprophytes: 1. catalase activity - formation of gas bubbles with 50% perhydrol solution is measured and determined in mm. This feature is higher in saprophytes. 2. Formamidase activity - a blue ring appears in a culture tube treated with formamide solution and several chemicals. This condition is manifested only in saprophytes. 3. Susceptibility to drugs-tuberculostat preparations (streptomycin, ftivazid, PASK, etc.) are studied in the nutrient medium.

M.tuberculosis and M.bovis are sensitive to them, while saprophytes and *M.avium* are resistant.

Biopreparations: BSG vaccine-dried live culture of M. Bovis vaccine strain.

Purified, dry PPD tuberculin, for mammals.

Alttuberculin, for mammals.

PPD tuberculin for poultry.

Control questions:

1. Obtaining patmaterial for testing for tuberculosis?
2. Tell me the methods of testing in the laboratory?
3. Morphological-tinctorial characteristics of the stimulus?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory study : Brucellosis to identify the disease

The purpose of the training: To get patmaterial and study the rule of sending to the laboratory. Mastering the examination of patmaterial for brucellosis by bacteriological, serological methods.

of brucellosis was isolated for the first time in 1886 by an English microbiologist, David Bruce, from the spleen of a person and called it *Micrcoccus melitensis* .

Brucellosis is a chronic infectious disease in animals and humans. The disease is usually asymptomatic, sometimes clinical symptoms such as abortion, bursitis, orchitis, epididymitis, endometritis appear.

At the beginning of brucellosis enzootic disease, gross abortion in animals, rapid separation of the placenta, endometritis, and infertility are manifested as a result. In most cases, it passes without clinical symptoms.

The migration of Brucella from one type of animal to another has important epizootological and epidemiological significance. For example *Br. melitensis* was found in cattle and pigs, so such animals become a source of human brucellosis

(Ye.V. Kozlovskii, 1954-1956, etc.). It was also found that *Br. suis* migrates to cattle and sheep, goats, and *Br. abortus migrates to sheep, goats and pigs.*

Humans can be infected by all types of Brucella germs, but sheep-goat Brucella is highly contagious to humans and the disease is severe.

The causative agent belongs to the genus *Brucella and consists of 6 species:*

1. *melitensis* (in sheep and goats)
2. *abortus* (in cattle)
3. *suis* (in pigs)
4. *ovis* (in rams)
5. *canis* (in dogs)
6. *neotoma* (in rats)

Brucella ovis causes infectious epididymitis in pigs.

Endurance . Brucella is resistant to the effects of the external environment. It lives 3-4 months in wet soil, water, 160 days at low temperature in cow milk, 1.5-5 months in sheep's wool, 2.5 hours in direct sunlight. Lives 8 days in milk, 45 days in brinza and cheese, 60 days in fat, 20 days in cold meat. Milk dies in 30 minutes when heated to 70 º C, and in 1-2 minutes when boiled. Milk 70^0Cis pasteurized for 30 minutes at 85°C or 20 seconds at 85 °C. 90^0C2% caustic soda, 20% freshly slaked lime, 2% formaldehyde, 4% creolin, etc. are used for disinfection.

Pathological material. *From a sick animal* - an abandoned fetus, fetal stomach with a fetal membrane or parts of a fetus with both sides tied: hygroma material, milk - after washing and disinfecting the udder in 70° alcohol, then from each teat into separate sterile test tubes 10-15ml is taken from the last portions). From sheep and goats, milk is taken sterilely from the udder with a syringe needle. The milk should be checked on the day of sampling. If it is not possible, it is preserved with boric acid in the amount of 0.1 g per 10 ml of milk **.**

It is taken from rams (at slaughter) with a sperm sac. A piece of feather material taken from each animal is individually wrapped in cellophane, parchment paper and placed in a waterproof container (polyethylene bag, box, jar). It is necessary to check the blood of pregnant animals (one week after pregnancy). The material comes complete with a referral to the laboratory.

1. Microscopy. Two smears are prepared from the pad material and stained by Gram and Kozlovsky methods. Brucella are small, rod-shaped or cocci-shaped bacteria, 0.6-1.5 μm in length, 0.3-0.5 μm in diameter, gram-negative, non-motile, non-spore-forming, single, double or clustered on smear. lib will be located. In smears stained by the Kozolsky method, Brucella are red, and other microbes are green.

2. Bacteriology. Brucella grows in special media: meat-peptone liver broth (GPJB), liver-glucose-glycerin agar (JGGA) and broth (JGGB), erythritol-agar, serum-dextrose agar, etc. on agar, two test tubes of broth from the stomach, five test tubes are planted on agar.

Cultivated media from ram's feather material is grown in an atmosphere with 10-15% carbon dioxide.

Half of the litter material seedlings obtained from cattle are grown with 10-15% carbon dioxide, and the rest are grown in a normal atmosphere.

Seedlings 38°Care grown in a thermostat at 37- for 30 days.

In a dense nutrient medium, small, clear, raised, round, shiny, smooth (*S* -shaped) and bluish (*R* -shaped) colonies are formed. When grown for a long time, the colonies become dull, and with the formation of pigment, they become black and join together.

In a liquid nutrient medium, uniform turbidity, blue formation of a ring is formed, and then less sedimentation occurs.

3. Biotest. First, 350-400 gram guinea pigs take blood from their hearts, and their serum is checked for brucellosis by the AR method. Only if a negative result is obtained in the ratio of 1:5, they can be biotested.

A 1:10 suspension of feather material is injected subcutaneously into the inner thigh of guinea pigs in a dose of 1 ml. On the 15th, 25th, and 40th days, blood is taken from them, and their serum is tested for brucellosis using the AR method in a ratio of 1:10 to 1:80. If a positive result is obtained in ratios of 1:10 and higher, the test result is considered positive, even if the culture is not isolated from the provided paternal material. The biotest is observed for two months. All isolated cultures are destroyed by autoclaving in an autoclave at 1.5 RPM for 1 hour at the end of the work.

From serological testing methods, AR, KBR, UKBR, RBN, milk ring AR are put. AR is placed in 1 ml in 4 proportions. Blood serum of sheep, goats, sheep, dogs is from 1:25 to 1:200 (positive result is 1:50 and higher titer). Y.sh.m, horses, camels from 1:50 to 1:400 (1:100 and higher titer is positive). 1:10 to 1:80 in guinea pigs and fur animals (1:10 and higher titers are positive).

RBN. 0.3 ml of serum is poured into the grooves of special enamel plates. 0.03 ml of Brucellosis antigen stained with pink Bengal is poured over it. Stir gently for 4 minutes. Antigen positive, negative sera, physiological solution are used for control. A positive result shows a pink agglutinate. A positive sera sample will be retested at AR, KBR.

Milk ring reaction. 2-3 ml of freshly expressed milk is poured into the test tube, and 0.2 ml (2 drops) of hemotoxylin-stained antigen is added to it. The test tubes are shaken, mixed well, and kept in a water bath or thermostat at 37ºC for 45-60 minutes. A positive result - a blue ring appears, the milk becomes discolored. In a negative result, the milk remains blue.

Allergic method . An allergic reaction occurs when brucellosis allergens are injected into the skin of animals infected with brucellosis. For cattle and pigs, an allergenic brucellisate VIEV prepared from a non-agglutinogenic strain of *Br.abortus* is used. The appearance of well-defined swelling at the site of allergen injection is considered a positive result of an allergic sample.

Infected cattle are not treated and are sent to meat. Vaccination against the disease is carried out using vaccines in sheep. Active immunization against brucellosis was initiated by Bang in 1906. Sht #19 was isolated in virulent form from cow's milk by Buk in 1923. The virulence of strain 19 was reduced by recultivation

on potato agar for 10 years. Because animals remained seropositive for a long time after the use of the vaccine ShT #19, it became difficult to distinguish animals infected with brucellosis. This prompted our scientists to conduct research to create more perfect live vaccines against brucellosis. As a result, vaccines prepared from sht #82 were put into practice and widely used.

Control questions:

1. Tell me the rules for receiving and sending the material to the laboratory?
2. Principles of laboratory diagnosis of brucellosis?
3. Cultural characteristics of Brussels?
4. Biotest for brucellosis?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory exercise : Methods of rabies diagnosis

Rabies is an acute infectious disease characterized by severe damage to the nervous system and death. Humans and all mammals are susceptible to the disease. Rabies is widespread everywhere. The infectious agent of the disease is spread by dogs, cats, wild rodents and predators, as well as blood-sucking bats (vampires). The duration of the latent period depends on the place of the bite and the force of the bite, the amount and virulence of the virus, and the resistance of the bitten animal. the period can be from 1-3 weeks to a year or even more. The disease is acute. Clinical signs are almost the same in all animals. In dogs, rabies is characterized by aggressiveness and calm (paralysis). Rabies in large horned animals (loss of appetite, atony of the large abdomen, salivation) is not specific. There may be no transformation stage. Pathologoanatomical changes are not specific. Inedible objects can be found in the stomach of carnivorous animals (mainly dogs). Rabies virus has remarkable neuroprobability. Bitten falls from the periphery to the place, moves to the central nervous system through the nerve fibers, spreads in the body through the peripheral nerves to various organs, including the salivary glands. The virus belongs to the Rhabdoviridae family, the Lussavirus genus. the part of the virus is cut off. The virion of the virus has helical symmetry, preserving the RNA, and has a lipoproteid shell. The virus is kept intact at low temperature. 60°CThe temperature kills the virus in 5-10 minutes, and sunlight kills it in 5-7 days. The activity of the virus is reduced by formalin, phenol, 5% hydrochloric acid in 5-10 minutes. The rabies virion contains glycoprotein (outer) and nucleocapsid (inner) antigens. If the glycoprotein antigen virus produces neutralizing antibodies, it produces nucleocapsid-complement binding and precipitating antibodies. Epizootic strains of rabies virus are similar in immunobiological characteristics, but differ from each other in terms of virulence. In

the body, the virus is mainly central stored in the nervous system, salivary gland and saliva. The virus is cultured in mice, rabbits, guinea pigs and other animals, and in primary cultured cells (kidneys of Syrian hamsters, sheep fetuses, calves, etc.) growing cells (VNK-21, KEM-1, etc.). Virus multiplication in cultured cells does not always show CPT. Chicken embryos preadapted to rabies virus are susceptible. The virus is able to form cytoplasmic inclusion bodies. These can often be found in the cells of the ammon horn, cerebrum, and cerebral cortex. The source of the disease is a sick animal. They transmit the virus through bites. Carnivores become infected with rabies and are infected when they eat the brain and spinal cord of a dead animal. It has been proven that rabies can be spread by air where there are bats. The main source of rabies is dogs and cats, foxes, wolves and other wild animals. Diagnosis of rabies is based on epizootological, clinical data and laboratory tests. When working with sick animals and infectious materials, strict personal safety measures should be taken: it is necessary to wear a rubber or polyethylene apron worn over the sleeve, rubber boots, protective glasses, a protective face mask. It is strictly forbidden to open the corpse of animals suspected of rabies in the field.

Getting patmaterial

For rabies examination, a fresh carcass, small animals whole, large and medium-sized animals with the head together with the second and first cervical vertebrae are sent to the laboratory.

Small animal carcasses are treated with insecticides before being sent for examination.

The pathological material is placed in a plastic bag. Then, a gasket treated with a moisture-absorbing disinfectant is placed inside the tightly closed box. In the material and referral letter, the sender and his address, the type of animal, the anamnesis data proving suspicion of rabies, the signature and date of the veterinary officer are sent through a special person.

Laboratory diagnostics.

It consists of antigen detection using IFR and DPR, visualization of Babesh-Negri bodies, and biological sampling in mice.

IFR produced fluorescent antirabies gammaglobulin for this reaction.

The method of putting IFR. Smears are prepared from the left and right sides of the brain (ammon horn, hemispheric cortex and medulla) on a degreased, glass slide or thin stamp.

At least two preparations are prepared from each part of the brain.

Spinal cord, submandibular salivary glands can also be checked. For control, preparations are prepared from the brain of a healthy animal (white mouse).

The preparations are air-dried and 20°Cfixed in cooled (minus 15-) acetone for 4 hours to 12 hours, air-dried, then fluorescent gamma-globulin is dropped and 37°Cplaced in a wet chamber for 25-30 minutes, then with physiological solution or pH 7, 4, washed with phosphate buffered saline, air-dried, blotted with non-fluorescent immersion oil, and viewed under a fluorescent microscope. In the granules of the drug containing the rabies virus antigen, in most cases, a yellow-green fluorescent color can be found outside the cells of various sizes. (See Figure 63).

In the control, almost without such light emission, the nerve cell emits a dull gray or green color. The speed of light scattering is estimated by crests. Absence of specific fluorescent emission indicates a negative result.

Pathological material obtained from animals vaccinated against rabies cannot be examined by IFR for three months, because the antigen of the vaccine virus can also fluoresce. Tissues preserved with glycerin, formalin, alcohol, etc., and with some signs of decay, cannot be examined by IFR.

DPR in agar gel.

This method is based on the properties of antigen and antibodies, and after the antigen and antibody are soaked in agar gel and mixed (antigen complex + antibody), they meet and form a visible precipitation line. It is used to find the antigen in the brain of an animal that has died due to the street virus of rabies or died from an experimental infection (biological sample).

When performing the reaction, a glass is used and 2.5-3 ml of 1.5% dissolved agar is poured onto it. After the agar hardens, 4-5mm holes are made on the stencil. If the columns are removed using a learning pen. The wells in the agar are filled with components arranged according to the scheme (Fig. 65).

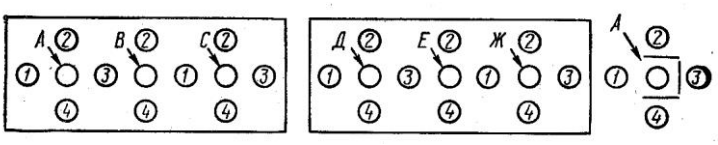


Fig. 65. Image of DPR in the diagnosis of rabies.

A-cerebral cortex (left hemisphere); B-cerebral cortex (right hemisphere); C-ammon horn (left); D-ammonium horn (right); E-brain; G-long brain; Dilution of 1,2,3,4,-globulin, 1:2,1:4,1:8,1:16.

On the right is a positive view of -1:4,1:8,1:16-PR diluted globulin.

In large animals, all parts of the brain (left and right hemispheres) are examined, in medium-sized animals (rat, hamster, etc.), any three parts of the brain are examined, and in mice, the entire brain is examined.

After preparing a paste-like mass from the brain with the help of tweezers, it is poured into the appropriate cavities. Controls with negative and positive antigens are placed separately on the same stencil in the window.

Agar gel: agar Difko agar-15g, sodium chloride-8.5g, 1% solution of methyl orange in 50% ethyl alcohol-10ml, merthiolate-0.01g, distilled water-1000ml.

After the wells are filled with components, the preparations are placed in a humid chamber, or 37°Cplaced in a thermostat for 6 hours, and then at room temperature for 18 hours. The calculation of the results is carried out after 48 hours. The formation of 2-3 precipitation lines around the wells between the brain suspension and antirabies gamma globulin indicates a positive reaction.

Bacterial sterility or rotting of the brain cannot be a barrier to DPR placement. Materials preserved with glycerin, formalin or other means are not suitable for DPR.

Finding Babesh-Negri corpuscles.

At least two of each section of the brain are prepared on a slide or stained with Sellers, Muromsev, Mann, Lens and other methods.

For example, Sellers staining: a fresh, unhardened preparation is kept for 10-30s after being coated with dye and washed with a phosphate-buffered solution (pH 7.0-7.5) in a dark place at room temperature dried in a vertical position, then viewed under a microscope using immersion oil.

A positive result is indicated by the appearance of sharply delineated oval or elongated granulated pale-red Babesh-Negri bodies located in or outside the cytoplasm of the cell . This method has the diagnostic value of identifying species-specific entries.

Biological sample.

More effective than all the methods mentioned above.

This method is tested when a negative result is obtained in the above methods and when it is suspected. White mice weighing 16-20 g are selected for the biological sample. The brain is removed from all parts of the nervous tissue and crushed in a mortar filled with sterile sand, then saline is added to make a 10% solution. After cooling for 30-40 minutes, the liquid on the sediment is removed and infected with mice.

If it is suspected to be contaminated with a bacterial infection, 500 units of penicillin or streptomycin are added to 1 ml of the suspension and kept at room temperature for 30-40 minutes. For a biological sample, 10-12 mice are infected: 0.03 ml intracerebral to half, and 0.1-0.2 ml to the other half under the skin of the nose or upper lip.

Mice infected with the virus are placed in glass jars (preferably in an aquarium) and observed for 30 days, recording the changes that occur.

Mice dying at the end of 48 hours were not considered disease-specific and were not taken into account in the outcome evaluation.

If the pathological material contains the rabies virus, the following symptoms appear within 7-10 days after infection of mice: fur loss, a characteristic hunching of the shoulders, impaired coordination of movement, paralysis of the hind legs, then the front legs, later death is observed.

Brains of dead mice are examined by IFR and DPR to detect Babesh-Negri corpuscles (Fig. 65a).

A positive diagnosis is biological testing if an antigen is detected by the IFR, DPR method due to the presence of Babesh-Negri bodies in preparations prepared from the brains of virus-infected mice. A negative diagnosis is considered if there is no death in mice for 30 days.

For biological testing, the method of tomorrow's diagnosis (when the tested animal bites the examiner) uses 20-30 mice instead of 10-12 mice per infection, and the brains of 1-2 mice are examined daily using IFR. This (in a positive case) shortens the inspection period by a few days.

A unique biological test method is used in laboratory practice.

Its essence lies in the fact that a suspension prepared from the brain tissue of an animal with rabies is infected with mice. If the brain tissue is pre 37°C-treated with anti-rabies serum for -10 minutes, the mice do not become infected.

Some investigators have suggested making a diagnosis by immunofluorescence by making a smear from the cornea of an animal suspected of having rabies while it is still alive.

But the efficiency of this method is not high.

In almost all laboratories, the examination is carried out in turn as follows:

For IFR, a smear-mark is prepared from the brain, a DPR is applied, and a biological sample is taken if the result is negative.

When IFR is performed with high proficiency, a result that corresponds to biological testing in 99-100% is obtained. In rabies, Babesh-Negri bodies can be detected in 65-85%, with DPR in 45-70%.

Review questions.

1. Describe the main characteristics of the rabies virus.
2. Talk about the epizootological features and symptoms of the disease caused by the rabies virus.
3. What methods of laboratory diagnosis of rabies do you know?
4. What kind of material is taken from animals suspected of rabies and what are the working rules.

Laboratory training : methods of determining pasteurellosis .

Purpose: To teach students how to organize prevention and countermeasures against pasteurellosis disease in farm animals and how to diagnose the disease.

Materials and equipment

Place and content of training. In the laboratory of the department, the teacher explains the subject material to the students for 5-10 minutes and asks questions. Then he explains the main features of diagnosing the disease.

Pasteurellosis - hemorrhagic septicemia, fowl plague, (lat. Ing. – pasteurellosis , moisture. french pasteurellosis) is characterized by acute septic and hemorrhagic inflammation of the mucous membrane of the respiratory tract and intestines. The disease infects all mammals, birds and humans.

The causative agent - Pasteurella multocida - is an inactive, gram-negative, aerobic bacterium that does not produce spores or spores. Stains well with Romanovsky - gimza and methyl blue. Grows well in normal artificial environments.

Making a diagnosis of pasteurellosis causes certain difficulties. Therefore, the following information is taken into account.

1) Firstly, in many cases, the disease of pasteurellosis is mixed with viral infections, and the clinical symptoms of the main disease disappear, as a result,

clinical symptoms that are not characteristic of the disease may appear , and as a result, the diagnosis becomes difficult. That is why bacteriological methods in the laboratory are the best factor and facilitate the solution of the problem. That is, it is necessary to make a complex diagnosis here .

2) Epizootological inspection of the farm is carried out on the basis of a generally accepted plan. Thorough As a result of epizootological analysis, many infectious diseases are suspected or, on the contrary, excluded. Thus, verification work is carried out in the desired direction.

3) When the disease of pasteurellosis is acute, the clinical appearance is sufficient and characteristic in all types of animals, and it helps in determining the diagnosis.

4) Pathologoanatomic changes also provide valuable information about the nature of the disease.

5) Bacteriological and virological tests complement the general information and help in making a final diagnosis of the disease .

6) For laboratory tests, blood, tubular bone, blood smear is prepared from the heart of a dead animal, and a piece of parenchymatous organs is removed.

Pathological materials should be sent no later than 3-5 hours after the death of the animal.

DIFFERENTIAL diagnosis

Pasteurellosis disease is distinguished from anthrax, piroplasmidoses and blackheads in large horned animals.

In young animals, staphylococcal and streptococcal infections, salmonellosis, colibacteriosis and viral respiratory infections are also taken into account. It is also separated from PG-2, IRT, etc.

In pigs - from anthrax, piroplasmosis, clostridiosis and streptococcal infections.

In poultry, it differs from Newcastle disease, spirochitosis, mycoplasmosis and infectious laryngotracheitis.

Measures to improve the health of unhealthy holdings (farms) with regard to HONEY

Sanitation measures are developed taking into account the results of the epizootological examination of the farm, the type of injured animals and their housing conditions. Using highly effective biological drugs and antibiotics, it is possible to quickly eliminate the focus of the disease.

There are the following guidelines and methodological guidelines for pasteurellosis:

1. AzNIVI semi-diluted formalin-hydro-oxide against pasteurellosis in large horned cattle, buffaloes Instructions for use of aluminum vaccine. VK 1 floor. 514 pages.
2. Instructions for the use of concentrated semi-valent formaldehyde vaccine against swine paratyphoid, pasteurellosis and diplococcal septicemia diseases. VK 1 floor. 550 pages.
3. Guidelines for the use of an emulsified vaccine against pasteurellosis in cattle, buffaloes and sheep. 1 t 542 pages.

4. Instructions for the use of precipitated formal vaccine against hemorrhagic septicemia (pasteurellosis) of cattle, sheep and pigs. VK 1 floor. 543 pages.

K immune preparations and antibiotics .

1. Interim guidance on the use of norsulfazole sodium against avian pasteurellosis. VK 1 floor. 778 pages
2. Instructions for the use of terramycin against pasteurellosis in cattle . VK 1 volume 679 pages.
3. Guidelines on measures to combat the disease of poultry pasteurellosis. VK Sh tom 94 pages.
4. Instructions for use of dry vaccine (first and second) prepared from Krasnodar NIVS strain "AV" and "K" against waterfowl pasteurellosis. VK Sh tom 198 pages.

Serums

1. Guidelines for the use of serum against pasteurellosis in cattle, buffalo, sheep and pigs. VK Sh roof. 215 pages.

Control questions.

1. How to identify pasteurellosis?
2. Making a final diagnosis of the disease.
3. Creating treatment plans for pasteurellosis in an unhealthy farm.

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent, 2021.
2. Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory training : Smallpox diagnosis , prevention and countermeasures .

Purpose: To learn to make a correct diagnosis of the disease and eliminate it when it appears, as well as to make a plan of activities for the health of the farm using the guidelines.

of training equipment : slides, pictures, tables, manuals and biological drugs. Complex methods of disease detection. The main methods of smallpox detection are epizootological inspection of the farm and study of clinical signs of the disease.

Basic concept

E piezootological method. During the epizootological examination of the farm, it is taken into account that the smallpox disease has its own characteristics in different animals. For example, in cattle pox, it is taken into account that people have been vaccinated against smallpox.

CLINICAL method . Explanation of the clinical signs in smallpox are very characteristic and therefore there is no doubt about the diagnosis (five stages). Laboratory testing methods are used in doubtful cases. For this, smears are examined

under a microscope, virological examination or biotest. A biotest is performed on rabbits with litter material obtained from cattle , two piglets in pigs, sheep and goats. Prepare a suspension (1:10) of virus-preserving material and inject 0.2 ml to rabbits and 0.5 ml to large animals.

DIFFERENTIAL diagnosis

Cattle from protein, vesicular stomatitis and feed rash, contagious ecthyma of sheep, scabies, itching and non-infectious eczema:

It differs from protein, vesicular disease of pigs, avitaminosis, hypocalcemia and hyperkeratosis, salmonellosis, flu, vesicular exanthema, streptococcosis, infectious laryngotracheitis in poultry, mycoplasmosis, avitaminosis A, candidamycosis.

REGARDING MEASURES AGAINST SMALLPOX DISEASE EXCERPT FROM MANUAL

Guidelines for smallpox control measures are specified for sheep and pigs only.

Measures for the prevention of sheep pox.

For the prevention of sheep pox disease, all sheep in farms and settlements are vaccinated against smallpox.

Farms and settlements are cured of diseases that were previously unhealthy, and after 3 years, sheep on farms are also vaccinated with a vaccine.

Heads of farms, owners of sheep are prohibited from bringing sheep, fodder and tools from unhealthy farms into farms, farms, and settlements.

Sheep brought to the farm are kept in preventive quarantine for 30 days. Pastures, watering places, livestock buildings should be in good veterinary and sanitary condition. Persons who provide permanent service to horses, pasture plots, watering places, and driving paths are closed. It is necessary to ensure systematic monitoring of sheep condition.

MEASURES TO BE CARRIED OUT WHEN YOU SUSPECT SMALLPOX DISEASE

If smallpox is suspected, the head of the farm, the managers of the farm should immediately inform the veterinarian and take measures to prevent the spread of the disease until the arrival of the veterinarian.

After arriving at the farm, the veterinarian should conduct a clinical examination of the animals suspected of having a disease and measure their body temperature, sick and high temperature animals are transferred to the unhealthy group.

Severely infected sheep are killed immediately. A sample is taken and sent to the laboratory for examination. Veterinary-sanitary measures and disinfection are carried out in order to detect the epizootic situation and prevent the spread of the disease.

The chief veterinarian of the region is informed about the occurrence of the disease and the measures taken.

Measures for the elimination of sheep pox in unhealthy areas.

Nokhia Chief Veterinarian should immediately take measures to determine the diagnosis after hearing the news about sheep pox. If the diagnosis is confirmed, the

farm (settlement) will be declared unhealthy due to smallpox and quarantined after submitting the documents to the governor.

Pets are prohibited during quarantine. Removing and bringing animals from the farm, replacing animals in the farm, feeding, watering and keeping sick animals with healthy animals. Removal of fodder from an unhealthy farm, use of sheep's milk and products derived from it without disinfection. Milk from sheep is pasteurized at a temperature of 85 g at 0 C for 30 minutes or boiled for 5 minutes and is used on the farm itself, shearing of the wool of the hands in the flock unhealthy due to smallpox, sale of livestock products and animals, exhibitions , fairs, markets and other events are strictly prohibited.

All hands on the farm are considered and must undergo a veterinary examination 1 time in 10 days. Sick animals are isolated and treated with symptomatic means.

Sheep that die with clinical signs are lost. The use of skins and wool is prohibited. All clinical milking is vaccinated against smallpox. The vaccinated sheep were under the supervision of a veterinarian for 14 days. After each lambing and carcass collection, as well as after vaccination of sheep, sheds and other areas are mechanically cleaned and disinfected.

The following disinfectants are used to disinfect buildings and places where animals are kept. Chlorinated lime or sodium hypochlorite with 2% lysol chloride boron, 2% formaldehyde solution. Manures are neutralized by biothermal method for 3 weeks. If smallpox disease is detected in farms that have not been registered for 3 years or more, according to the recommendation of the state veterinary inspector, all sheep slaughterhouses are subject to vet sanitary rules under the supervision of the farm's chief veterinarian. will cry.

When the quarantine farm is completely cured, the last animal dies or is slaughtered, the final disinfection is carried out 20 days later.

Although the clinic of smallpox in animals does not have the same appearance, it is very similar to each other.

Damage can be found on the mucous membranes and skin, in the more hairless, hairless areas of the body together with the generalization process.

It consists in the appearance (redness) of a certain type of roseola on the skin and mucous membranes, followed by the transition to a papule and the formation of vesicles (blisters). Then it becomes a pustule, and as a result of their rupture, liquid flow is observed, and a hardened crust (scrub) is formed on the wounds.

After the wounds heal, the crust falls off and scars remain. Due to the delay in the generalization process in smallpox, there is an increase in temperature, pallor, loss of appetite, swelling of the subcutaneous tissue, and in poultry, a diphtheroid layer appears in the respiratory tract. Smallpox viruses produce virions, 260x390 NM in size. They are the largest among the virions of other viruses.

They can be seen not only under an electron microscope, but also under a light microscope. Smallpox is easily transmitted from sick animals to healthy animals, and it is easy to infect experimental animals.

Some smallpox viruses multiply in the chorionallantois membrane of the chicken fetus and form smallpox (their form is not the same for different viruses).

Smallpox viruses multiply in primary cultured cells of a species-specific animal and exhibit SPT.

As with other viruses, antigens of smallpox virus in various materials can be found using FAU and DPR.

It is not difficult to make a diagnosis taking into account the clinical and epizootological data. However, it is recommended to confirm the clinical epizootological diagnosis based on laboratory examination data.

Among the laboratory methods, viroscopy is considered universal - it is possible to find smallpox virus virion in the material taken from a sick animal using a light microscope. For this, smears are made on a piece of glass from a piece of skin or a mucous membrane infected with smallpox (preferably in the stage of papules or vesicles). Smears are painted by various methods, the best of which is MAMorozov's method of silvering. When painting according to MAMorozov, 3 reagents are prepared.

Nl Reagent (Ruge liquid): Pour 1 ml of glacial acetic acid, 2 ml of 40% formaldehyde solution (commercial formalin) and 100 ml of distilled water into a container.

N2 Reagent (chemical substance): After dissolving 5g of tannin in 100ml of distilled water, 1ml of liquid carbolic acid (phenol) is added. Crystalline carbolic acid 56°Cis dissolved in a water bath to make it liquid. A good grade of tannin should be used. To find out the purity of lg tannin, it is dissolved in 5 ml of water and 10 ml of 96° alcohol is added to it. Cloudiness should not appear for an hour (dextrin, camel). Then add 5ml of ether (sugar, salt) to avoid clouding.

Reagent (ammonia silver solution): 5 g of crystalline silver nitrate is dissolved in 1 ml of distilled water. A small amount (one tenth) is poured into another container from the general solution . Ammonia solution (25%) is added dropwise to the remaining silver nitral solution. A dense brown precipitate forms at the beginning, which then dissolves due to the addition of ammonia.

If the solution is completely shiny, the stock solution is poured off before it becomes cloudy, and the resulting silver nitrate solution is added dropwise.

A stock solution of ammonia is added dropwise followed by a solution of silver nitrate until a slight turbidity is formed. The resulting cloudy solution is diluted 1:10 in distilled water and used for staining preparations. The solution is very stable and should be stored in a tightly closed glass container in a dark room.

Painting according to the MAMorozov method.

The drug is treated with reagent 1 for 3-5 minutes, then washed with distilled water;

It is immersed in reagent 2 and then heated for 1-2 minutes until steam is formed; It is heated and treated with the 3rd reagent for 1-2 minutes until a black-brown color appears, then it is thoroughly washed in distilled water; air-dried and viewed in the immersion system of the microscope. Result: a small black-brown, rounded oval-shaped, accumulating, row or diffuse mass or non-single cells can be found on a yellow background. These cells are the virions of the smallpox virus. (Fig. 66)

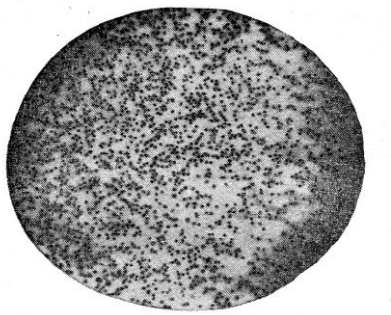


Figure 66. Viroscopy. Smallpox virus virions stained by the Morozov method (according to SSMarennikova).

An enameled cuvette (18x24) is convenient for staining, and 2 1-2 ml pipettes "bridge-like" are placed on its edges and fitted with a rubber band.

There should be a separate pipette for each reagent. The advantages of the viroscopy method are quick response, simple and easy to perform. The disadvantages of this method are that it gives an accurate result when examining vesicular fluid, but the result of the reaction decreases significantly when examining pustules and hardened crusts; har in the smallpox group

it will be impossible to distinguish different viruses; it is impossible to clearly distinguish virions from cellular elements (in any case, the level of the examiner should be much higher).

Biological sample. It is the second method that is widely used in the diagnosis of smallpox. In smallpox, biological sampling is always done in susceptible animals, in primary cultured cells prepared from that animal.

Rabbits are sensitive to mumps virus, cows, grass pox virus, and chicken fetuses are not only sensitive to fowl pox virus, but also to mumps vaccine, black cattle pox, pigeon pox. All smallpox viruses are dermatropes, and experimental infection is carried out intradermally, through scratched skin, by inoculation of chicken fetuses with XAP (the method of infection of animals and chicken fetuses is recorded in the relevant topics (Fig. 67).

The method of infecting the scratched skin of a rooster is easy, it is done with a needle or a broken Pasteur pipette, and the crown is scratched not very deep (blood should not come out. The virus suspension is applied to this place using a cotton swab or a toothbrush.

The chicken pox virus can easily be transmitted to the feather follicles of the rooster. To do this, the feather on the cock's thigh is plucked and the virus suspension is immediately applied to the open follicles using a tampon or brush.

If the examined material contains the virus of smallpox, 5-7 days after infection with the virus, smallpox-like folliculitis appears on the crown (Fig. 68), and smallpox-like folliculitis appears on the thigh (Fig. 69).

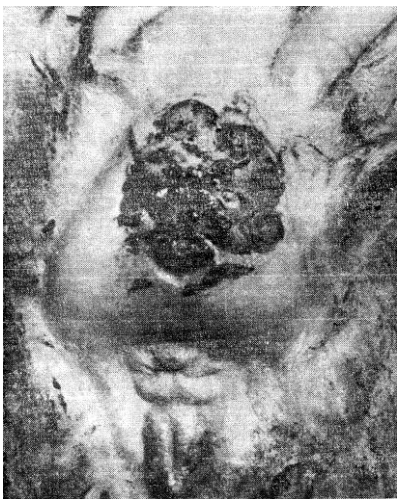


Figure 67. Biological testing of sheep pox virus by infecting the dung layer.

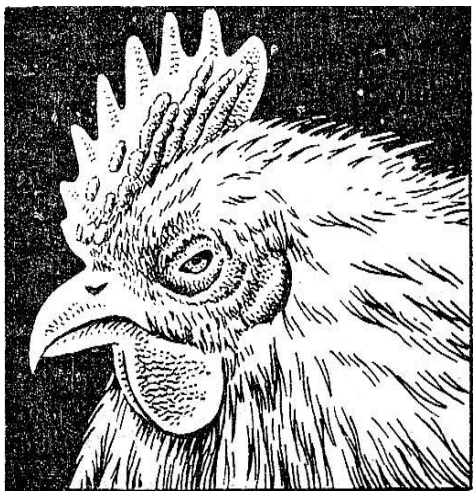


Figure 68. Damage to the cock's crown in smallpox.

It is not difficult to find virions in smallpox lesions or follicles in smears by viroscopy.

When infecting rabbits, the virus is applied to the scratched area after the skin is free of wool, which means it is no different from infecting the scratched area of a rooster.

The methods of FAU and DPR in smallpox are almost similar to those written in other subjects.

Material supply:

Smallpox virus and sensitive living objects (cock, pigeon, rabbit); sterilized toothbrush; sterile cotton swab; skimmed glass; sharp blade; tweezers; 1,2,3 reagents; immersion oil; illumination microscopes; spirit lamp; distilled water.

Approximate lesson plan (2 **hours)**

l. 5-7 days before the training, with the participation of all students of the group, infecting a rooster and a rabbit with the smallpox virus.

2. Questions for control.

3. Teacher's explanation.

4. To demonstrate: a) the result of a biological sample; b) the method of preparing smears for smallpox.

5. Independent work of students: a) taking swabs for smallpox; b) Painting smears according to the Morozov method; c) look at smears with a microscope; d) draw the results.

6. Ending the training.

7. Assignment for the next exercise.

Methodological instruction

Difficulties in preparing for this exercise are the selection of the smallpox virus and the living organism that is susceptible to it.

To infect roosters (on the scratched crown or feather follicles) NR-1 chicken pox virus vaccine strain is used.

Chicken pox is easy to find. is the N'yu-D jersey strain of pigeon pox virus used as a vaccine against the disease.

With these viruses, an effective biological sample can be placed in pigeons.

Until recently, injecting the smallpox virus into the skin of rabbits, which was used to vaccinate humans, showed good results.

When this is not possible in smallpox, many available chicken fetuses are used for biological specimens. Due to the infection of the chorionallantois membrane of the chicken fetus with the above-mentioned viruses, swelling develops and characteristic nodules (smallpox) appear.

Preparation of reagents for painting smears according to the Morozov method does not cause many difficulties. It is advisable to prepare sets for staining, which consist of reagents 1, 2 and 3, immersion oil, gasoline, penicillin vials, eye pipettes. All the vials in one set can be placed in the lid of a Petri dish and given to two people from one set.

Review questions

1. Give the diagnosis of smallpox.
2. Differential diagnosis of smallpox.
3. for suspected smallpox .

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory exercise : Diagnosis, prevention and countermeasures of pig disease .

Purpose: To acquaint the students with the diagnosis, differential diagnosis, immunity, general and special treatment, and preventive means of swine rabies disease.

Material and equipment:

Smear, microscope, color tables, standard strain, pat.material, paints for smear smear, standard stamp smear, immersion oil, special anatoxins, slide.

Basic concepts.

Diagnosis, differential diagnosis
1. Immunity
2. General and special treatment
3. Quarantine measures
4. Farm health, preventive means

SWINE SCURVY - *Erysipelas Suum* - Swine scurvy is an infectious disease that mainly affects 3-12-month-old pigs, almost acutely, septicemia is clearly visible, in chronic cases, verrucous endocarditis, serofibrinous polyarthritis and skin There are signs of necrosis.

The pathogen was discovered by L. Pasteur and Thume in 1882. In 1883, L. Pasteur developed a vaccine from a weakened strain. Anti-Saramas hyperimmune serum was developed by Lawrence and Leclanche in 1895-1899. D.F. Konev created the vaccine-producing strain using the L. Pasteur method.

Economic damage - death, treatment and preventive measures, costs of quarantine measures, reduced productivity, forced slaughter for meat.

The disease is widespread worldwide.

Trigger – Erysipelothrix insidiosa (Bact. Rhusiopatiae suis) occurs in the form of a rod or curved rod, sometimes in a filamentous form. Gram positive, produces spores and capsules. In normal solid nutrient environments, it forms smooth (S - ANTIGEN), FEATHERY (R - ANTIGEN), MIXED COLONY (O - ANTIGEN). Widely distributed in nature.

Disinfectants: 2% formaldehyde, NaON, 10% - chlorinated lime, 10-20% freshly slaked lime.

In addition to pigs, seramas infection is sporadically found in roe deer, big horned animals, sheep, reindeer, dogs and many other wild animals, in zoos - in zoos (spotted deer, gazelles, keguru, wild pig, seal, apple, mink, chicken, tussock, etc.). From poultry in turkeys and ducks. It has also been found in humans. White mice and pigeons are prone to laboratory animals.

The source of the disease is sick pigs and healthy pigs, insects, rodents, ticks belonging to the Ixoda family.

(Dermacentor pistus)

AND contaminated food, hygiene violations, and people in the service cause the spread of disease.

Transmission - *per oraeno* in the tonsils, intestines,

in *per os* } bridging lymph nodes.

It develops in the lymph nodes when passing through skin wounds.

When the disease is in an extremely acute form (when it passes at lightning speed) in pigs aged 7-10 months, when ventilation does not meet the requirements, during transportation - the temperature rises and the heart fails, and the pigs die.

Acute course, semi-acute course, chronic course - mild clinical signs.

In warm, black cattle, vultures, dogs - it often passes with secondary infection, in sheep it passes with the sign of polyarthritis, bronchopneumonia.

In poultry, saramas infection is epizootic.

Pathanatomy - foamy mucus flows from the nasal cavity, parenchymatous organs are in congestive ulcers, and it turns blue due to venous hyperemia. The nose is filled with venous blood, swollen, dark blue. Hepatization is observed in the bile. Verrucous inflammation, dystrophy is observed in the endocardial layer of the heart.

Granular necrotic nodules are present in the heart muscle.

Diagnosis:

- clinical
- epizootological
- administration of special serum and antibiotics
- pathanatomical
- bacteriological, microbiological
- bioassay methods
- feather the sample is sent in 30-40% glycerin
- method of using fluorescent antibodies

q is being used in the following years.

Differential diagnosis .

Plague, pasteurellosis, listeriosis, anthrax, sunstroke, sunstroke.

Plague is highly contagious, death is high, it spreads quickly, pigs of all ages are infected. In plague, blood flows from internal mucous membranes and serous membranes, and pneumonia is observed in the lungs.

In pasteurellosis - in pigs kept in poor hygienic conditions, signs of pleuropneumonia are observed.

In listeriosis - often occurs in young pigs, signs of meningoencephalitis are observed.

Anthrax is relatively rare, symptoms of angina appear, a form of carbunculosis.

Sunburn - occurs mainly in cancer, in hot weather. The main method is carried out by bacteriological examination.

Treatment .

Antiviral serum and antibiotics are used. The effect is better if both are used together. Special serum 1-1.5 ml/kg intramuscularly, after 8-12 hours of treatment, this dose is re-injected. Penicillin 2-3 thousand IU/kg, 3-4 times a day with an interval of 6-8 hours.

Ecmonovacillin 5-10 thousand IU/kg, 2-3 times, interval 24 hours. Erythromycin 5-8 mg/kg 2-4 times, interval 6-12 hours.

The result is better if special treatment is carried out along with symptomatic attendance. Special treatment is effective when combined with symptomatic treatment.

Immunity - animals that recover from illness develop strong immunity.

1. Live vaccine - depot vaccine is the 2nd vaccine of DFKonev. From the 2nd month, two vaccinations are carried out, subcutaneously, with an interval of 12-14 days.
2. VR-2 strain vaccine against Saramas (Romania). Vaccination is carried out from 2-4 months, revaccination is carried out after 4-5 months.
3. Formula vaccine with concentrated aluminum hydroxide is injected between the muscles in the sagittal region or neck, twice, the interval is 12-14 days, vaccination is given from 2-4 months, revaccination is carried out after 4-5 months.

***Preventive means* :**

2. Ration, hygienic requirements must be optimal.
3. Deratization should be done on time.

4. Vaccination must be carried out on time (in addition, it should be carried out continuously and planned).
5. It is necessary to prevent sarcoid, plague, pasteurellosis, paratyphoid and other dangerous infections in order to avoid secondary infection.
6. When the disease appears, sick pigs are quickly separated, isolated and treated, the rest are given preventive serum - passive immunization, after 10-12 days they are vaccinated.
7. When sick pigs are slaughtered for forced meat, the meat is only boiled and then sold.
8. Quarantine is canceled after the 14th day after the last sick animal is lost, the final disinfection is carried out.
9. Tools and equipment are also disinfected.

Control questions

1. Epizootology of swine fever?
2. Pathogenesis of the disease?
3. Measures to prevent disease from entering the country?
4. When a disease is suspected, should measures be taken to prevent its spread?

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory training : Serological reactions used in the diagnosis of infectious diseases.

Purpose . Organize a complete blood draw according to the plan, each student mastering the complete blood draw, this work is performed on different farm animals. Sending blood for laboratory examination, preservation (stabilization) of blood samples, filling out and sending a referral letter.

Blood sampling equipment; blood, serum, plasma samples: cotton, scissors, 5% iodine tincture, 70% ethanol, tourniquet, test tubes, tripod, needles for blood collection.

2. Serological reactions at the department, institute vivarium, educational experimental farm, regional veterinary laboratory: RA, RSK, RP, ... etc. to determine the result of performing the tasks, to master keeping a journal.

3. Students strengthen the theoretical and practical knowledge acquired in 2-3 courses (microbiology, physiology, pathophysiology, clinical diagnostics, etc.) by performing in epizootology practices and laboratories.

The role of serological testing in diagnosis. Organization of mass serological examination

1. Procedure for putting the Rozbengal reaction
2. Methods of obtaining blood from animals.
3. Preparation of documents for sending blood samples to the laboratory

Purpose: to organize a mass blood collection according to the plan, each student should master the complete execution of blood collection, this work is performed on different farm animals. Sending blood for laboratory examination, preservation (stabilization) of blood samples, filling out and sending a referral letter.

Blood collection devices, blood sample.

Place and methods of training. In the clinic of the department, they get acquainted with the instruments and blood samples. He learns the method of extracting serum from coagulated blood in a test tube (practicing taking blood from different q/x/h/ in aviaries). In farms, they get acquainted with taking blood from animals and sending it to the laboratory, as well as document work.

Blood sampling method. When taking blood from animals, in 2 different amounts:

1) less and
2) 2) can be carried out in large quantities.

For small amounts, a few drops, and for large amounts, a few ml or even more blood is drawn. All instruments used for blood collection are sterile, one per animal. The number of blood tubes should be 5-10% more than the number of animals. I-51, I-52, Casper needle, injection needle, Dufo needle, Saykovich, Ananyev needle are used for blood sampling.

The test tubes for blood collection must have a special stopper, a paper label, and a rubber sealing ring. The place where blood is to be drawn must be cleaned with Cooper's scissors, 3% phenol, 70% alcohol, bandage, cotton, iodine wipes. The blood should not foam in the test tube. In cold weather, blood is kept in water baths at 30-35 ^{o}C for 30-35 minutes. Then it is stored in a cool dark place at a temperature of 45 ^{o}C. If hemolysis is observed, the blood is considered unfit for testing. Pig blood is hemolyzed especially quickly.

Y.Sh.H and M.Sh.H, horses, camels, vena jugularis - blood is taken from the jugular vein, and in pigs, from the tip of the tail, in poultry, from the crown and underwing veins.

Serological test reactions are very important in the diagnosis of infectious diseases. Because of IFA, PSR, the accuracy level is very high, there is almost no error. Antigen and antibody titer-based methods are special test reactions and are widely used in veterinary practice.

The collected blood is sent to the veterinary laboratory with the following referral letter.

Veterinary laboratory mark _____ date of arrival of material _____
Brought the sample _____ invalidated _____
Which vet. laboratory _____
Address: _____

Sent _____ blood sample _____
_____ farm _____
(type of animal) (Farmer and Vet. point)
_____
_____
(province, district ...)
_____ _____
(which test reaction) (for which disease)
Farm goods _____
_____
(healthy, unhealthy, when, with what kind of vaccine)
The inspection is being carried out for the first time or again (underline as appropriate).
Time of blood draw _____

List of blood drawn animals.

No	Farmer x/address, animal owner I.Sh. and address	Animal sex, age, color	Tag number, nickname	Check result				
				RA		RSK positive, suspicious, negative	RMA and L	
				positive, suspicious, negative	titer		sero type	titer
1.								
2.								

Veterinarian (specimen sender) Veterinarian who performed the examination
_____ signature _____ signature.

Serological reactions:

1. RA- reaction agglutination
2. RSK - (KBR) - comp. the garden React.
3. RDSK –
4. RP - precipitation reaction.
5. RF-flocculation
6. IFM - immunoenzymatic method
7. RN - neutralization reaction
8. IFM - synonyms RI, SAF
9. MFA - fluorescence antibody method
10. RAL - reaction agglutination latex
11. RGA - hemagglutination reaction
12. RZG - reaction zaderzka geadsorption
13. RIA - radioimmunological analysis
14. RIM - radio immunological method

15. RINA - projection inhibition neural activity
16. RID - reaction immunodiffusion
17. RISAF - reaction immune absorption antibody, mechanical enzyme.
18. RIE - reaction inhibition elution
19. RKOA - reaction coagglutinase
20. RNGA - reaction indirect hemagglutination
21. RRG - the reaction of radial hemolysis
22. RTAL - reaction tormogenia agglutinasii latex
23. RTGA - reaction tormogenia agglutinasii
24. RTNGA - reaction tormogeny nepryamoy hemagglutinasii
25. RMA - reaction microagglutin.

Control questions

1. Writing and submitting abstracts about serological reactions performed in regional and district veterinary laboratories.
2. The role and importance of serological testing methods in measures against E. pizootics.
3. Procedures for taking blood samples from various animals, separating serum, preserving them, and sending them to the laboratory.
4. Say conservation (stabilization) of blood and serum.

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Laboratory training: Allergic testing methods in the diagnosis of infectious diseases (VITI brucellosis, tuberculosis in the research laboratory).

Purpose *:* To study allergy testing in different animals, to learn how to determine and document the results of allergy testing based on practical laboratory results.

Necessary equipment, reagents : Equipment, tools and allergens to induce an allergic reaction.

Place and style of training .

Training is held in aviaries. Students are introduced to the instruments used in allergy testing. Prepares the necessary reagents and solutions. Learns to induce allergic reactions in different animals, injects allergens.

Allergen diagnostic test (ADP) includes a reaction to the introduction of a specific allergen (specific) to an organism sensitized by a stimulus. In veterinary medicine, allergens are introduced into the body in 2 ways:

1. To the eye;
2. Between the skin;

Eye allergy test - each animal is injected with 3-4 drops of allergen (according to the instructions) using a sterilized pipette. The eye into which the allergen is injected must be healthy. The hands of the veterinarian must be sterilized. When the allergen is injected, the results are determined after every 3 hours, i.e. 3-6-9-12 and after 24 hours. Positive reaction - the animal is sick, the mucous membranes of the eyes are inflamed, red, swollen, pus accumulates, purulent liquid flows from the corners of the eyes. When the reaction is observed, the amounts of ammonia (NH_3), carbon dioxide (CO_2) and similar gases in the livestock should not exceed the norm. These reactions are often associated with mange in horses and tuberculosis (tuberculosis) in large animals.

Allergy probe injected between the skin - this probe is used more often in veterinary medicine. The allergen administered to each animal is administered according to the guidelines for the type of animal. In horses and cattle, in the middle part of the neck, in sheep and goats, under the tail or in the part of the chin, in pigs, on the base of the ear, i.e. on the lateral surface, in poultry, in the ear (below the muzzle), dogs, monkeys. , is injected into the medial part of the thigh (hairless area) in fur-bearing animals, in the abdomen in camels, and in the upper eyelid in beavers. The place where the allergen is to be injected is cleaned and disinfected with wool + **sharp (cooper) scissors and then the allergen is injected.** 70% alcohol, 3% phenol, 5% iodine tincture are used as disinfectants. The thickness of the goitre to be injected with the Stangen circle is measured and recorded, and then the allergen is injected according to the instructions. The result of the reaction is determined within 72 hours. If the reaction needs to be repeated, it will be done within 5 days.

Subcutaneous injection test - used in mange disease. Before this, the temperature of the animal is measured and the injection site is palpated. In recent years, this reaction is rarely used.

Devices that inject allergens between the skin are PIL-1, Ovod (without needle), IBV-01 without needle. Allergic reaction takes place in the following form.

Normergy is a normal reaction,

Hyperergy - high sensitivity,

Hypoergy - decreased sensitivity,

Anergy is the y- axis of sensitivity.

The hyperergic form of the allergic reaction is a protective reaction of the body and is related to the relative immunity of the body. An increase in hyperergic reaction indicates an increase in the pathological process in the body. This is a sign of a bad outcome of the disease.

An allergic reaction is observed to increase or decrease under certain conditions. Factors that weaken the allergic reaction include: 1. Many infectious and non-infectious diseases that pass acutely.

2. When sand diseases are severe,

3. In avitaminoses,

4. In liver diseases,

5. Dystrophy,
6. Strong sunlight ,
7. b Ogazik,
8. Observed in protein deficiencies.
9. Serum
10. dry, cracked skin.
11. treatment procedures, when various medicines are injected into the body.

Diminution of the allergic reaction is observed in the following cases: even in non-sensitized animals, when the metabolism of some substances is disturbed, after some vaccines, when pus accumulates in the body.

Increased allergic reaction in the body iodine, histamine and others. will be tracked when sent. The etiology of an allergic reaction can be various infectious (microbe, fungus, virus...) pathogenic or non-pathogenic types. Allergic reaction is used in the diagnosis of mange, tuberculosis, brucellosis, paratuberculosis diseases in veterinary practice.

Allergic and diagnostic drugs are being continuously worked on.

Diagnostic allergens are processed from pathogenic and non-pathogenic micro-bacteria, fungi, viruses ... their parts.

Questions for control .

1. Allergy and its types.
2. The main features of infectious allergy.
3. Factors affecting allergic reactions.
4. Ways of introduction of allergens into the body.

Books:

1. Urban VP "Praktikum po e pizootologii ii nf.bolezney " st 7-37.
2. Parmanov MP et al. "Epizootolgia" pp. 15-23.
3. Antonov V.YA, Blinov PN "Laboratornye issledovaniya v veterinirii" st 5-339.

Laboratory training: Pathanatomical examination methods in the diagnosis of infectious diseases (in the VITI pathanatomical laboratory) .

Plan :

1. The role and importance of pathologoanatomical examination in diagnosis.
2. Sampling procedure for bacteriological examination.
3. Smear preparation, hardening (fixation), painting, inspection.
4. Preparation of laboratory shipment documents of pathological material samples.

The goal is to learn the rules of dissecting animal carcasses, identifying pathanatomical and pathogistological changes, learning the methods of bacteriological examination of animal materials, and using the results in diagnostics.

Necessary equipment, reagents, equipment. Farmer x furnace model, models of internal organs, pathological material, fixation of pat materials, jars, cellophane bags, glass slide, cover glass, scissors, scalpel, tweezers, Pasteur pipette.

Place and methods of training are held at the department, veterinary laboratory, laboratories of UzVITI. The pathanatomical examination method is also a component of complex epizootological examination, and the information obtained in it is of great importance. At the end of the training, students must know and master the following questions: where dead animals are examined for pathanatomical examination on a farm where different animals are raised, the rules and procedure of examination, the rules for taking and sending samples for pathomorphological examination. Rules for disposal of residual materials after examination, rules for sampling and examination for histological examination, sterilization of used instruments and rules for personal hygiene.

Histological examination method is performed by a specialist. Histological examination is of great importance in the diagnosis of infectious diseases . Isolation of the causative agent from the patient material, cultivation of its pure culture is an etiological diagnosis. This part of the work is considered a bacteriological examination and consists of the following:

1. Smear microscopy of the stimulus.
2. Cultivation of Sof kutura in nutrient media.
3. Biotesting in laboratory animals.

These 3 tests form the basis of laboratory diagnostics. These methods are also useful in identifying mixed infections and are considered a reliable method of testing.

Sampling rule for bacteriological examination: sick or dead animals are sampled for bacteriological examination. The goal is to prove that the sick animal is a carrier and spreader of bacteria. Often milk, sputum, blood, feces, urine, pus, exudate, exudate from wounds on the skin, etc. it is necessary to follow the rules of asepsis and antiseptics when taking samples from sick animals for bacteriological examination.

When taking a sample of milk from a sick animal, the udder and udder teats are cleaned with hot water and soap. The teats are cleaned with cotton wool at 70 alcohol. Hands are washed with soap and then wiped with alcohol. The first milk that comes out is milked on the ground. The next one is milked into a container, 25 ml of milk is taken as a sample. Each teat is taken separately. Sometimes a sample is taken with a milk catheter.

Leptospirosis causative agents are sought in the urine sample. In this case, urine is taken from the bladder with the help of a catheter, 50-200 ml.

A stool sample is taken from the colon. If there is a change in the mucous membrane of the rectum, the mucus is removed. The nipple is removed using a spatula or a special surgical spoon. The rectal scope is shipped in a separate container.

To take mucus samples from the nose, mouth, and throat, wash them with distilled water using a syringe, then take them with a sterile swab and put them in 0.5

ml of physiological solution. Laryngeal fluid is taken using a special probe. A probe is sent to sheep, goats and cattle through the nose and mouth.

A swab is also taken from purulent wounds, and skin, wool... are also taken separately. Dead animals, a piece of parenchymatous organs from discarded fetuses, internal organs, fetal fluid, placenta ... are sent separately. If the die is small, it is sent completely in a cellophane bag. Blood, exudates are taken in a Pasteur pipette and fixed.

The smears are prepared and sent in 3-5 pieces. The sample sent to the laboratory is placed in 30% glycerin or petroleum jelly. The amount of applied solutions should be 4-5 times more than the sample.

The sample sent for virological examination is washed in glycerol prepared in 30-50% isotonic solution. Intestines and tubular bones are preserved in Nacl salt.

A letter is sent to the laboratory, the type of animal, sex, age, from which organs the pat material was taken, number, nickname, examination reaction, pathomorphological changes are written in the letter. It is necessary to send whether the animal was treated, the history of the disease, how the sample was preserved from the leaflet, and epizootic information.

1. The diagnosis of infectious diseases differs in reliability and accuracy.
2. Primary diagnosis is based on clinical signs and epizootic data.
3. In the primary diagnosis, the disease must be an infectious disease.
4. Suspicion of any infectious disease will definitely be confirmed by a complex epizootological examination.

We have no right to forget these rules.

_____v to the veterinary laboratory

Address: _____

Sent for review _____

Feather material sample _____ animal _____ belonging to the farm _____

The time when the disease was registered _____

Death registered _____

Clinical signs _____

Pathanatomic changes _____

Primary diagnosis _____

Sample sent time _____

Position _____ signature _____ seal

Control questions:

3. Rules for working with an animal that died of an infectious disease.
4. Rules for pathanatomical examination of an animal that died from an infectious disease in laboratory, f/ x, and field conditions.
5. Ensuring safety of activity in pathanatomical examination.
6. Rules for receiving and sending patmaterial.

7. Send a referral letter.
8. Importance of comprehensive epizootological investigation.

List of references

1. Antonov V. YA., Blinov PN "Laboratornye issledovanie v veterinirii". Moscow - 1971, str. 5-30.
2. Urban. VP "Praktikum po e pizootologii ii nf.bolezney".Uch.pos . L. 1987
3. MPParmanov et al . "Epizootology" 2007, pp. 30-50.

Laboratory training: Methods of clinical and allergic examination of animals (in educational and vivarium animals) .

Methods of clinical examination in animals.

Infectious diseases are fundamentally different from non-infectious diseases. It is examined by clinical and epizootological methods.

In the clinical method, animals are examined based on their clinical signs, body temperature, habitus, and the shape of the nose cheek. In the epizootological method, the causative agent of the disease is detected by laboratory examination, staining in special methods, taking samples from sick animals, and finding the causative agent of the disease in laboratory conditions under a microscope.

These diseases appear only when a disease-causing microorganism or virus enters the body. Secondly, the pathogen is characterized by its transfer from a sick animal to a healthy one. This allows the disease to continue to spread and become widespread. Therefore, the infectious disease spreads over a large area, and the epizootic process continues continuously, causing great economic damage to the national economy. Thirdly, antibodies are formed against the pathogen that entered the body, fourthly, it goes through stages (incubation period, manifestation of clinical signs, development and extinction).

Infection and infectious diseases, clinical forms and dynamics of infectious diseases, immunological reactivity and immunity, epizootic process and its driving forces, epizootic focus and natural focus, basics of epizootological investigation, nomenclature and classification of animal infectious diseases, infectious basic principles of disease prevention, treatment, rehabilitation and combating them.

When studying the nature of the epizootic process in a particular infectious disease, it is necessary to take into account that it is related to biological, natural-geographic and socio-economic (economic) conditions.

The method of comprehensive epizootological inspection includes the following: a) epizootological inspection and monitoring of farms; b) historical and geographical comparison of the epizootic process; c) epizootological experience; g) consists of statistical examination and epizootological analysis.

Epizootic control measures are scientifically based systems of preventive and health measures. Its main task is to protect people from zooanthroponous diseases and to create a stable healthy state in terms of infectious diseases in order to increase their productivity without allowing the death of animals.

Preventing the emergence and spread of infectious diseases is the main task of epizootology, because preventing the disease is easier than fighting it.

Another task of epizootology is quick and reliable diagnosis of an infectious disease in a farm, settlement or farm. Preventing the emergence and spread of infectious diseases is the main task of epizootology, because preventing the disease is easier than fighting it. taking measures to prevent the spread of the disease, and carrying out measures to treat and rehabilitate it, in compliance with the requirements of quarantine or restriction. It is very important to use modern laboratory diagnostic methods, treatment and mass vaccination, keep healthy animals under epizootic control, and control the veterinary-sanitary condition of the farm.

The occurrence, development, and fate of an infection depend not only on the virulence and amount of microorganisms and viruses entering the organism, but also on the natural resistance of the organism against these pathogens. Therefore, it is necessary to focus not only on pathogens, but also on strengthening the defenses of the macroorganism (increasing natural resistance) and on external environmental factors that contribute to the development of the disease.

Susceptibility is the ability of an animal organism to become infected and diseased when in contact with a pathogen. *Endurance* (natural resistance) is the opposite of susceptibility, a state of resistance of the organism to a stimulus. Resistance is directly related to immunoreactivity, and this condition depends on the organism (constitution, sex, age, anatomic-morphological characteristics of the animal species, development of the immune system, etc.) and several factors of the external environment (stress, quality of nutrition and zoohygienic standards storage based on). Therefore, for the emergence of an infectious disease, the causative agent must be pathogenic and virulent, it must fall on a genetically predisposed animal, the level of protection of the macroorganism against the disease is low, the development and reproduction of the causative agent that enters the body is external it is necessary to have conditions such as environmental support (aerogenous infections increase during cold and wet weather, and toxicoinfections increase during food in summer). Cold and very hot also reduce the body's resistance. Even a virulent pathogen introduced into a genetically predisposed organism may not be able to cause disease in a highly resistant animal. Only in a very small number of highly dangerous acute transmissible diseases (diseases, anthrax, swine fever, anthrax, sheep pox) does contact of the agent with a susceptible animal usually cause disease. The causative agents of these diseases fully satisfy all three conditions of Henley's and Koch's postulate: a) the causative agent is always isolated and observed in a diseased animal with clinical symptoms; b) this pathogen is not isolated in other infectious diseases; c) when a susceptible animal is infected with a pure culture of the causative agent, the same disease is called.

However, there are many infectious diseases of the 2nd group, the presence of micro- and macroorganisms is not enough to cause them artificially, they are caused by certain external conditions: stress, factors that lower the body's resistance or other stimuli. The assistance of shooters is required. These do not fully satisfy all three conditions of Koch's postulate. It will be very difficult to call their clinical and

pathologoanatomical signs of this disease in an experiment. Therefore, many factors are needed to cause an infectious disease: the virulence of viruses and microorganisms, the amount of them entering the body, the gate of infection, the resistance of a susceptible animal organism, and external environmental factors (cold, very hot, insufficient nutrition, chronic poisoning, tight storage) depends. All these together are the etiological factor that causes an infectious disease - *the etiology of the disease* . Thus, the etiology of the disease has a broader meaning than the cause of the disease.

Clinical forms of infectious diseases and dynamics of manifestation.

Infectious diseases differ from non-infectious diseases by their specificity - the presence of the causative agent. Each infectious disease is caused by its causative agent, so it does not cause a specific, different disease.

In animals Allergic testing methods .

Allergic testing is performed to detect animal and poultry tuberculosis. Purified PPD for mammals (protein, pouioified derivative, tuberculin and PPD tuberculin used for poultry).

In the allergic examination of animals and poultry, the allergic method is recognized, and in horses it is determined by the drip method.

The technique of intradermal injection

Before injecting tuberculin into the skin, the thickness of the skin is measured with a cutimeter, the wool is trimmed and treated with 70% ethyl alcohol. Tuberculin in large horned animals, buffaloes, deer in the middle of the neck, in bulls under the tail, in the belly of camels, in sheep, goats, dogs and monkeys in the inner side of the rump or in the armpit, in blackbirds - intrapalpebral, i.e. the top of the eye, the eyelid, is sent to one side of the earring in birds.

Tuberculin is administered to mammals (except monkeys and baboons) 0.2 ml, to monkeys, baboons and birds - 0.1 ml using special small needles and syringes. After the administration of tuberculin, the reaction to it is evaluated after 72 hours when examining large horned animals, buffaloes, camels and deer, after 48 hours when examining sheep, goats, pigs, dogs, cats, and fur animals. , and when checking birds, it is evaluated after 30-36 hours. Skin thickness is measured using a calliper to evaluate the reaction. Large horned animals, buffaloes, camels and deer, if the skin thickness is 3 mm and more, other animals, if the skin of the tuberculin injection site is swollen in poultry, if the eyelid is swollen in blackbirds, the result is considered positive.

Eye drop technique

The method of instillation of tuberculin into the eyes is carried out 2 times with an interval of 5-6 days. Allergen is instilled with an eye pipette in 3-5 drops on the mucous membrane of the lower eyelid with closed eyelids. This method cannot be used if the animal's eye is damaged.

The test results are taken into account every three hours after the first instillation of the allergen into the eye, i.e. after 3, 6, 9, 12 and 24 hours, and after 3, 6, 9 and 12 hours after the second instillation. In the case of a positive reaction, the mucous

membranes of the eyes turn red, and pus begins to flow from the inner corner of the eyeball.

Serological and bacteriological examination is carried out. Complement fixation reaction (CRT) is used in serological examination of large horned animals. The reaction is made with complex tuberculosis antigen (CTA) and is carried out at the diagnostic slaughterhouse of animals. Bacteriological tests are carried out if they are not visible or unclear, and to determine the type of mycobacteria.
Bacteriological examination uses the method of bacterioscopy (smears are prepared and stained by the Sil-Nilson method) to isolate the pure product (nutritional media are planted in Leventein-Gelberga and Petriani media with eggs and a bioassay is carried out).
A bioassay is used to determine which type the pathogen belongs to. Guinea pigs, rabbits and chickens are used for biotesting.
Differential diagnosis: When diagnosing tuberculosis, it is necessary to differentiate from contagious pleuromoniasis (PVL) and pasteurellosis (breast form).
Scheme of differential diagnosis of a tuberculosis patient.
Diseases with damage to the lungs.
PVL (poval. vospol. legkix) gross inflammation of the lungs
Course: tuberculosis chronic Course: Lightning fast, acute,
semi-acute and chronic
Superficial lymph nodes are enlarged, swollen in upper lymph nodes
there is no difference
When viewed in the lung, in the lymphatic layers, when viewed in the lung, in marble nodules, serous, in chronic cases
Sometimes in the tissue of the udder and in other organs and tissues, nodules with a curdled necrotic mass. The result of tuberculinization when testing for tuberculosis. Tuberculosis. The result of a negative reaction when testing for tuberculosis is contagious pleuropneumonia (PVA).

Allergy test. Allergens are used in this. VIEV allergen - brucellin gives good results. From it, 0.5 ml (under the skin) is injected into the left eyelid in the palpebral way to sheep and goats. It is injected into the skin on the outer part of the supra-ear in pigs. A positive result is evaluated by the appearance of a hard swelling under the lid, and in pigs by redness and swelling.

1. Epizootological method (incidence of the disease, spread of the disease, transmission)
2. Clinical method (throwing, bursitis, orchitis, abscess in horses and pigs)
3. Pathanatomic method (liver, spleen, kidney abscesses, fetal hemorrhage, pus necrotic nodes...)
4. Allergic method (brucellin is injected under the skin palpebrally).
5. Method of laboratory examination:
a) Bacteriological method. Gram negative, stained by the Kozlovsky method.
Grown on Ploskirev agar.

b) Serological method (RBP, RA, RSK. Milk ring reaction).
g) Biological test method (in guinea pigs, white mice) is carried out.
Differential diagnosis. Brucellosis should be distinguished from leptospirosis, campylobacteriosis, and trichomoniasis.

Diagnosis. Diagnosis of mange disease in horses, if the disease is acute, if clinical signs are clearly manifested, it is not difficult to make a diagnosis of the disease based on clinical and epizootological data. However, manga is mostly chronic or latent. For this reason, an allergic diagnosis is made. Mallein is used in allergy diagnosis.

Eye, subcutaneous, and intradermal methods of injecting **mallein are used** in practice .
Method of injecting mallein into the eye. The main method. In this case, 3-4 drops of mallein are instilled into the eye conjunctiva of the horse twice with an interval of 5-6 days with a sterile pipette. The result is determined 3, 6, 9 and 24 hours after the first drop:

positive result (+) if the eye has purulent conjunctivitis or pus in the inner corner of the eye and hangs like a thread; if purulent mucus flows from the nose or the 2nd eye also puss;

suspicious result (+ -) if the conjunctiva is red, the eyebrow is swollen, tears flow and there is a little pus in the inner corner of the eye;

a negative result (-) is when there is no change in the conjunctiva, but there may be tears and a little pus.
Allergic reaction is repeated after 5-6 days with a suspicious and negative result. The result of the reaction is calculated after 3, 6, 9, 12 hours.

The subcutaneous method is used if it is not possible to inject the allergen into the eye. For this, the body temperature of the horse is checked 3 times and it should not exceed 38.50C on average. 1 ml of mallein is injected under the skin of the neck. The next day at 6 a.m. body temperature is measured every 2 hours until 6 p.m. It is then measured after 24 and 36 hours.

a positive result (+) if the body temperature rises to 400C and stays for 6-8 hours, even if it then returns to normal; or if there is a hot, painful swelling at the injection site and the horse's body temperature is 39.60C;

suspicious result (+ -) if the local reaction at the injection site of mallein is weak, the body temperature is up to 39.60C or above 400 and there is no local reaction;

negative result (-) if there is no local inflammatory reaction and the body temperature does not exceed 390C. The subcutaneous method detects up to 95% of sick animals, but the technique is more difficult to perform.
The intradermal method is used for allergy testing of semi-wild, non-reining horses. It does not stay out of sight. In this method, mallein is injected into the skin of the neck in a dose of 0.2 ml, and the result of the reaction is counted after 48 hours. a positive result (+) if there is a circumscribed, pasty, hot, painful swelling measuring 2 x 3.5 or 14.5 x 25 cm at the injection site of mallein. Horses intended for export are

checked twice. Horses that did not respond to mallein were re-administered with mallein after 48 hours and scored after 24 hours.
Only horses that test positive for maleine are tested for the complement binding reaction (KBR). This makes it possible to distinguish horses that are undergoing an active mange process. Only horses that test positive for KBR are not considered mange without a positive allergic reaction.

Study materials on independent education

SUBJECT: Contributions of department scientists to the study of infectious diseases

(Prepare from the literature and complete assignments)

Main textbooks and training used list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Vaccines and other biological preparations made in K aphedra

(Prepare from literature and complete tasks)

Main textbooks and training used list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural magazine of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Infectious epididymitis of dogs .

(Prepare from the literature, write an abstract and submit it perform)

Main textbooks and training used list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis

9. www. fvat@.academy.uzsci.net

SUBJECT: Tularemia , mellioidosis . _

(Prepare from literature and complete tasks)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Dengue fever.

(Prepare from the literature and complete assignments)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Paratuberculosis.

(Prepare from the literature and complete assignments)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Chlamydia abortion in sheep

(Prepare from the literature and write an abstract)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Viral gastroenteritis disease of pigs .

(Prepared from the literature and assignments perform)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Rotavirus diarrhea in calves.

(Prepare from the literature, write an abstract and submit it perform)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Calves with coronavirus enteritis.
(Prepare from the literature and complete assignments)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Pleuropneumonia.
(Prepare from the literature and complete assignments)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural magazine of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Pig plague .

(Prepare from literature and complete tasks)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural magazine of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: B otulism disease.

(Prepared from the literature and assignments perform)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .
2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural magazine of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Lyme disease.

(Prepared from the literature and assignments perform)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural journal of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

SUBJECT: Dysentery.

(Prepare from the literature and complete assignments)

Main textbooks and training used
list of manuals

Basic textbooks and study guides

1. Salimov XS, Kambarov AA, Salimov IX, "Epizootology and Infectious Diseases" Textbook. Tashkent , 2021 .
2 . Salimov XS, Kambarov AA "Epizootology" Textbook. Samarkand, 2016.
3 . Parmanov MP et al., "Epizootology" Textbook. Samarkand, 2010.
4. Parmanov MP et al., "Epizootology" training manual. T, 2007.

Foreign books

1. M. Jackson Veterinary clinical pathology. America 2010 year .

2. Shevchenko A.A., Djailidi G.A., Shevchenko L.V., Chernykh O.Yu. Diagnostics of infectious and living diseases (educational posobie). – Krasnodar: KubGAU, OOO "Caucasian Typography", 2014 .

ADDITIONAL literature

1. Bessarobov BF i dr "Infectious diseases of life" uchebnik, Moscow 2007.
2. Veterinary legislation. Tashkent, 1998.
3. Agricultural magazine of Uzbekistan. Tashkent, 2008
4. Handbook of veterinary medicine. Uch.pos. Rostov-on-Don.; 2001
5. www.Ziyo.net.uz. _
6. www.vetjurnal.uz
7. www.sea@mail.net21
8. www.veterinariy.actavis
9. www. fvat@.academy.uzsci.net

GLOSSARY

Name of the term in Uzbek language	The name of the term in English	The meaning of the term
Epizootology	Epizootology	a science that studies the laws of emergence and development of epizootics, their prevention and control measures.
Epizootic	Epizootic	widespread spread of any infectious disease
Infection	infe s tio	entry of disease-causing viruses and microbes into the body. This condition causes an infectious disease with certain clinical symptoms. There are also asymptomatic infections. Viruses and germs that cause disease are sometimes called infections.
Alimentary infection	alimentary	entry of the causative agent into the body through the mouth.
A erogenous infection	a ergenes	an infection caused by airborne pathogens.
Exogenous infection	exogenous	a disease caused by pathogenic viruses and microbes entering the animal body from the external environment
Infectious exanthema	exanthema	is a focal inflammatory lesion of the skin, which appears as a result of some infectious diseases. For example, such a situation was observed in animal smallpox.
Interferon	interrio	a low-molecular glycoprotein, a protein present in a virus-infected cell that prevents the development of viral diseases. Its molecular weight is 20-40 thousand daltons, it does not kill the virus, it is non-toxic to cells. Interferon is important in the formation of general resistance. Interferon produced by one virus also prevents the reproduction of other viruses.

Capsid	cap	the component of the virion, the shell, protects its nucleic acid from the external environment, consisting of a combination of capsomeres
Quarantine	quarantine	as a system of temporary measures to limit the spread of infectious diseases, to make it possible to completely end the disease by keeping it in the center of its origin.
Clone	clone	the offspring of a bacterium, cell or virus, as well as a virus or a single-celled (multicellular) organism created by the vegetative way (asexual reproduction), having the same characteristic (sign) ancestor
Contamination	contamination	contamination of fodder, water, soil, working tools and external parts of the animal body and other objects with pathogenic microorganisms, viruses, infection of infectious substances
Contagiousness	contagion	a term used to describe the rapid spread of infectious diseases. Contagious diseases that spread very quickly and widely include smallpox, swine fever, and equine influenza.
Leukosis	Leukosis, leukemia	Infectious virus disease of a tumor nature is characterized by an excessive increase of immature leukocytes in the blood as a result of excessive activity of tissues that produce white blood cells. RNA oncovirus causes leukemia in cattle, sheep and poultry.
Pandemic	Pan, pandemos	epidemic diseases that cover several countries and are widespread among people. For example, influenza, AIDS, etc.
Panzootic	panzoonosis	the most intense high level of the epizootic process is the epizootic of animal diseases that covers several countries and continents. For example, protein, rinderpest.
Parainfection	Para infection	an infection caused by a change in the characteristics of another microorganism under the influence of a group of microbes
Passage	passage	animals susceptible to them with microorganisms and viruses, chicken embryos and cultured cells with pathogens serial infection. P. creates an opportunity to keep viruses clean, isolate them, increase

		their number and keep their activity constant.
Prolongation	extension	extend the duration of the effect. In most cases, it is a phrase that expresses the prolongation of the effect of drugs on the body. Various adjuvants and polymers are used for this purpose.
Ribonucleic acid	RNA	RNA) is a polymeric substance, the large molecule of which consists of polynucleotide helical chains. RNA is included in the cell cytoplasm and nucleus.
Vesicle	vesicle	one of the first morphological elements of skin rashes; a blister formed by the accumulation of exudate (fluid) in the outer layer of the skin (epidermis)
Vaccine	vaccine	is a special biological preparation, prepared from pathogens. It is mainly used to prevent disease.
Hematogenous	haematiogenes	spread of the pathogen through the blood.
Hemolysis	hymalysis	disintegration of erythrocytes in the blood and release of hemoglobin into the external environment.
Hemolysins	haemolysin	substances (antibodies) that cause the release of hemoglobin from red blood cells (erythrocytes), that is, cause hemolysis
Herpes viruses	herpes	a family of DNA viruses that replicate in the cell nucleus, many of which cause latent disease.
Hydrophobia	hydorphobos	fear, fear of water) - fear of water, such a sign is observed in human and animal rabies.
Abortive	abortivus	short duration of the disease and passing in a mild form
Absorption	absorber	absorption of a substance in a gas, light or liquid environment into the entire volume of an absorbing body (absorbent) as a physical process.

Aftar	epithelium	(ulcers) are small areas of dead (necrosis) epithelium of mucous membranes. It mainly appears on the mucous membranes of the oral cavity.
Agglutination	a gglutinatio	corpuscular particles - viruses, bacteria, erythrocytes, leukocytes, thrombocytes, tissue cells, corpuscular chemically active particles sticking together and sedimenting under the influence of antibodies - agglutinins formed against them.
Aggressiveness	Aggressive	aggression occurs, for example, in psychopathic cases.
Bacteriophage	Bacteriophage	A virus capable of infecting a bacterial cell, multiplying, and releasing phage particles into the bacterial environment by dissolving this process.
Disinsection	Disinsection	Arthropod control measures.
Bronchitis _	Bronchitis	Inflammation of the bronchi (macro and micro bronchitis
Symptom	Symptoms	Functional and morphological changes in the body under the influence of pathogenic factors i
Disinfection	Disinfection	destruction of viruses causing infectious diseases by physical and chemical action
Deoxyribonucleic acids	(DNA)	Deoxyribose nucleic acids are present in every cell, viruses, and microorganisms that store DNA.
Epizootic	Epizootic	widespread spread of any infectious disease.

Veterinary-sanitary regulations	Veterinary sanitary rules	It is a set of sanitary standards and requirements that must be fulfilled by livestock farms and other organizations, and is the core of measures aimed at protecting animals from infectious and parasitic diseases and producing livestock products of high sanitary quality.
Gen	Gen	They are special parts (loci) of chromosomes differentiated along their length and are the simplest units of heredity.
Embryo	Embryo	A developing organism (fetus) in the period from the crushing of the zygote to the end of ontogeny.
E. Epithelius	epithelium	tissue covering the surface of the skin and the organs whose cavity is in contact with the external environment. It limits the internal environment of the organism from the external environment. Between the epithelium and the underlying connective tissue lies the basement membrane. The fact that it is composed only of cells and does not penetrate blood vessels is one of the characteristic features of the epithelium.
Isobar	isobar	Elements with different sequence numbers and the same atomic mass-weight: 19 K-40, 20 Sa-40.
Diet	Diet	Therapeutic feeding, use of food for the purpose of treatment.
Ionization	Ionization	The formation of ions as a result of the destruction of electrons in an atom and the excitation and disintegration of atomic molecules.
Embryo _	Embryo	A developing organism (fetus) during the period from the crushing of the zygote until the end of organogenesis.
Ionization	Ionization	This is the separation of electrons from the atom and the formation of ions as a result of the excitation and disintegration of atomic molecules. to swallow, to absorb, to absorb.
Tropism	Tropism	is that a certain isotope, an element, likes a

		certain place. Urotropin drug (uro-urinary tropus-yul), which means that this drug affects the urinary tract.
Register now _	Registry	Calculation of the types, source and radiation dose of ionizing rays.
Atrophy	Atrophy	Shrinkage and weakening of body cells, tissues and organs.
Dose	Dose	Substance mass to the unit recoverable energy _ quantity.
Dedifferentia t sia	Dedifferentiation	The return of specialized cells to an immature state, losing their characteristic features.
Diagnosis	Diagnosis	A summary of the doctor's information about the essence of the disease and the condition of the sick animal in the form of current modern terms.
Hemoglobin	Hemoglobin	A complex protein belonging to the group of chromoproteins contained in erythrocytes, gives oxygen to the cells and acquires the properties of oxidation and reduction.
Gen	Gene	They are special parts (loci) of chromosomes differentiated along their length and are the simplest units of heredity.
Appetite	Appetite	Feeling the need to eat.
Capillary	Capillaries	Blood and lymph vessels of microscopic size. Their walls consist of endothelial cells, basement membrane and adventitial cells, capillaries actively participate in the control of substance transport and exchange.
X is a joy	Cell	It is a whole elemental living system consisting of two closely related parts - cytoplasm and nucleus, surrounded by plasmolemma, and [is] the basis of the structure, development and life activity of plant and animal organisms.
Ra t sion	ration	The composition of feed that meets the daily needs of animals in terms of nutrients.
Symptomatics	Symptomatology	As an auxiliary method to the pathogenetic method, it is used for the purpose of eliminating some symptoms of the disease and improving the condition of the sick animal. Examples of symptomatic therapy include the use of expectorant, antipyretic,

		pain reliever, cardiac and other drugs, physiotherapy and operative methods.

Questions for attestations held in science.

1-Oral questions for intermediate control .

1. How many different forms of rabies, laboratory diagnosis.
2. Toxic fungi and environments for their reproduction.
3. Understanding of special prevention.
4. Presidential decrees on ways to develop livestock breeding in the market economy.
5. General and special preventive measures in anthrax.
6. Mention measures to eliminate brucellosis.
7. Swine plague (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
8. Types of antibodies.
9. Types of immunity, immunological reactivity.
10. Diagnosis of brucellosis.
11. Ways of transmission of infectious diseases to healthy animals.
12. Bacteriological examination methods.
13. Treatment of iron deficiency.
14. The importance of microorganisms and viruses in the formation of infection and their pathogenicity.
15. Brucellosis control measures.
16. Diagnostic measures carried out in pig serum.
17. The role of serological diagnostic methods in the fight against infectious diseases.
18. Protection of people from zooanthropanous diseases.
19. Concept of immunity and its types.
20. Determination of protein diseases in laboratory conditions.
21. Enterotoxemia disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
22. Special prevention of infectious diseases.
23. Importance of formation and development of infection by microorganisms and viruses.
24. Antigens and their immunogenicity.
25. General and special preventive measures of protein disease.
26. Mange disease of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
27. Clinical signs of Bratzot's disease.
28. Diagnosis of swine scurvy.
29. Organization of treatment.

30. Mutism of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, prevention, and countermeasures).

31. Necrobacteriosis and its countermeasures.

32. What is the difference between communicable diseases and non-communicable diseases?

33. Epizootology of anthrax.

34. Epizootic, panzootic, sporadic, epidemic, pandemic.

35. Explain quarantine, restriction, isolators?

36. Rules for sending material to the laboratory.

37. African swine fever (economic damage, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

38. Manka disease (economic damage, epizootic chain, clinical symptoms, immunity, pathological changes, diagnosis, preventive measures).

39. Means and measures against epizootics.

40. Paratuberculosis disease of cattle (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

41. Smallpox and measures against it.

42. Diagnosis of infectious rhinotracheitis.

43. Vaccines and their types.

44. Anthrax disease (etiology, epizootic, chain pathogenesis, clinical symptoms, immune pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

45. Immunity and their types.

46. Epizootic process and its driving forces.

47. Infectious Disease Study Template.

48. Necrobacteriosis (clinical symptoms, pathological changes, diagnosis, differential diagnosis, treatment, immunity, preventive measures).

49. Ways of separation of pathogens from the body.

50. Avian flu disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

51. Infectious anemia disease of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

52. Comment on serological reactions?

53. Carnivore pest control measures.

54. Immune mechanism and factors.

55. Peculiarities of epizootology of protein disease.

56. Colibacteriosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

57. Disinfection and its types.

58. Explain the epizootic process?

59. Transmissive and vertical (from generation to generation, transovarial transmission) of pathogens.

60. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

61. Rhinopneumonia of horses (etiology, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment)

62. Measures to prevent protein disease from being imported from other countries.

63. Explain diseases transmitted by insects?

64. Susceptible animals are the driving forces of the epizootic process.

65. Formation of active and passive immunity.

66. Reasons for long storage of anthrax foci.

67. Camel plague and its social risk.

68. Explain the methods of virological testing?

69. Specific characteristics of anaerobic infections.

70. Pasteurellosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

71. General and special prevention of smallpox.

72. Explain viral diarrhea disease.

73. Pathogenesis and laboratory diagnosis of rabies.

74. Economic damage of infectious diseases and importance of anti-epizootic measures.

75. Campliobacteriosis of cattle.

76. Pullorosis disease of poultry (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

77. How to control the source of the pathogen.

78. Tuberculosis epizootology.

79. Importance of macroorganism and external environment in the development of infection.

80. Understanding of vaccines and their importance in preventing infection.

81. Goat pleuropneumonia (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

82. The problem of prevention of the disease of the blackhead.

83. Epizootics and measures against it.
84. Actions directed against the transmission mechanism of the pathogen.
85. Pathogenesis and diagnosis of brucellosis.
86. Etiology, epizootology and methods of laboratory investigation of leptospirosis.
87. Specific and non-specific prevention of pasteurellosis.
88. Explain the methods of physical disinfection.
89. Disinfection and its types.
90. Explain the epizootic process?
91. Transmissive and vertical (from generation to generation, transovarial transmission) of pathogens.
92. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
93. Rhinopneumonia of horses (etiology, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment)
94. Measures to prevent protein disease from being imported from other countries.
95. Explain diseases transmitted by insects?
96. Susceptible animals are the driving forces of the epizootic process.
97. Formation of active and passive immunity.
98. Reasons for long storage of anthrax foci.
99. Camel plague and its social risk.
100. Explain the methods of virological examination?
101. Specific characteristics of anaerobic infections.
102. Pasteurellosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
103. General and special prevention of smallpox.
104. Explain viral diarrhea disease.
105. Rabies pathogenesis and laboratory diagnosis.
106. Economic damage of infectious diseases and importance of anti-epizootic measures.
107. Campliobacteriosis of cattle.
108. Pullorosis disease of birds (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
109. How to combat the source of the pathogen.
110. Tuberculosis epizootology.
111. Importance of macroorganism and external environment bodies in the formation of infection.
112. Understanding of vaccines and their importance in preventing infection.

113. Pleuropneumonia of goats (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

114. The problem of prevention of rabies disease.

115. Epizootic and measures against it.

116. Actions directed against the transmission mechanism of the pathogen.

117. Pathogenesis and diagnosis of brucellosis.

118. Leptospirosis etiology, epizootology and laboratory testing methods.

119. Specific and non-specific prevention of pasteurellosis.

120. Comment on physical disinfection methods.

Oral questions for midterm assessment .

1. The role of disinsection in the elimination of transmissible diseases.
2. Listeriosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
3. Laboratory diagnosis of leukemia.
4. The fight against anthrax.
5. Laboratory diagnosis of brucellosis.
6. Economic damage of infectious diseases.
7. Infectious bronchitis disease of birds (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
8. Infectious hepatitis disease of meat-eating animals (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
9. Explain active and passive immunity?
10. Count the links of the epizootic chain?
11. Concept of epizootic, panzootic, sporadic, epidemic, pandemic.
12. Content, essence of sensitization and desensitization.
13. Explain general and specific treatments for infectious diseases.
14. Infectious mastitis disease of sheep (economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
15. Mange disease in horses (etiology, economic damage, clinical symptoms, pathanatomical changes, diagnosis, preventive measures).
16. Epizootic process and its driving forces.
17. Measles and measures to combat it.
18. Measures to prevent parainfluenza-3 disease.

19. Rules for obtaining pathological material in infectious diseases and sending it to the laboratory.
20. Poultry mycoplasmosis disease (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
21. General and special prevention of infectious diseases.
22. Avian flu disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
23. Measures against pasteurellosis.
24. Epizootology of mange disease of horses.
25. Explain about hapten?
26. Types of infectious diseases (zooanthroponosis diseases, zoonotic diseases, highly dangerous infectious diseases).
27. Cattle viral diarrhea disease (etiology, economic damage, epizootic chain pathogenesis, clinical signs, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).
28. Understanding of the use of hyperimmune blood sera, types.
29. Types of deratization, mechanism of action, quality assessment.
30. Protein disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).
31. Concept of epizootic, panzootic, sporadic epidemic, pandemic.
32. Explain the laboratory diagnosis of viral and infectious diseases?
33. Explain general and specific treatments for infectious diseases?
34. Infectious mastitis disease of sheep (economic damage, epizootic chain pathogenesis, clinical symptoms, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).
35. Mange disease in horses (etiology, economic damage, clinical signs, pathanatomical changes, diagnosis, treatment, preventive measures).
36. Forms of transmission of infectious diseases.
37. Infectious mastitis of sheep.
38. Diagnosis and epizootology of horse dumb disease.
39. Clinical forms of bovine infectious rhinotracheitis.
40. What is tolerance?
41. Epizootological classification of infectious diseases.
42. General and special prevention of iron deficiency disease.
43. Pasteurellosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
44. Phases of epizootics.

45. Epizootology of infectious laryngo-tracheitis of birds.
46. Determining the quality of disinfection.
47. Parainfluenza-3 disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
48. Eczema disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
49. Activities held in the risk area for protein disease.
50. Explain the bacteriological diagnosis?
51. Importance of microorganisms and viruses in the formation of infection.
52. Marek's disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
53. Sheep chlamydiosis and its control measures.
54. Cattle plague (etiology, epizootology, clinic, course, diagnosis, preventive and countermeasures).
55. Deratization and its importance.
56. Explain the diseases that are forbidden to treat?
57. The role of the science of epizootology in the training of a veterinary doctor.
58. Smallpox (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
59. Epizootology of leukemia.
60. Explain panzootic diseases?
61. Comment on the carrier status of pathogens?
62. Teshen's disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
63. Anthrax diagnosis and prevention.
64. Explain the pathogen reservoir?
65. List the diseases that can be transmitted from humans to animals and how it occurs.
66. The relationship of epizootology with other sciences.
67. Rules of procedure for working with infectious animals.
68. Explain the technique of tuberculinization?
69. Name the vaccines used for preventive purposes in protein disease?
70. Poultry infectious bronchitis disease (etiology, epizootology, economic damage, pathogenesis, immunity, clinical symptoms, pathanotomy, diagnosis, differential diagnosis, treatment, preventive measures).
71. How do you explain the government's strong focus on infectious diseases?

72. Infectious anemia disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, diagnosis, treatment, preventive and countermeasures).
73. Swine fever.
74. Goat pleuropneumonia and its control measures.
75. Comment on chemical disinfectants?
76. Explain antigens and haptens?
77. Paratuberculosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
78. Ways of transmission of infectious disease agents.
79. What measures are used against wild animals in the fight against rabies?
80. Mechanization of disinfection works.
81. The role of veterinary-sanitary measures in disease prevention.
82. Poultry Marek's disease (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
83. Diagnosis of smallpox, differential diagnosis.
84. Epizootology of protein disease.
85. Explain allergy diagnosis?
86. What is a vaccine, serum, hyperimmune serum, convalescent blood, and its use.
87. Explain the types of disinfection?
88. Explain the schematic template for the study of infectious diseases?
89. Bia salmonellosis, diarrhea (abortion) disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
90. Protein disease (etiology, economic damage, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
91. The relationship of the science of epizootology with other sciences.
92. Laboratory diagnosis of anthrax.
93. Tuberculosis pathogenesis and diagnostics.
94. Prevention of leukemia.
95. The role of veterinary-sanitary measures in the prevention of infection and elimination of the disease.
96. The role of bacteriological examination in the detection of infectious diseases.
97. Infectious laryngotracheitis disease of birds.
98. Plague of carnivores (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

99. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

100. Understanding anaphylaxis.

101. Application of immunology in practice.

102. General and special preventive measures of rabies.

103. Salmonellosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

104. Mechanization of disinfection measures.

105. Non-specific features of immunity.

106. Explain the nature of spread of infectious diseases in the form of sporadic, enzootic, epizootic and panzootic.

107. Diagnosis of rabies.

108. Measures to prevent infectious rhinotracheitis.

109. Types of allergy diagnosis.

110. Epizootology of leukemia.

111. Diseases transmitted by rodents and their control measures.

112. What is an epizootic focus and its definition.

113. Epizootology of bird flu disease.

114. Epizootology of protein disease.

115. Explain soil infectious diseases.

116. Humoral factors of immunity.

117. Allergic and serological diagnosis of mange disease in horses.

118. Clinical signs of cattle plague.

119. Clinical signs of anthrax.

120. In which diseases is toxicoinfection present?

Oral questions for final assessment

1. Types of infection and their economic damage to the economy.

2. Anti-tuberculosis measures.

3. Camel plague (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

4. Allergy and allergic diagnosis.

5. Measures to prevent leukemia.

6. The essence of the epizootic process.

7. Anti-tuberculosis measures.

8. Explain fully the bacteriological examination in the detection of infectious diseases.

9. The importance of phagocytosis in infectious diseases.
10. Diagnosis of leukemia.
11. Concept of infection.
12. List the modern diagnostic procedures performed in tuberculosis.
13. Diagnosis of protein disease, differential diagnosis.
14. Ways of transmission of the causative agent from one animal to another.
15. List the causative agents of salmonellosis by animal and poultry species.
16. History of the development of the science of epizootology.
17. Prophylactic means of tuberculosis (general and special).
18. General and special preventive measures of Ayeski's disease.
19. Infection and its types
20. Differences between viruses and bacteria
21. Importance of preventive measures in the national economy.
22. Diagnosis of tuberculosis.
23. Smallpox diagnosis, differential diagnosis, preventive measures.
24. Methods of reproduction of anaerobic microorganisms and the environments they need.
25. Methods of virus detection in pathological material.
26. The role of epizootology in the training of a veterinary doctor.
27. Anthrax control measures.
28. Listeriosis diagnosis, differential diagnosis, prevention.
29. Immunoglobulins, their types and importance in infectious diseases.
30. The causative agent of iron disease.
31. Decisions of the Cabinet of Ministers on ways to develop animal husbandry in the market economy.
32. Detection of anthrax in laboratory conditions.
33. How many different forms of rabies, laboratory diagnosis.
34. Toxic fungi and environments for their reproduction.
35. Understanding of special prevention.
36. Presidential decrees on ways to develop livestock breeding in the market economy.
37. General and special preventive measures in anthrax.
38. Mention measures to eliminate brucellosis.
39. Swine plague (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
40. Types of antibodies.
41. Types of immunity, immunological reactivity.
42. Diagnosis of brucellosis.
43. Ways of transmission of infectious diseases to healthy animals.

44. Bacteriological examination methods.

45. Treatment of iron deficiency.

46. The importance of microorganisms and viruses in the formation of infection and their pathogenicity.

47. Brucellosis control measures.

48. Diagnostic measures carried out in pig serum.

49. The role of serological diagnostic methods in the fight against infectious diseases.

50. Protection of people from zooanthropanous diseases.

51. Concept of immunity and its types.

52. Determination of protein diseases in laboratory conditions.

53. Enterotoxemia disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

54. Special prevention of infectious diseases.

55. Importance of formation and development of infection by microorganisms and viruses.

56. Antigens and their immunogenicity.

57. General and special preventive measures of protein disease.

58. Mange disease of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

59. Clinical signs of Bratzot's disease.

60. Diagnosis of swine scurvy.

61. Organization of treatment.

62. Mutism of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, prevention, and countermeasures).

63. Necrobacteriosis and its countermeasures.

64. What is the difference between communicable diseases and non-communicable diseases?

65. Epizootology of anthrax.

66. Epizootic, panzootic, sporadic, epidemic, pandemic.

67. Explain quarantine, restriction, isolators?

68. Rules for sending material to the laboratory.

69. African swine fever (economic damage, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

70. Manka disease (economic damage, epizootic chain, clinical symptoms, immunity, pathological changes, diagnosis, preventive measures).

71. Means and measures against epizootics.

72. Paratuberculosis disease of cattle (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

73. Smallpox and measures against it.

74. Diagnosis of infectious rhinotracheitis.

75. Vaccines and their types.

76. Anthrax disease (etiology, epizootic, chain pathogenesis, clinical symptoms, immune pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

77. Immunity and their types.

78. Epizootic process and its driving forces.

79. Infectious Disease Study Template.

80. Necrobacteriosis (clinical symptoms, pathological changes, diagnosis, differential diagnosis, treatment, immunity, preventive measures).

81. Ways of separation of pathogens from the body.

82. Avian flu disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

83. Infectious anemia disease of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

84. Comment on serological reactions?

85. Carnivore pest control measures.

86. Immune mechanism and factors.

87. Peculiarities of epizootology of protein disease.

88. Colibacteriosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

89. Disinfection and its types.

90. Explain the epizootic process?

91. Transmissive and vertical (from generation to generation, transovarial transmission) of pathogens.

92. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

93. Rhinopneumonia of horses (etiology, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment)

94. Measures to prevent protein disease from being imported from other countries.

95. Explain diseases transmitted by insects?

96. Susceptible animals are the driving forces of the epizootic process.

97. Formation of active and passive immunity.
98. Reasons for long storage of anthrax foci.
99. Camel plague and its social risk.
100. Explain the methods of virological testing?
101. Specific characteristics of anaerobic infections.
102. Pasteurellosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
103. General and special prevention of smallpox.
104. Explain viral diarrhea disease.
105. Pathogenesis and laboratory diagnosis of rabies.
106. Economic damage of infectious diseases and importance of anti-epizootic measures.
107. Campliobacteriosis of cattle.
108. Pullorosis disease of poultry (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
109. How to control the source of the pathogen.
110. Tuberculosis epizootology.
111. Importance of macroorganism and external environment in the development of infection.
112. Understanding of vaccines and their importance in preventing infection.
113. Goat pleuropneumonia (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
114. The problem of prevention of the disease of the blackhead.
115. Epizootics and measures against it.
116. Actions directed against the transmission mechanism of the pathogen.
117. Pathogenesis and diagnosis of brucellosis.
118. Etiology, epizootology and methods of laboratory investigation of leptospirosis.
119. Specific and non-specific prevention of pasteurellosis.
120. Explain the methods of physical disinfection.
121. The role of disinsection in the elimination of transmissible diseases.
122. Listeriosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
123. Laboratory diagnosis of leukemia.
124. The fight against anthrax.
125. Laboratory diagnosis of brucellosis.
126. Economic damage of infectious diseases.

127. Infectious bronchitis disease of birds (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

128. Infectious hepatitis disease of meat-eating animals (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

129. Explain active and passive immunity?

130. Count the links of the epizootic chain?

131. Concept of epizootic, panzootic, sporadic, epidemic, pandemic.

132. Content, essence of sensitization and desensitization.

133. Explain general and specific treatments for infectious diseases.

134. Infectious mastitis disease of sheep (economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

135. Mange disease in horses (etiology, economic damage, clinical symptoms, pathanatomical changes, diagnosis, preventive measures).

136. Epizootic process and its driving forces.

137. Measles and measures to combat it.

138. Measures to prevent parainfluenza-3 disease.

139. Rules for obtaining pathological material in infectious diseases and sending it to the laboratory.

140. Poultry mycoplasmosis disease (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

141. General and special prevention of infectious diseases.

142. Avian flu disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

143. Measures against pasteurellosis.

144. Epizootology of mange disease of horses.

145. Explain about hapten?

146. Types of infectious diseases (zooanthroponosis diseases, zoonotic diseases, highly dangerous infectious diseases).

147. Cattle viral diarrhea disease (etiology, economic damage, epizootic chain pathogenesis, clinical signs, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).

148. Understanding of the use of hyperimmune blood sera, types.

149. Types of deratization, mechanism of action, quality assessment.

150. Protein disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).

151. Concept of epizootic, panzootic, sporadic epidemic, pandemic.

152. Explain the laboratory diagnosis of viral and infectious diseases?

153. Explain general and specific treatments for infectious diseases?

154. Infectious mastitis disease of sheep (economic damage, epizootic chain pathogenesis, clinical symptoms, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).

155. Mange disease in horses (etiology, economic damage, clinical signs, pathanatomical changes, diagnosis, treatment, preventive measures).

156. Forms of transmission of infectious diseases.

157. Infectious mastitis of sheep.

158. Diagnosis and epizootology of horse dumb disease.

159. Clinical forms of bovine infectious rhinotracheitis.

160. What is tolerance?

161. Epizootological classification of infectious diseases.

162. General and special prevention of iron deficiency disease.

163. Pasteurellosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

164. Phases of epizootics.

165. Epizootology of infectious laryngo-tracheitis of birds.

166. Determining the quality of disinfection.

167. Parainfluenza-3 disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

168. Eczema disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

169. Activities held in the risk area for protein disease.

170. Explain the bacteriological diagnosis?

171. Importance of microorganisms and viruses in the formation of infection.

172. Marek's disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

173. Sheep chlamydiosis and its control measures.

174. Cattle plague (etiology, epizootology, clinic, course, diagnosis, preventive and countermeasures).

175. Deratization and its importance.

176. Explain the diseases that are forbidden to treat?

177. The role of the science of epizootology in the training of a veterinary doctor.
178. Smallpox (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
179. Epizootology of leukemia.
180. Explain panzootic diseases?
181. Comment on the carrier status of pathogens?
182. Teshen's disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
183. Anthrax diagnosis and prevention.
184. Explain the pathogen reservoir?
185. List the diseases that can be transmitted from humans to animals and how it occurs.
186. The relationship of epizootology with other sciences.
187. Rules of procedure for working with infectious animals.
188. Explain the technique of tuberculinization?
189. Name the vaccines used for preventive purposes in protein disease?
190. Poultry infectious bronchitis disease (etiology, epizootology, economic damage, pathogenesis, immunity, clinical symptoms, pathanotomy, diagnosis, differential diagnosis, treatment, preventive measures).
191. How do you explain the government's strong focus on infectious diseases?
192. Infectious anemia disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, diagnosis, treatment, preventive and countermeasures).
193. Swine fever.
194. Goat pleuropneumonia and its control measures.
195. Comment on chemical disinfectants?
196. Explain antigens and haptens?
197. Paratuberculosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
198. Ways of transmission of infectious disease agents.
199. What measures are used against wild animals in the fight against rabies?
200. Mechanization of disinfection works.
201. The role of veterinary-sanitary measures in disease prevention.
202. Poultry Marek's disease (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
203. Diagnosis of smallpox, differential diagnosis.

204. Epizootology of protein disease.
205. Explain allergy diagnosis?
206. What is a vaccine, serum, hyperimmune serum, convalescent blood, and its use.
207. Explain the types of disinfection?
208. Explain the schematic template for the study of infectious diseases?
209. Bia salmonellosis, diarrhea (abortion) disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
210. Protein disease (etiology, economic damage, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
211. The relationship of the science of epizootology with other sciences.
212. Laboratory diagnosis of anthrax.
213. Tuberculosis pathogenesis and diagnostics.
214. Prevention of leukemia.
215. The role of veterinary-sanitary measures in the prevention of infection and elimination of the disease.
216. The role of bacteriological examination in the detection of infectious diseases.
217. Infectious laryngotracheitis disease of birds.
218. Pestilence of carnivores (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
219. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
220. Understanding anaphylaxis.
221. Application of immunology in practice.
222. General and special preventive measures of rabies.
223. Salmonellosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
224. Mechanization of disinfection measures.
225. Non-specific features of immunity.
226. Explain the nature of spread of infectious diseases in the form of sporadic, enzootic, epizootic and panzootic.
227. Diagnosis of rabies.
228. Measures to prevent infectious rhinotracheitis.
229. Types of allergy diagnosis.

230. Epizootology of leukemia.
231. Diseases transmitted by rodents and their control measures.
232. What is an epizootic focus and its definition.
233. Epizootology of bird flu disease.
234. Epizootology of protein disease.
235. Explain soil infectious diseases.
236. Humoral factors of immunity.
237. Allergic and serological diagnosis of mange disease in horses.
238. Clinical signs of cattle plague.
239. Clinical signs of anthrax.
240. In which diseases is toxicoinfection present?
241. Protective features of the organism's normative microflora.
242. Diagnosis and epizootology of leptospirosis.
243. Clinical signs in pigs.
244. Clinical signs of Aujeski's disease.
245. What is the difference between an infectious disease and a non-infectious disease?
246. Understanding of epizootological investigation methods.
247. Methods of treatment of infectious diseases.
248. Brucellosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).
249. Preventive measures in rabies.
250. Listeriosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).
251. Epizootic focus, understanding of the mechanism of disease spread.
252. Newcastle disease and its countermeasures.
253. The importance of disinfection, disinsection and deratization measures in the elimination of infectious diseases.
254. Epizootology of rabies.
255. Briefly explain panzootic diseases?
256. Specific features of immunity formed against viral diseases.
257. Explain the essence of allergy diagnosis.
258. Forms of clinical manifestations of infectious rhinotracheitis.
259. The role of bulls in the spread of campylobacteriosis.
260. Actions against bovine leukemia.
261. Measures to prevent infectious diseases.
262. Methods of testing infectious diseases in the laboratory.

263. Peculiarities of diagnosis of infectious diseases.

264. Infectious pleuropneumonia disease of goats (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

265. Aujeski's disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

266. Understanding infection and immunity.

267. Explain the epizootic process?

268. Manka disease and measures to combat it.

269. Cattle plague (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

270. Infectious gastroenteritis of pigs (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

271. Rabies disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

272. Understanding of secondary infection, colostral immunity.

273. Explain allergy diagnosis?

274. Infectious atrophic rhinitis disease of pigs (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

275. Aujeski's disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

276. The role of deratization in the fight against infectious diseases.

277. Prophylactic means of tuberculosis (general and special).

278. Leptospirosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).

279. The role of wild animals in the spread of rabies.

280. Comment on serological diagnosis?

281. Concept of infection.

282. Infectious laryngotracheitis disease (causing agent, epizootology, economic damage, pathogenesis, clinical signs, course, pathology, general and special treatment, preventive measures).

283. Infectious hepatitis disease of carnivores (causing agent, epizootology, economic damage, pathogenesis, clinical symptoms, course, pathology, general and special treatment, preventive measures).

284. Infectious mastitis disease of sheep (causing agent, epizootology, economic damage, pathogenesis, clinical signs, course, pathology, general and special treatment, preventive measures).

285. Epizootology of anthrax.

286. Insulators and their requirements.

287. Describe zooanthroponous diseases?

288. Tell the laboratory diagnosis of brucellosis?

289. Explain the health of the economy from tuberculosis?

290. Poultry YLT (infectious laryngotracheitis) disease (etiology, economic damage, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

291. Enumerate the measures used against epizootics.

292. Explain primary, secondary infection, mixed infection.

293. Horses' INAN disease and measures to combat it.

294. Swine flu disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

295. Black disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

296. Classification of epizootological classification of infectious diseases.

297. The difference between infectious diseases and non-infectious diseases.

298. Enterotoxemia (etiology, economic damage, pathogenesis, clinical signs, course, pathological changes, diagnosis, differential diagnosis, preventive measures).

299. Diagnosis of Newcastle disease in poultry and measures to prevent it.

300. Salmonellosis of young animals (etiology, economic damage, pathogenesis, clinical symptoms, course, pathological changes, diagnosis, differential diagnosis, preventive measures).

1-Written work questions for midterm assessment.

1. The relationship of the science of epizootology with other sciences.
2. Laboratory diagnosis of anthrax.
3. Tuberculosis pathogenesis and diagnostics.
4. Prevention of leukemia.
5. The role of veterinary-sanitary measures in the prevention of infection and elimination of the disease.

6. The role of bacteriological examination in the detection of infectious diseases.
7. Infectious laryngotracheitis disease of birds.
8. Plague of carnivores (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
9. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
10. Understanding anaphylaxis.
11. Application of immunology in practice.
12. General and special preventive measures of rabies.
13. Salmonellosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
14. Mechanization of disinfection measures.
15. Non-specific features of immunity.
16. Explain the nature of spread of infectious diseases in the form of sporadic, enzootic, epizootic and panzootic.
17. Diagnosis of rabies.
18. Measures to prevent infectious rhinotracheitis.
19. Types of allergy diagnosis.
20. Epizootology of leukemia.
21. Diseases transmitted by rodents and their control measures.
22. What is an epizootic focus and its definition.
23. Epizootology of bird flu disease.
24. Epizootology of protein disease.
25. Explain soil infectious diseases.
26. Humoral factors of immunity.
27. Allergic and serological diagnosis of mange disease in horses.
28. Clinical signs of cattle plague.
29. Clinical signs of anthrax.
30. In which diseases is toxicoinfection present?
31. Protective features of the organism's normative microflora.
32. Diagnosis and epizootology of leptospirosis.
33. Clinical signs in pigs.
34. Clinical signs of Aujeski's disease.
35. What is the difference between an infectious disease and a non-infectious disease?
36. Understanding of epizootological investigation methods.
37. Methods of treatment of infectious diseases.

38. Brucellosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).

39. Preventive measures in rabies.

40. Listeriosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).

41. Epizootic focus, understanding of the mechanism of disease spread.

42. Newcastle disease and its countermeasures.

43. The importance of disinfection, disinsection and deratization measures in the elimination of infectious diseases.

44. Epizootology of rabies.

45. Briefly explain panzootic diseases?

46. Specific features of immunity formed against viral diseases.

47. Explain the essence of allergy diagnosis.

48. Forms of clinical manifestations of infectious rhinotracheitis.

49. The role of bulls in the spread of campylobacteriosis.

50. Actions against bovine leukemia.

51. Measures to prevent infectious diseases.

52. Methods of testing infectious diseases in the laboratory.

53. Peculiarities of diagnosis of infectious diseases.

54. Infectious pleuropneumonia disease of goats (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

55. Aujeski's disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

56. Understanding infection and immunity.

57. Explain the epizootic process?

58. Manka disease and measures to combat it.

59. Cattle plague (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

60. Infectious gastroenteritis of pigs (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

61. Rabies disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

62. Understanding of secondary infection, colostral immunity.

63. Explain allergy diagnosis?

64. Infectious atrophic rhinitis disease of pigs (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

65. Aujeski's disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

66. The role of deratization in the fight against infectious diseases.

67. Prophylactic means of tuberculosis (general and special).

68. Leptospirosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).

69. The role of wild animals in the spread of rabies.

70. Comment on serological diagnosis?

71. Concept of infection.

72. Infectious laryngotracheitis disease (causing agent, epizootology, economic damage, pathogenesis, clinical signs, course, pathology, general and special treatment, preventive measures).

73. Infectious hepatitis disease of carnivores (causing agent, epizootology, economic damage, pathogenesis, clinical symptoms, course, pathology, general and special treatment, preventive measures).

74. Infectious mastitis disease of sheep (causing agent, epizootology, economic damage, pathogenesis, clinical signs, course, pathology, general and special treatment, preventive measures).

75. Epizootology of anthrax.

76. Insulators and their requirements.

77. Describe zooanthroponous diseases?

78. Tell the laboratory diagnosis of brucellosis?

79. Explain the health of the economy from tuberculosis?

80. Poultry YLT (infectious laryngotracheitis) disease (etiology, economic damage, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

81. Enumerate the measures used against epizootics.

82. Explain primary, secondary infection, mixed infection.

83. Horses' INAN disease and measures to combat it.

84. Swine flu disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

85. Black disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

86. Classification of epizootological classification of infectious diseases.

87. The difference between infectious diseases and non-infectious diseases.

88. Enterotoxemia (etiology, economic damage, pathogenesis, clinical signs, course, pathological changes, diagnosis, differential diagnosis, preventive measures).

89. Diagnosis of Newcastle disease in poultry and measures to prevent it.

90. Salmonellosis of young animals (etiology, economic damage, pathogenesis, clinical symptoms, course, pathological changes, diagnosis, differential diagnosis, preventive measures).

91. The nature of antibodies.

92. Cattle plague (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

93. Explain the diseases that can be transmitted from humans to animals and their prevention measures.

94. Anthrax control measures.

95. Veterinary-sanitary measures in the fight against infectious diseases.

96. Horse flu disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

97. State the purpose of AR, KBR, Rosbengal reaction.

98. Content, essence of sensitization, desensitization.

99. Protein disease (etiology, economic damage, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

100. Epizootology of Karasan disease.

101. The difference between infectious diseases and non-infectious diseases.

102. The subject and task of the science of epizootology.

103. Bradzot's disease (economic damage, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

104. Camel plague and measures to combat it.

105. Leptospirosis prevention measures (general and special)

106. Explain allergy diagnosis?

107. Understanding of disinsection methods.

108. Concept of epizootology, panzootic, sporadic, epidemic, pandemic.

109. Swine flu (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

110. Infectious pleuropneumonia disease of goats (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

111. Epizootological process and its driving forces.

112. Methods of epizootic investigation.

113. Measures to prevent infectious diseases.

114. Infectious vaginitis disease of cattle (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

115. Campylobacteriosis (virus) disease (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

116. Special treatment against epizootics)

117. Types of disinfection, deratization, disinsection (sterkushka activities.

118. Treatment of infectious diseases (general and

119. ilization, pasteurization, tindalization).

120. Salmonellosis disease of young animals (causing agent, economic damage, epizootology, pathogenesis, clinical signs and course, pathology, general and special treatment, preventive measures).

121. Avian flu disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

122. What is a vaccine, serum, hyperimmune serum, convalescent blood, its use.

123. Tell about the types of disinfection?

124. Schematic template for the study of infectious diseases.

125. Biyalar salmonellosis diarrheal disease (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

126. protein disease (etiology, economic damage, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

127. Tasks of the science of epizootology.

128. The importance of vaccination in the prevention of infectious diseases.

129. Prevention of Newcastle disease in poultry.

130. Diagnosis of brucellosis.

131. How does the infectious process manifest itself?

132. Poultry scurvy disease (causing agent, economic damage, epizootology, pathogenesis, clinical signs and course, pathology, general and special treatment, preventive measures).

133. Methods of treatment of infectious diseases.
134. Immunity and its types
135. Paratuberculosis disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
136. Smallpox (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
137. Types and quality of disinsection.
138. Non-specific prevention of paratuberculosis.
139. Diagnosis and general prevention of campylobacteriosis.
140. Diagnosis of equine epizootic lymphangitis.
141. Ways of transmission of infectious disease agents.
142. What measures are used against wild animals in the fight against rabies?
143. Mechanization of disinfection works.
144. The role of veterinary-sanitary measures in disease prevention.
145. Poultry Marek's disease (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
146. Diagnosis of smallpox, differential diagnosis.
147. Epizootology of protein disease.
148. Explain allergy diagnosis?
149. What is a vaccine, serum, hyperimmune serum, convalescent blood, and its use.
150. Explain the types of disinfection?

2-Written work questions for midterm assessment.

1. What diseases are examples of endogenous infection?
2. Explain the infectious process.
3. Diagnosis of brucellosis and recovery of the farm from this disease.
4. Smallpox disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
5. Diagnosis of rabies.
6. Control measures in infectious diseases.
7. Types of disinfection.
8. Iron disease and its treatment methods.
9. Autovaccines and their importance.

10. Infectious laryngotracheitis disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
11. Explain diseases spread by rodents
12. Equine influenza disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
13. RA, RCK,R B H, State the purpose of Rosbengal reaction.
14. Meaning and essence of sensitization, desensitization.
15. Protein disease (etiology, economic damage, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
16. Epizootology and prevention of Ayeski's disease.
17. Understanding of active, passive and colostral immunity.
18. Diagnosis and epizootology of swine fever.
19. Prevention of Bradzot disease.
20. Pleuropneumonia disease of goats (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
21. Understanding of alimentary, aerogenous, contact and transmissible transmission factors of infectious diseases.
22. Manifestations of infectious diseases.
23. Diagnosis of tuberculosis and recovery of the farm from this disease.
24. Ayeski's disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
25. Infectious hepatitis disease of carnivores (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
26. Understanding quarantine and containment measures.
27. Serological reactions to the diagnosis of infectious diseases.
28. Understanding the types of infection.
29. Horizontal and vertical pathways of causative agents.
30. Measures to combat fowl pox.
31. Rabies and its control measures.
32. General and special prevention.
33. Measures to fight protein disease.
34. Infectious anemia disease of horses (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

35. Specificity of immunity in viral diseases.
36. Understanding the steps in the infectious process.
37. Understanding of epizootics, panzootics, sporagies, epidemics, pandemics.
38. Explain general and specific treatments for infectious diseases
39. Rules for sending material to the laboratory.
40. African swine fever (economic damage, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
41. Mute disease (economic damage, epizootic chain, clinical symptoms, immunity, pathanatomical changes, diagnosis, preventive measures).
42. Explain the immunity of the body's cells.
43. Distinguishing Newcastle Disease from Avian Influenza.
44. Prevention of plague of carnivorous animals
45. Pathologoanatomical changes in anthrax.
46. List the types of immunity.
47. The relationship of the science of epizootology with other sciences.
48. Brucellosis prevention measures (general and special).
49. Treatment and preventive measures of Karason disease.
50. List the types and serovars of protein sickness virus.
51. Prevention of iron disease.
52. The immunological protective function of the lymphoid system.
53. African plague of horses (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
54. Diagnosis of sheep enterotoxemia.
55. Prevention of colibacteriosis in young animals.
56. List the features of immunity that are always effective.
57. Measures to prevent infectious diseases.
58. Methods of allergy testing.
59. Methods of testing infectious diseases in the laboratory.
60. Anthrax disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment and preventive measures).
61. Necrobacteriosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment and preventive measures).
62. Understanding the types of disinfection.
63. Forms of transmission of infectious diseases.
64. Explain the diseases spread by rodents

65. Epizootology of anthrax.
66. Prevention of brucellosis.
67. Actions directed against the source of the disease.
68. Tuberculosis pathogenesis and diagnostics.
69. Measures to prevent protein disease.
70. Swine scurvy disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
71. Explain infectious diseases of young animals.
72. Explain the concepts of pathogenicity and virulence.
73. Bradzot's disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
74. Rinderpest control measures.
75. Colibacteriosis of young animals (etiology, economic damage, epizootology, clinical signs of pathogenesis, course, diagnosis, treatment, preventive and countermeasures).
76. Explain pasteurization activities.
77. Importance of macroorganism and environmental factors in the formation of infection.
78. Leptospirosis prevention measures (general and special).
79. Bradzot's disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
80. Immunological reactivity and its types.
81. Bacilli and their role in infectious diseases.
82. The mechanism of transmission of pathogens.
83. Prevention of carnivorous hepatitis disease.
84. Diagnosis of infectious bronchitis of birds.
85. Prevention of measles.
86. List the diseases for which wild animals are reservoirs.
87. Diagnosis of Manka disease.
88. Measures to combat paratuberculosis.
89. Salmonellosis disease of sheep and goats (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
90. Diagnosis of viral diarrhea of cattle.
91. The ability of bacterial pathogens to cause disease depends on what properties?
92. Epizootic process and its driving forces.
93. General and special prevention of tuberculosis.

94. Paratuberculosis disease (etiology, economic damage, epizootic chain, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

95. Smallpox (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).

96. Prevention of brucellosis.

97. Tell the procedure-instructions for malleinization.

98. Explain the health of the economy from tuberculosis.

99. Avian flu disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

100. Poultry infectious bronchitis disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

101. Name the vaccines used for preventive purposes in protein disease.

102. Swine fever disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

103. Explain the measures to prevent infectious diseases?

104. Measles (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

105. Explain the types of immunity (active and passive immunity)?

106. Pasteurellosis (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

107. General and special prevention of infectious diseases.

108. Healing the farm from brucellosis.

109. Protein disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).

110. Laboratory diagnosis of leukemia.

111. Temiratki disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).

112. Laboratory diagnosis of listeriosis.

113. Paratuberculosis detection, prevention and control measures.

114. Laboratory diagnosis of protein disease.

115. Parainfluenza 3 disease identification, prevention and control measures.
116. Laboratory diagnosis of Karasan disease.
117. Bradzot and enterotoxemia detection, prevention and control measures.
118. Measures for detection, prevention and control of equine influenza.
119. Measures to identify, prevent, and fight swine fever.
120. Detection, prevention and control measures of poultry laryngotracheitis.
121. Swine influenza detection, prevention and control measures.
122. Camel plague detection, prevention and control measures.
123. Laboratory diagnosis of leukemia.
124. Rabbit myxomatosis disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).
125. Laboratory diagnosis of camel plague.
126. Rinderpest disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
127. Explain the methods of microscopic and bacteriological examination?
128. Serological reactions used in the diagnosis of infectious diseases.
129. Temiratki disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
130. Prevention of furunculosis, pseudomanosis and aeromycosis of fish.
131. Preventive measures to prevent infectious diseases of poultry.
132. Avian pullorosis (etiology, economic damage, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
133. Carnivore plague (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).
134. American rot disease (etiology, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
135. Carnivore hepatitis disease (etiology, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).
136. European rot disease (etiology, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
137. Laboratory diagnosis of smallpox.
138. Pasteurellosis detection, prevention and control measures.
139. Laboratory diagnosis of anthrax.
140. Explain zooanthroponous diseases?

141. Necrobacteriosis (etiology, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
142. Fish pox detection, prevention and control measures.
143. Explain the infectious diseases of young animals?
144. Laboratory diagnosis of tuberculosis.
145. Measures to identify, prevent and combat infectious diseases of fish.
146. Laboratory diagnosis of listeriosis.
147. Biological drugs, their types and rules of use.
148. Analyzing the results of epizootological investigation.
149. What diseases are diagnosed using allergic methods.
150. Laboratory diagnosis of brucellosis.

Written work questions for final assessment.

1. Types of infection and their economic damage to the economy.
2. Anti-tuberculosis measures.
3. Camel plague (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
4. Allergy and allergic diagnosis.
5. Measures to prevent leukemia.
6. The essence of the epizootic process.
7. Anti-tuberculosis measures.
8. Explain fully the bacteriological examination in the detection of infectious diseases.
9. The importance of phagocytosis in infectious diseases.
10. Diagnosis of leukemia.
11. Concept of infection.
12. List the modern diagnostic procedures performed in tuberculosis.
13. Diagnosis of protein disease, differential diagnosis.
14. Ways of transmission of the causative agent from one animal to another.
15. List the causative agents of salmonellosis by animal and poultry species.
16. History of the development of the science of epizootology.
17. Prophylactic means of tuberculosis (general and special).
18. General and special preventive measures of Ayeski's disease.
19. Infection and its types
20. Differences between viruses and bacteria
21. Importance of preventive measures in the national economy.
22. Diagnosis of tuberculosis.

23. Smallpox diagnosis, differential diagnosis, preventive measures.

24. Methods of reproduction of anaerobic microorganisms and the environments they need.

25. Methods of virus detection in pathological material.

26. The role of epizootology in the training of a veterinary doctor.

27. Anthrax control measures.

28. Listeriosis diagnosis, differential diagnosis, prevention.

29. Immunoglobulins, their types and importance in infectious diseases.

30. The causative agent of iron disease.

31. Decisions of the Cabinet of Ministers on ways to develop animal husbandry in the market economy.

32. Detection of anthrax in laboratory conditions.

33. How many different forms of rabies, laboratory diagnosis.

34. Toxic fungi and environments for their reproduction.

35. Understanding of special prevention.

36. Presidential decrees on ways to develop livestock breeding in the market economy.

37. General and special preventive measures in anthrax.

38. Mention measures to eliminate brucellosis.

39. Swine plague (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

40. Types of antibodies.

41. Types of immunity, immunological reactivity.

42. Diagnosis of brucellosis.

43. Ways of transmission of infectious diseases to healthy animals.

44. Bacteriological examination methods.

45. Treatment of iron deficiency.

46. The importance of microorganisms and viruses in the formation of infection and their pathogenicity.

47. Brucellosis control measures.

48. Diagnostic measures carried out in pig serum.

49. The role of serological diagnostic methods in the fight against infectious diseases.

50. Protection of people from zooanthropanous diseases.

51. Concept of immunity and its types.

52. Determination of protein diseases in laboratory conditions.

53. Enterotoxemia disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

54. Special prevention of infectious diseases.

55. Importance of formation and development of infection by microorganisms and viruses.

56. Antigens and their immunogenicity.

57. General and special preventive measures of protein disease.

58. Mange disease of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

59. Clinical signs of Bratzot's disease.

60. Diagnosis of swine scurvy.

61. Organization of treatment.

62. Mutism of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, prevention, and countermeasures).

63. Necrobacteriosis and its countermeasures.

64. What is the difference between communicable diseases and non-communicable diseases?

65. Epizootology of anthrax.

66. Epizootic, panzootic, sporadic, epidemic, pandemic.

67. Explain quarantine, restriction, isolators?

68. Rules for sending material to the laboratory.

69. African swine fever (economic damage, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

70. Manka disease (economic damage, epizootic chain, clinical symptoms, immunity, pathological changes, diagnosis, preventive measures).

71. Means and measures against epizootics.

72. Paratuberculosis disease of cattle (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

73. Smallpox and measures against it.

74. Diagnosis of infectious rhinotracheitis.

75. Vaccines and their types.

76. Anthrax disease (etiology, epizootic, chain pathogenesis, clinical symptoms, immune pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

77. Immunity and their types.

78. Epizootic process and its driving forces.

79. Infectious Disease Study Template.

80. Necrobacteriosis (clinical symptoms, pathological changes, diagnosis, differential diagnosis, treatment, immunity, preventive measures).

81. Ways of separation of pathogens from the body.

82. Avian flu disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

83. Infectious anemia disease of horses (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

84. Comment on serological reactions?

85. Carnivore pest control measures.

86. Immune mechanism and factors.

87. Peculiarities of epizootology of protein disease.

88. Colibacteriosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

89. Disinfection and its types.

90. Explain the epizootic process?

91. Transmissive and vertical (from generation to generation, transovarial transmission) of pathogens.

92. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

93. Rhinopneumonia of horses (etiology, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment)

94. Measures to prevent protein disease from being imported from other countries.

95. Explain diseases transmitted by insects?

96. Susceptible animals are the driving forces of the epizootic process.

97. Formation of active and passive immunity.

98. Reasons for long storage of anthrax foci.

99. Camel plague and its social risk.

100. Explain the methods of virological testing?

101. Specific characteristics of anaerobic infections.

102. Pasteurellosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

103. General and special prevention of smallpox.

104. Explain viral diarrhea disease.

105. Pathogenesis and laboratory diagnosis of rabies.

106. Economic damage of infectious diseases and importance of anti-epizootic measures.

107. Campliobacteriosis of cattle.

108. Pullorosis disease of poultry (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

109. How to control the source of the pathogen.

110. Tuberculosis epizootology.

111. Importance of macroorganism and external environment in the development of infection.

112. Understanding of vaccines and their importance in preventing infection.

113. Goat pleuropneumonia (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

114. The problem of prevention of the disease of the blackhead.

115. Epizootics and measures against it.

116. Actions directed against the transmission mechanism of the pathogen.

117. Pathogenesis and diagnosis of brucellosis.

118. Etiology, epizootology and methods of laboratory investigation of leptospirosis.

119. Specific and non-specific prevention of pasteurellosis.

120. Explain the methods of physical disinfection.

121. The role of disinsection in the elimination of transmissible diseases.

122. Listeriosis (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

123. Laboratory diagnosis of leukemia.

124. The fight against anthrax.

125. Laboratory diagnosis of brucellosis.

126. Economic damage of infectious diseases.

127. Infectious bronchitis disease of birds (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

128. Infectious hepatitis disease of meat-eating animals (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

129. Explain active and passive immunity?

130. Count the links of the epizootic chain?

131. Concept of epizootic, panzootic, sporadic, epidemic, pandemic.

132. Content, essence of sensitization and desensitization.

133. Explain general and specific treatments for infectious diseases.

134. Infectious mastitis disease of sheep (economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

135. Mange disease in horses (etiology, economic damage, clinical symptoms, pathanatomical changes, diagnosis, preventive measures).

136. Epizootic process and its driving forces.

137. Measles and measures to combat it.

138. Measures to prevent parainfluenza-3 disease.

139. Rules for obtaining pathological material in infectious diseases and sending it to the laboratory.

140. Poultry mycoplasmosis disease (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

141. General and special prevention of infectious diseases.

142. Avian flu disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

143. Measures against pasteurellosis.

144. Epizootology of mange disease of horses.

145. Explain about hapten?

146. Types of infectious diseases (zooanthroponosis diseases, zoonotic diseases, highly dangerous infectious diseases).

147. Cattle viral diarrhea disease (etiology, economic damage, epizootic chain pathogenesis, clinical signs, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).

148. Understanding of the use of hyperimmune blood sera, types.

149. Types of deratization, mechanism of action, quality assessment.

150. Protein disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).

151. Concept of epizootic, panzootic, sporadic epidemic, pandemic.

152. Explain the laboratory diagnosis of viral and infectious diseases?

153. Explain general and specific treatments for infectious diseases?

154. Infectious mastitis disease of sheep (economic damage, epizootic chain pathogenesis, clinical symptoms, immunity, pathanatomical changes, course, diagnosis, treatment, preventive measures).

155. Mange disease in horses (etiology, economic damage, clinical signs, pathanatomical changes, diagnosis, treatment, preventive measures).

156. Forms of transmission of infectious diseases.

157. Infectious mastitis of sheep.

158. Diagnosis and epizootology of horse dumb disease.
159. Clinical forms of bovine infectious rhinotracheitis.
160. What is tolerance?
161. Epizootological classification of infectious diseases.
162. General and special prevention of iron deficiency disease.
163. Pasteurellosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
164. Phases of epizootics.
165. Epizootology of infectious laryngo-tracheitis of birds.
166. Determining the quality of disinfection.
167. Parainfluenza-3 disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
168. Eczema disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
169. Activities held in the risk area for protein disease.
170. Explain the bacteriological diagnosis?
171. Importance of microorganisms and viruses in the formation of infection.
172. Marek's disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
173. Sheep chlamydiosis and its control measures.
174. Cattle plague (etiology, epizootology, clinic, course, diagnosis, preventive and countermeasures).
175. Deratization and its importance.
176. Explain the diseases that are forbidden to treat?
177. The role of the science of epizootology in the training of a veterinary doctor.
178. Smallpox (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
179. Epizootology of leukemia.
180. Explain panzootic diseases?
181. Comment on the carrier status of pathogens?
182. Teshen's disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
183. Anthrax diagnosis and prevention.
184. Explain the pathogen reservoir?
185. List the diseases that can be transmitted from humans to animals and how it occurs.
186. The relationship of epizootology with other sciences.

187. Rules of procedure for working with infectious animals.
188. Explain the technique of tuberculinization?
189. Name the vaccines used for preventive purposes in protein disease?
190. Poultry infectious bronchitis disease (etiology, epizootology, economic damage, pathogenesis, immunity, clinical symptoms, pathanotomy, diagnosis, differential diagnosis, treatment, preventive measures).
191. How do you explain the government's strong focus on infectious diseases?
192. Infectious anemia disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, diagnosis, treatment, preventive and countermeasures).
193. Swine fever.
194. Goat pleuropneumonia and its control measures.
195. Comment on chemical disinfectants?
196. Explain antigens and haptens?
197. Paratuberculosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
198. Ways of transmission of infectious disease agents.
199. What measures are used against wild animals in the fight against rabies?
200. Mechanization of disinfection works.
201. The role of veterinary-sanitary measures in disease prevention.
202. Poultry Marek's disease (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
203. Diagnosis of smallpox, differential diagnosis.
204. Epizootology of protein disease.
205. Explain allergy diagnosis?
206. What is a vaccine, serum, hyperimmune serum, convalescent blood, and its use.
207. Explain the types of disinfection?
208. Explain the schematic template for the study of infectious diseases?
209. Bia salmonellosis, diarrhea (abortion) disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
210. Protein disease (etiology, economic damage, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
211. The relationship of the science of epizootology with other sciences.
212. Laboratory diagnosis of anthrax.

213. Tuberculosis pathogenesis and diagnostics.
214. Prevention of leukemia.
215. The role of veterinary-sanitary measures in the prevention of infection and elimination of the disease.
216. The role of bacteriological examination in the detection of infectious diseases.
217. Infectious laryngotracheitis disease of birds.
218. Pestilence of carnivores (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
219. Transmissible gastroenteritis disease of pigs (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
220. Understanding anaphylaxis.
221. Application of immunology in practice.
222. General and special preventive measures of rabies.
223. Salmonellosis disease (etiology, epizootology, economic damage, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
224. Mechanization of disinfection measures.
225. Non-specific features of immunity.
226. Explain the nature of spread of infectious diseases in the form of sporadic, enzootic, epizootic and panzootic.
227. Diagnosis of rabies.
228. Measures to prevent infectious rhinotracheitis.
229. Types of allergy diagnosis.
230. Epizootology of leukemia.
231. Diseases transmitted by rodents and their control measures.
232. What is an epizootic focus and its definition.
233. Epizootology of bird flu disease.
234. Epizootology of protein disease.
235. Explain soil infectious diseases.
236. Humoral factors of immunity.
237. Allergic and serological diagnosis of mange disease in horses.
238. Clinical signs of cattle plague.
239. Clinical signs of anthrax.
240. In which diseases is toxicoinfection present?
241. Protective features of the organism's normative microflora.
242. Diagnosis and epizootology of leptospirosis.
243. Clinical signs in pigs.

244. Clinical signs of Aujeski's disease.

245. What is the difference between an infectious disease and a non-infectious disease?

246. Understanding of epizootological investigation methods.

247. Methods of treatment of infectious diseases.

248. Brucellosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).

249. Preventive measures in rabies.

250. Listeriosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).

251. Epizootic focus, understanding of the mechanism of disease spread.

252. Newcastle disease and its countermeasures.

253. The importance of disinfection, disinsection and deratization measures in the elimination of infectious diseases.

254. Epizootology of rabies.

255. Briefly explain panzootic diseases?

256. Specific features of immunity formed against viral diseases.

257. Explain the essence of allergy diagnosis.

258. Forms of clinical manifestations of infectious rhinotracheitis.

259. The role of bulls in the spread of campylobacteriosis.

260. Actions against bovine leukemia.

261. Measures to prevent infectious diseases.

262. Methods of testing infectious diseases in the laboratory.

263. Peculiarities of diagnosis of infectious diseases.

264. Infectious pleuropneumonia disease of goats (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

265. Aujeski's disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

266. Understanding infection and immunity.

267. Explain the epizootic process?

268. Manka disease and measures to combat it.

269. Cattle plague (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

270. Infectious gastroenteritis of pigs (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

271. Rabies disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

272. Understanding of secondary infection, colostral immunity.

273. Explain allergy diagnosis?

274. Infectious atrophic rhinitis disease of pigs (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

275. Aujeski's disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

276. The role of deratization in the fight against infectious diseases.

277. Prophylactic means of tuberculosis (general and special).

278. Leptospirosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathological changes, diagnosis, differential diagnosis, treatment, preventive measures).

279. The role of wild animals in the spread of rabies.

280. Comment on serological diagnosis?

281. Concept of infection.

282. Infectious laryngotracheitis disease (causing agent, epizootology, economic damage, pathogenesis, clinical signs, course, pathology, general and special treatment, preventive measures).

283. Infectious hepatitis disease of carnivores (causing agent, epizootology, economic damage, pathogenesis, clinical symptoms, course, pathology, general and special treatment, preventive measures).

284. Infectious mastitis disease of sheep (causing agent, epizootology, economic damage, pathogenesis, clinical signs, course, pathology, general and special treatment, preventive measures).

285. Epizootology of anthrax.

286. Insulators and their requirements.

287. Describe zooanthroponous diseases?

288. Tell the laboratory diagnosis of brucellosis?

289. Explain the health of the economy from tuberculosis?

290. Poultry YLT (infectious laryngotracheitis) disease (etiology, economic damage, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

291. Enumerate the measures used against epizootics.

292. Explain primary, secondary infection, mixed infection.

293. Horses' INAN disease and measures to combat it.

294. Swine flu disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

295. Black disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

296. Classification of epizootological classification of infectious diseases.

297. The difference between infectious diseases and non-infectious diseases.

298. Enterotoxemia (etiology, economic damage, pathogenesis, clinical signs, course, pathological changes, diagnosis, differential diagnosis, preventive measures).

299. Diagnosis of Newcastle disease in poultry and measures to prevent it.

300. Salmonellosis of young animals (etiology, economic damage, pathogenesis, clinical symptoms, course, pathological changes, diagnosis, differential diagnosis, preventive measures).

301. The nature of antibodies.

302. Cattle plague (etiology, epizootology, economic damage, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

303. Explain the diseases that can be transmitted from humans to animals and their prevention measures.

304. Anthrax control measures.

305. Veterinary-sanitary measures in the fight against infectious diseases.

306. Horse flu disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

307. State the purpose of AR, KBR, Rosbengal reaction.

308. Content, essence of sensitization, desensitization.

309. Protein disease (etiology, economic damage, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

310. Epizootology of Karasan disease.

311. The difference between infectious diseases and non-infectious diseases.

312. The subject and task of the science of epizootology.

313. Bradzot's disease (economic damage, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

314. Camel plague and measures to combat it.

315. Leptospirosis prevention measures (general and special)

316. Explain allergy diagnosis?

317. Understanding of disinsection methods.

318. Concept of epizootology, panzootic, sporadic, epidemic, pandemic.

319. Swine flu (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

320. Infectious pleuropneumonia disease of goats (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

321. Epizootological process and its driving forces.

322. Methods of epizootic investigation.

323. Measures to prevent infectious diseases.

324. Infectious vaginitis disease of cattle (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

325. Campylobacteriosis (virus) disease (etiology, economic damage, epizootological chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

326. Special treatment against epizootics)

327. Types of disinfection, deratization, disinsection (sterkushka activities.

328. Treatment of infectious diseases (general and

329. ilization, pasteurization, tindalization).

330. Salmonellosis disease of young animals (causing agent, economic damage, epizootology, pathogenesis, clinical signs and course, pathology, general and special treatment, preventive measures).

331. Avian flu disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

332. What is a vaccine, serum, hyperimmune serum, convalescent blood, its use.

333. Tell about the types of disinfection?

334. Schematic template for the study of infectious diseases.

335. Biyalar salmonellosis diarrheal disease (etiology, economic damage, epizootic chain, pathogenesis, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

336. protein disease (etiology, economic damage, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

337. Tasks of the science of epizootology.

338. The importance of vaccination in the prevention of infectious diseases.

339. Prevention of Newcastle disease in poultry.
340. Diagnosis of brucellosis.
341. How does the infectious process manifest itself?
342. Poultry scurvy disease (causing agent, economic damage, epizootology, pathogenesis, clinical signs and course, pathology, general and special treatment, preventive measures).
343. Methods of treatment of infectious diseases.
344. Immunity and its types
345. Paratuberculosis disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
346. Smallpox (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).
347. Types and quality of disinsection.
348. Non-specific prevention of paratuberculosis.
349. Diagnosis and general prevention of campylobacteriosis.
350. Diagnosis of equine epizootic lymphangitis.
351. Clinical signs characteristic of Karason's disease.
352. Explain natural epizootic diseases.
353. Treatment and prevention of pleuropneumonia in goats.
354. Diagnosis and prevention of Bradzot disease.
355. Clinical signs and diagnosis of necrobacteriosis.
356. Epizootic foci are divided into several types depending on their activity.
357. Hematological changes in equine infectious anemia.
358. Infectious rhinotracheitis of black cattle should be distinguished from other diseases by clinical signs.
359. Pathologoanatomical changes in swine scurvy.
360. What are the forms of relationship between macroorganisms and pathogens? Describe them.
361. What diseases are examples of endogenous infection?
362. Explain the infectious process.
363. Diagnosis of brucellosis and recovery of the farm from this disease.
364. Smallpox disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
365. Diagnosis of rabies.
366. Control measures in infectious diseases.
367. Types of disinfection.
368. Iron disease and its treatment methods.

369. Autovaccines and their importance.

370. Infectious laryngotracheitis disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

371. Explain diseases spread by rodents

372. Equine influenza disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

373. RA, RCK,R B H, State the purpose of Rosbengal reaction.

374. Meaning and essence of sensitization, desensitization.

375. Protein disease (etiology, economic damage, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

376. Epizootology and prevention of Ayeski's disease.

377. Understanding of active, passive and colostral immunity.

378. Diagnosis and epizootology of swine fever.

379. Prevention of Bradzot disease.

380. Pleuropneumonia disease of goats (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

381. Understanding of alimentary, aerogenous, contact and transmissible transmission factors of infectious diseases.

382. Manifestations of infectious diseases.

383. Diagnosis of tuberculosis and recovery of the farm from this disease.

384. Ayeski's disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

385. Infectious hepatitis disease of carnivores (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

386. Understanding quarantine and containment measures.

387. Serological reactions to the diagnosis of infectious diseases.

388. Understanding the types of infection.

389. Horizontal and vertical pathways of causative agents.

390. Measures to combat fowl pox.

391. Rabies and its control measures.

392. General and special prevention.

393. Measures to fight protein disease.

394. Infectious anemia disease of horses (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).

395. Specificity of immunity in viral diseases.

396. Understanding the steps in the infectious process.

397. Understanding of epizootics, panzootics, sporagies, epidemics, pandemics.

398. Explain general and specific treatments for infectious diseases

399. Rules for sending material to the laboratory.

400. African swine fever (economic damage, clinical signs, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment, preventive measures).

401. Mute disease (economic damage, epizootic chain, clinical symptoms, immunity, pathanatomical changes, diagnosis, preventive measures).

402. Explain the immunity of the body's cells.

403. Distinguishing Newcastle Disease from Avian Influenza.

404. Prevention of plague of carnivorous animals

405. Pathologoanatomical changes in anthrax.

406. List the types of immunity.

407. The relationship of the science of epizootology with other sciences.

408. Brucellosis prevention measures (general and special).

409. Treatment and preventive measures of Karason disease.

410. List the types and serovars of protein sickness virus.

411. Prevention of iron disease.

412. The immunological protective function of the lymphoid system.

413. African plague of horses (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

414. Diagnosis of sheep enterotoxemia.

415. Prevention of colibacteriosis in young animals.

416. List the features of immunity that are always effective.

417. Measures to prevent infectious diseases.

418. Methods of allergy testing.

419. Methods of testing infectious diseases in the laboratory.

420. Anthrax disease (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment and preventive measures).

421. Necrobacteriosis (etiology, economic damage, epizootic chain, pathogenesis, clinical symptoms, immunity, pathanatomical changes, diagnosis, differential diagnosis, treatment and preventive measures).

422. Understanding the types of disinfection.
423. Forms of transmission of infectious diseases.
424. Explain the diseases spread by rodents
425. Epizootology of anthrax.
426. Prevention of brucellosis.
427. Actions directed against the source of the disease.
428. Tuberculosis pathogenesis and diagnostics.
429. Measures to prevent protein disease.
430. Swine scurvy disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
431. Explain infectious diseases of young animals.
432. Explain the concepts of pathogenicity and virulence.
433. Bradzot's disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
434. Rinderpest control measures.
435. Colibacteriosis of young animals (etiology, economic damage, epizootology, clinical signs of pathogenesis, course, diagnosis, treatment, preventive and countermeasures).
436. Explain pasteurization activities.
437. Importance of macroorganism and environmental factors in the formation of infection.
438. Leptospirosis prevention measures (general and special).
439. Bradzot's disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, course, diagnosis, treatment, preventive and countermeasures).
440. Immunological reactivity and its types.
441. Bacilli and their role in infectious diseases.
442. The mechanism of transmission of pathogens.
443. Prevention of carnivorous hepatitis disease.
444. Diagnosis of infectious bronchitis of birds.
445. Prevention of measles.
446. List the diseases for which wild animals are reservoirs.
447. Diagnosis of Manka disease.
448. Measures to combat paratuberculosis.
449. Salmonellosis disease of sheep and goats (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).
450. Diagnosis of viral diarrhea of cattle.
451. The ability of bacterial pathogens to cause disease depends on what properties?
452. Epizootic process and its driving forces.

453. General and special prevention of tuberculosis.

454. Paratuberculosis disease (etiology, economic damage, epizootic chain, epizootology, pathogenesis, clinical symptoms, course, diagnosis, treatment, preventive and countermeasures).

455. Smallpox (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).

456. Prevention of brucellosis.

457. Tell the procedure-instructions for malleinization.

458. Explain the health of the economy from tuberculosis.

459. Avian flu disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

460. Poultry infectious bronchitis disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

461. Name the vaccines used for preventive purposes in protein disease.

462. Swine fever disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

463. Explain the measures to prevent infectious diseases?

464. Measles (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

465. Explain the types of immunity (active and passive immunity)?

466. Pasteurellosis (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).

467. General and special prevention of infectious diseases.

468. Healing the farm from brucellosis.

469. Protein disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).

470. Laboratory diagnosis of leukemia.

471. Temiratki disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).

472. Laboratory diagnosis of listeriosis.

473. Paratuberculosis detection, prevention and control measures.

474. Laboratory diagnosis of protein disease.
475. Parainfluenza 3 disease identification, prevention and control measures.
476. Laboratory diagnosis of Karasan disease.
477. Bradzot and enterotoxemia detection, prevention and control measures.
478. Measures for detection, prevention and control of equine influenza.
479. Measures to identify, prevent, and fight swine fever.
480. Detection, prevention and control measures of poultry laryngotracheitis.
481. Swine influenza detection, prevention and control measures.
482. Camel plague detection, prevention and control measures.
483. Laboratory diagnosis of leukemia.
484. Rabbit myxomatosis disease (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).
485. Laboratory diagnosis of camel plague.
486. Rinderpest disease (etiology, economic damage, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
487. Explain the methods of microscopic and bacteriological examination?
488. Serological reactions used in the diagnosis of infectious diseases.
489. Temiratki disease (etiology, economic damage, epizootology, epizootic chain, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
490. Prevention of furunculosis, pseudomanosis and aeromycosis of fish.
491. Preventive measures to prevent infectious diseases of poultry.
492. Avian pullorosis (etiology, economic damage, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
493. Carnivore plague (etiology, economic damage, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).
494. American rot disease (etiology, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
495. Carnivore hepatitis disease (etiology, epizootology, pathogenesis, clinical symptoms, immunity, course, diagnosis, treatment, preventive and countermeasures).
496. European rot disease (etiology, epizootology, pathogenesis, clinical signs, immunity, course, diagnosis, treatment, preventive and countermeasures).
497. Laboratory diagnosis of smallpox.
498. Pasteurellosis detection, prevention and control measures.
499. Laboratory diagnosis of anthrax.

500. Explain zooanthroponous diseases?

Test questions for 1-Interim assessment

1. What is another name for Au e ski disease?
A. False rabies, infectious bulbar paralysis, infectious meningoencephalitis.
B. Morbus Au e ski, pseudo rabies.
S. Polyencephalitis, myelitis, paralysis.
D. Hydrophobia, ataxia, asthesia.
Who discovered Au e ski disease and when?
A. 1902 A. Auski, Hungary.
B. 1900 F. Gutira, Y. Marek, Czechoslovakia.
S. 1912 M. Eno, France.
D. 1920 Stapr et al., USA.
3. Which answer to Aueski's prompt is correct?
A. DNA storage, family Herperviridae.
B. RNA-protecting, neurotropic, epitheliotropic.
S. DNA-preserving, myotropic, parenchymatropic.
D. RNA-sparing, neurotropic, somatropic, heterotropic.
4. In which answer is the chronology according to the degree of tendency to Aueski correct?
A. Cattle, deer, sheep, pig, horse, cat, dog, fox....
B. One-hooved animals, two-hooved animals, carnivorous animals.
S. All pets and wild animals.
D. All animals living on land are prone, birds are prone.
What is the source of the disease in Au e ski?
A. Sick animals, virus carriers.
B. Often latently infected animals.
S. Infected and immunizing subinfection cases.
D. Relapsed patients, newly brought sick animals.
6. In what season is Aueski's disease more common?
A. In spring, autumn.
B. In autumn, winter.
S. In the summer, in the spring.
D. In winter, in spring.
7. Which animals do not experience severe itching in Aueski's disease?
A. In pigs.
B. In dogs, cats.
S. In sheep and goats.
D. Cattle, horses.
8. In Aueski in 6-8 days illness in pigs how many % organize enough?
A. 100%.
B. 80%.
S. 60%.
D. 40-60%.
9. Aueski in pigs most of the time how clinical syndromes with will it pass?

A. Epilepsy, pathological view
B. Polyneurosis, ataxia.
S. Asthesia, atony, asthenia.
D. Neurosis, neuritis, epilepsy.
10. Clinical symptoms characteristic of Aueski are given correctly in which answer?
A. Strong itching, scratching, biting itching
B. Epilepsy, angina, bradycardia.
S. Apnea, dyspnea, neurosis, hydrophobia.
D. Tachycardia, strong itching, discomfort.
11. In Aueski special healer biological preparations is there
A. Yes, hyperimmune blood serum, special gammaglobulin.
B. Yes, convalescent blood, novarsenol.
Q. Yes, sulfonamides, nitrofurans.
D. Yes, bacteriocide, herbicide, phytoncide.
12. Aueski in illness how event used?
A. Quarantine announcement will be done.
BC h eclov announcement will be done.
S. The household is healthy that announcement will be done.
D. " Stamping out " method is used.
13. Who and when invented the causative agent of necrobacteriosis?
ARKox (1881), F. Leffler (1884).
B. F. Leffler, A. Froggy (1884).
S. F.ssion, IP Pavlov (1901).
D. IM Sechenev, D. Aginer (1881).
14. Necrobacteriosis disease of the trigger to oxygen relatively optimum conditions tell me
A. Obligation anaerobic.
B. Obligation aerobic.
S. Optional anaerobic.
D. _ Optional aerobic.
15. Necrobacteriosis of the trigger which serotypes identified?
A. A, AB, B, Serotypes.
B. from 10 more than serotypes.
Q. 2 serotype.
D. _ 8 serotype.
16. Necrobacteriosis of triggers pathogen serotypes tell me
A.A andAB serotype.
B. All serotypes pathogen.
S. All strains pat ogen.
D. All strains lentogenic.
17. Necrobacteriosis triggers how exo, endotoxins separates?
A. Leukocidin, necrotoxin, hemolysin, cytotoxin.
B. Neurotoxin, myotoxin, epitheliotoxin.
S. Fibrotoxin, leukotoxin, phagotoxin.

D. _ Polytoxin, monotoxin, plasmotoxin.
18. Necrobacteriosis which to the group belong to?
A. Zooanthroponosis.
B. Kavshovchi to animals.
S. Bir to ungulates.
D. Pair to ungulates.
19. Necrobacteriosis trigger sources?
A. Sick animals, bacteria carriers.
B. Damaged fodder, water.
S. Alimentary, aerogenous factors.
D. Vertical, horizontal factors.
20. In what way is the causative agent of necrobacteriosis separated from a sick animal?
A. All excreta with, injury tissues with.
B. Saliva, feces, urine with.
S. Aerogen, genital, contact the way with.
D. Hemolytic, lympholytic, alimentary.
21. Necrobacteriosis young in animals skin and mucus on curtains how pathology with night?
A. Calves diphtheria, swine in children rhinitis, stomatitis, dermatitis.
B. Abscess, phlegmon, pleurisy, pneumonia.
S. Gangrene, abscess, dermatitis.
D. _ Gastritis, gastroenteritis, pododermatitis.
22. In necrobacteriosis special prevention is there
A. Bor, Nekovac, polyvalent, associated.
B. No, bacteriocide preparations used..
Q. No, anti septic preparations is used.
D. Not developed.
23. Necrobacteriosis to people is it contagious?
A. It is contagious.
B. It is not contagious.
S. It can only be transmitted through trauma.
D. Science sure information no.
24. What group does the causative agent of contagious pleuropneumonia of cattle belong to?
A. Mycoplasmas.
B. To rickettsiae.
S. To bacteria.
D. To viruses .
25. Mycoplasmas how pathogen descendants have
A. Mycoplasmas (type 76), ureoplasma (type 2).
B. 40 ha near types there is
From S. 20 more than types there is
D. Pathogen and a pathogen types there is

26. Mycoplasmas special features which in the answer right given?
A. Polymorphism, strict curtain no.
B. Coccoid, oval.
S. Rod-shaped, spiral.
D. With spores, without spores.
27. Cattle KPP – the causative agent of the disease grows in what nutrient environments?
A. In a natural nutrient medium with 10-20% horse serum and 10% yeast extract.
B. In all liquid, semi-liquid nutrient media.
S. In all solid environments.
D. Egg yolk, in food media with added glucose.
28. Which animals cattle KPP to the disease inclined?
A. Cattle, zebu-like cattle, buffalo, bison, yak.
B. All couple ungulates.
S. Experimental if infected all animals inclined
D. Bu of the instigator pathogenicity depends.
29. Small laboratory animals if the experimental y is flown get sick
A. No.
B. Yes.
S. Sometimes gets sick
D. Injury to the way depends.
30. Cattle KPP - in the disease to die what %?
A. 10-90%.
B. 10-20%.
S. 20-30%.
D. 30-40%.
31. Cattle KPP is a disease triggers how toxins separates?
A. Exo, endotoxins - in the composition lipo polysaccharide and galactan there is.
B. Exotoxin, universal toxin.
S. Endotoxin, hemolytic, proteolytic.
D. Neurotoxin, pneumotoxin, myotoxin.
32. Cattle CPP - how long does the disease last on average?
A. 40-45 days.
B. 10-12 days.
S. 15-20 days.
D. 20-25 days .
33. Foods KPP - disease special pathanatomical changes?
A. Injured in the lungs (up to 20 l.). fibrinous – reddish – yellowish exudate t o' plans.
B. Exudation, proliferation, hepatization.
S. Atelectasis, emphysema, pneumonia.
D. Pneumonia, pleurisy, spike.
34. Cattle KPP - how is the disease diagnosed?
A. Serological, bacteriological, clinical, pathanatomical, epizootological check with.
B.AR, KBR, GATR with.

S. Allergic and bioassay with.
D. With bacteriological examination.
35. Cattle KPP of XEB - in which case is it considered to have completely disappeared?
A. From the destruction of the last diseased animal in the furnace after one in another disease if it doesn't come out.
B. Last ill an animal no from being done then at 6 months disease if it doesn't come out.
S. Allergic 100% negative for the 2nd time in the tests the result if taken
D. The last sick animal no from being done then 2 years inside disease if it doesn't come out.
36. Goats KPP - in the disease scientist what %?
A. 70-100%.
B. 10-20%.
S. 30-40%.
D. 50-60%.
37. Goats KPP is a disease of the trigger morphological shape how?
A. Coxsimon, rod, thread.
B. S h arciform, prismatic, spiral.
S. Polymorph, all in forms.
D. This strain type depend _
38. In the flock goats KPP - in the disease illness level what %?
A. 100%.
B. 80%.
S. 60%.
D. 50%.
39. Goats with KPP - disease clinical others _ _ appear from being after when scientist will be
A. From 7-10 days after.
B. One moon inside.
S. 15-20 days after.
D. in 2-3 months.
40. In which case, the diagnosis of KPP-disease in goats is correct is it?
A. Culture if isolated, sick from a goat received in patmaterial instigator if found.
B. Serodiagnosis mu s bat the result if he gives
S. Allergic diagnosis positive the result if he gives
D. Disease public tu0Clsa.
41. Differentiate the KPP disease of goats from similar diseases must
A. Pasterell o z, agalactia.
B. Salmanell o z, diplococcosis.
S. Colibacteriosis, pneumonia.
D. Compilobacteriosis, smallpox.
42. Goats KPP in illness laboratory inspection term how much
A. 60-90 days.

B. 10-20 days.
S. Bir to the moon.
D. 30-40 days.
43. Doors KPP disease in prevention how vaccine used?
A. Hydro Okisammonian liquid-formal-vaccine, immunity 1 year continue is enough.
V. Live-dry vaccine, immunity 6 months continue is enough.
S. Water yu q-emulsion vaccine, immunity 10 months continue is enough.
D. Associate polyvalent vaccine immunity 1 year continue is enough.
44. Goats KPP in illness in the oven how event used?
A. Quarantine announcement will be done.
B. C h eklov announcement will be done.
S. Unhealthy that announcement will be done.
D. " Stamping out " method is used.
45. When will the quarantine be lifted in goats with KPP disease and how long will it be under control?
A. Two months after the last patient has been eliminated, there will be another 1 year of follow-up.
B. Last ill no from q i lingan then 21 days after that, another 6 months in control will be
Q. Last ill no from q i lingan then 14 days after that, another 1 year in control will be
D. Two months after the last patient was eliminated, another 6 months will be observed.
46. Cattle plague in the CIS countries since when since does not meet?
A. Since 1929.
B. Since 1970.
S. Since 1950.
D. since 1980.
47. The virus that causes rinderpest has antigenic and immunological similarities with which viruses?
A. Dogs the plague, people smallpox disease.
B. C h intestine, protein.
S. Kutyrish, Aueski.
D. Vesicular stomatitis, viral diarrhea.
48. Cattle plague to the trigger special cultural feature tell me
A. Chicken in the embryo to ssPT have
B. Cultural in tissue necrosis calls
S. Epithelio trop, neuro trope.
D. Bezli tissues lysis does.
49. Cattle plague stationary in their furnaces colostral immunity how much time continue enough?
A. 8-11 months.
B. 2-3 months.
S. 4-5 months.
D. 5-7 months.

50. Cattle in the plague primary in the oven scientist what %?
A. 90-100%.
B. 70-80%.
S. 60-70%.
D. 50-60%.
51. Cattle plague with sick an animal what will be done?
A. Sick animals without blood method with killed, skinned with will be burned.
B. Special to the graves will be buried.
S. is thrown into the wells of Becker.
D. It is buried at least 2m deep and disinfected with 20% freshly slaked lime.
52. Of cattle bad good quality catarrhal fever how disease?
A. Sporadic transient, acontagious, virosis disease.
B. Epizootic, contagious, viral disease.
S. Exotic transient, contagious, bacteriosis.
D. Panzootic transit, transcontagious, viriosis.
53. Yo mon good quality catarrhal fever more which age in animals occurs?
A. 1-4 years old cattle and in buffaloes occurs.
B. Up to 1 year all couple in ungulates occurs.
S. In 2-4 month old calves, lambs, goats.
D. All age forager in animals.
54. Yo mon good quality catarrhal fever against special prevention work is it done?
A. No, the vaccine work not released.
B. Yes, woven hydroxy-al y uminyli vaccine.
S. Associate vaccine.
D. Weakened alive vaccine.
55. Sick bad good quality catarrhal feverish an animal meats how neutralized?
A. 1.5-2 kg divided into pieces 2.5-3 atm. Boiling at 115-117 ºC for 1.5-2 hours is allowed for consumption inside the farm.
B. Without a rock that is found.
S. Boil, meat choir to animals is given.
D. Bone it, meat him is prepared.
56. In cattle infectious nodular dermatitis inciting virus which one
A. Neethling-typevirus.
B. Allerton-typevirus.
C. Orpheling-typevirus.
D. All answers right.
57. The causative agent of nodular dermatitis is morpho-physiologically similar to which virus?
AC h intestine to the virus.
B. Rabies to the virus.
S. White to the virus.
State to the virus.
58. ssPT of the causative agent of nodular dermatitis – in a 5-7-day-old chicken embryo when observed?

A. In 5-14 days.
B. In 2-4 days.
S. In 4-6 days.
D. In 6-8 days.
59. Nodular to dermatitis the most sensitive animal?
A. Lactation period a cow and buffalo
B. Service period cattle
S. Strait cows.
D. Mainly calves of 6-8 months.
60. Special prevention in nodular dermatitis and special treatment tell me
A. Special prevention, special treatment work not released.
B. Antibiotics, sulfamides.
S. Vitamin, hormonal therapy.
D. Novarsenol, osarsol, among others preparations.
61. Of sheep with chlamydia abortion (constipation) disease which to the group belong to?
A. To zoonoses.
B. Zooanthroponoses.
S. Anthropozoonoses.
D. Ubiquit to infections.
62. Chlamydia where _ _ lives?
A. Obligate, cells inside.
B. In the blood.
S. In the muscles.
D. Epithelium in the tissues.
63. Chlamydia development cycle how much time?
A. 40-48 hours.
B. 48-72 hours.
S. 20-24 hours.
D. 1 week.
64. In Chlamydia how antigen have
A. Thermostable.
B. Thermolabile.
S. Glyco polypeptide
D. Polypeptide, lipoprotein.
65. Which laboratory animals are susceptible to sheep chlamydia abortion?
A. Aq mouse, rat, sea pig, rabbit.
B. Cat a child, a dog a child, a rabbit.
S. Mouse rat, chicken chicks.
D. Lamb, goat, calf, rabbit.
66. Abortion what % - chlamydia (in sheep)?
A. Up to 60%.
B. Up to 70%.
S. Up to 30%.

D. Up to 20%.
67. When is the diagnosis of sheep chlamydiosis considered valid?
A. If a special body is found in the uterus, vaginal fluid, if it has a positive reaction to KBR, RNGA, if a pathogen is found in a chicken embryo.
B. Pathanatomical changes, abortion.
S. Epizootological, clinical analyses.
D. RA, KBR, RNGA to positive the result if he gives
68. Vaccine work released?
A. Yes, emulsin vaccine Russia VIEV.
V. No.
S. Yes, Czechia , emulsin vaccine.
D. Polyvalent water and white GOV – France.
69. Sheep and goats plague when , where _ _ studied?
A. In West Africa, 1942.
B. In Australia, 1910.
In S. Hungary, 1900.
D. in France, 1940.
70. In what kind of environment does the virus that causes plague of sheep and goats grow (pathogenic effect)?
A. Vegetation in lamb, rabbit organism, chicken embryo, lamb, calf kidney culture, ssPTgaega.
B. All natural food environments.
S. In selective nutrient environments.
D. In nutrient medium supplemented with blood serum and glucose.
71. Pestilence with sick sheep and goats what will be done?
A. Sick mandatory killed, burned.
B. Patients treated, healthy is vaccinated.
S. All to the sick hyperimmune blood serum is added.
D. Mandatory meat for will be slaughtered.
72. Sheep and goats to the plague against cultural virus vaccine with immunity when vaccinated (France). how much time continue enough?
A. 3 years.
B. 1 year.
S. 2 years.
D. 10 months.
73. Pestilence the disease in determining the most reliable sero diagnosis?
A. IFR, PCR.
B. AR, KBR, GATR.
S. RDP, GAR.
D. All sero tests reliable.
74. In which answer is the causative agent of manka disease correctly written?
A. Non-motile, spore, capsule harvest does not Bacteria.
B. Mycoplasma.
S. Spore, capsule harvest does, clostridia.

D. Rickettsia.
75. Which organ is most often injured in Manka's disease?
A. Lungs, nose (cavity...).
B. Stomach, intestines.
S. Skin, mucous curtains.
D. Nerve tissues, muscles.
76. Manga conditional respectively which in forms night?
A. Nose form, skin, lung, latent.
B. Nose, stomach, intestines shape
S. Skin, muscle shape
D. Head, feet, skin shape
77. Mangada in practice which diagnosis a lot used?
A. Allergic diagnosis.
B. Sero diagnosis.
S. Clinical-syndromatic diagnosis.
D. Laboratory diagnosis.
78. Malleinization how many kind of held?
A. In autumn, leather under, skin between
B. In autumn, muscle between
S. Theriosti, tail skin between
D. Only ophthalmo test
79. Manga the disease which from diseases differentiate should
A. Mit, epizootic lymphangitis.
B. Strigu sh ylishay, dermatitis.
S. Streptococcus, myeliosis.
D. Skin diseases.
80. Sap with ill an animal what will be done?
A. Mandatory kill and skin with will be burned.
B. Only pedigree animals is treated.
S. Scientific studies for-strain to get is sent.
D. Hyperimmune blood serum with treatment possible
81. Sap in illness quarantine void to be done which in the answer right given?
A. 45 days after final disinfection after destruction of the last sick animal after.
B. Last ill an animal no from being done after 6 months after.
Q. Last ill an animal no will be done, 21 days after
D. Last ill an animal no after 1 year after
82. Horses flu disease trigger strain m , serotypes in them to what depends?
A. Ickib-antigen, d-antigen and in neuroamin and to enzymes depends.
B. M, D, A to antigens.
S. Shell and internal antigens.
D. Properdin, globulin, interferons.
83. Horses in influenza, INAN diseases ECHT-acceleration reason what?
A. Erythropenia, globulinemia.
B. Erythrocytosis, anemia.

S. Anisatocytosis, poikilocytosis.
D. Leukocytosis, phagocytosis, leukopenia.
84. Viral pathogens in identification which of the methods used?
A. Viral my diagnosis with check
B. Bacterial from infections differentiate
S. Mycotic from infections differentiate
D. Prionli, haptenli from infections differentiate
85. C h furnaces plague the first times when , where _ _ studied?
A. 1833 USA at Ohio in the state.
B. 1890 in France.
S. 1901 Russia, Kaluga in the region.
D. 1603 China, Manguria in the region.
86. C h furnaces in the plague scientist what %?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
87. C h furnaces in the plague virus portability how much time continue enough?
A. 3-10 months.
B. 2-5 months.
S. 4-8 months.
D. 1 year.
88. C h furnaces in the plague virus antigen in determining which reaction put?
A. IFR, NR.
B. AR, KBR, GAR.
S. GATR, KUBR, RA.
D. All serological reactions.
89. Quarantine during swine fever void from being done how much from time after pig take exit can
A. From 12 months after.
B. Two from the moon after.
S. From 4 months after.
D. From 8 months after.
90. C h furnaces Africa plague of the trigger pathogenicity degree tell me
A. diluted 1:109 ill 1 ml of pork blood disease provokes
B. No, 4 ml disease provokes
S. 3-5 ml disease provokes
D. diluted 1:10 1 ml of blood disease provokes
91. C h furnaces Africa plague in illness which vaccine used?
A. Bloodless method killed and burned.
B. Becker to the well thrown away.
S. 2 m deep digging if buried will be
D. Only 20% diluted lime with disinfection to do a must
92. C h goat flu disease the first times when, which countries pandemic as note done?

A. 1918 USA.
B. 1860 France.
S. 1882 England.
D. 1920 Russia.
93. The virus that causes swine flu is similar to which viruses in terms of antigenic properties?
A. People flu the disease instigator to the virus.
B. Horses, pigs, dogs, poultry flu triggers.
S. Dog, pig, poultry flu triggers.
D. All animals flu triggers.
94. C h goat in the flu characteristic pathanatomical changes which in the answer right defined?
A. Lungs swollen nose cavity, larynx, bronchi blood mixed slimy mass is collected.
B. In veins sclerosis, thrombus, hemorrhage observed.
S. Gastritis, enteritis, pneumonia.
D. Kon' y unctivitis, keratitis, vulvitis.
95. C h goat the flu which from diseases differentiate should
A. Olat, pasterell o z, Salmanell o z, enzo o tik pneumonia.
B. Au e ski, rhinitis, transmissive gastritis.
S. Saramas, plague, pasteurellosis , colibacteriosis.
D. White, plague, Africa plague, rhinitis.
96. C h goat flu in illness how treatment is used.
A. Symptomatic treatment.
B. Special treatment.
S. Gamma globulins.
D. Hyperimmune blood serum.
97. C h goat from the flu recovered an animal blood in serum how antibodies have
A. Virus neutralizing, anti Hemagglutin.
B. Complement binding, precipitating.
S. Agglutination inhibitor.
D. Inhibitor of phagocytosis.
98. C h of goats viral gastroenteritis disease first there is when which in the country note done?
A. In the USA, 1946.
B. in Russia, 1920.
In S. China, 1900.
D. in England, 1910.
99. C h furnaces viral to gastroenteritis special clinical characters which in the answer right?
A. Catarrhal, hemorrhagic gastroenteritis, vomiting, diarrhea, organism dehydration, death a large %.
B. Bloody drink withdrawal, pneumonia, strong itching
S. Motion balance break down, cough.
D. Asthesia, asthenia, ataxia, adhesion.

100. The virus that causes swine viral gastroenteritis is antigenically similar to which viruses?
A. Hemagglutinating to coronaviruses.
B. Antiviral to poxviruses.
S. To rhabdoviruses that inhibit agglutination.
D. To rhabdo, corona, pox viruses.
101 . Tell the sources of the causative agent of swine viral gastroenteritis?
A. Sick and recovered pigs.
B. Products from sick pigs.
S. Rodents, blood-sucking insects.
D. Infected fodder, piglets.
102. How long does immunity last in a pig that has recovered from swine viral gastroenteritis?
A. Two years.
B. One year.
S. Up to three years.
D. Four years.
103. Name the clinical signs of infectious dysentery of piglets.
A. Weight loss, bloody-mucous diarrhea.
B. Pneumonia, fever, diarrhea.
S. Ataxia, asthenia, hydrophobia.
D. Rhinitis, pneumonia, cough, diarrhea.
104. When was the disease of piglet dysentery registered for the first time, in which country?
A. USA, 1921.
B. Canada, 1910.
S. Russia, 1901.
D. France, 1903.
105. What is the correct answer for the causative agent of piglet dysentery?
A. There is. hyodysentery, gram-negative, anaerobic, spirochete.
B. There is. hyodysentry, gram-positive, aerobic, mycoplasma.
S. There is. treponema, gram-positive, aerobic, rickettsia.
D. There is. treponema, gram negative, aerobic, bacteria.
106. Piglet dysentery is most common in pigs of which age?
A. 2-6 months old.
B. 6-12 months old.
S. 1-4 months old.
D. 1-2 months old.
107. The treatment of piglet dysentery is given correctly in which answer?
A. Soda-osarsol solution, thilan, pharmazine, trichopo.
B. Antibiotics, sulfonamides.
S. Bacteriophages, gammaglobulin.
D. Convalescent blood, hyperimmune blood serum.
108. Which clinical signs of infectious bronchitis in chickens?

A. Pathology of respiratory organs, pathology of reproductive organs.
B. Gastro-enteritis, intestinal pathology.
S. Pathology of head organs.
D. Conjunctivitis, keratoconjunctivitis.
109. When and where was infectious bronchitis registered for the first time?
A. In the USA, 1931.
B. in Italy, 1902.
In S. Germany, 1882.
D. in Russia, 1910.
110. What is the causative agent of infectious bronchitis, and how many serotypes have been identified?
A. RNA storage coronavirus, more than 20 serotypes.
B. DNA storage poxvirus, more than 20 serotypes.
S. RNA-storing herpesvirus, more than 10 serotypes.
D. DNA storage coronavirus, more than 10 serotypes.
111. Which animals are prone to experimental infection with the causative agent of infectious bronchitis?
A. Bat, hyena, rabbit babies.
B. Mouse, rat, kitten.
S. Guinea pig, mouse, rat.
D. Rabbit, mouse, rat.
112. Which serological reactions are used in the diagnosis of infectious bronchitis?
A. RN, RNGA, RDP, RIF, IFA, PZR.
B. RA, RP, KBR, RP.
S. RA, KBR, RP, PZR, IFA.
D. RA, RP, KBR are sufficient.
113. How is infectious bronchitis treated?
A. No treatment has been developed.
B. It is treated symptomatically.
S. Antibiotic powder is dusted by aerosol method.
D. 40% formaldehyde vapor is used.
114. What is the causative agent of poultry leukemia?
A. RNA-storing oncovirus, 5 serogroups, A,B,C,D,E.
B. DNA-storing coronavirus, there are 3 serogroups.
S. RNA-storing poxvirus, there are 5 serogroups.
D. DNA-storing oncovirus, there are 4 serogroups.
115. Poultry of which age are more susceptible to leukemia?
A. Poultry 4 months and older.
B. Up to 2 months.
S. Up to 1 year.
D. Up to 2 years.
116. Poultry leukosis should be distinguished from which diseases?
A. Tuberculosis, Marek, coligranulomatosis.
B. Salmonellosis, ILT, colibacteriosis.

S. Escherichia, lymphomatosis, salmonellosis.
D. Tuberculosis, brucellosis, Newcastle.
117. How is avian leukemia treated?
A. A treatment method has not been developed.
B. It is destroyed by burning.
S. Compulsory submission to meat.
D. " Stamping out " method is used.
118. Which organs are more inflamed in bird flu?
A. Respiratory, digestive organs.
B. All hemopoietic organs.
S. Nerve system, basically centers.
D. Subtraction and reproductive organs.
119. Poultry flu in the literature how called?
A. Exudative, y European plague, classic plague.
B. Croupous pneumonia, black death.
S. Hemophilic plague, necrotic pneumonia.
D. Liver necrosis, kidney softening.
120. Who are the Russian scientists who studied bird flu perfectly?
A. M. Tartakovsky, VN Syurin, NG Ozidze.
BIP Pavlov, IM Sechenev, RV Petrov.
S. Blondlo, Gols, R. Virchow.
DKI Skryabin, NV Badanin, II Arkhangelsky.
121. What is the causative agent of bird flu?
A. Orthomyxoviride, DNA storage, InfluenzavirusA.
B. Poxoviride, DNA protector, neurotrope.
S. Coronoviride, RNA protector, pantotrope.
D. Herpesviride, DNA-preserving, epitheliotropic.
122. Human influenza virus A_2- in domestic and wild birds, or vice versa, is the avian influenza virus in humans?
A. It has been experimentally proven.
B. Sometimes 3-5% occurs.
S. No, not studied.
D. It depends on many factors.
123. What is the morbidity and mortality rate of bird flu?
A. Morbidity is 80-100%, death is 10-90%.
B. Morbidity 40-50%, death 10-20%.
S. Morbidity 50-60%, death 20-30%.
D. Morbidity 60-70%, death 40-50%.
124. In which case is the diagnosis of bird flu considered correct?
A. Diagnosis based on the results of GAR, NR, GATR, IDR, IFR.
B. Based on epizootological, clinical, pathanatomical results.
S. Infected embryos - the diagnosis.
D. All answers are correct.
125. Bird flu should be distinguished from what diseases?

A. Newcastle, infectious bronchitis, infectious laryngotrachitis, pasteurellosis, mycoplasmosis.
B. Salmonellosis, colibacteriosis, eimeria.
S. Pasteurellosis, anthrax, bradzot.
D. Marek, protein, smallpox, bronchitis.
126. In which answer is the causative agent of fowl pox correctly written?
A. DNA storage, aviapoxviride, chicken, hyena, canoreika species.
B. RNA storage, coronavirus, chicken, duck, turkey.
S. DNA storage, rhabdovirus, goose, chicken, duck.
D. RNA storage, herpesvirus, turkey, chicken, hyena.
127. Where are colostral antibodies in smallpox?
A. In newly hatched chicks.
B. In chicks 10-30 days old.
S. in chicks 20-21 days old.
D. In a stationary oven - in chicks up to 1 month old.
128. Which clinical-syndromatic type of smallpox is most common in poultry?
A. Diphtheritic smallpox.
B. Combined (slivnoy) smallpox.
S. Hemmorrhagic smallpox.
D. Fibrinous pox.
129. What changes are observed in the smear prepared in smallpox?
A. Guarnielli, Bollinger inputs (set of viruses).
B. Babesh, Negri, abortive, atypical.
S. Hemophilic, lipolytic, neurolytic.
D. Phagolytic, cytopathogenic.
130. Poultry pox should be distinguished from which diseases?
A. Infectious laryngotracheitis, infectious bronchitis, candidomycosis, aspergillosis, avitaminosis A.
B. Pasteurellosis, Salmonellosis, colibacteriosis.
S. ILT, IB, escherichia, eimeria.
D. Plague, flu, marek, newcastle.
131. What antibodies appear when vaccinated against smallpox?
A. _ Virus-neutralizing, complement-binding, precipitating antibodies.
B. Cell and tissue immunity.
S. Humoral and macrophage immunity.
D. Specific and non-specific immunity.
How long does immunity last when vaccinated with the N-27 strain vaccine of VITI in chicken pox ?A_3
A. 4 months for chicks, 9-10 months for chickens.
B. Chicks 6 months, chickens 1 year.
S. 1 month in chicks, 2 years in chickens.
D. 5 months in chicks, 1 year in chickens.
133. The rule and procedure for canceling quarantine in case of poultry pox?

A. 2 months after the last patient has disappeared, the final disinfection is carried out... it is canceled.
B. 4 months after the end of the illness.
S. 6 months after the end of the illness.
D. 45 days after the end of the illness.
134. When and in which country was infectious laryngotracheitis first studied?
A. 1925 in the USA.
B. 1900 in Spain.
S. 1880 in Italy.
D. 1910 in England.
135. Who and when experimentally studied infectious laryngotracheitis in Uzbekistan?
A. 1970 BQ Kochkarov.
B. 1960 AO Oripov.
S. 1954 Sh.A. Azimov.
D. 1952 AK Sitdikov.
136. In which answer, the causative agent of infectious laryngotracheitis is correctly indicated?
A. DNA storage, herpesvirus.
B. DNA storage, coronavirus.
S. RNA storage, poxvirus.
D. RNA storage, epithelitropvirus.
137. How long does virus transmission last in birds that have recovered from infectious laryngotracheitis?
A. Up to 2 years.
B. Up to 1 year.
S. Lasts for life.
D. 3-4 years.
138. The trophic classification of the causative agent of infectious laryngotracheitis is given correctly in which answer?
A. Epileutrop.
B. Myotropic.
S. Neurotropic.
D. Sarcotrope.
139. In what answer is the classification according to the clinical-syndromal course of infectious laryngotracheitis correct?
A. Laryngotracheal, conjunctival form.
B. Pulmonary, enteral.
S. Parenchymal, gastroenteral.
D. Urogenital, pulmonary, hepatopathological.
140. How long does immunity last when vaccinated against infectious laryngotracheitis? Where is the vaccine sent?
A. Applied to the cloacal mucosa, immunity until the end of life, lifelong.
B. Under the skin, on the neck, lasts for 1 year.

S. Between the muscles, to the chest, lasts 2 years.
D. Skin feathers are plucked, scratched and vaccinated, lasting 4 years.
141. The procedure for canceling restrictions in case of infectious laryngotracheitis?
A. 2 months after the last patient has disappeared, the final disinfection is carried out and the restriction is taken.
B. 6 months after the termination of the disease.
S. 1 year after the termination of the disease.
D. 2 years after the termination of the disease.
142. When and in which country was infectious bronchitis studied in poultry?
A. In the USA, 1931.
B. in Germany, 1900.
In S. England, 1910.
D. in Italy, 1940.
143. In which answer is the pathogen of infectious bronchitis correctly written?
A. RNA storage, corona virus avium.
B. RNA storage, herpesviride.
S. DNA storage, poxviride.
D. DNA storage, coronavirus avia.
144. The age of chickens most susceptible to infectious bronchitis? What is the death rate?
A. Age up to 30 days, mortality 40-60%.
B. Age up to 60 days, mortality 80-100%.
S. Age up to 90 days, mortality 60-80%.
D. Age up to 4 months, mortality 70-80%.
145. What happens when chicken embryos are infected with infectious bronchitis?
A. Embryo development is inhibited, deafness (pacana) is observed.
B. Embryo development is accelerated, hyperkinesis is observed.
S. Embryo undergoes necrosis, dies.
D. Depends on how many doses are given.
146. How long do chickens that have recovered from infectious bronchitis remain carriers of the virus?
A. 49-105 days.
B. Up to 6 months.
S. Up to 1 year.
D. Up to 2 years.
147. How much egg production is reduced in case of infectious bronchitis?
A. 50% or more is reduced.
B. Decreases by 60% or more.
S. is reduced by 70% and more.
D. Decreases by 80% or more.
148. When chicks are infected with infectious bronchitis viruses, how long do clinical symptoms appear?
A. In 18-24 hours.
B. In 2-3 days.

S. Depends on the method of injury.
D. Depends on the dose of viruses.
149. How long is the incubation period when infected with infectious bronchitis under natural conditions?
A. 36 hours to 10 days.
B. 18-24 hours.
S. 2-3 days.
D. Up to 1 month.
150. What is the death rate when chickens with infectious bronchitis have symptoms of ephrit and nephrosis?
A. 57-70%.
B. 40-50%.
S. 70-80%.
D. 20-30%.
151. In chicken embryos infected with infectious bronchitis viruses, "smallness", "deafness" is observed on how many days?
A. In 6-9 days.
B. in 13-15 days.
S. in 4-5 days.
D. in 19-20 days.
152. In infectious bronchitis, the virus isolated from the paternal material is identified by which reactions?
A. NR, BGAR, IDR, IFR.
B. RP, IDR, KBR, PZR.
S. AR, PR, KBR, DPR.
D. Any serological reaction can be applied.
153. When do virus-neutralizing antibodies appear in infectious bronchitis and how long are they preserved?
A. Appears on the 11th day of the disease, lasts 483 days.
Appears on the 4th day of B. and is stored for 1 year.
Appears on the 6th day of S. and is stored for 10 months.
D. Appears on the 3rd day and is stored for 2 years.
154. Infectious bronchitis should be distinguished from which diseases?
A. ILT, Smallpox, Newcastle, respiratory mycoplasmosis.
B. Salmonellosis, escherichia, marek, bursitis.
S. Of all prion diseases.
D. From all viral infectious diseases.
155. Does a bird that has recovered from infectious bronchitis get infected with this disease a second time?
A. No.
B. Yes.
Q. It depends on many factors.
D. Depends on the virulence of the pathogen.
156. The rule to cancel the restriction in infectious bronchitis?

A. Final disinfection 3 months after the last patient has been eliminated.
B. 6 months after the last patient was eliminated.
S. 1 year after the last disease was eliminated.
D. 45 days after the last patient has been eliminated.
157. In what country was infectious bursitis studied for the first time?
A. 1957 in the United States.
B. 1920 in Italy.
S. 1910 in England.
D. 1930 in Hungary.
158. 2004 from infectious bursitis. What % of existing birds in Russia died?
A. 14%.
B. 20%.
S. 30%.
D. 42%.
159. Which definition given to the cause of infectious bursitis is correct?
A. RNA storage, one type.
B. DNA storage, herpesviride.
S. RNA storage, coronaviride.
D. DNA storage, poxviride.
160. Find the range of poultry most susceptible to infectious bursitis?
A. Broiler, 2-3 weeks to 11 weeks.
B. Loman-Brown up to 1 month.
S. Ru0Cq chickens, 1-2 weeks old.
D. All domestic chicken breeds, up to 1 year old.
161. Name the reservoir of viruses in infectious bursitis?
A. Black beetle, fly, rodents, mealworms, wild birds.
B. Fish, frogs, worms.
S. Flies, mosquitoes, blood-sucking insects.
D. All arthropods that live in water.
162. Infectious bursitis - Gamboro - which diseases should be distinguished?
A. IB, Newcastle, coccidiosis, avitaminosisE, K, toxic dystrophy.
B. Escherichia, Salmonellosis, ILT.
S. Bird flu, YUPPG, eimeria.
D. From all viral infectious diseases.
163. According to statistics, how much does the United States, England, and Italy suffer from avian leukosis every year?
A. USA $150 million, England $40 million, Italy $20 million.
B. $200-250 million.
S. $250-300 million.
D. $400-500 million.
164. 1970 How much has Russia suffered from avian leukemia?
A. 3 times the damage of all other diseases.
B. Equal to the damage of all other diseases.
S. Half the damage of remaining diseases.

D. 10 times more damage than other diseases.
165. In which answer is the causative agent of avian leukemia correctly written?
A. RNA storage, oncornavirus.
B. DNA storage, poxvirus.
S. RNA storage, herpesvirus.
D. DNA storage, coronavirus.
166. How many percent of poultry leukemia is lymphoid leukemia?
A. 80%.
B. 60%.
Q. 40%.
D. 30%.
167. How is avian leukemia treated?
A. Not treated, given to meat.
B. There is symptomatic treatment.
S. Gammaglobulin, convalescent blood.
D. Antibiotics, sulfonamides.
168. If leukemia is detected in a poultry farm, how many percent of the birds must be serologically tested?
A. 5%.
B. 10%.
Q. 20%.
D. 30%.
169. What is the correct answer for the causative agent of Marek's disease?
A. DNA storage, herpesvirus.
B. RNA storage, poxvirus.
S. DNA storage, oncocoronavirus.
D. RNA storage, coronavirus.
170. Classification of Marek's disease according to clinical manifestations?
A. Neural, ocular, visceral.
B. Myopathic, epiopathic, neuropathic.
S. Orthopedic, cranial, caudal.
D. Hormal, desmochorial, mesenchymal.
171. Tell the principle of exploitation of poultry houses in Marek's disease?
A. "Bariband", "baribush".
B. Filarbeiten vinikelessen.
S. " Stamping out ".
D. Well-well, out-out.
172. If all infectious diseases are taken as 100%, how many % corresponds to Salmonellosis?
A. 26-40%.
B. 10-14%.
S. 18-20%.
D. 20-24%.
173. What is the percentage of chicken Salmonellosis?

A. 80-90%.
B. 70-80%.
S. 60-70%.
D. 50-60%.
174. Before 1970, what was the name of Salmonellosis in poultry?
A. Typhoid, lack of money.
B. Escherichia coli, colibacteriosis.
S. Granulamatous, mucosa.
D. Bacteriosis, stachyobotriosis.
175. Who isolated Salmonella for the first time?
A. US scientists Smith, Salmon, 1885 from pig.
B. American scientist Retger, 1900.
S. Endo, Ploskirov, Italy, 1890 from a bird.
D. Gapten, Prion - 1910, Russia.
176. How is avian salmonellosis transmitted?
A. Alimentary, aerogenous, contact.
B. Vertical, horizontal, genital.
S. Sporadic, epizootic, exotic.
D. Mainly aerogenic.
177. What is the percentage of salmonella death in ducklings?
A. Up to 90%.
B. Up to 80%.
S. Up to 70%.
D. Up to 60%.
178. What other serological tests can be performed in addition to the Hemagglutination reaction with a drop of blood when diagnosing salmonellosis?
A. Coagglutination, IFA, PCR.
B. KBGAR, GAR, KTPR.
S. PR, AR, KBR, RDP.
D. IFA, PR, AR, KBR.
179. Poultry salmonellosis should be distinguished from what diseases?
A. Colibacteriosis, aspergillosis, Newcastle disease, pasteurellosis.
B. Escherichia, eimeria, marek, smallpox.
S. Psittacosis, eimeria, plague.
D. Influenza, infectious bursitis, Newcastle.
180. Explain the scientific basis of treatment against salmonellosis.
A. It is determined after the sensitivity to therapeutic agents is determined.
B. Antibiotics, sulfonamides.
S. Bacteriocidal and bacteriostatic agents.
D. Provitamin A, B (complex).
181. Name the clinical signs of poultry escherichia.
A. Diarrhea, enterotoxemia, sepsis.
B. Abscess, phlegmon, furunculosis.
S. Asphyxia, asthesia, ataxia.

D. Hemorrhage, hyperemia, diarrhea.
182. What antigens are found in the causative agent of poultry escherichia?
A. O, K, H - antigens
B. Endo, exotoxin.
S. A, S, O - antigens.
D. Protein, lipoid, lipoprotein.
183. What is the death rate in poultry (young chicks) if Escherichia and Eymeriosis occur together?
A. 80-100%, 2006. RB Davlatov.
B. 70-80%, 2001. FA Niyazov.
S. 50-60%, 2000. AI Ibragimov.
D. 40-50%, 1992. AO Aripov.
184. Poultry escherichia should be distinguished from which diseases?
A. Salmonellosis, pasteurellosis, respiratory mycoplasmosis.
B. Gripp, Newcastle, marek.
S. Bursit, YUPPG, Gamboro.
D. Salmonellosis, flu, scurvy.
185. How to cancel the restriction on poultry escherichia?
A. Final disinfection 2 months after the last patient has been eliminated.
B. 45 days after the last patient has been eliminated.
Q. Last ill no 30 days from date after
D. Last ill no 21 days after making.
186. Respirator mycoplasmosis disease name when acceptance done?
A. 1961 XEB at the congress.
B. 1934 Microbiologicals in the association.
S. 1920 XEB at the congress.
D. 1900 XEB in the assembly.
187. Poultry Escherichia coli trigger how toxin separates?
A. Exo, endotoxin.
B. a, b, g toxins.
S. Proteolytic, lipolytictoxin.
D. Epilitrop, polytroptoxin.
188. Mycoplasmagallisepticum which disease trigger?
A. Respirator mycoplasmosis.
B. Marek.
S. Bursitis.
D. Infection laryngotracheitis.
189. Micro of the climate very suboptimal to be infection pathology how effect enough?
A. Strengthens.
B. Attenuates.
S. To the stimulus the majority factors participates.
D. This macroorganism tolerance depends.
190. Respirator in mycoplasmosis ill chickens what will be done?

A. Killed and burned.
B. Special, in insulators is treated.
S. Mandatory to the meat is submitted.
D. Feeding and then consumption is allowed.
191. Itlarolatin trigger who, when determined?
A. 1905 French scientist Karre.
B. 1926 Dunkin, Laidlaw.
S. 1932 MirolYubovI.
D. 1938 SyurinN.V., PankovV.A., LyuboshenkoS.Ya.
192. Dogs plague trigger which in the answer right written?
A. RNA preservative, paromyxoviride.
B. DNA preservative, poxviride.
S. RNA storage, oncoviride.
D. DNA storage, herpesviride.
193. The antigenic characteristics of the causative agent of canine plague are similar to which causative agents?
A. People measles, cattle plague
B. Influenza, smallpox, rabies.
S. Infectious hepatitis, influenza, Newcastle.
D. Bursitis, infectious bronchitis, ebola.
194. Dogs in the plague breeder in dogs scientist what %?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
195. In nature dogs of the plague reservoir tell me
A. Eat not and eat dogs.
B. All rodents.
S. Ticks, ticks, blood-sucking insects.
D. Wild predators.
196. % to the index of contagion of canine diseases?
A. 70-100%.
B. 50-60%.
S. 40-50%.
D. Depending on the factors.
197. According to the leading clinical signs, what is the form of canine plague?
A. Lungs, intestines, nerves, skin, mixed.
B. Respiratory, mixed, genital.
S. Neurosis, subclinical, immune subinfection.
D. Complex syndromes, reproductive.
198. What are the 6 symptoms that must be taken into account in canine distemper, and how many of them are considered to be a valid diagnosis?
A. Diarrhea, shortness of breath, eye-nose inflammation, palm perkeratosis, neurosis, the disease lasts at least 3 weeks. 4 of them.

B. Diarrhea, atelectasis, epilepsy, emphysema, aphonia, ataxia, 3 of them.
S. Conjunctivitis, ataxia, asthesia, hydrophobia, agalactia, adynamia, 4 of them.
D. Hydrophobia, diarrhea, hemorrhage, sepsis, septicemia, ataxia, 3 of them.
199. Which answer is correct about the 3 components of the epizootic chain?
A. The causative agent of an infectious disease, the route of transmission to the body of a susceptible animal, a susceptible animal.
B. Alimentary, aerogenous, transmissible, genetic, sexual.
S. Horizontal, vertical.
D. Respiratory, conglomerative, contact way.
200. How is an infectious disease?
A. Clinical symptoms are clearly manifested, in the form of latent, immune subinfection.
B. Typical, atypical, abortive.
S. Acute, acute, semi-acute, chronic.
D. Reactive, areactive, tolerant.

2-Interim assessment test questions

1. What method is used to diagnose infectious diseases in the case of immune subinfection?
A. Diagnosis is made by the method of determining the titer of antibodies in the blood of a sick animal.
B. With bacteriological, virological, mycological examinations.
S. With RA, RP, KBR, RGA, GATR, IDR.
D. By the method of allergic examination.
2. What are the methods of malleinization and tuberculinization?
A. Oftalmoprobe, between the skin, under the skin.
B. RA, MRA in a glass slide, test tube.
S. With all seroreactions.
D. With special diagnostics.
3. In what case is the diagnosis of an infectious disease considered correct?
A. If there is a laboratory test report.
B. Epizootological, clinical, pathanatomical.
S. Pathohistological, pathanatomical.
D. Rengenoscopy, fluoroscopy, lazeroscopy.
4. Who created the allergy diagnostic drug Mallein?
AO Kalning.
BIS Andriyevsky.
SMA Dukalov.
DAX Sarkisov.
5. Who created the tuberculin allergic diagnostic drug?
AXI Gelman.
BO Cal's.

SSN Vishelesky.
DMA Dukalov.
6. BCG vaccine against tuberculosis is used to vaccinate people and, if necessary, also animals. What does the word BSJ mean?
A. Initial letters of the words Bacterium, Kalning, Gelman in Latin spelling.
B. Biopreparat, sovremenn iy, jivotnix.
S. Biological, serum, zhivotnix.
D. Biopreparation, svobodenium, gelatin.
7. Who created Etis-1, Etis-2 drugs, a prophylactic and therapeutic agent against tuberculosis ?
A. GX Mamadullayev.
BS Salimov.
SQN Norboyev.
DAO Oripov.
8. What is another name for follicular encephalopathy of cattle?
A. False escape.
B. False protein.
S. Fake Auyeski.
D. Bloody dysentery.
9. If all diseases are 100% according to the statistics of the Ministry of Health, how many of them are infectious diseases?
A. 0.3-0.5%.
B. 7-10%.
S. 14-16%.
D. 18-20%.
10. What kind of infection is called if the point of entry of the causative agent into the susceptible organism is not determined?
A. Cryptogenic infection.
B. Regional infection.
S. Super infection.
D. Latent infection.
11. What is the name of an infection that can be found everywhere?
A. Ubiquitous infection.
B. Soil infection.
S. Water-air infection.
D. Natural infection.
12. How many diseases do rodents transmit to humans as reservoirs of infectious diseases?
A. About 20 - rabies, listeriosis, leptospirosis, tularemia, Auyeski...
B. More than 10.
About 30 S.
D. 10-12 diseases.
13. How many viral diseases have been found to be transmitted to humans by flies?
A. More than 150.

B. About 100.
Around S. 40-50.
D. to 20-30.
14. How many veterinary border points are currently operating in our country?
A. 45.
B. on the scale of 14 Republics.
S. 8 provinces.
D. 12 railways, 11 airports.
15. Which method is the injection of hyperimmune blood serum?
A. Special prevention, special treatment.
B. General prevention.
S. General treatment.
D. Modern prevention, modern treatment.
16. What measure is used when an infectious disease is registered?
A. Quarantine, restriction, declared unhealthy.
B. Disinfection, disinsection, deratization.
S. Urbanization, dehydration, deacarization.
D. Termination of all communications.
17. Is it possible to sell healthy animals outside quarantined farms?
A. No, it is not possible.
B. You can check it again and then sell it.
S. If the serological reaction is healthy.
D. If it comes out healthy in IFA, PCR.
18. Give an example of pathogens that are highly resistant to the effects of disinfectants.
A. Anthrax, anaerobic dysentery, bradzot, enterotoxemia, malignant tumor, scabies and other spore infections.
B. All clostridia.
S. All mycoplasmas.
D. All rickettsiae.
19. Biological method of disinfection.
A. Detoxification of manure by biothermal method.
B. Use of special cameras.
S. Use of UBN, IQN.
D. Application of thermal, chemo, partial pressure.
20. After how many days can an anthrax-vaccinated animal be slaughtered for meat?
A. After 14 days.
B. After 21 days.
S. After 3 days.
D. After 7 days.
21. Who discovered that there are several types of viruses that cause protein disease?
A. Corret, Valle 1922.
B. Lefler, Frosh 1898y.
SD Fracastro 1546.

D. Blondlo, Basov 1842.
22. Tell me what materials are taken from the animal during protein disease?
A. Liquid scraped from aphids.
B. Oral fluid, milk, urine of a sick animal.
S. Sick animal feces, urine.
D. Blood, feces of a sick animal.
23. How do protein disease serovars differ from each other?
A. Variants with shell amino acids.
B. Variant capsomeres by molecular weight.
S. With its clinical course in animals.
D. With which animal sensitivity.
24. How long does it take for antibodies to form after exposure to the protein disease virus?
A. After 4 days.
B. After 10 days.
Q. After 20 days.
D. After 10-14 days.
25. How many days before the appearance of clinical symptoms, rabies viruses begin to be shed in saliva?
A. 10 days ago.
B. 20 days ago.
S. 3-4 days ago.
D. 12-14 days ago.
26. How is protein disease in piglets, what is the death rate?
A. Myocarditis without aphthous, gastroenteritis 60-80% mortality.
B. Neurosis, neuritis, nephritis, death 90-100%.
S. Polymyelitis, caprostasis, mortality 60-80%.
D. Diarrhea, keratoconjunctivitis, mortality 90-100%.
27. VM Zhukorev found that 76.8% of the animals recovered from protein how long the immunity lasts?
A. 6 years.
B. 4 years.
S. 5 years.
D. 3 years.
28. What is the advantage of a universal vaccine made against protein over a regular vaccine?
A. The efficiency is 100%, the amount of consumption is small.
B. Vaccination is carried out by aerosol method.
S. At least 4 types are prevented.
D. No complication of vaccination.
29. How long will it take to vaccinate against that type and serovariant in the area where the protein came out?
A. 2 years - continuous.
B. 4 years - continuous.

S. 5 years - in a row.
D. 6 years - in a row.
30. Who is the scientist who used the name tuberculosis in science and when?
A. France, 1819. Lennik.
B. Hippocrates - VI century.
S. Abu Ali Ibn Sina - XI century.
D. Aristotle - VIII century.
31. What is the average annual percentage of cattle in Uzbekistan that is tested by an allergic method against tuberculosis, who determined it?
A. 0.016%, GH Mamadullayev.
B. 3-5%, AP Lee.
S. 2-3 %, VI Kan.
D. 6-8%, AK Sitdikov.
32. What happens to an animal with tuberculosis?
A. Delivered to meat within 15 days.
B. Killed and burned.
S. The meat is disinfected and transferred to the sausage.
D. " Stamping out " method is used.
33. Ascoli reaction on the skin is used to diagnose which disease?
A. Anthrax.
B. Dermatoses.
S. Dermatomycoses.
D. Tuberculosis.
34. State the merits and demerits of the Rose Bengal test.
A. A sensitive reaction is performed in a short time in 2-3 minutes, it is impossible to distinguish between naturally diseased and vaccinated animals.
B. High reliability, only naturally diseased animals are detected.
S. The level of reliability is low, only vaccinated cattle are detected.
D. It is not always possible to find a Rosbengal diagnostician, we spend little time.
35. Name the allergic diagnostic drugs used to detect tuberculosis.
A. PPD for cattle, PPD for poultry, LOW.
B. Tuberculin, cachegin, alttuberculin.
S. Allergen, urobilin, stercobilin.
D. Primary and Secondary PPDs.
36. Mycobacterium t. bovis are birds prone to?
A. No.
B. Prone.
S. Chicks up to 2 months are prone.
D. Sometimes you can get sick.
37. What type of viruses does the polyvalent vaccine used for protein disease prevention prevent?
A. A, O, Asia-1.
B. A, O, S.
Q. Sat-1, Sat-2, Sat-3.

D. A, Asia-1, Sat-1.
38. After the recovery of the last sick animal in protein disease, the final disinfection is carried out and quarantine is canceled after how many days?
A. 21 days.
B. 14 days.
S. 30 days.
D. 45 days.
39. How much more time does vaccination have to be carried out on a registered protein farm?
A. At least 2 years.
B. 4-5 years.
S. 2-3 years.
D. This is determined by a thorough examination.
40. According to WHO, WHO, how many people die of tuberculosis in the world in one year?
A. 4 million people.
B. 10 million people.
S. 1-2 mln.
D. 2-3 mln.
41. According to statistics, what percentage of people with brucellosis are correctly diagnosed?
A. 10-20%.
B. 20-30%.
S. 30-40%.
D. 40-50%.
42. Which serological reactions are often used in the laboratory diagnosis of brucellosis in veterinary practice?
A. Probasi of Rosebengal, AR, KBR.
B. IFA, PCR, RP.
S. GAR, GATR, RP, Askoli.
D. Askoli, Rosebengal, KBR.
43. How much is a positive reaction for cattle, sheep, goats, and pigs in AR - brucellosis?
A. 1:100 in cattle, 1:50 in small cattle.
B. 1:50 in cattle, 1:100 in small cattle.
S. 1:200 in cattle, 1:400 in small cattle.
D. 1:25 in cattle, 1:100 in small cattle.
44. How is the reaction of formation of milk in milk carried out if there is no diagnosticum?
A. Transfer is not possible.
B. Determinable but not reliable.
Q. Can be detected in urine.
D. Can be detected in feces.
45. In vaccination against brucellosis m.sh.h. What strain is the vaccine used for?

A. Rev-1 strain.
B. Strain - 19.
S. Strain - 68.
D. Strain - 62.
46. What strain is used in the vaccine used to vaccinate cattle against brucellosis?
A. Strain -19.
B. Rev-1 strain.
S. Nevsky - 12.
D. Strain - 68.
47. When and where was the infectious epididymitis of rams diagnosed?
A. 1942, New Zealand, Australia.
B. 1902, Italy, Hungary.
S. 1910, England, Norway.
D. 1860, Germany, Czechoslovakia.
48. Infectious epididymitis of rams should be distinguished from which diseases?
A. Diplococcosis, compilobacteriosis, salmanellosis, listeriosis, chlamydiosis.
B. Vibriosis, pasteurellosis, tuberculosis.
S. Aujeski, plague, escherichia.
D. Dermatomycosis, salmanellosis, vibriosis.
49. Rams What to do with an animal with infectious epididymitis?
A. Submitted to the meat.
B. Killed and burned.
S. Only testicles and internal organs are disinfected.
D. Treated, with hyperimmune blood.
50. What happens if the rabies virus is injected into a turtle?
A. It loses its pathogenicity.
B. Rash gets sick.
S. The sent site becomes necrotic.
D. Allergy in the turtle, anaphylaxis.
51. What diseases must be distinguished from rabies?
A. Auyeski, canine distemper, listeriosis, equine infectious encephalomyelitis.
B. Aujeski, follicular encephalopathy, pasteurellosis.
S. Visna-medi, Acobaltosis, pestilence.
D. Inan, leptospirosis, listeriosis.
52. Is there an oral rabies vaccine?
A. Boron, an improved granular antirabies vaccine.
B. No, not created.
S. Developed in the USA.
D. Yes, but ineffective.
53. In rabies, how long after the last diseased animal is destroyed is the restriction lifted?
A. After 2 months.
B. 1 month later.
Q. After 40 days.

D. 6 months later.
54. In Aujeski's disease, strong itching is not observed in which animals?
A. Pig, marmot, marten.
B. Horse, donkey, mule.
S. Even ungulates.
D. Carnivores.
55. What is the death rate in Aujeski's disease in young animals?
A. 80-90%.
B. 70-80%.
S. 60-70%.
D. 50-60%.
56. What reactions are used in the serodiagnosis of Aujeski's disease?
A. IDR, KBR, IFR, BGAR.
B. RA, RP, RDP, KBR.
S. Askoli, RDP, RP, RA.
D. All serotests can be used.
57. What is the vaccine for Auyeski's disease?
A. BUK - 628 strain vaccine.
B. GOA, GOAF, live vaccine.
S. Leophile, leophobic vaccine.
D. Associated formal vaccine.
58. In what form does chicken pox occur?
A. 90-94% of diphtheria.
B. Vesicle - papule form.
S. Hemorrhagic, gonino-fibrinous.
D. Fibrinous, purulent, hemorrhagic.
59. What kind of inclusions appear in cells in smallpox?
A. Bollinger, Guarniella.
B. Babesh, Negri.
S. Emphysema, Atelectasis, Diuresis.
D. Hematopoiesis, lymphopoiesis.
60. Vesicular stomatitis is included in which group by XEB?
A. A group, extremely dangerous infectious diseases.
B. B - group, dangerous infectious diseases.
S. Anthropozoonous diseases.
D. Zooanthroponous diseases.
61. Who studied leptospirosis in the veterinary department of Uzbekistan?
ANJ Khudoiberdiyev, N. Shutyayev, E. Yaparov.
BAK Sitdikov, ID Burlussky, A. Abdusattarov.
S. Sh.A. Azimov, FI Ibodullayev, RE Sosov.
DAO Oripov, A. Rozimurodov, BS Murtazin.
62. Optimal conditions for leptospira?
A. Hydrobiont.
B. Thermostable.

S. Thermolabile.
D. Neuro, myotropic, anaerobic.
63. EChT in leptospirosis - what is acceleration related to?
A. Erythropenia, globulinemia.
B. Erythrocytosis, leukemia.
S. Poikilocytosis, hemoglobinemia.
D. Hemophilia, albumenemia.
64. Has special prevention of necrobacteriosis been developed?
A. 1997 AA Sidorchuk and others. Nekovak.
B. 1950 S. Ya. Lyubashenko, GOAF vaccine.
S. 1920 AX Sarkisov, LTF.
D. 1970 AA Konopatkin, GOAF vaccine.
65. What antigens have been isolated from Listeria?
A. 15 somatic, 5 antigens.
B. About 10 antigens.
S. About 30 antigens.
D. 8 antigens.
66. What should be done to increase the effectiveness of therapeutic agents?
A. Determine their sensitivity and then use it.
B. Administration of broad-spectrum drugs.
S. Administer the dose 2-3 times larger.
D. Carrying out therapy together with autohemotherapy.
67. What are the types of tularemia?
A. USA, Tuyare Governorate California, European type, Asian type.
B. England, Netherlands.
S. Hungary, Bulgaria, Italy.
D. Canada, USA, Australia.
68. How many species of animals have been diagnosed with tularemia?
A. 125 species of vertebrates, 101 species of invertebrates.
B. All rodents and farm animals.
S. All kinds of insects and wild animals.
D. Humans and all creatures.
69. Which type of farm animals is highly susceptible to tularemia?
A. Sheep and lambs.
B. Cattle and calves.
S. Pigs and carnivores.
D. Wild animals and carnivores.
70. What diseases should be distinguished from tularemia?
A. Anaplasmosis, pseudotuberculosis, tuberculosis, coccidiosis, brucellosis.
B. Hemosporidiosis, pasteurellosis, escherichia.
S. Ringworm, botulism, listeriosis, leptospirosis.
D. Aujeski, protein, smallpox, ephemeral fever.
71. Can the tularemia vaccine be used to vaccinate animals?
A. No, ineffective.

B. Yes, it will.
S. It is possible for cattle.
D. Available to carnivores.
72. Who and when invented the vaccine for humans against tularemia?
A. 1946 B. Ya. Elbert, NA Gaysky.
B. 1912 Stanton, Fletcher.
S. 1954 Verge, Perimura.
D. 1932 Olds, Lewis.
73. What is another name for melioidosis?
A. False manga, Stanton, Fletcher's disease, Rangoon manga, pneumoenteritis.
B. Epizootic lymphangitis, dermatomycosis.
S. Purulent sap, yellow mite.
D. Persistent fever, fibrillation.
74. What is the difference between melioidosis and manga?
A. With bacteriological examination and KBR.
B. With all serotests.
S. Allergy test.
D. Pathomorphological, pathogistological.
75. What are the special methods for treating melioidosis?
A. No specific treatment has been developed.
B. Symptomatic treatment is used.
S. Antibiotics, sulfonamides are used.
D. Convalescent blood and immunoglobulins.
76. What infection is the causative agent of the layer?
A. Wound infection.
B. Alimentary infection.
S. Respiratory infection.
D. Toxin infection.
77. What kind of toxin does the causative agent of the layer secrete?
A. Neurotoxin - tetanospasmin, hemotoxin tetanolysin.
B. Exo, endotoxin.
S. Mio and epidermotoxin.
D. Fibrino, peptidotoxins.
78. What special biopreparations have been developed against plaque?
A. Anotoxin (anti-inflammatory) half subcutaneous, half intravenous.
B. Immunoglobulin.
S. Hyperimmune serum.
D. So far no effective drug has been developed.
79. What kind of diseases should be distinguished from shingles?
A. Rabies, from acute rheumatism of the muscles.
B. Auyeski, pestilence, protein, smallpox.
S. Pasteurellosis, salmanellosis, colidyspepsia.
D. Escherichia, meningitis, epilepsy.
80. Name the clinical signs of rabies.

A. Lower jaw, paralysis of legs, salivation, red eyes, aggression, hydrophobia.
B. Ataxia, asthesia, atonia, asthenia.
S. Chewing all objects, diarrhea.
D. Hydrophobia, blindness, fear.
81. How to prevent the layer?
A. The injured animal is injected with an anti-fog anatoxin within 12 hours.
B. The wound is washed with disinfectants.
S. Aseptic, antiseptic agents are used.
D. It is placed in a warm room, glucose 40% is sent.
82. What was botulism called when it was first discovered?
A. 1820-1822. Kerner, Sausage poisoning.
B. 1900 IA Andrevsky, tetany.
S. 1842 A. Basov, meningitis.
D. 1910 IM Sechenev polyneuritis.
83. What does the word botulus mean in Latin?
A. Sausage.
B. Spoiled meat.
S. This tribe.
D. Poison.
84. How many types of toxins have been isolated from the causative agent of botulism?
A. 7 different.
B. 5 different.
Q. 3 different.
D. Exo and endotoxin.
85. Is the causative agent of botulism thermolabile or thermostable?
A. Thermostable.
B. Thermolabile.
S. This condition depends on the RN - environment.
D. Depends on the toxin released by the pathogen.
86. Which of the laboratory animals are sensitive to botulism toxin?
A. White mouse, guinea pig, rabbit.
B. Cats, puppies.
S. All young birds.
D. All animals are sentient.
87. What is the death rate in botulism?
A. 70-95%.
B. 50-60%.
S. 40-50%.
D. 100%.
88. Explain the mechanism of action of botulism toxins.
A. Acetyl choline is not released from the parasympathetic nerve endings, and communication with the central nervous system is interrupted, muscle tone decreases, angina pectoris in the heart, asphyxiation in breathing, and the animal dies.

B. As a result of cytopathogenic effect, myocarditis, endocarditis, epicarditis cause death.
As a result of S. Meningitis, the activity of the Kiss-Flyaka nerve node is disturbed, death occurs.
D. Angina, due to the failure of the ligament of Hissa, causes death.
89. What diseases should be distinguished from botulism?
A. Anthrax, rabies, Aujeski, listeriosis, stachyobotriotoxicosis, various poisonings, postpartum paralysis, acetonomy, Newcastle, Marek, canine plague.
B. Karason, bradzot, enterotoxemia.
S. Fodder poisoning.
D. Neuritis, neurosis, polymyelitis, epilepsy.
90. Can botulism be treated?
A. At the beginning of the disease, 600-900 thousand SB of special antitoxic serum is sent to horses.
B. Inefficient.
Q. It depends on the qualification of the treatment.
D. It can sometimes work.
91. When and in what country was the disease of ku-fever studied for the first time?
A. 1909-1910 USA, Ricketts.
B. 1812 France, N. Banaparte.
S. 1940 Russia, IP Pavlov.
D. 1970 Russia, RF Sosov.
92. Who are the reservoir of ku-fever disease?
A. All animals and ticks.
B. Wild animals and insects.
S. Rodents, flies.
D. Most animals that live in water.
93. What treatment methods are used in ku-fever?
A. Symptomatic (antibiotics, sulfonamides).
B. Special, hyperimmune blood serum.
S. Immunoglobulin, convalescent blood.
D. Treatment does not work.
94. What is the death rate in ku-fever disease?
A. Death is rare or rare.
B. 40-50%.
S. 20-30%.
D. 60-70%.
95. When and who diagnosed infectious hydropericarditis?
A. 1925 Cowdrey.
B. 1900 F. Majandi.
S. 1910 LD Vinci.
D. Hippocrates, Aristotle BC.
96. What is the death rate in infectious hydropericarditis?
A. 50-90%, sometimes 100%?

B. Death is not observed.
S. 20-30%.
D. 30-40%.
97. Infectious hydropericarditis-way of transmission?
A. Transmissive, iksod mites.
B. Alimentary, respiratory.
S. Retrospective, parenteral.
D. Vertical, horizontal, cryptogen.
98. Who and when was the first to detect iron deficiency in human hair?
A. 1841-1843. AA Cazenov.
B. 1845 Malstem.
S. 1858 A. Ardi.
D. 1970-1980. AH Sarkisov.
99. Which vaccine is used for preventive and blood treatment purposes?
A. LTF-130.
V. GOA-formal vaccine against trichophytosis.
S. Yam and yam jams.
D. Iodine salts, antibiotics.
100. Who received the state award for his tetanus vaccine?
A. AH Sarkisov.
B. Sh.T. Rasulov.
SMP Parmanov.
DAB Lee.
1 01. A mixture of 7% alkali, formalin, vaseline is effective in the treatment of which disease?
A. Temiratkini.
B. Psoriasis.
S. Dermatoses.
D. Vitiligo.
102. Who prepared the vaccine against sheep distemper?
A. Trichovis vaccine, MP Parmanov.
B. Mentovak, Sh.T. Rasulov.
S. SP-1, AB Lee.
D. LTF, LTF-130, AX Sadkisov.
103. The definition of microsporosis disease is correctly written in which answer?
A. Pathogenic fungi belonging to the genus Microsporum cause damage to the skin and skin products (wool, hair, nails).
B. Infectious skin disease, the causative agent is pathogenic bacteria.
S. Skin disease, not contagious, the causative agent is a pathogenic fungus.
D. All people, animals have skin disease, wool falls, injuries are limited, the causative agent is bacteria.
104. From dermatomycoses - microsporosis is more common in which animals?
A. In horses.
B. Donkeys, in mules.

S. In ungulates.
D. In carnivores.
1 05. Who is the reservoir of microsporosis disease?
A. Rodents.
B. Insects.
S. Ticks, flies.
D. Wild birds.
1 06. Microsporosis should be distinguished from which diseases?
A. Iron, eczema, scabies, dermatitis.
B. From all dermatomycoses.
S. Of all dermatitis.
D. From tuberculosis, anthrax, brucellosis.
107. Who created a vaccine from a local strain in Uzbekistan against cattle distemper?
A. 2008 IX Salimov.
B. 2004 GH Mamadullayev.
S. 2005 XO Khamdamov.
D. 2010 BA Elmurodov.
108. According to AA Sidorchuk, what kind of toxins does the causative agent secrete?
A. Alpha, Betta, delta toxins.
B. Exo, endotoxins.
S. Neuro, cyto, myotoxins.
D. Hemo, neuro, epitheliotoxins.
1 09. Optimum conditions of the Karason trigger in relation to oxygen?
A. Strict anaerobic.
B. Facultative anaerobic.
S. Strict aerobic.
D. Facultative aerobic.
110. What is the correct answer to the description of the death of a herd of rabies?
A. Occurs sporadically, mortality up to 80%.
B. In the form of epizootics, mortality is 60%.
S. In the form of enzootic, mortality is 40%.
D. In the form of panzootic disease, mortality is 40-50%.
111. Which laboratory animals are biotested for Karasan disease?
A. To guinea pigs.
B. To the rabbit.
S. To the white mouse.
D. To cats and dogs.
1 12. When will the quarantine be canceled in Karasan disease?
A. After final disinfection 14 days after the loss of the last sick animal.
B. 21 days after the loss of the last sick animal.
S. 30 days after the loss of the last sick animal.
D. 45 days after the loss of the last sick animal.

1 13. How many hours after the death of an animal in Karasan's disease should the pet material be delivered to the laboratory with a referral letter?
A. within 2-3 hours.
B. Within 8-10 hours.
S. Within 12-18 hours.
D. Within 1 day.
114. After how long an animal that has recovered from the disease is allowed to be slaughtered for meat?
A. After 30 days.
B. After 10 days.
Q. After 20 days.
D. After 14 days.
115. How long after the elimination of the last animal in Karasan disease is the quarantine canceled?
A. After 14 days.
B. After 21 days.
S. After 30 days.
D. After 45 days.
1 16. What are the permanent, observable clinical signs of paratuberculosis?
A. Diarrhea, rectal sphincter paralysis, weight loss.
B. Fever, fibrillation, polyuria.
S. Diarrhea, abdominal paralysis.
D. Ataxia, Atonia, Asthesia, hydrophobia.
1 17. Who and when invented the experimental module of paratuberculosis disease?
A. 1906 Bang, in the calves.
B. 1895 Yons, in cattle.
S. 1937 PP Vishnevsky, in cattle.
D. 1949 KA Dorofeyev, in sheep.
1 18. What allergen is used in allergic diagnosis in paratuberculosis?
A. Poultry alttuberculini, between the skin, 0.2-0.4 ml.
B. Tuberculin PPD for cattle, 0.2 ml.
S. Tuberculin PPD for poultry, 0.2 ml.
D. LESS kit.
119. Which serological reaction is more reliable in diagnosing paratuberculosis?
A. KBR - 83.3%.
B. RA, RP - 70%.
S. RDP, CUBR - 80%.
D. All serological reactions.
1 20. What treatment methods have been developed for paratuberculosis?
A. No effective treatment has been developed.
B. 1926 Valle, Ranger produced a live vaccine, but it caused an allergy in the body.
S. Hyperimmune blood serum developed.
D. It is recommended to determine the sensitivity to antibiotics and treat it.
1 21. What kind of disease is campylobacteriosis according to its clinical course?

A. Genital, reproductive disease.
B. Neurosis, polymyelitis.
S. Gastroenteritis diseases.
D. Heart and vascular diseases.
122. Cattle between the ages of 3 months and 4 years are more susceptible to black disease, tell me the reason?
A. Colostral immunity is stable in calves up to 3 months old, and animals over 4 years old have been vaccinated 8-9 times, so the immune background is at a high level.
B. Calves up to 3 months of age are mainly fed milk and receive lactoglobulins.
S. Tolerance develops in animals older than 4 years.
D. Answers B and C are correct.
1 23. Name the main way of spreading campylobacteriosis.
A. Use of sick bulls - in escape.
B. Sick cows, calves and bulls.
S. Rodents, contaminated feed.
D. Blood-sucking insects, mites.
124. What organs of experimentally infected cattle contagious pleuropneumonia was used as a vaccine - L. Pasteur?
A. Subcutaneous lymph nodes.
B. Neck, mesenteric lymph nodes.
S. Brain.
D. Thyroid gland, adrenal glands.
1 25. What happens to an animal infected with rinderpest?
A. It is lost by killing and burning.
B. Treated in a special place.
Q. Only hyperimmune serum is effective.
D. Compulsory submission to meat.
126. How long after the cancellation of the quarantine in case of rinderpest is it allowed to release the animals?
A. After 4.5 months.
B. After 6 months.
Q. After 1 year.
D. After 2-3 months.
127. What age group of cattle is more likely to suffer from catarrhal fever?
A. 1-4 years old.
B. 2-8 years old.
S. 6-10 years old.
D. Age does not matter.
1 28. Catarrhal fever of poor quality mainly occurs in which condition?
A. 60-80% sporadic.
B. 60-80% epizootic.
S. 40-50% exotic.
D. 60-80% panzootic.

129. The brief definition of leukemia is written correctly in which answer?
A. Blood cancer, appearance of immature stem blood cells - hemocytoblast, lymphoblast, meeloblast, prolymphocyte, monoblasts in peripheral blood.
B. Occurrence of tumors in the reticulo-endothelial system.
S. Chronic infectious disease, occurrence of hemophilia.
D. Obvious appearance of hemorrhage, complete loss of coagulation.
1 30. Who has studied leukemia in depth in our country?
A. Professor XS Salimov and his students.
B. Professor AO Oripov and his students.
S. Professor BS Salimov and his students.
D. Professor FI Ibodullayev and his students.
1 31. Who is the scientist who founded experimental leuko-oncology in dogs?
A. Veterinary doctor, MA Novinsky 1876.
BA Shastny 1876.
S. Ye. Neumann in 1870.
D. R. Virkhov 1680.
132. Are leukosis viruses transmitted through ticks, bull, ram sperm?
A. No - XS Salimov, MQ Butayev.
B. O'tadi - KA Nizametdinova, ZE Roziyev.
S. Sometimes passes 20-30%, S. Primov.
D. This question is not resolved.
1 33. What % of pathology is observed in the spleen in cattle leukemia?
A. 100%.
B. 90%.
S. 80%.
D. 70%.
1 34. What is done with cattle infected with leukemia?
A. Compulsorily submitted to the meat.
B. Treated with panflavin.
S. Treated with autovaccine.
D. It is lost by burning.
1 35. Which serological reaction is effective in bovine leukemia?
A. REED.
B. RA, KBR.
S. RP, Reaction Askoli.
D. Rosebengal, RA.
136. After the last leukosis seropositive animal is handed over for meat, after what other tests is the restriction lifted?
A. 3 times with an interval of 3 months, if a complete negative result is obtained.
B. 2 times with an interval of 2 months, if a completely negative result is obtained.
Q. 2 times with a 6-month interval, if the result is completely negative.
D. 1 time after 1 year with a completely negative serological test result.
1 37. What is the name of infectious rhinotracheitis of cattle in the literature?

A. Acute infection of the upper respiratory tract, conjunctivitis, infectious pustular vulvaginitis.
B. Lobar, lobular bronchopneumonia.
S. Emphysema, atelectasis, pleurisy.
D. Chronic, purulent pleuropneumonia.
138. Who studied infectious rhinotracheitis in our country?
AIX Salimov, ZJ Shapulatova, G. Uzakova.
BGX Mamadullayev, AG'. Gafurov.
SXS Salimov, FI Ibodullayev.
DAO Oripov, Sh.A. Azimov, TB Boymurodov.
139. In which answer is the causative agent of infectious rhinotracheitis correctly written?
A. DNA storage herpesvirus BHV-1.
B. RNA-sparing poxvirus.
S. DNA-storing coronavirus.
D. RNA storage orthomyxovirus.
140. How many different stages of infectious rhinotracheitis according to clinical manifestations?
A. Respiratory, vesicular rash, conjunctival, meningoenciofelatic form.
B. Respiratory, enteral, genital.
S. Respiratory, neuritis, nephrosis, nephritis.
D. Pulmonary, genital, gastroenteritis.
1 41. IX Salimov in the treatment of calves with infectious rhinotracheitis - which drugs were 100% effective?
A. 1% baytril, iodinol and etonio suspension, with furozolidon.
B. Antibiotics (ampicillin, streptomycin).
S. Etonium, furozolidone, gentamicin.
D. Norsulfasol, streptomycin.
1 42. What kind of antibodies are produced in animals that have recovered from infectious rhinotracheitis?
A. VN, KB precipitating.
B. Hemagglutinating, VN.
S. KB, phagocytosis inhibitor.
D. Neutralizing, enhancing phagocytosis.
1 43. Who studied viral diarrhea of cattle in our country?
A. 1991 IX Salimov.
B. 1990 BA Elmurodov.
S. 1995 SI Mavlanov.
D. 1998. Sh.A. Jabbarov.
144. In which answer is the causative agent of bovine viral diarrhea correctly written?
A. RNA storage tag .
B. DNA-storing herpesvirus.
S. An RNA-storing poxvirus.
D. DNA-storing orthomyxoviride.

1 45. Cattle of which age are prone to viral diarrhea of cattle?
A. From 2 days to 2 years IX Salimov.
B. 2-4 months old.
S. Up to 6 months.
D. Up to 1 month.
1 46. State the rule for lifting the restriction in case of viral diarrhea of cattle.
A. The restriction is canceled after the final disinfection is carried out one month after the last sick animal has been eliminated (healed).
B. 45 days after the last diseased animal has been eliminated.
S. 2 months after the last diseased animal has been eliminated.
D. The restriction is lifted 14 days after the final disinfection after the last diseased animal has been eliminated.
147. IX Salimov - IRT, PG-3, experimentally determined the therapeutic effect of hyperimmune blood serum prepared against colisalmanellosis?
A. 90.6%.
B. 80%.
S. 70-80%.
D. 50-60%.
1 48. What vaccines and biopreparations are available to prevent cattle from PG-3 disease?
A. Paravac, Bivak vaccines, Hyperimmune blood serum.
B. Chumvak, Rabikan vaccines, Convalescent blood.
S. Mono, polyvalent, 20A - vaccines.
D. Live, dead, lyophilized, weakened.
1 49. Who was the first to identify cattle ephemeral fever in Uzbekistan?
A. 1984 It was defined by XS Salimov and A. Mengliyev under the name Termiz fever.
B. 1984, 2002, 2013. A. Oripov identified.
S. 1984, 2010. T. Boymurodov identified.
D. 1980, 2006. F. Ibodullayev identified.
1 50. How is cattle ephemeral fever disease transmitted and spread?
A. Through Culex annulirosis, Anopheles annulipes and other blood-sucking flies.
B. Horizontal, vertical.
S. Alimentary, aerogenous, genital.
D. Contact, respiratory, genital.
1 51. How many cattle on the farm get sick when cattle ephemeral fever occurs for the first time?
A. 75-100%.
B. 20-30%.
S. 40-50%.
D. 60-70%.
1 52. What vaccines and biopreparations are used for cattle ephemeral fever?
A. Inactivated cultured vaccine, hyperimmune and convalescent serum.
B. Mono, polyvalent vaccines.

S. Leophilic, leophobic, live vaccines.
D. Quartz vaccine with GOA.
1 53. What is the Danish meaning of Bradzot?
A. Lightning fast death.
B. Liver necrosis.
S. Kidney turning into a pasty mass.
D. The transformation of the lungs, kidneys, spleen into a thick clot.
1 54. What is the death rate in Bradzot's disease?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
1 55. What diseases does ClostriDium DiDas cause?
A. Bradzot, infectious necrotic hepatitis.
B. All clostridiosis.
S. Layer, botulism.
D. Dangerous swelling, gas gangrene, blackness.
1 56. Bradzot bioassay test in which answer is correct?
A. The wound material was washed in GPB, filtered, and injected into 2 guinea pigs, dying in 16-48 hours (0.5-1 ml).
B. Patmaterial suspension 0.5 ml is injected into a white mouse, it dies in 7-8 hours.
S. Petmaterial filtrate 0.5 ml is injected into kittens, they die in 8-10 hours.
D. The bedding material is sterilized and the filtrate is given to lambs 2-4 months old, they die in 1-2 hours.
1 57. What biopreparations are available for the prevention of Bradzot's disease?
AF Kogan, AP Kolesev - vaccine and colianatoxin. Immunity - 10 months.
B. Polyvalent concentrated vaccine against clostriosis.
S. GOA, farmol, inactivated vaccine.
D. Live and dead, concentrated vaccine.
158. Anaerobic enterotoxemia of sheep is the causative agent of the disease and another name for the disease is correct in which answer?
A. Renal relaxation, Cl. perfingens-C.
B. Kidney stiffness, Cl. tetany.
S. Liver necrosis, Cl. oeDimatiens.
D. Pulmonary emphysema, Cl. DiDas.
1 59. Cl. What kind of colonies does perfingen0C form in the environment?
A. S-smooth, Rgadir-here, M-mushy.
B. Convex, concave, uneven edges.
S. Dark, reddish-brown.
D. Coral, emerald systems.
1 60. Do people also get sick with anaerobic enterotoxemia of sheep?
A. Gets sick.
B. Does not get sick.
S. Sometimes he gets sick.

D. Get sick if experimentally infected.
161. Is it possible to vaccinate sheep against anthrax, brazot, enterotoxemia and smallpox at the same time?
A. It is possible.
B. Impossible.
S. The interval must be at least 14 days.
D. Possible in extreme cases.
1 62. Chlamydial abortion disease of sheep was studied by whom in Uzbekistan?
A. GN Nosirov, AK Sitdikov, A. Abdusattarov.
BFI Ibodullayev, M. Aminjonov, Sh.A. Azimov.
SJA Azimov, BS Salimov, EX Ergashev.
DOS Sodikov, Yo.Kh Torakulov.
163. What serological reactions are used in the diagnosis of chlamydial abortion of sheep?
A. CBR and CUBR.
B. RA, RP, RDP.
S. RA, Ascoli reaction.
D. All serotests can be performed.
1 63. What serotests are used to detect sheep-goat plague?
A. 5 years.
B. Until death.
S. 3 years.
D. 2 years.
1 64. What is the name of infectious catarrhal fever of sheep in the literature?
A. Blutang, blue tongue, catarrhal fever.
B. Death at lightning speed, steppe.
S. Sudden death, cirrhosis of the liver.
D. Black spleen, hemophilic necrosis.
1 65. What is the death rate in Blutang disease?
A. 0-70%.
B. 90-100%.
S. 40-50%.
D. 30-40%.
1 66. What special treatment was created in Blutang disease?
A. Not developed.
B. A hyperimmune serum was developed in the USA.
S. Polyanatoxin was developed in Australia.
D. Gamma globulin, interferon.
1 67. Infectious hoof rot disease of sheep should be distinguished from which diseases?
A. Necrobacteriosis, contagious ecthyma, smallpox, protein, pododermatitis, mechanical injury.
B. Brucellosis, tuberculosis, layer.
S. Rabies, Auyeski, Listeriosis.

D. Aqsil, emkar, olat, saramas.
1 68. What biopreparations are available for the prevention of infectious agalactia in sheep and goats?
A. Russian, Mongolian, Romanian vaccines.
B. England, Australia, USA vaccines.
S. Italian, French, Caucasian vaccines.
D. Mongolia, Kazakhstan, Russian vaccines.
169. For the vaccine developed for the prevention of infectious pleuropneumonia disease in goats, who received the state award of VITI - scientists?
A. IA Dukalov, IS Polkovnikova, FD Lukashenko, SP Ilinov.
B. Sh.A. Azimov, Orifjanov, Burlusky.
SAK Siddikov, RX Khaitov, XZ Ibragimov.
DJK Sayfutdinov, EX Irgashev.
1 70. What is the death rate in infectious pleuropneumonia of goats?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
171. Who and when found out that the people who were infected with camel plague in the deserts of Astrakhan and ate the meat of forcibly slaughtered camels were infected with plague?
A. 1907-1914. NI Klodnisky.
B. 1891 Kitazato, Yersin.
S. 1899 Junkovsky.
D. 1900 R. Koch.
1 72. Who are the reservoirs of causative agents of camel plague?
A. Sand mice, rats, rabbits.
B. Mice, bats, insects.
S. Flies, mosquitoes, ticks.
D. Reptiles, arthropods.
1 73. Can people get infected with the causative agents of camel plague?
A. Gets sick.
B. Does not get sick.
S. Sometimes he gets sick.
D. It depends on the pathogenicity, virulence of the pathogen.
1 74. Who invented the drugs Mallein, tuberculin?
A. 1891 Gelman, Kalning.
B. 1882 Leffler, Schuetz.
S. 1907 Schubert, Schubert.
D. 1900 Romonovsky, Gimza.
175. Who else gets sick with Manka disease besides odd ungulates?
A. Camel, people and lion, tiger, lynx, bear.
B. All farm animals.
S. All wild animals.

D. All animals become infected when experimentally infected.
1 76. Ways to inject mallein in manka disease?
A. Eye, interdermal, subcutaneous.
B. Optoprobe, venous vessels.
S. The skin is scratched and applied.
D. The inhalation method is used.
1 77. Animals that have a positive reaction to Mallein must be checked by which method?
A. KBR is placed.
B. RA, RP are placed.
S. IDR, CUBR.
D. RP, RA, Askoli.
1 78. What is the accuracy rate when Malleinization is injected between the skin?
A. 95%.
B. 85%.
S. 75%.
D. 100%.
1 79. What should be done with an animal suffering from manka disease?
A. He is killed and burned with his skin.
B. Treated in a special place.
S. "Stamping out" method is used.
D. Must be slaughtered for meat.
180. How many days after the last diseased animal in Manka disease is lost, the final disinfection is carried out and the quarantine is canceled?
A. 45.
B. 21.
S. 14.
D. 2 months.
181. Who and when invented that the causative agent of equine disease is a virus and pathological changes in blood-producing organs?
A. 1904 Carré, Valle.
B. 1843 Ligney.
S. 1859 Anginiard.
D. 1932 Kolyakov, Romanov.
1 82. What is the reason for EChT acceleration in INAN disease?
A. Erythropenia, globulinemia.
B. Albuminemia, poikilocytosis.
S. Leukocytosis, monocytosis.
D. Hemophilia, lymphocytosis.
1 83. Tell the factors that cause the spread of INAN disease.
A. Swamps, puddles, blood-sucking insects.
B. Drought, ticks, ants.
S. Wild birds.
D. Thorny, stony pastures.

1 84. How are animals infected with INAN disease treated?
A. It is not treated, it is destroyed.
B. With special hyperimmune blood sera.
S. With convalescent serum.
D. Sensitivity to therapeutic agents is identified and then treated.
1 85. INAN disease should be distinguished from which diseases?
A. Piroplasmosis, nuttaliosis, trypanasomosis, leptospirosis, rhinopneumonia.
B. Manka, dumb, layered.
S. Epizootic lymphangitis, influenza.
D. Botulism, anthrax, colic.
186. In which answer is the description given to the dumb disease of horses correct?
A. It is acute, contagion, fever, purulent inflammation of the nasal cavity, throat mucous membranes, abscess under the jaw - appears.
B. It is chronic, contagion, fever, wounds appear on legs, abdomen, hooves.
S. Chronic night, head, eye, ear, throat abscesses.
D. Swelling appears in the ventral, caudal part of the body.
187. Clinical-syndromatic course of deafness is written correctly in which answer?
A. Typical, complicated, abortive, genital.
B. Typical, atypical, internal, external.
S. Alimentary, respiratory, skin form.
D. Exogenital, endogenital.
188. In which answer is the trigger of the mute shown correctly?
A. Streptococcus.
B. Staphylococcus.
S. Diplococcus.
D. Polyetiological characteristic.
1 89. How long does the immunity of people who recover from muteness last?
A. Lifetime.
B. 1 year.
S. 2 years.
D. Up to 5 years.
190. Horses are the causative agent of influenza in which answer is written correctly?
A. RNA-storing orthomyxovirus.
B. DNA-storing herpesvirus.
S. RNA-sparing poxvirus.
D. DNA-storing coronavirus.
1 91. What is another name for rhinopneumonia in horses?
A. Viral abortion of females.
B. Equine conjunctivitis.
S. Respiratory pneumonia.
D. Pneumothorax, hydremia.
1 92. How is rhinopneumonia abortion different from salmonellosis abortion?
A. Abortion in salmanellosis 4-8 months, rhinopneumonia 7-11 months, laboratory examination.

B. In salmonellosis - necrotic abortion, in rhinopneumonia hemorrhagic abortion.
S. Salmonellosis is observed in abortion - paralysis in bia, hemophilia in rhinopneumonia.
D. Only IFA differs from PCR.
193. Which definition is correct for equine contagious pleuropneumonia?
A. Inflammation of liver, lungs and pleura.
B. Conjunctivitis, rhinitis, fibrinous stomatitis.
S. Diarrhea, caprostasis, polyuria.
D. Atonia, colic, ataxia.
1 94. In which answer is the treatment of KPP - disease of horses correctly written?
A. 3-4.5 g. novarsenol 1:20, 1:30 in warm distilled water, intravenously (do not go under the skin!).
B. Glucose 40%, caffeine benzoate - intravenously.
S. Oxytetracycline 500 tb/kg - intramuscularly.
D. Laxatives.
1 95. Horses KPP - what to do with an animal that died?
A. The skin can be removed, the body is burned.
B. is thrown into the Becker well.
S. Given to carnivores.
D. It is destroyed by incineration in a special place.
1 96. Tell the optimum environment of Salmonella abortus equi.
A. Aerobic, anaerobic grow in all nutrient media.
B. Grows aerobically in a medium supplemented with egg yolk and serum.
S. Grows in an aerobic medium with added glucose and starch.
D. Grows only in tissue culture media.
1 97. Is there a recurrence of abortion with salmanellosis?
A. It doesn't happen.
B. Meets.
S. Occurs occasionally.
D. Occurs as a secondary infection.
1 98. What is the death rate of African plague in horses?
A. 95%.
B. 85%.
S. 75%.
D. 100%.
1 99. Name the reservoir in African swine fever?
A. Argas mites.
B. Bat.
S. Blood-sucking insects.
D. Birds, worms.
2 00. What are the main clinical signs of infectious atrophic rhinitis in piglets?
A. Deformation of the nose, pneumonia, pleurisy.
B. Diarrhea, ataxia.
S. Inability to stand, polyuria.

D. Symptoms of encephalitis.

Test questions for final assessment.

1. What is another name for Au e ski disease?
A. False rabies, infectious bulbar paralysis, infectious meningoencephalitis.
B. Morbus Au e ski, pseudo rabies.
S. Polyencephalitis, myelitis, paralysis.
D. Hydrophobia, ataxia, asthesia.
Who discovered Au e ski disease and when?
A. 1902 A. Auski, Hungary.
B. 1900 F. Gutira, Y. Marek, Czechoslovakia.
S. 1912 M. Eno, France.
D. 1920 Stapr et al., USA.
3. Which answer to Aueski's prompt is correct?
A. DNA storage, family Herperviridae.
B. RNA-protecting, neurotropic, epitheliotropic.
S. DNA-preserving, myotropic, parenchymatropic.
D. RNA-sparing, neurotropic, somatropic, heterotropic.
4. In which answer is the chronology according to the degree of tendency to Aueski correct?
A. Cattle, deer, sheep, pig, horse, cat, dog, fox....
B. One-hooved animals, two-hooved animals, carnivorous animals.
S. All pets and wild animals.
D. All animals living on land are prone, birds are prone.
What is the source of the disease in Au e ski?
A. Sick animals, virus carriers.
B. Often latently infected animals.
S. Infected and immunizing subinfection cases.
D. Relapsed patients, newly brought sick animals.
6. In what season is Aueski's disease more common?
A. In the spring, in the fall.
B. In autumn, winter.
S. In the summer, in the spring.
D. In winter, in spring.
7. Which animals do not experience severe itching in Aueski's disease?
A. In pigs.
B. In dogs, cats.
S. In sheep and goats.
D. Cattle, horses.
8. In Aueski in 6-8 days illness in pigs how many % organize enough?
A. 100%.

B. 80%.
S. 60%.
D. 40-60%.
9. Aueski in pigs most of the time how clinical syndromes with will it pass?
A. Epilepsy, pathological view
B. Polyneurosis, ataxia.
S. Asthesia, atony, asthenia.
D. Neurosis, neuritis, epilepsy.
10. Clinical symptoms characteristic of Aueski are given correctly in which answer?
A. Strong itching, scratching, biting itching
B. Epilepsy, angina, bradycardia.
S. Apnea, dyspnea, neurosis, hydrophobia.
D. Tachycardia, strong itching, discomfort.
11. In Aueski special healer biological preparations is there
A. Yes, hyperimmune blood serum, special gammaglobulin.
B. Yes, convalescent blood, novarsenol.
Q. Yes, sulfonamides, nitrofurans.
D. Yes, bacteriocide, herbicide, phytoncide.
12. Aueski in illness how event used?
A. Quarantine announcement will be done.
BC h eclov announcement will be done.
S. The household is healthy that announcement will be done.
D. " Stamping out " method is used.
13. Who and when invented the causative agent of necrobacteriosis?
ARKox (1881), F. Leffler (1884).
B. F. Leffler, A. Froggy (1884).
S. F.ssion, IP Pavlov (1901).
D. IM Sechenev, D. Aginer (1881).
14. Necrobacteriosis disease of the trigger to oxygen relatively optimum conditions tell me
A. Bond anaerobic.
B. Obligation aerobic.
S. Optional anaerobic.
D. _ Optional aerobic.
15. Necrobacteriosis of the trigger which serotypes identified?
A. A, AB, B, Serotypes.
B. from 10 more than serotypes.
Q. 2 serotype.
D. _ 8 serotype.
16. Necrobacteriosis of triggers pathogen serotypes tell me
A.A andAB serotype.
B. All serotypes pathogen.
S. All strains pat ogen.
D. All strains lentogenic.

17. Necrobacteriosis triggers how exo, endotoxins separates?
A. Leukocidin, necrotoxin, hemolysin, cytotoxin.
B. Neurotoxin, myotoxin, epitheliotoxin.
S. Fibrotoxin, leukotoxin, phagotoxin.
D. _ Polytoxin, monotoxin, plasmotoxin.
18. Necrobacteriosis which to the group belong to?
A. Zooanthroponosis.
B. Kavshovchi to animals.
S. Bir to ungulates.
D. Pair to ungulates.
19. Necrobacteriosis trigger sources?
A. Sick animals, bacteria carriers.
B. Damaged fodder, water.
S. Alimentary, aerogenous factors.
D. Vertical, horizontal factors.
20. In what way is the causative agent of necrobacteriosis separated from a sick animal?
A. All excreta with, injury tissues with.
B. Saliva, feces, urine with.
S. Aerogen, genital, contact the way with.
D. Hemolytic, lympholytic, alimentary.
21. Necrobacteriosis young in animals skin and mucus on curtains how pathology with night?
A. Calves diphtheria, swine in children rhinitis, stomatitis, dermatitis.
B. Abscess, phlegmon, pleurisy, pneumonia.
S. Gangrene, abscess, dermatitis.
D. _ Gastritis, gastroenteritis, pododermatitis.
22. In necrobacteriosis special prevention is there
A. Boron, Nekovac, polyvalent, associated.
B. No, bacteriocide preparations used..
Q. No, anti septic preparations is used.
D. Not developed.
23. Necrobacteriosis to people is it contagious?
A. It is contagious.
B. It is not contagious.
S. It can only be transmitted through trauma.
D. Science sure information no.
24. What group does the causative agent of contagious pleuropneumonia of cattle belong to?
A. Mycoplasmas.
B. To rickettsiae.
S. To bacteria.
D. To viruses .
25. Mycoplasmas how pathogen descendants have

A. Mycoplasmas (type 76), ureoplasma (type 2).
B. 40 ha near types there is
From S. 20 more than types there is
D. Pathogen and a pathogen types there is
26. Mycoplasmas special features which in the answer right given?
A. Polymorphism, strict curtain no.
B. Coccoid, oval.
S. Rod-shaped, spiral.
D. With spores, without spores.
27. Cattle KPP – the causative agent of the disease grows in what nutrient environments?
A. In a natural nutrient medium with 10-20% horse serum and 10% yeast extract.
B. In all liquid, semi-liquid nutrient media.
S. In all solid environments.
D. Egg yolk, in food media with added glucose.
28. Which animals cattle KPP to the disease inclined?
A. Cattle, zebu-like cattle, buffalo, bison, yak.
B. All couple ungulates.
S. Experimental if infected all animals inclined
D. Bu of the instigator pathogenicity depends.
29. Small laboratory animals if the experimental y is flown get sick
A. No.
B. Yes.
S. Sometimes gets sick
D. Injury to the way depends.
30. Cattle KPP - in the disease to die what %?
A. 10-90%.
B. 10-20%.
S. 20-30%.
D. 30-40%.
31. Cattle KPP is a disease triggers how toxins separates?
A. Exo, endotoxins - in the composition lipo polysaccharide and galactan there is.
B. Exotoxin, universal toxin.
S. Endotoxin, hemolytic, proteolytic.
D. Neurotoxin, pneumotoxin, myotoxin.
32. Cattle CPP - how long does the disease last on average?
A. 40-45 days.
B. 10-12 days.
S. 15-20 days.
D. 20-25 days .
33. Foods KPP - disease special pathanatomical changes?
A. Injured in the lungs (up to 20 l.). fibrinous – reddish – yellowish exudate t o' plans.
B. Exudation, proliferation, hepatization.
S. Atelectasis, emphysema, pneumonia.

D. Pneumonia, pleurisy, spike.
34. Cattle KPP - how is the disease diagnosed?
A. Serological, bacteriological, clinical, pathanatomical, epizootological check with.
B.AR, KBR, GATR with.
S. Allergic and bioassay with.
D. With bacteriological examination.
35. Cattle KPP of XEB - in which case is it considered to have completely disappeared?
A. From the destruction of the last diseased animal in the furnace after one in another disease if it doesn't come out.
B. Last ill an animal no from being done then at 6 months disease if it doesn't come out.
S. Allergic 100% negative for the 2nd time in the tests the result if taken
D. The last sick animal no from being done then 2 years inside disease if it doesn't come out.
36. Goats KPP - in the disease scientist what %?
A. 70-100%.
B. 10-20%.
S. 30-40%.
D. 50-60%.
37. Goats KPP is a disease of the trigger morphological shape how?
A. Coxsimon, rod, thread.
B. S h arciform, prismatic, spiral.
S. Polymorph, all in forms.
D. This strain type depend _
38. In the flock goats KPP - in the disease illness level what %?
A. 100%.
B. 80%.
S. 60%.
D. 50%.
39. Goats with KPP - disease clinical others _ _ appear from being after when scientist will be
A. From 7-10 days after.
B. One moon inside.
S. 15-20 days after.
D. in 2-3 months.
40. In which case, the diagnosis of KPP-disease in goats is correct is it?
A. Culture if isolated, sick from a goat received in patmaterial instigator if found.
B. Serodiagnosis mu s bat the result if he gives
S. Allergic diagnosis positive the result if he gives
D. Disease public tu0Clsa.
41. Differentiate the KPP disease of goats from similar diseases must
A. Pasterell o z, agalactia.
B. Salmanell o z, diplococcosis.

S. Colibacteriosis, pneumonia.
D. Compilobacteriosis, smallpox.
42. Goats KPP in illness laboratory inspection term how much
A. 60-90 days.
B. 10-20 days.
S. Bir to the moon.
D. 30-40 days.
43. Doors KPP disease in prevention how vaccine used?
A. Hydro Okisammonian liquid-formal-vaccine, immunity 1 year continue is enough.
V. Live-dry vaccine, immunity 6 months continue is enough.
S. Water yu q-emulsion vaccine, immunity 10 months continue is enough.
D. Associate polyvalent vaccine immunity 1 year continue is enough.
44. Goats KPP in illness in the oven how event used?
A. Quarantine announcement will be done.
B. C h eklov announcement will be done.
S. Unhealthy that announcement will be done.
D. " Stamping out " method is used.
45. When will the quarantine be lifted in goats with KPP disease and how long will it be under control?
A. Two months after the last patient has been eliminated, there will be another 1 year of follow-up.
B. Last ill no from q i lingan then 21 days after that, another 6 months in control will be
Q. Last ill no from q i lingan then 14 days after that, another 1 year in control will be
D. Two months after the last patient was eliminated, another 6 months will be observed.
46. Cattle plague in the CIS countries since when since does not meet?
A. Since 1929.
B. Since 1970.
S. Since 1950.
D. since 1980.
47. The virus that causes rinderpest has antigenic and immunological similarities with which viruses?
A. Dogs the plague, people smallpox disease.
B. C h intestine, protein.
S. Kutyrish, Aueski.
D. Vesicular stomatitis, viral diarrhea.
48. Cattle plague to the trigger special cultural feature tell me
A. Chicken in the embryo to ssPT have
B. Cultural in tissue necrosis calls
S. Epithelio trop, neuro trope.
D. Bezli tissues lysis does.
49. Cattle plague stationary in their furnaces colostral immunity how much time continue enough?

A. 8-11 months.
B. 2-3 months.
S. 4-5 months.
D. 5-7 months.
50. Cattle in the plague primary in the oven scientist what %?
A. 90-100%.
B. 70-80%.
S. 60-70%.
D. 50-60%.
51. Cattle plague with sick an animal what will be done?
A. Sick animals without blood method with killed, skinned with will be burned.
B. Special to the graves will be buried.
S. is thrown into the wells of Becker.
D. It is buried at least 2m deep and disinfected with 20% freshly slaked lime.
52. Of cattle bad good quality catarrhal fever how disease?
A. Sporadic transient, acontagious, virosis disease.
B. Epizootic, contagious, viral disease.
S. Exotic transient, contagious, bacteriosis.
D. Panzootic transit, transcontagious, viriosis.
53. Yo mon good quality catarrhal fever more which age in animals occurs?
A. 1-4 years old cattle and in buffaloes occurs.
B. Up to 1 year all couple in ungulates occurs.
S. In 2-4 month old calves, lambs, goats.
D. All age forager in animals.
54. Yo mon good quality catarrhal fever against special prevention work is it done?
A. No, the vaccine work not released.
B. Yes, woven hydroxy-al y uminyli vaccine.
S. Associate vaccine.
D. Weakened alive vaccine.
55. Sick bad good quality catarrhal feverish an animal meats how neutralized?
A. 1.5-2 kg divided into pieces 2.5-3 atm. Boiling at 115-117 ºC for 1.5-2 hours is allowed for consumption inside the farm.
B. Without a rock that is found.
S. Boil, meat choir to animals is given.
D. Bone it, meat him is prepared.
56. In cattle infectious nodular dermatitis inciting virus which one
A. Neethling-typevirus.
B. Allerton-typevirus.
C. Orpheling-typevirus.
D. All answers right.
57. The causative agent of nodular dermatitis is morpho-physiologically similar to which virus?
A. C h intestine to the virus.
B. Rabies to the virus.

S. White to the virus.
State to the virus.
58. ssPT of the causative agent of nodular dermatitis – in a 5-7-day-old chicken embryo when observed?
A. In 5-14 days.
B. In 2-4 days.
S. In 4-6 days.
D. In 6-8 days.
59. Nodular to dermatitis the most sensitive animal?
A. Lactation period a cow and buffalo
B. Service period cattle
S. Strait cows.
D. Mainly calves of 6-8 months.
60. Special prevention in nodular dermatitis and special treatment tell me
A. Special prevention, special treatment work not released.
B. Antibiotics, sulfamides.
S. Vitamin, hormonal therapy.
D. Novarsenol, osarsol, among others preparations.
61. Of sheep with chlamydia abortion (constipation) disease which to the group belong to?
A. To zoonoses.
B. Zooanthroponoses.
S. Anthropozoonoses.
D. Ubiquit to infections.
62. Chlamydia where _ _ lives?
A. Bond, cells inside.
B. In the blood.
S. In the muscles.
D. Epithelium in the tissues.
63. Chlamydia development cycle how much time?
A. 40-48 hours.
B. 48-72 hours.
S. 20-24 hours.
D. 1 week.
64. In Chlamydia how antigen have
A. Thermostable.
B. Thermolabile.
S. Glyco polypeptide
D. Polypeptide, lipoprotein.
65. Which laboratory animals are susceptible to sheep chlamydia abortion?
A. White mouse, rat, sea pig, rabbit.
B. Cat a child, a dog a child, a rabbit.
S. Mouse rat, chicken chicks.
D. Lamb, goat, calf, rabbit.

66. Abortion what % - chlamydia (in sheep)?
A. Up to 60%.
B. Up to 70%.
S. Up to 30%.
D. Up to 20%.
67. When is the diagnosis of sheep chlamydiosis considered valid?
A. If a special body is found in the uterus, vaginal fluid, if it has a positive reaction to KBR, RNGA, if a pathogen is found in a chicken embryo.
B. Pathanatomical changes, abortion.
S. Epizootological, clinical analyses.
D. RA, KBR, RNGA to positive the result if he gives
68. Vaccine work released?
A. Yes, emulsin vaccine Russia VIEV.
V. No.
S. Yes, Czechia , emulsin vaccine.
D. Polyvalent water and white GOV – France.
69. Sheep and goats plague when , where _ _ studied?
A. In West Africa, 1942.
B. In Australia, 1910.
In S. Hungary, 1900.
D. in France, 1940.
70. In what kind of environment does the virus that causes plague of sheep and goats grow (pathogenic effect)?
A. Vegetation in lamb, rabbit organism, chicken embryo, lamb, calf kidney culture, ssPTgaega.
B. All natural food environments.
S. In selective nutrient environments.
D. In nutrient medium supplemented with blood serum and glucose.
71. Pestilence with sick sheep and goats what will be done?
A. Sick mandatory killed, burned.
B. Patients treated, healthy is vaccinated.
S. All to the sick hyperimmune blood serum is added.
D. Mandatory meat for will be slaughtered.
72. Sheep and goats to the plague against cultural virus vaccine with immunity when vaccinated (France). how much time continue enough?
A. 3 years.
B. 1 year.
S. 2 years.
D. 10 months.
73. Pestilence the disease in determining the most reliable sero diagnosis?
A. IFR, PCR.
B. AR, KBR, GATR.
S. RDP, GAR.
D. All sero tests reliable.

74. In which answer is the causative agent of manka disease correctly written?
A. Non-motile, spore, capsule harvest does not Bacteria.
B. Mycoplasma.
S. Spore, capsule harvest does, clostridia.
D. Rickettsia.
75. Which organ is most often injured in Manka's disease?
A. Lungs, nose (cavity...).
B. Stomach, intestines.
S. Skin, mucous curtains.
D. Nerve tissues, muscles.
76. Manga conditional respectively which in forms night?
A. Nose form, skin, lung, latent.
B. Nose, stomach, intestines shape
S. Skin, muscle shape
D. Head, feet, skin shape
77. Mangada in practice which diagnosis a lot used?
A. Allergic diagnosis.
B. Sero diagnosis.
S. Clinical-syndromatic diagnosis.
D. Laboratory diagnosis.
78. Malleinization how many kind of held?
A. In autumn, leather under, skin between
B. In autumn, muscle between
S. Theriosti, tail skin between
D. Only ophthalmo test
79. Manga the disease which from diseases differentiate should
A. Mit, epizootic lymphangitis.
B. Strigu sh ylishay, dermatitis.
S. Streptococcus, myeliosis.
D. Skin diseases.
80. Sap with ill an animal what will be done?
A. Mandatory kill and skin with will be burned.
B. Only pedigree animals is treated.
S. Scientific studies for-strain to get is sent.
D. Hyperimmune blood serum with treatment possible
81. Sap in illness quarantine void to be done which in the answer right given?
A. 45 days after final disinfection after destruction of the last sick animal after.
B. Last ill an animal no from being done after 6 months after.
Q. Last ill an animal no will be done, 21 days after
D. Last ill an animal no after 1 year after
82. Horses flu disease trigger strain m , serotypes in them to what depends?
A. Ickib-antigen, d-antigen and in neuroamin and to enzymes depends.
B. M, D, A to antigens.
S. Shell and internal antigens.

D. Properdin, globulin, interferons.
83. Horses in influenza, INAN diseases ECHT-acceleration reason what?
A. Erythropenia, globulinemia.
B. Erythrocytosis, anemia.
S. Anisatocytosis, poikilocytosis.
D. Leukocytosis, phagocytosis, leukopenia.
84. Viral pathogens in identification which of the methods used?
A. Viral my diagnosis with check
B. Bacterial from infections differentiate
S. Mycotic from infections differentiate
D. Prionli, haptenli from infections differentiate
85. C h furnaces plague the first times when , where _ _ studied?
A. 1833 USA at Ohio in the state.
B. 1890 in France.
S. 1901 Russia, Kaluga in the region.
D. 1603 China, Manguria in the region.
86. C h furnaces in the plague scientist what %?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
87. C h furnaces in the plague virus portability how much time continue enough?
A. 3-10 months.
B. 2-5 months.
S. 4-8 months.
D. 1 year.
88. C h furnaces in the plague virus antigen in determining which reaction put?
A. IFR, NR.
B. AR, KBR, GAR.
S. GATR, KUBR, RA.
D. All serological reactions.
89. Quarantine during swine fever void from being done how much from time after pig take exit can
A. From 12 months after.
B. Two from the moon after.
S. From 4 months after.
D. From 8 months after.
90. C h furnaces Africa plague of the trigger pathogenicity degree tell me
A. diluted 1:109 ill 1 ml of pork blood disease provokes
B. No, 4 ml disease provokes
S. 3-5 ml disease provokes
D. diluted 1:10 1 ml of blood disease provokes
91. C h furnaces Africa plague in illness which vaccine used?
A. Bloodless method killed and burned.

B. Becker to the well thrown away.
S. 2 m deep digging if buried will be
D. Only 20% diluted lime with disinfection to do a must
92. C h goat flu disease the first times when, which countries pandemic as note done?
A. 1918 USA.
B. 1860 France.
S. 1882 England.
D. 1920 Russia.
93. The virus that causes swine flu is similar to which viruses in terms of antigenic properties?
A. People flu the disease instigator to the virus.
B. Horses, pigs, dogs, poultry flu triggers.
S. Dog, pig, poultry flu triggers.
D. All animals flu triggers.
94. C h goat in the flu characteristic pathanatomical changes which in the answer right defined?
A. Lungs swollen nose cavity, larynx, bronchi blood mixed slimy mass is collected.
B. In veins sclerosis, thrombus, hemorrhage observed.
S. Gastritis, enteritis, pneumonia.
D. Kon' y unctivitis, keratitis, vulvitis.
95. C h goat the flu which from diseases differentiate should
A. Olat, pasterell o z, Salmanell o z, enzo o tik pneumonia.
B. Au e ski, rhinitis, transmissive gastritis.
S. Saramas, plague, pasteurellosis , colibacteriosis.
D. White, plague, Africa plague, rhinitis.
96. C h goat flu in illness how treatment is used.
A. Symptomatic treatment.
B. Special treatment.
S. Gamma globulins.
D. Hyperimmune blood serum.
97. C h goat from the flu recovered an animal blood in serum how antibodies have
A. Virus neutralizing, anti Hemagglutin.
B. Complement binding, precipitating.
S. Agglutination inhibitor.
D. Inhibitor of phagocytosis.
98. C h of goats viral gastroenteritis disease first there is when which in the country note done?
A. In the USA, 1946.
B. in Russia, 1920.
In S. China, 1900.
D. in England, 1910.
99. C h furnaces viral to gastroenteritis special clinical characters which in the answer right?

A. Catarrhal, hemorrhagic gastroenteritis, vomiting, diarrhea, organism dehydration, death a large %.
B. Bloody drink withdrawal, pneumonia, strong itching
S. Motion balance break down, cough.
D. Asthesia, asthenia, ataxia, adhesion.
100. The virus that causes swine viral gastroenteritis is antigenically similar to which viruses?
A. Hemagglutinating to coronaviruses.
B. Antiviral to poxviruses.
S. To rhabdoviruses that inhibit agglutination.
D. To rhabdo, corona, pox viruses.
101 . Tell the sources of the causative agent of swine viral gastroenteritis?
A. Sick and recovered pigs.
B. Products from sick pigs.
S. Rodents, blood-sucking insects.
D. Infected fodder, piglets.
102. How long does immunity last in a pig that has recovered from swine viral gastroenteritis?
A. Two years.
B. One year.
S. Up to three years.
D. Four years.
103. Name the clinical signs of infectious dysentery of piglets.
A. Weight loss, bloody-mucous diarrhea.
B. Pneumonia, fever, diarrhea.
S. Ataxia, asthenia, hydrophobia.
D. Rhinitis, pneumonia, cough, diarrhea.
104. When was the disease of piglet dysentery registered for the first time, in which country?
A. USA, 1921.
B. Canada, 1910.
S. Russia, 1901.
D. France, 1903.
105. What is the correct answer for the causative agent of piglet dysentery?
A. There is. hyodysentery, gram-negative, anaerobic, spirochete.
B. There is. hyodysentry, gram-positive, aerobic, mycoplasma.
S. There is. treponema, gram-positive, aerobic, rickettsia.
D. There is. treponema, gram negative, aerobic, bacteria.
106. Piglet dysentery is most common in pigs of which age?
A. 2-6 months old.
B. 6-12 months old.
S. 1-4 months old.
D. 1-2 months old.
107. The treatment of piglet dysentery is given correctly in which answer?

A. Soda-osarsol solution, thilan, pharmazine, trichopo.
B. Antibiotics, sulfonamides.
S. Bacteriophages, gammaglobulin.
D. Convalescent blood, hyperimmune blood serum.
108. Which clinical signs of infectious bronchitis in chickens?
A. Pathology of respiratory organs, pathology of reproductive organs.
B. Gastro-enteritis, intestinal pathology.
S. Pathology of head organs.
D. Conjunctivitis, keratoconjunctivitis.
109. When and where was infectious bronchitis registered for the first time?
A. In the USA, 1931.
B. in Italy, 1902.
In S. Germany, 1882.
D. in Russia, 1910.
110. What is the causative agent of infectious bronchitis, and how many serotypes have been identified?
A. RNA storage coronavirus, more than 20 serotypes.
B. DNA storage poxvirus, more than 20 serotypes.
S. RNA-storing herpesvirus, more than 10 serotypes.
D. DNA storage coronavirus, more than 10 serotypes.
111. Which animals are prone to experimental infection with the causative agent of infectious bronchitis?
A. Bat, hyena, rabbit babies.
B. Mouse, rat, kitten.
S. Guinea pig, mouse, rat.
D. Rabbit, mouse, rat.
112. Which serological reactions are used in the diagnosis of infectious bronchitis?
A. RN, RNGA, RDP, RIF, IFA, PZR.
B. RA, RP, KBR, RP.
S. RA, KBR, RP, PZR, IFA.
D. RA, RP, KBR are sufficient.
113. How is infectious bronchitis treated?
A. No treatment has been developed.
B. It is treated symptomatically.
S. Antibiotic powder is dusted by aerosol method.
D. 40% formaldehyde vapor is used.
114. What is the causative agent of poultry leukemia?
A. RNA-storing oncovirus, 5 serogroups, A,B,C,D,E.
B. DNA-storing coronavirus, there are 3 serogroups.
S. RNA-storing poxvirus, there are 5 serogroups.
D. DNA-storing oncovirus, there are 4 serogroups.
115. Poultry of which age are more susceptible to leukemia?
A. Poultry 4 months and older.
B. Up to 2 months.

S. Up to 1 year.
D. Up to 2 years.
116. Poultry leukosis should be distinguished from which diseases?
A. Tuberculosis, Marek, coligranulomatosis.
B. Salmonellosis, ILT, colibacteriosis.
S. Escherichia, lymphomatosis, salmonellosis.
D. Tuberculosis, brucellosis, Newcastle.
117. How is avian leukemia treated?
A. A treatment method has not been developed.
B. It is destroyed by burning.
S. Compulsory submission to meat.
D. " Stamping out " method is used.
118. Which organs are more inflamed in bird flu?
A. Respiratory, digestive organs.
B. All hemopoietic organs.
S. Nerve system, basically centers.
D. Subtraction and reproductive organs.
119. Poultry flu in the literature how called?
A. Exudative, y European plague, classic plague.
B. Croupous pneumonia, black death.
S. Hemophilic plague, necrotic pneumonia.
D. Liver necrosis, kidney softening.
120. Who are the Russian scientists who studied bird flu perfectly?
A. M. Tartakovsky, VN Syurin, NG Ozidze.
BIP Pavlov, IM Sechenev, RV Petrov.
S. Blondlo, Gols, R. Virchow.
DKI Skryabin, NV Badanin, II Arkhangelsky.
121. What is the causative agent of bird flu?
A. Orthomyxoviride, DNA storage, InfluenzavirusA.
B. Poxoviride, DNA protector, neurotrope.
S. Coronoviride, RNA protector, pantotrope.
D. Herpesviride, DNA-preserving, epitheliotropic.
122. Human influenza virus A_2- in domestic and wild birds, or vice versa, is the avian influenza virus in humans?
A. It has been experimentally proven.
B. Sometimes 3-5% occurs.
S. No, not studied.
D. It depends on many factors.
123. What is the morbidity and mortality rate of bird flu?
A. Morbidity is 80-100%, death is 10-90%.
B. Morbidity 40-50%, death 10-20%.
S. Morbidity 50-60%, death 20-30%.
D. Morbidity 60-70%, death 40-50%.
124. In which case is the diagnosis of bird flu considered correct?

A. Diagnosis based on the results of GAR, NR, GATR, IDR, IFR.
B. Based on epizootological, clinical, pathanatomical results.
S. Infected embryos - the diagnosis.
D. All answers are correct.
125. Bird flu should be distinguished from what diseases?
A. Newcastle, infectious bronchitis, infectious laryngotrachitis, pasteurellosis, mycoplasmosis.
B. Salmonellosis, colibacteriosis, eimeria.
S. Pasteurellosis, anthrax, bradzot.
D. Marek, protein, smallpox, bronchitis.
126. In which answer is the causative agent of fowl pox correctly written?
A. DNA storage, aviapoxviride, chicken, hyena, canoreika species.
B. RNA storage, coronavirus, chicken, duck, turkey.
S. DNA storage, rhabdovirus, goose, chicken, duck.
D. RNA storage, herpesvirus, turkey, chicken, hyena.
127. Where are colostral antibodies in smallpox?
A. In newly hatched chicks.
B. In chicks 10-30 days old.
S. in chicks 20-21 days old.
D. In a stationary oven - in chicks up to 1 month old.
128. Which clinical-syndromatic type of smallpox is most common in poultry?
A. Diphtheritic smallpox.
B. Combined (slivnoy) smallpox.
S. Hemmorrhagic smallpox.
D. Fibrinous pox.
129. What changes are observed in the smear prepared in smallpox?
A. Guarnielli, Bollinger inputs (set of viruses).
B. Babesh, Negri, abortive, atypical.
S. Hemophilic, lipolytic, neurolytic.
D. Phagolytic, cytopathogenic.
130. Poultry pox should be distinguished from which diseases?
A. Infectious laryngotracheitis, infectious bronchitis, candidomycosis, aspergillosis, avitaminosis A.
B. Pasteurellosis, Salmonellosis, colibacteriosis.
S. ILT, IB, escherichia, eimeria.
D. Plague, flu, marek, newcastle.
131. What antibodies appear when vaccinated against smallpox?
A. Virus neutralizing, complement binding, precipitating antibodies.
B. Cell and tissue immunity.
S. Humoral and macrophage immunity.
D. Specific and non-specific immunity.
How long does immunity last when vaccinated with the N-27 strain vaccine of VITI in chicken pox ?A_3
A. 4 months for chicks, 9-10 months for chickens.

B. Chicks 6 months, chickens 1 year.
S. 1 month in chicks, 2 years in chickens.
D. 5 months in chicks, 1 year in chickens.
133. The rule and procedure for canceling quarantine in case of poultry pox?
A. 2 months after the last patient has disappeared, the final disinfection is carried out... it is canceled.
B. 4 months after the end of the illness.
S. 6 months after the end of the illness.
D. 45 days after the end of the illness.
134. When and in which country was infectious laryngotracheitis first studied?
A. 1925 in the USA.
B. 1900 in Spain.
S. 1880 in Italy.
D. 1910 in England.
135. Who and when experimentally studied infectious laryngotracheitis in Uzbekistan?
A. 1970 BQ Kochkarov.
B. 1960 AO Oripov.
S. 1954 Sh.A. Azimov.
D. 1952 AK Sitdikov.
136. In which answer, the causative agent of infectious laryngotracheitis is correctly indicated?
A. DNA storage, herpesvirus.
B. DNA storage, coronavirus.
S. RNA storage, poxvirus.
D. RNA storage, epithelitropvirus.
137. How long does virus transmission last in birds that have recovered from infectious laryngotracheitis?
A. Up to 2 years.
B. Up to 1 year.
S. Lasts for life.
D. 3-4 years.
138. The trophic classification of the causative agent of infectious laryngotracheitis is given correctly in which answer?
A. Epileutrop.
B. Myotropic.
S. Neurotropic.
D. Sarcotrope.
139. In what answer is the classification according to the clinical-syndromal course of infectious laryngotracheitis correct?
A. Laryngotracheal, conjunctival form.
B. Pulmonary, enteral.
S. Parenchymal, gastroenteral.
D. Urogenital, pulmonary, hepatopathological.

140. How long does immunity last when vaccinated against infectious laryngotracheitis? Where is the vaccine sent?
A. Applied to the cloacal mucosa, immunity until the end of life, lifelong.
B. Under the skin, on the neck, lasts for 1 year.
S. Between the muscles, to the chest, lasts 2 years.
D. Skin feathers are plucked, scratched and vaccinated, lasting 4 years.
141. The procedure for canceling restrictions in case of infectious laryngotracheitis?
A. 2 months after the last patient has disappeared, the final disinfection is carried out and the restriction is taken.
B. 6 months after the termination of the disease.
S. 1 year after the termination of the disease.
D. 2 years after the termination of the disease.
142. When and in which country was infectious bronchitis studied in poultry?
A. In the USA, 1931.
B. in Germany, 1900.
In S. England, 1910.
D. in Italy, 1940.
143. In which answer is the pathogen of infectious bronchitis correctly written?
A. RNA storage, corona virus avium.
B. RNA storage, herpesviride.
S. DNA storage, poxviride.
D. DNA storage, coronavirus avia.
144. The age of chickens most susceptible to infectious bronchitis? What is the death rate?
A. Age up to 30 days, mortality 40-60%.
B. Age up to 60 days, mortality 80-100%.
S. Age up to 90 days, mortality 60-80%.
D. Age up to 4 months, mortality 70-80%.
145. What happens when chicken embryos are infected with infectious bronchitis?
A. Embryo development is inhibited, deafness (pacana) is observed.
B. Embryo development is accelerated, hyperkinesis is observed.
S. Embryo undergoes necrosis, dies.
D. Depends on how many doses are given.
146. How long do chickens that have recovered from infectious bronchitis remain carriers of the virus?
A. 49-105 days.
B. Up to 6 months.
S. Up to 1 year.
D. Up to 2 years.
147. How much egg production is reduced in case of infectious bronchitis?
A. 50% or more is reduced.
B. Decreases by 60% or more.
S. is reduced by 70% and more.
D. Decreases by 80% or more.

148. When chicks are infected with infectious bronchitis viruses, how long do clinical symptoms appear?
A. In 18-24 hours.
B. In 2-3 days.
S. Depends on the method of injury.
D. Depends on the dose of viruses.
149. How long is the incubation period when infected with infectious bronchitis under natural conditions?
A. 36 hours to 10 days.
B. 18-24 hours.
S. 2-3 days.
D. Up to 1 month.
150. What is the death rate when chickens with infectious bronchitis have symptoms of ephrit and nephrosis?
A. 57-70%.
B. 40-50%.
S. 70-80%.
D. 20-30%.
151. In chicken embryos infected with infectious bronchitis viruses, "smallness", "deafness" is observed on how many days?
A. in 6-9 days.
B. in 13-15 days.
S. in 4-5 days.
D. in 19-20 days.
152. In infectious bronchitis, the virus isolated from the paternal material is identified by which reactions?
A. NR, BGAR, IDR, IFR.
B. RP, IDR, KBR, PZR.
S. AR, PR, KBR, DPR.
D. Any serological reaction can be applied.
153. When do virus-neutralizing antibodies appear in infectious bronchitis and how long do they last?
A. It appears on the 11th day of the disease, it is stored for 483 days.
Appears on the 4th day of B. and is stored for 1 year.
Appears on the 6th day of S. and is stored for 10 months.
D. Appears on the 3rd day and is stored for 2 years.
154. What diseases should be distinguished from infectious bronchitis?
A. ILT, Smallpox, Newcastle, respiratory mycoplasmosis.
B. Salmonellosis, escherichia, marek, bursitis.
S. Of all prion diseases.
D. From all viral infectious diseases.
155. Does a bird that has recovered from infectious bronchitis get infected with this disease a second time?
A. No.

B. Yes.
Q. It depends on many factors.
D. Depends on the virulence of the pathogen.
156. The rule to cancel the restriction in infectious bronchitis?
A. Final disinfection 3 months after the last patient has been eliminated.
B. 6 months after the last patient was eliminated.
S. 1 year after the last disease was eliminated.
D. 45 days after the last patient has been eliminated.
157. In what country was infectious bursitis studied for the first time?
A. 1957 in the USA.
B. 1920 in Italy.
S. 1910 in England.
D. 1930 in Hungary.
158. 2004 from infectious bursitis. What % of existing birds in Russia died?
A. 14%.
B. 20%.
S. 30%.
D. 42%.
159. Which definition given to the cause of infectious bursitis is correct?
A. RNA storage, one type.
B. DNA storage, herpesviride.
S. RNA storage, coronaviride.
D. DNA storage, poxviride.
160. Find the range of poultry most susceptible to infectious bursitis?
A. Broiler, 2-3 weeks to 11 weeks.
B. Loman-Brown up to 1 month.
S. Ru0Cq chickens, 1-2 weeks old.
D. All domestic chicken breeds, up to 1 year old.
161. Name the reservoir of viruses in infectious bursitis?
A. Black beetle, fly, rodents, mealworms, wild birds.
B. Fish, frogs, worms.
S. Flies, mosquitoes, blood-sucking insects.
D. All arthropods that live in water.
162. Infectious bursitis - Gamboro - which diseases should be distinguished?
A. IB, Newcastle, coccidiosis, avitaminosis E, K, toxic dystrophy.
B. Escherichia, Salmonellosis, ILT.
S. Bird flu, YUPPG, eimeria.
D. From all viral infectious diseases.
163. According to statistics, how much does the United States, England, and Italy suffer from avian leukemia every year?
A. USA $150 million, England $40 million, Italy $20 million.
B. $200-250 million.
S. $250-300 million.
D. $400-500 million.

164. 1970 How much has Russia suffered from avian leukemia?
A. 3 times the damage of all other diseases.
B. Equal to the damage of all other diseases.
S. Half the damage of remaining diseases.
D. 10 times more damage than other diseases.
165. In which answer is the causative agent of avian leukemia correctly written?
A. RNA storage, oncornavirus.
B. DNA storage, poxvirus.
S. RNA storage, herpesvirus.
D. DNA storage, coronavirus.
166. How many percent of poultry leukemia is lymphoid leukemia?
A. 80%.
B. 60%.
Q. 40%.
D. 30%.
167. How is avian leukemia treated?
A. Not treated, given to meat.
B. There is symptomatic treatment.
S. Gammaglobulin, convalescent blood.
D. Antibiotics, sulfonamides.
168. If leukemia is detected in a poultry farm, how many percent of poultry must undergo serological examination?
A. 5%.
B. 10%.
S. 20%.
D. 30%.
169. What is the correct answer for the causative agent of Marek's disease?
A. DNA storage, herpesvirus.
B. RNA storage, poxvirus.
S. DNA storage, oncocoronavirus.
D. RNA storage, coronavirus.
170. Classification of Marek's disease according to clinical manifestations?
A. Neural, ocular, visceral.
B. Myopathic, epiopathic, neuropathic.
S. Orthopedic, cranial, caudal.
D. Hormal, desmochorial, mesenchymal.
171. Tell the principle of exploitation of poultry houses in Marek's disease?
A. "Bariband", "baribush".
B. Filarbeiten vinikelessen.
S. " Stamping out ".
D. Well-well, out-out.
172. If all infectious diseases are taken as 100%, how many % corresponds to Salmonellosis?
A. 26-40%.

B. 10-14%.
S. 18-20%.
D. 20-24%.
173. What is the percentage of chicken Salmonellosis?
A. 80-90%.
B. 70-80%.
S. 60-70%.
D. 50-60%.
174. Before 1970, what was the name of Salmonellosis in poultry?
A. Typhoid, penniless.
B. Escherichia coli, colibacteriosis.
S. Granulamatous, mucosa.
D. Bacteriosis, stachyobotriosis.
175. Who isolated Salmonella for the first time?
A. US scientists Smith, Salmon, 1885 from pig.
B. American scientist Retger, 1900.
S. Endo, Ploskirov, Italy, 1890 from a bird.
D. Gapten, Prion - 1910, Russia.
176. How is avian salmonellosis transmitted?
A. Alimentary, aerogenous, contact.
B. Vertical, horizontal, genital.
S. Sporadic, epizootic, exotic.
D. Mainly aerogenic.
177. What is the percentage of salmonella death in ducklings?
A. up to 90%.
B. Up to 80%.
S. Up to 70%.
D. Up to 60%.
178. What other serological tests can be performed in addition to the Hemagglutination reaction with a drop of blood when diagnosing salmonellosis?
A. Coagglutination, IFA, PCR.
B. KBGAR, GAR, KTPR.
S. PR, AR, KBR, RDP.
D. IFA, PR, AR, KBR.
179. Poultry salmonellosis should be distinguished from what diseases?
A. Colibacteriosis, aspergillosis, Newcastle disease, pasteurellosis.
B. Escherichia, eimeria, marek, smallpox.
S. Psittacosis, eimeria, plague.
D. Influenza, infectious bursitis, Newcastle.
180. Explain the scientific basis of treatment against salmonellosis.
A. It is determined after the sensitivity to therapeutic agents is determined.
B. Antibiotics, sulfonamides.
S. Bacteriocidal and bacteriostatic agents.
D. Provitamin A, B (complex).

181. Name the clinical signs of poultry escherichia.
A. Diarrhea, enterotoxemia, sepsis.
B. Abscess, phlegmon, furunculosis.
S. Asphyxia, asthesia, ataxia.
D. Hemorrhage, hyperemia, diarrhea.
182. What antigens are found in the causative agent of poultry escherichia?
A. O, K, H - antigens
B. Endo, exotoxin.
S. A, S, O - antigens.
D. Protein, lipoid, lipoprotein.
183. What is the death rate in poultry (young chicks) if Escherichia and Eymeriosis occur together?
A. 80-100%, 2006. RB Davlatov.
B. 70-80%, 2001. FA Niyazov.
S. 50-60%, 2000. AI Ibragimov.
D. 40-50%, 1992. AO Aripov.
184. Poultry escherichia should be distinguished from which diseases?
A. Salmonellosis, pasteurellosis, respiratory mycoplasmosis.
B. Gripp, Newcastle, marek.
S. Bursit, YUPPG, Gamboro.
D. Salmonellosis, flu, scurvy.
185. How to cancel the restriction on poultry escherichia?
A. Final disinfection 2 months after the last patient has been eliminated.
B. 45 days after the last patient has been eliminated.
Q. Last ill no 30 days from date after
D. Last ill no 21 days after making.
186. Respirator mycoplasmosis disease name when acceptance done?
A. 1961 XEB at the congress.
B. 1934 Microbiologicals in the association.
S. 1920 XEB at the congress.
D. 1900 XEB in the assembly.
187. Poultry Escherichia coli trigger how toxin separates?
A. Exo, endotoxin.
B. a, b, g toxins.
S. Proteolytic, lipolytictoxin.
D. Epilitrop, polytroptoxin.
188. Mycoplasmagallisepticum which disease trigger?
A. Respirator mycoplasmosis.
B. Marek.
S. Bursitis.
D. Infection laryngotracheitis.
189. Micro of the climate very suboptimal to be infection pathology how effect enough?
A. Strengthens.

B. Attenuates.
S. To the stimulus the majority factors participates.
D. This macroorganism tolerance depends.
190. Respirator in mycoplasmosis ill chickens what will be done?
A. Killed and burned.
B. Special, in insulators is treated.
S. Mandatory to the meat is submitted.
D. Feeding and then consumption is allowed.
191. Itlarolatin trigger who, when determined?
A. 1905 French scientist Karre.
B. 1926 Dunkin, Laidlaw.
S. 1932 MirolYubovI.
D. 1938 SyurinN.V., PankovV.A., LyuboshenkoS.Ya.
192. Dogs plague trigger which in the answer right written?
A. RNA preservative, paromyxoviride.
B. DNA preservative, poxviride.
S. RNA storage, oncoviride.
D. DNA storage, herpesviride.
193. The antigenic characteristics of the causative agent of canine plague are similar to which causative agents?
A. People measles, cattle plague
B. Influenza, smallpox, rabies.
S. Infectious hepatitis, influenza, Newcastle.
D. Bursitis, infectious bronchitis, ebola.
194. Dogs in the plague breeder in dogs scientist what %?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
195. In nature dogs of the plague reservoir tell me
A. Eat not and eat dogs.
B. All rodents.
S. Ticks, ticks, blood-sucking insects.
D. Wild predators.
196. % to the index of contagion of canine diseases?
A. 70-100%.
B. 50-60%.
S. 40-50%.
D. Depending on the factors.
197. According to the leading clinical signs, what is the form of canine plague?
A. Lungs, intestines, nerves, skin, mixed.
B. Respiratory, mixed, genital.
S. Neurosis, subclinical, immune subinfection.
D. Complex syndromes, reproductive.

198. What are the 6 symptoms that must be taken into account in canine distemper, and how many of them are considered to be a valid diagnosis?
A. Diarrhea, shortness of breath, eye-nose inflammation, palm perkeratosis, neurosis, the disease lasts at least 3 weeks. 4 of them.
B. Diarrhea, atelectasis, epilepsy, emphysema, aphonia, ataxia, 3 of them.
S. Conjunctivitis, ataxia, asthesia, hydrophobia, agalactia, adynamia, 4 of them.
D. Hydrophobia, diarrhea, hemorrhage, sepsis, septicemia, ataxia, 3 of them.
199. Which answer is correct about the 3 components of the epizootic chain?
A. The causative agent of an infectious disease, the route of transmission to the organism of a susceptible animal, a susceptible animal.
B. Alimentary, aerogenous, transmissible, genetic, sexual.
S. Horizontal, vertical.
D. Respiratory, conglomerative, contact way.
200. How is an infectious disease?
A. Clinical symptoms are clearly manifested, in the form of latent, immune subinfection.
B. Typical, atypical, abortive.
S. Acute, acute, semi-acute, chronic.
D. Reactive, areactive, tolerant.
201. What method is used to diagnose infectious diseases in the case of immune subinfection?
A. Diagnosis is made by the method of determining the titer of antibodies in the blood of a sick animal.
B. With bacteriological, virological, mycological examinations.
S. With RA, RP, KBR, RGA, GATR, IDR.
D. By the method of allergic examination.
202. Malleinization, tuberculinization is carried out by what methods?
A. Oftalmoprobe, between the skin, under the skin.
B. RA, MRA in a glass slide, test tube.
S. With all seroreactions.
D. With special diagnostics.
203. In what case is the diagnosis of an infectious disease considered correct?
A. If there is a laboratory test report.
B. Epizootological, clinical, pathanatomical.
S. Pathohistological, pathanatomical.
D. Rengenoscopy, fluoroscopy, lazeroscopy.
204. Who created the allergy diagnostic drug Mallein?
A. O. Kalning.
BIS Andriyevsky.
SMA Dukalov.
DAX Sarkisov.
205. Who created tuberculin allergic diagnostic drug?
A. XI Gelman.
BO Cal's.

SSN Vishelesky.
DMA Dukalov.
206. BCG vaccine against tuberculosis is used to vaccinate people and, if necessary, also animals. What does the word BSJ mean?
A. Initial letters of the words Bacterium, Kalning, Gelman in Latin spelling.
B. Biopreparat, sovremenn iy, jivotnix.
S. Biological, serum, zhivotnix.
D. Biopreparation, svobodenium, gelatin.
207. Who created Etis-1, Etis-2 drugs, a prophylactic and therapeutic agent against tuberculosis ?
AGX Mamadullayev.
BS Salimov.
SQN Norboyev.
DAO Oripov.
208. What is another name for follicular encephalopathy disease of cattle?
A. False escape.
B. False protein.
S. Fake Auyeski.
D. Bloody dysentery.
209. If all diseases are 100% according to the statistics of the Ministry of Health, how many of them are infectious diseases?
A. 0.3-0.5%.
B. 7-10%.
S. 14-16%.
D. 18-20%.
210. What kind of infection is called if the causative agent enters the susceptible organism?
A. Cryptogenic infection.
B. Regional infection.
S. Super infection.
D. Latent infection.
211. What is the name of an infection that can be found everywhere?
A. Ubiquitous infection.
B. Soil infection.
S. Water-air infection.
D. Natural infection.
212. How many diseases do rodents transmit to humans as reservoirs of infectious diseases?
A. About 20 - rabies, listeriosis, leptospirosis, tularemia, Auyeski...
B. More than 10.
About 30 S.
D. 10-12 diseases.
213. How many viral diseases have been found to be transmitted to humans by flies?
A. More than 150.

B. About 100.
Around S. 40-50.
D. to 20-30.
214. How many veterinary border points are operating in our country now?
A. 45.
B. on the scale of 14 Republics.
S. 8 provinces.
D. 12 railways, 11 airports.
215. Which method is the injection of hyperimmune blood serum?
A. Special prevention, special treatment.
B. General prevention.
S. General treatment.
D. Modern prevention, modern treatment.
216. What measure is used when an infectious disease is registered?
A. Quarantine, restriction, declared unhealthy.
B. Disinfection, disinsection, deratization.
S. Urbanization, dehydration, deacarization.
D. Termination of all communications.
217. Is it possible to sell healthy animals outside quarantined farms?
A. No, it is not possible.
B. You can check it again and then sell it.
S. If the serological reaction is healthy.
D. If it comes out healthy in IFA, PCR.
218. Give an example of the causative agents of infectious diseases that are extremely resistant to the effect of disinfectants.
A. Anthrax, anaerobic dysentery, bradzot, enterotoxemia, malignant tumor, scabies and other spore infections.
B. All clostridia.
S. All mycoplasmas.
D. All rickettsiae.
219. Biological method of disinfection.
A. Detoxification of manure by biothermal method.
B. Use of special cameras.
S. Use of UBN, IQN.
D. Application of thermal, chemo, partial pressure.
220. After how many days can an anthrax-vaccinated animal be slaughtered for meat?
A. After 14 days.
B. After 21 days.
S. After 3 days.
D. After 7 days.
221. Who discovered that there are several types of viruses that cause protein disease?
A. Corret, Valle 1922.
B. Lefler, Frosh 1898y.

SD Fracastro 1546.
D. Blondlo, Basov 1842.
222. Tell me what materials are taken from the animal during protein disease?
A. Liquid scraped from aphids.
B. Oral fluid, milk, urine of a sick animal.
S. Sick animal feces, urine.
D. Blood, feces of a sick animal.
223. How do protein disease serovars differ from each other?
A. Variants with shell amino acids.
B. Variant capsomeres by molecular weight.
S. With its clinical course in animals.
D. With which animal sensitivity.
224. How long after protein disease viruses enter a susceptible organism, antibodies begin to form?
A. After 4 days.
B. After 10 days.
Q. After 20 days.
D. After 10-14 days.
225. How many days before the appearance of clinical symptoms, rabies viruses begin to be shed in saliva?
A. 10 days ago.
B. 20 days ago.
S. 3-4 days ago.
D. 12-14 days ago.
226. How is protein disease in piglets, what is the death rate?
A. Myocarditis without aphthous, gastroenteritis 60-80% mortality.
B. Neurosis, neuritis, nephritis, death 90-100%.
S. Polymyelitis, caprostasis, mortality 60-80%.
D. Diarrhea, keratoconjunctivitis, mortality 90-100%.
227. VM Zhukorev found that 76.8% of the animals recovered from protein how long the immunity lasts?
A. 6 years.
B. 4 years.
S. 5 years.
D. 3 years.
228. What is the advantage of a universal vaccine made against protein over a regular vaccine?
A. The efficiency is 100%, the amount of consumption is small.
B. Vaccination is carried out by aerosol method.
S. At least 4 types are prevented.
D. No complication of vaccination.
229. How long will it take to vaccinate against that type and serovariant in the area where the protein came out?
A. 2 years - continuous.

B. 4 years - continuous.
S. 5 years - in a row.
D. 6 years - in a row.
230. Who is the scientist who used the name tuberculosis in science and when?
A. France, 1819. Lennik.
B. Hippocrates - VI century.
S. Abu Ali Ibn Sina - XI century.
D. Aristotle - VIII century.
231. What is the average annual percentage of cattle in Uzbekistan that is tested by an allergic method against tuberculosis, who determined it?
A. 0.016%, GH Mamadullayev.
B. 3-5%, AP Lee.
S. 2-3 %, VI Kan.
D. 6-8%, AK Sitdikov.
232. What happens to an animal with tuberculosis?
A. Delivered to meat within 15 days.
B. Killed and burned.
S. The meat is disinfected and transferred to the sausage.
D. " Stamping out " method is used.
233. Ascoli reaction on the skin is used to diagnose which disease?
A. Anthrax.
B. Dermatoses.
S. Dermatomycoses.
D. Tuberculosis.
234. State the merits and demerits of the Rozbengal trial.
A. A sensitive reaction is performed in a short time in 2-3 minutes, it is impossible to distinguish between naturally diseased and vaccinated animals.
B. High reliability, only naturally diseased animals are detected.
S. The level of reliability is low, only vaccinated cattle are detected.
D. It is not always possible to find a Rosbengal diagnostician, we spend little time.
235. Name the allergic diagnostic drugs used to detect tuberculosis.
A. PPD for cattle, PPD for poultry, LOW.
B. Tuberculin, cachegin, alttuberculin.
S. Allergen, urobilin, stercobilin.
D. Primary and Secondary PPDs.
236. Mycobacterium t. bovis are birds prone to?
A. No.
B. Prone.
S. Chicks up to 2 months are prone.
D. Sometimes you can get sick.
237. What type of viruses does the polyvalent vaccine used for the prevention of protein disease prevent?
A. A, O, Asia-1.
B. A, O, S.

S. Sat-1, Sat-2, Sat-3.
D. A, Asia-1, Sat-1.
238. After the recovery of the last sick animal in protein disease, the final disinfection is carried out and quarantine is canceled after how many days?
A. 21 days.
B. 14 days.
S. 30 days.
D. 45 days.
239. How much more time does vaccination have to be carried out on a farm registered as a protein?
A. At least 2 years.
B. 4-5 years.
S. 2-3 years.
D. This is determined by a thorough examination.
240. According to WHO, WHO, how many people die from tuberculosis in a year?
A. 4 million people.
B. 10 million people.
S. 1-2 mln.
D. 2-3 mln.
241. According to statistics, how many percent of people with brucellosis are correctly diagnosed?
A. 10-20%.
B. 20-30%.
S. 30-40%.
D. 40-50%.
242. Which serological reactions are often used in veterinary practice in the laboratory diagnosis of brucellosis?
A. Probasi of Rosebengal, AR, KBR.
B. IFA, PCR, RP.
S. GAR, GATR, RP, Askoli.
D. Askoli, Rosebengal, KBR.
243. How much is a positive reaction for cattle, sheep, goats, pigs AR - brucellosis?
A. 1:100 in cattle, 1:50 in small cattle.
B. 1:50 in cattle, 1:100 in small cattle.
S. 1:200 in cattle, 1:400 in small cattle.
D. 1:25 in cattle, 1:100 in small cattle.
244. How is the reaction of formation of milk in milk carried out if there is no diagnosticum?
A. Transfer is not possible.
B. Determinable but not reliable.
Q. Can be detected in urine.
D. Can be detected in feces.
245. In vaccination against brucellosis m.sh.h. What strain is the vaccine used for?
A. Rev-1 strain.

B. Strain - 19.
S. Strain - 68.
D. Strain - 62.
246. What strain is used in the vaccine used to vaccinate cattle against brucellosis?
A. Strain -19.
B. Rev-1 strain.
S. Nevsky - 12.
D. Strain - 68.
247. When and where was the infectious epididymitis of rams diagnosed?
A. 1942, New Zealand, Australia.
B. 1902, Italy, Hungary.
S. 1910, England, Norway.
D. 1860, Germany, Czechoslovakia.
248. Infectious epididymitis of rams should be distinguished from which diseases?
A. Diplococcosis, compilobacteriosis, salmanellosis, listeriosis, chlamydiosis.
B. Vibriosis, pasteurellosis, tuberculosis.
S. Aujeski, plague, escherichia.
D. Dermatomycosis, salmanellosis, vibriosis.
249. Rams, what should be done with an animal suffering from infectious epididymitis?
A. Submitted to meat.
B. Killed and burned.
S. Only testicles and internal organs are disinfected.
D. Treated, with hyperimmune blood.
250. What happens if the rabies virus is injected into a turtle?
A. It loses its pathogenicity.
B. Rash gets sick.
S. The sent site becomes necrotic.
D. Allergy in the turtle, anaphylaxis.
251. What diseases must be distinguished from rabies?
A. Auyeski, canine distemper, listeriosis, equine infectious encephalomyelitis.
B. Aujeski, follicular encephalopathy, pasteurellosis.
S. Visna-medi, Acobaltosis, pestilence.
D. Inan, leptospirosis, listeriosis.
252. Is there an oral rabies vaccine?
A. Boron, an improved granular antirabies vaccine.
B. No, not created.
S. Developed in the USA.
D. Yes, but ineffective.
253. In rabies, how long after the last diseased animal is destroyed is the restriction lifted?
A. After 2 months.
B. 1 month later.
Q. After 40 days.

D. 6 months later.
254. Which animals do not experience severe itching in Aujeski's disease?
A. Pig, marmot, marten.
B. Horse, donkey, mule.
S. Even ungulates.
D. Carnivores.
255. What is the death rate in young animals in Auyeski's disease?
A. 80-90%.
B. 70-80%.
S. 60-70%.
D. 50-60%.
256. What reactions are used in the serodiagnosis of Auyeski's disease?
A. IDR, KBR, IFR, BGAR.
B. RA, RP, RDP, KBR.
S. Askoli, RDP, RP, RA.
D. All serotests can be used.
257. What is the vaccine for Auyeski's disease?
A. BUK - 628 strain vaccine.
B. GOA, GOAF, live vaccine.
S. Leophile, leophobic vaccine.
D. Associated formal vaccine.
258. In what form does chicken pox occur mainly?
A. 90-94% of diphtheria.
B. Vesicle - papule form.
S. Hemorrhagic, gonino-fibrinous.
D. Fibrinous, purulent, hemorrhagic.
259. What kind of inclusions appear in the cells in smallpox?
A. Bollinger, Guarniella.
B. Babesh, Negri.
S. Emphysema, Atelectasis, Diuresis.
D. Hematopoiesis, lymphopoiesis.
260. Vesicular stomatitis is included in which group by XEB?
A. A - group, extremely dangerous infectious diseases.
B. B - group, dangerous infectious diseases.
S. Anthropozoonous diseases.
D. Zooanthroponous diseases.
261. Who studied leptospirosis in the veterinary department of Uzbekistan?
A. NJ Khudoiberdiyev, N. Shutyayev, E. Yaparov.
BAK Sitdikov, ID Burlussky, A. Abdusattarov.
S. Sh.A. Azimov, FI Ibodullayev, RE Sosov.
DAO Oripov, A. Rozimurodov, BS Murtazin.
262. Optimal conditions for leptospira?
A. Hydrobiont.
B. Thermostable.

S. Thermolabile.
D. Neuro, myotropic, anaerobic.
263. EChT in leptospirosis - what is acceleration related to?
A. Erythropenia, globulinemia.
B. Erythrocytosis, leukemia.
S. Poikilocytosis, hemoglobinemia.
D. Hemophilia, albumenemia.
264. Has a special prevention of necrobacteriosis been developed?
A. 1997 AA Sidorchuk and others. Nekovak.
B. 1950 S. Ya. Lyubashenko, GOAF vaccine.
S. 1920 AX Sarkisov, LTF.
D. 1970 AA Konopatkin, GOAF vaccine.
265. What antigens have been isolated from Listeria?
A. 15 somatic, 5 antigens.
B. About 10 antigens.
S. About 30 antigens.
D. 8 antigens.
266. What should be done to increase the effectiveness of therapeutic agents?
A. Determine their sensitivity and then use it.
B. Administration of broad-spectrum drugs.
S. Administer the dose 2-3 times larger.
D. Carrying out therapy together with autohemotherapy.
267. What are the types of tularemia?
A. USA, Tuyare Governorate California, European type, Asian type.
B. England, Netherlands.
S. Hungary, Bulgaria, Italy.
D. Canada, USA, Australia.
268. How many species of animals have been diagnosed with tularemia?
A. 125 species of vertebrates, 101 species of invertebrates.
B. All rodents and farm animals.
S. All kinds of insects and wild animals.
D. Humans and all creatures.
269. Which type of farm animals is highly susceptible to tularemia?
A. Sheep and lambs.
B. Cattle and calves.
S. Pigs and carnivores.
D. Wild animals and carnivores.
270. What diseases should be distinguished from tularemia?
A. Anaplasmosis, pseudotuberculosis, tuberculosis, coccidiosis, brucellosis.
B. Hemosporidiosis, pasteurellosis, escherichia.
S. Ringworm, botulism, listeriosis, leptospirosis.
D. Aujeski, protein, smallpox, ephemeral fever.
271. Can the tularemia vaccine be used to vaccinate animals?
A. No, ineffective.

B. Yes, it will.
S. It is possible for cattle.
D. Available to carnivores.
272. Who and when invented the vaccine for humans against tularemia?
A. 1946 B. Ya. Elbert, NA Gaysky.
B. 1912 Stanton, Fletcher.
S. 1954 Verge, Perimura.
D. 1932 Olds, Lewis.
273. What is another name for melioidosis?
A. False manga, Stanton, Fletcher's disease, Rangoon manga, pneumoenteritis.
B. Epizootic lymphangitis, dermatomycosis.
S. Purulent sap, yellow mite.
D. Persistent fever, fibrillation.
274. How is melioidosis different from ringworm?
A. With bacteriological examination and KBR.
B. With all serotests.
S. Allergy test.
D. Pathomorphological, pathogistological.
275. What are the special methods for treating melioidosis?
A. No specific treatment has been developed.
B. Symptomatic treatment is used.
S. Antibiotics, sulfonamides are used.
D. Convalescent blood and immunoglobulins.
276. What infection is the causative agent of the layer?
A. Wound infection.
B. Alimentary infection.
S. Respiratory infection.
D. Toxin infection.
277. What kind of toxin does the causative agent of the layer secrete?
A. Neurotoxin - tetanospasmin, hemotoxin tetanolysin.
B. Exo, endotoxin.
S. Mio and epidermotoxin.
D. Fibrino, peptidotoxins.
278. What special biopreparations have been developed against plaque?
A. Anotoxin (anti-inflammatory) half subcutaneous, half intravenous.
B. Immunoglobulin.
S. Hyperimmune serum.
D. So far no effective drug has been developed.
279. What kind of diseases should be distinguished from scurvy?
A. Rabies, from acute rheumatism of the muscles.
B. Auyeski, pestilence, protein, smallpox.
S. Pasteurellosis, salmanellosis, colidyspepsia.
D. Escherichia, meningitis, epilepsy.
280. Name the clinical signs of rabies.

A. Lower jaw, paralysis of legs, drooling, red eyes, aggression, hydrophobia.
B. Ataxia, asthesia, atonia, asthenia.
S. Chewing all objects, diarrhea.
D. Hydrophobia, blindness, fear.
281. How should the layer be prevented?
A. The injured animal is injected with an anti-fog anatoxin within 12 hours.
B. The wound is washed with disinfectants.
S. Aseptic, antiseptic agents are used.
D. It is placed in a warm room, glucose 40% is sent.
282. What was botulism called when it was first discovered?
A. 1820-1822. Kerner, Sausage poisoning.
B. 1900 IA Andrevsky, tetany.
S. 1842 A. Basov, meningitis.
D. 1910 IM Sechenev polyneuritis.
283. What does the word Botulus mean in Latin?
A. Sausage.
B. Spoiled meat.
S. This tribe.
D. Poison.
284. How many different toxins have been isolated from the causative agent of botulism?
A. 7 different.
B. 5 different.
Q. 3 different.
D. Exo and endotoxin.
285. Is the causative agent of botulism thermolabile or thermostable?
A. Thermostable.
B. Thermolabile.
S. This condition depends on the RN - environment.
D. Depends on the toxin released by the pathogen.
286. Which of the laboratory animals are sensitive to botulism toxin?
A. White mouse, guinea pig, rabbit.
B. Cats, puppies.
S. All young birds.
D. All animals are sentient.
287. What is the death rate in botulism?
A. 70-95%.
B. 50-60%.
S. 40-50%.
D. 100%.
288. Explain the mechanism of action of botulism toxins.
A. Acetylcholine is not released from the parasympathetic nerve endings, and communication with the central nervous system is interrupted, muscle tone decreases, angina pectoris in the heart, asphyxiation in breathing, and the animal dies.

B. As a result of cytopathogenic effect, myocarditis, endocarditis, epicarditis cause death.
As a result of S. Meningitis, the activity of the Kiss-Flyaka nerve node is disturbed, death occurs.
D. Angina, due to the failure of the ligament of Hissa, causes death.
289. What diseases should be distinguished from botulism?
A. Anthrax, rabies, Aujeski, listeriosis, stachyobotriotoxicosis, various poisonings, postpartum paralysis, acetonomy, Newcastle, Marek, canine plague.
B. Karason, bradzot, enterotoxemia.
S. Fodder poisoning.
D. Neuritis, neurosis, polymyelitis, epilepsy.
290. Can botulism be treated?
A. At the beginning of the disease, 600-900 thousand SB of special antitoxic serum is sent to horses.
B. Inefficient.
Q. It depends on the qualification of the treatment.
D. It can sometimes work.
291. When and in which country was the disease Ku-fever studied for the first time?
A. 1909-1910. USA, Ricketts.
B. 1812 France, N. Banaparte.
S. 1940 Russia, IP Pavlov.
D. 1970 Russia, RF Sosov.
292. Who are the reservoirs of ku-fever disease?
A. All animals and ticks.
B. Wild animals and insects.
S. Rodents, flies.
D. Most animals that live in water.
293. What treatment methods are used in ku-fever?
A. Symptomatic (antibiotics, sulfonamides).
B. Special, hyperimmune blood serum.
S. Immunoglobulin, convalescent blood.
D. Treatment does not work.
294. What is the death rate in ku-fever disease?
A. Death is rare or rare.
B. 40-50%.
S. 20-30%.
D. 60-70%.
295. When and who diagnosed infectious hydropericarditis?
A. 1925 Cowdrey.
B. 1900 F. Majandi.
S. 1910 LD Vinci.
D. Hippocrates, Aristotle BC.
296. What is the death rate in infectious hydropericarditis?
A. 50-90%, sometimes 100%?

B. Death is not observed.
S. 20-30%.
D. 30-40%.
297. Infectious hydropericarditis-way of transmission?
A. Transmissive, iksod mites.
B. Alimentary, respiratory.
S. Retrospective, parenteral.
D. Vertical, horizontal, cryptogen.
298. Who and when was the first to detect iron deficiency in human hair?
A. 1841-1843. AA Cazenov.
B. 1845 Malstem.
S. 1858 A. Ardi.
D. 1970-1980. AH Sarkisov.
299. Which vaccine is used for preventive and blood treatment purposes?
A. LTF-130.
V. GOA-formal vaccine against trichophytosis.
S. Yam and yam jams.
D. Iodine salts, antibiotics.
300. Who received the state award for his tetanus vaccine?
AAX Sarkisov.
B. Sh.T. Rasulov.
SMP Parmanov.
DAB Lee.
301. A mixture of 7% alkali, formalin, vaseline is effective in the treatment of which disease?
A. Temiratkini.
B. Psoriasis.
S. Dermatoses.
D. Vitiligo.
302. Who prepared the vaccine against sheep distemper?
A. Trichovis vaccine, MP Parmanov.
B. Mentovak, Sh.T. Rasulov.
S. SP-1, AB Lee.
D. LTF, LTF-130, AX Sadkisov.
303. The definition of microsporosis disease is written correctly in which answer?
A. Pathogenic fungi belonging to the genus Microsporum cause damage to the skin and skin products (wool, hair, nails).
B. Infectious skin disease, the causative agent is pathogenic bacteria.
S. Skin disease, not contagious, the causative agent is a pathogenic fungus.
D. All human, animal skin disease, wool falls, the wound is limited, the causative agent is bacteria.
304. From dermatomycoses - microsporosis is more common in which animals?
A. In horses.
B. Donkeys, in mules.

S. In ungulates.
D. In carnivores.
305. Who is the reservoir of microsporosis disease?
A. Rodents.
B. Insects.
S. Ticks, flies.
D. Wild birds.
306. Microsporosis should be distinguished from which diseases?
A. Iron, eczema, scabies, dermatitis.
B. From all dermatomycoses.
S. Of all dermatitis.
D. From tuberculosis, anthrax, brucellosis.
307. Who created a vaccine from a local strain in Uzbekistan against cattle distemper?
A. 2008 IX Salimov.
B. 2004 GH Mamadullayev.
S. 2005 XO Khamdamov.
D. 2010 BA Elmurodov.
308. According to AA Sidorchuk, what kind of toxins does the causative agent secrete?
A. Alpha, Betta, delta toxins.
B. Exo, endotoxins.
S. Neuro, cyto, myotoxins.
D. Hemo, neuro, epitheliotoxins.
309. What is the optimal condition of the Karason trigger in relation to oxygen?
A. Strict anaerobic.
B. Facultative anaerobic.
S. Strict aerobic.
D. Facultative aerobic.
310. What is the correct answer to the description of the death of a herd of rabies?
A. Occurs sporadically, mortality up to 80%.
B. In the form of epizootics, mortality is 60%.
S. In the form of enzootic, mortality is 40%.
D. In the form of panzootic disease, mortality is 40-50%.
311. Which laboratory animals are biotested for Karasan disease?
A. To the guinea pigs.
B. To the rabbit.
S. To the white mouse.
D. To cats and dogs.
312. When will the quarantine be canceled in the case of Karason disease?
A. After final disinfection 14 days after the loss of the last sick animal.
B. 21 days after the loss of the last sick animal.
S. 30 days after the loss of the last sick animal.
D. 45 days after the loss of the last sick animal.

313. How many hours after the death of an animal in Karasan's disease should the pet material be delivered to the laboratory with a referral letter?
A. Within 2-3 hours.
B. Within 8-10 hours.
S. Within 12-18 hours.
D. Within 1 day.
314. After how long an animal that has recovered from the disease is allowed to be slaughtered for meat?
A. After 30 days.
B. After 10 days.
Q. After 20 days.
D. After 14 days.
315. How long after the elimination of the last animal in Karasan disease is the quarantine canceled?
A. After 14 days.
B. After 21 days.
S. After 30 days.
D. After 45 days.
316. What are the permanent, observable clinical signs of paratuberculosis?
A. Diarrhea, paralysis of the rectal sphincter, weight loss.
B. Fever, fibrillation, polyuria.
S. Diarrhea, abdominal paralysis.
D. Ataxia, Atonia, Asthesia, hydrophobia.
317. Who and when invented the experimental module of paratuberculosis disease?
A. 1906 Bang, in the calves.
B. 1895 Yons, in cattle.
S. 1937 PP Vishnevsky, in cattle.
D. 1949 KA Dorofeyev, in sheep.
318. What allergen is used in allergic diagnosis in paratuberculosis?
A. Poultry alttuberculini, between the skin, 0.2-0.4 ml.
B. Tuberculin PPD for cattle, 0.2 ml.
S. Tuberculin PPD for poultry, 0.2 ml.
D. LESS kit.
319. The result of which serological reaction is more reliable in diagnosing paratuberculosis?
A. KBR - 83.3%.
B. RA, RP - 70%.
S. RDP, CUBR - 80%.
D. All serological reactions.
320. What treatment methods have been developed for paratuberculosis?
A. No effective treatment has been developed.
B. 1926 Valle, Ranger produced a live vaccine, but it caused an allergy in the body.
S. Hyperimmune blood serum developed.
D. It is recommended to determine the sensitivity to antibiotics and treat it.

321. What kind of disease is campylobacteriosis according to its clinical course?
A. Genital, reproductive disease.
B. Neurosis, polymyelitis.
S. Gastroenteritis diseases.
D. Heart and vascular diseases.
322. Cattle between the ages of 3 months and 4 years are more prone to black disease, tell me the reason?
A. Colostral immunity is stable in calves up to 3 months of age, and animals older than 4 years have a high immune background due to the fact that they have been vaccinated 8-9 times.
B. Calves up to 3 months of age are mainly fed milk and receive lactoglobulins.
S. Tolerance develops in animals older than 4 years.
D. Answers B and C are correct.
323. Name the main way of spreading campylobacteriosis.
A. Use of sick bulls - in escape.
B. Sick cows, calves and bulls.
S. Rodents, contaminated feed.
D. Blood-sucking insects, mites.
324. L. Pasteur used which organs experimentally infected with contagious pleuropneumonia disease of cattle as a vaccine?
A. Subcutaneous lymph nodes.
B. Neck, mesenteric lymph nodes.
S. Brain.
D. Thyroid gland, adrenal glands.
325. What happens to an animal infected with rinderpest?
A. It is lost by killing and burning.
B. Treated in a special place.
Q. Only hyperimmune serum is effective.
D. Compulsory submission to meat.
326. How long after the cancellation of the quarantine in case of rinderpest is it allowed to release the animals?
A. After 4.5 months.
B. After 6 months.
Q. After 1 year.
D. After 2-3 months.
327. What age group of cattle is more likely to suffer from catarrhal fever?
A. 1-4 years old.
B. 2-8 years old.
S. 6-10 years old.
D. Age does not matter.
328. Catarrhal fever of poor quality mainly occurs in which case?
A. 60-80% sporadic.
B. 60-80% epizootic.
S. 40-50% exotic.

D. 60-80% panzootic.
329. The brief definition of leukemia is written correctly in which answer?
A. Blood cancer, the appearance of immature stem blood cells - hemocytoblast, lymphoblast, meoloblast, prolymphocyte, monoblasts in the peripheral blood.
B. Occurrence of tumors in the reticulo-endothelial system.
S. Chronic infectious disease, occurrence of hemophilia.
D. Obvious appearance of hemorrhage, complete loss of coagulation.
330. Who has studied leukemia in depth in our country?
A. Professor XS Salimov and his students.
B. Professor AO Oripov and his students.
S. Professor BS Salimov and his students.
D. Professor FI Ibodullayev and his students.
331. Who is the scientist who founded experimental leuko-oncology in dogs?
A. Veterinary doctor, MA Novinsky 1876.
BA Shastny 1876.
S. Ye. Neumann in 1870.
D. R. Virkhov 1680.
332. Are leukosis viruses transmitted through ticks, bull, ram sperm?
A. No - XS Salimov, MQ Butayev.
B. O'tadi - KA Nizametdinova, ZE Roziyev.
S. Sometimes passes 20-30%, S. Primov.
D. This question is not resolved.
333. What percentage of pathology is observed in the spleen in cattle leukemia?
A. 100%.
B. 90%.
S. 80%.
D. 70%.
334. What is done with cattle infected with leukemia?
A. Compulsory submission to meat.
B. Treated with panflavin.
S. Treated with autovaccine.
D. It is lost by burning.
335. Which serological reaction is effective in bovine leukemia?
A. REED.
B. RA, KBR.
S. RP, Reaction Askoli.
D. Rosebengal, RA.
336. After the last leukosis seropositive animal is handed over for meat, after what other tests is the restriction lifted?
A. 3 times with an interval of 3 months, if a complete negative result is obtained.
B. 2 times with an interval of 2 months, if a completely negative result is obtained.
Q. 2 times with a 6-month interval, if the result is completely negative.
D. 1 time after 1 year with a completely negative serological test result.
337. What is the name of infectious rhinotracheitis of cattle in the literature?

A. Acute infection of the upper respiratory tract, conjunctivitis, infectious pustular vulvaginitis.
B. Lobar, lobular bronchopneumonia.
S. Emphysema, atelectasis, pleurisy.
D. Chronic, purulent pleuropneumonia.
338. Who studied infectious rhinotracheitis in our country?
A. IX Salimov, ZJ Shapulatova, G. Uzakova.
BGX Mamadullayev, AG'. Gafurov.
SXS Salimov, FI Ibodullayev.
DAO Oripov, Sh.A. Azimov, TB Boymurodov.
339. In which answer is the causative agent of infectious rhinotracheitis correctly written?
A. DNA storage herpesvirus BHV-1.
B. RNA-sparing poxvirus.
S. DNA-storing coronavirus.
D. RNA storage orthomyxovirus.
340. How many different stages of infectious rhinotracheitis according to clinical manifestations?
A. Respiratory, vesicular rash, conjunctival, meningoenciofelatic form.
B. Respiratory, enteral, genital.
S. Respiratory, neuritis, nephrosis, nephritis.
D. Pulmonary, genital, gastroenteritis.
341. In the treatment of infectious rhinotracheitis in calves, IX Salimov - with which drugs was 100% effective?
A. 1% baytril, iodinol and etonio suspension, with furozolidon.
B. Antibiotics (ampicillin, streptomycin).
S. Etonium, furozolidone, gentamicin.
D. Norsulfasol, streptomycin.
342. What kind of antibodies are produced in animals that recover from infectious rhinotracheitis?
A. VN, KB precipitating.
B. Hemagglutinating, VN.
S. KB, phagocytosis inhibitor.
D. Neutralizing, enhancing phagocytosis.
343. Who studied viral diarrhea of cattle in our country?
A. 1991 IX Salimov.
B. 1990 BA Elmurodov.
S. 1995 SI Mavlanov.
D. 1998. Sh.A. Jabbarov.
344. In which answer is the causative agent of bovine viral diarrhea correctly written?
A. RNA storage tag .
B. DNA-storing herpesvirus.
S. An RNA-storing poxvirus.
D. DNA-storing orthomyxoviride.

345. Cattle of what age are prone to viral diarrhea of cattle?
A. From 2 days to 2 years IX Salimov.
B. 2-4 months old.
S. Up to 6 months.
D. Up to 1 month.
346. State the rule for lifting the restriction in case of viral diarrhea of cattle.
A. A month after the last sick animal has been eliminated (healed), the restriction is canceled after the final disinfection is carried out.
B. 45 days after the last diseased animal has been eliminated.
S. 2 months after the last diseased animal has been eliminated.
D. The restriction is lifted 14 days after the final disinfection after the last diseased animal has been eliminated.
347. IX Salimov - IRT, PG-3, experimentally determined the therapeutic effect of hyperimmune blood serum prepared against colisalmanellosis?
A. 90.6%.
B. 80%.
S. 70-80%.
D. 50-60%.
348. What vaccines and biopreparations are available to prevent cattle from PG-3 disease?
A. Paravac, Bivak vaccines, Hyperimmune blood serum.
B. Chumvak, Rabikan vaccines, Convalescent blood.
S. Mono, polyvalent, 20A - vaccines.
D. Live, dead, lyophilized, weakened.
349. Who was the first to identify cattle ephemeral fever in Uzbekistan?
A. 1984 It was defined by XS Salimov and A. Mengliyev under the name Termiz fever.
B. 1984, 2002, 2013. A. Oripov identified.
S. 1984, 2010. T. Boymurodov identified.
D. 1980, 2006. F. Ibodullayev identified.
350. How is cattle ephemeral fever disease transmitted and spread?
A. Through Culex annulirosis, Anopheles annulipes and other blood-sucking flies.
B. Horizontal, vertical.
S. Alimentary, aerogenous, genital.
D. Contact, respiratory, genital.
351. How many cattle on the farm get sick when cattle ephemeral fever occurs for the first time?
A. 75-100%.
B. 20-30%.
S. 40-50%.
D. 60-70%.
352. What vaccines and biopreparations are used for cattle ephemeral fever?
A. Inactivated cultured vaccine, hyperimmune and convalescent serum.
B. Mono, polyvalent vaccines.

S. Leophilic, leophobic, live vaccines.
D. Quartz vaccine with GOA.
353. What is the Danish meaning of Bradzot?
A. Lightning fast death.
B. Liver necrosis.
S. Kidney turning into a pasty mass.
D. The transformation of the lungs, kidneys, spleen into a thick clot.
354. What is the death rate in Bradzot's disease?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
355. What diseases does ClostriDium DiDas cause?
A. Bradzot, infectious necrotic hepatitis.
B. All clostridiosis.
S. Layer, botulism.
D. Dangerous swelling, gas gangrene, blackness.
356. Bradzot bioassay test in which answer is correct?
A. The wound material was washed in GPB, filtered, and injected into 2 guinea pigs, dying in 16-48 hours (0.5-1 ml).
B. Patmaterial suspension 0.5 ml is injected into a white mouse, it dies in 7-8 hours.
S. Petmaterial filtrate 0.5 ml is injected into kittens, they die in 8-10 hours.
D. The bedding material is sterilized and the filtrate is given to lambs 2-4 months old, they die in 1-2 hours.
357. What biopreparations are available for the prevention of Bradzot's disease?
A. F. Kogan, AP Kolesev - vaccine and colianatoxin. Immunity - 10 months.
B. Polyvalent concentrated vaccine against clostriosis.
S. GOA, farmol, inactivated vaccine.
D. Live and dead, concentrated vaccine.
358. Anaerobic enterotoxemia of sheep is the causative agent of the disease and another name for the disease is correct in which answer?
A. Renal relaxation, Cl. perfingens-C.
B. Kidney stiffness, Cl. tetany.
S. Liver necrosis, Cl. oeDimatiens.
D. Pulmonary emphysema, Cl. DiDas.
359. Cl. What kind of colonies does perfingen0C form in the environment?
A. S-smooth, Rg'adir-budur, M-mushy.
B. Convex, concave, uneven edges.
S. Dark, reddish-brown.
D. Coral, emerald systems.
360. Do people also get sick with anaerobic enterotoxemia of sheep?
A. Gets sick.
B. Does not get sick.
S. Sometimes he gets sick.

D. Get sick if experimentally infected.
361. Is it possible to vaccinate sheep against anthrax, brazot, enterotoxemia and smallpox at the same time?
A. It is possible.
B. Impossible.
S. The interval must be at least 14 days.
D. Possible in extreme cases.
362. Chlamydial abortion disease of sheep was studied by whom in Uzbekistan?
AGN Nosirov, AK Sitdikov, A. Abdusattarov.
BFI Ibodullayev, M. Aminjonov, Sh.A. Azimov.
SJA Azimov, BS Salimov, EX Ergashev.
DOS Sodikov, Yo.Kh Torakulov.
363. Which serological reactions are used in the diagnosis of chlamydial abortion of sheep?
A. CBR and CUBR.
B. RA, RP, RDP.
S. RA, Ascoli reaction.
D. All serotests can be performed.
363. What serotests are used to detect sheep-goat plague?
A. 5 years.
B. Until death.
S. 3 years.
D. 2 years.
364. What is the name of infectious catarrhal fever of sheep in the literature?
A. Blutang, blue tongue, catarrhal fever.
B. Death at lightning speed, steppe.
S. Sudden death, cirrhosis of the liver.
D. Black spleen, hemophilic necrosis.
365. What is the death rate in Blutang disease?
A. 0-70%.
B. 90-100%.
S. 40-50%.
D. 30-40%.
366. What special treatment was created for Blutang's disease?
A. Undeveloped.
B. A hyperimmune serum was developed in the USA.
S. Polyanatoxin was developed in Australia.
D. Gamma globulin, interferon.
367. Infectious hoof rot disease of sheep should be distinguished from what diseases?
A. Necrobacteriosis, contagious ecthyma, smallpox, protein, pododermatitis, mechanical injury.
B. Brucellosis, tuberculosis, layer.
S. Rabies, Auyeski, Listeriosis.
D. Aqsil, emkar, olat, saramas.

368. What biopreparations are available for the prevention of infectious agalactia disease in sheep and goats?
A. Vaccines from Russia, Mongolia, Romania.
B. England, Australia, USA vaccines.
S. Italian, French, Caucasian vaccines.
D. Mongolia, Kazakhstan, Russian vaccines.
369. Who received the state award from VITI scientists for the vaccine developed for the prevention of infectious pleuropneumonia disease in goats?
AIA Dukalov, IS Polkovnikova, FD Lukashenko, SP Ilinov.
B. Sh.A. Azimov, Orifjanov, Burlusky.
SAK Siddikov, RX Khaitov, XZ Ibragimov.
DJK Sayfutdinov, EX Irgashev.
370. What is the death rate in infectious pleuropneumonia of goats?
A. 90-100%.
B. 80-90%.
S. 70-80%.
D. 60-70%.
371. Who and when found out that the people who were infected with camel plague in the deserts of Astrakhan and ate the meat of forcibly slaughtered camels were infected with plague?
A. 1907-1914. NI Klodnisky.
B. 1891 Kitazato, Yersin.
S. 1899 Junkovsky.
D. 1900 R. Koch.
372. Who are the reservoirs of causative agents of camel plague?
A. Sand mice, rats, rabbits.
B. Mice, bats, insects.
S. Flies, mosquitoes, ticks.
D. Reptiles, arthropods.
373. Can people get infected with the causative agents of camel plague?
A. Gets sick.
B. Does not get sick.
S. Sometimes he gets sick.
D. It depends on the pathogenicity, virulence of the pathogen.
374. Who invented the drugs Mallein, Tuberculin?
A. 1891 Gelman, Kalning.
B. 1882 Leffler, Schuetz.
S. 1907 Schubert, Schubert.
D. 1900 Romonovsky, Gimza.
375. Who else gets sick with Manka disease besides odd ungulates?
A. Camel, people and lion, tiger, lynx, bear.
B. All farm animals.
S. All wild animals.
D. All animals become infected when experimentally infected.

376. Ways to inject mallein in manka disease?
A. Eye, interdermal, subcutaneous.
B. Optoprobe, venous vessels.
S. The skin is scratched and applied.
D. The inhalation method is used.
377. Animals that have a positive reaction to Mallein must be checked by what other method?
A. KBR is placed.
B. RA, RP are placed.
S. IDR, CUBR.
D. RP, RA, Askoli.
378. What is the accuracy rate when injecting malleinization between the skin?
A. 95%.
B. 85%.
S. 75%.
D. 100%.
379. What should be done with an animal suffering from manka disease?
A. He is killed and burned with his skin.
B. Treated in a special place.
S. "Stamping out" method is used.
D. Must be slaughtered for meat.
380. How many days after the last diseased animal in Manka disease is lost, the final disinfection is carried out and the quarantine is canceled?
A. 45.
B. 21.
S. 14.
D. 2 months.
381. Who and when invented that the causative agent of horse sickness is a virus and pathological changes in blood producing organs?
A. 1904 Carré, Valle.
B. 1843 Ligney.
S. 1859 Anginiard.
D. 1932 Kolyakov, Romanov.
382. What is the reason for EChT acceleration in INAN disease?
A. Erythropenia, globulinemia.
B. Albuminemia, poikilocytosis.
S. Leukocytosis, monocytosis.
D. Hemophilia, lymphocytosis.
383. Tell the factors that cause the spread of INAN disease.
A. Swamps, puddles, blood-sucking insects.
B. Drought, ticks, ants.
S. Wild birds.
D. Thorny, stony pastures.
384. How are animals infected with INAN disease treated?

A. It is not treated, it is destroyed.
B. With special hyperimmune blood sera.
S. With convalescent serum.
D. Sensitivity to therapeutic agents is identified and then treated.
385. INAN disease should be distinguished from which diseases?
A. Piroplasmosis, nuttaliosis, trypanasomosis, leptospirosis, rhinopneumonia.
B. Manka, dumb, layered.
S. Epizootic lymphangitis, influenza.
D. Botulism, anthrax, colic.
386. In which answer is the description given to the dumb disease of horses correct?
A. It is acute, contagion, fever, purulent inflammation of the nasal cavity, throat mucous membranes, abscess under the jaw - appears.
B. It is chronic, contagion, fever, wounds appear on legs, abdomen, hooves.
S. Chronic night, head, eye, ear, throat abscesses.
D. Swelling appears in the ventral, caudal part of the body.
387. Clinical-syndromatic course of muteness is written correctly in which answer?
A. Typical, complicated, abortive, genital.
B. Typical, atypical, internal, external.
S. Alimentary, respiratory, skin form.
D. Exogenital, endogenital.
388. What is the correct answer for the trigger of the mute?
A. Streptococcus.
B. Staphylococcus.
S. Diplococcus.
D. Polyetiological characteristic.
389. How long does the immunity of people who recover from muteness last?
A. Lifetime.
B. 1 year.
S. 2 years.
D. Up to 5 years.
390. Horses are the causative agent of influenza in which answer is written correctly?
A. An RNA-storing orthomyxovirus.
B. DNA-storing herpesvirus.
S. RNA-sparing poxvirus.
D. DNA-storing coronavirus.
391. What is another name for rhinopneumonia of horses?
A. Viral abortion of ewes.
B. Equine conjunctivitis.
S. Respiratory pneumonia.
D. Pneumothorax, hydremia.
392. How is rhinopneumonia abortion different from salmonellosis abortion?
A. Abortion in salmanellosis 4-8 months, rhinopneumonia 7-11 months, laboratory examination.
B. In salmonellosis - necrotic abortion, in rhinopneumonia hemorrhagic abortion.

S. Salmonellosis is observed in abortion - paralysis in bia, hemophilia in rhinopneumonia.
D. Only IFA differs from PCR.
393. Which definition is correct for equine contagious pleuropneumonia?
A. Inflammation of liver, lungs and pleura.
B. Conjunctivitis, rhinitis, fibrinous stomatitis.
S. Diarrhea, caprostasis, polyuria.
D. Atonia, colic, ataxia.
394. In which answer is the treatment of KPP - a disease of horses correctly written?
A. 3-4.5 g. novarsenol 1:20, 1:30 in warm distilled water, intravenously (do not go under the skin!).
B. Glucose 40%, caffeine benzoate - intravenously.
S. Oxytetracycline 500 tb/kg - intramuscularly.
D. Laxatives.
395. Horses KPP - what to do with an animal that died from the disease?
A. The skin can be separated, the body is burned.
B. is thrown into the Becker well.
S. Given to carnivores.
D. It is destroyed by incineration in a special place.
396. Tell the optimum environment of Salmonella abortus equi.
A. Aerobic, anaerobic grow in all nutrient media.
B. Grows aerobically in a medium supplemented with egg yolk and serum.
S. Grows in an aerobic medium with added glucose and starch.
D. Grows only in tissue culture media.
397. Is there a recurrence of abortion with salmanellosis?
A. It doesn't happen.
B. Meets.
S. Occurs occasionally.
D. Occurs as a secondary infection.
398. What is the death rate of African plague in horses?
A. 95%.
B. 85%.
S. 75%.
D. 100%.
399. Name the reservoir in African swine fever?
A. Argas mites.
B. Bat.
S. Blood-sucking insects.
D. Birds, worms.
400. What are the main clinical signs of infectious atrophic rhinitis in piglets?
A. Deformation of the nose, pneumonia, pleurisy.
B. Diarrhea, ataxia.
S. Inability to stand, polyuria.
D. Symptoms of encephalitis.

401. Which answer is correct for anthropozoonous diseases?
A. Diseases transmitted from sick people to healthy animals.
B. Diseases transmitted to healthy people by sick animals and their products.
S. Diseases affecting all kinds of animals.
D. Diseases affecting only one type of animal.
402. Which answer to zooanthroponous diseases is written correctly?
A. Diseases transmitted to healthy people through sick animals and their products.
B. Diseases transmitted from sick people to healthy animals.
Q. Diseases affecting only one type of animal.
D. Diseases affecting all kinds of animals.
403. Ascoli's reaction is used to diagnose which disease?
A. Anthrax.
B. Layer.
S. Tuberculosis.
D. Rabies.
404. Are prions antigenic, can they cause disease?
A. Can provoke.
B. Can't provoke.
S. Depends on the degree of virulence.
D. Depends on the level of resistance of the macroorganism.
405. What is needed to make special reactions in the diagnosis of infectious diseases?
A. Special diagnostics.
B. Feather material from diseased animals.
S. Excreta of sick animals.
D. Blood serum of sick animals.
406. Name the most common clinical sign of leptospirosis?
A. Hemoglobinuria.
B. Hemophilia.
S. Hemostasis.
D. Hemocoagulation.
407. Name the most common clinical sign of rabies?
A. Hydrophobia.
B. Hemoglobinemia.
S. Hyperergic allergy.
D. Ataxia, asthesia.
408. Is sensitization used in veterinary practice?
A. Yes (vaccinating...).
B. In meeting the need for vitamins.
S. In meeting the need for micronutrients.
D. Stabilizing blood pH .
409. Name special biopreparations for the treatment of infectious diseases.
A. Hyperimmune blood serum, convalescent blood serum.
B. Broad-spectrum antibiotics.
S. Compounds with nitrofuran.

D. Specific compounds.
410. Types of disinfection are given correctly in which answer?
A. Preventive, final, current, mandatory... disinfections.
B. It is classified according to which disinfectant is used.
S. It varies according to the seasons of the year.
D. It is classified according to object types.
411. In which answer is the abortive course of the disease written correctly?
A. Short duration of the disease, mild form, no symptoms characteristic of the disease.
B. Sporadic course of the disease, long duration.
S. Several occurrences of the disease in at least 20-30% of the animals in the herd.
D. Recurrence of the disease.
412. Types of agglutination reaction?
A. Direct and indirect.
B. Differentiates depending on the type of antigens.
S. Varies with execution style.
D. It is classified according to the quality of the result.
413. Use of allergic reactions in veterinary practice.
A. Allergic reactions are used in the diagnosis of infectious diseases.
B. According to the content of allergic reactions, it is considered a therapeutic agent.
S. Allergic reaction is necessary in the determination of preventive measures.
D. Allergic reactions are used to diagnose all infectious diseases.
414. Antigens and their types are written correctly in which answer?
A. Heterogeneous, metabolic, perfect, imperfect, protective antigens.
B, Protein and lipoid antigens.
S. Complex biopolymer antigens.
D. Antigens soluble in water, fat, alcohol.
415. Explain strict and facultative aerobic microorganisms?
A. Can only live where there is oxygen, can only live where there is no oxygen.
B. Aerobic microorganisms are encapsulated in the presence of oxygen.
S. Aerobic microorganisms do not grow in the absence of oxygen.
D. All aerobic microorganisms are the causative agents of infectious diseases.
416. Can haptens cause an infectious disease?
A. Can't trigger.
B. It can stimulate if combined with small molecular proteins.
S. Can trigger only under optimal conditions.
D. Depends on the level of resistance of the macroorganism.
417. Rose Bengal test is used in the diagnosis of which disease?
A. Brucellosis.
B. Tuberculosis.
S. White.
D. Pasteurellosis.
418. Where do tumors in Karason's disease appear more often?
A. In the parts where the muscles are well developed.

B. Mainly in the ventral parts of the body.
S. In the dorso-lateral part of the body.
D. In the caudal and ventral parts of the body.
419. Why is fowl pox called black pox?
A. As a result of secondary infection, purulent, diphtheritic inflammation is common.
B. Because the disease is chronic.
S. Because the lesions are superficial and deep.
D. Because the stimulus is epitheliotropic, myotropic.
420. Ways to inject mallein in manka disease?
A. Eye, interdermal, subcutaneous.
B. Optoprobe, venous vessels.
S. The skin is scratched and applied.
D. The inhalation method is used.
421. Adhesion feature?
A. Adhesion of two different bodies.
B. Tissue rupture.
S. A type of decay.
D. State of necrosis.
422. What is adenoma?
A. Benign tumor arising in glandular tissue.
B. Malignant tumor in muscle tissue.
S. Malignant tumor of connective tissue.
D. Bone, tendon tissue necrosis.
423. What are adsorbents?
A. Pharmacological agents that absorb other substances.
B. Substances soluble in all solvents.
S. Complex solutions.
D. Mucous substances.
424. What are allergens?
A. Allergy-causing substances of antigenic or hapten nature.
B. Inorganic substances causing agglutination.
S. Anaphylaxis-inducing substances.
D. Dissociated amino acids.
425. What is anaphylaxis?
A. In the second parenteral injection of an allergen into the body - a severe manifestation of an allergic reaction.
B. A type of parabiosis, agglutination.
S. Pinocytosis, a type of poikilocytosis.
D. Immunization subinfection.
426. What is Antigen Drift?
A. Small changes of some viruses during evolutionary development.
B. Changes in viruses during ontogenesis.
S. Replication of viruses in phylogeny.
D. Democracy line.

427. What is the antigen ceiling?
A. Change in the antigenic nature of viruses.
B. Increased virulence of viruses.
S. Reduction of pathogenicity of viruses.
D. Pathogenicity, increased virulence.
428. Antibodies are divided into what immunoglobulins?
A. A, M, Ye, D, G - immunoglobulins.
B. a, b, g - globulins.
S. Properdik, to interferons.
D. To leukocytes, neutrophils, lymphocytes.
429. What is the function of A - immunoglobulins?
A. It neutralizes bacteria, viruses..., exo, endotoxins, snake and blackworm poisons.
B. Neutralizes all chemical poisons.
C. Neutralizes protein poisons.
D. Neutralizes very strong poisons.
430. Ascoli's reaction is used in which disease and in the examination of what kind of material?
A. Anthrax, leather and leather products.
B. In tuberculosis, lungs.
S. Aksilda, aftani.
D. Brain in rabies.
431. What is asthenia?
A. A state of extreme fatigue, weakness, is observed in most infectious diseases.
B. Loss of sensitivity.
S. Loss of movement balance.
D. Absolute loss of reflexes.
432. What is ataxia?
A. Movement balance is lost.
B. Defecation stops.
S. Sensibility is lost.
D. Loss of vision.
433. What is an autovaccine?
A. A vaccine prepared from a culture isolated from sick animals, using it in that farm, area.
B. Vaccine prepared from special tissues.
S. A vaccine that meets standard requirements.
D. Associated vaccine.
434. What is autoimmunity?
A. Immunization with antigens from the body itself.
B. Type of monoimmunization.
S. Type of polyimmunization.
D. Repeat immunization.
435. What are aflatoxins?
A. Strong toxins secreted by fungi belonging to the genus Aspergillus.

B. Strong toxins released by viruses.
S. Toxins secreted by Clostridia.
D. Toxins for which antidotes have not been developed.
436. What event (indicator) is accepted as a unit of virulence?
A. Bacteria, viruses, 50% of laboratory animals killing dose accepted LD-50.
B. The unit of virulence is LD-100.
S. The unit of virulence is LD-1 %.
D. The unit of virulence is LD-25 %.
437. What is the infectious dose ID-50%?
A. Dose to produce disease symptoms in 50% of animals.
B. Inducing death in 50% of animals.
S. 50% disease of the whole animal.
D. One sentence with LD-50.
438. What is a dezobarrier?
A. At the entrance to the farm, there is a special concrete area where a disinfectant is placed.
B. Extermination of insects.
S. Extermination of mites.
D. Extermination of rodents.
439. What is interferon?
A. A small molecule glycoprotein present in a virus-infected cell.
B. Specific antibody.
S. Antibacterial antibody.
D. Antibodies that neutralize toxins.
440. What other diseases does the vaccine prepared against diplococcosis in young animals prevent? Who developed this vaccine when?
AAG Malyavin, 1956, diplococcosis, salmonellosis, pasteurellosis.
B. Streptococcus, colibacteriosis, pneumococcosis.
S. Scabies, necrobacteriosis, hoof rot.
D. Colibacteriosis, salmonellosis.
441. Who developed the vaccine against diplococcosis in young animals in Uzbekistan?
ABA Elmurodov.
BTB Boymurodov.
SFI Ibodullayev.
DA Rozimorodov.
442. Colibacteriosis is more common in calves of which age?
A. 2-7 days old.
B. At 1-2 months.
S. is 5-6 months old.
D. Age up to 1 month.
443. At what age is colibacteriosis more common in poultry?
A. From 1 day to 90 days, when entering the egg.
B. Up to 6 months, during the period of puberty.

S. Up to 1 year.
D. At 2-4 months.
444. What else is salmonellosis called in the literature?
A. Paratyph.
B. Belly fold.
S. Endometritis, enteritis.
D. Blood dysentery.
445. Who studied salmonellosis in Uzbekistan?
AAM Akhmedov.
BMM Muradov.
SBYa. Yarmatov.
DNG Shatonin.
446. What is the death rate in equine infectious encephalomyelitis?
A. 80%.
B. 60%.
Q. 40%.
D. 100%.
447. What is the death rate of African plague in horses?
A. 95%.
B. 85%.
S. 75%.
D. 100%.
448. What is the form of rhinopneumonia in horses?
A. Respiratory, abortive, nervous, genital.
B. Respiratory, gastrointestinal form.
S. Skin, genitals, eyes.
D. Genital, alimentary, myopathological.
449. Has a special treatment been developed for INAN disease?
A. Undeveloped.
B. Immunoglobulin.
S. Convalescent serum.
D. Yes, hyperimmune serum.
450. What is the reason for INAN to increase in July-August?
A. Increase in blood-sucking insects.
B. The heat of the temperature in summer.
S. Horses in the sun a lot in summer.
D. Infrared rays.
451. Who among Ru0Clims wrote the textbook "Private Epizootology" in what year and what prize did this book receive?
A. SN Vy Shelessky, Ye.Ya. Mazel - 1935. State award.
B. IP Pavlov, IM Sechenev - 1924. State award.
SSP Botkin - 1901. State award.
DMS Gannushkin - 1940. State award.
452. What is the incidence rate, %?

A. % of the number of infected animals in relation to the total number of susceptible animals.
B. % of the number of infected animals in relation to the total number of all species of animals on the farm.
S. % of the number of infected animals in relation to the number of susceptible animals in the district.
D. The ratio of the number of infected animals to the number of non-infected animals.
453. What is the gorge of fatality?
A. % of the number of animals that died from the disease in relation to the total number of susceptible animals.
B. Ratio of total susceptible animals to number of dead animals.
Q. How many infected animals died in %.
D. Rate of disease transmission.
454. What is the death rate?
A. Calculation of how many of the infected animals died in %.
B. % of the dead animal relative to the total number of animals on the farm.
S. % of infected animals in relation to the number of animals in the district.
D. Expression of the number of infected and healthy animals on the farm in %.
455. In what experiment did SS Andreyevsky prove that the causative agent of anthrax in humans and animals is 1?
A. July 18, 1788. In the city of Chelyabinsk, he injured himself with the blood of a sick animal.
B. Invented the Ascoli reaction.
S. Kultura isolated.
D. "Coral system" - discovered the experiment.
456. What is the death rate of anthrax in horses and sheep?
A. 90%.
B. 80%.
S. 70%.
D. 60%.
457. How is the causative agent of anthrax called according to its oxygen requirement?
A. Facultative aerobic.
B. Strict aerobic.
S. Facultative anaerobic.
D. Strict anaerobic.
458. In natural conditions, poultry are not susceptible to anthrax, what is the reason?
A. Their body temperature should be 41-42°.
B. That they have tolerance.
S. Strength of sensitization.
D. High antibody titer.
459. Can poultry be experimentally infected with anthrax?
A. Yes, he gets sick if he lowers his body temperature and gets infected.

B. Can be transmitted in thermostatic conditions.
S. Can be transmitted parenterally.
D. Can be transmitted by respiratory route.
460. Who updated the map of anthrax hotspots in Uzbekistan?
AXS Salimov, BS Saidkulov, G'. Mengliyev.
BRX Khaitov, AO Oripov.
S. Sh.A. Azimov, BS Salimov.
DA Orifjanov, ID Burlusky.
461. What is the death rate in sheep and lambs in relapsing disease?
A. 95-100%.
B. 80-90%.
S. 70-80%.
D. 50-60%.
462. Name the most effective special treatment for eczema?
A. Administration of anatoxin against the layer.
B. Administer the vaccine.
S. Hyperimmune serum.
D. Comprehensive treatment.
463. Name the most effective prevention of susceptible animals against scurvy.
A. Active immunization, inoculation with concentrated anatoxin against rabies.
B. Treatment of the injury.
S. Administration of antibiotics.
D. Administration of bactericidal drugs.
464. What kind of infection is the causative agent?
A. Wound infection.
B. Universal genital infection.
S. Severe respiratory infection.
D. Alimentary infection.
465. According to US scientists, 1 ounce (28.3 g) of botulism toxin kills how many cows?
A. 24 thousand.
B. 10 thousand.
S. 50 thousand.
D. 100 thousand.
466. How many mice does 1 g of botulism toxin kill?
A. 60 billion mice or 120 thousand tons.
B. 10 billion mice or 12 thousand tons.
S. 20 billion mice or 24 thousand t.
D. 30 billion mice or 36 thousand tons.
467. Name animals that are more resistant to botulism toxin?
A. Dog, wild carnivores, rat.
B. Horse, cattle, camel.
S. Pig, poultry, donkey.
D. Pets.

468. What is the optimal therapeutic dose of botulism special serum for horses?
A. 600 and more AYe, to the vein.
B. 200 and more AYe, to the vein
S. 500 and more AYe, between the muscle.
D. 100-200 AYe, intravenously.
469. How is pasteurellosis?
A. Septicemia.
B. Neuroplegia.
S. Atelectasis.
D. Angina.
470. What is the oxygen requirement of Pasteurella?
A. Facultative anaerobic.
B. Strict anaerobic.
S. Facultative aerobe.
D. Strict aerobic.
471. What is the difference between Pasteurella serotypes A, B, C, D?
A. It differs in morphological, virulence, pathogenicity, and antigenic characteristics.
B. Thermostable, with thermolability.
S. Aerobic, with anaerobic.
D. By type of toxins.
472. Pasteurellosis occurs in which species of animals in epizootic conditions?
A. In poultry.
B. In domestic animals.
S. In wild animals.
D. In carnivores.
473. How many types of tuberculosis causative agents are there?
A. Humanus, bovium, avium (Mycobact. root).
B. Sheep, goats, cattle, horses, camels.
S. Mouse, rat, bird.
D. Wild animals.
474. Tell the clinico-syndromatic classification of tuberculosis?
A. Lung, gastrointestinal, bone, skin form.
B. Muscle, nerve, tendon, glandular form.
S. Support organs, lungs, heart shape.
D. Udder, shape of lymph glands.
475. According to RF Sosov, how many pathogens does one sick cow excrete with feces in 1 day?
A. 37 million/day.
B. 10 million\ in 1 day.
S. 15 million\ in 1 day.
D. 20 million\ in 1 day.
476. Pathanatomical changes characteristic of tuberculosis - internal organs?
A. "Pearls of tuberculosis" - gemchug.
B. Pulmonary atelectasis.

S. Pulmonary emphysema.
D. Lobar and lobular inflammations.
477. Who discovered tuberculin?
AR Koch 1882, XI Gelman, Gering 1892.
BVA Bashenin, 1936.
S. Fontes, 1910.
D. Sil-Nielsen, 1890.
478. What drugs are used in allergic diagnosis of tuberculosis?
A. PPD for cattle, PPD for poultry, KAM.
B. BSJ - for people.
S. PPD and mallein.
D. A sick animal can use eye drops.
479. What are ETIS-1 and ETIS-2 drugs used for?
A. It is used as a preventive and therapeutic agent in tuberculosis.
B. As a diagnostic tool.
S. As a preventive measure.
D. Special remedy.
480. What should be done with animals that have a positive reaction to tuberculosis allergy test?
A. Within 15 days at the latest, it will be handed over to the meat.
B. Held in isolation.
S. Killed and burned.
D. It is fed to the stomach.
481. Do people also get infected with sheep pseudotuberculosis?
A. Get sick.
B. Does not get sick.
S. If the resistance decreases, he will get sick.
D. Get sick as a mixed infection.
482. How many groups are infectious diseases divided according to the XEB list?
A. into 3 groups, A, B, C.
B. into 2 groups, A, B.
S. to 5 groups.
D. into 4 groups.
483. Brucella was found to be released from a sick animal?
A. All excreta, discarded fetus.
B. Fecal, through urine.
S. Respiratory air, nasal fluid.
D. Through milk, urine.
484. Tell the advantage of Rozbengal test?
A. The level of accuracy is high, it is determined whether the animal is sick or healthy in a short time.
B. It costs less.
S. The work is easy, the result is reliable.
D. Can be won once a year.

485. Tularemia is more common in which animals?
A. In rodents.
B. In ungulates.
S. In ungulates.
D. In carnivores.
486. Is allergic diagnosis used in tularemia?
A. It applies.
B. Not applicable.
S. It is used when the disease is severe.
D. In some extreme cases.
487. Does tularemia also occur in humans?
A. It happens.
B. Does not occur.
S. Occurs occasionally.
D. Occurs in extreme conditions.
488. Tell listeriosis clinical-syndromatics.
A. Neurosis, sepsis, abortion, mastitis.
B. Pneumonia, necrotic hepatitis.
S. Nephritis, nephrosis, architis.
D. Encephalitis, genetic pathology.
489. What is another name for melioidosis?
A. False manka, Stanton's disease, Fletcher manka.
B. Lymphadenitis, pneumoenteritis.
S. Uremia, pneumonia.
D. Encephalomyelitis, Rangoon Manga.
490. What group of diseases does ku fever belong to?
A. Rickettsioses.
B. Clostridiosis.
S. Mycoses.
D. Mycotoxicosis.
491. Is ku malaria contagious to humans?
A. It is contagious.
B. It is not contagious.
S. If the resistance decreases.
D. It is sometimes contagious.
492. What group does hydropericarditis belong to?
A. Rickettsioses.
B. Clostridiosis.
S. Chlamydiases.
D. Mycoplasmosis.
493. Which group does keratoconjunctivitis belong to?
A. Rickettsioses.
B. Balantidiosis.
S. Psoroptosis.

D. Mycoplasmosis.
494. Who studied leptospirosis in Uzbekistan?
ANJ Khudoiberdiyev.
BDX Narziyev.
SNM Matchanov.
D. Sh.A. Azimov.
495. S. Ya. Lyubashenko - according to information, how can leptospira be distinguished from a sick animal?
A. With feces, urine, milk.
B. With all excreta.
S. With breath air.
D. With an abandoned fetus.
496. How is leptospirosis?
A. In the form of immune subinfection.
B. With clear clinical signs.
S. Without clinical signs.
D. Not all characters are known.
497. What is the death rate of leptospirosis in fur animals?
A. Up to 90%.
B. Up to 80%.
S. Up to 70%.
D. Up to 60%.
498. How is rabies transmitted?
A. When bitten by a rabid animal.
B. In contact.
S. Horizontal.
D. Vertical.
499. What to do with a rabid animal?
A. It is destroyed by burning with the skin.
B. Buried in a special place.
S. is thrown into Becker's well.
D. Disinfection with 20% slaked lime.

500. Is it possible to heal an animal with signs of rabies?
A. It is not possible.
B. If hyperimmune serum.
S. Possibly, if gammaglobulin.
D. If there is convalescent blood.

VIII. Evaluation

Students' mastery of subjects is evaluated on a 5-point scale.

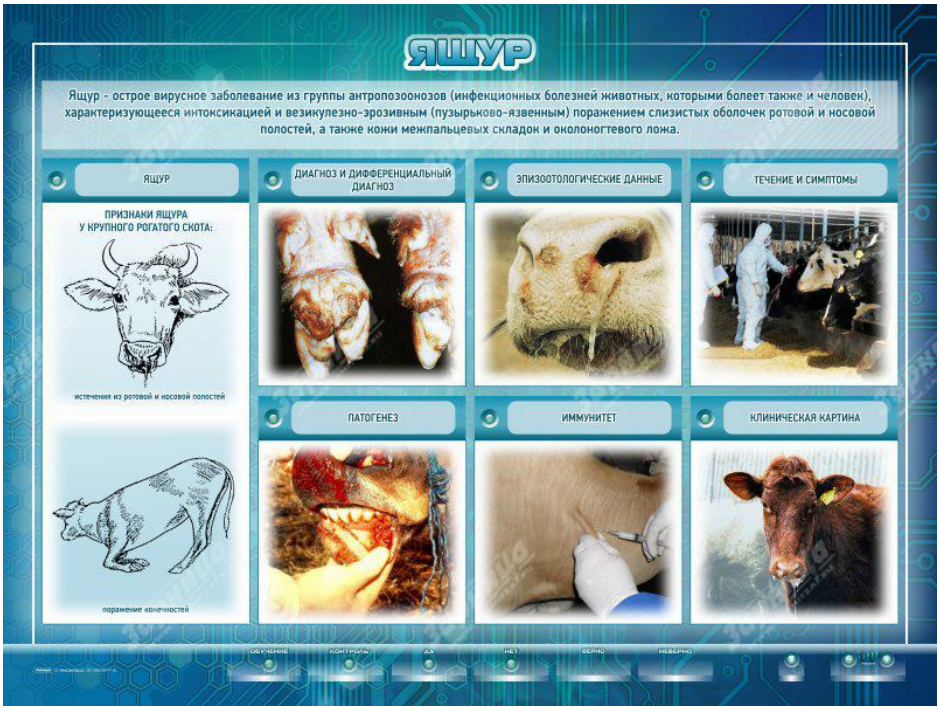
5 (excellent) rating:
Summary and decision acceptance to do;
Get creative ideas;
Independent in observation walk get;
Received knowledge in practice apply get;
The essence understanding;
To know, to tell;
Imagination have to be

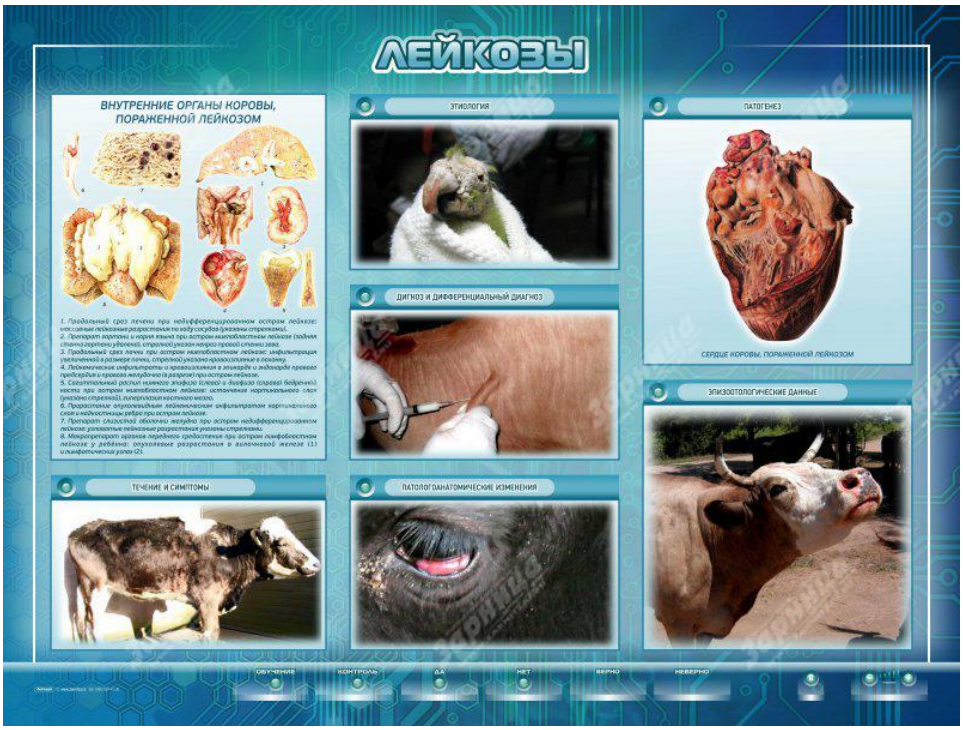
4 (good) grades:
Independent in observation walk get;
Received knowledge in practice use;
Understanding the essence;
To know, to tell;
Imagination have to be

3 (satisfactory) grade;
Understanding the essence;
Knowing, telling to give
Imagination have to be

2 (unsatisfied) rating:
Failure to master the program;
Ignorance of the essence of science;
Not having a clear vision;
He cannot think independently

Science handouts





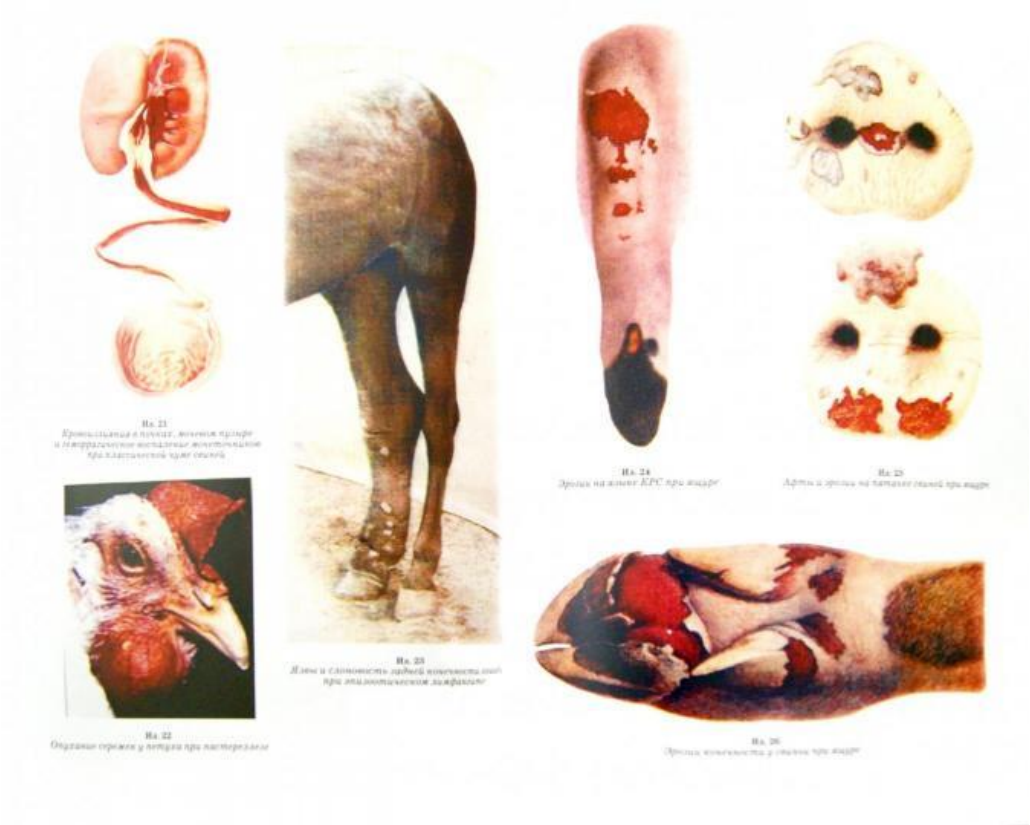

ВЕЩЕСТВА ДЕЙСТВУЮЩИЕ ПРЕИМУЩЕСТВЕННО НА ЦЕНТРАЛЬНУЮ НЕРВНУЮ СИСТЕМУ
АНАЛЬГЕТИЧЕСКИЕ ВЕЩЕСТВА
ПРОТИВОСУДОРОЖНЫЕ ВЕЩЕСТВА
НЕЙРОЛЕПТИЧЕСКИЕ ВЕЩЕСТВА
НАРКОТИЧЕСКИЕ ВЕЩЕСТВА
СЕДАТИВНЫЕ ВЕЩЕСТВА
СНОТВОРНЫЕ ВЕЩЕСТВА
СНОТВОРНОЕ СРЕДСТВО
ЖАРОПОНИЖАЮЩИЕ ВЕЩЕСТВА
ОБУЧЕНИЕ
КОНТРОЛЬ
ДА
НЕТ
ВЕРНО
НЕВЕРНО


ОСНОВНЫЕ ПРИЗНАКИ ОСТРЫХ ОТРАВЛЕНИЙ ЖИВОТНЫХ ЛЕКАРСТВЕННЫМИ ВЕЩЕСТВАМИ
Отравление лекарственными средствами – это распространенный тип отравлений, случающихся, в основном, по причине самостоятельного назначения препаратов владельцами своим животным.
Неотложная помощь:
– в первую очередь немедленно вызовите ветеринарного врача
– если животное находится в сознании и прошло мало времени с момента поглощения препарата – промойте желудок
– дайте любое обволакивающее средство для предотвращения дальнейшего всасывания вещества в организм (молоко, сливочное масло, сырое яйцо)
РАСШИРЕНИЕ ЗРАЧКОВ
КОЖА
ДЫХАНИЕ
РВОТА
ПРЕПАРАТЫ ВЫЗЫВАЮЩИЕ ОТРАВЛЕНИЕ У ЖИВОТНЫХ
– адреналин
– аминазин
– анальгин
– андипсин
– антабус
– антибиотики
– борная кислота
– витамин D2
– димедрол
– препараты железа
– кофеин
– йод
– калия перманганат
– медь сульфат
– новокаиновый спирт
– фенацетин
– противосудорожные препараты (седуксен, нейролептики, нитрофурал)
– парацетамол (ацетаминофен)
– пилокарпин
– перекись водорода
– эскадефедрин
– спирт этиловый (этанол)
ШОКОВОЕ
СУДОРОЖНОСТЬ
МЫШЕЧНАЯ СЛАБОСТЬ
ОБУЧЕНИЕ
КОНТРОЛЬ
ДА
НЕТ
ВЕРНО
НЕВЕРНО

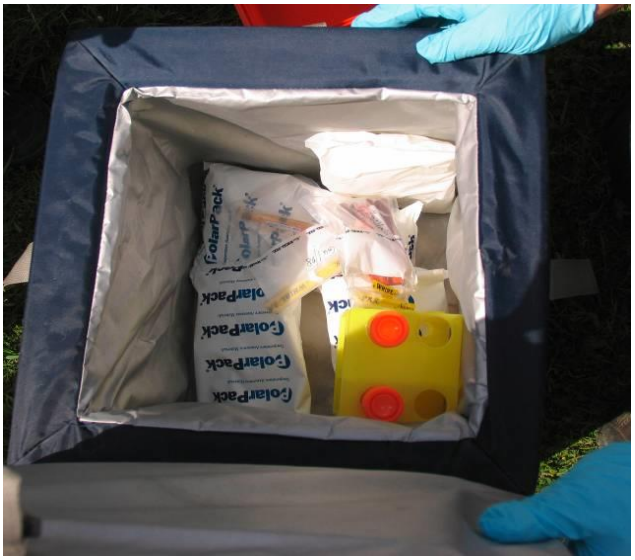

ПРОТИВОМИКРОБНЫЕ
И ПРОТИВОПАРАЗИТАРНЫЕ ВЕЩЕСТВА
ДЕЗИНФИЦИРУЮЩИЕ СРЕДСТВА
АНТИСЕПТИЧЕСКИЕ СРЕДСТВА
ХИМИОТЕРАПЕВТИЧЕСКИЕ СРЕДСТВА

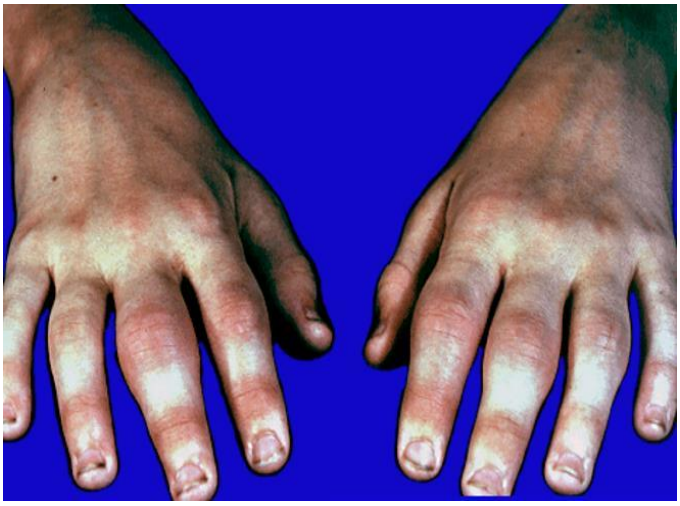

ЭПИЗООТОЛОГИЯ
ЯЩУР
БРУЦЕЛЛЕЗ
САП
ЧУМА КРУПНОГО РОГАТОГО СКОТА
ЧУМА СВИНЕЙ
ГРИПП ПТИЦ
ОБУЧЕНИЕ
КОНТРОЛЬ
ДА
НЕТ
ВЕРНО
НЕВЕРНО


ТУБЕРКУЛЕЗ, ПАСТЕРЕЛЛЕЗ, ОСПА ПТИЦ
ОСПА ПТИЦ
ОПИСАНИЕ
ИСТОРИЧЕСКАЯ СПРАВКА
ВОЗБУДИТЕЛЬ БОЛЕЗНИ
ЭПИЗООТОЛОГИЯ
ТЕЧЕНИЕ И КЛИНИЧЕСКОЕ ПРОЯВЛЕНИЕ
ЛЕЧЕНИЕ
МЕРЫ БОРЬБЫ
ТУБЕРКУЛЕЗ
РАСПРОСТРАНЕНИЕ
ЕСТЕСТВЕННЫЕ И ЭКСПЕРИМЕНТАЛЬНЫЕ ХОЗЯЕВА
ПЕРЕДАЧА
ПРИЗНАКИ ЗАБОЛЕВАНИЯ
ТУБЕРКУЛИНОВАЯ ПРОБА
ЛЕЧЕНИЕ ТУБЕРКУЛЕЗА
ПАСТЕРЕЛЛЕЗ
ОПИСАНИЕ
ТЕЧЕНИЕ И КЛИНИЧЕСКОЕ ПРОЯВЛЕНИЕ
ЛЕЧЕНИЕ
ОБУЧЕНИЕ
КОНТРОЛЬ
ДА
НЕТ
ВЕРНО
НЕВЕРНО




САП ЛОШАДЕЙ
ПРИЗНАКИ БОЛЕЗНИ


PolarPack




Основные причины заболеваний


Abdel Khaleq Sultan (RFE/RL)