№ СТОМ-21 -ИН

Federal State Budgetary Educational Institution of Higher Education

NORTH OSSETIAN STATE MEDICAL ACADEMY

Ministry of Health of the Russian Federation

Department of microbiology

METHODOLOGICAL MATERIALS

by discipline - microbiology, virology, immunology - microbiology of the oral cavity

the main professional educational program of higher education - specialist's programs in the specialty 31.05.03 Dentistry ,
approved on May 24, 2023

Vladikavkaz

LIST OF METHODOLOGICAL MATERIALS:

1. Teaching aids for students in the specialty "Dentistry" (part 1).
2. Teaching aids for students in the specialty "Dentistry" (part 2).
3. Teaching aids for teachers in the specialty "Dentistry" (part 1).
4. Teaching aids for teachers in the specialty "Dentistry" (part 2).
5. Test tasks for students of the Faculty of Dentistry in General Microbiology.
6. Test tasks for students of the Faculty of Dentistry in private microbiology.

No. СТОМ-21 ИН

Federal State Budgetary Educational Institution of Higher Education
"NORTH OSSETIAN STATE MEDICAL ACADEMY"
Ministry of Health of the Russian Federation

DEPARTMENT OF MICROBIOLOGY

COLLECTION METHODOLOGICAL DEVELOPMENT ON MICROBIOLOGY, VIROLOGY, IMMUNOLOGY -ORAL MICROBIOLOGY FOR STUDENTS DENTAL FACULTY

Autumn semester

Vladikavkaz

Authors: Associate Professor of the Department of Microbiology, FSBEI NOSMA of Ministry of Health Russia., Ph.D. Chertkoeva M.G., Assistant Khabieva B.A.

Main appointment developments - methodical help students to to each practical occupation in III semester. Directions drawn up incompliance With Federal public educational standard Supreme and vocational education.

REVIEWERS:
L.V. Bibaeva – MD, Professor, head department biology and histology FSBEI NOSMA of Ministry of Health Russia.

A.R. Kusova- MD, Professor, head department of hygiene and physicaleducation FSBEI NOSMA of Ministry of Health Russia.

PRACTICAL OCCUPATION No. 1.

Topic: Microscopic research method. Equipment and rules of work in bacteriological laboratory. Light microscopy. Immersion system microscope. Morphology microbes. Simple and complex ways coloring drugs. Structure Features eu- and prokaryotic cells.

Educational goal:

1. Familiarization with the rules work *in* microbiologicallaboratories.
2. To study the taxonomy and classification of microorganisms: morphological peculiarities microorganisms and simple coloring.
3. Explore morphology individual representatives bacteria.
4. master technique microscopy.
5. master simple method coloring microorganisms.

Student must know:

1. Device and equipment microbiological laboratories, mode work andappointment.
2. classification bacteria.
3. Structure microscope.
4. ***Methods microscopic research.***
5. Technique microscopic research.
6. Peculiarity buildings bacterial cells.
7. functional meaning various components prokaryoticcells.
8. Complex methods coloring (Gram, Tsilya-Nelsen, Ozheshko, Burri -Ginsa,Neisser).

Student must be able to:

1. cook smear from clean culture, paint simple way.
2. microscoping With immersion system.
3. cook smear With clean culture bacteria E. coli, S. aureus and color accordingmethod Gram.
4. microscoping smear.
5. Be able to cook drugs "hanging" and "crushed" drops.

Plan lessons:

1. Familiarity with the rules of work and the basics of safety inmicrobiological laboratories.
2. Device and equipment microbiological laboratories, mode work andappointment.
3. Classification bacteria.
4. Morphology of bacteria, methods of study (light, dark-field, phase contrast, electron microscopy).
5. Stages cooking smear.
6. Simple method coloring bacteria.
7. Cooking smears from culture staphylococcus and intestinal sticks, coloringsimple method.
8. Demonstration drugs from micrococci, diplococci, tetracoccus, sarcin,staphylococcus, streptococci, intestinal sticks, bacillus, vibrios.

Independent Work students

1. Smear preparation and coloring by a simple method (under the guidance of teacher).

2. Mastering the technique of microscopy. microscopic studymorphology bacteria:
3. View demonstration smear from clean culture staphylococcus (Staphylococcus aureus). Coloring gentian violet.
4. View demonstration smear from clean culture intestinalsticks (E. coli). Coloring - water fuchsin.
5. Decor protocol research.

INFORMATIONAL MATERIL ON THEME

REGULATIONS WORKS AT MICROBIOLOGICAL LABORATORIES.

one . Work in microbiological laboratories held With contagiousmaterial, what requires special discipline and thoroughness in work.

2.Student must be in laboratories in dressing gown and cap. 3.Material forwork taken on duty on group at laboratory assistant

departments and resounds students only With permissions teacher. 4.When implementation of practical classes, students obliged fulfill instructions teacher.

5.All items, which were in work, must to be disinfected. 6.Ifstudent will smash test tube With microbes, he must to report

about this teacher and neutralize working place.

7. Each student receives a microscope, which is assigned to him.8.Wo time fulfillment practical exercises on the table should not to be none strangers items.

9.Students obliged lead in order his working place; pass

duty officer materials and microscope; sign album With sketches. ten. ten. Wash uparms after work.

1. Technique cooking smears.

Smears are prepared on defatted glass slides, having previously outlined with a pencil on the glass, the place of the future smear on the opposite side of the subject glass. When bacteria grow on a liquid nutrient medium, the material is taken sterile bacterial loop, applied to glass and rubbed over the outlined area. AT case growth bacteria on the dense nutritional environment, on the subject glass previously applied with a loop drop water and material rubbed.

2. bacterial loop before using With remaining culture sterilizein the flame of a burner. The prepared smear is dried in air or held high aboveflame spirit lamps.

3. After this a drug fix, for what smear side, where not material, three times carry out through middle flame burners. Fixation allows kill microbes, attach them to the glass and finally, the killed microbes are stained better than alive.

2. Technique simple methods coloring

The staining of bacteria aims to make them sharply different from the background, which allowsexamine their morphology and structure under a microscope. used in microbiology simple and complex staining methods.

At SIMPLE WAY coloring, smear stain any one dye, for example, aqueous fuchsin (2-3 minutes) or methylene blue (2-3 minutes), washed water, dry and microscopic.

3. Technique microscopy

AT connections With very small dimensions bacteria the study them morphology Maybe only at big magnification, achieved at help immersion oils, which allows you to create a single system between the glass slide and special, x 90- multiple (With black stripe) lens.

At microscopy painted objects necessary create bright lighting With help concave mirrors, raised condenser and fully open diaphragm.

A drop of immersion oil is applied to the area of the smear on the glass lying on the table. The glass is then transferred to the microscope stage. The immersion lens is immersed in oil carefully, under the control of the eye until the lens is in direct contact with the oil. Then the lens is raised without removing it from the oil drop and looking into the eyepiece to find the object research ("field of view"). A clear image of the drug is achieved by adjusting first with a macro screw (for detecting an object), and then with a micro screw for adjustment sharpness Images.

Morphology major forms bacteria

known four main forms bacteria:

1. cocci - microbes rounded forms, having in diameter 1-2c. They are different among themselves according to the mutual arrangement of individual cells, which depends on the method them division. If a on graduation division cocci are separated on the individual balloons, are obtained single cells cocci -Micrococcus.
2. Group from two cocci wears title diplococcus -Diplococcus (meningococcus, gonococcus have resemblance With beans, and lanceolate shape - Pneumococcus).
3. If the division of cocci occurs in only one direction and the resulting cocci are not separated, then a thread of balls is obtained in the form of a chain, more or less long in dependencies from number cocci- Streptococcus.
4. When dividing in two mutually perpendicular directions, combinations arise on four coccus-Tetracoccus.
5. If division occurs in three mutually perpendicular directions, cocci connect in form packages (cubes) and get title — Sarcina.
6. Sharing in various directions without special correctness cocci form disorderly clusters cells, reminiscent of grape bunches, why they and got the name Staphylococcus.

rod-shaped microorganisms presented most numerous and diverse group bacteria. AT classification rod-shaped forms received name bacilli and clostridia those sticks, which able formcontroversy, a sticks incapable to spore formation called bacteria. Rod-shaped forms differ in size, location - singly, in pairs, chain, randomly and at an angle. The outline of the ends - rounded, chopped off, thickened, pointed.

Collection forms - *spirilla* and spirochetes having view corkscrew tortuous cells. Pathogenic spirillums include the causative agent of sodoku (rat bite disease). To tortuous also relate campylobacter, having curves how at wings flying seagulls.

Spirochetes — thin, long, curved (spiral forms) bacteria, differing from spirilla in mobility due to flexion changes cells. Spirochetes are represented by three genera pathogenic for humans: Treponema, Borrelia, Leptospira.

Methods diagnostics infectious diseases

1. *The microscopic method* consists in the preparation of preparations (native or painted simple or complex methods) from researched material and them microscopy With application various species microscopic technology (light, darkfield, phase contrast, electronic). AT bacteriology microscopic method received title bacterioscopic, in virology — viroscopy.
2. *cultural method* is in sowing researched material on the artificial nutrient media for the purpose of isolating and identifying a pure culture pathogens. AT bacteriology cultural method received title bacteriological, a in virology - virological.
3. *The biological method* (experimental or bioassay) is to infect test material of sensitive or other biological objects (chicken embryos, culture cells). His use for allocation clean culture pathogen, definitions type toxin, definition activity antimicrobial chemotherapy drugs.
4. *Serological method* - consists in determining the titer of antibodies in serum the patient's blood, less often - in the detection of microbial antigen in the test material. FROMthis reactions are used for the purpose immunity.
5. *The allergic method* consists in identifying an infectious allergy (HRT) to diagnostic microbial a drug — allergen. FROM this purpose put skin allergic samples from relevant allergens.

An object study medical microbiological laboratories — pathogenic biological agents are

microorganisms pathogenic for humans (viruses, bacteria, mushrooms, protozoa). AT compliance With types microorganisms allocate: bacteriological, virological, mycological, protozoological laboratories. Regulation conditions work With pathogens infectious diseases is produced in accordance with the degree of danger of microorganisms for person. Allocate four groups pathogens.

Group 1: pathogens of especially dangerous infections: plague, smallpox, fevers Lassa, Ebola.

Group 2: exciters highly contagious bacterial fungal and viral infections: anthrax, cholera, fever, typhus, rabies.

Group 3: pathogens bacterial fungal, viral and protozoan nosological forms (whooping cough, malaria, polio, leishmaniasis).

Group four: pathogens bacterial viral, fungal diseases (pseudomonal infection, amoebiasis, aspergillosis).

Microbiological laboratories work with PBA with highly dangerous pathogens infections (group 1 and 2). The special mode is maximally isolated by the individual and public risk.

Technique complex staining methods

Complex ways coloring include sequential application on the a drug dyes, different on chemical composition and color, mordant and differentiating agents. This allows pre-differentiation of microbes *(differential diagnostic methods)* and identify certain cell structures *(special ways).*

1. Way coloring on Gramu

Coloring on Gramu is important diagnostic sign identification bacteria. As a result of coloring Gram all bacteria are divided into two groups; gram- positive (blue colors) and gram negative (red colors).

Technique coloring on method Grama

1.A fixed smear is placed on a bacteriological bridge and covered with a strip filter paper impregnated with a solution of gentian violet on a paper strip inflict water. After 2 minutes, a strip are removed.

2. Not flushing a drug water, inflict solution Lugol on the one minute. Then solution drained.

3. A drug discolor alcohol 20-30 seconds (before discharge purple tricklepaints).

4. A drug washed water.

5. coloring water magenta — 2 minutes.

6. A drug washed water.

7. Dry up on the air or filtering paper.

2. Way coloring on Tsil-Nelsen

Applies for detection some germs, rich lipids (for example,pathogen tuberculosis, leprosy, etc.)

1. For staining, a concentrated solution of Ziel's carbolic fuchsin is used. In order to improve the penetration of the dye into the cell, the preparation with the stripe filtering paper and dye warm up above flame burners three times before steam appears.

2. Then the preparation is decolorized with a 5% solution of sulfuric acid, after removing filtering paper.

3. washed water.

4. Finishing up methylene blue in flow 3-5 minutes.

5. A drug washed water.

6. Dry up on the air or filtering paper.

Bleaching acid leads to loss dye acid-stable microbes, and they turn blue. Acid-fast microbes remain red.

3.Way coloring on Burri Guinsu

1. Mix a drop of culture of capsular bacteria with a drop of carcass at the end of the subjectglass. Then cook smear as usual his cook from a drop blood.

2. Smear dry up on the air and fix in flame burners.
3. For detection bacteria smear stain water fuchsin.

At this way coloring bacteria stained in red color, a unpaintedcapsules in contrast stand out how bezel on the black-brown background around bacteria.

4.Way coloring on method Neisser

1. On the fixed smear inflict bluing Neisser2-3 min.
2. Not flushing water, inflict solution Lugol 10-30sec.
3. Smear washed water.
4. Finishing up solution vesuvina — one min.

In the culture of yeast-like fungi, there are many grains of volutin. They represent connections, having, in difference from cytoplasm, alkaline reaction and because are dyed dark blue. The cytoplasm of the cell is acidic perceives alkaline dye vesuvine and stained in yellow color.

5.Way coloring on method Ozheshko

1. On the unfixed smear inflict 0.5% solution HCI and warm up on the flameburners in flow 2 minutes. Before appearance vapors.
2. A drug washed water, dry up and fix.
3. Finishing up on method Tsilya-Nelsen.

controversy bacteria after given coloring acquire red color, a body bacteria-blue.

TEST TASKS FOR CHECKS KNOWLEDGE

Specify one right answer:

1. Which scientist is associated with the discovery of the essence of fermentation (1857), microbial conditionality and contagiousness of infectious diseases (1881), manufacturing methods vaccines and methods for the prevention of chicken cholera, anthrax and rabies (1882-1885)?

a) Leeuwenhoek
b) Mechnikov c)
KochG) Pasteur

2. Microbe — this is:

a) a pre-cellular living being; b)
organism certain type;
c) a single-celled creature, invisible to the naked eye;G) infectious
protein particle;
e) unicellular organism.

3. Bacterium — this is:

a) virus;
b) unicellular creature certain kind, pertaining to to prokaryotes;c) a single-celled
creature of a certain species, belonging to eukaryotes; G) organism a certain type;
e) unicellular organism.

4. bacilli — this is:

a) cocci, generators disputes;
b) rods that do not form spores;c)
rods that form spores; G) tortuous
forms.

5. Alive maybe reckon system, capable to:

a) exchange substances With environmental environment;
b) maintaining its structural organization; in)
multiplication their structural components;
d) reproduction and implementation of the genetic program;e)
metabolism.

6. If a conditionally to choose three major functional and structural componentbacteria,

then it will be:
a) nucleus, cytoplasm, shell;
b) DNA, cytoplasmic membrane, inclusions;
c) cell wall, cytoplasmic membrane, nucleus;G) shell,
cytoplasm, DNA;
e) ribosome, cytoplasm, nucleus.
7. AT difference from eukaryotic cells bacteria have:
a) haploid set of chromosomes; b)
diploid set of chromosomes; in)
cell center;
G) histone proteins.
8. Structurally cytoplasmic membrane bacteria is different from membranes othersalive creatures in that:
a) is three-layer;
b) in its composition includes cholesterol;
c) is able to form the endoplasmic reticulum;G) capable form mesosome;
e) capable form spindle division.
9. Rigidity structures bacterial cells provided:a) capsule;
b) cellular wall;
c) cytoplasmic membrane;G) flagella;
d) saws.
10. The form bacteria determined structure her:
a) pili;
b) cytoplasmic membrane;in) cell
wall;
d) all three components;e)
unknown science.

PRACTICAL EXERCISE No. 2.

Topic: Bacteriological method research. Physiology bacteria. Nutrients environment. Them classification, ways cooking, sterilization. Technique crops material on nutritious environment.

Educational goal:

1. To master the bacteriological method of diagnosing infectious diseases. 2. Explore types nutrition bacteria, principles cultivation of microorganisms classification nutritional avg.
3. Explore methodology receiving pure cultures bacteria from researched material.
4. Explore methods sterilization (physical, mechanical, chemical).
5. Explore methods control efficiency sterilization.

Student must know:

1. Methods sterilization
2. The mechanism of action of sterilizing factors on the molecular structure microorganisms.
3. Concepts contamination and decontamination, disinfection and sterilization, asepsis and antiseptics.
4. Modern sterilization technologies and equipment. 5. Ways to control the effectiveness of sterilization and disinfection6.Control quality sterilization.
7. Principles cultivation microorganisms.

8. Bacteriological method diagnostics infectious diseases.
9. Nutrients environment, requirements, presented to nutritious Wednesdays
10. Classification nutritional environments, compound and cooking.

Student must be able to:

1. Estimate efficiency sterilization and disinfection.
2. Run the first stage allocation clean culture aerobic bacteria.

Plan lessons:

1. Nutrition of bacteria: types, mechanisms of nutrient entry into microbial cell.
2. Principles cultivation microorganisms.
3. Bacteriological method diagnostics infectious diseases.
4. Nutrient media: requirements for nutrient media ;classification, compound, cooking.
5. Demonstration nutritional avg.
6. Sowing researched material (suspended microorganisms) on the MPA method Drygalsky (Stage 1).
7. Methods sterilization: physical, chemical, biological, mechanical.
8. Device and application ovens Pasteur autoclave, apparatus Koch.
9. Sterilization various medicinal funds in dependencies from them nature,forms, lability to physical factor.
10. Control quality sterilization.
11. concept about asepsis, antiseptic and disinfection.
12. Antiseptics and disinfectants.
13. Principles control quality disinfection.
14. Demonstration antiseptic and disinfectants funds.

Independent Work students:

1. Sowing test material on method Drygalsky.
2. Familiarization With cooking nutritional avg.

3. Spend and take into account results experience on definition actions high temperature (80°C) into spore-forming (anthracoid) and asporogenic (intestinal wand and staphylococcus) microorganisms.
    - Fill protocol on form:

Accounting growth culture	Staphylococcus aureus	intestinal wand	Anthracoid
before warming up			
after warming up			

Vegetative forms of pathogenic microorganisms die at 50-60 ^{0} C during 30 minutes, and at a temperature of 70 ^{0} C for 5-10 minutes. Bacterial spores have greater resistance to high temperatures, due to the content in them water in related state, big content salts calcium, lipids and density, layering shells. Consequently, staphylococcus aureus and intestinal wand after warming up are dying a disputes anthracoid survive. it and necessary take account of in evaluation seeding results.

- Fill on one's own table:

No.	Way sterilization	Apparatus	Reliability	sterilizable material
one.	Sterilization in flame			
2.	Plasma Sterilization			
		four.	Ferry under pressure	
		5.	Fluid ferry	
		6.	Tyndalization	
		7.	Filtration	
		eight.	Physica l factors (UFL, gamma rays, ultrasound)	
		9.	Gas sterilization	
		ten.	Pasteurization	
3.	Dry heat			

INFORMATIONAL MATERIL ON THEME

Microbiological research is carried out in order to isolate pure cultures microorganisms, cultivation and study them properties. It necessary at diagnostics infectious diseases, for definitions specific accessories germs, in research work, for receiving products vital activity microbes (toxins, antibiotics, vaccines, etc.). To grow microorganisms in artificial conditions necessary special substrates — nutritious environment. They are are basis microbiological work and determine results Total research. environments must create optimal terms for vital activity microbes.

REQUIREMENTS PRESENT To WEDNESDAY:

1. Must be nutritious, i.e. contain in an easily digestible form all the substances necessary for satisfaction food and energy needs microorganisms.
2. Have optimal concentration hydrogen ions.
3. To be isotonic for microbial cells.
4. To be sterile.
5. To be wet.
6. Have certain redox potential.
7. To be on capabilities unified.

Need in nutritional substances and properties environments at different species microorganisms is not the same. it rules out possibility creation universal environment. Except Togo, on the choice toy or other environment affect goals research.

Group classification	Class	Examples
Composition	***Simple***	*Liquid — MPB, peptone water Dense — MPA*
	Complex	*Liquid — sugar bouillon Dense — sugar agar, blood agar*
Origin	***natural***	*Milk, folded serum, slice raw potatoes*
	artificial	*Milk Salt Agar Whey agar Ascites agar Blood agar*
	Synthetic	*Wednesday Needle, Wednesday 199*
By appointment	***selective (elective)***	*Milky salt agar, zhel-exactly-*

	-for staphylococcus: *-for gram(-) cocci anddiphtheroids:* *-for enterobacteria:* *-for cholera vibrio:* *-for lactobacilli and mushrooms* ***Differential-diagnostic Universal enrichment media Preservative***	*salt agar Serum media Media with tellurium salts Media with bile salts Peptone broth and alkaline agar Tomato agar, rice agar, agar Saburo* *endo, Ploskireva, Levin, Ressel,gissa MPB, MPA, blood agar Wednesday Muller environments With glycerin*
By consistency	***Liquid*** ***semi-liquid*** ***Dense***	*MPB, peptone water, sugarBCH MPJele, gelatinousMPA, blood agar*

STERILIZATION

Sterilization is deposition, t. e. complete release objects environmentalenvironments from microorganisms and their dispute.

Sterilization produce various ways:

1. **Physical** (exposure to high temperature, UV rays, increased pressure, steam, gamma rays, ultrasound).
2. **Chemical** (usage various disinfectants, antiseptics).
3. **biological** (application antibiotics).
4. **Mechanical** (filtration).

AT laboratory practice usually apply physical ways sterilization.

The possibility and feasibility of using one or another method of sterilization due to the characteristics of the material to be sterilized, its physical and chemical properties.

To *physical* ways sterilization can attributed calcination in flame, dry heat sterilization in a Pasteur oven, boiling, fluid steam sterilization in apparatus Koch, ferry under pressure in autoclave, tindalization, pasteurization sterilization UFL, ultrasound.

Mechanical sterilization carried out filtering With help bacterial filters made from various finely porous materials, pores filters should be fine enough to provide mechanical delay bacteria. This method sterilizes nutrient media containing protein, serum, antibiotics; separate bacteria from viruses phages, exotoxins.

AT microbiological practice use asbestos filters Seitz, membrane Chamberlain filters (candles) and Berkefeld.

a) *Seitz filters* — discs from mixtures asbestos With cellulose, thickness them 3-5mm, diameter 35-140mm;

b) *membrane* filters - made of nitrocellulose, 0.1 mm thick and 35 mm in diameter. AT dependencies from size pores are 1,2,3,4,5;

c) *Chamberlain and Berkefeld candles* - hollow cylinders closed at one end, cook them from kaolin With sand admixture and quartz.

Chemical ways sterilization apply limited but they serve for prevention of bacterial contamination of nutrient media and immunobiological drugs (vaccines and sera).

biological sterilization founded on the application antibiotics, sometimes phages.

Disinfection — usage chemical substances (phenol, lysol, chloramine, peroxide hydrogen, corrosive sublimate, alcohol, and t. e.) for destruction pathogenic bacteria in spent pathological material.

Systematization appliances, processes processing and funds for disinfection andsterilization

Classification tools	**Main typestools**	**The nature of processing and typesimpacts**
critical - penetrate in sterilefabrics or vessels	All invasive surgical tools that havecontact with blood-supplied fabrics, scalpels, needlessyringes, implants,burs, root needles,excavators, probes, trowels.	**Sterilization** - virucidal, sporicidal, tuberculocidal,bactericidal impact. **long exposure:** gamma- rays, plasma, continuous gas and chemical sterilization, autoclaving (2 atm. 15 min), dry heat (max. mode, 2 hours)
Semi -critical - come into contact with mucous shells (perexcept for som edental tools, listed above)	flexible endoscopes, catheters, toolssimilar to flexible endoscopes, mirrors, crowns, tips turbines, a also prints(casts) teeth.	**Disinfection high level** -virucidal, sporicidal, tuberculocidal, bactericidal impact. **Short exposure:**gamma rays, plasma, short-term gas and chemical sterilization, autoclaving (1-1.5 atm. fifteen min), dry heat.
	Thermometers for measuring mucosal temperature shells, baths for hydrotherapy.	**Medium level disinfection:** virucidal, tuberculocidal,bactericidal impact.
	Ultrasonic baths and UV lampsdentists physiotherapy tools , spoons casts.	**Means for chemical disinfection with** indication of labeling tuberculocidalactivity.
Non-critical come into contact withintact skin	Thermometers for measuring skin temperature covers, stethoscopes, cuff devices for measurements pressure, desktop appliances and etc.	**Disinfection level:** bactericidal effects. **Funds for chemical disinfection** without instructions on the presence marking tuberculocidal activity.

TEST TASKS

Specify correct answers:

1. What such sterilization?

a) complete decontamination of objects from all types of microbes and their sporesb) destruction of pathogenic microorganisms

c) destruction of vegetative forms of microorganisms d) prevention hits microorganisms in wound

e) destruction on the objects concrete species microbes

2. What factors are used in autoclaving?a) temperature

b) filters

c) steam

d) pressure

3. What kind factors are used in ovens Pasteur?a) pressure

b) steam

c) dry heat

d) antibiotics

4. To physical methods sterilization relate:

a) ultrasound

b) ultraviolet raysc) antibiotics

d) filtering

e) steam sterilization

e) dry heat sterilization

5. List ways sterilization, liberating an object from spore formsmicrobes:

a) exposure to ultraviolet light

b) autoclaving

c) pasteurization

d) dry heat

e) gamma irradiation

6. What culture media are simple?a) Wednesday Endo;

b) MPA;

c) blood agar;G) MPB;

e) peptonic water.

7. The density of nutrient media depends on the content in them:

a) chloride sodium;

b) peptone;

c) agar-agar;

G) sucrose;

e) serum blood.

8. At the 1st stage of the bacteriological research method, the following tasks are solved:a) pure culture identification microbes;

b) definition sensitivity to antibiotics;in) receiving isolated colonies;

d) determination of the type of microbe; e) receiving clean culture.

9.Preferential growth some species microbes at simultaneous suppression otherscan get on next types nutritional Wednesdays:
a) selective (elective);b)
simple;
in) complex;
G) preservative;
e) differential diagnostic;e) universal.
10. AT concept "cultural properties" microbe includes:
a) character growth on the nutritional environments;
b) macroscopic characteristic colonies;
c) morphology of microbial cells under microscopy;G)
fermentation carbohydrates on the environments Hiss;
e) color pigment colonies or culture;
e) attitude pathogen to coloration on Gram.

PRACTICAL OCCUPATION No. 3.

Topic: Ways allocation and identification pure cultures aerobic bacteria. The study enzymatic activity, factors virulence and sensitivity to antibiotics dedicated cultures. Peculiarities transportation material and allocation pure cultures anaerobic bacteria.Cultural and pathogenic properties mushrooms.

test control.

Educational goal:

1. master methods allocation pure cultures aerobes.
2. To study the types of bacterial respiration, ways to create conditions for anaerobiosis. Mastermethods isolation of pure cultures of anaerobes.
3. Explore mushrooms - pathogens mycoses and mycological method research

Student must know:

1. Principles cultivation microorganisms. 2. Bacteriological method for diagnosing infectious diseases.3.Nutritious environments for cultivation anaerobes
four. Stages and factors symbiosis human With microbes.

Student must be able to:

1. Run second stage allocation clean culture aerobic bacteria.

Plan lessons:

1. Types breathing bacteria.
2. Ways creation conditions anaerobiosis.
3. Methods allocation clean culture aerobes and anaerobes.
4. Sowing soil talkers on the Wednesday Kitta Tarozzi.
5. Nutrients environments for anaerobes, methods cultivation and selection cleanculture anaerobes.
6. The study cultural properties bacteria.
7. The study colonies, grown on the cups, sown Drygalsky's method .
8. Sowing microorganisms from studied colonies on the oblique agar for receivingclean culture (stage 2).

9. Demonstration pigment formation bacteria.
10. Demonstration character growth bacteria on the dense and liquid nutritional environments.
11. Change module.

Independent Work students

1. Completion 1st stage bacteriological method. The study culturalproperties bacteria.
2. From grown colonies on the MPA cook smear, paint on Gram.
3. Inoculation from the studied isolated colonies on agar slant for accumulation clean culture.
4. Demonstration technology anaerobic cultivation and Wednesdays for anaerobes: tall column of agar, Kitt-Tarozzi medium, thioglycol, Stuart. Demonstration microaerostat. Methods: Fortner, Weinberg.
5. Cooking smears from yeast mushrooms, color them with a simple method (methylene blue) and microscoping.
6. Preparation and microscopy native preparations from cultures moldy mushrooms.
7. Viewing and sketching demo preparations: a) actinomycetes, painted on Gram;
b) native preparations from cultures of mold fungi (mucor, aspergillus, penicillium);
in) yeast mushrooms, painted methylene blue;
8. Sowing material With fingers hands on the cup With MPA (method prints).
9. Sowing detachable from nose and pharynx on the MPA.

INFORMATIONAL MATERIL ON THEME

Breath bacteria. Classification bacteria on type breathing.

Essence breathing at microorganisms — receiving energy, emerging in process direct biological oxidation substances oxygen or through substrate dehydrogenation. The accumulation of energy occurs in special structures bacteria, called mesosomes.

Bacteria are classified according to their oxygen requirements. main groups:

1. Obligate (strict) aerobes - microorganisms that grow and reproduce only in presence oxygen. For example: Vibrio cholerae Pseudomonas aeriqinoza.

2. Obligate anaerobes are microorganisms that grow and reproduce only without access oxygen. For example: Clostridum botulinum, Clostridium te tani.

3. Facultative anaerobes are microorganisms that can grow and multiply both in the presence of oxygen and in anoxic conditions. For example: Escherichia coli, Salmonella typhi.

4. microaerophiles bacteria — microorganisms, which better are growing and thrive in high $CO2_{and}$ low oxygen levels. For example: Helicobacter pylori, Campylobacter coli.

Methods cultivation anaerobes

Ways creation anaerobic conditions a) mechanical - removal (pumping out)air from the anaerostat using vacuum suction. Then the anaerostat is filled gas mixture which consists from 80% nitrogen, ten% hydrogen and ten% carbon dioxide gas;

b) chemical — absorption oxygen per check chemical substances (alkalinesolution pyrogallol, bicarbonate soda);

in) biological (method Fortner) — a joint cultivation anaerobes andaerobes. At this on the one cup petri With dense nutritional environment (more often use Wednesday Zeissler) sow culture anaerobes, on the another — culture aerobes, able vigorously absorb oxygen. AT quality aerobes use culture miraculous sticks (Serratia marcescens). The edges cups Petri is waxed;

d) physical and chemical - inoculation of the test material on special media for anaerobes, for example, Kitt-Tarozzi and Wilson-Blair media (iron sulfite agar). The media are regenerated before inoculation (boil in a water bath for 15 minutes) to removal oxygen.

Compound environments Kitta-Tarozzi:

-pieces liver — for adsorption oxygen;

-one% glucose — for implementation anaerobic glycolysis;

- semi-liquid agar — not admits oxygen in thickness environment.

Receipt clean culture anaerobes

1. Method Weinberg (method dilution)

To obtain isolated colonies of anaerobes from the Kitt-Tarozzi medium with growth

anaerobic bacteria culture is taken with a Pasteur pipette with a sealed end and successively lowered this pipette at first in 3 test tubes With physiological solution, and then - 3 test tubes with melted semi-liquid sugar MPA. After temperature control at 37 ^{0} C in recent observed growth isolated colonies anaerobes.

2. Method Peretz.

One of the last Weinberg dilutions in semi-liquid agar is poured into the lid cups petri and close her bottom So, to delete air. The edges cups petri paraffin. Sowing researched material on the Wednesday Zeissler sectors With subsequent cultivation in anaerostat.

Mushrooms (Fungi, Mycetes) are a heterogeneous group of eukaryotic microorganisms. Mushrooms have nucleus With nuclear shell, cytoplasm With organelles cyto- plasmatic membrane (which contains phospholipids and sterols) and powerful cellular wall consisting from glucan, cellulose, chitin, squirrel, lipids and others Mushrooms consist of long thin filaments (hyphae) woven into a mycelium, or mycelium. Hyphae of lower fungi - phycomycetes - do not have partitions. In higher mushrooms - eumycetes — hyphae divided partitions; them mycelium multicellular. Mushrooms reproduce by spores, sexually, asexually, and vegetatively (budding or fragmentation gif). Mushrooms, breeding sexual and asexual by, relate to perfect. imperfect called mushrooms, at which missing or more not described sexual path breeding. asexual reproduction carried out at mushrooms With help endogenous dispute, maturing inside round structures - sporangia, and exogenous spores - conidia, formed at the tips fruitful hyphae.

Mushrooms can divide on the 7 classes: chytridiomycetes, hyphochytridiomycetes, oomycetes, zygomycetes, ascomycetes, basidiomycetes, deuteromycetes. overwhelmingmajority mushrooms, defiant diseases at human (mycoses), relate toimperfect mushrooms. For diagnostics mycoses may to be used microscopic (cultural), allergic, serological, biological and histological research methods. Material for research can be pus, sputum, affected hair, nails, scales skin, punctates bone brain, lymphatic nodes, internal organs, blood, bile, feces, tissue biopsyand t. P. For coloring smears more often Total use methods Grama, Ziel-Nielsen, Romanovsky-Giemsa

TEST TASKS FOR CHECKS KNOWLEDGE

Specify all correct answers:

1. For cultivation anaerobes without anaerostat used Wednesday:a) blood agar;
b) yolk-salt agar;in) Endo;
d) Kitta-Tarozzi;e) Clauberg.
2. With anaerobic respiration, bacteria lack a group of enzymes:a) dehydrogenases;
b) flavoproteins; c) cytochrome oxidases;
G) decitinases;
e) nucleases.
3. The final electron acceptor in aerobic respiration in bacteria is:a) molecular oxygen;
b) inorganic compounds;in) organic connections;
d) both organic and inorganic compounds;e) mitochondrial proteins.

4.Wednesday thioglycolic serves for highlights:
a) obligate aerobes; b) obligate anaerobes;
in) optional aerobes; G) optional anaerobes;

5.Wednesday Kitta-Tarozzi serves for highlights:
a) obligate aerobes; b) obligate anaerobes;
in) optional aerobes; G) optional anaerobes;

PRACTICAL OCCUPATION No. four.

Topic: Bacteriophage. Genetics bacteria. Molecular genetic method diagnostics. Structure and reproduction bacteriophages. Them medical meaning. Heredity and variability in bacteria. The polymerase chain reaction and its application . .

Educational goal:

1. Explore structure and morphology bacteriophages.
2. Explore heredity and variability at bacteria.

Student must know:

one. morphology, ultrastructure classification bacteriophages.

Student must be able to:

1. Find viral inclusion method light microscopy.
2. Find viral inclusion method luminescent microscopy.

Plan lessons:

1. Morphology and structure of bacteriophages, their practical application in medicine.
2. Kinds heredity bacteria.
3. Independent Work students:

- The study demonstrations phenomenon bacteriophage on the dense and liquid nutritional environments.
- The study demonstrations intracellular inclusions (body Babesha-Negri).

INFORMATIONAL MATERIL ON THEME

bacteriophages differ on chemical structure, type nucleic acid, morphology and nature of interaction with bacteria. Size of bacterial viruses in hundreds and a thousand times less microbial cells.

A typical phage particle (virion) consists of a head and a tail. Tail length is usually2-4 times the diameter of the head. The head contains genetic material - single stranded or double stranded RNA or DNA With enzyme transcriptase in inactive state, surrounded protein or lipoprotein shell - ***capsid*** , preserving genome outside cells.

Nucleic acid and capsid together make up the nucleocapsid. Bacteriophages can have icosahedral capsid, assembled from sets copies one or two specific proteins. Usually angles are made up of pentamers squirrel, and support each side from hexamers Togo same or similar squirrel. More Togo, phages on form may to be spherical, lemon-shaped or pleomorphic. Tail represents yourself protein tube - a continuation of the protein shell of the head, at the base of the tail there is ATPase, which regenerates energy for the injection of genetic material. There are alsobacteriophages short offshoot, not having offshoot and threadlike.

Phages, like all viruses, are absolute intracellular parasites. Although they endure all information for launch own reproductions in relevant host, at them missing mechanisms for workings energy and ribosomes for protein synthesis. Some phages have a genome containing several thousand bases, while the G phage, the largest phage sequenced, contains 480,000 steam grounds - twice more middle values for bacteria, although all same insufficient quantity genes for the most important bacterial organoid how ribosomes.

big amount dedicated and studied bacteriophages defines need them systematization. Classification viruses bacteria has undergonechanges: based on the characterization host virus, taken into account serological, morphological properties, a then structure and physical and chemical compound virion .

Currently, according to the International Classification and Nomenclature of Viruses bacteriophages, depending on the type of nucleic acid, are divided into DNA- and RNA-containing.

By morphological characteristics DNA containing phages highlighted in the following families: myoviridae, Siphoviridae, Podoviridae, Lipothrixviridae, Plasmaviridae, Corticoviridae, Fuselloviridae, Tectiviridae, Microviridae, Inoviridae Plectovirus and Inoviridae Inovirus.

RNA containing: cystoviridae, Leviviridae

By character interactions bacteriophage With bacterial cell distinguish virulent and moderate phages. Virulent phages may only increase in quantity through lytic cycle. Process interactions virulent bacteriophage with a cell consists of several stages: adsorption of the bacteriophage on cage, penetration in cell, biosynthesis components phage and them assembly, exit bacteriophages from the cell.

Initially bacteriophages attached to phage-specific receptors on the surfaces bacterial cells. Tail phage With help enzymes located on the its end (mainly lysozyme), locally dissolves the cell membrane, contracts and contained in head DNA injected in cell, at this protein shell bacteriophage remains outside. injected DNA causes complete perestroika metabolism cells: stops synthesis bacterial DNA, RNA and proteins. DNA bacteriophage starts be transcribed With help own enzyme transcriptase, which the after hits in bacterial cage is activated. First, early and then late mRNAs are synthesized, which enter the ribosomes. host cells, where early (DNA polymerases, nucleases) and late (proteins) are synthesized capsid and tail process, enzymes lysozyme, ATPase and transcriptase) squirrels bacteriophage. replication DNA bacteriophage going on on semi-conservative mechanism and carried out With participation own DNA polymerases. After synthesis late proteins and the completion of DNA replication, the final process begins - maturation phage particles or compound phage DNA With protein shells and education mature infectious phage particles.

Duration this process maybe make up from several minutes before several hours. Then going on lysis cells, and released new mature bacteriophages. Sometimes the phage initiates a lysis cycle, which leads to cell lysis and release new phages. AT quality alternatives phage maybe initiate lysogenic cycle, at which he instead of replication reversible interacts With the genetic system of the host cell, integrating into the chromosome or remaining in the form plasmids. Thus, the viral genome replicates synchronously with host DNA and cell division, and a similar state of the phage is called a prophage. bacteria containing prophage, becomes lysogenic before those since, bye at certain conditions or spontaneously prophage not will be stimulated on the implementation lysing cycle replication. Transition from lysogeny to lysis called lysogenic by induction or prophage induction. Phage induction is strongly influenced by the state of the cell host prior to induction, as well as nutrient availability and other conditions at the moment of induction. Poor growing conditions favor lysogenic way, then how good conditions encourage lysing reactions.

A very important property of bacteriophages is their specificity: bacteriophages lyse cultures of a certain type, moreover, there are so-called typicalbacteriophages, lysing options inside kind, although meet polyvalent bacteriophages, which parasitize in bacteria different types.

Viruses highlighted in separate "kingdom" - Viga. They are contain only one type of nucleic acid, not have cellular structures, not have independent exchange substances being intracellular parasites reproduction viruses carried out disunited way.

According to the international classification, all viruses are subdivided according to the type of nucleic acids on the 2 subtype - RNA- and containing DNA. Further separation viruses is carried out on the basis of the size of the viruses, the type of symmetry in the formation of capsids,availability or absence outer shells and quantity contained in them capsomeres.

TEST TASKS

1.What are bacteriophages?
   a) bacteria;
   b) viruses;
   in) phagocyte cells;
   G) mushrooms.
2.What kind microbes not have cellular buildings?a)
   viruses;
   b) mycoplasmas;
   in) chlamydia;
   G) mushrooms.
3.What contains a complexly organized virus?a)
   two type nucleic acid;

b) one type of nucleic acid (either DNA or RNA);in)
supercapsid;
G) capsid
4. Viruses opened:
a) L.
Pasteur;b) R.
Koch;
c) I. Ivanovsky;G)
AND. Mechnikov.
5. Extracellular form of existence of virusesa)
virion;
b) capsid;
in)
capsomere;
G) supercapsid;
e) elementary bodies.

PRACTICAL OCCUPATION No. 5.

Topic: Modern methods diagnostics in virology. concept about buildingviruses, viroids and prions. Methods diagnostics.

Educational goal:

1. Explore morphology and ultrastructure viruses.
2. Explore methods diagnostics viruses.

Student must know:

one. morphology, ultrastructure classification viruses.

Student must be able to:

1. Find viral inclusion method light microscopy.
2. Find viral inclusion method luminescent microscopy.

Plan lessons:

1. Peculiarities biology viruses **.**
2. Principles classification viruses.
3. Types interactions viruses With cell.

Independent Work students:

- The study demonstrations phenomenon bacteriophage on the dense and liquid nutritional environments.
- The study demonstrations intracellular inclusions (body Babesha-Negri).

INFORMATIONAL MATERIL ON THEME

VIRUSES

Viruses have properties that make it impossible to use conventional methods to study them. methods microbiological research.

Distinctive properties viruses:

1. smallest sizes, measurable thousandths shares micron - millimicrons
- from 8-10 m up to 300-400 m.
2. Filterability through special finely porous filters, not passingother microorganisms.
3. non-cellular structure.
4. Absolute parasitism, those. ability live and multiply only in alivecells.

The form viral particles It has several types:

1. rod-shaped
2. spherical (spherical)
3. Cuboid
4. Capitate (spermatozoa)
5. filiform

mature viral particle, called *virions*, have next scheme buildings: in central parts located molecule DNA or RNA, which forms *nucleoid.* Around situated protective protein shell, called *capsid,* built from morphological units, called *capsomeres.* Some complex virions have external shell, called *supercapsid.*

For microbiological diagnostics viral infections in the present time apply three main methodical approach:

1. **Virological diagnostics** - founded on the allocation from researched material virus and his subsequent identification.
2. **Serological diagnostics** - definition specific immunological changes in the body under the influence of viruses (most often with help diagnosticums reveal in serum blood antiviral antibodies).
3. **Molecular biological diagnostics** - detection in clinical the material of nucleic acid fragments of pathogenic viruses using probes (hybridization NK) or PCR.

Individual viruses larger than 200 m can be stained according to Romanovsky - Giemsa; smaller viruses (variola viruses) can only be detected using special processing methods.

VIROLOGICAL RESEARCH METHOD is the main and most reliable, allows you to isolate the virus from the test material with its subsequent identification. FROM purpose accumulation virus-containing material are used chicken embryos and cultures fabrics (artificially cultured cells one or other tissue). Tissue cultures are maintained on vivo (medium 27, Enders) and synthetic (Wednesday 199, Needle, Melnik-Riordan) nutritional environments prepared on the basis of solutions of Hanks and Earl. They are cultivated in the usual test tubes cups carrel, test tubes Barsky.

Methodology infections chicken embryo

There are several ways to infect a chicken embryo. Most often the material injected into the allantoic and amniotic cavities, onto the chorionallantoic membrane and into yolk bag. Before infection shell eggs above air camera treated with 70% alcohol, burned on a flame, smeared with 2% iodine tincture, secondarily wipe with alcohol and burn.

When infected in the allantoic cavity in the shell above the air chamber (borders which is outlined in advance with a pencil when the eggs are translucent in an ovoscope) are done small hole With help scissors or scalpel. Tuberculinov syringe introduce 0.1-0.2 ml virus-containing material on the depth 2-3 mm below borders air chamber. A puncture in the shell is filled with molten paraffin. Opening infected embryos produce in terms maximum accumulation virus (through 48-72 h incubation at temperature 37 FROM) after processing shells alcohol and 2% solution iodine her dissect and dump, filmed carefully shelledshell and consider chorionallantoic wrap around places infections on the Availability foci lesions (hemorrhages, whitish foci defeats).

Classification cellular crops:

• **primary** receive directly from fabrics animal and human through destruction proteolytic enzymes (trypsin, collagenase) intercellular substances. Disunited cells, placed in nutritional Wednesday, able attach to the surface of the culture vessel and multiply, forming a monolayer - layer one cell thick. With the help of special reagents, cells can be removed from surfaces one vessel and transplant in another. Such manipulation called **passage.** primary crops withstand not more 5-10 passages.

• **transplantable** (passage) cellular culture able withstand unlimited number of passages. They originate from tumor cells that have lostdifferentiation and not having restrictions growth.

• **semi-transplantable** (diploid) culture - fibroblast-like cells, which able to fast reproduction, withstand before 30-60 passages and save original set of chromosomes.

Viruses can reproduce only in the cells of a living organism. Concerning viruses cultivated through infections chicken embryos or cultures fabrics, a also suckling animals.

Detection (indication) of viruses *Virus*
detection in chick embryo1. Death
2. The appearance of an odor upon
opening 3. Cloudiness liquids in
cavities
four. Education sores and hemorrhages on the shells

Biological method research is in contagion sensitive to virus animal researched material, studying clinical and pathoanatomical paintings diseases. AT framework this method are used various animals: monkey, rabbits, maritime pigs, dogs, mice, rats. Ways infections: subdural, intracerebral, intranasal and other.

Methods for detecting the virus in the body of laboratory animals differ in dependencies from the view animal and type virus.

Detection viruses in culture cells

Revealing on cytopathic action (CPD). JPC represents yourself degenerative changes in cells that result from reproduction in them viruses.

Distinguish complete and partial degeneration cells monolayer.

With complete degeneration caused, for example, by polio viruses, Coxsackie and ECHO, cells of the monolayer undergo significant changes, more of themslough off co glass. Remaining single the cells are wrinkled

Partial degeneration has several varieties: one .Type cluster
formation (adenoviruses);
2 .By type focal destruction (smallpox, flu);
3.According to type symplast formation (measles, mumps, parainfluenza, herpes, HIV).

Proliferative type of changes typical for some oncogenic viruses, transforming cells in malignant.

Intracellular inclusion formed at reproductions some viruses in cytoplasm and core cells (smallpox, rabies, flu, herpes and etc.) Them discover at microscopy after coloring monolayer on Romanovsky - Giemse, a also at luminescent microscopy.

Salk color test. As a result of the vital activity of cells in a nutrient medium accumulate sour products. AT result this color incoming in compound environments indicator (phenolic red) becomes orange. At contagion culture cells with cytopathogenic viruses such as enteroviruses or reoviruses, metabolism cells suppressed medium pH and her color not are changing (Wednesday remains red).

Reaction hemagglutination. AT basis this reactions lies ability viruses, containing hemagglutinin receptors, "glue" erythrocytes. If a there is hemagglutinins - RGA+(umbrella), if No - RGA - (button).

Reaction hemadsorption. Mechanism similar With RGA.

TEST TASKS

1. For microbiological diagnostics viral infections apply the followingbasic methodological approach
A) bacteriological diagnostics;b)
virological diagnostics;
in) serological diagnostics;
G) molecular biological diagnostics.
2. Viruses multiply only:
a) in living systems;
b) on the meat-peptone agar;
c) on differential diagnostic media;G) on the
elective environments.

3. The first step in virological diagnosis is to obtain and preparea) cell cultures;
b) chicken embryos;
c) sensitive laboratory animals;G) differential
diagnostic Wednesdays
4. Reveal viruses:
A) By cytopathic action;b) Of
Education plaques;
vpo color sample;
G) By biochemical properties.
5. Virus found in chick embryos A) by changing the chorioallantoic membrane; b) RGA (Reaction agglutination);
in RSK (Complement fixation reaction); G)
RP (Precipitation reaction).

PRACTICAL OCCUPATION No. 6.

Topic: Symbiosis and antibiosis. Residential and pathogenic microflora. Factors virulence microbes. Synergy and antagonism at microbes. antibiotics, mechanism actions and methods definitions sensitivity to antibiotics.
test control.

Educational goal:

1. Explore stages and factors symbiosis human With microbes.
2. Explore mechanism actions antibiotics on the microbial cell.
3. Explore methodology definitions sensitivity bacteria to antibiotics.

Student must know:

1. Stages and factors symbiosis human With microbes.
2. Microflora body person.
3. Terms formation associations residents.
4. Differences pathogens from residents.
5. Spectrum actions antibiotics on the microbial cell.
6. Definition sensitivity (methods indicator disks and cassette).

Student must be able to:

1. Conduct sowing material With fingers hands per cup With MPA (method prints).
2. Sowing detachable from nose and pharynx on the MPA.
3. describe results sensitivity clean culture to antibiotics.
4. Define sensitivity of bacteria to antibioticsindicator disks.

Plan lessons:

1. Stages and factors symbiosis human With microbes.
2. Terms formation associations residents.
3. Differences pathogens from residents.
4. antibiotics, definition, classification on chemical structure, spectrum,types and mechanism actions.
5. Chemotherapeutic drugs, mechanism them actions on the microbial cell.
6. Mechanisms medicinal sustainability bacteria.
7. Side effects of antibiotics and synthetic antimicrobials medicines.
8. Methods and units measurements antimicrobial activity.
9. Antiviral chemotherapy drugs.
10. Demonstration antibiotics With various mechanisms and spectrum actions.
11. Change module.

Independent Work students:

1. Take into account results disk antibiograms.

2. Take into account results cassette micromethod.
3. Design protocol research.

INFORMATIONAL MATERIAL ON THEME

Microorganisms are in various relationships friend With friend. The coexistence of two different organisms is called *symbiosis.* Distinguish several options for useful relationships: metabiosis, mutualism, commensalism, satelliteism.

Antagonistic relationships expressed in form unfavorable the impact of one type of microorganism on another, leading to damage and even death the last one. Forms antagonism: competition, predation, parasitism.

Microflora organism human

organism human populated about 500 types germs, constituents his normal microflora, in form communities microorganisms *(microbiocenosis).* They are are in able equilibrium *(eubiose)* friend With friend and organism person. Distinguish normal microflora various biotopes: skin, mucous shells oral cavity, upper respiratory tract, gastrointestinal tract and genitourinary system. In the body allocate permanent and transitory microflora. *Constant* microflora presented microorganisms constantly present in body. *Transient* microflora not capable to long existence in body. permanent microflora can divide on the obligate and optional. Obligate microflora (bifidobacteria, lactobacilli, peptostreptococci, Escherichia coli and etc.) is basis microbiocenosis, a optional microflora (staphylococci, streptococci, klebsiella, clostridia, some fungi, etc.) includes a smaller part microbiocenosis. Microorganisms that make up the normal microflora are enclosed in highly hydrated exopolysaccharide nomycin matrix, forming a biological film, resistant to various influences.

Protocol research

No.	researched material	results research	Graphic image

All antibiotics possess selectivity actions. Them relative harmlessness for human determined, before Total, topics what they specifically suppress such metabolic processes in microbial cage or virus, which missing in eukaryotic cage or unavailable for them. AT this respect unique is the mechanism of action of beta-lactam antibiotics. Targets for them are transpeptidases, which complete synthesis peptidoglycan cellular walls. Since only prokaryotes have a cell wall, a eukaryotic cell does not have targets for beta-lactam antibiotics. Transpeptidases are a set enzyme proteins, localized in cytoplasmic membrane bacterial cells. Selected beta lactams vary in degree affinities to this or otherwise enzyme which got title penicillin-binding proteins. That's why biological Effect beta-lactam antibiotics different: bacteriostatic, bactericidal, lytic.

Except beta-lactam antibiotics, synthesis cellular walls amaze such antibiotics, how bacitracin, fosfomycin, cycloserine, vancomycin, ristomycin, but otherwise by, how penicillin. All they, Besides cycloserine, cause bactericidal Effect.

Mechanism actions such antibiotics, how chloramphenicol, tetracyclines, streptomycin, aminoglycosides, erythromycin, oleandromycin, spiramycin and other macrolides, lincosamides, fusidian acid, tied With oppression synthesis squirrel on the ribosome level 708. Although bacterial ribosomes 708 have the same in principle structure, how ribosomes 808 eukaryotic cells, them squirrels and protein factors involved in the work of the protein-synthesizing system differ from those of ribosomes 808. This explains the selectivity of the action of these antibiotics on the protein synthesis bacteria.

Different antibiotics block protein synthesis in different ways. Tetracyclines block binding at-RNA on the A-section ribosomes 708. Chloramphenicol suppresses peptidyl transferase reaction. Streptomycins impede transformation initiator complex into a functionally active ribosome. Erythromycin blocks translocation reaction. Puromycin, joining the growing end of the synthesized polypeptide chain, causing its premature separation from the ribosome. Mechanism actions fluoroquinolones tied With them electoral suppression bacterial DNA gyrase enzymes involved in DNA replication. Fluoroquinolones are associated with specific plots DNA,

which created impact DNA gyrase, and suppress her activity.

Rifampicins inhibit the activity of DNA-dependent RNA polymerases, as a result of which at bacteria inhibit transcription.

The activity of anticancer antibiotics is due to the fact that they are either inhibitor synthesis DNA (bruneomycin), or suppress activity DNA dependent RNA polymerase, t. e. blocks transcription (anthracyclines, actinomycins, olivomycin).

Accounting results definitions sensitivity dedicated from researched material microorganisms to antibiotics held next way: on the working table located a cup Petri, on the which was sown dedicated from of the microbe under study and were applied at an equal distance from each other discs With antibiotics (method this work outlined in practical guide).

student necessary do conclusion about degree sensitivity dedicated culture to antibiotics. Meaning given research comes down to next: surface nutritional environments on the cup moisten suspension dedicated clean culture in physical solution and so way achieved uniform distribution culture on all cup. "Over" sowing superimposed discs With antibiotics and cups incubate in thermostat. FROM disks, impregnated each separate antibiotic, the diffusion of antibiotics into the thickness of the agar occurs. The more sensitive culture to antibiotic topics less his efficiency concentration and topics morethe diameter of the culture stunting zone around a particular disk. At the same time, the result taken into account according to the following scheme (table).

culture highly sensitive	diameter zones oppression growth bacteria thirty and more mm.
culture medium sensitive	diameter zones oppression growth bacteria not less twenty mm.
culture weakly sensitive	diameter zones oppression growth bacteria not more ten mm.

test control

1. Synthesis cellular walls suppress antibiotics:
   a) polymyxin
   b) aminoglycosides
   c) cephalosporins G)
   tetracyclines
2. Violation functions cytoplasmic membranes noted under action:a) cephalosporin
b) macrolides c)
levomecithinG)
nystatin
3. antibiotics, inhibitory synthesis squirrel on the ribosomes bacterial cells:a) penicillin
b) polymyxin
c) aminoglycosides
G) amphotericin AT
4. Antibiotics acting on nucleic acid synthesisa) erythromycin
b) oleandomycin
in) rifampicin G)
lincomycin
5. Sensitivity to antibiotics is determined by:a)
method membrane filters
b) method paper disks
c) two-phase fermentation methodG)
sedimentation method
e) aspiration method.

PRACTICAL OCCUPATION No. 7.

Topic: Serological method diagnostics. Mechanisms non-specific human resistance. Phagocytosis, complement system, lysozyme, etc. Antigens and antibodies. Serological reactions: agglutination, precipitation, lysis, hemolysis and binding complement. Immunofluorescent, enzyme immunoassay and radioimmune analysis in diagnostics infectious diseases.

Educational goal:

1. Explore physiological mechanisms immunity.
2. Explore serological methods laboratory diagnostics.
3. Explore complement dependent serological reactions,
4. Explore reactions immunity With labeled components.

Student must know:

1. Statement of the agglutination reaction (expanded). 2. Setting up the precipitation reaction, practical application.3. Receipt diagnostic sera, classification.
4. Setting up an immune lysis reaction. 5.Staging reactions binding complement (RSK).
6. staging reactions immunity With labeled components.

Student must be able to:

1. Put indicative reaction agglutination on the subject glass.
2. Put extended reaction agglutination.
3. Put reaction ring precipitation.
4. Put reaction immune lysis.
5. Put reaction binding complement (RSK).
6. Put reaction immunity With labeled components.

Plan lessons

1. Antigens them nature. Gaptens. Antigens bacteria.
2. Antibodies, classification. Structure immunoglobulins, main classes.
3. humoral and cellular immune answer
4. Serological reactions, them essence and mechanism, practical application. Serodiagnostics. Seroidentification.
5. Agglutination reaction , staging methods , reaction phases , practicalapplication.
6. Reaction precipitation, ways performances, practical application.
7. diagnosticums, classification, application.
8. Diagnostic serum, receiving and kinds diagnostic sera - agglutinating (adsorbed and non-adsorbed, mono- andpolyvalent), precipitating.
9. Demonstration deployed reactions agglutination, reactions hemolysis.
10. staging reactions ring precipitation.
11. Demonstration diagnosticums and diagnostic sera.
12. Reactions immune lysis, Components.
13. Reaction hemolysis.
14. Reaction binding complement (RSK). staging and accounting reactions binding complement.
15. Reaction immunofluorescence, straight and indirect.
16. ELISA analysis, Components, application.
17. radioimmune analysis, Components, application.

Independent Work students:

1. staging and accounting indicative reactions agglutination on the subject glass With purpose identification dedicated clean culture Gram-negative sticks.
2. staging and accounting deployed reactions agglutination With purpose serodiagnosis abdominal typhus.
3. staging and accounting reactions thermoring precipitation With purpose of seroindicationSiberian ulcers.
4. staging and accounting reactions binding complement With purpose serodiagnosis syphilis

INFORMATIONAL MATERIAL ON THEME LESSONS

Under **immunity** (from Latin immunitas - liberation, getting rid of something) in biology and medicine understand complex reactions organism, directed on the preservation of its structural and functional integrity when exposed to the body genetically alien substances, both coming from outside and formed inside organism.

Distinguish several major species immunity:

-*Hereditary immunity* (congenital, species) conditioned development in process phylogenesis genetically fixed immunity kind to given antigen or microorganism.

-*Acquired immunity* specific and not transmitted on inheritance. He formed naturally and created artificially. Natural acquired immunity appears after transferred infectious disease (smallpox, measles and etc.). Artificial acquired immunity arises at vaccination.

Immunity happens *active* and *passive. Active immunity* produced organism in result impact antigen on the immune system (eg. at vaccination). *Passive immunity* conditioned antibodies transmitted from immune mother to the child at birth or by administering immune sera, and also when transplanting immune cells.

Active immunity can be *humoral* (caused by antibodies), *cellular* (due to immunocompetent cells) and *cellular humoral* (due to antibodies and immunocompetent cells). For example, antitoxic immunity to botulism and tetanus is humoral So how he conditioned antibodies circulating in the blood immunity to leprosy or tuberculosis is cellular, and to smallpox - cellular humoral.

Distinguish also immunity *sterile,* persisting in absence microorganism, and *non-sterile,* which exists only in the presence of a pathogen in body. The classic example of non-sterile immunity is immunity to tuberculosis.

Separately allocate So called *local immunity,* which the protects individual plots organism, for example, mucous shells from pathogens infectious diseases. It is formed with the participation of secretory immunoglobulin A and more active phagocytosis.

Antigens are any substances that are genetically foreign to a given organism. (usually biopolymers), which, having entered the internal environment of the body or formed in body, cause reciprocal specific immunological reaction: synthesis antibodies, the appearance of sensitized lymphocytes or the emergence of tolerance to this substance hypersensitivity delayed or immediate types, immunological memory.

Antigens have specificity, which is associated with a certain chemical group in composition molecules, called determinant or epitope. Determinants antigen are those parts of it that are recognized by antibodies and immunocompetent cells.

Distinguish *full-fledged* and *defective (haptens) antigens.* Antigens defiant full-fledged immune answer, having 2 and more determinants, called *complete.* it organic substances microbial, vegetable and animal origin. *haptens* may to be chemical substances With small molecular weight or more complex chemical substances not possessing properties of a complete antigen: some bacterial polysaccharides, a polypeptide tuberculosis bacillus (PPD), DNA, RNA, lipids, peptides. *Haptens* due to small molecular weight are not fixed by immunocompetent cells of the macroorganism and cannot elicit an immunological response. Semi- *haptens* - inorganic radicals (iodine, bromine, nitro group, nitrogen, etc.) attached to the protein molecule, may change immunological specificity squirrel.

Antibody formation. AT answer on the introduction antigen immune system produces antibodies — proteins, capable specifically unite With antigen, that caused their formation and,

thus, participate in immunological reactions. Relate antibodies to y-globulins, t. e. least mobile in electric field serum protein fractions. In the body, y-globulins are produced by special cells — plasma cells. AT compliance With International classification y- globulins, bearing functions antibodies, got title immunoglobulins and are designated symbol lg. Consequently, antibodies — this is immunoglobulins, produced in answer on the introduction antigen and capable specifically to interact With this same antigen.

Functions antibodies. Primary function antibodies consists in interaction them active centers With complementary them determinants antigens. Secondary function antibodies consists of them capabilities:

- bind antigen With purpose his neutralization and elimination from the body;
- participate in recognition "foreign" antigen;
- provide cooperation immunocompetent cells (macrophages, T- and AT- lymphocytes);
- participate in various forms of the immune response (phagocytosis, killer function, immunological tolerance, immunological memory, hypersensitivity immediate type, hypersensitivity slow type).

Proteins of immunoglobulins in chemical composition belong to glycoproteins, so how they are made up of protein and sugars; built from 18 amino acids. Distinguish 5 classes immunoglobulins: IqM, IgG, IgA, IgE, IgD. Immunoglobulins M, G, A have subclasses. For example, IgG It has four subclass (IgGl, IgG2, IgG3, IgG4).

Immunological memory called ability organism at repeated meeting With one and topics same antigen to react more active and more fast formation immunity those. to react on type secondary immune response.

Immunological tolerance phenomenon opposite immunological memory. AT this case in answer on the repeated introduction antigen organism instead of energetic workings immunity shows areactivity, not answers immune reaction i.e., tolerant to the antigen.

I. Reaction agglutination on the subject glass

Place three drops on a glass slide at a sufficient distance from each other: physiological solution, typhoid agglutinating serum (No. one) and dysentery agglutinating serum (No. 2). researched culture contribute in drop physiological solution and thoroughly grind in her before appearance expressed turbidity. bacterial loop prepared suspension postpone in serum number 1 and mix thoroughly. Next, the bacteriological loop is necessary sterilize calcination. Then take bacterial loop material from suspension culture in a drop of saline and add it to a drop of serum No. 2. Glass slightly and carefully wiggle for careful mixing. Accounting results reactions are carried out after 1-2 minutes: in a drop of saline, uniform turbidity, while agglutination is noted in a drop of one of the serums. Signs agglutination are: dropping out grains agglutinate and enlightenment liquids. AT case detection in control drop With physiological solutionspontaneous agglutination, the results of the reaction are not subject to further accounting, and the reaction requires re-setting.

II. deployed reaction agglutination

An extended agglutination reaction was set up to determine the antibody titer in serum the patient's blood.

Researched serum getting divorced physiological solution in fifty once, and received so way breeding (1:50) counts original. Further initial breeding serum successively twice getting divorced physiological solution. For this (see diagram productions):

a) in all agglutination tubes, Besides No. 6, are made on 1.0 mlphysiological solution;

b) 1.0 ml of serum is added to test tubes No. 1 and No. 6 in the initial dilution of 1:50, and, so the way serum in test tube No. 1 divorced more twice, then there is in 100 times;

c) 1.0 ml of serum from test tube No. 1 is transferred to test tube No. 2 to the available1.0 ml of saline, as a result of which the serum is diluted two more times, then there is in 200 once, so Further, up to to the test tube No. 5, where breeding reaches 1:1600;

d) it is obvious that tubes No. 1 - No. 4 contain 1.0 ml of serum, while vial 5 contains 2.0 ml of ee - the excess 1.0 ml is removed, and thus volumes in test tubes No. 1 - No. 5 are equalized. In test tube No. 6, serum control. Further, in each test tube, with the exception of test tube No. 6, add 2 drops DIAGNOSTICUM - formalin-treated suspension in physiological solution of Salmonella typhi culture cells, each milliliter of which contains 2 billion bacterial tel. Tripod

With test tubes shake and put in thermostat at t 37°C on the 2 hours. After excerpts in thermostat tripod With reaction withstand at room temperature or at cold" (+3° +5°C) in flow eighteen hours.

Components reactions	An experience					serum	diagnosticia n crazy
	one	2	3	four	5	6	7
one. Phys. Solution	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2. Serum under study (1:50); ml	1.0 1:100	1.0 1:200	1.0 1:400	1.0 1:800	1.0 1:1600	1.0 1:100	1.0
3. Diagnosticum, drops	2	2	2	2	2	-	2

The results are recorded in a day in the following sequence: first turn assess the state control test tubes (#6 and No. 7), in second turn
—experienced. In test tube No. 6 (serum control) should be absolutely transparent, devoid of any draft liquid. AT test tube No. 7 (control diagnosticum)
- Uniform haze. The results of test tubes should be evaluated starting from test tubes with the highest serum dilution (No. 5). The result of the reaction is taken into account according to fallout on the bottom test tubes flakes agglutinate and simultaneous enlightenment the contents of the test tube; by lightly tapping on the wall of the test tube or gently shaking agglutinate easily separates from bottom, pops up and, not changing his structures, returns to original position.

III. Reaction ring precipitation

The precipitation reaction is most often used to determine the presence in the materialsoluble antigens. into a control precipitating tube, up to approximatelyhalf of its volume normal serum is added. In the experimental the test tube is introduced same amount precipitating serum. Further in every test tube is brought small amount researched material — for example, extract from skins animal (sheep), deceased presumably from Siberian ulcers. researchedmaterial should bring in through careful layering on the internal wall a precipitation tube held in the hand at a height of 30-35 cm from the surface working table under angle 45° to horizontal.

In the test tube, at the border of the serum and the test material, there is education precipitate: whitish "disk" irreversibly collapsing at shaking test tubes. AT control test tube education precipitate not observed.

IV. Reaction indirect (passive) hemagglutination (RITA)

RPGA founded on the use erythrocytes With adsorbed on the them surfaces antigens (erythrocyte diagnosticum), interaction which With relevant antibodies serum blood sick causes dropping out erythrocytes in sediment on bottom test tubes (wells) in form "revealed umbrella."

The studied patient's serum is diluted 10 times and heated at 65°C for 20 minutes. in a water bath to remove nonspecific hemagglutinins, then prepare a series of dilutions from 1:100 to 1:3200 and poured into wells of 0.5 ml. Add to each well on 0.5 ml diagnosticum. AT each row holes added corresponding erythrocyte diagnosticum: to shigella Sonne, Flexner, Newcastle and polyvalent salmonella.

At the same time, control diagnosticums and control of the studied serum are put. The result of the reaction is taken into account after incubation in a thermostat for 2 hours at 37°C. or at room temperature for 1824 hours. The response is considered positive. subject to the location of erythrocytes in the form of an "umbrella" over the entire surface hole bottom and rated as "+"

Scheme productions

Breeding researched serum	DIAGNOSTICS				CONTROL				
	Sonne	Flexner	New castle	Salmon. watered.	CD 1	CD 2	CD 3	CD 4	Ks

1:100									
1:200									
1:400									
1:800									
1:1600									
1:3200									
Incubation at t 37 ^{0} C; 24 hours.									
Accounting results									

principled scheme productions binding reactions complement

Components reactions	No. test tubes		
	1(exp)	2 (counter)	3 (counter)
1. Исследуемая сыворотка (1:5)	0,5	0,5	-
2. Антиген в рабочей дозе	0,5	-	0,5
3Комплемент в рабочей дозе	0,5	0,5	0,5
4.Физиологический раствор	-	0,5	0,5
Incubation at t 370 C – — 40 minutes.			
5. Hemolytic system(sheep erythrocytes + hemolytic serum)	1.0	1.0	1.0

Incubation at t 370C – _ — 40 minutes.

Accounting results _____ Hemolysis + Hemolysis +

Conclusion:

In the presence of antibodies in the test serum (positive reaction) in the experimental there is no hemolysis in vitro. With a negative reaction (no antibodies) in all three test tubes hemolysis is observed.

The complement fixation reaction takes place in two phases: 1st phase - interaction researched serum With antigen and complement. 2nd phase — indicator — determination of the presence of free complement in the mixture by adding hemolytic system consisting of sheep erythrocytes and hemolytic serum containing antibodies to sheep erythrocytes. If in the first the reaction phase is the formation antigen-antibody complex, complement is bound by this complex in the second phase hemolysis of erythrocytes is absent (positive reaction). If antigen and antibody are not correspond to each other, the complement in the first phase of the reaction remains free and during in the second phase of the reaction, it joins the erythrocyte-hemolytic serum complex, causing hemolysis (negative reaction).

TEST TASKS

1. What kind Components participate in reactions indirect hemagglutination?

a) antibodies, antigens, complement;

b) antibodies, antigens, saline solution;in) antigens, physiological solution;

G) antigens, erythrocytes, antibodies, physiological solution.

2.What kind Components participate in reactions precipitation?

a) corpuscular antigens, antibodies, physiological saline;b) soluble antigens, antibodies, physiological solution;

in) antigens, antibodies, complement;
G) antigens, antibodies, erythrocytes, physiological solution.

3.What components are involved in the hemagglutination inhibition reaction?a
) antigens, antibodies, saline;
b) antigens, antibodies, complement;
c) corpuscular antigens, antibodies, saline solution;G) viruses,
erythrocytes, antibodies;
e) bacteria, erythrocytes, antibodies.
4.Additional component (Besides antigens and antibodies) participating in reactions agglutination is:
a) guinea pig serum complement;b) isotonic
solution NaCl;
in) erythrocytes;
G) hemolytic system.
5.Visual result reactions agglutination:
a) hemolysis erythrocytes ram;
b) delay hemolysis erythrocytes ram;
in) enlightenment cloudy environments reactions and education coarse (grainy)draft;
G) turbidity transparent environments reactions and education finely dispersed suspension (flocculate) or rings precipitation.
6. Varieties reactions immune lysis are:
a) hemagglutination;
b) hemolysis;
c) ring precipitation;G)
bacteriolysis;
e) cytolysis
7. What kind more Components (Besides antigens and antibodies) participate in reactions binding complement?
a) bacteria;
b) complement;
in) erythrocytes ram;
d) hemolytic serum;e)
physiological solution.
8. At staging enzyme immunoassay analysis antibodies or antigens tag:a)
peroxidase;
b) fluorochrome; c)
a radioisotope; G)
complement.
9. The most modern serological reactions are:a) agglutination reaction;
b) complement fixation reaction;in)
enzyme immunoassay analysis;
G) polymerase chain reaction;
e) reaction immunofluorescence.
10. Reveal Availability characteristic for certain microorganism site nucleic acid in the test material and multiply it many times allows:a) linked immunosorbent assay;
b) polymerase chain reaction;in)
radioimmune analysis;
G) reaction immunofluorescence.

PRACTICAL OCCUPATION No. eight.

Topic: Immunoprophylaxis, immunotherapy and immunocorrection. Assessment Methods immune status person: flowing cytofluorometry With monoclonalCD antibodies, leukocyte chemiluminescence, lymphocyte blast transformation and others Immunobiological drugs: vaccines, toxoids, serum. Immunomodulators. Probiotics.

Educational goal:

1. Explore tests first and second level, them clinical interpretation.
2. Explore pathogenesis secondary immunodeficiencies
3. Explore genetics immunodeficiencies, peculiarities inheritance.
4. Explore congenital immunodeficiencies
5. Decide and take into account functionally condition phagocytes,
6. Define activity complement blood

Student must know:

one Age peculiarities immune status.
2. Methods research lymphocytes, evaluation functional states phagocytes,
4. genetics immunodeficiencies, peculiarities inheritance.
5. Secondary immunological deficiency (SID) - classification, etiology,diagnostics

Student must be able to:

1. Decide and take into account functionally condition phagocytes,
2. Define activity complement blood
3. Estimate and interpret indicators immune status at secondaryimmunological insufficiency

Plan lessons:

1. Immune status and principles his estimates.
2. Age peculiarities immune status.
3. Methods research lymphocytes, grade functional states phagocytes.
4. Definition complement
5. Tests first and second level, them clinical interpretation.
6. Genetics immunodeficiencies, peculiarities inheritance.
7. Congenital immunodeficiencies (classification, diagnostics)
8. Congenital immunodeficiencies at children.
9. Secondary immunological failure (VIN) - classification, etiology,diagnostics

Independent Work students:

1. staging and accounting functional states phagocytes,
2. Definition complement
3. Assess and interpret indicators of the immune status in secondaryimmunological insufficiency in ready immunograms

INFORMATIONAL MATERIAL

Maturation immune reactivity fetus

The thymus is laid down in the second month of intrauterine life in the area of the third fourth gill pockets and at the sixth week has a pronounced epithelial character. On the 7-8 week he "settled" lymphocyte-like cells. To end third months formation body ends. AT further in thymus observed only quantitative changes. Lymphatic nodes and other secondary bodies immune systems are laid on the 4th month, them final formation ends in postnatal period. Lymphoid follicleslocated in iliac gut and appendix, in Peyer's plaques contain progenitor cells of plasma cells. They mature to plasma cells, synthesizing IgA to 14-16 weeks intrauterine development fetus.

Stem cells appear at 3-8 weeks of embryogenesis and are found in liver, blood islands of the yolk sac. Later, their main place of education becomes bone marrow. Lymphocytes are first detected at week 9 in the thymus, at 12- fifteen — in spleen. AT blood lymphocyte-like cells determined With 8-10 weeks. Lymphoid cells endowed with the function of T-lymphocytes are

detected at 10-11week. B-cells are determined in the liver from 10-12 weeks, in the spleen - from 12 weeks. Synthesis and secretion of IgM is registered in the cells on the 11th, IgG - on the 22nd week. IgM content is 1/10 from maternal, a IgG — yet less. Education components systems complement starts at fetus on the 8th week pregnancy. At this components C2 and C4 are synthesized by macrophages, C5 and C4 - in the liver, lungs, peritoneum; al cells, C3 and C1 - in the small and large intestine. At the 18th week of development, all these components are determined in the blood serum of the fetus. Cellular and humoral factors of non-specific anti-infectious immunity appear in the early ontogeny.

AT period embryonic development "Work" immune systems It has their peculiarities. AT in particular among T-dependent immunological reactions first appears ability to rejection transplant (13 a week), HRT implemented much later.

Despite on the Availability in body fetus significant quantity B cells With immunoglobulin receptors plasmatic cells, directly synthesizing AT, very few. Row very powerful factors suppresses function humoral part of the immune system. This is a choriotropic gonadotropin, a-fetoprote- in, a-2-globulin. During this period, the influence of T-lymphocytes on B-cells and macrophages.

Premature activation of the immune system is observed during intrauterine infection. Practically always this is accompanied by any immunopathological disorders. Thus, for the embryonic period typical stage immunogenesis is tolerance own immune systems and passive antibody immunity due to maternal IgG, the concentration of which progressively is growing in process pregnancy. Ability fetus form components of the complement system are defective. In the third trimester of her level, although increases, but is no more than 30-50% of the indicators of adults. Local immunity in early and late ontogeny not developed.

Immune status at children after birth

A healthy full-term baby born to a healthy mother with physiological flow pregnancy, It has definite immune status and corresponding level factors non-specific anti-infectious resistance. Peculiarthe nature of the passive immunity of the newborn has positive and negative sides. So, without receiving IgM from the mother, the fetus is not saturated with those associated with this class. group isohemagglutinins, what reduces risk development conflict at mismatch of group erythrocyte Ag. On the other hand, a low protection against gram-negative bacteria, since this fraction predominantly are AT against specified pathogens. AT moment birth at child physiological leukocytosis is observed, reaching up to 12-15 billion cells / l. From cells more 35% constitute lymphocytes. From general numbers lymphocytes near half make up T cells. In relative terms, their content is moderately reduced, and in absolute, given the high leukocytosis, is not changed. About 60% of all T-lymphocytes constitute cells With helper functions, fifteen% - T-suppressors.

Content antibody-dependent killers also strongly reduced from level adults. Functions of lymphocytes of newborns are changed. So, the intensity of the reaction blast transformation, induced T-mitogen FGA, "normal" or several lowered how at more senior children. reduced them ability produce lymphocytes,

induce skin reactions. At the same time, cells show a higherlevel metabolism, if judge on intensity synthesis nucleic acids.

Quantity B cells at newborns usually raised in relative and absolute values. How rule on the these cells are found IgM and IgE receptors. In the umbilical cord blood of newborns, IgM and IgG are detected, IgA and IgE are extremely rare. Synthesis of IgM increases sharply, reaching a maximum of 2-3 weeks life child, then to monthly age decreases in further slowly increases, reaching the level of adults by 6-12 months. Excessive increase in concentration IgM at newborns is evidence transferred intrauterine infections. Most often it is syphilis, rubella. Threefold increase in IgM level is evidence the presence of sepsis at child.

IgG concentration is very low at birth, increases to 7-8 years. In artificially fed children, this dynamics is realized much faster. IgA in serum blood newborns, how rule missing in flow first months life. AT further content immunoglobulin slowly is growing by the end of the first year, 28% of the level of this protein in adults. Normalization parameter is achieved by 8-15 years. IgD is usually not detected in newborns. This protein appears approximately at the 6th week, reaching the level of adults by 5-10-15 years.IgE is also not found in newborns, gradually increasing, it approaches values adults of people to 11-12 years. Acceleration accumulation

reagina is risk development at children bronchial asthma and others allergic and especially atopic diseases.

It is known that the content of immunoglobulins is determined by the amount of AT of various specificity. Earlier than others in children, the appearance of immune globulins has an effect microflora organism child. Main representative intestinal microflora in this

period are bifidumbacteria. That's why any unfavorable factors(artificial feeding, application antibiotics) inevitably entail per yourselfviolation specific composition microflora and changes spectrum emerging AT.Antibody formation at newborns, how rule leaks only on primarytype, requiring for implementation big quantity Ag. Much slowed downswitching synthesis from IgM to IgG, within 5-20 days in adults and 20-40 in children.AT moment birth phagocytes and serum blood newborns possess certain bactericidal activity against row microbial strains. Chemotaxisand functional activity macrophages reduced. Partially this is compensatedincrease content granulocytes, So same endowed phagocytic function.However, the digestive capacity of these cells is reduced due to the immaturity of the enzymes.Child is born co reduced on comparison co adults levels complement and properdine, which enough fast are growing. Initial activity lysozyme, against, significant.

Content lysozyme in body not always, depends from age, time of the year, vitamin balance and others More Total lysozyme in saliva children (before 200 mcg/ml), which is many times higher than its concentration in blood serum. The highest content lysozyme in saliva children first of the year life, in age 1-6 years it declining nearly in 3 times, to 7-15 years increases but not reaches original level.

Important factor local immunity is IgA, which the located in two forms

- serum and secretory. This y-globulin plays a major role in resistance organism against respiratory, viral, bacterial, parasitic infections, etc. Secretory IgA begins to be detected in the secrets of the first and early second weeks, continues progressively grow in subsequent months and years, in coprofilters is found from the third week of life. The amount of secretin is constantly replenished for account of secretory IgA of milk and, especially, colostrum, where its amount is 20 times or more surpasses level in serum adult. Usually after 3-5 days lactation concentration IgA sharp decreases but, Considering increasing consumption milk child, its quantity Plasma cells located in the mucous membranes, form IgA, IgM, IgG, IgD, IgE. Wall intestines synthesizes before 3 G immunoglobulins in day. IgG provide protection in mostly against toxins the rest are against bacteria and viruses. Formation of full-fledged local immunity on different data ends at one to twelve years life.

Ratio plasmatic cells gastrointestinal track, producing immune globulins, at some diseases is changing. So, at young children (from birth to three years) with chronic gastroduodenitis observed deficit IgA and

increase in IgM production. In patients with cholecystitis, there is a decrease IgA concentrations and an increase in IgM or IgG. With peptic ulcer of the duodenum 12 going on the fall level IgA in duodenal content. deficit local IgA facilitates binding immune globulins others classes With Ag.

Local immunity is determined not only by humoral, but also by cellular factors. It is shown that in the first 24 hours after the birth of a child there is a sharp increase in the number of alveolar macrophages. Their number continues to increasemonthly

age, after what stabilizes. Microbicidal properties macrophages and other phagocytic cells, as a rule, lag behind in children of the first weeks and even months life.

State immune systems child in first years life characterized high dynamism. So, after birth declining number leukocytes in circulation, the percentage of lymphocytes increases, the number of granulocytes.

Crossroads between curves reflective dynamics these cells, first occurs on the 5th day of life, after which a similar cross (decrease in specific weight lymphocytes and an increase in neutrophils) is noted only aged 4-5 years.

Highly slowly

rises relative content T cells level B-lymphocytes steadily decreases to normal.

Thus, for the embryonic period, tolerance and passive immunity per check maternal IgG, concentration which is growing in the process of pregnancy. The newborn is also dominated by

maternal passive immunity, although the beginning of the synthesis of own AT, endowed with a small12 months, immune reactivity matures. At the age of 1-3 years, T- cellular immunity. AT this same period actively are functioning and B-lymphocytes.

From stated follows, what organism newborn up to before annual age poorly protected from infectious agents. Active main way humoral link immunity. T-dependent reactions and phagocytosis developed not enoughand enter in complete force later. Sometimes only to period sexual maturation. Considering all these intelligence appointment children immunotropic funds must produced extremely carefully, to not pervert natural peculiarities response, mistaking for immune disorders physiological changes in immune reactions.

In many diseases in children, early involvement in the pathological process liver and spleen. These bodies in intrauterine period carry out hemo- and lymphopoiesis. That's why in answer on the damage or infection fetus answers activation of the reticuloendothelial system. After birth, its significance decreases, replaced by more advanced machines. However, some of the so-called "slowlystarting children" with delay maturation immune systems possible reaction on the pathogenic situation of these organs. At present, in the life of a child, several critical periods, which characterized greatest vulnerability organism (D.V. Stephanie Yu.E. Veltishchev, 1996). In intrauterine period critical should count age 8-12 week, when going on differentiation organs and cells of the immune system. First critical period after birth is period newborn™, when organism exposed action huge Ag numbers. The immune system at this time is subject to strong suppressive influences, passive humoral immunity conditioned maternal AT. noted functional imbalance of T-lymphocytes, the suppressor function is realized not only CD8+ cells, but and immature thymocytes and others cells.

The second critical age (3-6 months) is characterized by a weakening of the passive humoral immunity in connections With catabolism maternal AT. At this the suppressive orientation of immune peaktia is preserved in the presence of a pronounced lymphocytosis. This type of immune response occurs with tetanus vaccination, diphtheria, whooping cough, poliomyelitis, measles, and only after the 2-3rd revaccination develops secondary immune answer With IgG formation AT and persistent immune memory.

The third critical period is the 1st year of life. At this time, the primary character immune response on the many Ag, but already Maybe switching on the formation of IgG-AT. However, the synthesis of the IgG2 and lgG4 subclasses is delayed. suppressor the direction of the immune mechanisms begins to change into a helper one. Local immunity not developed, children sensitive to respiratory viral infections. Fourth critical period — 4th—6th years life. AT this age average the concentration of IgG and IgM in the blood corresponds to that in adults, the concentration of IgA in plasma has not yet reached the final values, the content of IgE in the blood reaches maximum values. This period is characterized by a high frequency of atopic, parasitic, immunocomplex diseases.

Fifth critical period — teenage age (at girls With 12-13, at boys from 14-15 years old). Pubertal growth spurt combined with weight loss lymphoid organs. Raise secretions genital hormones (before

total androgens) leads to suppression of the cellular link of immunity and stimulation his humoral mechanisms. AT in general at children meet the following peculiarities links immune status. T — link immunity. Quantity lymphocytes peripheral blood at birth in first day life is 24-30%, a absolute number - 3-9 • Yu'/l. Then their relative number increases and to 4-5- m days reaches 40-50%, absolute - 2.5 - 10 billion / l.

Lymphocytes of newborns are characterized by high metabolic activity, in they increased the synthesis of DNA and RNA. BTL when cultivated with PHA is well expressed as at full-term, So and at premature newborns. noted high level spontaneous transformation, in average near 6-10%, then how at adults this index is near 0.2%. AT — link immunity. System humoral immunity in difference from cellular starts actively function only afterbirth under the influence antigenic irritation. At birth child content IgG in his blood is usually greater than that of the mother, since the transplacental transition of this immunoglobulin is active process. IgM in serum usually missing or determined in minimal quantities. IgA are usually absent or present in trace concentrations. To end first weeks content IgA and IgM several increases IgG — to 2nd-3rd week noticeably declining and reaches minimal concentrations in age 1-4 months

phagocytic link. Number neutrophils in blood at birth relatively large: 50-70% and 4.5-

20 billion / l. From the 4th day, it begins to decrease to 30-40% - 2.5-6 billion/l Monocytes in flow Total period newborn™ constitute 4-9 %
- 0.6-2 billion / l. Absorptive capacity of neutrophils of newborns is not reduced, but digesting activity lowered what leads to unfinished phagocytosis. Number HCT-positive neutrophils in spontaneous reactions at children the first 2 weeks of life is 14-20%, while in older children - 2-10%. The rise in the number of these cells in the induced test is low; phagocytic reserve cells at children in age two weeks small. Monocytes newborns characterized low bactericidal activity and inadequate migratory ability. Immunodeficiencies (IDS) - disorders of immunological reactivity, caused by the loss of one or more components of the immune apparatus or closely interacting With him non-specific factors.

There is no single classification. By origin, immunodeficiencies are divided on the primary and secondary.Contents [put away]

Definition and classification

Primary immunodeficiencies are congenital (genetic or embryopathies) immune system defects. Depending on the level of violations and localization of the defect they there are:

humoral or antibody - with a predominant lesion of the B-system lymphocytes)
X-linked agammaglobulinemia (Bruton's disease)Hyper-
IgM syndrome
X-linked autosomal
recessive
deletion of immunoglobulin heavy chain genes
deficit k-chains
selective deficit subclasses IgG With or without deficit IgAdeficiency
of antibodies with normal levels of immunoglobulins general variable
immune deficiency
IgA
deficiency
cellular
syndrome Di Georgie primary
deficiency of CD4 cellsdeficit
CD7 T cells
deficit IL-2
multiple cytokine deficiencysignal
transmission defect combined:
syndrome Wiskott-Aldrich

ataxia-telangiectasia (syndrome Louis Bar)
severe combined immunodeficiencyX-linked With floor
autosomal recessive deficit
adenosine deaminase
deficit purine nucleoside phosphorylase
deficiency of MHC class II molecules (bald lymphocyte syndrome)
reticular dysgenesis
CD3γ or CD3ε deficiency
deficit CD8 lymphocytes
insufficiency of the complement system
defects phagocytosis
hereditary neutropenia
infantile lethal agranulocytosis (disease Kostman)cyclic neutropenia
familial benign neutropeniadefects in
phagocytic function chronic granulomatous
disease
X-linked autosomal
recessive
type I lymphocyte adhesion deficiency
deficit adhesion leukocytes 2 type
neutrophil glucose-6-dehydrogenase deficiency
myeloperoxidase deficiency
deficiency of secondary granules
Shwachman's syndrome Clinical
picture of IDS Clinic It has row
general crap:

1. Recurrent and chronic infections top respiratory ways, adnexal sinuses skin, mucous shells, gastrointestinal tract, often called opportunistic bacteria protozoa, mushrooms, having trend to generalization, septicemia and torpid to conventional therapy.

2. Hematological deficits: leukocytopenia, thrombocytopenia, anemia (hemolytic and megaloblastic).

3. Autoimmune disorders: SLE-like syndrome, arthritis, scleroderma, chronic active hepatitis, thyroiditis.

4. Often, IDS is combined with type 1 allergic reactions in the form of eczema, edema Quincke, allergic reactions on the introduction medicinal drugs, immunoglobulin, blood.

5.Tumors and lymphoproliferative diseases in IDS occur 1000 times more often, how without IDS. [one]

6. At sick With IDS often are celebrated disorders digestion, diarrheal syndrome and malabsorption syndrome.

7. Sick With IDS different unusual reactions on the vaccination, a application at them alive vaccines dangerous development sepsis.

8. Primary IDS often fit together With vices development, before Total With hypoplasia of cellular elements of cartilage and hair. Cardiovascular malformations are described, main Thus, with the syndrome Dee George.

[edit]

Treatment primary IDS

Etiotropic therapy is in corrections genetic defect methodsgenetic engineering. But such an approach is experimental. Main efforts at

established primary CID are aimed at: prevention

infections

substitution correction defective link immune systems in form transplants bone brain, substitution immunoglobulins, transfusions neutrophils.

enzyme replacement therapycytokine

therapy vitamin therapy

treatment of associated infections

Secondary immunodeficiencies

Factors that can cause secondary immunodeficiency, very varied. Secondary immunodeficiency can be caused by both environmental factors and internal factors of the body. In general, all adverse environmental factors environments that can disrupt the body's metabolism can cause the development secondary immunodeficiency. To most widespread factors environmental environment, defiant immunodeficiency relate pollution environmental environment, ionizing and microwave radiation, acute and chronic poisoning, long-term use some medicinal drugs, chronic stress and overwork. General a feature of the factors described above is a complex negative impact on all body systems, including the immune system. In addition, factors such as ionizing radiation render electoral inhibitory action on the immunity associated With oppression systems hematopoiesis. People, living or working in conditions polluted environmental environment, more often get sick various infectious diseases and are more likely to suffer from cancer. Obviously, what such promotion incidence at this categories of people related co declineactivity immune system.

The reasons

Secondary immunodeficiencies are frequent complication many diseasesand states. Main the reasons secondary IDS:

defect nutrition and general exhaustion organism also leads to decrease immunity. Against the background of general exhaustion of the body, the work of all internal organs. immune system especially sensitive to lack vitamins, minerals and nutritional substances So how implementation immune protection this is energy intensive process. Often decline immunity observed in time seasonal vitamin insufficiency (winter spring)

chronic bacterial and viral infections, as well as parasitic infestations (tuberculosis, staphylococcosis, pneumococcosis, herpes, chronic viral hepatitis, rubella, HIV, malaria, toxoplasmosis, leishmaniasis, ascariasis, etc.). With various chronic diseases infectious character immune system undergoes serious changes: violated immunoreactivity, develops increased sensitization to various microbial antigens. In addition, in the background chronic infectious process, intoxication of the body and oppressionfunctions hematopoiesis. Immunodeficiency in time infections HIV mediated electoral defeat cells immune system virus

helminthiases

loss of immune defense factors is observed during severe blood loss, with burns or with kidney disease (proteinuria, chronic renal failure). The common feature of these pathologies is a significant loss of blood plasma or proteins dissolved in it, part them which is immunoglobulins and others components immune systems (proteins systems compliment C-jet protein). In time bleeding not only plasma is lost, but also blood cells, therefore, against the background of severe bleeding decline immunity has combined character (cellular humoral)

diarrhea syndrome

stress syndrome

severe injuries and operations also occur with a decrease in the function of the immune systems. Generally any serious disease organism leads to secondary immunodeficiency. This is partly due to metabolic disorders and intoxication. organism, a partly With topics what in time injuries or operations stand out large quantity hormones adrenal, which oppress function immune systems

endocrinopathies (DM, hypothyroidism, hyperthyroidism) lead to a decrease in immunity due to metabolic disorders. The most pronounced decrease in immune reactivity organism observed at sugar diabetes and hypothyroidism. At these diseases declining production energy in fabrics, what leads to violation processes division and differentiation cells, in volume including and cells immune systems.Against the background of diabetes mellitus, the frequency of various infectious diseases is significantly rises. This is due not only to the suppression of the function of

the immune system, but also to the fact that what elevated content glucose in blood sick diabetes stimulates reproduction bacteria

sharp and chronic poisoning various xenobiotics (chemical toxic substances medicinal drugs, narcotic means). Especially expressed decline immune protection in time reception cytostatics,glucocorticoid hormones, antimetabolites, antibiotics

low weight body at birth

decreased immune defenses in the elderly, pregnant women and children related With age and physiological features organism these categories of people

malignant neoplasms - violate activity all systems organism. Most expressed decline immunity observed in case malignant blood diseases (leukemia) and red bone marrow replacementtumor metastases. Against the background of leukemia, the number of immune cells in the blood sometimes increases tens, hundreds and thousands of times, however, these cells are non-functional and therefore not can provide normal immune body protection

autoimmune diseases arise due to violations functions immune systems. Against the background of diseases of this type and in their treatment, the immune system works not enough and, sometimes, incorrectly, which leads to damage to their own tissues and inability fight the infection

Treatment secondary IDS

The mechanisms of immune suppression in secondary IDS are different, and, as a rule, there is a combination of several mechanisms, disorders of the immune system are expressed in to a lesser extent than in the primary. As a rule, secondary immunodeficiencies are coming character. AT connections With this treatment secondary immunodeficiencies much easier and more efficient on comparison With treatment primary violations functions immune systems. Usually treatment secondary immunodeficiency start With definitions and eliminate the reasons his occurrence. For example, treatment immunodeficiency on the background chronic infections start With sanitation foci chronic inflammation. Immunodeficiency on the background vitamin and mineral insufficiency start treat at help complexes vitamins and minerals. Recovery capabilities immune systems are great that's why elimination causes of immunodeficiency, usually leads to the restoration of the immune system. For acceleration recovery and stimulation immunity carry out well treatment immunostimulating drugs. AT the present time known big number immunostimulating drugs, With various mechanisms actions.

CLASSES IMMUNOGLOBULIN

IgA , Ig M, IgF, IgE , IgD
IgA , IgM , IgG , IgE , IgD (+)
IgA , Ig M, IgG , Ig E, IgF
IgM, IgG, IgE, IgF, IgD
IgA , IgG , Ig E, IgF, IgD

HIGH LEVEL GENERAL IgE CHARACTERIZES

helminthiases, allergy (+)
allergies, autoimmune diseases
helminthiases, immunodeficiencies
immunodeficiencies, allergies
helminthiases, viral infections

CLINICAL MANIFESTATIONS OF C -4 COMPONENT DEFICIENCYCOMPLEMENT

rheumatoid arthritis
tuberculosis periodic
illnessalveolitis
SLE (+)

A CASCADE SYSTEM OF BLOOD SERUM THAT CAN CAUSE LYSISCELLS, THIS IS

complement system (+)
cytokine network
interferons

kalecrein-kinin system
immunoglobulins

At SICK ALLERGY To YODUS, TO HIM CONTRAINDICATED

butadione brufen
enteroseptol (+)

PRACTICAL OCCUPATION No. 9.

TOPIC: Microbiological diagnostics bacterial infections. Working offmethods diagnostics For example the following pathogens:
1. staphylo-, entero- and streptococci (bacteriological method)
2. Neisseria and mycoplasma (microscopic method)

TRAINING GOAL:

1. Explore biological properties staphylococci.
2. To study methods of microbiological diagnostics of staphylococcal, streptococcal diseases.
3. To study the morphological and cultural properties of pathogenic gram-positive and gram-negative strepto- and diplococci (Neisserium).
4. master main methods laboratory diagnostics diseases, calledpathogenic diplococci.

Student must know :

1. Morphology, cultural, tinctorial properties of staphylococci and streptococci. Enzymatic activity.
2. pathogenicity factors and toxins. Them role in pathogenesis staphylococcal infections.
3. Main diseases called staphylococci, Pathogenesis, peculiaritiesimmunity at staphylococcal infections. Sources and way transmission infections.
4. Principles microbiological diagnostics, main method research, scheme identification dedicated clean culture, phage typing.
5. Specific prevention and treatment of staphylococcal infections. 6 . Morphological, cultural and biochemical features of diplococci;7. Factors pathogenicity, antigenic structure;
8. Sensitivity to antibiotics;
9. Main research methods : bacterioscopic, bacteriological,serological, bioassays, express diagnostics;
10. Prevention and treatment gonococcal infections.

Student must be able to :

1. Holding bacteriological research (on scheme). Accounting and interpretationresults.
2. Cooking smear and coloring on Gram.
3. Light microscopy of preparations from pure cultures of staphylococci,streptococci.
4. Light microscopy of preparations from pure cultures of meningococci, gonococci, pneumococcus, coloring by Gram.
5. Holding bacteriological research: spinal liquids on the suspicion meningococcal infections; slime from top respiratory ways sick on the pneumonia; detachable With urethra on the gonorrhea (on scheme).

PLAN LESSONS:

1. Morphology, cultural and biochemical properties staphylococcus.
2. Factors virulence staphylococcus.
3. Antigens staphylococcus.
4. diseases, called staphylococcus.
5. Methods diagnostics and researched material at staphylococcal diseases.
6. Preparations for specific prevention and treatmentstaphylococcal diseases.

7. Morphological characteristic pneumococcus (Streptococcus pneumoniae), meningococcus, gonococcus.
8. Comparative characteristics of biochemical activity and needs fornutritional environments for diplococci of different types.
9. Differential diagnostic signs (differences) of pathogenic andnon-pathogenic neisseria.
10. Factors virulence pathogenic diplococci.
11. Source infection, way transmission, input gates at diseases, causeddiplococci.
12. researched material and main methods diagnostics at pathologicalprocesses, called diplococci.

INDEPENDENT WORK STUDENTS:

1. To study the morphology of staphylococcus in a pure culture smear , describe, sketch.
2. To give macroscopic characteristic colonies on the milk-salt agar(bacteriological method diagnostics, 1st research phase).
3. To identify culture staphylococcus on morphological, cultural, biochemical properties, define factorsvirulence (2nd stage bacteriological method):
   a) recording the results of inoculation of staphylococcus culture on blood agarWith the purpose of determining hemolysin.
   b) taking into account the results of seeding in citrated plasma to determine plasmacoagulase. in) accounting seeding results on the yolk-salt agar for the purpose definitionslecithinases.
   G) accounting results sowing on the Wednesday With mannitol.
4. describe drugs for specific therapy and prevention staphylococcal diseases (staphylococcal toxoid, antistaphylococcal plasma, antistaphylococcal immunoglobulin, staphylococcal bacteriophage).
5. The study morphology pneumococci (str. pneumoniae) in smears- prints from bodies white mice, infected intraperitoneal phlegm sickpneumonia. Coloring according to Gram (table).
6. The study of biochemical activity pneumococci with the aim of differentiating themfrom streptococci. Sowing on the environments with inulin and bile.
7. Microscopic method diagnostics acute gonorrhea: microscopy smear purulent discharge from the urethra of a patient with acute gonorrhea. Coloring with methylene blue.
8. Serological method diagnostics chronic gonorrhea: estimate demonstration reaction binding complement (on Borde-Jangu), delivered with purpose detection antibodies in serum sick gonorrhea.
9. Decor protocol research.

METHODOLOGICAL RECOMMENDATIONS

The main method for diagnosing staphylococcal diseases - bacteriological. To isolate a pure culture, the test material is inoculated on yolk-salt, blood or milk-salt agar. Grown isolated colonies are subcultured on oblique agar to obtain pure culture.

Identification of a pure culture is carried out by morphological, cultural, biochemical properties, then determine factors virulence.

I. DETERMINATION OF HEMOLYTIC ACTIVITY BACTERIA.

On the cup With bloody agar made sowing culture staphylococcus. cups leave in thermostat on 24 hours at a temperature 37 degrees.

When evaluating the results, attention is paid to areas of hemolysis, i.e. enlightenment environment around grown colonies. The hemolytic properties of bacteria are associated with the presence of hemolysin (exotoxin).

II. DEFINITION LECITINASES

Staphylococcus aureus was inoculated on a plate with yolk-salt agar. Cups leave in thermostat on the 24 hours.

When evaluating the results, the presence of turbidity haloes around the colonies is taken into account, what testifies about education staphylococcus aureus enzyme lecithinases.

III. FOR DETECTIONS ENZYME PLASMACOAGULASES

A culture of staphylococcus aureus is inoculated into citrated plasma. The tubes are placed in thermostat. The results are taken into account after 24 hours. In the presence of the enzyme plasmacoagulase going on coagulation plasma With education clot fibrin. Availability enzyme plasma coagulase is main identification sign kind S.aureus, which the often is the causative agent nosocomial infection.

IV. MANNITE FERMENTATION DETERMINATIONAT ANAEROBIC CONDITIONS

For definitions this sign, confirming belonging clean culture of staphylococcus to the most aggressive species S.aureus, sowing was done on Wednesday with mannitol. When splitting mannitol, acidic products are formed that change the color of the indicator in the environment (the Andrede indicator - gives a red color to the environment, and the indicator VR - blue).

№№ P/P	researched material	results research	Graphic image

Informational material to topic

Of the 14 species of staphylococci that live on the skin and mucous membranes of humans, prevail and often cause diseases: S.aureus, S.epidermidis, S.saprophyticus. Staphylococci are Gram-positive cocci, non-motile, do not form spores or capsules. smears are arranged in clusters in the form of "clusters of grapes". **Cultural properties** . Not demanding on nutrient media: cultivated on MPA with formation of pigmented yellow colonies or white, in the MPB they give diffusely turbid growth. Character is important for identification of staphylococci growth on blood agar (hemolysis zone) and yolk-salt agar (YSA) (determinationlecithinase).
Biochemical properties . Staphylococci break down carbohydrates into acids. Important a differentiating feature of various types of staphylococci is the formationacids from mannitol in aerobic and anaerobic conditions.
pathogenicity factors .1.
Factors adhesion:
-teichoaceae acids provide adhesion on the cells organism;
"hospital strains" S. epidermidis produce a special kind of mucus, ensuring their attachment to polymeric materials of catheters, artificial valves hearts and creation on the them bacterial biofilms. it going on to developmentsepsis and endocarditis, conditioned " hospital strains" S. epidermidis.
2.Protein A non-specific binds Fc fragment IqQ that leads to oppressionphagocytosis, functions complement and opsonizing actions antibodies.
3.Eclipse antigens, having antigenic commonality With cells skin and kidneyperson.
one. Enzymes pathogenicity:
– hyaluronidase, splits hyaluronic acid in composition connective fabrics, what promotes dissemination

staphylococci;
-plasma coagulase causes clotting proteins serum blood, forming fibrin "pseudocapsule" protecting staphylococci from phagocytosis
-Plasmocoagulase is one of the important markers of various types of staphylococcifor differentiation. S.aureus has plasmacoagulase and belongs to coagulase-positive staphylococci; S.epidermidis and S. Saprophyticus do not have plasma coagulase and are to coagulase-negative (KOS).
fibrinolysin breaks down fibrin and promotes the breakdown of staphylococci intobody;
-lecithinase destroys lipid membranes cells organism;
nucleases (RNases, DNases) cleave DNA molecules and RNA, which leads todestruction synthesis squirrel in cells and their doom;
-β-lactamase destroys -β-lactam antibiotics (penicillins, cephalosporins).5. **Exotoxins:**
-hemolysins 4th types, in mostly possessing hemolytic and cytotoxicaction;
-leukocidin destroys leukocytes;
exfoliatins cause damage and detachment of the epidermis with accumulation of fluid and formation of blisters, causing the development of the syndrome of "scalded skin" (syndrome Lyell);
-toxic shock exotoxin (ECT) causes systemic damage to the body in the form ofsyndrome toxic shock (STSH) With high lethality;
-enterotoxins cause symptoms acute food poisoning. All toxins,Besides hemolysins, produces only S. aureus.
6.R- **_ plasmids (** factors multiple medicinal sustainability **).**
S.aureus - ubiquitous, part of the facultative microflora of the skinand mucous nasal membranes and nasopharynx.
Sources infections are sick human and bacteriocarrier. Often formedcarrier status medical staff.
Ways of infection: airborne, contact, alimentary. In individuals with reducedresistance possible endogenous way of infection.
Nosological forms of infections caused by S.aureus are diverse, because are amazedany fabrics and organs.

S.epidermidis colonizes the skin and mucous membranes. Most often causes nosocomial, iatrogenic infections: sepsis, endocarditis, urological infection,what is associated with the colonization of artificial heart valves by these microorganisms, catheters vascular prostheses.

S. Saprophyticus colonizes mucous shells urogenital tract and causesinflammation of various sections of the urinary tract in people with low resistance.

Main nosological forms staphylococcal infections

Forms diseases	Material for research
Local	
Purulent defeat skin (boils, carbuncles, abscesses.Phlegmon)	Purulent detachable, purulent content
mastitis	breast milk, pus from abscess
Angina, tonsillitis	Smear from pharynx, With tonsils
Pneumonia, bronchopneumonia	Sputum, flushing water bronchi, blood

Arthritis	articular liquid
Conjunctiva	Purulent detachable conjunctiva
infections urinary ways	Urine
food poisoning	Flushing water stomach, emetic masses, faeces, leftovers food
Generalized	
Sepsis	
Endocarditis	
Meningitis	
Hematogenous osteomyelitis	
Syndrome toxic shock (STSH)	Detachable from vagina, blood

specific treatment staphylococcal infections

Acute staphylococcal infections	Chronic staphylococcal infections
Immunoglobulin staphylococcal human	Anatoxin staphylococcal purified liquid
Staphylococcal bacteriophage	killed staphylococcal vaccine, chemical staphylococcal vaccines on the basis protective antigens

streptococci - gram-positive cocci, motionless, dispute and capsules not form, insmears arranged in chains.

cultural properties . Streptococci are demanding on nutrient media. AT sugar broth give near-wall type of growth. On blood agar they formsmall convex colonies. Optional anaerobes.

By character growth on the bloody agar allocate 3 groups streptococci:

1) α- hemolytic form around colonies zone greening ("greenstreptococci") in result transformation hemoglobin in methemoglobin;
2) β-hemolytic cause full lysis erythrocytes and form aroundcolonies transparent zone;
3) γ-streptococci do not cause hemolysis and are non-homolytic. **Biochemical properties** .

When identifying streptococci, their ability toferment carbohydrates, grow on media with bile, as well as on environments with high concentration NaCI and reduce in methylene blue in milk.

Antigenic structure . By antigenic structure (polysaccharide antigens of cellwalls) R. Lensfield divided streptococci into 20 serogroups - A, B, C, etc. To streptococcus group A include - S.pyoqenes (β-hemolytic - streptococcus), most pathogenic species.

α-hemolytic streptococci are mostly part of the normal microflora("oral streptococci", enterococci), but can cause pathology in humans when decline residence of the body.

Non-hemolytic streptococci are included in compound obligate microflora mucousshells human and usually not cause pathological processes.

The most epidemiologically significant for humans is the species S. pyoqenes, possessing significant a set **of pathogenicity factors:**

1. Factors adhesion : lipoteichoic acid cellular walls;
2. Protein M provides not only adhesion but and suppression phagocytosis;
3. Eclipse antigens having antigenic commonality With cloth hearts and kidneys.

Enzymes pathogenicity:

- hyaluronidase - promotes displacement microbes on connective fabrics;
- fibrinolysin (streptokinase) - causes the dissolution of fibrin thrombi,promotes dissemination by circulatory channel;
-DNA-aza- destroys molecules DNA.

Exotoxins :

-hemolysins (O- and S-streptolysins) - have hemolytic and cytotoxicaction on the cardiomyocytes and phagocytes;

-erythrogenic (pyrogenic) - lead to the formation of rashes on the skin, havepyrogenic action, cause development syndrome toxic shock.
Source infection :sick human and bacteriocarrier.
Ways of infection: airborne, contact, for S aqalactiae - intranatal(in time of birth).
The main method of microbiological diagnosis of streptococcal infectionsis bacteriological.

PROTOCOL RESEARCH

Stage (day research)	move research	Result
1st	Microscopy smears from pus, dyed according to Gramu Sowing in cups with bile-salt agar	Among leukocytes visible Gr + cocci, locatedsmall bunches andalso alone and in pairs. colony growth medium sizes with clouding around colonies and rainbows whisk
2 - th	Microscopy of smears from selected colonies, Gram-stained Screening of colonies with rainbowwhisk on the oblique agar	AT field view visible Gr + cocci locatedforms
3rd	Dedicatedclean culture. Definition signs pathogenicity: a) smear microscopy, painted on Gram; b) inoculation on Hiss media withmannitol and glucose anaerobic and anaerobicconditions; c) definition hyaluronidase activity, plasmacoagulation, DNA-basics; d) definition of α-hemolysin on plates bloody agar; e) phage typing.	

	Sensitivity testto antibiotics method paper disks.	
4th	Conclusion on carried out research	Highlighted culture pathogenic staphylococcus. Fagotype is --- sensitive to next antibiotics

Scarlet fever is an acute infectious disease, manifested by a small punctate rash, fever, general intoxication, tonsillitis. The causative agent of the disease - group streptococcus A (Streptococcus pyogenes). Infection occurs from airborne droplets (at cough, sneezing, conversation), a also through items everyday life (tableware, toys, underwear). Especially dangerous sick how sources infections in first days illness.

Sources infectious agent are sick scarlet fever or any another clinical form streptococcal infections and bacteriocarrier. More often get sick children 3-10 years, visiting children's preschool institutions and school. appearance cases scarlet fever in children's institutions, how rule preceded increased incidence of tonsillitis and acute respiratory viral infections. Children first of the year life (especially first half a year) and adults rarely get sick with scarlet fever. The main route of transmission of the pathogen is airborne drip.

Pathogenesis

Pathogen penetrates in organism human through mucous shells pharynx and nasopharynx, in rare cases, infection through the mucous membranes of the genital bodies or damaged skin. AT place adhesion bacteria formed local inflammatory-necrotic hearth. Development infectious-toxic syndrome conditioned in first turn admission in blood flow erythrogenic toxin streptococci (toxin Dick), a also action peptidoglycan cellular walls. Toxinemia leads to a generalized expansion of small vessels in all organs, including in the skin and mucous membranes, and the appearance of a characteristic rash. Synthesis and accumulation antitoxic antibodies in dynamics infectious process, binding them toxins in subsequent condition decrease and liquidation manifestations toxicosis and gradual disappearance rash. Simultaneously develop moderate phenomena perivascular infiltration and edema dermis. Epidermis impregnated exudate, his cells exposed keratinization, what in further leads to peeling of the skin after the extinction of the scarlatinal rash. Maintaining a strong connections between keratinized cells in the thick layers of the epidermis on the palms and soles explains large-lamellar character peeling in these places.

Components cellular walls streptococcus (group A-polysaccharide, peptidoglycan, protein M) and extracellular products (streptolysins, hyaluronidase, DNA- aza, etc.) cause the development of delayed-type hypersensitivity reactions, autoimmune reactions, formation and fixation of immune complexes, disorders systems hemostasis. In many cases them can count cause development glomerulonephritis, arteritis, endocarditis and others complications immunopathological character.

From the lymphatic formations of the mucous membrane of the oropharynx, pathogens lymphatic vessels enter the regional lymph nodes, where theyaccumulation, accompanied by the development of inflammatory reactions with foci of necrosis and leukocyte infiltration. Subsequent bacteremia may in some cases lead to penetration microorganisms in various bodies and systems, formation purulent-necrotic processes in them (purulent lymphadenitis, otitis, defeats bone fabrics temporal areas, solid cerebral shells, temporal sinuses and etc.).

Scarlet fever should be distinguished from measles, rubella, pseudotuberculosis, medicinal dermatitis. In rare cases development of fibrinous raids and especially when they outletper limits tonsils disease necessary differentiate from diphtheria.

Scarlet fever is distinguished bright spilled hyperemia oropharynx ("flaming yawn"), sharply limited at the point of transition of the mucous membrane to the hard palate, bright red language raspberry and hypertrophied papillae ("crimson language"), punctate elements of the rash on a general hyperemic background, thickening of the rash in the form dark red stripes on the skin folds in places natural folds, distinctly expressed white dermographism, pale nasolabial triangle (symptom Filatov). When pressing on the skin with the palm of your hand, the rash in

this place temporarily disappears ("symptom palms"), positive endothelial symptoms. After disappearance exanthems note finely scaly peeling skin (on the palms and soleslarge plate).

laboratory diagnostics

Diagnosis scarlet fever based on the clinical (acute Start diseases, fever, intoxication, acute catarrhal or catarrhal-purulent (with septic form of illness - necrotic), tonsillitis, profuse punctate rash, thickening into natural skin folds and laboratory (neutrophilic leukocytosis, increased ESR, abundant growth of beta-hemolytic streptococci when sowing material from the focus infections on blood agar, an increase in antibody titers to streptococcal antigens - M-protein A-polysaccharide, streptolysin-O and others) data.

1. When a white mouse is infected with the sputum of a patient with pneumonia, the mouse dies from pneumococcal sepsis. From organs of the deceased mice prepare smears-imprints. paint on Gram. On the pink background, educated cells fabrics, are found gram-positive diplococci slightly elongated forms, reminiscent of contours flame candles or lancet surrounded colorless capsule.

2. A characteristic feature of pneumococci that distinguishes them from most other species of streptococci, is related to bile and bile salts. Bile is not only kills, but and dissolves pneumococci. Against, in difference from verdant (S.faecalis, S.sanguis) and hemolytic streptococci (S.pyogenes), pneumococci decompose inulin.

3. Diagnosis acute gonorrhea put With help microscopic method research. From researched material make two strokes, one color by Gramu, another - methylene blue. At availability in smear gonococci visible gram negative diplococci, located inside leukocytes (unfinished phagocytosis).

4. So how at chronic gonorrhea gonococci are outside cells, have atypical form in form balls or very small entities, use bacterioscopic method for productions diagnosis is not possible. Therefore, in order to diagnose chronic gonorrhea apply: bacteriological, serological methods research.

Serological diagnosis of gonorrhea is made with the help of RSK. The reaction is set for detection antibodies in serum blood sick, With help famous antigen, which the represents yourself suspension killed gonococci.

Scheme productions RSK

Components reactions	1st (an experience)	2nd (control AG)	3rd (control AT)
one. Serum under study (1:5)	0.5	-	0.5
2. Antigen in working dose	0.5	0.5	-

3. Complement in working dose	0.5	0.5	0.5
four. Physiological solution	-	0.5	0.5
on the 45 minutes in thermostat			
5. Hemolytic system	1.0	1.0	1.0
on the 45 minutes in thermostat			
Accounting results		hemolysis	hemolysis

Accounting result reactions start With control tubes. At availability hemolysis in control test tubes about results reactions judge but experienced test tube.

INFORMATIONAL MATERIAL To TOPIC:

Meningococci (Neisseria meningitides) are Gram-negative bean diplococci. forms, flagella and dispute is not have, in body form a capsule.

Cultural properties . Highly demanding to conditions cultivation. grow up on the dense and liquid nutritional environments containing 20-25% serum (serum agar, whey broth). On a dense medium they form small smooth clear colonies. Strict temperature optimum - 37 ° C (at other temperatures meningococci die) necessary create how at cultivation, So and at transportation material from the patient to laboratory.

Among representatives kind Neisseria there is conditionally pathogenic kinds, inhabitants mucous shells nasopharynx - N. Sicca, N. mucosa and others At of people With weakened resistance they may call diseases clinically similar With meningococcal infection.

Antigenic structure. N meningitides has generic antigens common to all types. Within the species by capsular polysaccharide antigens distinguish between serogroups N meningitides- A,B,C,D,YZ and etc.

epidemiological outbreaks more often cause pathogens serogroups A, B, C.

Factors pathogenicity meningococci :

1. Pili - provide adhesion on the cells of the cylindrical epithelium of the nasopharynx. 2. Ig BUT- proteases - split molecules SIg BUT, lowering topics most local protection mucous membranes of the nasopharynx; 3.

Capsule - protects from phagocytosis;

4. Enzymes pathogenicity : hyaluronidase, neurominidase and others

5. Endotoxin (cell wall LPS) - causes damage to blood vessels, which is manifested by hemorrhages in the internal organs and a hemorrhagic rash on skin.

The source of infection is a sick person, or a bacteriocarrier. More often (in70-80% cases) sick children first three years of life.

Ways infections - airborne. Input gates infections - mucous shell nasopharynx. Meningococcal infection maybe leak in several clinical forms, which share on the localized and generalized.

Main clinical forms meningococcal infections and material for microbiological research

FORMS	DISEASES	MATERIAL FOR

		RESEARCH
Primary localized	meningococcal carriage	smear from nasopharynx
	Spicy nasopharyngitis	
Hematogenou sgeneralized	Meningococcemia	Smear from nasopharynx, blood
	Epidemiological cerebrospinal meningitis, meningoencephalitis	Smear from nasopharynx,blood. liquor

Microbiological diagnostics **meningococcal infections.**

1. Bacteriological method (basic) - isolation of pure culture pathogen on the serum environments and definition his antibiotic sensitivity.
2. Bacteriological method - uses how required indicative. AT smears from native material With coloration on Gram stands out intracellular location bacteria and characteristic painting unfinished phagocytosis meningococci.

Specific prevention of meningococcal infection are carried out only on epidemiological testimony meningococcal polysaccharide vaccine serogroups BUT and S.

Gonococci - (N. Gonorrhoeae) - gram-negative bean-shaped diplococci, form capsule in organism, flagella and the dispute is not have.

cultural properties . Demanding on nutrient media and temperature optimum - 37 FROM. Require freshly prepared wet nutritious environments With adding native proteins blood, serum, or ascitic fluid. Don't call hemolysis on media containing blood, on media containing milk, gelatin and potatoes not are growing.

Gonococci are characterized by pronounced antigenic variability, even within one strain.

Biochemical properties: decompose only glucose With education acids. Proteolytic activity missing, ammonia, hydrogen sulfide and indole not form.

Factors pathogenicity gonococos :

1. Pili - provide adhesion to the cells of the cylindrical epithelium urinary ways;
2. Capsule - in freshly isolated cultures, it has antiphagocyticaction;
3. Cellular wall contains endotoxin.
4. Surface protein one classes causes to bactericidal factors;
5. Surface protein 2 class forms separate protein fraction called turbidity proteins or Ora proteins (turbidity). Them consider first factors virulence gonococci, and they cause attachment to the epithelium.
6. R- plasmids multidrug resistance factors.For diagnostics are used:

Bacteriological method (basic) - isolation of pure culture pathogen on serum media and determination of its antibiotic sensitivity. Gram stain and characteristic pattern _ unfinished phagocytosis gonococci.

The serological method is used for chronic gonorrhea, in the absence of the patient's discharge. Carry out RSC on Borde-Jangu according to the standard scheme, which happens positive With 3-4 weeks. AT quality antigen for RSK apply gonovaccine or antigen from killed gonococci.

Genetic method - determination of sections of the gonococcus genome in the material from sick With using PCR.

For the **specific treatment of** chronic forms of gonorrhea, killed gonococcal vaccine.

Pneumococci - Streptococcus pneumoniae Gram-positive diplococcus, usually lanceolate or arranged in chains, having a polysaccharide capsule,which allows easily " type" them specific antisera. pneumococci motionless, dispute not form; optional anaerobes. At cultivation on artificial nutrient media lose the capsule, move from S-into an R-shape. They grow well on blood and serum media. When growing on agar with ram blood form colonies with zone α partial hemolysis and greening of the medium, β full hemolysis, γ-hemolysis visually invisible hemolysis.

Enzymatic activity glucose With education dairy acids.

Pneumococcus does not contain group antigen serologically heterogeneous in AG capsular polysaccharides are isolated 84 serovar.

With pneumococcal infection in order to isolate a pure culture of the pathogen put bioassay - intraperitoneally infect white mice material from sick.

situational tasks :

Microscopic examination of the detachable furuncle revealed S. aureus. To whichgroup representatives normal microflora skin applies this microorganism?

On the what from listed Wednesdays grow up staphylococci and kaike environments appropriateuse at bacteriological research?

a) BCH or MPA;

b) milk-salt agarin) bile

broth

G) blood agar

e) sugar agar or brothe) bile-

salt agar

What medium is selective for staphylococci? What makes selectivitythis environment?

What are the features of the structure of the cell wall of staphylococci in comparison withstructure cell wall Gram-negative bacteria?

What pathogenicity enzymes are produced by staphylococci? What is their role in pathogenesis infections?

What kind cells, Besides erythrocytes, maybe damage £ - toxin?

What explains the cardiotoxic, dermatotoxic and neurotoxic effects of α -toxin?

Questions for self-control:

1. Explain the origin of hemolysis zones. How to distinguish pneumococci – β hemolytic –from α – hemolytic?
2. List the possible methods of laboratory diagnostics for gonococcalinfections.
3. name main factors pathogenicity meningococcus.
4. Who is main source meningococcal infections?
5. List factors pathogenicity for meningococcus.

PRACTICAL OCCUPATION No. ten.

TOPIC: Microbiological diagnostics bacterial infections. Working offmethods diagnostics on the example the following pathogens:

1. corynebacteria, actinomycetes, listeria (microscopic andbacteriological methods)

2. anaerobic bacteria (microscopic, bacteriological methods)

Educational goal:

1. Train students methods microbiological diagnostics and specific prevention diphtheria, whooping cough
2. will study laboratory diagnostics actinomycete.

3. Explore modern methods microbiological diagnostics diseases,caused by anaerobes.
4. Explore drugs for specific prevention and therapy anaerobicdiseases.

Student must know:

1.Biological properties and laboratory diagnostics diphtheria, whooping cough
2. specific prevention diphtheria, whooping cough
3.Peculiarities morphology, tinctorial and cultural properties, biochemicalactivity.
4.Pathogenicity factors: toxins and their significance in the pathogenesis of anaerobic infections.
5.Distribution, source infection, way transmission diseases called atperson.
6.Microbiological diagnostics: bacterioscopic, bacteriological method, bioassays.
7.Specific prevention and treatment.

Student must be able to:

1.cook smear and paint on method Neisser.
2. cook smear and paint on method Gram.
3.Define toxigenicity diphtheria cultures on Ouchterlony.
4. Put sample on the cystinase and sample on the urease diphtheria and false - diphtheriasticks.
5.Conduct bacteriological research clean culture (on scheme).6.Cook swab and paint by Gram.
7.Smear microscopy. 8.Spend accounting results.

PLAN:

1. Taxonomy and basic biological properties of diphtheria pathogens , whooping cough
2. Epidemiology, pathogenesis, immunity called diseases.
3. Principles of microbiological diagnosis of diphtheria, whooping cough, Preparations for etiotropic therapy and specific prevention diphtheria, whooping cough
4. Modern ideas about the etiology of anaerobic infection. Clostridial and non-clostridial anaerobic infection.
5. Morphological, cultural and biochemical properties pathogens anaerobic infections: Clostridium (gas gangrene, tetanus, botulism), peptostreptococcus, bacteroids, fusobacteria, anaerobic vibrios, campylobacter and spirilla.
6. Pathogenetic Aspects anaerobic infections: primary exogenous and secondary, endogenous. Mechanisms occurrence. Opportunistic anaerobic and mixed infections.
7. Methods microbiological diagnostics anaerobic infections.
8. Principles specific prevention anaerobic infections. Preparations for active and passive immunization.
9. Principles specific therapy anaerobic infections. Etiotropic and pathogenetic therapy: antibacterial, hyperbaric oxygenation and etc.

INDEPENDENT WORK

1. Cooking smear and coloring on method Neisser.
2. Cooking smear and coloring on method Gram.
3. Definition toxigenicity diphtheria cultures on Ouchterlony.
4. Holding samples on the cystinase and on the urease diphtheria and false diphtheria sticks.
5. Microscopic method for the diagnosis of gas gangrene: the study of smear-imprint from a festering wound coloring by Gram.
6. Bacteriological method diagnostics anaerobic infections:
   1-th stage - the study on the 5% bloody agar isolated colonies bacteroids and peptostreptococcus, dedicated from purulent exudate.
   Further- receiving clean culture anaerobic bacteria in semi-liquid environment AS.

Demonstration selective Wednesdays for cultivation anaerobes: Kitta Tarozzi, "high" column sugar agar.

2-th stage - identification of a pure culture of anaerobic bacteria by biochemical properties With using test systems AP1-Ap (principle "variegated row").

7. Definition sensitivity anaerobic bacteria to antibiotics (micromethod). Demonstration of the results of inoculation of a pure culture in a microcassette withantibiotics.
8. Description drugs for specific prevention clostridial anaerobic infections: tetraanotoxin gas gangrene, pentaanotoxin (+tetanus toxoid), tetanus toxoid component ADS drugs and vaccines DPT, TABte.
9. Description drugs for specific therapy clostridial anaerobic infections: polyvalent antigangrenous serum, antitoxic tetanus toxoid serum, antitoxic monoclonal and polyvalent antibotulinum serum.
10. Decor protocol research.

METHODOLOGICAL RECOMMENDATIONS

diphtheria wand (Corynebacterium diphtheriae) — gram-positive rod-shaped bacteria of the genus Corynebacterium. The pathogen was first discovered on sections films, received from oropharynx sick in 1883 G. Edwin Klebs (German Edwin klebs, 1834-1913). Through year Friedrich Loeffler (German Friedrich august Johannes Loeffler, 1852-1915) was highlighted clean culture. diphtheria toxin received by E. Roux and A. Yersen (1884-1888). Anatoxin discovered by Ramon Gaston in 1923 G. and proposed use his for active immunization. Corynebacterium diphtheriae

- large (1-8 × 0.3-0.8 microns) straight, slightly curved polymorphic rod-shaped bacteria. Metachromatic grains of volutin are localized at the poles of cells, givingcells characteristic form "maces". grains volutin stained methylene blue on Neisser. On the micropreparations are located alone or due to features of cell division are arranged in the form of the Latin letter V or Y. Dispute and capsules not form.

Epidemiology. Humans are the source of infection in diphtheria - sick or healthy carriers toxigenic diphtheria microbes. the greatest epidemic danger is posed by patients with diphtheria of the pharynx, nose and larynx, activelyhighlighting pathogens diseases in external Wednesday With exhaled air. Minor in this respect meaning play sick diphtheria eye, skin, woundsand other localizations capable of spreading the infection by contact (through arms, Houseware).

Pathogenesis. The entrance gate of diphtheria pathogens can be practically all areas of the integument (skin and mucous membranes) of the macroorganism. However, most often they is mucous shell oropharynx, much less often - larynx, nose, conjunctiva genital bodies, wound surface, leather and others Toxigenic corynebacteria fixed on the cells fabrics, multiply and in process vital activity produce an exotoxin that has a local and general effect, causing almost all manifestations of the pathological process. microbial cells outside fabrics, being gate infection, how rule not spread and direct participation in defeat macroorganism not accept.

diphtheria exotoxin consists from several factions, each from which has independent biological action. One from them - hyaluronidase: destroys hyaluronic acid capillaries and increases their permeability. This leads to exit per limits vessels liquid parts blood, impregnation affected fabrics plasma, containing along with With others components fibrinogen. Second - necrotoxin

- causes necrosis of the epithelium at the site of the infection gate, accompanied by the release of epithelial cells thrombokinase. Last promotes transformation fibrinogen into fibrin and the formation of fibrin on the surface of the affected tissues films. Palatal tonsils, in difference from others bodies, covered multi-row epithelium. AT result emerging at diphtheria fibrinous film penetrates deep inside epithelial cover and tight soldered With tissues. Third fraction diphtheria toxin - true diphtheria toxin (basic his component) able displace from cellular structures cytochrome B and so way block in them processes cellular breathing and synthesis protein molecules. Most sensitive to these changes are the myocardium, capillaries and nerve cells. AT cardiomyocytes develop the phenomena of myocardial dystrophy with their subsequent necrosis, myolysis and development of infectious-toxic myocarditis. Capillary damage with diphtheria, it

is accompanied by infectious-toxic shock. Nerve damage cells accompanied dystrophic changes Shvanovsky cells and demyelination of nerve fibers. Along with the noted, the general effect of diphtheria toxin manifested by the phenomena of the general intoxication.

basis laboratory diagnostics constitute bacteriological research: selection pathogen from hearth inflammation, definition his type and toxigenicity. Material take away sterile wadded tampons dry or wetted (before sterilization!) 5% solution glycerin. At storage and transportation tampons protect from cooling and drying. Material must to be sown not later 2-4 h after taking. In patients with angina who were in contact with patients with diphtheria, as well as in persons With typical clinical manifestations diphtheria diagnosis put even at negative result bacteriological research.

Of secondary importance is the determination of titers of antitoxic antibodies in paired sera at staging RNGA. toxin formation reveal using RNGA with antibody erythrocyte diagnosticum. For identifying diphtheria toxin suggested use PCR.

Main in treatment diphtheria consider introduction antitoxic diphtheria serum. She is neutralizes toxin, circulating in blood, Consequently, renders greatest effect at an early application

Preventive Events. Vaccination remains main way to control diphtheria. The Children's Immunization Scheme includes immunization DTP vaccine starting from 3 months of life (vaccinated 3 times with an interval of 30-40 days). Revaccination is carried out 9-12 months after the completed vaccination. For revaccinationin 6-7, 11-12 and 16-17 years apply ADS-M. AT individual cases, for example at contraindications to whooping cough component DPT, ADS-M apply and for vaccination.

Whooping cough (wooping-cough - English; Keuchhusten - it; Coqueluche - French) and parapertussis - acute infectious diseases, clinically indistinguishable from each other. Characterized sharp Qatar respiratory ways and attacks spasmodic cough.

The causative agent of whooping cough (Bordetella pertussis) is a short stick with rounded ends (0.2-1.2 μm), gram negative motionless, Good staining aniline paints. AT antigenic respect heterogeneous. The antigen that causes the formation of agglutinins (agglutinogen) consists of multiple components. They are called factors and are numbered from 1 to 14. Factor 7 is generic, factor one contains AT. pertussis, fourteen - AT. parapertussis, the rest meet in different combinations; for the causative agent of whooping cough, these are factors 2, 3, four, 5, 6, for parapertussis - eight, 9, ten. Reaction agglutination With adsorbed factor sera allows you to differentiate the types of bordetella and determine them antigenic options. pathogens whooping cough and parapertussis very unstable in external environment, so sowing should be done immediately after taking the material. bacteria fast perish at drying ultraviolet irradiation, under influence disinfectants funds. sensitive to erythromycin, chloramphenicol, antibiotics tetracycline group, streptomycin.

Pathogenesis. Gates infections is mucous shell respiratory tract. whooping cough microbes attached to cells flickering epithelium, where they multiply on the surface of the mucous membrane, without penetrating into the bloodstream. On site implementation pathogen develops inflammatory process, oppressed activity ciliary apparatus of epithelial cells and increased secretion of mucus. AT further there is ulceration of the epithelium of the respiratory tract and focal necrosis. Pathological process most pronounced in bronchi and bronchioles, less pronounced changes develop in the trachea, larynx and nasopharynx. Mucopurulent plugs clog clearance small bronchi, develops focal atelectasis, emphysema. Observed peribronchial infiltration. AT genesis convulsive seizures It has meaning sensitization organism to toxins pertussis sticks. permanent irritation receptors respiratory ways conditions cough and leads to formation in respiratory center hearth arousal type dominants. Due to this typical seizures spasmodic cough may to be caused and non-specific irritants. From the dominant focus, excitation can radiate to other departments nervous systems, for example on the vasomotor (increase HELL, spasm vessels). irradiation arousal explained also appearance convulsive

contractions of the muscles of the face and trunk, vomiting and other symptoms of whooping cough. Postponed whooping cough (how and pertussis vaccinations) not provides tense lifelong immunity, so recurrence of whooping cough is possible (about 5% cases whooping cough falls on adults of people).

Reliable diagnosis in catarrhal period maybe to be staged after receiving results bacteriological research. foundation for research in these cases usually serve epidemiological data (contact With sick whooping cough absence data about vaccinations and etc.). AT period spasmodic cough diagnosis whooping cough is much easier to deliver, as typical attacks appear. However need take account of, what sometimes seizures cough, similar With pertussis, may to be conditioned others reasons (adenoviral infection, viral pneumonia, compression respiratory ways at malignant neoplasms, infectious mononucleosis and etc.), With another hand, whooping cough maybe leak atypical without characteristic seizures (at vaccinated children, at adults). Main method laboratory confirmation diagnosis is selection pathogen whooping cough Frequency allocation depends from timing taking material; on the 1st week diseases positive results can be obtained in 95% of patients, on the 4th - only in 50%, and starting from the 5th week, the microbe can no longer be isolated. The material is taken from the nasopharynx dry swab with immediate inoculation on plates with selective nutrient medium. The "cough plates" method is also used, in which a Petri dish with nutrient the medium is placed in front of the mouth of the coughing child (at a distance of about 10 cm), held in this position for several seconds to catch 5-6 coughing shocks. Cup With sowing fast close lid and put in thermostat. At transportation is protected from cooling (wrapped in paper, cotton wool, in a container put heating pad completed hot water). However on frequency allocation pathogens whooping cough method "cough records" much inferior taking swab material. Serological methods can be used for retrospective diagnostics, a also at sick With negative results bacteriological research. From old methods can use RSK, RPGA, reaction agglutination. An increase in antibody titers by 4 times or more is considered diagnostic, and also high antibody titers (1:80 and above).

AT last thing time successfully use enzyme immunoassay method for detection of antibodies in serum (class M immunoglobulins) and in nasopharyngeal mucus (immunoglobulins class BUT). These antibodies appear co 2nd-3rd weeks disease and persist in flow 3 months

1. Microscopic method for diagnosing gas gangrene. In a smear-print from purulent wound (Gram stain) shows purple rod-shaped cells colors.

2. Bacteriological method diagnostics anaerobic infections.

1-th stage. The first day . On the 5% bloody agar in cup petri (after cultivation in an anaerobic balloon: 80% N_2, 10% H_2, 10% CO_2) several species are determined isolated colonies, including those with various types of hemolysis (α, β) and pigment (for example, black pigment bacteroids groups melaninogenicus). Second day. AT in a test tube with a pure culture of peptostreptococci in a semi-liquid medium, AS are observed small white granules at the bottom parts of the test tube with the medium. In the control of cleanliness isolated culture (gentian violet staining), chains of elongated cocci blue colors.

2-th stage. AT test system API-An for identification pure cultures on biochemical properties determined fermentation glucose (change coloring indicator in yellow) in the absence of other manifestations of glycolytic, as well as proteolytic activity (negative samples for indole and hydrogen sulfide).

3-th stage. At definition sensitivity anaerobic bacteria to antibiotics in microcassette (after cultivation in anaerostat) are celebrated positive and negative options results.

4- stage . When studying ampoules with drugs for specific prophylaxis and therapy of anaerobic infections, the goals (prevention, treatment) are noted in the protocol, character immunization (active or passive, antitoxic or antibacterial), testimony to application and peculiarities use everyone drug.

PROTOCOL RESEARCH

№№ P/P	researched material	results research	Graphic image

one.	Smear-imprint from a festering wound.Coloring on Gram.		

Informational material

Tetanus heavy wound infection.

Morphology Gram-positive rods with rounded ends. located alone or chain. Spores are located terminally.

Cultural properties obligate anaerobe. On the MPA and gelatin in strictly anaerobic conditions pathogen growing slowly and forms thin transparent colonies. When sown in a column in semi-liquid agar, it forms colonies in 24-48 hours in form "lentils" R-shape or "fluff" S shape.

Pathogenicity factors are exotoxins tetanospasmin and tetanolysin.

Antigenic structure -O and H antigens.

Immunity. There is no natural immunity in humans to tetanus.Diagnostics: bacterioscopic, bacteriological and biological.

Treatment is aimed at neutralizing the tetanus toxin with toxoid. Apply tetanus toxoid horse serum in dose 50-100 thousand ME.

Prevention - surgical treatment of the wound. Creation of an artificial active immunity in planned okay vaccination DPT, ADSm. Primary vaccination carry out children in 3- monthly age.

Clostridia botulism

Botulism - acute food toxic infection, flowing predominantdefeat central and vegetative systems.

Morphology- sticks With rounded ends, mobile, peretrichia. controversylocated subterminally.

Cultural properties - strict anaerobes. Good are growing on the environments kitta-Tarozzi, bouillon from meat fish. calls turbidity environments and gas formation.

All types clostridia botulism form hydrogen sulfide.

The antigenic structure has group-specific (H) flagella andtype-specific somatic (O) antigens.

Factors pathogenicity - botulinum toxin protein, showing neurotoxicaction. Botulinum toxin is most strong poison known to a person.

Immunity. Natural immunity human missing.

Treatment. For treatment on Bezredko sick i/v introduce one internationalmedical dose (on 10000 IU serum types BUT and E and 5000 IU type AT).

Prevention. For emergency prevention used polyvalent (types A, B, E) equine serum.

Clostridia gas gangrene.

Anaerobic wound infection (gas gangrene, anaerobic myositis) - heavy wound infection human and animals, called bacilli kind Clostribiumperfinqens.

Morphology. Vegetative cells are large, gram-positive, immobile. Classic forms submitted under direct angle ends. AT body form capsules, they most pronounced in virulent strains. Resistant to phagocytosis.

cultural properties. On dense media, C Perfiinqens of type A forms S and R - round colonies. S - dome-shaped colonies, with smooth even edges. R - colonies wrong forms edges; in depth agar remind lumps cotton wool.

Growth on liquid and semi-liquid nutrient media, especially those containing glucose, very rapidly with the formation H2 and CO2 and usually ends after 8-12 hours Turbidity of the medium and active gas formation can be observed after 4-8 hours cultivation.

Biochemical activity- splits With education acids and gas glucose,sucrose, maltose, lactose, mannose, starch.

Proteolytic activity weak; liquefies gelatin, intensively curdle milk.

Antigenic structure - all serovars form α-toxin (lecithinase). Pathogen forms at least 12 identifying toxins and enzymes that play a role in pathogenesis gas gangrene.

Clostribium perfinqens is widely distributed in the environment; it is isolated from water, soil, sewage. Spores can persist in the environment for a long time.environment, able vegetate in soil. controversy distinguishes high sustainability to chemical and physical influences.

situational tasks

1.Answer the test question: select the media on which clostridia are cultivated:a) iron sulfite milk
b) a high column of sugar MPAin)
Wednesday Endo, Levina
d) Wilson-Blair
environmente) bile broth
e) blood agar
2.What methods of laboratory diagnostics can you note for gas gangrene andtetanus, proceeding from from knowledge pathogenesis, clinical paintings and conditions infections?
3. It is important to know that in the pathogenesis of diseases caused by gas gangrene and tetanus, main role belongs produced them toxins and enzymespathogenicity.
4.name them, give brief characteristics them properties.
5. Taking into account this fact, suggest drugs for a specific prevention and treatment anaerobic infections, caused gas gangrene andtetanus.

test control

1.Methods used for staining diphtheria bacillus:BUT) Gram's method
B) Neisser method
AT) method Ozheshko
G) Method Tsilya –Nelsen
2.Biological variants of diphtheria bacillus:BUT) Gravis
B) Mitis
G) Intermedius
3.What kind associated drugs use for preventiondiphtheria, whooping cough
BUT) DPT
B) typhoid vaccine With tetraanatoxin.

Task #1

When examining for diphtheria carriage from the pharynx of a kindergarten teacherisolated a microbe with the following properties: grains of volutin found in individual individuals, sucrose, glucose, starch does not break down, sampleson the cystinase and urease are negative.

PRACTICAL OCCUPATION No. 11

TOPIC: Microbiological diagnostics bacterial infections. Working offmethods diagnostics For example the following pathogens:
1. pathogens intestinal infections (bacteriological, serological methods)
2. pathogens STI (serological, molecular biological methods)

Learning goal: to train students in the methods of microbiological diagnostics andspecific prevention of intestinal diseases.

Student must know:

1. Biological properties and laboratory diagnosis of cholera. 2.Biological properties and laboratory diagnostics dysentery.3.Express diagnostics cholera. four. specific prevention cholera and dysentery.

Student must be able to:

1.Record and interpret the results of rapid diagnostics of cholera.2. Carry out bacteriological diagnosis of dysentery: do sowing on differential diagnostic Wednesday Ploskirev.

PLAN:

1. Taxonomy and main biological properties of pathogens intestinal escherichiosis and intestinal yersiniosis.
2. Epidemiology, pathogenesis, immunity called diseases.
3. Principles of microbiological diagnosis of intestinal escherichiosis andintestinal yersiniosis.
4. Preparations for etiotropic therapy and specific prevention of intestinal escherichiosis and intestinal yersiniosis.
5. Taxonomy and main biological properties of pathogens abdominal typhoid, salmonellosis.
6. Epidemiology, pathogenesis, immunity called diseases.
7. Principles microbiological diagnostics abdominal typhoid, salmonellosis.
8. Preparations for etiotropic therapy and specific prevention abdominaltyphoid, salmonellosis.
9. Taxonomy and main biological properties pathogens shigellosis, cholera.
10. Epidemiology, pathogenesis, immunity called diseases.
11. Principles microbiological diagnostics shigellosis, cholera.
12. Preparations for etiotropic therapy and specific prevention shigellosis,cholera.

INDEPENDENT WORK

Bacteriological method research

1. Selection clean culture from researched material (excreta patient).
2. Sowing researched material on the differential- diagnostic Wednesday Endo (demonstration).
3. Accounting results sowing researched material on the Wednesday Endo. Selection "suspicious" colonies and them the study on the environment endo, macroscopic characteristic colonies (demonstration).
4. Seeding "suspicious colonies" on the Wednesday Ressel and MPB.
5. Decor protocol research.
6. Accounting results on the differential diagnostic Wednesday Endo, bismuth -sulfite agar (demonstration).
7. Accounting results on the environment Ressel and MPB.
8. Accounting results reactions Vidal.
9. Accounting results express diagnostics cholera (demonstration).
10. Accounting for the results of sowing on a differential diagnostic mediumPloskireva (macro- and microscopic research).

PROTOCOL RESEARCH

№№ P/P	researched material	results research	Graphic image

METHODOLOGICAL RECOMMENDATIONS

AT connections With difficulty differentiation pathogens intestinal diseases, defiant similar clinical manifestations, it is necessary to conduct a comprehensive microbiological research, including simultaneous search in the investigated material pathogens escherichiosis, shigellosis, salmonellosis and cholera.

1. researched material (stool of the patient) sown on surface one from differential diagnostic Wednesdays for allocation pathogen intestinal diseases (Wednesday Endo) and one %alkaline agar for allocation pathogen cholera.

Sowing is carried out by a stroke on a dense surface nutrient medium in order to mechanical separation microbes and receiving isolated colonies.

Cups with 1% alkaline agar are incubated at 37 degrees. 10-12 hours, cups Wednesday Endo - 18-24 hours.

2. After incubation in thermostat crops on the cups co Wednesdays Endo and one % alkaline agar is viewed in transmitted and refractive light. With absence any signs of microbial growth on alkaline agar, a negative answer is given in respect finding pathogen cholera in researched material.

On the environment Endo through 18-24 h. growth in thermostat noted Availability colonies crimson red (fermenting lactose, which is part of the medium) and colorless (not fermenting lactose).

3. Colorless ("suspicious") colonies are sown on Ressel's medium. Medium composition Ressel: MPA, 1 % lactose, 0.1 % glucose and Andrede indicator.

Sowing is done as follows: the removed colony is carefully, without touching the edges test tubes, contribute in condensation liquid, then strokes sow all sloping surface of the medium and make an injection into the depth of the column. A test tube with inoculation environment Ressel put in thermostat (37°C) on day (18-24 hours).

Simultaneously for study proteolytic activity culture lactose-negative colonies are sown in a test tube with BCH with indicator papers, impregnated acetate lead and oxalic acid for definitions education hydrogen sulfide and indole. test tube placed in thermostat (37°C, 18-24 h.)

Escherichiosis (intestinal coliinfection) is an acute intestinal infection caused byvarious serological groups of enteropathogenic Escherichia coli (EPEC), occurring with symptoms of general intoxication and a syndrome of lesions of the gastrointestinal intestinal tract.

Etiology escherichiosis.

pathogens — enteropathogenic intestinal sticks —belong to to mind Escheirichia, kind Escherichia, family enterobacteroceae, present yourself Gram-negative rods that are stable in the environment. Can keep for months in soil, water, feces. They grow well on normal nutrient media. Fast die when boiled and exposed to disinfectants. Escherichia have complex antigenic structure: somatic O antigen (thermostable), surface (capsular) K antigen and flagellate H-antigen (thermolabile).

Intestinal infections caused by EPKD are more common in young childrenClassification escherichiosis:

- Enteropathogenic (salmonella-like).
- Enterotoxic (cholera-like).
- Enteroinvasive (dysentery-like).
- Enterohemorrhagic.

The diagnosis of escherichiosis can only be established by isolating the pathogen. For bacteriological examination, faeces, vomit, washings are taken stomach, at generalized forms - blood, CSF. Conduct studybowel movements need straightaway same, how only sick addressed per help to doctor, So how With flow time probability allocation pathogen fast decreases. Collection bowel movements held after natural defecation or With help tampons in test tubes With glycerin mixture in quantity not more 1/3 volume preservative a vomit and gastric lavage - in glass jars with a capacity of 200-250 ml. AT medical institution must to be carried out not less three diagnostic research (first - at admission sick before destination to him antibiotics, chemotherapy drugs).

FROM purpose allocation EPKP and ETCP should select samples bowel movements from recent servings, at research EICP - samples With impurity mucus.

The selected material is delivered to the laboratory within the first 2 hours, if it impossible - placed in a refrigerator and sent to the laboratory no later than 12 hours after fence.

At decision question about etiological roles pathogen at occurrence intestinal infection is necessary take account of the following criteria:

- selection Escherichia certain serovars, related to EPKP, EICP, ETCP, EGKP or EACP, in monoculture in combined With non-pathogenic serovars escherichia; if escherichia pathogenic diagnosis maybe to be installed on alone positive bakposeva;
- massive selection ETCP (106/g faeces and more) and significant them dominance above representatives another conditionally pathogenic flora.

certain diagnostic meaning have serological methods research, although they and less informative, unconvincing, So how possible false positive results due to antigenic similarities With others enterobacteria. Used for retrospective diagnosis, especially during outbreaks. Currently, RNHA is used from serological research methods. (diagnostic titer 1:200 - 1:400 for adults, 1:40 - 1:80 for children); reaction immunofluorescence; reaction immune sorption antibodies, labeled enzymes; neutralization reaction; agglutination reaction with autoculture with increasing titer antibodies in 4 or more once in dynamics diseases.

A promising diagnostic method is the polymerase chain reaction (PCR).To prove pathogenicity escherichia, need make sure, what she is It has receptors providing adhesiveness, can produce heat- labile andthermostable toxins, contains plasmid DNA encoding toxin formation (Protasov S.A., 2003).

If a stand out non-pathogenic escherichia, necessary suit to diagnostics how to such at others OKI, caused conditionally pathogenic flora: triple massive growth microorganism, no seeding pathogenic agents.

Diagnosis "escherichiosis" how noted unauthorized without bacteriological, a also serological confirmation. Exception is clinical- epidemiological justification diagnosis.

Instrumental methods surveys (sigmoidoscopy, colonoscopy) escherichiosis uninformative.

At design final diagnosis indicated view dedicated causative agent, gastrointestinal tract syndrome, severity of the disease. With a protracted course, the nature of the course of the disease is also noted. For example: escherichiosis (E. coli O111) in form acute gastroenteritis, moderate gravity.

The diagnosis of bacterial carriage can only be established when clinical symptoms of the disease are absent at the present time and have not been observed in previous 1-1.5 months Bacteriocarrier, as a rule, short-term (1-2 times pathogen release). In such cases, when making a diagnosis, only view pathogen. For example: bacteriocarrier enteropathogenic escherichia O125.

Etiology. Pathogen (Yersinia enterocolica) - gram negative wand, anaerobic, grows well on ordinary nutritional environments at low temperatures. Known thirty serovars. Disease at human more often cause 3rd, 5th, 8th and 9th serovars.

Intestinal yersiniosis.

Epidemiology. The source of infection are humans and animals, sick and carriers. Especially often pathogen is found at murine rodents, major horned livestock, pigs, dogs, cats, in dairy products, ice cream. Infection of a person occurs through the mouth when eating infected food, wateror contact way.

Disease meets in flow Total of the year.

Pathogenesis. Pathogen breeds in thin intestines, due to what develops enterocolitis or gastroenterocolitis. AT heavy cases in areas terminal department thin guts arises ulcerative process With involving mesenteric lymphatic nodes. At penetration pathogen in blood are celebrated bacteremia and generalization process With development inflammation in organs.

Clinic. The incubation period is 2-3 days. Clinical symptoms in patients practically not is different from such at pseudotuberculosis. However necessary have in mind what at intestinal yersiniosis disease often starts With intestinal disorders (copious watery stools mixed with blood), and damage to internal organs occurs, as it were, secondarily at the height of clinical manifestations and more often in severe cases.

In the diagnosis of intestinal yersiniosis , the leading role is played by bacteriological and serological methods research. Yersinia enterocolica can highlight from feces, blood, urine, pus, slime from pharynx, lymphatic node. From methods serological diagnostics use the agglutination reaction and the indirect hemagglutination reaction. Diagnostic titer 1:100 and above. A more reliable increase in the titer of specific antibodies in dynamics diseases.

Prevention of intestinal yersiniosis is carried out in the same way as for other intestinal diseases. infections. Specific prophylaxis not developed.

Abdominal typhus — acute cyclically flowing intestinal anthroponoticinfection caused by the bacteria Salmonella typhi (Salmonella enterica serotype typhi), with alimentary through transmission (fecal-oral), characterized fever phenomena general intoxication With development typhoid status, roseolous rashes on the skin, hepato- and splenomegaly and specific defeat lymphatic lower department thin intestines.

The causative agent is Salmonella typhi from the family Enterobacteriaceae of the genus Salmonella, movable gram-negative rod with rounded ends, highly stainable everyone aniline dyes. Works out endotoxin, pathogenic only for person. Not creates controversy.

Typhoid bacteria are quite stable in the external environment: in fresh water reservoirs, they persist for up to a month, on vegetables and fruits - up to 10 days, and in dairy products can reproduce and accumulate.

Under the influence of 3% chloramine solution, 5% carbolic acid solution, sublimate (1:1000), 96 % ethyl alcohol they perish through several minutes.

Typhoid salmonella have a complex antigenic structure. Various serovars contain a characteristic set of antigenic factors that are composed of combinations O- and H antigens.

laboratory diagnostics before Total is in bacteriologicalresearch blood, feces, urine, bile. Method blood cultures can use With first days diseases and before end feverish period, desirable before start treatment. To do this, 5-10 ml of blood from the cubital vein at the bedside of the patient is sown at 20% bile broth or Rapoport's medium, meat-peptone broth with 1% glucose, or even in sterile distilled water. The volume of the medium is 50-100 ml. Material ratio and Wednesday should be 1:10. Feces, urine, duodenal contents are examined from the 2nd week from start diseases, sowing on the environments Ploskireva, Levin, Muller and others The preliminary result of these studies is obtained after 2 days, the final - after 4 days. To detect typhoid bacillus in feces, urine, duodenal content use REEF With labeled sera to O- and Vi antigens. A preliminary response can be received within 1 hour, the final - after 5-20 h.

From serological methods use RA (Vidal) and RPGA With cysteine. The Vidal reaction is set with H- and O-antigens from the 7th-9th day of the disease, repeated on the 3rd-4th week for definitions growth titra (from 1:200 before 1:400-1:800-1:1600). Last thing is important to exclude a positive reaction result, which can bedue to prior immunization against typhoid fever. The answer might be received through 18-20 h. At staging RPGA accounting results carry out afterincubation of the plates at 37 ° C for 1.5-2 hours and again - after 24 hours of exposure at room temperature. Positive counts reaction in titre 1:40 and above. **Salmonellosis is an** acute intestinal infection of animals and humans caused by salmonella. Acute infectious zooanthroponotic disease, called salmonella and characterized in general case, development

intoxication and defeat gastrointestinal tract.

salmonellosis at human consider how certain disease (nosological form), distinguishing it from typhoid fever and paratyphoid fever. Main sourceinfections — food products, less often-sick animal, in individual cases the source of infection can be a person (sick or bacteriocarrier). Infection going on through infected food products, how rule animal origin (meat and meat products, milk, eggs, especially duck and goose), withforced, wrong slaughter animals, violation rules storage and food preparation (contact of finished and raw products, insufficient thermal treatment products before use and t. d.). salmonellosis develop in those cases when living organisms accumulated in foods enter the body salmonella.

On the territories RF most often meet the following serovars kind Salmonella enterica subspecies enterica: Salmonella enteritidis, Salmonella Typhimurium , Salmonella infantis.

Clinical manifestations salmonellosis varied — from asymptomatic carriage pathogen infections before heavy septic forms. Incubation period ranges from 2-6 hours to 2-3 days.
Distinguish several clinical forms salmonellosis:
1.Gastrointestinal form2.
Typhoid form 3.Septic the form

AT 15-17 % cases salmonellosis in period convalescence observed short-term bacteriocarrier. Possible "transient" carriage (single selection salmonella without clinical manifestations) and chronic bacteriocarrier.

Diagnostics salmonellosis carried out complex With taking into accountepidemiological data, symptoms and laboratory results, aimed at isolation and typing of the pathogen. The main type of typing salmonella is an agglutination reaction. For its holding until recently used hyperimmune sera, but now they have been replaced by monoclonal antibodies to salmonella.
Prevention.
Veterinary and sanitary supervision of slaughter and processing of carcasses ; performancesanitary rules for the preparation, storage and sale of food products;
examination of people entering work at catering and trade enterprises,children's institutions.

SITUATIONAL TASKS

1. From bowel movements sick allocated gr-, mobile vibrio, agglutinating about- agglutinating cholera serum, insensitive to action of a specific cholera phage, insensitive to polymyxin. Your indicative diagnosis? What need more do for confirmation diagnosis?

2. At sick With profuse diarrhea and vomiting and from bowel movements and emetic masses allocated gr- mobile wand, not losing mobility in the presence of agglutinating cholera serum, inoculated according to Polev-Ermolyeva after 3 hours in first test tube - diffuse haze, in second - diffuse haze, in third - at adding solution Lugol turning blue. Your sentence? Stages further laboratory research?

3. From water open reservoir highlighted microbe: gr- wand very mobile giving on the alkaline agar very gentle transparent, bluish in passing light colony, splitting glucose, maltose beckons, not splitting lactose dulcite, thinning gelatin funnel. Your indicative diagnosis? Your laboratory tactics?

PRACTICAL OCCUPATION No. 12.

TEST CONTROL.

No. СТОМ-21-ИН

Federal State Budgetary Educational Institution of Higher Education
NORTH OSSETIAN STATE MEDICAL ACADEMY
Ministry of Health of the Russian Federation

Department of microbiology

COLLECTION METHODOLOGICAL DEVELOPMENT ON MICROBIOLOGY, VIROLOGY, IMMUNOLOGY -ORAL MICROBIOLOGY FOR STUDENTS DENTAL FACULTY

SPRING SEMESTER

Vladikavkaz

Authors: Associate Professor of the Department of Microbiology, FSBEI NOSMA of Ministry of Health Russia., Ph.D. Chertkoeva M.G., Assistant Khabieva B.A.

Main appointment developments - methodical help students to to each practical occupation in IV semester. Directions drawn up in compliance With Federal public educational standard Supreme and vocational education (2011)

REVIEWERS:
L.V. Bibaeva – MD, Professor, head department biology and histology FGBOUIN SOGMA Ministry of Health Russia.

A.R. Kusova- MD, Professor, head department of hygiene and physical education FGBOU VO SOGMA Ministry of Health Russia.

PRACTICAL OCCUPATION No. 1

Topic: Microbiological diagnostics viral diseases. Indication and identification viruses in researched material. Serological method diagnostics viral diseases: reactions neutralization, passive hemagglutination, ELISA. Working off methods diagnostics on the example viraldiseases:
- cultivation in chicken embryo, colored try, hemagglutination and braking hemagglutination at identification viruses influenza and SARS;
-serological tests and polymerase chain reaction in the diagnosis of viral hepatitis B, C, herpes, HIV.

Educational goal:

1. Explore morphology and ultrastructure viruses.
2. Train students methods virological diagnostics and specific prevention flu, SARS.
3. Train students methods virological diagnostics and specific prevention hepatitis B, C, herpes, HIV infections.

Student must know:

1. morphology, ultrastructure classification viruses.
2. morphology, ultrastructure classification bacteriophages.
3. Biological properties and laboratory diagnostics flu, SARS.
4. specific prevention flu, SARS.
5. Biological properties and laboratory diagnostics hepatitis B, C, D, G, HIV-infections.
6. specific prevention hepatitis B, C, D, G, HIV infections.
7. Biological properties and laboratory diagnostics herpes

Student must be able to:

1. Find viral inclusion method light microscopy.
2. Find viral inclusion method luminescent microscopy.
3. Put and take into account results REEF at SARS.
4. Put and take into account results RTGA for seroidentification at flu.
5. Put and take into account results ELISA for serodiagnosis at SARS.
6. Set and take into account the results of the ELISA reaction for serodiagnosis andseroidentification at hepatitis B, C, D, G, HIV- infections.
7. Put and take into account results RPGA at hepatitis AT.
8. Put and take into account results RTGA and ELISA for serodiagnosis at herpes.

Lesson plan:

1. Peculiarities biology viruses **.**
2. Principles classification viruses.
3. Types interactions viruses With cell.
4. Taxonomy and main biological properties pathogens flu, SARS.
5. Epidemiology, pathogenesis, immunity called diseases
6. Principles microbiological influenza diagnosis, SARS.
7. Preparations for etiotropic therapy and specific prevention flu,SARS.
8. Taxonomy and main biological properties pathogens hepatitis b , FROM,HIV infection herpes
9. Epidemiology, pathogenesis, immunity called diseases.

10. Principles microbiological diagnostics hepatitis b , FROM, HIV infection,herpes.
11. Preparations for etiotropic therapy and specific prevention hepatitisB, C, HIV infections, herpes.

Independent Work students:

1. Parsing supplies and accounting results REEF at SARS (demonstration).
2. Parsing supplies and accounting results RTGA for seroidentification with flu (demonstration).
3. Parsing supplies and accounting results ELISA for serodiagnosis with SARS(demonstration).
4. Parsing productions and accounting reaction results ELISA for serodiagnosis andseroidentification at hepatitis B, C, HIV infections (demonstration).
5. Analysis of the formulation and accounting of the results of the reaction of RPHA in hepatitis B (demonstration).
6. Parsing productions and accounting RTGA results and ELISA for serodiagnosis (demonstration).

INFORMATIONAL MATERIL ON THEME

VIRUSES

Viruses have properties that make it impossible to use conventional methods to study them. methods microbiological research.

Distinctive properties viruses:

1. smallest sizes, measurable thousandths shares micron - millimicrons - from 8-10 m up to 300-400 m.
2. Filterability through special finely porous filters, not passingother microorganisms.
3. non-cellular structure.
4. Absolute parasitism, those. ability live and multiply only in alivecells.

The form viral particles It has several types:

6. rod-shaped
7. spherical (spherical)
8. Cuboid
9. Capitate (spermatozoa)10. Filiform

mature viral particle, called *virions,* have next scheme buildings: in central parts located molecule DNA or RNA, which forms *nucleoid.* Around situated protective protein shell, called *capsid,* built from morphological units, called *capsomeres.* Some complex virions have external shell, called *supercapsid.*

For microbiological diagnostics viral infections in the present time apply three main methodical approach:

4. **Virological diagnostics** - founded on the allocation from researched material virus and his subsequent identification.
5. **Serological diagnosis** - determination of specific immunological changes in body under action viruses (more often Total With help diagnosticums reveal in serum blood antiviral antibodies).
6. **Molecular biological diagnostics** - detection in clinical material fragments nucleic acids causative viruses With help probes (hybridization NK) or PCR.

Individual viruses larger than 200 m can be stained according to Romanovsky - Giemsa; smaller viruses (variola viruses) can only be detected using special processing methods.

bacteriophages differ on chemical structure, type nucleic acid, morphology and nature of interaction with bacteria. Size of bacterial viruses in hundreds and thousands of times less microbial cells.

A typical phage particle (virion) consists of a head and a tail. Tail length is usually2-4 times

the diameter of the head. The head contains genetic material - single stranded or double stranded RNA or DNA With enzyme transcriptase in inactive state, surrounded protein or lipoprotein shell - ***capsid*** , preserving genome outside cells.

Nucleic acid and capsid together make up the nucleocapsid. Bacteriophages can have icosahedral capsid, assembled from sets copies one or two specific proteins. Usually angles are made up of pentamers squirrel, and support each side from hexamers Togo same or similar squirrel. More Togo, phages on form may to be spherical, lemon-shaped or pleomorphic. Tail represents yourself protein tube - a continuation of the protein shell of the head, at the base of the tail there is ATPase, which regenerates energy for the injection of genetic material. There are alsobacteriophages With short offshoot, not having offshoot and filiform.

By international classification all viruses subdivided on type nucleicacids on the 2 subtype - RNA- and containing DNA. Further separation virusesis carried out on the basis of the size of the viruses, the type of symmetry in the formation of capsids,the presence or absence of outer shells and the number of capsomeres contained in them.VIROLOGICAL METHOD RESEARCH is main and most authentic, allows highlight virus from researched material With subsequent hisidentification. FROM purpose accumulation virus-containing material are used chicken embryos and culture fabrics (artificially cultivated cells toy ordifferent fabrics). cultures fabrics supported on the natural (Wednesday 27, Enders) andsynthetic (medium 199, Needle, Melnik-Riordan) nutrient media, cooked on the basis solutions Hanks and Earl. cultivated they in conventional test tubes cups carrel, test tubes Barsky.

Methodology infections chicken embryo

There are several ways to infect a chicken embryo. Most often the material injected into the allantoic and amniotic cavities, onto the chorionallantoic membrane and into yolk bag. Before infection shell eggs above air camera treated with 70% alcohol, burned on a flame, smeared with 2% iodine tincture, secondarily wipe with alcohol and burn.

At contagion in allantoic cavity in shell above air camera (borderswhich in advance circle pencil at translucence eggs in ovoscope) are doingsmall hole With help scissors or scalpel. Tuberculinov syringeintroduce 0.1-0.2 ml virus-containing material on the depth 2-3 mm below bordersair cameras. Puncture in shell poured molten paraffin. Openinginfected embryos produce in terms maximum accumulation virus (through 48-72 h incubation at temperature 37 FROM) after processing shells alcohol and 2% solution iodine her dissect and dump, filmed carefully shelledshell and consider chorionallantoic shell around places infections on theAvailability foci lesions (hemorrhages, whitish foci defeats).

Classification cellular crops:

• **primary** receive directly from fabrics animal and human through destruction proteolytic enzymes (trypsin, collagenase) intercellular substances. Disunited cells, placed in nutritional Wednesday, able attach to the surface of the culture vessel and multiply, forming a monolayer - layer one cell thick. With the help of special reagents, cells can be removed from surfaces one vessel and transplant in another. Such manipulation called **passage.** primary crops withstand not more 5-10 passages.

• **transplantable** (passage) cellular culture able withstand unlimited number of passages. They originate from tumor cells that have lostdifferentiation and not having restrictions growth.

• **semi-transplantable** (diploid) culture - fibroblast-like cells, which able to fast reproduction, withstand before 30-60 passages and save original set of chromosomes.

Viruses can reproduce only in the cells of a living organism. Concerning viruses cultivated through infections chicken embryos or cultures fabrics, a also suckling animals.

Detection (indication) of viruses *Virus*
detection in chick embryo1. Death
2. The appearance of an odor upon opening 3. Cloudiness liquids in cavities
four. Education sores and hemorrhages on the shells

Biological method research is in contagion sensitive to virus animal researched material, studying clinical and pathoanatomical paintings diseases. AT framework this method are used

various animals: monkey, rabbits, maritime pigs, dogs, mice, rats. Ways infections: subdural, intracerebral, intranasal and other.

Methods for detecting the virus in the body of laboratory animals differ in dependencies from the view animal and type virus.

Detection viruses in culture cells

Revealing on cytopathic action (CPD). JPC represents yourself degenerative changes in cells that result from reproduction in them viruses.

Distinguish complete and partial degeneration cells monolayer.

With complete degeneration caused, for example, by polio viruses, Coxsackie and ECHO, cells of the monolayer undergo significant changes, more of themslough off co glass. Remaining single the cells are wrinkled

Partial degeneration It has several varieties:

3 .By type cluster formation (adenoviruses);

4 .By type focal destruction (smallpox, flu);

3. By type symplast formation (measles, mumps, parainfluenza, herpes, HIV).

Proliferative type of changes typical for some oncogenic viruses, transforming cells in malignant.

Intracellular inclusion formed at reproductions some viruses in cytoplasm and nucleus of cells (smallpox, rabies, influenza, herpes, etc.) They are found when microscopy after coloring monolayer on Romanovsky - Giemse, a also at luminescent microscopy.

Salk color test. As a result of the vital activity of cells in a nutrient medium accumulate sour products. AT result this color incoming in compound environments indicator (phenolic red) becomes orange. At contagion culture cells with cytopathogenic viruses such as enteroviruses or reoviruses, metabolism cells suppressed medium pH and her color not are changing (Wednesday remains red).

Reaction hemagglutination. AT basis this reactions lies ability viruses, containing hemagglutinin receptors, "glue" erythrocytes. If a there is hemagglutinins - RGA+(umbrella), if No - RGA - (button).

Reaction hemadsorption. Mechanism similar With RGA.

Flu (from fr. grippe) — acute infectious disease respiratory ways, caused by the influenza virus. Included in the group of acute respiratory viral infections(ARVI). Periodically spreads in the form of epidemics and pandemics. Currentlymore than 2000 variants of the influenza virus have been identified, differing in antigenic spectrum.

First virus was highlighted in 30s of the year XX century. Viruses influenza relate to family Orthomyxoviridae, which includes childbirth Influenza A, b, FROM. Antigenic properties internal proteins virion (M1 and NP) determine belonging virus influenza to the genus BUT, AT or FROM.

Viruses containing three subtypes of HA are of epidemic importance to humans. (H1, H2, H3) and two NA subtypes (N1, N2). Influenza A and B viruses contain NA and HA in quality major structural and antigenic components viral particle, possessing hemagglutinating and neuraminidase activities. The influenza virus C no neuraminidase, it has instead a hemagglutinin-esterase (penetrating)protein (HEF). The RNA strand is surrounded by protein and packaged in a lipoprotein membrane. Virions able agglutinate erythrocytes and elute in them With help virus-specific enzymes.

Virus influenza It has spherical form diameter 80-120 nm, in center there are RNA fragments enclosed in a lipoprotein membrane on the surface which has "spikes" consisting of hemagglutinin (H) and neuraminidase (N). Antibodies produced in response to hemagglutinin (H) form the basis of immunity against certain subtype pathogen influenza

source infections is sick human With explicit or erased form disease that releases the virus with coughing, sneezing, etc. The patient is contagious from the first hours diseases and before 5th–7th days disease.[5] Characterized aerosol (inhalation tiny drops of saliva, mucus that contain the influenza virus) transmission mechanism and extremely fast spread in form epidemics and pandemics. Epidemics influenza caused by serotype A occurs approximately every 2-3 years, and those caused by serotype B - every 4-6 years. Serotype C does not cause epidemics, only sporadic outbreaks in children and debilitated people. In the form of epidemics, it occurs more often in the autumn winter period. Periodicity epidemics tied With frequent

change antigenic structures virus during your stay his in natural conditions.

The entry gate for the influenza virus is the cells of the ciliated epithelium. upper respiratory tract - nose, trachea, bronchi. The virus replicates in these cells and leads to them destruction and death. This explained irritation top respiratory tract cough, sneezing, nasal congestion. Penetrating into the blood and causing viremia, virus renders direct, toxic action, emerging inform raise temperature, chills myalgia, head pain. Except Togo, virus raises vascular permeability, causes development stasis and plasma hemorrhage.

Traditional way warnings diseases influenza is vaccination. Suggested vaccine for prevention influenza in form alive, killed (inactivated), subunit vaccine. Vaccination is especially indicated in groups risk - children, the elderly, patients with chronic heart and lung diseases, and also doctors. Usually carried out, when epidemiological forecast indicates the expediency of mass events (usually in the middle of autumn). Possible and second injection in middle winters.

For fast diagnostics influenza use "express method" detection influenza virus using fluorescent antibodies. The test material is taken from nose in first days illness. cooked from him smears handle specific flu-like fluorescent sera. formed the antigen-antibody complex glows brightly in the nucleus and cytoplasm of cylindrical cells epithelium and is clearly visible under a fluorescent microscope. The answer can be obtained through 2-3 h.

Serological research help retrospective diagnostics flu. Examine paired blood sera taken from patients in the acute period of the disease (up to the 5th days from start disease) and in period convalescence With interval 12-14 days. The most indicative in serological diagnostics are the binding reaction complement (RSC) with influenza antigens and hemagglutination inhibition reaction (RTGA). Diagnostic counts growth titra antibodies in 4 times and more.

Hepatitis B is a viral disease caused by the hepatitis B virus. (in the specialized literature it may be referred to as "HBV virus", HBV or HBV) from the family hepadnaviruses.

The virus is extremely resistant to various physical and chemical factors: low and high temperatures (in volume including boiling), repeated freezing and thawing, prolonged exposure to an acidic environment. In the external environment at room temperature, the hepatitis B virus can survive up to several weeks: even in a dried and imperceptible stain of blood, on a razor blade, the end needles. In blood serum at a temperature of +30°C, the infectivity of the virus remains flow 6 months at -20°C near fifteen years. Inactivated at autoclaving infor 30 minutes, dry heat sterilization at 160°C for 60 minutes, warming up at 60°C in flow 10 hours.

Mechanism transmission infections — parenteral. Infection going on natural (sexual, vertical, domestic) and artificial (parenteral) ways. The virus is present in the blood and various biological fluids - saliva, urine, semen, vaginal secret, menstrual blood and others contagiousness (infectiousness) virus hepatitis A B exceeds contagiousness HIV in 100 once.

Greatest meaning before everywhere had exactly parenteral path — infection during medical and diagnostic manipulations, accompanied by a violation integrity skin or mucous cover through medical, dental, manicure and other tools, transfusion blood and her drugs.

Pathogenesis. The most significant pathogenetic factor in viral hepatitis B is death of infected hepatocytes due to attack by their own immune agents. Massive death of hepatocytes leads to impaired liver function, primarily detox, in lesser degree - synthetic.

Incubation period (time With moment infections before appearance symptoms) hepatitis B averages 12 weeks, but can range from 2 to 6 months. The infectious process begins from the moment the virus enters the bloodstream. After viruses entering the liver through the blood, there is a latent phase of reproduction and accumulation viral particles. At achieving certain concentration virus in liver acute hepatitis B develops. Sometimes acute hepatitis goes away for a person practicallyimperceptibly, and is found by chance, sometimes leaks in light anicteric form

— appears only malaise and decline performance. Some researchers believe what asymptomatic flow, anicteric the form and

"icteric" hepatitis are equal in the number of affected persons of the group. That is identified diagnosed cases of acute hepatitis B account for only one third all cases acute hepatitis.

Vaccination. Mandatory vaccination. FROM recent time vaccination against hepatitis A AT was enabled in required calendar vaccination. newborns most sensitive to hepatitis b virus - in case of infection at this age, risk acquisition of a chronic form of hepatitis B is 100%. At the

same time, immunity created by the vaccine during this period of life, the most persistent. Recommended to vaccinate newborn still in the maternity hospital, then 1 month after the first vaccination, and through 6 months after first vaccinations (So called scheme 0-1-6). At pass the next injection should be remembered about the allowable intervals - 0-1 (4) -6 (4-18) months. However if were missed admissible intervals, necessary continue vaccination according to the scheme, as if there was no pass. If the vaccination was standard schedule, revaccination is usually not required because immunity preserved on lesser measure in flow fifteen years. For definitions, how much for a long time immunity persists throughout life, further research is needed - because Vaccination has been introduced relatively recently. Only after all vaccination course, almost 100% immunity is achieved. About 5% of the general population responds to vaccination, in these cases, other types of vaccines against hepatitis A AT.

laboratory diagnostics GW - founded on the identifying specific for GW antigens and corresponding antibodies in the blood, as well as viral nucleic acids, main from which are:

HB sAg - anti-HB s
anti-HBc class Ig M and IgG
HBe Ag - anti-HWe
DNA HBV

Most wide in diagnostics GV used definition HBsAg. The antigen is detected in both acute and chronic disease (however, acute infection is usually confirmed by the presence of high titers of anti-HBc IgM). With acute GV surface antigen virus is found through 3-5 weeks from momentinfection, that is, long before the appearance of clinical signs of the disease and in these cases is the only serological marker. HBsAg is constantly detected in the preicteric and icteric periods of the disease. HBsAg persistence for 6 months or more indicates a protracted or chronic course of the disease, and allows suppose chronic carriage virus. Elimination HBsAg and appearance antibodies to him is indispensable condition convalescence. Serological markers replication HBV are - anti-HBs class IgM, HBeAg, DNA and DNA- polymerase, which are found at sharp GV With first days clinical manifestations and can be detected during exacerbation of chronic hepatitis B. Serological HBV replication markers are determined both for general diagnostic purposes and for evaluation efficiency applied therapy.

Virus hepatitis A D (HDV) first was discovered in 1977 year. He not belongs neitherto one of the known families of viruses. HDV is a spherical particle, in the center of which is a spherical antigen (ND-Ag) containing RNA. Outdoor shell particles formed superficial antigen virus hepatitis A AT - HBs antigen (HBsAg). HDV cannot exist without HB virus replication, so itcalled a parasite virus, or a defective virus. The hepatitis B virus performsthis helper function, then there is role assistant for breeding NDV. That's why NDV
- infection always occurs together with HBV infection. NDV is located mainly in nuclei of hepatocytes and occasionally in cytoplasm.

Epidemiology. HDV infection is widespread. Circulation intensity NDV in different regions of the world varies significantly, but in general repeats the situation at HBV, although and not absolutely exactly. At acute hepatitis antibodies to NDV stand outin different regions in 2-7% of patients, and in chronic hepatitis - in 9-50% of patients. On the territory of the former USSR, among “healthy” carriers of HBsAg, the highest frequency (10-20 %) detection antibodies to NDV identified in Moldova Kazakhstan, Middle Asia, Tuva, that is, in areas hyperendemic for HBV. In the European part of Russia, the frequency identifying antibodies to NDV is 1.2-5.5 %.

source infections are sick sharp and chronic IOP, virus carriers, as well as carriers of anti-NDV, since it is known that in individuals with anti-NDV can be found at the same time RNA-NDV. Broadcast NDV is happening So same as and at HBV (parenteral, sexual by, from mothers fetus). To delta - infections susceptible individuals who have not had HBV (i.e., do not have anti-HBs), as well as carriers HB-virus (healthy carriers of HBsAg and patients with chronic HBV). delta infection occurs both sporadically and and in form outbreaks.

Pathogenesis, clinic. Infectious process, conditioned NDV, appearsbefore Total appearance ND-Ag in blood. Delta - antihemia maybe to be short-term or lengthy in dependencies from Togo, how happened infection and whether there is HB-virus integration into the hepatocyte genome. Distinguish acute, protracted and chronic flow delta- infections. Character her currents limited duration HBs- antigenemia: on measure her exhaustion stops and synthesis NDV, and ends delta- dependent pathological process.

Delta- infection develops in form co-infections or superinfection. At co-infection, simultaneous infection of HBV + HDV occurs in persons who have not been ill before HBV infection (not having HBV infection markers prior to infection). In that case, acute HBV+HDV-hepatitis develops with the appearance of serological markers two acute infections at once. In coinfection, HBV replication is most often HBV + HD - hepatitis A usually happens sharp and ends recovery.

In case of superinfection with HDV - the infection is superimposed on the current HBV infection in healthy carriers of HBsAg, in convalescents of the main HBV, in patients with chronic HBV. At this develops clinic acute viral hepatitis A delta, accompanied appearance antibodies to delta antigen.

laboratory diagnostics hepatitis A D (DG) Virus hepatitis A D (IOP) - this is defective virus, containing single helix RNA, to whom for replication the help of the HB virus is needed for the synthesis of envelope proteins consisting of HBsAg, which is used to encapsulate the HDD genome. IOP does not belong to any of the known families of animal viruses, in terms of its properties, HDV is closest to viroids and plant satellite viruses. Laboratory diagnostics is carried out by detecting serological markers of IOP, including the presence of antigen, antibodies to him and IOP RNA. Detection of HDV antigen and HDR RNA in blood serum or tissue liver indicates the presence of active HD infection, however, it should be noted that these markers may not show up in serum sick fulminant GD. Marker active replication IOP also is anti-CHD class IgM. Serological markers of HD infection depend on how the virus was acquired - in form of co-infection with HBV (in most patients, the disease has an acute course and ends recovery) or superinfection at sick With chronic GW- infection (flows heavier, how coinfection - in ten% develops fulminant hepatitis). In case of superinfection in patients with chronic hepatitis B infection, serological the picture has the following characteristic features: - the HBsAg titer decreases by the time appearance antigen IOP in serum; - antigen IOP and RNA-HVD continue be determined in serum, since usually in most patients with HD superinfection (70-80%) develops chronic infection, in difference from cases coinfections; - high titers of antibodies (anti-VGD) of both the IgM and IgG classes are determined, which persist indefinite time. Serological markers virus DG determine method enzyme immunoassay and radioimmune analysis, a RNA-HVD - method polymerase chain reaction.

Hepatitis C is an anthroponotic viral disease with a parenteral mechanism. infection, most often flowing in form post-transfusion hepatitis A With predominance anicteric and prone to chronization.

Hepatitis C is called the "gentle killer" because of its ability to mask the true reason under the guise of a multitude others diseases.

Parenteral viral hepatitis C called RNA containing virus With the size of the virion is 30-60 nm, belonging to the family Flaviviridae. virus particles HCV are enveloped, found in blood in trace amounts, and are associated with low-density lipoproteins and antibodies to proteins of the hepatitis C virus. Viruses, isolated from complexes with lipoproteins and anti-HCV antibodies have a diameter 60-70 nm. At electron microscopic studying on the surfaces virion identified well-defined ledges height 6-8 nm.

source infections are sick With active hepatitis C and latent sick — carriers virus. HCV infection is infection With parenteral mechanism infections — through infected blood and her Components. infection Maybe at parenteral manipulation, in volume including in medical institutions, including rendering dental services, through injection equipment, at acupuncture, piercing, drawing tattoos, at rendering row services in hairdressing, but at genital contacts probability get sick hepatitis FROM much less, how hepatitis AT, and comes down to minimal indicators.

Laboratory diagnosis of hepatitis C (HC). Laboratory diagnosis of HS was solved using modern methods of molecular biology, given that in HS the virus is in an extremely low concentration and its antigens are not available for detection with help contemporary methods indication, efforts researchers concentrated on the detection of antibodies to various antigenic components of the virus, the detection of which can serve as an indicator of the presence of the virus. Proteins were used as antigens. encoded by the structural and non-structural zone of HCV RNA, obtained using recombinant technology or synthesis (polypeptides used in modern immunological methods - C22-3; C33s, C100-3, C200, NS5, S-1-1). laboratory diagnosis of HC

is based on the detection of serological markers in HCV: antibodies to HC virus (anti-HCV, anti-HCV class IgM, IgG) by ELISA and RNA-HCV method PCR. To date, 4 generations of test systems have been developed to detect anti- HCV in enzyme immunoassay, but the first generation ELISA is not currently used due to for low sensitivity. HCV RNA is an indicator of active HCV replication and the earliest marker of infection, and can be detected by polymerase chain reaction as early as 1-2 weeks after infection, shortly before the increase serum transaminase levels. Anti-HCV is detected by 5-6 weeks after onset hepatitis in 80% of cases and by week 12 in 90% of individuals by enzyme immunoassay. When determining anti-HCV, in some cases a false positive is recorded reaction. To distinguish false positive samples from real samples containing antibodies to HCV developed additional tests - recombinant immunoblotting, definition spectrum anti-HCV proteins.

HIV — virus immunodeficiency human, defiant disease — HIV infection last stage which known how syndrome acquired immunodeficiency (AIDS) - in difference from congenital immunodeficiency.

Spreading HIV infections related, main the way With unprotected sexual contacts, using infected virus syringes, needles and others medical and paramedical instruments, transmission of the virus from an infected mother to child during childbirth or while breastfeeding. In developed countries Mandatory testing of donated blood has greatly reduced the possibility of transmission virus with her use.

HIV infects before Total cells immune systems (CD4+ T-lymphocytes, macrophages and dendritic cells) a also some other types cells. infected HIV CD4+ T-lymphocytes gradually die.

Virus immunodeficiency human refer to family retroviruses (Retroviridae), kind lentiviruses (Lentivirus). Name Lentivirus going on from Latin the words lente — slow. Such title reflects one from features viruses this groups, aexactly — slow and unequal speed development infectious process inmacroorganism. Lentiviruses also have a long incubation period.*Diagnostics.* Flow HIV infections characterized lengthy absence significant symptoms disease[81]. Diagnosis HIV infections put on the basislaboratory data: at identifying in blood antibodies to HIV. Antibodies to HIV in periodacute phase, how rule not discover. AT first 3 months after infections antibodies toHIV come to light at 96-97 % patients through 6 months — at the rest 2-3 %, a in morelate terms — only at 0.5-1 % (source Centers for disease control and preventionusa, 2009). AT stages AIDS register significant decline content antibodiesin the blood. The first weeks after infection are the "periodseronegative window", when antibodies to HIV not come to light. That's why negativeresult testing on the HIV in this period not means what human not infected HIV and not maybe infect others.

For the diagnosis of lesions of the oral mucosa in HIV-infected patients accepted working classification, approved in London, in September 1992 of the year. All defeat divided into 3 groups:

1 group - lesions clearly associated with HIV infection. This group includes the following nosological forms:

candidiasis (erythematous, pseudomembranous, hyperplastic, atrophic);hairy leukoplakia;
marginal gingivitis;
ulcerative necrotic gingivitis;
destructive periodontitis; sarcoma
Kaposi;
non-Hodgkin lymphoma.

2 group - lesions less clearly associated with HIV infection:bacterial
infections;
disease salivary glands; viral
infections; thrombocytopenic
purpura.

3 Group — defeat, which may to be at HIV infection but not related Withher.

Herpes (Greek ἕρπης - creeping, spreading skin disease) - viral disease With characteristic rash grouped bubbles on the skin and mucous shells.

Herpes simplex (Herpes simplex) - a group of crowded vesicles with a transparent

contents on an inflamed base. Herpes is preceded by itching, burning of the skin, sometimes chills, malaise.

Shingles (Herpes zoster) - characterized by pain along the nerve, head pain. Through several days on the site skin on move nerve appear rashes in the form of grouped vesicles, first with a transparent, and later purulent bloody content. Are increasing lymphatic nodes, rises body temperature, the general condition is disturbed. Neuralgic pains can last up to several months.

Pathogenesis. Virus herpes transmitted immediate contact by, a also through items everyday life. Possible also broadcast infections air-drip way. Herpes penetrates through mucous shells cavities mouth, top respiratory tract and genitals. Having overcome tissue barriers, the virus enters blood and lymph. Then gets into various domestic organs.

Virus penetrates in sensitive nervous graduation and embedded in genetic apparatus nervous cells. After this delete virus from organism impossible, he will remain with the person for life. The immune system responds to penetration herpes development specific antibodies, blocking circulating in blood viral particles. Characteristically awakening infections in cold season, with colds, with hypovitaminosis. reproduction herpes in the cells of the epithelium of the skin and mucous membranes leads to the development of dystrophy andcell death.

According to research scientists Colombian University, herpes is stimulating factor for the development of Alzheimer's disease. Later, these data were independently validated by researchers at the University of Manchester. Previously the same Group researchers under leadership Ruth Yitzhaki proved what virus simple herpes is found in the brains of almost 70% of patients with Alzheimer's disease. Except Togo, they confirmed what at infection virus culture cells brainthere is a significant increase in the level of beta-amyloid, from which plaques. In a recent study, scientists were able to find that 90% of plaques in brain patients With sickness Alzheimer's contain DNA simple herpes — HSV-1.

For diagnostics herpetic infections are used all laboratory reactions
— from cytological research before molecular biological methods.

Material for virus isolation for the purpose of diagnosing herpes infection maybe serve content herpetic bubbles, scrapings With horny shells andliquids from front cameras eyes, blood, saliva, urine, spinal liquid feces pieces fabrics brain, liver, kidney, spleen, lungs lymphatic nodes,taken on the bio- or autopsy.

Infectious material can for a long time keep at -70°C, then how at temperature -20°C he fast is inactivated. Virus containing fabrics may to be saved more 6 months at 4°C, if they are in fifty% solution glycerin.

There are a number of special methods for the detection of viral antigens, specific antibodies and virus-induced morphologically changed cells.

Most affordable and technically uncomplicated is cytological method, allowing to study morphological changes in cells infected with the virus herpes simplex. The effectiveness of the method depends on obtaining a sufficient amount cells for research. Availability intranuclear inclusions, characteristic for reproduction of the herpes virus serves as a confirmation of the diagnosis. It should be remembered that intranuclear inclusions are detected only after immediate fixation of smears scraping in absolute alcohol With subsequent coloration on Romanovsky-Giemsa. Morphological changes, induced virus simple herpes, can also detected in tissue sections of infected organs. characteristic of herpetic infections is: Availability multi-core cells, intranuclear inclusions and in some cases of hemorrhage. In the generalized form of the disease, multinuclear cells With eosinophilic inclusions find in zones necrotic fabrics various bodies (brain, liver, kidney, adrenal glands, epithelium bronchi and trachea).

Method immunofluorescence — is method express diagnostics herpetic infections and allows in flow 1-2 hours determine Availability herpesvirus antigens in clinical material (scraping With skin and mucous membranes, sections of biopsied organs). Identification of antigens of the simplex virus herpes maybe to be completed in various modifications method immunofluorescence
— straight, indirect, With application labeled complement.

From serological methods identification most often use reaction binding complement (RSK), especially in micromodifications her staging. micromethods use and for identifying virus simple herpes in reactions neutralization, passive hemagglutination and in others serological tests.

Sensitivity listed methods are different.

AT the present time one from most sensitive methods diagnostics herpetic infections is method enzyme immunoassay analysis (IFA), allowing find, in dependencies from kind biological material, how virus-specific antigens, So and virus-specific antibodies class IgM, IgG.

TEST TASKS

1. The avian influenza virus isa) to the virus influenza type FROM b) to the influenza virus type A c) to the influenza virus type B G) to virus influenza type D

2.Interferon provides antiviral protection cells, because prevents:a) virus adsorption on the cage;
b) penetration virus in cell;in) reproductions virus;
G) lysis affected cells;e) activation killers.

3. You can determine the serological type of the influenza virus using:a) agglutination reactions on the glass;
b) reactions braking hemagglutination;in) reactions indirect hemagglutination;
G) reactions hemagglutination.

4. AT pathogenesis viral diseases decisive role plays:a) virus virulence;
b) toxigenicity virus;G) level lysozyme;
e) reaction organism on the cells, affected virus.

5.HIV applies to group viruses:a) DNA-genomic;
b) RNA genomic;in) complex.

6. Family retroviruses is different presencea) RNA polymerase
b) DNA polymerases
in) endonucleases
d) reverse transcriptasee) exonucleases

7. Which type of nucleic acids contains virus hepatitis A AT?a) RNA
b) DNA
in) DNA and RNA

8.In the pathogenesis of AIDS, an important place is occupied by: a) transformation PrP^{c-} proteins in PrP^{sc} proteins;
b) unrestrained proliferation B-lymphocytes;

in) accumulation pathological myeloma proteins;G)
defeat T-helpers and macrophages.

9. AT pathogenesis viral diseases decisive role plays:a) virus
virulence;
b) toxigenicity virus;G)
level lysozyme;
e) reaction organism on the cells, affected virus.

PRACTICAL OCCUPATION No. 2.

Topic: Infectious control in dentistry. Disinfection, pre-sterilization treatment and sterilization tools, materials, equipment. Antiseptics and disinfectants. Ways fence material for researches from an oral cavity (for microbiological researches). Modern methods clinical immunology and molecular genetics.

Educational goal:

1. Explore peculiarities fence researched material from cavities mouth for holdingvarious methods of microbiological diagnosis.
2. Explore major representatives resident microflora cavities mouth.

Student must know:

1. methodology holding fence researched material from cavities mouth.
2. methodology holding various methods microbiological diagnostics.

Student must be able to:

1. cook smear and paint his by Gram.
2. Spend bacteriological study at dental diseases.

Plan lessons:

one. Features of the sampling of the test material from the oral cavity (oral fluid, dental plaque, content gingival groove, periodontal pocket, carious cavity, root channels and etc.).

Independent Work students:

1. Explore peculiarities fence researched material from cavities mouth.
2. Design protocols research.

INFORMATIONAL MATERIL ON THEME

1. sketch in protocol fence scheme researched material at complications cariesteeth and periodontitis.
2. Using the reference literature and the drawing "Microbiocenosis of the oral cavity",sketch representatives resident microflora cavities mouth at coloration on Gram.
3. results contribute in protocol.

Table. Fence researched material from content periodontal pocket

researched material	Description results microscopy	Picture

MICROBIOCENOSIS CAVITIES RTA

corynebacteria

lactobacilli

actinomycetes

Strept. mutans

peptostreptococci

spirochetes

lactobacilli

anaerovibrio

anaerobospirilla

Neisseria

leptotrichia

mushrooms candida

fusobacteria

trichomonas

amoeba

bacteroids

fusobacteria

anaerobovibrio

anaerobospirilla

spirochetes

bacteroids

veillonella

Designations:
1- dental plaque
2- microcracks and tubules enamel tooth3-
gingival groove
4- gaps mucous shells cavities mouth

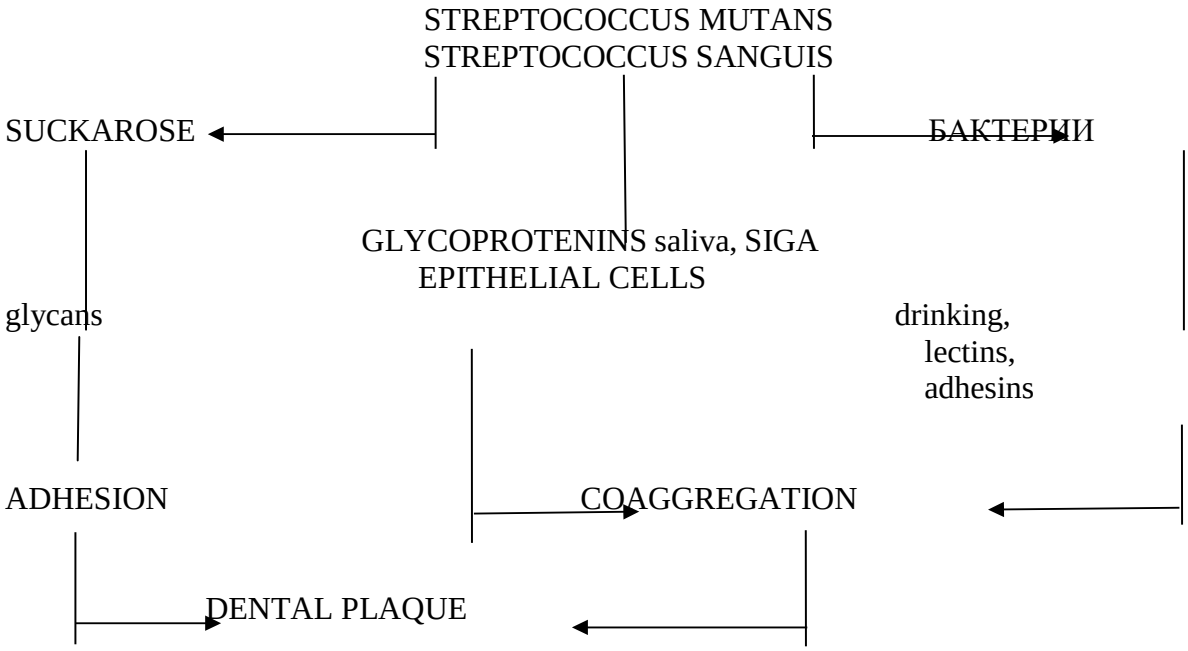
1-2. Smear from dental raid or scraping co mucous cook on the subjectglass. The material can be taken with a sterile spatula, trowel, match. The material taken from the interdental spaces or at the neck of the tooth is applied to the subject glass next to a drop of water and rub it dry, and then bring in a loop of water, gradually preparing a homogeneous suspension and evenly distributing it over the glass surface. Smear dried in air, fixed in a burner flame and stained by Gram. At microscopy under immersion study morphological peculiarities and attitude to Gram staining of representatives of the normal microflora of dental plaque biotopes and mucous language.

3. Decor protocol research.

Table. Fence researched material dental plaques

researched material	Description results microscopy	Picture

SCHEME FORMATIONS DENTAL PLAQUE (PLAQUES).



2. Microscopic examination of the demonstration clean smears cultures of bacteriaallocated from cavities mouth (lactobacilli, peptococci, bacteroids).
3. Decor protocol research.
Table. Fence researched material from horse channels.

researched material	Description results microscopy	Picture

TEST TASKS:

1) During sterilization, the following types of chemical bonds inpeptidoglycan bacterial cell wall:

a) peptide
b) glycosidicin)
hydrogen G)
covalent

2) Substances which cause delay breeding and death microorganisms innegligible small concentrations are called:

a) antibiotics b)
antiseptics c)
disinfectantsG)
preservatives

3) Complex events, directed on the destruction on/in objects pathogenicmicrobes called
a) asepsis
b) antiseptic c) disinfection d) sterilization e) tyndalization

4) pasteurization With subsequent fast cooling carry out in next mode:a) at t 100C in flow 30 seconds
b) at t 65-95C for 2-30 minutesin) at t 35-55C in flow 60 minutes G) all the answers are correct

5) If a means has detergent and antimicrobial properties, then:
a) combination of disinfection is allowed and pre-sterilizationclean-ups
b) disinfection and pre-sterilization cleaning must be carried outseparately
in) given means maybe used only for cleaning
G) given means maybe used only for disinfection

PRACTICAL OCCUPATION No. 3.

Topic: Sterilization and disinfection. Ways sterilization and disinfection laboratory crockery and medical tools. Peculiarities sterilization and pre-sterilization processing dental tools, hog, tips turbines etc .

Educational goal:

1. Familiarize With modern methods sterilization and disinfection in dentistry.
2. Familiarize yourself with the list of modern disinfectants and antisepticsdrugs.
3. Explore regulations precautions from infections infectious diseases on thereception at dentist.

Student must know:

1. methodology holding contemporary methods sterilization in dentistry.
2. methodology holding contemporary methods disinfection in dentistry.
3. Rules precautions from infections infectious diseases on thereception at dentist.

Student must be able to:

1. Conducting a microscopic examination (according to the scheme) in the diagnosisdental diseases.
2. Holding bacteriological research (on scheme) at diagnosticsdental diseases.
3. Conducting a serological study (according to the scheme) in the diagnosisdental diseases.

Lesson plan:

1. Features of microscopic, bacteriological and serological methodsresearch at diagnostics dental diseases.
2. Modern methods sterilization and disinfection in dentistry (ultrasound, UVgamma rays, laser)
3. Rules precautions from infections infectious diseases on the admission atdentist.
4. Instructions and normative the documents on disinfection and sterilization in dentistry.

Independent Work students:

1. Explore instructions and normative the documents on disinfection and sterilization in dentistry.
2. work out methodical recommendations to occupation and fill table.

3. Table

4. **Characteristic methods sterilization in dentistry**

5.

	Metho d	Apparatus	Mode	Reliability and testimony	Objects sterilization
one.	Ferry under pressure				
2.	Dry heat				
3.	Gas sterilization.				
four .	Chemical sterilization.				
5.	Ultrasound.				
b.	uv and gamma rays				
7.	laser				

INFORMATIONAL MATERIL ON THEME

In dentistry, more than in other areas of medicine, strict compliance rules asepsis and antiseptics, So how any dental intervention is performed on infected tissues. Not only the removal of carious tooth or treatment root channel, but and simple inspection cavities mouth sick connectedwith infection of the instruments used for these purposes, in order to exclude the transfer microbes from one sick in cavity mouth another a also prevent infection of healthy tissues, it is permissible to work only with a sterile instrument. Security sterile dressing material and tool - a task sisters,which must be supervised by a doctor.

I. PROCESSING OF THE DENTAL ROOMOFFICE

1. Track in office per temperature and humidity use air filters.
2. Before receiving patients, it is necessary to carry out wet cleaning using various disinfectants. Wipe 2-r. napkin every 15 minutes disinfectant solutions (3% chloramine, 6% hydrogen peroxide, 70 degrees alcohol and etc.) surfaces all items With purpose of destruction vegetative forms bacteria.

3. Then necessary turn on ultraviolet installation for destruction airborne and surface bacteria. (Calculation of a germicidal lamp at 2.5 W on the 1 cubic meters in flow 1 hour)

II. DISINFECTION, STERILIZATION IN DENTALOFFICE

1. All products that do not have contact with the wound must be disinfected. surface, blood or injections (solutions of 6% peroxides hydrogen, 3% chloramine, 70 ^{0} alcohol, etc.)
2. Sterilization - this is complete desolvation material. Sterilization must all objects in contact with the wound surface, in contact with blood and individual kinds medical tools, which in process work touch with mucous membrane and may call her damage.
3. Before sterilization, it is necessary to soak the entire tool, burs, a then clear them from protein, fatty, mechanical pollution and medicinal drugs. cleaning must produced jet, rotary methods, ruffing or With application ultrasonic baths, in which put 6 % solution peroxides hydrogen and detergent substance (powder "Lotus", "Progress" and etc.).
4. AT dependencies from sterilized material can use thermal, chemical and gas methods of sterilization. Preference should be given to thermalmethods like more reliable.

However, products made of rubber, polymers, optical equipment, some tools, devices heart-lungs, artificial bud not withstand thermal processing.

5. Sterilization ferry under pressure carried out in autoclaves. Mode sterilization allows you to destroy not only bacteria, spores, but also viruses such as hepatitis B virus (serum hepatitis) and HIV. Pressure 2 atm. (temperature 13 2 deg.) within 1 hour. Sterilization is carried out in sterilization boxes, bixes, bags from moisture-resistant paper with marking. This method recommended for products from corrosion resistant metal, glass syringes, rubber, textile materials,some polymers.

6. Some tool (especially cutting) recommended sterilize in glass-pearl sterilizer at temperature 240 deg. in flow 5-10 seconds.

7. Dry heat sterilization in dry heat ovens is carried out at a temperature 180 deg. in flow 150 minutes (2.5 hours). Duration impact also allows destroy hepatitis E and HIV viruses. Sterilization is subjected to dry products in packaging from paper (term storage twenty days). Sterilize can and without packaging, but then products must be used directly after sterilization.

8. The chemical method of sterilization consists in the fact that products are immersed in solution 6% hydrogen peroxide on 6 hours or

into a chamber with vapors of 40% formaldehyde in ethyl alcohol for several hours, which depends from the material to be sterilized.

9. Recently, in connection with the advent of new equipment, it has become more widely use the gas sterilization method. It is carried out in special chambers or tabletop gas sterilizers containing ethylene oxide or a mixture of ethylene and methyl bromide, Sterilization takes place at a temperature of 35 degrees. up to 42 degrees, duringseveral hours or days in special packages

With marking:

a) if contact with blood, tissues was less than 30 minutes, then metal products sterilize in for 4 hours, products rubber, plastics - 24 hours.

b) if contact with blood, tissues was more than 30 minutes, then metal products sterilized within 24 hours, products made of rubber, plastics - one week, the device lungs-heart-kidney in flow 2nd weeks.

Such long sterilization tied With prevention HIV and hepatitis A B.

10. FROM purpose prevention serum hepatitis A B and HIV recommended use items disposable use (syringes, injection needles, systems for blood transfusion and etc.)

Safety instructions for working with biomaterial, potentiallyinfected HIV

I. General provisions.

AIDS - disease co deadly outcome, developing in result dysfunctions of the immune system. The incubation period of the disease is 5-10 years. There are no cases of spontaneous recovery or cure from AIDS. Pathogens - T-lymphotropic retroviruses HTLV-3 (HIV-1) and HTLV-4 (HIV-2). Transmission routes - With blood (cells, serum), sexual, from mother to children with breast milk. Viruses unstable - die after a 30-minute exposure to a 20% solution of ethyl alcohol. Therefore, all measures provided to prevent infection with hepatitis viruses, sufficient to protect against infection with AIDS viruses. When dealing with infectious material necessary observe three major regulations: change robe, work in disposable gloves and more wash arms.

II. Rules work.

1. Work in the department should be in specially designed for this bathrobe. Keep them necessary in closet at entrance in branch, put on before work, take off on exit from departments.

2. All furniture and equipment in department must have plastic or metallic coating, easily amenable disinfection. On the tables must standcontainers With disinfectant solution (70% solution ethyl alcohol).

3. Tubes with biomaterial must be labeled carefully closed (traffic jams, parafilm, patch) and be delivered in unbreakable containers, easilyexposed disinfection.

4. All work, related With acceptance biomaterial and staging method, must be done with disposable gloves. All while working hand injuries must be closed (adhesive plaster, fingertip).

5. centrifugation test tubes With biosamples necessary conduct in centrifuge having individual lids on everyone glass.

6. At work With biomaterial should enjoy means, protective eyes from hits drops liquids (protective glass, shield, glasses).

7. All disposable materials, contact with the studied biomaterial (traffic jams, tips, adhesive paper, gloves) necessary straightaway same after use dump in special capacity With dez. solution (70% ethyl alcohol).

8. At the end of work, wipe all work surfaces (tables, equipment) swab dipped in disinfectant. solution. All used in production are disposable materials test test tube, gloves, traffic jams, plateau etc.) soak.

TEST TASKS

1) Arrange in correct sequences subsequence processes:a) pre-sterilization cleaning → sterilization

b) pre-sterilization cleaning → sterilization → disinfection c) pre-sterilization cleaning → disinfection → sterilizationG) disinfection → pre-sterilization cleaning → sterilization

2) Azopyrine test evaluates the quality of:a) disinfection

b) pre-sterilization cleaningin)

sterilization
G) tyndalization

3) To control the quality of pre-sterilization treatment,a) azopyrine sample
b) polymerase chain reactionin)
amidopyrine sample
G) bacteriological study

4) Fractional sterilization is used to process:a) medical
products from metals
b) dressing material
in) objects which may be nutritious substrate formicroorganisms
G) liquid medicines

5). At disinfection products medical destination boiling in distilledwater With 2% bicarbonate sodium (soda) exposure is:
a) at least 5 minutes
b) at least 10 minutes
c) at least 15 minutes
G) not less 40 minutes

PRACTICAL OCCUPATION No. 4.

Topic: Microbiocenosis of the oral cavity. Resident microflora of various biotopes oral cavity. Plaque and its study in assessing the hygienic condition oral cavities.

Educational goal:

1. Explore major representatives resident microflora cavities mouth.
2. Explore microflora dental raid.

Student must know:

1. The method of sampling the test material from the dental for carrying outbacteriological method diagnostics.
2. Compound microflora dental raid.

Student must be able to:

1. Spend fence material from dental raid.
2. Spend bacteriological study dental raid.
3. cook smear and paint his on Gram.

Plan lessons:

1. Symbiosis, stages symbiosis.
2. Cavity mouth how ecological niche organism.
3. Main representatives resident microflora cavities mouth, them properties.
4. dental plaque. Mechanism her formations. Localization.

Independent Work students:

1. sketch in form scheme morphology main residents cavities mouth:
    1) anaerobic gram-positive (Peptostreptococcus, actinomycetes, propion- and eubacteria) and gram-negative (veillonella, bacteroids, fusobacteria, tortuous forms);
    2) aerobic and facultative anaerobic gram-positive (streptococci, staphylococci,

Korine- and lactobacilli) and gram- negative (neisseria, pseudomonas),

2. Decor protocol research.

INFORMATIONAL MATERIL ON THEME

Species compound microbial flora cavities mouth in norm enough constant. TogetherWith topics amount microbes in cavities mouth susceptible significant fluctuations. AT the present time described several hundreds species microorganisms, constituents normal microflora of the oral cavity. It contains bacteria, viruses, fungi and protozoa.

Quantity microbial flora depends from hygienic content cavities mouth, smoking promotes reproduction microorganisms, causes chronic inflammation mucous shells. Solid food more affects on the decrease the number of microbes, because chewing promotes mechanical cleaning of the oral cavity from microorganisms. Disorder salivation, chewing and swallowing always leads to rising quantity microorganisms in oral cavities.

Availability carious cavities, gingival pockets, poorly stocked dental fixed prostheses and others condition enough high frequency formation foci chronic infections With subsequent allergization organism and high degree risk development general autoimmune diseases.

Microflora cavities mouth newborn presented in mostly milky - sour chopsticks, non-hemolytic streptococci and non-pathogenic staphylococci. Her enough fast (in flow weeks) replace microorganisms, characteristic for cavities mouth adult person.

Main inhabitants cavities mouth adult human are bacteria, predominantly (3/4 all microbial species) anaerobic type breathing. Among them meet various cocci, sticks, tortuous forms.

Despite the wide variety of microorganisms in the oral cavity, quantitatively in it is dominated by microbes of three groups: about half are facultative and obligate anaerobic streptococci, and the other half consists of veillonella (less than 1/4) and diphtheroids (less than 1/4). The remaining numerous groups of bacteria are staphylococci, lactobacilli, flagella, spirochetes, leptospira, fusobacteria, bacteroids, neisseria, hemophilus, mycoplasmas, yeast, protozoa - present yourself small populations on quantity, but equal groups on formation associations residents.

Table

Microflora cavities mouth in norm.

	AT saliva		**AT dental gums pockets (frequency detection in %)**
Group A: Residential flora 1. Aerobesand			
Streptococcus salvarius	100	10^7	100
[illegible]	100	10^6-10^8	100
Sapropliyuc e Neisseria	100	10^5-10^7	++
Lactobacillus	90	10^3-10^4	+
Staphylococci	80	10^3-10^4	-n-
Diphtheroids	80	Not def.	+
Hemophiles	60	Not def.	0
pneumococci	60	Not def.	Not defined
and others	thirty	10^2-10^4	++

Saprophytic e	++	Not def.	++
Tetracocci	++	Not def.	++
yeast-like s mushrooms	fifty	10^{2}-10^{3}	+
Mycoplasmas	fifty	10^{2}-10^{3}	+
Protozoa: Entamoesa gingivalis	0	0	45
Trichomonas	0	0	25
II. **Obligatory** **anaerobes**	100	10^{6}-10^{8}	100
Anaerobic streptococcus			

(peptostrep tococci)	100	He def.	100
Bacteroids	100	He def.	100
Fusobacter ai	75	ioMo4	100
filiform bacteria	100	102-104	100
Actinomyce you and anaerobic diphtheroids	100	He def.	++
Spirilla and vibrios	++	He def.	.one. ++
Spirochetes (saprophytic	±	He def.	100
Group B. Fickle	fifteen	I0-102	. 0
Klebsbrieila	2	10-I02	+
Esclierichia	3	10-102	0
Aerobacter	±	He def.	0
Pseudomona s	±	He def.	0
Proteus	±	He def.	0
Alcaligenes	±	He def.	0
bacilli			
II. obligatee anaerobes			
Clostridia	+	He def.	0
Cloatridium putrificun	±	He def.	0
Clostridium perfringens	+	He def.	0

Ob about h n a h en and e: ++ ha With t about, + ne about h enb ha With t about, ± rarely, 0 not discovered.

To about l and h e With t in about m and to R about b about in in P about l about With t and R t a n e about d and n a to about in about in R a h n s X e G about uh to about l about G and h e With t o and X n and sh a X: With l and h and With t about th about b about l about h t o e, h about n e de With n e in about G about and e l about b t o a, P R about t about t o a X With l Yu n n s X and e l e h, With l Yu n e and R about t about in about th and and d to about With t and, hat b n about th bl Ish t o e.

T a to, n a P R and m e R, With about d e R and a n and e b a t o t e R and a l b n s X to l e t about to in With l Yu n e (R about t about in about t h and and d to about With t and) With about With t a in l I et about t 50 m l n. d about 5 m l R d., P R and h e m b about l b sh and n With t in about b a t o t e R and th P about P a d a e t in With l Yu n at With about With P and n to and I h s to a. AT hat b n about m n a l e t e (bl Ish t o e) m and to R about b about in h n a h and t e l b n about b about l b sh e: about t one00 d about one000 m l R d. in GRamme mate Randala. FROMamat Yub about l b sh at Yu GR at PP at With Red and in With t R e h a Yu sch and X With I in P about l about With t and R t a b a t o t e R and th With about With t a in l I Yu t to about t o to and. (T a bl and c a 2).

Tablandca.

O With n about in n s e G R at P P s R e h and d e n t n about th f l about R s P about l about With t and R t a P about m about R f about l about G and and and t and P at d s X a n and I.

Coloring on Gramu	Morphology	Food kind
one GROUP: FROM anaerobic type breathing (obligate anaerobes)		
Gram-negative	cocci	VEILLONELLA
	sticks	BACTEROIDES PORPHYROMONAS PREVOTELLA FUSOBACTERIUM LEPTOTRICHIA
Gram positive	cocci	PEPTOSTREPTOCOCCUS PEPTOCOCCUS
	sticks indisputable	LACTOBACTERIUM BIFIDOBACTERJUM EUBACTERIUM PROPIONIBACTERIUM ACTINOMYCES
	sticks spore-forming	CLOSTRIDIUM
2 GROUP: FROM aerobic and mixed type breathing (aerobes andoptional anaerobes)		
Gram-negative	cocci	NELSSERIA
	sticks	PSEUDOMONAS BORDETELLA EIKENELLA
Gram positive	cocci	STREPTOCOCCUS STAPHYLOCOCCUS
	sticks indisputable	CORINEBACTERIUM NOCARD1A, ROTHIA
	sticks spore-forming	BACILLUS

Staphylococci. Division Firmicutes, family Micrococcaceae, genus Staphylococcus. AT genus Staphylococcus on classification Baird— Parker are included 3 type: S. aureus, S. epidermidis and S. saprophyticus. Recently proposed other classifications include more species of staphylococci, but they are used so far only in scientific research.

All types of staphylococci are rounded cells with a diameter of 0.5-1 μm. In a smear, they are usually arranged in asymmetrical clusters ("clusters of grapes"), but there are single cells, pairs of cells. Gram-positive. Dispute does not form flagella not have. At some strains can discover capsule. Can form L-shapes. The cell wall contains a large amount of peptidoglycan, related With him teichoids acids, protein BUT.

Staphylococci grow well on simple media (pH 7.0-7.5); optional anaerobes. On dense media they form smooth round convex colonies with different pigment. The pigment has no taxonomic value. Can grow on agar with juice content (8-10 %) NaCl. produce saccharolytic and proteolytic enzymes. Staphylococci produce hemolysins, fibrinolysin, phosphatase p-lactamase, bacteriocinins, enterotoxins, coagulase dna-ase,

leukocidins, lecitovitellase and others

Staphylococci very plastic: fast develop sustainability to antibacterial drugs. Substantial role in this play plasmids, transmitted With help transducing phages from one cells to another. R- plasmids determine sustainability to alone or several antibiotics, in volume including and per check extracellular products p-lactamase — enzyme, destructive penicillin, tearing his p-lactam ring.

Pathogen staphylococcal infections happens more often S. aureus, somewhat less often - S. epidermidis, very rarely - S.saprophyticus. Staphylococci are representatives normal microflora human body, that's why microbiological diagnostics staphylococcal infections not maybe confine highlighting and identification pathogens; necessary quantitative methods research, t. e. definition numbers microorganisms in sample.

Staphylococci in the oral cavity of a healthy person are found on average in 30% cases. In plaque on the gums of healthy people are present in mostly Stapf. epidermidis. Much more often pathogenic staphylococci are localized on the mucous pharynx and nose, causing the so-called "healthy bacteriocarrier". Possessing enzymatic activity, staphylococci take part in the breakdown of residuesfood in the mouth. Such permanent carriers of pathogenic staphylococcus are source airborne infections. Pathogenic staphylococci, encountered on the nasopharyngeal mucosa and in the oral cavity are a common cause of autoinfection, causing various purulent-inflammatory processes cavities mouth.

Treatment staphylococcal infections usually carry out antibiotics and sulfanilamide drugs. AT recent years from sick often allocate staphylococci resistant to most chemotherapy drugs. Such cases, antitoxic anti-staphylococcal plasma is used for treatment or immunoglobulin obtained from the blood of immunized donors staphylococcal toxoid. For active immunization (planned surgical sick, pregnant women women) maybe to be used adsorbed staphylococcal toxoid.

Streptococci. Department firmicutes, family streptococcaceae, genus Streptococcus. AT genus Streptococcus are included more twenty species, among which there is representatives normal microflora human body and cavities mouth, a also pathogens heavy infectious epidemic diseases person.

streptococci — small (less one μm) spherical cells, located chains or in pairs, Gram-positive dispute not form, motionless. Majority strains streptococci form capsule, consisting from hyaluronic acids. Cellular wall contains squirrels (M-, T- and R antigens), carbohydrates (group specific) and peptidoglycans. Easily go over in L-shapes.

Genetic exchange available per check transformation and transduction, but not conjugation. Sustainability to antibiotics produced slowly.

streptococci groups BUT develop more twenty extracellular substances possessing antigenic activity. Greatest meaning in pathogenesis streptococcal infections have:

- streptokinase (fibrinolysin) — proteolytic enzyme, splitting fibrin and other proteins;
- DNAase — enzyme, depolymerizing DNA. Mixture DNAases and fibrinolysin is able to liquefy exudates, lyse venous thrombi, so maybe to be used for removal pus and necrotic fabrics from wounds;
- hyaluronidase — enzyme aggression destructive hyaluro-new acid, incoming in compound connective fabrics ("factor permeability");
- erythrogenin — toxin, produced p-hemolytic

streptococci groups BUT, able call scarlet fever. stands out only lysogenic cultures.

Standardized diluted erythrogenin use at staging intradermal test (Dick test) for identifying sensitivity to this toxin (susceptibility to scarlet fever).

streptococci are main inhabitants cavities mouth. IN 1 ml saliva contains up to 10^8 - 10^9 streptococci. However, in the samples their saliva at about 2 times more than in plaque or gingival groove material. most significant group streptococci cavities mouth should count microaerophilic a- hemolytic ("green") streptococci and j - non-hemolytic forms. Should Mark, what from 40 - 90 % strains kind milled may to be B-hemolytic, which take an active part in the processes leading to lesions of solid tissues of the tooth and periodontium. This group includes Streptococcus mutants, S. sanguis, S. mitis, S. salivarium. They are differ from each

other in their ability to ferment carbohydrates and form hydrogen peroxide.

Shift pH in sour side leads to decalcification dental enamel. Especially should emphasize high capabilities microaerophilic streptococci to aggregation With others bacteria which shown, in in particular in respect actinomycetes, fusobacteria, lactobacilli. All this contributes to the detection of these species in the composition associations of pathogens in various purulent-inflammatory processes in the maxillo- facial area. But their role in the development of caries is especially significant. Leading position in This plan is occupied by two species that actively produce milk and milk from food carbohydrates. other acids on the enamel -S. mutans and S. sanguis.

All types of streptococci grow poorly and die on simple nutrient media, because in the process of growth and reproduction, streptococci secrete a lot of hydrogen peroxide, which has a detrimental effect on them, tk. they do not produce catalase. For creating optimal conditions growth, blood is usually added to the nutrient medium, in which contained catalase, destructive peroxide hydrogen. On the blood environments streptococci grow well under aerobic conditions, while some of them form on blood agar colonies surrounded by a zone of complete hemolysis (these are hemolytic (B) streptococci), other surrounded zone greenish colors (green streptococci), at third (y) hemolysis missing (non-hemolytic streptococci).

streptococci allocate exotoxin and enzymes aggression. In external environment less resistant, how staphylococci. Majority sensitive to penicillin and others antibiotics. By antigenic structure all streptococci divide on the 17 serological groups (A,B,C,D, ... and before S), bowl others in cavities mouth are found streptococci groups BUT, FROM, D, F, g, H and O.

Peptostreptococci - Gr + obligate anaerobic cocci, which include two kind - Peptostreptococcus and Peptococcus. Wide presented in all niches cavities mouth. More often Total peptococci found in association with fusobacteria and spirochetes at caries, pulpitis, periodontitis, abscesses maxillofacial areas.

Veillonelles - this is obligate anaerobic, Gr -, small cocco -bacteria, motionless, dispute not form. Are permanent inhabitants cavities mouth human and animals. Isolated colonies on the lactate agar have 1-3 mm in diameter, smooth, convex, lenticular, diamond-shaped or cardiac forms, yellow-white, soft in consistency. Representatives are found in the oral cavity two species of veillonella (V. parvula, V. alcalescens), which inhabit the mucous membrane cavities mouth, palate, are dominant in saliva and ducts salivary glands. Good ferment acetic, pyruvic and dairy acid, neutralizing sour products metabolism others bacteria, this is allows consider veillonella how the most important factor, resistance to caries teeth. pathogenic role veillonella not proven.

diphtheroids , or corynebacterium, present yourself group bacteria, quantitatively comparable With veillonella.

it polymorphic gram positive sticks, located orderly ("palisade" or groups) in a smear from a pure culture. Some types of microbes able form inclusion - grain volutin.

Classification diphtheroids cavities mouth before present time remains undeveloped. At research material diphtheroids often difficult differentiate from actinomycetes and propionibacteria. optional anaerobic kinds diphtheroids constitute approximately 13% of the number of residents, isolated from the back of the tongue, 15% from the gingival groove and 24% from dental plaque. Representatives of diphtheroids with an obligate anaerobic type of respiration make up in these materials respectively eight, twenty and eighteen%.

Diphtheroids play important role in cavities mouth as a stabilizing factor oral microbiocenosis, So how synthesize vitamins, in in particular vitamin TO, being stimulant growth anaerobic bacteria. Reducing in process breathing molecular oxygen, they actively promote development obligate anaerobic flora in aerobic conditions.

Shown powerful immunomodulatory activity antigens diphtheroids (corynebacteria) on the organism human, what used at treatment immunodeficiencies. Together With topics at corynebacteria discovered some enzymes aggression and toxic polymers, they often are found in associations With pathogens purulent inflammation.

Lactobacillus constantly are in cavities mouth, motionless, dispute and capsules do not form, Gr + are characterized by high polymorphism. Grow up on electives nutritional environments contain such factors growth, how vitamins and some amino acids. grow up in form small, colorless, compacted colonies. Possess rather low adhesive properties to the mucosal

epithelium and especially to enamel tooth, but presented in all niches cavities mouth. Stormy multiply at admission in cavity mouth carbohydrate food and plentifully produce dairy and other acids, which allows them to be considered as a cariogenic factor. Together with topics lactobacilli play the most important stimulating role at formation microbial associations cavities mouth, So how synthesize vitamins groups AT and TO, necessary for development others bacteria and organism.

In view of education big quantity dairy acids in process vital activity lactobacilli, they detain growth others microbes: staphylococcus, intestinal sticks, typhoid and dysentery sticks. Antagonistic properties of lactic acid bacteria in relation to a number of putrefactive microbes were noticed yet I.I. Mechnikov, which the proposed use curdled milk made from milk fermented with lactic acid sticks. Before 90% of the lactobacilli living in the oral cavity belong to the species Lactobacterium casei, Lactobacterium fermenti.

actinomycetes - presented small Gr + chopsticks, having trend to education intertwined and branching threads or shorter chains. Actinomycetes are located on the mucous membrane of the mouth, make up the stroma of the dental stone and are part of dental plaque. Along with this, they are contained in carious cavities of teeth, in pathological gingival pockets, in ducts salivary glands.

Representatives of this family can take part in the formation of dental plaques and in development caries teeth, a also diseases periodontium. AT cavities mouth there are favorite places of penetration of actinomycetes into the depths of tissues - inflamed gums near the wisdom tooth or near the destroyed roots of the teeth, pathological gingival pockets at periodontal disease, root channels teeth With dead pulp tonsils.

For occurrence diseases not enough only introduction actinomycete deep into fabrics, certain role plays and condition protective strength, downgrade resistance organism to infections.

Bacteroides - represent a group of coccoid, ovoid or polymorphic rod-shaped Gr - bacteria. FROM 1990 of the year divided on the three kind: Porpluiroman (representative - P. Gingivalis inhabit gingival groove, dental plaque), Prevotella (the most important species - P. Melaninogenica inhabits pockets of the mucous membrane, fissures of the tooth, gingival groove), Bacteroides (representative - AT. Fragilis meets in folds mucous at the base teeth, but more typical for intestines. For growth on nutritional environments this microorganisms needed hemotin and vitamin TO. On the bloody agar AT. melaninogenicus shapes black colonies. Availability proteolytic. enzymes in bacteroids (collagenase, hyaluronidase, heparinase, Jg A -; Jg W-; Jg M - protease) It has big pathogenetic meaning in disease development periodontal.

Fusobacteria - elongated Gr - sticks, more often With pointed ends, often formative chains and threads. inhabit how mucosa mouth, So and dental plaque.

Fusobacteria produce powerful histolytic enzymes - hyaluronidase, lecithinase, have endotoxin. Along With bacteroids and peptococci considered main pathogens diverse purulently - inflammatory processes in cavities mouth, including ulceratively -necrotic fasciitis.

Neisseria - genus Neisseria - Gr - diplococci, detectable in various niches cavities mouth, especially on the surfaces which constantly touch With air - back language, soft sky, enamel teeth. pathogenic role them not proven.

Yeast-like mushrooms in cavities mouth healthy of people meet in 40 - 50% of cases. Candida are oval or elongated cell size 7 - 10 micron, often bud off new cell. Aerobe grow up on the environment Saburo, containing yeast extract and maltose, where convex colonies of opaque colors.

The most common species found in the oral cavity are: Candida albicans, Candida tropicalis, Candida crusel. Pathogenic properties are most pronounced in C. aldicans. Mushrooms cause a general disease of the body - candidomycosis or local damage to the cavity mouth - "milkmaid".

Spirochetes - inhabit the oral cavity from the moment of eruption of milk teeth in a child and from that time become permanent inhabitants oral cavity. Gr -, mobile, strict anaerobes, grow on media containing serum, ascitic liquid With adding fresh pieces various bodies, on the environments form turbidity in the form of a cloud. High proteolytic activity, liquefy gelatin, egg protein, folded serum form indole, hydrogen sulfide, ammonia.

They are easiest to detect in the dark field of view with microscopy of native drug.

Spirochetes cause pathological processes in the oral cavity with significant reproduction all anaerobic microorganisms.

Protozoa cavities mouth - meet at fifty % healthy of people, predominantly in dental on the fly, crypts tonsils (Entamoeba gigivalis). They multiply with unhygienic maintenance of the oral cavity. They are found in pus from gum pockets in severe alveolar pyorrhea D - 20 - 30 nm. aerobes, mobile, visible better in native unpainted preparation (crushed a drop). Grow on blood or serum agar, drenched in a layer of Ringer's liquid and with adding solution tryptophan (1 : 10000).

Much more often, how amoeba, in cavities mouth healthy of people meet Trichomonas. Weak mobile, Good visible in native preparation, in alive condition.

At staining on Romanovsky - Giemse nucleus blepharoplast and flagella stained in red color, protoplasm - in blue. Reinforced reproduction Trichomonas going on just like amoeba, with unhygienic maintenance of the oral cavity. in a very large quantity they are found at periodontitis, at gingivitis.

Viruses oral cavity . Nearly at all healthy of people in oral cavity the herpes virus (Herpes vilgaris) is constantly present. This virus is transmitted even in childhood by airborne droplets from adult virus carriers. herpes vilgaris belongs to the group of DNA-containing herpesviruses, size 150 nm. Grown on chorionallantoic shell of the chick embryo.

At weakening protective forces macroorganism in result colds, overwork and etc. possible relapse illness.

Clostridia. Genus *Clostridium* - gram positive spore-forming sticks. Some kinds mobile thanks to availability flagella. Biochemically they active. AT norm are included in compound microbiocenosis intestines. AT cavities mouth determined some kinds not always.

stand out at sick With purulent wounds maxillofacial areas, rarely - at odontogenic inflammatory processes. At pollution wound surfaces and extensive traumatization fabrics Maybe development exogenous clostridial anaerobic infection, clinical manifestations which correspond classical picture gas gangrene. Main kinds: *FROM. perfringens, FROM. septicum, FROM. clostridiiforme, C. bifermentans* (the latter is found in odontogenic inflammatory processes).

other residents. Among bacteria with aerobic type breathing in cavities mouth meet also representatives actinomycete lines - nocardia and rotia (*Rothia dentocariosae),* which, possessing high adhesive and coaggregative properties, contribute formation dental plaques. Last view often determined in carious cavities and fistulas at actinomycosis, a also at non-specific osteomyelitis maxillofacial areas.

Non-fermenting gram-negative bacteria cavities mouth presented childbirth *pseudomonas, Bordetella, Eikenella (E.corrodens)* and some others. Among them most known bacteria *Pseudomonas (Actinobacillus) actinomycetemcommitam,* which violently develop at some young of people, causing progressive purulent juvenile periodontitis. Their role in the development of periodontitis adults in the present time being studied.

dental plaque. Her meaning in development caries teeth.

Using scanning electron and immunoluminescent microscopy shown what dental plaque consists in mostly from microbes With insignificant inclusion structureless substances organic nature.

AT formation dental plaques can highlight several major mechanisms.

1. Adhesion to enamel epithelial cells that are invadedbacteria With subsequent growth microcolonies,
2. precipitation extracellular glycans, produced S. mutans and S. Sanguis. 3 . Precipitation of salivary glycoproteins forming a pellicle with subsequent specific adhesion to her bacteria.
4 . Agglutination of bacteria with antibodies followed by fixation onsurfaces enamel.

Using immunoluminescence microscopy _ shown what bacteria in dental plaque covered immunoglobulins classes BUT and G.

dental plaque starts form already in first minutes after purges teeth, and in the dynamics of its formation there are significant changes character microbiocenosis. General trend is change composition flora from dominance aerobic and facultative anaerobic forms, predominantly gram-positive cocci, to obligate anaerobic gram negative chopsticks and tortuous forms.

1 phase formation dental plaques - first 1-4 hours after careful purges teeth (or processing ultrasound on the apparatus "Piezon-master"). She is predominantly consists from cocci (streptococci, neisseria, veillonella) and short sticks (diphtheroids). This, So called, "early" dental plaque.

2 phase - up to 4 - 5 days. Characterized by a decrease in the proportion of gram-positive cocci and growth shares gram-variable filiform forms - leptotrichian, a also gram-negative veillonella and fusobacteria. This phase maybe to be characterized how "balanced" or "dynamic" dental plaque. At persons With good adaptive abilities, With So called "high natural sanitation" microbiocenosis of dental plaque can be maintained in this able on the throughout significant segments life, not passing to next phase (when absence systematic purges teeth).

3 phase - from 6 - 7 days and Further. dental plaque accepts final on composition symbionts view, although quantitative shifts in her are happening constantly. The number of aerobic species - Neisseria, Rothium, facultative anaerobic streptococci. Dominated by gram-negative obligate anaerobic bacteroid bacteria, fusobacteria, veillonella and gram-positive actinomycetes, microaerophilic streptococci and peptostreptococci. it "mature" dental plaque. It characterizes the negative hygienic state cavities mouth and maybe induce development gingivitis in persons who are not regularly clean teeth.

Total number of bacteria in the dental plaque increases from 100-5000 in I phase formation before one - ten mln/g in 2 phase. AT 3 phase formations, in dependencies from many factors amount bacteria calculated dozens and hundred billion in one G.

established, what microbes possess different ability to adhesion even in respect various surfaces tooth. Except Togo, on the process adhesion affect and mechanical factors related with the process chewing, physical and chemical conditions andetc. Therefore, on different surfaces of the teeth, in the pits and fissures, the composition of the microflora several is different, even in within one tooth.

These data are of great practical importance due to the fact that condition dental plaques, how known is key mechanism occurrence and development caries teeth.

AT the present time established, what after reception food, especially rich carbohydrates in oral liquids going on sharp gain enzymatic activity bacteria - "metabolic explosion". basis "metabolic explosion" is activation glycolysis, what leads to sharp shift pH environments in sour side per check ejection sour catabolites - acetic, dairy, formic, pyruvic and others acids.

AT my turn, this is leads to exit ions calcium from solid fabrics tooth (demineralization), a also decrease content phosphates in process phosphorylation at bacteria. Except Togo, bacteria dental plaques accumulate excess carbohydrates in the form of reserve polysaccharides - dextrans and levans. In patients caries products organic acids much above, a normalization metabolic activity going on slower.

AT recent years installed role some resident-participants microbiocenosis dental plaques how antagonists cariogenic streptococci.

Before Total this is applies to veillonella - gram negative anaerobic coccam, which actively dispose of acids. it allows to consider veillonella how the most important microecological factor caries resistance.

dental plaque formed also and on the surfaces seals, and its composition somewhat different and depends on the nature and quality of the filling material. The most richly represented microbial flora on the cements and amalgams. Average level colonization typical for macrocomposite filling materials. And finally, on microcomposite and hybrid materials, dental plaque formed poorly due to the low affinity of bacteria. Usually in plaque microcomposite fillings are determined only microaerophilic streptococci and actinomycetes in small quantity.

These data have important practical meaning in connections With because condition dental plaques, how known is key mechanism occurrenceand development caries teeth.

AT the present time established, what after reception food, especially rich carbohydrates in oral liquids going on sharp gain enzymatic activity bacteria — "metabolic explosion". basis "metabolic explosion" is activation glycolysis, what leads to sharp shift pH environments in sour side per check ejection sour catabolites - acetic, dairy, formic, pyruvic acids.

AT my turn, this is leads to exit ions calcium from solid fabrics tooth (demineralization), a also decrease content phosphates in process phosphorylation at bacteria. Except Togo, bacteria dental plaques accumulate excess carbohydrates in form reserve polysaccharides - dextrans and Levanov.

For study composition dental plaques use methodology taking material probe, metallic spatula or swab With subsequent weighing on the analytical scales. After this, in dependencies from tasks research, carry out mechanical trituration plaques or her disintegration ultrasound and quantitative sowing With using technology anaerobic cultivation. Quantity bacteria express in colony-forming units (CFU) in gram material.

Identification dedicated cultures before kind and kind allowed reveal significant differences in the proportion of bacteria of different genera in dental plaque and mucosa membranes of the oral cavity. So, in the composition of the dental plaque dominated by the frequency of excretion actinomycetes (20.4 *%)* and anaerobic cocci kind *Peptostreptococcus* (17.2 %)- By comparison With frequency allocation co mucous shells in dental plaque It was also much more alpha green streptococci (nearly in 3 times), lacto- and bifidobacteria (in 9 once). bacteria groups bacteroids and kind *Fusobacterium* on frequency allocation competed With dominant flora (14.7 and 5.7%), but their share was significantly less than in allocation co mucous shells cavities mouth.

At identification strains microorganisms, dedicated co mucous shellsof the oral cavity, the dominant flora in terms of frequency of occurrence were non-spore-forming gram- negative anaerobes of the bacteroid group (23.7%) and the genus *Fusobacterium* (14.9%). Whereinit should be emphasized that bacteroids were isolated almost 2 times, and fusobacteria - 2.5 times more often,how from dental plaques. Peptostreptococci also enough often stood out co mucous(15.8%), although somewhat less frequently than from dental plaque. The frequency of allocation of other cocci - veillonella, peptococci, microaerophilic streptococci and staphylococci - practically not different in dental plaque and co mucous.

drew on the myself Attention much more low frequency allocation co mucous shells actinomycetes, alpha streptococci and lactobacilli. Share facultative anaerobic and aerobic bacteria in dental plaque and on the mucous practically not differed.

TEST TASKS

1) The quantitative ratio of residents in the ecological niche is determined by…a – presence at residents invasive factors
   b - the presence of infectivity factors among residentsin
   - state protective forces organism
   G - toxigenicity residents
2) After teething, a significant amount of ...a - Neisseria and hemophilus
   b - bacilli and Clostridium
   c - lactobacilli and corynebacteriaG -
   bacteroids and tortuous forms
3) The cariogenic action of bacteria at night is realized due to ...a – the presence of cell wall lectins
   b - polymerase productionin
   – synthesis glycans
   G - education capsules
4) Under "metabolic explosion" in cavities mouth understand...
   a - a sharp increase in glycolysis and phosphorylation after eatingb – degranulation immunocompetent cells
   in - activation complement alternative way
   G - release enzymes aggression and toxic metabolites microbes

5) Factors non-specific resistance oral liquids are…a – circulating immunoglobulins

b - secretory immunoglobulinsin –
myeloperoxidase saliva
G – T-lymphocytes

PRACTICAL OCCUPATION No. 5.

Topic: Cariogenic microflora. Microbiological methods of study microflora at caries teeth and his complications. Computer karyogram.

Educational goal:

1. Familiarize With methodology fence material at caries for bacteriologicalmethod research.
2. Explore microflora at caries.
3. Consider the role of microflora in the emergence and developmentcaries

Student must know:

1. methodology fence researched material from carious cavities for holding bacteriological method diagnostics.
2. Compound microflora cavities mouth at caries teeth.

Student must be able to:

1. Spend fence material from carious cavities.
2. Spend bacteriological study at caries teeth.
3. cook smear and paint his on Gram.

Plan lessons:

1. Peculiarities microflora cavities mouth at caries teeth.
2. Streptococcus mutans and his role in occurrence caries.
3. experimental confirmation roles microbes in development caries.
4. Role local factors resistance at caries. Vaccine for preventioncaries.
5. Features of sampling material from the carious cavity for bacteriological method research.

Independent Work students:

1. Familiarize yourself with the features of taking material for caries forbacteriological method research.
2. Microscopic examination of smears from pure cultures of cariogenicbacteria and them antagonists:

a) demonstration smear from a pure culture of Streptococcus mutans. okr.gentian violet;

b) demonstration smear from a pure culture of actinomycetes. okr. gentian violet;in) demonstration smear from clean veillonella cultures. okr. magenta.

3. Method for quantitative determination of cariogenic flora on an examplelactobacillin test.
4. decor protocol research.

INFORMATIONAL MATERIL ON THEME

Caries - this is pathological process, at which going on demineralization andsoftening solid fabrics tooth With subsequent education cavities.

AT the present time known what carious process maybe develop atnext conditions:

1. Availability sufficient quantity carbohydrates in food;
2. Availability microorganisms in cavities mouth;
3. Contact carbohydrates and microorganisms With teeth.

bright proof roles carbohydrates in occurrence carious process are conducted experimental research. All cariogenic diets contain more than 50% sucrose. Content in diet of experimental animals smaller amounts of carbohydrates either does not cause a carious process,

or it develops slowly.

There is now strong evidence that without contact teeth With carbohydrates carious process not arises. Undoubtedly important role belongs to the composition and structure of tooth enamel, saliva, as well as the nature of nutrition, composition drinking water. The study microbial flora at caries gave possibility install certain subsequence penetration various species microorganisms in fabrics carious tooth, a also reveal shifts in composition all microbial flora of the oral cavity in caries. Microorganisms are primarily penetrate into the enamel of a carious tooth after the destruction of the structure of all its layers. At initial lesions are found also microorganisms, which With points vision them biochemical activity may to be subdivided on the two groups: proteolytic and acid-forming.

The proteolytic group includes bacteroids and peptococci. They work out enzymes, capable split organic substances carious tooth.

To acid-forming group relate streptococci, lactobacilli and actinomycetes. Of the streptococci, enterococci are most often present here. All these microorganisms may participate in process demineralization solid fabrics carious tooth, because they intensively split carbohydrates and form a lot of organic acids.

All representatives of the permanent flora of the cavity are present in the carious cavity. mouth, main way strict anaerobes. On the cariogenic activity oral microorganisms are affected by saliva - its aggregating factors, which, on the one hand, contribute attachment microbial cells to surfaces tooth, a With another - delete them at washing cavities mouth.

The system of buffers has an anti-carious effect, bicarbonate - carboxylic acid, as well as protein and burn, which are in saliva. Prevention of caries be aimed at reducing the number of cariogenic microorganisms in the cavity mouth. Effective application various bactericidal and bacteriostatic drugs. Good results are obtained with antiseptics, in particular 0.2% chlorhexidine. At this amount cells S. mutans in dental plaques declining on the 80-
85 %, a in saliva on the 55 %. covering dental surface, chlorhexidine not only has a bactericidal effect on microorganisms, but also prevents their adhesion, does not disturbing the microbial balance. inhibitory effect on microorganisms has fluorine and his connections. Another path decrease acid formation and accumulationglucans - replacement sucrose others carbohydrates at enzymatic splitting which these products not are formed.

At fence material at caries for holding bacteriological researchnecessary stick to some rules:

a) for eliminate access saliva in carious cavity necessary isolate tooth,for what his cover with cotton rolls;
b) superficial layers softened dentine necessary delete sterileboron;
c) deep layer softened dentine take away sterile excavator andproduce sowing;
d) inoculation is carried out on appropriate nutrient media for isolationanaerobic and aerobic microflora.

2. Spend microscopy demonstration swabs from pure cultures Streptococcus mutans, actinomycetes, veillonella under immersion.

3. Lactobacillin test produce next way:

unstimulated saliva collect on an empty stomach in sterile test tube. Cooking serial dilutions of saliva under sterile conditions from 1:10 to 1:10 0.1 ml from each dilution is sown on a dense elective nutrient medium with a low pH, rubbing material spatula throughout agar surface.

After incubation at 37 hail.produce count quantity colonies, grown on the surfaces environment and recalculation for overall volume saliva.

For example, at breeding saliva 1:10 has grown ten colonies, means in 0.1 ml undiluted saliva contains 10 000 cells, and in 1 ml - 100,000 cells.

Exists certain addiction between quantity brown" sogenicbacteria in saliva (lactobacilli, streptococci) and them antagonists (Veyalonell), what allows give the following recommendations:

a) use fluoride preparations for the prevention of caries;b) restrict use in food carbohydrates;
in) strengthen hygiene oral cavity.

4. Decor protocol research:

Table. Fence researched material from carious cavities.

researched material	Description results microscopy	Picture

TEST TASKS

1) Aerobic bacteria that are antagonists of cariogenic flora can count…
   a - Neisseria
   b - veillonella
   c - Haemophilus influenzae
   G – fusobacteria

2) The main factor of infectivity in Str. mutans is…a - education hemolysin
   b - adhesins cellular walls
   c - dextrans produced during the utilization of sucroseG - lactic acid

3) According to WHO, the group of cariogenic microbes includes ...
   a - S. mutans S. sanguis, lactobacterium, Actinomyces
   b - S. sanguis, Fusobacterium, actinomyces, E. corrodens
   in - S. mutans, S. sanguis, bacteroides, R. dentocariosa, Neisseria
   G - lactobacterium, Bifidobacterium, Propionibacterium
4) The mucosal associations in the back of the tongue are characterized by dominance ...
   a - leptotrichia and candida fungi
   b - streptococci, in particular Str. salivarius
   c - filamentous forms, in particular roti R. dentocariosaG - bacteroids and fusobacteria

5) From the point of view of the occurrence of caries, antagonists are ...a - streptococci and veillonella
   b - streptococci and actinomycetes
   in - streptococci and bacteroids
   G - mushrooms and spirochetes

PRACTICAL OCCUPATION No. 6.

Topic: Periodontopathogenic microflora. Microbiological methods study microflora at diseases periodontal. Tactics antibacterial therapy anaerobic infections of the maxillofacial region.
test control

Educational goal:

1. Familiarize With features fence researched material for microscopicand bacteriological research methods.
2. Peculiarities composition microflora at non-specific lesions mucousshells cavities mouth (cheilitis, glossitis, stomatitis), the reasons their occurrence.

Student must know:

1. methodology fence researched material at gingivitis.
2. methodology fence researched material at periodontitis.
3. Modern methods treatment diseases periodontal.

Student must be able to:

1. Spend fence researched material from periodontal pocket.
2. Spend fence researched material from periodontal pocket.
3. Carrying out a bacteriological diagnostic method for inflammatorydiseases maxillofacial area.
4. cook smear and paint his on Gram.

Plan lessons:

1. Methods study quantitative and quality composition microflora gingivalgroove and periodontal pockets.
2. Main representatives resident microflora at absence pathology fabricsperiodontal.
3. Peculiarities composition microflora at gingivitis
4. Peculiarities composition microflora at periodontitis
5. Periodontogenic microbes. Proof of them participation in pathogenesis diseases
6. Immunological changes, ongoing in answer on the bacterial antigens andtoxins
7. Modern methods treatment diseases periodontal in compliance With lastscientific data
8. Change module.

Independent Work students:

1. microscopic study demonstration smear-imprint co mucous shells at ulcerative necrotic stomatitis (fusospirochetosis), coloring on Romanovsky.
2. microscopic study demonstration scraping smear co mucouslanguage with leptotrichosis, coloring on Romanovsky.
3. Methods laboratory diagnostics candidiasis.
4. decor protocol research.

INFORMATIONAL MATERIL ON THEME

Before present time not at all clear question - is whether periodontitis logical completion gingivitis. AT experiment on the animals (dogs) managed demonstrate this subsequence, but at of people gingivitis not always passes in periodontitis.

By about mechanism development periodontitis exist, how minimum, two points vision:

1. Exist certain germs, defiant destructive defeat fabrics periodontal.
2. To development periodontitis leads failure in functioning protective mechanisms organism and changes in composition and quantity microflora periodontal pocket.

At darkfield microscopy comes to light significant shift in side rod-shaped forms and spirochete, amount which increases before 40%. Attitude mobile forms to motionless increases before 1:1 (in norm 1:49).

Electron microscopic study subgingival plaques at periodontitis revealed what to cement attached, in basically, gram-positive microbes. Gram negative cells, flagella and spirochetes present in in large numbers in loose layers subgingival plaque that spreads before apical parts pocket.

At bacteriological research material from sick periodontitis established dominance Gram-negative anaerobic sticks, in basically, subspecies non-saccharolytic *Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, Selenomonas sputigena, Eikenella corrodens, Campylobacter rectus* and others However at some patients observed prevalence actinomycetes. Many kinds anaerobic bacteria not succeeded to identify.

AT the present time, on data WHO, to periodontopathogenic types refer, first of all, two representatives of the bacteroid group - *Porphyromonas gingivalis and Prevotella intermedia*. Data gram negative microbes possess ability cling in big quantity to epithelial cells hydroxyapatite and to gram positive bacteria. Them adhesive properties are inhibited in presence human saliva and serum blood. However ability to coaggregation With gram-positive bacteria at this not is inhibited.

At periodontitis characteristic is education microbial clusters, reminiscent corn cob, which consist from cocci and tortuous forms.

At research content gingival pocket at sick periodontitis determined immunoglobulins classes A, g, M, factions complement NW, C5, leukocytes. fabrics gums plentifully infiltrated plasmatic cells lymphocytes and macrophages (monocytes). All this is allows count, what many reactions antigen-antibody, manifestations cellular immunity are happening exactly here, in tissues periodontal and alveolar bones.

If we adhere only to the microbial etiology of periodontitis, then it is obvious that for development diseases must combine the following terms:

1. Presence periodontopathogenic species bacteria in quantity, sufficient for Togo, to has begun pathological process.
2. Terms a habitat in niche must promote growth and reproduction bacteria.
3. Periodontal tissues should be free of antagonist microbes periodontopathogenic bacteria.
4. The microbe must be spatially localized so that it orproducts his vital activity could act on the target cells.
5. organism human must to be sensitive to microbes or productsthem vital activity.

Understanding etiology and pathogenesis periodontitis necessary not only for establishing roles microbes in this process, but also and for clarification conditions, conducive growth plaques, definition roles local and systemic factors which may influence on the resistance or sensitivity fabrics periodontal to bacteria products them vital activity. The study individual features organism host in functioning destructive and protective mechanisms at periodontitis allows optimize comprehensive treatment given diseases.

Microflora at periodontitis - prevails streptococcal Flora above staphylococcal. AT primary stages inflammation this is usually green and non-hemolytic streptococci without group antigen. At transition acute periodontitis in a chronic main role is played by streptococcal anaerobic flora, those. peptostreptococcus, to which are joining other streptococci. AT apical granulomas are found actinomycetes, bacteroids, fusobacteria, vibrios and spirochetes

microbial Flora at periodontitis.

periodontal disease is one from most common diseases cavities mouth and represents yourself inflammatory -dystrophic process in alveolar processes, emerging due to violations nutrition alveoli.

By about mechanism development periodontal disease exists, how minimum, two points vision;

1. There are certain microbes that cause destructivedefeat fabrics periodontium.

2. To development periodontal disease leads failure in functioning protective mechanisms organism and change in composition and quantity microflora periodontal pocket.

All inflammatory processes in periodontium begin with education dental plaques predominantly subgingival, in result colonization surfaces teeth optional anaerobes.

AT the present time, on data WHO, to periodontopathogenic types refer, before Total, two representatives groups bacteroids -Porphyromonas gingivalis and Prevotella melaninogenica. These Gr - microbes have the ability to stick to in large numbers to epithelial cells and to Gr + bacteria. Their adhesive properties are inhibited in presence human saliva and serum blood. At periodontitis characteristic is education microbial clusters, which remind corn cob and consist from cocci and famous forms.

At research content gingival pocket at sick periodontitis determined immunoglobulins classes A, g, M, factions complement cj, C5, leukocytes. If a stick to only microbial etiology periodontitis, then obviously, what for development diseases must combine the following terms:

1. Presence of periodontopathogenic species bacteria in quantity,sufficient for Togo, to has begun pathological process.
2. Terms a habitat in niche must promote growth and reproductionbacteria.
3. Periodontal tissues should be free of microbes - antagonistsperiodontopathogenic bacteria.

4. human body must be sensitive to microbes or products their livelihoods.

1-2. scraping co mucous shells, backrests language can do sterile spatula, trowel. The material is applied to the surface of a glass slide, in a drop water. Gram or Romanovsky stain. Before taking material from erosions and ulcers it is advisable to remove surface plaque with a sterile cotton swab, without using at this antiseptic drugs.

At ulcerative necrotic stomatitis in smear watching abundance gram- negative spindle-shaped rods (fusobacteria) and convoluted forms (anaerobio- spirilla and spirochetes) on the background leukocytes and desiccated epi telium.

With leptotrichosis, a smear shows accumulations of gram-variable filamentous bacterial forms, and part bacteria situated how b in single case (leptotrichia).

3. For laboratory diagnosis of oral candidiasis, the following are used: methods:

a) Microscopic examination of a smear-imprint from the mucous membrane sick candidomycosis can see: oval and sharp elongated cells yeast-like fungus arranged in long chains (pseudomycelium). Not pseudomycelia form Xia flask-shaped swelling, from which lace up chlamydospores (feature of the species C. albicans).

b) When bacteriological method research material, received from sick, sown on Sabouraud media (agar-agar, carbohydrates, peptone) at t = 37 degrees, for 3-5 days. grown up colonies on the this environment studied macroscopically.

Colonies mushroom kind Candida round, whitish, convex, smooth With even edges, surface shiny. Sometimes the colony grows in in agar.

Microscopic examination of smears shows pseudomycelium, consisting of oval elongated, finger-shaped cells located in clusters. Chlamydospores round, resemble bundles of balls.

in) At visceral mycoses are used for serological diagnostics reactions

- agglutination and RSK.For these reactions at sick take serum and determine Availability antibodies in serum sick With candidal diagnosticum.

Scheme reactions agglutination

Components test tubes	one	2	3	four	5	6	7
one. Phys. rr	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2. Researched serum 1:40	1.0 1:80	1.0 1:160	1.0 1:320	1.0 1:640	1.0 1:1280	1.0 1:80	- -
3. Cell antigen (diagnosticum)	1.0	1.0	1.0	1.0	1.0	1.0	-
ACCOUNTING RESULTS							

Accounting results carry out, beginning With control test tubes (6 and 7). O results reactions judged by education draft in experimental test tubes.

4-6. In theory take apart clinical manifestations in cavities mouth at syphilis diphtheria, tuberculosis, herpes, foot and mouth disease using educational allowance. results design in form tables (on graphs: nosological the form; pathogen and his morphology; clinical symptoms in cavities mouth).

7. Decor protocol research on pp. No. 1-3.

Table. Fence researched material at periodontitis

Исследуемый материал	Описание результатов микроскопии	Рисунок

Table. Fence researched material at gingivitis

Исследуемый материал	Описание результатов микроскопии	Рисунок

TEST TASKS

1) Periodontopathogenic species includea)
   Porphyromonas gingivalis
   b) Str. mutans
   c) Prevotella melaninogenica
   G) Staff. Aureus
2) microbial Flora at pulpitis presented:
   a. fusobacteria
   b. staphylococci
   c. streptococci groups D
3) For diagnostics diseases periodontal apply methods:
   a. radiological
   b. definition index Fedorov - Volodkina
   c. definition index Green - Vermillion
   d. definition index SPITN
   e. clinical study blood
4) Basic method surveys dental patient:
   a. radiological
   b. clinical
   c. cytological
   d. laboratory
5) Sterilization this is complex activities directed on the:a) destruction on the objects concrete species microbes b) prevention hits microorganisms in wound c) complete decontamination of objects from all types of microbesd) destruction virulent species microbes.

PRACTICAL OCCUPATION No. 7.

Topic: The study microflora purulent detachable at inflammatory diseases maxillofacial areas. Technique anaerobic cultivation bacteria With quantitative accounting. Ways identification and definitions sensitivity anaerobes to antibiotics.

Educational goal:

1. Technique anaerobic cultivation bacteria With quantitative taking into account
2. Ways identification and definitions sensitivity to antibiotics.
3. Tactics of antibiotic therapy of anaerobic infections of the maxillofacial areas.
4. Peculiarities etiology inflammatory diseases maxillofacial areas (associations, mix-infections).
5. Familiarize With methods fence and microbiological research material for acute and chronic inflammatory diseases of the maxillo- facial areas.
6. To study the main representatives of opportunistic anaerobic infection maxillofacial areas.

Student must know:

1. What kind methods fence researched material use at bacteriologicalresearch microflora with pulpitis and chronic periodontitis?
2. What kind terms are obligatory at transportation material from sickanaerobic an infection?
3. What kind groups bacteria most often stand out at chronic inflammatorydiseases maxillofacial region?
4. What kind groups bacteria most often stand out at acute inflammatorydiseases maxillofacial region?
5. Methods cultivation anaerobic bacteria.
6. Methods definitions sensitivity bacteria to antibacterial drugs.
7. Mechanisms workings resistance to antibacterial drugs at bacteria -residents oral cavity.
8. Features of the composition of the microflora in acute odontogenic inflammatoryprocesses.
9. Peculiarities composition microflora at chronic odontogenic inflammation.
10. Peculiarities composition microflora at nonodontogenic inflammatory processes.

Plan lessons:

1. Methods study quantitative and quality composition microflora gingivalgroove and periodontal pockets.
2. Main representatives resident microflora at absence pathology fabricsperiodontal.
3. Peculiarities composition microflora at phlegmon CHLO.
4. Peculiarities composition microflora at abscesses CHLO.
5. Advantages and limitations the most important methods definitions sensitivitybacteria to antibiotics.
6. Indications and contraindications to application antibiotics at diseases CHLO.
7. Mechanism workings sustainability to antibiotics at bacteria.

Independent Work students:

1. Familiarize yourself with the features of the sampling of the test material from gingival groove and pathological gingival pockets for microscopic and bacteriological research methods.
2. microscopic study demonstration smears from content deep periodontal pockets. Coloring on Gram.
3. microscopic study smears from pure cultures "periodontopathogenic" anaerobic bacteria:
   a) smear from a pure culture of Prevotella melaninogenica, Gram stain; b) smear from clean Actinomyces cultures naeslundii, coloring on Gram.
4. disassemble bacteriological method research purulent exudate Withusing technology anaerobic cultivation (demonstration microanaerostat, transport medium, some environments: thioglycol medium, Wednesday With heminoma, brain heart agar and broth and etc.)
5. disassemble scheme pathogenesis inflammatory process and sketch.
6. Spend bacteriological study material from sick odontogenic infection (stage 1 research).
7. Decor protocol research.

INFORMATIONAL MATERIL ON THEME

1. Peculiarities fence researched material at diseases periodontal.

For microscopic research content periodontal pocketscollected with a curettage spoon or celluloid plate. From the inside sides of the plate - microbes stick on the surface of the root part tooth, a With outdoor — located in gum fluid.

For bacteriological research content periodontal pockets necessary place in transport nutritional Wednesday With purpose conservation viability of anaerobic bacteria. In case of examination of sub-gingival dental plaque removed from the periodontal pocket, it must be disintegrated before sowing With help ultrasound. Further selection pure cultures, them cultivation and identification carry out in anaerobic conditions on classical scheme.

2. At microscopic research content periodontal pocket in demonstration preparations reveal morphological forms characteristic for major paprodontopathogenic species - actinomycetes, streptococci, bacteroids, fusobacteria and spirochetes.

3. Detailed study of the morphology of periodontopathogenic bacterial species in smears from pure cultures. Coloring on Gram.

4. Decor protocol research

Table. Fence researched material at abscesses of the maxillofacial area

researched material	Description results microscopy	Picture

Pathological material at abscesses phlegmon and fasciitis take puncture With help thick needles and deliver in laboratory in syringe. AT process operational intervention, the material is taken using a standard cotton swab, which placed in a transport medium. Transport media, due to the peculiarities of its composition, provide sharp decline metabolism microbes and possibility long conservation them vitality (from 6 - one 2 hours).

I **stage bacteriological research** - receiving isolated colonies. Usually performed on the cups petri With 5 % anaerobic hemagar - nutritional environment, which besides native blood contains such factors growth anaerobic bacteria like hemin (vitamin K) and menadione. The medium is universal for growth majority species anaerobic and aerobic bacteria.

Petri dishes with an anaerobic hemagar are cultivated in an anaerostat or gas box at t - 37 ° C up to 7 - 10 days, although most of the anaerobes gives good colony growth already on the 3 - four day. At macroscopic and microscopic studying grown colonies compare the morphology of the bacteria themselves and the colonies they formed during cultivation. in an anaerobic balloon and received by 5 % blood agar in aerobic conditions.

Testing is essential for further identification. on the Availability catalase: material colonies mix on the subject glass With drop 0.5 % peroxides hydrogen - active education bubbles gas testifies about availability this microbe has an enzyme catalase, what usually characteristically for optional - anaerobic bacteria. Main kinds obligately - anaerobic bacteria catalase not produce.

II **stage bacteriological research** - receiving clean culture . Performed on the liquid (thioglycolic Wednesday, Wednesday kitta - Taroczi, cordially- cerebral bouillon) or semi-liquid environments (With adding 6.5 % agar agar). Material from isolated colonies endure in test tube With one of specified environments that then desirable place in anaerostat. Pure culture receive through 3 - 5 days cultivation at t- 37° FROM.

III **stage bacteriological research** - identification clean culture. For definitions kind dedicated culture used definition complex morphological, tinctorial, cultural, biochemical and chemotaxonomic properties.

results bacteriological research bring in in protocol.

acute odontogenic inflammatory diseases	abscess phlegmon osteomyelitis lymphadenitis	bacteroids, fusobacteria, peptostreptococcus, peptococci, less often actinomycetes, staphylococci
chronic odontogenic inflammatory diseases, actinomycosis		obligate associations anaerobic bacteria with facultative anaerobes (staphylococci, bacilli, streptococci) aerobic and anaerobic actinomycetes (rotia, nocardia and streptomyces).
nonodontogenic inflammatory diseases	furuncle festering atheroma infected injury people-persons.of the region	the predominance of staphylococci, streptococci, less often bacilli and obligate anaerobes, often stand out monocultures

TEST TASKS

1. Operating wounds called conditionally clean, if in process surgicalintervention contact occurs tools With:
a - mucous shell cavities mouth
b - mucous membrane of the paranasal sinusesin -
skin
G - purulent exudate inflammatory hearth
2. The causative agents of juvenile periodontitis are:a -
leptotrichia
b - streptococci

in - actinobacilli G -
bifidobacteria

3. From the purulent exudate of periodontal abscesses, the following are most often distinguished:a - Prevotella P.intermedia
b - porphyromonas P. gingivalisc
– peptostreptococci P.micros G -
staphylococci S. aureus
4. actinomycetes are group bacteria, With activation which binddevelopment next diseases:
a - phlegmon and abscesses maxillofacial areas
b - chronic inflammatory diseases soft and bone fabricsin - recurrent
aphthous stomatitis
G - periodontitis
5. Antibiotics groups macrolides apply for treatment:
a - candidiasis cavities mouth
b - leptotrichiasis of the
mucosain - periodontitis
G - returnable aphthous stomatitis

PRACTICAL OCCUPATION No. 8.

Topic: Chronic foci infections. pathogens tuberculosis and leprosy. Features of diagnosis and manifestation of infection in the oral cavity. Prevention and treatment tuberculosis and leprosy

Educational goal:

1. Bacterial infections and them manifestation in cavities mouth (tuberculosis).

Plan lessons:

1. Peculiarities composition microflora cavities mouth at tuberculosis.
2. Manifestations tuberculosis in cavities mouth.
3. Peculiarities fence researched material at tuberculosis.
4. Methods laboratory diagnostics tuberculosis.
5. Modern methods treatment tuberculosis.

Student must know:

1. methodology fence researched material for microscopic researchat non-specific lesions mucous membranes of the oral cavity.
2. methodology fence researched material for bacteriological researchat non-specific diseases mucous shell cavity mouth.
3. Modern methods treatment viral infections and them manifestation in cavities mouth.

Student must be able to:

1. Spend fence researched material for microscopic and bacteriological examination for nonspecific lesions of the mucosa membranes of the oral cavity.
2. Carrying out a bacteriological diagnostic method for viral infections and them manifestations in oral cavity.

Independent Work students:

1. Theoretical analysis of clinical manifestations in the oral cavity of diphtheria andtuberculosis, diagnostic methods.

2. Decor protocol research.

INFORMATIONAL MATERIL ON THEME

Tuberculosis of the mucous membrane of the mouth and lips is caused by mycobacteria tuberculosis, in mostly human type, and usually is secondary, less often primary tuberculosis of the oral mucosa develops in the form of primary tuberculosis complex. Mycobacteria tuberculosis may hit in mucosa shell mouth how endogenous through So and exogenous.

Mucous shell cavities mouth is bad environment for breeding tuberculosis mycobacteria; having hit in mucosa shell at majority sick tuberculosis, they die. If, nevertheless, its defeat occurs, then the clinical form diseases depends from row factors before Total from general currents tuberculosis process and immunological states organism, which determine With help tuberculin reactions. AT pathogenesis tuberculosis certain role play character food, neuro-endocrine disorders and others

From the forms of secondary tuberculosis with damage to the mucous membrane mouth can observed lupus erythematosus, scrofuloderma and miliary ulcerative tuberculosis, moreover, if two first forms flow usually on the background positive tuberculin reactions, then malarial and ulcerative tuberculosis arises predominantly on the background anergy, t. e. on the background negative tuberculin reactions.

Primary tuberculosis of the lips and oral mucosa. primary tuberculosis , or primary tuberculous complex, or primary tuberculous chancre, on the lips and oral mucosa is rare, mainly in children. He arises in result exogenous infection, which going on more often air- drip, less alimentary way. This form of tuberculosis can only develop in people who do not have mycobacterium tuberculosis in the body and tuberculin reactionsnegative.

After an incubation period of 8-30 days , at the site of the entrance gate of infection painful ulceration up to one- 1.5 cm with undermined uneven edges with a dirty gray bottom. The bottom and edges of the ulcer are slightly compacted, however, on the lips, the seal can be significant. After 2-4 weeks after education ulcers increase and submandibular lymphatic nodes. At first they are mobile, and then soldered to each other and to the skin. Often through some time these nodes suppurate and are opened.

Tuberculous lupus. Among tuberculosis diseases of the mucous membrane mouth and lips tuberculosis lupus is most frequent stubborn inclined to relapses, chronically current disease. Beloved localization lupus erythematosus is a person that is affected in approximately 75% of patients, and very often red is involved in the process border top lips on which process usually passes With nose. However maybe to be and isolated defeat red borders top lips.

Primary element at tuberculosis lupus is tubercle (lupoma). lupoma represents yourself limited, in early flat, with pin head or a little more red or yellowish red soft painless education, prone to peripheral growth and merger With neighboring elements. AT result mergers lupom formed foci defeat, having various dimensions and outlines.

Lupus foci on the red border of the lips and especially on the oral mucosa ulcerate. The edges emerging at this ulcers corroded, wrong forms. Bottom ulcers covered or dirty gray raid, or papillomatous expanding granulations, sometimes they resemble bright juicy raspberries. On the red border lips on the surfaces ulcers often formed crusts, sometimes very thick.

On the place defeat remains superficial cicatricial atrophy; characteristically re-occurrence of individual loops on such a scar. In places of ulceration, rough, disfiguring scars form. Lupus ulcerative process, although rare, leads to significant destruction fabrics.

In second stages on the background edema and hyperemia appear individual small tubercles, which present yourself papillary overgrowth, covered slightly tarnished epithelium. Merging with each other, they can resemble warty overgrowth. AT subsequent at majority sick tubercles break apart With the formation of an ulcer, which can be of different sizes, irregular outlines, often with corroded, but unburied edges, With granulations on the bottom and narrow

an inflammatory border around, against the background of which one can often see individual surviving tubercles, a also erosion. AT detachable from ulcers, how rule microscopically not succeed discover tuberculosis mycobacteria. At completion process scars are formed, and if the process proceeded without ulceration nia, then they smooth, shiny, atrophic. After ulceration scarring dense, rough, solder mucosa shell With subject tissues.

Clinical painting tuberculosis lupus It has some peculiarities, process localization. According to the location of the lesion on the mucosa shell gums distinguish four kind defeats: one) marginal encompassing gingival edge first in form banal infiltration and passing then in tuberculous-erosive (ulcerative) form; while the gingival margin and interdental papillae sharp swell up picture gingival the edges smoothed out mucous shell gums takes on a bright red color. The gum appears as if pierced pins, painless matte dim, easily bleeds; 2) supramarginal: infiltrative or tubercular-ulcerative lesion does not affect the gingival border; 3) total: process captures all outer surface gums on type infiltrative, often erosive, and sometimes ulcerative lupus. With this form often is affected bone the cloth alveoli, maybe develop "painting hypertrophic stupid gingivitis"; four) bilateral, flowing on type of ulcer lupus.

Treatment tuberculosis mucous shells cavities mouth, being one from manifestations general tuberculosis, carry out on generally accepted methodologies treatment tuberculosis usually in tuberculosis dispensaries. Most effective means treatment tuberculosis lupus are drugs hydrazideisonicotinic acids (ftivazid, isoniazid, or tubazid, salusite, metazid, larusan, INGA-17, etc.), which affect mycobacterium tuberculosis not only bacteriostatic, but and bactericidal action.

Prognosis in patients with tuberculosis of the oral mucosa at the present time,at availability powerful tuberculosis funds, good, but sick must for a long time, until a complete cure, be under dispensary observation. AT early identifying tuberculosis patients mucous shells cavities mouth, in direction them on the treatment in anti-tuberculosis institutions, in organizations dispensary observationsDentists play a big role. This is due to the fact that lupus erythematosus andmiliary-ulcerative tuberculosis can begin and exist for a long time only on mucous shell cavities mouth, in connections With how sick, naturally, apply todentist.

TEST TASKS

1. For the treatment of odontogenic sinusitis, drugs are used:a -
lincomycin 0.5 g 2 times in day in / m
b - erythromycin 500 mg 4 times + metronidazole 250 mg 3 times a day orallyin -
gentamicin 1 g 2 times in day in / m
G - rifampicin 500 mg 2 times in day oral

2. For stepped antibacterial therapy at heavy progressive phlegmon admissible apply:
a - dalacin C IM, then lincomycin orally b -
rovamycin IM, then spiramycin orally in -
clarithromycin in / in, then rulid orally
G - kanamycin in / m, then cephalexin orally

3. Components development odontogenic inflammation are:
a - microbial release beyond their ecological niche in the oral cavityb -
toxinemia
in - violation local circulation
G - invasion microbes in fabrics from environmental environments

4. When microbiological diagnosis of periodontitis is performed:a -
material sampling in transport system
b - quantitative assessment of different types of colonies when seeded on yolk-salt agarin-
quantitative evaluation different types colonies at sowing on the blood hemin agar
G - definition sensitivity to antibiotics dedicated culturesd - PCR diagnostics

5. etiological factors odontogenic infections are:
a - crowding out normal anaerobic flora virulent aerobes, for example,Pseudomonas

aeruginosa wand
b - decline redox potential fabrics and activation anaerobes
c - entry of spores of anaerobic clostridia into the wound from the environmentG - violation microcirculation in tissues at trauma.

PRACTICAL OCCUPATION No. 9.

Topic: Microbiological diagnostics dysbiosis cavities mouth and stomatitis. Dysbiosis and opportunistic stomatitis. Opportunistic processes how manifestations immunodeficiencies and HIV infections. laboratory diagnosticscandidiasis, leptotrichiasis, fusospirochetosis.

Educational goal:

1. Familiarize yourself with the features of sampling the material under study formicroscopic and bacteriological research methods.
2. Peculiarities composition microflora at non-specific lesions mucousshells cavities mouth (cheilitis, glossitis, stomatitis), the reasons them occurrence.
3. Bacterial infections and them manifestation in cavities mouth (gonococcal gingivo-stomatitis).
4. To master the technique of sampling the test material for microscopicresearch at non-specific lesions mucous shells cavities mouth.
5. master methodology fence researched material for bacteriologicalresearch at non-specific diseases mucous shells cavities mouth

Student must know:

1. methodology fence researched material for microscopic researchat non-specific lesions mucous membranes of the oral cavity.
2. methodology fence researched material for bacteriological researchat non-specific diseases mucous shell cavity mouth.
3. Modern methods treatment viral infections and them manifestation in cavities mouth.

Student must be able to:

1. Spend fence researched material for microscopic and bacteriological examination for nonspecific lesions of the mucosa membranes of the oral cavity.
2. Carrying out a bacteriological diagnostic method for viral infections and them manifestations in oral cavity.

Plan lessons:

1. Features of the sampling of the test material for microscopic andbacteriological research.
2. Peculiarities composition microflora at non-specific lesions mucous cavitiesmouth (cheilitis, glossitis, stomatitis), the reasons them occurrence.
3. HIV infection. Manifestations in cavities mouth.
4. Candidiasis. laboratory diagnostics candidiasis.
5. Leptotrichiasis. laboratory diagnostics.
6. Fusosperochitosis. laboratory diagnostics.

Independent Work students:

1. Theoretical analysis clinical manifestations in cavities cheilitis, glossites,stomatitis, HIV infection, diagnostic methods.
2. Decor protocol research.

INFORMATIONAL MATERIL ON THEME

Viruses are most frequent cause infectious diseases(measles, epidemic mumps, rubella, flu, windmill smallpox, hepatitis, AIDS and etc.).

Another virus for which parenteral transmission is also maybe to be basic, - this is virus immunodeficiency human (HIV). Virus HIVstrikes

lymphocytes (T-helpers) and macrophages. At long reproductions virus inbody populations helpers is shrinking in affected macrophages declining production interleukin-I. Except immune systems suffer other bodies and systems: nervous system, bodies digestion and breath, cardiovascular system , etc. Against the background of immunodeficiency , Kaposi 's sarcoma develops and opportunistic diseases (candidiasis, ulcerative necrotic stomatitis, cytomegalovirus infection, herpes, pneumocystosis).

Sources virus are sick and virus carriers. Available parenteral route of infection when using an infected virus blood with various surgical interventions, blood transfusion, etc., as well as sexual contact. Registered cases infections fetus in intrauterine period.

Diagnostics held main way serological method, through identifying antibodies With help enzyme immunoassay analysis.

Thus, patients who came to the reception with candidomycosis, relapses herpes, ulcerative necrotic defeat must to be necessarily verified on the infection HIV. special danger is posed by patients at risk - homosexuals, prostitutes, drug addicts, patients who often received blood transfusions (hemophilia).

A dentist can become infected with a virus from a patient airborne by and during a digital examination, as well as the doctor himself during his manipulations maybe infect patient (hepatitis AT, AIDS).

Additionally necessary Mark, what about near 46 % patients on the reception can be carriers in the oral cavity of Streptococcus puogeus, Staphul. aureus. They are can cause the doctor to develop various respiratory diseases, as well as cause an inflammatory process in case of a hand injury with a drill or a stab wound. Considering that, that many of the staphylococci are antibiotic-resistant, the entry of these microbes maybe make it difficult process treatment.

Leprosy

Leprosy (leprosy) called leprosy mycobacteria Hansen and represents yourself chronic generalized infection developing predominantly in derivatives ectoderm and in bodies and fabrics, rich elements active mesenchyme. Leprosy leaks chronically progressive With periodical exacerbations (leprosy reactions), leading in the absence of rational treatment for severe disability and through a lot of years to of death.

Allocate tuberculoid and lepromatous types leprosy. emergence Togo or another type of disease related with fortune resistance organism human to leprosy infection, which determine With help lepromine skin samples. Mucosal lesions shells cavities mouth may arise only at lepromatous type, which the develops at persons With sharp reduced reactivity (lepromine try negative). At such sick on the skin, mucous shell mouth, internal bodies, on move nervous trunks arise lepromatous infiltrates With leprosy cells not able (in difference from tuberculoid type) destroy phagocytosed them sticks Hansen, that's why they freely multiply in these cells.

Leprous process on mucous shell mouth begins with infiltrative stage, then on the this background arise tubercular rashes which through some time ulcerate. Process ends scarring. Highly often on the mucous shell mouth at one and Togo same sick can observe elements, characteristic for all four stages development leprosy defeat mucous shells, t. e. infiltration tubercles, ulcers and scarring. Clinical painting leprosy lesions of the oral mucosa is characterized by polymorphism, each of four stages characterized described signs.

Leprosy changes mucous shells mouth begin With education superficial limited infiltrate, slightly rising above the surrounding mucous shell and having grayish white color, sometimes With dark blue plots. Then on the infiltrated plots arise tubercles various sizes - from millet grain to a cherry stone. At first, the tubercles are dense, then soften. They are located haphazardly and inclined to peripheral growth and merger. Tubercles mostly dull pink, sometimes grayish pink colors. Them surface usually shiny. More often Total tubercles arise on the solid and soft sky, language and lips. Through some time tubercles usually ulcerate. ulcers on the place tubercles at first small, bottom them bumpy, dirty gray white colors, the edges uneven, swollen, soft consistency. Sometimes ulcerative process distributed by on the bone the cloth, causing her meltdown. More often Total at this collapses alveolar edge jaws. AT subsequent ulcers scarred but scarring may develop without previous ulceration tubercles, when the tubercle or infiltrate mucous shells mouth fibrosis. Education scars more not

means recovery, for simultaneously may arise new infiltrates, tubercles and ulcers. Leprosy scarring on the mucous shell cavities mouth may to be round or in form stripes — radiant. Scarring smooth, shiny, white colors. formed scarring in dependencies from them sizes and places location may cause functional disorders. Especially often the soft palate is deformed and tongue, which the maybe shift a sometimes even disappear.

Leprosy defeat may arise and on the lips. At this very often a pronounced infiltrate is formed, accompanied by edema, which entails education leprosy elephantiasis. Lip compacted, becomes more thick, roller-shaped and sedentary. Leather lips and red border on bloom few different friend from friend. On the this background arise Good contoured tubercles.

Leprosy changes on the inner surface lips begin With diffuse erythema, then an infiltration occurs view bluish spots, covered with thickened epithelium. Against this background, sometimes bumps appear, which nearly not act above mucous shell. Leprosy tubercles on the lips may long time remain unchanged but sometimes they ulcerate. The emerging shallow painless ulcers located on the surfaces tubercles. Them detachable dries up in light yellow crusts.

Ulcerative defeat mucous shells lips always ends scarring with deformity of the lips, resulting in a thinner lip, the mouth opening may narrow down. If a same deep located tubercles fibrosis, then lip wrinkled, due to what finds it difficult speech, leather lips in these cases happens dotted furrows a red border lips becomes wrinkled. Leprosy changes mucous shells lips rarely reach before transitional folds.

Beloved localization leprosy rashes are gums on the topjaws from the side of the tongue in the area of the frontal, less often molars. Leprosy changes in the gums begin with the formation of an infiltrate. The gums seem to swell are made loose, red, sometimes cyanotic slightly bleed; gingival papillae swell up picture gingival the edges smoothed out. Highly often to this increased salivation joins. Soon the gingival mucosa becomes matte, on the her surfaces formed sores, which then scarred what leads to wrinkling of the gingival margin and turning it inward. The gums retract, the roots of the teeth exposed. Simultaneously with scarring in other areas of the gums, fresh infiltrate. characteristic painless process.

Leprosy changes on the mucous shell solid sky are happening next way: in those cases, when defeat starts on the front thirds at first noted infiltrative stage, infiltrate formed behind necks central incisors and comes before fangs. Plot infiltrated mucous shells on direction back narrows and more often ends at the beginning middle thirds solid sky, in result what formed triangle, basis facing forward, and the top back. Significantly less often infiltrate in the direction narrows back to a strip 2 cm wide and extends along the midline to soft sky. Infiltrate on the lateral surfaces solid sky observed extremely rarely. infiltrated plot greater part It has grayish red color and absolutely painless. Then on the infiltrated plots arise grayish white tubercles magnitude With millet corn, which subsequently superficially ulcerate.

In the soft palate, the lesion begins with hyperemia, turning into an infiltrate dark coloring. Sometimes soft sky at first It has pale yellow color. tongue usually infiltrated dimensions his increase. On the infiltrated areas of the soft palate and uvula appear whitish-gray, focally located tubercles various quantities — from millet grain before peas. Then tubercles break apart and formed sores. Sometimes individual sores merge, forming greater or lesser quantities solid ulcerative surfaces. The edges ulcers slightly raised and undermined, bottom bumpy With grayish raid. Color them dirty grayish white. Due to ulcerative defeat usually collapses part or the whole tongue.The ulcerative process on the hard and soft palate ends with scarring. The emerging scarring have varied form: sometimes they round, but more often radiant or stellate. Scarring usually shiny, superficial, whitish colors. Cicatricial contraction on the solid sky near necks front teeth leads to wrinkling gums and retractions her With exposure roots teeth. The edges retracted gums wrap up inside in side roots, tight hugging them. Scarring on the soft palate sometimes form an arc, the convex part facing forward. fibrosis in areas soft sky often causes persistent deformation, due to what soft sky maybe pull up up, narrowing input in nasopharynx. Sometimes soft sky absolutely is destroyed.

Beloved localization leprosy elements on the language is average line his backs, beginning from root and before tip. Language maybe infiltrate increase, thicken, in result what

his mobility finds it difficult and speech becomes obscure. On the infiltrated surfaces backrests language appear dense tubercles various quantities, but not more bob, With flat surface and wide basis. Surface tubercles shiny, covered whitish raid thanks to desquamation epithelium ("silver" language). Number tubercles may enlarge and may coalesce (leprosy glossitis), resulting in what on the back of the tongue are formed roller-shaped elevations with deep furrows between them.

tubercles on the language inclined to decay and ulceration. formed ulcers located superficially and have jagged, undermined, infiltrated edges. The bottom of the ulcers shallow, rough, covered with a grayish coating. In some cases, these ulcers merge, forming a continuous ulcerative surface, covered with a thin gray coating. Emerging on the place ulcers various forms superficial scarring usually have greyishwhite color and brilliant surface.

For the diagnosis of leprosy , bacterioscopic studies of scrapings from mucous shells nasal partitions, a also co bottom and edges leprosy ulcers, in whicheasily discover sticks Hansen.

Leprosy lesions of the oral mucosa may resemble tertiary syphilis and lupus erythematosus. From the manifestation of lupus erythematosus and collicative tuberculosis, leprosy rashes are characterized by greater density, the presence of pronounced disorders sensitivity and leprosy mycobacteria in ulcerated dischargeelements.

Most effective antileprosy means are drugs sulfonic row, such, how diamino diphenyl sulfone (DS), avosulfone, dapsone, sulfetron (solusulfone, novotrope), which accept in flow long time. The less toxic drug thiocarbonylide has the same effect as sulfones. (derivative thiourea). Not lost meaning chaulmugrious oil and his derivatives, inin particular ethyl ethers and mugrol. In order to successfully treat patients with leprosy, always should apply combined therapy everyone famous antileprosymeans in combined With restorative drugs and physiotherapy.

Forecast at leprosy in recent decades in connections With introduction in practice treatment this disease of new, sufficiently effective drugs has become better. Main public prevention of leprosy is early detection and rapid isolation of patients with leprosy in special institutions - leper colonies, where patients are treated, live and work. At discovery sick or suspicious on the disease leprosy doctor immediately must to report about this authorities healthcare. At confirmation diagnosis leprosy sick With compliance measures precautions envisaged instruction People's Commissariat ways messages — People's Commissariat health care from 19/IV 1944 G., must to be delivered in leprosarium.

depending from localization inflammatory process mucous shells cavities mouth called various: stomatitis (cheek mucosa), glossitis (tongue), gingivitis (gums), cheilitis (lips). Stomatitis is usually either a consequence various dystrophic processes in body, infectious or somatic diseases, or result damaging physical or chemical impact on the mucosa at secondary roles resident microflora. At superficial catarrhal stomatitis usually find Gr + aerobic cocci and sticks, at deep stomatitis prevails strictly anaerobic Gr - Flora (fusobacteria, bacteroids, peptostreptococci).

At ulcerative necrotic stomatitis prevails anaerobic Flora, predominantly fusobacteria and spirochetes, but may attend and other microorganisms (veillonels, peptostreptococcus, bacteroids, vibrios, actinomycetes). To fusospirochetosis also refer ulceratively - necrotic sore throat Vincent, sore throat Ludwig, gangrene lung ulcerative colitis and etc.).

In recent years, there has been an increase in the incidence of candidomycosis. It's connected with widespread use of antibiotics, corticosteroids, cytostatics. Long them application leads to violation composition normal microbial flora (dysbacteriosis). Candida fungi are a resident of the mucous membranes of the oral cavity, digestive tract, respiratory ways, vagina, skin covers.

Process relationships yeast cells With epithelial cells oral mucosa begins with their adhesion. Sucrose, maltose, glucose and others carbohydrates increase activity adhesion. Adhesiveness yeast-like mushrooms of the genus Candida in many defines them virulence.

System complement, which activated mannan cellular walls yeast, inhibits their adhesion. Yeast-like mushrooms contribute to the destruction tooth enamel and the development of caries. Carious teeth in which yeast vegetates cells, can consider, how peculiar ecological niche-, thanks to which they may participate in development mycotic tonsillitis and stomatitis. Local manifestations candidiasis or primary candidiasis in cavities mouth leaks in form acute pseudomembranous candidiasis (thrush), acute or chronic candidiasis and hyperplastic

candidiasis.

Gonococcal stomatitis

Gonococcal lesions of the oral mucosa - gonococcal stomatitis - meets rarely. The disease occurs in newborns when gonococci enter the cavity mouth child in time childbirth at passing through infected generic way mother. Infection from caregivers and other patients is possible. Usually observed simultaneous defeat gonococcus mucous shells mouth, nose and conjunctiva. Gonococcal stomatitis has also been observed in adults (Besionck, Janet and etc.). Gonococcal stomatitis is more common than commonly thought, but it remains unrecognized because what, firstly, inspection cavities mouth and nose most sick gonorrhea not produce, Secondly, dentists and otolaryngologists, to which more often of all patients who are unfamiliar with this disease are treated, and, thirdly, gonorrheal stomatitis more often leaks without subjective sensations, inclined to self-healing and sick disappear from under observations doctors.

In recent years, there has been an increase in reports of extragenital gonorrhea in adults. In this case, the pharynx and tonsils are more often affected, less common stomatitis, gingivitis, laryngitis. The lesion of the oral mucosa is the main way at homosexual men and persons, who had orogenital contacts.

Flow gonorrhea mucous shells cavities mouth and pharynx, how rule asymptomatic. At children, the act of sucking is not violated. Pain is rare in adults in the throat, the body temperature rises. The first symptoms of gonococcal stomatitis - hyperemia, edema, small erosion on the mucous shell and viscous mucopurulent more or less abundant secret. AT more heavy cases at absence treatment process maybe to spread appears big amount erosion and ulcers on the mucous shell cheeks, language, gums sores superficial, small sizes, With wrong unburied or few undermined edges, soft, painless, With sparse yellow gray separating immovable, in which discover gonococcus, what confirms diagnosis.

Histologically determined inflammatory process in subepithelial connective tissue with infiltration by lymphocytes, neutrophils, plasma cells.

Treatment of gonorrheal stomatitis is carried out with antibiotics in the same doses as gonorrheal lesions of the genitourinary organs. Locally prescribed rinses 0.01-0.1% solution permanganate potassium.

Prevention of gonorrheal stomatitis in newborns born from mothers sick gonorrhea, consists in processing mucous shells mouth newborns immediately after birth 2% solution nitrate silver. Adult patients with gonorrhea genitourinary organs, the mucous membranes of the oral cavity and pharynx should be examined, with testimony carry out study detachable on the gonococcus

Laboratory diagnosis carried out through use next methods:

1. Microscopic examination (light, luminescent) pathological material (raid, pieces bodies and etc.) and discovery young or mature pseudomycelium.
2. Bacteriological study - sowing material on the Wednesday Saburo, tomato bouillon and rice agar With identification dedicated culture.
3. Serological method - staging reactions agglutination and binding complement With serum blood sick With purpose detection antibodies.

TEST TASKS

1. At the second stage of the disease with syphilis, diagnostic methods are used:a) bacterioscopic
b) bacteriologicalin)
serological
G) biological

2.Install conformity morphology and coloring With group anaerobic bacteria:

a - spore-forming Gram+ sticks	1. Clostridia.
b - non-spore-forming Gram+ sticks	2. Peptostreptococci.
in - non-spore-forming Gram+ cocci	3. Eubacteria.
G - non-spore-forming Gram- sticks	4. Bacteroids.

3. Practical application reactions precipitation:

a. serodiagnosis abdominal typhus - reaction Vidal,
b. serodiagnosis of syphilis - sedimentary reactions of Kahn and Sachs-Vitebsky,
in. serodiagnosis brucellosis - reaction Wright, Heddelson,
seroidentification of streptococci according to
Lensfield.d. indication AG Siberian ulcers by Askoli.
4. Roxithromycin (rulid) for perioperative prophylaxis is prescribed at a dose of:a - 100 mg IV for 60 min
b - 150 mg, intramuscularly per thirty min
c - 150 mg, orally for 30-60 minutesG
- 300 mg, orally per 2 hours

5. At disinfection products medical destination boiling in distilledwater With 2% bicarbonate sodium (soda) time exposure is:
a - not less 5 minutes
b - at least 10 minutes
in- at least 15 minutes
G - not less thirty
minutes

PRACTICAL OCCUPATION No. 10.

Topic: infectious stomatitis and diagnostics manifestations bacterial and viral infectious diseases in cavities mouth. laboratory diagnostics diphtheria, gonorrhea, syphilis. Prevention and treatment.
laboratory diagnostics herpetic, coxsackie- and echo viral stomatitis. Principles prevention
.

Educational goal:

1. Familiarize yourself with the features of sampling the material under study formicroscopic and bacteriological methods research.
2. Peculiarities composition microflora at diphtheria, gonorrhea, syphilis herpesthe reasons them occurrence.
3. To master the technique of sampling the test material for microscopicresearch at diphtheria, gonorrhea, syphilis, herpes.
4. master methodology fence researched material for bacteriologicalresearch at diphtheria, gonorrhea, syphilis, herpes.

Plan lessons:

1. infectious stomatitis. Manifestations in cavities mouth. laboratory diagnostics.
2. Diphtheria. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.
3. Gonorrhea. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.
4. Syphilis. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.
5. Herpes. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.

Student must zant:

1. methodology fence researched material for microscopic researchat non-specific lesions mucous membranes of the oral cavity.
2. methodology fence researched material for bacteriological researchat non-specific diseases mucous shell cavity mouth.
3. Modern methods treatment viral infections and them manifestation in cavities mouth.

Student must be able to:

1. Spend fence researched material for microscopic and bacteriological examination for nonspecific lesions of the mucosa membranes of the oral cavity.

2. Carrying out a bacteriological diagnostic method for viral infections and them manifestations in oral cavity.

Independent Work students:

1. Theoretical analysis of clinical manifestations in the cavity of diphtheria, gonorrhea,syphilis, herpes, diagnostic methods.
2. Decor protocol research.

INFORMATIONAL MATERIL ON THEME

Syphilis is a chronic infectious disease caused by Treponema pallidum. Syphilis characterized very peculiar flow: firstly, undulating change active manifestations and periods is hidden flowing infections; Secondly, gradual anda consistent change in the clinical and pathoanatomical picture of lesions bodies and fabrics from mild inflammatory phenomena before education specific deep infectious granuloma, squeezing and destructive bodies and tissues in which they are localized, which leads to loss of organ function, and sometimes to of death sick.

There are acquired and congenital syphilis. Congenital syphilis occurs when hit pale treponema in organism fetus through placenta from sick syphilis mother. For infections human syphilis necessary penetration pale treponemathrough skin or mucosa shell, integrity which violated.

Usually infection occurs sexually. Sexual infection can be professional, for example, in medical workers during operations, autopsy, dental or gynecological examination, etc., or occur when usinggeneral crockery, labial lipstick mouthpieces and others

Due to the undulating course of syphilis, the different nature of clinical and morphological changes that occur at various stages of the disease are distinguished incubation, primary, secondary and tertiary periods acquired syphilis, a also hidden, in volume including unknown, visceral syphilis and syphilis nervous systems.

Incubation period syphilis in average equals 3-4 weeks, but Maybe how hisshortening (before 10-12 days), So and elongation (before 6 months), which usually related With reception in time incubation small quantity antibiotics on about intercurrent diseases or gonorrhea.

The primary period of syphilis begins with the onset at the site of infection, i.e. the introduction of pale treponema, hard chancre (primary syphiloma). Primary Period lasts 6-7 weeks. 5-7 days after the formation of a hard chancre, a second an indispensable symptom of the primary period - regional lymph nodes (bubo, or regional scleradenitis) increase. These nodes thrive treponema. From the lymph nodes along the lymphatic pathways already at the beginning of the primary period treponema fall in blood, in answer on the this is gradually start be developed antibodies, which at the end of the 3rd week of the primary period of syphilis can be determined in blood using classical serological reactions (Wassermann reaction, sedimentary reactions), several before — with the reaction immunofluorescence (REEF), a a little later - and with the help of the immobilization reaction of pale treponema (RIBT, or RIT).

About at twenty% sick to end of primary period syphilis develop general symptoms (increase temperature body before 38-38.5°C, weakness, head pain, pain in bones especially on nights), in peripheral blood observed small anemia, leukocytosis, increase in ESR. Later 4-6 days on the this background on thethe skin of the body, and often on the mucous membrane of the oral cavity, a rash appears, which testifies about graduation primary and early secondary period syphilis.

Mucous shell cavities mouth and red border lips are localization syphilitic rashes in all stages diseases, in volume including and at primary syphilis. At non-sexual infections localization chancre on the lips and mucous shell cavities mouth meets most often. Solid chancre maybe arise on the any site red borders lips or mucous shells cavities mouth, but more often Total he localized on the lips language, tonsils.

The development of a hard chancre on the lip or oral mucosa, as well as on others places starts With appearance limited redness, in basis which within 2-3 days there is a seal due to the inflammatory infiltrate. it limited seal gradually increases and reaches usually 1-2 cm in diameter. In the central part of the lesion, necrosis occurs and erosion of the meat- red, rarely ulcer. Having reached full development within 1-2 weeks, solid chancre on the mucous membrane is usually a round or oval painless lesionny erosion of meat-red color or an ulcer with saucer-shaped edges ranging in size from 3 mm (pygmy chancre) up to 1.5 cm in diameter with dense elastic infiltrate in basis. In the scraping of the surface of the chancre, pale treponemas are easily detected. Sometimes erosions are covered with a grayish-white coating. When the chancre is located on the lips sometimes formed significant edema, due to whom lip sags, a chancre lasts longer than elsewhere. More often one hard chancre develops, less often - two or more. If a joins secondary infection, then erosion can deepen, at this formed ulcer With dirty gray necrotic raid.

On the language solid chancre usually happens single, arises more often in middle third. In addition to erosive and ulcerative forms, streets with folded language, with localization solid chancre along folds maybe arise slit-like the form. At location solid chancre on the backrest language due to significant infiltrate in basis chancre usually sharp speaks above environmental cloth, on the its surface has meat-red erosion. Note the absence inflammatory phenomena around chancre and his painlessness.

Solid chancre in areas gums It has view bright red smooth erosion,which in the form of a crescent surrounds one or two teeth. Ulcerative form hard chancre gums is very similar to banal ulceration and almost does not have any signs, characteristic for primary syphilomas. Diagnostics facilitates Availability bubo in submandibular areas.

At localization on the tonsil solid chancre maybe have one from three forms: ulcerative, angina-like (amygdalite) and combined — ulcerative on the angina-like background. The tonsil is affected on one side. With ulcerative amygdala increased dense Against this background, there is meat-red oval ulcer With canopies even edges. Mucous shell around ulcers hyperemic. Process accompanied painful sensations sometimes significant. At angina-like chancre erosion or ulcer missing, available unilateral significant enlargement of the tonsil. It acquires a copper-red color, painless nenaya, dense. Process is different from sore throats one-sidedness defeat, absence pain and acute inflammatory hyperemia. General phenomena No, temperature body normal.

Chancre on the lips should be differentiated from simple vesicular lichen, with which in difference from syphilis rashes preceded burning or itch, erosion situated on the hyperemic, slightly edematous basis and It has micropolycyclic outlines. Except Togo, at bubble lichen erosive rashes precede bubbles, which never not arise in processchancre formation. Unlike hard chancre, herpetic erosions are almost always characterized by rapid onset and rapid epithelialization, in addition, herpes in difference from solid chancre often It has recurrent flow. Should take into account, what atlong existence herpetic erosion on the lip in her basis appears infiltrative seal, what reinforces resemblance erosion With primary syphiloma.

secondary period of syphilis begins after 6-7 weeks. after the appearance of solid chancre, when, against the background of symptoms characteristic of the primary period of syphilis (solid chancre, regional scleradenitis, polyadenitis), abundant roseolous-papular rash. Secondary period syphilis continues in flow 3-5 years and accompanied positive serological tests. A feature of the secondary period of syphilisis an undulating course, when periods of active manifestation of the disease are replaced periods hidden, asymptomatic currents disease, and duration everyone from these periods individual (in average on 1.5-2 months).

The active stage of the disease, which develops at the beginning of the secondary period of syphilis due to the generalization of the infection, is characterized by a large amount of roseolous papular, and sometimes pustular rashes, polyadenitis, scleradenitis, remnants hard chancre and is called - secondary fresh syphilis. By the end of the secondary fresh syphilis, a hard chancre is resolved, roseolous-papular rashes disappear,liquidated regional scleradenitis and polyadenitis.

Mucous shell cavities mouth is frequent place localization syphilides secondary period, and at secondary recurrent syphilis rashes in mouth may to be the only clinical manifestation illness. Nearly at half sick With phenomena secondary syphilis observed defeat mucous shells mouth in form roseolous and papular elements, pustular rashes on the

mucous shell mouth arise extremely rarely.

roseolous rashes on the mucous shell mouth arise symmetrically on the temples, soft sky, tongue and tonsils. featureroseolous rashes in this areas is that they merge in solid foci defeat (erythematous angina). Struck region It has stagnant red color, sometimes with a copper tint, sharp borders. The mucous membrane in this the area is slightly swollen; patients feel awkward when swallowing, pain, but subjective Feel may and absent. Permission erythematous sore throats starts With central parts.

The most common manifestation of secondary syphilis on the oral mucosa are papular rashes. They can occur anywhere in the mucosa shells, but bowl on the tonsils, temples, soft sky, where often papules mergein solid foci defeat (papular angina), language, mucous shell cheeks, especially along the line of teeth closing, gums, etc. The type of papules depends on their duration. existence. at first papule - sharp limited Dark red hearth size up to 1 cm in diameter with a small infiltrate at the base. Some time later emerging in result what is happening inflammation exudate impregnates covering papule epithelium, and she is acquires very characteristic view.

The tertiary period of syphilis is not observed in all patients, even if they do not are being treated. It begins 4-6 years after the onset of the disease due to a change reactivity organism, sensitivity his to pale treponema and etc. and It has malignant flow. Tertiary period maybe continue decades characterized by the development of inflammatory infiltrates (gum and tubercles), prone to disintegration and often causing significant destructive, sometimes incompatible with life changes in organs and tissues. At the same time, rashes of tertiary syphilis do not contagious for surrounding, So how in them detachable missing pale treponema.

AT tertiary period syphilis on the mucous shell mouth may to appear gummas, gummy diffuse infiltration and tubercular rashes. At this mucous shell maybe to be the only place clinical manifestations diseases.

Gummy syphilis maybe localize in any mucosal site shells mouth. More often gummas formed on the soft and solid sky and language. Usually gumma appears in the only number. at first formed painless node, which the gradually increases a then opens up. Rejected gummy rod, after which a gummy ulcer is formed. This process lasts 3-4 months, sometimes accompanied insignificant subjective sensations. unopened gumma It has dense consistency smooth surface, mucous shell above the node is moderately inflamed, it has a stagnant red, sharply limited color. After branches rod gummy ulcer It has crater-shaped shape, dense the edges, painless bottom her covered granulations. Ulcer gradually heals With the formation of a stellate retracted scar. When localized in the sky at the place of the gumma often formed perforation, persisting after permissions process.

On the solid sky gumma usually situated on middle lines. Due to Togo, what mucous shell thin and intimately tied With periosteum sky, beginning gummy process very fast passes on the periosteum and bone. Infiltrate gummas fast breaks up and exposed bone, which necrotizesand sequestered arises message between cavities mouth and nose.

Treatment sick syphilis maybe to be started only after confirmation clinical diagnosis by detection pale treponem at primary and secondary syphilis or positive serological reactions. Under influence antisyphilitic treatment rashes fast disappear and already through 8-10 hour.after the start of penicillin therapy, pale treponemas are not found on the surface rashes. In this regard, patients with syphilis after 10-12 hours. after the start of treatment nicillin practically not contagious at household contact, a also at examination them doctors,in volume including dentists.

A dentist in his practice may encounter patients with tertiary syphilis, which the only manifestation of the disease may be gummy or tuberculate rashes on the mucous membrane of the mouth. Treatment of such patients should not begin with the introduction penicillin, as it will cause an exacerbation reaction, which will stimulate a rapid resorption syphilitic rashes what maybe lead to catastrophe, even to of deathsick, if such rashes are localized in vital important organs. it related With topics what at such treatment resorption infiltrate happen per 2-3 day , during which not the connective tissue replacing them will have time to form. For this reason , the treatment of patients tertiary syphilis should always start with iodine for 2-4 weeks, then enterhalf the course dose of the bismuth preparation and only then penicillin, after which the second half the course dose of the drug bismuth; the second and subsequent courses of treatment begin, how usually, those. With penicillin.

Dentist maybe meet With sick, which the moved tertiary or late congenital syphilis and at whom available perforation sky, requiring plastic surgery. It should be borne in mind that patients with syphilis after treatment 5 years are on the dispensary accounting, in flow this time at them determine curability syphilis. AT connections With this plastic operation so patients should be done after deregistration. If, however, there is a need for operations before this term, then operational intervention necessary conduct under protection of penicillin, in this case, the value of the total dose of the drug is determined collectively With venereologist, under observation whom located sick.

In the treatment of manifestations of syphilis in the oral cavity, complications may occur, related With application penicillin and drugs bismuth. Penicillin and his drugs can cause acute allergic drug-induced stomatitis, due to which necessary stop introduction penicillin, and candidiasis. Last thing complication at sick syphilis not requires mandatory cancellation penicillin. Complications from drugs bismuth are bismuth border, bismuth gingivitis and stomatitis.

Gonorrhea.

Gonorrhea is caused by Neisseria gonorrhoeae. It's highly contagious venereal disease characterized defeat not only urogenital tract. There are extragenital lesions: arthritis, oral lesions and throats. Percent recent manifestations sharp increased in connections With spread oral sex and homosexuality. On the lips at gonorrhea may to be ulcerative defeat, the gum becomes edematous, inflamed and resembles the picture is ulcerative - necrotic gingivitis. Language, mucous cheeks may to be hyperemic and ulceration.

Viruses are most frequent cause infectious diseases(measles, epidemic mumps, rubella, flu, windmill smallpox, hepatitis, AIDS and etc.).

Another virus for which parenteral transmission is also maybe to be basic, - this is virus immunodeficiency human (HIV). Virus HIVstrikes lymphocytes (T-helpers) and macrophages. At long reproductions virus inbody populations helpers is shrinking in affected macrophages declining production interleukin-I. Except immune systems suffer other bodies and systems: nervous system, bodies digestion and breath, cardiovascular system , etc. Against the background of immunodeficiency , Kaposi 's sarcoma develops and opportunistic diseases (candidiasis, ulcerative necrotic stomatitis, cytomegalovirus infection, herpes, pneumocystosis).

Sources virus are sick and virus carriers. Available parenteral route of infection when using an infected virus blood with various surgical interventions, blood transfusion, etc., as well as sexual contact. Registered cases infections fetus in intrauterine period.

Diagnostics held main way serological method, through identifying antibodies With help enzyme immunoassay analysis.

Thus, patients who came to the reception with candidomycosis, relapses herpes, ulcerative necrotic lesions must be checked on the infection HIV. special danger is posed by patients at risk - homosexuals, prostitutes, drug addicts, patients who often received blood transfusions (hemophilia).

A dentist can become infected with a virus from a patient airborne by and during a digital examination, as well as the doctor himself during his manipulations maybe infect patient (hepatitis AT, AIDS).

Additionally necessary Mark, what about near 46 % patients on the reception can be carriers in the oral cavity of Streptococcus puogeus, Staphul. aureus. They are can cause the doctor to develop various respiratory diseases, as well as cause an inflammatory process in case of a hand injury with a drill or a stab wound. Considering that, that many of the staphylococci are antibiotic-resistant, the entry of these microbes maybe make it difficult process treatment.

Leprosy

Leprosy (leprosy) called leprosy mycobacteria Hansen and represents yourself chronic generalized infection developing predominantly in derivatives ectoderm and in bodies and fabrics, rich elements active mesenchyme. Leprosy leaks chronically progressive With periodical exacerbations (leprosy reactions), leading in the absence of rational treatment for severe disability and through a lot of years to of death.

Allocate tuberculoid and lepromatous types leprosy. emergence Togo or another

type of disease related with fortune resistance organism human to leprosy infection, which determine With help lepromine skin samples. Mucosal lesions shells cavities mouth may arise only at lepromatous type, which the develops at persons With sharp reduced reactivity (lepromine try negative). At such sick on the skin, mucous shell mouth, internal bodies, on move nervous trunks arise lepromatous infiltrates With leprosy cells not able (in difference from tuberculoid type) destroy phagocytosed them sticks Hansen, that's why they freely multiply in these cells.

Leprous process on mucous shell mouth begins with infiltrative stage, then on the this background arise tubercular rashes which through some time ulcerate. Process ends scarring. Highly often on the mucous shell mouth at one and Togo same sick can observe elements, characteristic for all four stages development leprosy defeat mucous shells, t. e. infiltration tubercles, ulcers and scarring. Clinical painting leprosy lesions of the oral mucosa is characterized by polymorphism, each of four stages characterized described signs.

Leprosy changes mucous shells mouth begin With education superficial limited infiltrate, slightly rising above the surrounding mucous shell and having grayish white color, sometimes With dark blue plots. Then on the infiltrated plots arise tubercles various sizes - from millet grain to a cherry stone. At first, the tubercles are dense, then soften. They are located haphazardly and inclined to peripheral growth and merger. Tubercles mostly dull pink, sometimes grayish pink colors. Them surface usually shiny. More often Total tubercles arise on the solid and soft sky, language and lips. Through some time tubercles usually ulcerate. ulcers on the place tubercles at first small, bottom them bumpy, dirty gray white colors, the edges uneven, swollen, soft consistency. Sometimes ulcerative process distributed by on the bone the cloth, causing her meltdown. More often Total at this collapses alveolar edge jaws. AT subsequent ulcers scarred but scarring may develop without previous ulceration tubercles, when the tubercle or infiltrate of the oral mucosa are fibrosed. Scar formation is not yet means recovery, for simultaneously may arise new infiltrates, tubercles and ulcers. Leprosy scarring on the mucous shell cavities mouth may to be round or in form stripes — radiant. Scarring smooth, shiny, white colors. formed scarring in dependencies from them sizes and places location may cause functional disorders. Especially often the soft palate is deformed and tongue, which the maybe shift a sometimes even disappear.

Leprosy defeat may arise and on the lips. At this very often a pronounced infiltrate is formed, accompanied by edema, which entails education leprosy elephantiasis. Lip compacted, becomes more thick, roller-shaped and sedentary. Leather lips and red border on bloom few different friend from friend. On the this background arise Good contoured tubercles.

Leprosy changes on the inner surface lips begin With diffuse erythema, then an infiltration occurs view bluish spots, covered with thickened epithelium. Against this background, sometimes bumps appear, which nearly not act above mucous shell. Leprosy tubercles on the lips may long time remain unchanged but sometimes they ulcerate. The emerging shallow painless ulcers located on the surfaces tubercles. Them detachable dries up in light yellow crusts.

Ulcerative defeat mucous shells lips always ends scarring with deformity of the lips, resulting in a thinner lip, the mouth opening may narrow down. If a same deep located tubercles fibrosis, then lip wrinkled, due to what finds it difficult speech, leather lips in these cases happens dotted furrows a red border lips becomes wrinkled. Leprosy changes mucous shells lips rarely reach before transitional folds.

Beloved localization leprosy rashes are gums on the topjaws from the side of the tongue in the area of the frontal, less often molars. Leprosy changes in the gums begin with the formation of an infiltrate. The gums seem to swell are made loose, red, sometimes cyanotic slightly bleed; gingival papillae swell up picture gingival the edges smoothed out. Highly often to this

increased salivation joins. Soon the gingival mucosa becomes matte, on the her surfaces formed sores, which then scarred what leadsto wrinkling of the gingival margin and turning it inward. The gums retract, the roots of the teeth exposed. Simultaneously with scarring in other areas of the gums, fresh infiltrate. characteristic painless process.

Leprosy changes on the mucous shell solid sky are happening next way: in those cases, when defeat begins on the front thirds at first noted infiltrative stage, infiltrate formed behind necks central incisors and comes before fangs. Plot infiltrated mucous shells on direction back narrows and more often ends at the beginning middle thirds solid sky, in result what formed triangle, basis facing forward, and the top back. Significantly less often infiltrate in the direction narrows back to a strip 2 cm wide and extends along the midline to soft sky. Infiltrate on the lateral surfaces solid sky observed extremely rarely. infiltrated plot greater part It has grayish red color and absolutely painless. Then on the infiltrated plots arise grayish white tubercles magnitude With millet corn, which subsequently superficially ulcerate.

On the soft sky defeat starts With hyperemia, passing in infiltrate dark coloring. Sometimes soft sky at first It has pale yellow color. tongueusually infiltrated, its size increases. On the infiltrated plots soft sky and tongue appear whitish gray, focally located tubercles various quantities — from millet grain before peas. Then tubercles break apart and formed sores. Sometimes individual sores merge, forming greater or lesser quantities solid ulcerative surfaces. The edges ulcers slightlyraised and undermined, bottom bumpy With grayish raid. Color them dirty grayish white. Due to ulcerative defeat usually collapses part or the whole tongue.The ulcerative process on the hard and soft palate ends with scarring. The emerging scarring have varied form: sometimes they round, but more oftenradiant or stellate. Scarring usually shiny, superficial, whitish colors. Cicatricial contraction on the solid sky near necks front teeth leads to gingival wrinkling and retraction her with exposure of the roots of her teeth. The edges retracted gums wrap up inside in side roots, tight hugging them. Scarring on the soft palate sometimes form an arc, the convex part facing forward. fibrosis in areas soft sky often causes persistent deformation, due to what soft sky maybe pull up up, narrowing input in nasopharynx. Sometimes soft sky absolutely is destroyed.

Beloved localization leprosy elements on the language is average line his backs, beginning from root and before tip. Language maybe infiltrate increase, thicken, in result what his mobility finds it difficult and speech becomes obscure. On the infiltrated surfaces backrests language appear dense tubercles various quantities, but not more bob, With flat surface and wide basis. Surface tubercles shiny, covered whitish raid thanks to desquamation epithelium ("silver" language). Number tubercles may enlarge and may coalesce (leprosy glossitis), resulting in what on the back of the tongue are formed roller-shaped elevations with deep furrows between them.

tubercles on the language inclined to decay and ulceration. formed ulcers located superficially and have jagged, undermined, infiltrated edges. The bottom of the ulcers shallow, rough, covered with a grayish coating. In some cases, these ulcers merge, forming a continuous ulcerative surface, covered with a thin gray coating. Emerging on the place ulcers various forms superficial scarring usually have greyish white color and brilliant surface.

For the diagnosis of leprosy , bacterioscopic studies of scrapings from mucous shells nasal partitions, a also co bottom and edges leprosy ulcers, in whicheasily discover sticks Hansen.

Leprosy lesions of the oral mucosa may resemble tertiary syphilis and lupus erythematosus. From the manifestation of lupus erythematosus and collicative tuberculosis, leprosy rashes are characterized by greater density, the presence of pronounced disorders sensitivity and leprosy mycobacteria in ulcerated dischargeelements.

Most effective antileprosy means are drugs sulfonic row, such, how diamino diphenyl sulfone (DS), avosulfone, dapsone, sulfetron (solusulfone, novotrope), which accept in flow long time. The less toxic drug thiocarbonylide has the same effect as sulfones. (derivative thiourea). Not lost meaning chaulmugrious oil and his derivatives, inin particular ethyl ethers and mugrol. In order to successfully treat patients with leprosy, always should apply combined therapy everyone famous antileprosymeans in combined With restorative drugs and physiotherapy.

Forecast at leprosy in recent decades in connections With introduction in practice

treatment this disease of new, sufficiently effective drugs has become better. Main public prevention of leprosy is early detection and rapid isolation of patients with leprosy in special institutions - leper colonies, where patients are treated, live and work. At discovery sick or suspicious on the disease leprosythe doctor must immediately report this to the health authorities. Upon confirmation of the diagnosis leprosy sick With compliance measures precautions envisaged instruction People's Commissariat ways messages — People's Commissariat health care from 19/IV 1944 G., must to be delivered in leprosarium.

herpes

Lichen lichen simplex (syn. herpes simplex) is one of the most common diseases of the human oral mucosa. Lips and oral mucosa are favorite localization herpes. Virus herpes starts his development in cellular core.Once in the body and causing manifestations of a primary herpetic infection, it remains in body human, apparently in flow all life in latent able or causes relapses illness.

Herpes can occur in a person once in a lifetime, and may have recurrent flow. Relapses develop in different people at different times and are characterized by unequal intensity of the process. The occurrence of relapses is facilitated by many factors that reduce the body's defenses: cooling, overheating, pneumonia, tonsillitis, diseases of the gastrointestinal tract, menstruation, etc. More often herpetic infection appears in form local defeat, but sometimes she is acquires generalized character, more often at newborns.

Simple bubble lichen maybe arise at of people any age. Manifestationsherpes can be noted on the skin and mucous membranes. The disease begins withone or two, less often more limited foci of hyperemia, against which small, the size of a millet grain or a little more, bubbles are quickly formed. Quantityin bubbles maybe vary from 2-3 before 10-15 or more. bubbles are located groups,contain a clear liquid, then their contents become cloudy. Sometimes as a result of clusters liquids small bubbles merge in one or two bubble diameter before 1.5 cm.On the lips through 2-3 days liquid shrinks in yellowish gray peel.

Often bubbles open up With education erosion bright red colors With polycyclic outlines. On the oral mucosa, more pronounced inflammatory reaction, bubbles open up in first watch after appearance. erosion on the them place have wrong scalloped outlines and covered delicate fibrinous film. The process on the oral mucosa can be limited or widespread - herpetic stomatitis. After 8-12 days, and in in some cases, slower, epithelialization of erosion occurs. Eruptions of bubbles accompanied tingling. AT individual cases rashes accompanied strong swelling of the surrounding tissue. The general condition, as a rule, does not suffer, however, some patients report malaise, muscle pain, chills. Body temperature may be subfebrile or rise before 38-39°C.

A type of herpes simplex is recurrent herpes. Relapses occur with different frequency, at different times and regardless of the season. Some patients relapses are observed 3-4 times a month, the disease actually takes permanent flow. Recurrent herpes does not differ in clinical manifestationsfrom simple herpes.

Acute herpetic stomatitis. Until recently in domestic literature on dentistry and pediatrics described two independent diseases: spicyaphthous and spicy herpetic stomatitis. Clinical and laboratory survey a large group of patients using the arsenal of modern virological, serological, cytological and immunofluorescent methods research earnestly showed clinical and etiological unity these diseases.

Received data allowed recommend single term and call the disease "acute herpetic stomatitis", based on the viral etiology of the disease. This stomatitis occupies one of the leading places in children's infectious diseases. pathology, meeting more often scarlet fever, measles, epidemic mumps and only a littleyielding wind smallpox

Spicy herpetic stomatitis among non-immune persons It has relatively high contagiousness. So, in preschool institutions and in hospital wards at epidemic outbreak maybe get sick before $^{3/4}$ _ _ children.

Broadcast infections going on obviously, contact and airborne way.

For development diseases It has meaning violation intact mucous shells and skin covers. A clear seasonality of the occurrence of the disease can not be identified. The highest prevalence of the disease among children aged 6 months to 3 years explained topics

what at them disappear antibodies, received from mothers interplacentally, a also inadequate maturity systems specific immunity.

Spicy herpetic stomatitis, how and other infectious diseases, It has fiveperiods development: incubation, premonitory (catarrhal) periods developmentdiseases (rashes), extinction and clinical recovery (reconvalescence). AT dependencies from degree expressiveness general toxicosis and local manifestations in cavities mouthdisease maybe leak in light, middle and severe form.

From general symptoms characteristic hyperergic reaction With rise temperature body before four HS and above at severe form diseases, malaise, weakness, head pain,skin and muscular hyperesthesia, absence appetite pallor skin covers, nausea and vomiting of central origin, since the herpes simplex virus is encephalotropic. Already in the incubation and especially in the prodromal period, clearlycomes to light increase submandibular, a in heavy cases and cervical lymph nodes.

On the pique rise temperature arises hyperemia and puffiness mucous shellsoral cavity; on the inflamed mucous membrane of the lips, cheeks, tongue appear from 2-3 to several dozens close located friend to friend groups bubbles, which fast are opened. On the them place arise erosion With necrosis in center size 0.5—1 cm, very resembling aphthous elements. On the lips, rashes quickly become covered with crusts. At this defeat maybe localize not only in cavities mouth, but and on the skin face, especiallyoften this is observed in severe form of the disease. Due to the fact that the rash continue arise in flow several days at examinations can see elements lesions at different stages of development. Reappearance of rashes accompanied by a deterioration in the general condition of the child, anxiety or adynamia and rise temperature body on the 1-2°C on comparison With previous for days. Compulsory a symptom of acute herpetic stomatitis is hypersalivation, saliva becomes viscous and viscous, noted smell iso mouth.

Already in the catarrhal period of the disease, pronounced gingivitis often occurs, which in the future, especially in severe form, acquires an erosive-ulcerative character. noted pronounced bleeding gums and mucous shells cavities mouth.AT blood children With severe form diseases discover leukopenia, stabshift to the left, eosinophilia, single plasma cells, young forms of neutrophils.Sometimes in urine appears protein. AT saliva first determined shift pH in sour side, then - into alkaline, while interferon is usually absent in saliva, and the content lysozyme is markedly reduced. Humoral factors of the body's natural defenses, including including phagocytosis, in period swing diseases also sharp reduced.

The diagnosis of acute herpetic stomatitis is established on the basis of clinical patterns and epidemiology of the disease. To clarify the diagnosis, it is recommended to perform cytological examination of material from herpetic erosions, which is stained according to Romanovsky-Giemsa to discover the so-called giant multinucleated cells,which characteristic for herpes.

A very promising method for the etiological diagnosis of the disease is the method immunofluorescence, which the allows get result in flow 2.5-3 h With momentfence material.

Treatment of lichen lichen simplex in adults is to lubricate the focus lesions 2-3 times a day with alcohol solutions of aniline dyes or ointments, containing antiviral substances (1-3% oxolinic, 3% octathionic, 2-5% tebrofenovaya, etc.). A good effect is given by leukocyte interferon, the solution of which applied to the affected area 6-7 times a day. In the treatment of recurrent herpes necessary apply funds, increasing protective strength organism (gamma globulin, pyrogenal and etc.). positive action renders herpetic polio vaccine, whichinjected intramuscularly at 0.1-0.2 ml with an interval of 2-3 days, for a course of 10 injections. Deoxyribonuclease gives a good effect, which administered parenterally by 10-50 mg 2 times in a week on the well 6-10 injections.

Tactics doctor at treatment children, sick sharp herpetic stomatitis, must determined degree gravity diseases and period her development. At middle gravity andin severe cases of the disease , it is advisable to carry out general treatment together with the pediatrician. Considering,that these forms of the disease develop against the background of a significant decrease in the protective forces organism, appropriate apply means of stimulating immunity (ly-zozyme, prodigiosan, gamma globulin parenterally; methyluracil, pentoxyl, sodium nucleinate), at the same time , desensitizing therapy should be carried out (diphenhydramine, suprastin, pipolfen, calcium gluconate, etc.). Local treatment for acute herpetic stomatitis is antiviral therapy. Assign 0.25-1% oxolinic, 1-2% florenal, 1-2% tebrofen, four% heliomycin ointment, one% solution deoxyribonuclease, liniment helepin, mixture interferon With prodigiosan and others interferonogens. These drugs are used 3-4

times a day. Antivirals _necessary apply on the all mucosa shell, a not only on the affected plots, So as they provide both curative and preventive effects. Once a day oral cavity recommended handle 0.1— 0.5% solution proteolytic enzymes (trypsin, chemotrypsin, pancreatin and etc.), which contribute dissolution necrotic fabrics.

AT period fading disease basic meaning attach application weak antiseptics and keratoplastic funds. Good ones results give applications oil solutions vitamin A BUT, oils wild rose, carotolina, ointment and jelly solcoseryl, ointments With methyluracil, livian, levovy-nizol. AT quality antimicrobial funds can apply solutions furatsilina, ethacridine lactate, ectericide, aetonia and etc. Sick must accept liquid or semi-liquid writing, not annoying inflamed mucosa shell, receive sufficient amount liquids. Before reception food necessary anesthetize mucosa shell of the mouth 5% anesthetic emulsion.

AT conclusion should Mark, what spicy herpetic stomatitis, flowing in any form, is contagious disease , all cases required Attention co sides pediatrician and dentist for Togo to provide comprehensive treatment, exclude contact sick co healthy and spend preventive Events in children's collectives. AT children's institutions, especially in nurseries, should not be allowed to work with children employees in period manifestations herpes, regardless from his localization. At identifying in children's institution child, sick sharp herpetic stomatitis, he is not allowed to attend a kindergarten, even if disease leaks in very light form.

Shingles. This disease is caused by a virus identical in their antigenic properties virus wind smallpox. This virus is different neurodermatotropism With prevailing action on the central and peripheral nervous system. Disease often develops on the background sharp weakening protective properties organism, often how complication pneumonia, diseases blood and others exhausting diseases.

Disease starts With pain on move affected peripheral nerves always With one sides. On the face and mucous shell mouth appear paresthesia and pain on move one or two branches trigeminal or facial nerve. Then appears erythema in form spots, sometimes merging in stripes, where formed groups small bubbles, filled serous or hemorrhagic content. rashes, sharp limited, unilateral, come to light on move affected nerves. On the mucous shell cavities mouth these bubbles fast are opened, forming erosion With scalloped outlines.

The entire thickness of the cheek in the affected area may be edematous and infiltrated, submandibular lymphatic nodes increased painful. At defeat motor and sensitive fibers facial nerve sometimes develops Ramsay Hunt syndrome, including herpes zoster, facial paralysis nerve and ear pain. The disease lasts 3-6 weeks. and spontaneously resolved. Disease accompanied pains neuralgic character, which may persist and after liquidation rashes.

AT diagnostic respect important meaning It has one-sidedness defeat, painful syndrome, localization rashes strictly in zone innervation affected nerve, absence relapses. Last thing distinguishes herpes zoster lichen from Herpes simplex recurrences.

Treatment is in appointment antibiotics wide spectrum actions, salicylates, vitamins AT and At $_{12}$, deoxyribonuclease, interferon, analgesics and others Mucous shell mouth handle antiviral ointments (0.25-0.5% oxolinic, 0.5-2% tebrofen, 1-2% florenal) or interferon solution (32 U in 1 ml). During the period of resolution of rashes are shown keratoplastic funds: oil wild rose, sea buckthorn, carotenoline, ointments, containing vitamin BUT.

herpetic angina. Disease called enterovirus coxsackie groups BUT, starts acute: With rise temperature body, myalgic pain, general ailments. AT rear department mouth: on the soft sky, front temples, tonsils and rear wall pharynx –appear painful grouped and

single vesicles filled serous or hemorrhagic content. AT Subsequently, part of the vesicles disappears, others open, forming erosions. merger small erosion leads to the formation of eroded areas of different sizes with scalloped outlines. Some from erosion may remind aphthae. erosion not painful, epithelialize slowly, sometimes in flow 2-3 weeks Described cases diseases members one families and even epidemic outbreaks.

Treatment consists in symptomatic general therapy and topical application in the first 2-3 days of antiviral, and later keratoplastic agents. Frequent rinsing and lubrication slow down process epithelization erosion.

TEST TASKS:

1. Practical application RSC:
a. syphilis serodiagnosis - Wasserman reaction,b.
serodiagnosis tuberculosis
in. seroidentification of pure cultures of bacteria on glass,G.
seroidentification bacterial antigens
2. To treponemal tests include:
a) reaction immunofluorescence b)
treponema immobilization reactionin)
Wassermann reaction
G) sedimentary reactions

3. For systemic treatment candidiasis apply:
a - lamisil (terbinafine)b -
itraconazole (orungal)
c - fluconazole (diflucan)G -
ketoconazole (nizoral)

4. Group bacteria, With activation which bind development of periodontitis applies tochildbirth:
a - lactobacillib -
prevotell
c - staphylococciG
- actinomycetes

5. causative agents juvenile periodontitis are:
a - leptotrichia b -
streptococci
in - actinobacilli G -
bifidobacteria

PRACTICAL OCCUPATION No. 11.

Topic: Microflora in prosthetics and implantation of teeth. The study of adhesion and colonization of oral bacteria on dental materials. Diagnosticsperi-implantitis and their prevention.

Educational goal:

explore meaning resident microflora cavities mouth in development complications of dental implantation and principles of prevention of post-implantation complications inflammatory character.

Student must know:

1. Sampling material for microbiological examination _ _osteomyelitis.
2. Feature microorganisms, being most frequent pathogenspost-implantation complications.
3. Sensitivity to antibiotics.

Student must be able to:

1. Spend fence material on the antibiotic sensitivity.
2. Holding bacteriological method research (on scheme).
3. Spend accounting results.

Plan lessons:

1. Representatives of what oral biotopes are the most common pathogenspost-implantation complications?
2. Ways infections zones implantation, related With contamination bone lodgeimplant and seam lines.
3. Pathogenesis and clinical forms post-implantation complications inflammatorycharacter.
4. Fence material for research at peri-implantitis and osteomyelitis.
5. Prevention post-implantation complications inflammatory character.

Independent Work students:

1. Take into account results crops microflora mucous shells cavities mouth before and after use self-adhesive films DEPLEN-DENT and bring in to the protocol.
2. Microscopy smears from pure cultures anaerobic bacteria, dedicated atperi-implants:

a) convertibles
b) peptostreptococcic)
fusobacteria

3. Decor protocol research.

Table. Fence researched material from prosthetic lodge.

researched material	Description results microscopy	Picture

PRACTICAL OCCUPATION No. 12.

TEST CONTROL.

No. СТОМ-21-ИН

Federal State Budgetary Educational Institution of Higher Education
NORTH OSSETIAN STATE MEDICAL ACADEMY
Ministry of Health of the Russian Federation

Department of microbiology

COLLECTION METHODOLOGICAL DEVELOPMENT ON MICROBIOLOGY, VIROLOGY, IMMUNOLOGY - MICROBIOLOGY MOUTH FOR DENTAL FACULTYFOR TEACHER

Autumn semester

Vladikavkaz

Authors: Associate Professor of the Department of Microbiology, FSBEI NOSMA of Ministry of Health Russia., Ph.D. Chertkoeva M.G., Assistant Khabieva B.A.

The main purpose of the developments is methodological assistance to teachers to to each practical occupation in IV semester. Directions drawn up incompliance With Federal public educational standard Supreme and vocational education.

REVIEWERS:
L.V. Bibaeva – MD, Professor, head department biology and histology FGBOUIN SOGMA Ministry of Health Russia.

A.R. Kusova- MD, Professor, head department of hygiene and physical education FGBOU VO SOGMA Ministry of Health Russia.

methodical recommendations approved on the meeting TsUKMS FGBOU IN "North-Ossetian state medical academy Ministry of Health Russia" from , protocol

PRACTICAL OCCUPATION No. one.

Topic: Microbiological diagnostics viral diseases. Indication and identification viruses in researched material. Serological method diagnostics viral diseases: reactions neutralization, passive hemagglutination, ELISA. Working off methods diagnostics on the example viraldiseases:
- cultivation in chicken embryo, colored try, hemagglutination and braking hemagglutination at identification viruses influenza and SARS;
-serological tests and polymerase chain reaction in the diagnosis of viral hepatitis B, C, herpes, HIV.

Educational goal:

4. Explore morphology and ultrastructure viruses.
5. Train students methods virological diagnostics and specific prevention flu, SARS.
6. Train students methods virological diagnostics and specific prevention hepatitis B, C, herpes, HIV infections.

Plan lessons:

12. Peculiarities biology viruses **.**
13. Principles classification viruses.
14. Types interactions viruses With cell.
15. Taxonomy and main biological properties pathogens flu, SARS.
16. Epidemiology, pathogenesis, immunity called diseases
17. Principles microbiological influenza diagnosis, SARS.
18. Preparations for etiotropic therapy and specific prevention flu,SARS.
19. Taxonomy and main biological properties pathogens hepatitis b , FROM,HIV infection herpes
20. Epidemiology, pathogenesis, immunity called diseases.
21. Principles microbiological diagnostics hepatitis b , FROM, HIV infection,herpes.
22. Preparations for etiotropic therapy and specific prevention hepatitisB, C, HIV infections, herpes.

Independent Work students:

7. Parsing supplies and accounting results REEF at SARS (demonstration).
8. Parsing supplies and accounting results RTGA for seroidentification with flu (demonstration).
9. Parsing supplies and accounting results ELISA for serodiagnosis with SARS(demonstration).
10. Parsing productions and accounting reaction results ELISA for serodiagnosis andseroidentification at hepatitis B, C, HIV infections (demonstration).
11. Analysis of the formulation and accounting of the results of the reaction of RPHA in hepatitis B (demonstration).
12. Parsing productions and accounting RTGA results and ELISA for serodiagnosis

(demonstration).

EQUIPMENT

1. Accounting results REEF at SARS (demonstration).
2. Accounting results RTGA for seroidentification with flu (demonstration).
3. Accounting results ELISA for serodiagnosis at SARS (demonstration).
4. Accounting results reactions ELISA for serodiagnosis and seroidentification athepatitis B, C, HIV infections (demonstration).
5. Accounting results reactions RPGA at hepatitis AT (demonstration).
6. Accounting results RTGA and ELISA for serodiagnosis (demonstration).

INFORMATIONAL MATERIL ON THEME

VIRUSES

Viruses have properties that make it impossible to use conventional methods to study them. methods microbiological research.

Distinctive properties viruses:

1. smallest sizes, measurable thousandths shares micron - millimicrons
- from 8-10 m up to 300-400 m.
2. Filterability through special finely porous filters, not passingother microorganisms.
3. non-cellular structure.
4. Absolute parasitism, those. ability live and multiply only in alivecells.

The form viral particles It has several types:

11. Rod-shaped 12. Spherical
(spherical)13.Cuboid
14. Capitate (spermatozoa)15. Filiform

mature viral particle, called *virions,* have next scheme buildings: in central parts located molecule DNA or RNA, which forms *nucleoid.* Around situated protective protein shell, called *capsid,* built from morphological units, called *capsomeres.* Some complex virions have external shell, called *supercapsid.*

For microbiological diagnostics viral infections in the present time apply three main methodical approach:

7. **Virological diagnostics** - founded on the allocation from researched material virus and his subsequent identification.
8. **Serological diagnosis** - determination of specific immunological changes in body under action viruses (more often Total With help diagnosticums reveal in serum blood antiviral antibodies).
9. **Molecular biological diagnostics** - detection in clinical material fragments nucleic acids causative viruses With help probes (hybridization NK) or PCR.

Individual viruses larger than 200 m can be stained according to Romanovsky - Giemsa; smaller viruses (variola viruses) can only be detected using special processing methods.

bacteriophages differ on chemical structure, type nucleic acid, morphology and nature of interaction with bacteria. Size of bacterial viruses in hundreds and thousands of times less microbial cells.

A typical phage particle (virion) consists of a head and a tail. Tail length is usually2-4 times the diameter of the head. The head contains genetic material - single stranded or double stranded RNA or DNA With enzyme transcriptase in inactive state, surrounded protein or lipoprotein shell - ***capsid*** , preserving genome outside cells.

Nucleic acid and capsid together make up the nucleocapsid. Bacteriophages can have icosahedral capsid, assembled from sets copies one or two specific proteins. Usually angles are made up of pentamers squirrel, and support each side from hexamers Togo same or similar squirrel. More Togo, phages on form may to be spherical, lemon-shaped or pleomorphic. Tail represents yourself protein tube - a continuation of the protein shell of the head, at the base of the tail there is ATPase, which regenerates energy for the injection of genetic material. There are alsobacteriophages With short offshoot, not having offshoot and filiform.

By international classification all viruses subdivided on type nucleicacids on the 2 subtype - RNA- and containing DNA. Further separation virusesis carried out on the basis of the size of the viruses, the type of symmetry in the formation of capsids,the presence or absence of outer shells and the number of capsomeres contained in them.VIROLOGICAL METHOD RESEARCH is main and most authentic, allows highlight virus from researched material With subsequent hisidentification. FROM purpose accumulation virus-containing material are used chicken embryos and culture fabrics (artificially cultivated cells toy ordifferent fabrics). cultures fabrics supported on the natural (Wednesday 27, Enders) andsynthetic (medium 199, Needle, Melnik-Riordan) nutrient media, cooked on the basis solutions Hanks and Earl. cultivated they in conventional test tubes cups carrel, test tubes Barsky.

Methodology infections chicken embryo

There are several ways to infect a chicken embryo. Most often the material injected into the allantoic and amniotic cavities, onto the chorionallantoic membrane and into yolk bag. Before infection shell eggs above air camera treated with 70% alcohol, burned on a flame, smeared with 2% iodine tincture, secondarily wipe with alcohol and burn.

At contagion in allantoic cavity in shell above air camera (borderswhich in advance circle pencil at translucence eggs in ovoscope) are doingsmall hole With help scissors or scalpel. Tuberculinov syringeintroduce 0.1-0.2 ml virus-containing material on the depth 2-3 mm below bordersair cameras. Puncture in shell poured molten paraffin. Openinginfected embryos produce in terms maximum accumulation virus (through 48-72 h incubation at temperature 37 FROM) after processing shells alcohol and 2% solution iodine her dissect and dump, filmed carefully shelledshell and consider chorionallantoic shell around places infections on theAvailability foci lesions (hemorrhages, whitish foci defeats).

Classification cellular crops:

• **primary** receive directly from fabrics animal and human through destruction proteolytic enzymes (trypsin, collagenase) intercellular substances. Disunited cells, placed in nutritional Wednesday, able attach to the surface of the culture vessel and multiply, forming a monolayer - layer one cell thick. With the help of special reagents, cells can be removed from surfaces one vessel and transplant in another. Such manipulation called **passage.** primary crops withstand not more 5-10 passages.

• **transplantable** (passage) cellular culture able withstand unlimited number of passages. They originate from tumor cells that have lostdifferentiation and not having restrictions growth.

• **semi-transplantable** (diploid) culture - fibroblast-like cells, which able to fast reproduction, withstand before 30-60 passages and save original set of chromosomes.

Viruses can reproduce only in the cells of a living organism. Concerning viruses cultivated through infections chicken embryos or cultures fabrics, a also suckling animals.

Detection (indication) of viruses *Virus*
detection in chick embryo1. Death
2. The appearance of an odor upon
opening 3. Cloudiness liquids in
cavities
four. Education sores and hemorrhages on the shells

Biological method research is in contagion sensitive to virus animal researched material, studying clinical and pathoanatomical paintings diseases. AT framework this method are used various animals: monkey, rabbits, maritime pigs, dogs, mice, rats. Ways infections: subdural, intracerebral, intranasal and other.

Methods for detecting the virus in the body of laboratory animals differ in dependencies from the view animal and type virus.

Detection viruses in culture cells

Revealing on cytopathic action (CPD). JPC represents yourself degenerative changes in cells that result from reproduction in them viruses.

Distinguish complete and partial degeneration cells monolayer.

With complete degeneration caused, for example, by polio viruses, Coxsackie and ECHO, cells of the monolayer undergo significant changes, more of themslough off co glass. Remaining

single the cells are wrinkled

Partial degeneration It has several varieties:

5 .By type cluster formation (adenoviruses);

6 .By type focal destruction (smallpox, flu);

3.By type symplast formation (measles, mumps, parainfluenza, herpes, HIV).

Proliferative type of changes typical for some oncogenic viruses, transforming cells in malignant.

Intracellular inclusion formed at reproductions some viruses in cytoplasm and nucleus of cells (smallpox, rabies, influenza, herpes, etc.) They are found when microscopy after coloring monolayer on Romanovsky - Giemse, a also at luminescent microscopy.

Salk color test. As a result of the vital activity of cells in a nutrient medium accumulate sour products. AT result this color incoming in compound environments indicator (phenolic red) becomes orange. At contagion culture cells with cytopathogenic viruses such as enteroviruses or reoviruses, metabolism cells suppressed medium pH and her color not are changing (Wednesday remains red).

Reaction hemagglutination. AT basis this reactions lies ability viruses, containing hemagglutinin receptors, "glue" erythrocytes. If a there is hemagglutinins - RGA+(umbrella), if No - RGA - (button).

Reaction hemadsorption. Mechanism similar With RGA.

Flu (from fr. grippe) — acute infectious disease respiratory ways, caused by the influenza virus. Included in the group of acute respiratory viral infections(ARVI). Periodically spreads in the form of epidemics and pandemics. Currentlymore than 2000 variants of the influenza virus have been identified, differing in antigenic spectrum.

First virus was highlighted in 30s of the year XX century. Viruses influenza relate to family Orthomyxoviridae, which includes childbirth Influenza A, b, FROM. Antigenic properties internal proteins virion (M1 and NP) determine belonging virus influenza to the genus BUT, AT or FROM.

Viruses containing three subtypes of HA are of epidemic importance to humans. (H1, H2, H3) and two NA subtypes (N1, N2). Influenza A and B viruses contain NA and HA in quality major structural and antigenic components viral particle, possessing hemagglutinating and neuraminidase activities. The influenza virus C no neuraminidase, it has instead a hemagglutinin-esterase (penetrating)protein (HEF). The RNA strand is surrounded by protein and packaged in a lipoprotein membrane. Virions able agglutinate erythrocytes and elute in them With help virus-specific enzymes.

Virus influenza It has spherical form diameter 80-120 nm, in center there are RNA fragments enclosed in a lipoprotein membrane on the surface which has "spikes" consisting of hemagglutinin (H) and neuraminidase (N). Antibodies produced in response to hemagglutinin (H) form the basis of immunity against certain subtype pathogen influenza

source infections is sick human With explicit or erased form disease that releases the virus with coughing, sneezing, etc. The patient is contagious from the first hours diseases and before 5th–7th days disease.[5] Characterized aerosol (inhalation tiny drops of saliva, mucus that contain the influenza virus) transmission mechanism and extremely fast spread in form epidemics and pandemics. Epidemics influenza caused by serotype A occurs approximately every 2-3 years, and those caused by serotype B - every 4-6 years. Serotype C does not cause epidemics, only sporadic outbreaks in children and debilitated people. In the form of epidemics, it occurs more often in the autumn winter period. Periodicity epidemics tied With frequent change antigenic structures virus during your stay his in natural conditions.

The entry gate for the influenza virus is the cells of the ciliated epithelium. upper respiratory tract - nose, trachea, bronchi. The virus replicates in these cells and leads to them destruction and death. This explained irritation top respiratory tract cough, sneezing, nasal congestion. Penetrating into the blood and causing viremia, virus renders direct, toxic action, emerging inform raise temperature, chills myalgia, head pain. Except Togo, virus raises vascular permeability, causes development stasis and plasma hemorrhage.

Traditional way warnings diseases influenza is vaccination. Suggested vaccine for prevention influenza in form alive, killed (inactivated), subunit vaccine. Vaccination is especially indicated in groups risk - children, the elderly, patients with chronic heart and lung

diseases, and also doctors. Usually carried out, when epidemiological forecast indicates the expediency of mass events (usually in the middle of autumn). Possible and second injection in middle winters.

For fast diagnostics influenza use "express method" detection influenza virus using fluorescent antibodies. The test material is taken from nose in first days illness. cooked from him smears handle specific flu-like fluorescent sera. formed the antigen-antibody complex glows brightly in the nucleus and cytoplasm of cylindrical cells epithelium and is clearly visible under a fluorescent microscope. The answer can be obtained through 2-3 h.

Serological research help retrospective diagnostics flu. Examine paired blood sera taken from patients in the acute period of the disease (up to the 5th days from start disease) and in period convalescence With interval 12-14 days. Most demonstrative in serological diagnostics are reaction binding complement (RSC) with influenza antigens and hemagglutination inhibition reaction (RTGA). Diagnostic counts growth titra antibodies in four times and more.

Hepatitis B is a viral disease caused by the hepatitis B virus.(in the specialized literature it may be referred to as "HBV virus", HBV or HBV) from the family hepadnaviruses.

The virus is extremely resistant to various physical and chemical factors: low and high temperatures (in volume including boiling), repeated freezing and thawing, prolonged exposure to an acidic environment. In the external environment at room temperature, the hepatitis B virus can survive up to several weeks: even in a dried and imperceptible stain of blood, on a razor blade, the end needles. In blood serum at a temperature of +30°C, the infectivity of the virus remains flow 6 months at -20°C near fifteen years. Inactivated at autoclaving infor 30 minutes, dry heat sterilization at 160°C for 60 minutes, warming up at 60°C in flow 10 hours.

Mechanism transmission infections — parenteral. Infection going on natural (sexual, vertical, domestic) and artificial (parenteral) ways. The virus is present in the blood and various biological fluids - saliva, urine, semen, vaginal secret, menstrual blood and others contagiousness (infectiousness) virus hepatitis A B exceeds HIV contagiousness in 100 once.

Greatest meaning before everywhere had exactly parenteral path — infection during medical and diagnostic manipulations, accompanied by a violation integrity skin or mucous cover through medical, dental, manicure and other tools, blood transfusions and her drugs.

Pathogenesis. The most significant pathogenetic factor in viral hepatitis B is death of infected hepatocytes due to attack by their own immune agents. Massive death of hepatocytes leads to impaired liver function, primarily detox, in lesser degree - synthetic.

Incubation period (time With moment infections before appearance symptoms) hepatitis B averages 12 weeks, but can range from 2 to 6 months. The infectious process begins from the moment the virus enters the bloodstream. After viruses entering the liver through the blood, there is a latent phase of reproduction and accumulation viral particles. At achieving certain concentration virus in liver acute hepatitis B develops. Sometimes acute hepatitis goes away for a person practicallyimperceptibly, and is found by chance, sometimes leaks in light anicteric form

— appears only malaise and decline performance. Some researchers believe what asymptomatic flow, anicteric the form and

"icteric" hepatitis are equal in the number of affected persons of the group. That is identified diagnosed cases of acute hepatitis B account for only one third all cases acute hepatitis.

Vaccination. Mandatory vaccination. FROM recent time vaccination against hepatitis B was included in the mandatory immunization schedule. Newborns are the most sensitive to hepatitis b virus - in infection at this age, the risk acquisition of a chronic form of hepatitis B is 100%. At the same time, immunity created by the vaccine during this period of life, the most persistent. Recommended to vaccinate newborn still in the maternity hospital, then 1 month after the first vaccination, and through 6 months after first vaccinations (So called scheme 0-1-6). At pass the next injection should be remembered about the allowable intervals - 0-1 (4) -6 (4-18) months. However if were missed admissible intervals, necessary continue vaccination according to the scheme, as if there was no pass. If the vaccination was standard schedule, revaccination is usually not required because immunity preserved on lesser measure in flow fifteen years. For definitions, how much for a long time preserved immunity in flow life, necessary further research - after all Vaccination has been introduced relatively recently. Only after all vaccination course, almost 100% immunity is achieved. About 5% of the general population

responds to vaccination, in these cases, other types of vaccines against hepatitis A AT.

laboratory diagnostics GV - founded on the identifying specific for GV antigens and corresponding antibodies in the blood, as well as viral nucleic acids, main from which are:

HB sAg - anti-HB s

anti-HBc class Ig M and IgG

HBe Ag - anti-HWe

DNA HBV

Most wide in diagnostics GV used definition HBsAg. The antigen is detected in both acute and chronic disease (however, acute infection is usually confirmed by the presence of high titers of anti-HBc IgM). With acute GV surface antigen virus is found through 3-5 weeks from momentinfection, that is, long before the appearance of clinical signs of the disease and in these cases is the only serological marker. HBsAg is constantly detected in the preicteric and icteric periods of the disease. HBsAg persistence for 6 months or more indicates a protracted or chronic course of the disease, and allows suppose chronic carriage virus. Elimination HBsAg and appearance antibodies to him is indispensable condition convalescence. Serological markers replication HBV are - anti-HBs class IgM, HBeAg, DNA and DNA- polymerase, which are found at sharp GV With first days clinical manifestations and can be detected during exacerbation of chronic hepatitis B. Serological HBV replication markers are determined both for general diagnostic purposes and for evaluation efficiency applied therapy.

Virus hepatitis A D (HDV) first was discovered in 1977 year. He not belongs neitherto one of the known families of viruses. HDV is a spherical particle, in the center of which is a spherical antigen (ND-Ag) containing RNA. Outdoor shell particles formed superficial antigen virus hepatitis A AT - HBs antigen (HBsAg). HDV cannot exist without HB virus replication, so it called a parasite virus, or a defective virus. The hepatitis B virus performsthis helper function, then there is role assistant for breeding NDV. Therefore, NDV

- infection always occurs together with HBV infection. NDV is located mainly in nuclei of hepatocytes and occasionally in cytoplasm.

Epidemiology. HDV infection is widespread. Circulation intensity NDV in different regions of the world varies significantly, but in general repeats the situation at HBV, although and not absolutely exactly. At acute hepatitis antibodies to NDV stand outin different regions in 2-7% of patients, and in chronic hepatitis - in 9-50% of patients. On the territory of the former USSR, among “healthy” carriers of HBsAg, the highest frequency (10-20%) detection of antibodies to HDV detected in Moldova, Kazakhstan, Central Asia, Tuva, that is, in areas hyperendemic for HBV. In the European part of Russia, the frequency identifying antibodies to NDV is 1.2-5.5 %.

source infections are sick sharp and chronic IOP, virus carriers, as well as carriers of anti-NDV, since it is known that in individuals with anti-NDV at the same time, RNA-NDV can be detected. Broadcast NDV occurs in the same way as at HBV (parenteral, sexual by, from mothers fetus). To delta - infections susceptible individuals who have not had HBV (i.e., do not have anti-HBs), as well as carriers HB-virus (healthy carriers of HBsAg and patients with chronic HBV). delta infection arises both sporadically and form outbreaks.

Pathogenesis, clinic. Infectious process, conditioned NDV, appearsbefore Total appearance ND-Ag in blood. Delta - antihemia maybe to be short-term or lengthy in dependencies from Togo, how happened infection and whether there is HB-virus integration into the hepatocyte genome. Distinguish acute, protracted and chronic flow delta- infections. Character her currents limited duration HBs- antigenemia: on measure her exhaustion stops and synthesis NDV, and ends delta- dependent pathological process.

Delta- infection develops in form co-infections or superinfection. At co-infection, simultaneous infection of HBV + HDV occurs in persons who have not been ill before HBV infection (not having HBV infection markers prior to infection). In that case, acute HBV+HDV-hepatitis develops with the appearance of serological markers two acute infections at once. In coinfection, HBV replication is most often HBV + HD - hepatitis A usually happens sharp and ends recovery.

In case of superinfection with HDV - the infection is superimposed on the current HBV infection in healthy carriers of HBsAg, in convalescents of the main HBV, in patients with chronic HBV. At this develops clinic acute viral hepatitis A delta, accompanied appearance antibodies to delta antigen.

laboratory diagnostics hepatitis A D (DG) Virus hepatitis A D (IOP) - this is defective virus, containing single helix RNA, to whom for replication the help of the HB virus is needed for the synthesis of envelope proteins consisting of HBsAg, which is used to encapsulate the HDD genome. IOP does not belong to any of the known families of animal viruses, in terms of its properties, HDV is closest to viroids and plant satellite viruses. Laboratory diagnostics is carried out by detecting serological markers of IOP, including the presence of antigen, antibodies to him and IOP RNA. Detection of HDV antigen and HDR RNA in blood serum or tissue liver indicates the presence of active HD infection, however, it should be noted that these markers may not show up in serum sick fulminant GD. Marker active replication IOP also is anti-CHD class IgM. Serological markers of HD infection depend on how the virus was acquired - in form of co-infection with HBV (in most patients, the disease has an acute course and ends recovery) or superinfection at sick With chronic GW- infection (flows heavier, how coinfection - in ten% develops fulminant hepatitis). In case of superinfection in patients with chronic hepatitis B infection, serological the picture has the following characteristic features: - the HBsAg titer decreases by the time appearance antigen IOP in serum; - antigen IOP and RNA-HVD continue be determined in serum, since usually in most patients with HD superinfection (70-80%) develops chronic infection, in difference from cases coinfections; - high titers of antibodies (anti-VGD) of both the IgM and IgG classes are determined, which persist indefinite time. Serological markers virus DG determine method enzyme immunoassay and radioimmune analysis, a RNA-HVD - method polymerase chain reaction.

Hepatitis C is an anthroponotic viral disease with a parenteral mechanism. infection, most often flowing in form post-transfusion hepatitis A With predominance anicteric and prone to chronization.

Hepatitis C is called the "gentle killer" because of its ability to mask the true reason under the guise of a multitude others diseases.

Parenteral viral hepatitis C called RNA containing virus With the size of the virion is 30-60 nm, belonging to the family Flaviviridae. virus particles HCV are enveloped, found in blood in trace amounts, and are associated with low-density lipoproteins and antibodies to proteins of the hepatitis C virus. Viruses, isolated from complexes with lipoproteins and anti-HCV antibodies have a diameter 60-70 nm. At electron microscopic studying on the surfaces virion identified well-defined ledges height 6-8 nm.

source infections are sick With active hepatitis C and latent sick — carriers virus. HCV infection is infection With parenteral mechanism infections — through infected blood and her Components. infection Maybe at parenteral manipulation, in volume including in medical institutions, including rendering dental services, through injection equipment, at acupuncture, piercing, drawing tattoos, at rendering row services in hairdressing, but at genital contacts probability get sick hepatitis FROM much less, how hepatitis AT, and comes down to minimalindicators.

Laboratory diagnosis of hepatitis C (HC). Laboratory diagnosis of HS was solved using modern methods of molecular biology, given that in HS the virus is in an extremely low concentration and its antigens are not available for detection with help contemporary methods indication, efforts researchers concentrated on the detection of antibodies to various antigenic components of the virus, the detection of which can serve as an indicator of the presence of the virus. Proteins were used as antigens. encoded by the structural and non-structural zone of HCV RNA, obtained using recombinant technology or synthesis (polypeptides used in modern immunological methods - C22-3; C33s, C100-3, C200, NS5, S-1-1). laboratory diagnosis of HC is based on the detection of serological markers in HCV: antibodies to HC virus (anti-HCV, anti-HCV class IgM, IgG) by ELISA and RNA-HCV method PCR. To date, 4 generations of test systems have been developed to detect anti- HCV in enzyme immunoassay, but the first generation ELISA is not currently used due to for low sensitivity. HCV RNA is an indicator of active HCV replication and the earliest marker of infection, and can be detected by polymerase chain reaction as early as 1-2 weeks after infection, shortly before the increase serum transaminase levels. Anti-HCV is detected by 5-6 weeks after onset hepatitis in 80% of cases and by week 12 in 90% of individuals by enzyme immunoassay. When determining anti-HCV, in some cases a false positive is recorded reaction. To distinguish false positive samples from real samples containing antibodies to HCV developed additional tests - recombinant immunoblotting, definition spectrum anti-HCV proteins.

HIV — virus immunodeficiency human, defiant disease — HIV infection last stage which known how syndrome acquired immunodeficiency (AIDS) - in difference from congenital immunodeficiency.

Spreading HIV infections related, main the way With unprotected sexual contacts, using infected virus syringes, needles and others medical and paramedical instruments, transmission of the virus from an infected mother to child during childbirth or while breastfeeding. In developed countries Mandatory testing of donated blood has greatly reduced the possibility of transmission virus with her use.

HIV infects before Total cells immune systems (CD4+ T-lymphocytes, macrophages and dendritic cells) a also some other types cells. infected HIV CD4+ T-lymphocytes gradually die.

Virus immunodeficiency human refer to family retroviruses (Retroviridae), kind lentiviruses (Lentivirus). Name Lentivirus going on from Latin the words lente — slow. Such title reflects one from features viruses this groups, aexactly — slow and unequal speed development infectious process inmacroorganism. Lentiviruses also have a long incubation period.*Diagnostics.* Flow HIV infections characterized lengthy absence significant symptoms disease[81]. Diagnosis HIV infections put on the basislaboratory data: at identifying in blood antibodies to HIV. Antibodies to HIV in periodacute phase, how rule not discover. AT first 3 months after infections antibodies toHIV come to light at 96-97 % patients through 6 months — at the rest 2-3 %, a in more late terms - only 0.5-1% (source Centers for Disease Control and Prevention usa, 2009). AT stages AIDS register significant decline content antibodiesin blood. First weeks after infections present yourself "period seronegative window, when antibodies to HIV are not detected. Therefore negative an HIV test result during this period does not mean that the person is not infected HIV and not can infect others.

For the diagnosis of lesions of the oral mucosa in HIV-infected patients accepted working classification, approved in London, in September 1992 of the year. All defeat divided into 3 groups:

1 group - lesions clearly associated with HIV infection. This group includes the following nosological forms:

candidiasis (erythematous, pseudomembranous, hyperplastic, atrophic);hairy leukoplakia;
marginal gingivitis;
ulcerative necrotic gingivitis;
destructive periodontitis; sarcoma
Kaposi;
non-Hodgkin lymphoma.

2 group - lesions less clearly associated with HIV infection:bacterial
infections;
disease salivary glands; viral
infections; thrombocytopenic
purpura.

3 Group — defeat, which may to be at HIV infection but not related Withher.

Herpes (Greek ἕρπης - creeping, spreading skin disease) - viral disease With characteristic rash grouped bubbles on the skin and mucous shells.

Herpes simplex (Herpes simplex) - a group of crowded vesicles with a transparent contents on an inflamed base. Herpes is preceded by itching, burning of the skin, sometimes chills, malaise.

Shingles (Herpes zoster) - characterized by pain along the nerve, head pain. Through several days on the site skin on move nerve appear rashes in the form of grouped vesicles, first with a transparent, and later purulent bloody content. Are increasing lymphatic nodes, rises body temperature, the general condition is disturbed. Neuralgic pains can last up to several months.

Pathogenesis. Virus herpes transmitted immediate contact by, a also through household items. Airborne transmission is also possible. drip way. Herpes penetrates through mucous shells cavities mouth, top respiratory tract and genitals. Having overcome tissue barriers, the virus enters blood and lymph. Then gets into various domestic organs.

Virus penetrates in sensitive nervous graduation and embedded in genetic apparatus

nervous cells. After this delete virus from organism impossible, he will remain with the person for life. The immune system responds to penetration herpes development specific antibodies, blocking circulating in blood viral particles. Characteristically awakening infections in cold season, with colds, with hypovitaminosis. reproduction herpes in the cells of the epithelium of the skin and mucous membranes leads to the development of dystrophy andcell death.

According to research scientists Colombian University, herpes is stimulating factor for development disease Alzheimer's. Later these data were independently validated by researchers at the University of Manchester. Previously the same Group researchers under leadership Ruth Yitzhaki proved what virus simple herpes is found in the brains of almost 70% of patients with Alzheimer's disease. Except Togo, they confirmed what at infection virus culture cells brainthere is a significant increase in the level of beta-amyloid, from which plaques. In a recent study, scientists were able to find that 90% of plaques in brain patients With sickness Alzheimer's contain DNA simple herpes — HSV-1.

For diagnostics herpetic infections are used all laboratory reactions
— from cytological research before molecular biological methods.

Material for virus isolation for the purpose of diagnosing herpes infection maybe serve content herpetic bubbles, scrapings With horny shells andliquids from front cameras eyes, blood, saliva, urine, spinal liquid feces pieces fabrics brain, liver, kidney, spleen, lungs lymphatic nodes,taken on the bio- or autopsy.

Infectious material can for a long time keep at -70°C, then how at temperature -20°C he fast is inactivated. Virus containing fabrics may to be saved more 6 months at 4°C, if they are in fifty% solution glycerin.

There are a number of special methods for the detection of viral antigens, specific antibodies and virus-induced morphologically changed cells.

Most affordable and technically uncomplicated is cytological method, allowing to study morphological changes in cells infected with the virus herpes simplex. The effectiveness of the method depends on obtaining a sufficient amount cells for research. Availability intranuclear inclusions, characteristic for reproduction of the herpes virus serves as a confirmation of the diagnosis. It should be remembered that intranuclear inclusions are detected only after immediate fixation of smears scraping in absolute alcohol With subsequent coloration on Romanovsky-Giemsa. Morphological changes, induced virus simple herpes, can also detected in tissue sections of infected organs. characteristic of herpetic infections is: Availability multi-core cells, intranuclear inclusions and in some cases of hemorrhage. In the generalized form of the disease, multinuclear cells With eosinophilic inclusions find in zones necrotic fabrics various bodies (brain, liver, kidney, adrenal glands, epithelium bronchi and trachea).

Method immunofluorescence — is method express diagnostics herpetic infections and allows in flow 1-2 hours determine Availability herpesvirus antigens in clinical material (scraping With skin and mucous membranes, sections of biopsied organs). Identification of antigens of the simplex virus herpes maybe to be completed in various modifications method immunofluorescence
— straight, indirect, With application labeled complement.

Of the serological methods of identification, the most commonly used reaction binding complement (RSK), especially in micromodifications her staging. micromethods use and for identifying virus simple herpes in reactionsneutralization, passive hemagglutination and in others serological tests.Sensitivity listed methods are different.

AT the present time one from most sensitive methods diagnostics herpetic infections is method enzyme immunoassay analysis (IFA), allowing find, in dependencies from kind biological material, how virus-specific antigens, So and virus-specific antibodies class IgM, IgG.

TIMELINE

1. Definition original level knowledge ----------30 min.

2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15 min.

PRACTICAL OCCUPATION No. 2.

Topic: Infectious control in dentistry. Disinfection, pre-sterilization treatment and sterilization tools, materials, equipment. Antiseptics and disinfectants. Ways fence material for researches from an oral cavity (for microbiological researches). Modern methods clinical immunology and molecular genetics.

Educational goal:

1. Explore peculiarities fence researched material from cavities mouth for holdingvarious methods of microbiological diagnosis.
2. Explore major representatives of the resident microflora cavities mouth.

Plan lessons:

2. Features of the sampling of the test material from the oral cavity (oral fluid, dental plaque, content gingival groove, periodontal pocket, carious cavity, root channels and etc.).

Independent Work students:

3. Explore peculiarities fence researched material from cavities mouth.
4. Design protocols research.

EQUIPMENT

one. Preparations for asepsis and antiseptics.

INFORMATIONAL MATERIL ON THEME

4. sketch in protocol fence scheme researched material at complications cariesteeth and periodontitis.
5. Using the reference literature and the drawing "Microbiocenosis of the oral cavity",sketch representatives resident microflora cavities mouth at coloration on Gram.
6. results contribute in protocol.

Table. Fence researched material from content periodontal pocket

researched material	Description results microscopy	Picture

MICROBIOCENOSIS CAVITIES RTA

corynebacteria
lactobacilli
actinomycetes
Strept. mutans
peptostreptococci

spirochetes
lactobacilli

anaerovibrio
anaerobospirilla

Neisseria

leptotrichia
mushrooms candida
fusobacteria
bacteroids
veillonella

trichomonas
amoeba

bacteroids
fusobacteria
anaerobovibrio
anaerobospirilla
spirochetes

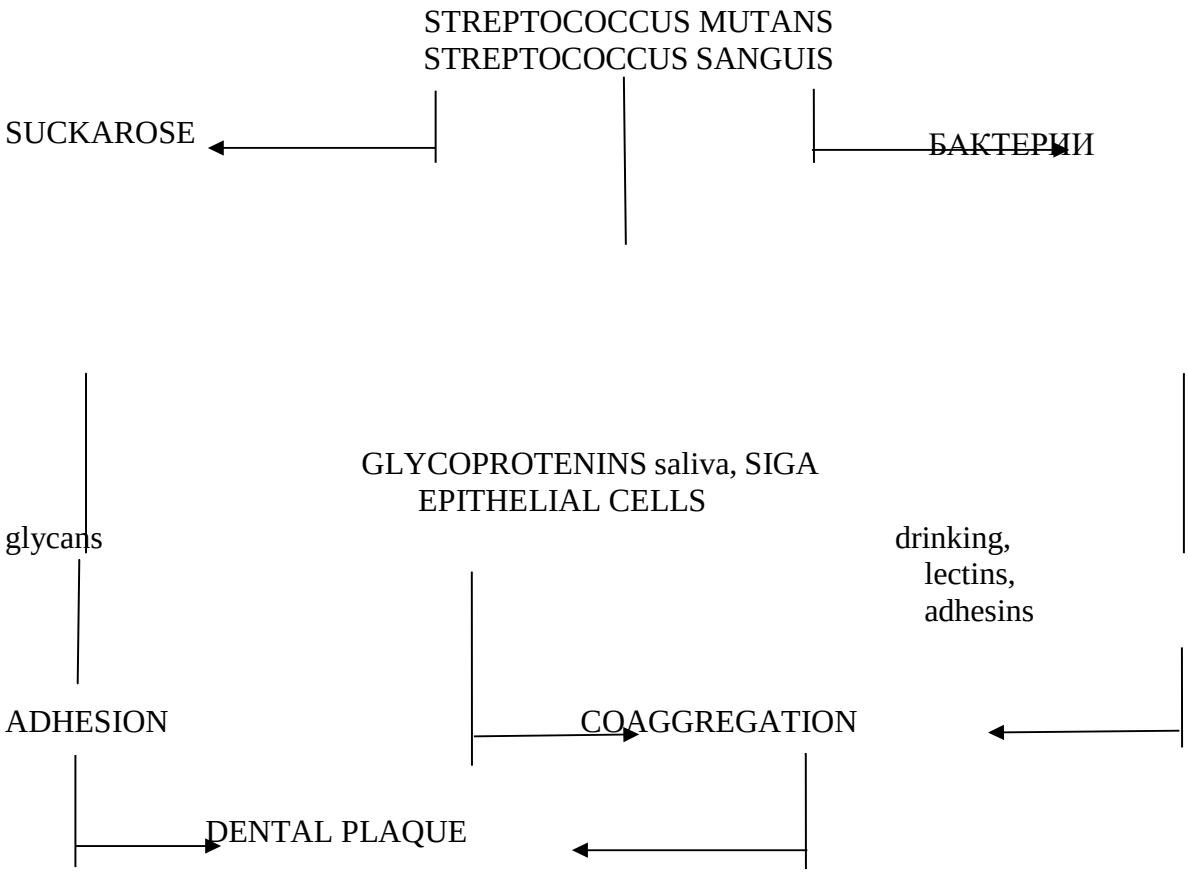
Designations:
5- dental plaque
6- microcracks and tubules enamel tooth7-
gingival groove
eight- gaps mucous shells cavities mouth

1-2. Smear from dental raid or scraping co mucous cook on the subjectglass. The material can be taken with a sterile spatula, trowel, match. The material taken from the interdental spaces or at the neck of the tooth is applied to the subject glass next to a drop of water and rub it dry, and then bring in a loop of water, gradually preparing a homogeneous suspension and evenly distributing it over the glass surface. Smear dried in air, fixed in a burner flame and stained by Gram. At microscopy under immersion study morphological peculiarities and attitude to Gram staining of representatives of the normal microflora of dental plaque biotopes and mucous language.
3. Decor protocol research.

Table. Fence researched material dental plaques

researched material	Description results microscopy	Picture

SCHEME FORMATIONS DENTAL PLAQUE (PLAQUES).



2. Microscopic examination of the demonstration clean smears cultures of bacteriaallocated from cavities mouth (lactobacilli, peptococci, bacteroids).
3. Decor protocol research.

Table. Fence researched material from horse channels.

researched material	Description results microscopy	Picture

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.

5. Control final level knowledge and exercise on the house ---------- 15 min.

PRACTICAL OCCUPATION No. 3.

Topic: Sterilization and disinfection. Ways sterilization and disinfection laboratory crockery and medical tools. Peculiarities sterilization and pre-sterilization processing dental tools, hog, tips turbines etc .

Educational goal:

1. Familiarize With modern methods sterilization and disinfection in dentistry.
2. Familiarize yourself with the list of modern disinfectants and antisepticsdrugs.
3. Explore regulations precautions from infections infectious diseases on thereception at dentist.

Plan lessons:

1.Features of microscopic, bacteriological and serological methodsresearch at diagnostics dental diseases.
5. Modern methods sterilization and disinfection in dentistry (ultrasound, UVgamma rays, laser)
6. Rules precautions from infections infectious diseases on the admission atdentist.
7. Instructions and normative the documents on disinfection and sterilization in dentistry.

Independent Work students:

6. Explore instructions and normative the documents on disinfection and sterilization in dentistry.
7. work out methodical recommendations to occupation and fill table.

8. Table

9. **Characteristic methods sterilization in dentistry**

10. ten.

	Metho d	Apparatus	Mode	Reliability and testimony	Objects sterilization
one.	Ferry under pressure				
2.	Dry heat				

3.	Gas sterilization.				
four.	Chemical sterilization.				
5.	Ultrasound.				
b.	uv and gamma rays				
7.	laser				

EQUIPMENT

1. Preparations for asepsis and antiseptics.
2. Equipment for disinfection and sterilization materials (demonstration).

INFORMATIONAL MATERIL ON THEME

In dentistry, more than in other areas of medicine, strict compliance rules asepsis and antiseptics, So how any dental intervention is performed on infected tissues. Not only the removal of carious tooth or treatment root channel, but and simple inspection cavities mouth sick connectedwith infection of the instruments used for these purposes, in order to exclude the transfer microbes from one sick in cavity mouth another a also prevent infection of healthy tissues, it is permissible to work only with a sterile instrument. Security sterile dressing material and tool - a task sisters,which must be supervised by a doctor.

I. PROCESSING OF THE DENTAL ROOMOFFICE

1.Track in office per temperature and humidity use air filters.

2.Before receiving patients, it is necessary to carry out wet cleaning using various disinfectants. Wipe 2-r. napkin every 15 minutes disinfectant solutions (3% chloramine, 6% hydrogen peroxide, 70 degrees alcohol and etc.) surfaces all items With purpose destruction vegetative forms bacteria.

3.Then necessary turn on ultraviolet installation for destruction airborne and surface bacteria. (Calculation of a germicidal lamp at 2.5 W on the 1 cubic meters in flow 1 hour)

II. DISINFECTION, STERILIZATION IN DENTALOFFICE

5. All products that do not have contact with the wound must be disinfected. surface, blood or injections (solutions of 6% peroxides hydrogen, 3% chloramine, 70 ^{0} alcohol, etc.)

6. Sterilization - this is complete desolvation material. Sterilization must all objects in contact with the wound surface, in contact with blood and individual kinds medical tools, which in process work touch co mucous membrane and may call her damage.

7. Before sterilization, it is necessary to soak the entire tool, burs, a then clear them from protein, fatty, mechanical pollution and medicinal drugs. cleaning must produced jet, rotary

methods, ruffing or With application ultrasonic baths, in which put 6 % solution peroxides hydrogen and detergent substance (powder "Lotus", "Progress" and etc.).

8. AT dependencies from sterilized material can use thermal, chemical and gas methods of sterilization. Preference should be given to thermalmethods like more reliable.

However, products made of rubber, polymers, optical equipment, some tools, devices heart-lungs, artificial bud not withstand thermal processing.

9. Sterilization ferry under pressure carried out in autoclaves. Mode sterilization allows you to destroy not only bacteria, spores, but also viruses such as hepatitis B virus (serum hepatitis) and HIV. Pressure 2 atm. (temperature 13 2 deg.) within 1 hour. Sterilization is carried out in sterilization boxes, bixes, wet-strength paper bags with markings. This method is recommended for products corrosion resistant metal, glass syringes, rubber, textile materials, some polymers.

10.Some tool (especially cutting) recommended sterilize in glass-pearl sterilizer at temperature 240 deg. in flow 5-10 seconds.

11.Dry heat sterilization in dry heat ovens is carried out at a temperature 180 deg. in flow 150 minutes (2.5 hours). Duration impact also allows destroy hepatitis E and HIV viruses. Sterilization is subjected to dry products in packaging from paper (term storage twenty days). Sterilize can and without packaging, but then products must be used directly after sterilization.

12.The chemical method of sterilization consists in the fact that products are immersed in solution 6% hydrogen peroxide on 6 hours or

into a chamber with vapors of 40% formaldehyde in ethyl alcohol for several hours, which depends from the material to be sterilized.

9. Recently, in connection with the advent of new equipment, it has become more widely use the gas sterilization method. It is carried out in special chambers or tabletop gas sterilizers containing ethylene oxide or a mixture of ethylene and methyl bromide, Sterilization takes place at a temperature of 35 degrees. up to 42 degrees, duringseveral hours or days in special packages With marking:

a) if contact with blood, tissues was less than 30 minutes, then metal products sterilize in for 4 hours, products rubber, plastics - 24 hours.

b) if contact with blood, tissues was more than 30 minutes, then metal products sterilized within 24 hours, products made of rubber, plastics - one week, the device lungs-heart-kidney in flow 2nd weeks.

Such long sterilization tied With prevention HIV and hepatitis A B.

10. FROM purpose prevention serum hepatitis A B and HIV recommended use items disposable use (syringes, injection needles, systems for transfusions blood and etc.)

Safety instructions for working with biomaterial, potentiallyinfected HIV

I. General provisions.

AIDS - disease co deadly outcome, developing in result dysfunctions of the immune system. The incubation period of the disease is 5-10 years. There are no cases of spontaneous recovery or cure from AIDS. Pathogens - T-lymphotropic retroviruses HTLV-3 (HIV-1) and HTLV-4 (HIV-2). Transmission routes - With blood (cells, serum), sexual, from mother to children with breast milk. Viruses unstable - die after a 30-minute exposure to a 20% solution of ethyl alcohol. Therefore, all measures provided to prevent infection with hepatitis viruses, sufficient to protect against infection with AIDS viruses. When dealing with infectious material necessary observe three major regulations: change robe, work in disposable gloves and more wash arms.

II. Rules work.

1. Work in the department should be in specially designed for this bathrobe. Keep them necessary in closet at entrance in branch, put on before work, take off on exit from departments.

2. All furniture and equipment in department must have plastic or metallic coating, easily amenable disinfection. On the tables must standcontainers With disinfectant solution (70% solution ethyl alcohol).

3. Tubes with biomaterial must be labeled carefully closed (traffic jams, parafilm, patch) and be delivered in unbreakable containers, easilyexposed disinfection.

4. All work, related With acceptance biomaterial and staging method, must be done with disposable gloves. All while working hand injuries must be closed (adhesive plaster, fingertip).

5. centrifugation test tubes With biosamples necessary conduct in centrifuge having individual lids on everyone glass.

6. At work With biomaterial should enjoy means, protective eyes from hits drops liquids (protective glass, shield, glasses).

7. All disposable materials, contact with the studied biomaterial (traffic jams, tips, adhesive paper, gloves) necessary straightaway same after use dump in special capacity With dez. solution (70% ethyl alcohol).

8. At the end of work, wipe all work surfaces (tables, equipment) swab dipped in disinfectant. solution. All used in production are disposable materials test test tube, gloves, traffic jams, plateau etc.) soak.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15 min.

PRACTICAL OCCUPATION No. four.

Topic: Microbiocenosis of the oral cavity. Resident microflora of various biotopes oral cavity. Plaque and its study in assessing the hygienic condition oral cavities.

Educational goal:

3. Explore major representatives resident microflora cavities mouth.
4. Explore microflora dental raid.

Plan lessons:

5. Symbiosis, stages symbiosis.
6. Cavity mouth how ecological niche organism.
7. Main representatives resident microflora cavities mouth, them properties.
8. dental plaque. Mechanism her formations. Localization.

Independent Work students:

3. sketch in form scheme morphology main residents cavities mouth:
    1) anaerobic gram-positive (Peptostreptococcus, actinomycetes, propion- and eubacteria) and gram-negative (veillonella, bacteroids, fusobacteria, tortuous forms);
    2) aerobic and facultative anaerobic gram-positive (streptococci, staphylococci, Korine- and lactobacilli) and gram- negative (neisseria, pseudomonas),
4. Decor protocol research.

EQUIPMENT

1. Preparations for asepsis and antiseptics.
2. Equipment for disinfection and sterilization materials (demonstration).

INFORMATIONAL MATERIL ON THEME

Species compound microbial flora cavities mouth in norm enough constant. TogetherWith topics amount microbes in cavities mouth susceptible significant fluctuations. AT the present time described several hundreds species microorganisms, constituents normal microflora of the oral cavity. It contains bacteria, viruses, fungi and protozoa.

Quantity microbial flora depends from hygienic content cavities mouth, smoking promotes reproduction microorganisms, causes chronic inflammation mucous shells. Solid food more affects on the decrease the number of microbes, because chewing promotes mechanical cleaning of the oral cavity from microorganisms. Disorder salivation, chewing and swallowing always leads to rising quantity microorganisms in oral cavities.

Availability carious cavities, gingival pockets, poorly stocked dental fixed prostheses and others condition enough high frequency formation foci chronic infections With subsequent allergization organism and high degree risk development general autoimmune diseases.

Microflora cavities mouth newborn presented in mostly milky - sour chopsticks, non-hemolytic streptococci and non-pathogenic staphylococci. Her enough fast (in flow weeks) replace microorganisms, characteristic for cavities mouth adult person.

Main inhabitants cavities mouth adult human are bacteria, predominantly (3/4 all microbial species) anaerobic type breathing. Among them meet various cocci, sticks, tortuous forms.

Despite the wide variety of microorganisms in the oral cavity, quantitatively in it is dominated by microbes of three groups: about half are facultative and obligate anaerobic streptococci, and the other half consists of veillonella (less than 1/4) and diphtheroids (less than 1/4). The remaining numerous groups of bacteria are staphylococci, lactobacilli, flagella, spirochetes, leptospira, fusobacteria, bacteroids, neisseria, hemophilus, mycoplasmas, yeast, protozoa - present yourself small populations on quantity, but equal groups on formation associations residents.

Table

Microflora cavities mouth in norm.

Microorganisms	**AT saliva**		**AT dental gums**
	frequency	**amount in 1**	**pockets (frequency**
Residential flora **1. Aerobesand**			
Strentococcus	100	10^7	100
Streptococcus mitis	100	10^6-10^8	100
Saprophytic e Neisseria	100	10^5-10^7	++
Lactobacillus	90	10^3-10^4	+
Staphylococci	80	10^3-10^4	-n-
Diphtheroids	80	Not def.	+
Hemophiles	60	Not def.	0
pneumococci	60	Not def.	Not defined
and others	thirty	10^2-10^4	++
Saprophytic e	++	Not def.	++
Tetracocci	++	Not def.	++

yeast-like s mushrooms	fifty	10^2-10^3	+
Mycoplasmas	fifty	10^2-10^3	+
Protozoa: Entamoesa gingivalis	0	0	45
Trichomonas	0	0	25
II. Obligatory anaerobes	100	10^6-10^8	100
Anaerobic streptococcus			
(peptostrep tococci)	100	He def.	100
Bacteroids	100	He def.	100

Fusobacter ai	75	ioMo4	100
filiform bacteria	100	102-104	100
Actinomyce you and anaerobic diphtheroids	100	He def.	++
Spirilla and vibrios	++	He def.	.one. ++
Spirochetes (saprophytic	±	He def.	100
Group B. Fickle	fifteen	I0-102	. 0
Klebsbrieila	2	10-I02	+
Esclierichia	3	10-102	0
Aerobacter	±	He def.	0
Pseudomona s	±	He def.	0
Proteus	±	He def.	0
Alcaligenes	±	He def.	0
bacilli			
II. obligatee anaerobes			
Clostridia	+	He def.	0
Cloatridium putrificun	±	He def.	0
Clostridium perfringens	+	He def.	0

Ob about hn a hen and e: ++ ha With t about, + ne about h enb ha With t about, ± rarely, 0 not discovered.

To about l and h e With t in about m and to R about b about in in P about l about With t and R t a n e about d and n a to about in about in R a h n s X e G about uh to about l about G and h e With to and X n and sh a X: With l and h and With t about th about b about l about h to e, h about n e de With n e in about G about and e l about b to a, P R about t about to a X With l Yu n n s X and e l e h, With l Yu n e and R about t about in about th and and d to about With t and, h at b n about th b l Ish to e.

Ta to, n a P R and m e R, With about d e R and a n and e b a to t e R and al b n s X to l e t about to in With l Yu n e (R about t about in about th and and d to about With t and) With about With t a in l I et about t 50 m l n. d about 5 m l R d., P R and h e m b about l b sh and n With t in about b a to t e R and th P about P a d a et in With l Yu n at With about With P and n to and I h s to a. AT h at b n about m n a l e t e (b l Ish to e) m and to R about b about in h n a h and t e l b n about b about l b sh e: about t one 00 d about one 000 m l R d. in G R a m m e mate R and a l a. FROM a m at Yu b about l b sh at Yu G R at P P at With R e d and in With t R e h a Yu s ch and X With I in P about l about With t and R t a b a to t e R and th With about With t a in l I Yu t to about to to and. (T a b l and c a 2).

T a b l a n d c a .

O With n about in n s e G R at P P s R e h and d e n t n about th f l about R s P about l about With t and R t a P about m about R f about l about G and and and

t and P at d s X a n and I .

Coloring on Gramu	Morphology	Food kind
one GROUP: FROM anaerobic type breathing (obligate anaerobes)		
Gram-negative	cocci	VEILLONELLA
	sticks	BACTEROIDES PORPHYROMONAS PREVOTELLA FUSOBACTERIUM LEPTOTRICHIA
Gram positive	cocci	PEPTOSTREPTOCOCCUS PEPTOCOCCUS
	sticks indisputable	LACTOBACTERIUM BIFIDOBACTERJUM EUBACTERIUM PROPIONIBACTERIUM ACTINOMYCES
	sticks spore-forming	CLOSTRIDIUM
2 GROUP: FROM aerobic and mixed type breathing (aerobes andoptional anaerobes)		
Gram-negative	cocci	NELSSERIA
	sticks	PSEUDOMONAS BORDETELLA EIKENELLA
Gram positive	cocci	STREPTOCOCCUS STAPHYLOCOCCUS
	sticks indisputable	CORINEBACTERIUM NOCARD1A, ROTHIA
	sticks spore-forming	BACILLUS

Staphylococci. Division Firmicutes, family Micrococcaceae, genus Staphylococcus. AT genus Staphylococcus on classification Baird— Parker are included 3 type: S. aureus, S. epidermidis and S. saprophyticus. Recently proposed other classifications include more species of staphylococci, but they are used so far only in scientific research.

All types of staphylococci are rounded cells with a diameter of 0.5-1 μm. In a smear, they are usually arranged in asymmetrical clusters (“clusters of grapes”), but there are single cells, pairs of cells. Gram-positive. Dispute does not form flagella not have. At some strains can discover capsule. Can form L-shapes. The cell wall contains a large amount of peptidoglycan, related With him teichoids acids, protein BUT.

Staphylococci grow well on simple media (pH 7.0-7.5); optional anaerobes. On dense media they form smooth round convex colonies with different pigment. The pigment has no taxonomic value. Can grow on agar with juice content (8-10 %) NaCl. produce saccharolytic and proteolytic enzymes. Staphylococci produce hemolysins, fibrinolysin, phosphatase p-lactamase, bacteriocinins, enterotoxins, coagulase dna-ase, leukocidins, lecitovitellase and others

Staphylococci very plastic: fast develop sustainability to antibacterial drugs. Substantial role in this play plasmids, transmitted With help transducing phages from one cells to another. R- plasmids determine sustainability to alone or several antibiotics, in volume including and per check extracellular products p-lactamase — enzyme, destructive penicillin, tearing his p-lactam ring.

Pathogen staphylococcal infections happens more often S. aureus, somewhat less often - S. epidermidis, very rarely - S.saprophyticus. Staphylococci are representatives normal microflora human body, that's why microbiological diagnostics staphylococcal infections not maybe confine highlighting and identification pathogens; necessary quantitative methods research, t. e. definition numbers microorganisms in sample.

Staphylococci in the oral cavity of a healthy person are found on average in 30% cases. In plaque on the gums of healthy people are present in mostly Stapf. epidermidis. Much more often pathogenic staphylococci are localized on the mucous pharynx and nose, causing the so-called "healthy bacteriocarrier". Possessing enzymatic activity, staphylococci take part in the breakdown of residuesfood in the mouth. Such permanent carriers of pathogenic staphylococcus are source airborne infections. Pathogenic staphylococci, encountered on the nasopharyngeal mucosa and in the oral cavity are a common cause of autoinfection, causing various purulent-inflammatory processes cavities mouth.

Treatment staphylococcal infections usually carry out antibiotics and sulfanilamide drugs. AT recent years from sick often allocate staphylococci resistant to most chemotherapy drugs. Such cases, antitoxic anti-staphylococcal plasma is used for treatment or immunoglobulin obtained from the blood of immunized donors staphylococcal toxoid. For active immunization (planned surgical sick, pregnant women women) maybe to be used adsorbed staphylococcal toxoid.

Streptococci. Department firmicutes, family streptococcaceae, genus Streptococcus. AT genus Streptococcus are included more twenty species, among which there is representatives normal microflora human body and cavities mouth, a also pathogens heavy infectious epidemic diseases person.

streptococci — small (less one μm) spherical cells, located chains or in pairs, Gram-positive dispute not form, motionless. Majority strains streptococci form capsule, consisting from hyaluronic acids. Cellular wall contains squirrels (M-, T- and R antigens), carbohydrates (group specific) and peptidoglycans. Easily go over in L-shapes.

Genetic exchange available per check transformation and transduction, but not conjugation. Sustainability to antibiotics produced slowly.

streptococci groups BUT develop more twenty extracellular substances possessing antigenic activity. Greatest meaning in pathogenesis streptococcal infections have:

- streptokinase (fibrinolysin) — proteolytic enzyme, splitting fibrin and other proteins;
- DNAase — enzyme, depolymerizing DNA. Mixture DNAases and fibrinolysin is able to liquefy exudates, lyse venous thrombi, so maybe to be used for removal pus and necrotic fabrics from wounds;
- hyaluronidase — enzyme aggression destructive hyaluro-new acid, incoming in compound connective fabrics ("factor permeability");
- erythrogenin — toxin, produced p-hemolyticstreptococci groups BUT, able call scarlet fever. stands out only lysogenic cultures.

Standardized diluted erythrogenin use at staging intradermal test (Dick test) for identifying sensitivity to this toxin (susceptibility to scarlet fever).

streptococci are main inhabitants cavities mouth. IN 1 ml saliva contains up to 10^8 - 10^9 streptococci. However, in the samples their saliva at about 2 times more than in plaque or gingival groove material. most significant group streptococci cavities mouth should count microaerophilic a- hemolytic ("green") streptococci and j - non-hemolytic forms. Should Mark, what from 40 - 90 % strains kind milled may to be B-hemolytic, which take an active part in the processes leading to lesions of solid tissues of the tooth and periodontium. This group includes Streptococcus mutants, S. sanguis, S. mitis, S. salivarium. They are differ from each other in their ability to ferment carbohydrates and form hydrogen peroxide.

Shift pH in sour side leads to decalcification dental enamel. Especially should emphasize high capabilities microaerophilic streptococci to aggregation With others bacteria which shown, in in particular in respect actinomycetes, fusobacteria, lactobacilli. All this contributes to the detection of these species in the composition associations of pathogens in various purulent-inflammatory processes in the maxillo- facial area. But their role in the development of caries is especially significant. Leading position in This plan is occupied by two species that actively produce milk and milk from food carbohydrates. other acids on the enamel -S. mutans and S. sanguis.

All types of streptococci grow poorly and die on simple nutrient media, because in the process of growth and reproduction, streptococci secrete a lot of hydrogen peroxide, which has a detrimental effect on them, tk. they do not produce catalase. For creating optimal conditions growth, blood is usually added to the nutrient medium, in which contained catalase, destructive peroxide hydrogen. On the blood environments streptococci grow well under aerobic conditions, while some of them form on blood agar colonies surrounded by a zone of complete hemolysis (these are hemolytic (B) streptococci), other surrounded zone greenish colors (green streptococci), at third (y) hemolysis missing (non-hemolytic streptococci).

streptococci allocate exotoxin and enzymes aggression. In external environment less resistant, how staphylococci. Majority sensitive to penicillin and others antibiotics. By antigenic structure all streptococci divide on the 17 serological groups (A,B,C,D, ... and before S), bowl others in cavities mouth are found streptococci groups BUT, FROM, D, F, g, H and O.

Peptostreptococci - Gr + obligate anaerobic cocci, which include two kind - Peptostreptococcus and Peptococcus. Wide presented in all niches cavities mouth. More often Total peptococci found in association with fusobacteria and spirochetes at caries, pulpitis, periodontitis, abscesses maxillofacial areas.

Veillonelles - this is obligate anaerobic, Gr -, small cocco -bacteria, motionless, dispute not form. Are permanent inhabitants cavities mouth human and animals. Isolated colonies on the lactate agar have 1-3 mm in diameter, smooth, convex, lenticular, diamond-shaped or cardiac forms, yellow-white, soft in consistency. Representatives are found in the oral cavity two species of veillonella (V. parvula, V. alcalescens), which inhabit the mucous membrane cavities mouth, palate, are dominant in saliva and ducts salivary glands. Good ferment acetic, pyruvic and dairy acid, neutralizing sour products metabolism others bacteria, this is allows consider veillonella how the most important factor, resistance to caries teeth. pathogenic role veillonella not proven.

diphtheroids , or corynebacterium, present yourself group bacteria, quantitatively comparable With veillonella.

it polymorphic gram positive sticks, located orderly ("palisade" or groups) in a smear from a pure culture. Some types of microbes able form inclusion - grain volutin.

Classification diphtheroids cavities mouth before present time remains undeveloped. At research material diphtheroids often difficult differentiate from actinomycetes and propionibacteria. optional anaerobic kinds diphtheroids constitute approximately 13% of the number residents, isolated from the back of the tongue, 15% from the gingival groove and 24% from dental plaque. Representatives of diphtheroids with an obligate anaerobic type of respiration make up in these materials respectively eight, twenty and eighteen%.

Diphtheroids play important role in cavities mouth as a stabilizing factor oral microbiocenosis, So how synthesize vitamins, in in particular vitamin TO, being stimulant growth anaerobic bacteria. Reducing in process breathing molecular oxygen, they actively promote development obligate anaerobic flora in aerobic conditions.

Shown powerful immunomodulatory activity antigens diphtheroids (corynebacteria) on the organism human, what used at treatment immunodeficiencies. Together With topics at corynebacteria discovered some enzymes aggression and toxic polymers, they often are found in associations With pathogens purulent inflammation.

Lactobacillus constantly are in cavities mouth, motionless, dispute and capsules do not form, Gr + are characterized by high polymorphism. Grow up on electives nutritional environments contain such factors growth, how vitamins and some amino acids. grow up in form small, colorless, compacted colonies. Possess rather low adhesive properties to the mucosal epithelium and especially to enamel tooth, but presented in all niches cavities mouth. Stormy

multiply at admission in cavity mouth carbohydrate food and plentifully produce dairy and other acids, which allows them to be considered as a cariogenic factor. Together with topics lactobacilli play the most important stimulating role at formation microbial associations cavities mouth, So how synthesize vitamins groups AT and TO, necessary for development others bacteria and organism.

In view of education big quantity dairy acids in process vital activity lactobacilli, they detain growth others microbes: staphylococcus, intestinal sticks, typhoid and dysentery sticks. Antagonistic properties of lactic acid bacteria in relation to a number of putrefactive microbes were noticed yet I.I. Mechnikov, which the proposed use curdled milk made from milk fermented with lactic acid sticks. Before 90% of the lactobacilli living in the oral cavity belong to the species Lactobacterium casei, Lactobacterium fermenti.

actinomycetes - presented small Gr + chopsticks, having trend to education intertwined and branching threads or shorter chains. Actinomycetes are located on the mucous membrane of the mouth, make up the stroma of the dental stone and are part of dental plaque. Along with this, they are contained in carious cavities of teeth, in pathological gingival pockets, in ducts salivary glands.

Representatives of this family can take part in the formation of dental plaques and in development caries teeth, a also diseases periodontium. AT cavities mouth there are favorite places of penetration of actinomycetes into the depths of tissues - inflamed gums near the wisdom tooth or near the destroyed roots of the teeth, pathological gingival pockets at periodontal disease, root channels teeth With dead pulp tonsils.

For occurrence diseases not enough only introduction actinomycete deep into fabrics, certain role plays and condition protective strength, downgrade resistance organism to infections.

Bacteroides - represent a group of coccoid, ovoid or polymorphic rod-shaped Gr - bacteria. FROM 1990 of the year divided on the three kind: Porpluiroman (representative - P. Gingivalis inhabit gingival groove, dental plaque), Prevotella (the most important species - P. Melaninogenica inhabits pockets of the mucous membrane, fissures of the tooth, gingival groove), Bacteroides (representative - AT. Fragilis meets in folds mucous at the base teeth, but more typical for intestines. For growth on nutritional environments this microorganisms needed hemotin and vitamin TO. On the bloody agar AT. melaninogenicus shapes black colonies. Availability proteolytic. enzymes in bacteroids (collagenase, hyaluronidase, heparinase, Jg A -; Jg W-; Jg M - protease) It has big pathogenetic meaning in disease development periodontal.

Fusobacteria - elongated Gr - sticks, more often With pointed ends, often formative chains and threads. inhabit how mucosa mouth, So and dental plaque.

Fusobacteria produce powerful histolytic enzymes - hyaluronidase, lecithinase, have endotoxin. Along With bacteroids and peptococci considered main pathogens diverse purulently - inflammatory processes in cavities mouth, including ulceratively -necrotic fasciitis.

Neisseria - genus Neisseria - Gr - diplococci, detectable in various niches cavities mouth, especially on the surfaces which constantly touch With air - back language, soft sky, enamel teeth. pathogenic role them not proven.

Yeast-like mushrooms in cavities mouth healthy of people meet in 40 - 50% of cases. Candida are oval or elongated cell size 7 - 10 micron, often bud off new cell. Aerobe grow up on the environment Saburo, containing yeast extract and maltose, where convex colonies of opaque colors.

The most common species found in the oral cavity are: Candida albicans, Candida tropicalis, Candida crusel. Pathogenic properties are most pronounced in C. aldicans. Mushrooms cause a general disease of the body - candidomycosis or local damage to the cavity mouth - "milkmaid".

Spirochetes - inhabit the oral cavity from the moment of eruption of milk teeth in a child and from that time become permanent inhabitants oral cavity. Gr -, mobile, strict anaerobes, grow on media containing serum, ascitic liquid With adding fresh pieces various bodies, on the environments form turbidity in the form of a cloud. High proteolytic activity, liquefy gelatin, egg protein, folded serum form indole, hydrogen sulfide, ammonia.

They are easiest to detect in the dark field of view with microscopy of native drug.

Spirochetes cause pathological processes in the oral cavity with significant reproduction all anaerobic microorganisms.

Protozoa cavities mouth - meet at fifty % healthy of people, predominantly in dental on the fly, crypts tonsils (Entamoeba gigivalis). They multiply with unhygienic maintenance of the oral cavity. They are found in pus from gum pockets in severe alveolar pyorrhea D - 20 - 30 nm. aerobes, mobile, visible better in native unpainted preparation (crushed a drop). Grow on blood or serum agar, drenched in a layer of Ringer's liquid and with adding solution tryptophan (1 : 10000).

Much more often, how amoeba, in cavities mouth healthy of people meet Trichomonas. Weak mobile, Good visible in native preparation, in alive condition.At staining on Romanovsky - Giemse nucleus blepharoplast and flagella stained in red color, protoplasm - in blue. Reinforced reproduction Trichomonas going on just like amoeba, with unhygienic maintenance of the oral cavity. in a very large quantity they are found at periodontitis, at gingivitis.

Viruses oral cavity . Nearly at all healthy of people in oral cavity the herpes virus (Herpes vilgaris) is constantly present. This virus is transmitted even in childhood by airborne droplets from adult virus carriers. herpes vilgaris belongs to the group of DNA-containing herpesviruses, size 150 nm. Grown on chorionallantoic shell of the chick embryo.

At weakening protective forces macroorganism in result colds, overwork and etc. possible relapse illness.

Clostridia. Genus *Clostridium* - gram positive spore-forming sticks. Some kinds mobile thanks to availability flagella. Biochemically they active.AT norm are included in compound microbiocenosis intestines. AT cavities mouth determined some kinds not always.

stand out at sick With purulent wounds maxillofacial areas, rarely - at odontogenic inflammatory processes. At pollution wound surfaces and extensive traumatization fabrics Maybe development exogenous clostridial anaerobic infection, clinical manifestations which correspond classical picture gas gangrene. Main kinds: *FROM. perfringens, FROM. septicum, FROM. clostridiiforme, C. bifermentans* (the latter is found in odontogenic inflammatory processes).

other residents. Among bacteria with aerobic type breathing in cavities mouth meet also representatives actinomycete lines - nocardia and rotia (*Rothia dentocariosae),* which, possessing high adhesive and coaggregative properties, contribute formation dental plaques. Last view often determined in carious cavities and fistulas at actinomycosis, a also at non-specific osteomyelitis maxillofacial areas.

Non-fermenting gram-negative bacteria cavities mouth presented childbirth *pseudomonas, Bordetella, Eikenella (E.corrodens)* and some others. Among them most known bacteria *Pseudomonas (Actinobacillus) actinomycetemcommitam,* which violently develop at some young of people, causing progressive purulent juvenile periodontitis. Their role in the development of periodontitis adults in the present time being studied.

dental plaque. Her meaning in development caries teeth.

Using scanning electron and immunoluminescent microscopy shown what dental plaque consists in mostly from microbes With insignificant inclusion structureless substances organic nature.

AT formation dental plaques can highlight several major mechanisms.

1. Adhesion to enamel epithelial cells that are invadedbacteria With subsequent growth microcolonies,
2. precipitation extracellular glycans, produced S. mutans and S. Sanguis.
5. Precipitation of salivary glycoproteins forming a pellicle withsubsequent specific adhesion to her bacteria.
6. Agglutination of bacteria with antibodies followed by fixation onsurfaces enamel.

Using immunoluminescence microscopy _ shown what bacteria in dental plaque covered immunoglobulins classes BUT and G.

dental plaque starts form already in first minutes after purges teeth, and in the dynamics of its formation there are significant changes character microbiocenosis. General trend is change

composition flora from dominance aerobic and facultative anaerobic forms, predominantly gram-positive cocci, to obligate anaerobic gram negative chopsticks and tortuous forms.

3 phase formation dental plaques - first 1-4 hours after careful purges teeth (or processing ultrasound on the apparatus "Piezon-master"). She is predominantly consists from cocci (streptococci, neisseria, veillonella) and short sticks (diphtheroids). This, So called, "early" dental plaque.

4 phase - up to 4 - 5 days. Characterized by a decrease in the proportion of gram-positive cocci and growth shares gram-variable filiform forms - leptotrichian, a also gram-negative veillonella and fusobacteria. This phase maybe to be characterized how "balanced" or "dynamic" dental plaque. At persons With good adaptive abilities, With So called "high natural sanitation" microbiocenosis of dental plaque can be maintained in this able on the throughout significant segments life, not passing to next phase (when absence systematic purges teeth).

3 phase - from 6 - 7 days and Further. dental plaque accepts final on composition symbionts view, although quantitative shifts in her are happening constantly. The number of aerobic species - Neisseria, Rothium, facultative anaerobic streptococci. Dominated by gram-negative obligate anaerobic bacteroid bacteria, fusobacteria, veillonella and gram- positive actinomycetes, microaerophilic streptococci and peptostreptococci. it "mature" dental plaque. It characterizes the negative hygienic state cavities mouth and maybe induce development gingivitis in persons who are not regularly clean teeth.

Total number of bacteria in the dental plaque increases from 100-5000 in I phase formation before one - ten mln/g in 2 phase. AT 3 phase formations, in dependencies from many factors amount bacteria calculated dozens and hundred billion in one G.

established, what microbes possess different ability to adhesion even in respect various surfaces tooth. Except Togo, on the process adhesion affect and mechanical factors related with the process chewing, physical and chemical conditions andetc. Therefore, on different surfaces of the teeth, in the pits and fissures, the composition of the microflora several is different, even in within one tooth.

These data are of great practical importance due to the fact that condition dental plaques, how known is key mechanism occurrence and development caries teeth.

AT the present time established, what after reception food, especially rich carbohydrates in oral liquids going on sharp gain enzymatic activity bacteria - "metabolic explosion". basis "metabolic explosion" is activation glycolysis, what leads to sharp shift pH environments in sour side per check ejection sour catabolites - acetic, dairy, formic, pyruvic and others acids.

AT my turn, this is leads to exit ions calcium from solid fabrics tooth (demineralization), a also decrease content phosphates in process phosphorylation at bacteria. Except Togo, bacteria dental plaques accumulate excess carbohydrates in the form of reserve polysaccharides - dextrans and levans. In patients caries products organic acids much above, a normalization metabolic activity going on slower.

AT recent years installed role some resident-participants microbiocenosis dental plaques how antagonists cariogenic streptococci.

Before Total this is applies to veillonella - gram negative anaerobic coccam, which actively dispose of acids. it allows to consider veillonella how the most important microecological factor caries resistance.

dental plaque formed also and on the surfaces seals, and its composition somewhat different and depends on the nature and quality of the filling material. The most richly represented microbial flora on the cements and amalgams. Average level colonization typical for macrocomposite filling materials. And finally, on microcomposite and hybrid materials, dental plaque formed poorly due to the low affinity of bacteria. Usually in plaque microcomposite fillings are determined only microaerophilic streptococci and actinomycetes in small quantity.

These data have important practical meaning in connections With because condition dental plaques, how known is key mechanism occurrenceand development caries teeth.

AT the present time established, what after reception food, especially rich carbohydrates in oral liquids going on sharp gain enzymatic activity bacteria — "metabolic explosion". basis "metabolic explosion" is activation glycolysis, what leads to sharp shift pH environments in

sour side per check ejection sour catabolites - acetic, dairy, formic, pyruvic acids.

AT my turn, this is leads to exit ions calcium from solid fabrics tooth (demineralization), a also decrease content phosphates in process phosphorylation at bacteria. Except Togo, bacteria dental plaques accumulate excess carbohydrates in form reserve polysaccharides - dextrans and Levanov.

For study composition dental plaques use methodology taking material probe, metallic spatula or swab With subsequent weighing on the analytical scales. After this, in dependencies from tasks research, carry out mechanical trituration plaques or her disintegration ultrasound and quantitative sowing With using technology anaerobic cultivation. Quantity bacteria express in colony-forming units (CFU) in gram material.

Identification dedicated cultures before kind and kind allowed reveal significant differences in the proportion of bacteria of different genera in dental plaque and mucosa membranes of the oral cavity. So, in the composition of the dental plaque dominated by the frequency of excretion actinomycetes (20.4 *%)* and anaerobic cocci kind *Peptostreptococcus* (17.2 %)- By comparison With frequency allocation co mucous shells in dental plaque It was also much more alpha green streptococci (nearly in 3 times), lacto- and bifidobacteria (in 9 once). bacteria groups bacteroids and kind *Fusobacterium* on frequency allocation competed With dominant flora (14.7 and 5.7%), but their share was significantly less than in allocation co mucous shells cavities mouth.

At identification strains microorganisms, dedicated co mucous shellsof the oral cavity, the dominant flora in terms of frequency of occurrence were non-spore-forming gram- negative anaerobes of the bacteroid group (23.7%) and the genus *Fusobacterium* (14.9%). Whereinit should be emphasized that bacteroids were isolated almost 2 times, and fusobacteria - 2.5 times more often,how from dental plaques. Peptostreptococci also enough often stood out co mucous(15.8%), although somewhat less frequently than from dental plaque. The frequency of allocation of other cocci - veillonella, peptococci, microaerophilic streptococci and staphylococci - practically not different in dental plaque and co mucous.

drew on the myself Attention much more low frequency allocation co mucous shells actinomycetes, alpha streptococci and lactobacilli. Share facultative anaerobic and aerobic bacteria in dental plaque and on the mucous practically not differed.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15 min.

PRACTICAL OCCUPATION No. 5.

Topic: Cariogenic microflora. Microbiological methods of study microflora at caries teeth and his complications. Computer karyogram.

Educational goal:

4. Familiarize With methodology fence material at caries for bacteriologicalmethod research.
5. Explore microflora at caries.
6. Consider the role of microflora in the emergence and developmentcaries

Plan lessons:

6. Peculiarities microflora cavities mouth at caries teeth.
7. Streptococcus mutans and his role in occurrence caries.

8. experimental confirmation roles microbes in development caries.
9. Role local factors resistance at caries. Vaccine for preventioncaries.
10. Features of sampling material from the carious cavity for bacteriological method research.

Independent Work students:

3. Familiarize yourself with the features of taking material for caries forbacteriological method research.
4. Microscopic examination of smears from pure cultures of cariogenicbacteria and them antagonists:

a) demonstration smear from a pure culture of Streptococcus mutans. okr.gentian violet;

b) demonstration smear from a pure culture of actinomycetes. okr. gentian violet;in) demonstration smear from clean veillonella cultures. okr. magenta.

3. Method for quantitative determination of cariogenic flora on an examplelactobacillin test.
4. decor protocol research.

EQUIPMENT

1. Demonstration drugs Streptococcus mutans.
2. Equipment for cooking smear and coloring on Gram.

INFORMATIONAL MATERIL ON THEME

Caries - this is pathological process, at which going on demineralization andsoftening solid fabrics tooth With subsequent education cavities.

AT the present time known what carious process maybe develop atnext conditions:

4. Availability sufficient quantity carbohydrates in food;
5. Availability microorganisms in cavities mouth;
6. Contact carbohydrates and microorganisms With teeth.

bright proof roles carbohydrates in occurrence carious process are conducted experimental research. All cariogenic diets contain more than 50% sucrose. Content in diet of experimental animals smaller amounts of carbohydrates either does not cause a carious process, or it develops slowly.

There is now strong evidence that without contact teeth With carbohydrates carious process not arises. Undoubtedly important role belongs to the composition and structure of tooth enamel, saliva, as well as the nature of nutrition, composition drinking water. The study microbial flora at caries gave possibility install certain subsequence penetration various species microorganisms in fabrics carious tooth, a also reveal shifts in composition all microbial flora of the oral cavity in caries. Microorganisms are primarily penetrate into the enamel of a carious tooth after the destruction of the structure of all its layers. At initial lesions are found also microorganisms, which With points vision them biochemical activity may to be subdivided on the two groups: proteolytic and acid-forming.

The proteolytic group includes bacteroids and peptococci. They work out enzymes, capable split organic substances carious tooth.

To acid-forming group relate streptococci, lactobacilli and actinomycetes. Of the streptococci, enterococci are most often present here. All these microorganisms may participate in process demineralization solid fabrics carious tooth, because they intensively split carbohydrates and form a lot of organic acids.

All representatives of the permanent flora of the cavity are present in the carious cavity. mouth, main way strict anaerobes. On the cariogenic activity oral microorganisms are affected by saliva - its aggregating factors, which, on the one hand, contribute attachment microbial cells to surfaces tooth, a With another - delete them at washing cavities mouth.

The system of buffers has an anti-carious effect, bicarbonate - carboxylic acid, as well as protein and burn, which are in saliva. Prevention of caries be aimed at reducing the number of

cariogenic microorganisms in the cavity mouth. Effective application various bactericidal and bacteriostatic drugs. Good results are obtained with antiseptics, in particular 0.2% chlorhexidine. At this amount cells S. mutans in dental plaques declining on the 80 -
85 %, a in saliva on the 55 %. covering dental surface, chlorhexidine not only renders on the microorganisms bactericidal action, but and hinders them adhesion, not disturbing the microbial balance. inhibitory effect on microorganisms has fluorine and his connections. Another path decrease acid formation and accumulationglucans - replacement sucrose others carbohydrates at enzymatic splitting which these products not are formed.

At fence material at caries for holding bacteriological researchnecessary stick to some rules:

a) for eliminate access saliva in carious cavity necessary isolate tooth,for what his cover with cotton rolls;
b) superficial layers softened dentine necessary delete sterileboron;
c) deep layer softened dentine take away sterile excavator andproduce sowing;
d) inoculation is carried out on appropriate nutrient media for isolationanaerobic and aerobic microflora.

2. Spend microscopy demonstration swabs from pure cultures Streptococcus mutans, actinomycetes, veillonella under immersion.
3. Lactobacillin test produce next way:
unstimulated saliva collect on an empty stomach in sterile test tube. Cooking serial dilutions of saliva under sterile conditions from 1:10 to 1:10 0.1 ml from each dilution is sown on a dense elective nutrient medium with a low pH, rubbing material spatula on the entire surface of the agar.

After incubation at 37 hail.produce count quantity colonies, grown on the surfaces environment and recalculation for overall volume saliva.

For example, at breeding saliva 1:10 has grown ten colonies, means in 0.1 ml undiluted saliva contained 10,000 cells a in one ml - 100,000 cells.

Exists certain addiction between quantity brown" sogenicbacteria in saliva (lactobacilli, streptococci) and them antagonists (Veyalonell), what allows give the following recommendations:

a) use fluoride preparations for the prevention of caries;b) restrict use in food carbohydrates;
in) strengthen hygiene oral cavity.

4. Decor protocol research:

Table. Fence researched material from carious cavities.

researched material	Description results microscopy	Picture

TIMELINE

one. Definition original level knowledge	-------- thirty min.
2. Independent Work	--------- 70 min.
3. Examination protocols	--------- 10 min.
four. Cleaning working places	--------- 10 min.
5. Control final level knowledge and exercise on the house	--------- 15 minutes.

PRACTICAL OCCUPATION No. 6.

Topic: Periodontopathogenic microflora. Microbiological methods study microflora at diseases periodontal. Tactics antibacterial therapy anaerobic infections maxillofacial area. ***test control***

Educational goal:

1. Familiarize With features fence researched material for microscopicand bacteriological research methods.
2. Peculiarities composition microflora at non-specific lesions mucousshells cavities mouth (cheilitis, glossitis, stomatitis), the reasons their occurrence.

Plan lessons:

1. Methods study quantitative and quality composition microflora gingivalgroove and periodontal pockets.
2. Main representatives resident microflora at absence pathology fabricsperiodontal.
3. Peculiarities composition microflora at gingivitis
4. Peculiarities composition microflora at periodontitis
5. Periodontogenic microbes. Proof of them participation in pathogenesis diseases
6. Immunological changes, ongoing in answer on the bacterial antigens andtoxins
7. Modern methods treatment diseases periodontal in compliance With lastscientific data
8. Change module.

Independent Work students:

4. microscopic study demonstration smear-imprint co mucous shells at ulcerative necrotic stomatitis (fusospirochetosis), coloring on Romanovsky.
5. microscopic study demonstration scraping smear co mucouslanguage with leptotrichosis, coloring on Romanovsky.
6. Methods laboratory diagnostics candidiasis.
4. decor protocol research.

EQUIPMENT

3. Demonstration drugs Porf. Gingivalis.
4. Equipment for cooking smear and coloring on Gram.

INFORMATIONAL MATERIL ON THEME

Before present time not at all clear question - is whether periodontitis logical completion gingivitis. AT experiment on the animals (dogs) managed demonstrate this subsequence, but at of people gingivitis not always passes in periodontitis.

By about mechanism development periodontitis exist, how minimum, two points vision:

1. Exist certain germs, defiant destructive defeat fabrics periodontal.
2. To development periodontitis leads failure in functioning protective mechanisms organism and changes in composition and quantity microflora periodontal pocket.

At darkfield microscopy comes to light significant shift in side rod-shaped forms and spirochete, amount which increases before 40%. Attitude mobile forms to motionless increases before 1:1 (in norm 1:49).

Electron microscopic study subgingival plaques at periodontitis revealed what to cement attached, in basically, gram-positive microbes. Gram negative cells, flagella and spirochetes

present in in large numbers in loose layers subgingival plaque that spreads before apical parts pocket.

At bacteriological research material from sick periodontitis established dominance Gram-negative anaerobic sticks, in basically, subspecies non-saccharolytic *Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, Selenomonas sputigena, Eikenella corrodens, Campylobacter rectus* and others However at some patients observed prevalence actinomycetes. Many kinds anaerobic bacteria not succeeded to identify.

AT the present time, on data WHO, to periodontopathogenic types refer, first of all, two representatives of the bacteroid group - *Porphyromonas gingivalis and Prevotella intermedia*. Data gram negative microbes possess ability cling in big quantity to epithelial cells hydroxyapatite and to gram positive bacteria. Them adhesive properties are inhibited in presence human saliva and serum blood. However ability to coaggregation With gram-positive bacteria at this not is inhibited.

At periodontitis characteristic is education microbial clusters, reminiscent corn cob, which consist from cocci and tortuous forms.

At research content gingival pocket at sick periodontitis determined immunoglobulins classes A, g, M, factions complement NW, C5, leukocytes. fabrics gums plentifully infiltrated plasmatic cells lymphocytes and macrophages (monocytes). All this is allows count, what many reactions antigen-antibody, manifestations cellular immunity are happening exactly here, in tissues periodontal and alveolar bones.

If we adhere only to the microbial etiology of periodontitis, then it is obvious that for development diseases must combine the following terms:

1. Presence periodontopathogenic species bacteria in quantity, sufficient for Togo, to has begun pathological process.
2. Terms a habitat in niche must promote growth and reproduction bacteria.
6. Periodontal tissues should be free of antagonist microbes periodontopathogenic bacteria.
7. The microbe must be spatially localized so that it orproducts his vital activity could act on the target cells.
8. organism human must to be sensitive to microbes or productsthem vital activity.

Understanding etiology and pathogenesis periodontitis necessary not only for establishing roles microbes in this process, but also and for clarification conditions, conducive growth plaques, definition roles local and systemic factors which may influence on the resistance or sensitivity fabrics periodontal to bacteria products them vital activity. The study individual features organism host in functioning destructive and protective mechanisms at periodontitis allows optimize comprehensive treatment given diseases.

Microflora at periodontitis - prevails streptococcal Flora above staphylococcal. AT primary stages inflammation this is usually green and non-hemolytic streptococci without group antigen. At transition acute periodontitis in a chronic main role is played by streptococcal anaerobic flora, those. peptostreptococcus, to which are joining other streptococci. AT apical granulomas are found actinomycetes, bacteroids, fusobacteria, vibrios and spirochetes.

microbial Flora at periodontitis.

periodontal disease is one from most common diseases cavities mouth and represents yourself inflammatory -dystrophic process in alveolar processes, emerging due to violations nutrition alveoli.

By about mechanism development periodontal disease exists, how minimum, two points vision;

1. Exist certain germs, defiant destructive defeat fabrics periodontium.

2. To development periodontal disease leads failure in functioning protective mechanisms organism and change in composition and quantity microflora periodontal pocket.

All inflammatory processes in periodontium begin with education dental plaques predominantly subgingival, in result colonization surfaces teeth optional anaerobes.

AT the present time, on data WHO, to periodontopathogenic types refer, before Total, two representatives groups bacteroids -Porphyromonas gingivalis and Prevotella melaninogenica. These Gr - microbes have the ability to stick to in large numbers to epithelial cells and to Gr + bacteria. Their adhesive properties are inhibited in presence human saliva and serum blood. At periodontitis characteristic is education microbial clusters, which remind corn cob and consist from cocci and famous forms.

At research content gingival pocket at sick periodontitis determined immunoglobulins classes A, g, M, factions complement cj, C5, leukocytes. If a stick to only microbial etiology periodontitis, then obviously, what for development diseases must combine the following terms:

one. Presence of periodontopathogenic species bacteria in quantity,sufficient for Togo, to has begun pathological process.

5. Terms a habitat in niche must promote growth and reproduction bacteria.
6. Periodontal tissues should be free of microbes - antagonistsperiodontopathogenic bacteria.
7. human body must be sensitive to microbes or products their livelihoods.

1-2. scraping co mucous shells, backrests language can do sterile spatula, trowel. The material is applied to the surface of a glass slide, in a drop water. Gram or Romanovsky stain. Before taking material from erosions and ulcers it is advisable to remove surface plaque with a sterile cotton swab, without using at this antiseptic drugs.

At ulcerative necrotic stomatitis in smear watching abundance gram- negative spindle-shaped rods (fusobacteria) and convoluted forms (anaerobio- spirilla and spirochetes) on the background leukocytes and desiccated epi telium.

With leptotrichosis, a smear shows accumulations of gram-variable filamentous bacterial forms, and part bacteria situated how b in single case (leptotrichia).

3. For laboratory diagnosis of oral candidiasis, the following are used: methods:

a) Microscopic examination of a smear-imprint from the mucous membrane sick candidomycosis can see: oval and sharp elongated cells yeast-like fungus, arranged in the form of long chains (pseudomycelium). Not pseudomycelia form Xia flask-shaped swelling, from which lace up chlamydospores (feature of the species C. albicans).

b) When bacteriological method research material, received from sick, sown on Sabouraud media (agar-agar, carbohydrates, peptone) at t = 37 degrees, for 3-5 days. grown up colonies on the this environment studied macroscopically.

Colonies of the fungus of the genus Candida are round, whitish, convex, smooth with even edges, shiny surface. Sometimes the colony grows in agar.

Microscopic examination of smears shows pseudomycelium, consisting of oval elongated, finger-shaped cells located in clusters. Chlamydospores round, resemble bundles of balls.

in) At visceral mycoses apply for serological diagnostics reactions

- agglutination and RSK.For these reactions at sick take serum and determine Availability antibodies in serum sick With candidal diagnosticum.

Scheme reactions agglutination

Components test tubes	one	2	3	four	5	6	7
one. Phys. rr	1.0	1.0	1.0	1.0	1.0	1.0	1.0

2. Researched serum 1:40	1.0 1:80	1.0 1:160	1.0 1:320	1.0 1:640	1.0 1:1280	1.0 1:80	- -
3. Cell antigen (diagnosticum)	1.0	1.0	1.0	1.0	1.0	1.0	-
ACCOUNTING RESULTS							

Accounting results carry out, beginning With control test tubes (6 and 7). O results reactions judged by education draft in experimental test tubes. 4-6. In theory take apart clinical manifestations in cavities mouth at syphilis diphtheria, tuberculosis, herpes, foot and mouth disease using educational allowance. results design in form tables (on graphs: nosological the form; pathogen and his morphology; clinical symptoms in cavities mouth).

7. Decor protocol research on pp. No. 1-3.

Table. Fence researched material at periodontitis

Исследуемый материал	Описание результатов микроскопии	Рисунок

Table. Fence researched material at gingivitis

Исследуемый материал	Описание результатов микроскопии	Рисунок

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15 min.

PRACTICAL OCCUPATION No. 7.

Topic: The study microflora purulent detachable at inflammatory diseases maxillofacial areas. Technique anaerobic cultivation bacteria With quantitative accounting. Ways identification and definitions sensitivity anaerobes to antibiotics.

Educational goal:

7. Technique anaerobic cultivation bacteria With quantitative taking into account
8. Ways identification and definitions sensitivity to antibiotics.
9. Tactics of antibiotic therapy of anaerobic infections of the maxillofacial areas.
10. Peculiarities etiology inflammatory diseases maxillofacial areas (associations, mix-infections).

11. Familiarize With methods fence and microbiological research material for acute and chronic inflammatory diseases of the maxillo- facial areas.
12. Explore major representatives opportunistic anaerobic infections maxillofacial areas.

Plan lessons:

8. Methods study quantitative and quality composition microflora gingivalgroove and periodontal pockets.
9. Main representatives resident microflora at absence pathology fabricsperiodontal.
10. Peculiarities composition microflora at phlegmon CHLO.
11. Peculiarities composition microflora at abscesses CHLO.
12. Advantages and limitations the most important methods definitions sensitivitybacteria to antibiotics.
13. Indications and contraindications to application antibiotics at diseases CHLO.
14. Mechanism workings sustainability to antibiotics at bacteria.

Independent Work students:

8. Familiarize yourself with the features of sampling the test material from the gingival groove and pathological gingival pockets for microscopic and bacteriological research methods.
9. microscopic study demonstration smears from content deep periodontal pockets. Coloring on Gram.
10. microscopic study smears from pure cultures "periodontopathogenic" anaerobic bacteria:
    a) smear from a pure culture of Prevotella melaninogenica, Gram stain; b) smear from clean Actinomyces cultures naeslundii, coloring on Gram.
11. disassemble bacteriological method research purulent exudate Withusing technology anaerobic cultivation (demonstration microanaerostat, transport medium, some environments: thioglycol medium, Wednesday With heminoma, brain heart agar and broth and etc.)
12. disassemble scheme pathogenesis inflammatory process and sketch.
13. Spend bacteriological study material from sick odontogenic infection (stage 1 research).
14. Decor protocol research.

EQUIPMENT

one. Equipment for cooking smear and coloring on Gram.

INFORMATIONAL MATERIL ON THEME

1. Peculiarities fence researched material at diseases periodontal.

For microscopic research content periodontal pocketscollected with a curettage spoon or celluloid plate. From the inside sides of the plate - microbes stick on the surface of the root part tooth, a With outdoor — located in gum fluid.

For bacteriological research content periodontal pockets necessary place in transport nutritional Wednesday With purpose conservation viability of anaerobic bacteria. In case of examination of sub-gingival dental plaque removed from the periodontal pocket, it must be disintegrated before sowing With help ultrasound. Further selection pure cultures, them cultivation and identification carry out in anaerobic conditions on classical

scheme.

2. At microscopic research content periodontal pocket in demonstration preparations reveal morphological forms characteristic for major paprodontopathogenic species - actinomycetes, streptococci, bacteroids, fusobacteria and spirochetes.

3. Detailed study of the morphology of periodontopathogenic bacterial species in smears from pure cultures. Coloring on Gram.

4. Decor protocol research

Table. Fence researched material at abscesses of the maxillofacial area

researched material	Description results microscopy	Picture

Pathological material at abscesses phlegmon and fasciitis take puncture With help thick needles and deliver in laboratory in syringe. AT process operational intervention, the material is taken using a standard cotton swab, which placed in a transport medium. Transport media, due to the peculiarities of its composition, provide sharp decline metabolism microbes and possibility long conservation them vitality (from 6 - one 2 hours).

I **stage bacteriological research** - receiving isolated colonies. Usually performed on the cups petri With 5 % anaerobic hemagar - nutritional environment, which besides native blood contains such factors growth anaerobic bacteria like hemin (vitamin K) and menadione. The medium is universal for growth majority species anaerobic and aerobic bacteria.

Petri dishes with an anaerobic hemagar are cultivated in an anaerostat or gas box at t - 37 ° C up to 7 - 10 days, although most of the anaerobes gives good colony growth already on the 3 - four day. At macroscopic and microscopic studying grown colonies compare the morphology of the bacteria themselves and the colonies they formed during cultivation. in an anaerobic balloon and received by 5 % blood agar in aerobic conditions.

Testing is essential for further identification. on the Availability catalase: material colonies mix on the subject glass With drop 0.5 % peroxides hydrogen - active education bubbles gas testifies about availability this microbe has an enzyme catalase, what usually characteristically for optional - anaerobic bacteria. Main kinds obligately - anaerobic bacteria catalase not produce.

II **stage bacteriological research** - receiving clean culture . Performed on the liquid (thioglycolic Wednesday, Wednesday kitta - Taroczi, cordially- cerebral bouillon) or semi-liquid environments (With adding 6.5 % agar agar). Material from isolated colonies endure in test tube With one of specified environments that then desirable place in anaerostat. Pure culture receive through 3 - 5 days cultivation at t- 37° FROM.

III **stage bacteriological research** - identification clean culture. For definitions kind dedicated culture used definition complex morphological, tinctorial, cultural, biochemical and chemotaxonomic properties.

results bacteriological research bring in in protocol.

acute odontogenic inflammatory diseases	abscess phlegmon osteomyelitis lymphadenitis	bacteroids, fusobacteria, peptostreptococcus, peptococci, less often actinomycetes, staphylococci
chronic odontogenic inflammatory diseases, actinomycosis		obligate associations anaerobic bacteria with facultative anaerobes (staphylococci, bacilli, streptococci) aerobic and anaerobic actinomycetes (rotia, nocardia and streptomyces).
nonodontogenic inflammatory diseases	furuncle festering atheroma infected injury people-persons.of the region	the predominance of staphylococci, streptococci, less often bacilli and obligate anaerobes, often stand out monocultures

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15 min.

PRACTICAL OCCUPATION No. eight.

Topic: Chronic foci infections. pathogens tuberculosis and leprosy. Features of diagnosis and manifestation of infection in the oral cavity. Prevention and treatment tuberculosis

Educational goal:

2. Bacterial infections and their manifestation in cavities mouth (tuberculosis).

Lesson plan:

6. Peculiarities composition microflora cavities mouth at tuberculosis.
7. Manifestations tuberculosis in cavities mouth.
8. Peculiarities fence researched material at tuberculosis.
9. Methods laboratory diagnostics tuberculosis.
10. Modern methods treatment tuberculosis.

Independent Work students:

3. Theoretical analysis of clinical manifestations in the oral cavity of diphtheria andtuberculosis, diagnostic methods.
4. Decor protocol research.

EQUIPMENT

1. Equipment for cooking smear and coloring on Gram.
2. Tables.

INFORMATIONAL MATERIL ON THEME

Tuberculosis of the mucous membrane of the mouth and lips is caused by mycobacteria tuberculosis, in mostly human type, and usually is secondary, less often primary tuberculosis of the oral mucosa develops in the form of primary tuberculosis complex. Mycobacteria tuberculosis may hit in mucosa shell mouth how endogenous through So and exogenous.

Mucous shell cavities mouth is bad environment for breeding tuberculosis mycobacteria; having hit in mucosa shell at majority sick tuberculosis, they die. If, nevertheless, its defeat occurs, then the clinical form diseases depends from row factors before Total from general currents tuberculosis process and immunological states organism, which determine With help tuberculin reactions. AT pathogenesis tuberculosis certain role play character food, neuro-endocrine disorders and others

From the forms of secondary tuberculosis with damage to the mucous membrane mouth can observed lupus erythematosus, scrofuloderma and miliary ulcerative tuberculosis, moreover, if two first forms flow usually on the background positive tuberculin reactions, then malarial and ulcerative tuberculosis arises predominantly on the background anergy, t. e. on the background negative tuberculin reactions.

Primary tuberculosis of the lips and oral mucosa. primary tuberculosis , or primary tuberculous complex, or primary tuberculous chancre, on the lips and oral mucosa is rare, mainly in children. He arises in result exogenous infection, which going on more often air- drip, less alimentary way. This form of tuberculosis can only develop in people who do not have mycobacterium tuberculosis in the body and tuberculin reactionsnegative.

After an incubation period of 8-30 days , at the site of the entrance gate of infection painful ulceration up to one- 1.5 cm with undermined uneven edges with a dirty gray bottom. The bottom and edges of the ulcer are slightly compacted, however, on the lips, the seal can be significant. After 2-4 weeks after education ulcers increase and submandibular lymphatic nodes. At first they are mobile, and then soldered to each other and to the skin. Often through some time these nodes suppurate and are opened.

Tuberculous lupus. Among tuberculosis diseases of the mucous membrane mouth and lips tuberculosis lupus is most frequent stubborn inclined to relapses, chronically current

disease. Beloved localization lupus erythematosus is a person that is affected in approximately 75% of patients, and very often red is involved in the process border top lips on which process usually passes With nose. However maybe to be and isolated defeat red borders top lips.

Primary element at tuberculosis lupus is tubercle (lupoma). lupoma represents yourself limited, in early flat, with pin head or a little more red or yellowish red soft painless education, prone to peripheral growth and merger With neighboring elements. AT result mergers lupom formed foci defeat, having various dimensions and outlines.

Lupus foci on the red border of the lips and especially on the oral mucosa ulcerate. The edges emerging at this ulcers corroded, wrong forms. Bottom ulcers covered or dirty gray raid, or papillomatous expanding granulations, sometimes they resemble bright juicy raspberries. On the red border lips on the surfaces ulcers often formed crusts, sometimes very thick.

On the place defeat remains superficial cicatricial atrophy; characteristically re-occurrence of individual loops on such a scar. In places of ulceration, rough, disfiguring scars form. Lupus ulcerative process, although rare, leads to significant destruction fabrics.

In second stages on the background edema and hyperemia appear individual small tubercles, which present yourself papillary overgrowth, covered slightly tarnished epithelium. Merging with each other, they can resemble warty overgrowth. AT subsequent at majority sick tubercles break apart With the formation of an ulcer, which can be of different sizes, irregular outlines, often with corroded, but unburied edges, With granulations on the bottom and narrow an inflammatory border around, against the background of which one can often see individual surviving tubercles, a also erosion. AT detachable from ulcers, how rule microscopically not succeed discover tuberculosis mycobacteria. At completion process scars are formed, and if the process proceeded without ulceration nia, then they smooth, shiny, atrophic. After ulceration scarring dense, rough, solder mucosa shell With subject tissues.

Clinical painting tuberculosis lupus It has some peculiarities, process localization. According to the location of the lesion on the mucosa shell gums distinguish four kind defeats: one) marginal encompassing gingival edge first in form banal infiltration and passing then in tuberculous-erosive (ulcerative) form; while the gingival margin and interdental papillae sharp swell up picture gingival the edges smoothed out mucous shell gums takes on a bright red color. The gum appears as if pierced pins, painless matte dim, easily bleeds; 2) supramarginal: infiltrative or tubercular-ulcerative defeat not affects gingival border; 3) total: process captures all outer surface gums on type infiltrative, often erosive, and sometimes ulcerative lupus. With this form often is affected bone the cloth alveoli, maybe develop "painting hypertrophic stupid gingivitis"; four) bilateral, flowing on type of ulcer lupus.

Treatment tuberculosis mucous shells cavities mouth, being one from manifestations general tuberculosis, carry out on generally accepted methodologies treatment tuberculosis usually in tuberculosis dispensaries. Most effective means treatment tuberculosis lupus are drugs hydrazide isonicotinic acids (ftivazid, isoniazid, or tubazid, salusite, metazid, larusan, INGA-17, etc.), which affect mycobacterium tuberculosis not only bacteriostatic, but and bactericidal action.

Prognosis in patients with tuberculosis of the oral mucosa at the present time, at availability powerful tuberculosis funds, good, but sick must for a long time, until a complete cure, be under dispensary observation. AT early identifying tuberculosis patients mucous shells cavities mouth, in direction them on the treatment in anti-tuberculosis institutions, in organizations dispensary observations Dentists play a big role. This is due to the fact that lupus erythematosus and miliary-ulcerative tuberculosis can begin and exist for a long time only on mucous shell cavities mouth, in connections With how sick, naturally, apply to dentist.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15 min.

PRACTICAL OCCUPATION No. 9.

Topic: Microbiological diagnostics dysbiosis cavities mouth and stomatitis. Dysbiosis and opportunistic stomatitis. Opportunistic processes how manifestations immunodeficiencies and HIV infections. laboratory diagnosticscandidiasis, leptotrichiasis, fusospirochetosis.

Educational goal:

6. Familiarize yourself with the features of sampling the material under study formicroscopic and bacteriological methods research.
7. Peculiarities composition microflora at non-specific lesions mucousshells cavities mouth (cheilitis, glossitis, stomatitis), the reasons them occurrence.
8. Bacterial infections and them manifestation in cavities mouth (gonococcal gingivo-stomatitis).
9. To master the technique of sampling the test material for microscopicresearch at non-specific lesions mucous shells cavities mouth.
10. master methodology fence researched material for bacteriologicalresearch at non-specific diseases mucous shells cavities mouth

Plan lessons:

1. Features of the sampling of the test material for microscopic andbacteriological research.
2. Peculiarities composition microflora at non-specific lesions mucous cavitiesmouth (cheilitis, glossitis, stomatitis), the reasons them occurrence.
3. HIV infection. Manifestations in cavities mouth.
4. Candidiasis. laboratory diagnostics candidiasis.
5. Leptotrichiasis. laboratory diagnostics.
6. Fusosperochitosis. laboratory diagnostics.

Independent Work students:

3. Theoretical analysis clinical manifestations in cavities cheilitis, glossites,stomatitis, HIV infection, diagnostic methods.
4. Decor protocol research.

EQUIPMENT

1. Equipment for cooking smear and coloring on Gram.
2. Tables.

INFORMATIONAL MATERIL ON THEME

Viruses are most frequent cause infectious diseases(measles, epidemic mumps, rubella, flu, windmill smallpox, hepatitis, AIDS and etc.).

Another virus for which parenteral transmission is also maybe to be basic, - this is virus immunodeficiency human (HIV). Virus HIVstrikes lymphocytes (T-helpers) and macrophages. At long reproductions virus inbody populations helpers is shrinking in affected macrophages declining production interleukin-I. Except immune systems suffer other bodies andsystems: nervous system, bodies digestion and breath, cardiovascularsystem , etc. Against the background of immunodeficiency , Kaposi 's sarcoma develops and opportunistic diseases (candidiasis, ulcerative necrotic stomatitis, cytomegalovirus infection, herpes, pneumocystosis).

Sources virus are sick and virus carriers. Available parenteral route of infection when using an infected virus blood with various surgical interventions, blood transfusion, etc., as well as sexual contact. Registered cases infections fetus in intrauterine period.

Diagnostics held main way serological method, through identifying antibodies With help enzyme immunoassay analysis.

Thus, patients who came to the reception with candidomycosis, relapses herpes, ulcerative necrotic lesions must be checked on the infection HIV. special danger is posed by patients at risk - homosexuals, prostitutes, drug addicts, patients who often received blood transfusions (hemophilia).

A dentist can become infected with a virus from a patient airborne by and during a digital examination, as well as the doctor himself during his manipulations maybe infect patient (hepatitis AT, AIDS).

Additionally necessary Mark, what about near 46 % patients on the reception can be carriers in the oral cavity of Streptococcus puogeus, Staphul. aureus. They are can cause the doctor to develop various respiratory diseases, as well as cause an inflammatory process in case of a hand injury with a drill or a stab wound. Considering that, that many of the staphylococci are antibiotic-resistant, the entry of these microbes maybe make it difficult process treatment.

Leprosy

Leprosy (leprosy) called leprosy mycobacteria Hansen and represents yourself chronic generalized infection developing predominantly in derivatives ectoderm and in bodies and fabrics, rich elements active mesenchyme. Leprosy leaks chronically progressive With periodical exacerbations (leprosy reactions), leading in the absence of rational treatment for severe disability and through a lot of years to of death.

Allocate tuberculoid and lepromatous types leprosy. emergence Togo or another type of disease related with fortune resistance organism human to leprosy infection, which determine With help lepromine skin samples. Mucosal lesions shells cavities mouth may arise only at lepromatous type, which the develops at persons With sharp reduced reactivity (lepromine try negative). At such sick on the skin, mucous shell mouth, internal bodies, on move nervous trunks arise lepromatous infiltrates With leprosy cells not able (in difference from tuberculoid type) destroy phagocytosed them sticks Hansen, that's why they freely multiply in these cells.

Leprous process on mucous shell mouth begins with infiltrative stage, then on the this background arise tubercular rashes which through some time ulcerate. Process ends scarring. Highly often on the mucous shell mouth at one and Togo same sick can observe elements, characteristic for all four stages development leprosy defeat mucous shells, t. e. infiltration tubercles, ulcers and scarring. Clinical painting leprosy lesions of the oral mucosa is characterized by polymorphism, each of four stages characterized described signs.

Leprosy changes mucous shells mouth begin With education superficial limited infiltrate, slightly rising above the surrounding mucous shell and having grayish white color, sometimes With dark blue plots. Then on the infiltrated plots arise tubercles various sizes - from millet grain to a cherry stone. At first, the tubercles are dense, then soften. They are located haphazardly and inclined to peripheral growth and merger. Tubercles mostly dull pink, sometimes grayish pink colors. Them surface usually shiny. More often Total tubercles arise on the solid and soft sky, language and lips. Through some time tubercles usually ulcerate. ulcers on the place tubercles at first small, bottom them bumpy, dirty gray white colors, the edges uneven, swollen, soft consistency. Sometimes ulcerative process distributed by on the bone the cloth, causing her meltdown. More often Total at this collapses alveolar edge jaws. AT subsequent ulcers scarred but scarring may develop without previous ulceration tubercles, when the tubercle or infiltrate of the oral mucosa are fibrosed. Scar formation is not yet means recovery, for simultaneously may arise new infiltrates, tubercles and ulcers. Leprosy scarring on the mucous shell cavities mouth may to be round or in form stripes — radiant. Scarring smooth, shiny, white colors. formed scarring in dependencies from them sizes and places location may cause functional disorders. Especially often the soft palate is deformed and tongue,

which the maybe shift a sometimes even disappear.

Leprosy defeat may arise and on the lips. At this very often a pronounced infiltrate is formed, accompanied by edema, which entails education leprosy elephantiasis. Lip compacted, becomes more thick, roller-shaped and sedentary. Leather lips and red border on bloom few different friend from friend. On the this background arise Good contoured tubercles.

Leprosy changes on the inner surface lips begin With diffuse erythema, then an infiltration occurs view bluish spots, covered with thickened epithelium. Against this background, sometimes bumps appear, which nearly not act above mucous shell. Leprosy tubercles on the lips may long time remain unchanged but sometimes they ulcerate. The emerging shallow painless ulcers located on the surfaces tubercles. Them detachable dries up in light yellow crusts.

Ulcerative defeat mucous shells lips always ends scarring with deformity of the lips, resulting in a thinner lip, the mouth opening may narrow down. If a same deep located tubercles fibrosis, then lip wrinkled, due to what finds it difficult speech, leather lips in these cases happens dotted furrows a red border lips becomes wrinkled. Leprosy changes mucous shells lips rarely reach before transitional folds.

Beloved localization leprosy rashes are gums on the topjaws from the side of the tongue in the area of the frontal, less often molars. Leprosy changes in the gums begin with the formation of an infiltrate. The gums seem to swell are made loose, red, sometimes cyanotic slightly bleed; gingival papillae swell up picture gingival the edges smoothed out. Highly often to this increased salivation joins. Soon the gingival mucosa becomes matte, on the her surfaces formed sores, which then scarred what leads to wrinkling of the gingival margin and turning it inward. The gums retract, the roots of the teeth exposed. Simultaneously with scarring in other areas of the gums, fresh infiltrate. characteristic painless process.

Leprosy changes on the mucous shell solid sky are happening next way: in those cases, when defeat starts on the front thirds at first noted infiltrative stage, infiltrate formed behind necks central incisors and comes before fangs. Plot infiltrated mucous shells on direction back narrows and more often ends at the beginning middle thirds solid sky, in result what formed triangle, basis facing forward, and the top back. Significantly less often infiltrate in the direction narrows back to a strip 2 cm wide and extends along the midline to soft sky. Infiltrate on the lateral surfaces solid sky observed extremely rarely. infiltrated plot greater part It has grayish red color and absolutely painless. Then on the infiltrated plots arise grayish white tubercles magnitude With millet corn, which subsequently superficially ulcerate.

On the soft sky defeat starts With hyperemia, passing in infiltrate dark coloring. Sometimes soft sky at first It has pale yellow color. tongueusually infiltrated, its size increases. On the infiltrated plots soft sky and tongue appear whitish gray, focally located tubercles various quantities — from millet grain before peas. Then tubercles break apart and formed sores. Sometimes individual sores merge, forming greater or lesser quantities solid ulcerative surfaces. The edges ulcers slightly raised and undermined, bottom bumpy With grayish raid. Color them dirty grayish white. Due to ulcerative defeat usually collapses part or the whole tongue.The ulcerative process on the hard and soft palate ends with scarring. The emerging scarring have varied form: sometimes they round, but more oftenradiant or stellate. Scarring usually shiny, superficial, whitish colors. Cicatricial contraction on the solid sky near necks front teeth leads to wrinkling gums and retractions her With exposure roots teeth. The edges retracted gums wrap up inside in side roots, tight hugging them. Scarring on the soft palate sometimes form an arc, the convex part facing forward.fibrosis in areas soft sky often causes persistent deformation, due to what soft sky maybe pull up up, narrowing input in nasopharynx. Sometimes soft sky absolutely is destroyed.

Beloved localization leprosy elements on the language is average line his backs, beginning from root and before tip. Language maybe infiltrate increase, thicken, in result what his mobility finds it difficult and speech becomes obscure. On the infiltrated surfaces backrests language appear dense tubercles various quantities, but not more bob, With flat surface and wide basis. Surface tubercles shiny, covered whitish raid thanks to desquamation epithelium ("silver" language). Number tubercles may enlarge and may coalesce (leprosy glossitis),

resulting in what on the back of the tongue are formed roller-shaped elevations with deep furrows between them.

tubercles on the language inclined to decay and ulceration. formed ulcers located superficially and have jagged, undermined, infiltrated edges. The bottom of the ulcers shallow, rough, covered with a grayish coating. In some cases, these ulcers merge, forming a continuous ulcerative surface, covered with a thin gray coating. Emerging on the place ulcers various forms superficial scarring usually have greyishwhite color and brilliant surface.

For the diagnosis of leprosy, bacterioscopic studies of scrapings from mucous shells nasal partitions, a also co bottom and edges leprosy ulcers, in whicheasily discover sticks Hansen.

Leprosy lesions of the oral mucosa may resemble tertiary syphilis and lupus erythematosus. From the manifestation of lupus erythematosus and collicative tuberculosis, leprosy rashes are characterized by greater density, the presence of pronounced disorders sensitivity and leprosy mycobacteria in ulcerated dischargeelements.

Most effective antileprosy means are drugs sulfonic row, such, how diamino diphenyl sulfone (DS), avosulfone, dapsone, sulfetron (solusulfone, novotrope), which accept in flow long time. The less toxic drug thiocarbonylide has the same effect as sulfones. (derivative thiourea). Not lost meaning chaulmugrious oil and his derivatives, inin particular ethyl ethers and mugrol. In order to successfully treat patients with leprosy, always should apply combined therapy everyone famous antileprosymeans in combined With restorative drugs and physiotherapy.

Forecast at leprosy in recent decades in connections With introduction in practice treatment this disease of new, sufficiently effective drugs has become better. Main public prevention of leprosy is early detection and rapid isolation of patients with leprosy in special institutions - leper colonies, where patients are treated, live and work. At discovery sick or suspicious on the disease leprosythe doctor must immediately report this to the health authorities. Upon confirmation of the diagnosis leprosy sick With compliance measures precautions envisaged instruction People's Commissariat ways messages — People's Commissariat health care from 19/IV 1944 G., must to be delivered in leprosarium.

depending from localization inflammatory process mucous shells cavities mouth called various: stomatitis (cheek mucosa), glossitis (tongue), gingivitis (gums), cheilitis (lips). Stomatitis is usually either a consequence various dystrophic processes in body, infectious or somatic diseases, or result damaging physical or chemical impact on the mucosa at secondary roles resident microflora. At superficial catarrhal stomatitis usually find Gr + aerobic cocci and sticks, at deep stomatitis prevails strictly anaerobic Gr - Flora (fusobacteria, bacteroids, peptostreptococci).

At ulcerative necrotic stomatitis prevails anaerobic Flora, predominantly fusobacteria and spirochetes, but may attend and other microorganisms (veillonels, peptostreptococcus, bacteroids, vibrios, actinomycetes). To fusospirochetosis also refer ulceratively - necrotic sore throat Vincent, sore throat Ludwig, gangrene lung ulcerative colitis and etc.).

In recent years, there has been an increase in the incidence of candidomycosis. It's connected with widespread use of antibiotics, corticosteroids, cytostatics. Long them application leads to violation composition normal microbial flora (dysbacteriosis). Candida fungi are a resident of the mucous membranes of the oral cavity, digestive tract, respiratory ways, vagina, skin covers.

Process relationships yeast cells With epithelial cells oral mucosa begins with their adhesion. Sucrose, maltose, glucose and others carbohydrates increase activity adhesion. Adhesiveness yeast-like mushrooms of the genus Candida in many defines them virulence.

System complement, which activated mannan cellular walls yeast, inhibits their adhesion. Yeast-like mushrooms contribute to the destruction tooth enamel and the development of caries. Carious teeth in which yeast vegetates cells, can consider, how peculiar ecological niche-, thanks to which they may participate in development mycotic tonsillitis and stomatitis. Local manifestations candidiasis or primary candidiasis in cavities mouth leaks in form acute pseudomembranous candidiasis (thrush), acute or chronic candidiasis and hyperplastic candidiasis.

Gonococcal stomatitis

Gonococcal lesions of the oral mucosa - gonococcal stomatitis - meets rarely. The disease occurs in newborns when gonococci enter the cavity mouth child in time childbirth at passing

through infected generic way mother. maybe entering infections from caring personnel and others sick.

Usually observed simultaneous defeat gonococcus mucous shells mouth, nose and conjunctiva. Gonococcal stomatitis has also been observed in adults (Besionck, Janet and etc.). Gonococcal stomatitis is more common than commonly thought, but it remains unrecognized because what, firstly, inspection cavities mouth and nose most sick gonorrhea not produce, Secondly, dentists and otolaryngologists, to which more often of all patients who are unfamiliar with this disease are treated, and, thirdly, gonorrheal stomatitis more often leaks without subjective sensations, inclined to self-healing and sick disappear from under observations doctors.

In recent years, there has been an increase in reports of extragenital gonorrhea in adults. In this case, the pharynx and tonsils are more often affected, less common stomatitis, gingivitis, laryngitis. The lesion of the oral mucosa is the main way at homosexual men and persons, who had orogenital contacts.

Flow gonorrhea mucous shells cavities mouth and pharynx, how rule asymptomatic. At children, the act of sucking is not violated. Pain is rare in adults in the throat, the body temperature rises. The first symptoms of gonococcal stomatitis - hyperemia, edema, small erosion on the mucous shell and viscous mucopurulent more or less abundant secret. AT more heavy cases at absence treatment process maybe to spread appears big amount erosion and ulcers on the mucous shell cheeks, language, gums sores superficial, small sizes, With wrong unburied or few undermined edges, soft, painless, With sparse yellow gray separating immovable, in which discover gonococcus, what confirms diagnosis.

Histologically determined inflammatory process in subepithelial connective tissue with infiltration by lymphocytes, neutrophils, plasma cells.

Treatment of gonorrheal stomatitis is carried out with antibiotics in the same doses as gonorrheal lesions of the genitourinary organs. Locally prescribed rinses 0.01-0.1% solution permanganate potassium.

Prevention of gonorrheal stomatitis in newborns born from mothers sick gonorrhea, consists in processing mucous shells mouth newborns immediately after birth 2% solution nitrate silver. Adult patients with gonorrhea genitourinary organs, the mucous membranes of the oral cavity and pharynx should be examined, with testimony carry out study detachable on the gonococcus

Laboratory diagnosis carried out through use next methods:

3. Microscopic examination (light, luminescent) pathological material (raid, pieces bodies and etc.) and discovery young or mature pseudomycelium.

4. Bacteriological study - sowing material on the Wednesday Saburo, tomato bouillon and rice agar With identification dedicated culture.

3. Serological method - staging reactions agglutination and binding complement With serum blood sick With purpose detection antibodies.

TIMELINE

1. Definition original level knowledge ---------- 30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ---------- 10 min.

5. Control final level knowledge and exercise on the house ---------- 15 min.

PRACTICAL OCCUPATION No. ten.

Topic: infectious stomatitis and diagnostics manifestations bacterial and viral infectious diseases in cavities mouth. laboratory diagnostics diphtheria, gonorrhea, syphilis. Prevention and treatment.
laboratory diagnostics herpetic, coxsackie- and echo viral stomatitis. Principles prevention
.

Educational goal:

5. Familiarize yourself with the features of sampling the material under study formicroscopic and bacteriological methods research.
6. Peculiarities composition microflora at diphtheria, gonorrhea, syphilis herpesthe reasons them occurrence.
7. To master the technique of sampling the test material for microscopicresearch at diphtheria, gonorrhea, syphilis, herpes.
8. master methodology fence researched material for bacteriologicalresearch at diphtheria, gonorrhea, syphilis, herpes.

Plan lessons:

6. infectious stomatitis. Manifestations in cavities mouth. laboratory diagnostics.
7. Diphtheria. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.
8. Gonorrhea. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.
9. Syphilis. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.
10. Herpes. Manifestations in cavities mouth. laboratory diagnostics. Prevention. Treatment.

Independent Work students:

3. Theoretical analysis of clinical manifestations in the cavity of diphtheria, gonorrhea,syphilis, herpes, diagnostic methods.
4. Decor protocol research.

EQUIPMENT

1. Equipment for cooking smear and coloring on Gram.
2. Tables.

INFORMATIONAL MATERIL ON THEME

Syphilis is a chronic infectious disease caused by Treponema pallidum. Syphilis characterized very peculiar flow: firstly, undulating change active manifestations and periods is hidden flowing infections; Secondly, gradual anda consistent change in the clinical and pathoanatomical picture of lesions bodies and fabrics from mild inflammatory phenomena before education specific deep infectious granuloma, squeezing and destructive bodies and tissues in which they are localized, which leads to loss of organ function, and sometimes to of death sick.

There are acquired and congenital syphilis. Congenital syphilis occurs when hit pale treponema in organism fetus through placenta from sick syphilis mother. For infections human syphilis necessary penetration pale treponemathrough skin or mucosa shell, integrity which violated.

Usually infection occurs sexually. Sexual infection can be professional, for example, in medical workers during operations, autopsy, dental or gynecological examination, etc., or occur when usinggeneral crockery, labial lipstick mouthpieces and others

Due to the undulating course of syphilis, the different nature of clinical and morphological changes that occur at various stages of the disease are distinguished incubation, primary, secondary and tertiary periods acquired syphilis, a also hidden, in volume including unknown, visceral syphilis and syphilis nervous systems.

Incubation period syphilis in average equals 3-4 weeks, but Maybe how hisshortening (before 10-12 days), So and elongation (before 6 month), which usually related With reception in time incubation small quantity antibiotics on about intercurrent diseases or gonorrhea.

The primary period of syphilis begins with the onset at the site of infection, i.e. the introduction of pale treponema, hard chancre (primary syphiloma). Primary Period lasts 6-7 weeks. 5-7 days after the formation of a hard chancre, a second an indispensable symptom of the primary period - regional lymph nodes (bubo, or regional scleradenitis) increase. These nodes thrive treponema. From the lymph nodes along the lymphatic pathways already at the beginning of the primary period treponema fall in blood, in answer on the this is gradually start be developed antibodies, which at the end of the 3rd week of the primary period of syphilis can be determined in blood using classical serological reactions (Wassermann reaction, sedimentary reactions), several before — with the reaction immunofluorescence (REEF), a a little later - and with the help of the immobilization reaction of pale treponema (RIBT, or RIT).

About at twenty% sick to end of primary period syphilis develop general symptoms (increase temperature body before 38-38.5°C, weakness, head pain, pain in bones especially on nights), in peripheral blood observed small anemia, leukocytosis, increase in ESR. After 4-6 days against this background the skin of the body, and often on the mucous membrane of the oral cavity, a rash appears, which testifies about graduation primary and early secondary period syphilis.

Mucous shell cavities mouth and red border lips are localization syphilitic rashes in all stages diseases, in volume including and at primary syphilis. At non-sexual infections localization chancre on the lips and mucous shell cavities mouth meets most often. Solid chancre maybe arise on the any site red borders lips or mucous shells cavities mouth, but more often Total he localized on the lips language, tonsils.

The development of a hard chancre on the lip or oral mucosa, as well as on others places starts With appearance limited redness, in basis which within 2-3 days there is a seal due to the inflammatory infiltrate. it limited seal gradually increases and reaches usually 1-2 cm in diameter. In the central part of the lesion, necrosis occurs and erosion of the meat- red, rarely ulcer. Having reached full development within 1-2 weeks, solid chancre on the mucous membrane is usually a round or oval painless lesionny erosion of meat-red color or an ulcer with saucer-shaped edges ranging in size from 3 mm (pygmy chancre) up to 1.5 cm in diameter with dense elastic infiltrate in basis. In the scraping of the surface of the chancre, pale treponemas are easily detected. Sometimes erosions are covered with a grayish-white coating. When the chancre is located on the lips sometimes formed significant edema, due to whom lip sags, a chancre lasts longer than elsewhere. More often one hard chancre develops, less often - two or more. If a joins secondary infection, then erosion can deepen, at this formed ulcer With dirty gray necrotic raid.

On the language solid chancre usually happens single, arises more often in middle third. In addition to erosive and ulcerative forms, streets with folded language, with localization solid chancre along folds maybe arise slit-like the form. At location solid chancre on the backrest language due to significant infiltrate in basis chancre usually sharp speaks above environmental cloth, on the its surface has meat-red erosion. Note the absence inflammatory phenomena around chancre and his painlessness.

Solid chancre in areas gums It has view bright red smooth erosion,which in the form of a crescent surrounds one or two teeth. Ulcerative form hard chancre gums is very similar to banal ulceration and almost does not have any signs, characteristic for primary syphilomas. Diagnostics facilitates Availability bubo in submandibular areas.

At localization on the tonsil solid chancre maybe have one from three forms: ulcerative, angina-like (amygdalite) and combined — ulcerative on the angina-like background. The tonsil is affected on one side. With ulcerative amygdala increased dense Against this background, there is meat-red oval ulcer With canopies even edges. Mucous shell around ulcers hyperemic. Process accompanied painful sensations sometimes significant.

At angina-like chancre erosion or ulcer missing, available unilateral significant enlargement of the tonsil. It acquires a copper-red color, painless nenaya, dense. Process is different from sore throats one-sidedness defeat, absence pain and acute inflammatory hyperemia. General phenomena No, temperature body normal.

Chancre on the lips should be differentiated from simple vesicular lichen, with which in difference from syphilis rashes preceded burning or itch, erosion situated on the hyperemic, slightly edematous basis and It has micropolycyclic outlines. Except Togo, at bubble lichen erosive rashes precede bubbles, which never not arise in processchancre formation. Unlike hard chancre, herpetic erosions are almost always characterized by rapid onset and rapid epithelialization, in addition, herpes in difference from solid chancre often It has recurrent flow. Should take into account, what atlong existence herpetic erosion on the lip in her basis appears infiltrative seal, what reinforces resemblance erosion With primary syphiloma.

secondary period of syphilis begins after 6-7 weeks. after the appearance of solid chancre, when, against the background of symptoms characteristic of the primary period of syphilis (solid chancre, regional scleradenitis, polyadenitis), abundant roseolous-papular rash. The secondary period of syphilis lasts for 3-5 years and is accompanied by positive serological tests. A feature of the secondary period of syphilisis an undulating course, when periods of active manifestation of the disease are replaced periods hidden, asymptomatic currents disease, and duration everyonefrom these periods individual (in average on 1.5-2 months).

The active stage of the disease, which develops at the beginning of the secondary period of syphilis due to the generalization of the infection, is characterized by a large amount of roseolous papular, and sometimes pustular rashes, polyadenitis, scleradenitis, remnants hard chancre and is called - secondary fresh syphilis. By the end of the secondary fresh syphilis, a hard chancre is resolved, roseolous-papular rashes disappear,liquidated regional scleradenitis and polyadenitis.

Mucous shell cavities mouth is frequent place localization syphilides secondary period, and at secondary recurrent syphilis rashes in mouth may to be the only clinical manifestation illness. Nearly at half sick With phenomena secondary syphilis observed defeat mucous shells mouth in form roseolous and papular elements, pustular rashes on the mucous shell mouth arise extremely rarely.

roseolous rashes on the mucous shell mouth arise symmetrically on the temples, soft sky, tongue and tonsils. featureroseolous rashes in this areas is that they merge in solid foci defeat (erythematous angina). Struck region It has stagnant red color, sometimes with a copper tint, sharp borders. The mucous membrane in this the area is slightly swollen; patients feel awkward when swallowing, pain, but subjective Feel may and absent. Permission erythematous sore throats starts With central parts.

The most common manifestation of secondary syphilis on the oral mucosa are papular rashes. They can occur anywhere in the mucosa shells, but bowl on the tonsils, temples, soft sky, where often papules mergein solid foci defeat (papular angina), language, mucous shell cheeks, especially along the line of teeth closing, gums, etc. The type of papules depends on their duration. existence. at first papule - sharp limited Dark red hearth size up to 1 cm in diameter with a small infiltrate at the base. Some time later emerging in result what is happening inflammation exudate impregnates covering papule epithelium, and she is acquires very characteristic view.

The tertiary period of syphilis is not observed in all patients, even if they do not are being treated. It begins 4-6 years after the onset of the disease due to a change reactivity organism, sensitivity his to pale treponema and etc. and It has malignant flow. Tertiary period maybe continue decades characterized by the development of inflammatory infiltrates (gum and tubercles), prone to disintegration and often causing significant destructive, sometimes incompatible with life changes in organs and tissues. At the same time, rashes of tertiary syphilis do not contagious for surrounding, So how in them detachable missing pale treponema.

AT tertiary period syphilis on the mucous shell mouth may to appear gummas, gummy diffuse infiltration and tubercular rashes. At this mucous shell maybe to be the only place clinical manifestations diseases.

Gummy syphilis maybe localize in any mucosal site shells mouth. More often gummas formed on soft and solid sky and language. Usually gumma appears in the only number. at first formed painless node, which the gradually increases a then opens up. Rejected

gummy rod, after which a gummy ulcer is formed. This process lasts 3-4 months, sometimes accompanied insignificant subjective sensations. unopened gumma It has dense consistency smooth surface, mucous shell above the node is moderately inflamed, it has a stagnant red, sharply limited color. After branches rod gummy ulcer It has crater-shaped shape, dense the edges, painless bottom her covered granulations. Ulcer gradually heals With the formation of a stellate retracted scar. When localized in the sky at the place of the gumma often formed perforation, persisting after permissions process.

On the solid sky gumma usually situated on middle lines. Due to Togo, what mucous shell thin and intimately tied With periosteum sky, beginning gummy process very fast passes on the periosteum and bone. Infiltrate gummas fast breaks up and exposed bone, which necrotizesand sequestered arises message between cavities mouth and nose.

Treatment sick syphilis maybe to be started only after confirmation clinical diagnosis discovery pale treponem at primary and secondary syphilis or positive serological reactions. Under influence antisyphilitic treatment rashes fast disappear and already through 8-10 hour. after the start of penicillin therapy, pale treponemas are not found on the surface rashes. In this regard, patients with syphilis after 10-12 hours. after the start of treatment nicillin practically not contagious at household contact, a also at examination them doctors,in volume including dentists.

A dentist in his practice may encounter patients with tertiary syphilis, which the only manifestation of the disease may be gummy or tuberculate rashes on the mucous membrane of the mouth. Treatment of such patients should not begin with the introduction penicillin, as it will cause an exacerbation reaction, which will stimulate a rapid resorption syphilitic rashes what maybe lead to catastrophe, even to of deathsick, if such rashes are localized in vital important organs. it related With topics what at such treatment resorption infiltrate happen per 2-3 day , during which not the connective tissue replacing them will have time to form. For this reason , the treatment of patients tertiary syphilis should always start with iodine for 2-4 weeks, then enterhalf the course dose of the bismuth preparation and only then penicillin, after which the second half the course dose of the drug bismuth; the second and subsequent courses of treatment begin, how usually, those. With penicillin.

Dentist maybe meet With sick, which the moved tertiary or late congenital syphilis and at whom available perforation sky, requiring plastic surgery. It should be borne in mind that patients with syphilis after treatment 5 years are on the dispensary accounting, in flow this time at them determine curability syphilis. AT connections With this plastic operation so patients should be done after deregistration. If, however, there is a need for operations before this term, then operational intervention necessary conduct under protection of penicillin, in this case, the value of the total dose of the drug is determined collectively With venereologist, under observation whom located sick.

In the treatment of manifestations of syphilis in the oral cavity, complications may occur, related With application penicillin and drugs bismuth. Penicillin and his drugs can cause acute allergic drug-induced stomatitis, due to which necessary stop introduction penicillin, and candidiasis. Last thing complication at sick syphilis not requires mandatory cancellation penicillin. Complications from drugs bismuth are bismuth border, bismuth gingivitis and stomatitis.

Gonorrhea.

Gonorrhea is caused by Neisseria gonorrhoeae. It's highly contagious venereal disease characterized defeat not only urogenital tract. There are extragenital lesions: arthritis, oral lesions and throats. Percent recent manifestations sharp increased in connections With spread oral sex and homosexuality. On the lips at gonorrhea may to be ulcerative defeat, the gum becomes edematous, inflamed and resembles the picture is stingingly - necrotic gingivitis. Language, mucous cheeks may to be hyperemic and ulceration.

Viruses are most frequent cause infectious diseases (measles, epidemic mumps, rubella, flu, windmill smallpox, hepatitis, AIDS and etc.).

Another virus, for which parenteral route transfers also maybe to be basic, - this is virus immunodeficiency human (HIV). Virus HIV affects lymphocytes (T-helpers) and macrophages. With prolonged reproduction of the virus in body populations helpers is shrinking in affected macrophages declining production interleukin-I. Except immune

systems suffer other bodies and systems: nervous system, bodies digestion and breath, cardiovascularsystem , etc. Against the background of immunodeficiency , Kaposi 's sarcoma develops andopportunistic diseases (candidiasis, ulcerative necrotic stomatitis, cytomegalovirus infection, herpes, pneumocystosis).

Sources virus are sick and virus carriers. Available parenteral route of infection when using an infected virus blood with various surgical interventions, blood transfusion, etc., as well as sexual contact. Registered cases infections fetus in intrauterine period.

Diagnostics held main way serological method, through identifying antibodies With help enzyme immunoassay analysis.

Thus, patients who came to the reception with candidomycosis, relapses herpes, ulcerative necrotic lesions must be checked on the infection HIV. special danger is posed by patients at risk - homosexuals, prostitutes, drug addicts, patients who often received blood transfusions (hemophilia).

A dentist can become infected with a virus from a patient airborne by and during a digital examination, as well as the doctor himself during his manipulations maybe infect patient (hepatitis AT, AIDS).

Additionally necessary Mark, what about near 46 % patients on the reception can be carriers in the oral cavity of Streptococcus puogeus, Staphul. aureus. They are can cause the doctor to develop various respiratory diseases, as well as cause an inflammatory process in case of a hand injury with a drill or a stab wound. Considering that, that many of the staphylococci are antibiotic-resistant, the entry of these microbes maybe make it difficult process treatment.

Leprosy

Leprosy (leprosy) called leprosy mycobacteria Hansen and represents yourself chronic generalized infection developing predominantly in derivatives ectoderm and in bodies and fabrics, rich elements active mesenchyme. Leprosy leaks chronically progressive With periodical exacerbations (leprosy reactions), leading in the absence of rational treatment for severe disability and through a lot of years to of death.

Allocate tuberculoid and lepromatous types leprosy. emergence Togo or another type of disease related with fortune resistance organism human to leprosy infection, which determine With help lepromine skin samples. Mucosal lesions shells cavities mouth may arise only at lepromatous type, which the develops at persons With sharp reduced reactivity (lepromine try negative). At such sick on the skin, mucous shell mouth, internal bodies, on move nervous trunks arise lepromatous infiltrates With leprosy cells not able (in difference from tuberculoid type) destroy phagocytosed them sticks Hansen, that's why they freely multiply in these cells.

Leprous process on mucous shell mouth begins with infiltrative stage, then on the this background arise tubercular rashes which through some time ulcerate. Process ends scarring. Highly often on the mucous shell mouth at one and Togo same sick can observe elements, characteristic for all four stages development leprosy defeat mucous shells, t. e. infiltration tubercles, ulcers and scarring. Clinical painting leprosy lesions of the oral mucosa is characterized by polymorphism, each of four stages characterized described signs.

Leprosy changes mucous shells mouth begin With education superficial limited infiltrate, slightly rising above the surrounding mucous shell and having grayish white color, sometimes With dark blue plots. Then on the infiltrated plots arise tubercles various sizes - from millet grain to a cherry stone. At first, the tubercles are dense, then soften. They are located haphazardly and inclined to peripheral growth and merger. Tubercles mostly dull pink, sometimes grayish pink colors. Them surface usually shiny. More often Total tubercles arise on the solid and soft sky, language and lips. Through some time tubercles usually ulcerate. ulcers on the place tubercles at first small, bottom them bumpy, dirty gray white colors, the edges uneven, swollen, soft consistency. Sometimes ulcerative process distributed by on the bone the cloth, causing her meltdown. More often Total at this collapses alveolar edge jaws. AT subsequent ulcers scarred but scarring may develop without previous ulceration tubercles, when the tubercle or infiltrate of the oral mucosa are fibrosed. Scar formation is not yet means

recovery, for simultaneously may arise new infiltrates, tubercles and ulcers. Leprosy scarring on the mucous shell cavities mouth may to be round or in form stripes — radiant. Scarring smooth, shiny, white colors. formed scarring in dependencies from them sizes and places location may cause functional disorders. Especially often the soft palate is deformed and tongue, which the maybe shift a sometimes even disappear.

Leprosy defeat may arise and on the lips. At this very often a pronounced infiltrate is formed, accompanied by edema, which entails education leprosy elephantiasis. Lip compacted, becomes more thick, roller-shaped and sedentary. Leather lips and red border on bloom few different friend from friend. On the this background arise Good contoured tubercles.

Leprosy changes on the inner surface lips begin With diffuse erythema, then an infiltration occurs view bluish spots, covered with thickened epithelium. Against this background, sometimes bumps appear, which nearly not act above mucous shell. Leprosy tubercles on the lips may long time remain unchanged but sometimes they ulcerate. The emerging shallow painless ulcers located on the surfaces tubercles. Them detachable dries up in light yellow crusts.

Ulcerative defeat mucous shells lips always ends scarring with deformity of the lips, resulting in a thinner lip, the mouth opening may narrow down. If a same deep located tubercles fibrosis, then lip wrinkled, due to what finds it difficult speech, leather lips in these cases happens dotted furrows a red border lips becomes wrinkled. Leprosy changes mucous shells lips rarely reach before transitional folds.

Beloved localization leprosy rashes are gums on the topjaws from the side of the tongue in the area of the frontal, less often molars. Leprosy changes in the gums begin with the formation of an infiltrate. The gums seem to swell are made loose, red, sometimes cyanotic slightly bleed; gingival papillae swell up picture gingival the edges smoothed out. Highly often to this increased salivation joins. Soon the gingival mucosa becomes matte, on the her surfaces formed sores, which then scarred what leads to wrinkling of the gingival margin and turning it inward. The gums retract, the roots of the teeth exposed. Simultaneously with scarring in other areas of the gums, fresh infiltrate. characteristic painless process.

Leprosy changes on the mucous shell solid sky are happening next way: in those cases, when defeat starts on the front thirds at first noted infiltrative stage, infiltrate formed behind necks central incisors and comes before fangs. Plot infiltrated mucous shells on direction back narrows and more often ends at the beginning middle thirds solid sky, in result what formed triangle, basis facing forward, and the top back. Significantly less often infiltrate in the direction narrows back to a strip 2 cm wide and extends along the midline to soft sky. Infiltrate on the lateral surfaces solid sky observed extremely rarely. infiltrated plot greater part It has grayish red color and absolutely painless. Then on the infiltrated plots arise grayish white tubercles magnitude With millet corn, which subsequently superficially ulcerate.

On the soft sky defeat starts With hyperemia, passing in infiltrate dark coloring. Sometimes soft sky at first It has pale yellow color. tongueusually infiltrated, its size increases. On the infiltrated plots soft sky and tongue appear whitish gray, focally located tubercles various quantities — from millet grain before peas. Then tubercles break apart and formed sores. Sometimes individual sores merge, forming greater or lesser quantities solid ulcerative surfaces. The edges ulcers slightlyraised and undermined, bottom bumpy With grayish raid. Color them dirty grayish white. Due to ulcerative defeat usually collapses part or the whole tongue.The ulcerative process on the hard and soft palate ends with scarring. The emerging scarring have varied form: sometimes they round, but more oftenradiant or stellate. Scarring usually shiny, superficial, whitish colors. Cicatricial contraction on the solid sky near necks front teeth leads to gingival wrinkling and retraction her with exposure of the roots of her teeth. The edges retracted gums wrap up inside in side roots, tight hugging them. Scarring on the soft palate sometimes form an arc, the convex part facing forward. fibrosis in areas soft sky often causes persistent deformation, due to what soft sky maybe pull up up, narrowing input in nasopharynx. Sometimes soft sky absolutely is destroyed.

Beloved localization leprosy elements on the language is average line his backs, beginning from root and before tip. Language maybe infiltrate increase, thicken, in result what

his mobility finds it difficult and speech becomes obscure. On the infiltrated surfaces backrests language appear dense tubercles various quantities, but not more bob, With flat surface and wide basis. Surface tubercles shiny, covered whitish raid thanks to desquamation epithelium ("silver" language). Number tubercles may enlarge and may coalesce (leprosy glossitis), resulting in what on the back of the tongue are formed roller-shaped elevations with deep furrows between them.

tubercles on the language inclined to decay and ulceration. formed ulcers located superficially and have jagged, undermined, infiltrated edges. The bottom of the ulcers shallow, rough, covered with a grayish coating. In some cases, these ulcers merge, forming a continuous ulcerative surface, covered with a thin gray coating. Emerging on the place ulcers various forms superficial scarring usually have greyishwhite color and brilliant surface.

For the diagnosis of leprosy , bacterioscopic studies of scrapings from mucous shells nasal partitions, a also co bottom and edges leprosy ulcers, in whicheasily discover sticks Hansen.

Leprosy lesions of the oral mucosa may resemble tertiary syphilis and lupus erythematosus. From the manifestation of lupus erythematosus and collicative tuberculosis, leprosy rashes are characterized by greater density, the pre sence of pronounced disorders sensitivity and leprosy mycobacteria in ulcerated discharge elements.

Most effective antileprosy means are drugs sulfonic row, such, how diamino diphenyl sulfone (DS), avosulfone, dapsone, sulfetron (solusulfone, novotrope), which accept in flow long time. The less toxic drug thiocarbonylide has the same effect as sulfones. (derivative thiourea). Not lost meaning chaulmugrious oil and his derivatives, inin particular ethyl ethers and mugrol. In order to successfully treat patients with leprosy, always should apply combined therapy everyone famous antileprosymeans in combined With restorative drugs and physiotherapy.

Forecast at leprosy in recent decades in connections With introduction in practice treatment this disease of new, sufficiently effective drugs has become better. Main public prevention of leprosy is early detection and rapid isolation of patients with leprosy in special institutions - leper colonies, where patients are treated, live and work. At discovery sick or suspicious on the disease leprosythe doctor must immediately report this to the health authorities. Upon confirmation of the diagnosis leprosy sick With compliance measures precautions envisaged instruction People's Commissariat ways messages — People's Commissariat health care from 19/IV 1944 G., must to be delivered in leprosarium.

herpes

Lichen lichen simplex (syn. herpes simplex) is one of the most common diseases of the human oral mucosa. Lips and oral mucosa are favorite localization herpes. Virus herpes starts his development in cellular core. Once in the body and causing manifestations of a primary herpetic infection, it remains in body human, apparently in flow all life in latent able or causes relapses illness.

Herpes can occur in a person once in a lifetime, and may have recurrent flow. Relapses develop in different people at different times and are characterized by unequal intensity of the process. The occurrence of relapses is facilitated by many factors that reduce the body's defenses: cooling, overheating, pneumonia, tonsillitis, diseases of the gastrointestinal tract, menstruation, etc. More often herpetic infection appears in form local defeat, but sometimes she is acquires generalized character, more often at newborns.

Simple bubble lichen maybe arise at of people any age. Manifestationsherpes can be noted on the skin and mucous membranes. The disease begins withone or two, less often more limited foci of hyperemia, against which small, the size of a millet grain or a little more, bubbles are quickly formed. Quantityin bubbles maybe vary from 2-3 before 10-15 or more. bubbles are located groups, contain a clear liquid, then their contents become cloudy. Sometimes as a result of clusters liquids small bubbles merge in one or two bubble diameter before 1.5 cm. On the lips through 2-3 days liquid shrinks in yellowish gray peel.

Often bubbles open up With education erosion bright red colors With polycyclic outlines. On the oral mucosa, more pronounced inflammatory reaction, bubbles open up in first watch after appearance. erosion on the them place have wrong scalloped outlines and covered delicate fibrinous film. The process on the oral mucosa can be limited or widespread - herpetic stomatitis. After 8-12 days, and in in some cases, slower, epithelialization of erosion occurs. Eruptions of bubbles accompanied tingling. AT individual cases rashes accompanied strong

swelling of the surrounding tissue. The general condition, as a rule, does not suffer, however, some patients report malaise, muscle pain, chills. Body temperature may be subfebrile or rise before 38-39°C.

Variety simple herpes is recurrent herpes. Relapses occur with different frequency, at different times and regardless of the season. Some patients relapses are observed 3-4 times a month, the disease actually takes permanent flow. Recurrent herpes does not differ in clinical manifestations from simple herpes.

Acute herpetic stomatitis. Until recently in domestic literature on dentistry and pediatrics described two independent diseases: spicyaphthous and spicy herpetic stomatitis. Clinical and laboratory survey a large group of patients using the arsenal of modern virological, serological, cytological and immunofluorescent methods research earnestly showed clinical and etiological unity these diseases.

Received data allowed recommend single term and call the disease "acute herpetic stomatitis", based on the viral etiology of the disease. This stomatitis occupies one of the leading places in children's infectious diseases. pathology, meeting more often scarlet fever, measles, epidemic mumps and only a littleyielding wind smallpox

Spicy herpetic stomatitis among non-immune persons It has relatively high contagiousness. So, in preschool institutions and in hospital wards at epidemic outbreak maybe get sick before $^{3/4}$ _ _ children.

Broadcast infections going on obviously, contact and airborne way.

For development diseases It has meaning violation intact mucous shells and skin covers. A clear seasonality of the occurrence of the disease can not be identified. The highest prevalence of the disease among children aged 6 months to 3 years explained topics what at them disappear antibodies, received from mothers interplacentally, a also inadequate maturity systems specific immunity.

Spicy herpetic stomatitis, how and other infectious diseases, It has fiveperiods development: incubation, premonitory (catarrhal) periods developmentdiseases (rashes), extinction and clinical recovery (reconvalescence). AT dependencies from degree expressiveness general toxicosis and local manifestations in cavities mouthdisease maybe leak in light, middle and severe form.

From general symptoms characteristic hyperergic reaction With rise temperature body before four HS and above at severe form diseases, malaise, weakness, head pain,skin and muscular hyperesthesia, absence appetite pallor skin covers, nausea and vomiting of central origin, since the herpes simplex virus is encephalotropic. Already in the incubation and especially in the prodromal period, clearlycomes to light increase submandibular, a in heavy cases and cervical lymph nodes.

On the pique rise temperature arises hyperemia and puffiness mucous shellsoral cavity; on the inflamed mucous membrane of the lips, cheeks, tongue appear from 2-3 to several dozens close located friend to friend groups bubbles, which fast are opened. On the them place arise erosion With necrosis in center size 0.5—1 cm, veryreminiscent of aphthous elements. On the lips rashes fast covered crusts. Atthis defeat maybe localize not only in cavities mouth, but and on the skin face, especiallyoften this is observed in severe form of the disease. Due to the fact that the rash continue to occur for several days, during examinations you can see elements lesions at different stages of development. Reappearance of rashes accompanied by a deterioration in the general condition of the child, anxiety or adynamia and rise temperature body on the 1-2°C on comparison With previous for days. Compulsory a symptom of acute herpetic stomatitis is hypersalivation, saliva becomes viscous and viscous, noted smell iso mouth.

Already in the catarrhal period of the disease, pronounced gingivitis often occurs, which in the future, especially in severe form, acquires an erosive-ulcerative character. noted pronounced bleeding gums and mucous shells cavities mouth.

AT blood children With severe form diseases discover leukopenia, stab shift to the left, eosinophilia, single plasma cells, young forms of neutrophils.Sometimes in urine appears protein. AT saliva first determined shift pH in sour side, then - into alkaline, while interferon is usually absent in saliva, and the content lysozyme is markedly reduced. Humoral factors of the body's natural defenses, including including phagocytosis, in period swing diseases also sharp reduced.

The diagnosis of acute herpetic stomatitis is established on the basis of clinical patterns and

epidemiology of the disease. To clarify the diagnosis, it is recommended to perform cytological examination of material from herpetic erosions, which is stained according to Romanovsky-Giemsa to discover the so-called giant multinucleated cells,which characteristic for herpes.

A very promising method for the etiological diagnosis of the disease is the method immunofluorescence, which the allows get result in flow 2.5-3 h With momentfence material.

Treatment of lichen lichen simplex in adults is to lubricate the focus lesions 2-3 times a day with alcohol solutions of aniline dyes or ointments, containing antiviral substances (1-3% oxolinic, 3% octathionic, 2-5% tebrofenovaya, etc.). A good effect is given by leukocyte interferon, the solution of which applied to the affected area 6-7 times a day. In the treatment of recurrent herpes necessary apply funds, increasing protective strength organism (gamma globulin, pyrogenal and etc.). positive action renders herpetic polio vaccine, whichinjected intramuscularly at 0.1-0.2 ml with an interval of 2-3 days, for a course of 10 injections. Deoxyribonuclease gives a good effect, which administered parenterally by 10-50 mg 2 times in a week on the well 6-10 injections.

Tactics doctor at treatment children, sick sharp herpetic stomatitis, must determined degree gravity diseases and period her development. At middle gravity andinsevere cases of the disease , it is advisable to carry out general treatment together with the pediatrician. Considering, that these forms of the disease develop against the background of a significant decrease in the protective forces organism, appropriate apply means of stimulating immunity (ly-zozyme, prodigiosan, gamma globulin parenterally; methyluracil, pentoxyl, sodium nucleinate), at the same time , desensitizing therapy should be carried out (diphenhydramine, suprastin, pipolfen, calcium gluconate, etc.). Local treatment for acute herpetic stomatitis is antiviral therapy. Assign 0.25-1% oxolinic, 1-2% florenal, 1-2% tebrofen, four% heliomycin ointment, one% solution deoxyribonuclease, liniment helepin, mixture interferon With prodigiosan and others interferonogens. These drugs are used 3-4 times a day. Antivirals _necessary apply on the all mucosa shell, a not only on the affected plots, So as they provide both curative and preventive effects. Once a day oral cavity recommended handle 0.1— 0.5% solution proteolytic enzymes (trypsin, chemotrypsin, pancreatin and etc.), which contribute dissolution necrotic fabrics.

AT period fading disease basic meaning attach application weak antiseptics and keratoplastic funds. Good ones results give applications oil solutions vitamin A BUT, oils wild rose, carotolina, ointment and jelly solcoseryl, ointments With methyluracil, livian, levovy-nizol. AT quality antimicrobial funds can apply solutions furatsilina, ethacridine lactate, ectericide, aetonia and etc. Sick must accept liquid or semi-liquid writing, not annoying inflamed mucosa shell, receive sufficient amount liquids. Before reception food necessary anesthetize mucosa shell of the mouth 5% anesthetic emulsion.

AT conclusion should Mark, what spicy herpetic stomatitis, flowing in any form, is contagious disease, in all cases required Attention co sides pediatrician and dentist for Togo to provide comprehensive treatment, exclude contact sick co healthy and spend preventive Events in children's collectives. AT children's institutions, especially in nurseries, should not be allowed to work with children employees in period manifestations herpes, regardless from his localization. At identifying in children's institution child, sick sharp herpetic stomatitis, he is not allowed to attend a kindergarten, even if disease leaks in very light form.

Shingles. This disease is caused by a virus identical in their antigenic properties virus wind smallpox. This virus is different neurodermatotropism With prevailing action on the central and peripheral nervous system. Disease often develops on the background sharp weakening protective properties organism, often how complication pneumonia, diseases blood and others exhausting diseases.

Disease starts With pain on move affected peripheral nerves always With one sides. On the face and mucous shell mouth appear paresthesia and pain on move one or two branches trigeminal or facial nerve. Then appears erythema in form spots, sometimes merging in stripes, where formed groups small bubbles, filled serous or hemorrhagic content. rashes, sharp limited, unilateral, come to light on move affected nerves. On the mucous shell cavities mouth these bubbles fast are opened, forming erosion With scalloped outlines.

The entire thickness of the cheek in the affected area may be edematous and infiltrated, submandibular lymphatic nodes increased painful. At defeat motor and sensitive

fibers facial nerve sometimes develops Ramsay Hunt syndrome, including herpes zoster, facial paralysis nerve and ear pain. The disease lasts 3-6 weeks. and spontaneously resolved. Disease accompanied pains neuralgic character, which may persist and after liquidation rashes.

AT diagnostic respect important meaning It has one-sidedness defeat, painful syndrome, localization rashes strictly in zone innervation affected nerve, absence relapses. Last thing distinguishes herpes zoster lichen from Herpes simplex recurrences.

Treatment is in appointment antibiotics wide spectrum actions, salicylates, vitamins AT and At $_{12}$, deoxyribonuclease, interferon, analgesics and others Mucous shell mouth handle antiviral ointments (0.25-0.5% oxolinic, 0.5-2% tebrofen, 1-2% florenal) or interferon solution (32 U in 1 ml). During the period of resolution of rashes are shown keratoplastic funds: oil wild rose, sea buckthorn, carotenoline, ointments, containing vitamin BUT.

herpetic angina. Disease called enterovirus coxsackie groups BUT, starts acute: With rise temperature body, myalgic pain, general ailments. AT rear department mouth: on the soft sky, front temples, tonsils and back of the throat –appear painful grouped and single vesicles filled serous or hemorrhagic content. AT Subsequently, part of the vesicles disappears, others open, forming erosions. merger small erosion leads to the formation of eroded areas of different sizes with scalloped outlines. Some from erosion may remind aphthae. erosion not painful, epithelialize slowly, sometimes in flow 2-3 weeks Described cases diseases members one families and even epidemic outbreaks.

Treatment consists in symptomatic general therapy and local applicationin first 2-3 days antiviral, a in further keratoplastic funds.

Frequent rinsing and lubrication slow down process epithelization erosion.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15 min.

PRACTICAL OCCUPATION No. eleven.

Topic: Microflora in prosthetics and implantation of teeth. The study of adhesion and colonization of oral bacteria on dental materials. Diagnosticsperi-implantitis and their prevention.

Educational goal:

explore meaning resident microflora cavities mouth in development complications of dental implantation and principles of prevention of post-implantation complications inflammatory character.

Plan lessons:

6. Representatives of what oral biotopes are the most common pathogenspost-implantation complications?
7. Ways infections zones implantation, related With contamination bone lodgeimplant and seam lines.
8. Pathogenesis and clinical forms post-implantation complications inflammatorycharacter.
9. Sampling of material for research in peri-implantitis and osteomyelitis. 10.

Prevention post-implantation complications inflammatory character.

Independent Work students:

1. Take into account results crops microflora mucous shells cavities mouth before and after use self-adhesive films DEPLEN-DENT and bring in to the protocol.
2. Microscopy smears from pure cultures anaerobic bacteria, dedicated atperi-implants:
a) convertibles
b) peptostreptococcic)
fusobacteria

3. Decor protocol research.

Table. Fence researched material from prosthetic lodge.

researched material	Description results microscopy	Picture

EQUIPMENT

3. Equipment for cooking smear and coloring on Gram.
4. Tables.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 70 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------15min

PRACTICAL OCCUPATION No. 12.

MODULE No. 4.

No. СТОМ-21-ИН

Federal State Budgetary Educational Institution of Higher Education
NORTH OSSETIAN STATE MEDICAL ACADEMY
Ministry of Health of the Russian Federation

Department of microbiology

COLLECTION METHODOLOGICAL DEVELOPMENT ON MICROBIOLOGY, VIROLOGY, IMMUNOLOGY-MICROBIOLOGY MOUTH FOR DENTAL FACULTYFOR TEACHER

Spring semester

Vladikavkaz

Authors: Associate Professor of the Department of Microbiology, FSBEI NOSMA of Ministry of Health Russia., Ph.D. Chertkoeva M.G., Assistant Khabieva B.A.

The main purpose of the developments is methodological assistance to teachers to to each practical occupation in III semester. Directions drawn up incompliance With Federal public educational standard Supreme and vocational education.

REVIEWERS:
L.V. Bibaeva – MD, Professor, head department biology and histology FGBOUIN SOGMA Ministry of Health Russia.

A.R. Kusova- MD, Professor, head department of hygiene and physical education FGBOU VO SOGMA Ministry of Health Russia.

PRACTICAL OCCUPATION No. 1.

Topic: Microscopic research method. Equipment and rules of work in bacteriological laboratory. Light microscopy. Immersion system microscope. Morphology microbes. Simple and complex ways coloring drugs. Structure Features eu- and prokaryotic cells.

Educational goal:

6. Familiarization with the rules work *in* microbiologicallaboratories.
7. To study the taxonomy and classification of microorganisms: morphological peculiarities microorganisms and simple coloring.
8. Explore morphology individual representatives bacteria.
9. master technique microscopy.
10. master simple method coloring microorganisms.

Plan lessons:

9. Familiarity with the rules of work and the basics of safety inmicrobiological laboratories.
10. Device and equipment microbiological laboratories, mode work andappointment.
11. Classification bacteria.
12. Morphology of bacteria, methods of study (light, dark-field, phase contrast, electron microscopy).
13. Stages cooking smear.
14. Simple method coloring bacteria.
15. Cooking smears from culture staphylococcus and intestinal sticks, coloringsimple method.
16. Demonstration drugs from micrococci, diplococci, tetracoccus, sarcin,staphylococcus, streptococci, intestinal sticks, bacillus, vibrios.

Independent Work students

3. Smear preparation and coloring by a simple method (under the guidance of teacher).
4. Mastering the technique of microscopy. microscopic studymorphology bacteria:
6. View demonstration smear from clean culture staphylococcus (Staphylococcus aureus). Coloring gentian violet.
7. View demonstration smear from clean culture intestinalsticks (E. coli). Coloring - water fuchsin.
8. Decor protocol research.

EQUIPMENT

1. Set for bacteriological research:Tripod- 8 pcs.
   Tweezers - 8 pcs. Bacteriological
   loop-8 pcs.Tray With stand - on
   8pcs
   glass slidesAlcohol
   lamp -8 pcs.
   Bottle With physical solution -8pcs.
2. Kit colors: Methylene blue -
   8 pcs.Water magenta - 8 pcs.
3. Tubes with the growth of cultures S.aureus -8

pcs.test tubes With growth cultures E.coli -
eight PCS.

4. Microscopes - 8 pcs.
   Immersion oil - four PCS.
5. Demo micropreparations: sarcinas, streptococci, diplococci, intestinal wand, actinomycetes, staphylococci, spirochetes, vibrios, anthracoid.

6.Tables.

INFORMATIONAL MATERIL ON THEME

REGULATIONS WORKS AT MICROBIOLOGICAL LABORATORIES.

one . Work in microbiological laboratories held With contagiousmaterial, what requires special discipline and thoroughness in work.

2.Student must be in laboratories in dressing gown and cap. 3.Material forwork taken on duty on group at laboratory assistant departments and resounds students only With permissions teacher. 4.When implementation of practical classes, students obliged fulfill instructions teacher.

5.All items, which were in work, must to be disinfected. 6.Ifstudent will smash test tube With microbes, he must to report about this teacher and neutralize working place.

7. Each student receives a microscope, which is assigned to him.8.Wo time fulfillment practical exercises on the table should not to be none strangers items.

9.Students obliged lead in order his working place; pass duty officer materials and microscope; sign album With sketches. ten. ten. Wash uparms after work.

4. Technique cooking smears.

Smears are prepared on defatted glass slides, having previously outlined with a pencil on the glass, the place of the future smear on the opposite side of the subject glass. When bacteria grow on a liquid nutrient medium, the material is taken sterile bacterial loop, applied to glass and rubbed over the outlined area. AT case growth bacteria on the dense nutritional environment, on the subject glass previously inflict loop drop water and material rubbed.

5. bacterial loop before using With remaining culture sterilizein the flame of a burner. The prepared smear is dried in air or held high aboveflame spirit lamps.

6. After this a drug fix, for what smear side, where No material, three times carry out through middle flame burners. Fixation allows kill microbes, attach them to the glass and finally, the killed microbes are stained better than alive.

2. Technique simple methods coloring

The staining of bacteria aims to make them sharply different from the background, which allowsexamine their morphology and structure under a microscope. used in microbiology simple and complex staining methods.

At SIMPLE WAY coloring, smear stain any one dye, for example, aqueous fuchsin (2-3 minutes) or methylene blue (2-3 minutes), washed water, dry and microscopic.

3. Technique microscopy

AT connections With very small dimensions bacteria the study them morphology Maybe only at big magnification, achieved at help immersion oils, which allows you to create a single system between the glass slide and special, x 90- multiple (With black stripe) lens.

At microscopy painted objects necessary create bright lighting With help concave mirrors, raised condenser and fully open aperture.

A drop of immersion oil is applied to the area of the smear on the glass lying on the table. The glass is then transferred to the microscope stage. The immersion lens is immersed in oil carefully, under the control of the eye until the lens is in direct contact with the oil. Then the lens

is raised without removing it from the oil drop and looking into the eyepiece to find the object research ("field of view"). A clear image of the drug is achieved by adjusting first with a macro screw (for detecting an object), and then with a micro screw for adjustment sharpness Images.

Morphology major forms bacteria

known four main forms bacteria:

7. cocci - microbes rounded forms, having in diameter 1-2c. They are different among themselves according to the mutual arrangement of individual cells, which depends on the method them division. If a on graduation division cocci are separated on the individual balloons, are obtained single cells cocci -Micrococcus.

8. Group from two cocci wears title diplococcus -Diplococcus (meningococcus, gonococcus have resemblance With beans, and lanceolate form - Pneumococcus).

9. If the division of cocci occurs in only one direction and the resulting cocci are not separated, then a thread of balls is obtained in the form of a chain, more or less long in dependencies from number cocci- Streptococcus.

10. When dividing in two mutually perpendicular directions, combinations arise on four coccus-Tetracoccus.

11. If division occurs in three mutually perpendicular directions, cocci connect in form packages (cubes) and get title - Sarcina.

12. Sharing in various directions without special correctness cocci form disorderly clusters cells, reminiscent of grape bunches, why they and got the name Staphylococcus.

rod-shaped microorganisms presented most numerous and diverse group bacteria. AT classification rod-shaped forms received name bacilli and clostridia those sticks, which able formcontroversy, a sticks incapable to spore formation called bacteria. Rod-shaped forms differ in size, location - singly, in pairs, chain, randomly and at an angle. The outline of the ends - rounded, chopped off, thickened, pointed.

Collection forms - *spirilla* and spirochetes having view corkscrew tortuous cells. Pathogenic spirillums include the causative agent of sodoku (rat bite disease). To tortuous also relate campylobacter, having curves how at wings flying seagulls.

Spirochetes — thin, long, curved (spiral forms) bacteria, differing from spirilla in mobility due to flexion changes cells. Spirochetes are represented by three genera pathogenic for humans: Treponema, Borrelia, Leptospira.

Methods diagnostics infectious diseases

3. *The microscopic method* consists in the preparation of preparations (native or painted simple or complex methods) from researched material and them microscopy With application various species microscopic technology (light, darkfield, phase contrast, electronic). AT bacteriology microscopic method received title bacterioscopic, in virology — viroscopy.

4. *cultural method* is in sowing researched material on the artificial nutrient media for the purpose of isolating and identifying a pure culture pathogens. AT bacteriology cultural method received title bacteriological, a in virology - virological.

6. *The biological method* (experimental or bioassay) is to infect test material of sensitive or other biological objects (chicken embryos, culture cells). His use for allocation clean culture pathogen, definitions type toxin, definition activity antimicrobial chemotherapy drugs.

7. *Serological method* - consists in determining the titer of antibodies in serum the patient's blood, less often - in the detection of microbial antigen in the test material. FROMthis reactions are used for the purpose immunity.

8. *The allergic method* consists in identifying an infectious allergy (HRT) to diagnostic microbial a drug — allergen. FROM this purpose put skin allergic samples from relevant allergens.

An object study medical microbiological laboratories — pathogenic biological agents are microorganisms pathogenic for humans (viruses, bacteria, mushrooms, protozoa). AT compliance With types microorganisms allocate: bacteriological, virological, mycological, protozoological laboratories. Regulation conditions work With pathogens infectious diseases is produced in accordance with the degree of danger of microorganisms for person. Allocate four groups pathogens.

Group 1: pathogens of especially dangerous infections: plague, smallpox, fevers Lassa, Ebola.

Group 2: exciters highly contagious bacterial fungal and viral infections: anthrax, cholera, fever, typhus, rabies.

Group 3: pathogens bacterial fungal, viral and protozoan nosological forms (whooping cough, malaria, polio, leishmaniasis).

Group four: pathogens bacterial viral, fungal diseases (pseudomonal infection, amoebiasis, aspergillosis).

Microbiological laboratories work with PBA with highly dangerous pathogens infections (group 1 and 2). The special mode is maximally isolated by the individual and public risk.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent job ---------- 30 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ---------- 10 min.
5. Control final level knowledge and exercise on the house ----------10min

PRACTICAL EXERCISE No. 2.

Topic: Bacteriological method research. Physiology bacteria. Nutrients environment. Them classification, ways cooking, sterilization. Technique crops material on nutritious environment.

Educational goal:

1. Master the bacteriological method for diagnosing infectious diseases. 2. Explore types nutrition bacteria, principles cultivation of microorganisms classification nutritional avg.
3. Explore methodology receiving pure cultures bacteria from researched material.
4. Explore methods sterilization (physical, mechanical, chemical).
5. Explore methods control efficiency sterilization.

Plan lessons:

15. Nutrition of bacteria: types, mechanisms of nutrient entry into microbial cell.
16. Principles cultivation microorganisms.
17. Bacteriological method diagnostics infectious diseases.
18. Nutrient media: requirements for nutrient media ;classification, compound, cooking.
19. Demonstration nutritional avg.
20. Sowing researched material (suspended microorganisms) on the MPA method Drygalsky (Stage 1).
21. Methods sterilization: physical, chemical, biological, mechanical.
22. Device and application ovens Pasteur autoclave, apparatus Koch.
23. Sterilization various medicinal funds in dependencies from them nature,forms, lability to physical factor.
24. Control quality sterilization.
25. concept about asepsis, antiseptic and disinfection.
26. Antiseptics and disinfectants.
27. Principles control quality disinfection.
28. Demonstration antiseptic and disinfectants funds.

Independent Work students:

4. Sowing test material on method Drygalsky.
5. Familiarization With cooking nutritional avg.

6. Spend and take into account results experience on definition actions high temperature (80°C) into spore-forming (anthracoid) and asporogenic (intestinal wand and staphylococcus) microorganisms.
    - Fill protocol on form:

Accounting growth culture	Staphylococcus aureus	intestinal wand	Anthracoid
before warming up			
after warming up			

Vegetative forms of pathogenic microorganisms die at 50-60 ^{0} C during 30 minutes, and at a temperature of 70 ^{0} C for 5-10 minutes. Bacterial spores have greater resistance to high temperatures, due to the content in them water in related state, big content salts calcium, lipids and density, layering shells. Consequently, staphylococcus aureus and intestinal wand after warming up are dying a disputes anthracoid survive. it and necessary take account of in evaluation seeding results.

- Fill on one's own table:

No.	Way sterilization	Apparatus	Reliability	sterilizable material
one.	Sterilization in flame			
2.	Plasma Sterilization			
3.	Dry heat			
four.	Ferry under pressure			
5.	Fluid ferry			
6.	Tyndalization			
7.	Filtration			
eight.	Physical factors (UFL, gamma rays, ultrasound)			
9.	Gas sterilization			
ten.	Pasteurization			

EQUIPMENT

1. Set for bacteriological researchTripod- 8 pcs.
   Tweezers - 8 pcs. Bacteriological
   loop-8 pcs.Tray With stand - on
   8pcs
   glass slidesspirit
   lamp -8pcs
   Bottle with physical
solution - 8 pcs.2. Sowing on
Drygalsky:
   cups With MPA 3 pcs.-4 set
   Test tubes with a suspension of
   microorganisms - 4 pcs.Sterile spatulas - 4 pcs.
3. Demonstration: nutrient media: MPA, KA, Endo medium, Kitt medium
   Tarozzi, agar-agar, environments gissa, JSA, SCHA, Wednesday bismuth
   sulfite, MPB.

4. tables

INFORMATIONAL MATERIL ON THEME

Microbiological research is carried out in order to isolate pure cultures microorganisms, cultivation and study them properties. It necessary at diagnostics infectious diseases, for definitions specific accessories germs, in research work, for receiving products vital activity microbes (toxins, antibiotics, vaccines, etc.). To grow microorganisms in artificial conditions necessary special substrates — nutritious environment. They are are basis microbiological work and determine results Total research. environments must create optimal terms for vital activity microbes.

REQUIREMENTS PRESENT To WEDNESDAY:

8. Must be nutritious, i.e. contain in an easily digestible form all the substances necessary for satisfaction food and energy needs microorganisms.
9. Have an optimal concentration of hydrogen ions.10. Be isotonic for microbial cells.
11. Be sterile.12. Be wet.
13. Possess a certain redox potential.14. Be possible unified.

Need in nutritional substances and properties environments at different species microorganisms is not the same. it rules out possibility creation universal environment. Except Togo, on the choice of one or the other environments affect goals research.

Group classification	Class	Examples
Composition	***Simple***	*Liquid — MPB, peptone water Dense — MPA*
	Complex	*Liquid — sugar bouillon Dense — sugar agar, blood agar*
Origin	***natural***	*Milk, folded serum, slice raw potatoes*
	artificial	*Milky salt agar Serum agar Ascites agarBlood agar*
	Synthetic	*Wednesday Needle, Wednesday 199*
By appointment	***selective (elective)*** *-for staphylococcus: -for gram(-) cocci anddiphtheroids: -for enterobacteria: -for cholera vibrio: -for lactobacilli and mushrooms*	*Milk-salt agar, bile-acid- salt agar Serum media Media with tellurium salts Media with bile salts Peptone broth and alkaline agarTomato agar, rice agar, agar Saburo*
	Differential-diagnostic Universal enrichment media Preservative	*endo, Ploskireva, Levin, Ressel,gissa MPB, MPA, blood agar Wednesday Muller*

		environments With glycerin
By consistency	***Liquid*** ***semi-liquid*** ***Dense***	*MPB, peptonic water, sugarBCH* *MPJele, gelatinousMPA,* *blood agar*

STERILIZATION

Sterilization is deposition, t. e. complete release objects environmentalenvironments from microorganisms and their dispute.

Sterilization produce various ways:

10. Physical (impact high temperature, UV rays, enhanced pressure, steam, gamma rays, ultrasound).

11. Chemical (usage various disinfectants, antiseptics).

12. biological (application antibiotics).

13. Mechanical (filtration).

AT laboratory practice usually apply physical ways sterilization.

The possibility and feasibility of using one or another method of sterilization due to the characteristics of the material to be sterilized, its physical and chemical properties.

To *physical* ways sterilization can attributed calcination in flame, dry heat sterilization in a Pasteur oven, boiling, fluid steam sterilization in apparatus Koch, ferry under pressure in autoclave, tyndalization, pasteurization sterilization UFL, ultrasound.

Mechanical sterilization carried out filtering With help bacterial filters made from various finely porous materials, pores filters should be fine enough to provide mechanical delay bacteria. This method sterilizes nutrient media containing protein, serum, antibiotics; separate bacteria from viruses phages, exotoxins.

AT microbiological practice use asbestos filters Seitz, membrane Chamberlain filters (candles) and Berkefeld.

a) *Seitz filters* — discs from mixtures asbestos With cellulose, thickness them 3-5mm, diameter 35-140mm;

b) *membrane* filters - made of nitrocellulose, 0.1 mm thick and 35 mm in diameter. AT dependencies from size pores are 1,2,3,4,5;

c) *Chamberlain and Berkefeld candles* - hollow cylinders closed at one end, cook them from kaolin With sand admixture and quartz.

Chemical ways sterilization apply limited but they serve for prevention of bacterial contamination of nutrient media and immunobiological drugs (vaccines and sera).

biological sterilization founded on the application antibiotics, sometimes phages.

Disinfection — usage chemical substances (phenol, lysol, chloramine, peroxide hydrogen, corrosive sublimate, alcohol, and t. e.) for destruction pathogenic bacteria in spent pathological material.

Systematization appliances, processes processing and funds for disinfection andsterilization

Classification tools	Main typestools	The nature of processing and typesimpacts
critical - penetrate in sterilefabrics or vessels	All invasive surgical tools that havecontact with blood-supplied fabrics, scalpels, needlessyringes, implants,burs, root needles,excavators, probes, trowels.	**Sterilization** - virucidal, sporicidal, tuberculocidal,bactericidal impact. **long exposure:** gamma- rays, plasma, continuous gas and chemical sterilization, autoclaving (2 atm. 15 min),dry heat (maximum mode, 2 hours)
Semi -critical - come into contact with mucous shells (perexcept for som edental tools, listed above)	flexible endoscopes, catheters, toolssimilar to flexible endoscopes, mirrors, crowns, tips turbines, a also prints(casts) teeth.	**Disinfection high level** -virucidal, sporicidal, tuberculocidal, bactericidal impact. **Short exposure:**gamma rays, plasma, short-term gas and chemical sterilization, autoclaving (1-1.5 atm. fifteen min), dry heat.
	Thermometers for measuring mucosal temperature shells, baths for hydrotherapy.	**Medium level disinfection:** virucidal, tuberculocidal,bactericidal impact.
	Ultrasonic baths and UV lampsdentists physiotherapy tools , spoons casts.	**Means for chemical disinfection with** indication of labeling tuberculocidalactivity.
Non-critical come into contact withintact skin	Thermometers for measuring skin temperature covers, stethoscopes, cuff devices for measurements pressure, desktop appliances and etc.	**Disinfection level:** bactericidal effects. **Funds for chemical disinfection** without instructions on the presence marking tuberculocidal activity.

TIMELINE

1. Definition baseline knowledge ----------30 min.
2. Independent work ---------- 30 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------10 min.

PRACTICAL OCCUPATION No. 3.

Topic: Ways allocation and identification pure cultures aerobic bacteria. The study enzymatic activity, factors virulence and sensitivity to antibiotics dedicated cultures. Peculiarities transportation material and allocation pure cultures anaerobic bacteria.Cultural and pathogenic properties mushrooms.

test control.

Educational goal:

14.master methods allocation pure cultures aerobes.
15. Explore types breathing bacteria, ways creation conditions anaerobiosis. Mastermethods isolation of pure cultures of anaerobes.
16. Explore mushrooms - pathogens mycoses and mycological method research

Plan lessons:

12. Types breathing bacteria.
13. Ways creation conditions anaerobiosis.
14. Methods allocation clean culture aerobes and anaerobes.
15. Sowing soil talkers on the Wednesday Kitta Tarozzi.
16. Nutrients environments for anaerobes, methods cultivation and selection cleanculture anaerobes.
17. The study cultural properties bacteria.
18. The study colonies, grown on the cups, sown Drygalsky's method .
19. Sowing microorganisms from studied colonies on the oblique agar for receivingclean culture (stage 2).
20. Demonstration pigment formation bacteria.
21. Demonstration character growth bacteria on the dense and liquid nutritional environments.
22. Change module.

Independent Work students

17.Completion 1st stage bacteriological method. The study cultural properties bacteria.
18.From grown colonies on the MPA cook smear, paint on Gram.
19.Sowing from researched isolated colonies on the oblique agar for accumulation clean culture.
20.Demonstration technology anaerobic cultivation and Wednesdays for anaerobes: tall column of agar, Kitt-Tarozzi medium, thioglycol, Stuart. Demonstration microaerostat. Methods: Fortner, Weinberg.
21.Cooking smears from yeast mushrooms, color them with a simple method (methylene blue) and microscoping.
22.Preparation and microscopy native preparations from cultures moldy mushrooms.
23.Viewing and sketching demo preparations: a) actinomycetes, painted according to Gram;
b) native preparations from cultures of mold fungi (mucor, aspergillus, penicillium);
in) yeast mushrooms, painted methylene blue;
24.Sowing material With fingers hands on the cup With MPA (method prints).25. Sowing discharge from the nose and pharynx on the MPA.

EQUIPMENT

1. Swab kit:Tripod- 8 pcs.
   Tweezers - 8 pcs. Bacteriological
   loop-8 pcs.Tray With stand - on

8pcs
glass slidesAlcohol
lamp -8 pcs.
Bottle With physical solution -8pcs.

2. Paint set: By
Gram– eight
PCS.

3.microscopes - eight PCS.
Immersion oil - four PCS.

4. 3 cups with colony growth on MPA-4 pcs.

5.Slanted agar for reseeding - 8 pcs.

6. Demonstration of anaerobic culture techniques and media for anaerobes:
high column of agar, Kitt-Tarozzi medium, thioglycol, Stuart.

7.Demonstration of a microanaerostat.

8.Demonstration pigment formation bacteria.

9.Demonstration cultural properties on the liquid and densenutritional environments.

eight. Tables.

INFORMATIONAL MATERIL ON THEME

Breath bacteria. Classification bacteria on type breathing.

Essence breathing at microorganisms — receiving energy, emerging in process direct biological oxidation substances oxygen or through substrate dehydrogenation. The accumulation of energy occurs in special structures bacteria, called mesosomes.

Bacteria are classified according to their oxygen requirements. main groups:

1. Obligate (strict) aerobes - microorganisms that grow and multiply only in presence oxygen. For example: Vibrio cholerae Pseudomonas aeriqinoza.

5. Obligate anaerobes are microorganisms that grow and reproduce only without access oxygen. For example: Clostridum botulinum, Clostridium te tani.

6. Facultative anaerobes are microorganisms that can grow and multiply both in the presence of oxygen and in anoxic conditions. For example: Escherichia coli, Salmonella typhi.

7. microaerophiles bacteria — microorganisms, which better are growing and thrive in high $CO2$ and low oxygen levels. For example: Helicobacter pylori, Campylobacter coli.

Methods cultivation anaerobes

Ways creation anaerobic conditions a) mechanical - removal (pumping out)air from the anaerostat using vacuum suction. Then the anaerostat is filled gas mixture which consists from 80% nitrogen, ten% hydrogen and ten% carbon dioxide gas;

b) chemical — absorption oxygen per check chemical substances (alkalinesolution pyrogallol, bicarbonate soda);

c) biological (method Fortner) — a joint cultivation anaerobes and aerobes. At the same time, one Petri dish with a dense nutrient medium (more often used Wednesday Zeissler) sow culture anaerobes, on the another — culture aerobes, able vigorously absorb oxygen. AT quality aerobes use culture miraculous sticks (Serratia marcescens). The edges cups Petri is waxed;

d) physical and chemical - inoculation of the test material on special media for anaerobes, for example, Kitt-Tarozzi and Wilson-Blair media (iron sulfite agar). The media are regenerated before inoculation (boil in a water bath for 15 minutes) to removal oxygen.

Compound environments Kitta-Tarozzi:

-pieces liver - for adsorption oxygen;

-one% glucose — for implementation anaerobic glycolysis;

- semi-liquid agar — not admits oxygen in thickness environment.

Receipt clean culture anaerobes

1. Method Weinberg (method dilution)

To obtain isolated colonies of anaerobes from the Kitt-Tarozzi medium with growth anaerobic bacteria culture is taken with a Pasteur pipette with a sealed end and successively lowered this pipette at first in 3 test tubes With physiological solution, and then - 3 test tubes with melted semi-liquid sugar MPA. After temperature control at 37 ^{0} C in recent observed

growth isolated colonies anaerobes.

2. Method Peretz.

One of the last Weinberg dilutions in semi-liquid agar is poured into the lid cups petri and close her bottom So, to delete air. The edges cups petri paraffin. Sowing researched material on the Wednesday Zeissler sectors With subsequent cultivation in anaerostat.

Mushrooms (Fungi, Mycetes) are a heterogeneous group of eukaryotic microorganisms. Mushrooms have nucleus With nuclear shell, cytoplasm With organelles cyto- plasmatic membrane (which contains phospholipids and sterols) and powerful cellular wall consisting from glucan, cellulose, chitin, squirrel, lipids and others Mushrooms consist of long thin filaments (hyphae) woven into a mycelium, or mycelium. Hyphae of lower fungi - phycomycetes - do not have partitions. In higher mushrooms - eumycetes — hyphae divided partitions; them mycelium multicellular. Mushrooms reproduce by spores, sexually, asexually, and vegetatively (budding or fragmentation gif). Mushrooms, breeding sexual and asexual by, relate to perfect. imperfect called mushrooms, at which missing or more not described sexual path breeding. asexual reproduction carried out at mushrooms With help endogenous dispute, maturing inside round structures - sporangia, and exogenous spores - conidia, formed at the tips fruitful hyphae.

Mushrooms can divide on the 7 classes: chytridiomycetes, hyphochytridiomycetes, oomycetes, zygomycetes, ascomycetes, basidiomycetes, deuteromycetes. overwhelmingmajority mushrooms, defiant diseases at human (mycoses), relate toimperfect mushrooms. For diagnostics mycoses may to be used microscopic (cultural), allergic, serological, biological and histological research methods. Material for research can be pus, sputum, affected hair, nails, scales skin, punctates bone brain, lymphatic nodes, internal organs, blood, bile, feces, tissue biopsyand t. P. For coloring smears more often Total use methods Grama, Ziel-Nielsen, Romanovsky-Giemsa

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 30 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------10 min.

PRACTICAL OCCUPATION No. four.

Topic: Bacteriophage. Genetics bacteria. Molecular genetic method diagnostics. Structure and reproduction bacteriophages. Them medical meaning. Heredity and variability in bacteria. The polymerase chain reaction and its application .

.

Educational goal:

1. Explore structure and morphology bacteriophages.
2. Explore heredity and variability at bacteria.

Plan lessons:

1. Morphology and structure of bacteriophages, their practical application in medicine.
2. Kinds heredity bacteria.

Independent Work students:

- The study demonstrations phenomenon bacteriophage on the dense and liquid nutritional environments.
- The study demonstrations intracellular inclusions (body Babesha-Negri).

EQUIPMENT

1. Demonstration phage typing.
2. Demonstration phenomenon bacteriophage on the liquid and dense
   a. nutritional environments.
3. Demonstration Wednesdays for cultivation cultures fabrics: environments Hanks,

199,Needle.

INFORMATIONAL MATERIL ON THEME

bacteriophages differ on chemical structure, type nucleic acid, morphology and nature of interaction with bacteria. Size of bacterial viruses in hundreds and thousands of times less microbial cells.

A typical phage particle (virion) consists of a head and a tail. Tail length is usually2-4 times the diameter of the head. The head contains genetic material - single stranded or double stranded RNA or DNA With enzyme transcriptase in inactive state, surrounded protein or lipoprotein shell - ***capsid*** , preserving genome outside cells.

Nucleic acid and capsid together make up the nucleocapsid. Bacteriophages can have icosahedral capsid, assembled from sets copies one or two specific proteins. Usually angles are made up of pentamers squirrel, and support each side from hexamers Togo same or similar squirrel. More Togo, phages on form may to be spherical, lemon-shaped or pleomorphic. Tail represents yourself protein handset — continuation protein shells heads, in basis tail available ATPase, which regenerates energy for the injection of genetic material. There are also bacteriophages With short offshoot, not having offshoot and filiform.

Phages, like all viruses, are absolute intracellular parasites. Although they endure all information for launch own reproductions in relevant host, at them missing mechanisms for workings energy and ribosomes for protein synthesis. Some phages have a genome containing several thousand bases, while the G phage, the largest phage sequenced, contains 480,000 steam grounds - twice more middle values for bacteria, although all same insufficient quantity genes for the most important bacterial organoid how ribosomes.

big amount dedicated and studied bacteriophages defines need them systematization. Classification viruses bacteria has undergone changes: based on the characterization host virus, taken into account serological, morphological properties, a then structure and physical and chemical compound virion .

Currently, according to the International Classification and Nomenclature of Viruses bacteriophages, depending on the type of nucleic acid, are divided into DNA- and RNA-containing.

By morphological characteristics DNA containing phages highlighted in the following families: myoviridae, Siphoviridae, Podoviridae, Lipothrixviridae, Plasmaviridae, Corticoviridae, Fuselloviridae, Tectiviridae, Microviridae, Inoviridae Plectovirus and Inoviridae Inovirus.

RNA containing: cystoviridae, Leviviridae

By character interactions bacteriophage With bacterial cell distinguish virulent and moderate phages. Virulent phages may only increase in quantity through lytic cycle. Process interactions virulent bacteriophage with a cell consists of several stages: adsorption of the bacteriophage on cage, penetration in cell, biosynthesis components phage and them assembly, exit bacteriophages from the cell.

Initially bacteriophages attached to phage-specific receptors on the surface of the bacterial cell. The tail of the phage with the help of enzymes located on its end (mainly lysozyme), locally dissolves the cell membrane, contracts and contained in head DNA injected in cell, at this protein shell bacteriophage remains outside. injected DNA causes complete perestroika metabolism cells: stops synthesis bacterial DNA, RNA and proteins. DNA bacteriophage starts be transcribed With help own enzyme transcriptase, which the after hits in bacterial cage is activated. First, early and then late mRNAs are synthesized, which enter the ribosomes. host cells, where early (DNA polymerases, nucleases) and late (proteins) are synthesized capsid and tail process, enzymes lysozyme, ATPase and transcriptase) squirrels bacteriophage. replication DNA bacteriophage going on on semi-conservative mechanism and carried out With participation own DNA polymerases. After synthesis late proteins and the completion of DNA replication, the final process begins - maturation phage particles or compound phage DNA With protein shells andeducation mature infectious phage particles.

Duration this process maybe make up from several minutes before several hours. Then going on lysis cells, and released new mature bacteriophages. Sometimes the phage initiates a lysis cycle, which leads to cell lysis and release new phages. AT quality alternatives phage maybe

initiate lysogenic cycle, at which he instead of replication reversible interacts With the genetic system of the host cell, integrating into the chromosome or remaining in the form plasmids. So the way viral genome replicated synchronously With DNA host and cell division, and a similar state of the phage is called a prophage. bacteria containing prophage, becomes lysogenic before those since, bye at certain conditions or spontaneously prophage not will be stimulated on the implementation lysing cycle replication. Transition from lysogeny to lysis called lysogenic by induction or prophage induction. Phage induction is strongly influenced by the state of the cell host prior to induction, as well as nutrient availability and other conditions at the moment of induction. Poor growing conditions favor lysogenic way, then how good ones terms contribute lysing reactions.

A very important property of bacteriophages is their specificity: bacteriophages lyse cultures of a certain type, moreover, there are so-called typicalbacteriophages, lysing options inside kind, although meet polyvalent bacteriophages, which parasitize in bacteria different types.

Viruses highlighted in separate "kingdom" - Viga. They are contain only one type of nucleic acid, not have cellular structures, not have independent exchange substances being intracellular parasites reproduction viruses carried out disunited way.

According to the international classification, all viruses are subdivided according to the type of nucleic acids on the 2 subtype - RNA- and containing DNA. Further separation viruses is carried out on the basis of the size of the viruses, the type of symmetry in the formation of capsids,availability or absence outer shells and quantity contained in them capsomeres.

TIMELINE

6. Definition baseline knowledge	--------	thirty min.
7. Independent Work	---------	thirty min.
eight. Examination protocols	---------	10 min.
9. Cleaning working places	---------	10 min.
ten. Control final level knowledge and exercise on the house eleven.	---------	10 min.

PRACTICAL OCCUPATION No. 5.

Topic: Modern methods diagnostics in virology. concept about buildingviruses, viroids and prions. Methods diagnostics.

Educational goal:

1. Explore morphology and ultrastructure viruses.
2. Explore methods diagnostics viruses.

Plan lessons:

1. Peculiarities biology viruses **.**
2. Principles classification viruses.
3. Types interactions viruses With cell.

Independent Work students:

- The study demonstrations phenomenon bacteriophage on the dense and liquid nutritional environments.
- The study demonstrations intracellular inclusions (body Babesha-Negri).

EQUIPMENT

1. Demonstration phage typing.
2. Demonstration phenomenon bacteriophage on the liquid and dense
   a. nutritional environments.

3. Demonstration Wednesdays for cultivation cultures fabrics: environments Hanks, 199,Needle.
4. Demonstration ovoscope With chicken embryo.
5. Demonstration micropreparations: intranuclear inclusions at measles, bodies Pashen at smallpox, bodies Babesh –Negri at rabies.
6. Demo micropreparations: cultures fabrics, cytopathic
   a. action virus, reaction hemadsorption.
7. Demonstration reactions hemagglutination.
8. Demonstration color samples.

INFORMATIONAL MATERIL ON THEME

VIRUSES

Viruses have properties that make it impossible to use conventional methods to study them. methods microbiological research.

Distinctive properties viruses:

1. smallest sizes, measurable thousandths shares micron - millimicrons - from 8-10 m up to 300-400 m.
2. Filterability through special finely porous filters, not passingother microorganisms.
3. non-cellular structure.
4. Absolute parasitism, those. ability live and multiply only in alivecells.

The form viral particles It has several types:

16. Rod-shaped 17. Spherical (spherical)18.Cuboid
19. Capitate (spermatozoa)20. Filiform

mature viral particle, called *virions,* have next scheme buildings: in central parts located molecule DNA or RNA, which forms *nucleoid.* Around situated protective protein shell, called *capsid,* built from morphological units, called *capsomeres.* Some complex virions have external shell, called *supercapsid.*

For microbiological diagnostics viral infections in the present time apply three main methodical approach:

10. **Virological diagnostics** - founded on the allocation from researched material virus and his subsequent identification.
11. **Serological diagnostics** - definition specific immunological changes in the body under the influence of viruses (most often with help diagnosticums reveal in serum blood antiviral antibodies).
12. **Molecular biological diagnostics** - detection in clinical the material of nucleic acid fragments of pathogenic viruses using probes (hybridization NK) or PCR.

Individual viruses larger than 200 m can be stained according to Romanovsky - Giemsa; smaller viruses (variola viruses) can only be detected using special processing methods.

VIROLOGICAL RESEARCH METHOD is the main and most reliable, allows you to isolate the virus from the test material with its subsequent identification. FROM purpose accumulation virus-containing material are used chicken embryos and culture fabrics (artificially cultivated cells toy or

other tissue). Tissue cultures are maintained on vivo (medium 27, Enders) and synthetic (Wednesday 199, Needle, Melnik-Riordan) nutritional environments prepared on the basis of solutions of Hanks and Earl. They are cultivated in the usual test tubes cups carrel, test tubes Barsky.

Methodology infections chicken embryo

There are several ways to infect a chicken embryo. Most often the material injected into the allantoic and amniotic cavities, onto the chorionallantoic membrane and into yolk bag. Before infection shell eggs above air camera treated with 70% alcohol, burned on a flame, smeared with 2% iodine tincture, secondarily wipe with alcohol and burn.

At contagion in allantoic cavity in shell above air camera (borderswhich in advance circle pencil at translucence eggs in ovoscope) are doingsmall hole With help scissors or scalpel. Tuberculinov syringeintroduce 0.1-0.2 ml virus-containing material on the depth 2-3 mm below bordersair cameras. Puncture in shell poured molten paraffin. Openinginfected embryos produce in terms maximum accumulation virus (through 48-72 h incubation at temperature 37 FROM) after processing shells alcohol and 2% solution iodine her dissect and dump, filmed carefully shelledshell and consider chorionallantoic shell around places infections on theAvailability foci lesions (hemorrhages, whitish foci defeats).

Classification cellular crops:

• **primary** receive directly from fabrics animal and human through destruction proteolytic enzymes (trypsin, collagenase) intercellular substances. Disunited cells, placed in nutritional Wednesday, able attach to the surface of the culture vessel and multiply, forming a monolayer - layer one cell thick. With the help of special reagents, cells can be removed from surfaces one vessel and transplant in another. Such manipulation called **passage.** primary crops withstand not more 5-10 passages.

• **transplantable** (passage) cellular culture able withstand unlimited number of passages. They originate from tumor cells that have lostdifferentiation and not having restrictions growth.

• **semi-transplantable** (diploid) culture - fibroblast-like cells, which able to fast reproduction, withstand before 30-60 passages and save original set of chromosomes.

Viruses can reproduce only in the cells of a living organism. Concerning viruses cultivated through infections chicken embryos or cultures fabrics, a also suckling animals.

Detection (indication) of viruses *Virus*
detection in chick embryo1. Death
2. The appearance of an odor upon
opening 3. Cloudiness liquids in
cavities
four. Education sores and hemorrhages on the shells

Biological method research is in contagion sensitive to virus animal researched material, studying clinical and pathoanatomical paintings diseases. AT framework this method are used various animals: monkey, rabbits, maritime pigs, dogs, mice, rats. Ways infections: subdural, intracerebral, intranasal and other.

Methods for detecting the virus in the body of laboratory animals differ in dependencies from the view animal and type virus.

Detection viruses in culture cells

Revealing on cytopathic action (CPD). JPC represents yourself degenerative changes in cells that result from reproduction in them viruses.

Distinguish complete and partial degeneration cells monolayer.

With complete degeneration caused, for example, by polio viruses, Coxsackie and ECHO, cells of the monolayer undergo significant changes, more of themslough off co glass. Remaining single the cells are wrinkled

Partial degeneration has several varieties: 7 .Type cluster formation (adenoviruses);
eight .By type focal destruction (smallpox, flu);
3.By type symplast formation (measles, mumps, parainfluenza, herpes, HIV).

Proliferative type of changes typical for some oncogenic viruses, transforming cells in malignant.

Intracellular inclusion formed at reproductions some viruses in cytoplasm and nucleus of cells (smallpox, rabies, influenza, herpes, etc.) They are found when microscopy after coloring monolayer on Romanovsky - Giemse, a also at luminescent microscopy.

Salk color test. As a result of the vital activity of cells in a nutrient medium accumulate sour products. AT result this color incoming in compound environments indicator (phenolic red) becomes orange. At contagion culture cells with cytopathogenic viruses such as enteroviruses or reoviruses, metabolism cells suppressed medium pH and her color not are changing (Wednesday remains red).

Reaction hemagglutination. AT basis this reactions lies ability viruses, containing hemagglutinin receptors, "glue" erythrocytes. If a there is hemagglutinins - RGA+(umbrella), if No - RGA - (button).

Reaction hemadsorption. Mechanism similar With RGA.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 30 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------10 min.

PRACTICAL OCCUPATION No. 6.

Topic: Symbiosis and antibiosis. Residential and pathogenic microflora. Factors virulence microbes. Synergy and antagonism at microbes. antibiotics, mechanism actions and methods definitions sensitivity to antibiotics.
test control.

Educational goal:

4. Explore stages and factors symbiosis human With microbes.
5. Explore mechanism actions antibiotics on the microbial cell.
6. Explore methodology definitions sensitivity bacteria to antibiotics.

Plan lessons:

12. Stages and factors symbiosis human With microbes.
13. Terms formation associations residents.

14. Differences pathogens from residents.
15. antibiotics, definition, classification on chemical structure, spectrum,types and mechanism actions.
16. Chemotherapeutic drugs, mechanism them actions on the microbial cell.
17. Mechanisms medicinal sustainability bacteria.
18. Side effects of antibiotics and synthetic antimicrobials medicines.
19. Methods and units measurements antimicrobial activity.
20. Antiviral chemotherapy drugs.
21. Demonstration antibiotics With various mechanisms and spectrum actions.
22. Change module.

Independent Work students:

6. Take into account results disk antibiograms.
7. Take into account results cassette micromethod.
8. Design protocol research.

EQUIPMENT

1. cups petri With MPA With antibiotic sensitivity -four PCS.
2. Demo: indicator paper discs With antibiotics.
3. Demonstration: method serial dilutions.
4. Demonstration: antibiotics various spectrum actions.
5. Tables.

INFORMATIONAL MATERIAL ON THEME

Microorganisms are in various relationships friend With friend. The coexistence of two different organisms is called *symbiosis.* Distinguish several options for useful relationships: metabiosis, mutualism, commensalism, satelliteism.

Antagonistic relationships expressed in form unfavorable the impact of one type of microorganism on another, leading to damage and even death the last one. Forms antagonism: competition, predation, parasitism.

Microflora organism human

organism human populated about 500 types germs, constituents his normal microflora, in form communities microorganisms *(microbiocenosis).* They are are in able equilibrium *(eubiose)* friend With friend and organism person. Distinguish normal microflora various biotopes: skin, mucous shells oral cavity, upper respiratory tract, gastrointestinal tract and genitourinary system. In the body allocate permanent and transitory microflora. *Constant* microflora presented microorganisms constantly present in body. *Transient* microflora not capable to long existence in body. Permanent microflora can be divided into obligate and facultative. obligate microflora (bifidobacteria, lactobacilli, peptostreptococci, Escherichia coli and etc.) is basis microbiocenosis, a optional microflora (staphylococci, streptococci, klebsiella, clostridia, some fungi, etc.) includes a smaller part microbiocenosis. Microorganisms that make up the normal microflora are enclosed in highly hydrated exopolysaccharide nomycin matrix, forming a biological film, resistant to various influences.

Protocol research

No.	researched material	results research	Graphic image

All antibiotics possess selectivity actions. Them relative harmlessness for human determined, before Total, topics what they specifically suppress such metabolic processes in microbial cage or virus, which missing in eukaryotic cage or unavailable for them. AT this respect unique is the mechanism of action of beta-lactam antibiotics. Targets for them are transpeptidases, which complete synthesis peptidoglycan cellular walls. Since only prokaryotes have a cell wall, a eukaryotic cell does not have targets for beta-lactam antibiotics. Transpeptidases are a set enzyme proteins, localized in cytoplasmic membrane bacterial cells. Selected beta lactams vary in degree affinities to this or otherwise enzyme which got title penicillin-binding proteins. That's why biological Effect beta-lactam antibiotics different: bacteriostatic, bactericidal, lytic.

Except beta-lactam antibiotics, synthesis cellular walls amaze such antibiotics, how bacitracin, fosfomycin, cycloserine, vancomycin, ristomycin, but otherwise by, how penicillin. All they, Besides cycloserine, cause bactericidal Effect.

Mechanism actions such antibiotics, how chloramphenicol, tetracyclines, streptomycin, aminoglycosides, erythromycin, oleandromycin, spiramycin and other macrolides, lincosamides, fusidian acid, tied With oppression synthesis squirrel on the ribosome level 708. Although bacterial ribosomes 708 have basically the same structure, how ribosomes 808 eukaryotic cells, them squirrels and protein factors involved in the work of the protein-synthesizing system differ from those of ribosomes 808. This explains the selectivity of the action of these antibiotics on the protein synthesis bacteria.

Different antibiotics block protein synthesis in different ways. Tetracyclines block binding at-RNA on the A-section ribosomes 708. Chloramphenicol suppresses peptidyl transferase reaction. Streptomycins impede transformation initiator complex into a functionally active ribosome. Erythromycin blocks translocation reaction. Puromycin, joining the growing end of the synthesized polypeptide chain, causing its premature separation from the ribosome. Mechanism actions fluoroquinolones tied With them electoral suppression bacterial DNA gyrase enzymes involved in DNA replication. Fluoroquinolones are associated with specific plots DNA, which created impact DNA gyrase, and suppress her activity.

Rifampicins inhibit the activity of DNA-dependent RNA polymerases, as a result of which at bacteria inhibit transcription.

The activity of anticancer antibiotics is due to the fact that they are either inhibitor of DNA synthesis (bruneomycin), or suppress the activity of DNA-dependent RNA polymerase, t. e. blocks transcription (anthracyclines, actinomycins, olivomycin).

Accounting results definitions sensitivity dedicated from researched material microorganisms to antibiotics held next way: on the working table located a cup Petri, on the which was sown dedicated from of the microbe under study and were applied at an equal distance from each other discs With antibiotics (method this work outlined in practical guide).

student necessary do conclusion about degree sensitivity dedicated culture to antibiotics. Meaning given research comes down to next: surface nutritional environments on the cup moisten suspension dedicated clean culture in physical solution and so way achieved uniform distribution culture on all cup. "Over" sowing superimposed discs With antibiotics and cups incubate in thermostat. FROM disks, impregnated each separate antibiotic, the diffusion of antibiotics into the thickness of the agar occurs. The more sensitive culture to antibiotic topics less his efficiency concentration and topics morethe diameter of the culture stunting zone around a particular disk. At the same time, the result taken into account according to the following scheme (table).

culture highly sensitive	diameter zones oppression growth bacteria thirty and more mm.
culture medium sensitive	diameter zones oppression growth bacteria not less twenty mm.
culture weakly sensitive	diameter zones oppression growth bacteria not more ten mm.

TIMELINE

1. Definition original level knowledge ----------30 min.
2. Independent work ---------- 30 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------10 min.

PRACTICAL OCCUPATION No. 7.

Topic: Serological method diagnostics. Mechanisms non-specific human resistance. Phagocytosis, complement system, lysozyme, etc. Antigens and antibodies. Serological reactions: agglutination, precipitation, lysis, hemolysis and binding complement. Immunofluorescent, enzyme immunoassay and radioimmune analysis in diagnostics infectious diseases.

Educational goal:

5. Explore physiological mechanisms immunity.
6. Explore serological methods laboratory diagnostics.
7. Explore complement dependent serological reactions,
8. Explore reactions immunity With labeled components.

Plan lessons

18. Antigens them nature. Gaptens. Antigens bacteria.
19. Antibodies, classification. Structure immunoglobulins, main classes.
20. humoral and cellular immune answer
21. Serological reactions, them essence and mechanism, practical application. Serodiagnostics. Seroidentification.

22. Agglutination reaction , staging methods , reaction phases , practicalapplication.
23. Reaction precipitation, ways performances, practical application.
24. diagnosticums, classification, application.
25. Diagnostic serum, receiving and kinds diagnostic sera - agglutinating (adsorbed and non-adsorbed, mono- andpolyvalent), precipitating.
26. Demonstration deployed reactions agglutination, reactions hemolysis.
27. staging reactions ring precipitation.
28. Demonstration diagnosticums and diagnostic sera.
29. Reactions immune lysis, Components.
30. Reaction hemolysis.
31. Reaction binding complement (RSK). staging and accounting reactions binding complement.
32. Reaction immunofluorescence, straight and indirect.
33. ELISA analysis, Components, application.
34. radioimmune analysis, Components, application.

Independent Work students:

5. staging and accounting indicative reactions agglutination on the subject glass With purpose identification dedicated clean culture Gram-negative sticks.
6. staging and accounting deployed reactions agglutination With purpose serodiagnosis abdominal typhus.
7. staging and accounting reactions thermoring precipitation With purpose of seroindicationSiberian ulcers.
8. staging and accounting reactions binding complement With purpose serodiagnosis syphilis

EQUIPMENT

1. Set for microbiological research:Tripod- 8 pcs.
   Tweezers - 8 pcs. Bacteriological
   loop-8 pcs.Tray With stand - on
   8pcs
   Subject glasses.
   Alcohol lamp -8 pcs.
   vials With physical r-rum.-8pcs.
2. Statement and accounting of the approximate reaction of agglutination to glass slide to identify isolated pure culture Gram-negative sticks:
   Tube with slant agar with E.coli growth – 4 pcs. subject glass -4 things.
   test tube With polyvalent coli-serum -4 PCS.
3. Statement and accounting of an extended agglutination reaction in order toserodiagnosis typhoid fever.
   Researched serum-8pcs.
   Diagnosticum "O" -2 pcs.
   Diagnosticum "N" - 2 pcs.
   Diagnosticum "OH (A)" -2 pcs.
   Diagnosticum "HE IS IN)"- 2
   PCS.

test tubes clean 8pcs- eight sets.
Test tubes with physiological solution - 8 pcs.
Sterile pipettes.

4. staging and taking into account the reaction thermoring precipitation Ascoli With purposeseroindication anthrax.
   Test tube with immune serum - 4 pcs. Test tube with normal serum - 4 pcs. Test tube with physiological solution - 4 pcs.Precipitinogen -4 things.
   Pasteur pipettes - 4 pcs.
5. Demonstration: reaction Vidal.
6. Demonstration reactions precipitation in gel on Ouchterlony.
7. Demonstration bacterial preparations: serum, diagnostics.
8. Demonstration: RPGA.
9. tables

INFORMATIONAL MATERIAL ON THEME LESSONS

Under **immunity** (from Latin immunitas - liberation, getting rid of something) in biology and medicine understand complex reactions organism, directed on the preservation of its structural and functional integrity when exposed to the body genetically alien substances, both coming from outside and formed inside organism.

Distinguish several major species immunity:

-*Hereditary immunity* (congenital, species) conditioned development in process phylogenesis genetically fixed immunity kind to given antigen or microorganism.

-*Acquired immunity* specific and not transmitted on inheritance. He formed naturally and created artificially. Natural acquired immunity appears after transferred infectious disease (smallpox, measles and etc.). Artificial acquired immunity arises at vaccination.

Immunity happens *active* and *passive. Active immunity* produced organism in result impact antigen on the immune system (eg. at vaccination). *Passive immunity* conditioned antibodies transmitted from immune mother to the child at birth or by administering immune sera, and also when transplanting immune cells.

Active immunity can be *humoral* (caused by antibodies), *cellular* (due to immunocompetent cells) and *cellular humoral* (due to antibodies and immunocompetent cells). For example, antitoxic immunity to botulism and tetanus is humoral So how he conditioned antibodies circulating in the blood immunity to leprosy or tuberculosis is cellular, and to smallpox - cellular humoral.

Distinguish also immunity *sterile,* persisting in absence microorganism, and *non-sterile,* which exists only in the presence of a pathogen in body. The classic example of non-sterile immunity is immunity to tuberculosis.

Separately allocate So called *local immunity,* which the protects individual plots organism, for example, mucous shells from pathogens infectious diseases. It is formed with the participation of secretory immunoglobulin A and more active phagocytosis.

Antigens are any substances that are genetically foreign to a given organism. (usually biopolymers), which, having entered the internal environment of the body or formed in body, cause reciprocal specific immunological reaction: synthesis

antibodies, the appearance of sensitized lymphocytes or the emergence of tolerance to this substance hypersensitivity delayed or immediate types, immunological memory.

Antigens have specificity, which is associated with a certain chemical a group within a molecule called a determinant or epitope. determinants antigen are those parts of it that are recognized by antibodies and immunocompetent cells.

Distinguish *full-fledged* and *defective (haptens) antigens.* Antigens defiant full-fledged immune answer, having 2 and more determinants, called *complete.* it organic substances microbial, vegetable and animal origin. *haptens* may to be chemical substances With small molecular weight or more complex chemical substances not possessing properties of a complete antigen: some bacterial polysaccharides, a polypeptidetuberculosis bacillus (PPD), DNA, RNA, lipids, peptides. *Haptens* due to small molecular weight are not fixed by immunocompetent cells of the macroorganism and cannot elicit an immunological response. Semi- *haptens* - inorganic radicals (iodine, bromine, nitro group, nitrogen, etc.) attached to the protein molecule, may change immunological protein specificity.

Antibody formation. AT answer on the introduction antigen immune system produces antibodies — proteins, capable specifically unite With antigen, that caused their formation and, thus, participate in immunological reactions. Relate antibodies to y-globulins, t. e. least mobile in electric fieldserum protein fractions. In the body, y-globulins are produced by special cells — plasma cells. AT compliance With International classification y- globulins, bearing functions antibodies, got title immunoglobulins and are designated symbol lg. Consequently, antibodies — this is immunoglobulins, produced in answer on the introduction antigen and capable specifically to interact With this same antigen.

Functions antibodies. Primary function antibodies consists in interaction them active centers With complementary them determinants antigens. Secondary function antibodies consists of them capabilities:

- bind antigen With purpose his neutralization and elimination from the body;
- participate in recognition "foreign" antigen;
- provide cooperation immunocompetent cells (macrophages, T- and AT- lymphocytes);
- participate in various forms of the immune response (phagocytosis, killer function, immunological tolerance, immunological memory, hypersensitivity immediate type, hypersensitivity slow type).

Proteins of immunoglobulins in chemical composition belong to glycoproteins, so how they are made up of protein and sugars; built from 18 amino acids. Distinguish 5 classes immunoglobulins: IqM, IgG, IgA, IgE, IgD. Immunoglobulins M, G, A have subclasses. For example, IgG It has four subclass (IgGl, IgG2, IgG3, IgG4).

Immunological memory called ability organism at repeated meeting With one and topics same antigen to react more active and more fast formation immunity those. to react on type secondary immune response.

Immunological tolerance phenomenon opposite immunological memory. AT this case in answer on the repeated introduction antigen organism instead of energetic workings immunity shows areactivity, not answers immune reaction i.e., tolerant to the antigen.

I. Reaction agglutination on the subject glass

Place three drops on a glass slide at a sufficient distance from each other: physiological solution, typhoid agglutinating serum (No. one) and

dysentery agglutinating serum (No. 2). researched culture contribute in drop physiological solution and thoroughly grind in her before appearance expressed turbidity. bacterial loop prepared suspension postpone in serum number 1 and mix thoroughly. Next, the bacteriological loop is necessary sterilize by heating. Then take the material from the suspension with a bacterial loop culture in a drop of saline and add it to a drop of serum No. 2. Glass slightly and carefully wiggle for careful mixing. Accounting results reactions are carried out after 1-2 minutes: in a drop of saline, uniform turbidity, while agglutination is noted in a drop of one of the serums. Signs agglutination are: dropping out grains agglutinate and enlightenment liquids. AT case detection in control drop With physiological solutionspontaneous agglutination, the results of the reaction are not subject to further accounting, and the reaction requires re-setting.

II. deployed reaction agglutination

An extended agglutination reaction was set up to determine the antibody titer in serum the patient's blood.

Researched serum getting divorced physiological solution in fifty once, and received so way breeding (1:50) counts original. Further original breeding serum successively twice getting divorced physiological solution. For this (see diagram productions):

a) in all agglutination tubes, Besides No. 6, are made on 1.0 mlphysiological solution;

b) 1.0 ml of serum is added to test tubes No. 1 and No. 6 in the initial dilution of 1:50, and, so the way serum in test tube No. 1 divorced more twice that is in 100 once;

c) 1.0 ml of serum from test tube No. 1 is transferred to test tube No. 2 to the available 1.0 ml of saline, as a result of which the serum is diluted two more times, then there is in 200 times so further, up to to the test tube No. 5, where breeding reaches 1:1600;

d) it is obvious that tubes No. 1 - No. 4 contain 1.0 ml of serum, while vial 5 contains 2.0 ml of ee - the excess 1.0 ml is removed, and thus volumes in test tubes No. 1 - No. 5 are equalized. In test tube No. 6, serum control. Further, in each test tube, with the exception of test tube No. 6, add 2 drops DIAGNOSTICUM - formalin-treated suspension in physiological solution of Salmonella typhi culture cells, each milliliter of which contains 2 billion bacterial tel. Tripod With test tubes shake and put in thermostat at t 37°C on the 2 hours. After excerpts in thermostat tripod With reaction withstand at room temperature or at cold" (+3° +5°C) in flow eighteen hours.

Components reactions	An experience					serum	diagnosticia n crazy
	one	2	3	four	5	6	7
one. Phys. Solution	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2. Serum under study (1:50); ml	1.0 1:100	1.0 1:200	1.0 1:400	1.0 1:800	1.0 1:1600	1.0 1:100	1.0
3. Diagnosticum, drops	2	2	2	2	2	-	2

Accounting results produce through day in next sequences: in firstturn evaluate condition control test tubes (#6 and No. 7), in second turn

—experienced. In test tube No. 6 (serum control) should be absolutely transparent, devoid of any draft liquid. AT test tube No. 7 (control diagnosticum)
- Uniform haze. The results of test tubes should be evaluated starting from test tubes with the highest serum dilution (No. 5). The result of the reaction is taken into account according to fallout on the bottom test tubes flakes agglutinate and simultaneous enlightenment the contents of the test tube; by lightly tapping on the wall of the test tube or gently shaking agglutinate easily separates from bottom, pops up and, not changing his structures, returns to original position.

III. Reaction ring precipitation

The precipitation reaction is most often used to determine the presence in the materialsoluble antigens. into a control precipitating tube, up to approximatelyhalf of its volume is brought in by normal serum. In the experimental the test tube is introduced same amount precipitating serum. Further in every test tube is brought small amount researched material — for example, extract from skins animal (sheep), deceased presumably from Siberian ulcers. researchedmaterial should bring in through careful layering on the internal wall a precipitation tube held in the hand at a height of 30-35 cm from the surface working table under angle 45° to horizontal.

In the test tube, at the border of the serum and the test material, there is education precipitate: whitish "disk" irreversibly collapsing at shaking test tubes. AT control test tube education precipitate not observed.

IV. Reaction indirect (passive) hemagglutination (RITA)

RPGA founded on the use erythrocytes With adsorbed on the them surfaces antigens (erythrocyte diagnosticum), interaction which With relevant antibodies serum blood sick causes dropping out erythrocytes in sediment on bottom test tubes (wells) in form "revealed umbrella."

The studied patient's serum is diluted 10 times and heated at 65°C for 20 minutes. in a water bath to remove nonspecific hemagglutinins, then prepare a series of dilutions from 1:100 to 1:3200 and poured into wells of 0.5 ml. Add to each well on 0.5 ml diagnosticum. AT each row holes added corresponding erythrocyte diagnosticum: to shigella Sonne, Flexner, Newcastle and polyvalent salmonella.

At the same time, control diagnosticums and control of the studied serum are put. The result of the reaction is taken into account after incubation in a thermostat for 2 hours at 37°C. or at room temperature for 1824 hours. The response is considered positive. at condition location erythrocytes in form "umbrella" on all surfaces bottom holesand rated as "+".

Scheme productions

Breeding researched serum	DIAGNOSTICS				CONTROL				
	Sonne	Flexner	New castle	Salmon. watered.	CD 1	CD 2	CD 3	CD 4	Ks
1:100									
1:200									
1:400									
1:800									

1:1600									
1:3200									
Incubation at t 37 ^{0} C; 24 hours.									
Accounting results									

principled scheme productions reactions binding complement

Components reactions	No. test tubes		
	1(exp)	2 (counter)	3 (counter)
one. Serum under study (1:5)	0.5	0.5	-
2. Antigen in working dose	0.5	-	0.5
3Complement in working dose	0.5	0.5	0.5
4.Physiological solution	-	0.5	0.5
Incubation at t 370C – _ — 40 minutes.			
5. Hemolytic system(sheep erythrocytes + hemolytic serum)	1.0	1.0	1.0

Incubation at t 370C – _ — 40 minutes.

Accounting results _____ Hemolysis + Hemolysis +

Conclusion:

In the presence of antibodies in the test serum (positive reaction) in the experimental there is no hemolysis in vitro. With a negative reaction (no antibodies) in all three test tubes hemolysis is observed.

The complement fixation reaction takes place in two phases: 1st phase - interaction researched serum With antigen and complement. 2nd phase — indicator — determination of the presence of free complement in the mixture by adding hemolytic system consisting of sheep erythrocytes and hemolytic serum containing antibodies to sheep erythrocytes. If in the first phase of the reaction, the formation antigen-antibody complex, complement is bound by this complex in the second phase hemolysis of erythrocytes is absent (positive reaction). If antigen and antibody are not correspond to each other, the complement in the first phase of the reaction remains free and during in the second phase of the reaction, it joins the erythrocyte-hemolytic serum complex, causing hemolysis (negative reaction).

TIMELINE

one. Definition baseline knowledge -------- thirty min.

2. Independent Work --------- thirty min.

3. Examination protocols --------- 10 min.

four. Cleaning working places --------- 10 min.

5. Control final level knowledge and exercise on the house --------- 10 min.

PRACTICAL OCCUPATION No. eight.

Topic: Immunoprophylaxis, immunotherapy and immunocorrection. Assessment Methods immune status person: flowing cytofluorometry With monoclonalCD antibodies, leukocyte chemiluminescence, lymphocyte blast transformation and others Immunobiological drugs: vaccines, toxoids, serum. Immunomodulators. Probiotics.

Educational goal:

7. Explore tests first and second level, them clinical interpretation.
8. Explore pathogenesis secondary immunodeficiencies
9. Explore genetics immunodeficiencies, peculiarities inheritance.
10. Explore congenital immunodeficiencies
11. Decide and take into account functionally condition phagocytes,
12. Define activity complement blood

Plan lessons:

10. Immune status and principles his estimates.
11. Age peculiarities immune status.
12. Methods research lymphocytes, grade functional states phagocytes.
13. Definition complement
14. Tests first and second level, them clinical interpretation.
15. Genetics immunodeficiencies, peculiarities inheritance.
16. Congenital immunodeficiencies (classification, diagnostics)
17. Congenital immunodeficiencies at children.
18. Secondary immunological failure (VIN) - classification, etiology,diagnostics

Independent Work students:

1. staging and accounting functional states phagocytes,
2. Definition complement
3. Assess and interpret indicators of the immune status in secondaryimmunological insufficiency in ready immunograms

EQUIPMENT

1. Immunograms
2. smears With phagocytosis

INFORMATIONAL MATERIAL

Maturation immune reactivity fetus

The thymus is laid down in the second month of intrauterine life in the area of the third fourth gill pockets and at the sixth week has a pronounced epithelial character. On the 7-8 week he "settled" lymphocyte-like cells. To end third months formation body ends. AT further in thymus only quantitative changes are observed. Lymph nodes and other secondary bodies immune systems are laid on the 4th month, them final formation ends in postnatal period. Lymphoid follicles located in iliac gut and appendix, in Peyer's plaques contain

progenitor cells of plasma cells. They mature to plasma cells, synthesizing IgA to 14-16 weeks intrauterine development fetus.

Stem cells appear at 3-8 weeks of embryogenesis and are found in liver, blood islands of the yolk sac. Later, their main place of education becomes bone marrow. Lymphocytes are first detected at week 9 in the thymus, at 12- fifteen — in spleen. AT blood lymphocyte-like cells determined With 8-10 weeks. Lymphoid cells endowed with the function of T-lymphocytes are detected at 10-11week. B-cells are determined in the liver from 10-12 weeks, in the spleen - from 12 weeks. Synthesis and secretion of IgM is registered in the cells on the 11th, IgG - on the 22nd week. IgM content is 1/10 from maternal, a IgG — yet less. Education components systems complement starts at fetus on the 8th week pregnancy. At this components C2 and C4 are synthesized by macrophages, C5 and C4 - in the liver, lungs, peritoneum; al cells, C3 and C1 - in the small and large intestine. At the 18th week of development, all these components are determined in the blood serum of the fetus. Cellular and humoral factors of non-specific anti-infectious immunity appear in the early ontogeny.

AT period embryonic development "Work" immune systems It has their peculiarities. AT in particular among T-dependent immunological reactions first appears ability to rejection transplant (13 a week), HRT implemented much later.

Despite on the Availability in body fetus significant quantity B cells With immunoglobulin receptors plasmatic cells, directly synthesizing AT, very few. Row very powerful factors suppresses function humoral part of the immune system. This is a choriotropic gonadotropin, a-fetoprote- in, a-2-globulin. During this period, the influence of T-lymphocytes on B-cells and macrophages.

Premature activation of the immune system is observed during intrauterine infection. Practically always this is accompanied by any immunopathological disorders. Thus, for the embryonic period typical stage immunogenesis is tolerance own immune systems and passive antibody immunity due to maternal IgG, the concentration of which progressively is growing in process pregnancy. Ability fetus form components of the complement system are defective. In the third trimester of her level, although increases, but is no more than 30-50% of the indicators of adults. Local immunity in early and late ontogeny not developed.

Immune status at children after birth

A healthy full-term baby born to a healthy mother with physiological flow pregnancy, It has definite immune status and corresponding level factors non-specific anti-infectious resistance. Peculiarthe nature of the passive immunity of the newborn has positive and negative sides. So, without receiving IgM from the mother, the fetus is not saturated with those associated with this class. group isohemagglutinins, what reduces risk development conflict at mismatch of group erythrocyte Ag. FROM the other side is induced low protection against gram-negative bacteria, since this fraction predominantlyare AT against specified pathogens. AT moment birth at child physiological leukocytosis is observed, reaching up to 12-15 billion cells / l. From cells more 35% constitute lymphocytes. From general numbers lymphocytes near half make up T cells. In relative terms, their content is moderately reduced, and in absolute, given the high leukocytosis, is not changed. About 60% of all T-lymphocytes constitute cells With helper functions, fifteen% - T-suppressors.

Content antibody-dependent killers also strongly reduced from level adults. Functions of lymphocytes of newborns are changed. So, the intensity of the reaction blast transformation, induced T-mitogen FGA, "normal" or several lowered how at more senior children. reduced them ability produce lymphocytes,

induce skin reactions. At the same time, cells show a higherlevel metabolism, judging by on intensity synthesis nucleic acids.

Quantity B cells at newborns usually raised in relative and absolute values. How rule on the these cells are found IgM and IgE receptors. In the umbilical cord blood of newborns, IgM and IgG are detected, IgA and IgE are extremely rare. Synthesis of IgM increases sharply, reaching a maximum of 2-3 weeks life child, then to monthly age decreases in further slowly increases, reaching the level of adults by 6-12 months. Excessive increase in concentration IgM at newborns is evidence transferred intrauterine infections. Most often it is syphilis, rubella. Threefold increase in IgM level is evidence the presence of sepsis at child.

IgG concentration is very low at birth, increases to 7-8 years. In artificially fed children, this dynamics is realized much faster. IgA in serum blood newborns, how rule missing in flow first months life. AT further content immunoglobulin slowly is growing by the end of the first year, 28% of the level of this protein in adults. Normalization parameter is achieved by 8-15 years. IgD is usually not detected in newborns. This protein appears approximately at the 6th week, reaching the level of adults by 5-10-15 years.IgE is also not found in newborns, gradually increasing, it approaches values adults of people to 11-12 years. Acceleration accumulation reagina is risk development at children bronchial asthma and others allergic and especially atopic diseases

It is known that the content of immunoglobulins is determined by the amount of AT of various specificity. Earlier than others in children, the appearance of immune globulins has an effect microflora organism child. Main representative intestinal microflora in this

period are bifidumbacteria. Therefore, any unfavorable factors (artificial feeding, application antibiotics) is inevitable entail per yourself violation specific composition microflora and changes spectrum emerging AT. Antibody formation in newborns, as a rule, proceeds only through the primary type, requiring for implementation big quantity Ag. Much slowed down switching synthesis from IgM to IgG, in during 5-20 days at adults and 20-40 — at children.

AT moment birth phagocytes and serum blood newborns possess certain bactericidal activity against a number of microbial strains. Chemotaxis and functional activity macrophages reduced. Partially this is compensated an increase in the content of granulocytes, also endowed with a phagocytic function. However, digesting ability these cells lowered per check immaturity enzymes.

Child is born co reduced on comparison co adults levels complement and properdin, which increase rather quickly. Initial Activity lysozyme, against, significant.

Content lysozyme in body not always, depends from age, time of the year, vitamin balance and others More Total lysozyme in saliva children (before 200 mcg/ml), which is many times higher than its concentration in blood serum. The highest content lysozyme in saliva children first of the year life, in age 1-6 years it declining nearly in 3 times, to 7-15 years increases but not reaches original level.

Important factor local immunity is IgA, which the located in two forms
- serum and secretory. This y-globulin plays a major role in resistance organism against respiratory, viral, bacterial, parasitic infections, etc. Secretory IgA begins to be detected in the secrets of the first and early second weeks, continues progressively grow in subsequent months and years, in coprofilters is found from the third week of life. The amount of secretin is constantly replenished for account of secretory IgA of milk and, especially, colostrum, where its amount is 20 times or more surpasses level in serum adult. Usually after 3-5 days lactation concentration IgA sharp decreases but, Considering increasing consumption milk child, its quantity Plasma cells located in the mucous membranes, form IgA, IgM, IgG, IgD, IgE. Wall intestines synthesizes before 3 G immunoglobulins in day. IgG provide protection in mostly against toxins the rest are against bacteria and viruses. Formation of full-fledged local immunity on different data ends at one to twelve years life.

Ratio plasmatic cells gastrointestinal track, producing immune globulins, at some diseases is changing. So, at young children (from birth to three years) with chronic gastroduodenitis observed deficit IgA and

increase in IgM production. In patients with cholecystitis, there is a decrease IgA concentrations and an increase in IgM or IgG. With peptic ulcer of the duodenum 12 going on the fall level IgA in duodenal content. deficit local IgA facilitates binding immune globulins others classes With Ag.

Local immunity is determined not only by humoral, but also by cellular factors. It is shown that in the first 24 hours after the birth of a child there is a sharp increase in the number of alveolar macrophages. Their number continues to increasemonthly

age, after what stabilizes. Microbicidal properties macrophages and other phagocytic cells, as a rule, lag behind in children of the first weeks and even months life.

State immune systems child in first years life characterized high dynamism. So, after birth declining number leukocytes in circulation, the percentage of lymphocytes increases, the number of granulocytes.

Crossroads between curves reflective dynamics these cells, first occurs on the 5th day of life, after which a similar cross (decrease in specific weight lymphocytes and an increase in neutrophils) is noted only aged 4-5 years.
Highly slowly

rises relative content T cells level B-lymphocytes steadily decreases to normal.

Thus, for the embryonic period, tolerance and passive immunity per check maternal IgG, concentration which is growing in the process of pregnancy. The newborn is also dominated by maternal passive immunity, although the beginning of the synthesis of own AT, endowed with a small12 months, immune reactivity matures. At the age of 1-3 years, T- cellular immunity. AT this same period actively are functioning and B-lymphocytes.

From stated follows, what organism newborn up to before annual age poorly protected from infectious agents. Active main way

humoral link immunity. T-dependent reactions and phagocytosis developed not enough and enter in complete force later. Sometimes only to period sexual maturation. Considering all these intelligence appointment children immunotropic funds must produced extremely carefully, to not pervert natural peculiarities response, mistaking for immune disorders physiological changes in immune reactions.

In many diseases in children, early involvement in the pathological process liver and spleen. These bodies in intrauterine period carry out hemo- and lymphopoiesis. That's why in answer on the damage or infection fetus answers activation of the reticuloendothelial system. After birth, its significance decreases, replaced by more advanced machines. However, some of the so-called "slowlystarting children" with delay maturation immune systems possible reaction on the pathogenic situation of these organs. At present, in the life of a child, several critical periods, which characterized greatest vulnerability organism (D.V. Stephanie Yu.E. Veltishchev, 1996). In intrauterine period critical should count age 8-12 week, when going on differentiation organs and cells of the immune system. The first critical period after birth is period newborn™, when organism exposed action huge Ag numbers. The immune system at this time is subject to strong suppressive influences, passive humoral immunity conditioned maternal AT. noted functional imbalance of T-lymphocytes, the suppressor function is realized not only CD8+ cells, but and immature thymocytes and others cells.

The second critical age (3-6 months) is characterized by a weakening of the passive humoral immunity in connections With catabolism maternal AT. At this the suppressive orientation of immune peaktia is preserved in the presence of a pronounced lymphocytosis. This type of immune response occurs with tetanus vaccination, diphtheria, whooping cough, poliomyelitis, measles, and only after the 2-3rd revaccination develops secondary immune answer With IgG formation AT and persistent immune memory.

The third critical period is the 1st year of life. At this time, the primary character immune response on the many Ag, but already Maybe switching on the formation of IgG-AT. However, the synthesis of the IgG2 and lgG4 subclasses is delayed. suppressor the direction of the immune mechanisms begins to change into a helper one. Local immunity not developed, children sensitive to respiratory viral infections. Fourth critical period — 4th—6th years life. AT this age average the concentration of IgG and IgM in the blood corresponds to that in adults, the concentration of IgA in plasma has not yet reached the final values, the content of IgE in the blood reaches maximum values. This period is characterized by a high frequency of atopic, parasitic, immunocomplex diseases.

Fifth critical period — teenage age (at girls With 12-13, at boys from 14-15 years old). Pubertal growth spurt combined with weight loss lymphoid organs. Raise secretions genital hormones (before

total androgens) leads to suppression of the cellular link of immunity and stimulation his humoral mechanisms. AT in general at children meet the following peculiarities links immune status. T — link immunity. Quantity lymphocytes peripheral blood at birth in first day life is 24-30%, a absolute number - 3-9 • Yu'/l. Then their relative number increases and to 4-5- m days reaches 40-50%, absolute - 2.5 - 10 billion / l.

Lymphocytes of newborns are characterized by high metabolic activity, in they increased the synthesis of DNA and RNA. BTL when cultivated with PHA is well expressed as at full-term, so at premature newborns. noted high level

spontaneous transformation, in average near 6-10%, then how at adults this index is near 0.2%. AT — link immunity. System humoral immunity in difference from cellular starts actively function only afterbirth under the influence antigenic irritation. At birth child content IgG in his blood is usually greater than that of the mother, since the transplacental transition of this immunoglobulin is an active process. IgM is usually absent in serum or determined in minimal quantities. IgA are usually absent or present in trace concentrations. To end first weeks content IgA and IgM several increases IgG — to 2nd-3rd week noticeably declining and reaches minimal concentrations in age 1-4 months

phagocytic link. Number neutrophils in blood at birth relatively large: 50-70% and 4.5-20 billion / l. From the 4th day, it begins to decrease to 30-40% - 2.5-6 billion/l Monocytes in flow Total period newborn™ constitute 4-9 %
— 0.6-2 billion/l. Absorptive capacity of neutrophils of newborns is not reduced, but digesting activity lowered what leads to unfinished phagocytosis. Number HCT-positive neutrophils in spontaneous reactions at children the first 2 weeks of life is 14-20%, while in older children - 2-10%. The rise in the number of these cells in the induced test is low; phagocytic reserve cells at children in age two weeks small. Monocytes newborns characterized low bactericidal activity and inadequate migratory ability.

Immunodeficiencies (IDS) — violations immunological reactivity, caused by the loss of one or more components of the immune apparatus or closely interacting With him non-specific factors.

There is no single classification. By origin, immunodeficiencies are divided on the primary and secondary.Contents [put away]

Definition and classification

Primary immunodeficiencies are congenital (genetic or embryopathies) immune system defects. Depending on the level of violations and localization of the defect they there are:

humoral or antibody - with a predominant lesion of the B-system lymphocytes)
X-linked agammaglobulinemia (Bruton's disease)Hyper-
IgM syndrome
X-linked autosomal
recessive
deletion of immunoglobulin heavy chain genes
deficit k-chains
selective deficit subclasses IgG With or without deficit IgAdeficiency
of antibodies with normal levels of immunoglobulins general variable
immune deficiency
IgA
deficiency
cellular
syndrome Di Georgie

primary deficiency of CD4 cells
deficit CD7 T cells
deficit IL-2
multiple cytokine deficiencysignal
transmission defect combined:
syndrome Wiskott-Aldrich
ataxia-telangiectasia (syndrome Louis Bar)
severe combined immunodeficiencyX-linked With floor
autosomal recessive deficit
adenosine deaminase
deficit purine nucleoside phosphorylase
deficiency of MHC class II molecules (bald lymphocyte syndrome)
reticular dysgenesis
CD3γ or CD3ε deficiency
deficit CD8 lymphocytes
insufficiency of the complement system
defects phagocytosis
hereditary neutropenia
infantile lethal agranulocytosis (disease Kostman)cyclic neutropenia
familial benign neutropeniadefects in
phagocytic function chronic granulomatous
disease
X-linked autosomal
recessive
type I lymphocyte adhesion deficiency
deficit adhesion leukocytes 2 type
neutrophil glucose-6-dehydrogenase deficiency
deficit myeloperoxidase
deficiency of secondary granules
Shwachman's syndrome Clinical
picture of IDS Clinic It has row
general crap:

1. Recurrent and chronic infections top respiratory ways, adnexal sinuses skin, mucous shells, gastrointestinal tract, often called opportunistic bacteria protozoa, mushrooms, having trend to generalization, septicemia and torpid to conventional therapy.

2. Hematological deficits: leukocytopenia, thrombocytopenia, anemia (hemolytic and megaloblastic).

3. Autoimmune disorders: SLE-like syndrome, arthritis, scleroderma, chronic active hepatitis, thyroiditis.

4. Often, IDS is combined with type 1 allergic reactions in the form of eczema, edema Quincke, allergic reactions on the introduction medicinal drugs,immunoglobulin, blood.

5.Tumors and lymphoproliferative diseases in IDS occur 1000 times more often, how without IDS. [one]

6. At sick With IDS often are celebrated disorders digestion, diarrheal syndrome and malabsorption syndrome.

7. Sick With IDS different unusual reactions on the vaccination, a application at them alive vaccines dangerous development sepsis.

8. Primary IDS often fit together With vices development, before Total With hypoplasia of cellular elements of cartilage and hair. Cardiovascular malformations are described, main Thus, with the syndrome Dee George.

[edit]

Treatment primary IDS

Etiotropic therapy consists in the correction of a genetic defect by methods genetic engineering. But this approach is experimental. The main efforts in established primary IDS are aimed at:

prevention infections

substitution correction defective link immune systems in form transplants bone brain, substitution immunoglobulins, transfusions neutrophils.

enzyme replacement therapycytokine

therapy vitamin therapy

treatment of associated infections

Secondary immunodeficiencies

Factors that can cause secondary immunodeficiency, very varied. Secondary immunodeficiency can be caused by both environmental factors and internal factors of the body. In general, all adverse environmental factors environments that can disrupt the body's metabolism can cause the development secondary immunodeficiency. To most widespread factors environmental environment, defiant immunodeficiency relate pollution environmental environment, ionizing and microwave radiation, acute and chronic poisoning, long-term use some medicinal drugs, chronic stress and overwork. General a feature of the factors described above is a complex negative impact on all body systems, including the immune system. In addition, factors such as ionizing radiation render electoral inhibitory action on the immunity associated With oppression systems hematopoiesis. People, living or working in conditions polluted environmental environment, more often get sick various infectious diseases and are more likely to suffer from cancer. Obviously, what such promotion incidence at this categories of people related co declineactivity immune system.

The reasons

Secondary immunodeficiencies are frequent complication many diseasesand states. Main the reasons secondary IDS:

defect nutrition and general exhaustion organism also leads to decrease immunity. Against the background of general exhaustion of the body, the work of all internal organs. immune system especially sensitive to lack vitamins, minerals and nutritional substances So how implementation immune protection this is energy intensive process. Often decline immunity observed in time seasonal vitamin insufficiency (winter spring)

chronic bacterial and viral infections, as well as parasitic infestations (tuberculosis, staphylococcosis, pneumococcosis, herpes, chronic viral hepatitis, rubella, HIV, malaria, toxoplasmosis, leishmaniasis, ascariasis, etc.). With various chronic diseases infectious character immune system undergoes serious changes: violated immunoreactivity, develops increased sensitization to various microbial antigens. In addition, in the background chronic infectious process, intoxication of the body and oppression functions hematopoiesis. Immunodeficiency in time infections HIV mediated electoral defeat cells immune system virus

helminthiases

loss of immune defense factors is observed during severe blood loss, with burns or with kidney disease (proteinuria, chronic renal failure). The common feature of these pathologies is a significant loss of blood plasma or proteins dissolved in it, part them which is immunoglobulins and others components immune systems (proteins systems compliment C-jet protein). In time bleeding not only plasma is lost, but also blood cells, therefore, against the background of severe bleeding decline immunity has combined character (cellular humoral)

diarrhea syndrome

stress syndrome

severe injuries and operations also occur with a decrease in the function of the immune systems. Generally any serious disease organism leads to secondary immunodeficiency. This is partly due to metabolic disorders and intoxication. organism, a partly With topics what in time injuries or operations stand out large quantity hormones adrenal, which oppress function immune systems

endocrinopathies (DM, hypothyroidism, hyperthyroidism) lead to a decrease in immunity due to metabolic disorders. The most pronounced decrease in immune reactivity organism observed at sugar diabetes and hypothyroidism. At these diseases declining production energy in fabrics, what leads to violation fission processes and differentiation cells, including immune cells systems. Against the background of diabetes mellitus, the frequency of various infectious diseases is significantly rises. This is due not only to the suppression of the function of the immune system, but also to the fact that what elevated content glucose in blood sick diabetes stimulates reproduction bacteria

sharp and chronic poisoning various xenobiotics (chemical toxic substances medicinal drugs, narcotic means). Especially expressed decline immune protection in time reception cytostatics,glucocorticoid hormones, antimetabolites, antibiotics

low weight body at birth

decreased immune defenses in the elderly, pregnant women and children related With age and physiological features organism these categories of people

malignant neoplasms - violate activity all systems organism. Most expressed decline immunity observed in case malignant blood diseases (leukemia) and red bone marrow replacementtumor metastases. Against the background of leukemia, the number of immune cells in the blood sometimes increases tens, hundreds and thousands of times, however, these cells are non-functional and therefore not can provide normal immune body protection

autoimmune diseases arise due to violations functions immune systems. Against the background of diseases of this type and in their treatment, the immune system works not enough and, sometimes, incorrectly, which leads to damage to their own tissues and inability fight the infection

Treatment secondary IDS

The mechanisms of immune suppression in secondary IDS are different, and, as a rule, there is a combination of several mechanisms, disorders of the immune system are expressed in to a lesser extent than in the primary. As a rule, secondary immunodeficiencies are coming character. AT connections With this treatment secondary immunodeficiencies much easier and more efficient on comparison With treatment primary violations functions immune systems. Usually treatment secondary immunodeficiency start With definitions and eliminate the reasons his occurrence. For example, treatment immunodeficiency on the background chronic infections start With sanitation foci chronic inflammation. Immunodeficiency on the background vitamin and mineral insufficiency start treat at help complexes vitamins and minerals.

Recovery capabilities immune systems are great that's why elimination causes of immunodeficiency, usually leads to the restoration of the immune system. For acceleration recovery and stimulation immunity carry out well treatment immunostimulating drugs. AT the present time known big number immunostimulating drugs, With various mechanisms of action.

TIMELINE

one. Definition baseline knowledge	--------	thirty min.
2. Independent Work	---------	thirty min.
3. Examination protocols	---------	10 min.
four. Cleaning working places	---------	10 min.
5. Control final level knowledge and exercise on the house	---------	10 min.

PRACTICAL OCCUPATION No. 9.

TOPIC: Microbiological diagnostics bacterial infections. Working offmethods diagnostics For example the following pathogens:
1. staphylo-, entero- and streptococci (bacteriological method)
2. Neisseria and mycoplasma (microscopic method)

TRAINING GOAL:

5. Explore biological properties staphylococci.
6. To study methods of microbiological diagnostics of staphylococcal, streptococcal diseases.
7. To study the morphological and cultural properties of pathogenic gram-positive and gram-negative strepto- and diplococci (Neisserium).
8. master main methods laboratory diagnostics diseases, calledpathogenic diplococci.

PLAN LESSONS:

13. Morphology, cultural and biochemical properties staphylococcus.
14. Factors virulence staphylococcus.
15. Antigens staphylococcus.
16. diseases, called staphylococcus.
17. Methods diagnostics and researched material at staphylococcal diseases.
18. Preparations for specific prevention and treatmentstaphylococcal diseases.
19. Morphological characteristic pneumococcus (Streptococcus pneumoniae), meningococcus, gonococcus.
20. Comparative characteristics of biochemical activity and needs fornutritional environments for diplococci of different types.
21. Differential diagnostic signs (differences) of pathogenic andnon-pathogenic neisseria.
22. Factors virulence pathogenic diplococci.
23. Source infection, way transmission, input gates at diseases, causeddiplococci.

24. researched material and main methods diagnostics at pathological processes, called diplococci.

INDEPENDENT WORK STUDENTS:

10. Explore morphology staphylococcus in smear from clean culture, describe sketch.
11. To give macroscopic characteristic colonies on the milk-salt agar (bacteriological method diagnostics, 1st research phase).
12. To identify culture staphylococcus on morphological, cultural, biochemical properties, define factorsvirulence (2nd stage bacteriological method):
    a) recording the results of inoculation of staphylococcus culture on blood agarWith the purpose of determining hemolysin.
    b) taking into account the results of seeding in citrated plasma to determine plasmacoagulase. in) accounting seeding results on the yolk-salt agar for the purpose definitionslecithinases.
    G) accounting results sowing on the Wednesday With mannitol.
13. describe drugs for specific therapy and prevention staphylococcal diseases (staphylococcal toxoid, antistaphylococcal plasma, antistaphylococcal immunoglobulin, staphylococcal bacteriophage).
14. The study morphology pneumococci (str. pneumoniae) in smears- prints from bodies white mice, infected intraperitoneal phlegm sickpneumonia. Coloring according to Gram (table).
15. The study of biochemical activity pneumococci with the aim of differentiating themfrom streptococci. Sowing on the environments with inulin and bile.
16. Microscopic method diagnostics acute gonorrhea: microscopy smear purulent discharge from the urethra of a patient with acute gonorrhea. Coloring with methylene blue.
17. Serological method diagnostics chronic gonorrhea: estimate demonstration reaction binding complement (on Borde-Jangu), delivered with purpose detection antibodies in serum sick gonorrhea.
18. Decor protocol research.

EQUIPMENT

1. Set for bacteriological researchTripod – 8 pcs.

Tweezers -8 pcs. Bacteriological
loop -8 pcs.Bottle With physical
solution – 8 pcs.
spirit lamp -eight PCS.

2. Microscopes - 8 pcs.

Immersion oil

3. Kit colors on Gramu - 8pcs
4. cups With Kas growth staphylococcus -8pcs
5. test tubes With MPA With growth staphylococcus- 8pcs
6. test tubes With BCH - eight PCS.
7. cups petri With lecithinase activity- eight PCS
8. cups petri With milk-salt agar and growth staphylococcus-8pcs
9. test tubes With curled up citrate plasma - four PCS.

10. Bacterial preparations.

METHODOLOGICAL RECOMMENDATIONS

The main method for diagnosing staphylococcal diseases - bacteriological. To isolate a pure culture, the test material is inoculated on yolk-salt, blood or milk-salt agar. Grown isolated colonies are subcultured on oblique agar to obtain pure culture.

Identification of a pure culture is carried out by morphological, cultural, biochemical properties, then determine factors virulence.

I. DETERMINATION OF HEMOLYTIC ACTIVITY BACTERIA.

On the cup With bloody agar made sowing culture staphylococcus. cups leave in thermostat on 24 hours at a temperature 37 degrees.

When evaluating the results, attention is paid to areas of hemolysis, i.e. enlightenment environment around grown colonies. The hemolytic properties of bacteria are associated with the presence of hemolysin (exotoxin).

IV. DEFINITION LECITINASES

Staphylococcus aureus was inoculated on a plate with yolk-salt agar. Cups leave in thermostat on the 24 hours.

When evaluating the results, the presence of turbidity haloes around the colonies is taken into account, what testifies about education staphylococcus aureus enzyme lecithinases.

V. FOR DETECTION OF PLASMOCOAGULASE ENZYME Produce sowing culture staphylococcus in citrate plasma. test tubes put in thermostat. The results are taken into account after 24 hours. In the presence of the enzyme plasmacoagulase going on coagulation plasma With education clot fibrin. Availability enzyme plasma coagulase is main identification sign kind S.aureus, which the often is the causative agent nosocomial infection.

IV.DETERMINATION OF MANNITE FERMENTATIONAT ANAEROBIC CONDITIONS

For definitions this sign, confirming belonging clean culture of staphylococcus to the most aggressive species S.aureus, sowing was done on Wednesday with mannitol. When splitting mannitol, acidic products are formed that change the color of the indicator in the environment (the Andrede indicator - gives a red color to the environment, and the indicator VR - blue).

№№ P/P	researched material	results research	Graphic image

Informational material to topic

Of the 14 species of staphylococci that live on the skin and mucous membranes of humans, prevail and often cause diseases: S.aureus, S.epidermidis, S.saprophyticus. Staphylococci are Gram-positive cocci, non-motile, do not form spores or capsules. smears are arranged in clusters in the form of "clusters of grapes". **Cultural properties** . Not demanding on nutrient media: cultivated on MPA with formation of pigmented yellow colonies or white, in the MPB they give diffusely turbid growth. Character is important for identification of staphylococci growth on blood agar (hemolysis zone) and yolk-salt agar (YSA) (determinationlecithinase).
Biochemical properties . Staphylococci break down carbohydrates into acids. Important a differentiating feature of various types of staphylococci is the formationacids from mannitol in aerobic and anaerobic conditions.
pathogenicity factors .1.
Factors adhesion:
-teichoaceae acids provide adhesion on the cells organism;
"hospital strains" S. epidermidis produce a special kind of mucus, ensuring their attachment to polymeric materials of catheters, artificial valves hearts and creation on the them bacterial biofilms. it going on to developmentsepsis and endocarditis, conditioned " hospital strains" S. epidermidis.
2.Protein A non-specific binds Fc fragment IqQ that leads to oppressionphagocytosis, functions complement and opsonizing actions antibodies.
3.Eclipse antigens, having antigenic commonality With cells skin and kidneyperson.
2. Enzymes pathogenicity:
– hyaluronidase, splits hyaluronic acid in composition connective fabrics, whatpromotes the spread of staphylococci;
-plasma coagulase causes clotting proteins serum blood, forming fibrin "pseudocapsule" protecting staphylococci from phagocytosis
-Plasmocoagulase is one of the important markers of various types of staphylococcifor differentiation. S.aureus has plasmacoagulase and belongs to coagulase-positive staphylococci; S.epidermidis and S. Saprophyticus do not have plasma coagulase and are to coagulase-negative (KOS).
fibrinolysin breaks down fibrin and promotes the breakdown of staphylococci intobody;
-lecithinase destroys lipid membranes cells organism;
nucleases (RNases, DNases) cleave DNA molecules and RNA, which leads todestruction synthesis squirrel in cells and their doom;
-β-lactamase destroys -β-lactam antibiotics (penicillins, cephalosporins).5. **Exotoxins:**
-hemolysins 4th types, in mostly possessing hemolytic and cytotoxicaction;
-leukocidin destroys leukocytes;
exfoliatins cause damage and detachment of the epidermis with accumulation of fluid and formation of blisters, causing the development of the syndrome of "scalded skin" (syndrome Lyell);
-toxic shock exotoxin (ECT) causes systemic damage to the body in the form ofsyndrome toxic shock (STSH) With high lethality;
-enterotoxins cause symptoms acute food poisoning. All toxins,

Besides hemolysins, produces only S. aureus.
6.R- **_ plasmids (** factors multiple medicinal sustainability **).**
S.aureus - ubiquitous, part of the facultative microflora of the skinand mucous nasal membranes and nasopharynx.
Sources infections are sick human and bacteriocarrier. Often formedcarrier status medical staff. Ways of infection: airborne, contact, alimentary. In individuals with reducedresistance possible endogenous way of infection.
Nosological forms of infections caused by S.aureus are diverse, because are amazedany fabrics and organs.

S.epidermidis colonizes the skin and mucous membranes. Most often causes nosocomial, iatrogenic infections: sepsis, endocarditis, urological infection,what is associated with the colonization of artificial heart valves by these microorganisms, catheters vascular prostheses.

S. Saprophyticus colonizes mucous shells urogenital tract and causesinflammation of various sections of the urinary tract in people with low resistance.

Main nosological forms staphylococcal infections

Forms diseases	Material for research
Local	
Purulent skin lesions (boils, carbuncles, abscesses. Phlegmon)	Purulent detachable, purulent content
mastitis	breast milk, pus from abscess
Angina, tonsillitis	Smear from pharynx, With tonsils
Pneumonia, bronchopneumonia	Sputum, flushing water bronchi, blood
Arthritis	articular liquid
Conjunctiva	Purulent detachable conjunctiva
infections urinary ways	Urine
food poisoning	Flushing water stomach, emetic masses, faeces, leftovers food
Generalized	
Sepsis	
Endocarditis	
Meningitis	
Hematogenous osteomyelitis	
Syndrome toxic shock (STSH)	Detachable from vagina, blood

specific treatment staphylococcal infections

Acute staphylococcal infections	Chronic staphylococcal infections
Immunoglobulin staphylococcal human	Anatoxin staphylococcal purified liquid
Staphylococcal bacteriophage	killed staphylococcal vaccine, chemical staphylococcal vaccines on the basis protective antigens

streptococci - gram-positive cocci, motionless, dispute and capsules not form, insmears arranged in chains.
cultural properties . Streptococci are demanding on nutrient media. AT sugar broth give near-wall type of growth. On blood agar they formsmall convex colonies. Optional anaerobes.
By character growth on the bloody agar allocate 3 groups streptococci:

4) α- hemolytic form around colonies zone greening ("greenstreptococci") in result transformation hemoglobin in methemoglobin;
5) β-hemolytic cause full lysis erythrocytes and form aroundcolonies transparent zone;
6) γ-streptococci do not cause hemolysis and are non-homolytic. **Biochemical properties** .

When identifying streptococci, their ability toferment carbohydrates, grow on media with bile, as well as on environments with high concentration NaCI and reduce in methylene blue in milk.
Antigenic structure . By antigenic structure (polysaccharide antigens of cellwalls) R. Lensfield divided streptococci into 20 serogroups - A, B, C, etc. To streptococcus group A include - S.pyoqenes (β-hemolytic - streptococcus), most pathogenic species.
α-hemolytic streptococci are mostly part of the normal microflora("oral streptococci", enterococci), but can cause pathology in humans when decline residence of the body.
Non-hemolytic streptococci are included in compound obligate microflora mucousshells human and usually not cause pathological processes.
The most epidemiologically significant for humans is the species S. pyoqenes, possessing significant a set **of pathogenicity factors:**

4. Factors adhesion : lipoteichoic acid cellular walls;
5. Protein M provides not only adhesion but and suppression phagocytosis;
6. Eclipse antigens having antigenic commonality With cloth hearts and kidneys.

Enzymes pathogenicity:
- hyaluronidase - promotes displacement microbes on connective fabrics;
- fibrinolysin (streptokinase) - causes the dissolution of fibrin thrombi,promotes distribution in the bloodstream channel;
-DNA-aza- destroys molecules DNA.
Exotoxins :
-hemolysins (O- and S-streptolysins) - have hemolytic and cytotoxicaction on the cardiomyocytes and phagocytes;
-erythrogenic (pyrogenic) - lead to the formation of rashes on the skin, havepyrogenic action, cause development syndrome toxic shock.
Source infection :sick human and bacteriocarrier.
Ways of infection: airborne, contact, for S aqalactiae - intranatal(in time of birth).
The main method of microbiological diagnosis of streptococcal infectionsis bacteriological.

PROTOCOL RESEARCH

Stage (day research)	move research	Result
	Microscopy smears from pus, dyed according to Gramu	Among leukocytes visible Gr + cocci, located

1st	Sowing in cups with bile-salt agar	small bunches, aalso alone and in pairs. colony growth medium sizes with clouding around colonies and iridescent whisk
2 - th	Microscopy of smears from selected colonies, Gram-stained screening colonies With iridescent whisk on the obliqueagar	AT field view visible Gr + cocci locatedforms
3rd	Dedicatedclean culture. Definition signs pathogenicity: a) smear microscopy, painted on Gram; b) inoculation on Hiss media withmannitol and glucose anaerobic and anaerobicconditions; c) definition hyaluronidase activity, plasmacoagulation, DNA-basics; d) definition of α-hemolysin on plates bloody agar; e) phage typing. Sensitivity testto antibiotics paper discs.	
4th	Conclusion on carried out research	Highlighted culture pathogenic staphylococcus. Fagotype is --- sensitive to next antibiotics

Scarlet fever is an acute infectious disease, manifested by a small punctate rash, fever, general intoxication, tonsillitis. The causative agent of the disease - group streptococcus A (Streptococcus pyogenes). Infection occurs from airborne droplets (at cough, sneezing, conversation), a also through items everyday life (tableware, toys, underwear). Especially dangerous sick how sources infections in first days illness.

Sources infectious agent are sick scarlet fever or any another clinical form streptococcal infections and bacteriocarrier. More often get sick children 3-10 years, visiting children's preschool institutions and school. appearance cases scarlet fever in children's institutions, how rule preceded increased incidence of tonsillitis and acute respiratory viral infections. Children first of the year life (especially first half a year) and adults scarlet fever get sick rarely. Basic path transmission pathogen infections — air-

drip.

Pathogenesis

Pathogen penetrates in organism human through mucous shells pharynx and nasopharynx, in rare cases, infection through the mucous membranes of the genital bodies or damaged skin. AT place adhesion bacteria formed local inflammatory-necrotic hearth. Development infectious-toxic syndrome conditioned in first turn admission in blood flow erythrogenic toxin streptococci (toxin Dick), a also action peptidoglycan cellular walls. Toxinemia leads to a generalized expansion of small vessels in all organs, including in the skin and mucous membranes, and the appearance of a characteristic rash. Synthesis and accumulation antitoxic antibodies in dynamics infectious process, binding them toxins in subsequent condition decrease and liquidation manifestations toxicosis and gradual disappearance rash. Simultaneously develop moderate phenomena perivascular infiltration and edema dermis. Epidermis impregnated exudate, his cells exposed keratinization, what in further leads to peeling of the skin after the extinction of the scarlatinal rash. Maintaining a strong connections between keratinized cells in the thick layers of the epidermis on the palms and soles explains large-lamellar character peeling in these places.

Components cellular walls streptococcus (group A-polysaccharide, peptidoglycan, protein M) and extracellular products (streptolysins, hyaluronidase, DNA- aza, etc.) cause the development of delayed-type hypersensitivity reactions, autoimmune reactions, formation and fixation of immune complexes, disorders systems hemostasis. In many cases them can count cause development glomerulonephritis, arteritis, endocarditis and others complications immunopathological character.

From the lymphatic formations of the mucous membrane of the oropharynx, pathogens on lymphatic vessels enter the regional lymph nodes, where they accumulation, accompanied by the development of inflammatory reactions with foci of necrosis and leukocyte infiltration. Subsequent bacteremia may in some cases lead to penetration microorganisms in various bodies and systems, formation purulent-necrotic processes in them (purulent lymphadenitis, otitis, defeats bone fabrics temporal areas, solid cerebral shells, temporal sinuses etc.).

Scarlet fever should be distinguished from measles, rubella, pseudotuberculosis, medicinal dermatitis. In rare cases development of fibrinous raids and especially when they outletper limits tonsils disease necessary differentiate from diphtheria.

Scarlet fever is distinguished bright spilled hyperemia oropharynx ("flaming yawn"), sharply limited at the point of transition of the mucous membrane to the hard palate, bright red language raspberry and hypertrophied papillae ("crimson language"), punctate elements of the rash on a general hyperemic background, thickening of the rash in the form dark red stripes on the skin folds in places natural folds, distinctly expressed white dermographism, pale nasolabial triangle (symptom Filatov). When pressing on the skin with the palm of your hand, the rash in this place temporarily disappears ("symptom palms"), positive endothelial symptoms. After disappearance exanthems note finely scaly peeling skin (on the palms and soleslarge plate).

laboratory diagnostics

Diagnosis scarlet fever based on the clinical (acute Start diseases, fever, intoxication, acute catarrhal or catarrhal-purulent (with septic form of illness - necrotic), tonsillitis, profuse punctate rash, thickening into natural folds skin and laboratory (neutrophilic leukocytosis, increased

ESR, abundant growth of beta-hemolytic streptococci when sowing material from the focus infections on blood agar, an increase in antibody titers to streptococcal antigens - M-protein A-polysaccharide, streptolysin-O and others) data.

5. When a white mouse is infected with the sputum of a patient with pneumonia, the mouse dies from pneumococcal sepsis. From the organs of the deceased mice prepare smears-imprints. paint on Gram. On the pink background, educated cells fabrics, are found gram-positive diplococci slightly elongated forms, reminiscent of contours flame candles or lancet surrounded colorless capsule.

6. A characteristic feature of pneumococci that distinguishes them from most other species of streptococci, is related to bile and bile salts. Bile is not only kills, but and dissolves pneumococci. Against, in difference from verdant (S.faecalis, S.sanguis) and hemolytic streptococci (S.pyogenes), pneumococci decompose inulin.

7. Diagnosis acute gonorrhea put With help microscopic method research. From researched material make two strokes, one color by Gramu, another - methylene blue. At availability in smear gonococci visible gram negative diplococci, located inside leukocytes (unfinished phagocytosis).

8. So how at chronic gonorrhea gonococci are outside cells, have atypical form in form balls or very small entities, use bacterioscopic method for productions diagnosis is not possible. Therefore, in order to diagnose chronic gonorrhea apply: bacteriological, serological methods research.

Serological diagnosis of gonorrhea is made with the help of RSK. The reaction is set for detection antibodies in serum blood sick, With help famous antigen, which the represents yourself suspension killed gonococci.

Scheme productions RSK

Components reactions	1st (an experience)	2nd (control AG)	3rd (control AT)
one. Serum under study (1:5)	0.5	-	0.5
2. Antigen in working dose	0.5	0.5	-
3. Complement in working dose	0.5	0.5	0.5
four. Physiological solution	-	0.5	0.5
on the 45 minutes in thermostat			
5. Hemolytic system	1.0	1.0	1.0
on the 45 minutes in thermostat			
Accounting results		hemolysis	hemolysis

Accounting result reactions start With control tubes. At availability hemolysisin control test tubes about results reactions judge but experienced test tube.

INFORMATIONAL MATERIAL To TOPIC:

meningococci (Neisseria meningitis)- gram negative diplococci bean-shapedforms, flagella and dispute is not have, in body form a capsule.

Cultural properties . Highly demanding to conditions cultivation. grow up on the dense and liquid nutritional environments containing 20-25% serum (serum agar, whey broth). On a dense medium they form small smooth clear colonies. Strict temperature optimum - 37 ° C (at other temperatures meningococci die) necessary create how at cultivation, So and at transportation material from the patient to laboratory.

Among representatives kind Neisseria there is conditionally pathogenic kinds, inhabitants mucous shells nasopharynx - N. Sicca, N. mucosa and others At of people With weakened resistance they may call diseases clinically similar With meningococcal infection.

Antigenic structure. N meningitides has generic antigens common to all types. Within the species by capsular polysaccharide antigens distinguish between serogroups N meningitides-A,B,C,D,YZ and etc.

epidemiological outbreaks more often cause pathogens serogroups A,B,C.

Factors pathogenicity meningococci :

1. Pili - provide adhesion on the cells of the cylindrical epithelium of the nasopharynx. 2. Ig BUT- proteases - split molecules SIg BUT, lowering topics most local protection mucous membranes of the nasopharynx; 3.

Capsule - protects from phagocytosis;

4. Enzymes pathogenicity : hyaluronidase, neurominidase and others

5. Endotoxin (cell wall LPS) - causes damage to blood vessels, which is manifested by hemorrhages in the internal organs and a hemorrhagic rash on skin.

The source of infection is a sick person, or a bacteriocarrier. More often (in70-80% cases) sick children first three years of life.

Ways infections - airborne. Input gates infections - mucous shell nasopharynx. Meningococcal infection maybe leak in several clinical forms, which share on the localized and generalized.

Main clinical forms meningococcal infections and material for microbiological research

FORMS	DISEASES	MATERIAL FOR RESEARCH
Primary localized	meningococcal carriage	smear from nasopharynx
	Spicy nasopharyngitis	
Hematogenou sgeneralized	Meningococcemia	Smear from nasopharynx, blood
	Epidemiological cerebrospinal meningitis, meningoencephalitis	Smear from nasopharynx,blood. liquor

Microbiological diagnostics **meningococcal infections.**

3. Bacteriological method (basic) - isolation of pure culture pathogen on the serum environments and definition his antibiotic sensitivity.

4. Bacteriological method - uses how required indicative. AT smears from native material With coloration on Gram

stands out intracellular location bacteria and characteristic painting unfinished phagocytosis meningococci.

Specific prevention of meningococcal infection is carried out only according to epidemiological testimony meningococcal polysaccharide vaccineserogroups BUT and S.

Gonococci - (N. Gonorrhoeae) - gram-negative bean-shaped diplococci, form capsule in organism, flagella and the dispute is not have.

cultural properties . Demanding on nutrient media and temperature optimum - 37 FROM. Require freshly prepared wet nutritious environments With adding native proteins blood, serum, or ascitic fluid. Don't call hemolysis on media containing blood, on media containing milk, gelatin and potatoes not are growing.

Gonococci are characterized by pronounced antigenic variability, even within one strain.

Biochemical properties: decompose only glucose With education acids. Proteolytic activity missing, ammonia, hydrogen sulfide and indole not form.

Factors pathogenicity gonococos :

7. Pili - provide adhesion to the cells of the cylindrical epithelium urinary ways;
8. Capsule - in freshly isolated cultures, it has antiphagocyticaction;
9. Cellular wall contains endotoxin.
10. Surface protein one classes causes to bactericidal factors;
11. Surface protein 2 class forms separate protein fraction called turbidity proteins or Ora proteins (turbidity). Them consider first factors virulence gonococci, and they cause attachment to the epithelium.
12. R- plasmids multidrug resistance factors.For diagnostics are used:

Bacteriological method (basic)- selection clean culture pathogen on the serum environments and definition his antibiotic sensitivity. Coloring on Gramu and characteristic painting unfinished phagocytosis gonococci.

The serological method is used for chronic gonorrhea, in the absence of the patient's discharge. Carry out RSC on Borde-Jangu according to the standard scheme, which happens positive With 3-4 weeks. AT quality antigen for RSK apply gonovaccine or antigen from killed gonococci.

Genetic method - determination of sections of the gonococcus genome in the material from sick With using PCR.

For the **specific treatment of** chronic forms of gonorrhea, killed gonococcal vaccine.

Pneumococci - Streptococcus pneumoniae Gram-positive diplococcus, usually lanceolate or arranged in chains, having a polysaccharide capsule,which allows easily " type" them specific antisera. pneumococci motionless, dispute not form; optional anaerobes. At cultivation on the artificial nutrients environments lose the capsule moving from S-

into an R-shape. They grow well on blood and serum media. When growing on agar with ram blood form colonies with zone α partial hemolysis and greening of the medium, β full hemolysis, γ-hemolysis visually invisible hemolysis.

Enzymatic activity glucose With education dairy acids.

Pneumococcus not contains group antigen serologically heterogeneous on AGcapsular polysaccharides are isolated 84 serovar.

At pneumococcal infections With purpose allocation clean culture pathogenput bioassay - intraperitoneally infect white mice material from sick.

TIMELINE

1. Definition original level knowledge ---------- 30 min.
2. Independent job ---------- 30 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------10 min.

PRACTICAL OCCUPATION No. ten.

TOPIC: Microbiological diagnostics bacterial infections. Working offmethods diagnostics For example the following pathogens:

1. corynebacteria, actinomycetes, listeria (microscopic andbacteriological methods)

2. anaerobic bacteria (microscopic, bacteriological methods)

Educational goal:

1. Train students methods microbiological diagnostics and specific prevention diphtheria, whooping cough
2. will study laboratory diagnostics actinomycete.
3. To study modern methods of microbiological diagnostics of diseases,caused by anaerobes.
4. Explore drugs for specific prevention and therapy anaerobicdiseases.

PLAN:

1. Taxonomy and basic biological properties causative agents of diphtheria, whooping cough.2. Epidemiology, pathogenesis, immunity caused diseases.
3. Principles microbiological diagnostics diphtheria, whooping cough Preparations for etiotropic therapy and specific prevention of diphtheria, whooping cough. 4.Modern ideas about the etiology of anaerobic infection. Clostridial andnon-clostridial anaerobic infection.
5. Morphological, cultural and biochemical properties pathogens anaerobic infections: Clostridium (gas gangrene, tetanus, botulism), peptostreptococcus, bacteroids, fusobacteria, anaerobic vibrios, campylobacter and spirilla.
6. Pathogenetic aspects of anaerobic infection: primary exogenous and secondary, endogenous. Mechanisms occurrence. Opportunistic anaerobic and mixed infections.
7. Methods microbiological diagnostics anaerobic infections.

8. Principles specific prevention anaerobic infections. Preparations foractive and passive immunization.
9. Principles of specific therapy of anaerobic infection. Etiotropic andpathogenetic therapy: antibacterial, hyperbaric oxygenation and etc.

INDEPENDENT WORK

1. Cooking smear and coloring on method Neisser.2.Cooking smear and coloring on method Gram.
3. Definition toxigenicity diphtheria cultures on Ouchterlony.
4. Holding samples on the cystinase and on the urease diphtheria and false diphtheria sticks.
5. Microscopic method diagnostics gas gangrene: the study smear-imprintfrom purulent wound, staining by Gram.
6. Bacteriological method diagnostics anaerobic infections:
   1-th stage - the study on the 5% bloody agar isolated colonies bacteroids and peptostreptococcus, dedicated from purulent exudate.
   Further- receiving clean culture anaerobic bacteria in semi-liquid environment AS.
   Demonstration selective Wednesdays for cultivation anaerobes: Kitta Tarozzi, "high" column sugar agar.
   2-th stage - identification of a pure culture of anaerobic bacteria by biochemical properties With using test systems AP1-Ap (principle "variegated row").
7. Definition sensitivity anaerobic bacteria to antibiotics (micromethod).Demonstration of the results of inoculation of a pure culture in a microcassette with antibiotics. 8. Description of drugs for the specific prevention of clostridialanaerobic infection: gas gangrene tetraanotoxin, pentaanotoxin (+tetanus toxoid), tetanus toxoid component drugs ADS and vaccinesDPT, TABte.
9. Description drugs for specific therapy clostridial anaerobic infections: polyvalent antigangrenous serum, antitoxic tetanus toxoid, antitoxic monoclonal and polyvalent antibotulinum serum.
10Design protocol research.

EQUIPMENT

1. Set for bacteriological researchTripod – 8 pcs.
Tweezers -8 pcs. Bacteriological
loop -8 pcs.Bottle With physical
solution – 8 pcs.
spirit lamp -eight PCS.
2. Microscopes - 8 pcs.
Immersion oil
3. Kit colors on Gramu - 8pcs
4. Kit colors on Neisser - 8pcs
5. test tubes With diphthyroid - 8pcs
6. Demonstration: toxigenicity diphtheria cultures on Ouchterlony.
7. Demonstration: micropreparation pathogen diphtheria.
8. Demonstration: samples pisa, samples Zaks

9. Demonstration: Kitta-Tarozzi and sugar column- four PCS.
10. Demonstration: micropreparations pathogenic anaerobes
11. Tank. drugs
12. tables

METHODOLOGICAL RECOMMENDATIONS

diphtheria wand (Corynebacterium diphtheriae) — gram-positive rod-shaped bacteria of the genus Corynebacterium. The pathogen was first discovered on sections films obtained from oropharynx sick in 1883 G. Edwin Klebs (German Edwin klebs, 1834-1913). Through year Friedrich Loeffler (German Friedrich august Johannes Loeffler, 1852-1915) was highlighted clean culture. diphtheria toxin got E. RU and BUT. Yersin (1884-1888 gg.). Anatoxin discovered Ramon Gaston in 1923 G. and proposed use his for active immunization. Corynebacterium diphtheriae
— large (1–8 × 0.3–0.8 μm) straight, slightly curved polymorphic rod-shaped bacteria. Metachromatic grains of volutin are localized at the poles of cells, givingcells characteristic form "maces". grains volutin stained methylene blue on Neisser. On the micropreparations are located alone or due to features of cell division are arranged in the form of the Latin letter V or Y. Dispute and capsules not form.

Epidemiology. Humans are the source of infection in diphtheria - sick or healthy carriers toxigenic diphtheria microbes. the greatest epidemic danger is posed by patients with diphtheria of the pharynx, nose and larynx, activelyhighlighting pathogens diseases in external Wednesday With exhaled air. Minor in this respect meaning play sick diphtheria eye, skin, woundsand other localizations capable of spreading the infection by contact (through arms, Houseware).

Pathogenesis. The entrance gate of diphtheria pathogens can be practically all areas of the integument (skin and mucous membranes) of the macroorganism. However, most often they is mucous shell oropharynx, much less often - larynx, nose, conjunctiva genital bodies, wound surface, leather and others Toxigenic corynebacteria fixed on the cells fabrics, multiply and in process vital activity produce an exotoxin that has a local and general effect, causing almost all manifestations of the pathological process. microbial cells outside fabrics, being gate infection, how rule not spread and direct participation in defeat macroorganism not accept.

diphtheria exotoxin consists from several factions, each from which has independent biological action. One from them - hyaluronidase: destroys hyaluronic acid capillaries and increases their permeability. This leads to exit per limits vessels liquid parts blood, impregnation affected fabrics plasma, containing along with With others components fibrinogen. Second - necrotoxin
- causes necrosis of the epithelium at the site of the infection gate, accompanied by the release of epithelial cells thrombokinase. Last promotes transformation fibrinogen into fibrin and the formation of fibrin on the surface of the affected tissues films. Palatal tonsils, in difference from others bodies, covered multi-row epithelium. AT result emerging at diphtheria fibrinous film penetrates deep inside epithelial cover and tight soldered With tissues. Third fraction diphtheria toxin - true diphtheria toxin (basic his component) able displace from cellular structures cytochrome B and so way block in them processes cellular breathing and synthesis protein molecules. Most sensitive to these changes are the myocardium, capillaries and nerve cells. AT cardiomyocytes develop the phenomena of myocardial dystrophy with their subsequent necrosis, myolysis and development infectious-toxic myocarditis. Defeat capillaries

with diphtheria, it is accompanied by infectious-toxic shock. Nerve damage cells accompanied dystrophic changes Shvanovsky cells and demyelination of nerve fibers. Along with the noted, the general effect of diphtheria toxin manifested by the phenomena of the general intoxication.
basis laboratory diagnostics constitute bacteriological research: selection pathogen from hearth inflammation, definition his type and toxigenicity. Material take away sterile wadded tampons dry or wetted (before sterilization!) 5% solution glycerin. At storage and transportation tampons protect from cooling and drying. Material must to be sown not later 2-4 h after taking. In patients with angina who were in contact with patients with diphtheria, as well as in persons With typical clinical manifestations diphtheria diagnosis put even at negative result bacteriological research.

Of secondary importance is the determination of titers of antitoxic antibodies in paired sera at staging RNGA. toxin formation reveal using RNGA with antibody erythrocyte diagnosticum. For identifying diphtheria toxin suggested use PCR.

Main in treatment diphtheria consider introduction antitoxic diphtheria serum. She is neutralizes toxin, circulating in blood, Consequently, renders greatest Effect at an early application

Preventive Events. Vaccination remains main way to control diphtheria. The Children's Immunization Scheme includes immunization DTP vaccine starting from 3 months of life (vaccinated 3 times with an interval of 30-40 days). Revaccination is carried out 9-12 months after the completed vaccination. For revaccination in 6-7, 11-12 and 16-17 years apply ADS-M. AT individual cases, for example at contraindications to whooping cough component DPT, ADS-M apply and for vaccination.

Whooping cough (wooping-cough - English; Keuchhusten - it; Coqueluche - French) and parapertussis - acute infectious diseases, clinically indistinguishable from each other. Characterized sharp Qatar respiratory ways and attacks spasmodic cough.

The causative agent of whooping cough (Bordetella pertussis) is a short stick with rounded ends (0.2-1.2 μm), gram negative motionless, Good staining aniline paints. AT antigenic respect heterogeneous. The antigen that causes the formation of agglutinins (agglutinogen) consists of multiple components. They are called factors and are numbered from 1 to 14. Factor 7 is generic, factor one contains AT. pertussis, fourteen - AT. parapertussis, the rest meet in different combinations; for the causative agent of whooping cough, these are factors 2, 3, four, 5, 6, for parapertussis - eight, 9, ten. Reaction agglutination With adsorbed factor sera allows you to differentiate the types of bordetella and determine them antigenic options. pathogens whooping cough and parapertussis very unstable in external environment, so sowing should be done immediately after taking the material. bacteria fast perish at drying ultraviolet irradiation, under influence disinfectants funds. sensitive to erythromycin, chloramphenicol, antibiotics tetracycline groups, streptomycin.
Pathogenesis. Gates infections is mucous shell respiratory tract. whooping cough microbes attached to cells flickering epithelium, where they multiply on the surface of the mucous membrane, without penetrating into the bloodstream. On site implementation pathogen develops inflammatory process, oppressed activity ciliary apparatus of epithelial cells and increased secretion of mucus. Further there is ulceration of the epithelium of the respiratory tract and focal necrosis. Pathological process most pronounced in bronchi and bronchioles, less pronounced changes develop in trachea, larynx and nasopharynx. Mucopurulent cork cork up

clearance small bronchi, develops focal atelectasis, emphysema. Observed peribronchial infiltration. AT genesis convulsive seizures It has meaning sensitization organism to toxins pertussis sticks. permanent irritation receptors respiratory ways conditions cough and leads to formation in respiratory center hearth arousal type dominants. Due to this typical seizures spasmodic cough may to be caused and non-specific irritants. From the dominant focus, excitation can radiate to other departments nervous systems, for example on the vasomotor (increase HELL, spasm vessels). irradiation arousal explained also appearance convulsive contractions of the muscles of the face and trunk, vomiting and other symptoms of whooping cough. Postponed whooping cough (how and pertussis vaccinations) not provides tense lifelong immunity, so recurrence of whooping cough is possible (about 5% cases whooping cough falls on adults of people).

Reliable diagnosis in catarrhal period may to be staged after receiving results bacteriological research. foundation for research in these cases usually serve epidemiological data (contact With sick whooping cough absence data about vaccinations and etc.). AT period spasmodic cough diagnosis whooping cough is much easier to deliver, as typical attacks appear. However need take account of, what sometimes seizures cough, similar With pertussis, may to be conditioned others reasons (adenoviral infection, viral pneumonia, compression respiratory ways at malignant neoplasms, infectious mononucleosis and etc.), With another hand, whooping cough maybe leak atypical without characteristic seizures (at vaccinated children, at adults). Main method laboratory confirmation diagnosis is selection pathogen whooping cough Frequency allocation depends from timing taking material; on the 1st week diseases positive results can be obtained in 95% of patients, on the 4th - only in 50%, and starting from the 5th week, the microbe can no longer be isolated. The material is taken from the nasopharynx dry swab with immediate inoculation on plates with selective nutrient medium. The "cough plates" method is also used, in which a Petri dish with nutrient the medium is placed in front of the mouth of the coughing child (at a distance of about 10 cm), held in this position for several seconds to catch 5-6 coughing shocks. Cup With sowing fast close lid and put in thermostat. At transportation is protected from cooling (wrapped in paper, cotton wool, in a container put heating pad completed hot water). However on frequency allocation pathogens whooping cough method "cough records" much inferior taking swab material. Serological methods can be used for retrospective diagnostics, a also at sick With negative results bacteriological research. From old methods can use RSK, RPGA, reaction agglutination. An increase in antibody titers by 4 times or more is considered diagnostic, and also high antibody titers (1:80 and above).

AT last thing time successfully use enzyme immunoassay method for detection of antibodies in serum (class M immunoglobulins) and in nasopharyngeal mucus (immunoglobulins class BUT). These antibodies appear co 2nd-3rd weeks disease and persist in flow 3 months

4. Microscopic method for diagnosing gas gangrene. In a smear-print from purulent wound (Gram stain) shows purple rod-shaped cells colors.

5. Bacteriological method diagnostics anaerobic infections.

1-th stage. The first day . On the 5% bloody agar in cup petri (after cultivation in an anaerobic balloon: 80% N_2, 10% H_2, 10% CO_2) several species are determined isolated colonies, including those with various types of hemolysis (α, β) and pigment (for example, black pigment bacteroids groups melaninogenicus). Second day. AT

in a test tube with a pure culture of peptostreptococci in a semi-liquid medium, AS are observed small white granules at the bottom parts of the test tube with the medium. In the control of cleanliness isolated culture (gentian violet staining), chains of elongated cocci blue colors.

2-th stage. AT test system API-An for identification pure cultures on biochemical properties determined fermentation glucose (change coloring indicator in yellow) in the absence of other manifestations of glycolytic, as well as proteolytic activity (negative samples for indole and hydrogen sulfide).

3-th stage. At definition sensitivity anaerobic bacteria to antibiotics in microcassette (after cultivation in anaerostat) are celebrated positive and negative options results.

4- stage . When studying ampoules with drugs for specific prophylaxis and therapy of anaerobic infections, the goals (prevention, treatment) are noted in the protocol, character immunization (active or passive, antitoxic or antibacterial), testimony to application and peculiarities use everyone drug.

PROTOCOL RESEARCH

№№ P/P	researched material	results research	Graphic image
one.	Smear-imprint from a festering wound.Coloring on Gram.		

Informational material

Tetanus heavy wound infection.

Morphology Gram-positive rods with rounded ends. located alone or chain. Spores are located terminally.

Cultural properties obligate anaerobe. On the MPA and gelatin in strictly anaerobic conditions pathogen growing slowly and forms thin transparent colonies. When sown in a column in semi-liquid agar, it forms colonies in 24-48 hours in form "lentils" R-shape or "fluff" S shape.

Pathogenicity factors are exotoxins tetanospasmin and tetanolysin.

Antigenic structure -O and H antigens.

Immunity. There is no natural immunity in humans to tetanus.Diagnostics: bacterioscopic, bacteriological and biological.

Treatment is aimed at neutralizing the tetanus toxin with toxoid. Apply tetanus toxoid horse serum in dose 50-100 thousand ME.

Prevention - surgical treatment of the wound. Creation of an artificial active immunity in planned okay vaccination DPT, ADSm. Primary vaccination carry out children in 3- monthly age.

Clostridia botulism

Botulism - acute food toxic infection, flowing predominantdefeat central and vegetative systems.

Morphology- sticks With rounded ends, mobile, peretrichia. controversy

located subterminally.

Cultural properties - strict anaerobes. Good are growing on the environments kitta-Tarozzi, bouillon from meat fish. calls turbidity environments and gas formation.

All types clostridia botulism form hydrogen sulfide.

The antigenic structure has group-specific (H) flagella andtype-specific somatic (O) antigens.

Factors pathogenicity - botulinum toxin protein, showing neurotoxicaction. Botulinum toxin is most strong poison known to a person.

Immunity. Natural immunity human missing.

Treatment. For treatment on Bezredko sick i/v introduce one internationalmedical dose (on 10000 IU serum types BUT and E and 5000 IU type AT).

Prevention. For emergency prevention used polyvalent (typesA, B, E) horse serum.

Clostridia gas gangrene.

Anaerobic wound infection (gas gangrene, anaerobic myositis) - heavy wound infection human and animals, called bacilli kind Clostribiumperfinqens.

Morphology. Vegetative cells are large, gram-positive, immobile. Classic forms submitted under direct angle ends. AT body form capsules, they most pronounced in virulent strains. Resistant to phagocytosis.

cultural properties. On dense media, C Perfiinqens of type A forms S and R - round colonies. S - dome-shaped colonies, with smooth even edges. R - colonies wrong forms edges; in depth agar remind lumps cotton wool.

Growth on liquid and semi-liquid nutrient media, especially those containing glucose, very rapidly with the formation H2 and CO2 and usually ends after 8-12 hours Turbidity of the medium and active gas formation can be observed after 4-8 hours cultivation.

Biochemical activity- splits With education acids and gas glucose,sucrose, maltose, lactose, mannose, starch.

Proteolytic activity weak; liquefies gelatin, intensively curdle milk.

Antigenic structure - all serovars form α-toxin (lecithinase). Pathogen forms at least 12 identifying toxins and enzymes that play a role in pathogenesis gas gangrene.

Clostribium perfinqens is widely distributed in the environment; it is isolated from water, soil, sewage. Spores can persist in the environment for a long time.environment, able vegetate in soil. controversy distinguishes high sustainability to chemical and physical influences.

TIMELINE

one. Definition baseline knowledge	--------	thirty min.
2. Independent Work	---------	thirty min.
3. Examination protocols	---------	10 min.
four. Cleaning working places	---------	10 min.
5. Control final level knowledge and exercise on the house	---------	10 min.

PRACTICAL OCCUPATION No. eleven

TOPIC: Microbiological diagnostics bacterial infections. Working offmethods diagnostics For example the following pathogens:
1. pathogens intestinal infections (bacteriological, serological methods)
2. pathogens STI (serological, molecular biological methods)

Learning goal: to train students in the methods of microbiological diagnostics andspecific prevention of intestinal diseases.

PLAN:

13. Taxonomy and main biological properties of pathogens intestinal escherichiosis and intestinal yersiniosis.
14. Epidemiology, pathogenesis, immunity called diseases.
15. Principles of microbiological diagnosis of intestinal escherichiosis andintestinal yersiniosis.
16. Preparations for etiotropic therapy and specific prevention of intestinal escherichiosis and intestinal yersiniosis.
17. Taxonomy and main biological properties of pathogens abdominal typhoid, salmonellosis.
18. Epidemiology, pathogenesis, immunity called diseases.
19. Principles microbiological diagnostics abdominal typhoid, salmonellosis.
20. Preparations for etiotropic therapy and specific prevention abdominaltyphoid, salmonellosis.
21. Taxonomy and main biological properties pathogens shigellosis, cholera.
22. Epidemiology, pathogenesis, immunity called diseases.
23. Principles microbiological diagnostics shigellosis, cholera.
24. Preparations for etiotropic therapy and specific prevention shigellosis,cholera.

INDEPENDENT WORK

Bacteriological method research

11. Selection clean culture from researched material (excreta patient).
12. Sowing researched material on the differential- diagnostic Wednesday Endo (demonstration).
13. Accounting results sowing researched material on the Wednesday Endo. Selection "suspicious" colonies and them the study on the environment endo, macroscopic characteristic colonies (demonstration).
14. Seeding "suspicious colonies" on the Wednesday Ressel and MPB.
15. Decor protocol research.
16. Accounting results on the differential diagnostic Wednesday Endo, bismuth -sulfite agar (demonstration).
17. Accounting results on the environment Ressel and MPB.
18. Accounting results reactions Vidal.
19. Accounting results express diagnostics cholera (demonstration).
20. Accounting for the results of sowing on a differential diagnostic mediumPloskireva (macro- and microscopic research).

EQUIPMENT

1. Set for bacteriological researchTripod – 8 pcs.
   Tweezers -8 pcs. Bacteriological
   loop -8 pcs.Bottle With physical
   solution – 8 pcs.
   Spirit lamp -8 pcs.

2.Microscopes - 8pcs
   Immersion oil 1.Set colors on
Gramu - 8pcs

2. cups co environment Endo With growth intestinal sticks -8pcs
3. test tubes co environment Ressel - 8pcs
4. test tubes With BCH - eight PCS.
5. cups petri With indicator stripes on the indole- eight PCS
6. cups petri With indicator stripes on the hydrogen sulfide- eight PCS
7. Tank drugs

PROTOCOL RESEARCH

№№ P/P	researched material	results research	Graphic image

METHODOLOGICAL RECOMMENDATIONS

AT connections With difficulty differentiation pathogens intestinal diseases, defiant similar clinical manifestations, it is necessary to conduct a comprehensive microbiological research, including simultaneous search in the investigated material pathogens escherichiosis, shigellosis, salmonellosis and cholera.

1. The test material (stools of the patient) is inoculated on the surface of one of the differential diagnostic Wednesdays for allocation pathogen intestinal diseases (Wednesday Endo) and one %alkaline agar for allocation pathogen cholera.

Sowing is carried out with a stroke on dense surface nutrient medium in order to mechanical separation microbes and receiving isolated colonies.

Cups with 1% alkaline agar are incubated at 37 degrees. 10-12 hours, cups Wednesday Endo - 18-24 hours.

2. After incubation in thermostat crops on the cups co Wednesdays Endo and one % alkaline agar is viewed in transmitted and refractive light. With absence any signs of microbial growth on alkaline agar, a negative answer is given in respect finding pathogen cholera in researched material.

On the environment Endo through 18-24 h. growth in thermostat noted Availability colonies crimson red (fermenting lactose, which is part of the medium) and colorless (not fermenting lactose).

3. Colorless ("suspicious") colonies are sown on Ressel's medium. Medium composition Ressel: MPA, 1 % lactose, 0.1 % glucose and Andrede indicator.

Sowing is done as follows: the removed colony is carefully, without touching the edges test tubes, contribute in condensation liquid, then strokes sow all beveled surface environments and do injection in column depth. test tube With sowing on the

environment Ressel put in thermostat (37°C) on the day (18-24 h.).

Simultaneously for study proteolytic activity culture lactose-negative colonies are sown in a test tube with BCH with indicator papers, impregnated acetate lead and oxalic acid for definitions education hydrogen sulfide and indole. test tube placed in thermostat (37°C, 18-24 h.)

Escherichiosis (intestinal coliinfection) is an acute intestinal infection caused byvarious serological groups of enteropathogenic Escherichia coli (EPEC), occurring with symptoms of general intoxication and a syndrome of lesions of the gastrointestinal intestinal tract.

Etiology escherichiosis.

pathogens — enteropathogenic intestinal sticks —belong to to mind Escheirichia, kind Escherichia, family enterobacteroceae, present yourself Gram-negative rods that are stable in the environment. Can keep for months in soil, water, feces. They grow well on normal nutrient media. Fast are killed by boiling and exposure disinfectants. Escherichia have complex antigenic structure: somatic O antigen (thermostable), surface (capsular) K antigen and flagellate H-antigen (thermolabile).

Intestinal infections caused by EPKD are more common in young childrenClassification escherichiosis:

- Enteropathogenic (salmonella-like).
- Enterotoxic (cholera-like).
- Enteroinvasive (dysentery-like).
- Enterohemorrhagic.

The diagnosis of escherichiosis can only be established by isolating the pathogen. For bacteriological examination, faeces, vomit, washings are taken stomach, at generalized forms - blood, CSF. Conduct studybowel movements need straightaway same, how only sick addressed per help to doctor, So how With flow time probability allocation pathogen fast decreases. Collection bowel movements held after natural defecation or With help tampons in test tubes With glycerin mixture in quantity not more 1/3 volume preservative a vomit and gastric lavage - in glass jars with a capacity of 200-250 ml. AT medical institution must to be carried out not less three diagnostic research (first - at admission sick before destination to him antibiotics, chemotherapy drugs).

FROM purpose allocation EPKP and ETCP should select samples bowel movements from recent servings, at research EICP - samples With impurity mucus.

The selected material is delivered to the laboratory within the first 2 hours, if it impossible - placed in a refrigerator and sent to the laboratory no later than 12 hours after fence.

At decision question about etiological roles pathogen at occurrence intestinal infection is necessary take account of the following criteria:

- selection Escherichia certain serovars, related to EPKP, EICP, ETCP, EGKP or EACP, in monoculture in combined With non-pathogenic serovars escherichia; if escherichia pathogenic diagnosis maybe to be installed on alone positive bakposeva;
- massive selection ETCP (106/g faeces and more) and significant them dominance above representatives another conditionally pathogenic flora.

certain diagnostic meaning have serological methods research, although they and less informative, unconvincing, So how possible false positive results due to antigenic similarities With others enterobacteria. Are used for retrospective diagnostics, especially in time

outbreaks. Currently, RNHA is used from serological research methods. (diagnostic titer 1:200 - 1:400 for adults, 1:40 - 1:80 for children); reaction immunofluorescence; reaction immune sorption antibodies, labeled enzymes; neutralization reaction; agglutination reaction with autoculture with increasing titer antibodies in 4 or more once in dynamics diseases.

A promising diagnostic method is the polymerase chain reaction (PCR).To prove pathogenicity escherichia, need make sure, what she is It has receptors providing adhesiveness, can produce heat- labile andthermostable toxins, contains plasmid DNA encoding toxin formation (Protasov S.A., 2003).

If a stand out non-pathogenic escherichia, necessary suit to diagnostics how to such at others OKI, caused conditionally pathogenic flora: triple massive growth microorganism, no seeding pathogenic agents.

Diagnosis "escherichiosis" how noted unauthorized without bacteriological, a also serological confirmation. Exception is clinical- epidemiological justification diagnosis.

Instrumental methods surveys (sigmoidoscopy, colonoscopy) escherichiosis uninformative.

At design final diagnosis indicated view dedicated causative agent, gastrointestinal tract syndrome, severity of the disease. With a protracted course, the nature of the course of the disease is also noted. For example: escherichiosis (E. coli O111) in form acute gastroenteritis, moderate gravity.

The diagnosis of bacterial carriage can only be established when clinical symptoms of the disease are absent at the present time and have not been observed in previous 1-1.5 months Bacteriocarrier, as a rule, short-term (1-2 times pathogen release). In such cases, when making a diagnosis, only view pathogen. For example: bacteriocarrier enteropathogenic Escherichia O125.

Etiology. Pathogen (Yersinia enterocolica) - gram negative wand, anaerobic, grows well on ordinary nutritional environments at low temperatures. Known thirty serovars. Disease at human more often cause 3rd, 5th, 8th and 9th serovars.

Intestinal yersiniosis.

Epidemiology. Humans are the source of infection animals, sick and carriers. Especially often pathogen is found at murine rodents, major horned livestock, pigs, dogs, cats, in dairy products, ice cream. Infection of a person occurs through the mouth when eating infected food, wateror contact way.

Disease meets in flow Total of the year.

Pathogenesis. Pathogen breeds in thin intestines, due to what develops enterocolitis or gastroenterocolitis. AT heavy cases in areas terminal department thin guts arises ulcerative process With involving mesenteric lymphatic nodes. At penetration pathogen in blood are celebrated bacteremia and generalization process With development inflammation in organs.

Clinic. The incubation period is 2-3 days. Clinical symptoms in patients practically not is different from such at pseudotuberculosis. However necessary have in mind what at intestinal yersiniosis disease often starts With intestinal disorders (copious watery stools mixed with blood), and damage to internal organs occurs, as it were, secondarily at the height of clinical manifestations and more often in severe cases.

In the diagnosis of intestinal yersiniosis , the leading role is played by bacteriological and serological methods research. Yersinia enterocolica can highlight from feces, blood, urine, pus, slime from pharynx, lymphatic node. From methods serological

diagnostics use the agglutination reaction and the indirect hemagglutination reaction. Diagnostic titer 1:100 and above. A more reliable increase in the titer of specific antibodies in dynamics diseases.

Prevention of intestinal yersiniosis is carried out in the same way as for other intestinal diseases. infections. Specific prophylaxis not developed.

Abdominal typhus — acute cyclically flowing intestinal anthroponoticinfection caused by the bacteria Salmonella typhi (Salmonella enterica serotype typhi), with alimentary through transmission (fecal-oral), characterized fever phenomena general intoxication With development typhoid status, roseolous rashes on the skin, hepato- and splenomegaly and specific defeat lymphatic lower department thin intestines.

The causative agent is Salmonella typhi from the family Enterobacteriaceae of the genus Salmonella, movable gram-negative rod with rounded ends, highly stainable everyone aniline dyes. Works out endotoxin, pathogenic only for person. Not creates controversy.

Typhoid bacteria are quite stable in the external environment: in fresh water reservoirs, they persist for up to a month, on vegetables and fruits - up to 10 days, and in dairy products may breed and accumulate.

Under the influence of 3% chloramine solution, 5% carbolic acid solution, sublimate (1:1000), 96 % ethyl alcohol they perish through several minutes.

Typhoid salmonella have a complex antigenic structure. Various serovars contain a characteristic set of antigenic factors that are composed of combinations O- and H antigens.

laboratory diagnostics before Total is in bacteriologicalresearch blood, feces, urine, bile. Method blood cultures can use With first days diseases and before end feverish period, desirable before start treatment. To do this, 5-10 ml of blood from the cubital vein at the bedside of the patient is sown at 20% bile broth or Rapoport's medium, meat-peptone broth with 1% glucose, or even in sterile distilled water. The volume of the medium is 50-100 ml. Material ratio and Wednesday should be 1:10. Feces, urine, duodenal contents are examined from the 2nd week from start diseases, sowing on the environments Ploskireva, Levin, Muller and others The preliminary result of these studies is obtained after 2 days, the final - after 4 days. To detect typhoid bacillus in feces, urine, duodenal content use REEF With labeled sera to O- and Vi antigens. The preliminary response may be received within 1 hour, final - after 5-20 h.

From serological methods use RA (Vidal) and RPGA With cysteine. The Vidal reaction is set with H- and O-antigens from the 7th-9th day of the disease, repeated on the 3rd-4th week for definitions growth titra (from 1:200 before 1:400-1:800-1:1600). Last thing is important to exclude a positive reaction result, which can bedue to prior immunization against typhoid fever. The answer might be received through 18-20 h. At staging RPGA accounting results carry out afterincubation of the plates at 37 ° C for 1.5-2 hours and again - after 24 hours of exposure at room temperature. Positive counts reaction in titre 1:40 and above. **Salmonellosis is an** acute intestinal infection of animals and humans caused by salmonella. Acute infectious zooanthroponotic disease, called salmonella and characterized in general case, development intoxication and defeat gastrointestinal tract.

salmonellosis at human consider how certain disease (nosological form), distinguishing it from typhoid fever and paratyphoid fever. Main sourceinfections — food products, less often-sick animal, in individual cases source infections maybe to be human (sick or bacteriocarrier). Infection

going on through infected food products, how rule animal origin (meat and meat products, milk, eggs, especially duck and goose), with forced, wrong slaughter animals, violation rules storage and food preparation (contact of finished and raw products, insufficient thermal treatment products before use and t. d.). salmonellosis develop in those cases when living organisms accumulated in foods enter the body salmonella.

On the territories RF most often meet the following serovars kind Salmonella enterica subspecies enterica: Salmonella enteritidis, Salmonella Typhimurium , Salmonella infantis.

Clinical manifestations salmonellosis varied — from asymptomatic carriage pathogen infections before heavy septic forms. Incubation period ranges from 2-6 hours to 2-3 days.
Distinguish several clinical forms salmonellosis:
1.Gastrointestinal form2.
Typhoid form 3.Septic the form

AT 15-17 % cases salmonellosis in period convalescence observed short-term bacteriocarrier. Possible "transient" carriage (single selection salmonella without clinical manifestations) and chronic bacteriocarrier.

Diagnostics salmonellosis carried out complex With taking into account epidemiological data, symptoms and laboratory results, aimed at isolation and typing of the pathogen. The main type of typing salmonella is an agglutination reaction. For its holding until recently used hyperimmune sera, but now they have been replaced by monoclonal antibodies to salmonella.
Prevention.
Veterinary and sanitary supervision per slaughter livestock and processing carcasses; performance sanitary rules cooking, storage and implementation food products; examination of people entering work at catering and trade enterprises,children's institutions.

TIMELINE

1. Definition baseline knowledge ----------30 min.
2. Independent work ---------- 30 min.
3. Examination protocols ---------- 10 min.
4. Cleaning working places ----------10 min.
5. Control final level knowledge and exercise on the house ----------10 min.

PRACTICAL OCCUPATION No. 12.

TEST CONTROL.

TEST TASKS

ON MICROBIOLOGY, VIROLOGY, IMMUNOLOGISTS - MICROBIOLOGY OF THE ORAL CAVITY
FOR DENTAL FACULTY(2 WELL, SPRING SEMESTER)

Vladikavkaz

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»I OPTION

(Choose one or several correct answers)

1. Essence scientific discoveries D.I. Ivanovsky:

A) the creation of the first microscope;B) opening viruses;
AT) opening phenomena phagocytosis;
G) receiving anti-rabies vaccines.

2. Main morphological varieties bacteria:

BUT) cocci;
B) Sticks;
AT) Vintage;
G) Branching.

3. Prokaryotes that do not have a cell wall and do not synthesizepredecessors peptidoglycan:

A) Staphylococci;
B) Neisseria;
B) streptococci;G) Mycoplasmas.

4. To spore-forming bacteria relate:

A) streptococci;B) Clostridia; AT) Neisseria;
G) Salmonella.

5. What properties possess spirochetes?

BUT) have thin cellular wall;B) gram-negative;
C) Thin spirally curved cells;G) Have cytoplasmic cylinder

6. Chlamydia:

A) Gram-negative;B) form disputes;
AT) Prokaryotes;
G) obligate intracellular parasites.

7. Properties LPS:

A) It is an endotoxin;B) Thermolabile;
AT) Is an O-antigen;
G) Contains peptidoglycan.

8. Outdoor membrane Gram-negative bacteria It has:

BUT) LPS;
B) Porins;
AT) Lipid BUT;
G) Peptidoglycan.

9. Gram positive bacteria:

BUT) Escherichia;
B) Staphylococci;
B) Vibrios;

G) Streptococci.

10. Tough mucus layer outside the cell wall bacteria:

BUT) Case;
B) Mucoid;
B) Outer membrane;G)
Capsule.

11. Non-spore-forming acid resistant bacteria:

A) ClostridiaB)
Escherichia;
AT) bacilli;
G) Mycobacteria.

12. Chromosomes bacteria:

A) Associated with the cytoplasmic membrane;B)
Contain histones;
B) have the shape of a
ring;G) Connected with
LPS.

13. Darkfield microscopy applied for study:

A) Escherichia coli;B)
Rickettsia;
AT) Staphylococcus;
G) pale treponema.

14. name method coloring, applicable for pathogens tuberculosis:

A) Ziel-Nielsen;B)
Ozheshko;
B) Burri-Gins;G)
Neisser.

15. Methods definitions availability flagella bacteria :

A) Etching and impregnation with silver or mercury salts;B)
Coloring according to Neisser;
AT) Coloring on Leffler;
G) By directed character movements at bacteria in preparations"crushed" and " hanging" a drop.

16. What such transformation?

BUT) Broadcast genetic information at contact bacterialcells different "sexual" orientation;
B) Recovery damaged DNA;
C) The transfer of genetic material throughhighly polymerized DNA.

17. signs mushrooms:

A) The main component of the cell wall is chitin;B)
Have chlorophyll;
C) Contain ergosterols in the cytoplasmic membrane;G) Has a core nuclear shell.

18. protozoa, having apical complex:

BUT) Balantidia;
B) Malarial Plasmodium;AT)
Trichomonas;
G) Toxoplasma.

19. To microorganisms With prokaryotic type organizations cellsrelate:

a) moldy mushrooms; b) spirochetes; in) chlamydia;
G) mycoplasmas; e) actinomycetes.

Choose the only combination that takes into account allcorrect answers:

BUT) a, b, in;
B) b, in, G, d;
AT) in, G, d;
G) a, in, G, d;
D) b, d, d.

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

20.

1. Organs movements at bacteria:
2. bacteria, covered flagella co all parties cells:

BUT) Drank;
B) Flagella;
B) Pseudopodia;
D) Trichomonas;
D) Peritrichs.

21.

1. **microorganisms, not having cellular walls:**
2. Adhesion bacteria to eukaryotic cells:

A) amphitriches;
B) Spirochetes;
B) Mycoplasmas;
G) Porins;
D) Drank;
E) flagella.

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»II OPTION

(Choose one or several correct answers)

1. founding scientists physiological period development microbiology:

A)
Leeuwenhoek
;B) Pasteur;
AT)
Mechnikov;
G) Koch.

2. Structural features of prokaryotes: A)
ribosome sedimentation constant 70S;B)
There is a nucleoid;
C) there is no Golgi apparatus; G)
Missing nuclear membrane.

3. Not have complete cellular walls:

A) Chlamydia;
B) L-shape;
AT)
Rickettsia;
G) Mycoplasmas.

4. What morphology have sarcinas?

A) rod-shaped;B)
coccoid;

AT) twisted;
G) filiform.

5. To spirochetes relate:

BUT)
Treponema;
B) Borrelia;
AT) Leptospira;
G)
Mycoplasmas.

6. controversy actinomycetes participate in:

BUT) reproduction;
B) Protection from adverse external influences;C)
Settlement of the microbe or colonization of the
substrate; G) Transfer genes.

7. Rickettsia:

A) obligate intracellular parasites;B)
Prokaryotes;
AT) Gram-negative;
G) Are stained on method Zdrodovsky.

8. Functions of the bacterial cell wall: A)
Participation in the process of cell
division;B) Participation in exchange
substances;
C) Protection from external harmful factors;G)
maintenance permanent form.

9. Components LPS bacteria:

BUT) Lipid BUT;
B) Peptidoglycan;
B) polysaccharide side chain;G)
Porin protein.

10. Microcapsule:

A) Formed in most bacteria; B) It is clearly
visible in a light microscope;AT) Thickness
less 0.2 μm;
G) Attaches bacteria acid resistance.

11. Sustainability non-spore-forming bacteria to acids alkalis and alcoholsconditioned high content in cell wall:

A) Peptidoglycan; B)
Teikhoevs acids;
C) Peptide bridges;G)
Voskov and lipids.

12. Peculiarities grains volutin?

A) Relate to cytoplasmic inclusions;B) Are stained
according to Neisser;
C) Differ in methochromasia;G)
Contain polyphosphates.

13. Tinctorial properties bacteria characterize:

BUT) Sustainability in external environment
B) Resistance to physical factorsAT) Sensitivity to
bacteriophages
G) Attitude to certain method staining

14. Methods staining rickettsia: A)
Coloring according to Romanovsky-
Giemsa;B) Coloring according to
Neisser;
AT) Coloring on Zdrodovsky;

G) Coloring on Aueske.

15. For detection dispute at bacteria use coloration:

BUT) By Neisser;
B) According to Romanovsky-Giemsa;AT) By BurrioGuinsu;
G) By Ozheshki.

16.What such conjugation?

BUT) Correction damaged plots DNA;
B) Broadcast genetic information at help bacteriophage;AT) hereditary spasmodic change sign;
G) Broadcast genetic information at crossing bacteriumthrough genital villi.

17. signs mushrooms:

BUT) Missing chlorophyll;
B) Have rigid cellular wall;
AT) Contain ergosterols in cytoplasmic membrane;G) Eukaryotes.

18. Protozoa:

BUT) eukaryotes;
B) belong to the animal kingdom;
AT) Have cellular structure;
G) Relate to prokaryotes.

19. Light microscopy includes the following varieties: a) phase-contrast microscopy; b) electron microscopy; c) darkfieldmicroscopy; d) microscopy in dark field; e) immersion microscopy.

Choose the only combination that takes into account allcorrect answers:

BUT) a, in, G, d;
B) a, b, G, d;
AT) b, in, G, d;
G) b, in, G;
D) in, G, d.

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

20.

1. Functions pili (villi, fimbria):

2. Nucleoid bacteria:

BUT) Adhesion bacteria to substrate;B) Are antigens;
C) Serve as a receptor for bacteriophages;G) Contains 2-3 nucleoli;
E) The DNA strand is closed in a ring;E) It has nuclear shell.

21.

1. Collection forms bacteria:

2. spore-forming bacteria:

A) ClostridiaB) bacilli;
AT) Actinomycetes;
G) Spirilla;

D) Mycoplasmas;
E) Spirochetes.

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»III OPTION
(Choose one or several correct answers)

1.FROM name what scientist related opening entities fermentation [1857], microbial conditionality and contagiousness of infectious diseases [1881], methods of making vaccines and methods of prevention chicken cholera anthrax and rabies [1882-1885] ?
A) Leeuwenhoek ; B) Mechnikov;
B) Koch;
G) Pasteur.

2. Determine concept "taxon":
A) Genetically homogeneous pure culture of microbes;B) culture germs, ongoing from one cells;
C) A culture of a certain type of microbe isolated from the environment environment, pathological materials of humans and animals or received from the museum;
G) Group microorganisms, united in systematiccategory on the basis commonality properties and signs.

3. Eukaryotes:
A) the simplest;
B) Eubacteria;
AT) Mushrooms;
G) Prions.

4. bacteria, at which missing complete cellular wall:
BUT) Rickettsia; B) Mycoplasmas;
AT) Chlamydia;
G) Spirochetes.

5. Curled bacteria: A) Actinomycetes; B) Spirilla;
B) Mycobacteria;
G) Spirochetes.

6. Actinomycetes:
BUT) Gram positive microbes;
B) Cells have view branched threads;AT) Form exospores;
G) Prokaryotes.

7. Microorganisms, partially or completely lost cellularwall Under the influence external environments:
BUT) Spheroplasts;
B) protoplasts;
AT) L-shape;

G) Mycoplasmas.

8. Functions LPS:

BUT) antigenic;
B) Enzymatic;
C) Toxic (endotoxin);G)
Hereditary.

9. Microbes in which cell wall rigidity causespeptidoglycan:

A) Gram-negative bacteria;B)
Viruses;
B) Gram-positive bacteria;G)
Mushrooms.

10. Acid resistant microorganisms:

A) Mycobacteria;
B) Streptococci; B)
Vibrios;
G) Staphylococci.

11. Functions pili (villi, fimbria):

BUT) Adhesion bacteria to
substrate;B) Participation in
transfer genes;
C) Serve as a receptor for bacteriophages;G)
They are antigens.

12. Education endospore at bacteria stimulate:

BUT) Flaw oxygen;
B) Change in ambient temperature;AT) deficit
nutrients;
G) hit in organism human or animal.

13. signs gram-positive bacteria:

A) There are teichoic acids in the cell wall;B) Can
form disputes;
C) The main component of the cell wall is peptidoglycan;G)
Separate representatives of acid-resistant.

14. What kind peculiarities characteristic for mesosomes at bacteria?

BUT) formed in result invaginations cytoplasmicmembranes in cytoplasm;
B) Performs functions digestive vacuoles;AT)
Synthesize protein;
G) Reveal on Ziel-Nielsen.

15. For Capsule Detection bacteria in pure culture usecoloring:

BUT) simple;
B) According to
Neisser;AT) By
Gram;
G) By Burri Guinsu.

16. What such transformation?

A) Transfer of genetic information upon contact of bacterialcells
different "sexual" orientation;
B) Recovery damaged DNA;
C) The transfer of genetic material throughhighly
polymerized DNA.

17. higher mushrooms:

BUT) Have axial thread;

B) Have septate mycelium;
C) Form vegetative endospores;G) form exospores (conidia).

18. Give characteristic protozoa:

A) have a cellular structure;B) Relate to eukaryotes;
C) Outside surrounded by pellicle;G) Relate to kingdom animals.

19. To microorganisms with a prokaryotic type of cell organization include: a) mold fungi; b) spirochetes; c) chlamydia; G) mycoplasmas; e) actinomycetes.
Choose the only combination that takes into account allcorrect answers:

BUT) a, b, in;
B) b, in, G, d;
AT) a, c, G, d;
G) a, b, G, d;

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

20.

1. For detection acid resistant bacteria apply?
2. For detection grains volutin bacteria?

BUT) Coloring on Buri-Ginsu;
B) Coloring on Ziel-Nielsen;
AT) Coloring on Romanovsky-Giemsa;
D) Staining with diluted carbolic fuchsin;D) Coloring on Neisser.

21.

1. Function synthesis squirrel performs:
2. chromosomal genetic structures at bacteria:

A) mesosomes; B) Ribosomes;
AT) Plasmids;
D) transposons;
D) Nucleoid.

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»IV OPTION

(Choose one or several correct answers)

1. Who is one of the founders of the immunological stagedevelopment of microbiology and the creator of the phagocytic theory immunity?

BUT) Rarely;
B) Mechnikov;
B) Koch;
G) Pasteur.

2. What kind microbes relate to eukaryotes?

BUT) Protozoa;

B) Mycoplasmas;
AT) Mushrooms;
G) Chlamydia.

3. Structural features of prokaryotes: A) ribosome sedimentation constant 70S;B) There is a nucleoid;
C) there is no Golgi apparatus; G) Missing nuclear membrane.

4. What such streptobacilli?

A) Cocci forming a chain; B) sticks, generators chain;AT) Collection forms;
G) spore-forming sticks, conducive chain.

5. branching forms bacteria:

BUT) Actinomycetes;
B) Spirilla;
AT) Mycoplasmas;
G) Spirochetes.

6.microorganisms, at which absence cellular walls alwaysdetermined genetically:

A) protoplasts B) Spheroplasts AT) Chlamydia
G) Mycoplasmas

7. What kind properties characteristic for chlamydia?

BUT) Gram-negative;
B) Prokaryotes;
C) Obligate intracellular parasites;G) Have a twisted form.

8. Lipopolysaccharide bacterial cells located in:

BUT) cytoplasmic membrane;
B) the outer membrane of gram-positive bacteria;AT) Mesosomes;
G) Outdoor membrane Gram-negative bacteria.

9. AT compound peptidoglycan includes:

BUT) Teichoaceae acids
B) N-acetylglucosamine and M-acetylmuramic acidAT) Lipopolysaccharide (LPS)
G) molecules glycan.

10. What kind structures required for L-shapes bacteria?

A) a capsule
B) CPM;
B) cytoplasm;G) Nucleoid;
D) Cellular wall.

11. Non-spore-forming bacteria, most resistant to actionacids, alkalis and alcohol:

A) Mycobacteria;
B) Clostridia;
B) Escherichia;G) Bacillus.

12. Intracellular inclusion bacteria:

A) Glycogen grains;
B) Mitochondria; C)
grains of volutin;G)
Ribosomes.

13. Complex differential diagnostic methods coloring:

A) Tsil-Nelsen stain;B) Coloring
blue Leffler; AT) Coloring by
Gram;
G) Coloring divorced carbolic fuchsin.

14. On microscopy of the test material, rickettsia usuallydiscover:

A) in the cytoplasmic membrane;B)
AT mesosomes;
AT) extracellular;
G) AT cytoplasm cells.

15. For detection grains volutin at bacteria use coloration:

BUT) By Neisser;
B) According to Romanovsky-
Giemsa;AT) By BurrioGuinsu;
G) By Ozheshki.

16. What such mutagens?

A) Genes providing mutation;B)
Factors defiant mutation
AT) Factors regenerating DNA;
G) Fatory, transmitting genetic information.

17. Mycelium mushrooms - this is:

A) A cell without a cytoplasmic membrane;B)
The totality of hyphae;
AT) Aggregate chlamydospores;

G) multi-core structure.

18. Protozoa:

BUT) eukaryotes;
B) Contain a well-formed nucleus with a nuclear
membrane;AT) Arranged more difficult than bacterial
cells;
G) Prokaryotes.

19. To microorganisms With prokaryotic type organizations cellsinclude:
a) chlamydia; b) viruses; c) mold fungi; G) spirochetes; e) actinomycetes; e) mycoplasmas.
Choose the only combination that takes into account allcorrect answers:

BUT) a, b, in;
B) a, G, d, f;
AT) in, G, d;
G) a, in, G, d;
D) b, d, d.

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

1. Transduction:
2. Conjugation:
BUT) Correction damaged plots DNA;
B) Broadcast genetic information at help bacteriophage;AT) hereditary spasmodic change sign;
G) Broadcast genetic information at crossing bacteriumthrough genital villi.
E) Transfer of genetic material usinghighly polymerized DNA.

21.
1. Actinomycetes:
2. Chlamydia:
A) Gram-positive microbes;B) Gram negative microbes;
AT) Cells have view branched threads;G) Form exospores;
D) Obligate intracellular parasites.E) Eukaryotes.

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»I OPTION

(Choose one or several correct answers)

1. Essence scientific discoveries D.I. Ivanovsky:
A) the creation of the first microscope;B) opening viruses;
AT) opening phenomena phagocytosis;
G) receiving anti-rabies vaccines.

2. Main morphological varieties bacteria:
BUT) cocci;
B) Sticks;
AT) Vintage;
G) Branching.

3. Prokaryotes that do not have a cell wall and do not synthesizepredecessors peptidoglycan:
A) Staphylococci;
B) Neisseria;
B) streptococci;G) Mycoplasmas.

4. To spore-forming bacteria relate:
A) streptococci;B) Clostridia; AT) Neisseria;
G) Salmonella.

5. What properties possess spirochetes?
BUT) have thin cellular wall;B) gram-negative;
C) Thin spirally curved cells;G) Have cytoplasmic cylinder

6. Chlamydia:

A) Gram-negative;B)
form disputes;
AT) Prokaryotes;
G) obligate intracellular parasites.

7. Properties LPS:

A) It is an endotoxin;B)
Thermolabile;
AT) Is O antigen; G)
Contains peptidoglycan.

8. Outdoor membrane Gram-negative bacteria It has:

BUT) LPS;
B) Porins;
AT) Lipid
BUT;
G) Peptidoglycan.

9. Gram positive bacteria:

BUT) Escherichia;
B) Staphylococci;
B) Vibrios;
G) Streptococci.

10. Tough mucus layer outside the cell wall bacteria:

BUT) Case;
B) Mucoid;
B) Outer membrane;G)
Capsule.

11. Non-spore-forming acid resistant bacteria:

A) ClostridiaB)
Escherichia;
AT) bacilli;
G) Mycobacteria.

12. Chromosomes bacteria:

A) Associated with the cytoplasmic membrane;B)
Contain histones;
B) have the shape of a
ring;G) Connected with
LPS.

13. Darkfield microscopy applied for study:

A) Escherichia coli;B)
Rickettsia;
AT) Staphylococcus;
G) pale treponema.

14. name method coloring, applied for pathogens tuberculosis:

A) Ziel-Nielsen;B)
Ozheshko;
B) Burri-Gins;G)
Neisser.

15. Methods definitions availability flagella bacteria :

A) Etching and impregnation with silver or mercury salts;B)
Coloring according to Neisser;
AT) Coloring on Leffler;
G) By directed character movements at bacteria in preparations"crushed"
and " hanging" a drop.

16. What such transformation?

BUT) Broadcast genetic information at contact bacterialcells different "sexual" orientation;
B) Recovery damaged DNA;
C) The transfer of genetic material throughhighly polymerized DNA.

17. signs mushrooms:

A) The main component of the cell wall is chitin;B) Have chlorophyll;
C) Contain ergosterols in the cytoplasmic membrane;G) Has a core nuclear shell.

18. protozoa, having apical complex:

BUT) Balantidia;
B) Malarial Plasmodium;AT) Trichomonas;
G) Toxoplasma.

19. To microorganisms With prokaryotic type organizations cellsrelate:
a) moldy mushrooms; b) spirochetes; in) chlamydia;
G) mycoplasmas; e) actinomycetes.
Choose the only combination that takes into account allcorrect answers:

BUT) a, b, in;
B) b, in, G, d;
AT) in, G, d;
G) a, in, G, d;
D) b, d, d.

MAKE LOGICAL PAIRS: QUESTION AND ANSWER
twenty.

1. Organs movements at bacteria:
2. bacteria, covered flagella co all parties cells:

BUT) Drank;
B) Flagella;
B) Pseudopodia;
D) Trichomonas;
D) Peritrichs.

21.

1. **microorganisms, not having cellular walls:**
2. Adhesion bacteria to eukaryotic cells:

A) amphitriches;
B) Spirochetes;
B) Mycoplasmas;
G) Porins;
D) Drank;
E) flagella.

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»II OPTION

(Choose one or several correct answers)

1. founding scientists physiological period development microbiology:

A) Leeuwenhoek ;B) Pasteur;
AT) Mechnikov;

G) Koch.

2. Structural features of prokaryotes: A) ribosome sedimentation constant 70S;B) There is a nucleoid;
C) there is no Golgi apparatus; G) Missing nuclear membrane.

3. Not have complete cellular walls:
A) Chlamydia;
B) L-shape;
AT) Rickettsia;
G) Mycoplasmas.

4. What morphology have sarcinas?
A) rod-shaped;B) coccoid; AT) twisted;
G) filiform.

5. To spirochetes relate:
BUT) Treponema;
B) Borrelia;
AT) Leptospira;
G) Mycoplasmas.

6. controversy actinomycetes participate in:
BUT) reproduction;
B) Protection from adverse external influences;C) Settlement of the microbe or colonization of the substrate; G) Transfer genes.

7. Rickettsia:
A) obligate intracellular parasites;B) Prokaryotes;
AT) Gram-negative;
G) Are stained on method Zdrodovsky.

8. Functions of the bacterial cell wall: A) Participation in the process of cell division;B) Participation in exchange substances;
C) Protection from external harmful factors;G) Maintenance permanent form.

9. Components LPS bacteria:
BUT) Lipid BUT;
B) Peptidoglycan;
B) polysaccharide side chain;G) Porin protein.

10. Microcapsule:
A) Formed in most bacteria; B) It is clearly visible in a light microscope;AT) Thickness less 0.2 μm;
G) Attaches bacteria acid resistance.

11. Sustainability non-spore-forming bacteria to acids alkalis and alcoholsconditioned high content in cell wall:
A) Peptidoglycan; B) Teikhoevs acids;
C) Peptide bridges;G) Voskov and lipids.

12. Peculiarities grains volutin?

A) Relate to cytoplasmic inclusions;B) Are stained according to Neisser;
C) Differ in methochromasia;G) Contain polyphosphates.

13. Tinctorial properties bacteria characterize:

BUT) Sustainability in external environment
B) Resistance to physical factorsAT) Sensitivity to bacteriophages
G) Attitude to certain method staining

14. Methods staining rickettsia: A) Coloring according to Romanovsky-Giemsa;B) Coloring according to Neisser;
AT) Coloring on Zdrodovsky;
G) Coloring according to Aueska.

15. For detection dispute at bacteria use coloration:

BUT) By Neisser;
B) According to Romanovsky-Giemsa;AT) By BurrioGuinsu;
G) By Ozheshki.

16.What such conjugation?

BUT) Correction damaged plots DNA;
B) Broadcast genetic information at help bacteriophage;AT) hereditary spasmodic change sign;
G) Broadcast genetic information at crossing bacteriumthrough genital villi.

17. signs mushrooms:

BUT) Missing chlorophyll;
B) Have rigid cellular wall;
AT) Contain ergosterols in cytoplasmic membrane;G) Eukaryotes.

18. Protozoa:

BUT) eukaryotes;
B) belong to the animal kingdom;
AT) Have cellular structure;
G) Relate to prokaryotes.

19. Light microscopy includes the following varieties: a) phase-contrast microscopy; b) electron microscopy; c) darkfieldmicroscopy; d) microscopy in dark field; e) immersion microscopy.

Choose the only combination that takes into account allcorrect answers:

BUT) a, in, G, d;
B) a, b, G, d;
AT) b, in, G, d;
G) b, in, G;
D) in, G, d.

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

20.

1. Functions pili (villi, fimbria):

2. Nucleoid bacteria:

BUT) Adhesion bacteria to substrate;B) Are antigens;
C) Serve as a receptor for bacteriophages;G) Contains 2-3 nucleoli;
E) The DNA strand is closed in a ring;E) It has nuclear shell.

21.

1. Collection forms bacteria:

2. spore-forming bacteria:

A) ClostridiaB) bacilli;
AT) Actinomycetes;
G) Spirilla;
D) Mycoplasmas;
E) Spirochetes.

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»III OPTION

(Choose one or several correct answers)

1.FROM name what scientist related opening entities fermentation [1857], microbial conditionality and contagiousness of infectious diseases [1881], methods of making vaccines and methods of prevention chicken cholera anthrax and rabies [1882-1885] ?

A) Leeuwenhoek; B) Mechnikov;
B) Koch;
G) Pasteur.

2. Determine concept "taxon":

A) Genetically homogeneous pure culture of microbes;B) culture germs, ongoing from one cells;
C) A culture of a certain type of microbe isolated from the environment environment, pathological materials of humans and animals or received from the museum;
G) Group microorganisms, united in systematiccategory on the basis commonality properties and signs.

3. Eukaryotes:

A) the simplest;
B) Eubacteria;
AT) Mushrooms;
G) Prions.

4. bacteria, at which missing complete cellular wall:

BUT) Rickettsia; B) Mycoplasmas;
AT) Chlamydia;
G) Spirochetes.

5. Curled bacteria: A) Actinomycetes; B) Spirilla;

B) Mycobacteria;
G) Spirochetes.

6. Actinomycetes:

BUT) Gram positive microbes;
B) Cells have view branched threads;AT) Form exospores;
G) Prokaryotes.

7. Microorganisms, partially or completely lost cellularwall Under the influence environmental factors:

BUT) Spheroplasts;
B) protoplasts;
AT) L-shape;
G) Mycoplasmas.

8. Functions LPS:

BUT) antigenic;
B) Enzymatic;
C) Toxic (endotoxin);G) Hereditary.

9. Microbes in which cell wall rigidity causespeptidoglycan:

A) Gram-negative bacteria;B) Viruses;
B) Gram-positive bacteria;G) Mushrooms.

10. Acid resistant microorganisms:

A) Mycobacteria;
B) Streptococci; B) Vibrios;
G) Staphylococci.

11. Functions pili (villi, fimbria):

BUT) Adhesion bacteria to substrate;B) Participation in transfer genes;
C) Serve as a receptor for bacteriophages;G) They are antigens.

12. Education endospore at bacteria stimulate:

BUT) Flaw oxygen;
B) Change in ambient temperature;AT) deficit nutrients;
G) hit in organism human or animal.

13. signs gram-positive bacteria:

A) There are teichoic acids in the cell wall;B) Can form disputes;
C) The main component of the cell wall is peptidoglycan;G) Separate representatives of acid-resistant.

14. What kind peculiarities characteristic for mesosomes at bacteria?

BUT) formed in result invaginations cytoplasmicmembranes in cytoplasm;
B) Performs the functions of a digestive vacuole;AT) Synthesize protein;
G) Reveal on Ziel-Nielsen.

15. For Capsule Detection bacteria in pure culture usecoloring:

BUT) simple;
B) According to
Neisser;AT) By
Gram;
G) By Burri Guinsu.

16. What such transformation?

A) Transfer of genetic information upon contact of bacterialcells different "sexual" orientation;
B) Recovery damaged DNA;
C) The transfer of genetic material throughhighly polymerized DNA.

17. higher mushrooms:

BUT) Have axial thread;
B) Have septate mycelium;
C) Form vegetative endospores;G) form exospores (conidia).

18. Give characteristic protozoa:

A) have a cellular structure;B) Relate to eukaryotes;
C) Outside surrounded by pellicle;G) Relate to kingdom animals.

19. To microorganisms With prokaryotic type organizations cellsinclude: a) mold fungi; b) spirochetes; c) chlamydia; G) mycoplasmas; e) actinomycetes.
Choose the only combination that takes into account allcorrect answers:

BUT) a, b, in;
B) b, in, G, d;
AT) a, c, G, d;
G) a, b, G, d;

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

20.

1. For detection acid resistant bacteria apply?
2. For detection grains volutin bacteria?

BUT) Coloring on Buri-Ginsu;
B) Coloring on Ziel-Nielsen;
AT) Coloring on Romanovsky-Giemsa;
D) Staining with diluted carbolic fuchsin;D) Coloring on Neisser.

21.

1. Function synthesis squirrel performs:
2. chromosomal genetic structures at bacteria:

A) mesosomes; B) Ribosomes;
AT) Plasmids;
D) transposons;
D) Nucleoid.

«GENERAL MICROBIOLOGY. STRUCTURE OF A BACTERIAL CELL»IV OPTION

(Choose one or several correct answers)

1. Who is one of the founders of the immunological stagedevelopment of microbiology and the creator of the phagocytic theory immunity?

BUT) Rarely;
B)
Mechnikov;
B) Koch;
G) Pasteur.

2. What kind microbes relate to eukaryotes?

A) the simplest;
B)
Mycoplasmas;
AT) Mushrooms;
G) Chlamydia.

3. Structural features of prokaryotes: A)
ribosome sedimentation constant 70S;B)
There is a nucleoid;
C) there is no Golgi apparatus; G)
Missing nuclear membrane.

4. What such streptobacilli?

A) Cocci forming a chain; B) sticks,
generators chain;AT) Collection
forms;
G) spore-forming sticks, conducive chain.

5. branching forms bacteria:

BUT)
Actinomycetes;
B) Spirilla;
AT)
Mycoplasmas;
G) Spirochetes.

6.microorganisms, at which absence cellular walls alwaysdetermined genetically:

A) protoplasts B)
Spheroplasts AT)
Chlamydia
G) Mycoplasmas

7. What kind properties characteristic for chlamydia?

BUT) Gram-negative;
B) Prokaryotes;
C) Obligate intracellular parasites;G) Have a
twisted form.

8. Lipopolysaccharide bacterial cells located in:

BUT) cytoplasmic membrane;
B) the outer membrane of gram-positive bacteria;AT)
Mesosomes;
G) outdoor membrane Gram-negative bacteria.

9. AT compound peptidoglycan includes:

BUT) Teichoaceae acids
B) N-acetylglucosamine and M-acetylmuramic acidAT)
Lipopolysaccharide (LPS)
G) molecules glycan.

10. What kind structures required for L-shapes bacteria?

A) a capsule
B) CPM;
B) cytoplasm;G)
Nucleoid;
D) Cellular wall.

11. Non-spore-forming bacteria, most resistant to actionacids, alkalis and alcohol:

A) Mycobacteria;
B) Clostridia;
B)
Escherichia;G)
Bacillus.

12. Intracellular inclusion bacteria:

A) Glycogen grains;
B) Mitochondria; C)
grains of volutin;G)
Ribosomes.

13. Complex differential diagnostic methods coloring:

A) Tsil-Nelsen stain;B) Coloring
blue Leffler; AT) Coloring by
Gram;
G) Coloring divorced carbolic fuchsin.

14. On microscopy of the test material, rickettsia usuallydiscover:

A) in the cytoplasmic membrane;B)
AT mesosomes;
AT) extracellular;
G) AT cytoplasm cells.

15. For detection grains volutin at bacteria use coloration:

BUT) By Neisser;
B) According to Romanovsky-
Giemsa;AT) By BurrioGuinsu;
G) By Ozheshki.

16. What such mutagens?

A) Genes providing mutation;B)
Factors defiant mutation
AT) Factors regenerating DNA;
G) Fatory, transmitting genetic information.

17. Mycelium mushrooms - this is:

A) A cell without a cytoplasmic membrane;B)
The totality of hyphae;
AT) Aggregate chlamydospores;
G) multi-core structure.

18. Protozoa:

BUT) eukaryotes;
B) Contain a well-formed nucleus with a nuclear
membrane;AT) Arranged more difficult than bacterial
cells;
G) Prokaryotes.

19. To microorganisms With prokaryotic type organizations cellsinclude: a) chlamydia; b) viruses; c) mold fungi; G) spirochetes; e) actinomycetes; e) mycoplasmas.
Choose the only combination that takes into account allcorrect answers:

BUT) a, b, in;
B) a, G, d, f;
AT) in, G, d;
G) a, in, G, d;
D) b, d, d.

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

20.

1. Transduction:

2. Conjugation:

BUT) Correction damaged plots DNA;
B) Broadcast genetic information at help bacteriophage;AT) hereditary spasmodic change sign;
D) Transfer of genetic information when crossing bacteriumthrough genital villi.
E) Transfer of genetic material usinghighly polymerized DNA.

21.

1. Actinomycetes:

2. Chlamydia:

A) Gram-positive microbes;B)
Gram negative microbes;
AT) Cells have view branched threads;G)
Form exospores;
D) Obligate intracellular parasites.E)
Eukaryotes.

«INFECTION AND IMMUNITY. STRUCTURE AND FUNCTIONS OF ANTIGENS ANDANTIBODIES"

I OPTION

(Choose one or several correct answers)

1. pathogenicity microbe - this is sign:
A. Genotypic;B.
Potential;
AT. Inherent mind microbe;
G. influencing on the susceptibility macroorganism.

2. Dlm is unit measurements:
A. Virulence of microbes;B.
Antigenicity of microbes; AT.
Toxigenicity microbes;
G. Immunogenicity microbes.

3. characteristic properties endotoxin:
BUT. Protein nature;
B. Causes an increase in body temperature;
AT. translates into toxoid;
G. Is factor pathogenicity.

4. characteristic signs infectious diseases:
A. The presence of a microbe-
causative agent;B. contagiousness;
B. Formation of the immune response;
G. Cyclic flow.

5. For septicopyemia characteristic:
A. Hematogenous spread of bacteria in the macroorganism;B.
reproduction bacteria in blood;
G. Formation of secondary purulent foci in the internal organs;D.
microbial circulation toxins in blood.

6. Name the form of the infectious process, in which the pathogen for a long time time is in the body, not showing pathogenic properties and not standing out in surrounding Wednesday:
A. Bacteriocarrier;B.
Latent infection; B. Slow
infection; G. Acute
infection.

7. Naturally acquired immunity:
A. After the introduction of immune sera;B.
post-infectious;
B. Post-vaccination; G.
Transplacental.

8. Acquired artificial active immunity:
A. After the introduction of antitoxic serum;B.
Post-vaccination;
B. Transplacental;G.
Post-infectious.

9. Name the process that protects the body from repeated antigenic interventions:
A. Immune tolerance;B.
immune memory;
B. Hypersensitivity;G.
Immune paralysis.

10. Alternative path activation complement starts:
BUT. Histamine;
B. Components of the bacterial cell wall;AT.
Complex "antigen-antibody";
G. Lipopolysaccharide.

11. Immunoglobulin class G:
BUT. binds complement;
B. Found in mucous secretions;AT. passes
through the placenta;
G. Provides local immunity.

12. Antibodies:
BUT. Are synthesized plasma cells;
B. Able bind complement; AT. Able
neutralize toxins;
G. Agglutinate corpuscular antigens.

13. Check effector cells immune systems:
A. T-killers;B.
T-helpers;
B. Dendritic cells;G. B-
lymphocytes.
14. Phenomenon immunological memory founded on the:
BUT. oppression T-helpers;
B. The absence of certain clones of immune cells;AT.
Absence antigens histocompatibility;
G. education cells memory.

15. name signs hypersensitivity delayed type:
A. Lymphocyte-macrophage reaction;B.
Synthesis Ig E;
B. Participation of T-
lymphocytes;G.
Participation B-
lymphocytes.

16. Immunomodulators:
A. Influence the pathological process through the genome;B.
Possess immunotropic action;
B. Influence the pathological process through the immune system;G. AT
basis mechanism actions are immunological reactions.

17. Antitoxic immunity:
BUT. Absorption toxin macrophages;
B. Development of antitoxic antigens;AT.
Activation of T-killers;
G. Working out antitoxic antibodies.

18. Purpose RPGA:
A. Serodiagnosis of infectious diseases;B. Microbe
identification;
B. Determination of specific antibodies;G.
Titration complement.
MAKE LOGICAL PAIRS: QUESTION AND ANSWER19 .
1. relapse
2. reinfection
3. Superinfection
A. A disease that arose after an infection due to repeatedinfections topics same pathogen;
B. Repeated infection macroorganism topics same pathogen beforerecovery;
AT. Both;

G. Neither then, neither other.

twenty.

1. Passive, naturally acquired immunity

2. Active, naturally acquired immunity

A. Postinfectious; B.
Post-vaccination; B.
Transplacental; G.
Transplant.

21.

1. Antigen in reactions agglutination

2. Antigen in reactions precipitation

A. Molecular; B.
Corpuscular;AT.
Both;
G. Neither then, neither other.

«INFECTION AND IMMUNITY. STRUCTURE AND FUNCTIONS OF ANTIGENS ANDANTIBODIES"

II OPTION

(Choose one or several correct answers)

1. Virulence microbes:
A. Controlled by the genes of the chromosome and plasmids;B. Determine on the sensitive animals;
B. Changes under the influence of external factors;
G. Is specific sign.

2. Adhesins microbes:
A. Hyaluronidase;
B. Endotoxins; AT.
Exotoxins; G. Pili.

3. Properties bacterial endotoxins:
BUT. Lipopolysaccharide nature;
B. They are excreted by bacteria in the process of life;B. Not has specificity actions in body;
G. Under action formalin are turning into toxoid.

4. Periods in development infectious process
A. Prodromal; B.
Reconvalescence;AT.
Incubation;
G. Superinfection.

5. Forms infections:
BUT. reinfection;
B. convalescence;

AT. relapse;
G. Incubation.

6. Author phagocytic theories immunity:
A. Burnet F.;
B. erne N.;
AT. Erlich P.;
G. Mechnikov I.I.

7. Artificially acquired immunity:
A. After the introduction of immune sera;B.
post-infectious;
B. Post-vaccination; G.
Transplacental.

8. Complete antigens:
BUT. specific;
B. Interact with specific antibodies;AT. Have high
molecular weight mass;
G. Possess immunogenicity.

9. AT structure bacterial cells may enter antigens:
A. H-antigen;
B. K-antigen;
AT. O
antigen;
G. HLA antigens.

10. Biological liquid, in which contained lysozyme:
BUT. Tears;
B. Tissue fluid;AT.
Saliva;
G. Serum.

11. Interferons:
A. Produced by fibroblasts and T-lymphocytes;B.
Produced by leukocytes;
B. They have immunomodulatory properties;G.
Possess specific specificity.

12. Immunoglobulin class M:
BUT. binds complement; B.
Passes through the placenta;
AT. Pentamer;
G. It has 2 center binding antigen.

13. Secretory immunoglobulin class A:
A. Provides local immunity;B. Is a
pentamer;
B. Contains a secretory component;G.
passes through the placenta.

14. Local immunity provide immunoglobulins:

A. Class G;
B. Class E;
B. Class D;
G. class
BUT.

15. phagocytes may to be cells:

A. Monocyte;
B. Neutrophil;
B. Alveolar macrophage;G.
Erythrocyte.

16. Specify the forms of immunity in which it participates complement:

A. Mucosal immunity;B. Antitoxic;
B. Antibacterial humoral;G. Humoral
viral.

17. Specify the forms of immunity in which T-killers:

A. Transplant;B.
Antitumor; AT. Antiviral;
G. Antibacterial.

18. List Components RPGA:

BUT. red blood cells;
B. Erythrocyte diagnosticum;AT.
Hemolytic serum;
G. Researched serum.

MAKE LOGICAL PAIRS: QUESTION AND ANSWER19.

1. **Exotoxins**
2. **Endotoxins**
3. **Anatoxins**

A. Do not have toxic properties;B. Produced
by a microbe in environment;
B. Released when bacteria are destroyedG.
Neither one nor the other.

twenty.

1. **Complete antigen**
2. **Defective antigen**

A.
Polysaccharide;
B. Protein;
AT. Both;
G. Neither then, neither other.

21.

1. **Definition molecular antigens**

2. Definition corpuscular antigens
A. Precipitation reaction; B.
Agglutination reaction; AT.
Both;
G. Neither then, neither other.

«INFECTION AND IMMUNITY. STRUCTURE AND FUNCTIONS OF ANTIGENS ANDANTIBODIES"

III OPTION

(Choose one or several correct answers)

1. pathogenicity microbes - this is sign:
BUT. Species;
B. Arose in the process of evolution of
parasitism; AT. Genotypic;
G. Fast changes under influence factors environmental environment.

2. adhesive ability bacteria due to:
BUT. The presence pili;
B. The presence peptidoglycan;
B. The presence of lipoteichoic acids;
G. education protein toxins.

3. characteristic properties endotoxins:
BUT. Strong antigens;
B. They are found in the cell wall of gram-negative bacteria; AT.
Thermolabile;
G. Not sensitive to formalin.

4. Premonitory period - this is period:
A. From the moment of infection to the onset of clinical manifestations of
the disease; B. intensive breeding pathogen together entrance gate;
AT. Liberations macroorganism from microbes;
G. Appearances non-specific symptoms infectious illness.

5. Recurrent manifestations of the disease caused by the same pathogens:
BUT. relapse;
B. Secondary infection;
AT. reinfection;
G. mixed infection.

6. Author humoral theories immunity:
BUT. Burnet
F.
B. erne N.
V. Mechnikov I.I.
G. Erlich P.

7. Active immunity:
BUT. After introductions immune serums;

B. Post-vaccination; B.
Transplacental;G. Post-
infectious.

8. Chemical substances being full-fledged antigens:
BUT. Protein;
B. Mineral salts;AT.
Polysaccharide;
G. Lipid.

9. To factors non-specific resistance relate:
A.
Phagocytosis;
B. Lysozyme;
AT. Complement;
G. Normal microflora.

10. Interferons:
A. Produced by fibroblasts and T-lymphocytes;B.
Produced by leukocytes;
B. They have immunomodulatory properties;G.
Possess specific specificity.

11. Immunoglobulin class M:
BUT. binds complement; B.
Passes through the placenta;
AT. Pentamer;
G. It has 2 center binding antigen.

12. Immunoglobulin class E:
BUT. passes through placenta;
B. Pentamer;
AT. Provides local immunity;
G. Possesses cytophilicity to obese cells and basophils;

13. Monoclonal antibodies:
A. Possess heterogeneity;B. Are
synthesized hybridoma;
B. Synthesized in the human body;G.
Highly specific.

14. AT formation non-specific resistance participatecells:
A. T-helpers;
B.
macrophages;
AT. B-lymphocytes;
G. natural killers.

15. Functions T-helpers:
A. Production of
antibodies;B.
Phagocytosis;
B. The manifestation of
cytotoxicity;G. Regulation immune
response.

16. Neutralization virus outside cells (virion) carried out:
A. Class A immunoglobulins;B.
Interferons;
B. Class G immunoglobulins;G. T
cells.

17. For creation artificial active immunity use:
BUT. Vaccines;
B. Immune sera;AT.
Anatoxins;
G. Tolerogens.

18. List Components reactions precipitation:
BUT. red blood cells;
B. Molecular antigen;
AT. Hemolytic serum;
G. Specific immune serum.
MAKE LOGICAL PAIRS: QUESTION AND ANSWER19.
1. aerogenic mechanism transmission pathogen
2. fecal-oral mechanism transmission pathogen
3. Transmissible mechanism transmission pathogen
BUT. Transfer pathogen through allocation his With faeces and penetration in organism through gastrointestinal tract;
B. Transfer of the pathogen through blood-sucking arthropods;
AT. Both;
G. Neither then, neither other.

twenty.
1. cells, not having antigens histocompatibility
2. cells, having antigens histocompatibility
A.
Lymphocytes;B.
Erythrocytes;
AT. Both;
G. Neither then, neither other.

21.
1. Titer agglutinating serum
2. Titer hemolytic serum
A. The highest dilution of serum that causes complete lysiserythrocytes in the presence of a complement;
B. The minimum serum dilution at which hemolysis occurs;AT. Both;
G. Neither then, neither other.

"INFECTION And IMMUNITY. STRUCTURE And FUNCTIONS ANTIGENS And

ANTIBODIE
S" IV
OPTION
(Choose one or several correct answers)

1. Factors conditioning pathogenicity microbes:
A. Production of aggression
enzymes;B. Toxin formation;
B. Capsule formation;G.
Availability adhesins.

2. Factors pathogenicity bacteria With invasive function:
A. Membranotoxins;B.
Hyaluronidase;
AT. Capsule;
G. Neuraminidase.

3. Describe protein toxins bacteria:
BUT. Are synthesized gram-positive bacteria;
B. Are released into the environment in the process of life;AT. Can
partially secreted;
G. Not possess specificity actions.

4. name forms infections on sign localization pathogen:
A. Manifesto;B.
Sepsis;
AT. relapse;
G. Septicopyemia.

5. Forms of infections characterized by long staymicrobes in macroorganism:
A. Bacteriocarrier;B.
persistence;
AT. relapse;
G. Secondary infection.

6. Peripheral bodies immune systems:
A. Bone marrow;
B. thymus;
AT. Plasma cells;
G. Lymphatic nodes.

7. Passive immunity:
A. After the introduction of immune sera;B.
Post-vaccination;
B. Transplacental;G.
Post-infectious.

8. Haptens:
A. Determined in the agglutination reaction;
B. interact With antibodies;
B. Induce an immune response in the macroorganism;
G. Have low molecular weight mass.

9. Activation complement maybe start off With component:
BUT. C1;
B. C2;
AT. C3;
G. C4.

10. Immunoglobulin class M:
BUT. binds complement; B.
Passes through the placenta;
AT. Pentamer;
G. It has 2 center binding antigen.

11. Immunoglobulin class E has tropism to:
A. Basophilam;
B. macrophages;
B. mast cells;G.
fibroblasts.

12. Monoclonal antibodies:
BUT. High specific;
B. Possess structural heterogeneity;
B. Used as diagnostic preparations;G. Produced by
macrophages.

13.Phenomena of the immune response, in which B-lymphocytes:
A. Production of
antibodies;B.
Phagocytosis;
B. Immunological memory;G.
killer function.

14. Specify immunocompetent cells withcytotoxicity:
A. Natural killers;B. T-
helpers;
B. T-killers;G.
Basophils.

15. For antibacterial immunity characteristically participation:
A. Complement;
B. Phagocytes;
AT. Antibodies;
G. B-lymphocytes.
16. signs hypersensitivity I type (anaphylaxis):
A. Immediate reaction development;
B. Possibility of desensitization;AT.
Participation B-lymphocytes;
G. Participation Ig E.

17. For creation artificial passive immunity use:
BUT. Vaccines;

B. Immune sera;AT.
Immunoglobulins;
G. Adjuvants.

18. Purpose reactions precipitation:
A. Determination of unknown antibodies against a known antigen;B. Definition quantity erythrocytes;
B. Determination of an unknown antigen by known antibodies;G. Definition titra complement.
MAKE LOGICAL PAIRS: QUESTION AND ANSWER19.
1. **Anthroponosis**
2. **zooanthroponosis**
3. **Sapronose**

A. The source of infection is a person; B. The source of infection is an animal;AT. Both;
G. Neither then, neither other.
twenty.
1. **Flagellate antigen bacteria**
2. **Somatic antigen bacterial cells**

A. H-antigen;
B. O-antigen;
AT. Both;
G. Neither then, neither other.

21.
1. **Bacterial diagnosticum**
2. **Diagnostic serum**

A. Contains specific antibodies;B. Antigen in corpuscular form; AT. Both;
G. Neither then, neither other.

TEST TASKS

ON MICROBIOLOGY, VIROLOGY, IMMUNOLOGISTS - MICROBIOLOGY OF THE ORAL CAVITY
FOR DENTAL FACULTY(2 WELL, AUTUMN SEMESTER)

Vladikavkaz

"THE ACTIVATIVES DISEASES, CALLED PATHOGENIC COCCAS. CAUSES OF BACTERIAL INTESTINAL INFECTIONS»I OPTION
(Choose one or several correct answers)

1.Purulent-inflammatory diseases, called conditionally pathogenic cocci are characterized by:
BUT. Various localization;
B. Diversity clinical forms;
AT. Decrease resistance macroorganism; G. Weak immune answer.

2. For prevention which infections _ maybe to be and used toxoid? BUT. Staphylococcal;
B. Streptococcal; AT. gonococcal;
G. Meningococcal.

3. Nutrients environment, which can use for isolation of opportunistic pathogens staphylococci:
BUT. ten % yolk - salt agar;
B. blood agar;
AT. Whey agar;
G. MPA.

four. ***At what diseases applied method "provocations"***
BUT. Rheumatism;
B. meningitis;
AT. gonorrhea;
G. Syndrome toxic shock.

5. Which from microorganism coccoid forms produces toxin "syndrome toxic shock":
A. Pneumococcus;
B. Staphylococcus;
B. Streptococcus;
G. Meningococcus.

6. Material for bacteriological method research at meningococcal infections:
BUT. Liquor;
B. Smear from nasopharynx;
AT. Blood;
G. Serum.

7. Properties bacteria kind Salmonella:
BUT. Produce H_2S;
B. Lactose-negative; AT. mobile;

G. Gram-positive.

8. Material for bacteriological research at cholera:
BUT. Blood;
B. vomit masses;
AT. Urine;
G. Excreta;

9. For serological method diagnostics abdominal typhus apply reactions:
BUT.
RNGA;
B.
ELISA;
AT. PCR;
G. RA on the glass.

10. diarrheagenic intestinal sticks:
A. Produce enterotoxins;B.
Lactose-positive;
B. Have pathogenicity plasmids;G.
They have endotoxin.

11. Nutrients environments for allocation and identification pathogen shigellosis:
A. Ploskireva;
B. Kligler;
AT. Endo;
G. alkaline peptonic water.

12. Properties bacteria kind Shigella
BUT. form disputes;
B. Lactose-negative;AT.
Have N- antigen;
G. Not produce H2S . _

13. Factors pathogenicity pathogens cholera:
A. Invasive outer membrane proteins;B.
Enterotoxin;
B. Toxin Shiga; G.
Neuraminidase.

14. Serological method diagnostics abdominal typhus allows:
A. Assess the dynamics of the
disease;B. Reveal bacteriocarrier;
AT. Spend retrospective diagnostics;
G. Define biochemical properties pathogen.

15. Material for bacteriological research on the 1st week diseasesabdominal typhus:
BUT. Urine;
B. Excreta;
AT. Serum;
G. Blood.

16. Useful functions intestinal sticks for macroorganism:
A. Antagonist of pathogenic putrefactive microflora;B.
Not breaks down fiber
B. Participate in the synthesis of
vitamins;G. Partially splits fiber.

17. Methods microbiological diagnostics abdominal typhus on the 3rd weekdiseases:
BUT. Bacterioscopic;

B. Bacteriological;AT.
Biological;
G. Serological.

18. Development diarrheal syndrome at salmonellosis is result:
BUT. Actions enterotoxin;
B. Reproduction of Salmonella in epithelial cells of the surface epithelium;AT.
Activation by endotoxin cascade arachidonic acids;
G. Actions Shiga-like toxin.

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

19.
1. Unfinished phagocytosis:
2.Forms chains cells in bouillon culture:
3.Maybe produce enterotoxin:
4.calls blennorey:
A. S. aureus; B. S.pyogene s; AT. N. gonorrhoeae.

twenty.
1. Cholera:
2. Shigellosis:
3. Salmonellosis:
4. Intestinal escherichiosis:
A.ETKP;
B. S enteritidis;
AT. S.typhi;
G. V.cholerae;D. S. sonnei.

21.
1. Agglutinated by polyvalent escherichial OK-serum(antibodies to 0157):
2. Cause purulent-inflammatory diseases of variouslocalization:
3. produce enterotoxins:
4. Possesses psychrophilia:
A. Conditionally pathogenic Escherichia coli;
B. diarrheagenic intestinal sticks;
B. Both;
G. Neither then, neither other.

"THE ACTIVATIVES DISEASES, CALLED PATHOGENIC COCCAS. CAUSES OF BACTERIAL INTESTINAL INFECTIONS»II OPTION

(Choose one or several correct answers)

1. Gonococci and meningococci in clean culture and researched material usuallylocated:
A. Single;

B. Chains;AT.
in pairs; G.
Clusters.

2. Material for bacteriological research at scarlet fever:

A. Blood;
B. Urine;
AT. Serum; G.
Smear from
pharynx.

3. antigens staphylococci are:

BUT. Protein BUT;
B. Ther X about ev s e ki layer t s;
AT. toxins;
G. Lipopolysaccharide.

4. For treatment: heavy acute staphylococcal infections (sepsis and etc.)can use:

BUT. Immunoglobulin;
B. killed vaccine;
AT. Hyperimmune plasma;
G. live vaccine.

5. For what pathogen typical unfinished phagocytosis?

BUT. Staphylococcus aureus;B.
C **tre Fri about to to to;**
B. Staphylococcus epidermidis;G.
Gonococcus.

6. AT what forms maybe leak meningococcal infection?

BUT. Meningitis;
B. Nasopharyngitis;
B. "Healthy" carriage;G.
Furunculosis.

7. What kind methods are used for diagnostics abdominal typhus?
BUT. Bacterioscopic;
B. Bacteriological;AT.
Biological;
G. Serological.

8. Properties bacteria kind Escherichia:
BUT. Gram-positive;
B. Lactose-positive;AT.
form disputes;
G. Not produce H 2 S.

9. What properties do bacteria have? Enterobacteriaceae:BUT. Gram
negative sticks;
B. Not form dispute;
AT. Optional anaerobes;

G. Have grain volutin.

10. name factors pathogenicity shigella:
A. Invasive outer membrane proteins;B. W, V
antigens;
B. Shiga-like toxin;G.
Cholerogen.

11. What pathogenicity factors does the causative agent of cholera
have:BUT. Invasive proteins outer membrane;
B. Enterotoxin;
AT. Toxin Shiga;
G. Neuraminidase.

12. The serological method for diagnosing typhoid fever allows:BUT.
Assess the dynamics infectious process;
B. Reveal bacteriocarrier;
B. Conduct a retrospective diagnosis;G.
Seropify the pathogen.

13. What medium is used to isolate the cholera pathogen?BUT.
Alkaline peptone water;
B. Kligler;
AT. Alkaline agar;
G. biliary bouillon.

14. What are the properties of diarrheal E. coli?BUT. The presence of
plasmids virulence;
B. Lactose negativity; B.
Antigenic structure;G.
Products H2S . _

15.name drugs for specific prevention abdominal typhus:BUT. Chemical
vaccine;
B. Inactivated corpuscular vaccine;AT.
Bacteriophage;
G. Anatoxin.

16. What drugs are used to treat and prevent dysentery?BUT. Intesti
bacteriophage;
B. Dysenteric bacteriophage;AT.
Vi bacteriophage;
G. Pyocyneus bacteriophage.

17. What methods of diagnosing cholera are accelerated?BUT.
Immobilization in the dark field;

B. Dark field agglutination;AT.
Method Ermolyeva;
G. Immunofluorescent method.

oneeight. Hazovite semoatars holernoGohweebryona?
BUT. OGa
wa;B. Ying
aba;
V. Giko shima;
G. XolepeWith
uiWith.

COMPOSE BRAIN TEASER COUPLES: QUESTION ANSWER

19.
1. Lecitovitellase activity on the yolk-salt agar:
2. Availability plasma coagulase:
3. Absence plasma coagulase:
4. Pigmentation:
A. Staphylococcus aureus;B.
Streptococci;
B. Both;
G. Neither then, neither
other.

twenty.
1. Cholera:
2. Paratyphoid BUT:
3. Intestinal escherichiosis:
4. Shigellosis:
A. S.
dysenteriae;B.
V. cholerae;
B. S.
typhimurium;G.
EPKP;
D. S. paratyphi.

21.
1. Applies to serogroup O1:
2. sustainable to polymyxin:
3. sensitive to bacteriophage FROM:
4. Produces enterotoxin:
A. biovar cholerae;
B. Biovar eltor;
AT. Both;
G. Neither then, neither other.

"THE ACTIVATIVES DISEASES, CALLED PATHOGENIC COCCAS. CAUSES OF BACTERIAL INTESTINAL INFECTIONS»III OPTION
(Choose one or several correct answers)

1. Methods of microbiological diagnosis of pneumococcal infection :BUT.
Bacteriological;
B. Serological;

B. Biological;G.
Allergic.

2. What bacteria are located in pure culture and test material?in pairs?
A. Pneumococci;
B. Staphylococci;
AT. meningococci;
G. All the above right.

3. What material for microbiological research should be taken from the patient at suspicion of gonorrhea?
A. Detachable urethra;
B. Vaginal swab; AT.
smear from pharynx;
G. Rectal smear.

4. After what disease of streptococcal etiology is formed a strongimmunity?
A. Tonsillitis;
B. Rheumatism;
B. Scarlet
fever;G. Sepsis.

5. name pathogen toxin scarlet fever:
A. Fibrinolysin;B.
Erythrogenin; AT.
Erythrolysin;
G. Plasmocoagulase.

6. Basic path transmission blenorei newborns:
BUT. Contact;
B. Contact household;
AT. Sexual;
G. Water.

7. What properties are characteristic of representatives of the Enterobacteriaceae family:BUT. Need alkaline nutrient media;
B. Gram-negative rods;AT. form
disputes;
G. fermented carbohydrates.

8. What media are used to isolate and identify the pathogencolienteritis?
BUT. Endo;
B. Kligler;
AT. Levin;
G. biliary bouillon.

9. reactions, used for serological method diagnostics abdominaltyphus:

BUT.
RNGA;
B. ELISA;
AT. deployed RA;
G. RA on the glass.

10. By what properties differ biovars vibrio cholerae and eltor?
BUT. By reactions agglutination With *01* - serum;
B. By sensitivity to polymyxin;
AT. By relation to serum Inaba;
G. By sensitivity to specific bacteriophages.

11. The development of diarrheal syndrome in salmonellosis is associated with:BUT. action enterotoxin;
B. Reproduction of salmonella in epithelial cells of the surface epithelium;AT. Activation endotoxin cascade arachidonic acids;
G. blocking neurovascular receptors.

one2. Hazovite semoatars holerno Goh weebryona?
BUT. OGa
wa;B. Ying
aba;
V. Giko shima;
G. XolepeWith
uiWith.

***13.* Pabout tolikem St.oystinam rahwhether chautWithi diareegenowe and atWithloclearly -PataboutGennese kishehns Palaboutchki?**
BUT. PnowRofiflaxst;
B. SpoWithupdateosbe utilizi roinat lAct
ram;AT. spaboutcobnost produce H_2S;
***G*.** AntiGennand I structuretura.

onefour. Mnoones microbiolaboutgiheucoy diagnostiki brYushaboutth tand F na3-th nunitsatee habolevaniI:
BUT. BActeriologiheu
cue; B. BActeriaboutsave
CheWithcue;AT. Biological;
G. Serologiheucue.

one5. Ha Pepinm thisupe baktериolaboutgiChesk aboutth andsslunitsovaniI at infekciI, inzvanns etcunitsstavitelami Witheatacetina kishehnsXbActeriy, Paboutsein isetcawenii prodinauditionWithI on the Wedfood:
BUT. MPA;
B. KliGepa;
AT. peptone water;
G. Lactosis aboutcodepandashie diffepencialbut-diagnostiheskiesRunitss.

16. what kind **Wedunitss andspoehhatut at inidateeresearch institutes and identandficationinaboutzbfishesel I Withalumheelle per:**
BUT. In andWithmut-Withstbfitnth aGar;
B. PlaboutskyrеIn

a;AT. Toleagues l
epa;

G. Selenito inth bst yon .

17. Preparations for specific prevention abdominal typhus:
BUT. Chemical vaccine;
B. inactivated corpuscular vaccine;AT.
Bacteriophage;
G. Anatoxin.

18. Material for bacteriological research at cholera:
BUT. Blood;
B. vomit masses;
AT. Urine;
G. Excreta;
MAKE LOGICAL PAIRS: QUESTION AND ANSWER19.
1. Absence tropism
2. Low enzymatic activity
3. High enzymatic activity
4. Growth on the saline environments
5. Growth only on the rich protein environments
A. Pathogenic Neisseria;B.
Staphylococci;
AT. Both;
G. Neither then, neither other.

twenty.
1. Transcytosis of the epithelium of the small intestine with reproduction in the regionallymphoid intestinal tissue:
2. Invasion and intracellular reproduction in the epithelium of the colonintestines:
3. attachment and colonization surfaces epithelium thin intestines:
BUT. Shigella;
B. Salmonella;
B. Vibrio cholerae;G.
EPKP.
21.
1. split mannitol:
2. More often transmitted water by:
3. More often transmitted contact household by:
4. breeds in fabrics intestines:
BUT. S. flexneri;
B. S. dysenteriae;
B. Both;
G. Neither then, neither other.

"PATHOGENS OF DISEASES CAUSED BY PATHOGENIC COCCAS. PATHOGENS BACTERIAL INTESTINAL INFECTIONS"

IV **OPTION**

(Choose one or several correct answers)

1. As **if pathogenic cocci that can cause diseases of variouslocalization:**
A. Staphylococci;
B. Pneumococcus;
B. Streptococci; G.
Meningococcus.

2. Biological properties pathogen gonorrhea:
A. Sensitive to environmental factors;B.
pathogenic only for person;
B. Grows on nutrient media supplemented with human protein;G. Mobile.

3. Main methods diagnostics pneumococcal infections:
BUT. Allergic;
B. Bacteriological;AT.
Serological;
G. Biological.

4. Multiple medicinal resistance at staphylococci conditionedpresence:
BUT. Capsules;
B. Ent-plasmids;
AT. Hyaluronidase;
G. R-plasmids.

5. What kind cocci sensitive to optochin and bile:
A.
Streptococcus; B.
Staphylococcus;
AT. Gonococcus;
G. Meningococcus.

6. ®– **hemolytic streptococci are characterized by :**A.
They have adhesins - a complex of lipoteichoic acid; B.
Motionless, dispute and capsules not forms;
AT. produce enzymes: streptokinase and hyaluronidase;G.
Release toxins **.**

7. What kind antigens virulence there is in E.coli?
BUT. E;
B. W;
AT. H;
G. K;
D. Oh

8. What properties possess bacteria kind Shigella?
BUT. form disputes;
B. Lactose-negative; B.
Possess H-antigen;G. Not
produce H_2S . _

9. What kind vaccines are used for specific prevention abdominal typhus?
BUT. O-vaccines;
B. AKSD vaccines;
B. Typho-paratyphoid-tetanus vaccines;G.
BCG.

10. Pathogen abdominal typhus strikes:
A. The mucous membrane of the stomach;B. Epithelium of the small intestine;AT. Heart;
G. Kidneys.

11. causative agents salmonellosis are:
A. S. enterica;B. S. typhi;
B. S. typhimurium;
G. S. enteritidis.

12. Nutrients environments for allocation and identification pathogen shigellosis:
A. Ploskireva;
B. Kligler;
AT. Endo;
G. alkaline peptonic water.

13. Methods of microbiological diagnosis of typhoid fever at 3 weeksdiseases:
A. Bacterioscopic;B. Bacteriological; AT. Biological;
G. Serological.

14. Serological method diagnostics abdominal typhus allows:
A. Assess the dynamics of the disease;B. Reveal bacteriocarrier;
AT. Spend retrospective diagnostics;G. Serotype pathogen.

15. Material for bacteriological research at shigellosis:
BUT. Blood;
B. Urine;
AT. Excreta;
G. Serum.

16. What kind environments use for accumulation cholera vibrio?
A. Sugar broth;B. biliary bouillon;
AT. Whey bouillon;
G. alkaline peptonic water.

17. What kind antigens have salmonella abdominal typhus?
BUT. O;

B. H;
AT. Vi;
G. TO;
D. W.

18. For serological method diagnostics typhoid fever apply reactions:
BUT.
RNGA;
B.
ELISA;
AT. PCR;
G. RA on the glass.

LEAVE BRAIN TEASER COUPLES: QUESTION ANSWER
19.
1. erysipelatous inflammation:
2. Blennorea:
3. Syndrome toxic shock:
4. Rheumatism:
A. Staphylococcu
s;B. Gonococcus;
B. Group A beta-hemolytic streptococci;G.
Pneumococcus.
twenty.
1. attachment and colonization surfaces epithelium thin intestines:
2. Transcytosis epithelium thin guts With breeding in regional lymphoidfabrics intestines:
3. Invasion and intracellular reproduction in epithelium thick intestines:
BUT. Shigella;
B. Salmonella;
AT. cholera vibrio;
21.

1. Basic path transmission - contact household:
2. Basic path transmission - water:
3. Produces a Shiga-like toxin:
4. Works out Shiga -toxin:
BUT. S. sonnei;
B. S.
dysenteriae;AT.
Both;
G. Neither then, neither other.

"THE CAUSES OF DIPTHTERIA, WHOOPING COUGH, PARACCOUSH, TUBERCULOSIS,ZOONOUS INFECTIONS"

I OPTION

(Choose one or several correct answers)

1. What form maybe have pathogen diphtheria?
BUT. coccoid;
B. Polymorphic sticks;B.
Curved (2-3 curls); G.
Branching.

2. Microscopy pathogen diphtheria carry out:
A. When stained according to Tsil -
Nelsen;B. AT dark field vision;
B. When stained according to
Neisser;G. negative way.

3. For coloring mycobacteria use method:
BUT. Ozheshko;
B. Tsilya - Nelsen;
AT. Leffler;
G. Romanovsky-Giemsa;
D. Neisser.

4. The sequence of stages of bacteriological research method fordiphtheria:
BUT. Definition toxicity;
B. Sowing the test material on special media;AT. The study
biochemical properties;
G. Reseeding colonies for receiving clean culture.

5. Toxicity diphtheria sticks determine With help reactions:
A. Agglutinations on glass;B.
hemagglutination;
B. Ring precipitation;G.
precipitation in gel.

6. name main methods microbiological diagnostics diphtheria:
A. Microscopic;B.
Biological;
B. Bacteriological;G.
Allergic.

7.Methods microbiological diagnostics whooping cough
A. Bacterioscopic;B.
Bacteriological; AT.
Allergic;
G. Serological.

8. What method is used for accelerated bacteriological diagnosistuberculosis?
BUT. Homogenization;
B. Microcultivation;AT.
precipitation;
G. Method Price.

9. Vaccine for specific prevention tuberculosis:
BUT.
killed;
B. Live;
B. Anatoxin;
G. BCG.

10. Distinguish the causative agent of tuberculosis from the causative agent of leprosy duringmicrobiological diagnostics can be done by:
BUT. acid resistance;
B. Rostu on artificial nutrient media;AT. results
PCR;
G. results bioassays.

11. For prevention leprosy apply:
A. Dry purified tuberculin;B.
Integral lepromin;
AT. DPT;
G. BCG.

12. For serodiagnosis brucellosis apply:
BUT. reaction Vidal;
B. reaction Wright;
B. Weil-Felix reaction;G.
ELISA.

13. For serodiagnosis tularemia apply:
BUT.
RNGA;
B. RSK;
AT. REEF;
G. deployed RA.

14. Nutrients environments for cultivation anthrax bacilli:
BUT. JSA;
B. Alkaline agar;
AT. biliary bouillon;
G. MPA.

15. Express- diagnostics plague:
A. Gas-liquid chromatography;B.
REEF;
B. Phage typing;G.
Phage diagnostics.

16. Vaccines for prevention zoonotic bacterial infections:
BUT. killed;
B. Anatoxin; B.
Chemical;G.
Live.

17. The form plague, source infections at which is only human:
BUT.
bubonic;
B. Intestinal;
AT. Skin-bubonic;
G. Pulmonary.

18. Infection of people brucellosis going on :
A. In contact with sick animals;B. Through
milk and dairy products;

AT. Through postpartum discharge; G.
At contact With sick people.

19. Make up brain teaser couples: question answer

1. split urea	A. Pathogen diphtheria
2. Not possess cystinase	B. Conditionally pathogenic corynebacteria
3. Not have urease	B. Both
4. Work out cystinase	G. Neither then, not other

twenty. Make up brain teaser couples: question answer

1. M. leprae	A. located intracellularly, forming balls
2. M. bovis	B. Gram negative cocci
3. M. tuberculosis	B. Long thin sticks
	G. Short thick sticks

21. Make up brain teaser couples: question answer

1. Gram positive sticks	A. Pathogen Siberian ulcers
2. Gram negative sticks	B. Pathogen brucellosis
3. Maybe form capsule	B. Both
4. moving	G. Neither then, neither other

"THE CAUSES OF DIPTHTERIA, WHOOPING COUGH, PARACCOUSH, TUBERCULOSIS,ZOONOUS INFECTIONS"

II OPTION

(Choose one or several correct answers)

1. What morphological structures has pathogen diphtheria?
A. Spores;B.
Pilyami;
AT. flagella;
G. grains volutin.

2. Ways transmission pathogen tuberculosis:
A. Airborne;B. Sexual;
B. Air and dust;G.
Transmissible.

3.name main sources tuberculosis:
A. Patients with an open form of tuberculosis; B.
Patients with a closed form of tuberculosis; B.
Patients farm animals;G. Marine pigs.

4. Which material take for research at pulmonary forms tuberculosis?
BUT. Sputum;
B. Pleural liquid;

B. Flushing water of the
bronchi;G. ascitic liquid.

5. diseases, called mycobacteria:
A. actinomycosis;
B. Tuberculosis;
AT. Deep mycoses;
G. Leprosy.

6. Try mantoux put for:
A. Selection of persons to be revaccination;B.
medical purpose;
AT. Prevention tuberculosis;
G. control efficiency treatment.
7. What kind drugs use for specific prevention tuberculosis?
BUT. ZhKSV-E;
B. BCG-M;
AT. DPT;
G. BCG.

8. At diagnostics diphtheria do sowing researched material on the Wednesday:
BUT. RU;
B. Endo;
AT. Levin;
G. Clauberg; D.
Ploskirev.

9.Factors virulence pathogen tuberculosis are:
BUT. Capsule;
B. Cord factor;
B. Endotoxin; G.
Exotoxin;
D. Lipids cellular walls.

10. What kind methods "enrichment » apply at microscopic diagnosticstuberculosis?
A. Homogenization and
precipitation;B. Method Price;
AT. Method flotation;
G. Method deep cultivation.

11. Factors pathogenicity pathogen whooping cough
A. Filamentous hemagglutinin;B.
whooping cough toxin;
B. Extracellular adenylate cyclase;G.
Endotoxin.

12. Property pathogen whooping cough
A. Demanding on nutrient media;B.
Biochemically few active;
B. Highly sensitive to environmental factors;G. grows on
simple environments.

13. On the what environments growing pathogen whooping cough?
BUT. MPA;
B. Caseinovo - coal agar;AT.
Wednesday Clauberg;
G. Wednesday Borde-Jangu.

14. What kind epidemiological peculiarities characteristic for leprosy?
A. The source is a sick person;B.
Contact path transfers;
B. Airborne transmission;G. Source -
rodents.

15. What principles underlie the clinical and immunological classificationleprosy?
A. Histological manifestations;B.
Bacterioscopic data;
B. Results of a skin-allergic test;G.
Bacteriological data.

16. What methods allow to distinguish the causative agents of tuberculosis from the pathogenleprosy at carrying out microbiological diagnostics?
BUT. Coloring on Tsil-Nelsen;
B. Growth on artificial nutrient media;AT.
staging skin-allergic samples;
G. Definition pathogenicity for guinea pigs and rabbits.

17. Methods microbiological diagnostics plague:
A. Bacteriological; B.
Bacterioscopic;AT.
Biological;
G. Serological.

18. Methods differentiation species brucella:
BUT. Need in CO2;
B. Tinctorial properties;
B. Bacteriostatic action of cells;G.
Antigenic structure.

19. Make logical pairs: question-answer
Susceptible animals:

1. M. Bovis	A. Marine pigs
2. M. leprae	B. rabbits
3. M. tuberculosis	B. armadillos

21. Make up brain teaser couples: question answer

1. M. leprae	A. Leprosy
2. M. kansasii	B. Mycobacteriosis
3. M. africanum	B. Tuberculosis
4. M. Avium	

21. Make up brain teaser couples:

Main methods microbiological diagnostics

BUT. Bacterioscopic
B. bacteriological
AT. Serological
G. Biological
Allergological

1. Tularemia;
2. Siberian ulcer;
3. Both.
4. Neither other.D.

"THE CAUSES OF DIPTHTERIA, WHOOPING COUGH, PARACCOUSH, TUBERCULOSIS,ZOONOUS INFECTIONS"

III OPTION

(Choose one or several correct answers)

1. Infection of people brucellosis happens:
A. In contact with sick animals;B. Through milk and dairy products; AT. Through postpartum discharge;
G. At contact With sick people.

2. For productions samples Burne apply:
BUT. Pestin;
B. Antraksin;
AT. Tulyarin;
G. Brucellin.

3. How in clean culture located diphtheria sticks?
BUT. Messy;
B. Location cells in form chains;
AT. Location cells in form "palisade";G. Location cells in form V, x.

4. Ways transmission diphtheria:
A. Airborne;B. Contact; B. Alimentary;
G. Transmissible.

5. What material for microbiological research should be taken fromsick on suspicion for diphtheria?
A. Mucus from the pharynx; B. Film from the pharynx;
B. Mucus from the nose; G. Blood.

6. Nutrients environments for cultivation pathogen diphtheria:
BUT. MPA;
B. Tellurite blood agar;AT. Yolk-salt agar;

G. Rolled up serum.

7. How conditioned acid resistance mycobacteria?
A. A large amount of peptidoglycan;B. The
presence tuberculin;
AT. The presence LPS in outdoor
membrane;G. Mycolic acids.

8. For cultivation M. leprae carry out:
A. Blood agar culture;B.
Infection armadillos;
B. Infection of a rabbit in testis;
G. Infection clothes lice.

9.characteristic location pathogen leprosy in affected fabrics:
A. In intercellular spaces;B.
Intracellularly;
AT. AT form long chains;
G. Forms clusters cells in form balls.

10. Method accelerated bacteriological diagnostics tuberculosis:
BUT. Homogenization;
B. Microcultivation;AT.
precipitation;
G. Method Price.

11. What kind cultural properties characteristic for M.tuberculosis?
A. Dry colonies with uneven edges;B. R-
shape;
C. Delicate wrinkled film on the surface of the liquid nutrient
environment;
G. Smooth even colonies white or gray colors.

12. Which antigen use pi staging reactions Mitsuda?
BUT. autoclaved suspension pathogen leprosy, received throughhomogenization
contents of leprosy;
B. Lepromin-A;
AT. Integral lepromin;
G. Dry purified tuberculin.

13. Planned specific diphtheria prophylaxis postponed until 3-4monthly child's age in links with:
BUT. Admission secretory Ig A With milk mothers;
B. Lack of formed normal microflora;AT. Working out high
credits own antibodies;
G. Availability Ig g, received from mothers through placenta.

14. For the treatment of the chronic form of what zoonotic infections are used killed vaccines?
BUT. Plague;
B. Tularemia;
AT. Siberian ulcer;

G. Brucellosis.

15. bacteria virulent in R-shape:
BUT. Brucella;
B. Anthrax bacilli;AT.
Francisella;
G. Yersinia.

16. Most often encountered pathogens brucellosis:
A.V. melitensis;
B. B. ovis;
AT. B.
abortus;
G. b. neotome.

17. Factors pathogenicity brucella:
A. Endotoxin;B.
Exotoxin;
B. Enzymes of
aggression;G. Capsule.

18. For serodiagnosis brucellosis apply:
BUT. reaction Wright;
B. Opson-phagocytic reaction;AT.
Huddleson reaction;
G. RPGA.

19. Make up brain teaser couples: question answer

1. Biovar gravis	A. Forms large smooth red colonies
2. Biovar mitis	B. Forms small black colonies
	B. Forms large rough gray colonies.

twenty. Make up brain teaser couples: question answer

1. M. Bovis	A. Marine pigs;
2. M.leprae	B. rabbits;
3. M. tuberculosis	B. Battleships.

21. Make up brain teaser couples: question answer
1. Allocate very a lot of pathogens
2. Allocate a little pathogens
3. More dangerous for surrounding
4. Can to be source infections at diphtheria

BUT. Sick diphtheria;
B. Bacteriocarriers pathogens of diphtheria;AT.
Both;
G. Neither then, neither other.

"THE CAUSES OF DIPTHTERIA, WHOOPING COUGH, PARACCOUSH, TUBERCULOSIS,ZOONOUS INFECTIONS"

IV OPTION

(Choose one or several correct answers)

1. For cultivation pathogens tuberculosis use nutritious environments:
A. Levenshtein-Jensen;B.
Levin;
AT. Petragnani;
G. Clauberg.
2. characteristic location diphtheria sticks in clean culture:
BUT. bunches;
B. AT form chains;
AT. AT form "palisade";
G. Under angle friend to friend.

3. Antidiphtheria antitoxic serum applied for:
A. Emergency prevention;B.
Planned prevention; AT.
treatment;
G. Dermal staging - allergic samples.

4.Main tests, used for identification diphtheria sticks:
A. Test for cystinase;B.
samples on the indole;
AT. Try on the
urease;G. Try on the
H2S.

5. Properties pathogen whooping cough
A. Gram-negative rod;B. Forms
exotoxin;
B. Biochemical is not very active;
G. Generates controversy.

6. On the what kind bodies renders pathological action diphtheria toxin?
A. Cardiac muscle;B.
kidneys;
AT. Adrenals; G.
nervous ganglia.
7. On the what environments can cultivate pathogen diphtheria?
BUT. MPA;
B. Tellurite blood agar;AT. Yolk-
salt agar;
G. Rolled up serum **.**

8. What properties has pathogen whooping cough?
A. Demanding on nutrient media;B.
Biochemical few active;
B. Highly sensitive to environmental factors;G. grows on
simple environments.

9. Ways transmission pathogen leprosy:
A. Airborne;B. Sexual;
V. Contact;
G.Transmissive.
10. Prevention tuberculosis held introduction:

A. Anatoxin; B.
Antitoxin;V.
Tuberculin;G.
BCG.

11. Biological models for cultivation pathogen leprosy:
A. Guinea pigs;B.
rabbits;
B. Golden hamsters;G.
Battleships.

12. Methods of "enrichment" of the studied material with microscopic diagnostics tuberculosis:
A. Homogenization and
precipitation;B. Method Price;
B. Flotation method;
G. PCR.

13. For prevention leprosy apply:
A. Dry purified tuberculin;B.
Integral lepromin;
AT. DPT;
G. BCG.

14. What kind epidemiological peculiarities characteristic for leprosy?
A. The source is a sick person;B.
Contact path transfers;
B. Airborne transmission;G. Source -
rodents.

15. Vaccine, applied for prevention brucellosis:
BUT. STI;
B. Living corpuscular Elbert-Gaisky;AT. EV;
G. live corpuscular Vershilova (VA-19A).

16. Material from the patient for bacteriological research at tularemia:
BUT. Blood;
B. Puncture of lymph
nodes;AT. Sputum;
G. Serum blood.

17. Factors pathogenicity anthrax bacilli:
A. Pili; B.
disputes;
B. Endotoxin;G.
Exotoxin.

18. The pearl necklace test on penicillin medium is used toidentification:
BUT. Yersinia;
B. Francisell;
AT. Brucella;

G. Anthrax bacilli

19. Make up brain teaser couples: question answer

1. b. Pertussis A. parapertussis;
2. L. Pneumophila b. Whooping cough;
3. b. Parapertussis B. Paratyphoid;
   G. Legionellosis.

20. Make logical pairs: question-answer
Susceptible animals:

1. M. Bovis A. Marine pigs
2. M. leprae B. rabbits
3. M. tuberculosis B. armadillos

21. Morphological and tinctorial properties of causative agents of bloodinfections:

1. Gram positive sticks A. Yersinia plague
2. Gram negative sticks B. Pathogen tularemia
3. Ovoid form B. Both
4. Forms subterminal controversy g . Neither then, neither other

«PATHOGENS OF ANAEROBIC CLOSTRIDIAL INFECTIONS.PATHOGENIC SPIROCHETES AND SPIROCHETOSES. MYCOPLASMS. CHLAMYDIA"

I OPTION

(Choose one or several correct answers)

1. The onset of tetanus is caused by the ingestion ofbody:
A. Brucella melitensis; B. Exotoxins
Clostridium difficile;
B. Clostridium tetani and its exotoxin;G.
Clostridium novyi through the wound.

2. By type breathing clostridia:
BUT. obligate anaerobes;
B. Optional anaerobes;AT.
obligate aerobes;
D. Facultative aerobes;D.
Microaerophiles.

3. To pathogens gas gangrene relate:
A. Clostridium perfringens;
B. Clostridium tetani;
B. Clostridium botulinum;
G. Clostridium novyi.

4. Ways transmission botulism:
A. Parenteral;B.
Wound;
B. Contact household;G.
Food.

5. pathogens tetanus are:
A. Fusobacteria;B.
Clostridia; AT.
Bacteroids; G.
Peptococci.

6. For clostridia characteristic:
A. Capsule formation;B.
Education dispute;
C. Presence of volutin grains;
G. Anaerobic type of
breathing.

7. Immunity after transferred botulism:
BUT. Antitoxic; B.
Antibacterial;AT. Local;
G. Not is being formed.

8. Non-spore-forming anaerobes:
BUT. Bacteroids;
B. Clostridia;
AT. Fusobacteria;
G. Waylonelles.

9. Immunobiological drugs for prevention and treatment botulism:
A. Antitoxic serum;B. DPT;
B. Tetraanatoxin;G.
ADS.

10. Pathogen syphilis:
A. T. pertenue;
B. T. pallidum;
B. N.
gonorrhoeae;G. N.
meningitidis.

11. Describe pathogen leptospirosis:
BUT. Thin light threads With bent ends;B. Are
stained in violet color;
B. Number of curls 20-40;
G. form cysts.

12. Sustainability pathogen syphilis in environmental environment:
BUT. sustainable to disinfectants;
B. Weakly resistant to the environment;
AT. sustainable to elevated temperature;

G. sustainable to drying out.

13. For syphilis characteristic:
A. Penetration of the pathogen through skin and mucous membranes;B. Infection transplacental by;
AT. leaking cyclically;
G. leaking in form sepsis.

14. What properties possess spirochetes?
BUT. Have thin cellular wall;B. Gram-negative;
B. Thin spirally curved cells; G. Have cytoplasmic cylinder.

15. What kind properties characteristic for chlamydia?
A. Gram-negative;B. Prokaryotes;
B. Obligate intracellular parasites;G. Have a twisted form.

16. Source infections at loose typhus:
BUT. Sick; B. Carrier; B. Animals;G. Lice.

17. Immunity at loose typhus:
A. Antibacterial;B. Antitoxic; AT. non-sterile;
G. Local.

18. Methods cultivation rickettsia:
A. On blood agar;B. AT anaerostat;
AT. AT chicken embryo;
G. On the serum environments.

19. Make up brain teaser couples:
BUT. bioassay on the suckling rabbits
B. Aniline dyes are well perceivedAT. Microscoping in dark field vision

1. pathogens disease Lyme
2. Pathogen leptospirosis
3. Both
4. Neither then, neither other.

20. Immunological drugs for creation active immunity:

BUT. DTP	1. Tetanus
B. Tetanus toxoid serum	2. Gas gangreneAT.
ADS-m	3. Both
G. Antigangrenous serum	4. None then, not other.

21. Make up brain teaser couples: question answer
BUT. For bacterioscopic diagnostics use microscopy in dark field.
B. For bacterioscopic diagnostics carry out microscopy smears, painted on

Gram.
AT. For diagnostics put skin-allergic sample.G. Available
contact-household way transmission.
D. Anthroponosis.

1. Syphilis
2. Gonorrhea
3. Both
4. Neither then, neither other

«PATHOGENS OF ANAEROBIC CLOSTRIDIAL INFECTIONS.PATHOGENIC SPIROCHETES AND SPIROCHETOSES. MYCOPLASMS. CHLAMYDIA"

II OPTION

(Choose one or several correct answers)

1. To pathogens anaerobic infection applies to:
A. Clostridia; B.
Mycoplasmas;
B. Mycobacteria;
G. Chlamydia.

2. Disease botulism conditioned hit in organism person:
BUT. Brucella bovis;
B. Exotoxins Clostridium tetani;
AT. Clostridium botulinum and their exotoxins;
G. Clostridium spore difficile.

3. Clostridia form:
A. Hemozolin;
B. catalase;
B. Lecithinase;
G. DNA azu.

4. Clostridial anaerobes cultivate in environment:
A. Wilson-Blair; B.
Salt environment;
AT. biliary bouillon;
G. High column sugar agar.

5. For diagnostics gas gangrene apply:
BUT. Bacteriological method research;B.
Biological;
AT. Microscopic;
G. Serological method.

6. Tetanus - this is infection:
A. Anaerobic;
B. Contact; AT.
Intestinal;

G. Wound.

7. Clostridia - this is:
BUT. Gram+
aerobes;
B. Gram-
aerobes;
AT. Gram+
anaerobes;
G. Gram- anaerobes.

8. name peculiarities spirochete:
BUT. Gram negative bacteria;
B. Have motor fibrillar apparatus;AT. Have a
twisted form.
G. Are absolute parasites.

9. Peculiarities Borrelia:
BUT. Collection bacteria With 3-8 curls
B. Thin tortuous cells With bent ends
B. Stained according to Romanovsky-Giemsa in purpleG. Weak
perceive aniline dyes.

10. Morphology pathogen syphilis:
A. A thin bacterium of a spiral shape;B. Thick
stick;
B. Bean-shaped cocci;G.
Vibrio.

11. Cultural properties pathogen syphilis:
BUT. Can cultivate in testicle a rabbit;
B. May be cultured on media containing organ pieces;AT. Cultivated in
anaerobic conditions;
G. Cultivate can in aerobic conditions.

12. Methods bacterioscopic diagnostics syphilis:
BUT. Coloring silvering;
B. Methylene blue stain;B. Dark
field microscopy; G. Coloring by
Gram.

13. name obligate intracellular parasites:
BUT. Rickettsia;
B. Actinomycetes;
AT. Spirochetes;
G. Chlamydia.

14. Serological reactions, used at diagnostics loose typhus:
BUT.
Agglutination;
B. RSK;
AT. RPGA;
G. precipitation.

15. Mycoplasmas cause:
BUT. Atypical pneumonia;

B. Lesions of the genitourinary tract;
AT. Typhus;
G. returnable typhoid.

16. Main method identifying chlamydia is:
BUT. Coloring on Romanovsky-
Giemsa;B. Coloring on Neisser;
B. Coloring according to
Zdradovsky;G. Coloring on
Burri.

17. name main factors pathogenicity rickettsia:
A. Microcapsule; B.
Phospholipase A2;
AT. Adhesins *(OmpA, OmpB);*
G. Exotoxin.

18. Check pathogens defiant disease respiratory tract, atwhich source infection is a person:
A. C. trachomatis;
B. M.
pneumoniae;AT.
C. psittaci;
G. C. pneumoniae.

19. Make up brain teaser couples: question answer
A. Transmitted by airborne dropletsB.
Sexually transmitted through
AT. Have lipoid antigen, identical lipoid extract bullish heartsG. At hit in phagocytes cause unfinished phagocytosis

1. T. pallidum
2.N.gonorrhoeae
3. Both
4. Neither then, neither other

20. Make logical pairs: question-answer Morphological and tinctorial properties of pathogens:BUT. Gram positive sticks 1.
Pathogen tetanus
B. Terminal spores 2. Causative agents of gas
gangreneAT. Subterminal disputes 3. Both
G. located chain 4. Neither then, neither other

21. Make up brain teaser couples: question answer
BUT. Epidemic returnable typhoid 1. b. burgdorferi
B. syphilis 2. L. interrogans
AT. Disease Lyme 3. B. recurrentis
G. Leptospirosis 4. T. pallidum

«PATHOGENS OF ANAEROBIC CLOSTRIDIAL INFECTIONS.PATHOGENIC SPIROCHETES AND SPIROCHETOSES. MYCOPLASMS. CHLAMYDIA"

III OPTION

(Choose one or several correct answers)

1. pathogens tetanus are:
A. Fusobacteria;B.
Clostridia; AT.
Bacteroids; G.
Peptococci.

2. For clostridia characteristic:
A. Capsule formation;B.
Education dispute;
C. Presence of volutin grains;
G. Anaerobic type of
breathing.

3. Methods microbiological diagnostics botulism:
A. Bacterioscopic;B.
Bacteriological; AT.
Biological;
G. Serological.

4. Clostridium perfringens is pathogen:
A. Food poisoning; B.
Pseudomembranous colitis;AT.
gas gangrene;
G. Toxinemic infections.

5. Non-spore-forming anaerobes:
BUT. Bacteroids;
B. Clostridia;
AT. Fusobacteria;
G. Waylonelles.

6. Immunobiological drugs for prevention and treatment botulism:
A. Antitoxic serum;B. DPT;
B. Tetraanatoxin;G.
ADS.

7. The onset of tetanus is caused by the ingestion ofbody:
A. Brucella melitensis; B. Exotoxins
Clostridium difficile;
AT. Clostridium tetani and her exotoxin;

G. Clostridium novyi through wound.

8. By type breathing clostridia:
BUT. obligate anaerobes;
B. Optional anaerobes;AT.
obligate aerobes;
G. Microaerophiles.
9. To pathogens gas gangrene relate:
A. Clostridium perfringens;
B. Clostridium tetani;
B. Clostridium botulinum;
G. Clostridium novyi.
10. Ways transmission botulism:
A. Parenteral;B.
Wound;
B. Contact household;G.
Food.
11. For syphilis characteristic:
A. Penetration of the pathogen through skin and mucous
membranes;B. infection transplacental by;
AT. leaking cyclically;
G. leaking in form sepsis.

12. Antigens used for productions RSK at diagnostics syphilis:
BUT. O antigen;
B. Cardiolipin;
AT. Soluble antigen;
G. Treponemal specific.

13. What properties possess spirochetes?
BUT. Have thin cellular wall;B. Gram-
negative;
B. Thin spirally curved cells; G. Have
cytoplasmic cylinder.

14. What kind properties characteristic for chlamydia?
A. Gram-negative;B.
Prokaryotes;
AT. obligate intracellular parasites;
G. Have twisted form.
15. Describe pathogen leptospirosis:
A. Thin light threads with curved ends;B. Are
stained in violet color;
B. Number of curls 20-40;
G. form cysts.
16. Source infections at loose typhus:
BUT. Sick; B.
Carrier; B.
Animals;G.
Lice.

17. Immunity at loose typhus:
A. Antibacterial;B.
Antitoxic; AT. non-
sterile;
G. Local.

18. Methods cultivation rickettsia:
A. On blood agar;B.
AT anaerostat;
AT. AT chicken embryo;
G. On the serum environments.

19. Make up brain teaser couples: question answer
BUT. For bacterioscopic diagnostics use microscopy in dark field.
B. For bacterioscopic diagnostics carry out microscopy smears, painted onGram.
B. For diagnosis, a skin-allergic test is performed.G.
Available contact-household way transmission.
D. Anthroponosis.

1. Syphilis
2. Gonorrhea
3. Both
4. Neither then, neither other

20. Immunobiological drugs for creation active immunity:

BUT. DTP	1. Tetanus
B. Tetanus toxoid serum	2. Gas gangreneAT.
ADS-M	3. Both
G. Antigangrenous serum.	four. Neither then, neither other.

21. Install conformity called infections and kind pathogen:

BUT. Tetanus	1. Cl. botulinum
B. Gas gangrene	2. Cl. tetani
AT. Botulism	3. F. nucleatum
G. Fusospirochetosis	4. Cl. novyi

«PATHOGENS OF ANAEROBIC CLOSTRIDIAL INFECTIONS.PATHOGENIC SPIROCHETES AND SPIROCHETOSES. MYCOPLASMS. CHLAMYDIA"

IV OPTION

(Choose one or several correct answers)

1. Tetanus - this is infection:
BUT. anaerobic;
B. Contact;
AT. Intestinal;
G. Wound.

2. To pathogens anaerobic infections applies to:
A. Clostridia; B.
Mycoplasmas;

B. Mycobacteria;
G. Chlamydia.

3. Clostridia - this is:
BUT. Gram+
aerobes;
B. Gram-
aerobes;
AT. Gram+
anaerobes;
G. Gram- anaerobes.

4. For diagnostics gas gangrene apply:
BUT. Bacteriological method research;B.
Biological;
AT. Microscopic;
G. Serological method.

5. Disease botulism conditioned hit in organism person:
BUT. Brucella bovis;
B. Exotoxins Clostridium tetani;
AT. Clostridium botulinum and their exotoxins;
G. Clostridium spore difficile.

6. Clostridia form:
A. Hemozolin;
B. catalase;
B. Lecithinase;
G. DNA azu.

7. Clostridial anaerobes cultivate in environment:
A. Wilson-Blair; B.
Salt environment;
AT. biliary bouillon;
G. High column sugar agar.

8. name peculiarities spirochete:
BUT. Gram negative bacteria;
B. Have motor fibrillar apparatus;AT. Have a
twisted form.
G. Are absolute parasites.

9. Morphology pathogen syphilis:
A. A thin bacterium of a spiral shape;B. Thick
stick;
B. Bean-shaped cocci;G.
Vibrio.

10. Peculiarities Borrelia:
BUT. Collection bacteria With 3-8 curls
B. Thin tortuous cells With bent ends
B. Stained according to Romanovsky-Giemsa in purpleG. Weak
perceive aniline dyes.

11. Methods bacterioscopic diagnostics syphilis:

BUT. Coloring silvering;
B. Methylene blue stain;B. Dark field microscopy; G. Coloring by Gram.

12. Cultural properties pathogen syphilis:
BUT. Can cultivate in testicle a rabbit;
B. May be cultured on media containing organ pieces;AT. Cultivated in anaerobic conditions;
G. Cultivate can in aerobic conditions.

13. name obligate intracellular parasites:
BUT. Rickettsia;
B. Actinomycetes;
AT. Spirochetes;
G. Chlamydia.

14. Serological reactions, used at diagnostics loose typhus:
BUT. Agglutination;
B. RSK;
AT. RPGA;
G. precipitation.

15. Mycoplasmas cause:
BUT. atypical pneumonia;
B. Lesions of the genitourinary tract;
AT. Typhus;
G. returnable typhoid.

16. name main factors pathogenicity rickettsia:
A. Microcapsule; B. Phospholipase A2;
AT. Adhesins *(OmpA, OmpB);*
G. Exotoxin.

17. Check pathogens defiant disease respiratory tract, atwhich source infection is a person:
A. C. trachomatis;
B. M. pneumoniae;AT. C. psittaci;
G. C. pneumoniae.

18. Main method identifying chlamydia is:
BUT. Coloring on Romanovsky-Giemsa;B. Coloring on Neisser;
B. Coloring according to Zdradovsky;G. Coloring on Burri.

19. Make up brain teaser couples: question answer

BUT. Epidemic returnable typhoid	1. b. burgdorferi
B. syphilis	2. L. interrogans
AT. Disease Lyme	3. b. recurrentis

G. Leptospirosis 4. T. pallidum

20. Make up brain teaser couples: question answer

BUT. Gram negative	1. Bacteroids
B. Kokki	2. Waylonelles
AT. sticks	3. Both
G. form subterminal controversy	4. Neither then, neither other.

21. Make up brain teaser couples: question answer

BUT. Gr+Cocci	1. Peptostreptococci
B. Gr+ sticks	2. Clostridia
AT. Aerobes	3. Both
G. Anaerobes	4. Neither then, neither other

«PRIVATE VIROLOGY. PATHOGENIC FUNGI»I OPTION
(Choose one or several correct answers)

1. **AT pathogenesis viral diseases decisive role plays:**
   a) the virulence of the virus;b) toxigenicity of the virus;in) level lysozyme;
   G) reaction organism on the cells, affected virus.

2. **Install serological type of virus influenza can With help:**
   a) reactions agglutination on the glass;
   b) reactions braking hemagglutination;in) reactions indirect hemagglutination;
   G) reactions hemagglutination.

3. **AT pathogenesis AIDS important place occupies:**
   a) transformation of PrP^{c} proteins into PrP^{sc} proteins; b) unrestrained proliferation B-lymphocytes;
   in) accumulation pathological myeloma proteins;G) defeat T-helpers and macrophages.

4. **Interferon provides antiviral protection cells, because prevents:**
   a) adsorption virus on the cage;
   b) penetration virus in cell;in) reproductions virus;
   G) lysis affected cells;

5. **HIV applies to group viruses:**
   a) DNA-genomic;

b) RNA genomic;in)
complex;
G) simple.

6. For serodiagnosis viral hepatitis apply:
a) hemagglutination inhibition reaction;b)
enzyme immunoassay analysis;
c) reaction of indirect (passive) hemagglutination;G)
hemagglutination reaction;

7. Neurotropic viruses are considered:
a) virus flu;
b) hepatitis C virus;
c) rabies virus; G)
virus rubella.

8. Virus Epstein-Barr calls:
a) sarcoma Kaposi;
b) Infectious mononucleosis;in)
Shingles;
G) Cytomegaly.

9. For planned specific prevention polyemylite use:
a) Sabin live vaccine;b)
toxoid;
in) killed vaccine;
G) specific serum;

10. Virus rubella calls:
a) Panencephalitis;
b) acute respiratory infection;in)
congenital pathology;
G) acute intestinal infection.

11. Virus avian influenza applies to:
a) to the influenza virus type C; b) to the influenza virus type A; c) to the influenza virus type B;G) to virus influenza type D.

12. Viruses poliomyelitis refer to family:
a) caliciviruses;b)
retroviruses; in)
poxviruses;
G) picornaviruses.

13. Basic path transmission virus hepatitis A BUT:
a) parenteral;
b) airborne;c) fecal-oral;
G) contact.

14. Which type of nucleic acids contains virus hepatitis A AT?

a) RNA;
b) DNA;
in) DNA and RNA.

15. What represents yourself mycelium mushrooms?

a) this is cell, devoid of cytoplasmic membranes;b) it is a collection of hyphae;
c) it is a collection of chlamydospores;G) this is multi-core structure.

16. Yeast-like mushrooms are characterized by:

a) presence round or oval cells;
b) ability multiply sexual by;
c) the ability to reproduce only asexually;G) ability to form disputes.

17. Mushrooms kind Candida

a) belong to yeast-like fungi;b) refer to filamentous mushrooms;
c) belong to filamentous fungi;G) are pathogenic.

18. At keratomycosis are affected:

a) the stratum corneum of the epidermis;b) bones;
in) hair;
G) domestic organs.

Make up brain teaser couples: question answer

19.

1. Conditionally pathogenic mushrooms:	a. Trichophyton
2. Dermatophytes	b. Genus Aspergillus
3. form conidia:	c. Both
4. form aflatoxins:	d. Neither then, neither other

20. Specify conformity between through transmission virus and disease

one. fecal-oral	a. Hepatitis AT
2. Parenteral	b. Polio
3. airborne	in. Hepatitis BUT
	G. Rubella

21.

1. To rhabdoviruses relate:

2. To orthomyxoviruses relate:A.
mumps virus. B. Virus rabies
B. Tick-borne encephalitis virus;
G. Influenza viruses.

«PRIVATE VIROLOGY. PATHOGENIC FUNGI»II OPTION

(Choose one or several correct answers)

1. Determine the antibodies in the patient's blood to a specific serotype virus flu can be help:
a) reactions agglutination on the glass;
b) reactions hemagglutination;
in) enzyme immunoassay analysis.

2. Reaction braking hemagglutination maybe to be applied for:
a) detection virus influenza in researched material;b) influenza virus identification;
in) definition quantity virus in researched material;G) antibody detection to virus in blood.

3. Specify virus hepatitis A, demanding for replication participation helper virus:
a) VGA;
b) VGB;
c) VGC;
G) VGD.

4. Interferon has next action:
a) lysing in respect affected cells;b) stimulating phagocytosis;
in) inhibitory broadcast;
G) specific binding With virus.

5. Virus influenza applies to group viruses:
a) DNA-genomic;b) RNA-genomic; in) complex;
G) families orthomyxoviruses.

6. characteristic signs families retroviruses are:
a) H and N antigens capsid;
b) enzyme reverse transcriptase;in) fragmentation genome;
G) two identical threads RNA in genome.

7. Enterotropic are considered:
a) virus poliomyelitis;b) virus hepatitis C;

in) virus rabies;
G) viruses coxsackie and Echo.

8. Viruses flu - this is:
a) DNA-containing virusesb)
simple viruses
c) RNA-containing virusesG)
complex viruses.

9. For specific prevention rabies use:
a) a live vaccine;b)
toxoid;
in) inactivated vaccine;G)
gamma globulin.

10. Antigenic drift and shift are related to the following virus antigensflu:
a) ribonucleoprotein NP;b)
matrix squirrel M;
c) neuraminidase N;
G) hemagglutinin N.

11. Which type of ~~—well-~~klein acids contains virus wind smallpox?
a) RNA;
b) DNA;
in) DNA and RNA;
G) not contains nucleic acid.

12. Polio viruses are:a) DNA-
containing viruses; b) simple
viruses;
c) RNA-containing viruses;G)
complex viruses.

13. Which type of nucleic acids contain viruses hepatitis BUT and E?
a) DNA;
b) RNA;
in) DNA and RNA;
G) not contains nucleic acid.

14. To systemic, or deep mycoses applies to:
a)
Histoplasmosis;b)
Favus (scab);c)
Sporotrichosis; G)
Microsporia.

15. What such conidia?
a) Endospores;
b) Exospores;
in) spore-forming structures; G)
transverse partition in hyphae.

16. Opportunistic mycoses:

a) Cause pathogenic mushrooms;
b) Cause conditionally pathogenic mushrooms;
c) Cause unclassified pathogenic fungi;G) They are called dermatophytes.

17. Mycoses - this is diseases, caused by:

a) bacteriab) mushrooms;
c) the simplest;G) Chlamydia.

18. For allocation mushrooms from pathological material use:

a) MPA;
b) Wednesday Saburo;
c) serum agar;G) MPB.

Make up brain teaser couples: question answer

19.

1. Keratomycosis:	A. microsporum
2. subcutaneous mycoses	b. Pathogen colorful lichen
3. deep mycoses	B. Sporotrichosis
4. Epidermophytosis:	G. Blastomycosis.

20. Install conformity between through infections and view virus hepatitis A

one. fecal-oral	BUT. VGA
2. Parenteral	B. VG B
3. Sexual	AT. VGE

21. Which type of nucleic acids contain viruses?

1. Herpesviruses
2. Virus parainfluenza
3. Polio virusBUT. DNA
B. RNA
AT. DNA and RNA
G. neither then, neither other.

«PRIVATE VIROLOGY. PATHOGENIC FUNGI»III OPTION

(Choose one or several correct answers)

1. specific factors protection organism from viruses are:

a) NK - cells (normal killers);

b) interferons;in)
slgA;
G) CD8 - cells (T-killers).

2. HIV is cultivated in: a)
chicken embryo; b)
culture cells PE4;in)
lungs whites mice;
G) culture CD4 lymphocytes.

3. Synthesis interferons encoded:
a) genome virus;b)
HLA genes;
in) prophage;
G) provirus.

4. Virus influenza cultivated in:
a) culture CD4 lymphocytes;b)
lungs whites mice;
in) chicken embryo;
G) culture cells PE4.

5. Nonspecific resistance to viruses influenza depends from availability:
a) lysozyme;
b) complement; c)
inhibitors; G)
interferons.

6. To viruses hepatitis A, having complicated structure include:
a) VGA;
b) VGB;
in) VGC;
G) VGE.

7. Slow viral disease are characterized by:
a) incubation period continues months and years;
b) recurrent damage to the central nervous system and immune
system;in) progressive flow from lethal outcome;
G) sharp flow With defeat vital important organs.

8. The AIDS clinic is defined by a number of complications caused by opportunistic agents:
a) herpes viruses;
b) pathogen diphtheria;in)
mushrooms Candida;
G) mycobacteria tuberculosis.

9. For specific prevention tick-borne encephalitis use:
a) a live vaccine;b)
toxoid;
in) killed vaccine;G)
antigrippin.

10. Viruses influenza refer to family:

a) coronaviruses;b)
adenoviruses;
c) paramyxoviruses;G)
orthomyxoviruses.

11. For virus natural smallpox characteristic:

a) RNA-containing simple virus; b)
DNA containing difficult virus;in)
contains hemagglutinin;
G) not contains hemagglutinin.

12. What class of immunoglobulins in the blood serum of a patient with hepatitis Aindicates activity (sharpness) of the process?

a) lgG;
b) IgA;
c) Ig M;
G) Ig E.

13. How serotypes have viruses polio?

a) 5
6) 7
in) 3
G) 2

14. Virus immunodeficiency human characterized next properties:

a) DNA-containing; b)
RNA-containing; in)
simple virus;
G) difficult virus.

15. What represents yourself mycelium mushrooms?

a) this is cell, devoid of cytoplasmic membranes;b) this is set
of hyphae;
c) it is a collection of
chlamydospores;G) this is multi-
core structure.

16. Yeast-like mushrooms not are characterized by:

a) presence round or oval cells;
b) ability multiply sexual by;
in) ability multiply only asexual by;G) ability to form
disputes.

17. Mushrooms kind Candida

a) belong to yeast-like fungi;b) refer to
filamentous mushrooms;
c) belong to filamentous fungi;G) are
pathogenic.

18. By relation to temperature pathogenic mushrooms are:

a) psychrophiles;

b) mesophylls; in)
thermophiles;
G) all answers correct.

Make up brain teaser couples: question answer

19.

1. Conditionally pathogenic mushrooms a	. Trichophyton
2. Dermatophytes	b. Genus Aspergillus
3. form conidia:	B. Both
4. form aflatoxins:	G. Neither then, neither other.

twenty.

Specify conformity between through transmission virus and disease

one. fecal-oral	BUT. Hepatitis AT
2. Parenteral	B. Polio
3. airborne	AT. Hepatitis BUT
	G. Rubella

21.

At what viruses discovered the following antigens?

1. HBs -antigen

2. Hemagglutinin

BUT. Virus measles
B. Virus hepatitis A AT
AT. Virus poliomyelitis.

«PRIVATE VIROLOGY. PATHOGENIC FUNGI»IV OPTION
(Choose one or several correct answers)

1. HIV applies to group viruses:
a) DNA-genomic;b)
RNA-genomic; in)
complex;
G) families orthomyxoviruses;

2. Viruses parainfluenza - this is:
a) DNA-containing viruses;b)
simple viruses;
c) RNA-containing viruses;G)
complex viruses.

3. Interferon provides antiviral protection cells, because prevents:
a) reproductions virus;

b) lysis of the affected cell;in)
activation killers;
G) adsorption virus on the cage

4. Nonspecific factors protecting the body from influenzaare:
a) the complement
system;b) inhibitors;
c) interferons;G)
slgA;

5. Broadcast HIV infections going on next ways:
a) parenteral;b)
alimentary; in)
sexual;
G) airborne.

6. For serodiagnosis viral hepatitis apply:
a) hemagglutination inhibition reaction;b)
enzyme immunoassay analysis;
c) reaction of indirect (passive) hemagglutination;G)
hemagglutination reaction.

7. causative agents slow infections may to be:
a) prions;
b) tick-borne encephalitis virus;
in) virus poliomyelitis;
e) virus flu.

8. Enterotropic are considered:
a) polio virus;b) virus
rabies;
in) virus hepatitis A FROM;
G) viruses coxsackie and Echo.

9. For planned specific prevention influenza use:
a) a live vaccine;b)
toxoid;
in) inactivated whole virion vaccine;G) antigrippin.

10. Virus measles on structure:
a) a simple virus; b)
difficult virus;
in) It has supercapsid;
G) not It has supercapsid.

11. Viruses parainfluenza include:
a) to the genus
Paramyxovirus;b) to the
genus Lyssavirus;
in) to the genus Pneumovirus;

G) to the genus Morbillivirus.

12. For specific prevention poliomyelitis use:

a) BCG;
b) DPT;
in) live vaccine, received Smorodintsev A.A. and ChumakovM.P.;
G) anti-rabies vaccine.

13. Which path transmission hepatitis AT, FROM, D, G is main?

a) fecal-oral;b)
parenteral;
c) airborne;G) contact.

14. Which type of nucleic acids contain viruses hepatitis BUT and E?

a) DNA;
b) RNA;
in) DNA and RNA;
G) not contains nucleic acid.

15. ringworm lichen called mushrooms kind:

a) Trichophyton;
b) Aspergillus;
in) Candida
G) Fusarium.

16. To systemic, or deep mycoses applies to:

a) Histoplasmosis;
b) Favus (scab);c)
Sporotrichosis; G)
Microsporia.

17. What such conidia?

a) Endospores;
b) Exospores;
in) spore-forming structures; G)
transverse partition in hyphae.

18. Opportunistic mycoses:

a) cause pathogenic mushrooms;
b) cause opportunistic pathogens mushrooms;
c) cause unclassified pathogenic fungi;G) cause
dermatophytes.

Make up brain teaser couples: question answer

19.

1. Keratomycosis:	A. microsporum
2. subcutaneous mycoses	b. Pathogen colorful lichen
3. Deep mycoses	B. Sporotrichosis
4. Epidermophytosis:	G. Blastomycosis

20. Install conformity between type nucleic acids genome and view virus hepatitis A

1-DNA	A.VGA
2- RNA	B.VGB
	V.VGC
	G.VGD
	D.VGE

21. What kind reactions use at diagnostics

1. **Polio**
2. **Hepatitis A AT**

BUT. reaction neutralization color samplesB. reaction indirect hemagglutination AT. Both
G. Neither then, neither other