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

"Physiology of Animals"

Field of knowledge:	400000 - Agriculture and water management
Field of education:	440000 – Veterinary3
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:

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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

2/27

O‘ZBEKISTON RESPUBLIKASI
OLIY VA O‘RTA MAXSUS TA’LIM VAZIRLIGI

SAMARQAND VETERINARIYA MEDITSINASI INSTITUTI

"TASDIQLAYMAN"
Samarqand veterinariya meditsinasi instituti rektori
_____
2022 yil "05" 08

"KELISHILDI"
Oliy va o‘rta maxsus ta’lim vazirligi
_____
2022 yil "12" 08

Ro‘yxatga olindi: №BD-5440100-2.05
2022 yil "17" 08

HAYVONLAR FIZIOLOGIYASI

FAN DASTURI

Bilim sohasi:	400000 – Qishloq va suv xo‘jaligi
Ta’lim sohasi:	440000 – Veterinariya
Ta’lim yo‘nalishlari:	5440100 – Veterinariya meditsinasi (faoliyat turlari bo‘yicha) 5440200 – Veterinariya farmatsevtikasi 5440300 – Veterinariya diagnostikasi va laboratoriya ishlari 5440400 – Veterinariya sanitariya ekspertizasi

Toshkent – 2022

Fan/modul kodi HFM2306	O‘quv yili 2022 - 2023	Semestr 3	ECTS – Kreditlar 6
Fan/modul turi Majburiy	Ta’lim tili O‘zbek-ing		Haftadagi dars soatlari 6

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

2.

I. Fanning mazmuni

Fanni o‘qitishdan maqsad - talabalarda har xil turga mansub bo‘lgan hayvonlar organizmidagi va uning ayrim qismlari: sistemalari, organlari, to‘qimalari, hujayralarida kechayotgan hayotiy jarayonlarni, bu jarayonlarning hayvonlar turiga. jinsiga, zotiga, yashash sharoitiga, mahsuldorligiga va boshqa omillarga qarab, qanday o‘zgarishini o‘rgatish hamda ularni amaliyotda tadbiq etish ko‘nikmasini hosil qilishdan iborat.

Fanning vazifasi - Ushbu maqsadga erishish uchun fan talabalarni nazariy bilimlar, amaliy ko‘nikmalar, hayvonlar organizmida kechayotgan hayotiy jarayonlarga, ularning asoslari va qonuniyatlariga, fiziologik ko‘rsatkichlarni bilish va ularni 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 fiziologiyasi fani va uning rivojlanish tarixi

“Hayvonlar fiziologiyasi” fanning mazmuni, maqsadi, vazifasi va boshqa fanlar bilan o‘zaro bog‘liqligi. Fiziologiya fanining rivojlanish tarixi. Organizm va muhit. Organizmdagi hayotiy jarayonlarning neyrogumoral boshqarilishi.

2-mavzu. Qon sistemasi fiziologiyasi

Qon va limfa haqida tushuncha. Qonning vazifalari, ahamiyati va turli hayvonlar organizmidagi miqdori. Qonning tarkibi va fizik-kimyoviy xossalari.

Qon plazmasining xususiyatlari va uning organizm uchun ahamiyati.

3-mavzu. Yurak fiziologiyasi

Qon aylanishi haqida tushuncha. Qon aylanishini o‘rganish tarixi va bu borada Ibn-Sino ta’limoti. Katta va kichik qon aylanish doiralari.

Hayvonlar yuragining tuzilishi va ishi. Yurak ishining tashqi belgilari: tonlari va turtkisi. Yurak muskullarining xususiyatlari. Yurak ishining neyro-gumoral boshqarilishi.

4-mavzu. Qon tomirlari fiziologiyasi

Qon tomirlari fiziologiyasi. Gemodinamika ta’limoti haqida tushuncha. Qonning tomirlardagi harakati va uni ta’minlovchi omillar, qon bosimi, qonning harakat tezliklari, arteriya va vena pulsi.

Qon tomirlari faoliyatining boshqarilishi. Refleksogen qismlar va ularning

yurak tomirlar faoliyatini boshqarilishidagi ahamiyati.

5-mavzu. Nafas sistemasi fiziologiyasi

Nafasning mohiyati, bosqichlari va mexanizmi. Nafas olish tiplari va tezligi. O‘pkaning ventilyatsiya koeffitsenti. O‘pkada gazlar almashinuvi.

Gazlarning qon bilan tashilishi. Qonning kislorod sig‘imi. Qon bilan to‘qimalar o‘rtasida gazlar almashinuvi. Gazlarning parsial bosimi.

Nafas jarayonining boshqarilishi va ularga turli omillarning ta’siri. Turli sharoitlarda nafas olish xususiyatlari.

6-mavzu. Og‘izda va me’dada ozuqalarninig hazm bo‘lishi

Ozuqa hazmi to‘g‘risida tushuncha. Hazm sistemasining vazifalari va uni o‘rganish usullari. Og‘izda ozuqalarninig hazm bo‘lishi va uning turli hayvonlardagi xususiyatlari. So‘lakning tarkibi, ahamiyati, turli hayvonlardagi miqdori va xususiyatlari. So‘lak ajralishi va uning boshqarilishi. Me’dada ozuqa hazmining umumiy qonuniyatlari. Me’da shirasi, tarkibi, ahamiyati, ajralishi va uning boshqarilishi. Me’da harakati. Me’dadan ichakka ozuqa moddalarning o‘tish qonuniyatlari.

7-mavzu. Kavsh qaytaruvchi hayvonlarda ozuqa hazmining xususiyatlari

Kavsh qaytaruvchi hayvonlarda ozuqa hazmining xususiyatlari. Me’da oldi bo‘lmalarida ozuqalarning hazm bo‘lishi. Mikrofloralarning ahamiyati.

Kavsh qaytaruvchi hayvonlar me’dasining harakati. Shirdonda ozuqalarning hazm bo‘lishi.

8-mavzu. Ichaklarda ozuqalarninig hazm bo‘lishi

Ozuqalarning ingichka ichaklarda hazm bo‘lishi. Ichak shirasi, me’da osti bezi shirasi, o‘t suyuqligining hazm jarayonidagi ahamiyati, ularning ajralishi va boshqarilishi. Ingichka ichaklar harakati (motorikasi).

Ozuqalarning yo‘g‘on ichaklarda hazm bo‘lishi. Yo‘g‘on ichak shirasi, uning xususiyatlari, ajralishi va boshqarilishi. Yo‘g‘on ichaklar harakati (motorikasi).

Tezak (najasning) shakllanishi va chiqarilishi. Hazm sistemasining ekskretor vazifasi.

9-mavzu. Modda va energiya almashinuvi fiziologiyasi

Modda va energiya almashinuvi hayotning mazmuni ekanligi, uni o‘rganish usullari. Oqsillar almashinuvi. To‘la qiymatli va to‘la qiymatsiz oqsillar. Azot balansi va muvozanati. Oqsil minimumi. Oqsillar almashinuvining boshqarilishi.

10-mavzu. Yog‘lar va uglevodlar almashinuvi fiziologiyasi

Yog‘lar va lipoidlar almashinuvi, ularning organizm uchun ahamiyati. Uglevodlar almashinuvi va uning organizm uchun ahamiyati.

Yog‘lar va uglevodlar almashinuvining boshqarilishi. Jigarning moddalar almashinuvidagi ahamiyati.

11-mavzu. Energiya va issiqlik almashinuvi fiziologiyasi

Energiya almashinuvi, mohiyati va ahamiyati. Vositasiz va vositali kalorimetriya. Nafas koeffitsenti.

Turli omillarning moddalar almashinuviga ta’siri.

Issiqlik almashinuvi. Poykilotermli va gomoyotermli hayvonlar. Issiqlik hosil bo‘lishi va uning uzatilishi.

Hayvonlarning tana harorati va uning boshqarilishi-termoregulyatsiya.

12-mavzu. Buyraklar fiziologiyasi

Ayiruv organlari va ularning ahamiyati. Buyraklar fiziologiyasi. Siydik hosil bo‘lishi. Birlamchi va oxirgi siydiklar haqida tushuncha. Pog‘onali va pog‘onasiz moddalar.

Siydik hosil bo‘lishining boshqarilishi. Diurez haqida tushuncha. Siydik ajralishining boshqarilishi.

13-mavzu. Teri fiziologiyasi

Terining tuzilishi, vazifalari va ahamiyati. Ter bezlari. Ter suyuqligining ajralishi va boshqarilishi.

Terining yog‘ bezlari, harorati va muhiti. Teri pigmentatsiyasi. Teridagi mavsumiy o‘zgarishlar. Tullash va uning ahamiyati.

14-mavzu. Markaziy va vegetativ nerv sistemasi fiziologiyasi

Markaziy nerv sistemasi haqida tushuncha. MNSning neyron tuzilishi va reflektor faoliyati. Refleks va ularning turlari. Nerv markazlari va ularning xususiyatlari. Vegetativ nerv sistemasi fiziologiyasi. Simpatik va parasimpatik nerv sistemasi. Nerv sistemasining trofik faoliyati.

15- mavzu. Oliy nerv faoliyati fiziologiyasi

Oliy nerv faoliyati haqida tushuncha. Bosh miya yarim sharlari po‘stlog‘i va uning faoliyatini o‘rganishda I.M.Sechenov va I.P.Pavlovlarning xizmati. Determinizm, analiz va sintez, tuzilish tamoyillari. Shartli va shartsiz reflekslar. Shartli reflekslarning biologik ahamiyati. Dinamik stereotip. Nerv sistemasining tiplari. I.P.Pavlov talimotining chorvachilikdagi ahamiyati.

III. Amaliy mashg‘ulotlar bo‘yicha ko‘rsatma va tavsiyalar

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

1. Hayvonlar fiziologiyasi fanining tajribalari.
2. Qonning shaklli elementlarini sanash uslubi.
3. Qon surtmasini tayyorlash va leykotsitar formulani aniqlash.
4. Yurak va tomirlar faoliyatini o‘rganish usullari.
5. Ekstrasistola va kompensator pauza hosil qilish.
6. Yurak avtomatiyasi.
7. Yurak faoliyatining reflektor boshqarilishi.
8. Qon bosimi va uni aniqlash usullari.
9. Qon tomirlari faoliyatining boshqarilishi.
10. Qonning tomirlardagi harakatini kuzatish (kapillyaroskopiya)
11. O‘pka faoliyatini o‘rganish usullari. Nafas olish va nafas chiqarish mexanizmi.
12. O‘pkaning tiriklik havo sig‘imini o‘lchash.

13. Ichaklar avtomatiyasi.
14. Hayvonlarda tana haroratini o‘lchash.
15. Nerv - muskul preparatini tayyorlash.

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 texnologiyalarini qo‘llanilishi maqsadga muvofiq.

III.II. Laboratoriya mashg‘ulotlar uchun quyidagi mavzular tavsiya etiladi:

1. Qon plazmasi va qon zardobini ajratib olish.
2. Eritrotsitlar va leykotsitlar sonini sanash.
3. Gemoglobin miqdorini aniqlash.
4. Eritrotsitlarning cho‘kish tezligini aniqlash.
5. Gemoliz. Eritrotsitlarning osmotik rezistentligini aniqlash.
6. Qonning ivish tezligini aniqlash.
7. Qon guruhlarini aniqlash.
8. Yurak faoliyatini gumoral boshqarilishi.
9. So‘lak fermentlari ta’sirida kraxmalning gidrolizlanishi.
10. Katta qorindagi mikroorganizmlar miqdorini aniqlash.
11. Me’da shirasining ahamiyati va uning kislotalik darajasini aniqlash.
12. Ichaklarning bir tomonlama o‘tkazuvchanlik xususiyati.
13. Siydikning tarkibi va xususiyatlarini o‘rganish.
14. Muskullarning yakka va tetonik qisqarishi. Fiziologik tinch va faoliyat davridagi biotoklarni aniqlash.
15. Refleks yoyi, vaqti va maydonini aniqlash.

Laboratoriya mashg‘ulotlarini 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 auditoriyalarda bir guruhni ikkiga bo‘lib o‘tkazilishi lozim.
Laboratoriya mashg‘ulotlarida talabalar turli qishloq xo‘jalik va laboratoriya hayvonlari organizmida kechayotgan hayotiy jarayonlarning hayvonlar turiga, jinsiga, zotiga, yashash sharoitiga, mahsuldorligiga va boshqa omillarga qarab, qanday o‘zgarishini 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. Immun sistema fiziologiyasi.
2. Parrandalarning yurak va qon tomirlari fiziologiyasi.

3. Parrandalarda nafas olishning o‘ziga xos xususiyatlari.
4. Ot va cho‘chqalar me’dasida ozuqa hazmining xususiyatlari.
5. Parrandalarda ozuqa hazmining o‘ziga xos xususiyatlari.
6. Suv almashinuvining fiziologiyasi.
7. Makro va mikro elementlar fiziologiyasi.
8. Suvda va yog‘da eruvchi vitaminlar fiziologiyasi.
9. Parrandalarda siydik ajralishi.
10. Bug‘ozlik va tug‘ish fiziologiyasi.
11. Homilada qon aylanishi.
12. Parrandalarning ko‘payish fiziologiyasi.
13. Mashina bilan sut sog‘ishning fiziologik asoslari.
14. Hayvonlarni ozuqlantirish va parvarish qilishning sut miqdori hamda tarkibiga ta’siri.
15. Ichki sekretsiya bezlari fiziologiyasi.
16. Bosh va orqa miya, ularning vazifalari.
17. Uyqu va gipnoz.
18. Signal sistemalar.
19. Analizatorlar fiziologiyasi.

Mustaqil o‘zlashtiriladigan mavzular bo‘yicha talabalar tomonidan fan bo‘yicha internet ma’lumotlarini to‘plash, ularni o‘rganish, o‘quv adabiyotlari yordamida referat tayyorlash va uni taqdimot qilish tavsiya etiladi.

V. Malakaviy amaliyotni o‘tkazish tartibi

“Hayvonlar fiziologiyasi” fanidan malakaviy amaliyot Samarqand veterinariya meditsinasi institutining vivariumi va o‘quv-tajriba xo‘jaligida, Samarqand viloyati hayvon kasalliklari tashxisi va ozuq-ovqat mahsulotlari xavfsizligi davlat markazi va chorvachilik fermer xo‘jaliklarida o‘tkaziladi.

Malakaviy amaliyotni o‘tkazish bo‘yicha mavzular:

1. Qishloq xo‘jalik va laboratoriya hayvonlaridan qon olish usullari.
2. Sitrat, oksalat, fibrinsizlantirilgan va gemolizlangan qon hosil qilish.
3. Qishloq xo‘jalik va laboratoriya hayvonlarida yurak chastotasini aniqlash.
4. Qishloq xo‘jalik va laboratoriya hayvonlarida nafas chastotasini aniqlash.
5. Qishloq xo‘jalik va laboratoriya hayvonlarida tana haroratini aniqlash.
6. Hayvonlarning xulq-atvorlari va nerv sistemasi tiplarini o‘rganish.

Malakaviy amaliyot bo‘yicha bir kunga bitta amaliy mashg‘ulot rejalashtirilgan bo‘lib, u 6 soatga mo‘ljallangan. Har bir guruh talabalari 3-4 tadan kichik guruhlarga bo‘linadi va ketma-ket, navbati bilan kafedra o‘qituvchilarning rahbarligida berilgan amaliy mashg‘ulot vazifalarini mustaqil bajarishadi.

Olingan natijalarni daftarga qayd etib, me’yordagi fiziologik ko‘rsatkichlar bilan taqqoslab, tahlil qiladi va xulosa chiqaradi.

Malakaviy amaliyotni o‘tkazish vaqtida har bir talaba kafedra o‘qituvchilari tomonidan yozilgan va chop etilgan uslubiy qo‘llanmalaridan foydalanishlari shart.

Ana shunda malakaviy amaliyot mashg‘ulotlari bo‘yicha qo‘yilgan vazifalarni har bir talaba to‘liq bajarishga erishadi.

3	**VI. Fan o‘qitilishining natijalari (shakllanadigan kompetensiyalar).** Fanni o‘zlashtirish natijasida talaba: - hayvon organizmida kechadigan fiziologik jarayonlarning yaxlitligi va gomeostaz, hayvonlarning qoni, plazmasi va qon zardobi, hayvonlar organizmida qon aylanishi, hayvonlarning nafas olishi va hazm faoliyati, moddalar va energiya almashinuvi, ayiruv organlari va teri faoliyati, hayvonlarning ko‘payishi va laktatsiyasi, endokrin bezlar va gormonlar, qo‘zg‘aluvchan to‘qimalar, markaziy va oliy nerv sistemalari faoliyati ***haqida tasavvurga ega bo‘lishi;*** -hayvonlar organizmida kechadigan hayotiy jarayonlarni, qon sistemasi fiziologiyasini, qon va limfa aylanishi sistemasi fiziologiyasini, nafas sistemasi fiziologiyasini, hazm sistemasi fiziologiyasini, moddalar va energiya almashinuvi fiziologiyasini, umumiy va asosiy almashinuv fiziologiyasini, issiqlik almashinuvi va uning boshqarilishini, ayiruv organlari sistemasi fiziologiyasini, teri fiziologiyasini, endokrin - ichki sekretsiya bezlari sistemasi fiziologiyasini, garmonlar va ularning ta’sirini, ko‘payish fiziologiyasini, laktatsiya fiziologiyasini, qo‘zg‘aluvchan to‘qimalar (muskul, nerv, bez) fiziologiyasini, markaziy va periferik nerv sistemasi fiziologiyasini, oliy nerv faoliyati va uni o‘rganish usullarini, qo‘zg‘alish va tormozlanishlarni, etologiya – hayvonlar xulq-atvorini, analizatorlar - sezgi organlari fiziologiyasini ***bilishi va ulardan amaliyotda foydalana olishi;*** -hayvonlar organizmidagi fiziologik ko‘rsatkichlarni aniqlash, hayvonlarda o‘tkir va surunkali tajribalar o‘tkazish hamda o‘tkir va surunkali tajribalarni modellashtirish, klinik va laboratoriya tekshiruvlarini o‘tkazish ***ko‘nikmalariga ega bo‘lishi;*** - hayvonlar va parrandalardan qon olish, hayvonlar va parrandalar qonidan plazma va qon zardobini ajratish; gemotologik va serologik tekshirishlarni tahlil qilish va baholash ***malakalariga ega bo‘lishi kerak.***
4.	**VII. Ta’lim texnologiyalari va metodlari:** • ma’ruzalar; • interfaol keys-stadilar; • seminarlar (mantiqiy fikrlash, tezkor savol-javoblar); • guruhlarda ishlash; • taqdimotlarni qilish; • individual loyihalar; • jamoa bo‘lib ishlash va himoya qilish uchun loyihalar.
5.	VIII. 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.

Asosiy adabiyotlar

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Qo'shimcha adabiyotlar

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7.	Fan dasturi Oliy va o'rta maxsus professional ta'lim yo'nalishlari bo'yicha O'quv-uslubiy birlashmalar faoliyatini Muvofiqlashtiruvchi Kengashning 2022 yil "17" 08 dagi 3 -sonli bayonnomasi bilan ma'qullangan.

Oliy ta'limning 400000-Qishloq va suv xo'jaligi bilim sohasi va 440000 – Veterinariya ta'lim sohasining 5440100 – Veterinariya meditsinasi (faoliyat turlari bo'yicha), 5440200 – Veterinariya farmatsevtikasi, 5440300 – Veterinariya diagnostikasi va laboratoriya ishlari, 5440400 – Veterinariya sanitariya ekspertizasi bakalavriat ta'lim yo'nalishlari uchun dotsentlar R.F.Ro'ziqulov va D.E.Eshimovlar tomonidan tayyorlangan "Hayvonlar fiziologiyasi" fan dasturiga

TAQRIZ

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

Hayvonlar fiziologiyasi – har xil turga mansub bo'lgan hayvonlar organizmidagi va uning ayrim qismlari: sistemalari, organlari, to'qimalari, hujayralarida kechayotgan hayotiy jarayonlarni, o'rganadigan, bu jarayonlarning hayvonlar turiga, jinsiga, zotiga, yashash sharoitiga, mahsuldorligiga va boshqa omillarga qarab, qanday o'zgarishini tekshiradigan fandir.

Fanni o'rganish uchun ma'ruza, amaliy mashg'ulotlar, laboratoriya darslari va mustaqil ishlash uchun yetarli darajada soatlar ajratilgan bo'lib, veterinariya vrachlari, zootexniya va kasb ta'limi bo'yicha mutaxassislikka oid tafakkur va dunyoqarashni shakllantirishda katta ahamiyatga egadir.

Dasturning "Kirish" qismida: fanning dolzarbligi va oliy kasbiy ta'limdagi o'rni, fanning maqsadi va vazifalari; fan bo'yicha talabalarning bilimiga, o'quv va ko'nikmalariga qo'yiladigan talablar; o'qitish sistemasi va uslubiy ko'rsatmalar berilgan.

Dasturning "Asosiy qismi"da modullar va mavzular asosida fanning mazmuni va ma'ruza mashg'ulotlari, amaliy va laboratoriya mashg'ulotlari bo'yicha ko'rsatma va tavsiyalar, mustaqil ta'lim va mustaqil ishlar uchun tavsiya etilgan mavzular malakaviy amaliyotni o'tkazish bo'yicha ko'rsatma va tavsiyalar alohida-alohida berilgan.

Asosiy, xorijiy va qo'shimcha adabiyotlar hamda axborot manbalari ham alohida-alohida berilgan.

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

Shuning uchun "Hayvonlar fiziologiyasi" 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.

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

Oliy ta'limning 400000-Qishloq va suv xo'jaligi bilim sohasi va 440000 – Veterinariya ta'lim sohasining 5440100 – Veterinariya meditsinasi (faoliyat turlari bo'yicha), 5440200 – Veterinariya farmatsevtikasi, 5440300 – Veterinariya diagnostikasi va laboratoriya ishlari, 5440400 – Veterinariya sanitariya ekspertizasi bakalavriat ta'lim yo'nalishlari uchun dotsentlar R.F.Ro'ziqulov va D.E.Eshimovlar tomonidan tayyorlangan "Hayvonlar fiziologiyasi" fan dasturiga

TAQRIZ

"Ta'lim" va "Kadrlar tayyorlash milliy dasturi" to'g'risidagi qonunlarga mos ravishda qishloq xo'jaligini raqobatbardosh, malakali mutaxassislar bilan ta'minlash borasida bakalavriat ta'lim yo'nalishlari bo'yicha ta'lim olayotgan talabalar "Hayvonlar fiziologiyasi" fanidan sog'lom hayvonlar organizmida kechayotgan hayotiy jarayonlar, ularning asoslari, qonuniyatlarini o'zlashtirib, fiziologik ko'rsatkichlarni bilishi va ularni aniqlashni, chorvachilikda hayvonlar mahsuldorligini oshirish hamda iqtisodiy samaradorligini yuksaltirishning ilmiy va amaliy qonuniyatlarini yaratishni o'rganishdan iboratdir.

Dasturning "Kirish" qismida: fanning dolzarbligi va oliy kasbiy ta'limdagi o'rni, fanning maqsadi va vazifalari; fan bo'yicha talabalarning bilimiga, o'quv va ko'nikmalariga qo'yiladigan talablar; o'qitish sistemasi va uslubiy ko'rsatmalar berilgan.

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Dasturda dars uchun kerak bo'ladigan didaktik vositalar va o'qitish uslublari o'z aksini topgan.

Umuman olganda "Hayvonlar fiziologiyasi" fanidan yozib tayyorlangan dastur hozirgi davr va Malaka talablariga javob beradi deb hisoblayman va uni tasdiqlash hamda o'quv jarayonida qo'llash uchun tavsiya etaman.

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

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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

"I APPROVE"
Vice rector for academic affairs
Amurodov AA
"___" _____ 2022

ANIMAL PHYSIOLOGY

WORKING CURRICULUM OF SCIENCE

Field of knowledge: 400000 - Agriculture and Water Management

Field of education: 440000 - Veterinary

Areas of study: 5440100 - Veterinary medicine (by type 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 the subject:
Code of the subject: **HFM 2306**
Name of the subject: **Animal physiology**
Semester / year: **3rd semester / 2021-2022 year of study**
Department: 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 - Students learn about the vital processes in the body of animals of different species and their parts: systems, organs, tissues, cells, the type of these processes in animals. to teach how they change depending on gender, breed, living conditions, productivity and other factors, and to develop the ability to apply them in practice.

II. Science function - To achieve this goal, science provides students with theoretical knowledge, practical skills, a methodological approach to the life processes in animals, their foundations and laws, physiological indicators and their definition, as well as the formation of a scientific worldview.

The structure of science: plan of lectures

Topic: THE SCIENCE OF ANIMAL PHYSIOLOGY AND HISTORY OF ITS DEVELOPMENT

Plan:

1.1. Animal Physiology ”the content, purpose, tasks and interrelation of science with other sciences

1.2. Animal Physiology ”the content, purpose, tasks and interrelation of science with other sciences

1.3. Organism and environment. Neurohumoral control of vital processes in the body.

Basic phrases:

Physiology, organ, tissue, cell, general physiology, specific physiology, evolutionary physiology, ecological physiology, age physiology, human physiology, plant physiology, microbial physiology, animal physiology, organism and environment, metabolism, susceptibility, excitability, growth, development, reproduction, heredity, nerve, humoral, reflex, receptors, homeostasis, Hippocrates, Aristotle, Claudius Galen, Abu Ali ibn Sino, A. Vezali, V. Garvey, A. Levenguk, Rene-Descartes, F.Majandi, I. Müller, Purkine, CH.Darvin, AMFilomafitskiy, VABasov, Zion, IMSechenov, VVPashutin, NEVvedenskiy, IPPavlov, LAOrbeli, PKAnoxin,

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www:zooveterinariya. @ mail.ru www. zootechniya.ru

1. The science, purpose, function and

connection with other sciences.

Physiology - one of the biological sciences, which studies the vital processes that take place in the whole organism and in some parts of it: systems, organs, tissues, cells, the laws underlying them, the connections between these laws, the processes closely related to the external environment It is a science that examines the state of affairs.

Physiology consists of two words, Latin physis - nature, logos- doctrine.

Physiology is divided into two interrelated parts:

1. General physiology. 2. Private physiology.

Of the science of physiology *in the general part* common to all cells, tissues, organs, and systems in the body: vital processes such as metabolism, exposure, reproduction, growth, and development are studied.

Of the science of physiology *in the private part*and the vital processes specific to each organ and system in the body are studied. For example, the activity of organs such as the heart, liver, kidneys, blood, blood circulation, respiration, digestion, digestion and other systems are studied.

This means that as many organs and systems as there are in the body, so is the specific part of physiology.

There are several branches of physiology:

1. Evolutionary physiology. 2. Ecological physiology. 3. Physiology of age.
4. Human physiology. 5. Plant physiology. 6. Physiology of microorganisms. 7. Physiology of fish. 8. Physiology of birds.
9. Physiology of bees. 10. Animal physiology.

Evolutionary physiology studies the vital processes that take place during the historical development of the organism.

Ecological physiology learns the activity of the organism and the life processes in it in different environmental conditions.

Ysoup physiology studies the physiological processes that take place at different stages of ontogenetic development of the organism.

Human physiology studies the life processes that take place in the human body and the laws that underlie them.

Plant physiology studies the vital processes that take place in the organism of different plants and the laws that underlie them.

Physiology of microorganisms studies the vital processes that take place in the body of various microorganisms and the laws that underlie them.

Physiology of fish studies the vital processes that take place in the body of various fish and the laws that underlie them.

Physiology of birds studies the life processes that take place in the bodies of various birds and the laws that underlie them.

Physiology of bees studies the life processes that take place in the body of bees and the laws that underlie them.

Animal physiology is a branch of physiology that studies the vital processes that take place in the body of pets of different species, examining how these processes

change depending on the type, age, sex, breed, nutrition, living conditions, productivity, and other factors. fandir.

Animal physiology the purpose of science - Students learn about the life processes in the body and its parts: systems, organs, tissues, cells of animals of different species, depending on the type, sex, breed, living conditions, productivity and other factors of these processes. to explain the change, to form professional thinking and worldview, as well as knowledge, skills and competencies in the field.

Animal physiology The task of science - to students mastering the basics and laws of life processes in the body of animals, learning to know and identify physiological indicators, increase the productivity of animals in animal husbandry and create scientific and practical laws to increase economic efficiency.

The impact of the results of socio-economic reforms in the country on the prospects of veterinary and animal husbandry, The development of livestock farms, increasing the number of healthy animals and poultry, as well as quality livestock products (meat, milk, fat and eggs) poses new challenges for physiologists.

Cfor veterinary and animal husbandry specialists need to know the physiological parameters and physiological processes taking place in the body in order to fertilize, breed, feed, care for animals, increase productivity, prevent and treat diseases.

Thus, the subject of "Animal Physiology" is of great importance in the study of veterinary and animal husbandry, and serves as both a theoretical and practical basis.

2. Organism and environment.

First of all, it is necessary to pay special attention to the meaning of the two words organism and environment.

An organism is a highly developed organic universe consisting of a collection of living cells, tissues, organs and systems, capable of self-regulation, independent living, development, reproduction, and responsive to the effects of the external environment. is an integrated system.

The environment is the cause that determines the external conditions of an organism.

Every organism can survive only if it is in constant contact with the external environment. This communication takes place due to metabolism. This is because oxygen and nutrients enter the body from the external environment, and carbon dioxide and other unwanted substances are released into the external environment.

Depending on the metabolism, the following general features of the organism appear:

1. Sensitivity. 2. Mobility.

3. Growth. 4. Development. 5. Reproduction. 6. Heredity.

Susceptibility is a characteristic feature of all living things, all cells and tissues of the organism, and is characterized by changes in metabolism.

Excitability is a characteristic feature of nerve, muscle and glandular tissue, characterized by gross changes in metabolism and the appearance of tissue-specific symptoms. For example: nerve tissue generates an impulse and transmits it along nerve fibers to organs and systems; glandular tissue secretes fluid; the muscle contracts.

Growth is the process by which an organism's live weight continues to increase.

Development is the process by which an organism is fully formed from a zygote.

Reproduction refers to the process by which an organism leaves offspring and ensures the stability of a species.

Heredity refers to the ability of an organism to pass on its traits and characteristics to the next generation.

So, the only event that determines the vitality of an organism is metabolism. Its basis is nutrition. With the cessation of metabolism, the life of the organism dies.

3. Neurohumoral pathways of vital processes in the body
be managed with.

All processes in the body are continuously controlled by 2 systems:

Nervous system.

Humoral system.

It is controlled by the reflex pathway through the nervous system.

It is administered through the humoral path in the presence of biologically active substances (hormones, vitamins, minerals, etc.) in the blood, lymph and other fluids.

Latin humor - means liquid.

First of all, the humoral system appeared in lower animals. Later, due to the development of the animal kingdom, the development and complexity of the vital processes that take place in them, the nervous system based on the humoral system emerged. As a result, the processes that take place in animals where the nervous system is formed remain controlled by the neuro-humoral pathway.

Although the nervous system is formed later than the humoral system, it takes the lead in controlling the processes in the body and also regulates the activity of the humoral system. But the activities of these two systems are inseparable.

Despite any change in the external environment, the organism always maintains and maintains its internal environment, vital functions to a certain extent.

The ability of an organism to constantly maintain its internal environment is called homeostasis. This doctrine was first founded in 1878 by the French scientist Claude Bernard. This doctrine was later developed by Canadian scientist Walter Kennon, who coined the term homeostasis.

Blood, lymph, osmotic pressure of interstitial fluids, concentration of hydrogen ions, amount of protein, sugar, anions and cations in tissues, body temperature, etc. are important vital indicators of the organism. These figures are maintained throughout life. But this does not mean that the indicators are variable, absolute. They vary to a very small extent depending on the general condition of the organism. Only when these indicators are stable, the conditions are created for the proper conduct of vital processes in the tissues and cells of the body.

The activity of the kidneys, lungs, skin, digestive organs, their regulation by the nervous and humoral pathways play an important role in the uniformity of vital indicators.

4. History of the development of physiology.

Russian physiologist academician IPPavlov: *"Only by knowing the ancient, past state of science, that is, its achievements, can we know the present state."* he said.

The purpose of knowing the history of science: to study the origin, creation, period and conditions of science; to get acquainted with the methods, ideas and theories of science; to get acquainted with the works of people (scientists) who created science and to arouse interest; to study the mistakes and shortcomings in the development of science; to explore new research being conducted to address these errors and omissions; is the study of terms and innovations in science. Thus, knowledge of the history of science expands the thinking and worldview of students in the field and allows them to form knowledge, skills and competencies in the field.

Topic: Physiology of the circulatory system.

Plan:

1 The concept of blood and lymph.
2.Functions, importance and amount of blood in the body of various animals.
3. Composition and physicochemical properties of blood
4.Properties of blood plasma and its importance for the body

Every organism, regardless of any changes in the external environment, always maintains and maintains its internal environment and vital functions to a certain extent. The ability of an organism to constantly maintain its internal environment is called homeostasis. This doctrine was introduced to science in 1878 by the French scientist Claude Bernard. This doctrine is later developed by Canadian scholar Colter Kennon, who explains the essence of the content.

The internal environment of the body consists of the following 3 fluids.

1.Blood 2. Lymph 3. Tissue interstitial fluid

Blood is one of the most important tissues of the body. Blood is a red, short-tasting, liquid connective tissue.

Lymph is a colorless fluid that travels through the lymphatic vessels.

Interstitial fluid is the fluid that fills the intercellular spaces of all tissues in the body. These three fluids are involved in the metabolism of intermediates by washing the cells in the tissues. In all tissues and cells of the body, their composition and properties are relatively constant, they can live normally in the environment of these fluids.

Tissue interstitial fluid is formed in the smallest blood vessels of the blood and acts as an environment that directly nourishes the body.

Because cells get the substances they need for nutrition and growth from the tissue interstitial fluid. It removes unnecessary substances from the metabolism. The corresponding portion of these substances is absorbed from the tissue interstitial fluid into the venous portion of the capillaries. The rest is poured into the lymphatic vessels. It is then enriched with lymphocytes in the lymph nodes and turned into lymph fluid.

Lymph differs slightly in its composition and properties, even though it is close to the blood plasma and tissue space. Lymph plays an important role in the formation of

milk, the exchange of water and salts, the elimination of various unwanted substances from the body and the protective activity of the body.

The importance of blood is determined by the following functions it performs in the body.

Transportation function

a) the task of transporting gases

b) the function of transporting nutrients

c) the function of transporting metabolites to the excretory organs

2. The function of thermoregulation - the state of body temperature control

3. The function of protection

4. Correlation - management function

5. Ensuring homeostasis

If we take the blood in the body as 100%, 54% of it is constantly on the move. The remaining 46 percent is stored in the following depot bodies. 20% in the liver, 16% in the spleen, 10% in the skin.

About 55% of the blood in the body is in the veins, 20% in the pulmonary veins, 15% in the arteries, 5% in the capillaries, and 5% in the heart.

The amount of blood relative to the body weight of farm animals:

8% in cattle 9.8% in horses 8.1% in sheep and goats 4.6% in pigs and 7% in humans.

Blood consists of two parts. Plasma and shaped elements.

1. Blood plasma is 55-60%.

Plasma content: 90-92% water, 0.8-1% inorganic matter, 0.7-0.8% Organic matter, 2.1-4% Albumins, 2-4% Globulins, Fibrinogen 0.2-0, Will be in the amount of 4%.

2. The form elements of the blood make up 40-45% of them

a) erythrocytes - red blood cells

b) leukocytes - white blood cells

c) platelets - blood platelets.

These 3 cells have specific properties, functions, importance, and quantity.

Physicochemical properties of blood. The color of the blood is red

a) light red blood - arterial blood

b) dark red blood-venous blood

2. The taste of blood is sour

3. Blood viscosity -4.0-6.0 (taken in relation to water)

4. Specific gravity of blood-1,050-1,060

5. The osmotic pressure of the blood forms salts and is normally equal to 7 atmospheres.

6. Oncotic pressure in the blood - produces proteins in it and is normally equal to 25-30 mm Hg.

7. The environment of the blood is weakly alkaline and differs little from one animal to another. pH 7.0-7.2

a) pH-7.42 in arterial blood

b) Venous blood has a pH of 7.35.

8. Blood buffering - plays an important role in maintaining a normal blood environment.

a) Hemoglobin buffer system - Hemoglobin buffer makes up 75% of all buffers.

b) Protein buffer system c) Phosphate buffer system d) Carbonate buffer system

1. Blood cells unite in the organs and blood system where they are formed and broken down. These include the red marrow, spleen, liver and lymph nodes. For the normal functioning of the organs, they must be constantly supplied with blood. Irreversible changes result from a very short cessation of blood circulation (a few minutes to the brain). It is characterized by the fact that the blood performs important functions for the life of the organism.

Topic: Physiology of the heart.

Plan:

1.The concept of blood circulation.
2. The history of the study of blood circulation and the teachings of Ibn Sina in this regard.
3 Large and small circulatory circles The structure and function of the animal heart
4. The structure and function of the animal heart in large and small circulatory circles

Basic expressions.

Circulatory system, large and small circulatory system, heart, aorta, arteries, veins, capillaries, Aristotle, Hippocrates, K. Galen, Abu Ali ibn Sino, V. Garvey, Malpighi, Weber, I.Sion, heart, epicardium, pericardium, myocardium, endocardium, ventricle, ventricle, diaphragmatic valve, crescent valve, spinal cord, systole, diastole, pause, cardiac cycle, heart tones, systolic and diastolic tones, heart rate, systolic and minute volume of the heart , excitability, conduction, automation, refractoriness, bioelectric current, nerve node, Kiss - Fleka node, Ashof - Commodity node, Giss tuft and legs, Purkinje fibers, blockade, myogenic theory, neurogenic theory, extrasystole, compensator pause, ECG.

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3. V.F.Lisov, V.I.Maksimov. Osnovy fiziologii i etologii jivotnyx. Moscow, Kolos, 2004.

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5. Websites:

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www:zooveterinariya. @ mail.ru www. zootechniya.ru

1. General concept of blood circulation. Large and small circulatory circles.

In the body, blood performs its various functions only when it is in constant motion. Due to the constant work of the heart, there is a continuous movement along the arteries.

Circulation refers to the constant movement of blood in special tubes, that is, in the blood vessels, without stopping.

To the circulatory system:

1. Yurak. 2. Large circulatory area. 3. Small circulatory area.

4. Blood vessels: include aorta, arteries, veins and capillaries.

Arterial blood vessels to all blood vessels from the heart, The blood vessels that flow to the heart are called venous blood vessels, and the blood vessels that connect them to each other are called capillaries.

There are 2 circulatory systems in mammals and poultry:

1. Large circulatory area. 2. Small circulatory area.

The great circle of circulation begins with the outflow of the aortic artery from the left ventricle of the heart and ends with the inflow of the anterior and posterior vena cava into the right ventricle of the heart.

The small circulatory system begins with the outflow of the pulmonary artery from the right ventricle of the heart, travels to the lungs, gas exchange takes place in the lungs, and ends with the inflow of the pulmonary vein into the left ventricle of the heart. Blood from the right ventricle of the heart to the pulmonary artery is venous blood, and blood from the left ventricle of the heart is arterial blood. Each circulatory system is made up of specific arteries, veins, and capillaries. This means that both circulatory systems begin in the heart and end in the heart.

2. The history of the circulatory system and the teachings of Ibn Sina in this regard.

The study of the physiology of the cardiovascular system has its own historical development. Preliminary data on the study of the activity of the cardiovascular system were described by Aristotle and Hippocrates. Hippocrates discovered that there is a heart and a place in the body. K. Galen had a misconception about the circulation, that there is air without blood in the arteries, that the liver moves the blood, not the heart, that there is an oval hole between the chambers of the heart. The famous orientalist Abu Ali Ibn Sina (980-1037) expressed his opinion on the dependence of the pulse on the nature of the organism and the activity of the nervous system, as well as the small

circulatory system. In 1628, W. Garvey identified the major and minor circulatory systems and the way blood circulates through the lungs. In 1661, Malpighi identified the capillary vascular system and thoroughly studied the major and minor circulatory systems. In 1885, the Weber brothers studied the effects of the stray nerve, the IFSion sympathetic nerve, on heart function.

3. The structure and function of the animal heart

The heart is an integral hollow organ located inside the chest and is an organism
0.6 - 1% of live weight.

The following types of heart are distinguished:

- 2-chambered heart - in fish; - 3-chambered heart - in amphibians;
- 4-chambered heart - occurs in mammals and birds.

The structure of the animal heart. The heart of highly developed warm-blooded animals and birds consists of 4 chambers: 2 compartments (right and left) and 2 ventricles (right and left).

Ythe left and right parts of the heart are separated by a barrier, and there are atrioventricular openings provided with stratified valves between the heart chambers and the ventricles. There is a 2-layer valve between the left ventricle and the left ventricle, and a 3-layer valve between the right ventricle and the right ventricle, which opens to the side of the ventricles. The threads that hold the ventricles do not allow the valves to open on the side of the heart chambers. Where the aorta and pulmonary artery begin, there are crescent-shaped valves reminiscent of the shape of pockets, which open into the blood vessels. At the junction of the anterior and posterior vena cava to the right ventricle there is a sphincteric structure consisting of annular muscles - the ventricles of the heart.

YThe wall of the heart consists of three layers:

1. The epicardium is the outer layer.
2. The myocardium is the middle muscle layer.
3. The endocardium is the inner layer.

Above the heart is the "shirt", ie the heart sac - the pericardium. Between the pericardium and the epicardium is a fluid with a specific property. This fluid rubs against the heart as it works and keeps it from overheating. The heart muscle is made up of transverse sphincter muscles and is connected to each other by protoplasmic bridges.

Ythe work of the heart. The heart is constantly working at a steady pace. As a result, blood moves throughout the body in only one direction: from the heart chambers to the ventricles, from the ventricles to the blood vessels, and from them to the chambers.

This activity is characterized by contraction of the heart muscle - systole, dilation of diastole and relaxation - pause.

YThe heart works alternately in two phases:

Phase 1: compartment systole and diastole.

Phase 2: ventricular systole and diastole.

Then the muscles of the pelvis and ventricles relax for a while.

Ythe work of the heart begins with the contraction-systole of both heart chambers. The right ventricle contracts 0.01 seconds before the left ventricle. During the operation

of the compartments, the pressure inside them is equal to 30-70 mm of mercury. The ventricles are in an diastole state, and blood collects in the ventricles because the stratified valves between the ventricles and the ventricles are open. During the period when the stratum valves close and ventricular systole begins, the pressure in the ventricles is not enough to open the crescentic valves, and the ventricular muscle is forced to contract for 0.03-0.06 seconds. begins and the tension phase of the ventricles occurs. As a result, the pressure inside the ventricles increases to 130-150 mm Hg, and the crescent valves open and are pumped into the blood vessels. From this point on, the crescent-shaped valves close and the layered valves open. During total diastole, blood continues to flow and fills the ventricles and compartments.

Ya working cycle of the heart is said to be the period from one systole of the heart chambers to another. If we take one work cycle of the heart as 100%, 10% of it is related to the work of the ventricles, 30% to the work of the ventricles, and 60% to the pause, that is, to rest. That is why the heart works in a rhythm without stopping for a lifetime. Cardiogram: 1. The work of the heart chambers. 2. The work of the ventricles of the heart. 3. Pause - rest.

The following factors affect the activity of the heart in different ways:

1. Type of animal. 2. The breed of animals. 3. Gender of animals.
4. Age of animals. 5. Animal productivity. 6. The body of an animal.7. Condition of the organism. 8. Time of day. 9. Ambient temperature.
10. Physical work and others.

In particular, the functioning of the heart depends on the speed or slowness of the metabolism in the body. This means that the work of the heart can change under the influence of various factors. Arrhythmia is a change in the heart rhythm. Tachycardia is an increase in heart rate. Bradycardia is a slowing of the heart.

The number and frequency of heartbeats vary in different animals and birds.

Heart rate, minute and systolic volume

Animals and birds type	Heart frequency (In 1 minute)	The minuteness of the heart volume (liters)	Systolic heart volume (ml)
On the horse	25-42	23	700
In cattle	50-75	45	700
Tuyada	32-52	24	700
In sheep and goats	60-80	5	70
In the pig	60-80	4.5	60
Itda	80-120	2	20
In the rabbit	100-140		
In mice	550-720		
In the chicken	130-200	0.4	2.5

If we know the number of heartbeats per minute, we can determine the minute volume of the heart by multiplying it by the systolic volume of the heart. Ythe

amount of blood pumped from the ventricles to the arteries in each systole of the heart is called the systolic volume of the heart. The systolic volume of the heart depends on the size of the heart cavities, the amount of blood flowing to the heart, the force of contraction of the heart muscle, the resistance of the arteries to blood flow.

4. External signs of heart function: tones and pulse. Due to the functioning of the heart, 2 different physiological sounds are heard from it and they are called heart tones:

1. Systolic tone. 2. Diastolic tone.

The systolic tone is formed by the closing of the stratified valves when the muscles of the ventricles contract, and sounds "bu-u" in the form of elongated, muffled, lower. Diastolic tone, on the other hand, results from the closing of the crescentic valves when the heart's ventricular muscles dilate and relax, and sounds short, resonant, and loud in the form of a "dup." If sounds other than these sounds appear in the heart, they are pathological sounds, signaling heart disease. A heartbeat is a change in the state of the heart during a systole of the ventricles of the heart, which strikes the chest wall. There are two types of heart attacks:

1. Yblow with the tip of the heart. (Observed in dogs and humans).

2. Heartbeat. (Observed well in all farm animals, especially horses).

5. Features of the heart muscle. The heart muscle has the following characteristics:

1. Mobility. 2. Conductivity. 3. Automation. 4. Refractoriness. 5. Formation of biotoxins.

Excitability property. The heart muscle is excited in response to the same impact as the skeletal muscle. But the excitation of the heart muscle is slow and lasts a long time. Cbecause the fibers in the heart muscle are symplastically interconnected by a protoplasmic bridge and are structured interconnected. For this reason, the effect on the heart muscle spreads evenly across all the fibers, generating excitation only when the force is higher than the force of the jump, and the heart is constantly working.

Conductivity property. The heart muscle has a special conducting system consisting of nerve nodes and muscle fibers.

The conduction system of the heart consists of:

1. Kiss-Flek or sinus node.

2. Ashof - Commodity or atrioventricular node.

3. Giss tuft and legs. 4. Purkine fibers.

Excitement occurs at the Kiss-Fleck node, first spreads to the heart chambers, and they contract. It is then transferred to the Ashof-Tovar node, from there through the Giss tuft and Purkinje fibers to the ventricles of the heart, and they contract. Excitement through the conduction system of the heart passes 10 times faster than in the skeletal muscles.

YThe excitation in the hurricane passes at the following speed:

1. Y1000-1200 mm / sec in the muscles of the heart chambers;

2. 0.02-0.05 at the Ashof-Commodity node *mm / sec;*

3. Giss tuft and legs 1500-5000 *mm / sec;*

4. 1000-5000 in Purkinje fibers *mm / sec;*

5. Y300-500 mm / sec in the muscles of the ventricles of the heart.

The slow passage of the movement in the Ashof-Tovar node is of great importance for the alternating contraction and relaxation of the various parts of the heart.

Cardiac automation refers to the ability of the heart to work independently outside the body in certain, specific conditions.

Ythe heart muscle is able to function independently under the influence of impulses generated directly in it, even when no impulse comes to it from the center.

There are 2 different theories that explain cardiac automation:

1. Neurogenic theory. 2. Myogenic theory.

Each theory is based on specific evidence and proofs. It would be expedient for us to study neurogenic and myogenic theories together as a neuromyogenic theory, without denying them. Because the nervous and muscular elements of the conduction system in the heart are so interconnected, their activities are inseparable.

Refractory property. Tetanic contraction is not typical for the heart muscle. Cbecause the heart muscle must definitely relax after a contraction. Tetanic contraction is characteristic of skeletal muscle. For example, when an animal is standing, the leg muscles are in a tetanic contraction and remain so for a long time. If an additional effect is given during the systole of the heart muscle, the heart muscle is excited to this effect and does not respond with contraction. The property of the heart muscle not to respond to a given effect is called refractoriness.

The refractory properties of the heart muscle are manifested in 2 phases:

1. Absolute refractory phase. 2. Relative refractoriness phase.

The phase of the heart muscle that does not respond at all to the given effect is called absolute refractoriness.

The phase of response of the heart muscle to a given strong impact with additional contraction is called relative refractoriness.

The extra, extraordinary contraction that occurs during the relative refractory phase is called an extrasystole. After an extrasystole, the pause time is prolonged. This is called a compensatory pause.

The refractory properties of the heart muscle are of great importance for cardiac activity. If the heart responds to various stimuli with contractions, its function would be impaired.

Properties of biotoxins. The heart muscle also has the ability to produce biocurrents similar to other excitable tissues.

The main reason for the formation of biocurrents is the formation of negative and positive electropotentials. Cbecause the excited part of the living tissue is negatively charged and the unexcited part is positively charged.

YThe bits formed in the urethra are studied by recording them using an electrocardiograph. This is called an electrocardiogram, and the recorded curve is called an electrocardiogram.

The study of the bits that form in the heart allows us to think about the activity of the heart. This is of great importance in veterinary practice.

Topic: Vascular physiology

Plan:

1. Physiology of blood vessels. The concept of the doctrine of hemodynamics.
2. The movement of blood in the arteries and the factors that supply it, blood pressure, velocities of blood, arterial and venous pulse.
3. Management of vascular activity. Reflexogenic components and their role in the regulation of cardiovascular activity.

Basic phrases:

Hemodynamics, hydrodynamics, blood vessels, aorta, arteries, arterioles, veins, hollow veins, capillaries, vascular anastomosis, blood flow rate, blood pressure, maximum pressure, minimum pressure, pulse (pressure) difference, pulse, arterial pulse, vein pulse, sphygmography, sphygmogram, phlebography, phlebogram, vascular tone, sympathetic nerve fiber, parasympathetic nerve fiber, vasodilators, vasoconstrictors, adrenaline, vasopressin, renin, serotonin, histamine, acetylcholine, ATF.

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1. The concept of the doctrine of hemodynamics. The movement of blood in the arteries and the factors that supply it. Hemodynamics is the study of the flow of blood

through blood vessels. Hydrodynamics is the study of the flow of fluids through tubes. Because blood vessels are tubular in nature and blood is fluid, the flow of blood through the vessels is governed by the laws of hemodynamics, hydrodynamics. This means that, like other fluids, blood flows from a place of high pressure to a place of low pressure. The heart works in a rhythm and becomes a blood vessel-and, although fired in separate portions, flows continuously in the veins as a continuous stream. This allows the blood to flow through the larger arteries without flowing along the arterioles and capillaries.

So, if the main cause of blood flow in the arteries is pressure, there are 3 factors that cause blood to flow from the veins to the heart:

1. Contraction of muscles by contraction of veins.
2. Valves within the veins.
3. Negative pressure inside the chest.

As the muscles contract, the veins constrict and move the blood forward.

The valves inside the veins prevent the blood that has been pumped forward from returning. Negative pressure inside the chest is formed periodically during cardiac diastole. This allows the blood to move towards the heart.

2. Blood pressure and factors affecting it. Blood pressure is the pressure that the blood exerts on the arterial wall due to the work of the heart.This is mainly due to the work of the heart and the tone of the vascular wall. In an organism, the arteries, like the branches of a tree, branch out and become narrower in diameter, and so does its resistance to the flowing blood. As a result, blood pressure drops as much. Therefore, the highest pressure is observed in the aorta, and the pressure gradually decreases as it passes through the arteries and capillaries.

In small diameter veins, the pressure is very low (equivalent to a 58 mm Hg column) and decreases in large veins. As a result, the pressure in the vena cava becomes even negative. During systole of the ventricles of the heart, the pressure in the arteries rises to a maximum, and during diastole falls to a minimum.

Accordingly, two types of blood pressure are distinguished: 1. Maximum (systolic) pressure. 2. Minimum (diastolic) pressure.

The amplitude of pressure change between systolic pressure and diastolic pressure is called pulse pressure or pulse difference.

Pulse waves of blood pressure in the arterioles and capillaries are not observed. There are two ways to measure blood pressure: 1. Blood method (K. Ludwig method)

2. Bloodless method.

Blood pressure is much more difficult to determine by the bloody method. A symbolic manometer is used to determine blood pressure in the arteries, and an aqueous manometer is used because the pressure in the veins is low. Blood pressure in capillaries Krog method measured with. The bloodless method is widely used to determine blood pressure. A sphygmomanometer is used for this purpose. Blood pressure of different animals

(in mm of mercury)

T / r	Animals	Maximum	Minimum	Location to be

	type	pressure	pressure	identified
1	Ot	100120	3550	Tail artery
2	Cattle	110140	3540	Tail artery
3	Camels	130 - 155	50 - 70	Tail artery
4	Sheep and goats	110120	5065	Hip artery
5	Dogs	120140	3040	Hip artery

Factors affecting blood pressure:

1. Acceleration and deceleration of the heart. 2. Narrowing and widening of the diameter of the vessels. 3. Increase and decrease in blood volume. 4. Excessive blood loss. 5. Viscosity of blood. 6. Resistance of arterioles and capillaries to blood. 7. Systolic and minute volume of the heart. 8. Physical work.
9. Nervous system. 10. The state of the organism. 11. The period of the day and the ambient temperature. 12. Type, breed, age, productivity, etc. of the animal.

Acceleration of heart rate, narrowing of the diameter of the vessels leads to an increase in blood pressure, and vice versa. Excessive blood loss from the arteries leads to a drop in blood pressure. Blood pressure is lower at night than during the day. As the animal ages, blood pressure increases due to the loss of vascular elasticity. An increase of 10 l of milk in milk causes a 30 mm rise in blood pressure. The pressure in the small circulatory system is 56 times lower than the pressure in the large circulatory system.

3. Blood velocities, arterial and venous pulses.

The movement of blood in the body in the arteries, i.e. the flow rate, varies in different arteries. The narrowest part of the vascular system is the aorta. Blood:

1. Aorta 400-500 mm / sec. 2. Blood in arteries of medium diameter 150-200 mm / sec. 3. 0.5 mm / sec in capillaries. 4. In medium diameter veins 6-14 cm / sec. 5. The hollow veins move at a speed of 20 cm / sec.

The total diameter of the capillaries in the body is on average 800 times the diameter of the aorta. Because there are more veins in the body than arteries, they have lower pressure.

Circulation time is the time it takes for a blood particle to circulate all the blood vessels in the major and minor circulatory systems.

Time for blood to circulate the body once: 1. 40 seconds in horses. 2. 32 seconds in cattle. 3. 14 seconds in sheep. 4. 13 seconds in goats.

5. In rabbits, it is 8 seconds.

4/5 of the circulatory time is spent passing through the large circulatory area and 1/5 of the small circulatory area.

This is because the blood vessels of the small circulatory area are short and large in diameter. The blood vessels of the greater circulatory system are longer and smaller in diameter. A pulse, or pulse, is said to be the rhythmic undulating movement of the blood vessel wall due to heart activity.

There are two types of pulses in the body: 1. Arterial pulse 2. Venous pulse

Arterial pulse depends on the activity of the ventricles of the heart.

Because the heart is in a certain rhythm, the blood to the aorta-pumps. An arterial pulse propagates much faster than the rate at which blood flows. For example, the rate

of blood flow in the aorta is 400-At 500 mm / sec, the pulse wave propagates at a speed of 79 m / sec. This means that the pulse travels 1418 times faster than the blood in the aorta.

As it moves away from the heart, the pulse wave fades and disappears in the capillaries.

Arterial pulse:

1. From the external jaw artery in horses and cattle.

2. In small animals it can be detected by palpation of the femoral artery.

3. Cbecause the subcutaneous fat layer of pigs is much thicker, the pulse is more difficult to detect by palpation. The arterial pulse can be studied graphically by recording it sphygmographically. Ythe thinned line is called the sphygmogram.

Pulse is characterized by the following signs:

1. Cthe number of contractions and expansions of the heart per unit time.

2. Rhythm is the repetition of pulse waves at equal intervals of time.

3. Velocity is the propagation of pulse waves along the vessel walls.

4. Elevation is the extent to which a pulse wave can expand the vessel wall.

5. Pulse force is the force required to press on the vessel wall for the pulse wave to disappear.

Pulse:

1. Depending on the speed: accelerated or decelerated. 2. By frequency: more or less. 3. Depending on the rhythm: rhythmic or non-rhythmic. 4. Depending on how much the pulse wave can dilate the vein: high or low. 5. Depending on strength: can be loose or rigid (strong or weak).

The pulse changes during various diseases. Therefore, the detection of arterial pulse is of great importance in the study of various diseases in the body.

The venous pulse depends on the activity of the heart chambers. The venous pulse is mainly observed in the areas of the large vena cava near the heart. Pulse waves are not recorded in small, small-diameter veins. Vienna pulse phlebograph instrument can be recorded and studied using. The recorded curve is called a phlebogram. The study of venous pulse recording is of great importance in the examination of cardiac activity.

4. Management of vascular activity. Reflexogenic components and their importance. All blood vessels in the body dilate as a result of various influences. This is called vascular tone.

Vascular tone of the central nervous system - controlled by sympathetic and parasympathetic nerve fibers.

There are nerve fibers that narrow and dilate blood vessels:

1. Vasoconstrictors are nerve fibers belonging to the sympathetic nervous system, which increase vascular tone and narrow blood vessels. However, the coronary arteries, the nerves that control the cerebral arteries, are an exception, because when the sympathetic nerve fibers are excited, the wall of these arteries expands.

2. Vasodilators are nerve fibers that reduce vascular tone, dilate blood vessels, and although some of them belong to the parasympathetic nervous system, most of them are part of the trunk of the sympathetic nervous system.

While the nerve fibers that constrict the arteries have a continuous effect on the vessel wall, the nerve fibers that dilate the arteries do not have a continuous effect.

Centers that control vascular tone:

1. The elongated brain is the main center, which was discovered in 1871 by the Russian scientist FV Ovsyannikov. 2. The lateral branches of the spinal cord are the secondary center. 3. The cortex of the midbrain and cerebral hemispheres.

Vascular tone is also controlled by the reflex pathway. Reflexogenic parts - zones - play an important role in the reflex control of vascular activity. In the body reflexogenic components:

1. In the aortic arch. 2. At the junction of the carotid artery with the external and internal carotid arteries. 3. The vena cava is at the point of infusion into the right ventricle.

The receptors of the reflexogenic zones in the vessels play an important role in maintaining the stability of blood pressure.

Due to their activity, 2 different reflexes are formed:

1. Depressor reflexes - reflexes that lower blood pressure.

2. Pressor reflexes - reflexes that increase blood pressure.

Depressor reflexes lower blood pressure due to slowing of heart rate and dilation of blood vessels.

Pressor reflexes, on the other hand, increase blood pressure due to increased heart rate and narrowing of the arteries.

Vascular tone can also be managed humorally. It is different in the blood-The district contains biologically active substances and hormones:

1. Adrenaline is a hormone produced by the adrenal cortex that narrows all the blood vessels except the coronary arteries and the blood vessels of the brain. 2. Vasopressin is a hormone released from the back of the pituitary gland that narrows arterioles and capillaries to a lesser extent and arteries and veins to a greater extent. 3. Renin is a chemical produced by the kidneys that cannot constrict blood vessels on its own. It acts on hypertensinogen, one of the plasma globulin proteins, converting it to hypertensin and narrowing the arteries. 4. Serotonin is a substance that breaks down and separates from platelets when blood vessels are injured, narrowing the arteries and creating favorable conditions for blood clotting.

5. Histamine is a chemical that is formed as a result of metabolism and dilates blood vessels.

6. Acetylcholine is a mediator that is released from the ends of parasympathetic nerve fibers and slightly dilates blood vessels.

7. ATF (Adenosine triphosphate) is a substance formed due to the activity of skeletal muscles and dilates blood vessels.

The humoral system is definitely involved in the regulation of vascular activity in close connection with the nervous system.

Topic: Physiology of the respiratory system.

Plan:

1. The essence, stages and mechanism of respiration. Types and rate of respiration.
2. Ventilation coefficient of the lungs. Gas exchange in the lungs.
3. Transport of gases by blood. Oxygen capacity of the blood. Gas exchange between blood and tissues.
4. Partial pressure of gases.
5. Management of the respiratory process and the influence of various factors on them. Respiratory properties in different conditions.

Basic expressions.

Breathing, external respiration, internal respiration, oxygen, carbon dioxide, lungs, nasal cavity, oral cavity, throat, larynx, larynx, bronchi, alveoli, external and internal dentate muscles, elasticity , negative pressure, inspiration, expiration, living air capacity, total air capacity, dead space, ventilation coefficient, minute ventilation volume, gas exchange, gas transport, gas melting coefficient, partial pressure of gases, blood oxygen capacity, hypoxemia, hypoxia, anoxia, sympathetic and parasympathetic nerves, reflexogenic part, gipernoe, apnea.

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Respiration is a physiological act involving a number of biochemical processes that ensure the release of carbon dioxide and water as a result of the consumption of oxygen in the body.

Energy is required for the occurrence of various physiological processes in the body. This energy is mainly produced as a result of oxidation-reduction processes in the body. Oxidation processes take place in the presence of oxygen. Therefore, for the vital activity of all the cells in the body, the process of respiration must be continuous.

In highly developed organisms, the process of respiration consists of the following stages:

1. External breathing:
a) air exchange between the external environment and the alveoli of the lungs;
b) gas exchange between alveolar air and blood.
2. Transport of gases in the blood:
a) transport of oxygen from the lungs to the tissues by blood;
b) transport of carbon dioxide from the tissues to the lungs by blood.
3. Inhalation:
a) exchange of gases between blood and tissues;
b) the release of carbon dioxide by cells by consuming oxygen (cell respiration).

This means that the lungs are involved only in external respiration, that is, the exchange of gases between the external environment and the blood. The lungs are a well-developed pair of organs that are connected to the external environment through the nasal and oral cavities, throat, larynx, larynx, and bronchi. The bronchi branch off and form bronchioles, which end in air bubbles - alveoli. In the wall of the alveoli, capillaries clump together to form a network. The wall of the alveoli and capillaries consists of a single layer of cells, which is a very good condition for the exchange of gases between them. In order for gases to be exchanged through the lungs, they must constantly expand and contract. But he doesn’t have the specific muscles that can keep him from expanding and contracting. But because it is located in a closed cavity of the chest, the chest expands as it expands, and narrows as it contracts. Hence, the lungs move passively, following the active movement of the thorax. This movement occurs with the birth of the animal.

Physiology of the first breath. When the baby is born, the umbilical cord is severed and the communication between the baby and the mother ceases immediately. As a result, carbon dioxide accumulates in the baby's blood, increasing its amount and stimulating the respiratory center. Impulses generated in the respiratory center due to agitation cause contraction of the external intercostal dentate muscles in the chest. As a result, the thorax dilates, the heads of the ribs fall into the corresponding grooves of the vertebrae, and the lifespan does not return. Negative pressure is created inside the chest and it is less than the external ambient (atmospheric) pressure by 6-15 mm Hg. As a result, oxygen begins to enter the lungs from the outside environment, and the baby animal breathes for the first time.

Mechanism of inhalation and exhalation. Inhalation is called inspiration, exhalation is called expiration.

Respiratory mechanism. Breathing occurs due to the expansion of the chest to width, height, and height due to contraction of the intercostal external dentate muscles. At this point, the sternum is pulled down and the diaphragm is pulled toward the abdomen. As a result, the lungs also expand behind the chest, the pressure inside it

decreases, and air is absorbed into it. Air absorption continues until the pressure inside the lungs equals atmospheric pressure.

The intercostal external dentate muscles involved in the expansion of the thorax are called inspiratory muscles. As you finish breathing, you begin to exhale.

Exhalation mechanism. Exhalation occurs due to the narrowing of the chest in width, height, and height due to contraction of the intercostal muscles. At this point, the sternum and diaphragm return to their previous position. As a result, the lungs constrict and air is expelled from the chest. The intercostal muscles, which are involved in the contraction of the thorax, are called expiratory muscles. Hence, the processes of respiration and expiration continuously control and maintain each other's activities.

There are 3 types of breathing:

1. Chest-to-rib breathing. 2. Breathing through the abdomen-diaphragm.
3. Mixed, i.e. chest-abdominal breathing.

Chest-to-rib breathing is observed in dogs and women. Breathing through the abdominal diaphragm is observed in men. Mixed-type respiration is observed in farm animals. Respiratory types can change during various physiological conditions and diseases of the body. For example, animals breathe in the thoracic-rib type during pregnancy and in the abdominal-diaphragm type in lung diseases. Respiratory rate (frequency) varies in animals and birds.

Respiratory rate (frequency) of different animals

Animal type	The number of breaths per minute
Horses	8-16
Cattle	10-30
Sheep and goats	16-30
Pigs	8-18
Camels	5-12
Dogs	10-30
Rabbits	50-60
Mice	200

Respiratory rate depends on the following factors:

1. Type of animal. 2. To the body of an animal. 3. To the breed of animals.
4. The age of the animals. 5. The sex of the animals. 6. To the productivity of animals. 7. To the physiological state of animals. 8. Intensity of metabolism. 9. The season of the year. 10. Depends on ambient temperature and so on.

For example, respiration is faster in small animals than in large animals, in young animals it is faster than in older animals, in high-productivity animals it is faster to breathe than in low-productivity animals, and vice versa.

Lung ventilation coefficient. Not all of the air inhaled reaches the alveoli of the lungs. About 30% of it remains in the upper respiratory tract and is not involved in the exchange of gases in the lungs.

This air is called "harmful" or "dead" air.

This air is of great importance for the process of respiration and for the organism. This is because the air we breathe is heated, purified and saturated with water vapor in

the upper respiratory tract. If such functions were not performed in the upper respiratory tract, various diseases would have arisen in the respiratory system, in the body as a whole.

The ventilation coefficient of the lungs is the ratio of the portion of inhaled air that reaches the alveoli of the lungs to the alveolar air.

For example, if a horse inhales 5 liters of air each time, 30% of that air, or 1.5 liters, is trapped in the upper respiratory tract, and the remaining 3.5 liters of air reaches the alveoli of the lungs. If we assume that the amount of alveolar air in horses is 22 l, then the ventilation coefficient of the lungs is 3.5: 22 = 1.6.

This means that 6 to 1 part of the alveolar air is exchanged with the air we breathe each time the animal breathes.

The amount of air taken into the lungs per minute is called the minute ventilation volume of the lungs.

Per minute ventilation volume of the lungs:

1. The speed of breathing movements. 2. Animal nutrition. 3. Time of day. 4. The season of the year. 5. Physiological state of the body (pregnancy). 6. Intensity of metabolism and others are affected.

For example, pulmonary ventilation increases 5 times when the horse is walking and 8 times when walking slowly. The minute ventilation volume of the lungs does not fully reflect the state of pulmonary ventilation. Gas exchange in the lungs. There is a continuous gas exchange between the alveolar air in the alveoli of the lungs and the blood in the capillaries in the alveolar wall. Due to the diffusion phenomenon of gases move from a place where the partial pressure is high to a place where the partial pressure is low. This means that gases pass from the alveolar air to the blood in the lungs and from the blood to the alveolar air.

The partial pressure of gases is the fraction of the total pressure of a mixture of gases that corresponds to a certain proportion of gas in the mixture.

№	Gases in the atmosphere:		
	name	amount (%)	partial pressure (mm.mercury column)
1	Oxygen	20.96	159.29
2	Carbon dioxide	0.03	0.28
3	Nitrogen	78.13	593.79
4	Inert gases	0.88	6.69
	TOTAL:	100.0	760

Transport of gases by blood. One of the most important functions of the blood in the body is to transport gases, that is, to transport O2 from the lungs to the tissues and cells, and SO2 from the tissues and cells to the lungs.

The blood does not fully absorb SO2 in the lungs and O2 in the tissues. A certain amount of O2, SO2 and N2 in the blood is constantly circulating throughout the body.

The transport of gases in the blood depends on their state in the blood.

Gases in the blood are in 2 different states:

1. Physically dissolved, in a free state. 2. In a chemically bound state.

In the blood, 0.3% of O2 is dissolved, 99.7% is dissolved, 2.7% of SO2 is dissolved, 97.3% is dissolved, and the amount of N2 is 1%, only in the dissolved state.
This means that some of the gases in the blood are dissolved in the plasma and most are transported in combination with the hemoglobin (Nb) in the erythrocytes.
According to Henry's law, the solubility of gases in a liquid depends on their nature, the partial pressure, and the temperature of the liquid.
The specific gas volume that can be dissolved in 1 ml of liquid under normal conditions (0o temperature and 760 mm Hg) is called the melting coefficient of the gas.
The lower the temperature of the liquid, the higher the pressure of the gas, the more gas dissolves in that liquid, and vice versa. When the body temperature is normal and the pressure is 760 mm Hg, the melting coefficient of gases in the blood plasma is as follows:
1. Oxygen - 0.022. 2. Carbon dioxide - 0.511. 3. Nitrogen - 0.011.

The melting coefficient of nitrogen can fully express the amount in the blood. The melting coefficient of oxygen and carbon dioxide in the blood does not fully represent the amount of these gases in the blood.

If both oxygen and carbon dioxide in the blood were only in the dissolved state, their melting point would not exceed 0.3% of the amount of O2 in the blood, and that of SO2 would not exceed 2.7%.

But in fact, arterial blood contains 20% oxygen, carbon dioxide 30-40%, venous blood 12% oxygen and 50-55% carbon dioxide.

This means that less of the O2 and SO2 in the blood is freely dissolved and more is chemically bound.

Oxygen capacity of the blood. One of the remarkable properties of hemoglobin in the blood is that it binds oxygen very lightly at high partial pressure - in the lungs, where it is low at partial pressure - and easily excretes it in the tissues. The oxygen capacity of the blood is defined as the amount of oxygen required to convert hemoglobin in 100 ml of blood into complete oxyhemoglobin.

When 1 gram of hemoglobin is converted to complete oxyhemoglobin, it attaches 1.34 cm2 of oxygen. If we multiply it by 1.34 cm2, taking into account that the blood of various farm animals contains an average of 13-15 grams of hemoglobin, the average oxygen content of the blood in them is 17.32 - 20.0 cm2. does. By knowing the oxygen capacity of the blood, it is possible to determine the amount of O2 in the freshly drawn blood from the blood vessel and to think about how saturated this blood is with O2.

If the blood is not well saturated with oxygen, various changes will take place in the body. For example:

Hypoxemia is a decrease in the amount of oxygen in the blood.

Hypoxia is a decrease in the amount of oxygen in the tissues.

Anoxia is the complete absence of oxygen to the tissues.

If no emergency measures are taken in such cases, the organism dies. Therefore, it is impossible to reduce the organism of animals to such conditions.

Management of the respiratory process. Respiratory processes in the body are controlled in 2 different ways: 1. Through the nervous system. 2. In a humorous way.

The main center that controls the activity of the respiratory system is located in the cerebellum, which was identified and studied in 1885 by the Russian physiologist NDMislavsky. The center of the elongated brain is made up of a pair of symmetrical parts, each side of which controls the breathing movements on the corresponding side of the chest. Therefore, whichever side of the respiratory center is injured, the breathing movements on that side of the chest stop. The secondary lower center that controls respiration is located in the cortex of the spinal cord and cerebral hemispheres.

The respiratory center responds to stimuli from various parts of the body: the lungs, the walls of blood vessels, the sinuses of the carotid arteries, the pleura, and other organs through the corresponding fibers of the sympathetic and parasympathetic nerves. In the lungs, there are two fibers of the parasympathetic nerve, one of which is excited when the pressure in the lungs decreases and the other when the pressure rises. It is these fibers that make breathing self-regulating.

The sinuses of the carotid arteries and the reflexogenic parts of the aortic arch play a special role in respiratory activity. The chemoreceptors located in these reflexogenic parts are excited when SO2 increases or O2 decreases in the blood and change respiration. The nervous system plays a leading role in the functioning of the cardiovascular system and the respiratory system in close connection with each other. For example, when the reflexogenic part of the carotid artery is affected, the heart slows down, the blood vessels dilate, and breathing slows down. When the blood pressure drops, the heart rate speeds up, the blood vessels constrict, breathing speeds up and deepens a bit. When the mucous membranes of the larynx, larynx, and bronchi are affected, the slowing of breathing causes the heart to slow down.

The functioning of the cardiovascular system and the respiratory system in close connection with each other is of great importance in the adaptation of all vital processes in the body to the changing external environment.

Humoral control of the respiratory process. The humoral system also plays an important role in the regulation of respiration. The most important of the substances involved in the management of asthma is carbonic acid.

An increase in carbonic acid in the blood speeds up and deepens breathing. For example, if the airways are closed for 20-30 seconds, breathing becomes faster and deeper (hypernoeal) due to an increase in carbonic acid in the blood. If a person exhales more often than at rest, during this time the breathing becomes sparse and shallow (apnea) due to a temporary decrease in carbon dioxide in the blood.

This means that carbonic acid in the blood is an active stimulant of the respiratory center.

In the mouth and digestion of nutrients in the stomach

Plan:

1. The concept of digestion. Functions of the digestive system and methods of its study.
2. Digestion of nutrients in the mouth and its properties in different animals. The composition, importance, quantity and properties of saliva in different animals.
3. Salivation and its management. General laws of digestion of food in the stomach.

4. Gastric juice, composition, importance, separation and its administration. Stomach movement. Laws of transfer of nutrients from the stomach to the intestine.

Basic expressions.

Digestive system, digestive process, food, animals, proteins, fats, carbohydrates, vitamins, minerals, enzymes, proteolytic, amylolytic, lipolytic, fermentation, absorption, secretion, motor, incretory, lips, teeth, tongue, chewing , swallowing, bite, salivary glands: auricle, submandibular, sublingual, salivary, lysozyme, amylase, maltase, maltose, glucose, glycopolysaccharide, mutsin, urea, ammonia, carbon dioxide, ascorbic acid, reflector, humoral, sympathetic and parasympathetic nerve fibers, conditioned and unconditioned reflexes, kallikrein. Stomach, single-chambered simple and complex stomach, 2,3 and 4-chambered stomach, serous membrane, muscle layer, submucosa, mucous membrane, cardial, fundal, pylorus, secretory glands, extra cells, stem or head cells, coating cells, pepsin, pepsinogen, chymosin, catepsin, gelatinase, lipase, hydrochloric acid, reflector phase, chemical phase, MNS, receptor, sympathetic nerve, stray nerve, motor, tonic movement, rhythmic movement, worm-like movement.

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1. General concept of digestion. Functions of the digestive system.

Digestion is the first stage of the constant metabolism between the organism and the external environment.

From 2 different food sources for feeding mammals:

1. 60-70% of plant food.

2. 20-30% use animal food.

Nutrients contain proteins, fats, carbohydrates, vitamins, minerals and others necessary for the body.

Digestion is a complex physiological process that involves the absorption of vital substances into the digestive system, where they are broken down, the complex substances are broken down into simple substances, the vital substances are absorbed into the blood and lymph, and the unnecessary ones are excreted.

The body constantly needs energy to survive. This energy is due to the breakdown of proteins, fats and carbohydrates in the food consumed by the animal.

In this case, the importance of various biocatalysts, ie enzymes, contained in the fluids of the digestive system: saliva, stomach, pancreas, intestinal juices. Digestive enzymes are divided into three groups depending on which organic matter they break down:

1. Proteolytic, i.e. enzymes that break down proteins.
2. Glycolytic or amylolytic, i.e. enzymes that break down carbohydrates.
3. Lipolytic, i.e. enzymes that break down fats.

In addition to enzymes, bile fluid, which is formed in liver cells, also plays an important role in the digestion of nutrients.

Functions of the digestive system:

1. Secretary - shiraajrat. 2. Motor - movement. 3. Ensuring the fermentation process. 4. Ensure the absorption process. 5. Incretor - the function of hormone production. Digestive processes begin in the oral cavity of animals and poultry and continue in the stomach and intestines.

2. Digestion of nutrients in the mouth and its variety
properties in animals.

Digestion of nutrients in the mouth is divided into three stages:

1. Take food by mouth. 2. Direct digestion of food in the mouth.

3. Swallowing. Taking food by mouth. Before taking food into the mouth, the animal sees it, smells it, and distinguishes it from non-food, junk, and toxins. The food taken into the mouth is analyzed due to the sense of taste, and the unnecessary part is removed. The oral intake of food varies in farm animals, and the animal's lips, teeth, and tongue are involved in this process.

1. Horses, sheep, and goats cut grain feed using their lips and direct it into their mouths using their tongues.

2. The lips of cattle are less mobile, and their tongue is mainly involved in food intake, and to a lesser extent the shovel teeth. They twist and wrap the food with their tongues, press it to the upper jaw and direct it to the mouth.

3. The feeding of pigs is also carried out mainly by the teeth and tongue of the shovel. Animals lick soft food and salt with their tongues.

YThe soup is fed to the mother suckling animals. At this point, the lower jaw is lowered and pulled through the tongue. As a result, negative pressure is created in the oral cavity and ensures that milk is absorbed into the mouth.

Although the process of drinking water does not occur uniformly in different animals, the law of its soil is almost the same. As the animal drinks water, it dips its beak into it, then moves its lower jaw in the opposite direction to its upper jaw, a phenomenon that continues with repeated repetition of the tongue. This movement of the jaw and tongue ensures that water is absorbed into the mouth.

Food intake and water intake inevitably take place under the control of the central nervous system.

Chaynash. The nutrients taken into the mouth are repeatedly chewed, mixed with saliva, and made into a bite.

Chewing takes place as follows: due to the activity of the jaw muscles, the lower jaw moves downwards and then upwards, towards the upper jaw. The jaws move in opposite directions, and the teeth (in ruminants, the upper jaw gums collide with the lower jaw teeth), then they move sideways. As a result, the chewed food is cut, crushed, rubbed and crushed. The unevenness of the tooth surface is of great importance in the grinding of food. Animals usually chew food on one side of their mouths, at which time the mouth is closed. Cattle, on the other hand, keep their mouths open when chewing food, so they keep their heads in a horizontal position.

Different animals chew food at different levels.

Chewing animals swallow food from afar.

Horses and pigs chew well and swallow finely.

Carnivores, on the other hand, break the food into pieces and swallow it without chewing.

Chewing is an optional process and is controlled reflexively. Nutrients affect mechanoreceptors in the oral cavity. The resulting excitation impulse is transmitted through the afferent nerve fibers to the masticatory center in the medulla oblongata. The response of the center is transmitted to the jaw muscles through the corresponding efferent nerve fibers. As a result, the jaw muscles contract, become active, and perform chewing movements.

Cthe midbrain, the hypothalamus, and the cortex are also involved in the regulation of edema.

Swallowing. Once the food taken into the mouth is well chewed and mixed with saliva, it is pushed towards the root of the tongue due to the movement of the tongue and cheek in the form of a bite. As the tongue contracts and moves, the bite rests on the soft palate and is directed toward the base of the tongue and into the larynx. The food bite transferred to the larynx causes the soft palate-lifting muscles to contract as a result of the receptors in the laryngeal mucosa, and the soft palate rises, thus blocking the passage to the nose. The base of the tongue, on the other hand, lifts the uncle above the larynx and closes the laryngeal mouth. Sthus, food will not enter the upper respiratory tract. As a result of the contraction of the laryngeal muscles, food is transferred to the esophagus.

Swallowing is a complex reflex process located in the central cerebral cortex that controls it.

3. Separation, composition and physicochemical properties of saliva.

Separation of saliva. Saliva mainly consists of three pairs of glands:

1. He listened. 2. Til osti. 3. Produces salivary glands under the jaw.

In addition, salivary glands are involved in the formation of salivary glands in the mouth, at the base of the tongue, in the throat, and in some other cells. The anterior salivary gland is made up of serous cells that secrete an aqueous fluid containing protein.

The submandibular and sublingual salivary glands are composed of serous and mucous cells and secrete a mucous fluid called mucin.

Saliva is a colorless, mucous and sticky liquid.

The composition of saliva consists of 2 parts:

1. Water (99.0 - 99.4%). 2. Dry matter (0.6 - 1%).

Organic and inorganic substances are stored in the dry matter. Saliva:

1. Inorganic substances include chlorides, sulfates, carbonates, calcium, potassium and others.

2. Organic substances include amylase, maltase, lysozyme enzymes, adhesive - glycopolysaccharide - mutsin, as well as metabolic products - urea, ammonia, carbon dioxide and others.

Physicochemical properties of saliva. Amylolytic enzymes are very rare in saliva, and the amylase in it breaks down starch to maltose, and maltase breaks down maltose to glucose.

Lysozyme has bactericidal properties, kills a variety of microorganisms, and is more abundant in the saliva of carnivorous animals.

Mutsin is a slimy substance that makes the saliva sticky and plays an important role in making the food a bite and easy to swallow.

The specific gravity of the saliva of different animals averaged 1,002 - 1,012, while the environment pH = around 7.32 - 8.1, osmotic and oncotic pressures are low.

4. The amount and separation properties of saliva in different animals.

Saliva secretion in different animals is more or less different from each other.

Salivation in horses. Horses occasionally secrete saliva when food falls into their mouths. Horses secrete about 40 liters of saliva a day, and its main function is to wet and swell the feed. Amylolytic in horse salivaenzymes (amylase, maltase) begin to break down carbohydrates in the mouth, albeit in small amounts.

Csalivation in pigs. It is not much different in essence from horses. Adult pigs secrete about 15 liters of saliva per day, and it contains a lot of amylolytic enzymes. This is why more carbohydrates are broken down in the mouths of pigs.

In ruminants, salivation is a constant food in the large abdomen, where the salivary glands secrete saliva without interruption, as digestive processes continue uninterrupted. Other salivary glands occasionally secrete saliva only when food falls into the mouth.Adult cattle produce 90-190 liters of saliva, and sheep 6-10 liters. The significant alkalinity of the saliva of ruminants is of great importance for the proper conduct of digestive processes in the large intestine. This is because the acidic substances formed

in the large abdomen are neutralized. Ascorbic acid (vitamin C) in saliva has a positive effect on the growth of microorganisms in the large intestine, enhances the activity of enzymes.The saliva of ruminants contains about 15 - 36 mg.% Of urea, which is absorbed by microorganisms in the large intestine. This means that saliva is also involved in nitrogen metabolism in the body of ruminants. Saliva secretion in young animals is somewhat different. The sublingual and submandibular salivary glands of milk-sucking calves, which have not yet developed anterior gastric compartments, secrete more saliva than the ear glands. After the animal begins to feed entirely on coarse food, the auricle salivary glands become a constant source of saliva due to the intensification of fermentation processes in the large abdomen.

Saliva plays an important role in the digestion of milk. Cbecause proteolytic enzymes in milk have a better effect on milk mixed well with saliva. Therefore, when feeding calves by hand, special attention should be paid to drinking milk, using special pacifiers. It is not possible to drink milk directly from a bucket or other container. Because the calf drinks milk in a hurry, as a result, the milk does not mix well with saliva and digestion is impaired.

5. Management of salivary secretion.

The activity of the salivary glands is controlled by 2 systems:

1. Nervous system. 2. Humoral system.

The main center that controls salivation is located in the medulla oblongata.

Foods that enter the mouth affect a variety of receptors (mechano, chemo, thermo) on the oral wall, cheek, and tongue. The resulting excitation impulse is transmitted through the corresponding afferent nerve fibers to the salivary separation center in the medulla oblongata, and the center is excited. The response of the center comes to the salivary glands through the corresponding fibers of the parasympathetic and sympathetic nervous systems. When the parasympathetic nerve fibers are stimulated, a large amount of fluid is released, and when the sympathetic nerve fibers are stimulated, a small amount of thick saliva is released. This is the unconditioned reflex salivation. Certain groups of nerve cells in the midbrain, thalamus, hypothalamus, and cerebral cortex are also involved in regulating salivation.

In daily life, the appearance or smell of food in the animal's eyes or in the nose causes the saliva to separate independently, i.e., before the animal has eaten the food. This is the release of saliva by a conditioned reflex.

Hence, the process of salivary separation is controlled by a complex reflex pathway using conditioned and unconditioned reflexes. Along with the nervous system, the humoral system is also involved in the management of saliva secretion.

For example, when the parasympathetic nervous system is stimulated, a tissue hormone called kallikrein is produced. It dilates blood vessels and alters the permeability of cells to the skin. This has a positive effect on saliva secretion.

1. General laws of digestion of food in the stomach.

Properly digested in the mouth, mixed with saliva and swallowed in the form of a bite, the food enters the stomach through the esophagus. In the stomach, food mixes with gastric juice, undergoes mechanical, physical, chemical effects, is broken down, and some parts are partially absorbed into the bloodstream.

In animals and birds, the stomach has several chambers:
1. A single chamber is found in the normal stomach - dogs, cats and rats.
2. One-chambered complex stomach - occurs in horses, pigs and rabbits.
3. Two-chambered stomach - occurs in birds.
4. Three-chambered stomach - occurs in camels and deer.
5. Four-chambered stomach - occurs in cattle, sheep and goats.
In animals and poultry, the stomach has a different structure.
The structure of the stomach itself is divided into 3 parts:

1. Cardiac - the part of the esophagus that enters the stomach. 2. Fundal - the bottom of the stomach, the main part. 3. Pylorus is the part of the 12-finger bowel exit.

1. The wall of the stomach consists of 4 layers:
1. Outer serous membrane layer. 2. The middle muscle layer. 3. Subcutaneous layer of mucous membrane. 4. The inner mucous membrane layer.
The stomach wall is made up of three types: longitudinal, circumferential, and curved.
There are 3 types of gastric mucosa:
1. Additional gland cells. 2. Stem or head gland cells.
3. The lining contains glandular cells that produce gastric juice.
The extra glandular cells of the stomach produce mucus, the main gland cells produce enzymes, and the lining gland cells produce hydrochloric acid.
Stomach:
1. Only additional glandular cells in the cardiac part.
2. Additional, head, covering gland cells in the fundal part.
3. The pylorus contains additional and primary glandular cells.
Stomach:
1. Only mucus in the cardiac part.
2. Mucus, enzymes, hydrochloric acid in the fundal part.
3. Mucus and pepsin enzyme are produced in the pylorus.
Hence, gastric juice plays an important role in the digestion of nutrients.
2. Gastric juice, composition, importance, separation and administration.
Gastric juice is a liquid with a clear, colorless, acidic environment, formed due to the activity of additional, primary and covering glandular cells located in the gastric wall.

Me'da The juice contains more than 99% water, various inorganic and organic substances.

Inorganic substances include chlorine, phosphorus, carbonate, sulfuric salts and hydrochloric acid of elements such as Ca, K, Na, Mg, ammonium.

Organic substances include: lactic acid, creatinine, ATF, urea, uric (uric) acid, proteins, amino acids and enzymes.

Chloric acid in gastric juice plays an important role in the proper conduct of digestive processes in the stomach.
Functions of hydrochloric acid:
1. Gives gastric juice an acidic environment. 2. Carries out fermentation processes in the stomach. 3. Participates in the dissolution of minerals.

4. Exaggerates proteins and helps digestion. 5. Converts the enzyme pepsinogen to active pepsin. 6. Demonstrates bactericidal properties. 7. 12 fingers from the stomach reflexively provide the passage of food to the intestine.

Chloric acid in gastric juice is around 0.4-0.5% in 2 different states:

1. In the case of free hydrochloric acid - around 0.15-0.25%. 2. When combined with organic matter - more part.

Chloric acid is slightly more in carnivorous animals and less in herbivores.

Gastric juice contains the following enzymes:

1. Pepsin. 2. Chymosin. 3. Katepsin. 4. Gelatinase. 5. Lipase.

Pepsin is released in the inactive state of pepsinogen and converted to active pepsin under the influence of hydrochloric acid, in a highly acidic environment (pH = 0.8 - 2.0) and at a temperature (+ 38 + 40o). Pepsin breaks down proteins into albumin and peptones. Pepsin breaks down meat protein quickly and egg protein slowly.

Chymosin is active in a weakly acidic, weakly alkaline, ie neutral environment in the presence of Sa + ions, and the milk protein reacts with the caseinogen, converting it to casein, i.e., fermenting milk. This enzyme is abundant in the stomachs of young animals, and as the animals age, this enzyme decreases and other enzymes increase. This enzyme is used as a yeast in the preparation of cheese, which is separated from the juices of young lambs when slaughtered on the skin, dried.

Cathepsin breaks down the halogenated parts of the protein, the connective tissue gelatin.

The amount of gelatinase is very low and breaks down the connective tissue protein - gelatin.

Lipase only breaks down emulsified fats. Gastric lipase is much weaker than intestinal lipase.

Pure gastric juice does not contain amylolytic enzymes. But under the influence of saliva and food enzymes that fall into the stomach with a bite, carbohydrates are partially broken down. This is because the syrup is slowly absorbed into the food bite that enters the stomach, and the environment there becomes alkaline. Then, with the absorption of gastric juice into the bite, the environment changes to an acidic side, the activity of amylolytic enzymes ceases, and the enzymes of gastric juice begin to act.

The most active proteolytic enzyme in gastric juice is pepsin, which does not break down the gastric wall. Because: antipepsin substances are formed in the stomach wall; gastric mucosa - has a protective barrier property; the blood flowing along the stomach wall has an alkaline environment. All this reduces the activity of pepsin and it cannot break down the stomach wall.

Separation and administration of gastric juice. Gastric juice is secreted from time to time as food is consumed.

Neurohumoral effects play a key role in the secretion of sap from the glands in the stomach wall.

There are reflex and humoral, ie chemical phases of gastric juice secretion.

In the reflex phase of mucus secretion, 4-5 minutes after the nutrients enter the mouth or stomach, the mucus separates from the glands in the gastric wall, which is caused by the excitation of the centers that control mucus secretion through the CNS.

The main center that controls the secretion of sap is in the cerebellum, which is connected to the central brain. The excitation of the center occurs as a result of various non-spherical and conditioned effects. Syrup is secreted from the side effects by the action of nutrients on receptors in the mouth or stomach wall. Reflex secretion is mediated by the VNS and is mediated by sympathetic and sciatic nerves. The stray nerve is a secretory nerve that enhances the secretion of sap, is rich in fluid, salts and acids, has low fermentation capacity, under the influence of the sympathetic nerve secretes a small amount but thick syrup. The juice has a lot of organic compounds, a lot of enzymes from substances of a protein nature, and the fermentation power is high. The juice secreted in the reflex phase is generally considered to be the appetite juice that affects the breakdown of primary foods. The separation of aphids in the reflector phase depends on the properties and characteristics of various foods, including yogurt when a large amount of liquid syrup is released under the influence of coarse food, but under the influence of liquid, soft foods syrup separation is less, dark syrup is released, The fermentation capacity varies. The fermentation power of the juice is studied by the Matt (tube) method. (A special microcapillary tube is divided into scales, in which food is placed into the tube, leaving the effect of the syrup in a thermastat condition, and digestion is determined by looking at these scales). The animal consumes meat, protein, fat products, is digested in a certain period, and the ability to ferment is studied. Digestion depends on the temperature, composition and other characteristics of the food.

In the chemical phase of gastric juice secretion in the stomach, about 20-30 minutes after ingestion of food, the secretion of juice begins and lasts 40-45 minutes. The secretion of sap is influenced by the nutrients in the blood. Proteins, fats, carbohydrates, various salts in food NaCl, various acids (amino acids, fatty acids) act as a cause that enhances the process of syrup separation.

Proteins are broken down into amino acids, absorbed into the bloodstream, and affect the secretion of gastric juice. Fats are broken down into fatty acids and glycine, which are absorbed into the bloodstream and affect the secretion of sap through the blood. In the chemical phase, the separation of the lion can only be determined by the addition of food in a small stomach prepared by the Heidengain method. the syrup separated in the chemical phase is separated over a long period of time, and the fermentation capacity of the syrup is lower than that of the reflector phase.

3. Stomach movement. Laws of transfer of nutrients from the stomach to the intestine.

When the stomach is empty, the stomach walls touch each other, the cardial opening is closed, and the pyloric sphincter is open. With the absorption of food, the cardiac sphincter opens reflexively, the food is layered in the stomach, and the pyloric sphincter closes. The lining of the stomach causes the evacuation of nutrients from it.

Gastric motility is caused by the activity of muscles in the stomach wall that are longitudinal, circular, and curved.

Stomach movements are studied in two ways:

1. Tonic actions. 2. Rhythmic or peristaltic movements.

The tonic action of the stomach is caused by the contraction of the longitudinal and curved muscles in it, which, while not mixing the food, help to squeeze it into the pylorus.

The rhythmic movement of the stomach occurs when one side of the muscles located in the stomach wall contracts, resulting in the expansion of the muscles of the anterior part. This movement begins in the cardiac part of the stomach and extends to the pylorus. At this time the cardiac and fundal part of the stomach is weakly contracted and the pylorus part is strongly contracted. As a result, parts of the stomach move like a worm. This is called a worm-like or peris-talt movement. Rhythmic movements mix the food well with the gastric juice and direct it towards the intestine.

The following factors affect gastric motility:

1. The amount of hydrochloric acid. 2. How full is the stomach with nutrients. 3. Histamine. 4. Various products formed from the breakdown of proteins. 5. Ambient temperature.

Gastric motility is controlled by stray and sympathetic nerves coming from the central nervous system. While the stray nerve accelerates gastric motility, the sympathetic nerve slows down.

It shrinks independently even when there is no impulse from the central nervous system to the stomach. This movement depends on the nerve and muscle elements in the stomach wall. But these actions do not meet the ever-changing needs of the organism.

A.Yu. Yunusov observed that under the influence of heat and sunlight the secretory and motor activity of the stomach is inhibited, and then studied these conditions and observed that these activities are normalized.

Thus, in the digestion of food in the stomach, gastric motility, ie the production of various secretions from the motility and secretory glands located in its walls, as well as hormones play a key role.

TOPIC: Features of food digestion in ruminants.

Plan:

1. Features of food digestion in ruminants.
2. Digestion of nutrients in the anterior pancreas. Importance of microflora.
3. The movement of the stomach of ruminants. Digestion of nutrients in milk.

BASIC EXPRESSIONS.

Large stomach, retina, flat stomach, nectar, organic acids, fermentation, nutrients, meat, milk, herbs, coarse foods, protein, fat carbohydrates, grains, legumes, alfalfa , hay, lip, tongue, teeth, salivary glands, 25 days, reflex, 2 months, 3 months, 40-60 l, nitrogen, 30-35%, enzyme, porridge, tube, tarnov, rot, meconium , uvuz milk, starch, fiber, protein, fat, bacteria, infusoria, fungus, nerve, humoral, MNS, absorption, stray nerve, sympathetic nerve.

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1. Peculiarities of food digestion in ruminants.

Ruminants play a key role in the livestock sector, providing humans with a major source of food: meat, fat, milk and dairy products.

It is not for nothing that our people say, "In the language of the milk of cattle." Because if the animals are fed good, quality food, the product will be plentiful and of good quality. Therefore, it is necessary to know the digestive processes in the body of ruminants and to study the peculiarities of food digestion in them. The digestion of food in ruminants is different from the digestion process that takes place in the stomachs of other species.

The stomach of ruminants consists of four chambers:
1. Large abdomen. 2. Retina. 3. Flat stomach. 4.Shirdon.
The first three chambers (large, retina, and folded abdomen) are the pancreas compartments,
the fourth chamber is the chin, which is the chin or true stomach.
The specificity of the processes that take place in each of these chambers indicates the complexity of the digestive processes in ruminants.

The inner mucous membrane of the anterior gastric compartments is covered with a smooth epithelium without horns, and there are no glandular cells that secrete mucus.

The mucous membrane of the shirdon is structured similar to the stomach of a single-chambered animal, and its extra, head, and covering cells produce gastric juice. That's why it's called a real stomach.

In order to better understand the digestion of food in ruminants, it is necessary to know the process of digestion in each cell and the importance of the red decimal variety.

The large abdomen is the initial and largest part of the stomach, occupying the left side of the abdomen completely and the right side partially. Its volume is 100-300 liters in cattle and 13-23 liters in sheep and goats.

On the surface of the large abdominal mucosa there are suckers that come in 0.5 cm in young animals and 1 cm in large animals, and the function it performs corresponds to the structure of the large abdomen.

The retina is the second part and is connected by special paths with the large and folded abdomen and, accordingly, with the red tentacles. It is reminiscent of a round sack, and the inner mucous membrane consists of a net resembling a beehive. That is why it is called the retina. Its volume is 5-10 liters in cows and 1.5-2.0 liters in sheep and goats.

The abdomen is the third part, and the abdomen is connected to the abdomen, retina, and pelvis. It contains large, medium, and small leaflets. This part is well developed in cattle, its volume is 7-18 liters in cattle, 0.3-0.9 liters in sheep and goats.

The red ten novi is of great importance in the digestion of food in young animals. The retina ends in the abdominal cavity with the large abdominal corridor, from where the red duodenum begins as a continuation of the retina, and the retina reaches the fold abdomen with the base of the abdomen.

When young animals suckle their mother or drink water, the lips of the esophageal sphincter move and form a tube. The result is breast milk or drinking waterwithout falling through the tube into the large abdomen, the net passes through the abdomen into the folded abdomen and from there into the larynx. It is a reflex process in which the lips of the red right nostril move and form a tube. Receptors in the mucous membranes of the tongue and throat are affected and stimulated by breastfeeding, drinking water or milk. The excitation travels to the center of the elongated brain through the centripetal nerve, and the response produced by the excitation of the center is transmitted through the centrifugal fibers of the stray nerve to the labia minora of the red right nostril and forms a tube. . This reflex is lost when the stray nerve is cut on both sides. The excitation of the lips of the red ten novi can also occur as a conditioned reflex. This involves the crust of large hemispheres.

When young calves are fed by hand, the milk is given to them in small portions, at which time the lips of the esophageal sphincter do not move completely and do not have time to form a tube. As a result, some of the milk you drink falls into the large stomach and stays there for a long time, rots and disrupts digestive processes. For this reason, young ruminants should drink milk with caution and patience. In lambs and calves, the anterior chambers are smaller and less well developed than in the esophagus. In the first days of their lives, the anterior gastric compartments develop slowly. As the animal

grows, the red ten-layered novi lags behind the anterior chambers of the stomach in growth, and its lips become rough, unable to move, unable to form a tube. As the animal grows older, it loses its significance.

2. Digestion of food in the anterior pancreas.

Digestion of food in the large abdomen. In chewing gum, superficially chewed food in the mouth falls into the large stomach. There, the food is softened, inflated with saliva, and a variety of microorganisms are involved in the process. Microorganisms play a key role in the digestion of food in the large intestine.

They break down organic matter, synthesizing some.

It is known that food is not digested in the large abdomen of young ruminants fed only milk or liquid foods that replace it. Because they do not have microorganisms in their large stomachs. Microorganisms enter the large intestine only with coarse foods, multiply rapidly, and determine the level of digestive processes in the large abdomen. After that, the digestion of food in the large intestine takes place in the presence of microorganisms throughout life.

Infusoria, streptococci, ruminococci, succinogens and cellulose-degrading bacteria are the most important microflora of the large intestine. The type and amount of microorganisms in the large intestine depends on the type of food consumed, its composition, the age of the animal, its nutrition, productivity, and so on. 1 gram of food in the large abdomen of ruminants contains up to 10 billion bacteria and up to 1 million infusoria belonging to 20 species. 120 species of infusoria can be found in the large abdomen of animals.

Microorganisms: 1. Has a mechanical effect on food. 2. Decomposes nutrients. 3. Proteins in food, nitrogen compounds, partially fiber,
assimilates starch and others, synthesizes proteins and polysaccharides in its body.

Hence, the body of microorganisms is a source of nutrients for the animal organism. The protein of microorganisms is more valuable for the animal body than the feed protein. Because the proteins of microorganisms are close to the protein of the organism in terms of their amino acid composition. This means that microbial proteins replace proteins that are completely valuable for the animal body.

4. Due to the activity of microorganisms in the large intestine there is a process of fermentation, the formation of various gases and various volatile fatty acids.

2-3 hours after feeding the animals, the maximum amount of gases is formed, which is an average of 35 liters per hour and *gas formation up to 1000 liters per day* possible. The large intestine produces mainly carbon dioxide and methane. Mono and disaccharides produce up to 4 liters of various volatile fatty acids per day: lactic, acetic, propionic, and fatty acids. They are absorbed into the blood here and assimilated by the body.

5. Vitamins are synthesized in the large abdomen due to the activity of microorganisms. Including b group vitamins: riboflavin, thiamine, nicotinic acid, folic acid, pantothenic acid, biotin, pyridoxine, b_{12}vitamin, fat-soluble vitamin K is synthesized. Proper digestion of food in the large intestine depends on the activity of microorganisms, the formation of many useful parts of the species.

Normal large abdominal temperature is 38-41^{0}However, the pH is around 6.5-7.4, which is very favorable for the survival of microorganisms. The environment in the large abdomen is stable and it does not change easily. In this case, the continuous secretion of saliva from the parotid glands is of great importance. Various acids and other acidic substances formed in the large abdomen can alter the environment of the large abdomen and prevent microorganisms from living. It is at this point that the saliva of the parotid gland neutralizes substances with such acidic properties. In the stability of the environment in the large abdomen, it is important that nutrients are mixed and absorbed from time to time. The presence of the species of microorganisms in the large intestine necessary for digestive processes also depends on how often the composition of the ration changes. If animals are fed a particular food for a long time, only certain types of microorganisms, that is, the species involved in the digestion of the food eaten, will live in the large intestine at this time. If we suddenly switch to feeding this animal with another food, the digestion of food may be disturbed to a certain extent. This is because the animal's large abdomen does not contain the microorganisms that are required to participate in the digestion of freshly consumed food. Therefore, if it is necessary to convert ruminants from one food type to another, it is advisable to do it gradually.

In the large stomach, the undigested parts of the food are periodically returned to the mouth without being completely broken down, re-chewed, enough after being crushed and softened, the appropriately digested portion of the food is transferred from the large abdomen to the next portions of the anterior gastric compartments.

In the large intestine is digested carbohydrates, ie complex polysaccharide-fiber. Because 40-50% of the plant food eaten by ruminants is fiber. In the digestive system, 80% of the digestible fiber is broken down in the large intestine in the presence of cellulose-degrading bacteria. Because fiber is a polysaccharide, it serves as an energy source. Bacterial enzymes break down fiber into disaccharides and then into monosaccharides.

Digestion of proteins. In the large intestine, proteins, peptides, amino acids are then broken down to ammonia by enzymes of microorganisms. 40-80% of the proteins in the food are converted into protein by the body of the microorganism during digestion, the rest is passed unchanged to the intestine, where it continues to be digested.

Digestion of nutrients in the retina and flat stomach. In the large abdomen, the next portion of the properly digested food is transferred to the retina, then to the retina. Between the corridor part of the large abdomen and the retina is a fold of mucous membrane, which, when the corridor of the large abdomen is shortened, closes the hole between it and the retina to a certain extent, and only in the large abdomen r allows the passage to the abdomen. The well-digested portion of the food remains in the large abdomen, and after being properly digested, the net is transferred to the abdomen. The nutrients that fall into the retina are immediately transferred to the abdomen and the layer is squeezed between the abdominal sheets, more or less crushed, crushed. Water is perfectly absorbed here. After proper digestion in the abdomen, the food is transferred to the udder.

Chewing process. The process of chewing is important in the digestion of nutrients in the large intestine. Chewing gum is one of the unique characteristics of ruminants. As a result of chewing gum, the animals re-chew the food they have chewed from afar, grind it, and mix it with saliva. Cbecause half-chewed food is not well digested in the digestive system and is not fully absorbed by the body. Therefore, the process of chewing gum is one of the important stages of the digestive process.

*Chewing gum is a reflex process,*it occurs as follows: coarse portions of nutrients in the large abdomen stimulate the tactile receptors in the esophageal sphincter and retina, the excitation being transmitted through the corresponding afferent fibers of the sciatic nerve to the rumen return center in the elongated brain. The excitatory response produced by the center is transmitted through the efferent fibers of the stray nerve to the retina and abdomen, as well as to other organs involved in the return of food to the mouth. The return of nutrients to the mouth then begins with an additional contraction of the retina. At this point, the breathing movements stop during exhalation, the hiccups close, and you try to breathe out. The pressure at the entrance to the thorax and the greater abdomen of the esophagus decreases and the cardiac sphincter of the esophagus opens. As a result, a certain portion of the food in the large abdominal corridor moves towards the esophagus and exits the red esophagus, and the food is excreted into the mouth due to the antiperistaltic action of the esophagus. The food is compressed in the cranial sphincter of the esophagus in the larynx, and the liquid portion of the food falls back into the large abdomen. Long re-chewing of food returned to the mouth, depending on its type, takes an average of 20-60 seconds. The food is swallowed again after a good re-chewing. The return of the food to the mouth is then repeated. This means that the chewing return can be repeated. Long re-chewing of food returned to the mouth, depending on its type, takes an average of 20-60 seconds. The food is swallowed again after a good re-chewing. The return of the food to the mouth is then repeated. This means that the chewing return can be repeated. Long re-chewing of food returned to the mouth, depending on its type, takes an average of 20-60 seconds. The food is swallowed again after a good re-chewing. The return of the food to the mouth is then repeated. This means that the chewing return can be repeated.

Chewing begins 20-45 minutes in sheep and 30-70 minutes in cattle. During this time, the food in the large abdomen swells, softens, and becomes lightly chewy. Chewing gum consists of separate periods that come in succession, each of which averages 30-60 minutes. The duration of the chewing period depends on how well the various parts of the complex stomach are filled with food, the degree to which the rough parts of the food affect the large abdominal, retinal walls.

The better the food is digested and diluted in the large abdomen, the shorter the chewing time. Chewing gum also depends on external environmental influences. It is good to chew gum at night when it is quiet, when it is cool. In addition to the cerebellum, chewing is also controlled by the reticular formation of the midbrain, the hypothalamic part of the midbrain, the subcortical nuclei, and the premature zone of the cortex.

3. The movement (motility) of the stomach of ruminants.

The anterior chambers of the stomach are constantly shrinking and moving. The movement of the anterior gastric compartments begins with the retinal movement of the retina and is marked by a two-phase contraction. First, the retina is partially contracted, that is, it is reduced by half, and then it relaxes and returns to its previous position. Then it shrinks completely. The two-phase movement of the retina is repeated in 30-60 seconds. The contraction of the retina occurs simultaneously with the contraction of the red tentacle. This is followed by different parts of the large abdomen: the large abdominal corridor, then the dorsal sac, then the ventral sac, the koudodarsal, the caudoventiral sacs, respectively.

These movements mix the nutrients well and drive them out of the shrinking portion. After one or two contractions of the retina, the large abdomen is further shortened. In the same way, the abdomen shrinks. The movement of the anterior chambers of the stomach is controlled by the center of the elongated brain.

The movement of the anterior gastric compartments is also controlled by a conditioned reflex pathway.

4. Digestion of nutrients in milk.

The digestion of nutrients in the stomach is similar to the digestive processes that take place in a single-chambered stomach.

Shirdon juice is secreted by different animals in different amounts: sheep secrete 4.2 l of juice per day, cattle 40-80 l, calves 30 l. The total acidity of the medium pH 1.0-1.5 is 0.27-0.39, free hydrochloric acid is 0.23-0.29%.

The amount of enzymes in milkweed juice varies depending on the age and nutrition of the animals. Juice of young dairy animals is rich in the enzyme chymosin. As nutrients are continuously transferred from the anterior chambers of the stomach to the spleen, the spleen is continuously separated in the spleen.

Separation of sap from syrup occurs in two periods, in the reflex and neurochemical periods. The sap is also separated by a conditioned reflector pathway.

Topic: Digestion of nutrients in the intestine.

Plan:

1. Digestion of nutrients in the small intestine. Intestinal juice, pancreatic juice, the role of bile in the digestive process, their separation and administration.
2. Movement of the small intestine (motility).

Digestion of nutrients in the colon. Colon juice, its properties, separation and administration.

3. Motor movement of the colon. Formation and excretion of feces. Excretory function of the digestive system.

Basic expressions

Small and large intestine, 12 small intestine, small intestine, small intestine, appendix, small intestine, rectum, liver, bile duct, pancreas, Langerhans island, bilirubin, biliverdin, hemoglobin, status, glycocholate, tavroxolat, insulin, alkaline and acidic environment, inorganic and organic matter, entirakinase, trypsin, trypsinogen, chemotrypsin, carboxypeptidase, lipase, elastase, ribonuclease, lapeptide, dipeptidase, protamine lipase, alkaline phosphatase, chymus, evacuation, defecation, nerve and humoral regulation.

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1. Digestion of nutrients in the small intestine.

There are 2 types of intestines in humans and animals:
1. Small intestine: Includes duodenum, small and large intestines.
2. Colon: Includes appendix, small intestine, and rectum.
Foods that are thoroughly digested in the stomach are transferred to the small intestine.
In the digestion of nutrients in the small intestine:
1. Intestinal juice. 2. The juice of the pancreas. 3. Bile fluid is involved.
Nutrients that pass through the stomach are broken down in the small intestine into all its components and absorbed into the blood and lymph.

The nutrients mix well with the various fluids in the small intestine to form a homogeneous slurry. This is called chymus. The composition of chime is unchanged and maintained at the same rate. Complete excretion of chimera will inevitably lead to the destruction of the organism. In the small intestine of different animals is formed humus around 14.5–15.0 liters per 1 kg of dry food.
The average daily amount of hummus:
1. 250-300 l in cattle. 2. 28-50 l in sheep. 3. 260-265 l in grass.
4. In pigs it is around 75-80 l. 2. Intestinal juice, pancreatic juice, the participation of bile in the digestive process, their separation and administration.

Pure intestinal juice is slightly opaque, colorless, alkaline environment (pH-8.2-8.7), specific gravity is around 1,005-1,015, contains an average of 97.6% water, 0.8% proteins, 0.73% organic matter, 0.87% minerals (sodium bicarbonate and sodium chloride), mucus, cholesterol, a fluid that stores epithelial cells and enzymes.

The main enzymes in intestinal juice are:

1. Entrokinase - affects trypsinogen and procarboxypeptidase, converting them to active trypsin and carboxypeptidase.

2.Aminopeptidase 3. Dipeptidase These act on peptides and break them down into amino acids.

4. Maltase 5. Invertase These three enzymes break down various sugars.

6. Lactase 7. Lipase - breaks down fats into fatty acids and glycerin

8. Alkaline phosphatase - although not a specific enzyme for intestinal juice, phosphate separates phosphates from acid monoesters, plays an important role in the phosphorylation of carbohydrates and fats, the absorption of amino acids.

The pancreas plays an important role in the digestion of nutrients in the intestine. Pancreatic juice has a clear colorless, alkaline environment (7.8-8.0), contains 3.0 - 4.5% protein and sodium carbonate from inorganic substances.

During the day from the pancreas:

1. 6-7 l in cattle. 2. 0.36 l in sheep. 3. In pigs up to 8 liters of juice is released.

From the enzymes in the juice of the pancreas:

1. Trypsin. 2. Chymotrypsin. 3. Carboxypolypeptidase. 4. Ribonuclease.

5. Elastase. 6. Dipeptidase. 7. Protaminase. 8. Amylase. 9. Lactose.

10. Invertase. 11. There is lipase.

Bile fluid and its importance. Bile fluid is produced in the liver cells and begins to be excreted into the duodenum from time to time, i.e. 5-10 minutes after the animal has eaten, and lasts for 6-8 hours. Prior to this, bile fluid collects in the gallbladder. The gallbladder is first excreted in the intestine, then the liver.

Horses, camels, and reindeer have no gall bladder. In these animals, the gallbladder acts as an enlarged bile duct. Cbecause the digestion of nutrients in their intestines continues unabated.

Grass fluid is very important for digestion, and there are two types:

1. Liver is a fairly clear, light yellow, light green liquid. Specific gravity 1,009–1,031, pH 7.5, alkaline environment. It contains 96-99% water.

2. The gallbladder is a much thicker, darker liquid. The specific gravity is 1,026–1,048, with a pH of 6.8. contains 80-86% water and mucus separated from the mucous membrane of the bladder.

Grass also contains pigments and acids.

There are two types of grass pigments:

1. Bilirubin. 2. You know.

Bilirubin gives the grass a yellow color, biliverdin gives a green color. The color of herbivores is dark green, while the color of herbivores is reddish-yellow.

There are two types of bile acids:

1. Glycochoic acid. 2. Tauroxolate acid.

Bile acids play an important role in the absorption of fat and fatty acids through the intestinal wall into the lymph and blood. This is because fatty acids only become absorbed when they combine with bile acids to form water-soluble complex compounds.

During the day, horses weigh 6.0-7.2 liters, cattle 7.0-9.5 liters, sheep and goats 1.0-1.5 liters, pigs 2.4-3.8 liters. t is expelled into the duodenum.

The formation and excretion of bile is regularly controlled by the neurohumoral pathway and adapted to the needs of the organism.

3. Digestion of nutrients in the colon.

Nutrients are digested in the colon under the influence of enzymes in the colonic juice, enzymes supplied with food from the small intestine, and enzymes of bacteria.

Up to 15 billion bacteria are stored in 1 g of the product of the colon of herbivores, which, due to their activity:

1. Ferments carbohydrates. 2. Decomposes proteins. 3. Breaks down the fiber. 4. Different acids are formed. 5. Gases such as hydrogen sulfide, carbon dioxide, methane, hydrogen are formed. 6. As a result of the decomposition of proteins, various toxins are formed: cresol, phenol, skatole, indole, etc., which are absorbed into the blood and they are neutralized in the liver.

4. Bowel movement. The formation and removal of sediment.

It moves continuously as a result of the contraction of the muscles located longitudinally and circumferentially in the small intestinal wall. Due to these actions, nutrients are mixed with juices, transferred from one part of the intestine to another.

There are 3 types of movements in the small intestinal wall:

1. Due to the pendulum action, nutrients in the intestine mix well with digestive juices. 2. Peristaltic movement - is observed alternately, allowing the chymus to move from front to back. 3. Rhythmic movements - due to which the humus is divided into separate parts and mixed thoroughly, as well as very close to the intestinal wall.

Bowel movements:

1. By the central nervous system. 2. By conditioned reflector path. 3. Independently (automatically). 4. Managed in a humoral way.

The intestinal wall has the ability to contract independently (automatically) even when no impulses come from the central nervous system. At this time, the movement of the bowel occurs due to the excitation of the Auerbach nerve structures and muscular elements in its wall.

The movement of the colon is similar to the movement of the small intestine. Its counter-antiperistaltic action combined with peristaltic action in the caecum and small intestine mixes nutrients very well.

Nutrients affect all parts of the digestive system:

1. 94-100 hours on horses. 2. 24-36 hours in pigs. 3. In ruminants, it lasts from 14-16 hours to 14-20 days.

In the last part of the colon, water is absorbed more rapidly, the substances thicken 15-20 times, where mainly waste is collected, resulting in the formation of feces. The amount of feces excreted depends on the amount of food eaten and is more common in

herbivores than in carnivores. In particular, cows produce up to 40 kg per day, sheep up to 3 kg per day, horses produce 16-17 kg of hay and 9-10 kg of oats.

Defecation is the excretion of feces that have accumulated at the end of the rectum. Defecation is a reflex process that occurs from time to time, depending on the need, i.e. the need. Defecation in farm animals can occur when lying down, standing, or while moving.Defecation occurs 5-12 times a day in horses and 10-20 times a day in ruminants.

TOPIC: Physiology of metabolism and energy metabolism.

Plan:

1. That metabolism and energy are the meaning of life, ways to study it.
2. Protein metabolism. Completely valuable and completely worthless proteins. Nitrogen balance and equilibrium.
3.Protein minimum. Regulation of protein metabolism.

Basic phrases:

Growth, development, productivity, fertility, reproduction, decay, decomposition, shape, assimilation, dissimilation, construction, plastic, plant, animal, acute, chronic, protein, fat, carbohydrate, macro and micronutrients , observation, isolation, angiostamia, balance experiment, mediated, indirect colorimetry, respiration rate, marked, marked, blood, lymph, glycerin, fatty acids, fat, heart, kidney fat, nutritious, malnourished , Lebedov, coke oil, monosaccharide, essential fatty acid, Chervinsky, sheep fat, brown oil, barrier, water source, fat-soluble vitamins, A, D, E, K, phosphatides, sterols, poultry eggs, milk, nervous system, VNS, Claude Bernard, 4.1 k jaul, 9.3 kjaul, peas, beans, mosh, cambicorma, concentrate, fattening, nucleic acid, DNA, RNA , carnivorous, mixed feeder, sheep, goat,cattle.

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1.The occurrence of the main vital processes of the body is based on the process of continuous and uninterrupted exchange of substances and energy in the body. Metabolism and energy metabolism are the main factors that determine the vitality of an organism. Metabolism and energy exchange ensure the growth, development, formal and functional dependence of the organism on the basis of life. All vital processes in the body are based on the metabolism of matter and energy.

The metabolism of matter and energy takes place in all branches of nature. In living nature, the organism grows and provides development, productivity and fertility. In the dead nature, the processes of decomposition, disintegration, and transition of the organism from one form to another are ensured.

Assimilation or anabolism is the process by which an organism assimilates substances that are necessary for different organisms in accordance with its own characteristics. 'is assimilated in an adapted state for sections and cells.

Dissimilation or catabolism is the inequality or separation of various substances from organs in modified forms.

The processes of assimilation and dissimilation are complementary organic phenomena that provide the biological basis of an organism.

2. Protein is the life of an organism. Without protein, there is no life in the body. Protein is taken into the body in the form of various proteins along with various nutrients. The main sources of protein are plant and animal life. The protein content has a complex structure and differs in its nitrogen storage. Proteins differ in the storage of amino acids in their composition, and proteins are of two types. Full-value proteins contain all the essential amino acids. The source of such proteins is animal product and partly plant product. Completely worthless proteins — such proteins lack some essential amino acids. When proteins enter the digestive system with food, they are broken down by proteolytic enzymes to form various amino acids, which are absorbed into the bloodstream. It is distributed throughout the body through the blood, travels to organs, tissues and systems and serves as a building material.

3. To meet the protein needs of the body, each organism has its own specific protein requirement, and the amount of protein that is sufficient only as a plastic material is called the protein minimum. The minimum protein is determined by the live weight of the organism, for carnivorous and mixed-feeding organisms requires 1 gram or 1.2 grams per 1 kg of live weight, for a herbivore 0.6-0.8 grams of protein per 1 kg of body weight. is done. The level of this minimum protein varies depending on the living conditions of the organism, the work it performs, its physiological condition and productivity, and an average of around 25-30% of protein is added. Organisms that work hard, organisms that recover after a chronic illness,

Given that the bulk of the protein is excreted as nitrogen and only nitrogen is present, the nitrogen balance is checked to determine the level of protein metabolism. Nitrogen balance can be both positive and negative and equal.

Agar When the amount of nitrogen introduced into the body exceeds the amount of nitrogen excreted from the body, it is called a positive nitrogen balance, and vice versa, when the amount of nitrogen excreted exceeds the amount of nitrogen introduced, it is called negative nitrogen balance. When the nitrogen balance in the body is equal, the protein ingested with food is used to increase the growth, development and productivity of the organism. To check the nitrogen balance in the laboratory, the animals are kept in a special balance. The amount of nitrogen introduced into the body is checked by the composition of the food included in the diet. Nitrogen excreted from the body is detected in urine and feces.

Nitrogen is rapidly excreted as sweat, urine, hippuric acid, urea. It is necessary to determine the amount of nitrogen in these products. Protein metabolism is controlled by the nervous and humoral systems. In the hypothalamus part of the midbrain is the center that regulates protein metabolism, its gray matter accelerates the breakdown of protein from the action of the nuclei in the part and accelerates the excretion of nitrogen in the urine. Other parts of the hypothalamus may also have a center that inhibits protein metabolism. The nervous system alters the metabolism of proteins by enhancing the release of thyroxine, the somatotropic hormone of the pituitary gland from the humoral system, ie the thyroid gland. The effects of protein metabolism on the cerebral cortex have been studied using the false feeding method.

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Plan:

1. That metabolism and energy are the meaning of life, ways to study it.
2. Protein metabolism. Completely valuable and completely worthless proteins. Nitrogen balance and equilibrium.
3.Protein minimum. Regulation of protein metabolism.

Basic phrases:

Growth, development, productivity, fertility, reproduction, decay, decomposition, shape, assimilation, dissimilation, construction, plastic, plant, animal, acute, chronic, protein, fat, carbohydrate, macro and micronutrients , observation, isolation, angiostamia, balance experiment, mediated, indirect colorimetry, respiration rate, marked, marked, blood, lymph, glycerin, fatty acids, fat, heart, kidney fat, nutritious, malnourished , Lebedov, coke oil, monosaccharide, essential fatty acid, Chervinsky, sheep fat, brown oil, barrier, water source, fat-soluble vitamins, A, D, E, K, phosphatides, sterols, poultry eggs, milk, nervous system, VNS, Claude Bernard, 4.1 k jaul, 9.3 kjaul, peas, beans, mosh, cambicorma, concentrate, fattening, nucleic acid, DNA, RNA , carnivorous, mixed feeder, sheep, goat,cattle.

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1.The occurrence of the main vital processes of the body is based on the process of continuous and uninterrupted exchange of substances and energy in the body. Metabolism and energy metabolism are the main factors that determine the vitality of an organism. Metabolism and energy exchange ensure the growth, development, formal and functional dependence of the organism on the basis of life. All vital processes in the body are based on the metabolism of matter and energy.

The metabolism of matter and energy takes place in all branches of nature. In living nature, the organism grows and provides development, productivity and fertility. In the dead nature, the processes of decomposition, disintegration, and transition of the organism from one form to another are ensured.

Assimilation or anabolism is the process by which an organism assimilates substances that are necessary for different organisms in accordance with its own characteristics. 'is assimilated in an adapted state for sections and cells.

Dissimilation or catabolism is the inequality or separation of various substances from organs in modified forms.

The processes of assimilation and dissimilation are complementary organic phenomena that provide the biological basis of an organism.

2. Protein is the life of an organism. Without protein, there is no life in the body. Protein is taken into the body in the form of various proteins along with various nutrients. The main sources of protein are plant and animal life. The protein content has a complex structure and differs in its nitrogen storage. Proteins differ in the storage of amino acids in their composition, and proteins are of two types. Full-value proteins

contain all the essential amino acids. The source of such proteins is animal product and partly plant product. Completely worthless proteins — such proteins lack some essential amino acids. When proteins enter the digestive system with food, they are broken down by proteolytic enzymes to form various amino acids, which are absorbed into the bloodstream. It is distributed throughout the body through the blood, travels to organs, tissues and systems and serves as a building material.

3. To meet the protein needs of the body, each organism has its own specific protein requirement, and the amount of protein that is sufficient only as a plastic material is called the protein minimum. The minimum protein is determined by the live weight of the organism, for carnivorous and mixed-feeding organisms requires 1 gram or 1.2 grams per 1 kg of live weight, for a herbivore 0.6-0.8 grams of protein per 1 kg of body weight. is done. The level of this minimum protein varies depending on the living conditions of the organism, the work it performs, its physiological condition and productivity, and an average of around 25-30% of protein is added. Organisms that work hard, organisms that recover after a chronic illness,

Given that the bulk of the protein is excreted as nitrogen and only nitrogen is present, the nitrogen balance is checked to determine the level of protein metabolism. Nitrogen balance can be both positive and negative and equal.

Agar When the amount of nitrogen introduced into the body exceeds the amount of nitrogen excreted from the body, it is called a positive nitrogen balance, and vice versa, when the amount of nitrogen excreted exceeds the amount of nitrogen introduced, it is called negative nitrogen balance. When the nitrogen balance in the body is equal, the protein ingested with food is used to increase the growth, development and productivity of the organism. To check the nitrogen balance in the laboratory, the animals are kept in a special balance. The amount of nitrogen introduced into the body is checked by the composition of the food included in the diet. Nitrogen excreted from the body is detected in urine and feces.

Nitrogen is rapidly excreted as sweat, urine, hippuric acid, urea. It is necessary to determine the amount of nitrogen in these products. Protein metabolism is controlled by the nervous and humoral systems. In the hypothalamus part of the midbrain is the center that regulates protein metabolism, its gray matter accelerates the breakdown of protein from the action of the nuclei in the part and accelerates the excretion of nitrogen in the urine. Other parts of the hypothalamus may also have a center that inhibits protein metabolism. The nervous system alters the metabolism of proteins by enhancing the release of thyroxine, the somatotropic hormone of the pituitary gland from the humoral system, ie the thyroid gland. The effects of protein metabolism on the cerebral cortex have been studied using the false feeding method.

TOPIC: Energy and heat exchange physiology.

Plan :

1. Energy exchange, essence and significance. Instrumental and instrumental calorimetry. Breathing coefficient.

2. The effect of various factors on metabolism. Heat metabolism. Poikilothermic and homothermic animals.
3. Heat generation and its transfer.
4. Body temperature of animals and its control-thermoregulation.

Basic expressions

Energy, heat, fat, protein, carbohydrates, urine, waste, milk, fermentation processes, gases, mechanical, physical, chemical, osmotic, synthesis, water, food, calorimetric pressure, vositalikalorimetry, medium calorimetry, AALixachev, VVPashutin , Etuoter, F. Benedict, breath coefficient, SO_2, O2, caloric equivalent, respiratory chamber, respirator, gasometer, isotherm, homothermic animals, poikilotherm animals, exothermic reactions, thermoregulation.

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1. The essence and importance of energy exchange.

Metabolism in the body is accompanied by the release and absorption of energy. The rate at which metabolism occurs and the rate at which it occurs is determined by the amount of energy expended on it.

The body takes into account the nutrients it consumes energy. Energy is released when complex organic compounds in foods - fats, carbohydrates and proteins - are broken down.

The potential energy released is:

1. Not everything is spent for the realization of vital processes taking place in the body. 2. Part of it is used to heat urine, feces, milk, gases produced by fermentation processes, as well as drinking water and food consumed. 3. The main part is used for the occurrence of vital processes in the body in the form of mechanical, chemical, electrical, osmotic energy.

All kinds of energy used for vital processes in the body are converted into heat energy and transferred to the environment outside the body.

The body releases heat as follows:

9.3 kcal when 1 g of fat is oxidized. 4.1 kcal when 1 g of carbohydrate is oxidized.

4.1 kcal per 1 g of protein oxidized

To know the amount of energy produced in the body is the amount of heat released from it per unit time.

The following methods are used to calculate the amount of heat released from the body per unit time:

1. Toolless calorimetry. 2. Instrumental calorimetry.2. Immediate and indirect calorimetry. Breathing coefficient.

In indirect calorimetry, the amount of heat released in the body per unit time is measured. To do this, special biocalorimeters are used and the animal to be examined is placed inside the biocalorimeter. The size of biocameras varies depending on the size of the animal. Such calorimeters were first developed in Russia in 1893 by AA Likhachev and VV Pashutin, and later in 1898 in the United States by Etuoter and F. Benedict.

Determining the body's energy expenditure using calorimeters created so far is much more complicated and labor-intensive. Therefore, indirect calorimetry, ie the method of studying gas exchange, is more widely used.

The method of indirect calorimetry measures the amount of oxygen taken into the body and the amount of carbon dioxide released from it.

The amount of heat generated when the body receives 1 liter of oxygen or releases 1 liter of carbon dioxide is called the caloric equivalent of oxygen or carbon dioxide.

Respiratory chambers are used to determine the amount of oxygen received or carbon dioxide released.

There are two types of respiratory cameras:

1. Sealed cameras. 2. Cameras operating in contact with atmospheric air.

Data obtained using indirect and indirect calorimetry methods are not always accurate.

The respiration rate is the ratio of the volume of carbon dioxide excreted from the body per unit time to the volume of oxygen absorbed.

This is as follows NK = SO2 O2 is used.

The respiration rate varies depending on the type of substances that are broken down in the body.

If the nutrients in the body are broken down:

1. If carbohydrates are oxidized, the respiration rate is 1.

2. Yif the whites are oxidized, the respiration coefficient is 0.7.

3. If the proteins are oxidized, the respiration coefficient is 0.8.

Fats, carbohydrates and proteins are not broken down separately in the body. On the contrary, all of these substances are broken down in a certain amount at the same time.

3. The effect of various factors on metabolism.

To the metabolic process in the body:

1. Type, age, breed, sex, weight, size, productivity and nutrition of animals. 2. The state of the organism. 3. The season of the year. 4. The period of the day.

5. Ambient temperature 6. Physical work. 7. The body is affected by starvation and other factors.

Metabolism is several times faster in small-bodied animals than in large-bodied animals, and several times faster in mice and rats than in horses and cattle. Metabolism is 10-26% faster in bulls than in cows, 20% faster in females than in males, and 20-30% faster in hens than in chickens. YIn animals, the metabolism is faster and slows down as the animal gets older.

Metabolism is faster in winter than in summer, during the day than at night. The more milk the cows give, the higher the fat content of the milk, the faster the metabolism.

When muscles work, energy expenditure increases and metabolism speeds up. The harder the muscle works, the higher the energy expenditure. For example, for walking 1 m, dogs have 0.58 kcal per 1 kg of live weight when walking on a flat road, 7.26 kcal per 1 kg of live weight when climbing a mountain, and 0.59 kcal per sheep when walking on a flat road. and 6.45 kcal energy expenditure during climbing.

When an animal is hungry, its metabolism slows down. In this case, the body meets its energy needs first at the expense of carbohydrates, then fats and proteins.

4. Heat exchange. Heat generation and its transfer.

The heat generated by the metabolism gives the body heat.

The body temperature of highly developed, warm-blooded animals and humans is always kept at a certain level, regardless of changes in the ambient temperature in which they live. This constant of body temperature is called isotherm.

Isotherm is unique to warm-blooded animals.

The body temperature of cold-blooded - poikilotherm animals varies slightly depending on the ambient temperature.

The systems that control heat exchange in homothermic animals are highly developed and perfected. That is why their body temperature is always the same, and finally in a small range1°varies around. Factors such as the time of day, the age of the animal, sex, nutrition, condition of the organism, the intensity of metabolism affect the temperature fluctuations in such a small range.

In poikilothermic animals, however, the systems that control heat exchange are not as highly developed as in homothermic animals. Therefore, their body temperature varies slightly depending on the ambient temperature.The rate of heat production in all

organs of the body is not the same. Because the intensity of metabolism in different organs varies to some extent. In organs such as the liver and kidneys, where metabolism is fast, heat is generated several times more than in bones, joints, and connective tissues, where metabolism is slow.

Heat production increases during physical activity, nutrition, and active movement.

The heat generated is continuously transferred to the external environment. The subcutaneous fat layer, wool, feathers have some resistance to heat transfer.

Heat transfer from the body is as follows: 1. Heating of the exhaled air. 2. Radiation. 3. Convection is the scattering of heat into the surrounding air. 4. Evaporation of sweat. 5. The surface of an animal's body is exposed in ways that scatter invisible infrared rays.

For example, 580 kcal of heat loss from the body has been proven when 1 liter of sweat fluid evaporates. Even with saliva, a small amount of heat is lost.

5. Animal body temperature and its regulation thermoregulation.

Body temperature of agricultural and domestic animals belonging to different species is 37-43 ℃range. These animals have a body temperature of 24℃ fall below or vice versa 44 ℃ is dangerous for life.

Body temperature of animals
(when measured from the rectum)

Animals type	Body temperature (in degrees)	Animal type	Body temperature (in degrees)
Ot	37.5-38.5	Cat	38.0-39.5
Cattle	37.5-39.5	Rabbit	38.5-39.5
Put it down	38.8-40.0	Goose	40.0-41.0
Goat	38.5-40.0	Chicken	40.5-42.0
Pig	38.0-40.0	Duck	41.0-43.0
It	37.5-39.0	Pigeon	41.0-43.0

Only when the amount of heat lost in the body is equal to the amount of heat lost can the body temperature remain constant.

Depending on the physiological mechanism, there are two types:

1. Chemical thermoregulation. 2. Physical thermoregulation is different.

Chemical thermoregulation occurs by accelerating or slowing down the formation of heat in the body.

Physical thermoregulation, on the other hand, is accomplished by altering heat transfer from the body.

For example, when the ambient temperature decreases, metabolism speeds up. As a result, heat generation increases. This is because under the influence of cold, the cold-sensitive receptors of the muscles are affected, the muscles begin to vibrate reflexively and their energy expenditure increases. This leads to an acceleration of metabolism.

In addition to muscles, the liver and kidneys also play a role in chemical thermoregulation. Cbecause under the influence of cold the metabolism in these organs is accelerated several times.

The nervous system plays a leading role in thermoregulation. In the hypothalamus, there is a center in the hypothalamus that controls heat exchange. This center is evoked by appropriate reflexive and humoral impressions. The impulse generated by the action of the corresponding receptors is transmitted to the center, the excitation of the center causes a change in the corresponding processes - oxidative processes in muscles and organs, vascular capacity, sweat secretion. As a result, heat generation and transmission also change.

Topic: Kidney physiology.

Plan :

1. Separation bodies and their importance.
2. Physiology of the kidneys. Urine formation. The concept of primary and final urine. Stepped and non-stepped substances.
3. Management of urine production. The concept of diuresis. Management of urinary excretion.

Basic expressions

Kidneys, sweat glands, lungs, intestines, metabolism, protein, fat, carbohydrates, water, salts, toxins, nephron, Bauman-Shumlyansky capsule, Malpighian ball, Genle's cortex, twisted tubules, filtration, reabsorption, step, stepless, nerve, humoral, stray nerve, sympathetic nerve, elongated brain, pituitary, hypothalamus, adrenaline, aldesterone, thyroxine, parathyroid hormone, glucosuria, hematuria, albuminuria, hemoglobinuria, osmotic pressure, oncotic pressure, renal factors, extrarenal factors, diuresis.

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1. Separation bodies and their importance.

The end products of metabolism in the body are excreted through the digestive organs.

Separation bodies: 1. Kidneys. 2. Sweat glands. 3.Lungs. 4. Intestines included.

The sweat glands excrete some of the excess water and salts, and a small amount of the products of protein metabolism.

Water, carbon dioxide, and some volatile substances are excreted through the lungs.

Intestinal orgasm removes heavy metal salts, various waste substances, products of bile pigments from the body.

The main excretory organ of the body is the kidneys. Cbecause most of the waste products formed in the body are excreted through them.

Through the kidneys, water, the products of protein metabolism - urea, uric acid, creatinine, minerals and other wastes are excreted in the urine.

Separation organs are of great importance for the organism. Chunki, due to the activities of the separation bodies:

1. The body is free from various toxins that are unnecessary and toxic to itself.
2. The composition and properties of the internal environment of the organism are always maintained to a certain extent.
3. Stability of ionic content of blood, lymph, tissue fluids is provided.
4. Stability of osmotic pressure of blood, lymph, tissue fluids is provided.
5. Excess heat is released from the body.

This means that the digestive organs are also involved in thermoregulation.

2. Physiology of the kidneys. Its the formation of urine management

Physiology of the kidneys. Animal and human kidneys are a pair of organs located in the lumbar region of the body.

Each kidney consists of two layers.

1. Skin layer. 2. The core layer.

The cortex of the kidney is made up of nephrons, and all the processes of urine formation take place in it.

Nephrons were first identified in 1872 by Sstudied by Humlyansky. Each nephron, taken separately, begins with a small double-walled Bauman-Shumlyansky capsule, in which the capillaries are intertwined, that is, the Malpighian ball.

The canal begins in the space between the walls of the double-walled capsule. The tube is bent two or three times near the capsule. This is called a first-order torsion channel. This duct straightens and narrows at the boundary between the cortex and the nucleus accumbens of the kidney, and descends into the nucleus accumbens. Eventually, Genle forms a loop and the bark returns to the floor. Kanalcha the bark

is bent two or three more times on the floor. This is called a second-order torsion tube. The second-order screw is poured into the collecting channel.

The collecting ducts of several nephrons join to form a common output duct. The common excretory duct passes through the nucleus accumbens and into the renal pelvis.

The amount of nephrons in both kidneys of animals belonging to different species varies:

1. In cattle 8 mln. close to 2. C1400,000 in pigs. 3. 1,000,000 in sheep. 4. There are 700,000. 5. A frog has 2,000 nephrons. Humans have 4,000,000 nephrons in both kidneys.

Urine formation. Urine is formed in the kidneys, nephrons. The basic theory that explains the formation of urine in nephrons is the theory of filtration-reabsorption.

According to this theory, there are two phases in the formation of urine:

1. Filtration phase. 2. There is a phase of reabsorption (reabsorption).

During the filtration phase, soluble substances in the blood plasma flowing from the capillaries of the ball are absorbed into the Bauman-Shumlyansky capsule and filtered. The presence of high blood pressure (90 mm Hg) in the capillaries of the ball has a significant effect on the performance of filtration. The oncotic pressure (20-30 mm Hg) generated by the plasma proteins resists filtration.

As a result of filtration, a certain amount of fluid is absorbed from the capillaries into the capsule and the tube. Primary,*provizor* urine is formed.

It differs from blood plasma in the absence of proteins in the primary urine.

In the reabsorption phase, the primary urine flows through the tubules and is reabsorbed. As a result, most of the substances in it are reabsorbed into the bloodstream, eventually forming a slightly final, true urine that is "thickened."

Due to reabsorption, glucose and amino acids in the primary urine are completely reabsorbed.

Stepped and non-stepped substances. Substances are divided into 2 depending on their reabsorption:

1. Stepped substances. 2. Non-step substances.

Non-step substances are substances that are not reabsorbed, regardless of their concentration in plasma, or are reabsorbed in very small amounts and excreted in the final urine. These substances include urea, creatinine, sulfates. They have a positive effect on the amount of urine excreted.

Stepwise substances are substances that are not found in the urine or are found in very small amounts, usually due to complete reabsorption. These include glucose, amino acids, various ions.

Management of urine production. The urinary activity of the kidneys is controlled by the nervous and humoral systems.

The kidneys are supplied with stray and sympathetic nerve fibers of the nervous system.

When a stray nerve fiber is exposed, water excretion accelerates, resulting in an increase in urine volume and a decrease in nitrogen content.

When the sympathetic nerve fiber is affected, the amount of sodium chloride in the urine decreases and increases slightly.

Different parts of the central nervous system are involved in the management of renal function. Urinary excretion is increased when the bottom of the fourth cerebral ventricle in the medulla oblongata is affected by a gray lump in the midbrain.

Urine production is also controlled under cortical control, i.e. by a conditioned reflex pathway. For example, pain causes a sharp decrease in urinary excretion (painful anuria).

Humoral factors also play an important role in the management of renal function.

The following have a major effect on urine production: 1. Antidiuretic hormone of the pituitary gland. 2. Thyroxine hormone of the thyroid gland. 3. Adrenaline, aldosterone hormones of the adrenal glands. 4. Urea, minerals in the blood (especially sodium chloride).

Antidiuretic hormone dramatically reduces the amount of urine excreted.

Thyroxine reduces the binding of water and salts to tissues, enhances their absorption into the bloodstream, and promotes urine formation.

Reduces adrenaline-urinary excretion.

Aldosterone affects the tubular epithelium and helps reabsorb sodium.

4. The composition, amount and properties of urine.

The composition of animal urine consists of 2 parts:1. Water - 96%. 2. Dry matter 4%.

The dry matter is composed of organic and inorganic substances.

Organic substances in urine:

1. Waste substances formed as a result of protein breakdown: urea (urea), uric acid, purine bases (guanine, adenine, hypoxanthine, xanthine) and creatine.

2. Urea nitrogen. 3. Indoxyl sulfate (indicane) skatoxyl sulfate, oxyphenyl acetate and oxyphenyl propionate acids, formed by the decomposition of proteins in the intestine and neutralized by sulfuric acid in the liver. 4. Urochrome and urobilin formed in the intestine from bile pigments. 5. Contains hippuric acid, which is synthesized in the kidneys.

Inorganic substances in urine:

1. Sodium salts. 2. Potassium salts. 3. Sulfates. 4. Phosphates are released.

Protein and sugar are usually absent in the urine of a healthy animal.

However, the presence of significant amounts of protein in the urine indicates the onset of the disease in the body.

Albuminuria is the excretion of proteins in the urine.

Glucosuria is the excretion of glucose in the urine. It is observed in some physiological conditions (as a result of nervousness, agitation of the sympathetic nervous system, excessive release of adrenaline) and in diabetes mellitus.

Hematuria is defined as bleeding in the urine.

It is observed in various kidney diseases, urinary tract injuries and other cases.

Hemoglobinuria is the excretion of hemoglobin in the urine.

It is observed during a number of infectious and parasitic diseases of animals.

Physicochemical properties of urine:

1. Type of animal. 2. The sex of the animals. 3. The nutritional nature of animals.

4. The physical performance of animals. 5. Animals depend on the general condition of the organism.

The color of urine depends on the amount of pigments (urochrome, urobilin, food pigments consumed), the amount of urine excreted, the concentration. The urine of most animals is clear, yellowish in color.

If the urine is expelled quickly and a lot, the color will be pale yellow.

As the amount of urine excreted by the animal decreases due to excessive sweating and its concentration increases, its color becomes darker yellow. It seems a bit blurry because calcium carbonate crystals are more common in the urine of ungulates.

The specific gravity of the urine of different animals differs from each other:

1. In horses 1,040 ha. 2. 1,032 ha in cattle. 3. In sheep 1,042 ha.

4. C1,018 ha in pigs 5. 1,025 ha in dogs. 6. 1,035 ha in cats.

7. In rabbits, it is equal to 1,015.

Urine environment depends on the type and composition of food consumed by the animal, the state of the organism:

1. The environment of the urine of herbivores pH = 8.7-7.1, alkaline.

2. The environment of the urine of carnivorous animals pH = 5.7-7.0, slightly acidic.

Cbecause they consume large amounts of protein.

3. The urinary environment of animals that consume mixed foods is alkaline or acidic.

Urination. Urine is formed continuously in the kidneys and is poured into the cup. When the renal cup fills with urine, it contracts, eventually pushing urine through the internal urinary tract (ureters) into the bladder (bladder).

Urination is a reflex act. The activity of the bladder, sphincters is controlled by sympathetic and parasympathetic nerve fibers.

When the sympathetic nerve is stimulated, the bladder dilates, the sphincters close tightly. At this time, conditions are created for urine to accumulate in the bladder. When the parasympathetic nerve fiber is stimulated, the tone of the bladder increases, resulting in its contraction, the sphincters relax and open. As a result, there is an opportunity for urination.

The center that controls urination is located in the lumbar region of the spine.

The urinary reflex occurs as follows: After the bladder is full, the receptors on the walls are stimulated. The resulting impulses urinateis transmitted to the center, resulting in the center being moved. The response is transmitted to the bladder via the parasympathetic nerve fiber, and the bladder contracts and the sphincters relax, so urine is expelled.

The center in the spinal cord that controls urination works under the control of the cortex of the cerebellum, midbrain, and cerebral hemispheres. This is manifested by the suspension or intensification of urination, i.e., voluntary, involuntary urination.

5. The concept of diuresis. Diuresis is the amount of urine that is expelled at one time.

Animals average daily:

1. Herbs 2.5 l.
2. Cattle 5-12 l.
3. Put 1-1.5 l.
4. Dogs excrete 0.5-1.0 liters of urine.

The amount of diuresis:

1. Type of animal
2. The amount of water or other liquids drunk.
3. Type and composition of foods consumed.
4. The state of the organism.
5. Climate.
6. The period of the day and a number of other factors are affected.

The body is more active during the day than at night. The amount of diuresis also increases when you drink a lot of water or consume excessive amounts of wet foods. Conversely, if the animal sweats a lot (when doing heavy physical work), diuresis is somewhat reduced.

Topic: Skin physiology

Plan:

1. The structure, functions and importance of the skin. Sweat glands. Separation and administration of sweat fluid.
2. Sebaceous glands of the skin, temperature and environment. Skin pigmentation.
3. Seasonal changes in the skin. Throwing and its importance.

BASIC EXPRESSIONS.

Skin, epidermis, dermis, subcutaneous tissue, thermoregulation, depot organ, analyzer, receptors, heat-sensitive Ruffin bodies, cold-sensitive Krause tubes, tactile-sensitive Meissner bodies and Markel cells, pressure-sensitive Fatter-Pachchini bodies, sweat and fat glands, sweat fluid, secretory epithelium, sweating, calcium salts, phosphates, sulfates, proteins, urea, uric acid, creatinine, ammonia, volatile fatty acids, vitamins, sympathetic and sciatic nerves, holocrine, giraffe, pigments, melanin, tyrosine , pigmentation, wool, wool fiber, wool.

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I. STRUCTURE, FUNCTION AND IMPORTANCE OF SKIN.

Teri -is a very important organ for life, covering the surface of the organism. This is because the skin is constantly exposed to various influences of the external environment and protects the body from them. The skin is a mobile, elastic organ that connects through the subcutaneous tissue without directly contacting the muscles.

THE SKIN IS MADE OF 3 FLOORS:

1. Epidermis. 2. Derma-specific or genuine skin. 3. Subcutaneous tissue.

Epidermisis the outer layer of the skin, which consists of several rows of flat epithelium. The upper part of the epidermis is gradually branched, frozen and continuously separated, and regenerates due to the inner granular - malpighian layer. There are lymphatic vessels in this layer, but no blood vessels. The epidermis and dermis are connected to each other by a thin membrane through which the metabolism between the two layers takes place. The dermis is the main part of the skin, it is a layer rich in blood vessels, nerve and muscle fibers, sweat and sebaceous glands, wool glands. The thickness of the skin depends on how developed the dermis is.

AVERAGE SKIN THICKNESS:

2.7-4.6 mm in cattle; 1-5 mm in horses; in sheep it is 0.7-3.1 mm.

The thickness of the dermis is different in different types of animal skin and not the same in different parts of the same animal skin, depending on the age, sex, breed and climatic conditions of the animal.The task of participating in metabolism.Because the vital processes that take place in the body, their changes are also reflected in the skin to one degree or another. Therefore, depending on the condition of the hair on the skin, its luster, it is possible to think about the condition of the organism. Sweating function - due to which the skin secretes sweat fluid.

II. SEPARATION AND MANAGEMENT OF SWEAT FLUID.

The secretion of sweat fluid is called sweating. Sweating is important. Because due to sweating, the body transfers a certain amount of heat, removes excess water and mineral salts, some of the unwanted substances, and regulates the osmoregulation of the internal environment.

Sweat fluid is continuously formed and secreted in the secretory epithelium of the sweat glands. Their excretory pathways open to the skin surface.

Sweat glands are spread over the entire surface of the body. The amount of sweat glands is not the same in different parts of an individual’s skin and in the skin of

different types of animals. For example, sweat glands are more common in the head of cattle and pigs than in other parts. On the surface of 1 cm2 of skincattle have up to 2,500, horses, up to 1,500, sheep, up to 500 sweat glands. Sweat glands are extremely rare in rodents and absent in wild animals.

A person has about 3 million sweat glands, including 400-500 in the arms, legs, palms and armpits.

Sweating is not the same in different animals. This is because sweat secretion is influenced by factors such as the type of animal, breed, condition of the organism, climatic conditions.

For example, in one day: horses 2.5-3 liters, cattle 4-5 liters, people 1.5-2.5 liters of sweat.

Sweat fluidis a saline, aqueous liquid with a weak alkaline environment (pH = 7.5–8.5) and a specific gravity of 1,005–1,021. Usually the sweat fluid mixes with the skin oil, resulting in a slightly acidic reaction.

Sweat fluidhas a weakly alkaline environment (pH = 7.5–8.5) and is a saline, aqueous liquid with a specific gravity of 1,005–1,021. Usually the sweat fluid mixes with the skin oil, resulting in a slightly acidic reaction. sodium chloride potassium chloride calcium salts phosphates sulfates Proteins urea uric acid creatinine ammonia volatile fatty acids pigments vitamins and other substances. Sweat secretion is controlled by neuro-humoral pathways.Sympathetic and sciatic nerve fibers are the secretory nerves of the sweat glands. Sweating is a reflex process. External ambient temperature plays a key role in the occurrence of this reflex. This is because when the temperature rises, the thermoreceptors in the skin are affected and stimulated. The resulting excitation impulses travel to the center of the sweat glands located in the lateral branches of the spinal cord through nerve fibers that tend to the center and excite it. The resulting response impulses are transmitted to the nodes of the sympathetic nervous system. The excitation originates from these nodes and travels to the sweat glands through the secretory (centrifugal) nerves and alters their activity. As a result, sweat is released.

In addition to the spinal cord, the medulla oblongata also has a center that controls sweat secretion. This center works under the control of a center that controls heat exchange in the midbrain. Increased sweating in a variety of emotional states suggests that the cerebral cortex is involved in the management of sweat secretion.

III. OIL GLANDS AND SKIN PIGMENTATION OF THE SKIN.

In addition to sweat glands, the skin also has sebaceous glands, which produce sebum. The sebaceous glands are superficially located and their pathways open into the wool sacs. The wall of the sebaceous glands consists of multilayered epithelium, which grows close to the glandular pathway, turns into fat and dies. Skin oil is composed of complex esters of unsaturated glycerin and cholesterol formed with fatty acids. Skin oil is broken down in the presence of acids in the sweat fluid. The result is a variety of volatile fatty acids that emit a distinctive odor. Skin oil is of great importance for the body. Because 1. Fetal skin oil: prevents the absorption of amniotic fluid into the body; 2. Skin oilmakes the skin smooth and facilitates childbirth. It lubricates and protects the epidermis of animal skin, such skin is less injured and becomes elastic; lubricates the

hair, making them shiny, soft and elastic. Sheep skin oil is mixed with sweat fluid and mixed. This is called giraffe (or fat-sweat compound).

Giropot is of great importance in the good growth of wool, in its proper, fibrous placement, in its durability, in ensuring that it is not contaminated. Giraffes are more common in fine-wool sheep than in coarse-wool sheep (merinos)in sheep 7-30% of the total weight of wool is giraffe). Pure giraffe contains lanolin, cholesterol and isocholesterol, which are used in perfumery and pharmaceuticals in the preparation of various ointments (ointments). The activity of the sebaceous glands is influenced by the nutrition of animals, the condition of the organism and other factors.

Skin temperaturebelow body temperature. Different animals also have different temperatures in different parts of an individual's skin. For example, skin temperature:

in cattle 32-35o in horses 27.5-30.2o in renos sheep around 30.4-33.7o.

The temperature of the skin of the feet is higher than the temperature of the skin of the feet. Skin temperature can vary around 5-6o. But higher variability is a disease-specific feature. carbohydrates are broken down a lot due to the high production of lactic acid during physical activity; elastic collagen, carotene, glutamine, vitamin D and glycogen are synthesized; lysozyme enzyme with bactericidal properties is formed; immune cells are formed; found in various protein fractions, urea, creatinine, uric acid, amino acids, pigments. Skin pigmentation. The color of the skin and wool depends on the pigments in it. Pigments protect the skin and the body in general, that is, they absorb the short-wave rays of the sun and eliminate their harmful effects on the body.

There are basically two types of pigment in the skin:

2. Melanin - a black pigment synthesized from the amino acid tyrosine.

1. Hemosiderin- a red pigment formed by the breakdown of hemoglobin and found in the nucleus of wool fibers. The quinine in melanin gives it a black color. Melanin occurs between the malpighian layer of the dermis and the epidermis and continuously passes into the malpighian layer. The formation of melanin depends on the presence of ascorbic acid, the amount of sulfhydryl (Sh) group, the state of the endocrine glands. For example, if the adrenal glands are removed, skin pigmentation increases. Therefore, if the activity of the cortical layer of this gland decreases, people develop "bronze" disease. Skin pigmentation decreases as thyroid activity increases. Skin pigmentation is also affected by the activity of the pituitary and gonads, as well as the ambient temperature.

IV. SEASONAL CHANGES AND SKIN IN THE SKIN.

Jun is a leather product. The skin of most animals is covered with wool, the length and density of which depends on the type, breed, diet, living conditions and other factors of the animal. For example, the hair of animals living in cold climates is longer and denser than in warm climates. According to Troitsky, 1 cm^2On the surface of the skin there are on average 700 wool fibers in horses, 600-1200 in chinchilla rabbits, 5000 in Romanov sheep, up to 8000 in merino sheep. The fur of young animals grows faster and better than that of older ones, and in summer than in autumn and winter. For the good growth of wool, the food consumed must have enough proteins, especially the amino acid cystine. The growth of wool is also affected by the endocrine glands. For example, if the thyroid gland is removed, the growth of wool slows down and its quality

deteriorates. The fur of the animals gradually separates from the epithelium of the wool sac. After a certain time, the animal jumps.

Pay it is said that the animal completely replaces the old wool cover with a new wool cover. There are three types of shedding in warm-blooded animals

1. Continuous throwing.2. Periodic or seasonal throwing. 3. Age by age.

But none of these throws leave the animal's skin completely exposed.

Continuous throwingobserved in horses and sheep.Because the wool of sheep in the mane and fine wool with the tail of horses falls and alternates continuously throughout the year. Many animals spawn periodically, that is, seasonally, especially in spring and summer. For example, fox fur starts in March-April in the spring and ends in July-August in the summer. The wool cover of the horses 'bodies is changed 2 times in spring and autumn, i.e. they shed 2 times in 2 seasons. Age-related shedding is an example of calving at 6-7 months of age, regardless of the season of the calf and the wedding. During urination, the body expends a certain amount of matter and energy. Because plastic (building) material is needed for the growth of new wool.

To throw a number of internal and external factors. Among them, light and ambient temperature play a special role. While light exerts its effects through the pituitary gland, the pituitary gland exerts its effects through the thyroid gland. If the thyroid gland is removed, the animal's ejaculation is inhibited, and conversely, an increase in the activity of this gland accelerates ejaculation.

Throwit is not the fall of the wool by itself. There are certain laws on its ground. But these laws have not been fully studied.

Topic: Physiology of the central and autonomic nervous system

Plan:

1. Central understanding of the nervous system. Neuronal structure and reflex activity of the CNS. Reflexes and their types.
2. Nerve centers and their properties. Physiology of the autonomic nervous system.
3. Sympathetic and parasympathetic nervous system. Trophic activity of the nervous system.

Basic expressions

Central nervous system, brain, spinal cord, neuron, reflex, Rene Descartes, Proxasco, receptors, reflexes, extroceptive, introceptive, proprioceptive, activity balance, metabolism, arousal transformation, arousal, irradiation, center tone, center plasticity, dominant property, inertia property, inertia property, braking property, postsynaptic, perisynaptic braking, coordination of centers activity, convergence and occlusion phenomenon, reticular formation. Vegetative nervous system, somatic nervous system, peripheral nervous system, sympathetic nervous system, parasympathetic nervous system, trophic activity of the nervous system, preganglionic fibers, postganglionic fibers, antagonists, synergists, higher vegetative centers, reticular pharmacy, adrenaline and norepinephrine,

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1. The neural structure of the central nervous system and reflector activity.

The central nervous system controls the vital processes of the body, ensures the interaction, coordination and integrity of the organs, and the adaptation of the organism to the external environment.

The central nervous system consists of two parts: 1. Brain 2. Spine.

The upper part of the brain is made up of the cortex of the cerebral hemispheres, which is the youngest, the upper part where the function is complex and important.

Impulses come to the central nervous system from various organs and tissues of the body through the nerve fibers that travel to the center, are analyzed and synthesized in the relevant parts of the central nervous system, and the responses come to the relevant organs through the centrifugal nerve fibers and perform certain physiological functions.
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The anterior enlarged part of the MNS is called the brain, the posterior tubular part is called the spinal cord. The more well-developed an organism is, the more strongly it develops its brain and the frontal lobe of the brain, that is, the cortex of the cerebral hemispheres. In the human body, the cortex of the cerebral hemispheres is highly developed.

The nervous system is made up of nerve cells - neurons.

The structure, shape, and size of neurons vary in size, and they are divided into 2:

1. Body part (consisting of several long protrusions of a single length).

2. Dendrites (short tumors that branch into a neuron).

The ability to excite and generate nerve impulses and to transmit these impulses to neighboring nerve cells represents the content of neuronal activity.

The body of neurons is located in the spinal cord or brain in the MNS and forms the gray matter of the brain, the tumors - the white matter of the brain.

The bodies of VNS neurons are also located in the nerve nodes. They leave the CNS and spread throughout the body, forming the peripheral nervous system. The central and peripheral nervous systems are an integral system, and both systems are equally involved in responding to the impressions received.

There are several types of neurons:

1. Receptor neurons are neurons that receive impulses from nerve fibers that tend to the center.

2. Contact (intermediate) neurons - neurons that transmit existing effects from receptor neurons to effector neurons

3. Effector neurons are neurons that transmit impulses from contact neurons to centrifugal nerve fibers.

Effects are transmitted from receptor neurons to effector neurons through one or more contact neurons.

The main and specific feature of MNS activity is the emergence of reflex processes.

IPPavlov commented on this: “Reflex is the most important and most common nervous phenomenon in the life of a complex organism. The constant, correct and precise ratio of the parts of the body and the attitude of the whole organism to the surrounding conditions are determined by reflexes.

2. Reflexes and types of reflexes.

Reflex refers to the body's response to external and internal influences in the presence of the CNS.

All the activity of the body is based on the reflex act.

All the organs in the body work on the basis of reflexes, their connection, activity adapts and harmonizes, and functions come together as a whole.

The doctrine of reflex was first introduced to science in 1644 by the French scientist Rene Descartes (1595-1650). He said that the sensory organs receive the effect, and the impressions transmitted to the brain through the nerves are reflected in it and returned to the nerves.

This doctrine was later developed in 1817 by the Czech scholar Proxaska.

The reflex activity of the cerebral cortex of the large hemispheres has been fully and comprehensively studied by IMSechenov and IPPavlov.

There are no non-reflex processes in the body.

Types of reflexes.

I. There are 3 different reflexes depending on which part of the body the receptors are involved first:

1. Extroreceptive reflexes. 2. Introceptive reflexes. 3. Proprioceptive reflexes.

Extroreceptive reflexes occur when the body’s external receptors are affected, introreceptive reflexes are affected when receptors in the internal organs are affected,

and proprioceptive reflexes are caused when receptors located in skeletal muscles, joints, and scars are affected.

II. Depending on which part of the MNS is involved, the following reflexes are distinguished:

1. Spinal reflexes are reflexes that occur in the presence of the spinal cord.

2. Bulbar reflexes are reflexes that occur in the presence of the elongated brain.

3. Mesencephalic reflexes are reflexes whose central part is located in the midbrain.

4. Diencephalic reflexes - reflexes whose central part is located in the midbrain.

5. Cortical reflexes are reflexes in which the center of the reflex arc is located in the cerebral cortex.

In the occurrence of each reflex, the role of neurons scattered in different parts of the MNS, rather than part of the MNS, is important. Therefore, the division of the brain into parts depending on the formation of reflexes is relative, because just as reflexes do not occur in the presence of the hindbrain or medulla oblongata, there are no reflexes that occur in the presence of the interstitial or cerebral cortex.

III. The following reflexes are distinguished by the nature of the reflex reaction that occurs at the beginning of the reflexes: 1. Motor reflexes. 2. Secretory reflexes. 3. Reflexes that move blood vessels.

IV. Depending on the biological importance of the organism necessary for life, the following reflexes are distinguished:

1. Nutritional reflexes. 2. Defensive reflexes. 3. Sexual reflexes. 4. Approximate reflexes. 5. Condition (tonic) reflexes. 6. Locomotor (phase movement of the body) reflexes. 7. Sound reflexes.

V. These reflexes, in turn, are divided into 2:

1. Unconditioned - spinal reflexes. 2. Conditional - brain reflexes.

There is a circular connection between the MNS and the working body, which is called feedback.

The activity of the working organ changes to the impulse from the center, and then the working organ sends impulses to the center via MIN fibers, and the center sends new impulses to the working body, analyzing and synthesizing these impulses accordingly. Under the influence of these impulses, the working body again informs the center about the extent to which it responds to impulses and the state of the working body.

Hence, an organic connection is formed between the nerve center and the controlled organ, and re-afferentation occurs.

When the impulse coming to the center informs about the change in the activity of the working organ, the center changes the activity of the organ as needed in order to meet the needs of the organism.

This means that just as the central body actively influences the activity of the body, so does the organ actively influence the activity of the center.

3. Nerve centers and their properties. A nerve center is a group of MNS neurons that work together to control an activity in the body. Certain activities are controlled by neurons scattered in different parts of the MNS, and while a certain group of them is

important for life, neurons in another part of the brain are somewhat less important. For example, cardiac activity is controlled by the cerebellum, but certain groups of neurons in the spinal cord, midbrain, and cortex of the large hemispheres are also involved in the regulation of cardiac activity. However, when the elongated brain is damaged, the heart's work is disrupted and the animal dies. MN

Excitations that start in parts of the body are first transmitted to the posterior or longitudinal brain neurons and then spread to the cerebral cortex and all parts of the CNS.

Hence, all parts of the MNS participate as a whole system in responding to impressions.

Nerve centers have a number of properties that depend on the structure of the chain of neurons that make up the nerve centers and the processes by which nerve impulses pass through synapses.

Features of nerve centers: 1. Unilateral conduction of excitation. 2. Slow down the drive. 3. Activity balance. 4. Rapid metabolism in the nerve center. 5. Fatigue of nerve centers. 6. Addition (concentration) of impulses in the nerve center. 7. Drive transformation.

Once the impulses reach the nerve center, the center is able to convert them into other impulses by changing their rhythm and strength.

8. Facilitate the passage of motion in the center. 9. Road opening incident.

The phenomenon of increasing the excitability of one center while the other is in the working position is called the center-opening feature of the center.

10. Irritation of motion.

The excitation that occurs at a particular location in the MNS not only stops at that location, but also spreads to neighboring centers is called irradiation.

11. Tone of centers.

The slightly excited state of the nerve centers is called the tone of the centers.

12. Plasticity of centers.

The ability of the center to change its activity depending on the activity of the working body is called the plasticity of the center.

13. The dominant feature of the center.

The dominant feature of the center was first identified by AAUkhtomsky.

Dominant is said to dominate the MNS with a strong agitation of a particular center.

14. Inertial properties of the center.

Inertia is when the nerve center is excited and retains traces of excitation for a long time.

He remembers things and events and can remember them after a long time.

15. Braking properties of centers.

16. Coordination of the activities of the centers.

17. Convergence events.

Convergence refers to the fact that in the MNS, the impulses coming through the center-oriented nerve fibers meet each other in the intermediate and effector neutrons.

18. Occlusion - the phenomenon of obstruction.

The reflex act effect that occurs when the MINs of two reflexes (two reflexes that cause muscle contraction) are affected simultaneously is that the effect observed when these reflexes occur separately is smaller than the arithmetic sum of the effects observed.

19. The last general road principle.

Reflexes, which are the last common path, are divided into two: while reciprocal reflexes support each other, antagonistic reflexes fight for the last common path and inhibit each other.

4. Braking events observed in the nerve center.

Braking is of great importance, preventing the continuous propagation of the excitation throughout the MNS, allowing the reflex acts to take place precisely. Braking and fatigue are like the same change from the outside, in both specific organ activity declines and stops. However, when the body is tired, the organ activity gradually decreases, fatigue increases, the organ activity decreases gradually, and the decline in activity lasts for a long time. Because braking is an active process, the organ immediately slows down and stops.

Postsynaptic braking. As a result of the examination, it was found that the MNS has neurons that provide its excitation and inhibition. Inhibitory neurons differ in the way they produce mediators from neurons that provide MNS excitation, and these mediators cause the neurons to hyperpolarize without depolarizing the postsynaptic membrane as they pass through synapses formed by neurons that provide MNS excitation.

Perisynaptic braking. This inhibition is common in the CNS, where nerve cell tumors secrete a special inhibitory mediator at the synapse where nerve cell axons pass to nerve fibers, depolarizing the perisynaptic membrane, making it difficult or impossible to pass excitation through the synapse. 'yadi.

1. One-way transmission. Nerve center neurons are made up of interconnected chains, synapses, and because these synapses conduct excitation unilaterally, excitations from nerve centers are also conducted only unilaterally.

2. Slow down the drive. Nerve centers are made up of a chain of neurons connected by synapses, which synapses slow down and slow down excitation. Because this condition depends on the structure and activity of synapses, excitation in the nerve centers is slowed down.

3. Activity balance. When the center is affected, due to a nerve impulse from the center, the activity of the internal organ does not cease with the cessation of this effect, but the working organ continues to function for some time. There are various explanations for the residual activity, with some scientists linking it to the isdepolarization observed in the possinaptic membrane when a neuron is excited, while others attribute it to the circulation of impulses in a chain of closed neurons in the nerve center.

4. Metabolism in the nerve center. Metabolism in nerve centers is accelerated relative to nerve fibers, and excitation of the center further activates metabolism, increasing 02 consumption and SO2 secretion.

5. Fatigue of nerve centers. Increased metabolism in the center exhausts the center, i.e., due to chronic excitation of the center, the passage of excitation from the synapses is disrupted, as the separation of mediators at the synapses is reduced, the sensitivity of the possinaptic membrane to mediators is reduced and the energy source of nerve cells is reduced. That's why the center gets tired.

6. Addition (concentration) of impulses in the nerve center. When the individual nerve impulses below the level of the nerve center do not move, and when these impressions are intensely superimposed, these impulses join together in the center, and they co-exist. excites the center. At the center, the impressions are added in series and at a distance, i.e., in phase. When impulses go through a single afferent nerve in series and join at the center, it is called a series connection, and when several afferent nerves go in parallel at the same time and join at one center, it is called a long-distance connection.

7. Drive transformation. Once the impulses reach the nerve center, the center is able to transform them into other impulses by changing their rhythm and strength.

For example, when a single impact comes to the center, it emits a lot of response impulses from the center, and the center conducts strong impulses weaker and weak impulses stronger.

8. Facilitate the passage of motion in the center. Extremely weak impact increases center excitability, making it easier for the impact to pass through the center. As the receptors are excited, the pulses through the MIN come to the center quickly and one after the other, and each previous pulse completes the reflex act, facilitating the passage of the next pulse through the center.

9. Road opening incident. The fact that one center is in working condition increases the excitability of the other center, and this phenomenon is called the center's cross-linking feature.

For example: If we scratch the dog's skin a little, the dog does not produce any noticeable reaction, and after passing this scratch with a strong sound effect, the dog's itching reflex occurs.

10. Irritation of motion. The excitation that occurs at a particular location in the MNS does not stop there, but spreads to neighboring centers, called irradiation, which means that a limited part of the brain is involved in the formation of a reflex reaction. The whole part of the MNS is involved to one degree or another. When the force of influence coming to the center is strong, the neighboring centers move widely, resulting in many reflex reactions. However, synapses prevent the propagation of excitation in the MNS, preventing fatigue of brain cells if certain neurons inhibit it.

11. Tone of centers. The state of some excitation of the nerve centers is called the tone of the centers, the tone of the center depends on the influence of hormones in the blood and lymph, SO2, biologically active substances from the working body, sends impulses to their working organs with a continuous rhythm and provides working body tone.

12. Plasticity of centers. The ability of the center to change its activity, the function of which depends on the function of the working body, is called the plasticity of the center. When PKAnoxin connects the central end of the dog's lost nerve to the end of the wrist and restores nerve connectivity, itching of the dog's leg results in vomiting and

coughing, which are characteristic of previously lost nerve activity, and then the dog's foot function is fully restored. This means that impulses from the foot often alter the activity of the lost nerve center, the nerve adapted to control the activity of the internal organs, and then the function of the dog's foot is restored by adapting to control the activity of the leg muscles. EAAsratyan proved the importance of the cortex of large hemispheres in the formation of plastic properties of nerve centers,

13. The dominant feature of the center. The dominant feature of the center was first identified by AAUkhtomsky. Dominant is said to dominate the MNS by strongly stirring a particular center. The strongly excited part of the YA attracts the excitations coming to the other parts of the NS, causing a strong excitation at the expense of these excitations. As a result, it overreacts, dominating other centers and slowing down their activities. When a dominant center is formed in the CNS, it produces reactions specific to the activity of the dominant center, rather than simple reactions to influences from different parts of the nervous system. For example, a bridle placed on the lip when tying a horse does not resist the tying horse because it causes severe pain in the lip and strongly stimulates the corresponding center in the brain. The pain generated during the application strongly stimulates the center of the severely sore lip. AAUkhtomsky noted that dominance is the main working principle of the nerve center. According to AAUkhtomsky, an over-excited center alters or subjugates the activity of other centers and ensures that the nervous system functions as a whole.

14. Inertial properties of the center. Inertia is when the nerve center is excited and retains traces of excitation for a long time. Inertia is well observed in the dominant center and is also characteristic of non-dominant centers. Inertia is well manifested in the cells of the cerebral cortex, and due to the fact that the centers have the property of inertia, the organism adapts to the conditions. He remembers things and events and remembers them after a long time.

15. Braking properties of centers. Inhibition of nerve centers is a complex process that counteracts the propagation of excitation. The nature of braking and driving is the same, and if the center moves excessively beyond the norm, it brakes. Hence, braking resists strong center excitation and protects nerve cells from fatigue. To prove the braking phenomenon in the MNS in 1862, IMSechenov determined the reflex time to bend the legs by cutting the frog's brain crosswise from the field of view, removing the large hemispheres, immersing the frog's limbs in a solution of sulfuric acid. IMSechenov then placed salt crystals in the area of the frog's drying tubes and immersed the frog's legs in an acid solution. observed that the reflex time was prolonged when determining the leg bending reflex time. Based on these experiments, he concluded that there were special braking centers in the brain, and in 1866 he proved that braking was observed in warm-blooded animals.

16. Coordination of the centers.

Since the braking processes in different parts of the MNS are compatible, interrelated, and consistent, the reflex acts that occur in the body are clear and mutually compatible. When a center is moved in the MNS, the center that is opposite to that center is braked and a certain function is performed. For example, a joint in the leg or arm is equipped with flexor and extension muscles. When the joint bends, the flexor

muscle center is excited and the extensor muscle center is twisted. When the joint is stretched, the center of the flexor muscle is excited and the center of the flexor muscle is braked. So, although these muscles are antagonists, they help one another work precisely. The proper functioning of the muscles depends on the compatibility and coordination of the processes of excitation and deceleration in the MNS. In 1896, NE Vvedinsky studied the reciprocal functioning of muscles, and called such innervation of muscles reciprocal innervation. These features were described by the English physiologist Ch. Studied in detail by Sherington. Because of the reciprocal innervation of the muscles, the animals move freely, the reciprocal innervation does not change, and this relationship can change under the influence of the brain without a stable event. For example: an animal bends both legs when jumping. We also see reciprocal innervation in the formation of other reflexes. For example: breathing, when one of the nerves affecting the activity of the heart is stimulated, the other is inhibited. When the reciprocal innervation is disturbed, when it is insufficient, the reflex act does not come out clearly, it is not well manifested. When we are tired, this happens, and the movements are intermittent. resiprok is formed from innervation-cyanosis. This means that one center drive opens the way for a second center brake, and one center brake opens the way for a second center drive. The law that expresses such a relationship between excitation and braking is called the law of induction. The relationship between drive and braking is of two types, with simultaneous induction and series (alternating) induction. The induction in which one center is excited and the braking of another adjacent center is simultaneously observed is called induction. It is called negative induction if braking occurs as a result of excitation, and positive induction if excitation occurs as a result of braking. If a brake is applied to one center after excitation, it is called alternating induction.

17. Convergence events. In MNS, convergence refers to the interaction of impulses coming through MIN fibers with the intermediate and effector neutrons.

While impulses from different parts of a given reflex area meet in neurons in the lower parts of the MNS, impulses from different reflex areas meet in the subcortical nuclei and cortical cells. Hence, a single intermediate and effector neuron in the lower part of the MNS is excited by impulses from different parts of the same reflex area. will receive. Initially, Ch. This principle is explained by Sherrington

The addition of pulses at a distance in the MNS explains the nature of the occlusion phenomenon.

18. Occlusion - the phenomenon of obstruction. The effect of the reflex act that occurs when the MINs of two reflexes (two reflexes that cause muscle contraction) are affected simultaneously is that the effects observed when these reflexes occur separately are smaller than the arithmetic sum.

For example, if each reflex is a reflex that causes four neurons to be excited each time it occurs, four to eight neurons are excited each time these reflexes occur independently. But if these reflexes are formed at the same time, the number of excited neurons will be six. This is because two neurons are excited simultaneously by the effects coming from both reflex fields, and the effect from the MINs of the two reflexes converges on these two neurons. The addition of pulses at a distance is generated in the

same way on the basis of convergence. While strong effects cause occlusion, weak effects cause impulses to be added at a distance.

19. The last general road principle. In the MNS, receptor (afferent) neurons are more numerous than effector neurons, so a single effector neuron communicates with several afferent neurons through an intermediate neuron and is part of different reflex arcs. Hence, impulses coming from several afferent neurons are eventually forced to pass through a single effector neuron. As a result, neurons form a common and final path. Reflexes, which are the last common pathway, are divided into two: while reciprocal reflexes support each other, atogenic reflexes fight for the last common pathway and inhibit each other.

Braking is of great importance, preventing the continuous propagation of excitation throughout the MNS, creating the conditions for the precise occurrence of reflex acts. Braking and fatigue are like the same change from the outside, in both specific organ activity declines and stops. However, when the body is tired, the organ activity gradually decreases, fatigue increases, the organ activity decreases gradually, and the decline in activity lasts for a long time. Because braking is an active process, the organ immediately slows down and stops.

Postsynaptic braking. As a result of the examination, it was found that the MNS has neurons that provide its excitation and inhibition. Inhibitory neurons differ in that they produce mediators from neurons that provide MNS excitation, and these mediators cause the neurons to hyperpolarize without inhibiting the possinaptic membrane as they pass through the synapses formed by the neurons that provide MNS excitation.

Perisynaptic braking. This inhibition is common in the CNS, where nerve cell tumors secrete a specific inhibitory mediator at the synapse where nerve cell axons pass to nerve fibers, depolarizing the perisynaptic membrane, making it difficult for excitation to pass through the synapse or at all. will stop.

1. Physiology of the autonomic nervous system.

All centrifugal nerve fibers fall into two major groups:

1. Somatic nerve fibers. 2. Vegetative nerve fibers.

The first group includes the motor nerve fibers of skeletal muscle. These fibers are also called animal nerve fibers because they are involved in the motor activities of animals.

The second group includes the remaining nerve fibers.

Based on this, the nervous system is divided into 2 parts:

1. Somatic nervous system. 2. Vegetative nervous system.

The autonomic nervous system is the part of the nervous system that controls the activity of all internal organs, metabolic processes in tissues, growth and reproduction. Hence, blood circulation, digestion, urination, respiration, reproduction, in short, the activity of all internal organs, blood vessels and skin glands are controlled by the autonomic nervous system.

The term autonomic nervous system was coined in 1800 by the French scientist M. Bisha. M. Bisha divided the whole nervous system into the autonomic nervous system, which controls the functions that give rise to the animal's senses and movements - feeding, breathing, reproduction, growth, and so on.

The functions controlled by the autonomic nervous system do not depend on the will of the organism, nor can they be stopped or altered in any way by the animal itself. In this regard, the English physiologist J. Lengley called the autonomic nervous system an autonomous (independent) nervous system.

However, the "autonomy" of the autonomic nervous system from the higher parts of the brain is relative. Because impulses coming from the cortex of the cerebral hemispheres to the center of the autonomic nervous system can also change the function of internal organs. The autonomic nervous system differs from the somatic nervous system in a number of features:

1. Once the fibers of the somatic nervous system leave the central nervous system, they reach the internal organs directly without breaking anywhere. The autonomic nervous system, on the other hand, has a nodular structure, meaning that its fibers begin at the central nervous system and end at different distances, including the abdominal cavity, in the immediate vicinity of various organs and directly inside the nodes. From these nodes, a second neuron begins, and its tumor ends directly in the organ tissue.

Accordingly, the fibers of the autonomic nervous system are divided into two: a) preganglionic (pre-knot) fibers b) postganglionic (knot-like) fibers.

The fibers from the neurons in the central nervous system to the nodes are called preganglionic fibers, and the fibers that start from the neurons in the nodes and come directly to the organ are called postganglionic fibers.

2. The fibers of the somatic nervous system extend in different order from the four vertebrae in the brain to the dorsal part of the spinal cord. The centers of the fibers of the autonomic nervous system are located only in certain parts of the central nervous system. For example, if the centers of the sciatic nerve, facial nerve, sublingual nerve, etc. are located in the medulla oblongata, the spinal cord from the first thoracic segment to the third, fourth lumbar segments and in the second to fourth segments of the autonomic nervous system. other centers are located.

3. The myelin sheath of somatic nervous system fibers is well developed, so these fibers are somewhat thicker, while most of the fibers of the autonomic nervous system do not have myelin sheath and they are much thinner.

4. The fibers of the somatic nervous system are more excitable, even more excited. The lower the excitability of the fibers of the autonomic nervous system, the slower they conduct excitation. Impulses from somatic fibers pass at a speed of 60-120 m / sec, while impulses from vegetative fibers pass at a speed of 1-18 m / sec.

5. Synaptic capture of nerve impulses at the synapses of the autonomic ganglia lasts a long time.

6. The action potentials generated in the autonomic nerve fibers last longer than the action potentials of the somatic fibers.

2. Sympathetic and parasympathetic nervous systems.

The autonomic nervous system is divided into two parts according to several anatomical and physiological features:

1. Sympathetic nervous system 2. Parasympathetic nervous system

The sympathetic and parasympathetic nervous systems, which are part of the autonomic nervous system, also differ in a number of features:

1. While the centers of the sympathetic nervous system are located in the thoracic and lumbar segments of the spinal cord, the centers of the parasympathetic nervous system are located in the midbrain and longitudinal brain, as well as in the dorsal part of the spinal cord.

2. If the nodes of the parasympathetic nervous system are located near the working organ or directly in it, the nodes of the sympathetic nervous system are located far away from the working organ, usually near the central nervous system. Therefore, the priganglionic fiber of the sympathetic nervous system is long, the postganglionic fiber of the parasympathetic nervous system is short.

3. All organs in the body are supplied with fibers of the sympathetic nervous system, but some organs, including the adrenal glands, spleen, skin, blood vessels, sweat glands, wool sacs, body muscles (except the genitals and cerebral arteries) are parasympathetic. does not receive nerve fibers.

Most of the parasympathetic fibers are contained in the stray nerve. The stray nerve travels to the organs in the chest and abdomen - the larynx, bronchi, heart chambers, stomach, small intestine, liver - and controls their work. The fibers of the third, seventh, and ninth pairs of cerebral nerves go to the saliva, tear glands, and blood vessels. The parasympathetic fibers in the humerus go to the kidneys, bladder, genitals, colon and control their work.

4. The multiplication phenomenon is peculiar to the sympathetic nervous system. The meaning of this word is that postganglionic fibers are larger than preganglionic fibers. Accordingly, impulses from a single preganglionic fiber of the sympathetic nervous system can propagate much more widely through postganglionic fibers. This phenomenon is not specific to the parasympathetic nervous system.

5. When the fibers of the parasympathetic nervous system are excited, only acetylcholine is released from the ends, while most of the fibers of the sympathetic nervous system secrete an adrenaline-like substance - sympatin, a small part - acetylcholine.

The sympathetic and parasympathetic nervous systems also differ in their activity, ie in the reactions they produce in the body:

1. When the sympathetic nervous system is stimulated, the pupils dilate, the animal sweats a lot, most of the arteries (except the coronary arteries of the heart, brain and some other vessels) constrict, the heart works hard and fast, blood pressure rises decreases, the activity of the adrenal glands increases, diuresis decreases.

2. When the parasympathetic nervous system is stimulated, the opposite is observed, including narrowing of the pupil, narrowing of the coronary arteries of the heart, and so on.

The activity of the sympathetic and parasympathetic nervous systems only seems to be opposite to each other on the surface, when in fact their activities are interdependent and mutually compatible. These nervous systems change the activity of this or that organ in one direction depending on the needs of the organism, because when the center of one is stimulated, the other is inhibited. This means that although

these nervous systems seem to be antagonistic to each other on the surface, they are synergistic in nature. Almost all organs of the body are supplied with fibers of the sympathetic and parasympathetic nervous systems. Therefore, the activity of the organs adapts to the changing needs of the organism.

Higher vegetative centers. The activity of the autonomic nervous system is controlled by the cortex of the large hemispheres, the subcortical nuclei, the reticular pharmacy, and the brain. There is a group of neurons that affect the sympathetic and parasympathetic nuclei in the subcortical nucleus, including the subcortical body.

The formation of adrenaline and noradrenaline-like substances in reticular pharmacy proves that this part of the brain is connected to the sympathetic nervous system. Reticular pharmacy maintains a certain level of activity of the centers that control vegetative functions. When the brain is removed or affected in some way, internal organs, including heart activity, vascular tone, etc., change, which affects the brain's autonomic nervous system through reticular pharmacy and the hypothalamus. All vegetative centers function under the control of the cortex of the cerebral hemisphere. Many vegetative functions are affected by changes in the V.YA. Danilevskiy, VM Bexterov. NA Mislavskiy. J. Fulton and other researchers observed. When the frontal part of the cerebral cortex is affected, the higher centers of respiration, digestion, blood circulation, and sexual activity in the cerebral cortex are considered to be in the frontal cortex. In the laboratory of KM Bykov it has been proved that it is possible to form conditioned reflexes to various vegetative functions.

3. Trophic activity of the nervous system.

Trophic activity of the nervous system refers to its ability to affect the metabolism, which occurs continuously in organs and tissues. It has been proven in many experiments that if the activity of the nervous system changes in any way, it can lead to metabolic disorders in the tissues.

Information on the trophic activity of the nervous system is well developed in the work of IP Pavlov. The works of LAOrbeli and AGGinetsinsky show the restoration of tired organ function due to the excitation of the sympathetic nervous system. Hence, under the influence of the sympathetic nervous system, tissue metabolism is accelerated.

Nerve fibers that positively or negatively affect the metabolism of tissue: 1. Nutrition; 2. Susceptibility; 3. Mobility; 4. Conductivity; 5. Affects performance.

Axon reflexes are reflexes that are formed without the involvement of the central nervous system.

This is why they are also called false reflexes.

The nerve fiber leading to the organ is branched, and if one of its branches is connected to another organ, axon reflexes can occur at this time. At this time, the excitation generated by the action of an organ passes through a nerve fiber that originates from that organ, travels to the adjacent organ, and activates that organ. Thus a reflex act takes place without the involvement of the central nervous system. For example, some time after a frog's brain is injured, if its gut is affected, a change in heart activity is observed.

Hence, due to axon reflexes, one organ can affect and alter the activity of another organ without the involvement of the central nervous system.

Vegetative reflexes are a constant component of all reactions of the organism, all unconditioned and conditioned reflexes.

As muscle activity increases, so does blood circulation, respiration, digestion, and the activity of the endocrine glands.

Vegetative reflexes are very important in maintaining the relative environment of the organism in various changes in the external environment.

Topic: Physiology of higher nervous activity

Plan:

1. The concept of higher nervous activity. The role of IMSechenov and IPPavlov in the study of the cerebral cortex and its activity.
2. Determinism, analysis and synthesis, principles of structure. Conditioned and unconditioned reflexes. Biological significance of conditioned reflexes.
3. Dynamic stereotype. Types of the nervous system. The importance of IPPavlov's teaching in animal husbandry.

Basic phrases:

ONF, cortex, subcortical structure, evolution, gray and white matter, warm-blooded animals, observation, exposure, partial and complete removal, electroencephalography, cybernetics, clinical, conditioned and spherical reflex, neuroglia, IMSechenov and IPPavlov, determinism, analysis and synthesis, structure and structure, analyzers receptors, food appearance, ringing or metronome vibration, beast, adaptation, force of impact, saliva separation method, natural and artificial conditioned reflex.

1. General concept of high nerve activity.

Higher nervous activity refers to reflex reactions that occur in the presence of an unprecedentedly developed upper part of the central nervous system - the cerebral cortex and subcortical structures of the brain.

Reflex reactions that occur in the presence of the large hemisphere cortex and subcortical structures are conditioned reflexes.

Hence, conditioned reflexes determine the behavior of the organism, forming the content and essence of cortical activity, that is, higher nervous activity.

The cerebral cortex is in constant contact with the external environment through the lower parts of the central nervous system. Through the activity of the cerebral cortex, the organism adapts to the constantly changing external environmental conditions and takes a favorable position in relation to various influences. The more developed mammals are, the more developed their cerebral cortex is. The cerebral cortex of the large hemispheres has evolved to an unprecedented extent in humans from representatives of mammals. Therefore, humans are qualitatively different from other mammals in terms of behavior and consciousness.

The activity of the cerebral cortex is studied using a variety of methods:

1. Observation method. 2. Method of affecting the cerebral cortex. 3. A method of complete or partial removal of the cerebral cortex. 4. Recording of cortical biocurrents - a method of electroencephalography. The recorded curve is called an electroencephalogram. 5. Cybernetic methods.

A method of studying brain activity by artificially embodying, modeling, using precise and precisely working mechanisms.

6. Clinical methods. Study of brain activity during various diseases.

7. The original physiological process of the cortex is studied using the method of conditioned reflexes.

Anatomical, histological, histochemical, biochemical, biophysical methods are also widely used in the study of cortical activity.

2. In the study of the activity of the cerebral cortex

Service of IMSechenov and IPPavlov.

The cortex of the cerebral hemispheres was first studied by IMSechenov, and his work "Brain Reflexes" laid the foundation for the doctrine of higher nervous activity.

Later bark activity was studied by IPPavlov. IPPavlov's conditioned reflex method is of great importance in the study of the activity of the cortex of the large hemispheres.

IPPavlov studied the activity of the cortex in detail and put forward three materialist principles of higher nervous activity.

1. The principle of determinism - just as the causes of any events that occur in nature, the events that occur in the crust of large hemispheres depend on a specific cause. If we sometimes fail to determine the cause of a process, this should not lead to the conclusion that they have no cause, our inability to determine it may be due to the imperfection of the methods we use.

2. The principle of analysis and synthesis. The cerebral cortex analyzes the effects and breaks them down into parts. It immediately combines, rounds and synthesizes these parts.

According to the analytical activity of the cortex, the individual shape of the object is distinguished, color, odor are distinguished, the synthesis activity creates an understanding of the known object and concludes about the object that gives the impression.

3. The principle of structure. At the heart of any process in the body lies a definite structure, which justifies the fact that each process produces an anatomical-physiological unit, an appropriate structure. So, since the processes of the organism are material, the processes in the cortex are also material, and they also have a material basis - a structure.

3. Conditioned and unconditioned reflexes.

All reflexes of the body are studied in two groups:

1. Conditioned reflexes. 2. Unconditioned reflexes.

Unconditioned reflexes are congenital, hereditary, reactions that remain virtually unchanged throughout life and occur without the involvement of the cerebral hemisphere cortex.

Conditioned reflexes are formed throughout life on the basis of unconditioned reflexes, disappear when they are no longer needed, do not pass from generation to

generation, and occur in the presence of the cerebral cortex. To better understand the difference between conditioned and unconditioned reflexes, let us consider conditioned and unconditioned reflexes related to salivary separation. The salivary glands of a newborn lamb do not secrete saliva without sucking the mother. When a lamb sucks on its mother, saliva begins to separate and it emerges through an unconditioned reflex. In the days that follow, the lamb sees its mother from a distance and the salivary glands begin to secrete saliva without yet sucking it. In this case, the lamb does not suck the milk, but sees its mother from a distance, which is enough to separate the saliva, that is, the saliva is separated by a conditioned reflex. Because every time the lamb tried to suckle, he saw and suckled his mother. Each time, the visual center in the cerebral cortex is stimulated, followed by the salivary separation center. In this order, after the movement is repeated several times, these centers become interconnected. The appearance of the mother at this time is conditional, the milk sucked in the mouth is an unconditional effect. Hence, for a conditioned reflex to take place, the conditioned effect must act before the unconditional effect, and must continue and strengthen it along with that effect. While unconditioned reflexes are species-specific, conditioned reflexes are reflex reactions specific to the individual. for a conditioned reflex to take place, the conditioned effect must act before the unconditional effect, and must continue to strengthen it along with that effect. While unconditioned reflexes are species-specific, conditioned reflexes are reflex reactions specific to the individual. for a conditioned reflex to take place, the conditioned effect must act before the unconditional effect, and must continue to strengthen it along with that effect. While unconditioned reflexes are species-specific, conditioned reflexes are reflex reactions specific to the individual.

Salivation is common to all animals, and salivation is the same in all species, and all mammals secrete saliva when food enters their mouths. The animal produces the necessary conditioned reflexes for feeding, protection, or self, and restores them when the need arises, losing the conditioned reflexes that have lost their significance for life in the living environment.

4. Biological significance of conditioned reflexes, rules and methods of formation. Conditioned reflexes are important for the body. This is because the environment is constantly changing, and the organism is exposed to a variety of influences, to which the organism responds clearly and perfectly, forming a conditioned reflex. Because of conditioned reflexes, the organism knows in advance that an effect will occur, prepares to respond, and assumes the most favorable position for the effect.

As a result of the formation of a conditioned reflex, the animal is protected from various hazards.

The following laws and rules must be followed in the formation of conditioned reflexes.

1. The conditioned effect should take effect 10-20 seconds before the unconditional effect.

2. Both conditioned and unconditional effects must be affected several times together. 3. The strength of conditioned and unconditional effects should be moderate. 4. The activity of the cortex of the animal cerebral hemispheres should be normal, there

should be no pathological processes in the body, no foreign influences other than the conditioned and unconditional effects used in the experiment.

The conditioned reflex can be generated in several different ways.

1. Saliva separation method.

IPPavlov studied the activity of the cerebral hemispheres using this method.

2. Protection is a method of action.

3. Action eating method.

In farm animals, the defensive-action, movement-feeding methods of conditioned reflexes are used more.

Types of conditioned reflexes.

There are several types of conditioned reflexes, depending on the type of conditioned effect:

1. Natural conditioned reflexes.

2. Artificial conditioned reflexes are different.

To generate a conditioned reflex, the bell, various light signals, the metranom's vibration are used as conditioned influences from artificial influences, and the conditioned reflexes that are formed are called artificial conditioned reflexes.

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.

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:

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.

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.

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.

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.

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.

, 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 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.

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.

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.

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:

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.

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.

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. 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.

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 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:

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.

, 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

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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.

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:

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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
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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.
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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
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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)

T/r	O‘zbek tilidagi		Русский язык		Ingliz tilidagi	
	nomi	**mazmuni**	**название**	**содержание**	**nomi**	**mazmuni**
	Hayvonlar fiziologiyasi fani va uning rivojlanish tarixi.					
1	Fiziologiya	Fizis-tabiat logos-o‘rganaman.	Физиология	Fizis- природа, logos-изучаю	physiology	Explore the physical nature logos-.
2	Hayvonlar fiziologiyasi	Qishloq xo‘jalik hayvonlarining fiziologik holatlarini o‘rnanadigan fan	Физиология животных	Наука изучающая физиологическое состояние животных	Animals physiology	
3	Tajriba	Hayvonlar fiziologiyasi fanining asosiy va bosh usuli	Опыт	Основной и главный метод предмета физиологии животных	experiment	Other methods of animal physiology and science
4	O‘tkir tajriba usullari (O‘tkir tajriba)	Eng zamonaviy usullar yordamida jarrohlik yo‘li bilan tajribalar o‘tkazish	Острые методы	Проведение опытов хирургическим путем при помощи современных методов	Acute experimental methods	Using the most modern methods of surgical experiments
5	Ta’sirlanuvchanlik	Har qanday tirik hujayra yoki to‘qimaning ta’sirotga javoban moddalar almashinuvini o‘zgartirishi	Чувствительность	Особенность клетки или ткани отвечать изменением обмена веществ на какой-либо	sensitiveness	Any live cell or tissue ta'sirotga metabolism,

		bilan ifodalanadigan xususiyatidir		раздражитель		characterized by the change in response characteristics.
6	Rivoj lanish	Zigotadan to‘la shakllangan organizm hosil bo‘lish jarayoni	Развитие	Процесс образования организма с появления зиготы	development	The process of the formation of the zygote is fully formed organism
7	Irsiyat	Organizmning o‘z belgilari va rivojlanish xususiyatlarini navbatdagi naslga o‘tkazish xossasi	Наследственность	Свойства организма передавать очередному поколению признаки и особенности развития	heredity	The body's own brands, and the characteristics of the development of the next generation of the property
Qon sistemasi fiziologiyasi						
8	Gidrolimfa	past taraqqiy etgan hayvonlarning tomirida oqadigan suyuqlik	Гидролимфа	Жидкость протекающая в сосудах низших животных	Gidrolimfa	low fluid flows through the roof of the civilize

						d animals
9	To‘qima oraliq suyuqlik	Organizmning ichki suyuq muhiti	Межтканевая жидкость	Жилкая среда организма	interstitial fluid	The body's internal fluid environment
10	Qonning yopishqoqligi	Suvga nisbatan 4-6 marta ko‘p	Вязкость крови	4-6 раз гуще воды	The viscosity of the blood	4-6 times more water
11	Oqsil koeffitsienti	Albuminlarning globulinlarga bo‘lgan nisbatiga aytiladi.	Коэффициент белка	Соотношение альбуминов к глобулинам	the rate of protein	Albumin globulin grew says.
12	Fibrinogen	Plazma oqsili (0,2-0,4%) bo‘ladi.	Фибриноген	Белок плазмы (0,2-0,4%)	fibrinogen	Plasma protein (0,2-0,4%).
13	Fiziologik eritma	0,9% osh tuzi eritmasi	Физиологический раствор	0,9% раствор поваренной соли	saline solution	0.9% saltsolution
14	Gipertonik eritma	0,9% osh tuzi eritmasidan yuqori bo‘lgan eritma	Гипертонический раствор	Раствор поваренной соли выше 0,9% и с большим осмотическим давлением	hypertonic solution	0.9% solution of high salt solution
15	Gipotonik eritma	0,9% osh tuzi eritmasidan past bo‘lgan eritma	Гипотонический раствор	Раствор поваренной соли ниже 0,9% и с меньшим осмотическим давлением	hypotonic solution	The solution below 0.9% salt solution
17	Atsidoz	Qon muhitining kislotali tomonga siljishi	Ацидоз	Изменение реакции организма в кислую	acidosis	To shift towards the acidic

				стонону		environment in the blood
18	Alkaloz	Qon muhitining ishqoriy tomonga siljishi	Алкалоз	Изменение реакции организма в щелочную стонону	alkalosis	Environment to shift towards the alkaline
19	Trombotsit	Qon plastinkalari. 1882 yilda italyan olimi Bitsotsero 1- bo‘lib fanga kiritgan.	Тромбоцит	Кровяные пластинки. Впервые в науку внес итальянский ученый Бицоцеро	platelets	Blood platelets. 1, 1882, the Italian scientist Bitsotsero personal goals.
23	Oksigemoglobin	Kislorod bilan birikkan gemoglobin	Оксигемоглобин	Гемоглобин присоединенный с кислородом	oxyhemoglobin	Linkage of oxygen and hemoglobin
24	Karbogemoglobin	Karbonat angidrid gazi bilan birikkan gemoglobin	Карбогемоглобин	Гемоглобин присоединенный с углекистым газом	carbohemoglobin	Hemoglobin linkage of carbon dioxide
25	Karboksigemoglobin	Is gazi bilan birikkan gemoglobin	Карбоксигемоглобин	Гемоглобин присоединенный с угарным газом	carboxyhemoglobin	Hemoglobin is connected with the gas
26	Metgemoglobin	Dorivor moddalardan zaharlanish oqibatida yuzaga keladi.	Метгемоглобин	Возникает вследствие отравления лекарственными препаратами	hemiglobin	Medicinal caused by toxic substances.

27	Anoksiya	Organizmda kislorod tanqisligi.	Аноксия	Нехватка кислорода в организме	Anoxia	A deficiency of oxygen in the body.
28	Granulotsitlar	Donali leykotsitlar	Гранулоцитлар	Зернистые лейкоциты	granulocytes	podsofleukocytes
29	Agranulotsitlar	Donasiz leykotsitlar	Агранулоциты	Незернистые лейкоциты	agranulocytes	leukocytes
30	Leykotsitar formula	Leykotsit turlarining bir-biriga bo‘lgan % nisbati	Лейкоцитарная формула	Процентное соотношение различных видов лейкоцитов	leukocyte formula (wbc)	The ratio of each species of leucocytes%
31	Gemotsitoblast	Qonning ona hujayralari	Гемоцитобласт	Материнская клетка крови	haemocytoblast	The blood cells of the mother
32	Eritroblast	Gematsitoblastlardan eritroblast keyin eritrotsitlar hosil bo‘ladi.	Эритробласт	Возникновение эритробласта из гемоцитобласта	erythroblast	Gematsitoblastlardaneritroblast then the red blood cells are formed.
33	Angioblastik qon hosil bo‘lish davri	Gematsitoblastlardan eritroblast keyin eritrotsitlar hosil bo‘lish davri	Ангиобластическое образование крови	Период образование эритробласта из гемацитобласта затем эритроцитов	Period of the formation of blood Angioblastik	Gematsitoblastlardaneritroblast period after the formation of red

						blood cells
34	Megal oblast	Qon hujayralari	Мегалобласт	Кровяные клетки	megaloblasts	bloodcells
35	Promi elotsit	Keyinchalik mielotsit, eozinofil, bazofil va neytrofillarga aylanuvchi qon hujayralari	Промиэлоцит	Затем образуются миэлоциты, эозинофилы, базофилы	progranulocyte	Later mielotsit, eosinophil, basophil and neutrophil blood cells rotating
36	Mega kariotsitlar	Ko‘mik va taloqdagi gigant hujayralar. Ulardan trombotsitlar hosil bo‘ladi.	Мегакариоцитлар	Образуются тромбоциты	megakaryocyte	Coal and spleen giant cells. Theplateletsare formed.
37	Fagotsitoz	Em-emirish. Organizmga tushgan et moddalarni fagotsitoz qilish.	Фагоцитоз	Поедание. Фагоцитирование чужеродных веществ попавших в организм.	Phagocytosis	Eat-repairable. Diagnostic substances in the body phagocytosis.
38	Antiko agulyant	Qonning ivishiga qarshilik qiluvchi moddalar.	Антикоагулянт	Вещества препятствующие свертыванию крови	Anticoagulant	Resistance to blood clotting agents.

39	Aggluti nogen	Tabiatan oqsil moddalar. Yopishuvchi moddalar.	Агглютиноген	Вещества белковой природы. Склеивающиеся	agglutinogens	The nature of the protein. Adhesive substances.
40	Aglyutinin	Tabiatan oqsil moddalar. Yopishtiruvchi moddalar	Аглютинин	Вещества белковой природы. Склеивающие.	agglutinin	The nature of the protein. Adhesive
41	Donor	Qon beruvchi	Донор	Дающий кровь	Donor	employer
42	Retsipient	Qon oluvchi	Реципиент	Кровь берущий	recipients	recipient
43	Agglyutinatsiya	Bir-biriga yopishib qolish.	Агглютинация	Склеивание	Agglutination	Stuck to each other.
44	Rezus faktor	Rh- dastlab makakus rezus maymunlarida aniqlangan.	Резус фактор	Rh- впервые выявлен у обезьян макаккус резус	Rh factor	makakus first detected in rhesus monkeys.
Qon aylanish sistemasi fiziologiyasi						
45	Yurak	Ichi kovak, 4 kameradan iborat muskulli organ	Сердце	Бесполый, мышечный орган состоящий из 4 камер	heart	Hollow, 4 camera muscular body
46	Sistola	Yurak muskullarining qisqarishi	Систола	Сокращение сердечных мышц	systolic	Reduction of the heart muscle
47	Diastola	Yurak muskullarining kengayishi	Диастола	Расслабление сердечных мышц	diastolic	The expansion of the heart

						muscle
48	Yurak avtomati yasi	Yurak muskullarining xususiyatlaridan biri.	Автоматия сердца	Одна из особенностей сердечной мышцы	The heart's conduction automatic	One of the features of heart muscle.
49	Refrakterlik	Yurak muskullarining biron bir ta’sirotga javob bermasligi	Рефрактерность	Безответность сердечной мышцы на какой-нибудь раздражитель	Refractories	The heart muscle does not respond to any ta'sirotga
50	Mutlaq refrakterlik	Yurak muskullarining ta’sirotga umuman javob bermasligi	Абсолютная рефрактерность	Абсолютная безответность сердечной мышцы	absolute refractory	The heart muscle does not meet the general ta'sirotga
51	Nisbiy refrakterlik	Yurak muskullarining ta’sirotga qisman javob berishi	Относительная рефрактерность	Частичное сокращение сердечной мышцы	relative Refractories	To give a partial answer to the heart muscle ta'sirotga
52	Ekstrasistola	Yurak muskullarining ta’sirotga qo‘shimcha qisqa qisqarish bilan javob berishi	Экстрасистола	Преждевременная добавочная систола	extrasystole	The heart muscles respond with a brief description of the addition

						al ta'sirotga
53	Kompenstor pauza	Ekstrasistoladan keyinga uzaygan pauza	Компенсаторная пауза	Удлиненная пауза после экстасистолы	compensator pause	Ekstrasistolada nafter a prolonged pause
54	Yurakning sistolik hajmi	Yurakning har sistolasida tomirlarga haydab chiqaradigan qon miqdori	Систолический объем сердца	Колическтво крови вытекающее при каждой систоле	The heart's systolic volume	The amount of blood out of the heart sistolasida vessels
55	Yurakning minutlik hajmi	Yurakning sistolik hajmining minutdagi qisqarishlar soni	Минутный объем сердца	Количество минутных сокращений систолического объема сердца	The volume of the heart minute	Systolic volume of the heart in one minute contractions
56	Yurakning 1 ish sikli.	Yurakning 1 ish sikli ikkala bo‘lmachaning qisqarishi bilan boshlanib, qorinchalar doastolasi bilan tugaydi.	Рабочий цикл сердца	Рабочий цикл сердца это сокращение двух предсердий и диастола желудочков	1 cycle of the heart.	1 of the heart cycle, starting with a reduction in both bo'lmachaning'redoastolasi ends.
57	Manfiy xronotrop effekt	Yurak ish ritmining kamayishi	Отрицательный хронотропный эффект	Уменьшение частоты сердечных сокращений	Negative effects xronotrop	The decreas e in heart

						rhythm
75	Manfiy batmotrop effekt	Yurak muskullarining qo‘zg‘aluvchanligi pasayadi.	Отрицательный батмотропный эффект	Уменьшение порога возбуждения	Negative effects batmotrop	Excitability of heart muscle.
76	Manfiy inotrop effekt	Yurak muskullarining qisqarish kuchi kamayadi	Отрицательный инотропный эффект	Уменьшение силы сердечной мышцы	Negative inotropic effect	Reduces the force of heart muscle contraction,
77	Manfiy dromotrop effekt	Yurak muskullarining o‘tkazuvchanligi susayadi.	Отрицательный дромотропный эффект	Уменьшение скорости проведения возбуждения по сердцу	Negative effects dronotrop	Conductivity of the heart muscle.
78	Musbat xronotrop effekt	Yurak ish ritmining tezlashishi	Положительный хронотропный эффект	Увеличение частоты сердечных сокращений	Positive effect xronotrop	Heartrhythmaccelerati on
79	Musbat batmotrop effekt	Yurak muskullarining qo‘zg‘aluvchanligi oshadi .	Положительный батмотропный эффект	Увеличение порога возбуждения	Positive effect batmotrop	Excitabilityofheartmuscle.
80	Musbat inotrop effekt	Yurak muskullarining qisqarish kuchi ko‘payadi	Положительный инотропный эффект	Увеличение силы сердечной мышцы	Positive inotropic effect	Increase the force of contraction of the heart muscle
81	Musbat dronotrop effekt	Yurak muskullarining o‘tkazuvchanligi tezlashadi.	Положительн ый дронотропный эффект	Увеличение скорости проведения возбуждения по сердцу	Positive effect dronotrop	Accelerates the conductivity of the heart muscle.

82	Gemodinamika	Qonning tomirlar bo‘ylab oqish qonuni	Гемодинамика	Закон течения крови по сосудам	hemodynamics	Leakage of blood vessels around the law
83	Gidrodinamika	Suyuqlikning naychalar bo‘ylab oqish qonuni	Гидродинамика	Закон течения жидкости по трубкам	Hydrodynamic	Law to flow through the fluid tubes
84	Maksimal (sistolik) bosim	Bo‘lmachalarning sistolasi davridagi bosim	Максимальное (систолическое давление)	Давление в период систолы предсердий	The maximum (systolic) pressure	Bo'lmachalarningpressureduringSystole
85	Minamal (diastolik) bosim	Bo‘lmachalarning diastolasi davridagi bosim	Минимальное (диастолическое давление)	Давление в период диастолы сердца	Minamar (diastolic) pressure	Bo'lmachalarningpressureduringdiastole
86	Puls bosimi yoki puls ayirmasi	Bosimning o‘zgarish amplitudasi	Пульсовое давление	Амплитуда изменения давления	pulse pressure	A pressurefluctuationamplitude
87	Sfigmogramma	Arteriya pulsini aniqlash	Сфигмограмма	Измерение артериального пульса	sphygmograms	Arterypulsetodetermine
88	Flebogramma	Vena pulsini aniqlash	Флебограмма	Измерение венозного пульса	venogram	Vienna pulse
89	Anakrota	Yurak qorinchalarining sistolasida tomir devorilarining kengayishi tufayli yuqoriga ko‘tarilgan egri	Анакрота	Кривая поднявшаяся вверх вследствие расширения стенок сосудов при	Anacrota	Ventricular stroke sistolasida due to the expansi

		chiziq		систоле		on of the devorilarining up curve
90	katakrota	Yurak qorinchalarining diastolasi tufayli pastga tushgan egri chiziq	Катакрота	Кривая упавшая вниз вследствие диастолы	decaying limb	Down due to ventricular diastole curve
91	Puls		Пульс		Pulse	
92	Puls tezligi	Puls to‘lqinlarining tomir devori bo‘ylab naqadar tarqalishi	Скорость пульса	Распространение по стенкам сосудов пульсовых волн	pulse rate	Pulse waves spread through the vessel wall so
93	Puls balandligi	Puls to‘lqinining tomir devorini qay darajada kengaytira olishi	Высота пульса	Степень расширениястенок сосудов пульсовыми волнами	pulse height	Expand the extent to which the wall of the pulse wave stroke
94	Puls kuchi	Puls to‘lqinining yo‘qolishi uchun tomir devoriga bosish zarur bo‘lgan kuch.	Сила пульса	Сила необходимая для исчезновения пульсовых волн	pulse power	For the loss of the pulse wave of the vascular wall by pressing the power.

95	Vazodilyatorlar	Tomirlar tonusini pasaytiruvchi tomirlarni kengaytiruvchi nerv tolalar	Вазодилятаторы	Сосудосуживающие волокна и уменьшающие тонус сосудов	vasodilators	Veins, arteries, lowering the tone of the nerve fibers that extend
96	Vazokonstriktorlar	Tomirlar tonusini oshiruvchi tomirlarni toraytiruvchi nerv tolalar	Вазоконстрикторы	Сосудорасширяющие волокна и увеличивающие тонус сосудов	vasoconstrictor	Vascular tone narrowed arteries carrying nerve fibers
97	Baroretseptorlar	Aorta yoyi sinokarotid refleksogen zonalardagi bosimni sezuvchi retseptorlar.	Барорецепторы	Рецепторы чувствительные к давлению	baroreceptor	Aortic arch sinokarotidrefleksogen receptors pressure.
Nafas sistemasi fiziologiyasi						
99	Jabra (oyqulоqlar)	Baliqlarning asosiy nafas organi	Жабры	Основные дыхательные органы рыб	Jabra	Fish main body to breathe
100	Pnevmotoraks	Ko‘krak qafasi devorining teshilib, plevralar oralig‘iga havo kiritilsa	Пневмоторакс	Прохождение воздуха в межплевральную полость вследствие повреждения стенок грудной клетки	pneumothorax	The hole in the wall of the chest, entered the air between the pleural

101	Inspiratsiya	Nafas olish. Ko‘krak qafasining eniga, bo‘yiga va balandligiga kengayishi.	Инспирация	Вдох, увеличение размеров грудной клетки	InspiRator	Breathing. The expansion of the chest width and height.
102	Ekspiratsiya	Nafas chiqarish	Экспирация	выдох	expiratory	expiration
103	Nafas havosi	Har bir nafas olganda o‘pkaga qabul qilinadigan va chiqariladigan havo.	Дыхательный воздух	Воздух вдыхаемый и выдыхаемый при каждом дыхательном движении	breathing air	Every breathing and production of air taken into the lungs.
104	Qo‘shimcha havo	Nafas havosi bilan chuqurroq olingan havo	Дополнительный воздух	Дополнительный воздух вдыхаемый с дыхательным воздухом	More weather	Deeper breathing air from the air
105	Rezerv havo	CHiqariladigan nafas havosi bilan qo‘shimcha cho‘qur chiqarilgan havo	Резервный воздух	Воздух выдыхаемый после спкойного выдоха	Reserve air	In addition to the production of breathable air devoured
106	O‘pkaning tiriklik sig‘imi.	Nafas havosi, qo‘shimcha va rezerv havolar yig‘indisi	Жизненная емкость легких	Сумма дыхательного, дополнительного и резервного воздуха	The lung capacity of life	Breathing air, and the sum of the Reserve
107	Qoldiq havo	O‘pka alveolalarida qolgan havo	Остаточный воздух	Некоторое количество воздуха оставшееся	the residual air	The rest of the lung alveoli

				после максимальног о выдоха		
108	O‘pkaning umumiy sig‘imi	O‘pkaning umumiy sig‘imi va qoldiq havo yig‘indisi	Общая емкость легких	Сумма жизненной емкости легкгих и остаточный воздух	The total capacity of the lungs	The sum total lung capacity and residual air
109	O‘pkaning ventilyasi ya koeffitsienti	Nafasga olingan havoning o‘pka alveolalariga etib borgan qisminingalveola havosiga bo‘lgan nisbati	Коэффициент вентиляции легких	Соотношение альвеолярного воздуха к вдыхаемому воздуху легких	The rate of ventilation of the lungs	Manhattan reached the lung alveoli, which is qisminingalveola air
110	O‘pkaning minutlik ventilyasi ya hajmi	O‘pkaga bir minutda qabul qilingan havo miqdori	Минутный объем вентиляции легких	Количество воздуха вдыхаемое в минуту	Pulmonary minute ventilation volume	A minute amount of air taken into the lungs
111	Gipoksemiya	Qonning kislorod bilan yaxshi to‘yinmay qolishi	Гипоксемия	Неполное насыщение крови кислородом	hypoxemia	Keepbloodoxygensaturation
112	Gipoksiya	To‘qimalarda kislorodning kamchiligi	Гипоксия	Уменьшение кислорода в тканях	hypoxia	Thedisadvantageofoxygen
113	Anoksiya	To‘qimalarga kislorodning mutlaqo bormay qo‘yishi	Аноксия	Прекращение доступа кислорода к тканям	Anoxia	Oxygen to tissues is completely

						stopped
114	Giperpnoe	Nafasning tezlashib chuqurlashishi	Гиперпное	Ускорение дыхания	hyperpnoea	Manhattanaccelerategrowth
115	Apnoe	Nafasning siyraklashib, yuzakilashishi	Апноэ	Урежение дыхания	Apnea	Manhattanthinningyuzakilashishi
116	Gipokapniya	qonda karbonat angidridning kamayib ketishi.	Гипокапния	Уменьшение в крови количества углекислого газа	hypocapnia	the overall carbon dioxide in the blood.
117	Giperventilyasiya	Nafas tezlashib o‘pka ventilyasiyasining kuchayishi	Гипервентиляция	Усиление вентиляции при ускорении дыхания	hyperventilation	Breathing accelerated increase in pulmonary ventilation
118	Adaptatsiya	Organizning turli xil holat yoki sharoitiga moslashishi	Адаптация	Приспособление организма к различное среде обитания	Adaptation	The body with a variety of flexible or
Hazm sistemasi fiziologiyasi						
119	So‘lak	3 juft bez quloq oldi, til osti va jag‘ osti so‘lak bezlaridan ajraladigan suyuq shilimshiq modda	Слюна	Слизистое вещество выделяемое тремя видами желез:околоушной, подязычной и подчелюстной	saliva	3 pairs of ear, subling ual and subman dibular salivary glands of

						liquid slime
120	Chaynash	Og‘izga olingan oziqaning mexanik ishlov berilib, so‘lak bilan aralashtirilib ozuqani 1 jinsli luqma holiga keltirish	Жевание	Жевание взятого корма и смешивание со слюной	chewing	The mouth from the lack of mechanical processing, mixed with saliva to make it 1 have a bite of food
121	Salivatsiya	So‘lakning ajralishi	Саливация	Выделение слюны	ptyalism	a division of the saliva
122	Gipersalivatsiya	So‘lakning ko‘p ajralishi	Гиперсаливация	Увеличение выделения слюны	hyperptyalism	Divorce a lot of saliva
123	Giposalivatsiya	So‘lakning kam ajralishi	Гипосаливация	Уменьшение выделения слюны	hyperptyalism	Divorce lesssaliva
124	Asalivatsiya	So‘lakning ajralmay qolishi	Асаливация	Прекращение выделения слюны	no ptyalism	Remain part of the saliva
125	Ezofagotomiya	Qizilo‘ngachdan sun’iy teshik ochish	Эзофаготомия	Разрез пищевода	esophagotomy	Esophagus to open a hole
127	Kardiya	Me’daning qizilo‘ngachdan kirish qismi	Кардиальная часть	Передняя часть желудка	cardia	Part of the stomach to the esophag

						us
128	Fundal	Me’daning tubi	Фундальная часть	Основание желудка	fundazol	the bottom of the stomach
129	Pilorus	Me’daning 12 barmoqli ichakka chiqish qismi	Пилорус	Выходная часть двенадцатиперстной кишки желудка	pylorus	12 duodenum, stomach, intestine,
130	Me’da shirasi	Me’da devorida joylashgan qo‘shimcha bosh va qoplama hujayralar faoliyati tufayli hosil bo‘ladigan moddalar aralashmasi	Желудочный сок	Сок выделяемый дополнительными и обкладочными клетками расположенные на стенке желудка	gastric juice	In addition to the wall of the stomach due to the activity of the cells in the skin and the formation of a mixture of substances
131	Qusish	Organizmning himoya akti	Рвота	Защитная реакция организма	KAY	Act to protect the body
132	Katta qorin	Oshqozonning dastlabki eng katta qismi	Рубец	Большая и первая часть желудка	rumen	Stomach or the first part of the largest

133	To‘r qorin	murakkab oshqozonning 2-bo‘lmasi	Сетка	Вторая часть желудка	the net profit	2 compartmentstomachcomplex
134	Qat qorin	Murakkab oshqozonning 3 bo‘limi. Qat qorin to‘r qorin va shirdon bilan tutashgan.	Книжка	Третья часть желудка.	floor without distinction abdomen	Complex stomach 3. Solid profit with net profit and rennet.
135	Kavsh qaytarish	Qayta chaynash. Naridan beri chaynab yutilgan oziqani keyinchalik yaxshilab chaynab maydalanadi va so‘lak bilan obdon aralashtirilib yutiladi.	Жевачка	Повторное жевание.тщательное прожевывание пищи и смешивание со слюной	Rumination	Re-chewing. The other has been swallowed and then pulverized and thoroughly chewing food thoroughly mixed with saliva and absorbed.
136	Bilirubin	Sariq rangli o‘t pigmenti	Билирубин	Пигмент желчи желтого цвета	Bilirubin	Yellow pigment fire
137	Bilieverdin	Yashil rangli o‘t pigmenti	Биливердин	Пигмент желчи	dehydrobilirubin	Greengrasspigm

				зеленого цвета		ent
Modda va energiya almashinuvi fiziologiyasi						
139	Azot balansi	Ozuqalar bilan organizmga kirgan va siydik orqali chiqqan azotning bir-biriga nisbati	Азотистый баланс соотношение азота попавшего в организм с кормом и выделенный с мочой		nitrogen balance	Feed the body and the ratio of nitrogen in the urine through a
140	Musbat azot balansi	Organizmga kirgan azot miqdorining organizmdan chiqqan ya’ni parchalangan azot miqdoridan ko‘p bo‘lishiga aytiladi	Положительный азотистый баланс	Количество входящего азота больше, чем количество выходящего	Positive nitrogen balance	The amount of nitrogen entering the body is said to be more than the amount of nitrogen in the body that is broken
141	Manfiy azot balansi	Organizmga kiritilgan azot undan chiqayotgan azotdan kam bo‘lsa.	Отрицательный азотистый баланс	Если количество азота попавшего в организм меньше выходящего азота	Negative nitrogen balance	Less nitrogen, or nitrogen in the body.
143	Giperglikemiya	Qonda qand miqdorining ko‘payishi	Гипергликемия	Увеличение количества сахара в крови	Hyperglycemia	An increase in blood sugar levels

144	Gipoglikemiya	Qonda qand miqdorining kamayishi	Гипогликемия	Уменьшение количества сахара в крови	hypoglycemia	A decrease in blood sugar levels
145	Tuz isitmasi	Organizmning haddan tashqari ko‘p tuz natijasida zaharlanishi	Солевая лихорадка	Отравление организма большим количеством солей	salt fever	The body as a result of too much salt poisoning
147	Avitaminoz	Vitamin etishmasligi	Авитаминоз	Нехватка витамина	Avitaminosis	Thelackofvitamin
148	Poliavitaminoz	Bir necha vitamin etishmasligi	Полиавитаминоз	Нехватка нескольких витаминов	Poliavitaminosis	A lackofvitamin
149	Gipovitaminoz	Biron vitaminning nisbiy etishmasligi	Гиповитаминоз	Относительная нехватка какого-либо витамина	Hypovitaminosis	There is a relative lack of vitamin
150	Gelirolopiya	SHabko‘rlik	Гелиролопия	Косоглазие	day-blindness	Shabko'rlik
151	Vositali kalorimetriya	Organizmga yutilgan kislorod va undan ajralib chiqadigan karbonat angidridni o‘lchashga asoslangan	Посредственная калориметрия	Основано на измерении поглощенного кислорода и выделяемого углекислого газа	indirect calorimeter	The absorbed oxygen in the body and is based on measurements of the emission of carbon dioxide

152	Vositasiz kalorimetriya	Vaqt birligida ajralib chiqqan issiqlik miqdori o‘lchanadi.	Непосредственная калориметрия	Измеряемое количество тепла выделенное в еденице времени	Calorimetry	Separated from a unit of time is measured by the amount of heat.
153	Nafas koeffitsienti	Vaqt birligida organizmdan ajralib chiqqan karbonat angidrid hajmining yutilgan kislorod hajmiga nisbatiga aytiladi.	Дыхательный коэффициент	Соотношение выдыхаемого углекислого газа к вдыхаемому кислороду	the rate of breathing	Time per unit volume of the carbon dioxide separated from the body of the absorbed volume of oxygen in it.
154	Izodinamiya	Organizmda 1 moddaning o‘rnini 2 modda qoplashi	Изодинамия	Замещение в организме одного вещества другим	isodynam	Article 1 paragraph 2 position of the body to compensate
155	Izotermiya	Tana haroratining doimiyligi	Изотермия	Постоянство температуры тела	isotherm	The continuity of the body temperature
15	Gomo	Issiq qonli	Гомойотерм	Теплокровные	homeotherm	Warm-

6	yoterm	hayvonlar	ные животные	животные	y	blooded animals
157	Poykiloterm	Sovuq qonli hayvonlar	Пойкилотермные животные	Холоднокровные животные	poikilothermic	Cold-blooded animals
158	Kimyoviy termoregulyasiya	Organizmda issiqlik hosil bo‘lishini tezlashtirish yoki sekinlashtirish yo‘li bilan yuzaga chiqadi.	Химическая терморегуляция	Появляется при ускорении и замедлении тепла в организме	chemical thermotaxis	By body heat to speed up or slow to emerge.
159	Fizikaviy termoregulyasiya	Organizmda issiqlik uzatilishini o‘zgartirish yo‘li bilan amalga oshiriladi	Физическая терморегуляция	Проведение тепла в организме при помощи изменения	physical thermotaxis	Is performed by changing the transmission of heat in the body,
160	Termoregulyasiya	Tana haroratining boshqarilishi	Терморегуляция	Управление температуры тела	thermotaxis	Bodytemperatureregulation
Ayiruv organlari sistemasi fiziologiyasi						
161	Vakuola	1 hujayrali organizmlarda (tufelkalarda) dastlabki chiqaruv organi	Вакуола	Первичный выделительный орган у одноклеточных	vacuole	1-celled organisms (tufelkalarda) in the first body
162	Nefridiyalar	Ko‘pchilik umurtqasiz ko‘p hujayrali hayvonlarda chiqaruv organi vazifasini bajaruvchi shoxlangan ingichka	Нефридии	Разветвленные тонкие трубочки выполняющие выделительную функцию у многих беспозвоночных	nefridiya	Many of the invertebrate multicellular animal body, acting

		naychalar.		многоклеточных животных		as the thin branches of the tubes.
163	Birlamchi buyrak	Past taraqqiy etgan umurtqali hayvonlarda chiqaruv vazifasini bajaradi.	Первичная почка	Выделительный орган у низших позвоночных животных	Initial kidney	Low developed the function of vertebrate animals.
164	Buyraklar	Hayvonlar va odamlar buyragi juft organ bo‘lib, organizmning bel qismida joylashgan.	Почки	Парный орган у человека и животных	kidneys	Animal and human body kidney pair was located in the back part of the body.
165	Nefronlar	Buyraklarning po‘stloq qismini tashkil etgan qismi	Нефроны	Расположен в корковой части почки	nephron	Part of the crust and part of the kidneys
166	Malpigiy koptokchasi	har bir nefronning ichida qo‘sh devorli kichik kapsuladagi kapillarlar chigali (tuguni)	Мальпигиев клубочек	Составляющая часть нефрона	Malpigiy ball	Each nephron in double-walled small seeds kapillarlar capsulated (tie)

167	SHumlyanskiy – Boumen kapsulasi	Qo‘sh devorli kapsula. Ushbu bo‘shliqdan kanalchalar boshlanadi.	Капсула Шумлянского-Боумена	Составляющая часть нефрона	Shumlyanskiy -Boumen capsule	Double-walled capsules. This empty tubules.
168	Filtratsiya	Siydik hosil bo‘lishining 1-fazasi	Фильтрация	Первая фаза образования мочи	Filter	Phase 1 of the urine formation
169	Reabsorbsiya	Qayta so‘rilish – siydik hosil bo‘lishining 2-fazasi	Реабсорбция	Вторая фаза образования мочи, повторное всасывание	Reabsorbtsiya	Phase 2 of the re-absorption of urine formation
170	Og‘riq anuriyasi	Og‘riq natijasida siydik ajralishining kamayishi.	Оғриқ анурияси	Уменьшение выделения мочи вследствие боли	pain anurii	Pain reduction in urine as a result of divorce.
171	Anuriya	Siydik ajralmasligi	Анурия	Прекращение выделения мочи	anuria	Separationofurine
172	Albuminuriya	Siydik orqali oqsillarning ajralishi	Албуминурия	Выделение белков с мочой	Albuminuriya	Separation of proteins in the urine
173	Glyukozuriya	Siydik bilan qand, glyukoza chiqarilishi	Глюкозурия	Выделение сахара с мочой	glycosuria	Urinesugar, glucose production
174	Gematuriya	Siydik bilan qon chiqarilishi	Гематурия	Выделение крови с мочой	hematuria	Blood in the urine product

						ion
175	Gemo globin uriya	Siydik bilan gemoglobin chiqarilishi	Гемоглобин урия	Выделение гемоглобина с мочой	hemoglobinu ria	Product ion of hemogl obin in the urine
176	Ureter	ichki siydik yo‘llari	Уретер	Внутренние мочевые пути	The ureters	urinaryt ract
177	Diurez	Bir yo‘la ajraladigan siydik miqdoriga	Диурез	Количество выделяемой мочи	diuresis	With the amount of urine
178	Kloak a	Qushlarning to‘g‘ri ichagi	Клоака	Прямая кишка птиц	Kloaka	Birdrect um

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?
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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?
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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?
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?
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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 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?
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?
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82. Explain the development of the science of pathological physiology in Russia?
83. Explain hypoglycemic shock and the mechanism of its formation?
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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?

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

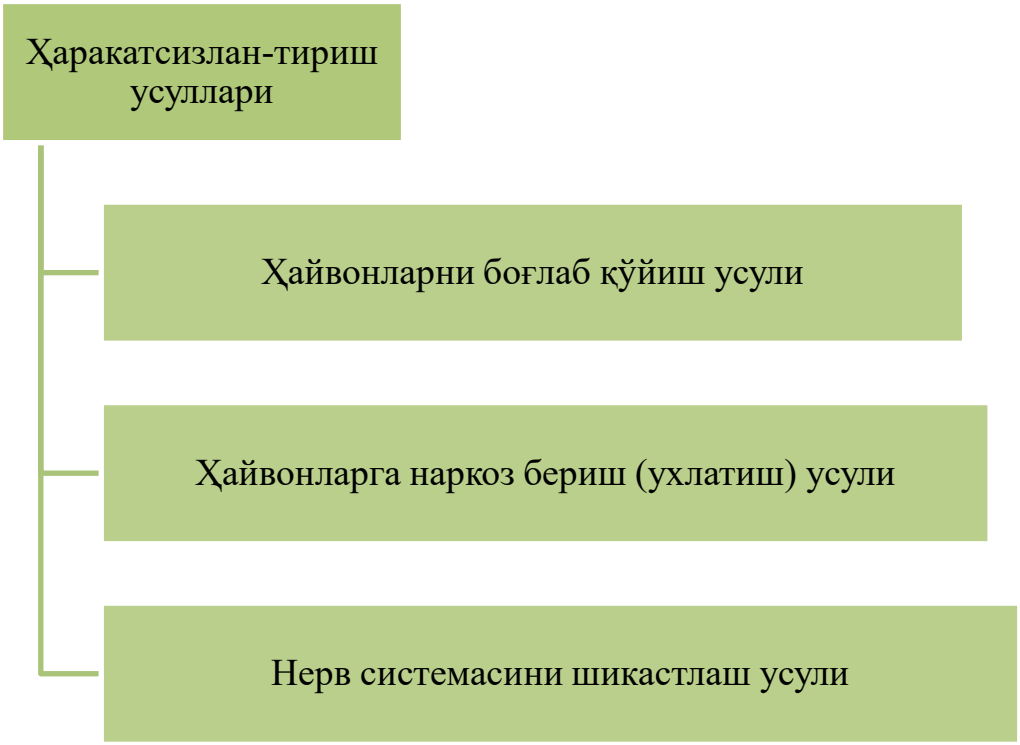
500. What is the attachment of the liver instead of parenchymal cells called tissue outflow?

A. Hepatoma
B. Hepatosis
D. Hepatitis
E. Cirrhosis

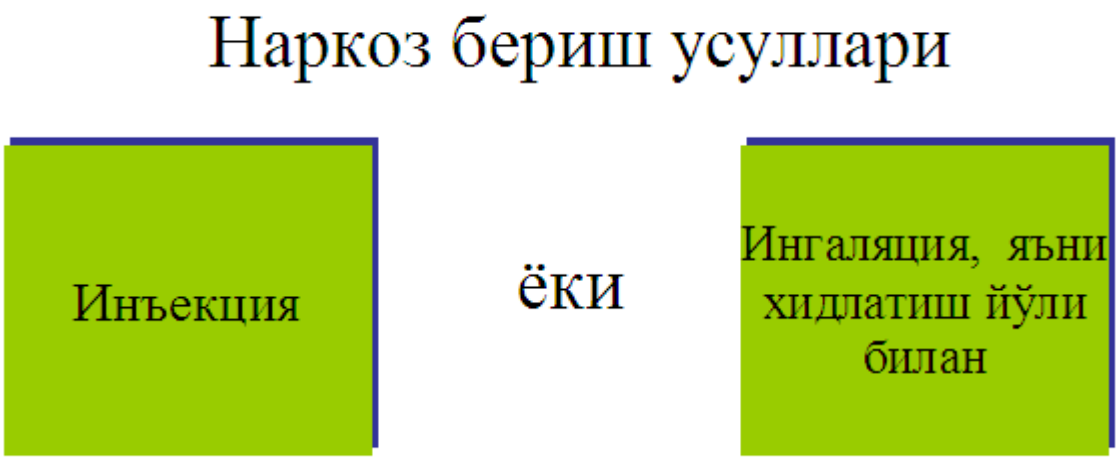
Science Assessment Criteria

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

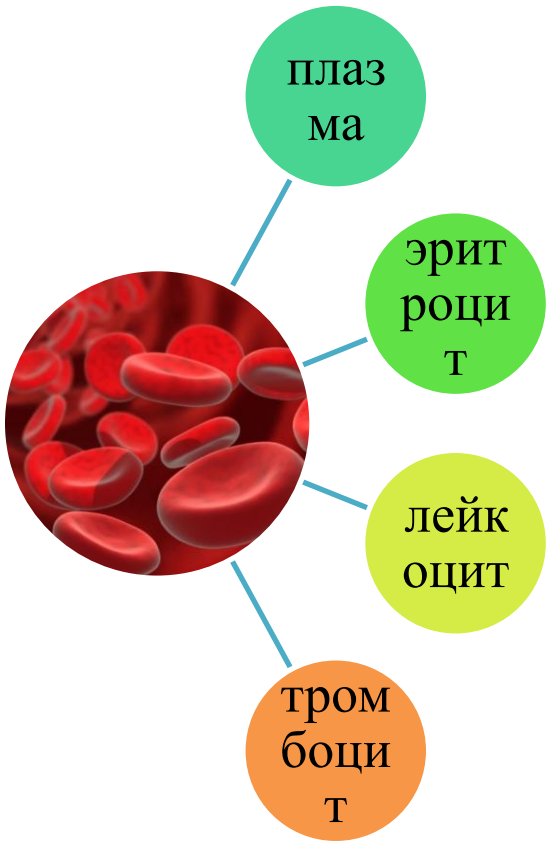

Наркоз бериш усуллари
Иньекция
ёки
Ингаляция, яъни хидлатиш йўли билан


Вивисекция усули
Органларни изоляция килиш усули
Экстерпация усули
Ўткир тажриба усуллари
томирлар анастомози
Трансплантация усули
лигатуралар солиш усули
Денервация усули

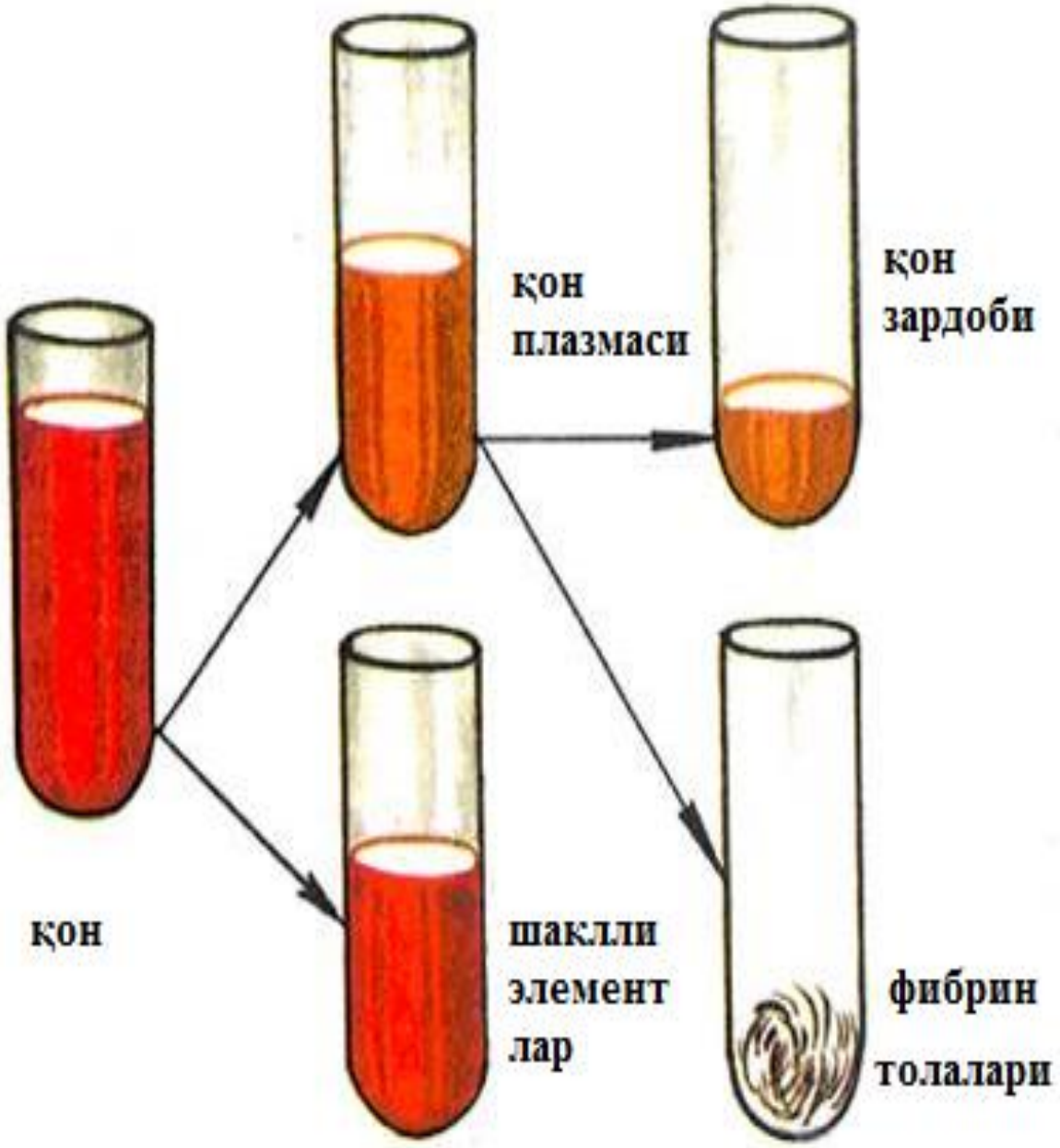
Xronik(surunkali) usullar:

2-Mavzu. QON PLAZMASI VA QON ZARDOBINI AJRATIB OLISh

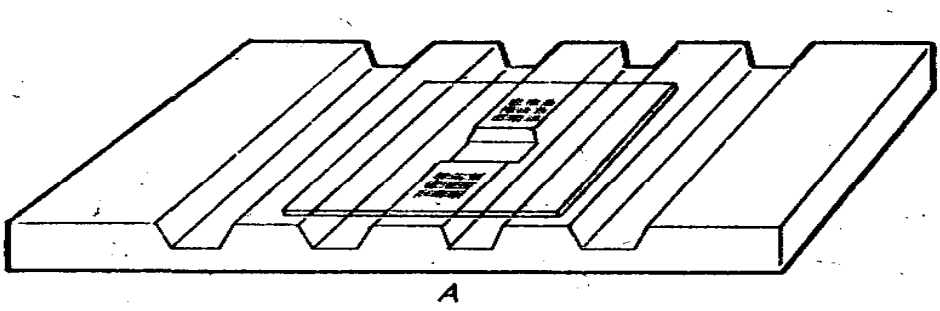
Darsning maqsadi: Qon, qon plazmasi, qon zardobi va fibrinsizlantirilgan qon haqida tushunchaga ega bo'lish hamda qon plazmasi, qon zardobi va fibrinni ajratib olishni o'rganish.



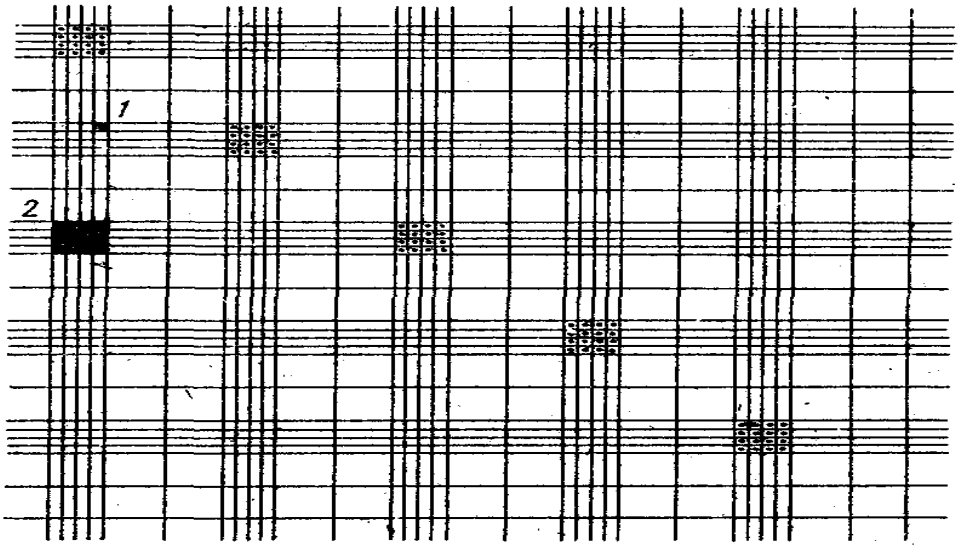
Қон олиниб центрофуга қилингач, 2 қисмга ажралади, плазма - яъни сариқ тиниқ суюқлик ва шаклли элементларга



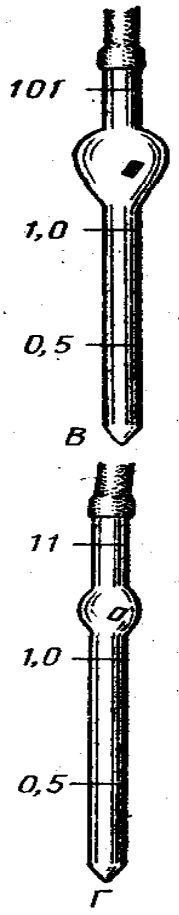
Demak, qon plazmasida 0,4% gacha fibrinogen bo’lishi bilan zardob farqlanadi.

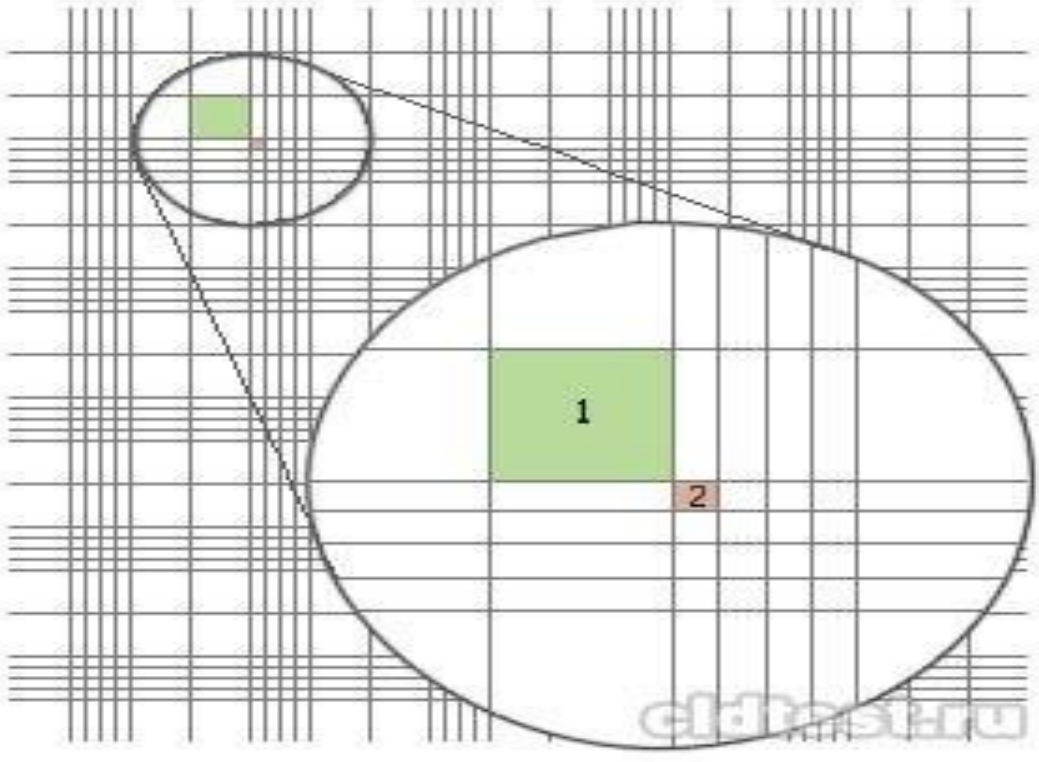


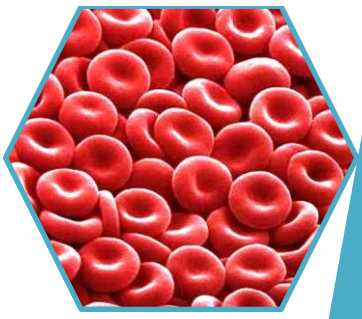
А



Б



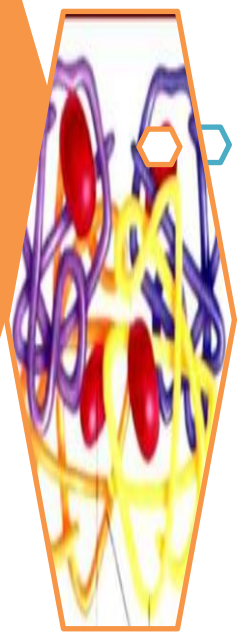




Эритроцитларнинг ўртача диаметри 4-7 микронга тенг

• 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

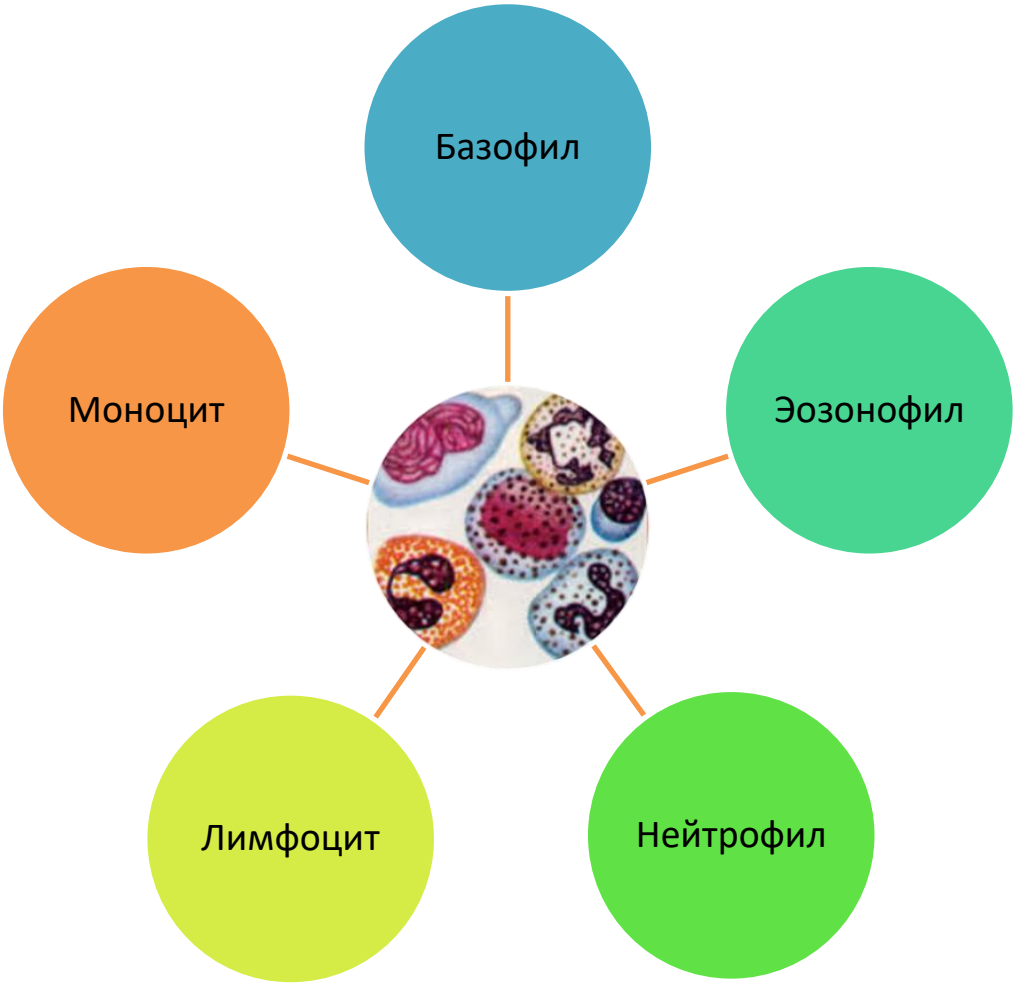
o’rtacha 120 kun muddatda yashaydi. Umri tugagan eritrositlar jigar va taloqda parchalanadi.

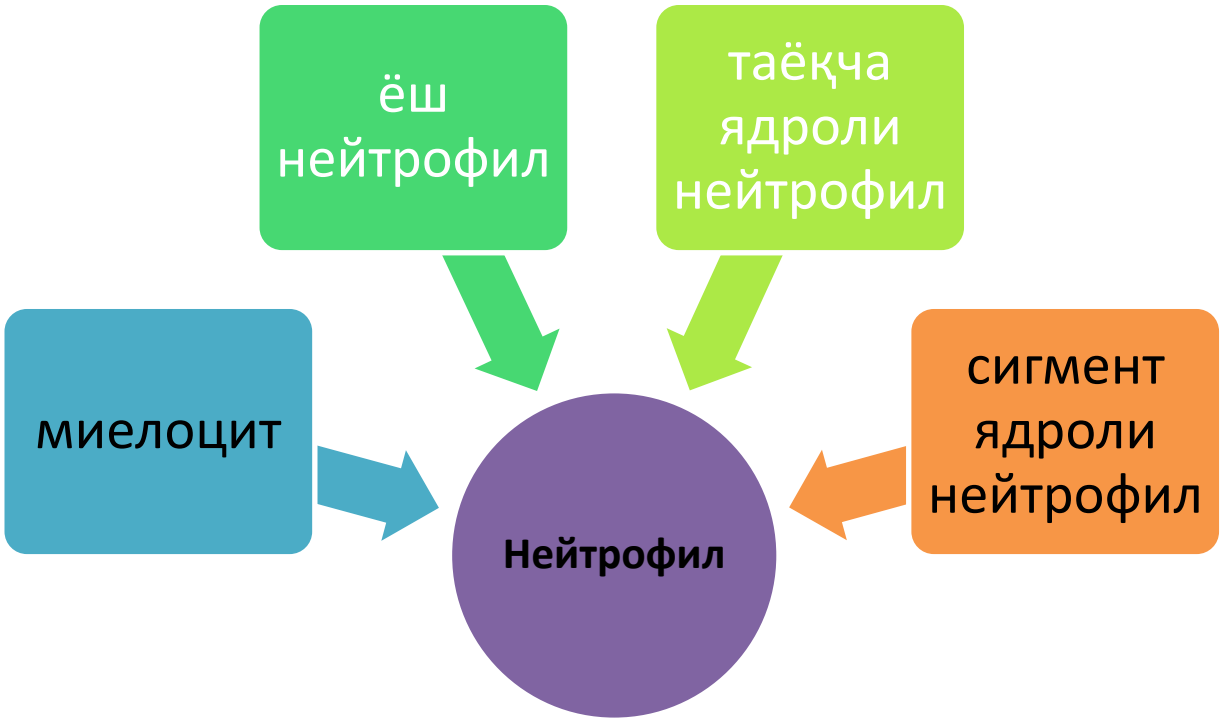
Грек.
- leucocytus
- яъни оқ қон таначалари

грек.
- leucocytosis
- яъни лейкоцитларнинг кўпайиб кетиши

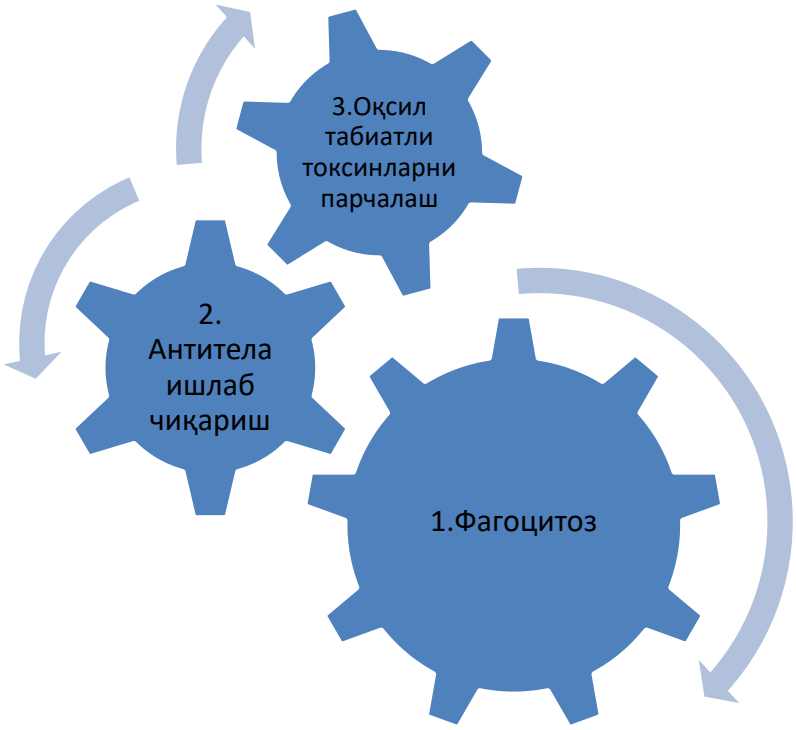
грек. .
- leucopenia
- яъни лейкоцитларнинг камайиб кетиши

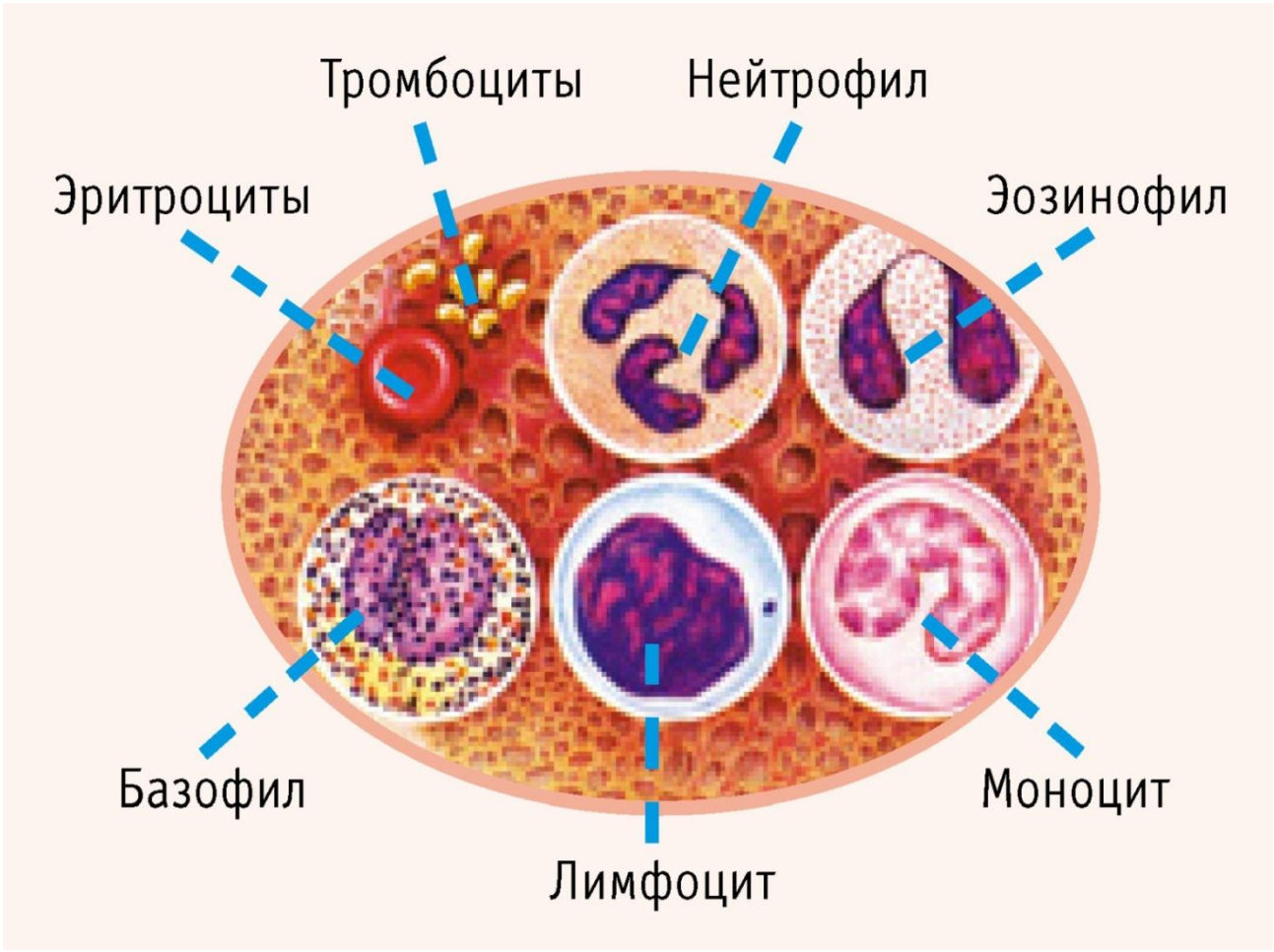
грек. .
- leucopoieses
- яъни лейкоцитларнинг ҳосил бўлиши

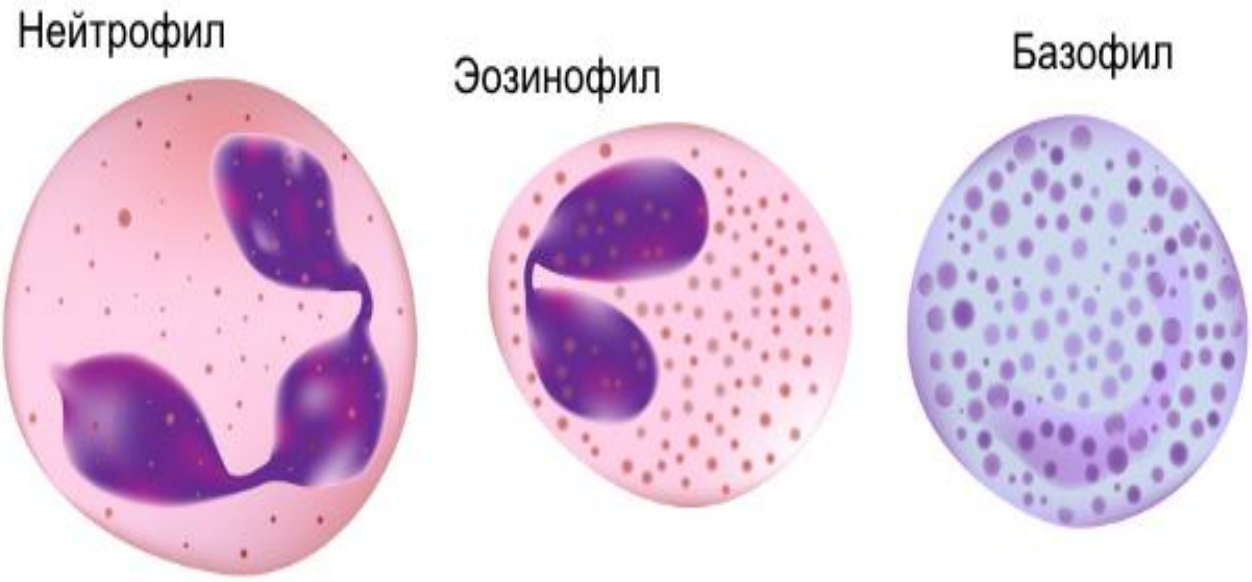




Leykositlarning organizmdagi asosiy vazifasi:




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.

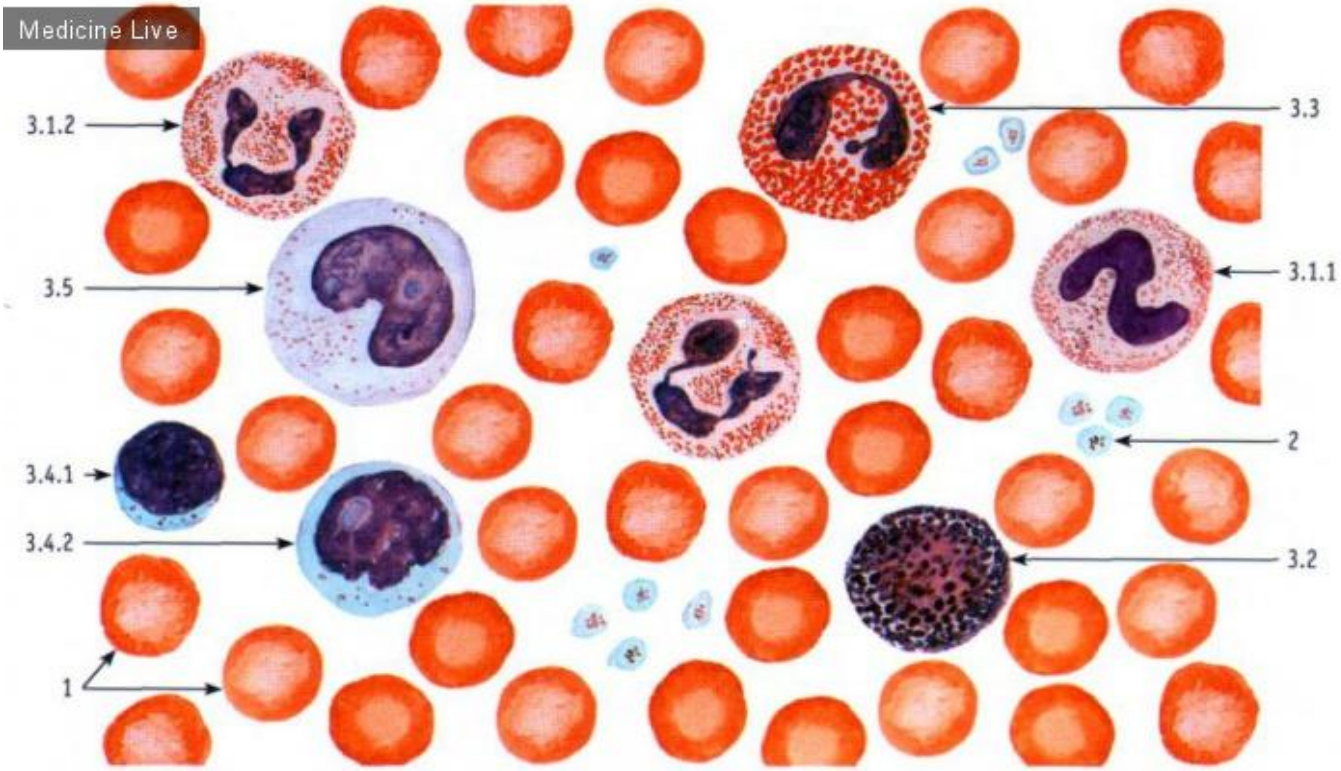
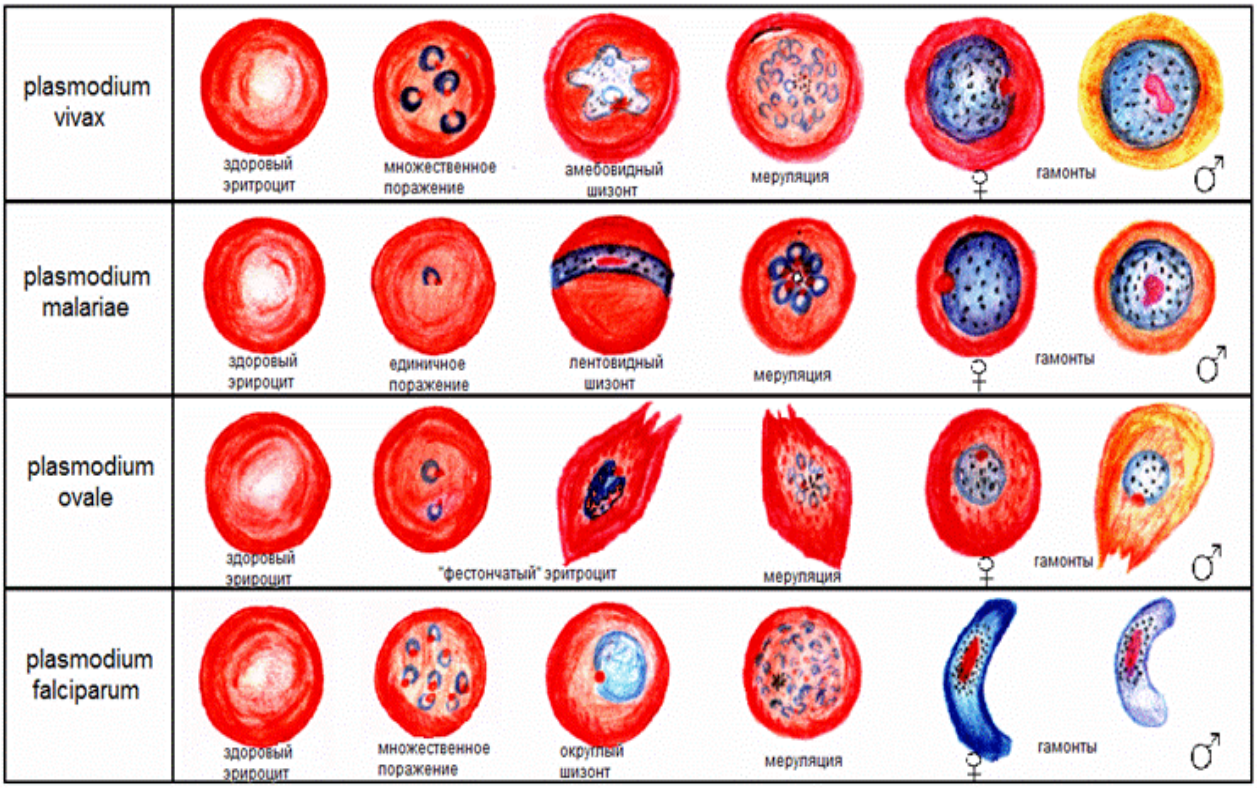
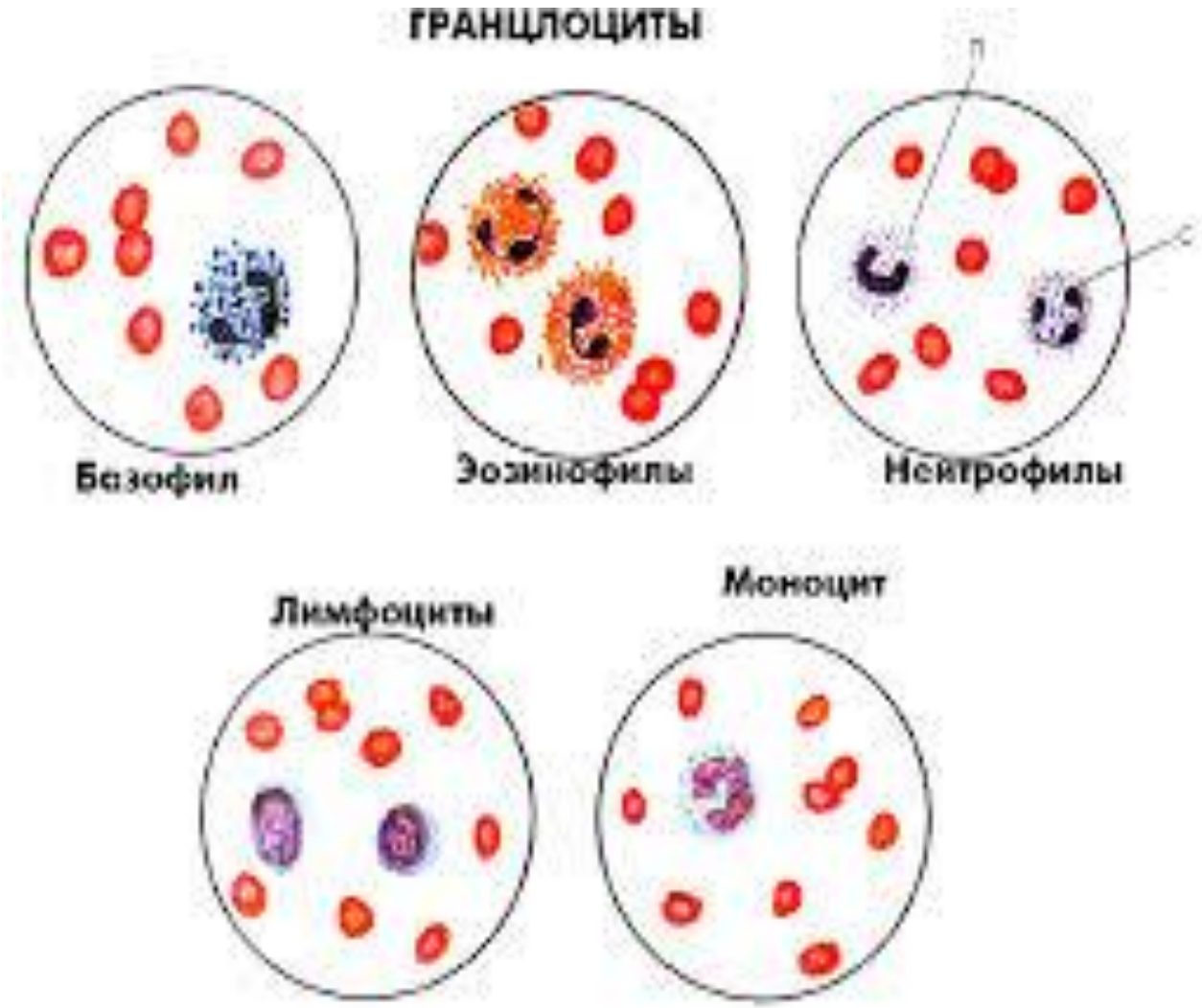


Рис. 47. Кровь человека (мазок)

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



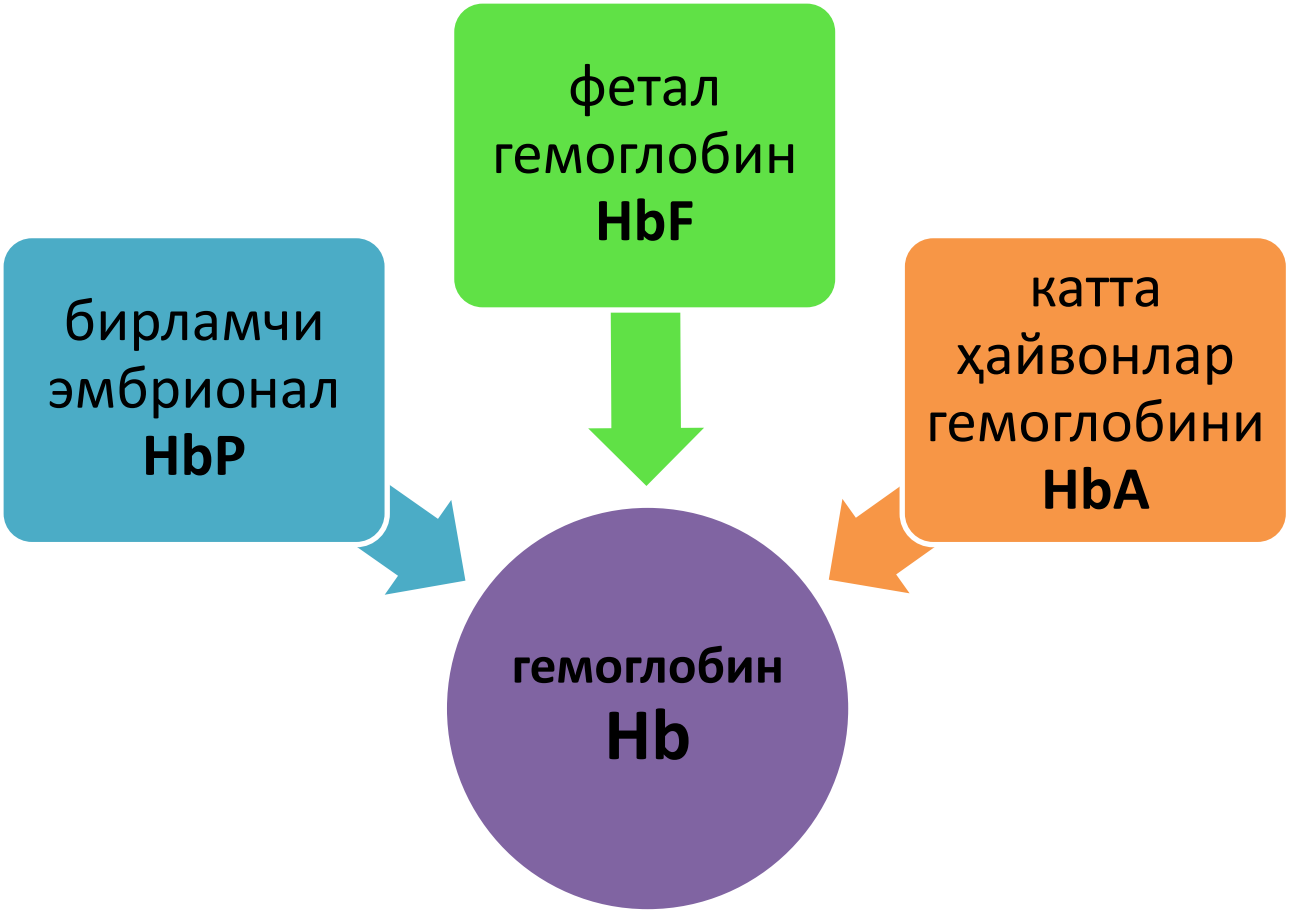


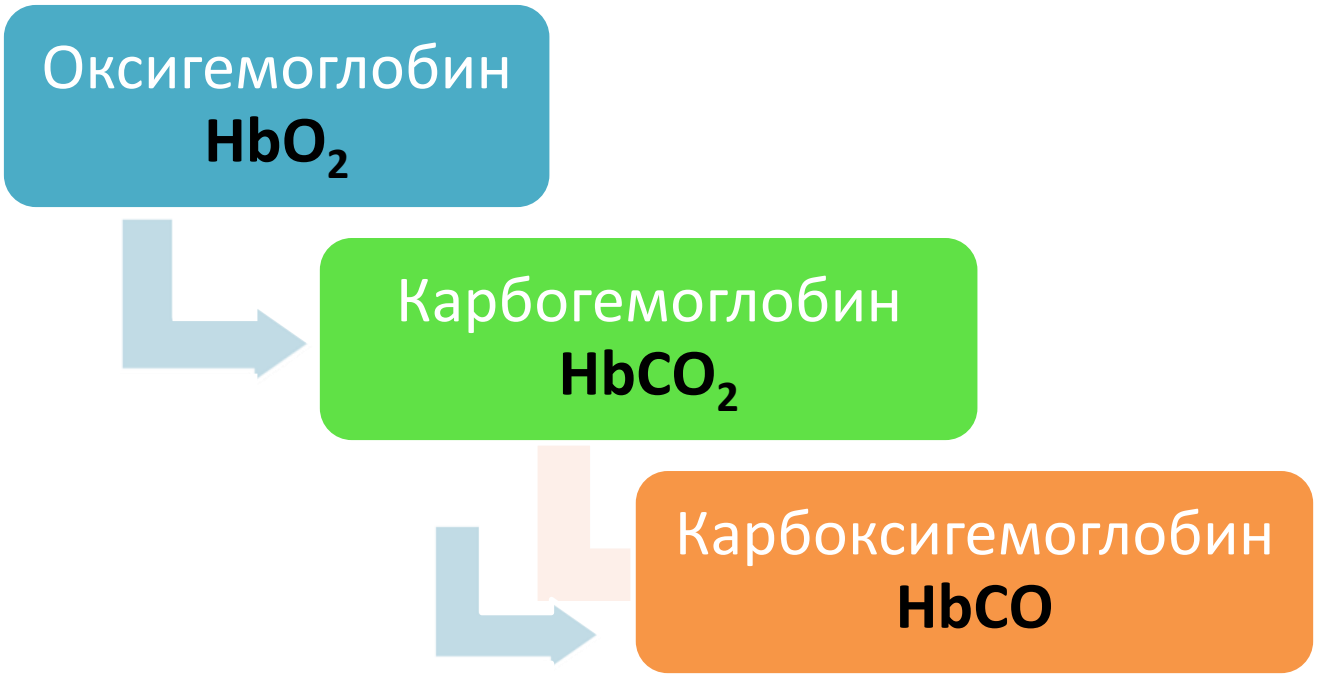
Лейкоцитларни санаш усуллари

- Тўрт майдон усули
- Филлипченко усули
- Суртманинг ўртасидан санаш усули

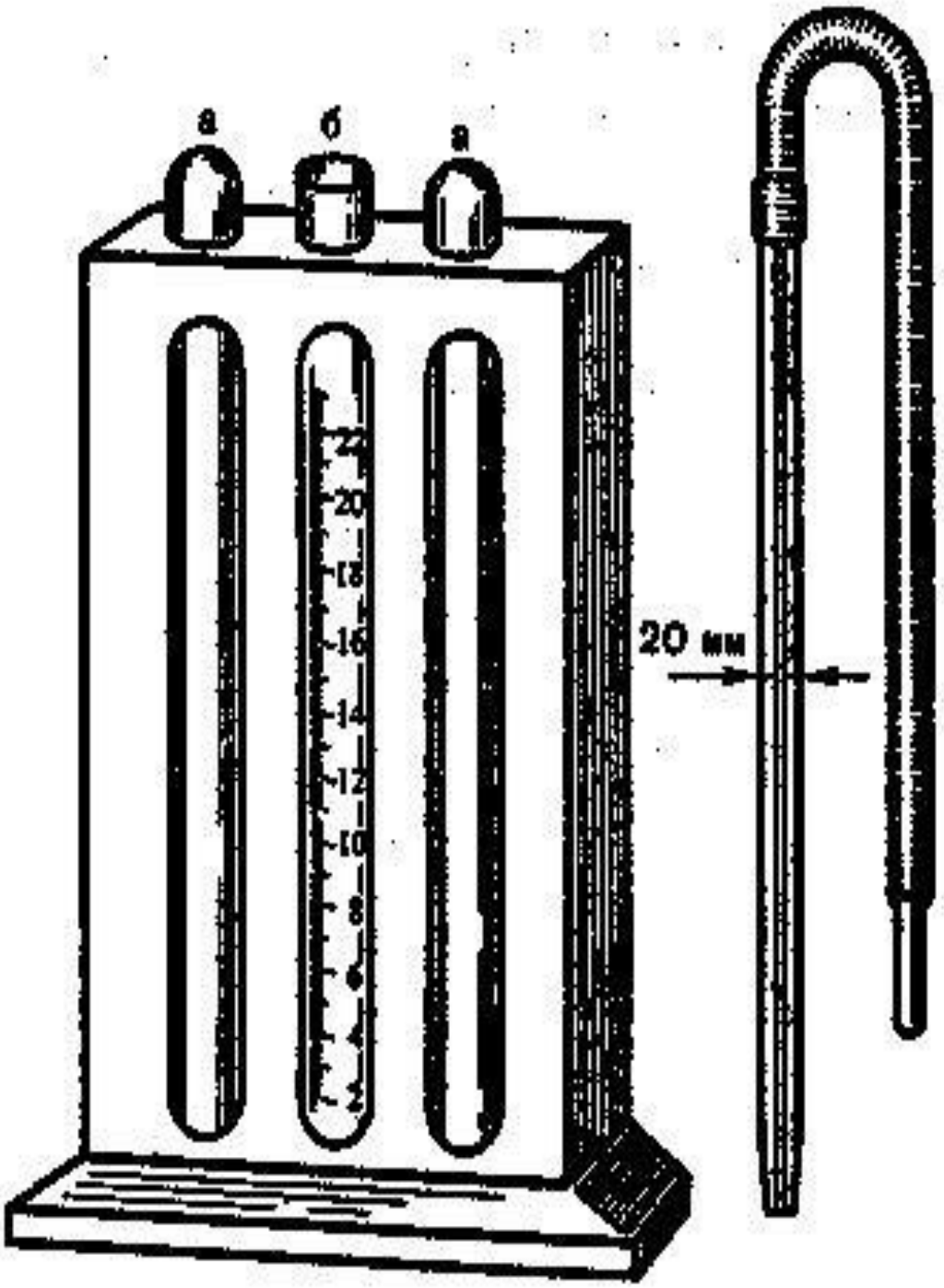
Животные	азофилы	Эозинофилы	Нейтрофилы			Лимфоциты	Моноциты
			Ю				
Крупн. рог. скот	0-2	5-8	0-1	2-5	20-35	40-65	2-7
Овца	0-1	4-12	0-2	3-6	35-45	40-50	2-5
Коза	0-1	3-12	-	1-5	29-38	47-64	2-4
Верблюд	0-1	4-12	0-2	1-6	40-52	29-45	1-5
Олень	0-1	3-7	0-1	2-5	55-66	21-37	1-4
Буйвол	0-2	3-10	-	1-6	24-46	45-66	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

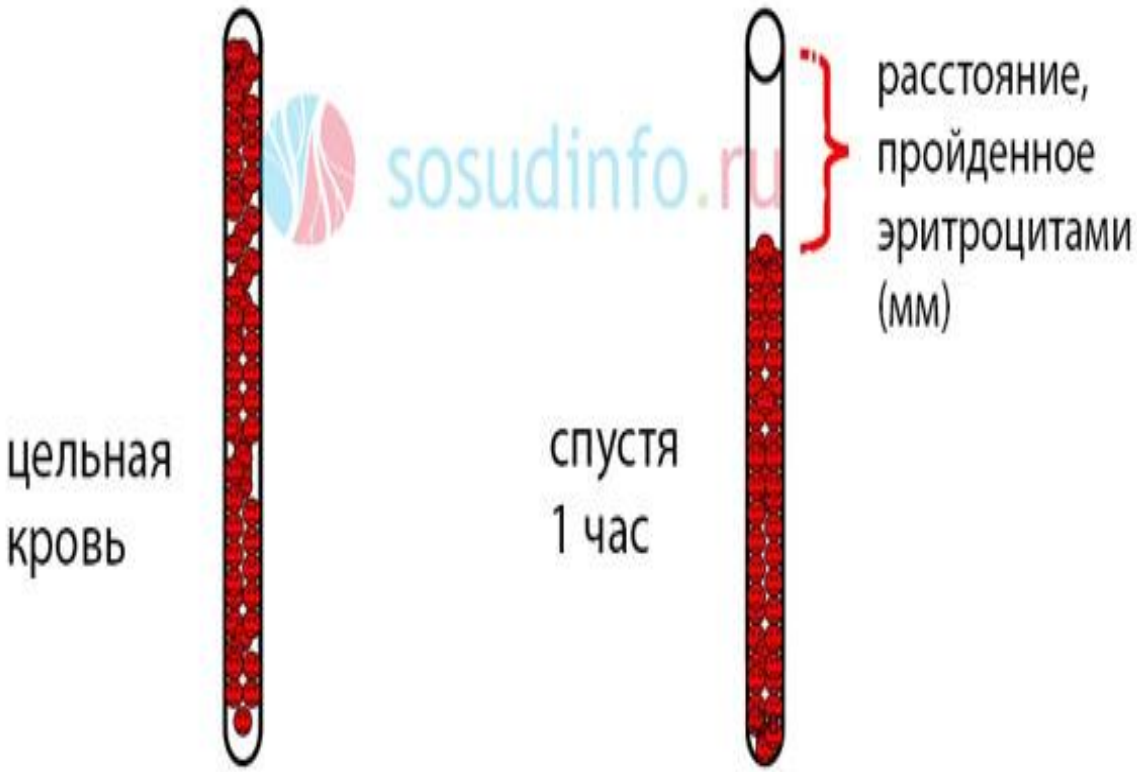
Fiziologik gemoglobinning 3 xili farqlanadi.

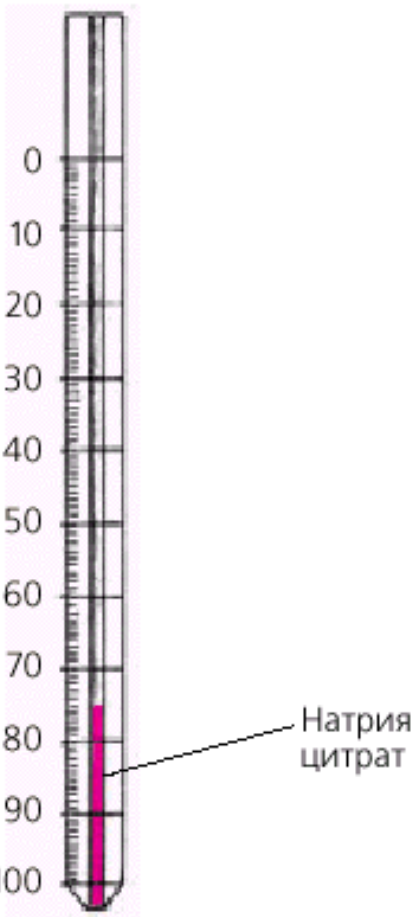


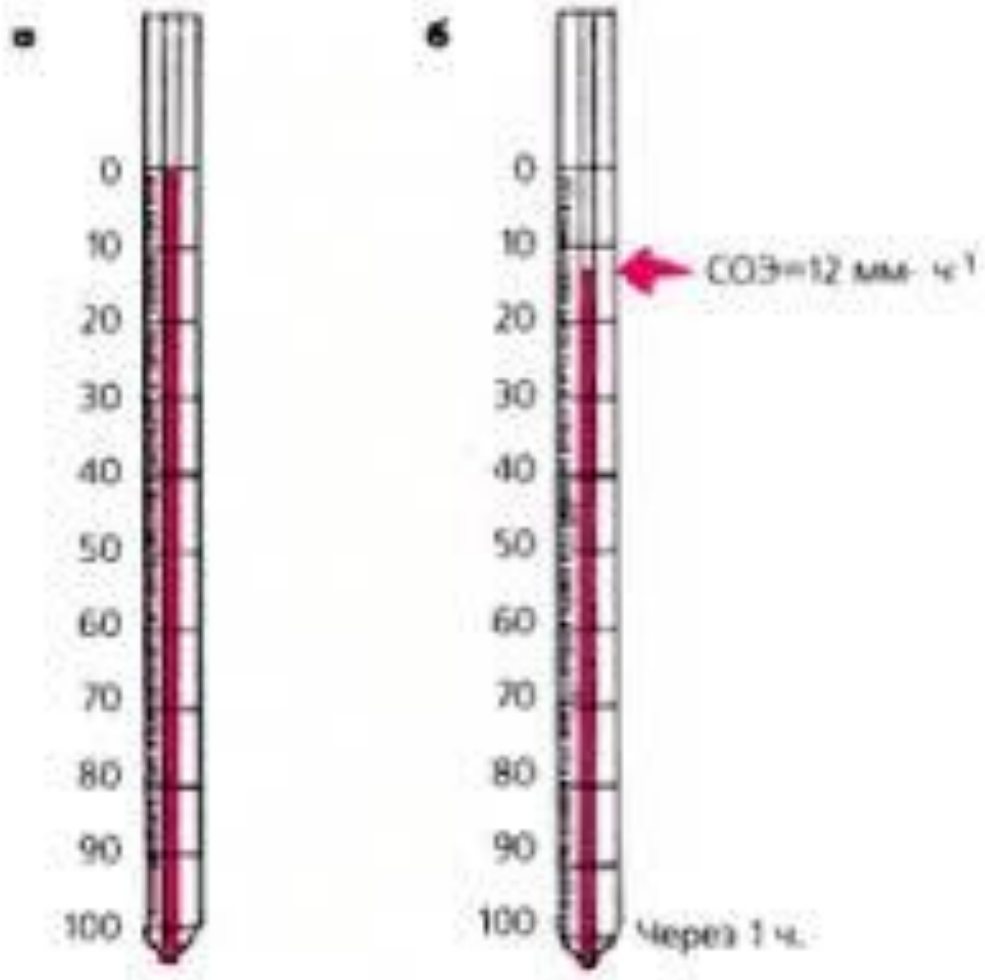

Оксигемоглобин
HbO2
Карбогемоглобин
HbCO2
Карбоксигемоглобин
HbCO


Метгемоглобин
HbO
Миоглобин

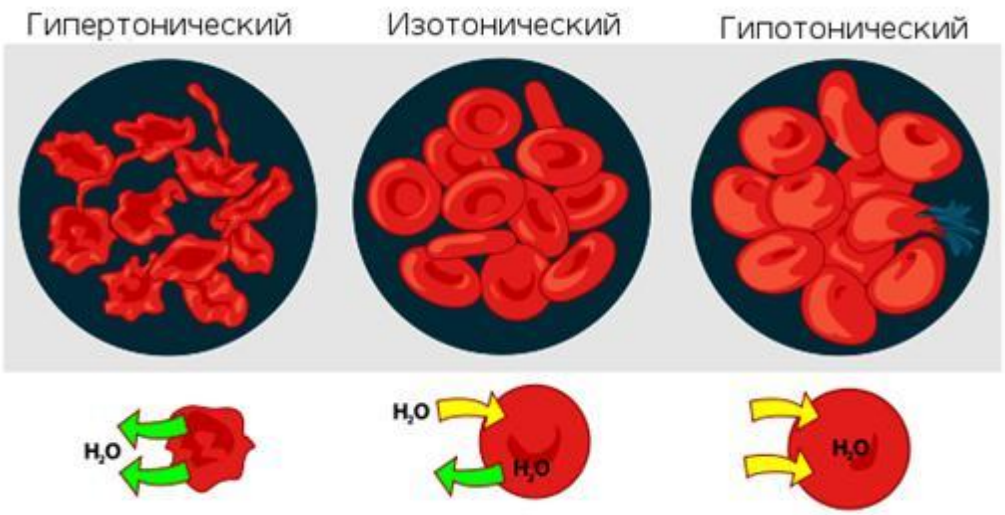

а
б
в
20 мм



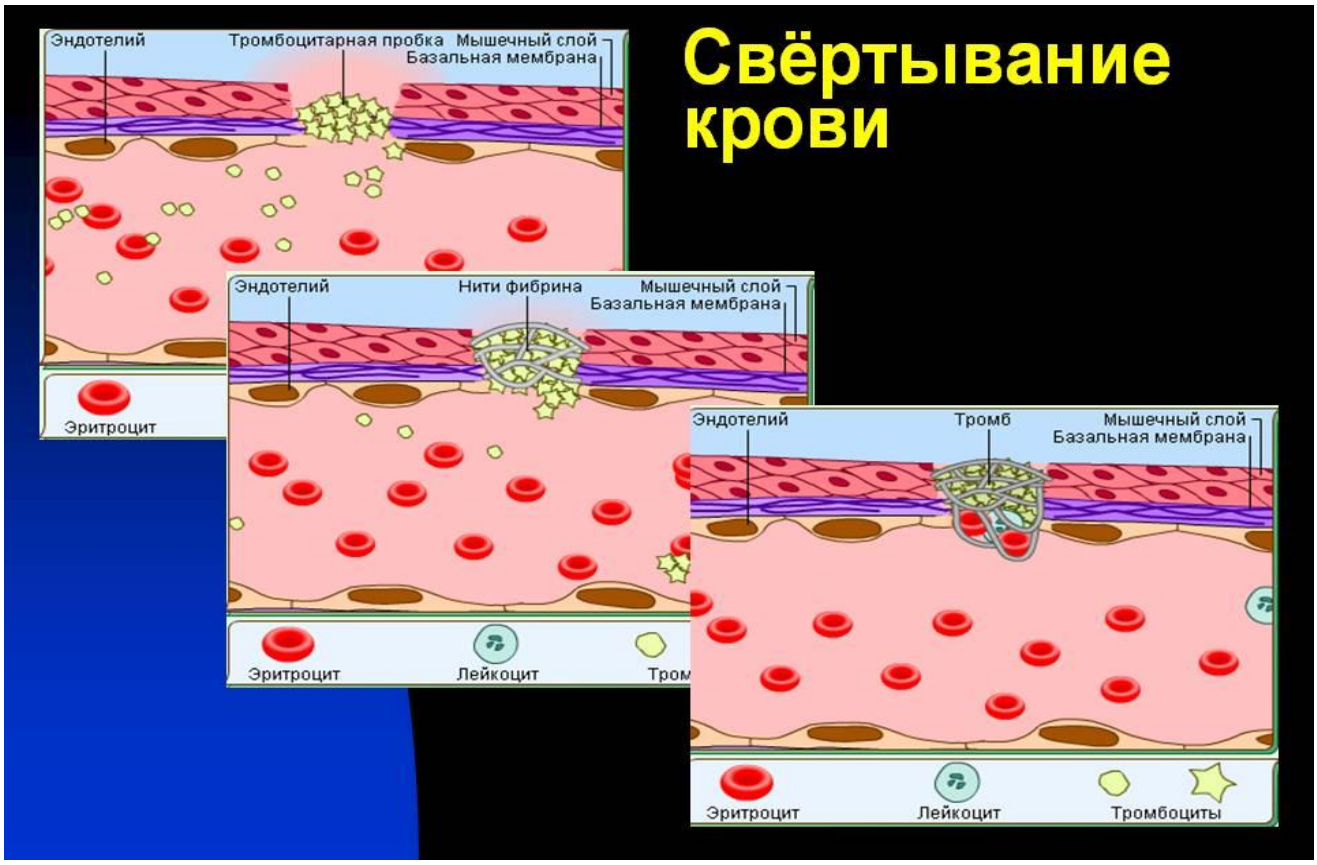

0
10
20
30
40
50
60
70
80
90
100
Натрия
цитрат


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.

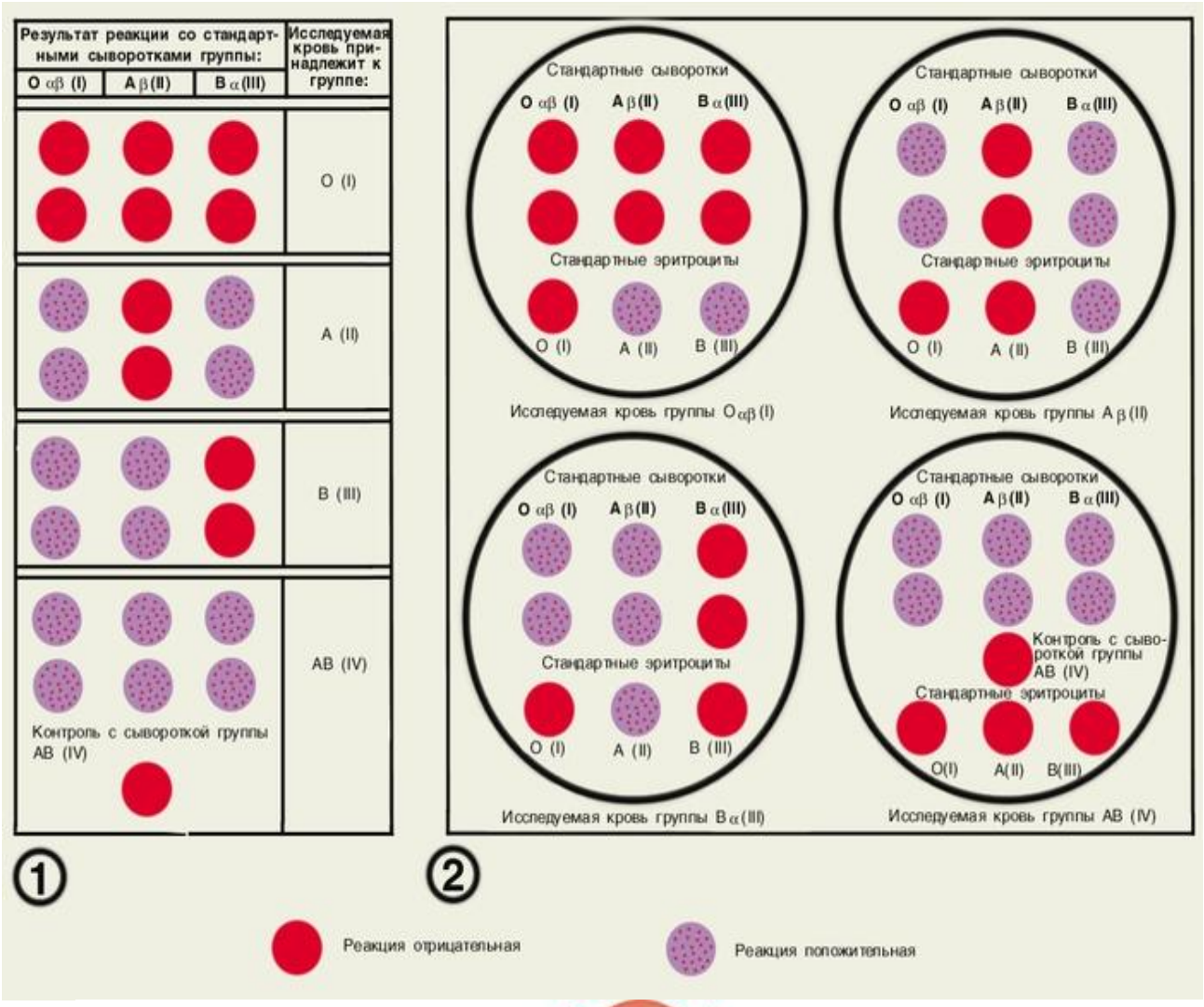
10-Mavzu: GEMOLIZ. ERITROSITLARNING OSMOTIK REZISTENTLIGINI ANIQLASh

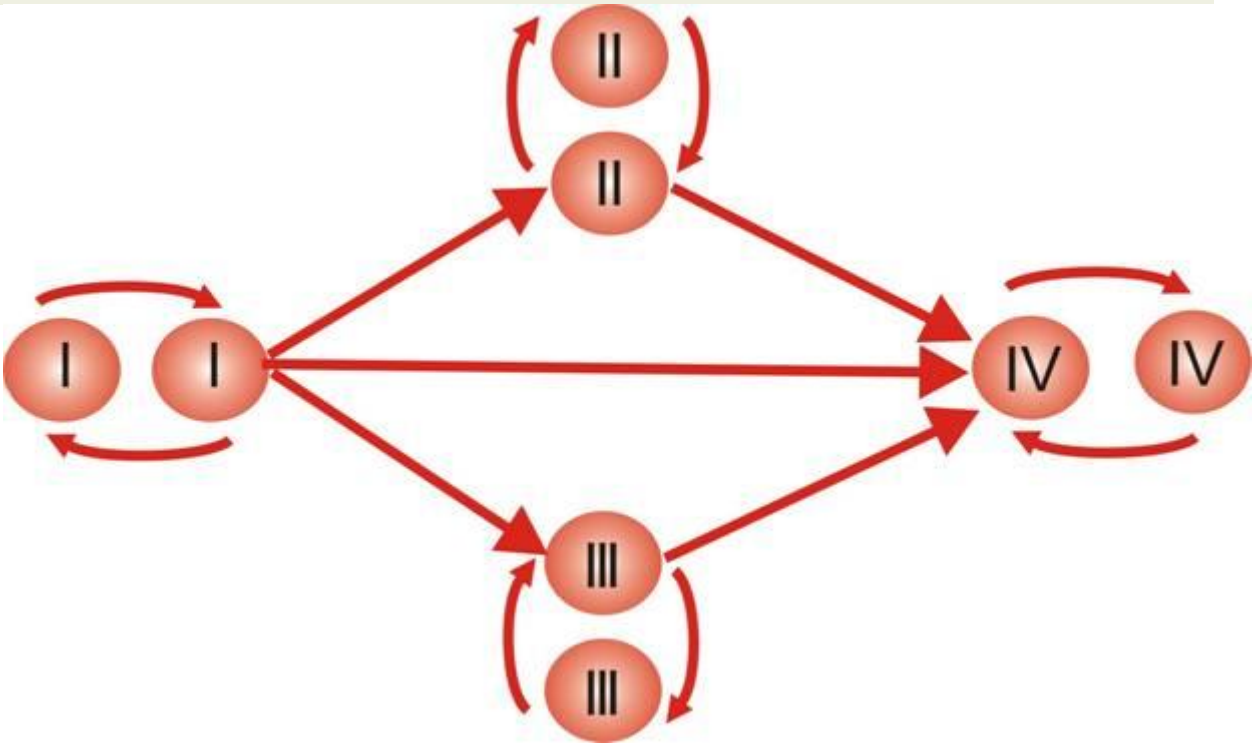


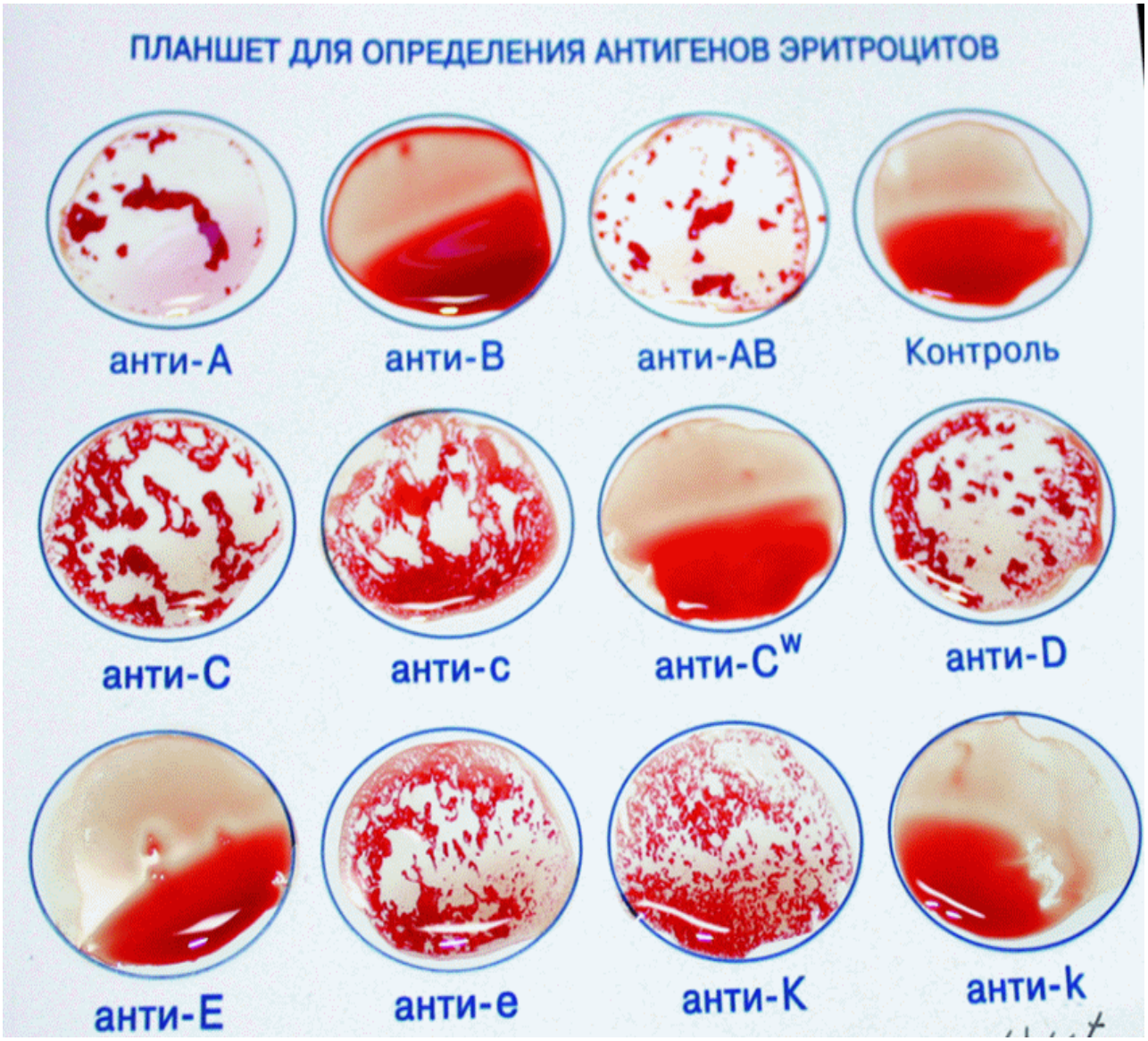
11-Mavzu: QONNING IVISh TEZLIGINI ANIQLASh



12-Mavzu: QON GURUXLARINI ANIQLASh





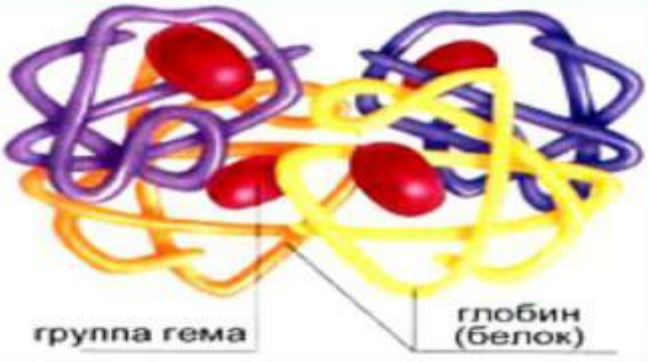


резус-фактор		Rh- (--)		Rh+ (-+)		Rh+ (++)	
	ген	-	-	-	+	+	+
Rh- (--)	-	Rh- (--)	Rh- (--)	Rh- (--)	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)
	-	Rh- (--)	Rh- (--)	Rh- (--)	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)
Rh+ (-+)	-	Rh- (--)	Rh- (--)	Rh- (--)	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)
	+	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)	Rh+ (++)	Rh+ (++)	Rh+ (++)
Rh+ (++)	+	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)	Rh+ (++)	Rh+ (++)	Rh+ (++)
	+	Rh+ (-+)	Rh+ (-+)	Rh+ (-+)	Rh+ (++)	Rh+ (++)	Rh+ (++)

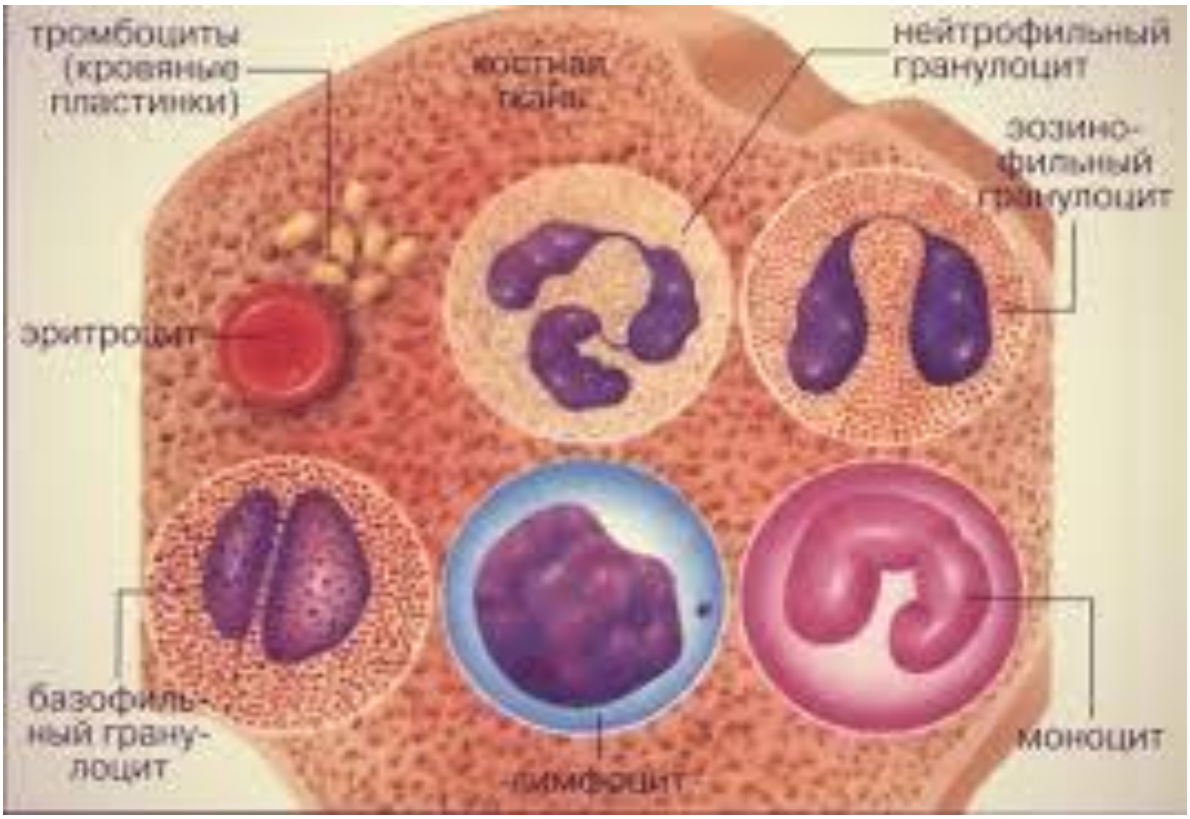
эритроцит заполнен гемоглобином

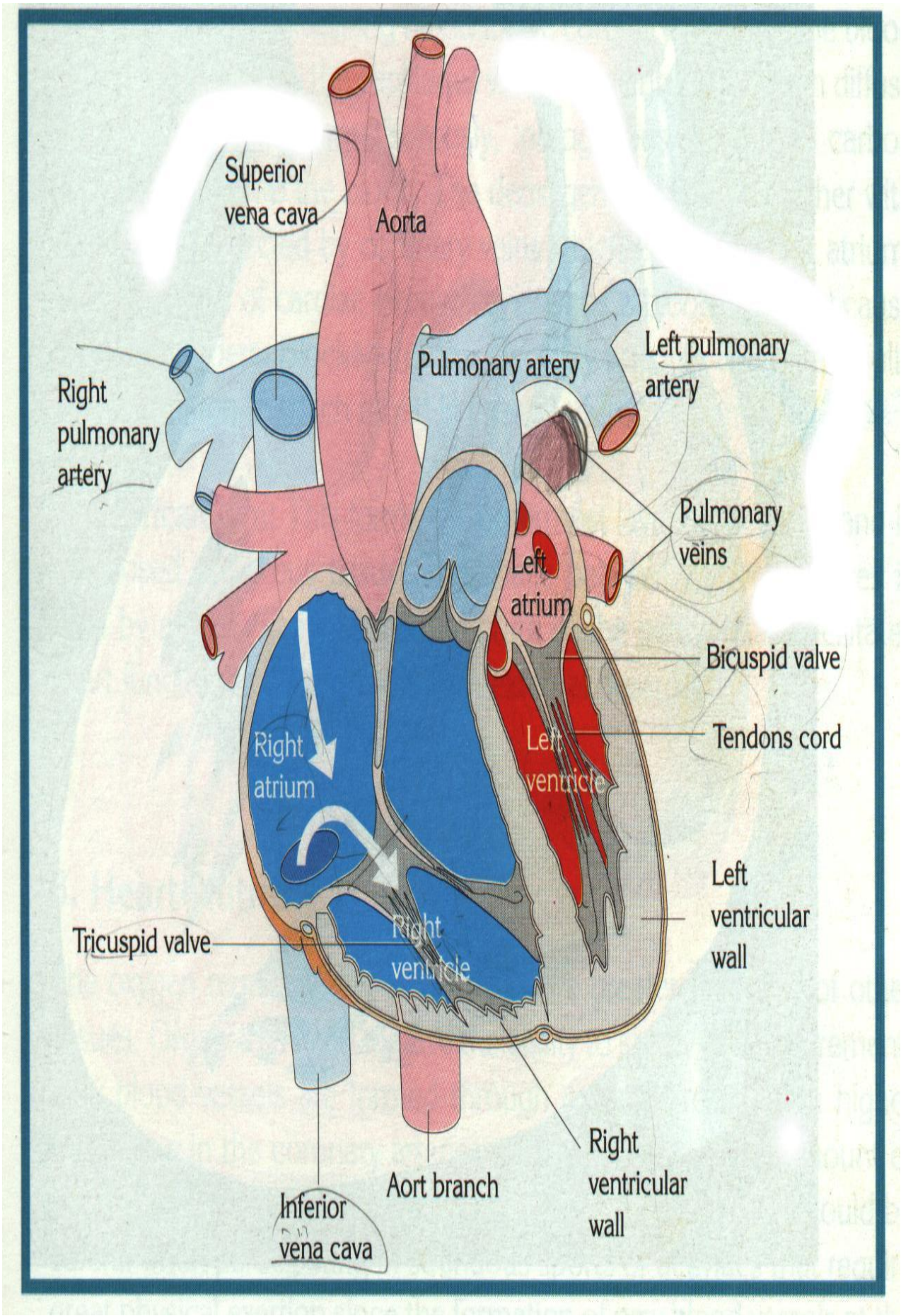
△ ЭРИТРОЦИТ наполнен специальным белком — гемоглобином

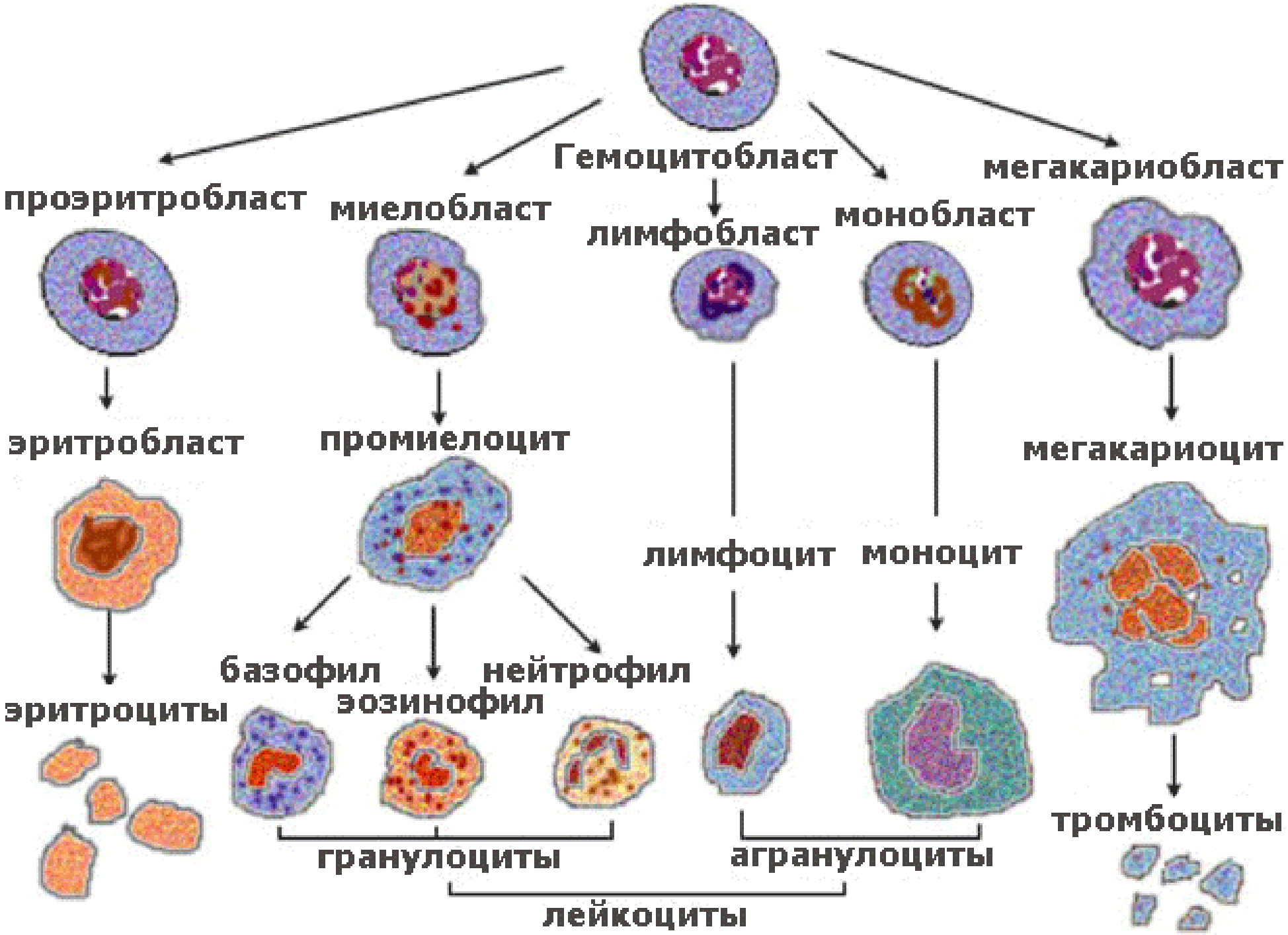
- 1. Гемоглобин состоит из четырех белковых нитей.
- 2. К каждой нити прикреплен один гем.
- 3. Гем содержит атом железа и способен удерживать одну молекулу кислорода.

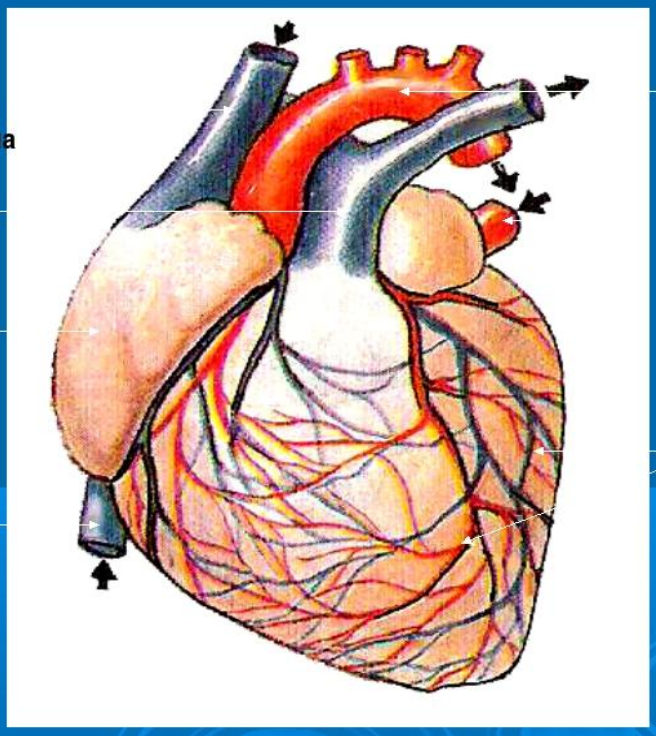


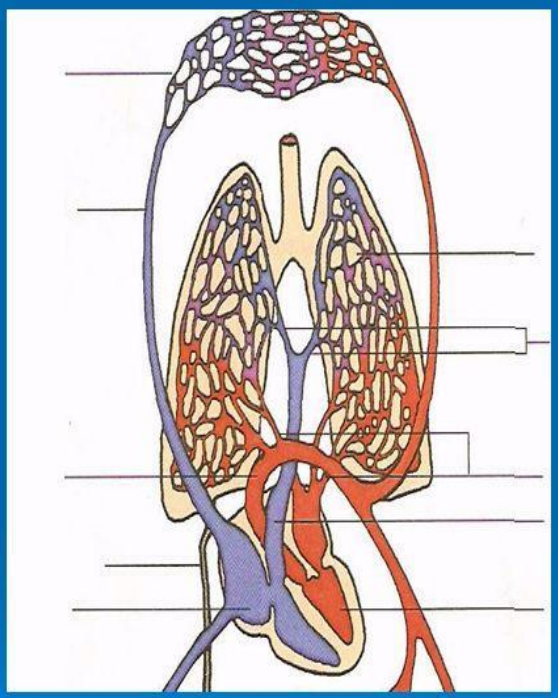
PPt4WEB.ru




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

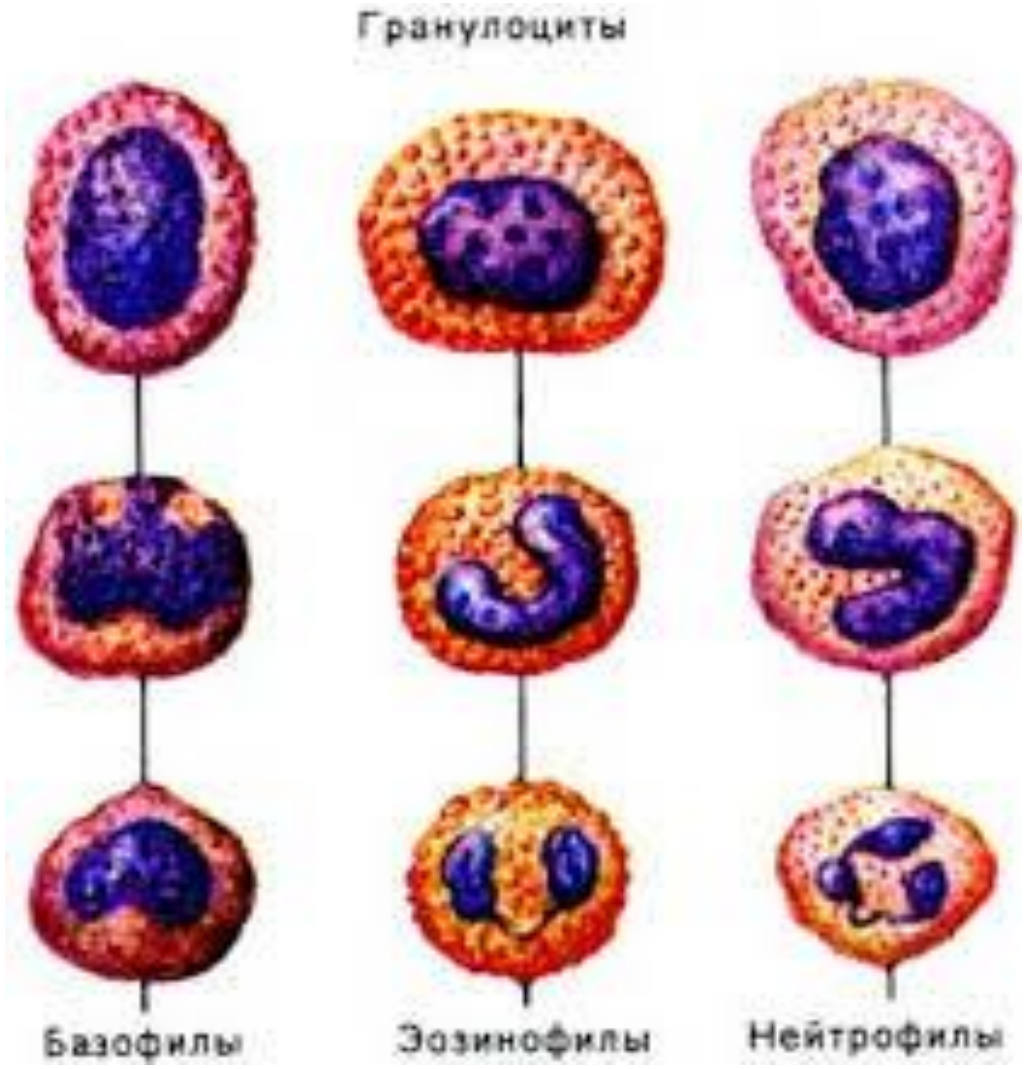

Гемоцитобласт
проэритробласт
миелобласт
лимфобласт
монобласт
мегакариобласт
эритробласт
промиелоцит
мегакариоцит
лимфоцит
моноцит
базофил
эозинофил
нейтрофил
эритроциты
тромбоциты
гранулоциты
агранулоциты
лейкоциты

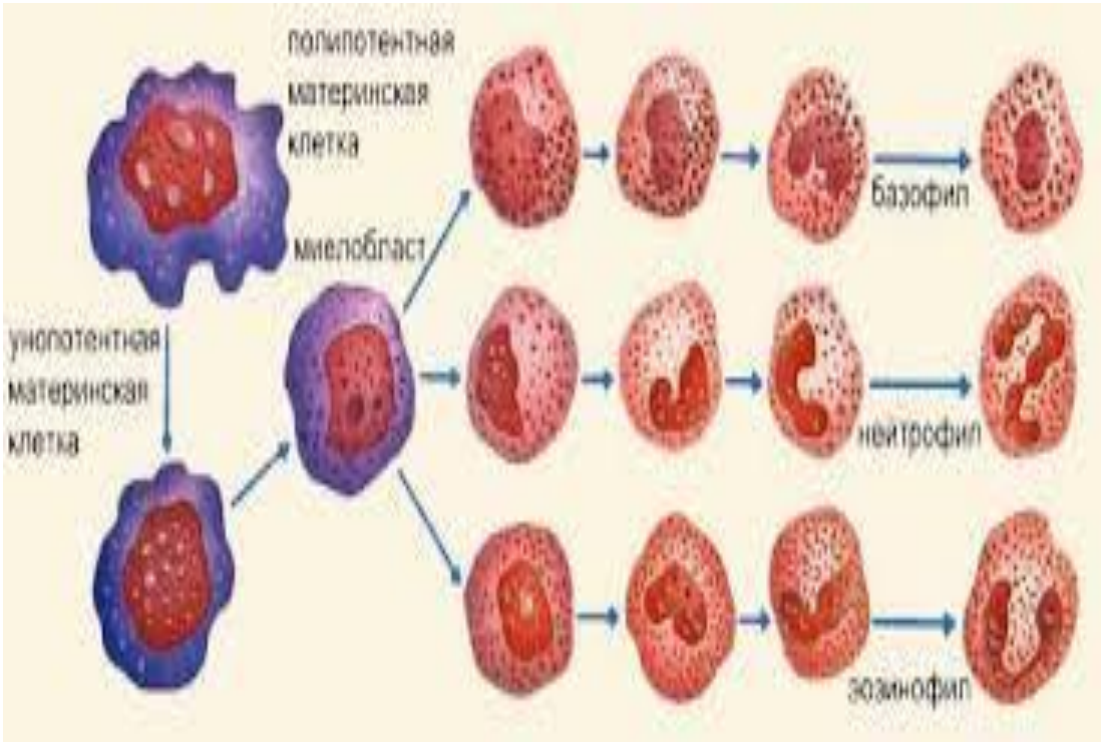




Малый круг:

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.


Гранулоциты
Базофилы
Эозинофилы
Нейтрофилы


полипотентная
материнская
клетка
миелобласт
унипотентная
материнская
клетка
базофил
нейтрофил
эозинофил