REPUBLIC OF UZBEKISTAN
MINISTRY OF HIGHER AND SECONDARY SPECIAL EDUCATION

SAMARKAND INSTITUTE OF VETERINARY MEDICINE

«DEPARTMENT OF ANIMAL PHYSIOLOGY, BIOCHEMISTRY AND PATHOLOGICAL PHYSIOLOGY

"I APPROVE"
Vice rector for academic affairs
Elmurodov AA
" " 2022

TRAINING AND METODOLOGY COMPLEX

by subject

"Phatophysiology of Animals"

Field of knowledge:	400000 - Agriculture and water management
Field of education:	440000 - Veterinary
Areas of study:	5440100-Veterinary medicine (by type of activity) 5440300-Veterinary diagnostics and laboratory work

Samarkand - 2022

The working curriculum (syllabus) of science has been developed in accordance with the approved curriculum and science program for 2022.

Developers:

Eshimov DE Head of the Department of "Animal Physiology, Biochemistry and Pathological Physiology", Associate Professor, Candidate of Biological Sciences.

Ruzikulov RF - Associate Professor of "Animal Physiology, Biochemistry and Pathological Physiology", Candidate of Veterinary Sciences.

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Reviewers:

Toshmuratov EA - Head of the Samarkand Regional Department of Veterinary and Livestock Development.

Eshburiyev B. - Sam IVM, Department of Veterinary Surgery and Obstetrics, c.v.s. Professor,

Working curriculum of science "Animal physiology, biochemistry and pathological physiology" Discussed at the meeting of the department "26" in 08. 2022 "1" and recommended for discussion at the faculty council.

Chair holder, c.b.s., Associate Professor _____ **Eshimov.DE**

The working curriculum of the subject was discussed and recommended for use by the Board of the Faculty of Veterinary Diagnostics and Food Safety (Protocol No. 1 of 27.08., 2022).

Faculty Council Chairman, c.v.s., Professor _____ **Davlatov.RB**

Agreed:
Educational-methodical head of department, c.v.s, Associate Professor _____ **Ruzikulov RF**

CONTENTS

Study program

2/28

O'ZBEKISTON RESPUBLIKASI
OLIY VA O'RTA MAXSUS TA'LIM VAZIRLIGI

SAMARQAND VETERINARIYA MEDITSINASI INSTITUTI

"TASDIQLAYMAN"
Samarqand veterinariya meditsinasi instituti rektori
_____
"05" 08 2021 yil

"KELISHILDI"
Oliy va o'rta maxsus ta'lim vazirligi
_____
2021 yil "__" _____

Ro'yxatga olindi: №BD-5440100-2.10
2022 yil "__" _____

HAYVONLAR PATOFIZIOLOGIYASI
FAN DASTURI

Bilim sohasi:	400000 – Qishloq va suv xo'jaligi
Ta'lim sohsi:	440000 –Veterinariya
Ta'lim yo'nalishlari:	5440100-Veterinariya meditsinasi (faoliyat turlari bo'yicha) 5440300-Veterinariya diagnostikasi va laboratoriya ishlari

Toshkent – 2022

Fan/modul kodi	O‘quv yili	Semestr	ECTS – Kreditlar
HPM 2306	2021 - 2022	4	6

Fan/modul turi	Ta’lim tili	Haftadagi dars soatlari
Majburiy	O‘zbek/rus	6

1.	Fanning nomi	Auditoriya mashg‘ulotlari (soat)	Mustaqil ta’lim (soat)	Jami yuklama (soat)
	Hayvonlar patofiziologiyasi	90	90	180

2. I. Fanning mazmuni

Ushbu fan Respublikamizdagi ijtimoiy-iqtisodiy islohatlar natijalarining chorvachilik va veterinariya istiqboliga, kasal hayvonlar va parrandalar organizmida ro‘y beradigan umumiy o‘zgarishlar, ularning hayvonlar mahsuldorligiga ta’siri, hayvonlar organizmidagi turli sistemalarning patofiziologiyasi kabi mavzularni qamrab oladi.

“Hayvonlar patofiziologiyasi” fani talabalarga hayvonlar organizmida kechadigan patologik jarayonlarni va ularni hayvonlar mahsuldorligiga ta’sirini tushintirish, hayvonlar organizmida me’yor va patologiyani farqlash, kasalliklar rivojlanishining ilmiy va amaliy qonuniyatlarini o‘rgatish borasida veterinariya tizimining ajralmas bo‘g‘ini bo‘lib hisoblanadi.

“Hayvonlar patofiziologiyasi” umumkasbiy fanlar blokiga kiritilgan fan hisoblanib, 2-bosqichda o‘qitiladi. Mazkur fan veterinariya fanlarining nazariy hamda amaliy asosini tashkil qilib, o‘z rivojida veterinariya ta’lim yo‘nalishidagi umumkasbiy hamda ixtisoslik fanlari uchun zamin bo‘lib xizmat qiladi.

Fanning vazifasi - talabalarga kasal organizmda ro‘y beradigan umumiy o‘zgarishlarni, kasalliklarning paydo bo‘lishi, sabablari, shart-sharoitlari, rivojlanish mexanizmi, avj olishi, kechishi va oqibatlarini, ularning hayvonlar mahsuldorligiga ta’sirini o‘rgatish hamda ularni amaliyotda tatbiq etish ko‘nikmasini hosil qilishdan iborat.

Ushbu maqsadga erishish uchun fan talabalarni nazariy bilimlar, amaliy ko‘nikmalar, hayvonlar organizmida kechayotgan patologik jarayonlarga, ularning asoslari va qonuniyatlarini aniqlashga uslubiy yondoshuv hamda ilmiy dunyo qarashini shakillantirish vazifalarini bajaradi.

II. Asosiy nazariy qism (ma’ruza mashg‘ulotlari)

II.I. Fan tarkibiga quyidagi mavzular kiradi:

1-mavzu. Hayvonlar patofiziologiyasi fani va uning rivojlanish tarixi

Fanning mazmuni, maqsadi, vazifasi va boshqa fanlar bilan o‘zaro bog‘liqligi. Hayvonlar patofiziologiyasi fanining rivojlanish tarixi.

2-mavzu. Nozologiya

Sog‘lomlik (me’yor) va kasallik haqida tushuncha. Patologik reaksiya, patologik jarayon va patologik holatlar haqida tushuncha. Kasallik ta’rifi, turlari, kechishi, organizmdagi hayotiy jarayonlarni qayta tiklash - reanimatsiya. Anabioz: mavsumiy va latergik uyqu.

3-mavzu. Etiopatogenez

Etiologiya va patogenez haqida tushuncha. Etiologiyaga doir metafizik qarashlarni tanqid qilish: monokauzalizm, konditsionalizm, konstitusionalizm oqimlari. Kasalliklarni keltirib chiqaruvchi sabablar, shart-sharoitlari va oldini olish chora-tadbirlari.

Patogenez va unda etiologik omillarni ta’sir qilish mexanizimi. Kasallikning asosiy rivojlanish mexanizmlari. Organizmda kasallik qo‘zg‘atuvchi agentlarning tarqalish yo‘llari. Patogenezda neyro-gumoral jarayonlarning ahamiyati. Hayvonlar turi, zoti, jinsi va yoshining patogenezdagi ahamiyati. Sanogenez - sog‘ayish. Organizmning sog‘ayish jarayonini ta’minlab beradigan himoya-kompensator mexanizmlari. Sanogenezda nerv va endokrin sistemalarining ahamiyati.

4-mavzu. Organizm reaktivligi va rezistentligi

Organizm reaktivligi va rezistentligi haqida tushuncha. Reaktivlik va rezistentlik turlari. Organizm reaktivligining namoyon bo‘lish shakllari va unga ta’sir etuvchi omillar. Reaktivlikda nerv va endokrin sistemalarning ahamiyati.

5-mavzu. Allergiya va allergik kasalliklar

Allergiya, sabablari, turlari va rivojlanish mexanizmi. Allergenlar va ularning turlari. Allergik kasalliklar va ularning turlari. Yuqumli kasalliklarni aniqlashda allergik reaksiyalarning ahamiyati.

6-mavzu. Yallig‘lanish

Yallig‘lanish haqida tushuncha. Yallig‘lanishning etiologiyasi va tashqi belgilari. Yallig‘lanishning asosiy bosqichlari: alteratsiya, ekssudatsiya va proliferatsiya. Yallig‘lanishda qon aylanishining buzilishi.

Yallig‘lanish tasnifi. Ekssudat, uning turlari va xususiyatlari. Yallig‘lanishda nerv, endokrin va immun tizimlarining o‘rni. Yallig‘lanishning oqibatlari va organizm uchun ahamiyati.

7-mavzu. O‘smalar

O‘smalar haqida tushuncha. O‘smalarning turlari, etiologiyasi, patogenezi. Xavfli va xavfsiz o‘smalarning asosiy xususiyatlari. O‘smalarning hayvonot dunyosida tarqalishi va biologik xususiyatlari. Anaplaziya: morfologik, bioximiyaviy, fiziko-kimyoviy, energetik. O‘smalarda moddalar almashinuvi. O‘sma bilan organizmning o‘zaro aloqadorligi. O‘smalar etiologiyasi to‘g‘risidagi nazariyalar.

8-mavzu. Issiqlik almashinuvining buzilishi

Organizmda issiqlik almashinuvining boshqarilishini buzulishi va sabablari. Gipotermiya. Gipertermiya. Isitmaning etiologiyasi, patogenezi, bosqichlari va turlari. Isitmada moddalar almashinuvi va organ-sistemalar faoliyatidagi o'zgarishlar. Isitmaning organizm uchun biologik ahamiyati.

9-mavzu. Qon patofiziologiyasi

Umumiy qon miqdorining o'zgarishi. Normovolemiya, Gipervolemiya (pletora), gipovolemiya (oligemiya), ularning turlari va hosil bo'lish mexanizmi. Qon quyish usullari. Gemotransfuzion shok.

10-mavzu. Qon aylanishning patofiziologiyasi

Qon aylanishini va uni buzuvchi umumiy sabablar. Qon aylanishi yetishmovchiligi va uning asosiy klinik belgilari.

Qon tomirlar faoliyati buzilganda umumiy qon aylanishidagi o'zgarishlar. Arteriosklerozning qon aylanishiga ta'siri.

Yurak faoliyatidagi yetishmovchiliklar, etiologiyasi, patogenezi va oqibati. Yurak nuqsonlari va ularning turlari. Perikard va miokard patologiyasi davrida qon aylanishining buzilishi. Yurak ish ritmining buzilishlari, aritmiyalar.

11-mavzu. Nafas patofiziologiyasi

Tashqi nafas faoliyatining buzilishi. Nafas markazi faoliyatining o'zgarishi, sabablari va patogenezi.

Davriy nafas olish: Cheyn-Stokscha, Biotcha va Kussmaulcha. Hansirash turlari, sabablari va patogenezi. Yo'tal, aksa urish. Asfiksiya.

O'pka kasalliklarida (bronxit, pnevmoniya, bronxopnevmoniya, atelektaz, emfizema), qon va atmosfera havosi tarkibini o'zgarishidan nafas faoliyatining buzilishi.

12-mavzu. Hazm tizimining patofiziologiyasi

Hazm jarayonining buzilishi va uning sabablari. Ishtaha va chanqashning buzilishi. Og'iz bo'shlig'ida oziqa hazmining buzilishi. Chaynashning buzilishi, sabablari va oqibatlari. So'lak ajralishining buzilishi.

Me'dada oziqa hazmining buzilishi. Patologik sekresiyaning to'rt tipi. Gipersekresiya. Giposekresiya. Axiliya.

Ichaklarda oziqa hazmining buzilishi. Me'da osti bezi tashqi sekretsiyasining patologiyasi. Ichaklarda shira ajralishi va so'rilish jarayonlarining buzilishi. Ichaklar motorikasining buzilishi. Ich yurishmay qolishi – qabziyat. Dispepsiya turlari, sabablari, patogenezi, belgilari va oqibatlari.

13-mavzu. Jigar patofiziologiyasi

Jigar faoliyatining buzilishi, sabablari, patogenezi, belgilari va oqibatlari. Jigar faoliyati buzilganda moddalar almashinuvining buzilishi.

O't hosil bo'lishi va ajralishining buzilishi. Sariqlik turlari, sabablari, patogenezi, belgilari va oqibatlari. Bilirubinemiya. Xolemiya. Urobilinuriya. O't toshi kasalligi.

14-mavzu. Buyrak patofiziologiyasi

Siydik hosil bo'lishi va ajralishining buzilishi. Buyrak faoliyati buzilishining etiopatogenezi. Buyrak faoliyati buzilishiga olib keluvchi ekstrarenal va renal omillar.

Siydik miqdori va tarkibining o'zgarishi. Buyrak faoliyati buzilishining umumiy oqibatlari: siydik toshi kasalligi, buyrak shishlari va gipertoniyasi. Uremiya, turlari, sabablari, patogenezi, belgilari va oqibati.

15-mavzu. Nerv tizimining patofiziologiyasi

Nerv tizimi faoliyati buzilishining etiologiyasi.

Nerv tizimining harakatlantiruvchi faoliyatini buzilishi. Parez va falajlanish. Gipokinez. Giperkinez. Akinez. Ataksiya. Asteniya. Astaziya.

Sezuvchanlikning buzilishi. Gipesteziya, giperstesiya, anesteziya, paresteziya. Ichki organlar sezuvchanligining buzilishi.

Vegetativ nerv sistemasi faoliyatining buzilishi. Nerv sistemasi trofik faoliyatining buzilishi.

III. Amaliy mashg'ulotlari bo'yicha ko'rsatma va tavsiyalar

III.I. Amaliy mashg'ulotlar uchun quyidagi mavzular tavsiya etiladi:

1. Eksperiment va uning patologiyadagi ahamiyati.
2. Kasallik davrlari va oqibatlari.
3. Organizmga elektr tokining patologik ta'siri
4. Hayvonlar organizmiga yuqori va past atmosfera bosimining patologik ta'siri.
5. Hayvonlar organizmiga yuqori va past haroratning patologik ta'siri.
6. Allergiyaning mahalliy alomatlari.
7. Arteriya va vena giperemiyasi.
8. Staz. Ishemiya.
9. Qon oqishi – gemmoragiya.
10. Tromboz.
11. Emboliya.
12. Yallig'lanishning tashqi mahalliy belgilari va qon tomirlar reaksiyasi.
13. Shish va istisqo.
14. Gipertoniya va gipotoniya.
15. Nafas faoliyatidagi o'zgarishlar.

Amaliy mashg'ulotlarni tashkil etish bo'yicha kafedra professor-o'qituvchilari tomonidan uslubiy ko'rsatma va tavsiyalar ishlab chiqiladi. Unda talabalar asosiy ma'ruza mavzulari bo'yicha olgan bilim va ko'nikmalarini amaliy mashg'ulotlarda tajribalarni bajarish orqali yanada boyitadilar.

Amaliy mashg'ulotlar zarur asbob uskunalar bilan jihozlangan

	auditoriyada bir guruhga bir o‘qituvchi tomonidan o‘tkazilishi lozim. Mashg‘ulotlar faol va interfaol usullar yordamida o‘tilishi, mos ravishda munosib pedagogik va axborot texnologiyalarni qo‘llanilishi maqsadga muvofiq. **III.II. Laboratoriya mashg‘ulotlar uchun quyidagi mavzular tavsiya etiladi:** 1. Hayvonlar organizmiga kimiyoviy moddalarning patologik ta’siri . 2. Hayvonlar organizmining bar’erlik xususiyatlari. 3. Fagotsitoz va pinotsitoz jarayonlari. 4. Anafilaktik shokni namoish etish. 5. Yiringli ekssudatning morfologik tarkibi va xususiyatlari. 6. Medikamentoz va tuz isitmalari. 7. Gipoglikemik shokni namoish etish. 8. Eritrotsitlarning son va sifat jihatidan o‘zgarishlari. 9. Eksperimental gemolitik anemiya. 10. Leykotsitoz va leykopeniya. 11. Leykoformula va leykoprofilni aniqlash. 12. Me’dada shira ajralishining buzilishi. 13. O‘t suyuqligining organlar faoliyatiga patologik ta’siri. 14. Diurezga gormonlarning ta’siri. 15. Nerv tizimining reflektor faoliyatini buzilishi. Laboratoriya mashg‘ulotlarni tashkil etish bo‘yicha kafedra professor-o‘qituvchilari tomonidan laboratoriya mashg‘ulotlarining pasportlari, ularni bajarish bo‘yicha uslubiy ko‘rsatma va tavsiyalar ishlab chiqiladi. Laboratoriya mashg‘ulotlari zarur asbob uskunalar va reaktivlar bilan jihozlangan auditoriyada bir guruhni ikkiga bo‘lib o‘tkazilishi lozim. Laboratoriya mashg‘ulotlarida talabalar turli qishloq xo‘jalik va laboratoriya hayvonlari organizmida kasalliklarni chaqiruvchi sabablar ta’sirida ro‘y beradigan o‘zgarishlarni laboratoriya (gemotologik, serologik va boshqa) tekshirishlar orqali o‘rganadilar. Olingan natijalarni tahlil qilish va baholash malakalariga ega bo‘ladilar. **IV. Mustaqil ta’lim va mustaqil ishlar.** Mustaqil ta’lim uchun tavsiya etiladigan mavzular: 1. Irsiyat, irsiy va tug‘ma kasalliklar. 2. Hayvon yoshi va konstitutsiyasining patogenezdagi ahamiyati. 3. Immun tizim patofiziologiyasi. 4. Atrofiya va uning turlari. 5. Gipertrofiya va uning turlari. 6. Regeneratsiya, transplantatsiya va ularning turlari. 7. Moddalar almashinuvining buzilishi. 8. Uglevodlar almashinuvining buzilishi. 9. Yog‘ almashinuvining buzilishi. 10.Oqsil almashinuvining buzilishi.
	11.Suv almashinuvining buzilishi va oqibatlari. 12.Och qolish va uning turlari. 13.Mineral moddalar yetishmovchiligi. 14.Vitaminlar yetishmovchiligi. 15.Yurak muskullarining xususiyatlarining buzilishlari. 16.Kavsh qaytaruvchi hayvonlarning me’da oldi bo‘limlarida oziqa hazmining buzulishi. 17.Ichki sekretsiya bezlari patologiyasi. 18.Stress va adaptatsion sindrom. 19.Oliy nerv faoliyatining buzulishi. 20.O‘rta miyya va miyyacha faoliyatining buzulishi Mustaqil ta’limning turli xil shakllari mavjud bo‘lib, bunda asosiy e’tibor talabaning berilgan mavzular (amaliy masalalar, topshiriqlar va keys-stadilar)ni mustaqil ravishda, ya’ni auditoriyadan tashqarida bajarishi, o‘qib o‘rganishi va shu yo‘nalish bo‘yicha bilim va ko‘nikmalarini chuqurlashtirishiga qaratiladi. Ushbu shakllarga quyidagilar tegishlidir: - darslik va o‘quv qo‘llanmalar bo‘yicha fanlar boblari va mavzularini o‘rganish orqali referatlar tayyorlash; - o‘qituvchi rahbarligida ilmiy-amaliy izlanishlar olib borish orqali to‘garaklar va semenarlar uchun ma’ruzalar tayyorlash: - kompyuterda amaliy dasturlar yordamida sohaga oid masalalrni yechish, yechimni tahlil qilish va qaror qabul qilish ko‘nikmalarini shakllantirish; -masofaviy (distatsion) ta’lim vositalari orqali berilgan mavzularni o‘rganish, nazoratlarni topshirish.
3	**V. Fan o‘qitilishining natijalari (shakllanadigan kompetensiyalar).** Fanni o‘zlashtirish natijasida talaba: - hayvon organizmida kechadigan patologik jarayonlar; sog‘lomlik va kasallik; patologik reaksiya; patologik jarayon; patologik holat; organizm reaktivligi; organizm rezistentligi; allergiya va anafilaksiya; yallig‘lanish; o‘smalar; isitma, gipotermiya va gipertermiyalar; qon aylanishining mahalliy buzilishlari; qon, qon aylanishi, nafas, hazm, endokrin va nerv tizimlarining patofiziologiyalari; gipo va giperfunksiyalar to‘g‘risida ***tasavvurga ega bo‘lishi;*** - hayvon organizmida kechadigan patologik jarayonlar; sog‘lomlik va kasallikni; kasallik haqidagi nazariyalarni; patologik reaksiyani, patologik jarayonni, patologik holatni; kasallik oqibati va tarqalish yo‘llarini; organizm reaktivligini patologiyadagi ahamiyatini; allergiya va anafilaksiyani; mahalliy qon aylanishining buzilishlarini; yallig‘lanish sabablari, mahalliy belgilari, bosqichlari va kechishini; atrofiya, gipertrofiya, regeneratsiya va transplantatsiyani; o‘smalarning sabablari, turlari va hayvonat olamida tarqalishini; isitmaning sabablari, bosqichlari hamda oqibatlarini; gipotermiya va gipertermiyalarning farqini; moddalar almashinishini buzilishlarini; qon,

	qon plazmasi va shaklli elementlarning o‘zgarish sabablarini; qon ivishining buzilishlarini; yurak va tomirlar tizimi o‘zgarishlari va oqibatlarini; nafas va uning bosqichlarini; nafasni plevra, o‘pka patologiyasida, qon va atmosfera havosi tarkibi o‘zgarishidan buzilishlarini; kislorod tanqisligini; hazm sistemasi organlari patofiziologiyasini; oshqozon shirasining sekresiya tiplarini; ichaklar o‘tkazuvchanligining buzilishlarini; jigar va buyrak faoliyati buzilishlarini; siydik miqdorini o‘zgarish turlari va sabablarini; ichki sekresiya bezlari faoliyatining buzilishlarini; asab tizimi faoliyatini buzuvchi umumiy sabablarni; asab tizimini harakatlantiruvchi, sezuvchanlik va trofik faoliyati buzilishlarini; vegetativ nerv sistemasi faoliyatlarining buzilishlarini ***bilishi va ulardan amaliyotda foydalana olishi;*** -hayvon organizmida kechadigan patologik jarayonlarni aniqlash; hayvonlarda o‘tkir va surunkali tajribalar o‘tkazish; hayvonlarda o‘tkir va surunkali tajribalarni modellashtirish; sog‘lom va kasal hayvonlarda klinik tekshiruvlarni o‘tkazish; laboratoriya tekshiruvlarini o‘tkazish ***ko‘nikmalariga ega bo‘lishi;*** -me’yor va patologiyani farqlash; o‘tkir va surunkali tajriba natijalarini tahlil qilish va baholash; sog‘lom va kasal hayvonlarning klinik, gemotologik ko‘rsatkichlarini aniqlash va ularni tahlil qilish ***malakalariga ega bo‘lishi kerak.***
4.	**VI. Ta’lim texnologiyalari va metodlari:** • ma’ruzalar; • amaliy va laboratoriya mashg‘ulotlari; • muammoli ta’lim; • kichik guruhlarda ishlash; • taqdimotlarni qilish; • individual loyihalar; • jamoa bo‘lib ishlash; • assisment.
5.	**VII. Kreditlarni olish uchun talablar:** Fanga oid nazariy va uslubiy tushunchalarni to‘la o‘zlashtirish, tahlil natijalarini to‘g‘ri aks ettira olish, o‘rganilayotgan jarayonlar haqida mustaqil mushoxada yuritish va joriy, oraliq nazorat shakllarida berilgan vazifa va topshiriqlarni bajarish, yakuniy nazorat bo‘yicha yozma ishni topshirish.

6.	**Asosiy adabiyotlar** 1. R.X. Xaitov., D.E. Eshimov. “Hayvonlar patologik fiziologiyasi”. Darslik. Toshkent, “Ilm Ziyo” – 2013 yil. 2. D.E. Eshimov., R.F. Ro‘ziqulov. “Hayvonlar patofiziologiyasi fanidan amaliy-laboratoriya mashg‘ulotlari”. O‘quv qo‘llanma. Toshkent, “O‘zbekiston faylasuflari milliy jamiyati nashriyoti” – 2017 yil. **Xorijiy adabiyotlar** 1. Ganti A.Sastry. Veterinary Patology. CBS Publishers. Distributors P Ltd (7 th edition). USA, 2011. 2. С.И.Лютинский. Патологическая физиология сельскохозяйственных животных. Учебник. Москва, Колос, 2011 год. **Qo‘shimcha adabiyotlar** 1. Mirziyoyev Sh.M. Erkin va farovon demokratik O‘zbekiston davlatini birgalikda barpo etamiz. Toshkent, “O‘zbekiston” NMIU, 2017 yil. 2. Mirziyoyev Sh.M. Qonun ustuvorligi va inson manfaatlarini ta’minlash yurt taraqqiyoti va xalq farovonligining garovi.“O‘zbekiston” NMIU, 2017 yil. 3. Mirziyoyev Sh.M. Buyuk kelajagimizni mard va olijanob xalqimiz bilan birga quramiz. “O‘zbekiston” NMIU, 2017 yil. 4. O‘zbekiston Respublikasi Prezidentining 2017 yil 7 fevraldagi “O‘zbekiston Respublikasini yanada rivojlantirish bo‘yicha harakatlar strategiyasi to‘g‘risida”gi PF-4947-sonli Farmoni. O‘zbekiston Respublikasi qonun hujjatlari to‘plami, 2017 yil, 6-son, 70-modda. 5. D.E. Eshimov., R.F. Ro‘ziqulov. “Hayvonlar fiziologiyasi va patofiziologiyasi fanidan amaliy-laboratoriya mashg‘ulotlari”. O‘quv qo‘llanma. Toshkent, “Tafakkur bo‘stoni” - 2011 yil. **Internet saytlari** 1. www.ziyonet.uz. 2. www.vetjurnal.uz 3. www.lex.uz 4. www.veterinariy.actavis 5. www.kodges.ru

7.	Fan dasturi Oliy va o'rta maxsus professional ta'lim yo'nalishlari bo'yicha O'quv-uslubiy birlashmalar faoliyatini Muvofiqlashtiruvchi Kengashning 2021 yil "17" 08 dagi 3 -sonli bayonnomasi bilan ma'qullangan. O'zbekiston Respublikasi Oliy va o'rta maxsus ta'lim vazirligining 2021 yil "18" 08 dagi 355 sonli buyrug'i bilan ma'qullangan fan dasturlarini tayanch oliy ta'lim muassasasi tomonidan tasdiqlashga rozilik berilgan.
8.	**Fan/modul uchun mas'ul:** **D.Eshimov** – SamVMI, "Hayvonlar fiziologiyasi, biokimyosi va patologik fiziologiya" kafedrasi mudiri b.f.n., dotsent.
9.	**Taqrizchilar:** **E.A.Toshmuratov**- Samarqand viloyat veterinariya va chorvachilikni rivojlantirish boshqarmasi boshlig'i. **B. Eshburiyev** - Sam VMI, "Veterinariya jarrohligi va akusherlik" kafedrasi, professori, v.f.d.

Oliy ta'limning 100000-Gumanitar bilim sohasining hamda 400000-Qishloq xo'jaligi bilim sohasining 5440100-Veterinariya meditsinasi (faoliyati turlari bo'yicha) 5440300-Veterinariya diagnostikasi va laboratoriya ishlari bakalavriat ta'lim yo'nalishlari uchun dotsentlar D.E.Eshimov va R.F.Ro'ziqulovlar tomonidan tayyorlangan "Hayvonlar patofiziologiyasi" fanining fan dasturiga

TAQRIZ

"Ta'lim" to'g'risidagi qonun va "Kadrlar tayyorlash milliy dasturi"ga mos ravishda qishloq xo'jaligini etuk, malakali mutaxassislar bilan ta'minlash borasida tayyorlanayotgan veterinariya vrachlari raqobatbardosh, yuqori saviyali, nazariy va amaliy bilimlarga ega bo'lishi lozim.

"Hayvonlar patofiziologiya"si fani kasal hayvon organizmidagi patologik jarayonlarni aniqlashda, ularni umumiy qonuniyatlarini o'rganishda, me'yor va patologiyani farqlashda etakchi o'rinda turadi.

Dasturda fanning maqsadi va vazifalari, fan bo'yicha talabalarning bilimi, malaka va ko'nikmalariga qo'yiladigan DTSga muvofiq talablar, o'quv rejasidagi boshqa fanlar bilan o'zaro bog'liqligi va uslubiy jihatdan uzviy ketma-ketligi, fanni o'qitishda zamonaviy axborot va pedagogik texnologiyalar, o'qitish semestrlari va uslubiy ko'rsatmalar, fanning bo'limlari bo'yicha mazmuni, amaliy mashg'ulotlar, laboratoriya darslari va mustaqil ishni tashkil etish uchun ko'rsatmalar, dasturning informatsion-uslubiy ta'minoti hamda foydalaniladigan asosiy darslik va o'quv qo'llanmalar ro'yxati alohida-alohida berilgan.

Hayvonlar patofiziologiyasi fani uch qismga: umumiy nozologiya, tipik patologik jarayonlar, organ va sistemalar patologiyasiga bo'lib o'rganiladi.

Umumiy nozologiya qismida kasallik haqida tushuncha, kasallikga sabab bo'ladigan fizik, ximik, biologik omillar, kasallikning kechishi, rivojlanish mexanizmi (patogenez) va oqibatlari o'rganiladi. Dasturning bu qismida organizmning reaktivligi va uning patologiyadagi ahamiyatiga ham keng o'rin berilgan.

Dasturning tipik patologik jarayonlari qismida esa qon aylanishining mahalliy buzilishlari, yallig'lanish, istma, o'smalar, modda almashinuvi va och qolish patologiyasi kabi mavzularningetiologiyasi, patogenezi, mohiyati o'rganilib, veterinariya vrachlarining tafakkuri va dunyoqarashini shakllantirishda katta yordam beradi.

Dasturning organ va sistemalar patofiziologiyasi qismida qishloq xo'jalik hayvonlari organizmidagi organ va sistemalarda uchraydigan patologik jarayonlarning etiopatogenezi, oqibatlari va immun tizimning patofiziologiyasi o'rganiladi.

Umuman olganda "Hayvonlar patofiziologiya"si fanidan yozib tayyorlangan dastur hozirgi davr va DTS talablariga javob beradi deb hisoblayman va uni tasdiqlab, o'quv jarayonida qo'llash uchun tavsiya etaman.

Samarqand viloyat veterinariya va chorvachilikni rivojlantirish boshqarmasi boshlig'i **E.A.Toshmuratov**

Oliy ta'limning 100000-Gumanitar bilim sohasining hamda 400000-Qishloq xo'jaligi bilim sohasining 5440100-Veterinariya meditsinasi (faoliyati turlari bo'yicha) 5440300-Veterinariya diagnostikasi va laboratoriya ishlari bakalavriat ta'lim yo'nalishlari uchun dotsentlar D.E.Eshimov va R.F.Ro'ziqulovlar tomonidan tayyorlangan "Hayvonlar patofiziologiyasi" fanining fan dasturiga

TAQRIZ

Mustaqil Respublikamizning qishloq xo'jaligini malakali mutexassislar bilan ta'minlash borasida tayyorlanayotgan veterinariya vrachlari yuqori saviyali, nazariy va amaliy bilimlarga ega bo'lishi lozim.

"Hayvonlar patofiziologiyasi" fani uchta modulga: umumiy nozologiya, tipik patologik jarayonlar, organ va sistemalar patologiyasiga bo'lib o'rganiladi.

"Hayvonlar patofiziologiyasi" umumkasbiy fanlardan biri bo'lib, veterinariya vrachlarining kasal hayvonlar organizmidagi patologik jarayonlarni aniqlashda, ularning umumiy qonuniyatlarini o'rganishda, me'yor va patologiyani farqlashda etakchi o'rinda turadi.

Fanni o'rganish uchun ma'ruza, amaliy mashg'ulot, laboratoriya darslari va mustaqil ishlash uchun etarli darajada dars soatlari ajratilgan.

Dasturning "Kirish" qismida: fanning maqsadi va vazifalari, fan bo'yicha talabalarning bilimi, malaka va ko'nikmalariga qo'yiladigan DTSga muvofiq talablar; o'quv rejasidagi boshqa fanlar bilan o'zaro bog'liqligi, fanning ishlab chiqarishdagi o'rni, o'qitish semestri va uslubiy ko'rsatmalar berilgan.

Dasturning "Asosiy qismi"da fanning mazmuni, amaliy mashg'ulotlar, laboratoriya darslari, mustaqil ish uchun ko'rsatmalar, dars uchun kerak bo'ladigan "Informatsion uslubiy ta'minot" hamda "Darslik va o'quv qo'llanmalar ro'yxati" alohida-alohida berilgan.

Dasturda ma'ruza va amaliy-laboratoriya darslarining mavzulari rejalashtirilgan soatlarga qarab taqsimlangan.

Shuning uchun "Hayvonlar patofiziologiyasi" fanidan yozib tayyorlangan o'quv dasturi hozirgi davr talabiga javob beradi deb hisoblayman va uni tasdiqlashga hamda o'quv jarayonida qo'llash uchun tavsiya etaman.

Sam VMI, "Veterinariya jarrohligi va akusherlik" kafedrasi, professori, v.b.d. **B. Eshburiyev**

Science worker study program

REPUBLIC OF UZBEKISTAN
MINISTRY OF HIGHER AND SECONDARY SPECIAL EDUCATION

SAMARKAND INSTITUTE OF VETERINARY MEDICINE

Registered:
№ BP-5440100-2.10
"___" _____ 2022

"APPROVED"
Vice rector for academic affairs
Eimurodov AA
"___" _____ 2022

ANIMAL PATHOPHYSIOLOGY

WORKING CURRICULUM OF SCIENCE

Field of knowledge: 400000 - Agriculture and Water Management

Field of education: 440000 - Veterinary

Areas of study: 5440100-Veterinary medicine (by types of activity)

Samarkand - 2022

The working curriculum (syllabus) of science has been developed in accordance with the approved curriculum and science program for 2021.

Developers:

Eshimov DE Head of the Department of "Animal Physiology, Biochemistry and Pathological Physiology", Associate Professor, Candidate of Biological Sciences.

Ruzikulov RF - Associate Professor of "Animal Physiology, Biochemistry and Pathological Physiology", Candidate of Veterinary Sciences.

Babayeva Sh.A. - assistant of the department of physiology biochemistry and pathology of animal physiology

Reviewers:

Toshmuratov EA - Head of the Samarkand Regional Department of Veterinary and Livestock Development.

Eshburiyev B. - Sam IVM, Department of Veterinary Surgery and Obstetrics, c.v.s. Professor,

Working curriculum of science "Animal physiology, biochemistry and pathological physiology" Discussed at the meeting of the department "26" in 08 2021 "1" and recommended for discussion at the faculty council.

Chair holder, c.b.s., Associate Professor _____ **Eshimov.DE**

The working curriculum of the subject was discussed and recommended for use by the Board of the Faculty of Veterinary Diagnostics and Food Safety (Protocol No. 1 of 27 08, 2021).

Faculty Council Chairman, c.v.s., Professor _____ **Davlatov.RB**

Agreed:
Educational-methodical head of department, c.v.s, Associate Professor _____ **Ruzikulov RF**

Syllabus of science
Samarkand Institute of Veterinary Medicine

Information of science
Code of science:HPM 2306
Name of science: **Animal pathophysiology**
Semester / year: **4th semester / 2021-2022 year of study**
Chair: Animal physiology, biochemistry and pathological physiology
Hours / credits: 6.0 ECTS **(90 a classroom hours, 90 hours of independent study)**

Lecture	Practical exercise	Laboratory exercises	Independent education	Total
30	30	30	90	180

Location of science classes:
Audience time: according to the course schedule
Requirements:
Department of Science: **Animal physiology, biochemistry and pathological physiology**
Information about instructors
Candidate of Biological Sciences, Associate Professor Eshimov DE
Phone: +998979218087
E.mail.
Candidate of Veterinary Sciences, Associate Professor. Ruzikulov RF
Phone: +998937231300
E.mail.
Assistant Babayeva Sh.A.Phone: +998906560849
E.mail.shaxlo-babayeva@mail.ru
Location of the department: SamVMI, 2nd educational building, room 241
Hours: By appointment

I. The purpose of teaching science -This subject covers such topics as the results of socio-economic reforms in the country, the prospects of animal husbandry and veterinary medicine, general changes in the body of sick animals and poultry, their impact on animal productivity, pathophysiology of various systems in animals.

The subject "Animal Pathophysiology" is an integral part of the veterinary system to explain to students the pathological processes in animals and their impact on animal productivity, to distinguish between norms and pathologies in animals, to teach scientific and practical laws of disease development. is calculated.

"Animal pathophysiology" is a subject included in the block of general professional disciplines and is taught in the 2nd stage. This science forms the theoretical and practical basis of veterinary sciences and in its development serves as a basis for general and specialized disciplines in the field of veterinary education.

II. Science function - To teach students the general changes that occur in the diseased organism. the emergence, causes, conditions, mechanisms of development,

Training materials for lectures

THE SCIENCE OF ANIMAL PATHOPHYSIOLOGY AND THE HISTORY OF ITS DEVELOPMENT.

Plan:

1. The content, purpose, function and interrelation of science with other sciences.
2. History of the development of animal pathophysiology.

Main publications:

1. Khaitov RX, Eshimov DE Pathological physiology of animals, Textbook Tashkent, "Ilim Ziyo" 2013.
2. M.Donald., James F. “Pathologic basis of veterinary disese” Humana Press; 2011 edition.
3. S.I.Lyutinskiy. Pathological physiology of agricultural animals. Moscow, Kolos, 2001.

Additional literature

1. DEEshimov., RFRuzikulov. "Practical and laboratory classes in animal physiology and pathophysiology." Study guide. Tashkent. Tafakkur Bostoni - 2011.

Internet information:

1. www.ziyonet.uz.
2. www.vetjurnal.uz
3. www.lex.uz
4. www.veterinariy.actavis
5. www.kodges.ru

Base phrases.

Animal pathophysiology general law, artificial model, K.Bernard, Mering, Minkovsky, Fleming, Peer Marie, biochemistry, clinical diagnostics, experiment, general, typical and specific pathophysiology, general nosology, etiology, pathogenesis, reactivity, inflammation, local circulation disorder, fever, tumor, hyper and hypobiotic processes, STZabelin, AMFilomafitskiy, VVPashutin, ABFoxt, GPSakharov, AFAndreev, VVVoronin, VANegovskiy, CMPavlenko, ADAdo, AAJuravel, AVABodniyskov, NVPodvisotskiy, ADAdo, AAJuravel, NVPodvisotskiy , LRPepelman, MPTushnov, VVKoropov VMVolkov, RXXaitov, RPPo'latov.

1. Animal pathophysiology is the study of changes in the body of sick animals, the causes, conditions, mechanisms and consequences of disease, or the physiology of diseased animals.

One of the main tasks of the science of animal pathophysiology is to reveal the general laws of the development of pathological processes, to study the causes and mechanisms of development and course and consequences of the disease.

The science of animal pathophysiology reveals the general laws of the mechanisms of development of diseases, opens the way to the essence of the disease, a clear diagnosis, prevention and treatment of the disease.

The science of animal pathophysiology is one of the most important disciplines in the field of veterinary medicine, which forms the worldview of the doctor (judge), scientifically substantiates the origin of diseases and creates new, modern methods to replace outdated, unconventional preventive measures and treatment processes.

Animal pathophysiology is an experimental science and consists of two words: Greek Pathos - disease, illness, logos - doctrine.

The main and main method of the science of animal pathophysiology is 'experiment'. This science seeks to teach in-depth, comprehensive study of various pathological processes, diseases and

their artificial models, artificially using the method of experiments. It teaches the importance of various factors in the pathogenesis of the disease, the mechanisms of disease development, the consequences of the flow.

With the help of pathological experience, the necessary conditions are created to study the causes of diseases in the past, present and future, and this is important. In studying the glycogen-forming properties of the liver, K. Bernard studied the amount of carbohydrate in the blood that goes to the liver and is present in the blood vessels leaving the liver, and found that the blood leaving the liver is low in carbohydrates.

The glycogen-forming properties of the liver were also studied by Mering and Minkovsky, who observed an increase in the amount of glucose in the blood when they examined the blood by tying two pancreatic ducts, thereby demonstrating the importance of hormones in the body. In experiments, Peer Marie proved that hypofunction of the pituitary gland leads to stunting, and hyperfunction leads to acromegaly. American scientist Simones studied the occurrence of cachexia when the function of the pituitary gland is reduced. When the Russian scientist Lunin took two groups of mice and fed one group with artificial and the other group with natural milk, a few days later the artificial milk-fed mice lost weight, lost their growth and their hair fell out, and their skin began to change. .

Trying to determine the importance of vitamins, VVPashutin feeds rabbits with sauerkraut and observes that rabbits are susceptible to sinus disease, but cannot explain the mechanism of its development.

The hypothesis of vitamins was given in 1911 by Kazimir Funk, a Polish biochemist working in London. He isolated a white crystalline substance from rice bran that could cure the disease and called it a vitamin. Latin-Vita means life amine, a chemical compound that contains nitrogen. K.Funk believes that diseases such as scurvy, pellagra, rickets, and beriberi are caused by a lack of vitamins in the body. Studies in recent years have confirmed that most vitamins do not contain nitrogen. Nitrogen-free vitamins include A, D, E, K, C. In the past, experiments have been conducted in a short period of time, using sharp experiments.

Therefore, the experiment was developed in the hands of IPPavlov, who conducted it using chronic methods.

IPPavlov spent 10 years in the SPBotkin laboratory, where the effects of caffeine, camphor, bromine on blood vessels, in particular, affecting the heart nerves, changes in blood pressure, changes in blood pressure in dogs under the influence of drugs, suturing the carotid artery in dogs , learns.

For 20 years, IPPavlov improved the methods of fistula in the physiology of the digestive system. 'rganadi. To study the role of the nervous system in digestion, the method of esophagotomy of animals explains the reflex separation of gastric juice as a result of "lying" feeding. Based on these methods, creates a diet.

IPPavlov devoted 35 years of his life to the study of mental activity and behavior of humans and animals.

The pathophysiologist uses pathological experimentation to study the causes of the disease, determine its course, find measures to prevent the disease, and develop ways and means of treating the disease, which in turn helps the practice. In particular, in the 18th century, when French wines began to turn into vinegar, IPPaster developed a method of washing and disinfecting wine containers in boiling water. When silkworm disease occurs, it is recommended that the silkworm storage rooms be cleaned of contaminants, proving that silkworm disease is caused by microorganisms.

When Louis Pasteur grows bacteria that cause cholera (malaria) and the thermostat door is accidentally left open, a few days later, he observes that the growth of cholera microbes is weakened and when he injects this microbial wash into the chickens, the chickens do not get sick. Thus, a vaccination method is created. British scientist Fleming planted in petri dishes to study the disease-causing properties of streptococcal microbes, and when the surface was left open, fungi fell on the planted microbe, partially killing the microbes and, based on this, the first antibiotic, penicillin, was created. So the importance of experiments is significant. On the importance of experiments, IPPavlov recommends paying attention to the following two important processes:

1. Observations should be given close attention;

2.He says we study nature by focusing on the experimental method.

The French scientist Couve says that by the method of observation we hear nature and in practice we force the opening and submission of nature.

Three different problems are studied in the science of animal pathophysiology:

1.Nosology is the general doctrine of disease. In nosology, the doctor faces two different issues: one is why the disease occurs and what is the mechanism of its development (etiology, pathogenesis)? In the origin of the disease is studied the importance of the type, breed, sex, heredity and constitution of the animal, as well as the characteristics of disease resistance - reactivity.

2.The general typical cases that occur in all diseases and underlie all diseases or are observed in their origin are studied:

a). Local circulatory disorders;

b). Inflammation;

v). Fever;

g). Hyper and hypobioses.

In the special pathophysiology part of the science of animal pathophysiology teaches pathologies of organs or systems: blood, blood circulation, respiration, digestion, liver, digestive organs, endocrine glands and nervous system.

Later he began to teach pathophysiology and normal physiology AMFilomafitsky (Head of the Department of Physiology, Moscow University). Since he was not divided into in-depth knowledge at the time, he taught only some of the symptoms of the disease, without knowing the course of the disease. It teaches the origin of diseases by linking them to divine power. Therefore, AMFilofitsky begins to study a number of diseases in practice, as it is expedient to observe and study the disease. For example: the importance of the nervous system in cough, the method of blood transfusion, transfusion of fibrin-deficient blood, reviving dogs, and writing a work in this area, he has not lost its value so far. Nutritional chemistry is studied in the laboratory of AMFilomafitsky, and in 1842 in this laboratory VABasov developed a method of inserting a tube-fistula in the stomach of a dog. AMFilomafitsky studies various pathological processes in Russia under a microscope. For example: erythrocytes from the shaped elements of the blood, observed changes in urine output during the disease. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science.

VVPashutin, based on several experiments, knowing the importance of the nervous system, opposes R. Virkhov's cell pathology and explains that the processes taking place in the cells depend on the nervous system. Experimental observation of the formation of various pathological processes in the body as a result of lack of various substances, the study of the mechanism of origin of scurvy, feeding rabbits with sauerkraut. As a result, it is concluded that the disease is caused by a lack of any additional nutrients to the organisms. Lunin then justifies the lack of vitamins. That is why VVPashutin is called the gift-pioneer of the doctrine of vitamins.

VVPashutin organizes the largest school of pathophysiologists in Russia. One of his students was MPAlbitsky (after Pashutin he was the head of the department), AVReprov was the head of the physiology department at the Khorkov Medical Institute, X-ray exposure, endocryology. He founded an independent school of pathophysiologists at the Kharkiv Medical Institute, where he studied the pathology of gas, heat, metabolism and endocrine systems from his students DEAlperin, SMLeytes and others. Academician ADTimofeevsky worked on tumors and studied whether tumors can be

grown under artificial conditions. It is a state award winner for growing large tumors from a single cell in vivo and in vitro (inside and outside the body). Lunin works in the field of vitamins. AP Likhachev works in the field of gas exchange. VVPashutin died of a heart attack in 1901 while working as the rector of the Academy of Medical Surgery.

2- The School of Animal Pathophysiology was founded at the University of Moscow under the direction of Alexander Bogdanovich Foxt (1848-1930), a student of AIPolunin. It studies the pathological processes occurring in organ tissues, including: lungs, heart system. Creates a model of artificial pores of the heart and studies it in detail. He studies the formation of constipation in the lungs and heart in cardiovascular pathology, pulmonary, cardiac dysfunction. Professor Govril Petrovich Sakharov from the ABFoxt laboratory in the field of allergy and endocrinology, AI Talyansev develops methods of peripheral circulatory pathology, VVVoronin inflammation, AFAndreev clinical death and general resuscitation of the organism. VANegovsky studied animal pathophysiology of the cardiovascular system, on this basis he created a complex method of resurrection. GPSakharov and his students SMPavlenko and AAJuravel worked in the field of reactivity, immunology and endocrinology.

3- The School of Animal Pathophysiology in Kiev and Odessa was founded by Vladimir Valeryanovich Podvesotsky (1857-1913), who developed the humoral theory of immunity, a parasitic theory in the field of tumors. He worked on the regeneration process. He has written a textbook on animal pathophysiology and has published it in several languages. He published a journal, The Archive of Pathology and Medicine, to promote the science of animal pathophysiology. His students are LATarasevich and ITSavchenko, academician AABogomolets and others. They studied the problems of immunology, reactivity of the organism, endocrinology, and they always worked under the direction of II Mechnikov. LATarasevich and IT Savchenko worked on agglutinin, precipitate, antibodies in France at the suggestion of IIMechnikov.

Academician AABogomolets works in the field of animal pathophysiology, studying the role of reactivity in pathology, its relationship to endocrine management. He was born in 1881 in Petropavlovsk Prison and died in 1946. His mother was imprisoned for being a member of Russia's "southern liberation group."

Academician AABogomolets is the President of the Ukrainian Academy of Sciences and the First Deputy Chairman of the Presidium of the Supreme Soviet of Ukraine. Pathophysiologist-pathologist since 1924. By developing the pathophysiology of animals, he created the original pathological doctrine in medicine, which is called the physiological system of connective tissue. In addition to the supporting function of the connective tissue, it performs a trophic function and a plastic-building function. As it is composed of RES cells, it enhances phagocytosis and antibody production. Improves connective tissue function using antiretroviral cytotoxic serum. It was actively used in the treatment of many diseases during World War II. Academician AABogomolets Director of the All-Union Blood Transfusion Institute, developed a method of conserving blood (the first among physicians to be awarded the title of sos. labor hero). He identified 4 different types of constitutions depending on the nature of the connective tissue and observed more or less common diseases, depending on these constitutions. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize.

Academician IISirotin worked on the field of acclimatization of the organism and the reactivity of the organism.

Academician REKovetsy studied the origin of tumors and the characteristics of their development in different conditions, the course of metabolism in tumors.

Academic ADAdo has worked on allergic diseases, anaphylaxis, lung disease, and has written a textbook on Animal Pathophysiology.

The new schools of animal pathophysiology were headed by well-known scientists LATaraseevich, AVReprev, ESLondon, AABogomolets SSKholatov, GPSakharov, NNAnichkov, ADSperansky.

Academician NNAnichkov (1885-1965) studied in depth the pathophysiology of the cardiovascular system, the involvement of RES cells in pathological processes and the mechanisms of origin of arteriosclerosis in the Department of Pathophysiology, Pathanatomy of the Military Medical Academy.

Experiments show that indifferent influencers play an important role in the development of diseases. For example, if dogs are injected with apomorphine for 15 days and supported with light, in the following days only the lighting of the lamp causes them to vomit reflexively. He suggested that organs could not be studied in isolation from the body, and that systematic scientific work should be carried out. He founded a large school of pathophysiologists, and even today scientists from the ADSperansky school are actively working in research institutes and universities.

IIRavich, the founder of veterinary pathophysiology, worked in the veterinary department of the Academy of Medical Surgery in St. Petersburg, critically examining Virkhov's cell theory and acknowledging the importance of the nervous system in the origin of the disease. He wrote a textbook on general zoopathology and lectured to students on the subject.

Academician MPTushnov (1879-1935), Head of the Department of Pathophysiology of the Kazan Veterinary Institute, created an original drug in pathophysiology, the lysates of which are the products of the decomposition of various organs. For example, muscle lysates are called myolysates, and when animals are released when they are tired, their ability to work is restored, mammolysates are prepared from the udder and increase the amount of milk, and ovariolysates accelerate the maturation of egg cells. Lysates are now the most common and widespread type - biostimulants. They are used in the growth and development of young animals, increase productivity and treat many diseases. Biostimulants are widely used in fattening. Including, Chlorella, which is found in billions of water, has been used to enhance productivity by enhancing all the processes that take place in animals. Currently, there are more than 45 departments of veterinary pathophysiology in veterinary institutes and faculties of the CIS countries, which are studying the effects of biostimulants on the characteristics of the organism. Most research veterinary institutes are studying the effects of biostimulants on the body's reactivity, metabolism and neuro-endocrine control processes.

The contribution of the French scientist Claude Bernard (1813-1878) in the development of the science of animal pathophysiology was significant. K. Bernard's work is studied in two periods:

The first period involved 20 years of normal physiology, proving the liver’s glycogen production function and determining its reflex mechanism. The origin of diabetes in the body proves that it is associated with dysfunction of the CNS. Demonstrates the importance of pancreatic juice and bile in the breakdown of nutrients, as well as observed an increase in body temperature. Blood and lymph determine the organization of the internal environment of the body and determine vital processes.

The second period. He has been working in experimental physiology for 10 years. It studies the importance and function of various nerve fibers in the body, the electrical properties of nerve and muscle tissue, the properties of blood, and the effects of SO2 on the body. Proves a violation of saliva production from salivary glands. A substance called Curare affects the endocrine glands and observes a decrease in the secretory process. He studied various pathological processes of the respiratory system and wrote more than 180 scientific sources, which consist of 18 volumes. K. Bernard did a lot of work despite experiencing great difficulties. He teaches that the processes that take place in the body depend on the vital force, and that that force is random.

IPPavlov says of K. Bernard, "K. Bernard is a scientist who thought broadly and deeply in his mind, generalized physiology, experimental physiology, and experimental therapy as a whole, or combined the achievements of physiology with practice."

The famous chemist Dumas says, "K. Bernard is not only a physiologist, but he is a physiologist."

IPPavlov's doctrine is important in the development of animal pathophysiology. Prior to IPPavlov, observations were made in pathophysiology using analytical methods. Diseases of isolated organs, their integral parts have not been studied with attention to the living conditions of the animal, changes in the external environment and other related connections. IPPavlov, on the other hand, pays great attention to experimental scientific work and observes changes in body systems in healthy organisms in chronic experiments. According to IPPavlov's theory of nervousness, it is emphasized that any pathological processes in complex organisms are carried out with the participation of the nervous system, in particular, with the participation of higher nervous activity.

The organization and development of the science of animal pathophysiology in Uzbekistan was associated with the formation of the former Soviet Union, which began with the establishment of universities and research institutes in accordance with the decree of the Soviet government. As a result, the medical faculty of the Central Asian State University was established in Tashkent, which was later transformed into Tashkent State University, and intensive work in this area began. In 1921, the first department of "General Pathology" was established at Tashkent State University, which was later renamed the Department of Animal Pathophysiology. The first departments of pathophysiology were established in Samarkand in 1930, in Andijan in 1957, and in 1972 at the Central Asian Institute of Pediatrics.

At the Uzbek State Agricultural Institute in Samarkand, Farkhodi first studied veterinary pathophysiology, and from 1936, the head of the department, Associate Professor Vladimir Valerianovich Volkov. VVVolkov was an encyclopedic lecturer, a skilled experimenter, an excellent pedagogue-coach. VVVolkov was the initiator and organizer of several original scientific works with the staff of the department:

1. The causes and mechanisms of development of allergies and anaphylaxis in astrakhan sheep and goats in hot conditions;

2.Study the causes and mechanisms of development of pneumonia in sheep and goats during the summer months;

3. He has done a lot of research in the field of pathology of the region, the causes of the disease "Suyluk" in horses, the mechanism of its development and the development of methods for its detection. Today, the disease is found in humans and animals and is called trichodesmatoxicosis. In this field NXShevchenko and FIIbodullaev defended their doctoral dissertations and supervised several candidate dissertations.

4. The study of the enhancing effect of cytotoxins formed in tissues on the immunological properties of the organism of various laboratory animals (accelerated formation of antibodies to paratyphoid and colibotseliosis strains).

5. A detailed study of the effects of the parasympathetic division of the autonomic nervous system on the organism of experimental animals.

6. He made a great contribution to the training of a large number of highly qualified personnel. After the untimely death of VVVolkov in 1953, the department was headed by Associate Professor Anton Ivanovich Yarmashkeevich.

Extensive development of scientific work carried out at the department, mainly since 1961 under the leadership of Associate Professor, now Professor Ruzi Haitovich Haitov. By this time, the staff of the department sent different amounts of extracts from the liver, spleen and other parenchymal organs to healthy and sick animals, depending on the timing of their delivery, studied the mechanism of their action and developed a number of recommendations. In the Department of Animal Physiology and Pathophysiology, tissue feeding of animal feeds has proven to have a positive effect on the growth and development of the organism and the treatment and prevention of various diseases.

Having studied the effects of many drugs against helminthiasis, a number of recommendations have been developed. The genetic features of natural immunity, especially in karakul sheep and lambs of different colors, have been extensively studied and are still being studied. In this area, Associate Professor ADDushanov developed a synthetic vaccine, which gave good results, and Associate Professor MAAbdullaev in collaboration with the senior lecturer of the department RFRuzikulov conducts important research. Under the leadership of Professor RXKhaitov«Veterinary basics»Volume 1-2, 1972, RHHaitov and A.Dushanov on "Animal Physiology" in 1975, RHHaitov and Associate Professor MA Abdullaev on "Animal Pathophysiology of Agricultural Animals" in 1980 in Uzbek, a number of manuals, He has published more than 400 scientific articles in various collections, scientific collections of universities, research institutes, international and CIS congresses and conferences. Under the direct supervision of scientists of the department 10 doctoral and 342 candidate dissertations were defended in specialized scientific councils. Researchers of the department have been writing reviews and defending PhD and doctoral dissertations in many fields of physiology. And so, In Uzbekistan, pathophysiologists study the theoretical and practical processes of modern veterinary and medical science at the Department of Pathophysiology of the Veterinary Faculty of Samarkand Agricultural Institute, the Uzbek Veterinary Research Institute, pathophysiology laboratories of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad.

In order to strengthen the study of pathophysiology, the "Society of Pathophysiologists of Uzbekistan" was established, which includes more than 100 pathophysiologists. Pathophysiology and research work have been carried out in cooperation with veterinary institutes in Moscow, St. Petersburg, Kiev, Kazan, Almaty, Yerevan, and are still connected. As a confirmation of this strong unity, the fact that the 2nd pathophysiologists' session was held in 1972 in Tashkent is a proof of our opinion.

QUESTIONS FOR CONTROL

1. What is the science of animal pathophysiology?
2. Understand the functions of the science of animal pathophysiology?
3. What is the main method of animal pathophysiology?
4. Explain the importance of experiments in animal pathophysiology?

NOSOLOGY

Plan:

1. The concept of health (norm) and disease.
2. The concept of pathological reaction, pathological process and pathological conditions.
3. Description of the disease, types, course, restoration of vital processes in the body - resuscitation.
4. Anabiosis: seasonal and latergic sleep.

Main publications:

1. Khaitov RX, Eshimov DE "Pathological physiology of animals" Textbook Tashkent, "Ilim Ziyo" 2013.
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BASIC EXPRESSIONS.

Animism, evil spirit, divine or spiritual power, pneumonia of life, Archaea, humoral, Hippocrates, solid, Democritus, cellular, R. Virkhov, Golen, Sels, Avicenna, yatrophysics, iatrochemical, Paracelsus, vivisection, nervousness, norm, disease, contagious , infectious, organ system, acute, moderately acute, recovery and death, symptoms, acute, chronic, incubation, latent, prodramol, period of clinical manifestations, outcome, sanogenesis, relapsing , death, anabiosis, secondary sleep, prophylaxis.

1.Information about the disease has been of interest to people since ancient times. Because science and enlightenment did not develop in the primitive community system, and people did not know the origin of natural phenomena, they thought only about the visible and the invisible. That is why the organism has been described as composed of mythical things found in nature, such as soil, air, water, wood (metal), and fire. Illness, on the other hand, was interpreted as being caused by an invisible divine (supernatural force) or "SPIRIT" - anima. This current is called the "ANIMISM" current or theory, and it is a picture that all diseases are invoked by this supernatural force, the evil spirit. Talented physicians began to appear in Greece 4-5 thousand years BC, who wrote down what they knew, what they asked someone, their observations on the patient, and bequeathed this knowledge to their descendants. As a result, medical science began to develop slowly. For example, they recorded discharge from the mouth, nose, and ears in various diseases, fever, foul odors, and so on. Later in Greece, doctors explained that a living organism was composed of 4 different fluids in addition to 5 different elements (blood, mucus, black and yellow grass). Thus, the current that explains health and disease with these four different fluid properties is called the Humoral Flow or Theory. So, if the fluids are normally mixed properly, health is a sign of health, and this condition is called krazia or krazis. If, for some reason, the ratio of fluids is disturbed or the juices are contaminated, improper mixing, the disease can lead to dyscrasia or**«Discrasion»**The founder of this movement is the Greek scientist Hippocrates, who lived in the 4th-5th centuries BC.

Hippocrates was an observer, a disease-seeker, a traveling physician, who always traveled from village to village, making many observations on patients, studying the symptoms, various features, currents, and consequences of many diseases, and writing dozens of works. The role of the external environment in the origin of diseases, with great emphasis on cleanliness, developed methods of diagnosis and treatment of many diseases. He developed the laws of medicine, and in medicine there is

the Hippocratic oath in medicine. The teachings of Hippocrates have been proven to be true for centuries and even now, and his works have not lost their value.

In addition to diseases, Hippocrates also tried to create constitutions of human temperaments, which included four different temperaments: choleric (yellow grass), melancholic (black grass), sanguine (blood), and phlegmatic (mucous fluid). 'p or less depending on.

The contemporary philosopher Democritus of Hippocrates also developed a theory of diseases, which he called the solid (atomic, particle) theory, which explains that diseases are caused by changes in the spacing of atomic particles in the body. This theory explains that the disease is caused by the narrowing or widening and thinning of the spacing of the particles. At the same time, idealistic schools of thought have sprung up in Greece, claiming that diseases are called by divine power, explaining that organ function, organ diseases, and their causes depend on a particular pneumonia of life. According to Plato, Aristotle explained that there are three kinds of divine or spiritual power that govern the lives of people and animals:

1. Spiritual power is located in the brain and controls the mental function of people.
2. The spirit of the animal is located in the heart and controls the movement and warmth of the animal.
3. Explains that the spirit of the plant is located in the liver and regulates digestion.

They explain that they believe that the causes of diseases are not in the external environment, but in the mental origin. At the beginning of the twentieth century, knowledge of the disease was developed by Roman physicians Galen and Sels, who, in addition to the three zinc origins, based their humoral flow on explaining that diseases often resulted from the breakdown of juices, distinguishing between hot and cold discrasions. developed treatment options. Based on the symptoms of the disease, they observed four specific symptoms of the disease: redness, edema, edema, pain, and these changes, which lead to dysfunction, called functio laesa. . Galen introduces the vivisection method into science.

After Galen, our compatriot was the famous scientist and philosopher Abu Ali Ibn Sino (Avicenna), who made a great contribution to the development of medicine. He was born in 980 in the village of Afshona, Romitan district of Bukhara region and died in 1037 in Hamadan. In 1980, Avicenna's 1000th anniversary was celebrated and her works were published. He wrote more than 300 works in various fields, especially in the field of medicine, and in 1020 wrote a book on the laws of medicine. It consists of 6 books in 5 volumes:

1. The book is devoted to the anatomy, physiology, causes, appearance, general treatment of diseases. Attention was paid to nutrition, health, deportation, vomiting, and blood transfusions.
2. The book describes more than 800 drugs derived from plants and animals.
3. The book is about diseases from head to toe, this book is dedicated to specific pathology and therapy.
4. The book deals with fever, various tumors, rashes, wounds, burns, bone fractures and dislocations, nerve injuries, injuries to the skull, chest, spine and limbs, poisons and poisonings - toxicology, makeup - is dedicated to keeping people beautiful. Recommended remedies against hair loss, obesity or weight loss. He wrote about rabies, smallpox, measles, leprosy, and plague.
5. The book describes the methods of preparation and use of drugs.

Avicenna's book, The Laws of Medicine, pays great attention to the methods of observation and experimentation in the study of diseases, and widely uses this method on various diseases. developed He identified many diseases, developed treatment methods, studied urinary incontinence, urinary tract infections, worm diseases, pulse heart disease.

In his multifaceted scientific work, Avicenna concluded that diseases must have had invisible causes, not divine powers, and that they were now identified as microorganisms.

Avicenna studied in detail the wounds, lung diseases, diabetes, plague, cholera, smallpox, leprosy, tuberculosis (tuberculosis) and many other diseases, especially in the origin of the disease. , boiled, proved that it is important to follow hygiene. He studied the effects of many drugs and found that mercury is important in diseases such as gonorrhea and syphilis. It has been proven that following a meal plan-diet is important in diseases. Although he did not know the functioning of the nervous

system, he thought about the nervous system, that is, tied the sheep to the wolf, and observed that a few weeks later the sheep became frightened.

Avicenna's work on TIB laws has been reprinted 25 to 30 times in Europe and Asia, and is still being published today, and has served as a guide for physicians. By the 14th and 15th centuries, Copernicus, a Polish scientist, described the movements of the planets in the sky, Giordano Bruno's rotation of the earth around the sun, the Spanish Servetus's small circulatory system, and Leonardo-Da Vinci's anatomical tracts. V. Garvey discovers a large circulatory system based on his experiments on rabbits and dogs.

By the fifteenth century, a new direction in medicine, the iatrochemical and iatrophysical currents, began to emerge, meaning Iatros-physician.

The chemist Paracelsus conducted many experiments to prove the structure of the organism, the need for chemical elements to survive, the importance of mercury, matches, steel, iron and other elements in the health or illness of the organism. concludes that it contributes, and explains that when archaea get angry, they cause disease without releasing these elements into the body.

Iatrophysicists connect the organs of the body to the parts of a machine and pump the heart, explaining health and disease according to the laws of physics and mechanics.

In the XV1-XV11 centuries, the pathological-anatomical direction developed, and Morgani, Bish, and others began to study the body structure of animals and humans. In 1543, the Italian scientist A. Vezali began to study the structure of the body by tearing apart the bodies. 1640 Descartes wrote the reflex doctrine, 1660 Malpighi lens using the lens, renal capillaries, liver, spleen, skin structure, erythrocytes, 1674 Levenguk lens sperm movement. Morgan and Bish wrote about the changes that occur in different organs in different diseases, which led to the development of the study of pathological processes.

This means that the external environment has had two different effects on the organism over a long period of evolution, and that the organism has become accustomed to these favorable and unfavorable effects, adapted and balanced. -slowly studied and adapted, these effects are called daily or physiological, adequate effects. The processes that take place under the influence of these influences are called physiological processes and are called the norm, abbreviated for short. The second type of effects are often referred to as sudden, strong, sudden, adverse effects, which are called harmful or disease-causing, inadequate effects, and the processes that take place under the influence of these effects are called pathological processes.

Norm or health is a set of influences, conditions, adapting to their currents in a certain period of time, making them suitable for life, necessary or physiological effects, and the processes that take place and develop under their influence. called normal processes. Norma is a process that takes place in a period of stagnation, when the organism is calm and peaceful.

1.Norma-Sergey Petrovich Botkincha stagnation of life processes
is the sum.
2. Norma-Ivan Mikhailovich Sechenov and Claude Bernard describe the organism
with the balance of the external environment.
3. Norma-Victor Vasilevich Pashutin described the structure of the organism and
is said to harmonize its functions.
4.Norma-Vladimir Valerianovich Podvisosky to the conditions of our body

The structure of normative organ systems, the state in which they function without disruption. In real life, the norm is a relatively stable, changeable situation, because the absolute norm does not exist in real life. For example: consider pulse, temperature, respiration.

When one wants to study a disease, one must study it by comparing it with the norm. Both disease and health are ongoing processes in the body, which differ from each other in quantitative and qualitative changes. At the heart of both processes are two opposing processes of assimilation and dissimilation. It is impossible to know the exact time of onset of the disease, but it can be determined only by the symptoms that appear at a certain stage of development. For example, sleep is caused by fatigue as a result of overwork, which is considered a normal physiological state of the body, but in

some severe infectious diseases, drowsiness also occurs, indicating a disease of the body: anthrax, typhoid, diabetes , tuberculosis and others.

1.SPBotkin described the disease as a disorder of the vital processes of the organism.

2.IMSechenov and K.Bernar described the disease as a violation of the balance of the external environment in contact with the organism.

3. VVPashutin explains the disease as a violation of the harmony of the structure and function of the organism. These descriptions of the disease provide insights into unilateral changes in the disease, ignoring various complex quantitative and qualitative changes and active processes during the course of the disease. Therefore, these definitions do not fully describe the diseases.

4. In an attempt to fully express the disease, IPPavlov proposed the following definition: a disease is an encounter of an organism with an awkward, pathogenic, gross cause and condition that affects it suddenly, suddenly, collision, ie mechanical shock, crushing, injury, exposure to chemical, physical influences or attack by microorganisms, this encounter is the beginning of a struggle between the organism and the cause , by activating all defense mechanisms against, removing pathogenic causes, cleared or enzymes, phagocytes, Acute flow diseases - from a few minutes, hours to several weeks: For example: infectious and parasitic diseases.

1. Moderate acute flow illnesses — from a few weeks to several months.

Chronic recurrent diseases are those that last for months or years, most of which are non-communicable and non-infectious.

Diseases occur in several stages as they develop in the body.

a). An incubation or latent or latent period is the time that elapses between the onset of the disease and the onset of the first symptoms of the disease. This period can range from a few minutes to a few hours, weeks, months, and even years. Tuberculosis, brucellosis, non-communicable diseases, leprosy, AIDS and others.

b). The prodromal or disease-reporting period has its own characteristics, during which general symptoms for the disease appear. For example: increase in body temperature, decrease in appetite, heart rate, rapid breathing, etc.

v). Outbreaks appear to be exacerbated during clinical trials.

g). The consequences of diseases are twofold: the animal is either cured of the disease, or the sick animal dies.

1.Diseases spread throughout the body - per kontinuitatem. As the disease progresses, one organ spreads due to adhesions to the other organ. For example, inflammation of the oral cavity continues to spread to the red intestine, then to the stomach, intestines, and so on.

2.The disease is spread by means of friction, adhesions - per kontiguitatem. Pulmonary pneumonia to the pleura and pericarditis - myocarditis, liver - stomach, etc.

The disease is transmitted through the blood and lymph - permestastazine. Many microorganisms are spread through the blood and lymph.

3. Diseases are transmitted through the nervous system - per nervorum, through nerve fibers, stolbnyak - congestion, botulism, polio and other diseases.

4. Diseases are spread by secretions, saliva, sweat, urine and feces.

Intermittent course of illness is a period of illness that is sometimes mild and sometimes severe.

The complete recovery of the body from disease is called sanogenesis. The consequences of the disease are of two types:

a). The body recovers from the disease.

b). The disease ends in death.

3. There are two types of recovery:

a). The body recovers completely from the disease.

b). The body recovers from the disease.

Recovery comes in two different ways: simple and complex. Simple ways of recovery are carried out by revealing various reflexes. For example: reflex agitation, excessive salivation, wiping

tears, vomiting, sweating, coughing, diarrhea, excessive urination and excretion, tickling of the nervous system, and others

In complex treatment, the body is decontaminated by complex processes using barrier barriers, RES organs - liver, spleen, lymph nodes, red marrow, leukocytes, especially T and B lymphocytes, antibodies, etc. the cause is removed, then partially or completely repaired as a result of the recovery process. Restitution is called ad integrum if the body is completely cured of the disease. Sometimes the body can recover from the disease and recur, and the body can be severely damaged, and this is called a lytic transition to a critical and mild course.

3. Diseases can lead to dysfunction of the body without complete recovery. When the body's ability to heal is completely reduced, the body dies from the disease if the doctor's treatment does not help.

3.Death - mortis, morbi -characterized by the cessation of the continuous process of assimilation and dissimilation in the body and the cessation of heart function and respiration.

There are two types of death depending on their origin:

1. Natural or physiological death.
2. Death due to disease or pathological condition.

If 100% of all deaths are considered, only 2% of them are natural deaths and the remaining 98% are deaths due to diseases.

The doctrine that explains the formation of death is called tanatogenesis. Death occurs in several stages and is called the terminal state, they are:

1. Agony-pre-death convulsions: (consisting of peripoganal and oganal period).
2. Clinical death.
3. Biological death

As a result of death, the following changes occur in the corpse:

1.The body cools - algar mortis drops from 10 in the first days and cools to 0.20 on the second day. Of course, these changes are due to environmental changes.

2. The appearance of spots on the body - livoris mortis on the side on which the animal is lying, more spots appear and look good in hairless, unpigmented areas.

3. Hardening of the body - rigor mortis solidification of colloidal substances. Hardening begins after 8-10 hours and goes from head to toe.

4.Decomposition of the body - maceration or autolysis is formed under the influence of putrefactive and microorganisms from the external environment in the body, and the carcass begins to smell foul. If these bacteria are not present in the body, the body will become waxy.

Observations show that the animal continues to live in organs and tissues for some time after death. For example: nails, hair, hair, growth, movement of the stomach, intestinal muscles, contractions and other signs are observed. Much work has been done on the possibility of resurrecting the organism at the time of death. This condition is called resuscitation. It has now been discovered and proven that it is possible to resurrect organisms that have died by accident, and that people and animals who have died from various traumas, excessive blood loss, suffocation during anesthesia, electric trauma, various tragic events is being resurrected. Kulyabko, a professor of physiology at Tomsk University, was the first in this field in 1902.

From 1912 to 1919, the American physiologist Karel was able to use a burdock chicken heart under artificial conditions.

In the laboratory, Academician Kravkov observed the growth of nails and fur when rabbits' ears and fingers were removed and placed in special liquids. So it is possible to resurrect individual organs.

Professor FA Andreev conducted many experiments on dogs in 1913 and concluded that by anesthetizing dogs, the dogs were resuscitated by sending blood to the body and the whole organism could be revived.

1928 At a congress of physiologists and biochemists in Tbilisi, Bryukhonenko and Chechulin demonstrate an interesting experience: cutting off a dog's head, injecting blood into its veins through rubber tubes, and observing the dog's condition. saliva begins to separate when you put the sausage in the bur. In 1966 he was posthumously awarded the Lenin Prize for his invention of the AIK instrument. In 1940, Sinitsin was able to transplant and hold the hearts of frogs and fish. Academician

VANegovsky created a common method of resuscitation in 1941-1945, which was suitable for the resurrection of many soldiers and officers during the Great Patriotic War. In nature, it is a near-fatal condition and is called anabiosis: and we can find it in the plant and animal worlds. In the process of long evolution, plants, animals, and microorganisms go into a state of anabiosis, adapting, in order to survive various adverse effects. For example, by reducing the osmotic pressure from extreme cold or heat, by reducing the oxygen in the air, by freezing and drying, special chemical conditions can be created, that is, by using protective substances, anabiosis can be formed. During anabiosis, all functions in animals are sharply reduced (body temperature, heart rate, respiration, metabolism are sharply reduced, reflexes are lost). Anabiosis occurs in worms, fish, frogs, hedgehogs, lizards, bears, and frogs.

In humans, a condition close to anabiosis is called secondary sleep. Lattergic sleep is caused by severe effects, severe illness, and nervous mental illness.

Aging is a three-phase process:

1. Aging in infancy.
2. Aging in adulthood.
3. Aging.

The main task of veterinarians is the prevention and treatment of various diseases. General prevention is a measure of disease prevention using various ways, methods and measures, which consists of complex economic, organizational and veterinary-sanitary measures, which are:

1. The work of improving the external environment, for this it is necessary to create cultural meadows, the transition to the zagon system, the exchange of meadows, the removal of poisonous plants found in the meadows, various harmful substances. Grasslands, barns need to be disinfected and mechanically cleaned. Surrounding the farm, arranging insulators, building cemeteries and animal cremation rooms, improving the reclamation condition of meadows, drying or increasing moisture, washing away salts and other activities:
2. Bacteriological, serological, biochemical, radioactive isotopes and other methods are used to determine the latent stages of the disease by various methods, with regular examinations, taking appropriate measures, ie X-ray machines, allergic methods, blood tests. Twice a year in spring and autumn medical examination is obligatory:
3. It is necessary to strengthen the natural resistance or reactivity of the organism in various ways:
4. It is necessary to treat with various drugs, vaccinations, extensive use of drugs prepared from immune serum, biostimulants, proteins and enzymes:
5. Promotion of veterinary and sanitary knowledge, etc.

General principles of treatment:

1. Treatment of the causes of diseases. Use of antibiotics, hormones, vitamins, etc.:
2. Treatment against the pathogenesis of diseases:
3. Against the symptoms of the disease: treatment of diarrhea, tachycardia, fever, cough, etc .:
4. Treatment by replacement.

QUESTIONS FOR CONTROL.

1. What is nosology?
2. What are the directions that explain the origin of diseases?
3. Explain diseases in relation to divine power?
4. Explain the diseases associated with fluid contamination?
5. Explain the disease by linking changes in the distance between atoms and particles?
6. Explain the disease by linking it to changes in the cells?

ETIOPATHOGENESIS.

Plan:

1. The concept of etiology and pathogenesis. Criticism of metaphysical views on etiology: monocaualism, conditionalism, currents of constitutionalism.

2. Causes, conditions and prevention of diseases. Pathogenesis and the mechanism of action of etiological factors in it. The main mechanisms of disease development. Ways of transmission of pathogenic agents in the body.
3. The role of neuro-humoral processes in pathogenesis. The role of animal type, breed, sex, and age in pathogenesis. Sanogenesis - healing. Protective-compensatory mechanisms that ensure the healing process of the body. The role of the nervous and endocrine systems in sanogenesis.

Main publications:

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2. M.Donald., James F. “Pathologic basis of veterinary disese” Humana Press; 2011 edition.
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BASIC EXPRESSIONS.

Etiology, A. Vezali, Malpighi, Louis Pasteur, Robert Cox, wine fermentation, silkworm disease, mechanical processing, boiling water, monocaualism, one cause, conditioning, condition, constitutionalism, racist theory, neuralism theory and practice unit, exogenous : mechanical, physical, chemical, biological, endogenous: circulatory disorders, mining, factory, cotton, inadequate, psychogenic, etiological factors, burns, poisoning, corticosteroid, stress, tension, resistance, breed, gender, age, compensatory organs mechanisms.

1. Etiology - teaches the general laws of origin of diseases in the body, their causes, a set of conditions. Etiology is the Greek word for aitia-cause, logos-doctrine.

According to IP Pavlov, the future should become a hygienic veterinary, hygiene. Therefore, it is necessary to protect the external environment, and a lot of work is being done in this area. IPPavlov said that it is necessary to know all the causes and conditions of the disease.

The doctrine that teaches the causes of disease is the result of a struggle between materialist and idealistic currents. This doctrine has explained the origin of diseases in a simple, mythical, teleological way, i.e. the disease is caused by the influence of zinc, contamination of juices, changes in their composition, decrease or increase, thinning of particles in the body or indicates that the disease is caused by thickening. Later in the Middle Ages the origin of diseases was badjahil zinc«archetypal»explained in connection with the wrath of God. As a result of observations, A. Vezali and Malpighi began to study the structure of the organism in depth. By this time, the development of industry, the production of dyes, the increase in the production of equipment, created favorable conditions for the study of the functions of the organism.

At the end of the 19th century, the production of wine and silk in many countries, including France, fell into disrepair. This poses great challenges for French scientists. As a result, Louis Pasteur, under his leadership, began to search for and find the causes of many diseases. As a result, they discover that microorganisms are the cause of wine fermentation and silkworm disease. Microorganisms can be used to prevent the deterioration of wine quality by washing wine containers

with boiling water and disinfecting silkworm rooms. Thus, by identifying the real causes of the disease, now world scientists are doing a lot of research behind microorganisms, and German scientist Robert Cox is discovering the causes of tuberculosis, Louis Pasteur cholera, rabies and other diseases. The discovery of these diseases, on the other hand, follows a certain pattern, and this current is called the monocaual current. Mopo-single, single, couza - means cause. This doctrine is one of the most advanced doctrines of this period and deals a severe blow to religious doctrines. However, this doctrine does not fully explain the causes and conditions that cause disease, because the entry of microorganisms into the body does not always cause disease. As a result, the doctrine arises that diseases are caused by changes in the sum of many conditions, not microorganisms, and this doctrine means the conditionic conditions called the doctrine of conditionalism. This doctrine is contradicted by the inability to explain the disease, claiming that there is no clear cause for the disease, negating the importance of microorganisms in the origin of the disease.

Constitutionalismproponents of the theory explain that the disease arises from the genotypic structure of the organism, as a result of a deficiency in the constitution. The constitution and genotype do not change at all, so the disease is interpreted as a fatal process or a top-down process. With the emergence of the theory of constitutionalism, many erroneous theories have emerged. There is a misconception that people with low genes and low constitutions should be confused with people with high genes and high constitutions. As a result, Nazi Germany wiped out many nations in order to create a new race, and racist theories still prevail in many countries. These teachings exaggerate the causes of disease,

Nervism explains that the organism is closely connected with the external environment, which is due to the nervous system.

In studying the doctrine of etiology, we must take into account the structure of the organism and the principles of their solidarity, that is, we must combine theory and practice closely, which can explain the etiology in detail.

The causes of the disease are studied into 2 major groups: external or exogenous, internal or endogenous causes.

External causative agents include mechanical, physical, chemical, biological, and other causes.

3. External environmental factors that cause disease.

External causes of the disease are those influencers that affect the body from the external environment and create a pathological process. The causes of the disease are studied in close connection with the organism without self-study of the external environmental factors, and the degree of origin of the disease depends on its nature. Environmental factors that cause disease include mechanical, physical, chemical, and biological causes. As a result of absorption (reserves and electricity, light energy) or reflexively (conditionally and unconditionally) into the closed automatic (IPPavlov) MNS through the place where all factors directly affect the organism of highly developed animals by reflector).

Mechanical factors causing the disease.

An influencer that affects the body from the external environment, causing an injury to this or that in the body, is called trauma.

In such cases, the injury can be caused by mechanical (shock, bruising), thermal (hot and cold), electric current, chemical, X-rays, and even heat (fear, strong impact) and other changes. 'ladi.

Usually the term trauma or injury is used in a narrow sense to refer to changes that occur mechanically. All changes to mechanical injuries are made by crushing, wounding, sharp, impenetrable, shot bullets, pressure objects.

Stretching, crushing, beating, injuring blood vessels and nerve fibers at the site of mechanical impact. The pathological changes that occur as a result of stretching or traction depend on the strength of the causative agent, the duration of exposure, and the physiological properties and condition of the organ or tissue that is stretched or stretched.

The bones and tendons are also stretched and stretched, and when the muscles contract, they are pulled less than when they are still.

If an organ is strongly pulled and stretched (skin, muscle ligaments, bones, etc.), it is torn and torn. Slow but long and repetitive pulling stretches (e.g. in joints) causes the connecting parts to loosen, causing the joints to play, come out, and so on.

Strong and long-term filling of internal organs (stomach, intestines, bladder). This causes dystrophy of the organ wall and glandular cells.

While changes in organ and tissue compression cause disruption of blood supply, long-term compression of organ or tissue causes tissue nutrition to deteriorate, leading to atrophy and even necrosis.

Strong organ dysfunction occurs when animals are rescued from being trapped underground, resulting in frequent traumatic shock-like disturbances in renal function.

Injuries occur in animals as a result of exposure to cold or firearms, thunder, and air waves. Falling from height or rupture of spleen and blood vessels of deep tissues and organs under the influence of thunder waves is observed fracture of bones without changing the skin lining system.

Traumatic injuries in farm animals (from the coldness of animal caregivers) are caused by the impact of equipment and tools used in various industries (machine mechanisms, washers, dots, etc.).

The following types of traumatic injuries are distinguished:

1. Closed injuries in which the integrity of the skin covering system is not compromised include: compression of the tissue (with tumor, wash, and puncture). Stretching, pulling, breaking, breaking bones, breaking, cracking under the influence of impenetrable weapons.
2. Injuries to the skin lining system, open changes include injury, destruction of the skin lining of the bone, tearing. Depending on the strength of the impact, torn, incised wounds are formed.

One of the characteristic or characteristic changes when an injury occurs is the sensation of pain. The formation of pain is associated with exposure of the organ to extra and introceptors, the breakdown of toxins, tissue breakdown, and the accumulation of toxins of microorganisms in the injured area.

In addition to local changes during injuries, general changes in some organs (heart, respiratory organs, endocrine and external organs) are observed with reflex dysfunction, accompanied by tachycardia, shortness of breath, hyperglycemia, increased blood pressure and other changes. characterized.

Injury to tissues on the surface of the body causes microorganisms to enter the internal parts of the body and cause them to become inflamed. Normally, pathogenic changes are limited due to the activity of protective flexibility mechanisms that protect our body when tissue injury occurs, only in some cases the process is exacerbated by insufficient resistance of the body's protective flexibility mechanisms, leading to the development of pneumonia and then sepsis.

The dead-necrotic tissue in the injured parts forms a large part, and the direct effect of the cause of the injury is due to the wash. The occurrence of such changes is associated with the restoration of tissue nutrition and metabolism by narrowing and rupture of blood vessels, disruption of the integrity of the innervation, and finally compression of the injured tissue and adjacent healthy tissue with exudate.

Long-term purulent wounds are a debilitating weight loss due to the body not healing. Injury weight loss leads to severe damage to internal organs (pleura, lungs, ribs, pelvis and stones). In such cases, the process of tissue regeneration is weakened, atrophy develops in the skin, subcutaneous tissue, transverse skeletal muscles, some internal organs: the animal's appetite is suffocated, sleep is disturbed, liver and intestinal function is impaired, some parts of the bed lie together. becomes lifeless.

Toxins produced by microbes during chronic injuries, the products of tissue breakdown, poison the body and cause it to lose weight. At the same time, many proteins in the pus are released from the body, which weakens the body's resistance to pathogens.

Traumatic shock is one of the most severe pathological conditions of the body.

During a period of traumatic shock, after a short period of agitation, a strong inhibition of the basic physiological functions of the body occurs. Characteristic changes during traumatic shock include acceleration of breathing and pulse, increase in blood pressure, increase in blood glucose and adrenaline. Subsequently, blood pressure decreases, the amount of blood circulating in the blood

vessels decreases, body temperature decreases, reflex activity weakens, the animal becomes insensitive to environmental changes, pain sensitivity decreases, alkaline blood reserve and tissue oxygen consumption decrease. The excitability of the cerebral hemisphere cortex and vegetative centers, the formation of biopathy is weakened. A traumatic shock condition occurs after trauma or exposure to a traumatic agent (primary shock). Primary shock is caused by the reflex excitation of sensory nerve endings under the influence of traumatic factors. The peripheral nerves are irradiated to the subcortical parts, first causing excitation and then braking in the cortex. It weakens all the physiological functions of the body, in particular by lowering vascular tone, leading to a decrease in blood pressure. Many scientists explain the secondary development of shock as poisoning caused by the absorption of histamine-like substances into the body through the blood vessels in the crushed part of the tissues. This is supported by the following supporting information. When histamine and other biologically active substances are released into an animal's bloodstream, a secondary shock-like condition occurs, but histamine and peptone shock, although similar to this shock, do not resemble the shock that results from the injury itself. The formation of traumatic shock is accompanied by additional changes in the body, adverse factors (blood loss, fever or heat, hunger, fatigue), the width of the injured area (nerve columns), due to the abundance of receptors and many other factors. In the development of traumatic shock and subsequent restoration of impaired function occurs the influence of pituitary, adrenal hormones, nervous system and other organs.

The outcome of trauma depends on the type of organ, its vital importance. Death can occur if the heart, large diameter blood vessels, nerve centers, etc. are injured. The changes resulting from the effects of mechanical influences on the nervous system are severe and complex. When peripheral nerves are injured, the motor and sensory properties of organ systems change. Mechanical injury of the central nervous system causes severe functional changes in the body (the affected area depends on the degree of injury). Severe bruising, bullet and skull injuries, causing general bruising, can sometimes injure the brain, blocking blood vessels and the respiratory center. This results in cessation of breathing or paralysis of the heart.

Spinal cord injury paralyzes the leg and impairs the function of pelvic organs (urine, fecal excretion, etc.). Sometimes when a strong blow to the podcherevnoy (abdominal) part, the heartbeat weakens and even stops. Injuries to the heart and large blood vessels are dangerous for the body. When a heart is injured, death usually occurs from exposure to its neuromuscular apparatus, thrombus and blood flow to the heart cavities.

Rupture or injury to the artery of the hip, pelvis, and mesentery results in external and internal rupture, resulting in death. Rupture of the tissues in the chest causes air to enter the interstitial spaces and compress the lungs, leading to disruption of the reflex.

Disease-causing sound wavesdepending on the strength, frequency and duration of exposure to sound waves can have a detrimental effect on the body. Noisy mixtures of different strengths and heights have a detrimental effect on the body. Under the influence of these noises, strong agitation, fatigue, changes in the respiratory process, worsening of hearing, increased intracranial pressure and other pathological changes occur.

Accidental, sudden loud noise can damage the hearing aid: a long and strong generated sound wave can affect the activity of the central nervous system. Pathological changes in the body (metabolic disorders, changes in cell structure, accumulation of heat in the body, when the ultrasound is exposed to a sound that is too long and strong) an increase in glucose and cholesterol in the blood, a change in the shape and structure of the shaped elements of the blood i.e. deformation can cause protein coagulation and other changes).

The causes of internal disease often include the factors that contribute to the onset of the disease in the body. For example, as a result of working in mines, factories, and mines, toxins that enter the body in different ways are absorbed into the tissues, and the dust settles in the lung tissue, causing various deficiencies in these tissues and causing disease. causes. Circulatory disorders are also among the internal causes of the disease. Changes in hereditary traits also cause disease under the influence of mutagenic causes.

Pavlov recommends studying the causes of IP disease in three groups:

1. All exogenous and endogenous causes are the first group of causes to which the body responds with an unconditional reflex.

2. The indifferent effects created by IPPavlov's work, that is, the influence of the causative agent, if supported by normal conditions, then the natural effect of this supporter is called by the disease itself. For example, if you take an apomorphine in a syringe, tie the dog to a machine, and then send the apomorphine to the dog every time it is supported by a light or a bell, then turning on the light bulb will cause illness and the dog will vomit. called syrotchis. The body of animals responds to the causes of this disease by producing conditioned reflexes.

3. Psychogenic causes have also been proven in experiments and are of great importance for human beings, that is, affecting the body by speaking, drawing, grieving, and writing harsh insults can also lead to diseases.

1.Pathogenesis is the study of the origin, mechanism of development, pathogenesis, course, and consequences of diseases.

Greek pathos-victim, genesis-formation. Diseases develop by different mechanisms when different pathological causes affect the body. To make the doctrine of pathogenesis easier to understand, it is distinguished that etiological causes affect 3 different types.

Type 1 causes diseases that affect all stages of development. For example, in acute poisonings, until the toxin is released from the body, it affects the development of the disease in the body, or a similar change occurs when an electric shock.

Type 2 causes serve as a driving force, developing the mechanism of the disease. For example, as a result of a single exposure to hot water, it acts as a starting force. The following substances are formed and poison the body, disrupt the permeability of blood vessels, create an acidic environment and create oxygen deficiency.

Type 3 etiological causes continue to affect themselves depending on the duration of disease development.

The basic structure of the mechanisms of disease development is that when various causes affect the body, there is a lack of oxygen in the body, that is, the metabolism changes, which disrupts the function of various organs and the mechanisms of disease begin to develop.

1. Corticoviceral doctrine is a two-way connection, ie a doctrine that explains that the nervous system is connected to all internal organs. The effect on the body is affected either by a conditioned or unconditioned reflex pathway and responds using unconditioned reflexes. The mechanism of disease development also depends on the reactivity properties of the organism. If reactivity is strongly developed, the disease may not develop. If the body is deficient in various micro and macronutrients, the nutrient content is incomplete, or the body is tired, the development of the disease can occur slowly.

2. Depending on the types of nervous system. If the animals fall into the fragile type, the disease develops more strongly.

3. Explains the development of the disease under the influence of stressors. When inadequate effects on the body are given to the pituitary and adrenal glands over a long period of time, they produce 3 different changes to the effects as they control the body’s reactivity.

1.The properties of tension The pituitary and adrenal glands produce a lot of hormones, adapt to stress by inadequate action, strong excitation, and produce a variety of hormones. If the hormone-producing function either increases or decreases, the body's function is impaired.

2. In the stage of resistance, the body is resistant to any pathogenic influences, because the hormones of the pituitary and adrenal glands increase the energy and plastic mobilization of the body. In the stage of resistance, when the body can not cope with the pathogenic force, the stage of general weakness, without exhaustion begins.

3. At the stage of general weakness, the body loses flexibility, immunological reactions, regeneration state decreases.

3. Examination of cell composition in animals and humans revealed that the development of pathological processes depends on chromosomes: for example, defects in the development of sex, ie

secondary sexual characteristics, infertility and other changes. Males have one more sex chromosome and females have one less sex chromosome.

The role of constitution in pathogenesis. The disease arises from the encounter of disease-causing causes with the organism. Therefore, in addition to qualitative and quantitative changes in the pathogen, the characteristics of the animal organism are important in the origin of the disease. The individual reactivity of the organism takes the first place in the origin of diseases in the organism, because the effect of a certain pathogen on the organism of animals does not lead to the disease of all animals, but to some of them.

What is the constitution? Although there is still no complete answer to the question, constitution refers to the general morphological and physiological features of an organism, which are the product of long-term evolution from the interaction of the organism with the external environment, and these properties are stable. Due to these features, the reaction of the organism to the external environment is determined, comparing close species.

The constitution of agricultural animals means that it increases the resilience, resilience, disease resistance, flexibility and productivity of the farm and the environment. Thus, the constitution of farm animals means not only the morphological and physiological characteristics of the organism, but also the reactivity of the organism to the external environment, including the development of a response to the causes of the disease.

The whole organism can be afflicted with various diseases, and it is impossible to know in advance for what reasons they occur. It depends on external influences, hunger, poisoning, fatigue, exposure to cold and other causes that change resistance and their effects. Due to congenital malformations of the organs in some organisms, the influence of the above external causes causes the disease. In recent times, it has become common to study the constitution in two parts:

1. The constitution of the breath.
2. The constitution of digestion.

Importance of breed, sex and age in pathogenesis. Animal breeds play an important role in the origin of the disease, and Algerian sheep do not suffer from anthrax. Horses of the Budyonny breed are not susceptible to lung diseases. Caucasian mountain merinos do not suffer from pyrapylazmosis, but other breeds are highly susceptible to the cause of this disease. Depending on age, young animals suffer from diseases of the digestive organs, pneumonia, some infectious diseases. As the animals mature, many diseases become more resistant.

4. Restoration of body activity. Protective resilience mechanisms in the body that have the ability to restore impaired function under the influence of pathogenic influences, including excess energy generated in the body, surfaces, stored blood, chemicals and biochemicals. For example: under normal physiological conditions, 17-20% of the heart muscle, the respiratory surface of the lungs, the absorption surface of the intestine, 20-25% of the glomeruli of the kidneys, 12-15% of the liver, 10-15% of the blood vessels, 50 of hemoglobin -60% and nervous, endocrine systems are rarely used. Therefore, the organism adapts to any difficult conditions. For example: in bilateral pneumonia, dystrophy and fatty heart muscle, severe liver injury, removal of a single kidney, functions are also compensated when a large part of the stomach and intestines are cut, when a lot of blood is lost, when many capillaries become loose and clogged, and when nerves and endocrine glands are injured. The patient's kidney function is performed by a healthy kidney, and lymph nodes perform blood formation when the spleen is removed or diseased.

Control questions.

1. What is the etiology?
2. What theories do you know that explain etiology?
3. What are the types of etiological factors?
4. What is pathogenesis?
5. Explain the mechanism of action of etiological causes that explain the pathogenesis?

ORGANIZATIONAL REACTIVITY AND RESISTANCE.

Plan:

1. The concept of reactivity and resistance of the organism.
2. Types of reactivity and resistance. Forms of manifestation of the organism's reactivity and factors influencing it.
3. The role of nervous and endocrine systems in reactivity..

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Basic expressions

Reactivity, resistance, immunity, NNSirotinin, R.Virkhov, IIMechnikov, VVPashutin, AABogomolets, IPPavlov, nerve, humoral, ADSperanskiy, AAUkhtomskiy, AMMonaenkov, NEVvedinsky, IMSechenov, heat, cold, physiology, heat, cold, poison, , hereditary, AMBezredko, barrier, phagocytosis, immunantana, full-value, worthless, macro-and micronutrients.

Organisms have developed resistance mechanisms to exposure to the external environment at different times. First of all, the general reactivity in the body, that is, the resistance to various toxins, and then the types of immunological reactivity developed. As organisms now develop, the reactive function is performed by cells, which later develop a response using the humoral system and eventually the nervous system.

The properties of reactivity depend on the age of the animal, the nervous and humoral systems, the external environment and the general condition of the organism. For example, when the embryo develops in the mother's womb, it responds to the stimuli through the mother's body, ie through the placenta. When a baby is born, its reactivity is weak and responds only by a phagocytic reaction or by immune cells that pass through the mother’s blood. That is why young animals often get sick and die. Young animals are weakly adaptable to changes in ambient temperature, and their dyspepsia, salmonellosis, colibacillosis, rickets and other diseases are common. Reactivity in adult animals is manifested in the fight against microorganisms by antibodies, phagocytes and macrocytes that have accumulated in their bodies. As the body ages, its reactivity decreases. phagocytes, immune cells are reduced, and the incidence of disease increases with susceptibility to disease. As a result, tumors, hypertension increase, regeneration is weakened, and the body's reactivity is low, so they have severe infectious diseases.

Sirotin NN and other scientists note that the cerebral cortex of cold-blooded and young animals is poorly developed and is less sensitive to strong toxins (histamine, diphtheria, stolbyank toxin). During

anabiosis, animals do not develop sensitivity to very strong toxins and infectious agents (plague, tuleremia, anthrax, tuberculosis).

Due to reactivity, the body responds to disease-causing causes, and the sensitivity of different individuals to infectious agents varies. Such cases can be observed in various pathological processes. For example, when an animal with a high reactivity burns, it recovers quickly and an animal with a low reactivity recovers later. The reactivity of the animal organism depends on the metabolism, the immunological properties of the organism, the functional state of the animal organism, the vascular reaction and chronaxy to the excitability of the nervous system.

Concepts of reactivity R Virkhov's cellular theory developed at a time when the theory of cells gave a misunderstanding of the general reactivity properties of individual cells, tissues and organs, ie the fact that pathological processes take place only in cells. 'did not notice. In contrast, IIMechinkov in his many years of observations shows that the reactivity of organisms at different stages of evolutionary development is also formed under the influence of disease-causing factors of the external environment. As organisms become more complex and the nervous system develops, the body's reactivity to inflammatory agents becomes more complex. For example: cold-blooded frogs, inflammation in fish, develops very poorly in warm-blooded animals. Even when these properties were observed by NNSirotinin sending proteins to the body, it was observed that the body of cold-blooded animals produced very weak responses. Gradually, as a result of the development of the nervous system of the organism, the reactivity or sensitivity of the organism to many toxins, formed a changing response.

Reactivity is a characteristic feature of all animals, and in the field of reactivity IIMechnikov, VVPashutin, AABogomolets, NNSirotinins have done a lot of research. In their laboratories, these scientists studied reactivity by linking it to metabolism and other areas. IPPavlov and IMSechenov confirmed that the nervous system plays a leading role in the development of reactivity. In the IPPavlov laboratory, MKPetrova et al observed that the reactivity of animals was impaired by inhibiting the cerebral cortex by giving bromine preparations.

The importance of the types of nervous system in reactivity is also great. To study the importance of types of nervous system in reactivity, they took two groups of dogs:

1. The group includes dogs with a weak nervous system.
2. Dogs with a strong type nervous system in the group.

In animals of both groups, when exposed to strong toxins, cyanic acid, bacterial toxins, dogs with a weak nervous system became ill due to weak barrier properties of the organism, in animals with a strong nervous system AMMonaenkov and others explain that the diseases have not developed because their barriers are strong, their neutralizing properties are high.

In the IPPavlov laboratory, pigeons became infected with anthrax when a certain part of their brain was removed.

Academician ADSperansky observed that when dogs opened their brains and placed a ball in the midbrain, mechanical effects resulted in ulcers in the lungs and digestive systems, weakening their resistance to infection. He drew attention to the fact that the traces of the nervous system in the origin and development of pathological processes, that is, pathological processes in the nervous system, even after their recovery, retain their complications for a long time. In many experiments, that is, when animals are exposed to different stimuli after treatment of the disease, the effect of these stimuli spreads to the entire nervous system, leaving traces of old disease in the affected area. observed that it had survived and accumulated, leading to the onset of the disease. This feature of the nervous system is called AA

Reactivity is also affected by the autonomic nervous system. Reactivity changes when the function of the autonomic nervous system increases or slows down. Excitation of the sympathetic nervous system enhances phagocytosis, enhances metabolism, and increases reactivity. Excitation of the parasympathetic nervous system increases the production of antibodies, produces short-term leukocytosis, followed by leukopenia, exposure to certain toxins (phenol, aniline, etc.), lymph nodes, liver barrier - barrier properties increases.

Reflexivity changes reflexively from the pathological effects of heat and cold. For example, as a result of colds, people get the flu, pneumonia, that is, the body's reactivity decreases. In experiments,

it is possible to cool the body of chickens, reduce their reactivity and lead to anthrax, or to heat the body of guinea pigs and reduce their sensitivity to proteins.

Toxic substances, alcohol, carbon monoxide, lead, mercury, cyanic acid weaken the internal braking. Pigeons were poisoned with alcohol, which reduced their reactivity to anthrax, or when people consumed alcohol for a long time, they observed a decrease in the general reactivity of the organism, and xko.

While ultraviolet light from light energy increases the stability of an organism to a certain extent, it weakens the stability of an organism to a certain extent. X-rays and gamma rays have a detrimental effect on the body's reactivity. The reactivity of the organism also decreases under the influence of mechanical influences. Thus, the role of nervous endocrine systems in the formation of reactivity of the organism is important, but different effects of the external environment affect the activity of various organ systems of the organism, affecting their metabolism, neurohumoral control mechanisms.

There are several classifications of reactivity, and most scientists classify the organism according to its state of health or disease:

1. Physiological reactivity.
2. Pathological reactivity.

Physiological and pathological reactivity can be individual or individual, as well as group. Individual or specific reactivity depends on hereditary traits and can be passed down from generation to generation. Physiological reactivity develops the body's response to natural (adequate) influences, while pathological reactivity develops the body's response to the causes of the disease. Allergic and immunological types of pathological reactivity are distinguished, and the manifestation of these types of reactivity is formed in relation to foreign proteins, microbes and their toxins. (Allergy, Anaphylaxis, Immunity). Typically, biological or species reactivity is differentiated and is specific to animals belonging to a particular species, ranging from seasonal changes in animals to: seasonal sleep, migration of animals from one place to another, animals are not exposed to microorganisms, ie chickens are not infected with anthrax, specific reactivity is a characteristic feature of a particular individual, it depends on the constitution, sex, age, nutrition and storage characteristics, newborn reactivity in animals is low, reactivity is well developed during sexual maturation, phagocytosis and the formation of immunoassays are well demonstrated, in older animals the reactivity of the organism is low due to the weakening of their barrier properties. Hence, the specific reactivity is that during the period of complete vaccination of animals, their reactivity is formed differently, with strong antibodies in some and weak antibodies in others.

The resistance of an organism, as the Latin resisteo (resist, resist), is the resistance of an organism to physical, chemical, and biological causes of disease. This means that the body's resistance is understood to be resistance to many different causes.

During phylogenetic development, when the resistance of the organism changes and invertebrates are resistant to bacterial toxins, the susceptibility of warm-blooded animals is high. Resistance is associated with the functioning of organ systems, depending on the type, sex, age, constitution, anatomical and physiological characteristics of the animal, the level of development of the organism, the development of the RES and lymphoid system. In the early stages of ontogenetic development of animals, resistance to various harmful agents is high (partial pressure reduction, some bacterial toxins), resistance to sexual development is well developed, and resistance decreases with age.

Resistance:

1. Natural-born,
2. Acquired-generated species are different.

Congenital resistance is passed down from generation to generation. For example, Algerian sheep are more resistant to anthrax than European sheep.

Acquired generated resistance depends on the individual characteristics of the organism and is formed when immunized against infectious diseases. Resistance is formed depending on the activity of the pituitary, adrenal glands, colon, gonads. Barrier properties of the organism, biologically active substances in the blood and phagocytosis play a key role in resistance. When the body is tired, very

productive, living conditions are poor, resistance is weakened, and conditions are created for the development of diseases.

2. Animals and humans live in a world of microorganisms. Immunity, on the other hand, as a controller, rigorously tests agents for various causes that have entered the body.

Immunity - Latin Immunitas - means purification, deliverance. Immunity is the ability of an organism to be exposed to antigenic pathogens, their products and hereditary foreign substances, or to be resistant to various disease-causing microorganisms, viruses and their products, as well as to non-infectious modes. , forms a special view of the overall resistance.

Immunity is divided into two depending on the nature of the mechanism and causes that cause it:

1. Congenital immunity or hereditary immunity from generation to generation.
2. Acquired immunity

Congenital or natural species-specific immunity is a specific resistance of an organism that is passed from generation to generation and is specific to a species, breed, and population. For example, in cattle, horses are resistant to microorganisms that cause croupous inflammation of the lungs, and animals are highly resistant to human diarrhea. Dogs are not infected with pleural pneumonia in cattle. Cattle do not suffer from horse manure, infectious (infectious) anemia.

Inter-species immunity is also different, Algerian sheep are resistant to anthrax, Breton sheep are resistant to smallpox, light-bodied pigs are resistant to yellow fever, Mongolian cattle are resistant to plague, and other animals of this type are infected with the above diseases. Congenital immunity is formed not only against an infectious agent, but also against their toxins. The barrier properties of animals with innate immunity are strong and do not transmit microorganisms into the body or prevent the growth of microorganisms by altering the environment.

These organisms have high phagocytic activity and bactericidal properties in fluids, which prevents the development of microorganisms and forms specific immune cells against these microorganisms.

Acquired immunity is formed during the ontogenetic development of certain microorganisms in the body of animals. Acquired immunity is created by natural and artificial means. For example, naturally acquired immunity is formed after recovery from mango, smallpox, proteinuria and other diseases. Artificial active immunity is created by vaccinating animals against various infectious diseases. Hence, acquired immunity is generated by natural and artificial means.

Artificial immunity is studied as active and passive immunity. Passive immunity is formed when hyperimmune serums are sent, through the passage of immunoassays through milk, through the placenta. Due to passive immunity, the body's resistance is maintained for some time. RES plays a leading role in the formation of immunity, and the formation and formation of immunity is controlled by the nervous system.

During the period of immunity against infectious diseases, if the organism is completely cleansed of infectious agents, sterile immunity is formed and the organism is provided with sterility to this antigen. If the immunity formed in the body does not maintain complete sterility, and the antigen is retained in the body, it is called nosteril immunity, which is characteristic of tuberculosis and brucellosis.

Immunity can be formed not only against microorganisms themselves, but also against their toxins, which is called antitoxic immunity and is observed during exotoxin-producing microorganisms: tetanus, botulism, gas gangrene and other infections. Hence, toxins act as antigens in this process.

In addition, the body has special organs and factors that fight microbes and foreign substances, which are called barrier properties of the organism. The barrier-barrier properties of the organism are studied as external and internal barriers.

External barriers of the body include the skin and its products (accumulations), mucous membranes in various parts, the oscillating epithelium of the respiratory tract, microorganisms of the digestive system and hydrochloric acid.

Control questions

1. What is reactivity?
2. Explain the role of the nervous system in reactivity?
3. Explain the role of VNS in reactivity?

4.Explain the role of high nerve activity in reactivity?
5. Explain the role of endocrine glands in reactivity?
6.Explain the effect of the external environment on reactivity?

ALLERGY AND ALLERGIC DISEASES

1. Allergy, causes, types and mechanism of development.
2. Allergens and their types.
3. Allergic diseases and their types. The role of allergic reactions in the diagnosis of infectious diseases.

Main publications:

1. Khaitov RX, Eshimov DE "Pathological physiology of animals" Textbook Tashkent, "Ilim Ziyo" 2013.
2. M.Donald., James F. “Pathologic basis of veterinary disese” Humana Press; 2011 edition.
3. S.I.Lyutinskiy. Pathological physiology of agricultural animals. Moscow, Kolos, 2001.

Additional literature

1. DEEshimov., RFRuzikulov. "Practical and laboratory classes in animal physiology and pathophysiology." Study guide. Tashkent. Tafakkur Bostoni - 2011.

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5. www.kodges.ru

The body's internal barriers include a number of cellular and humoral factors, various histiocytes, reticular cells, plasma cells, epithelial cells of the inner wall of blood vessels, and leukocytes. RES cells, which are involved in protecting the body, are active, they absorb microbes and other particles that enter the body, they are very rich in RES in the lymph nodes, spleen, liver, lungs, kidneys, meninges, blood-forming organs, skin . This means that RES is present to one degree or another in various organs of the body, and phagocytic activity is much higher in leukocytes, including neutrophils. In his long-term observations, IIMechnikov argued that the process of phagocytosis plays an important role in the formation of immunological features. microbes and their toxins, cellular elements, tissue breakdown products, other particles are digested in cells. Phagocytosis is the process by which particles are trapped in a cell and then digested. Phagocytosis is common in nature, with feeding and protection of single and multicellular simple animals occurring in a single cell, while in highly developed animals these systems are isolated and protected by specific mesenchymal cells (blood leukocytes, lymph nodes, red blood cells). bone marrow, spleen, liver, connective tissue histiocytes) - by phagocytes. Studies have shown that there is a direct link between the process of

phagocytosis and the resistance of the organism. increased phagocytosis indicates a weakened immunity in the body. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens.

1. Antitoxins and antifenzymes, immune cells that inactivate by binding toxins and enzymes.
2. Agglutinin and persipitins, antibodies that change the colloidal chemical structure of microorganisms, immobilize them, bind them to the sediment.
3. Cytolysins or cytotoxins - antibodies that break down cells under the influence of enzymatic complement substances.
4. Opsonins and bacteriotropins - change the appearance of microorganisms, facilitating phagocytosis.

If antibodies are formed under the influence of antigens, what are the antigens themselves?

Antigens are substances that enhance the formation of immune bodies and react selectively with them. These include microbes, toxins, erythrocytes and serum of other animals, as well as high-molecular compounds.

There are two types of antigens.

1. Full value antigens.
2. Incomplete antigens - haptens.

Complete antigens include complete proteins, ie serum, various proteins, microorganism toxins and filtrate colonies. Antigens have specific properties that react with the antibodies they produce.

Incomplete antigens, ie haptens, cannot enter the body to form antibodies and only bind to the protein molecule to achieve antigenic properties.

Antigens must be administered parenterally to the body to form immune cells. Antigens are exogenous and endogenous substances that are foreign to the body. The body's own proteins also sometimes exhibit antigenic properties. To do this, the body's proteins meet with the infectious agent, toxins, and form an autoantigen. In order to form immune cells against antigens, the antigen remains in the body for a certain period of time, is captured in the liver, spleen, lymph nodes and stored in the blood for 2-3 weeks. Immunological reactivity is formed not only from the encounter of macro and micro organisms, but also from other types of individuals and even in the same organism itself when tumors grow, become inflamed and in other cases have antigenic properties against their own organism. In all cases, there are antigen and antibody reactions and phagocytosis between body tissues and other tissues. The tissue formed during embryonic development serves as an antigen for older tissues. Tissue does not fit the transplanted tissue or organ due to the immune barrier property of these organisms when transplanting organs into one species or individual, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by.

Decreased or complete loss of antibody production as a result of exposure of antigens to the body is called immunological tolerance or non-response. This condition is caused by antigen transmission during the embryonic period or after the animal is born. In older animals, immunological tolerance can be established by transferring large amounts of antigen or exposing them to X-rays. Immunological tolerance is characterized by the loss of these antigens of their antigenic properties, which is observed when transplanted into other animal tissues, and the transplant grows well. It is currently used in blood transplants to remove tissue barriers from immunological tolerance.

Control questions

1. What is immunity?
2. What types of immunity do you know?
3. On the basis of what theories do you explain the formation of immunity?
4. What is an antigen?
5. What types of antigens do you know?
6. What is an antibody?

Inflammation

Plan:

1. The concept of inflammation. Etiology and external signs of inflammation. The main stages of inflammation: alteration, exudation and proliferation.

2. Circulatory disorders in inflammation. Classification of inflammation. Exudate, its types and properties.

3. The role of nervous, endocrine and immune systems in inflammation. Consequences of inflammation and its importance for the body.

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BASIC EXPRESSIONS.

Exogenous, endogenous causes, mechanical, physical, chemical, biological, necrotic tissue, infarction, salt accumulation, thaw, galenic, redness, swelling, local temperature rise, pain, dysfunction, alteration, dystrophy, phagocytosis, proliferation, exudation, emigration, protein, fat, carbohydrate, osmotic, oncotic pressure, vascular reaction loss of vascular tone, unevenness of the inner surface of the vessel, transudate, mechanical, biological, chemical theory, macrophage, granulison tissue , complete incomplete healing, transition to a chronic state, exudates. Serous, serous catarrhal, fibrinous, purulent, bloody or hemorrhagic, purulent or esophageal, normiergic, hyperarrgic, hepatic, reactivity, autoantigen, autoantetela, R. Virkhov, congeym, Ricker, IIMechnikov, Shade, Samuel, V.Ya. Danelevskiy, IP Pavlov, NI Simonovskaya, AD Speranskiy,

Inflammation is the most common, most complex pathological process known since ancient times, and in ancient times all diseases accompanied by a rise in local temperature were called inflammation. Inflammation is a typical pathological change (disruption of tissue function and changes in structure) that is common in various diseases, as well as the activation of the body's protective resilience properties and the restoration of impaired function. Although inflammation in this area delays the organism as a process with protective properties against the effects, the mechanism of its development, the formation of symptoms depends on the state of the organism, the activity of neuro-humoral systems. For example: Inflammation of the skin can be caused by affecting some endocrine glands of the gillpotalamus or peripheral nerves. Glandular is a local manifestation of the general reactivity of the organism, the degree of reactivity of the organism depends on the course of inflammation and, conversely, on the reactivity of the organism to inflammation, neurohumoral control, thermoregulation and other mechanisms. All substances that cause inflammation are called phylogenetic substances, and we study them in two groups, namely, exogenous and endogenous substances. Inflammation occurs under the influence of phylogenetic substances, and the name of the inflamed organ or tissue is read by adding the suffix "IT", "IYA". For example. Inflammation of the liver is called hepatitis, inflammation of the kidneys is called nephritis, inflammation of the lungs is called pneumonia, and xzo

Inflammation is caused by mechanical, physical, chemical, and biological causes of external disease, and often the contribution of microorganisms and viruses is important in causing inflammation.

Sometimes inflammation can also be generated under the influence of conditioned indeferent stimuli.

Ichki yalligg‘lanish chaqiruvchi sabablarga nekrotik to‘qima, infarkt, gematoma, turli qismlarda to‘plangan tuzlar kiradi. Yallig‘lanish chaqiruvchi sabab, ko‘pincha yallig‘lanish reaksiyalarini hosil bo‘lish intensivligini belgilab beradi: Masalan. Rengen nuri, zaharli modda, mexanik jarohatlar, kuyish,sovuq urish va boshqalar oldin to‘qimalarni parchalab, keyin shu joyda fiziologik aktiv moddalar to‘planib, ular ishtirokida yallig‘lanish jarayonlari ro‘yobga chiqa boshlaydi. Surunkali

kechuvchi kasalliklarda , kasallik chaqiruvchi sababni, begona tasirotchini uzoq vaqt tasiridan, yoki ximiyaviy qo‘zg‘atuvchining tasiridan proliferativ jarayonlar kuchayaadi.

Yallig‘lanishni kechishi kasallik chaqiruvchi sabab tushgan joyga bog‘liq bo‘lib, amyoba jigarga tushib absets chaqirsa, ichaklarda yarali yallig‘lanish chaqiradi. Masalan. Stafilokok, streptokoklarni yiringli infeksion jarayon hosil qilish aniq, lekin skipidarlarni teritagiga yoki muskullar orasiga yuborib yiringli yallig‘lanish chaqirish mumkin. Shunday qilib yallig‘lanishni xususiyati, uni hosil bo‘lish tezligini qo‘zg‘atuvchi xususiyatiga hamda yallig‘lanish kechayotgan muhitga bog‘liq ekan. Yallig‘lanishning tashqi mahalliy belgilari Sels va Galenlar tomonidan sharxlangan bo‘lib: qizarish-chivoch, shish tishoch, harorat ko‘tarilishi-saloch og‘riq - doloch, funksiyani buzilishi fipstto laesa deyiladi. Har qanday yallig‘lanish ham bir qancha asosiy bir-biri bilan bog‘liq jarayon bilan kechadi: altteratsiya-to‘qimalardagi distrofik o‘zgarishlar-to‘qimalarning yallig‘lanish chaqiruvchi agent ta’sirida qitiqlanishi va parchalanishi, maxalliy qon aylanishini buzilishi-ekssudatsiya va emigratsiya, fagotsitoz hamda proliferativ o‘zgarishlar. Yallig‘lanish chaqiruvchi agent to‘qimalarni qitiqlashi, parchalashi, ulardagi moddalar almashinuvini, tuzilish va funksiyani buzilishiga sabab bo‘ladi. Distrofik o‘zgarishlar yallig‘lanish chaqiruvchi sabab ta’sir etgan vaqtdan hosil bo‘lib, kam chegaralangan bo‘ladi. Keyinchalik ta’sirotchining ta’siri kuchayishi bilan yallig‘lanish kuchayadi, to‘qimalarda moddalar almashinuvi kuchayadi, qon aylanishi buzilib, distrofik o‘zgarish kuchayadi. Kasallik chaqiruvchi sabab organizmga tushib birinchi navbatda retseptorlarga tasir qiladi. Agar ta’sirotchi kuchi etarli bo‘lsa nerv oxirlarida parabioz xolatini hosil qiladi.

At the onset of inflammation, the tissue bends the cells, fat granules appear, protein and fat dystrophies are observed, then the cell structure is disrupted and even severely damaged and dies. Necrabiotic processes during inflammation are caused by the bending and melting of collagen and elastic fibers of tissue interstitials. In inflammation, necrobiotic processes are formed when tissue burns, under the influence of strong acids and alkalis, sometimes in relation to weak influences from increased sensitivity of the organism. There is a certain association between them and dystrophic changes in the body, and sometimes due to the injured part there is a compensatory restoration of their functions, despite the presence of destructive changes in the salivary glands, stomach and other organs. ladi. The development of destructive changes during the period of inflammation depends on the organ, and such changes can be observed in injuries of parinchyomous organs. The degree of dystrophic changes depends on the strength and nature of the pathogen, where the pathogen enters, the nature of the injured organ or tissue, and the reactivity of the organism. Physiologically active substances formed as a result of dystrophic changes in the source of inflammation and metabolic disorders are absorbed into the blood, reducing vascular tone, causing emigration, phagocytosis and proliferation of cellular elements. These biologically active substances include histamine and histamine-like substances, acetylcholine, ATF, creatine phosphoric acid and other necrogorms that dilate blood vessels and enhance proliferation, trephon tissue proteases and cathepsins. Thus, the strong passage of alternative, proliferative and exudative processes during the inflammatory period leads to tissue bending and the development of dystrophic changes that complicate blood circulation.

Metabolism at the source of inflammation undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. Metabolism between the inflamed part and healthy tissue is enhanced. The increase in metabolism is due to easily oxidized carbohydrates, which form many weak acids as they take place in an oxygen-free environment. The breakdown of carbohydrates in the anaerobic phase increases due to leukocytes released during emigration, but these changes can be seen in the oxygen consumed and the carbon dioxide excreted before the breakdown is broken down into the final product. During this process, the respiration rate decreases as more carbonic acid is released.

During inflammation, the metabolism undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. The metabolism between the inflamed part and the healthy tissue becomes enhanced. Metabolism will be enhanced. Lactic acids are formed due to the fact that the increase in metabolism is due to easily oxidized carbohydrates, which take place in an oxygen-free environment. Due to the leukocytes released during emigration, the breakdown of carbohydrates in the anaerobic phase

increases, but without decomposition to the final product, these changes can be determined by the oxygen consumed and the carbonic acid released. In this process, the respiration rate decreases as more carbonic acid is released.

Fats and proteins also form ketone bodies, albumin-peptones, which are not completely broken down in the center of inflammation. Excessive increase in carbohydrate protein and fat metabolism, complete oxidation of milk at the source of inflammation, pyruvic acid, fatty acids lead to an increase in ketone bodies, amino acids and peptones, and acidosis develops. Acedosis is compensated first at the expense of the body's alkaline reserve, then it is not compensated.

(N hyperonia is formed). Depending on the nature of the process taking place in the tissue, the change in the environment of the tissue becomes 7.1-6.6, ie weakly alkaline, in the acute process 6.5-5.4 in the acute flow process. Increased acidosis increases the dissociation of salts, changes the electrolyte ratio, increases the amount of potassium, increases metabolism, breaks down large molecules into small molecules, increases the amount of ions, increases the osmotic pressure at the source of inflammation. Similarly, oncotic pressure increases. Osmotic and oncotic pressure decrease as you move away from the source of inflammation. Thus, changes in the quality and quantity of tissues during inflammation cause physicochemical changes in tissues, including: hyperionia, hyperosmia and hyperonkia. The causative agent causes a short-term narrowing of the blood vessels by reflex action on the blood vessels and then dilation of the blood vessels.

The slowing of blood flow in the blood vessels is due to the following reasons:

1. Paralysis of the vascular neuromuscular apparatus causes loss of vascular tone.
2. Causes excessive dilation of the vascular surface.
3. It causes the blood to thicken and become sticky.
4. Slows down blood flow as a result of cutting blood vessels with fluids in the surrounding tissues.
5. Due to the adhesion of leukocytes to the inner wall of blood vessels, the unevenness of the inner surface of blood vessels is formed, and sometimes clogging with thrombi leads to a slowing of blood flow.

The vascular response at the source of inflammation varies under the influence of various pathogens. For example: vasoconstrictor (adrenaline caffeine, etc.) and vasoconstrictor sympathetic nerve effect. Slowing of blood circulation changes until complete cessation of blood flow in the arteries, leading to changes similar to thrombosis and hemorrhage. Disruption of blood circulation at the source of inflammation worsens metabolism, disrupts the nutrition of cells in the inflammatory center, and these changes themselves lead to increased inflammation.

Dilation of blood vessels and slowing of blood flow increase the permeability of blood vessels, resulting in leakage of shaped elements with liquid parts of the blood, and this process is called exudation. The fluid released is called exudate. The exudate differs from the transudate in the presence of 2-4 times the protein, shaped elements, local tissue elements, tissue breakdown products, some enzymes and other products. The process of exudation depends on several factors, the main of which are capillary permeability, high blood pressure in the vessels, osmotic and oncotic pressure at the source of inflammation.

Capillary permeability depends on the physiologically active substances histamine, bradykinin, serotonin, as well as potassium and hydrogen ions accumulated at the source of inflammation, which ions swell the blood vessel wall, dilute colloidal substances and disrupt vascular nutrition.

Healthy capillaries pass water and crystolloids, increasing permeability from colloidal substances to proteins primarily albumins (low molecular weight) substances.

In inflammation, more blood flows to the source of inflammation, weakening the bleeding and increasing the pressure in the blood vessels, which allows more fluid to leak out of the blood vessels. Such strong exudation lowers blood pressure in the blood vessels and weakens blood flow. Exudation is also affected by the osmotic and oncotic pressure at the source of inflammation.

During exudation, water, salt, protein, or cell-free products are released from the blood vessels, and then leukocytes are released from the blood vessels into the tissues, called leukocyte emigration. During leukocyte emigration, the localization of leukocytes along the walls of blood vessels occurs, resulting in the redistribution of blood-forming elements, which is associated with slowing of blood

flow. In normal life processes, the blood is characterized by the placement of two layers of thin, plasma at the edges of the blood vessels and shaped elements moving in the center, the specific gravity of erythrocytes is heavy between the blood vessels, leukocytes move lightly on the periphery.

As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cytoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Remaining leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that die in large numbers in high osmotic pressure and atsedosis.

Monocytes show their resistance even at pH 5.5. While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. AS Shklyarevisky, a proponent of the mechanical theory that explains the location of leukocytes along blood vessels, explains that leukocytes are pushed aside by other shaped elements because of their light weight.

Proponents of the second type of this theory explain that leukocyte emigration is a passive process in which leukocytes flow out of the general fluid flow and remain outside the blood vessels. If this is the case, then why do neutrophils come out in one case, lymphocytes and monocytes in the other. Thus, without mechanical factors playing a major role in the location of leukocytes along the vessel wall, this theory cannot explain the formation of these processes. Because the location of leukocytes along the walls of blood vessels is a complex biological process, the active processes in which leukocytes approach the wall of blood vessels, push it out of the blood vessels and participate in phagocytosis.

According to IIMechnekov's biological theory, leukocyte emigration is called a positive hemataxis feature. Positive chemotaxis properties include staphylococcus, streptococcus and other substances that are formed as a result of their activity, as well as products of nucleic metabolism, some globulins, liver and kidney proteins, meat peptone broth, some medicinal substances.

The repulsion of leukocytes from these chemicals is called negative chimataxis, and the negative chymataxis property is characteristic of quinine, chlorochrome, benzene, alcohols.

The development of physkaloid chemistry leads to the emergence of a new direction that explains the emigration of leukocytes, i.e. leukocyte emigration is associated with physicochemical changes in tissues.

Increased metabolism in the inflammatory center results in the formation of completely unoxidized substances, leading to an increase in N ions. Thus, due to different charges, negatively charged leukocytes move towards the center of positively charged inflammation. Leukocyte emigration is also caused by the continuous release of fluid from the blood vessels into the inflamed parts. Energy processes in leukocytes also play an important role in leukocyte emigration. On the side of leukocytes facing the source of inflammation, the protoplasm melts to form pseudopodia and amoeba-like action due to the energy generated during the metabolism of leukocytes. Emigrated leukocytes partially die under the influence of the environment at the source of inflammation, while others are actively

involved in the process of phagocytosis. While the process of phagocytosis is influenced by the tissue environment and physiologically active substances, the acidic environment and alkaline environment inhibit the process of phagocytosis, while the normalization of the environment ensures the normal course of phagocytosis. Thus, leukocyte emigration is an active biological process in which mechanical and physicochemical changes play an important role.

Proliferatsiya jarayoni yallig‘lanishning barcha davrlarida hosil bo‘lib, alteratsiya kechayotgan davrda kam miqdorda bo‘lsada to‘qima hujayralari ko‘payib o‘zining eng kuchli ko‘payish davriga yallig‘lanishning oxirgi davrlarida etiladi. To‘qima hujayralarni ko‘payishini kuchayishini parchalangan mahsulotlar va to‘qimalarda moddalar almashinuvini buzilishidan hosil bo‘lgan moddalar hamda patogen agentining o‘zining ta’siridan hosil bo‘ladi. To‘qima va hujayralarni tiklanishida yallig‘lanish markazidagi RES hujayralari ya’ni qon tomirlar endoteliyasi, advintitsiyasi, fibroblastlar, gistiositlar, fibrotsitlar va qon tomirlari orqali emigratsiyalangan monotsitlar ishtirok etadi. Hujayra elementlari harakatchan bo‘lib fagotsitoz jarayonida ishtirok etadi. Bularni makrafaglar deyilib, ularga Ranve plazmatsitlari, poliblastlar, Maksmovning tinchlikdagi adashgan hujayralari, turli gistiotsitlar kiradi. Yallig‘lanish manbaida hosil qiluvchi plazmatik hujayralarni parchalanish mahsulotlarini fermentativ yo‘l bilan emiradi.

After the process of proliferation, the process of regeneration develops, the growth of connective tissue, blood vessels, connective tissue proliferates and glandular cells are regenerated. Young fast-growing connective tissue is rich in blood vessels and is called granulation tissue. The connective tissue grows from the periphery to the center, creating a barrier between healthy tissue and inflamed tissue, preventing microorganisms from spreading from the source of inflammation to the body. Upon completion of the inflammation, interstitial fibrous substances are formed in the granulated tissue, the blood vessels shrink, the young mesenchymal cells stop growing, and eventually a dense connective tissue chandelier is formed. The resulting scars cause various dysfunctions, including esophagus, stomach, if it is formed in the urinary tract, it causes them to narrow, the mobility of the joints changes, and so on. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little sluggishly. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little

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Yallig‘lanish morfologik va etiologik belgilariga qarab bir necha turlarga bo‘linadi. Yallig‘lanishning morfologik belgisiga karab alterativ, ekssudativ va proliferativ xillarga bo‘linadi.

Alterativ yallig‘lanish davrida to‘qimalarda distrofik va nekrobiotik jarayonlar, ekssudatsiya va proliferatsiya jarayonlariga nisbatan kuchli rivojlanib bu turdagi yallig‘lanishlarni turli zaharli moddalardan bakteriya toksinlari, ba’zi bir tuzlar ta’sirida parenximotoz organlardan buyrakda, jigarda, yurak va kam xollarda miyada uchraydi.

Ekssudativ yallig‘lanishda ekssudatsiya va emigratsiya jarayonlari boshqa jarayonlardan ustun turib, ekssudat turiga bog‘liq holda serroz-zardobli, kataral-shilliqli, fibrinli, yiringli, ixoroz yallig‘lanishlar farq qilinadi.

Seroz yallig‘lanishlarda suyuqlik tiniq, sarg‘imtir rangli, solishtirma og‘irligi 1,018-1,-20 tarkibida 5-6% oqsil va kam miqdorda shaklli elementlar saqlaydi. Qon tomirlar reaksiyasi to‘liq rivojlanmay to‘qima kam parchalanib ekssudat tez so‘rilib faqat plevra va qorin bo‘shlig‘ini yallig‘lanishi bir muncha qiyin kechadi.

Catarrhal inflammation is a mixture of serum and mucous substances, which is more pronounced at the level of the mucous membranes, and leukocytes are less in the exudate. In fibrinous inflammation, the exudate is high in fibrin, which indicates an increase in vascular permeability. As a result, in addition to albumin and globulins, fibrinogen leaks into the interstitial fluid, forming fibrin fibers and membranes, which coagulate. Diphtheria is when the fibrin sits flat between the tissue and on the surface, moves hard on the surface of the organ, and forms a wound.

During inflammation, krupoz inflammation is when fibrin sticks to the surface of the tissue and between them and moves easily without forming a wound.

Purulent inflammation occurs in all parts of the body, with the accumulation of pus in the inflamed parts. This fluid contains a large number of leukocytes, tissue fragments with a high specific gravity. Purulent exudates fill the space in the interstitial space and form an abscess or abscess, inflammation of the sebaceous glands and hair follicles-boils, inflammation of a group of fat and wool bulbs is called carbuncle.

When putrefactive bacteria enter the inflamed parts and dissolve the tissue, the ulcer is called dissolved inflammation and is well manifested in alteration processes.

In hemorrhagic inflammation, the exudate becomes red due to the retention of erythrocytes. Vascular permeability results from acute and severe infectious diseases and poisonings.

In proliferative inflammation, cell proliferation increases oncotic pressures above other processes.

During exudation, water, salt, protein, or cell-free products are released from the blood vessels, and then leukocytes are released from the blood vessels into the tissues, called leukocyte emigration. During leukocyte emigration, the localization of leukocytes along the vascular walls occurs, resulting in the redistribution of mine-shaped elements, which is associated with slowing of blood flow. In normal life processes, the blood is characterized by the placement of two layers of thin, plasma at the edges of the blood vessels and shaped elements moving in the center, the specific gravity of erythrocytes is heavy between the blood vessels, leukocytes move lightly on the periphery.

As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cetoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of

inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Intact leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that degrade in large acidic environments and under osmotic pressure

Neutrophils exhibit their resistance at pH 5.5.

While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. According to AS Shklyarevisky, one of the proponents of the mechanical theory explaining the location of leukocytes in the blood vessels, the specific gravity of leukocytes is light, including inflammation of the connective tissue at the site of inflammation, sepsis, actinomycosis, proteinuria and other diseases. 'sib, granuloma is formed, resulting in the passage of toxins and microorganisms from the inflamed area to healthy tissue. Biologically active substances released from leukocytes and other cells, as well as changes in osmotic and oncotic pressure in inflamed parts play an important role in the occurrence of proliferative processes. These modes tickle the receptors in the injured parts by the reflex pathway.

Depending on the immunobiological reactivity of the organism, normergic, hyperergic and hypergic inflammations are distinguished.

Normergic inflammation is caused by the primary exposure of microbes or toxins to organisms that are not sensitized and have normal immune properties. Hyperergic inflammation occurs after repeated exposure of the body to the cause of the disease. This inflammation is accompanied by a strong acute flow, alternating and exudative processes. Changes in this period do not depend on the strength of the antigen, but rather on the increase in the sensitivity of the organism. Alterative changes in hyperergic inflammation begin with fibrin bending and necrosis of halogenated and smooth muscle fibers. The fibrin in the exudate is hemorrhagic because it is a mixed serum. Examples of local allergies to hyperergic inflammation are pulmonary embolism and infectious inflammation in acute rheumatism.

Hypergic inflammation is slow, weak. Hypergic inflammation occurs in organisms that may have immunity to this antigen, or are very weak, emaciated, and less reactive. For example, if a diphtheria toxin is injected into the skin of an animal vaccinated against diphtheria, a very slow local change occurs. Such a sluggish response is observed due to decreased reactivity in animals with strong lean and malignant tumors.

Why does inflammation manifest as a general organism change?

Yallig‘lanish manbai bilan organizm o‘rtasida o‘zaro aloqadorlik va bir-biriga ta’sir etish hosil bo‘lib turadi birinchidan yallig‘lanishning hosil bo‘lishi va rivojoanishi organizm reaktivliligiga, uning boshqaruvchi mexanizmi, moddalar almashinuvi va boshqalarga bog‘liq ikkinchidan yallig‘lanish manbai organizmdagi moddalar almashinuvi, immunologik xususiyatlarga ya’ni barcha organizmga ta’sir qiladi. Sensibilizatsiyalangan hayvon organizmga zaharli bo‘lmagan qo‘zg‘atuvchilar bilan ta’sir etilganda kuchli giperergik yallig‘lanish kelib chiqishini, immunlangan organizmlarda zaharli moddalarga xos yalig‘lanish jarayonlarini chiqaradi. Yallig‘lanishning shakllanishida nerv reflektor jarayonlar muhim ahamiyatga ega. Masalan: retseptorlarni blokada qilib yallig‘lanishni susaytirish yoki umuman hosil qilmaslik mumkin. Nervsizlantirilgan to‘qimada yallig‘lanish juda sust va belgilarsiz kechadi. Simpatik nervning qo‘zg‘alishi yalig‘lanishni susaytirsa, parasimpatik nerv kuchaytiradi. Oraliq miyadagi kulrang do‘mboqchaning uzluksiz qo‘zg‘atilishi organizm turli qismlarida: terida, ichki organlarda keng yallig‘lanish jarayonini chaqiradi. Hayvonlar narkoz xolatda, qishqi uyqu vaqtida va po‘stloq tormozlanganida harqanday kuchli qo‘zg‘atuvchi ham yallig‘lanish chiqarolmaydi. Hayvonlar organizmining murakkablashishi, nerv sistemasining diferensiyalangan bo‘lishi, ularda yallig‘lanishni to‘la belgilari bilan aniq kechishiga, organizmning ximoyaviy

xususiyatlarida fagotsitoz, leykotsitlar emigratsiyasi va proliferativ jarayonlar yaqqol kechishini ta’minlaydi.

Inflammation is also affected by the endocrine glands, while thyroxine, aldesterone and somatotron hormones increase inflammation, while AKGT, cortisone and sex hormones histamine, acetylcholine, serotonin and others.

Inflammation depends on the age, type, constitution, sex, and other characteristics of the animal, and hyperergic inflammation cannot occur in young animals. If the signs of inflammation are well manifested with the age of the animal, in old, loose constitution, inert nerve-type animals, inflammation is slowed down and conditions are created for the spread of the pathogenic agent in the body. Inflammation of the abdominal cavity of horses is more acute and severe than in cattle, or if we send tuberculosis rods under the skin to guinea pigs, they form a long-term incurable wound at the injection site. calls. The development of inflammation depends on the anatomophysiological structure of the organism, if the inflamed parts are well supplied with blood vessels, the inflammation will be so strong and, conversely, if the blood vessels are poorly supplied, the inflammation will be asymptomatic. Inflammation is affected by animal nutrition, metabolism, low protein content in the diet, reduces the formation of immune cells in the body of the animal, weakens the resilience of patients, vitamin A deficiency from avitaminosis can lead to easy inflammation of the eyes and respiratory tract. causes. The intensity of inflammation varies in different vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different vitamin deficiencies.

How does the source of inflammation affect the body?

Yallig‘lanish organizmning mahaliy qon tomirlar reaksiyasi sifatida nomoyon bo‘lishiga qaramasdan, organizmning umumiy xolatiga, moddalar almashinuviga, immunobiologik reaktivliligiga, qon tarkibiga, termoregulyasiya va jarohatlanmagan to‘qimalarga ta’sir qiladi. Yallig‘lanish davrida moddalar almashinuvining buzilishidan, glikoliz jarayoni kuchayib qonda qand miqdorini ko‘payishiga, albumin-globulin indeksini o‘zgarishiga, globulinlarni ko‘payishiga, qonda qoldiq azotni, albumoz-peptonlarni, gistamin, nukleinlar almashinuvining oraliq mahsulotlari va atseton tanachalarini ko‘payishiga olib keladi. Qonda leykotsitlar ko‘payadi, ECHT tezlashadi, tana harorati ko‘tariladi. Immunobiologik reaktivlik yo immunitetni hosil bo‘lishini kuchayishi yo pasayishi bilan harakterlanadi: emlash va kasallikdan tuzalgandan keyin antitela hosil bo‘lishi va fagotsitoz kuchaysa, surunkali kechadigan yallig‘lanish jarayonida immunobiologik reaktivlik va rezistentlik susayishi madorni qurishiga olib keladi. Yallig‘lanish manbai o‘ziga yaqin to‘qima va organlarga ta’sir qilib hayvonlar qorin bo‘shlig‘iga filogen moddalar ta’sirida qorin devoriga yuborilgan mikrobga turg‘unligi kuchayib, bu mahalliy to‘qimalarni immunologik xususiyatlarini kuchayishidan hosil bo‘ladi. Yallig‘lanish manbailarini jarohatlanmagan to‘qimalarga ta’sirini ba’zan organizmdagi qorin sohasining yallig‘lanishi appenditsit yoki aritmiyalarini hosil bo‘lishida ko‘rish mumkin.

The inflammatory center affects the whole organism, affecting its metabolism, reactivity, uninjured organs and systems due to the microorganisms accumulated in these inflamed parts, their breakdown products, toxins, biologically active substances that are absorbed into the blood and tickle the receptors. . The body is also affected by painful stimuli coming from the source of inflammation. The increase in body temperature is caused by the effect of completely undigested substances formed in these parts on the thermoregulatory center in the midbrain. Thus, the source of inflammation affects the body through nerve reflex and neurohumoral pathways.

What do you mean by the mechanism of development of inflammatory processes?

It is a complex reaction of the organism to inflammatory influences that appeared very early, and theories explaining these processes have also been known since very ancient times. The protective

properties of inflammation are also stated in the ideas of Hippocrates, who have different views and worldviews on the essence of inflammation.

According to R. Virkhov's 1958 theory of nutrition, inflammation is the transition of cells to a high functional state under the influence of inflammatory factors, a state of intensive consumption of nutrients. However, cells not only undergo a high functional state under the influence of a phlogogenic agent, but also under a high functional state during other effects. R. Virkhov equated inflammation with a simple arousal phenomenon and could not explain that arousal is another qualitatively specific phenomenon. If the proliferative and exudative processes in inflammation are considered a high functional state, the alternative process cannot be considered as such. By binding the inflammation to the cell,

Congeym's theory of vascular changes in 1885. It is said to cause changes in the blood vessels leading to inflammation. Congeym says that the changes that occur in inflammation are due to increased vascular permeability, i.e., exudation and emigration. This theory ignores the fact that other tissues, not blood vessels, play an important role in the development of inflammation. The fact that there is an inflammatory process even in animals with underdeveloped vascular systems did not take into account the fact that vascular permeability is controlled by the nervous and humoral systems.

In Ricker's vasamotor theory, inflammation is explained as a phenomenon associated with changes in the vasomotor nerves under the influence of a phylogenetic agent. Inflammatory nerve exposure causes changes in vascular permeability and tone, leading to the formation of inflammatory-specific metabolic changes in tissues. In this theory, the interaction between the flogen agent and the tissue is ignored and the role of the nervous system is limited. IIMechnikov's phagocytic theory was stated in 1892. Inflammation is a protective reaction formed as a result of evolutionary development, in which specific cells of inflammation (RES cells) are considered active in response to the action of a phlogogenic agent. This theory suggests that vessels, other than phagocytes, are cells of the nervous system,

In Shaden’s physicochemical theory of 1923, he explained that inflammation under the influence of a phylogenetic agent disrupts tissue metabolism and alters the physicochemical properties of colloidal substances as the main pathogenetic chain of inflammation. Inflammation is only a local process, it does not take into account the reactivity of the organism, the state of the regulatory mechanisms that play an important role in the development of inflammation. Thus, inflammation is associated with alteration, necrobiosis, venous hyperemia, stasis, intoxication, dysfunction and other events, on the one hand, arterial hyperemia with protective compensatory properties, accelerated metabolism, leukocytosis, phagocytosis, emigration, multiple antibodies. and the formation of biostimulants, proiferation,

At the end of the twentieth century, the role of the nervous system in the development of inflammation was raised. Samuel recognizes and promotes the importance of the nervous system, saying that neurotrophic processes play an important role in the origin of inflammation, that the influencer affects the cell through the nervous system.

While V.Ya. Danilevsky cut the sympathetic nerve and observed strong inflammation in the tissue controlled by this nerve, Ricker explained that inflammation is caused by dysfunction of vasomotor nerves, and these theories led to the notion that inflammation occurs in the organs. will come.

Only IPPavlov tries to explain that with the development of the theory of nervousness and its role in the nutrition and metabolism of the nervous system is important, that inflammation develops on the basis of important laws. IPPavlov observes that wounds on the skin and mucous membranes of dogs with tubes are formed under the influence of chronic pathogens. These chronic movements are caused by improper placement of the tubes. Inflammation is provided only by the injured nerve and has been observed in other organs or tissues as well, not only in the tissues. For example, inflammation of the cornea of the eye was observed when the sciatic nerve, the cervical sympathetic node and the gray ball and some centers were stimulated. The effect of the cerebral hemisphere on the inflammatory process, when the bark is removed or the animal is anesthetized, the inflammation is sluggish and goes unnoticed. Similar changes are not caused by inflammation during the hibernation of animals, in severe poisoning (mustard, when large amounts of leucites are introduced into the body). Loss of

receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body.

Control questions

1. What is inflammation?
2. What are the main pathological changes observed during inflammation?
3. What are the causes of inflammation?
4. What are the external local changes that occur during inflammation?
5. What is the morphological classification of inflammation?
6. What is the classification of inflammation according to the composition of the exudate?
7. What is the classification of inflammation according to the immunobiological properties of the organism?

O 'SMALA R.

Plan:

1. The concept of tumors. Types, etiology, pathogenesis of tumors.
2. The main features of malignant and benign tumors. Distribution and biological properties of tumors in the animal kingdom. Anaplasia: morphological, biochemical, physicochemical, energetic.
3. Metabolism in tumors. Interaction of the organism with the tumor. Theories of tumor etiology.

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BASIC EXPRESSIONS.

Tumor, oncology, oma, histoid, orgonoid, sarcoma, cancer, metastasis, recurrent, infiltration, Hippocrates, treatable, incurable, phosphorus plant, turmeric disease, chemical, exposure, embryonic

bulge, virus, maleg nization, anaplasia, glycolysis, RVKavetskiy, LAZilber, MKPetrova, AABogomolets, carcinolysis, B.Zbarskiy, Stern, Wildheim, Rondoni, MANavinskiy, Ivan Polikarpovich Mishchenko, anemia, Raus, ADTimofeevskiy.

Tumors are defined as the growth of tissues in a stable pathological state, the inability of tissues to have specific biological properties, unlimited growth and control, and changes in the structure and function of tumor cells. These properties in tumor tissue are caused by the influence of external and internal environment on disease-causing causes in healthy cells in the body. Tumor tissue, unlike other pathological changes in the tissue, does not have the properties of regeneration and flexibility (regeneration, hypertrophy, proliferative inflammation) in the body. Not only does the tumor increase in size when the tumor grows, but the tumor can also break down the surrounding tissue.

The branch of pathological physiology that teaches the problems of tumors is called oncology-Greek-oncos-tumor or neorlasma-new abnormal formation, Latin-tumor-tumor. Tumors can form and develop from healthy tissues in the body (epithelial, connective, muscle, nerve). Tumor-forming substances are called carcinogens. The transformation of healthy cells into tumor cells is called malignancy. Tumors are formed by adding a suffix "oma" to the name of the tissue from which they are formed: For example: epithelioma, fibroids, lipoma, osteoma, chondroma, adenoma and others. Some tumors, as they are called by their historical name, are called malignant tumors (sapsech, sachstpoma) formed from epithelial tissue and malignant tumors formed from connective tissue. Tumors have a parenchyma and a stroma, and the characteristics of the tumor depend on its parenchyma. Blood vessels and nerves pass through the tumor stroma and are composed of connective tissue. Because malignant tumor stroma is so poorly developed, these tumors are called histoid tumors. In benign tumors, the stroma is well developed, surrounded by a thick shell, and is called an orgonoid tumor, reminiscent of a parinchymatous organ. If the tumor parinchyma is composed of multiple tissues. These tumors are called mixed tumors. Hence, we study all tumors into two groups i.e. malignant and benign tumors. Malignant tumors include cancer and sarcoma, all remaining tumors include fibroids, fibroids, ostiomas, chondromas, adenomas, and other benign tumors.

Safe tumors are called tumors that are close to the mother cell and mature due to their morphological structure. As benign tumors grow, they grow from the center to the periphery, enlarging to form a connective tissue shell and compressing the surrounding tissue as they grow. Because benign tumors have a connective tissue shell that is confined to the surrounding tissue, they grow slowly and sometimes temporarily stop growing. In dogs, the size of the tumor increases and the dogs become 1/3 of their body weight. The expansion of a tumor without growing into other tissue is called expansive growth. Safe tumors do not recur and metastasize when surgically removed because they are surrounded by a good connective tissue shell. Of course, a safe tumor is a relative concept. the formation of this benign tumor in the brain leads to disruption of the activity of various nerve centers by squeezing the brain. Safe tumors that form from the endocrine glands cause the production of many hormones and disrupt the functions of the endocrine glands. Safe tumors grow around the red eyelids and other tubular organs, squeezing them, causing dysfunction.

Malignant tumors grow rapidly, irregularly, and are not limited to the surrounding tissues, but grow into them and are called infiltrative growths. Malignant tumors injure the surrounding tissue. The central part of malignant tumors disintegrates without good nutrition and does not become large in size. Tumor growth is variable, sometimes rapid, sometimes slower than in benign tumors. When malignant tumors grow, there is no boundary between the tumor and the healthy tissue, so the malignant tumor cannot be separated from the body. If the sma cell remains, it recurs. Recurrence is a characteristic feature of malignant tumors. A recurrent tumor can form long after it has been removed. Malignant tumor metastasis-Greek metastasis - displacement, interference, which causes tumors to grow into the blood and lymph vessels, staring at the capillaries and forming an embolus. Cancer often metastasizes through lymphatic vessels. Wherever tumors develop when they metastasize, they retain the characteristics of maternal tumors. For example, regardless of which part of the body the hepatoma is formed, it produces urethra, a tumor formed by the thyroid gland is rich in iodine. The formation of

metastases depends on which blood vessel the embolus flows through. For example: If the cancer has developed in the stomach, it metastasizes primarily to the liver. In other cases, the formation of metastases depends on the biochemical properties of the tissue in which the metastasis occurs. If you have lung cancer, metastasis will form in the brain and adrenal glands. Thyroid, malignant tumors of the prostate and mammary glands often metastasize to bone tissue. However, the entry of tumor cells into the organs does not always lead to the formation of tumors because they are broken down by macrophages. For example, the flow of cancer cells in the spleen does not cause metastasis. Malignant tumors are so different from benign tumors that in malignant tumors the metabolism changes more deeply than in benign tumors, causing the animals to lose weight.

Tumors are found in all farm and domestic animals, birds, amphibians, and fish. It is even found in various invertebrates as shown in the literature. Sarcoma from malignant tumors in cattle, lipoma, fibroma, ostioma from malignant tumors, melanosarcoma, osteosarcoma and cancer from more dangerous tumors in horses are found in cattle. Tumors in the genitals and other parts of bulls and stallions are more common. Tumors rarely form in the stomach and uterus of animals.

Tumors are more common in older animals. Dogs of purebred and older than 5 years of age have a variety of tumors, most commonly tumors of the genitals and mammary glands. Tumors are rare in rabbits, and tumor damage is very rare in guinea pigs. While laboratory animals are more likely to develop cancer in mice, sarcoma is more common in rats. According to some data, 6-8% of mice die from cancer. Tumors also occur in chickens, where they develop sarcoma. Similarly, geese and ducks are also affected by tumors. In birds, malignant tumors grow and metastasize. In fish, as in other vertebrates, epithelial and connective tissue tumors are different. Tumors are more common when fish are artificially bred and are less common in free-living fish.

Tumor formation also depends on the age of the animals, with tumors occurring in humans after the age of 40, in dogs after the age of 5, in chickens at the age of one year, and in older animals 10%. The occurrence of tumors in older animals is associated, firstly, with the long-term effects of etiological causes, and secondly, with a decrease in the body's protective functions.

The importance of hereditary traits in the origin of tumors has not yet been definitively studied. However, cancer is caused by viruses, and if an animal is born with cancer after birth, it will develop cancer. This condition is well studied by infecting the animal's udder with the virus.

2. The causes of tumors have not yet been fully studied, and the first information about tumors dates back to 1500-2000 BC in ancient Egypt and Rome, and Hippocrates in those days. tumors can be treated or untreated. In the seventeenth century in England in the cleaners of factory pipes - a disease of pipe workers, in the United States - tumors in the clockmakers of a phosphorus plant. In the first half of the 19th century, œcìàëàð was found to be composed of cells, like other tissues, and the origin of tumors has been explained by various theories. One of these theories is the theory of embryonic buds, in which Congeim argues that during the embryonic development of an organism, some of the cells fail to develop, and that various causes, strikes, due to inflammation and other causes, growth energy is formed in cells that live in secret and begin to grow. Tumor feature is formed. Tumors begin to form. Proponents of this theory explain that tumors and embryonic tissues have morphological similarities, that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor.

R. Virkhov's theory of exposure was developed in 1885 and explains that it is caused by the action of long-term pathogens on tumors, resulting in the formation of lesions in many tissues. This theory explains that tumors are formed in humans and animals in the processes of tissue breakdown, inflammation, and regeneration due to long-term mechanical, thermal, chemical, and other effects. It is

said that cancerous tumors are formed as a result of long-term exposure of certain parts in people performing the same functions, from proliferative inflamed parts to the differentiation of cells. But not all formed scars and wounds form tumors. This theory seeks to explain that tumors are formed under the influence of chronic influencers of the external environment. VVPodvesotsky observed that tumors do not form when the body is exposed to mechanical and chemical agents for a long time. However, due to this theory, conditions have been created for many studies and the causes of tumors have not been identified. As a result, in 1916, Japanese scientists K. Ishikova and K. Yamagiwa discovered that tumors are caused by chemicals. They rubbed dyogt charcoal on the inside (skin) of rabbit ears for a long time, causing malignant tumors. Diagnostic cancer was later invoked from experimental animals in mice, rats, and dogs. Two weeks after the coal tar has been applied, the wool from these resinous parts falls off and new wool emerges, and after this change is repeated 6-7 times, the wool does not grow on the skin at all. the skin thickens, roughs, cracks, the outer surface of the skin sheds and alternates. If we observe these parts under a microscope, we will see acute, moderately acute and chronic inflammation of the skin after a month in the place where the coal tar was applied. 3-4 months later, sometimes earlier, sometimes later, one or more questions arise. These tumors then grow, enlarge, infiltrate, and metastasize to a cancerous tumor. Subsequent research has shown that carcinogenic chemical compounds are synthesized from various resins that cause tumors. Carcinogens are polycyclic carbohydrates with their chemical structure. Carcinogens form tumors after several latent periods after they enter our body. If left untreated, a rapid tumor can form. Cancer tumors form by the 31st to 179th days after the skin is coated with methylcholentren. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat in mice and rats from hungry animals.

Cholesterol, sex hormones, vitamin D, carcinogens in the benzperin group are chemically close and they are phenanthrene products. Some substances change their carcinogenic properties as a result of various effects. For example, cholesterol in grass can be turned into a carcinogen under the influence of radiation. NILazerev's observations show that when hormones are overproduced or a decrease in their antagonists leads to tumor formation. This means that an adequate stimulus forms a

tumor when it changes in quantity. The process of cell dedifferentiation and rapid proliferation to form a tumor can lead to malignancy and tumor formation.

Impaired sterein metabolism from fats and lipids is a factor that contributes to the growth of tumors. The formation of malignant tumors under the influence of carcinogens is one of the important achievements of experimental oncology. However, the mechanisms of action of carcinogens have not yet been elucidated. Perhaps the effects of carcinogens acquire biological properties by altering the genetic properties of cells by disrupting the structure and function of nucleic acids. Even chemical theory cannot fully explain the formation of tumors. He explains that chemicals only create the conditions for viruses to affect the body.

From the end of the last century to the present day, tumors have an infectious nature, they explain the parasitic ducts that cause disease in various animals and plants, worms-worm-like parasites, fungi are specific pathogens of tumors. During the study of tumors, many microorganisms were isolated, but all of them were found to be saprophytic microbes and not related to tumors. Malignant or malignant tumors also occur when infected with certain parasites: Cancer can occur in dogs and cats when infected with Oristorshis felineus, which belongs to the class of suckers. Cancer develops when rats are fed cockroaches, or when cattle become infected with fasciola, which causes liver cancer.

The notion that tumors are caused by viruses was first proposed by II Mechnikov in 1910, and in 1911 an English scientist, P. Rose, observed that tumors were formed by sending a filtrate made from sarcoma-infected chicken tissue. P.Rous virus is found not only in tumors but also in the heels, liver, brain, blood and other fluids of chickens, the size of the virus is 01 m. Low resistance to chemical and physical influences. For example, it decomposes in 2-3 days at a temperature of 00, and in 15 minutes at 550. Antisetics have a strong effect on the virus. Some tumors can grow in an environment made of tissue. Safe tumors formed under the influence of viruses have been observed in various animals to develop into malignant tumors. For example: papilloma of wild rabbits, in dogs and cattle papillomatosis is similar to the warts that occur in humans, and the virus isolated in these animals causes tumors only in this type of animal. Most tumors can only develop in a healthy organism when transplanted. Proponents of viral theory, such as LAZilber et al. The tumor-causing virus may not show its pathogenicity for a long time, even in all vital processes. For example, while some species of mice reach a certain age, most of them become infected with tumors, while others develop one or two tumors. Because tumors can also call a healthy animal child by suckling an infected animal, this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact

that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories.

3. Tumor growth begins with the transformation of normal healthy cells into tumor cells, and the metabolism in these cells changes. produces qualitative changes from the biological properties of the cell. Later tumors grow only due to the proliferation of tumor cells. Of course, not all tumor cells turn into tumors, some are absorbed, and some form multiple tumors.

One of the main characteristics of tumors is that they can grow continuously and, if not removed by a doctor, squeeze the animal's organs, causing death under the influence of toxins. As a result of continuous growth of tumors, the fibroma in cattle reaches 100 cm in diameter and weighs up to 100 kg, about half the weight of the animal. In humans, uterine fibroids weigh 20-25 kg, and ovarian cysts range from 50 kg. By transplanting tumors in the same species, it is possible to ensure their growth for several years. One of the characteristic features of tumors is the transformation of tumor tissue into low-differentiated tissue.

Anaplasia refers to low-level morphological differentiation of mother cells into tumor cells, and Greek means mother-back, down, plasis-formation. In a cell that is becoming a tumor, the rate of growth and proliferation increases. The faster the growth in the tumor cell, the better the anaplasia develops. Usually morphological, biochemical, physicochemical and energy anaplasia are distinguished.

1. In morphological anaplasia, changes occur in the tumor cell and tissue, and according to the morphological features, the tumor tissue is close to the embryonic tissue. The shape and size of the parenchyma of tumor cells vary. In some cells, the normal ratio of nucleus and protoplasm is different, the number and shape of chromosomes change. The division of tumor cells is atypically malformed, disrupting the mutual arrangement of cells. For example, glandular tumors do not have or have a malformed structure that produces glandular fluid, but retains the functional properties of tumor cells despite having such an atypical structure. That is, tumors formed from melanblasts melanin, tumors formed from liver cells, tumors formed from grass, glandular cells, maintains the function of hormone production. Morphological atypicality is not specific to tumors but can also result in cell growth and proliferation in a variety of pathological conditions. For example: During regeneration and proliferative inflammation.

2. During biochemical anaplasia, the biochemical properties of tumors change, that is, as in embryonic tissues, the amount of water increases to 90%. Potassium salts increase and calcium salts decrease from normal. The faster the tumor grows, the more the ratio of potassium and calcium changes.

Tumors increase cholesterol from lipoids. Tumors accumulate a lot of glycogen, which does not absorb glycogen well. This glycogen accumulates as a result of disruption of carbohydrate metabolism and is associated with an increase in lactic and pyruvic acids in tumors.

DNA and RNA increase in tumor tissue. As a result of the strong breakdown of nucleic acids, pentoses are formed in tumors, the amino acid composition changes, ie cystine, methyanine, tyrosine are reduced in tumors, and histidine, arginine and lysine are increased. Tumors are rich in protolytic enzymes.

3. In physicochemical anaplasia, the surface tension properties of colloidal substances are reduced, many completely unoxidized intermediates are formed, changing the acid-base balance to acidic. Osmotic pressure rises in tumors. Tumor tissue has a higher electrical charge than healthy tissue. Tissue and cell membranes have strong permeability properties. Biochemical and physicochemical anaplasia occurs in the process of regeneration or proliferative inflammation without any specific changes for the tumor. The stronger the growth of a charged tumor, the better the biochemical and physical anaplasia.

Energy anaplasia is caused by changes in metabolism and excessive metabolism in tumors, disruption of carbohydrate and protein metabolism.

4. Metabolism in tumors differs from that in healthy tissues, i.e. we can better observe these changes in carbohydrate metabolism: in healthy tissues, carbohydrate metabolism takes place in 2 periods: anaerobic and aerobic.

As a result of many intermediate changes in the anaerobic period, lactic acid is broken down - called glycolysis.

In the aerobic cycle, 1/5 of lactic acid is oxidized to SO2 and N2O, and the remaining 4/5 is converted to glucose due to energy generated by oxidation.

During glycolysis, 5% of potential energy is wasted on carbohydrates, the remainder being oxidized to form S2O and N2O from lactic acid. When the oxidizing properties decrease, a lot of lactic acid is formed, and acidic substances accumulate in the tissues. Glycolytic processes are dangerous tumors, the breakdown of glucose to lactic acid is 200 times faster than in resting muscles and 8 times faster than in maximally working muscles. Malignant tumors can produce lactic acid equal to their own weight in 10-12 hours. Therefore, the amount of lactic acid in the blood is higher in cancer-prone organisms. Glycolytic changes in malignant tumors are more active than in benign tumors. The formation of large amounts of lactic acid, changes in the surface tension of tumor tissue, etc. are characteristic of tumors. Cancer cells break down glucose 4-5 times more strongly and oxidation is very slow. Glycol = dog processes are not characteristic of tumors, because glycolytic processes occur in the retina, leukocytes in healthy life processes, increased glycolysis, decreased oxygen consumption are also observed in the process of inflammation and regeneration . Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a decrease in oxygen consumption are also observed in the process of inflammation and regeneration. Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a decrease in oxygen consumption are also observed in the process of inflammation and regeneration. Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body

In tumors, protein metabolism is severely impaired, albumin and nucleoproteins are increased in tumor proteins, and proteins that are not found in healthy tissue are found. The formation of these nucleoproteins has not been studied, but other proteins or viruses of a different nature (LAZilber) or proteins that have been altered by the body in the formation of tumors.

In malignant tumors, full-value and full-value amino acids can also be formed. Proteins in this change can disrupt the activity of enzymes. BIZbarsky determined that specific protein synthesis occurs in tumors and is called tumoproteins.

The disruption of specific nucleic acid metabolism in tumors was discovered in 1934 by Stern and Wilheim, and later in 1941 by Rondoni in tumors where DNA was more than RNA. It has been studied that protein synthesis in tumors is superior to its breakdown by sending various identified atoms into the body. The fact that purine and pyrimidine bases from large amounts of amino acids fall into the

tumor tissue and that the amount of residual nitrogen in the tumors is high indicates that the protein metabolism in tumors is faster than in healthy tissue.

The metabolism of fats and lipids is strong in tumors and varies depending on the nature of the tumor. Fats are high in unsaturated fatty acids, cholesterol and acetone cells.

Relationship of tumors with the organism. Based on the data collected in the experiments, MKPetrova explained that the effect of the body on the growth of tumors can affect the nervous system in tumors. The creation of conditions for the origin of tumors in chronic functional disorders of the nervous system (neuroses) in the animal body has been studied experimentally by calling dangerous and benign tumors. During the period of chronic functional disorders of the nervous system, the formation of tumors under the influence of carcinogens is accelerated. The role of the nervous system in the mechanism of tumor development has been observed to slow the growth of tumors under the influence of carcinogens during the hibernation of animals or the inhibition of nervous system activity, and accelerated tumor growth in controlled animals receiving so many carcinogens. If we send sodium bromine to the body, the activity of the nervous system decreases and the formation and development of tumors slows down. It is during this period that the effects of caffeine or nervous system stimulants on rabbits accelerate tumor growth.

Injury to peripheral nerves contributes to the formation of metastases. If the sympathetic nerve of the neck is cut, malignant tumors will form, which will help the transplant to grow. The effect of RES tissue on tumor growth is significant, as macrophages can break down the tumor without developing it, preventing it from growing. Macrophages resist metastasis by trapping malignant tumor fragments that enter the blood and lymph. AABogomolets and MANavinsky in 1877 observed that activation of RES tissue function prevents the transplantation of transplanted tumor tissue, or blockade of RES tissue creates conditions for the growth of transplants.

The body influences the growth of tumors through hormones produced in the endocrine glands. While one of these hormones inhibits the growth of tumors, the other accelerates the growth of tumors. For example, while somatotron hormone in the pituitary gland enhances tumor growth, hormones in the pancreas and adrenal cortex inhibit tumor growth. When we send estrogen hormones to an animal's body, a tumor develops in the animal's udder and genitals. Testosterone and progesterone inhibit tumor formation in the udder and genitals.

As the body reacts to tumors, so do tumors. The effect of tumors on the body depends on the nature of the tumor, its growth and the location chosen. If there are small tumors on the surface of the hand, they fall into the category of benign tumors, which only cause discomfort when doing any work. possible. Safe tumors compress the surrounding tissues, disrupting their nutrition and leading to atrophy. If the sap compresses the separating pathways, the sap becomes difficult to separate, and so on

Although malignant tumors are small, they degrade the body and lead to death due to impaired growth and metabolic disorders. The cause of weight loss in animals is caused by metabolic disorders, poisoning the body with intermediate products of metabolism and due to the breakdown products of tumor tissue. From it, the dysfunction of the organ in which the tumor grows also causes the body to lose weight. Tumors show antigenic properties to the organism as they begin to grow, but the structure of these antigens has not been determined, but antibodies to these antigens are formed. Antigens are sufficiently foreign, due to the lack of foreign antigenic properties, as well as the weakening of the immune-forming functions of the immune system and the low production of immunogens, which can not protect the body. The presence of malignant tumors in the body disrupts the overall metabolism. In the initial period of tumor formation, metabolism increases and decreases in the next period. Blood glucose may increase or decrease.

Increased activity of enzymes involved in carbohydrate metabolism increases lactic acid in the blood, including in the veins. A decrease in serum albumin in the blood leads to a decrease in protein and an increase in residual nitrogen. Decreased albumins are associated with decreased protein synthesis. When tumors grow, the activity of arginase, catalase, oxidase in the liver decreases, glycogen synthesis, urea, guipuric acid formation is impaired, the total amount of nitrogen excreted from the body increases, and urinary urea decreases. In the urine, lactic acid, polypeptides, some

amino acids increase, and acetone cells appear. According to NBMedvedev, in cancer, carbohydrates are 6-7 times more than nitrogen. Tumors cause hypochromic anemias in the body, decreases to 0.5 to the color index of the blood. Anemia is caused by the breakdown of erythrocytes under the influence of various charged substances, ie not completely oxidized. Disruption of the control of the activity of blood-forming organs by the formation of erythrocytes by nerves and endocrine glands leads to anemia.

During the transplants, he observed that the infinite features of the tumors were visible. Tumor strains are also present today, including the well-studied Erlix mouse cancer, Jensen's rat sarcoma, Raus's chicken sarcoma, and others, which have been transferred from organism to organism for hundreds of years and have existed for 50 years or more. The nutrition of the experimental animal plays an important role in transplant growth, and if the caloric content of the food is low, i.e. lysine, arginine, histidine, the growth of tumors is inhibited. If it contains a lot of carbohydrates, cholesterol and potassium in the diet, the growth of the tumor will accelerate. Liver cancer can develop even if the animal does not have enough choline in its diet. But the growth of tumors did not stop as a result of complete starvation of animals SAMMI researcher IP Mishenko observed in chickens and rats. Experiments have shown that tumors can be grown outside the body by creating special nutritional conditions, as observed by ADTimofeevsky et al. Thus, the role of the nervous system in the origin of the tumor is also important, as the causes of the tumor include chemicals, mechanical stimuli, light energies.

Control questions.

1. What is a tumor?
2. What types of tumors do you know?
3. What are tumors called?
4. What is the name of the science that studies the concepts of tumors?
5. Explain the difference between malignant and benign tumors?

HEAT EXCHANGE DISORDER.

Plan:

1. Disorders and causes of heat exchange regulation in the body. Hypothermia. Hyperthermia.
2. Etiology, pathogenesis, stages and types of fever. Changes in metabolism and organ-system function in fever.
3. The biological significance of fever for the body.

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BASIC EXPRESSIONS.

Interstitial brain, gray matter, MNS, conditioned and unconditioned reflex, hypothalamus, cortex, vitality, thermal stimulus, Krause cone, Ruffin's body, vascular tone, sweating, breathing, muscle, liver, neurohumoral system , hypothermia, hyperthermia, pyrogenic substance, exogenous, endogenous, environmental change, humidity, wind, medicinal substances, light, poisoning, injury, newborn, lean, old, animal species, poultry, pigs, cattle, four-period change, compensation, heat, poor ventilation, transport, species, breed, age, color, three-period change, fever, infectious and non-infectious fever, indifferent agent, subfebrile, febrile, hyperpertensive, fever, fluctuating, decreased, types of fever, nerves, heart, blood vessels, appetite, digestive organs, kidneys, endocrine, L.Paster, IIMechnikov, Hippocrates, IPPavlov, NFGamaley, antibody, phagocytosis, vaccination, sympathetic, parasympathetic, barrier barrier.

In the body of highly developed warm-blooded animals, body temperature changes in a very short time, and their body temperature depends on the specific condition of the animal, type, development of sweat glands, time of day, age. The temperature is not the same in different parts of the body of an animal of the same species. Relatively uniform temperature maintenance in the body is ensured by physical and chemical thermoregulatory mechanisms, a process controlled by the CNS and endocrine glands.

Heat exchange is provided by the MNS using conditioned and unconditioned reflexes. Experiments have shown that in the back of the gray matter of the midbrain is a center that controls the formation and transmission of heat. This control is controlled by the centers of metabolism, vascular tone, respiration, and sweat secretion, and these processes are related to the activity of the hypothalamus and cerebellum. Needle puncture in the hypothalamus raises the body temperature of the animal to 2.5–30. Heat exchange depends on the activity of the shell, and in animals where the shell is removed, the heat exchange is disrupted. In dogs, it is possible to control heat exchange by a conditioned reflector pathway.

The heat exchange is controlled as follows: thermally excited cold-floating Krauze flasks excite the heat-floating Ruffin bodies and transmit the effect to the MNS. From there, impulses are transmitted to various organs, altering vascular tone, sweating, respiration, altering metabolism in the muscles and liver, and regulating heat exchange also depends on blood temperature. The pituitary gland, thyroid gland, adrenal gland, pancreas and other glands from the endocrine glands are involved in the regulation of heat exchange in conjunction with the nervous system. For example: if the body temperature rises when we send hormones or extracts of the pituitary gland, thyroid gland, adrenal glands, lower the body temperature by sending pancreatic extracts, or such changes in the pituitary gland, observed when the thyroid gland and adrenal gland are removed. As the body cools, the pituitary gland begins to secrete AKTG and the animal’s resistance to the cold increases. If the center that controls heat exchange in the midbrain is injured, the body does not respond to a decrease in ambient temperature with an increase in metabolism, and vice versa. Thus, the depletion of heat exchange in the body of animals is observed when the activity of the nervous and endocrine systems, as well as the activity of peripheral organs and systems is impaired. Disorders of heat exchange are manifested in the form of hypothermia, hyperthermia and fever, all of which are caused by a violation of the control of heat exchange and are accompanied by changes in body temperature of the animal.

Hypothermia is derived from the Greek word hypo- low, terme- heat, and is characterized by a decrease in body temperature as a result of the regulation of heat exchange. Hypothermia is caused by exogenous and endogenous causes. Exogenous causes of hypothermia include a decrease in ambient temperature: humidity, increased wind, exposure to medicinal substances, and radiation poisoning.

Hypothermia caused by endogenous causes: severe blood loss, starvation, starvation, weight loss, injury to the CNS (heat exchange control center), prolonged dilation of peripheral blood vessels (shock), neonatal, other in, the activity of the center that regulates heat exchange in older animals is weakened, leading to a decrease in body temperature. Pigs cool faster than cattle because a lot of heat is generated in cattle due to the activity of the anterior chambers. Birds are resistant to cold, geese do

not change body temperature at ambient temperature - 90–1020 chickens - 500, ducks - 400. Chickens are also resistant to temperature drops.

There are four periods of hypothermia:

1- During this period, the animal's body activates compensatory mechanisms that increase heat production and reduce heat transfer: narrowing of blood vessels, shrinkage, increased heat production due to muscle activity, movement and tremors, accelerated heart rate and respiration. blood pressure rises. Increases the activity of the thyroid, pituitary, adrenal glands, autonomic nervous system. General and basic metabolism, oxidation and other processes are enhanced.

2. The flexibility mechanisms of heat exchange are exhausted, heat transfer is increased, and some oxygen deficiencies are formed. But the metabolism is high and the rectal temperature drops to 29-270.

3- During this period, metabolism, cardiovascular activity decreases, respiration and rectal temperature decreases to 27-190, but during this period, if the animal is immediately warmed up, we can return to normal life processes. Cooling in the next period reduces vital processes, blood pressure, metabolism, the formation of heat completely stops, sleep is suppressed, fibrillation occurs first in the heart chambers, then in the ventricles of the heart, the heart stops working and the respiratory center is paralyzed. the temperature in the rectum cools to 12-100.

Characteristic signs for hypothermia are the weakening of the protective mechanisms of the animal organism, phagocytosis, immune formation, oxidation-reduction processes, changes in carbohydrate metabolism, the formation of oxygen deficiency. When an animal that has died from hypothermia is dissected, we see that dystrophic changes have occurred in the liver, kidneys, heart, and CNS. In recent years, artificial hypothermia has been used in surgical practice, especially in cardiac operations, to increase the resistance of the heart muscle to oxygen deficiency. During this time, the body's metabolism slows down and oxygen consumption in cells and tissues decreases. A similar situation is observed during the hibernation of animals.

Hyperthermia (Greek hyper- high, terme- heat) is an increase in body temperature of an animal as a result of a violation of the regulation of heat exchange. It is said to overheat. Hyperthermia is caused by an increase in ambient temperature, an increase in humidity without wind. At this time, heat is radiated and decomposed to the outside, which is not formed because there is no difference in temperature between the organism and the environment. Heat transfer is a key part of heat exchange control, and even the smallest metabolism in the body ensures that there is a lot of heat and that the body temperature is kept constant. Therefore, the excess heat must be expelled from the body.

Keeping animals in tight spaces, moving them in warm rooms, in poorly ventilated vehicles, doing heavy physical work and overheating the pasture can cause the animals to overheat. The high temperature resistance and flexibility of animals depends on their type, breed, age, color, and skin coating system. Sheep are resistant to high temperatures and only after the ambient temperature is 400 and above will their rectal temperature change. The resistance of animals to high temperatures depends on the development of their sweat-sweating system.

While an increase in the ambient temperature of cattle above 300 causes an increase in rectal temperature, pigs are intolerant to this temperature due to the underdeveloped mechanism of sweating. When pigs are kept at an ambient temperature of 310, their rectal temperature rises to an ambient temperature of 0.70, causing them to die without adaptation because they do not have sweat glands. They lose steam and adapt to the heat. Excessive heat increases metabolism and disturbs rectal temperature up to 440. From small animals (piglets and calves) are heat-resistant, while chickens are heat-resistant. Under the influence of heat, the appetite of animals decreases, productivity decreases, blood composition changes, breathing and heart rate increase. The strong heat of the environment in the body causes a change in three periods.

In the 1st period, the compensating mechanisms ensure a decrease in heat generation and an increase in heat transfer. In animals, metabolism decreases, sweating increases, peripheral blood vessels dilate, blood circulation accelerates, respiration accelerates. All this increases heat transfer and ensures that the rectal temperature is maintained without rising. Increased heat transfer is associated

with the passage of heated blood in the centers in the medulla oblongata (breathing, heart, blood vessels, sweat secretion, etc.). In the following periods, as a result of overheating of the organism, a second period occurs without adequate mechanisms of adaptation of the organism.

In stage II, the animal becomes agitated, pulse, respiration is accelerated, saliva excretion is accelerated, metabolism is increased, the final product is not broken down, protein is formed in the urine, rectal temperature rises to 2-30. If the heat effect still does not disappear, a third period will occur.

In period III, the activity of the nervous system decreases sharply, the heart and respiration slow down, blood pressure drops, fainting, and rectal temperature rises to 5-60. When the animal's body heats up, it stops breathing, and the heart stops beating during systole. When we examine such animals, we observe that profound changes have taken place in the parenchymal organs.

One of the conditions similar to hyperthermia is the heat stroke of the animal's body. Such changes are observed in animals during intense muscle activity, when the temperature is high and the humidity increases. Acute heat stroke can lead to death from impaired heart function.

2. Disorders of heat exchange are characterized not only by hypo and hyperthermia, but also by the formation of fever.

Fever-fenbris is a general change of the organism in relation to the pathogenic, more infectious causes, and as a result of violation of the regulation of heat exchange in the body, the animal's body temperature rises, independent of the ambient temperature. Fever is a manifestation of disease formation, which is caused by a violation of the regulation of heat exchange, including the disruption of metabolism in relation to the causes of the disease as a secondary process in the body.

There is a difference in the regulation of physical and chemical heat exchange, while maintaining the process of thermoregulation in the body of an animal with a fever. The body that produces the fever becomes resistant to the effects of heat and cold. In a fever-producing animal, the disruption of heat exchange control depends on the type of animal, age, type of nervous system, and so on. The causes of fever are diverse, and pyro-pyrogens are substances that cause fever, and we study them into two major groups depending on their properties:

1. Causes of infectious fever - various infectious diseases.

2. Causes of non-infectious fever are protein, saline, medicated, fever caused by injury to the nervous system.

Fever is caused by the action of various pyrogenic substances on the control centers of heat exchange. Fever is hypothalamic thermal, and the delivery of these substances under the skin or into the composition of venous blood does not cause any changes. A similar situation can be caused by fever by observing the thermal pathways in the gray matter of the interstitial brain of animals or the nerve pathways leading to that part. Fever cannot be caused if the back and brain are cut apart during exposure to pyrogens. Hence, peeling is also important in the formation of fever, which can also increase injury under the influence of indifferent pathogens.

Along with the nervous system, the role of endocrine glands in the formation of heat is also important. does not participate properly. For example: removal of endocrine glands and pituitary gland, adrenal gland, thyroid gland, pancreas does not cause fever, but the endocrine glands only increase the development of fever, changing the overall biotonus of the organism, reactivity, heat exchange. affects by changing the tone of the control centers. Thus, the nervous system serves as the mechanism that initiates the formation of fever.

Depending on the degree of fever in animals with fever: in subfebrile animals the temperature rises above the upper limit of 10, in febrile animals the temperature rises above the upper limit of 20, in hyperpyretic animals the temperature rises above 30 and above. The rate and degree of fever depends on the ability of the causative agent, the reactivity of the organism, the activity of the immune system, the age of the animal, the type of nervous system, obesity, storage and nutrition.

There are three stages in the development of fever in the body:

1. Temperature rise period - stadium incrementi.

2. Maintaining a high temperature-stadium fastigil from 2-3 hours to 2-3 weeks.

3. Period of temperature decrease - stadium decrementi.

With the formation of heat in each period there is a difference in heat transfer, metabolism, activity of various systems, the reactivity of the organism. Depending on the functional state of the thermoregulatory mechanisms to the reactivity of the organism, the type and strength of the pyrogenic agent, fever occurs at different levels and in different cases. In this process, the thermoregulatory nervous mechanisms, the cardiovascular system, the respiratory system, the functional state of the sweat glands play a determining role.

Whether pyrogenic agents are always present in the body during the course of the disease. Depending on whether the thermoregulatory mechanisms work like this, the following types of fever are distinguished:

1. Permanent type fever-febris continia. The high temperature does not return to normal and causes a change around 10 in the morning and evening. In croupous inflammation of the lungs, acute anaerobic and viral diseases, the temperature may rise in the first period and fall slowly or rapidly in 3 periods.

2. Relieving or remitting fever-febris remittens. Daily changes in temperature are 10 and above in the morning and evening, due to the intense relaxation of the effects of the pyrogenic agent, which occurs in catarrhal pneumonia, sepsis and others.

3. Rising or falling intermittent-febris intermittens. In fever, the thermoregulatory mechanisms are very stable, decreasing to normal when the temperature drops to 2-30 and beyond. In acute hepatitis, people encounter malaria.

4. Tinka dryer or hectic fever-febris nectica. Body temperature fluctuates between 3-50, some temperatures fall below normal and rise again. This type of fever is observed in tuberculosis and septic processes. In animals, thermoregulatory mechanisms are formed when they are stressed, weakened, and their productivity decreases.

5. Recurrent fever-febris recurrens. Body temperature is high and normal for several days, with the pyrogenic agent intensifying from time to time. This type of fever is caused by infectious anemia in horses and recurrent typhoid fever in humans.

6. Atypical fever-febris atypica. Even if the disease progresses, the temperature does not rise, and the disappearance of the disease is accompanied by a rise in temperature, which changes several times a day. This type of fever is observed in horses' mango, sepsis.

7. Ephemeral fever-febris ephemera. It lasts from a few hours to 1-2 days. This type of fever is when vaccinated against tuberculosis and mango, after giving birth to animals, after heavy muscle work, when walking a lot in the heat, or when animals are moved in wagons. It is observed in diarrhea.

During fever, changes in the activity of the nervous system, cardiovascular system, respiration and digestion, kidneys, endocrine glands may occur. Changes occur in the nervous system that lead to disruption of thermoregulation. When the body temperature rises, the SNS is stimulated and then braked. Changes in the nervous system can also be due to the pyrogenic nature of the toxins that accumulate in the body. A characteristic change in the nervous system is caused by a sudden rise in temperature at the onset of fever. It does not cause changes in higher nerf activity as adaptation to pyrogenic substances is formed in the nervous system. This indicates that the organism is poisoned and not regenerated in the MNS. The nervous system of lean animals is impaired, The sympathetic nerve activity of the VNS increases. Changes in temperature rise in young animals are stronger than in older animals. Circulatory disorders are characterized by the redistribution of blood in the body, which causes more blood flow to the internal organs and less in the skin, and later the blood vessels in the skin dilate and more blood flows. The work of the heart is accelerated by the rise in temperature to this maximum, which is caused by the excitation of the sympathetic nerve, the excitation of the cardiac nervous muscle apparatus by hot blood, pyrogens and toxins. Usually a rise in temperature to 10 causes the heart to beat 8-10 times faster. In diseases such as tuberculosis and meningitis, pulse formation weakens when the temperature rises, which is a sign that the disease is getting worse. Some fever develops arrhythmia, In the third period of fever the heart rhythm slows down. While blood pressure rises first, which is associated with increased heart rate, vascular spasm, in the third period,

blood vessels dilate, heart rate slows, and blood pressure returns to normal. Sometimes in the third period the blood drops sharply, ie collapse occurs.

Fever changes the quantity and composition of the blood, the intermediate products of protein metabolism in the blood are residual nitrogen, acidic substances increase, alkaline reserve decreases, leukocytes either increase or decrease. ECHT is accelerating. The presence of microbial plaque and even microbes in the blood of animals with fever, the formation of antibodies, etc.

Respiration is accelerated by the excitation of the respiratory center by pyrogenic substances and toxic products contained in warm blood, depending on the activity of the heart. Acceleration of respiration is observed in anthrax, swine fever, pneumonia. Acceleration of respiration has a compensatory effect, increasing heat transfer and increasing the body's oxygen saturation.

Digestive system activity is inhibited, appetite is lost, gastric and endocrine and motor activity is inhibited, and absorption is impaired. The process of putrefaction in the intestine intensifies, gas accumulates and flatulence develops. Digestive disorders lead to the development of autointaxia and deepening of pathological processes due to impaired absorption of nutrients. Disorders of the digestive organs are associated with increased activity of the nervous system, including the sympathetic nervous system in the VNS.

In ruminants, the motility of the pancreas is disturbed during fever, the secretion into the pancreas is reduced, the acidity is increased, and the microflora and microfauna of the large intestine and microbiological processes in general are disrupted. As a result, the chewing period is broken. Hypo and atony of pre-gastric lesions develop. Food is not digested by stopping in the pancreas. In other animals, movement, motor, secretory, and absorption processes are disrupted throughout the intestinal system during fever. At this time, only water is absorbed from the intestine. During the heating period, animals should be given plenty of water and easily digestible carbohydrate foods to reduce the amount of concentrates in the feed.

There are also changes in the digestive system during the fever period, in the first period there is a lot of blood flow to the internal organs and a lot of urine, while in the second period there is a decrease due to water retention in the body. In the third period, urinary excretion increases again, and the composition of urine changes, glucose sometimes appears protein, albumen.

Sweating decreases in the first and second periods of inhibition of nerve centers, and increases strongly in the third period. Increased digestive processes have a compensatory effect, releasing fever from the body, the release of toxic and pyrogenic substances in the tissues, as well as certain products of metabolic processes in the tissues, and normalize body temperature.

During fever in the liver, the ability of machevina and glycogen production is weakened, the residual nitrogen in the venous blood from the liver increases, and in some fevers, bile secretion decreases.

From the endocrine glands, changes occur in the pituitary, thyroid and adrenal glands, the secretion of AKTG in the pituitary gland increases, and the activity of the thyroid gland increases. The amount of corticosteroids in the blood and urine increases.

Pathological anatomical changes cause dystrophic changes in the parenchymal organs, swelling of the organ, fatty infiltrations.

When there is a dystrophic condition in the organs, including parenchymal dystrophic changes, they disrupt their function, which in turn affects the process of fever. The formation of dystrophic changes in the organs occurs under the influence of overheating, infection and intoxication of the organ.

4. Metabolic disorders during fever are associated on the one hand with the rise of pyrogens in the body. In addition, fever leads to starvation from decreased intake and absorption of nutrients.

Metabolism is disturbed in various ways during the period of fever, however, the general laws specific to fever are not absent. During many fevers, an increase in metabolism, with an increase in dissimilation - an increase in heat production and an increase in basal metabolism by 5-10%, an increase in cardiac and respiratory activity - intensifies the oxidation process.

During the fever, protein metabolism changes, protein breakdown increases due to toxic and thermal factors, instead of the normal 15-20%, proteins are used as a source of 30% energy, 30% of

nitrogen-fixing substances in the urine are ammonia, creatinine, urea and others. substances are separated. As a result, the body loses a lot of protein, at which time the body needs to be fed with easily digestible carbohydrates, if the fever is infectious, it is necessary to put glucose.

In chronic infectious fever, fat metabolism is increased, at which time excessive fat consumption is not only associated with fever, but also with starvation and poisoning of the animal. According to some scientists, changes in the activity of the gray matter in the midbrain, the center that regulates fat metabolism, lead to disruption of fat metabolism. Infectious and aseptic fevers are rarely accompanied by hyperglycemia, glucosuria, which is associated with a strong breakdown of glycogen in the liver and muscles and a violation of the regulation of carbohydrate metabolism.

Water - salt metabolism changes during the heating period, the accumulation in the tissues of incompletely degraded products of protein and fat metabolism, causing a lot of water retention in the tissues. Renal function plays an important role in this process, high temperature and toxins are reduced in the second period of diuresis, disrupting the filtration of the kidneys. In the third period, heat transfer, sweating, and diuresis increase, and large amounts of water are released. Salts also increase in the body as water is retained, many chlorides are retained, and many begin to be excreted in the third period. The release of phosphorus and potassium salts in fever is also enhanced by the intensification of decomposition processes in tissues.

Failure to raise or weaken the temperature during certain diseases in humans and animals has had serious consequences. Other investigators recommend the use of antipyretics during fever, given the toxicity of the organism during fever. When the problem is solved correctly, IPPavlov looks at the disease from the worldview, and if the disease simultaneously disrupts the activity of the organism, the second eliminates the cause of the disease. According to IPPavlov, when the body is affected by adverse causes, the body reacts sharply to this cause. From this process we must be able to distinguish the true disease and the physiological protective process. Therefore, considering fever as a complex process, there are mechanisms to combat the disease using real disease and physiological systems.

Control questions.

1. Where is the center of heat exchange?
2. What changes lead to a violation of heat exchange?
3. How to provide heat exchange in the body do you know the mechanisms?
4. What is hypothermia?
5. What is hyperthermia?
6. What is a fever?
7. What are the causes of fever?
8. What are the names of antipyretics?

Pathophysiology of blood

Plan:

1. Changes in total blood volume.

2. Normovolemia, Hypervolemia (pletora), hypovolemia (olighemia), their types and mechanism of formation. Blood transfusion methods. Hemotransfusion shock.

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BASIC EXPRESSIONS:

Blood, interstitial fluid, lymph, thermoregulation, transport, correlation, physicochemical environment, blood depot, hypervolymia, hypovolemia, anticoagulants, microelements, macronutrients, hyproteinemia, hyperprotenemia, azetemia, hypoglycemia, hypoglycemia, hyperglycemia, hyperglycemia ETCH, atsedosis, alcoholism. Hypervolimy, hypovolimy, simple, polycythemic, oligocythemic, pleura, hydremia, anhydremia, blood transfusion, agglutinin, agglutinogen, Landtshteiner, Yanskiy, Bogomoets, hemotransformation shock, hematopoiesis, leukocytosis, polycystic, erythropoiesis, erythropoiesis, rez leukopimia, leukemia, platelets.

Blood, interstitial fluid and lymph together form the internal environment of the body. Oxygen and nutrients are delivered to the tissues through the blood, and the final products of the metabolic process are delivered to the digestive organs.

1. In normal single-celled animals, the ore is a load that receives nutrients through the cell membrane and excretes unnecessary substances through the cell membrane.

2. Proteins and nitrogenous substances move in the vessels of low-growing animals.

3. With the development of the animal kingdom, hemolymph is formed, and hemolymph is rich in inorganic and organic substances, which contain proteins and oxygen-carrying pigments.

4. In the organism of hot-blooded animals there is a liquid tissue deposit, the composition of which has complex and extremely important functions. The importance of blood in the body depends on its function. Blood transport in the body. thermoregulation. The physicochemical environment for cells and tissues is very important in the protection and correlation, ie the coordination of neuro-humoral processes. Therefore, changes in the composition of the blood have a huge impact on all functions of the body.

There are several theories about the formation of blood, of which AAMaximov's unitary theory explains the formation of blood in hemocytoblasts - the mother cells of the blood, while later proponents of the duolistic theory explain that Erlix is formed in myeloblasts in monocytic sand.

Changes in the total amount of blood Depending on the type of animal, the amount of blood in the body is 4-5% of the body weight of 8 guinea pigs on horseback and up to 15% on reindeer. 55-60% of the total amount of blood falls on the liquid part of the blood (plasma), and 40-45% on the form elements of the blood (erythrocytes, leukocytes, platelets). Animals that are well fattened will have a much lower amount of blood than lean cattle. The better the muscle tissue is developed, the greater the amount of blood in the animal’s body.

The bulk of the blood (around 50%) is in the blood depot. The amount of moving and stored blood depends on the functional state of the organism. The amount of blood in the body increases or decreases under various pathological influences, during which time the ratio between the liquid part of the blood and the shaped elements changes.

An increase in the total amount of blood. An increase in the total amount of blood in the body means hypervolemia or pleural effusion in Latin huper- excessive, volumen- volume, and there are simple polycythemic and oligocytic types.

1. In normal hypervolemia, the ratio between plasma and erythrocytes is almost unchanged. Under normal conditions, this type of hypervolemia does not occur. Normal hypervolemia occurs after blood transfusion, and such artificially generated hypervolemia quickly return to normal due to the breakdown of erythrocytes in the blood that are then implanted in the body after first plasma filtration (transfer to surrounding tissues).

It is not dangerous to transfuse around 60-80% of the total blood volume of this organism into the body.

2. Polycythemic or true hypervolemia is caused by an increase in the total amount of blood in the body at the expense of erythrocytes. In this type of hypervolemia, an increase in blood volume leads to hyperemia in the mucous membranes, an increase in blood pressure and hypertrophy of the heart.

The blood-forming properties of the red marrow increase — in the tubular bones, the fatty marrow is replaced by red marrow, and young erythrocytes appear in the blood. Polycythemic hypervolymia is caused by chronic oxygen deficiency.

3. In oligocytic hypervolemia, the total amount of blood increases at the expense of the liquid part of the blood, i.e., the amount of water increases. This type of hypervolemia is called serous or hydremic pleurisy. This type of pleurisy occurs in kidney disease, which causes excessive water retention in the body when drinking too much water. Hydremic pleurisy cannot be called experimentally, because no matter how much saline is added to an animal's body, the deposited fluids pass into the interstitial spaces and are expelled from the body, or a very short-term increase in blood pressure occurs. observed. An increase in water content (hydremia) in the blood can occur even without an increase in the total amount of blood. This hydremia is caused by a decrease in dry matter and protein in the blood, when there is severe weight loss (cachexia), when a lot of blood is lost,

Decreased total blood volume is called hypovolemia or oligemia, which means hypo-less, decreased, volumen-volume, and is divided into simple, pilitsetemic, and oligocytemic types.

1. In normal hypovolemia, erythrocyte and plasma ratios are unchanged, resulting in a decrease in total blood volume and excessive blood loss. Injury to the vessel wall with mechanical injury or tumor. excessive blood loss due to inflammation or wound processes can lead to hypovolemia.

Sometimes a decrease in blood can also be caused by taking blood from a donor. Older and younger animals are more susceptible to blood loss than middle-aged or adult animals, while diseased organisms are more susceptible to blood loss than healthy organisms. It is dangerous for the body when the body loses 60-70% of blood and 15-30% of blood loss when the body overheats causes death. Death occurs even if the body loses about 50% of its blood quickly and in a short time. If the amount of blood lost in the body does not exceed 25%, the blood pressure in the blood vessels decreases for a short time and immediately normalizes due to an increase in vascular tone by reflex and the release of stored blood into the blood vessels. If the body loses more than 25% of its blood, a long-term stable blood pressure drop occurs. When there is a lot of blood loss, the number of erythrocytes decreases, oxidation processes in the body are provided by oxygen transported by erythrocytes present in the body. A similar situation is observed when the blood is thinned (hydremia), that is, when interstitial fluid flows into the bloodstream. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases.

Excessive blood loss leads to oxygen deficiency. When the nervous system is excited first, it then exhausts the centers that control respiratory and vascular tone by creating a wide-section braking. Cardiac function weakens, body temperature drops, and death occurs from paralysis of the respiratory center. Changes in body functions, hypovolemia or a decrease in total blood volume play a key role in lowering blood pressure. When blood is lost, it is important to put blood in the body, because if we put

a saline solution at this time, the liquid part of the delivered solution passes from the blood vessel to the tissue.

2. In polycythemic hypovolemia, the total amount of blood decreases due to the liquid part of the blood, and the amount of erythrocytes increases per unit volume. In polycythemic hypervolemia, the absolute or absolute amount of erythrocytes is normal and the dry matter and viscosity of the blood increases. The decrease in the fluid content of the blood may be due to the body not consuming water. The strong viscosity of the blood prevents it from passing through the bloodstream, including through the capillaries.

3. In oligocytic hypovolemia, a decrease in total blood volume is associated with a decrease in erythrocytes in the blood. This type of hypovolemia can be observed in cases of excessive blood loss due to incomplete recovery of the fluid portion of the blood and some anemia and anemia.

Blood transfusion. When transfusing blood: a) lost blood - proteins, enzymes, hormones of the form elements of the blood are replaced, and the transfused blood participates in the performance of biological functions.

b) has a stimulating effect - that is, increases metabolism and blood formation.

c) increases blood clotting and stops bleeding.

g) cleanses the blood of toxins because erythrocytes and proteins in the transfused blood absorb toxins. Due to blood transfusion, blood pressure is restored, the body's stability is increased. It is used in cases of severe blood loss from burns, shock, collapse, diseases that reduce the reactivity of the organism, and general weight loss, because the blood affects various functions.

Until the twentieth century, blood transfusions were not widely used due to various tragic changes as a result of blood transfusions. The creation of the teachings of K. Landsteiner and Yansky on blood groups opened a wide way for blood transfusion.

The presence of blood groups is associated with antigenic causes in erythrocytes — isohemohagoglutinogen and antibody-isohemohaglutinins in serum. In determining blood groups, agglutinogen A and B in erythrocytes of blood are taken into account. These agglutinogens can occur in erythrocytes separately and both together or not at all. In accordance with these agglutinogens, agglutinins are also denoted by the Greek letters alpha and beta. An animal does not have similar agglutinogens and agglutinins.

Heterohemoagglutinins are also present in the blood at the same time as isohemoagglutinins.

Among the animals, the blood groups of horses are very clear, cattle, goats. in pigs and dogs, low levels of agglutinins in serum and low erythrocyte adhesion properties make it difficult to determine blood groups. Therefore, their blood will always need to be tested before a blood transfusion. To do this, take 2 drops of recipient serum on a vial, dilute 1 drop of donor blood 5 times in saline solution, and if agglutination does not occur within 10 minutes, this blood can be considered as recipient blood. If it does not resemble the recipient's blood, the donor solution will agglutinate. When solutions are gradually applied to the recipient, the agglutinating property is lost by repeatedly diluting with donor blood. Therefore, in practice, the focus is primarily on the donor agglutinogen and the recipient agglutinogen. If a large amount of blood is transfused, the recipient does not lose the agglutinating properties of the donor agglutinin and may cause shock in the body.

Hemotransfusion shock is a reaction that occurs when groups of blood are improperly placed in the body, and for the development of shock it is enough to put 80-120 ml of blood in groups that do not correspond to groups. As a result, the animal develops strong agitation, rapid breathing and heart rate - tachycardia. Decreased blood pressure makes breathing difficult, mucous membranes turn blue, vomit, urine and feces are no longer dependent on the activity of the organism. Shock often occurs within a short time, sometimes a few hours after a blood transfusion, and causes death. If the blood groups are not matched enough, the shock will pass immediately.

Some scientists explain that shock groups are formed by improper blood transfusion due to embolism of blood vessels in the brain, lungs, kidneys, while others explain that they are formed due to the breakdown products of erythrocytes in the recipient organism. Not all scientists agree with such analyzes. Experiments have shown that the mass formed by the adhesion of erythrocytes breaks down quickly without being stable and does not disrupt the activity of the organism. Even when hemolyzed

blood is transfused, there is no shock in the animal's body. Academic AABogomolets binds to changes in the electric charge of colloidal substances during shock, as the colloidal structure of blood and tissue proteins plays a key role in the formation of hemotransfusion shock.

Due to improper blood transfusion, the structure of the recipient and donor proteins changes and the deposition of the protein micelles leads to a severe impairment of the body's function. This theory unilaterally explains the formation of shock.

In the pathogenesis of hemotransfusion shock is manifested as a major change in the reflex activity of the organism. When blood is burned in groups that do not match, it stimulates the vascular receptor to produce multiple impulses, creating a short-term strong excitation in the nervous system and then braking large parts. It therefore disrupts blood circulation, respiration, metabolism and other physiological functions.

Osmotic resistance of erythrocytes. EOR is the resistance of red blood cells to hypotonic solutions, and there is a difference between minimum and maximum resistance.

Minimum resistance is defined as the level of hypotension in which gamma-resistant erythrocytes break down and hemolyze. At maximum resistance, all erythrocytes are broken down, and the concentration of the saline solution is taken into account when assessing the degree of hypotension.

The resistance of erythrocytes depends on their structure, the resistance of erythrocytes in the changed form is low and hemolysis occurs. In addition, the resistance of erythrocytes to hypotonic solutions depends on the layer of lipoid protein formed on the erythrocytes. Due to the lack of lipids and phosphorus in the newly released erythrocytes, they break down earlier than the old erythrocytes. The state of maximum resistance indicates that the bulk of the erythrocytes are mature erythrocytes. An increase in EOR is observed in mechanical jaundice, in cases of poisoning with hemolytic toxins, in pathological conditions accompanied by tissue breakdown. Increased osmotic resistance of erythrocytes is also associated with the deposition of cholesterol and broken down tissue proteins in the body of erythrocytes.

Decreased EOR occurs when starving, in hemolytic jaundice, and in other diseased states of the organism.

Hemolysis is the rupture of red blood cells and the release of hemoglobin into the surrounding fluids. Blood or erythrocytes become discolored after hemolysis. Hemolysis occurs in and outside the blood vessel. Some erythrocytes also break down due to their own death. If in the physiological state erythrocytes are broken down by splenic macrophages, in pathological cases the breakdown of erythrocytes also involves the macrophages of the liver, red marrow and other organs.

Causes of hemolysis include:

1. Infusion of erythrocytes into hypotonic solutions.
2. Heating of blood or erythrocytes 62-630.
2. Re-freeze and thaw the blood.
3. The effect of rays.
4. The effect of electric current.

The hemolytic effect of light energy occurs in the presence of photosensitizers such as eosin, fluoroacin and others.

Hemolytic effect is manifested by chemicals such as nitrite, nitrobenzene, ether, benzene, case and deoxycholate acids, and others. Under the influence of chemicals, the erythrocyte membrane breaks down, disrupting the binding of hemoglobin to erythrocyte strain. Hemolysis-causing substances include bee venom, chaen snake venom, tetanolysin, staphylolysin, and many other microbial toxins. The hemolytic effect of toxins is based on the hydrolysis and softening of the erythrocyte shell by phospholipids. Erythrocytes are also broken down by blood parasites. Specific immunoassays to erythrocytes may be the effect of hemolysins as the cause of hemolysis. Sometimes substances in the blood serum that are formed under the influence of tumors, radiation and other diseases break down erythrocytes to form autohemolysins.

From the breakdown of erythrocytes in the bloodstream, hemoglobin dissolves in blood plasma and is excreted in the urine. In the gradual breakdown of erythrocytes, hemoglobin and erythrocyte

fragments are captured by RES macrophages, resulting in complex changes to form the pigments bilirubin and hemosiderin.

Multiple breakdown of erythrocytes primarily increases the excretion of bilirubin by bile, which in turn increases stercobilin in the feces and excretes urobilinogen in the urine.

Iron released from erythrocyte breakdown is stored in liver and spleen macrophages. Here, after complex chemical changes, iron is released into the bloodstream and transported to the red marrow, where it is used in hemoglobin biosynthesis.

From the disruption of the normal change of hemoglobin, excess porphyrins-red violet-colored pigment is formed, which separates with the urine and turns the urine red. Due to the sensitization of porphyrin to light, its sensitivity to sunlight is increased. There are reports of parfirinuria as an inherited disease in Shortgorn pedigree cattle. Parfirinuria also occurs when poisoned with mercury, lead and sulfonamides.

Anemia is a decrease in hemoglobin and erythrocytes per unit volume of blood. In anemia, erythrocytes undergo qualitative changes, pathological forms of erythrocytes are formed, which differ in size, shape, saturation with hemoglobin. The total amount of blood in anemia is either reduced or maintained at normal.

Classification of anemia. One of the most common classifications of anemias is to classify them according to their origin. Depending on the origin of anemia is divided into pasthemorrhagic, hemolytic, elemental and infectious types.

1. Posthemorrhagic anemia occurs when there is a lot of blood loss in the body. Acute posthemorrhagic anemia occurs as a result of sudden multiple or multiple - multiple chronic blood loss. Bleeding from blood vessels due to injury, ulceration of the intestines and stomach from internal organs, tuberculosis of the lungs, bleeding in the nasal cavity, tumor growth, bleeding as a result of childbirth, etc. is formed.

Restoration of the blood component after blood loss Normal red blood cell count is restored in a few days to 2-3 weeks, depending on the amount of blood lost by the body. Recovery of hemoglobin after extensive blood loss occurs gradually. In the blood, hypochromic erythrocytes are formed polychromatophils, reticulocytes and normocytes. The color of the blood decreases, the amount of leukocytes increases. Chronic diseases, changes in the quality of nutrition, reduce the regenerative properties of red blood cells and cause severe anemia. Decreased red marrow activity leads to anisocytosis and paikilocytosis, and sometimes to the formation of extramedullary blood in the spleen, liver, lymph nodes.

Hemolytic-toxic anemia is caused by toxins that break down erythrocytes. Some substances break down erythrocytes, directly in the blood vessels, some break down blood cells and then break down in RES macrophages. In the origin of toxic anemias, the formation of blood and the violation of the reflex control of its breakdown are of great importance. does not cause anemia when administered.

In hemolytic anemia, bilirubin in the blood increases, urobilinogen is excreted in the urine, and sometimes free hemoglobin is also excreted. First of all, the color of the blood is suddenly higher, and undigested erythrocytes are absorbed into the body, absorbed. Blood formation is enhanced by strong breakdown of erythrocytes. In the blood there are large numbers of polychromatophiles, reticulocytes and sometimes normoblasts. The color index of the blood suddenly decreases. Due to the good regenerative properties of red marrow, the composition of the blood is quickly restored with the loss of toxic effects. In chronic hemolytic anemia, the blood-forming organ becomes tired, its activity weakens, and erythrocytes with various defects in the blood fall into the bloodstream, and anisocytosis and poikilocytosis are observed. The amount of erythrocytes in the blood decreases sharply.

3. Alimentary anemia is caused by a lack of vitamins, proteins, trace elements in the diet, cobalt and copper, ie substances involved in the synthesis of hemoglobin. Alimentary anemia has a hypochromic character and the blood color index is less than one. Alimentary anemia is observed in young animals, especially piglets. Alimentary anemia is caused by inability to assimilate nutrients well during diseases of the gastrointestinal tract.

a). Anemia caused by iron deficiency is caused by a disorder of iron metabolism in the body. In this type of anemia, not only is there a decrease in erythrocytes, but also a decrease in the amount of

hemoglobin. In severe anemias, anisocytosis and poikilocytosis occur. In pigs, iron deficiency in pigs resulted in the development of anemia in piglets at 1–6 weeks and up to 70% mortality.

b). Anemia caused by protein deficiency As a result of a lack of proteins in the diet or a decrease in their absorption, the synthesis of globulin protein is disrupted and hemoglobin is not formed.

4. Infectious anemia is caused by filtering viruses in horses and other ungulates. While some scientists explain the formation of this anemia as a direct breakdown of erythrocytes under the influence of viruses, others explain that the viruses are associated with causing red marrow hypofunction. The amount of erythrocytes in 1mm3 of blood of animals with infectious anemia is reduced by 1-2 million. Anisocytosis, poikilocytosis and other changes occur in the blood. In infectious anemia in the red marrow occurs the replacement of the yellow marrow with red marrow, the formation of extramedullary blood in the spleen, liver, lymph nodes.

Regenerative and oregenerative anemia occur depending on the functional state of the blood-forming organ.

In regenerative anemia, the regenerative properties of the blood-forming organs are well manifested. As a sign of regenerative status in the peripheral blood are formed hypochromic, polychromatophilic erythrocytes, reticulocytes, erythrocyte nucleus remnants (Jolie bodies and Cape rings), normoblasts. When strong regenerative properties are manifested, the yellow marrow turns into red marrow, and in the liver spleen, extromedular blood formation occurs in the lymph nodes. Such changes disrupt blood formation and are formed from cells of the embryonic period — megoloblasts, macrocytes. Oxygen deficiency is an intermediate product formed during anemia, as a cause of regenerative processes in the blood-forming organs.

Aregenerative or hypoplastic anemia results from fatigue of the blood-forming feature of the red marrow. In hypoplastic anemia, the red marrow loses its ability to form erythrocytes, young erythrocytes in the blood decrease, the red marrow turns into yellow marrow, and has a hypochromic character. Weakening of the blood-forming organ is observed during avitaminosis, infections (tuberculosis, paratuberculosis, infectious anemia, sepsis), strong toxins, radiation sickness. Under certain conditions, any anemia can progress to a type of hypoplastic anemia. In most cases of anemia, erythropoiesis is not impaired, but leukopoiesis is also impaired.

In organisms, the compensatory mechanisms in anemia change. The function of oxygen supply to the blood is weakened, a number of flexibility mechanisms are formed: accelerated respiration, increased blood circulation and blood formation. As the heart beats faster, blood circulation speeds up and more blood flows through the capillaries over time. Accelerated and deepened respiration increases the saturation of the blood with oxygen in the lungs, increasing the formation of broken erythrocytes in the blood-forming organs. Compensatory properties are associated with the ability of tissues to fully absorb oxygen from arterial blood.

In severe hemoglobin deficiency in anemia, normal gas exchange is ensured in animals due to the activities of compensatory mechanisms. But weak movements during anemia cause a lot of oxygen demand, accelerated breathing movements, and tachycardias. Acedosis develops when there is an increase in incompletely broken down intermediates in the blood.

Polycythemia - or polyglobulia (Greek poly poly, globulus-ball, kutos-cell) is an increase in the number of erythrocytes in the blood per unit volume. Polycythemia is divided into absolute and relative types. In relative (false) polycythemia, the fluid content of the blood decreases and the number of erythrocytes does not change. This type of polycythemia occurs when sweating, severe diarrhea, diabetes mellitus, severe isthmus, dehydration and other pathological processes. In relative polycythemia, the total amount of blood is often reduced or unchanged.

In absolute polycythemia, erythrocytes proliferate due to increased erythropoiesis. In most cases, absolute polycythemia serves as a resilience reaction in the absence of oxygen to the body. Lack of oxygen increases the flow of erythrocytes from blood depots and blood-forming organs into the bloodstream. Absolute polycythemia develops when external respiration is disrupted (pulmonary emphysema, when the upper airway narrows, O2 partial pressure decreases in atmospheric air), when blood circulation is disrupted. Polycythemia also occurs when poisoned with copper, phosphorus,

cobalt, arsenic. Polycythemia is a physiological condition in newborns, ie in the first days of life of calves erythrocytes in 1 mm3 of blood are 10.5 million. and a month later it dropped to 7.5 million.

Changes in white blood cells. Leukocytes, i.e. white blood cells, are formed in the red marrow, lymph nodes, and spleen. The stem cells that produce leukocytes are called hemocytoblasts, and the hemocytoblasts form myeloblasts, the primary cell of granular leukocytes in the red marrow. Lymphoblasts and monocytes are produced in the lymph node and spleen. In the blood of a healthy animal, there are many joint nuclei, and a small number of rod nuclei are found. Young neutrophils are not always present, and when blood-forming organs are tickled, rod nuclei proliferate, and in some cases myelocytes also occur.

Leukocytes include plasma cells, i.e., lymph nodes, spleen, and products of reticular and endothelial cells of the red marrow. Immune cells are formed due to the activity of plasma cells. During normal blood formation, plasma cells are found in the blood-forming organs, while in healthy animals, they are almost never found in the peripheral blood. The cytoplasm of plasma cells is stained dark orange, and the nucleus is round or oval in shape.

A leukoformula is a list of leukocyte types to determine the percentage of individual leukocyte species. In the leukoform of cattle, sheep and pigs, lymphocytes are abundant in the blood of horses, dogs and cats, and neutrophil leukocytes are abundant. White blood cells differ in type, and the leukoforms of young organisms are slightly different from those of older animals.

In determining the functional status of blood-forming organs, it is necessary to know not only the amount of leukocyte-forming organs, but also the absolute amount of leukocytes. The determination of the ratio of the main group of leukocytes in numbers is called leukocytic profile.

The main function of leukocytes is a protective function, i.e. phagocytosis. Leukocytes play an important role in the repair of damaged tissue, clearing the injured area of necrotic cells. Leukocytes produce a substance that stimulates regeneration, basophils and eosinophils are involved in neutralizing toxins. Quantitative changes in leukocytes are caused by an increase or decrease in leukopoiesis, as well as redistribution of blood in the blood vessels. As a result of dilation of blood vessels, blood flow slows down, leukocytes settle along the walls of blood vessels, and their amount in these blood vessels increases. Where blood vessels constrict and as a result blood flow accelerates, the amount of leukocytes in the blood decreases.

4.Myeloid, lymphoid leukemia and reticuloendotheliosis are distinguished depending on which part of the hematopoietic system is hyperplastic. Lymphoid leukemia is found in cattle, horses, and pigs, while myeloid leukemia is observed in dogs.

Myeloid leukemia or myelosis is characterized by hyperplasia of myeloid tissue. The yellow marrow turns into a red marrow, causing extromedular blood to form in the spleen, lymph nodes, liver, and sometimes other organs. Leukoblasts are more common in erythroblasts than erythroblasts. Myeloid leukemia is divided into leukemic and aleichemic types. In leukemic myelosis, the number of leukocytes in 1 mm3 of blood can be a hundred thousand or more. The main part of leukocytes, ie 90% and more, are granulocytes. The bulk of granulocytes are young cells, ie myelocytes, promyelocytes and myoblasts, and sometimes the number of unexposed eosinophils, basophils and erythroblasts also increases. In aleukemic leukemia, the number of leukocytes is increased around the norm and or in very small amounts. Examination of the leukoformula shows a strong rejuvenation of leukocytes. However, although their phagocytic properties are preserved, they are slightly lower than the phagocytic activity of mature neutrophils. In myeloid leukemia, the spleen becomes enlarged.

Some scientists attribute the formation of extromedular blood in leukemia to the introduction of myeloid cells into tissues and the formation of metastases, while others explain that the formation of extromedular blood is caused by the influence of etiological causes of leukemia on mesenchymal cells.

There are leukemic and aleukemic types of myeloid leukemia. In leukemic leukemia, the number of leukocytes in 1mm3 of blood reaches 100,000. The main part of leukocytes is granulocytes, which account for 90%. Granulocytes are composed of young cells - myelocytes, promyelocytes, sometimes non-myeloblastic eosinophils, basophils, erythroblasts. In aleukemic myelosis, the leukocytes in the blood increase normally or very little. In leukoform, young cells are weaker than phagocytosis in neutrophils, whose main part is phagocytic function (myelocytes, etc.).

During lymphoid leukemia or lymphodenosis, lymphoid tissue grows and is characterized by enlargement of the lymph nodes, spleen and liver. As leukemia develops, the myeloid tissue is replaced by lymphoid tissue in the red marrow. During leukemic lymphodenosis, the amount of white blood cells in 1 mm3 of blood reaches 1.5 million, and lymphocytes make up 98% of all leukocytes. In aleukemic lymphodenosis, the number of leukocytes is normal or partially increased, lymphocytosis develops in the leukocyte formula, and lymphoblasts are also found among the lymphocytes.

Reticuloendotheliosis is characterized by proliferation of reticular cells in the red bone marrow, spleen, lymph nodes, and liver. There are leukemic and aleukemic types of reticuloendotheliosis. In leukemic retiuloendotheliosis, there is a strong increase in monocytes in the blood. In acute leukemia the metabolism is disturbed, the productivity of the animals decreases, anemia develops and severe weight loss occurs, in chronic leukemia the animal seems to be healthy for a long time, the animal dies from malnutrition and other diseases.

Leukemia etiopathogenesis. At present, leukemia with all its symptoms is recognized as a pathological process specific to the inflammatory process. Symptoms related to the theory of blastomatosis of leukemia include:

1. The growth of hematopoietic tissue during leukemia is not differentiated like tumor cells.
2. Changes in metabolism during leukemia are similar to those in malignant tumors.
3. Carcinogens have leukogenic properties in the experiment.
4. The therapeutic effect is due to the same substances in leukemia and tumors. (M. X-rays, radioactive phosphorus, chemicals that affect cells).

In leukemia, the leukocytes are in such an atypical state that it is difficult to consider them as this or that blood-forming element. However, the process of phagocytosis is worse than in normal leukocytes. Leukemia differs from normal tumors in the formation and growth of blood in the blood-forming organs. In aleukemic leukemia, destructive symptoms characteristic of the growth of all tumors are observed.

The causes of leukemia and tumor formation are not yet fully understood. Chicken leukemia is caused by viruses. This has been studied in leukemia by sending cell-free filtrate to healthy chickens. All leukemias can be formed by injecting carcinogens. Leukemia is caused by long-term ionizing radiation in the body, the mechanism of action of which has not yet been determined.

Changes in blood plastics. Blood plastics play an important role in platelet coagulation and are a source of the enzyme thrombocytosis. Platelets are formed in large cells of the red marrow - megakaryocytes. Therefore, the factors that affect the red marrow affect the amount of blood platelets. A decrease in the amount of platelets in the blood is called thrombopenia, which causes a weakening of the blood clotting process. In thrombopenia, the retraction of the blood clot is weakened The blood clot is soft and does not provide a tight closure of the injured blood vessel.

The causes of thrombopenia are as follows:

1. Redistribution of platelets, ie accumulation of platelets in the blood vessels of the internal organs and a decrease in the peripheral blood vessels.
2. Weakening of platelet formation in the red marrow.
3. Strong breakdown of platelets in peripheral blood.

Thrombopenia in some infectious diseases is caused by physical, chemical causes, disruption of the activity of blood-forming organs or strong breakdown of platelets.

When thrombocytosis or an increase in the amount of platelets in the blood is cured of many infectious diseases, in myeloid leukemia, anemia is formed during the recovery of blood composition, and blood clotting is enhanced.

Simultaneously with the change in the number of platelets, a qualitative change occurs, the shape changes, does not wrinkle and undergoes other changes. The agglutination property of such blood plastics is lost, and blood flow and blood clot retraction are impaired.

Changes in blood coagulation. Blood coagulation is recognized as a three-phase process as explained on the basis of modern theories. The first phase is a complex biochemical process in which active thrombokinase is formed from active tissue thromboplastins and the action of blood platelets on serum proteins. From the inactive prothrombin enzyme in the second phase: active thrombin is formed

in the blood plasma. Calcium ion, active thrombokinase and plasma protein - globulin accelerator are involved in the activation of prothrombin. Prothrombin is formed in the liver in the presence of vitamin K. The liver is one of the main sites where fibrinogen is synthesized. In the third phase, fibrin is formed from the action of active thrombin on fibrinogen. As a result, fibrin filaments are formed and blood clots form. In the body, along with the blood coagulation system, there is also an anti-coagulation system, these substances are formed in the tissues and released into the blood under the control of the nervous system. Anti-coagulation systems include 1) heparin-liver physiologically active substance formed in the lungs and blood vessels, 2) fibrinolysin-plasmin, 3) protein substances that inhibit the formation of thrombin and thromboplastin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin.

The blood coagulation and anticoagulant system are two interconnected parts of the blood's coagulation system. Because these two systems are mutually balanced, the blood moves in a fluid state without clotting in the blood vessels.

Weakening of blood clotting. Weakening of blood clotting: 1) due to insufficient intake of vitamin K in the body or impaired synthesis of prothrombin and fibrinogens in pathological processes of the liver. 2) when there is a decrease in platelets in the blood - in thrombocytopenia. 3) decrease in calcium ions in the blood. 4) excessive development of the anticoagulant system in the body - heparin and others. 5) when anti-coagulants, ie substances that weaken blood clotting, are injected into the body.

When animal blood has a low coagulation property, a small mechanical injury can cause bleeding into the subcutaneous tissue, mucous membranes, muscles, and other tissues. The easiest bleeding occurs in the nose, lungs, intestines.

By treating the blood vessels with paraffin, if blood collects in the arteries, the blood becomes coagulated. A 5% sodium hydroxide solution of citric acid is often used to make the blood non-coagulating. Anticoagulants include dicoumarin and other anticoagulants extracted from the head of the leech. These substances stabilize the blood by inactivating thrombin. We can use the stabilizing properties of these substances by injecting them directly into the body or adding them to freshly drawn blood.

Acceleration of blood clotting. Accelerated blood clotting is associated with vascular injury. Blood platelets easily sink into the injured vascular wall, break down due to low resistance, and form active thromboplastin-thrombokinase. Blood coagulation can be formed by the strong breakdown of tissues by sending to the body extracts prepared from blood serum and organs. Increased blood coagulation after excessive blood loss is associated with the influx of many interstitial fluids rich in thromboplastin factor into the blood. Based on this mechanism, the delivery of calcium salts, multi-vitamin K, when hypertonic solutions are injected into the blood, increases blood coagulation. Increased blood clotting in the body can lead to thrombosis and embolism.

Changes in the biochemical composition of the blood. Minerals are ionized in the blood and are in a molecular state as well as in a state of binding to proteins from colloidal substances. Minerals are involved in blood osmotic pressure and other complex physicochemical processes. Minerals are not evenly distributed between the blood plasma and trace elements, the amount of calcium, potassium, sodium and other minerals in the blood of healthy animals is always kept the same, even when saline solutions are sent to the body.

Calcium. Ionized calcium is physiologically active, accounting for 45-55% of total calcium. Combined with non-ionized calcium mining proteins. The amount of calcium in the blood depends on the functional state of the autonomic nervous system. Calcium decreases when sympathetic nerve tone

decreases, and calcium increases in blood when parasympathetic nerve tone decreases. Calcium salts thicken cell and tissue membranes.

A sharp decrease in calcium levels is caused by a deficiency of glands near the thyroid gland and causes hypoproteinemia due to the fact that part of the calcium is bound to proteins. The amount of calcium in the blood is reduced in nephritic anemia congenital paresis. Decreased calcium intake increases vascular permeability, excitability of the CNS and peripheral nervous system. Calcium intake is also caused by impaired intestinal absorption in chronic diarrhea.

Potassium. In many animals, the amount of potassium in erythrocytes is higher than in plasma, and the amount of potassium in plasma increases when erythrocytes break down. Damage to erythrocytes causes the release of potassium from erythrocytes into plasma due to increased permeability without breaking them down. The amount of potassium in the serum increases in severe diseases when the tone of the parasympathetic nervous system increases, regardless of its nature. Potassium and calcium affect the excitability of the nervous system. Deficiency of potassium in the body leads to weakening of muscle activity.

Sodium. Occurs in the blood plasma mainly in the form of chlorides, partly biocarbonate and other salts. Chlorides are reduced in the blood when sweating, diarrhea, vomiting, weight loss, impaired intestinal permeability, kidney disease. Decreased chlorides affect osmotic pressure and increase the breakdown of tissue proteins, weakening the activity of the adrenal cortex. The amount of chlorides increases in the blood during kidney disease, ie nephritis. The onset of hyperchloremia is caused by increased pulmonary ventilation, as a result of which chlorine ions pass from the tissues into the blood.

Phosphorusoccurs in the form of organic and inorganic compounds. In animals, inorganic phosphorus in the blood is reduced in pregnancy, rickets and osteomalacia. Hyperphosphatemia is caused by fever, lack of oxygen, uremia, exposure to vitamin D and ultraviolet light, as well as a lack of glands under the thyroid gland.

Ironenters hemoglobin and occurs in the form of other compounds only in 2% of cases. Therefore, iron varies depending on the amount of hemoglobin. In anemia, iron in the blood is reduced. Blood contains trace elements such as iodine, bromine, fluorine, magnesium, copper, manganese and others. The amount of micronutrients in the blood is affected by the nervous and endocrine systems. Detection of micronutrients in the blood is important in the diagnosis of metabolic diseases.

Proteins and products of protein metabolism. Protein and its fractions are different in the blood of different animals. Some proteins combine with fats and carbohydrates to form double compounds - lipoproteins or glycoproteins. Although many proteins (e.g. enzymes) are present in very small amounts in the blood, they have very important physiological activity. Most of the blood plasma proteins are synthesized in the liver. Decreased total protein in the blood (hypoproteinemia) is caused by eating disorders (malnutrition, protein starvation). Causes of hypoproteinemia include urinary excretion of proteins, liver toxicity, excessive blood loss, severe degenerative diseases of animals (tuberculosis, malignant tumors, chronic purulent processes, etc.). In hypoproteinemia, mainly albumin function is reduced, while the globulin fraction is significantly reduced. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in

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Blood plasma increases globulins in infectious disease and starvation. After immunization, gamma globulins in the blood increase sharply. However, an increase in gamma globulins is not associated with an increase in antibody levels. An increase in non-specific gamma globulins in the blood and an increase in gamma globulins may be due to a decrease in specific antibodies, as AE Gurvich found. Decreased albumin fraction in the blood is observed in hepatitis and cirrhosis. Therefore, in patients with impaired liver function, the total amount of proteins in the blood plasma and some fractions are variable.

Residual nitrogen in the blood is the protein-free nitrogen of the blood or the nitrogenous substances that remain after the deposition of proteins in the blood is 20-40 mg%. Increased residual nitrogen in the blood (azotemia) is observed in disorders of renal, hepatic and intestinal permeability. The amount of residual nitrogen in the blood is 200 mg% and more when the renal excretory function is impaired. In azotemia associated with renal (retention) activity, an increase in the amount of residual nitrogen occurs due to urea.

In cachexia, leukemia, and infectious diseases, the accumulation of large amounts of nitrogen-fixing substances in the blood due to the breakdown of tissue proteins causes azotemia. In hepatitis, azotemia is caused by polypeptides, which can also lead to a decrease in the amount of urea in the blood. Such a change is observed in liver disease when the deamination of amino acids is impaired, the synthesis of urea is weakened, and the transfer of ammonia salts into the blood is increased.

Accumulation of uric acid in the blood is observed in disorders of purine metabolism, gout, diseases associated with tissue breakdown, and leukemia.

There are a certain amount of free amino acids in the blood, which are intermediate products of protein metabolism. An increase in the amount of free amino acids is caused by liver disease, ie severe atrophy, poisoning by carbon tetrachloride.

Carbohydrates and products of carbohydrate metabolism.

Blood contains products of glucose, glycogen, lactic acid and other carbohydrate metabolism. The amount of glucose in the erythrocytes of most species is lower than in plasma, and this is more pronounced in pigs. Most of the glycogen is found in leukocytes. An increase in the amount of glucose in the blood (hyperglycemia) occurs when consuming easily digestible carbohydrate foods (elemental hyperglycemia), when the regulation of carbohydrates through the nervous and endocrine systems is impaired. Hyperglycemia occurs when poisoned with physostigmine, pilocarpine and other substances that affect the nervous system. The origin of hyperglycemia is in the pathology of the endocrine system, ie in the hypofunction of the islets of the pancreas Langerhans, formed in inflammation and dystrophic changes of the liver. Decreased blood glucose (hypoglycemia) is observed in chronic insufficiency of nutrition, excessive infusion or delivery of insulin into the blood, hypofunction of the

adrenal hypo-thyroid gland. The manifestation of severe hypoglycemia is observed in patients with chronic cachexia.

An increase in the amount of lactic acid in the blood is observed in muscle work and pathological processes in the disruption of oxidative processes in the body, when there is a lot of blood loss, pulmonary edema, suffocation, the formation of malignant tumors. All the factors that increase the formation of lactic acid in the blood cause an increase in the amount of pyruvic acid in the blood.

Lipids. Neutral fats, lysine, cholesterol and their products are stored in the blood from lipids.

The amount of neutral fats in the blood increases during feeding. Pathological lipemia is observed at the onset of starvation, and the development of lipemia during starvation is associated with the excretion of fats from fat depots and transport to the liver. Lipemia in all cases results from the release of fats from fat depots when glycogen in the liver is depleted. Lipimia is caused by liver and kidney damage.

An increase in the amount of cholesterol in the blood is increased by food (elemental hypercholesterolemia), inflammation or dystrophic processes in the liver, leukemia, severe forms (forms) of tuberculosis, some infectious diseases, arteriosclerosis, pregnancy. Underlying pathological hypercholesterolemia is the excretion of excess cholesterol from the tissues, insufficient excretion of cholesterol from the liver and intestines, and impaired cholesterol oxidation.

Changes in the amount of phospholipids (lecithin) in the blood are caused by disorders of fat metabolism. An increase in phospholipids is associated with lipemia. An increase in ketone bodies in the blood (ketinemia, acetonemia) is observed when the body's carbohydrate intake decreases or carbohydrates are replenished in the diet at the expense of fats and proteins.

Due to the lack of carbohydrates, the breakdown of fats increases and many ketone bodies are formed. The main cause of ketonemia is a decrease in glycogen storage in the liver, during which the oxidation of fats in the liver is disrupted.

Changes in pigments. An increase in the amount of bilirubin in the blood (bilirubinemia) is caused by a strong breakdown of erythrocytes, a violation of the bile secretion function of the liver.

The formation of dissolved hemoglobin in the blood plasma is called hemoglobinemia. Causes of hemoglobinemia include: when erythrocytes are broken down by blood parasites or blood toxins, blood clots whose groups do not match. Formation of methemoglobin in the blood-met hemoglobinemia is formed by nitrates, nitrites, bertolet salt and other substances. Methemoglobin cannot perform the function of transporting oxygen to the blood, and the conversion of 20-40% of hemoglobin to methemoglobin results in oxygen deficiency.

When inhaled air (SO) contains carbon monoxide, carboxyhemoglobin is formed in the blood. The binding of hemoglobin to carbon monoxide is 300 times stronger than that of oxygen. Carboxyhemoglobin, like methemoglobin, cannot carry oxygen to the blood. Therefore, the presence of small amounts of carbon monoxide in the air we breathe also causes oxygen deficiency.

Changes in the amount of enzymes, hormones and vitamins in the blood and plasma are formed, and these changes are important in the diagnosis of diseases.

Changes in the physicochemical properties of blood.

THE SCIENCE OF ANIMAL PATHOPHYSIOLOGY AND THE HISTORY OF ITS DEVELOPMENT.

Plan:

1. The content, purpose, function and interrelation of science with other sciences.
2. History of the development of animal pathophysiology.

Main publications:

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Base phrases.

Animal pathophysiology general law, artificial model, K.Bernard, Mering, Minkovsky, Fleming, Peer Marie, biochemistry, clinical diagnostics, experiment, general, typical and specific pathophysiology, general nosology, etiology, pathogenesis, reactivity, inflammation, local circulation disorder, fever, tumor, hyper and hypobiotic processes, STZabelin, AMFilomafitskiy, VVPashutin, ABFoxt, GPSakharov, AFAndreev, VVVoronin, VANegovskiy, CMPavlenko, ADAdo, AAJuravel, AVABodniyskov, NVPodvisotskiy, ADAdo, AAJuravel, NVPodvisotskiy , LRPepelman, MPTushnov, VVKoropov VMVolkov, RXXaitov, RPPo'latov.

1. Animal pathophysiology is the study of changes in the body of sick animals, the causes, conditions, mechanisms and consequences of disease, or the physiology of diseased animals.

One of the main tasks of the science of animal pathophysiology is to reveal the general laws of the development of pathological processes, to study the causes and mechanisms of development and course and consequences of the disease.

The science of animal pathophysiology reveals the general laws of the mechanisms of development of diseases, opens the way to the essence of the disease, a clear diagnosis, prevention and treatment of the disease.

The science of animal pathophysiology is one of the most important disciplines in the field of veterinary medicine, which forms the worldview of the doctor (judge), scientifically substantiates the origin of diseases and creates new, modern methods to replace outdated, unconventional preventive measures and treatment processes.

Animal pathophysiology is an experimental science and consists of two words: Greek Pathos - disease, illness, logos - doctrine.

The main and main method of the science of animal pathophysiology is 'experiment'. This science seeks to teach in-depth, comprehensive study of various pathological processes, diseases and their artificial models, artificially using the method of experiments. It teaches the importance of various factors in the pathogenesis of the disease, the mechanisms of disease development, the consequences of the flow.

With the help of pathological experience, the necessary conditions are created to study the causes of diseases in the past, present and future, and this is important. In studying the glycogen-forming properties of the liver, K. Bernard studied the amount of carbohydrate in the blood that goes to the liver and is present in the blood vessels leaving the liver, and found that the blood leaving the liver is low in carbohydrates.

The glycogen-forming properties of the liver were also studied by Mering and Minkovsky, who observed an increase in the amount of glucose in the blood when they examined the blood by tying two pancreatic ducts, thereby demonstrating the importance of hormones in the body. In experiments, Peer Marie proved that hypofunction of the pituitary gland leads to stunting, and hyperfunction leads to acromegaly. American scientist Simones studied the occurrence of cachexia when the function of the pituitary gland is reduced. When the Russian scientist Lunin took two groups of mice and fed one group with artificial and the other group with natural milk, a few days later the artificial milk-fed mice lost weight, lost their growth and their hair fell out, and their skin began to change. .

Trying to determine the importance of vitamins, VVPashutin feeds rabbits with sauerkraut and observes that rabbits are susceptible to sinus disease, but cannot explain the mechanism of its development.

The hypothesis of vitamins was given in 1911 by Kazimir Funk, a Polish biochemist working in London. He isolated a white crystalline substance from rice bran that could cure the disease and called it a vitamin. Latin-Vita means life amine, a chemical compound that contains nitrogen. K.Funk believes that diseases such as scurvy, pellagra, rickets, and beriberi are caused by a lack of vitamins in the body. Studies in recent years have confirmed that most vitamins do not contain nitrogen. Nitrogen-free vitamins include A, D, E, K, C. In the past, experiments have been conducted in a short period of time, using sharp experiments.

Therefore, the experiment was developed in the hands of IPPavlov, who conducted it using chronic methods.

IPPavlov spent 10 years in the SPBotkin laboratory, where the effects of caffeine, camphor, bromine on blood vessels, in particular, affecting the heart nerves, changes in blood pressure, changes in blood pressure in dogs under the influence of drugs, suturing the carotid artery in dogs , learns.

For 20 years, IPPavlov improved the methods of fistula in the physiology of the digestive system. 'rganadi. To study the role of the nervous system in digestion, the method of esophagotomy of animals explains the reflex separation of gastric juice as a result of "lying" feeding. Based on these methods, creates a diet.

IPPavlov devoted 35 years of his life to the study of mental activity and behavior of humans and animals.

The pathophysiologist uses pathological experimentation to study the causes of the disease, determine its course, find measures to prevent the disease, and develop ways and means of treating the disease, which in turn helps the practice. In particular, in the 18th century, when French wines began to turn into vinegar, IPPaster developed a method of washing and disinfecting wine containers in boiling water. When silkworm disease occurs, it is recommended that the silkworm storage rooms be cleaned of contaminants, proving that silkworm disease is caused by microorganisms.

When Louis Pasteur grows bacteria that cause cholera (malaria) and the thermostat door is accidentally left open, a few days later, he observes that the growth of cholera microbes is weakened and when he injects this microbial wash into the chickens, the chickens do not get sick. Thus, a vaccination method is created. British scientist Fleming planted in petri dishes to study the disease-causing properties of streptococcal microbes, and when the surface was left open, fungi fell on the planted microbe, partially killing the microbes and, based on this, the first antibiotic, penicillin, was created. So the importance of experiments is significant. On the importance of experiments, IPPavlov recommends paying attention to the following two important processes:

1. Observations should be given close attention;

2.He says we study nature by focusing on the experimental method.

The French scientist Couve says that by the method of observation we hear nature and in practice we force the opening and submission of nature.

Three different problems are studied in the science of animal pathophysiology:

1.Nosology is the general doctrine of disease. In nosology, the doctor faces two different issues: one is why the disease occurs and what is the mechanism of its development (etiology, pathogenesis)? In the origin of the disease is studied the importance of the type, breed, sex, heredity and constitution of the animal, as well as the characteristics of disease resistance - reactivity.

2.The general typical cases that occur in all diseases and underlie all diseases or are observed in their origin are studied:

a). Local circulatory disorders;

b). Inflammation;

v). Fever;

g). Hyper and hypobioses.

In the special pathophysiology part of the science of animal pathophysiology teaches pathologies of organs or systems: blood, blood circulation, respiration, digestion, liver, digestive organs, endocrine glands and nervous system.

Later he began to teach pathophysiology and normal physiology AMFilomafitsky (Head of the Department of Physiology, Moscow University). Since he was not divided into in-depth knowledge at the time, he taught only some of the symptoms of the disease, without knowing the course of the disease. It teaches the origin of diseases by linking them to divine power. Therefore, AMFilofitsky begins to study a number of diseases in practice, as it is expedient to observe and study the disease. For example: the importance of the nervous system in cough, the method of blood transfusion, transfusion of fibrin-deficient blood, reviving dogs, and writing a work in this area, he has not lost its value so far. Nutritional chemistry is studied in the laboratory of AMFilomafitsky, and in 1842 in this laboratory VABasov developed a method of inserting a tube-fistula in the stomach of a dog. AMFilomafitsky studies various pathological processes in Russia under a microscope. For example: erythrocytes from the shaped elements of the blood, observed changes in urine output during the disease. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science.

VVPashutin, based on several experiments, knowing the importance of the nervous system, opposes R. Virkhov's cell pathology and explains that the processes taking place in the cells depend on the nervous system. Experimental observation of the formation of various pathological processes in the body as a result of lack of various substances, the study of the mechanism of origin of scurvy, feeding rabbits with sauerkraut. As a result, it is concluded that the disease is caused by a lack of any additional nutrients to the organisms. Lunin then justifies the lack of vitamins. That is why VVPashutin is called the gift-pioneer of the doctrine of vitamins.

VVPashutin organizes the largest school of pathophysiologists in Russia. One of his students was MPAlbitsky (after Pashutin he was the head of the department), AVReprov was the head of the physiology department at the Khorkov Medical Institute, X-ray exposure, endocryology. He founded an independent school of pathophysiologists at the Kharkiv Medical Institute, where he studied the pathology of gas, heat, metabolism and endocrine systems from his students DEAlperin, SMLeytes and others. Academician ADTimofeevsky worked on tumors and studied whether tumors can be grown under artificial conditions. It is a state award winner for growing large tumors from a single cell in vivo and in vitro (inside and outside the body). Lunin works in the field of vitamins. AP Likhachev works in the field of gas exchange. VVPashutin died of a heart attack in 1901 while working as the rector of the Academy of Medical Surgery.

2- The School of Animal Pathophysiology was founded at the University of Moscow under the direction of Alexander Bogdanovich Foxt (1848-1930), a student of AIPolunin. It studies the pathological processes occurring in organ tissues, including: lungs, heart system. Creates a model of artificial pores of the heart and studies it in detail. He studies the formation of constipation in the lungs

and heart in cardiovascular pathology, pulmonary, cardiac dysfunction. Professor Govril Petrovich Sakharov from the ABFoxt laboratory in the field of allergy and endocrinology, AI Talyansev develops methods of peripheral circulatory pathology, VVVoronin inflammation, AFAndreev clinical death and general resuscitation of the organism. VANegovsky studied animal pathophysiology of the cardiovascular system, on this basis he created a complex method of resurrection. GPSakharov and his students SMPavlenko and AAJuravel worked in the field of reactivity, immunology and endocrinology.

3- The School of Animal Pathophysiology in Kiev and Odessa was founded by Vladimir Valeryanovich Podvesotsky (1857-1913), who developed the humoral theory of immunity, a parasitic theory in the field of tumors. He worked on the regeneration process. He has written a textbook on animal pathophysiology and has published it in several languages. He published a journal, The Archive of Pathology and Medicine, to promote the science of animal pathophysiology. His students are LATarasevich and ITSavchenko, academician AABogomolets and others. They studied the problems of immunology, reactivity of the organism, endocrinology, and they always worked under the direction of II Mechnikov. LATarasevich and IT Savchenko worked on agglutinin, precipitate, antibodies in France at the suggestion of IIMechnikov.

Academician AABogomolets works in the field of animal pathophysiology, studying the role of reactivity in pathology, its relationship to endocrine management. He was born in 1881 in Petropavlovsk Prison and died in 1946. His mother was imprisoned for being a member of Russia's "southern liberation group."

Academician AABogomolets is the President of the Ukrainian Academy of Sciences and the First Deputy Chairman of the Presidium of the Supreme Soviet of Ukraine. Pathophysiologist-pathologist since 1924. By developing the pathophysiology of animals, he created the original pathological doctrine in medicine, which is called the physiological system of connective tissue. In addition to the supporting function of the connective tissue, it performs a trophic function and a plastic-building function. As it is composed of RES cells, it enhances phagocytosis and antibody production. Improves connective tissue function using antiretroviral cytotoxic serum. It was actively used in the treatment of many diseases during World War II. Academician AABogomolets Director of the All-Union Blood Transfusion Institute, developed a method of conserving blood (the first among physicians to be awarded the title of sos. labor hero). He identified 4 different types of constitutions depending on the nature of the connective tissue and observed more or less common diseases, depending on these constitutions. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize.

Academician IISirotin worked on the field of acclimatization of the organism and the reactivity of the organism.

Academician REKovetsy studied the origin of tumors and the characteristics of their development in different conditions, the course of metabolism in tumors.

Academic ADAdo has worked on allergic diseases, anaphylaxis, lung disease, and has written a textbook on Animal Pathophysiology.

The new schools of animal pathophysiology were headed by well-known scientists LATaraseevich, AVReprev, ESLondon, AABogomolets SSKholatov, GPSakharov, NNAnichkov, ADSperansky.

Academician NNAnichkov (1885-1965) studied in depth the pathophysiology of the cardiovascular system, the involvement of RES cells in pathological processes and the mechanisms of origin of arteriosclerosis in the Department of Pathophysiology, Pathanatomy of the Military Medical Academy.

Experiments show that indifferent influencers play an important role in the development of diseases. For example, if dogs are injected with apomorphine for 15 days and supported with light, in the following days only the lighting of the lamp causes them to vomit reflexively. He suggested that organs could not be studied in isolation from the body, and that systematic scientific work should be carried out. He founded a large school of pathophysiologists, and even today scientists from the ADSperansky school are actively working in research institutes and universities.

IIRavich, the founder of veterinary pathophysiology, worked in the veterinary department of the Academy of Medical Surgery in St. Petersburg, critically examining Virkhov's cell theory and acknowledging the importance of the nervous system in the origin of the disease. He wrote a textbook on general zoopathology and lectured to students on the subject.

Academician MPTushnov (1879-1935), Head of the Department of Pathophysiology of the Kazan Veterinary Institute, created an original drug in pathophysiology, the lysates of which are the products of the decomposition of various organs. For example, muscle lysates are called myolysates, and when animals are released when they are tired, their ability to work is restored, mammolysates are prepared from the udder and increase the amount of milk, and ovariolysates accelerate the maturation of egg cells. Lysates are now the most common and widespread type - biostimulants. They are used in the growth and development of young animals, increase productivity and treat many diseases. Biostimulants are widely used in fattening. Including, Chlorella, which is found in billions of water, has been used to enhance productivity by enhancing all the processes that take place in animals. Currently, there are more than 45 departments of veterinary pathophysiology in veterinary institutes and faculties of the CIS countries, which are studying the effects of biostimulants on the characteristics of the organism. Most research veterinary institutes are studying the effects of biostimulants on the body's reactivity, metabolism and neuro-endocrine control processes.

The contribution of the French scientist Claude Bernard (1813-1878) in the development of the science of animal pathophysiology was significant. K. Bernard's work is studied in two periods:

The first period involved 20 years of normal physiology, proving the liver’s glycogen production function and determining its reflex mechanism. The origin of diabetes in the body proves that it is associated with dysfunction of the CNS. Demonstrates the importance of pancreatic juice and bile in the breakdown of nutrients, as well as observed an increase in body temperature. Blood and lymph determine the organization of the internal environment of the body and determine vital processes.

The second period. He has been working in experimental physiology for 10 years. It studies the importance and function of various nerve fibers in the body, the electrical properties of nerve and muscle tissue, the properties of blood, and the effects of SO2 on the body. Proves a violation of saliva production from salivary glands. A substance called Curare affects the endocrine glands and observes a decrease in the secretory process. He studied various pathological processes of the respiratory system and wrote more than 180 scientific sources, which consist of 18 volumes. K. Bernard did a lot of work despite experiencing great difficulties. He teaches that the processes that take place in the body depend on the vital force, and that that force is random.

IPPavlov says of K. Bernard, "K. Bernard is a scientist who thought broadly and deeply in his mind, generalized physiology, experimental physiology, and experimental therapy as a whole, or combined the achievements of physiology with practice."

The famous chemist Dumas says, "K. Bernard is not only a physiologist, but he is a physiologist."

IPPavlov's doctrine is important in the development of animal pathophysiology. Prior to IPPavlov, observations were made in pathophysiology using analytical methods. Diseases of isolated organs, their integral parts have not been studied with attention to the living conditions of the animal, changes in the external environment and other related connections. IPPavlov, on the other hand, pays

great attention to experimental scientific work and observes changes in body systems in healthy organisms in chronic experiments. According to IPPavlov's theory of nervousness, it is emphasized that any pathological processes in complex organisms are carried out with the participation of the nervous system, in particular, with the participation of higher nervous activity.

The organization and development of the science of animal pathophysiology in Uzbekistan was associated with the formation of the former Soviet Union, which began with the establishment of universities and research institutes in accordance with the decree of the Soviet government. As a result, the medical faculty of the Central Asian State University was established in Tashkent, which was later transformed into Tashkent State University, and intensive work in this area began. In 1921, the first department of "General Pathology" was established at Tashkent State University, which was later renamed the Department of Animal Pathophysiology. The first departments of pathophysiology were established in Samarkand in 1930, in Andijan in 1957, and in 1972 at the Central Asian Institute of Pediatrics.

At the Uzbek State Agricultural Institute in Samarkand, Farkhodi first studied veterinary pathophysiology, and from 1936, the head of the department, Associate Professor Vladimir Valerianovich Volkov. VVVolkov was an encyclopedic lecturer, a skilled experimenter, an excellent pedagogue-coach. VVVolkov was the initiator and organizer of several original scientific works with the staff of the department:

1. The causes and mechanisms of development of allergies and anaphylaxis in astrakhan sheep and goats in hot conditions;

2.Study the causes and mechanisms of development of pneumonia in sheep and goats during the summer months;

3. He has done a lot of research in the field of pathology of the region, the causes of the disease "Suyluk" in horses, the mechanism of its development and the development of methods for its detection. Today, the disease is found in humans and animals and is called trichodesmatoxicosis. In this field NXShevchenko and FIIbodullaev defended their doctoral dissertations and supervised several candidate dissertations.

4. The study of the enhancing effect of cytotoxins formed in tissues on the immunological properties of the organism of various laboratory animals (accelerated formation of antibodies to paratyphoid and colibotseliosis strains).

5. A detailed study of the effects of the parasympathetic division of the autonomic nervous system on the organism of experimental animals.

6. He made a great contribution to the training of a large number of highly qualified personnel. After the untimely death of VVVolkov in 1953, the department was headed by Associate Professor Anton Ivanovich Yarmashkeevich.

Extensive development of scientific work carried out at the department, mainly since 1961 under the leadership of Associate Professor, now Professor Ruzi Haitovich Haitov. By this time, the staff of the department sent different amounts of extracts from the liver, spleen and other parenchymal organs to healthy and sick animals, depending on the timing of their delivery, studied the mechanism of their action and developed a number of recommendations. In the Department of Animal Physiology and Pathophysiology, tissue feeding of animal feeds has proven to have a positive effect on the growth and development of the organism and the treatment and prevention of various diseases.

Having studied the effects of many drugs against helminthiasis, a number of recommendations have been developed. The genetic features of natural immunity, especially in karakul sheep and lambs of different colors, have been extensively studied and are still being studied. In this area, Associate Professor ADDushanov developed a synthetic vaccine, which gave good results, and Associate Professor MAAbdullaev in collaboration with the senior lecturer of the department RFRuzikulov conducts important research. Under the leadership of Professor RXKhaitov«Veterinary basics»Volume 1-2, 1972, RHHaitov and A.Dushanov on "Animal Physiology" in 1975, RHHaitov and Associate Professor MA Abdullaev on "Animal Pathophysiology of Agricultural Animals" in 1980 in Uzbek, a number of manuals, He has published more than 400 scientific articles in various collections, scientific collections of universities, research institutes, international and CIS congresses and conferences. Under

the direct supervision of scientists of the department 10 doctoral and 342 candidate dissertations were defended in specialized scientific councils. Researchers of the department have been writing reviews and defending PhD and doctoral dissertations in many fields of physiology. And so, In Uzbekistan, pathophysiologists study the theoretical and practical processes of modern veterinary and medical science at the Department of Pathophysiology of the Veterinary Faculty of Samarkand Agricultural Institute, the Uzbek Veterinary Research Institute, pathophysiology laboratories of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad.

In order to strengthen the study of pathophysiology, the "Society of Pathophysiologists of Uzbekistan" was established, which includes more than 100 pathophysiologists. Pathophysiology and research work have been carried out in cooperation with veterinary institutes in Moscow, St. Petersburg, Kiev, Kazan, Almaty, Yerevan, and are still connected. As a confirmation of this strong unity, the fact that the 2nd pathophysiologists' session was held in 1972 in Tashkent is a proof of our opinion.

QUESTIONS FOR CONTROL

5. What is the science of animal pathophysiology?
6. Understand the functions of the science of animal pathophysiology?
7. What is the main method of animal pathophysiology?
8. Explain the importance of experiments in animal pathophysiology?

NOSOLOGY

Plan:

1. The concept of health (norm) and disease.
2. The concept of pathological reaction, pathological process and pathological conditions.
3. Description of the disease, types, course, restoration of vital processes in the body - resuscitation.
4. Anabiosis: seasonal and latergic sleep.

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BASIC EXPRESSIONS.

Animism, evil spirit, divine or spiritual power, pneumonia of life, Archaea, humoral, Hippocrates, solid, Democritus, cellular, R. Virkhov, Golen, Sels, Avicenna, yatrophysics, iatrochemical, Paracelsus, vivisection, nervousness, norm, disease, contagious , infectious, organ system, acute, moderately acute, recovery and death, symptoms, acute, chronic, incubation, latent, prodramol, period of clinical manifestations, outcome, sanogenesis, relapsing , death, anabiosis, secondary sleep, prophylaxis.

1.Information about the disease has been of interest to people since ancient times. Because science and enlightenment did not develop in the primitive community system, and people did not know the origin of natural phenomena, they thought only about the visible and the invisible. That is why the organism has been described as composed of mythical things found in nature, such as soil, air, water, wood (metal), and fire. Illness, on the other hand, was interpreted as being caused by an invisible divine (supernatural force) or "SPIRIT" - anima. This current is called the "ANIMISM" current or theory, and it is a picture that all diseases are invoked by this supernatural force, the evil spirit. Talented physicians began to appear in Greece 4-5 thousand years BC, who wrote down what they knew, what they asked someone, their observations on the patient, and bequeathed this knowledge to their descendants. As a result, medical science began to develop slowly. For example, they recorded discharge from the mouth, nose, and ears in various diseases, fever, foul odors, and so on. Later in Greece, doctors explained that a living organism was composed of 4 different fluids in addition to 5 different elements (blood, mucus, black and yellow grass). Thus, the current that explains health and disease with these four different fluid properties is called the Humoral Flow or Theory. So, if the fluids are normally mixed properly, health is a sign of health, and this condition is called krazia or krazis. If, for some reason, the ratio of fluids is disturbed or the juices are contaminated, improper mixing, the disease can lead to dyscrasia or**«Discrasion»**The founder of this movement is the Greek scientist Hippocrates, who lived in the 4th-5th centuries BC.

Hippocrates was an observer, a disease-seeker, a traveling physician, who always traveled from village to village, making many observations on patients, studying the symptoms, various features, currents, and consequences of many diseases, and writing dozens of works. The role of the external environment in the origin of diseases, with great emphasis on cleanliness, developed methods of diagnosis and treatment of many diseases. He developed the laws of medicine, and in medicine there is the Hippocratic oath in medicine. The teachings of Hippocrates have been proven to be true for centuries and even now, and his works have not lost their value.

In addition to diseases, Hippocrates also tried to create constitutions of human temperaments, which included four different temperaments: choleric (yellow grass), melancholic (black grass), sanguine (blood), and phlegmatic (mucous fluid). 'p or less depending on.

The contemporary philosopher Democritus of Hippocrates also developed a theory of diseases, which he called the solid (atomic, particle) theory, which explains that diseases are caused by changes in the spacing of atomic particles in the body. This theory explains that the disease is caused by the narrowing or widening and thinning of the spacing of the particles. At the same time, idealistic schools of thought have sprung up in Greece, claiming that diseases are called by divine power, explaining that organ function, organ diseases, and their causes depend on a particular pneumonia of life. According to

Plato, Aristotle explained that there are three kinds of divine or spiritual power that govern the lives of people and animals:

1. Spiritual power is located in the brain and controls the mental function of people.

2. The spirit of the animal is located in the heart and controls the movement and warmth of the animal.

3. Explains that the spirit of the plant is located in the liver and regulates digestion.

They explain that they believe that the causes of diseases are not in the external environment, but in the mental origin. At the beginning of the twentieth century, knowledge of the disease was developed by Roman physicians Galen and Sels, who, in addition to the three zinc origins, based their humoral flow on explaining that diseases often resulted from the breakdown of juices, distinguishing between hot and cold discrasions. developed treatment options. Based on the symptoms of the disease, they observed four specific symptoms of the disease: redness, edema, edema, pain, and these changes, which lead to dysfunction, called functio laesa. . Galen introduces the vivisection method into science.

After Galen, our compatriot was the famous scientist and philosopher Abu Ali Ibn Sino (Avicenna), who made a great contribution to the development of medicine. He was born in 980 in the village of Afshona, Romitan district of Bukhara region and died in 1037 in Hamadan. In 1980, Avicenna's 1000th anniversary was celebrated and her works were published. He wrote more than 300 works in various fields, especially in the field of medicine, and in 1020 wrote a book on the laws of medicine. It consists of 6 books in 5 volumes:

1. The book is devoted to the anatomy, physiology, causes, appearance, general treatment of diseases. Attention was paid to nutrition, health, deportation, vomiting, and blood transfusions.

2. The book describes more than 800 drugs derived from plants and animals.

3. The book is about diseases from head to toe, this book is dedicated to specific pathology and therapy.

4. The book deals with fever, various tumors, rashes, wounds, burns, bone fractures and dislocations, nerve injuries, injuries to the skull, chest, spine and limbs, poisons and poisonings - toxicology, makeup - is dedicated to keeping people beautiful. Recommended remedies against hair loss, obesity or weight loss. He wrote about rabies, smallpox, measles, leprosy, and plague.

5. The book describes the methods of preparation and use of drugs.

Avicenna's book, The Laws of Medicine, pays great attention to the methods of observation and experimentation in the study of diseases, and widely uses this method on various diseases. developed He identified many diseases, developed treatment methods, studied urinary incontinence, urinary tract infections, worm diseases, pulse heart disease.

In his multifaceted scientific work, Avicenna concluded that diseases must have had invisible causes, not divine powers, and that they were now identified as microorganisms.

Avicenna studied in detail the wounds, lung diseases, diabetes, plague, cholera, smallpox, leprosy, tuberculosis (tuberculosis) and many other diseases, especially in the origin of the disease. , boiled, proved that it is important to follow hygiene. He studied the effects of many drugs and found that mercury is important in diseases such as gonorrhea and syphilis. It has been proven that following a meal plan-diet is important in diseases. Although he did not know the functioning of the nervous system, he thought about the nervous system, that is, tied the sheep to the wolf, and observed that a few weeks later the sheep became frightened.

Avicenna's work on TIB laws has been reprinted 25 to 30 times in Europe and Asia, and is still being published today, and has served as a guide for physicians. By the 14th and 15th centuries, Copernicus, a Polish scientist, described the movements of the planets in the sky, Giordano Bruno's rotation of the earth around the sun, the Spanish Servetus's small circulatory system, and Leonardo-Da Vinci's anatomical tracts. V. Garvey discovers a large circulatory system based on his experiments on rabbits and dogs.

By the fifteenth century, a new direction in medicine, the iatrochemical and iatrophysical currents, began to emerge, meaning Iatros-physician.

The chemist Paracelsus conducted many experiments to prove the structure of the organism, the need for chemical elements to survive, the importance of mercury, matches, steel, iron and other

elements in the health or illness of the organism. concludes that it contributes, and explains that when archaea get angry, they cause disease without releasing these elements into the body.

Iatrophysicists connect the organs of the body to the parts of a machine and pump the heart, explaining health and disease according to the laws of physics and mechanics.

In the XV1-XV11 centuries, the pathological-anatomical direction developed, and Morgani, Bish, and others began to study the body structure of animals and humans. In 1543, the Italian scientist A. Vezali began to study the structure of the body by tearing apart the bodies. 1640 Descartes wrote the reflex doctrine, 1660 Malpighi lens using the lens, renal capillaries, liver, spleen, skin structure, erythrocytes, 1674 Levenguk lens sperm movement. Morgan and Bish wrote about the changes that occur in different organs in different diseases, which led to the development of the study of pathological processes.

This means that the external environment has had two different effects on the organism over a long period of evolution, and that the organism has become accustomed to these favorable and unfavorable effects, adapted and balanced. -slowly studied and adapted, these effects are called daily or physiological, adequate effects. The processes that take place under the influence of these influences are called physiological processes and are called the norm, abbreviated for short. The second type of effects are often referred to as sudden, strong, sudden, adverse effects, which are called harmful or disease-causing, inadequate effects, and the processes that take place under the influence of these effects are called pathological processes.

Norm or health is a set of influences, conditions, adapting to their currents in a certain period of time, making them suitable for life, necessary or physiological effects, and the processes that take place and develop under their influence. called normal processes. Norma is a process that takes place in a period of stagnation, when the organism is calm and peaceful.

1.Norma-Sergey Petrovich Botkincha stagnation of life processes
is the sum.

2. Norma-Ivan Mikhailovich Sechenov and Claude Bernard describe the organism
with the balance of the external environment.

3. Norma-Victor Vasilevich Pashutin described the structure of the organism and
is said to harmonize its functions.

4.Norma-Vladimir Valerianovich Podvisosky to the conditions of our body

The structure of normative organ systems, the state in which they function without disruption. In real life, the norm is a relatively stable, changeable situation, because the absolute norm does not exist in real life. For example: consider pulse, temperature, respiration.

When one wants to study a disease, one must study it by comparing it with the norm. Both disease and health are ongoing processes in the body, which differ from each other in quantitative and qualitative changes. At the heart of both processes are two opposing processes of assimilation and dissimilation. It is impossible to know the exact time of onset of the disease, but it can be determined only by the symptoms that appear at a certain stage of development. For example, sleep is caused by fatigue as a result of overwork, which is considered a normal physiological state of the body, but in some severe infectious diseases, drowsiness also occurs, indicating a disease of the body: anthrax, typhoid, diabetes , tuberculosis and others.

1.SPBotkin described the disease as a disorder of the vital processes of the organism.

2.IMSechenov and K.Bernar described the disease as a violation of the balance of the external environment in contact with the organism.

5. VVPashutin explains the disease as a violation of the harmony of the structure and function of the organism. These descriptions of the disease provide insights into unilateral changes in the disease, ignoring various complex quantitative and qualitative changes and active processes during the course of the disease. Therefore, these definitions do not fully describe the diseases.

6. In an attempt to fully express the disease, IPPavlov proposed the following definition: a disease is an encounter of an organism with an awkward, pathogenic, gross cause and condition that affects it suddenly, suddenly, collision, ie mechanical shock, crushing, injury, exposure to chemical, physical influences or attack by microorganisms, this encounter is the beginning of a struggle between the

organism and the cause , by activating all defense mechanisms against, removing pathogenic causes, cleared or enzymes, phagocytes, Acute flow diseases - from a few minutes, hours to several weeks: For example: infectious and parasitic diseases.

2. Moderate acute flow illnesses — from a few weeks to several months.

Chronic recurrent diseases are those that last for months or years, most of which are non-communicable and non-infectious.

Diseases occur in several stages as they develop in the body.

a). An incubation or latent or latent period is the time that elapses between the onset of the disease and the onset of the first symptoms of the disease. This period can range from a few minutes to a few hours, weeks, months, and even years. Tuberculosis, brucellosis, non-communicable diseases, leprosy, AIDS and others.

b). The prodromal or disease-reporting period has its own characteristics, during which general symptoms for the disease appear. For example: increase in body temperature, decrease in appetite, heart rate, rapid breathing, etc.

v). Outbreaks appear to be exacerbated during clinical trials.

g). The consequences of diseases are twofold: the animal is either cured of the disease, or the sick animal dies.

1.Diseases spread throughout the body - per kontinuitatem. As the disease progresses, one organ spreads due to adhesions to the other organ. For example, inflammation of the oral cavity continues to spread to the red intestine, then to the stomach, intestines, and so on.

2.The disease is spread by means of friction, adhesions - per kontiguitatem. Pulmonary pneumonia to the pleura and pericarditis - myocarditis, liver - stomach, etc.

The disease is transmitted through the blood and lymph - permestastazine. Many microorganisms are spread through the blood and lymph.

3. Diseases are transmitted through the nervous system - per nervorum, through nerve fibers, stolbnyak - congestion, botulism, polio and other diseases.

4. Diseases are spread by secretions, saliva, sweat, urine and feces.

Intermittent course of illness is a period of illness that is sometimes mild and sometimes severe.

The complete recovery of the body from disease is called sanogenesis. The consequences of the disease are of two types:

a). The body recovers from the disease.

b). The disease ends in death.

3. There are two types of recovery:

a). The body recovers completely from the disease.

b). The body recovers from the disease.

Recovery comes in two different ways: simple and complex. Simple ways of recovery are carried out by revealing various reflexes. For example: reflex agitation, excessive salivation, wiping tears, vomiting, sweating, coughing, diarrhea, excessive urination and excretion, tickling of the nervous system, and others

In complex treatment, the body is decontaminated by complex processes using barrier barriers, RES organs - liver, spleen, lymph nodes, red marrow, leukocytes, especially T and B lymphocytes, antibodies, etc. the cause is removed, then partially or completely repaired as a result of the recovery process. Restitution is called ad integrum if the body is completely cured of the disease. Sometimes the body can recover from the disease and recur, and the body can be severely damaged, and this is called a lytic transition to a critical and mild course.

3. Diseases can lead to dysfunction of the body without complete recovery. When the body's ability to heal is completely reduced, the body dies from the disease if the doctor's treatment does not help.

3.Death - mortis, morbi -characterized by the cessation of the continuous process of assimilation and dissimilation in the body and the cessation of heart function and respiration.

There are two types of death depending on their origin:

1. Natural or physiological death.

2. Death due to disease or pathological condition.

If 100% of all deaths are considered, only 2% of them are natural deaths and the remaining 98% are deaths due to diseases.

The doctrine that explains the formation of death is called tanatogenesis. Death occurs in several stages and is called the terminal state, they are:

1. Agony-pre-death convulsions: (consisting of peripoganal and oganal period).
2. Clinical death.
3. Biological death

As a result of death, the following changes occur in the corpse:

1.The body cools - algar mortis drops from 10 in the first days and cools to 0.20 on the second day. Of course, these changes are due to environmental changes.

2. The appearance of spots on the body - livoris mortis on the side on which the animal is lying, more spots appear and look good in hairless, unpigmented areas.

3. Hardening of the body - rigor mortis solidification of colloidal substances. Hardening begins after 8-10 hours and goes from head to toe.

4.Decomposition of the body - maceration or autolysis is formed under the influence of putrefactive and microorganisms from the external environment in the body, and the carcass begins to smell foul. If these bacteria are not present in the body, the body will become waxy.

Observations show that the animal continues to live in organs and tissues for some time after death. For example: nails, hair, hair, growth, movement of the stomach, intestinal muscles, contractions and other signs are observed. Much work has been done on the possibility of resurrecting the organism at the time of death. This condition is called resuscitation. It has now been discovered and proven that it is possible to resurrect organisms that have died by accident, and that people and animals who have died from various traumas, excessive blood loss, suffocation during anesthesia, electric trauma, various tragic events is being resurrected. Kulyabko, a professor of physiology at Tomsk University, was the first in this field in 1902.

From 1912 to 1919, the American physiologist Karel was able to use a burdock chicken heart under artificial conditions.

In the laboratory, Academician Kravkov observed the growth of nails and fur when rabbits' ears and fingers were removed and placed in special liquids. So it is possible to resurrect individual organs.

Professor FA Andreev conducted many experiments on dogs in 1913 and concluded that by anesthetizing dogs, the dogs were resuscitated by sending blood to the body and the whole organism could be revived.

1928 At a congress of physiologists and biochemists in Tbilisi, Bryukhonenko and Chechulin demonstrate an interesting experience: cutting off a dog's head, injecting blood into its veins through rubber tubes, and observing the dog's condition. saliva begins to separate when you put the sausage in the bur. In 1966 he was posthumously awarded the Lenin Prize for his invention of the AIK instrument. In 1940, Sinitsin was able to transplant and hold the hearts of frogs and fish. Academician VANegovsky created a common method of resuscitation in 1941-1945, which was suitable for the resurrection of many soldiers and officers during the Great Patriotic War. In nature, it is a near-fatal condition and is called anabiosis: and we can find it in the plant and animal worlds. In the process of long evolution, plants, animals, and microorganisms go into a state of anabiosis, adapting, in order to survive various adverse effects. For example, by reducing the osmotic pressure from extreme cold or heat, by reducing the oxygen in the air, by freezing and drying, special chemical conditions can be created, that is, by using protective substances, anabiosis can be formed. During anabiosis, all functions in animals are sharply reduced (body temperature, heart rate, respiration, metabolism are sharply reduced, reflexes are lost). Anabiosis occurs in worms, fish, frogs, hedgehogs, lizards, bears, and frogs.

In humans, a condition close to anabiosis is called secondary sleep. Lattergic sleep is caused by severe effects, severe illness, and nervous mental illness.

Aging is a three-phase process:

1. Aging in infancy.

2. Aging in adulthood.

3. Aging.

The main task of veterinarians is the prevention and treatment of various diseases. General prevention is a measure of disease prevention using various ways, methods and measures, which consists of complex economic, organizational and veterinary-sanitary measures, which are:

1. The work of improving the external environment, for this it is necessary to create cultural meadows, the transition to the zagon system, the exchange of meadows, the removal of poisonous plants found in the meadows, various harmful substances. Grasslands, barns need to be disinfected and mechanically cleaned. Surrounding the farm, arranging insulators, building cemeteries and animal cremation rooms, improving the reclamation condition of meadows, drying or increasing moisture, washing away salts and other activities:

2. Bacteriological, serological, biochemical, radioactive isotopes and other methods are used to determine the latent stages of the disease by various methods, with regular examinations, taking appropriate measures, ie X-ray machines, allergic methods, blood tests. Twice a year in spring and autumn medical examination is obligatory:

3. It is necessary to strengthen the natural resistance or reactivity of the organism in various ways:

4. It is necessary to treat with various drugs, vaccinations, extensive use of drugs prepared from immune serum, biostimulants, proteins and enzymes:

5. Promotion of veterinary and sanitary knowledge, etc.

General principles of treatment:

1. Treatment of the causes of diseases. Use of antibiotics, hormones, vitamins, etc.:

2. Treatment against the pathogenesis of diseases:

3. Against the symptoms of the disease: treatment of diarrhea, tachycardia, fever, cough, etc .:

4. Treatment by replacement.

QUESTIONS FOR CONTROL.

1. What is nosology?
2. What are the directions that explain the origin of diseases?
3. Explain diseases in relation to divine power?
4. Explain the diseases associated with fluid contamination?
5. Explain the disease by linking changes in the distance between atoms and particles?
6. Explain the disease by linking it to changes in the cells?

ETIOPATHOGENESIS.

Plan:

1. The concept of etiology and pathogenesis. Criticism of metaphysical views on etiology: monocaualism, conditionalism, currents of constitutionalism.

2. Causes, conditions and prevention of diseases. Pathogenesis and the mechanism of action of etiological factors in it. The main mechanisms of disease development. Ways of transmission of pathogenic agents in the body.

3. The role of neuro-humoral processes in pathogenesis. The role of animal type, breed, sex, and age in pathogenesis. Sanogenesis - healing. Protective-compensatory mechanisms that ensure the healing process of the body. The role of the nervous and endocrine systems in sanogenesis.

Main publications:

1. Khaitov RX, Eshimov DE "Pathological physiology of animals" Textbook Tashkent, "Ilim Ziyo" 2013.

2. M.Donald., James F. “Pathologic basis of veterinary disese” Humana Press; 2011 edition.

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BASIC EXPRESSIONS.

Etiology, A. Vezali, Malpighi, Louis Pasteur, Robert Cox, wine fermentation, silkworm disease, mechanical processing, boiling water, monocaualism, one cause, conditioning, condition, constitutionalism, racist theory, neuralism theory and practice unit, exogenous : mechanical, physical, chemical, biological, endogenous: circulatory disorders, mining, factory, cotton, inadequate, psychogenic, etiological factors, burns, poisoning, corticosteroid, stress, tension, resistance, breed, gender, age, compensatory organs mechanisms.

1. Etiology - teaches the general laws of origin of diseases in the body, their causes, a set of conditions. Etiology is the Greek word for aitia-cause, logos-doctrine.

According to IP Pavlov, the future should become a hygienic veterinary, hygiene. Therefore, it is necessary to protect the external environment, and a lot of work is being done in this area. IPPavlov said that it is necessary to know all the causes and conditions of the disease.

The doctrine that teaches the causes of disease is the result of a struggle between materialist and idealistic currents. This doctrine has explained the origin of diseases in a simple, mythical, teleological way, i.e. the disease is caused by the influence of zinc, contamination of juices, changes in their composition, decrease or increase, thinning of particles in the body or indicates that the disease is caused by thickening. Later in the Middle Ages the origin of diseases was badjahil zinc«archetypal»explained in connection with the wrath of God. As a result of observations, A. Vezali and Malpighi began to study the structure of the organism in depth. By this time, the development of industry, the production of dyes, the increase in the production of equipment, created favorable conditions for the study of the functions of the organism.

At the end of the 19th century, the production of wine and silk in many countries, including France, fell into disrepair. This poses great challenges for French scientists. As a result, Louis Pasteur, under his leadership, began to search for and find the causes of many diseases. As a result, they discover that microorganisms are the cause of wine fermentation and silkworm disease. Microorganisms can be used to prevent the deterioration of wine quality by washing wine containers with boiling water and disinfecting silkworm rooms. Thus, by identifying the real causes of the disease, now world scientists are doing a lot of research behind microorganisms, and German scientist Robert Cox is discovering the causes of tuberculosis, Louis Pasteur cholera, rabies and other diseases. The discovery of these diseases, on the other hand, follows a certain pattern, and this current is called the monocaual current. Mopo-single, single, couza - means cause. This doctrine is one of the most advanced doctrines of this period and deals a severe blow to religious doctrines. However, this doctrine does not fully explain the causes and conditions that cause disease, because the entry of microorganisms into the body does not always cause disease. As a result, the doctrine arises that diseases are caused by changes in the sum of many conditions, not microorganisms, and this doctrine means the conditionic conditions called the doctrine of conditionalism. This doctrine is contradicted by the inability to explain the disease, claiming that there is no clear cause for the disease, negating the importance of microorganisms in the origin of the disease.

Constitutionalismproponents of the theory explain that the disease arises from the genotypic structure of the organism, as a result of a deficiency in the constitution. The constitution and genotype

do not change at all, so the disease is interpreted as a fatal process or a top-down process. With the emergence of the theory of constitutionalism, many erroneous theories have emerged. There is a misconception that people with low genes and low constitutions should be confused with people with high genes and high constitutions. As a result, Nazi Germany wiped out many nations in order to create a new race, and racist theories still prevail in many countries. These teachings exaggerate the causes of disease,

Nervism explains that the organism is closely connected with the external environment, which is due to the nervous system.

In studying the doctrine of etiology, we must take into account the structure of the organism and the principles of their solidarity, that is, we must combine theory and practice closely, which can explain the etiology in detail.

The causes of the disease are studied into 2 major groups: external or exogenous, internal or endogenous causes.

External causative agents include mechanical, physical, chemical, biological, and other causes.

3. External environmental factors that cause disease.

External causes of the disease are those influencers that affect the body from the external environment and create a pathological process. The causes of the disease are studied in close connection with the organism without self-study of the external environmental factors, and the degree of origin of the disease depends on its nature. Environmental factors that cause disease include mechanical, physical, chemical, and biological causes. As a result of absorption (reserves and electricity, light energy) or reflexively (conditionally and unconditionally) into the closed automatic (IPPavlov) MNS through the place where all factors directly affect the organism of highly developed animals by reflector).

Mechanical factors causing the disease.

An influencer that affects the body from the external environment, causing an injury to this or that in the body, is called trauma.

In such cases, the injury can be caused by mechanical (shock, bruising), thermal (hot and cold), electric current, chemical, X-rays, and even heat (fear, strong impact) and other changes. 'ladi.

Usually the term trauma or injury is used in a narrow sense to refer to changes that occur mechanically. All changes to mechanical injuries are made by crushing, wounding, sharp, impenetrable, shot bullets, pressure objects.

Stretching, crushing, beating, injuring blood vessels and nerve fibers at the site of mechanical impact. The pathological changes that occur as a result of stretching or traction depend on the strength of the causative agent, the duration of exposure, and the physiological properties and condition of the organ or tissue that is stretched or stretched.

The bones and tendons are also stretched and stretched, and when the muscles contract, they are pulled less than when they are still.

If an organ is strongly pulled and stretched (skin, muscle ligaments, bones, etc.), it is torn and torn. Slow but long and repetitive pulling stretches (e.g. in joints) causes the connecting parts to loosen, causing the joints to play, come out, and so on.

Strong and long-term filling of internal organs (stomach, intestines, bladder). This causes dystrophy of the organ wall and glandular cells.

While changes in organ and tissue compression cause disruption of blood supply, long-term compression of organ or tissue causes tissue nutrition to deteriorate, leading to atrophy and even necrosis.

Strong organ dysfunction occurs when animals are rescued from being trapped underground, resulting in frequent traumatic shock-like disturbances in renal function.

Injuries occur in animals as a result of exposure to cold or firearms, thunder, and air waves. Falling from height or rupture of spleen and blood vessels of deep tissues and organs under the influence of thunder waves is observed fracture of bones without changing the skin lining system.

Traumatic injuries in farm animals (from the coldness of animal caregivers) are caused by the impact of equipment and tools used in various industries (machine mechanisms, washers, dots, etc.).

The following types of traumatic injuries are distinguished:

3. Closed injuries in which the integrity of the skin covering system is not compromised include: compression of the tissue (with tumor, wash, and puncture). Stretching, pulling, breaking, breaking bones, breaking, cracking under the influence of impenetrable weapons.

4. Injuries to the skin lining system, open changes include injury, destruction of the skin lining of the bone, tearing. Depending on the strength of the impact, torn, incised wounds are formed.

One of the characteristic or characteristic changes when an injury occurs is the sensation of pain. The formation of pain is associated with exposure of the organ to extra and introceptors, the breakdown of toxins, tissue breakdown, and the accumulation of toxins of microorganisms in the injured area.

In addition to local changes during injuries, general changes in some organs (heart, respiratory organs, endocrine and external organs) are observed with reflex dysfunction, accompanied by tachycardia, shortness of breath, hyperglycemia, increased blood pressure and other changes. characterized.

Injury to tissues on the surface of the body causes microorganisms to enter the internal parts of the body and cause them to become inflamed. Normally, pathogenic changes are limited due to the activity of protective flexibility mechanisms that protect our body when tissue injury occurs, only in some cases the process is exacerbated by insufficient resistance of the body's protective flexibility mechanisms, leading to the development of pneumonia and then sepsis.

The dead-necrotic tissue in the injured parts forms a large part, and the direct effect of the cause of the injury is due to the wash. The occurrence of such changes is associated with the restoration of tissue nutrition and metabolism by narrowing and rupture of blood vessels, disruption of the integrity of the innervation, and finally compression of the injured tissue and adjacent healthy tissue with exudate.

Long-term purulent wounds are a debilitating weight loss due to the body not healing. Injury weight loss leads to severe damage to internal organs (pleura, lungs, ribs, pelvis and stones). In such cases, the process of tissue regeneration is weakened, atrophy develops in the skin, subcutaneous tissue, transverse skeletal muscles, some internal organs: the animal's appetite is suffocated, sleep is disturbed, liver and intestinal function is impaired, some parts of the bed lie together. becomes lifeless.

Toxins produced by microbes during chronic injuries, the products of tissue breakdown, poison the body and cause it to lose weight. At the same time, many proteins in the pus are released from the body, which weakens the body's resistance to pathogens.

Traumatic shock is one of the most severe pathological conditions of the body.

During a period of traumatic shock, after a short period of agitation, a strong inhibition of the basic physiological functions of the body occurs. Characteristic changes during traumatic shock include acceleration of breathing and pulse, increase in blood pressure, increase in blood glucose and adrenaline. Subsequently, blood pressure decreases, the amount of blood circulating in the blood vessels decreases, body temperature decreases, reflex activity weakens, the animal becomes insensitive to environmental changes, pain sensitivity decreases, alkaline blood reserve and tissue oxygen consumption decrease. The excitability of the cerebral hemisphere cortex and vegetative centers, the formation of biopathy is weakened. A traumatic shock condition occurs after trauma or exposure to a traumatic agent (primary shock). Primary shock is caused by the reflex excitation of sensory nerve endings under the influence of traumatic factors. The peripheral nerves are irradiated to the subcortical parts, first causing excitation and then braking in the cortex. It weakens all the physiological functions of the body, in particular by lowering vascular tone, leading to a decrease in blood pressure. Many scientists explain the secondary development of shock as poisoning caused by the absorption of histamine-like substances into the body through the blood vessels in the crushed part of the tissues. This is supported by the following supporting information. When histamine and other biologically active substances are released into an animal’s bloodstream, a secondary shock-like condition occurs, but histamine and peptone shock, although similar to this shock, do not resemble the shock that results from the injury itself. The formation of traumatic shock is accompanied by additional changes in the body, adverse factors (blood loss, fever or heat, hunger, fatigue), the width of the injured area (nerve

columns), due to the abundance of receptors and many other factors. In the development of traumatic shock and subsequent restoration of impaired function occurs the influence of pituitary, adrenal hormones, nervous system and other organs.

The outcome of trauma depends on the type of organ, its vital importance. Death can occur if the heart, large diameter blood vessels, nerve centers, etc. are injured. The changes resulting from the effects of mechanical influences on the nervous system are severe and complex. When peripheral nerves are injured, the motor and sensory properties of organ systems change. Mechanical injury of the central nervous system causes severe functional changes in the body (the affected area depends on the degree of injury). Severe bruising, bullet and skull injuries, causing general bruising, can sometimes injure the brain, blocking blood vessels and the respiratory center. This results in cessation of breathing or paralysis of the heart.

Spinal cord injury paralyzes the leg and impairs the function of pelvic organs (urine, fecal excretion, etc.). Sometimes when a strong blow to the podcherevnoy (abdominal) part, the heartbeat weakens and even stops. Injuries to the heart and large blood vessels are dangerous for the body. When a heart is injured, death usually occurs from exposure to its neuromuscular apparatus, thrombus and blood flow to the heart cavities.

Rupture or injury to the artery of the hip, pelvis, and mesentery results in external and internal rupture, resulting in death. Rupture of the tissues in the chest causes air to enter the interstitial spaces and compress the lungs, leading to disruption of the reflex.

Disease-causing sound wavesdepending on the strength, frequency and duration of exposure to sound waves can have a detrimental effect on the body. Noisy mixtures of different strengths and heights have a detrimental effect on the body. Under the influence of these noises, strong agitation, fatigue, changes in the respiratory process, worsening of hearing, increased intracranial pressure and other pathological changes occur.

Accidental, sudden loud noise can damage the hearing aid: a long and strong generated sound wave can affect the activity of the central nervous system. Pathological changes in the body (metabolic disorders, changes in cell structure, accumulation of heat in the body, when the ultrasound is exposed to a sound that is too long and strong) an increase in glucose and cholesterol in the blood, a change in the shape and structure of the shaped elements of the blood i.e. deformation can cause protein coagulation and other changes).

The causes of internal disease often include the factors that contribute to the onset of the disease in the body. For example, as a result of working in mines, factories, and mines, toxins that enter the body in different ways are absorbed into the tissues, and the dust settles in the lung tissue, causing various deficiencies in these tissues and causing disease. causes. Circulatory disorders are also among the internal causes of the disease. Changes in hereditary traits also cause disease under the influence of mutagenic causes.

Pavlov recommends studying the causes of IP disease in three groups:

1. All exogenous and endogenous causes are the first group of causes to which the body responds with an unconditional reflex.

2. The indifferent effects created by IPPavlov's work, that is, the influence of the causative agent, if supported by normal conditions, then the natural effect of this supporter is called by the disease itself. For example, if you take an apomorphine in a syringe, tie the dog to a machine, and then send the apomorphine to the dog every time it is supported by a light or a bell, then turning on the light bulb will cause illness and the dog will vomit. called syrotchis. The body of animals responds to the causes of this disease by producing conditioned reflexes.

3. Psychogenic causes have also been proven in experiments and are of great importance for human beings, that is, affecting the body by speaking, drawing, grieving, and writing harsh insults can also lead to diseases.

1.Pathogenesis is the study of the origin, mechanism of development, pathogenesis, course, and consequences of diseases.

Greek pathos-victim, genesis-formation. Diseases develop by different mechanisms when different pathological causes affect the body. To make the doctrine of pathogenesis easier to understand, it is distinguished that etiological causes affect 3 different types.

Type 1 causes diseases that affect all stages of development. For example, in acute poisonings, until the toxin is released from the body, it affects the development of the disease in the body, or a similar change occurs when an electric shock.

Type 2 causes serve as a driving force, developing the mechanism of the disease. For example, as a result of a single exposure to hot water, it acts as a starting force. The following substances are formed and poison the body, disrupt the permeability of blood vessels, create an acidic environment and create oxygen deficiency.

Type 3 etiological causes continue to affect themselves depending on the duration of disease development.

The basic structure of the mechanisms of disease development is that when various causes affect the body, there is a lack of oxygen in the body, that is, the metabolism changes, which disrupts the function of various organs and the mechanisms of disease begin to develop.

1. Corticoviceral doctrine is a two-way connection, ie a doctrine that explains that the nervous system is connected to all internal organs. The effect on the body is affected either by a conditioned or unconditioned reflex pathway and responds using unconditioned reflexes. The mechanism of disease development also depends on the reactivity properties of the organism. If reactivity is strongly developed, the disease may not develop. If the body is deficient in various micro and macronutrients, the nutrient content is incomplete, or the body is tired, the development of the disease can occur slowly.

2. Depending on the types of nervous system. If the animals fall into the fragile type, the disease develops more strongly.

3. Explains the development of the disease under the influence of stressors. When inadequate effects on the body are given to the pituitary and adrenal glands over a long period of time, they produce 3 different changes to the effects as they control the body’s reactivity.

1.The properties of tension The pituitary and adrenal glands produce a lot of hormones, adapt to stress by inadequate action, strong excitation, and produce a variety of hormones. If the hormone-producing function either increases or decreases, the body's function is impaired.

2. In the stage of resistance, the body is resistant to any pathogenic influences, because the hormones of the pituitary and adrenal glands increase the energy and plastic mobilization of the body. In the stage of resistance, when the body can not cope with the pathogenic force, the stage of general weakness, without exhaustion begins.

3. At the stage of general weakness, the body loses flexibility, immunological reactions, regeneration state decreases.

3. Examination of cell composition in animals and humans revealed that the development of pathological processes depends on chromosomes: for example, defects in the development of sex, ie secondary sexual characteristics, infertility and other changes. Males have one more sex chromosome and females have one less sex chromosome.

The role of constitution in pathogenesis. The disease arises from the encounter of disease-causing causes with the organism. Therefore, in addition to qualitative and quantitative changes in the pathogen, the characteristics of the animal organism are important in the origin of the disease. The individual reactivity of the organism takes the first place in the origin of diseases in the organism, because the effect of a certain pathogen on the organism of animals does not lead to the disease of all animals, but to some of them.

What is the constitution? Although there is still no complete answer to the question, constitution refers to the general morphological and physiological features of an organism, which are the product of long-term evolution from the interaction of the organism with the external environment, and these properties are stable. Due to these features, the reaction of the organism to the external environment is determined, comparing close species.

The constitution of agricultural animals means that it increases the resilience, resilience, disease resistance, flexibility and productivity of the farm and the environment. Thus, the constitution of farm animals means not only the morphological and physiological characteristics of the organism, but also the reactivity of the organism to the external environment, including the development of a response to the causes of the disease.

The whole organism can be afflicted with various diseases, and it is impossible to know in advance for what reasons they occur. It depends on external influences, hunger, poisoning, fatigue, exposure to cold and other causes that change resistance and their effects. Due to congenital malformations of the organs in some organisms, the influence of the above external causes causes the disease. In recent times, it has become common to study the constitution in two parts:

1. The constitution of the breath.
2. The constitution of digestion.

Importance of breed, sex and age in pathogenesis. Animal breeds play an important role in the origin of the disease, and Algerian sheep do not suffer from anthrax. Horses of the Budyonny breed are not susceptible to lung diseases. Caucasian mountain merinos do not suffer from pyrapylazmosis, but other breeds are highly susceptible to the cause of this disease. Depending on age, young animals suffer from diseases of the digestive organs, pneumonia, some infectious diseases. As the animals mature, many diseases become more resistant.

4. Restoration of body activity. Protective resilience mechanisms in the body that have the ability to restore impaired function under the influence of pathogenic influences, including excess energy generated in the body, surfaces, stored blood, chemicals and biochemicals. For example: under normal physiological conditions, 17-20% of the heart muscle, the respiratory surface of the lungs, the absorption surface of the intestine, 20-25% of the glomeruli of the kidneys, 12-15% of the liver, 10-15% of the blood vessels, 50 of hemoglobin -60% and nervous, endocrine systems are rarely used. Therefore, the organism adapts to any difficult conditions. For example: in bilateral pneumonia, dystrophy and fatty heart muscle, severe liver injury, removal of a single kidney, functions are also compensated when a large part of the stomach and intestines are cut, when a lot of blood is lost, when many capillaries become loose and clogged, and when nerves and endocrine glands are injured. The patient's kidney function is performed by a healthy kidney, and lymph nodes perform blood formation when the spleen is removed or diseased.

Control questions.

1. What is the etiology?
2. What theories do you know that explain etiology?
3. What are the types of etiological factors?
4. What is pathogenesis?
5. Explain the mechanism of action of etiological causes that explain the pathogenesis?

ORGANIZATIONAL REACTIVITY AND RESISTANCE.

Plan:

1. The concept of reactivity and resistance of the organism.
2. Types of reactivity and resistance. Forms of manifestation of the organism's reactivity and factors influencing it.
3. The role of nervous and endocrine systems in reactivity..

Main publications:

1. Khaitov RX, Eshimov DE "Pathological physiology of animals" Textbook Tashkent, "Ilim Ziyo" 2013.
2. M.Donald., James F. “Pathologic basis of veterinary disese” Humana Press; 2011 edition.
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Basic expressions

Reactivity, resistance, immunity, NNSirotinin, R.Virkhov, IIMechnikov, VVPashutin, AABogomolets, IPPavlov, nerve, humoral, ADSperanskiy, AAUkhtomskiy, AMMonaenkov, NEVvedinsky, IMSechenov, heat, cold, physiology, heat, cold, poison, , hereditary, AMBezredko, barrier, phagocytosis, immunantana, full-value, worthless, macro-and micronutrients.

Organisms have developed resistance mechanisms to exposure to the external environment at different times. First of all, the general reactivity in the body, that is, the resistance to various toxins, and then the types of immunological reactivity developed. As organisms now develop, the reactive function is performed by cells, which later develop a response using the humoral system and eventually the nervous system.

The properties of reactivity depend on the age of the animal, the nervous and humoral systems, the external environment and the general condition of the organism. For example, when the embryo develops in the mother's womb, it responds to the stimuli through the mother's body, ie through the placenta. When a baby is born, its reactivity is weak and responds only by a phagocytic reaction or by immune cells that pass through the mother’s blood. That is why young animals often get sick and die. Young animals are weakly adaptable to changes in ambient temperature, and their dyspepsia, salmonellosis, colibacillosis, rickets and other diseases are common. Reactivity in adult animals is manifested in the fight against microorganisms by antibodies, phagocytes and macrocytes that have accumulated in their bodies. As the body ages, its reactivity decreases. phagocytes, immune cells are reduced, and the incidence of disease increases with susceptibility to disease. As a result, tumors, hypertension increase, regeneration is weakened, and the body's reactivity is low, so they have severe infectious diseases.

Sirotin NN and other scientists note that the cerebral cortex of cold-blooded and young animals is poorly developed and is less sensitive to strong toxins (histamine, diphtheria, stolbyank toxin). During anabiosis, animals do not develop sensitivity to very strong toxins and infectious agents (plague, tuleremia, anthrax, tuberculosis).

Due to reactivity, the body responds to disease-causing causes, and the sensitivity of different individuals to infectious agents varies. Such cases can be observed in various pathological processes. For example, when an animal with a high reactivity burns, it recovers quickly and an animal with a low reactivity recovers later. The reactivity of the animal organism depends on the metabolism, the immunological properties of the organism, the functional state of the animal organism, the vascular reaction and chronaxy to the excitability of the nervous system.

Concepts of reactivity R Virkhov's cellular theory developed at a time when the theory of cells gave a misunderstanding of the general reactivity properties of individual cells, tissues and organs, ie the fact that pathological processes take place only in cells. 'did not notice. In contrast, IIMechinkov in his many years of observations shows that the reactivity of organisms at different stages of evolutionary development is also formed under the influence of disease-causing factors of the external environment. As organisms become more complex and the nervous system develops, the body's reactivity to

inflammatory agents becomes more complex. For example: cold-blooded frogs, inflammation in fish, develops very poorly in warm-blooded animals. Even when these properties were observed by NNSirotinin sending proteins to the body, it was observed that the body of cold-blooded animals produced very weak responses. Gradually, as a result of the development of the nervous system of the organism, the reactivity or sensitivity of the organism to many toxins, formed a changing response.

Reactivity is a characteristic feature of all animals, and in the field of reactivity IIMechnikov, VVPashutin, AABogomolets, NNSirotinins have done a lot of research. In their laboratories, these scientists studied reactivity by linking it to metabolism and other areas. IPPavlov and IMSechenov confirmed that the nervous system plays a leading role in the development of reactivity. In the IPPavlov laboratory, MKPetrova et al observed that the reactivity of animals was impaired by inhibiting the cerebral cortex by giving bromine preparations.

The importance of the types of nervous system in reactivity is also great. To study the importance of types of nervous system in reactivity, they took two groups of dogs:

1. The group includes dogs with a weak nervous system.
2. Dogs with a strong type nervous system in the group.

In animals of both groups, when exposed to strong toxins, cyanic acid, bacterial toxins, dogs with a weak nervous system became ill due to weak barrier properties of the organism, in animals with a strong nervous system AMMonaenkov and others explain that the diseases have not developed because their barriers are strong, their neutralizing properties are high.

In the IPPavlov laboratory, pigeons became infected with anthrax when a certain part of their brain was removed.

Academician ADSperansky observed that when dogs opened their brains and placed a ball in the midbrain, mechanical effects resulted in ulcers in the lungs and digestive systems, weakening their resistance to infection. He drew attention to the fact that the traces of the nervous system in the origin and development of pathological processes, that is, pathological processes in the nervous system, even after their recovery, retain their complications for a long time. In many experiments, that is, when animals are exposed to different stimuli after treatment of the disease, the effect of these stimuli spreads to the entire nervous system, leaving traces of old disease in the affected area. observed that it had survived and accumulated, leading to the onset of the disease. This feature of the nervous system is called AA

Reactivity is also affected by the autonomic nervous system. Reactivity changes when the function of the autonomic nervous system increases or slows down. Excitation of the sympathetic nervous system enhances phagocytosis, enhances metabolism, and increases reactivity. Excitation of the parasympathetic nervous system increases the production of antibodies, produces short-term leukocytosis, followed by leukopenia, exposure to certain toxins (phenol, aniline, etc.), lymph nodes, liver barrier - barrier properties increases.

Reflexivity changes reflexively from the pathological effects of heat and cold. For example, as a result of colds, people get the flu, pneumonia, that is, the body's reactivity decreases. In experiments, it is possible to cool the body of chickens, reduce their reactivity and lead to anthrax, or to heat the body of guinea pigs and reduce their sensitivity to proteins.

Toxic substances, alcohol, carbon monoxide, lead, mercury, cyanic acid weaken the internal braking. Pigeons were poisoned with alcohol, which reduced their reactivity to anthrax, or when people consumed alcohol for a long time, they observed a decrease in the general reactivity of the organism, and xko.

While ultraviolet light from light energy increases the stability of an organism to a certain extent, it weakens the stability of an organism to a certain extent. X-rays and gamma rays have a detrimental effect on the body's reactivity. The reactivity of the organism also decreases under the influence of mechanical influences. Thus, the role of nervous endocrine systems in the formation of reactivity of the organism is important, but different effects of the external environment affect the activity of various organ systems of the organism, affecting their metabolism, neurohumoral control mechanisms.

There are several classifications of reactivity, and most scientists classify the organism according to its state of health or disease:

1. Physiological reactivity.

2. Pathological reactivity.

Physiological and pathological reactivity can be individual or individual, as well as group. Individual or specific reactivity depends on hereditary traits and can be passed down from generation to generation. Physiological reactivity develops the body's response to natural (adequate) influences, while pathological reactivity develops the body's response to the causes of the disease. Allergic and immunological types of pathological reactivity are distinguished, and the manifestation of these types of reactivity is formed in relation to foreign proteins, microbes and their toxins. (Allergy, Anaphylaxis, Immunity). Typically, biological or species reactivity is differentiated and is specific to animals belonging to a particular species, ranging from seasonal changes in animals to: seasonal sleep, migration of animals from one place to another, animals are not exposed to microorganisms, ie chickens are not infected with anthrax, specific reactivity is a characteristic feature of a particular individual, it depends on the constitution, sex, age, nutrition and storage characteristics, newborn reactivity in animals is low, reactivity is well developed during sexual maturation, phagocytosis and the formation of immunoassays are well demonstrated, in older animals the reactivity of the organism is low due to the weakening of their barrier properties. Hence, the specific reactivity is that during the period of complete vaccination of animals, their reactivity is formed differently, with strong antibodies in some and weak antibodies in others.

The resistance of an organism, as the Latin resisteo (resist, resist), is the resistance of an organism to physical, chemical, and biological causes of disease. This means that the body's resistance is understood to be resistance to many different causes.

During phylogenetic development, when the resistance of the organism changes and invertebrates are resistant to bacterial toxins, the susceptibility of warm-blooded animals is high. Resistance is associated with the functioning of organ systems, depending on the type, sex, age, constitution, anatomical and physiological characteristics of the animal, the level of development of the organism, the development of the RES and lymphoid system. In the early stages of ontogenetic development of animals, resistance to various harmful agents is high (partial pressure reduction, some bacterial toxins), resistance to sexual development is well developed, and resistance decreases with age.

Resistance:

1. Natural-born,

2. Acquired-generated species are different.

Congenital resistance is passed down from generation to generation. For example, Algerian sheep are more resistant to anthrax than European sheep.

Acquired generated resistance depends on the individual characteristics of the organism and is formed when immunized against infectious diseases. Resistance is formed depending on the activity of the pituitary, adrenal glands, colon, gonads. Barrier properties of the organism, biologically active substances in the blood and phagocytosis play a key role in resistance. When the body is tired, very productive, living conditions are poor, resistance is weakened, and conditions are created for the development of diseases.

2. Animals and humans live in a world of microorganisms. Immunity, on the other hand, as a controller, rigorously tests agents for various causes that have entered the body.

Immunity - Latin Immunitas - means purification, deliverance. Immunity is the ability of an organism to be exposed to antigenic pathogens, their products and hereditary foreign substances, or to be resistant to various disease-causing microorganisms, viruses and their products, as well as to non-infectious modes. , forms a special view of the overall resistance.

Immunity is divided into two depending on the nature of the mechanism and causes that cause it:

1. Congenital immunity or hereditary immunity from generation to generation.

3. Acquired immunity

Congenital or natural species-specific immunity is a specific resistance of an organism that is passed from generation to generation and is specific to a species, breed, and population. For example, in cattle, horses are resistant to microorganisms that cause croupous inflammation of the lungs, and

animals are highly resistant to human diarrhea. Dogs are not infected with pleural pneumonia in cattle. Cattle do not suffer from horse manure, infectious (infectious) anemia.

Inter-species immunity is also different, Algerian sheep are resistant to anthrax, Breton sheep are resistant to smallpox, light-bodied pigs are resistant to yellow fever, Mongolian cattle are resistant to plague, and other animals of this type are infected with the above diseases. Congenital immunity is formed not only against an infectious agent, but also against their toxins. The barrier properties of animals with innate immunity are strong and do not transmit microorganisms into the body or prevent the growth of microorganisms by altering the environment.

These organisms have high phagocytic activity and bactericidal properties in fluids, which prevents the development of microorganisms and forms specific immune cells against these microorganisms.

Acquired immunity is formed during the ontogenetic development of certain microorganisms in the body of animals. Acquired immunity is created by natural and artificial means. For example, naturally acquired immunity is formed after recovery from mango, smallpox, proteinuria and other diseases. Artificial active immunity is created by vaccinating animals against various infectious diseases. Hence, acquired immunity is generated by natural and artificial means.

Artificial immunity is studied as active and passive immunity. Passive immunity is formed when hyperimmune serums are sent, through the passage of immunoassays through milk, through the placenta. Due to passive immunity, the body's resistance is maintained for some time. RES plays a leading role in the formation of immunity, and the formation and formation of immunity is controlled by the nervous system.

During the period of immunity against infectious diseases, if the organism is completely cleansed of infectious agents, sterile immunity is formed and the organism is provided with sterility to this antigen. If the immunity formed in the body does not maintain complete sterility, and the antigen is retained in the body, it is called nosteril immunity, which is characteristic of tuberculosis and brucellosis.

Immunity can be formed not only against microorganisms themselves, but also against their toxins, which is called antitoxic immunity and is observed during exotoxin-producing microorganisms: tetanus, botulism, gas gangrene and other infections. Hence, toxins act as antigens in this process.

In addition, the body has special organs and factors that fight microbes and foreign substances, which are called barrier properties of the organism. The barrier-barrier properties of the organism are studied as external and internal barriers.

External barriers of the body include the skin and its products (accumulations), mucous membranes in various parts, the oscillating epithelium of the respiratory tract, microorganisms of the digestive system and hydrochloric acid.

Control questions

1. What is reactivity?
2. Explain the role of the nervous system in reactivity?
3. Explain the role of VNS in reactivity?

ALLERGY AND ALLERGIC DISEASES

1. Allergy, causes, types and mechanism of development.
2. Allergens and their types.
3. Allergic diseases and their types. The role of allergic reactions in the diagnosis of infectious diseases.

Main publications:

1. Khaitov RX, Eshimov DE "Pathological physiology of animals" Textbook Tashkent, "Ilim Ziyo" 2013.
2. M.Donald., James F. “Pathologic basis of veterinary disese” Humana Press; 2011 edition.
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The body's internal barriers include a number of cellular and humoral factors, various histiocytes, reticular cells, plasma cells, epithelial cells of the inner wall of blood vessels, and leukocytes. RES cells, which are involved in protecting the body, are active, they absorb microbes and other particles that enter the body, they are very rich in RES in the lymph nodes, spleen, liver, lungs, kidneys, meninges, blood-forming organs, skin . This means that RES is present to one degree or another in various organs of the body, and phagocytic activity is much higher in leukocytes, including neutrophils. In his long-term observations, IIMechnikov argued that the process of phagocytosis plays an important role in the formation of immunological features. microbes and their toxins, cellular elements, tissue breakdown products, other particles are digested in cells. Phagocytosis is the process by which particles are trapped in a cell and then digested. Phagocytosis is common in nature, with feeding and protection of single and multicellular simple animals occurring in a single cell, while in highly developed animals these systems are isolated and protected by specific mesenchymal cells (blood leukocytes, lymph nodes, red blood cells). bone marrow, spleen, liver, connective tissue histiocytes) - by phagocytes. Studies have shown that there is a direct link between the process of phagocytosis and the resistance of the organism. increased phagocytosis indicates a weakened immunity in the body. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are

substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens.

1. Antitoxins and antifenzymes, immune cells that inactivate by binding toxins and enzymes.

2. Agglutinin and persipitins, antibodies that change the colloidal chemical structure of microorganisms, immobilize them, bind them to the sediment.

3. Cytolysins or cytotoxins - antibodies that break down cells under the influence of enzymatic complement substances.

4.Opsonins and bacteriotropins - change the appearance of microorganisms, facilitating phagocytosis.

If antibodies are formed under the influence of antigens, what are the antigens themselves?

Antigens are substances that enhance the formation of immune bodies and react selectively with them. These include microbes, toxins, erythrocytes and serum of other animals, as well as high-molecular compounds.

There are two types of antigens.

1. Full value antigens.

2. Incomplete antigens - haptens.

Complete antigens include complete proteins, ie serum, various proteins, microorganism toxins and filtrate colonies. Antigens have specific properties that react with the antibodies they produce.

Incomplete antigens, ie haptens, cannot enter the body to form antibodies and only bind to the protein molecule to achieve antigenic properties.

Antigens must be administered parenterally to the body to form immune cells. Antigens are exogenous and endogenous substances that are foreign to the body. The body’s own proteins also sometimes exhibit antigenic properties. To do this, the body's proteins meet with the infectious agent, toxins, and form an autoantigen. In order to form immune cells against antigens, the antigen remains in the body for a certain period of time, is captured in the liver, spleen, lymph nodes and stored in the blood for 2-3 weeks. Immunological reactivity is formed not only from the encounter of macro and micro organisms, but also from other types of individuals and even in the same organism itself when tumors grow, become inflamed and in other cases have antigenic properties against their own organism. In all cases, there are antigen and antibody reactions and phagocytosis between body tissues and other tissues. The tissue formed during embryonic development serves as an antigen for older tissues. Tissue does not fit the transplanted tissue or organ due to the immune barrier property of these organisms when transplanting organs into one species or individual, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by.

Decreased or complete loss of antibody production as a result of exposure of antigens to the body is called immunological tolerance or non-response. This condition is caused by antigen transmission during the embryonic period or after the animal is born. In older animals, immunological tolerance can be established by transferring large amounts of antigen or exposing them to X-rays. Immunological tolerance is characterized by the loss of these antigens of their antigenic properties, which is observed

when transplanted into other animal tissues, and the transplant grows well. It is currently used in blood transplants to remove tissue barriers from immunological tolerance.

Control questions

1. What is immunity?
2. What types of immunity do you know?
3. On the basis of what theories do you explain the formation of immunity?
4. What is an antigen?
5. What types of antigens do you know?
6. What is an antibody?

Inflammation

Plan:

1. The concept of inflammation. Etiology and external signs of inflammation. The main stages of inflammation: alteration, exudation and proliferation.
2. Circulatory disorders in inflammation. Classification of inflammation. Exudate, its types and properties.
3. The role of nervous, endocrine and immune systems in inflammation. Consequences of inflammation and its importance for the body.

Main publications:

1. Khaitov RX, Eshimov DE "Pathological physiology of animals" Textbook Tashkent, "Ilim Ziyo" 2013.
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BASIC EXPRESSIONS.

Exogenous, endogenous causes, mechanical, physical, chemical, biological, necrotic tissue, infarction, salt accumulation, thaw, galenic, redness, swelling, local temperature rise, pain, dysfunction, alteration, dystrophy, phagocytosis, proliferation, exudation, emigration, protein, fat, carbohydrate, osmotic, oncotic pressure, vascular reaction loss of vascular tone, unevenness of the inner surface of the vessel, transudate, mechanical, biological, chemical theory, macrophage, granulison tissue , complete incomplete healing, transition to a chronic state, exudates. Serous, serous catarrhal, fibrinous, purulent, bloody or hemorrhagic, purulent or esophageal, normiergic, hyperarrgic, hepatic, reactivity, autoantigen, autoantetela, R. Virkhov, congeym, Ricker, IIMechnikov, Shade, Samuel, V.Ya. Danelevskiy, IP Pavlov, NI Simonovskaya, AD Speranskiy,

Inflammation is the most common, most complex pathological process known since ancient times, and in ancient times all diseases accompanied by a rise in local temperature were called inflammation.

Inflammation is a typical pathological change (disruption of tissue function and changes in structure) that is common in various diseases, as well as the activation of the body's protective resilience properties and the restoration of impaired function. Although inflammation in this area delays the organism as a process with protective properties against the effects, the mechanism of its development, the formation of symptoms depends on the state of the organism, the activity of neuro-humoral systems. For example: Inflammation of the skin can be caused by affecting some endocrine glands of the gillpotalamus or peripheral nerves. Glandular is a local manifestation of the general reactivity of the organism, the degree of reactivity of the organism depends on the course of inflammation and, conversely, on the reactivity of the organism to inflammation, neurohumoral control, thermoregulation and other mechanisms. All substances that cause inflammation are called phylogenetic substances, and we study them in two groups, namely, exogenous and endogenous substances. Inflammation occurs under the influence of phylogenetic substances, and the name of the inflamed organ or tissue is read by adding the suffix "IT", "IYA". For example. Inflammation of the liver is called hepatitis, inflammation of the kidneys is called nephritis, inflammation of the lungs is called pneumonia, and xzo

Inflammation is caused by mechanical, physical, chemical, and biological causes of external disease, and often the contribution of microorganisms and viruses is important in causing inflammation.

Sometimes inflammation can also be generated under the influence of conditioned indeferent stimuli.

Ichki yalligg‘lanish chaqiruvchi sabablarga nekrotik to‘qima, infarkt, gematoma, turli qismlarda to‘plangan tuzlar kiradi. Yallig‘lanish chaqiruvchi sabab, ko‘pincha yallig‘lanish reaksiyalarini hosil bo‘lish intensivligini belgilab beradi: Masalan. Rengen nuri, zaharli modda, mexanik jarohatlar, kuyish,sovuq urish va boshqalar oldin to‘qimalarni parchalab, keyin shu joyda fiziologik aktiv moddalar to‘planib, ular ishtirokida yallig‘lanish jarayonlari ro‘yobga chiqa boshlaydi. Surunkali kechuvchi kasalliklarda , kasallik chaqiruvchi sababni, begona tasirotchini uzoq vaqt tasiridan, yoki ximiyaviy qo‘zg‘atuvchining tasiridan proliferativ jarayonlar kuchayaadi.

Yallig‘lanishni kechishi kasallik chaqiruvchi sabab tushgan joyga bog‘liq bo‘lib, amyoba jigarga tushib absets chaqirsa, ichaklarda yarali yallig‘lanish chaqiradi. Masalan. Stafilokok, streptokoklarni yiringli infeksion jarayon hosil qilish aniq, lekin skipidarlarni teritagiga yoki muskullar orasiga yuborib yiringli yallig‘lanish chaqirish mumkin. Shunday qilib yallig‘lanishni xususiyati, uni hosil bo‘lish tezligini qo‘zg‘atuvchi xususiyatiga hamda yallig‘lanish kechayotgan muhitga bog‘liq ekan. Yallig‘lanishning tashqi mahalliy belgilari Sels va Galenlar tomonidan sharxlangan bo‘lib: qizarish-chivoch, shish tishoch, harorat ko‘tarilishi-saloch og‘riq - doloch, funksiyani buzilishi fipstto laesa deyiladi. Har qanday yallig‘lanish ham bir qancha asosiy bir-biri bilan bog‘liq jarayon bilan kechadi: altteratsiya-to‘qimalardagi distrofik o‘zgarishlar-to‘qimalarning yallig‘lanish chaqiruvchi agent ta’sirida qitiqlanishi va parchalanishi, maxalliy qon aylanishini buzilishi-ekssudatsiya va emigratsiya, fagotsitoz hamda proliferativ o‘zgarishlar. Yallig‘lanish chaqiruvchi agent to‘qimalarni qitiqlashi, parchalashi, ulardagi moddalar almashinuvini, tuzilish va funksiyani buzilishiga sabab bo‘ladi. Distrofik o‘zgarishlar yallig‘lanish chaqiruvchi sabab ta’sir etgan vaqtdan hosil bo‘lib, kam chegaralangan bo‘ladi. Keyinchalik ta’sirotchining ta’siri kuchayishi bilan yallig‘lanish kuchayadi, to‘qimalarda moddalar almashinuvi kuchayadi, qon aylanishi buzilib, distrofik o‘zgarish kuchayadi. Kasallik chaqiruvchi sabab organizmga tushib birinchi navbatda retseptorlarga tasir qiladi. Agar ta’sirotchi kuchi etarli bo‘lsa nerv oxirlarida parabioz xolatini hosil qiladi.

At the onset of inflammation, the tissue bends the cells, fat granules appear, protein and fat dystrophies are observed, then the cell structure is disrupted and even severely damaged and dies. Necrabiotic processes during inflammation are caused by the bending and melting of collagen and elastic fibers of tissue interstitials. In inflammation, necrobiotic processes are formed when tissue burns, under the influence of strong acids and alkalis, sometimes in relation to weak influences from increased sensitivity of the organism. There is a certain association between them and dystrophic changes in the body, and sometimes due to the injured part there is a compensatory restoration of their functions, despite the presence of destructive changes in the salivary glands, stomach and other organs. ladi. The development of destructive changes during the period of inflammation depends on the organ,

and such changes can be observed in injuries of parinchyomous organs. The degree of dystrophic changes depends on the strength and nature of the pathogen, where the pathogen enters, the nature of the injured organ or tissue, and the reactivity of the organism. Physiologically active substances formed as a result of dystrophic changes in the source of inflammation and metabolic disorders are absorbed into the blood, reducing vascular tone, causing emigration, phagocytosis and proliferation of cellular elements. These biologically active substances include histamine and histamine-like substances, acetylcholine, ATF, creatine phosphoric acid and other necrogorms that dilate blood vessels and enhance proliferation, trephon tissue proteases and cathepsins. Thus, the strong passage of alternative, proliferative and exudative processes during the inflammatory period leads to tissue bending and the development of dystrophic changes that complicate blood circulation.

Metabolism at the source of inflammation undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. Metabolism between the inflamed part and healthy tissue is enhanced. The increase in metabolism is due to easily oxidized carbohydrates, which form many weak acids as they take place in an oxygen-free environment. The breakdown of carbohydrates in the anaerobic phase increases due to leukocytes released during emigration, but these changes can be seen in the oxygen consumed and the carbon dioxide excreted before the breakdown is broken down into the final product. During this process, the respiration rate decreases as more carbonic acid is released.

During inflammation, the metabolism undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. The metabolism between the inflamed part and the healthy tissue becomes enhanced. Metabolism will be enhanced. Lactic acids are formed due to the fact that the increase in metabolism is due to easily oxidized carbohydrates, which take place in an oxygen-free environment. Due to the leukocytes released during emigration, the breakdown of carbohydrates in the anaerobic phase increases, but without decomposition to the final product, these changes can be determined by the oxygen consumed and the carbonic acid released. In this process, the respiration rate decreases as more carbonic acid is released.

Fats and proteins also form ketone bodies, albumin-peptones, which are not completely broken down in the center of inflammation. Excessive increase in carbohydrate protein and fat metabolism, complete oxidation of milk at the source of inflammation, pyruvic acid, fatty acids lead to an increase in ketone bodies, amino acids and peptones, and acidosis develops. Acedosis is compensated first at the expense of the body's alkaline reserve, then it is not compensated.

(N hyperonia is formed). Depending on the nature of the process taking place in the tissue, the change in the environment of the tissue becomes 7.1-6.6, ie weakly alkaline, in the acute process 6.5-5.4 in the acute flow process. Increased acidosis increases the dissociation of salts, changes the electrolyte ratio, increases the amount of potassium, increases metabolism, breaks down large molecules into small molecules, increases the amount of ions, increases the osmotic pressure at the source of inflammation. Similarly, oncotic pressure increases. Osmotic and oncotic pressure decrease as you move away from the source of inflammation. Thus, changes in the quality and quantity of tissues during inflammation cause physicochemical changes in tissues, including: hyperionia, hyperosmia and hyperonkia. The causative agent causes a short-term narrowing of the blood vessels by reflex action on the blood vessels and then dilation of the blood vessels.

The slowing of blood flow in the blood vessels is due to the following reasons:

1. Paralysis of the vascular neuromuscular apparatus causes loss of vascular tone.
2. Causes excessive dilation of the vascular surface.
3. It causes the blood to thicken and become sticky.
4. Slows down blood flow as a result of cutting blood vessels with fluids in the surrounding tissues.
5. Due to the adhesion of leukocytes to the inner wall of blood vessels, the unevenness of the inner surface of blood vessels is formed, and sometimes clogging with thrombi leads to a slowing of blood flow.

The vascular response at the source of inflammation varies under the influence of various pathogens. For example: vasoconstrictor (adrenaline caffeine, etc.) and vasoconstrictor sympathetic

nerve effect. Slowing of blood circulation changes until complete cessation of blood flow in the arteries, leading to changes similar to thrombosis and hemorrhage. Disruption of blood circulation at the source of inflammation worsens metabolism, disrupts the nutrition of cells in the inflammatory center, and these changes themselves lead to increased inflammation.

Dilation of blood vessels and slowing of blood flow increase the permeability of blood vessels, resulting in leakage of shaped elements with liquid parts of the blood, and this process is called exudation. The fluid released is called exudate. The exudate differs from the transudate in the presence of 2-4 times the protein, shaped elements, local tissue elements, tissue breakdown products, some enzymes and other products. The process of exudation depends on several factors, the main of which are capillary permeability, high blood pressure in the vessels, osmotic and oncotic pressure at the source of inflammation.

Capillary permeability depends on the physiologically active substances histamine, bradykinin, serotonin, as well as potassium and hydrogen ions accumulated at the source of inflammation, which ions swell the blood vessel wall, dilute colloidal substances and disrupt vascular nutrition.

Healthy capillaries pass water and crystolloids, increasing permeability from colloidal substances to proteins primarily albumins (low molecular weight) substances.

In inflammation, more blood flows to the source of inflammation, weakening the bleeding and increasing the pressure in the blood vessels, which allows more fluid to leak out of the blood vessels. Such strong exudation lowers blood pressure in the blood vessels and weakens blood flow. Exudation is also affected by the osmotic and oncotic pressure at the source of inflammation.

During exudation, water, salt, protein, or cell-free products are released from the blood vessels, and then leukocytes are released from the blood vessels into the tissues, called leukocyte emigration. During leukocyte emigration, the localization of leukocytes along the walls of blood vessels occurs, resulting in the redistribution of blood-forming elements, which is associated with slowing of blood flow. In normal life processes, the blood is characterized by the placement of two layers of thin, plasma at the edges of the blood vessels and shaped elements moving in the center, the specific gravity of erythrocytes is heavy between the blood vessels, leukocytes move lightly on the periphery.

As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cytoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Remaining leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that die in large numbers in high osmotic pressure and atsedosis.

Monocytes show their resistance even at pH 5.5. While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. AS Shklyarevisky, a proponent of the mechanical theory that explains the location of leukocytes along blood vessels, explains that leukocytes are pushed aside by other shaped elements because of their light weight.

Proponents of the second type of this theory explain that leukocyte emigration is a passive process in which leukocytes flow out of the general fluid flow and remain outside the blood vessels. If this is the case, then why do neutrophils come out in one case, lymphocytes and monocytes in the other. Thus, without mechanical factors playing a major role in the location of leukocytes along the vessel wall, this theory cannot explain the formation of these processes. Because the location of leukocytes along the walls of blood vessels is a complex biological process, the active processes in which leukocytes approach the wall of blood vessels, push it out of the blood vessels and participate in phagocytosis.

According to IIMechnekov's biological theory, leukocyte emigration is called a positive hemataxis feature. Positive chemotaxis properties include staphylococcus, streptococcus and other substances that are formed as a result of their activity, as well as products of nucleic metabolism, some globulins, liver and kidney proteins, meat peptone broth, some medicinal substances.

The repulsion of leukocytes from these chemicals is called negative chimataxis, and the negative chymataxis property is characteristic of quinine, chlorochrome, benzene, alcohols.

The development of physkaloid chemistry leads to the emergence of a new direction that explains the emigration of leukocytes, i.e. leukocyte emigration is associated with physicochemical changes in tissues.

Increased metabolism in the inflammatory center results in the formation of completely unoxidized substances, leading to an increase in N ions. Thus, due to different charges, negatively charged leukocytes move towards the center of positively charged inflammation. Leukocyte emigration is also caused by the continuous release of fluid from the blood vessels into the inflamed parts. Energy processes in leukocytes also play an important role in leukocyte emigration. On the side of leukocytes facing the source of inflammation, the protoplasm melts to form pseudopodia and amoeba-like action due to the energy generated during the metabolism of leukocytes. Emigrated leukocytes partially die under the influence of the environment at the source of inflammation, while others are actively involved in the process of phagocytosis. While the process of phagocytosis is influenced by the tissue environment and physiologically active substances, the acidic environment and alkaline environment inhibit the process of phagocytosis, while the normalization of the environment ensures the normal course of phagocytosis. Thus, leukocyte emigration is an active biological process in which mechanical and physicochemical changes play an important role.

Proliferatsiya jarayoni yallig‘lanishning barcha davrlarida hosil bo‘lib, alteratsiya kechayotgan davrda kam miqdorda bo‘lsada to‘qima hujayralari ko‘payib o‘zining eng kuchli ko‘payish davriga yallig‘lanishning oxirgi davrlarida etiladi. To‘qima hujayralarni ko‘payishini kuchayishini parchalangan mahsulotlar va to‘qimalarda moddalar almashinuvini buzilishidan hosil bo‘lgan moddalar hamda patogen agentining o‘zining ta’siridan hosil bo‘ladi. To‘qima va hujayralarni tiklanishida yallig‘lanish markazidagi RES hujayralari ya’ni qon tomirlar endoteliyasi, advintitsiyasi, fibroblastlar, gistiositlar, fibrotsitlar va qon tomirlari orqali emigratsiyalangan monotsitlar ishtirok etadi. Hujayra elementlari harakatchan bo‘lib fagotsitoz jarayonida ishtirok etadi. Bularni makrafaglar deyilib, ularga Ranve plazmatsitlari, poliblastlar, Maksmovning tinchlikdagi adashgan hujayralari, turli gistiotsitlar kiradi. Yallig‘lanish manbaida hosil qiluvchi plazmatik hujayralarni parchalanish mahsulotlarini fermentativ yo‘l bilan emiradi.

After the process of proliferation, the process of regeneration develops, the growth of connective tissue, blood vessels, connective tissue proliferates and glandular cells are regenerated. Young fast-growing connective tissue is rich in blood vessels and is called granulation tissue. The connective tissue grows from the periphery to the center, creating a barrier between healthy tissue and inflamed tissue, preventing microorganisms from spreading from the source of inflammation to the body. Upon completion of the inflammation, interstitial fibrous substances are formed in the granulated tissue, the blood vessels shrink, the young mesenchymal cells stop growing, and eventually a dense connective tissue chandelier is formed. The resulting scars cause various dysfunctions, including esophagus, stomach, if it is formed in the urinary tract, it causes them to narrow, the mobility of the joints changes, and so on. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles

recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little sluggishly. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little sluggishly. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed.

Yallig‘lanish morfologik va etiologik belgilariga qarab bir necha turlarga bo‘linadi. Yallig‘lanishning morfologik belgisiga karab alterativ, ekssudativ va proliferativ xillarga bo‘linadi.

Alterativ yallig‘lanish davrida to‘qimalarda distrofik va nekrobiotik jarayonlar, ekssudatsiya va proliferatsiya jarayonlariga nisbatan kuchli rivojlanib bu turdagi yallig‘lanishlarni turli zaharli moddalardan bakteriya toksinlari, ba’zi bir tuzlar ta’sirida parenximotoz organlardan buyrakda, jigarda, yurak va kam xollarda miyada uchraydi.

Ekssudativ yallig‘lanishda ekssudatsiya va emigratsiya jarayonlari boshqa jarayonlardan ustun turib, ekssudat turiga bog‘liq holda serroz-zardobli, kataral-shilliqli, fibrinli, yiringli, ixoroz yallig‘lanishlar farq qilinadi.

Seroz yallig‘lanishlarda suyuqlik tiniq, sarg‘imtir rangli, solishtirma og‘irligi 1,018-1,-20 tarkibida 5-6% oqsil va kam miqdorda shaklli elementlar saqlaydi. Qon tomirlar reaksiyasi to‘liq rivojlanmay to‘qima kam parchalanib ekssudat tez so‘rilib faqat plevra va qorin bo‘shlig‘ini yallig‘lanishi bir muncha qiyin kechadi.

Catarrhal inflammation is a mixture of serum and mucous substances, which is more pronounced at the level of the mucous membranes, and leukocytes are less in the exudate. In fibrinous inflammation, the exudate is high in fibrin, which indicates an increase in vascular permeability. As a result, in addition to albumin and globulins, fibrinogen leaks into the interstitial fluid, forming fibrin fibers and membranes, which coagulate. Diphtheria is when the fibrin sits flat between the tissue and on the surface, moves hard on the surface of the organ, and forms a wound.

During inflammation, krupoz inflammation is when fibrin sticks to the surface of the tissue and between them and moves easily without forming a wound.

Purulent inflammation occurs in all parts of the body, with the accumulation of pus in the inflamed parts. This fluid contains a large number of leukocytes, tissue fragments with a high specific gravity. Purulent exudates fill the space in the interstitial space and form an abscess or abscess, inflammation of the sebaceous glands and hair follicles-boils, inflammation of a group of fat and wool bulbs is called carbuncle.

When putrefactive bacteria enter the inflamed parts and dissolve the tissue, the ulcer is called dissolved inflammation and is well manifested in alteration processes.

In hemorrhagic inflammation, the exudate becomes red due to the retention of erythrocytes. Vascular permeability results from acute and severe infectious diseases and poisonings.

In proliferative inflammation, cell proliferation increases oncotic pressures above other processes.

During exudation, water, salt, protein, or cell-free products are released from the blood vessels, and then leukocytes are released from the blood vessels into the tissues, called leukocyte emigration. During leukocyte emigration, the localization of leukocytes along the vascular walls occurs, resulting in the redistribution of mine-shaped elements, which is associated with slowing of blood flow. In normal life processes, the blood is characterized by the placement of two layers of thin, plasma at the edges of the blood vessels and shaped elements moving in the center, the specific gravity of erythrocytes is heavy between the blood vessels, leukocytes move lightly on the periphery.

As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cetoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Intact leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that degrade in large acidic environments and under osmotic pressure

Neutrophils exhibit their resistance at pH 5.5.

While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. According to AS Shklyarevisky, one of the proponents of the mechanical theory explaining the location of leukocytes in the blood vessels, the specific gravity of leukocytes is light, including inflammation of the connective tissue at the site of inflammation, sepsis, actinomycosis, proteinuria and other diseases. 'sib, granuloma is formed, resulting in the passage of toxins and microorganisms from the inflamed area to healthy tissue. Biologically active substances released from leukocytes and other cells, as well as changes in osmotic and oncotic pressure in inflamed parts play an important role in the occurrence of proliferative processes. These modes tickle the receptors in the injured parts by the reflex pathway.

Depending on the immunobiological reactivity of the organism, normergic, hyperergic and hypergic inflammations are distinguished.

Normergic inflammation is caused by the primary exposure of microbes or toxins to organisms that are not sensitized and have normal immune properties. Hyperergic inflammation occurs after repeated exposure of the body to the cause of the disease. This inflammation is accompanied by a strong acute flow, alternating and exudative processes. Changes in this period do not depend on the strength of the

antigen, but rather on the increase in the sensitivity of the organism. Alterative changes in hyperergic inflammation begin with fibrin bending and necrosis of halogenated and smooth muscle fibers. The fibrin in the exudate is hemorrhagic because it is a mixed serum. Examples of local allergies to hyperergic inflammation are pulmonary embolism and infectious inflammation in acute rheumatism.

Hypergic inflammation is slow, weak. Hypergic inflammation occurs in organisms that may have immunity to this antigen, or are very weak, emaciated, and less reactive. For example, if a diphtheria toxin is injected into the skin of an animal vaccinated against diphtheria, a very slow local change occurs. Such a sluggish response is observed due to decreased reactivity in animals with strong lean and malignant tumors.

Why does inflammation manifest as a general organism change?

Yallig‘lanish manbai bilan organizm o‘rtasida o‘zaro aloqadorlik va bir-biriga ta’sir etish hosil bo‘lib turadi birinchidan yallig‘lanishning hosil bo‘lishi va rivojoanishi organizm reaktivliligiga, uning boshqaruvchi mexanizmi, moddalar almashinuvi va boshqalarga bog‘liq ikkinchidan yallig‘lanish manbai organizmdagi moddalar almashinuvi, immunologik xususiyatlarga ya’ni barcha organizmga ta’sir qiladi. Sensibilizatsiyalangan hayvon organizmga zaharli bo‘lmagan qo‘zg‘atuvchilar bilan ta’sir etilganda kuchli giperergik yallig‘lanish kelib chiqishini, immunlangan organizmlarda zaharli moddalarga xos yalig‘lanish jarayonlarini chiqaradi. Yallig‘lanishning shakllanishida nerv reflektor jarayonlar muhim ahamiyatga ega. Masalan: retseptorlarni blokada qilib yallig‘lanishni susaytirish yoki umuman hosil qilmaslik mumkin. Nervsizlantirilgan to‘qimada yallig‘lanish juda sust va belgilarsiz kechadi. Simpatik nervning qo‘zg‘alishi yalig‘lanishni susaytirsa, parasimpatik nerv kuchaytiradi. Oraliq miyadagi kulrang do‘mboqchaning uzluksiz qo‘zg‘atilishi organizm turli qismlarida: terida, ichki organlarda keng yallig‘lanish jarayonini chaqiradi. Hayvonlar narkoz xolatda, qishqi uyqu vaqtida va po‘stloq tormozlanganida harqanday kuchli qo‘zg‘atuvchi ham yallig‘lanish chiqarolmaydi. Hayvonlar organizmining murakkablashishi, nerv sistemasining diferensiyalangan bo‘lishi, ularda yallig‘lanishni to‘la belgilari bilan aniq kechishiga, organizmning ximoyaviy xususiyatlarida fagotsitoz, leykotsitlar emigratsiyasi va proliferativ jarayonlar yaqqol kechishini ta’minlaydi.

Inflammation is also affected by the endocrine glands, while thyroxine, aldesterone and somatotron hormones increase inflammation, while AKGT, cortisone and sex hormones histamine, acetylcholine, serotonin and others.

Inflammation depends on the age, type, constitution, sex, and other characteristics of the animal, and hyperergic inflammation cannot occur in young animals. If the signs of inflammation are well manifested with the age of the animal, in old, loose constitution, inert nerve-type animals, inflammation is slowed down and conditions are created for the spread of the pathogenic agent in the body. Inflammation of the abdominal cavity of horses is more acute and severe than in cattle, or if we send tuberculosis rods under the skin to guinea pigs, they form a long-term incurable wound at the injection site. calls. The development of inflammation depends on the anatomophysiological structure of the organism, if the inflamed parts are well supplied with blood vessels, the inflammation will be so strong and, conversely, if the blood vessels are poorly supplied, the inflammation will be asymptomatic. Inflammation is affected by animal nutrition, metabolism, low protein content in the diet, reduces the formation of immune cells in the body of the animal, weakens the resilience of patients, vitamin A deficiency from avitaminosis can lead to easy inflammation of the eyes and respiratory tract. causes. The intensity of inflammation varies in different vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different vitamin deficiencies.

How does the source of inflammation affect the body?

Yallig‘lanish organizmning mahaliy qon tomirlar reaksiyasi sifatida nomoyon bo‘lishiga qaramasdan, organizmning umumiy xolatiga, moddalar almashinuviga, immunobiologik reaktivliligiga, qon tarkibiga, termoregulyasiya va jarohatlanmagan to‘qimalarga ta’sir qiladi. Yallig‘lanish davrida moddalar almashinuvining buzilishidan, glikoliz jarayoni kuchayib qonda qand miqdorini ko‘payishiga, albumin-globulin indeksini o‘zgarishiga, globulinlarni ko‘payishiga, qonda qoldiq azotni, albumoz-peptonlarni, gistamin, nukleinlar almashinuvining oraliq mahsulotlari va atseton tanachalarini ko‘payishiga olib keladi. Qonda leykotsitlar ko‘payadi, ECHT tezlashadi, tana harorati ko‘tariladi. Immunobiologik reaktivlik yo immunitetni hosil bo‘lishini kuchayishi yo pasayishi bilan harakterlanadi: emlash va kasallikdan tuzalgandan keyin antitela hosil bo‘lishi va fagotsitoz kuchaysa, surunkali kechadigan yallig‘lanish jarayonida immunobiologik reaktivlik va rezistentlik susayishi madorni qurishiga olib keladi. Yallig‘lanish manbai o‘ziga yaqin to‘qima va organlarga ta’sir qilib hayvonlar qorin bo‘shlig‘iga filogen moddalar ta’sirida qorin devoriga yuborilgan mikrobga turg‘unligi kuchayib, bu mahalliy to‘qimalarni immunologik xususiyatlarini kuchayishidan hosil bo‘ladi. Yallig‘lanish manbailarini jarohatlanmagan to‘qimalarga ta’sirini ba’zan organizmdagi qorin sohasining yallig‘lanishi appenditsit yoki aritmiyalarini hosil bo‘lishida ko‘rish mumkin.

The inflammatory center affects the whole organism, affecting its metabolism, reactivity, uninjured organs and systems due to the microorganisms accumulated in these inflamed parts, their breakdown products, toxins, biologically active substances that are absorbed into the blood and tickle the receptors. . The body is also affected by painful stimuli coming from the source of inflammation. The increase in body temperature is caused by the effect of completely undigested substances formed in these parts on the thermoregulatory center in the midbrain. Thus, the source of inflammation affects the body through nerve reflex and neurohumoral pathways.

What do you mean by the mechanism of development of inflammatory processes?

It is a complex reaction of the organism to inflammatory influences that appeared very early, and theories explaining these processes have also been known since very ancient times. The protective properties of inflammation are also stated in the ideas of Hippocrates, who have different views and worldviews on the essence of inflammation.

According to R. Virkhov's 1958 theory of nutrition, inflammation is the transition of cells to a high functional state under the influence of inflammatory factors, a state of intensive consumption of nutrients. However, cells not only undergo a high functional state under the influence of a phlogogenic agent, but also under a high functional state during other effects. R. Virkhov equated inflammation with a simple arousal phenomenon and could not explain that arousal is another qualitatively specific phenomenon. If the proliferative and exudative processes in inflammation are considered a high functional state, the alternative process cannot be considered as such. By binding the inflammation to the cell,

Congeym's theory of vascular changes in 1885. It is said to cause changes in the blood vessels leading to inflammation. Congeym says that the changes that occur in inflammation are due to increased vascular permeability, i.e., exudation and emigration. This theory ignores the fact that other tissues, not blood vessels, play an important role in the development of inflammation. The fact that there is an inflammatory process even in animals with underdeveloped vascular systems did not take into account the fact that vascular permeability is controlled by the nervous and humoral systems.

In Ricker's vasamotor theory, inflammation is explained as a phenomenon associated with changes in the vasomotor nerves under the influence of a phylogenetic agent. Inflammatory nerve exposure causes changes in vascular permeability and tone, leading to the formation of inflammatory-specific metabolic changes in tissues. In this theory, the interaction between the flogen agent and the tissue is ignored and the role of the nervous system is limited. IIMechnikov's phagocytic theory was stated in 1892. Inflammation is a protective reaction formed as a result of evolutionary development, in which specific cells of inflammation (RES cells) are considered active in response to the action of a phlogogenic agent. This theory suggests that vessels, other than phagocytes, are cells of the nervous system,

In Shaden’s physicochemical theory of 1923, he explained that inflammation under the influence of a phylogenetic agent disrupts tissue metabolism and alters the physicochemical properties of colloidal

substances as the main pathogenetic chain of inflammation. Inflammation is only a local process, it does not take into account the reactivity of the organism, the state of the regulatory mechanisms that play an important role in the development of inflammation. Thus, inflammation is associated with alteration, necrobiosis, venous hyperemia, stasis, intoxication, dysfunction and other events, on the one hand, arterial hyperemia with protective compensatory properties, accelerated metabolism, leukocytosis, phagocytosis, emigration, multiple antibodies. and the formation of biostimulants, proiferation,

At the end of the twentieth century, the role of the nervous system in the development of inflammation was raised. Samuel recognizes and promotes the importance of the nervous system, saying that neurotrophic processes play an important role in the origin of inflammation, that the influencer affects the cell through the nervous system.

While V.Ya. Danilevsky cut the sympathetic nerve and observed strong inflammation in the tissue controlled by this nerve, Ricker explained that inflammation is caused by dysfunction of vasomotor nerves, and these theories led to the notion that inflammation occurs in the organs. will come.

Only IPPavlov tries to explain that with the development of the theory of nervousness and its role in the nutrition and metabolism of the nervous system is important, that inflammation develops on the basis of important laws. IPPavlov observes that wounds on the skin and mucous membranes of dogs with tubes are formed under the influence of chronic pathogens. These chronic movements are caused by improper placement of the tubes. Inflammation is provided only by the injured nerve and has been observed in other organs or tissues as well, not only in the tissues. For example, inflammation of the cornea of the eye was observed when the sciatic nerve, the cervical sympathetic node and the gray ball and some centers were stimulated. The effect of the cerebral hemisphere on the inflammatory process, when the bark is removed or the animal is anesthetized, the inflammation is sluggish and goes unnoticed. Similar changes are not caused by inflammation during the hibernation of animals, in severe poisoning (mustard, when large amounts of leucites are introduced into the body). Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body.

Control questions

1. What is inflammation?
2. What are the main pathological changes observed during inflammation?
3. What are the causes of inflammation?
4. What are the external local changes that occur during inflammation?
5. What is the morphological classification of inflammation?
6. What is the classification of inflammation according to the composition of the exudate?
7. What is the classification of inflammation according to the immunobiological properties of the organism?

O 'SMALA R.

Plan:

1. The concept of tumors. Types, etiology, pathogenesis of tumors.
2. The main features of malignant and benign tumors. Distribution and biological properties of tumors in the animal kingdom. Anaplasia: morphological, biochemical, physicochemical, energetic.
3. Metabolism in tumors. Interaction of the organism with the tumor. Theories of tumor etiology.

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BASIC EXPRESSIONS.

Tumor, oncology, oma, histoid, orgonoid, sarcoma, cancer, metastasis, recurrent, infiltration, Hippocrates, treatable, incurable, phosphorus plant, turmeric disease, chemical, exposure, embryonic bulge, virus, maleg nization, anaplasia, glycolysis, RVKavetskiy, LAZilber, MKPetrova, AABogomolets, carcinolysis, B.Zbarskiy, Stern, Wildheim, Rondoni, MANavinskiy, Ivan Polikarpovich Mishchenko, anemia, Raus, ADTimofeevskiy.

Tumors are defined as the growth of tissues in a stable pathological state, the inability of tissues to have specific biological properties, unlimited growth and control, and changes in the structure and function of tumor cells. These properties in tumor tissue are caused by the influence of external and internal environment on disease-causing causes in healthy cells in the body. Tumor tissue, unlike other pathological changes in the tissue, does not have the properties of regeneration and flexibility (regeneration, hypertrophy, proliferative inflammation) in the body. Not only does the tumor increase in size when the tumor grows, but the tumor can also break down the surrounding tissue.

The branch of pathological physiology that teaches the problems of tumors is called oncology-Greek-oncos-tumor or neorlasma-new abnormal formation, Latin-tumor-tumor. Tumors can form and develop from healthy tissues in the body (epithelial, connective, muscle, nerve). Tumor-forming substances are called carcinogens. The transformation of healthy cells into tumor cells is called malignancy. Tumors are formed by adding a suffix "oma" to the name of the tissue from which they are formed: For example: epithelioma, fibroids, lipoma, osteoma, chondroma, adenoma and others. Some tumors, as they are called by their historical name, are called malignant tumors (sapsech, sachstpoma) formed from epithelial tissue and malignant tumors formed from connective tissue. Tumors have a parenchyma and a stroma, and the characteristics of the tumor depend on its parenchyma. Blood vessels and nerves pass through the tumor stroma and are composed of connective tissue. Because malignant tumor stroma is so poorly developed, these tumors are called histoid tumors. In benign tumors, the stroma is well developed, surrounded by a thick shell, and is called an orgonoid tumor, reminiscent of a parinchymatous organ. If the tumor parinchyma is composed of multiple tissues. These tumors are called mixed tumors. Hence, we study all tumors into two groups i.e.

malignant and benign tumors. Malignant tumors include cancer and sarcoma, all remaining tumors include fibroids, fibroids, ostiomas, chondromas, adenomas, and other benign tumors.

Safe tumors are called tumors that are close to the mother cell and mature due to their morphological structure. As benign tumors grow, they grow from the center to the periphery, enlarging to form a connective tissue shell and compressing the surrounding tissue as they grow. Because benign tumors have a connective tissue shell that is confined to the surrounding tissue, they grow slowly and sometimes temporarily stop growing. In dogs, the size of the tumor increases and the dogs become 1/3 of their body weight. The expansion of a tumor without growing into other tissue is called expansive growth. Safe tumors do not recur and metastasize when surgically removed because they are surrounded by a good connective tissue shell. Of course, a safe tumor is a relative concept. the formation of this benign tumor in the brain leads to disruption of the activity of various nerve centers by squeezing the brain. Safe tumors that form from the endocrine glands cause the production of many hormones and disrupt the functions of the endocrine glands. Safe tumors grow around the red eyelids and other tubular organs, squeezing them, causing dysfunction.

Malignant tumors grow rapidly, irregularly, and are not limited to the surrounding tissues, but grow into them and are called infiltrative growths. Malignant tumors injure the surrounding tissue. The central part of malignant tumors disintegrates without good nutrition and does not become large in size. Tumor growth is variable, sometimes rapid, sometimes slower than in benign tumors. When malignant tumors grow, there is no boundary between the tumor and the healthy tissue, so the malignant tumor cannot be separated from the body. If the sma cell remains, it recurs. Recurrence is a characteristic feature of malignant tumors. A recurrent tumor can form long after it has been removed. Malignant tumor metastasis-Greek metastasis - displacement, interference, which causes tumors to grow into the blood and lymph vessels, staring at the capillaries and forming an embolus. Cancer often metastasizes through lymphatic vessels. Wherever tumors develop when they metastasize, they retain the characteristics of maternal tumors. For example, regardless of which part of the body the hepatoma is formed, it produces urethra, a tumor formed by the thyroid gland is rich in iodine. The formation of metastases depends on which blood vessel the embolus flows through. For example: If the cancer has developed in the stomach, it metastasizes primarily to the liver. In other cases, the formation of metastases depends on the biochemical properties of the tissue in which the metastasis occurs. If you have lung cancer, metastasis will form in the brain and adrenal glands. Thyroid, malignant tumors of the prostate and mammary glands often metastasize to bone tissue. However, the entry of tumor cells into the organs does not always lead to the formation of tumors because they are broken down by macrophages. For example, the flow of cancer cells in the spleen does not cause metastasis. Malignant tumors are so different from benign tumors that in malignant tumors the metabolism changes more deeply than in benign tumors, causing the animals to lose weight.

Tumors are found in all farm and domestic animals, birds, amphibians, and fish. It is even found in various invertebrates as shown in the literature. Sarcoma from malignant tumors in cattle, lipoma, fibroma, ostioma from malignant tumors, melanosarcoma, osteosarcoma and cancer from more dangerous tumors in horses are found in cattle. Tumors in the genitals and other parts of bulls and stallions are more common. Tumors rarely form in the stomach and uterus of animals.

Tumors are more common in older animals. Dogs of purebred and older than 5 years of age have a variety of tumors, most commonly tumors of the genitals and mammary glands. Tumors are rare in rabbits, and tumor damage is very rare in guinea pigs. While laboratory animals are more likely to develop cancer in mice, sarcoma is more common in rats. According to some data, 6-8% of mice die from cancer. Tumors also occur in chickens, where they develop sarcoma. Similarly, geese and ducks are also affected by tumors. In birds, malignant tumors grow and metastasize. In fish, as in other vertebrates, epithelial and connective tissue tumors are different. Tumors are more common when fish are artificially bred and are less common in free-living fish.

Tumor formation also depends on the age of the animals, with tumors occurring in humans after the age of 40, in dogs after the age of 5, in chickens at the age of one year, and in older animals 10%. The occurrence of tumors in older animals is associated, firstly, with the long-term effects of etiological causes, and secondly, with a decrease in the body's protective functions.

The importance of hereditary traits in the origin of tumors has not yet been definitively studied. However, cancer is caused by viruses, and if an animal is born with cancer after birth, it will develop cancer. This condition is well studied by infecting the animal's udder with the virus.

3. The causes of tumors have not yet been fully studied, and the first information about tumors dates back to 1500-2000 BC in ancient Egypt and Rome, and Hippocrates in those days. tumors can be treated or untreated. In the seventeenth century in England in the cleaners of factory pipes - a disease of pipe workers, in the United States - tumors in the clockmakers of a phosphorus plant. In the first half of the 19th century, œcìàëàð was found to be composed of cells, like other tissues, and the origin of tumors has been explained by various theories. One of these theories is the theory of embryonic buds, in which Congeim argues that during the embryonic development of an organism, some of the cells fail to develop, and that various causes, strikes, due to inflammation and other causes, growth energy is formed in cells that live in secret and begin to grow. Tumor feature is formed. Tumors begin to form. Proponents of this theory explain that tumors and embryonic tissues have morphological similarities, that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor.

R. Virkhov's theory of exposure was developed in 1885 and explains that it is caused by the action of long-term pathogens on tumors, resulting in the formation of lesions in many tissues. This theory explains that tumors are formed in humans and animals in the processes of tissue breakdown, inflammation, and regeneration due to long-term mechanical, thermal, chemical, and other effects. It is said that cancerous tumors are formed as a result of long-term exposure of certain parts in people performing the same functions, from proliferative inflamed parts to the differentiation of cells. But not all formed scars and wounds form tumors. This theory seeks to explain that tumors are formed under the influence of chronic influencers of the external environment. VVPodvesotsky observed that tumors do not form when the body is exposed to mechanical and chemical agents for a long time. However, due to this theory, conditions have been created for many studies and the causes of tumors have not been identified. As a result, in 1916, Japanese scientists K. Ishikova and K. Yamagiwa discovered that tumors are caused by chemicals. They rubbed dyogt charcoal on the inside (skin) of rabbit ears for a long time, causing malignant tumors. Diagnostic cancer was later invoked from experimental animals in mice, rats, and dogs. Two weeks after the coal tar has been applied, the wool from these resinous parts falls off and new wool emerges, and after this change is repeated 6-7 times, the wool does not grow on the skin at all. the skin thickens, roughs, cracks, the outer surface of the skin sheds and alternates. If we observe these parts under a microscope, we will see acute, moderately acute and chronic inflammation of the skin after a month in the place where the coal tar was applied. 3-4 months later, sometimes earlier, sometimes later, one or more questions arise. These tumors then grow, enlarge, infiltrate, and metastasize to a cancerous tumor. Subsequent research has shown that carcinogenic chemical compounds are synthesized from various resins that cause tumors. Carcinogens are polycyclic carbohydrates with their chemical structure. Carcinogens form tumors after several latent periods after they enter our body. If left untreated, a rapid tumor can form. Cancer tumors form by the 31st to 179th days after the skin is coated with methylcholentren. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a

sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat in mice and rats from hungry animals.

Cholesterol, sex hormones, vitamin D, carcinogens in the benzperin group are chemically close and they are phenanthrene products. Some substances change their carcinogenic properties as a result of various effects. For example, cholesterol in grass can be turned into a carcinogen under the influence of radiation. NILazerev's observations show that when hormones are overproduced or a decrease in their antagonists leads to tumor formation. This means that an adequate stimulus forms a tumor when it changes in quantity. The process of cell dedifferentiation and rapid proliferation to form a tumor can lead to malignancy and tumor formation.

Impaired sterein metabolism from fats and lipids is a factor that contributes to the growth of tumors. The formation of malignant tumors under the influence of carcinogens is one of the important achievements of experimental oncology. However, the mechanisms of action of carcinogens have not yet been elucidated. Perhaps the effects of carcinogens acquire biological properties by altering the genetic properties of cells by disrupting the structure and function of nucleic acids. Even chemical theory cannot fully explain the formation of tumors. He explains that chemicals only create the conditions for viruses to affect the body.

From the end of the last century to the present day, tumors have an infectious nature, they explain the parasitic ducts that cause disease in various animals and plants, worms-worm-like parasites, fungi are specific pathogens of tumors. During the study of tumors, many microorganisms were isolated, but all of them were found to be saprophytic microbes and not related to tumors. Malignant or malignant tumors also occur when infected with certain parasites: Cancer can occur in dogs and cats when infected with Oristorshis felineus, which belongs to the class of suckers. Cancer develops when rats are fed cockroaches, or when cattle become infected with fasciola, which causes liver cancer.

The notion that tumors are caused by viruses was first proposed by II Mechnikov in 1910, and in 1911 an English scientist, P. Rose, observed that tumors were formed by sending a filtrate made from sarcoma-infected chicken tissue. P.Rous virus is found not only in tumors but also in the heels, liver, brain, blood and other fluids of chickens, the size of the virus is 01 m. Low resistance to chemical and physical influences. For example, it decomposes in 2-3 days at a temperature of 00, and in 15 minutes at 550. Antisetics have a strong effect on the virus. Some tumors can grow in an environment made of tissue. Safe tumors formed under the influence of viruses have been observed in various animals to develop into malignant tumors. For example: papilloma of wild rabbits, in dogs and cattle papillomatosis is similar to the warts that occur in humans, and the virus isolated in these animals causes tumors only in this type of animal. Most tumors can only develop in a healthy organism when

transplanted. Proponents of viral theory, such as LAZilber et al. The tumor-causing virus may not show its pathogenicity for a long time, even in all vital processes. For example, while some species of mice reach a certain age, most of them become infected with tumors, while others develop one or two tumors. Because tumors can also call a healthy animal child by suckling an infected animal, this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories.

4. Tumor growth begins with the transformation of normal healthy cells into tumor cells, and the metabolism in these cells changes. produces qualitative changes from the biological properties of the cell. Later tumors grow only due to the proliferation of tumor cells. Of course, not all tumor cells turn into tumors, some are absorbed, and some form multiple tumors.

One of the main characteristics of tumors is that they can grow continuously and, if not removed by a doctor, squeeze the animal’s organs, causing death under the influence of toxins. As a result of continuous growth of tumors, the fibroma in cattle reaches 100 cm in diameter and weighs up to 100 kg, about half the weight of the animal. In humans, uterine fibroids weigh 20-25 kg, and ovarian cysts range from 50 kg. By transplanting tumors in the same species, it is possible to ensure their growth for several years. One of the characteristic features of tumors is the transformation of tumor tissue into low-differentiated tissue.

Anaplasia refers to low-level morphological differentiation of mother cells into tumor cells, and Greek means mother-back, down, plasis-formation. In a cell that is becoming a tumor, the rate of growth and proliferation increases. The faster the growth in the tumor cell, the better the anaplasia develops. Usually morphological, biochemical, physicochemical and energy anaplasia are distinguished.

3. In morphological anaplasia, changes occur in the tumor cell and tissue, and according to the morphological features, the tumor tissue is close to the embryonic tissue. The shape and size of the parenchyma of tumor cells vary. In some cells, the normal ratio of nucleus and protoplasm is different, the number and shape of chromosomes change. The division of tumor cells is atypically malformed, disrupting the mutual arrangement of cells. For example, glandular tumors do not have or have a malformed structure that produces glandular fluid, but retains the functional properties of tumor cells despite having such an atypical structure. That is, tumors formed from melanblasts melanin, tumors formed from liver cells, tumors formed from grass, glandular cells, maintains the function of hormone production. Morphological atypicality is not specific to tumors but can also result in cell growth and

proliferation in a variety of pathological conditions. For example: During regeneration and proliferative inflammation.

4. During biochemical anaplasia, the biochemical properties of tumors change, that is, as in embryonic tissues, the amount of water increases to 90%. Potassium salts increase and calcium salts decrease from normal. The faster the tumor grows, the more the ratio of potassium and calcium changes.

Tumors increase cholesterol from lipoids. Tumors accumulate a lot of glycogen, which does not absorb glycogen well. This glycogen accumulates as a result of disruption of carbohydrate metabolism and is associated with an increase in lactic and pyruvic acids in tumors.

DNA and RNA increase in tumor tissue. As a result of the strong breakdown of nucleic acids, pentoses are formed in tumors, the amino acid composition changes, ie cystine, methyanine, tyrosine are reduced in tumors, and histidine, arginine and lysine are increased. Tumors are rich in protolytic enzymes.

4. In physicochemical anaplasia, the surface tension properties of colloidal substances are reduced, many completely unoxidized intermediates are formed, changing the acid-base balance to acidic. Osmotic pressure rises in tumors. Tumor tissue has a higher electrical charge than healthy tissue. Tissue and cell membranes have strong permeability properties. Biochemical and physicochemical anaplasia occurs in the process of regeneration or proliferative inflammation without any specific changes for the tumor. The stronger the growth of a charged tumor, the better the biochemical and physical anaplasia.

Energy anaplasia is caused by changes in metabolism and excessive metabolism in tumors, disruption of carbohydrate and protein metabolism.

5. Metabolism in tumors differs from that in healthy tissues, i.e. we can better observe these changes in carbohydrate metabolism: in healthy tissues, carbohydrate metabolism takes place in 2 periods: anaerobic and aerobic.

As a result of many intermediate changes in the anaerobic period, lactic acid is broken down - called glycolysis.

In the aerobic cycle, 1/5 of lactic acid is oxidized to SO2 and N2O, and the remaining 4/5 is converted to glucose due to energy generated by oxidation.

During glycolysis, 5% of potential energy is wasted on carbohydrates, the remainder being oxidized to form S2O and N2O from lactic acid. When the oxidizing properties decrease, a lot of lactic acid is formed, and acidic substances accumulate in the tissues. Glycolytic processes are dangerous tumors, the breakdown of glucose to lactic acid is 200 times faster than in resting muscles and 8 times faster than in maximally working muscles. Malignant tumors can produce lactic acid equal to their own weight in 10-12 hours. Therefore, the amount of lactic acid in the blood is higher in cancer-prone organisms. Glycolytic changes in malignant tumors are more active than in benign tumors. The formation of large amounts of lactic acid, changes in the surface tension of tumor tissue, etc. are characteristic of tumors. Cancer cells break down glucose 4-5 times more strongly and oxidation is very slow. Glycol = dog processes are not characteristic of tumors, because glycolytic processes occur in the retina, leukocytes in healthy life processes, increased glycolysis, decreased oxygen consumption are also observed in the process of inflammation and regeneration . Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a decrease in oxygen consumption are also observed in the process of inflammation and regeneration. Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a

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In tumors, protein metabolism is severely impaired, albumin and nucleoproteins are increased in tumor proteins, and proteins that are not found in healthy tissue are found. The formation of these nucleoproteins has not been studied, but other proteins or viruses of a different nature (LAZilber) or proteins that have been altered by the body in the formation of tumors.

In malignant tumors, full-value and full-value amino acids can also be formed. Proteins in this change can disrupt the activity of enzymes. BIZbarsky determined that specific protein synthesis occurs in tumors and is called tumoproteins.

The disruption of specific nucleic acid metabolism in tumors was discovered in 1934 by Stern and Wilheim, and later in 1941 by Rondoni in tumors where DNA was more than RNA. It has been studied that protein synthesis in tumors is superior to its breakdown by sending various identified atoms into the body. The fact that purine and pyrimidine bases from large amounts of amino acids fall into the tumor tissue and that the amount of residual nitrogen in the tumors is high indicates that the protein metabolism in tumors is faster than in healthy tissue.

The metabolism of fats and lipids is strong in tumors and varies depending on the nature of the tumor. Fats are high in unsaturated fatty acids, cholesterol and acetone cells.

Relationship of tumors with the organism. Based on the data collected in the experiments, MKPetrova explained that the effect of the body on the growth of tumors can affect the nervous system in tumors. The creation of conditions for the origin of tumors in chronic functional disorders of the nervous system (neuroses) in the animal body has been studied experimentally by calling dangerous and benign tumors. During the period of chronic functional disorders of the nervous system, the formation of tumors under the influence of carcinogens is accelerated. The role of the nervous system in the mechanism of tumor development has been observed to slow the growth of tumors under the influence of carcinogens during the hibernation of animals or the inhibition of nervous system activity, and accelerated tumor growth in controlled animals receiving so many carcinogens. If we send sodium bromine to the body, the activity of the nervous system decreases and the formation and development of tumors slows down. It is during this period that the effects of caffeine or nervous system stimulants on rabbits accelerate tumor growth.

Injury to peripheral nerves contributes to the formation of metastases. If the sympathetic nerve of the neck is cut, malignant tumors will form, which will help the transplant to grow. The effect of RES tissue on tumor growth is significant, as macrophages can break down the tumor without developing it, preventing it from growing. Macrophages resist metastasis by trapping malignant tumor fragments that enter the blood and lymph. AABogomolets and MANavinsky in 1877 observed that activation of RES tissue function prevents the transplantation of transplanted tumor tissue, or blockade of RES tissue creates conditions for the growth of transplants.

The body influences the growth of tumors through hormones produced in the endocrine glands. While one of these hormones inhibits the growth of tumors, the other accelerates the growth of tumors. For example, while somatotron hormone in the pituitary gland enhances tumor growth, hormones in the pancreas and adrenal cortex inhibit tumor growth. When we send estrogen hormones to an animal's body, a tumor develops in the animal's udder and genitals. Testosterone and progesterone inhibit tumor formation in the udder and genitals.

As the body reacts to tumors, so do tumors. The effect of tumors on the body depends on the nature of the tumor, its growth and the location chosen. If there are small tumors on the surface of the hand, they fall into the category of benign tumors, which only cause discomfort when doing any work. possible. Safe tumors compress the surrounding tissues, disrupting their nutrition and leading to atrophy. If the sap compresses the separating pathways, the sap becomes difficult to separate, and so on

Although malignant tumors are small, they degrade the body and lead to death due to impaired growth and metabolic disorders. The cause of weight loss in animals is caused by metabolic disorders, poisoning the body with intermediate products of metabolism and due to the breakdown products of tumor tissue. From it, the dysfunction of the organ in which the tumor grows also causes the body to lose weight. Tumors show antigenic properties to the organism as they begin to grow, but the structure of these antigens has not been determined, but antibodies to these antigens are formed. Antigens are sufficiently foreign, due to the lack of foreign antigenic properties, as well as the weakening of the immune-forming functions of the immune system and the low production of immunogens, which can not protect the body. The presence of malignant tumors in the body disrupts the overall metabolism. In the initial period of tumor formation, metabolism increases and decreases in the next period. Blood glucose may increase or decrease.

Increased activity of enzymes involved in carbohydrate metabolism increases lactic acid in the blood, including in the veins. A decrease in serum albumin in the blood leads to a decrease in protein and an increase in residual nitrogen. Decreased albumins are associated with decreased protein synthesis. When tumors grow, the activity of arginase, catalase, oxidase in the liver decreases, glycogen synthesis, urea, guipuric acid formation is impaired, the total amount of nitrogen excreted from the body increases, and urinary urea decreases. In the urine, lactic acid, polypeptides, some amino acids increase, and acetone cells appear. According to NBMedvedev, in cancer, carbohydrates are 6-7 times more than nitrogen. Tumors cause hypochromic anemias in the body, decreases to 0.5 to the color index of the blood. Anemia is caused by the breakdown of erythrocytes under the influence of various charged substances, ie not completely oxidized. Disruption of the control of the activity of blood-forming organs by the formation of erythrocytes by nerves and endocrine glands leads to anemia.

During the transplants, he observed that the infinite features of the tumors were visible. Tumor strains are also present today, including the well-studied Erlix mouse cancer, Jensen’s rat sarcoma, Raus’s chicken sarcoma, and others, which have been transferred from organism to organism for hundreds of years and have existed for 50 years or more. The nutrition of the experimental animal plays an important role in transplant growth, and if the caloric content of the food is low, i.e. lysine, arginine, histidine, the growth of tumors is inhibited. If it contains a lot of carbohydrates, cholesterol and potassium in the diet, the growth of the tumor will accelerate. Liver cancer can develop even if the animal does not have enough choline in its diet. But the growth of tumors did not stop as a result of complete starvation of animals SAMMI researcher IP Mishenko observed in chickens and rats. Experiments have shown that tumors can be grown outside the body by creating special nutritional conditions, as observed by ADTimofeevsky et al. Thus, the role of the nervous system in the origin of the tumor is also important, as the causes of the tumor include chemicals, mechanical stimuli, light energies.

Control questions.

1. What is a tumor?
2. What types of tumors do you know?
3. What are tumors called?
4. What is the name of the science that studies the concepts of tumors?
5. Explain the difference between malignant and benign tumors?

HEAT EXCHANGE DISORDER.

Plan:

1. Disorders and causes of heat exchange regulation in the body. Hypothermia. Hyperthermia.

2. Etiology, pathogenesis, stages and types of fever. Changes in metabolism and organ-system function in fever.

3. The biological significance of fever for the body.

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BASIC EXPRESSIONS.

Interstitial brain, gray matter, MNS, conditioned and unconditioned reflex, hypothalamus, cortex, vitality, thermal stimulus, Krause cone, Ruffin's body, vascular tone, sweating, breathing, muscle, liver, neurohumoral system , hypothermia, hyperthermia, pyrogenic substance, exogenous, endogenous, environmental change, humidity, wind, medicinal substances, light, poisoning, injury, newborn, lean, old, animal species, poultry, pigs, cattle, four-period change, compensation, heat, poor ventilation, transport, species, breed, age, color, three-period change, fever, infectious and non-infectious fever, indifferent agent, subfebrile, febrile, hyperpertensive, fever, fluctuating, decreased, types of fever, nerves, heart, blood vessels, appetite, digestive organs, kidneys, endocrine, L.Paster, IIMechnikov, Hippocrates, IPPavlov, NFGamaley, antibody, phagocytosis, vaccination, sympathetic, parasympathetic, barrier barrier.

In the body of highly developed warm-blooded animals, body temperature changes in a very short time, and their body temperature depends on the specific condition of the animal, type, development of sweat glands, time of day, age. The temperature is not the same in different parts of the body of an animal of the same species. Relatively uniform temperature maintenance in the body is ensured by physical and chemical thermoregulatory mechanisms, a process controlled by the CNS and endocrine glands.

Heat exchange is provided by the MNS using conditioned and unconditioned reflexes. Experiments have shown that in the back of the gray matter of the midbrain is a center that controls the formation and transmission of heat. This control is controlled by the centers of metabolism, vascular tone, respiration, and sweat secretion, and these processes are related to the activity of the hypothalamus and cerebellum. Needle puncture in the hypothalamus raises the body temperature of the animal to 2.5–30. Heat exchange depends on the activity of the shell, and in animals where the shell is removed, the heat exchange is disrupted. In dogs, it is possible to control heat exchange by a conditioned reflector pathway.

The heat exchange is controlled as follows: thermally excited cold-floating Krauze flasks excite the heat-floating Ruffin bodies and transmit the effect to the MNS. From there, impulses are transmitted to various organs, altering vascular tone, sweating, respiration, altering metabolism in the

muscles and liver, and regulating heat exchange also depends on blood temperature. The pituitary gland, thyroid gland, adrenal gland, pancreas and other glands from the endocrine glands are involved in the regulation of heat exchange in conjunction with the nervous system. For example: if the body temperature rises when we send hormones or extracts of the pituitary gland, thyroid gland, adrenal glands, lower the body temperature by sending pancreatic extracts, or such changes in the pituitary gland, observed when the thyroid gland and adrenal gland are removed. As the body cools, the pituitary gland begins to secrete AKTG and the animal's resistance to the cold increases. If the center that controls heat exchange in the midbrain is injured, the body does not respond to a decrease in ambient temperature with an increase in metabolism, and vice versa. Thus, the depletion of heat exchange in the body of animals is observed when the activity of the nervous and endocrine systems, as well as the activity of peripheral organs and systems is impaired. Disorders of heat exchange are manifested in the form of hypothermia, hyperthermia and fever, all of which are caused by a violation of the control of heat exchange and are accompanied by changes in body temperature of the animal.

Hypothermia is derived from the Greek word hypo- low, terme- heat, and is characterized by a decrease in body temperature as a result of the regulation of heat exchange. Hypothermia is caused by exogenous and endogenous causes. Exogenous causes of hypothermia include a decrease in ambient temperature: humidity, increased wind, exposure to medicinal substances, and radiation poisoning.

Hypothermia caused by endogenous causes: severe blood loss, starvation, starvation, weight loss, injury to the CNS (heat exchange control center), prolonged dilation of peripheral blood vessels (shock), neonatal, other in, the activity of the center that regulates heat exchange in older animals is weakened, leading to a decrease in body temperature. Pigs cool faster than cattle because a lot of heat is generated in cattle due to the activity of the anterior chambers. Birds are resistant to cold, geese do not change body temperature at ambient temperature - 90–1020 chickens - 500, ducks - 400. Chickens are also resistant to temperature drops.

There are four periods of hypothermia:

2- During this period, the animal's body activates compensatory mechanisms that increase heat production and reduce heat transfer: narrowing of blood vessels, shrinkage, increased heat production due to muscle activity, movement and tremors, accelerated heart rate and respiration. blood pressure rises. Increases the activity of the thyroid, pituitary, adrenal glands, autonomic nervous system. General and basic metabolism, oxidation and other processes are enhanced.

3. The flexibility mechanisms of heat exchange are exhausted, heat transfer is increased, and some oxygen deficiencies are formed. But the metabolism is high and the rectal temperature drops to 29-270.

4- During this period, metabolism, cardiovascular activity decreases, respiration and rectal temperature decreases to 27-190, but during this period, if the animal is immediately warmed up, we can return to normal life processes. Cooling in the next period reduces vital processes, blood pressure, metabolism, the formation of heat completely stops, sleep is suppressed, fibrillation occurs first in the heart chambers, then in the ventricles of the heart, the heart stops working and the respiratory center is paralyzed. the temperature in the rectum cools to 12-100.

Characteristic signs for hypothermia are the weakening of the protective mechanisms of the animal organism, phagocytosis, immune formation, oxidation-reduction processes, changes in carbohydrate metabolism, the formation of oxygen deficiency. When an animal that has died from hypothermia is dissected, we see that dystrophic changes have occurred in the liver, kidneys, heart, and CNS. In recent years, artificial hypothermia has been used in surgical practice, especially in cardiac operations, to increase the resistance of the heart muscle to oxygen deficiency. During this time, the body's metabolism slows down and oxygen consumption in cells and tissues decreases. A similar situation is observed during the hibernation of animals.

Hyperthermia (Greek hyper- high, terme- heat) is an increase in body temperature of an animal as a result of a violation of the regulation of heat exchange. It is said to overheat. Hyperthermia is caused by an increase in ambient temperature, an increase in humidity without wind. At this time, heat is radiated and decomposed to the outside, which is not formed because there is no difference in

temperature between the organism and the environment. Heat transfer is a key part of heat exchange control, and even the smallest metabolism in the body ensures that there is a lot of heat and that the body temperature is kept constant. Therefore, the excess heat must be expelled from the body.

Keeping animals in tight spaces, moving them in warm rooms, in poorly ventilated vehicles, doing heavy physical work and overheating the pasture can cause the animals to overheat. The high temperature resistance and flexibility of animals depends on their type, breed, age, color, and skin coating system. Sheep are resistant to high temperatures and only after the ambient temperature is 400 and above will their rectal temperature change. The resistance of animals to high temperatures depends on the development of their sweat-sweating system.

While an increase in the ambient temperature of cattle above 300 causes an increase in rectal temperature, pigs are intolerant to this temperature due to the underdeveloped mechanism of sweating. When pigs are kept at an ambient temperature of 310, their rectal temperature rises to an ambient temperature of 0.70, causing them to die without adaptation because they do not have sweat glands. They lose steam and adapt to the heat. Excessive heat increases metabolism and disturbs rectal temperature up to 440. From small animals (piglets and calves) are heat-resistant, while chickens are heat-resistant. Under the influence of heat, the appetite of animals decreases, productivity decreases, blood composition changes, breathing and heart rate increase. The strong heat of the environment in the body causes a change in three periods.

In the 1st period, the compensating mechanisms ensure a decrease in heat generation and an increase in heat transfer. In animals, metabolism decreases, sweating increases, peripheral blood vessels dilate, blood circulation accelerates, respiration accelerates. All this increases heat transfer and ensures that the rectal temperature is maintained without rising. Increased heat transfer is associated with the passage of heated blood in the centers in the medulla oblongata (breathing, heart, blood vessels, sweat secretion, etc.). In the following periods, as a result of overheating of the organism, a second period occurs without adequate mechanisms of adaptation of the organism.

In stage II, the animal becomes agitated, pulse, respiration is accelerated, saliva excretion is accelerated, metabolism is increased, the final product is not broken down, protein is formed in the urine, rectal temperature rises to 2-30. If the heat effect still does not disappear, a third period will occur.

In period III, the activity of the nervous system decreases sharply, the heart and respiration slow down, blood pressure drops, fainting, and rectal temperature rises to 5-60. When the animal's body heats up, it stops breathing, and the heart stops beating during systole. When we examine such animals, we observe that profound changes have taken place in the parenchymal organs.

One of the conditions similar to hyperthermia is the heat stroke of the animal’s body. Such changes are observed in animals during intense muscle activity, when the temperature is high and the humidity increases. Acute heat stroke can lead to death from impaired heart function.

3. Disorders of heat exchange are characterized not only by hypo and hyperthermia, but also by the formation of fever.

Fever-fenbris is a general change of the organism in relation to the pathogenic, more infectious causes, and as a result of violation of the regulation of heat exchange in the body, the animal's body temperature rises, independent of the ambient temperature. Fever is a manifestation of disease formation, which is caused by a violation of the regulation of heat exchange, including the disruption of metabolism in relation to the causes of the disease as a secondary process in the body.

There is a difference in the regulation of physical and chemical heat exchange, while maintaining the process of thermoregulation in the body of an animal with a fever. The body that produces the fever becomes resistant to the effects of heat and cold. In a fever-producing animal, the disruption of heat exchange control depends on the type of animal, age, type of nervous system, and so on. The causes of fever are diverse, and pyro-pyrogens are substances that cause fever, and we study them into two major groups depending on their properties:

3. Causes of infectious fever - various infectious diseases.

4. Causes of non-infectious fever are protein, saline, medicated, fever caused by injury to the nervous system.

Fever is caused by the action of various pyrogenic substances on the control centers of heat exchange. Fever is hypothalamic thermal, and the delivery of these substances under the skin or into the composition of venous blood does not cause any changes. A similar situation can be caused by fever by observing the thermal pathways in the gray matter of the interstitial brain of animals or the nerve pathways leading to that part. Fever cannot be caused if the back and brain are cut apart during exposure to pyrogens. Hence, peeling is also important in the formation of fever, which can also increase injury under the influence of indifferent pathogens.

Along with the nervous system, the role of endocrine glands in the formation of heat is also important. does not participate properly. For example: removal of endocrine glands and pituitary gland, adrenal gland, thyroid gland, pancreas does not cause fever, but the endocrine glands only increase the development of fever, changing the overall biotonus of the organism, reactivity, heat exchange. affects by changing the tone of the control centers. Thus, the nervous system serves as the mechanism that initiates the formation of fever.

Depending on the degree of fever in animals with fever: in subfebrile animals the temperature rises above the upper limit of 10, in febrile animals the temperature rises above the upper limit of 20, in hyperpyretic animals the temperature rises above 30 and above. The rate and degree of fever depends on the ability of the causative agent, the reactivity of the organism, the activity of the immune system, the age of the animal, the type of nervous system, obesity, storage and nutrition.

There are three stages in the development of fever in the body:

4. Temperature rise period - stadium incrementi.
5. Maintaining a high temperature-stadium fastigil from 2-3 hours to 2-3 weeks.
6. Period of temperature decrease - stadium decrementi.

With the formation of heat in each period there is a difference in heat transfer, metabolism, activity of various systems, the reactivity of the organism. Depending on the functional state of the thermoregulatory mechanisms to the reactivity of the organism, the type and strength of the pyrogenic agent, fever occurs at different levels and in different cases. In this process, the thermoregulatory nervous mechanisms, the cardiovascular system, the respiratory system, the functional state of the sweat glands play a determining role.

Whether pyrogenic agents are always present in the body during the course of the disease. Depending on whether the thermoregulatory mechanisms work like this, the following types of fever are distinguished:

8. Permanent type fever-febris continia. The high temperature does not return to normal and causes a change around 10 in the morning and evening. In croupous inflammation of the lungs, acute anaerobic and viral diseases, the temperature may rise in the first period and fall slowly or rapidly in 3 periods.

9. Relieving or remitting fever-febris remittens. Daily changes in temperature are 10 and above in the morning and evening, due to the intense relaxation of the effects of the pyrogenic agent, which occurs in catarrhal pneumonia, sepsis and others.

10. Rising or falling intermittent-febris intermittens. In fever, the thermoregulatory mechanisms are very stable, decreasing to normal when the temperature drops to 2-30 and beyond. In acute hepatitis, people encounter malaria.

11. Tinka dryer or hectic fever-febris nectica. Body temperature fluctuates between 3-50, some temperatures fall below normal and rise again. This type of fever is observed in tuberculosis and septic processes. In animals, thermoregulatory mechanisms are formed when they are stressed, weakened, and their productivity decreases.

12. Recurrent fever-febris recurrens. Body temperature is high and normal for several days, with the pyrogenic agent intensifying from time to time. This type of fever is caused by infectious anemia in horses and recurrent typhoid fever in humans.

13. Atypical fever-febris atypica. Even if the disease progresses, the temperature does not rise, and the disappearance of the disease is accompanied by a rise in temperature, which changes several times a day. This type of fever is observed in horses' mango, sepsis.

14. Ephemeral fever-febris ephemera. It lasts from a few hours to 1-2 days. This type of fever is when vaccinated against tuberculosis and mango, after giving birth to animals, after heavy muscle work, when walking a lot in the heat, or when animals are moved in wagons. It is observed in diarrhea.

During fever, changes in the activity of the nervous system, cardiovascular system, respiration and digestion, kidneys, endocrine glands may occur. Changes occur in the nervous system that lead to disruption of thermoregulation. When the body temperature rises, the SNS is stimulated and then braked. Changes in the nervous system can also be due to the pyrogenic nature of the toxins that accumulate in the body. A characteristic change in the nervous system is caused by a sudden rise in temperature at the onset of fever. It does not cause changes in higher nerf activity as adaptation to pyrogenic substances is formed in the nervous system. This indicates that the organism is poisoned and not regenerated in the MNS. The nervous system of lean animals is impaired, The sympathetic nerve activity of the VNS increases. Changes in temperature rise in young animals are stronger than in older animals. Circulatory disorders are characterized by the redistribution of blood in the body, which causes more blood flow to the internal organs and less in the skin, and later the blood vessels in the skin dilate and more blood flows. The work of the heart is accelerated by the rise in temperature to this maximum, which is caused by the excitation of the sympathetic nerve, the excitation of the cardiac nervous muscle apparatus by hot blood, pyrogens and toxins. Usually a rise in temperature to 10 causes the heart to beat 8-10 times faster. In diseases such as tuberculosis and meningitis, pulse formation weakens when the temperature rises, which is a sign that the disease is getting worse. Some fever develops arrhythmia, In the third period of fever the heart rhythm slows down. While blood pressure rises first, which is associated with increased heart rate, vascular spasm, in the third period, blood vessels dilate, heart rate slows, and blood pressure returns to normal. Sometimes in the third period the blood drops sharply, ie collapse occurs.

Fever changes the quantity and composition of the blood, the intermediate products of protein metabolism in the blood are residual nitrogen, acidic substances increase, alkaline reserve decreases, leukocytes either increase or decrease. ECHT is accelerating. The presence of microbial plaque and even microbes in the blood of animals with fever, the formation of antibodies, etc.

Respiration is accelerated by the excitation of the respiratory center by pyrogenic substances and toxic products contained in warm blood, depending on the activity of the heart. Acceleration of respiration is observed in anthrax, swine fever, pneumonia. Acceleration of respiration has a compensatory effect, increasing heat transfer and increasing the body's oxygen saturation.

Digestive system activity is inhibited, appetite is lost, gastric and endocrine and motor activity is inhibited, and absorption is impaired. The process of putrefaction in the intestine intensifies, gas accumulates and flatulence develops. Digestive disorders lead to the development of autointaxia and deepening of pathological processes due to impaired absorption of nutrients. Disorders of the digestive organs are associated with increased activity of the nervous system, including the sympathetic nervous system in the VNS.

In ruminants, the motility of the pancreas is disturbed during fever, the secretion into the pancreas is reduced, the acidity is increased, and the microflora and microfauna of the large intestine and microbiological processes in general are disrupted. As a result, the chewing period is broken. Hypo and atony of pre-gastric lesions develop. Food is not digested by stopping in the pancreas. In other animals, movement, motor, secretory, and absorption processes are disrupted throughout the intestinal system during fever. At this time, only water is absorbed from the intestine. During the heating period, animals should be given plenty of water and easily digestible carbohydrate foods to reduce the amount of concentrates in the feed.

There are also changes in the digestive system during the fever period, in the first period there is a lot of blood flow to the internal organs and a lot of urine, while in the second period there is a decrease due to water retention in the body. In the third period, urinary excretion increases again, and the composition of urine changes, glucose sometimes appears protein, albumen.

Sweating decreases in the first and second periods of inhibition of nerve centers, and increases strongly in the third period. Increased digestive processes have a compensatory effect, releasing fever from the body, the release of toxic and pyrogenic substances in the tissues, as well as certain products of metabolic processes in the tissues, and normalize body temperature.

During fever in the liver, the ability of machevina and glycogen production is weakened, the residual nitrogen in the venous blood from the liver increases, and in some fevers, bile secretion decreases.

From the endocrine glands, changes occur in the pituitary, thyroid and adrenal glands, the secretion of AKTG in the pituitary gland increases, and the activity of the thyroid gland increases. The amount of corticosteroids in the blood and urine increases.

Pathological anatomical changes cause dystrophic changes in the parenchymal organs, swelling of the organ, fatty infiltrations.

When there is a dystrophic condition in the organs, including parenchymal dystrophic changes, they disrupt their function, which in turn affects the process of fever. The formation of dystrophic changes in the organs occurs under the influence of overheating, infection and intoxication of the organ.

5. Metabolic disorders during fever are associated on the one hand with the rise of pyrogens in the body. In addition, fever leads to starvation from decreased intake and absorption of nutrients.

Metabolism is disturbed in various ways during the period of fever, however, the general laws specific to fever are not absent. During many fevers, an increase in metabolism, with an increase in dissimilation - an increase in heat production and an increase in basal metabolism by 5-10%, an increase in cardiac and respiratory activity - intensifies the oxidation process.

During the fever, protein metabolism changes, protein breakdown increases due to toxic and thermal factors, instead of the normal 15-20%, proteins are used as a source of 30% energy, 30% of nitrogen-fixing substances in the urine are ammonia, creatinine, urea and others. substances are separated. As a result, the body loses a lot of protein, at which time the body needs to be fed with easily digestible carbohydrates, if the fever is infectious, it is necessary to put glucose.

In chronic infectious fever, fat metabolism is increased, at which time excessive fat consumption is not only associated with fever, but also with starvation and poisoning of the animal. According to some scientists, changes in the activity of the gray matter in the midbrain, the center that regulates fat metabolism, lead to disruption of fat metabolism. Infectious and aseptic fevers are rarely accompanied by hyperglycemia, glucosuria, which is associated with a strong breakdown of glycogen in the liver and muscles and a violation of the regulation of carbohydrate metabolism.

Water - salt metabolism changes during the heating period, the accumulation in the tissues of incompletely degraded products of protein and fat metabolism, causing a lot of water retention in the tissues. Renal function plays an important role in this process, high temperature and toxins are reduced in the second period of diuresis, disrupting the filtration of the kidneys. In the third period, heat transfer, sweating, and diuresis increase, and large amounts of water are released. Salts also increase in the body as water is retained, many chlorides are retained, and many begin to be excreted in the third period. The release of phosphorus and potassium salts in fever is also enhanced by the intensification of decomposition processes in tissues.

Failure to raise or weaken the temperature during certain diseases in humans and animals has had serious consequences. Other investigators recommend the use of antipyretics during fever, given the toxicity of the organism during fever. When the problem is solved correctly, IPPavlov looks at the disease from the worldview, and if the disease simultaneously disrupts the activity of the organism, the second eliminates the cause of the disease. According to IPPavlov, when the body is affected by adverse causes, the body reacts sharply to this cause. From this process we must be able to distinguish the true disease and the physiological protective process. Therefore, considering fever as a complex process, there are mechanisms to combat the disease using real disease and physiological systems.

Control questions.

1. Where is the center of heat exchange?
2. What changes lead to a violation of heat exchange?

3.How to provide heat exchange in the body
do you know the mechanisms?
4. What is hypothermia?
5. What is hyperthermia?
6. What is a fever?
7. What are the causes of fever?
8. What are the names of antipyretics?

Pathophysiology of blood

Plan:

1. Changes in total blood volume.
2. Normovolemia, Hypervolemia (pletora), hypovolemia (olighemia), their types and mechanism of formation. Blood transfusion methods. Hemotransfusion shock.

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BASIC EXPRESSIONS:

Blood, interstitial fluid, lymph, thermoregulation, transport, correlation, physicochemical environment, blood depot, hypervolymia, hypovolemia, anticoagulants, microelements, macronutrients, hyproteinemia, hyperprotenemia, azetemia, hypoglycemia, hypoglycemia, hyperglycemia, hyperglycemia ETCH, atsedosis, alcoholism. Hypervolimy, hypovolimy, simple, polycythemic, oligocythemic, pleura, hydremia, anhydremia, blood transfusion, agglutinin, agglutinogen, Landtshteiner, Yanskiy, Bogomoets, hemotransformation shock, hematopoiesis, leukocytosis, polycystic, erythropoiesis, erythropoiesis, rez leukopimia, leukemia, platelets.

Blood, interstitial fluid and lymph together form the internal environment of the body. Oxygen and nutrients are delivered to the tissues through the blood, and the final products of the metabolic process are delivered to the digestive organs.

1. In normal single-celled animals, the ore is a load that receives nutrients through the cell membrane and excretes unnecessary substances through the cell membrane.
2. Proteins and nitrogenous substances move in the vessels of low-growing animals.
3. With the development of the animal kingdom, hemolymph is formed, and hemolymph is rich in inorganic and organic substances, which contain proteins and oxygen-carrying pigments.
4. In the organism of hot-blooded animals there is a liquid tissue deposit, the composition of which has complex and extremely important functions. The importance of blood in the body depends on its function. Blood transport in the body. thermoregulation. The physicochemical environment for cells and tissues is very important in the protection and correlation, ie the coordination of neuro-humoral

processes. Therefore, changes in the composition of the blood have a huge impact on all functions of the body.

There are several theories about the formation of blood, of which AAMaximov's unitary theory explains the formation of blood in hemocytoblasts - the mother cells of the blood, while later proponents of the duolistic theory explain that Erlix is formed in myeloblasts in monocytic sand.

Changes in the total amount of blood Depending on the type of animal, the amount of blood in the body is 4-5% of the body weight of 8 guinea pigs on horseback and up to 15% on reindeer. 55-60% of the total amount of blood falls on the liquid part of the blood (plasma), and 40-45% on the form elements of the blood (erythrocytes, leukocytes, platelets). Animals that are well fattened will have a much lower amount of blood than lean cattle. The better the muscle tissue is developed, the greater the amount of blood in the animal's body.

The bulk of the blood (around 50%) is in the blood depot. The amount of moving and stored blood depends on the functional state of the organism. The amount of blood in the body increases or decreases under various pathological influences, during which time the ratio between the liquid part of the blood and the shaped elements changes.

An increase in the total amount of blood. An increase in the total amount of blood in the body means hypervolemia or pleural effusion in Latin huper- excessive, volumen- volume, and there are simple polycythemic and oligocytic types.

2. In normal hypervolemia, the ratio between plasma and erythrocytes is almost unchanged. Under normal conditions, this type of hypervolemia does not occur. Normal hypervolemia occurs after blood transfusion, and such artificially generated hypervolemia quickly return to normal due to the breakdown of erythrocytes in the blood that are then implanted in the body after first plasma filtration (transfer to surrounding tissues).

It is not dangerous to transfuse around 60-80% of the total blood volume of this organism into the body.

3. Polycythemic or true hypervolemia is caused by an increase in the total amount of blood in the body at the expense of erythrocytes. In this type of hypervolemia, an increase in blood volume leads to hyperemia in the mucous membranes, an increase in blood pressure and hypertrophy of the heart.

The blood-forming properties of the red marrow increase — in the tubular bones, the fatty marrow is replaced by red marrow, and young erythrocytes appear in the blood. Polycythemic hypervolymia is caused by chronic oxygen deficiency.

4. In oligocytic hypervolemia, the total amount of blood increases at the expense of the liquid part of the blood, i.e., the amount of water increases. This type of hypervolemia is called serous or hydremic pleurisy. This type of pleurisy occurs in kidney disease, which causes excessive water retention in the body when drinking too much water. Hydremic pleurisy cannot be called experimentally, because no matter how much saline is added to an animal's body, the deposited fluids pass into the interstitial spaces and are expelled from the body, or a very short-term increase in blood pressure occurs. observed. An increase in water content (hydremia) in the blood can occur even without an increase in the total amount of blood. This hydremia is caused by a decrease in dry matter and protein in the blood, when there is severe weight loss (cachexia), when a lot of blood is lost,

Decreased total blood volume is called hypovolemia or oligemia, which means hypo-less, decreased, volumen-volume, and is divided into simple, pilitsetemic, and oligocytemic types.

5. In normal hypovolemia, erythrocyte and plasma ratios are unchanged, resulting in a decrease in total blood volume and excessive blood loss. Injury to the vessel wall with mechanical injury or tumor. excessive blood loss due to inflammation or wound processes can lead to hypovolemia.

Sometimes a decrease in blood can also be caused by taking blood from a donor. Older and younger animals are more susceptible to blood loss than middle-aged or adult animals, while diseased organisms are more susceptible to blood loss than healthy organisms. It is dangerous for the body when the body loses 60-70% of blood and 15-30% of blood loss when the body overheats causes death. Death occurs even if the body loses about 50% of its blood quickly and in a short time. If the amount of blood lost in the body does not exceed 25%, the blood pressure in the blood vessels

decreases for a short time and immediately normalizes due to an increase in vascular tone by reflex and the release of stored blood into the blood vessels. If the body loses more than 25% of its blood, a long-term stable blood pressure drop occurs. When there is a lot of blood loss, the number of erythrocytes decreases, oxidation processes in the body are provided by oxygen transported by erythrocytes present in the body. A similar situation is observed when the blood is thinned (hydremia), that is, when interstitial fluid flows into the bloodstream. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases.

Excessive blood loss leads to oxygen deficiency. When the nervous system is excited first, it then exhausts the centers that control respiratory and vascular tone by creating a wide-section braking. Cardiac function weakens, body temperature drops, and death occurs from paralysis of the respiratory center. Changes in body functions, hypovolemia or a decrease in total blood volume play a key role in lowering blood pressure. When blood is lost, it is important to put blood in the body, because if we put a saline solution at this time, the liquid part of the delivered solution passes from the blood vessel to the tissue.

4. In polycythemic hypovolemia, the total amount of blood decreases due to the liquid part of the blood, and the amount of erythrocytes increases per unit volume. In polycythemic hypervolemia, the absolute or absolute amount of erythrocytes is normal and the dry matter and viscosity of the blood increases. The decrease in the fluid content of the blood may be due to the body not consuming water. The strong viscosity of the blood prevents it from passing through the bloodstream, including through the capillaries.

5. In oligocytic hypovolemia, a decrease in total blood volume is associated with a decrease in erythrocytes in the blood. This type of hypovolemia can be observed in cases of excessive blood loss due to incomplete recovery of the fluid portion of the blood and some anemia and anemia.

Blood transfusion. When transfusing blood: a) lost blood - proteins, enzymes, hormones of the form elements of the blood are replaced, and the transfused blood participates in the performance of biological functions.

b) has a stimulating effect - that is, increases metabolism and blood formation.

c) increases blood clotting and stops bleeding.

g) cleanses the blood of toxins because erythrocytes and proteins in the transfused blood absorb toxins. Due to blood transfusion, blood pressure is restored, the body's stability is increased. It is used in cases of severe blood loss from burns, shock, collapse, diseases that reduce the reactivity of the organism, and general weight loss, because the blood affects various functions.

Until the twentieth century, blood transfusions were not widely used due to various tragic changes as a result of blood transfusions. The creation of the teachings of K. Landsteiner and Yansky on blood groups opened a wide way for blood transfusion.

The presence of blood groups is associated with antigenic causes in erythrocytes — isohemohagoglutinogen and antibody-isohemohaglutinins in serum. In determining blood groups, agglutinogen A and B in erythrocytes of blood are taken into account. These agglutinogens can occur in erythrocytes separately and both together or not at all. In accordance with these agglutinogens, agglutinins are also denoted by the Greek letters alpha and beta. An animal does not have similar agglutinogens and agglutinins.

Heterohemoagglutinins are also present in the blood at the same time as isohemoagglutinins.

Among the animals, the blood groups of horses are very clear, cattle, goats. in pigs and dogs, low levels of agglutinins in serum and low erythrocyte adhesion properties make it difficult to determine blood groups. Therefore, their blood will always need to be tested before a blood transfusion. To do this, take 2 drops of recipient serum on a vial, dilute 1 drop of donor blood 5 times in saline solution, and if agglutination does not occur within 10 minutes, this blood can be considered as recipient blood. If it does not resemble the recipient's blood, the donor solution will agglutinate. When solutions are gradually applied to the recipient, the agglutinating property is lost by repeatedly diluting with donor blood. Therefore, in practice, the focus is primarily on the donor agglutinogen and the recipient agglutinogen. If a large amount of blood is transfused, the recipient does not lose the agglutinating properties of the donor agglutinin and may cause shock in the body.

Hemotransfusion shock is a reaction that occurs when groups of blood are improperly placed in the body, and for the development of shock it is enough to put 80-120 ml of blood in groups that do not correspond to groups. As a result, the animal develops strong agitation, rapid breathing and heart rate - tachycardia. Decreased blood pressure makes breathing difficult, mucous membranes turn blue, vomit, urine and feces are no longer dependent on the activity of the organism. Shock often occurs within a short time, sometimes a few hours after a blood transfusion, and causes death. If the blood groups are not matched enough, the shock will pass immediately.

Some scientists explain that shock groups are formed by improper blood transfusion due to embolism of blood vessels in the brain, lungs, kidneys, while others explain that they are formed due to the breakdown products of erythrocytes in the recipient organism. Not all scientists agree with such analyzes. Experiments have shown that the mass formed by the adhesion of erythrocytes breaks down quickly without being stable and does not disrupt the activity of the organism. Even when hemolyzed blood is transfused, there is no shock in the animal's body. Academic AABogomolets binds to changes in the electric charge of colloidal substances during shock, as the colloidal structure of blood and tissue proteins plays a key role in the formation of hemotransfusion shock.

Due to improper blood transfusion, the structure of the recipient and donor proteins changes and the deposition of the protein micelles leads to a severe impairment of the body's function. This theory unilaterally explains the formation of shock.

In the pathogenesis of hemotransfusion shock is manifested as a major change in the reflex activity of the organism. When blood is burned in groups that do not match, it stimulates the vascular receptor to produce multiple impulses, creating a short-term strong excitation in the nervous system and then braking large parts. It therefore disrupts blood circulation, respiration, metabolism and other physiological functions.

Osmotic resistance of erythrocytes. EOR is the resistance of red blood cells to hypotonic solutions, and there is a difference between minimum and maximum resistance.

Minimum resistance is defined as the level of hypotension in which gamma-resistant erythrocytes break down and hemolyze. At maximum resistance, all erythrocytes are broken down, and the concentration of the saline solution is taken into account when assessing the degree of hypotension.

The resistance of erythrocytes depends on their structure, the resistance of erythrocytes in the changed form is low and hemolysis occurs. In addition, the resistance of erythrocytes to hypotonic solutions depends on the layer of lipoid protein formed on the erythrocytes. Due to the lack of lipids and phosphorus in the newly released erythrocytes, they break down earlier than the old erythrocytes. The state of maximum resistance indicates that the bulk of the erythrocytes are mature erythrocytes. An increase in EOR is observed in mechanical jaundice, in cases of poisoning with hemolytic toxins, in pathological conditions accompanied by tissue breakdown. Increased osmotic resistance of erythrocytes is also associated with the deposition of cholesterol and broken down tissue proteins in the body of erythrocytes.

Decreased EOR occurs when starving, in hemolytic jaundice, and in other diseased states of the organism.

Hemolysis is the rupture of red blood cells and the release of hemoglobin into the surrounding fluids. Blood or erythrocytes become discolored after hemolysis. Hemolysis occurs in and outside the

blood vessel. Some erythrocytes also break down due to their own death. If in the physiological state erythrocytes are broken down by splenic macrophages, in pathological cases the breakdown of erythrocytes also involves the macrophages of the liver, red marrow and other organs.

Causes of hemolysis include:

1. Infusion of erythrocytes into hypotonic solutions.
2. Heating of blood or erythrocytes 62-630.
6. Re-freeze and thaw the blood.
7. The effect of rays.
8. The effect of electric current.

The hemolytic effect of light energy occurs in the presence of photosensitizers such as eosin, fluoroacin and others.

Hemolytic effect is manifested by chemicals such as nitrite, nitrobenzene, ether, benzene, case and deoxycholate acids, and others. Under the influence of chemicals, the erythrocyte membrane breaks down, disrupting the binding of hemoglobin to erythrocyte strain. Hemolysis-causing substances include bee venom, chaen snake venom, tetanolysin, staphylolysin, and many other microbial toxins. The hemolytic effect of toxins is based on the hydrolysis and softening of the erythrocyte shell by phospholipids. Erythrocytes are also broken down by blood parasites. Specific immunoassays to erythrocytes may be the effect of hemolysins as the cause of hemolysis. Sometimes substances in the blood serum that are formed under the influence of tumors, radiation and other diseases break down erythrocytes to form autohemolysins.

From the breakdown of erythrocytes in the bloodstream, hemoglobin dissolves in blood plasma and is excreted in the urine. In the gradual breakdown of erythrocytes, hemoglobin and erythrocyte fragments are captured by RES macrophages, resulting in complex changes to form the pigments bilirubin and hemosiderin.

Multiple breakdown of erythrocytes primarily increases the excretion of bilirubin by bile, which in turn increases stercobilin in the feces and excretes urobilinogen in the urine.

Iron released from erythrocyte breakdown is stored in liver and spleen macrophages. Here, after complex chemical changes, iron is released into the bloodstream and transported to the red marrow, where it is used in hemoglobin biosynthesis.

From the disruption of the normal change of hemoglobin, excess porphyrins-red violet-colored pigment is formed, which separates with the urine and turns the urine red. Due to the sensitization of porphyrin to light, its sensitivity to sunlight is increased. There are reports of parfirinuria as an inherited disease in Shortgorn pedigree cattle. Parfirinuria also occurs when poisoned with mercury, lead and sulfonamides.

Anemia is a decrease in hemoglobin and erythrocytes per unit volume of blood. In anemia, erythrocytes undergo qualitative changes, pathological forms of erythrocytes are formed, which differ in size, shape, saturation with hemoglobin. The total amount of blood in anemia is either reduced or maintained at normal.

Classification of anemia. One of the most common classifications of anemias is to classify them according to their origin. Depending on the origin of anemia is divided into pasthemorrhagic, hemolytic, elemental and infectious types.

2. Posthemorrhagic anemia occurs when there is a lot of blood loss in the body. Acute posthemorrhagic anemia occurs as a result of sudden multiple or multiple - multiple chronic blood loss. Bleeding from blood vessels due to injury, ulceration of the intestines and stomach from internal organs, tuberculosis of the lungs, bleeding in the nasal cavity, tumor growth, bleeding as a result of childbirth, etc. is formed.

Restoration of the blood component after blood loss Normal red blood cell count is restored in a few days to 2-3 weeks, depending on the amount of blood lost by the body. Recovery of hemoglobin after extensive blood loss occurs gradually. In the blood, hypochromic erythrocytes are formed polychromatophils, reticulocytes and normocytes. The color of the blood decreases, the amount of leukocytes increases. Chronic diseases, changes in the quality of nutrition, reduce the regenerative

properties of red blood cells and cause severe anemia. Decreased red marrow activity leads to anisocytosis and paikilocytosis, and sometimes to the formation of extramedullary blood in the spleen, liver, lymph nodes.

Hemolytic-toxic anemia is caused by toxins that break down erythrocytes. Some substances break down erythrocytes, directly in the blood vessels, some break down blood cells and then break down in RES macrophages. In the origin of toxic anemias, the formation of blood and the violation of the reflex control of its breakdown are of great importance. does not cause anemia when administered.

In hemolytic anemia, bilirubin in the blood increases, urobilinogen is excreted in the urine, and sometimes free hemoglobin is also excreted. First of all, the color of the blood is suddenly higher, and undigested erythrocytes are absorbed into the body, absorbed. Blood formation is enhanced by strong breakdown of erythrocytes. In the blood there are large numbers of polychromatophiles, reticulocytes and sometimes normoblasts. The color index of the blood suddenly decreases. Due to the good regenerative properties of red marrow, the composition of the blood is quickly restored with the loss of toxic effects. In chronic hemolytic anemia, the blood-forming organ becomes tired, its activity weakens, and erythrocytes with various defects in the blood fall into the bloodstream, and anisocytosis and poikilocytosis are observed. The amount of erythrocytes in the blood decreases sharply.

4. Alimentary anemia is caused by a lack of vitamins, proteins, trace elements in the diet, cobalt and copper, ie substances involved in the synthesis of hemoglobin. Alimentary anemia has a hypochromic character and the blood color index is less than one. Alimentary anemia is observed in young animals, especially piglets. Alimentary anemia is caused by inability to assimilate nutrients well during diseases of the gastrointestinal tract.

a). Anemia caused by iron deficiency is caused by a disorder of iron metabolism in the body. In this type of anemia, not only is there a decrease in erythrocytes, but also a decrease in the amount of hemoglobin. In severe anemias, anisocytosis and poikilocytosis occur. In pigs, iron deficiency in pigs resulted in the development of anemia in piglets at 1–6 weeks and up to 70% mortality.

b). Anemia caused by protein deficiency As a result of a lack of proteins in the diet or a decrease in their absorption, the synthesis of globulin protein is disrupted and hemoglobin is not formed.

4. Infectious anemia is caused by filtering viruses in horses and other ungulates. While some scientists explain the formation of this anemia as a direct breakdown of erythrocytes under the influence of viruses, others explain that the viruses are associated with causing red marrow hypofunction. The amount of erythrocytes in 1mm3 of blood of animals with infectious anemia is reduced by 1-2 million. Anisocytosis, poikilocytosis and other changes occur in the blood. In infectious anemia in the red marrow occurs the replacement of the yellow marrow with red marrow, the formation of extramedullary blood in the spleen, liver, lymph nodes.

Regenerative and oregenerative anemia occur depending on the functional state of the blood-forming organ.

In regenerative anemia, the regenerative properties of the blood-forming organs are well manifested. As a sign of regenerative status in the peripheral blood are formed hypochromic, polychromatophilic erythrocytes, reticulocytes, erythrocyte nucleus remnants (Jolie bodies and Cape rings), normoblasts. When strong regenerative properties are manifested, the yellow marrow turns into red marrow, and in the liver spleen, extromedular blood formation occurs in the lymph nodes. Such changes disrupt blood formation and are formed from cells of the embryonic period — megoloblasts, macrocytes. Oxygen deficiency is an intermediate product formed during anemia, as a cause of regenerative processes in the blood-forming organs.

Aregenerative or hypoplastic anemia results from fatigue of the blood-forming feature of the red marrow. In hypoplastic anemia, the red marrow loses its ability to form erythrocytes, young erythrocytes in the blood decrease, the red marrow turns into yellow marrow, and has a hypochromic character. Weakening of the blood-forming organ is observed during avitaminosis, infections (tuberculosis, paratuberculosis, infectious anemia, sepsis), strong toxins, radiation sickness. Under certain conditions, any anemia can progress to a type of hypoplastic anemia. In most cases of anemia, erythropoiesis is not impaired, but leukopoiesis is also impaired.

In organisms, the compensatory mechanisms in anemia change. The function of oxygen supply to the blood is weakened, a number of flexibility mechanisms are formed: accelerated respiration, increased blood circulation and blood formation. As the heart beats faster, blood circulation speeds up and more blood flows through the capillaries over time. Accelerated and deepened respiration increases the saturation of the blood with oxygen in the lungs, increasing the formation of broken erythrocytes in the blood-forming organs. Compensatory properties are associated with the ability of tissues to fully absorb oxygen from arterial blood.

In severe hemoglobin deficiency in anemia, normal gas exchange is ensured in animals due to the activities of compensatory mechanisms. But weak movements during anemia cause a lot of oxygen demand, accelerated breathing movements, and tachycardias. Acedosis develops when there is an increase in incompletely broken down intermediates in the blood.

Polycythemia - or polyglobulia (Greek poly poly, globulus-ball, kutos-cell) is an increase in the number of erythrocytes in the blood per unit volume. Polycythemia is divided into absolute and relative types. In relative (false) polycythemia, the fluid content of the blood decreases and the number of erythrocytes does not change. This type of polycythemia occurs when sweating, severe diarrhea, diabetes mellitus, severe isthmus, dehydration and other pathological processes. In relative polycythemia, the total amount of blood is often reduced or unchanged.

In absolute polycythemia, erythrocytes proliferate due to increased erythropoiesis. In most cases, absolute polycythemia serves as a resilience reaction in the absence of oxygen to the body. Lack of oxygen increases the flow of erythrocytes from blood depots and blood-forming organs into the bloodstream. Absolute polycythemia develops when external respiration is disrupted (pulmonary emphysema, when the upper airway narrows, O2 partial pressure decreases in atmospheric air), when blood circulation is disrupted. Polycythemia also occurs when poisoned with copper, phosphorus, cobalt, arsenic. Polycythemia is a physiological condition in newborns, ie in the first days of life of calves erythrocytes in 1 mm3 of blood are 10.5 million. and a month later it dropped to 7.5 million.

Changes in white blood cells. Leukocytes, i.e. white blood cells, are formed in the red marrow, lymph nodes, and spleen. The stem cells that produce leukocytes are called hemocytoblasts, and the hemocytoblasts form myeloblasts, the primary cell of granular leukocytes in the red marrow. Lymphoblasts and monocytes are produced in the lymph node and spleen. In the blood of a healthy animal, there are many joint nuclei, and a small number of rod nuclei are found. Young neutrophils are not always present, and when blood-forming organs are tickled, rod nuclei proliferate, and in some cases myelocytes also occur.

Leukocytes include plasma cells, i.e., lymph nodes, spleen, and products of reticular and endothelial cells of the red marrow. Immune cells are formed due to the activity of plasma cells. During normal blood formation, plasma cells are found in the blood-forming organs, while in healthy animals, they are almost never found in the peripheral blood. The cytoplasm of plasma cells is stained dark orange, and the nucleus is round or oval in shape.

A leukoformula is a list of leukocyte types to determine the percentage of individual leukocyte species. In the leukoform of cattle, sheep and pigs, lymphocytes are abundant in the blood of horses, dogs and cats, and neutrophil leukocytes are abundant. White blood cells differ in type, and the leukoforms of young organisms are slightly different from those of older animals.

In determining the functional status of blood-forming organs, it is necessary to know not only the amount of leukocyte-forming organs, but also the absolute amount of leukocytes. The determination of the ratio of the main group of leukocytes in numbers is called leukocytic profile.

The main function of leukocytes is a protective function, i.e. phagocytosis. Leukocytes play an important role in the repair of damaged tissue, clearing the injured area of necrotic cells. Leukocytes produce a substance that stimulates regeneration, basophils and eosinophils are involved in neutralizing toxins. Quantitative changes in leukocytes are caused by an increase or decrease in leukopoiesis, as well as redistribution of blood in the blood vessels. As a result of dilation of blood vessels, blood flow slows down, leukocytes settle along the walls of blood vessels, and their amount in these blood vessels increases. Where blood vessels constrict and as a result blood flow accelerates, the amount of leukocytes in the blood decreases.

4.Myeloid, lymphoid leukemia and reticuloendotheliosis are distinguished depending on which part of the hematopoietic system is hyperplastic. Lymphoid leukemia is found in cattle, horses, and pigs, while myeloid leukemia is observed in dogs.

Myeloid leukemia or myelosis is characterized by hyperplasia of myeloid tissue. The yellow marrow turns into a red marrow, causing extromedular blood to form in the spleen, lymph nodes, liver, and sometimes other organs. Leukoblasts are more common in erythroblasts than erythroblasts. Myeloid leukemia is divided into leukemic and aleichemic types. In leukemic myelosis, the number of leukocytes in 1 mm3 of blood can be a hundred thousand or more. The main part of leukocytes, ie 90% and more, are granulocytes. The bulk of granulocytes are young cells, ie myelocytes, promyelocytes and myoblasts, and sometimes the number of unexposed eosinophils, basophils and erythroblasts also increases. In aleukemic leukemia, the number of leukocytes is increased around the norm and or in very small amounts. Examination of the leukoformula shows a strong rejuvenation of leukocytes. However, although their phagocytic properties are preserved, they are slightly lower than the phagocytic activity of mature neutrophils. In myeloid leukemia, the spleen becomes enlarged.

Some scientists attribute the formation of extromedular blood in leukemia to the introduction of myeloid cells into tissues and the formation of metastases, while others explain that the formation of extromedular blood is caused by the influence of etiological causes of leukemia on mesenchymal cells.

There are leukemic and aleukemic types of myeloid leukemia. In leukemic leukemia, the number of leukocytes in 1mm3 of blood reaches 100,000. The main part of leukocytes is granulocytes, which account for 90%. Granulocytes are composed of young cells - myelocytes, promyelocytes, sometimes non-myeloblastic eosinophils, basophils, erythroblasts. In aleukemic myelosis, the leukocytes in the blood increase normally or very little. In leukoform, young cells are weaker than phagocytosis in neutrophils, whose main part is phagocytic function (myelocytes, etc.).

During lymphoid leukemia or lymphodenosis, lymphoid tissue grows and is characterized by enlargement of the lymph nodes, spleen and liver. As leukemia develops, the myeloid tissue is replaced by lymphoid tissue in the red marrow. During leukemic lymphodenosis, the amount of white blood cells in 1 mm3 of blood reaches 1.5 million, and lymphocytes make up 98% of all leukocytes. In aleukemic lymphodenosis, the number of leukocytes is normal or partially increased, lymphocytosis develops in the leukocyte formula, and lymphoblasts are also found among the lymphocytes.

Reticuloendotheliosis is characterized by proliferation of reticular cells in the red bone marrow, spleen, lymph nodes, and liver. There are leukemic and aleukemic types of reticuloendotheliosis. In leukemic retiuloendotheliosis, there is a strong increase in monocytes in the blood. In acute leukemia the metabolism is disturbed, the productivity of the animals decreases, anemia develops and severe weight loss occurs, in chronic leukemia the animal seems to be healthy for a long time, the animal dies from malnutrition and other diseases.

Leukemia etiopathogenesis. At present, leukemia with all its symptoms is recognized as a pathological process specific to the inflammatory process. Symptoms related to the theory of blastomatosis of leukemia include:

1. The growth of hematopoietic tissue during leukemia is not differentiated like tumor cells.
2. Changes in metabolism during leukemia are similar to those in malignant tumors.
3. Carcinogens have leukogenic properties in the experiment.
4. The therapeutic effect is due to the same substances in leukemia and tumors. (M. X-rays, radioactive phosphorus, chemicals that affect cells).

In leukemia, the leukocytes are in such an atypical state that it is difficult to consider them as this or that blood-forming element. However, the process of phagocytosis is worse than in normal leukocytes. Leukemia differs from normal tumors in the formation and growth of blood in the blood-forming organs. In aleukemic leukemia, destructive symptoms characteristic of the growth of all tumors are observed.

The causes of leukemia and tumor formation are not yet fully understood. Chicken leukemia is caused by viruses. This has been studied in leukemia by sending cell-free filtrate to healthy chickens. All leukemias can be formed by injecting carcinogens. Leukemia is caused by long-term ionizing radiation in the body, the mechanism of action of which has not yet been determined.

Changes in blood plastics. Blood plastics play an important role in platelet coagulation and are a source of the enzyme thrombocytosis. Platelets are formed in large cells of the red marrow - megakaryocytes. Therefore, the factors that affect the red marrow affect the amount of blood platelets. A decrease in the amount of platelets in the blood is called thrombopenia, which causes a weakening of the blood clotting process. In thrombopenia, the retraction of the blood clot is weakened The blood clot is soft and does not provide a tight closure of the injured blood vessel.

The causes of thrombopenia are as follows:

1. Redistribution of platelets, ie accumulation of platelets in the blood vessels of the internal organs and a decrease in the peripheral blood vessels.
2. Weakening of platelet formation in the red marrow.
3. Strong breakdown of platelets in peripheral blood.

Thrombopenia in some infectious diseases is caused by physical, chemical causes, disruption of the activity of blood-forming organs or strong breakdown of platelets.

When thrombocytosis or an increase in the amount of platelets in the blood is cured of many infectious diseases, in myeloid leukemia, anemia is formed during the recovery of blood composition, and blood clotting is enhanced.

Simultaneously with the change in the number of platelets, a qualitative change occurs, the shape changes, does not wrinkle and undergoes other changes. The agglutination property of such blood plastics is lost, and blood flow and blood clot retraction are impaired.

Changes in blood coagulation. Blood coagulation is recognized as a three-phase process as explained on the basis of modern theories. The first phase is a complex biochemical process in which active thrombokinase is formed from active tissue thromboplastins and the action of blood platelets on serum proteins. From the inactive prothrombin enzyme in the second phase: active thrombin is formed in the blood plasma. Calcium ion, active thrombokinase and plasma protein - globulin accelerator are involved in the activation of prothrombin. Prothrombin is formed in the liver in the presence of vitamin K. The liver is one of the main sites where fibrinogen is synthesized. In the third phase, fibrin is formed from the action of active thrombin on fibrinogen. As a result, fibrin filaments are formed and blood clots form. In the body, along with the blood coagulation system, there is also an anti-coagulation system, these substances are formed in the tissues and released into the blood under the control of the nervous system. Anti-coagulation systems include 1) heparin-liver physiologically active substance formed in the lungs and blood vessels, 2) fibrinolysin-plasmin, 3) protein substances that inhibit the formation of thrombin and thromboplastin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin.

The blood coagulation and anticoagulant system are two interconnected parts of the blood's coagulation system. Because these two systems are mutually balanced, the blood moves in a fluid state without clotting in the blood vessels.

Weakening of blood clotting. Weakening of blood clotting: 1) due to insufficient intake of vitamin K in the body or impaired synthesis of prothrombin and fibrinogens in pathological processes of the liver. 2) when there is a decrease in platelets in the blood - in thrombocytopenia. 3) decrease in calcium ions in the blood. 4) excessive development of the anticoagulant system in the body - heparin and others. 5) when anti-coagulants, ie substances that weaken blood clotting, are injected into the body.

When animal blood has a low coagulation property, a small mechanical injury can cause bleeding into the subcutaneous tissue, mucous membranes, muscles, and other tissues. The easiest bleeding occurs in the nose, lungs, intestines.

By treating the blood vessels with paraffin, if blood collects in the arteries, the blood becomes coagulated. A 5% sodium hydroxide solution of citric acid is often used to make the blood non-coagulating. Anticoagulants include dicoumarin and other anticoagulants extracted from the head of the leech. These substances stabilize the blood by inactivating thrombin. We can use the stabilizing properties of these substances by injecting them directly into the body or adding them to freshly drawn blood.

Acceleration of blood clotting. Accelerated blood clotting is associated with vascular injury. Blood platelets easily sink into the injured vascular wall, break down due to low resistance, and form active thromboplastin-thrombokinase. Blood coagulation can be formed by the strong breakdown of tissues by sending to the body extracts prepared from blood serum and organs. Increased blood coagulation after excessive blood loss is associated with the influx of many interstitial fluids rich in thromboplastin factor into the blood. Based on this mechanism, the delivery of calcium salts, multi-vitamin K, when hypertonic solutions are injected into the blood, increases blood coagulation. Increased blood clotting in the body can lead to thrombosis and embolism.

Changes in the biochemical composition of the blood. Minerals are ionized in the blood and are in a molecular state as well as in a state of binding to proteins from colloidal substances. Minerals are involved in blood osmotic pressure and other complex physicochemical processes. Minerals are not evenly distributed between the blood plasma and trace elements, the amount of calcium, potassium, sodium and other minerals in the blood of healthy animals is always kept the same, even when saline solutions are sent to the body.

Calcium. Ionized calcium is physiologically active, accounting for 45-55% of total calcium. Combined with non-ionized calcium mining proteins. The amount of calcium in the blood depends on the functional state of the autonomic nervous system. Calcium decreases when sympathetic nerve tone decreases, and calcium increases in blood when parasympathetic nerve tone decreases. Calcium salts thicken cell and tissue membranes.

A sharp decrease in calcium levels is caused by a deficiency of glands near the thyroid gland and causes hypoproteinemia due to the fact that part of the calcium is bound to proteins. The amount of calcium in the blood is reduced in nephritic anemia congenital paresis. Decreased calcium intake increases vascular permeability, excitability of the CNS and peripheral nervous system. Calcium intake is also caused by impaired intestinal absorption in chronic diarrhea.

Potassium. In many animals, the amount of potassium in erythrocytes is higher than in plasma, and the amount of potassium in plasma increases when erythrocytes break down. Damage to erythrocytes causes the release of potassium from erythrocytes into plasma due to increased permeability without breaking them down. The amount of potassium in the serum increases in severe diseases when the tone of the parasympathetic nervous system increases, regardless of its nature. Potassium and calcium affect the excitability of the nervous system. Deficiency of potassium in the body leads to weakening of muscle activity.

Sodium. Occurs in the blood plasma mainly in the form of chlorides, partly biocarbonate and other salts. Chlorides are reduced in the blood when sweating, diarrhea, vomiting, weight loss, impaired intestinal permeability, kidney disease. Decreased chlorides affect osmotic pressure and increase the breakdown of tissue proteins, weakening the activity of the adrenal cortex. The amount of chlorides increases in the blood during kidney disease, ie nephritis. The onset of hyperchloremia is caused by increased pulmonary ventilation, as a result of which chlorine ions pass from the tissues into the blood.

Phosphorusoccurs in the form of organic and inorganic compounds. In animals, inorganic phosphorus in the blood is reduced in pregnancy, rickets and osteomalacia. Hyperphosphatemia is caused by fever, lack of oxygen, uremia, exposure to vitamin D and ultraviolet light, as well as a lack of glands under the thyroid gland.

Ironenters hemoglobin and occurs in the form of other compounds only in 2% of cases. Therefore, iron varies depending on the amount of hemoglobin. In anemia, iron in the blood is reduced. Blood contains trace elements such as iodine, bromine, fluorine, magnesium, copper, manganese and others. The amount of micronutrients in the blood is affected by the nervous and

endocrine systems. Detection of micronutrients in the blood is important in the diagnosis of metabolic diseases.

Proteins and products of protein metabolism. Protein and its fractions are different in the blood of different animals. Some proteins combine with fats and carbohydrates to form double compounds - lipoproteins or glycoproteins. Although many proteins (e.g. enzymes) are present in very small amounts in the blood, they have very important physiological activity. Most of the blood plasma proteins are synthesized in the liver. Decreased total protein in the blood (hypoproteinemia) is caused by eating disorders (malnutrition, protein starvation). Causes of hypoproteinemia include urinary excretion of proteins, liver toxicity, excessive blood loss, severe degenerative diseases of animals (tuberculosis, malignant tumors, chronic purulent processes, etc.). In hypoproteinemia, mainly albumin function is reduced, while the globulin fraction is significantly reduced. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. leads to a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. leads to a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases.

Blood plasma increases globulins in infectious disease and starvation. After immunization, gamma globulins in the blood increase sharply. However, an increase in gamma globulins is not associated with an increase in antibody levels. An increase in non-specific gamma globulins in the blood and an increase in gamma globulins may be due to a decrease in specific antibodies, as AE Gurvich found. Decreased albumin fraction in the blood is observed in hepatitis and cirrhosis. Therefore, in patients with impaired liver function, the total amount of proteins in the blood plasma and some fractions are variable.

Residual nitrogen in the blood is the protein-free nitrogen of the blood or the nitrogenous substances that remain after the deposition of proteins in the blood is 20-40 mg%. Increased residual nitrogen in the blood (azotemia) is observed in disorders of renal, hepatic and intestinal permeability. The amount of residual nitrogen in the blood is 200 mg% and more when the renal excretory function

is impaired. In azotemia associated with renal (retention) activity, an increase in the amount of residual nitrogen occurs due to urea.

In cachexia, leukemia, and infectious diseases, the accumulation of large amounts of nitrogen-fixing substances in the blood due to the breakdown of tissue proteins causes azotemia. In hepatitis, azotemia is caused by polypeptides, which can also lead to a decrease in the amount of urea in the blood. Such a change is observed in liver disease when the deamination of amino acids is impaired, the synthesis of urea is weakened, and the transfer of ammonia salts into the blood is increased.

Accumulation of uric acid in the blood is observed in disorders of purine metabolism, gout, diseases associated with tissue breakdown, and leukemia.

There are a certain amount of free amino acids in the blood, which are intermediate products of protein metabolism. An increase in the amount of free amino acids is caused by liver disease, ie severe atrophy, poisoning by carbon tetrachloride.

Carbohydrates and products of carbohydrate metabolism.

Blood contains products of glucose, glycogen, lactic acid and other carbohydrate metabolism. The amount of glucose in the erythrocytes of most species is lower than in plasma, and this is more pronounced in pigs. Most of the glycogen is found in leukocytes. An increase in the amount of glucose in the blood (hyperglycemia) occurs when consuming easily digestible carbohydrate foods (elemental hyperglycemia), when the regulation of carbohydrates through the nervous and endocrine systems is impaired. Hyperglycemia occurs when poisoned with physostigmine, pilocarpine and other substances that affect the nervous system. The origin of hyperglycemia is in the pathology of the endocrine system, ie in the hypofunction of the islets of the pancreas Langerhans, formed in inflammation and dystrophic changes of the liver. Decreased blood glucose (hypoglycemia) is observed in chronic insufficiency of nutrition, excessive infusion or delivery of insulin into the blood, hypofunction of the adrenal hypo-thyroid gland. The manifestation of severe hypoglycemia is observed in patients with chronic cachexia.

An increase in the amount of lactic acid in the blood is observed in muscle work and pathological processes in the disruption of oxidative processes in the body, when there is a lot of blood loss, pulmonary edema, suffocation, the formation of malignant tumors. All the factors that increase the formation of lactic acid in the blood cause an increase in the amount of pyruvic acid in the blood.

Lipids. Neutral fats, lysine, cholesterol and their products are stored in the blood from lipids.

The amount of neutral fats in the blood increases during feeding. Pathological lipemia is observed at the onset of starvation, and the development of lipemia during starvation is associated with the excretion of fats from fat depots and transport to the liver. Lipemia in all cases results from the release of fats from fat depots when glycogen in the liver is depleted. Lipimia is caused by liver and kidney damage.

An increase in the amount of cholesterol in the blood is increased by food (elemental hypercholesterolemia), inflammation or dystrophic processes in the liver, leukemia, severe forms (forms) of tuberculosis, some infectious diseases, arteriosclerosis, pregnancy. Underlying pathological hypercholesterolemia is the excretion of excess cholesterol from the tissues, insufficient excretion of cholesterol from the liver and intestines, and impaired cholesterol oxidation.

Changes in the amount of phospholipids (lecithin) in the blood are caused by disorders of fat metabolism. An increase in phospholipids is associated with lipemia. An increase in ketone bodies in the blood (ketinemia, acetonemia) is observed when the body's carbohydrate intake decreases or carbohydrates are replenished in the diet at the expense of fats and proteins.

Due to the lack of carbohydrates, the breakdown of fats increases and many ketone bodies are formed. The main cause of ketonemia is a decrease in glycogen storage in the liver, during which the oxidation of fats in the liver is disrupted.

Changes in pigments. An increase in the amount of bilirubin in the blood (bilirubinemia) is caused by a strong breakdown of erythrocytes, a violation of the bile secretion function of the liver.

The formation of dissolved hemoglobin in the blood plasma is called hemoglobinemia.

Training materials for practical training

ANIMAL pathophysiology is an experimental science and consists of two words: Greek Pathos - disease, illness, logos - doctrine.

The main and main method of the science of animal pathophysiology is 'experiment'. This science seeks to teach in-depth, comprehensive study of various pathological processes, diseases and their artificial models, artificially using the method of experiments. It teaches the importance of various factors in the pathogenesis of the disease, the mechanisms of disease development, the consequences of the flow.

With the help of pathological experience, the necessary conditions are created to study the causes of diseases in the past, present and future, and this is important. In studying the glycogen-forming properties of the liver, K. Bernard studied the amount of carbohydrate in the blood that goes to the liver and is present in the blood vessels leaving the liver, and found that the blood leaving the liver is low in carbohydrates.

The glycogen-forming properties of the liver were also studied by Mering and Minkovsky, who observed an increase in the amount of glucose in the blood when they examined the blood by tying two pancreatic ducts, thereby demonstrating the importance of hormones in the body. In experiments, Peer Marie proved that hypofunction of the pituitary gland leads to stunting, and hyperfunction leads to acromegaly. American scientist Simones studied the occurrence of cachexia when the function of the pituitary gland is reduced. When the Russian scientist Lunin took two groups of mice and fed one group with artificial and the other group with natural milk, a few days later the artificial milk-fed mice lost weight, lost their growth and their hair fell out, and their skin began to change. .

Trying to determine the importance of vitamins, VVPashutin feeds rabbits with sauerkraut and observes that rabbits are susceptible to sinus disease, but cannot explain the mechanism of its development.

The hypothesis of vitamins was given in 1911 by Kazimir Funk, a Polish biochemist working in London. He isolated a white crystalline substance from rice bran that could cure the disease and called it a vitamin. Latin-Vita means life amine, a chemical compound that contains nitrogen. K.Funk believes that diseases such as scurvy, pellagra, rickets, and beriberi are caused by a lack of vitamins in the body. Studies in recent years have confirmed that most vitamins do not contain nitrogen. Nitrogen-free vitamins include A, D, E, K, C. In the past, experiments have been conducted in a short period of time, using sharp experiments.

Therefore, the experiment was developed in the hands of IPPavlov, who conducted it using chronic methods.

IPPavlov spent 10 years in the SPBotkin laboratory, where the effects of caffeine, camphor, bromine on blood vessels, in particular, affecting the heart nerves, changes in blood pressure, changes in blood pressure in dogs under the influence of drugs, suturing the carotid artery in dogs , learns.

For 20 years, IPPavlov improved the methods of fistula in the physiology of the digestive system. 'rganadi. To study the role of the nervous system in digestion, the method of esophagotomy of animals explains the reflex separation of gastric juice as a result of "lying" feeding. Based on these methods, creates a diet.

IPPavlov devoted 35 years of his life to the study of mental activity and behavior of humans and animals.

The pathophysiologist uses pathological experimentation to study the causes of the disease, determine its course, find measures to prevent the disease, and develop ways and means of treating the disease, which in turn helps the practice. In particular, in the 18th century, when French wines began to turn into vinegar, IPPaster developed a method of washing and disinfecting wine containers in boiling water. When silkworm disease occurs, it is recommended that the silkworm storage rooms be cleaned of contaminants, proving that silkworm disease is caused by microorganisms.

When Louis Pasteur grows bacteria that cause cholera (malaria) and the thermostat door is accidentally left open, a few days later, he observes that the growth of cholera microbes is weakened and when he injects this microbial wash into the chickens, the chickens do not get sick. Thus, a vaccination method is created. British scientist Fleming planted in petri dishes to study the disease-causing properties of streptococcal microbes, and when the surface was left open, fungi fell on the

planted microbe, partially killing the microbes and, based on this, the first antibiotic, penicillin, was created. So the importance of experiments is significant. On the importance of experiments, IPPavlov recommends paying attention to the following two important processes:

1. Observations should be given close attention;

2.He says we study nature by focusing on the experimental method.

The French scientist Couve says that by the method of observation we hear nature and in practice we force the opening and submission of nature.

Three different problems are studied in the science of animal pathophysiology:

1.Nosology is the general doctrine of disease. In nosology, the doctor faces two different issues: one is why the disease occurs and what is the mechanism of its development (etiology, pathogenesis)? In the origin of the disease is studied the importance of the type, breed, sex, heredity and constitution of the animal, as well as the characteristics of disease resistance - reactivity.

2.The general typical cases that occur in all diseases and underlie all diseases or are observed in their origin are studied:

a). Local circulatory disorders;

b). Inflammation;

v). Fever;

g). Hyper and hypobioses.

In the special pathophysiology part of the science of animal pathophysiology teaches pathologies of organs or systems: blood, blood circulation, respiration, digestion, liver, digestive organs, endocrine glands and nervous system.

Later he began to teach pathophysiology and normal physiology AMFilomafitsky (Head of the Department of Physiology, Moscow University). Since he was not divided into in-depth knowledge at the time, he taught only some of the symptoms of the disease, without knowing the course of the disease. It teaches the origin of diseases by linking them to divine power. Therefore, AMFilofitsky begins to study a number of diseases in practice, as it is expedient to observe and study the disease. For example: the importance of the nervous system in cough, the method of blood transfusion, transfusion of fibrin-deficient blood, reviving dogs, and writing a work in this area, he has not lost its value so far. Nutritional chemistry is studied in the laboratory of AMFilomafitsky, and in 1842 in this laboratory VABasov developed a method of inserting a tube-fistula in the stomach of a dog. AMFilomafitsky studies various pathological processes in Russia under a microscope. For example: erythrocytes from the shaped elements of the blood, observed changes in urine output during the disease. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science.

VVPashutin, based on several experiments, knowing the importance of the nervous system, opposes R. Virkhov's cell pathology and explains that the processes taking place in the cells depend on the nervous system. Experimental observation of the formation of various pathological processes in the body as a result of lack of various substances, the study of the mechanism of origin of scurvy, feeding rabbits with sauerkraut. As a result, it is concluded that the disease is caused by a lack of any additional nutrients to the organisms. Lunin then justifies the lack of vitamins. That is why VVPashutin is called the gift-pioneer of the doctrine of vitamins.

VVPashutin organizes the largest school of pathophysiologists in Russia. One of his students was MPAlbitsky (after Pashutin he was the head of the department), AVReprov was the head of the

physiology department at the Khorkov Medical Institute, X-ray exposure, endocryology. He founded an independent school of pathophysiologists at the Kharkiv Medical Institute, where he studied the pathology of gas, heat, metabolism and endocrine systems from his students DEAlperin, SMLeytes and others. Academician ADTimofeevsky worked on tumors and studied whether tumors can be grown under artificial conditions. It is a state award winner for growing large tumors from a single cell in vivo and in vitro (inside and outside the body). Lunin works in the field of vitamins. AP Likhachev works in the field of gas exchange. VVPashutin died of a heart attack in 1901 while working as the rector of the Academy of Medical Surgery.

2- The School of Animal Pathophysiology was founded at the University of Moscow under the direction of Alexander Bogdanovich Foxt (1848-1930), a student of AIPolunin. It studies the pathological processes occurring in organ tissues, including: lungs, heart system. Creates a model of artificial pores of the heart and studies it in detail. He studies the formation of constipation in the lungs and heart in cardiovascular pathology, pulmonary, cardiac dysfunction. Professor Govril Petrovich Sakharov from the ABFoxt laboratory in the field of allergy and endocrinology, AI Talyansev develops methods of peripheral circulatory pathology, VVVoronin inflammation, AFAndreev clinical death and general resuscitation of the organism. VANegovsky studied animal pathophysiology of the cardiovascular system, on this basis he created a complex method of resurrection. GPSakharov and his students SMPavlenko and AAJuravel worked in the field of reactivity, immunology and endocrinology.

3- The School of Animal Pathophysiology in Kiev and Odessa was founded by Vladimir Valeryanovich Podvesotsky (1857-1913), who developed the humoral theory of immunity, a parasitic theory in the field of tumors. He worked on the regeneration process. He has written a textbook on animal pathophysiology and has published it in several languages. He published a journal, The Archive of Pathology and Medicine, to promote the science of animal pathophysiology. His students are LATarasevich and ITSavchenko, academician AABogomolets and others. They studied the problems of immunology, reactivity of the organism, endocrinology, and they always worked under the direction of II Mechnikov. LATarasevich and IT Savchenko worked on agglutinin, precipitate, antibodies in France at the suggestion of IIMechnikov.

Academician AABogomolets works in the field of animal pathophysiology, studying the role of reactivity in pathology, its relationship to endocrine management. He was born in 1881 in Petropavlovsk Prison and died in 1946. His mother was imprisoned for being a member of Russia's "southern liberation group."

Academician AABogomolets is the President of the Ukrainian Academy of Sciences and the First Deputy Chairman of the Presidium of the Supreme Soviet of Ukraine. Pathophysiologist-pathologist since 1924. By developing the pathophysiology of animals, he created the original pathological doctrine in medicine, which is called the physiological system of connective tissue. In addition to the supporting function of the connective tissue, it performs a trophic function and a plastic-building function. As it is composed of RES cells, it enhances phagocytosis and antibody production. Improves connective tissue function using antiretroviral cytotoxic serum. It was actively used in the treatment of many diseases during World War II. Academician AABogomolets Director of the All-Union Blood Transfusion Institute, developed a method of conserving blood (the first among physicians to be awarded the title of sos. labor hero). He identified 4 different types of constitutions depending on the nature of the connective tissue and observed more or less common diseases, depending on these constitutions. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known

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Academician IISirotin worked on the field of acclimatization of the organism and the reactivity of the organism.

Academician REKovetsy studied the origin of tumors and the characteristics of their development in different conditions, the course of metabolism in tumors.

Academic ADAdo has worked on allergic diseases, anaphylaxis, lung disease, and has written a textbook on Animal Pathophysiology.

The new schools of animal pathophysiology were headed by well-known scientists LATaraseevich, AVReprev, ESLondon, AABogomolets SSKholatov, GPSakharov, NNAnichkov, ADSperansky.

Academician NNAnichkov (1885-1965) studied in depth the pathophysiology of the cardiovascular system, the involvement of RES cells in pathological processes and the mechanisms of origin of arteriosclerosis in the Department of Pathophysiology, Pathanatomy of the Military Medical Academy.

Experiments show that indifferent influencers play an important role in the development of diseases. For example, if dogs are injected with apomorphine for 15 days and supported with light, in the following days only the lighting of the lamp causes them to vomit reflexively. He suggested that organs could not be studied in isolation from the body, and that systematic scientific work should be carried out. He founded a large school of pathophysiologists, and even today scientists from the ADSperansky school are actively working in research institutes and universities.

IIRavich, the founder of veterinary pathophysiology, worked in the veterinary department of the Academy of Medical Surgery in St. Petersburg, critically examining Virkhov's cell theory and acknowledging the importance of the nervous system in the origin of the disease. He wrote a textbook on general zoopathology and lectured to students on the subject.

Academician MPTushnov (1879-1935), Head of the Department of Pathophysiology of the Kazan Veterinary Institute, created an original drug in pathophysiology, the lysates of which are the products of the decomposition of various organs. For example, muscle lysates are called myolysates, and when animals are released when they are tired, their ability to work is restored, mammolysates are prepared from the udder and increase the amount of milk, and ovariolysates accelerate the maturation of egg cells. Lysates are now the most common and widespread type - biostimulants. They are used in the growth and development of young animals, increase productivity and treat many diseases. Biostimulants are widely used in fattening. Including, Chlorella, which is found in billions of water, has been used to enhance productivity by enhancing all the processes that take place in animals. Currently, there are more than 45 departments of veterinary pathophysiology in veterinary institutes and faculties of the CIS countries, which are studying the effects of biostimulants on the characteristics of the organism. Most research veterinary institutes are studying the effects of biostimulants on the body's reactivity, metabolism and neuro-endocrine control processes.

The contribution of the French scientist Claude Bernard (1813-1878) in the development of the science of animal pathophysiology was significant. K. Bernard's work is studied in two periods:

The first period involved 20 years of normal physiology, proving the liver’s glycogen production function and determining its reflex mechanism. The origin of diabetes in the body proves that it is associated with dysfunction of the CNS. Demonstrates the importance of pancreatic juice and bile in the breakdown of nutrients, as well as observed an increase in body temperature. Blood and lymph determine the organization of the internal environment of the body and determine vital processes.

The second period. He has been working in experimental physiology for 10 years. It studies the importance and function of various nerve fibers in the body, the electrical properties of nerve and muscle tissue, the properties of blood, and the effects of SO2 on the body. Proves a violation of saliva production from salivary glands. A substance called Curare affects the endocrine glands and observes a decrease in the secretory process. He studied various pathological processes of the respiratory system

and wrote more than 180 scientific sources, which consist of 18 volumes. K. Bernard did a lot of work despite experiencing great difficulties. He teaches that the processes that take place in the body depend on the vital force, and that that force is random.

IPPavlov says of K. Bernard, "K. Bernard is a scientist who thought broadly and deeply in his mind, generalized physiology, experimental physiology, and experimental therapy as a whole, or combined the achievements of physiology with practice."

The famous chemist Dumas says, "K. Bernard is not only a physiologist, but he is a physiologist."

IPPavlov's doctrine is important in the development of animal pathophysiology. Prior to IPPavlov, observations were made in pathophysiology using analytical methods. Diseases of isolated organs, their integral parts have not been studied with attention to the living conditions of the animal, changes in the external environment and other related connections. IPPavlov, on the other hand, pays great attention to experimental scientific work and observes changes in body systems in healthy organisms in chronic experiments. According to IPPavlov's theory of nervousness, it is emphasized that any pathological processes in complex organisms are carried out with the participation of the nervous system, in particular, with the participation of higher nervous activity.

The organization and development of the science of animal pathophysiology in Uzbekistan was associated with the formation of the former Soviet Union, which began with the establishment of universities and research institutes in accordance with the decree of the Soviet government. As a result, the medical faculty of the Central Asian State University was established in Tashkent, which was later transformed into Tashkent State University, and intensive work in this area began. In 1921, the first department of "General Pathology" was established at Tashkent State University, which was later renamed the Department of Animal Pathophysiology. The first departments of pathophysiology were established in Samarkand in 1930, in Andijan in 1957, and in 1972 at the Central Asian Institute of Pediatrics.

At the Uzbek State Agricultural Institute in Samarkand, Farkhodi first studied veterinary pathophysiology, and from 1936, the head of the department, Associate Professor Vladimir Valerianovich Volkov. VVVolkov was an encyclopedic lecturer, a skilled experimenter, an excellent pedagogue-coach. VVVolkov was the initiator and organizer of several original scientific works with the staff of the department:

1. The causes and mechanisms of development of allergies and anaphylaxis in astrakhan sheep and goats in hot conditions;

2.Study the causes and mechanisms of development of pneumonia in sheep and goats during the summer months;

3. He has done a lot of research in the field of pathology of the region, the causes of the disease "Suyluk" in horses, the mechanism of its development and the development of methods for its detection. Today, the disease is found in humans and animals and is called trichodesmatoxicosis. In this field NXShevchenko and FIIbodullaev defended their doctoral dissertations and supervised several candidate dissertations.

4. The study of the enhancing effect of cytotoxins formed in tissues on the immunological properties of the organism of various laboratory animals (accelerated formation of antibodies to paratyphoid and colibotseliosis strains).

5. A detailed study of the effects of the parasympathetic division of the autonomic nervous system on the organism of experimental animals.

6. He made a great contribution to the training of a large number of highly qualified personnel. After the untimely death of VVVolkov in 1953, the department was headed by Associate Professor Anton Ivanovich Yarmashkeevich.

Extensive development of scientific work carried out at the department, mainly since 1961 under the leadership of Associate Professor, now Professor Ruzi Haitovich Haitov. By this time, the staff of the department sent different amounts of extracts from the liver, spleen and other parenchymal organs to healthy and sick animals, depending on the timing of their delivery, studied the mechanism of their action and developed a number of recommendations. In the Department of Animal Physiology

and Pathophysiology, tissue feeding of animal feeds has proven to have a positive effect on the growth and development of the organism and the treatment and prevention of various diseases.

Having studied the effects of many drugs against helminthiasis, a number of recommendations have been developed. The genetic features of natural immunity, especially in karakul sheep and lambs of different colors, have been extensively studied and are still being studied. In this area, Associate Professor ADDushanov developed a synthetic vaccine, which gave good results, and Associate Professor MAAbdullaev in collaboration with the senior lecturer of the department RFRuzikulov conducts important research. Under the leadership of Professor RXKhaitov«Veterinary basics»Volume 1-2, 1972, RHHaitov and A.Dushanov on "Animal Physiology" in 1975, RHHaitov and Associate Professor MA Abdullaev on "Animal Pathophysiology of Agricultural Animals" in 1980 in Uzbek, a number of manuals, He has published more than 400 scientific articles in various collections, scientific collections of universities, research institutes, international and CIS congresses and conferences. Under the direct supervision of scientists of the department 10 doctoral and 342 candidate dissertations were defended in specialized scientific councils. Researchers of the department have been writing reviews and defending PhD and doctoral dissertations in many fields of physiology. And so, In Uzbekistan, pathophysiologists study the theoretical and practical processes of modern veterinary and medical science at the Department of Pathophysiology of the Veterinary Faculty of Samarkand Agricultural Institute, the Uzbek Veterinary Research Institute, pathophysiology laboratories of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad.

In order to strengthen the study of pathophysiology, the "Society of Pathophysiologists of Uzbekistan" was established, which includes more than 100 pathophysiologists. Pathophysiology and research work have been carried out in cooperation with veterinary institutes in Moscow, St. Petersburg, Kiev, Kazan, Almaty, Yerevan, and are still connected. As a confirmation of this strong unity, the fact that the 2nd pathophysiologists' session was held in 1972 in Tashkent is a proof of our opinion.

1.Information about the disease has been of interest to people since ancient times. Because science and enlightenment did not develop in the primitive community system, and people did not know the origin of natural phenomena, they thought only about the visible and the invisible. That is why the organism has been described as composed of mythical things found in nature, such as soil, air, water, wood (metal), and fire. Illness, on the other hand, was interpreted as being caused by an invisible divine (supernatural force) or "SPIRIT" - anima. This current is called the "ANIMISM" current or theory, and it is a picture that all diseases are invoked by this supernatural force, the evil spirit. Talented physicians began to appear in Greece 4-5 thousand years BC, who wrote down what they knew, what they asked someone, their observations on the patient, and bequeathed this knowledge to their descendants. As a result, medical science began to develop slowly. For example, they recorded discharge from the mouth, nose, and ears in various diseases, fever, foul odors, and so on. Later in Greece, doctors explained that a living organism was composed of 4 different fluids in addition to 5 different elements (blood, mucus, black and yellow grass). Thus, the current that explains health and disease with these four different fluid properties is called the Humoral Flow or Theory. So, if the fluids are normally mixed properly, health is a sign of health, and this condition is called krazia or krazis. If, for some reason, the ratio of fluids is disturbed or the juices are contaminated, improper mixing, the

disease can lead to dyscrasia or**«Discrasion»**The founder of this movement is the Greek scientist Hippocrates, who lived in the 4th-5th centuries BC.

Hippocrates was an observer, a disease-seeker, a traveling physician, who always traveled from village to village, making many observations on patients, studying the symptoms, various features, currents, and consequences of many diseases, and writing dozens of works. The role of the external environment in the origin of diseases, with great emphasis on cleanliness, developed methods of diagnosis and treatment of many diseases. He developed the laws of medicine, and in medicine there is the Hippocratic oath in medicine. The teachings of Hippocrates have been proven to be true for centuries and even now, and his works have not lost their value.

In addition to diseases, Hippocrates also tried to create constitutions of human temperaments, which included four different temperaments: choleric (yellow grass), melancholic (black grass), sanguine (blood), and phlegmatic (mucous fluid). 'p or less depending on.

The contemporary philosopher Democritus of Hippocrates also developed a theory of diseases, which he called the solid (atomic, particle) theory, which explains that diseases are caused by changes in the spacing of atomic particles in the body. This theory explains that the disease is caused by the narrowing or widening and thinning of the spacing of the particles. At the same time, idealistic schools of thought have sprung up in Greece, claiming that diseases are called by divine power, explaining that organ function, organ diseases, and their causes depend on a particular pneumonia of life. According to Plato, Aristotle explained that there are three kinds of divine or spiritual power that govern the lives of people and animals:

1. Spiritual power is located in the brain and controls the mental function of people.
2. The spirit of the animal is located in the heart and controls the movement and warmth of the animal.
3. Explains that the spirit of the plant is located in the liver and regulates digestion.

They explain that they believe that the causes of diseases are not in the external environment, but in the mental origin. At the beginning of the twentieth century, knowledge of the disease was developed by Roman physicians Galen and Sels, who, in addition to the three zinc origins, based their humoral flow on explaining that diseases often resulted from the breakdown of juices, distinguishing between hot and cold discrasions. developed treatment options. Based on the symptoms of the disease, they observed four specific symptoms of the disease: redness, edema, edema, pain, and these changes, which lead to dysfunction, called functio laesa. . Galen introduces the vivisection method into science.

After Galen, our compatriot was the famous scientist and philosopher Abu Ali Ibn Sino (Avicenna), who made a great contribution to the development of medicine. He was born in 980 in the village of Afshona, Romitan district of Bukhara region and died in 1037 in Hamadan. In 1980, Avicenna's 1000th anniversary was celebrated and her works were published. He wrote more than 300 works in various fields, especially in the field of medicine, and in 1020 wrote a book on the laws of medicine. It consists of 6 books in 5 volumes:

1. The book is devoted to the anatomy, physiology, causes, appearance, general treatment of diseases. Attention was paid to nutrition, health, deportation, vomiting, and blood transfusions.
2. The book describes more than 800 drugs derived from plants and animals.
3. The book is about diseases from head to toe, this book is dedicated to specific pathology and therapy.
4. The book deals with fever, various tumors, rashes, wounds, burns, bone fractures and dislocations, nerve injuries, injuries to the skull, chest, spine and limbs, poisons and poisonings - toxicology, makeup - is dedicated to keeping people beautiful. Recommended remedies against hair loss, obesity or weight loss. He wrote about rabies, smallpox, measles, leprosy, and plague.
5. The book describes the methods of preparation and use of drugs.

Avicenna's book, The Laws of Medicine, pays great attention to the methods of observation and experimentation in the study of diseases, and widely uses this method on various diseases. developed He identified many diseases, developed treatment methods, studied urinary incontinence, urinary tract infections, worm diseases, pulse heart disease.

In his multifaceted scientific work, Avicenna concluded that diseases must have had invisible causes, not divine powers, and that they were now identified as microorganisms.

Avicenna studied in detail the wounds, lung diseases, diabetes, plague, cholera, smallpox, leprosy, tuberculosis (tuberculosis) and many other diseases, especially in the origin of the disease. , boiled, proved that it is important to follow hygiene. He studied the effects of many drugs and found that mercury is important in diseases such as gonorrhea and syphilis. It has been proven that following a meal plan-diet is important in diseases. Although he did not know the functioning of the nervous system, he thought about the nervous system, that is, tied the sheep to the wolf, and observed that a few weeks later the sheep became frightened.

Avicenna's work on TIB laws has been reprinted 25 to 30 times in Europe and Asia, and is still being published today, and has served as a guide for physicians. By the 14th and 15th centuries, Copernicus, a Polish scientist, described the movements of the planets in the sky, Giordano Bruno's rotation of the earth around the sun, the Spanish Servetus's small circulatory system, and Leonardo-Da Vinci's anatomical tracts. V. Garvey discovers a large circulatory system based on his experiments on rabbits and dogs.

By the fifteenth century, a new direction in medicine, the iatrochemical and iatrophysical currents, began to emerge, meaning Iatros-physician.

The chemist Paracelsus conducted many experiments to prove the structure of the organism, the need for chemical elements to survive, the importance of mercury, matches, steel, iron and other elements in the health or illness of the organism. concludes that it contributes, and explains that when archaea get angry, they cause disease without releasing these elements into the body.

Iatrophysicists connect the organs of the body to the parts of a machine and pump the heart, explaining health and disease according to the laws of physics and mechanics.

In the XV1-XV11 centuries, the pathological-anatomical direction developed, and Morgani, Bish, and others began to study the body structure of animals and humans. In 1543, the Italian scientist A. Vezali began to study the structure of the body by tearing apart the bodies. 1640 Descartes wrote the reflex doctrine, 1660 Malpighi lens using the lens, renal capillaries, liver, spleen, skin structure, erythrocytes, 1674 Levenguk lens sperm movement. Morgan and Bish wrote about the changes that occur in different organs in different diseases, which led to the development of the study of pathological processes.

This means that the external environment has had two different effects on the organism over a long period of evolution, and that the organism has become accustomed to these favorable and unfavorable effects, adapted and balanced. -slowly studied and adapted, these effects are called daily or physiological, adequate effects. The processes that take place under the influence of these influences are called physiological processes and are called the norm, abbreviated for short. The second type of effects are often referred to as sudden, strong, sudden, adverse effects, which are called harmful or disease-causing, inadequate effects, and the processes that take place under the influence of these effects are called pathological processes.

Norm or health is a set of influences, conditions, adapting to their currents in a certain period of time, making them suitable for life, necessary or physiological effects, and the processes that take place and develop under their influence. called normal processes. Norma is a process that takes place in a period of stagnation, when the organism is calm and peaceful.

1.Norma-Sergey Petrovich Botkincha stagnation of life processes
is the sum.

2. Norma-Ivan Mikhailovich Sechenov and Claude Bernard describe the organism
with the balance of the external environment.

3. Norma-Victor Vasilevich Pashutin described the structure of the organism and
is said to harmonize its functions.

4.Norma-Vladimir Valerianovich Podvisosky to the conditions of our body

The structure of normative organ systems, the state in which they function without disruption. In real life, the norm is a relatively stable, changeable situation, because the absolute norm does not exist in real life. For example: consider pulse, temperature, respiration.

When one wants to study a disease, one must study it by comparing it with the norm. Both disease and health are ongoing processes in the body, which differ from each other in quantitative and qualitative changes. At the heart of both processes are two opposing processes of assimilation and dissimilation. It is impossible to know the exact time of onset of the disease, but it can be determined only by the symptoms that appear at a certain stage of development. For example, sleep is caused by fatigue as a result of overwork, which is considered a normal physiological state of the body, but in some severe infectious diseases, drowsiness also occurs, indicating a disease of the body: anthrax, typhoid, diabetes , tuberculosis and others.

1.SPBotkin described the disease as a disorder of the vital processes of the organism.

2.IMSechenov and K.Bernar described the disease as a violation of the balance of the external environment in contact with the organism.

7. VVPashutin explains the disease as a violation of the harmony of the structure and function of the organism. These descriptions of the disease provide insights into unilateral changes in the disease, ignoring various complex quantitative and qualitative changes and active processes during the course of the disease. Therefore, these definitions do not fully describe the diseases.

8. In an attempt to fully express the disease, IPPavlov proposed the following definition: a disease is an encounter of an organism with an awkward, pathogenic, gross cause and condition that affects it suddenly, suddenly, collision, ie mechanical shock, crushing, injury, exposure to chemical, physical influences or attack by microorganisms, this encounter is the beginning of a struggle between the organism and the cause , by activating all defense mechanisms against, removing pathogenic causes, cleared or enzymes, phagocytes, Acute flow diseases - from a few minutes, hours to several weeks: For example: infectious and parasitic diseases.

3. Moderate acute flow illnesses — from a few weeks to several months.

Chronic recurrent diseases are those that last for months or years, most of which are non-communicable and non-infectious.

Diseases occur in several stages as they develop in the body.

a). An incubation or latent or latent period is the time that elapses between the onset of the disease and the onset of the first symptoms of the disease. This period can range from a few minutes to a few hours, weeks, months, and even years. Tuberculosis, brucellosis, non-communicable diseases, leprosy, AIDS and others.

b). The prodromal or disease-reporting period has its own characteristics, during which general symptoms for the disease appear. For example: increase in body temperature, decrease in appetite, heart rate, rapid breathing, etc.

v). Outbreaks appear to be exacerbated during clinical trials.

g). The consequences of diseases are twofold: the animal is either cured of the disease, or the sick animal dies.

1.Diseases spread throughout the body - per kontinuitatem. As the disease progresses, one organ spreads due to adhesions to the other organ. For example, inflammation of the oral cavity continues to spread to the red intestine, then to the stomach, intestines, and so on.

2.The disease is spread by means of friction, adhesions - per kontiguitatem. Pulmonary pneumonia to the pleura and pericarditis - myocarditis, liver - stomach, etc.

The disease is transmitted through the blood and lymph - permestastazine. Many microorganisms are spread through the blood and lymph.

3. Diseases are transmitted through the nervous system - per nervorum, through nerve fibers, stolbnyak - congestion, botulism, polio and other diseases.

4. Diseases are spread by secretions, saliva, sweat, urine and feces.

Intermittent course of illness is a period of illness that is sometimes mild and sometimes severe.

The complete recovery of the body from disease is called sanogenesis. The consequences of the disease are of two types:

a). The body recovers from the disease.

b). The disease ends in death.

3. There are two types of recovery:

a). The body recovers completely from the disease.

b). The body recovers from the disease.

Recovery comes in two different ways: simple and complex. Simple ways of recovery are carried out by revealing various reflexes. For example: reflex agitation, excessive salivation, wiping tears, vomiting, sweating, coughing, diarrhea, excessive urination and excretion, tickling of the nervous system, and others

In complex treatment, the body is decontaminated by complex processes using barrier barriers, RES organs - liver, spleen, lymph nodes, red marrow, leukocytes, especially T and B lymphocytes, antibodies, etc. the cause is removed, then partially or completely repaired as a result of the recovery process. Restitution is called ad integrum if the body is completely cured of the disease. Sometimes the body can recover from the disease and recur, and the body can be severely damaged, and this is called a lytic transition to a critical and mild course.

3. Diseases can lead to dysfunction of the body without complete recovery. When the body's ability to heal is completely reduced, the body dies from the disease if the doctor's treatment does not help.

3.Death - mortis, morbi -characterized by the cessation of the continuous process of assimilation and dissimilation in the body and the cessation of heart function and respiration.

There are two types of death depending on their origin:

1. Natural or physiological death.

2. Death due to disease or pathological condition.

If 100% of all deaths are considered, only 2% of them are natural deaths and the remaining 98% are deaths due to diseases.

The doctrine that explains the formation of death is called tanatogenesis. Death occurs in several stages and is called the terminal state, they are:

1. Agony-pre-death convulsions: (consisting of peripoganal and oganal period).

2. Clinical death.

3. Biological death

As a result of death, the following changes occur in the corpse:

1.The body cools - algar mortis drops from 10 in the first days and cools to 0.20 on the second day. Of course, these changes are due to environmental changes.

2. The appearance of spots on the body - livoris mortis on the side on which the animal is lying, more spots appear and look good in hairless, unpigmented areas.

3. Hardening of the body - rigor mortis solidification of colloidal substances. Hardening begins after 8-10 hours and goes from head to toe.

4.Decomposition of the body - maceration or autolysis is formed under the influence of putrefactive and microorganisms from the external environment in the body, and the carcass begins to smell foul. If these bacteria are not present in the body, the body will become waxy.

Observations show that the animal continues to live in organs and tissues for some time after death. For example: nails, hair, hair, growth, movement of the stomach, intestinal muscles, contractions and other signs are observed. Much work has been done on the possibility of resurrecting the organism at the time of death. This condition is called resuscitation. It has now been discovered and proven that it is possible to resurrect organisms that have died by accident, and that people and animals who have died from various traumas, excessive blood loss, suffocation during anesthesia, electric trauma, various tragic events is being resurrected. Kulyabko, a professor of physiology at Tomsk University, was the first in this field in 1902.

From 1912 to 1919, the American physiologist Karel was able to use a burdock chicken heart under artificial conditions.

In the laboratory, Academician Kravkov observed the growth of nails and fur when rabbits' ears and fingers were removed and placed in special liquids. So it is possible to resurrect individual organs.

Professor FA Andreev conducted many experiments on dogs in 1913 and concluded that by anesthetizing dogs, the dogs were resuscitated by sending blood to the body and the whole organism could be revived.

1928 At a congress of physiologists and biochemists in Tbilisi, Bryukhonenko and Chechulin demonstrate an interesting experience: cutting off a dog's head, injecting blood into its veins through rubber tubes, and observing the dog's condition. saliva begins to separate when you put the sausage in the bur. In 1966 he was posthumously awarded the Lenin Prize for his invention of the AIK instrument. In 1940, Sinitsin was able to transplant and hold the hearts of frogs and fish. Academician VANegovsky created a common method of resuscitation in 1941-1945, which was suitable for the resurrection of many soldiers and officers during the Great Patriotic War. In nature, it is a near-fatal condition and is called anabiosis: and we can find it in the plant and animal worlds. In the process of long evolution, plants, animals, and microorganisms go into a state of anabiosis, adapting, in order to survive various adverse effects. For example, by reducing the osmotic pressure from extreme cold or heat, by reducing the oxygen in the air, by freezing and drying, special chemical conditions can be created, that is, by using protective substances, anabiosis can be formed. During anabiosis, all functions in animals are sharply reduced (body temperature, heart rate, respiration, metabolism are sharply reduced, reflexes are lost). Anabiosis occurs in worms, fish, frogs, hedgehogs, lizards, bears, and frogs.

In humans, a condition close to anabiosis is called secondary sleep. Lattergic sleep is caused by severe effects, severe illness, and nervous mental illness.

Aging is a three-phase process:

1. Aging in infancy.
2. Aging in adulthood.
3. Aging.

The main task of veterinarians is the prevention and treatment of various diseases. General prevention is a measure of disease prevention using various ways, methods and measures, which consists of complex economic, organizational and veterinary-sanitary measures, which are:

1. The work of improving the external environment, for this it is necessary to create cultural meadows, the transition to the zagon system, the exchange of meadows, the removal of poisonous plants found in the meadows, various harmful substances. Grasslands, barns need to be disinfected and mechanically cleaned. Surrounding the farm, arranging insulators, building cemeteries and animal cremation rooms, improving the reclamation condition of meadows, drying or increasing moisture, washing away salts and other activities:

2. Bacteriological, serological, biochemical, radioactive isotopes and other methods are used to determine the latent stages of the disease by various methods, with regular examinations, taking appropriate measures, ie X-ray machines, allergic methods, blood tests. Twice a year in spring and autumn medical examination is obligatory:

1. Etiology - teaches the general laws of origin of diseases in the body, their causes, a set of conditions. Etiology is the Greek word for aitia-cause, logos-doctrine.

According to IP Pavlov, the future should become a hygienic veterinary, hygiene. Therefore, it is necessary to protect the external environment, and a lot of work is being done in this area. IPPavlov said that it is necessary to know all the causes and conditions of the disease.

The doctrine that teaches the causes of disease is the result of a struggle between materialist and idealistic currents. This doctrine has explained the origin of diseases in a simple, mythical, teleological way, i.e. the disease is caused by the influence of zinc, contamination of juices, changes in their composition, decrease or increase, thinning of particles in the body or indicates that the disease is caused by thickening. Later in the Middle Ages the origin of diseases was badjahil zinc«archetypal»explained in connection with the wrath of God. As a result of observations, A. Vezali and Malpighi began to study the structure of the organism in depth. By this time, the development of industry, the production of dyes, the increase in the production of equipment, created favorable conditions for the study of the functions of the organism.

At the end of the 19th century, the production of wine and silk in many countries, including France, fell into disrepair. This poses great challenges for French scientists. As a result, Louis Pasteur, under his leadership, began to search for and find the causes of many diseases. As a result, they discover that microorganisms are the cause of wine fermentation and silkworm disease. Microorganisms can be used to prevent the deterioration of wine quality by washing wine containers with boiling water and disinfecting silkworm rooms. Thus, by identifying the real causes of the disease, now world scientists are doing a lot of research behind microorganisms, and German scientist Robert Cox is discovering the causes of tuberculosis, Louis Pasteur cholera, rabies and other diseases. The discovery of these diseases, on the other hand, follows a certain pattern, and this current is called the monocaual current. Mopo-single, single, couza - means cause. This doctrine is one of the most advanced doctrines of this period and deals a severe blow to religious doctrines. However, this doctrine does not fully explain the causes and conditions that cause disease, because the entry of microorganisms into the body does not always cause disease. As a result, the doctrine arises that diseases are caused by changes in the sum of many conditions, not microorganisms, and this doctrine means the conditionic conditions called the doctrine of conditionalism. This doctrine is contradicted by the inability to explain the disease, claiming that there is no clear cause for the disease, negating the importance of microorganisms in the origin of the disease.

Constitutionalismproponents of the theory explain that the disease arises from the genotypic structure of the organism, as a result of a deficiency in the constitution. The constitution and genotype do not change at all, so the disease is interpreted as a fatal process or a top-down process. With the emergence of the theory of constitutionalism, many erroneous theories have emerged. There is a misconception that people with low genes and low constitutions should be confused with people with high genes and high constitutions. As a result, Nazi Germany wiped out many nations in order to create a new race, and racist theories still prevail in many countries. These teachings exaggerate the causes of disease,

Nervism explains that the organism is closely connected with the external environment, which is due to the nervous system.

In studying the doctrine of etiology, we must take into account the structure of the organism and the principles of their solidarity, that is, we must combine theory and practice closely, which can explain the etiology in detail.

The causes of the disease are studied into 2 major groups: external or exogenous, internal or endogenous causes.

External causative agents include mechanical, physical, chemical, biological, and other causes.

3. External environmental factors that cause disease.

External causes of the disease are those influencers that affect the body from the external environment and create a pathological process. The causes of the disease are studied in close connection with the organism without self-study of the external environmental factors, and the degree of origin of the disease depends on its nature. Environmental factors that cause disease include mechanical, physical, chemical, and biological causes. As a result of absorption (reserves and electricity, light energy) or reflexively (conditionally and unconditionally) into the closed automatic (IPPavlov) MNS through the place where all factors directly affect the organism of highly developed animals by reflector).

Mechanical factors causing the disease.

An influencer that affects the body from the external environment, causing an injury to this or that in the body, is called trauma.

In such cases, the injury can be caused by mechanical (shock, bruising), thermal (hot and cold), electric current, chemical, X-rays, and even heat (fear, strong impact) and other changes. 'ladi.

Usually the term trauma or injury is used in a narrow sense to refer to changes that occur mechanically. All changes to mechanical injuries are made by crushing, wounding, sharp, impenetrable, shot bullets, pressure objects.

Stretching, crushing, beating, injuring blood vessels and nerve fibers at the site of mechanical impact. The pathological changes that occur as a result of stretching or traction depend on the strength

of the causative agent, the duration of exposure, and the physiological properties and condition of the organ or tissue that is stretched or stretched.

The bones and tendons are also stretched and stretched, and when the muscles contract, they are pulled less than when they are still.

If an organ is strongly pulled and stretched (skin, muscle ligaments, bones, etc.), it is torn and torn. Slow but long and repetitive pulling stretches (e.g. in joints) causes the connecting parts to loosen, causing the joints to play, come out, and so on.

Strong and long-term filling of internal organs (stomach, intestines, bladder). This causes dystrophy of the organ wall and glandular cells.

While changes in organ and tissue compression cause disruption of blood supply, long-term compression of organ or tissue causes tissue nutrition to deteriorate, leading to atrophy and even necrosis.

Strong organ dysfunction occurs when animals are rescued from being trapped underground, resulting in frequent traumatic shock-like disturbances in renal function.

Injuries occur in animals as a result of exposure to cold or firearms, thunder, and air waves. Falling from height or rupture of spleen and blood vessels of deep tissues and organs under the influence of thunder waves is observed fracture of bones without changing the skin lining system.

Traumatic injuries in farm animals (from the coldness of animal caregivers) are caused by the impact of equipment and tools used in various industries (machine mechanisms, washers, dots, etc.).

The following types of traumatic injuries are distinguished:

5. Closed injuries in which the integrity of the skin covering system is not compromised include: compression of the tissue (with tumor, wash, and puncture). Stretching, pulling, breaking, breaking bones, breaking, cracking under the influence of impenetrable weapons.
6. Injuries to the skin lining system, open changes include injury, destruction of the skin lining of the bone, tearing. Depending on the strength of the impact, torn, incised wounds are formed.

One of the characteristic or characteristic changes when an injury occurs is the sensation of pain. The formation of pain is associated with exposure of the organ to extra and introceptors, the breakdown of toxins, tissue breakdown, and the accumulation of toxins of microorganisms in the injured area.

In addition to local changes during injuries, general changes in some organs (heart, respiratory organs, endocrine and external organs) are observed with reflex dysfunction, accompanied by tachycardia, shortness of breath, hyperglycemia, increased blood pressure and other changes. characterized.

Injury to tissues on the surface of the body causes microorganisms to enter the internal parts of the body and cause them to become inflamed. Normally, pathogenic changes are limited due to the activity of protective flexibility mechanisms that protect our body when tissue injury occurs, only in some cases the process is exacerbated by insufficient resistance of the body's protective flexibility mechanisms, leading to the development of pneumonia and then sepsis.

The dead-necrotic tissue in the injured parts forms a large part, and the direct effect of the cause of the injury is due to the wash. The occurrence of such changes is associated with the restoration of tissue nutrition and metabolism by narrowing and rupture of blood vessels, disruption of the integrity of the innervation, and finally compression of the injured tissue and adjacent healthy tissue with exudate.

Long-term purulent wounds are a debilitating weight loss due to the body not healing. Injury weight loss leads to severe damage to internal organs (pleura, lungs, ribs, pelvis and stones). In such cases, the process of tissue regeneration is weakened, atrophy develops in the skin, subcutaneous tissue, transverse skeletal muscles, some internal organs: the animal's appetite is suffocated, sleep is disturbed, liver and intestinal function is impaired, some parts of the bed lie together. becomes lifeless.

Toxins produced by microbes during chronic injuries, the products of tissue breakdown, poison the body and cause it to lose weight. At the same time, many proteins in the pus are released from the body, which weakens the body's resistance to pathogens.

Traumatic shock is one of the most severe pathological conditions of the body.

During a period of traumatic shock, after a short period of agitation, a strong inhibition of the basic physiological functions of the body occurs. Characteristic changes during traumatic shock include acceleration of breathing and pulse, increase in blood pressure, increase in blood glucose and adrenaline. Subsequently, blood pressure decreases, the amount of blood circulating in the blood vessels decreases, body temperature decreases, reflex activity weakens, the animal becomes insensitive to environmental changes, pain sensitivity decreases, alkaline blood reserve and tissue oxygen consumption decrease. The excitability of the cerebral hemisphere cortex and vegetative centers, the formation of biopathy is weakened. A traumatic shock condition occurs after trauma or exposure to a traumatic agent (primary shock). Primary shock is caused by the reflex excitation of sensory nerve endings under the influence of traumatic factors. The peripheral nerves are irradiated to the subcortical parts, first causing excitation and then braking in the cortex. It weakens all the physiological functions of the body, in particular by lowering vascular tone, leading to a decrease in blood pressure. Many scientists explain the secondary development of shock as poisoning caused by the absorption of histamine-like substances into the body through the blood vessels in the crushed part of the tissues. This is supported by the following supporting information. When histamine and other biologically active substances are released into an animal's bloodstream, a secondary shock-like condition occurs, but histamine and peptone shock, although similar to this shock, do not resemble the shock that results from the injury itself. The formation of traumatic shock is accompanied by additional changes in the body, adverse factors (blood loss, fever or heat, hunger, fatigue), the width of the injured area (nerve columns), due to the abundance of receptors and many other factors. In the development of traumatic shock and subsequent restoration of impaired function occurs the influence of pituitary, adrenal hormones, nervous system and other organs.

The outcome of trauma depends on the type of organ, its vital importance. Death can occur if the heart, large diameter blood vessels, nerve centers, etc. are injured. The changes resulting from the effects of mechanical influences on the nervous system are severe and complex. When peripheral nerves are injured, the motor and sensory properties of organ systems change. Mechanical injury of the central nervous system causes severe functional changes in the body (the affected area depends on the degree of injury). Severe bruising, bullet and skull injuries, causing general bruising, can sometimes injure the brain, blocking blood vessels and the respiratory center. This results in cessation of breathing or paralysis of the heart.

Spinal cord injury paralyzes the leg and impairs the function of pelvic organs (urine, fecal excretion, etc.). Sometimes when a strong blow to the podcherevnoy (abdominal) part, the heartbeat weakens and even stops. Injuries to the heart and large blood vessels are dangerous for the body. When a heart is injured, death usually occurs from exposure to its neuromuscular apparatus, thrombus and blood flow to the heart cavities.

Rupture or injury to the artery of the hip, pelvis, and mesentery results in external and internal rupture, resulting in death. Rupture of the tissues in the chest causes air to enter the interstitial spaces and compress the lungs, leading to disruption of the reflex.

Disease-causing sound wavesdepending on the strength, frequency and duration of exposure to sound waves can have a detrimental effect on the body. Noisy mixtures of different strengths and heights have a detrimental effect on the body. Under the influence of these noises, strong agitation, fatigue, changes in the respiratory process, worsening of hearing, increased intracranial pressure and other pathological changes occur.

Accidental, sudden loud noise can damage the hearing aid: a long and strong generated sound wave can affect the activity of the central nervous system. Pathological changes in the body (metabolic disorders, changes in cell structure, accumulation of heat in the body, when the ultrasound is exposed to a sound that is too long and strong) an increase in glucose and cholesterol in the blood, a change in the shape and structure of the shaped elements of the blood i.e. deformation can cause protein coagulation and other changes).

The causes of internal disease often include the factors that contribute to the onset of the disease in the body. For example, as a result of working in mines, factories, and mines, toxins that enter the body in different ways are absorbed into the tissues, and the dust settles in the lung tissue,

causing various deficiencies in these tissues and causing disease. causes. Circulatory disorders are also among the internal causes of the disease. Changes in hereditary traits also cause disease under the influence of mutagenic causes.

Pavlov recommends studying the causes of IP disease in three groups:

1. All exogenous and endogenous causes are the first group of causes to which the body responds with an unconditional reflex.

2. The indifferent effects created by IPPavlov's work, that is, the influence of the causative agent, if supported by normal conditions, then the natural effect of this supporter is called by the disease itself. For example, if you take an apomorphine in a syringe, tie the dog to a machine, and then send the apomorphine to the dog every time it is supported by a light or a bell, then turning on the light bulb will cause illness and the dog will vomit. called syrotchis. The body of animals responds to the causes of this disease by producing conditioned reflexes.

3. Psychogenic causes have also been proven in experiments and are of great importance for human beings, that is, affecting the body by speaking, drawing, grieving, and writing harsh insults can also lead to diseases.

1.Pathogenesis is the study of the origin, mechanism of development, pathogenesis, course, and consequences of diseases.

Greek pathos-victim, genesis-formation. Diseases develop by different mechanisms when different pathological causes affect the body. To make the doctrine of pathogenesis easier to understand, it is distinguished that etiological causes affect 3 different types.

Type 1 causes diseases that affect all stages of development. For example, in acute poisonings, until the toxin is released from the body, it affects the development of the disease in the body, or a similar change occurs when an electric shock.

Type 2 causes serve as a driving force, developing the mechanism of the disease. For example, as a result of a single exposure to hot water, it acts as a starting force. The following substances are formed and poison the body, disrupt the permeability of blood vessels, create an acidic environment and create oxygen deficiency.

Type 3 etiological causes continue to affect themselves depending on the duration of disease development.

The basic structure of the mechanisms of disease development is that when various causes affect the body, there is a lack of oxygen in the body, that is, the metabolism changes, which disrupts the function of various organs and the mechanisms of disease begin to develop.

1. Corticoviceral doctrine is a two-way connection, ie a doctrine that explains that the nervous system is connected to all internal organs. The effect on the body is affected either by a conditioned or unconditioned reflex pathway and responds using unconditioned reflexes. The mechanism of disease development also depends on the reactivity properties of the organism. If reactivity is strongly developed, the disease may not develop. If the body is deficient in various micro and macronutrients, the nutrient content is incomplete, or the body is tired, the development of the disease can occur slowly.

2. Depending on the types of nervous system. If the animals fall into the fragile type, the disease develops more strongly.

3. Explains the development of the disease under the influence of stressors. When inadequate effects on the body are given to the pituitary and adrenal glands over a long period of time, they produce 3 different changes to the effects as they control the body’s reactivity.

1.The properties of tension The pituitary and adrenal glands produce a lot of hormones, adapt to stress by inadequate action, strong excitation, and produce a variety of hormones. If the hormone-producing function either increases or decreases, the body's function is impaired.

2. In the stage of resistance, the body is resistant to any pathogenic influences, because the hormones of the pituitary and adrenal glands increase the energy and plastic mobilization of the body. In the stage of resistance, when the body can not cope with the pathogenic force, the stage of general weakness, without exhaustion begins.

3. At the stage of general weakness, the body loses flexibility, immunological reactions, regeneration state decreases.

3. Examination of cell composition in animals and humans revealed that the development of pathological processes depends on chromosomes: for example, defects in the development of sex, ie secondary sexual characteristics, infertility and other changes. Males have one more sex chromosome and females have one less sex chromosome.

The role of constitution in pathogenesis. The disease arises from the encounter of disease-causing causes with the organism. Therefore, in addition to qualitative and quantitative changes in the pathogen, the characteristics of the animal organism are important in the origin of the disease. The individual reactivity of the organism takes the first place in the origin of diseases in the organism, because the effect of a certain pathogen on the organism of animals does not lead to the disease of all animals, but to some of them.

What is the constitution? Although there is still no complete answer to the question, constitution refers to the general morphological and physiological features of an organism, which are the product of long-term evolution from the interaction of the organism with the external environment, and these properties are stable. Due to these features, the reaction of the organism to the external environment is determined, comparing close species.

The constitution of agricultural animals means that it increases the resilience, resilience, disease resistance, flexibility and productivity of the farm and the environment. Thus, the constitution of farm animals means not only the morphological and physiological characteristics of the organism, but also the reactivity of the organism to the external environment, including the development of a response to the causes of the disease.

The whole organism can be afflicted with various diseases, and it is impossible to know in advance for what reasons they occur. It depends on external influences, hunger, poisoning, fatigue, exposure to cold and other causes that change resistance and their effects. Due to congenital malformations of the organs in some organisms, the influence of the above external causes causes the disease. In recent times, it has become common to study the constitution in two parts:

1. The constitution of the breath.

2. The constitution of digestion.

Importance of breed, sex and age in pathogenesis. Animal breeds play an important role in the origin of the disease, and Algerian sheep do not suffer from anthrax. Horses of the Budyonny breed are not susceptible to lung diseases. Caucasian mountain merinos do not suffer from pyrapylazmosis, but other breeds are highly susceptible to the cause of this disease. Depending on age, young animals suffer from diseases of the digestive organs, pneumonia, some infectious diseases. As the animals mature, many diseases become more resistant.

4. Restoration of body activity. Protective resilience mechanisms in the body that have the ability to restore impaired function under the influence of pathogenic influences, including excess energy generated in the body, surfaces, stored blood, chemicals and biochemicals. For example: under normal physiological conditions, 17-20% of the heart muscle, the respiratory surface of the lungs, the absorption surface of the intestine, 20-25% of the glomeruli of the kidneys, 12-15% of the liver, 10-15% of the blood vessels, 50 of hemoglobin -60% and nervous, endocrine systems are rarely used. Therefore, the organism adapts to any difficult conditions. For example: in bilateral pneumonia, dystrophy and fatty heart muscle, severe liver injury, removal of a single kidney, functions are also compensated when a large part of the stomach and intestines are cut, when a lot of blood is lost, when many capillaries become loose and clogged, and when nerves and endocrine glands are injured. The patient's kidney function is performed by a healthy kidney, and lymph nodes perform blood formation when the spleen is removed or diseased.

environment at different times. First of all, the general reactivity in the body, that is, the resistance to various toxins, and then the types of immunological reactivity developed. As organisms now

develop, the reactive function is performed by cells, which later develop a response using the humoral system and eventually the nervous system.

The properties of reactivity depend on the age of the animal, the nervous and humoral systems, the external environment and the general condition of the organism. For example, when the embryo develops in the mother's womb, it responds to the stimuli through the mother's body, ie through the placenta. When a baby is born, its reactivity is weak and responds only by a phagocytic reaction or by immune cells that pass through the mother’s blood. That is why young animals often get sick and die. Young animals are weakly adaptable to changes in ambient temperature, and their dyspepsia, salmonellosis, colibacillosis, rickets and other diseases are common. Reactivity in adult animals is manifested in the fight against microorganisms by antibodies, phagocytes and macrocytes that have accumulated in their bodies. As the body ages, its reactivity decreases. phagocytes, immune cells are reduced, and the incidence of disease increases with susceptibility to disease. As a result, tumors, hypertension increase, regeneration is weakened, and the body's reactivity is low, so they have severe infectious diseases.

Sirotin NN and other scientists note that the cerebral cortex of cold-blooded and young animals is poorly developed and is less sensitive to strong toxins (histamine, diphtheria, stolbyank toxin). During anabiosis, animals do not develop sensitivity to very strong toxins and infectious agents (plague, tuleremia, anthrax, tuberculosis).

Due to reactivity, the body responds to disease-causing causes, and the sensitivity of different individuals to infectious agents varies. Such cases can be observed in various pathological processes. For example, when an animal with a high reactivity burns, it recovers quickly and an animal with a low reactivity recovers later. The reactivity of the animal organism depends on the metabolism, the immunological properties of the organism, the functional state of the animal organism, the vascular reaction and chronaxy to the excitability of the nervous system.

Concepts of reactivity R Virkhov's cellular theory developed at a time when the theory of cells gave a misunderstanding of the general reactivity properties of individual cells, tissues and organs, ie the fact that pathological processes take place only in cells. 'did not notice. In contrast, IIMechinkov in his many years of observations shows that the reactivity of organisms at different stages of evolutionary development is also formed under the influence of disease-causing factors of the external environment. As organisms become more complex and the nervous system develops, the body's reactivity to inflammatory agents becomes more complex. For example: cold-blooded frogs, inflammation in fish, develops very poorly in warm-blooded animals. Even when these properties were observed by NNSirotinin sending proteins to the body, it was observed that the body of cold-blooded animals produced very weak responses. Gradually, as a result of the development of the nervous system of the organism, the reactivity or sensitivity of the organism to many toxins, formed a changing response.

Reactivity is a characteristic feature of all animals, and in the field of reactivity IIMechnikov, VVPashutin, AABogomolets, NNSirotinins have done a lot of research. In their laboratories, these scientists studied reactivity by linking it to metabolism and other areas. IPPavlov and IMSechenov confirmed that the nervous system plays a leading role in the development of reactivity. In the IPPavlov laboratory, MKPetrova et al observed that the reactivity of animals was impaired by inhibiting the cerebral cortex by giving bromine preparations.

The importance of the types of nervous system in reactivity is also great. To study the importance of types of nervous system in reactivity, they took two groups of dogs:

1. The group includes dogs with a weak nervous system.
2. Dogs with a strong type nervous system in the group.

In animals of both groups, when exposed to strong toxins, cyanic acid, bacterial toxins, dogs with a weak nervous system became ill due to weak barrier properties of the organism, in animals with a strong nervous system AMMonaenkov and others explain that the diseases have not developed because their barriers are strong, their neutralizing properties are high.

In the IPPavlov laboratory, pigeons became infected with anthrax when a certain part of their brain was removed.

Academician ADSperansky observed that when dogs opened their brains and placed a ball in the midbrain, mechanical effects resulted in ulcers in the lungs and digestive systems, weakening their resistance to infection. He drew attention to the fact that the traces of the nervous system in the origin and development of pathological processes, that is, pathological processes in the nervous system, even after their recovery, retain their complications for a long time. In many experiments, that is, when animals are exposed to different stimuli after treatment of the disease, the effect of these stimuli spreads to the entire nervous system, leaving traces of old disease in the affected area. observed that it had survived and accumulated, leading to the onset of the disease. This feature of the nervous system is called AA

Reactivity is also affected by the autonomic nervous system. Reactivity changes when the function of the autonomic nervous system increases or slows down. Excitation of the sympathetic nervous system enhances phagocytosis, enhances metabolism, and increases reactivity. Excitation of the parasympathetic nervous system increases the production of antibodies, produces short-term leukocytosis, followed by leukopenia, exposure to certain toxins (phenol, aniline, etc.), lymph nodes, liver barrier - barrier properties increases.

Reflexivity changes reflexively from the pathological effects of heat and cold. For example, as a result of colds, people get the flu, pneumonia, that is, the body's reactivity decreases. In experiments, it is possible to cool the body of chickens, reduce their reactivity and lead to anthrax, or to heat the body of guinea pigs and reduce their sensitivity to proteins.

Toxic substances, alcohol, carbon monoxide, lead, mercury, cyanic acid weaken the internal braking. Pigeons were poisoned with alcohol, which reduced their reactivity to anthrax, or when people consumed alcohol for a long time, they observed a decrease in the general reactivity of the organism, and xko.

While ultraviolet light from light energy increases the stability of an organism to a certain extent, it weakens the stability of an organism to a certain extent. X-rays and gamma rays have a detrimental effect on the body's reactivity. The reactivity of the organism also decreases under the influence of mechanical influences. Thus, the role of nervous endocrine systems in the formation of reactivity of the organism is important, but different effects of the external environment affect the activity of various organ systems of the organism, affecting their metabolism, neurohumoral control mechanisms.

There are several classifications of reactivity, and most scientists classify the organism according to its state of health or disease:

1. Physiological reactivity.
2. Pathological reactivity.

Physiological and pathological reactivity can be individual or individual, as well as group. Individual or specific reactivity depends on hereditary traits and can be passed down from generation to generation. Physiological reactivity develops the body's response to natural (adequate) influences, while pathological reactivity develops the body's response to the causes of the disease. Allergic and immunological types of pathological reactivity are distinguished, and the manifestation of these types of reactivity is formed in relation to foreign proteins, microbes and their toxins. (Allergy, Anaphylaxis, Immunity). Typically, biological or species reactivity is differentiated and is specific to animals belonging to a particular species, ranging from seasonal changes in animals to: seasonal sleep, migration of animals from one place to another, animals are not exposed to microorganisms, ie chickens are not infected with anthrax, specific reactivity is a characteristic feature of a particular individual, it depends on the constitution, sex, age, nutrition and storage characteristics, newborn reactivity in animals is low, reactivity is well developed during sexual maturation, phagocytosis and the formation of immunoassays are well demonstrated, in older animals the reactivity of the organism is low due to the weakening of their barrier properties. Hence, the specific reactivity is that during the period of complete vaccination of animals, their reactivity is formed differently, with strong antibodies in some and weak antibodies in others.

The resistance of an organism, as the Latin resisteo (resist, resist), is the resistance of an organism to physical, chemical, and biological causes of disease. This means that the body's resistance is understood to be resistance to many different causes.

During phylogenetic development, when the resistance of the organism changes and invertebrates are resistant to bacterial toxins, the susceptibility of warm-blooded animals is high. Resistance is associated with the functioning of organ systems, depending on the type, sex, age, constitution, anatomical and physiological characteristics of the animal, the level of development of the organism, the development of the RES and lymphoid system. In the early stages of ontogenetic development of animals, resistance to various harmful agents is high (partial pressure reduction, some bacterial toxins), resistance to sexual development is well developed, and resistance decreases with age.

Resistance:

1. Natural-born,

2. Acquired-generated species are different.

Congenital resistance is passed down from generation to generation. For example, Algerian sheep are more resistant to anthrax than European sheep.

Acquired generated resistance depends on the individual characteristics of the organism and is formed when immunized against infectious diseases. Resistance is formed depending on the activity of the pituitary, adrenal glands, colon, gonads. Barrier properties of the organism, biologically active substances in the blood and phagocytosis play a key role in resistance. When the body is tired, very productive, living conditions are poor, resistance is weakened, and conditions are created for the development of diseases.

2. Animals and humans live in a world of microorganisms. Immunity, on the other hand, as a controller, rigorously tests agents for various causes that have entered the body.

Immunity - Latin Immunitas - means purification, deliverance. Immunity is the ability of an organism to be exposed to antigenic pathogens, their products and hereditary foreign substances, or to be resistant to various disease-causing microorganisms, viruses and their products, as well as to non-infectious modes. , forms a special view of the overall resistance.

Immunity is divided into two depending on the nature of the mechanism and causes that cause it:

1. Congenital immunity or hereditary immunity from generation to generation.

4. Acquired immunity

Congenital or natural species-specific immunity is a specific resistance of an organism that is passed from generation to generation and is specific to a species, breed, and population. For example, in cattle, horses are resistant to microorganisms that cause croupous inflammation of the lungs, and animals are highly resistant to human diarrhea. Dogs are not infected with pleural pneumonia in cattle. Cattle do not suffer from horse manure, infectious (infectious) anemia.

Inter-species immunity is also different, Algerian sheep are resistant to anthrax, Breton sheep are resistant to smallpox, light-bodied pigs are resistant to yellow fever, Mongolian cattle are resistant to plague, and other animals of this type are infected with the above diseases. Congenital immunity is formed not only against an infectious agent, but also against their toxins. The barrier properties of animals with innate immunity are strong and do not transmit microorganisms into the body or prevent the growth of microorganisms by altering the environment.

These organisms have high phagocytic activity and bactericidal properties in fluids, which prevents the development of microorganisms and forms specific immune cells against these microorganisms.

Acquired immunity is formed during the ontogenetic development of certain microorganisms in the body of animals. Acquired immunity is created by natural and artificial means. For example, naturally acquired immunity is formed after recovery from mango, smallpox, proteinuria and other diseases. Artificial active immunity is created by vaccinating animals against various infectious diseases. Hence, acquired immunity is generated by natural and artificial means.

Artificial immunity is studied as active and passive immunity. Passive immunity is formed when hyperimmune serums are sent, through the passage of immunoassays through milk, through the placenta. Due to passive immunity, the body's resistance is maintained for some time. RES plays a leading role in the formation of immunity, and the formation and formation of immunity is controlled by the nervous system.

During the period of immunity against infectious diseases, if the organism is completely cleansed of infectious agents, sterile immunity is formed and the organism is provided with sterility to this antigen.

If the immunity formed in the body does not maintain complete sterility, and the antigen is retained in the body, it is called nosteril immunity, which is characteristic of tuberculosis and brucellosis.

Immunity can be formed not only against microorganisms themselves, but also against their toxins, which is called antitoxic immunity and is observed during exotoxin-producing microorganisms: tetanus, botulism, gas gangrene and other infections. Hence, toxins act as antigens in this process.

In addition, the body has special organs and factors that fight microbes and foreign substances, which are called barrier properties of the organism. The barrier-barrier properties of the organism are studied as external and internal barriers.

External barriers of the body include the skin and its products (accumulations), mucous membranes in various parts, the oscillating epithelium of the respiratory tract, microorganisms of the digestive system and hydrochloric acid.

The body's internal barriers include a number of cellular and humoral factors, various histiocytes, reticular cells, plasma cells, epithelial cells of the inner wall of blood vessels, and leukocytes. RES cells, which are involved in protecting the body, are active, they absorb microbes and other particles that enter the body, they are very rich in RES in the lymph nodes, spleen, liver, lungs, kidneys, meninges, blood-forming organs, skin . This means that RES is present to one degree or another in various organs of the body, and phagocytic activity is much higher in leukocytes, including neutrophils. In his long-term observations, IIMechnikov argued that the process of phagocytosis plays an important role in the formation of immunological features. microbes and their toxins, cellular elements, tissue breakdown products, other particles are digested in cells. Phagocytosis is the process by which particles are trapped in a cell and then digested. Phagocytosis is common in nature, with feeding and protection of single and multicellular simple animals occurring in a single cell, while in highly developed animals these systems are isolated and protected by specific mesenchymal cells (blood leukocytes, lymph nodes, red blood cells). bone marrow, spleen, liver, connective tissue histiocytes) - by phagocytes. Studies have shown that there is a direct link between the process of phagocytosis and the resistance of the organism. increased phagocytosis indicates a weakened immunity in the body. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or

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1. Antitoxins and antifenzymes, immune cells that inactivate by binding toxins and enzymes.

2. Agglutinin and persipitins, antibodies that change the colloidal chemical structure of microorganisms, immobilize them, bind them to the sediment.

3. Cytolysins or cytotoxins - antibodies that break down cells under the influence of enzymatic complement substances.

4.Opsonins and bacteriotropins - change the appearance of microorganisms, facilitating phagocytosis.

If antibodies are formed under the influence of antigens, what are the antigens themselves?

Antigens are substances that enhance the formation of immune bodies and react selectively with them. These include microbes, toxins, erythrocytes and serum of other animals, as well as high-molecular compounds.

There are two types of antigens.

1. Full value antigens.

2. Incomplete antigens - haptens.

Complete antigens include complete proteins, ie serum, various proteins, microorganism toxins and filtrate colonies. Antigens have specific properties that react with the antibodies they produce.

Incomplete antigens, ie haptens, cannot enter the body to form antibodies and only bind to the protein molecule to achieve antigenic properties.

Antigens must be administered parenterally to the body to form immune cells. Antigens are exogenous and endogenous substances that are foreign to the body. The body’s own proteins also sometimes exhibit antigenic properties. To do this, the body's proteins meet with the infectious agent, toxins, and form an autoantigen. In order to form immune cells against antigens, the antigen remains in the body for a certain period of time, is captured in the liver, spleen, lymph nodes and stored in the blood for 2-3 weeks. Immunological reactivity is formed not only from the encounter of macro and micro organisms, but also from other types of individuals and even in the same organism itself when tumors grow, become inflamed and in other cases have antigenic properties against their own organism. In all cases, there are antigen and antibody reactions and phagocytosis between body tissues and other tissues. The tissue formed during embryonic development serves as an antigen for older tissues. Tissue does not fit the transplanted tissue or organ due to the immune barrier property of these organisms when transplanting organs into one species or individual, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by.

Decreased or complete loss of antibody production as a result of exposure of antigens to the body is called immunological tolerance or non-response. This condition is caused by antigen transmission during the embryonic period or after the animal is born. In older animals, immunological tolerance can

be established by transferring large amounts of antigen or exposing them to X-rays. Immunological tolerance is characterized by the loss of these antigens of their antigenic properties, which is observed when transplanted into other animal tissues, and the transplant grows well. It is currently used in blood transplants to remove tissue barriers from immunological

Inflammation is the most common, most complex pathological process known since ancient times, and in ancient times all diseases accompanied by a rise in local temperature were called inflammation. Inflammation is a typical pathological change (disruption of tissue function and changes in structure) that is common in various diseases, as well as the activation of the body's protective resilience properties and the restoration of impaired function. Although inflammation in this area delays the organism as a process with protective properties against the effects, the mechanism of its development, the formation of symptoms depends on the state of the organism, the activity of neuro-humoral systems. For example: Inflammation of the skin can be caused by affecting some endocrine glands of the gillpotalamus or peripheral nerves. Glandular is a local manifestation of the general reactivity of the organism, the degree of reactivity of the organism depends on the course of inflammation and, conversely, on the reactivity of the organism to inflammation, neurohumoral control, thermoregulation and other mechanisms. All substances that cause inflammation are called phylogenetic substances, and we study them in two groups, namely, exogenous and endogenous substances. Inflammation occurs under the influence of phylogenetic substances, and the name of the inflamed organ or tissue is read by adding the suffix "IT", "IYA". For example. Inflammation of the liver is called hepatitis, inflammation of the kidneys is called nephritis, inflammation of the lungs is called pneumonia, and xzo

Inflammation is caused by mechanical, physical, chemical, and biological causes of external disease, and often the contribution of microorganisms and viruses is important in causing inflammation.

Sometimes inflammation can also be generated under the influence of conditioned indeferent stimuli.

Ichki yalligg‘lanish chaqiruvchi sabablarga nekrotik to‘qima, infarkt, gematoma, turli qismlarda to‘plangan tuzlar kiradi. Yallig‘lanish chaqiruvchi sabab, ko‘pincha yallig‘lanish reaksiyalarini hosil bo‘lish intensivligini belgilab beradi: Masalan. Rengen nuri, zaharli modda, mexanik jarohatlar, kuyish,sovuq urish va boshqalar oldin to‘qimalarni parchalab, keyin shu joyda fiziologik aktiv moddalar to‘planib, ular ishtirokida yallig‘lanish jarayonlari ro‘yobga chiqa boshlaydi. Surunkali kechuvchi kasalliklarda , kasallik chaqiruvchi sababni, begona tasirotchini uzoq vaqt tasiridan, yoki ximiyaviy qo‘zg‘atuvchining tasiridan proliferativ jarayonlar kuchayaadi.

Yallig‘lanishni kechishi kasallik chaqiruvchi sabab tushgan joyga bog‘liq bo‘lib, amyoba jigarga tushib absets chaqirsa, ichaklarda yarali yallig‘lanish chaqiradi. Masalan. Stafilokok, streptokoklarni yiringli infeksion jarayon hosil qilish aniq, lekin skipidarlarni teritagiga yoki muskullar orasiga yuborib yiringli yallig‘lanish chaqirish mumkin. Shunday qilib yallig‘lanishni xususiyati, uni hosil bo‘lish tezligini qo‘zg‘atuvchi xususiyatiga hamda yallig‘lanish kechayotgan muhitga bog‘liq ekan. Yallig‘lanishning tashqi mahalliy belgilari Sels va Galenlar tomonidan sharxlangan bo‘lib: qizarish-chivoch, shish tishoch, harorat ko‘tarilishi-saloch og‘riq - doloch, funksiyani buzilishi fipstto laesa deyiladi. Har qanday yallig‘lanish ham bir qancha asosiy bir-biri bilan bog‘liq jarayon bilan kechadi: altteratsiya-to‘qimalardagi distrofik o‘zgarishlar-to‘qimalarning yallig‘lanish chaqiruvchi agent ta’sirida qitiqlanishi va parchalanishi, maxalliy qon aylanishini buzilishi-ekssudatsiya va emigratsiya, fagotsitoz hamda proliferativ o‘zgarishlar. Yallig‘lanish chaqiruvchi agent to‘qimalarni qitiqlashi, parchalashi, ulardagi moddalar almashinuvini, tuzilish va funksiyani buzilishiga sabab bo‘ladi. Distrofik o‘zgarishlar yallig‘lanish chaqiruvchi sabab ta’sir etgan vaqtdan hosil bo‘lib, kam chegaralangan bo‘ladi. Keyinchalik ta’sirotchining ta’siri kuchayishi bilan yallig‘lanish kuchayadi, to‘qimalarda moddalar almashinuvi kuchayadi, qon aylanishi buzilib, distrofik o‘zgarish kuchayadi. Kasallik chaqiruvchi sabab organizmga tushib birinchi navbatda retseptorlarga tasir qiladi. Agar ta’sirotchi kuchi etarli bo‘lsa nerv oxirlarida parabioz xolatini hosil qiladi.

At the onset of inflammation, the tissue bends the cells, fat granules appear, protein and fat dystrophies are observed, then the cell structure is disrupted and even severely damaged and dies. Necrabiotic processes during inflammation are caused by the bending and melting of collagen and

elastic fibers of tissue interstitials. In inflammation, necrobiotic processes are formed when tissue burns, under the influence of strong acids and alkalis, sometimes in relation to weak influences from increased sensitivity of the organism. There is a certain association between them and dystrophic changes in the body, and sometimes due to the injured part there is a compensatory restoration of their functions, despite the presence of destructive changes in the salivary glands, stomach and other organs. ladi. The development of destructive changes during the period of inflammation depends on the organ, and such changes can be observed in injuries of parinchyomous organs. The degree of dystrophic changes depends on the strength and nature of the pathogen, where the pathogen enters, the nature of the injured organ or tissue, and the reactivity of the organism. Physiologically active substances formed as a result of dystrophic changes in the source of inflammation and metabolic disorders are absorbed into the blood, reducing vascular tone, causing emigration, phagocytosis and proliferation of cellular elements. These biologically active substances include histamine and histamine-like substances, acetylcholine, ATF, creatine phosphoric acid and other necrogorms that dilate blood vessels and enhance proliferation, trephon tissue proteases and cathepsins. Thus, the strong passage of alternative, proliferative and exudative processes during the inflammatory period leads to tissue bending and the development of dystrophic changes that complicate blood circulation.

Metabolism at the source of inflammation undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. Metabolism between the inflamed part and healthy tissue is enhanced. The increase in metabolism is due to easily oxidized carbohydrates, which form many weak acids as they take place in an oxygen-free environment. The breakdown of carbohydrates in the anaerobic phase increases due to leukocytes released during emigration, but these changes can be seen in the oxygen consumed and the carbon dioxide excreted before the breakdown is broken down into the final product. During this process, the respiration rate decreases as more carbonic acid is released.

During inflammation, the metabolism undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. The metabolism between the inflamed part and the healthy tissue becomes enhanced. Metabolism will be enhanced. Lactic acids are formed due to the fact that the increase in metabolism is due to easily oxidized carbohydrates, which take place in an oxygen-free environment. Due to the leukocytes released during emigration, the breakdown of carbohydrates in the anaerobic phase increases, but without decomposition to the final product, these changes can be determined by the oxygen consumed and the carbonic acid released. In this process, the respiration rate decreases as more carbonic acid is released.

Fats and proteins also form ketone bodies, albumin-peptones, which are not completely broken down in the center of inflammation. Excessive increase in carbohydrate protein and fat metabolism, complete oxidation of milk at the source of inflammation, pyruvic acid, fatty acids lead to an increase in ketone bodies, amino acids and peptones, and acidosis develops. Acedosis is compensated first at the expense of the body's alkaline reserve, then it is not compensated.

(N hyperonia is formed). Depending on the nature of the process taking place in the tissue, the change in the environment of the tissue becomes 7.1-6.6, ie weakly alkaline, in the acute process 6.5-5.4 in the acute flow process. Increased acidosis increases the dissociation of salts, changes the electrolyte ratio, increases the amount of potassium, increases metabolism, breaks down large molecules into small molecules, increases the amount of ions, increases the osmotic pressure at the source of inflammation. Similarly, oncotic pressure increases. Osmotic and oncotic pressure decrease as you move away from the source of inflammation. Thus, changes in the quality and quantity of tissues during inflammation cause physicochemical changes in tissues, including: hyperionia, hyperosmia and hyperonkia. The causative agent causes a short-term narrowing of the blood vessels by reflex action on the blood vessels and then dilation of the blood vessels.

The slowing of blood flow in the blood vessels is due to the following reasons:

1. Paralysis of the vascular neuromuscular apparatus causes loss of vascular tone.
2. Causes excessive dilation of the vascular surface.
3. It causes the blood to thicken and become sticky.

4. Slows down blood flow as a result of cutting blood vessels with fluids in the surrounding tissues.

5. Due to the adhesion of leukocytes to the inner wall of blood vessels, the unevenness of the inner surface of blood vessels is formed, and sometimes clogging with thrombi leads to a slowing of blood flow.

The vascular response at the source of inflammation varies under the influence of various pathogens. For example: vasoconstrictor (adrenaline caffeine, etc.) and vasoconstrictor sympathetic nerve effect. Slowing of blood circulation changes until complete cessation of blood flow in the arteries, leading to changes similar to thrombosis and hemorrhage. Disruption of blood circulation at the source of inflammation worsens metabolism, disrupts the nutrition of cells in the inflammatory center, and these changes themselves lead to increased inflammation.

Dilation of blood vessels and slowing of blood flow increase the permeability of blood vessels, resulting in leakage of shaped elements with liquid parts of the blood, and this process is called exudation. The fluid released is called exudate. The exudate differs from the transudate in the presence of 2-4 times the protein, shaped elements, local tissue elements, tissue breakdown products, some enzymes and other products. The process of exudation depends on several factors, the main of which are capillary permeability, high blood pressure in the vessels, osmotic and oncotic pressure at the source of inflammation.

Capillary permeability depends on the physiologically active substances histamine, bradykinin, serotonin, as well as potassium and hydrogen ions accumulated at the source of inflammation, which ions swell the blood vessel wall, dilute colloidal substances and disrupt vascular nutrition.

Healthy capillaries pass water and crystolloids, increasing permeability from colloidal substances to proteins primarily albumins (low molecular weight) substances.

In inflammation, more blood flows to the source of inflammation, weakening the bleeding and increasing the pressure in the blood vessels, which allows more fluid to leak out of the blood vessels. Such strong exudation lowers blood pressure in the blood vessels and weakens blood flow. Exudation is also affected by the osmotic and oncotic pressure at the source of inflammation.

During exudation, water, salt, protein, or cell-free products are released from the blood vessels, and then leukocytes are released from the blood vessels into the tissues, called leukocyte emigration. During leukocyte emigration, the localization of leukocytes along the walls of blood vessels occurs, resulting in the redistribution of blood-forming elements, which is associated with slowing of blood flow. In normal life processes, the blood is characterized by the placement of two layers of thin, plasma at the edges of the blood vessels and shaped elements moving in the center, the specific gravity of erythrocytes is heavy between the blood vessels, leukocytes move lightly on the periphery.

As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cytoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Remaining leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that die in large numbers in high osmotic pressure and atsedosis.

Monocytes show their resistance even at pH 5.5. While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose

fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. AS Shklyarevisky, a proponent of the mechanical theory that explains the location of leukocytes along blood vessels, explains that leukocytes are pushed aside by other shaped elements because of their light weight.

Proponents of the second type of this theory explain that leukocyte emigration is a passive process in which leukocytes flow out of the general fluid flow and remain outside the blood vessels. If this is the case, then why do neutrophils come out in one case, lymphocytes and monocytes in the other. Thus, without mechanical factors playing a major role in the location of leukocytes along the vessel wall, this theory cannot explain the formation of these processes. Because the location of leukocytes along the walls of blood vessels is a complex biological process, the active processes in which leukocytes approach the wall of blood vessels, push it out of the blood vessels and participate in phagocytosis.

According to IIMechnekov's biological theory, leukocyte emigration is called a positive hemataxis feature. Positive chemotaxis properties include staphylococcus, streptococcus and other substances that are formed as a result of their activity, as well as products of nucleic metabolism, some globulins, liver and kidney proteins, meat peptone broth, some medicinal substances.

The repulsion of leukocytes from these chemicals is called negative chimataxis, and the negative chymataxis property is characteristic of quinine, chlorochrome, benzene, alcohols.

The development of physkaloid chemistry leads to the emergence of a new direction that explains the emigration of leukocytes, i.e. leukocyte emigration is associated with physicochemical changes in tissues.

Increased metabolism in the inflammatory center results in the formation of completely unoxidized substances, leading to an increase in N ions. Thus, due to different charges, negatively charged leukocytes move towards the center of positively charged inflammation. Leukocyte emigration is also caused by the continuous release of fluid from the blood vessels into the inflamed parts. Energy processes in leukocytes also play an important role in leukocyte emigration. On the side of leukocytes facing the source of inflammation, the protoplasm melts to form pseudopodia and amoeba-like action due to the energy generated during the metabolism of leukocytes. Emigrated leukocytes partially die under the influence of the environment at the source of inflammation, while others are actively involved in the process of phagocytosis. While the process of phagocytosis is influenced by the tissue environment and physiologically active substances, the acidic environment and alkaline environment inhibit the process of phagocytosis, while the normalization of the environment ensures the normal course of phagocytosis. Thus, leukocyte emigration is an active biological process in which mechanical and physicochemical changes play an important role.

Proliferatsiya jarayoni yallig‘lanishning barcha davrlarida hosil bo‘lib, alteratsiya kechayotgan davrda kam miqdorda bo‘lsada to‘qima hujayralari ko‘payib o‘zining eng kuchli ko‘payish davriga yallig‘lanishning oxirgi davrlarida etiladi. To‘qima hujayralarni ko‘payishini kuchayishini parchalangan mahsulotlar va to‘qimalarda moddalar almashinuvini buzilishidan hosil bo‘lgan moddalar hamda patogen agentining o‘zining ta’siridan hosil bo‘ladi. To‘qima va hujayralarni tiklanishida yallig‘lanish markazidagi RES hujayralari ya’ni qon tomirlar endoteliyasi, advintitsiyasi, fibroblastlar, gistiositlar, fibrotsitlar va qon tomirlari orqali emigratsiyalangan monotsitlar ishtirok etadi. Hujayra elementlari harakatchan bo‘lib fagotsitoz jarayonida ishtirok etadi. Bularni makrafaglar deyilib, ularga Ranve plazmatsitlari, poliblastlar, Maksmovning tinchlikdagi adashgan hujayralari, turli gistiotsitlar kiradi. Yallig‘lanish manbaida hosil qiluvchi plazmatik hujayralarni parchalanish mahsulotlarini fermentativ yo‘l bilan emiradi.

After the process of proliferation, the process of regeneration develops, the growth of connective tissue, blood vessels, connective tissue proliferates and glandular cells are regenerated. Young fast-growing connective tissue is rich in blood vessels and is called granulation tissue. The connective tissue grows from the periphery to the center, creating a barrier between healthy tissue and inflamed tissue, preventing microorganisms from spreading from the source of inflammation to the body. Upon completion of the inflammation, interstitial fibrous substances are formed in the granulated tissue, the

blood vessels shrink, the young mesenchymal cells stop growing, and eventually a dense connective tissue chandelier is formed. The resulting scars cause various dysfunctions, including esophagus, stomach, if it is formed in the urinary tract, it causes them to narrow, the mobility of the joints changes, and so on. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little sluggishly. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little sluggishly. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed.

Yallig‘lanish morfologik va etiologik belgilariga qarab bir necha turlarga bo‘linadi. Yallig‘lanishning morfologik belgisiga karab alterativ, ekssudativ va proliferativ xillarga bo‘linadi.

Alterativ yallig‘lanish davrida to‘qimalarda distrofik va nekrobiotik jarayonlar, ekssudatsiya va proliferatsiya jarayonlariga nisbatan kuchli rivojlanib bu turdagi yallig‘lanishlarni turli zaharli moddalardan bakteriya toksinlari, ba’zi bir tuzlar ta’sirida parenximotoz organlardan buyrakda, jigarda, yurak va kam xollarda miyada uchraydi.

Ekssudativ yallig‘lanishda ekssudatsiya va emigratsiya jarayonlari boshqa jarayonlardan ustun turib, ekssudat turiga bog‘liq holda serroz-zardobli, kataral-shilliqli, fibrinli, yiringli, ixoroz yallig‘lanishlar farq qilinadi.

Seroz yallig‘lanishlarda suyuqlik tiniq, sarg‘imtir rangli, solishtirma og‘irligi 1,018-1,-20 tarkibida 5-6% oqsil va kam miqdorda shaklli elementlar saqlaydi. Qon tomirlar reaksiyasi to‘liq rivojlanmay to‘qima kam parchalanib ekssudat tez so‘rilib faqat plevra va qorin bo‘shlig‘ini yallig‘lanishi bir muncha qiyin kechadi.

Catarrhal inflammation is a mixture of serum and mucous substances, which is more pronounced at the level of the mucous membranes, and leukocytes are less in the exudate. In fibrinous inflammation, the exudate is high in fibrin, which indicates an increase in vascular permeability. As a

result, in addition to albumin and globulins, fibrinogen leaks into the interstitial fluid, forming fibrin fibers and membranes, which coagulate. Diphtheria is when the fibrin sits flat between the tissue and on the surface, moves hard on the surface of the organ, and forms a wound.

During inflammation, krupoz inflammation is when fibrin sticks to the surface of the tissue and between them and moves easily without forming a wound.

Purulent inflammation occurs in all parts of the body, with the accumulation of pus in the inflamed parts. This fluid contains a large number of leukocytes, tissue fragments with a high specific gravity. Purulent exudates fill the space in the interstitial space and form an abscess or abscess, inflammation of the sebaceous glands and hair follicles-boils, inflammation of a group of fat and wool bulbs is called carbuncle.

When putrefactive bacteria enter the inflamed parts and dissolve the tissue, the ulcer is called dissolved inflammation and is well manifested in alteration processes.

In hemorrhagic inflammation, the exudate becomes red due to the retention of erythrocytes. Vascular permeability results from acute and severe infectious diseases and poisonings.

In proliferative inflammation, cell proliferation increases oncotic pressures above other processes.

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As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cetoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Intact leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that degrade in large acidic environments and under osmotic pressure

Neutrophils exhibit their resistance at pH 5.5.

While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. According to AS Shklyarevisky, one of the proponents of the mechanical theory explaining the location of leukocytes in the blood vessels, the specific gravity of leukocytes is light, including inflammation of the connective tissue at the site of inflammation, sepsis, actinomycosis, proteinuria and other diseases. 'sib, granuloma is formed, resulting in the passage of toxins and microorganisms from the inflamed area to healthy tissue. Biologically active substances released from leukocytes and other cells, as well as changes in osmotic and oncotic pressure in inflamed parts play an important role in the occurrence of proliferative processes. These modes tickle the receptors in the injured parts by the reflex pathway.

Depending on the immunobiological reactivity of the organism, normergic, hyperergic and hypergic inflammations are distinguished.

Normergic inflammation is caused by the primary exposure of microbes or toxins to organisms that are not sensitized and have normal immune properties. Hyperergic inflammation occurs after repeated exposure of the body to the cause of the disease. This inflammation is accompanied by a strong acute flow, alternating and exudative processes. Changes in this period do not depend on the strength of the antigen, but rather on the increase in the sensitivity of the organism. Alterative changes in hyperergic inflammation begin with fibrin bending and necrosis of halogenated and smooth muscle fibers. The fibrin in the exudate is hemorrhagic because it is a mixed serum. Examples of local allergies to hyperergic inflammation are pulmonary embolism and infectious inflammation in acute rheumatism.

Hypergic inflammation is slow, weak. Hypergic inflammation occurs in organisms that may have immunity to this antigen, or are very weak, emaciated, and less reactive. For example, if a diphtheria toxin is injected into the skin of an animal vaccinated against diphtheria, a very slow local change occurs. Such a sluggish response is observed due to decreased reactivity in animals with strong lean and malignant tumors.

Why does inflammation manifest as a general organism change?

Yallig‘lanish manbai bilan organizm o‘rtasida o‘zaro aloqadorlik va bir-biriga ta’sir etish hosil bo‘lib turadi birinchidan yallig‘lanishning hosil bo‘lishi va rivojoanishi organizm reaktivliligiga, uning boshqaruvchi mexanizmi, moddalar almashinuvi va boshqalarga bog‘liq ikkinchidan yallig‘lanish manbai organizmdagi moddalar almashinuvi, immunologik xususiyatlarga ya’ni barcha organizmga ta’sir qiladi. Sensibilizatsiyalangan hayvon organizmga zaharli bo‘lmagan qo‘zg‘atuvchilar bilan ta’sir etilganda kuchli giperergik yallig‘lanish kelib chiqishini, immunlangan organizmlarda zaharli moddalarga xos yalig‘lanish jarayonlarini chiqaradi. Yallig‘lanishning shakllanishida nerv reflektor jarayonlar muhim ahamiyatga ega. Masalan: retseptorlarni blokada qilib yallig‘lanishni susaytirish yoki umuman hosil qilmaslik mumkin. Nervsizlantirilgan to‘qimada yallig‘lanish juda sust va belgilarsiz kechadi. Simpatik nervning qo‘zg‘alishi yalig‘lanishni susaytirsa, parasimpatik nerv kuchaytiradi. Oraliq miyadagi kulrang do‘mboqchaning uzluksiz qo‘zg‘atilishi organizm turli qismlarida: terida, ichki organlarda keng yallig‘lanish jarayonini chaqiradi. Hayvonlar narkoz xolatda, qishqi uyqu vaqtida va po‘stloq tormozlanganida harqanday kuchli qo‘zg‘atuvchi ham yallig‘lanish chiqarolmaydi. Hayvonlar organizmining murakkablashishi, nerv sistemasining diferensiyalangan bo‘lishi, ularda yallig‘lanishni to‘la belgilari bilan aniq kechishiga, organizmning ximoyaviy xususiyatlarida fagotsitoz, leykotsitlar emigratsiyasi va proliferativ jarayonlar yaqqol kechishini ta’minlaydi.

Inflammation is also affected by the endocrine glands, while thyroxine, aldesterone and somatotron hormones increase inflammation, while AKGT, cortisone and sex hormones histamine, acetylcholine, serotonin and others.

Inflammation depends on the age, type, constitution, sex, and other characteristics of the animal, and hyperergic inflammation cannot occur in young animals. If the signs of inflammation are well manifested with the age of the animal, in old, loose constitution, inert nerve-type animals, inflammation is slowed down and conditions are created for the spread of the pathogenic agent in the body. Inflammation of the abdominal cavity of horses is more acute and severe than in cattle, or if we send tuberculosis rods under the skin to guinea pigs, they form a long-term incurable wound at the injection site. calls. The development of inflammation depends on the anatomophysiological structure of the organism, if the inflamed parts are well supplied with blood vessels, the inflammation will be so strong and, conversely, if the blood vessels are poorly supplied, the inflammation will be asymptomatic. Inflammation is affected by animal nutrition, metabolism, low protein content in the diet, reduces the formation of immune cells in the body of the animal, weakens the resilience of patients, vitamin A deficiency from avitaminosis can lead to easy inflammation of the eyes and respiratory tract. causes. The intensity of inflammation varies in different vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different

vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different vitamin deficiencies.

How does the source of inflammation affect the body?

Yallig‘lanish organizmning mahaliy qon tomirlar reaksiyasi sifatida nomoyon bo‘lishiga qaramasdan, organizmning umumiy xolatiga, moddalar almashinuviga, immunobiologik reaktivliligiga, qon tarkibiga, termoregulyasiya va jarohatlanmagan to‘qimalarga ta’sir qiladi. Yallig‘lanish davrida moddalar almashinuvining buzilishidan, glikoliz jarayoni kuchayib qonda qand miqdorini ko‘payishiga, albumin-globulin indeksini o‘zgarishiga, globulinlarni ko‘payishiga, qonda qoldiq azotni, albumoz-peptonlarni, gistamin, nukleinlar almashinuvining oraliq mahsulotlari va atseton tanachalarini ko‘payishiga olib keladi. Qonda leykotsitlar ko‘payadi, ECHT tezlashadi, tana harorati ko‘tariladi. Immunobiologik reaktivlik yo immunitetni hosil bo‘lishini kuchayishi yo pasayishi bilan harakterlanadi: emlash va kasallikdan tuzalgandan keyin antitela hosil bo‘lishi va fagotsitoz kuchaysa, surunkali kechadigan yallig‘lanish jarayonida immunobiologik reaktivlik va rezistentlik susayishi madorni qurishiga olib keladi. Yallig‘lanish manbai o‘ziga yaqin to‘qima va organlarga ta’sir qilib hayvonlar qorin bo‘shlig‘iga filogen moddalar ta’sirida qorin devoriga yuborilgan mikrobga turg‘unligi kuchayib, bu mahalliy to‘qimalarni immunologik xususiyatlarini kuchayishidan hosil bo‘ladi. Yallig‘lanish manbailarini jarohatlanmagan to‘qimalarga ta’sirini ba’zan organizmdagi qorin sohasining yallig‘lanishi appenditsit yoki aritmiyalarini hosil bo‘lishida ko‘rish mumkin.

The inflammatory center affects the whole organism, affecting its metabolism, reactivity, uninjured organs and systems due to the microorganisms accumulated in these inflamed parts, their breakdown products, toxins, biologically active substances that are absorbed into the blood and tickle the receptors. . The body is also affected by painful stimuli coming from the source of inflammation. The increase in body temperature is caused by the effect of completely undigested substances formed in these parts on the thermoregulatory center in the midbrain. Thus, the source of inflammation affects the body through nerve reflex and neurohumoral pathways.

What do you mean by the mechanism of development of inflammatory processes?

It is a complex reaction of the organism to inflammatory influences that appeared very early, and theories explaining these processes have also been known since very ancient times. The protective properties of inflammation are also stated in the ideas of Hippocrates, who have different views and worldviews on the essence of inflammation.

According to R. Virkhov's 1958 theory of nutrition, inflammation is the transition of cells to a high functional state under the influence of inflammatory factors, a state of intensive consumption of nutrients. However, cells not only undergo a high functional state under the influence of a phlogogenic agent, but also under a high functional state during other effects. R. Virkhov equated inflammation with a simple arousal phenomenon and could not explain that arousal is another qualitatively specific phenomenon. If the proliferative and exudative processes in inflammation are considered a high functional state, the alternative process cannot be considered as such. By binding the inflammation to the cell,

Congeym's theory of vascular changes in 1885. It is said to cause changes in the blood vessels leading to inflammation. Congeym says that the changes that occur in inflammation are due to increased vascular permeability, i.e., exudation and emigration. This theory ignores the fact that other tissues, not blood vessels, play an important role in the development of inflammation. The fact that there is an inflammatory process even in animals with underdeveloped vascular systems did not take into account the fact that vascular permeability is controlled by the nervous and humoral systems.

In Ricker's vasamotor theory, inflammation is explained as a phenomenon associated with changes in the vasomotor nerves under the influence of a phylogenetic agent. Inflammatory nerve exposure causes changes in vascular permeability and tone, leading to the formation of inflammatory-specific metabolic changes in tissues. In this theory, the interaction between the flogen agent and the tissue is ignored and the role of the nervous system is limited. IIMechnikov's phagocytic theory was stated in 1892. Inflammation is a protective reaction formed as a result of evolutionary development, in which

specific cells of inflammation (RES cells) are considered active in response to the action of a phlogogenic agent. This theory suggests that vessels, other than phagocytes, are cells of the nervous system,

In Shaden's physicochemical theory of 1923, he explained that inflammation under the influence of a phylogenetic agent disrupts tissue metabolism and alters the physicochemical properties of colloidal substances as the main pathogenetic chain of inflammation. Inflammation is only a local process, it does not take into account the reactivity of the organism, the state of the regulatory mechanisms that play an important role in the development of inflammation. Thus, inflammation is associated with alteration, necrobiosis, venous hyperemia, stasis, intoxication, dysfunction and other events, on the one hand, arterial hyperemia with protective compensatory properties, accelerated metabolism, leukocytosis, phagocytosis, emigration, multiple antibodies. and the formation of biostimulants, proiferation,

At the end of the twentieth century, the role of the nervous system in the development of inflammation was raised. Samuel recognizes and promotes the importance of the nervous system, saying that neurotrophic processes play an important role in the origin of inflammation, that the influencer affects the cell through the nervous system.

While V.Ya. Danilevsky cut the sympathetic nerve and observed strong inflammation in the tissue controlled by this nerve, Ricker explained that inflammation is caused by dysfunction of vasomotor nerves, and these theories led to the notion that inflammation occurs in the organs. will come.

Only IPPavlov tries to explain that with the development of the theory of nervousness and its role in the nutrition and metabolism of the nervous system is important, that inflammation develops on the basis of important laws. IPPavlov observes that wounds on the skin and mucous membranes of dogs with tubes are formed under the influence of chronic pathogens. These chronic movements are caused by improper placement of the tubes. Inflammation is provided only by the injured nerve and has been observed in other organs or tissues as well, not only in the tissues. For example, inflammation of the cornea of the eye was observed when the sciatic nerve, the cervical sympathetic node and the gray ball and some centers were stimulated. The effect of the cerebral hemisphere on the inflammatory process, when the bark is removed or the animal is anesthetized, the inflammation is sluggish and goes unnoticed. Similar changes are not caused by inflammation during the hibernation of animals, in severe poisoning (mustard, when large amounts of leucites are introduced into the body). Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body.

inability of tissues to have specific biological properties, unlimited growth and control, and changes in the structure and function of tumor cells. These properties in tumor tissue are caused by the influence of external and internal environment on disease-causing causes in healthy cells in the body. Tumor tissue, unlike other pathological changes in the tissue, does not have the properties of regeneration and flexibility (regeneration, hypertrophy, proliferative inflammation) in the body. Not only does the tumor increase in size when the tumor grows, but the tumor can also break down the surrounding tissue.

The branch of pathological physiology that teaches the problems of tumors is called oncology-Greek-oncos-tumor or neorlasma-new abnormal formation, Latin-tumor-tumor. Tumors can form and develop from healthy tissues in the body (epithelial, connective, muscle, nerve). Tumor-forming substances are called carcinogens. The transformation of healthy cells into tumor cells is called malignancy. Tumors are formed by adding a suffix "oma" to the name of the tissue from which they are formed: For example: epithelioma, fibroids, lipoma, osteoma, chondroma, adenoma and others. Some tumors, as they are called by their historical name, are called malignant tumors (sapsech,

sachstpoma) formed from epithelial tissue and malignant tumors formed from connective tissue. Tumors have a parenchyma and a stroma, and the characteristics of the tumor depend on its parenchyma. Blood vessels and nerves pass through the tumor stroma and are composed of connective tissue. Because malignant tumor stroma is so poorly developed, these tumors are called histoid tumors. In benign tumors, the stroma is well developed, surrounded by a thick shell, and is called an orgonoid tumor, reminiscent of a parinchymatous organ. If the tumor parinchyma is composed of multiple tissues. These tumors are called mixed tumors. Hence, we study all tumors into two groups i.e. malignant and benign tumors. Malignant tumors include cancer and sarcoma, all remaining tumors include fibroids, fibroids, ostiomas, chondromas, adenomas, and other benign tumors.

Safe tumors are called tumors that are close to the mother cell and mature due to their morphological structure. As benign tumors grow, they grow from the center to the periphery, enlarging to form a connective tissue shell and compressing the surrounding tissue as they grow. Because benign tumors have a connective tissue shell that is confined to the surrounding tissue, they grow slowly and sometimes temporarily stop growing. In dogs, the size of the tumor increases and the dogs become 1/3 of their body weight. The expansion of a tumor without growing into other tissue is called expansive growth. Safe tumors do not recur and metastasize when surgically removed because they are surrounded by a good connective tissue shell. Of course, a safe tumor is a relative concept. the formation of this benign tumor in the brain leads to disruption of the activity of various nerve centers by squeezing the brain. Safe tumors that form from the endocrine glands cause the production of many hormones and disrupt the functions of the endocrine glands. Safe tumors grow around the red eyelids and other tubular organs, squeezing them, causing dysfunction.

Malignant tumors grow rapidly, irregularly, and are not limited to the surrounding tissues, but grow into them and are called infiltrative growths. Malignant tumors injure the surrounding tissue. The central part of malignant tumors disintegrates without good nutrition and does not become large in size. Tumor growth is variable, sometimes rapid, sometimes slower than in benign tumors. When malignant tumors grow, there is no boundary between the tumor and the healthy tissue, so the malignant tumor cannot be separated from the body. If the sma cell remains, it recurs. Recurrence is a characteristic feature of malignant tumors. A recurrent tumor can form long after it has been removed. Malignant tumor metastasis-Greek metastasis - displacement, interference, which causes tumors to grow into the blood and lymph vessels, staring at the capillaries and forming an embolus. Cancer often metastasizes through lymphatic vessels. Wherever tumors develop when they metastasize, they retain the characteristics of maternal tumors. For example, regardless of which part of the body the hepatoma is formed, it produces urethra, a tumor formed by the thyroid gland is rich in iodine. The formation of metastases depends on which blood vessel the embolus flows through. For example: If the cancer has developed in the stomach, it metastasizes primarily to the liver. In other cases, the formation of metastases depends on the biochemical properties of the tissue in which the metastasis occurs. If you have lung cancer, metastasis will form in the brain and adrenal glands. Thyroid, malignant tumors of the prostate and mammary glands often metastasize to bone tissue. However, the entry of tumor cells into the organs does not always lead to the formation of tumors because they are broken down by macrophages. For example, the flow of cancer cells in the spleen does not cause metastasis. Malignant tumors are so different from benign tumors that in malignant tumors the metabolism changes more deeply than in benign tumors, causing the animals to lose weight.

Tumors are found in all farm and domestic animals, birds, amphibians, and fish. It is even found in various invertebrates as shown in the literature. Sarcoma from malignant tumors in cattle, lipoma, fibroma, ostioma from malignant tumors, melanosarcoma, osteosarcoma and cancer from more dangerous tumors in horses are found in cattle. Tumors in the genitals and other parts of bulls and stallions are more common. Tumors rarely form in the stomach and uterus of animals.

Tumors are more common in older animals. Dogs of purebred and older than 5 years of age have a variety of tumors, most commonly tumors of the genitals and mammary glands. Tumors are rare in rabbits, and tumor damage is very rare in guinea pigs. While laboratory animals are more likely to develop cancer in mice, sarcoma is more common in rats. According to some data, 6-8% of mice die from cancer. Tumors also occur in chickens, where they develop sarcoma. Similarly, geese and ducks

are also affected by tumors. In birds, malignant tumors grow and metastasize. In fish, as in other vertebrates, epithelial and connective tissue tumors are different. Tumors are more common when fish are artificially bred and are less common in free-living fish.

Tumor formation also depends on the age of the animals, with tumors occurring in humans after the age of 40, in dogs after the age of 5, in chickens at the age of one year, and in older animals 10%. The occurrence of tumors in older animals is associated, firstly, with the long-term effects of etiological causes, and secondly, with a decrease in the body's protective functions.

The importance of hereditary traits in the origin of tumors has not yet been definitively studied. However, cancer is caused by viruses, and if an animal is born with cancer after birth, it will develop cancer. This condition is well studied by infecting the animal's udder with the virus.

4. The causes of tumors have not yet been fully studied, and the first information about tumors dates back to 1500-2000 BC in ancient Egypt and Rome, and Hippocrates in those days. tumors can be treated or untreated. In the seventeenth century in England in the cleaners of factory pipes - a disease of pipe workers, in the United States - tumors in the clockmakers of a phosphorus plant. In the first half of the 19th century, œcìàëàð was found to be composed of cells, like other tissues, and the origin of tumors has been explained by various theories. One of these theories is the theory of embryonic buds, in which Congeim argues that during the embryonic development of an organism, some of the cells fail to develop, and that various causes, strikes, due to inflammation and other causes, growth energy is formed in cells that live in secret and begin to grow. Tumor feature is formed. Tumors begin to form. Proponents of this theory explain that tumors and embryonic tissues have morphological similarities, that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor.

R. Virkhov's theory of exposure was developed in 1885 and explains that it is caused by the action of long-term pathogens on tumors, resulting in the formation of lesions in many tissues. This theory explains that tumors are formed in humans and animals in the processes of tissue breakdown, inflammation, and regeneration due to long-term mechanical, thermal, chemical, and other effects. It is said that cancerous tumors are formed as a result of long-term exposure of certain parts in people performing the same functions, from proliferative inflamed parts to the differentiation of cells. But not all formed scars and wounds form tumors. This theory seeks to explain that tumors are formed under the influence of chronic influencers of the external environment. VVPodvesotsky observed that tumors do not form when the body is exposed to mechanical and chemical agents for a long time. However, due to this theory, conditions have been created for many studies and the causes of tumors have not been identified. As a result, in 1916, Japanese scientists K. Ishikova and K. Yamagiwa discovered that tumors are caused by chemicals. They rubbed dyogt charcoal on the inside (skin) of rabbit ears for a long time, causing malignant tumors. Diagnostic cancer was later invoked from experimental animals in mice, rats, and dogs. Two weeks after the coal tar has been applied, the wool from these resinous parts falls off and new wool emerges, and after this change is repeated 6-7 times, the wool does not grow on the skin at all. the skin thickens, roughs, cracks, the outer surface of the skin sheds and alternates. If we observe these parts under a microscope, we will see acute, moderately acute and chronic inflammation of the skin after a month in the place where the coal tar was applied. 3-4 months later, sometimes earlier, sometimes later, one or more questions arise. These tumors then grow, enlarge, infiltrate, and metastasize to a cancerous tumor. Subsequent research has shown that carcinogenic chemical compounds are synthesized from various resins that cause tumors. Carcinogens are polycyclic carbohydrates with their chemical structure. Carcinogens form tumors after several latent periods after they enter our body. If left untreated, a rapid tumor can form. Cancer tumors form

by the 31st to 179th days after the skin is coated with methylcholentren. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat in mice and rats from hungry animals.

Cholesterol, sex hormones, vitamin D, carcinogens in the benzperin group are chemically close and they are phenanthrene products. Some substances change their carcinogenic properties as a result of various effects. For example, cholesterol in grass can be turned into a carcinogen under the influence of radiation. NILazerev's observations show that when hormones are overproduced or a decrease in their antagonists leads to tumor formation. This means that an adequate stimulus forms a tumor when it changes in quantity. The process of cell dedifferentiation and rapid proliferation to form a tumor can lead to malignancy and tumor formation.

Impaired sterein metabolism from fats and lipids is a factor that contributes to the growth of tumors. The formation of malignant tumors under the influence of carcinogens is one of the important achievements of experimental oncology. However, the mechanisms of action of carcinogens have not yet been elucidated. Perhaps the effects of carcinogens acquire biological properties by altering the genetic properties of cells by disrupting the structure and function of nucleic acids. Even chemical theory cannot fully explain the formation of tumors. He explains that chemicals only create the conditions for viruses to affect the body.

From the end of the last century to the present day, tumors have an infectious nature, they explain the parasitic ducts that cause disease in various animals and plants, worms-worm-like parasites, fungi are specific pathogens of tumors. During the study of tumors, many microorganisms were isolated, but all of them were found to be saprophytic microbes and not related to tumors. Malignant or malignant tumors also occur when infected with certain parasites: Cancer can occur in dogs and cats when infected with Oristorshis felineus, which belongs to the class of suckers. Cancer develops when rats are fed cockroaches, or when cattle become infected with fasciola, which causes liver cancer.

The notion that tumors are caused by viruses was first proposed by II Mechnikov in 1910, and in 1911 an English scientist, P. Rose, observed that tumors were formed by sending a filtrate made from sarcoma-infected chicken tissue. P.Rous virus is found not only in tumors but also in the heels, liver,

brain, blood and other fluids of chickens, the size of the virus is 01 m. Low resistance to chemical and physical influences. For example, it decomposes in 2-3 days at a temperature of 00, and in 15 minutes at 550. Antisetics have a strong effect on the virus. Some tumors can grow in an environment made of tissue. Safe tumors formed under the influence of viruses have been observed in various animals to develop into malignant tumors. For example: papilloma of wild rabbits, in dogs and cattle papillomatosis is similar to the warts that occur in humans, and the virus isolated in these animals causes tumors only in this type of animal. Most tumors can only develop in a healthy organism when transplanted. Proponents of viral theory, such as LAZilber et al. The tumor-causing virus may not show its pathogenicity for a long time, even in all vital processes. For example, while some species of mice reach a certain age, most of them become infected with tumors, while others develop one or two tumors. Because tumors can also call a healthy animal child by suckling an infected animal, this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories.

5. Tumor growth begins with the transformation of normal healthy cells into tumor cells, and the metabolism in these cells changes. produces qualitative changes from the biological properties of the cell. Later tumors grow only due to the proliferation of tumor cells. Of course, not all tumor cells turn into tumors, some are absorbed, and some form multiple tumors.

One of the main characteristics of tumors is that they can grow continuously and, if not removed by a doctor, squeeze the animal’s organs, causing death under the influence of toxins. As a result of continuous growth of tumors, the fibroma in cattle reaches 100 cm in diameter and weighs up to 100 kg, about half the weight of the animal. In humans, uterine fibroids weigh 20-25 kg, and ovarian cysts range from 50 kg. By transplanting tumors in the same species, it is possible to ensure their growth for several years. One of the characteristic features of tumors is the transformation of tumor tissue into low-differentiated tissue.

Anaplasia refers to low-level morphological differentiation of mother cells into tumor cells, and Greek means mother-back, down, plasis-formation. In a cell that is becoming a tumor, the rate of growth and proliferation increases. The faster the growth in the tumor cell, the better the anaplasia develops. Usually morphological, biochemical, physicochemical and energy anaplasia are distinguished.

5. In morphological anaplasia, changes occur in the tumor cell and tissue, and according to the morphological features, the tumor tissue is close to the embryonic tissue. The shape and size of the

parenchyma of tumor cells vary. In some cells, the normal ratio of nucleus and protoplasm is different, the number and shape of chromosomes change. The division of tumor cells is atypically malformed, disrupting the mutual arrangement of cells. For example, glandular tumors do not have or have a malformed structure that produces glandular fluid, but retains the functional properties of tumor cells despite having such an atypical structure. That is, tumors formed from melanblasts melanin, tumors formed from liver cells, tumors formed from grass, glandular cells, maintains the function of hormone production. Morphological atypicality is not specific to tumors but can also result in cell growth and proliferation in a variety of pathological conditions. For example: During regeneration and proliferative inflammation.

6. During biochemical anaplasia, the biochemical properties of tumors change, that is, as in embryonic tissues, the amount of water increases to 90%. Potassium salts increase and calcium salts decrease from normal. The faster the tumor grows, the more the ratio of potassium and calcium changes.

Tumors increase cholesterol from lipoids. Tumors accumulate a lot of glycogen, which does not absorb glycogen well. This glycogen accumulates as a result of disruption of carbohydrate metabolism and is associated with an increase in lactic and pyruvic acids in tumors.

DNA and RNA increase in tumor tissue. As a result of the strong breakdown of nucleic acids, pentoses are formed in tumors, the amino acid composition changes, ie cystine, methyanine, tyrosine are reduced in tumors, and histidine, arginine and lysine are increased. Tumors are rich in protolytic enzymes.

5. In physicochemical anaplasia, the surface tension properties of colloidal substances are reduced, many completely unoxidized intermediates are formed, changing the acid-base balance to acidic. Osmotic pressure rises in tumors. Tumor tissue has a higher electrical charge than healthy tissue. Tissue and cell membranes have strong permeability properties. Biochemical and physicochemical anaplasia occurs in the process of regeneration or proliferative inflammation without any specific changes for the tumor. The stronger the growth of a charged tumor, the better the biochemical and physical anaplasia.

Energy anaplasia is caused by changes in metabolism and excessive metabolism in tumors, disruption of carbohydrate and protein metabolism.

6. Metabolism in tumors differs from that in healthy tissues, i.e. we can better observe these changes in carbohydrate metabolism: in healthy tissues, carbohydrate metabolism takes place in 2 periods: anaerobic and aerobic.

As a result of many intermediate changes in the anaerobic period, lactic acid is broken down - called glycolysis.

In the aerobic cycle, 1/5 of lactic acid is oxidized to SO2 and N2O, and the remaining 4/5 is converted to glucose due to energy generated by oxidation.

During glycolysis, 5% of potential energy is wasted on carbohydrates, the remainder being oxidized to form S2O and N2O from lactic acid. When the oxidizing properties decrease, a lot of lactic acid is formed, and acidic substances accumulate in the tissues. Glycolytic processes are dangerous tumors, the breakdown of glucose to lactic acid is 200 times faster than in resting muscles and 8 times faster than in maximally working muscles. Malignant tumors can produce lactic acid equal to their own weight in 10-12 hours. Therefore, the amount of lactic acid in the blood is higher in cancer-prone organisms. Glycolytic changes in malignant tumors are more active than in benign tumors. The formation of large amounts of lactic acid, changes in the surface tension of tumor tissue, etc. are characteristic of tumors. Cancer cells break down glucose 4-5 times more strongly and oxidation is very slow. Glycol = dog processes are not characteristic of tumors, because glycolytic processes occur in the retina, leukocytes in healthy life processes, increased glycolysis, decreased oxygen consumption are also observed in the process of inflammation and regeneration . Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic

processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a decrease in oxygen consumption are also observed in the process of inflammation and regeneration. Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a decrease in oxygen consumption are also observed in the process of inflammation and regeneration. Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body

In tumors, protein metabolism is severely impaired, albumin and nucleoproteins are increased in tumor proteins, and proteins that are not found in healthy tissue are found. The formation of these nucleoproteins has not been studied, but other proteins or viruses of a different nature (LAZilber) or proteins that have been altered by the body in the formation of tumors.

In malignant tumors, full-value and full-value amino acids can also be formed. Proteins in this change can disrupt the activity of enzymes. BIZbarsky determined that specific protein synthesis occurs in tumors and is called tumoproteins.

The disruption of specific nucleic acid metabolism in tumors was discovered in 1934 by Stern and Wilheim, and later in 1941 by Rondoni in tumors where DNA was more than RNA. It has been studied that protein synthesis in tumors is superior to its breakdown by sending various identified atoms into the body. The fact that purine and pyrimidine bases from large amounts of amino acids fall into the tumor tissue and that the amount of residual nitrogen in the tumors is high indicates that the protein metabolism in tumors is faster than in healthy tissue.

The metabolism of fats and lipids is strong in tumors and varies depending on the nature of the tumor. Fats are high in unsaturated fatty acids, cholesterol and acetone cells.

Relationship of tumors with the organism. Based on the data collected in the experiments, MKPetrova explained that the effect of the body on the growth of tumors can affect the nervous system in tumors. The creation of conditions for the origin of tumors in chronic functional disorders of the nervous system (neuroses) in the animal body has been studied experimentally by calling dangerous and benign tumors. During the period of chronic functional disorders of the nervous system, the formation of tumors under the influence of carcinogens is accelerated. The role of the nervous system in the mechanism of tumor development has been observed to slow the growth of tumors under the influence of carcinogens during the hibernation of animals or the inhibition of nervous system activity, and accelerated tumor growth in controlled animals receiving so many carcinogens. If we send sodium bromine to the body, the activity of the nervous system decreases and the formation and development of tumors slows down. It is during this period that the effects of caffeine or nervous system stimulants on rabbits accelerate tumor growth.

Injury to peripheral nerves contributes to the formation of metastases. If the sympathetic nerve of the neck is cut, malignant tumors will form, which will help the transplant to grow. The effect of RES tissue on tumor growth is significant, as macrophages can break down the tumor without developing it, preventing it from growing. Macrophages resist metastasis by trapping malignant tumor fragments that enter the blood and lymph. AABogomolets and MANavinsky in 1877 observed that activation of RES tissue function prevents the transplantation of transplanted tumor tissue, or blockade of RES tissue creates conditions for the growth of transplants.

The body influences the growth of tumors through hormones produced in the endocrine glands. While one of these hormones inhibits the growth of tumors, the other accelerates the growth of tumors. For example, while somatotron hormone in the pituitary gland enhances tumor growth, hormones in the pancreas and adrenal cortex inhibit tumor growth. When we send estrogen hormones to an animal's body, a tumor develops in the animal's udder and genitals. Testosterone and progesterone inhibit tumor formation in the udder and genitals.

As the body reacts to tumors, so do tumors. The effect of tumors on the body depends on the nature of the tumor, its growth and the location chosen. If there are small tumors on the surface of the hand, they fall into the category of benign tumors, which only cause discomfort when doing any work. possible. Safe tumors compress the surrounding tissues, disrupting their nutrition and leading to atrophy. If the sap compresses the separating pathways, the sap becomes difficult to separate, and so on

Although malignant tumors are small, they degrade the body and lead to death due to impaired growth and metabolic disorders. The cause of weight loss in animals is caused by metabolic disorders, poisoning the body with intermediate products of metabolism and due to the breakdown products of tumor tissue. From it, the dysfunction of the organ in which the tumor grows also causes the body to lose weight. Tumors show antigenic properties to the organism as they begin to grow, but the structure of these antigens has not been determined, but antibodies to these antigens are formed. Antigens are sufficiently foreign, due to the lack of foreign antigenic properties, as well as the weakening of the immune-forming functions of the immune system and the low production of immunogens, which can not protect the body. The presence of malignant tumors in the body disrupts the overall metabolism. In the initial period of tumor formation, metabolism increases and decreases in the next period. Blood glucose may increase or decrease.

Increased activity of enzymes involved in carbohydrate metabolism increases lactic acid in the blood, including in the veins. A decrease in serum albumin in the blood leads to a decrease in protein and an increase in residual nitrogen. Decreased albumins are associated with decreased protein synthesis. When tumors grow, the activity of arginase, catalase, oxidase in the liver decreases, glycogen synthesis, urea, guipuric acid formation is impaired, the total amount of nitrogen excreted from the body increases, and urinary urea decreases. In the urine, lactic acid, polypeptides, some amino acids increase, and acetone cells appear. According to NBMedvedev, in cancer, carbohydrates are 6-7 times more than nitrogen. Tumors cause hypochromic anemias in the body, decreases to 0.5 to the color index of the blood. Anemia is caused by the breakdown of erythrocytes under the influence of various charged substances, ie not completely oxidized. Disruption of the control of the activity of blood-forming organs by the formation of erythrocytes by nerves and endocrine glands leads to anemia.

During the transplants, he observed that the infinite features of the tumors were visible. Tumor strains are also present today, including the well-studied Erlix mouse cancer, Jensen’s rat sarcoma, Raus’s chicken sarcoma, and others, which have been transferred from organism to organism for hundreds of years and have existed for 50 years or more. The nutrition of the experimental animal plays an important role in transplant growth, and if the caloric content of the food is low, i.e. lysine, arginine, histidine, the growth of tumors is inhibited. If it contains a lot of carbohydrates, cholesterol and potassium in the diet, the growth of the tumor will accelerate. Liver cancer can develop even if the animal does not have enough choline in its diet. But the growth of tumors did not stop as a result of complete starvation of animals SAMMI researcher IP Mishenko observed in chickens and rats. Experiments have shown that tumors can be grown outside the body by creating special nutritional conditions, as observed by ADTimofeevsky et al. Thus, the role of the nervous system in the origin of the tumor is also important, as the causes of the tumor include chemicals, mechanical stimuli, light energies.

In the body of highly developed warm-blooded animals, body temperature changes in a very short time, and their body temperature depends on the specific condition of the animal, type, development of sweat glands, time of day, age. The temperature is not the same in different parts of the body of an

animal of the same species. Relatively uniform temperature maintenance in the body is ensured by physical and chemical thermoregulatory mechanisms, a process controlled by the CNS and endocrine glands.

Heat exchange is provided by the MNS using conditioned and unconditioned reflexes. Experiments have shown that in the back of the gray matter of the midbrain is a center that controls the formation and transmission of heat. This control is controlled by the centers of metabolism, vascular tone, respiration, and sweat secretion, and these processes are related to the activity of the hypothalamus and cerebellum. Needle puncture in the hypothalamus raises the body temperature of the animal to 2.5–30. Heat exchange depends on the activity of the shell, and in animals where the shell is removed, the heat exchange is disrupted. In dogs, it is possible to control heat exchange by a conditioned reflector pathway.

The heat exchange is controlled as follows: thermally excited cold-floating Krauze flasks excite the heat-floating Ruffin bodies and transmit the effect to the MNS. From there, impulses are transmitted to various organs, altering vascular tone, sweating, respiration, altering metabolism in the muscles and liver, and regulating heat exchange also depends on blood temperature. The pituitary gland, thyroid gland, adrenal gland, pancreas and other glands from the endocrine glands are involved in the regulation of heat exchange in conjunction with the nervous system. For example: if the body temperature rises when we send hormones or extracts of the pituitary gland, thyroid gland, adrenal glands, lower the body temperature by sending pancreatic extracts, or such changes in the pituitary gland, observed when the thyroid gland and adrenal gland are removed. As the body cools, the pituitary gland begins to secrete AKTG and the animal's resistance to the cold increases. If the center that controls heat exchange in the midbrain is injured, the body does not respond to a decrease in ambient temperature with an increase in metabolism, and vice versa. Thus, the depletion of heat exchange in the body of animals is observed when the activity of the nervous and endocrine systems, as well as the activity of peripheral organs and systems is impaired. Disorders of heat exchange are manifested in the form of hypothermia, hyperthermia and fever, all of which are caused by a violation of the control of heat exchange and are accompanied by changes in body temperature of the animal.

Hypothermia is derived from the Greek word hypo- low, terme- heat, and is characterized by a decrease in body temperature as a result of the regulation of heat exchange. Hypothermia is caused by exogenous and endogenous causes. Exogenous causes of hypothermia include a decrease in ambient temperature: humidity, increased wind, exposure to medicinal substances, and radiation poisoning.

Hypothermia caused by endogenous causes: severe blood loss, starvation, starvation, weight loss, injury to the CNS (heat exchange control center), prolonged dilation of peripheral blood vessels (shock), neonatal, other in, the activity of the center that regulates heat exchange in older animals is weakened, leading to a decrease in body temperature. Pigs cool faster than cattle because a lot of heat is generated in cattle due to the activity of the anterior chambers. Birds are resistant to cold, geese do not change body temperature at ambient temperature - 90–1020 chickens - 500, ducks - 400. Chickens are also resistant to temperature drops.

There are four periods of hypothermia:

3- During this period, the animal's body activates compensatory mechanisms that increase heat production and reduce heat transfer: narrowing of blood vessels, shrinkage, increased heat production due to muscle activity, movement and tremors, accelerated heart rate and respiration. blood pressure rises. Increases the activity of the thyroid, pituitary, adrenal glands, autonomic nervous system. General and basic metabolism, oxidation and other processes are enhanced.

4. The flexibility mechanisms of heat exchange are exhausted, heat transfer is increased, and some oxygen deficiencies are formed. But the metabolism is high and the rectal temperature drops to 29-270.

5- During this period, metabolism, cardiovascular activity decreases, respiration and rectal temperature decreases to 27-190, but during this period, if the animal is immediately warmed up, we can return to normal life processes. Cooling in the next period reduces vital processes, blood pressure, metabolism, the formation of heat completely stops, sleep is suppressed, fibrillation occurs first in the

heart chambers, then in the ventricles of the heart, the heart stops working and the respiratory center is paralyzed. the temperature in the rectum cools to 12-100.

Characteristic signs for hypothermia are the weakening of the protective mechanisms of the animal organism, phagocytosis, immune formation, oxidation-reduction processes, changes in carbohydrate metabolism, the formation of oxygen deficiency. When an animal that has died from hypothermia is dissected, we see that dystrophic changes have occurred in the liver, kidneys, heart, and CNS. In recent years, artificial hypothermia has been used in surgical practice, especially in cardiac operations, to increase the resistance of the heart muscle to oxygen deficiency. During this time, the body's metabolism slows down and oxygen consumption in cells and tissues decreases. A similar situation is observed during the hibernation of animals.

Hyperthermia (Greek hyper- high, terme- heat) is an increase in body temperature of an animal as a result of a violation of the regulation of heat exchange. It is said to overheat. Hyperthermia is caused by an increase in ambient temperature, an increase in humidity without wind. At this time, heat is radiated and decomposed to the outside, which is not formed because there is no difference in temperature between the organism and the environment. Heat transfer is a key part of heat exchange control, and even the smallest metabolism in the body ensures that there is a lot of heat and that the body temperature is kept constant. Therefore, the excess heat must be expelled from the body.

Keeping animals in tight spaces, moving them in warm rooms, in poorly ventilated vehicles, doing heavy physical work and overheating the pasture can cause the animals to overheat. The high temperature resistance and flexibility of animals depends on their type, breed, age, color, and skin coating system. Sheep are resistant to high temperatures and only after the ambient temperature is 400 and above will their rectal temperature change. The resistance of animals to high temperatures depends on the development of their sweat-sweating system.

While an increase in the ambient temperature of cattle above 300 causes an increase in rectal temperature, pigs are intolerant to this temperature due to the underdeveloped mechanism of sweating. When pigs are kept at an ambient temperature of 310, their rectal temperature rises to an ambient temperature of 0.70, causing them to die without adaptation because they do not have sweat glands. They lose steam and adapt to the heat. Excessive heat increases metabolism and disturbs rectal temperature up to 440. From small animals (piglets and calves) are heat-resistant, while chickens are heat-resistant. Under the influence of heat, the appetite of animals decreases, productivity decreases, blood composition changes, breathing and heart rate increase. The strong heat of the environment in the body causes a change in three periods.

In the 1st period, the compensating mechanisms ensure a decrease in heat generation and an increase in heat transfer. In animals, metabolism decreases, sweating increases, peripheral blood vessels dilate, blood circulation accelerates, respiration accelerates. All this increases heat transfer and ensures that the rectal temperature is maintained without rising. Increased heat transfer is associated with the passage of heated blood in the centers in the medulla oblongata (breathing, heart, blood vessels, sweat secretion, etc.). In the following periods, as a result of overheating of the organism, a second period occurs without adequate mechanisms of adaptation of the organism.

In stage II, the animal becomes agitated, pulse, respiration is accelerated, saliva excretion is accelerated, metabolism is increased, the final product is not broken down, protein is formed in the urine, rectal temperature rises to 2-30. If the heat effect still does not disappear, a third period will occur.

In period III, the activity of the nervous system decreases sharply, the heart and respiration slow down, blood pressure drops, fainting, and rectal temperature rises to 5-60. When the animal's body heats up, it stops breathing, and the heart stops beating during systole. When we examine such animals, we observe that profound changes have taken place in the parenchymal organs.

One of the conditions similar to hyperthermia is the heat stroke of the animal’s body. Such changes are observed in animals during intense muscle activity, when the temperature is high and the humidity increases. Acute heat stroke can lead to death from impaired heart function.

4. Disorders of heat exchange are characterized not only by hypo and hyperthermia, but also by the formation of fever.

Fever-fenbris is a general change of the organism in relation to the pathogenic, more infectious causes, and as a result of violation of the regulation of heat exchange in the body, the animal's body temperature rises, independent of the ambient temperature. Fever is a manifestation of disease formation, which is caused by a violation of the regulation of heat exchange, including the disruption of metabolism in relation to the causes of the disease as a secondary process in the body.

There is a difference in the regulation of physical and chemical heat exchange, while maintaining the process of thermoregulation in the body of an animal with a fever. The body that produces the fever becomes resistant to the effects of heat and cold. In a fever-producing animal, the disruption of heat exchange control depends on the type of animal, age, type of nervous system, and so on. The causes of fever are diverse, and pyro-pyrogens are substances that cause fever, and we study them into two major groups depending on their properties:

5. Causes of infectious fever - various infectious diseases.

6. Causes of non-infectious fever are protein, saline, medicated, fever caused by injury to the nervous system.

Fever is caused by the action of various pyrogenic substances on the control centers of heat exchange. Fever is hypothalamic thermal, and the delivery of these substances under the skin or into the composition of venous blood does not cause any changes. A similar situation can be caused by fever by observing the thermal pathways in the gray matter of the interstitial brain of animals or the nerve pathways leading to that part. Fever cannot be caused if the back and brain are cut apart during exposure to pyrogens. Hence, peeling is also important in the formation of fever, which can also increase injury under the influence of indifferent pathogens.

Along with the nervous system, the role of endocrine glands in the formation of heat is also important. does not participate properly. For example: removal of endocrine glands and pituitary gland, adrenal gland, thyroid gland, pancreas does not cause fever, but the endocrine glands only increase the development of fever, changing the overall biotonus of the organism, reactivity, heat exchange. affects by changing the tone of the control centers. Thus, the nervous system serves as the mechanism that initiates the formation of fever.

Depending on the degree of fever in animals with fever: in subfebrile animals the temperature rises above the upper limit of 10, in febrile animals the temperature rises above the upper limit of 20, in hyperpyretic animals the temperature rises above 30 and above. The rate and degree of fever depends on the ability of the causative agent, the reactivity of the organism, the activity of the immune system, the age of the animal, the type of nervous system, obesity, storage and nutrition.

There are three stages in the development of fever in the body:

7. Temperature rise period - stadium incrementi.

8. Maintaining a high temperature-stadium fastigil from 2-3 hours to 2-3 weeks.

9. Period of temperature decrease - stadium decrementi.

With the formation of heat in each period there is a difference in heat transfer, metabolism, activity of various systems, the reactivity of the organism. Depending on the functional state of the thermoregulatory mechanisms to the reactivity of the organism, the type and strength of the pyrogenic agent, fever occurs at different levels and in different cases. In this process, the thermoregulatory nervous mechanisms, the cardiovascular system, the respiratory system, the functional state of the sweat glands play a determining role.

Whether pyrogenic agents are always present in the body during the course of the disease. Depending on whether the thermoregulatory mechanisms work like this, the following types of fever are distinguished:

15. Permanent type fever-febris continia. The high temperature does not return to normal and causes a change around 10 in the morning and evening. In croupous inflammation of the lungs, acute anaerobic and viral diseases, the temperature may rise in the first period and fall slowly or rapidly in 3 periods.

16. Relieving or remitting fever-febris remittens. Daily changes in temperature are 10 and above in the morning and evening, due to the intense relaxation of the effects of the pyrogenic agent, which occurs in catarrhal pneumonia, sepsis and others.

17. Rising or falling intermittent-febris intermittens. In fever, the thermoregulatory mechanisms are very stable, decreasing to normal when the temperature drops to 2-30 and beyond. In acute hepatitis, people encounter malaria.

18. Tinka dryer or hectic fever-febris nectica. Body temperature fluctuates between 3-50, some temperatures fall below normal and rise again. This type of fever is observed in tuberculosis and septic processes. In animals, thermoregulatory mechanisms are formed when they are stressed, weakened, and their productivity decreases.

19. Recurrent fever-febris recurrens. Body temperature is high and normal for several days, with the pyrogenic agent intensifying from time to time. This type of fever is caused by infectious anemia in horses and recurrent typhoid fever in humans.

20. Atypical fever-febris atypica. Even if the disease progresses, the temperature does not rise, and the disappearance of the disease is accompanied by a rise in temperature, which changes several times a day. This type of fever is observed in horses' mango, sepsis.

21. Ephemeral fever-febris ephemera. It lasts from a few hours to 1-2 days. This type of fever is when vaccinated against tuberculosis and mango, after giving birth to animals, after heavy muscle work, when walking a lot in the heat, or when animals are moved in wagons. It is observed in diarrhea.

During fever, changes in the activity of the nervous system, cardiovascular system, respiration and digestion, kidneys, endocrine glands may occur. Changes occur in the nervous system that lead to disruption of thermoregulation. When the body temperature rises, the SNS is stimulated and then braked. Changes in the nervous system can also be due to the pyrogenic nature of the toxins that accumulate in the body. A characteristic change in the nervous system is caused by a sudden rise in temperature at the onset of fever. It does not cause changes in higher nerf activity as adaptation to pyrogenic substances is formed in the nervous system. This indicates that the organism is poisoned and not regenerated in the MNS. The nervous system of lean animals is impaired, The sympathetic nerve activity of the VNS increases. Changes in temperature rise in young animals are stronger than in older animals. Circulatory disorders are characterized by the redistribution of blood in the body, which causes more blood flow to the internal organs and less in the skin, and later the blood vessels in the skin dilate and more blood flows. The work of the heart is accelerated by the rise in temperature to this maximum, which is caused by the excitation of the sympathetic nerve, the excitation of the cardiac nervous muscle apparatus by hot blood, pyrogens and toxins. Usually a rise in temperature to 10 causes the heart to beat 8-10 times faster. In diseases such as tuberculosis and meningitis, pulse formation weakens when the temperature rises, which is a sign that the disease is getting worse. Some fever develops arrhythmia, In the third period of fever the heart rhythm slows down. While blood pressure rises first, which is associated with increased heart rate, vascular spasm, in the third period, blood vessels dilate, heart rate slows, and blood pressure returns to normal. Sometimes in the third period the blood drops sharply, ie collapse occurs.

Fever changes the quantity and composition of the blood, the intermediate products of protein metabolism in the blood are residual nitrogen, acidic substances increase, alkaline reserve decreases, leukocytes either increase or decrease. ECHT is accelerating. The presence of microbial plaque and even microbes in the blood of animals with fever, the formation of antibodies, etc.

Respiration is accelerated by the excitation of the respiratory center by pyrogenic substances and toxic products contained in warm blood, depending on the activity of the heart. Acceleration of respiration is observed in anthrax, swine fever, pneumonia. Acceleration of respiration has a compensatory effect, increasing heat transfer and increasing the body's oxygen saturation.

Digestive system activity is inhibited, appetite is lost, gastric and endocrine and motor activity is inhibited, and absorption is impaired. The process of putrefaction in the intestine intensifies, gas accumulates and flatulence develops. Digestive disorders lead to the development of autointaxia and deepening of pathological processes due to impaired absorption of nutrients. Disorders of the digestive

organs are associated with increased activity of the nervous system, including the sympathetic nervous system in the VNS.

In ruminants, the motility of the pancreas is disturbed during fever, the secretion into the pancreas is reduced, the acidity is increased, and the microflora and microfauna of the large intestine and microbiological processes in general are disrupted. As a result, the chewing period is broken. Hypo and atony of pre-gastric lesions develop. Food is not digested by stopping in the pancreas. In other animals, movement, motor, secretory, and absorption processes are disrupted throughout the intestinal system during fever. At this time, only water is absorbed from the intestine. During the heating period, animals should be given plenty of water and easily digestible carbohydrate foods to reduce the amount of concentrates in the feed.

There are also changes in the digestive system during the fever period, in the first period there is a lot of blood flow to the internal organs and a lot of urine, while in the second period there is a decrease due to water retention in the body. In the third period, urinary excretion increases again, and the composition of urine changes, glucose sometimes appears protein, albumen.

Sweating decreases in the first and second periods of inhibition of nerve centers, and increases strongly in the third period. Increased digestive processes have a compensatory effect, releasing fever from the body, the release of toxic and pyrogenic substances in the tissues, as well as certain products of metabolic processes in the tissues, and normalize body temperature.

During fever in the liver, the ability of machevina and glycogen production is weakened, the residual nitrogen in the venous blood from the liver increases, and in some fevers, bile secretion decreases.

From the endocrine glands, changes occur in the pituitary, thyroid and adrenal glands, the secretion of AKTG in the pituitary gland increases, and the activity of the thyroid gland increases. The amount of corticosteroids in the blood and urine increases.

Pathological anatomical changes cause dystrophic changes in the parenchymal organs, swelling of the organ, fatty infiltrations.

When there is a dystrophic condition in the organs, including parenchymal dystrophic changes, they disrupt their function, which in turn affects the process of fever. The formation of dystrophic changes in the organs occurs under the influence of overheating, infection and intoxication of the organ.

6. Metabolic disorders during fever are associated on the one hand with the rise of pyrogens in the body. In addition, fever leads to starvation from decreased intake and absorption of nutrients.

Metabolism is disturbed in various ways during the period of fever, however, the general laws specific to fever are not absent. During many fevers, an increase in metabolism, with an increase in dissimilation - an increase in heat production and an increase in basal metabolism by 5-10%, an increase in cardiac and respiratory activity - intensifies the oxidation process.

During the fever, protein metabolism changes, protein breakdown increases due to toxic and thermal factors, instead of the normal 15-20%, proteins are used as a source of 30% energy, 30% of nitrogen-fixing substances in the urine are ammonia, creatinine, urea and others. substances are separated. As a result, the body loses a lot of protein, at which time the body needs to be fed with easily digestible carbohydrates, if the fever is infectious, it is necessary to put glucose.

In chronic infectious fever, fat metabolism is increased, at which time excessive fat consumption is not only associated with fever, but also with starvation and poisoning of the animal. According to some scientists, changes in the activity of the gray matter in the midbrain, the center that regulates fat metabolism, lead to disruption of fat metabolism. Infectious and aseptic fevers are rarely accompanied by hyperglycemia, glucosuria, which is associated with a strong breakdown of glycogen in the liver and muscles and a violation of the regulation of carbohydrate metabolism.

Water - salt metabolism changes during the heating period, the accumulation in the tissues of incompletely degraded products of protein and fat metabolism, causing a lot of water retention in the tissues. Renal function plays an important role in this process, high temperature and toxins are reduced in the second period of diuresis, disrupting the filtration of the kidneys. In the third period, heat transfer, sweating, and diuresis increase, and large amounts of water are released. Salts also increase in

the body as water is retained, many chlorides are retained, and many begin to be excreted in the third period. The release of phosphorus and potassium salts in fever is also enhanced by the intensification of decomposition processes in tissues.

Failure to raise or weaken the temperature during certain diseases in humans and animals has had serious consequences. Other investigators recommend the use of antipyretics during fever, given the toxicity of the organism during fever. When the problem is solved correctly, IPPavlov looks at the disease from the worldview, and if the disease simultaneously disrupts the activity of the organism, the second eliminates the cause of the disease. According to IPPavlov, when the body is affected by adverse causes, the body reacts sharply to this cause. From this process we must be able to distinguish the true disease and the physiological protective process.

, hemolymph is formed, and hemolymph is rich in inorganic and organic substances, which contain proteins and oxygen-carrying pigments.

4. In the organism of hot-blooded animals there is a liquid tissue deposit, the composition of which has complex and extremely important functions. The importance of blood in the body depends on its function. Blood transport in the body. thermoregulation. The physicochemical environment for cells and tissues is very important in the protection and correlation, ie the coordination of neuro-humoral processes. Therefore, changes in the composition of the blood have a huge impact on all functions of the body.

There are several theories about the formation of blood, of which AAMaximov's unitary theory explains the formation of blood in hemocytoblasts - the mother cells of the blood, while later proponents of the duolistic theory explain that Erlix is formed in myeloblasts in monocytic sand.

Changes in the total amount of blood Depending on the type of animal, the amount of blood in the body is 4-5% of the body weight of 8 guinea pigs on horseback and up to 15% on reindeer. 55-60% of the total amount of blood falls on the liquid part of the blood (plasma), and 40-45% on the form elements of the blood (erythrocytes, leukocytes, platelets). Animals that are well fattened will have a much lower amount of blood than lean cattle. The better the muscle tissue is developed, the greater the amount of blood in the animal’s body.

The bulk of the blood (around 50%) is in the blood depot. The amount of moving and stored blood depends on the functional state of the organism. The amount of blood in the body increases or decreases under various pathological influences, during which time the ratio between the liquid part of the blood and the shaped elements changes.

An increase in the total amount of blood. An increase in the total amount of blood in the body means hypervolemia or pleural effusion in Latin huper- excessive, volumen- volume, and there are simple polycythemic and oligocytic types.

3. In normal hypervolemia, the ratio between plasma and erythrocytes is almost unchanged. Under normal conditions, this type of hypervolemia does not occur. Normal hypervolemia occurs after blood transfusion, and such artificially generated hypervolemia quickly return to normal due to the breakdown of erythrocytes in the blood that are then implanted in the body after first plasma filtration (transfer to surrounding tissues).

It is not dangerous to transfuse around 60-80% of the total blood volume of this organism into the body.

4. Polycythemic or true hypervolemia is caused by an increase in the total amount of blood in the body at the expense of erythrocytes. In this type of hypervolemia, an increase in blood volume leads to hyperemia in the mucous membranes, an increase in blood pressure and hypertrophy of the heart.

The blood-forming properties of the red marrow increase — in the tubular bones, the fatty marrow is replaced by red marrow, and young erythrocytes appear in the blood. Polycythemic hypervolymia is caused by chronic oxygen deficiency.

5. In oligocytic hypervolemia, the total amount of blood increases at the expense of the liquid part of the blood, i.e., the amount of water increases. This type of hypervolemia is called serous or hydremic pleurisy. This type of pleurisy occurs in kidney disease, which causes excessive water retention in the body when drinking too much water. Hydremic pleurisy cannot be called

experimentally, because no matter how much saline is added to an animal's body, the deposited fluids pass into the interstitial spaces and are expelled from the body, or a very short-term increase in blood pressure occurs. observed. An increase in water content (hydremia) in the blood can occur even without an increase in the total amount of blood. This hydremia is caused by a decrease in dry matter and protein in the blood, when there is severe weight loss (cachexia), when a lot of blood is lost,

Decreased total blood volume is called hypovolemia or oligemia, which means hypo-less, decreased, volumen-volume, and is divided into simple, pilitsetemic, and oligocytemic types.

9. In normal hypovolemia, erythrocyte and plasma ratios are unchanged, resulting in a decrease in total blood volume and excessive blood loss. Injury to the vessel wall with mechanical injury or tumor. excessive blood loss due to inflammation or wound processes can lead to hypovolemia.

Sometimes a decrease in blood can also be caused by taking blood from a donor. Older and younger animals are more susceptible to blood loss than middle-aged or adult animals, while diseased organisms are more susceptible to blood loss than healthy organisms. It is dangerous for the body when the body loses 60-70% of blood and 15-30% of blood loss when the body overheats causes death. Death occurs even if the body loses about 50% of its blood quickly and in a short time. If the amount of blood lost in the body does not exceed 25%, the blood pressure in the blood vessels decreases for a short time and immediately normalizes due to an increase in vascular tone by reflex and the release of stored blood into the blood vessels. If the body loses more than 25% of its blood, a long-term stable blood pressure drop occurs. When there is a lot of blood loss, the number of erythrocytes decreases, oxidation processes in the body are provided by oxygen transported by erythrocytes present in the body. A similar situation is observed when the blood is thinned (hydremia), that is, when interstitial fluid flows into the bloodstream. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases.

Excessive blood loss leads to oxygen deficiency. When the nervous system is excited first, it then exhausts the centers that control respiratory and vascular tone by creating a wide-section braking. Cardiac function weakens, body temperature drops, and death occurs from paralysis of the respiratory center. Changes in body functions, hypovolemia or a decrease in total blood volume play a key role in lowering blood pressure. When blood is lost, it is important to put blood in the body, because if we put a saline solution at this time, the liquid part of the delivered solution passes from the blood vessel to the tissue.

6. In polycythemic hypovolemia, the total amount of blood decreases due to the liquid part of the blood, and the amount of erythrocytes increases per unit volume. In polycythemic hypervolemia, the absolute or absolute amount of erythrocytes is normal and the dry matter and viscosity of the blood increases. The decrease in the fluid content of the blood may be due to the body not consuming water. The strong viscosity of the blood prevents it from passing through the bloodstream, including through the capillaries.

7. In oligocytic hypovolemia, a decrease in total blood volume is associated with a decrease in erythrocytes in the blood. This type of hypovolemia can be observed in cases of excessive blood loss due to incomplete recovery of the fluid portion of the blood and some anemia and anemia.

Blood transfusion. When transfusing blood: a) lost blood - proteins, enzymes, hormones of the form elements of the blood are replaced, and the transfused blood participates in the performance of biological functions.

b) has a stimulating effect - that is, increases metabolism and blood formation.

c) increases blood clotting and stops bleeding.

g) cleanses the blood of toxins because erythrocytes and proteins in the transfused blood absorb toxins. Due to blood transfusion, blood pressure is restored, the body's stability is increased. It is used in cases of severe blood loss from burns, shock, collapse, diseases that reduce the reactivity of the organism, and general weight loss, because the blood affects various functions.

Until the twentieth century, blood transfusions were not widely used due to various tragic changes as a result of blood transfusions. The creation of the teachings of K. Landsteiner and Yansky on blood groups opened a wide way for blood transfusion.

The presence of blood groups is associated with antigenic causes in erythrocytes — isohemohagoglutinogen and antibody-isohemohaglutinins in serum. In determining blood groups, agglutinogen A and B in erythrocytes of blood are taken into account. These agglutinogens can occur in erythrocytes separately and both together or not at all. In accordance with these agglutinogens, agglutinins are also denoted by the Greek letters alpha and beta. An animal does not have similar agglutinogens and agglutinins.

Heterohemoagglutinins are also present in the blood at the same time as isohemoagglutinins.

Among the animals, the blood groups of horses are very clear, cattle, goats. in pigs and dogs, low levels of agglutinins in serum and low erythrocyte adhesion properties make it difficult to determine blood groups. Therefore, their blood will always need to be tested before a blood transfusion. To do this, take 2 drops of recipient serum on a vial, dilute 1 drop of donor blood 5 times in saline solution, and if agglutination does not occur within 10 minutes, this blood can be considered as recipient blood. If it does not resemble the recipient's blood, the donor solution will agglutinate. When solutions are gradually applied to the recipient, the agglutinating property is lost by repeatedly diluting with donor blood. Therefore, in practice, the focus is primarily on the donor agglutinogen and the recipient agglutinogen. If a large amount of blood is transfused, the recipient does not lose the agglutinating properties of the donor agglutinin and may cause shock in the body.

Hemotransfusion shock is a reaction that occurs when groups of blood are improperly placed in the body, and for the development of shock it is enough to put 80-120 ml of blood in groups that do not correspond to groups. As a result, the animal develops strong agitation, rapid breathing and heart rate - tachycardia. Decreased blood pressure makes breathing difficult, mucous membranes turn blue, vomit, urine and feces are no longer dependent on the activity of the organism. Shock often occurs within a short time, sometimes a few hours after a blood transfusion, and causes death. If the blood groups are not matched enough, the shock will pass immediately.

Some scientists explain that shock groups are formed by improper blood transfusion due to embolism of blood vessels in the brain, lungs, kidneys, while others explain that they are formed due to the breakdown products of erythrocytes in the recipient organism. Not all scientists agree with such analyzes. Experiments have shown that the mass formed by the adhesion of erythrocytes breaks down quickly without being stable and does not disrupt the activity of the organism. Even when hemolyzed blood is transfused, there is no shock in the animal's body. Academic AABogomolets binds to changes in the electric charge of colloidal substances during shock, as the colloidal structure of blood and tissue proteins plays a key role in the formation of hemotransfusion shock.

Due to improper blood transfusion, the structure of the recipient and donor proteins changes and the deposition of the protein micelles leads to a severe impairment of the body's function. This theory unilaterally explains the formation of shock.

In the pathogenesis of hemotransfusion shock is manifested as a major change in the reflex activity of the organism. When blood is burned in groups that do not match, it stimulates the vascular receptor to produce multiple impulses, creating a short-term strong excitation in the nervous system and then braking large parts. It therefore disrupts blood circulation, respiration, metabolism and other physiological functions.

Osmotic resistance of erythrocytes. EOR is the resistance of red blood cells to hypotonic solutions, and there is a difference between minimum and maximum resistance.

Minimum resistance is defined as the level of hypotension in which gamma-resistant erythrocytes break down and hemolyze. At maximum resistance, all erythrocytes are broken down, and the concentration of the saline solution is taken into account when assessing the degree of hypotension.

The resistance of erythrocytes depends on their structure, the resistance of erythrocytes in the changed form is low and hemolysis occurs. In addition, the resistance of erythrocytes to hypotonic solutions depends on the layer of lipoid protein formed on the erythrocytes. Due to the lack of lipids and phosphorus in the newly released erythrocytes, they break down earlier than the old erythrocytes. The state of maximum resistance indicates that the bulk of the erythrocytes are mature erythrocytes. An increase in EOR is observed in mechanical jaundice, in cases of poisoning with hemolytic toxins, in pathological conditions accompanied by tissue breakdown. Increased osmotic resistance of erythrocytes is also associated with the deposition of cholesterol and broken down tissue proteins in the body of erythrocytes.

Decreased EOR occurs when starving, in hemolytic jaundice, and in other diseased states of the organism.

Hemolysis is the rupture of red blood cells and the release of hemoglobin into the surrounding fluids. Blood or erythrocytes become discolored after hemolysis. Hemolysis occurs in and outside the blood vessel. Some erythrocytes also break down due to their own death. If in the physiological state erythrocytes are broken down by splenic macrophages, in pathological cases the breakdown of erythrocytes also involves the macrophages of the liver, red marrow and other organs.

Causes of hemolysis include:

1. Infusion of erythrocytes into hypotonic solutions.
2. Heating of blood or erythrocytes 62-630.
10. Re-freeze and thaw the blood.
11. The effect of rays.
12. The effect of electric current.

The hemolytic effect of light energy occurs in the presence of photosensitizers such as eosin, fluoroacin and others.

Hemolytic effect is manifested by chemicals such as nitrite, nitrobenzene, ether, benzene, case and deoxycholate acids, and others. Under the influence of chemicals, the erythrocyte membrane breaks down, disrupting the binding of hemoglobin to erythrocyte strain. Hemolysis-causing substances include bee venom, chaen snake venom, tetanolysin, staphylolysin, and many other microbial toxins. The hemolytic effect of toxins is based on the hydrolysis and softening of the erythrocyte shell by phospholipids. Erythrocytes are also broken down by blood parasites. Specific immunoassays to erythrocytes may be the effect of hemolysins as the cause of hemolysis. Sometimes substances in the blood serum that are formed under the influence of tumors, radiation and other diseases break down erythrocytes to form autohemolysins.

From the breakdown of erythrocytes in the bloodstream, hemoglobin dissolves in blood plasma and is excreted in the urine. In the gradual breakdown of erythrocytes, hemoglobin and erythrocyte fragments are captured by RES macrophages, resulting in complex changes to form the pigments bilirubin and hemosiderin.

Multiple breakdown of erythrocytes primarily increases the excretion of bilirubin by bile, which in turn increases stercobilin in the feces and excretes urobilinogen in the urine.

Iron released from erythrocyte breakdown is stored in liver and spleen macrophages. Here, after complex chemical changes, iron is released into the bloodstream and transported to the red marrow, where it is used in hemoglobin biosynthesis.

From the disruption of the normal change of hemoglobin, excess porphyrins-red violet-colored pigment is formed, which separates with the urine and turns the urine red. Due to the sensitization of porphyrin to light, its sensitivity to sunlight is increased. There are reports of parfirinuria as an

inherited disease in Shortgorn pedigree cattle. Parfirinuria also occurs when poisoned with mercury, lead and sulfonamides.

Anemia is a decrease in hemoglobin and erythrocytes per unit volume of blood. In anemia, erythrocytes undergo qualitative changes, pathological forms of erythrocytes are formed, which differ in size, shape, saturation with hemoglobin. The total amount of blood in anemia is either reduced or maintained at normal.

Classification of anemia. One of the most common classifications of anemias is to classify them according to their origin. Depending on the origin of anemia is divided into pasthemorrhagic, hemolytic, elemental and infectious types.

3. Posthemorrhagic anemia occurs when there is a lot of blood loss in the body. Acute posthemorrhagic anemia occurs as a result of sudden multiple or multiple - multiple chronic blood loss. Bleeding from blood vessels due to injury, ulceration of the intestines and stomach from internal organs, tuberculosis of the lungs, bleeding in the nasal cavity, tumor growth, bleeding as a result of childbirth, etc. is formed.

Restoration of the blood component after blood loss Normal red blood cell count is restored in a few days to 2-3 weeks, depending on the amount of blood lost by the body. Recovery of hemoglobin after extensive blood loss occurs gradually. In the blood, hypochromic erythrocytes are formed polychromatophils, reticulocytes and normocytes. The color of the blood decreases, the amount of leukocytes increases. Chronic diseases, changes in the quality of nutrition, reduce the regenerative properties of red blood cells and cause severe anemia. Decreased red marrow activity leads to anisocytosis and paikilocytosis, and sometimes to the formation of extramedullary blood in the spleen, liver, lymph nodes.

Hemolytic-toxic anemia is caused by toxins that break down erythrocytes. Some substances break down erythrocytes, directly in the blood vessels, some break down blood cells and then break down in RES macrophages. In the origin of toxic anemias, the formation of blood and the violation of the reflex control of its breakdown are of great importance. does not cause anemia when administered.

In hemolytic anemia, bilirubin in the blood increases, urobilinogen is excreted in the urine, and sometimes free hemoglobin is also excreted. First of all, the color of the blood is suddenly higher, and undigested erythrocytes are absorbed into the body, absorbed. Blood formation is enhanced by strong breakdown of erythrocytes. In the blood there are large numbers of polychromatophiles, reticulocytes and sometimes normoblasts. The color index of the blood suddenly decreases. Due to the good regenerative properties of red marrow, the composition of the blood is quickly restored with the loss of toxic effects. In chronic hemolytic anemia, the blood-forming organ becomes tired, its activity weakens, and erythrocytes with various defects in the blood fall into the bloodstream, and anisocytosis and poikilocytosis are observed. The amount of erythrocytes in the blood decreases sharply.

5. Alimentary anemia is caused by a lack of vitamins, proteins, trace elements in the diet, cobalt and copper, ie substances involved in the synthesis of hemoglobin. Alimentary anemia has a hypochromic character and the blood color index is less than one. Alimentary anemia is observed in young animals, especially piglets. Alimentary anemia is caused by inability to assimilate nutrients well during diseases of the gastrointestinal tract.

a). Anemia caused by iron deficiency is caused by a disorder of iron metabolism in the body. In this type of anemia, not only is there a decrease in erythrocytes, but also a decrease in the amount of hemoglobin. In severe anemias, anisocytosis and poikilocytosis occur. In pigs, iron deficiency in pigs resulted in the development of anemia in piglets at 1–6 weeks and up to 70% mortality.

b). Anemia caused by protein deficiency As a result of a lack of proteins in the diet or a decrease in their absorption, the synthesis of globulin protein is disrupted and hemoglobin is not formed.

4. Infectious anemia is caused by filtering viruses in horses and other ungulates. While some scientists explain the formation of this anemia as a direct breakdown of erythrocytes under the influence of viruses, others explain that the viruses are associated with causing red marrow hypofunction. The amount of erythrocytes in 1mm3 of blood of animals with infectious anemia is reduced by 1-2 million. Anisocytosis, poikilocytosis and other changes occur in the blood. In

infectious anemia in the red marrow occurs the replacement of the yellow marrow with red marrow, the formation of extramedullary blood in the spleen, liver, lymph nodes.

Regenerative and oregenerative anemia occur depending on the functional state of the blood-forming organ.

In regenerative anemia, the regenerative properties of the blood-forming organs are well manifested. As a sign of regenerative status in the peripheral blood are formed hypochromic, polychromatophilic erythrocytes, reticulocytes, erythrocyte nucleus remnants (Jolie bodies and Cape rings), normoblasts. When strong regenerative properties are manifested, the yellow marrow turns into red marrow, and in the liver spleen, extromedular blood formation occurs in the lymph nodes. Such changes disrupt blood formation and are formed from cells of the embryonic period — megoloblasts, macrocytes. Oxygen deficiency is an intermediate product formed during anemia, as a cause of regenerative processes in the blood-forming organs.

Aregenerative or hypoplastic anemia results from fatigue of the blood-forming feature of the red marrow. In hypoplastic anemia, the red marrow loses its ability to form erythrocytes, young erythrocytes in the blood decrease, the red marrow turns into yellow marrow, and has a hypochromic character. Weakening of the blood-forming organ is observed during avitaminosis, infections (tuberculosis, paratuberculosis, infectious anemia, sepsis), strong toxins, radiation sickness. Under certain conditions, any anemia can progress to a type of hypoplastic anemia. In most cases of anemia, erythropoiesis is not impaired, but leukopoiesis is also impaired.

In organisms, the compensatory mechanisms in anemia change. The function of oxygen supply to the blood is weakened, a number of flexibility mechanisms are formed: accelerated respiration, increased blood circulation and blood formation. As the heart beats faster, blood circulation speeds up and more blood flows through the capillaries over time. Accelerated and deepened respiration increases the saturation of the blood with oxygen in the lungs, increasing the formation of broken erythrocytes in the blood-forming organs. Compensatory properties are associated with the ability of tissues to fully absorb oxygen from arterial blood.

In severe hemoglobin deficiency in anemia, normal gas exchange is ensured in animals due to the activities of compensatory mechanisms. But weak movements during anemia cause a lot of oxygen demand, accelerated breathing movements, and tachycardias. Acedosis develops when there is an increase in incompletely broken down intermediates in the blood.

Polycythemia - or polyglobulia (Greek poly poly, globulus-ball, kutos-cell) is an increase in the number of erythrocytes in the blood per unit volume. Polycythemia is divided into absolute and relative types. In relative (false) polycythemia, the fluid content of the blood decreases and the number of erythrocytes does not change. This type of polycythemia occurs when sweating, severe diarrhea, diabetes mellitus, severe isthmus, dehydration and other pathological processes. In relative polycythemia, the total amount of blood is often reduced or unchanged.

In absolute polycythemia, erythrocytes proliferate due to increased erythropoiesis. In most cases, absolute polycythemia serves as a resilience reaction in the absence of oxygen to the body. Lack of oxygen increases the flow of erythrocytes from blood depots and blood-forming organs into the bloodstream. Absolute polycythemia develops when external respiration is disrupted (pulmonary emphysema, when the upper airway narrows, O2 partial pressure decreases in atmospheric air), when blood circulation is disrupted. Polycythemia also occurs when poisoned with copper, phosphorus, cobalt, arsenic. Polycythemia is a physiological condition in newborns, ie in the first days of life of calves erythrocytes in 1 mm3 of blood are 10.5 million. and a month later it dropped to 7.5 million.

Changes in white blood cells. Leukocytes, i.e. white blood cells, are formed in the red marrow, lymph nodes, and spleen. The stem cells that produce leukocytes are called hemocytoblasts, and the hemocytoblasts form myeloblasts, the primary cell of granular leukocytes in the red marrow. Lymphoblasts and monocytes are produced in the lymph node and spleen. In the blood of a healthy animal, there are many joint nuclei, and a small number of rod nuclei are found. Young neutrophils are not always present, and when blood-forming organs are tickled, rod nuclei proliferate, and in some cases myelocytes also occur.

Leukocytes include plasma cells, i.e., lymph nodes, spleen, and products of reticular and endothelial cells of the red marrow. Immune cells are formed due to the activity of plasma cells. During normal blood formation, plasma cells are found in the blood-forming organs, while in healthy animals, they are almost never found in the peripheral blood. The cytoplasm of plasma cells is stained dark orange, and the nucleus is round or oval in shape.

A leukoformula is a list of leukocyte types to determine the percentage of individual leukocyte species. In the leukoform of cattle, sheep and pigs, lymphocytes are abundant in the blood of horses, dogs and cats, and neutrophil leukocytes are abundant. White blood cells differ in type, and the leukoforms of young organisms are slightly different from those of older animals.

In determining the functional status of blood-forming organs, it is necessary to know not only the amount of leukocyte-forming organs, but also the absolute amount of leukocytes. The determination of the ratio of the main group of leukocytes in numbers is called leukocytic profile.

The main function of leukocytes is a protective function, i.e. phagocytosis. Leukocytes play an important role in the repair of damaged tissue, clearing the injured area of necrotic cells. Leukocytes produce a substance that stimulates regeneration, basophils and eosinophils are involved in neutralizing toxins. Quantitative changes in leukocytes are caused by an increase or decrease in leukopoiesis, as well as redistribution of blood in the blood vessels. As a result of dilation of blood vessels, blood flow slows down, leukocytes settle along the walls of blood vessels, and their amount in these blood vessels increases. Where blood vessels constrict and as a result blood flow accelerates, the amount of leukocytes in the blood decreases.

4.Myeloid, lymphoid leukemia and reticuloendotheliosis are distinguished depending on which part of the hematopoietic system is hyperplastic. Lymphoid leukemia is found in cattle, horses, and pigs, while myeloid leukemia is observed in dogs.

Myeloid leukemia or myelosis is characterized by hyperplasia of myeloid tissue. The yellow marrow turns into a red marrow, causing extromedular blood to form in the spleen, lymph nodes, liver, and sometimes other organs. Leukoblasts are more common in erythroblasts than erythroblasts. Myeloid leukemia is divided into leukemic and aleichemic types. In leukemic myelosis, the number of leukocytes in 1 mm3 of blood can be a hundred thousand or more. The main part of leukocytes, ie 90% and more, are granulocytes. The bulk of granulocytes are young cells, ie myelocytes, promyelocytes and myoblasts, and sometimes the number of unexposed eosinophils, basophils and erythroblasts also increases. In aleukemic leukemia, the number of leukocytes is increased around the norm and or in very small amounts. Examination of the leukoformula shows a strong rejuvenation of leukocytes. However, although their phagocytic properties are preserved, they are slightly lower than the phagocytic activity of mature neutrophils. In myeloid leukemia, the spleen becomes enlarged.

Some scientists attribute the formation of extromedular blood in leukemia to the introduction of myeloid cells into tissues and the formation of metastases, while others explain that the formation of extromedular blood is caused by the influence of etiological causes of leukemia on mesenchymal cells.

There are leukemic and aleukemic types of myeloid leukemia. In leukemic leukemia, the number of leukocytes in 1mm3 of blood reaches 100,000. The main part of leukocytes is granulocytes, which account for 90%. Granulocytes are composed of young cells - myelocytes, promyelocytes, sometimes non-myeloblastic eosinophils, basophils, erythroblasts. In aleukemic myelosis, the leukocytes in the blood increase normally or very little. In leukoform, young cells are weaker than phagocytosis in neutrophils, whose main part is phagocytic function (myelocytes, etc.).

During lymphoid leukemia or lymphodenosis, lymphoid tissue grows and is characterized by enlargement of the lymph nodes, spleen and liver. As leukemia develops, the myeloid tissue is replaced by lymphoid tissue in the red marrow. During leukemic lymphodenosis, the amount of white blood cells in 1 mm3 of blood reaches 1.5 million, and lymphocytes make up 98% of all leukocytes. In aleukemic lymphodenosis, the number of leukocytes is normal or partially increased, lymphocytosis develops in the leukocyte formula, and lymphoblasts are also found among the lymphocytes.

Reticuloendotheliosis is characterized by proliferation of reticular cells in the red bone marrow, spleen, lymph nodes, and liver. There are leukemic and aleukemic types of reticuloendotheliosis. In leukemic retiuloendotheliosis, there is a strong increase in monocytes in the blood. In acute leukemia

the metabolism is disturbed, the productivity of the animals decreases, anemia develops and severe weight loss occurs, in chronic leukemia the animal seems to be healthy for a long time, the animal dies from malnutrition and other diseases.

Leukemia etiopathogenesis. At present, leukemia with all its symptoms is recognized as a pathological process specific to the inflammatory process. Symptoms related to the theory of blastomatosis of leukemia include:

1. The growth of hematopoietic tissue during leukemia is not differentiated like tumor cells.
2. Changes in metabolism during leukemia are similar to those in malignant tumors.
3. Carcinogens have leukogenic properties in the experiment.
4. The therapeutic effect is due to the same substances in leukemia and tumors. (M. X-rays, radioactive phosphorus, chemicals that affect cells).

In leukemia, the leukocytes are in such an atypical state that it is difficult to consider them as this or that blood-forming element. However, the process of phagocytosis is worse than in normal leukocytes. Leukemia differs from normal tumors in the formation and growth of blood in the blood-forming organs. In aleukemic leukemia, destructive symptoms characteristic of the growth of all tumors are observed.

The causes of leukemia and tumor formation are not yet fully understood. Chicken leukemia is caused by viruses. This has been studied in leukemia by sending cell-free filtrate to healthy chickens. All leukemias can be formed by injecting carcinogens. Leukemia is caused by long-term ionizing radiation in the body, the mechanism of action of which has not yet been determined.

Changes in blood plastics. Blood plastics play an important role in platelet coagulation and are a source of the enzyme thrombocytosis. Platelets are formed in large cells of the red marrow - megakaryocytes. Therefore, the factors that affect the red marrow affect the amount of blood platelets. A decrease in the amount of platelets in the blood is called thrombopenia, which causes a weakening of the blood clotting process. In thrombopenia, the retraction of the blood clot is weakened The blood clot is soft and does not provide a tight closure of the injured blood vessel.

The causes of thrombopenia are as follows:

1. Redistribution of platelets, ie accumulation of platelets in the blood vessels of the internal organs and a decrease in the peripheral blood vessels.
2. Weakening of platelet formation in the red marrow.
3. Strong breakdown of platelets in peripheral blood.

Thrombopenia in some infectious diseases is caused by physical, chemical causes, disruption of the activity of blood-forming organs or strong breakdown of platelets.

When thrombocytosis or an increase in the amount of platelets in the blood is cured of many infectious diseases, in myeloid leukemia, anemia is formed during the recovery of blood composition, and blood clotting is enhanced.

Simultaneously with the change in the number of platelets, a qualitative change occurs, the shape changes, does not wrinkle and undergoes other changes. The agglutination property of such blood plastics is lost, and blood flow and blood clot retraction are impaired.

Changes in blood coagulation. Blood coagulation is recognized as a three-phase process as explained on the basis of modern theories. The first phase is a complex biochemical process in which active thrombokinase is formed from active tissue thromboplastins and the action of blood platelets on serum proteins. From the inactive prothrombin enzyme in the second phase: active thrombin is formed in the blood plasma. Calcium ion, active thrombokinase and plasma protein - globulin accelerator are involved in the activation of prothrombin. Prothrombin is formed in the liver in the presence of vitamin K. The liver is one of the main sites where fibrinogen is synthesized. In the third phase, fibrin is formed from the action of active thrombin on fibrinogen. As a result, fibrin filaments are formed and blood clots form. In the body, along with the blood coagulation system, there is also an anti-coagulation system, these substances are formed in the tissues and released into the blood under the control of the nervous system. Anti-coagulation systems include 1) heparin-liver physiologically active substance formed in the lungs and blood vessels, 2) fibrinolysin-plasmin, 3) protein substances that inhibit the formation of thrombin and thromboplastin. Heparin activates the lipase of lipoproteins that

are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin.

The blood coagulation and anticoagulant system are two interconnected parts of the blood's coagulation system. Because these two systems are mutually balanced, the blood moves in a fluid state without clotting in the blood vessels.

Weakening of blood clotting. Weakening of blood clotting: 1) due to insufficient intake of vitamin K in the body or impaired synthesis of prothrombin and fibrinogens in pathological processes of the liver. 2) when there is a decrease in platelets in the blood - in thrombocytopenia. 3) decrease in calcium ions in the blood. 4) excessive development of the anticoagulant system in the body - heparin and others. 5) when anti-coagulants, ie substances that weaken blood clotting, are injected into the body.

When animal blood has a low coagulation property, a small mechanical injury can cause bleeding into the subcutaneous tissue, mucous membranes, muscles, and other tissues. The easiest bleeding occurs in the nose, lungs, intestines.

By treating the blood vessels with paraffin, if blood collects in the arteries, the blood becomes coagulated. A 5% sodium hydroxide solution of citric acid is often used to make the blood non-coagulating. Anticoagulants include dicoumarin and other anticoagulants extracted from the head of the leech. These substances stabilize the blood by inactivating thrombin. We can use the stabilizing properties of these substances by injecting them directly into the body or adding them to freshly drawn blood.

Acceleration of blood clotting. Accelerated blood clotting is associated with vascular injury. Blood platelets easily sink into the injured vascular wall, break down due to low resistance, and form active thromboplastin-thrombokinase. Blood coagulation can be formed by the strong breakdown of tissues by sending to the body extracts prepared from blood serum and organs. Increased blood coagulation after excessive blood loss is associated with the influx of many interstitial fluids rich in thromboplastin factor into the blood. Based on this mechanism, the delivery of calcium salts, multi-vitamin K, when hypertonic solutions are injected into the blood, increases blood coagulation. Increased blood clotting in the body can lead to thrombosis and embolism.

Changes in the biochemical composition of the blood. Minerals are ionized in the blood and are in a molecular state as well as in a state of binding to proteins from colloidal substances. Minerals are involved in blood osmotic pressure and other complex physicochemical processes. Minerals are not evenly distributed between the blood plasma and trace elements, the amount of calcium, potassium, sodium and other minerals in the blood of healthy animals is always kept the same, even when saline solutions are sent to the body.

Calcium. Ionized calcium is physiologically active, accounting for 45-55% of total calcium. Combined with non-ionized calcium mining proteins. The amount of calcium in the blood depends on the functional state of the autonomic nervous system. Calcium decreases when sympathetic nerve tone decreases, and calcium increases in blood when parasympathetic nerve tone decreases. Calcium salts thicken cell and tissue membranes.

A sharp decrease in calcium levels is caused by a deficiency of glands near the thyroid gland and causes hypoproteinemia due to the fact that part of the calcium is bound to proteins. The amount of calcium in the blood is reduced in nephritic anemia congenital paresis. Decreased calcium intake increases vascular permeability, excitability of the CNS and peripheral nervous system. Calcium intake is also caused by impaired intestinal absorption in chronic diarrhea.

Potassium. In many animals, the amount of potassium in erythrocytes is higher than in plasma, and the amount of potassium in plasma increases when erythrocytes break down. Damage to erythrocytes

causes the release of potassium from erythrocytes into plasma due to increased permeability without breaking them down. The amount of potassium in the serum increases in severe diseases when the tone of the parasympathetic nervous system increases, regardless of its nature. Potassium and calcium affect the excitability of the nervous system. Deficiency of potassium in the body leads to weakening of muscle activity.

Sodium. Occurs in the blood plasma mainly in the form of chlorides, partly biocarbonate and other salts. Chlorides are reduced in the blood when sweating, diarrhea, vomiting, weight loss, impaired intestinal permeability, kidney disease. Decreased chlorides affect osmotic pressure and increase the breakdown of tissue proteins, weakening the activity of the adrenal cortex. The amount of chlorides increases in the blood during kidney disease, ie nephritis. The onset of hyperchloremia is caused by increased pulmonary ventilation, as a result of which chlorine ions pass from the tissues into the blood.

Phosphorusoccurs in the form of organic and inorganic compounds. In animals, inorganic phosphorus in the blood is reduced in pregnancy, rickets and osteomalacia. Hyperphosphatemia is caused by fever, lack of oxygen, uremia, exposure to vitamin D and ultraviolet light, as well as a lack of glands under the thyroid gland.

Ironenters hemoglobin and occurs in the form of other compounds only in 2% of cases. Therefore, iron varies depending on the amount of hemoglobin. In anemia, iron in the blood is reduced. Blood contains trace elements such as iodine, bromine, fluorine, magnesium, copper, manganese and others. The amount of micronutrients in the blood is affected by the nervous and endocrine systems. Detection of micronutrients in the blood is important in the diagnosis of metabolic diseases.

Proteins and products of protein metabolism. Protein and its fractions are different in the blood of different animals. Some proteins combine with fats and carbohydrates to form double compounds - lipoproteins or glycoproteins. Although many proteins (e.g. enzymes) are present in very small amounts in the blood, they have very important physiological activity. Most of the blood plasma proteins are synthesized in the liver. Decreased total protein in the blood (hypoproteinemia) is caused by eating disorders (malnutrition, protein starvation). Causes of hypoproteinemia include urinary excretion of proteins, liver toxicity, excessive blood loss, severe degenerative diseases of animals (tuberculosis, malignant tumors, chronic purulent processes, etc.). In hypoproteinemia, mainly albumin function is reduced, while the globulin fraction is significantly reduced. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. leads to a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. leads to a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such

as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases.

Blood plasma increases globulins in infectious disease and starvation. After immunization, gamma globulins in the blood increase sharply. However, an increase in gamma globulins is not associated with an increase in antibody levels. An increase in non-specific gamma globulins in the blood and an increase in gamma globulins may be due to a decrease in specific antibodies, as AE Gurvich found. Decreased albumin fraction in the blood is observed in hepatitis and cirrhosis. Therefore, in patients with impaired liver function, the total amount of proteins in the blood plasma and some fractions are variable.

Residual nitrogen in the blood is the protein-free nitrogen of the blood or the nitrogenous substances that remain after the deposition of proteins in the blood is 20-40 mg%. Increased residual nitrogen in the blood (azotemia) is observed in disorders of renal, hepatic and intestinal permeability. The amount of residual nitrogen in the blood is 200 mg% and more when the renal excretory function is impaired. In azotemia associated with renal (retention) activity, an increase in the amount of residual nitrogen occurs due to urea.

In cachexia, leukemia, and infectious diseases, the accumulation of large amounts of nitrogen-fixing substances in the blood due to the breakdown of tissue proteins causes azotemia. In hepatitis, azotemia is caused by polypeptides, which can also lead to a decrease in the amount of urea in the blood. Such a change is observed in liver disease when the deamination of amino acids is impaired, the synthesis of urea is weakened, and the transfer of ammonia salts into the blood is increased.

Accumulation of uric acid in the blood is observed in disorders of purine metabolism, gout, diseases associated with tissue breakdown, and leukemia.

There are a certain amount of free amino acids in the blood, which are intermediate products of protein metabolism. An increase in the amount of free amino acids is caused by liver disease, ie severe atrophy, poisoning by carbon tetrachloride.

Carbohydrates and products of carbohydrate metabolism.

Blood contains products of glucose, glycogen, lactic acid and other carbohydrate metabolism. The amount of glucose in the erythrocytes of most species is lower than in plasma, and this is more pronounced in pigs. Most of the glycogen is found in leukocytes. An increase in the amount of glucose in the blood (hyperglycemia) occurs when consuming easily digestible carbohydrate foods (elemental hyperglycemia), when the regulation of carbohydrates through the nervous and endocrine systems is impaired. Hyperglycemia occurs when poisoned with physostigmine, pilocarpine and other substances that affect the nervous system. The origin of hyperglycemia is in the pathology of the endocrine system, ie in the hypofunction of the islets of the pancreas Langerhans, formed in inflammation and dystrophic changes of the liver. Decreased blood glucose (hypoglycemia) is observed in chronic insufficiency of nutrition, excessive infusion or delivery of insulin into the blood, hypofunction of the adrenal hypo-thyroid gland. The manifestation of severe hypoglycemia is observed in patients with chronic cachexia.

An increase in the amount of lactic acid in the blood is observed in muscle work and pathological processes in the disruption of oxidative processes in the body, when there is a lot of blood loss, pulmonary edema, suffocation, the formation of malignant tumors. All the factors that increase the formation of lactic acid in the blood cause an increase in the amount of pyruvic acid in the blood.

Training materials for laboratory classes

ANIMAL pathophysiology is an experimental science and consists of two words: Greek Pathos - disease, illness, logos - doctrine.

The main and main method of the science of animal pathophysiology is 'experiment'. This science seeks to teach in-depth, comprehensive study of various pathological processes, diseases and their artificial models, artificially using the method of experiments. It teaches the importance of various factors in the pathogenesis of the disease, the mechanisms of disease development, the consequences of the flow.

With the help of pathological experience, the necessary conditions are created to study the causes of diseases in the past, present and future, and this is important. In studying the glycogen-forming properties of the liver, K. Bernard studied the amount of carbohydrate in the blood that goes to the liver and is present in the blood vessels leaving the liver, and found that the blood leaving the liver is low in carbohydrates.

The glycogen-forming properties of the liver were also studied by Mering and Minkovsky, who observed an increase in the amount of glucose in the blood when they examined the blood by tying two pancreatic ducts, thereby demonstrating the importance of hormones in the body. In experiments, Peer Marie proved that hypofunction of the pituitary gland leads to stunting, and hyperfunction leads to acromegaly. American scientist Simones studied the occurrence of cachexia when the function of the pituitary gland is reduced. When the Russian scientist Lunin took two groups of mice and fed one group with artificial and the other group with natural milk, a few days later the artificial milk-fed mice lost weight, lost their growth and their hair fell out, and their skin began to change. .

Trying to determine the importance of vitamins, VVPashutin feeds rabbits with sauerkraut and observes that rabbits are susceptible to sinus disease, but cannot explain the mechanism of its development.

The hypothesis of vitamins was given in 1911 by Kazimir Funk, a Polish biochemist working in London. He isolated a white crystalline substance from rice bran that could cure the disease and called it a vitamin. Latin-Vita means life amine, a chemical compound that contains nitrogen. K.Funk believes that diseases such as scurvy, pellagra, rickets, and beriberi are caused by a lack of vitamins in the body. Studies in recent years have confirmed that most vitamins do not contain nitrogen. Nitrogen-free vitamins include A, D, E, K, C. In the past, experiments have been conducted in a short period of time, using sharp experiments.

Therefore, the experiment was developed in the hands of IPPavlov, who conducted it using chronic methods.

IPPavlov spent 10 years in the SPBotkin laboratory, where the effects of caffeine, camphor, bromine on blood vessels, in particular, affecting the heart nerves, changes in blood pressure, changes in blood pressure in dogs under the influence of drugs, suturing the carotid artery in dogs , learns.

For 20 years, IPPavlov improved the methods of fistula in the physiology of the digestive system. 'rganadi. To study the role of the nervous system in digestion, the method of esophagotomy of animals explains the reflex separation of gastric juice as a result of "lying" feeding. Based on these methods, creates a diet.

IPPavlov devoted 35 years of his life to the study of mental activity and behavior of humans and animals.

The pathophysiologist uses pathological experimentation to study the causes of the disease, determine its course, find measures to prevent the disease, and develop ways and means of treating the disease, which in turn helps the practice. In particular, in the 18th century, when French wines began to turn into vinegar, IPPaster developed a method of washing and disinfecting wine containers in boiling water. When silkworm disease occurs, it is recommended that the silkworm storage rooms be cleaned of contaminants, proving that silkworm disease is caused by microorganisms.

When Louis Pasteur grows bacteria that cause cholera (malaria) and the thermostat door is accidentally left open, a few days later, he observes that the growth of cholera microbes is weakened and when he injects this microbial wash into the chickens, the chickens do not get sick. Thus, a vaccination method is created. British scientist Fleming planted in petri dishes to study the disease-causing properties of streptococcal microbes, and when the surface was left open, fungi fell on the

planted microbe, partially killing the microbes and, based on this, the first antibiotic, penicillin, was created. So the importance of experiments is significant. On the importance of experiments, IPPavlov recommends paying attention to the following two important processes:

1. Observations should be given close attention;

2.He says we study nature by focusing on the experimental method.

The French scientist Couve says that by the method of observation we hear nature and in practice we force the opening and submission of nature.

Three different problems are studied in the science of animal pathophysiology:

1.Nosology is the general doctrine of disease. In nosology, the doctor faces two different issues: one is why the disease occurs and what is the mechanism of its development (etiology, pathogenesis)? In the origin of the disease is studied the importance of the type, breed, sex, heredity and constitution of the animal, as well as the characteristics of disease resistance - reactivity.

2.The general typical cases that occur in all diseases and underlie all diseases or are observed in their origin are studied:

a). Local circulatory disorders;

b). Inflammation;

v). Fever;

g). Hyper and hypobioses.

In the special pathophysiology part of the science of animal pathophysiology teaches pathologies of organs or systems: blood, blood circulation, respiration, digestion, liver, digestive organs, endocrine glands and nervous system.

Later he began to teach pathophysiology and normal physiology AMFilomafitsky (Head of the Department of Physiology, Moscow University). Since he was not divided into in-depth knowledge at the time, he taught only some of the symptoms of the disease, without knowing the course of the disease. It teaches the origin of diseases by linking them to divine power. Therefore, AMFilofitsky begins to study a number of diseases in practice, as it is expedient to observe and study the disease. For example: the importance of the nervous system in cough, the method of blood transfusion, transfusion of fibrin-deficient blood, reviving dogs, and writing a work in this area, he has not lost its value so far. Nutritional chemistry is studied in the laboratory of AMFilomafitsky, and in 1842 in this laboratory VABasov developed a method of inserting a tube-fistula in the stomach of a dog. AMFilomafitsky studies various pathological processes in Russia under a microscope. For example: erythrocytes from the shaped elements of the blood, observed changes in urine output during the disease. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science. His work in the field of anesthesia is of great importance in the operation. He also managed to save the lives of many people in the war between Russia and Turkey by creating a powerful weapon-anesthesia method for the famous surgeon of that time Pirogov. Thus, despite his short life, AMFilomafitsky is a scientist who has left a big mark in the field of science.

VVPashutin, based on several experiments, knowing the importance of the nervous system, opposes R. Virkhov's cell pathology and explains that the processes taking place in the cells depend on the nervous system. Experimental observation of the formation of various pathological processes in the body as a result of lack of various substances, the study of the mechanism of origin of scurvy, feeding rabbits with sauerkraut. As a result, it is concluded that the disease is caused by a lack of any additional nutrients to the organisms. Lunin then justifies the lack of vitamins. That is why VVPashutin is called the gift-pioneer of the doctrine of vitamins.

VVPashutin organizes the largest school of pathophysiologists in Russia. One of his students was MPAlbitsky (after Pashutin he was the head of the department), AVReprov was the head of the

physiology department at the Khorkov Medical Institute, X-ray exposure, endocryology. He founded an independent school of pathophysiologists at the Kharkiv Medical Institute, where he studied the pathology of gas, heat, metabolism and endocrine systems from his students DEAlperin, SMLeytes and others. Academician ADTimofeevsky worked on tumors and studied whether tumors can be grown under artificial conditions. It is a state award winner for growing large tumors from a single cell in vivo and in vitro (inside and outside the body). Lunin works in the field of vitamins. AP Likhachev works in the field of gas exchange. VVPashutin died of a heart attack in 1901 while working as the rector of the Academy of Medical Surgery.

2- The School of Animal Pathophysiology was founded at the University of Moscow under the direction of Alexander Bogdanovich Foxt (1848-1930), a student of AIPolunin. It studies the pathological processes occurring in organ tissues, including: lungs, heart system. Creates a model of artificial pores of the heart and studies it in detail. He studies the formation of constipation in the lungs and heart in cardiovascular pathology, pulmonary, cardiac dysfunction. Professor Govril Petrovich Sakharov from the ABFoxt laboratory in the field of allergy and endocrinology, AI Talyansev develops methods of peripheral circulatory pathology, VVVoronin inflammation, AFAndreev clinical death and general resuscitation of the organism. VANegovsky studied animal pathophysiology of the cardiovascular system, on this basis he created a complex method of resurrection. GPSakharov and his students SMPavlenko and AAJuravel worked in the field of reactivity, immunology and endocrinology.

3- The School of Animal Pathophysiology in Kiev and Odessa was founded by Vladimir Valeryanovich Podvesotsky (1857-1913), who developed the humoral theory of immunity, a parasitic theory in the field of tumors. He worked on the regeneration process. He has written a textbook on animal pathophysiology and has published it in several languages. He published a journal, The Archive of Pathology and Medicine, to promote the science of animal pathophysiology. His students are LATarasevich and ITSavchenko, academician AABogomolets and others. They studied the problems of immunology, reactivity of the organism, endocrinology, and they always worked under the direction of II Mechnikov. LATarasevich and IT Savchenko worked on agglutinin, precipitate, antibodies in France at the suggestion of IIMechnikov.

Academician AABogomolets works in the field of animal pathophysiology, studying the role of reactivity in pathology, its relationship to endocrine management. He was born in 1881 in Petropavlovsk Prison and died in 1946. His mother was imprisoned for being a member of Russia's "southern liberation group."

Academician AABogomolets is the President of the Ukrainian Academy of Sciences and the First Deputy Chairman of the Presidium of the Supreme Soviet of Ukraine. Pathophysiologist-pathologist since 1924. By developing the pathophysiology of animals, he created the original pathological doctrine in medicine, which is called the physiological system of connective tissue. In addition to the supporting function of the connective tissue, it performs a trophic function and a plastic-building function. As it is composed of RES cells, it enhances phagocytosis and antibody production. Improves connective tissue function using antiretroviral cytotoxic serum. It was actively used in the treatment of many diseases during World War II. Academician AABogomolets Director of the All-Union Blood Transfusion Institute, developed a method of conserving blood (the first among physicians to be awarded the title of sos. labor hero). He identified 4 different types of constitutions depending on the nature of the connective tissue and observed more or less common diseases, depending on these constitutions. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known scientists EATatarinov, NNSirotinin, P.Gorizontov, ADAdo, LRPepelman and others. Academician AABogomolets wrote a textbook on pathophysiology, created a multi-volume work in the field of pathophysiology and was awarded the State Prize. observed more or less frequent occurrence of diseases. He founded a large school of pathophysiologists in Saratov, from which well-known

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Academician IISirotin worked on the field of acclimatization of the organism and the reactivity of the organism.

Academician REKovetsy studied the origin of tumors and the characteristics of their development in different conditions, the course of metabolism in tumors.

Academic ADAdo has worked on allergic diseases, anaphylaxis, lung disease, and has written a textbook on Animal Pathophysiology.

The new schools of animal pathophysiology were headed by well-known scientists LATaraseevich, AVReprev, ESLondon, AABogomolets SSKholatov, GPSakharov, NNAnichkov, ADSperansky.

Academician NNAnichkov (1885-1965) studied in depth the pathophysiology of the cardiovascular system, the involvement of RES cells in pathological processes and the mechanisms of origin of arteriosclerosis in the Department of Pathophysiology, Pathanatomy of the Military Medical Academy.

Experiments show that indifferent influencers play an important role in the development of diseases. For example, if dogs are injected with apomorphine for 15 days and supported with light, in the following days only the lighting of the lamp causes them to vomit reflexively. He suggested that organs could not be studied in isolation from the body, and that systematic scientific work should be carried out. He founded a large school of pathophysiologists, and even today scientists from the ADSperansky school are actively working in research institutes and universities.

IIRavich, the founder of veterinary pathophysiology, worked in the veterinary department of the Academy of Medical Surgery in St. Petersburg, critically examining Virkhov's cell theory and acknowledging the importance of the nervous system in the origin of the disease. He wrote a textbook on general zoopathology and lectured to students on the subject.

Academician MPTushnov (1879-1935), Head of the Department of Pathophysiology of the Kazan Veterinary Institute, created an original drug in pathophysiology, the lysates of which are the products of the decomposition of various organs. For example, muscle lysates are called myolysates, and when animals are released when they are tired, their ability to work is restored, mammolysates are prepared from the udder and increase the amount of milk, and ovariolysates accelerate the maturation of egg cells. Lysates are now the most common and widespread type - biostimulants. They are used in the growth and development of young animals, increase productivity and treat many diseases. Biostimulants are widely used in fattening. Including, Chlorella, which is found in billions of water, has been used to enhance productivity by enhancing all the processes that take place in animals. Currently, there are more than 45 departments of veterinary pathophysiology in veterinary institutes and faculties of the CIS countries, which are studying the effects of biostimulants on the characteristics of the organism. Most research veterinary institutes are studying the effects of biostimulants on the body's reactivity, metabolism and neuro-endocrine control processes.

The contribution of the French scientist Claude Bernard (1813-1878) in the development of the science of animal pathophysiology was significant. K. Bernard's work is studied in two periods:

The first period involved 20 years of normal physiology, proving the liver’s glycogen production function and determining its reflex mechanism. The origin of diabetes in the body proves that it is associated with dysfunction of the CNS. Demonstrates the importance of pancreatic juice and bile in the breakdown of nutrients, as well as observed an increase in body temperature. Blood and lymph determine the organization of the internal environment of the body and determine vital processes.

The second period. He has been working in experimental physiology for 10 years. It studies the importance and function of various nerve fibers in the body, the electrical properties of nerve and muscle tissue, the properties of blood, and the effects of SO2 on the body. Proves a violation of saliva production from salivary glands. A substance called Curare affects the endocrine glands and observes a decrease in the secretory process. He studied various pathological processes of the respiratory system

and wrote more than 180 scientific sources, which consist of 18 volumes. K. Bernard did a lot of work despite experiencing great difficulties. He teaches that the processes that take place in the body depend on the vital force, and that that force is random.

IPPavlov says of K. Bernard, "K. Bernard is a scientist who thought broadly and deeply in his mind, generalized physiology, experimental physiology, and experimental therapy as a whole, or combined the achievements of physiology with practice."

The famous chemist Dumas says, "K. Bernard is not only a physiologist, but he is a physiologist."

IPPavlov's doctrine is important in the development of animal pathophysiology. Prior to IPPavlov, observations were made in pathophysiology using analytical methods. Diseases of isolated organs, their integral parts have not been studied with attention to the living conditions of the animal, changes in the external environment and other related connections. IPPavlov, on the other hand, pays great attention to experimental scientific work and observes changes in body systems in healthy organisms in chronic experiments. According to IPPavlov's theory of nervousness, it is emphasized that any pathological processes in complex organisms are carried out with the participation of the nervous system, in particular, with the participation of higher nervous activity.

The organization and development of the science of animal pathophysiology in Uzbekistan was associated with the formation of the former Soviet Union, which began with the establishment of universities and research institutes in accordance with the decree of the Soviet government. As a result, the medical faculty of the Central Asian State University was established in Tashkent, which was later transformed into Tashkent State University, and intensive work in this area began. In 1921, the first department of "General Pathology" was established at Tashkent State University, which was later renamed the Department of Animal Pathophysiology. The first departments of pathophysiology were established in Samarkand in 1930, in Andijan in 1957, and in 1972 at the Central Asian Institute of Pediatrics.

At the Uzbek State Agricultural Institute in Samarkand, Farkhodi first studied veterinary pathophysiology, and from 1936, the head of the department, Associate Professor Vladimir Valerianovich Volkov. VVVolkov was an encyclopedic lecturer, a skilled experimenter, an excellent pedagogue-coach. VVVolkov was the initiator and organizer of several original scientific works with the staff of the department:

1. The causes and mechanisms of development of allergies and anaphylaxis in astrakhan sheep and goats in hot conditions;

2.Study the causes and mechanisms of development of pneumonia in sheep and goats during the summer months;

3. He has done a lot of research in the field of pathology of the region, the causes of the disease "Suyluk" in horses, the mechanism of its development and the development of methods for its detection. Today, the disease is found in humans and animals and is called trichodesmatoxicosis. In this field NXShevchenko and FIIbodullaev defended their doctoral dissertations and supervised several candidate dissertations.

4. The study of the enhancing effect of cytotoxins formed in tissues on the immunological properties of the organism of various laboratory animals (accelerated formation of antibodies to paratyphoid and colibotseliosis strains).

5. A detailed study of the effects of the parasympathetic division of the autonomic nervous system on the organism of experimental animals.

6. He made a great contribution to the training of a large number of highly qualified personnel. After the untimely death of VVVolkov in 1953, the department was headed by Associate Professor Anton Ivanovich Yarmashkeevich.

Extensive development of scientific work carried out at the department, mainly since 1961 under the leadership of Associate Professor, now Professor Ruzi Haitovich Haitov. By this time, the staff of the department sent different amounts of extracts from the liver, spleen and other parenchymal organs to healthy and sick animals, depending on the timing of their delivery, studied the mechanism of their action and developed a number of recommendations. In the Department of Animal Physiology

and Pathophysiology, tissue feeding of animal feeds has proven to have a positive effect on the growth and development of the organism and the treatment and prevention of various diseases.

Having studied the effects of many drugs against helminthiasis, a number of recommendations have been developed. The genetic features of natural immunity, especially in karakul sheep and lambs of different colors, have been extensively studied and are still being studied. In this area, Associate Professor ADDushanov developed a synthetic vaccine, which gave good results, and Associate Professor MAAbdullaev in collaboration with the senior lecturer of the department RFRuzikulov conducts important research. Under the leadership of Professor RXKhaitov«Veterinary basics»Volume 1-2, 1972, RHHaitov and A.Dushanov on "Animal Physiology" in 1975, RHHaitov and Associate Professor MA Abdullaev on "Animal Pathophysiology of Agricultural Animals" in 1980 in Uzbek, a number of manuals, He has published more than 400 scientific articles in various collections, scientific collections of universities, research institutes, international and CIS congresses and conferences. Under the direct supervision of scientists of the department 10 doctoral and 342 candidate dissertations were defended in specialized scientific councils. Researchers of the department have been writing reviews and defending PhD and doctoral dissertations in many fields of physiology. And so, In Uzbekistan, pathophysiologists study the theoretical and practical processes of modern veterinary and medical science at the Department of Pathophysiology of the Veterinary Faculty of Samarkand Agricultural Institute, the Uzbek Veterinary Research Institute, pathophysiology laboratories of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad. is studying the theoretical and practical processes of modern veterinary and medical science in the laboratories of pathophysiology of several medical universities and research institutes. Many PhD and PhDs in the field of pathophysiology have been developed and are operating in these institutes and are recognized in the CIS and abroad.

In order to strengthen the study of pathophysiology, the "Society of Pathophysiologists of Uzbekistan" was established, which includes more than 100 pathophysiologists. Pathophysiology and research work have been carried out in cooperation with veterinary institutes in Moscow, St. Petersburg, Kiev, Kazan, Almaty, Yerevan, and are still connected. As a confirmation of this strong unity, the fact that the 2nd pathophysiologists' session was held in 1972 in Tashkent is a proof of our opinion.

1.Information about the disease has been of interest to people since ancient times. Because science and enlightenment did not develop in the primitive community system, and people did not know the origin of natural phenomena, they thought only about the visible and the invisible. That is why the organism has been described as composed of mythical things found in nature, such as soil, air, water, wood (metal), and fire. Illness, on the other hand, was interpreted as being caused by an invisible divine (supernatural force) or "SPIRIT" - anima. This current is called the "ANIMISM" current or theory, and it is a picture that all diseases are invoked by this supernatural force, the evil spirit. Talented physicians began to appear in Greece 4-5 thousand years BC, who wrote down what they knew, what they asked someone, their observations on the patient, and bequeathed this knowledge to their descendants. As a result, medical science began to develop slowly. For example, they recorded discharge from the mouth, nose, and ears in various diseases, fever, foul odors, and so on. Later in Greece, doctors explained that a living organism was composed of 4 different fluids in addition to 5 different elements (blood, mucus, black and yellow grass). Thus, the current that explains health and disease with these four different fluid properties is called the Humoral Flow or Theory. So, if the fluids are normally mixed properly, health is a sign of health, and this condition is called krazia or krazis. If, for some reason, the ratio of fluids is disturbed or the juices are contaminated, improper mixing, the

disease can lead to dyscrasia or**«Discrasion»**The founder of this movement is the Greek scientist Hippocrates, who lived in the 4th-5th centuries BC.

Hippocrates was an observer, a disease-seeker, a traveling physician, who always traveled from village to village, making many observations on patients, studying the symptoms, various features, currents, and consequences of many diseases, and writing dozens of works. The role of the external environment in the origin of diseases, with great emphasis on cleanliness, developed methods of diagnosis and treatment of many diseases. He developed the laws of medicine, and in medicine there is the Hippocratic oath in medicine. The teachings of Hippocrates have been proven to be true for centuries and even now, and his works have not lost their value.

In addition to diseases, Hippocrates also tried to create constitutions of human temperaments, which included four different temperaments: choleric (yellow grass), melancholic (black grass), sanguine (blood), and phlegmatic (mucous fluid). 'p or less depending on.

The contemporary philosopher Democritus of Hippocrates also developed a theory of diseases, which he called the solid (atomic, particle) theory, which explains that diseases are caused by changes in the spacing of atomic particles in the body. This theory explains that the disease is caused by the narrowing or widening and thinning of the spacing of the particles. At the same time, idealistic schools of thought have sprung up in Greece, claiming that diseases are called by divine power, explaining that organ function, organ diseases, and their causes depend on a particular pneumonia of life. According to Plato, Aristotle explained that there are three kinds of divine or spiritual power that govern the lives of people and animals:

1. Spiritual power is located in the brain and controls the mental function of people.
2. The spirit of the animal is located in the heart and controls the movement and warmth of the animal.
3. Explains that the spirit of the plant is located in the liver and regulates digestion.

They explain that they believe that the causes of diseases are not in the external environment, but in the mental origin. At the beginning of the twentieth century, knowledge of the disease was developed by Roman physicians Galen and Sels, who, in addition to the three zinc origins, based their humoral flow on explaining that diseases often resulted from the breakdown of juices, distinguishing between hot and cold discrasions. developed treatment options. Based on the symptoms of the disease, they observed four specific symptoms of the disease: redness, edema, edema, pain, and these changes, which lead to dysfunction, called functio laesa. . Galen introduces the vivisection method into science.

After Galen, our compatriot was the famous scientist and philosopher Abu Ali Ibn Sino (Avicenna), who made a great contribution to the development of medicine. He was born in 980 in the village of Afshona, Romitan district of Bukhara region and died in 1037 in Hamadan. In 1980, Avicenna's 1000th anniversary was celebrated and her works were published. He wrote more than 300 works in various fields, especially in the field of medicine, and in 1020 wrote a book on the laws of medicine. It consists of 6 books in 5 volumes:

1. The book is devoted to the anatomy, physiology, causes, appearance, general treatment of diseases. Attention was paid to nutrition, health, deportation, vomiting, and blood transfusions.
2. The book describes more than 800 drugs derived from plants and animals.
3. The book is about diseases from head to toe, this book is dedicated to specific pathology and therapy.
4. The book deals with fever, various tumors, rashes, wounds, burns, bone fractures and dislocations, nerve injuries, injuries to the skull, chest, spine and limbs, poisons and poisonings - toxicology, makeup - is dedicated to keeping people beautiful. Recommended remedies against hair loss, obesity or weight loss. He wrote about rabies, smallpox, measles, leprosy, and plague.
5. The book describes the methods of preparation and use of drugs.

Avicenna's book, The Laws of Medicine, pays great attention to the methods of observation and experimentation in the study of diseases, and widely uses this method on various diseases. developed He identified many diseases, developed treatment methods, studied urinary incontinence, urinary tract infections, worm diseases, pulse heart disease.

In his multifaceted scientific work, Avicenna concluded that diseases must have had invisible causes, not divine powers, and that they were now identified as microorganisms.

Avicenna studied in detail the wounds, lung diseases, diabetes, plague, cholera, smallpox, leprosy, tuberculosis (tuberculosis) and many other diseases, especially in the origin of the disease. , boiled, proved that it is important to follow hygiene. He studied the effects of many drugs and found that mercury is important in diseases such as gonorrhea and syphilis. It has been proven that following a meal plan-diet is important in diseases. Although he did not know the functioning of the nervous system, he thought about the nervous system, that is, tied the sheep to the wolf, and observed that a few weeks later the sheep became frightened.

Avicenna's work on TIB laws has been reprinted 25 to 30 times in Europe and Asia, and is still being published today, and has served as a guide for physicians. By the 14th and 15th centuries, Copernicus, a Polish scientist, described the movements of the planets in the sky, Giordano Bruno's rotation of the earth around the sun, the Spanish Servetus's small circulatory system, and Leonardo-Da Vinci's anatomical tracts. V. Garvey discovers a large circulatory system based on his experiments on rabbits and dogs.

By the fifteenth century, a new direction in medicine, the iatrochemical and iatrophysical currents, began to emerge, meaning Iatros-physician.

The chemist Paracelsus conducted many experiments to prove the structure of the organism, the need for chemical elements to survive, the importance of mercury, matches, steel, iron and other elements in the health or illness of the organism. concludes that it contributes, and explains that when archaea get angry, they cause disease without releasing these elements into the body.

Iatrophysicists connect the organs of the body to the parts of a machine and pump the heart, explaining health and disease according to the laws of physics and mechanics.

In the XV1-XV11 centuries, the pathological-anatomical direction developed, and Morgani, Bish, and others began to study the body structure of animals and humans. In 1543, the Italian scientist A. Vezali began to study the structure of the body by tearing apart the bodies. 1640 Descartes wrote the reflex doctrine, 1660 Malpighi lens using the lens, renal capillaries, liver, spleen, skin structure, erythrocytes, 1674 Levenguk lens sperm movement. Morgan and Bish wrote about the changes that occur in different organs in different diseases, which led to the development of the study of pathological processes.

This means that the external environment has had two different effects on the organism over a long period of evolution, and that the organism has become accustomed to these favorable and unfavorable effects, adapted and balanced. -slowly studied and adapted, these effects are called daily or physiological, adequate effects. The processes that take place under the influence of these influences are called physiological processes and are called the norm, abbreviated for short. The second type of effects are often referred to as sudden, strong, sudden, adverse effects, which are called harmful or disease-causing, inadequate effects, and the processes that take place under the influence of these effects are called pathological processes.

Norm or health is a set of influences, conditions, adapting to their currents in a certain period of time, making them suitable for life, necessary or physiological effects, and the processes that take place and develop under their influence. called normal processes. Norma is a process that takes place in a period of stagnation, when the organism is calm and peaceful.

1.Norma-Sergey Petrovich Botkincha stagnation of life processes
is the sum.

2. Norma-Ivan Mikhailovich Sechenov and Claude Bernard describe the organism
with the balance of the external environment.

3. Norma-Victor Vasilevich Pashutin described the structure of the organism and
is said to harmonize its functions.

4.Norma-Vladimir Valerianovich Podvisosky to the conditions of our body

The structure of normative organ systems, the state in which they function without disruption. In real life, the norm is a relatively stable, changeable situation, because the absolute norm does not exist in real life. For example: consider pulse, temperature, respiration.

When one wants to study a disease, one must study it by comparing it with the norm. Both disease and health are ongoing processes in the body, which differ from each other in quantitative and qualitative changes. At the heart of both processes are two opposing processes of assimilation and dissimilation. It is impossible to know the exact time of onset of the disease, but it can be determined only by the symptoms that appear at a certain stage of development. For example, sleep is caused by fatigue as a result of overwork, which is considered a normal physiological state of the body, but in some severe infectious diseases, drowsiness also occurs, indicating a disease of the body: anthrax, typhoid, diabetes , tuberculosis and others.

1.SPBotkin described the disease as a disorder of the vital processes of the organism.

2.IMSechenov and K.Bernar described the disease as a violation of the balance of the external environment in contact with the organism.

9. VVPashutin explains the disease as a violation of the harmony of the structure and function of the organism. These descriptions of the disease provide insights into unilateral changes in the disease, ignoring various complex quantitative and qualitative changes and active processes during the course of the disease. Therefore, these definitions do not fully describe the diseases.

10. In an attempt to fully express the disease, IPPavlov proposed the following definition: a disease is an encounter of an organism with an awkward, pathogenic, gross cause and condition that affects it suddenly, suddenly, collision, ie mechanical shock, crushing, injury, exposure to chemical, physical influences or attack by microorganisms, this encounter is the beginning of a struggle between the organism and the cause , by activating all defense mechanisms against, removing pathogenic causes, cleared or enzymes, phagocytes, Acute flow diseases - from a few minutes, hours to several weeks: For example: infectious and parasitic diseases.

4. Moderate acute flow illnesses — from a few weeks to several months.

Chronic recurrent diseases are those that last for months or years, most of which are non-communicable and non-infectious.

Diseases occur in several stages as they develop in the body.

a). An incubation or latent or latent period is the time that elapses between the onset of the disease and the onset of the first symptoms of the disease. This period can range from a few minutes to a few hours, weeks, months, and even years. Tuberculosis, brucellosis, non-communicable diseases, leprosy, AIDS and others.

b). The prodromal or disease-reporting period has its own characteristics, during which general symptoms for the disease appear. For example: increase in body temperature, decrease in appetite, heart rate, rapid breathing, etc.

v). Outbreaks appear to be exacerbated during clinical trials.

g). The consequences of diseases are twofold: the animal is either cured of the disease, or the sick animal dies.

1.Diseases spread throughout the body - per kontinuitatem. As the disease progresses, one organ spreads due to adhesions to the other organ. For example, inflammation of the oral cavity continues to spread to the red intestine, then to the stomach, intestines, and so on.

2.The disease is spread by means of friction, adhesions - per kontiguitatem. Pulmonary pneumonia to the pleura and pericarditis - myocarditis, liver - stomach, etc.

The disease is transmitted through the blood and lymph - permestastazine. Many microorganisms are spread through the blood and lymph.

3. Diseases are transmitted through the nervous system - per nervorum, through nerve fibers, stolbnyak - congestion, botulism, polio and other diseases.

4. Diseases are spread by secretions, saliva, sweat, urine and feces.

Intermittent course of illness is a period of illness that is sometimes mild and sometimes severe.

The complete recovery of the body from disease is called sanogenesis. The consequences of the disease are of two types:

a). The body recovers from the disease.

b). The disease ends in death.

3. There are two types of recovery:

a). The body recovers completely from the disease.

b). The body recovers from the disease.

Recovery comes in two different ways: simple and complex. Simple ways of recovery are carried out by revealing various reflexes. For example: reflex agitation, excessive salivation, wiping tears, vomiting, sweating, coughing, diarrhea, excessive urination and excretion, tickling of the nervous system, and others

In complex treatment, the body is decontaminated by complex processes using barrier barriers, RES organs - liver, spleen, lymph nodes, red marrow, leukocytes, especially T and B lymphocytes, antibodies, etc. the cause is removed, then partially or completely repaired as a result of the recovery process. Restitution is called ad integrum if the body is completely cured of the disease. Sometimes the body can recover from the disease and recur, and the body can be severely damaged, and this is called a lytic transition to a critical and mild course.

3. Diseases can lead to dysfunction of the body without complete recovery. When the body's ability to heal is completely reduced, the body dies from the disease if the doctor's treatment does not help.

3.Death - mortis, morbi -characterized by the cessation of the continuous process of assimilation and dissimilation in the body and the cessation of heart function and respiration.

There are two types of death depending on their origin:

1. Natural or physiological death.

2. Death due to disease or pathological condition.

If 100% of all deaths are considered, only 2% of them are natural deaths and the remaining 98% are deaths due to diseases.

The doctrine that explains the formation of death is called tanatogenesis. Death occurs in several stages and is called the terminal state, they are:

1. Agony-pre-death convulsions: (consisting of peripoganal and oganal period).

2. Clinical death.

3. Biological death

As a result of death, the following changes occur in the corpse:

1.The body cools - algar mortis drops from 10 in the first days and cools to 0.20 on the second day. Of course, these changes are due to environmental changes.

2. The appearance of spots on the body - livoris mortis on the side on which the animal is lying, more spots appear and look good in hairless, unpigmented areas.

3. Hardening of the body - rigor mortis solidification of colloidal substances. Hardening begins after 8-10 hours and goes from head to toe.

4.Decomposition of the body - maceration or autolysis is formed under the influence of putrefactive and microorganisms from the external environment in the body, and the carcass begins to smell foul. If these bacteria are not present in the body, the body will become waxy.

Observations show that the animal continues to live in organs and tissues for some time after death. For example: nails, hair, hair, growth, movement of the stomach, intestinal muscles, contractions and other signs are observed. Much work has been done on the possibility of resurrecting the organism at the time of death. This condition is called resuscitation. It has now been discovered and proven that it is possible to resurrect organisms that have died by accident, and that people and animals who have died from various traumas, excessive blood loss, suffocation during anesthesia, electric trauma, various tragic events is being resurrected. Kulyabko, a professor of physiology at Tomsk University, was the first in this field in 1902.

From 1912 to 1919, the American physiologist Karel was able to use a burdock chicken heart under artificial conditions.

In the laboratory, Academician Kravkov observed the growth of nails and fur when rabbits' ears and fingers were removed and placed in special liquids. So it is possible to resurrect individual organs.

Professor FA Andreev conducted many experiments on dogs in 1913 and concluded that by anesthetizing dogs, the dogs were resuscitated by sending blood to the body and the whole organism could be revived.

1928 At a congress of physiologists and biochemists in Tbilisi, Bryukhonenko and Chechulin demonstrate an interesting experience: cutting off a dog's head, injecting blood into its veins through rubber tubes, and observing the dog's condition. saliva begins to separate when you put the sausage in the bur. In 1966 he was posthumously awarded the Lenin Prize for his invention of the AIK instrument. In 1940, Sinitsin was able to transplant and hold the hearts of frogs and fish. Academician VANegovsky created a common method of resuscitation in 1941-1945, which was suitable for the resurrection of many soldiers and officers during the Great Patriotic War. In nature, it is a near-fatal condition and is called anabiosis: and we can find it in the plant and animal worlds. In the process of long evolution, plants, animals, and microorganisms go into a state of anabiosis, adapting, in order to survive various adverse effects. For example, by reducing the osmotic pressure from extreme cold or heat, by reducing the oxygen in the air, by freezing and drying, special chemical conditions can be created, that is, by using protective substances, anabiosis can be formed. During anabiosis, all functions in animals are sharply reduced (body temperature, heart rate, respiration, metabolism are sharply reduced, reflexes are lost). Anabiosis occurs in worms, fish, frogs, hedgehogs, lizards, bears, and frogs.

In humans, a condition close to anabiosis is called secondary sleep. Lattergic sleep is caused by severe effects, severe illness, and nervous mental illness.

Aging is a three-phase process:

1. Aging in infancy.
2. Aging in adulthood.
3. Aging.

The main task of veterinarians is the prevention and treatment of various diseases. General prevention is a measure of disease prevention using various ways, methods and measures, which consists of complex economic, organizational and veterinary-sanitary measures, which are:

1. The work of improving the external environment, for this it is necessary to create cultural meadows, the transition to the zagon system, the exchange of meadows, the removal of poisonous plants found in the meadows, various harmful substances. Grasslands, barns need to be disinfected and mechanically cleaned. Surrounding the farm, arranging insulators, building cemeteries and animal cremation rooms, improving the reclamation condition of meadows, drying or increasing moisture, washing away salts and other activities:

2. Bacteriological, serological, biochemical, radioactive isotopes and other methods are used to determine the latent stages of the disease by various methods, with regular examinations, taking appropriate measures, ie X-ray machines, allergic methods, blood tests. Twice a year in spring and autumn medical examination is obligatory:

1. Etiology - teaches the general laws of origin of diseases in the body, their causes, a set of conditions. Etiology is the Greek word for aitia-cause, logos-doctrine.

According to IP Pavlov, the future should become a hygienic veterinary, hygiene. Therefore, it is necessary to protect the external environment, and a lot of work is being done in this area. IPPavlov said that it is necessary to know all the causes and conditions of the disease.

The doctrine that teaches the causes of disease is the result of a struggle between materialist and idealistic currents. This doctrine has explained the origin of diseases in a simple, mythical, teleological way, i.e. the disease is caused by the influence of zinc, contamination of juices, changes in their composition, decrease or increase, thinning of particles in the body or indicates that the disease is caused by thickening. Later in the Middle Ages the origin of diseases was badjahil zinc«archetypal»explained in connection with the wrath of God. As a result of observations, A. Vezali and Malpighi began to study the structure of the organism in depth. By this time, the development of industry, the production of dyes, the increase in the production of equipment, created favorable conditions for the study of the functions of the organism.

At the end of the 19th century, the production of wine and silk in many countries, including France, fell into disrepair. This poses great challenges for French scientists. As a result, Louis Pasteur, under his leadership, began to search for and find the causes of many diseases. As a result, they discover that microorganisms are the cause of wine fermentation and silkworm disease. Microorganisms can be used to prevent the deterioration of wine quality by washing wine containers with boiling water and disinfecting silkworm rooms. Thus, by identifying the real causes of the disease, now world scientists are doing a lot of research behind microorganisms, and German scientist Robert Cox is discovering the causes of tuberculosis, Louis Pasteur cholera, rabies and other diseases. The discovery of these diseases, on the other hand, follows a certain pattern, and this current is called the monocaual current. Mopo-single, single, couza - means cause. This doctrine is one of the most advanced doctrines of this period and deals a severe blow to religious doctrines. However, this doctrine does not fully explain the causes and conditions that cause disease, because the entry of microorganisms into the body does not always cause disease. As a result, the doctrine arises that diseases are caused by changes in the sum of many conditions, not microorganisms, and this doctrine means the conditionic conditions called the doctrine of conditionalism. This doctrine is contradicted by the inability to explain the disease, claiming that there is no clear cause for the disease, negating the importance of microorganisms in the origin of the disease.

Constitutionalismproponents of the theory explain that the disease arises from the genotypic structure of the organism, as a result of a deficiency in the constitution. The constitution and genotype do not change at all, so the disease is interpreted as a fatal process or a top-down process. With the emergence of the theory of constitutionalism, many erroneous theories have emerged. There is a misconception that people with low genes and low constitutions should be confused with people with high genes and high constitutions. As a result, Nazi Germany wiped out many nations in order to create a new race, and racist theories still prevail in many countries. These teachings exaggerate the causes of disease,

Nervism explains that the organism is closely connected with the external environment, which is due to the nervous system.

In studying the doctrine of etiology, we must take into account the structure of the organism and the principles of their solidarity, that is, we must combine theory and practice closely, which can explain the etiology in detail.

The causes of the disease are studied into 2 major groups: external or exogenous, internal or endogenous causes.

External causative agents include mechanical, physical, chemical, biological, and other causes.

3. External environmental factors that cause disease.

External causes of the disease are those influencers that affect the body from the external environment and create a pathological process. The causes of the disease are studied in close connection with the organism without self-study of the external environmental factors, and the degree of origin of the disease depends on its nature. Environmental factors that cause disease include mechanical, physical, chemical, and biological causes. As a result of absorption (reserves and electricity, light energy) or reflexively (conditionally and unconditionally) into the closed automatic (IPPavlov) MNS through the place where all factors directly affect the organism of highly developed animals by reflector).

Mechanical factors causing the disease.

An influencer that affects the body from the external environment, causing an injury to this or that in the body, is called trauma.

In such cases, the injury can be caused by mechanical (shock, bruising), thermal (hot and cold), electric current, chemical, X-rays, and even heat (fear, strong impact) and other changes. 'ladi.

Usually the term trauma or injury is used in a narrow sense to refer to changes that occur mechanically. All changes to mechanical injuries are made by crushing, wounding, sharp, impenetrable, shot bullets, pressure objects.

Stretching, crushing, beating, injuring blood vessels and nerve fibers at the site of mechanical impact. The pathological changes that occur as a result of stretching or traction depend on the strength

of the causative agent, the duration of exposure, and the physiological properties and condition of the organ or tissue that is stretched or stretched.

The bones and tendons are also stretched and stretched, and when the muscles contract, they are pulled less than when they are still.

If an organ is strongly pulled and stretched (skin, muscle ligaments, bones, etc.), it is torn and torn. Slow but long and repetitive pulling stretches (e.g. in joints) causes the connecting parts to loosen, causing the joints to play, come out, and so on.

Strong and long-term filling of internal organs (stomach, intestines, bladder). This causes dystrophy of the organ wall and glandular cells.

While changes in organ and tissue compression cause disruption of blood supply, long-term compression of organ or tissue causes tissue nutrition to deteriorate, leading to atrophy and even necrosis.

Strong organ dysfunction occurs when animals are rescued from being trapped underground, resulting in frequent traumatic shock-like disturbances in renal function.

Injuries occur in animals as a result of exposure to cold or firearms, thunder, and air waves. Falling from height or rupture of spleen and blood vessels of deep tissues and organs under the influence of thunder waves is observed fracture of bones without changing the skin lining system.

Traumatic injuries in farm animals (from the coldness of animal caregivers) are caused by the impact of equipment and tools used in various industries (machine mechanisms, washers, dots, etc.).

The following types of traumatic injuries are distinguished:

7. Closed injuries in which the integrity of the skin covering system is not compromised include: compression of the tissue (with tumor, wash, and puncture). Stretching, pulling, breaking, breaking bones, breaking, cracking under the influence of impenetrable weapons.
8. Injuries to the skin lining system, open changes include injury, destruction of the skin lining of the bone, tearing. Depending on the strength of the impact, torn, incised wounds are formed.

One of the characteristic or characteristic changes when an injury occurs is the sensation of pain. The formation of pain is associated with exposure of the organ to extra and introceptors, the breakdown of toxins, tissue breakdown, and the accumulation of toxins of microorganisms in the injured area.

In addition to local changes during injuries, general changes in some organs (heart, respiratory organs, endocrine and external organs) are observed with reflex dysfunction, accompanied by tachycardia, shortness of breath, hyperglycemia, increased blood pressure and other changes. characterized.

Injury to tissues on the surface of the body causes microorganisms to enter the internal parts of the body and cause them to become inflamed. Normally, pathogenic changes are limited due to the activity of protective flexibility mechanisms that protect our body when tissue injury occurs, only in some cases the process is exacerbated by insufficient resistance of the body's protective flexibility mechanisms, leading to the development of pneumonia and then sepsis.

The dead-necrotic tissue in the injured parts forms a large part, and the direct effect of the cause of the injury is due to the wash. The occurrence of such changes is associated with the restoration of tissue nutrition and metabolism by narrowing and rupture of blood vessels, disruption of the integrity of the innervation, and finally compression of the injured tissue and adjacent healthy tissue with exudate.

Long-term purulent wounds are a debilitating weight loss due to the body not healing. Injury weight loss leads to severe damage to internal organs (pleura, lungs, ribs, pelvis and stones). In such cases, the process of tissue regeneration is weakened, atrophy develops in the skin, subcutaneous tissue, transverse skeletal muscles, some internal organs: the animal's appetite is suffocated, sleep is disturbed, liver and intestinal function is impaired, some parts of the bed lie together. becomes lifeless.

Toxins produced by microbes during chronic injuries, the products of tissue breakdown, poison the body and cause it to lose weight. At the same time, many proteins in the pus are released from the body, which weakens the body's resistance to pathogens.

Traumatic shock is one of the most severe pathological conditions of the body.

During a period of traumatic shock, after a short period of agitation, a strong inhibition of the basic physiological functions of the body occurs. Characteristic changes during traumatic shock include acceleration of breathing and pulse, increase in blood pressure, increase in blood glucose and adrenaline. Subsequently, blood pressure decreases, the amount of blood circulating in the blood vessels decreases, body temperature decreases, reflex activity weakens, the animal becomes insensitive to environmental changes, pain sensitivity decreases, alkaline blood reserve and tissue oxygen consumption decrease. The excitability of the cerebral hemisphere cortex and vegetative centers, the formation of biopathy is weakened. A traumatic shock condition occurs after trauma or exposure to a traumatic agent (primary shock). Primary shock is caused by the reflex excitation of sensory nerve endings under the influence of traumatic factors. The peripheral nerves are irradiated to the subcortical parts, first causing excitation and then braking in the cortex. It weakens all the physiological functions of the body, in particular by lowering vascular tone, leading to a decrease in blood pressure. Many scientists explain the secondary development of shock as poisoning caused by the absorption of histamine-like substances into the body through the blood vessels in the crushed part of the tissues. This is supported by the following supporting information. When histamine and other biologically active substances are released into an animal's bloodstream, a secondary shock-like condition occurs, but histamine and peptone shock, although similar to this shock, do not resemble the shock that results from the injury itself. The formation of traumatic shock is accompanied by additional changes in the body, adverse factors (blood loss, fever or heat, hunger, fatigue), the width of the injured area (nerve columns), due to the abundance of receptors and many other factors. In the development of traumatic shock and subsequent restoration of impaired function occurs the influence of pituitary, adrenal hormones, nervous system and other organs.

The outcome of trauma depends on the type of organ, its vital importance. Death can occur if the heart, large diameter blood vessels, nerve centers, etc. are injured. The changes resulting from the effects of mechanical influences on the nervous system are severe and complex. When peripheral nerves are injured, the motor and sensory properties of organ systems change. Mechanical injury of the central nervous system causes severe functional changes in the body (the affected area depends on the degree of injury). Severe bruising, bullet and skull injuries, causing general bruising, can sometimes injure the brain, blocking blood vessels and the respiratory center. This results in cessation of breathing or paralysis of the heart.

Spinal cord injury paralyzes the leg and impairs the function of pelvic organs (urine, fecal excretion, etc.). Sometimes when a strong blow to the podcherevnoy (abdominal) part, the heartbeat weakens and even stops. Injuries to the heart and large blood vessels are dangerous for the body. When a heart is injured, death usually occurs from exposure to its neuromuscular apparatus, thrombus and blood flow to the heart cavities.

Rupture or injury to the artery of the hip, pelvis, and mesentery results in external and internal rupture, resulting in death. Rupture of the tissues in the chest causes air to enter the interstitial spaces and compress the lungs, leading to disruption of the reflex.

Disease-causing sound wavesdepending on the strength, frequency and duration of exposure to sound waves can have a detrimental effect on the body. Noisy mixtures of different strengths and heights have a detrimental effect on the body. Under the influence of these noises, strong agitation, fatigue, changes in the respiratory process, worsening of hearing, increased intracranial pressure and other pathological changes occur.

Accidental, sudden loud noise can damage the hearing aid: a long and strong generated sound wave can affect the activity of the central nervous system. Pathological changes in the body (metabolic disorders, changes in cell structure, accumulation of heat in the body, when the ultrasound is exposed to a sound that is too long and strong) an increase in glucose and cholesterol in the blood, a change in the shape and structure of the shaped elements of the blood i.e. deformation can cause protein coagulation and other changes).

The causes of internal disease often include the factors that contribute to the onset of the disease in the body. For example, as a result of working in mines, factories, and mines, toxins that enter the body in different ways are absorbed into the tissues, and the dust settles in the lung tissue,

causing various deficiencies in these tissues and causing disease. causes. Circulatory disorders are also among the internal causes of the disease. Changes in hereditary traits also cause disease under the influence of mutagenic causes.

Pavlov recommends studying the causes of IP disease in three groups:

1. All exogenous and endogenous causes are the first group of causes to which the body responds with an unconditional reflex.

2. The indifferent effects created by IPPavlov's work, that is, the influence of the causative agent, if supported by normal conditions, then the natural effect of this supporter is called by the disease itself. For example, if you take an apomorphine in a syringe, tie the dog to a machine, and then send the apomorphine to the dog every time it is supported by a light or a bell, then turning on the light bulb will cause illness and the dog will vomit. called syrotchis. The body of animals responds to the causes of this disease by producing conditioned reflexes.

3. Psychogenic causes have also been proven in experiments and are of great importance for human beings, that is, affecting the body by speaking, drawing, grieving, and writing harsh insults can also lead to diseases.

1.Pathogenesis is the study of the origin, mechanism of development, pathogenesis, course, and consequences of diseases.

Greek pathos-victim, genesis-formation. Diseases develop by different mechanisms when different pathological causes affect the body. To make the doctrine of pathogenesis easier to understand, it is distinguished that etiological causes affect 3 different types.

Type 1 causes diseases that affect all stages of development. For example, in acute poisonings, until the toxin is released from the body, it affects the development of the disease in the body, or a similar change occurs when an electric shock.

Type 2 causes serve as a driving force, developing the mechanism of the disease. For example, as a result of a single exposure to hot water, it acts as a starting force. The following substances are formed and poison the body, disrupt the permeability of blood vessels, create an acidic environment and create oxygen deficiency.

Type 3 etiological causes continue to affect themselves depending on the duration of disease development.

The basic structure of the mechanisms of disease development is that when various causes affect the body, there is a lack of oxygen in the body, that is, the metabolism changes, which disrupts the function of various organs and the mechanisms of disease begin to develop.

1. Corticoviceral doctrine is a two-way connection, ie a doctrine that explains that the nervous system is connected to all internal organs. The effect on the body is affected either by a conditioned or unconditioned reflex pathway and responds using unconditioned reflexes. The mechanism of disease development also depends on the reactivity properties of the organism. If reactivity is strongly developed, the disease may not develop. If the body is deficient in various micro and macronutrients, the nutrient content is incomplete, or the body is tired, the development of the disease can occur slowly.

2. Depending on the types of nervous system. If the animals fall into the fragile type, the disease develops more strongly.

3. Explains the development of the disease under the influence of stressors. When inadequate effects on the body are given to the pituitary and adrenal glands over a long period of time, they produce 3 different changes to the effects as they control the body’s reactivity.

1.The properties of tension The pituitary and adrenal glands produce a lot of hormones, adapt to stress by inadequate action, strong excitation, and produce a variety of hormones. If the hormone-producing function either increases or decreases, the body's function is impaired.

2. In the stage of resistance, the body is resistant to any pathogenic influences, because the hormones of the pituitary and adrenal glands increase the energy and plastic mobilization of the body. In the stage of resistance, when the body can not cope with the pathogenic force, the stage of general weakness, without exhaustion begins.

3. At the stage of general weakness, the body loses flexibility, immunological reactions, regeneration state decreases.

3. Examination of cell composition in animals and humans revealed that the development of pathological processes depends on chromosomes: for example, defects in the development of sex, ie secondary sexual characteristics, infertility and other changes. Males have one more sex chromosome and females have one less sex chromosome.

The role of constitution in pathogenesis. The disease arises from the encounter of disease-causing causes with the organism. Therefore, in addition to qualitative and quantitative changes in the pathogen, the characteristics of the animal organism are important in the origin of the disease. The individual reactivity of the organism takes the first place in the origin of diseases in the organism, because the effect of a certain pathogen on the organism of animals does not lead to the disease of all animals, but to some of them.

What is the constitution? Although there is still no complete answer to the question, constitution refers to the general morphological and physiological features of an organism, which are the product of long-term evolution from the interaction of the organism with the external environment, and these properties are stable. Due to these features, the reaction of the organism to the external environment is determined, comparing close species.

The constitution of agricultural animals means that it increases the resilience, resilience, disease resistance, flexibility and productivity of the farm and the environment. Thus, the constitution of farm animals means not only the morphological and physiological characteristics of the organism, but also the reactivity of the organism to the external environment, including the development of a response to the causes of the disease.

The whole organism can be afflicted with various diseases, and it is impossible to know in advance for what reasons they occur. It depends on external influences, hunger, poisoning, fatigue, exposure to cold and other causes that change resistance and their effects. Due to congenital malformations of the organs in some organisms, the influence of the above external causes causes the disease. In recent times, it has become common to study the constitution in two parts:

1. The constitution of the breath.

2. The constitution of digestion.

Importance of breed, sex and age in pathogenesis. Animal breeds play an important role in the origin of the disease, and Algerian sheep do not suffer from anthrax. Horses of the Budyonny breed are not susceptible to lung diseases. Caucasian mountain merinos do not suffer from pyrapylazmosis, but other breeds are highly susceptible to the cause of this disease. Depending on age, young animals suffer from diseases of the digestive organs, pneumonia, some infectious diseases. As the animals mature, many diseases become more resistant.

4. Restoration of body activity. Protective resilience mechanisms in the body that have the ability to restore impaired function under the influence of pathogenic influences, including excess energy generated in the body, surfaces, stored blood, chemicals and biochemicals. For example: under normal physiological conditions, 17-20% of the heart muscle, the respiratory surface of the lungs, the absorption surface of the intestine, 20-25% of the glomeruli of the kidneys, 12-15% of the liver, 10-15% of the blood vessels, 50 of hemoglobin -60% and nervous, endocrine systems are rarely used. Therefore, the organism adapts to any difficult conditions. For example: in bilateral pneumonia, dystrophy and fatty heart muscle, severe liver injury, removal of a single kidney, functions are also compensated when a large part of the stomach and intestines are cut, when a lot of blood is lost, when many capillaries become loose and clogged, and when nerves and endocrine glands are injured. The patient's kidney function is performed by a healthy kidney, and lymph nodes perform blood formation when the spleen is removed or diseased.

environment at different times. First of all, the general reactivity in the body, that is, the resistance to various toxins, and then the types of immunological reactivity developed. As organisms now

develop, the reactive function is performed by cells, which later develop a response using the humoral system and eventually the nervous system.

The properties of reactivity depend on the age of the animal, the nervous and humoral systems, the external environment and the general condition of the organism. For example, when the embryo develops in the mother's womb, it responds to the stimuli through the mother's body, ie through the placenta. When a baby is born, its reactivity is weak and responds only by a phagocytic reaction or by immune cells that pass through the mother’s blood. That is why young animals often get sick and die. Young animals are weakly adaptable to changes in ambient temperature, and their dyspepsia, salmonellosis, colibacillosis, rickets and other diseases are common. Reactivity in adult animals is manifested in the fight against microorganisms by antibodies, phagocytes and macrocytes that have accumulated in their bodies. As the body ages, its reactivity decreases. phagocytes, immune cells are reduced, and the incidence of disease increases with susceptibility to disease. As a result, tumors, hypertension increase, regeneration is weakened, and the body's reactivity is low, so they have severe infectious diseases.

Sirotin NN and other scientists note that the cerebral cortex of cold-blooded and young animals is poorly developed and is less sensitive to strong toxins (histamine, diphtheria, stolbyank toxin). During anabiosis, animals do not develop sensitivity to very strong toxins and infectious agents (plague, tuleremia, anthrax, tuberculosis).

Due to reactivity, the body responds to disease-causing causes, and the sensitivity of different individuals to infectious agents varies. Such cases can be observed in various pathological processes. For example, when an animal with a high reactivity burns, it recovers quickly and an animal with a low reactivity recovers later. The reactivity of the animal organism depends on the metabolism, the immunological properties of the organism, the functional state of the animal organism, the vascular reaction and chronaxy to the excitability of the nervous system.

Concepts of reactivity R Virkhov's cellular theory developed at a time when the theory of cells gave a misunderstanding of the general reactivity properties of individual cells, tissues and organs, ie the fact that pathological processes take place only in cells. 'did not notice. In contrast, IIMechinkov in his many years of observations shows that the reactivity of organisms at different stages of evolutionary development is also formed under the influence of disease-causing factors of the external environment. As organisms become more complex and the nervous system develops, the body's reactivity to inflammatory agents becomes more complex. For example: cold-blooded frogs, inflammation in fish, develops very poorly in warm-blooded animals. Even when these properties were observed by NNSirotinin sending proteins to the body, it was observed that the body of cold-blooded animals produced very weak responses. Gradually, as a result of the development of the nervous system of the organism, the reactivity or sensitivity of the organism to many toxins, formed a changing response.

Reactivity is a characteristic feature of all animals, and in the field of reactivity IIMechnikov, VVPashutin, AABogomolets, NNSirotinins have done a lot of research. In their laboratories, these scientists studied reactivity by linking it to metabolism and other areas. IPPavlov and IMSechenov confirmed that the nervous system plays a leading role in the development of reactivity. In the IPPavlov laboratory, MKPetrova et al observed that the reactivity of animals was impaired by inhibiting the cerebral cortex by giving bromine preparations.

The importance of the types of nervous system in reactivity is also great. To study the importance of types of nervous system in reactivity, they took two groups of dogs:

1. The group includes dogs with a weak nervous system.
2. Dogs with a strong type nervous system in the group.

In animals of both groups, when exposed to strong toxins, cyanic acid, bacterial toxins, dogs with a weak nervous system became ill due to weak barrier properties of the organism, in animals with a strong nervous system AMMonaenkov and others explain that the diseases have not developed because their barriers are strong, their neutralizing properties are high.

In the IPPavlov laboratory, pigeons became infected with anthrax when a certain part of their brain was removed.

Academician ADSperansky observed that when dogs opened their brains and placed a ball in the midbrain, mechanical effects resulted in ulcers in the lungs and digestive systems, weakening their resistance to infection. He drew attention to the fact that the traces of the nervous system in the origin and development of pathological processes, that is, pathological processes in the nervous system, even after their recovery, retain their complications for a long time. In many experiments, that is, when animals are exposed to different stimuli after treatment of the disease, the effect of these stimuli spreads to the entire nervous system, leaving traces of old disease in the affected area. observed that it had survived and accumulated, leading to the onset of the disease. This feature of the nervous system is called AA

Reactivity is also affected by the autonomic nervous system. Reactivity changes when the function of the autonomic nervous system increases or slows down. Excitation of the sympathetic nervous system enhances phagocytosis, enhances metabolism, and increases reactivity. Excitation of the parasympathetic nervous system increases the production of antibodies, produces short-term leukocytosis, followed by leukopenia, exposure to certain toxins (phenol, aniline, etc.), lymph nodes, liver barrier - barrier properties increases.

Reflexivity changes reflexively from the pathological effects of heat and cold. For example, as a result of colds, people get the flu, pneumonia, that is, the body's reactivity decreases. In experiments, it is possible to cool the body of chickens, reduce their reactivity and lead to anthrax, or to heat the body of guinea pigs and reduce their sensitivity to proteins.

Toxic substances, alcohol, carbon monoxide, lead, mercury, cyanic acid weaken the internal braking. Pigeons were poisoned with alcohol, which reduced their reactivity to anthrax, or when people consumed alcohol for a long time, they observed a decrease in the general reactivity of the organism, and xko.

While ultraviolet light from light energy increases the stability of an organism to a certain extent, it weakens the stability of an organism to a certain extent. X-rays and gamma rays have a detrimental effect on the body's reactivity. The reactivity of the organism also decreases under the influence of mechanical influences. Thus, the role of nervous endocrine systems in the formation of reactivity of the organism is important, but different effects of the external environment affect the activity of various organ systems of the organism, affecting their metabolism, neurohumoral control mechanisms.

There are several classifications of reactivity, and most scientists classify the organism according to its state of health or disease:

1. Physiological reactivity.
2. Pathological reactivity.

Physiological and pathological reactivity can be individual or individual, as well as group. Individual or specific reactivity depends on hereditary traits and can be passed down from generation to generation. Physiological reactivity develops the body's response to natural (adequate) influences, while pathological reactivity develops the body's response to the causes of the disease. Allergic and immunological types of pathological reactivity are distinguished, and the manifestation of these types of reactivity is formed in relation to foreign proteins, microbes and their toxins. (Allergy, Anaphylaxis, Immunity). Typically, biological or species reactivity is differentiated and is specific to animals belonging to a particular species, ranging from seasonal changes in animals to: seasonal sleep, migration of animals from one place to another, animals are not exposed to microorganisms, ie chickens are not infected with anthrax, specific reactivity is a characteristic feature of a particular individual, it depends on the constitution, sex, age, nutrition and storage characteristics, newborn reactivity in animals is low, reactivity is well developed during sexual maturation, phagocytosis and the formation of immunoassays are well demonstrated, in older animals the reactivity of the organism is low due to the weakening of their barrier properties. Hence, the specific reactivity is that during the period of complete vaccination of animals, their reactivity is formed differently, with strong antibodies in some and weak antibodies in others.

The resistance of an organism, as the Latin resisteo (resist, resist), is the resistance of an organism to physical, chemical, and biological causes of disease. This means that the body's resistance is understood to be resistance to many different causes.

During phylogenetic development, when the resistance of the organism changes and invertebrates are resistant to bacterial toxins, the susceptibility of warm-blooded animals is high. Resistance is associated with the functioning of organ systems, depending on the type, sex, age, constitution, anatomical and physiological characteristics of the animal, the level of development of the organism, the development of the RES and lymphoid system. In the early stages of ontogenetic development of animals, resistance to various harmful agents is high (partial pressure reduction, some bacterial toxins), resistance to sexual development is well developed, and resistance decreases with age.

Resistance:

1. Natural-born,
2. Acquired-generated species are different.

Congenital resistance is passed down from generation to generation. For example, Algerian sheep are more resistant to anthrax than European sheep.

Acquired generated resistance depends on the individual characteristics of the organism and is formed when immunized against infectious diseases. Resistance is formed depending on the activity of the pituitary, adrenal glands, colon, gonads. Barrier properties of the organism, biologically active substances in the blood and phagocytosis play a key role in resistance. When the body is tired, very productive, living conditions are poor, resistance is weakened, and conditions are created for the development of diseases.

2. Animals and humans live in a world of microorganisms. Immunity, on the other hand, as a controller, rigorously tests agents for various causes that have entered the body.

Immunity - Latin Immunitas - means purification, deliverance. Immunity is the ability of an organism to be exposed to antigenic pathogens, their products and hereditary foreign substances, or to be resistant to various disease-causing microorganisms, viruses and their products, as well as to non-infectious modes. , forms a special view of the overall resistance.

Immunity is divided into two depending on the nature of the mechanism and causes that cause it:

1. Congenital immunity or hereditary immunity from generation to generation.
5. Acquired immunity

Congenital or natural species-specific immunity is a specific resistance of an organism that is passed from generation to generation and is specific to a species, breed, and population. For example, in cattle, horses are resistant to microorganisms that cause croupous inflammation of the lungs, and animals are highly resistant to human diarrhea. Dogs are not infected with pleural pneumonia in cattle. Cattle do not suffer from horse manure, infectious (infectious) anemia.

Inter-species immunity is also different, Algerian sheep are resistant to anthrax, Breton sheep are resistant to smallpox, light-bodied pigs are resistant to yellow fever, Mongolian cattle are resistant to plague, and other animals of this type are infected with the above diseases. Congenital immunity is formed not only against an infectious agent, but also against their toxins. The barrier properties of animals with innate immunity are strong and do not transmit microorganisms into the body or prevent the growth of microorganisms by altering the environment.

These organisms have high phagocytic activity and bactericidal properties in fluids, which prevents the development of microorganisms and forms specific immune cells against these microorganisms.

Acquired immunity is formed during the ontogenetic development of certain microorganisms in the body of animals. Acquired immunity is created by natural and artificial means. For example, naturally acquired immunity is formed after recovery from mango, smallpox, proteinuria and other diseases. Artificial active immunity is created by vaccinating animals against various infectious diseases. Hence, acquired immunity is generated by natural and artificial means.

Artificial immunity is studied as active and passive immunity. Passive immunity is formed when hyperimmune serums are sent, through the passage of immunoassays through milk, through the placenta. Due to passive immunity, the body's resistance is maintained for some time. RES plays a leading role in the formation of immunity, and the formation and formation of immunity is controlled by the nervous system.

During the period of immunity against infectious diseases, if the organism is completely cleansed of infectious agents, sterile immunity is formed and the organism is provided with sterility to this antigen.

If the immunity formed in the body does not maintain complete sterility, and the antigen is retained in the body, it is called nosteril immunity, which is characteristic of tuberculosis and brucellosis.

Immunity can be formed not only against microorganisms themselves, but also against their toxins, which is called antitoxic immunity and is observed during exotoxin-producing microorganisms: tetanus, botulism, gas gangrene and other infections. Hence, toxins act as antigens in this process.

In addition, the body has special organs and factors that fight microbes and foreign substances, which are called barrier properties of the organism. The barrier-barrier properties of the organism are studied as external and internal barriers.

External barriers of the body include the skin and its products (accumulations), mucous membranes in various parts, the oscillating epithelium of the respiratory tract, microorganisms of the digestive system and hydrochloric acid.

The body's internal barriers include a number of cellular and humoral factors, various histiocytes, reticular cells, plasma cells, epithelial cells of the inner wall of blood vessels, and leukocytes. RES cells, which are involved in protecting the body, are active, they absorb microbes and other particles that enter the body, they are very rich in RES in the lymph nodes, spleen, liver, lungs, kidneys, meninges, blood-forming organs, skin . This means that RES is present to one degree or another in various organs of the body, and phagocytic activity is much higher in leukocytes, including neutrophils. In his long-term observations, IIMechnikov argued that the process of phagocytosis plays an important role in the formation of immunological features. microbes and their toxins, cellular elements, tissue breakdown products, other particles are digested in cells. Phagocytosis is the process by which particles are trapped in a cell and then digested. Phagocytosis is common in nature, with feeding and protection of single and multicellular simple animals occurring in a single cell, while in highly developed animals these systems are isolated and protected by specific mesenchymal cells (blood leukocytes, lymph nodes, red blood cells). bone marrow, spleen, liver, connective tissue histiocytes) - by phagocytes. Studies have shown that there is a direct link between the process of phagocytosis and the resistance of the organism. increased phagocytosis indicates a weakened immunity in the body. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. The formation of immune cells depends not only on the activity of cells, but also on the action of body fluids. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or being vaccinated, immune cells are formed in the blood and other fluids, neutralizing certain microorganisms and toxins. Immune cells are formed as a result of the transmission of antigens in the fluids of the animal's body, and are substances that selectively react with them. Immune cells are substances close to gamma globulins in the blood due to their chemical composition. The following antibodies are distinguished depending on their reactions with antigens. As a result of the animal recovering from the disease or

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1. Antitoxins and antifenzymes, immune cells that inactivate by binding toxins and enzymes.

2. Agglutinin and persipitins, antibodies that change the colloidal chemical structure of microorganisms, immobilize them, bind them to the sediment.

3. Cytolysins or cytotoxins - antibodies that break down cells under the influence of enzymatic complement substances.

4.Opsonins and bacteriotropins - change the appearance of microorganisms, facilitating phagocytosis.

If antibodies are formed under the influence of antigens, what are the antigens themselves?

Antigens are substances that enhance the formation of immune bodies and react selectively with them. These include microbes, toxins, erythrocytes and serum of other animals, as well as high-molecular compounds.

There are two types of antigens.

1. Full value antigens.

2. Incomplete antigens - haptens.

Complete antigens include complete proteins, ie serum, various proteins, microorganism toxins and filtrate colonies. Antigens have specific properties that react with the antibodies they produce.

Incomplete antigens, ie haptens, cannot enter the body to form antibodies and only bind to the protein molecule to achieve antigenic properties.

Antigens must be administered parenterally to the body to form immune cells. Antigens are exogenous and endogenous substances that are foreign to the body. The body’s own proteins also sometimes exhibit antigenic properties. To do this, the body's proteins meet with the infectious agent, toxins, and form an autoantigen. In order to form immune cells against antigens, the antigen remains in the body for a certain period of time, is captured in the liver, spleen, lymph nodes and stored in the blood for 2-3 weeks. Immunological reactivity is formed not only from the encounter of macro and micro organisms, but also from other types of individuals and even in the same organism itself when tumors grow, become inflamed and in other cases have antigenic properties against their own organism. In all cases, there are antigen and antibody reactions and phagocytosis between body tissues and other tissues. The tissue formed during embryonic development serves as an antigen for older tissues. Tissue does not fit the transplanted tissue or organ due to the immune barrier property of these organisms when transplanting organs into one species or individual, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by. when organs are transplanted to a species or individual, they do not fit the transplanted tissue or organ due to the immune barrier property of these organisms, which is called immunological tolerance. To ensure the growth of the transplanted tissue, it is necessary to eliminate tissue incompatibility. Problems of tissue incompatibility 1971 Lopukhin YU.M. studied by.

Decreased or complete loss of antibody production as a result of exposure of antigens to the body is called immunological tolerance or non-response. This condition is caused by antigen transmission during the embryonic period or after the animal is born. In older animals, immunological tolerance can

be established by transferring large amounts of antigen or exposing them to X-rays. Immunological tolerance is characterized by the loss of these antigens of their antigenic properties, which is observed when transplanted into other animal tissues, and the transplant grows well. It is currently used in blood transplants to remove tissue barriers from immunological

Inflammation is the most common, most complex pathological process known since ancient times, and in ancient times all diseases accompanied by a rise in local temperature were called inflammation. Inflammation is a typical pathological change (disruption of tissue function and changes in structure) that is common in various diseases, as well as the activation of the body's protective resilience properties and the restoration of impaired function. Although inflammation in this area delays the organism as a process with protective properties against the effects, the mechanism of its development, the formation of symptoms depends on the state of the organism, the activity of neuro-humoral systems. For example: Inflammation of the skin can be caused by affecting some endocrine glands of the gillpotalamus or peripheral nerves. Glandular is a local manifestation of the general reactivity of the organism, the degree of reactivity of the organism depends on the course of inflammation and, conversely, on the reactivity of the organism to inflammation, neurohumoral control, thermoregulation and other mechanisms. All substances that cause inflammation are called phylogenetic substances, and we study them in two groups, namely, exogenous and endogenous substances. Inflammation occurs under the influence of phylogenetic substances, and the name of the inflamed organ or tissue is read by adding the suffix "IT", "IYA". For example. Inflammation of the liver is called hepatitis, inflammation of the kidneys is called nephritis, inflammation of the lungs is called pneumonia, and xzo

Inflammation is caused by mechanical, physical, chemical, and biological causes of external disease, and often the contribution of microorganisms and viruses is important in causing inflammation.

Sometimes inflammation can also be generated under the influence of conditioned indeferent stimuli.

Ichki yalligg‘lanish chaqiruvchi sabablarga nekrotik to‘qima, infarkt, gematoma, turli qismlarda to‘plangan tuzlar kiradi. Yallig‘lanish chaqiruvchi sabab, ko‘pincha yallig‘lanish reaksiyalarini hosil bo‘lish intensivligini belgilab beradi: Masalan. Rengen nuri, zaharli modda, mexanik jarohatlar, kuyish,sovuq urish va boshqalar oldin to‘qimalarni parchalab, keyin shu joyda fiziologik aktiv moddalar to‘planib, ular ishtirokida yallig‘lanish jarayonlari ro‘yobga chiqa boshlaydi. Surunkali kechuvchi kasalliklarda , kasallik chaqiruvchi sababni, begona tasirotchini uzoq vaqt tasiridan, yoki ximiyaviy qo‘zg‘atuvchining tasiridan proliferativ jarayonlar kuchayaadi.

Yallig‘lanishni kechishi kasallik chaqiruvchi sabab tushgan joyga bog‘liq bo‘lib, amyoba jigarga tushib absets chaqirsa, ichaklarda yarali yallig‘lanish chaqiradi. Masalan. Stafilokok, streptokoklarni yiringli infeksion jarayon hosil qilish aniq, lekin skipidarlarni teritagiga yoki muskullar orasiga yuborib yiringli yallig‘lanish chaqirish mumkin. Shunday qilib yallig‘lanishni xususiyati, uni hosil bo‘lish tezligini qo‘zg‘atuvchi xususiyatiga hamda yallig‘lanish kechayotgan muhitga bog‘liq ekan. Yallig‘lanishning tashqi mahalliy belgilari Sels va Galenlar tomonidan sharxlangan bo‘lib: qizarish-chivoch, shish tishoch, harorat ko‘tarilishi-saloch og‘riq - doloch, funksiyani buzilishi fipstto laesa deyiladi. Har qanday yallig‘lanish ham bir qancha asosiy bir-biri bilan bog‘liq jarayon bilan kechadi: altteratsiya-to‘qimalardagi distrofik o‘zgarishlar-to‘qimalarning yallig‘lanish chaqiruvchi agent ta’sirida qitiqlanishi va parchalanishi, maxalliy qon aylanishini buzilishi-ekssudatsiya va emigratsiya, fagotsitoz hamda proliferativ o‘zgarishlar. Yallig‘lanish chaqiruvchi agent to‘qimalarni qitiqlashi, parchalashi, ulardagi moddalar almashinuvini, tuzilish va funksiyani buzilishiga sabab bo‘ladi. Distrofik o‘zgarishlar yallig‘lanish chaqiruvchi sabab ta’sir etgan vaqtdan hosil bo‘lib, kam chegaralangan bo‘ladi. Keyinchalik ta’sirotchining ta’siri kuchayishi bilan yallig‘lanish kuchayadi, to‘qimalarda moddalar almashinuvi kuchayadi, qon aylanishi buzilib, distrofik o‘zgarish kuchayadi. Kasallik chaqiruvchi sabab organizmga tushib birinchi navbatda retseptorlarga tasir qiladi. Agar ta’sirotchi kuchi etarli bo‘lsa nerv oxirlarida parabioz xolatini hosil qiladi.

At the onset of inflammation, the tissue bends the cells, fat granules appear, protein and fat dystrophies are observed, then the cell structure is disrupted and even severely damaged and dies. Necrabiotic processes during inflammation are caused by the bending and melting of collagen and

elastic fibers of tissue interstitials. In inflammation, necrobiotic processes are formed when tissue burns, under the influence of strong acids and alkalis, sometimes in relation to weak influences from increased sensitivity of the organism. There is a certain association between them and dystrophic changes in the body, and sometimes due to the injured part there is a compensatory restoration of their functions, despite the presence of destructive changes in the salivary glands, stomach and other organs. ladi. The development of destructive changes during the period of inflammation depends on the organ, and such changes can be observed in injuries of parinchyomous organs. The degree of dystrophic changes depends on the strength and nature of the pathogen, where the pathogen enters, the nature of the injured organ or tissue, and the reactivity of the organism. Physiologically active substances formed as a result of dystrophic changes in the source of inflammation and metabolic disorders are absorbed into the blood, reducing vascular tone, causing emigration, phagocytosis and proliferation of cellular elements. These biologically active substances include histamine and histamine-like substances, acetylcholine, ATF, creatine phosphoric acid and other necrogorms that dilate blood vessels and enhance proliferation, trephon tissue proteases and cathepsins. Thus, the strong passage of alternative, proliferative and exudative processes during the inflammatory period leads to tissue bending and the development of dystrophic changes that complicate blood circulation.

Metabolism at the source of inflammation undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. Metabolism between the inflamed part and healthy tissue is enhanced. The increase in metabolism is due to easily oxidized carbohydrates, which form many weak acids as they take place in an oxygen-free environment. The breakdown of carbohydrates in the anaerobic phase increases due to leukocytes released during emigration, but these changes can be seen in the oxygen consumed and the carbon dioxide excreted before the breakdown is broken down into the final product. During this process, the respiration rate decreases as more carbonic acid is released.

During inflammation, the metabolism undergoes quantitative and qualitative changes, strong disintegrations are formed in the inflammatory center, and metabolic and oxidative processes are reduced. The metabolism between the inflamed part and the healthy tissue becomes enhanced. Metabolism will be enhanced. Lactic acids are formed due to the fact that the increase in metabolism is due to easily oxidized carbohydrates, which take place in an oxygen-free environment. Due to the leukocytes released during emigration, the breakdown of carbohydrates in the anaerobic phase increases, but without decomposition to the final product, these changes can be determined by the oxygen consumed and the carbonic acid released. In this process, the respiration rate decreases as more carbonic acid is released.

Fats and proteins also form ketone bodies, albumin-peptones, which are not completely broken down in the center of inflammation. Excessive increase in carbohydrate protein and fat metabolism, complete oxidation of milk at the source of inflammation, pyruvic acid, fatty acids lead to an increase in ketone bodies, amino acids and peptones, and acidosis develops. Acedosis is compensated first at the expense of the body's alkaline reserve, then it is not compensated.

(N hyperonia is formed). Depending on the nature of the process taking place in the tissue, the change in the environment of the tissue becomes 7.1-6.6, ie weakly alkaline, in the acute process 6.5-5.4 in the acute flow process. Increased acidosis increases the dissociation of salts, changes the electrolyte ratio, increases the amount of potassium, increases metabolism, breaks down large molecules into small molecules, increases the amount of ions, increases the osmotic pressure at the source of inflammation. Similarly, oncotic pressure increases. Osmotic and oncotic pressure decrease as you move away from the source of inflammation. Thus, changes in the quality and quantity of tissues during inflammation cause physicochemical changes in tissues, including: hyperionia, hyperosmia and hyperonkia. The causative agent causes a short-term narrowing of the blood vessels by reflex action on the blood vessels and then dilation of the blood vessels.

The slowing of blood flow in the blood vessels is due to the following reasons:

1. Paralysis of the vascular neuromuscular apparatus causes loss of vascular tone.
2. Causes excessive dilation of the vascular surface.
3. It causes the blood to thicken and become sticky.

4. Slows down blood flow as a result of cutting blood vessels with fluids in the surrounding tissues.

5. Due to the adhesion of leukocytes to the inner wall of blood vessels, the unevenness of the inner surface of blood vessels is formed, and sometimes clogging with thrombi leads to a slowing of blood flow.

The vascular response at the source of inflammation varies under the influence of various pathogens. For example: vasoconstrictor (adrenaline caffeine, etc.) and vasoconstrictor sympathetic nerve effect. Slowing of blood circulation changes until complete cessation of blood flow in the arteries, leading to changes similar to thrombosis and hemorrhage. Disruption of blood circulation at the source of inflammation worsens metabolism, disrupts the nutrition of cells in the inflammatory center, and these changes themselves lead to increased inflammation.

Dilation of blood vessels and slowing of blood flow increase the permeability of blood vessels, resulting in leakage of shaped elements with liquid parts of the blood, and this process is called exudation. The fluid released is called exudate. The exudate differs from the transudate in the presence of 2-4 times the protein, shaped elements, local tissue elements, tissue breakdown products, some enzymes and other products. The process of exudation depends on several factors, the main of which are capillary permeability, high blood pressure in the vessels, osmotic and oncotic pressure at the source of inflammation.

Capillary permeability depends on the physiologically active substances histamine, bradykinin, serotonin, as well as potassium and hydrogen ions accumulated at the source of inflammation, which ions swell the blood vessel wall, dilute colloidal substances and disrupt vascular nutrition.

Healthy capillaries pass water and crystolloids, increasing permeability from colloidal substances to proteins primarily albumins (low molecular weight) substances.

In inflammation, more blood flows to the source of inflammation, weakening the bleeding and increasing the pressure in the blood vessels, which allows more fluid to leak out of the blood vessels. Such strong exudation lowers blood pressure in the blood vessels and weakens blood flow. Exudation is also affected by the osmotic and oncotic pressure at the source of inflammation.

During exudation, water, salt, protein, or cell-free products are released from the blood vessels, and then leukocytes are released from the blood vessels into the tissues, called leukocyte emigration. During leukocyte emigration, the localization of leukocytes along the walls of blood vessels occurs, resulting in the redistribution of blood-forming elements, which is associated with slowing of blood flow. In normal life processes, the blood is characterized by the placement of two layers of thin, plasma at the edges of the blood vessels and shaped elements moving in the center, the specific gravity of erythrocytes is heavy between the blood vessels, leukocytes move lightly on the periphery.

As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cytoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Remaining leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that die in large numbers in high osmotic pressure and atsedosis.

Monocytes show their resistance even at pH 5.5. While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose

fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. AS Shklyarevisky, a proponent of the mechanical theory that explains the location of leukocytes along blood vessels, explains that leukocytes are pushed aside by other shaped elements because of their light weight.

Proponents of the second type of this theory explain that leukocyte emigration is a passive process in which leukocytes flow out of the general fluid flow and remain outside the blood vessels. If this is the case, then why do neutrophils come out in one case, lymphocytes and monocytes in the other. Thus, without mechanical factors playing a major role in the location of leukocytes along the vessel wall, this theory cannot explain the formation of these processes. Because the location of leukocytes along the walls of blood vessels is a complex biological process, the active processes in which leukocytes approach the wall of blood vessels, push it out of the blood vessels and participate in phagocytosis.

According to IIMechnekov's biological theory, leukocyte emigration is called a positive hemataxis feature. Positive chemotaxis properties include staphylococcus, streptococcus and other substances that are formed as a result of their activity, as well as products of nucleic metabolism, some globulins, liver and kidney proteins, meat peptone broth, some medicinal substances.

The repulsion of leukocytes from these chemicals is called negative chimataxis, and the negative chymataxis property is characteristic of quinine, chlorochrome, benzene, alcohols.

The development of physkaloid chemistry leads to the emergence of a new direction that explains the emigration of leukocytes, i.e. leukocyte emigration is associated with physicochemical changes in tissues.

Increased metabolism in the inflammatory center results in the formation of completely unoxidized substances, leading to an increase in N ions. Thus, due to different charges, negatively charged leukocytes move towards the center of positively charged inflammation. Leukocyte emigration is also caused by the continuous release of fluid from the blood vessels into the inflamed parts. Energy processes in leukocytes also play an important role in leukocyte emigration. On the side of leukocytes facing the source of inflammation, the protoplasm melts to form pseudopodia and amoeba-like action due to the energy generated during the metabolism of leukocytes. Emigrated leukocytes partially die under the influence of the environment at the source of inflammation, while others are actively involved in the process of phagocytosis. While the process of phagocytosis is influenced by the tissue environment and physiologically active substances, the acidic environment and alkaline environment inhibit the process of phagocytosis, while the normalization of the environment ensures the normal course of phagocytosis. Thus, leukocyte emigration is an active biological process in which mechanical and physicochemical changes play an important role.

Proliferatsiya jarayoni yallig‘lanishning barcha davrlarida hosil bo‘lib, alteratsiya kechayotgan davrda kam miqdorda bo‘lsada to‘qima hujayralari ko‘payib o‘zining eng kuchli ko‘payish davriga yallig‘lanishning oxirgi davrlarida etiladi. To‘qima hujayralarni ko‘payishini kuchayishini parchalangan mahsulotlar va to‘qimalarda moddalar almashinuvini buzilishidan hosil bo‘lgan moddalar hamda patogen agentining o‘zining ta’siridan hosil bo‘ladi. To‘qima va hujayralarni tiklanishida yallig‘lanish markazidagi RES hujayralari ya’ni qon tomirlar endoteliyasi, advintitsiyasi, fibroblastlar, gistiositlar, fibrotsitlar va qon tomirlari orqali emigratsiyalangan monotsitlar ishtirok etadi. Hujayra elementlari harakatchan bo‘lib fagotsitoz jarayonida ishtirok etadi. Bularni makrafaglar deyilib, ularga Ranve plazmatsitlari, poliblastlar, Maksmovning tinchlikdagi adashgan hujayralari, turli gistiotsitlar kiradi. Yallig‘lanish manbaida hosil qiluvchi plazmatik hujayralarni parchalanish mahsulotlarini fermentativ yo‘l bilan emiradi.

After the process of proliferation, the process of regeneration develops, the growth of connective tissue, blood vessels, connective tissue proliferates and glandular cells are regenerated. Young fast-growing connective tissue is rich in blood vessels and is called granulation tissue. The connective tissue grows from the periphery to the center, creating a barrier between healthy tissue and inflamed tissue, preventing microorganisms from spreading from the source of inflammation to the body. Upon completion of the inflammation, interstitial fibrous substances are formed in the granulated tissue, the

blood vessels shrink, the young mesenchymal cells stop growing, and eventually a dense connective tissue chandelier is formed. The resulting scars cause various dysfunctions, including esophagus, stomach, if it is formed in the urinary tract, it causes them to narrow, the mobility of the joints changes, and so on. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. If small parts are injured, the tissue is regenerated at the expense of special cells and no scars are formed. Full recovery is observed in the skin, mucous membranes, and the muscles recover a little slower. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little sluggishly. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed. the muscles recover a little sluggishly. The importance of hyperemia at the source of inflammation in the proliferative process is important. After inflammation, the structure and function of the tissue is completely restored to its original state. In this case, harmful agents and metabolites are neutralized and absorbed. If there are any defects, the functional capacity will decrease. If the process is chronic, a large area or organ is damaged, connective tissue grows, scars appear, function is impaired, and sometimes irreparable wounds are formed.

Yallig‘lanish morfologik va etiologik belgilariga qarab bir necha turlarga bo‘linadi. Yallig‘lanishning morfologik belgisiga karab alterativ, ekssudativ va proliferativ xillarga bo‘linadi.

Alterativ yallig‘lanish davrida to‘qimalarda distrofik va nekrobiotik jarayonlar, ekssudatsiya va proliferatsiya jarayonlariga nisbatan kuchli rivojlanib bu turdagi yallig‘lanishlarni turli zaharli moddalardan bakteriya toksinlari, ba’zi bir tuzlar ta’sirida parenximotoz organlardan buyrakda, jigarda, yurak va kam xollarda miyada uchraydi.

Ekssudativ yallig‘lanishda ekssudatsiya va emigratsiya jarayonlari boshqa jarayonlardan ustun turib, ekssudat turiga bog‘liq holda serroz-zardobli, kataral-shilliqli, fibrinli, yiringli, ixoroz yallig‘lanishlar farq qilinadi.

Seroz yallig‘lanishlarda suyuqlik tiniq, sarg‘imtir rangli, solishtirma og‘irligi 1,018-1,-20 tarkibida 5-6% oqsil va kam miqdorda shaklli elementlar saqlaydi. Qon tomirlar reaksiyasi to‘liq rivojlanmay to‘qima kam parchalanib ekssudat tez so‘rilib faqat plevra va qorin bo‘shlig‘ini yallig‘lanishi bir muncha qiyin kechadi.

Catarrhal inflammation is a mixture of serum and mucous substances, which is more pronounced at the level of the mucous membranes, and leukocytes are less in the exudate. In fibrinous inflammation, the exudate is high in fibrin, which indicates an increase in vascular permeability. As a

result, in addition to albumin and globulins, fibrinogen leaks into the interstitial fluid, forming fibrin fibers and membranes, which coagulate. Diphtheria is when the fibrin sits flat between the tissue and on the surface, moves hard on the surface of the organ, and forms a wound.

During inflammation, krupoz inflammation is when fibrin sticks to the surface of the tissue and between them and moves easily without forming a wound.

Purulent inflammation occurs in all parts of the body, with the accumulation of pus in the inflamed parts. This fluid contains a large number of leukocytes, tissue fragments with a high specific gravity. Purulent exudates fill the space in the interstitial space and form an abscess or abscess, inflammation of the sebaceous glands and hair follicles-boils, inflammation of a group of fat and wool bulbs is called carbuncle.

When putrefactive bacteria enter the inflamed parts and dissolve the tissue, the ulcer is called dissolved inflammation and is well manifested in alteration processes.

In hemorrhagic inflammation, the exudate becomes red due to the retention of erythrocytes. Vascular permeability results from acute and severe infectious diseases and poisonings.

In proliferative inflammation, cell proliferation increases oncotic pressures above other processes.

During exudation, water, salt, protein, or cell-free products are released from the blood vessels, and then leukocytes are released from the blood vessels into the tissues, called leukocyte emigration. During leukocyte emigration, the localization of leukocytes along the vascular walls occurs, resulting in the redistribution of mine-shaped elements, which is associated with slowing of blood flow. In normal life processes, the blood is characterized by the placement of two layers of thin, plasma at the edges of the blood vessels and shaped elements moving in the center, the specific gravity of erythrocytes is heavy between the blood vessels, leukocytes move lightly on the periphery.

As blood flow slows, light leukocytes accumulate at the edge of the blood vessel, collide, and move to be absorbed along the vascular wall. They then cling to the blood vessels in groups. This accumulation of white blood cells in the inner wall of the blood vessels is called the placement of leukocytes along the blood vessels. As a result of the location of leukocytes along the walls of blood vessels, they change their circular structure, forming a thin protoplasmic tumor-pseudopodia, piercing the blood vessels and forming a fold on the outside. This rash gradually enlarges and the leukocyte cetoplasm is deposited, resulting in leukocyte emigration outside the blood vessels. The emigrated leukocyte moves amoebae through the tissue interstitial spaces and passes to the center of inflammation, and II Mechnikov found that bacteria, dead tissue, carry out the process of phagocytosis against foreign particles. Some leukocytes die under the influence of intermediates formed as a result of metabolic disorders at the source of inflammation, forming many proteases, lipases, catalase nucleases and other enzymes, breaking down tissue fragments, bacteria, neutralizing harmful substances. Intact leukocytes either enter the bloodstream with interstitial fluids or participate in the recovery process that takes place there. Depending on the type and period of inflammation, different leukocytes are released at different times, usually neutrophils, then lymphocytes, and monocytes at the end of inflammation. Neutrophils are highly resistant leukocytes that degrade in large acidic environments and under osmotic pressure

Neutrophils exhibit their resistance at pH 5.5.

While neutrophils enter migrophages and phagocytose pus-producing microorganisms, lymphocytes and monocyte-pharyngeal phagocytose fragmented cell fragments. The location of leukocytes along the walls of blood vessels and their exit from blood vessels is explained on the basis of three different theories: mechanical, biological and physical-chemical theories. According to AS Shklyarevisky, one of the proponents of the mechanical theory explaining the location of leukocytes in the blood vessels, the specific gravity of leukocytes is light, including inflammation of the connective tissue at the site of inflammation, sepsis, actinomycosis, proteinuria and other diseases. 'sib, granuloma is formed, resulting in the passage of toxins and microorganisms from the inflamed area to healthy tissue. Biologically active substances released from leukocytes and other cells, as well as changes in osmotic and oncotic pressure in inflamed parts play an important role in the occurrence of proliferative processes. These modes tickle the receptors in the injured parts by the reflex pathway.

Depending on the immunobiological reactivity of the organism, normergic, hyperergic and hypergic inflammations are distinguished.

Normergic inflammation is caused by the primary exposure of microbes or toxins to organisms that are not sensitized and have normal immune properties. Hyperergic inflammation occurs after repeated exposure of the body to the cause of the disease. This inflammation is accompanied by a strong acute flow, alternating and exudative processes. Changes in this period do not depend on the strength of the antigen, but rather on the increase in the sensitivity of the organism. Alterative changes in hyperergic inflammation begin with fibrin bending and necrosis of halogenated and smooth muscle fibers. The fibrin in the exudate is hemorrhagic because it is a mixed serum. Examples of local allergies to hyperergic inflammation are pulmonary embolism and infectious inflammation in acute rheumatism.

Hypergic inflammation is slow, weak. Hypergic inflammation occurs in organisms that may have immunity to this antigen, or are very weak, emaciated, and less reactive. For example, if a diphtheria toxin is injected into the skin of an animal vaccinated against diphtheria, a very slow local change occurs. Such a sluggish response is observed due to decreased reactivity in animals with strong lean and malignant tumors.

Why does inflammation manifest as a general organism change?

Yallig‘lanish manbai bilan organizm o‘rtasida o‘zaro aloqadorlik va bir-biriga ta’sir etish hosil bo‘lib turadi birinchidan yallig‘lanishning hosil bo‘lishi va rivojoanishi organizm reaktivliligiga, uning boshqaruvchi mexanizmi, moddalar almashinuvi va boshqalarga bog‘liq ikkinchidan yallig‘lanish manbai organizmdagi moddalar almashinuvi, immunologik xususiyatlarga ya’ni barcha organizmga ta’sir qiladi. Sensibilizatsiyalangan hayvon organizmga zaharli bo‘lmagan qo‘zg‘atuvchilar bilan ta’sir etilganda kuchli giperergik yallig‘lanish kelib chiqishini, immunlangan organizmlarda zaharli moddalarga xos yalig‘lanish jarayonlarini chiqaradi. Yallig‘lanishning shakllanishida nerv reflektor jarayonlar muhim ahamiyatga ega. Masalan: retseptorlarni blokada qilib yallig‘lanishni susaytirish yoki umuman hosil qilmaslik mumkin. Nervsizlantirilgan to‘qimada yallig‘lanish juda sust va belgilarsiz kechadi. Simpatik nervning qo‘zg‘alishi yalig‘lanishni susaytirsa, parasimpatik nerv kuchaytiradi. Oraliq miyadagi kulrang do‘mboqchaning uzluksiz qo‘zg‘atilishi organizm turli qismlarida: terida, ichki organlarda keng yallig‘lanish jarayonini chaqiradi. Hayvonlar narkoz xolatda, qishqi uyqu vaqtida va po‘stloq tormozlanganida harqanday kuchli qo‘zg‘atuvchi ham yallig‘lanish chiqarolmaydi. Hayvonlar organizmining murakkablashishi, nerv sistemasining diferensiyalangan bo‘lishi, ularda yallig‘lanishni to‘la belgilari bilan aniq kechishiga, organizmning ximoyaviy xususiyatlarida fagotsitoz, leykotsitlar emigratsiyasi va proliferativ jarayonlar yaqqol kechishini ta’minlaydi.

Inflammation is also affected by the endocrine glands, while thyroxine, aldesterone and somatotron hormones increase inflammation, while AKGT, cortisone and sex hormones histamine, acetylcholine, serotonin and others.

Inflammation depends on the age, type, constitution, sex, and other characteristics of the animal, and hyperergic inflammation cannot occur in young animals. If the signs of inflammation are well manifested with the age of the animal, in old, loose constitution, inert nerve-type animals, inflammation is slowed down and conditions are created for the spread of the pathogenic agent in the body. Inflammation of the abdominal cavity of horses is more acute and severe than in cattle, or if we send tuberculosis rods under the skin to guinea pigs, they form a long-term incurable wound at the injection site. calls. The development of inflammation depends on the anatomophysiological structure of the organism, if the inflamed parts are well supplied with blood vessels, the inflammation will be so strong and, conversely, if the blood vessels are poorly supplied, the inflammation will be asymptomatic. Inflammation is affected by animal nutrition, metabolism, low protein content in the diet, reduces the formation of immune cells in the body of the animal, weakens the resilience of patients, vitamin A deficiency from avitaminosis can lead to easy inflammation of the eyes and respiratory tract. causes. The intensity of inflammation varies in different vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different

vitamin deficiencies. Vitamin A deficiency from avitaminosis causes easy inflammation of the eyes and respiratory tract, while affecting metabolism and low protein content in the diet weakens the resilience of patients by reducing the formation of immune cells in the animal. The intensity of inflammation varies in different vitamin deficiencies.

How does the source of inflammation affect the body?

Yallig‘lanish organizmning mahaliy qon tomirlar reaksiyasi sifatida nomoyon bo‘lishiga qaramasdan, organizmning umumiy xolatiga, moddalar almashinuviga, immunobiologik reaktivliligiga, qon tarkibiga, termoregulyasiya va jarohatlanmagan to‘qimalarga ta’sir qiladi. Yallig‘lanish davrida moddalar almashinuvining buzilishidan, glikoliz jarayoni kuchayib qonda qand miqdorini ko‘payishiga, albumin-globulin indeksini o‘zgarishiga, globulinlarni ko‘payishiga, qonda qoldiq azotni, albumoz-peptonlarni, gistamin, nukleinlar almashinuvining oraliq mahsulotlari va atseton tanachalarini ko‘payishiga olib keladi. Qonda leykotsitlar ko‘payadi, ECHT tezlashadi, tana harorati ko‘tariladi. Immunobiologik reaktivlik yo immunitetni hosil bo‘lishini kuchayishi yo pasayishi bilan harakterlanadi: emlash va kasallikdan tuzalgandan keyin antitela hosil bo‘lishi va fagotsitoz kuchaysa, surunkali kechadigan yallig‘lanish jarayonida immunobiologik reaktivlik va rezistentlik susayishi madorni qurishiga olib keladi. Yallig‘lanish manbai o‘ziga yaqin to‘qima va organlarga ta’sir qilib hayvonlar qorin bo‘shlig‘iga filogen moddalar ta’sirida qorin devoriga yuborilgan mikrobga turg‘unligi kuchayib, bu mahalliy to‘qimalarni immunologik xususiyatlarini kuchayishidan hosil bo‘ladi. Yallig‘lanish manbailarini jarohatlanmagan to‘qimalarga ta’sirini ba’zan organizmdagi qorin sohasining yallig‘lanishi appenditsit yoki aritmiyalarini hosil bo‘lishida ko‘rish mumkin.

The inflammatory center affects the whole organism, affecting its metabolism, reactivity, uninjured organs and systems due to the microorganisms accumulated in these inflamed parts, their breakdown products, toxins, biologically active substances that are absorbed into the blood and tickle the receptors. . The body is also affected by painful stimuli coming from the source of inflammation. The increase in body temperature is caused by the effect of completely undigested substances formed in these parts on the thermoregulatory center in the midbrain. Thus, the source of inflammation affects the body through nerve reflex and neurohumoral pathways.

What do you mean by the mechanism of development of inflammatory processes?

It is a complex reaction of the organism to inflammatory influences that appeared very early, and theories explaining these processes have also been known since very ancient times. The protective properties of inflammation are also stated in the ideas of Hippocrates, who have different views and worldviews on the essence of inflammation.

According to R. Virkhov's 1958 theory of nutrition, inflammation is the transition of cells to a high functional state under the influence of inflammatory factors, a state of intensive consumption of nutrients. However, cells not only undergo a high functional state under the influence of a phlogogenic agent, but also under a high functional state during other effects. R. Virkhov equated inflammation with a simple arousal phenomenon and could not explain that arousal is another qualitatively specific phenomenon. If the proliferative and exudative processes in inflammation are considered a high functional state, the alternative process cannot be considered as such. By binding the inflammation to the cell,

Congeym's theory of vascular changes in 1885. It is said to cause changes in the blood vessels leading to inflammation. Congeym says that the changes that occur in inflammation are due to increased vascular permeability, i.e., exudation and emigration. This theory ignores the fact that other tissues, not blood vessels, play an important role in the development of inflammation. The fact that there is an inflammatory process even in animals with underdeveloped vascular systems did not take into account the fact that vascular permeability is controlled by the nervous and humoral systems.

In Ricker's vasamotor theory, inflammation is explained as a phenomenon associated with changes in the vasomotor nerves under the influence of a phylogenetic agent. Inflammatory nerve exposure causes changes in vascular permeability and tone, leading to the formation of inflammatory-specific metabolic changes in tissues. In this theory, the interaction between the flogen agent and the tissue is ignored and the role of the nervous system is limited. IIMechnikov's phagocytic theory was stated in 1892. Inflammation is a protective reaction formed as a result of evolutionary development, in which

specific cells of inflammation (RES cells) are considered active in response to the action of a phlogogenic agent. This theory suggests that vessels, other than phagocytes, are cells of the nervous system,

In Shaden's physicochemical theory of 1923, he explained that inflammation under the influence of a phylogenetic agent disrupts tissue metabolism and alters the physicochemical properties of colloidal substances as the main pathogenetic chain of inflammation. Inflammation is only a local process, it does not take into account the reactivity of the organism, the state of the regulatory mechanisms that play an important role in the development of inflammation. Thus, inflammation is associated with alteration, necrobiosis, venous hyperemia, stasis, intoxication, dysfunction and other events, on the one hand, arterial hyperemia with protective compensatory properties, accelerated metabolism, leukocytosis, phagocytosis, emigration, multiple antibodies. and the formation of biostimulants, proiferation,

At the end of the twentieth century, the role of the nervous system in the development of inflammation was raised. Samuel recognizes and promotes the importance of the nervous system, saying that neurotrophic processes play an important role in the origin of inflammation, that the influencer affects the cell through the nervous system.

While V.Ya. Danilevsky cut the sympathetic nerve and observed strong inflammation in the tissue controlled by this nerve, Ricker explained that inflammation is caused by dysfunction of vasomotor nerves, and these theories led to the notion that inflammation occurs in the organs. will come.

Only IPPavlov tries to explain that with the development of the theory of nervousness and its role in the nutrition and metabolism of the nervous system is important, that inflammation develops on the basis of important laws. IPPavlov observes that wounds on the skin and mucous membranes of dogs with tubes are formed under the influence of chronic pathogens. These chronic movements are caused by improper placement of the tubes. Inflammation is provided only by the injured nerve and has been observed in other organs or tissues as well, not only in the tissues. For example, inflammation of the cornea of the eye was observed when the sciatic nerve, the cervical sympathetic node and the gray ball and some centers were stimulated. The effect of the cerebral hemisphere on the inflammatory process, when the bark is removed or the animal is anesthetized, the inflammation is sluggish and goes unnoticed. Similar changes are not caused by inflammation during the hibernation of animals, in severe poisoning (mustard, when large amounts of leucites are introduced into the body). Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body. Loss of receptor-receptor properties triggers inflammatory processes that either do not produce or weak inflammation. However, some signs of inflammation can be observed in degenerated or growing tissues from the body.

inability of tissues to have specific biological properties, unlimited growth and control, and changes in the structure and function of tumor cells. These properties in tumor tissue are caused by the influence of external and internal environment on disease-causing causes in healthy cells in the body. Tumor tissue, unlike other pathological changes in the tissue, does not have the properties of regeneration and flexibility (regeneration, hypertrophy, proliferative inflammation) in the body. Not only does the tumor increase in size when the tumor grows, but the tumor can also break down the surrounding tissue.

The branch of pathological physiology that teaches the problems of tumors is called oncology-Greek-oncos-tumor or neorlasma-new abnormal formation, Latin-tumor-tumor. Tumors can form and develop from healthy tissues in the body (epithelial, connective, muscle, nerve). Tumor-forming substances are called carcinogens. The transformation of healthy cells into tumor cells is called malignancy. Tumors are formed by adding a suffix "oma" to the name of the tissue from which they are formed: For example: epithelioma, fibroids, lipoma, osteoma, chondroma, adenoma and others. Some tumors, as they are called by their historical name, are called malignant tumors (sapsech,

sachstpoma) formed from epithelial tissue and malignant tumors formed from connective tissue. Tumors have a parenchyma and a stroma, and the characteristics of the tumor depend on its parenchyma. Blood vessels and nerves pass through the tumor stroma and are composed of connective tissue. Because malignant tumor stroma is so poorly developed, these tumors are called histoid tumors. In benign tumors, the stroma is well developed, surrounded by a thick shell, and is called an orgonoid tumor, reminiscent of a parinchymatous organ. If the tumor parinchyma is composed of multiple tissues. These tumors are called mixed tumors. Hence, we study all tumors into two groups i.e. malignant and benign tumors. Malignant tumors include cancer and sarcoma, all remaining tumors include fibroids, fibroids, ostiomas, chondromas, adenomas, and other benign tumors.

Safe tumors are called tumors that are close to the mother cell and mature due to their morphological structure. As benign tumors grow, they grow from the center to the periphery, enlarging to form a connective tissue shell and compressing the surrounding tissue as they grow. Because benign tumors have a connective tissue shell that is confined to the surrounding tissue, they grow slowly and sometimes temporarily stop growing. In dogs, the size of the tumor increases and the dogs become 1/3 of their body weight. The expansion of a tumor without growing into other tissue is called expansive growth. Safe tumors do not recur and metastasize when surgically removed because they are surrounded by a good connective tissue shell. Of course, a safe tumor is a relative concept. the formation of this benign tumor in the brain leads to disruption of the activity of various nerve centers by squeezing the brain. Safe tumors that form from the endocrine glands cause the production of many hormones and disrupt the functions of the endocrine glands. Safe tumors grow around the red eyelids and other tubular organs, squeezing them, causing dysfunction.

Malignant tumors grow rapidly, irregularly, and are not limited to the surrounding tissues, but grow into them and are called infiltrative growths. Malignant tumors injure the surrounding tissue. The central part of malignant tumors disintegrates without good nutrition and does not become large in size. Tumor growth is variable, sometimes rapid, sometimes slower than in benign tumors. When malignant tumors grow, there is no boundary between the tumor and the healthy tissue, so the malignant tumor cannot be separated from the body. If the sma cell remains, it recurs. Recurrence is a characteristic feature of malignant tumors. A recurrent tumor can form long after it has been removed. Malignant tumor metastasis-Greek metastasis - displacement, interference, which causes tumors to grow into the blood and lymph vessels, staring at the capillaries and forming an embolus. Cancer often metastasizes through lymphatic vessels. Wherever tumors develop when they metastasize, they retain the characteristics of maternal tumors. For example, regardless of which part of the body the hepatoma is formed, it produces urethra, a tumor formed by the thyroid gland is rich in iodine. The formation of metastases depends on which blood vessel the embolus flows through. For example: If the cancer has developed in the stomach, it metastasizes primarily to the liver. In other cases, the formation of metastases depends on the biochemical properties of the tissue in which the metastasis occurs. If you have lung cancer, metastasis will form in the brain and adrenal glands. Thyroid, malignant tumors of the prostate and mammary glands often metastasize to bone tissue. However, the entry of tumor cells into the organs does not always lead to the formation of tumors because they are broken down by macrophages. For example, the flow of cancer cells in the spleen does not cause metastasis. Malignant tumors are so different from benign tumors that in malignant tumors the metabolism changes more deeply than in benign tumors, causing the animals to lose weight.

Tumors are found in all farm and domestic animals, birds, amphibians, and fish. It is even found in various invertebrates as shown in the literature. Sarcoma from malignant tumors in cattle, lipoma, fibroma, ostioma from malignant tumors, melanosarcoma, osteosarcoma and cancer from more dangerous tumors in horses are found in cattle. Tumors in the genitals and other parts of bulls and stallions are more common. Tumors rarely form in the stomach and uterus of animals.

Tumors are more common in older animals. Dogs of purebred and older than 5 years of age have a variety of tumors, most commonly tumors of the genitals and mammary glands. Tumors are rare in rabbits, and tumor damage is very rare in guinea pigs. While laboratory animals are more likely to develop cancer in mice, sarcoma is more common in rats. According to some data, 6-8% of mice die from cancer. Tumors also occur in chickens, where they develop sarcoma. Similarly, geese and ducks

are also affected by tumors. In birds, malignant tumors grow and metastasize. In fish, as in other vertebrates, epithelial and connective tissue tumors are different. Tumors are more common when fish are artificially bred and are less common in free-living fish.

Tumor formation also depends on the age of the animals, with tumors occurring in humans after the age of 40, in dogs after the age of 5, in chickens at the age of one year, and in older animals 10%. The occurrence of tumors in older animals is associated, firstly, with the long-term effects of etiological causes, and secondly, with a decrease in the body's protective functions.

The importance of hereditary traits in the origin of tumors has not yet been definitively studied. However, cancer is caused by viruses, and if an animal is born with cancer after birth, it will develop cancer. This condition is well studied by infecting the animal's udder with the virus.

5. The causes of tumors have not yet been fully studied, and the first information about tumors dates back to 1500-2000 BC in ancient Egypt and Rome, and Hippocrates in those days. tumors can be treated or untreated. In the seventeenth century in England in the cleaners of factory pipes - a disease of pipe workers, in the United States - tumors in the clockmakers of a phosphorus plant. In the first half of the 19th century, œcìàëàð was found to be composed of cells, like other tissues, and the origin of tumors has been explained by various theories. One of these theories is the theory of embryonic buds, in which Congeim argues that during the embryonic development of an organism, some of the cells fail to develop, and that various causes, strikes, due to inflammation and other causes, growth energy is formed in cells that live in secret and begin to grow. Tumor feature is formed. Tumors begin to form. Proponents of this theory explain that tumors and embryonic tissues have morphological similarities, that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor. explains that they are formed from parts that are very difficult to differentiate in embryogenesis. Only teratomic tumors are formed from embryonic cells, which do not enter malignant tumors, enter the altered state of the organism, and cannot fully explain the origin of the tumor.

R. Virkhov's theory of exposure was developed in 1885 and explains that it is caused by the action of long-term pathogens on tumors, resulting in the formation of lesions in many tissues. This theory explains that tumors are formed in humans and animals in the processes of tissue breakdown, inflammation, and regeneration due to long-term mechanical, thermal, chemical, and other effects. It is said that cancerous tumors are formed as a result of long-term exposure of certain parts in people performing the same functions, from proliferative inflamed parts to the differentiation of cells. But not all formed scars and wounds form tumors. This theory seeks to explain that tumors are formed under the influence of chronic influencers of the external environment. VVPodvesotsky observed that tumors do not form when the body is exposed to mechanical and chemical agents for a long time. However, due to this theory, conditions have been created for many studies and the causes of tumors have not been identified. As a result, in 1916, Japanese scientists K. Ishikova and K. Yamagiwa discovered that tumors are caused by chemicals. They rubbed dyogt charcoal on the inside (skin) of rabbit ears for a long time, causing malignant tumors. Diagnostic cancer was later invoked from experimental animals in mice, rats, and dogs. Two weeks after the coal tar has been applied, the wool from these resinous parts falls off and new wool emerges, and after this change is repeated 6-7 times, the wool does not grow on the skin at all. the skin thickens, roughs, cracks, the outer surface of the skin sheds and alternates. If we observe these parts under a microscope, we will see acute, moderately acute and chronic inflammation of the skin after a month in the place where the coal tar was applied. 3-4 months later, sometimes earlier, sometimes later, one or more questions arise. These tumors then grow, enlarge, infiltrate, and metastasize to a cancerous tumor. Subsequent research has shown that carcinogenic chemical compounds are synthesized from various resins that cause tumors. Carcinogens are polycyclic carbohydrates with their chemical structure. Carcinogens form tumors after several latent periods after they enter our body. If left untreated, a rapid tumor can form. Cancer tumors form

by the 31st to 179th days after the skin is coated with methylcholentren. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. After 4-6 months, a sarcoma tumor is formed at the site of methylcholentren injection. Nowadays, 300-400 different compounds of tumor-causing chemicals are known, and even disorders of fat metabolism - disturbances in the metabolism of streins - can lead to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat from mice and rats from hungry animals. even a violation of fat metabolism - a violation of the metabolism of streins, which leads to the formation of tumors. The organism also contains substances similar to carcinogens in their chemical structure, of which 1,2-benzpyrene, 5,6-cyclopentene 1,2-benzathratsene affect the sex hormones of female animals, castrated It produces active carcinogenicity at the same time by invoking heat in mice and rats from hungry animals.

Cholesterol, sex hormones, vitamin D, carcinogens in the benzperin group are chemically close and they are phenanthrene products. Some substances change their carcinogenic properties as a result of various effects. For example, cholesterol in grass can be turned into a carcinogen under the influence of radiation. NILazerev's observations show that when hormones are overproduced or a decrease in their antagonists leads to tumor formation. This means that an adequate stimulus forms a tumor when it changes in quantity. The process of cell dedifferentiation and rapid proliferation to form a tumor can lead to malignancy and tumor formation.

Impaired sterein metabolism from fats and lipids is a factor that contributes to the growth of tumors. The formation of malignant tumors under the influence of carcinogens is one of the important achievements of experimental oncology. However, the mechanisms of action of carcinogens have not yet been elucidated. Perhaps the effects of carcinogens acquire biological properties by altering the genetic properties of cells by disrupting the structure and function of nucleic acids. Even chemical theory cannot fully explain the formation of tumors. He explains that chemicals only create the conditions for viruses to affect the body.

From the end of the last century to the present day, tumors have an infectious nature, they explain the parasitic ducts that cause disease in various animals and plants, worms-worm-like parasites, fungi are specific pathogens of tumors. During the study of tumors, many microorganisms were isolated, but all of them were found to be saprophytic microbes and not related to tumors. Malignant or malignant tumors also occur when infected with certain parasites: Cancer can occur in dogs and cats when infected with Oristorshis felineus, which belongs to the class of suckers. Cancer develops when rats are fed cockroaches, or when cattle become infected with fasciola, which causes liver cancer.

The notion that tumors are caused by viruses was first proposed by II Mechnikov in 1910, and in 1911 an English scientist, P. Rose, observed that tumors were formed by sending a filtrate made from sarcoma-infected chicken tissue. P.Rous virus is found not only in tumors but also in the heels, liver,

brain, blood and other fluids of chickens, the size of the virus is 01 m. Low resistance to chemical and physical influences. For example, it decomposes in 2-3 days at a temperature of 00, and in 15 minutes at 550. Antisetics have a strong effect on the virus. Some tumors can grow in an environment made of tissue. Safe tumors formed under the influence of viruses have been observed in various animals to develop into malignant tumors. For example: papilloma of wild rabbits, in dogs and cattle papillomatosis is similar to the warts that occur in humans, and the virus isolated in these animals causes tumors only in this type of animal. Most tumors can only develop in a healthy organism when transplanted. Proponents of viral theory, such as LAZilber et al. The tumor-causing virus may not show its pathogenicity for a long time, even in all vital processes. For example, while some species of mice reach a certain age, most of them become infected with tumors, while others develop one or two tumors. Because tumors can also call a healthy animal child by suckling an infected animal, this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. this leads to the conclusion that viruses in diseased organisms can pass through blood-sucking insects. Viral theory also cannot fully explain the origin of tumors, as tumors can often be induced even under the influence of chemicals. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories. The occurrence of tumors in different animals, their formation from different tissues, viruses perform the function of non-specific causative agents of viruses. Thus, despite the fact that the above theories explain the formation of tumors to one degree or another, all of these theories are polyetiological theories.

6. Tumor growth begins with the transformation of normal healthy cells into tumor cells, and the metabolism in these cells changes. produces qualitative changes from the biological properties of the cell. Later tumors grow only due to the proliferation of tumor cells. Of course, not all tumor cells turn into tumors, some are absorbed, and some form multiple tumors.

One of the main characteristics of tumors is that they can grow continuously and, if not removed by a doctor, squeeze the animal’s organs, causing death under the influence of toxins. As a result of continuous growth of tumors, the fibroma in cattle reaches 100 cm in diameter and weighs up to 100 kg, about half the weight of the animal. In humans, uterine fibroids weigh 20-25 kg, and ovarian cysts range from 50 kg. By transplanting tumors in the same species, it is possible to ensure their growth for several years. One of the characteristic features of tumors is the transformation of tumor tissue into low-differentiated tissue.

Anaplasia refers to low-level morphological differentiation of mother cells into tumor cells, and Greek means mother-back, down, plasis-formation. In a cell that is becoming a tumor, the rate of growth and proliferation increases. The faster the growth in the tumor cell, the better the anaplasia develops. Usually morphological, biochemical, physicochemical and energy anaplasia are distinguished.

7. In morphological anaplasia, changes occur in the tumor cell and tissue, and according to the morphological features, the tumor tissue is close to the embryonic tissue. The shape and size of the

parenchyma of tumor cells vary. In some cells, the normal ratio of nucleus and protoplasm is different, the number and shape of chromosomes change. The division of tumor cells is atypically malformed, disrupting the mutual arrangement of cells. For example, glandular tumors do not have or have a malformed structure that produces glandular fluid, but retains the functional properties of tumor cells despite having such an atypical structure. That is, tumors formed from melanblasts melanin, tumors formed from liver cells, tumors formed from grass, glandular cells, maintains the function of hormone production. Morphological atypicality is not specific to tumors but can also result in cell growth and proliferation in a variety of pathological conditions. For example: During regeneration and proliferative inflammation.

8. During biochemical anaplasia, the biochemical properties of tumors change, that is, as in embryonic tissues, the amount of water increases to 90%. Potassium salts increase and calcium salts decrease from normal. The faster the tumor grows, the more the ratio of potassium and calcium changes.

Tumors increase cholesterol from lipoids. Tumors accumulate a lot of glycogen, which does not absorb glycogen well. This glycogen accumulates as a result of disruption of carbohydrate metabolism and is associated with an increase in lactic and pyruvic acids in tumors.

DNA and RNA increase in tumor tissue. As a result of the strong breakdown of nucleic acids, pentoses are formed in tumors, the amino acid composition changes, ie cystine, methyanine, tyrosine are reduced in tumors, and histidine, arginine and lysine are increased. Tumors are rich in protolytic enzymes.

6. In physicochemical anaplasia, the surface tension properties of colloidal substances are reduced, many completely unoxidized intermediates are formed, changing the acid-base balance to acidic. Osmotic pressure rises in tumors. Tumor tissue has a higher electrical charge than healthy tissue. Tissue and cell membranes have strong permeability properties. Biochemical and physicochemical anaplasia occurs in the process of regeneration or proliferative inflammation without any specific changes for the tumor. The stronger the growth of a charged tumor, the better the biochemical and physical anaplasia.

Energy anaplasia is caused by changes in metabolism and excessive metabolism in tumors, disruption of carbohydrate and protein metabolism.

7. Metabolism in tumors differs from that in healthy tissues, i.e. we can better observe these changes in carbohydrate metabolism: in healthy tissues, carbohydrate metabolism takes place in 2 periods: anaerobic and aerobic.

As a result of many intermediate changes in the anaerobic period, lactic acid is broken down - called glycolysis.

In the aerobic cycle, 1/5 of lactic acid is oxidized to SO2 and N2O, and the remaining 4/5 is converted to glucose due to energy generated by oxidation.

During glycolysis, 5% of potential energy is wasted on carbohydrates, the remainder being oxidized to form S2O and N2O from lactic acid. When the oxidizing properties decrease, a lot of lactic acid is formed, and acidic substances accumulate in the tissues. Glycolytic processes are dangerous tumors, the breakdown of glucose to lactic acid is 200 times faster than in resting muscles and 8 times faster than in maximally working muscles. Malignant tumors can produce lactic acid equal to their own weight in 10-12 hours. Therefore, the amount of lactic acid in the blood is higher in cancer-prone organisms. Glycolytic changes in malignant tumors are more active than in benign tumors. The formation of large amounts of lactic acid, changes in the surface tension of tumor tissue, etc. are characteristic of tumors. Cancer cells break down glucose 4-5 times more strongly and oxidation is very slow. Glycol = dog processes are not characteristic of tumors, because glycolytic processes occur in the retina, leukocytes in healthy life processes, increased glycolysis, decreased oxygen consumption are also observed in the process of inflammation and regeneration . Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic

processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a decrease in oxygen consumption are also observed in the process of inflammation and regeneration. Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body because glycolytic processes occur in the retina of the eye, in healthy life processes in leukocytes, an increase in the process of glycolysis and a decrease in oxygen consumption are also observed in the process of inflammation and regeneration. Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body Glycolytic changes are intensified during the vigorous growth processes of various animals. But REKovetsky found that the property of strong glycolysis is a constant change, mainly characteristic of aerobic glycolysis tumors. Metabolic disorders are formed before the tumor is formed and spread throughout the body

In tumors, protein metabolism is severely impaired, albumin and nucleoproteins are increased in tumor proteins, and proteins that are not found in healthy tissue are found. The formation of these nucleoproteins has not been studied, but other proteins or viruses of a different nature (LAZilber) or proteins that have been altered by the body in the formation of tumors.

In malignant tumors, full-value and full-value amino acids can also be formed. Proteins in this change can disrupt the activity of enzymes. BIZbarsky determined that specific protein synthesis occurs in tumors and is called tumoproteins.

The disruption of specific nucleic acid metabolism in tumors was discovered in 1934 by Stern and Wilheim, and later in 1941 by Rondoni in tumors where DNA was more than RNA. It has been studied that protein synthesis in tumors is superior to its breakdown by sending various identified atoms into the body. The fact that purine and pyrimidine bases from large amounts of amino acids fall into the tumor tissue and that the amount of residual nitrogen in the tumors is high indicates that the protein metabolism in tumors is faster than in healthy tissue.

The metabolism of fats and lipids is strong in tumors and varies depending on the nature of the tumor. Fats are high in unsaturated fatty acids, cholesterol and acetone cells.

Relationship of tumors with the organism. Based on the data collected in the experiments, MKPetrova explained that the effect of the body on the growth of tumors can affect the nervous system in tumors. The creation of conditions for the origin of tumors in chronic functional disorders of the nervous system (neuroses) in the animal body has been studied experimentally by calling dangerous and benign tumors. During the period of chronic functional disorders of the nervous system, the formation of tumors under the influence of carcinogens is accelerated. The role of the nervous system in the mechanism of tumor development has been observed to slow the growth of tumors under the influence of carcinogens during the hibernation of animals or the inhibition of nervous system activity, and accelerated tumor growth in controlled animals receiving so many carcinogens. If we send sodium bromine to the body, the activity of the nervous system decreases and the formation and development of tumors slows down. It is during this period that the effects of caffeine or nervous system stimulants on rabbits accelerate tumor growth.

Injury to peripheral nerves contributes to the formation of metastases. If the sympathetic nerve of the neck is cut, malignant tumors will form, which will help the transplant to grow. The effect of RES tissue on tumor growth is significant, as macrophages can break down the tumor without developing it, preventing it from growing. Macrophages resist metastasis by trapping malignant tumor fragments that enter the blood and lymph. AABogomolets and MANavinsky in 1877 observed that activation of RES tissue function prevents the transplantation of transplanted tumor tissue, or blockade of RES tissue creates conditions for the growth of transplants.

The body influences the growth of tumors through hormones produced in the endocrine glands. While one of these hormones inhibits the growth of tumors, the other accelerates the growth of tumors. For example, while somatotron hormone in the pituitary gland enhances tumor growth, hormones in the pancreas and adrenal cortex inhibit tumor growth. When we send estrogen hormones to an animal's body, a tumor develops in the animal's udder and genitals. Testosterone and progesterone inhibit tumor formation in the udder and genitals.

As the body reacts to tumors, so do tumors. The effect of tumors on the body depends on the nature of the tumor, its growth and the location chosen. If there are small tumors on the surface of the hand, they fall into the category of benign tumors, which only cause discomfort when doing any work. possible. Safe tumors compress the surrounding tissues, disrupting their nutrition and leading to atrophy. If the sap compresses the separating pathways, the sap becomes difficult to separate, and so on

Although malignant tumors are small, they degrade the body and lead to death due to impaired growth and metabolic disorders. The cause of weight loss in animals is caused by metabolic disorders, poisoning the body with intermediate products of metabolism and due to the breakdown products of tumor tissue. From it, the dysfunction of the organ in which the tumor grows also causes the body to lose weight. Tumors show antigenic properties to the organism as they begin to grow, but the structure of these antigens has not been determined, but antibodies to these antigens are formed. Antigens are sufficiently foreign, due to the lack of foreign antigenic properties, as well as the weakening of the immune-forming functions of the immune system and the low production of immunogens, which can not protect the body. The presence of malignant tumors in the body disrupts the overall metabolism. In the initial period of tumor formation, metabolism increases and decreases in the next period. Blood glucose may increase or decrease.

Increased activity of enzymes involved in carbohydrate metabolism increases lactic acid in the blood, including in the veins. A decrease in serum albumin in the blood leads to a decrease in protein and an increase in residual nitrogen. Decreased albumins are associated with decreased protein synthesis. When tumors grow, the activity of arginase, catalase, oxidase in the liver decreases, glycogen synthesis, urea, guipuric acid formation is impaired, the total amount of nitrogen excreted from the body increases, and urinary urea decreases. In the urine, lactic acid, polypeptides, some amino acids increase, and acetone cells appear. According to NBMedvedev, in cancer, carbohydrates are 6-7 times more than nitrogen. Tumors cause hypochromic anemias in the body, decreases to 0.5 to the color index of the blood. Anemia is caused by the breakdown of erythrocytes under the influence of various charged substances, ie not completely oxidized. Disruption of the control of the activity of blood-forming organs by the formation of erythrocytes by nerves and endocrine glands leads to anemia.

During the transplants, he observed that the infinite features of the tumors were visible. Tumor strains are also present today, including the well-studied Erlix mouse cancer, Jensen’s rat sarcoma, Raus’s chicken sarcoma, and others, which have been transferred from organism to organism for hundreds of years and have existed for 50 years or more. The nutrition of the experimental animal plays an important role in transplant growth, and if the caloric content of the food is low, i.e. lysine, arginine, histidine, the growth of tumors is inhibited. If it contains a lot of carbohydrates, cholesterol and potassium in the diet, the growth of the tumor will accelerate. Liver cancer can develop even if the animal does not have enough choline in its diet. But the growth of tumors did not stop as a result of complete starvation of animals SAMMI researcher IP Mishenko observed in chickens and rats. Experiments have shown that tumors can be grown outside the body by creating special nutritional conditions, as observed by ADTimofeevsky et al. Thus, the role of the nervous system in the origin of the tumor is also important, as the causes of the tumor include chemicals, mechanical stimuli, light energies.

In the body of highly developed warm-blooded animals, body temperature changes in a very short time, and their body temperature depends on the specific condition of the animal, type, development of sweat glands, time of day, age. The temperature is not the same in different parts of the body of an

animal of the same species. Relatively uniform temperature maintenance in the body is ensured by physical and chemical thermoregulatory mechanisms, a process controlled by the CNS and endocrine glands.

Heat exchange is provided by the MNS using conditioned and unconditioned reflexes. Experiments have shown that in the back of the gray matter of the midbrain is a center that controls the formation and transmission of heat. This control is controlled by the centers of metabolism, vascular tone, respiration, and sweat secretion, and these processes are related to the activity of the hypothalamus and cerebellum. Needle puncture in the hypothalamus raises the body temperature of the animal to 2.5–30. Heat exchange depends on the activity of the shell, and in animals where the shell is removed, the heat exchange is disrupted. In dogs, it is possible to control heat exchange by a conditioned reflector pathway.

The heat exchange is controlled as follows: thermally excited cold-floating Krauze flasks excite the heat-floating Ruffin bodies and transmit the effect to the MNS. From there, impulses are transmitted to various organs, altering vascular tone, sweating, respiration, altering metabolism in the muscles and liver, and regulating heat exchange also depends on blood temperature. The pituitary gland, thyroid gland, adrenal gland, pancreas and other glands from the endocrine glands are involved in the regulation of heat exchange in conjunction with the nervous system. For example: if the body temperature rises when we send hormones or extracts of the pituitary gland, thyroid gland, adrenal glands, lower the body temperature by sending pancreatic extracts, or such changes in the pituitary gland, observed when the thyroid gland and adrenal gland are removed. As the body cools, the pituitary gland begins to secrete AKTG and the animal's resistance to the cold increases. If the center that controls heat exchange in the midbrain is injured, the body does not respond to a decrease in ambient temperature with an increase in metabolism, and vice versa. Thus, the depletion of heat exchange in the body of animals is observed when the activity of the nervous and endocrine systems, as well as the activity of peripheral organs and systems is impaired. Disorders of heat exchange are manifested in the form of hypothermia, hyperthermia and fever, all of which are caused by a violation of the control of heat exchange and are accompanied by changes in body temperature of the animal.

Hypothermia is derived from the Greek word hypo- low, terme- heat, and is characterized by a decrease in body temperature as a result of the regulation of heat exchange. Hypothermia is caused by exogenous and endogenous causes. Exogenous causes of hypothermia include a decrease in ambient temperature: humidity, increased wind, exposure to medicinal substances, and radiation poisoning.

Hypothermia caused by endogenous causes: severe blood loss, starvation, starvation, weight loss, injury to the CNS (heat exchange control center), prolonged dilation of peripheral blood vessels (shock), neonatal, other in, the activity of the center that regulates heat exchange in older animals is weakened, leading to a decrease in body temperature. Pigs cool faster than cattle because a lot of heat is generated in cattle due to the activity of the anterior chambers. Birds are resistant to cold, geese do not change body temperature at ambient temperature - 90–1020 chickens - 500, ducks - 400. Chickens are also resistant to temperature drops.

There are four periods of hypothermia:

4- During this period, the animal's body activates compensatory mechanisms that increase heat production and reduce heat transfer: narrowing of blood vessels, shrinkage, increased heat production due to muscle activity, movement and tremors, accelerated heart rate and respiration. blood pressure rises. Increases the activity of the thyroid, pituitary, adrenal glands, autonomic nervous system. General and basic metabolism, oxidation and other processes are enhanced.

5. The flexibility mechanisms of heat exchange are exhausted, heat transfer is increased, and some oxygen deficiencies are formed. But the metabolism is high and the rectal temperature drops to 29-270.

6- During this period, metabolism, cardiovascular activity decreases, respiration and rectal temperature decreases to 27-190, but during this period, if the animal is immediately warmed up, we can return to normal life processes. Cooling in the next period reduces vital processes, blood pressure, metabolism, the formation of heat completely stops, sleep is suppressed, fibrillation occurs first in the

heart chambers, then in the ventricles of the heart, the heart stops working and the respiratory center is paralyzed. the temperature in the rectum cools to 12-100.

Characteristic signs for hypothermia are the weakening of the protective mechanisms of the animal organism, phagocytosis, immune formation, oxidation-reduction processes, changes in carbohydrate metabolism, the formation of oxygen deficiency. When an animal that has died from hypothermia is dissected, we see that dystrophic changes have occurred in the liver, kidneys, heart, and CNS. In recent years, artificial hypothermia has been used in surgical practice, especially in cardiac operations, to increase the resistance of the heart muscle to oxygen deficiency. During this time, the body's metabolism slows down and oxygen consumption in cells and tissues decreases. A similar situation is observed during the hibernation of animals.

Hyperthermia (Greek hyper- high, terme- heat) is an increase in body temperature of an animal as a result of a violation of the regulation of heat exchange. It is said to overheat. Hyperthermia is caused by an increase in ambient temperature, an increase in humidity without wind. At this time, heat is radiated and decomposed to the outside, which is not formed because there is no difference in temperature between the organism and the environment. Heat transfer is a key part of heat exchange control, and even the smallest metabolism in the body ensures that there is a lot of heat and that the body temperature is kept constant. Therefore, the excess heat must be expelled from the body.

Keeping animals in tight spaces, moving them in warm rooms, in poorly ventilated vehicles, doing heavy physical work and overheating the pasture can cause the animals to overheat. The high temperature resistance and flexibility of animals depends on their type, breed, age, color, and skin coating system. Sheep are resistant to high temperatures and only after the ambient temperature is 400 and above will their rectal temperature change. The resistance of animals to high temperatures depends on the development of their sweat-sweating system.

While an increase in the ambient temperature of cattle above 300 causes an increase in rectal temperature, pigs are intolerant to this temperature due to the underdeveloped mechanism of sweating. When pigs are kept at an ambient temperature of 310, their rectal temperature rises to an ambient temperature of 0.70, causing them to die without adaptation because they do not have sweat glands. They lose steam and adapt to the heat. Excessive heat increases metabolism and disturbs rectal temperature up to 440. From small animals (piglets and calves) are heat-resistant, while chickens are heat-resistant. Under the influence of heat, the appetite of animals decreases, productivity decreases, blood composition changes, breathing and heart rate increase. The strong heat of the environment in the body causes a change in three periods.

In the 1st period, the compensating mechanisms ensure a decrease in heat generation and an increase in heat transfer. In animals, metabolism decreases, sweating increases, peripheral blood vessels dilate, blood circulation accelerates, respiration accelerates. All this increases heat transfer and ensures that the rectal temperature is maintained without rising. Increased heat transfer is associated with the passage of heated blood in the centers in the medulla oblongata (breathing, heart, blood vessels, sweat secretion, etc.). In the following periods, as a result of overheating of the organism, a second period occurs without adequate mechanisms of adaptation of the organism.

In stage II, the animal becomes agitated, pulse, respiration is accelerated, saliva excretion is accelerated, metabolism is increased, the final product is not broken down, protein is formed in the urine, rectal temperature rises to 2-30. If the heat effect still does not disappear, a third period will occur.

In period III, the activity of the nervous system decreases sharply, the heart and respiration slow down, blood pressure drops, fainting, and rectal temperature rises to 5-60. When the animal's body heats up, it stops breathing, and the heart stops beating during systole. When we examine such animals, we observe that profound changes have taken place in the parenchymal organs.

One of the conditions similar to hyperthermia is the heat stroke of the animal’s body. Such changes are observed in animals during intense muscle activity, when the temperature is high and the humidity increases. Acute heat stroke can lead to death from impaired heart function.

5. Disorders of heat exchange are characterized not only by hypo and hyperthermia, but also by the formation of fever.

Fever-fenbris is a general change of the organism in relation to the pathogenic, more infectious causes, and as a result of violation of the regulation of heat exchange in the body, the animal's body temperature rises, independent of the ambient temperature. Fever is a manifestation of disease formation, which is caused by a violation of the regulation of heat exchange, including the disruption of metabolism in relation to the causes of the disease as a secondary process in the body.

There is a difference in the regulation of physical and chemical heat exchange, while maintaining the process of thermoregulation in the body of an animal with a fever. The body that produces the fever becomes resistant to the effects of heat and cold. In a fever-producing animal, the disruption of heat exchange control depends on the type of animal, age, type of nervous system, and so on. The causes of fever are diverse, and pyro-pyrogens are substances that cause fever, and we study them into two major groups depending on their properties:

7. Causes of infectious fever - various infectious diseases.

8. Causes of non-infectious fever are protein, saline, medicated, fever caused by injury to the nervous system.

Fever is caused by the action of various pyrogenic substances on the control centers of heat exchange. Fever is hypothalamic thermal, and the delivery of these substances under the skin or into the composition of venous blood does not cause any changes. A similar situation can be caused by fever by observing the thermal pathways in the gray matter of the interstitial brain of animals or the nerve pathways leading to that part. Fever cannot be caused if the back and brain are cut apart during exposure to pyrogens. Hence, peeling is also important in the formation of fever, which can also increase injury under the influence of indifferent pathogens.

Along with the nervous system, the role of endocrine glands in the formation of heat is also important. does not participate properly. For example: removal of endocrine glands and pituitary gland, adrenal gland, thyroid gland, pancreas does not cause fever, but the endocrine glands only increase the development of fever, changing the overall biotonus of the organism, reactivity, heat exchange. affects by changing the tone of the control centers. Thus, the nervous system serves as the mechanism that initiates the formation of fever.

Depending on the degree of fever in animals with fever: in subfebrile animals the temperature rises above the upper limit of 10, in febrile animals the temperature rises above the upper limit of 20, in hyperpyretic animals the temperature rises above 30 and above. The rate and degree of fever depends on the ability of the causative agent, the reactivity of the organism, the activity of the immune system, the age of the animal, the type of nervous system, obesity, storage and nutrition.

There are three stages in the development of fever in the body:

10. Temperature rise period - stadium incrementi.

11. Maintaining a high temperature-stadium fastigil from 2-3 hours to 2-3 weeks.

12. Period of temperature decrease - stadium decrementi.

With the formation of heat in each period there is a difference in heat transfer, metabolism, activity of various systems, the reactivity of the organism. Depending on the functional state of the thermoregulatory mechanisms to the reactivity of the organism, the type and strength of the pyrogenic agent, fever occurs at different levels and in different cases. In this process, the thermoregulatory nervous mechanisms, the cardiovascular system, the respiratory system, the functional state of the sweat glands play a determining role.

Whether pyrogenic agents are always present in the body during the course of the disease. Depending on whether the thermoregulatory mechanisms work like this, the following types of fever are distinguished:

22. Permanent type fever-febris continia. The high temperature does not return to normal and causes a change around 10 in the morning and evening. In croupous inflammation of the lungs, acute anaerobic and viral diseases, the temperature may rise in the first period and fall slowly or rapidly in 3 periods.

23. Relieving or remitting fever-febris remittens. Daily changes in temperature are 10 and above in the morning and evening, due to the intense relaxation of the effects of the pyrogenic agent, which occurs in catarrhal pneumonia, sepsis and others.

24. Rising or falling intermittent-febris intermittens. In fever, the thermoregulatory mechanisms are very stable, decreasing to normal when the temperature drops to 2-30 and beyond. In acute hepatitis, people encounter malaria.

25. Tinka dryer or hectic fever-febris nectica. Body temperature fluctuates between 3-50, some temperatures fall below normal and rise again. This type of fever is observed in tuberculosis and septic processes. In animals, thermoregulatory mechanisms are formed when they are stressed, weakened, and their productivity decreases.

26. Recurrent fever-febris recurrens. Body temperature is high and normal for several days, with the pyrogenic agent intensifying from time to time. This type of fever is caused by infectious anemia in horses and recurrent typhoid fever in humans.

27. Atypical fever-febris atypica. Even if the disease progresses, the temperature does not rise, and the disappearance of the disease is accompanied by a rise in temperature, which changes several times a day. This type of fever is observed in horses' mango, sepsis.

28. Ephemeral fever-febris ephemera. It lasts from a few hours to 1-2 days. This type of fever is when vaccinated against tuberculosis and mango, after giving birth to animals, after heavy muscle work, when walking a lot in the heat, or when animals are moved in wagons. It is observed in diarrhea.

During fever, changes in the activity of the nervous system, cardiovascular system, respiration and digestion, kidneys, endocrine glands may occur. Changes occur in the nervous system that lead to disruption of thermoregulation. When the body temperature rises, the SNS is stimulated and then braked. Changes in the nervous system can also be due to the pyrogenic nature of the toxins that accumulate in the body. A characteristic change in the nervous system is caused by a sudden rise in temperature at the onset of fever. It does not cause changes in higher nerf activity as adaptation to pyrogenic substances is formed in the nervous system. This indicates that the organism is poisoned and not regenerated in the MNS. The nervous system of lean animals is impaired, The sympathetic nerve activity of the VNS increases. Changes in temperature rise in young animals are stronger than in older animals. Circulatory disorders are characterized by the redistribution of blood in the body, which causes more blood flow to the internal organs and less in the skin, and later the blood vessels in the skin dilate and more blood flows. The work of the heart is accelerated by the rise in temperature to this maximum, which is caused by the excitation of the sympathetic nerve, the excitation of the cardiac nervous muscle apparatus by hot blood, pyrogens and toxins. Usually a rise in temperature to 10 causes the heart to beat 8-10 times faster. In diseases such as tuberculosis and meningitis, pulse formation weakens when the temperature rises, which is a sign that the disease is getting worse. Some fever develops arrhythmia, In the third period of fever the heart rhythm slows down. While blood pressure rises first, which is associated with increased heart rate, vascular spasm, in the third period, blood vessels dilate, heart rate slows, and blood pressure returns to normal. Sometimes in the third period the blood drops sharply, ie collapse occurs.

Fever changes the quantity and composition of the blood, the intermediate products of protein metabolism in the blood are residual nitrogen, acidic substances increase, alkaline reserve decreases, leukocytes either increase or decrease. ECHT is accelerating. The presence of microbial plaque and even microbes in the blood of animals with fever, the formation of antibodies, etc.

Respiration is accelerated by the excitation of the respiratory center by pyrogenic substances and toxic products contained in warm blood, depending on the activity of the heart. Acceleration of respiration is observed in anthrax, swine fever, pneumonia. Acceleration of respiration has a compensatory effect, increasing heat transfer and increasing the body's oxygen saturation.

Digestive system activity is inhibited, appetite is lost, gastric and endocrine and motor activity is inhibited, and absorption is impaired. The process of putrefaction in the intestine intensifies, gas accumulates and flatulence develops. Digestive disorders lead to the development of autointaxia and deepening of pathological processes due to impaired absorption of nutrients. Disorders of the digestive

organs are associated with increased activity of the nervous system, including the sympathetic nervous system in the VNS.

In ruminants, the motility of the pancreas is disturbed during fever, the secretion into the pancreas is reduced, the acidity is increased, and the microflora and microfauna of the large intestine and microbiological processes in general are disrupted. As a result, the chewing period is broken. Hypo and atony of pre-gastric lesions develop. Food is not digested by stopping in the pancreas. In other animals, movement, motor, secretory, and absorption processes are disrupted throughout the intestinal system during fever. At this time, only water is absorbed from the intestine. During the heating period, animals should be given plenty of water and easily digestible carbohydrate foods to reduce the amount of concentrates in the feed.

There are also changes in the digestive system during the fever period, in the first period there is a lot of blood flow to the internal organs and a lot of urine, while in the second period there is a decrease due to water retention in the body. In the third period, urinary excretion increases again, and the composition of urine changes, glucose sometimes appears protein, albumen.

Sweating decreases in the first and second periods of inhibition of nerve centers, and increases strongly in the third period. Increased digestive processes have a compensatory effect, releasing fever from the body, the release of toxic and pyrogenic substances in the tissues, as well as certain products of metabolic processes in the tissues, and normalize body temperature.

During fever in the liver, the ability of machevina and glycogen production is weakened, the residual nitrogen in the venous blood from the liver increases, and in some fevers, bile secretion decreases.

From the endocrine glands, changes occur in the pituitary, thyroid and adrenal glands, the secretion of AKTG in the pituitary gland increases, and the activity of the thyroid gland increases. The amount of corticosteroids in the blood and urine increases.

Pathological anatomical changes cause dystrophic changes in the parenchymal organs, swelling of the organ, fatty infiltrations.

When there is a dystrophic condition in the organs, including parenchymal dystrophic changes, they disrupt their function, which in turn affects the process of fever. The formation of dystrophic changes in the organs occurs under the influence of overheating, infection and intoxication of the organ.

7. Metabolic disorders during fever are associated on the one hand with the rise of pyrogens in the body. In addition, fever leads to starvation from decreased intake and absorption of nutrients.

Metabolism is disturbed in various ways during the period of fever, however, the general laws specific to fever are not absent. During many fevers, an increase in metabolism, with an increase in dissimilation - an increase in heat production and an increase in basal metabolism by 5-10%, an increase in cardiac and respiratory activity - intensifies the oxidation process.

During the fever, protein metabolism changes, protein breakdown increases due to toxic and thermal factors, instead of the normal 15-20%, proteins are used as a source of 30% energy, 30% of nitrogen-fixing substances in the urine are ammonia, creatinine, urea and others. substances are separated. As a result, the body loses a lot of protein, at which time the body needs to be fed with easily digestible carbohydrates, if the fever is infectious, it is necessary to put glucose.

In chronic infectious fever, fat metabolism is increased, at which time excessive fat consumption is not only associated with fever, but also with starvation and poisoning of the animal. According to some scientists, changes in the activity of the gray matter in the midbrain, the center that regulates fat metabolism, lead to disruption of fat metabolism. Infectious and aseptic fevers are rarely accompanied by hyperglycemia, glucosuria, which is associated with a strong breakdown of glycogen in the liver and muscles and a violation of the regulation of carbohydrate metabolism.

Water - salt metabolism changes during the heating period, the accumulation in the tissues of incompletely degraded products of protein and fat metabolism, causing a lot of water retention in the tissues. Renal function plays an important role in this process, high temperature and toxins are reduced in the second period of diuresis, disrupting the filtration of the kidneys. In the third period, heat transfer, sweating, and diuresis increase, and large amounts of water are released. Salts also increase in

the body as water is retained, many chlorides are retained, and many begin to be excreted in the third period. The release of phosphorus and potassium salts in fever is also enhanced by the intensification of decomposition processes in tissues.

Failure to raise or weaken the temperature during certain diseases in humans and animals has had serious consequences. Other investigators recommend the use of antipyretics during fever, given the toxicity of the organism during fever. When the problem is solved correctly, IPPavlov looks at the disease from the worldview, and if the disease simultaneously disrupts the activity of the organism, the second eliminates the cause of the disease. According to IPPavlov, when the body is affected by adverse causes, the body reacts sharply to this cause. From this process we must be able to distinguish the true disease and the physiological protective process.

, hemolymph is formed, and hemolymph is rich in inorganic and organic substances, which contain proteins and oxygen-carrying pigments.

4. In the organism of hot-blooded animals there is a liquid tissue deposit, the composition of which has complex and extremely important functions. The importance of blood in the body depends on its function. Blood transport in the body. thermoregulation. The physicochemical environment for cells and tissues is very important in the protection and correlation, ie the coordination of neuro-humoral processes. Therefore, changes in the composition of the blood have a huge impact on all functions of the body.

There are several theories about the formation of blood, of which AAMaximov's unitary theory explains the formation of blood in hemocytoblasts - the mother cells of the blood, while later proponents of the duolistic theory explain that Erlix is formed in myeloblasts in monocytic sand.

Changes in the total amount of blood Depending on the type of animal, the amount of blood in the body is 4-5% of the body weight of 8 guinea pigs on horseback and up to 15% on reindeer. 55-60% of the total amount of blood falls on the liquid part of the blood (plasma), and 40-45% on the form elements of the blood (erythrocytes, leukocytes, platelets). Animals that are well fattened will have a much lower amount of blood than lean cattle. The better the muscle tissue is developed, the greater the amount of blood in the animal’s body.

The bulk of the blood (around 50%) is in the blood depot. The amount of moving and stored blood depends on the functional state of the organism. The amount of blood in the body increases or decreases under various pathological influences, during which time the ratio between the liquid part of the blood and the shaped elements changes.

An increase in the total amount of blood. An increase in the total amount of blood in the body means hypervolemia or pleural effusion in Latin huper- excessive, volumen- volume, and there are simple polycythemic and oligocytic types.

4. In normal hypervolemia, the ratio between plasma and erythrocytes is almost unchanged. Under normal conditions, this type of hypervolemia does not occur. Normal hypervolemia occurs after blood transfusion, and such artificially generated hypervolemia quickly return to normal due to the breakdown of erythrocytes in the blood that are then implanted in the body after first plasma filtration (transfer to surrounding tissues).

It is not dangerous to transfuse around 60-80% of the total blood volume of this organism into the body.

5. Polycythemic or true hypervolemia is caused by an increase in the total amount of blood in the body at the expense of erythrocytes. In this type of hypervolemia, an increase in blood volume leads to hyperemia in the mucous membranes, an increase in blood pressure and hypertrophy of the heart.

The blood-forming properties of the red marrow increase — in the tubular bones, the fatty marrow is replaced by red marrow, and young erythrocytes appear in the blood. Polycythemic hypervolymia is caused by chronic oxygen deficiency.

6. In oligocytic hypervolemia, the total amount of blood increases at the expense of the liquid part of the blood, i.e., the amount of water increases. This type of hypervolemia is called serous or hydremic pleurisy. This type of pleurisy occurs in kidney disease, which causes excessive water retention in the body when drinking too much water. Hydremic pleurisy cannot be called

experimentally, because no matter how much saline is added to an animal's body, the deposited fluids pass into the interstitial spaces and are expelled from the body, or a very short-term increase in blood pressure occurs. observed. An increase in water content (hydremia) in the blood can occur even without an increase in the total amount of blood. This hydremia is caused by a decrease in dry matter and protein in the blood, when there is severe weight loss (cachexia), when a lot of blood is lost,

Decreased total blood volume is called hypovolemia or oligemia, which means hypo-less, decreased, volumen-volume, and is divided into simple, pilitsetemic, and oligocytemic types.

13. In normal hypovolemia, erythrocyte and plasma ratios are unchanged, resulting in a decrease in total blood volume and excessive blood loss. Injury to the vessel wall with mechanical injury or tumor. excessive blood loss due to inflammation or wound processes can lead to hypovolemia.

Sometimes a decrease in blood can also be caused by taking blood from a donor. Older and younger animals are more susceptible to blood loss than middle-aged or adult animals, while diseased organisms are more susceptible to blood loss than healthy organisms. It is dangerous for the body when the body loses 60-70% of blood and 15-30% of blood loss when the body overheats causes death. Death occurs even if the body loses about 50% of its blood quickly and in a short time. If the amount of blood lost in the body does not exceed 25%, the blood pressure in the blood vessels decreases for a short time and immediately normalizes due to an increase in vascular tone by reflex and the release of stored blood into the blood vessels. If the body loses more than 25% of its blood, a long-term stable blood pressure drop occurs. When there is a lot of blood loss, the number of erythrocytes decreases, oxidation processes in the body are provided by oxygen transported by erythrocytes present in the body. A similar situation is observed when the blood is thinned (hydremia), that is, when interstitial fluid flows into the bloodstream. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases. If the total amount of blood is restored 3 days after blood loss, the shaped elements can be restored after 2-3 weeks. The recovery of the total amount of blood depends on the amount of blood lost from the body and the activity of the blood-forming organs. As the activity of blood-forming organs increases, the number of young erythrocytes, leukocytes and platelets in the blood increases.

Excessive blood loss leads to oxygen deficiency. When the nervous system is excited first, it then exhausts the centers that control respiratory and vascular tone by creating a wide-section braking. Cardiac function weakens, body temperature drops, and death occurs from paralysis of the respiratory center. Changes in body functions, hypovolemia or a decrease in total blood volume play a key role in lowering blood pressure. When blood is lost, it is important to put blood in the body, because if we put a saline solution at this time, the liquid part of the delivered solution passes from the blood vessel to the tissue.

8. In polycythemic hypovolemia, the total amount of blood decreases due to the liquid part of the blood, and the amount of erythrocytes increases per unit volume. In polycythemic hypervolemia, the absolute or absolute amount of erythrocytes is normal and the dry matter and viscosity of the blood increases. The decrease in the fluid content of the blood may be due to the body not consuming water. The strong viscosity of the blood prevents it from passing through the bloodstream, including through the capillaries.

9. In oligocytic hypovolemia, a decrease in total blood volume is associated with a decrease in erythrocytes in the blood. This type of hypovolemia can be observed in cases of excessive blood loss due to incomplete recovery of the fluid portion of the blood and some anemia and anemia.

Blood transfusion. When transfusing blood: a) lost blood - proteins, enzymes, hormones of the form elements of the blood are replaced, and the transfused blood participates in the performance of biological functions.

b) has a stimulating effect - that is, increases metabolism and blood formation.

c) increases blood clotting and stops bleeding.

g) cleanses the blood of toxins because erythrocytes and proteins in the transfused blood absorb toxins. Due to blood transfusion, blood pressure is restored, the body's stability is increased. It is used in cases of severe blood loss from burns, shock, collapse, diseases that reduce the reactivity of the organism, and general weight loss, because the blood affects various functions.

Until the twentieth century, blood transfusions were not widely used due to various tragic changes as a result of blood transfusions. The creation of the teachings of K. Landsteiner and Yansky on blood groups opened a wide way for blood transfusion.

The presence of blood groups is associated with antigenic causes in erythrocytes — isohemohagoglutinogen and antibody-isohemohaglutinins in serum. In determining blood groups, agglutinogen A and B in erythrocytes of blood are taken into account. These agglutinogens can occur in erythrocytes separately and both together or not at all. In accordance with these agglutinogens, agglutinins are also denoted by the Greek letters alpha and beta. An animal does not have similar agglutinogens and agglutinins.

Heterohemoagglutinins are also present in the blood at the same time as isohemoagglutinins.

Among the animals, the blood groups of horses are very clear, cattle, goats. in pigs and dogs, low levels of agglutinins in serum and low erythrocyte adhesion properties make it difficult to determine blood groups. Therefore, their blood will always need to be tested before a blood transfusion. To do this, take 2 drops of recipient serum on a vial, dilute 1 drop of donor blood 5 times in saline solution, and if agglutination does not occur within 10 minutes, this blood can be considered as recipient blood. If it does not resemble the recipient's blood, the donor solution will agglutinate. When solutions are gradually applied to the recipient, the agglutinating property is lost by repeatedly diluting with donor blood. Therefore, in practice, the focus is primarily on the donor agglutinogen and the recipient agglutinogen. If a large amount of blood is transfused, the recipient does not lose the agglutinating properties of the donor agglutinin and may cause shock in the body.

Hemotransfusion shock is a reaction that occurs when groups of blood are improperly placed in the body, and for the development of shock it is enough to put 80-120 ml of blood in groups that do not correspond to groups. As a result, the animal develops strong agitation, rapid breathing and heart rate - tachycardia. Decreased blood pressure makes breathing difficult, mucous membranes turn blue, vomit, urine and feces are no longer dependent on the activity of the organism. Shock often occurs within a short time, sometimes a few hours after a blood transfusion, and causes death. If the blood groups are not matched enough, the shock will pass immediately.

Some scientists explain that shock groups are formed by improper blood transfusion due to embolism of blood vessels in the brain, lungs, kidneys, while others explain that they are formed due to the breakdown products of erythrocytes in the recipient organism. Not all scientists agree with such analyzes. Experiments have shown that the mass formed by the adhesion of erythrocytes breaks down quickly without being stable and does not disrupt the activity of the organism. Even when hemolyzed blood is transfused, there is no shock in the animal's body. Academic AABogomolets binds to changes in the electric charge of colloidal substances during shock, as the colloidal structure of blood and tissue proteins plays a key role in the formation of hemotransfusion shock.

Due to improper blood transfusion, the structure of the recipient and donor proteins changes and the deposition of the protein micelles leads to a severe impairment of the body's function. This theory unilaterally explains the formation of shock.

In the pathogenesis of hemotransfusion shock is manifested as a major change in the reflex activity of the organism. When blood is burned in groups that do not match, it stimulates the vascular receptor to produce multiple impulses, creating a short-term strong excitation in the nervous system and then braking large parts. It therefore disrupts blood circulation, respiration, metabolism and other physiological functions.

Osmotic resistance of erythrocytes. EOR is the resistance of red blood cells to hypotonic solutions, and there is a difference between minimum and maximum resistance.

Minimum resistance is defined as the level of hypotension in which gamma-resistant erythrocytes break down and hemolyze. At maximum resistance, all erythrocytes are broken down, and the concentration of the saline solution is taken into account when assessing the degree of hypotension.

The resistance of erythrocytes depends on their structure, the resistance of erythrocytes in the changed form is low and hemolysis occurs. In addition, the resistance of erythrocytes to hypotonic solutions depends on the layer of lipoid protein formed on the erythrocytes. Due to the lack of lipids and phosphorus in the newly released erythrocytes, they break down earlier than the old erythrocytes. The state of maximum resistance indicates that the bulk of the erythrocytes are mature erythrocytes. An increase in EOR is observed in mechanical jaundice, in cases of poisoning with hemolytic toxins, in pathological conditions accompanied by tissue breakdown. Increased osmotic resistance of erythrocytes is also associated with the deposition of cholesterol and broken down tissue proteins in the body of erythrocytes.

Decreased EOR occurs when starving, in hemolytic jaundice, and in other diseased states of the organism.

Hemolysis is the rupture of red blood cells and the release of hemoglobin into the surrounding fluids. Blood or erythrocytes become discolored after hemolysis. Hemolysis occurs in and outside the blood vessel. Some erythrocytes also break down due to their own death. If in the physiological state erythrocytes are broken down by splenic macrophages, in pathological cases the breakdown of erythrocytes also involves the macrophages of the liver, red marrow and other organs.

Causes of hemolysis include:

1. Infusion of erythrocytes into hypotonic solutions.
2. Heating of blood or erythrocytes 62-630.
14. Re-freeze and thaw the blood.
15. The effect of rays.
16. The effect of electric current.

The hemolytic effect of light energy occurs in the presence of photosensitizers such as eosin, fluoroacin and others.

Hemolytic effect is manifested by chemicals such as nitrite, nitrobenzene, ether, benzene, case and deoxycholate acids, and others. Under the influence of chemicals, the erythrocyte membrane breaks down, disrupting the binding of hemoglobin to erythrocyte strain. Hemolysis-causing substances include bee venom, chaen snake venom, tetanolysin, staphylolysin, and many other microbial toxins. The hemolytic effect of toxins is based on the hydrolysis and softening of the erythrocyte shell by phospholipids. Erythrocytes are also broken down by blood parasites. Specific immunoassays to erythrocytes may be the effect of hemolysins as the cause of hemolysis. Sometimes substances in the blood serum that are formed under the influence of tumors, radiation and other diseases break down erythrocytes to form autohemolysins.

From the breakdown of erythrocytes in the bloodstream, hemoglobin dissolves in blood plasma and is excreted in the urine. In the gradual breakdown of erythrocytes, hemoglobin and erythrocyte fragments are captured by RES macrophages, resulting in complex changes to form the pigments bilirubin and hemosiderin.

Multiple breakdown of erythrocytes primarily increases the excretion of bilirubin by bile, which in turn increases stercobilin in the feces and excretes urobilinogen in the urine.

Iron released from erythrocyte breakdown is stored in liver and spleen macrophages. Here, after complex chemical changes, iron is released into the bloodstream and transported to the red marrow, where it is used in hemoglobin biosynthesis.

From the disruption of the normal change of hemoglobin, excess porphyrins-red violet-colored pigment is formed, which separates with the urine and turns the urine red. Due to the sensitization of porphyrin to light, its sensitivity to sunlight is increased. There are reports of parfirinuria as an

inherited disease in Shortgorn pedigree cattle. Parfirinuria also occurs when poisoned with mercury, lead and sulfonamides.

Anemia is a decrease in hemoglobin and erythrocytes per unit volume of blood. In anemia, erythrocytes undergo qualitative changes, pathological forms of erythrocytes are formed, which differ in size, shape, saturation with hemoglobin. The total amount of blood in anemia is either reduced or maintained at normal.

Classification of anemia. One of the most common classifications of anemias is to classify them according to their origin. Depending on the origin of anemia is divided into pasthemorrhagic, hemolytic, elemental and infectious types.

4. Posthemorrhagic anemia occurs when there is a lot of blood loss in the body. Acute posthemorrhagic anemia occurs as a result of sudden multiple or multiple - multiple chronic blood loss. Bleeding from blood vessels due to injury, ulceration of the intestines and stomach from internal organs, tuberculosis of the lungs, bleeding in the nasal cavity, tumor growth, bleeding as a result of childbirth, etc. is formed.

Restoration of the blood component after blood loss Normal red blood cell count is restored in a few days to 2-3 weeks, depending on the amount of blood lost by the body. Recovery of hemoglobin after extensive blood loss occurs gradually. In the blood, hypochromic erythrocytes are formed polychromatophils, reticulocytes and normocytes. The color of the blood decreases, the amount of leukocytes increases. Chronic diseases, changes in the quality of nutrition, reduce the regenerative properties of red blood cells and cause severe anemia. Decreased red marrow activity leads to anisocytosis and paikilocytosis, and sometimes to the formation of extramedullary blood in the spleen, liver, lymph nodes.

Hemolytic-toxic anemia is caused by toxins that break down erythrocytes. Some substances break down erythrocytes, directly in the blood vessels, some break down blood cells and then break down in RES macrophages. In the origin of toxic anemias, the formation of blood and the violation of the reflex control of its breakdown are of great importance. does not cause anemia when administered.

In hemolytic anemia, bilirubin in the blood increases, urobilinogen is excreted in the urine, and sometimes free hemoglobin is also excreted. First of all, the color of the blood is suddenly higher, and undigested erythrocytes are absorbed into the body, absorbed. Blood formation is enhanced by strong breakdown of erythrocytes. In the blood there are large numbers of polychromatophiles, reticulocytes and sometimes normoblasts. The color index of the blood suddenly decreases. Due to the good regenerative properties of red marrow, the composition of the blood is quickly restored with the loss of toxic effects. In chronic hemolytic anemia, the blood-forming organ becomes tired, its activity weakens, and erythrocytes with various defects in the blood fall into the bloodstream, and anisocytosis and poikilocytosis are observed. The amount of erythrocytes in the blood decreases sharply.

6. Alimentary anemia is caused by a lack of vitamins, proteins, trace elements in the diet, cobalt and copper, ie substances involved in the synthesis of hemoglobin. Alimentary anemia has a hypochromic character and the blood color index is less than one. Alimentary anemia is observed in young animals, especially piglets. Alimentary anemia is caused by inability to assimilate nutrients well during diseases of the gastrointestinal tract.

a). Anemia caused by iron deficiency is caused by a disorder of iron metabolism in the body. In this type of anemia, not only is there a decrease in erythrocytes, but also a decrease in the amount of hemoglobin. In severe anemias, anisocytosis and poikilocytosis occur. In pigs, iron deficiency in pigs resulted in the development of anemia in piglets at 1–6 weeks and up to 70% mortality.

b). Anemia caused by protein deficiency As a result of a lack of proteins in the diet or a decrease in their absorption, the synthesis of globulin protein is disrupted and hemoglobin is not formed.

4. Infectious anemia is caused by filtering viruses in horses and other ungulates. While some scientists explain the formation of this anemia as a direct breakdown of erythrocytes under the influence of viruses, others explain that the viruses are associated with causing red marrow hypofunction. The amount of erythrocytes in 1mm3 of blood of animals with infectious anemia is reduced by 1-2 million. Anisocytosis, poikilocytosis and other changes occur in the blood. In

infectious anemia in the red marrow occurs the replacement of the yellow marrow with red marrow, the formation of extramedullary blood in the spleen, liver, lymph nodes.

Regenerative and oregenerative anemia occur depending on the functional state of the blood-forming organ.

In regenerative anemia, the regenerative properties of the blood-forming organs are well manifested. As a sign of regenerative status in the peripheral blood are formed hypochromic, polychromatophilic erythrocytes, reticulocytes, erythrocyte nucleus remnants (Jolie bodies and Cape rings), normoblasts. When strong regenerative properties are manifested, the yellow marrow turns into red marrow, and in the liver spleen, extromedular blood formation occurs in the lymph nodes. Such changes disrupt blood formation and are formed from cells of the embryonic period — megoloblasts, macrocytes. Oxygen deficiency is an intermediate product formed during anemia, as a cause of regenerative processes in the blood-forming organs.

Aregenerative or hypoplastic anemia results from fatigue of the blood-forming feature of the red marrow. In hypoplastic anemia, the red marrow loses its ability to form erythrocytes, young erythrocytes in the blood decrease, the red marrow turns into yellow marrow, and has a hypochromic character. Weakening of the blood-forming organ is observed during avitaminosis, infections (tuberculosis, paratuberculosis, infectious anemia, sepsis), strong toxins, radiation sickness. Under certain conditions, any anemia can progress to a type of hypoplastic anemia. In most cases of anemia, erythropoiesis is not impaired, but leukopoiesis is also impaired.

In organisms, the compensatory mechanisms in anemia change. The function of oxygen supply to the blood is weakened, a number of flexibility mechanisms are formed: accelerated respiration, increased blood circulation and blood formation. As the heart beats faster, blood circulation speeds up and more blood flows through the capillaries over time. Accelerated and deepened respiration increases the saturation of the blood with oxygen in the lungs, increasing the formation of broken erythrocytes in the blood-forming organs. Compensatory properties are associated with the ability of tissues to fully absorb oxygen from arterial blood.

In severe hemoglobin deficiency in anemia, normal gas exchange is ensured in animals due to the activities of compensatory mechanisms. But weak movements during anemia cause a lot of oxygen demand, accelerated breathing movements, and tachycardias. Acedosis develops when there is an increase in incompletely broken down intermediates in the blood.

Polycythemia - or polyglobulia (Greek poly poly, globulus-ball, kutos-cell) is an increase in the number of erythrocytes in the blood per unit volume. Polycythemia is divided into absolute and relative types. In relative (false) polycythemia, the fluid content of the blood decreases and the number of erythrocytes does not change. This type of polycythemia occurs when sweating, severe diarrhea, diabetes mellitus, severe isthmus, dehydration and other pathological processes. In relative polycythemia, the total amount of blood is often reduced or unchanged.

In absolute polycythemia, erythrocytes proliferate due to increased erythropoiesis. In most cases, absolute polycythemia serves as a resilience reaction in the absence of oxygen to the body. Lack of oxygen increases the flow of erythrocytes from blood depots and blood-forming organs into the bloodstream. Absolute polycythemia develops when external respiration is disrupted (pulmonary emphysema, when the upper airway narrows, O2 partial pressure decreases in atmospheric air), when blood circulation is disrupted. Polycythemia also occurs when poisoned with copper, phosphorus, cobalt, arsenic. Polycythemia is a physiological condition in newborns, ie in the first days of life of calves erythrocytes in 1 mm3 of blood are 10.5 million. and a month later it dropped to 7.5 million.

Changes in white blood cells. Leukocytes, i.e. white blood cells, are formed in the red marrow, lymph nodes, and spleen. The stem cells that produce leukocytes are called hemocytoblasts, and the hemocytoblasts form myeloblasts, the primary cell of granular leukocytes in the red marrow. Lymphoblasts and monocytes are produced in the lymph node and spleen. In the blood of a healthy animal, there are many joint nuclei, and a small number of rod nuclei are found. Young neutrophils are not always present, and when blood-forming organs are tickled, rod nuclei proliferate, and in some cases myelocytes also occur.

Leukocytes include plasma cells, i.e., lymph nodes, spleen, and products of reticular and endothelial cells of the red marrow. Immune cells are formed due to the activity of plasma cells. During normal blood formation, plasma cells are found in the blood-forming organs, while in healthy animals, they are almost never found in the peripheral blood. The cytoplasm of plasma cells is stained dark orange, and the nucleus is round or oval in shape.

A leukoformula is a list of leukocyte types to determine the percentage of individual leukocyte species. In the leukoform of cattle, sheep and pigs, lymphocytes are abundant in the blood of horses, dogs and cats, and neutrophil leukocytes are abundant. White blood cells differ in type, and the leukoforms of young organisms are slightly different from those of older animals.

In determining the functional status of blood-forming organs, it is necessary to know not only the amount of leukocyte-forming organs, but also the absolute amount of leukocytes. The determination of the ratio of the main group of leukocytes in numbers is called leukocytic profile.

The main function of leukocytes is a protective function, i.e. phagocytosis. Leukocytes play an important role in the repair of damaged tissue, clearing the injured area of necrotic cells. Leukocytes produce a substance that stimulates regeneration, basophils and eosinophils are involved in neutralizing toxins. Quantitative changes in leukocytes are caused by an increase or decrease in leukopoiesis, as well as redistribution of blood in the blood vessels. As a result of dilation of blood vessels, blood flow slows down, leukocytes settle along the walls of blood vessels, and their amount in these blood vessels increases. Where blood vessels constrict and as a result blood flow accelerates, the amount of leukocytes in the blood decreases.

4.Myeloid, lymphoid leukemia and reticuloendotheliosis are distinguished depending on which part of the hematopoietic system is hyperplastic. Lymphoid leukemia is found in cattle, horses, and pigs, while myeloid leukemia is observed in dogs.

Myeloid leukemia or myelosis is characterized by hyperplasia of myeloid tissue. The yellow marrow turns into a red marrow, causing extromedular blood to form in the spleen, lymph nodes, liver, and sometimes other organs. Leukoblasts are more common in erythroblasts than erythroblasts. Myeloid leukemia is divided into leukemic and aleichemic types. In leukemic myelosis, the number of leukocytes in 1 mm3 of blood can be a hundred thousand or more. The main part of leukocytes, ie 90% and more, are granulocytes. The bulk of granulocytes are young cells, ie myelocytes, promyelocytes and myoblasts, and sometimes the number of unexposed eosinophils, basophils and erythroblasts also increases. In aleukemic leukemia, the number of leukocytes is increased around the norm and or in very small amounts. Examination of the leukoformula shows a strong rejuvenation of leukocytes. However, although their phagocytic properties are preserved, they are slightly lower than the phagocytic activity of mature neutrophils. In myeloid leukemia, the spleen becomes enlarged.

Some scientists attribute the formation of extromedular blood in leukemia to the introduction of myeloid cells into tissues and the formation of metastases, while others explain that the formation of extromedular blood is caused by the influence of etiological causes of leukemia on mesenchymal cells.

There are leukemic and aleukemic types of myeloid leukemia. In leukemic leukemia, the number of leukocytes in 1mm3 of blood reaches 100,000. The main part of leukocytes is granulocytes, which account for 90%. Granulocytes are composed of young cells - myelocytes, promyelocytes, sometimes non-myeloblastic eosinophils, basophils, erythroblasts. In aleukemic myelosis, the leukocytes in the blood increase normally or very little. In leukoform, young cells are weaker than phagocytosis in neutrophils, whose main part is phagocytic function (myelocytes, etc.).

During lymphoid leukemia or lymphodenosis, lymphoid tissue grows and is characterized by enlargement of the lymph nodes, spleen and liver. As leukemia develops, the myeloid tissue is replaced by lymphoid tissue in the red marrow. During leukemic lymphodenosis, the amount of white blood cells in 1 mm3 of blood reaches 1.5 million, and lymphocytes make up 98% of all leukocytes. In aleukemic lymphodenosis, the number of leukocytes is normal or partially increased, lymphocytosis develops in the leukocyte formula, and lymphoblasts are also found among the lymphocytes.

Reticuloendotheliosis is characterized by proliferation of reticular cells in the red bone marrow, spleen, lymph nodes, and liver. There are leukemic and aleukemic types of reticuloendotheliosis. In leukemic retiuloendotheliosis, there is a strong increase in monocytes in the blood. In acute leukemia

the metabolism is disturbed, the productivity of the animals decreases, anemia develops and severe weight loss occurs, in chronic leukemia the animal seems to be healthy for a long time, the animal dies from malnutrition and other diseases.

Leukemia etiopathogenesis. At present, leukemia with all its symptoms is recognized as a pathological process specific to the inflammatory process. Symptoms related to the theory of blastomatosis of leukemia include:

1. The growth of hematopoietic tissue during leukemia is not differentiated like tumor cells.
2. Changes in metabolism during leukemia are similar to those in malignant tumors.
3. Carcinogens have leukogenic properties in the experiment.
4. The therapeutic effect is due to the same substances in leukemia and tumors. (M. X-rays, radioactive phosphorus, chemicals that affect cells).

In leukemia, the leukocytes are in such an atypical state that it is difficult to consider them as this or that blood-forming element. However, the process of phagocytosis is worse than in normal leukocytes. Leukemia differs from normal tumors in the formation and growth of blood in the blood-forming organs. In aleukemic leukemia, destructive symptoms characteristic of the growth of all tumors are observed.

The causes of leukemia and tumor formation are not yet fully understood. Chicken leukemia is caused by viruses. This has been studied in leukemia by sending cell-free filtrate to healthy chickens. All leukemias can be formed by injecting carcinogens. Leukemia is caused by long-term ionizing radiation in the body, the mechanism of action of which has not yet been determined.

Changes in blood plastics. Blood plastics play an important role in platelet coagulation and are a source of the enzyme thrombocytosis. Platelets are formed in large cells of the red marrow - megakaryocytes. Therefore, the factors that affect the red marrow affect the amount of blood platelets. A decrease in the amount of platelets in the blood is called thrombopenia, which causes a weakening of the blood clotting process. In thrombopenia, the retraction of the blood clot is weakened The blood clot is soft and does not provide a tight closure of the injured blood vessel.

The causes of thrombopenia are as follows:

1. Redistribution of platelets, ie accumulation of platelets in the blood vessels of the internal organs and a decrease in the peripheral blood vessels.
2. Weakening of platelet formation in the red marrow.
3. Strong breakdown of platelets in peripheral blood.

Thrombopenia in some infectious diseases is caused by physical, chemical causes, disruption of the activity of blood-forming organs or strong breakdown of platelets.

When thrombocytosis or an increase in the amount of platelets in the blood is cured of many infectious diseases, in myeloid leukemia, anemia is formed during the recovery of blood composition, and blood clotting is enhanced.

Simultaneously with the change in the number of platelets, a qualitative change occurs, the shape changes, does not wrinkle and undergoes other changes. The agglutination property of such blood plastics is lost, and blood flow and blood clot retraction are impaired.

Changes in blood coagulation. Blood coagulation is recognized as a three-phase process as explained on the basis of modern theories. The first phase is a complex biochemical process in which active thrombokinase is formed from active tissue thromboplastins and the action of blood platelets on serum proteins. From the inactive prothrombin enzyme in the second phase: active thrombin is formed in the blood plasma. Calcium ion, active thrombokinase and plasma protein - globulin accelerator are involved in the activation of prothrombin. Prothrombin is formed in the liver in the presence of vitamin K. The liver is one of the main sites where fibrinogen is synthesized. In the third phase, fibrin is formed from the action of active thrombin on fibrinogen. As a result, fibrin filaments are formed and blood clots form. In the body, along with the blood coagulation system, there is also an anti-coagulation system, these substances are formed in the tissues and released into the blood under the control of the nervous system. Anti-coagulation systems include 1) heparin-liver physiologically active substance formed in the lungs and blood vessels, 2) fibrinolysin-plasmin, 3) protein substances that inhibit the formation of thrombin and thromboplastin. Heparin activates the lipase of lipoproteins that

are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin. Heparin activates the lipase of lipoproteins that are part of thromboplastins. Fibrinolysin is formed from plasminogen, which is released from tissues into the blood. Under the influence of fibrinolysis, fibrinogen is hydrolytically broken down into fibrin.

The blood coagulation and anticoagulant system are two interconnected parts of the blood's coagulation system. Because these two systems are mutually balanced, the blood moves in a fluid state without clotting in the blood vessels.

Weakening of blood clotting. Weakening of blood clotting: 1) due to insufficient intake of vitamin K in the body or impaired synthesis of prothrombin and fibrinogens in pathological processes of the liver. 2) when there is a decrease in platelets in the blood - in thrombocytopenia. 3) decrease in calcium ions in the blood. 4) excessive development of the anticoagulant system in the body - heparin and others. 5) when anti-coagulants, ie substances that weaken blood clotting, are injected into the body.

When animal blood has a low coagulation property, a small mechanical injury can cause bleeding into the subcutaneous tissue, mucous membranes, muscles, and other tissues. The easiest bleeding occurs in the nose, lungs, intestines.

By treating the blood vessels with paraffin, if blood collects in the arteries, the blood becomes coagulated. A 5% sodium hydroxide solution of citric acid is often used to make the blood non-coagulating. Anticoagulants include dicoumarin and other anticoagulants extracted from the head of the leech. These substances stabilize the blood by inactivating thrombin. We can use the stabilizing properties of these substances by injecting them directly into the body or adding them to freshly drawn blood.

Acceleration of blood clotting. Accelerated blood clotting is associated with vascular injury. Blood platelets easily sink into the injured vascular wall, break down due to low resistance, and form active thromboplastin-thrombokinase. Blood coagulation can be formed by the strong breakdown of tissues by sending to the body extracts prepared from blood serum and organs. Increased blood coagulation after excessive blood loss is associated with the influx of many interstitial fluids rich in thromboplastin factor into the blood. Based on this mechanism, the delivery of calcium salts, multi-vitamin K, when hypertonic solutions are injected into the blood, increases blood coagulation. Increased blood clotting in the body can lead to thrombosis and embolism.

Changes in the biochemical composition of the blood. Minerals are ionized in the blood and are in a molecular state as well as in a state of binding to proteins from colloidal substances. Minerals are involved in blood osmotic pressure and other complex physicochemical processes. Minerals are not evenly distributed between the blood plasma and trace elements, the amount of calcium, potassium, sodium and other minerals in the blood of healthy animals is always kept the same, even when saline solutions are sent to the body.

Calcium. Ionized calcium is physiologically active, accounting for 45-55% of total calcium. Combined with non-ionized calcium mining proteins. The amount of calcium in the blood depends on the functional state of the autonomic nervous system. Calcium decreases when sympathetic nerve tone decreases, and calcium increases in blood when parasympathetic nerve tone decreases. Calcium salts thicken cell and tissue membranes.

A sharp decrease in calcium levels is caused by a deficiency of glands near the thyroid gland and causes hypoproteinemia due to the fact that part of the calcium is bound to proteins. The amount of calcium in the blood is reduced in nephritic anemia congenital paresis. Decreased calcium intake increases vascular permeability, excitability of the CNS and peripheral nervous system. Calcium intake is also caused by impaired intestinal absorption in chronic diarrhea.

Potassium. In many animals, the amount of potassium in erythrocytes is higher than in plasma, and the amount of potassium in plasma increases when erythrocytes break down. Damage to erythrocytes

causes the release of potassium from erythrocytes into plasma due to increased permeability without breaking them down. The amount of potassium in the serum increases in severe diseases when the tone of the parasympathetic nervous system increases, regardless of its nature. Potassium and calcium affect the excitability of the nervous system. Deficiency of potassium in the body leads to weakening of muscle activity.

Sodium. Occurs in the blood plasma mainly in the form of chlorides, partly biocarbonate and other salts. Chlorides are reduced in the blood when sweating, diarrhea, vomiting, weight loss, impaired intestinal permeability, kidney disease. Decreased chlorides affect osmotic pressure and increase the breakdown of tissue proteins, weakening the activity of the adrenal cortex. The amount of chlorides increases in the blood during kidney disease, ie nephritis. The onset of hyperchloremia is caused by increased pulmonary ventilation, as a result of which chlorine ions pass from the tissues into the blood.

Phosphorusoccurs in the form of organic and inorganic compounds. In animals, inorganic phosphorus in the blood is reduced in pregnancy, rickets and osteomalacia. Hyperphosphatemia is caused by fever, lack of oxygen, uremia, exposure to vitamin D and ultraviolet light, as well as a lack of glands under the thyroid gland.

Ironenters hemoglobin and occurs in the form of other compounds only in 2% of cases. Therefore, iron varies depending on the amount of hemoglobin. In anemia, iron in the blood is reduced. Blood contains trace elements such as iodine, bromine, fluorine, magnesium, copper, manganese and others. The amount of micronutrients in the blood is affected by the nervous and endocrine systems. Detection of micronutrients in the blood is important in the diagnosis of metabolic diseases.

Proteins and products of protein metabolism. Protein and its fractions are different in the blood of different animals. Some proteins combine with fats and carbohydrates to form double compounds - lipoproteins or glycoproteins. Although many proteins (e.g. enzymes) are present in very small amounts in the blood, they have very important physiological activity. Most of the blood plasma proteins are synthesized in the liver. Decreased total protein in the blood (hypoproteinemia) is caused by eating disorders (malnutrition, protein starvation). Causes of hypoproteinemia include urinary excretion of proteins, liver toxicity, excessive blood loss, severe degenerative diseases of animals (tuberculosis, malignant tumors, chronic purulent processes, etc.). In hypoproteinemia, mainly albumin function is reduced, while the globulin fraction is significantly reduced. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. albumin function decreases, while the globulin fraction decreases insignificantly. Hypoproteinemia causes blood thinning (hydremia) and a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. leads to a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such as severe burns of the body, as well as other types of pathological processes that cause dehydration. In such cases, all fractions of proteins increase equally. In most cases, an increase in individual fractions is observed, and sometimes, only the amount of fibrinogen in the blood increases. leads to a decrease in colloid-osmotic pressure in the blood. An increase in protein in the blood plasma (hyperproteinemia) often occurs in blood clots, such

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Blood plasma increases globulins in infectious disease and starvation. After immunization, gamma globulins in the blood increase sharply. However, an increase in gamma globulins is not associated with an increase in antibody levels. An increase in non-specific gamma globulins in the blood and an increase in gamma globulins may be due to a decrease in specific antibodies, as AE Gurvich found. Decreased albumin fraction in the blood is observed in hepatitis and cirrhosis. Therefore, in patients with impaired liver function, the total amount of proteins in the blood plasma and some fractions are variable.

Residual nitrogen in the blood is the protein-free nitrogen of the blood or the nitrogenous substances that remain after the deposition of proteins in the blood is 20-40 mg%. Increased residual nitrogen in the blood (azotemia) is observed in disorders of renal, hepatic and intestinal permeability. The amount of residual nitrogen in the blood is 200 mg% and more when the renal excretory function is impaired. In azotemia associated with renal (retention) activity, an increase in the amount of residual nitrogen occurs due to urea.

In cachexia, leukemia, and infectious diseases, the accumulation of large amounts of nitrogen-fixing substances in the blood due to the breakdown of tissue proteins causes azotemia. In hepatitis, azotemia is caused by polypeptides, which can also lead to a decrease in the amount of urea in the blood. Such a change is observed in liver disease when the deamination of amino acids is impaired, the synthesis of urea is weakened, and the transfer of ammonia salts into the blood is increased.

Accumulation of uric acid in the blood is observed in disorders of purine metabolism, gout, diseases associated with tissue breakdown, and leukemia.

There are a certain amount of free amino acids in the blood, which are intermediate products of protein metabolism. An increase in the amount of free amino acids is caused by liver disease, ie severe atrophy, poisoning by carbon tetrachloride.

Carbohydrates and products of carbohydrate metabolism.

Blood contains products of glucose, glycogen, lactic acid and other carbohydrate metabolism. The amount of glucose in the erythrocytes of most species is lower than in plasma, and this is more pronounced in pigs. Most of the glycogen is found in leukocytes. An increase in the amount of glucose in the blood (hyperglycemia) occurs when consuming easily digestible carbohydrate foods (elemental hyperglycemia), when the regulation of carbohydrates through the nervous and endocrine systems is impaired. Hyperglycemia occurs when poisoned with physostigmine, pilocarpine and other substances that affect the nervous system. The origin of hyperglycemia is in the pathology of the endocrine system, ie in the hypofunction of the islets of the pancreas Langerhans, formed in inflammation and dystrophic changes of the liver. Decreased blood glucose (hypoglycemia) is observed in chronic insufficiency of nutrition, excessive infusion or delivery of insulin into the blood, hypofunction of the adrenal hypo-thyroid gland. The manifestation of severe hypoglycemia is observed in patients with chronic cachexia.

An increase in the amount of lactic acid in the blood is observed in muscle work and pathological processes in the disruption of oxidative processes in the body, when there is a lot of blood loss, pulmonary edema, suffocation, the formation of malignant tumors. All the factors that increase the formation of lactic acid in the blood cause an increase in the amount of pyruvic acid in the blood.

Learning materials for independent study

Topic: Physiology of the immune system

Plan:

1. The concept of immunity.
2. Importance of immunity for the organism.
3. Types of immunity.
4. Organism and environment.
5. Analysis and conclusions of the data obtained.

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3. Information on scientific articles on the subject.
4. Internet information:
   www.Ziyonet.uz
   www.vetjurnal.uz
   www.goldenpages.uz
   www.zootehniya.ru

Topic: Cardiovascular physiology of birds.

Plan:

1. The concept of the structure of the heart in birds.
2. Specific features of the circulatory system in birds.
3. Peculiarities of blood circulation in birds.
4. Management of blood circulation in birds.
5. Analysis and conclusions of the data obtained

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.

5. Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Specific features of respiration in birds.

Plan:

1. The concept of the respiratory organs of birds.
2. Specific features of the respiratory system in birds.
3. Specific features of respiration in birds.
4. Control of respiration in birds.
5. Analysis and conclusions of the data obtained.

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3. Information on scientific articles on the subject.
4. Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Features of digestion in the stomach of horses and pigs.

Plan:

1. The concept of the digestive organs of horses and pigs.
2. Specific features of the digestive organs in horses and pigs.
3. Features of digestion in the stomach of various farm animals.
4. Management of syrup secretion in the stomach of horses.
5. Analysis and conclusions of the data obtained.

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3. Information on scientific articles on the subject.
4. Internet information:
   www.Ziyonet.uz
   www.vetjurnal.uz
   www.goldenpages.uz
   www.zootehniya.ru

Topic: Peculiarities of digestion in birds.

Plan:

1. The concept of the digestive organs of birds.
2. Peculiarities of digestive organs in birds.
3. Peculiarities of digestion in poultry.
4. Management of digestion in poultry.
5. Analysis and conclusions of the obtained data.

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.
5. Internet information:

www.Ziyonet.uz

www.vetjurnal.uz

www.goldenpages.uz

www.zootehniya.ru

Topic: Physiology of macro and micro elements.

Plan:

1. The importance of minerals for the animal body.
2. The importance of macronutrients (sodium, potassium, calcium, chlorine, sulfur, phosphorus, magnesium) for the animal body.
3. The importance of trace elements (iron, copper, cobalt, iodine, manganese, zinc, strontium, cesium, bromine, fluorine) for the animal body.
4. Management of mineral metabolism.
5. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.
5.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Physiology of water and fat soluble vitamins

Plan:

1. The importance of vitamins for the animal body.
2. Physiology of water-soluble vitamins (B1, B2, B3, B6, B12, B15, Bt, PP, N, S, rutin, inositol, choline, paraaminobenzoic acid, folic acid, lipoic acid).
3. Physiology of fat-soluble vitamins (A, D, E, K).
4. Avitaminosis, hypovitaminosis, and polyavitaminosis.
5. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.
5.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Urine excretion in birds.

Plan:

1. The concept of the digestive organs of birds.
2. Urine excretion in birds.
3. Peculiarities of urinary excretion in birds.
4. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.
5.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Blood circulation in the fetus.

Plan:

1. The concept of blood circulation in the fetus.
2. Peculiarities of blood circulation in the fetus
3. Analysis and conclusions of the obtained data.

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.
5. Internet information:

www.Ziyonet.uz

www.vetjurnal.uz

www.goldenpages.uz

www.zootehniya.ru

Topic: Reproductive physiology of birds.

Plan:

1. The concept of the reproductive organs of birds.
2. The period of sexual activity of birds.
3. Composition and weight of poultry eggs.
4. Egg laying, egg laying cycle and its management.
5. Analysis and conclusions of the obtained data.

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.
5. Internet information:

www.Ziyonet.uz

www.vetjurnal.uz

www.goldenpages.uz www.zootehniya.ru

Topic: Physiological basis of machine milking

Plan:

1. A general understanding of the physiological basis of machine milking.
2. The importance of machine milking for the body.
3. Physiological changes observed during machine milking. .....
4. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
4. Information on scientific articles on the subject.
5.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Animal feeding and care
The effect on the amount and composition of milk.

Plan:

1. General concept of animal feeding and care.
2. For the organism of feeding and caring for animals importance
3. The effect of animal nutrition on milk quantity and composition.
4. Influence of animal care on milk quantity and composition
5. Analysis and conclusions of the obtained data.

List of used literature:

1. RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2. V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3. Information on scientific articles on the subject.
4. Internet information:

www.Ziyonet.uz

www.vetjurnal.uz

www.goldenpages.uz

www.zootehniya.ru

Topic: Physiology of endocrine glands

Plan:

1. The concept of endocrine and exocrine glands and hormones.
2. The relationship of the activity of the endocrine glands with the nervous system.
3. Methods of studying the activity of endocrine glands
4. The activity of the endocrine glands and its management:
- thyroid gland
- parathyroid glands
- adrenal glands
- pancreas
- pituitary gland
- Gonads and placenta
- epiphyseal gland
- thymus gland.

5. Internal secretory activity of other bodies.
6. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3. Information on scientific articles on the subject.
4.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Ethology. Behavior of farm animals and its characteristics.

Plan:

1. The concept of ethology.
2. Behavior of animals and their types.
3. The role of sensory organs in ethology.
4. The importance of the method of conditioned reflexes in the study of animal behavior.
5. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3. Information on scientific articles on the subject.
4.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootehniya.ru

Topic: Sleep and hypnosis.

Plan:

1. The concept of sleep.
2. The importance of sleep for the body.
3. Types of sleep.
4. The concept of hypnosis and its importance for the body.
5. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3.VIGeorgievskiy. "Physiology of agricultural animals". Moscow, Agropromizdat - 1990.
4. Information on scientific articles on the subject.
5.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
www.goldenpages.uz
www.zootechniya.ru

Topic: Alarm systems

Plan:

1. The concept of signal systems.
2. The importance of signaling systems.
3. The first alarm systems.
4. Secondary alarm systems.
5. Analysis and conclusions of the obtained data.

List of used literature:

1.RXKhaitov, BZZaripov, ZTRajamurodov "Animal physiology". Tashkent "Teacher" - 2005.
2.V.Husainova, E.Toshpulatov. "Physiology of farm animals." Tashkent "Uzbekistan" - 1994.
3.VIGeorgievskiy. "Physiology of agricultural animals". Moscow, Agropromizdat - 1990.
4. Information on scientific articles on the subject.
5.Internet information:
www.Ziyonet.uz
www.vetjurnal.uz
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Topic: Physiology of analyzers

Plan:

1. The importance of analyzers (sensory organs) for the body.
2. General features of analyzers (sensory organs).
3. Physiology of vision, hearing, body balance, skin, sense of smell, taste, internal and musculoskeletal or motion analyzers.
.4Interaction of analyzers (sensory organs).
5. Analysis and conclusions of the obtained data.

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Glossary (in Uzbek, English)

O‘zbek tili	O‘zbekcha ma’nosi	Ingliz tili	Ingliz ma’nosi
	Kirish		
Patologiya	Kasallik sababi, rivojlanishi mexanizmi, oqibati	pathophysiology	The mechanism of the development of the cause of the disease, the consequences
	Patologik fiziologik fanining tekshirish usullari		
Viviseksiya	Tabiy sharoitda juda qisqa vaqitda, kupincha, organizm funk-siyalarini o‘rganish	Vivisection	Natural environment, in a very short exponents of the body often funk-budsman
Fistulalar urnatish	Ko‘pincha katta qorin, me’da, ichak, so‘lak bezlari, oshqoz-onosti bezi chiqaruv yo‘ll-ariga va boshqa-larga fistula qo‘yish	the installation of the fistula	Most of the big belly, stomach, intestine, salivary glands, pancreas onos through the bee and you have to put fistula
Angiostomiya	Organga oqib kirgan va undan chiqayotgan qon tarkibini o‘rganish	angiostomy	The study of blood flow into and out of the body
Radioizotopli skanerlash	Radioaktiv atomga ega maxsus nishonlangan moddalardan foydalanish	radioisotope scan	Substances labeled with a radioactive atom
Steriotaktik	Oliy nerv faoliyatini o‘rganish	Steriotaktik	Study of the higher nervous activity of
Laporoskopiya	Ichki organlar-ning (oshqozon, ichak, jigar, o‘t xaltasi) shakli, rangini ko‘z bilan kuzatib o‘rganish	laparoscope	Internal organs (stomach, colon, liver, gall bladder), with a keen eye color
Biokimyoviy tekshirish	Kasal hayvon organizmidagi almashinish jarayonlarining ko‘rsatkichlari (protrombin faqat jigarda ishlaganligi tufayli uning qondagi miqdori) aniqlash	To check the biochemical	Animal indicators of metabolic processes in the body of the patient (PT only in the liver due to its amount in the blood)
	Patologiyaning rivojlanish davirlari xaqida qisqacha ma’lumot		
Gumoral		humoral	
Kpazis	Shiralarning nisbatan to‘g‘ri aralashishi sog‘lomlikni bildirib	Kpazis	Chirac said the right to intervene in health
Diskpazis	Organizimga tashqi muxit ta’sirida shiralarning ifloslanishi	Diskpazis	Contamination of the juice of the influence of the external environment of the organism
	Nazologiya		
Nazologiya	Kasallik xaqida umumiy ta’limot		Teaching about the disease
Sog‘lomlik	Tabiy tarizda yoki inson tomo-nidan yaratilgan yashash sharoitl-ariga javoban o‘zidan kutilayo-tgan maxsulotni optimal miqdor-larda beradigan hayvonni sog‘lom hayvon deb xisoblash	nosologic health	Natural or tarizda Human side to meet living conditions in response to the bee products itself kutilayo passing calculate the optimal amount of animal health and animal

			gods
Kasallik	Bemorlik	disease	of patients
Patologik jarayon	Patogen omil ta’sirida molek-ulalar, xo‘jayra va to‘qimalarda organizmning javob reaksiyasi bilan birgalikda amalga oshadigan strukturaviy va funksional o‘zgarishi	pathological process	Mole-ulalar the influence of the pathogenic factor, together with the cell and tissues in the body's response is to be implemented structural and functional changes
Patologik xolat	Strkuturaviy o‘zgarishlar bilan xaraktirlanadigan doimiy tarizdagi me’yoriy chetga chiqishdir	pathological conditions	Strkuturaviy permanent changes xaraktirlanadigan tarizdagi standard deviations

Kasallik kechishi va rivojlanish davirlari

Kasallikning latent yashirin davri	Kasallik chaqiruvchi sabab ta’sir eta boshlagan paytdan boshlab, kasallikning dastlabki klinik belgilari paydo bo‘la boshlagan paytgacha davom etishi	Hidden latent period of the disease,	Since begun to affect the cause of the disease calling for the start of the first clinical signs of the disease can continue
Inkubatsiya davri	Mikroorganizm yoki parazit bo‘lsa yuqumli yoki invazion kasallikning bu davri deb yuritilishi	Inkubattsiya period	Micro-organisms or parasites, infectious or parasitic disease in this period that the
Kasallikning prodromal	Kasallikdan darak beruvchi	prodromal illness	Indication of the disease
Prodrom	Nishona	Prodrom	a sign
Sog‘ayish sanogenizi	Kasal hayvon organizmi buzilgan funksiyalarining tiklanib, uni yashayotgan muhitga moslashib ish qobiliyati, maxsuldorligini tiklanishi	healing sanogenizi	Sick animal functions of the body to repair, and restoration of the ability to adapt to the living environment, business productivity
O‘lim	Organizm xayot faoliyatining to‘xtashi	death	Termination of the life of the organism
Tantogeniz	Organizmga kasallik chaqiruvchi sabab tushgan zaxotiyooq kompensator mexanizmlar ishga tushishi	Tantogenisis	The launch of an organism called zaxotiyooq compensatory mechanisms that cause the disease
Agoniya	Kurash	Agony	struggle
Mors perasfixiam	Nafas olishning tuxtashi bilan boshlanadigan o‘lim	Morse perasfixiam	breathing stopped, starting with the death
Mors persinkopen	YUrak urushining tuxtashi bilan boshlanadigan o‘lim.	Morse persinkopen	Heart stopped, starting with the death.
Mors klinikalis	Klinik o‘lim.	Morse klinikalis	Clinical death.
Algar mortis	Gavda sovishi	Algar mortis	cools body
Regor mortis	Gavda qotishi	Rego mortis	body hardening
Livaris mortis	Gavdada dog‘lar paydo bo‘lishi	Livaris mortis	His body, which can be
Masirati yoki autoliz	Gavdaning chirishi	Masirah or autoliz	decomposing body
Anabiosis	Uyqusimon	Anabiosis	sleep

Umumiy etiologiya

Etiologiya	Kasallikning sabablari va kelib	etiology	Science about the causes

	chiqish shart sharoitlari xaqidagi fan		and circumstances of the origin of the disease
Monokauzalizim	Etiologiyadagi shunday yo‘nalishki, bunga ko‘ra xar bir kasallik faqat bittagina sabab tomonidan chiqariladi	Monokauzalizim	According to the direction of the etiology of each disease is only one caused by the
Konditsionalizim	Kasalliklar sabablarini izoxlashdagi navbatdagi yangi yo‘nalish.	Kondittsionalizim	Because of izoxlashdagi new direction.
Konstitutiya			

Tashqi muxit omillarining patogen ta’siri

Biosopromat	Veterinariya tibbiyotining tirik tuqima bilan mexanik bosim o‘rtasidagi o‘zaro munosabatl-arini o‘rganuvchi bo‘limi	Biosopromat	Veterinary medicine foolish living with the study of the relationship between the mechanical pressure grease
Termik effekt	Lazer nuri yutilgan paytda energiyaning asosiy qismi	thermal effect	The main part of the energy when the laser route
Rezonanis effekti	Biomolekula murakkab tebranish tizimini anglatishi	the effect of Rezonanis	Biomolekula involves a complex vibration system
Stimullash effekti	Geliyali-neonli lazerning qizil nuri ta’sirida nerv retseptorlari	stimulatory effect	Helium-neon laser red light sensitive nerves rettseptorlari
Surunkali nurlanish kasalligi	O‘tkr zaxarlanish kasalligi	Chronic radiation disease	Acute poisoning disease
Autointoksikatsiya	Endotoksinlar bilan organizmlarning zaxarlanishi	Autointoksikattsiya	Endotoxin poisoning organisms
Rezorbsion autointoksikatsiya	Zaxarli moddalarni nekroz, yringli bo‘shliqlari	Rezorbtsion autointoksikatsiya	Poisonous substances necrosis cavities yringli
Metobolitik autointoksikatsiya	Turli kasalliklar davrida moddalar almashinuvining bo‘zilishi	metabolic autointoksikatsiya	A variety of diseases, metabolic deterioration during
Ximoya	Kompensator jarayonlar	protection	compensatory processes
Kompensare	Tenglashtirish, o‘rnini to‘ldirish	Kompensare	Matching, fill the

Umumiy patogeniz

Albinizim	Ko‘z ter va jun qoplama pigmenti melaninning to‘liq va qisman mavjud bo‘lmas-ligi bilan namoyon buladigan irsiy patologiya	Eye and skin and hair pigment melanin skin components are stained with immediately genetic pathology	albinizm
Orqa chiqaruv teshigining atreziyasi	Teshikning bo‘lmasligi	holes	Atreziyasi anus
Gernatologiya	Organizmning qarshi tabiy jarayon bo‘lib ushbu jarayon	The body's natural process	Gernatologiya

	bilan bog‘liq bo‘lgan o‘zgarishlarni o‘rganadigan fan	associated with this process changes the science	
Geriatriya	Kasalliklarni o‘rganadigan davolaydigan va ularning oldini olish bilan shug‘illanadigan fan	Learn science involved with the treatment and prevention of diseases	Geriatrics
Geron	Kasallik	disease	Heron
Latria	Davolash	cure	Latria
	Organizimning reaktevligi va uning patologiyadagi axamiyati		
Reaksion	Qarshi ta’sir	reactionary	opposite effect
Labillik	To‘qimalarning funksional xarakatchanligi	can be used	Functional mobility of tissues
Xronaksiya	Elektr tokining yoki nerv impulislari	Xronaktsiya	Electric shock or nervous impulislari
Sezuvchanlik	Adekvat ta’sirotning minimal kuchida sezgi organlarining qo‘zg‘aalgan xolatga kelish qobiliyati	sensitivity	The power of the minimum adequate reaction of sensory qo'zg'aalgan position
Xuper	YUqori ortiqcha	Marayati	plus
Ergon	Ta’sir	ergo	effect
Dizergiya	Sovuq va bazi bir dorivor moddalar ta’sirida kasallikning atipik kechishi ya’ni tomirlarning kengayishi va teri ajiralishi	Dizergiya	Cold and disease under the influence of some medicinal substances atypically that the expansion of the blood vessels and skin divorce
Nofaol rezistentlik	Ximoya mexanizmlarning faol funksiya bajarishi bilan bog‘liq bo‘lmasdan barer sistemalar	Inactive resistance	Protection mechanisms related to the performance of the active function barrier systems
Xellikobakter pyuulori	Qo‘zg‘atuvchilar	Xellikobakter pyuulori	pathogens
Fagatsitoz	Organizm nospetsifik ximoyalanish markaziy bo‘g‘imi ikki sistema xujayralari	Fagattsitoz	Non-Defense cells in the central system of the two joints of the body
Polimorf yadiroli lekotsitlar sistemasi	Polimorf yadroli lekotsitlar	Polymorphic yadiroli system lekotsitlar	Polymorphic core lekottsitlar
Interferonlar sistemasi	Interferonlar oz sondagi aminokislotalar va uglevodlarga ega kichik molekular pepetidlardir	interferons system	Interferons a small number of amino acids and carbohydrates with a small molecular pepetidlardir
Lizotsim sistemasi	Lizotsim glikozidaz guruxiga kiruvchi fermentdir	Lizottsim system	Lizottsim glycosides group, out of the fermenter
Propedin sistemasi	Bakteritsidlik xususiyatlariga ega bo‘lgan qon zardobining globulin tipidagi oqsil	Propedin system	Bactericidal properties of blood serum globulin type of protein
Perdere	Parchalash	perdere	shred
Nospetsifik	Organizimning turli tuman	Nospettsifik	Organizimning various

immunitet	infeksion agentga nisbatan turg‘unligini namoyon qilishi	immunity	infectious agents to show a relatively stable
Endoantegnlar	Tabiy yoki birlamchi va orttirilgan yoki ikkilamchi antigenlar	Endoantegnlar	Natural or acquired or primary and secondary antigene
Splenomegaliya	Rant kasalligining xarakterli belgilaridir	splenomegalia	Ranta nature of the disease,
	Xujayra patologik fiziologiyasi		
Ekzotropiya	Membrananing strukturaviy o‘zgarishlari uning tashqariga bo‘rtib chiqishi	exotropia	Membrane structural changes out of his bulging
Endotropiya	Sitoplazma ichiga kirishi	Endotropiya	Entry into the cytoplasm
Mitoxondriyalarning shikastlanishi	Mitoxondriyalar energiya hosil qiluvchi strukturalar	mitochondrion injury	Form of energy to the mitochondria structures
Tashqi membrana	Silliq bo‘lib organoidni o‘rab turishi	the outer membrane	Smooth and surrounded by Jude
Ichki membrana	Ko‘plab burmachalar tishchalarni xosil qilishi	the inner membrane	Playing a lot of burmachalar spikes
Donador endoplazmatik to‘r	Membranalarda ribasomalar uchrashi	Granular network endoplazmatik	Membrane ribasomalar encounter
Kapillar yoki xaqiqiy staz	qon oqib ketishning qiyinlashishi bilan bog‘liq bo‘lmay u juda kuchli ta’sirotchilari	Stasis in the capillaries or true	blood flow associated with the departure difficult it is a very strong ta'sirotchilari
Gemotogen ishemiya	arteriya qon tomirlari torayib, tromb yoki embollar bilan tiqilib qolishidan xosil bo‘ladi	Gemotogen ischemia	narrowing of the arteries, the blood vessels, it would clogged with clot, or embolus formation
Endogen ishemiya	arteriya qon tomirlari ichki devorining ateroskelerozlar davrida qalinlashib torayishi oqibatida xosil bo‘ladi	endogenous ischemia	a result of the narrowing of the artery wall during ateroskelerozlar widens dressing
Reflektor ishemiya	vozokonstriktor nerv apparatining reflektor ta’sirlanishidan rivojlanishi	reflex ischemia	vozokonstriktor the development of the nervous reflex reaction to the device
Kollaterial ishemiya	bir organdan qo‘shni yoki boshqa ayrim o‘zoq organ yoki to‘qimadan oqib ketishidan hosil bo‘ladi	Kollaterial ischemia	Joint theoretical or some other organ or tissue is formed by flowing out
Angiospatik konstriktiv ishemiya	qon tomir muskullarining yoki tomirlarni toraytiruvchi nervlarning ta’sirlanish oqibatida arteriya qon tomirlarining qisqarishidan hosil bo‘lishi.	Angiospatik constrictive ischemia	stroke caused by narrowed arteries or nerves to the muscles to react to the formation of arterial blood vessels reduction.
Kollaterial qon aylanish	xatto katta diametrli qon tomirlar tiqilib qolganida xam organ yoki uning biror qismining qon bilan ta’minlanishi yonbosh qon tomirlari tarmoqlari orqali tiklanishi	Kollaterial blood circulation	even before the current blockage of blood vessels of large diameter, or any part of the body to restore blood supply through the branches of the iliac blood vessels

Infarkt	mayda ko‘pincha terminal arteriya tomirining berkilishi natijasida biror qism to‘qimalarning nobo‘d bo‘lishi.	heart attack	small artery as a result of closure of the terminal to be a part of tissue nobo'd.
Xaematemesis	Oshqozonda	Xaematemesis	stomach
Xaemorrxagia per rxexin	qon tomirining yorilishi	Xaemorrxagia per rxexin	a ruptured blood vessel
Xaemorrxagia per diabrosin	qon tomirlarining yaralanishi	Xaemorrxagia per diabrosin	blood vessels wounded
Diabrosin	emirilish yoki eroziya	Diabrosin	degradation or erosion
Xaemorrxagia per diapedesis	jaroxatlanmagan qon tomirlardan diapedezi	Xaemorrxagia per diapedesis	lesion of blood vessels diapedezi
Tromboz	organizmning xayotiy jarayonlarda qon tomirlar devorida qon tarkibida hosil bo‘lishi	thrombosis	the body's vital processes of the formation of blood on the wall of blood vessels
Tromb	qon tomirlarida qon tarkibiy qismlaridan hosil bo‘lgan zichlashgan qattiq massa	stickiness	that the density of the solid mass of the components of the blood in the blood vessels
Aralash tromb	tromda oq va qizil qismlar navbatma navbat kelsa	mixed clot	Troms members of the white and red parts in order to come
Abturatsiyalovchi tromb	trombning qon tomirlarda joylashishiga qarabqon tomirlarini butunlay berkituvchi	Abturattsiyalovchi clot	thrombin blood vessels located qarabqon vessels completely sealed
Devoroldi tromb	qon tomirlar devorining bir tomoniga jipishlashishi	Devoroldi clot	one side of the wall of the blood vessels jipishlashishi
Tromb organizatsiyasim	bazan tromblarga tomirlar devoridan biriktiruvchi to‘qimalar o‘sib kirishi	stickiness organizattsiyasim	sometimes blood clots enter the vessel wall tissue growth
Flebolitlar	venalarda xosil bulgan bunday o‘zgarishlar	Flebolitlar	Such changes which playing in Vienna
Arterioletlar	arterialarda hosil bo‘lishi	Arterioletlar	The formation of artery
Emboli	ponaga o‘xshab orasiga kirib borish	embolism	Pona like to go into the
Embollar	zarrachalar	embolus	particles
Xavoli emboliya	atmosfera xavosining qon tomirlariga tushushidan hosil bo‘lish	air embolism	air formation of blood vessels shunned
Gazli emboliya	tomirlarning gaz pufakchalari bilan tiqilishidan hosil bo‘ladi	gas embolism	the gas bubbles formed overcrowding of vessels
Bakteriya emboliyasi	qon tomirlar o‘zaning mikroorganizmlarning konglomeratlari tushganda ko‘zatiladi	bacteria embolism	when the blood vessels are conglomerations of transmission of microorganisms ko'zatiladi
Parazit emboliyasi	gelmintlar lichenkali shakillarining qonga tushushi	Noise embolism	helminths kits blood forms lichenkali
Eidogen emboliya	embol tashkil topgan materialiga bog‘liq ravishda bir necha turdagi emboliyalar farq qilinadi	Eidogen embolism	embolic material consisting of several different types of embolism
Tromb emboliyasi	uzilgan tromb bulakchasi sabab	clot embolism	of fresh clot bulakchasi

	buladi		status
Parenximatozxujayrali emboliya	parenximatoz organlar va bir vaqitda ular venalarning jaroxati yoki degenirativ o‘zgarishlaridan hosil bo‘ladi	Parenximatozxujayrali embolism	parenchymal organs, and the exponents of their veins injuries or degenirativ changes
	Yallig‘lanish		
Inflammariya	alangalanish	Inflammariya	fire
Flogogen sabablar yalig‘lanish	chaqiruvchi sabablar turli tuman bo‘lib yallig‘lanish chiqaruvchi barcha moddalar	Flogogen reasons yalig'lanish	called causes all kinds of inflammatory substances
Pxlogosis	shikastlanish	Pxlogosis	strain
Gepatit	jigarning yallig‘lanishi	hepatitis	inflammation of the liver
Nefrit	bo‘yrakning yallig‘lanishi	jade	Inflammation bo'yrakning
Pnevmoniya	o‘pkaning yallig‘lanishi	pneumonia	inflammation of the lungs
Rubor	qizarish	rubor	redness
Tumor	SHish	Tumor	tumor
Kolor	xarorat ko‘tarilishi	Kolor	the rise in temperature
Dolor	Og‘riq	dolor	pain
Funktio laesa	funksiyaning bo‘zilishi	Funktion Laeso	deterioration of the function
Ekssudat	tomirlardan to‘qimalarga chiqqan suyuqlik	exudate	fluid from the tissues vessels
Ekssudatsiya	Jarayon	Ekssudattsiya	process
Eksuda	terilash ajirataman	exudative	to separate the skin
Granulatsitlovchi tuqima	tez usuvchi biriktiruvchi tuqima qon tomirlariga boy bo‘lishi	Granulatsitlovchi foolish	astringent foolish of the priorities to be rich in blood vessels
Zardobli kataral yallig‘lanish	shilliq pardalarda rivojlanadi	Whey catarrhal inflammation	develop on the mucous membranes
Fibrinli yallig‘lanish	ekssudat tarkibida fibrin kup bo‘lib qon plazmasining oqsillarinikup saqlaydi	inflammation of fibrin	exudate contains fibrin, a lot of blood plasma stores oqsillarinikup
Krupoz yalig‘lanish	fibrinning organ yuzasida erkin joylashishi xarakterli bo‘lib u fibrin ipchalaridan tashkil topgan parda hosil qilib engil ko‘chadi	croupous yalig'lanish	The location of the access to the fibrin on the surface of the body with a characteristic fibrin branches of the screen, you can see how easy
Gemorragik	yallig‘lanishlarda ekssudat tarkibida ko‘p yoki kam eritrotsitlar saqlangani uchun u qizil ranga ega buladi	haemorrhagic	more or less inflammation exudate cells saved, it will be red color
Abssess	aniq biriktiruvchi to‘qimali qobiq bilan chegaralangan su’niy bo‘shliqda yiring tuplanishi	abscess	Kit of fabric shell with limited space artificial pus
Frunkul	jun xaltachasi yog‘ bezchasining ularni urab turuvchi yumshoq biriktiruvchi tuqimalarning yiringli yallig‘lanishi	Furuncle	wool pouch of fat which surround them bezchasining purulent inflammation of soft astringent tuqimalarning
Karbunkula	bir qancha frunkullarning	Karbunkula	joining a number of

	qo‘shilishidan yoki bir guruh jun xaltachasi va yog‘ bezchalarining yallig‘lanishidan xosil bulishi		furuncles or a group of wool pouch and become fat dressing bezchalarining fever
Flegmona	teri tagida muskullar aro kletchatkada interstitsial to‘qimalarda me’da va ichak shilliq pardasi ostki qavatida kapsulaga o‘ralmasdan keng qisimlarga tarqalgan yiring hosil bo‘lishi	phlegmon	between the muscles under the skin fat interstitial tissue in the stomach and intestinal mucous membrane of the sub-floor capsules o'ralmasdan large parts of the formation of pus in common
Empiyaema	yopiq bo‘shliqlarda yiring to‘planishi	Empiyaema	The accumulation of pus in the closed cavities
Piyemiya	qonga yiring hosil qiluvchi mikroorganizmlarning kirishi bilan to‘qima va organlarda ko‘plab metastatik abssesslar hosil bo‘ladi.	Piyemiya	blood, pus-forming microorganisms to enter the tissues and organs of many metastatic abssesslar formed.
Oqma	tananing chuqur yiringli yallig‘lanish o‘choqlarining tashqi muhit bilan bog‘lovchi xaltachasimon yo‘lidan yiring sizib turishi	fistulas	deep abscesses, inflammation of the body into the furnace of pus in the external environment and the way of connecting xaltachasimon
Ixoroz yallig‘lanish gangrena	shikastlangan qisimlarga chirituvchi bakteriyalar tushash va tuqimalarni iritish ixoroz-irigan yallig‘lanish	Ixoroz yalig'lanish gangrene	damaged parts of putrefactive bacteria on the bed and tuqimalarni irritating ixoroz putrid inflammation
Kataral yallig‘lanish	nafas, hazim qilish, siydik tanasul kanali, sut bezlari yo‘llari, ko‘z shilliq pardalarida rivojlanish	catarrhal yalig'lanish	respiratory, humor, urine, body canal, mammary glands, eyes, mucous membranes of the development
Giperergik yallig‘lanish	organizmga antigen tabiatli kasallik chaqiruvchi sababning bir necha marta ta’sirida keyin paydo bulishi	Giperergik yalig'lanish	antigen challenge the nature of the disease in the body cause a number of times and then appear under the influence of
Gipergik yoki gipergik yallig‘lanish	tashqi belgilari namayon bo‘lishining pasayishi yoki mutloqa bo‘lmasligi bilan farq qilishi	Gipergik or gipergik yalig'lanish	whereby the symptoms that the decrease or totally not vary with
	To‘qimalarda kuzatiladigan tipik – patologik jarayonlar		
Gipoplaziya	tuqima yoki organ o‘sishining etishmovchiligiga aytiladi.	Gipoplaziya	the growth of the body of foolish or lack of it.
Atrofiya	to‘qima organ xajmi va o‘lchami kattaligining kichrayib funksiyasining zaiflashishi bilan xarakterlanadigan jarayonga	atrophy	Tissue and organ size and function of size, decreasing in size, characterized by a weakening of the process

	aytiladi.		said.
Nierogen atrofiya	to‘qimalar innervatsiyasining bo‘zilishi natijasida hosil buladi.	Nierogen atrophy	innervattsiyasining tissue deterioration resulting status.
Gormonal atrofiya	ayrim ichki sekritsiya bezlari asosan gipofiz, qalqonsimon bez funksiyasi etishmovchiliklari organizmni o‘sish va rivojlanishdan to‘xtatadi.	hormonal atrophy	sekritsiya glands, pituitary gland, thyroid function problem stops the growth and development of the organism.
Funksional atrofiya	tuqimaning funksiyanal faoliyati pasayganligi oqibatida rivojlanadi.	functional atrophy	tuqimaning functions develops a result of the decrease of the activity.
Alimentar atrofiya	maxalliy va umumiy bo‘ladi	nutritional atrophy	local and general
Kaxekssiya	organizmning umumiy atrofiyasi bo‘lib tana vazni keskin kamayib barcha fiziologik funksiyalarning pasayishi bilan namayon bo‘ladi.	Kaxekssiya	atrophy of the body's total body weight significantly decreased with the decline of physiological functions pursuit.
Nekros	bir guruh xujayralar butun organ yoki bir qismining o‘lishiga aytiladi.	necrosis	a group of cells on the death of the whole body or a part of it.
Nekrobioz	tuqimalarning sog‘lom xolatdan o‘lim xolatiga o‘tish jarayoniga	Nekrobioz	tuqimalarning healthy patch mortality process
Nekros	O‘lik	necrosis	dead
bios	Tirik	BIOS	live
Nam kollikvatsion nekros	o‘lgan tuqimalar yumshab va parchalanib bo‘tqasimon massaga aylanishi bilan xaraktirlanadi	Nam kollikvattsion necrosis	mitigate tuqimalar died and disintegrated into a mass bo'tqasimon xaraktirlanadi
Gepertrofiya	tuqima organ yoki uning biror qismining ayrim xujayralari o‘lchamining oshishi natijasida xajmining kattalashishidir	Gepertrofiya	foolish organ or cells in a certain part of the volume as a result of the increase in the size kattalashishidir
Giperplaziya	to‘qima xajmi xujayralarning miqdoriy ko‘payishi evaziga kattalashishi.	Giperplaziya	due to the increase in the volume of tissue cells whose sizes.
Patologik gepertrofiya	patologik jarayonlar davrida paydo bo‘ladi	pathological gepertrofiya	will appear during pathological processes
Ishchi gepertorofiya	patologik gipertrofiya turlari	Working gepertorofiya	pathological hypertrophy
Regenratsiyalanuvchi gepertrofiya	organning bir qismi olib tashlanganida qolgan qismi xisobiga rivojlanishidan xosil bo‘ladi	Regenrattsiyalanuvchi gepertrofiya	Removing part of the body will be playing the expense of the rest of the development
Korrelatsion gepertrofiya	regulator funksional aloqadorlikka ega sistimalarga xarakterli.	Korrelattsion gepertrofiya	regulators functional characteristic in connection with sistimalarga.
Fiziologig regeneratsiya	sog‘lom organizmning xayot faoliyati davrida parchalangan to‘qima va organlarning	Fiziologig regenerattsiya	healthy activities during the life of the organism, said the rebuilding of broken

	tiklanishiga aytiladi		tissues and organs
Patologik yoki reprativ regeneratsiya	organizmda turli patologik ta’sirotchi tuqimalar shikastlanishi kuzatiladi.	Pathological or reprativ regenerattsiya	various pathological reaction of the organism tuqimalar injury.
	O‘smalar		
O’sma	tuqimalarning barqaror patologik o‘sishi o‘ziga xos biologik xususiyatlariga ega bulishi, o‘sishning chegaralanmasligi va boshqarilmasligi xo‘jayralarning tuzilishi va funksiyasining o‘zgarishiga aytiladi	O‘sma	tuqimalarning stable pathological growth of specific biological characteristics of the country, said growth is limited and is governed by changes in cell structure and function
Konsepogen	o‘smalarni xosil qiladigan moddalar	Konsepogen	tumor formation
Gistoid usmalar	bu o‘smalar faqat o‘sma xo‘jayralaridan iborat bo‘lganligi uchun ataladi.	Gistoid o‘smalar	These tumors for which only the tumor cell referred to.
Ekspansiv	o‘smaning boshqa to‘qima ichiga o‘sib kirmay kattalashishi.	Ekspansiv	shall not grow into other tissues of the tumor sizes.
Metastaz	xavfsiz o‘smalar jarroxlik yo‘li bilan olib tashlangani	Metastaz	tumors surgical removed through
Destruktiv	xavfli o‘smalar o‘sganida atrof to‘qimalarni ymiradi.	Destruktiv	malignant tissue growth Ymir.
Yetuk o’smalar	xavfsiz o‘smalar o‘zlarining morfologik tuzilishiga ko‘ra sog‘lom to‘qimani eslatadi	Etuk o‘smalar	According to their morphological structure of the tumor resembles the healthy tissue
Yetilmagan o’sma	xavfli o‘sma xavfsiz o‘smalarga nisbatan sog‘lom to‘qimadan kuchli farq qilganligi uchun ularni	Etilmagan usma	malignant tumor tumor healthy tissue differs from them
O’sma shtammlari	uzoq va ko‘p marotab ko‘chirib o‘tkazilgan o‘smalar	O‘sma shtammlari	long and repeated transplantation tumors
	Issiqlik almashinuvining buzilishi		
Gipotermiya	issiqlik almashinuvi boshqarilishining buzilishi hayvon tana xaroratining pasayishi bilan namayon bo‘lishi	hypothermia	heat exchange boshqarilishining be a violation of animal body temperature falls whereby
Xupo	past	Xupo	low
Gipertirmiya	hayvon tana xaroratining ko‘tarilishi va uning ortiqcha qizishi bilan boradigan termoregulatsiyaning bo‘zilishi	Gipertirmiya	animal body temperature and excessive heat termoregulatsiyaning deterioration
Xyuper	YUqori	Xyuper	high
Txerma	Issiqlik	Txerma	heat
Pirogen moddalar	isitma hosil qiluvchi moddalar	Pirogov	fever-forming substances
Pir	issiqlik.	Pir	heat.
Medikamentoz	dori moddalari ta’sirida hosil bo‘ladigan issitma	medication	formed under the influence of the drug substance slay-
Stadium	tana haroratining ko‘tarilish	Stadium	body temperature rise

inkerementi		inkerementi	
Stadium fastigii	tana harorati yuqori darajada ko‘tarilgan xolda saqlanishi.	Stadium fastigii	body temperature rose to the highest level with the safety.
Stadium dekrementi	tana haroratining pasayishi	Stadium decrement	The decrease in body temperature
Fibris kontinua	doimiy tipdagi isitma	fibrin kontinua	permanent type of fever
Fibris remittens	bo‘shashtiruvchi yokremittirlovchi isitma	fibrin remittens	relaxing yokremittirlovchi fever
Fibris intermittens	ko‘tarilib to‘shib turuvchi yoki intermitterlovchi isitma	fibrillar intermittent	rose and waved a higher fever or intermitterlovchi
Fibris rekurreni	qaytalanuvchi isitma	fibrin rekurreni	recurrent fever
Fibris xiktik	tinkani qurutuvchi yoki gektik isitma	fibrin xiktik	Tinker fever or drying hectares
Fibris atxyupika	atipik isitma	fibrin atxyupika	atypical fever
Fibris epxemera	qisqa vaqitli efemer isitma	fibrin epxemera	short exponents of ephemeral fever
Nerv sistemasi	termoregulatsiya bo‘zilishining asosida yotadigan o‘zgarishlar paydo bo‘lishi.	the nervous system	termoregulatsiya bo'zilishining foundation changes.

Moddalar almashinuvining patofiziologiyasi

Alimentar giperglikemiya	hayvonlar juda kup engil o‘zlashtiriladigan uglivodli oziqalar istemol qilishidan hosil bo‘ladi.	nutritional Hyperglycemia	The food is a lot of light uglivodli consumer to make sure.
Giperglikemiya	alimentar emotsional va pankreatik turlari farq qilinadi	Hyperglycemia	different types of nutritional emottsional and pancreatic
Emotsional giperglikemiya	markaziy nerv sistemasining juda kuchli qo‘zg‘alishi oqibatida hosil bo‘ladi.	emotional Hyperglycemia	a result of the excitation of the central nervous system is a very powerful form.
Gepoglikemiya	kelib chiqishi bir qancha sabablarga yoki bu sabab turlarining miqdoriga bog‘liq bo‘ladi.	Gepoglikemiya	a number of reasons, or because the origin of the species depends on the amount.
Gepoglikemiya sindrom	kavush qaytaruvchi hayvonlarda ham o‘ziga hos o‘zgarishlar hosil bo‘ladi.	Gepoglikemiya syndrome	sandals animals to specific changes.
Alimentar giperlipemiya	yog‘lar so‘rilganda qonda netral yog‘lar ko‘payadi	alimentary hyperlipemia	increase the absorption of fats in the blood netron fats
Lipidemiya	bu moddalar miqdorining qonda ko‘payishi	Lipidemiya	an increase in the amount of these substances in the blood
Transport giperlipemiya	agar giperlipemiya yog‘ning depolardan jigarga tashilishida yuz berishi	hyperlipemia	If hyperlipemia oil depot in the occurrence of liver tashilishida
Retension giperlipemiya	natijada qonda yog‘ miqdori ko‘payishi	Retension hyperlipemia	resulting in an increase in the amount of fat in the blood

Lipuriya	lipoidonefrozlarda yog‘lar kam miqdorda siydikdapaydo bo‘lishi.	Lipuriya	lipoidonefrozlarda siydikdapaydo less fat.
Xiluriya	siydikda juda katta miqdorda yog‘ xilez tomirlaridan limfa siydik chiqaruv yo‘llarigatushganida kuzatilishi.	Xiluriya	a very large amount of fat in the urine Hillel lymph vessels of the urine Quality yo'llarigatushganida.
Giperketonemiya	ba’zan qonda atseton tanachalarining miqdori ko‘payishi	Giperketone miya	sometimes increase the amount of acetone in the blood cell
Ketonuriya	katta miqdorda qon bilan ajiralishi	ketonuria	a large amount of blood divorce
Yog‘li infiltratsiya	to‘qimalarda o‘zoq muddat katta miqdorda yog‘ saqlanishi.	fatty infiltration	Ozal while a large amount of fat stored in the tissues.
Lipodistrofiya	muayyan joylarda yog‘larning katta miqdorda to‘planishi.	Lipodystrop hy	The accumulation of large amounts of fat in certain areas.
Ateroskleroz	arteriyalar ayniqsa yirik arteriyalar subendotelial tqimasining xolesterin va uning efirlari bilan infiltratsiyasi.	atherosclero sis	especially in the large arteries, the arteries of cholesterol tqimasining ellastic and its ethers infiltration.
Geperproteinemiya	oqsil almashinuvining bo‘zilishi qon plazmasi yoki zardobida oqsillar miqdor ko‘payishi bilan ifodalanishi	Giperprotei nemiya	protein metabolism deterioration expressed in the increase in the amount of protein in the blood plasma or serum
Giperglobulinemiya	ko‘pincha qon plazmasidagi oqsillarning ko‘payishi globulin fraksiyasining ko‘payishi.	Giperglobul inemiya	often increased globulin fraction of proteins in blood plasma increased.
Eksikoz	to‘qimalarning qurishi	Eksikoz	drying of the tissues
Atsidoz	Kislotali	acidosis	acidic
Alkaloz	ishqoriy	alkalosis	alkaline
Gazli atsidoz	o‘pka kasalliklari bilan kasalanishi	gas acidosis	lung diseases safes
Gazli alkaloz	organizmdan xaddan tashqari ko‘p miqdorda karbonat angidrid ajiralishi natijasida ishqoriy rezer kamayishi.	gas alkalosis	the body is too much carbon dioxide as a result of divorce decrease in alkaline prefere.
Metabolitik atsidoz	organizmda organik kislotalardan sut asetosirka va beta-oksimoy kislotalarining juda ko‘p miqdorda to‘planishi	metabolic acidosis	the body of organic acids in milk asetosirka accumulation of large amounts of acid and beta-oksimoy
Metabolitik alkaloz	organizmda ishqor saqlovchi moddalarning	metabolic alkalosis	base storage substances accumulation in the body,

	to‘planib qolishi		
Poliuriya	hayvonlarda solishtirma og‘irligi past bo‘lgan ko‘p miqdorda suyuqliklar ajiralishi.	polyuria	animals, the specific weight of a large amount of liquids divorce.
Istisqo	suyuqlik zardob bo‘shliqqa yo‘nalib u erda katta miqdorda to‘planishi	dropsy	The accumulation of large amounts of liquid whey space where he directed
Gidrotoraks	plevra bo‘shlig‘ining istisqosi	effusions	pleural cavity istisqosi
Perikardiya gidroperikardiya	yurak perikardining istisqosi	pericarditis gidroperikardiya	pericarditis istisqosi
Gidrosefals	miya qorinchasi istisqosi	Gidrosefals	brain're istisqosi
Assit	qorin bo‘shlig‘ining istisqosi.	Assis	istisqosi abdomen.

Och qolish patofiziologiyasi

Valin	oziqada etishmasligi muskullar darmonsizlanishi	valine	lack of muscle darmonsizlanishi
Tirozin	etishmovchilik	tyrosine	lack of
Metionin va sistin	gipofizoldingi bo‘lagining adrenakortikotrop va o‘sish gormonining hosil bo‘lishi.	Methionine and cysteine	piece gipofizoldingi adrenakortikotrop and the production of growth hormone.
Avitaminoz	vitaminlardan birortasi bo‘lmasligidan hosil bo‘ladigan kasallik	Avitaminosis	the disease will not be any vitamins
Poliavitaminoz	tarkibida birdaniga bir qancha vitaminlar etishmasligi	Poliavitaminoz	once the structure of a lack of vitamins
Gipovitaminoz	vitaminlardan birortasi etishmasligi	scurvy	The lack of any of the vitamins
Poligipovitaminoz	vitaminlar etishmasa	Poligipovitaminoz	vitamins
Akseroftol	A vitamin	Akseroftol	vitamin A
Kalsiferol	D vitamin	Kalsiferol	vitamin D
Tokoferol	E vitamin	tocopherol	vitamin E
Filloxinon	K vitamin	Filloxinon	vitamin K
Tiamin	B 1 vitamin	thiamine	Vitamin B 1
Riboflavin	B 2 vitamin	riboflavin	Vitamin B 2
Nikotin kislotasi	PP vitamin	nicotinic acid	vitamin PP
Kobalamin	B12 vitamini	Kobolamin	vitamin B12
Pangamat kislota	B 15 vitamini	Pangamat acid	Vitamin B 15
C-metilmetionin	U vitamini	C-metilmetionin	He vitamin

Normovomeliya	qon umumiy miqdorining normal bo‘lishi	Normovolemiya	the total amount of blood to be normal
Valume	xajm	Valume	volume
Politsitemik normovolemiya	qonning umumiy xajmi o‘zgarmasdan shaklli elementlarni sonining ko‘payishi.	Polisitemik normovolemiya	an increase in the total volume remained unchanged shaped elements of the blood.
Gipervolemiya	plazma va eritrotsitlar o‘rtasidagi nisbat.	Gipervolemiya	The ratio between the plasma and red blood cells.
Oligositemik gipervolemiya	qonning umumiy miqdori asosan qonning suyuq qismi hisobiga ko‘payishi	Oligositemik gipervolemiya	the total amount of the increase, mainly due to the liquid part of the blood of the blood
Angidremiya	polisitemik gipovolemiya	Angidremiya	polisitemik hypovolemia
Anemiya	qonning xajm birligida eritrotsitlar soni va gemogloben miqdorining kamayishi.	anemia	The decrease in the number per unit volume of blood cells and hemoglobin.
Gemolitik anemiya	anemiya eritrotsitlarni parchalovchi zaharli moddalar bilan hayvonlarning zaharlanishidan kelib chiqadi.	hemolytic Animal	anemia, red blood cells with toxic substances from degradation due to the poisoning of animals.
Regenerativ anemiya	qon hosil qiluvchi organlarda tiklanish jarayonining yaxshi namoyoon bo‘lishi.	regenerative anemia	blood-forming organs and the recovery process showed.
Aregenerativ anemiya	qizil elik qon hosil qiluvchi xususyatining zaiflashishidan hosil buladi.	Aregenerativ anemia	move to weaken scientists to make red blood, quietly formed.
Gemotstoblastlar	leykotsitlarni hosil qiladigan ona hujayralar	Gemositoblastlar	make sure that the mother's cells, leukocytes
Donador leykotsitlar	leykotsitlarni hosil qiladigan ona hujayralar	granular leykosidlar	make sure that the mother's cells, leukocytes
Granulotsitlar	gemostablastladan qizil ilikda	granulocytes	gemostablastladan red marrow
Mieloblastlar	boshlang‘ich hujayralar	myeloblastic	primary cells
Limfoblast monoblast	limfa tuguni va taloqda ishlab chiqarilishi	Limfoblast monoblast	the output of the lymph node and spleen
Pilazmotsitlar	leykotsitlar orasida ataluvchi xujayralar	Pilazmotsitlar	cells called leukocytes in
Leykoformula	oq qon xujayralari tarkibining sifat ko‘rsatkichi	Leykoformula	indicator of the quality of the structure of white blood cells
Leykotsitar profil	leykotsitlar asosiy turlarining mutloq sonlarda ifodalanishi	Leykositar profile	The main species are expressed in absolute numbers of leukocytes

Leykotsitoz	qonda leykotsitlarning muayyan turdagi hayvon uchun me’yordagi nisbatdan kupayib ketishi.	Leykositoz	leukocytes in the blood of certain types of animal than normal, keeps bleeding.
Miogen leykotsitoz	muskullari ishi qanchalik og‘ir va o‘zoq bo‘lsa shunchalik kuchli ifodalanishi.	Myogenic leykositoz	theoretical work and how hard muscles are so strongly expressed.
Patologik leykotsitozlar	barcha infeksion va invazion kasalliklarga xos leykotsitozning rivojlanishi	pathological leykositozlar	The development of specific infectious and parasitic diseases, leukocytosis
Neytrofiliya leykotsitoz	ko‘pchilik utkir oqimli infeksion kasalliklar	neutrophils leykositoz	most acute infectious diseases
Qon yadrosining chapga siljishi	leykoformulada yosh neytrofillar ko‘payib ketishi	The movement of the blood to the left of the core	leykoformulada young neutrophils proliferation
Regenerativ siljish	leykotsitlar tarkibidagi bunday o‘zgarishi	regenerative progress	such a change in the structure of leukocytes
Degenerativ o‘zgarishlar	qonda umumiy lekotsitlar kamayib tayoqcha yadirolilar ko‘paysa bu qizil ilik faoliyatining zaiflashishi	degenerative changes	lekotsitlar in the blood decrease the activity of the bone, decreasing the number of stick yadirolilar red
Regenerativ –degenerativ siljish	qon yadirosining chapga siljishi leykatsitlarning deginerativ o‘zgarishlar bilan birgalikda ko‘zatilishi.	Regenerative and degenerative progress	yadirosining leykatsitlarning shift to the left, together with the changes in the deginerativ ko'zatilishi.
Eozinofilli leykotsitoz	qonda eozinofillarni ko‘payishi	eosinophilia leykositoz	an increase in blood eosinophilia
Limfotsitoz	qonda limfotsitlarni ko‘payishi	Limfositoz	an increase in blood lymphocytes
Monotsitoz	qonda monotsitlarning mutloq va nisbiy ko‘payishi	mono	monottsitlarning absolute and relative increase in blood
Gistotsitlar	surinkali kechuvchi kasalliklar	Gistiotsitlar	Diseases accompanied surinkali
Leykopeniya	oq qon xujayralarning muayyan hayvon turi uchun me’yordan kamayishi	leukopenia	The decrease in white blood cells, a particular type of animal norm
Neytropeniya	qonda neytrofil leykotsitlar miqdorining kamayishi	Netropeniya	The decrease in the amount of blood neutrophil leykottsitlar
Agranulotsitoz	donali leykotsitlarning qonda keskin kamayishi	agranulocytosis	a sharp decline in blood-grained leykottsitlarning
Eozinopeniya	ko‘pchilik infeksion kasalliklar to‘liq rivojlanganda sodir bo‘ladi.	Eozinopeniya	the development of many infectious diseases.
Limfotsitopeniya	qonda limfotsitlarning	Limfositope	a decrease in blood

	kamayishi.	niya	limfottsitlarning.
Monositopeniya	monotsitlarning qonda kamayishi.	Monositopeniya	The decrease in blood monottsitlarning.
Haima	qon	Haiming	blood
Blastoz	kurtak	blastoma	bud
Leykemiya	oq qon kasalligi	leukemia	white blood disease
Miyeloidli leykoz	miyeloid to‘qimalarning o‘sishi	Miyeloidli leukemia	myeloid tissue growth
Aleykemik miyelozda	qonda leykotsitlar miqdori norma atrofida yoki o‘rtacha ko‘payishi	Aleykemik miyelozda	leykottsitlar blood in norm or an increase in the average
Limfoid leykoz	limfoid to‘qimalar o‘sib limfa tugini taloq va jigarning keskin kattalashishi	lymphoid leukemia	growth of lymphoid tissue found in the lymph heart severely enlarged liver and spleen
Retikuloendotelioz	qizil ilik, taloq, limfa tuguni va jigarda ritikular xujayralarning katta miqdorda o‘sishi	Retikuloendotelioz	red bone marrow, spleen, lymph nodes and liver ritikular cells to grow large quantities of
Trombositoz	qondagi trombotsitlar miqdorining ko‘payishi	thrombocytosis	an increase in the amount of blood trombottsitlar
Philia	moyillik	Philip	inclination
Letsitin	qonda fosfolipidlar miqdorining o‘zgarishi	lecithin	changes in the amount of phospholipids in the blood
Ketonemiya asetonemiya	qonda keton tanachalarining ko‘payishi	Ketonemiya asetonemiya	increased ketones in the blood cell
	Immun sistema patofiziologiyasi		
Gistamin	allergiyaning eng muhim mediatorlaridan biri	histamine	One of the most important mediators of allergies
Serotonin	silliq muskullar qisqarishlari	serotonin	smooth muscle
Bradikinin	kinin sistemaning eng faol komponenti	bradykinin	the reproductive system is the most active component
Geparin	antitrombin bilan trombinning koagulatsiya qilishi	heparin	antithrombin with thrombin koagulattsiya
Prostaglandin	siklopentan xalqali to‘yinmagan yog‘ kislotalari	prostaglandin	cyclopentane ring of unsaturated fatty acids
Atopiya	takroriy tipdagi reaksiyalar	atopic	the second type of reactions
Anafilaksiya	hayvonlar organizmining antigenni muayyan yashirin davirdan keyin takroriy parenteral yo‘l bilan yuborilishi	anaphylaxis	specific antigen animals hidden Davis and then re-sent to the parenteral route
Sensibilizatsiya	hayvonlar organizmini antigenni takroriy tushishi	sensitizing	the second drop antigens in animals
Zaif sensibilizatsiya	sinsibilatsiya qilinmagan hayvonga aktiv – sinsibilatsiya qilingan	weak sensitizing	sinsibilatsiya yet the animal has been active - sinsibilatsiya animal serum

	hayvondan olingan qon zardobi		
Antianafilaksiya va desensibilizatsiya	anafilaktik shok bilan kasallanib o‘tgan hayvon	Antianafilaksiya and desensitizing	anaphylactic shock sick animal

Qon aylanishning patofiziologiyasi

Insufficientia valvulae aortaye	aorta klapinlarining etishmovchiligi	Insufficientia valvulae aortaye	failure of the aorta klapinlarining
Pulsus celer et altus	tezkor puls	Pulse Celera Diagnostics Altus	rapid pulse
Stenosis ostii aortae	aorta teshigining torayishi	Stenosis aorta	narrowing of the aortic valve opening
Pulsus parvus et tardus	aorta bo‘g‘zi toraygan paytda puls sekinlashadi va kechik bo‘lishi.	Pulse Parvus et Tardush	aortic neck narrowed pulse is slow and late.
Insufficientia valvulae mitralis	ikki tabaqali klapan etishmovchiligi	Insufficientia valvulae mitralis	two-level valve failure
Stenosis ostii mitralis	chap atrioventrikular teshikning torayishi	Stenosis mitralis	narrowing of the left atrioventricular opening
Yurak gipertrofiyasi	yurak muskuli vaznining asosan muskullari	cardiac hypertrophy	according to the weight of heart muscle muscles
Fiziologik gipertrofiya	yurak vazni skelet muskullari rivojlanishi	physiological hypertrophy	weight development of the skeletal muscles of the heart
Patologik gipertrofiya	yurak vazni skelet muskullariga bog‘liq bo‘lmagan xolatdagi o‘tishi	pathological hypertrophy	cases are not related to the weight of the skeletal muscles of the heart to
Fibrinli perikardit	perikard varaqlari fibrin qavati bilan qoplanishi	fibrin pericarditis	pericardial sheets covered with a layer of fibrin
Traxikardiya	yurak ish ritmining tezlashishi	Traxikardiya	heart rhythm acceleration
Bradikardiya	- yurak ish ritmining sekinlashishi	bradycardia	- Slow heart rhythm
Sinus aritmiyasi	yurak qisqarishlari har xil vaqit oraliqlarida takrorlanishi	sinus arrhythmia	repeat heart Waqif different frames,
Sinusli ekstrasistola	bo‘lmachalar va qorinchalar qisqarishlarining to‘liq saqlanishi	sinusitis ekstrasistola	have signs and kept're reduced
Atrioventrikular blokada	tugunda yoki giss bog‘lamida o‘tkazuvchanlikning bo‘zilishi oqibatida paydo bo‘lishi	atrioventricular block	ball or Gissing hub transmission deterioration caused
Hilpillovchi aritmiya	aritmiyaning bu turi yurak ishining butunlay bitartibligi bilan namayon bo‘lishi	Hilpillovchi arrhythmia	the work of this type of cardiac arrhythmias completely bitartibligi

			pursuit
Tomirlar sklerozi	arteriyalar elastikligi yuqolib qattiqlashib paydo bo‘lishi	vascular sclerosis	elasticity of the arteries inclement appeared to be hardened,
Endokrin gipertoniya	qon tomirlar tonusi ortishi	endocrine hypertension	increased tone of blood vessels
Nerogen gipertoniya	qon tomirlarini xarakatga keltiruvchi markazlarning qo‘zg‘alishidan kelib chiqishi	Nerogen hypertension	The origin of the blood vessels movement, the centers bring excitement
Kollaps	qon aylanishining o‘tkir qon tomir etishmasligi	collapse	The lack of blood circulation in acute stroke,
	Nafas patologiyasi		
Kussmaul	nafas siyrak chuqur va qaltiroq nafas olish xamda uzoq pauza bilan xaraktirlanish	KUSSMAUL	Rare breathing deep, quavering breath and a long pause with xaraktirlanish
Agonal nafas	o‘limoldi xolati paytida kuzatilish	Agony breath	seen during the state of o'limoldi
Dispnoe	nafas ritmining buzilishi	Dispnoe	respiratory failure
Pnevmotoraks	plevra bo‘shliqlariga xavo to‘planishidir	pneumothorax	the collection of pleural air pockets
Klapanli pnevmotoraks	eirtilgan joy xarakatchan po‘stloq bilan qoplanishi	valve pneumothorax	eirtilgan covered with bark xarakatchan
Gipoksik gipoksiya	arteriya qoni kislorod bilan etarlicha boyimasligi	hypoxic hypoxia	Arterial blood oxygen enrichment
Turg’un gipoksiya	qonning organizm bo‘ylab xarakatlanishi	persistent hypoxemia	blood throughout the body movements
Gistotoksik gipoksiya	qon orqali kislorod tashilishi etarli darajada bo‘lishi	Gistotoksik hypoxia	sufficient transport of oxygen through the blood
	Ovqat xazm bo‘lishining buzilishi yoki patologiyasi		
Gipodupsia	hayvonlar suv istemol qilishining kamayishi	Gipodupsia	to minimize water consumption
Adupsia	hayvonlar suv istemol qilishining yoki suvga extiyojining yuqolishi	Adupsia	water consumption, or water demand has occurred
Gipersalivatsiya	su’lak ajiralishining sezilarli darajada ko‘payishi	salivation	increased significantly separated from the su'lak
Giposalivatsiya	su’lak ajiralishining kamayishi	Giposalivatsiya	The decrease separated from su'lak
Ataniya	me’daoldi bo‘lmalar ataniyasi	Lawmakers from	me'daoldi upon ataniyasi
Hepotoniya	me’daoldi bo‘lmalari gepotaniyasi	Hepotoniya	me'daoldi gepotaniyasi
Tympania meteorismus	katta qorinning gaz bilan to‘lib ketishi	Tympania meteorismus	filled with gas abdominal departure
Gipersekretsiya-	me’dada oziqa xazimlanishining barcha	Gipersekretsiya-	stomachs, food xazimlanishining

	davirlarida shira ajiralishining kuchayishi		davirlarida an increase in glow separated
Giposekretsiya	-xazimlanishning barcha davirlarida me’da shirasining kam miqdorda ajiralishi	Giposekretsiya	-xazimlanishning davirlarida divorce less gastric juice
Giperkinez	me’da qisqarishining kuchayishi	Giperkinez	increase in the reduction of the stomach
Vomitus	og‘iz bo‘shlig‘i orqali oziqa massasini tashqi muxitga ixtiyorsiz chiqarilishi	Vomitus	mass production of the EIA, a reflex through the oral cavity
Ileus	ichaklar tiqilishi	ileus	obstruction of the intestine
	Jigar faoliyatining buzilishi		
Gepatozlar	moddalar almashinuvining buzilishi xisobiga distrofik o‘zgarish	hepatosis	dystrophic change at the expense of metabolic disorders
Urobilinemiya	katta miqdorda umumiy qon aylanish doirasiga tushadi	Urobilinemiya	will be a large amount of general circulation within the
Urobilinuriya	siydik orqali ajiralishi	Urobilinuriya	excreted through divorce
Sirroz	jigarning surunkali kasalligi bo‘lishi	cirrhosis	chronic liver disease
	Ayiruv jarayonlarning patofiziologiyasi		
Pog’onasis moddalar	kanalchalarda so‘rilmaydigan moddalar	Pog'onasis	Caudan absorbable substances
Ketonuriya	buyrak orqali siydikda chiqarilishi	ketonuria	production of urine by the kidneys
Glukozuriya	qonda glukoza miqdorining ko‘payishi ortiqcha glukozaning siydik bilan chiqarilishi	Glukozuriya	an increase in the amount of glucose in the blood excessive production of urine glukozaning
Gipoproteinemiya	qon plazmasida oqsil moddalar kamayishi	Gipoproteinemiya	The decrease in the blood plasma protein
Antidiuretik gormoni	buyrak kanalchalarida suvning reabsorbsiyalanishini kuchaytirishi	antidiuretic hormone	water in the renal tubules increase reabsorbsiyalanishini
Tireoglobulin gormoni-	moddalar almashinuvini ko‘chaytirib to‘qimalar suv saqlash xususiyatining pasayishidan diurezni kuchaytirishi	Tireoglobulin gormoni-	declining to save water in tissue metabolism ko'chaytirib enhance diuresis
Tireotrop gormoni	diurez gipofizi	Tireotrop hormone	urine pituitary
Nefrit	patogenizda shamollash omillarining axamiyati	jade	patogenizda inflammatory factors of significance
Nefroz-	siydik kanalchalarining distrofik o‘zgarishlari	Nefroz-	changes in the urinary tubules dystrophic

Nefroskleroz	buyraklardagi sklerot o‘zgarishlar umumiy arterioskleroz paytida va buyraklarning o‘zidagi surunkali yallig‘lanish	nephroscler osis	
Poliuriya	buyrak orqali katta miqdordr siydik ajiralishi	polyuria	changes in the kidney scleral general arteriosclerosis and chronic inflammation of the kidneys
Diurez	kuchayishi ko‘pincha buyrakning surunkali yallig‘lanishi	diuresis	miqdordr urine through the kidneys divorce
Pollakuriya	tez-tez siydik ajiralishini farqlash	Pollakuriya	often a chronic inflammation of the kidneys to increase
Oliguriya	buyrak orqali siydik miqdori ajiralishining kamayishi	oliguria	frequent urine to distinguish divorce
Anuriya	buyrak orqali siydikni qisqa yoki uzoq muddatga ajiralmay qolishi	anuria	The decrease separated from the urine by the kidneys
Proteinuriya	sog‘lom hayvon siydigida oqsil miqdori jo‘da kam ya’ni izi bo‘lib an’anaviy tekshirish usullari bilan aniqlab bo‘lmaslik	proteinuria	the kidneys through urine short or long term stay in divorce
Gematuriya	siydikda qon ajiralishi	hematuria	The amount of protein in the urine of healthy animals jo'da exact footprint of the traditional methods
Gemoglobinuriya	siydikda eritrotsitlar bilan birikmagan erkin gimogilobinning ajiralishi	hemoglobin uria	blood in the urine divorce
Glukozuriya	siydik bilan qand ajiralishi	Glukozuriy a	red blood cells in the urine sediment gimogilobinning divorce
Silindruriya	siydikda siydik silindirlarining paydo bo‘lishi	Silindruriya	urine sugar divorce
Epiteliy silindr	buyrak kanalcha epiteliyalarning ekssudati bilan yopishgan va kanalchalarda siydik oqimi bilan olib chiqilgan bo‘lishi	a cylindrical epithelium	the formation of urine urine cylinder
Indikanuriya	hayvonlarning normal siydigida kam miqdorda saqlanishi	Indikanuriy a	Kidney channels epiteliyalarning exudate must have been taken to block the flow of urine and tubules
Indikan	indoksilsulfat kislotasining kaliliy tuzi	Indikan	stored in a small amount of urine of normal animals
Bakteriuriya	yangi ajiralgan siydikda mikroorganizmlarning	bacteriuria	indoksilsulfat acid salt of the key

	bo‘lishi		
Aldosteron	buyrak shikastlangan kasallar qoni va siydigida organizmda suv-tuz almashinuvinigg asosiy boshqaruvchisidir	aldosterone	a new separate microorganisms in urine
Gipertenzin	qon tomirlarini kuchli toraytiruvchi xususiyatiga ega bo‘lishi	Hypertensio n	the urine and blood of patients with kidney damage in the body's water and salt authority to manage almashinuvinigg
Azotemik uremiya	urimiya	uraemia azotemia	urine
Eklamptik uremiya	o‘tkir nefritda yoki surunkali nefrit asoratlari kuchayganida rivojlanishi	Eklamptik uremia	Urim
	Endokrin boshqarilish patologiyasi		
Gipofiz ekstirpatsiya	olib tashlanish	pituitary ekstirpatsiya	throw
Gipofiz patologiyasi	gipofiz giperfunksiyasi bilan bog‘liq bo‘lgan kasalliklardagi o‘sishning kuchayishi	pituitary pathology	Increased growth in non-communicable diseases associated with pituitary giperfunktsiyasi
Gigantizim	butun tananing proporsional o‘sishi rivojlanishi	gigantism	the development of the entire increase in the proportion of the body
Akromegaliya	bunda kasallik metafizlar suyaklanishi yakunlanganiligidan keyin rivojlanishi	acromegaly	These patients metaphysical suyaklanishi development yakunlanganiligidan
Insulin	oshqozon osti bezi orolchalarining beta-xujayralarida bezning asosiy garmoni hosil bo‘lishi	insulin	pockets of pancreatic beta-cells in the gland to be the main accordion
Lipokain	oshqozonosti bezining mayda yo‘llari epiteliyalarida garmoni hosil bo‘lishi	Lipokain	pancreas and small ways epiteliyalarida accordion
Giperketonemiya	diabet davrida yog‘lar almashinuvi ham buzilib qonda keton tanachalarini ko‘payishi	Giperketonemi ya	diabetes increased ketone violated during the exchange of fats in the blood
Infiltratsiya	jigarda kuchli yog‘ to‘planib ruyberishi	infiltration	-Microsomal strong fat accumulation ruyberishi
Testosterone	erkaklar jinsiy garmoni	Testosterone	Men accordion
Ekstradiol (follikulin	follikulalarda urg‘ochi hayvonlarning jinsiy garmonlari ishlab chiqarilishi	Ekstradiol (follicle	follicles production of female sex hormones in animals
Progesteron	tuxumdonda sariq tanada	progesterone	The production of yellow

	ishlab chiqarilishi		body of the ovary
Ganadotrop gormoni	jinsiy bezlar faoliyatini boshqarishda gipofiz oldingi qismidan ajiralishi	Ganadotrop hormone	sex in the management of the activity of the pituitary glands in the front part of the divorce
Kriptorxizm	rivojlanishdagi nuqsonlar davrida xam axtalangandagiga o‘xshash ko‘p belgilari ko‘zatilishi	Kriptorxizm	malformations period, the symptoms are similar to axtalangandagiga ko'zatilishi
	Nerv sistemasining patologik fiziologiyasi		
Faol giperemiya	bosh miya arteriyalari tonusining pasayishi bilan birgalikda yurak ishining tezlashishiga olib keladigan barcha xolatlarda paydo bo‘lishi	active hyperemia	to minimize water consumption
Travmatik shok	nerv sistemasining mexank shkastlanishlar natijasida rivojlanishi	traumatic shock	increased significantly separated from the su'lak
Paradoksal	oliy nerv faoliyatining juda chuqur bo‘zilishlari paytida ro‘y berishi	paradoxicall y	The decrease separated from su'lak
Ultraparadoksal	kirish bilan bosh miya pustlog‘ining shartli qitiqlovchi ta’sirga nisbatan reaksiyasi	Ultraparado ksal	me'daoldi upon ataniyasi
Tormoz	hayvonning javob qaytish qobiliyati susayadi yoki batomom yo‘qoladi	brake	me'daoldi gepotaniyasi
Gipokinez	xarakat funksiyasining to‘liq ishdan chiqishi	Gipokinez	stomachs, food xazimlanishining davirlarida an increase in glow separated
Tetraplegiya	barcha oyoqlarning falajlanishi	Tetraplegiy a	-xazimlanishning davirlarida divorce less gastric juice
Paraplegiya	gavda bir bir tomonining falajlanishi	Paraplegiya	increase in the reduction of the stomach
Monoplegiya	bitta oyoqning falajlanishi	Monoplegiy a	mass production of the EIA, a reflex through the oral cavity
Tremor	titrash	tremor	dystrophic change at the expense of metabolic disorders
Ataksiya	xarakat muvofiqligi va tana muvozanatining bo‘zilishi	ataxia	will be a large amount of general circulation within the
Ataxia-	tartibsiz	Ataxia-	excreted through divorce
Periferik ataksiya	periferik nervlarning shikastlanishi va	peripheral ataxia	chronic liver disease

	sezuvchanlikni chuqur bo‘zilishi oqibatida pydo bo‘lishi		
Vestibule ataksiya	-vestibular apparatining shikastlanishi	Vestibule ataxia	Minus pathophysiological processes
Interoseptiv	ichki organlar sezuvchanligi	Interoseptiv	Caudan absorbable substances
Talamik	sezuvchanlik ta’sirotni qo‘pol ravishda qabul qilish bilan xaraktirlanish	adolescents	production of urine by the kidneys
Giperesteziya	nerv sistemasining yuqori qo‘zg‘aluvchanlik xolati	Giperestezi ya	an increase in the amount of glucose in the blood excessive production of urine glukozaning
Paresteziya	soxta sezgi	paresthesia	The decrease in the blood plasma protein

Questions for Science Certifications:

Oral questions for OB 1 (120)

1. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
2. Explain fever, etiopathogenesis, types, stages and significance?
3. Causes and consequences of impaired liver production and excretion?
4. Explain the pathological effects of disorders of bile formation on the body?
5. Explain inflammation, causes, stages, classification, significance and consequences?
6. Give an idea of the general directions that explain the origin of the disease?
7 What is the mechanism of recovery of impaired functions based on?
8. Explain the tumors, types, differences in development, biological properties?
9. Explain the pathology of white blood cell formation?
10. Give an idea of the development of pathophysiology in Uzbekistan?
11. Explain the importance of arterial and venous hyperemia for the body?
12. Give an idea about the disorders of sensory activity of the nervous system and its consequences?
13. Give an idea of tanotogenesis and its periods?
14. Explain the theories that explain the formation of tumors?
15. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
16. Give an idea about illness and health?
17. Explain the local circulatory disorders and its types?
18. Changes caused by dysfunction of the pituitary gland?
19. Give a general idea about nosology?
20. Explain atrophy, hypertrophy, regeneration and their types?
21. Explain the reasons for changes in the amount and composition of urine?
22. Etiology, give an idea of the types of etiological factors?
23. Explain that tumors are related to the organism?
24. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
25. Explain that animals are not susceptible to infectious diseases?
26. Explain the mechanism of development of diabetes?
27. Explain the mechanism of development of hypertension and hypotension?
28. Explain the importance of the nervous and humoral systems in reactivity?
29. Explain the mechanism of development of edema and inflammation?
30. Explain the violation of the incretory function of the pancreas?
31. General principles of disease classification?
32. Explain the protein and carbohydrate, fat, and vitamin starvation and its consequences?
33. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
34. Explain the theories that clarify the etiology and their essence?
35. Explain the types and consequences of starvation?
36. What are the causes of dysfunction of the secretory organs of the digestive tract?
37. Explain the pathological effects of changes in the composition of soil, water and atmospheric air on the body?

38. Explain the changes in organs and systems during fever?
39. Explain the disorder of digestion in the stomach?
40. Explain the pathological effects of electricity on animals?
41. Explain the causes, types and consequences of stasis, local anemia and heart attack?
42. Explain the disorder of appetite and thirst for water?
43. Explain the pathological effects of heat and cold on the body?
44. Dystrophic changes and metabolic disorders in the inflammatory focus?
45. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.
46. Explain the development of the science of pathological physiology in Russia?
47. Explain hypoglycemic shock and the mechanism of its formation?
48. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
49. Explain the importance of heredity, constitution, breed, age and sex in pathology?
50. Explain the consequences of disturbances in the metabolism of minerals and water?
51. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
52. Explain the pathogenesis of anaphylactic shock?
53. Metabolism in tumors. Experimental oncology and its importance?
54. Disorders of the endocrine function of the gonads?
55. Explain the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
56. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
57. Give an idea about the types of heart defects?

58. Explain biological causes as disease-causing causes?
59. Explain the effect of the organism on tumor growth?
60. Explain the pathological effects of adrenal insufficiency on animals?
61. Give an idea about general adaptation syndrome or G. Sele doctrine?
62. Explain the metabolic disorders during fever?
63. Give an idea about the pathology of internal respiration, lack of oxygen?
64. Explain the mechanism of action of etiological causes?
65. What is anaplasia, give an idea of its types?
66. Explain circulatory disorders in pericardial and myocardial pathology?
67. Explain allergies, allergic diseases, infectious allergies, autoallergies?
68. Vascular reaction in inflammation. Explain exudate and its types?
69. Explain respiratory disorders in pulmonary pathology?
70. Explain the mechanical causes of disease?
71. When does hemotransfusion shock occur?
72. Explain the renal and extrarenal causes of urinary disorders?

73. How to study the science of animal pathophysiology, give them an idea?
74. What are hypo and hyperthermia, explain their periods and significance?
75. Explain the causes, types and consequences of changes in total blood volume?
76. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
77. Explain fever, etiopathogenesis, types, stages and significance?
78. Causes and consequences of impaired hepatic function of the liver?
79. How to study the science of animal pathophysiology, give them an idea?
82. What is hypo and hyperthermia, explain the periods and significance?
81. Explain the causes, types and consequences of changes in total blood volume?
82. Explain the development of the science of pathological physiology in Russia?
83. Explain hypoglycemic shock and the mechanism of its formation?
84. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
85. Explain the mechanical causes of disease?
86. When does hemotransfusion shock occur?
87. Explain the renal and extrarenal causes of urinary disorders?
88. Give a general idea about nosology?
89. Explain atrophy, hypertrophy, regeneration and their types?
90. Explain the reasons for changes in the amount and composition of urine?
91 What is the mechanism of recovery of impaired functions based on?
92. Explain the tumors, types, differences in development, biological properties?
93. Explain the pathology of the formation of white blood cells?
94. Give an idea about tanotogenesis and its periods?
95. Give an idea of the theories that explain the formation of tumors?
96. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
97. Etiology, give an idea of the types of etiological factors?
98. Tumor, explain how tumors are related to the organism?
99. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
100. Explain the importance of heredity, constitution, breed, age and sex in pathology?
101. Explain the consequences of disturbances in the metabolism of minerals and water?
102. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
103. Explain that animals are not susceptible to infectious diseases?
104. Explain the mechanism of development of diabetes?
105. Explain the mechanism of development of hypertension and hypotension?
106. Explain the theories that clarify the etiology and their essence?
107. Explain the types and consequences of starvation?
108. What are the causes of disorders of the secretory function of the digestive organs?
109. Explain the importance of the nervous and humoral systems in reactivity?
110. Explain the mechanism of development of edema and inflammation?

111. Explain the violation of the incretory function of the pancreas?
112. General principles of disease classification?
113. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?
114. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
115. Explain the pathological effects of electricity on animals?
116. Explain the causes, types and consequences of stasis, local anemia and heart attack?
117. Explain the disorder of appetite and thirst for water?
118. Explain the pathological effects of heat and cold on the body?
119. Dystrophic changes in the foci of inflammation and metabolic disorders?
120. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.

Oral questions for OB 2 (120)

1. Explain the pathological effects of changes in the composition of soil, water and atmospheric air on the body?
2. Explain the changes in organs and systems during fever?
3. Explain digestive disorders in the stomach?
4. Explain the development of pathophysiology in Uzbekistan?
5. Explain the importance of arterial and venous hyperemia for the body?
6. Give an idea about the disorders of sensory activity of the nervous system and its consequences?
7. Give an idea about illness and health?
8. Explain the local circulatory disorders and its types?
9. Changes caused by dysfunction of the pituitary gland?
10. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
11. Explain the general reaction of the body to inflammation and the effect of the source of inflammation on the body?
12. Give an idea about the types of heart defects?
13. Explain allergies, allergic diseases, infectious allergies, autoallergies?
14. Vascular reaction in inflammation. Explain exudate and its types?
15. Explain respiratory disorders in pulmonary pathology?
16. Give an idea about general adaptation syndrome or G. Sele doctrine?
17. Explain the metabolic disorders during fever?
18. Give an idea about the pathology of internal respiration, lack of oxygen?
19. Explain the mechanism of action of etiological causes?
20. What is anaplasia, give an idea of its types?
21. Explain circulatory disorders in pericardial and myocardial pathology?
22. Explain the pathogenesis of anaphylactic shock?
23. Metabolism in tumors. Experimental oncology and its importance?
24. Disorders of the endocrine function of the gonads?
25. Explain biological causes as the cause of the disease?
26. Explain the effect of the organism on tumor growth?
27. Explain the pathological effect of adrenal insufficiency on animals?
28. Explain the pathological effects of disorders of bile formation on the body?
29. Explain inflammation, causes, stages, classification, significance and consequences?
30. Give an idea of the general directions that explain the origin of the disease?
31. Explain the science, functions and relationship of animal pathophysiology to other sciences? (anatomy, physiology, biochemistry, biophysics, pathoanatomy, microbiology, virology, animal nutrition and clinical sciences),
32. Explain fever, etiopathogenesis, types, stages and significance?
33. Causes and consequences of impaired function of the liver to produce and excrete bile?
34. Explain the pathological effect on the body of disorders of the formation and excretion of bile?

35. Explain inflammation, causes, stages, classification, significance and consequences? (microorganisms, viruses, fungi, simple animals, helminths, alternative, exudative, infiltrative, normergic, hyperergic, hypergic)
36. The main stages of development of pathology (animism, humoral, solid, cellular, ytrophysical, ytrochemical, nervousism).
37 What is the mechanism of recovery of impaired functions based on?
38. Explain the tumors, types, differences in development, biological properties? (dangerous, safe, orgonoid and histoid, infiltrative, exponential, relapsing, anaplasia)
39. Explain the pathology of the formation of white blood cells? (myeloblasts, lymphoblasts, plasma cells, leukoformula)
40. Explain the development of pathophysiology in Uzbekistan?
41. Explain the importance of arterial and venous hyperemia for the body? ()
42. Give an idea of the disorders of sensory activity of the nervous system and its consequences?
43. Give an idea about tanotogenesis and its periods?
44. Give an idea of the theories that explain the formation of tumors?
45. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
46. Give an idea about illness and health?
47. Explain the local circulatory disorders and its types?
48. Changes caused by dysfunction of the pituitary gland?
49. Give a general idea about nosology?
50. Explain atrophy, hypertrophy, regeneration and their types?
51. Explain the reasons for changes in the amount and composition of urine?
52. Etiology, give an idea of the types of etiological factors?
53. Tumor, explain how tumors are related to the organism?
54. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
55. Explain that animals are not susceptible to infectious diseases?
56. Explain the mechanism of development of diabetes?
57. Explain the mechanism of development of hypertension and hypotension?
58. Explain the importance of the nervous and humoral systems in reactivity?
59. Explain the mechanism of development of edema and inflammation?
60. Explain the violation of the incretory function of the pancreas?
61. General principles of disease classification?
62. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?
63. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
64. Explain the theories that clarify the etiology and their essence?
65. Explain the types and consequences of starvation?
66. What are the causes of disorders of the secretory function of the digestive organs?
67. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
68. Explain the changes in organs and systems during fever?
69. Explain the disorder of digestion in the stomach?

70. Explain the pathological effects of electricity on animals?
71. Explain the causes, types and consequences of stasis, local anemia and heart attack?
72. Explain the disorder of appetite and thirst for water?
73. Explain the pathological effects of heat and cold on the body?
74. Dystrophic changes in the foci of inflammation and metabolic disorders?
75. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the imprinting reactions of the nervous system and the types of the nervous system in pathology ?.
76. Explain the development of the science of pathological physiology in Russia?
77. Explain hypoglycemic shock and the mechanism of its formation?
78. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
79. Explain the importance of heredity, constitution, breed, age, and gender in pathology?
80. Explain the consequences of disturbances in the metabolism of minerals and water?
81. Explain the consequences of a violation of the biochemical and physicochemical properties of blood?
82. Explain the pathogenesis of anaphylactic shock?
83. Metabolism in tumors. Experimental oncology and its importance?
84. Disorders of the endocrine function of the gonads?
85. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
86. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
87. Give an idea about the types of heart defects?
88. Explain biological causes as disease-causing?
89. Explain the effect of the organism on tumor growth?
90. Explain the pathological effect of adrenal insufficiency on the body of animals?
91. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
92. Explain the metabolic disorders during fever?
93. What is the concept of internal respiratory pathology, lack of oxygen?
94. Explain the mechanism of influence of etiological causes?
95. What is anaplasia, give an idea of its types?
96. Explain circulatory disorders in pericardial and myocardial pathology?
97. Explain allergies, allergic diseases, infectious allergies, autoallergies?
98. Vascular reaction in inflammation. Explain exudate and its types?
99. Explain the disruption of the respiratory process in lung pathology?
100. Explain the mechanical causes of disease?
101. When does hemotransfusion shock occur?
102. Explain the renal and extrarenal causes of urinary disorders?
103. How to study the science of animal pathophysiology, give them an idea?
104. What are hypo and hyperthermia, explain their periods and significance?
105. Explain the causes, types and consequences of changes in total blood volume?

106. Explain the science, functions and relationship of animal pathophysiology to other sciences?
107. Explain fever, etiopathogenesis, types, stages and significance? ()
108. Causes and consequences of impaired function of the liver to produce and excrete bile?
109. How to study the science of animal pathophysiology, give them an idea?
110. What are hypo and hyperthermia, explain their periods and significance?
111. Explain the causes, types and consequences of changes in total blood volume?
112. Explain the development of the science of pathological physiology in Russia?
113. Explain hypoglycemic shock and the mechanism of its formation?
114. Explain the causes, types and consequences of anemia, quantitative and qualitative changes in red blood cells?
115. Explain the mechanical causes of disease?
116. When does hemotransfusion shock occur?
117. Explain the renal and extrarenal causes of urinary disorders?
118. Give a general idea about nosology?
119. Explain atrophy, hypertrophy, regeneration and their types?
120. Explain the reasons for changes in the amount and composition of urine?

Oral questions for GP (300)

1. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
2. Explain fever, etiopathogenesis, types, stages and significance?
3. Causes and consequences of impaired liver production and excretion?
4. Explain the pathological effects of disorders of bile formation on the body?
5. Explain inflammation, causes, stages, classification, significance and consequences?
6. Give an idea of the general directions that explain the origin of the disease?
7 What is the mechanism of recovery of impaired functions based on?
8. Explain the tumors, types, differences in development, biological properties?
9. Explain the pathology of white blood cell formation?
10. Give an idea of the development of pathophysiology in Uzbekistan?
11. Explain the importance of arterial and venous hyperemia for the body?
12. Give an idea about the disorders of sensory activity of the nervous system and its consequences?
13. Give an idea of tanotogenesis and its periods?
14. Explain the theories that explain the formation of tumors?
15. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
16. Give an idea about illness and health?
17. Explain the local circulatory disorders and its types?
18. Changes caused by dysfunction of the pituitary gland?
19. Give a general idea about nosology?
20. Explain atrophy, hypertrophy, regeneration and their types?
21. Explain the reasons for changes in the amount and composition of urine?
22. Etiology, give an idea of the types of etiological factors?
23. Explain that tumors are related to the organism?
24. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
25. Explain that animals are not susceptible to infectious diseases?
26. Explain the mechanism of development of diabetes?
27. Explain the mechanism of development of hypertension and hypotension?
28. Explain the importance of the nervous and humoral systems in reactivity?
29. Explain the mechanism of development of edema and inflammation?
30. Explain the violation of the incretory function of the pancreas?
31. General principles of disease classification?
32. Explain the protein and carbohydrate, fat, and vitamin starvation and its consequences?
33. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
34. Explain the theories that clarify the etiology and their essence?
35. Explain the types and consequences of starvation?
36. What are the causes of dysfunction of the secretory organs of the digestive tract?
37. Explain the pathological effects of changes in the composition of soil, water and atmospheric air on the body?

38. Explain the changes in organs and systems during fever?
39. Explain the disorder of digestion in the stomach?
40. Explain the pathological effects of electricity on animals?
41. Explain the causes, types and consequences of stasis, local anemia and heart attack?
42. Explain the disorder of appetite and thirst for water?
43. Explain the pathological effects of heat and cold on the body?
44. Dystrophic changes and metabolic disorders in the inflammatory focus?
45. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.
46. Explain the development of the science of pathological physiology in Russia?
47. Explain hypoglycemic shock and the mechanism of its formation?
48. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
49. Explain the importance of heredity, constitution, breed, age and sex in pathology?
50. Explain the consequences of disturbances in the metabolism of minerals and water?
51. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
52. Explain the pathogenesis of anaphylactic shock?
53. Metabolism in tumors. Experimental oncology and its importance?
54. Disorders of the endocrine function of the gonads?
55. Explain the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
56. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
57. Give an idea about the types of heart defects?

58. Explain biological causes as disease-causing causes?
59. Explain the effect of the organism on tumor growth?
60. Explain the pathological effects of adrenal insufficiency on animals?
61. Give an idea about general adaptation syndrome or G. Sele doctrine?
62. Explain the metabolic disorders during fever?
63. Give an idea about the pathology of internal respiration, lack of oxygen?
64. Explain the mechanism of action of etiological causes?
65. What is anaplasia, give an idea of its types?
66. Explain circulatory disorders in pericardial and myocardial pathology?
67. Explain allergies, allergic diseases, infectious allergies, autoallergies?
68. Vascular reaction in inflammation. Explain exudate and its types?
69. Explain respiratory disorders in pulmonary pathology?
70. Explain the mechanical causes of disease?
71. When does hemotransfusion shock occur?
72. Explain the renal and extrarenal causes of urinary disorders?

73. How to study the science of animal pathophysiology, give them an idea?
74. What are hypo and hyperthermia, explain their periods and significance?
75. Explain the causes, types and consequences of changes in total blood volume?
76. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
77. Explain fever, etiopathogenesis, types, stages and significance?
78. Causes and consequences of impaired hepatic function of the liver?
79. How to study the science of animal pathophysiology, give them an idea?
82. What is hypo and hyperthermia, explain the periods and significance?
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90. Explain the reasons for changes in the amount and composition of urine?
91 What is the mechanism of recovery of impaired functions based on?
92. Explain the tumors, types, differences in development, biological properties?
93. Explain the pathology of the formation of white blood cells?
94. Give an idea about tanotogenesis and its periods?
95. Give an idea of the theories that explain the formation of tumors?
96. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
97. Etiology, give an idea of the types of etiological factors?
98. Tumor, explain how tumors are related to the organism?
99. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
100. Explain the importance of heredity, constitution, breed, age and sex in pathology?
101. Explain the consequences of disturbances in the metabolism of minerals and water?
102. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
103. Explain that animals are not susceptible to infectious diseases?
104. Explain the mechanism of development of diabetes?
105. Explain the mechanism of development of hypertension and hypotension?
106. Explain the theories that clarify the etiology and their essence?
107. Explain the types and consequences of starvation?

108. What are the causes of disorders of the secretory function of the digestive organs?
109. Explain the importance of the nervous and humoral systems in reactivity?
110. Explain the mechanism of development of edema and inflammation?
111. Explain the violation of the incretory function of the pancreas?
112. General principles of disease classification?
113. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?
114. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
115. Explain the pathological effects of electricity on animals?
116. Explain the causes, types and consequences of stasis, local anemia and heart attack?
117. Explain the disorder of appetite and thirst for water?
118. Explain the pathological effects of heat and cold on the body?
119. Dystrophic changes in the foci of inflammation and metabolic disorders?
120. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.
121. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
122. Explain the changes in organs and systems during fever?
123. Explain the disorder of digestion in the stomach?
124. Explain the development of pathophysiology in Uzbekistan?
125. Explain the importance of arterial and venous hyperemia for the body?
126. Give an idea of the disorders of sensory activity of the nervous system and its consequences?
127. Give an idea about disease and health?
128. Explain the local circulatory disorders and its types?
129. Changes caused by dysfunction of the pituitary gland?
130. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
131 Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
132. Give an idea about the types of heart defects?
133. Explain allergies, allergic diseases, infectious allergies, autoallergies?
134. Vascular reaction in inflammation. Explain exudate and its types?
135. Explain the disruption of the respiratory process in lung pathology?
136. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
137. Explain the metabolic disorders during fever?
138. What is the concept of internal respiratory pathology, lack of oxygen?
139. Explain the mechanism of influence of etiological causes?
140. What is anaplasia, give an idea of its types?
141. Explain circulatory disorders in pericardial and myocardial pathology?
142. Explain the pathogenesis of anaphylactic shock?
143. Metabolism in tumors. Experimental oncology and its importance?
144. Disorders of the endocrine function of the gonads?

145. Explain biological causes as disease-causing causes?
146. Explain the effect of the organism on tumor growth?
147. Explain the pathological effects of adrenal insufficiency on animals?
148. Explain the pathological effects of disorders of bile formation on the body?
149. Explain inflammation, causes, stages, classification, significance and consequences?
150. Give an idea of the general directions that explain the origin of the disease?
151. Explain the science, functions and relationship of animal pathophysiology with other sciences? (anatomy, physiology, biochemistry, biophysics, pathoanatomy, microbiology, virology, animal nutrition and clinical sciences),
152. Explain fever, etiopathogenesis, types, stages and significance?
153. Causes and consequences of impaired function of the liver to produce and excrete?
154. Explain the pathological effect on the body of disorders of the formation and excretion of bile?
155. Explain inflammation, causes, stages, classification, significance and consequences? (microorganisms, viruses, fungi, simple animals, helminths, alternative, exudative, infiltrative, normergic, hyperergic, hypergic)
156. The main stages of development of pathology (animism, humoral, solid, cellular, ytrophysical, ytrochemical, nervousism).
157 What is the mechanism of recovery of impaired functions based on?
158. Explain the tumors, types, differences in development, biological properties? (dangerous, safe, orgonoid and histoid, infiltrative, exponential, relapsing, anaplasia)
159. Explain the pathology of the formation of white blood cells? (myeloblasts, lymphoblasts, plasma cells, leukoformula)
160. Explain the development of pathophysiology in Uzbekistan?
161. Explain the importance of arterial and venous hyperemia for the organism? ()
162. Give an idea about the disturbance of sensory activity of the nervous system and its consequences?
163. Give an idea about tanotogenesis and its periods?
164. Give an idea of the theories that explain the formation of tumors?
165. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
166. Give an idea about disease and health?
167. Explain the local circulatory disorders and its types?
168. Changes caused by dysfunction of the pituitary gland?
169. Give a general idea about nosology?
170. Explain atrophy, hypertrophy, regeneration and their types?
171. Explain the reasons for changes in the amount and composition of urine?
172. Etiology, give an idea of the types of etiological factors?
173. Tumor, explain how tumors are related to the organism?
174. Common causes of disorders of the nervous system, pathology of the upper nervous system and reticular formation?
175. Explain that animals are not susceptible to infectious diseases?
176. Explain the mechanism of development of diabetes?

177. Explain the mechanism of development of hypertension and hypotension?
178. Explain the importance of the nervous and humoral systems in reactivity?
179. Explain the mechanism of development of edema and constipation?
180. Explain the violation of the incretory function of the pancreas?
181. General principles of disease classification?
182. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?
183. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
184. Explain the theories that clarify the etiology and their essence?
185. Explain the types and consequences of starvation?
186. Causes of disorders of the secretory function of the digestive organs?
187. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
188. Explain the changes in organs and systems during fever?
189. Explain the disorder of digestion in the stomach?
190. Explain the pathological effect of electric current on the body of animals?
191. Explain the causes, types and consequences of stasis, local anemia and heart attack?
192. Explain the disorder of appetite and thirst for water?
193. Explain the pathological effects of heat and cold on the body?
194. Dystrophic changes in the foci of inflammation and metabolic disorders?
195. Explain the role of experimental neuroses, the effect of endocrine glands on high nervous activity, the traces of the nervous system and the types of the nervous system in pathology ?.
196. Explain the development of the science of pathological physiology in Russia?
197. Explain hypoglycemic shock and the mechanism of its formation?
198. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
199. Explain the importance of heredity, constitution, breed, age, and gender in pathology?
200. Explain the consequences of disturbances in the metabolism of minerals and water?
201. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
202. Explain the pathogenesis of anaphylactic shock?
203. Metabolism in tumors. Experimental oncology and its importance?
204. Disorders of the endocrine function of the gonads?
205. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
206. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
207. Give an idea about the types of heart defects?
208. Explain biological causes as disease-causing causes?
209. Explain the effect of the organism on tumor growth?
210. Explain the pathological effects of adrenal insufficiency on animals?

211. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
212. Explain the metabolic disorders during fever?
213. What is the concept of internal respiratory pathology, lack of oxygen?
214. Explain the mechanism of influence of etiological causes?
215. What is anaplasia, give an idea of its types?
216. Explain circulatory disorders in pericardial and myocardial pathology?
217. Explain allergies, allergic diseases, infectious allergies, autoallergies?
218. Vascular reaction in inflammation. Explain exudate and its types?
219. Explain the disruption of the respiratory process in lung pathology?
220. Explain the mechanical causes of disease?
221. When does hemotransfusion shock occur?
222. Explain the renal and extrarenal causes of urinary disorders?
223. How to study the science of animal pathophysiology, give them an idea?
224. What are hypo and hyperthermia, explain their periods and significance?
225. Explain the causes, types and consequences of changes in the total amount of blood?
226. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
227. Explain fever, etiopathogenesis, types, stages and significance? ()
228. Causes and consequences of impaired function of the liver to produce and excrete bile?
229. How to study the science of animal pathophysiology, give them an idea?
230. What are hypo and hyperthermia, explain their periods and significance?
231. Explain the causes, types and consequences of changes in the total amount of blood?
232. Explain the development of the science of pathological physiology in Russia?
233. Explain hypoglycemic shock and the mechanism of its formation?
234. Explain the causes, types and consequences of anemia, quantitative and qualitative changes in red blood cells?
235. Explain the mechanical causes of disease?
236. When does hemotransfusion shock occur?
237. Explain the renal and extrarenal causes of urinary disorders?
238. Give a general idea about nosology?
239. Explain atrophy, hypertrophy, regeneration and their types?
240. Explain the reasons for changes in the amount and composition of urine?
241 What is the mechanism of recovery of impaired functions based on?
242. Explain the tumors, types, differences in development, biological properties?
243. Explain the pathology of the formation of white blood cells?
244. Give an idea about tanotogenesis and its cycles?
245. Give an idea of the theories that explain the formation of tumors?
246. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
247. Etiology, give an idea of the types of etiological factors?
248. Tumor, explain how tumors are related to the organism?

249. Common causes of disorders of the nervous system, pathology of the upper nervous system and reticular formation?
250. Explain the importance of heredity, constitution, breed, age and sex in pathology?
251. Explain the consequences of disturbances in the metabolism of minerals and water?
252. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
253. Explain that the organism of animals is not susceptible to infectious diseases?
254. Explain the mechanism of development of diabetes?
255. Explain the mechanism of development of hypertension and hypotension?
256. Explain the theories that clarify the etiology and their essence?
257. Explain the types and consequences of starvation?
258. What are the causes of disorders of the secretory function of the digestive organs?
259. Explain the importance of the nervous and humoral systems in reactivity?
260. Explain the mechanism of development of edema and constipation?
261. Explain the violation of the incretory function of the pancreas?
262. General principles of disease classification?
263. Explain the hunger and consequences of protein, carbohydrate, fat, continental starvation?
264. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
265. Explain the pathological effect of electric current on the body of animals?
266. Explain the causes, types and consequences of stasis, local anemia and heart attack?
267. Explain the disorder of appetite and thirst for water?
268. Explain the pathological effects of heat and cold on the body?
269. Dystrophic changes in the foci of inflammation and metabolic disorders?
270. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the imprinting reactions of the nervous system and the types of the nervous system in pathology ?.
271. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
272. Explain the changes in organs and systems during fever?
273. Explain the disorder of digestion in the stomach?
274. Explain the development of pathophysiology in Uzbekistan?
275. Explain the importance of arterial and venous hyperemia for the organism?
276. Give an idea of the disorders of sensory activity of the nervous system and its consequences?
277. Give an idea about disease and health?
278. Give an idea of local circulatory disorders and its types?
279. Changes caused by dysfunction of the pituitary gland?
280. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
281. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?

282. Give an idea about the types of heart defects?
283. Explain allergies, allergic diseases, infectious allergies, autoallergies?
284. Vascular reaction in inflammation. Explain exudate and its types?
285. Explain the violation of the respiratory process in lung pathology?
286. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
287. Explain the metabolic disorders during fever?
288. What is the concept of internal respiratory pathology, lack of oxygen?
289. Explain the mechanism of influence of etiological causes?
290. What is anaplasia, give an idea of its types?
291. Explain circulatory disorders in pericardial and myocardial pathology?
292. Explain the pathogenesis of anaphylactic shock?
293. Metabolism in tumors. Experimental oncology and its importance?
294. Disorders of the endocrine function of the gonads?
295. Explain biological causes as disease-causing causes?
296. Explain the effect of the organism on tumor growth?
297. Explain the pathological effect of adrenal insufficiency on animals?
298. Explain the pathological effects of disorders of bile formation on the body?
299. Explain inflammation, causes, stages, classification, significance and consequences?
300. Give an idea of the general directions that explain the origin of the disease?

1 Written work questions for OB (200 pieces)

1. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
2. Explain fever, etiopathogenesis, types, stages and significance?
3. Causes and consequences of impaired liver production and excretion?
4. Explain the pathological effects of disorders of bile formation on the body?
5. Explain inflammation, causes, stages, classification, significance and consequences?
6. Give an idea of the general directions that explain the origin of the disease?
7 What is the mechanism of recovery of impaired functions based on?
8. Explain the tumors, types, differences in development, biological properties?
9. Explain the pathology of white blood cell formation?
10. Give an idea of the development of pathophysiology in Uzbekistan?
11. Explain the importance of arterial and venous hyperemia for the body?
12. Give an idea about the disorders of sensory activity of the nervous system and its consequences?
13. Give an idea of tanotogenesis and its periods?
14. Explain the theories that explain the formation of tumors?
15. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
16. Give an idea about illness and health?
17. Explain the local circulatory disorders and its types?
18. Changes caused by dysfunction of the pituitary gland?
19. Give a general idea about nosology?
20. Explain atrophy, hypertrophy, regeneration and their types?
21. Explain the reasons for changes in the amount and composition of urine?
22. Etiology, give an idea of the types of etiological factors?
23. Explain that tumors are related to the organism?
24. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
25. Explain that animals are not susceptible to infectious diseases?
26. Explain the mechanism of development of diabetes?
27. Explain the mechanism of development of hypertension and hypotension?
28. Explain the importance of the nervous and humoral systems in reactivity?
29. Explain the mechanism of tumor and esophageal development?
30. Explain the violation of the incretory function of the pancreas?
31. General principles of disease classification?
32. Explain the effects of protein, carbohydrate, fat, and vitamin starvation?
33. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
34. Explain the theories that clarify the etiology and their essence?
35. Explain the types and consequences of starvation?
36. What are the causes of dysfunction of the secretory organs of the digestive tract?
37. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
38. Explain the changes in organs and systems during fever?

39. Explain the disorder of digestion in the stomach?
40. Explain the pathological effects of electricity on animals?
41. Explain the causes, types and consequences of stasis, local anemia and heart attack?
42. Explain the disorder of appetite and thirst for water?
43. Explain the pathological effects of heat and cold on the body?
44. Dystrophic changes in the inflammatory focus and metabolic disorders?
45. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.
46. Explain the development of the science of pathological physiology in Russia?
47. Explain hypoglycemic shock and the mechanism of its formation?
48. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
49. Explain the importance of heredity, constitution, breed, age and sex in pathology?
50. Explain the consequences of disturbances in the metabolism of minerals and water?
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69. Explain respiratory disorders in pulmonary pathology?
70. Explain the mechanical causes of disease?
71. When does hemotransfusion shock occur?
72. Explain the renal and extrarenal causes of urinary disorders?
73. How to study the science of animal pathophysiology, give them an idea?
74. What are hypo and hyperthermia, explain their periods and significance?
75. Explain the causes, types and consequences of changes in total blood volume?

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91 What is the mechanism of recovery of impaired functions based on?
92. Explain the tumors, types, differences in development, biological properties?
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94. Give an idea about tanotogenesis and its periods?
95. Give an idea of the theories that explain the formation of tumors?
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97. Etiology, give an idea of the types of etiological factors?
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99. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
100. Explain the importance of heredity, constitution, breed, age and sex in pathology?
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108. What are the causes of disorders of the secretory function of the digestive organs?
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110. Explain the mechanism of development of edema and inflammation?
111. Explain the violation of the incretory function of the pancreas?
112. General principles of disease classification?
113. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?

114. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
115. Explain the pathological effects of electricity on animals?
116. Explain the causes, types and consequences of stasis, local anemia and heart attack?
117. Explain the disorder of appetite and thirst for water?
118. Explain the pathological effects of heat and cold on the body?
119. Dystrophic changes in the foci of inflammation and metabolic disorders?
120. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.
121. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
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124. Explain the development of pathophysiology in Uzbekistan?
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126. Give an idea of the disorders of sensory activity of the nervous system and its consequences?
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131 Explain the general organism reaction of inflammation and the effect of the inflammatory focus on the organism?
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146. Explain the effect of the organism on tumor growth?
147. Explain the pathological effects of adrenal insufficiency on animals?
148. Explain the pathological effects of disorders of bile formation on the body?
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150. Give an idea of the general directions that explain the origin of the disease?
151. Explain the science, functions and relationship of animal pathophysiology with other sciences? (anatomy, physiology, biochemistry, biophysics, pathoanatomy, microbiology, virology, animal nutrition and clinical sciences),
152. Explain fever, etiopathogenesis, types, stages and significance?
153. Causes and consequences of impaired function of the liver to produce and excrete?
154. Explain the pathological effect on the body of disorders of the formation and excretion of bile?
155. Explain inflammation, causes, stages, classification, significance and consequences? (microorganisms, viruses, fungi, simple animals, helminths, alternative, exudative, infiltrative, normergic, hyperergic, hypergic)
156. The main stages of development of pathology (animism, humoral, solid, cellular, ytrophysical, ytrochemical, nervousism).
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158. Explain the tumors, types, differences in development, biological properties? (dangerous, safe, orgonoid and histoid, infiltrative, exponential, relapsing, anaplasia)
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164. Give an idea of the theories that explain the formation of tumors?
165. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
166. Give an idea about disease and health?
167. Explain the local circulatory disorders and its types?
168. Changes caused by dysfunction of the pituitary gland?
169. Give a general idea about nosology?
170. Explain atrophy, hypertrophy, regeneration and their types?
171. Explain the reasons for changes in the amount and composition of urine?
172. Etiology, give an idea of the types of etiological factors?
173. Tumor, explain how tumors are related to the organism?
174. Common causes of disorders of the nervous system, pathology of the upper nervous system and reticular formation?
175. Explain that animals are not susceptible to infectious diseases?
176. Explain the mechanism of development of diabetes?
177. Explain the mechanism of development of hypertension and hypotension?
178. Explain the importance of the nervous and humoral systems in reactivity?
179. Explain the mechanism of tumor and esophageal development?
180. Explain the violation of the incretory function of the pancreas?
181. General principles of disease classification?
182. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?

183. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
184. Explain the theories that clarify the etiology and their essence?
185. Explain the types and consequences of starvation?
186. Causes of disorders of the secretory function of the digestive organs?
187. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
188. Explain the changes in organs and systems during fever?
189. Explain the disorder of digestion in the stomach?
190. Explain the pathological effect of electric current on the body of animals?
191. Explain the causes, types and consequences of stasis, local anemia and heart attack?
192. Explain the disorder of appetite and thirst for water?
193. Explain the pathological effects of heat and cold on the body?
194. Dystrophic changes in the foci of inflammation and metabolic disorders?
195. Explain the role of experimental neuroses, the effect of endocrine glands on high nervous activity, the traces of the nervous system and the types of the nervous system in pathology ?.
196. Explain the development of the science of pathological physiology in Russia?
197. Explain hypoglycemic shock and the mechanism of its formation?
198. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
199. Explain the importance of heredity, constitution, breed, age and sex in pathology?
200. Explain the consequences of disturbances in the metabolism of minerals and water?

Written work questions for OB 2 (200 pieces)

1. Explain the consequences of a violation of the biochemical and physicochemical properties of blood?
2. Explain the pathogenesis of anaphylactic shock?
3. Metabolism in tumors. Experimental oncology and its importance?
4. Disorders of the endocrine function of the gonads?
5. Explain the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
6. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
7. Give an idea about the types of heart defects?
8. Explain biological causes as the cause of the disease?
9. Explain the effect of the organism on tumor growth?
10. Explain the pathological effects of adrenal insufficiency on animals?
11. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
12. Explain the metabolic disorders during fever?
13. Give an idea about the pathology of internal respiration, lack of oxygen?
14. Explain the mechanism of action of etiological causes?
15. What is anaplasia, give an idea of its types?
16. Explain circulatory disorders in pericardial and myocardial pathology?
17. Explain allergies, allergic diseases, infectious allergies, autoallergies?
18. Vascular reaction in inflammation. Explain exudate and its types?
19. Explain respiratory disorders in pulmonary pathology?
20. Explain the mechanical causes of disease?
21. When does hemotransfusion shock occur?
22. Explain the renal and extrarenal causes of urinary disorders?
23. How to study the science of animal pathophysiology, give them an idea?
24. What are hypo and hyperthermia, explain their periods and significance?
25. Explain the causes, types and consequences of changes in total blood volume?
26. Explain the science, functions and relationship of animal pathophysiology to other sciences?
27. Explain fever, etiopathogenesis, types, stages and significance? ()
28. Causes and consequences of impaired function of the liver to produce and excrete bile?
29. How to study the science of animal pathophysiology, give them an idea?
30. What are hypo and hyperthermia, explain their periods and significance?
31. Explain the causes, types and consequences of changes in total blood volume?
32. Explain the development of the science of pathological physiology in Russia?
33. Explain hypoglycemic shock and the mechanism of its formation?
34. Explain the causes, types and consequences of anemia, quantitative and qualitative changes in red blood cells?
35. Explain the mechanical causes of disease?
36. When does hemotransfusion shock occur?
37. Explain the renal and extrarenal causes of urinary disorders?

38. Give a general idea about nosology?
39. Explain atrophy, hypertrophy, regeneration and their types?
40. Explain the reasons for changes in the amount and composition of urine?
41 What is the mechanism of recovery of impaired functions based on?
42. Explain tumors, types, differences in development, biological properties?
43. Explain the pathology of the formation of white blood cells?
44. Give an idea about tanotogenesis and its periods?
45. Give an idea of the theories that explain the formation of tumors?
46. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
47. Etiology, give an idea of the types of etiological factors?
48. Explain that the tumor is related to the organism?
49. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
50. Explain the importance of heredity, constitution, breed, age and sex in pathology?
51. Explain the consequences of disturbances in the metabolism of minerals and water?
52. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
53. Explain that animals are not susceptible to infectious diseases?
54. Explain the mechanism of development of diabetes?
55. Explain the mechanism of development of hypertension and hypotension?
56. Explain the theories that clarify the etiology and their essence?
57. Explain the types and consequences of starvation?
58. What are the causes of dysfunction of the secretory organs of the digestive tract?
59. Explain the importance of the nervous and humoral systems in reactivity?
60. Explain the mechanism of tumor and esophageal development?
61. Explain the violation of the incretory function of the pancreas?
62. General principles of disease classification?
63. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?
64. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
65. Explain the pathological effects of electricity on animals?
66. Explain the causes, types and consequences of stasis, local anemia and heart attack?
67. Explain the disorder of appetite and thirst for water?
68. Explain the pathological effects of heat and cold on the body?
69. Dystrophic changes and metabolic disorders in the inflammatory focus?
70. Explain the role of experimental neuroses, the effect of endocrine glands on high nervous activity, the imprinting reactions of the nervous system and the types of nervous system in pathology ?.
71. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
72. Explain the changes in organs and systems during fever?
73. Explain the disorder of digestion in the stomach?
74. Explain the development of pathophysiology in Uzbekistan?
75. Explain the importance of arterial and venous hyperemia for the body?

76. Give an idea about the disturbance of sensory activity of the nervous system and its consequences?
77. Give an idea about disease and health?
78. Explain the local circulatory disorders and its types?
79. Changes caused by dysfunction of the pituitary gland?
80. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
81. Explain the effect of inflammation on the general reaction of the organism and the source of inflammation in the body?
82. Give an idea about the types of heart defects?
83. Explain allergies, allergic diseases, infectious allergies, autoallergies?
84. Vascular reaction in inflammation. Explain exudate and its types?
85. Explain the disruption of the respiratory process in lung pathology?
86. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
87. Explain the metabolic disorders during fever?
88. What is the concept of internal respiratory pathology, lack of oxygen?
89. Explain the mechanism of influence of etiological causes?
90. What is anaplasia, give an idea of its types?
91. Explain circulatory disorders in pericardial and myocardial pathology?
92. Explain the pathogenesis of anaphylactic shock?
93. Metabolism in tumors. Experimental oncology and its importance?
94. Disorders of the endocrine function of the gonads?
95. Explain biological causes as the cause of the disease?
96. Explain the effect of the organism on tumor growth?
97. Explain the pathological effects of adrenal insufficiency on animals?
98. Explain the pathological effects of disorders of bile formation on the body?
99. Explain inflammation, causes, stages, classification, significance and consequences?
100. Give an idea of the general directions that explain the origin of the disease?
101. Give an idea of the science of animal pathophysiology, its functions and its relationship with other sciences (anatomy, histology, biochemistry, biophysics, zoohygiene, nutrition and other sciences).
102. Give an idea of the pathophysiology of the circulatory system (large and small circulatory circles, heart, arteries, veins, capillaries, arterial and venous blood).
103. Give an idea about digestive disorders. Explain the pathophysiology of secretions of the digestive system (saliva, stomach, pancreas and intestinal juices) and enzymes (amylolytic, proteolytic, glycolytic).
104. Understand the pathophysiology of the digestive organs (kidneys, lungs, intestines).
105. Understand the mechanism of development of anaphylactic shock and changes in organ systems (humoral, cellular, lung, liver, etc.)
106. The science of animal pathophysiology and its methods of investigation (experiment, acute and chronic methods, their importance).

107. Give an idea about anaphylactic shock and allergic diseases (anaphylaxis, sensitization desensitization antianaphylaxis and idiosyncrasy, bronchial asthma, hay fever, etc.).
108. Explain the pathophysiology of digestion in the mouth (taking food into the mouth, chewing, salivating, swallowing).
109.Explain the pathophysiology of the formation and excretion of urine (phases of filtration and reabsorption, primary-pharmacological and final, actual urine, diuresis).
110. Give an idea of the typical pathological processes observed in tissues (atrophy, dystrophy, hypertrophy, hyperplasia, regeneration).
111. A brief history of the development of the science of pathophysiology. (Service of Hippocrates, Aristotle, K. Galen, Abu Ali ibn Sino, Vezaliy, V. Garvey, MVLomonosov, F. Majandi, I. Müller, K. Ludwig, VV Pashutin, ABFoxt., VVpodvitsotsky, K. Bernard and others).
112. Give an idea about arrhythmias (pulse, heart sounds and impulses, heart defects).
113. Pathophysiology of salivation (salivation, hypersalivation, hyposalivation).
114. Explain the quantitative and qualitative changes in urinary excretion (composition, color, specific gravity, pH i, osmotic pressure, albuminuria, glucosuria, hematuria).
115. Give an idea about the disorders of the movement of the pancreas (atony, hypotension, hyperkinesis).
116. Services of IMSechenov and IPPavlov in the field of pathophysiology (conditioned reflexes, small gastric formation, the idea of nervousness).
117. Pathological features of the heart muscle (excitability, permeability, refractoriness, automation and formation of biotoxins).
118. Disorders of secretion of gastric juice. (HCl, pepsin, cathepsin, chymosin, gelatinase, lipase, reflex, and neurochemical phases).
119. Pathology of urinary excretion and factors influencing it (renal, blood pressure, nerve and humoral control, blood volume in the body).
120. Sensitivity disorders (extraceptive, introceptive, proprioceptive, etc.).
121. Explain the theories that clarify the etiology (monocaualism, conditionalism, constitutionalism, hormones, nerve cells, afferent and efferent nerve fibers, receptors).
122.Explain the pathophysiology of the excitability and conduction properties of the heart (conduction system of the heart: Kiss-Fleka and Ashof-Tovar nodes, Giss ligament and legs, Purkinje fibers).
123. Explain the consequences of the violation of the secretion of pancreatic juice (composition, importance, pH i, enzymes, neuro-humoral pathway).
124. Understand the pathological changes of urinary excretion (renal and extrarenal causes)
125. Explain the general causes (mechanical, physical, chemical, biological) that disrupt the nervous system.
126. Give an idea about the disorders of blood formation (shaped elements, plasma, serum, homeostasis).

127. Explain the violation of the automatic and refractory properties of the heart (myogenic and neurogenic theory, Stannius connections, absolute and relative refractoriness, extrasystole, compensatory pause).
128. Understand the barrier properties of the organism (skin, wool, hooves, lysozyme, mine, placenta, etc.).
129. Understand the consequences of impaired spinal function (disruption of spinal centers, structure, reflex and conduction pathways).
130. Explain the violation of the physicochemical properties of blood (color, taste, specific gravity, pH, osmotic and oncotic pressure, reaction, buffering).
131. Understand the pathophysiology (color, pH i, specific gravity, enzymes, neuro-humoral pathway) of the composition, importance, separation and administration of intestinal juice.
132. Give an idea about hypertensive diseases (productivity, hormonal, arteriosclerotic, etc.)
133. Give an idea of the pathophysiology of brain activity (elongated brain, variola bridge, cerebellum, midbrain, cerebellum, septum and cortex of the large hemispheres).
134. Understand the concept of antigen and antibody. Explain allergens and anaphyloctogens, their distribution (blood plasma and serum, willow, walnut, wormwood, flowers, dust).
135. Explain the causes (mechanical, hemolytic, infectious, invasive, etc.) that disrupt the ability of the liver to produce bile.
136. Understand the pathophysiology of gastric digestion (structure of the gastric wall, the main, lining and accessory gland cells; motility).
137. Give an idea of hypertrophy and its types (worker, vakat, vicar).
138. Give an idea of the pathophysiology of the cerebellum and midbrain (understanding of the centers, location, reflex and conductive functions).
139. Explain the pathology of blood reaction, buffering, osmotic and oncotic pressures (environment, acidosis, alkalosis, hemoglobin, carbonate and phosphate buffer systems, plasma proteins).
140. Understand the theories (embryonic bud, exposure, chemistry and biology) that explain the formation of tumors.
141. Understand the violation of the physicochemical properties of blood (pH, buffer, surface tension, etc.).
142. Give an idea about hypo and hyperthermia. Heating stages (about stages 3 and 4, stages 1-2-3).
143. Give an idea of the pathology of the secretory and incretory properties of glands with mixed activity (pancreas, gonads, secretions, sperm and egg cells, hormones).
144. Give an idea of the main stages of development of pathology (animism, malignant zinc, humoral, solid, yatrophysical and yatra chemical, cellular, nervous, etc.).
145. Understand the pathophysiology of erythrocytes (shape, composition, function, erythrocytosis, erythropenia, poikilocytosis, anisocytosis).
146. Explain the causes of changes in the speed of blood flow in the arteries (heart function, muscle contraction, intravenous capillaries, negative pressure in the chest).

147. Give an idea about adrenal endocrine dysfunction and stress (adrenaline, noradrenaline, androids, estrogens, glucocorticoids, mineralcorticoids).
148. Explain the pituitary gland and its pathophysiology (hypofunction, hyperfunction, dysfunction).
149. Give an understanding of the pathophysiology of blood platelets (shape, function, thrombocytosis, thrombopenia).
150. Explain arterial and venous hyperemia (heat, sunlight, mechanical injury, constriction of vessels, stasis, induration, heart rate, blood pressure).
151. Understand the pathophysiology of digestion of nutrients in the small and large intestine (intestinal juice, pancreatic juice, bile, digestion, absorption of nutrients, microflora).
152. The doctrine of pathogenesis. Give an idea of the ways and consequences of the disease (blood and lymph, nerve, friction, continuation).
153. Violation of the sensitivity of internal organs. (viceral, viceroviceral, vicerosezor) give an idea.
154. Leukocytosis, their types, causes and consequences (bacteria, parasitic worms, myeloid, viruses).
155. Explain arterial and venous hyperemia. (miapara6lytic, neurotonic, neuroparalitic, collateral).
156. Explain the pathophysiology of the digestive system (separation-secretory, excretory-excretory, motor-motor).
157. Give an idea of the chemical causes of the disease (inorganic, organic, natural, artificial).
158. Give an idea of the reactivity and resistance of the organism (nerve, endocrine, age, micro and macronutrients).
159. Explain the pathology of pancreatic juice secretion and incretory activity (trypsin, amylase, maltase, lipase, insulin, glucogon, lipocoin)
160. Give an idea of the classification of inflammation (alternative, exudative-emigrant, proliferative, normergic, hyperergic, hypergic, etc.).
161. Explain the pathological changes in leukocytes (granulocytes, agranulocytes, basophils, eosinophils, neutrophils, lymphocytes, monocytes)
362. Explain the consequences of circulatory disorders in various organs (thrombosis, stasis, anemia, hyperemia).
163. Understand the violation of the sensory properties of the nervous system (hyposthesia and anesthesia excitability, mobility, inertia, weakness).
164. Explain the classification of inflammation depending on the morphological and immunological characteristics of the organism and the consequences of inflammation (alternative, exudative, proliferative, normergic, hyperergic, hypergic, complete healing, scarring or non-healing).
165. Explain the physical causes of disease (light, heat and cold, the effect of atmospheric pressure, the effect of electric current)
166. Give an idea of the theories (nutritional, biological, physicochemical, etc.) that explain the inflammatory process.

167. Give an idea of the pathophysiology of the digestive organs (kidneys, lungs, intestines).
168. Explain the mechanism of development of anaphylactic shock and changes in organ systems (humoral, cellular, lung, liver, etc.)
169. Give an idea about hypertensive diseases (productivity, hormonal, arteriosclerotic, etc.)
170. Give an idea of anaphylactic shock and allergic diseases (anaphylaxis, sensitization desensitization antianaphylaxis and idiosyncrasy, bronchial asthma, hay fever, etc.).
171. Give an idea of the pathophysiology of salivation (salivation, hypersalivation, hyposalivation).
172. Give an idea of the pathology of urinary excretion and the factors influencing it (blood pressure, nervous and humoral control, blood volume in the body).
173. Explain the pathological changes of the shaped elements of the blood (erythrocytosis, erythropenia, leukocytosis, leukopenia, thrombocytosis, thrombopenia).
174. Explain the pathophysiology of endocrine activity of the pineal gland and pituitary glands (serotonin, melotonin, TV lymphocytes).
175. Give an idea of the pathology of the formation and excretion of bile (glycocolate and tauraholate acids, bilirubin and biliverdin pigments).
176. Give an idea of the barrier properties of the organism (skin, wool, hooves, lysozyme, mine, placenta, etc.).
177. Give an idea of the brief history of the development of the science of pathophysiology. (Service of Hippocrates, Aristotle, K. Galen, Abu Ali ibn Sino, Vezaliy, V. Garvey, MVLomonosov, F. Majandi, I. Müller, K. Ludwig, VV Pashutin, ABFoxt., VV Podvitsotsky, K. Bernard and others).
178. Explain the pathophysiology of brain activity (elongated brain, varioli bridge, cerebellum, midbrain, cerebellum, septum, and cerebral cortex).
179. Understand the pathophysiology of the composition, importance, secretion and administration of intestinal juice (color, pH i, specific gravity, enzymes, neuro-humoral pathway).
180. Give an idea of the main stages of development of pathology (animism, malignant zinc, humoral, solid, iatrophysical and yatra chemistry, cellular, nervousism, etc.).
181. Give an idea about hypo and hyperthermia. Heating stages (about stages 3 and 4, stages 1-2-3).
182. Understand the theories that explain the formation of tumors (embryonic bud, exposure, chemistry and biology).
183. Give an idea of hypertrophy and its types (worker, vakat, vicar).
184. Understand the concept of antigen and antibody. Allergens and anaphyloctogens, their distribution (blood plasma and blood serum, willow, walnut, wormwood, flowers, dust).
185. Give an idea about arterial and venous hyperemia. (myaparalytic, neurotonic, neuroparalitic, collateral).
186. Explain the pathophysiology of the digestive system (separation-secretory, excretory-excretory, motor-motor).

187. Give an idea about hypertensive diseases (maximum, minimum, causes of hypertensive diseases).
188. Give an idea of the pituitary gland and its pathophysiology (hypofunction, hyperfunction, dysfunction).
189. Give an idea of changes in the number and quality of erythrocytes (shape, composition, function, erythrocytosis, erythropenia, poikilocytosis, anisocytosis).
190. Give an idea of arteries, venous hyperemia and their importance in the body (heat, sunlight, constriction of the arteries, stasis, induration).
191. Explain the physical causes of disease (light, heat and cold, the effect of atmospheric pressure, the effect of electric current)
192. Give an idea of the classification of inflammation (alternative, exudative-emigrant, proliferative, normergic, hyperergic, hypergic, etc.)
193. Explain the chemical causes of disease (inorganic, organic, natural, artificial)
194. Understand the pathophysiology of digestion of nutrients in the small and large intestine (intestinal juice, pancreatic juice, bile, digestion, absorption of nutrients, microflora).
195. The doctrine of pathogenesis. Give an idea of the ways and consequences of the disease (blood and lymph, nerve, friction, continuation).
196. Give an idea about the disorders of endocrine function of the adrenal glands and stress (adrenaline, noradrenaline, androids, estrogens, glucocorticoids, mineralcorticoids).
197. Explain the pathological changes of leukocytes. (granulocytes, agranulocytes, basophils, eosinophils, neutrophils, lymphocytes, monocytes)
198. Give an idea of the pathophysiology of metabolism. (assimilation and dissimilation, decomposition of nutrients).
199. Understand the classification of inflammation depending on the morphological and immunological characteristics of the organism and the consequences of inflammation (alternative, exudative, proliferative, normergic, hyperergic, hypergic, complete healing, scarring or non-healing).
200. Explain the violation of the sensitivity of the internal organs. (viceral, vicerovitseral, vicerosezor).

Written work questions for GP (500)

1. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
2. Explain fever, etiopathogenesis, types, stages and significance?
3. Causes and consequences of impaired liver production and excretion?
4. Explain the pathological effects of disorders of bile formation on the body?
5. Explain inflammation, causes, stages, classification, significance and consequences?
6. Give an idea of the general directions that explain the origin of the disease?
7 What is the mechanism of recovery of impaired functions based on?
8. Explain the tumors, types, differences in development, biological properties?
9. Explain the pathology of white blood cell formation?
10. Give an idea of the development of pathophysiology in Uzbekistan?
11. Explain the importance of arterial and venous hyperemia for the body?
12. Give an idea about the disorders of sensory activity of the nervous system and its consequences?
13. Give an idea of tanotogenesis and its periods?
14. Explain the theories that explain the formation of tumors?
15. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
16. Give an idea about illness and health?
17. Explain the local circulatory disorders and its types?
18. Changes caused by dysfunction of the pituitary gland?
19. Give a general idea about nosology?
20. Explain atrophy, hypertrophy, regeneration and their types?
21. Explain the reasons for changes in the amount and composition of urine?
22. Etiology, give an idea of the types of etiological factors?
23. Explain that tumors are related to the organism?
24. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
25. Explain that animals are not susceptible to infectious diseases?
26. Explain the mechanism of development of diabetes?
27. Explain the mechanism of development of hypertension and hypotension?
28. Explain the importance of the nervous and humoral systems in reactivity?
29. Explain the mechanism of tumor and esophageal development?
30. Explain the violation of the incretory function of the pancreas?
31. General principles of disease classification?
32. Explain the effects of protein, carbohydrate, fat, and vitamin starvation?
33. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
34. Explain the theories that clarify the etiology and their essence?
35. Explain the types and consequences of starvation?
36. What are the causes of dysfunction of the secretory organs of the digestive tract?
37. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?

38. Explain the changes in organs and systems during fever?
39. Explain the disorder of digestion in the stomach?
40. Explain the pathological effects of electricity on animals?
41. Explain the causes, types and consequences of stasis, local anemia and heart attack?
42. Explain the disorder of appetite and thirst for water?
43. Explain the pathological effects of heat and cold on the body?
44. Dystrophic changes in the inflammatory focus and metabolic disorders?
45. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.
46. Explain the development of the science of pathological physiology in Russia?
47. Explain hypoglycemic shock and the mechanism of its formation?
48. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
49. Explain the importance of heredity, constitution, breed, age and sex in pathology?
50. Explain the consequences of disturbances in the metabolism of minerals and water?
51. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
52. Explain the pathogenesis of anaphylactic shock?
53. Metabolism in tumors. Experimental oncology and its importance?
54. Disorders of the endocrine function of the gonads?
55. Explain the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
56. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
57. Give an idea about the types of heart defects?
58. Explain biological causes as disease-causing causes?
59. Explain the effect of the organism on tumor growth?
60. Explain the pathological effects of adrenal insufficiency on animals?
61. Give an idea about general adaptive syndrome or G. Sele doctrine?
62. Explain the metabolic disorders during fever?
63. Give an idea about the pathology of internal respiration, lack of oxygen?
64. Explain the mechanism of action of etiological causes?
65. What is anaplasia, give an idea of its types?
66. Explain circulatory disorders in pericardial and myocardial pathology?
67. Explain allergies, allergic diseases, infectious allergies, autoallergies?
68. Vascular reaction in inflammation. Explain exudate and its types?
69. Explain respiratory disorders in pulmonary pathology?
70. Explain the mechanical causes of disease?
71. When does hemotransfusion shock occur?
72. Explain the renal and extrarenal causes of urinary disorders?
73. How to study the science of animal pathophysiology, give them an idea?
74. What are hypo and hyperthermia, explain their periods and significance?
75. Explain the causes, types and consequences of changes in total blood volume?

76. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
77. Explain fever, etiopathogenesis, types, stages and significance?
78. Causes and consequences of impaired hepatic function of the liver?
79. How to study the science of animal pathophysiology, give them an idea?
82. What are hypo and hyperthermia, explain their periods and significance?
81. Explain the causes, types and consequences of changes in total blood volume?
82. Explain the development of the science of pathological physiology in Russia?
83. Explain hypoglycemic shock and the mechanism of its formation?
84. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
85. Explain the mechanical causes of disease?
86. When does hemotransfusion shock occur?
87. Explain the renal and extrarenal causes of urinary disorders?
88. Give a general idea about nosology?
89. Explain atrophy, hypertrophy, regeneration and their types?
90. Explain the reasons for changes in the amount and composition of urine?
91 What is the mechanism of recovery of impaired functions based on?
92. Explain the tumors, types, differences in development, biological properties?
93. Explain the pathology of the formation of white blood cells?
94. Give an idea about tanotogenesis and its periods?
95. Give an idea of the theories that explain the formation of tumors?
96. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
97. Etiology, give an idea of the types of etiological factors?
98. Tumor, explain how tumors are related to the organism?
99. Common causes of dysfunction of the nervous system, pathology of the upper nervous system and reticular formation?
100. Explain the importance of heredity, constitution, breed, age and sex in pathology?
101. Explain the consequences of disturbances in the metabolism of minerals and water?
102. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
103. Explain that animals are not susceptible to infectious diseases?
104. Explain the mechanism of development of diabetes?
105. Explain the mechanism of development of hypertension and hypotension?
106. Explain the theories that clarify the etiology and their essence?
107. Explain the types and consequences of starvation?
108. What are the causes of disorders of the secretory function of the digestive organs?
109. Explain the importance of the nervous and humoral systems in reactivity?
110. Explain the mechanism of development of edema and inflammation?
111. Explain the violation of the incretory function of the pancreas?
112. General principles of disease classification?
113. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?

114. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
115. Explain the pathological effects of electricity on animals?
116. Explain the causes, types and consequences of stasis, local anemia and heart attack?
117. Explain the disorder of appetite and thirst for water?
118. Explain the pathological effects of heat and cold on the body?
119. Dystrophic changes in the foci of inflammation and metabolic disorders?
120. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the traces of the nervous system and the types of the nervous system in pathology ?.
121. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
122. Explain the changes in organs and systems during fever?
123. Explain the disorder of digestion in the stomach?
124. Explain the development of pathophysiology in Uzbekistan?
125. Explain the importance of arterial and venous hyperemia for the body?
126. Give an idea of the disorders of sensory activity of the nervous system and its consequences?
127. Give an idea about disease and health?
128. Explain the local circulatory disorders and its types?
129. Changes caused by dysfunction of the pituitary gland?
130. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
131 Explain the general organism reaction of inflammation and the effect of the inflammatory focus on the organism?
132. Give an idea about the types of heart defects?
133. Explain allergies, allergic diseases, infectious allergies, autoallergies?
134. Vascular reaction in inflammation. Explain exudate and its types?
135. Explain the disruption of the respiratory process in lung pathology?
136. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
137. Explain the metabolic disorders during fever?
138. What is the concept of internal respiratory pathology, lack of oxygen?
139. Explain the mechanism of influence of etiological causes?
140. What is anaplasia, give an idea of its types?
141. Explain circulatory disorders in pericardial and myocardial pathology?
142. Explain the pathogenesis of anaphylactic shock?
143. Metabolism in tumors. Experimental oncology and its importance?
144. Disorders of the endocrine function of the gonads?
145. Explain biological causes as disease-causing causes?
146. Explain the effect of the organism on tumor growth?
147. Explain the pathological effects of adrenal insufficiency on animals?
148. Explain the pathological effects of disorders of bile formation on the body?
149. Explain inflammation, causes, stages, classification, significance and consequences?

150. Give an idea of the general directions that explain the origin of the disease?
151. Explain the science, functions and relationship of animal pathophysiology with other sciences? (anatomy, physiology, biochemistry, biophysics, pathoanatomy, microbiology, virology, animal nutrition and clinical sciences),
152. Explain fever, etiopathogenesis, types, stages and significance?
153. Causes and consequences of impaired function of the liver to produce and excrete?
154. Explain the pathological effect on the body of disorders of the formation and excretion of bile?
155. Explain inflammation, causes, stages, classification, significance and consequences? (microorganisms, viruses, fungi, simple animals, helminths, alternative, exudative, infiltrative, normergic, hyperergic, hypergic)
156. The main stages of development of pathology (animism, humoral, solid, cellular, ytrophysical, ytrochemical, nervousism).
157 What is the mechanism of recovery of impaired functions based on?
158. Explain the tumors, types, differences in development, biological properties? (dangerous, safe, orgonoid and histoid, infiltrative, exponential, relapsing, anaplasia)
159. Explain the pathology of the formation of white blood cells? (myeloblasts, lymphoblasts, plasma cells, leukoformula)
160. Explain the development of pathophysiology in Uzbekistan?
161. Explain the importance of arterial and venous hyperemia for the organism?
162. Give an idea about the disturbance of sensory activity of the nervous system and its consequences?
163. Give an idea about tanotogenesis and its periods?
164. Give an idea of the theories that explain the formation of tumors?
165. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
166. Give an idea about disease and health?
167. Explain the local circulatory disorders and its types?
168. Changes caused by dysfunction of the pituitary gland?
169. Give a general idea about nosology?
170. Explain atrophy, hypertrophy, regeneration and their types?
171. Explain the reasons for changes in the amount and composition of urine?
172. Etiology, give an idea of the types of etiological factors?
173. Tumor, explain how tumors are related to the organism?
174. Common causes of disorders of the nervous system, pathology of the upper nervous system and reticular formation?
175. Explain that animals are not susceptible to infectious diseases?
176. Explain the mechanism of development of diabetes?
177. Explain the mechanism of development of hypertension and hypotension?
178. Explain the importance of the nervous and humoral systems in reactivity?
179. Explain the mechanism of tumor and esophageal development?
180. Explain the violation of the incretory function of the pancreas?
181. General principles of disease classification?
182. Explain the consequences of starvation with protein, carbohydrates, fats, vitamins?

183. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
184. Explain the theories that clarify the etiology and their essence?
185. Explain the types and consequences of starvation?
186. Causes of disorders of the secretory function of the digestive organs?
187. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
188. Explain the changes in organs and systems during fever?
189. Explain the disorder of digestion in the stomach?
190. Explain the pathological effect of electric current on the body of animals?
191. Explain the causes, types and consequences of stasis, local anemia and heart attack?
192. Explain the disorder of appetite and thirst for water?
193. Explain the pathological effects of heat and cold on the body?
194. Dystrophic changes in the foci of inflammation and metabolic disorders?
195. Explain the role of experimental neuroses, the effect of endocrine glands on high nervous activity, the traces of the nervous system and the types of the nervous system in pathology ?.
196. Explain the development of the science of pathological physiology in Russia?
197. Explain hypoglycemic shock and the mechanism of its formation?
198. Explain the causes, types and consequences of anemia, changes in the number and quality of red blood cells?
199. Explain the importance of heredity, constitution, breed, age and sex in pathology?
200. Explain the consequences of disturbances in the metabolism of minerals and water?
201. Explain the consequences of violation of the biochemical and physicochemical properties of blood?
202. Explain the pathogenesis of anaphylactic shock?
203. Metabolism in tumors. Experimental oncology and its importance?
204. Disorders of the endocrine function of the gonads?
205. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
206. Explain the general reaction of the organism to inflammation and the effect of the source of inflammation on the body?
207. Give an idea about the types of heart defects?
208. Explain biological causes as disease-causing causes?
209. Explain the effect of the organism on tumor growth?
210. Explain the pathological effects of adrenal insufficiency on animals?
211. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
212. Explain the metabolic disorders during fever?
213. What is the concept of internal respiratory pathology, lack of oxygen?
214. Explain the mechanism of influence of etiological causes?
215. What is anaplasia, give an idea of its types?
216. Explain circulatory disorders in pericardial and myocardial pathology?
217. Explain allergies, allergic diseases, infectious allergies, autoallergies?

218. Vascular reaction in inflammation. Explain exudate and its types?
219. Explain the disruption of the respiratory process in lung pathology?
220. Explain the mechanical causes of disease?
221. When does hemotransfusion shock occur?
222. Explain the renal and extrarenal causes of urinary disorders?
223. How to study the science of animal pathophysiology, give them an idea?
224. What are hypo and hyperthermia, explain their periods and significance?
225. Explain the causes, types and consequences of changes in the total amount of blood?
226. Explain the science of animal pathophysiology, its functions and its relationship with other sciences?
227. Explain fever, etiopathogenesis, types, stages and significance? ()
228. Causes and consequences of impaired function of the liver to produce and excrete bile?
229. How to study the science of animal pathophysiology, give them an idea?
230. What are hypo and hyperthermia, explain their periods and significance?
231. Explain the causes, types and consequences of changes in the total amount of blood?
232. Explain the development of the science of pathological physiology in Russia?
233. Explain hypoglycemic shock and the mechanism of its formation?
234. Explain the causes, types and consequences of anemia, quantitative and qualitative changes in red blood cells?
235. Explain the mechanical causes of disease?
236. When does hemotransfusion shock occur?
237. Explain the renal and extrarenal causes of urinary disorders?
238. Give a general idea about nosology?
239. Explain atrophy, hypertrophy, regeneration and their types?
240. Explain the reasons for changes in the amount and composition of urine?
241 What is the mechanism of recovery of impaired functions based on?
242. Explain the tumors, types, differences in development, biological properties?
243. Explain the pathology of the formation of white blood cells?
244. Give an idea about tanotogenesis and its cycles?
245. Give an idea of the theories that explain the formation of tumors?
246. Explain the causes and consequences of dysfunction of the renal capillaries and capillaries?
247. Etiology, give an idea of the types of etiological factors?
248. Tumor, explain how tumors are related to the organism?
249. Common causes of disorders of the nervous system, pathology of the upper nervous system and reticular formation?
250. Explain the importance of heredity, constitution, breed, age and sex in pathology?
251. Explain the consequences of disturbances in the metabolism of minerals and water?
252. Explain the consequences of violation of the biochemical and physicochemical properties of blood?

253. Explain that the organism of animals is not susceptible to infectious diseases?
254. Explain the mechanism of development of diabetes?
255. Explain the mechanism of development of hypertension and hypotension?
256. Explain the theories that clarify the etiology and their essence?
257. Explain the types and consequences of starvation?
258. What are the causes of disorders of the secretory function of the digestive organs?
259. Explain the importance of the nervous and humoral systems in reactivity?
260. Explain the mechanism of tumor and esophageal development?
261. Explain the violation of the incretory function of the pancreas?
262. General principles of disease classification?
263. Explain the hunger and consequences of protein, carbohydrate, fat, continental starvation?
264. Explain the causes and consequences of arrhythmia, tachycardia and bradycardia?
265. Explain the pathological effect of electric current on the body of animals?
266. Explain the causes, types and consequences of stasis, local anemia and heart attack?
267. Explain the disorder of appetite and thirst for water?
268. Explain the pathological effects of heat and cold on the body?
269. Dystrophic changes and metabolic disorders in the inflammatory focus?
270. Explain the role of experimental neuroses, the effect of endocrine glands on the activity of the upper nervous system, the imprinting reactions of the nervous system and the types of the nervous system in pathology ?.
271. Explain the pathological effect of changes in the composition of soil, water and atmospheric air on the body?
272. Explain the changes in organs and systems during fever?
273. Explain the disorder of digestion in the stomach?
274. Explain the development of pathophysiology in Uzbekistan?
275. Explain the importance of arterial and venous hyperemia for the organism?
276. Give an idea of the disorders of sensory activity of the nervous system and its consequences?
277. Give an idea about disease and health?
278. Give an idea of local circulatory disorders and its types?
279. Changes caused by dysfunction of the pituitary gland?
280. What is the mechanism of anaphylaxis, sensitization, antianaphylaxis, desensitization?
281. Explain the general inflammatory reaction of inflammation and the effect of the inflammatory focus on the body?
282. Give an idea about the types of heart defects?
283. Explain allergies, allergic diseases, infectious allergies, autoallergies?
284. Vascular reaction in inflammation. Explain exudate and its types?
285. Explain the violation of the respiratory process in lung pathology?
286. Give an idea about the general adaptive syndrome or the doctrine of G. Sele?
287. Explain the metabolic disorders during fever?
288. What is the concept of internal respiratory pathology, lack of oxygen?

289. Explain the mechanism of influence of etiological causes?
290. What is anaplasia, give an idea of its types?
291. Explain circulatory disorders in pericardial and myocardial pathology?
292. Explain the pathogenesis of anaphylactic shock?
293. Metabolism in tumors. Experimental oncology and its importance?
294. Disorders of the endocrine function of the gonads?
295. Explain biological causes as disease-causing causes?
296. Explain the effect of the organism on tumor growth?
297. Explain the pathological effect of adrenal insufficiency on animals?
298. Explain the pathological effects of disorders of bile formation on the body?
299. Explain inflammation, causes, stages, classification, significance and consequences?
300. Give an idea of the general directions that explain the origin of the disease?
301. Give an idea of the science of animal pathophysiology, its functions and its relationship with other sciences (anatomy, histology, biochemistry, biophysics, zoohygiene, nutrition and other sciences).
302. Give an idea of the pathophysiology of the circulatory system (large and small circulatory circles, heart, arteries, veins, capillaries, arterial and venous blood).
303. Give an idea about digestive disorders. Explain the pathophysiology of secretions of the digestive system (saliva, stomach, pancreas and intestinal juices) and enzymes (amylolytic, proteolytic, glycolytic).
304. Give an idea of the pathophysiology of the digestive organs (kidneys, lungs, intestines).
305. Explain the mechanism of development of anaphylactic shock and changes in organ systems (humoral, cellular, lung, liver, etc.)
306. The science of animal pathophysiology and its methods of investigation (experiment, acute and chronic methods, their importance).
307. Give an idea about anaphylactic shock and allergic diseases (anaphylaxis, sensitization desensitization antianaphylaxis and idiosyncrasy, bronchial asthma, hay fever, etc.).
308. Explain the pathophysiology of digestion in the mouth (taking food into the mouth, chewing, salivation, swallowing).
309.Explain the pathophysiology of the formation and excretion of urine (phases of filtration and reabsorption, primary-pharmacological and final, actual urine, diuresis).
310. Give an idea of the typical pathological processes observed in tissues (atrophy, dystrophy, hypertrophy, hyperplasia, regeneration).
311. A brief history of the development of the science of pathophysiology. (Service of Hippocrates, Aristotle, K. Galen, Abu Ali ibn Sino, Vezaliy, V. Garvey, MVLomonosov, F. Majandi, I. Müller, K. Ludwig, VV Pashutin, ABFoxt., VVpodvitsotsky, K. Bernard and others).
312. Give an idea about arrhythmias (pulse, heart sounds and impulses, heart defects).
313. Pathophysiology of salivation (salivation, hypersalivation, hyposalivation).

314. Explain the quantitative and qualitative changes in urine output (composition, color, specific gravity, pH i, osmotic pressure, albuminuria, glucosuria, hematuria).
315. Give an idea about the movement disorders of the pre-gastric compartments (atony, hypotension, hyperkinesis).
316. Services of IMSechenov and IPPavlov in the field of pathophysiology (conditioned reflexes, small gastric formation, the idea of nervousness).
317. Pathological features of the heart muscle (excitability, permeability, refractoriness, automation and formation of biocurrents).
318. Disorders of secretion of gastric juice. (HCl, pepsin, cathepsin, chymosin, gelatinase, lipase, reflex, and neurochemical phases).
319. Pathology of urinary excretion and factors influencing it (renal, blood pressure, nerve and humoral control, blood volume in the body).
320. Sensitivity disorders (extraceptive, introceptive, proprioceptive, etc.).
321. Explain the theories that clarify the etiology (monocaualism, conditionalism, constitutionalism, hormones, nerve cells, afferent and efferent nerve fibers, receptors).
322.Explain the pathophysiology of the excitability and conduction properties of the heart (conduction system of the heart: Kiss-Fleka and Ashof-Tovar nodes, Giss ligament and legs, Purkinje fibers).
323. Explain the consequences of the violation of the secretion of pancreatic juice (composition, importance, pH i, enzymes, neuro-humoral pathway).
324. Understand the pathological changes of urinary excretion (renal and extrarenal causes)
325. Explain the general causes (mechanical, physical, chemical, biological) that disrupt the functioning of the nervous system.
326. Give an idea about the disorders of blood formation (shaped elements, plasma, serum, homeostasis).
327. Explain the violation of the automatic and refractory properties of the heart (myogenic and neurogenic theory, Stannius connections, absolute and relative refractoriness, extrasystole, compensatory pause).
328. Understand the barrier properties of the organism (skin, wool, hooves, lysozyme, mine, placenta, etc.).
329. Understand the consequences of dysfunction of the spinal cord (disruption of spinal centers, structure, reflex and conduction pathways).
330. Explain the violation of the physicochemical properties of blood (color, taste, specific gravity, pH, osmotic and oncotic pressure, reaction, buffering).
331. Understand the pathophysiology (color, pH i, specific gravity, enzymes, neuro-humoral pathway) of the composition, importance, separation and administration of intestinal juice.
332. Give an idea about hypertensive diseases (productivity, hormonal, arteriosclerotic, etc.)
333. Give an idea of the pathophysiology of brain activity (elongated brain, varioli bridge, cerebellum, midbrain, cerebellum, septum and cortex of the large hemispheres).

334. Understand the concept of antigen and antibody. Explain allergens and anaphyloctogens, their distribution (blood plasma and serum, willow, walnut, wormwood, flowers, dust).
335. Explain the causes (mechanical, hemolytic, infectious, invasive, etc.) that disrupt the ability of the liver to produce bile.
336. Understand the pathophysiology of digestion of food in the stomach (structure of the stomach wall, the main, lining and accessory glandular cells in it; motility).
337. Give an idea of hypertrophy and its types (worker, vakat, vicar).
338. Give an idea of the pathophysiology of the cerebellum and midbrain (understanding of the centers, location, reflex and conductive functions).
339. Explain the pathology of blood reaction, buffering, osmotic and oncotic pressures (environment, acidosis, alkalosis, hemoglobin, carbonate and phosphate buffer systems, plasma proteins).
340. Explain the theories (embryonic bud, exposure, chemistry and biology) that explain the formation of tumors.
341. Understand the violation of the physicochemical properties of blood (pH, buffer, surface tension, etc.).
342. Give an idea about hypo and hyperthermia. Heating stages (about stages 3 and 4, stages 1-2-3).
343. Understand the pathology of the secretory and incretory properties of glands with mixed activity (pancreas, gonads, secretions, sperm and egg cells, hormones).
344. Give an idea of the main stages of development of pathology (animism, malignant zinc, humoral, solid, yatrophysical and yatra chemistry, cellular, nervousism, etc.).
345. Understand the pathophysiology of erythrocytes (shape, composition, function, erythrocytosis, erythropenia, poikilocytosis, anisocytosis).
346. Explain the causes of changes in the speed of blood flow in the arteries (heart function, muscle contraction, intravenous capillaries, negative pressure in the chest).
347. Give an idea about the disorders of endocrine function of the adrenal glands and stress (adrenaline, noradrenaline, androids, estrogens, glucocorticoids, mineralcorticoids).
348. Explain the pituitary gland and its pathophysiology (hypofunction, hyperfunction, dysfunction).
349. Give an understanding of the pathophysiology of blood platelets (shape, function, thrombocytosis, thrombopenia).
350. Explain arterial and venous hyperemia (heat, sunlight, mechanical injury, constriction of vessels, stasis, induration, heart rate, blood pressure).
351. Understand the pathophysiology of digestion of nutrients in the small and large intestine (intestinal juice, pancreatic juice, bile, digestion, absorption of nutrients, microflora).
352. The doctrine of pathogenesis. Give an idea of the ways and consequences of the disease (blood and lymph, nerve, friction, continuation).
353. Violation of the sensitivity of internal organs. (viceral, viceroviceral, vicerosezor) give an idea.

354. Leukocytosis, their types, causes and consequences (bacteria, parasitic worms, myeloid, viruses).
355. Explain arterial and venous hyperemia. (miapara6lytic, neurotonic, neuroparalitic, collateral).
356. Explain the pathophysiology of the digestive system (separation-secretory, excretory-excretory, motor-motor).
357. Give an idea of the chemical causes of the disease (inorganic, organic, natural, artificial).
358. Give an idea of the reactivity and resistance of the organism (nerve, endocrine, age, micro and macronutrients).
359. Explain the pathology of pancreatic juice secretion and incretory activity (trypsin, amylase, maltase, lipase, insulin, glucogon, lipocoin)
360. Give an idea of the classification of inflammation (alternative, exudative-emigrant, proliferative, normergic, hyperergic, hypergic, etc.).
361. Understand the pathological changes in leukocytes (granulocytes, agranulocytes, basophils, eosinophils, neutrophils, lymphocytes, monocytes)
362. Explain the consequences of circulatory disorders in various organs (thrombosis, stasis, anemia, hyperemia).
363. Understand the violation of the sensory properties of the nervous system (hypostasis and anesthesia excitability, mobility, inertia, weakness).
364. Explain the classification of inflammation depending on the morphological and immunological characteristics of the organism and the consequences of inflammation (alternative, exudative, proliferative, normergic, hyperergic, hypergic, complete recovery, scarring or non-healing).
365. Explain the physical causes of disease (light, heat and cold, the effect of atmospheric pressure, the effect of electric current)
366. Give an idea of the theories (nutritive, biological, physicochemical, etc.) that explain the inflammatory process.
367. Give an idea of the pathophysiology of the digestive organs (kidneys, lungs, intestines).
368. Explain the mechanism of development of anaphylactic shock and changes in organ systems (humoral, cellular, lung, liver, etc.)
369. Give an idea about hypertensive diseases (productivity, hormonal, arteriosclerotic, etc.)
370. Give an idea of anaphylactic shock and allergic diseases (anaphylaxis, sensitization desensitization antianaphylaxis and idiosyncrasy, bronchial asthma, hay fever, etc.).
371. Give an idea of the pathophysiology of salivation (salivation, hypersalivation, hyposalivation).
372. Understand the pathology of urinary excretion and the factors influencing it (blood pressure, nervous and humoral control, blood volume in the body).
373. Explain the pathological changes of the shaped elements of the blood (erythrocytosis, erythropenia, leukocytosis, leukopenia, thrombocytosis, thrombopenia).

374. Explain the pathophysiology of endocrine activity of the pineal gland and pituitary glands (serotonin, melotonin, TV lymphocytes).
375. Give an idea of the pathology of the formation and excretion of bile (glycoholate and tauraholate acids, bilirubin and biliverdin pigments).
376. Understand the barrier properties of the organism (skin, wool, hooves, lysozyme, mine, placenta, etc.).
377. Give a brief history of the development of the science of pathophysiology. (Service of Hippocrates, Aristotle, K. Galen, Abu Ali ibn Sino, Vezaliy, V. Garvey, MVLomonosov, F. Majandi, I. Müller, K. Ludwig, VV Pashutin, ABFoxt., VV Podvitsotsky, K. Bernard and others).
378. Explain the pathophysiology of brain activity (elongated brain, varioli bridge, cerebellum, midbrain, cerebellum, septum, and cortex of the large hemispheres).
379. Understand the pathophysiology of the composition, importance, secretion and administration of intestinal juice (color, pH i, specific gravity, enzymes, neuro-humoral pathway).
380. Give an idea of the main stages of development of pathology (animism, malignant zinc, humoral, solid, yatrophysical and yatra chemical, cellular, nervous, etc.).
381. Give an idea about hypo and hyperthermia. Heating stages (about stages 3 and 4, stages 1-2-3).
382. Give an idea of the theories that explain the formation of tumors (embryonic bud, exposure, chemistry and biology).
383. Give an idea of hypertrophy and its types (worker, vakat, vicar).
384. Understand the concept of antigen and antibody. Allergens and anaphyloctogens, their distribution (blood plasma and blood serum, willow, walnut, wormwood, flowers, dust).
385. Give an idea about arterial and venous hyperemia. (myaparalytic, neurotonic, neuroparalitic, collateral).
386. Explain the pathophysiology of the digestive system (separation-secretory, excretory-excretory, motor-motor).
387. Give an idea about hypertensive diseases (maximum, minimum, causes of hypertensive diseases).
388. Give an idea of the pituitary gland and its pathophysiology (hypofunction, hyperfunction, dysfunction).
389. Give an idea of changes in the number and quality of erythrocytes (shape, composition, function, erythrocytosis, erythropenia, poikilocytosis, anisocytosis).
390. Give an idea about arterial and venous hyperemia and their importance in the body (heat, sunlight, constriction of the arteries, stasis, induration).
391. Understand the physical causes of disease (light, heat and cold, the effect of atmospheric pressure, the effect of electric current)
392. Give an idea of the classification of inflammation (alternative, exudative-emigrant, proliferative, normergic, hyperergic, hypergic, etc.)
393. Explain the chemical causes of the disease (inorganic, organic, natural, artificial)

394. Understand the pathophysiology of digestion of nutrients in the small and large intestine (intestinal juice, pancreatic juice, bile, digestion, absorption of nutrients, microflora).
395. The doctrine of pathogenesis. Give an idea of the ways and consequences of the disease (blood and lymph, nerve, friction, continuation).
396. Give an idea about the disorders of endocrine function of the adrenal glands and stress (adrenaline, noradrenaline, androids, estrogens, glucocorticoids, mineralcorticoids).
397. Explain the pathological changes of leukocytes. (granulocytes, agranulocytes, basophils, eosinophils, neutrophils, lymphocytes, monocytes)
398. Give an idea of the pathophysiology of metabolism. (assimilation and dissimilation, decomposition of nutrients).
399. Understand the classification of inflammation depending on the morphological and immunological characteristics of the organism and the consequences of inflammation (alternative, exudative, proliferative, normergic, hyperergic, hypergic, complete healing, scarring or non-healing).
400. Explain the violation of the sensitivity of the internal organs. (viceral, vicerovitseral, vicerosezor).
401. Understand the pathophysiology of endocrine activity of the thyroid gland and thyroid gland (thyroxine, triiodothyronine, triocalcitron, parathyroid hormone, kyretinism, mexedema).
402. Give an idea of experimental neuroses and their periods? (behavior, agitation, braking, moving process).
403. Give an idea of the definition of erythrocytes and their quantitative and qualitative changes (polycythemia, reticulocytes, polychromatophilic erythrocytes, macrocytes, hyperchromic and hypochromic erythrocytes).
404. Give an idea of the pathophysiology of the endocrine activity of the adrenal glands (adrenaline, norepinephrine, androgens, estrogens, gestogens).
405. Give an idea of health and disease, an understanding of the periods of illness (latent, prodromal, clinical and consequential)?
406. Understand breathing (hypoxia, hypoxemia, hypocapnia, gas alkalosis, mountain and caisson disease, compensatory properties of the organism) during physical activity and in conditions of changing atmospheric pressure.
407. Explain the rate of respiration and the factors affecting it and the pathology of the pleura? (emphysema, atelectasis, pneumonia, pleurisy).
408. Explain the pathophysiology of endocrine activity of the pituitary gland (somatotropic, AKTG, thyrotropic, gonadotropic, luteinotropic, vasopressin, antidiuretic, oxytocin, intermedine).
409. Give an idea of the reactivity of the organism and its role in pathology (lymph node, blood, etc.)?
410. Explain the humoral and cellular theory of immunity. (nerve, fluid, antigen, and antibody).

411. Understand the pathophysiology of endocrine activity of the pancreas (insulin, glycogen, lipocaine, vagotonin, diabetes, hyperglycemia, glucosuria, comatose state).
412. Understand the pathological role of the liver in metabolism (processes of decomposition and synthesis, participation in depot, hematopoiesis and heat metabolism).
413. Fever and factors influencing it (isotherm, homothermic and poikilotherm animals, chemical and physical thermoregulation, fever, types of fever).
414. Give an idea of the pathology of the gonads (androgens, estrogens, progesterone, relaxin, hermaphroditism, eunuchoidism, infantilism).
415. Give an idea of the theories (nutritional, biological, physicochemical, etc.) that explain the inflammatory process.
416. Theories explaining blood formation and its disorders (hematopoiesis, erythropoiesis, leukopoiesis, thrombopoiesis, glomerulus, lymph nodes, unitary and dualistic).
417. Describe disease and health. Explain the practical significance of death? (disease, health, description of diseases of farm animals)
418. Explain the pathophysiology of endocrine activity of the pineal gland and pituitary glands (serotonin, melotonin, TV lymphocytes).
419. Explain the pathophysiology of endocrine activity of the pineal gland and pituitary glands (serotonin, melotonin, T. V lymphocytes).
420. Explain the pathophysiology of the autonomic nervous system (sympathetic and parasympathetic nervous systems, the difference from the somatic nervous system).
421. Humoral and cellular theory of immunity. (nerve, fluid, antigen, and antibody).
422. Explain the pathophysiology of digestion of nutrients in the large intestine (type, number, importance of microorganisms, breakdown, synthesis, absorption of nutrients).
423. Explain the pathology of the endocrine activity of the genitals (endocrine glands of males and females).
424. The role of the lungs in the respiratory process, lung diseases (pneumonia, atelectasis, emphysema
425. Explain the pathophysiology of endocrine activity of the thyroid gland and thyroid gland (thyroxine, triiodothyronine, triocalcitron, parathyroid hormone, kyretinism, mexedema).
426. Pathophysiology of endocrine and exocrine glands (hyper and hypofunction of endocrine and exocrine glands, dysfunction).
427. Factors influencing changes in erythrocyte resistance. (hemolysis, maximum and minimum resistance, fast and slow deposition of erythrocytes in animals, Panchenkov apparatus, Nevedov test tube, hypotonic solutions).
428. Biological causes of disease (bacteria, viruses, fungi, parasites, simple animals).
429. Respiration during physical activity and in conditions of changing atmospheric pressure (hypoxia, hypoxemia, hypocapnia, gas alkalosis, mountain and caisson disease, compensatory properties of the organism).

430. Lack of water and minerals for the body (macro and micronutrients, the amount of water consumed by animals, edema, constipation).
431. Explain the pathophysiology of endocrine activity of the adrenal glands (adrenaline, norepinephrine, androgens, estrogens, gestogens).
432. Explain the periods of illness (latent, prodromal, clinical and consequential), the concept of health and disease?
433. Humoral and cellular theory of immunity. (nerve, fluid, antigen, and antibody).
434. Theories explaining the inflammatory process (nutritive, biological, physicochemical, etc.).
435. Theories explaining blood formation and its disorders (hematopoiesis, erythropoiesis, leukopoiesis, thrombopoiesis, glomerulus, lymph nodes, unitary and dualistic).
436. Explain respiratory rate and factors affecting it and pleural pathology? (emphysema, atelectasis, pneumonia, pleurisy).
437. The concept of pathophysiology of vitamins (water and fat-soluble vitamins, avitaminosis, polyavitaminosis, hypovitaminosis).
438. Explain the pathology of the gonads (androgens, estrogens, progesterone, relaxin, hermaphroditism, eunuchoidism, infantilism).
439. Pathological role of the liver in metabolism (processes of decomposition and synthesis, participation in depot, hematopoiesis and heat exchange).
440. Give an idea of the theories that clarify the etiology (monocaualism, conditionalism, constitutionalism, hormones, nerve cells, afferent and efferent nerve fibers, receptors).
441. Give an idea about hypertensive diseases (productivity, hormonal, arteriosclerotic, etc.)
442. Give an idea of anaphylactic shock and allergic diseases (anaphylaxis, sensitization desensitization antianaphylaxis and idiosyncrasy, bronchial asthma, hay fever, etc.).
443. Understand the barrier properties of the organism (skin, wool, hooves, lysozyme, mine, placenta, etc.).
444. Explain the physiology and pathology of blood reaction, buffering, osmotic and oncotic pressures (environment, acidosis, alkalosis, hemoglobin, carbonate and phosphate buffer systems, plasma proteins).
445. Give an idea of the pathophysiology of high nervous activity. (activity of the cerebral cortex and conditioned reflexes).
446. Give an idea of the reactivity and resistance of the organism (nerve, endocrine, age, micro and macronutrients).
447. Understand the pathophysiology of digestion of nutrients in the small and large intestine (intestinal juice, pancreatic juice, bile, digestion, absorption of nutrients, microflora).
448. Explain the chemical causes of disease (inorganic, organic, natural, artificial)
449. The doctrine of pathogenesis. Give an idea of the ways and consequences of the disease (blood and lymph, nerve, friction, continuation).
450. Understand the pathological changes of leukocytes. (granulocytes, agranulocytes, basophils, eosinophils, neutrophils, lymphocytes, monocytes)

451. Understand the classification of inflammation depending on the morphological and immunological properties of the organism and the consequences of inflammation (alternative, exudative, proliferative, normergic, hyperergic, hypergic, complete healing, scarring or non-healing).
452. Give an idea of experimental neuroses and their periods? (behavior, agitation, braking, moving process).
453. Give an idea of the pathophysiology of the endocrine activity of the adrenal glands (adrenaline, norepinephrine, androgens, estrogens, gestogens).
454. Give an idea of health and disease, an understanding of the periods of illness (latent, prodromal, clinical and consequential)?
455. Understand breathing (hypoxia, hypoxemia, hypocapnia, gas alkalosis, mountain and caisson disease, compensatory properties of the organism) during physical activity and in conditions of changing atmospheric pressure.
456. Explain the rate of respiration and the factors affecting it and the pathology of the pleura? (emphysema, atelectasis, pneumonia, pleurisy).
457. Understand the pathophysiology of endocrine activity of the pancreas (insulin, glycogen, lipocaine, vagotonin, diabetes, hyperglycemia, glucosuria, comatose state).
458. Fever and factors affecting it (isotherm, homothermic and poikilotherm animals, chemical and physical thermoregulation, fever, types of fever).
459. Explain the pathophysiology of endocrine activity of the pineal gland and pituitary glands (serotonin, melotonin, TV lymphocytes).
460. Explain the pathophysiology of endocrine activity of the pituitary gland (somatotropic, AKTG, thyrotropic, gonadotropic, luteinotropic, vasopressin, antidiuretic, oxytocin, intermedine).
461. Explain the pathology of the gonads (androgens, estrogens, progesterone, relaxin, hermaphroditism, eunuchoidism, infantilism).
462. What changes occur in the organs of the body as a result of inflammation?
463. What is the reason for the appearance of round, oozing, rigid knots around the affected area?
464. What is the effect of high atmospheric pressure on the body?
465. What poisons are included in industrial products (organochlorine, organophosphorus compounds, herbicides)?
466. Formed in the body as a result of metabolic disorders What is poisoning called?
467. What is a self-poisoning of an organism?
468. What are the names of toxic substances that affect the deposit and its composition?
469. Toxic substances affecting the heart what is it called
470. Low temperature What is the local image of the orgasm?
471. What is called the transformation of healthy tissue into tumor tissue??
472. In febrile fever, how many degrees does the body temperature rise above the upper limit of the body temperature of a healthy animal?
473. How many degrees per hour does the temperature of the corpse decrease in the first and subsequent days?

474. What is the absorption of hemolyzed koii between tissues?
475. What is the reason for a person to fall asleep for a long time and to slow down the metabolism in his body?
476. What is the name of the current that explains that there is only one cause of the disease, if it affects the body, the disease occurs?
477. What are the signs of a cold?
478. What is the name of the toxins released during the decomposition of microorganisms?
479. What is the name of the appearance of spots on a corpse?
480. What is the artificial slowing of vital processes in an animal body?
481. What are the theories that explain the etiology?
482. The passage of electric current through which organs is very dangerous for the organism?
483. If at the level of the tissue bubbles appear in the clear liquid, it is strong If the pain subsides, what is the degree of burning?
484. What are the types of colds?
485. What are the signs of a cold, when the organs begin to weaken, the formation of large wounds, the accumulation of toxins in them and the appearance of signs of general intoxication in the body?
486. What characterizes the general effect of high temperature on an organism? '
487. The effect of low atmospheric pressure on the organism of animals causes what disease?
488. What products of microorganisms, flora and fauna (toxins glucosides, saponins, alkaloids, insect and snake venoms) are included in poisons?
489. In the body itself What are the names of toxins that can cause damage to bacterial and bacterial processes?
Who is the founder of the science of pathophysiology and veterinary pathophysiology?
490. Indicate the scientists who have revived the organism, the method of complex resurrection?
491. What is an injury caused by an electric current?
492. Which answer is given by veterinary pathophysiologists?
493. Who beat the tumors in Samarkand?
494. What kind of animals are resistant to the poison?
495. Indicate the founders of cellular and srlidar theories?
496. What is the cause and mechanism of the disease and the mechanism of its development?
497. Canadian pathophysiologist Hans Sele showed which system plays a leading role in the pathogenesis of diseases?
498. The ability of an organism to respond to an influence in a physiological way what is it called
499. What is the name of the extremely low reactivity of the organism?
500. How much does oxygen uptake increase in fat starvation?

Test questions for OB 1 (200)

1. What is the name of the science that teaches the changes that occur in the body of the patient, the causes of the disease, the conditions, the mechanism of development, the consequences of the flow?

A. Epizootology
B. Pathological anatomy
D. Clinical diagnostics
E. Pathology physiology

2. What experiments are used in the study of pathological processes?

A. Chronic experiments
B. Auscultation, percussion, palpation
D. Acute experiments
E. Acute and chronic experiences

3. What is the name of the theory that explains the origin of the disease by connecting the divine forces?

A. Nervism
B. Animism
D. Humoral
E. Solidar

4. What is the name of the theory that explains the origin of the disease in relation to changes observed in cells?

A. Humoral
B. Yatroximik
D. Cellular
E.Solidar

5. Who is the founder of humoral theory?

ARVirxov
B. Democritus
DIPPavlov
E. Hippocrates

6. Who is the founder of the solitary theory?

ARVirxov
B. Hippocrates
DIPPavlov
E Democritus

7. Who is the founder of the science of pathological physiology?

AESLondon
BIIRavich
DAABogomoles
EVVPashutin

8. Who is the founder of the science of veterinary pathological physiology?

AVVPashutin
BIIRavich

DABogomoles
EESLondon

9. What is the general doctrine of disease called?

A. Pathogenesis
B. Etiology
D. Nosology
E. Pathology

10. What stages of the disease do you know?

A. Latent, prodromal, and clinical periods
B. Incubation, clinical, and termination periods
D. Incubation, prodromal, consequence, and end periods
E. Latent, prodromal, clinical, and concluding periods

11. Do you know the consequences of the disease?

A. The disease can be completely cured
B. Heals or dies from illness
D. The disease can be completely and partially cured
E. The disease ends in death

12. What is the recurrence of the disease in the body?

A. Tonatogenesis
B. Remission
D. Recidivism
E. Pathogenesis

13. What is the complete recovery of the body from disease?

A. Tonatogenesis
B. Remission
D. Sanogenesis
E. Pathogenesis

14. How many oC per hour does the temperature of the corpse decrease in the first and subsequent days?

A. The first day is 30, the following days are 0.30
B. The first day 20, the next days 0.50
D. The first day is 10, the next days are 0.20
E. The first day 40, the following days 0.40}

15. Agony - how long does a pre-death seizure last?

A. 2-3 left
B. 5-6 minutes
D.3-5 hours
E. A few hours

16. How long does clinical death last?

A. 5-6 minutes
B. 2-3 left
D. 3-5 hours
E. 10 left

17. Name the stages of death.

A. Agony, clinical and biological death
B. Clinical death
D. Biological death
E. Clinical and biological death

18. What is the mechanism of disease progression and development?

A. Pathogenesis
B. Sanogenesis
D. Tonatogenesis
E. Etiology

19. Who is the founder of the doctrine of stress?

A. Gans Selye
B. Foxt
D. Galen
EIPPavlov

20. What is the ability of an organism to respond physiologically to an influence?

A. Allergy
B. Resistance
D. Reactivity
E. Anaphylaxis

21. What is the level of resistance of the organism to pathogenic forces?

A. Allergy
B. Reactivity
D. Resistance
E. Anaphylaxis

22. What is called high reactivity of the organism?

A. Energy
B. Hyperglycemia
D. Dysergia
E. Hyperergy

23. What is called low reactivity of the organism?

A. Hyperglycemia
B. Hyperergy
D. Dysergia
E. Energy

24. What is the complete loss of reactivity of the organism?

A. Energy
B. Hyperglycemia
D. Dysergia
E. Hyperergy

25. What is the ability of an organism to respond to an impact involving physiological systems?

A. Reactivity
B. Resistance
D. Allergy

E. Anaphylaxis

26. What is the level of resistance of the organism to pathogenic forces?
A. Reactivity
B. Resistance
D. Allergy
E. Anaphylaxis
27. What is the complete loss of reactivity of the organism?
A. Hypoergia
B. Dysergia
D. Energy
E. Hyperergy
28. What is the deterioration of the reactivity of the organism?
A. Energy
B. Hypoergia
D. Hyperergy
E. Dysergia
29. What is the name of a separate system consisting of bone marrow, lymph nodes, reticular connective tissue cells in the spleen, endothelial cells, Kupfer cells in the liver and leukocytes?
A. Reticular-endothelial system
B. Humoral system
D. Neuro-humoral system
E. Endocrine system
30. What are the inactive cells that make up RES called?
A. Faglar
B. Macrophages
D. Microphages
E. Phagocytosis
31. What are the motile cells that make up RES called?
A. Macrophages
B. Microphages
D. Faglar
E. Phagocytosis
32. What is the process by which cells absorb and digest foreign substances entering the body?
A. Allergy
B. Phagocytosis
D. Immunity
E. Chemotaxis
33. What is called the absorption and absorption of liquids and solutes in the environment by the cell?
A. Phagocytosis
B. Allergy

D. Pinocytosis
E. Chemotaxis

34. What is the movement of a phagocyte towards a foreign substance?

A. Phagocytosis
B. Allergy
D. Chemotaxis
E. Immunity

35. What is called the attachment of a phagocyte to a foreign substance?

A. Attraction
B. Allergy
D. Phagocytosis
E. Chemotaxis

36. How do phagocytes digest foreign substances entering the body?

A. With false legs
B. With oils
D. With proteins
E. With enzymes

37. At what stage does the process of phagocytosis take place?

A. In three stages
B. In five stages
D. In two stages
E. In four stages

38. Who created the phagocytic theory of immunity and when?

ARKox (1881)
BIIMechnikov (1883)
DAABogomolets (1805)
EAAdo (1950)

39. What theories explain the formation of immunity?

A. Phagocytic, humoral theory
B. Humoral theory
D. Neuro-humoral theory
E. Physicochemical theory

40. What is the property of the organism to resist the action of various microorganisms that cause disease and their toxins?

A. Allergy
B. Immunity
D. Reactivity
E. Resistance

41. What is the type of hereditary immunity of an organism?

A. Congenital immunity
B. Acquired immunity
D. Active immunity
E. Passive immunity

42. What is the immunity that is formed during the life of an organism?

A. Acquired immunity
B. Congenital immunity
D. Active immunity
E. Passive immunity

43. What is the immunity that an organism develops after suffering from a certain infectious disease?

A. Naturally acquired immunity
B. Artificially acquired immunity
D. Active immunity
E. Passive immunity

44. What is the immunity created by vaccination by injecting vaccines and blood serum into the body?

A. Artificially acquired immunity
B. Naturally acquired immunity
D. Active immunity
E. Passive immunity

45. What is the immunity that develops in the body as a result of natural disease or vaccination with vaccines against the disease?

A. Congenital immunity
B. Acquired immunity
D. Active immunity
E. Passive immunity

46. What is the immunity created by the passage of immune cells through the mother's oral milk to a newborn animal or by the delivery of serum containing immune antibodies?

A. Active immunity
B. Acquired immunity
D. Passive immunity
E. Congenital immunity

47. What is the immunity formed against the toxins of microorganisms?

A. Acquired immunity
B. Antitoxic immunity
D. Congenital immunity
E. Passive immunity

48. What is the immunity that can ensure the complete cleansing of the body from infectious agents?

A. Passive immunity
B. Congenital immunity
D. Active immunity
E. Sterile immunity

49. What is immunity called, which does not ensure complete cleansing of the body from infectious agents?

A. Active immunity
B. Sterile immunity

D. Nosteril immunity
E. Congenital immunity

50. What are the substances that act on the immunocompetent organs of the body, forming antibodies and reacting with them?

A. Antibodies
B. Antigens
D. Allergens
E. Anophylactogen

51. What are the specific proteins that are produced in the immunocompetent organs of the body against antigens and react with them?

A. Antibodies
B. Antigens
D. Allergens
E. Anophylactogen

52 What are the substances that make the body hypersensitive to foreign substances?

A. Antigens
B. Allergens
D. Antibodies
E. Anophylactogen

53. What are the substances that can cause anaphylactic shock?

A. Antigens
B. Anophylactogen
D. Allergens
E. Antibodies

54. What is an increase in the body's sensitivity to certain nutrients and drugs?

A. Idiosyncrasy
B. Autoallergic
D. Allergic disease
E. Autoallergic disease

55. What is a disease that occurs suddenly due to an increase in the body's sensitivity to certain foreign substances and passes in the form of attacks?

A. Allergic diseases
B. Hereditary diseases
D. Congenital diseases
E. Infectious diseases

56. What are the protein molecules that accelerate antigen-antibody reactions in animal blood?

A. Interferon
B. Antibody
D. Antigen
E. Complement

57. What is the hypersensitivity of the organism to foreign substances?

A. Desensitization

B. Anaphylaxis
D. Sansibilization
E. Allergy

58. What is called hypersensitivity of the organism with special substances to cause anaphylaxis?

A. Desensitization
B. Anaphylaxis
D. Anaphylactic shock
E. Sensitization

59. What is the release of an animal from a state of sensitization?

A. Anaphylactic shock
B. Anaphylaxis
D. Sensitization
E. Desensitization

60. What is a circulatory disorder of an individual organ or part of it without changing the total amount of blood in the body?

A. Local circulatory disorders
B. Collaterial circulation
D. Decreased blood volume
E. General circulatory disorders

61. What is the increase in blood volume due to increased blood flow to organs and tissues?

A. Hyperemia
B. Ischemia
D. Arterial hyperemia
E. Venous hyperemia

62. What is a decrease in the amount of blood in a particular organ or part of it due to a decrease in blood flow in the veins?

A. Ischemia
B. Hyperemia
D. Collaterial circulation
E. Staz

63. What are the main symptoms of arterial hyperemia?

A. The organ turns reddish-purple, enlarges, becomes hot
B. The organ turns blue, enlarges, decreases in temperature
D. The organ turns red and blue, shrinks
E. The organ becomes pale, small, pale, and painful

64. What are the main symptoms of venous hyperemia?

A. The organ turns blue, enlarges, decreases in temperature.
B. The organ turns reddish-purple, enlarges, becomes hot
D. The organ turns red and blue, shrinks
E. The organ becomes pale, small, pale, and painful

65. What is the increase in the amount of blood flowing from an artery to an organ or part of it and the change that occurs when the amount of blood flowing does not change?

A. Venous hyperemia
B. Ischemia
D. Hyperemia
E. Arterial hyperemia

66. What are the main symptoms of ischemia?

A. The organ becomes pale, small, pale, and painful
B. The organ turns blue, enlarges, decreases in temperature
D.Organ turns red and blue, shrinks
E. The organ turns reddish-purple, enlarges, becomes hot

67. What is the cessation of blood flow in the capillaries or venous blood vessels of an organ?

A. Ischemia
B. Staz
D. Collaterial circulation
E. Hyperemia

68. What is the bleeding from a vein when the vessel wall is not damaged or their permeability is increased?

A. External bleeding
B. Hemorrhage
D. Diapedez
E. Internal bleeding

69. What is the name given to the fact that blood clots in the blood vessels of a living organism, forming blockages and resisting blood flow?

A. Blood clot
B. Thrombosis
D. Thrombogenesis
E. Thrombosis

70. What is called the clogging of blood and lymph vessels by certain particles that are not commonly found in the blood and lymph, but are brought in by the flow of these fluids?

A. Staz
B. Thrombosis
D. Embolism
E. Embol

71. What are the external signs of inflammation?

A. Redness, swelling, redness, pain, dysfunction
B. Swelling, fever, pain
D. Redness, swelling, pain
E. Swelling, pain, dysfunction

72. Who identified and interpreted the external signs of inflammation?

A. Sels and Galen

B. Sels and Parasels
D. Galen and Garvey
E. Hippocrates and Democritus

73. What are the main stages of inflammation?
A. Alteration, exudation and emigration, proliferation
B. Alteration, exudation, regeneration, and emigration
D. Dystrophy, exudation, regeneration, and emigration
E. Exudation, emigration, proliferation, and regeneration

74. How can inflammation occur?
A. Acute and chronic
B. Acute, moderately acute, chronic
D. Moderately acute
E. Chronic

75. What is the stage of inflammation characterized by tissue damage, dystrophy, disruption of its structure and function?
A. Proliferation
B. Exudation
D. Emigration
E. Alteration

76. When naming an inflamed tissue or organ, what Greek or Latin word is added to their name?
A. «oma» «iya»
B. «it» «iya»
D. «genesis» «iya»
E. «iya» «pir»

77. What is the inflammation that occurs in organisms with high reactivity?
A. Emigrant inflammation
B. Exudative inflammation
D. Hyperergic inflammation
E. Hypergic inflammation

78. What is the inflammation that occurs in organisms with low reactivity?
A. Hypergic inflammation
B. Exudative inflammation
D. Emigrant inflammation
E. Hyperergic inflammation

79. What is the release of a liquid portion of blood through the vascular wall of inflamed tissue?
A. Alteration
B. Exudation
D. Emigration
E. Proliferation

80. What is the release of leukocytes from the blood through the vascular wall of inflamed tissue?
A. Alteration

B. Emigration
D. Exudation
E. Proliferation

81. What is the proliferation of cellular elements in the site of inflammation?
A. Proliferation
B. Alteration
D. Emigration
E. Exudation

82. What is called inflammation, characterized by a predominance of dystrophy, necrosis and necrobiosis in tissues?
A. Proliferative inflammation
B. Emigrant inflammation
D. Exudative inflammation
E. Alterative inflammation

83. What is the inflammation that occurs in organisms with optimal reactivity?
A. Hyperergic inflammation
B. Hypergic inflammation
D. Emigrant inflammation
E. Normergic inflammation

84. What is inflammation, which is characterized by an increase in tissue productivity, ie the proliferation of cells?
A. Exudative inflammation
B. Proliferative inflammation
D. Emigrant inflammation
E. Alterative inflammation

85. What is the inflammation that occurs with a stronger manifestation of the vascular reaction and the predominance of exudation and emigration processes?
A. Exudative and emigrant inflammation
B. Proliferative and hypergic inflammation
D. Normergic and alternative inflammation
E. Alterative and hyperergic inflammation

86. What is inflammation, which is characterized by the accumulation of protein and fluid accumulation?
A. Serous inflammation
B. Catarrhal inflammation
D. Fibrinous inflammation
E. Hemorrhagic inflammation

87. State the inflammation characterized by the accumulation of exudate consisting of a mixture of serum and mucus.
A. Catarrhal inflammation
B. Serous inflammation
D. Fibrinous inflammation
E. Hemorrhagic inflammation

88. State the inflammation characterized by the accumulation of exudate, which contains more fibrin.
A. Fibrinous inflammation
B. Catarrhal inflammation
D. Serous inflammation
E. Diphtheria inflammation
89. Describe the inflammation characterized by the fact that the fibrin membrane at the level of the organ moves to the saliva and does not form a wound in its place.
A. Krupoz inflammation
B. Diphtheria inflammation
D. Fibrinous inflammation
E. Hemorrhagic inflammation
90. Describe the inflammation characterized by difficult removal of the fibrin membrane at the level of the organ and the formation of a wound in its place.
A. Diphtheria inflammation
B. Fibrinous inflammation
D. Krupoz inflammation
E. Hemorrhagic inflammation
91. What is the name of inflammation characterized by the presence of erythrocytes in the exudate?
A. Hemorrhagic inflammation
B. Fibrinous inflammation
D. Icrosis inflammation
E. Purulent inflammation
92. What type of inflammation do you know that is characterized by tissue erosion?
A. Inflammation of the esophagus
B. Hemorrhagic inflammation
D. Fibrinous inflammation
E. Purulent inflammation
93. What is the inflammation characterized by the accumulation of purulent exudate in the tissue, forming an interstitial space?
A. Carbuncle
B. Phlegmon
D. Furuncle
E. Abscess
94. What is inflammation called subcutaneous tissue, characterized by the spread of pus through a large part of the tissue through the muscles?
A. Abscess
B. Phlegmon
D. Pustule
E. Carbuncle
95. What is the inflammation characterized by the formation of a purulent blister under the epidermis of the skin?

A. Furuncle
B. Abscess
D. Pustule
E. Carbuncle

96. What is purulent inflammation of the sebaceous glands and wool sac called?

A. Pustule
B. Furuncle
D. Carbuncle
E. Abscess

97. What is the transfer of pus from the source of purulent inflammation and the transfer of pus into the blood?

A. Septicopiemia
B. Empiema
D. Sepsis
E. Abscess

98. What is a group of purulent inflammation of the sebaceous glands and wool sacs called?

A. Carbuncle
B. Pustule
D. Abscess
E. Furuncle

99. What is the accumulation of pus in the cavities of the body?

A. Empiema
B. Abscess
D. Sepsis
E. Septicopiemia

100. What is the transformation of healthy cells into tumor cells?

A. Malignancy
B. Oncology
D. Blastoma
E. Anaplasia

101. What is a malignant tumor formed from epithelial tissue?

A. Sarcoma
B. Cancer
D. Myoma
E. Epithelioma

102. What is a malignant tumor formed from connective tissue?

A. Mioma
B. Cancer
D. Sarcoma
E. Lipoma

103. What is a tissue or organ growth deficiency?

A. Hypoplasia
B. Aplasia

D. Atrophy
E. Hyperplasia

104. What characterizes the lack of nutrients in the tissue or organ?

A. With a hypobiotic process
B. With hyperbiotic process
D. With hypoplastic process
E. With aplastic process

105. What is the weakening of the function of a tissue or organ by reducing its size and dimension?

A. Aplasia
B. Atrophy
D. Hypoplasia
E. Hyperplasia

106. What is a sharp decrease in body weight and a decrease in all physiological functions?

A. Aplasia
B. Cachexia
D. Atrophy
E. Hyperplasia

107. What is called an increase in body temperature depending on the ambient temperature?

A. Fever
B. Hyperthermia
D. Hypothermia
E. Inflammation

108. What is the general reaction of an organism characterized by an increase in body temperature, regardless of changes in ambient temperature, relatively under the influence of harmful, often infectious agents?

A. Inflammation
B. Fever
D. Hypothermia
E. Hyperthermia

109. What is the decrease in body temperature depending on the ambient temperature?

A. Fever
B. Hypothermia
D. Inflammation
E. Hyperthermia

110. What determines the accumulation of glycogen in tissues?

A. It depends on the rate of glycogen re-synthesis and breakdown
B. Glycogen is re-synthesized and broken down in the body at the onset of liver disease
D. Glycogen is re-synthesized and broken down in the body in kidney disease
E. Glycogen is involved in the re-synthesis and breakdown of glycogen in muscle diseases in the body

111. What are the names of heat-generating substances?
A. Pyrogenic substances
B. Infectious substances
D. Hematogenous substances
E. Harmful substances
112. When does the main exchange process slow down?
A. When the activity of the nervous system decreases, when drugs enter the body, when the thyroid gland, adrenal gland hypofunction
B. When the activity of the nervous system is increased, when the activity of the thyroid, pituitary glands is increased
D. When the activity of the nervous system is strained and the activity of the thyroid gland is disturbed
E. When the activity of the nervous system deteriorates and the activity of the gonads increases
113. When does the main exchange process intensify?
A. The pituitary gland, when the activity of the thyroid gland is increased, in winter, in various diseases accompanied by fever
B. When the activity of the pancreas, pineal gland increases, in summer, in various diseases without fever
D. In the autumn, when the activity of the glands near the thyroid gland is increased, in various diseases accompanied by low fever
E. When the activity of the pancreas increases, in the spring, when the heat strikes
114. What is glycogenolysis?
A. It depends on the re-synthesis and breakdown of glycogen in the body
B. Glycogen is re-synthesized and broken down in the body in kidney disease
D. Glycogen is re-synthesized and broken down in the body in kidney disease
E. Improves the re-synthesis and breakdown of glycogen in the body in kidney disease}
115. When is the production of glycogen from glucose limited?
A. When the hormone adrenaline is deficient
B. When the insulin hormone is deficient
D. When thyroid hormone is deficient
E. Parat hormone deficiency
116. When is the absorption of fats disrupted?
A. When the external secretory activity of the pancreas is impaired and lipase is poorly secreted
B. When the endocrine function of the pancreas is impaired and lipocaine is poorly secreted
D. When the secretory activity of the pancreas is impaired and glucogon is poorly released
E. When factor F of fatty acids is deficient

117. What is the increase in neutral fats in the blood when fats are absorbed?
A. Transport hyperglycemia
B. Aleventar hyperglycemia

D. Emotional hyperglycemia
E. Retention hyperglycemia

118. When does hyperglycemia occur?

A. When large amounts of blood sugar are absorbed from the digestive system and cannot be assimilated as an energy source and are not converted into a reserve substance
B. Decomposition of large amounts of glycogen when significant amounts of carbohydrates are consumed in the tissues
D. When the formation of blood is increased, when it is killed in the tissues
E. When the production of insulin increases and the conversion of glucose to glycogen increases

119. When does hyperglycemia occur?

A. When insulin production is enhanced, when glucose is converted to glycogen
B. Decomposition of large amounts of glycogen when significant amounts of carbohydrates are consumed in the tissues
D. When the formation of blood is increased, when it is killed in the tissues
E. When a large amount of sugar is absorbed from the digestive system into the blood and cannot be assimilated as an energy source and is not converted into a reserve substance.

120. What hyperglycemia is observed due to the difficulty of the transfer of large particulate neutral fats from the blood to the body?

A. Aleventar hyperglycemia
B. Transport hyperglycemia
D. Emotional hyperglycemia
E. Retention hyperglycemia

121. What is the name of hyperglycemia, which occurs when fat is transported from the depots to the liver?

A. Retention hyperglycemia
B. Transport hyperglycemia
D. Emotional hyperglycemia
E. Aleventar hyperglycemia

122. What is the name of the protein and fat complex?

A. Glycoprotein
B. Lipoprotein
D. Lipodystrophy
E. Hyperproteinemia

123. What is lipuria?

A. Protein excretion in urine
B. Fat excretion in urine
D. Carbohydrate excretion in urine
E. Protein excretion in urine

124. What is hyper ketonemia?

A. An increase in acetone cells in the blood
B. Increased cholesterol in the blood
D. Increased lipoproteins in the blood

E. Increased glycoproteins in the blood

125. What changes occur in the body when ketone bodies increase in the blood?

A. Acedosis develops and the activity of enzymatic systems is disrupted

B. As alcoholism develops, the activity of enzymatic systems is disrupted

D. The concentration of H ions decreases and the activity of enzymatic systems is disrupted

E. The properties of buffer systems change and the activity of enzymatic systems is disrupted

126. When the body is fat?

A. When the caloric content of nutrients is higher than the energetic needs of the organism

B. When the caloric content of nutrients is less than the energetic needs of the organism

D. When the caloric content of nutrients is sufficient for the energetic needs of the organism

E. When the caloric content of nutrients is not sufficient to meet the energy needs of the organism

127. What is the increase in the amount of protein in the blood when protein metabolism is disturbed?

A. Hypoproteinemia

B. Hyperproteinemia

D. Hyperlipoproteinemia

E. Hyperglycoproteinemia

128. What is hyperhydrenemia?

A. When the water balance is positive or when water is retained in the body

B. When the water balance is positive or when he urinates a lot

D. When the water balance is negative or the tissue begins to dry out

E. When the water balance is negative or when it is excreted in the urine

129. What percentage of water is lost when an organism loses it?

A. 5% D. 20%

B. 10% E. 15%

130. What is a tumor?

A. Accumulation of water between tissues due to disruption of water exchange between blood and tissue

B. Accumulation of fluids in serum cavities

D. Accumulation of fluids in anatomical cavities

E. Accumulation of fluids between organs

131. What is hydrothorax?

A. Accumulation of desire in the abdominal cavity

B. Accumulation of desire in the pericardium of the heart

D. Istesco accumulation in the pleural cavity

E. The accumulation of desire in the ventricles of the brain

132. What is pericardium, hydropericardium?

A. The formation of cravings in the ventricles of the brain

B. The formation of cravings in the pericardium of the heart

D. Accumulation of desire by forming a cavity in the kidney
E. The accumulation of desire by forming a cavity in the liver

133. What is hydrocephalus?

A. The formation of cravings in the pericardium of the heart
B. The formation of cravings in the ventricles of the brain
D. Accumulation of desire by forming a cavity in the kidney
E. The accumulation of desire by forming a cavity in the liver

134. What is istesqo?

A. Accumulation of fluids in serum cavities
B. Accumulation of water between tissues due to disruption of water exchange between blood and tissue
D. Accumulation of fluids in anatomical cavities
E. Accumulation of fluids between organs

135. How much carbohydrates, fats and proteins are absorbed during complete starvation?

A. Carbohydrates 99-100%, Fats 95-98%, Proteins 40-45%
B. Carbohydrates 96-97, Fats 93-95%, Proteins 39-40%
D. Carbohydrates 97-98%, Fats 92-94%, Proteins 38-39%
E. Carbohydrates 90-91%, Fats 90-91%, Proteins 37-38%

136. How many periods of starvation are divided according to changes in metabolism?

A. In 5 periods
B. in period 2
D. 4 periods
E. 3 periods

137. What substance must enter the body in order to synthesize vitamin B12?

A. Yod
B. Mis
D. Iron
E. Cobalt

138. What is avitaminosis?

A. Lack of any vitamin in the diet
B. Lack of several vitamins in the diet
D. Lack of most vitamins in food
E. Lack of certain groups of vitamins in the diet

139. What is polyavitaminosis?

A. Lack of any vitamin in the diet
B. Lack of several vitamins in the diet
D. Lack of most vitamins in food
E. Lack of certain groups of vitamins in the diet

140. What disease is caused by vitamin D deficiency in older animals?

A. Osteomalacia
B. Infertility
D. Raxit

E. Drug intolerance

141. What disease causes vitamin D deficiency in young animals?

A. Drug intolerance
B. Osteomalacia
D. Infertility
E. Raxit

142. When does hypercalcemia develop?

A. Decreased filtration of phosphate and calcium salts in the renal tubules and increased reabsorption in the tubules
B. Filtration of phosphate and calcium salts in the renal tubules is normal, when reabsorption in the tubules is delayed.
D. When the filtration of phosphate and calcium salts in the glomeruli is impaired and the reabsorption in the tubules is severely impaired
E. Decreased filtration of phosphate and calcium salts in the renal tubules and increased reabsorption in the tubules

143. Indicate a vitamin that eliminates the formation of ulcers in the gastrointestinal tract?

A. F omili
B. A vitamin
D. B vitamins
E. U vitamins

144. How much water is formed when proteins, fats and carbohydrates are oxidized?

A. 41.5 liters, 107.1 liters, 55.5 liters
B. 40.5 liters, 105.1 liters, 53.5 liters
D. 39.5 liters, 104.1 liters, 54.5 liters
E. 40.0 liters, 106.1 liters, 52.5 liters

145. In which organ is the formation of glucose from glycogen?

A. Muskulda
B. Liver
D. Divorced
E. In the stomach

146. What is the process of formation of glucose from glycogen?

A. Hyperglycemia
B. Gluconeogenesis
D. Hypoglycemia
E. Ketonomy

147. Describe the disease that occurs when the production of insulin from the pancreas decreases.

A. Diabed without blood
B. Kandli diabed
D. Hypoglycemia
E. Hyperglycemia

148. How was the textbook of animal pathophysiology created?

Collecting AABFoxt reports
Collecting BVVPashutin reports
Collecting DIIRavich reports
Collecting EVVPodvitsotskiy reports

149. In caisson's disease, why are the gases in the blood dissolved?

A. Because there are a lot of fluids in the body
B. Because the gases melt under high pressure
D. Because the blood circulation in the body is weakened
E. Because of increased blood circulation in the body

150. In which tissue nitrogen is slowly dissolved and slowly released?

A. Adipose tissue is slowly saturated with nitrogen and slowly decomposes
B. Protein tissue is slowly saturated with nitrogen and slowly decomposes
D. Carbohydrate tissue slowly saturates with nitrogen and slowly separates
E. Body fluids are slowly saturated with nitrogen and slowly excreted

151. How fast do cancer cells break down glucose?

A. Cancer cells break down glycolysis products more than 4-5 times faster
B. Cancer cells break down glycolysis products more than 4-5 times faster
D. Cancer cells break down glycolysis products more than 4-5 times faster
E. Cancer cells break down glycolysis products more than 4-5 times faster

152. Who found out that in Samarkand, even when an animal is hungry, tumors do not stop growing?

AJYo'lchiev
BIPMishenko
DRPXaitov
ERXXaitov

153. What is the increase in total blood volume?

A. Hypovolemia
B. Hypervolemia
D. Anemia
E. Hyperemia

154. What is a decrease in total blood volume?

A. Hypervolemia
B. Hypovolemia
D. Anemia
E. Hyperemia

155. What is called normal blood volume?

A. Hypovolemia
B. Norvolemia
D. Hypervolemia
E. Hyperemia

156. What is an increase in total blood volume called?

A. Normovolemia
B. Pletora
D. Oligemia

E. Hyperemia

157. What is a decrease in total blood volume called?

A. Oligemia
B. Hypovolemia
D. Hypervolemia
E. Hyperemia

158. What is the decrease in the amount of erythrocytes and hemoglobin per unit volume of blood?

A. Hypovolemia
B. Anemia
D. Hypervolemia
E. Hyperemia

159. What is called hypervolemia, characterized by an increase in the number of erythrocytes?

A. Plethora brain
B. Normal hypervolemia
D. Oligocytomic hypervolemia
E. Polycythemic hypervole

160. What is the name of hypervolemia, which is characterized by an increase in the total amount of blood plasma?

A. Polycythemic hypervolemia
B. Oligocytomic hypervolemia
D. Normal hypervolemia
E. False hypervolemia

161. What is the name of hypervolemia, which is characterized by a moderate increase in the amount of plasma and erythrocytes?

A. Oligocytomic hypervolemia
B. Polycythemic hypervolemia
D. Normal hypervolemia
E. Oligemia

162. What is the name of hypovolemia, characterized by a decrease in the number of erythrocytes?

A. Oligemia
B. Oligocytomic hypovolemia
D. Polycythemic hypovolemia
E. Normal hypovolemia

163. What is the name of hypovolemia, which is characterized by a decrease in the amount of blood plasma without changing the number of erythrocytes per unit volume of blood?

A. Simple hypovolemia
B. Polycythemic hypovolemia
D. Oligocytomic hypovolemia
E. Oligemia

164. State hypovolemia, characterized by a moderate decrease in the amount of plasma and erythrocytes.
A. Simple hypovolemia
B. Polycythemic hypovolemia
D. Oligocytomic hypovolemia
E. Oligemia
165. What is the total volume of blood when the solid part of it decreases and becomes thin?
A. Hypervolemia
B. Hydremic pleura
D. Oligocytemic norvolemia
E. Polycythemic normolemia
166. What is a transfusion without change in total blood volume?
A. Hypovolemia
B. Polycythemic hypovolemia
D. Oligocytemic normovolemia
E. Polycythemic normovolemia
167. What is the increase in the number of erythrocytes in the blood?
A. Oligocytemia
B. Polycythemia
D. Anisocytosis
E. Poikilocytosis
168. What is a decrease in the number of erythrocytes in the blood?
A. Poikilocytosis
B. Polycythemia
D. Anisocytosis
E. Erythropenia
169. What is the formation of large or small red blood cells in the blood?
A. Poikilocytosis
B. Polycythemia
D. Oligocytemia
E. Anisocytosis
170. What is the formation of deformed erythrocytes in the blood?
A. Oligocytemia
B. Polycythemia
D. Anisocytosis
E. Poikilocytosis
171. What is called a large volume of erythrocytes?
A. Poikilocytosis
B. Macrocytosis
D. Anisocytosis
E. Microcytosis
172. What is the small size of erythrocytes?
A. Macrocytosis

B. Poikilocytosis
D. Microcytosis
E. Anisocytosis

173. What is anemia caused by excessive blood loss called?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

174. What is anemia caused by a lack of necessary nutrients?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

175. What is anemia caused by excessive breakdown of erythrocytes under the influence of toxins?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Alimentary anemia
E. Infectious anemia

176. What is anemia caused by filtered viruses in ungulates called?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Infectious anemia
E. Alimentary anemia

177. What is anemia caused by a violation of hematopoiesis?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Dysgemoetic anemia
E. Infectious anemia

178. What is anemia caused by iron and cobalt deficiency called?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Alimentary anemia
E. Infectious anemia

179. What is anemia caused by vitamin V12 deficiency called?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

180. What is anemia caused by a lack of complete protein?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

181. What is the increase in the number of leukocytes per unit volume of blood?
A. Leukopenia
B. Aleykemia
D. Leukocytosis
E. Leukemia
182. What is the decrease in the number of leukocytes per unit volume of blood?
A. Leukocytosis
B. Leukopenia
D.Aleukemia
E. Leukemia
183. What is the increase in the number of leukocytes in different physiological conditions?
A. Physiological leukocytosis
B. Degenerative leukocytosis
D. Regenerative leukocytosis
E. Pathological leukocytosis
184. What is the increase in the number of leukocytes in various diseases?
A. Degenerative leukocytosis
B. Pathological leukocytosis
D. Physiological leukocytosis
E. Regenerative leukocytosis
185. What is leukocytosis, characterized by an increase in the number of young neurophils in the blood?
A. Degenerative leukocytosis
B. Regenerative leukocytosis
D. Physiological leukocytosis
E. Pathological leukocytosis
186.What is leukocytosis, characterized by an increase in aging neutrophils in the blood?
A. Degenerative leukocytosis
B. Regenerative leukocytosis
D. Physiological leukocytosis
E. Pathological leukocytosis
187. What is the increase in the amount of basophils in the blood?
A. Neutrophilia
B. Eosinophilia
D. Basophilia
E. Monocytosis
188. What is the increase in the number of eosinophils in the blood?
A. Neutrophilia
B. Basophilia
D. Eosinophilia
E. Monocytosis
189. What is the increase in the number of neutrophils in the blood?

A. Eosinophilia
B. Basophilia
D. Neutrophilia
E. Monocytosis

190. What is the increase in the number of lymphocytes in the blood?

A. Monocytosis
B. Basophilia
D. Neutrophilia
E. Lymphocytosis

191. What is the increase in the number of monocytes in the blood?

A. Monocytosis
B. Basophilia
D. Neutrophilia
E. Eosinophilia

192. What is the name of leukocytosis observed in hemophilia?

A. Basophilia
B. Eosinophilia
D. Neutrophilia
E. Lymphocytosis

193. What is the name of leukocytosis observed in allergic and infectious diseases?

A. Eosinophilia
B. Monocytosis
D. Neutrophilia
E. Lymphocytosis

194. Name the leukocytosis observed in acute infectious diseases.

A. Neutrophilia
B. Eosinophilia
D. Monocytosis
E. Lymphocytosis

195. Name the leukocytosis observed in chronic infectious and endocrine diseases.

A. Eosinophilia
B. Lymphocytosis
D. Neutrophilia
E. Monocytosis

196. Name the leukocytosis observed in chronic infectious and protozoal diseases and with increased RES activity.

A. Eosinophilia
B. Monocytosis
D. Neutrophilia
E. Lymphocytosis

197. What is an increase in the number of platelets in the blood?

A. Thrombocytosis
B. Thrombopoiesis
D. Thrombocytopenia

E. Hemophilia

198. What is a decrease in the number of platelets in the blood?

A. Thrombocytopenia
B. Thrombocytosis
D. Thrombopoiesis
E. Hemophilia

199. What is the increase in the number of erythrocytes in the blood?

A. Cryoglobulins
B. Hemoglobinopathy
D. Polyglobulia
E. Pyroglobulins

200. What is the appearance of atypical forms of hemoglobin in the blood?

A. Hemoglobinopathy
B. Polyglobulia
D. Cryoglobulins
E. Pyroglobulins

2 Test Questions for Evil (200)

1. What is the formation of proteins that are not normally found in the blood?

A. Hemoglobinopathy
B. Paraproteinemia
D. Cryoglobulins
E. Pyroglobulins

2. What is the change in total blood volume without change?

A. Hypervolemia
B. Hydremia
D. Oligocytemic norvolemia
E. Polycythemic normolemia

3. What is a total blood volume transfusion without change?

A. Hypervolemia
B. Anhydremia
D. Oligocytemic normovolemia
E. Polycythemic normovolemia

4. What is the acceleration of the heart?

A. Tachycardia
B. Cyanosis
D. Bradycardia
E. Xansirash

5. What is called bruising of the skin and mucous membranes due to heart failure?

A. Tachycardia
B. Cyanosis
D. Bradycardia
E. Xansirash

6. What is slow heart rate?
A. Bradycardia
B. Tachycardia
D. Cyanosis
E. Xansirash
7. What is the acceleration of breathing due to circulatory failure?
A. Tachycardia
B. Xansirash
D. Bradycardia
E. Cyanosis
8. What is called cardiopathy, which is caused by damage to the heart valves with various diseases?
A. Cardiac hypertrophy
B. Heart defects
D. Heart strain
E. Inflammation of the heart muscle
9. What types of heart defects do you know?
A. Congenital, acquired, simple, complex
B. Acquired
D. Congenital
E. Simple and complex
10. What are the names of heart defects that occur during the ontogeny of an animal?
A. Acquired powders
B. Congenital malformations
D. Simple powders
E. Complex powders
11. What is the narrowing of blood vessels?
A. Cyanosis
B. Stenosis
D. Porok
E. Arteriosclerosis
12. What is the name of the defect, which is characterized by the presence of defects in some valves of the heart?
A. Simple powders
B. Acquired powders
D. Congenital malformations
E. Complex powders
13. What are the defects characterized by the presence of damage to several valves at the same time?
A. Complex powders
B. Acquired powders
D. Simple powders
E. Congenital malformations

14. During the postnatal life of an animal, what are the names of heart defects that occur as a result of various diseases?
A. Acquired powders
B. Congenital malformations
D. Simple powders
E. Complex powders
15. What is the appearance of a barrier, characterized by a deterioration of the conduction of impulses through the conduction system of the heart?
A. Blockade
B. Stenosis
D. Cyanosis
E. Porok
16. What is a cardiac arrhythmia?
A. Steno
B. Arrhythmia
D. Porok z
E. Blockade
17. What is called an extraordinary contraction of the heart or part of it due to the formation of an additional impulse?
A. Extrasystole
B. Diastola
D. Sistola
E. Hyposystolic condition
18. What is called a sudden increase in heart rate?
A. Paroxysmal tachycardia
B. Diastola
D. Hyposystolic condition
E. Tachycardia
19. What is an increase in blood pressure in the arteries?
A. Hypertension
B. Hypotension
D. Collapse
E. Shok
20. What is a drop in blood pressure in the arteries?
A. hypotonia
B. Hypertension
D. Collapse
E. Shok
21. What is a severe reaction that occurs in the body in response to overly strong influences that disrupt the control of the most vital processes for life?
A. Hypertension
B. Shok
D. Collapse
E. Hypotension

22. What is an acute deficiency of the vascular system, characterized by impaired metabolism and hypotension and hypovolemia?
A. Hypertension
B. Collapse
D. Hypotension
E. Shok
23. What is cardiac arrest?
A. Angina
B. Bradycardia
D. Tachycardia
E. Arrhythmia
24. How much fluid accumulates in the pericardial cavity in small and large animals?
A. Up to one liter in small animals and up to ten liters in large animals
B. Up to one and a half liters in small animals and up to eleven liters in large animals
D. Up to two liters in small animals and up to ten and a half liters in large animals
E. Up to three liters in small animals and up to fifteen liters in large animals
25. When is there a violation of the contraction of the heart at the same time interval?
A. In sinus arrhythmia
B. In atrioventricular arrhythmia
D. In case of hyposystole
E. In paroxysmal tachycardia
26. What is the pathological process that develops in the myocardium with the growth of connective tissue and hardening of the heart muscle?
A. Cardiosclerosis
B. Arteriosclerosis
D. Sclerosis
E. Atriosclerosis
27. What is called short-term fainting, which occurs suddenly as a result of acute disruption of blood supply to the brain?
A. Obmorok
B. Shok
D. Collapse
E. Sclerosis
28. What causes the conduction disturbance between the sinus node and the compartments of the heart?
A. As a result of sino-auricular blockade
B. As a result of atrioventricular block
D. As a result of the cross siege
E. As a result of the siege of Uzina
29. What causes conduction disturbances in the atrioventricular node or GIS joints?
A. As a result of atrioventricular block

B. As a result of sino-auricular blockade
D. As a result of the cross siege
E. As a result of the siege of Uzina

30. When is it observed that the scar tissue formed in the heart is pulled by blood pressure and bulges?

A. In a cardiac aneurysm
B. In a heart attack
D. In myocarditis
E. In cardiosclerosis

31. What is the name of hypertension that occurs during various diseases?

A. Symptomatic hypertension
B. Atherosclerotic hypertension
D. Neurotonic hypertension
E. Hypertension related to renal function

32. What types of periodic breathing do you know%

A. Cheyn-Stokscha, biotcha, kussmaulcha
B. Kussmaulcha, Sechenovcha
D. Biotcha, Cheyn-Stokscha
E. Biotcha-kussmaulcha, Pavlovcha

33. What is the accumulation of carbon dioxide in the tissues when there is not enough oxygen?

A. Asphyxia
B. Dispnoe
D. Taxipnoe
E. Apnea

34. What is the acceleration and shallowness of breathing?

A. Dispnoe
B. Apnea
D. Taxipnoe
E. Asphyxia

35. What is called slowing and deep breathing?

A. Apnea
B. Dispnoe
D. Taxipnoe
E. Bradipnoe

36. What is called complete cessation of breathing?

A. Dispnoe
B. Apnea
D. Taxipnoe
E. Bradipnoe

37. What is the suffocation of an animal due to the lack of O2 in the tissues and the accumulation of SO2 in them?

A. Taxipnoe
B. Apnea

D. Asphyxia
E. Bradipnoe

38. What is called an overgrowth of the lungs and insufficient compression?

A. Atelectasis
B. Asphyxia
D. Pneumothorax
E. Emphysema

39. What is called shrinkage, shrinkage and shrinkage of the lungs?

A. Asphyxia
B. Emphysema
D. Pneumothorax
E. Atelectasis

40. What is inflammation of the lungs and bronchi?

A. Bronchitis
B. Pneumonia
D. Bronchopneumonia
E. Pneumothorax

41. What is called pneumonia?

A. Bronchitis
B. Pneumothorax
D. Pneumonia
E. Bronchopneumonia

42. What is inflammation of the mucous membranes of the bronchi?

A. Pneumonia
B. Bronchitis
D. Pneumothorax
E. Bronchopneumonia

43. What is the violation of the tightness of the chest due to the entry of air or gas into the pleural cavity?

A. Emphysema
B. Pneumothorax
D. Asphyxia
E. Atelectasis

44. What is the accumulation of transudate in the alveoli and the swelling of the alveolar barriers?

A. Bronchitis
B. Pneumonia
D. Lung tumor
E. Bronchopneumonia

45. What is the conversion of venous blood flowing into the lungs into arterial blood?

A. Hypoxia
B. Hypoxemia
D. Arteriolysis

E. Hypocapnia

46. What is the oxygen deficiency observed in tissues?

A. Hypoxia
B. Hypoxemia
D. Hypercapnia
E. Hypocapnia

47. What changes are caused by a decrease in oxygen in the air by 4-5%?

A. Severe hypoxia
B. Hypoxemia
D. Hypercapnia
E. Hypocapnia

48. What is a decrease in oxygen in the blood?

A. Hypoxia
B. Hypoxemia
D. Hypercapnia
E. Hypocapnia

49. What is a decrease in the amount of SO2 in the blood?

A. Hypocapnia
B. Hypercapnia
D. Hypoxia
E. Hypoxemia

50. What is an increase in the amount of SO2 in the blood?

A. Hypoxemia
B. Hypercapnia
D. Hypoxia
E. Hypocapnia

51. What is the name of hypoxia caused by insufficiency of O2 in arterial blood due to low content of O2 in the inhaled air?

A. Anemic hypoxia
B. Dimmed or ischemic hypoxia
D. Hypoxic hypoxia
E. Histotoxic hypoxia

52. Describe hypoxia caused by low levels of hemoglobin in the blood.

A. Hypoxic hypoxia
B. Anemic hypoxia
D. Mixed hypoxia
E. Histotoxic hypoxia

53. What hypoxia occurs as a result of local circulatory disorders?

A. Stagnant hypoxia
B. Hypoxic hypoxia
D. Anemic hypoxia
E. Histotoxic hypoxia

54. Describe the hypoxia caused by a decrease in the ability of tissues to use the oxygen supplied by the blood.

A. Histotoxic hypoxia
B. Hypoxic hypoxia
D. Anemic hypoxia
E. Dim or ischemic hypoxia

55. Name the hypoxia that occurs as a result of traumatic shock, intoxication and metabolic disorders in tissues.

A. Hypoxic hypoxia
B. Histotoxic hypoxia
D. Anemic hypoxia
E. Mixed hypoxia

56. What are the reflex protective reactions that help clear the airways called?

A. Asphyxia
B. Cough and wheezing
D. Cough
E. Accentuation

57. When is it observed that the throat is constricted and air escapes through the nose?

A. When coughing
B. In asphyxia
D. Aksa urganda
E. When suffocated

58. What is the name of the pathological process manifested by wavy exhalation?

A. Asphyxia
B. Cough
D. Choking
E. Accentuation

59. What is the air velocity in a cough?

A. 60-130 m / sec
B. 50-120 m / sec
D. 70-140 m / sec
E. 40-100 m / sec

60. What are the stages of asphyxia?

A. in two stages
B. in three stages
D. in four stages
E. in five stages

61. What causes respiratory disorders in hypoxemia?

A. The ability of hemoglobin to bind oxygen
B. The ability of hemoglobin to carry oxygen
D. The ability of hemoglobin to replace oxygen
E. The ability of hemoglobin to deliver oxygen to tissue

62. What is called a lack of carbon dioxide in the blood?

A. Hypercapnia
B. Hypocapnia

D. Acopnia
E. Hypoxemia

63. What is called complete loss of appetite?
A. Anorexia
B. Polyphagia
D. Arection
E. Bulimia

64. What is a decrease in appetite?
A. Polyphagia
B. Anorexia
D. Bulimia
E. Arection

65. What is an increase in appetite?
A. Perorection
B. Polyphagia
D. Anorexia
E. Bulimia

66. What is anorexia nervosa?
A. Parorection
B. Polyphagia
D. Anorexia
E. Bulimia

67. What is Hadeb called overeating?
A. Polyphagia
B. Bulimia
D. Anorexia
E. Perorection

68. What is an animal called to drink a lot of water (thirsty)?
A. Adipsia
B. Polydipsia
D. Hypersalivation
E. Hyposalivation

69. What is an animal called low water intake?
A. Hypersalivation
B. Polydipsia
D. Adipsia
E. Hyposalivation

70. What is the increase in salivation?
A. Hypersalivation
B. Polydipsia
D. Hydrophobia
E. Hyposalivation

71. What is a decrease in salivation?
A. Hyposalivation

B. Polydipsia
D. Hypersalivation
E. Hydrophobia

72. What is the fear of water of a rabid animal called?

A. Hydrophobia
B. Polydipsia
D. Hypersalivation
E. Hyposalivation

73. What is called a decrease in the metricity of the stomach and pre-gastric compartments?

A. Hyperkinesis
B. Atony
D. Hypotension
E. Timpania

74. What is called the cessation of gastric and pre-gastric metastases?

A. Hypotension
B. Atony
D. Hyperkinesis
E. Timpania

75. What is the accumulation of gas in the large abdomen?

A. Timpania
B. Hypotension
D. Hyperkinesis
E. Atony

76. What is called excessive secretion of gastric juice?

A. Hyposecretion
B. Hypersecretion
D. Hyperacidity
E. Hypoaciditis

77. What is the complete cessation of gastric juice secretion?

A. Achilles
B. Hypersecretion
D. Hyperacidity
E. Hypoaciditis

78. What is called high acidity in gastric juice?

A. Hyposecretion
B. Hypersecretion
D. Hyperacidity
E. Hypoaciditis

79. What is the decrease in acids in gastric juice?

A. Hypoaciditis
B. Hypersecretion
D. Hyperacidity
E. Hyposecretion

80. What is called the accumulation of chymus due to a decrease in evacuation in the small intestine?
A. Kaprostasis
B. Chemostasis
D. Hypoxia
E. Axoliya
81. What is called the accumulation of feces due to a decrease in evacuation in the colon?
A. Kaprostasis
B. Chemostasis
D. Hypoxia
E. Axoliya
82. What is called low bile secretion?
A. Chemostasis
B. Hypoxia
D. Cholemia
E. Axoliya
83 What is the inseparability of bile fluid?
A. Hypoxia
B. Cholemia
D. Axoliya
E. Kaprostasis
84. What is the accumulation of gas in the intestines?
A. Dyspepsia
B. Flatulence
D. Constipation
E. Ileus
85. What is the disease characterized by disruption of all digestive processes in young animals?
A. Enterolite
B. Dyspepsia
D. Enterit
E. Gastritis
86. What are intestinal stones called?
A. Enterolite
B. Dyspepsia
D. Enterit
E. Gastritis
87. What is inflammation of the intestine?
A. Enterit
B. Enterolite
D. Gastroenteritis
E. Gastritis
88. What is inflammation of the stomach?

A. Gastritis
B. Enterolite
D. Enterit
E. Gastroenteritis

89. What is inflammation of the stomach and intestines?

A. Enterolite
B. Gastroenteritis
D. Enterit
E. Gastritis

90. Why is it impossible to grind food when the mucous membrane of the oral cavity is inflamed?

A. The upper and lower jaws are not closed
B. Because it is difficult to chew
D. Because the food damages the oral mucosa
E. For toothache and gum disease

91. Why is the digestion of food in the stomach disrupted during hyposalivation?

A. Because the alkaline substances in the stomach are low
B. Because a lot of alkaline substances get into the stomach
D. Because of the excess of alkaline substances in the stomach
E. Because alkaline substances are significantly absorbed in the stomach

92. Which volatile fatty acids are rapidly and which are slowly absorbed in the large intestine?

A. Fatty acid is absorbed quickly and propionic and acetic acids are absorbed slowly
B. Propionic and fatty acids are absorbed quickly, acetic acid is absorbed slowly
D. Propionic and acetic acids are absorbed quickly and fatty acids are absorbed slowly
E. Both fatty acids and propionic and acetic acids are absorbed either rapidly or slowly, depending on the conditions

93. What is the role of acetic acid in the body, in addition to the process of metabolism in tissues?

A. In the formation of milk sugar
B. In the formation of milk fat
D. In the formation of milk protein
E. In the formation of milk glycosides

94. How many liters of fluid are absorbed in the retina and folds of the abdomen?

A. 90 liters
B. up to 100 liters
D. up to 80 liters
E. up to 70 liters

95. What percentage of fluid is absorbed in the retina and retina?

A. 70-80 percent
B. up to 60-70 percent
D. 80-90 percent
E. 90-100 percent

96. When the contraction of the anterior pancreas weakens?

A. When drinking cold water, when the moisture content of food in the large abdomen is up to 70% or more than 95%
B. When not drinking cold water, when the moisture content of food in the large abdomen reaches 60% or more than 75%
D. When drinking hot water, when the moisture content of food in the large abdomen reaches 65% or more than 70%
E. Where to drink water, when the moisture content of food in the large abdomen reaches 50% or more than 65%

97. Where is the hormone villi, which affects the contraction of intestinal villi, formed?

A. 12 fingers formed in the intestinal mucosa
B. formed in the mucous membranes of the small intestine
D. formed in the mucous membranes of the lateral intestine
E. is formed in the mucous membranes of the appendix

98. What substances have a detrimental effect on areas with impaired intestinal permeability?

A. Adrenaline and sympathin
B. Histamine and choline
D. Noradrenaline and sympathin
E. Noradrenaline and glutamine

99. What determines the biochemical balance in the large intestine?

A. Ingested nutrients and microflora in them
B. Changes in the composition and quality of microorganisms
D. The formation of volatile fatty acids
E. The formation of propionic and acetic acids

100. What changes occur during ketosis?

A. Alkaline phosphatase, lipase, catalase, protease activity is lost, oxidation-reduction is weakened
B. Alkaline phosphatase, carbohydrate lipase activity decreases, oxidation-reduction disappears
D. Increases the activity of alkaline phosphatase, lipase, catalase, protease, increases oxidation-reduction
E. Increases the activity of alkaline phosphatase, lipase, catalase, protease, increases oxidation-reduction

101. What changes occur during pregnancy toxemia?

A. The antitoxic activity of the liver is weakened
B. Increases the antitoxic activity of the liver
D. The antitoxic activity of the liver stops
E. Increases antitoxic activity of the liver

102. What is the property of alkaline hematin?

A. A potent toxin that affects the nervous system
B. A potent toxin that does not affect the nervous system
D. A weak toxin that does not affect the nervous system
E. A simple toxin that does not affect the nervous system

103. What change is caused by the weakening of the contraction of the anterior chambers?

A. The accumulation of large amounts of lactic acid and the change in pH of the product in them

B. Many lactic acids are formed and do not change the pH of the product in them

D. The accumulation of a lot of lactic acid and the product in them does not change the pH

E. Many lactic acids change the pH of the product without accumulating}

104. Why does hydremia develop in liver pathology?

A. Although diuresis does not decrease, the body retains a lot of water

B. Diuresis increases and more water is retained in the body

D. Diuresis is reduced, more water is retained in the body

E. Diuresis increases and more water is retained in the body

105. What changes lead to disruption of the formation of gamma globulins in the liver?

A. Decreases blood coagulation by disrupting immunity, fibrinogen and prothrombin production in the body

B. Immunity, fibrinogen, and prothrombin production in the body remain unchanged and blood clotting decreases

D. Increases blood clotting without disrupting the body's immune system, fibrinogen and prothrombin production

E. Increases blood clotting by boosting immunity, fibrinogen and prothrombin production in the body

106. How does non-hepatic RES bilirubin differ from hepatic bilirubin?

A. It is excreted in the urine through the kidneys

B. It is not excreted in the urine through the kidneys

D. It is excreted extensively in the urine through the kidneys

E. It is slightly excreted in the urine through the kidneys

107. What is the difference between non-hepatic RES bilirubin and hepatic bilirubin?

A. Passes lightly into the tissue and stains it easily

B. Passes hard on the tissue and stains it lightly

D. Easily passes into tissue and stains it

E. Passes into the tissue and stains it lightly

108. How is hemolytic jaundice different from mechanical jaundice?

A. With non-toxic effects of bile pigments in hemolytic jaundice

B. With toxic effects of bile pigments in hemolytic jaundice

D. With no effect of bile pigments in hemolytic jaundice

E. With no effect at all on bile pigments in hemolytic jaundice

109. What is the attachment of the liver instead of parenchymal cells called tissue growth?

A. Hepatosis

B. Cirrhosis

D. Hepatitis

E. Hepatoma

110. What is a dystrophic change of liver tissue called?

A. Hepatosis
B. Hepatitis
D. Cirrhosis
E. Hepatoma

111. What is inflammation of the liver?

A. Hepatosis
B. Hepatitis
D. Cirrhosis
E. Hepatoma

112. What is the formation of a tumor in the liver?

A. Cirrhosis
B. Hepatosis
D. Hepatoma
E. Hepatitis

113. What is an increase in blood pressure due to the accumulation of blood in the portal vein of the liver?

A. Hepatosis
B. Portal hypertension
D. Cirrhosis
E. Hepatoma

114. Which organ activity is most affected by liver pathology?

A. Divorce
B. To the heart
D. Kidney
E. Intestine

115. What are the types of jaundice?

A. Mechanical, hemolytic, and parenchymal
B. Obturation and parenchymatosis
D. Mechanical and hemolytic
E. Infectious-toxic

116. What jaundice occurs when the bile ducts are blocked?

A. Mechanical jaundice
B. Hemolytic jaundice
D. Parenchymal jaundice
E. Normal jaundice

117. What jaundice occurs when the activity of liver parenchyma cells is impaired?

A. Hemolytic jaundice
B. Parenchymal jaundice
D. Complex jaundice
E. Normal jaundice

118. What is the name of jaundice caused by the formation of excess bilirubin in the blood due to excessive breakdown of erythrocytes in the peripheral blood?

A. Mechanical jaundice
B. Hemolytic jaundice
D. Parenchymal jaundice
E. Normal jaundice

119. What is the most important sign of a disorder of grass formation and separation?

A. Cholemia
B. Bilirubinemia
D. Jaundice
E. Urobilinemia

120. What is an increase in bile acids and its components in the blood?

A. Bilirubinemia
B. Jaundice
D. Cholemia
E. Urobilinemia

121. What is the increase in urobilin in the blood?

A. Urobilinuria
B. Urobilinemia
D. Bilirubinemia
E. Cholemia

122. What is the increase in bilirubin in the blood?

A. Urobilinemia
B. Jaundice
D. Bilirubinemia
E. Bilirubinuria

123. What causes disorders of urine formation and excretion?

A. Kidney-related factors
B. Renal and extrarenal factors
D. Disorders of water and salt metabolism
E. Extrarenal factors

124. What is inflammation of the kidneys?

A. Cystitis
B. Nephrosis
D. Nephrosclerosis
E. Jade

125. What is a dystrophic change of the urinary tract?

A. Sisti
B. Jade
D. t Nephrosis
E. Uremia

126. What is the appearance of sclerotic changes in the small arteries of the kidney?

A. Uremia
B. Nephrosis

D. Jade
E. Nephrosclerosis

127. What is blood urination called?

A. Nephrosis
B. Uremia
D. Nephrosclerosis
E. Jade

128. What is the increase in urine production and excretion?

A. Oliguria
B. Pollakuria
D. Polyuria
E. Anuria

129. What is the decrease in urine formation and excretion?

A. Pollakuria
B. Polyuria
D. Oliguria
E. Anuria

130. What is the complete cessation of urine formation and excretion?

A. Oliguria
B. Polyuria
D. Anuria
E. Pollakuria

131. What is the name of a small, frequent urination of an animal?

A. Anuria
B. Polyuria
D. Pollakuria
E. Oliguria

132. What are the consequences of impaired renal function?

A. Kidney tumors, hypertension, uremia
B. Renal hypertension and uremia
D. Kidney tumors and hypertension
E. Azotemic and eclamptic uremia

133. Depending on the amount of which hormone in the blood, urine can be formed or increased or decreased?

A. When the hormone adrenaline is low in the blood, it increases urine production and greatly reduces it
B. When the hormone thyroxine is low in the blood, it increases urine production and greatly reduces it
D. Parathyroid hormone increases urine production when it is low in the blood and greatly reduces it
E. When the hormone insulin is low in the blood, it increases urine production and greatly reduces it

134. How many millimeters of mercury in the renal arteries stops the formation of urine?

A. When it reaches a 40-50 mm mercury column
B. When it reaches 50-60 mm Hg
D. When it reaches 60-70 mm Hg
E. When it reaches 70-80 mm Hg

135. What causes the formation of stones in the urinary tract?

A. On an organic basis
B. Inorganic basis
D. On a biological basis
E. At the base of the urinary tract

136. What is hypostenuria?

A. Decreased ability of the kidneys to produce primary urine
B. Increased ability of the kidneys to produce primary urine
D. Loss of the ability of the kidneys to produce primary urine
E. Increased ability of the kidney to produce primary urine

137. What is isostenuria?

A. Absolute loss of the ability of the kidneys to produce primary urine
B. Gradual recovery of the kidney's ability to produce primary urine
D. Increased ability of the kidneys to produce primary urine
E. Increased ability of the kidney to produce primary urine

138. What are the disorders of the endocrine glands?

A. Endocrinopathy
B. Hypofunction
D. Dysfunction
E. Hyperfunction

139. What is the physiologically active substance produced by the endocrine glands?

A. Histamine
B. Metabolite
D. Hormone
E. Neurosecret

140. How is the activity of the endocrine glands studied?

A. Hyperfunction
B. Hypofunction
D. Extirpation
E. Endocrinopathy

141. What is called an increase in the activity of the endocrine glands?

A. Dysfunction
B. Hypofunction
D. Hyperfunction
E. Endocrinopathy

142. What is a decrease in the activity of endocrine glands?

A. Dysfunction
B. Hyperfunction
D. Hypofunction

E. Endocrinopathy

143. What is the disorder of endocrine glands?

A. Hypofunction
B. Dysfunction
D. Hyperfunction
E. Endocrinopathy

144. What disease is caused by dysfunction of the posterior pituitary gland?

A. Acromegaly
B. Diabetes mellitus
D. Diabetes mellitus
E. Gigantism

145. What disease is caused by hyperfunction of the anterior pituitary gland in older people?

A. Diabetes mellitus
B. Diabetes mellitus
D. Acromegaly
E. Gigantism

146. What disease occurs in humans due to adrenal hypofunction?

A. Diabetes mellitus
B. Acromegaly
D. Addison's disease
E. Diabetes mellitus

147. What disease is caused by hypofunction of the pancreas?

A. Diabetes mellitus
B. Acromegaly
D. Addison's disease
E. Diabetes mellitus

148. What disease is caused by hyperthyroidism?

A. Bazedov's disease
B. Diabetes mellitus
D. Myxidema
E. Diabetes mellitus

149. What disease is caused by hypofunction of the thyroid gland?

A. Acromegaly
B. Myxidema
D. Diabetes mellitus
E. Diabetes mellitus

150. What is the increase in the effect of thyroid hormones in the body?

A. Hyperthyroidism
B. Thyrotoxicosis
D. Hypothyroidism
E. Hypergonadism

151. What is a decrease in the effect of thyroid hormones in the body?

A. Hypothyroidism

B. Hyperthyroidism
D. Thyrotoxicosis
E. Hypergonadism

152. What is the poisoning of the body due to an increase in thyroid hormones in the blood?

A Hyperthyroidism
B. Thyrotoxicosis
D. Hypothyroidism
E. Hypergonadism

153. What is called an increase in the incretory activity of the gonads?

A. Hypothyroidism
B. Hyperthyroidism
D. Hypergonadism
E. Hypogonadism

154. What is called a decrease in the incretory activity of the gonads?

A. Hyperthyroidism
B. Hypogonadism
D. Hypothyroidism
E. Hypergonadism

155. What is it called if the sperm remains in the abdominal cavity or duct?

A. Infantilism
B. Hypogonadism
D. Cryptorchidism
E. Castration

156. What is the method of removal of the gonads called?

A. Hypogonadism
B. Castration
D. Cryptorchidism
E. Infantilism

157. What disease occurs in young children due to hypersecretion of somatropic hormones?

A. Diabetes mellitus
B. Acromegaly
D. Diabetes mellitus
E. Gigantism

158. What change occurs if the anterior pituitary gonadotropic hormone is not produced?

A. Reproductive organs and secondary sexual characteristics are not formed
B. Although reproductive organs develop, secondary sexual characteristics do not form
D. Reproductive organs and secondary sexual characteristics are well formed
E. Reproductive organs do not produce sexual characteristics}

159. What change occurs when the thyroid gland is removed in large animals due to metabolic disorders?

A. Tireopriv cachexia

B. Bazedov's disease
D. Mixedema
E. Diabetes mellitus

160. What happens in the hypersecretion of gonadotropic hormones?
A. The animal reaches sexual maturity early
B. The animal abandons the child and becomes barren
D. Tetanic contraction occurs
E. Hypoglycemic shock occurs

161. What happens in the hypersecretion of oxytocin?
A. The animal abandons the child and becomes barren
B. The animal reaches sexual maturity early
D. Urinary excretion decreases
E. Hypoglycemic shock occurs

162. What happens in hypersecretion of antidiuretic?
A. Urinary excretion decreases
B. The animal reaches sexual maturity early
D. Tetanic contraction occurs
E. Hypoglycemic shock occurs

163. What happens in insulin hypersecretion?
A. Tetanic contraction occurs
B. The animal reaches sexual maturity early
D. Hypoglycemic shock occurs
E. The animal abandons the child and becomes barren

164. What happens in parathyroid hormone hypersecretion?
A. Ionized calcium increases and nervous system excitability decreases
B. The animal abandons the child and becomes barren
D. The animal reaches sexual maturity early
E. Hypoglycemic shock occurs

165. Who created the doctrine of higher nervous activity?
AIMSechenov
BIPPavlov
DADSperanskiy
ENEVvedenskiy

166. Who studied the typological features of the nervous system?
AADSperanskiy
BIMSechenov
DIPPavlov
ENEVvedenskiy

167. Who proved that it is possible to form pathological conditioned reflexes under experimental conditions?
AIMSechenov
BIPPavlov
DADSperanskiy
ENEVvedenskiy

168. Who studied the doctrine of pathological dominance?
AADSperanskiy
BIPPavlov
DAAUxtomskiy
ENEVvedenskiy
169. Who founded the doctrine of trophic activity of the nervous system?
AIPPavlov and ADSperanskiy
BIMSechenov and IPPavlov
D. Sechenok and NEVvedenskiy
E. Speransky and Ukhtomsky
170. What is called a decrease in organ movement due to the nervous system?
A. Hyperkinesis
B. Paralysis
D. Hypokinesis
E. Parez
171. What is the complete cessation of organ movement due to the nervous system?
A. Parez
B. Hypokinesis
D. Hyperkinesis
E. Paralich
172. What is the decline in the activity of the movement?
A. Paralysis
B. Hyperkinesis
D. Parez
E. Hypokinesis
173. What is it called when a paralyzed muscle loses its specific tone and becomes loose?
A. Muscle atony
B. Muscle hypotension
D. Muscle contraction
E. Muscle weakness
174. What is an involuntary action that is not in accordance with the purpose?
A. Paralysis
B. Hypokinesis
D. Hyperkinesis
E. Parez
175. What is paralysis of the quadriceps muscles?
A. Tetrapligiya
B. Monoplegia
D. Paraplegia
E. Hemiplegia
176. What is paralysis of the muscles of both forelegs or limbs?
A. Paraplegia
B. Monoplegia

D. Tetrapligiya
E. Hemiplegia

177. What is paralysis of one leg muscle called?

A. Monoplegia
B. Tetrapligiya
D. Paraplegia
E. Hemiplegia

178. What is paralysis of one side of the body called?

A. Hemiplegia
B. Monoplegia
D. Paraplegia
E. Tetrapligiya

179. What is it called when a muscle stays in a contracted state for a long time?

A. Clonic shooting
B. Tetanic convulsions
D. Tonic shooting
E. Convulsion

180. What is the involuntary, occasional, rhythmic contraction and relaxation of certain muscles or the contraction of an injured part of the body?

A. Convulsion
B. Clonic shooting
D. Tetanic convulsions
E. Tonic shooting

181. What is a clonic shot that covers a large part of the body?

A. Atetaz
B. Convulsion
D. Clonic shooting
E. Tonic shooting

182.What is a clonic gravity that covers a large part of the body or completely?

A. Clonic shooting
B. Tetanic convulsions
D. Convulsion
E. Tonic shooting

183. What is the distribution of tonic tension to all skeletal muscles?

A. Convulsion
B. Tetanic shooting
D. Clonic shooting
E. Tonic shooting

184. What is a clonic contraction involving one or more muscles called?

A. Atetaz
B. Astasia
D. Asthenia
E. Tik

185. What is a violation of coordination and balance of the body?

A. Astasia
B. Ataxia
D. Asthenia
E. Atetase

186. What is the condition of an animal characterized by involuntary oscillations of the body and head as a result of a violation of the tone of the antagonistic muscles?

A. Asthenia
B. Ataxia
D. Astasia
E. Atetase

187. What is it called when an animal's muscle tone weakens and it quickly becomes tired?

A. Ataxia
B. Astasia
D. Asthenia
E. Atetase

188. What is the condition of the head and hoof, which is manifested by frequent uncontrolled involuntary contractions of the muscles of each group of synergistic functions?

A. Astasia
B. Chorea
D. Ataxia
E. Atetase

189. What is observed when successive contractions of antagonistic muscles or changes in their tone?

A. Ataxia
B. Astasia
D. Titrash-drajanie
E. Asthenia

190. What is a severe pathological condition characterized by inhibition of the nervous system, sometimes tremors, decreased blood pressure, hypothermia, respiratory and other physiological processes?

A. Diabetic condition
B. Diabetic syndrome
D. Diabetic coma
E. Diabetic change

191. What is an increase in organ sensitivity?

A. Analgesia
B. Hypersthesia
D. Hypesthesia
E. Paresthesia

192. What is a decrease in organ sensitivity?

A. Hypersthesia
B. Hypesthesia

D. Anesthesia
E. Paresthesia

193. What is called complete loss of organ sensitivity?

A. Hypesthesia
B. Hypersthesia
D. Anesthesia
E. Analgesia

194. What is an organ sensitivity disorder called?

A. Hypesthesia
B. G hypersthesia
D. Paresthesia
E. Analgesia

195. What is the loss of sensation of pain in the body?

A. Analgesia
B. Hypersthesia
D. Hypesthesia
E. Paresthesia

196. What is the increase in pain in the body?

A. Hypesthesia
B. Paraesthesia
D. Hypersthesia
E. Hyperalgesia

197. Who identified the problem of neutralizing the effects of putrefactive bacteria in the digestive system by stopping their activity?

A. Gaydengayn
BIPPavlov
DIIMechnikov
EVABasov

198. What is it called that some muscles involuntarily, occasionally, rhythmically contract and relax?

A. Clonic strain
B. Convulsion
D. Tonic effort
E. Tetanic convulsions

199. What is a clonic strain that covers most of the body?

A. Clonic strain
B. Convulsion
D. Tonic effort
E. Tetanic convulsions

200. What is a tonic effort that covers the whole body?

A. Tonic effort
B. Clonic strain
D. Convulsion
E. Tetanic convulsions

Test questions for YaB (500)

1. What is the name of the science that teaches the changes that occur in the body of the patient, the causes of the disease, the conditions, the mechanism of development, the consequences of the flow?
A. Epizootology
B. Pathological anatomy
D. Clinical diagnostics
E. Pathology physiology
2. What experiments are used in the study of pathological processes?
A. Chronic experiments
B. Auscultation, percussion, palpation
D. Acute experiments
E. Acute and chronic experiences
3. What is the name of the theory that explains the origin of the disease by connecting the divine forces?
A. Nervism
B. Animism
D. Humoral
E. Solidar
4. What is the name of the theory that explains the origin of the disease in relation to changes observed in cells?
A. Humoral
B. Yatroximik
D. Cellular
E.Solidar
5. Who is the founder of humoral theory?
ARVirxov
B. Democritus
DIPPavlov
E. Hippocrates
6. Who is the founder of the solitary theory?
ARVirxov
B. Hippocrates
DIPPavlov
E Democritus
7. Who is the founder of the science of pathological physiology?
AESLondon
BIIRavich
DAABogomoles
EVVPashutin
8. Who is the founder of the science of veterinary pathological physiology?
AVVPashutin
BIIRavich

DABogomoles
EESLondon

9. What is the general doctrine of disease called?

A. Pathogenesis
B. Etiology
D. Nosology
E. Pathology

10. What stages of the disease do you know?

A. Latent, prodromal, and clinical periods
B. Incubation, clinical, and termination periods
D. Incubation, prodromal, consequence, and end periods
E. Latent, prodromal, clinical, and concluding periods

11. Do you know the consequences of the disease?

A. The disease can be completely cured
B. Heals or dies from illness
D. The disease can be completely and partially cured
E. The disease ends in death

12. What is the recurrence of the disease in the body?

A. Tonatogenesis
B. Remission
D. Recidivism
E. Pathogenesis

13. What is the complete recovery of the body from disease?

A. Tonatogenesis
B. Remission
D. Sanogenesis
E. Pathogenesis

14. How many oC per hour does the temperature of the corpse decrease in the first and subsequent days?

A. The first day is 30, the following days are 0.30
B. The first day 20, the next days 0.50
D. The first day is 10, the next days are 0.20
E. The first day 40, the following days 0.40}

15. Agony - how long does a pre-death seizure last?

A. 2-3 left
B. 5-6 minutes
D.3-5 hours
E. A few hours

16. How long does clinical death last?

A. 5-6 minutes
B. 2-3 left
D. 3-5 hours
E. 10 left

17. Name the stages of death.

A. Agony, clinical and biological death
B. Clinical death
D. Biological death
E. Clinical and biological death

18. What is the mechanism of disease progression and development?

A. Pathogenesis
B. Sanogenesis
D. Tonatogenesis
E. Etiology

19. Who is the founder of the doctrine of stress?

A. Gans Selye
B. Foxt
D. Galen
EIPPavlov

20. What is the ability of an organism to respond physiologically to an influence?

A. Allergy
B. Resistance
D. Reactivity
E. Anaphylaxis

21. What is the level of resistance of the organism to pathogenic forces?

A. Allergy
B. Reactivity
D. Resistance
E. Anaphylaxis

22. What is called high reactivity of the organism?

A. Energy
B. Hyperglycemia
D. Dysergia
E. Hyperergy

23. What is called low reactivity of the organism?

A. Hyperglycemia
B. Hyperergy
D. Dysergia
E. Energy

24. What is the complete loss of reactivity of the organism?

A. Energy
B. Hyperglycemia
D. Dysergia
E. Hyperergy

25. What is the ability of an organism to respond to an impact involving physiological systems?

A. Reactivity
B. Resistance
D. Allergy

E. Anaphylaxis

26. What is the level of resistance of the organism to pathogenic forces?
A. Reactivity
B. Resistance
D. Allergy
E. Anaphylaxis
27. What is the complete loss of reactivity of the organism?
A. Hypoergia
B. Dysergia
D. Energy
E. Hyperergy
28. What is the deterioration of the reactivity of the organism?
A. Energy
B. Hypoergia
D. Hyperergy
E. Dysergia
29. What is the name of a separate system consisting of bone marrow, lymph nodes, reticular connective tissue cells in the spleen, endothelial cells, Kupfer cells in the liver and leukocytes?
A. Reticular-endothelial system
B. Humoral system
D. Neuro-humoral system
E. Endocrine system
30. What are the inactive cells that make up RES called?
A. Faglar
B. Macrophages
D. Microphages
E. Phagocytosis
31. What are the motile cells that make up RES called?
A. Macrophages
B. Microphages
D. Faglar
E. Phagocytosis
32. What is the process by which cells absorb and digest foreign substances entering the body?
A. Allergy
B. Phagocytosis
D. Immunity
E. Chemotaxis
33. What is called the absorption and absorption of liquids and solutes in the environment by the cell?
A. Phagocytosis
B. Allergy

D. Pinocytosis
E. Chemotaxis

34. What is the movement of a phagocyte towards a foreign substance?

A. Phagocytosis
B. Allergy
D. Chemotaxis
E. Immunity

35. What is called the attachment of a phagocyte to a foreign substance?

A. Attraction
B. Allergy
D. Phagocytosis
E. Chemotaxis

36. How do phagocytes digest foreign substances entering the body?

A. With false legs
B. With oils
D. With proteins
E. With enzymes

37. At what stage does the process of phagocytosis take place?

A. In three stages
B. In five stages
D. In two stages
E. In four stages

38. Who created the phagocytic theory of immunity and when?

ARKox (1881)
BIIMechnikov (1883)
DAABogomolets (1805)
EAAdo (1950)

39. What theories explain the formation of immunity?

A. Phagocytic, humoral theory
B. Humoral theory
D. Neuro-humoral theory
E. Physicochemical theory

40. What is the property of the organism to resist the action of various microorganisms that cause disease and their toxins?

A. Allergy
B. Immunity
D. Reactivity
E. Resistance

41. What is the type of hereditary immunity of an organism?

A. Congenital immunity
B. Acquired immunity
D. Active immunity
E. Passive immunity

42. What is the immunity that is formed during the life of an organism?

A. Acquired immunity
B. Congenital immunity
D. Active immunity
E. Passive immunity

43. What is the immunity that an organism develops after suffering from a certain infectious disease?

A. Naturally acquired immunity
B. Artificially acquired immunity
D. Active immunity
E. Passive immunity

44. What is the immunity created by vaccination by injecting vaccines and blood serum into the body?

A. Artificially acquired immunity
B. Naturally acquired immunity
D. Active immunity
E. Passive immunity

45. What is the immunity that develops in the body as a result of natural disease or vaccination with vaccines against the disease?

A. Congenital immunity
B. Acquired immunity
D. Active immunity
E. Passive immunity

46. What is the immunity created by the passage of immune cells through the mother's oral milk to a newborn animal or by the delivery of serum containing immune antibodies?

A. Active immunity
B. Acquired immunity
D. Passive immunity
E. Congenital immunity

47. What is the immunity formed against the toxins of microorganisms?

A. Acquired immunity
B. Antitoxic immunity
D. Congenital immunity
E. Passive immunity

48. What is the immunity that can ensure the complete cleansing of the body from infectious agents?

A. Passive immunity
B. Congenital immunity
D. Active immunity
E. Sterile immunity

49. What is immunity called, which does not ensure complete cleansing of the body from infectious agents?

A. Active immunity
B. Sterile immunity

D. Nosteril immunity
E. Congenital immunity

50. What are the substances that act on the immunocompetent organs of the body, forming antibodies and reacting with them?

A. Antibodies
B. Antigens
D. Allergens
E. Anophylactogen

51. What are the specific proteins that are produced in the immunocompetent organs of the body against antigens and react with them?

A. Antibodies
B. Antigens
D. Allergens
E. Anophylactogen

52 What are the substances that make the body hypersensitive to foreign substances?

A. Antigens
B. Allergens
D. Antibodies
E. Anophylactogen

53. What are the substances that can cause anaphylactic shock?

A. Antigens
B. Anophylactogen
D. Allergens
E. Antibodies

54. What is an increase in the body's sensitivity to certain nutrients and drugs?

A. Idiosyncrasy
B. Autoallergic
D. Allergic disease
E. Autoallergic disease

55. What is a disease that occurs suddenly due to an increase in the body's sensitivity to certain foreign substances and passes in the form of attacks?

A. Allergic diseases
B. Hereditary diseases
D. Congenital diseases
E. Infectious diseases

56. What are the protein molecules that accelerate antigen-antibody reactions in animal blood?

A. Interferon
B. Antibody
D. Antigen
E. Complement

57. What is the hypersensitivity of the organism to foreign substances?

A. Desensitization

B. Anaphylaxis
D. Sansibilization
E. Allergy

58. What is called hypersensitivity of the organism with special substances to cause anaphylaxis?

A. Desensitization
B. Anaphylaxis
D. Anaphylactic shock
E. Sensitization

59. What is the release of an animal from a state of sensitization?

A. Anaphylactic shock
B. Anaphylaxis
D. Sensitization
E. Desensitization

60. What is a circulatory disorder of an individual organ or part of it without changing the total amount of blood in the body?

A. Local circulatory disorders
B. Collaterial circulation
D. Decreased blood volume
E. General circulatory disorders

61. What is the increase in blood volume due to increased blood flow to organs and tissues?

A. Hyperemia
B. Ischemia
D. Arterial hyperemia
E. Venous hyperemia

62. What is a decrease in the amount of blood in a particular organ or part of it due to a decrease in blood flow in the veins?

A. Ischemia
B. Hyperemia
D. Collaterial circulation
E. Staz

63. What are the main symptoms of arterial hyperemia?

A. The organ turns reddish-purple, enlarges, becomes hot
B. The organ turns blue, enlarges, decreases in temperature
D. The organ turns red and blue, shrinks
E. The organ becomes pale, small, pale, and painful

64. What are the main symptoms of venous hyperemia?

A. The organ turns blue, enlarges, decreases in temperature.
B. The organ turns reddish-purple, enlarges, becomes hot
D. The organ turns red and blue, shrinks
E. The organ becomes pale, small, pale, and painful

65. What is the increase in the amount of blood flowing from an artery to an organ or part of it and the change that occurs when the amount of blood flowing does not change?
A. Venous hyperemia
B. Ischemia
D. Hyperemia
E. Arterial hyperemia
66. What are the main symptoms of ischemia?
A. The organ becomes pale, small, pale, and painful
B. The organ turns blue, enlarges, decreases in temperature
D.Organ turns red and blue, shrinks
E. The organ turns reddish-purple, enlarges, becomes hot
67. What is the cessation of blood flow in the capillaries or venous blood vessels of an organ?
A. Ischemia
B. Staz
D. Collaterial circulation
E. Hyperemia
68. What is the bleeding from a vein when the vessel wall is not damaged or their permeability is increased?
A. External bleeding
B. Hemorrhage
D. Diapedez
E. Internal bleeding
69. What is the name given to the fact that blood clots in the blood vessels of a living organism, forming blockages and resisting blood flow?
A. Blood clot
B. Thrombosis
D. Thrombogenesis
E. Thrombosis
70. What is called the clogging of blood and lymph vessels by certain particles that are not commonly found in the blood and lymph, but are brought in by the flow of these fluids?
A. Staz
B. Thrombosis
D. Embolism
E. Embol
71. What are the external signs of inflammation?
A. Redness, swelling, redness, pain, dysfunction
B. Swelling, fever, pain
D. Redness, swelling, pain
E. Swelling, pain, dysfunction
72. Who identified and interpreted the external signs of inflammation?
A. Sels and Galen

B. Sels and Parasels
D. Galen and Garvey
E. Hippocrates and Democritus

73. What are the main stages of inflammation?
A. Alteration, exudation and emigration, proliferation
B. Alteration, exudation, regeneration, and emigration
D. Dystrophy, exudation, regeneration, and emigration
E. Exudation, emigration, proliferation, and regeneration

74. How can inflammation occur?
A. Acute and chronic
B. Acute, moderately acute, chronic
D. Moderately acute
E. Chronic

75. What is the stage of inflammation characterized by tissue damage, dystrophy, disruption of its structure and function?
A. Proliferation
B. Exudation
D. Emigration
E. Alteration

76. When naming an inflamed tissue or organ, what Greek or Latin word is added to their name?
A. «oma» «iya»
B. «it» «iya»
D. «genesis» «iya»
E. «iya» «pir»

77. What is the inflammation that occurs in organisms with high reactivity?
A. Emigrant inflammation
B. Exudative inflammation
D. Hyperergic inflammation
E. Hypergic inflammation

78. What is the inflammation that occurs in organisms with low reactivity?
A. Hypergic inflammation
B. Exudative inflammation
D. Emigrant inflammation
E. Hyperergic inflammation

79. What is the release of a liquid portion of blood through the vascular wall of inflamed tissue?
A. Alteration
B. Exudation
D. Emigration
E. Proliferation

80. What is the release of leukocytes from the blood through the vascular wall of inflamed tissue?
A. Alteration

B. Emigration
D. Exudation
E. Proliferation

81. What is the proliferation of cellular elements in the site of inflammation?
A. Proliferation
B. Alteration
D. Emigration
E. Exudation

82. What is called inflammation, characterized by a predominance of dystrophy, necrosis and necrobiosis in tissues?
A. Proliferative inflammation
B. Emigrant inflammation
D. Exudative inflammation
E. Alterative inflammation

83. What is the inflammation that occurs in organisms with optimal reactivity?
A. Hyperergic inflammation
B. Hypergic inflammation
D. Emigrant inflammation
E. Normergic inflammation

84. What is inflammation, which is characterized by an increase in tissue productivity, ie the proliferation of cells?
A. Exudative inflammation
B. Proliferative inflammation
D. Emigrant inflammation
E. Alterative inflammation

85. What is the inflammation that occurs with a stronger manifestation of the vascular reaction and the predominance of exudation and emigration processes?
A. Exudative and emigrant inflammation
B. Proliferative and hypergic inflammation
D. Normergic and alternative inflammation
E. Alterative and hyperergic inflammation

86. What is inflammation, which is characterized by the accumulation of protein and fluid accumulation?
A. Serous inflammation
B. Catarrhal inflammation
D. Fibrinous inflammation
E. Hemorrhagic inflammation

87. State the inflammation characterized by the accumulation of exudate consisting of a mixture of serum and mucus.
A. Catarrhal inflammation
B. Serous inflammation
D. Fibrinous inflammation
E. Hemorrhagic inflammation

88. State the inflammation characterized by the accumulation of exudate, which contains more fibrin.
A. Fibrinous inflammation
B. Catarrhal inflammation
D. Serous inflammation
E. Diphtheria inflammation
89. Describe the inflammation characterized by the fact that the fibrin membrane at the level of the organ moves to the saliva and does not form a wound in its place.
A. Krupoz inflammation
B. Diphtheria inflammation
D. Fibrinous inflammation
E. Hemorrhagic inflammation
90. Describe the inflammation characterized by difficult removal of the fibrin membrane at the level of the organ and the formation of a wound in its place.
A. Diphtheria inflammation
B. Fibrinous inflammation
D. Krupoz inflammation
E. Hemorrhagic inflammation
91. What is the name of inflammation characterized by the presence of erythrocytes in the exudate?
A. Hemorrhagic inflammation
B. Fibrinous inflammation
D. Icrosis inflammation
E. Purulent inflammation
92. What type of inflammation do you know that is characterized by tissue erosion?
A. Inflammation of the esophagus
B. Hemorrhagic inflammation
D. Fibrinous inflammation
E. Purulent inflammation
93. What is the inflammation characterized by the accumulation of purulent exudate in the tissue, forming an interstitial space?
A. Carbuncle
B. Phlegmon
D. Furuncle
E. Abscess
94. What is inflammation called subcutaneous tissue, characterized by the spread of pus through a large part of the tissue through the muscles?
A. Abscess
B. Phlegmon
D. Pustule
E. Carbuncle
95. What is the inflammation characterized by the formation of a purulent blister under the epidermis of the skin?

A. Furuncle
B. Abscess
D. Pustule
E. Carbuncle

96. What is purulent inflammation of the sebaceous glands and wool sac called?

A. Pustule
B. Furuncle
D. Carbuncle
E. Abscess

97. What is the transfer of pus from the source of purulent inflammation and the transfer of pus into the blood?

A. Septicopiemia
B. Empiema
D. Sepsis
E. Abscess

98. What is a group of purulent inflammation of the sebaceous glands and wool sacs called?

A. Carbuncle
B. Pustule
D. Abscess
E. Furuncle

99. What is the accumulation of pus in the cavities of the body?

A. Empiema
B. Abscess
D. Sepsis
E. Septicopiemia

100. What is the transformation of healthy cells into tumor cells?

A. Malignancy
B. Oncology
D. Blastoma
E. Anaplasia

101. What is a malignant tumor formed from epithelial tissue?

A. Sarcoma
B. Cancer
D. Myoma
E. Epithelioma

102. What is a malignant tumor formed from connective tissue?

A. Mioma
B. Cancer
D. Sarcoma
E. Lipoma

103. What is a tissue or organ growth deficiency?

A. Hypoplasia
B. Aplasia

D. Atrophy
E. Hyperplasia

104. What characterizes the lack of nutrients in the tissue or organ?

A. With a hypobiotic process
B. With hyperbiotic process
D. With hypoplastic process
E. With aplastic process

105. What is the weakening of the function of a tissue or organ by reducing its size and dimension?

A. Aplasia
B. Atrophy
D. Hypoplasia
E. Hyperplasia

106. What is a sharp decrease in body weight and a decrease in all physiological functions?

A. Aplasia
B. Cachexia
D. Atrophy
E. Hyperplasia

107. What is called an increase in body temperature depending on the ambient temperature?

A. Fever
B. Hyperthermia
D. Hypothermia
E. Inflammation

108. What is the general reaction of an organism characterized by an increase in body temperature, regardless of changes in ambient temperature, relatively under the influence of harmful, often infectious agents?

A. Inflammation
B. Fever
D. Hypothermia
E. Hyperthermia

109. What is the decrease in body temperature depending on the ambient temperature?

A. Fever
B. Hypothermia
D. Inflammation
E. Hyperthermia

110. What determines the accumulation of glycogen in tissues?

A. It depends on the rate of glycogen re-synthesis and breakdown
B. Glycogen is re-synthesized and broken down in the body at the onset of liver disease
D. Glycogen is re-synthesized and broken down in the body in kidney disease
E. Glycogen is involved in the re-synthesis and breakdown of glycogen in muscle diseases in the body

111. What are the names of heat-generating substances?
A. Pyrogenic substances
B. Infectious substances
D. Hematogenous substances
E. Harmful substances
112. When does the main exchange process slow down?
A. When the activity of the nervous system decreases, when drugs enter the body, when the thyroid gland, adrenal gland hypofunction
B. When the activity of the nervous system is increased, when the activity of the thyroid, pituitary glands is increased
D. When the activity of the nervous system is strained and the activity of the thyroid gland is disturbed
E. When the activity of the nervous system deteriorates and the activity of the gonads increases
113. When does the main exchange process intensify?
A. The pituitary gland, when the activity of the thyroid gland is increased, in winter, in various diseases accompanied by fever
B. When the activity of the pancreas, pineal gland increases, in summer, in various diseases without fever
D. In the autumn, when the activity of the glands near the thyroid gland is increased, in various diseases accompanied by low fever
E. When the activity of the pancreas increases, in the spring, when the heat strikes
114. What is glycogenolysis?
A. It depends on the re-synthesis and breakdown of glycogen in the body
B. Glycogen is re-synthesized and broken down in the body in kidney disease
D. Glycogen is re-synthesized and broken down in the body in kidney disease
E. Improves the re-synthesis and breakdown of glycogen in the body in kidney disease}
115. When is the production of glycogen from glucose limited?
A. When the hormone adrenaline is deficient
B. When the insulin hormone is deficient
D. When thyroid hormone is deficient
E. Parat hormone deficiency
116. When is the absorption of fats disrupted?
A. When the external secretory activity of the pancreas is impaired and lipase is poorly secreted
B. When the endocrine secretory activity of the pancreas is impaired and lipocaine is poorly secreted
D. When the secretory activity of the pancreas is impaired and glucogon is poorly released
E. When factor F of fatty acids is deficient

117. What is the increase in neutral fats in the blood when fats are absorbed?
A. Transport hyperglycemia
B. Aleventar hyperglycemia

D. Emotional hyperglycemia
E. Retention hyperglycemia

118. When does hyperglycemia occur?

A. When large amounts of blood sugar are absorbed from the digestive system and cannot be assimilated as an energy source and are not converted into a reserve substance
B. Decomposition of large amounts of glycogen when significant amounts of carbohydrates are consumed in the tissues
D. When the formation of blood is increased, when it is killed in the tissues
E. When the production of insulin increases and the conversion of glucose to glycogen increases

119. When does hyperglycemia occur?

A. When insulin production is enhanced, when glucose is converted to glycogen
B. Decomposition of large amounts of glycogen when significant amounts of carbohydrates are consumed in the tissues
D. When the formation of blood is increased, when it is killed in the tissues
E. When a large amount of sugar is absorbed from the digestive system into the blood and cannot be assimilated as an energy source and is not converted into a reserve substance.

120. What hyperglycemia is observed due to the difficulty of the transfer of large particulate neutral fats from the blood to the body?

A. Aleventar hyperglycemia
B. Transport hyperglycemia
D. Emotional hyperglycemia
E. Retention hyperglycemia

121. What is the name of hyperglycemia, which occurs when fat is transported from the depots to the liver?

A. Retention hyperglycemia
B. Transport hyperglycemia
D. Emotional hyperglycemia
E. Aleventar hyperglycemia

122. What is the name of the protein and fat complex?

A. Glycoprotein
B. Lipoprotein
D. Lipodystrophy
E. Hyperproteinemia

123. What is lipuria?

A. Protein excretion in urine
B. Fat excretion in urine
D. Carbohydrate excretion in urine
E. Protein excretion in urine

124. What is hyper ketonemia?

A. An increase in acetone cells in the blood
B. Increased cholesterol in the blood
D. Increased lipoproteins in the blood

E. Increased glycoproteins in the blood

125. What changes occur in the body when ketone bodies increase in the blood?

A. Acedosis develops and the activity of enzymatic systems is disrupted

B. As alcoholism develops, the activity of enzymatic systems is disrupted

D. The concentration of H ions decreases and the activity of enzymatic systems is disrupted

E. The properties of buffer systems change and the activity of enzymatic systems is disrupted

126. When the body is fat?

A. When the caloric content of nutrients is higher than the energetic needs of the organism

B. When the caloric content of nutrients is less than the energetic needs of the organism

D. When the caloric content of nutrients is sufficient for the energetic needs of the organism

E. When the caloric content of nutrients is not sufficient to meet the energy needs of the organism

127. What is the increase in the amount of protein in the blood when protein metabolism is disturbed?

A. Hypoproteinemia

B. Hyperproteinemia

D. Hyperlipoproteinemia

E. Hyperglycoproteinemia

128. What is hyperhydrenemia?

A. When the water balance is positive or when water is retained in the body

B. When the water balance is positive or when he urinates a lot

D. When the water balance is negative or the tissue begins to dry out

E. When the water balance is negative or when it is excreted in the urine

129. What percentage of water is lost when an organism loses it?

A. 5% D. 20%

B. 10% E. 15%

130. What is a tumor?

A. Accumulation of water between tissues due to disruption of water exchange between blood and tissue

B. Accumulation of fluids in serum cavities

D. Accumulation of fluids in anatomical cavities

E. Accumulation of fluids between organs

131. What is hydrothorax?

A. Accumulation of desire in the abdominal cavity

B. Accumulation of desire in the pericardium of the heart

D. Istesco accumulation in the pleural cavity

E. The accumulation of desire in the ventricles of the brain

132. What is pericardium, hydropericardium?

A. The formation of cravings in the ventricles of the brain

B. The formation of cravings in the pericardium of the heart

D. Accumulation of desire by forming a cavity in the kidney
E. The accumulation of desire by forming a cavity in the liver

133. What is hydrocephalus?

A. The formation of cravings in the pericardium of the heart
B. The formation of cravings in the ventricles of the brain
D. Accumulation of desire by forming a cavity in the kidney
E. The accumulation of desire by forming a cavity in the liver

134. What is istesqo?

A. Accumulation of fluids in serum cavities
B. Accumulation of water between tissues due to disruption of water exchange between blood and tissue
D. Accumulation of fluids in anatomical cavities
E. Accumulation of fluids between organs

135. How much carbohydrates, fats and proteins are absorbed during complete starvation?

A. Carbohydrates 99-100%, Fats 95-98%, Proteins 40-45%
B. Carbohydrates 96-97, Fats 93-95%, Proteins 39-40%
D. Carbohydrates 97-98%, Fats 92-94%, Proteins 38-39%
E. Carbohydrates 90-91%, Fats 90-91%, Proteins 37-38%

136. How many periods of starvation are divided according to changes in metabolism?

A. In 5 periods
B. in period 2
D. 4 periods
E. 3 periods

137. What substance must enter the body in order to synthesize vitamin B12?

A. Yod
B. Mis
D. Iron
E. Cobalt

138. What is avitaminosis?

A. Lack of any vitamin in the diet
B. Lack of several vitamins in the diet
D. Lack of most vitamins in food
E. Lack of certain groups of vitamins in the diet

139. What is polyavitaminosis?

A. Lack of any vitamin in the diet
B. Lack of several vitamins in the diet
D. Lack of most vitamins in food
E. Lack of certain groups of vitamins in the diet

140. What disease is caused by vitamin D deficiency in older animals?

A. Osteomalacia
B. Infertility
D. Raxit

E. Drug intolerance

141. What disease causes vitamin D deficiency in young animals?

A. Drug intolerance
B. Osteomalacia
D. Infertility
E. Raxit

142. When does hypercalcemia develop?

A. Decreased filtration of phosphate and calcium salts in the renal tubules and increased reabsorption in the tubules
B. Filtration of phosphate and calcium salts in the renal tubules is normal, when reabsorption in the tubules is delayed.
D. When the filtration of phosphate and calcium salts in the glomeruli is impaired and the reabsorption in the tubules is severely impaired
E. Decreased filtration of phosphate and calcium salts in the renal tubules and increased reabsorption in the tubules

143. Indicate a vitamin that eliminates the formation of ulcers in the gastrointestinal tract?

A. F omili
B. A vitamin
D. B vitamins
E. U vitamins

144. How much water is formed when proteins, fats and carbohydrates are oxidized?

A. 41.5 liters, 107.1 liters, 55.5 liters
B. 40.5 liters, 105.1 liters, 53.5 liters
D. 39.5 liters, 104.1 liters, 54.5 liters
E. 40.0 liters, 106.1 liters, 52.5 liters

145. In which organ is the formation of glucose from glycogen?

A. Muskulda
B. Liver
D. Divorced
E. In the stomach

146. What is the process of formation of glucose from glycogen?

A. Hyperglycemia
B. Gluconeogenesis
D. Hypoglycemia
E. Ketonomy

147. Describe the disease that occurs when the production of insulin from the pancreas decreases.

A. Diabed without blood
B. Kandli diabed
D. Hypoglycemia
E. Hyperglycemia

148. How was the textbook of animal pathophysiology created?

Collecting AABFoxt reports
Collecting BVVPashutin reports
Collecting DIIRavich reports
Collecting EVVPodvitsotskiy reports

149. In caisson's disease, why are the gases in the blood dissolved?

A. Because there are a lot of fluids in the body
B. Because the gases melt under high pressure
D. Because the blood circulation in the body is weakened
E. Because of increased blood circulation in the body

150. In which tissue nitrogen is slowly dissolved and slowly released?

A. Adipose tissue is slowly saturated with nitrogen and slowly decomposes
B. Protein tissue is slowly saturated with nitrogen and slowly decomposes
D. Carbohydrate tissue slowly saturates with nitrogen and slowly separates
E. Body fluids are slowly saturated with nitrogen and slowly excreted

151. How fast do cancer cells break down glucose?

A. Cancer cells break down glycolysis products more than 4-5 times faster
B. Cancer cells break down glycolysis products more than 4-5 times faster
D. Cancer cells break down glycolysis products more than 4-5 times faster
E. Cancer cells break down glycolysis products more than 4-5 times faster

152. Who found out that in Samarkand, even when an animal is hungry, tumors do not stop growing?

AJYo'lchiev
BIPMishenko
DRPXaitov
ERXXaitov

153. What is the increase in total blood volume?

A. Hypovolemia
B. Hypervolemia
D. Anemia
E. Hyperemia

154. What is a decrease in total blood volume?

A. Hypervolemia
B. Hypovolemia
D. Anemia
E. Hyperemia

155. What is called normal blood volume?

A. Hypovolemia
B. Norvolemia
D. Hypervolemia
E. Hyperemia

156. What is an increase in total blood volume called?

A. Normovolemia
B. Pletora
D. Oligemia

E. Hyperemia

157. What is a decrease in total blood volume called?

A. Oligemia
B. Hypovolemia
D. Hypervolemia
E. Hyperemia

158. What is the decrease in the amount of erythrocytes and hemoglobin per unit volume of blood?

A. Hypovolemia
B. Anemia
D. Hypervolemia
E. Hyperemia

159. What is called hypervolemia, characterized by an increase in the number of erythrocytes?

A. Plethora brain
B. Normal hypervolemia
D. Oligocytomic hypervolemia
E. Polycythemic hypervole

160. What is the name of hypervolemia, which is characterized by an increase in the total amount of blood plasma?

A. Polycythemic hypervolemia
B. Oligocytomic hypervolemia
D. Normal hypervolemia
E. False hypervolemia

161. What is the name of hypervolemia, which is characterized by a moderate increase in the amount of plasma and erythrocytes?

A. Oligocytomic hypervolemia
B. Polycythemic hypervolemia
D. Normal hypervolemia
E. Oligemia

162. What is the name of hypovolemia, characterized by a decrease in the number of erythrocytes?

A. Oligemia
B. Oligocytomic hypovolemia
D. Polycythemic hypovolemia
E. Normal hypovolemia

163. What is the name of hypovolemia, which is characterized by a decrease in the amount of blood plasma without changing the number of erythrocytes per unit volume of blood?

A. Simple hypovolemia
B. Polycythemic hypovolemia
D. Oligocytomic hypovolemia
E. Oligemia

164. State hypovolemia, characterized by a moderate decrease in the amount of plasma and erythrocytes.
A. Simple hypovolemia
B. Polycythemic hypovolemia
D. Oligocytomic hypovolemia
E. Oligemia
165. What is the total volume of blood when the solid part of it decreases and becomes thin?
A. Hypervolemia
B. Hydremic pleura
D. Oligocytemic norvolemia
E. Polycythemic normolemia
166. What is a transfusion without change in total blood volume?
A. Hypovolemia
B. Polycythemic hypovolemia
D. Oligocytemic normovolemia
E. Polycythemic normovolemia
167. What is the increase in the number of erythrocytes in the blood?
A. Oligocytemia
B. Polycythemia
D. Anisocytosis
E. Poikilocytosis
168. What is a decrease in the number of erythrocytes in the blood?
A. Poikilocytosis
B. Polycythemia
D. Anisocytosis
E. Erythropenia
169. What is the formation of large or small red blood cells in the blood?
A. Poikilocytosis
B. Polycythemia
D. Oligocytemia
E. Anisocytosis
170. What is the formation of deformed erythrocytes in the blood?
A. Oligocytemia
B. Polycythemia
D. Anisocytosis
E. Poikilocytosis
171. What is called a large volume of erythrocytes?
A. Poikilocytosis
B. Macrocytosis
D. Anisocytosis
E. Microcytosis
172. What is the small size of erythrocytes?
A. Macrocytosis

B. Poikilocytosis
D. Microcytosis
E. Anisocytosis

173. What is anemia caused by excessive blood loss called?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

174. What is anemia caused by a lack of necessary nutrients?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

175. What is anemia caused by excessive breakdown of erythrocytes under the influence of toxins?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Alimentary anemia
E. Infectious anemia

176. What is anemia caused by filtered viruses in ungulates called?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Infectious anemia
E. Alimentary anemia

177. What is anemia caused by a violation of hematopoiesis?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Dysgemoetic anemia
E. Infectious anemia

178. What is anemia caused by iron and cobalt deficiency called?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Alimentary anemia
E. Infectious anemia

179. What is anemia caused by vitamin V12 deficiency called?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

180. What is anemia caused by a lack of complete protein?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia

181. What is the increase in the number of leukocytes per unit volume of blood?
A. Leukopenia
B. Aleykemia
D. Leukocytosis
E. Leukemia
182. What is the decrease in the number of leukocytes per unit volume of blood?
A. Leukocytosis
B. Leukopenia
D.Aleukemia
E. Leukemia
183. What is the increase in the number of leukocytes in different physiological conditions?
A. Physiological leukocytosis
B. Degenerative leukocytosis
D. Regenerative leukocytosis
E. Pathological leukocytosis
184. What is the increase in the number of leukocytes in various diseases?
A. Degenerative leukocytosis
B. Pathological leukocytosis
D. Physiological leukocytosis
E. Regenerative leukocytosis
185. What is leukocytosis, characterized by an increase in the number of young neurophils in the blood?
A. Degenerative leukocytosis
B. Regenerative leukocytosis
D. Physiological leukocytosis
E. Pathological leukocytosis
186.What is leukocytosis, characterized by an increase in aging neutrophils in the blood?
A. Degenerative leukocytosis
B. Regenerative leukocytosis
D. Physiological leukocytosis
E. Pathological leukocytosis
187. What is the increase in the amount of basophils in the blood?
A. Neutrophilia
B. Eosinophilia
D. Basophilia
E. Monocytosis
188. What is the increase in the number of eosinophils in the blood?
A. Neutrophilia
B. Basophilia
D. Eosinophilia
E. Monocytosis
189. What is the increase in the number of neutrophils in the blood?

A. Eosinophilia
B. Basophilia
D. Neutrophilia
E. Monocytosis

190. What is the increase in the number of lymphocytes in the blood?

A. Monocytosis
B. Basophilia
D. Neutrophilia
E. Lymphocytosis

191. What is the increase in the number of monocytes in the blood?

A. Monocytosis
B. Basophilia
D. Neutrophilia
E. Eosinophilia

192. What is the name of leukocytosis observed in hemophilia?

A. Basophilia
B. Eosinophilia
D. Neutrophilia
E. Lymphocytosis

193. What is the name of leukocytosis observed in allergic and infectious diseases?

A. Eosinophilia
B. Monocytosis
D. Neutrophilia
E. Lymphocytosis

194. Name the leukocytosis observed in acute infectious diseases.

A. Neutrophilia
B. Eosinophilia
D. Monocytosis
E. Lymphocytosis

195. Name the leukocytosis observed in chronic infectious and endocrine diseases.

A. Eosinophilia
B. Lymphocytosis
D. Neutrophilia
E. Monocytosis

196. Name the leukocytosis observed in chronic infectious and protozoal diseases and with increased RES activity.

A. Eosinophilia
B. Monocytosis
D. Neutrophilia
E. Lymphocytosis

197. What is an increase in the number of platelets in the blood?

A. Thrombocytosis
B. Thrombopoiesis
D. Thrombocytopenia

E. Hemophilia

198. What is a decrease in the number of platelets in the blood?

A. Thrombocytopenia
B. Thrombocytosis
D. Thrombopoiesis
E. Hemophilia

199. What is the increase in the number of erythrocytes in the blood?

A. Cryoglobulins
B. Hemoglobinopathy
D. Polyglobulia
E. Pyroglobulins

200. What is the appearance of atypical forms of hemoglobin in the blood?

A. Hemoglobinopathy
B. Polyglobulia
D. Cryoglobulins
E. Pyroglobulins

201. What is the formation of proteins that are not normally found in the blood?

A. Hemoglobinopathy
B. Paraproteinemia
D. Cryoglobulins
E. Pyroglobulins

202. What is the loss of total blood volume without change?

A. Hypervolemia
B. Hydremia
D. Oligocytemic norvolemia
E. Polycythemic normolemia

203. What is called a transfusion without change in total blood volume?

A. Hypervolemia
B. Anhydremia
D. Oligocytemic normovolemia
E. Polycythemic normovolemia

204. What is the acceleration of the heart?

A. Tachycardia
B. Cyanosis
D. Bradycardia
E. Xansirash

205. What is bruising of the skin and mucous membranes due to heart failure?

A. Tachycardia
B. Cyanosis
D. Bradycardia
E. Xansirash

206. What is the slowing down of the heart?

A. Bradycardia
B. Tachycardia

D. Cyanosis
E. Xansirash

207. What is the acceleration of respiration due to circulatory failure?

A. Tachycardia
B. Xansirash
D. Bradycardia
E. Cyanosis

208. What is called cardiopathy caused by damage to the heart valves with various diseases?

A. Cardiac hypertrophy
B. Heart defects
D. Heart strain
E. Inflammation of the heart muscle

209. What types of heart defects do you know?

A. Congenital, acquired, simple, complex
B. Acquired
D. Congenital
E. Simple and complex

210. What are the names of heart defects that occur during the ontogeny of an animal?

A. Acquired powders
B. Congenital malformations
D. Simple powders
E. Complex powders

211. What is the narrowing of blood vessels?

A. Cyanosis
B. Stenosis
D. Porok
E. Arteriosclerosis

212. What are the defects characterized by the presence of defects in some valves of the heart?

A. Simple powders
B. Acquired powders
D. Congenital malformations
E. Complex powders

213. What is a powdery mildew characterized by the presence of damage to several valves at the same time?

A. Complex powders
B. Acquired powders
D. Simple powders
E. Congenital malformations

214. During the postnatal life of an animal, what are the names of heart defects that occur as a result of various diseases?

A. Acquired powders

B. Congenital malformations
D. Simple powders
E. Complex powders

215. What is the appearance of a barrier, characterized by a deterioration of the conduction of impulses through the conduction system of the heart?

A. Blockade
B. Stenosis
D. Cyanosis
E. Porok

216. What is a cardiac arrhythmia?

A. Steno
B. Arrhythmia
D. Porok z
E. Blockade

217. What is called an extraordinary contraction of the heart or part of it due to the formation of an additional impulse?

A. Extrasystole
B. Diastola
D. Sistola
E. Hyposystolic condition

218. What is called a sudden increase in heart rate?

A. Paroxysmal tachycardia
B. Diastola
D. Hyposystolic condition
E. Tachycardia

219. What is an increase in blood pressure in the arteries?

A. Hypertension
B. Hypotension
D. Collapse
E. Shok

220. What is a decrease in blood pressure in the arteries?

A. hypotonia
B. Hypertension
D. Collapse
E. Shok

221. What is a severe reaction that occurs in the body in response to overly strong influences that disrupt the control of the processes necessary for life?

A. Hypertension
B. Shok
D. Collapse
E. Hypotension

222. What is an acute deficiency of the vascular system, characterized by impaired metabolism and hypotension and hypovolemia?

A. Hypertension

B. Collapse
D. Hypotension
E. Shok

223. What is called cardiac arrest?

A. Angina
B. Bradycardia
D. Tachycardia
E. Arrhythmia

224. How much fluid accumulates in the pericardial cavity in small and large animals?

A. Up to one liter in small animals and up to ten liters in large animals
B. Up to one and a half liters in small animals and up to eleven liters in large animals
D. Up to two liters in small animals and up to ten and a half liters in large animals
E. Up to three liters in small animals and up to fifteen liters in large animals

225. When is there a violation of the contraction of the heart at the same time interval?

A. In sinus arrhythmia
B. In atrioventricular arrhythmia
D. In case of hyposystole
E. In paroxysmal tachycardia

226. What is the pathological process that develops in the myocardium with the growth of connective tissue and hardening of the heart muscle?

A. Cardiosclerosis
B. Arteriosclerosis
D. Sclerosis
E. Atriosclerosis

227. What is a short-term fainting that occurs suddenly as a result of acute disruption of blood supply to the brain?

A. Obmorok
B. Shok
D. Collapse
E. Sclerosis

228. What causes a violation of the conduction formed between the sinus node and the compartments of the heart?

A. As a result of sino-auricular blockade
B. As a result of atrioventricular block
D. As a result of the cross siege
E. As a result of the siege of Uzina

229. What causes conduction disturbances in the atrioventricular node or GIS joints?

A. As a result of atrioventricular block
B. As a result of sino-auricular blockade
D. As a result of the cross siege
E. As a result of the siege of Uzina

230. When is it observed that the scar tissue formed in the heart is pulled by blood pressure and bulges?
A. In a cardiac aneurysm
B. In a heart attack
D. In myocarditis
E. In cardiosclerosis
231. What is the name of hypertension that occurs during various diseases?
A. Symptomatic hypertension
B. Atherosclerotic hypertension
D. Neurotonic hypertension
E. Hypertension related to renal function
232. What periodic breathing types do you know%
A. Cheyn-Stokscha, biotcha, kussmaulcha
B. Kussmaulcha, Sechenovcha
D. Biotcha, Cheyn-Stokscha
E. Biotcha-kussmaulcha, Pavlovcha
233. What is the accumulation of carbon dioxide in the tissues when there is not enough oxygen?
A. Asphyxia
B. Dispnoe
D. Taxipnoe
E. Apnea
234. What is the acceleration and shallowness of breathing?
A. Dispnoe
B. Apnea
D. Taxipnoe
E. Asphyxia
235. What is called slowing and deepening of breathing?
A. Apnea
B. Dispnoe
D. Taxipnoe
E. Bradipnoe
236. What is called complete cessation of breathing?
A. Dispnoe
B. Apnea
D. Taxipnoe
E. Bradipnoe
237. What is called suffocation of an animal due to the lack of O2 in the tissues and the accumulation of SO2 in them?
A. Taxipnoe
B. Apnea
D. Asphyxia
E. Bradipnoe
238. What is called an over-expansion of the lungs and insufficient compression?

A. Atelectasis
B. Asphyxia
D. Pneumothorax
E. Emphysema

239. What is called shrinkage, shrinkage and shrinkage of the lungs?

A. Asphyxia
B. Emphysema
D. Pneumothorax
E. Atelectasis

240. What is inflammation of the lungs and bronchi called?

A. Bronchitis
B. Pneumonia
D. Bronchopneumonia
E. Pneumothorax

241. What is inflammation of the lungs?

A. Bronchitis
B. Pneumothorax
D. Pneumonia
E. Bronchopneumonia

242. What is inflammation of the mucous membranes of the bronchi?

A. Pneumonia
B. Bronchitis
D. Pneumothorax
E. Bronchopneumonia

243. What is a violation of the tightness of the chest due to the entry of air or gas into the pleural cavity?

A. Emphysema
B. Pneumothorax
D. Asphyxia
E. Atelectasis

244. What is the accumulation of transudate in the alveoli and the swelling of the alveolar barriers?

A. Bronchitis
B. Pneumonia
D. Lung tumor
E. Bronchopneumonia

245. What is the conversion of venous blood flowing into the lungs into arterial blood?

A. Hypoxia
B. Hypoxemia
D. Arteriolysis
E. Hypocapnia

246. What is the observed oxygen deficiency in tissues?

A. Hypoxia

B. Hypoxemia
D. Hypercapnia
E. Hypocapnia

247. What changes are caused by a decrease in oxygen in the air by 4-5%?
A. Severe hypoxia
B. Hypoxemia
D. Hypercapnia
E. Hypocapnia

248. What is a decrease in oxygen in the blood?
A. Hypoxia
B. Hypoxemia
D. Hypercapnia
E. Hypocapnia

249. What is a decrease in the amount of SO2 in the blood?
A. Hypocapnia
B. Hypercapnia
D. Hypoxia
E. Hypoxemia

250. What is an increase in the amount of SO2 in the blood?
A. Hypoxemia
B. Hypercapnia
D. Hypoxia
E. Hypocapnia

251. What is the name of hypoxia caused by insufficiency of arterial blood with O2 due to low O2 content in the inhaled air?
A. Anemic hypoxia
B. Dimmed or ischemic hypoxia
D. Hypoxic hypoxia
E. Histotoxic hypoxia

252. Describe hypoxia caused by low levels of hemoglobin in the blood.
A. Hypoxic hypoxia
B. Anemic hypoxia
D. Mixed hypoxia
E. Histotoxic hypoxia

253. What hypoxia occurs as a result of local circulatory disorders?
A. Stagnant hypoxia
B. Hypoxic hypoxia
D. Anemic hypoxia
E. Histotoxic hypoxia

254. State the hypoxia caused by a decrease in the ability of tissues to use the oxygen supplied by the blood.
A. Histotoxic hypoxia
B. Hypoxic hypoxia
D. Anemic hypoxia

E. Dim or ischemic hypoxia

255. Name the hypoxia that occurs as a result of traumatic shock, intoxication and disorders of tissue metabolism in tissues.

A. Hypoxic hypoxia
B. Histotoxic hypoxia
D. Anemic hypoxia
E. Mixed hypoxia

256. What are the reflex protective reactions that help clear the airways called?

A. Asphyxia
B. Cough and wheezing
D. Cough
E. Accentuation

257. When is it observed that the throat is constricted and air escapes through the nose?

A. When coughing
B. In asphyxia
D. Aksa urganda
E. When suffocated

258. What is the name of the pathological process manifested by wavy exhalation?

A. Asphyxia
B. Cough
D. Choking
E. Accentuation

259. What is the speed of air in a cough?

A. 60-130 m / sec
B. 50-120 m / sec
D. 70-140 m / sec
E. 40-100 m / sec

260. What are the stages of asphyxia?

A. in two stages
B. in three stages
D. in four stages
E. in five stages

261. What causes respiratory disorders in hypoxemia?

A. The ability of hemoglobin to bind oxygen
B. The ability of hemoglobin to carry oxygen
D. The ability of hemoglobin to replace oxygen
E. The ability of hemoglobin to deliver oxygen to tissue

262. What is called a lack of carbon dioxide in the blood?

A. Hypercapnia
B. Hypocapnia
D. Acopnia
E. Hypoxemia

263. What is called complete loss of appetite?

A. Anorexia
B. Polyphagia
D. Arection
E. Bulimia

264. What is a decrease in appetite?
A. Polyphagia
B. Anorexia
D. Bulimia
E. Arection

265. What is called an increase in appetite?
A. Perorection
B. Polyphagia
D. Anorexia
E. Bulimia

266. What is called anorexia?
A. Parorection
B. Polyphagia
D. Anorexia
E. Bulimia

267. What is Hadeb called overeating?
A. Polyphagia
B. Bulimia
D. Anorexia
E. Perorection

268. What is an animal called to drink a lot of water (thirsty)?
A. Adipsia
B. Polydipsia
D. Hypersalivation
E. Hyposalivation

269. What is it called that an animal drinks less water?
A. Hypersalivation
B. Polydipsia
D. Adipsia
E. Hyposalivation

270. What is called increased salivation?
A. Hypersalivation
B. Polydipsia
D. Hydrophobia
E. Hyposalivation

271. What is a decrease in salivation?
A. Hyposalivation
B. Polydipsia
D. Hypersalivation
E. Hydrophobia

272. What is the fear of water of a rabid animal?
A. Hydrophobia
B. Polydipsia
D. Hypersalivation
E. Hyposalivation
273. What is called a decrease in the metricity of the stomach and pre-gastric compartments?
A. Hyperkinesis
B. Atony
D. Hypotension
E. Timpania
274. What is called the cessation of gastric and pre-gastric metastases?
A. Hypotension
B. Atony
D. Hyperkinesis
E. Timpania
275. What is the accumulation of gas in the large abdomen?
A. Timpania
B. Hypotension
D. Hyperkinesis
E. Atony
276. What is called excessive secretion of gastric juice?
A. Hyposecretion
B. Hypersecretion
D. Hyperacidity
E. Hypoaciditis
277. What is called the complete cessation of gastric juice secretion?
A. Achilles
B. Hypersecretion
D. Hyperacidity
E. Hypoaciditis
278. What is the high content of acids in gastric juice?
A. Hyposecretion
B. Hypersecretion
D. Hyperacidity
E. Hypoaciditis
279. What is the decrease in acids in gastric juice?
A. Hypoaciditis
B. Hypersecretion
D. Hyperacidity
E. Hyposecretion
280. What is called the accumulation of chymus due to a decrease in evacuation in the small intestine?
A. Kaprostasis

B. Chemostasis
D. Hypoxia
E. Axoliya

281. What is called the accumulation of feces due to a decrease in evacuation in the colon?

A. Kaprostasis
B. Chemostasis
D. Hypoxia
E. Axoliya

282. What is called low secretion of bile fluid?

A. Chemostasis
B. Hypoxia
D. Cholemia
E. Axoliya

283 What is the inseparability of bile fluid?

A. Hypoxia
B. Cholemia
D. Axoliya
E. Kaprostasis

284. What is the accumulation of gas in the intestines?

A. Dyspepsia
B. Flatulence
D. Constipation
E. Ileus

285. What is the disease characterized by disruption of all digestive processes in young animals?

A. Enterolite
B. Dyspepsia
D. Enterit
E. Gastritis

286. What are intestinal stones called?

A. Enterolite
B. Dyspepsia
D. Enterit
E. Gastritis

287. What is inflammation of the intestine?

A. Enterit
B. Enterolite
D. Gastroenteritis
E. Gastritis

288. What is inflammation of the stomach?

A. Gastritis
B. Enterolite
D. Enterit

E. Gastroenteritis

289. What is inflammation of the stomach and intestines?

A. Enterolite

B. Gastroenteritis

D. Enterit

E. Gastritis

290. Why is it impossible to grind food when the mucous membrane of the oral cavity is inflamed?

A. The upper and lower jaws are not closed

B. Because it is difficult to chew

D. Because the food damages the oral mucosa

E. For toothache and gum disease

291. Why is the digestion of food in the stomach during hyposalivation?

A. Because the alkaline substances in the stomach are low

B. Because a lot of alkaline substances get into the stomach

D. Because of the excess of alkaline substances in the stomach

E. Because alkaline substances are significantly absorbed in the stomach

292. Which volatile fatty acids are rapidly and which are slowly absorbed in the large intestine?

A. Fatty acid is absorbed quickly and propionic and acetic acids are absorbed slowly

B. Propionic and fatty acids are absorbed quickly, acetic acid is absorbed slowly

D. Propionic and acetic acids are absorbed quickly and fatty acids are absorbed slowly

E. Both fatty acids and propionic and acetic acids are absorbed either rapidly or slowly, depending on the conditions

293. What is the role of acetic acid in the body in addition to the process of metabolism in tissues?

A. In the formation of milk sugar

B. In the formation of milk fat

D. In the formation of milk protein

E. In the formation of milk glycosides

294. How many liters of fluid are absorbed in the retina and retina?

A. 90 liters

B. up to 100 liters

D. up to 80 liters

E. up to 70 liters

295. What percentage of fluid is absorbed in the retina and retina?

A. 70-80 percent

B. up to 60-70 percent

D. 80-90 percent

E. 90-100 percent

296. When the contraction of the anterior pancreas weakens?

A. When drinking cold water, when the moisture content of food in the large abdomen is up to 70% or more than 95%

B. When not drinking cold water, when the moisture content of food in the large abdomen reaches 60% or more than 75%
D. When drinking hot water, when the moisture content of food in the large abdomen reaches 65% or more than 70%
E. Where to drink water, when the moisture content of food in the large abdomen reaches 50% or more than 65%

297. Where is the hormone villi, which affects the contraction of intestinal villi, formed?

A. 12 fingers formed in the intestinal mucosa
B. formed in the mucous membranes of the small intestine
D. formed in the mucous membranes of the lateral intestine
E. is formed in the mucous membranes of the appendix

298. What substances have a detrimental effect on areas with impaired intestinal permeability?

A. Adrenaline and sympathin
B. Histamine and choline
D. Noradrenaline and sympathin
E. Noradrenaline and glutamine

299. What determines the biochemical balance in the large intestine?

A. Ingested nutrients and microflora in them
B. Changes in the composition and quality of microorganisms
D. The formation of volatile fatty acids
E. The formation of propionic and acetic acids

300. What changes occur during ketosis?

A. Alkaline phosphatase, lipase, catalase, protease activity is lost, oxidation-reduction is weakened
B. Alkaline phosphatase, carbohydrate lipase activity decreases, oxidation-reduction disappears
D. Increases the activity of alkaline phosphatase, lipase, catalase, protease, increases oxidation-reduction
E. Increases the activity of alkaline phosphatase, lipase, catalase, protease, increases oxidation-reduction

301. What changes occur during pregnancy toxemia?

A. The antitoxic activity of the liver is weakened
B. Increases the antitoxic activity of the liver
D. The antitoxic activity of the liver stops
E. Increases antitoxic activity of the liver

302. What is the property of alkaline hematin?

A. A potent toxin that affects the nervous system
B. A potent toxin that does not affect the nervous system
D. A weak toxin that does not affect the nervous system
E. A simple toxin that does not affect the nervous system

303. What change is caused by the weakening of the contraction of the anterior chambers?

A. The accumulation of large amounts of lactic acid and the change in pH of the product in them
B. Many lactic acids are formed and do not change the pH of the product in them
D. The accumulation of a lot of lactic acid and the product in them does not change the pH
E. Many lactic acids change the pH of the product without accumulating}

304. What causes hydremia in liver pathology?

A. Although diuresis does not decrease, the body retains a lot of water
B. Diuresis increases and more water is retained in the body
D. Diuresis is reduced, more water is retained in the body
E. Diuresis increases and more water is retained in the body

305. What changes lead to disruption of the formation of gamma globulins in the liver?

A. Decreases blood coagulation by disrupting immunity, fibrinogen and prothrombin production in the body
B. Immunity, fibrinogen, and prothrombin production in the body remain unchanged and blood clotting decreases
D. Increases blood clotting without disrupting the body's immune system, fibrinogen and prothrombin production
E. Increases blood clotting by boosting immunity, fibrinogen and prothrombin production in the body

306. How does non-hepatic RES bilirubin differ from hepatic bilirubin?

A. It is excreted in the urine through the kidneys
B. It is not excreted in the urine through the kidneys
D. It is excreted extensively in the urine through the kidneys
E. It is slightly excreted in the urine through the kidneys

307. How does non-hepatic RES bilirubin differ from hepatic bilirubin?

A. Passes lightly into the tissue and stains it easily
B. Passes hard on the tissue and stains it lightly
D. Easily passes into tissue and stains it
E. Passes into the tissue and stains it lightly

308. How is hemolytic jaundice different from mechanical jaundice?

A. With non-toxic effects of bile pigments in hemolytic jaundice
B. With toxic effects of bile pigments in hemolytic jaundice
D. With no effect of bile pigments in hemolytic jaundice
E. With no effect at all on bile pigments in hemolytic jaundice

309. What is the attachment of the liver instead of parenchymal cells called tissue growth?

A. Hepatosis
B. Cirrhosis
D. Hepatitis
E. Hepatoma

310. What is called dystrophic change of liver tissue?

A. Hepatosis

B. Hepatitis
D. Cirrhosis
E. Hepatoma

311. What is inflammation of the liver?
A. Hepatosis
B. Hepatitis
D. Cirrhosis
E. Hepatoma

312. What is the formation of a tumor in the liver?
A. Cirrhosis
B. Hepatosis
D. Hepatoma
E. Hepatitis

313. What is an increase in blood pressure due to accumulation of blood in the portal vein of the liver?
A. Hepatosis
B. Portal hypertension
D. Cirrhosis
E. Hepatoma

314. Which organ activity is most affected by liver pathology?
A. Divorce
B. To the heart
D. Kidney
E. Intestine

315. What are the types of jaundice?
A. Mechanical, hemolytic, and parenchymal
B. Obturation and parenchymatosis
D. Mechanical and hemolytic
E. Infectious-toxic

316. What jaundice occurs when the bile ducts are blocked?
A. Mechanical jaundice
B. Hemolytic jaundice
D. Parenchymal jaundice
E. Normal jaundice

317. What jaundice occurs when the activity of liver parenchyma cells is impaired?
A. Hemolytic jaundice
B. Parenchymal jaundice
D. Complex jaundice
E. Normal jaundice

318. What is the name of jaundice caused by the formation of excess bilirubin in the blood due to excessive breakdown of erythrocytes in the peripheral blood?
A. Mechanical jaundice
B. Hemolytic jaundice
D. Parenchymal jaundice

E. Normal jaundice

319. What is the most important sign of a disorder of grass formation and separation?

A. Cholemia
B. Bilirubinemia
D. Jaundice
E. Urobilinemia

320. What is the increase in bile acids and its components in the blood?

A. Bilirubinemia
B. Jaundice
D. Cholemia
E. Urobilinemia

321.What is the increase in the amount of urobilin in the blood?

A. Urobilinuria
B. Urobilinemia
D. Bilirubinemia
E. Cholemia

322.What is the increase in bilirubin in the blood?

A. Urobilinemia
B. Jaundice
D. Bilirubinemia
E. Bilirubinuria

323. What causes disorders of urine formation and excretion?

A. Kidney-related factors
B. Renal and extrarenal factors
D. Disorders of water and salt metabolism
E. Extrarenal factors

324. What is inflammation of the kidneys?

A. Cystitis
B. Nephrosis
D. Nephrosclerosis
E. Jade

325. What is a dystrophic change of the urinary tract?

A. Sisti
B. Jade
D. t Nephrosis
E. Uremia

326. What is called the appearance of sclerotic changes in the small arteries of the kidney?

A. Uremia
B. Nephrosis
D. Jade
E. Nephrosclerosis

327. What is blood urination called?

A. Nephrosis
B. Uremia
D. Nephrosclerosis
E. Jade

328. What is the increase in urine production and excretion?

A. Oliguria
B. Pollakuria
D. Polyuria
E. Anuria

329. What is the decrease in urine formation and excretion?

A. Pollakuria
B. Polyuria
D. Oliguria
E. Anuria

330. What is the complete cessation of urine formation and excretion?

A. Oliguria
B. Polyuria
D. Anuria
E. Pollakuria

331. What is the name of a small, frequent urination of an animal?

A. Anuria
B. Polyuria
D. Pollakuria
E. Oliguria

332. What are the consequences of impaired renal function?

A. Kidney tumors, hypertension, uremia
B. Renal hypertension and uremia
D. Kidney tumors and hypertension
E. Azotemic and eclamptic uremia

333. Depending on the amount of which hormone in the blood, urine can be either increased or decreased?

A. When the hormone adrenaline is low in the blood, it increases urine production and greatly reduces it
B. When the hormone thyroxine is low in the blood, it increases urine production and greatly reduces it
D. Parathyroid hormone increases urine production when it is low in the blood and greatly reduces it
E. When the hormone insulin is low in the blood, it increases urine production and greatly reduces it

334. How many millimeters of mercury in the renal arteries stops the formation of urine?

A. When it reaches a 40-50 mm mercury column
B. When it reaches 50-60 mm Hg
D. When it reaches 60-70 mm Hg

E. When it reaches 70-80 mm Hg

335. What causes the formation of stones in the urinary tract?

A. On an organic basis
B. Inorganic basis
D. On a biological basis
E. At the base of the urinary tract

336. What is hypostenuria?

A. Decreased ability of the kidneys to produce primary urine
B. Increased ability of the kidneys to produce primary urine
D. Loss of the ability of the kidneys to produce primary urine
E. Increased ability of the kidney to produce primary urine

337. What is isostenuria?

A. Absolute loss of the ability of the kidneys to produce primary urine
B. Gradual recovery of the kidney's ability to produce primary urine
D. Increased ability of the kidneys to produce primary urine
E. Increased ability of the kidney to produce primary urine

338. What are the disorders of the endocrine glands?

A. Endocrinopathy
B. Hypofunction
D. Dysfunction
E. Hyperfunction

339. What is the physiologically active substance produced by the endocrine glands?

A. Histamine
B. Metabolite
D. Hormone
E. Neurosecret

340. How is the activity of endocrine glands studied?

A. Hyperfunction
B. Hypofunction
D. Extirpation
E. Endocrinopathy

341. What is called an increase in endocrine glands?

A. Dysfunction
B. Hypofunction
D. Hyperfunction
E. Endocrinopathy

342. What is a decrease in the activity of endocrine glands?

A. Dysfunction
B. Hyperfunction
D. Hypofunction
E. Endocrinopathy

343. What is the disorder of endocrine glands?

A. Hypofunction

B. Dysfunction
D. Hyperfunction
E. Endocrinopathy

344. What disease is caused by dysfunction of the posterior pituitary gland?
A. Acromegaly
B. Diabetes mellitus
D. Diabetes mellitus
E. Gigantism

345. What disease is caused by hyperfunction of the anterior pituitary gland in older people?
A. Diabetes mellitus
B. Diabetes mellitus
D. Acromegaly
E. Gigantism

346. What disease occurs in humans due to adrenal hypofunction?
A. Diabetes mellitus
B. Acromegaly
D. Addison's disease
E. Diabetes mellitus

347. What disease is caused by hypofunction of the pancreas?
A. Diabetes mellitus
B. Acromegaly
D. Addison's disease
E. Diabetes mellitus

348. What disease is caused by hyperthyroidism?
A. Bazedov's disease
B. Diabetes mellitus
D. Myxidema
E. Diabetes mellitus

349. What disease is caused by hypofunction of the thyroid gland?
A. Acromegaly
B. Myxidema
D. Diabetes mellitus
E. Diabetes mellitus

350. What is the increase in the effect of thyroid hormones in the body?
A. Hyperthyroidism
B. Thyrotoxicosis
D. Hypothyroidism
E. Hypergonadism

351. What is a decrease in the effect of thyroid hormones in the body?
A. Hypothyroidism
B. Hyperthyroidism
D. Thyrotoxicosis
E. Hypergonadism

352. What is the poisoning of the body due to an increase in thyroid hormones in the blood?
A Hyperthyroidism
B. Thyrotoxicosis
D. Hypothyroidism
E. Hypergonadism
353. What is called an increase in the incretory activity of the gonads?
A. Hypothyroidism
B. Hyperthyroidism
D. Hypergonadism
E. Hypogonadism
354. What is a decrease in the incretory activity of the gonads?
A. Hyperthyroidism
B. Hypogonadism
D. Hypothyroidism
E. Hypergonadism
355. What is it called when the sperm remains in the abdominal cavity or duct?
A. Infantilism
B. Hypogonadism
D. Cryptorchidism
E. Castration
356. What is the method of removal of the gonads called?
A. Hypogonadism
B. Castration
D. Cryptorchidism
E. Infantilism
357. What disease occurs in young children due to hypersecretion of somatropic hormones?
A. Diabetes mellitus
B. Acromegaly
D. Diabetes mellitus
E. Gigantism
358. What changes occur when the anterior pituitary gonadotropic hormone is not produced?
A. Reproductive organs and secondary sexual characteristics are not formed
B. Although reproductive organs develop, secondary sexual characteristics do not form
D. Reproductive organs and secondary sexual characteristics are well formed
E. Reproductive organs do not produce sexual characteristics}
359. What change occurs when the thyroid gland is removed in large animals due to metabolic disorders?
A. Tireopriv cachexia
B. Bazedov's disease
D. Mixedema
E. Diabetes mellitus

360. What happens in the hypersecretion of gonadotropic hormones?
A. The animal reaches sexual maturity early
B. The animal abandons the child and becomes barren
D. Tetanic contraction occurs
E. Hypoglycemic shock occurs
361. What happens in the hypersecretion of oxytocin?
A. The animal abandons the child and becomes barren
B. The animal reaches sexual maturity early
D. Urinary excretion decreases
E. Hypoglycemic shock occurs
362. What happens in hypersecretion of antidiuretic?
A. Urinary excretion decreases
B. The animal reaches sexual maturity early
D. Tetanic contraction occurs
E. Hypoglycemic shock occurs
363. What happens in insulin hypersecretion?
A. Tetanic contraction occurs
B. The animal reaches sexual maturity early
D. Hypoglycemic shock occurs
E. The animal abandons the child and becomes barren
364. What happens in parathyroid hormone hypersecretion?
A. Ionized calcium increases and nervous system excitability decreases
B. The animal abandons the child and becomes barren
D. The animal reaches sexual maturity early
E. Hypoglycemic shock occurs
365. Who created the doctrine of higher nervous activity?
AIMSechenov
BIPPavlov
DADSperanskiy
ENEVvedenskiy
366. Who studied the typological features of the nervous system?
AADSperanskiy
BIMSechenov
DIPPavlov
ENEVvedenskiy
367. Who proved that it is possible to form pathological conditioned reflexes under experimental conditions?
AIMSechenov
BIPPavlov
DADSperanskiy
ENEVvedenskiy
368. Who studied the doctrine of pathological dominance?
AADSperanskiy
BIPPavlov

DAAUxtomskiy
ENEVvedenskiy
369. Who founded the doctrine of trophic activity of the nervous system?
AIPPavlov and ADSperanskiy
BIMSechenov and IPPavlov
D. Sechenok and NEVvedenskiy
E. Speransky and Ukhtomsky
370. What is the decrease in organ movement due to the nervous system?
A. Hyperkinesis
B. Paralysis
D. Hypokinesis
E. Parez
371. What is the complete cessation of organ movement due to the nervous system?
A. Parez
B. Hypokinesis
D. Hyperkinesis
E. Paralich
372. What is the decline in the activity of the movement?
A. Paralysis
B. Hyperkinesis
D. Parez
E. Hypokinesis
373. What is it called when a paralyzed muscle loses its specific tone and becomes loose?
A. Muscle atony
B. Muscle hypotension
D. Muscle contraction
E. Muscle weakness
374. What is an involuntary action that is not appropriate to the purpose?
A. Paralysis
B. Hypokinesis
D. Hyperkinesis
E. Parez
375. What is paralysis of the quadriceps muscles?
A. Tetrapligiya
B. Monoplegia
D. Paraplegia
E. Hemiplegia
376. What is paralysis of the muscles of both forelegs or limbs?
A. Paraplegia
B. Monoplegia
D. Tetrapligiya
E. Hemiplegia
377. What is paralysis of the muscles of one leg?

A. Monoplegia
B. Tetrapligiya
D. Paraplegia
E. Hemiplegia

378. What is paralysis of one side of the body called?

A. Hemiplegia
B. Monoplegia
D. Paraplegia
E. Tetrapligiya

379. What is it called when a muscle stays in a contracted state for a long time?

A. Clonic shooting
B. Tetanic convulsions
D. Tonic shooting
E. Convulsion

380. What is it called when some muscles involuntarily, occasionally, rhythmically contract and relax, or pull the injured part of the body?

A. Convulsion
B. Clonic shooting
D. Tetanic convulsions
E. Tonic shooting

381. What is a clonic shot that covers a large part of the body?

A. Atetaz
B. Convulsion
D. Clonic shooting
E. Tonic shooting

382.What is a clonic gravity that covers a large part of the body or completely?

A. Clonic shooting
B. Tetanic convulsions
D. Convulsion
E. Tonic shooting

383. What is the distribution of tonic tension to all skeletal muscles?

A. Convulsion
B. Tetanic shooting
D. Clonic shooting
E. Tonic shooting

384. What is a clonic contraction involving one or more muscles called?

A. Atetaz
B. Astasia
D. Asthenia
E. Tik

385. What is a violation of coordination and balance of the body?

A. Astasia
B. Ataxia
D. Asthenia

E. Atetase

386. What is the condition of an animal characterized by involuntary vibration of the body and head as a result of a violation of the tone of the antagonistic muscles?

A. Asthenia
B. Ataxia
D. Astasia
E. Atetase

387. What is it called when an animal's muscle tone weakens and it quickly becomes tired?

A. Ataxia
B. Astasia
D. Asthenia
E. Atetase

388. What is the condition of the head and hooves, which is manifested by the uncoordinated involuntary frequent contraction of the muscles of each group of synergistic functions?

A. Astasia
B. Chorea
D. Ataxia
E. Atetase

389. What is observed when successive contractions of antagonistic muscles or changes in their tone?

A. Ataxia
B. Astasia
D. Titrash-drajanie
E. Asthenia

390. What is a severe pathological condition characterized by inhibition of the nervous system, sometimes tremors, decreased blood pressure, hypothermia, respiratory and other physiological processes?

A. Diabetic condition
B. Diabetic syndrome
D. Diabetic coma
E. Diabetic change

391. What is an increase in organ sensitivity?

A. Analgesia
B. Hypersthesia
D. Hypesthesia
E. Paresthesia

392. What is a decrease in organ sensitivity?

A. Hypersthesia
B. Hypesthesia
D. Anesthesia
E. Paresthesia

393. What is called complete loss of organ sensitivity?

A. Hypesthesia
B. Hypersthesia
D. Anesthesia
E. Analgesia

394. What is an organ sensitivity disorder called?

A. Hypesthesia
B. G hypersthesia
D. Paresthesia
E. Analgesia

395. What is the loss of sensation of pain in the organ?

A. Analgesia
B. Hypersthesia
D. Hypesthesia
E. Paresthesia

396. What is the increase in pain in the body?

A. Hypesthesia
B. Paraesthesia
D. Hypersthesia
E. Hyperalgesia

397. Who identified the problem of neutralizing the effects of putrefactive bacteria in the digestive system by stopping their activity?

A. Gaydengayn
BIPPavlov
DIIMechnikov
EVABasov

398. What is it called that some muscles involuntarily, occasionally, rhythmically contract and relax?

A. Clonic strain
B. Convulsion
D. Tonic effort
E. Tetanic convulsions

399. What is a clonic strain that covers most of the body?

A. Clonic strain
B. Convulsion
D. Tonic effort
E. Tetanic convulsions

400. What is a tonic effort that covers the whole body?

A. Tonic effort
B. Clonic strain
D. Convulsion
E. Tetanic convulsions

401. What is the decrease in the amount of erythrocytes and hemoglobin per unit volume of blood?

A. Hyperemia

B. Hypovolemia
D. Hypervolemia
E. Anemia

402. What is the increase in total blood volume?

A. Pletora
B. Normovolemia
D. Olegemia
E. Hyperemia

403. What is a decrease in total blood volume?

A. Hyperemia
B. Hypervolemia
D. Normovolemia
E. Oligemia

404. What is the name of hypervolemia, characterized by an increase in the number of erythrocytes ?.

A. Pletora
B. Simple hypervolemia
D. Oligocytomic hypervolemia
E. Polycythemic hypervolemia

405. What is the name of hypervolemia, which is characterized by an increase in the amount of plasma?

A. Normal hypervolemia
B. Polycythemic hypervolemia
D. Oligocytomic hypervolemia
E. False hypervolemia

406. What is the name of hypervolemia, which is characterized by a moderate increase in the amount of plasma and erythrocytes?

A. Pletora
B. Polycythemic hypervolemia
D. Oligocytomic hypervolemia
E. Normal hypervolemia

407. What is the name of hypovolemia, characterized by a decrease in the number of erythrocytes?

A. Normal hypervolemia
B. Polycythemic hypervolemia
D. Oligocytomic hypervolemia
E. Pletora

408. What is the name of hypovolemia, which is characterized by a decrease in plasma and an increase in the number of erythrocytes?

A. Normal hypervolemia
B. Polycythemic hypervolemia
D. Oligocytomic hypovolemia
E. Pletora

409. What is the name of hypovolemia, which is characterized by a moderate decrease in the amount of plasma and erythrocytes?
A. Pletora
B. Polycythemic hypervolemia
D. Oligocytomic hypervolemia
E. Normal hypovolemia
410. What is the formation of large or small red blood cells in the blood?
A. Oligocytemia
B. Polycythemia
D. Anisocytosis
E. Poikilocytosis
411. What is the formation of deformed erythrocytes in the blood?
A. Polycythemia
B. Poikilocytosis
D.Anizocytosis
E. Oligocytemia
412. What is called a large volume of erythrocytes?
A. Anisocytosis
B. Poikilocytosis
D. Macrocytosis
E. Microcytosis
413. What is the small size of erythrocytes?
A. Macrocytosis
B. Poikilocytosis
D. Microcytosis
E. Anisocytosis
414. What is anemia caused by excessive blood loss?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia
415. What is anemia caused by a lack of necessary nutrients?
A. Alimentary anemia
B. Posthemorrhagic anemia
D. Hemolytic anemia
E. Infectious anemia
416. What is anemia caused by excessive breakdown of erythrocytes under the influence of toxins?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D.Alimentar anemia
E. Infectious anemia
417. What is the anemia caused by filtering viruses in ungulates?
A. Alimentary anemia

B. Infectious anemia
D. Posthemorrhagic anemia
E. Hemolytic anemia

418. What is anemia caused by a violation of hematopoiesis?
A. Hemolytic anemia
B. Dysgemoetic anemia
D. Posthemorrhagic anemia
E. Infectious anemia

419. What is anemia caused by iron and cobalt deficiency called?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Alimentary anemia
E. Infectious anemia

420. What is anemia caused by vitamin B12 deficiency called?
A. Posthemorrhagic anemia
B. Hemolytic anemia
D. Alimentary anemia
E. Infectious anemia

421. What is anemia caused by a lack of complete proteins?
A. Hemolytic anemia
B. Posthemorrhagic anemia
D. Alimentary anemia
E. Infectious anemia

422. What is the increase in the number of leukocytes in various diseases?
A. Physiological leukocytosis
B. Degenerative leukocytosis
D. Pathological leukocytosis
E. Regenerative leukocytosis

423. What is the name of leukocytosis, characterized by an increase in the number of young neurophils in the blood?
A. Regenerative leukocytosis
B. Degenerative leukocytosis
D.Physiological leukocytosis
E. Pathological leukocytosis

424. What is the name of leukocytosis, characterized by an increase in aging neutrophils in the blood?
A. Degenerative leukocytosis
B. Regenerative leukocytosis
D.Physiological leukocytosis
E. Pathological leukocytosis

425. What is an increase in the amount of basophils in the blood?
A. Eosinophilia
B. Basophilia
D. Neutrophilia

E.Monocytosis

426. What is an increase in the amount of eosinophils in the blood?

A. Basophilia
B. Eosinophilia
D. Neutrophilia
E.Monocytosis

427. What is the increase in the number of neutrophils in the blood?

A. Eosinophilia
B. Basophilia
D. Neutrophilia
E.Monocytosis

428. What is an increase in the number of lymphocytes in the blood?

A. Monocytosis
B. Basophilia
D. Neutrophilia
E. Lymphocytosis

429. What is the increase in the number of monocytes in the blood?

A.Monocytosis
B. Basophilia
D. Neutrophilia
E. Eosinophilia

430. What is the name of leukocytosis observed in hemophilia?

A. Basophilia
B. Eosinophilia
D. Neutrophilia
E. Lymphocytosis

431. What leukocytosis is observed in allergic and infectious diseases?

A. Eosinophilia
B. Monocytosis
D. Neutrophilia
E. Lymphocytosis

432. What leukocytosis is observed in acute infectious diseases ?.

A. Monocytosis
B. Eosinophilia
D. Neutrophilia
E. Lymphocytosis

433. What leukocytosis is observed in chronic infectious and endocrine diseases?

A. Monocytosis
B. Eosinophilia
D. Neutrophilia
E. Lymphocytosis

434. What leukocytosis is observed in the last period of the disease and when RES activity increases?

A. Eosinophilia

B. Monocytosis
D. Neutrophilia
E. Lymphocytosis

435. What is the increase in the number of erythrocytes in the blood?

A. Hemoglobinopathy
B. Polyglobulia
D. Cryoglobulins
E. Pyroglobulins

436. What is the formation of proteins that are not normally found in the blood?

A. Hemoglobinopathy
B. Paraproteinemia
D. Cryoglobulins
E. Pyroglobulins

437. What is the acceleration of the heartbeat?

A. Cyanosis
B. Tachycardia
D. Bradycardia
E. Xansirash

438. What is bruising of the skin and mucous membranes due to heart failure?

A.Sianosis
B. Tachycardia
D. Bradycardia
E.Xansirash

439. What is the slowing of the heartbeat?

A. Bradycardia
B. Tachycardia
D.Sianoz
E.Xansirash

440. What is the acceleration of respiration due to circulatory failure?

A.Xansirash
B. Tachycardia
D. Bradycardia
E.Sianoz

441. What is called cardiopathy caused by damage to the heart valves with various diseases?

A. Heart defects
B. Cardiac hypertrophy
D. Tension of the heart
E. Deficiency in valves

442. What are the heart defects that occur during the ontogeny of an animal?

A. Congenital malformations
B. Acquired powders
D. Simple powders
E. Complex powders

443. What is the narrowing of blood vessels called?
A. Porok
B.Sianoz
D. Stenosis
E.Arteriosclerosis
444. Name the heart defects that occur during the postnatal life of an animal, as a result of various diseases.
A. Simple powders
B. Congenital malformations
D. Acquired powders
E. Complex powders
445. What are the defects characterized by the presence of defects in some valves of the heart?
A. Acquired powders
B. Simple powders
D. Congenital malformations
E. Complex powders
446. What is a powdery mildew characterized by the presence of damage to several valves at the same time?
A. Complex powders
B. Acquired powders
D. Simple powders
E. Congenital malformations
447. What is the appearance of a barrier, characterized by deterioration of the conduction of impulses through the conduction system of the heart?
A. Cyanosis
B.Stenoz
D.Porok
E. Blockade
448. What is a cardiac arrhythmia?
A.Arhythmia
B.Stenoz
D.Porok
E.Blockade
449. What is called an extraordinary contraction of the heart or part of it due to the formation of an additional impulse?
A. Diastola
B. Hyposystolic condition
D. Extrasystole
E.Sistola
450. What is called a sudden increase in heart rate?
A. Diastola
B. Paroxysmal tachycardia
D. Hyposystole condition

E. Tachycardia

451. What is an increase in blood pressure in the veins?

A. Collapse
B. Hypotension
D. Hypertension
E.Shok

452. What is a decrease in blood pressure in the arteries?

A. Hypertension
B. Hypotension
D. Kollaps
E.Shok

453. What is a severe reaction that occurs in the body in response to overly strong influences that disrupt the control of the processes necessary for life?

A. Collapse
B. Hypertension
D. Shok
E. Hypotension

454. What is an acute failure of the vascular system, characterized by hypotension and hypovolemia?

A.Kollaps
B. Hypertension
D. Hypotension
E.Shok

455. What is shortness of breath or wheezing?

A.Dispnoe
B.Apnoe
D.Taxipnoe
E.Asfiksiya

456. What is the acceleration and shallowness of breathing?

A.Dispnoe
B.Apnoe
D.Taxipnoe
E.Asfiksiya

457. What is the slowing down and deepening of breathing?

A. Bradipnoe
B.Apnoe
D.Taxipnoe
E. Dispnoe

458. What is called complete cessation of breathing?

A.Apnoe
B.Dispnoe
D.Taxipnoe
E.Bradipnoe

459. What is the deficiency of O2 in tissues and the accumulation of SO2 in them?

A. Apnea
B. Asphyxia
D.Taxipnoe
E.Bradipnoe

460. What is called an over-expansion of the lungs and insufficient compression?

A. Pneumothorax
B.Asfection
D. Emphysema
E.Atelectasis

461. What is the reduction, shrinkage and shrinkage of the lungs?

A. Pneumothorax
B.Asfection
D. Emphysema
E. Atelectasis

462. What is called inflammation of the mucous membranes of the bronchi?

A. Bronchitis
B. Pneumonia
D.Pneumothorax
E. Bronchopneumonia

463. What is inflammation of the lungs?

A. Pneumonia
B.Pneumothorax
D.Bronchitis
E. Bronchopneumonia

464. What is the violation of the tightness of the chest due to the entry of air or gas into the pleural cavity?

a.Pneumothorax
b.Emphysema
d.Asfection
e.Atelectasis

465. What is inflammation of the lungs and bronchi?

A. Bronchopneumonia
B. Pneumonia
D.Bronchitis
E.Pneumothorax

466. What is the accumulation of transudate in the alveoli and the swelling of the alveolar barriers?

A. Bronchitis
B. Pneumonia
D. Lung tumor
E. Bronchopneumonia

467. What is the conversion of venous blood flowing into the lungs into arterial blood?

A. Hypoxia

B. Hypoxemia
D. Arteriolysis
E. Hypocapnia
468. What is the observed oxygen deficiency in tissues?
a.Hypoxia
b.Hypoxemia
d.Hypercapnia
e.Gipokapniya
469. What is a decrease in oxygen in the blood?
A. Hypoxemia
B. Hypoxia
D. Hypercapnia
E. Hypocapnia
470. What is the name of hypoxia caused by a decrease in the ability of tissues to use the oxygen supplied by the blood?
A. Histotoxic hypoxia
B. Hypoxic hypoxia
D.Anemic hypoxia
E. Dimmed or ischemic hypoxia
471. What is the name of hypoxia caused by insufficiency of arterial blood with O2 due to low content of O2 in the inhaled air?
A. Anemic hypoxia
B. Dimmed or ischemic hypoxia
D. Hypoxic hypoxia
E. Histotoxic hypoxia
472. What is the name of hypoxia caused by low hemoglobin in the blood?
A. Mixed hypoxia
B. Hypoxic hypoxia
D. Anemic hypoxia
E. Histotoxic hypoxia
473. What is the name of hypoxia, which occurs as a result of local circulatory disorders?
A. Anemic hypoxia
B. Hypoxic hypoxia
D. Stagnant hypoxia
E. Histotoxic hypoxia
474. What is an increase in the amount of SO2 in the blood?
A. Hypoxemia
B. Hypercapnia
D. Hypoxia
E. Hypocapnia
475. What is the name of hypoxia that occurs as a result of traumatic shock, intoxication and metabolic disorders in tissues?
A. Hypoxic hypoxia

B. Histotoxic hypoxia
D. Anemic hypoxia
E. Mixed hypoxia
476. What are the reflex protective reactions that help to clear the airways?
A. Asphyxia
B. Cough and wheezing
D. Cough
E. Accentuation
477. What is the complete loss of appetite?
A. Arection
B. Polyphagia
D. Anorexia
E. Bulimia
479. What is a decrease in appetite?
A. Bulimia
B. Polyphagia
D. Anorexia
E. Arection
480. What is called an increase in appetite?
A. Bulimia
B. Polyphagia
D. Anorexia
E. Perorection
481. What is called anorexia?
A. Bulimia
B. Perorection
D. Polyphagia
E. Anorexia
482. What is Hadeb called overeating?
A. Anorexia
B. Polyphagia
D. Bulimia
E. Perorection
483. What is it called when an animal drinks a lot of water (thirst)?
A. Polydipsia
B. Adipsia
D. Hypersalivation
E. Hyposalivation
484. What is an animal's low water intake called?
A. Hyposalivation
B. Polydipsia
D. Hypersalivation
E. Adipsia
485. What is the increase in salivation?

A. Hydrophobia
B. Polydipsia
D. Hypersalivation
E. Hyposalivation
486. What is a decrease in salivation?
A. Hypersalivation
B. Polydipsia
D. Hyposalivation
E. Hydrophobia
487. What is the fear of water of a rabid animal?
A. Hydrophobia
B. Polydipsia
D. Hypersalivation
E. Hyposalivation
488. What is called a decrease in the metricity of the stomach and pre-gastric compartments?
A. Hypotension
B. Atony
D. Hyperkinesis
E. Timpania
489. What is called the cessation of gastric and pre-gastric metastases?
A. Atony
B. Hypotension
D. Hyperkinesis
E. Timpania
490. What is the accumulation of gas in the large abdomen?
A. Timpania
B. Hypotension
D. Hyperkinesis
E. Atony
491. What is called excessive secretion of gastric juice?
A. Hypersecretion
B. Hyposecretion
D. Hyperacidity
E. Hypoaciditis
492. What is called the complete cessation of gastric secretion?
A. Achilles
B. Hypersecretion
D. Hyperacidity
E. Hypoaciditis
493. What is called high acidity in gastric juice?
A. Hypersecretion
B. Hyperacidity
D. Hyposecretion

E. Hypoaciditis

494. What is the decrease in acids in gastric juice?

A. Hypersecretion
B. Hypoaciditis
D. Hyperacidity
E. Hyposecretion

495. What is said to cause the humus to begin to accumulate due to a decrease in evacuation in the small intestinein?

A. Hypoxia
B. Kaprostasis
D. Chemostasis
E. Axoliya

496. What is called the accumulation of feces due to a decrease in evacuation in the colon?

A. Hypoxia
B. Chemostasis
D. Kaprostasis
E. Axoliya

497. What is called low bile secretion?

A. Chemostasis
B. Hypoxia
D. Cholemia
E. Axoliya

498. What is the inseparability of bile fluid?

E. Axoliya
D. Cholemia
B. Hypoxia
A. Kaprostasis

499. What is the accumulation of gas in the intestines?

A. Constipation
B. Dyspepsia
D. Ileus
E. Flatulence

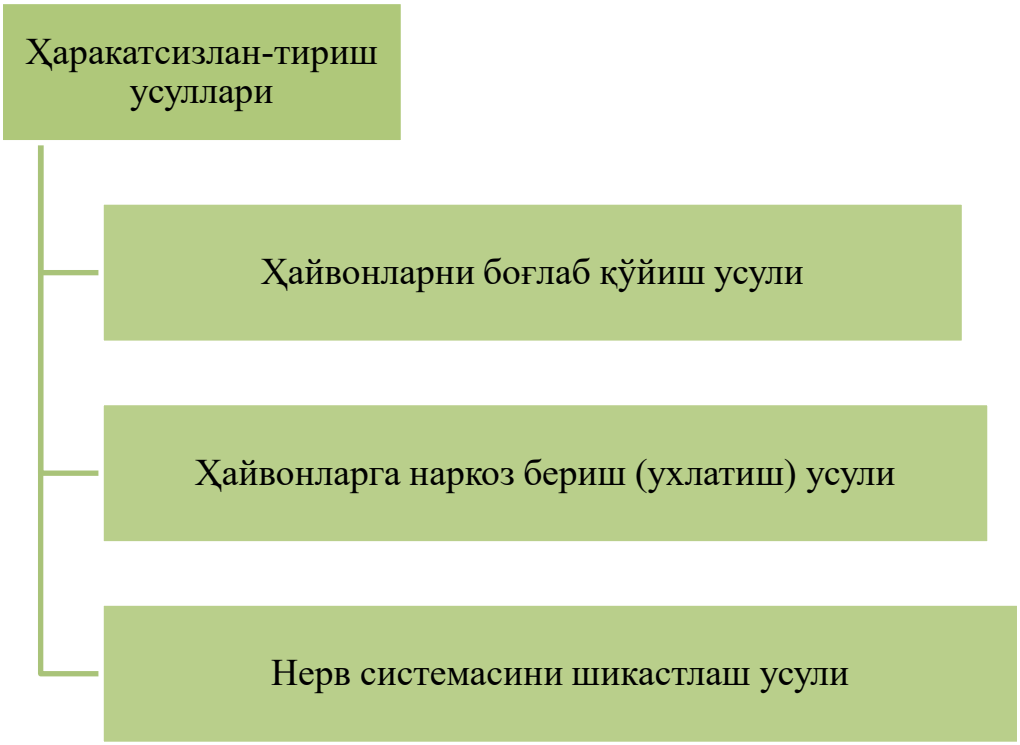
500. What is the attachment of the liver instead of parenchymal cells called tissue outflow?

A. Hepatoma
B. Hepatosis
D. Hepatitis
E. Cirrhosis

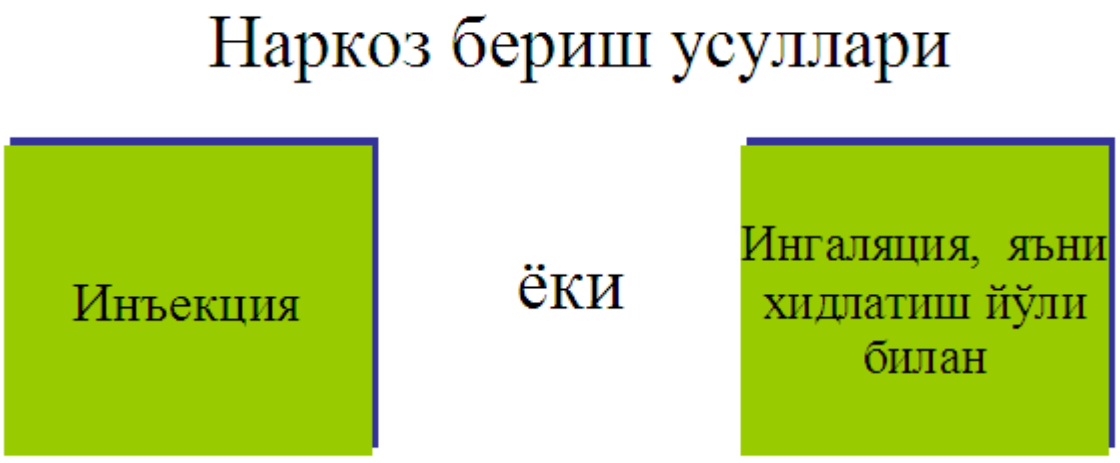
Science Assessment Criteria

Rating
Students' academic performance is assessed on a 5-point scale.
5 (excellent) rating:
Conclusion and decision making; Getting creative ideas;
Ability to observe independently; To be able to apply the acquired knowledge in practice; Understand the essence;
To know, to tell; Imagination; 4 (good) rating:
Ability to observe independently; To be able to apply the acquired knowledge in practice; Understand the essence;
To know, to tell; Imagination; 3 (satisfactory) evaluation; Understand the essence; To know, to tell; Imagination; 2 (unsatisfactory) rating:
Not mastering the program; Not knowing the essence of science; Lack of clarity; Inability to think independently.

Handouts on science




Тажриба усуллари икки катта гуруҳга бўлинади
Ўткир тажриба усуллари
Сурункали тажриба усуллари

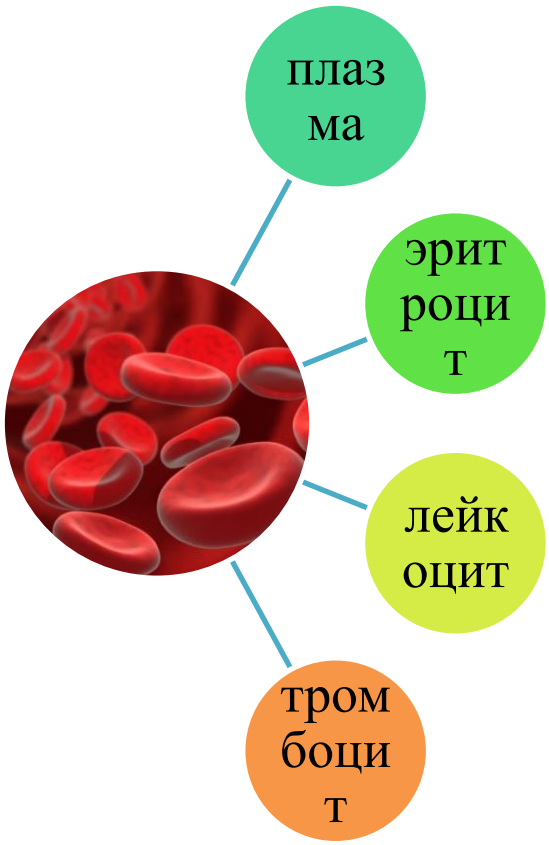

Наркоз бериш усуллари
Иньекция
ёки
Ингаляция, яъни хидлатиш йўли билан

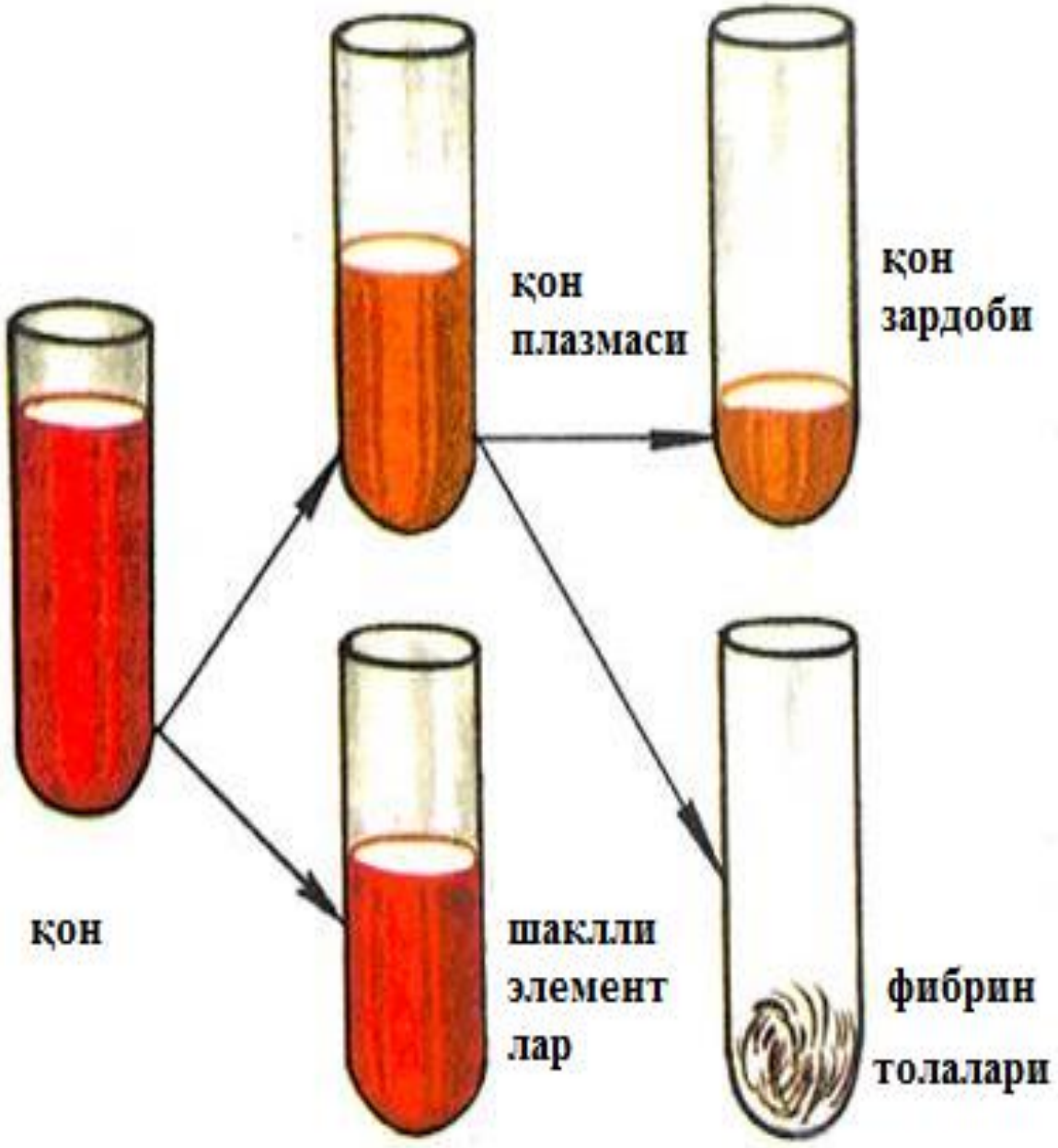

Вивисекция усули
Органларни изоляция килиш усули
Экстерпация усули
Ўткир тажриба усуллари
томирлар анастомози
Трансплантация усули
лигатуралар солиш усули
Денервация усули

Xronik(surunkali) usullar:

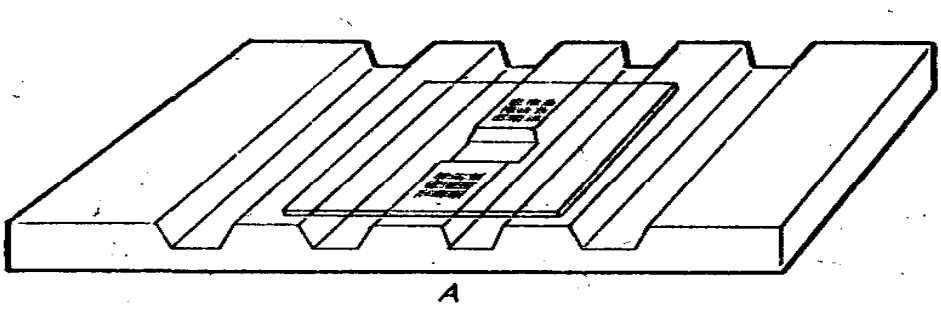
2-Mavzu. QON PLAZMASI VA QON ZARDOBINI AJRATIB OLISh

Darsning maqsadi: Qon, qon plazmasi, qon zardobi va fibrinsizlantirilgan qon haqida tushunchaga ega bo'lish hamda qon plazmasi, qon zardobi va fibrinni ajratib olishni o'rganish.

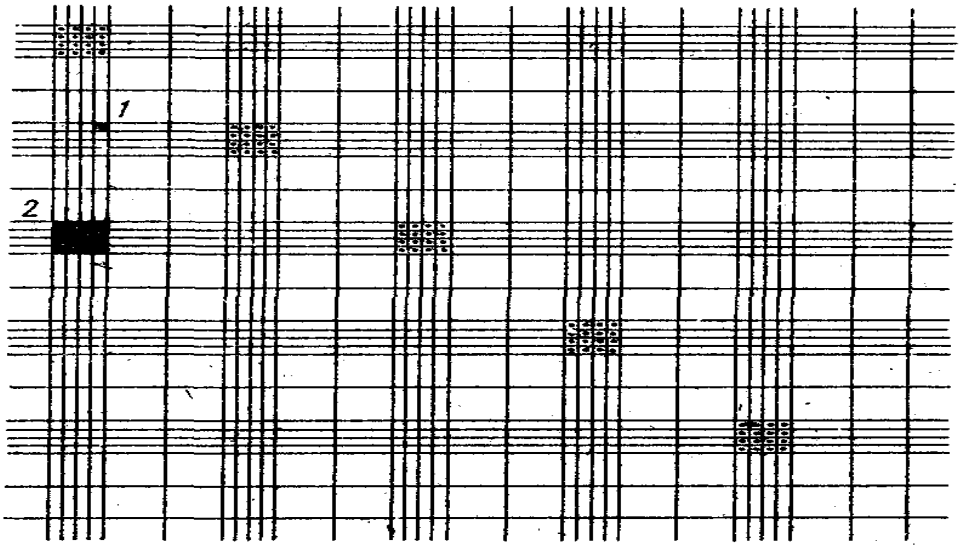




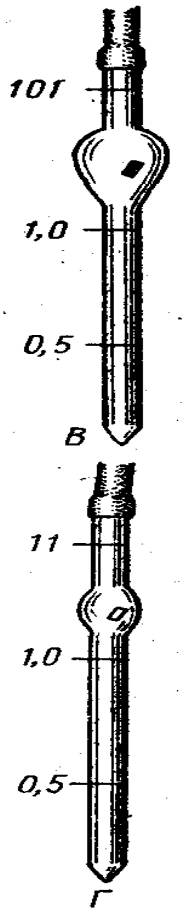
Demak, qon plazmasida 0,4% gacha fibrinogen bo’lishi bilan zardob farqlanadi.

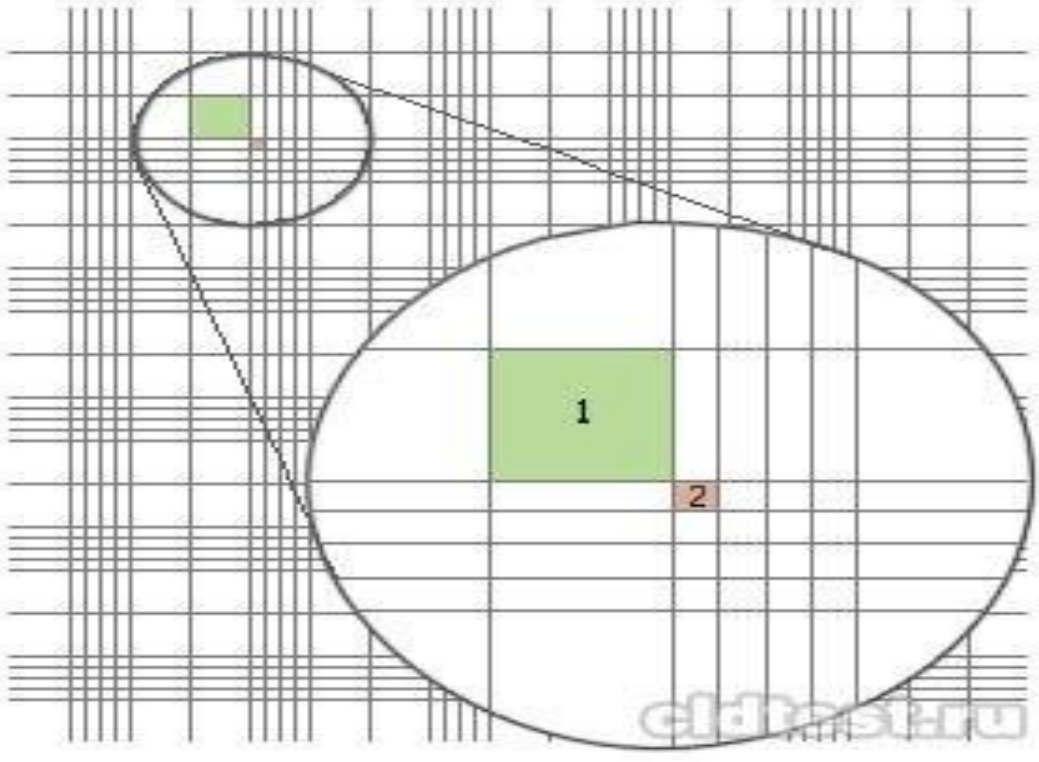


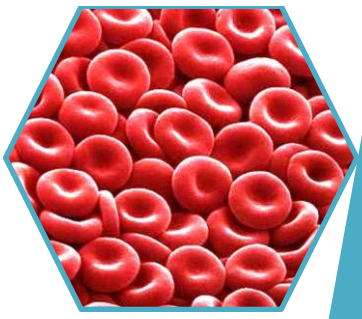
А



Б



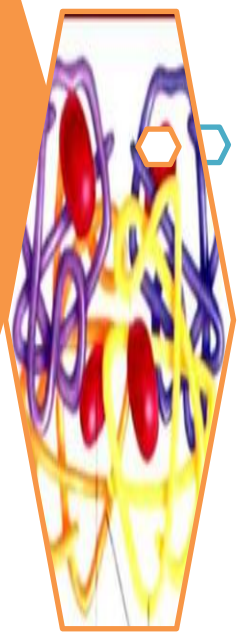




Эритроцитларнинг ўртача диаметри 4-7 микронга тенг

- **таркиби 60 % сув, 40% қуруқ модда**

қуруқ модданинг 90% ни гемоглобин, 5,8 % ни оқсиллар, қолган қисмини липоидлар, глюкоза, минерал тузлар ташкил қилади.



Грек.
- erythrocyti (erythros kutos)
- яъни қизил қон таначалари

грек.
- erythrocytosis
- яъни эритроцитларнинг кўпайиб кетиши

грек. .
- erythropenia
- яъни эритроцитларнинг камайиб кетиши

грек. .
- erythropoieses
- яъни эритроцитларнинг ҳосил бўлиши

Kavsh qaytaruvchi hayvonlarda va cho’chqalarda 1-1,5 oy

Otlarda 95 kun

Qoramollarda 130 kun

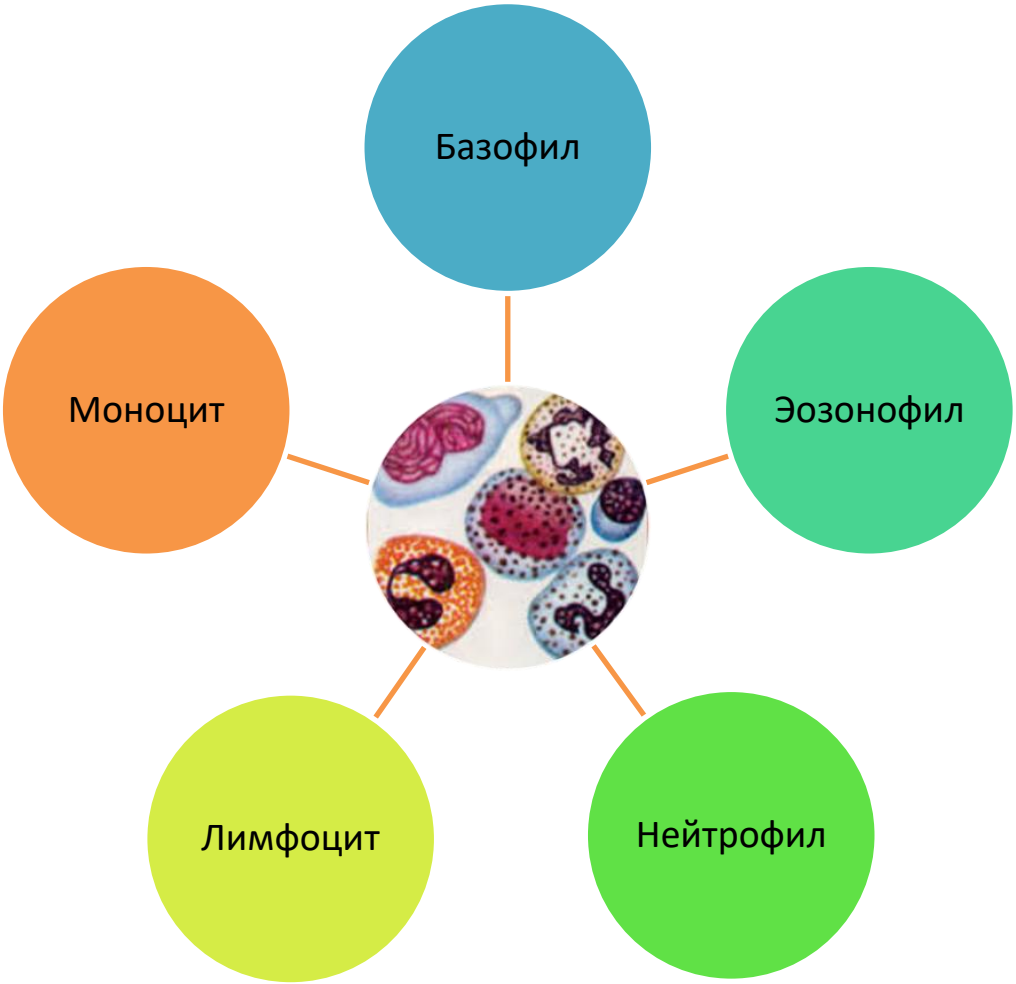
o’rtacha 120 kun muddatda yashaydi. Umri tugagan eritrositlar jigar va taloqda parchalanadi.

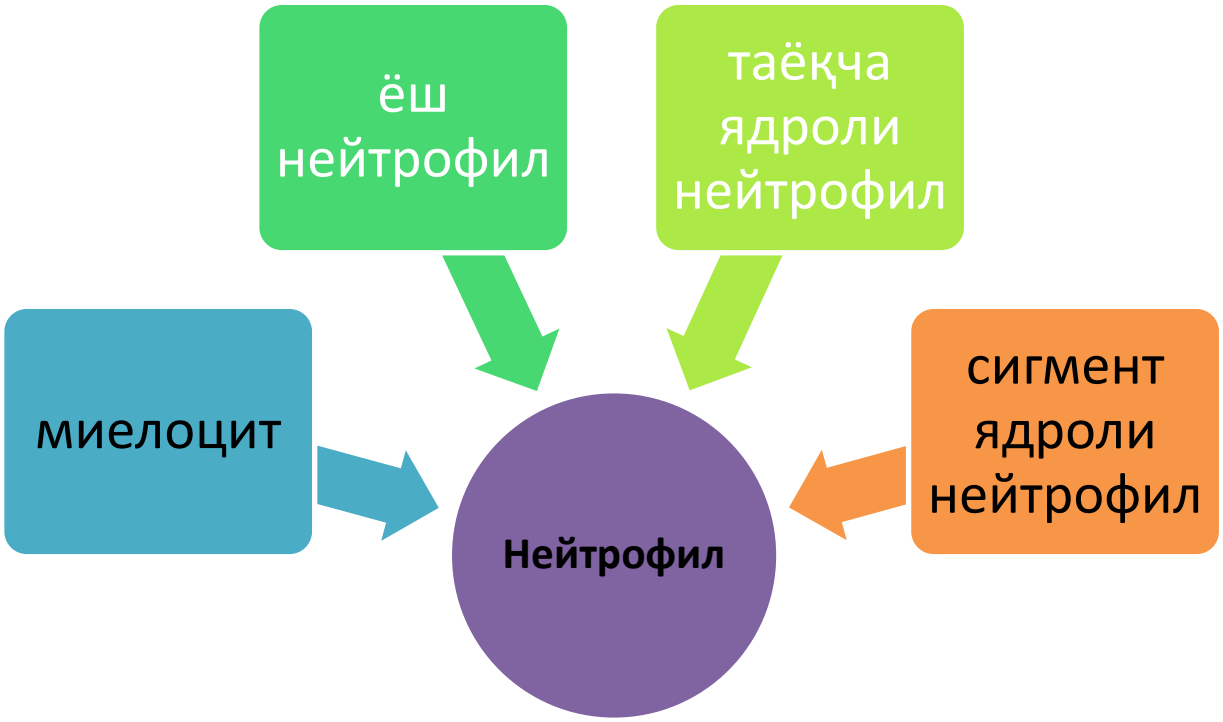
Грек.
- leucocytus
- яъни оқ қон таначалари

грек.
- leucocytosis
- яъни лейкоцитларнинг кўпайиб кетиши

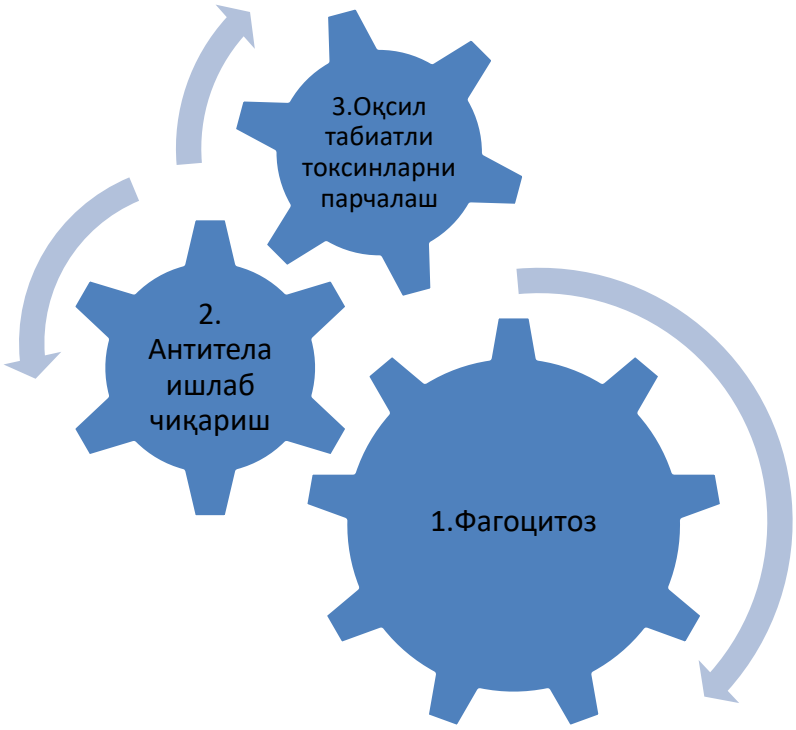
грек. .
- leucopenia
- яъни лейкоцитларнинг камайиб кетиши

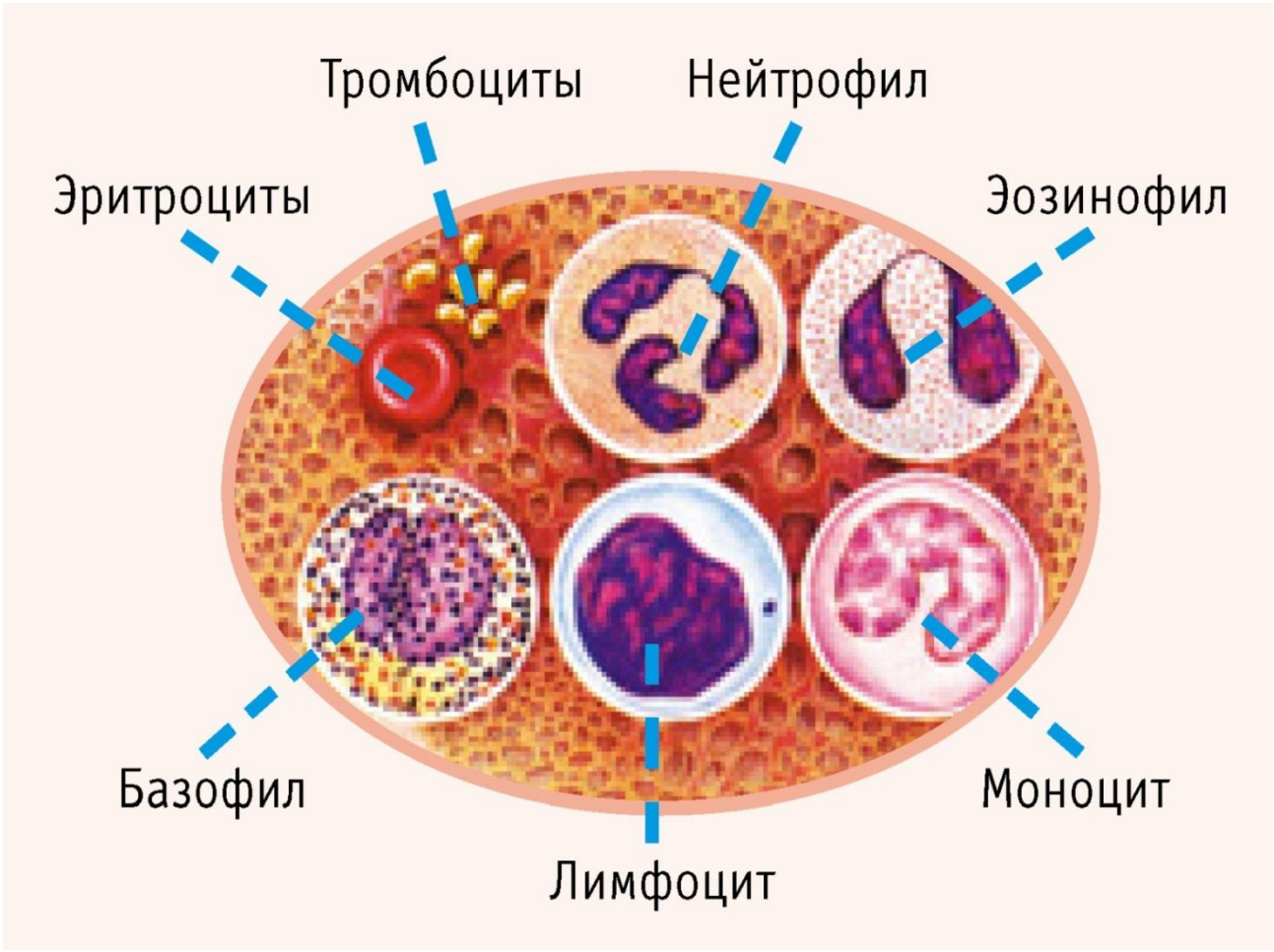
грек. .
- leucopoieses
- яъни лейкоцитларнинг ҳосил бўлиши

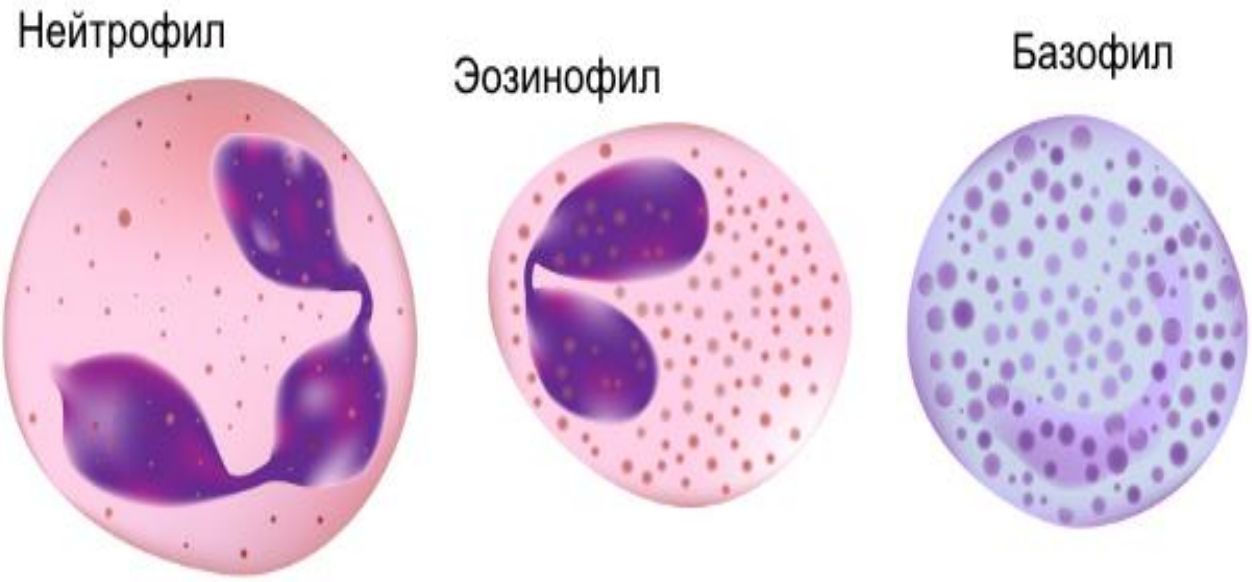




Leykositlarning organizmdagi asosiy vazifasi:




Тромбоциты
Нейтрофил
Эритроциты
Эозинофил
Базофил
Моноцит
Лимфоцит


Нейтрофил
Эозинофил
Базофил

Leykositlar klassifikasiyasi

Базофиллар - ишқорли бўёқлар билан бўялади ва лейкоцитларнинг 0-7 % ни ташкил этади. Базофилларнинг доначаларида қоннинг ивишига тўс-қинлик қилувчи ***антикоагулянт*** - ***гепарин*** деган модда ишлаб чиқарилади. Бу модда яллиғланган тўқимада қоннинг ивишига ёрдам беради.

Эозинофиллар - кислотали буёқлар билан бўялади ва лейкоцитларнинг 2-12% ни ташкил килади.Эозинофиллар оқсил табиатли токсинларни ней-траллаш вазифасини бажаради.

Нейтрофиллар - ҳам кислотали, ҳам ишқорли бўёқлар билан бўялади ва лейкоцитларнинг 18-60% ни ташкил қилади. Нейтрофиллар фагоцитоз қилиш хусусиятига эгадир.

Лимфоцитлар - ядроси билан протоплазмаси ўртасида перенуклеар, яъни бўялмайдиган қисмининг борлиги билан ифодаланади ва лейкоцитлар-нинг 20-65% ни ташкил этади. Лимфоцитлар антителалар ишлаб чиқишда ва иммунитет ҳосил бўлишида катта ролъ ўйнайди.

Моноцитлар - ядроси ҳар хил тузилишга эга бўлган энг йирик ҳужай-ралар бўлиб лейкоцитларнинг 1-7% ни ташкил этади. Моноцитлар регене-рация, яъни тикланиш жараёнларида катта аҳамиятга эгадир.

Medicine Live

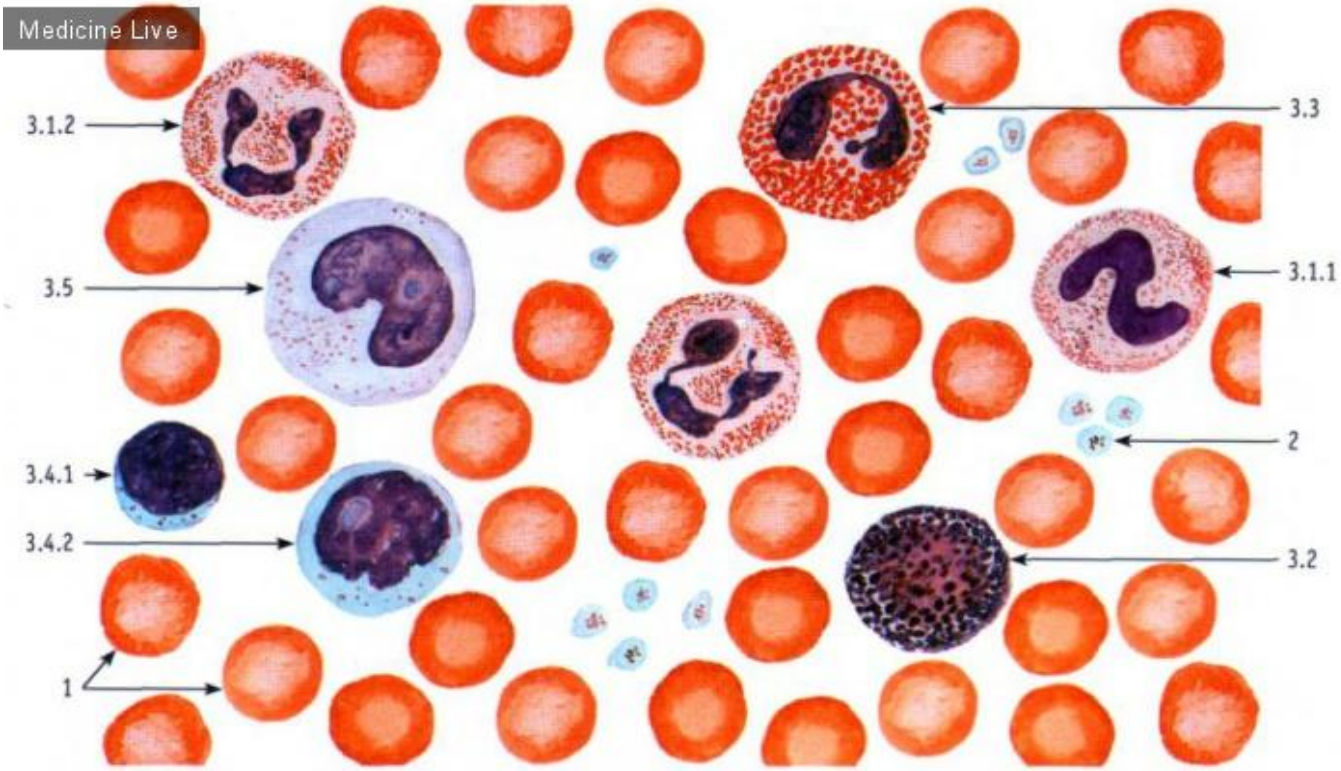
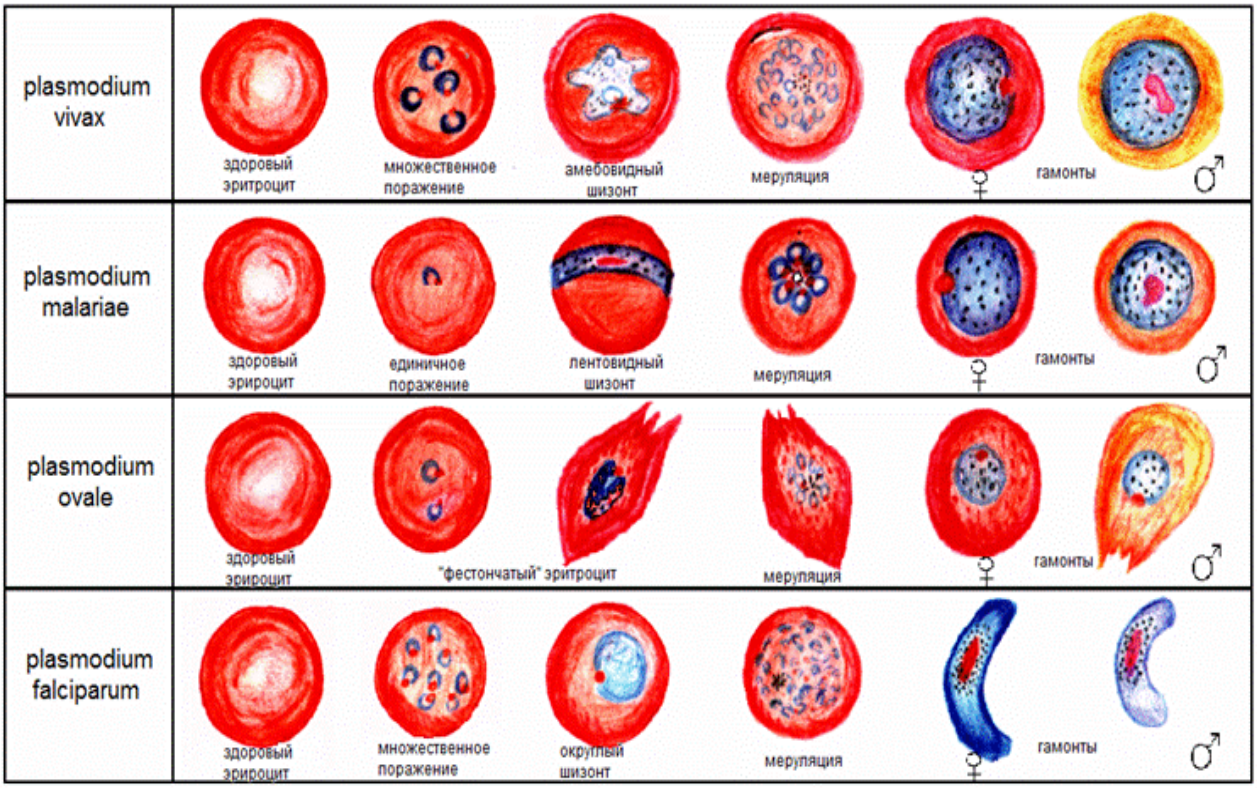
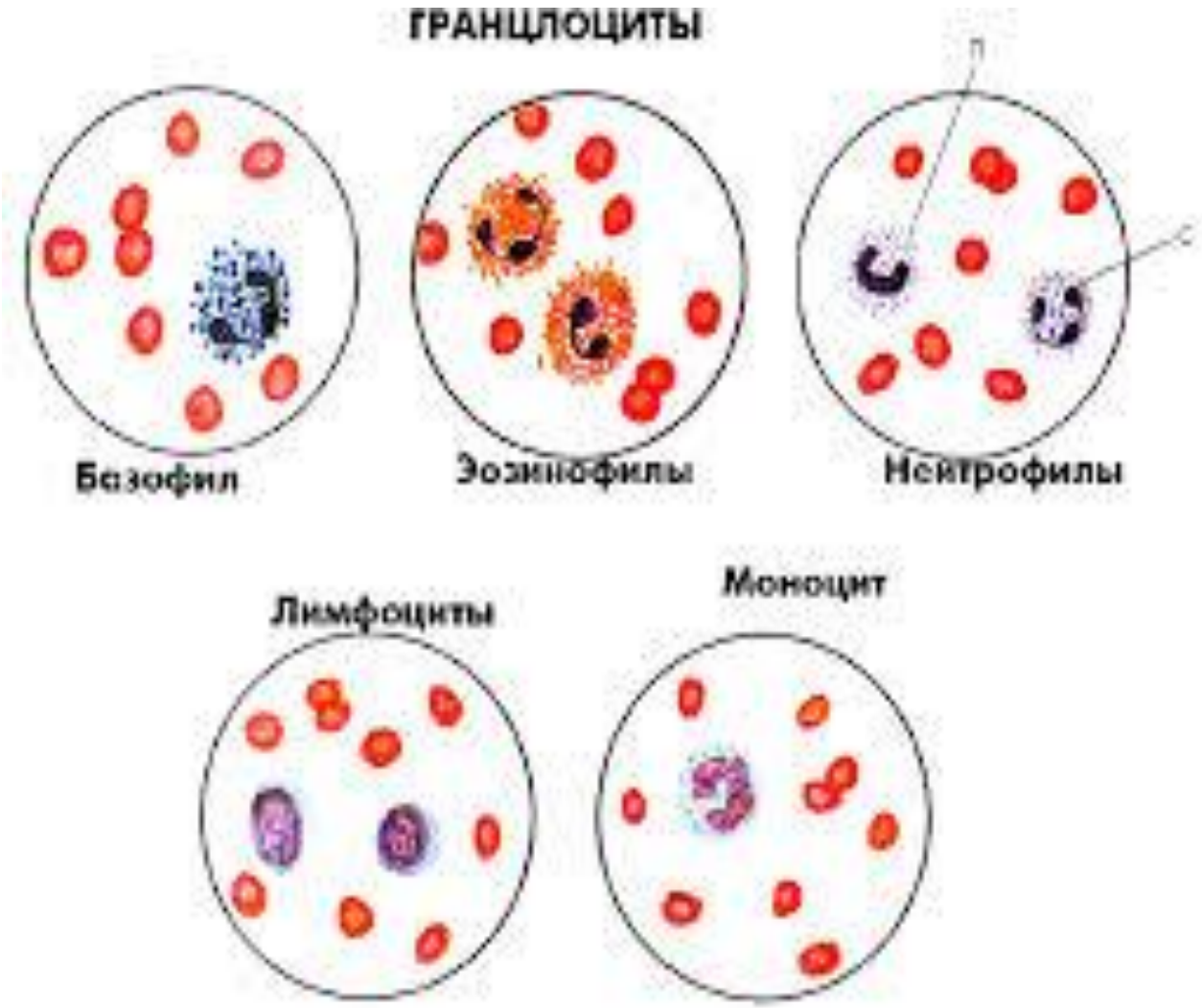


Рис. 47. Кровь человека (мазок)

Окраска: по Романовскому-Гимзе

1 – эритроциты; 2 – тромбоциты; 3 – лейкоциты: 3.1 – нейтрофильные гранулоциты (3.1.1 – палочкоядерный, 3.1.2 – сегментоядерный), 3.2 – базофильный гранулоцит, 3.3 – эозинофильный гранулоцит, 3.4 – лимфоциты (3.4.1 – малый лимфоцит, 3.4.2 – средний лимфоцит), 3.5 – моноцит



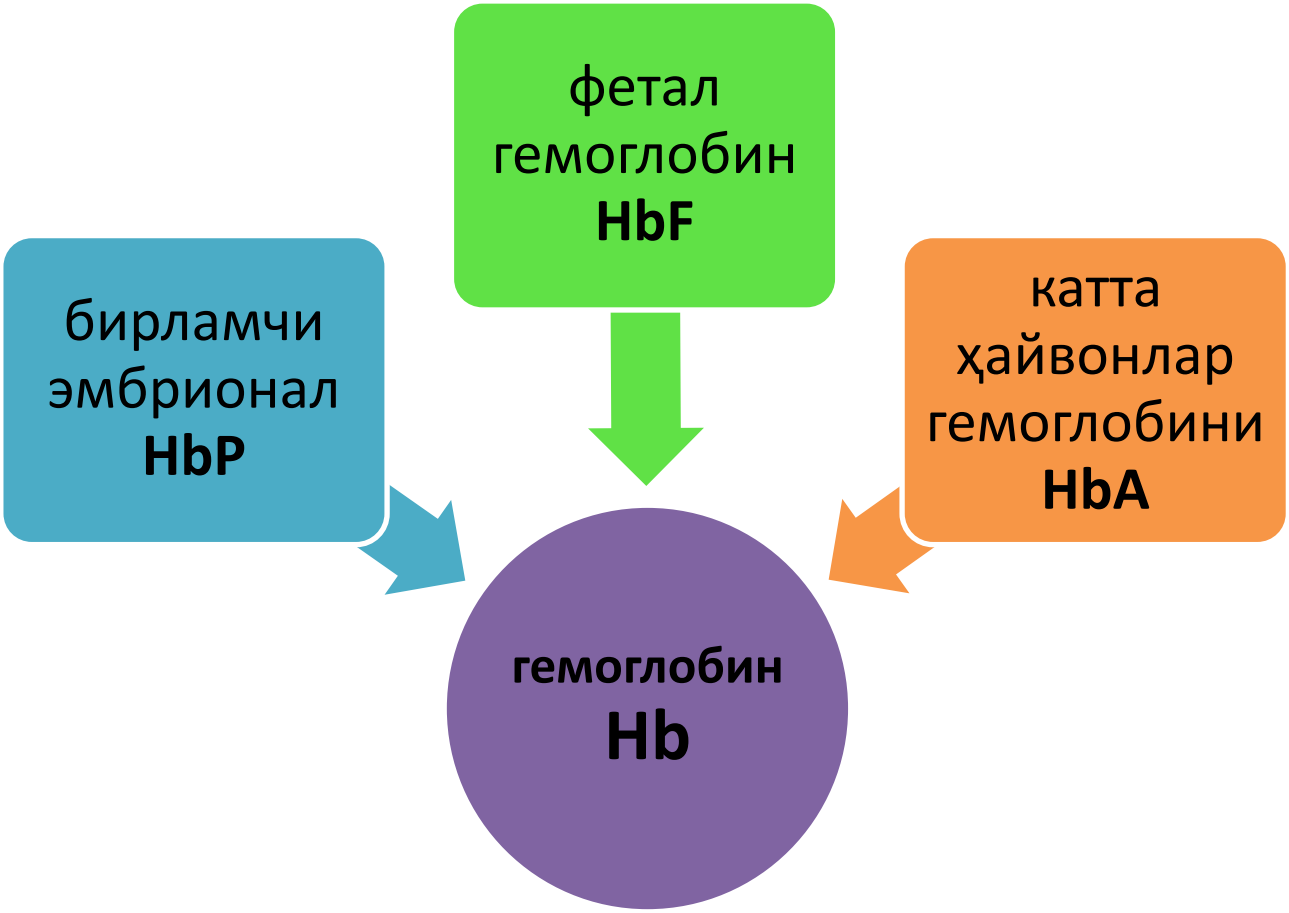


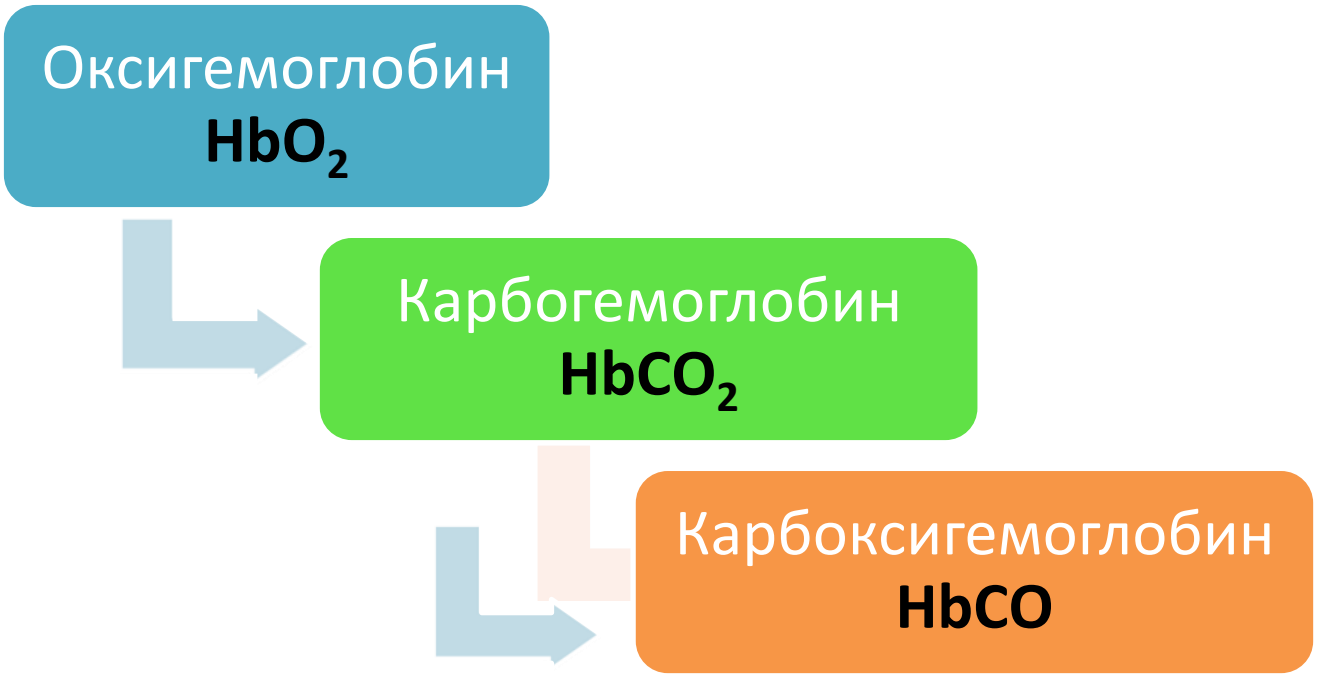
Лейкоцитларни санаш усуллари

- Тўрт майдон усули
- Филлипченко усули
- Суртманинг ўртасидан санаш усули

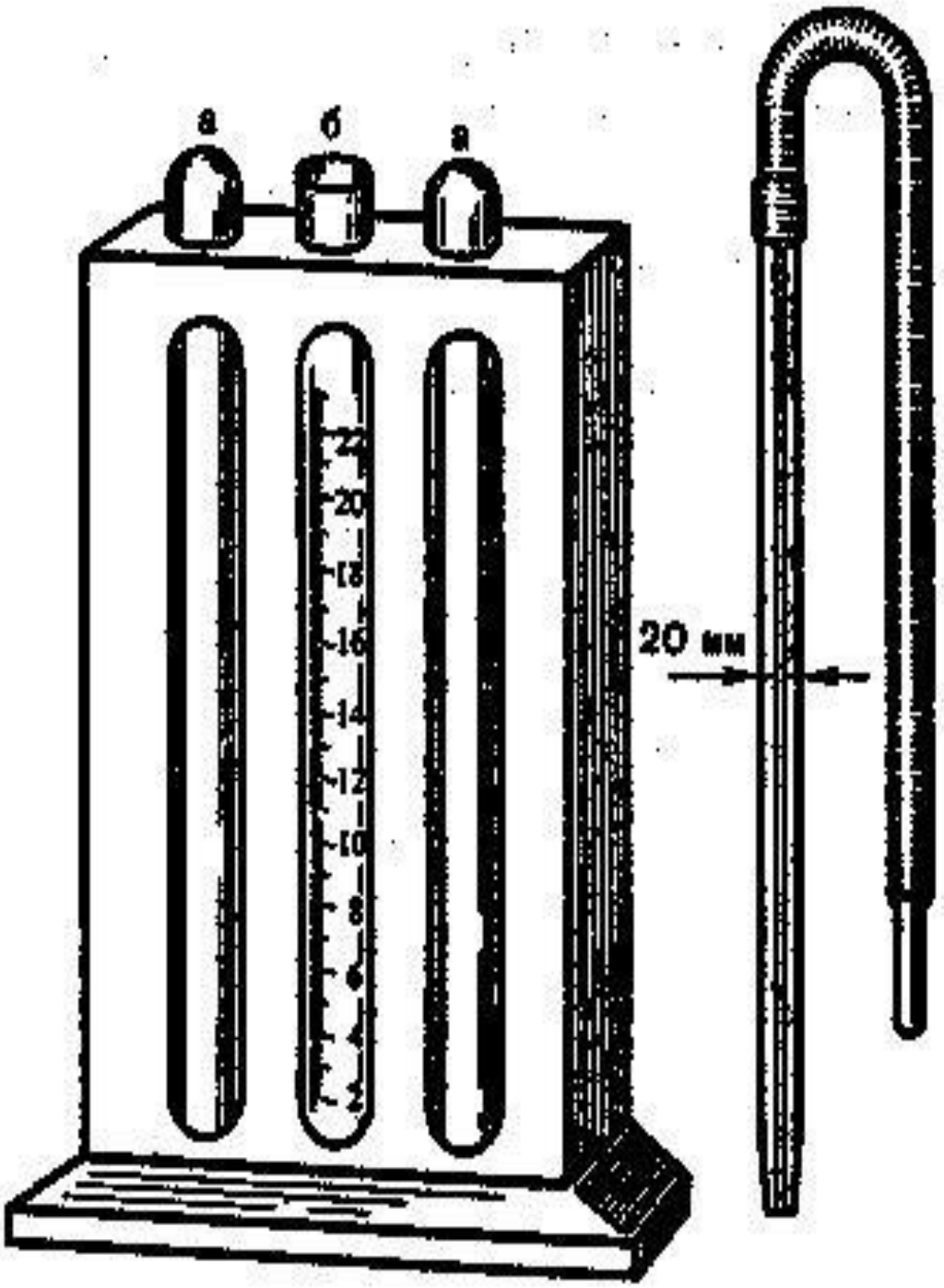
Животные	азофилы	Эозинофилы	Нейтрофилы			Лимфоциты	Моноциты
			Ю				
Крупн. рог. скот	0-2	5-8	0-1	2-5	20-35	40-65	2-7
Овца	0-1	4-12	0-2	3-6	35-45	40-50	2-5
Коза	0-1	3-12	-	1-5	29-38	47-64	2-4
Верблюд	0-1	4-12	0-2	1-6	40-52	29-45	1-5
Олень	0-1	3-7	0-1	2-5	55-66	21-37	1-4
Буйвол	0-2	3-10	-	1-6	24-46	45-66	2-5
Як	0-2	2-3	0-1	2-8	20-43	40-76	2-9
Лошадь	0-1	2-6	0-1	3-6	45-62	25-44	2-4
Осел	0-1	2-4	-	2-6	50-80	18-38	1-3
Свинья	0-1	1-4	0-2	2-4	40-80	40-50	2-6
Собака	0-1	3-9	-	1-6	43-71	21-40	1-5
Кошка	0-1	2-8	0-1	3-9	40-45	36-51	1-5
Лиса сер.-черн.	0-1	3-20	0-1	3-10	20-50	22-60	2-4
Кролик	0-2	1-3	-	5-9	33-39	43-62	1-3
Норка	0-1	2-8	0-1	5-10	45-65	26-45	2-4
Песец	0-1	1-9	0-2	1-25	29-54	25-78	1-8
Соболь	0-2	3-13	0-2	2-8	15-35	40-75	2-5
Морская свинка	0-2	4-12	0	1-5	30-45	36-54	3-8
Крыса белая	0-1	1-5	0	1-4	20-35	55-75	1-5
Мышь белая	0-2	0-4	0	1-5	18-30	60-78	2-5
Хомяк золот.	0-1	0-1	0	3-10	22-32	58-72	1-2
Еж	1-5	2-7	0	2-4	15-30	57-80	0-3
Курица	1-3	6-10	-	-	24-30*	52-60	4-10
Гусь	1-4	3-9	-	-	30-44*	40-56	2-6
Утка	0-5	4-12	-	-	30-42*	42-59	2-7
Голубь	1-5	2-8	-	-	28-54	38-54	1-5
Индейка	0-3	0-3	-	-	30-42	49-60	4-8
Цесарка	0-3	6-10	-	-	30-42	45-55	2-6

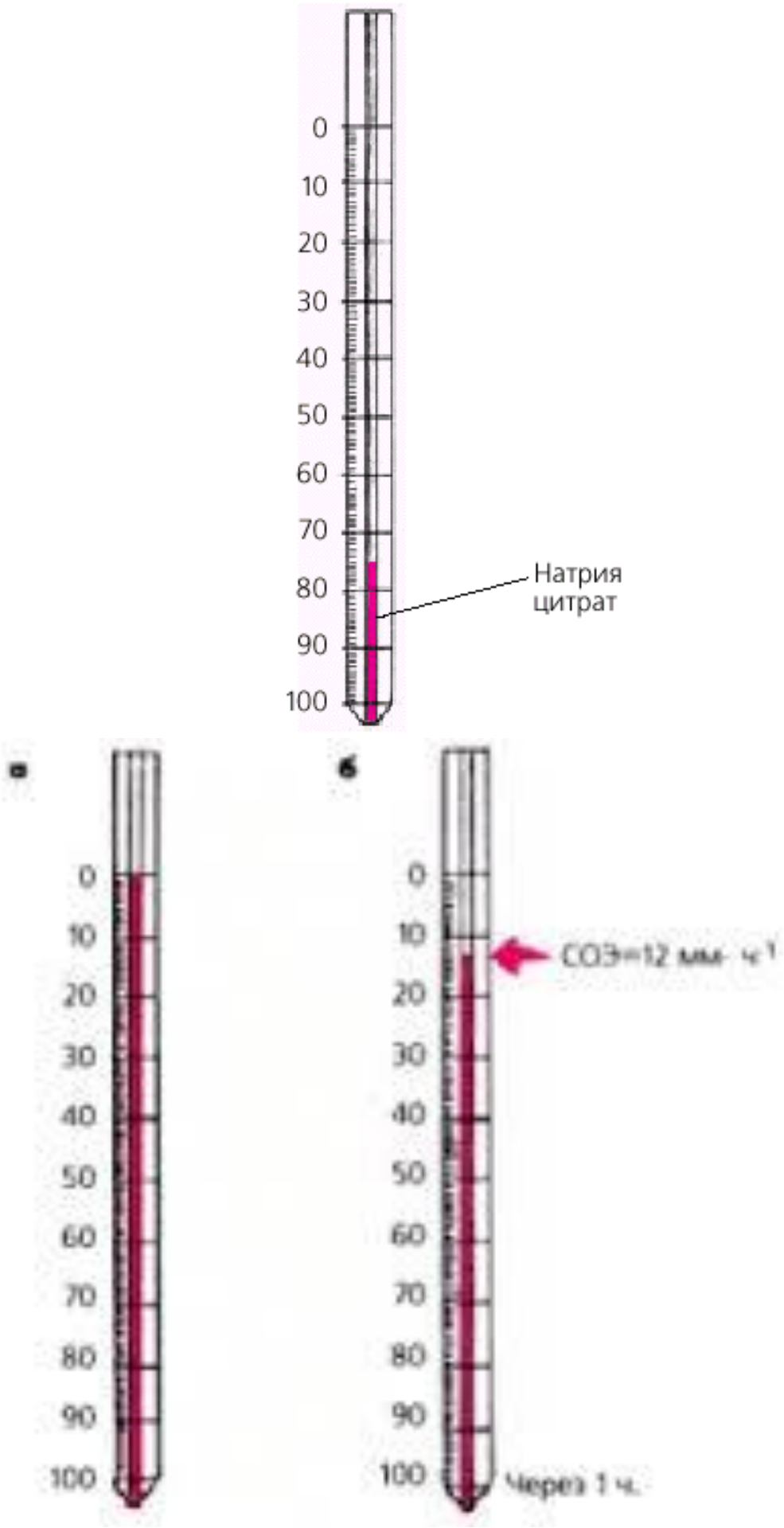
Fiziologik gemoglobinning 3 xili farqlanadi.



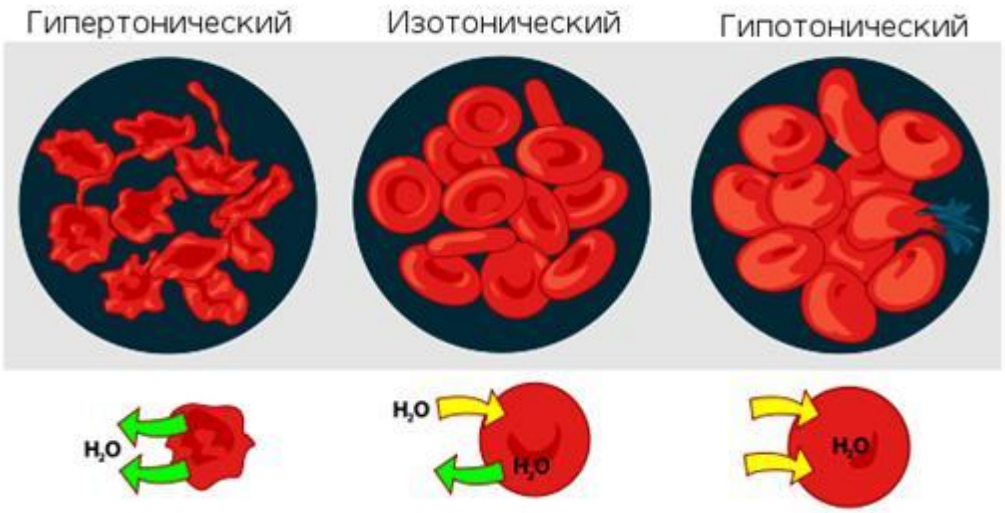

Оксигемоглобин
HbO2
Карбогемоглобин
HbCO2
Карбоксигемоглобин
HbCO


Метгемоглобин
HbO
Миоглобин


а
б
в
20 мм

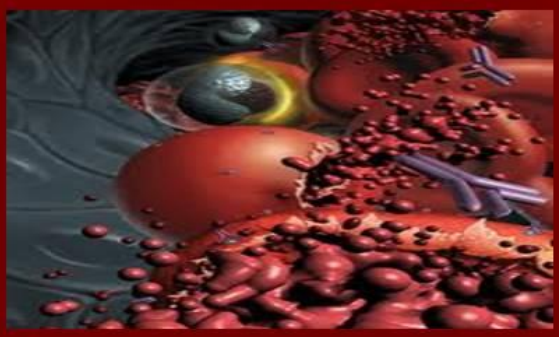

0
10
20
30
40
50
60
70
80
90
100
Натрия цитрат
а
0
10
20
30
40
50
60
70
80
90
100
б
0
10
20
30
40
50
60
70
80
90
100
СОЭ=12 мм· ч⁻¹
Через 1 ч.

10-Mavzu: GEMOLIZ. ERITROSITLARNING OSMOTIK REZISTENTLIGINI ANIQLASh



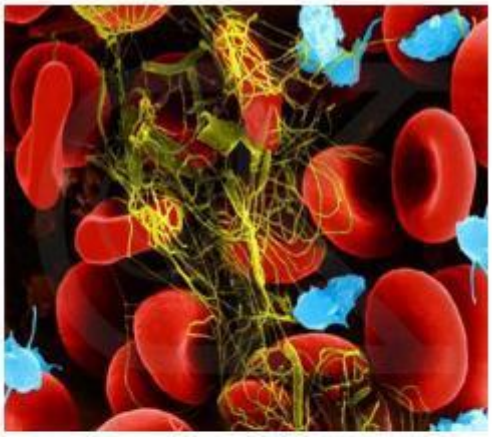
Виды гемолиза эритроцитов

- Осмотический
- Механический
- Термический
- Биологический
- Химический



- Электронная микрофотография гемолиза эритроцитов

11-Mavzu: QONNING IVISh TEZLIGINI ANIQLASh



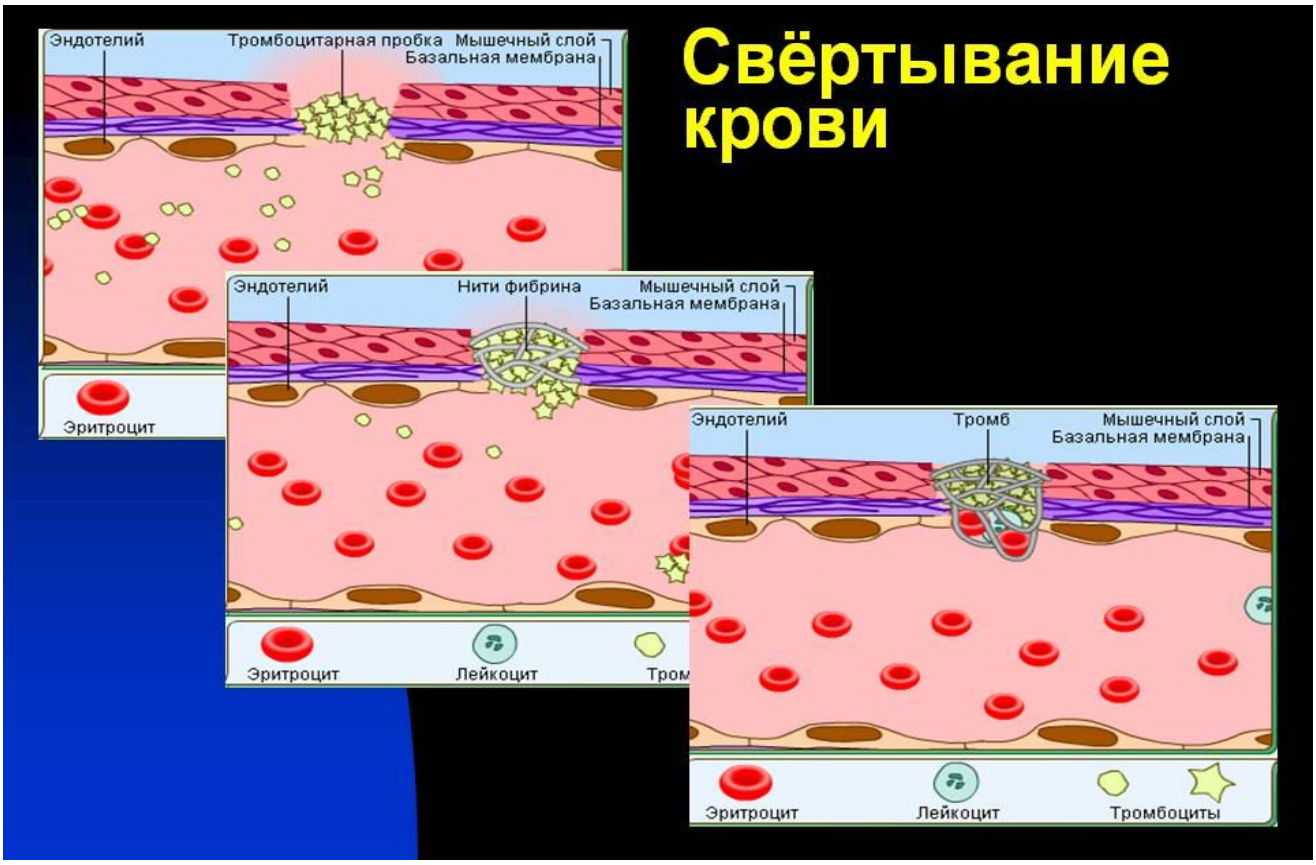
Свертывание крови

Коагуляция — процесс свёртывания крови.

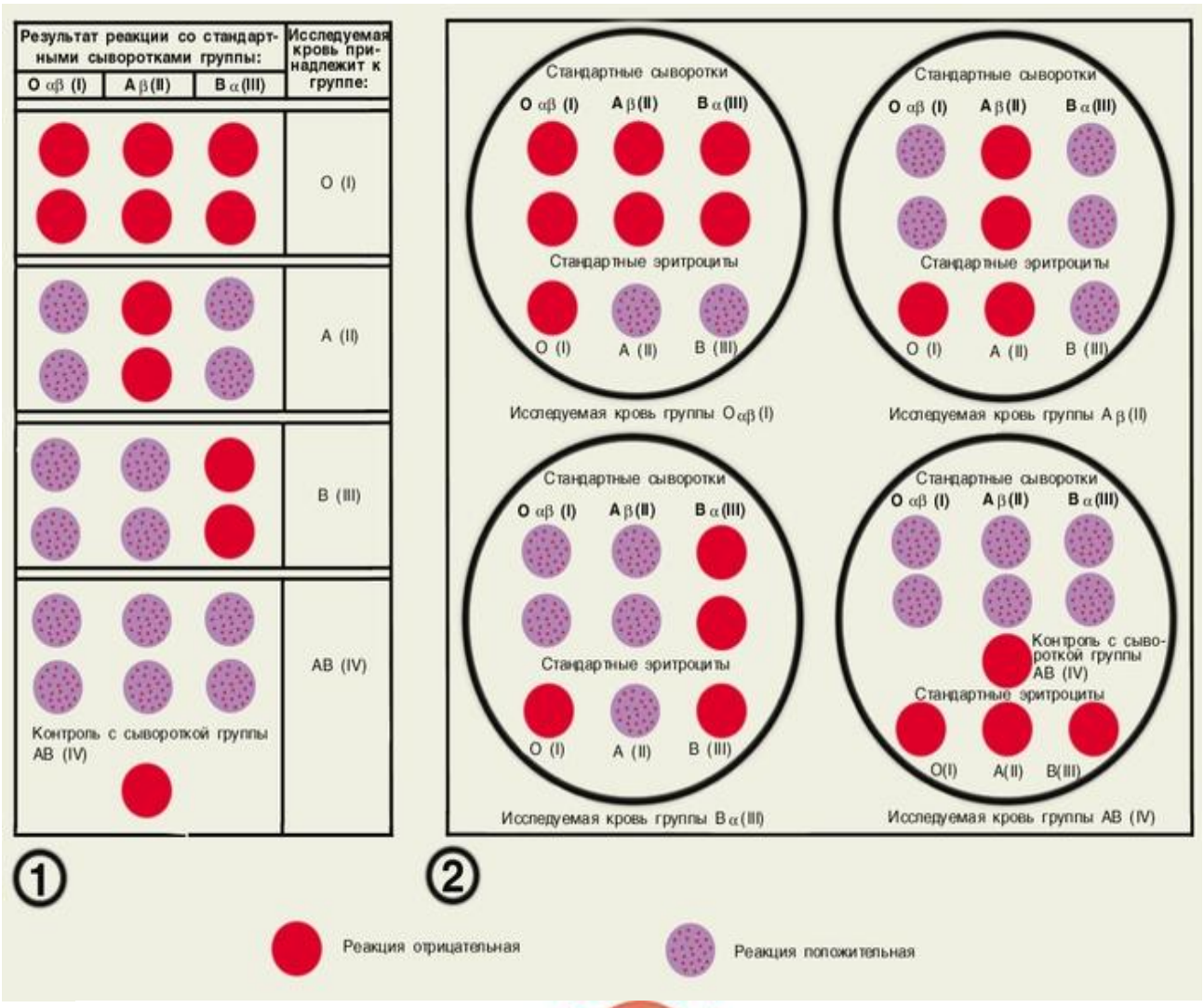
тромбоциты

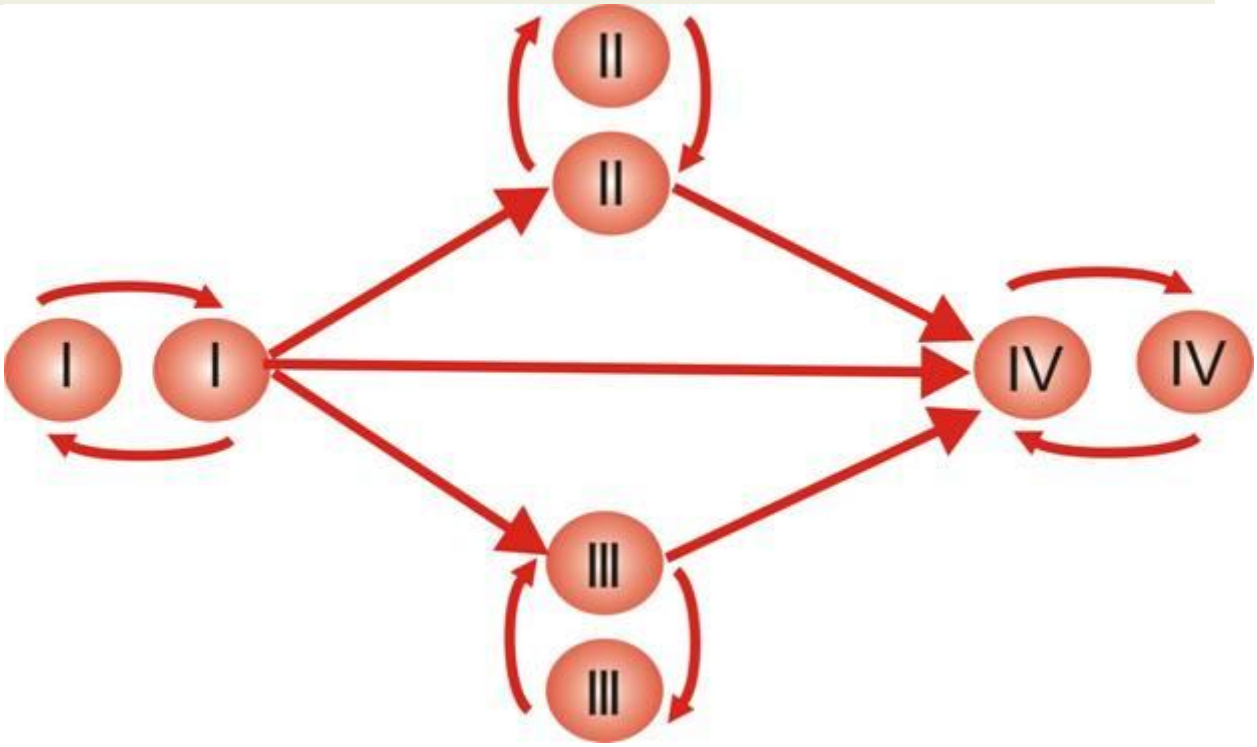
тромбопластин + кальций + витамин К + протромбин

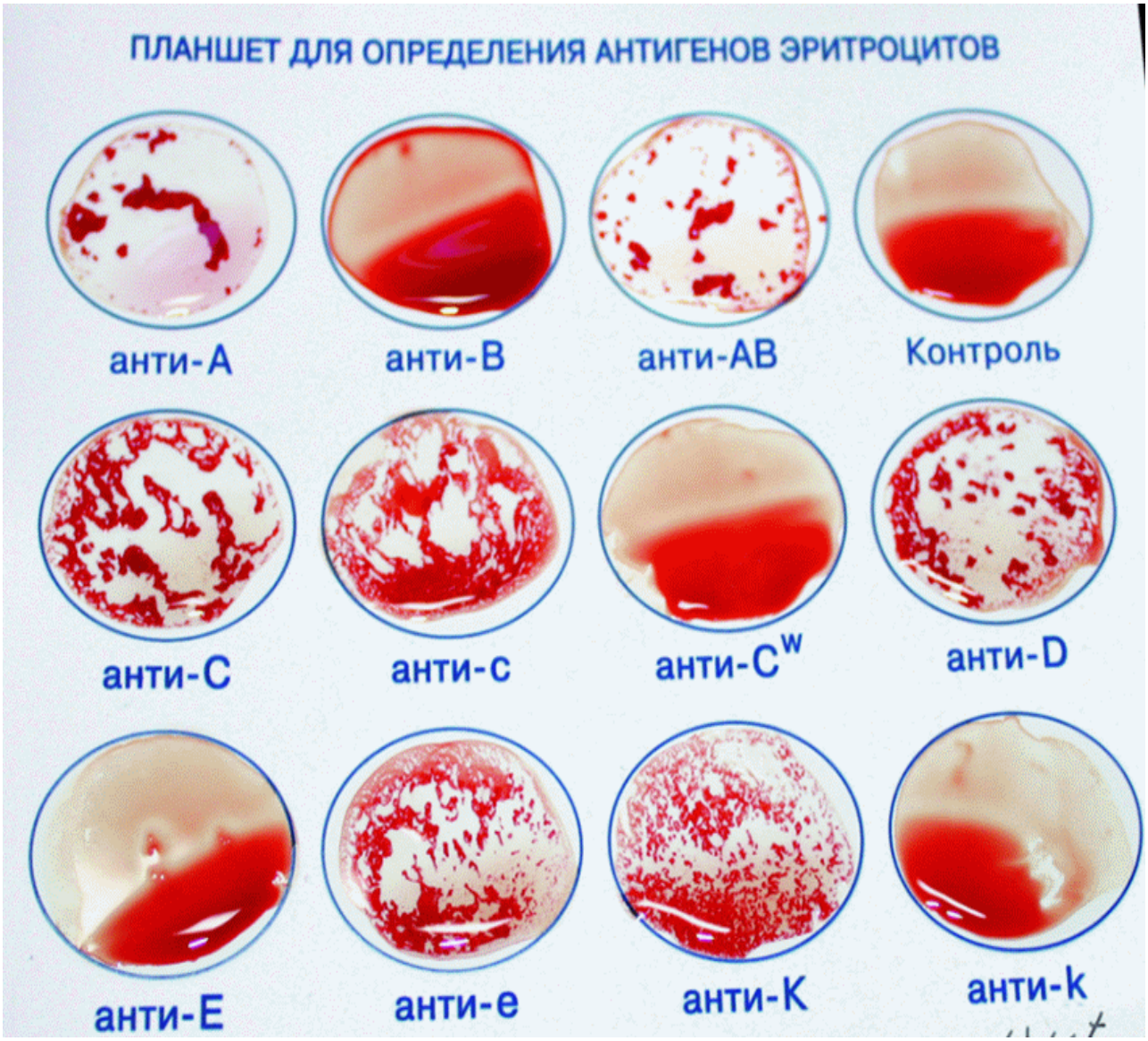
Фибриноген → Фибрин → Тромб



12-Mavzu: QON GURUXLARINI ANIQLASh







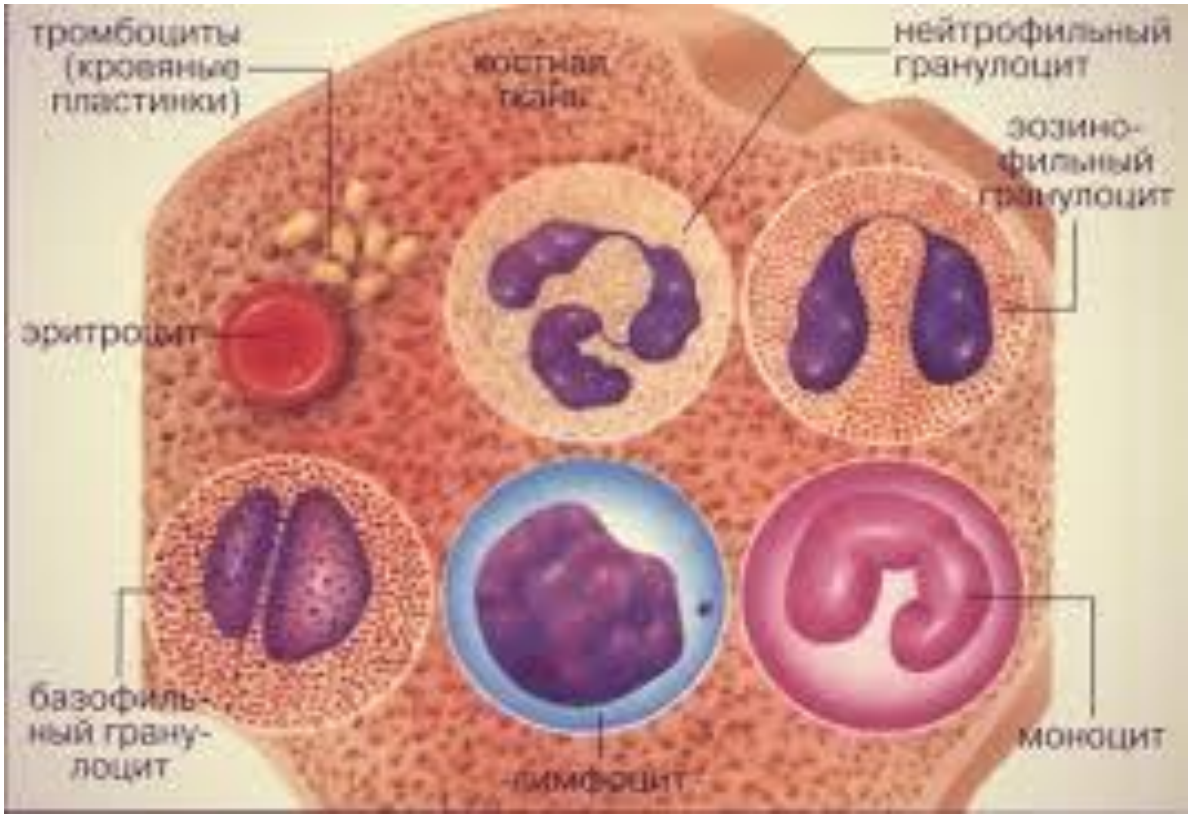
резус-фактор		Rh- (--)		Rh+ (-+)		Rh+ (++)	
	ген	-	-	-	+	+	+
Rh- (--)	-	Rh- (--)	Rh- (--)	Rh- (--)	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)
	-	Rh- (--)	Rh- (--)	Rh- (--)	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)
Rh+ (-+)	-	Rh- (--)	Rh- (--)	Rh- (--)	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)
	+	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)	Rh+ (++)	Rh+ (++)	Rh+ (++)
Rh+ (++)	+	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)	Rh+ (++)	Rh+ (++)	Rh+ (++)
	+	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)	Rh+ (++)	Rh+ (++)	Rh+ (++)

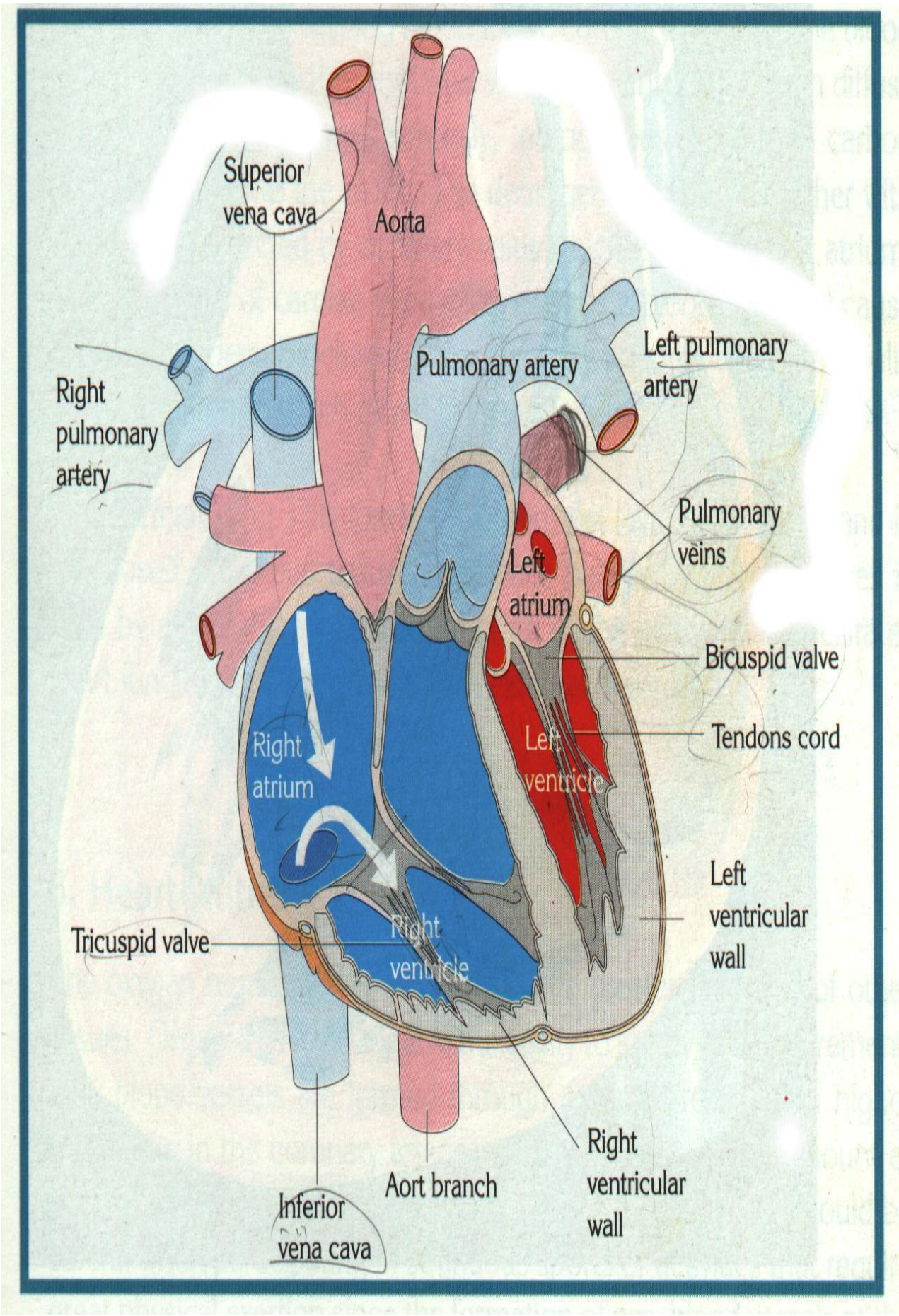
эритроцит заполнен гемоглобином

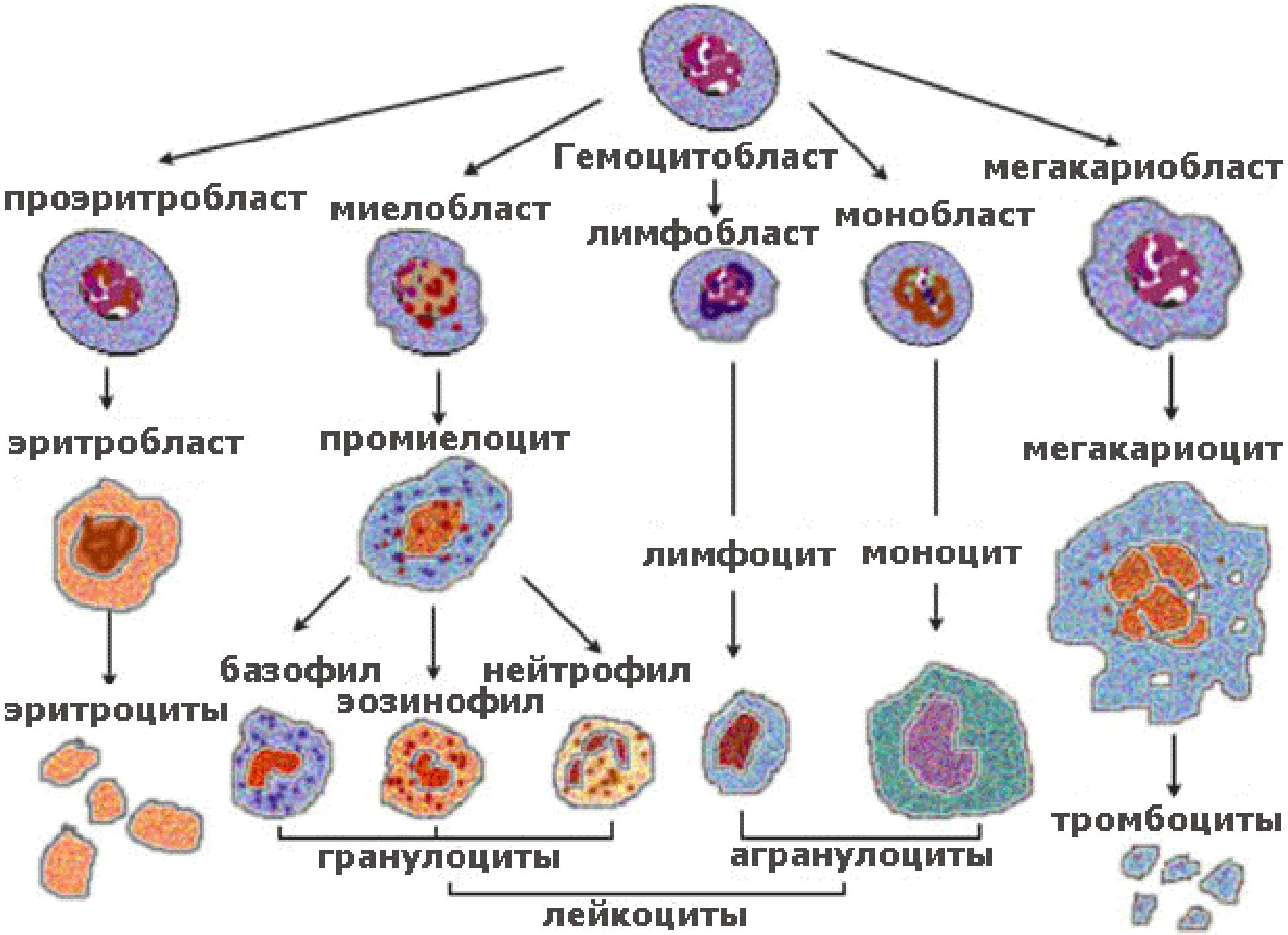
△ *ЭРИТРОЦИТ наполнен специальным белком — гемоглобином*

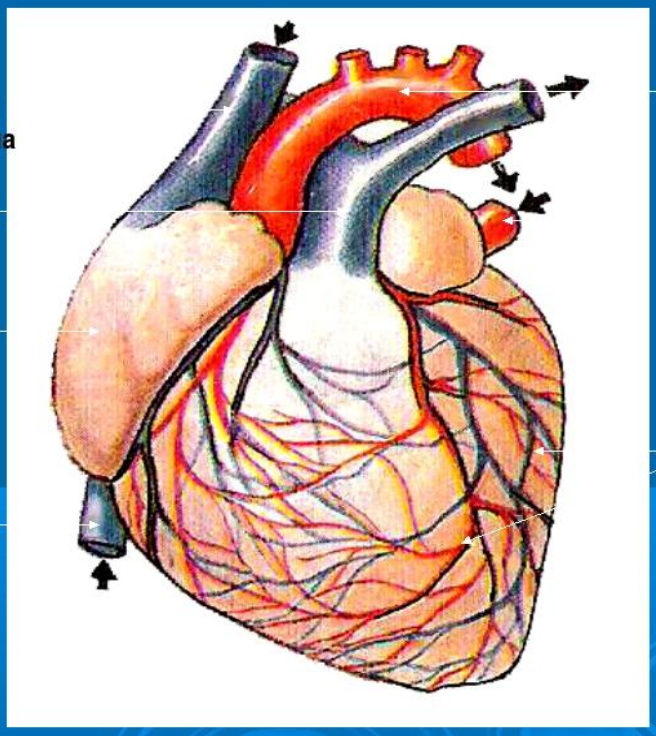
- 1. Гемоглобин состоит из четырех белковых нитей.
- 2. К каждой нити прикреплен один гем.
- 3. Гем содержит атом железа и способен удерживать одну молекулу кислорода.

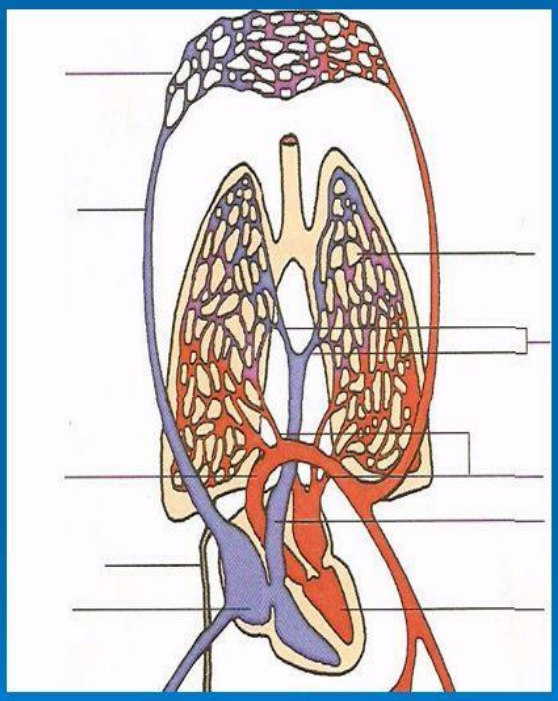
PPt4WEB.ru




Superior vena cava
Aorta
Pulmonary artery
Left pulmonary artery
Right pulmonary artery
Pulmonary veins
Left atrium
Bicuspid valve
Tendons cord
Right atrium
Left ventricle
Left ventricular wall
Tricuspid valve
Right ventricle
Right ventricular wall
Aort branch
Inferior vena cava


Гемоцитобласт
проэритробласт
миелобласт
лимфобласт
монобласт
мегакариобласт
эритробласт
промиелоцит
мегакариоцит
лимфоцит
моноцит
базофил
эозинофил
нейтрофил
эритроциты
тромбоциты
гранулоциты
агранулоциты
лейкоциты

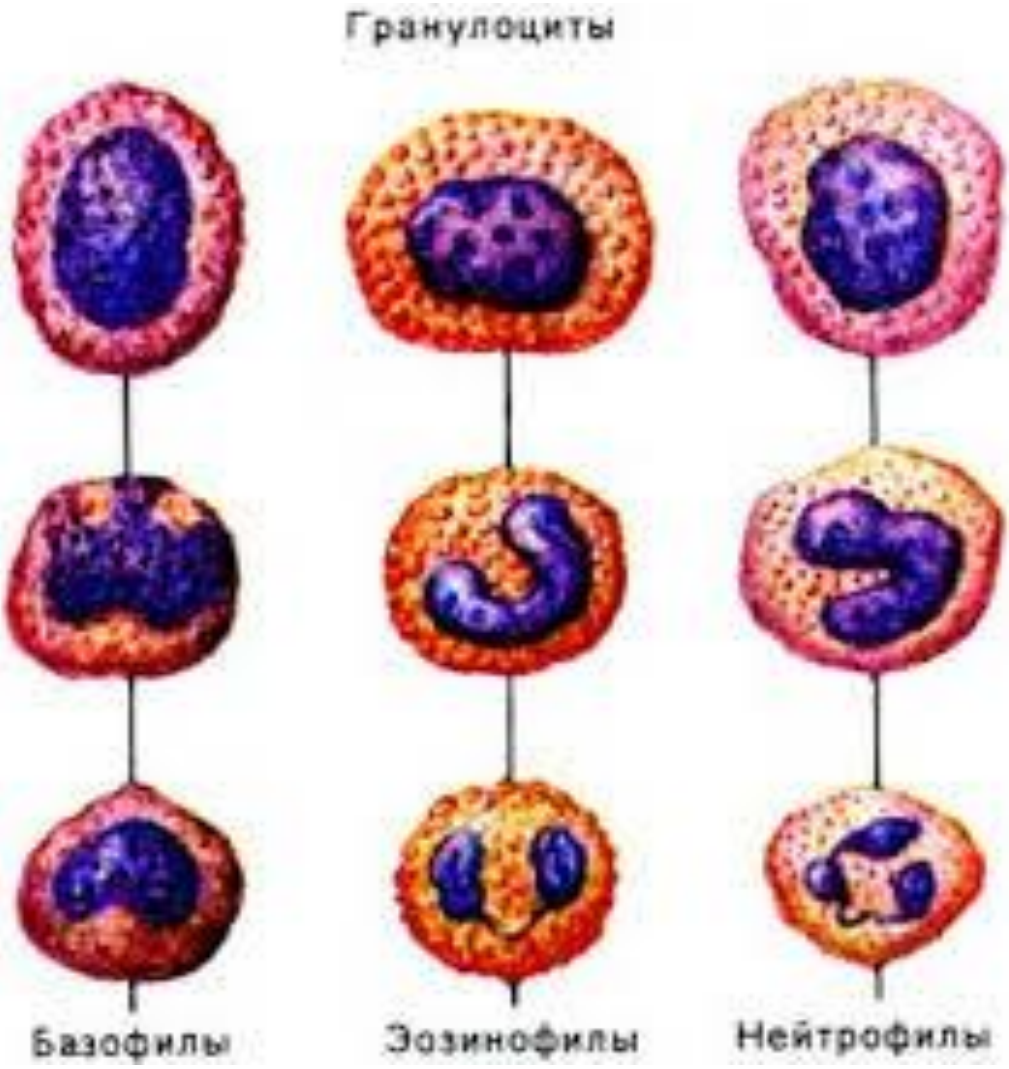


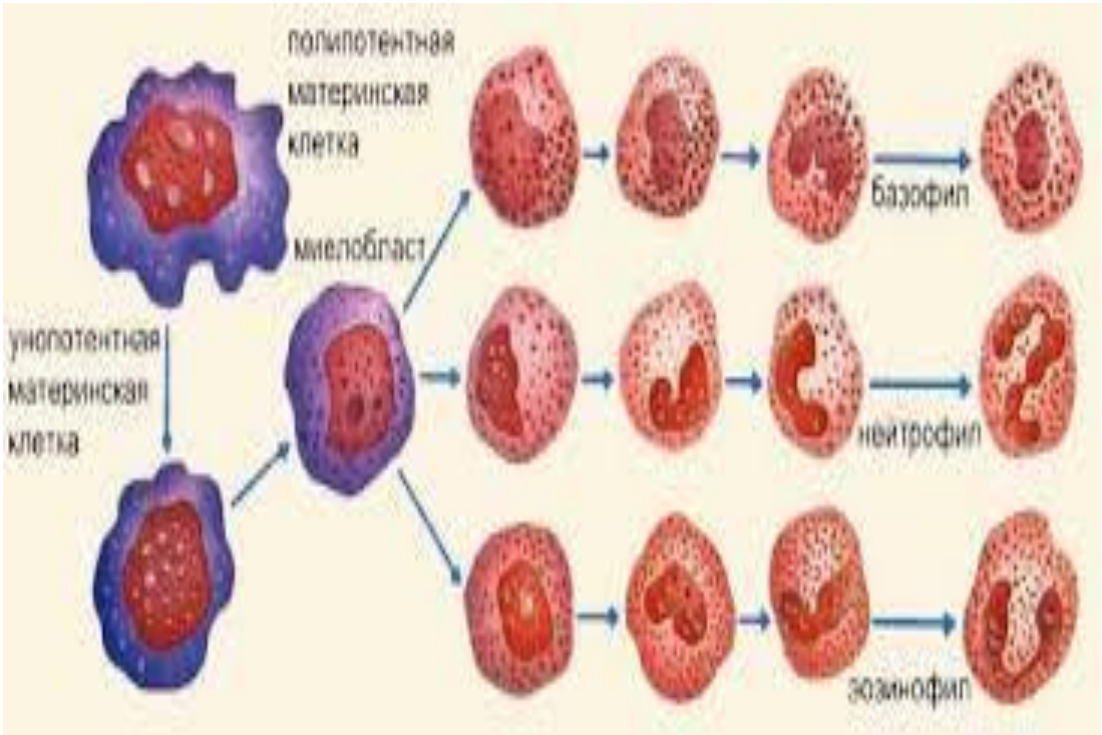


Малый круг:

Начинается от правого желудочка. Сокращениями желудочек выталкивает венозную кровь в лёгочную артерию, откуда она разносится к лёгочным капиллярам. Здесь кровь отдаёт углекислый газ, насыщается кислородом и по лёгочным венам течёт к левому предсердию. Из левого предсердия через левый желудочек кровь вновь поступает в большой круг кровообращения.

MyShared


Гранулоциты
Базофилы
Эозинофилы
Нейтрофилы


полипотентная материнская клетка
миелобласт
унипотентная материнская клетка
базофил
нейтрофил
эозинофил