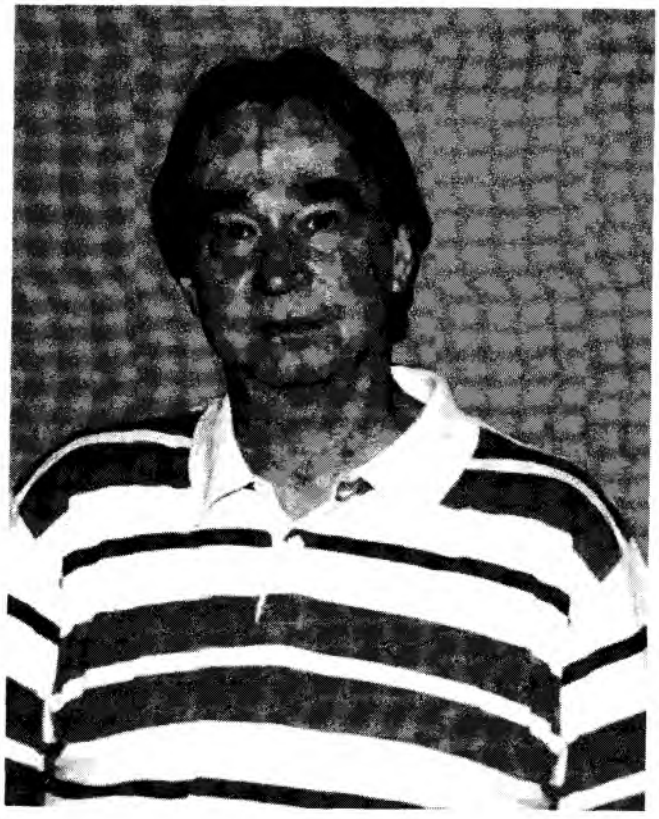
Чурилов Л. П., Строев Ю. И., Утехин В. И.,
Конашенок И. Н., Мясников А. А.,
С. Г. Ханикатт, У. Дж. Скоггинс

Английский язык для медиков

English for Medical Students

Руководство
с аудиодиском



We dedicate this project to the memory of
Dr. William Gene Scoggins, M.D., D.C.
(1948–2010)

To Alaskan native, Russian medical student,
Scottish bagpipes player,
Irish physician, Citizen of Universe,
to real Man, reader of Dostoevsky and friend of Russia,
to Bill, who always inspired us greatly and was with us
during hard times,
to One who has contributed much into this book
Bill, we will remember you forever...

**Л. П. Чурилов, В. И. Утехин, Ю. И. Строев,
И. Н. Конашенок, А. А. Мясников,
С. Г. Ханикатт, У. Дж. Скоггинс**

АНГЛИЙСКИЙ ЯЗЫК ДЛЯ МЕДИКОВ

*Рекомендовано для преподавания английского языка
студентам-медикам и для
последипломного образования врачей
редакционно-издательским объединением
Филологического факультета СПбГУ
и Межвузовским
редакционно-издательским советом Санкт-Петербурга
по медицинской литературе*

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2012

УДК 811. 111 (075.8)
ББК 81.2 Англ-932

Churilov L. P., Stroev Yu. I., Utekhin V. J., Konashenok I. N., Myasnikov A. A., Huneycutt S. G., Scoggins W. G.
English for Medical Students. Saint Petersburg: ELBI-SPb 2012. — 312 p. [the manual complemented by an Audio CD].

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This manual is created at Saint Petersburg State University. The work is done by the teachers of the Faculty of Medicine and the Faculty of Philology with Dr. S.G. Huneycutt from the USA and Dr. W.G. Scoggins from the UK. The Manual is complemented by an Audio CD. The recordings are done by British and American doctors. These doctors have good knowledge of British, American and Russian health care and medical education systems. There have not been any manuals of this type in the system of Russian medical education yet, though the similar books have been in use abroad and they are popular in many countries. This manual is based upon teaching English through Medicine. It contains the authors' texts which are the main source of input. These are the fragments from the orginal manuals on Pathophysiology and Internal Medicine. This manual is designated for teaching the second-year, the third-year and the fourth-year medical students. It helps learning medical subjects in parallels with learning English. The manual sets out to maximize medical students' abilities to perform speech activities in English and to encourage their future language development. It is easy and interesting in use. It contains lots of historical and medical facts and the biographies of outstanding English-speaking and Russian-speaking medical people. This manual can be successfully used in teaching medical post-graduate students and can be recommended for medical postgraduate education institutes. It can also draw the interest of those doctors who have received their medical education in Russia and who are planning to make a career in an English-speaking country. Among the authors of this manual there are the doctors who have been teaching Medicine in English in Russia and have been working successfully in English-speaking countries after they received their medical education in Russia. Medical foreign students learning in Russia can use this manual for better studying the courses of Pathology, Immunology, Internal Medicine and the History of Medicine. This manual is also aimed for self-studying and can be useful for doctors wishing to master English or to prepare for USML examination or similar tests. This manual includes 25 pictures, 25 tables, the bibliography of 37 references.

The peer review readers:

Vera A. Mayevskaya, Doctor of Philology, Professor, Chairperson of Foreign Language Dept. Saint Petersburg State University of Engineering and Economics (ENGECON).

Alexandr S. Gerd, Doctor of Philology, Professor, Head of Mathematical Linguistics Department at the Faculty of Philology, Saint Petersburg State University.

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Чурилов Л. П., Строев Ю. И., Утехин В. И., Конашенок И. Н., Мясников А. А., Ханикатт С. Г., Скоггинс У. Дж.

Английский язык для медиков. СПб.: ЭЛБИ-СПб, 2012. — 312 с. [руководство с электронным аудиодиском]

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Учебное пособие подготовлено преподавателями медицинского и филологического факультетов Санкт-Петербургского государственного университета под общим руководством заведующего кафедрой патологии медицинского факультета СПбГУ Л. П. Чурилова при участии доктора медицины С. Г. Ханикатта (США) и доктора медицины У. Дж. Скоггинса (Великобритания). Пособие использует аудиоматериал, основанный на аутентичном тексте и озвученный носителями языка–врачами профессионалами, изнутри знающими как англо-американское, так и с отечественное здравоохранение и медицинское образование. Книг такого типа в практике российской высшей медицинской школы еще не было, хотя за рубежом аналоги создавались и пользуются широкой популярностью. Пособие основано на преподавании языка через профессиональный предмет медицины. Оно содержит авторские тексты, представляющие собой фрагменты учебников патофизиологии и пропедевтики внутренних болезней. При его изучении на 2–4 курсах обеспечивается параллельность освоения соответствующих тем этих медицинских предметов с преподаванием английского языка, вовлечение студентов-медиков в иноязычную речевую деятельность путем интеграции языкового и профессионального обучения. Пособие написано живо и интересно, содержит много исторических и медицинских фактов, биографий известных англоязычных и русскоязычных ученых–медиков, которые используются в качестве обучающего материала, и может с успехом применяться при занятиях английским языком аспирантами и соискателями медицинских специальностей, рекомендовано для последипломного обучения врачей, представит интерес для медиков, получивших образование в России и планирующих или продолжающих свою карьеру в англоязычных странах, тем более, что его авторы либо преподавали медицину на английском языке в отечественных и зарубежных вузах, либо сами получили диплом врача в России и успешно работают по специальности в англоязычных странах. Студенты-иностранцы, обучающиеся в России, могут использовать книгу, как пособие для лучшего овладения отечественными курсами патологии, иммунологии, внутренних болезней и истории медицины. Книга может быть использована и при самостоятельных занятиях всеми медиками, стремящимися к свободному владению английским языком, готовящимися к экзамену USMLE или аналогичным экзаменам (25 рис., 25 табл., библ.: 37 ист.).

Рецензенты:

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Издается при финансовой поддержке
ЗАО ПНТ-ГСМ

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CONTENTS

INTRODUCTION

On the photo above you can see Dr. Yu. I. Stroev and Assoc. Prof. A. G. Vassil'ev with a group of American students during classes in Internal Medicine (1997)

This book to a certain degree is a fruit of the Russian-American M.D. programme, the first one in the long history of Russian and English-medium medical education, which was successfully performed in 1993–1998 at St. Petersburg State Pediatric Medical Academy with contribution of I. P. Pavlov St. Petersburg State Medical University. Some authors of this textbook were active members of this innovative project.

Dr. Leonid P. Churilov was the dean of English-medium M.D. programme,

Dr. Yury I. Stroev was a teacher of Internal Medicine,

Dr. Vladimir I. Utekhin was a lecturer in Pathophysiology,

Prof. Vera A. Mayevskaya was a teacher of Russian for American medical students.

Dr. Steven Huneycutt and Dr. William Scoggins both graduated from this programme and have got M. D. Diplomas in Russia.

The original texts in Pathophysiology, used in this textbook, were written for this unique educational project, by L. P. Churilov and V. J. Utekhin, the original texts in Internal Medicine are by Yu. I. Stroev and L. P. Churilov with participation of Dr. W. G. Scoggins and Dr. S. G. Huneycutt.

The authors keep a good memory of this remarkable event and created this book in order to share their experience with new generations of medical professionals. For this purpose medical doctors entered into collaboration with philologists:

Mr. Alexei Myasnikov, Ph.D. and Mrs. Irina Konashenok, Senior Lecturer.

On the photo above — first American alumni of Russian Medical School (1998). American students of St. Petersburg Pediatric Medical Academy and members of State Examination Board after the last state examination.

Standing, left to right: Dr. William G. Scoggins, Dr. Richard E. Staffel, Dr. Charlie Nguen, Dean of English-medium M. D. Programme Assoc. Prof. Leonid P. Churilov, Dr. Renald O. Dordon, Dr. Abbas I. Safieddine, Dr. James Inklebarger, Dr. Asqhar Baharahchi.

Sitting left to right: Dr. Robert J. Staffel, Assoc. Prof. Yury I. Stroev, Prof. Alexander T. Zhurkin, Prof. Lilia I. Levina, Prof. Oxana I. Korol'.

PREFACE OF THE AUTHORS

This textbook was written by the authors on the basis of their forty-five years of personal clinical and thirty years of pathophysiological experience. Admittedly, in the context of the tremendous amount of knowledge yet to be discovered, it is not a very long term.

St. Petersburg was founded more than three centuries ago, and today it is a modern city where students from many countries come to study an array of disciplines, including Medicine.

The field of Medicine has become international, and there is no national Medicine, as well as there is no national Mathematics or national Physics. Medicine is based on international laws of natural sciences. Every time a medical doctor checks the arterial blood pressure from one of his or her patients, this doctor (in any country of the world) applies the achievements of different national medical schools. This is plain medical fact, because the sphygmomanometer devise for this purpose was invented by an Italian doctor Scipione Riva-Rocci (1863–1937), the stethoscope used for this procedure was invented by a French physician René-Théophile-Hyacinthe Laennec (1781–1826) and the physiological phenomenon which produces the specific sounds heard for arterial blood pressure check was discovered by a Russian surgeon Nicolai Sergeievich Korotkov (1874–1920).

So, in a simple medical procedure, the achievements of three different national medical schools are combined, and the same principle applies in other areas of medicine. After the years of medical teaching, learning, practice and research in Russia, USA, Great Britain, Irish Republic, Romania, Japan, China, Canada, Sweden, Finland, Holland and few other countries, with experience of instructing medical and dentistry students from almost 60 different states, we do believe that the principles of medical education are similar all over the world.

In our current teaching, at the Medical School of St. Petersburg State University, we subscribe to the principles of early contact with patients, full integration between basic sciences and practical medicine, and the interpretive interdisciplinary approach which combines the concepts and findings taken from different medical disciplines.

Although the Science of Medicine is similar worldwide, the sphere of Health Care still is divided with national borders and has pretty much of specifics in every country, with their different legislations, standards, traditions, mentality and cultures. Even in the epoch of globalization this should be taken in account by international medical students and

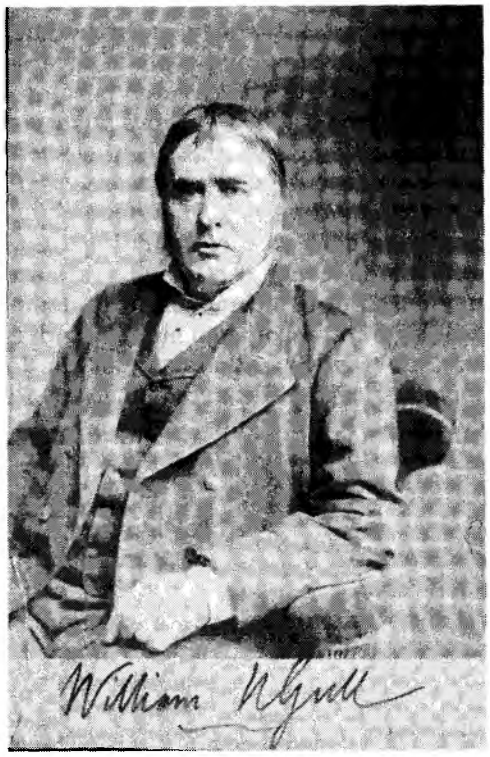

Fig. 1. Sir William W. Gull

medical doctors migrating for studies, research or employment. The object, which is called "white thrombus" on continental side of English Channel, is at the same time called "platelet plug" in Britain and all its former colonies. It does not mean that medical doctors living on a particular shore of the sea are more (or less) sophisticated. But it means difference in their thesaurus and in their traditions.

The main foundation for clinical thinking is a clinical language. But this great prerequisite of medical professionalism is not identical in different countries and even in the same country, but of various specialties and occupations of medicine.

In order to improve the performance of international medical students and make the tasks of the guest physicians or visiting scientists easier, we have composed this textbook both for Russian and Foreign readers, persuading the goal to acquire medical skills.

Three centuries ago, the Emperor of Russia, Peter the Great, had founded the city of St. Petersburg (1703) and — a bit later — also established our University (1724). His idea was to create a place that was suitable for hybridization and interaction between the Russian spirit and European culture. That is why today, St.Petersburg is probably the right place to study Medicine. Here you can combine the achievements of Russian and European cultures, with Russian and

International Medicine. The University puts together the intellectual efforts and creative potential of many people of diverse ethnic origin. In our School languages contribute the greater part of M.D. Programme curriculum, than anywhere else. The authors wish you success on your way to medical professionalism and great achievements in your future career of physician. *In order to accomplish this goal modern medical doctor has to be a multilingual person.* The great physicians of the past, those giants, whose shoulders we are standing on, in majority were polyglots. Our textbook is composed of several issues, dedicated to professional language used in Basic and Clinical Medicine. First issue is based on Pathophysiology and recommended for studies in 4th or 5th term of M.D. Programme, along with Pathology. Let the following words of famous English physician Sir William Whitney Gull (1816–1890) — fig. 1; p. 11, accompany you on your way:

"The road to a clinic goes through the pathologic museum and not through the apothecary's shop"

FOREWORD OF THE REVIEWER

In following up of what has been said by the colleagues the objectives they set designing this unique textbook for medical students are in fact challenging and rewarding. In the thick of day — to-day teaching, trying to cram endless syllabus into few hours we try to find balance relying on something tangible under our finger-tips. It is an attempt to overcome endless "must know", "should be able", etc to be inserted within a week's two or four academic hours. Let us being at the chalkface give a thought to how to make teaching process really intelligible and tuitional.

It is the truth universally acknowledged from the time immemorial that three factors are at work when we really want to succeed in teaching: a good textbook, a talented teacher and a diligent student. But let us be realistic: a perfect textbook is a rare thing even these days when the access to information you need is actually infinite since it is not a compilation of the material for studies but the way you think, treat the students and are interested in the pupils' advancement and know how to put these things together. The teacher should measure how heavy the load on the shoulders of his students is, how fast the students should be to bear the load and,finally, what rewards the students will have in the end.

The intellectual product of our colleagues is the sample of a textbook combining advantageous approach to teaching with many years of their medical experience. They made efforts to smooth away difficulties that might prevent from proper interactivity and have regular and reliable feedback which plays a key role in modern management of the teaching process. The textbook is a well developed system aiming at continuous improvement of various skills and based on gradual expansion of general vocabulary, terms and professional idiomatics. Great importance is attached to phonetic skills which are developed from lesson after lesson by means of intensive listening to original recordings performed by the native speakers — M.D. William G. Scoggins and M.D. Steven G. Huneycutt, which is of great value in itself. The effect is achieved, primarily, by the recurrence of the samples after the speakers.

Student — centered principle taking as its starting point the needs and interests of the learners aims at involving them as fully as possible

Part I

PATHOPHYSIOLOGY

(for the students of 4th and 5th terms)

Module 1
REACTIVITY and RESISTANCE

Unit 1
REACTIVITY

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

acinus	ацинус (1. легочный мешочек; 2. железистый ацинус)
adenylate-cyclase receptor subunit	рецепторная субъединица аденилат-циклазы
catalytic subunit	каталитическая субъединица
complementary	комплементарный (обладающий однозначным структурным соответствием), распознающий, дополнительный
endocrine	эндокринный
immune	иммунный, невосприимчивый, обладающий иммунитетом
inflammation	воспаление
microcirculation vessels	микроциркуляторные сосуды
nephron	нефрон (структурно-функциональная единица почки)
nervous	нервный (о системе); относящийся к нерву
ontogenetic	онтогенетический
organogenesis	органогенез (формирование органов в пренатальный период)
parenchyma	паренхима (совокупность основных функционирующих элементов внутреннего органа)
pathogenic	патогенный, болезнетворный
reactivity	реактивность
stroma	строма (соединительнотканная опорная структура органа или опухоли)
trophic	1) трофический (связанный с митозами и/или синтезом ДНК); 2) алиментарный (связанный с питанием)

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

REACTIVITY of the organism is the ability to respond adequately to changing conditions of the internal and external environment. The adequacy of the reaction is by no means absolute. The reaction is not always useful. It is more or less correspondent to the nature and amount of the acting factors. Mechanisms of reactivity are relatively beneficial and potentially pathogenic.

REACTIVITY is, in fact, a complete repertoire of reactions that the organism is capable of, including all normal hereditary determined reactions, as well as acquired reactions based on ontogenetic experience. REACTIVITY is not just a library of programs but an internal manager, *dealing with* and *selecting from* and *blending* these programs. The reactivity is the ability to be adaptive.

Although the concept of "reactivity" is associated with the organism as a whole, the concrete mechanisms of reactivity are realized at a certain level of the structure in question. There are several levels of reactivity distinguished in the organism: molecular, subcellular, cellular, tissue, organ, whole-body and population ones. The complementary interactions are particularly important at a molecular level. For instance, the adenylate-cyclase receptor subunit recognizes its peptide bio-regulator in accordance with the key-lock principle resulting in the activation of a catalytic subunit. The best example of a tissue level of reactivity is inflammation, i. e., local response of the vascularized tissue to any acute damage. The formation of system responses starts with the onset of the period of organogenesis. An important component of the tissue and organ substratum of reactivity is the structure-functional unit of the organ (a nephron, a liver acinus, a pancreatic acinus, a lung acinus etc.). The reliability of the system is provided by doubling of the function of a great number of structure-function units the organ consists of. The connective tissue elements of the organ stroma form a group around microcirculation vessels carrying out the supporting trophic and protective function for elements of organ parenchyma. The basis of the functioning of every system that integrates the mechanisms of reactivity (nervous, endocrine, immune) is the complementary interaction of the regulator with its receptor-discriminator system.

3. Do the following statements agree with the information given in the text?

Write

TRUE *if the statement agrees with the information*
FALSE *if the statement contradicts the information*

1. The reactivity of the organism is the ability to give an adequate response to changing conditions of the internal and external environment.
2. Each response is always useful.
3. All reactivity mechanisms are beneficial.
4. Reactivity displays a total repertory of reactions the organism is capable of.
5. The concrete mechanisms of reactivity are realized at the level of the whole organism.
6. There exist several levels of reactivity: molecular, subcellular, cellular, tissue, organ, whole-body and population ones.
7. Complementary interactions are especially obvious at a molecular level.
8. The functional basis of every system that integrates the mechanisms of reactivity (nervous, endocrine, immune) is the main interaction of the regulator with its receptor — discriminator system.

4. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner.

1. *What is reactivity?*
2. *Is the adequacy of the organism's reaction absolute?*
3. *Is reactivity always useful?*
4. *Does reaction depend on the nature and amount of active factors?*
5. *Can it be pathogenic?*
6. *What are the mechanisms of reactivity?*
7. *Why is reactivity compared with a manager?*
8. *What levels of reactivity are distinguished in the organism?*
9. *Can you illustrate any interactions at different levels of reactivity?*
10. *At what stage of ontogenesis does the formation of system responses start?*
11. *What role does a structure-functional unit of the organ play?*
12. *How is the reliability of the system provided?*

5. Match the words and expressions in column A with the words and expressions in column B.

A	*B*
the reactivity	of reactivity
internal and external	elements
mechanisms and levels	receptor
hereditary and acquired	environment
ontogenetic	experience
library	vessels
internal	principle
adenylate-cyclase	of the organism
peptide	reactions
key-lock	damage
result	parenchyma
the activation	of a catalytic subunit
acute	manager
tissue and organ substratum of	reactivity
connective tissue	system
the organ	stroma
microcirculation	of programs
elements of organ	in
receptor-discriminator	bio-regulator

6. Complete the sentences below.

1. The reactivity of the organism is the _____ to respond to changing conditions of the _____ environment.
2. Mechanisms of reactivity are relatively beneficial and potentially _____.
3. The reactivity is the ability to be _____.
4. The concrete mechanisms of reactivity are realized at a certain level of the structure _____.
5. The complementary interactions are particularly important at a _____.
6. The best example of a tissue level of reactivity is inflammation, i. e., local response of the vascularized tissue to any acute _____.
7. The formation of system responses starts with the onset of the period of _____.
8. An important component of the tissue and organ substratum of reactivity is the structure–functional unit of the _____.
9. The reliability of the system is provided by _____ of the function of a great number of structure–function units the organ _____.
10. The basis of the functioning of every system is the _____ interaction of the regulator with its _____ system.

7. Unjumble the words below.

oansmrgi
meenronntiv
teacinro
tannectioir
detpiep
minlmationaf
ustratumbs
lyreliabiit
ofuctinn
yehcparman

8. Read the task card below.

Describe the main peculiarities of reactivity following the plan:
What is reactivity?
What reactivity levels are distinguished in the organism?
How is the reliability of the system provided?

9. Now, using the prompts from exercise 8, talk on the topic: REACTIVITY.

10. Translate into English.

1. Реактивность организма — это его способность адекватно реагировать на изменяющиеся условия внешней и внутренней среды.
2. Механизмы реактивности характеризуются как относительно полезные и потенциально патогенные.
3. Конкретные механизмы реактивности реализуются на определенных структурных уровнях организма.
4. Выделяют несколько уровней реактивности: молекулярный, внутриклеточный, клеточный, тканевой, уровни органа, организма и популяции.
5. Наиболее ярким примером реактивности на тканевом уровне является воспаление, которое представляет собой местную реакцию васкуляризованной ткани на любое повреждение.
6. Формирование системных реакций начинается с началом процесса органогенеза.
7. Надежность системы реакций обеспечивается дублированием функций большого количества структурно-функциональных элементов органа.
8. Соединительнотканные элементы стромы органа группируются вокруг микроциркуляторных сосудов и выполняют опорно-трофическую и защитную функции в отношении элементов его паренхимы.

9. В основе функционирования каждой интегрирующей механизмы реактивности системы (нервной, эндокринной, иммунной), лежит комплементарное взаимодействие регулятора и рецепторно-дискриминаторного аппарата.

Unit 2
RESISTANCE

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

cardiovascular	сердечно-сосудистый
confrontation	противостояние, сопротивление
hibernation	гибернация, зимняя спячка
hypercapnia	гиперкапния (повышенное содержание двуокиси углерода в крови)
hypophysis	гипофиз
hypothalamus	гипоталамус, гипоталамическая область, подбугорная область, подбугорье
hypothermia	гипотермия (1. пониженная температура тела; 2. искусственное понижение температуры тела с лечебными целями)
hypoxia	гипоксия
integument	покровы тела
opioid peptides	опиоидные пептиды
tolerance	переносимость, толерантность
phagocytosis	фагоцитоз
resistance	резистентность
respiratory	респираторный, дыхательный

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo.

After that read the text aloud, trying to imitate the intonation.

RESISTANCE is a measure of the defiance of the organism to a certain factor. Unlike reactivity which is a *qualitative* property, resistance can be measured *quantitatively*. REACTIVITY is a full set of reactions the organism is capable of, while RESISTANCE is a concrete manifestation of reactivity. Thus, during hibernation, opioid peptides and somatostatin generated in the organism of an animal depress the activity of the hypothalamus and the hypophysis, and many manifestations of reactivity are depressed as well. At the same time resistance to different pathogenic factors (e. g., hypoxia, hypothermia, hypercapnia, infections, poisoning) is distinctly increased. Resistance cannot be universal because the nature of pathogenic factors is different. At the same time, some pathogenic factors are widespread, acting in many pathologic processes, that is why resistance to the factor of such kind is actually the ability of the organism to withstand a great many unfavorable factors. Thus, since hypoxia is the most wide-spread pathologic process always accompanying death, the stress improving resistance to acute hypoxia will adapt the organism nonspecifically to a large number of various influences. That is why the term "nonspecific resistance" is *de facto* an "anti-hypoxic resistance". Resistance to hypoxia depends on the ability of the respiratory and cardiovascular system to bring oxygen to tissues, as well as on the capacity of the tissues to do without oxygen, i. e., to exist anaerobically. Resistance may be provided by the passive mechanisms of tolerance (e. g., barriers of integument, or incapsulation) and active mechanisms of confrontation (e. g., phagocytosis and an immune response).

3. Do the following statements agree with the information given in the text?

Write

TRUE	***if the statement agrees with the information***
FALSE	***if the statement contradicts the information***

1. Defiance is the synonym of resistance.
2. Resistance is a quantitative property.
3. Reactivity is a concrete manifestation of resistance.
4. Resistance is the ability of the organism to withstand a great many unfavorable factors.
5. The term "nonspecific resistance" actually means "anti-hypoxic resistance".
6. Resistance can be provided by active mechanisms of confrontation (e. g., phagocytosis, an immune response) and passive mechanisms of tolerance (e. g. integument barriers, incapsulation).

4. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner.

1. *What is resistance?*
2. *What is the difference between reactivity and resistance?*
3. *Why can't resistance be universal?*
4. *Can resistance be measured quantitavely?*
5. *Is hypoxia the most wide-spread pathological process accompanying death?*
6. *What does resistance to hypoxia depend on?*
7. *Why is resistance to hypoxia treated as the ability of the organism to withstand a great many unfavourable factors?*
8. *By what passive and active mechanisms can resistance be provided?*
9. *What is the difference between tolerance and resistability?*

5. Match the words and expressions in column A with the words and expressions in column B.

A	*B*
defiance	death
qualitative	peptides
manifestation	response
opioid	property
depress	mechanisms
resistance	resistance
pathologic	without oxygen
to withstand	system
acute	of the organism
nonspecific	to different pathogenic factors
depend	of reactivity
cardiovascular	a great many unfavorable factors
to do	processes
passive	the activity of the hypothalamus and the hypophysis
immune	on

6. Complete the sentences below.

1. Resistance is a measure of the _____ of the organism to a certain factor.
2. Resistance can be measured _____.
3. Resistance is a concrete manifestation of _____.

4. Resistance cannot be universal because the nature of _____ factors is different.
5. Hypoxia is the most wide-spread pathologic process always accompanying _____.
6. Resistance to hypoxia depends on the ability of the _____ and _____ system to bring oxygen to tissues, as well as on the capacity of the tissues to do without oxygen.
7. Resistance may be provided by the _____ mechanisms (e. g., barriers of integument, incapsulation) and _____ mechanisms (e. g., phagocytosis, an immune response).

7. Unjumble the words below.

ristanscee
morgaisn
yectivirta
heyrnationb
dpetpie
mypothalaush
xhpoiay
gpthoenica
eautc
vliardasccuaro
ssreepon

8. Read the task card below.

Describe the main peculiarities of resistance following the plan:
What is resistance?
What is the difference between resistance and reactivity?
By what mechanisms may resistance be provided?

9. Now, using the prompts from exercise 8 talk on the topic: RESISTANCE.

10. Translate into English.

1. Резистентность отражает устойчивость организма к конкретному патогенному фактору.
2. Резистентность может быть измерена количественно.
3. Реактивность включает в себя весь набор доступных организму ответов, тогда как резистентность является конкретным проявлением реактивности.
4. При гибернации опиатные пептиды и соматостатин в организме некоторых животных тормозят активность гипоталамо-гипофизарной системы.
5. Резистентность не может быть универсальной из-за различий в природе патогенных факторов.

6. Поскольку гипоксия встречается при множестве различных болезней и сопровождает гибель организма, стресс как фактор, повышающий резистентность к острой гипоксии, будет неспецифически адаптировать организм к крайне широкому кругу разнообразных влияний.
7. Термин «неспецифическая резистентность» может быть уточнен как «антигипоксическая резистентность».
8. Резистентность включает факторы пассивной переносимости (барьерная роль покровов тела, инкапсуляция) и активного сопротивления (фагоцитоз, иммунный ответ).

Unit 3
ONTOGENESIS OF REACTIVITY

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

acidosis	ацидоз
adrenal medulla	мозговое вещество надпочечника
basal metabolic rate (BMR)	основной обмен
bradycardia	брадикардия
brown fat	бурая жировая клетчатка
cardiomyocyte	кардиомиоцит
catatoxic reactions	кататоксические реакции
catecholamine	катехоламин
epinephrine	адреналин, эпинефрин
fetal hemoglobin (Hb)	фетальный гемоглобин
phosphofructokynase	фосфофруктокиназа
lactate	лактат
pathogenic	патогенный, болезнетворный
sanogenic	саногенный, оздоравливающий
secretory	секреторный (относящийся к процессу или продуктам секреции)
syntoxic reactions	синтоксические реакции

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo.

After that read the text aloud, trying to imitate the intonation.

According to Hans Selye[1], the term "catatoxic reactions" means active protection while the term "syntoxic reactions" is used to define passive protection. Catatoxic and syntoxic reactions are intermingled parts of any adaptive process.

The dependence of many protective reactions on central mechanisms integrating reactivity is not too strong. Being adaptive at the level of the cell, tissue and organ, they may be pathogenic at the level of the organism as a whole. Thus, the effects of the mediators of inflammation within the limits of the inflammed zone may be sanogenic; however, the effects of their massive penetration into the systemic circulation are pathogenic. Mechanisms of reactivity are only relatively beneficial and potentially pathogenic.

Ontogenesis is the process of asynchronous expression and repression of the unfolding genetic programs. That is why individuals of different ages have different reactivity. New born human beings, like vertebrate and invertebrate animals at early stages of their ontogenesis, are more resistant to acute hypoxia (Sirotinin's[2] rule), since:

1) isoenzymes of PhFK (phosphofructokynase) are insensitive to increased levels of lactate acidosis;

2) the amount of fetal Hb in blood circulation is much greater;

3) the absolute basal metabolic rate (BMR) is markedly lower;

4) there are certain peculiarities in the catecholamine status and the amount of receptors of catecholamines on cardiomyocytes, such as:

a) secretion of catecholamines by the adrenal medulla cells occur in response to hypoxia in secretory cells;

b) epinephrine/nor-epinephrine ratios in adrenal medulla secretions are 1:1; compared to predominance of epinephrine in adults. Nor-epinephrine effectively increases surfactant secretion in lungs. It is also significant for cold adaptation of neonates via urgent lipolysis in brown fat.

c) the number of epinephrine receptors in cardiomyocytes is not sufficient for induction of tachycardia. Bradycardia is more effective response for hypoxia for fetal conditions. All this is also true for anti-hypoxial mechanisms of diving animals, like seal or cachalot.

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[1] **Hans Hugo Bruno Selye** (see p. 31 below), Austrian-Hungarian (after 1932 — Canadian) pathophysiologist and endocrinologist, born January 26, 1907, Vienna, Austria-Hungary, died October 16, 1982, Montreal, Canada. Discovered stress, formulated the concept of eustress and distress, experimentally proved the existence of polyetiological diseases.

[2] **Sirotinin Nicolay Nicolaevich** — Russian pathophysiologist, born November 26, 1896, Saratov, Russia, died April 4, 1977, Kyev, USSR. Founder of Aerospace Pathophysiology, made outstanding contribution in research of high altitude disease, invented stepwise principle of mountain acclimatization.

3. Do the following statements agree with the information given in the text?

Write

TRUE ***if the statement agrees with the information***
FALSE ***if the statement contradicts the information***

1. The term "catatoxic reactions" is used to define passive protection while the term "syntoxic reactions" is used to define active protection.
2. Catatoxic and syntoxic reactions are parts of any adaptive process.
3. The dependence of many protective reactions on central mechanisms integrating reactivity is very strong.
4. The effects of the mediators of inflammation within the limits of the inflammation zone may be pathogenic; however, the effects of their penetration into the circulation are sanogenic.
5. Mechanisms of reactivity are only relatively beneficial and potentially pathogenic.
6. Ontogenesis is the process of asynchronous repression and expression of the unfolding genetic programmes.
7. Individuals of different ages have different reactivity.
8. New born human beings, unlike vertebrate and invertebrate animals at early stages of their ontogenesis, are more resistant to acute hypoxia.

4. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner.

1. *Is the term "catatoxic reaction" used to define passive protection?*
2. *What does the term "catatoxic reactions" express?*
3. *Parts of what process are catatoxic and syntoxic reactions?*
4. *How strong is the dependence of protective reactions on central mechanisms integrating reactivity?*
5. *How may the mechanisms of reactivity be characterized in terms of usefulness?*
6. *What is ontogenesis?*
7. *Why are new born human beings more resistant to acute hypoxia then adults?*
8. *Who was Nicolay Sirotinin and what is his contribution into ontogenetic aspects of reactivity doctrine? (Use the text* ***"Medicine through Biographies"*** *below, p. 33)*
9. *Who was Hans Selye and what is his contribution into reactivity doctrine? (Use the text* ***"Medicine through Biographies"*** *below, p. 31)*

5. Match the words and expressions in column A with the words and expressions in column B.

A	*B*
catatoxic and syntoxic	of reactivity
active	hypoxia
adaptive	human beings
the level	cells
within the limits	metabolic rate
mechanisms	reactions
unfolding	receptors
new born	process
acute	protection
basal	of the inflammation zone
adrenal medulla	of the cell, tissue and organ
epinephrine	genetic programmes

6. Complete the sentences below.

1. The term "catatoxic reactions" means _____ protection while the term "syntoxic reactions" is used to define _____ protection.
2. Catatoxic and syntoxic reactions are parts of any _____ process.
3. The _____ of many protective reactions on central mechanisms integrating reactivity is not too strong.
4. The effects of the mediators of inflammation within the limits of the inflammation _____ may be sanogenic; however, the effects of their massive penetration into the systemic circulation are _____.
5. Mechanisms of reactivity are only relatively _____ and potentially pathogenic.
6. Ontogenesis is the process of asynchronous expression and _____ of the unfolding _____ programmes.
7. Individuals of different ages have different _____.
8. New born human at early stages of their _____ are more resistant to _____ hypoxia.

7. Unjumble the words below.

cttcaaoxi
tiynoxcs
veayctiitr
nthoicgepa

csnogenia
oontngeesis
yphoxai
sbaal
hepieprinen

8. Read the task card below.

Describe the main peculiarities of ontogenesis following the plan:
What are catatoxic and syntoxic reactions?
What is ontogenesis?
Why are new born human beings more resistant to acute hypoxia then adults?

9. Now, using the prompts from exercise 8 talk on the topic: REACTIVITY AND ONTOGENESIS.

10. Translate into English.

1. Кататоксические реакции обеспечивают активную защиту, а синтоксические реакции — пассивную защиту.
2. Приспособляемость достигается в соотношении кататоксических и синтоксических реакций.
3. Многие защитные реакции мало зависят от центральных механизмов, интегрирующих реактивность.
4. Будучи адаптивными для организма как целого, защитные реакции могут быть патогенны для его элементов, и наоборот.
5. Действие медиаторов в пределах очага воспаления может быть саногенным.
6. Онтогенез — это процесс неодновременной экспрессии и репрессии развертываемых генетических программ.
7. Представители разных возрастных групп имеют разную реактивность.
8. Новорожденные имеют повышенную устойчивость к острой гипоксии, что известно как «правило Сиротинина».

Medicine Through Biographies

Hans Hugo Bruno Selye (1907–1982)

Hans (Janoš) Selye, an eminent endocrinologist and pathophysiologist, known as "the father of stress", was born into the family of Dr. Hugo Selye, a Hungarian military surgeon. His mother Maria Felicita was an Austrian noble lady. He received his basic schooling in Komárno, Slovakia, where his father was stationed during the First World War. His mother made him a polyglot speaking fluently four European languages: English, French, Hungarian and German. He began his studies at the medical faculty in Prague, but also took part of the curriculum at the Universities of Paris and Rome. Later Selye recalled that medical professors mostly instructed students in how to distinguish between different diseases in diagnosing. But for him the most intriguing question was of an absolutely opposite nature: "Why are different illnesses so similar in their initial symptoms?" As early as in 1926, still only in his second year of medical school, Selye began developing his now-famous theory of universal response and its influence on ability to cope with and adapt to the pressures of injury and disease. He graduated from the German University of Prague in 1929 and subsequently obtained a position of assistant at the histological laboratory of the Institute of Experimental Pathology. He held this position until 1931, the year he obtained his Ph.D. That year he received a Rockefeller scholarship and moved to Johns Hopkins University, where he began his research. The discovery of stress which made Selye the most outstanding pathophysiologist of XX century and most cited medical scientist of his time (with 320 000 of references), resulted from a project which seemed to end in a complete fiasco. The young researcher studied the influence of different organ extracts on experimental rats in the hope to reveal the specific hormone tropic to ovary. But all was in vain, because various experimental and control stimuli only produced almost identical non-specific changes. Selye's supervisor advised him to quit and "cease to study the Pharmacology of dirt", but the researcher interpreted these results in accordance with an absolutely original concept of "general adaptation syndrome"

and demonstrated that typical neuroendocrine response involving suprarenal glands and pituitary is an inevitable element of any disease and, moreover, obligatory component of any adaptive reaction for a variety of extraordinary stimuli. Selye's contribution into General Nosology is difficult to ovestimate. He coined several crucial terms in Pathology: stress, eustress, distress, catatoxic and syntoxic reactions, introduced the principle "Acton causes Reacton", which illustrates the bilateral character of cause in Pathology. He authored the concept of the "diseases of failed adaptation" and experimentally proved the real existence of polyetiological diseases, using the original model of calciphylaxis.

Selye conducted studies on laboratory rats and found that the same physical responses are displayed by animals when they were put under different stressors. He concluded that stress plays some role in the development of every disease and that failure to cope with "stressors", which can be any non-routine stimuli, can result in "diseases of adaptation" such as peptic ulcer and high blood pressure. He claimed that it is not stress that harms us but distress or failure of stress mechanisms.

In 1932 he settled in his final academic destinations, Montreal, taking over a lectureship of biochemistry at the Mc Gill University. In Montreal Hans Selye worked during 50 years. He became Professor of Histology in 1941 and from 1945 he was the first Director of the Institute of Experimental Medicine and Surgery, Université de Montreal. He held this position until 1976. In 1979, Hans Selye and Alvin Toffler founded the world known Canadian Institute of Stress.

Bringing new notions and terms into the academic turnover in any area of knowledge and in all languarges — is a sign of supreme recognition and of utmost significance of a scientific discovery. When Selye was asked to present his paper in France, it turned out that there was no equivalent for "stress" to be found in French, so they coined one: *Le stress*. Similarly, when asked to speak in Germany and later in the Soviet Union, there was no German or Russian word for stress, so it was named "*Der Stress*" or "*стресс*". Selye's discoveries altered greatly not only experimental and clinical medicine; they also made deep influence on all human and behavioral Sciences.

Selye held three doctorates and was elected Doctor Honoris Causa 43 times at several universities in different countries, including USSR. He received numerous honours (M.D., Ph.D., D.Sc.) and wrote some 39 books in Medicine and Philosophy, more than 1,700 articles on stress and related items. Even between 1936 and 1951 he was 10 times nominated for Nobel Prize (later archives of Nobel Committee are still not available, otherwise we could certainly know much more nominations). However, the greatest pathophysiologist of twentieth century never was awarded a Nobel Prize in Physiology and Medicine, which many historians consider to be the second biggest mistake of

the Nobel Committee, after the rejection of Leo Tolstoy's candidature for the Prize in Literature (1906)[1]. The prize, bestowed Selye is much more honorable than even the Nobel Prize: he was entered into history with several eponyms (Selye's syndrome, Selye — Ovary phenomenon etc).

Nicolay Nicolaevich Sirotinin (1896–1977)

Only one pathophysiologist in the whole history of this basic medical discipline was awarded the prestigious "K. E. Tsiolkovsky's Gold Medal" for outstanding contribution into space research and cosmonautics. It was the renowned Soviet scientist Nicolay Nicolaevich Sirotinin. Sirotinin was born 26th November 1896 in the city of Saratov, on the Volga River. His father, also Nicolay Nicolaevich, was a mathematician, specializing in Statistics. He worked as a municipal official (secretary of Saratov City Duma) and was a person of leftist views combined with great energy: in his youth Sirotinin-senior was an active socialist revolutionary, in elderly years he became well-known for his progressive social projects. It was thanks to his enthusiastic initiative, supported by Prime Minister P. A. Stolypin (former governer of Saratov) that the Nicolaevsky University of Saratov was established in 1909. Previously this city had never had a university. New educational establishment began with a single faculty — the medical one. In 1921 Sirotinin-senior died of cholera. The Sirotinins had five children; all of them graduated from the newly established higher school and dedicated their careers to Biology and Medicine. Nicolay Sirotinin-junior was a 3rd child. In 1919 he graduated from the Faculty of Medicine, Saratov State University, where he was a disciple of an outstanding pathophysiologist Alexander Alexandrovich Bogomolets (1881–1946), who belonged to Mechnikov's school. After graduation the young scholar worked at *alma mater* under the guidance of his teacher until 1925 and later moved to Moscow following Bogomolets, who accepted a position there. In 1925–1929 Sirotinin subsequently held the positions of assistant professor and associate professor at the 2nd Moscow Medical Institute. In 1929 at Kazan State Medical Institute he became the Chairman of Pathophysiology Department (the oldest in Russia), thus returning to

[1] Leo Tolstoy was rejected for "lack of idealistic trend" in his novels, Nobel Committee preferred an Italian poet G. Carducci, an active freemason, nowadays completely forgotten beyond Italy

Volga River. Main area of Sirotinin's research was reactivity and resistance of human organism. He paid great attention to the problems of allergy and immunity and to the evolutionary interplay of these phenomena, especially as regards the mechanisms of anaphylaxis and rheumatic fever. But, his most valuable discoveries were made in the field of hypoxia and anti-hypoxic resistance. He organized several research expeditions in the high altitudes of Pamir and Caucausus mountains and established a permanent research station on the peak of Elbrus, the highest mountain of Europe. This happened for the first time in the whole world history of medicine. Sirotinin has demonstrated that all immature animal forms are more resistant to acute hypoxia than mature ones (for example, puppies compared to dogs, larvae compared to insects or tadpoles in comparision to frogs). This phenomenon is now known as "Sirotinin's rule". Also he has discovered the protective role of hypercapnia during acute hypoxia and the aggravating influence of hypocapnia on the course of hypoxia, revealed the increase of body resistance under the combined influence of hypercapnia, hypoxia and hypothermia, and explored the mechanisms of hibernation. The stepwise method of high mountain acclimatization and a special mixture for the adaptation of the mountain climbers ("sirotinovka") were invented by him as well. These discoveries were of great practical significance and soon were broadly applied in aerospace, navy and military medicine. In 1934 Sirotinin moved to Kiev, where he worked at the Institute of Physiology for all the rest of his life. In 1939 he was elected corresponding member of the Ukranian Academy of Sciences. During World War II Kyev was temporarily occupied by German troops and Sirotinin was captured by the Nazis. Since he already was a scholar of European fame, they suggested him to be the director of some research institute in Reich. Sirotinin categorically rejected the collaboration, although it was a fatally dangerous act. The Nazis tried to spoil his good reputation. In order to defame him, they spread misinformation about his "positive contacts" with new administration, after that Sirotinin was secretly transported to the Soviet territory and liberated. But the truth about the patriotic act of Sirotinin was revealed. After the war Nicolay Nicolaevich became one of the main experts in the biomedical part of Soviet space research program. In 1957 he became a member of the Soviet Academy of Medical Sciences. It was he who was in charge of the training of Soviet cosmonaut Alexei Leonov before his first heroic trip to open Cosmos (1965). For a long time Sirotinin was the Chairman of the Pathophysiology Department at Kiev State Medical Institute. He was worldwide leading specialist in pathogenesis and treatment of high altitude disease. Some of his gerontological ideas had predicted the dependence of oxygen metabolism on senescence, which is now considered to be proven.

Module 2

SEX CHROMATIN DETERMINATION IN THE DIAGNOSTICS OF HEREDITARY DISEASES

Unit 1

HEREDITARY DISEASES

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

ailment	недомогание, нездоровье
counter mutation	контрмутация
gamete	гамета, зрелая половая клетка
hemophilia	гемофилия
Mendelian laws (distribution)	законы (распределение) Менделя
monogenous hereditary diseases	моногенные наследственные заболевания
polygenous hereditary diseases	полигенные наследственные заболевания

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

Hereditary diseases are ailments caused by inherent defects in the genetic apparatus of the cell that are transmitted hereditarily through gametes. Monogenous hereditary diseases are controlled by one gene and are subject to Mendelian laws (distribution), i. e. hemo-

philia A. *Polygenous hereditary diseases* are controlled by a summary effect of several differently located genes and are inherited according to the additive-polygenous principle, most likely with a threshold effect of the limiting factor. Although mutations are the basis of hereditary pathology, they are not identical to the hereditary diseases. Like all other ailments, hereditary diseases are caused by certain factors effective in particular conditions against the background of the reactivity of the organism. The effect of gene mutations may be compensated or altered either by the action of other genes, or by the environment, or by defensive mechanisms: counter mutation at the cellular, or other factors at the organism level, etc. There are no diseases determined exclusively by mutation. On the other hand, there are no diseases whose cause, development and effects are completely unaffected by heredity. Heredity is the basis for the reactivity of the organism. The effect of age and sex on the reactivity can be seen as the result of unfolding of the genetic programme.

3. Answer the questions.

1. *How can hereditary diseases be characterized in the most general terms?*
2. *Are monogenous hereditary diseases controlled by individual genes?*
3. *How many genes control polygenous hereditary diseases?*
4. *What distribution are monogenous hereditary diseases subject to?*
5. *What are polygenous hereditary diseases controlled by?*
6. *What constitutes the basis of hereditary pathology?*
7. *What factors cause hereditary diseases?*
8. *By what may the effect of gene mutations be compensated?*
9. *Are there any diseases determined exclusively by mutation?*
10. *Are there any diseases completely unaffected by heredity?*
11. *How are the notions of heredity and reactivity correlated?*

4. Work in pairs. Ask and answer the questions above but not in the order they are listed in exercise 3.

5. Complete these words and word combinations below. (All the words may be found in the text above.)

1. r _ _ _ _ _ _ _ _ _ _ y
2. e _ _ _ _ _ _ _ _ _ _ t
3. c _ _ _ _ _ _ _ r
4. h _ _ _ _ _ _ _ _ _ a
5. M _ _ _ _ _ _ _ n d _ _ _ _ _ _ _ _ _ _ n
6. g _ _ _ _ _ s

6. Complete the dialogue using words and expressions from the completed list above.

Examiner: So how are hereditary diseases transmitted?

Student: They are transmitted hereditarily through _____.

Examiner: Right. What is the difference between monogenous hereditary diseases and polygenous hereditary diseases in terms of control by genes?

Student: Well, monogenous hereditary diseases are controlled by one gene and are subject to _____, for example, _____. Meantime polygenous hereditary diseases are controlled by a summary effect of several different genes.

Examiner: And do you think gene mutations are **not** identical to hereditary pathology?

Student: I'm definitely sure they are **not.** The point is that the effect of gene mutations can be compensated or altered either by the action of other genes, or by the _____, or by counter mutation defensive mechanisms at the _____ or organism level and so on.

Examiner: One more thing. Could you complete the following statement: Heredity is the basis for...

Student: ...the _____ of the organism.

7. Complete each word combination by choosing the best word from the box below.

genetic genetic reactivity Mendelian additive-polygenous inherent threshold hereditary

_____ programme
_____ apparatus
_____ of the organism
_____ distribution
_____ principle
_____ defects
_____ effect
_____ diseases

8. Listen to the following expressions. Listen again and repeat after the speaker. Think of their Russian equivalents.

1. To put it briefly...
2. It depends...
3. On one hand... but on the other hand...
4. It is common knowledge that...
5. Obviously...
6. In the long run...

9. Use the expressions given in the box below to complete the responses to some questions in exercise 3.

To put it briefly... It depends... On one hand... but on the other hand... It is common knowledge that... Obviously... In the long term...

1. _____ hereditary diseases are ailments caused by inherent defects in the genetic apparatus of the cell that are transmitted hereditarily through gametes.
2. _____ Monogenous hereditary diseases are controlled by one gene and are subject to Mendelian laws (i. e. hemophilia A). Polygenous hereditary diseases are controlled by a summary effect of several different genes and are inherited according to the additive-polygenous principle, most likely with a threshold effect of the limiting factor.
3. _____ mutations are the basis of hereditary pathology, _____ they are not identical to the hereditary pathology.
4. _____ like all other ailments, hereditary diseases are caused by certain factors effective in particular conditions against the background of the reactivity of the organism.
5. _____ The effect of gene mutations may be compensated or altered either by the action of other genes, or by the environment, or by counter mutation defensive mechanisms at the cellular or organism level and so on.
6. _____ there are no diseases determined exclusively by mutation. There are no diseases whose cause, development and effects are completely unaffected by heredity. Heredity is the basis for the reactivity of the organism. The effect of age and sex on the reactivity can be seen as the result of unfolding of the genetic programme.

10. Read the task card below.

Describe the main peculiarities of hereditary diseases following the plan: **What types of hereditary diseases are distinguished?** **By what factors are hereditary diseases caused?** **What role does heredity play in reactivity?**

11. Now, using the prompts from exercise 10 talk on the topic: HEREDITARY DESEASES.

12. Translate into English.

1. Наследственные заболевания вызываются нарушениями генетического аппарата клетки, которые передаются по наследству через гаметы.
2. Моногенные наследственные заболевания представляют результат мутации одного гена и подчиняются законам Менделя.
3. Полигенные наследственные заболевания определяются сложением действия нескольких различных генов и передаются в соответствии с аддитивно-полигенным типом наследования с пороговым эффектом, который оказывает некий лимитирующий фактор внешней среды.
4. В основе патогенеза наследственных заболеваний лежат мутации, однако понятия «мутация» и «наследственное заболевание» не являются тождественными.
5. Для возникновения наследственных болезней необходимо взаимодействие причинных факторов и реактивности организма в определенных условиях.
6. Последствия поражения генов могут компенсироваться или изменяться либо посредством воздействия других генов, либо посредством воздействия окружающей среды, либо защитными механизмами: контрмутациями на уровне клеток или факторами, действующими на уровне всего организма.
7. Не существует болезней, определяемых исключительно одной наследственностью.
8. Не существует болезней, определяемых исключительно одной мутацией. С другой стороны, не существует болезней, причины возникновения, развитие и последствия которых совершенно не зависят от наследственности.
9. Реактивность организма зависит, прежде всего, от наследственности.
10. Возрастная и половая зависимость реактивности определяется развертыванием генетической программы.

Unit 2

SEX CHROMATIN

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

Barr body	тельце Барра
buccal	1) буккальный, относящийся к щеке; щечный; трансбуккальный (о методе введения лекарственного средства); 2) внутриротовой

chromosome	хромосома
cytosine-guanine	цитозин-гуаниновый
diploid	диплоидный, с двойным набором хромосом
filament	филамент, нить
epithelium (*pl.*: epithelia)	эпителий, эпителиальная ткань
karyotype	кариотип (совокупность особенностей числа и формы хромосом клетки)
leukocyte	лейкоцит, белое кровяное тельце
methylation	метилирование
mitotic	митотический
neutrophilic	нейтрофильный, характеризующийся наличием нейтрофилов
nucleus (*pl.*: nuclei)	ядро, мн.: ядра
sex chromatine	половой хроматин
somatic	соматический
trisomy	трисомия (наличие в клетке лишней хромосомы)

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

"SEX CHROMATIN" ("Barr body") is the substance of genetically inactivated X-chromosome detected in normal conditions only in somatic (diploid) cells in females. In granular neutrophilic leukocytes "sex-chromatin" appears as a "drumstick". The number of X-chromosomes is equal to the number of bodies of sex-chromatin plus one. In the cells of different tissues the ocurrence of "sex chromatin" is different. Thus, 80–90% of neuron nuclei contain "sex chromatin", while only 20–40% of cell nuclei of the buccal epithelium contain it. "Sex chromatin" is localized in the nucleus, mostly attached to the nuclear membrane by thin filaments. In the case when "sex chromatin" is attached to the nuclear membrane, it has a triangle shape with the apex directed towards the nucleus centre. All organisms with two X-chromosomes in their karyotypes contain only one body of "sex- chromatin". In the case of trisomy of X-chromosome the nucleus contains two bodies of "sex chromatin".

There is a reverse correlation between the mitotic activity of the cell population and the frequency of sex-chromatin occurrence.

On the fifth and sixth days of embryonic development in a female organism inactivation of one of the two "X-chromosomes" takes place. The

mechanism of this phenomenon is conditioned by methylation of the DNA in the inactivated chromosome. There is a special site Xq27.3 (CpG islet) containing multiple cytosine-guanine repeats which can be methylated. They are methylated in the Barr body but are not methylated in the second (active) female X-chromosome, nor are they methylated in male X-chromosome (see fig. 4).

Fig. 4. Barr body

3. Answer the questions.

1. *What is sex chromatin?*
2. *What is the number of X-chromosomes?*
3. *Does the occurrence of sex chromatin in the cells depend on the type of the tissue?*
4. *Where is sex chromatin localized?*
5. *What shape does sex chromatin have in the case when it is attached to the nuclear membrane?*
6. *How many bodies of sex- chromatin are contained in the cell nuclei of chromosomes in normal female karyotypes?*
7. *How many bodies of sex- chromatin does a cell nucleus contain in the case of X-trisomy?*
8. *What sort of correlation is there between the mitotic activity of the cell population and the frequency of sex–chromatin occurrence?*
9. *What happens on the fifth and sixth days of embryonic development in a female organism?*
10. *By what is the mechanism of this phenomenon conditioned?*
11. *Describe the figure "Barr body" above?*

4. Work in pairs. Ask and answer the questions above but not in the order they are listed in exercise 3.

5. Complete this list of words and word combinations. (All these words may be found in the text above.)

1. n _ _ _ _ n
2. m _ _ _ _ _ _ _ _ _ _ n
3. b _ _ _ _ l epithelium

9. Read the task card below.

Describe the main peculiarities of sex-chromatin following the plan:

What is sex-chromatin?
Where is it localized?
How many bodies of sex-chromatin do organisms contain?
What is its function?

10. Now, using the prompts from exercise 9 talk on the topic: SEX CHROMATIN.

11. Translate into English.

1. Половой хроматин или тельце Барра — это материал генетически инактивированной Х-хромосомы, в норме присутствующей только в диплоидных соматических клетках женщин.
2. В гранулоцитах половой хроматин выглядит как небольшой дополнительный сегмент ядра — барабанная палочка.
3. Число Х-хромосом равно числу телец Барра плюс единица.
4. Тельце Барра наблюдается не одинаково часто в клетках различных тканей.
5. Лишь 20–40% клеток щечного эпителия содержат половой хроматин.
6. При синдроме «трипло-Х» клетки имеют два тельца Барра.
7. На 5–6-й день эмбрионального развития в женском организме осуществляется инактивация одной из двух Х-хромосом.
8. Механизм инактивации этого процесса связан с метилированием ДНК инактивируемой хромосомы.
9. Специальный участок Xq27.3, так называемый CpG-островок, содержит множественные цитозин-гуаниновые повторы, которые могут метилироваться.
10. Цитозин-гуаниновые повторы метилированы в тельце Барра и не метилированы в активной женской Х-хромосоме и в мужской Х-хромосоме.

Unit 3

SRY-GENE

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

assay	1) анализ; пробирный анализ; биологическое испытание, тест, проба; количественный анализ; производить анализ; испытывать; 2) проба; образец для анализа
dihydro-testosterone	дигидротестостерон
embryo	зародыш, эмбрион (на протяжении первых восьми недель внутриутробного развития)
embryogenesis	эмбриональное развитие, зародышевое развитие, эмбриогенез
estrogene	эстроген (1. женский половой гормон; 2. эстрогенное средство)
inhibiting	ингибирующий
luteinising hormone, LH	лютеинизирующий гормон, лютропин, пролан Б (*устар.*), ЛГ
polymerase	полимераза
SRY-gene	ген SRY
testosterone	тестостерон
trigger	триггер, спусковой крючок, запускающий элемент
tumor	опухоль
Shereshevsky–Turner's syndrome	синдром Шерешевского–Тернера

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo.

After that read the text aloud, trying to imitate the intonation.

In humans the development of masculine sex is predetermined by the presence of Y-chromosome. Provided there is a Y-chromosome present, the number of X-chromosomes is irrelevant. There is a special SRY-gene located in a Y-chromosome that begins functioning on the 6^{th}–7^{th} week

of embryogenesis. SRY-gene is a trigger activating a number of genes located in other chromosomes providing for the programme of testosterone synthesis in the embryo. It is the fetal testosterone that controls the masculine-type development of the embryo determining (together with anti-Müllerian inhibiting peptide) the development of internal, and, indirectly (through 5-α dihydro-testosterone), external masculine sex organs. SRY-gene also determines a high estrogene content (with part of testosterone converted into estrogens), thus suppressing the feedback between the luteinising hormone and estrogens in the developing brain. Such is the mechanism of the development of a non-cyclic hypothalamo-hypophysial regulation type of sex function and of the masculine type of brain. In the absence of Y-chromosome (and SRY-gene), when the karyotype is 46, XX or 45, X0 (Shereshevsky–Turner's[1] syndrome), the aforementioned events do not take place and the somatic and psychic type developed is feminine.

The information on sex chromatin is used for the verification of chromosomal sex and sex chromosomes aberrations and for the approximation of the proliferative activity of tumors. There are several modern methods used for the verification of genuine sex, one of them is immune-enzyme assay of "masculine" antigen encoded by SRY-gene. Polymerase chain reaction (PCR) may also be used for verification of genuine gender because it can detect any gene. PCR may also be used for revealing genetic defects.

3. Answer the questions.

1. *The presence of what chromosome is responsible for the development of masculine sex?*
2. *Does it matter how many X-chromosomes are present, provided there is a Y-chromosome?*
3. *How many Y-chromosomes are required for the development of an organism according to the masculine type?*
4. *On which week of embryogenesis does a SRY-gene located in a Y-chromosome begin to function?*
5. *What genes does a SRY-gene activate?*
6. *What does the fetal testosterone control?*
7. *What is high estrogene content determined by?*
8. *Under what conditions is the feminine somatic and psychic type developed?*
9. *What are the data on sex chromatin used for?*
10. *What are modern methods such as immune-enzyme assay and polymerase chain reaction (PCR) used for?*

[1] Turner Henry Hubert, American endocrinologist, born August 28, 1892, Harrisburg, Illinois; died 1970. One of the founders of modern Endocrinology

4. Work in pairs. Ask and answer the questions above but not in the order they are listed in exercise 3.

5. Complete this list of words and word combinations. (All the words may be found in the text above.)

1. b _ _ _ n
2. d _ _ _ _ _ _ _ _ _ _ t
3. k _ _ _ _ _ _ _e
4. s _ _ _ _ _ c
5. t _ _ _ _ _ _ _ _ _ _ _ _ e
6. t _ _ _ _ _ s
7. e _ _ _ _
8. m _ _ _ _ _ _ _ _ e sex
9. t _ _ _ _ _ r
10. c _ _ _ _ _ _ _ _ _ _s
11. v _ _ _ _ _ _ _ _ _ _ _ n
12. e _ _ _ _ _ _ _ e content

6. Complete this extract using words and expressions from the list above.

Examiner: What is the development of _____ sex predetermined by in humans?

Student: It is predetermined by the presence of Y-chromosome.

Examiner: Is it important how many X-chromosomes are present for the development of the organism according to the masculine type?

Student: No matter how many X-chromosomes are present, provided there is a Y-chromosome. One Y-chromosome is enough for the _____ of the organism according to the masculine type

Examiner: What important function does the SRY-gene implement?

Student: SRY-gene is a _____ which activates a number of genes located in other _____ providing for the program of _____ synthesis in the _____.

Examiner: Is it the only function?

Student: SRY-gene also determines a high _____ content (part of testosterone is converted into estrogens), thus suppressing the feedback between the luteinising hormone and estrogens in the developing _____.

Examiner: And what happens in the absence of Y-chromosome (and SRY-gene), when the _____ is 46, XX or 45, X0 (Shereshevsky — Turner's syndrome)?

Student: In this case the _____ and psychic type developed is feminine.

Examiner: How are the data of sex chromatin used?

The data of sex chromatin are used for the _____ of chromosomal sex and sex chromosomes aberrations and for the approximation of the proliferative activity of _____.

7. Complete each word combination by choosing the suitable word from the box below.

estrogene sex Luteinising chromosomal sex chromosomes fetal polymerase masculine Shereshevsky — Turner's testosterone sex genetic

type
synthesis
testosterone
organs
content
hormone
function
syndrome
sex
aberrations
chain reaction
defects

8. Complete gaps 1–9 with the appropriate word from the box.

that because that thus also indirectly providing for such provided

Polymerase chain reaction (PCR) may also be used for verification of genuine sex _____ it can detect any gene.

SRY-gene _____ determines a high estrogene content (part of testosterone is converted into estrogens), _____ suppressing the feedback between the luteinising hormone and estrogens in the developing brain. _____ is the mechanism of the development of a non-cyclic hypothalamo — hypophysial regulation type of sex function and of the masculine type of brain.

There is a special SRY-gene located in a Y-chromosome _____ begins functioning on the 6^{th}–7^{th} week of embryogenesis.

No matter how many X-chromosomes are present, _____ there is an Y-chromosome.

SRY-gene is a trigger activating a number of genes located in other chromosomes _____ the program of testosterone synthesis in the embryo. It is the fetal testosterone _____ controls the masculine-type development of the embryo determining (together with anti-Müllerian inhibiting peptide) the development of internal, and,

_____, (through 5-α dihydro-testosterone), external masculine sex organs.

9. Read the task card below.

Describe the main peculiarities of SRY–gene following the plan:
What is SRY-gene?
Why does it affect brain development?
Where and how are the data on SRY–gene used?

10. Now, using the prompts from exercise 9 talk on the topic: SRY-GENE.

11. Translate into English.

1. У человека развитие мужского пола предопределено наличием Y-хромосомы.
2. В присутствии любого количества X-хромосом одной Y-хромосомы достаточно для формирования организма по мужскому типу.
3. Ген SRY, находящийся в Y-хромосоме, срабатывает на 6–7-й неделе эмбрионального развития и запускает в действие ряд генов, локализованных в других хромосомах, и обеспечивающих программу синтеза тестостерона у плода.
4. Фетальный тестостерон направляет развитие зародыша по мужскому варианту.
5. Продукция тестостерона и антимюллерова ингибирующего пептида предопределяет развитие внутренних половых органов, а метаболит андрогенов 5-α-дигидротестостерон — наружных гениталий — по мужскому типу.
6. Часть тестостерона метаболизируется в эстрогены.
7. Ген SRY обеспечивает высокое содержание эстрогенных метаболитов, подавляющее формирование обратной связи между продукцией лютеинизирующего гормона и эстрогенов в развивающемся мозге.
8. Наличие Y-хромосомы и высокая продукция тестостерона ведут к установлению мужского нециклического типа гипоталамо-гипофизарной регуляции половых функций и мужского типа дифференцировки головного мозга.
9. В отсутствие гена SRY, даже если кариотип не 46XX, а 45X0 (синдром Шерешевского — Тернера), формируется соматический и психический женский пол.
10. Результаты исследований полового хроматина используются при определении истинного пола, аберраций половых хромосом, при оценке пролиферативной активности опухолей.

Medicine Through Biographies

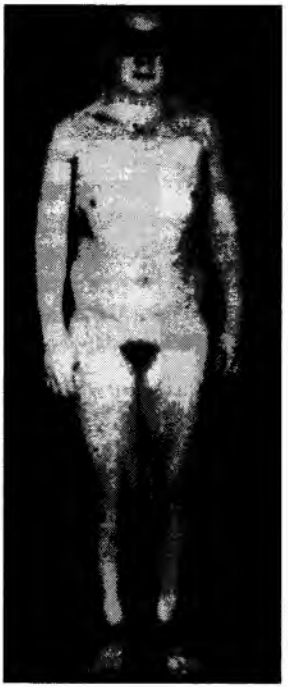
Barr, Murray Llewellyn (1908–1995) was a Canadian physician and medical researcher who discovered together with his graduate student Ewart George Bertram, in 1948, an important cell structure, the "Barr body". Born in Belmont, Ontario, he was educated at the University of Western Ontario, where he received his B.A. in 1930, M.D. in 1933, and M.Sc. in 1938. He was nominated for the Nobel Prize in Physiology or Medicine. In 1968, he was made an Officer of the Order of Canada. In 1959, he received the Royal Society of Canada's Flavelle Medal. In 1962, he won a Joseph P. Kennedy Jr. Foundation Award for his contributions to the understanding of the causes of mental retardation. In 1963, he received the Gairdner Foundation International Award and in 1972 he was elected Fellow of the Royal Society[1] of London. In 1998, he was posthumously inducted into Canadian Medical Hall of Fame.

Reference:

Barr ML, Bertram EG "A Morphological Distinction between Neurons of the Male and Female, and the Behavior of the Nucleolar Satellite during accelerated nucleoprotein synthesis". [Nature, N 163, P. 676 (April 30, 1949)]

Shereshevsky, Nicolay Adolfovich (1885–1961) was a Soviet endocrinologist and internist, who described in 1925 an infertile woman with primary hypogonadism, low stature, alar-shaped skin folds between neck and deltoid area and other signs of disease later named after him and Dr. Turner. The description of seven other cases was completed in 1938. Shereshevsky was educated at the Medical School of Moscow University, where he received his M.D. in 1911, worked as an internist at a sanatorium and served as military doctor in Red Army. In 1921 he became associate professor at the Second Moscow University, where he delivered one of the first courses on Clinical Endocrinology, later he headed the first Research Institute of Endocrinology and Hormone Chemistry (1934–1953).

[1] British equivalent of the title of Academician.



In 1953, during a campaign of mass repressions against Kremlin physicians, he was arrested and accused of being a "foreign spy". He was released from prison after Stalin's death. Nikolai Adolphovich Shereshevsky published over 100 articles.

Reference: *Shereshevsky N.A.* Clinical Endocrinology. Moscow: Medgiz, 1957.

Fig. 7. Phenotype in Shereshevsky-Turner syndrome (after: Kolarov P., Dokumov S., 1968)

12. Read the text above, discuss the biographies of Barr and Shereshevsky with your partner. Answer the following questions:

1. *What was common in the lives and careers of the Soviet and the Canadian scientists?*
2. *What was different?*
3. *Describe the main manifestations in Shereshevsky-Turner's syndrome and the patient's habitus, using text and fig. 7 above.*

Module 3

PATHOPHYSIOLOGY OF MICROCIRCULATION

Unit 1

MICROCIRCULATION AND ITS CHANGES

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

active (arterial) hyperemia	активное (артериальное) полнокровие
arteriole	артериола
blood flow	кровоток
blood vessel	кровеносный сосуд
capillary bed	капиллярное русло
coin stacks	«монетные столбики» (неустойчивые агрегаты эритроцитов)
elasticity	эластичность
embolus (*pl.*: emboli)	эмбол
endogenous/vascular ischemia	эндогенная/сосудистая ишемия
hematogenous/obturation ischemia	гематогенная/обтурационная ишемия
inflow	приток
ligature	1) лигатура; 2) перевязка, лигирование (напр. сосуда), наложение лигатуры; перевязывать, лигировать (напр. сосуд), накладывать лигатуру
metarteriole	прекапиллярная артериола, метартериола, прекапилляр
microcirculation	микроциркуляция
microcirculatory bed	микроциркуляторное русло
mixed/combined hyperemia	смешанная гиперемия

outflow	отток
passive (venous) hyperemia, congestion	пассивное (венозное) полнокровие, венозный застой
plasma capillary	плазматический капилляр
precapillary	предкапилляр; предкапиллярный
rheologic	реологический, связанный с текучестью
rouleaux	(*pl* от rouleau, франц.) «монетные столбики» (неустойчивые агрегаты эритроцитов)
sludge of erythrocytes	сладж эритроцитов
sphincter	сфинктер, жом (круговая мышца, сжимающая полый орган)
stasis	стаз (остановка кровотока или тока других жидкостей организма)
terminal	конечная часть, окончание; конец; конечный, терминальный
thoroughfare, preferencial channel	главный капилляр, магистральный канал
thrombus (pl. trombi)	тромб (*мн.*: тромбы)
tissue-type/compression ischemia	тканевая/компрессионная ишемия
true	истинный
venule	венула
viscosity	вязкость

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

MICROCIRCULATION is the movement of blood in small blood vessels not more than 100 mcm in diameter: arterioles, terminal arterioles with precapillary sphincters, metarterioles (with the thoroughfare, i. e. preferencial channel), true capillaries, plasma capillaries, postcapillary venules (8–30 mcm), collective venules (30–50 mcm), and muscle venules (50–100 mcm). A metarteriole branches from the middle part of the arteriole, passing through the capillary bed to the venule. It is wider than a true capillary and is surrounded in the first half (the proximal part) of its way through the capillary bed with smooth muscle cells. A proximal part of the metarteriole gives rise to numerous true capillar-

ies. The precapillary sphincters of the arteriole and metarteriole control the entry of blood into the capillaries. The distal segment of the thoroughfare receives capillaries from the microcirculatory bed, but no sphincters are present where the afferent capillaries enter the thoroughfare. The thoroughfare, or principal channel, is characterized by low resistance to blood flow.

The changes occurring in microcirculation are referred to as **hyperemia** and **ischemia**. Hyperemia is a phenomenon of increased blood filling in an organ or in a portion of tissue. There are several variations of hyperemia: arterial, venous and mixed type.

Arterial hyperemia (Latin: fluxio, active hyperemia) is a dynamic increase of blood filling in a portion of tissue due to enhanced inflow of blood through arteries. Outflow is also increased here in accordance with the enhanced inflow, as well as the linear and volume velocities of blood flow. In this case the number of blood capillaries, partial pressure of oxygen in blood, and oxygen supply of the tissue are increased, too.

Venous hyperemia (Latin: *congestio,* passive hyperemia. congestion) develops due to obstructed venous drainage of blood, whereas the inflow remains normal. Practically, the etiology of venous hyperemia is limited to mechanical obstruction to drainage inside the veins (thrombi or emboli), or to external pressure (ligature, tumor, etc). In the case of venous hyperemia the blood flow is slowed down. Postcapillary venules and capillaries appear distinctly enlarged above the area obstructing the outflow. The linear and volume velocities of the blood flow are decreased. Partial pressure of oxygen in venous blood is low. Oxygen supply of the tissue is poor.

Mixed (combined) hyperemia results from a combination of venous and arterial types, in which case obstructed venous drainage of blood takes place simultaneously with increased inflow. It may occur as transient state — from active hyperemia to congestion.

Ischemia is charecterised by the decrease of blood flow within an organ or tissue on account of decreased or inadequate inflow through the arteries. Ischemia may be hematogenous (obturation), endogenous (vascular) and tissue-type (compression). Progressive slowing down of blood flow in the case of venous hyperemia or ischemia may result in complete cessation of blood flow and, thus, cause congestive, or ischemic **stasis**. Stasis (genuine, or capillary) may also develop due to changes in the rheologic properties of blood within microvessels (viscosity and elasticity).

Stasis is preceded by *pre-static phenomena*: jerk-like or pendulum-like blood motions, sludge of erythrocytes, forming of coin stacks (rouleaux), etc.

The biophysics of blood microcirculation during pre-static phenomena and stasis was thoroughly investigated by Alexander Leonidovich Chizhevsky (see his biography: p. 67 below).

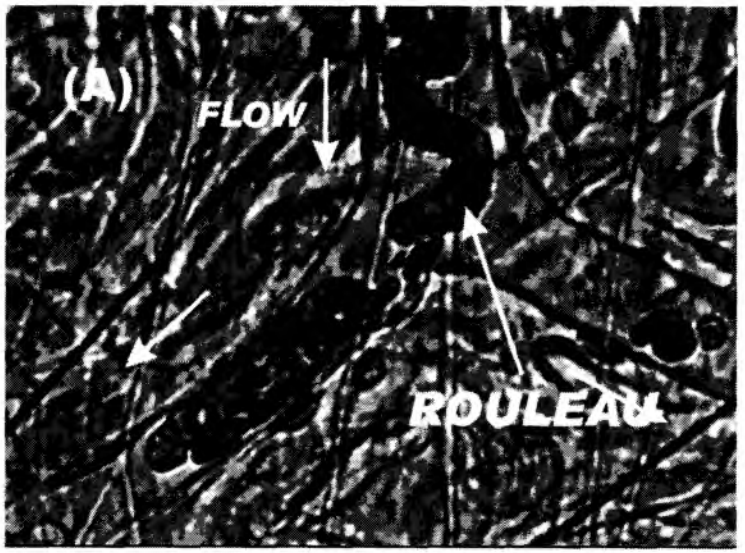


Fig. 8. RBC rouleau in a capillary (Lipowsky H. H., available from: http://www.bioe.psu.edu/labs/microcirc.html)

3. Answer the questions.

1. *What type of blood movement is referred to as microcirculation?*
2. *What types of blood vessels provide for microcirculation?*
3. *Which part of the metarteriole gives rise to numerous true capillaries?*
4. *Which sphincters control the entry of blood into the capillaries?*
5. *Whichs segments of the thoroughfare (principal channel) receive capillaries from the microcirculatory bed?*
6. *What resistance to blood flow is characteristic of the thoroughfare?*
7. *What two main types of abnormality occur in microcirculation?*
8. *What is hyperemia?*
9. *What variations of hyperemia may be distinguished?*
10. *What peculiarities are characteristic of arterial or active hyperemia?*
11. *What causes venous or passive hyperemia (congestion)?*
12. *What combination gives rise to mixed or combined hyperemia?*
13. *What is ischemia characterized by?*
14. *What variations of ischemia exist?*
15. *What processes may cause congestive or ischemic stasis?*
17. *By what kind of pre-static phenomena is stasis preceded?*
18. *What is rouleaux phenomenon? (use the text and the fig. 8 above)*

4. Work in pairs. Ask and answer the questions above but not in the order they are listed in exercise 3.

5. Find the matching words from the box below to complete the gaps.

precapillary obstructed ischemia true postcapillary branches motions stasis passive flow increased terminal channel phenomena bed external

Blood vessels

1. _____ arterioles
2. _____ sphincters
3. _____ capillaries
4. _____ venules

Microcirculation

5. metarteriole _____
6. microcirculatory _____
7. principal _____
8. blood _____

Hyperemia

9. _____ hyperemia
10. _____ drainage
11. _____ pressure
12. _____ inflow

Ischemia

13. vascular _____
14. ischemic _____
15. pre-static _____
16. jerk-like _____

6. Choose the right word from among those in italics.

1. Microcirculation is the movement of blood in small blood *blisters/banks/ vessels* not more than 100 mcm in diameter.
2. Metarteriole *branches/cells/fragments* from the middle part of the arteriole pass through the capillary bed to the venule.
3. A proximal *line/part/bronchus* of the metarteriole gives rise to numerous true capillaries/dwarves/believers.
4. The distal *cusp/radius/segment* of the thoroughfare receives capillaries from the microcirculatory bed.

5. The principal *challenge/channel/host* is characterized by low *fever/resistance/jaw* to blood flow.
6. *Arterial/venous/mixed* hyperemia is a dynamic increase of blood filling in a portion of tissue due to enhanced *movement/inflow/outflow* of blood through arteries.
7. Outflow is also increased here in accordance with the enhanced inflow, as well as the linear and volume *characteristics/velocities/data* of blood flow.
8. The etiology of venous hyperemia is limited to mechanical *obstacle/hindrance/obstruction* to drainage inside the veins or to external storage/pressure/use.
9. In the case of venous hyperemia the blood *supply/sugar/flow* is slowed down.
10. Mixed (combined) hyperemia results from a combination of venous and arterial *types/grooves/pressure.*

7. Read the conversation between an examiner and a student. Complete the student's responses using phrases a–f.

a) They seem to be
b) As a matter of fact
c) ideally
d) I'll almost certainly work in a hospital as a hematologist.
e) It's probably
f) They're almost certainly

Examiner: Now let's talk about the phenomenon of microcirculation and microcirculation changes. How do you think the term "microcirculation" can be defined?

Student: 1 _____, microcirculation is the movement of blood in small blood vessels not more than 100 mcm in diameter such as arterioles, terminal arterioles with precapillary sphincters, metarterioles, true capillaries, plasma capillaries, postcapillary, collective and muscle venules.

Examiner: What is the diameter of muscle venules?

Student: 2 _____ 50 to 100 microcentimetres wide.

Examiner: Precisely. And which part of the metarteriole gives rise to numerous true capillaries?

Student: I'm not really sure. 3 _____ a proximal part of the metarteriole.

Examiner: Now, could you tell me which blood vessels control the entry of blood into the capillaries?

Student: Let me see. 4 _____ the precapillary sphincters of the arteriole and metarteriole.

Unit 2

HYPEREMIA AND ISCEMIA MODELS IN THE FROG

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

axial	аксиальный, осевой
bundle	пучок
forceps	1) щипцы, зажим; 2) пинцет
frog	лягушка
interdigital	межпальцевой
intestinal	интестинальный, относящийся к кишечнику, кишечный
lateral	латеральный, боковой; удаленный от средней линии
lumen	просвет (сосуда)
marginal	маргинальный, краевой
medial	медиальный, относящийся к середине или центру
membrane	мембрана
mesentery	брыжейка
microcirculatory disorders	нарушения микроциркуляторного кровообращения
neurogenous	нейрогенный; неврогенный
neuroparalytic	нейропаралитический (механизм); нервно-паралитический (о ядах)
neurotonic	1) нейротонический (механизм); 2) улучшающий тонус нервной системы (о лекарственном средстве)
neurovascular	нейроваскулярный, невроваскулярный, нервно-сосудистый
plasmatic	плазменный, плазматический (кровоток), относящийся к плазме
sciatic nerve	седалищный нерв
session	время, отведенное какой-л. деятельности или занятию
spinal cord	спинной мозг
stratum (*pl.* strata)	слой
swab	тампон
tongue	язык
tongue root	корень языка
translucent	полупрозрачный
turpentine	скипидар
vessel	сосуд; полость трубчатого органа

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

Microcirculatory disorders may easily be modeled using certain translucent organs of frog (tongue, interdigital swimming membrane, intestinal mesentery).

Arterial hyperemia model in the frog tongue

After destruction of the spinal cord (with a long needle through the spinal canal) place the frog (belly down) upon the board with an elliptic hole at one side, so that the lower jaw is fastened with two pins near the hole. Then pull out the tongue and spread it over the hole fastening it with pins at its muscle angles. Be careful not to use force in order to avoid precipitous development of microcirculation disorders. Examine the preparation at low magnification (x10). Select a portion abundant in blood vessels (arterioles, venules and capillaries). Pay attention to the size of the vessel lumen, the blood velocity and the separation of the lumen into axial and marginal plasmatic strata. Count the number of functioning capillaries. To maintain normal circulation do not bend the tongue or overextend it. Remember to moisten it with saline solution.

For modeling arterial hyperemia (neurotonic type) place a cotton swab soaked in turpentine and plant oil on the tongue. After that examine the preparation again, making notes of the changes in the aforementioned parameters.

Venous hyperemia model in the frog tongue

Use the frog from the previous session after rinsing its tongue with saline solution. To model venous hyperemia, tie ligatures around the veins in the tongue root. There are two pairs of large parallel blood vessels in the root of the tongue: the lateral vessels are veins, whereas the medial ones are arteries. To tie a ligature around the vein, pull the tongue by its tip with forceps and insert a needle with the ligature between the vein and the artery, then bring it out. After this, the procedure is repeated on the second vein. Tie both ligatures to form a loop on each side but do not fasten the knots tightly. Study the normal circulation at low magnification (x10). Then, without moving the preparation,

pull one of the tied ligatures' ends and fasten the knot. Note the changes in the size of the vessel lumen, the blood velocity, the separation of the lumen into axial and marginal plasmatic strata and the number of functioning capillaries. Then tie firmly the second ligature and inspect the preparation microscopically. Note the microcirculatory changes taking into consideration the same parameters. Also pay attention to the outward appearance of the tongue (colour, size, etc).

Observe the microcirculation changes after the ligatures on both sides have been removed.

Neurogenous ischemia model in the frog interdigital swimming membrane

Cut the skin longitudinally with scissors on the posterior surface of the thigh along the groove containing the neurovascular bundle. Dissect the sciatic nerve and place a ligature beneath it but do not tie. Spread the swimming membrane over the round hole in the board fastening its ends with pins. Find an appropriate portion of the membrane rich in blood vessels and examine the circulation at low magnification (x10). Then tie the ligature tightly and cut the nerve above the ligature. Note the microcirculation changes correspondent to neuroparalytic arterial hyperemia. Then, tugging by the ligature and, in this way, exciting the peripheral end of the severed sciatic nerve, produce neurotonic ischemia against the background of neuroparalitic arterial hyperemia.

3. Answer the questions.

1. *The organs of which animals are used to model microcirculatory disorders and why?*
2. *Which organ of the frog is used to model arterial hyperemia?*
3. *What test is performed on a frog for modeling arterial hyperemia?*
4. *Which organ is used to model venous hyperemia?*
5. *What test is performed on a frog for modeling venous hyperemia?*
6. *What parameters must be taken into consideration while observing microcirculatory changes?*
7. *Which organ is used to model neurogenous ischemia?*
8. *What test is performed on a frog for modeling neurogenous ischemia?*
9. *Why are animals' organs used in experiments?*

4. Work in pairs. Ask and answer the questions above but not in the order they are listed in exercise 3.

5. Complete the collocations below by choosing the words from the box.

nerve spinal cotton membrane low disorders blood tongue saline lower vessel

1) _____ solution
2) _____ cord
3) _____ jaw
4) microcirculation _____
5) _____ lumen
6) _____ velocity
7) swimming _____
8) sciatic _____
9) frog _____
10) _____ swab
11) _____ magnification

6. Use the collocations from exercise 5 in the sentences below.

1. Having destroyed _____, place the frog (belly down) upon the board.
2. The frog's _____ must be fastened with two pins near the hole.
3. Then pull out the _____ and spread it over the hole fastening it with pins at its muscle angles.
4. You should not use force in order to avoid precipitous development of _____.
5. You ought to pay attention to the size of the _____, the separation of the lumen into axial and marginal plasmatic strata and _____.
6. Remember to moisten the frog tongue with _____.
7. For modeling arterial hyperemia place a _____ soaked in turpentine and plant oil on the tongue.
8. Study the normal circulation at _____ (x10).
9. Dissect the _____ and place a ligature beneath it but do not tie.
10. Spread the _____ over the hole in the board fastening its ends with pins.

7. Read the conversation between an examiner and a student. Complete the student's responses using logical links from the box.

after that and so that consequently while at the same time then such as also under such conditions (x2) firstly

Examiner: As we know, microcirculatory disorders may be easily modeled by using certain translucent organs of a frog. How can we use a frog's tongue for arterial hyperemia modeling?

Student: 1. _____ we ought to destruct the spinal cord with a long needle through the spinal canal. 2. _____ The frog is placed belly down on a board having an elliptic hole at one side. 3. _____ the lower jaw is fastened with two pins near the hole. 4. _____ we ought to pull out the tongue. 5. _____ we ought to spread the tongue over the hole fastening it with pins at its muscle angles. 6. _____ we should not not use force in order to avoid precipitous development of microcirculation disorders. 7. _____ we ought to examine the preparation at low magnification (x10). 8. _____ we ought to select a portion abundant in blood vessels. 9. _____ arterioles, venules and capillaries. 10. _____ we ought to pay attention to the size of the vessel lumen, blood velocity, separation of the lumen into axial and marginal plasmatic strata. 11. _____ we have to count the number of functioning capillaries.

8. Look at the list of topics below. Using the example, write two questions on topics 2 and 3.

Example. **Topic 1.** Arterial hyperemia model in a frog tongue
How can arterial hyperemia be modeled in a frog tongue?
What benefits can it bring to medical students?
Topic 2. Venous hyperemia model in the frog tongue
Topic 3. Neurogenous ischemia model in the frog interdigital swimming membrane

9. Look back at exercise 8. Answer the questions you wrote on these topics using the language and ideas from exercises 2–7.

10. Read the task card below.

Using the prompts speak on the following:
How can microcirculatory disorders be modeled?
How can arterial and venous hyperemia models in the frog tongue be produced?
How can neurogenous ischemia model in the frog interdigital swimming membrane be produced?

11. Now, using the prompts from exercise 9, talk on the topic: HYPEREMIA AND ISCHEMIA MODELS IN THE FROG.

12. Translate into English.

1. Нарушения микроциркуляторного кровообращения несложно смоделировать на лягушке, используя для этого ее определенные полупрозрачные органы (язык, плавательная перепонка, брыжейка тонкой кишки).
2. При моделировании артериальной гиперемии на языке лягушки необходимо предварительно разрушить спиной мозг через позвоночный канал с помощью длинной иглы. Затем лягушку кладут на живот на дощечку с овальным отверстием таким образом, чтобы нижняя челюсть располагалась рядом с отверстием. Двумя булавками нижняя челюсть крепится к дощечке.
3. Не используя силу, во избежание нарушения движения крови по сосудам языка, вытащите язык лягушки, расправьте его над отверстием, зафиксировав с помощью булавок.
4. Наблюдайте за происходящим при "малом" (десятикратном) увеличении микроскопа.
5. Выберите участок, богатый кровеносными сосудами (артериолами. венулами и капиллярами), обратите внимание на размер кровеносных сосудов, скорость кровотока, особенности разделения кровотока на осевой слой и периферический слои.
5. Определите количество функционирующих капилляров и, чтобы не нарушать нормального кровообращения, не сгибайте язык и не вытаскивайте его слишком далеко.
6. Чтобы смоделировать артериальную гиперемию нейротонического типа с помощью ватного тампона нанесите скипидар на язык лягушки, после чего наблюдайте изменения исследуемых вами параметров.
7. Венозная гиперемия моделируется на языке лягушки.
8. Для моделирования венозной гиперемии перевязывают вены языка, располагающиеся латерально в основании языка.
9. Сначала наблюдают нормальное кровообращение при десятикратном увеличении, а затем наложенные на вены лигатуры затягивают и наблюдают изменение размеров просвета кровеносных сосудов, скорости кровотока, особенности разделения кровотока на осевой и периферический слои, количество функционирующих капилляров.
10. Нейрогенная ишемия моделируется на межпальцевой плавательной перепонке лапки лягушки.

Medicine Through Biographies

Bernard, Claude, 1813–1878, a French physician and pathophysiologist, was born in the village of Saint-Julien near Villefranche-sur-Saône. He received his early education in the Jesuit school of that town, and then proceeded to the college at Lyon, which, however, he soon left to become assistant in a drugstore. His leisure hours were devoted to the composition of a vaudeville comedy, and the success it achieved moved him to attempt a prose drama in five acts, "Arthur de Bretagne". At the age of twenty-one, in 1834, he went to Paris, armed with this play and an introduction to Saint-Marc Girardin, Professor of Literature at Sorbonne, but the critic dissuaded him from adopting literature as a profession, and urged him rather to take up the study of medicine. This advice Bernard followed, entering the School of Medicine at the University of Paris, and became an interne at the Hotel Dieu (with 26th rank of academic standing among 29 students of his year!). At the hospital he was brought into contact with the great physiologist, FranHois Magendie, who worked as a physician there, and whose official "preparateur" at the Collége de France Bernard became in 1841. Bernard's first paper on the Chorda tympani was published in 1843, the year he received his M.D. degree. Three years later he was appointed Magendie's associate professor at the college, and in 1855 succeeded him as full professor.

Claude Bernard coined the concept of "milieu interieur" — constant liquid environment for body cells. "The constancy of the internal environment is the condition for a free and independent life", — he wrote. He was the founder of the concept of neurism in medicine, he discovered vasomotor nerves and neurogenic microcirculatory disorders (1851), glycogen production in the liver and the ability of this organ to liberate glucose in blood after neural stimulus (1848), he formulated the hepatogenic theory of diabetes mellitus pathogenesis (1877), introduced the practice of blind experiments and wrote the first textbook on experimental pathophysiology (1865). French Emperor Louis-Napoleon personally financed his lab. When Bernard died he was accorded a public funeral — an honor which had never before been bestowed by France on a man of science.

He wrote: "I consider the hospital to be the antechamber of medicine, it is the first place where the physician makes his observations. But the laboratory is the temple of the science of medicine".

Reference: Bernard C. Recherches expérimentales sur les fonctions du nerf spinal ou accessoire de Willis. Paris, 1851 (French).

Chizhevsky Alexander Leonidivich, 1897–1964, a Soviet biophysicist, pathophysiologist, poet, artist and philosopher. Chizhevsky was born in the small town of Zehanovec̆ to the family of a Russian artillery officer. His father was talented inventor. Alexander graduated from Kaluga technical college, where one of his teachers was the founder of cosmonautics K. E. Tsiolkovsky, who had greatly influenced him. Chizhevsky entered Moscow Higher School of Commerce and later moved to Moscow Archeological Institute, but World War I ruined his plans. He joined the Russian Army as a volunteer, fought for 2 years, was wounded and awarded The Cross of St. George for courage; in 1918–1922 he completed his education at the Faculty of Natural Sciences and Mathematics, and later — at the Faculty of Medicine, at Moscow University.

In 1924 Chizhevsky came to the conclusion that not only biological, but also social and historical phenomena on the Earth are controlled by cyclic electromagnetic and radiation activity of the Sun. He is the founder of Heliobiology and the term "cosmic weather" was suggested by him. He discovered and experimentally investigated the opposite influence of positively and negatively charged aeroions on cells and organisms and applied this phenomenon in physiotherapy and communal hygiene; he invented "Chizhevsky's chandelier" for artificial aeroionization. Chizhevsky was the first to carry out research of the electromagnetic properties of erythrocytes in circulating blood; he is a pioneer in biophysics of microcirculation: he suggested an explanation for the mechanisms of rouleau and other pre-static phenomena (1932), which is broadly used in the erythrocyte sedimentation rate (ESR) test. He also discovered metachromasia in bacterial cells (1935). A. L. Chizhevsky was elected Honorary President of the 1st World Congress in Biophysics and Cosmic Biology (1939) and nominated by scientists of different countries for the Nobel prize. In 1942 the scientist was falsely accused of anti-Soviet conspiracy, imprisoned for 8 years, and later exiled. In 1954 he was released, but continued his research in Kazakhstan at the city of Karaganda, far from the main academic centers. He devoted this period to studies in flow structure of moving blood. In 1958 Chizhevsky returned to Moscow where he published several books on aeroionification of buildings and on biophysics. He was also a gifted poet, a philosopher in the spirit of Russian Cosmism and an original painter. Historians of science called this polymath: "Soviet Leonardo".

Reference: A. L. Chizhevsky, Structural Analysis of the Mooving Blood. Moscow: USSR Acad. of Sci. Publ., 1959. 266 Pp. (Russian).

Module 4
THROMBOSIS AND EMBOLISM

Unit 1
THROMBOSIS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

accelerator	акселератор, ускоритель; катализатор
agglutination	агглютинация, склеивание
allergy	аллергия
Bernard-Soulier[1] syndrome	синдром Бернара-Сулье
clot	сгусток
coagulation	коагуляция, коагулирование; свертывание
collagen	коллаген
congenital	врожденный
endotheliocyte	эндотелиоцит, эндотелиальная клетка
enzyme	фермент, энзим
fibrin	фибрин
fibrinogen	фибриноген
glycoprotein	гликопротеид, гликопротеин
heart cavity	полость сердца
hemorrhage	1) кровотечение; 2) кровоизлияние
hemostasis	1) остановка кровотечения, гемостаз
inflammation	воспаление
phospholipid	фосфолипид
platelet	тромбоцит, кровяная пластинка, бляшка Бицоцеро[2]
prothrombin	фактор II (свертывающей системы крови), протромбин; протромбиновый
receptor	рецептор
retraction	1) втяжение; западение; 2) ретракция; стягивание, сокращение

[1] Jean Bernard, French physician, oncologist and haematologist, born May 26, 1907, Paris, died 2006. Jean-Pierre Soulier (1915–2003), French hematologist.

[2] Giulio Bizzozero (1846–1901), Italian pathologist and histologist.

subendothelial	субэндотелиальный
substrate	1) субстрат; 2) подложка (в культуре клеток)
supravital	суправитальный, прижизненный
thrombasthenia	тромбастения, тромбастеническая гемофилия
thrombin	тромбин, фибрин-фермент (*устар.*)
thrombosis	тромбоз
von Willebrand[1] disease	болезнь фон Виллебранда

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

Thrombosis is a typical pathological process of *supravital* forming a conglomerate (thrombus) consisting of plasma proteins and blood cells *primarily attached* to the injured inner surface of the blood vessel or heart cavities.

A thrombus should be distinguished from a blood clot: a clot may be generated *in vivo* and *in vitro*, whereas a thrombus may be generated only within a cardio-vascular system; from the very beginning of its formation a true thrombus is continuously fastened to the inner surface of the wall of a blood vessel or heart cavity, whereas a clot may be formed inside the vessel, unattached to the wall.

There are three conditions determining the formation of a thrombus ("The Virchow triad"): damage of the vessel wall, slowing down of the velocity of the blood stream, and changes in blood composition.

The stages of thrombosis are: agglutination, coagulation, retraction.

Thrombosis is one of the components of tissue response to injury. It is a *local* reversible process necessary for an adequate hemostasis, which prevents hemorrhage and blood loss and restores the wall of the blood vessel. Thrombosis in itself is a component of several other, more complex, pathologic processes such as inflammation, allergy, etc.

After a blood vessel injury, the von Willebrand factor (vWF) from the injured endotheliocytes functions as an adhesion bridge between subendothelial collagen and the Ib glycoprotein (GpIb) platelet receptor (primary reversible aggregation). The aggregation is accomplished through the fibrinogen binding to platelet Gp IIb-IIIa receptors and bringing many platelets together (secondary irreversible aggregation).

[1] Erik Adolf von Willebrand, Finnish internist, born February 1, 1870, Vaasa; Russia, died December 12, 1949, Perna, Finland.

Congenital deficiencies in various receptors, or in bridging molecules, called factors (f), lead either to Glanzmann[1] thrombasthenia (deficiency of Gp IIb–IIIa receptors), or to Bernard-Soulier syndrome (deficiency of Gp Ib receptor), or to von Willebrand disease (deficiency of vWF) . The initial reaction complex consists of an enzyme (f. IXa), a substrate (f. X) and a reaction accelerator (f. VIIIa) which are assembled on the phoshpolipid surface of platelets. Calcium ions (f. IV) hold the assembled components together and are essential for the reaction. The activated f. Xa then becomes the enzyme part of the second adjacent complex in the coagulation cascade, converting the prothrombin (f. II, substrate) to thrombin (f. IIa) in cooperation with the reaction accelerator (f. Va). Thrombin converts fibrinogen to fibrin within and around the platelet plug, essentially cementing the platelets in place.

3. Do the following statements agree with the information in the text above?

Write

TRUE	*if the statement agrees with the information*
FALSE	*if the statement contradicts the information*
NOT GIVEN	*if there is no information on the subject concerned*

1. Thrombosis is a process of forming a blood clot.
2. A clot can be generated only *in vivo*, not *in vitro*, and a thrombus can be generated only in a cardio-vascular system.
3. "The Virchow triad" contains three conditions determining the formation of a thrombus.
4. Inflammation and allergy are more complex pathologic processes than thrombosis.
5. Coagulation is one of the stages of thrombosis.
6. After a blood vessel injury the process of aggregation takes place.
7. The process of aggregation comprises three stages.
8. Such phenomena as Glanzman thrombasthenia, Bernard-Soulier syndrome and von Willebrand disease result from congenital deficiencies in various receptors.
9. It is sodium ions that are essential for the reaction.
10. It is thrombin that converts fibrinogen to fibrin within and around the platelet plug, essentially cementing the platelets in place.

4. Write questions based on the statements above.

[1] Eduard Glanzmann, Swiss paediatrician, born April 12, 1887, Luzern; died February 2, 1959.

5. Work in pairs. Ask and answer the questions from exercise 4.

6. Complete this mindmap below using the prompts given.

1) th _ _ m _ _ s (generated only in a cardio-vascular system)
2) c _ _ t (generated *in vivo* and *in vitro*)
3) ag _ _ ut _ _ at _ _ n
4) c _ _ g _ _ at _ _ n
5) r _ _ r _ _ t _ _ n (the stages of thrombosis)
6) I _ _ l _ _ mat _ _ n
7) a _ _ e _ _ y (complex pathologic processes)
8) a _ _ re _ _ t _ _ n
9) c _ _ g _ _ at _ _ n (processes which take place after a blood vessel injury)
10) f _ _ r _ _ og _ n
11) t _ _ o _ _ in
12) f _ _ r _ n (substances)

7. Now complete these sentences using words from the completed mindmap.

Examiner: Let's talk about thrombosis. What can be generated *in vivo* and *in vitro*, and what can be generated only in a cardio-vascular system?

Student: Well, a 1. _____ can be generated *in vivo* and *in vitro*, whereas a 2. _____ can be generated only in a cardio-vascular system.

Examiner: What are the stages of thrombosis?

Student: They are 3. _____, 4. _____ and 5. _____.

Examiner: A component of which complex pathologic process is thrombosis?

Student: It is a component of such processes as 6. _____ and 7. _____.

Examiner: *What happens after* a blood vessel injury?

Student: The Von Willebrand factor from the injuried endotheliocytes functions as an adhesion bridge between subendothelial collagen and the Ib glycoprotein platelet receptor (primary reversible 8. _____).

Examiner: As we know, calcium ions hold the assembled components together and are essential for the reaction. What happens in this situation?

Student: The activated f. Xa then becomes the enzyme part of the second adjacent complex in the 9. _____ cascade, converting the prothrombin (f. II, substrate) to 10. _____ (f. IIa) in cooperation with the reaction accelerator f.Va. Thrombin converts 11. _____ to 12. _____ within and around the platelet plug, essentially cementing the platelets in place.

8. Complete each sentence by choosing the best ending from the list a–g below.

1. A thrombus consists of plasma proteins and blood cells...
2. A clot may be generated *in vivo* and *in vitro*...
3. Thrombosis is one of the components...
4. Congenital deficiencies in various receptors, or in bridging molecules...
5. Calcium ions hold the assembled components together...
6. The activated f. Xa then becomes the enzyme part of the second adjacent complex in the coagulation cascade...
7. Thrombin converts fibrinogen to fibrin within and around the platelet plug...

a) essentially cementing the platelets in place.
b) and are essential for the reaction.
c) whereas a thrombus may be generated only in a cardio-vascular system.
d) converting the prothrombin (f II, substrate) to thrombin.
e) primarily attached to the injured inner surface of the blood vessel or heart cavities.
f) lead to Glanzmann thrombasthenia, to Bernard-Soulier syndrome, or to von Willebrand disease.
g) of tissue response to injury.

9. Read the task card below.

Describe the main peculiarities of thrombosis following the plan:
What is thrombosis?
How can a thrombus be distinguished from a clot?
What are the stages of thrombosis?
What is the function of thrombin?

10. Now, using the prompts from exercise 9, talk on the topic: Thrombosis.

11. Translate into English.

1. Тромбоз — типовой патологический процесс прижизненного формирования в сосудах или полостях сердца тромба — первично прикрепленного к сосудистой стенке конгломерата стабилизированного фибрина и форменных элементов крови.
2. Тромб следует отличать от кровяного сгустка: сгусток может формироваться и *in vivo*, и *in vitro*, но тромб — только *in vivo* в сосудах и полостях сердца.
3. Важнейшими тромбогенными факторами (триада Вирхова) являются: повреждение сосудистой стенки, замедление скорости кровотока и нарушение состава крови.

4. Стадиями тромбоза являются агглютинация, коагуляция и ретракция.
5. Тромбоз — один из компонентов ответа ткани на повреждение.
6. Совокупность механизмов, обеспечивающих остановку кровотечения, называется системой гемостаза, задача которого — остановка кровотечений и восстановление целостности сосудистой стенки.
7. Тромбоз является компонентом ряда других, более комплексных патологических процессов, таких как воспаление и аллергия.
8. Недостаточная экспрессия vWF ведет к болезни фон Виллебранда.
9. Дефект гликопротеина gp IIb–IIIa ведет к тромбоастении Гланцманна.
10. Для осуществления процесса тромбообразования необходим ионизированный кальций.

Unit 2
PLATELET GRANULES

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

adenine	аденин
agglutination	агглютинация, склеивание; 2) образование групп или скоплений (микроорганизмов)
anticoagulation	антикоагуляция, противосвертывание
angiogenesis	ангиогенез
antithrombin	антитромбин (название группы веществ — антагонистов тромбина)
biogenous amine	биогенный амин
calcium.	кальций
coagulation factor	фактор свертывания крови
epinephrine	адреналин, эпинефрин
fibronectin	фибронектин
fibrinogenesis	фибриногенез
endothelium	эндотелий
heparin	гепарин (естественный антикоагулянт; содержится во многих тканях и тучных клетках)
glycoprotein	гликопротеин
hemostasis	остановка кровотечения, гемостаз
histamine	гистамин
hydrolase	гидролаза

immunity	иммунитет, невосприимчивость
ionised	ионизированный
lysosome	лизосома
nucleotide	нуклеотид
peptide mediator	пептидный медиатор
phosphatidylcholine	фосфатидилхолин
phospholipide	фосфолипид
platelet-leukocyte activator of aggregation or (P-selectin)	тромбоцитарно-лейкоцитарный активатор агрегации (пи-селектин)
regeneration	регенерация, восстановление
serotonin	серотонин
sphyngomyelin	сфингомиелин
thrombocytopenia	тромбо(цито)пения (пониженное содержание тромбоцитов в крови)
thrombomodulin	тромбомодулин
thromboplastin	тромбопластин
thrombospondin	тромбоспондин
vasoconstrictor	сосудосуживающий фактор, вазоконстриктор;
vasopathia (*pl.*: vasopathiae)	вазопатия
Zahn thrombus	тромб Цаана, белый тромб

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

Alfa-granules contain a set of specific, as well as non-specific peptide mediators of coagulation and anticoagulation, inflammation, regeneration and immunity. Their membranes reveal an early platelet-leukocyte activator of aggregation (P-selectin) that appears on their surfaces during the activation of platelets. α-granules also contain thrombospondin (another aggregation activator), vWF, and VIII and V coagulation factors, 4th thrombocytic heparin-neutralizing factor, β-thrombomodulin, and, finally, fibrinogen and fibronectin.

δ-granules (dense bodies) are rich in biogenous amines (histamine, serotonin and epinephrine), adenine nucleotides (ADP and ATP), ionised calcium.

So-called λ-granules are lysosomes with typical enzymatic content including acid hydrolases.

Major functions of platelets are:

— forming of a white thrombus (also called primary, or agglutination, or *the Zahn thrombus*) which is responsible for primary hemostasis (hemostasis in the vessels of diameters less than 50–100 mcm);

— triggering out a spasm in the injured blood vessels through lipid and amine vasoconstrictors;

— speeding up fibrin formation (activation of platelets results in decrease of sphyngomyelin content on their surface while its phosphatidylcholine content increases — formerly this phenomenon was called "blood thromboplastin" or "3rd platelet factor"; this results, in its turn, in the development of phospholipide-glycoprotein cradle for fibrinogenesis and growth of the thrombus; in addition, platelets produce I, V, and VIII coagulation factors);

— initiation of tissue reparation and angiogenesis on account of the growth factors (chronic thrombocytopenias result in faulty reparation of blood vessels and development of secondary vasopathiae);

— stabilization of fibrin (XIII factor) and thrombus retraction;

— anti-hemostatic functions effected through storage and liberation of antithrombin III, S- and C-proteins;

— pro- and anti- inflammatory functions effected through a set of mediators.

Hemostasis in the vessels of diameters more than 50–100 mcm takes place owing to the formation of a secondary (coagulation) thrombus. The thrombus of such kind consists of a head, a body and a tail. The head is the primary agglutination thrombus. For the coagulation thrombus to form, the presence of a great amount of stabilized fibrin polymer is necessary. This task is accomplished through the coagulation system (that is, fibrin-formation), which is activated in response to the appearance of the primary white thrombus with a negatively charged surface, or the activation of the system may be initiated by the contact of the system's proteins with polyanion molecules, or as a result of contact of f. XII with a rough surface of basal membrane revealed after endothelium damage.

3. Do the following statements agree with the information given in the reading passage?

Write

TRUE	***if the statement agrees with the information***
FALSE	***if the statement contradicts the information***
NOT GIVEN	***if there is no information***

1. Platelet granules are responsible for forming thrombi.
2. There are more than three types of platelet granules.
3. α-granules are rich in biogenous amines.
4. δ-granules also contain fibrinogen and fibronectin.
5. λ-granules contain hydrolase.

6. A white thrombus is the same as an agglutination thrombus.
7. An agglutination thrombus is the same as Zahn thrombus.
8. Zahn thrombus is responsible for primary hemostasis in the vessels of diameters more than 50–100 mcm.
9. Platelets trigger out spasm in the injured blood vessels.
10. Activation of platelets results in increase of sphyngomyelin content on their surface while its phosphatidylcholine content decreases.
11. Chronic thrombocytopenias result in regular reparation of blood vessels.
12. Platelets are responsible for stabilization of fibrin (XIII factor) and thrombus retraction.
13. Hemostasis in the vessels of diameters more than 50–100 mcm takes place owing to the formation of an agglutination thrombus.
14. A coagulation thrombus consists of a head, a body and a tail.

4. Write questions based on the statements above.

5. Work in pairs. Ask and answer the questions from exercise 4.

6. Complete the mindmaps below.

peptide mediators of
1) c _ _ gul _ ti _ n
2) i _ _ u _ ity
3) i _ fla _ _ at_ _ n
4) r _ gen _ rat _ _ n
5) a _ _ icoag _ lat _ _ n

α-granules content
6) t _ r _ m _ ospo _ _ in
7) t _ _ om _ _ mod _ l _ n
8) f _ br _ _ og _ n
9) f _ _ ron _ c _ in

δ-granules content
10) h _ st _ _ ine
11) s _ r _ t _ nin
12) ep _ n _ p _ r _ ne
13) ad _ n _ ne nucleotides
14) I _nis _ d calcium

λ-granules characteristics
15) l _ sos _ me
16) h _ dr _ lase

hemostasis
17) s _ _sm
18) v _ so _ on _ tr _ ctor
19) f _ _ rin
20) f _ b _ in _ g _n _ sis
21) r _ p _ rat _ on
22) a _ _ iog _ nes _ s
23) r _tr _ ct _ on
24) a _ ti _ _ romb _ n

thrombus
25) a _ _ lut _ na _ ion
26) c _ _ gul _ t _ on
27) h _ _ d
28) b _ _ y
29) t _ _ l

7. Now complete these sentences using the words from the completed mindmap.

Examiner: Now let's talk about platelets. What do α-granules contain?

Student: They contain a set of specific, as well as non-specific peptide mediators of *c_ _ gul _ ti _ n* and *a _ _ icoag _ lat _ _ n*, *i _ fla _ _ at _ _ n*, *r _ gen _ rat _ _ n* and *i _ _ u _ ity*.

Examiner: And can δ-granules be characterized?

Student: They are rich in biogenous amines such as *h _st _ _ine*, epinephrine, *s _ r _t _ nin* and *ep _ n _ p _ r _ ne*, *ad _ n _ ne* nucleotides and *i _nis _ d* calcium.

Examiner: Well done. What are so-called λ-granules like?

Student: They are *l_sos _ mes* with typical enzymatic content including acid *h _dr_lases*.

Examiner: Precisely. What parts does a *c_ _ gul _ ti _ n* thrombus consist of? ***Student:*** The thrombus of such kind consists of a *h _ _ d*, a *b _ _ y* and a *t _ _ l*. The head is the primary *a _ _lut _na _ion* thrombus.

8. Complete each sentence by choosing the best ending from the list a–g below.

1. α-granules contain a set of specific, as well as non-specific...
2. α-granule membranes reveal an early platelet-leukocyte activator of aggregation (P-selectin)...
3. So-called λ-granules are lysosomes...
4. White thrombus, which is also called primary, or agglutination, or the Zahn thrombus...
5. Hemostasis in the vessels of diameters more than 50–100 mcm takes place...

6. For a coagulation thrombus to form...
7. The task of a coagulation thrombus formation is accomplished through the coagulation system...

a) the presence of a great amount of fibrin is necessary.

b) that appears on their surfaces during the activation of platelets.

c) is responsible for primary hemostasis.

d) which is activated in response to the appearance of the primary white thrombus with a negatively charged surface.

e) peptide mediators of coagulation and anticoagulation, inflammation, regeneration and immunity.

f) owing to the formation of a secondary (coagulation) thrombus.

g) with typical enzymatic content including hydrolases.

9. Read the task card below.

Describe the main peculiarities of thrombosis following the plan:
What are platelet granules?
What are their functions?
How does hemostasis in the vessels of diameters more than 50–100 mcm take place?

10. Now, using the prompts from exercise 9, talk on the topic: PLATELET GRANULES.

11. Translate into English.

1. Тромбоцит окружен мембраной, под которой располагается субмембранный слой. В цитоплазме тромбоцита располагаются 4 типа содержащих медиаторы гранул.
2. α-гранулы содержат набор специфических, а также неспецифических пептидных медиаторов коагуляции и антикоагуляции, воспаления, регенерации и иммунитета.
3. δ-гранулы (плотные тельца) богаты аминами (гистамином, серотонином и адреналином), содержат АТФ, АДФ, ионизированный кальций.
4. λ-гранулы — лизосомы, содержащие активные ферменты, в том числе кислые гидролазы.
5. Основные функции тромбоцитов заключаются:

— в формировании белого тромба;

— в запуске спазма пораженных сосудов через липидные и аминовые вазоконстрикторы;

— в ускорении образования фибрина;

— в инициации восстановления ткани и ангиогенеза;

— в стабилизации полимерного фибрина и ретракции тромба;
— в хранениии и высвобождении антитромбина III, S- и C-протеинов;
— в осуществлении воспалительного и антивоспалительного эффектов посредством ряда медиаторов.

6. Гемостаз в сосудах, диаметром более 100 микрон осуществляется благодаря формированию коагуляционных тромбов.
7. Коагуляционный тромб состоит из головки, тела и хвоста.
8. Процесс тромбообразования начинается с формирования агглютинационного белого тромба.
9. Коагуляционный тромб требует образования избытка стабилизированного полимерного фибрина.

Unit 3
EMBOLISM

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

anastomosis	анастомоз, соустье
antegrade embolism	ортоградная эмболия
aorta	аорта
cerebral	церебральный, мозговой
coronary	венечный, коронарный (о сосудах сердца)
diver's disease	кессонная болезнь
embolus	(*pl.*: emboli) эмбол (циркулирующий в крови субстрат, способный вызвать закупорку кровеносного сосуда)
extremity	1) край, конец; 2) конечность
infarction	инфаркт
intravascular	внутрисосудистый, интраваскулярный
lymph	лимфа
necrosis	некроз, омертвение
occlusion	1) окклюзия, смыкание челюстей, прикус; 2) обтурация; *окклюзия*; закупорка
pulmonary	легочный, пульмональный
retrograde embolism	ретроградная эмболия
shunt	шунт; (обходной) анастомоз; шунтировать
septum (*pl.*: septa)	перегородка
sepsis	сепсис, общая гнойная инфекция
oncogenesis	онкогенез, бластомогенез
thromboembolism	*тромбоэмболия* (эмболия вследствие тромбоза)

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

Embolism is a typical pathological process of the blood or lymph transportation of emboli — particles and conglomerates not characteristic of the normal blood flow (thrombi, gas bubbles, pieces of tumor, bacterial clots, bone fragments, fat droplets, etc).

An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood or lymph to a site distant from its point of origin.

Virtually, 99% of all emboli represent some part of a dislodged thrombus, hence — the commonly used term thromboembolism.

Embolism should be considered to be thrombotic in origin. Inevitably, emboli lodge in vessels too small to permit further passage, which results in partial or complete vascular occlusion. A potential consequence of such thromboembolic events in arteries is the ischemic necrosis of down-stream tissue, known as infarction.

Embolism is classified as antegrade and retrograde, depending on whether the emboli move along with, or counter to, the normal blood- or lymph- flow. In the case of antegrade embolism the patterns of emboli travel as follows (Virchow's rules):

Emboli from veins of the major circulation of blood and the right part of the heart move into the minor circulation.

Emboli from pulmonary veins, the left part of the heart and the aorta are transported into arteries of the major circulation (coronary, cerebral, organ, extremities, etc).

Emboli from unpaired organs of the abdominal cavity are brought into the portal vein and into the liver.

In the case of retrograde embolism, emboli move against the normal blood flow. Paradoxical embolism is a variety of antegrade embolism when the emboli travel uncharacteristically due to the presence of pathologic anastomoses between the arterial and the venous parts of the circulation (intercordial septum defects, pathologic shunts).

Embolism is an important component of various pathological processes, diseases (oncogenesis, acute circulatory disorders, diver's disease, sepsis, etc).

3. Do the following statements agree with the information given in the text?

Write

TRUE	***if the statement agrees with the information***
FALSE	***if the statement contradicts the information***
NOT GIVEN	***if there is no information on this***

1. Emboli are thrombi, gas bubbles, pieces of tumor, bacterial clots, bone fragments, fat droplets which are transported by blood or lymph.
2. Embolism is a typical process of transportation of particles and conglomerates characteristic of the normal blood flow.
3. An embolus is a kind of detached intravascular mass that is carried by blood to a site distant from its point of origin.
4. Nearly all emboli are sort of dislodged thrombi.
5. Initially, the term thromboembolism was to emphasize the fact that embolism should be considered as thrombotic in origin.
6. Emboli presence in vessels results in partial or complete vascular occlusion.
7. Thromboembolic events in arteries may result in infarction.
8. Embolism is classified as antegrade and retrograde, which depends on the trajectory of the emboli movement.
9. In the case of antegrade embolism the emboli are transported from veins of the greater circulation into the lesser circulation.
10. In the case of retrograde embolism, emboli are transported in the direction counter to the normal blood flow.
11. Paradoxical embolism is a variety of antegrade embolism.
12. Such diseases as oncogenesis, acute circulatory disorders, diver's disease, sepsis are important components of embolism.

4. Write questions based on the statements above.

5. Work in pairs. Ask and answer the questions from exercise 4.

6. Complete the mindmaps below.

emboli

1) t _ _ ombi
2) gas b _ _ bles
3) pieces of t _ _ or
4) bacterial c _ _ ts
5) bone fra _ _ ents
6) fat dro _ _ ets

embolism
7) t _ _ ombotic
8) o _ _ lusion
9) in _ _ rction
10) a _ _ egrade
11) re _ _ ograde

cardiovascular system
12) v _ _ in
13) p _ _ monary
14) a _ _ rta
15) a _ _ tery
16) c _ _ onary

organs
17) e _ tr _ mities
18) ab _om _nal cavity
19) l _ _ er

diseases
20) o _ _ ogenesis
21) acute c _ _ culatory disorders
22) d _ _ er's disease
23) s _ _ sis

7. Now complete these sentences using the words from the mindmap above.

Examiner: I'd like to ask you some questions. What particles can we characterize as emboli?

Student: They are *t _ _ ombi, gas b _ _ bles, pieces of t _ _ or, bacterial c _ _ ts, bone fra _ _ ents, fat dro _ _ ets.*

Examiner: Some more questions. How should embolism be considered in terms of origin?

Student: It should be considered as *t _ _ ombotic.*

Examiner: Right you are. What dangerous situation does the presence of emboli lead to?

Student: It leads to partial or complete vascular *o _ _ lusion.*

Examiner: Precisely. So how is the potential consequence of thromboembolic events in arteries is the ischemic necrosis of down-stream tissue known?

Student: Such consequence is known as *in _ _ rction.*

Examiner: How can embolism be classified depending on whether the emboli move along with or counter to the normal blood- or lymph- flow?

Student: It can be classified as *a _ _ egrade* and *re _ _ ograde.*

Examiner: How do emboli travel in case of antegrade embolism?

Student: Emboli from *v _ _ ins* of greater circulation and the right part of the heart move into the lesser circulation. Emboli from *p _ _ monary*

veins, the left part of the heart and the *a _ _ rta* are transported into *a _ _ teries* of the greater circulation: *c _ _ onary*, cerebral, organ, extremities. At last emboli from unpaired organs of the abdominal cavity are brought into the portal vein and into the liver.

Examiner: And embolism is an important component of various diseases, isn't it?

Student: Yes, it is. Embolism is an important component of such pathological processes and diseases as

o _ _ ogenesis, acute c _ _ culatory disorders, d _ _ er's disease, *s _ _ sis*.

8. Complete each sentence by choosing the best ending from the list a–g below.

1. Emboli such as thrombi, gas bubbles, pieces of tumor, bacterial clots, bone fragments, fat droplets...
2. The presence of emboli results in...
3. A potential consequence of such thromboembolic events is the ischemic necrosis of down-stream tissue...
4. Embolism is classified as antegrade and retrograde.
5. Emboli from unpaired organs of the abdominal cavity...
6. In the case of retrograde embolism...
7. Paradoxical embolism is a variety of antegrade embolism...

a) are brought into the portal vein and into the liver.
b) when the emboli travel uncharacteristically.
c) partial or complete vascular occlusion
d) depending on whether the emboli move along with, or counter to, the normal blood- or lymph-flow.
e) are particles and conglomerates not characteristic of the normal blood flow.
f) emboli move against the normal blood flow.
g) known as infarction.

9. Read the task card below.

Describe the main peculiarities of thrombosis following the plan:
What does the term "emboli" define?
What is embolism?
What types of embolism are distinguished?
How do emboli travel in the case of antegrade embolism?
... and in the case of retrograde embolism?

10. Now, using the prompts from exercise 9, talk on the topic: EMBOLISM.

11. Translate into English.

1. Эмболией называется типовой патологический процесс циркуляции в крови и лимфе частиц и конгломератов, не свойственных нормальному кровотоку.
2. Эмбол — твердая частица, газ или жидкость, распространяемая в токе крови или лимфы.
3. Практически 99% всех случаев эмболии относятся к эмболии оторвавшимися тромбами или их фрагментами, которая носит название тромбоэмболии.
4. Эмболия чаще всего возникает вследствие тромбоза.
5. В зависимости от того, распространяются ли эмболы по току крови или лимфы или против тока, выделяют ортоградную и ретроградную формы эмболии.
6. По Р. Вирхову, в случае ортоградной эмболии:

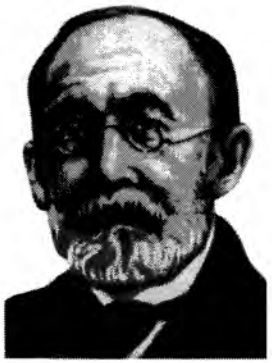
— эмболы из венозной системы большого круга кровообращения и правого сердца попадают в сосуды малого круга кровообращения;

— эмболы из легочных вен, левого сердца и аорты заносятся в артерии большого круга (коронарные, церебральные, внутренних органов, конечностей);

— эмболы, порожденные в непарных органах брюшной полости, заносятся в портальную вену и в печень.

7. При ретроградной форме эмболы движутся против тока крови.
8. Парадоксальная эмболия является разновидностью ортоградной эмболии, при которой эмболы распространяются по току крови, но из-за наличия дефектов межпредсердной либо межжелудочковой перегородки и при других пороках сердца с право-левым шунтом, получают возможность двигаться нехарактерно.
9. Эмболия является важным компонентом различных патологических процессов и заболеваний (онкогенез, острые нарушения кровообращения, кессонная болезнь, сепсис и другие).

Medicine Through Biographies



Virchow, Rudolf Ludwig Karl; "Father of Pathology" born, October 13, 1821, Schivelbein, Pomerania, Prussia, died September 5, 1902, Berlin.

German pathologist, anthropologist, archaeologist and statesman, one of the most prominent physicians of the 19th century. He pioneered the modern concept of pathological processes by his application of the cell theory to explain the effects of disease in the organs and tissues of the body. He gave the first descriptions of necrosis, apoplexy, agglutination thrombus, leukemia, formulated the first theories for pathogenesis of inflammation and neoplasia. Moreover, he founded the first research institute and published the first journal in the field of pathology, campaigned vigorously for social reforms and contributed to the development of Anthropology as a modern science. A computer quest game "Professor Virchow's Castle" created by teachers of Pathology and Morphology Departments of St. Petersburg State University is an interactive compendium of neurosciences, dedicated to Virchow's memory.

His main works (all in German) are:

Mittelheilungen über die Typhus-Epidemie, (1848)

Die Cellularpathologie, (1858), Russian translation by A. Polunin — 1859, English translation — 1860.

Handbuch Media: der speciellen Pathologie und Therapie, (1854-62)

Vorlesungen über Pathologie, (1862–1872)

Die krankhaften Geschwülste, (1863–1867)

Gegen den Antisemitismus, (1880)

Zahn, Friedrich Wilhelm born Feb. 14, 1845 in Switzerland, died Aug. 16, 1904, Switzerland.

Swiss pathologist and bacteriologist. The first head of the department of Pathology at Geneva University. He was the first to describe the white, or primary, or agglutination thrombus named after him.

Lines of Zahn are the characteristics of the thrombi that appear when formed in the heart or aorta. They have visible and microscopic laminations produced by alternating pale layers of platelets mixed with fibrin and darker layers containing red blood cells. Their presence implies thrombosis at the site of rapid blood flow. In veins or smaller arteries, where the flow is not so constant, they are less apparent. Also, Zahn was first who described paradoxical embolism.

Module 5

PATHOPHYSIOLOGY OF HEAT BALANCE. FEVER AS AN ELEMENT OF AN ACUTE PHASE RESPONSE

Unit 1

THE SYSTEM OF THERMOREGULATION. FEVER

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

anterior hypothalamus	передний гипоталамус
arachidonic acid	арахидоновая кислота
cytokine	цитокин
endogenous	эндогенный, возникший внутри организма
endotheliocyte	эндотелиоцит, эндотелиальная клетка
endotoxin	эндотоксин
exogenous	экзогенный, вызываемый внешними причинами
fever	жар, лихорадочное состояние; лихорадка; вызывать жар; бросать в жар, лихорадить
fungus	fungus (*pl.*: fungi); 1) гриб; грибок; плесень; 2) грибовидное разрастание (ткани)
intact	1) интактный, здоровый; незараженный, неосложненный; неповрежденный; 2) исходный (напр. о штамме микроорганизмов)
metabolite	метаболит (продукт метаболизма)
mononuclear	мононуклеар, одноядерная клетка; мононуклеарный лейкоцит, одноядерный
polymorphonuclear	полиморфно-ядерный (лейкоцит)
polysaccharide	полисахарид
prostaglandin	простагландин

pyrogen	1) пироген, пирогенное вещество; 2) фактор, вызывающий лихорадку
rear hypothalamus	задний гипоталамус
thermoregulation	терморегуляция
thermosensor	термосенсор
vasodilatation	вазодилатация, расширение кровеносных сосудов
zymosan	зимозан

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo.

After that read the text aloud, trying to imitate the intonation.

The system of thermoregulation consists of a number of elements with interconnected functions. Thermal status information is brought by afferent nerves from the surface and deep-seated thermosensors to the thermoregulation centre in the hypothalamus ("a thermostat of the organism"). After that the centre of thermoregulation activates various mechanisms providing either for heat production or heat emission. Feed-back control is accomplished through the nervous system and blood circulation that change the sensitivity of the thermosensors, thus completing the process. Surgical separation of the hypothalamus from the underlying parts of the brain and spinal cord makes regulation of the body's temperature impossible.

Motor centres of the anterior hypothalamus are sensitive to the temperature of the blood flow. They control heat emission through vasodilatation, sweating and heat breathing, thereby preventing overheating.

The center located in the rear hypothalamus controls the level of thermoproduction through activating shivering and non-shivering thermogenesis, so preventing overcooling.

Fever (febris, pyresis) is a general typical pathological process (TPP) developing in response to pyrogens occuring in the highest homeothermal animals and in man.

This TPP is characterized by a temporal change of the set-point in the system of temperature homeostasis at a higher level, with mechanisms of thermoregulation remaining intact. Fever is the result of the influence of endogenous pyrogens upon hypothalamic nuclei responsible for thermoregulation.

As endogenous pyrogens arise during preimmune (acute-phase) response, fever may be regarded as an element of preimmune (acute-phase) response.

Exogenous pyro-(febri-)gens are bacterial endotoxins (lipopolysacharides — LPS, soluble germ antigens), zymosan and other polysaccharide substances produced by fungi, etc. Exogenous pyro-(febri-)gens induce production of cytokines: interleukin-1 (IL-1), tumor necrosis factor (TNF) and interleukin-6 (IL-6) by mononuclear and polymorphonuclear leukocytes, endotheliocytes and certain macrophage-like antigen-presenting cells. These cytokines are known as endogenous pyrogens.

In their turn, IL-1, TNF and IL-6 exert influence upon non-specific central thermal sensors which generate "the reference signal" ("set-point"), and shift the "set-point" to a higher level. At the same time, endogenous pyrogens induce the endotheliocytes of OVLT (Organum Vasculosum Laminae Terminalis) to produce metabolites of arachidonic acid. These metabolites (prostaglandins, PG) diffuse into the neurons of the preoptic part of the frontal hypothalamus and induce the neurons to generate the "set-point signal". The main mediator among prostaglandins is prostaglandin E_2.

3. Complete the sentences below. Insert NO MORE THAN THREE WORDS in each gap.

1. Thermal status information is brought by _____ to the thermoregulation centre in the hypothalamus.
2. The centre of thermoregulation activates various mechanisms providing either for _____ or heat emission.
3. Surgical separation of the _____ from the underlying parts of the brain and spinal cord makes regulation of the body's temperature impossible.
4. The system of thermoregulation consists of a _____ with interconnected functions.
5. Feed-back control is accomplished through the nervous system and _____ that change the sensitivity of the thermosensors, thus completing the process.
6. Motor centres of the anterior hypothalamus control _____.

4. Choose the correct answer from A, B or C.

1. Motor centres prevent organism from _____.

A overheating.
B thermoproduction.
C vasodilatation.

2. The center located in the rear hypothalamus controls the level of _____.

A thermoproduction.
B thermogenesis.
A overheating.

3. Fever is a general typical pathological process developing in response to _____ occuring in the highest homeothermal animals and in man.

A thermogenesis.

B overheating.

C pyrogens.

4. Fever is characterized by the temporal change of the set-point in the system of temperature homeostasis at a _____.

A higher level.

B lower level.

C medium level.

5. Fever is the result of the influence of _____ pyrogens upon hypothalamic nuclei responsible for thermoregulation.

A intact.

B exogenous.

C endogenous.

6. Exogenous pyrogens are bacterial endotoxins, zymosan and other polysaccharide substances produced by _____.

A bacteria.

B germs.

C fungi.

7. Fever may be regarded as an element of _____ response.

A immune.

B preimmune.

C autoimmune.

8 Exogenous pyrogens induce production of _____ pyrogens.

A exogenous.

B intact.

C endogenous.

5. Answer the questions:

1. *Which organ is responsible for the accumulation of thermal status information?*
2. *Which organ activates various thermoproduction mechanisms?*
3. *Which organ activates various heat emission mechanisms?*
4. *What kind of surgical operation makes regulation of the body's temperature impossible?*
5. *What do motor centres of the anterior hypothalamus control?*
6. *What does the centre located in the rear hypothalamus control?*
7. *What causes the development of fever?*
8 *Do thermoregulation mechanisms remain intact or out of function during fever?*
9. *What exogenous pyrogens are referred to bacterial endotoxins?*

10. What pyrogens induce production of some endogenous pyrogens?
11. What function do endogenous pyrogens perform?

6. Complete the following sentences using an appropriate word from the box.

homeostasis frontal anterior elements thermal endogenous polysaccharide preimmune rear hypothalamus circulation pyrogens thermoproduction

1. The system of thermoregulation consists of a number of _____.
2. Thermal status information is brought by afferent nerves to the _____.
3. The centre of thermoregulation in the hypothalamus activates various mechanisms of _____ or heat emission.
4. Feed-back control is accomplished through the nervous system and blood _____.
5. Motor centres of the _____ hypothalamus control heat emission.
6. The center located in the _____ hypothalamus controls the level of thermoproduction.
7. Fever develops in response to _____.
8. Fever is characterized by temporal change of the set-point in the system of temperature _____.
9. Fever may be regarded as an element of _____ response.
10. Exogenous pyrogens are bacterial endotoxins, exotoxins, zymosan and other _____ substances.
11. Exogenous pyrogens induce production of IL-1, TNF and IL-6 _____ pyrogens.
12. TNF and IL-6 exert influence upon non-specific central _____ sensors.
13. Prostaglandins diffuse into neurons of preoptic part of the _____ hypothalamus and induce the neurons to generate the "set-point signal".

7. Complete the conversation with the words from the box.

surgical pyrogens prostaglandins underlying vasodilatation set-point thermogenesis arachidonic intact hypothalamus afferent fungi induce circulation heat emission heat breathing

Examiner: Now let's talk about the relation between thermoregulation and fever. I'd like you to tell me where the thermoregulation centre is located and what kind of information is brought to it by afferent nerves.

Student: Well, the thermoregulation centre is located in the **1.** _____ and thermal status information is brought to it by **2.** _____ nerves.

Examiner: And what happens after that?

Student: After that the centre of thermoregulation activates various mechanisms providing either for thermoproduction or **3.** _____.

Examiner: Right. And what is feed-back control accomplished through?

Student: It is accomplished through the nervous system and blood **4.** _____.

Examiner: What kind of surgical operation makes regulation of the body's temperature impossible?

Student: It happens in the case of **5.** _____ separation of the hypothalamus from the **6.** _____ parts of the brain and spinal cord.

Examiner: How do motor centres of the anterior hypothalamus control heat emission to prevent overheating?

Student: They control it through **7.** _____, sweating and **8.** _____.

Examiner: And what does the center located in the rear hypothalamus control to prevent overcooling?

Student: It controls the level of thermoproduction through activating shivering and non-shivering **9.** _____.

Examiner: What causes the development of fever?

Student: Fever develops in response to **10.** _____ occurring in the highest homeothermal animals and in man.

Examiner: What kind of temporal change is fever characterized?

Student: It is characterized by the temporal change of the **11.** _____ in the system of temperature homeostasis at a higher level, with mechanisms of thermoregulation remaining **12.** _____.

Examiner: By what organisms are exogenous pyrogens produced?

Student: They're produced by **13.** _____.

Examiner: And what production do exogenous pyrogens bring about?

Student: Exogenous pyrogens **14.** _____ production of IL-1, TNF and IL-6 endogenous pyrogens.

Examiner: By what are endogenous pyrogens connected with the "set-point signal" generation?

Student: They induce the endotheliocytes of OVLT or Organum Vasculosum Laminae Terminalis to produce metabolites of **15.** _____ acid. These metabolites, named **16.** _____, diffuse into the neurons of the preoptic part of the frontal hypothalamus and induce the neurons to generate the "set-point signal".

8. Work in pairs. Act out the dialogue from exercise 7 with your partner.

9. Read the task card below.

Describe the main peculiarities of the system of thermoregulation and fever following the plan:

How does the system of thermoregulation work?
What is fever?
What are exogenous and endogenous pyrogens?

10. Now, using the prompts from exercise, talk on the topic: the system of thermoregulation. fever.

11. Translate into English.

1. Центр терморегуляции активирует различные механизмы, обеспечивающие как теплопродукцию, так и теплоотдачу.
2. Моторные центры переднего гипоталамуса чувствительны к температуре крови, снабжающей их.
3. Центр, расположенный в заднем гипоталамусе, контролирует уровень термопродукции через управление сократительным термогенезом, а также несократительным термогенезом.
4. Лихорадка — общий типовой патологический процесс, присутствующий у человека и гомойотермных животных, и состоящий в ответе на пирогены, которые смещают «установочную точку» на более высокий уровень при сохранении механизмов терморегуляции.
5. Лихорадка характеризуется временным смещением установочной точки температурного гомеостаза на более высокий уровень при сохранении механизмов терморегуляции.
6. Экзогенные пирогены — компоненты инфекционных возбудителей, индуцирующие производство лейкоцитами, эндотелием и макрофагоподобными антигенпредставляющими клетками эндогенных пирогенов (цитокинов).
7. К эндогенным пирогенам относятся ИЛ-1, ИЛ-6, ФНО.
8. ИЛ-1, ИЛ-6 и ФНО приводят в действие механизм запуска лихорадки.

Unit 2

FEVER IN ADULTS EXPERIMENTAL FEVER· HYPERTHERMIA

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

adrenal medulla	мозговое вещество надпочечников
aldosterone	альдостерон
antipyretic	жаропонижающее средство, антипиретик; жаропонижающий, противолихорадочный
arginine-vasopressin	агринин-вазопрессин
arterial blood pressure	артериальное кровяное давление
axis	ось (в эндокринологии — регуляторный контур, включающий гипоталамо-гипофизарное звено и периферическую эндокринную железу с их прямыми и обратными связями)
bacterium (*pl.*: bacteria)	бактерия (бактерии)
carbonuria	карбонурия (повышение содержания общего углерода в моче)
cerebral ventricles	желудочки головного мозга
circulatory disorders	нарушения ковообращения
collapse	коллапс (острая недостаточность кровообращения без его централизации)
cortisol	кортизол, гидрокортизон
critical	критический, угрожающий
dehydration	обезвоживание, дегидратация
dipeptide	дипептид
diuresis	диурез (1) процесс образования и выделения мочи; 2) количество мочи, выводимое из организма за определенное время)
heat loss (heat emission)	теплоотдача
hyperthermia	гипертермия, перегревание организма
hypophysis (*syn.*: pituitary)	гипофиз, питуитарная железа, нижний мозговой придаток
hypoxia	гипоксия, кислородное голодание, кислородная недостаточность
intravenous injection	внутривенная инъекция
febrile	лихорадочный, фебрильный, имеющий отношение к лихорадке
ketone	кетон
oscillator	осциллятор, колебательный контур
phagocyte	фагоцит, фагоцитирующая клетка; фагоцитировать

Pyrogenalum	Пирогенал *(препарат)*
respiratory coefficient	дыхательный коэффициент
somatostatin	соматостатин
stadium decrementi	стадия понижения температуры
stadium fastigii or acme	стадия стояния температуры (акме)
stadium incrementi	стадия повышения температуры
sympathetic	симпатический (относящийся к симпатической нервной системе)
thyroid	щитовидная железа; щитовидный, тироидный
total nitrogen	общий азот (биохимический показатель)
urinary	мочевой, мочеиспускательный
vagal	вагусный (относящийся к блуждающему нерву)
vasoconstriction	вазоконстрикция, сужение кровеносных сосудов

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

The mechanism of febrile increasing temperature in adults is characterized by the limitation of heat emission with the simultaneous intensification of heat production. Limitation of heat loss is realized by means of sympathetic vasoconstriction of skin blood vessels. The decrease in skin temperature is responsible for the afferent signalization of cold thermo receptors and shivering. The spinal oscillator is responsible for the shivering reflex (simultaneous contraction of flexors and extensors). Contractile and non-contractile thermogeneses are increased by 20 to 60%, first, on account of sympathetic reflexes and stimulation of the adrenal medulla and, further, on account of the activation of the hypothalamus — hypophysis–thyroid axis. These changes are the first stage of the fever — stadium incrementi — (the stage of increasing temperature lasting 3 to 4 hours). Domination of carbohydrates oxidation causes the increase of the respiratory coefficient up to 1.0.

Stadium fastigii, or acme (the stage of constant temperature) is the evidence of a new set-point of temperature homeostasis having been reached. From this point thermoregulation is accomplished through the normal mechanisms. Heat production and heat emission are balanced. Skin vessels are widened. Breathing is frequent. Heart beats are frequent. Skin temperature is increased. Chill and shivering are gone.

Diuresis is limited. Activation of the hypothalamus-hypophysis-adrenal cortex axis and hypothalamus-hypophysis-thyroid axis causes changes in the hormonal status (increased cortisol, aldosterone and thyroid hormone levels) and, as a consequence, there are changes in metabolism. The dominance of contrainsulin hormones results in gluconeogenesis activation, decrease in protein synthesis and intensification in aminoacid and fat disintegration. The resulting aminoacids and fatty acid metabolites, like the metabolites of Krebs[1], cycle, serve as gluconeogenesis substrates. The dominance of fat oxidation causes a decrease of the respiratory coefficient down to 0.7. Rubner[2]'s urinary coefficient (total carbon (C) / total nitrogen (N) in urine) increases as a result of urinary excretion of a considerable amount of underoxidized carbon as part of ketones ("Disoxidative carbonuria").

Stadium decrementi (the stage of temperature decrease) occurs as a result of recourse of the set-point of temperature homeostasis down to the normal level after the exogenous pyrogens have been exhausted, while the endogenous ones are no longer formed.

The levels of natural antipyretics, sometimes called "anti-pyrogenes": arginine-vasopressin, $ACTH_{1-39}$, MSH, endorphins, somatostatin and others) are increased. The previously increased levels of some hormones (aldosterone, cortisol, thyroid hormones) return down to normal. Metabolism is normalized. The respiratory coefficient rises up to 0.8, the Rubner's urinary coefficient decreases. Heat loss is considerably intensified. Skin vessels are enlarged. Diuresis and sweating are increased. Heat emission exceeds heat production.

Temperature fall may be gradual (lytic) or rapid (critical). In the latter case abrupt dilatation of blood vessels ("vagal enlargement") may cause sharp decrease of peripheral resistance and arterial blood pressure. This may be complicated by vaso-vagal collapse.

EXPERIMENTAL FEVER

Pyrogenalum is a highly potent nonprotein exogenous pyrogen consisting of LPS. It may be synthesized or produced by Pseudomonas aeruginosa bacteria and by Salmonellae of Typhus-Paratyphus group. The onset of fever in response to pyrogen injection begins after a certain latent period whose duration is shorter in the case of an endogenous pyrogen injection. The latent period depends on the nature and potency of pyrogen, and the way of injection. In the case of intravenous injection the latent period is 15–40 minutes, in the case of subcutaneous injection it is 30 to 80 minutes, in the case of injection into cerebral ventricles —

[1] Sir Hans Adolf Krebs, German (after 1933 — English) biochemist, born August 25, 1900, Hildesheim; died November 22, 1981, Oxford. Nobel prize winner of 1953 in Physiology or Medicine.

[2] Max Rubner (1854–1932), German physiologist, pathologist and hygienist. Founder of scientific Dietetics and Nutritiology, pioneer of biocalorimetry, who has precisely correlated animal heat to oxidation and catabolism in the body.

it is considerably less than 15 minutes. Repeated injections of exogenous pyrogens produce tolerance, while repeated injections of endogenous ones do not. The blockade of the system of mononuclear phagocytes with Indian ink prevents development of fever after introduction of exogenous pyrogens.

Hyperthermia, unlike fever, is a temporary increase of body temperature due to the disturbance of thermoregulatory mechanisms and brake-down of the balance between thermogenesis and heat emission (loss). Hyperthermia is extremal heat stress, when the ability of the organism to emit heat is lower than heat production, hence an abnormal rise of body temperature against the background of thermoregulatory failure. Hyperthermia results in dehydration, circulatory disorders and hypoxia. Fever does not produce either of the aforementioned changes characteristic of hyperthermia.

3. Complete the sentences below. Insert NO MORE THAN THREE WORDS in each gap.

1. Fever in adults is characterized by the limitation of _____ with the simultaneous intensification of _____.

The limitation of heat loss is carried out by the vasoconstriction of _____.

2. The decrease in skin temperature results in the _____ signalization of cold thermo receptors and shivering.

3. The spinal oscillator is responsible for the shivering reflex showing itself as simultaneous contraction of _____.

4. The first stage of the fever is _____.

5. Contractile and non-contractile thermogeneses increase from 20% to 60% due to _____ and the stimulation of the _____.

6. At stadium incrementi the activation of the hypothalamus-hypophysis-_____ takes place.

4. Choose the correct answer from A, B or C.

1. Domination of carbohydrates oxidation causes the increase of the respiratory _____ up to 1.0.

A capacity

B expansion

C coefficient

2. Heat production and heat _____ are balanced at stadium fastigii.

A exhaustion

B elimination

C emission

3. Stadium decrementi results from a recourse of the set-point of temperature _____ down to normal.

A homeostasis

B drop

C indicator

4. The onset of fever in response to pyrogen injection begins after a certain _____ period.

A calm

B latent

C important

5. The latent period depends on the nature and potency of _____, and the way of injection.

A solution

B drug

C pyrogen

6. Repeated injections of _____ pyrogens produce tolerance.

A exogenous

B different

C endogenous

7. Hyperthermia, unlike fever, is a _____ increase of body temperature.

A constant

B temporary

C sudden

8 Hyperthermia results in dehydration, circulatory _____ and hypoxia.

A effect

B collapse

C disorders

5. Answer the questions.

1. *What are the characteristics of the mechanism of fever?*
2. *In what way is limitation of heat emission realized?*
3. *What does the decrease in skin temperature result in?*
4. *What is the spinal oscillator responsible for?*
5. *Why are contractile and non-contractile thermogeneses increased?*
6. *How long does the first stage of fever last?*
7. *What does stadium fastigii, or acme, signify?*
8. *What is the heat production and heat emission relationship like at stadium fastigii?*
9. *What does stadium decrementi result from?*
10. *What is the relationship between heat production and heat emission at stadium decrementi?*
11. *What can complicate abrupt enlargement of blood vessels in the case of critical temperature fall?*
12. *What is the effect of repeated injections of exogenous pyrogens and of endogenous pyrogens?*
13. *What is the difference between hyperthermia and fever?*

6. Complete the following sentences using an appropriate verb from the box.

Heat emission heat production temporary mechanisms thermogenesis vasoconstriction afferent spinal extensors stadium incrementi coefficient set-point constant homeostasis collapse pyrogen latent injection Indian ink hyperthermia dehydration hypoxia

1. Fever is characterized by the limitation of _____ with the simultaneous intensification of _____.
2. Hyperthermia, unlike fever, is a _____ increase of body temperature due to the disturbance of thermoregulatory _____ and brake-down of balance between _____ and heat emission (evolution).
3. Limitation of heat emission is realized by sympathetic _____ of skin blood vessels.
4. The _____ signalization of cold thermo receptors and shivering result from the decrease in skin temperature.
5. The _____ oscillator is responsible for the shivering reflex (simultaneous contraction of flexors and _____).
6. The first stage of fever is _____, characterized by increasing temperature lasts, from 3 to 4 hours.
7. At the first stage of fever the respiratory _____ increases up to 1.0.
8. A new _____ of temperature homeostasis is reached at the stage of _____ temperature.
9. The stage of temperature decrease results from the recourse of the set-point of temperature _____ down to the normal level.
10. In case of critical temperature fall abrupt enlargement of blood vessels may be complicated by _____.
11. The onset of fever in response to _____ injection begins after a certain _____ period.
12. The latent period depends on the nature and potency of pyrogen, and the way of _____.
13. The blockade of the system of mononuclear phagocytes with _____ prevents the development of fever after introduction of exogenous pyrogens.
14. At _____ the ability of the organism to emit heat is lower than heat production.
15. Hyperthermia results in _____, circulatory disorders and _____.

7. Complete the conversation with the words from the box.

homeostasis shivering skin sympathetic (x2) hypoxia heat loss potency exogenous dehydration flexors activation adrenal medulla sympathetic injection latent collapse vagal exceeds endogenous

Examiner: Let's discuss the fever in adults as well as experimental fever and hyperthermia. To begin with, how do fever and hyperthermia differ?

Student: Well, fever is characterized by the limitation of **1.** _____ with the simultaneous intensification of heat production while hyperthermia is a temporary increase of body temperature due to the disturbance of thermoregulatory mechanisms and brake-down of brake-down of the balance between thermogenesis and heat emission.

Examiner: What does hyperthermia result in?

Student: Hyperthermia results in **2.** _____, circulatory disorders and **3.** _____ Fever does not produce any of the aforementioned consequences.

Examiner: What is the limitation of heat emission realized by?

Student: It's realized by means of **4.** _____ vasoconstriction of skin blood vessels.

Examiner: What do the afferent signalization of cold thermo receptors and shivering result from?

Student: They result from the decrease in **5.** _____ temperature.

Examiner: What is the spinal oscillator responsible for?

Student: It is responsible for the **6.** _____ reflex, that is simultaneous contraction of **7.** _____ and extensors.

Examiner: Why are contractile and non-contractile thermogeneses are increased?

Student: They're increased on account of **8.** _____ reflexes and stimulation of the **9.** _____ and, further, on account of the **10.** _____ of the hypothalamus-hypophysis-thyroid axis.

Examiner: What does the stage of constant temperature signify?

Student: This stage is the evidence of a new set-point of temperature **11.** _____ having been reached.

Examiner: What does the stage of temperature decrease result from?

Student: This stage results from the recourse of the set-point of temperature homeostasis down to the normal level after the **12.** _____ pyrogens have been exhausted, while the **13.** _____ ones are no longer formed.

Examiner: And what is the balance of heat loss and heat production at this stage?

Student: Heat emission **14.** _____ heat production.

Examiner: OK. What can happen in the case of quick temperature fall?

Student: In this case abrupt enlargement of blood vessels, or **15.** _____ enlargement, may be complicated by **16.** _____.

Examiner: When does the onset of fever in response to pyrogen injection occur?

Student: It begins after a certain **17.** _____period whose duration is shorter in the case of an endogenous pyrogen **18.** _____.

Examiner: And, finally, what does the length of the latent period depend on?

Student: In depends on the nature and **19.** _____ of a pyrogen, and the way of injection.

8. Work in pairs. Act out the dialogue from exercise 7 with your partner.

9. Read the task card below.

Describe the main peculiarities of fever following the plan:
What is the mechanism of fever?
How is experimental fever effected?
How do fever and hyperthermia differ?

10. Now, using the prompts from exercise 9, talk on the topic: fever.

11. Translate into English.

1. Первый период лихорадки у взрослых характеризуется значительным уменьшением теплоотдачи и одновременно некоторым увеличением теплопродукции.
2. Первая стадия лихорадки, которая называется также стадией повышения температуры или восходящей стадией, длится 3–4 часа.
3. Стадия стояния температуры означает, что достигнута новая установочная точка температурного гомеостаза. На высоте этой стадии терморегуляция осуществляется по механизмам, аналогичным норме. Теплопродукция и теплоотдача уравновешены. Кожные сосуды расширены. Дыхание учащено. Температура кожи увеличилась, а озноб и дрожь исчезли. Диурез ограничен.
4. Стадия понижения температуры наступает при исчерпании экзогенных пирогенов, прекращении продукции эндогенных пирогенов и под действием естественных антипиретиков. В эту стадию увеличиваются перспирация и диурез. Теплоотдача превышает теплопродукцию.

5. Падение температуры может быть постепенным — литическим и быстрым — критическим. В последнем случае обычно наблюдается настолько резкое расширение кожных кровеносных сосудов, что это может осложниться снижением периферического сопротивления, падением артериального кровяного давления и коллапсом.
6. Приступ лихорадки, вызванной инъекцией пирогенов, наступает вслед за латентным периодом, продолжительность которого уменьшена после инъекции эндогенных пирогенов.
7. Гипертермия (перегревание) приводит к обезвоживанию, нарушениям кровообращения и гипоксии.

Medicine Through Biographies

Veselkin Pyotr Nikolaevich (1904–1984) — outstanding Soviet pathophysiologist, grandson of prominent Russian pathophysiologist P. M. Albitsky, pupil of N. N. Anichkoff and L.R.Perelman. He gradiuated from the Military Medical Academy and worked at the Institute of Experimental Medicine in Leningrad. He is the founder of the modern doctrine of fever and pyrogens, and predecessor of the modern concept of cytokine regulation in acute phase response. P. N. Veselkin was a teacher of academician Helena A. Korneva and other outstanding pathophysiologists.

Dinarello, Charles (born 1943 in Boston, MA), American pathologist, internist and pediatrician, professor of Medicine in the University of Colorado at Denver. He is an expert on inflammatory and febrile cytokines, specifically interleukin-1 (IL-1). Dinarello received his M.D. degree in 1969 at Yale University, and his clinical training at the Massachusetts General Hospital, being a pupil of Sheldon M. Wolff, a prominent clinician. From 1971 to 1974, he was a clinical associate and from 1975 to 1977 a senior investigator at the National Institute of Health in Bethesda. Dr. Dinarello has published over

450 original research articles. The Institute for Scientific Information lists him as the world's third most cited life scientist. He is considered to be one of the founding fathers of cytokine regulation doctrine, having purified and cloned IL-1. This important step established the validation of cytokines as mediators of disease, particularly of inflammation, acute phase response, immune response and fever. Dinarello's recent studies on blocking IL-1 in humans (anti-cytokine therapy) by means of specific antibodies support pivotal contributions to cytokine network understanding and to the pathogenesis of inflammatory diseases (adopted from: www.frontiersin.org/people/charlesdinarello/19927).

Module 6

THE ORIGIN AND EVOLUTION OF THE CONCEPT OF ALLERGY

Unit 1

ALLERGY[1]

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

aerial	воздушный; передаваемый воздушно-капельным путем (напр. об инфекции), переносимый воздушным путем (напр. о поллютанте)
allergen	аллерген
anaphylaxis	анафилаксия (реагин-опосредованная аллергическая реакция немедленного типа). *В американских текстах может использоваться как синоним слова «аллергия», что семантически неточно*
antigenic	антигенный
antioxidant	антиоксидант, ингибитор окисления
autoallergy	аутоаллергия
autoantigen	аутоантиген
hapten	гаптен (неполный антиген)
hyperergic inflammation	гиперегическое воспаление
hypersensitivity	гиперчувствительность, повышенная чувствительность; *в американских текстах используется вместо термина «аллергия»*

[1] The term "allergy" in the sense of "immunological hypersensitivity of any type" is used in European texts. American special literature avoids the use of this term in broad meaning. American medical tradition most frequently uses the term "hypersensitivity" as a collection name for different types of immunopathologic reactions. Many American authors limit the meaning of the term "allergy" and interpret it exclusively as a synonym of "anaphylaxis", which is not considered to be right by European authors.

idiosyncrasy	идиосинкразия, индивидуальная непереносимость, *исторически применялось к любой, а в настоящее время — лишь к неиммунологической повышенная чувствительности,*
immunoglobulin	иммуноглобулин, антитело (устар.: иммунный глобулин)
life span	продолжительность (срок) жизни, время жизни
nitrophenol	нитрофенол
pediatrician	педиатр
pollutant	загрязняющее вещество, загрязнитель, поллютант; примеси (в воздухе)
practitioner	практический врач, практикующий врач, врач-практик
sensitization	сенсибилизация; в аллергологии — первичный иммунный ответ
systemic	системный, общий, относящийся ко всему организму.

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

A. ***Allergy*** (after Greek αλλον — another, εργον — I act.) also called in many American texts "***Hypersensitivity***" is a collectiive term designating a group of various typical immunopathological processes which develop in the sensitized organisms of genetically predisposed individuals during repeated immune response to the antigen that earlier provoked sensitization. Each one of these processes, in spite of the difference in their mechanisms and signs, is, as a rule, manifested in hyperergic inflammation. When regarded as the cause of allergy, antigens are called ***allergens.***

B. In clinical language, any adverse immune reaction which is too intensive, or qualitatively unusual, is referred to as "hypersensitivity", because it incurs pathological sequels. It is necessary to stress that, for practitioners, allergy is a relatively defined term applicable, first of all, to excessive or poorly regulated immune response causing obvious tissue injury not justified by the scale of achieved defensive effect.

C. If an allergic reaction is directed against autoantigens, it is called autoallergy. Abnormal autoallergy is distinct from physiological autoimmunity, as well as normoergic immune response is distinct from hyperergic allergy, although their mechanisms are similar. Moderate

autoimmunity is necessary for regulation of cell growth and functions, but excessive autoallergy is able to cause autoimmune diseases. Allergy as an experimental phenomenon was originally developed by French pathophysiologists Ch. Richet[1] and P. Portier[2] (1902) on the yacht of Grimaldi, Prince of Monaco during their sea trip on the Mediterranean. The phenomenon was reproduced as an extraordinarily strong systemic reaction in dogs after repeated injection of sea anemones' extract into them. Clinical cases of allergy were described long before this research. Ancient Egyptian chroniclers reported that one of the Pharaohs died, after being stung by a bee, with the manifestations resembling what we now call ***systemic anaphylaxis***. F. Magandie[3] (1809) described cases of "idiosyncrasy" towards some substances, to which the majority of patients are indifferent, and explained them by individual specificity of the nervous system.

D. In the beginning of the twentieth century allergy was considered to be a rare anomaly of body constitution. It is witnessed by the very origin of the word "allergy", coined by the Austrian pediatrician C. von Pirquet[4] (1906) to designate a deviating reactivity. But nowadays, when about 10% of the Earth's population, according WHO estimates, suffer from some allergic disorders, allergy is no longer regarded as something extraodinary, but rather as a general calamity! Among children the percentage of allergic individuals is greater — up to 20% and even 30%.

E. Allergy is a problem of an organism which is genetically predetermined to respond exceptionally to an antigenic challenge. It is necessary to emphasize that allergy does not only result from the evil character of an allergen itself. But some antigens, for example, the components of ragweed or mites, of worms provoke allergy in especially large contingents of people.

F. It seems that a considerable percentage of individuals, genetically predisposed to allergy, have always existed in human population, while, previously such reactivity was rarely displayed. Now this peculiar reactivity has become more pronounced due to the longer duration of life span and some other reasons.

Such broad manifestation of allergy in the population can be accounted for by the deplorable state of antigenic and haptenic burden of a modern man. First of all, it is the deteriorating ecological environment. For example, in Australia in the mid — 1970-s the annual consumption of different food additives, alien to human body (preservants, antioxi-

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[1] Charles Robert Richet, French pathophysiologist, born August 26, 1850, Paris; died December 4, 1935, Paris. Nobel Prize winner of 1913 in Physiology or Medicine. Also was prolific writer and poet, dedicated pacifist and metaphysical philosopher interested in Occultism and Ezotericism (non-scholarly science).

[2] Paul Portier (1866–1952), French pathophysiologist and immunologist.

[3] François Magendie, French physiologist and pathophysiologist, born October 6, 1783, Bordeaux; died October 7, 1855, Sannois, département Seine-et-Oise. Authored the first textbook in Experimental Pathophysiology, also made an outstanding contribution into Pharmacology.

[4] Clemens Peter Pirquet von Cesenatico, Austrian paediatrician, born May 12, 1874, Hirschstetten near Vienna; died February 28, 1929, Vienna.

dants, dyes, pollutants, and drugs) reached the level of about 23 kilos per capita! The famous Russian internist academician Ye. M. Tareev[1] once said that the passion to swallow tablets is as characteristic of a modern man, as it was natural for a pre-historical man to hold himself erect.

G. In the good olden times our ancestors lived all their lifes on relatively small territories, accustomed to a constant antigenic environment. Nowadays people travel a lot over great distances, and willingly introduce innovations into their home environment. And even without distant travel, just living day by day somewhere in Peoria, State of Illinois, one has every opportunity to visit the nearest supermarket risking direct contact with all kinds of allergens from all 6 continents and 5 oceans, including the Aerial one. For example, nitrophenols, which are universally spread in the air, water and food, may act as haptens, cross-reacting with some autoantigens (brain proteins, MHC markers and immunoglobulins), causing various manifestations of autoallergy.

Let us dwell no longer on the subject of poor ecology so often blamed by journalists and public activists so that this important cause of growing allergy was not reduced for triviality.

3. Look through the text to find out what topic each paragraph is concerned with.

Autoallergy	**A**
Environment and allergy	**B**
General definition of allergy	**C**
The cause of current broad manifestation of allergy	**D**
Little spread of allergy at the beginning of the twentieth century	**E**
Predisposition to allergy of a considerable percentage of individuals	**F**
Sources of allergy	**G**
Definition of allergy in terms of clinical language	**H**

4. Complete each sentence with the correct ending A–K.

A body constitution
B tissue injury
C allergic disorders
D antigenic challenge
E clinical language
F modern man
G French scientists
H antigenic environment

[1] Yevgenij Mikhailovich Tareev, born May 13 (25) 1895, Pskov — died August 17 1986, Moscow, outstanding Soviet internist, made great contribution into Nephrology, Rheumatology and Hepatology.

I systemic anaphylaxis
J predisposed individuals
K autoimmune diseases

1. Allergy develops in sensitized organisms of genetically _____.
2. Any adverse immune reaction which is too intensive or qualitatively unusual is referred to as "hypersensitivity" in _____.
3. For many practitioners, allergy is defined as poorly regulated immune response causing obvious _____.
4. Under certain conditions autoallergy is able to cause _____.
5. Allergy as an experimental phenomenon was originally developed by _____.
6. One of the Egyptian Pharaohs died, after being stung by a bee, with the manifestations resembling what we now call _____.
7. At the beginning of the twentieth century allergy was considered to be a rare anomaly of _____.
8. Nowadays about 10% of the Earth's population suffer from some _____.
9. Allergy is genetically predetermined to respond exceptionally to an _____.
10. Broad manifestation of allergy can be accounted for by a deplorable state of antigenic and haptenic burden of a _____.
11. Our ancestors were accustomed to a constant _____.

5. Unjumble these jumbled words.

1) oensitiszatin
2) ihpersestinvityy
3) snaphylaxia
4) lergensal
5) pnistrohenol
6) nutoantigeas
7) hallceeng
8) llinicac
9) geviatdin
10) tnigenica
11) chatenip
12) eaditivds
13) yllearg

6. Use the unjumbled words from exercise 5 to complete the sentences.

1. In many American texts allergy is called "*ihpersestinvityy*".
2. *oensitiSzatin* is provoked by antigens.
3. When regarded as the cause of *ylalerg*, antigens are called allergens.

4. In *llinicac* language, any adverse immune reaction which is too intensive, or qualitatively unusual, is referred to as "hypersensitivity".
5. In the case of autoallergy an allergic reaction is directed against *nutoantigeas*.
6. One of the Egyptian Pharaohs died, after being stung by a bee, with the manifestations resembling what we now call "systemic *snaphylaxia*".
7. In the beginning of the twentieth century the word "allergy" designated a *geviatdin* reactivity.
8. Now allergy is treated as a problem of an organism which is genetically predetermined to respond exceptionally to an antigenic *hallceeng*.
9. Broad manifestation of allergy can be explained by the deplorable state of antigenic and *chatenip* burden of a modern man.
10. In Australia in the mid — 1970s the annual consumption of different food *eaditivds* alien to human body reached the level of about 23 kilos per capita.
11. A long time ago our ancestors were accustomed to *tnigenica* environment.
12. Visiting a modern supermarket a person takes a risk of direct contacting with different kinds of *lergensal*.
13. Nistrohenol are universally spread in the air, water and food.

7. Read the conversation between an examiner and a student. Complete the student's responses using sentences A–H from below.

A. *But some antigens, for example, the components of ragweed or mites of worms provoke allergy in especially large masses of people.*

B. *Secondly, it can be accounted for by the deplorable state of antigenic and haptenic burden of a modern man.*

C. *The phenomenon was discovered in dogs after repeated injection of sea anemones' extract.*

D. *The manifestations of his disease resembled those we now call* ***systemic anaphylaxis.***

E. *Under certain conditions autoallergy is able to cause autoimmune diseases.*

F. *Because it incurs pathological sequels.*

G. *But nowadays 10 per cent of the population of the Earth suffer from allergic disorders.*

H. *Each process is manifested in hyperergic inflammation.*

Examiner: Now let's talk about allergy. What do you think this term mean?

Student: Well, this term designates a group of various typical immunopathological processes. **1** _____.

Examiner: Why is the result of any intensive or unusual immune reaction referred to as sensitization?

Student: 2. _____. For practitioners the term "allergy" is applicable to excessive or poorly regulated immune response causing obvious tissue injury.

Examiner: How do you call an allergic reaction directed against autoantigens?

Student: 3. _____. If an allergic reaction is directed against autoantigens, it is called autoallergy.

Examiner: When was allergy as an experimental phenomenon originally developed? Who did it?

Student: 4. _____. Allergy as an experimental phenomenon was originally developed in 1902 by French scientists Richet and Portier during their sea trip on the Mediterranean.

Examiner: Do you know of any clinical cases of allergy described long before the aforementioned research?

Student: 5. _____. I do: one of the Egyptian Pharaohs died having been stung by a bee.

Examiner: Was allergy as widespread at the beginning of the twentieth century as it is now?

Student: No, it wasn't. At the beginning of the twentieth century allergy was considered to be a rare anomaly of body constitution. 6. _____.

Examiner: Does allergy only result from the evil character of an allergen itself?

Student: No, it doesn't. It is necessary to emphasize that allergy does not only result from the evil character of an allergen itself. 7. _____.

Examiner: How can such growing occurrence of allergy in the population be explained?

Student: Firstly, it can be explained by the greater number of people genetically predisposed to allergy. 8. _____.

Examiner: And... Are you allergic to anything?

Student: Well, I'm allergic to some poplar seed tufts.

8. Work in pairs. Act out the dialogue from exercise 8 with your partner.

9. Read the task card below.

Describe the main peculiarities of allergy following the plan:
What are allergy and autoallergy?
When was allergy first discovered?
How can broad manifestation of allergy be explained?

10. Now, using the prompts from exercise 9, talk on the topic: ALLERGY.

11. Translate into English.

1. Аллергия (от греч. αλλον — иной, ερογον — действую) или так называемая гиперчувствительность — явление, в основе которого лежат типовые иммунопатологические реакции, развивающиеся в сенсибилизированном организме при контакте с антигеном, вызвавшим сенсибилизацию. Итогом такого контакта является иммунологическое повреждение, а его следствием — гиперергическое воспаление.
2. Антигены, провоцирующие аллергию, называются аллергенами.
3. Если аллергические реакции направлены на аутоантигены, говорят об аутоаллергических реакциях, которые в известных условиях способны привести к аутоиммунным заболеваниям.
4. Аллергию как экспериментальный феномен впервые описали Ш. Рише и П. Портье (1902) во время плавния по Средиземному морю на яхте Гримальди, князя Монако, в виде необычайно сильной реакции при повторном введении собакам экстрактов морских лилий.
5. Еще в работах Франсуа Мажанди и даже в древнеегипетских хрониках описывались клинические случаи аллергии.
6. В начале двадцатого столетия аллергия принципиально рассматривалась как редкое явление. Об этом свидетельствует и этимология самого термина, введенного в 1906 году австрийским педиатром К. фон Пирке для обозначения «иной», то есть не обычной, а исключительной реактивности.
7. В настоящее время аллергические заболевания перестали быть редкостью. ВОЗ считает, что до 10% взрослого населения планеты и до 30% детей страдает от тех или иных аллергических болезней.
8. Аллергия — проблема организма, который генетически предрасположен к необычной реакции на заурядный для многих других индивидов антигенный раздражитель, а не следствие "злобного нрава" самого антигена.
9. К некоторым антигенам, например, к амброзии, клещам или гельминтам аллергичны особенно большие группы людей.
10. По всей видимости, существенный процент генетически предрасположенных к аллергии людей всегда существовал в человеческих популяциях, однако раньше такие люди выявлялись редко.
11. В настоящее время многие люди имеют возможность проявить свою особую реактивность на протяжении жизни. Причиной столь широкого распространения аллергии является многократно возросшая антигенная и гаптенная нагрузка на современного человека. Прежде всего, это является следствием

экологических проблем. Например, в 70-е годы XX в. в Австралии потребление различных чужеродных для организма непищевых веществ (консервантов, актиоксидантов, красителей, поллютантов и лекарств) составляло 23 килограмма на одного человека в год.

12. По словам известного терапевта Е. М. Тареева, страсть глотать таблетки — такое же неотъемлемое свойство современного человека, как прямохождение.
13. Наши предки проводили всю свою жизнь в антигенно-привычном окружении, но современный человек, путешествуя, меняет свою антигенную среду. Любой супермаркет, расположенный на где-нибудь в Пеории, штат Иллинойс, предоставляет ее жителям возможность контакта с антигенами шести континентов и пяти океанов, включая воздушный.

Unit 2
IMMUNIZATION AND ALLERGY

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

abdominal typhus	брюшной тиф
anaphylaxis	анафилаксия
anti-rabies boost	прививка от бешенства
antiserum	иммунная сыворотка, антисыворотка
BCG vaccine	Вакцина БЦЖ (*живая ослабленная противотуберкулезная вакцина Кальмета — Герена на основе bacille de Calmette-Guérin*[1] — бацилл Кальметта-Герена)
cholera	холера
diphtheria	дифтерия
endemic	эндемический
gastrointestinal tract	желудочно-кишечный тракт
gangrenous	гангренозный
globulin	глобулин
hepatitis B	гепатит В
gut	кишка, кишечный тракт
incidence	заболеваемость, коэффициент заболеваемости
hypersensitivity disorders	аллергические заболевания

[1] Léon Charles Albert Calmette, French physician and bacteriologist, born July 12, 1863, Nice; died October 29, 1933, Paris.

Jean-Marie Camille Guérin, French veterinarian and immunologist, born December 22, 1872, Poitiers; died June 9, 1961, Paris.

iatrogenic	ятрогенный, причиненный медицинским вмешательством
immunization	1) иммунизация, предохранительные прививки, профилактические прививки; 2) вакцинация
immunopathology	иммунопатология
Japanese encephalitis	японский (комариный) энцефалит
measles	корь
meningitis	менингит
mumps	эпидемический паротит, свинка
parenteral	парэнтеральный (способ введения лекарств и нутриентов в кровь)
parenteric	сходный с тифо-паратифозной группой инфекционных заболеваний, но вызванный иными возбудителями
pertussis	коклюш
poliomyelitis	полиомиелит, атрофический острый спинальный детский паралич, эпидемический детский паралич
protozoan	вызванный простейшими, принадлежащий к простейшим (*лат.*: Protozoa)
rubella, *syn.*: German measles	краснуха, син.: коревая краснуха
serotherapy	серотерапия
serum	1) сыворотка; 2) сыворотка крови; 3) иммунная сыворотка; 4) серозная жидкость; серозный экссудат
smallpox	натуральная оспа
tetanic	столбнячный
tetanus	столбняк
toxoid	токсоид, анатоксин (обезвреженный бактериальный токсин)
tuberculosis	туберкулез
vaccination	вакцинация
viral infection	вирусная инфекция
yellow fever	желтая лихорадка
whooping cough	коклюш

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo.

After that read the text aloud, trying to imitate the intonation.

A. There is also another factor directly related to the increased incidence of allergy in the twentieth century. And this matter is much less spoken about. But if we omit the discussion of this factor here, it can be interpreted as lack of proper medical self-criticism. The generations worldwide, which grew up allergic or inclined to hypersensitivity disorders, are those that have been subjected to mass national programs of cohort immunization.

B. The authors of this text are staunch supporters of vaccination and serotherapy. Since E. Jenner[1] in 1796 proposed *active immunization*[2] against smallpox, this disease has been universally eradicated. The annals of history keep the name of the last smallpox patient infected and recovered in 1977 — Ali Maow Maalin, a hospital cook and WHO vaccination team volunteer from Somalia. The effective vaccines are routinely and effectively used in many WHO countries for the prevention of poliomyelitis, measles, mumps, rubella, whooping cough, diphtheria, tetanus, Haemophilus influenzae type b meningitis. In some countries there is obligatory immunization with BCG vaccine against tuberculosis. In endemic regions of the world there is also mass immunization for prevention of abdominal typhus, cholera, yellow fever, hepatitis B, Japanese encephalitis and some other infections. Everyone is aware of the saving role of anti-rabies boosts. The means of active immunization — vaccines and toxoids (e. g. tetanic one) and the means of *passive immunization*[3] — antisera and specific γ-globulins (anti-diphtheria, anti-tetanic, anti-gangrenous, anti-hepatitis etc.) — virtually saved millions of lives and changed the history of civilization. Their creators, beginning from the inventor of anti-diphtheria serum *E. von Behring*[4] are listed in Nobel annals. In early post-Soviet Russia, as a result of a non-professional campaign against vaccination organized by medically ignorant journalists, a lot of parents refused to accept anti-diphtheria boosting for their children, misinterpreting mandatory vaccination as "violation of human rights". As a result, big cities saw an immediate revival of diphtheria previously not observed by Russian doctors for many decades. Some developing countries regularly modernizing their armies but still not performing mass anti-tetanus vaccination, allow tetanus to plunder about 1 million victims annually. This can clearly demonstrate how catastrophic the results of infectious diseases could have been but for the great achievements of practical immunology — sera and vaccines. An anticipated breakthrough in the field is the elaboration of vac-

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[1] Edward Jenner, English physician, surgeon and naturalist, born May 17, 1749, Berkeley, Gloucestershire; died January 26, 1823, Berkeley, author of first vaccination method, first who has clinically described delayed hypersebsitivity reaction.

[2] Induction of immune defenses by the administration of vaccines or toxoids

[3] Provision of temporary protection by means of exogenously produced immune substances.

[4] Emil Adolf von Behring (15 March 1854 — 31 March 1917) was a German physician, military doctor and hygienist, who received the 1901 Nobel Prize in Physiology or Medicine.

cines against Protozoan diseases and viral infections without the use of infectious agents, based on *anti-idiotypic* principle.

C. Now, let us turn to "the dark side of the Moon". On the reverse of the coin world medicine could be awarded for the control over infections, one can read a sad word in bold letters: **"ALLERGY".**

D. In order to be effective, any vaccine must be accepted by the immune system seriously. Although vaccination does not provoke the disease it is aimed at, immunization with DTP, for example, must be an equivalent of diphtheria, tetanus and pertussis developing in one body at the same time. Quite a substantial challenge! A challenge of this kind gives the immune system a chance to display all its abilities, not excluding the hidden predisposition to allergy. Not only natural, but also iatrogenic methods of protection against diseases are imperfect. That is why immunization is a weapon for cautious use in experienced hands.

E. Besides vaccination, during the last 80 years there has been rapid progress in the medical application of different antigenic preparations, injected via the parenteral[1] route (blood and blood products, parenteral nutrition, peptide drugs etc.). Avoiding the gastrointestinal tract, they are not inactivated in the gut and carry antigenic epitopes.

F. There has been a wave of allergies accrued in parallel with these medical changes. It is quite characteristic that both anaphylaxis and immune complex reactions in clinical conditions were first described as adverse effects of serotherapy in children treated with anti-diphtheric sera (C. von Pirquet, B. Schick[2], 1902). Therefore, one practical conclusion drawn from this is the necessity to be very meticulous about observing all restrictions, precautions and counterindications which are recommended by the Advisory Committee on Immunization Practices and other authorities concerned. "Boosting for boosting" is not the correct way of prophylaxis.

G. To meet these recommendations, medical doctors need broad knowledge in Immunopathology. It is essential to know the great variance of autoimmune and allergic disorders, and it is for this purpose that we have included in our Pathophysiology textbooks not only the general description of allergic mechanisms but also multiple concrete examples of immunopathological disorders in various organs and systems.

3. Look through the text to find out what topic each paragraph is concerned with.

The necessity to avoid dander or making mistakes at immunization	**A**
The necessity of doctors' training in immunopathology	**B**
The correct way of prophylaxis	**C**

[1] Compare with the meaning of the different, but sounding closely term *"parenteric"*: parenteric fever, parenteric infection

[2] Béla Schick (July 16, 1877 — December 6, 1967), was a Hungarian (after 1923 — American) pediatrician. He is the founder of the Schick allergotest and one who first described serum sickness in humans.

Application of different parenterial antigenic preparations	**D**
Mass national immunization programs	**E**
Allergy as a sad word	**F**
The history of immunization	**G**

4. Complete each sentence with the correct ending A–J.

A endemic regions
B anti-tetanus vaccination
C anti-rabies boosts
D active immunization
E human rights
F antigenic preparations
G allergic disorders
H effective vaccines
I experienced hands
J anti-diphtheria serum

1. It was E. Jenner who suggested _____.
2. In many WHO countries medical workers use some _____.
3. There is mass immunization for the prevention of abdominal typhus, cholera, yellow fever, hepatitis B, Japanese encephalitis in _____.
4. Everyone is aware of the saving role of _____.
5. E. von Behring was the creator of _____.
6. In early post-Soviet Russia a lot of parents refused to accept anti-diphtheria boosting for their children, misinterpreting this mandatory vaccination as "violation of _____".
7. Some developing countries regularly modernize their armies but they do not perform mass _____.
8. Immunization is a weapon for cautious use in _____.
9. During the last 80 years there has been rapid progress in the medical application of different _____.
10. It is essential to understand the great variance of autoimmune and _____.

5. Unjumble these jumbled words.

1 teroheraspy
2 timumnizaion
3 maslplox
4 saccinev
5 denmice
6 bsotos.
7 lviationo
8 reas
9 ciarogenit

10 pretparasion
11 recutiopnas
12 ropyplaxish
13 toimamunue

6. Use the unjumbled words from exercise 5 to complete the sentences.

1. Many doctors are strong supporters of vaccination and *teroheraspy*.
2. In 1796 E. Jenner proposed active *timumnizaion* against smallpox.
3. A hospital cook from Somalia Ali Maow Maalin is known as the last *maslplox* patient recovered in 1977.
4. Effective *saccinev* are routinely used in many WHO countries.
5. In *denmice* regions of the world there is also mass immunization for

the prevention of some infections.

6. Everyone is aware of the saving role of anti-rabies *bsotos*.
7. In early post-Soviet Russia a lot of parents misinterpreted mandatory vaccinations for their children as "*lviationo* of human rights".
8. The results of infectious could have been catastrophic but for the great achievements of practical immunology — *reas* and vaccines.
9. Not only natural, but also *ciarogenit* methods of defense against diseases are imperfect.
10. Besides vaccination, during the last 80 years there has been rapid progress in the medical application of different antigenic *pretparasion*.
11. It is necessary to be very meticulous about observing all limitations, *recutiopnas* and counterindications which are recommended by the Advisory Committee on Immunization Practices.
12. "Boosting for boosting" is not the correct way of *ropyplaxish*.
13. It is essential to know the great variance of *toimamunue* and allergic disorders.

7. Read the conversation between an examiner and a student. Complete the student's responses using sentences A–H from below.

A. As a result, big cities saw an immediate revival of diphtheria previously not observed by Russian doctors for decades.

B. Medical specialists must be very meticulous about observing all limitations, precautions and counterindications which are recommended by the Advisory Committee on Immunization Practices and other authorities concerned.

C. The annals of history keep the name of a hospital cook from Somalia Ali Maow Maalin, who was the last smallpox patient recovered in 1977.

D. At the same time it must give the immune system a chance to display all its abilities, not excluding the hidden predisposition to allergy.

E. In some countries there is obligatory immunization with BCG vaccine against tuberculosis.

F. It is essential for them to know a great variance of autoimmune and allergic disorders as well as multiple concrete immunopathological disorders in various organs and systems.

G. It happened in 1796.

H. The means of passive immunization are antisera and specific γ-globulins.

Examiner: And now let's talk about the problems of control over infections and allergy. Could you remind me of the name of the person who introduced active immunization?

Student: Well, it was Edward Jenner. **1.** _____.

Examiner: Right. And how effective it was?

Student: Smallpox was universally eradicated. **2.** _____.

Examiner: What diseases are vaccines used against?

Student: They are used against such diseases as poliomyelitis, measles, mumps, rubella, whooping cough, diphtheria, tetanus.

3. _____. In endemic regions of the world there is also mass immunization for the prevention of abdominal typhus, cholera, yellow fever, hepatitis B, Japanese encephalitis and some other infections.

Examiner: What are the means of active and passive immunization?

Student: The means of active immunization are vaccines and toxoids. **4.** _____.

Examiner: What do you know about the effect of one well-known non-professional campaign against vaccination?

Student: *In early post-Soviet Russia a lot of parents refused to accept* anti- diphtheria boosting for their children, misinterpreting mandatory vaccination as "violation of human rights". **5.** _____.

Examiner: How do you think a good vaccine must be accepted by the immune system?

Student: Vaccination must not provoke the disease. **6.** _____.

Examiner: What antigenic preparations are used besides vaccination?

Student: Besides vaccination different antigenic preparations such as blood

and blood products, parenteral nutrients, peptide drugs are used. Being injected via the parenteral route they avoid the gastrointestinal tract and are not inactivated in the gut.

Examiner: What do you think the correct way of prophylaxis is?

Student: "Boosting for boosting" is not the correct way of prophylaxis. 7. _____.

Examiner: What do doctors need to meet these recommendations?

Student: To meet these recommendations, doctors need broad knowledge in immunopathology. 8. _____.

8. Work in pairs. Act out the dialogue from exercise 8 with your partner.

9. Read the task card below.

Describe the main peculiarities of immunization following the plan:

Why are vaccination and serotherapy so important?
Why should immunization programmmes be carried out cautiously?
What knowledge in immunopathology is indispensible for a doctor?

10. Now, using the prompts from exercise 9, talk on the topic: IMMUNIZATION AND ALLERGY.

11. Translate into English.

1. Существует определенный фактор, непосредственно связанный с увеличением заболеваемости аллергией в XX столетии и не упомянуть об этом факторе было бы несамокритично.
2. Аллергичными и наклонными к иммунным заболеваниям выросли те поколения, которые прошли через программы массовой иммунизации.
3. Авторы данного текста являются убежденными сторонниками применения вакцин и сывороток.
4. В результате применения активной иммунизации против натуральной оспы после ее разработки Эдвардом Дженнером в 1796 году эта болезнь была совершенно искоренена.
5. История сохранила имя последнего человека, переболевшего натуральной оспой, больничного повара из Сомали — Али Маув Маалина.
6. Эффективные вакцины существуют и рутинно применяются во многих странах для предупреждения полиомиелита, кори, свинки, краснухи, коклюша, дифтерии, столбняка.
7. В некоторых странах является обязательной массовая иммунизация против туберкулеза, а в эндемичных районах мира — против брюшного тифа, холеры, желтой лихорадки, гепатита В.
8. Известна спасительная роль вакцинации против бешенства при риске заражения.

9. Для того чтобы быть эффективной, вакцина должна восприниматься иммунной системой всерьез.
10. Поэтому, несмотря на то, что вакцина не вызывает болезни, против которой она прививает, для иммунной системы введение, например, АКДС означает необходимость вести себя так, как если бы в организме одновременно протекали коклюш, дифтерия и столбняк.
11. Антигенный вызов такого рода позволяет иммунной системе проявить свои потенции, включая и скрытую предрасположенность к аллергии.
12. Несовершенны не только естественные, но и ятрогенные способы защиты от болезней. Поэтому иммунизацией следует пользоваться осторожно.
13. Помимо вакцинации за последние 80 лет многократно расширилась практика парэнтерального введения в лечебно-профилактических целях различных антигенных препаратов, не проходящих инактивацию и обезличивание в желудочно-кишечном тракте.
14. Анафилаксия и иммунокомплексная аллергия в клинике впервые были описаны в 1902 году Клеменсом фон Пирке и Белой Шиком у детей, получавших курсы серотерапии против дифтерии.
15. Необходимо детально соблюдать те прививочные противопоказания, ограничения и медотводы, которые вводятся прививочными инструкциями для соответствующих контингентов прививаемых. Иными словами «прививки ради прививок» недопустимы.
16. Врач должен быть широко эрудирован в вопросах иммунопатологических болезней.

Module 7
ANTIGENS

Unit 1
ANTIGENS AND EPITOPES

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

alien	чужой, чужеродный, чуждый
antigen	антиген
biopolymer	биополимер
cholesterol	холестерин
dextran	декстран (водорастворимый высокомолекулярный полимер глюкозы)
epitope	эпитоп
ester	эфир
ficoll	фиколл
flagellate	жгутиковый
gelatin	желатин(а)
hapten	гаптен
heterocyclic substance	гетероциклическое соединение
immunoglobulin	иммуноглобулин, антитело (*устар.*: *иммунный глобулин*)
levan	леван
lymphocytic antigen-specific receptor	лимфоцитарный антигенспецифический рецептор
polynucleotide	полинуклеотид
polypeptide	полипептид
tertiary	третичный

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

The living organism persists in the unfriendly environment in spite of mechanical, physical, chemical and biological pathogenic factors (pathogens) tending to harm it from outside and a lot of unfavorable tendencies injuring it from the inside. The only way for an organism to survive and flourish is to have all its systems permanently strained in order to achieve a balance between all kinds of damaging influences and the efforts of the organism to compensate, thus maintaining its homeostasis.

The role of the immune system in achieving this goal is unrivalled, because the immune system sustains the homeostasis on the sub-cellular level, being uniquely equipped with a mechanism capable of producing a host of special tools, each of them specifically complementary to one out of a billion various biopolymer molecular types. These molecules are called *antigens* and the specific tools of the immune system are referred to as *antigen-specific lymphocyte receptors*.

A substance is antigenic provided it has a three-dimensional structure capable of key-lock complementary principle interactions. To put it in other words, it must be able to recognize and to be recognized. Paul Ehrlich[1] formulated this principle in a proverbial form: "*Corpora non agunt nisi fixata.*" — "No bodies can act unless they bind (recognize)". Antigenicity requires some degree of structural complexity and spatial rigidity.

Even high molecular weight substances can be non-antigenic if they have a flat structure (cholesterol). Gelatin is a high molecular weight protein devoid of tertiary structure, and, consequently, it is non-antigenic or is regarded as very weak antigen (unable to form "key-hole", according the example given by W. Boyd)[2].

Thus, antigens are biopolymeric molecules capable of inciting immune response in the organism by specifically binding to the lymphocytic antigenic receptors.

Antigens are a wide variety of natural and biosynthetic molecules ranging in size between 1 and 10 kD (proteins, polysaccharides, complex esters and heterocyclic substances, nucleic acids, polynucleotides and their complexes, including lipid-containing ones).

The definition of an antigen has changed drastically during the last 60 years. Initially, it was regarded simply as a synonym of a "pathogen"; later, immunologists re-interpreted this term as a direct comple-

[1] Paul Ehrlich, German physician, pathologist and biochemist, founder of Immunology, born March 14, 1854, Strehlen, Schlesien; died August 20, 1915, Bad Homburg vor der Höhe.Awared Nobel Prize in Medicine or Physiology in 1908.

[2] William Clouser Boyd (March 4, 1903 — February 19, 1983) was an American immunochemist and anthropologist, who with his wife Lyle, during the 1930s, made a worldwide survey of the distribution of blood types. Also he discovered lectins in plants. Boyd published textbook of immunology, considered to be one of the best for his time. Together with his wife he wrote also science fiction novels under the nickname "Boyd Ellanbee".

mentary cast of an antibody, and, finally, it came to be defined as mentioned above. Hence, antigens are polymeric molecules that can trigger specific immune responses when introduced into the organism. Interestingly, only polar molecules are highly immunogenic.

At the same time, the wide-spread popular interpretation of the word "antigen" as an "alien protein" must be rejected from the very beginning of the present discussion because of being totally obsolete. Moreover, many low molecular weight substances (for instance, iodine) may generate unique three-dimensional complexes with biopolymeric carriers, which provoke immune responses. Such low molecular weight substances are called *haptens*.

The immunochemical treatment of an antigen is possible only if it has covalent chemical bonds. From this standpoint, cells, bacteria and multimolecular complexes possess many antigenic specificities. The minimal structural unit of an antigen, recognizable for antibodies and lymphocyte antigenic receptors, is known as an *epitope* (immunodominant region).

As a rule, epitopes are located in the most mobile and configurable parts of quite rigid antigen structures. The theory of clonal selection insists, that each clone of lymphocytes is genetically programmed to recognize only one epitope by means of an antigen-specific receptor on its surface, so that these cells display fine antigen specificity. It is believed that billions of different epitopes may exist, consequently, the equivalent number of lymphoid cell clones with their unique receptors is required for the recognition of this multitude.

Any immune response requires a certain densitiy of epitopes: it disappears if their compactness is either too high or too low. Single epitopes are non-immunogenic, since for an immune response to occur the recognizing molecules must be cross-linked. Due to this overwhelming majority of immune responses the organism needs cooperation of several specialized cells (*B-lymphocytes, T-lymphocytes, antigen-presenting cells*).

Some antigens include panels of numerous identical epitopes, among them: bacterial *lipopolysaccharides*, polymeric protein of Flagellates-flagellin, dextran, levan and other bacterial capsule polysaccharides, ficoll and polypeptides composed of D-aminoacids. They are hardly digestible for antigen-presenting cells, but able to cross-link the recognizing lymphocyte receptors directly. This group of antigens provokes polyclonal B-cell activation and immunoglobulin production without T-cell cooperation. Many individual antigens have several or even numerous different epitopes and provoke simultaneous immune response of various cellular clones (both antibody secretion and clonal expansion of cellular effectors).

3. Do the following statements agree with the information given in the text?

Write

TRUE *if the statement agrees with the information*

FALSE *if the statement contradicts the information*
NOT GIVEN *if there is no such information*

1. Any living organism has to sustain a variety of pathogenic factors to survive.
2. Pathogens tend to destroy an organism from inside.
3. Homeostasis is maintained by the immune system.
4. A substance is antigenic as it has the ability to recognize and to be recognized within immune system.
5. A substance is antigenic provided it has a three-dimensional structure.
6. High molecular weight substances are non-antigenic if they have a flat structure.
7. In accordance with classical views gelatin is either a non-antigenic or very weak antigen.
8. Antigens stimulate immune response via binding to the lymphocytic antigen-specific receptors.
9. Antigens are a limited variety of natural and biosynthetic molecules.
10. Haptens are high molecular weight substances.
11. The immunochemical treatment of an antigen is possible only if it has divalent chemical bonds.
12. An epitope is the minimal structural antigenic unit.
13. It is believed that billions of different cell clones are required for the recognition of billions of different epitopes.
14. Each clone of lymphocytes is genetically programmed to recognize several epitopes.
15. B-lymphocytes and T-lymphocytes can cooperate with single epitopes.

4. Correct the false statements from exercise 3.

5. Make questions based on the statements in exercise 3.

6. Work in pairs. In turns ask and answer the questions you have made.

7. Look at the dialogue below. Complete the student's answers with the sentences from the box.

A. *The equivalent number of lymphoid cell clones with their unique receptors is required for the recognition of their multitude.*

B. *Due to this overwhelming majority of immune responses the organism needs cooperation of several specialized cells such as B-lymphocytes, T-lymphocytes, antigen-presenting cells.*

C. *These molecules are called antigens and the specific tools of the immune system are antigen-specific lymphocyte receptors.*

D. *As a rule, epitopes are located in the most mobile and configurable parts of quite rigid antigen structures.*

E. *Antigens are capable of inciting immune response in the organism by specifically binding to the lymphocytic antigen-specific receptors.*

F. *For example, gelatin is a high molecular weight protein devoid of tertiary structure, and, consequently, it is either a non-antigenic or very weak antigen.*

G. *It maintains genetic homeostasis so the organism can survive*

H. *That may be recognized by the receptors of T- and B-lymphocytes and may generate immune response*

Examiner: Let's talk about antigens. What is the main function of the immune system?

Student: Well. **1.** _____.

Examiner: How can the immune system fight against different pathogens?

Student: By means of a mechanism capable of producing a host of special tools, which bind pathogens neutralizing them. Each tool is specifically complementary to one out of a billion various biopolymer molecular types. **2.** _____.

Examiner: What structural substance can be called antigenic?

Student: A substance is antigenic provided it has a three-dimensional structure that may be recognized by the receptors of T- and B-lymphocytes and may generate immune response. . **3.** _____.

Examiner: Can high molecular weight substances be non-antigenic?

Student: Yes, they can provided they have a flat structure. **4.** _____.

Examiner: So, what are antigens?

Student: They are a wide variety of natural and biosynthetic molecules ranging in size between 1 and 10 kD. **5.** _____.

Examiner: What is known as epitopes? And where are they located?

Student: This is the minimal structural unit of an antigen recognizable for antibodies and lymphocyte antigenic receptors. **6.** _____.

Examiner: How many clones of lymphocytes are programmed to epitopes?

Student: It is believed that billions of different epitopes may exist. **7.** _____.

Examiner: Can a single epitope initiate an immune response?

Student: Single epitopes are non-immunogenic. That is why for an immune response to occur the recognizing molecules must be cross-linked. **8.** _____.

8. Work in pairs. Act out the dialogue form exercise 7 with your partner.

9. Read the task card below.

Describe the main peculiarities of antigens and epitopes following the plan:

What are antigens?
What is the role of antigens for the immune system?
What are epitopes?

10. Now, using the prompts from exercise 9, talk on the topic: ANTIGENS AND EPITOPES.

11. Translate into English.

1. Живой организм существует в условиях недружественной окружающей среды: при воздействии извне механических, физических, химических и биологических патогенных факторов (патогенов) и неблагоприятных процессах внутри организма.
2. Единственным путем для поддержания жизни организма является поддержание его систем в рабочем состоянии, компенсация неблагоприятных воздействий, сохранение гомеостаза.
3. Иммунная система обеспечивает осуществление гомеостаза на субклеточном уровне с помощью уникального механизма, ответственного за создание инструментов, каждый из которых предназначен для распознавания одного вида биополимерных молекул из огромного множества.
4. Данные молекулы носят название антигенов, а специальные инструменты иммунной системы носят название антител и антигенраспознающих рецепторов лимфоцитов.
5. Субстанция является антигенной, если имеет трехмерную структуру, способную к комплементарным взаимодействиям. Другими словами, она должна распознаваться и распознавать. Антигенность требует определенной структурной сложности и пространственной ригидности.
6. Даже при большом молекулярном весе молекула может не быть антигенной, если ее структура плоскостная (холестерин).
7. Молекула желатины — высокомолекулярного белка — неантигенна, поскольку лишена третичной структуры, что не дает ей возможности участвовать во взаимодействиях типа «ключ — замочная скважина».
8. Антигены — биополимерные молекулы, вызывающие иммунный ответ организма, способные специфическим образом взаимодействовать с рецепторами лимфоцитов.
9. Антигенами называют биополимерные природные и синтетические молекулы размером от 1 до 10 кД (белки, липиды, по-

лисахариды, сложные эфиры, нуклеиновые кислоты, полинуклеотиды и их комплексы).

10. Индивидуальными антигенами считаются только такие биополимеры, все составляющие которых объединены ковалентными связями.
11. Эпитоп — минимальная единица структуры антигена, распознаваемая антителами или лимфоцитарными антигенными рецепторами.
12. Как правило, эпитоп — это наиболее подвижная и конфигурабельная часть относительно ригидной антигенной структуры.
13. Согласно клонально-селекционной теории, клон лимфоцитов отвечает на один вид таких детерминант.
14. Иммунный ответ требует определенной плотности эпитопов: при очень высокой или очень низкой плотности он исчезает.

Unit 2
CLASSIFICATION OF ANTIGENS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

autoantigen	аутоантиген
conformation determinant	конформационная (создаваемая третичной структурой биополимера) детерминанта
fibril	фибрилла
heteroantigen	гетероантиген
immunologic tolerance	иммунологическая толерантность
lipopolysaccharide, LPS	липополисахарид (соединение липида и углевода), ЛПС
sequential determinant	секвенциальная детерминанта
trauma	травма

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

All antigens may be formally classified into exogenous and endogenous, depending on their origin. This distinction is relative. Endogenous and exogenous antigens do not differ in shape, nature, size, smell or taste. The response of the immune system depends very little on whether the antigen is endogenous or exogenous, e. g., exogenous antigens of transfused donor red blood cells are tolerated, whereas the endogenous antigens arising on the recipients' own older erythrocytes incite a cold-blooded murder of these battered veterans by the immune system.

Of much greater significance for the classification of antigens is their division into *autoantigens* (self) and *heteroantigens* (non-self). Endoantigens are not necessarily autoantigens. Autoantigens are biopolymers characteristic of normal unimpaired young cells; although these molecules are superabundant, they normally produce no other response but *immunologic tolerance*. The markers of senescence expressed on aged cells and pathologic *neoantigens* induced by viral infection, trauma, and oncogenesis, do not belong to autoantigens. All these antigens are included in a self modified group and treated by the immune system as "aliens".

On the other hand, heteroantigens are molecules that are not found in normal unimpaired young cells; on the contrary, they appear in sick, older, injured or tumor cells, as well as in alien ones, or invade the organism from outside. These heteroantigens are not normally tolerated but rather produce full-fledged immune responses. No doubt, heteroantigens may be of exogenous as well as endogenous origin (neoantigens and self modified ones). At the same time, there are both endogenous autoantigens (which is only natural), and exogenous (cross-reactive) ones.

According so called "*danger hypothesis*", all antigens principally can be recognized by immune cells, regardless of their self or alien character. But, alien ones produce more intensive response. It occur because their presence is accompanied by presense of "obligatory pathogenic complexes", which are viral or prokaryotic biopolymers, absent in eukaryotic organism (in infectious aggression) and also few molecules normally presenting almost exclusively within living cells, like ATP and DNA. The extracellular abundance of such molecules is automatically interpreted as sign of aggression either cell death, which facilitates the longer and more productive cooperation of cells in immune response.

It takes all sorts of antigens to make the immune system work properly, for they are the major object it manipulates.

In fact, an antigen is not only a material molecule, but also an informational unit or signal. As any kind of new information, antigens need ***presentation*** for broad recognition and acknowledgement.

According to the classic concept, antigens display epitopes of different types. *Conformation determinants* represent forms created by tertiary structure of biopolymers. For example, in protein antigens it may be represented by amino acid residues located far from each other in primary polypeptide chains, but put together in spatial three-dimensional

globes of fibrils. As a rule, they protrude out of the antigen molecule. *Sequential determinants* are all but simple and short linear sequences produced by primary structure, for example short peptides of neighboring 9–11 amino acids in protein molecules.

In the course of immune responses to extracellular antigens (during antigen processing after *phagocytosis)*, conformation epitopes are lost, but sequential ones are exposed and presented by antigen-presenting cells on their surface as a rind or panel. In the course of immune responses for intracellular antigens, sequential determinants are created in translation and presented by antigen-presenting cells before a complete set of conformation determinants is acquired in post-translational modifications. Cytophysiological mechanisms of antigen presentation for *exocellular* antigens (both self and non-self), taken from outside of the antigen-presenting cells, and for endocellular antigens (both self and alien), produced within such cells, are not identical, for example, they involve MHC proteins of different classes and may be displayed in different types of hypersensitivity.

3. Do the following statements agree with the information given in the text?

Write

TRUE	*if the statement agrees with the information*
FALSE	*if the statement contradicts the information*
NOT GIVEN	*if there is no such information*

1. All antigens may be informally classified into exogenous and endogenous depending on their origin.
2. Endoantigens are always autoantigens.
3. Unimpaired young cells normally produce immunologic tolerance.
4. The markers of senescence belong to autoantigens.
5. Heteroantigens sometimes invade an organism from without.
6. Heteroantigens can be tolerated.
7. According to the classical concept, antigens display epitopes of different types.
8. Conformation determinants represent forms created by linear structure of biopolymers.
9. Sequential determinants are all but simple and short tertiary sequences.
10. In the course of immune responses to extracellular antigens (during antigen processing after phagocytosis), conformation epitopes are lost, but sequential ones are exposed and presented by antigen-presenting cells on their surface as a rind or panel.

4. Correct the false statements from exercise 3.

5. Make questions based on the statements in exercise 3.

6. Work in pairs. In turn ask and answer the questions you have made.

7. Look at the dialogue below. Complete the student's answers with the sentences from the box.

A. *On the contrary, they appear in sick, older, injured or tumor cells, as well as in alien ones, or invade the organism from without.*

B. *Antigens display conformation and sequential epitopes.*

C. *Conformation epitopes are lost, but sequential ones are exposed and presented by antigen-presenting cells on their surface as a rind or panel.*

D. *All antigens can be divided into exogenous and endogenous.*

E. *Of much greater significance for the classification of antigens is their division into autoantigens and heteroantigens.*

F. *In fact, an antigen is not only a material molecule, but also an informational unit or signal.*

G. *They are found in normal unimpaired young cells.*

Examiner: In what way are all antigens classified depending on their origin?

Student: **1.** _____.
Examiner: May antigens be divided in another way?
Student: Well. **2.** _____.
Examiner: Where are autoantigens found?
Student: **3.** _____.
Examiner: Where are heteroantigens found?
Student: They are *not* found in normal unimpaired young cells. **4.** _____.
Examiner: Should we interpret an antigen only as a material molecule?
Student: No, we shouldn't. **5.** _____.
Examiner: What types of epitopes do antigens display?
Student: **6.** _____.
Examiner: What happens with these epitopes in the course of immune responses to extracellular antigens?
Student: **7.** _____.

8. Work in pairs. Act out the dialogue form exercise 7 with your partner.

9. Read the task card below.

Describe the main peculiarities of the classification of antigens following the plan:

How can antigens be classified?
What are exogenous and endogenous antigens?
What are autoantigens and heteroantigens?

10. Now, using the prompts from exercise 9, talk on the topic: CLASSIFICATION OF ANTIGENS.

11. Translate into English.

1. Различают антигены экзогенного и эндогенного происхождения.
2. Далеко не все эндогенные антигены являются аутоантигенами.
3. Нормальные неповрежденные молодые клетки характеризуются наличием аутоантигенов и, несмотря на то, что данные молекулы имеются в изобилии, они обычно не вызывают иного ответа кроме иммунологической толерантности.
4. Маркеры старения, выраженные в старых клетках, и неоантигены, экспрессия которых вызвана вирусной инфекцией, травмой, и онкогенезом, не относятся к аутоантигенам. Данные антигены входят в особую группу и воспринимаются иммунной системой как «чужие».
5. Гетероантигены не обнаружены в нормальных неповрежденных молодых клетках, и, напротив, они появляются в больных, старых, поврежденных опухолевых, «чужеродных» клетках либо проникают в организм извне.
6. Организм обычно не толерантен к гетероантигенам, напротив, они вызывают полноценный иммунный ответ. Гетероантигены могут быть как экзогенными, так и эндогенными по происхождению.
7. По классическим представлениям, конформационные детерминанты антигена представляют форму, созданную третичной структурой биополимера. Секвенциальные детерминанты представляют собой простые линейные последовательности, созданные первичной структурой биополимера.
8. В ходе иммунного ответа при процессировании антигенов после их фагоцитоза конформационные детерминанты утрачиваются, зато секвенциальные обнажаются и презентируются фагоцитирующей клеткой на поверхности в виде панели.

Medicine Through Biographies

Read the text below, pausing after every paragraph to find the main fact (or idea) around which it is centered.

Then, read the text again and prepare a list of questions covering the main periods of Karl Otto Landsteiner's life. Using this list, discuss the facts with a partner/partners.

Then, make a detailed list of questions covering Landsteiner's main scientific achievements. Discuss the importance of his discoveries with a partner/partners.

Write a summary of Landsteiner's most significant contribution to the medical practice.

Discuss Landsteiner's role and contribution to various fields of medical theory and practice.

Notice the terms, given in bold script. Make a list of them. Without looking into the text give their definitions.

Karl Otto Landsteiner (1868–1943)

(after Ole-Daniel Enersen)

Karl Landsteiner was one of the first scientists to study the chemical and physical processes, underlying ***immunity*** and is reckoned as the founder of ***serology*** and the doctrine of ***antigens***. He discovered that there are several ***types of human blood*** and established the ABO and Rh systems based on ***haemagglutination***. This blood typing made ***blood transfusion*** a routine medical practice. In 1930 he was awarded the Nobel Prize in Physiology or Medicine "for his discovery of human blood groups".

Landsteiner was the only child of Dr. Leopold Landsteiner, a famous journalist and the founder of the Vienna daily newspaper "PRESSE", and Fanny Hess Landsteiner. The family lived in Baden bei Wien, an upper-middle-class suburb of Vienna. Karl was four years old when his father *suffered a massive heart attack* and died. He was placed under the guardianship of a family friend, but remained close to his mother.

In 1885, when he was seventeen, Landsteiner *passed entrance examinations* and *was admitted to* the Medical school at the University of Vienna. He took a year off from school at the age of twenty for his obligatory military service. When he was twenty-one, Landsteiner and his mother *converted* from Judaism to Catholicism and Karl was *christened* Karl Otto Landsteiner. Besides the medical *curriculum* Landsteiner had a thorough grounding in the basic sciences, particularly chemistry.

His first scientific work was in chemistry, which he began to study in Ernst Ludwig's (1842–1915) laboratory in Vienna while still a student. He also *attended* lectures by Ludwig Mauthner (1840–1894). He *received his medical degree* from the University of Vienna in 1891, aged 23. For the next three years he continued his chemical studies in Germany and Switzerland. In 1892 he ***synthesized glycolaldehyde*** at Würzburg with Emil Fischer (1852–1919), later to become the *winner of the Nobel prize in* Chemistry for 1902. During 1892–1893 he learned the chemistry of benzene derivatives from Eugen von Bamberger (1858–1921) in Munich, and in Zurich in 1893–1894 he studied organic chemistry under Arthur Rudolf Hantzsch (1857–1935) and Roland Scholl (1865–1945). With these famous scientists he published many journal articles.

Medicine, however, remained Landsteiner's chief interest. For a short time after receiving his M.D. he worked with Otto Kahler (1849–1893) at the Second Medical University Clinic in Vienna; and from 1894 to 1895 he served with Eduard Albert (1841–1900) at the First Surgical University Clinic. During 1896–1897 Landsteiner was an assistant to Max von Gruber (1853–1927) in the newly established Institute of Hygiene at the University of Vienna. At this Institute, then intensely interested in experimenting on bacterial agglutination, his interest was awakened in ***serology***, ***immunology*** and the ***nature of antibodies***, and he published his first papers on ***agglutination*** and immunology.

In 1897 Landsteiner moved to Vienna's Institute of Pathology, where he was hired to *perform autopsies*. Here his new teacher was Anton Weichselbaum (1845–1920) whose assistant he was from 1897 to 1908. Under Weichselbaum's *supervision,* Landsteiner conducted 3,639 postmortem examinations and also began his work on serology. In a footnote to his only paper published in 1900, Landsteiner reported his perhaps most important discovery: the *interagglutination* occurring between serum and blood cells of different humans. He suggested that this was not pathological, as was the prevailing belief at the time, but rather a physiological phenomenon due to the unique nature of the individual's blood.

In an article the following year he described a simple technique of agglutination and was able to show that there were at least three major types of human blood that vary according to the kinds of sugar-containing substances, known as antigens, attached to the plasma membrane of the red blood cells. Based on this he divided human blood into three groups: A, B, and C (later 0). Two of his co-workers, the clinicians Alfred von Decastello-Rechtwehr, (born 1872) and Adriano Sturli (1873–1964), examined a lot of additional individuals and in 1902 found the fourth blood group, later named AB. For his experiments, Landsteiner *drew blood* from himself and *collected blood samples* from his colleagues, doctors Jakob Erdheim (1874–1937), Oskar Stoerk (1870–1926) and Adriano Sturli. In 1903 Landsteiner became a Privatdozent at the University of Vienna.

The discovery of blood groups made possible *safe transfusion* of blood from one person to another. The first successful transfusions were achieved in 1907 by Dr. Reuben Ottenberg (1882–1959) of Mount Sinai Hospital, New York. In 1913, Richard Lewisohn (1875–1961), a surgeon at the Mount Sinai Hospital, re-discovered and inculcated in practice the fact, first experimentally shown in 1910 by two Russian military physicians Vadim Alexandrovich Jurevich (1873–1962) and Nicolay Konstantinovich Rosenberg (1876-1933), that adding citrates to blood *prevented* it *from* coagulation. This was the basis for his introduction of the indirect technique of blood transfusion, and the last prerequisite for the *establishment* of the modern blood bank, since blood could now be preserved for two- to three-week periods under refrigeration. The first transfusion on a human using Lewisohn's citrate method was performed by Dr. Howard Lilienthal (1860–1946). During World War I, transfusion of *compatible blood* was first performed on a large scale and saved many lives. Operations on the heart, lungs, and circulatory system, previously impracticable because of enormous blood loss involved, were now feasible, as were *complete blood exchange* in cases, for example, of intoxication or severe jaundice of the newborn. Later the Russian physician, philosopher and pathophysiologist Alexander Alexandrovich Bogdanov (1873–1928), influenced by Landsteiner's doctrine, broadly implemented these methods and founded in the USSR the first blood transfusion research institute and first all-national blood transfusion service in the world.

Besides ***transfusiology***, Landsteiner's work added an important chapter to the development of ***forensic medicine***, providing admissible evidence in *paternity suits* and *murder trials*. In 1902 Landsteiner presented a lecture, together with Max Richter (1867–1932) of the Vienna University Institute of Forensic Medicine, in which the two scientists reported on a new method of *typing dried blood stains* to help solve crimes in which blood stains are left at the scene. At first Landsteiner did not guess that blood types were *inheritable*, for Mendel's (Johann Gregor Mendel, 1822–1884) *laws of heredity*, first presented in 1865, had passed into oblivion. In 1900 the laws were rediscovered by the German botanist and geneticist Carl Erich Correns (1863–1933), the Austrian botanist Erich Tschermak von Seysenegg (1871–1962), and the Dutch botanist and geneticist Hugo de Vries (1848–1935). Ten years later Emil Freiherr von Dungern (born 1867) and Ludwig Hirschfeld (born 1884) postulated the first hypothesis for the inheritance of blood groups. This theory was corrected in 1924 by the German mathematician Felix Bernstein (1878–1956) and was finally established. Since then, denying paternity has never been the same.

During this period Landsteiner also worked on characterizing and evaluating the physiological meaning of cold agglutinations in human blood serum. In 1904 he and the Austrian internist Julius Donath (1870–1950) described a test for the diagnosis of ***paroxysmal cold haemoglobinuria***. This test is now known as Donath-Landsteiner phenomenon or test. In fact, the disorder for which it was suggested was

the first autoallergic disease of humans ever medically described. Landsteiner also made a great contribution into *infectious immunology*. In 1905–1906 Landsteiner and Ernest Finger (1856–1939), then chief of the Clinic for Venereal Diseases and Dermatology in Vienna, were successful in infecting monkeys with syphilis, making possible experimentation with Spirochaeta pallida, the causal agent of the disease. In collaboration with the neurologist Otto Pötzl (1877–1962) and the serologist Rudolf Müller (1877–1934), they elucidated the previously unknown mechanism occurring in the Wassermann reaction (August Paul von Wassermann, German bacteriologist, 1866–1925). In 1906, Landsteiner and Viktor Mucha, a scientist from the Chemical Institute at Finger's clinic, developed the technique of dark-field microscopy to identify and study the microorganisms that cause syphilis.

In 1908 he left the Pathology Institute to accept a position as director of the laboratories of the Royal-Imperial Wilhelmina Hospital in Vienna. This position afforded him the opportunity to become his own chief, thus allowing him to delegate routine pathological work to an assistant and continue his personal research along the lines of his own choosing. Landsteiner now concerned himself extensively with ***poliomyelitis***. One day in 1908 the body of a young polio victim was brought in for autopsy. After the autopsy, Landsteiner injected a homogenate of its brain and spinal cord into the abdominal cavity of two rabbits, two guinea pigs, two mice and two monkeys (Cynocephalus hamadryas and Macacus rhesus). The rabbits, guinea pigs and mice remained well, but on the sixth day following the injections the Cynocephalus became sick and showed signs of paralysis similar to those in poliomyelitis patients. Two days later he died. The histological appearance of the central nervous system of the monkey also was similar to that of the humans who had died of the disease. Since he could not prove the presence of bacteria in the spinal cord of the child who had died, Landsteiner postulated the existence of a virus: "The supposition is hence near, that a so-called invisible virus or a virus belonging to the class of protozoa causes the disease". The disease could then be transmitted from monkey to monkey and eventually it was possible to transfer a strain of the virus to the rat and to the mouse, which could be used in sufficient numbers ***to establish the existence and virulence of the polio virus***. The experiment was conducted with his assistant Erwin Popper. In volume 2, 1909 of the Zeitschrift für Immunitätsforschung, they reported that they might have found an 'invisible virus' that had caused the polio epidemics. Today the World Health Organisation (WHO) still credits Landsteiner and Popper as having found the poliovirus with this experiment.

Landsteiner spent the years 1909–1912 in Paris collaborating with the Romanian bacteriologist, Constantin Levaditi (1874–1953) of the Pasteur Institute. Working together, the two were able to trace poliomyelitis to a virus, describe the manner of its transmission, time its incubation phase, and show how it could be neutralized in the laboratory when mixed with the serum of a *convalescing patient*. In 1911 Landsteiner was promoted to Professor Extraordinarius. Landsteiner's work

schedule allowed little time for social activity. He was serving at a war hospital in 1916 when, at the age of 48, he married Leopoldine Helene Wlasto. Their only child, a son, was born the following year and was christened Ernst Karl on April 8, 1917. After the World War I, Austria was in chaos, with hyperinflation and extreme shortages of food and fuel, conditions similar to those experienced by Germany a few years later. Landsteiner accepted a position as chief dissector in a small Catholic hospital, R. K. Ziekenhuis, in The Hague, Holland. There, from 1919 to 1922, he performed routine laboratory tests on urine and blood. It wasn't much more than a technician's job, but it protected him and his family at least against hunger and cold.

In 1921, utilizing investigations dating as far back as 1904, Landsteiner began working on ***the concept of haptens***, small molecular weight chemicals such as fats or sugars that determine the specificity of antigen-antibody reactions when combined with a protein "carrier." This discovery was influential in the development of immunology and launched Landsteiner into a study of the phenomenon of ***allergic reactions***. However, the working conditions in The Hague were no better than in Vienna. He therefore accepted an offer from the Board of Directors of the Rockefeller Institute in New York, and went to the United States in 1922.

Throughout the 1920s Landsteiner worked on the problems of ***immunity and allergy***. In 1927 he discovered new blood groups: M, N and P, *refining* the work he had begun 20 years before. Soon after Landsteiner and his collaborator, Philip Levine, published the work and, in 1927, the types began to be used in paternity suits. Karl Landsteiner became a United States citizen in 1929. He continued broad research in the field of ***applied immunology*** and related areas. Landsteiner and Clara Nigg were successful in 1930–1932 in culturing Rickettsia prowazekii, the *causative agent* of dotty typhus, on living media. In 1934, together with W. Strutton and Merril W. Chase (born 1905) he described a blood antigen found only in Negroes, which today is called ***the Hunter-Henshaw system***.

In this fruitful late period of his biography Landsteiner was appreciated by world *scientific community* and achieved many *awards* and *honorary* positions. Among them: 1926 Hans Aronson Foundation Prize, 1929 Presidentship of the American Association of Immunologists, 1930 Nobel Prize for Physiology or Medicine and Paul Ehrlich Medal for Chemistry. He became also Chevalier of the French Legion of Honour and was awarded the Dutch Red Cross Medal. *Honorary doctorates* from the Universities of Chicago (1927) and Cambridge (1934), Université Libre de Bruxelles (1934), and Harvard University (1936) were adequate to his academic merits. Landsteiner officially retired in 1939, at the age of seventy-one, but went on working.

Soon, in 1940 he and his pupils Alexander Wiener (1907–1976) and Philip Levine (1900–1987) made an important discovery, the last in the long creative life of Karl Otto Landsteiner. They described a new antigen in the human blood, ***the Rh factor***, so called after Rhesus monkeys in which the factor was first discovered. Levine was the first to see the

connection between this Rh-factor and pathological jaundice occurring in some newborn children, thus explaining its pathogenesis. The disorder now known as Rh-conflict results from the isoimmune cytotoxic allergic reaction of the Rh-negative mother against the red blood cells of Rh-positive fetus.

Landsteiner was described as a modest, self critical, rather timid man known for his wide reading. He was not only a medical scientist, but also an excellent pianist. Over the years he became ever more committed to his work, and in his later years he had little time for friendships. Upon his death, tributes were published around the world, but no mention of his passing away was published in his native Austria or Germany until the defeat of Nazism. Motherland of Austria finally also commemorated him. Before the introduction of the Euro currency, Landsteiner was the last to have his portrait on a one thousand Austrian shillings banknote, sharing such honour with the eminent physicist Erwin Schredinger (1887–1961).

IDEAS FOR WRITING A COMPOSITION

! General tips

— **Always** practice timed writing to improve your speed.
— **Never** write answers below the minimum length.

For each writing task you should use the following outline:
Paragraph 1: Introduction: Introduce the topic
Paragraph 2: Main Part: Give the main ideas and details
Paragraph 3: Overview: Summarize the information

Module 1
Topic 1. Reactivity. **Write at least 70 words.**
Topic 2. Resistance. **Write at least 70 words.**
Topic 3. Ontogenesis. **Write at least 70 words.**

Module 2
Topic 4. Hereditary diseases. **Write at least 70 words.**
Topic 5. Sex chromatin. **Write at least 70 words.**
Topic 6. SRY-gene. **Write at least 70 words.**

Module 3
Topic 7. The microcirculatory disorders. **Write at least 70 words.**
Topic 8. Microcirculatory disorders modeling. **Write at least 70 words.**

Module 4
Topic 9. Thrombosis. **Write at least 70 words.**
Topic 10. Platelet granules. **Write at least 70 words.**
Topic 11. Embolism. **Write at least 70 words.**

Module 5

Topic 12. The system of thermoregulation. Fever. **Write at least 70 words.**

Topic 13. Fever of adults. Experimental Fever. Hyperthermia. **Write at least 70 words.**

Module 6

Topic 14. Allergy. **Write at least 70 words.**

Topic 15. Immunization and allergy. **Write at least 70 words.**

Module 7

Topic 16. Antigens and epitopes. **Write at least 70 words.**

Topic 17. The classification of antigens. **Write at least 70 words.**

Topic 18. Medicine via Biographies. **Write at least 70 words about life and deeds of some outstanding physician or medical scientist, mentioned above or not mentioned here, but known to you from other literature.**

VOCABULARY

a

abdominal typhus	брюшной тиф
accelerator	акселератор, ускоритель; катализатор
acidosis	ацидоз
acinus	ацинус (1. легочный мешочек; 2. железистый ацинус)
active (arterial) hyperemia	активное (артериальное) полнокровие
adenine	аденин
adenylate-cyclase receptor subunit	рецепторная субъединица аденилат-циклазы
adrenal medulla	мозговое вещество надпочечников
aerial	воздушный; передаваемый воздушно-капельным путем (напр. об инфекции), переносимый воздушным путем (напр. о поллютанте)
agglutination	1) агглютинация, склеивание; 2) образование групп или скоплений (микроорганизмов)
ailment	недомогание, нездоровье
aldosterone	альдостерон
alien	чужой, чужеродный, чуждый
allergen	аллерген
allergy	аллергия
anaphylaxis	анафилаксия (реагин-опосредованная аллергическая реакция немедленного типа) В американских текстах может использоваться как синоним слова «аллергия», что семантически неточно
anastomosis	анастомоз, соустье
angiogenesis	ангиогенез

antegrade embolism	ортоградная эмболия
anterior hypothalamus	передний гипоталамус
anticoagulation	антикоагуляция, противосвертывание
antigen	антиген
antigenic	антигенный
antioxidant	антиоксидант, ингибитор окисления
antipyretic	жаропонижающее средство, антипиретик; жаропонижающий, противолихорадочный
anti-rabies boost	прививка от бешенства
antiserum	иммунная сыворотка, антисыворотка
antithrombin	антитромбин (название группы веществ — антагонистов тромбина)
aorta	аорта
arachidonic acid	арахидоновая кислота
arginine-vasopressin	агринин-вазопрессин
arterial blood pressure	артериальное кровяное давление
arteriole	артериола
assay	1) анализ; пробирный анализ; биологическое испытание, тест, проба; количественный анализ; производить анализ; испытывать; 2) проба; образец для анализа
autoallergy	аутоаллергия
autoantigen	аутоантиген
axial	аксиальный, осевой
axis	ось (в эндокринологии — регуляторный контур, включающий гипоталамо-гипофизарное звено и периферическую эндокринную железу с их прямыми и обратными связями)

b

bacterium (*pl.:* bacteria)	бактерия (бактерии)
Barr body	тельце Барра
basal metabolic rate (BMR)	основной обмен
BCG vaccine	Вакцина БЦЖ (*живая ослабленная противотуберкулезная вакцина Кальмета — Герена на основе bacille de Calmette-Guérin* — бацилл Кальметта-Герена)
Bernard-Soulier syndrome	синдром Бернара-Сулье
biogenous amine	биогенный амин
biopolymer	биополимер
blood flow	кровоток
blood vessel	кровеносный сосуд
bradycardia	брадикардия
brown fat	бурая жировая клетчатка

buccal	1) буккальный, относящийся к щеке; щечный; трансбуккальный (о методе введения лекарственного средства); 2) внутриротовой
bundle	пучок

c

calcium	кальций
capillary bed	капиллярное русло
carbonuria	карбонурия (повышение содержания общего углерода в моче)
cardiomyocyte	кардиомиоцит
cardiovascular	сердечно-сосудистый
catalytic subunit	каталитическая субъединица
catatoxic reactions	кататоксические реакции
catecholamine	катехоламин
cerebral	церебральный, мозговой
cerebral ventricles	желудочки головного мозга
cholera	холера
cholesterol	холестерин
chromosome	хромосома
circulatory disorders	нарушения ковообращения
clot	сгусток
coagulation	коагуляция, коагулирование; свертывание
coagulation factor	фактор свертывания крови
coin stacks	"монетные столбики" (неустойчивые агрегаты эритроцитов)
collagen	коллаген
collapse	коллапс (острая недостаточность кровообращения без его централизации)
complementary	комплементарный (обладающий однозначным структурным соответствием), распознающий, дополнительный
conformation determinant	конформационная (создаваемая третичной структурой биополимера) детерминанта
confrontation	противостояние, сопротивление
congenital	врожденный
coronary	венечный, коронарный (о сосудах сердца)
cortisol	кортизол, гидрокортизон
counter mutation	контрмутация
critical	критический, угрожающий
cytokine	цитокин
cytosine-guanine	цитозин-гуаниновый

d

dehydration	обезвоживание, дегидратация
dextran	декстран (водорастворимый высокомолекулярный полимер глюкозы)
dihydro-testosterone	дигидротестостерон
dipeptide	дипептид

diphtheria	дифтерия
diploid	диплоидный, с двойным набором хромосом
diuresis	диурез (1. процесс образования и выделения мочи; 2. количество мочи, выводимое из организма за определенное время)
diver's disease	кессонная болезнь

e

elasticity	эластичность
embolus (*pl.*: emboli)	эмбол (циркулирующий в крови субстрат, способный вызвать закупорку кровеносного сосуда)
embryo	зародыш, эмбрион (на протяжении первых восьми недель внутриутробного развития)
embryogenesis	эмбриональное развитие, зародышевое развитие, эмбриогенез
endemic	эндемический
endocrine	эндокринный
endogenous	эндогенный, возникший внутри организма
endogenous/vascular ischemia	эндогенная/сосудистая ишемия
endotheliocyte	эндотелиоцит, эндотелиальная клетка
endothelium	эндотелий
endotoxin	эндотоксин
enzyme	фермент, энзим
epinephrine	адреналин, эпинефрин
epithelium (*pl.*: epithelia)	эпителий, эпителиальная ткань
epitope	эпитоп
ester	эфир
estrogene	эстроген (1. женский половой гормон; 2. эстрогенное средство)
exogenous	экзогенный, вызываемый внешними причинами
extremity	1) край, конец; 2) конечность

f

febrile	лихорадочный, фебрильный, имеющий отношение к лихорадке
fetal hemoglobin (Hb)	фетальный гемоглобин
fever	жар, лихорадочное состояние; лихорадка; вызывать жар; бросать в жар, лихорадить
fibril	фибрилла
fibrin	фибрин
fibrinogen	фибриноген
fibrinogenesis	фибриногенез
fibronectin	фибронектин
ficoll	фиколл
filament	филамент, нить

flagellate	жгутиковый
forceps	1) щипцы; зажим; 2) пинцет
frog	лягушка
fungus (*pl.*: fungi)	1) гриб; грибок; плесень; 2) грибовидное разрастание (ткани)

g

gamete	гамета, зрелая половая клетка
gangrenous	гангренозный
gastrointestinal tract	желудочно-кишечный тракт
gelatin	желатин(а)
globulin	глобулин
glycoprotein	гликопротеид, гликопротеин
gut	кишка, кишечный тракт

h

hapten	гаптен (неполный антиген)
heart cavity	полость сердца
heat loss (heat emission)	теплоотдача
hematogenous/obturation ischemia	гематогенная/обтурационная ишемия
hemophilia	гемофилия
hemorrhage	1) кровотечение; 2) кровоизлияние
hemostasis	остановка кровотечения, гемостаз
heparin	гепарин (естественный антикоагулянт; содержится во многих тканях и тучных клетках)
hepatitis B	гепатит В
heteroantigen	гетероантиген
heterocyclic substance	гетероциклическое соединение
hibernation	гибернация, зимняя спячка
histamine	гистамин
hydrolase	гидролаза
hypercapnia	гиперкапния (повышенное содержание двуокиси углерода в крови)
hyperergic inflammation	гиперергическое воспаление
hypersensitivity	гиперчувствительность, повышенная чувствительность; *в американских текстах используется вместо термина «аллергия»*
hypersensitivity disorders	аллергические заболевания
hyperthermia	гипертермия, перегревание организма
hypophysis (syn.: pituitary)	гипофиз, питуитарная железа, нижний мозговой придаток
hypothalamus	гипоталамус, гипоталамическая область, подбугорная область, подбугорье
hypothermia	гипотермия (1. пониженная температура тела; 2. искусственное понижение температуры тела с лечебными целями)
hypoxia	гипоксия, кислородное голодание, кислородная недостаточность

i

iatrogenic	ятрогенный, причиненный медицинским вмешательством
idiosyncrasy	идиосинкразия, индивидуальная непереносимость, *исторически применялось к любой, а в настоящее время — лишь к неиммунологической повышенная чувствительности,*
immune	иммунный, невосприимчивый, обладающий иммунитетом
immunity	иммунитет, невосприимчивость
immunization	1) иммунизация, предохранительные прививки, профилактические прививки; 2) вакцинация
immunoglobulin	иммуноглобулин, антитело (*устар.*: иммунный глобулин)
immunologic tolerance	иммунологическая толерантность
immunopathology	иммунопатология
incidence	заболеваемость, коэффициент заболеваемости
infarction	инфаркт
inflammation	воспаление
inflow	приток
inhibiting	ингибирующий
intact	1) интактный, здоровый; незараженный, неосложненный; неповрежденный; 2) исходный (напр. о штамме микроорганизмов)
integument	покровы тела
interdigital	межпальцевой
intestinal	интестинальный, относящийся к кишечнику, кишечный
intravascular	внутрисосудистый, интраваскулярный
intravenous injection	внутривенная инъекция
ionised	ионизированный
Japanese encephalitis	японский (комариный) энцефалит
karyotype	кариотип (совокупность особенностей числа и формы хромосом клетки)
ketone	кетон
lactate	лактат
lateral	латеральный, боковой; удаленный от средней линии
leukocyte	лейкоцит, белое кровяное тельце
levan	леван
life span	продолжительность (срок) жизни, время жизни
ligature	1) лигатура; 2) перевязка, лигирование (напр. сосуда), наложение лигатуры; перевязывать, лигировать (напр. сосуд), накладывать лигатуру
lipopolysaccharide, LPS	липополисахарид (соединение липида и углевода), ЛПС
lumen	просвет (сосуда)

luteinising hormone, LH	лютеинизирующий гормон, лютропин, пролан Б (*устар.*), ЛГ
lymph	лимфа
lymphocytic antigen-specific receptor	лимфоцитарный антигенспецифический рецептор
lysosome	лизосома
marginal	маргинальный, краевой
measles	корь
medial	медиальный, относящийся к середине или центру
membrane	мембрана
Mendelian laws (distribution)	законы (распределение) Менделя
meningitis	менингит
mesentery	брыжейка
metabolite	метаболит (продукт метаболизма)
metarteriole	прекапиллярная артериола, метартериола, прекапилляр
methylation	метилирование
microcirculation	микроциркуляция
microcirculation vessels	микроциркуляторные сосуды
microcirculatory bed	микроциркуляторное русло
microcirculatory disorders	нарушения микроциркуляторного кровообращения
mitotic	митотический
mixed/combined hyperemia	смешанная гиперемия
monogenous hereditary diseases	моногенные наследственные заболевания
mononuclear	мононуклеар, одноядерная клетка; мононуклеарный лейкоцит, одноядерный
mumps	эпидемический паротит, свинка

n

necrosis	некроз, омертвение
nephron	нефрон (структурно-функциональная единица почки)
nervous	нервный (о системе); относящийся к нерву
neurogenous	нейрогенный; неврогенный
neuroparalytic	нейропаралитический (механизм); нервно-паралитический (о ядах)
neurotonic	1) нейротонический (механизм); 2) улучшающий тонус нервной системы (о лекарственном средстве)
neurovascular	нейроваскулярный, невроваскулярный, нервно-сосудистый

neutrophilic	нейтрофильный, характеризующийся наличием нейтрофилов
nitrophenol	нитрофенол
nucleotide	нуклеотид
nucleus (*pl.*: nuclei)	ядро, *мн.*: ядра

o

occlusion	1) окклюзия, смыкание челюстей, прикус; 2) обтурация; окклюзия; закупорка
oncogenesis	онкогенез, бластомогенез
ontogenetic	онтогенетический
opioid peptides	опиоидные пептиды
organogenesis	органогенез (формирование органов в пренатальный период)
oscillator	осциллятор, колебательный контур
outflow	отток

p

parenchyma	паренхима (совокупность основных функционирующих элементов внутреннего органа)
parenteral	парэнтеральный (способ введения лекарств и нутриентов в кровь)
parenteric	сходный с тифо-паратифозной группой инфекционных заболеваний, но вызванный иными возбудителями
passive (venous) hyperemia, congestion	пассивное (венозное) полнокровие, венозный застой
pathogenic	патогенный, болезнетворный
pediatrician	педиатр
peptide mediator	пептидный медиатор
pertussis	коклюш
phagocyte	фагоцит, фагоцитирующая клетка; фагоцитировать
phagocytosis	фагоцитоз
phoshpolipid	фосфолипид
phosphatidylcholine	фосфатидилхолин
phosphofructokynase	фосфофруктокиназа
plasma capillary	плазматический капилляр
plasmatic	плазменный, плазматический (кровоток), относящийся к плазме
platelet	тромбоцит, кровяная пластинка, бляшка Биццоцеро
platelet-leukocyte activator of aggregation or (P-selectin)	тромбоцитарно-лейкоцитарный активатор агрегации (пи-селектин)

poliomyelitis	полиомиелит, атрофический острый спинальный детский паралич, эпидемический детский паралич
pollutant	загрязняющее вещество, загрязнитель, поллютант; примеси (в воздухе)
polygenous hereditary diseases	полигенные наследственные заболевания
polymerase	полимераза
polymorphonuclear	полиморфно-ядерный (лейкоцит)
polynucleotide	полинуклеотид
polypeptide	полипептид
polysaccharide	полисахарид
practitioner	практический врач, практикующий врач, врач-практик
precapillary	предкапилляр; предкапиллярный
prostaglandin	простагландин
prothrombin	фактор II (свертывающей системы крови), протромбин; протромбиновый
protozoan	вызванный простейшими, принадлежащий к простейшим (*лат.*: Protozoa)
pulmonary	легочный, пульмональный
pyrogen	1) пироген, пирогенное вещество; 2) фактор, вызывающий лихорадку
Pyrogenalum	Пирогенал (*препарат*)

r

reactivity	реактивность
rear hypothalamus	задний гипоталамус
receptor	рецептор
regeneration	регенерация, восстановление
resistance	резистентность
respiratory	респираторный, дыхательный
respiratory coefficient	дыхательный коэффициент
retraction	1) втяжение; западение; 2) ретракция; стягивание, сокращение
retrograde embolism	ретроградный эмболизм
rheologic	реологический, связанный с текучестью
rouleaux	(*pl.* от rouleau, франц.) «монетные столбики» (неустойчивые агрегаты эритроцитов)
rubella, *syn.*: German measles	краснуха, син.: коревая краснуха

s

sanogenic	саногенный, оздоравливающий
sciatic nerve	седалищный нерв

secretory	секреторный (относящийся к процессу или продуктам секреции)
sensitization	сенсибилизация; в аллергологии — первичный иммунный ответ
sepsis	сепсис, общая гнойная инфекция
septum (*pl.*: septa)	перегородка
sequential determinant	секвенциальная детерминанта
serotherapy	серотерапия
serotonin	серотонин
serum	1) сыворотка; 2) сыворотка крови; 3) иммунная сыворотка; 4) серозная жидкость; серозный экссудат
session	время, отведенное какой-либо деятельности или занятию
sex chromatine	половой хроматин
Shereshevsky–Turner's syndrome	синдром Шерешевского–Тернера
shunt	шунт; (обходной) анастомоз; шунтировать
sludge of erythrocytes	сладж эритроцитов
smallpox	натуральная оспа
somatic	соматический
somatostatin	соматостатин
sphincter	сфинктер, жом (круговая мышца, сжимающая полый орган)
sphyngomyelin	сфингомиелин
spinal cord	спинной мозг
SRY-gene	ген SRY
stadium decrementi	стадия понижения температуры
stadium fastigii or acme	стадия стояния температуры (акме)
stadium incrementi	стадия повышения температуры
stasis	стаз (остановка кровотока или тока других жидкостей организма)
stratum (pl. strata)	слой
stroma	строма (соединительнотканная опорная структура органа или опухоли)
subendothelial	субэндотелиальный
substrate	1) субстрат; 2) подложка (в культуре клеток)
supravital	суправитальный, прижизненный
swab	тампон
sympathetic	симпатический (относящийся к симпатической нервной системе)
syntoxic reactions	синтоксические реакции
systemic	системный, общий, относящийся ко всему организму

t

terminal	конечная часть, окончание; конец; конечный, терминальный
tertiary	третичный
testosterone	тестостерон

tetanic	столбнячный
tetanus	столбняк
thermoregulation	терморегуляция
thermosensor	термосенсор
thoroughfare, preferencial channel	главный капилляр, магистральный канал
thrombasthenia	тромбастения, тромбастеническая гемофилия
thrombin	тромбин, фибрин-фермент (*устар.*)
thrombocytopenia	тромбо(цито)пения (пониженное содержание тромбоцитов в крови)
thromboembolism	тромбоэмболия (эмболия вследствие тромбоза)
thrombomodulin	тромбомодулин
thromboplastin	тромбопластин
thrombosis	тромбоз
thrombospondin	тромбоспондин
thrombus (*pl.*: trombi)	тромб (*мн.*: тромбы)
thyroid	щитовидная железа; щитовидный, тироидный
tissue-type/compression ischemia	тканевая/компрессионная ишемия
tolerance	переносимость, толерантность
tongue	язык
tongue root	корень языка
total nitrogen	общий азот (биохимический показатель)
toxoid	токсоид, анатоксин (обезвреженный бактериальный токсин)
translucent	полупрозрачный
trauma	травма
trigger	триггер, спусковой крючок, запускающий элемент
trisomy	трисомия (наличие в клетке лишней хромосомы)
trophic	1) трофический (связанный с митозами и/или синтезом ДНК); 2) алиментарный (связанный с питанием)
true	истинный
tuberculosis	туберкулез
tumor	опухоль
turpentine	скипидар

U

urinary	мочевой, мочеиспускательный
urtica	волдырь

V

vaccination	вакцинация
vagal	вагусный (относящийся к блуждающему нерву)
vasoconstriction	вазоконстрикция, сужение кровеносных сосудов

vasoconstrictor	сосудосуживающий фактор, вазоконстриктор
vasodilatation	вазодилатация, расширение кровеносных сосудов
vasopathia (*pl.*: vasopathiae)	вазопатия
venule	венула
vessel	сосуд; полость трубчатого органа
viral infection	вирусная инфекция
viscosity	вязкость
von Willebrand disease	болезнь фон Виллебранда

W

whooping cough	коклюш

Y

yellow fever	желтая лихорадка

Z

Zahn thrombus (*syn.*: platelet plug)	тромб Цаана, белый тромб
zymosan	зимозан

Part II

INTRODUCTION INTO INTERNAL MEDICINE

(for the students of 6th and 7th terms

Preface

This part of the book is addressed to the third and forth year medical students. It deals with introduction into internal medicine. The target of this book is to develop the English language relevant to professional purposes. We hope this book will help medical students in mastering their English. International medical students may use it also for better learning of Internal Medicine, while studying at Russian medical schools.

The authors

PHYSICAL METHODS OF DIAGNOSIS IN CARDIOLOGY

Module 1

PATIENT'S INTERWIEW

Unit 1.1

INTRODUCTION. THE SIGNIFICANCE OF ANAMESIS

1. **Without looking into the text listen to the recording.**

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Despite all its achivements, modern medicine is unable to establish the correct diagnosis without taking into account the results of a conversation between a doctor and a patient, because there is no instrumental or laboratory screening method which can entirely substitute for it. Moreover, it is not possible to use in full the results of instrumental and laboratory probing without analyzing the concrete results of direct interaction with a patient.

The first contact with a patient must begin with an interview conducted by the doctor. Carring out an interview with a patient is a matter of great proficiency. The goal of such an interview is to obtain maximum information concerning the patient as a result of a relatively short

dialogue with a viewer to construct mentally the appropriate diagnostic suppositions. The beginners of medical practice sometimes fail to reach this goal even at the end of a prolonged intercourse. But such kind of experience is vital for a doctor because it lays the foundation of "first glance diagnosis" art for further more adept practice.

Establishing a correct diagnosis after the very first interview requires that a doctor should poswsess a special gift and an immense practical experience. If a doctor interviews a patient for the first time, the most essential thing is to provoke the patient's frankness in order to complete the comprehension of the patient's personality, motivation to recuperate, and desire to be treated. It helps to understand if the patient is alarmed with his/her status, which plays an important part in the treatment as a psychotherapeutic effect.

Asking questions about the patient's complaints is considered to be an intricate and difficult part of the medical interview and, at the same time, the most crucial one. In the majority of cases the key role in distinguishing a disease belongs to patient's complaints. For freshmen of medical practice it is vital to beware of hasty questioning and examination, which may often cause a wrong guess as regards the disease. The questions a doctor puts forth must be absolutely clear for a patient of any intellectual and educational level. It is important in order to avoid misleading and ambiguous answers. A doctor can not be satisfied with any patient's single answer, but only earnestly clear, competent and unequivocal ones will do. It is malpractice for a doctor to include any descriptions, not named by a patient himself, into the list of symptoms. Otherwise, the doctor might put into a patient's head the symptoms of a disease mistakenly suggested at first glance.

The doctor should never accept desirable things instead of real ones. During the initial steps of diagnosis, establishing a mistaken tentative diagnosis is not at all impossible. This situation is not dangerous. The real danger is in involuntarily putting this false point of a doctor's view into a patient's head. As a result of this you may plant in the mind of a patient the suspicions of symptoms which he does not actually have. The hypnotizing style of interviewing should be carefully avoided.

2. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *Why is it impossible in modern medicine to make a correct diagnosis without taking into account the results of the conversation between a doctor and a patient?*
2. *Is it possible to use in full scale the results of instrumental and laboratory probing without analyzing the results of direct interaction with a patient?*

3. How should the first contact with a patient begin?
4. What is the goal of the interview?

3. Read the task card below.

Describe the main peculiarities of interview following the plan:
What is an interview?
How should an interview be carried out?
Why is an interview important?

4. Now, using the prompts from exercise 4, talk on the topic: INTERVIEW.

Unit 1.2
INTERVIEWING A PATIENT

1. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

The questioning is an important and difficult diagnostic method. To acquire the necessary skill, a doctor must strive to improve his/her proficiency and master her/his abilities. You may auscultate your patient by means of a stethoscope, but it is more important to listen to him/her with ears and calm the patient down. The lower the doctor's proficiency level, the less he/she tends to speak to a patient. Patients often give different descriptions of the symptoms of their diseases to different doctors. Much depends on the doctor' experience: the greater the doctor's experience, the more information he/she will obtain through questioning patients.

The great Russian physician Sergey Petrovich Botkin[1] used to underline the role of "a certain dominant idea" governing the collection of anamnestic facts.

Usually, a patient starts with complaints, which are the most important in his opinion, but objectively they are not always the principal

[1] Botkin (also spelled: Botkine), Sergey Petrovich, 1832–1889, Russian internist and pathophysiologist, discoverer of infectious hepatitis (Botkine's disease).

ones. Therefore it would be incorrect to passively listen to the patient's complaints. Active questioning is of extremely necessary importance. Thus, an interview must consist of two parts: a passive one attending to a patient's spontaneous narration, and an active one — skillful professional questioning by the doctor.

The most significant part of the interview is the anamnesis. Anamnesis (from Greek бнбмнзуйу — remembrance) is a patient's recollections concerning the onset and development of the disease, as the patient himself interprets it. There is a proverb, that proper anamnesis is half of the correct diagnosis. Anamnesis consists of an unconstrained patient's description of the onset and development of the disease and a targeted doctor's questioning. During the course of the anamnesis, the doctor appreciates the meaningful and secondary information in the patient's story, at the same time observing the patient's neuropsychological status. Hence, the questioning is a process which is planned and organized by the physician. It usually takes more time than other methods of diagnostics. But a doctor should never save time at the expense of anamnesis.

Not infrequently, during subsequent clinical curation, the necessity of recourse to the anamnesis arises several times to add something, to reach proper exactness or re-estimate data.

The common therapeutic mistakes in the anamnesis collection are the following:

— Underestimation of the character of the complaints;

— Misjudgment of the difference between the onset and exacerbation of the illness;

— Neglecting of epidemiological, pharmacological and allergologic anamnesis;

— Muscalculation of living conditions and family-sexual problems.

— Turning to effective investigation after the clarification of complaints and "anamnesis morbi et vitae" detailed collection, the doctor proceeds to objective investigations

2. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What is questioning?*
2. *What does a doctor have to do to master his/her skills of questioning?*
3. *How is a doctor's proficiency level connected with the ability to speak with a patient?*
4. *Do you agree that the greater a doctor's experience, the more information may be obtained by means of questioning a patient?*

Dialogue 1. Interview at the doctor's office

3. Without looking into the dialogue listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the dialogue silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the dialogue aloud, trying to imitate the intonation.

Doctor. Good morning. What's your name, please? How old are you? What do you do?

Patient. My name is Natalia. I am 54 years old. I am a mathematics teacher.

D. What's troubling you?

P. Well, doctor, I have had a bad headache for the whole month and I have palpitation, especially at the end of my workday, and I can't stand stuffy and smoky premises.

D. What do you do to get rid of your headache?

P. My mother advised me to take some pills, like No-Spa, but I have not noticed any effect. And Dibazolum[1] is helpful for ache relief. Citramonum[2] is also helpful but I have heartburn from it. It seems to contain Aspirin, and I have peptic ulcer, so I can't bear Aspirin. However, in winter the ulcer does not bother me.

D. Your profession is very difficult and nervous, isn't it?

P. Exactly. It was much easier to be a teacher about thirty years ago. And now the requirements are getting more complicated and the amount of the school material is getting bigger. It drives me mad. The children are overloaded too, and everyone gets nervous with stresses...

D. I can guess the reason of your complaints. It is, most likely, essential hypertension. It can be regarded as nearly occupational disease of teachers. It usually appears at the background of chronic stresses, and you have these stresses every day. I'd like to check your blood pressure...

Can you see it? You have increased blood pressure: 150 to 100 millimeters of mercury. And you have rapid pulse, which is 98 beats per minute.

P. Is this that bad, Doctor?

D. No, it isn't. Your have only recently developed your disease; therefore it will be easier to cope with it. Nowadays there are lots of medicines for hypertension, it is only necessary to choose the right one.

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[1] Russian commercial name. International name: Bendazolum.

[2] Russian commercial name. International name: tablets of Acetylsalicylic acid+Paracetamol+Caffeine+Ascorbic acid.

Each course of treatment is individual. With the help of modern medicines it is possible to normalize any high blood pressure. The initial stages of essential hypertension can be treated by rest and sedatives. Also it is necessary to decrease the consumption of salt. But the essential condition is the elimination of stressful situations. Winter vacation is coming. Can leave from school for about 7 or 10 days? You could go to a board and lodging health centre, for example.

P. We have a country house and I was planning to go there for some days to ski. In fact I used to do sports before. And what medicines do I have to take?

D. As your high blood pressure is accompanied with tachycardia, which is most likely connected with epinephrine, a hormone of stress, the best medications for you are those which suppress its influence on the vessels and the heart. They are called "beta-adrenoblockers". I will prescribe you Egilok-25[1]. It is the minimal doze. You will take 1 tablet in the morning daily. Have you suffered from chronic bronchitis or bronchial asthma?

P. No, I haven't. But, does it have todo with the essential hypertension?

D. Well, patients suffering from chronic bronchitis and bronchial asthma cannot bear beta-adrenoblockers properly, as it may cause breathlessness and bronchospasm. Here is the prescription for the Egilok. It will help you. I advise you to buy a tonometer to be able to control your arterial pressure by yourself. The price of a tonometer is reasonable nowadays.

P. Thank you, doctor. I feel relieved even now after I have talked to you. Good buy.

4. Translate into English.

I. На Вы жалуетесь?

Чем Вы снимаете головные боли?

У вас очень трудная, нервная профессия, не так ли?

Давайте измерим кровяное давление.

Оно повышено: 150/100 мм ртутного столба. Пульс учащенный — 98 ударов в минуту.

II. Это гипертоническая болезнь. Ее можно считать почти профессиональной болезнью учителей. Она обычно возникает на фоне хронических стрессов, а они у учителей почти ежедневные.

III. Гипертоническая болезнь возникла недавно, поэтому с ней будет легче справиться. В настоящее время существует масса лекарств от гипертонии, надо только подобрать нужное. Лечение подбирается индивидуальное. С помощью современных лекарств можно нормализовать любое повышенное давление крови. Начальные стадии гипертонической болезни можно лечить покоем и успокоительными средствами. И надо обязательно снизить потребление соли. Но необходимым условием является устранение стрессовых ситуаций.

[1] Hungarian commercial name. International name: Metoprololum.

IV. Повышение кровяного давления сопровождается сердцебиениями, что, вероятнее всего, связано с гормоном стресса — адреналином. В этом случае для лечения лучше всего подойдут препараты, подавляющие влияние адреналина на сосуды и сердце. Они называются бета-адреноблокаторы.

V. Пациенты с хроническим бронхитом и бронхиальной астмой бета-адреноблокаторы переносят плохо — у них появляется одышка, и может возникнуть спазм бронхов. Больному гипертонией рекомендуется приобрести тонометр, чтобы контролировать артериальное давление. Тонометры доступны по цене.

Unit 1.3
TYPES OF DIAGNOSIS AND METHODS OF PHYSICAL INVESTIGATION

1. **Without looking into the text listen to the recording.**

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

In diagnostic process a medical doctor creats mental model of a disease, explaining the observed manifestation and rules out other possible explanations.

In common practice three kinds of diagnosis are in use: provisional, main and final. They reflect three separate steps of the diagnostic process.

Provisional (or preliminary) diagnosis is established on the initial examination of a patient, at the beginning of questioning. At this point, the reliability of diagnosis is doubtful because the doctor still has not got suffitient information about the patient. Of course, one may refer to a long list of cases where even provisional diagnosis appeared to be correct and well established.

The main (clinical, or detailed) diagnosis arises as a result of the dynamic observation and investigation of a patient by means of the analysis and synthesis of numerous pathologic symptoms and the conclusion made due to the doctor's brainwork. Rules, accepted by world medicine, require the diagnosis be established during the first three days of curation.

The final (firm, conclusive) diagnosis is established by the doctor after repetitive examinations and on the basis of interpretation of the results at the final stage of observation or in connection with a patient's death (i. e. postmortem). In some cases the diagnosis may be established only as a result of prolonged observation. This diagnosis is called retrospective.

An interview of a patient gives only preliminary data to be used by a physician for a rational, optimally planned objective physical investigation.

The basic investigation methods are the following: examination, palpation, percussion, auscultation and checking of certain specific symptoms, depending on the particular disease.

The advances in medical science and health industry give the possibility to improve and refine available simple physical methods, by means of instrumental approaches, which, undoubtedly, improve the quality of diagnostics.

Yet, it is estimated that all modern achievements in cardiologic laboratory and instrumental diagnostics, although still in fashion, contribute only 10% of diagnostic value, while the old simplest physical methods, beginning with questioning and ending with auscultation, contribute 90% of diagnostic value.

As a rule, physical methods are more revealing than instrumental ones; the main thing is to apply them properly, which requires continuous, serious and prolonged training.

2. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What kinds of diagnosis are in use?*
2. *What do they reflect?*
3. *What is provisional or preliminary diagnosis established on?*
4. *How does it start?*

3. Read the task card below.

Describe the main peculiarities of interview following the plan:
How many kinds of diagnosis are distinguished?
What are they?
What methods are used at diagnostics?

4. Now, using the prompts from exercise 4, talk on the topic: INTERVIEW.

Module 2

CARDIOLOGICAL PATIENT'S COMPLAINTS AND HISTORY

Unit 2.1

EVALUATION OF PATIENT'S COMPLAINTS IN CARDIOLOGY: DYSPNEA

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

arterialization	артериализация (превращение венозной крови в артериальную)
atherosclerotic cardiosclerosis	атеросклеротический кардеосклероз
breathlessness (dyspnea)	одышка (диспноэ)
cardiomyopathy	кардиомиопатия
ciliary arrhythmia	мерцательная аритмия
claudication	хромота
dropsy	водянка (скопление жидкости в какой-либо полости тела)
dyspepsia	диспепсия (расстройство пищеварения)
erethism	эретизм (болезненное состояние возбуждения или раздражения)
expectoration	мокрота
extremities	конечности
fatiguability, fatigability	утомляемость
hypercapnic	дыхательный
hypertension	гипертензия, гипертония
hypoxemic	гипоксемический
mitral stenosis	митральный стеноз
myocardial infarction	инфаркт миокарда
nephritis	нефрит (воспаление почек)
palpitation	учащенное сердцебиение
pericarditis	перикардит (воспаление перикарда)
pulse intermission	перебои пульса
retrosternal	загрудинный

sputum	мокрота
subcostal	подреберный
valvular	вальвулярный, клапанный
vertigo	головокружение

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Recognition of cardiovascular diseases also starts with questioning of a patient. First of all, the character of complaints should be revealed.

The list of main cardiovascular patient's complaints includes:

— Breathlessness
— Cardiac and retrosternal pains
— Palpitations
— Pulse intermissions
— Occasional bloody expectoration
— Heavy feeling in the right subcostal area
— Dropsy
— Dyspepsia
— Undue fatigability, reduced work capacity
— Erethism, sleep disorders
— Not infrequently — headache and vertigo
— In some cardiovascular diseases — elevated temperature, pain in low extremities in and after walking (called: intermittent claudications), etc.

Breathlessness

Breathlessness (or dyspnea) — is breathing disorder, obviously very distressing for any patient. In cardiac patients dyspnea is the manifestation of circulatory insufficiency. It may be classified according to its severity. That is why it is necessary to reveal when and under what conditions it arises in a patient (e. g., in a state of rest, in walking, in running, in going upstairs, during climbing a hill or, finally, during arduous exercises, physical work or sport training). Ask if the patient gets relief in some specific posture/position.

A doctor needs to know if the breathlessness in question is permanent or intermittent, setting in with attacks, and whether is it accompanied by a sense of fear. Dyspnea in heart failure is caused by hypercapnic, hypoxemic and other reflex influences exerted on the integrative brain respiratory center. The most severe dyspnea is that of a

quiescent state, when a patient may suffer from it even in bed. Dyspnea is common for heart disease patients, particularly in mitral stenosis, but also occurs in atherosclerotic cardiosclerosis, pericarditis, myocardial infarction and other cardiac diseases, e. g. cardiomyopathy.

Commonly, the shortness of breath in cardiac diseases is of inspiratory (Traube's[1] type) or mixed variety. It is both an objective and subjective symptom. The extreme severity of dyspnea may force a patient to keep a sitting position, holding to some support with his hands in order to bring the additional respiratory musculature in action (constrained posture).

Dyspnea is brought in by congestive disorders in the lesser circulation which effect gas exchange in the blood and impair blood arterialization by the lungs, hampering the blood flow in the greater circulation. As a result, the growing levels of carbon dioxide in the blood and metabolic products of incomplete oxidation overexcite the respiratory center. The extreme irritation of the center decreases its excitability, causing Chayne-Stokes[2]' (tidal) respiration which is considered to be rather unfavorable for prognosis. Usually it occurs in the nighttime, during sleep.

Sometimes sudden assaults of breath shortness occur — so called asphyxia attacks. They should be distinguished from breathlessness of constant character. Asphyxia may occur in a state of rest, after exercises or stress, more often in wee hours, while sleeping. Permanent dyspnea may serve as a background for asphyxia assaults. The useful questions to the patient of this kind will be: if he has some moist gurgling rales in his chest on breathing; whether he/she suffers from breath shortness mostly when breathing in or out (or both phases are involved); if there is some sputum, rusty or with blood expectoration of scarlet color. This asphyxia attack is referred to as "cardiac asthma". Typically it is caused by the left ventricular pump failure, e. g., in mitral stenosis or other heart disease decompensation, in aortic valvular heart diseases, in myocardial infarction, cardiosclerosis or aneurysm of left-ventricular localization, hypertension of greater circulation (e. g., provoked by acute nephritis). Sudden asphyxia may accompany an attack of ciliary arrhythmia.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. At what stage of an interview can cardiovascular diseases be recognized?

[1] Ludwig Traube, Austro-German internist and pathophysiologist, born January 12, 1818, Ratibor in Oberschlesien; died April 11, 1876, Vienna.

[2] Cheyne John, 1777–1836, Scottish physician; Stokes William, 1804–1878, Irish physician.

2. *What must be revealed first of all?*
3. *What does the list of the main cardiovascular patient's complaints include?*
4. *What kind of malaise is breathlessness?*

4. Read the task card below.

Describe the main peculiarities of breathlessness following the plan:

What are the main complaints of cardiovascular patients?
What is breathlessness?
In what patients is breathlessness common?

5. Now, using the prompts from exercise 4, talk on the topic: BREATHLESSNESS.

Unit 2.2

EVALUATION OF PATIENT'S COMPLAINTS IN CARDIOLOGY: PAIN

Aches and Pains

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

analgesic	болеутоляющее средство, аналгетик
aortic aneurysm	аневризма аорты
cardioneurosis	кардионевроз, невроз сердца (*устар.*)
diaphragmatic	диафрагмальный
endocarditis	эндокардит (воспаление эндокарда)
fracture	перелом
Glisson's capsule	фиброзная капсула печени, глиссонова капсула
hernia	грыжа
herpes zoster	опоясывающий герпес, опоясывающий лишай
intercostal	межреберный
ischemic heart disease	ишемическая болезнь сердца
lumen	просвет; полость трубчатого органа

mesaortitis	мезаортит (воспаление мышечной оболочки аорты)
myeloma	миеломная болезнь, множественная миелома, болезнь Рустицкого–Калера
myocarditis	миокардит (воспаление сердечной мышцы)
myositis	миозит (воспаление мышцы)
neuralgia	невралгия
osteochondrosis	остеохондроз
pancreatitis	панкреатит (воспаление поджелудочной железы)
pericarditis	перикардит (воспаление перикарда)
periostitis	периостит (воспаление надкостницы)
plethora	плетора, гиперволемия
pleurisy	плеврит (воспаление плевры)
stenocardia, angina pectoris	стенокардия, грудная жаба
syphilitic	сифилитический

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Complaints about pains in cardiac area are of great importance. A doctor must ask the following necessary questions:

Where do you get this pain?

— Show me exactly (mind the retrosternal or heart apical localization).

— What kind of pain is it? (Mind the character of the pain: pang, gnawing, burning, smarting, stabbing, piercing, beating, bursting, aching, dull, boring, throbbing, sharp, steady, cramp, spasmodic, cutting, shooting, clasping, dragging, drawing, gnawing, pressing, knife-like, straining, tearing, tingling, twinkling, gripping etc.).

— Are the pains accompanied by the sensation of retrosternal squeezing and heaviness or pressure?

— Are they constant or spasmodic?

— Did it come on slowly or suddenly?

— What was the duration of the attack?

— Does anything special bring it on? (e. g., rest position, excitement, walking, exercises, beginning of meal or satiety).

— Do they occur in the daytime or at night?

— Does it radiate anywhere else? (e. g., to the left or right shoulder, towards the left arm, to the abdomen, under the left shoulder blade).

— Does anything relieve it? (Any drug, a comfortable position, turning the right side down or lying on the back or the tummy, a bowed position etc.).

The case history without a detailed description of the character of the pain is invalid. A diligent registration of the details of these signs is very helpful in making a diagnosis. Most often, pains in the heart are connected with coronary circulation insufficiency. These pains are due to ischemia provoked by permanent or intermittent spasms or occlusion of coronary arteries. Unfortunately, a typical angina pectoris with characteristic spasmodic gripping or burning retrosternal pain ("squeezing", as described and usually accompanied by an appropriate gesture showing tightening) takes place only when the arterial lumen is already narrowed to a quarter of the normal.

Besides the ischemic heart disease, cardiac pains may be a result of myocarditis, pericarditis, endocarditis, aortic aneurysm, for example, in syphilitic mesaortitis, as well as of cardioneurosis.

Finally, they may be of noncardiac origin (left dry pleurisy, intercostal neuralgia, herpes zoster, myositis, spinal osteochondrosis, pancreatitis, diaphragmatic hernia, costal fractures, periostitis, tuberculosis, systemic myeloma disease etc.). Sometimes detailed questioning about the character of cardiac pains gives the possibility to establish the correct diagnosis. For example, pains in angina pectoris (stenocardia) are quite specific. They arise in exercise or in walking, forcing a patient to stop. After stopping he/she may be subdued. These pains (stenocardia of strain) may be provoked by wind, by coming out of a warm chamber into the open air, sometimes — by overeating.

More rarely, such pains occur at night time, when sleeping (stenocardia of rest, Prinzmetal's[1] stenocardia).

Such pains are usually retrosternal most often of squeezing, and pinching character. Pain may occur as spasmodic attacks and last a few minutes or about half an hour.

Common radiation is to the left arm and shoulder blade, sporadically to the mandible. It is relieved by rest or drugs (glonoin, methyl valerate). Frequently patients feel anxiety, suffer from the fear of death and try to lie still in bed, quite motionless.

The pain in myocardial infarction is of quite different kind. It is more intensive and prolonged, lasting for several hours or even days, it does not calm down after taking glonoin and other vasodilatation drugs. In the majority of patients, infarction follows some physical or negative emotional stress. The patient with infarction does not suffer from the fear of death and usually can walk. It is common knowledge that in a lot of cases myocardial infarction was revealed long after the stenocardia attack, or occasionally — by accidental ECG or postmortem in autopsy. Thus, a patient may sustain through infarction "on feet".

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[1] Prinzmetal Myron (1908–1987), American cardiologist.

In myocarditis the pain is less pronounced, weak, dull, changeable, usually pressing, pinching, but much weaker than in angina pectoris.

In pericarditis the pain is durable and varies in intensity; some postures (e. g. half-bowed, sitting with the hands around the knees, lying on the tummy) may relieve it. In endocarditis the pain is, usually, dull and constant, not so severe, as in infarction and angina. In aortitis (including syphilitic type) retrosternal pain is permanent, not depending on worry or exercises, resistant to coronarolytic therapy and curable only by narcotic analgesics.

Cardioneurosis is commonly manifested by durable piercing or vague pain in the heart apical region, without any radiation, but proved by different emotions.

In heart disease, pain in the right subcostal area may be a result of the congestion of the greater circulation and liver plethora, leading to extreme distension of Glisson's capsule, rich in pain receptors. Acute pain of extreme severity in the right or left side may arise in thromboembolism of the pulmonary artery and its branches. Cough and bloody sputum expectoration accompanies it.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What are the necessary questions concerning pains in the cardiac area?*
2. *Why is detaled registration of the signs important?*
3. *What are pains in the heart connected with most often?*

4. Read the task card below.

Describe the main peculiarities of pains following the plan:
What are the necessary questions concerning pains in cardiac area?
What causes cardiac pains?
What diseases may cardiac pains result from?

5. Now, using the prompts from exercise 4, talk on the topic: PAINS.

Unit 2.3

EVALUATION OF PATIENT'S COMPLAINTS IN CARDIOLOGY: EDEMA AND RELATED COMPLAINTS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

anticoagulant	антикоагулянт, противосвертывающее средство
belching, eructation	отрыжка
congestion	застой (напр. крови, желчи)
constipation	констипация, запор, обстипация
diapedesis	диапедез
dicumarol	дикумарол
distension	вздутие (живота)
embolism	эмболия (закупорка кровеносного сосуда эмболом)
erythrocytosis	эритроцитоз, полицитемия
fenilin	фенилин
flatulence	метеоризм (вздутие живота вследствие скопления газов)
gut	кишка
left ventricular failure	левожелудочковая недостаточность
mitral valve	левый митральный клапан
nodular	нодозный, узелковый; узловатый
pelvic	тазовый
periarteritis	периартериит (воспаление адвентиции артерии)
RBC	1) [red blood cell] эритроцит; 2) [red blood count] количество эритроцитов
stool	стул
thrombophlebitis	тромбофлебит
warfarin	варфарин

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Edema. Usually, edema accompanies advanced congestion in the greater circulation. A doctor must clarify the localization of dropsy. The most frequent correlations are the following:

Low extremities — advanced heart failure.

All over the body and extremities — dreadfully severe heart failure (anasarca).

Face — it can be renal disorders.

The useful questions are:

— When does the dropsy appear?

(After walking, at night, in the morning)

— Does it subside after resting in bed?

— Is the dropsy connected with the hot season?

(Some women in menopausal period suffer from leg dropsy in hot weather.)

The edema may be aggravated by consuming too much salt and excessive drink.

Ascites (which is known as abdominal dropsy) is the accumulation of the fluid collection in the peritoneal cavity which causes complaints of abdominal protrusion, heavy feeling "in the tummy", flatulence, stretching in abdomen. Partly heaviness and distension are caused on by liver swelling and Glisson's[1] capsule strain. In congestion of the greater circulation we first observe liver enlargement, and later cardiac dropsy may be observed.

Blood expectoration

Blood expectoration is a sign of noticeable congestion in the lesser circulation and consequent diapedesis of RBC out of dilated capillaries. Most often it is observable in mitral stenosis, and may occur in the left ventricular failure, or myocardial infarction. The sputum may be either rusty or scarlet. Blood expectoration is typical of pulmonary artery embolism and subsequent lung infarction. Blood expectoration also accompanies some cases of mitral valve disease, myocardial infarction, pelvic vein thrombophlebitis, erythrocytosis, and pulmonary nodular periarteriitis.

Iatrogenic blood expectoration is provoked by indirect anticoagulants in overdosing (fenilin, warfarin, dicumarol). This cause should be excluded by questioning. Blood expectoration may progress to real pulmonary bleeding.

Cardiac asthma is characterized by foamy rose-coloured sputum, containing a small amount of blood.

Dyspepsia

In cardiovascular diseases dyspepsia manifests as sickness, sometimes vomiting. The patients complain of belching (gaseous, or meal eructation), celiectasia (abdominal distension) may occur. Sometimes it appears as constipation or, vice versa, loose (watery) stools. The circulatory congestion within liver and gut causes these digestive disorders.

[1] Francis Glisson — British physician, anatomist, physiologist, and pathologist, born 1597, Rampisham, Dorsetshire; died October 14, 1677, London.

But a cardiologist has to bear in mind that the gut symptoms of digitalis overdosing are similar.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What must the doctor clarify?*
2. *What are the most frequent correlations?*
3. *What are the useful questions?*

4. Read the task card below.

Describe the main peculiarities of edema, blood expectoration and dyspepsia following the plan:

What are the most frequent correlations in the case of edema?
What are the useful questions to be asked in the case of edema?
What is blood expectoration?
What is dyspepsia?

5. Now, using the prompts from exercise 4, talk on the topic: EDEMA, BLOOD EXPECTORATION AND DYSPEPSYA?

Unit 2.4

EVALUATION OF PATIENT'S COMPLAINTS IN CARDIOLOGY: PALPITATIONS AND OTHER COMPLAINTS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

aggravator	фактор, вызывающий обострение
anamnesis morbi	анамнез morbi (совокупность сведений о болезни)
atropine	атропин
belladonna	белладонна, красавка обыкновенная
bigeminy	бигеминия (форма аллоритмии)
cardiomyopathy	кардиомиопатия
carditis	кардит (воспаление каких-либо структур сердца

cervical	цервикальный (1. относящийся к области шеи; 2. относящийся к шейке какого-либо органа)
cholinolytics	холинолитики (противодействующие эффекту ацетилхолина)
congestive heart failure	застойная сердечная недостаточность
endocarditis	эндокардит (воспаление эндокарда)
ephedrine	эфедрин (гидрохлорид эфедрина — сосудосуживающее и бронхорасширяющее средство)
epinephrine	адреналин, эпинефрин (*устар.*)
hypotensive	больной с постоянно сниженным артериальным давлением, гипотоник
meninx (*pl.*: meninges)	мозговая оболочка головного и спинного мозга
mesenteric	мезентериальный, брыжеечный
neurocirculatory dystonia	нейроциркуляторная дистония
pancarditis	панкардит (воспаление всех слоев стенки сердца)
paroxysmal	пароксизмальный
pulmonary embolism	эмболия сосудов легких; эмболия легочной артерии
sympathomimetics	симпатомиметики
tachycardia	тахикардия
thyrotoxic	тиротоксический (обусловленный повышенной функцией щитовидной железы)
vascular	васкулярный, сосудистый
vasculitis	васкулит, ангиит (воспаление стенок кровеносных сосудов)

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Palpitation is a relatively frequent symptom in cardiovascular patients, one of the main complaints.

Palpitation is a feeling of abnormally high pulse rate, usually arising in real tachycardia, but sporadically occurring as only a subjective sensation, in normal or low heart rate. Commonly, palpitation is related to increased excitability of the neuroendocrine apparatus regulating heart activity. Palpitation may occur in an absolutely healthy person, which makes it useful to ask if palpitation arises in any special condition (e. g., in a state of rest, in making efforts, in doing exercises etc., after drink-

ing alcohol, strong tea or coffee, when smoking or being anxious). High temperature, infections and various cardiac diseases may be accompanied by palpitations (e. g. including in congestive heart failure, pulmonary embolism, pancarditis, toxic carditis, myocardial infarction, risk of vascular collapse, thyrotoxic cardiomyopathy, neurocirculatory dystonia[1]). Palpitation may be brought on by certain drugs: sympathomimetics (epinephrine, ephedrin, alupent, asthmopent, euspiran), cholinolytics (atropine, remedies with belladonna)

The attacks of palpitation with heart rate > 160 beats per minute are called paroxysmal tachycardia. The latter may be the result of improper neuroendocrine regulation, cervical spinal disorders, latent foci of infection and toxic or degenerative cardiomyopathy. The ventricular form is connected with cardiosclerosis and has a less favorable prognosis.

Pulse intermissions

Intermissions of pulse are also a frequent complaint of cardiologic patients. Patients may say that their "heart stops or sinks", they may have a sense of "a lump in the throat." Usually, it is a sign of extrasystoles. The latter may be registered in persons with a delicate autonomous nervous system, but sometimes also present a manifestation of serious heart desease. A regular type of pulse intermissions (like bigeminy) suggests possible a Digitalis overdosage.

Patient's general condition

Cardiac patients may feel exhausted, weak. They complain of easy fatigability. Work capacity is diminished. Immoderate nervousness is common. Sleep disorder, headaches and dizziness are usually displayed in arterial hypertension, particularly in the "essential" type. This may also be conditioned by meninx swelling and congestion in cerebral circulation. A typical cerebral form of myocardium infarction is manifested mainly by neurologic symptoms. Hypotensive patients regularly suffer from cerebral disorders, particularly depending on weather changes.

Temperature elevation

Temperature elevation may be revealed in several inflammatory diseases of cardiovascular system (myocarditis and endocarditis, vasculitis of various etiologies). Sporadically, the temperature may be quite high. Ask if the patient has chills or profuse sweating in fever, whether is it accompanied by pain in the joint, how long it lasts, etc.

Other complaints

A variety of other complaints may be observed in cardiovascular patients. Some of them seem, at first glance, unrelated to the heart and vessels.

Examples may include:

[1] "Neurocirculatory dystonia" or "vegetative vascular dystonia" — an outdated term, which nevertheless is still in broad use in Russian medical texts, authored by specialists from ex-USSR republics. It is never used in English medical literature. The closest correct English equivalent is: **Somatoform autonomic dysfunction.** Some types of it are qualified by International Classification of Diseases (10th revised edition) as: **Panic attacks.** We still keep this term in the texts of this book, because it is designated for the readers, learning or working in Russia.

— Hoarse voice in advanced mitral stenosis;
— Cough in lesser circulation congestion;
— Stomachache in an atypical abdominal form of myocardium infarction and thromboembolism of mesenteric artery;
— Black stools and coffee-like vomits in acute gut ulcer, caused by stress in myocardium infarction.

Muscular and joint pains and ache in the left side manifest Dressler's post-infarction syndrome. Every complaint must be carefully analyzed and explained in combination with the others. Patients usually tell the truth, with the exeption of aggravators. It is advisable for a beginning practitioner to write down the case history in the course of questioning lest he/she should forget some important details. Growing experience will train the memory and allow postponing this process. Tape recording is not a proper aid during an interview, because some patients get too conocious of it, so the attempt of tape recording makes a patient inhibited and less communicable. After the complaints have been acquired, it is necessary to proceed to anamnesis morbi.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *Is palpitation quite a frequent symptom in cardiovascular patients?*
2. *What is palpitation?*
3. *What is palpitation commonly related to?*
4. *Can palpitation occur in an absolutely healthy person?*

4. Read the task card below.

Describe the main peculiarities of palpitation, pulse intermissions, patient's general condition, temperature elevation and other complaints following the plan:
What is palpitation?
What are other complaints?

5. Now, using the prompts from exercise 4, talk on the topic: PALPITATION, PULSE INTERMISSIONS, PATIENT'S GENERAL CONDITION, TEMPERATURE ELEVATION AND OTHER COMPLAINTS.

Module 3
ANAMNESIS MORBI ET VTAE (CASE AND LIFE HISTORY)

Unit 3.1
THE HISTORY OF THE CASE (ANAMNESIS MORBI)

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

atherosclerosis	атеросклероз
cardialgia	кардиалгия (боль в сердце)
Doppler ultrasonography	допплеровская ультрасонография, эхография, допплеровское УЗИ
ECG [electrocardiogram]	электрокардиограмма, ЭКГ
exacerbation	обострение болезни
impedance pletismography	импедансная плетизмография, реоплетизмография
neurocirculatory dystonia = somatoform autonomic dysfunction	нейроцикуляторная дистония, вегетососудистая дистония, вегетоневроз (*рус.*)
hypertension	гипертензия, гипертония
rheumatic fever	ревматическая атака
streptococcal	стрептококковый
valvular heart disease	клапанный порок сердца

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

It is well known that many medical doctors (A. P. Chekhov, M. A. Bulgakov, A. Conan Doyle, F. Rables, A. J. Cronin) achieved great public recognition as prominent writers. This may witness for the exclusive importance of literary gift to tell the stories in the medical profession. Probably, these writers-physicians have polished their literary skills in collecting anamnesis vitae et morbi from their patients. Indeed, the ability to collect and interpret information about a patient's life and the history of a current disease is a very essential part of a doctor's proficiency.

While questioning the patient, the doctor must reveal the history of the current disease development. It means, for example, the time of its onset. Rheumatic fever takes onset in childhood. The symptomatic arterial hypertensions arise more commonly in younger persons than the essential one. Somatoform autonomic dysfunction and other distress-related disorders off circulation are peculiar to adolescence. The manifested complications of atherosclerosis take place in elder persons and mature age. It is also quite important to take a patient's gender into account. If a young man has cardiac pain of gripping character, it is a very danderous sign, because ischemic heart disease develops in males much quicker and earlier than in females. At the same time, cardialgia in young females is not usually related to coronary artery disease and the forecast is more favorable. Useful questions will be: What symptoms were the first to appear? (Pain, breathlessness... etc.) When did it start? What was the character of the symptoms? When was the patient consulted by the doctor for the first time? What kind of examination and treatment was prescribed at that time?

For a cardiologic patient it is vitally important to archive and keep all the results of previous examinations (especially laboratory and graphic ones — ECG, Doppler ultrasonography, polycardiography, impedance pletismography etc.).

It will provide the opportunity to compare previous and current pictures, which is invaluable for complicated cases like ECG-diagnostics of myocardial infarction on the background of preliminary arrhythmia. Do ask the patient if he/she has such an archive. Verbal description of the previous objective data is not reliable, because the patient may be incompetent in cardiologic functional diagnostics and may misinform his doctor.

It is essential to clear the probable relation between cardiac disease and prerequisite infections, especially streptococcal ones, and also the connection with other diseases and stresses. For example, Georgiy Feodorovich Lang[1] believed that chronic stress might be an important risk factor for essential hypertension. It is necessary to obtain information concerning the previous treatment: its character, effectiveness and regularity, which are especially vital for the day-by-day treatment of hypertension. If the disease develops with exacerbation, ask the patients what provoked it? The cause may be irregular treatment, stresses etc.

[1] Lang, Georgiy Feodorovich, 1875–1948, Russian internist, the discoverer of hypertonic disease.

Anamnesis morbi should be completed with a detailed description of the latest exacerbation, which brings the patient to your clinic or outpatient department. If these particular complaints arose for the first time, the doctor must clear the information about probable health disorders in the patient's past life, even in his/her childhood.

The following situation is not unique: a patient of mature age has the first appearance of arrhythmia and dyspnea. In early childhood he had joint pain and swelling of joints. Rheumatic fever proceeded obscurely, and heart disease was latent. The picture may be connected with non-recognized rheumatic valvular heart disease, freshly progressed to non-compensated state. It is especially characteristic of rheumatic aortic valvular diseases, which may be firstly recognized only in a senescent age as a result of left ventricular failure. The next part of an interview is questioning about the patient's life history.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What are the names of some prominent writers who were medical doctors by profession?*
2. *Do you think writers-physicians polished their literary skills in collecting anamnesis vitae et morbi from their patients?*
3. *What ability is an essential part of a doctor's proficiency?*

4. Read the task card below.

Describe the main peculiarities of work aimed at questioning a patient following the plan:

What doctors became outstanding writers?

What should a doctor reveal while questioning a patient?

5 Now, using the prompts from exercise 4, talk on the topic: WORK AIMED AT QUESTIONING A PATIENT.

Unit 3.2

HISTORY OF THE PATIENT'S LIFE (ANAMNESIS VITAE)

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

AIDS (acquired immune deficiency syndrome)	СПИД (синдром приобретенного иммунного дефицита)
angina	1) ангина; 2) стенокардия, грудная жаба
brucellosis	бруцеллез
cardiomyopathy	кардиомиопатия
case history file	история болезни
complication	осложнение
contagious	контагиозный, заразный
diabetes mellitus	сахарный диабет
endocarditis	эндокардит (воспаление эндокарда)
enterovirus	энтеровирус
erysipelas	рожа; рожистое воспаление
exudative	экссудативный, выпотной
helminthiasis	гельминтоз, глистная инвазия
labor	роды, родовой акт
lesion	повреждение, поражение, патологическое изменение
lesion	повреждение, поражение, патологическое изменение
malaria, jungle fever	малярия
obesity	ожирение
pelvic	тазовый
pericarditis	перикардит (воспаление перикарда)
phthisiologic	фтизиатрический
pregnancy	беременность
pseudotuberculosis	псевдотуберкулез
staphylococcus (staphylococci)	стафилококк
streptococcus (streptococci)	стрептококк
syphilis	сифилис
thromboembolism	тромбоэмболия (эмболия вследствие тромбоза)
thrombophlebitis	тромбофлебит (воспаление вены с ее тромбозом)
tonsillitis	1) ангина; 2) тонзиллит, амигдалит
tricuspid valve правый	трехстворчатый клапан
Trypanosoma	трипаносома

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

The aim of questioning in cardiology is to disclose all possible risk factors that may contribute to the development of a peculiar pathology in every particular case. Usually, it starts with the date and place of birth and social origin of the patient interviewed. Some cardiovascular diseases are connected with local ecological factors, early living conditions and psychological climate of the family, where the patient grew up. It is recommended to find out how the patient grew and develop, wether he was successful in childish games, sports, and competitive exercises with his mates (it may not be essential in some congenital and acquired heart diseases), wether he made good progress at school. It is worth checking patients' occupational history from the beginning of their working life. Several occupations exert a negative influence on the cardiovascular system. It is a well-known fact that telephone operators are a leading group in the incidence of essential hypertension. This disease also frequently affects young people, who combine work and studies due to excess fatigue and lack of sleep. Some professions are connected with hazards and toxic products. The examples may be found among cases such as overeating in confectioners, excessive beer and alcohol drinking by restaurant and coffee shop staff, cardiovascular toxins exposure in chemistry etc.

General anamnesis is also of great significance. It is necessary to ask, what diseases can be treated in the patient's parents and close relatives. Genetic predisposition is especially important in certain multifactorial diseases such as essential hypertension, diabetes mellitus, hypertrophic cardiomyopathy, rheumatic fever, familiar dyslipoproteinemias, obesity and others.

Any infectious disease a patient has recovered from, beginning from his/her childhood up to the day of examination, should not escape the doctor's attention. It includes childhood infections, streptococci and staphylococci infection (tonsillitis, angina, erysipelas etc.). The diphtheria endured in childhood may later cause myocarditic cardiosclerosis in an adult. Frequent streptococcal angina may cause rheumatic heart damage such as endocarditis. Some contagious agents may later, after infections, provoke a disease of a latent autoimmune process, directed towards the cardiovascular system, causing late subsequent therapeutic pathology of the heart and vessels (chlamydiae, trypanosomae, enteroviruses and other).

Phthisiologic anamnesis is also important. Try to clear out, if the patient himself or somebody among his closest persons has suffered from tuberculosis. It may have caused subsequent exudative pericarditis.

It is very important to be assured that there have been no previous sexually transmitted diseases, syphilis in particular, because they may be the cause of aortic and coronary lesion in elder age.

We must bear in mind that any infectious disease can provoke a myocarditis, which is responsible for subsequent arrhythmia and heart failure due to myocarditic cardiosclerosis in mature age.

Special attention must be paid to the patient's bad habits: smoking, alcohol abuse and drug addiction. Smoking is a risk factor in atherosclerosis, alcohol drinking is considered to be a risk factor in arterial hypertension and cardiomyopathy. But moderate doses of alcohol are recognized as an anti-risk factor in atherosclerosis. Drug addiction with intravenous narcotic injections via non-sterile syringes may facilitate the development of septic endocarditis of tricuspid valve, thrombophlebitis and thromboembolism. When questioning a female patient, the doctor should not forget gynecological anamnesis: the debuting of periods, age of marriage, how the pregnancies proceeded, if the patient has had abortions. Ask if hypertension has ever occured in pregnancy or labor? The post-partial complication of pelvic vein thrombosis is a frequent reason for bacterial endocarditis or pulmonary thromboembolism.

Sexual anamnesis is necessary in interviewing both males and females. This is particularly important when dealing with a myocardial infarction patient, who may subsequently suffer from some disease-derived sexual problems. Questions, regarding the possibility of AIDS infection are also acceptable, because this is helpful in identifying feverish patients with cardiac diseases.

The epidemiological anamnesis is of great significance, because it enables us to investigate the patient's contacts with the sources of contagious diseases, such as birds (doves and hens), animals (danger of brucellosis, pseudotuberculosis). Ask if the patient has visited any regions endemic in malaria and other tropical diseases (jungle fevers, helminthiases), and if he had got any immunizations shortly before the current disease (danger of autoimmune and allergic lesions of heart and vessels).

When collecting the allergologic anamnesis, it is worthwhile to take into consideration not only food allergy, but also drug allergy. Commonly, the data about the food and drugs, which usually provoke allergic and pseudo-allergic (allergoid) reactions in particular patient must be shown at some conspicuous place (for example, at the title page of case history file) because of their great role in vessel and heart condition. Ask the patient about his/her living conditions, financial security, habits, way of life, usual diet, attitude to exercises and sport, hygienic habits, family and personal affairs.

The history of a patient's life is also of merit, because it is related to cardiovascular diseases forecast.

The next part of the doctor's direct interaction with the patient is devoted to physical methods of diagnosis.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What is the aim of questioning in cardiology?*
2. *What does it usually start with?*
3. *What details of a patient's life are recommended to be clarified?*

4. Read the task card below.

Describe the main peculiarities of questioning in cardiology following the plan:

What is the aim of an interview in cardiology?
How is questioning organized?
What is blood expectoration?
What is dyspepsia?

5. Now, using the prompts from exercise 4, talk on the topic: EDEMA, BLOOD EXPECTORATION AND DYSPEPSYA?

Module 4

OBJECTIVE PHYSICAL EXAMINATION IN CARDIOVASCULAR DISEASES: VISUAL EXAMINATION

Unit 4.1

PATIENT'S POSITION, GENERAL STATE AND SOMATOTYPE EVALUATION

1 Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

aneurysm	аневризма
ascites	асцит, брюшная водянка
asthenic	астенический
auscultation	аускультация, выслушивание
clonic convulsion	клоническая судорога
coma	кома, коматозное состояние; глубокое бессознательное состояние
consciousness	сознание
dilatation	дилатация, расширение
dystonia	дистония, нарушение тонуса
ectomorphic	астенического телосложения
encopresis	энкопрез, недержание кала (чаще функциональное)
endomorphic	пикнического типа телосложения
enuresis	недержание мочи, энурез; ночное недержание мочи
etiology	этиология
general state	общее самочувствие
hypersthenic	гиперстенический (характеризующийся гиперстенией)
hypotension	гипотензия, гипотония
mesomorphic	мезоморфного типа телосложения
neurocirculatory	нейроцикуляторный
normosthenic	нормостенический

palpation	пальпация, ощупывание, прощупывание
percussion	перкуссия, выстукивание
somatotype	тип конституции, тип телосложения
stupor	1) помрачение сознания; оглушение; 2) ступор
syncope	синкопе, обморок
tonic convulsion	тоническая судорога
vasculitis	васкулит, ангиит (воспаление стенок кровеносных сосудов)
visual examination	визуальный осмотр

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

In cardiovascular patients, objective physical investigation commonly proceeds according to the following sequence: visual examination, palpation, percussion, and auscultation. Both the heart and the vessels should be carefully examined. During a visual examination, the doctor must primarily inspect the patient's position. In a compensated state of the disease the mode of life of a cardiovascular patient is ordinary and normal. A patient can walk easily and usually has no complaints. In overt heart failure such patients instinctively prefer to keep to bed remaining in Fawler's position[1], half-risen, almost half-sitting, getting from it some relief. As a rule, in this station they avoid lying on their left side to escape unpleasant sensations in the heart. Severe breathlessness may prompt a patient to sit on the bed with the feet on the floor. This position facilitates blood deposition in the lower extremities and relieves congestion in the lesser circulation. Such a position is also helpful for ascites patients since it facilitates diaphragmatic respiratory movements. In severe heart failure the patients would sit in this position round the clock all night and even for weeks unless the proper therapy is applied. Patients with left ventricular failure (caused by myocardial infarction, noticeable cardiosclerosis, left ventricular postinfarction aneurysm, valvular heart diseases etc) also keep to this position. It is typical that patients with right ventricular failure ("Cor pulmonale") rarely assume this attitude and commonly prefer a normal

[1] George Ryerson Fowler: American surgeon, born December 25, 1848, New York City; died February 6, 1906, Albany, New York.

laying position in spite of dyspnea. In acute vascular failure (shock, collapse) patients also keep to a horizontal position, because a minor elevation provokes insufficient cerebral circulation and syncope.

In certain cardiac diseases patient have to remain in some peculiar forced positions, relieving their suffering (e. g., in exudative pericarditis they may keep on all fours or lie on the stomach, sometimes sit holding their knees). In noticeable heart dilatation patients commonly lie on the right side. In thrombophlebitis and other lower extremities' vein diseases patients may keep the legs in a raised position, trying to relieve pain and tension in the low extremities. The position with feet end of the bed raised is called "Trendelenburg's pose[1]".

The next thing to evaluate is the patient's general state. It may be either satisfactory or serious (extremely serious). In a satisfactory state a patient can usually walk in a normal way, does not take on any forced position, can easily take care of himself. In a serious case, somebody usually has to look after the patient who is unable to take care of himself or even change his position. During examination, the doctor also assesses the patient's level of consciousness. It may be clear when a patient takes part in the conversation and answers questions, and is properly oriented in time and circumstances. In a stunned (staggered somnolent) state the patient is hardly aware of the time and circumstances and may be inadequate in his answers or hesitates with answers. In a stupor state, the patient is insensible, does not answer the questions, but responds to the strong stimuli (e. g. injections), and is able to maintain all the physiological reflexes, usually keeping stools and urine. In a comatose state consciousness is absolutely lost, normal reflexes are absent, pathologic ones may arise, which are not normally observable. Tonic or (more often) clonic convulsions may accompany coma. A state like this can be registered in the cerebral form of myocardial infarction. Patients with rheumatic heart diseases may develop it after cerebrovascular thrombotic embolism or in heavy cerebral vasculitis of different etiology. Commonly, comatose patients are unable to keep stools and urine and have involuntary enuresis and encopresis. Visual examination is to determine a patient's somatotype (constitution). It may be normosthenic, asthenic or hypersthenic (picnic). M. V. Tchernorutzky[2] introduced these terms in Russia. In western countries analogous division by Shelton's[3] criteria (ectomorphic, endomorphic and mesomorphic somatotypes) exists. The type of human constitution is quite important in cardiovascular diseases spreading and diagnostics. Persons of hypersthenic somatotype are genetically more susceptible to arterial hypertension and atherosclerosis, asthenic ones — to somatoform autonomic dysfunction and arterial hypotension.

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[1] Friedrich Trendelenburg, German surgeon, born May 24, 1844, Berlin; died December 15, 1924, Nikolassee near Berlin.

[2] Mikhail Vasil'evich Tchernorutzky, a Russian internist, 1884–1957.

[3] William Herbert Sheldon (November 19, 1898 — September 17, 1977), an American psychologist, eugeneticist and numismatist.

cyanosis	цианоз (синюшний оттенок кожи и слизистых оболочек, обусловленный недостаточным насыщением крови кислородом)
diabetes mellitus	сахарный диабет
diaphragmatic	диафрагмальный
dystrophic	страдающий дистрофией
erythema	эритема (ограниченная гиперемия кожи)
extraneous	инородный, посторонний
heart disease	сердечное заболевание; порок сердца
heart failure	сердечная недостаточность
hemoglobin	гемоглобин
hemorrhage	1) кровотечение; 2) кровоизлияние
hemorrhagic	геморрагический, относящийся к кровотечению
hepatic	печеночный
hyperemia	гиперемия, полнокровие
hypopituitarism	гипопитуитаризм (недостаточность функций гипофиза)
hypothyroidism	гипотироз
jaundice	желтуха
lymph node	лимфатический узел, лимфоузел
methemoglobin	метгемоглобин, ферригемоглобин
MSH, melanocyte-stimulating hormone	МСГ, меланоцитстимулирующий гормон
mucous membrane	слизистая оболочка
nasolabial	носогубной
noncompensated	некомпенсированный
oxyhemoglobin	оксигемоглобин, оксигенированный гемоглобин
pallor	бледность
panhypopituitarism	пангипопитуитаризм, гипофизарная кахексия, болезнь Симмондса
pericarditis	перикардит (воспаление перикарда)
pituitary necrosis	гипофизарный некроз
pneumothorax	пневмоторакс (наличие воздуха или газа в плевральной полости)
pseudocirrhosis	1) сердечный фиброз печени; 2) псевдоцирроз, перикардитический цирроз печени
rash	сыпь, (особенно быстропроходящая, мимолетная)
regurgitate	1) течь в обратном направлении; 2) срыгивать
rheumatic	ревматический
sclerosis	1) фиброзное уплотнение; 2) склероз
sclerotic coat	склера, белочная оболочка глаза
septic	септический, относящийся к сепсису
shock	шок
subicteric	субиктеричность (легкая желтушность)
syncope	синкопе, обморок
telangiectasia	телеангиэктазия (локальное чрезмерное расширение мелких сосудов)

thoracic	торакальный, грудной
thrombophlebitis	тромбофлебит (воспаление вены с ее тромбозом)
trophic disorders	трофический
ulceration	1) образование язвы; 2) изъязвление, язвы
vasculitis	васкулит, ангиит (воспаление стенок кровеносных сосудов)
vena cava superior	верхняя полая вена

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

The color of the skin is of great importance in cardiovascular diagnostics. Simultaneously you must check the color of the mouth mucous membranes. Visually, a doctor can observe color changes, as well as hemorrhagic rush, telangiectases, the manifestations of low extremities trophic disorders in peripheral circulation disturbance, resulting from chronic heart failure (right until the skin ulceration). In chronic and acute thrombophlebitis local hyperemia may be revealed, diabetes mellitus is characteristic for skin atrophy, dystrophic nails and pallor of the cruses and feet. Skin hemorrhagic rash may occur in septic conditions (e. g. bacterial endocarditis), rheumatic attacks (with annular erythema in joint areas), in vasculitis (for example, Schönlein-Henoch's[1] disease). Focus your attention on the localization of the rash and the presence or absence of symmetry. Cyanosis often occurs in cardiovascular diseases. In an extreme degree, both a patient's face and trunk appear almost black (severe non-compensated Cor pulmonale). At the initial stage cyanosis is more noticeable in the areas of thin skin: nose, lips, chicks, hands (fingers). Progressing cyanosis spreads onto the body and extremities. Usually cyanosis marks the elevated blood level of reduced hemoglobin, which has darker color as compared with oxyhemoglobin. The causes of cyanosis are different. In central cyanosis blood arterelization is disturbed, as a result of lung diseases. In peripheral cyanosis blood circulation is retarded, thus facilitating oxygen diffusion into tissues. As a result, venous blood also receives reduced amounts of hemoglobin. Patients with cor pulmonale of different etiology usually have most pronounced central cyanosis (for either bronchopulmonary or

[1] Johann Lukas Schönlein, German physician, born November 30, 1793, Bamberg; died January 22, 1864, Bamberg.; Eduard Heinrich Henoch, German paediatrician, born June (30: July) 16, 1820, Berlin; died 1910, August 25, Dresden.

thoracic-diaphragmatic and vascular reasons). Different cardiac diseases commonly produce peripheral cyanosis due to circulation disturbance. Cyanosis may be either local (in thrombophlebitis of lower extremities veins and mechanical compression of the veins by enlarged lymph nodes), or extended (spread). The later is especially noticeable in severe congenital heart diseases, pulmonary artery sclerosis (Ayerza's disease[1]). The cyanoderma may be permanent (chronic heart failure) or sets in abruptly (pulmonary artery branches embolism, pneumothorax). If vena cava superior is compressed (by the tumor) isolated cyanosis of the head and neck may occur. In poisoning with certain drugs and poisons, cyanosis may be a result of extraneous methemoglobin or sulphohemoglobin production. Any efforts and exercises tend to increase both cyanosis and dyspnea.

The presence of unusual skin and mucous membrane pallor in the absence of anemia may be observed in stenotic aortal heart disease, while mitral heart diseases are always accompanied by so called "mitral face": cyanosis plus typical appearance — bluish lips and cheeks and pale nasolabial triangle. Pallor in aortal stenosis is caused by insufficient blood supply into the arterial system from the left ventricle. But, pallor may accompany aortal insufficiency as well due to a large portion of regurgitated blood and the decrease in peripheral blood supply. Patients with bacterial endocarditis are particularly pale; this tinge of the skin is called "white coffee."

Noticeable skin pallor is typical for hypopituitarism, including panhypopituitarism and so called Sheehan's[2] syndrome (due to declined MSH production). Sheehan's syndrome may develop after hard labor in persons with high blood pressure. It is believed to be caused by pituitary necrosis due to either tromboembolism of the supplying artery or intrapituitary hemorrhage. There are also signs of autoimmune hypophysitis in this disease. Paleness is also characteristic of patients with hypertonic disease, as well as in collapse, shock and syncope.

Yellowish skin and mucous membranes are frequent manifestations of advanced chronic heart failure because of liver swelling and hepatic dysfunction. Subicterus of sclerotic coats is commonly observed in this case. Subicterus of skin may be observed in diabetes mellitus and hypothyroidism. Constrictive pericarditis provokes both functional and morphologic changes within the liver (due to congestion in venae cavae) known as Niemann-Pick[3] pseudocirrhosis. In this case the signs of jaundice are quite pronounced.

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[1] Abel Ayerza, Argentinian physician, 1861–1918. Buenos Aires

[2] Harold Leeming Sheehan, English pathologist, born 1900, Carlisle; died October 25th, 1988, Liverpool.

[3] Albert Niemann, German paediatrician, born February 23, 1880, Berlin; died March 22, 1921, Berlin. Ludwig Pick, German pathologist, born August 31, 1868, Landsberg an der Warthe; died February 3, 1944, Theresienstadt Concentration Camp, where he was killed by the Nazis.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *How important is the color of the skin in cardiovascular diagnostics?*
2. *What should a doctor check simultaneously with the color of the skin?*
3. *What can a doctor disclose visually?*

4. Read the task card below.

Describe the main characteristics of checking the color of the skin following the plan:

Why is the color of the skin so important in cardiovascular diagnostics?

What diseases connected with the color of the skin can be revealed?

5. Now, using the prompts from exercise 4, talk on the topic: CHECKING THE COLOR OF THE SKIN.

Unit 4.3
EVALUATION OF EDEMAS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

ad oculus	визуально
alopecia	алопеция, облысение
anasarca	анасарка (распространенный отек подкожной клетчатки)
atherosclerosis	атеросклероз
blisters	буллезное поражение
bronchiectasis = bronchiectasia	бронхоэктаз (расширение ограниченных участков бронха)
cardiac dropsy	гидроперикард, водянка перикарда
cerebral	церебральный, мозговой
contusion	1) ушиб; контузия; 2) закрытая травма
crural	голенный, находящийся на голени

cutaneous	кожный
diuretic	мочегонное средство, диуретическое средство, диуретик; мочегонный, диуретический
Dressler's postinfarction syndrome	постинфарктный синдром; синдром Дресслера (сочетание перикардита с плевритом и синовиитом)
hepatic cirrhosis	цирроз печени
hydropericardium = hydropericarditis	гидроперикард, водянка перикарда
hypothyroidism	гипотироз
metastasis	метастаз
myxedema	микседема; синдром Галла, гипотироидный отек
ovary	яичник
palsied	1) парализованный; 2) дрожащий, трясущийся
paroxysmal	пароксизмальный
pericardial = pericardiac	перикардиальный, относящийся к перикарду
peritoneal	перитонеальный, брюшинный
pilosis (in females — hirsutism)	избыточное оволосение (у женщин — гирсутизм)
pitting edema	образование углублений при надавливании на отечную область
renal dropsy	почечный отек
sacral	крестцовый, сакральный
scrotal	мошоночный
scrotum	мошонка
subcutaneous	подкожный
tachycardia attack	приступ тахикардии
tetralogy of Fallot	тетрада Фалло, тетралогия Фалло
ulceration	изъязвление, образование язвы

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

During an *ad oculus* examination the doctor recognizes the existence of edema (dropsy) and its localization (face, extremities, and trunk). Edemas are common in cardiovascular patients. When patients spend much time walking or standing, dropsy is usually located in their low

extremities. If a patient spends most of the time lying in bed, edema may be observed on his back, sacral area, or the right side, when he tends to lie predominantly with the right side down. Both cardiac and renal dropsy react with dimple (pit) at the place of probing, which will slowly flatten out after the doctor's finger is taken away (pitting edema). Only one disease always gives dropsy without showing pits after pressure — it is hypothyroidism, known as myxedema. Extremely manifested total edema of subcutaneous fat in abdominal and chest area plus fluid collection within the peritoneal, pleural and pericardial cavities is called anasarca. The preponderant fluid congestion in pleural cavity is referred to as hydrothorax (left, right, bilateral); in abdominal cavity — ascites; in pericardial cavity — hydropericardium. Left hydrothorax may be caused by heart failure or Dressler's[1] postinfarction syndrome, right hydrothorax — by compression of vena cava superior. Hydropericardium may be a manifestation of anasarca or pericarditis (exudative with tuberculosis, rheumatic and other etiologies). Isolated ascites not accompanied by signs of heart failure or hepatic cirrhosis, may be strongly suggestive of some oncologic disease (most frequently, tumor of ovaries with peritoneal metastases). In edema of low extremities the skin is smooth, pale and tightened with a waxen appearance. Chronic edema is manifested in skin trophic disorders; skin becomes firm, stiff, non-elastic and dark, sometimes with crural ulceration. Vascular diseases may cause local edemas, for example in venous hyperemia due to some vein compression by either tumor, or enlarged lymph nodes.

Improper renal function in circulation diseases always contributes to edema formation. One should bear in mind that that cardiac dropsy, unlike renal one, always behaves in accordance with hydrostatic laws, locating at the lowest possible places due to gravitation. So, in renal diseases facial edema is common, although in chronic heart failure feet dropsy is more typical. The development of edema is, mostly, gradual. But dropsy may set in quite rapidly, for example in case of long paroxysmal tachycardia attack, quickly disappearing after the attack is over. Internal dropsy usually grows in parallel to the external one, although it is not visible from outside. So, dyspepsia in cardiac diseases may be related to liver and gut organ dropsy. In cerebral stroke a doctor may observe edema of palsied extremities. Before the introduction of diuretics into broad clinical practice, doctors have not infrequently encountered extreme degrees of edema, which may be manifested in cutaneous bullae (blisters) filled with plasma. The generalized edema in males usually involves scrotum, so it is essential to examine it, because scrotal dropsy may hurt a patient, the phenomenon of "drum stick fingers" may be revealed when examining a cardiologic patient both on the hands and feet. This symptom is typical for bronchiectases, but exists also in severe congenital heart diseases (like Fallot[2] tetralogy) or prolonged course of bacterial endocarditis. When examining the hair, doctor must pay attention

[1] W. Dressler, American physician, 1890–1969.

[2] Etienne-Louis Arthur Fallot, French physician, born September 29, 1850, Sète outside Marseille; died April 30, 1911.

to the degree of pilosis. Early and frequent baldness (alopecia) is noticeable for atherosclerotic patients. Certain types of arterial hypertension (post-contusion one) may bring in early hair loss.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What does a doctor determine during an ad oculus examination?*
2. *In what patients are edemas common?*
3. *What conditions the location of dropsy in the low extremities?*

4. Read the task card below.

Describe the main peculiarities of edema following the plan: **How does a doctor diagnose the existence of edema (dropsy) and its localization?** **In what patients is edema common?** **What may be caused by renal function?**

5. Now, using the prompts from exercise 4, talk on the topic: DIAGNOSING THE PRESENCE OF EDEMA

Unit 4.4

VISUAL EXAMINATION OF CARDIAC AREA

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

abdominal	абдоминальный
aneurysm	аневризма (расширение просвета кровеносного сосуда или полости сердца вследствие патологических изменений их стенок)
aortic	аортальный
ascending	восходящий, афферентный
beat	1) систола (сердца); 2) систолический шум; 3) пульс
Botallo's duct patency	Боталлов (артериальный) проток
cardiac apex	верхушка сердца

cardiac humpback	сердечный горб
duct	проток; канал; ход; проход-
epigastric pulse	надчревная пульсация
exudative pericarditis	экссудативный перикардит, выпотной перикардит
hydropneumothorax	гидропневмоторакс
hydrothorax	гидроторакс
hypersthenic	гиперстенический (характеризующийся гиперстенией)
hypertrophy	гипертрофия
insufficiency	недостаточность
mediastinal	медиастинальный, средостенный
patency	раскрытое состояние сосуда, канюли
pneumothorax	пневмоторакс
protrusion	выступание вперед, выпячивание; протрузия, выбухание
pulsation	пульсация, биение
retraction	1) втяжение; западение; 2) ретракция; стягивание, сокращение epigastrium надчревье, надчревная область, эпигастрий
somatotype	тип конституции, тип телосложения
spondylitis	спондилит (воспаление всех или некоторых структурных элементов позвоночника)
thoracic wall	стенка грудной клетки
thoracophrenic	грудобрюшный
tricuspid	трехстворчатый
unilateral	односторонний (о локализации патологического процесса)
ventricle	желудочек

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

The cardiac area should be examined with the light coming from the side, because this angle may enable to reveal protrusion or some movements there. The protrusion of the whole cardiac area is common for congenital heart diseases, manifested since early childhood (Botallo's[1]

[1] *The discovery of ductus arteriosus erroneously attributed to an Italian anatomist Leonardo Botallo (1530–1600). In fact it was discovered by another Italian anatomist and surgeon, Giulio Cesare Aranzi, born 1529/1530, Bologna; died April 7, 1589, Bologna.*

duct patency and other) and it persists during the patient's entire life. But, the "cardiac humpback" may be unrelated to heart pathology (it may occur in congenital anomalies of skeleton, chest and rib tumors). In adults some protrusion of cardiac area is observable in exudative pericarditis. The protrusion above the cardiac area may be caused by aortic aneurysm. Anterior mediastinal and thoracic tumors may also be accompanied by cardiac humpback. Severe chest deformations of different origin (e. g., in tuberculosis spondylitis) may lead to thoracophrenic cor pulmonale. Healthy non-obese individuals always have visible rhythmic pulsation in the cardiac area, caused by the impacts of beating cardiac apex and thoracic wall. The name of this phenomenon is apex (cardiac) beat (AB). Naturally, apex beat is situated a little bit upper and to the medium from the actual position of cardiac apex, which is covered by lungs. Apex beat is produced by left ventricle contractions.

In individuals of normal somatotype, AB should be in 5th intercostal space, 1,5–2 cm to the medial from the medioclavicular line. In asthenic persons AB is one intercostal space lower, and in hypersthenics — one space upper and a little to the left. Some cardiologists consider apex beat to be normal, and use the term "cardiac beat" only in the case of pathology accompanied by right ventricular hypertrophy (cor pulmonale, mitral stenosis, and tricuspid valve disease). These conditions produce rising pulsation in the area of left sternal edge, related to movements of hypertrophic right ventricle. The pulsation may also be observed to the right of sternum — in aorta ascendant aneurysm, aortal valve insufficiency. Cardiac aneurysm produces pathologic pulsation in 3^{rd}–4^{th} left intercostal space. In children before they reach the age of 5, AB may be situated in 4^{th} space laterally from medioclavicular line. Changing the position of the body shifts AB 3–5 cm away from its normal location. AB and the whole heart are moved aside in unilateral hydrothorax, pneumothorax or hydropneumothorax. Mind, that the heart in these cases is displaced to the opposite side from the side involved. In left hydrothorax AB is invisible and impalpable. It is hardly recognizable in obesity and marked pulmonary emphysema. Sporadically, retraction (retrograde motion) may be seen in cardiac area instead of protrusion. This is so called negative AB, characteristic of adhesive pericarditis, due to adherence and sticking pericardium layers together.

The area of epigastrium also has to be examined attentively. There may be three kinds of epigastric pulsation: from up-to-down (in right ventricle hypertrophy) from right-to-left (in liver enlargement due to tricuspid valve insufficiency) from back-forward (derived from abdominal aortic pulsations, as a rule in slim patients).

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *Why should the cardiac area be examined with the light coming from aside?*
2. *For what diseases is the protrusion of the whole cardiac area common?*
3. *May the "cardiac humpback" be unrelated to heart pathology?*
4. *Where is some protrusion of cardiac area observable in adults?*

4. Read the task card below.

Describe the main peculiarities of protrusion of cardiac area following the plan:

In what diseases is the protrusion of the entire cardiac area observable?

What are apex beat characteristics in different individuals?

How does changing the position of a body affect AB?

5. Now, using the prompts from exercise 4, talk on the topic: THE PROTRUSION OF CARDIAC AREA

Unit 4.5

VISUAL EXAMINATION OF MAJOR PERIPHERAL BLOOD VESSELS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

apex beat, AB *(note, that the abbreviation is used in case histories to designate some other terms also)*	верхушечный толчок. Сокращение также используется в историях болезни для обозначений: 1) [abdominal bloating] метеоризм, вздутие живота; 2) [aspiration biopsy] аспирационная биопсия
aortic aneurysm	аневризма аорты
aortic insufficiency	недостаточность клапана аорты, аортальная недостаточность
aponeurosis	апоневроз
arteritis	артериит (воспаление стенки артерии)
atherogenic	атерогенный, способствующий развитию атеросклероза

atrium	1) полость; пазуха; синус; 2) предсердие; 3) отверстие бронхиолы (открывающееся в альвеолы); 4) преддверие; 5) входные ворота инфекции
bending	1) сгибание; изгибание; 2) изгиб; кривизна; изогнутость
brachial	плечевой
buccal	1) буккальный, относящийся к щеке; щечный; трансбуккальный (о методе введения лекарственного средства) 2) внутриротовой
carotid	сонная артерия
cartilage	хрящ
cervical	цервикальный (1. относящийся к области шеи; 2. относящийся к шейке какого-либо органа)
compression	компрессия, сдавление; сжатие
cornea	роговица
corneal	роговичный, корнеальный
dilatation = dilation	дилатация, расширение
Dupuytren's contracture, palmar sclerosis	контрактура ладонного апоневроза, контрактура Дюпюитрена, синдром Дюпюитрена
fusiform, spindle-like	веретеновидный, веретенобразный
gland	железа
gout	подагра
Graves' disease	диффузный токсический зоб, базедова болезнь, *тж.:* болезнь фон Базедова, болезнь Грейвса, болезнь Парри, болезнь Флаяни
Horton's disease	гистаминовая цефалгия, синдром Хортона, мигрень Хортона, височный артериит
hypercholesterolemia = hypercholesteremia	гиперхолестеринемия
hyperlipoproteinemia, hyperlipoproteidemia	гиперлипопротеинемия, гиперлипопротеидемия
jugular	1) яремный; 2) яремная вена
jugular fossa	яремная ямка, надгрудинная ямка
lid	веко
malady	болезнь, заболевание
microangiopathy	микроангиопатия
moniliform	монилиформный, бусообразный
musculus sternocleidomastoideus	грудино-ключично-сосцевидная мышца
Musset's sign	симптом Мюссе (синхронное с ритмом сердца покачивание головы вперед-назад; признак недостаточности клапана сердца)
nasal	носовой, назальный
navel	пупок
nodous = nodular	нодозный, узелковый; узловатый
occlusion	обтурация; окклюзия; закупорка
palmar	ладонный

periarteritis	периартериит (воспаление адвентиции артерии)
plantar	подошвенный, плантарный
plaque	1) бляшка; 2) тромбоцит, кровяная пластинка, бляшка Бицоцеро
popliteal	подколенный
portocaval shunt	портокавальный анастомоз
pulsation	пульсация, биение
radial	1) лучевой (напр. о кости); 2) радиальный (напр. о расположении волокон)
reticulum	(*pl.*: reticula) 1) ретикулум, (тонкая) сеть, сеточка, сетчатое строение; 2) нейроглия; 3) ретикулярная ткань
systole	систола сердца
temporal	1) височный; 2) временный, преходящий
thrombotic	тромботический
thyroid	щитовидная железа; щитовидный
tricuspid valve	правый предсердно-желудочковый клапан, правый трехстворчатый клапан
umbilical	пупочный, умбиликальный
varicosity	1) варикозно расширенная вена; 2) варикозное расширение вен, варикоз
vasopathy	вазопатия
vermiform	червеобразный
xanthelasma	(плоская) ксантелазма, плоская ксантома
xanthoma	ксантома (патологическое скопление макрофагов, перегруженных липидами, в коже, сухожилиях и др. при нарушении липидного обмена)
xanthomatosis	ксантоматоз, экстрацеллюлярный холестериноз (*устар.*), болезнь Керля–Урбаха

3. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Jugular pit pulsation is observable in aortic aneurysms of various etiology. This may be accompanied by rhythmic pulsation over the thyroid cartilage (Oliver-Cardarelli's[1] symptom).

[1] William Silver Oliver, English military surgeon, born 1836; died 1908, Farnborough. Antonio Cardarelli, Italian physician, born March 29, 1831, Civitanova del Sannio; died January 8, 1926.

The pulsation of peripheral vessels is normally invisible. Under pathologic conditions the vessels may display abnormal pulsation and some visual changes. The vermiform bending of temporal arteries is observed in essential hypertension and atherosclerosis. The moniliform (beaded) temporal arteries may be in nodous periarteriitis and Horton's disease (temporal arteriitis).

In neck area of healthy persons only carotid arterial pulsation may be observed, usually synchronous with AB. In aortic insufficiency a noticeable abnormal pulsation of cervical arterial vessels is observed. A patient may even nod and shake involuntarily, simultaneously with the heartbeats (de Musset's[1] symptom). Nodding may accompany aortic aneurysm as well. In these conditions other big vessels (brachial, radial, popliteal etc) may also pulsate, which is a characteristic of aortic insufficiency and atherosclerosis. In aortic insufficiency capillary pulsation in nail areas is also typical. While examining the cervical area, a doctor should bear in mind a possible thyroid gland enlargement, because it is helpful in Graves'[2] disease diagnosis if tachycardia is also present. On examination the veins may display the condition of dilatation, overfilling and even pulsation. Cervical venous pulsation is peculiar exclusively for tricuspid valve insufficiency due to blood regurgitation into right atrium and, consequently, into venae cavae in right ventricle systoles. In this case ("Homo pulsans") venous pulsation may be even in peripheral veins (hands). There is also simultaneous liver pulsation (sometimes, visible).

Cervical veins swelling may result from heart congestion, but also from tumoral compression of the vein(s) or their thrombotic occlusion. Commonly it is accompanied by local edema. In congestive liver (portal venous hyperemia) the net of distended umbilical veins is observable in navel region due to porto-caval anastomoses ("caput Medusae"). To distinguish between arterial and venous cervical pulsation, remember that the former is situated to the medium from musculus sternocleidomastoideus, but the latter — to the exterior from this muscle. In leg phlebopathies varicosity and venous swelling with the signs of inflammation are usually obvious. After elevating lower extremities the veins usually abate due to improvement of blood outflow. On examining the eyes, mind the presence of corneal lipid arch (senescent arch of cornea), typical for certain atherogenic hyperlipoproteinemias and atherosclerosis. The xanthomas of lids (xanthelasmas) may be revealed in atherosclerosis, especially in familiar inherited hypercholesterolemia (HLP IIa, according Fredrickson's[3] classification). Xanthomatosis with eruptive, plane or tendinous xanthomas of various localization is peculiar to

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[1] Louis Charles Alfred de Musset, French Romantic poet and playwrighter, born December 11, 1810, Paris; died May 2, 1857, Paris. The symptom was first noticed in him in 1842 by his brother, physician Paul de Musset (1804–1880).

[2] Robert James Graves, Irish physician, born March 27, 1797, Dublin; died March 20, 1853, Dublin.

[3] Donald Sharp Fredrickson (also self-spelled: Frederickson) (August 8, 1924 — June 7, 2002), American pathologist, particularly known for his research of lipid and cholesterol metabolism disorders.

hyperlipoproteinemias of types I, IIa, II, III, IV and V. The nasal or buccal vascular reticulum may be observed in patients suffering from essential hypertension or diabetes mellitus complicated by microangiopathy. This phenomenon is observable also in alcoholic vasopathy. Scouring reveals numerous pigmented skin plaques with candle wax constituency, not removable by scouring, revealed in old persons with atherosclerosis. Finally, visual general examination must enable the doctor to conclude whether a patient has an excessive body mass. It is of great significance, because obesity is epidemiologically related to higher frequency of cardiovascular maladies, metabolic disorders and diabetes mellitus. Check, if possible, a patient's growth and weight by measuring, in order to calculate an exact surplus of body mass by means of special tables (Aub-Dubois'[1] tables or American insurance companies' tables). Palmar and plantar surfaces are also subject to careful examination. The contracture of palmar or plantar aponeuroses (Dupuytren's[2] contracture or palmar sclerosis) may be an indirect sign of atherosclerosis, non-insulin dependant diabetes mellitus or other metabolic disorders; palmar xanthomas may occur in certain atherogenic hyperlipoproteinemias. Joint deformation may be related to systemic autoimmune diseases of conjunctive tissue or gout, which are connected with cardiovascular involvement or acceleration of cardiovascular disorder development.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *In what kind of aneurysms is jugular pit pulsation observable?*
2. *What may jugular pit pulsation be accompanied by?*
3. *Is the pulsation of peripheral vessels normally visible or invisible?*
4. *What may vessels display under pathologic conditions?*

4. Read the task card below.

Describe the main peculiarities of jugular pulse following the plan: **Under what conditions is jugular pulse observed?** **What kinds of pulsation are observed in people?** **What diseases may be accompanied by jugular pulse?**

5 Now, using the prompts from exercise 4, talk on the topic: JUGULAR PULSE.

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[1] Joseph Charles Aub, American physician, 1890–1973. Eugene Floyd Dubois, American physician, born 1882; died February 12, 1959, New York.

[2] Baron Guillaume Dupuytren, French surgeon, born October 5, 1777, Pierre-Buffière near Limoges, département Haute-Vienne; died February 8, 1835, Paris.

Module 5

PHYSICAL METHODS OF EXAMINATION IN CARDIOLOGY: "TOUCHING" EXAMINATION

Unit 5.1

PALPATION OF APEX BEAT

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

axillary	подмышечный, подкрыльцовый
cachexia	кахексия, общая атрофия
cardiac muscle	миокард, сердечная мышца
flatulence	метеоризм (вздутие живота вследствие скопления газов в кишечнике)
hypertrophic	гипертрофический
hypertrophy	гипертрофия (увеличение органа или его части)
mammary glands	молочные железы
medioclavicular	средне-ключичный, относящийся к срединной части ключицы
phalanx (phalanges)	фаланга пальца
phrenic	диафрагмальный, грудобрюшинный
pneumosclerosis	пневмосклероз, пневмофиброз, склероз легких
sternum	грудина
sympathicotonia	симпатикотония
thyrotoxicosis	тиротоксикоз
tricuspid	трехстворчатый (о клапане)
ventricular hypertrophy	гипертрофия желудочка; гипертрофия желудочков
visceroptosis = visceroptosia	спланхноптоз, висцероптоз, симптомокомплекс Гленара

The strength of AB is evaluated by palpation. It increases in left ventricular hypertrophy and decreases in pulmonary emphysema, obesity, chest edemas, and exudative pericarditis. Female patients with large mammary glands may have weakened AB on palpation. The compactness of cardiac muscle itself may be reflected in so called resistance of AB, evaluated also by palpation. AB firmness is noticeably increased in any kind of left ventricular hypertrophy. So, the AB in left ventricular hypertrophy is high, resistant and strong. In extreme hypertrophy it is described as a dome-like one.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *When does a doctor start physical investigation?*
2. *What does physical investigation include?*
3. *What does palpation allow a doctor to estimate?*
4. *How should a doctor position himself while palpating?*

4. Read the task card below.

Describe the main peculiarities of palpation following the plan:
What is palpation?
How is palpation carried out?
What results can be obtained by percussion?

5. Now, using the prompts from exercise 4, talk on the topic: PALPATION

Unit 5.2
PULSE CHECKING

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

adhesive pericarditis	спаечный перикардит, адгезивный перикардит, слипчивый перикардит
aortic insufficiency	недостаточность клапана аорты, аортальная недостаточность
aortic stenosis	стеноз устья аорты, аортальный стеноз
aphonia	афония (отсутствие звучности голоса)

arrhythmia	аритмия (сердца)
atrioventricular	атриовентрикулярный, предсердно-желудочковый
bradycardia	брадикардия (пониженная частота сердечных сокращений)
calcinosis	кальциноз, обызвествление, кальцификация
capillary pulse	капиллярный пульс, прекапиллярный пульс Квинке, симптом Квинке
cardiac	сердечный
cardiac conduction system	проводящая система сердца
coarctation	коарктация (аорты), сужение, стеноз; стриктура (сосуда, канала, отверстия)
collapse	коллапс (острая сосудистая недостаточность)
extrasystole	экстрасистола
hyperthyroidism	гипертироз, гипертироидизм (синдром, обусловленный избыточностью действия гормонов щитовидной железы)
hypotension	гипотензия, гипотония
induration	индурация, уплотнение (органа или ткани); затвердение, отвердение
intracranial	интракраниальный, внутричерепной
lesion	повреждение, поражение, патологическое изменение
nailbed	ногтевое ложе
nervus	нерв
neurosis, neurotic disorder	невроз
nodular = nodous	нодозный, узелковый; узловатый
periarteritis	периартериит (воспаление адвентиции артерии)
posterior	задний
recurrence	рецидив
respiration rate	частота дыхания
rupture	разрыв; прободение; перфорация
sclerotic	склеротический
septum (*pl.*: septa)	перегородка
sinoatrial node	синусно-предсердный узел, синусный узел, узел Киса–Флека
sinoauricular block	синоаурикулярная блокада
skull	череп
stratification	расслоение
subclavicular	подключичный
sympathetic	симпатический (*относящийся к* симпатической нервной системе)
sympathomimetic	симпатомиметическое средство; симпатомиметический
Takayasu's disease	болезнь отсутствия пульса, болезнь Такаясу
thread pulse	нитевидный пульс
thyrotoxicosis	тиротоксикоз

upstream	перед, выше по течению (о кровеносных сосудах)
vagus = vagus nerve	блуждающий нерв

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Palpation gives information concerning a patient's pulse. The pulse may be checked in any palpable artery, but on account of convenience it is common to determine it on the the radial artery. However, in some arterial diseases radial artery pulse is not palpable. There may be also some anomalies of radial artery position. Hence, bilateral simultaneous pulse palpation on both radial (brachial, carotid or low extremities) arteries is necessary. Otherwise, the doctor may miss certain important diseases, manifested in the lack of pulse on the side of injury (upstream thromboembolism of the artery examined, Takayasu's[1] disease, aortic coarctation, peripheral atherosclerosis, compression of the artery with tumor or enlarged lymph node, thoracic aortal aneurysm, pneumothorax, excess of fluid in pleural cavity, sugar diabetes with asymmetric lesion of low extremities' arteries etc). Sometimes the pulse is weakened in the left radial artery in patients with mitral stenosis due to megaloatrium (left atrial enlargement), because the atrium compresses left subclavicular artery.

At the same time, due to recurrent nerve compression, aphonia may occur (Ortner's symptom I[2]). Along with estimation of even or unsteady character of the pulse, the following pulse characteristics may be described: frequency, rhythm, pulse curve shape, hardness.

To check pulse frequency it is important to count pulses during 1 minute. It is obligatory in unsteady pulse or arrhythmia. In steady rhythmic pulse it is possible to count pulse beats per 30 seconds and double the result. Normal pulse frequency in adults is 70–76 bpm. In children it is higher and is closely dependant on age: the younger the child, the higher the rate. The 4:1 equation between the pulse and respiration rates is a normal index of great importance. In pneumonia it may be disturbed, specifically in children.

Physical strains, excitement, overeating, hot weather — all these factors increase pulse rate. Tachycardia is pulse rate accelerated upper than 90 bpm. Pulse rate may be accelerated due to: pain of different origin (stenocardia, infarction, pulmonary artery thromboembolism,

[1] Mikito Takayasu, Japanese ophthalmologist, 1860–1938, Kanazawa.

[2] Norbert Ortner, Austrian internist, born August 10, 1865, Linz; died 1935.

rupture or stratification of aortic wall), various neuroses (including cardioneurosis), palsy of vagus nerve or excitation of stimulatory sympathetic (Pavlov's[1]) cardiac nerve, paroxysmal tachycardia, hyperthyroidism, heart failure, belladonna and sympathomimetic drugs overdose, sometimes digitalis overdose.

Pathologic bradycardia with pulse rate < 60 bpm may be caused by cardiac conductivity system disorders (blockades). It may be observed in sinoatrial node weakness, sinoauricular block, atrioventricular block of different nature (related to myocarditis, cardiosclerosis, myocardial infarction with the injure of septum and posterior wall, digitalis overdose etc). Complete atrioventricular block produces cerebral circulation disorder due to rare pulse, which is known as Morgagni — Adams — Stokes'[2] syndrome.

Stroke, caused by hypertension, may produce either bradycardia or tachycardia, as well as brain tumors, aneurysms or other sizable processes within the skull, provoking intracranial fluid hypertension. Pulse is regular and steady if the intervals between the pulse waves are stable, otherwise it is irregular (arrhythmic). In arrhythmia, the pulse should be checked simultaneously both on peripheral artery and heart (by means of stethoscope). It may occur that the number of heartbeats outnumber number of pulse beats. This phenomenon is referred to as pulse deficit, and it may occur only in absolute arrhythmia (ciliary arrhythmia, irregular pulse). Other arrhythmias are not accompanied by this phenomenon. Deficit of pulse is not observed in extrasystoles.

Respiratory arrhythmia is accompanied by increased pulse rate during inspiration and decreased in an expiratory phase. Paradoxical pulse is the pulse disappearing on expiration (as in exudative or adhesive pericarditis). Hard (firm) pulse — *pulsus durus* — is characteristic of hypertension and artheriosclerosis. Soft pulse (*pulsus mollus*) is effortlessly compressible and typical for decreased vascular tone (collapse, shock, hypotension).

Almost non-palpable pulse can be named pulsus filiformis — a thread pulse; if pulse waves show rapid elevation and quick fall, we call that "pulsus celer (Corrigan's[3] pulse)" and high pulse or "*pulsus altus*". This kind of pulse is typical for aortic insufficiency and thyrotoxicosis. On the contrary, slow pulse (pulsus tardus) with small filling (*pulsus parvus*, or small pulse) is characteristic of aortic stenosis and advanced artheriosclerosis. Gliding movement of fingers along the palpable vessel may reveal the peculiarities of vascular wall. Perceptive induration of the whole artery may be in atherosclerosis, especially in M`nckeberg's[4]

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[1] Ivan Petrovich Pavlov (born: September 14, 1849, Ryazan'; died: February 27, 1936, Leningrad), Russian physiologist, psychologist, and physician, Nobel Prize winner of 1904 in Physiology or Medicine.

[2] Giovanni Battista Morgagni, Italian anatomist and pathologist, born: February 25, 1682, ForlX; died : December 5, 1771, Padua. Robert Adams, Irish surgeon, born: 1791, Dublin; died: January 13 or 16, 1875, Dublin. John Stokes — see footnote 2 above, page 161.

[3] Sir Dominic John Corrigan, Irish physician, born December 1, 1802, Dublin; died February 1, 1880, Dublin.

[4] Johann Georg Mönckeberg, German pathologist, born August 5, 1877, Hamburg; died March 22, 1925, Bonn.

sclerotic calcinosis. Nodular separated indurations are typical for nodous periarteriitis. In high arterial hypertension pulse can hardly be compressed. Capillary pulse, rhythmic changes of nailbed color — pallor in diastole and redness in systole, is observable in aortic insufficiency (Quincke's[1] symptom).

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What kind of information does palpation give?*
2. *On what artery is it common to check pulse?*
3. *In what diseases is radial artery pulse not palpable?*
4. *Are there any anomalies of radial artery position?*

4. Read the task card below.

Describe the main peculiarities of pulse check following the plan: **What is the most suitable way to check the pulse?** **What is important for checking pulse frequency?** **What dideases can be diagnosed by pulse check?**

5. Now, using the prompts from exercise 4, talk on the topic: THE PULSE CHECK.

Dialogue 2. At the doctor's office.

4. Without looking into the dialogue listen to the recording.

Say what information you have gathered.

Listen to the dialogue again.

Now, read the dialogue silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the dialogue aloud, trying to imitate the intonation.

[1] Heinrich Irenaeus Quincke, German internist, born August 26, 1842, Frankfurt an der Oder; died May 19, 1922, Frankfurt am Main.

Patient. Excuse me, doctor. May I come in?

Doctor. Come in, please. Well, what's your family name? How old are you?

P. My family name is Petrova and I am 70.

D. What's the problem? I understand you're gasping for breath.

P. Doctor, you see I had been relatively well until yesterday, for my age, I would say. Well, I went shopping yesterday and... When I was approaching my home, I started gasping for breath. I had never felt like that before. I even could not walk, sat down on a bench in front of the house for a while to catch my breath. After that I could hardly walk upstairs as I live on the third floor. There is no elevator. I had had a bad night and I couldn't lie down and sleep as I was gasping for breath. So I slept keeping a sitting down. Today it was only with great difficulty that I came to see you. What's wrong with me?

D. Now tell me, please, do you have palpitations?

P. I am sorry I have forgotten to tell you about it. I used to have high pulse before. And yesterday I suddenly got irregularity in my heart beat and immediately it was difficult to breath.

D. Have you suffered from high blood pressure before? Have you had angina?

P. I sometimes have had heartaches. Valocordinum[1] drops and Validolum[2] were helpful. My blood pressure has always been normal. But I used to feel pain in my joints when I was young and doctors found heart valve disease, probably aortic one. From time to time I was given Bicillinum[3] injections. But everything calmed down eventually.

D. Strip to the waist, please. I'd like to check your heart. Your heart is increased by 3 centimeters to the left. I can hear coarse systolic cardiac murmur over the aorta. The beats of your heart are not regular. Sometimes they accelerate and sometimes they slow down and your heart seems to come to a standstill. This is cardiac fibrillation.

P. And why it is that I immediately felt like a sick, disabled person?

D. In this arrhythmia your heart beats irregularly. You have pulmonary congestion. It causes dyspnea and the deficiency of oxygen in your organism. Hypoxia develops and due to it your organism, especially your brain, suffers. Therefore you suddenly felt so unwell. The patients with ciliary arrhythmia or cardiac fibrillation can tell you a sudden onset of dyspnea within a minute. And this is an attack of cardiac asthma. By the way, is it more difficult for you to inhale or to exhale?

P. It is more difficult for me to breathe in than to breathe out, Doctor.

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[1] German commercial name. ATX code: N05C B02, mixture of Phenobarbitalum — 2 g, Ethylbromisovaleratum — 2 g, Oil of Hops — 0,2 g, Oil of Peppermint — 0,14 g, solvent added up to 100 ml

[2] Russian commercial name. International non-patented name: Menthol solution in menthyl isovalerate.

[3] Russian commercial name. There is no international non-patented name for this drug; its acting substance is Benzylpenicillinum (Penicillin G of British Pharmacopoeia), Western synonyms: Benzathine benzylpenicillin, Benzylpenicillin procaine.

D. That is all. You have so-called inspiratory dyspnea, which is the sign of heart failure.

P. Well, doctor. Are you sure that this is cardiac fibrillation?

D. Your heart throws into the aorta different portions of blood with this arrhythmia going on. Therefore your pulse rate is always less than the number of heart beats. I have heard 112 beats of your heart per minute, and the pulse on your hand, which is less frequent, equals 80 beats. This difference of 32 beats is called pulse deficit, and it is observed only in cardiac fibrillation. Now, can you go to the ECG ward, please? As soon as your electrocardiogram has been done, do come back to see me: I will prescribe you additional checkup and treatment. I will prescribe you antiarrhythmic drugs combined with cardiac glycosides. The arrhythmia you have can sometimes disappear even without treatment. But you should not wait until it disappears by itself, otherwise you will have edemas. Apparently, the reason of this arrhythmia is your aortic valve disease you forgot about a long time ago. It often reminds of itself characteristically in middle age.

5. Translate into English.

I. Что с Вами случилось?

Я вижу, что Вы задыхаетесь.

Скажите, пожалуйста, а нет ли у Вас сердцебиений?

У Вас не было раньше повышения кровяного давления? Не было стенокардии?

Разденьтесь до пояса, пожалуйста.

Я хочу послушать Ваше сердце.

II. Сердце расширено влево на 3 см. Выслушивается грубый систолический шум над аортой. Сердцебиения нерегулярные: то замедляются, то ускоряются, а сердце временами будто замирает. Это мерцательная аритмия. При этой аритмии сердце сокращается нерегулярно. Кровь в легких застаивается. Это вызывает одышку и дефицит кислорода в организме. Развивается гипоксия, от которой страдает весь организм, особенно головной мозг. Пациенты при мерцательной аритмии с точностью до минуты могут назвать время возникновения внезапной одышки. Это — приступ сердечной астмы.

III. При мерцательной аритмии сердце выбрасывает в аорту разные порции крови. Тогда частота пульса всегда меньше, чем число ударов сердца. Эта разница называется дефицитом пульса и наблюдается только при мерцательной аритмии.

Больного направляют в электрокардиографический кабинет, снимают ЭКГ, назначают дополнительные обследования и лечение.

Причиной мерцательной аритмии может быть аортальный порок сердца, который часто напоминает о себе в пожилом возрасте.

Unit 5.3
PERCUSSION

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

auricle	ушко (предсердия)
cardiac dullness = dulness	тупость (перкуторного звука); приглушенность, притупленность
cardiomyopathy	кардиомиопатия
cardiosclerosis	кардиосклероз
hemopericardium	гемоперикард (кровь в полости сердечной сорочки)
hypertrophy	гипертрофия (увеличение органа или его части)
parasternal	парастернальный
plessimeter	плессиметр (приспособление для перкуссии)
pneumopericardium	пневмоперикард (наличие газа в перикардиальной полости)
relative cardiac dullness border (RCDB)	относительная граница сердечной тупости
superficial cardiac dullness border (SCDB)	поверхностная граница сердечной тупости

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Percussion is applied to determine the heart's size, configuration, position and big vessel dimensions. It is convenient to percuss a patient standing upright, but a heavy patient's heart may be percussed in a lying position. Heart borders are usually slightly broader when a person is lying on the bed.

Percussion may be either mediated (through special plessimeter or finger-plessimeter) or immediate (by Ebstein[1], by Obraztsov[2]).

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[1] Wilhelm Ebstein, German internist, born November 27, 1836, Jauer (Schlesien); died October 22, 1912. Göttingen.

[2] Vasiliy Parmenovich Obraztsov, Russian internist, born January, 1st 1849, Gryazovets, Vologodskaya region; died December 14th, 1920, Kyev.

There are 6 laws of percussion:

1) Finger-plessimeter should be tightly attached to the thorax

2) Finger-plessimeter should always be situated parallel to the organ border, as the doctor sees it.

3) While checking relative cardiac dullness, a doctor should percuss by finger strikes of medium strength.

4) When checking superficial cardiac dullness (over the portion of heart, non-covered by the lungs and directly attached to the thoracic wall) use the smoothest and lightest percussion (after Goldscheider[1] or after Plesch[2]).

5) The courses of percussion always change from clear sound to a dull one (in cardiology practice — from the lungs to the heart).

6) The organ border as determined by means of percussion is considered to run along the external side of the plessimeter finger.

The sequence of percussion is as fololows:

1) The right cardiac dullness border,

2) The left cardiac dullness border,

3) The upper cardiac dullness border.

Relative dullness is determined before superficial one. At first, the doctor determines the upper right phrenic cupola border, then raises plessimeter finger 1 intercostal space up and percusses the right cardiac border until the transition of clear sound into a dullish one.

The normal right cardiac border in healthy individuals is revealed 1–1,5 cm to the right from the right sternal margin. It is formed by the right atrium. The left border of relative cardiac dullness is examined after the determination of apex beat. Usually, AB is placed on the left relative dullness border.

If AB is not palpable, plessimeter finger is placed in the 5th intercostal space and percussion goes parallel to the expected left superficial cardiac dullness border starting from the medial axillary line. Normally, a relative cardiac dullness border is found 1–2 cm to the medium from the medioclavicular line, being formed by left ventricle.

To determine the upper relative cardiac dullness border (RCDB), it is necessary to place plessimeter finger near the left sternal edge parallel to the ribs, and percuss starting from 1 space downwards. Normally, the left border of relative cardiac dullness is at the upper margin of the 3rd rib, being formed by the left auricle and the pulmonary artery cone. Examining RCDB at different points and different spaces, the doctor may obtain the outline of heart configuration. It is changeable, depending on cardiac pathology. In mitral valve diseases all cameras of the heart are dilated, producing typical "mitral" heart configuration. In aortic valve diseases configuration is "aortic" (resembling the profile of sitting duck or a top boot). In exudative pericarditis the heart is of trapezium shape, narrowing up towards the vascular bundle.

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[1] Johannes Karl August Eugen Alfred Goldscheider (1858–1935), German internist, worked at Berlin.

[2] Janos (John) Oscar Plesch (1878–1957) — Hungarian, later — German and English internist.

After the examination of RCDBs following the same procedure the superficial cardiac dullness borders (SCDB) are determined. The right SCDB normally runs along the left sternal edge, the left one runs along the left RCDB or is positioned 1 cm to the medium from it. The upper normal SCDB is under the cartilage of the 4th rib. The percussion zone of the vascular bundle is measured in the 2nd space from both sides. As it is difficult use subtle percussion. The normal width is about 5–6 cm. In aortic diseases it is considerably broadened. Mind that you should not confuse expanded bundles with enlarged mediastinal lymph nodes in pulmonary tuberculosis.

SCDB and RCDB may vary depending on phrenic position, somatotype, cardiopulmonary pathology. In pulmonary emphysema the borders are squeezed, as well as in asthenics (suspended, hanging or drop heart, Cor pendulum, cardioptosia). RCDB is expanded in hypersthenic somatotype, ascites, pregnancy, flatulence, but most frequently — in cardiac dilatation and (to a minor extent) — due to cardiac hypertrophy itself.

Right RCDB is broadened in dilatation of right atrium or/and ventricle (tricuspid disease, pulmonary artery stenosis resulted from Ayerza's disease or congenital reasons) as well as in cor pulmonale.

Left RCDB is dilated in left ventricle dilatation and hypertrophy (mitral insufficiency and aortic valve diseases, arterial hypertensions, left ventricular aneurysm). It is accompanied by palpable pathologic precardial pulsation of a peculiar kind in the cardiac area. In advanced heart valve diseases, specifically, tricuspid ones, there is total dilatation of heart borders in all directions (Cor bovinum).

Cor bovinum sometimes accompany advanced cardiosclerosis and dilatation cardiomyopathy, usually against the background of severe heart failure and breathlessness. RCDB is displaced upwards in mitral stenosis due to dilatation of left atrium ("parasternal dullness symptom", when not only the border is dilated, but also cardiac waist thickens, or even protrudes). SCDB is dilated totally in exudative pericarditis, hydropericardium and hemopericardium, usually together with RCDB dilatation. SCDB area is increased in mediastinal tumors, pulmonary margins squeezing, and also in bending ahead. It is, vice versa, decreased on deep inhalation, emphysema (both pulmonary and subcutaneous paracardial one), in pneumothorax, pneumopericardium. Paracardial subcutaneous emphysema may occur as a complication of left subclavicular artery catheterization when the lung is injured.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What is percussion applied for?*
2. *In what position is it convenient to percuss a patient?*
3. *In what position may a critical patient's heart be percussed?*

4. Read the task card below.

Describe the main peculiarities of percussion following the plan: **What is percussion applied for?** **What are the laws of percussion?** **How do SCDB and RCDB vary?**

5 Now, using the prompts from exercise 4, talk on the topic: PERCUSSION.

Module 6

OBJECTIVE PHYSICAL EXAMINATION IN CARDIOLOGY: SOUND DATA

Unit 6.1

AUSCULTATION OF HEART SOUNDS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

anatomical axis	анатомическая ось
aperture	1) апертура, отверстие, щель; 2) диаметр объектива микроскопа
atherosclerosis	атеросклероз
atria	предсердие
atrial	предсердный
atrioventricular	атриовентрикулярный, предсердно-желудочковый
cachexia	кахексия, общая атрофия
diastolic	диастолический
dystonia	дистония, нарушение тонуса
dystrophy	дистрофия, гипобиоз, дегенерация, перерождение, *тж.:* голодное истощение (алиментарная)
ergometry	эргометрия; динамометрия
exhaustion	истощение; утомление, крайняя усталость
extrasystole	экстрасистола
extreme fatigue	состояние крайнего утомления
hydropericardium = hydropericarditis	гидроперикард, водянка перикарда
hyperthyroidism	гипертироз, гипертироидизм (синдром, обусловленный избыточным влиянием гормонов щитовидной железы)
hypertrophy	гипертрофия (увеличение органа или его части без увеличения числа клеток в них)
kyphoscoliosis	кифосколиоз
mitral stenosis	стеноз левого атриовентрикулярного отверстия, митральный стеноз
mitral valve	левый предсердно-желудочковый клапан, левый двустворчатый клапан, левый митральный клапан

neurocirculatory	нейроцикуляторный
opening snap	щелчок открытия (клапана сердца)
orifice	отверстие; устье; вход; проход
ostium	отверстие (вход в полый орган или канал)
pneumonia	пневмония, воспаление легких
presystolic	пресистолический
sternal	стернальный, грудинный
systolic	систолический
tricuspid valve	правый предсердно-желудочковый клапан, правый трехстворчатый клапан
ventricle	желудочек (сердца)
xiphoid process = xiphoid appendix	мечевидный отросток (грудины)

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

The heart is to be auscultated, if possible, both in vertical and horizontal positions. When a patient's general condition permits, it is desirable also to perform auscultation after special loading tests (running, bicycle ergometry, knees-bend squatting etc.) Heart auscultation by the stethoscope usually reveals 2 rhythmic reiterating tones (heart sounds). Immediate auscultation by ear, as a rule, makes also the 3rd sound audible (following the 2nd one), which is much weaker than the other sounds and presents not only an audible, but also a tangible phenomenon. The 1st heart sound is named systolic, and 2nd is diastolic, and the space between them is a systolic pause (normally, 0,2 sec); the space after the 2nd sound, before the 1st sound is a diastolic pause (normally 0,43 sec). The heart sounds are complexes, or combinations of several audible constituents.

The 1st sound is the effect of the following 8 factors: simultaneous contraction of both ventricles, coincident closure of both mitral and tricuspid valves, synchronous opening of both aortic and pulmonary artery orifices, and distension of their orifices; muscle component, caused by both left and right atria contraction. The 2nd sound is formed by two phenomena: simultaneous closure of aortic and pulmonary artery crescent valves.

The 3d sound is produced by the injection of blood in the beginning of diastole from the atria into the emptied, dilating ventricle cavities, distending their walls. The 1st sound lasts 0,11–0,12 sec, the 2nd — 0,7–0,8 sec. The 3d sound is considered to be physiologic up till 25 years

of age, and is more common in nervous persons and in heavy physical strain. In left ventricular insufficiency the 3rd sound is calculated as abnormality. Its duration is 0,2–0,6 sec. As a result of its pathologic appearance, a so called "gallop rhythm" may arise. Sporadically, in atrial hypertrophy a presystolic 4th heart sound may arise. This produces the phenomenon of presystolic "gallop rhythm". It is a matter of proficiency to distinguish the constituents of the sounds, because they vary depending on the type and location of a particular pathology.

It is advisable to auscultate every sound at the point closest to the heart ostium, which serves a source of this particular audible phenomenon. The mitral valve's projection is at the point of the 3rd left rib cartilage attachment to sternum. The tricuspid valve's projection is in the middle of sternum on the line that joins the attachment points of the 3rd right and the 5th left ribs. The projection of the aortic valve is at the level of the 3rd ribs' attachment in the middle of aorta. The projection of the pulmonary artery orifice is in 2nd intercostal space near the left margin of sternum. All the projections listed are situated in the closest neighborhood . Thus, it is necessary to choose the points of auscultation so as to provide maximal loudness for the sound under examination and weaken noticeably all other sounds. That's why the stethoscope is positioned not exactly at the points of projections, but at some special loci. Mitral sounds are spread well to cardiac apex. Because of this, at first the doctor palpates the AB, then places the stethoscope exactly on AB area. Normally, the 1st sound is louder than the 2nd at the apical point. The 1st sound coinciding with carotid pulsation and AB is named systolic sound. It is better to auscultate the tricuspid valve over the base of processus xiphoideus. For the crescent valves of the aorta, it is better to listen in right 2nd space near sternum. For the 2nd, diastolic sound, it is better to auscultate over the base of the heart, at the projection point of aortic crescent valves.

The classic point for aortic valve auscultation is Botkine–Erb[1] point. It is located at the focus of intersection between the anatomical heart axis and the left sternal edge. Sounds are more pronounced in a vertical position. The sound at a certain point is referred to as an accent. The accent of 1st sound over AB and accents of 2nd sound over the aorta and the pulmonary artery are of great clinical significance. The accent of the 1st sound over the apex is a classical and, sometimes, single sign of mitral stenosis. The same accent appears in extrasystoles, which produce a clapping (cracking) 1st sound. The 1st sound accent is caused by low diastolic filling of the left ventricle due to the narrowing of the atrioventricular aperture or resulting from premature contraction in response to extraordinary extrasystole. Aortic accent of the 2nd sound is related to the systematic hypertension of the greater circulation, but may also occur in decreased aortic wall elasticity due to either atherosclerosis or aortitis of the initial aortic portion, producing local blood

[1] Wilhelm Heinrich Erb, German neurologist, born November 30, 1840, Winnweiler, Palatinate; died October 21, 1921. S. P. Botkin — see footnote 1 above, p. 153.

pressure elevation. In noticeable atherosclerosis, the 2nd sound accent is of metallic character. In aortic insufficiency, the 2nd sound accent is not audible, in spite of high systolic pressure, due to incomplete closure of squeezed valves and their deformities, although sometimes the 2nd sound does not change. Somatoform autonomic dysfunction, especially manifested in panic attacks, sporadically may be accompanied by an aortic accent of the 2nd sound. The 2nd sound accent over pulmonary artery is characteristic of lesser circulation hypertensions (mitral valve diseases, pulmonary emphysema, severe kyphoscoliosis, pneumoniae, marked pleural exudates). It is peculiar to all situations of lesser circulation hardened, but right ventricle strength remaining sufficient.

The sounds are weakened in obese patients and females with big breasts, in pulmonary emphysema, cachexia, exhaustion, extreme fatigue, myocardial diseases (myocarditis, dystrophy, and some cardiomyopathies). The cardiac muscle in these conditions is contracting weaker and slower. The 2nd sound over large vessels may be weak in shock and collapse. Sometimes it is not audible at all. A weakened 1st sound over the apex is displayed in atrioventricular valves insufficiency, surplus diastolic filling of ventricles, slower ventricle's contraction due to hypertrophy, myocarditis, cardiosclerosis, cardiomyopathies. In these cases 1st sound becomes dull. Hydropericardium makes the heart sounds very dull. In slim subtle individuals all heart sounds are amplified, as well as in pneumosclerosis, exercises, stress, under the influence of excitatory drugs, in hyperthyroidism, fever, and anemia. A thin chest wall, as well as the existence of some sounding boards (cavities filled with air located in the vicinity of the heart: lung caverns, pneumothorax, gut gases, big phrenic hernias etc) both lead to enhancement of heart sounds. Splitting of heart sounds (distinguished from the 3rd and the 4th audible sounds) is not uncommon. Splitting of the 1st sound is a result of a non-synchronous contraction of the right and left ventricles. This may result from an isolated left or right bundle branch block (His' bundle branch blockade), and also from a separate hypertrophy of one ventricle. The 2nd split sound is typical of the non-synchronous end of left and right ventricle systole, causing a lag between aortic and pulmonary artery crescent valves closure. A striking example is mitral stenosis, when the left ventricle's systole become shorter than the right one, and the aorta is closed earlier than the pulmonary artery. Sound splitting may be real and apparent, produced by additional sounds. An example of additional sound is the sound of mitral valve opening, which arises soon after the 2nd sound, imitating its splitting. Unlike the real split of the 2nd sound, this additional sound is audible best of all not over the heart base, but over the apex. The traditional name of this phenomenon is "mitral opening snap". The 1st sound accent over AB plus a mitral opening snap compose a classic melody of mitral stenosis. The 3rd and the 4th sounds when enhanced as it was mentioned above, produce a three tact rhythm of the heart — "gallop rhythm". Gallop rhythm is the heart's appeal for help. It arises in noticeable weakness of myocardium (infarction, non-compen-

sated valve diseases, myocarditis, severe cardiosclerosis, hypertonic disease, chronic nephritis).

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *In what positions is the heart to be auscultated?*
2. *After what tests is it desirable to perform auscultation when a patient's general condition permits?*
3. *How many rhythmic reiterating tones does heart auscultation by stethoscope usually reveal?*
4. *Which sound does immediate auscultation by ear identify as a rule?*

4. Read the task card below.

Describe the main peculiarities of heart auscultation following the plan:

How is a patient's heart auscultated?
What are heart sounds?
How are heart sounds auscultated?

5. Now, using the prompts from exercise 4, talk on the topic: HEART AUSCULTATION.

Unit 6.2
AUSCULTATION OF HEART MURMURS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

autoimmune	аутоиммунный
basal	базальный, расположенный у основания
cardiac	1) сердечный; 2) кардиальный
conjunctive tissue, connective tissue	соединительная ткань
dehydration	обезвоживание, дегидратация
dilatation	дилатация, расширение
Dressler's syndrome	постинфарктный синдром, синдром Дресслера

dullness = dulness	тупость (перкуторного звука); приглушенность, притупленность
extracardiac	некардиальный
fibrinous pericarditis	фибринозный перикардит
His' bundle	пучок Гиса
functional	функциональный
hypertonicity	1) гипертоничность; 2) повышение осмотического давления
lupus erythematosus, LE	красная волчанка, СКВ
metabolite	метаболит (продукт метаболизма)
metastasis	1) метастаз; 2) метастазирование
mitral insufficiency	митральная недостаточность, недостаточность левого предсердно-желудочкового клапана
mitral insufficiency	митральная недостаточность, недостаточность левого предсердно-желудочкового клапана
mitral stenosis	стеноз левого атриовентрикулярного отверстия, митральный стеноз
murmur	шум, аускультативный феномен
organic	1) относящийся к органу или органам; 2) связанный с жизнью организма; органический
papillary	сосочковый, папиллярный
pleuropericardial	плевроперикардиальный
recurrent	рецидивирующий, повторяющийся, возвратный
regurgitation	1) регургитация (перемещение содержимого полого органа в направлении, противоположном физиологическому, в результате сокращения его мышц); 2) отрыжка; срыгивание
renal failure	почечная недостаточность
rupture	разрыв; прободение
sc [subcutaneous]	п/к, подкожный
scapula (*pl.* scapulae)	лопатка
stenotic	стенозированный, вызванный стенозом, характеризующийся сужением
tricuspid disease	порок трехстворчатого клапана сердца
urate	урат, соль мочевой кислоты

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Heart noises — *murmurs* — are of great significance in ausculating diagnostics. They may be divided into three groups: organic, functional and related to dilatation.

Organic murmurs are derived from valve lesions and, quite naturally, may occur in heart diseases, papillary muscle rupture after myocardial infarction and acute mitral insufficiency.

Functional murmurs may be of cardiac and extracardiac origin, always un-related to valve diseases. They arise in anemia, sympathicotonia, and papillary muscle hypertonus in young persons with instability of autonomous nervous regulation. Presystolic functional murmur is sporadically audible in the projection point of the mitral valve in patients with aortic insufficiency (Flint[1]'s murmur).

The extracardial murmurs are: pericardial murmur due to fibrinous pericarditis and rough pericardial layers friction. An extracardial murmur may be smooth or coarse. This murmur occurs also in chronic renal failure due to pericardial deposits of urates and other metabolites, in metastases of tumors, in dehydration, systemic autoimmune non-organ specific conjunctive tissue diseases (lupus erythematosus), in post-infarction Dressler's syndrome, sometimes in thyrotoxicosis or heart contusion. Pericardial exudate leads to provisional disappearance of this murmur, but withdrawal of fluid allows it arise again. Its characteristics may be diverse (crisping, scratching, rustling etc.). This murmur is recurrent and most pronounced over SC dullness area, enhanced on bending forward and, commonly, does not spread anywhere. Pericardial murmur must be distinguished from pleuropericardial murmur, which is audible in RC dullness area. Pericardial murmur becomes audible when patients hold their breath, as distinguished from the pleuropericardial form. Heart murmurs are less musical than heart sounds, and more prolonged. It is a difficult phenomenon for verbal description, and that's why it is better to hear it at least for once. The origin of the murmurs is stenotic, because a laminar blood flow changes into a turbulent one due to the narrowing of lumen or orifices. The resulting vibration of heart valves and walls produces these murmurs. In valvular insufficiency, the murmur is caused by blood regurgitation via an improperly closed valve which quickens is the blood flow, making the murmur louder. In ghastly stenosis of the valve, as well as its extreme insufficiency, murmurs may be weak. They may be like seesawing, rubbing. musical, sometimes even with a squeak. Physical exercise provokes them due to acceleration of blood flow. Unlike the heart sounds, murmurs are stronger when a patient in a horizontal position. The following parameters of murmurs may occur: phase characteristic (systolic or diastolic). strength, duration, timbre, place of best auscultation, direction of spreading. All this is important in cardiac diagnostics. Murmurs, close to sounds, may be conducted for a long distance from the place of origin even to the back between scapulae (in aortic stenosis). Therefore, during auscultation it is important to find the point of their origin by means of placing the stethoscope nearer and nearer to the point where they are

[1] Austin Flint (senior), American physician, born October 20, 1812, Petersham, Massachusetts; died March 13, 1886, Brooklyn, New York.

the loudest. In general, valvular murmurs are best audible in valvular sounds auscultation points. But, aortic murmurs are best heard in the aortic aperture projection point (see above).

Pulmonary artery murmurs are extremely rare in adults, which is helpful in making diagnostics of aortic types. The murmurs are easily spread along the direction of blood flow, but not counter to blood flow. Therefore, the murmur of regurgitation in aortic insufficiency is not spread along the aorta. If the loudness of a murmur increases as the stethoscope moves to AB, this murmur is probably of mitral nature. If the loudness is maximal at the 3rd rib level in the middle of sternum, a murmur is of aortic origin. In combined heart diseases murmurs are certainly difficult to distinguish. The remnants of normal sounds facilitate the diagnosis. The murmur audible at the moment of valve closure is the evidence of its insufficiency. The murmur, corresponding with the moment of valve opening is related to its stenosis. Organic systolic murmur over AB is a sign of mitral insufficiency, but a diastolic murmur over the same point is a sign of mitral stenosis.

Valvular lesions may be combined with one or another predominant, forming the preponderance of proper murmur. In aortic valve diseases insufficiency is manifested in diastolic murmur, while in the case of stenosis — in systolic murmur. Tricuspid disease rarely produces murmurs. So, we may assume that an apical systolic murmur is the manifestation of mitral insufficiency, a basal systolic one is the manifestation of mitral stenosis. A diastolic murmur over the apex is a sign of mitral stenosis, and over the heart base is a sign of aortic insufficiency.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *How important are heart noises or murmurs in auscultating diagnostics?*
2. *How can murmurs be classified?*
3. *What are organic murmurs?*
4. *Where can organic murmurs occur?*

4. Read the task card below.

Describe the main peculiarities of murmurs following the plan: **Into what groups are heart murmurs divided?** **How are these groups characterized?** **What are the parameters of murmurs?**

5. Now, using the prompts from exercise 4, talk on the topic: MURMURS.

Unit 6.3

AUSCULTATION OF "FRÉMISSEMENT CATAIRE" AND VASCULAR MURMURS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

aortic insufficiency	недостаточность клапана аорты, аортальная недостаточность
backflow	1) обратный ток (жидкости), противоток; 2) рефлюкс; обратное забрасывание; ретроградный кровоток
coarctation	коарктация (аорты), сужение, стеноз; стриктура (сосуда, канала, отверстия)
congenital	врожденный
diagnosis	1) диагноз; 2) диагностика
diagnostics	учение о диагностике заболеваний
Duroziez double murmur	двойной шум Дюрозье
dysplasia	дисплазия (нарушение формирования ткани или органа)
femoral	бедренный
foramen (*pl.* foramina)	отверстие
iatrogenic	ятрогенный (о заболевании)
interventricular	межжелудочковый
jugular	1) яремный; 2) яремная вена
pansystolic	пансистолический (о шуме сердца)
septal	септальный, перегородочный
sternal	стернальный, грудинный
Traube's double tone	двойной тон Траубе, двойной симптом Траубе
vessel	сосуд

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

The murmurs may be not only auscultated, but also tangible. A cat's purring phenomenon ("frémissement cataire" — in French) is a sensation of trembling palpated, most often, in mitral stenosis, by laying the hand on the patient's chest. It is called so because it is similar to the sensation experienced by hand laid on the back of a purring cat.

Presystolic flutter formed near the mitral aperture is transferred to the thoracic wall. Palpatory trembling in the heart area is also perceptible in interventricular septal defects, accompanied by specific pansystolic murmur in the 4th space near the left sternal edge.

The diastolic murmur over the aortic base is observable not only in aortic insufficiency, but in an open foramen ovale joining the atria. It is a common case with approximately 25% of people having an open foramen ovale.

The vessels are also a subject of auscultation. Commonly, the doctor auscultates the vessels of medium caliber (carotid, subclavicular, femoral arteries). An artery is first palpated, then auscultated with a stethoscope. Be careful not to press the artery too firmly with the stethoscope, lest it should produce an artificial stenosis and create an iatrogenic murmur.

The murmurs of valve diseases, especially those of aortic stenosis, are often spread along the large and medium vessels. Auscultation of vascular murmurs in the abdominal area is of major importance for the differential diagnosis of arterial hypertension. In renal arteries disorders (caused by atherosclerosis, congenital stenosis of dysplasia) there is a specific systolic murmur over umbilical area. In congenital coarctation of abdominal aorta the murmur is also clear.

In aortic insufficiency, a high systolic blood pressure and a big pulse difference produce large fluctuations of the vascular wall during the systole and diastole. It results in Traube's double tone over the femoral artery. Pressing upon this vessel with a stethoscope produces Duroziez double murmur.

The 1st component of this murmur is produced by stenosis of the artery after pressing with the stethoscope, and the 2nd component results from the acceleration of backflow in diastole (blood regurgitation)

Healthy people usually have no murmurs over their veins. But, a spinning top murmur over the jugular veins, produced in anemia, may be of some diagnostical significance. It has a buzzing or blowing tune, more distinct over the right jugular vein increasing when a patient turns his head to the left.

After completing the cardiovascular system examination, it is obligatory to check patients for other systems and organs: respiratory, digestive, urinary, endocrine etc. Only a complex examination of the body as a whole may be helpful in establishing the diagnosis of any disease. Physical examination of other body systems will be discussed in the next sections of this textbook. Only after completing a physical examination can a doctor proceed to laboratory and instrumental examination in order to establish an accurate and definite diagnosis.

In conclusion, we would like to underline once more, that the simplest physical method of examination is of greatest significance in diagnostics.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *Can murmurs be palpable?*
2. *What is cat's purring phenomenon?*
3. *Why does cat's purring phenomenon have this name?*
4. *Why is it obligatory to check patients for other systems and organs after completing the cardiovascular system examination?*

4. Read the task card below.

Describe the main peculiarities of physical examination of murmurs following the plan:
How is cat's purring phenomenon found out?
How are vessels examined?
What kind of examination may be helpful in the diagnosis of any disease?

5. Now, using the prompts from exercise 4, talk on the topic: PHYSICAL EXAMINATION OF MURMURS.

Medicine Through Biographies

Read the text below, pausing after every paragraph to find the main fact (or idea) around which it is centered.

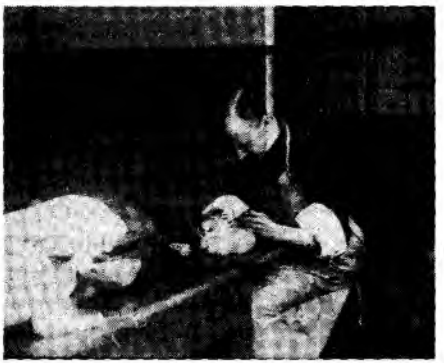
Then, read the text again and prepare a list of questions covering the main periods of William Osler's life. Using this list, discuss the facts with a partner/partners.

Then, make a detailed list of questions covering Osler's main academic and research achievements. Discuss the importance of his works with a partner/partners.

Write a summary of Osler's most significant contribution to the medical theory and practice. Compare the significance of Osler and Pirogov for British-American and Russian Medicine accordingly.

Discuss Osler's personal features and their influence on his professional activities.

LIFE AND WORK OF WILLIAM OSLER (1849–1919)



Sir William Osler (Fig. 15 — Osler performing an autopsy) was an outstanding physician and pathologist. He was one of the founders of clinical pathophysiology, the reformer of the British and American medical education, who had moved it onto practical rails. Osler was born in Canada in 1849 to the family of an Anglican preacher, the youngest of his nine children. In 1870 he graduated from a medical school in Toronto, then he continued his medical education at McGill University in Montreal. In 1872 he received a medical degree. Like other great clinicians, he started his work from dealing with pathological anatomy and general pathology, improving his knowledge and professional skills in Europe, learning from G. Burden-Sanderson, R. Virchow, L. Traube, B. Langenbek, F. von Hebra and K. Rokitansky. After a short period of medical practice in Ontario, he became in 1875 a professor of the Department of internal diseases at McGill University, Montreal (Canada), where he worked also as a pathologist since 1876, and as a doctor at Montreal hospital — since 1878. At McGill University Osler taught not only internal diseases, but also physiology and pathology. As a matter of fact, he was the first pathophysiologist of the New World. In 1884 he received a job offer from Pennsylvania University (the USA), and, *by tossing a coin*, made his choice and went to teach in Philadelphia. There he founded the American Medical Association. In 1888 Osler accepted an offer from the newly organized Johns Hopkins University in Baltimore. There he became the first clinician professor, and, together with his colleagues, he laid the foundation of principally new methods of teaching medicine, thereby bringing this new university to leading positions. There he created his immortal manual "The Principles and Practice of Medicine", which inspired a millionaire-philanthropist John D. Rockefeller to open the Rockefeller Foundation and the Institute of Medical Research. In this period he married a great-great-granddaughter of the legendary hero of the American Revolution Paul Revere. Osler played the same role for English-speaking medical world as did N. I. Pirogov for the Russian-speaking one. Osler's glory soon went beyond the borders of America, quite provincial place in terms of the academic world of the nineteenth century.

Therefore, since 1905 Osler worked in Oxford (GB) where he succeeded to his teacher G. Burden-Sanderson. In Britain he organized the best medical library of that time. After the death of the great scientist, it was bequeathed to McGill University. William Osler was a bibliophile,

even bibliomaniac. He said: "*To study medicine without reading textbooks is like going to sea without charts, but to study medicine without dealing with patients is not going to sea at all*". The libraries shaped by Osler are exemplary.

The total number of academic works written by W. Osler created on the internal diseases, surgery, parasitology, the history of medicine, culturology, and teaching exceeds 700! He discovered new species of nematodes (Filaria Osleri) and trematodes (Sphryanura Osleri), he was the first (1873) who described in detail the earlier discovered platelets. First description of subacute bacterial endocarditis with with typical red nodules on the skin of fingers (Osler's symptom) also belongs to him. He described Osler-Vaquez disease. His classical manual on internal medicine was published and republished in different languages, even in Chinese, worldwide. Osler's aphorisms are very well known, for example his famous phrase about "relative uselessness of a men over forty years of age". He had a rare gift for teaching young doctors. He was called the elect of the nature. He "denied chauvinism and national limitation" (L. Ashoff), having the reputation of the apostle of international medicine. Osler considered that studying medicine is studying the illness by means of sincere supervision without prejudiced theories.

He used to tell his students that the secret of successful work consists in regular arrangement of what it is necessary to do, and methodical performance of it. "His broadest humanism was combined with the deepest knowledge" (M. J. Breitman). Osler had a reputation of the kindest and a most hospitable person in Oxford. Sick children waited for him impatiently and were disappointed on parting with him. Once in a cold season, walking near the university, he came across a sick old beggar, and immediately donated his coat to that person. Few weeks later, after beggar's death a gift returned to owner with a last message of gratitude from the deceased person. Professor Osler continued to wear this coat for many years.

A number of important works by Osler were devoted to lung diseases and to tuberculosis particularly. He himself suffered from pneumoniae and periodic attacks of a bronchitis which quite often ended in pneumoniae. Nevertheless he continued to work. During one of his pneumonic episodes he wrote an important work devoted to this particular disease. Osler's last pneumonia was complicated with pleurisy and empyema. The operation was followed by the development of pulmonary abscess, and after the second operation on December the 29th, 1919 — a tragic outcome happened. Sir William Osler, a baronet of the British Crown was a person of brilliant mind and flashing wit. He is a famous medical writer not only for his textbooks but in connection with his well-known classification of medical doctors. He divided them into four groups: those who do much without telling anything, those who talk much without doing anything, those who talk much and do much and finally, those who don't tell anything and don't do anything. He emphasized that the latter ones earn most of all. Using a long pseudonym "Egerton Yorrick Davis, a retired captain of the US Army, a military

surgeon", he wrote a lot of parodies which were seriously republished in medical journals as journal workers took Osler's parodies at face value.

In 2010 the authors of this book had a happy chance to visit Oslerian Library at McGill University (fig. 16) and experienced great excitement there.

One can feel the spirit of Osler, walking along the book shelves. It seems that theimpact of his genius is still there.

Fig. 16. Russian and Chinese pathophysiologists in Osler Library at McGill University. First from the right — Mrs. Pamela Miller, History of Medicine Librarian of the Osler Library of the History of Medicine

PHYSICAL METHODS OF DIAGNOSIS IN PULMONOLOGY

Module 1

PATIENT'S INTERVIEW IN PULMONOLOGY

Unit 1.1

EVALUATION OF PATIENT'S COMPLAINTS: DYSPNEA

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

airway	дыхательные пути
alveolar	альвеолярный
asthma	астма; удушье
cardiorespiratory	кардиопульмональный, сердечно-легочный
combined dyspnea	комбинированная (смешанная) одышка
croup	круп (острый ларингит или ларинготрахеит, сопровождающийся явлениями спазматического стеноза гортани)
diphtheria	дифтерия
excursion	движение, подвижность, экскурсия
expiratory dyspnea	экспираторная одышка
exudative pleuritis	экссудативный плеврит, выпотной плеврит
facet	небольшая суставная поверхность
failure	недостаточность, декомпенсация; нарушение; расстройство
false croup	ложный круп, псевдокруп

findings	данные
general sickness	общее недомогание
goiter	зоб, струма
gross	макроскопический
hypercapnia	гиперкапния (повышенное содержание двуокиси углерода в крови)
hypoxemia	гипоксемия, аноксемия (пониженное содержание кислорода в крови)
inspiratory dyspnea	инспираторная одышка
laryngeal edema	отек гортани; отек подсвязочного пространства
neurotic	невротик, человек, страдающий неврозом
paralysis	паралич
pulmonary	легочный, пульмональный
strain	напряжение, нагрузка
stridor	стридор (свистящее дыхание)
vocal cords	голосовые связки
whooping cough	коклюш

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Pulmonological diagnostics starts with the interview of a patient. First of all, the character of complaints should be clarified, traditionally followed by anamnesis morbi et vitae. After that, the doctor turns to the physical investigation of the respiratory system, which is carried out in accordance with the conventional procedure of visual examination, palpation, percussion, auscultation, and special symptoms check. The process is then completed by proper laboratory and instrumental methods of respiratory system investigation. The doctor establishes the correct diagnosis by means of analysis and synthesis of the findings obtained. The complaints are usually typical in respiratory system disorders. They are: dyspnea, cough, chest pain, fever and general sickness.

DYSPNEA is the most serious disorder of normal respiration. But, breathlessness may be not only pathologic, but also physiologic. The disturbance of respiratory rhythm and breath rate is common in heavy physical strain even in healthy and well-trained individuals. In this case, however, dyspnea is transient, breath is rapidly restored. The latter phenomenon is known in sports medicine as "second wind". If the second wind does not come or comes too late, this may be an evidence of

hidden cardiorespiratory failure and pathologic dyspnea of pulmonary or cardiac origin.

Dyspnea has subjective and objective facets. If a patient complains of dyspnea, but the visible signs are absent, the dyspnea is called subjective. Objective dyspnea is registered as a complex of disorders in breath rate, depth and rhythm as well as in relative duration of breathing phases. Surely, in overwhelming majority of cases, the objective sides of dyspnea comply with a subjective feeling of dyspnea. Subjective dyspnea without any objective grounds may occur, most often, in neurotic patients.

The mechanism of dyspnea is related to poor alveolar ventilation and subsequent gaseous exchange disorder and hypoxemia. It is accompanied by hypercapnia and the accumulation of other pathologic metabolites collected in blood. Due to this, the respiratory center, being irritated, changes the program of breathing and provokes dyspnea. Sudden paroxysmal dyspnea is referred to as asthma. It manifests itself in attacks of breathlessness. Commonly, dyspnea is divided into 3 types, depending on the disorder of separate breath phases — inhalation and expiration.

These are: *inspiratory dyspnea* (connected with inhalation); *expiratory dyspnea* (appearing during expiration) and *combined dyspnea* (linked with both faulty phases).

Inspiratory dyspnea develops commonly in gross airway stenosis. The etiology may be either inflammation or tumor, as well as derived from compression of lung or bronchi by neighboring organs, goiter, aortic aneurism etc. Other causes may be: paralysis of vocal cords, laryngeal edema (for example, in diphtheria — true croup; in allergic edema — false croup), or a foreign body within a bronchus.

All these, and similar conditions, are characterized by difficult inhalation and active work of the muscles in inhalation. If the extent of stenosis is considerable, inhalation is accompanied by a specific sound of high-pitched noisy respiration — a so called "stridor", easily audible from some distance. A bright example of stridor is a so called "reprise" in whooping cough patients — a musical noisy inhalation after the attack of cough. Inspiratory dyspnea is usually accompanied by normal breath rate (16–18 breaths per min for adults). The correlation of breath to pulse rates is usually 1 to 4, as normally should be in adults.

Expiratory dyspnea is mostly caused by congestion and collection of excessive secretum in respiratory airways, e. g. in bronchioli (bronchiolitis). Edema of mucous membranes, detachment of dead cells from the inner surface of bronchi and spasm of finest bronchi (in bronchiolitis and bronchial asthma) or loss of normal pulmonary tissue elasticity and spring effect, causing the impossibility of passive expiration (in pulmonary emphysema) — all mentioned above may also generate expiratory dyspnea.

In cases like these constricted or dropsy bronchioli are still able to dilate during inhalation and give way to the airflow, reaching alveolae. But, during breathing out, which normally is passive, the expiration of

air through the narrowed airways is retarded and obstructed. It is accompanied by the generation of specific musical sounds, even whistling.

Combined dyspnea with both phases obstructed is characteristic of the restriction of pulmonary area (surface) of restriction of lung excursion and gas exchange (in pneumonia, exudative pleuritis, pneumothorax, atelectases, high position of phrenic muscle). It must be clarified, during patient interviewing, what phase of breathing is more difficult for him/her — exhalation or inhalation, or both. Ask the patient, when the dyspnea most frequently occurs: in a state of rest or only in exercise such as physical work, running, bicycle riding, hill climbing or walking upstairs. It is also important to ask if the patient hears some whistling or wheezing noises when breathing.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *How does pulmonary diagnosing start?*
2. *What should be clarified first?*
3. *What should be done after the character of complaints has been clarified?*
4. *What should be done after that?*

4. Read the task card below.

Describe the main peculiarities of pulmonological diagnostics following the plan:
How is pulmonological diagnostics carried out?
What are the characteristics of dyspnea?
What kinds of dyspnea are known?

5. Now, using the prompts from exercise 4, talk on the topic: PULMONOLOGICAL DIAGNOSTICS.

Unit 1.2

EVALUATION OF PATIENT'S COMPLAINTS: COUGH

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

abscess	абсцесс, гнойник
adenoid (s)	аденоид; аденоидный; аденоиды, аденоидные разращения, аденоидные вегетации
bronchiectasis = bronchiectasia	бронхоэктаз (расширение ограниченных участков бронха)
bronchitis	бронхит
bronchopulmonary	бронхолегочный
cancer	рак, карцинома, злокачественная эпителиома
ENT [ear, nose, throat]	ЛОР [ухо, нос, горло]
esophagus = oesophagus	пищевод
gangrene	гангрена; вызывать гангрену
gastric	гастральный, желудочный
gingiva (*pl.*: gingivae)	десна
grippe	грипп
hemoptysis = blood expectoration	кровохарканье
impairment	ухудшение (в результате травмы или болезни)
laryngitis	ларингит (воспаление слизистой оболочки гортани)
larynx	гортань
meager	бедный содержанием
measles	корь
nasopharynx	носовая часть глотки, носоглотка, эпифаринкс
nodose	нодозный, узелковый; узловатый
Osler's disease	истинная полицитемия, эритремия, болезнь Вакеза–Ослера
periarteritis	периартериит (воспаление адвентиции артерии)
polyp	полип
recurrence	рецидив
stomatologic	стоматологический
syndrome	синдром, симптомокомплекс
syphilis сифилис	
telangiectasia	телеангиэктазия (локальное чрезмерное расширение мелких сосудов)
tracheitis	трахеит (воспаление слизистой оболочки трахеи)
tracheobronchial	трахеобронхиальный
trauma	травма, повреждение
tuberculosis	туберкулез

ulorrhagia	десневое кровотечение
viscous	вязкий; липкий, клейкий; тягучий
vocal ligaments palsy	паралич голосовых связок

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

COUGH is the most meaningful complaint of bronchopulmonary disorders. It is a defensive reflex, consisting of forced expiration after deep inhalation with true glottis either closed or considerably narrowed. It produces the so called "push of cough", which is helpful in cleaning up the airways from the excess of sputum or foreign substances (caseous masses, food, blood, pus etc).

Cough may be permanent or recurrent. It may come in attacks (like in whooping cough or tracheobronchial foreign bodies). Permanent cough is most common in patients with chronic bronchopulmonary diseases (laryngitis, bronchitis, bronchiectasis, tuberculosis, pulmonary tumors). Cough may be very weak and even soundless in vocal cords lesions (syphilis, tuberculosis), vocal ligaments palsy (due to compression or surgical impairment of n. laryngeus recurrens). In vocal cords swelling resulting from inflammation, cough may be of a "barking" type (e. g., measles). Barking cough may be observed without any signs of inflammation in hysteric individuals. Cough may be wet (with sputum expectoration) or dry. Dry cough is the result of swelling of bronchial mucous membrane, when meager and viscous sputum is trapped in the lumen of bronchi and has difficulty finding its way out. It is observable in laryngitis, tracheitis, pneumosclerosis, dry pleuritis, and bronchial asthma. In light forms of respiratory tract inflammation, a cough is weak and occurs as in mild coughing.

Wet cough always has some specific timbre, influenced by the noise produced by movement of excessive discharge along the bronchi, added to cough push. Cough with sputum (secretum) expectoration is observed in pneumonia, chronic bronchitis, pulmonary abscess, tuberculosis and, especially, in bronchiectasis disease, when the amount of expectorated sputum may be up to one liter per day.

Checking the character of sputum is very significant. Ask the patient about the amount, consistence, scent, color, streaks of something, e. g. blood in sputum. Blood-streaked sputum is an evidence of hemoptysis. Blood may present in sputum in different forms, depending on its quantity: most often as blood-streaked sputum or rusty sputum, as well as in

separate blood spites or massive hemoptysis. Sometimes prune-juice sputum is observed.

Blood expectoration may result from tuberculosis of the lungs, but also occurs in bronchiectases, croupous pneumonia, lung abscess, gangrene and cancer of the lung, virus respiratory infections (like grippe), pneumothorax, pulmonary arteries embolism (pulmonary infarction), chest trauma, hemorrhagic syndrome.

Do not forget that blood expectoration may accompany mitral stenosis, myocardial infarction, periarteriitis nodosa. Moreover, blood may come to sputum from damaged gingivae (so called *ulorrhagia*, or minor bleeding during brushing of teeth), from inflamed nasopharynx, from disturbed telangiectases (Osler's[1] disease), from bleeding damaged adenoids or polyps. The nature may be clarified after special stomatologic and ENT examination of a patient.

The sputum may have some blood from the the gut. Commonly, in gastric or oesophagus bleeding the blood, mixed with acid gastric secretum, is dark in color (coffee dregs color) and has acid pH; at the same time blood in true hemoptysis is scarlet and with alkaline pH. The sputum in the latter case is usually foamy. Rusty sputum is common in croupous pneumonia, mitral valve disease and expectoration of the pulmonary hemorrhage remnants.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What is the most meaningful complaint of pulmonary disorders?*
2. *What kind of reflex is cough?*
3. *What does it consist of?*
4. *What is "push of cough" produced by?*

4. Read the task card below.

Describe the main peculiarities of cough following the plan: **How is cough related to pulmonologic disorders?** **How may cough be characterized?** **Why is sputum checking important?**

5. Now, using the prompts from exercise 4, talk on the topic: COUGH.

[1] Sir William Osler (see p. 221 above), Baronet, Canadian, American and British physician, born July 12, 1849, Bond Head, Tecumseth, Canada West; died December 29, 1919, Oxford, Great Britain.

Unit 1.3
EVALUATION OF PATIENT'S COMPLAINTS: CHEST PAIN AND FEVER

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

Addison-Biermer anemia = Addison-Biermer disease	пернициозная анемия, мегалобластическаяая анемия с первичным аутоиммунным атрофическим гастритом, болезнь Аддисона–Бирмера
analgesic	аналгезирующее средство, болеутоляющее средство, аналгетик
appendicitis	аппендицит (воспаление червеобразного отростка слепой кишки)
cholecystitis	холецистит (воспаление желчного пузыря)
cholelithiasis	желчнокаменная болезнь, холелитиаз
dizziness	головокружение
hematologic	гематологический
heterotopic	отраженный
herpes zoster	опоясывающий герпес, опоясывающий лишай
hyperthyroidism	гипертиреоз, гипертиреоидизм (синдром, обусловленный повышением активности щитовидной железы и ее увеличением)
interlobar	интерлобарный, междолевой, находящийся между долями
leukemia	лейкоз, лейкемия, белокровие
mesothelioma	мезотелиома, целотелиома
misdiagnosis	ошибочный диагноз
myelomatosis	миеломная болезнь, множественная миелома, болезнь Рустицкого–Калера
myositis	миозит (воспаление мышцы)
narcotic	наркотизирующий, вызывающий наркоз
neuralgia	невралгия
pancreatitis	панкреатит (воспаление поджелудочной железы)
periostitis	периостит (воспаление надкостницы)
prodromal stage	продромальный период
pulmonary tumor	опухоль легкого
purulent pleuritis	гнойный плеврит
remittent	перемежающийся (о лихорадке); ремиттирующий (о течении болезни)
subfebrile	субфебрильный
visceral	висцеральный (относящийся к внутренним органам)

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

CHEST PAIN — is often an evidence of pleura involvement, but can be registered also in pulmonary diseases (such as: croupous pneumonia, pulmonary infarction, lung cancer, tuberculosis), although in these cases pain is also produced by involvement of visceral, especially interlobar pleura, rich with nocireceptors.

Pleural pain is usually observed on inhalation, especially a deep one (in dry pleuritis, in the very beginning of adhesive pleuritis, in pleural mesothelioma). The inflamed pleural layers are rubbing against each other, thus producing pain. That is why patients stick to practice shallow breathing and avoid coughing in order to protect themselves from the pain.

Pleural cancer (mesothelioma) and pulmonary infarction (brought in by pulmonary artery branches embolism) both produce extremely severe pain resistant to narcotic analgesics.

Involvement of phrenic pleura may spark abdominal pain which can be mistakenly treated as a sign of appendicitis or cholecystitis. Croupous pneumonia of the inferior lobes results in such misdiagnosis quite regularly.

Severe chest pain is characteristic also of pneumothorax.

Herpes zoster may also be the reason of chest pain, even in prodromal stage, when a typical skin rush is still absent. Sometimes chest pain is produced by costal trauma, periostitis, intercostal neuralgia, myositis, myocardial infarction, some hematologic diseases (like systemic myelomatosis, Addison-Biermer's[1] anemia, leukemia).

Chest pain may be heterotopic in origin: in some abdominal diseases, like pancreatitis, cholecystitis, cholelithiasis.

FEVER — is a common complaint in pulmonologic patients. Most frequently, pneumonia is of infectious origin causing fever. The most pronounced fever is characteristic of the heaviest croupous pneumonia. It may be accompanied by high temperature up to 39–40 degrees centigrade (C°) and by chills. In focal (broncho) pneumonia and in pleuritis the temperature rises slightly and not so abruptly as in croupous (lobar) pneumonia.

[1] Thomas Addison (see p. 235 below), English physician, born October 1795, Long Benton, Northumberland, near Newcastle; died June 29, 1860, 15 Wellington Villas, Brighton.

High temperature, fever, chills and excessive sweating are typical of such lung diseases as abscess, purulent pleuritis, and gangrene. Purulent lung and pleural illnesses are accompanied by a remittent temperature curve, with big swings between morning and evening temperatures.

In tuberculosis, the temperature may be low, high or subfebrile. High temperature along with excessive sweating characterizes severe, disseminated forms of tuberculosis. Most of the TB patients have subfebrile temperature below 37,5°. It is distinguished for its constant character, lasting for weeks and even months. Evening fever of this kind requires profound examination for the exclusion of tuberculosis. The combination of such kind of fever like this with sweating and weight loss is observable also in hyperthyroidism, in which case differential investigations are required.

General sickness and nonspecific dizziness — are characteristic complaints of pulmonary patients. It may be observed both in acute and chronic pulmonary pathology. In acute pneumonia headache, poor appetite, constipation are not rare. Noticeable weakness is typical in pulmonary tumors and cor pulmonale. Evening weakness and exhaustion after a working day may be present in tuberculosis of the lungs.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *In what pulmonary diseases can chest pain be registered?*
2. *What deseases is severe chest pain characteristic of?*
3. *What temperature do most of the TB patients have?*
4. *What are characteristic complaints of pulmonary patients?*

4. Read the task card below.

Describe the main peculiarities of chest pain and fever following the plan:

With what diseases is chest pain connected?
What reason causes fever most often?
What kinds of ailment are common for pulmonary patients?

5. Now, using the prompts from exercise 4, talk on the topic: CHEST PAIN AND FEVER.

6. Work in pairs. Act out the dialogue below between physician and patient with your partner:

AT THE DOCTOR'S

Patient. Good morning, doctor. I've suddenly fallen ill. Before that I felt relatively well. However I suffer from varicose veins in my legs. People have advised me to rub heparin ointment into the skin of my shins. Yesterday before going to sleeping I massaged my legs and went to bed. Suddenly I had a pricking pain in my right side and I had coughed producing a small amount of phlegm... But the most worrying part was that I saw some blood streaks in the phlegm. Could it be tuberculosis?

Doctor. Had you caught a cold the night before? But then again it's summer now and it should be warm enough outside already. Did you have a fever? Did you have high temperature? Did you sweat at night?

Patient. No, doctor, I didn't't. I'm not inclined to catching colds. I don't get fevers. So I didn't take my temperature as I thought it was unnecessary. I don't sweat. I just feel weak and out of breath today.

Doctor. Do you smoke? Have your ever had pain your lungs? Does tuberculosis run in your family?

Patient. I've never smoked. I've only suffered from childhood infections such as measles and mumps. Pirquet's reaction and Mantoux test used to be normal.

Doctor. Please strip to the waist. I am going to examine you. The skin and the mucous are clean, but the tongue and the lips are slightly cyanotic. You've got tachypnoe. You've got rapid pulse which counts 90 beats per minute. Arterial pressure is normal. In the lungs under your left scapula I can make out a small dullness of percussion sound. In this place I can hear pleural friction rub but the rales in the lungs are absent. This is the symptom for right dry pleurisy

The tops of your lungs are ideal. The liver and the spleen are not increased. And the veins in your legs are indeed dilated, and on the right there is a limited reddening of the shin skin. It is probably thrombophlebitis. This seems to be the cause of your pleurisy.

Patient. Does it have to do with my sick veins if I have pleurisy? Once I read in "Health" that pleurisies appear usually in connection with tuberculosis. And besides I have hemoptysis.

Doctor. The matter is that you were too zealous in the treatment of your veins. One can use heparin ointment, it certainly helps. But you've made a big mistake, like many other patients with varicose veins. People with such a disease, which is complicated by thrombophlebitis shouldn't massage legs. Thrombi occasionally appear in widened shin veins. By massaging your legs you can dislodge these thrombi, which in turn can trouble the right ventricle and enter the lungs. In the lungs they get stuck in small pulmonary vessels. This is called thromboembolism. In this situation a heart attack can occur which typically is characterized by stitch, pleurisy and hemoptysis.

Patient. What can I do doctor?

Doctor. Pulmonary thromboembolism is a serious disease. You can not cope with it alone at home. You have to require medical treatment

with diluted remedies and a course of antibiotics to prevent complications in connection with heart arrest. Now I shall call an ambulance which will take you to the admission department of our hospital. I can not allow you to go home as it is dangerous for your life. This is very serious. And cease to massage your legs.

Patient. OK. Thank you very much. I'm really scared. How could I have known that massaging my legs is dangerous in case of thromboembolism? But I want to live. I'm only fifty years old.

Medicine Through Biographies

Read the text below, pausing after every paragraph to find the main fact (or idea) around which it is centered.

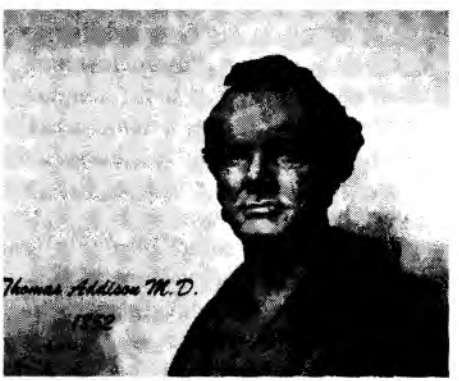
Then, read the text again and prepare a list of questions covering the main periods of Thomas Addison's life. Using this list, discuss the facts with a partner/partners.

Then, make a detailed list of questions covering Addison's main academic and research achievements. Discuss the importance of his works with a partner/partners.

Write a summary of Addison's most significant contribution to the medical theory and practice. Discuss Addison's personal features and their influence on his professional activities. Describe the last period of his life.

Thomas Addison (1795–1860)

(after Ole-Daniel Enersen and Charles Douglas Wehner
Fig. 17 — photo by Ch. D. Wehner after sculpture by J. Towne, available from: http://wehner.org/addison/images/himself.jpg)



A prominent British physician Thomas Addison was the son of Sarah and Joseph Addison, a grocer and flower-seller in Long Benton, Northumberland. He attended the local village school and then went to the Royal Free Grammar School in Newcastle-upon-Tyne. There he learned Latin so well that later used to make his personal notes in that language and spoke it fluently. This probably influenced both his diction and writing style, known for habitual exactitude. His father had wished him to become a lawyer, but in 1812 Thomas entered the Univer-

sity of Edinburgh as a medical student. He graduated in 1815 and was conferred Doctor of Medicine with the graduation thesis titled: "Dissertatio medica inauguralis quaedam de syphilide et hydrargyro complectens — Concerning Syphilis and Mecury". The same year Addison moved to London where he took up his residence at Skinner Street, Snow Hill, and became house surgeon at the Lock Hospital. He commenced practice, while at the same time he was physician at an open ward reception on Carey Street. Thanks to his superiors he became especially interested in dermatology. This interest prevailed over his whole life and led to him being the first to describe the changes in skin pigmentation typical of chronic primary adrenal insufficiency or Addison's disease. Addison's brilliant career as a physician and scientist began in 1817 when he was enrolled as a physician pupil at Guy's Hospital in London. Guy's Medical School book records his entrance: "Dec. 13, 1817, from Edinburgh, T. Addison, M. D., paid pounds 22... to be a perpetual Physician's pupil." He obtained his licentiateship in the Royal College of Physicians on 22nd of December, 1819 and was elected a fellow on July 4th, 1838.

On January 14th, 1824 he was promoted to assistant physician, In 1827 he was appointed "Lecturer of Materia Medica". At that time, when medical students paid fees for separate courses of lectures, they sought throughout the metropolis for the most attractive teachers. A lot of pupils kept attending Addison's lectures, and so great was the attendance that his lecture-fees must have amounted to $ 700 or $ 800 a year. In 1835 Addison was joint lecturer with a famous founder of nephrology Richard Bright (1789–1858) on practical medicine, and in 1837 Addison became a full physician at Guy's Hospital. When Bright retired from the lectureship in 1840 Addison became sole lecturer. He held this position until either 1854 or 1855. Addison was a brilliant lecturer and diagnostician but a rather shy and taciturn individual, and, as a result, had a small practice, at a time when doctors of his position evidently all had large practices. Probably, it was due to his introverted nature and lack of any self-advertising, that he was underestimated by medical community and his career developed quite slowly. Addison received all official merits and honors with some delay, much later than he actually deserved it. Nevertheless, he was one of the most respected physicians at the Guy's Hospital where he exerted a great deal of influence and taught in a dogmatic and forceful manner, devoting himself almost wholly to his students and patients. He was described as the type of doctor who is always trying to discover the re-arrangement in a piece of machinery, rather. The discovery of "bronze disease" immortalized Addison's name. The adrenal glands were described by Eustachius as early as in 1563, but it was many years before their function was elucidated. The story of Addison's disease begins with Addison's first description in a short note in an article for "London Medical Gazette" entitled: "Anaemia — disease of the suprarenal capsules" in which the disease is not distinctly separated from a new form of anemia. This article was followed up in his monograph: "On the Constitutional and Local Ef-

fects of Disease of the Suprarenal Capsule" which was published in London in 1855 and represented the beginning of the study of these endocrine glands. This work was much debated in England and Scotland and largely discounted, John Hughes Bennett (1812–1875) in Edinburgh denying the existence of the disease. Armand Trousseau (1801–1867) in Paris, however, was quick to recognize adrenal failure and gave it the eponym "Addison's disease". In his monograph, one of the truly remarkable medical books of the nineteenth century. Addison points out that it was really under his attempts at elucidating the base of a peculiar form of anaemia that he happened to find bilateral pathological changes comprising both suprarenal glands. He maintains that the disease of the adrenal glands could not be connected with the anaemia, as he had previously thought. His classical descriptions of the symptoms of the eleven patients with altered adrenal cortex collected by himself and his younger associate Samuel Wilks (1824–1911) is worth quoting:

"The leading and characteristic features of the morbid state to which I would direct your attention are: anaemia, general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of the color in the skin, occurring in connection with a diseased condition of the suprarenal capsules... The discoloration pervades the whole surface of the body, but is commonly most strongly manifested on the face, neck, superior extremities, penis, scrotum, and in the flexures of the axillae and around the navel."

One of the eleven patients in the monograph is of special interest. This patient had been treated by Bright, who had noted pigmentation of the skin, the irritability of the stomach, the emaciation, and the asthenia which quickly lead to the patient's death. He also described the characteristic changes: the adrenal glands much enlarged with deposits of a "scrofulous kind", and partial deterioration of the glands with deposits of pus. It seems that Bright observed a case of tuberculosis adrenalitis and has been confused by the fact that the patient also had tumors of the chest and a swelling of parotids. Anyway, he did not connect the subsequent classic manifestations with the changes of the adrenal glands. This description came many years before Addison's monograph and, if Bright had understood the connection his name, not Addison's, would have been attached to the disease.

Addison's comment to Bright's account tells something of why Addison enjoyed modest popularity: "It did not appear that Dr. Bright either entertained a suspicion of the disease of the capsules before death, or was led at any period to associate the color of the skin with the diseased condition of the organs, although his well-known sagacity induced him to suggest the probable existence of some internal malignant disease. In this as in most other cases, we have the same remarkable prostration, the usual gastric symptoms, the same absence of any very obvious and adequate cause of the patient's actual condition together with a discoloration of the skin, sufficiently striking to have arrested Dr. Bright's attention even during the life of the patient." The disease now known as Addison's anaemia was first described in detail by Addison in 1849 in a

lecture in the South London Medical Society, but it was apparently not published in an ordinary way. He is also said to have lectured on the disease already since 1843. It appears from these accounts that what Addison called "this remarkable form of anaemia" was probably pernicious anaemia. Nowadays we know that both adrenalitis and pernicious anemia with primary atrophic gastritis may occur in parallels in patients with combined multi-organ autoimmunity. It means that combination of symptoms noticed by Addison is not occasional, but pathogenetically linked. An overlapping of this two diseases on the side of Addison, who tried to unite "his" diseases into one entity, colored the struggle about priority to the idiopathic or pernicious anemia. In "The Medical Times" and in "Gazette of London" in 1874, one could read that Anton Biermer in Zurich has described a new type of anemia, "idiopathic anemia", and that this disease was not yet described in England. The paper adds: "...no doubt there will soon be many observers on the lookout for it." This caused Samuel Wilks seven days later in a letter to "British Medical Journal" to inform that the disease was well known in England as Addison had lectured on it in 1843.

Today Addison's discoveries are regarded as fundamentally significant in the study of endocrine glands and multi-targeted autoimmunity.

In 1839 together with Bright, Addison was to write a textbook of medicine, "Elements of the Practice of Medicine", but only one volume was written and that by Addison. This joint undertaking by two of the most famous European physicians of XIX century is quite scarce. It was intended as a "work at once elementary and practical to which teachers might refer their pupils as a companion and assistant during the period of their studies". This book contains if not the first, then the most lucid and complete description of "Inflammation of the caecum and appendix vermiformis" — from the earliest symptom to the buildup of abscess and death in peritonitis, of course with significant autopsy findings. Besides, in co-authorship with Sir William Whitey Gull (1816–1890), Addison described xanthoma diabeticorum, and he also first described morphea, or circumscribed scleroderma (Alibert's keloid syndrome), which is sometimes called Addison's keloid. Addison took a great interest in diseases of the lungs and in 1843 described the pathomorphology of pneumonia, which so far in accordance with René-Théophile-Hyacinthe Laennec (1781–1826) had been considered an inflammation of the interstitial tissues of the lungs. Addison followed the fine bronchial branches to their very end, and found that the inflammation consisted of "pneumonic deposits in the air cells", the alveolae. Addison's lifelong interest in dermatology is evident from some of his writings. One article in particular deserving to be mentioned is: "On a certain affection of the skin, vitilogoidea plana tuberosa", in which he presents the first account of xanthoma planum et tuberosum. This symptom is now known to be associated with hypercholersterolaemia. He founded the Department of Dermatology at Guy's in 1824 and his influence is still evident in the collection of wax models of skin disorders which were prepared under his supervision. Addison was at his best at the bedside, always

moving to one side since he was slightly deaf in one ear. He used to tell his students that if he could not reach a diagnosis in a patient he would think of all the possible explanations for his patient's symptoms on his way to and from the hospital. His abilities to sift evidence and come up with a diagnosis were unrivalled in his day, but he did not devote the same energies to alleviation or cure. It is evident from literature that Addison and Bright were among the first to introduce scientific principles in the diagnostics of diseases, demanding that the physician should try to correlate physiological findings during life with the pathomorphological observations done at autopsy, something which was still quite rare at the time. To a certain degree, his role in development of British medicine is analogous to that of his younger contemporary: Sergei Petrovich Botkin (1832–1889) — in Russian medicine. This dawn of a new era was initially met with cynicism and resistance to changes amongst the establishment — a situation all too familiar to many scientists of our time. The old school even protested against the use of the new approaches, like stethoscope, which had been introduced by Laennec, whom Addison admired so much. Addison, probably, was a naturalist in Medicine, even more than physician, at least to recognize a disease for him was more important than to treat it. Once, when called in to see a patient he spent a long time in finally arriving at the diagnosis of an abdominal cancer. He discussed this with the attending doctor and the patient's friends and relatives and was leaving when he was reminded that he had not written a prescription. He asked what he was already being given and when told "a magnesium mixture" he said "a very good medicine, go on with it". This probably explains why his practice was not as big as it might have been.

Both marital happiness and fame came late to Addison. In September 1847, at the age of 52 years, he married Elizabeth Catherine Hauxwell at Lanercost Church. They were childless, although she had two children by her first marriage. His membership in the Royal College of Physicians, invitation to lecture at the Royal Society, to be physician to the court, honorary titles, etc, all came later, often decades later, than what would have been "normal" for a medical scientist of his importance. He must have been pleased to read the following text in "The Medical Times" and "Gazette": "We believe that Dr. Addison has made a discovery which is the most important practical medicine has produced for many years, and one in every way worthy of the untiring zeal and energy in professional pursuits which has characterized his life." After ill-health forced him to leave Guy's Hospital Addison received an admiring letters from his pupils. He is considered by many to be the greatest of the triumvirate Addison-Bright-Hodgkin, "so that every Guy's man during the 30 or 40 years of his teaching, was a disciple of Addison holding his name in the greatest reverence and regarding his authority as the best guide to the practice of the profession". The following statement in the medical press adds to the picture: "He is a fine, dashing, big, burly, busting man, proud and pompous as a parish beadle in his robe of office. Dark, and of sallow complexion, an intelligent counte-

nance and noble forehead, he is what the ladies would renounce a fine man. He had mentally and physically a tall idea of himself. Every sentence is polished, is powerful: he prefers the grandiloquent. Slow and studied are his opening sentences, studied the regularity of his intonations. The advantages of his tall and graceful person are artfully employed to add to the favorable impression; his attitudes, tones and manner are studied and systematic."

Addison had a number of episodes of severe depression which he greatly feared. He retired in 1860 because of an incipient cerebral disorder depression and wrote to his students: "A considerable breakdown in my health has scared me from the anxieties, responsibilities and excitement of my profession; whether temporarily or permanently cannot yet be determined but, whatever may be the issue, be assured that nothing was better calculated to soothe me than the kind interest manifested by the pupils of Guy's Hospital during the many trying years devoted to that institution." Three months later, on June 29, 1860, he committed suicide. On July 7, 1860, "The Medical Times" and "Gazette" published a note of Addison's death, but neither the "Lancet" nor the "British Medical Journal" recorded it, something that was considered almost obligatory. The "Brighton Herald" of the 30th June, 1860 published a description of Dr. Addison's death: "Dr Addison, formerly a physician to Guy`s Hospital, committed suicide by jumping down the area (i. e. the space between the front of the house and the street) of 15 Wellington Villas, where he had for some time been residing, under the care of two attendants, having before attempted self-destruction. He was 72 years of age (sic), and laboured under the form of insanity called melancholia, resulting from overwork of the brain. He was walking in the garden with his attendants, when he was summoned in to dinner. He made as if towards the front door, but suddenly threw himself over a dwarf-wall into the area — a distance of nine feet — and, falling on his head, the frontal bone was fractured, and death resulted at one o`clock yesterday morning"

He was buried in Lanercost Abbey, Cumberland, near his childhood home. In his memory the university placed a bust of him in the pathological museum, named a hall of the new part of the hospital after him, and perpetuated his memory with a marble wall table in the chapel.

Unit 1.4
ANAMNESIS MORBI ET VITAE IN PULMONOLOGY

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

alcohol abuse = alcohol addiction	алкоголизм
allergic rhinitis	аллергический ринит
alveolitis	альвеолит (1. альвеолярная пневмония; 2. воспаление стенок альвеолы зуба)
antipyretic	жаропонижающее средство, антипиретик; жаропонижающий, противолихорадочный
bronchospasm	бронхоспазм
carcinogen	канцероген, канцерогенное вещество, онкогенное вещество
farmer's lungs	"легкие фермера", экзогенный плесневый аллергический альвеолит
hay fever = hay catarrh	поллиноз, сенная лихорадка
idiosyncrasy	идиосинкразия, повышенная чувствительность, индивидуальная непереносимость
immunization	1) иммунизация, предохранительные прививки, профилактические прививки; 2) иммунизация, вакцинация
morbidity rate	коэффициент заболеваемости
occupational bronchitis	профессиональный бронхит
occupational hazards	профессиональные вредности
pigeon breeder's lung	болезнь голубеводов, экзогенный плесневый аллергический альвеолит
pneumoconiosis	пневмокониоз (название профессиональных болезней органов дыхания, обусловленных воздействием производственной пыли)
pollutant	загрязняющее вещество, загрязнитель; примеси (в воздухе)
purulent	гнойный; гноящийся
silicosis	силикоз, силикатоз (форма пневмокониоза, возникающая вследствие вдыхания кремний-содержащей пыли)

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

ANAMNESIS is registered in order to check: how did the disease start (abruptly or gradually, was the fever present from the very beginning? or was the body temperature normal? did the patient suffer from:

— night sweats;
— side pain on breathing and in resting state;
— cough of some kind;

was there any expectoration of sputum... etc.?

The disease could be triggered by exposure to cold in case of improper clothing or cold shower taken after sport exercises and work, or by exposure to draught. In croupous pneumonia a patient can sometimes indicate the exact hour of the onset of the disease, while in pleuritis the disease sets in, little by little, with complaints (side pain on breath, cough, dyspnea, fever) gradually growing.

The checking of childhood anamnesis and previous diseases is important. Even childhood infections like measles, whooping cough, grippe may be of big subsequent meaning. In every concrete case it is necessary to check all probable contacts with patients infected with TB and other contagious diseases involving respiratory tracts.

Ask the patient about any possible self-treatment undertaken at the initial stage of the disease before deciding to visit the doctor (what drugs were taken? in what doses? were any antipyretics and antibacterial remedies used? were any home or traditional remedies used? were they effective or not?).

In anamnesis vitae the doctor must reveal the patient's history of growth and development. Were any infections, common colds and sore throat episodes frequent or not? There exist certain genetic predispositions to pulmonary pathology. It requires careful genetic and family anamnesis. Case histories of relatives are also important for TB patients.

The doctor must be aware of the patient's living conditions. Damp air, poor ventilation of the room, smoking and/or cohabitating with smokers, working in the same room with smokers, living in a bad climate — all these factors may increase the risk of pulmonary diseases.

Passive smoking is almost as dangerous as active. It is especially true of the diagnosis of oncologic pulmonary pathology. It has been demonstrated, for example, that smokers have a 50 times greater lung cancer morbidity rate than non-smokers. Alcohol abuse also increases the risk of development of certain pulmonary diseases: purulent pulmonary processes, tuberculosis. It is related with diminished resistance of alcoholics to infection and poor diet and social condition of many alcoholics.

Occupational hazards are of great importance in pulmonology. The following works are especially hazardous: work in dusty rooms, weaving mills, mills, mains, in building materials production (sand, concrete, cement, bricks). Air contact with irritating pollutants or carcinogens is

essential. For example, work with asbestos is considered to be associated with higher risk of mesothelioma. Sometimes it is occupational exposure to certain saprophytic fungi growing on some animal- or vegetable-born dusty particles and provoking extrinsic allergic alveolitis (farmer's lung, pigeon breeder's or bird fancier's lung, maple bark stripper's lung, cheese washer's lung and even sauna frequenter's lung etc). Neglect of safety rules in industry may lead to occupational bronchitis, pneumoconiosis (like silicosis, or antracosis). Professional bronchitis is described even in dentists, who neglected the protective respirators and inhaled for a long time the dental dust produced by a drilling machine. Allergologic anamnesis is of great significance. Be sure to ask if the patient had previously suffered from bronchial asthma or some bronchospasm, from any drug, food or odor idiosyncrasy, hay fever or allergic rhinitis; reveal, how he/she reacted to immunizations. Of course, a doctor must ask lung patient about probable previous and currently diagnosed diseases of other organs and systems. Cardiovascular diseases, diabetes mellitus, systemic illnesses — are of special interest. It is well-known, for example, that the diabetic patients are liable to tuberculosis.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *Why is anamnesis registered?*
2. *What the disease could be triggered by?*
3. *In what disease can a patient indicate the exact hour of disease onset?*
4. *How does a disease set in pleuritis?*

4. Read the task card below.

Describe the main peculiarities of anamnesis vitae following the plan:

Why is anamnesis registered?
Why is checking of childhood anamnesis and previous disea-s es important?
What does a doctor have to reveal in anamnesis vitae?
Why is the analisis of a patient's living conditions very important for a pulmonoilogist?

5. Now, using the prompts from exercise 4, talk on the topic: ANAMNESIS.

Module 2

OBJECTIVE PHYSICAL EXAMINATION IN PULMONOLOGY

Unit 2.1

GENERAL VISUAL EXAMINATION: POSITION, INTEGUMENT, SOMATOTYPE AND CHEST EXCURTIONS

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

alcoholic intoxication	алкогольная интоксикация
apnea	1) апноэ (временная остановка дыхания); 2) асфиксия, удушье
apneustic breath	1) апноэ (временная остановка дыхания); 2) глубокое редкое дыхание
atherosclerosis	атеросклероз
Ayerza's disease	первичный атеросклероз легочной артерии, синдром Айерсы
carbon dioxide acidosis	газовый ацидоз, дыхательный ацидоз, респираторный ацидоз
cerebral injury	повреждение мозга
Cheyne-Stokes respiration	дыхание Чейна-Стокса
coma	кома, коматозное состояние; глубокое бессознательное состояние (с расстройством жизненно важных функций)
comatose	коматозный, находящийся в состоянии комы
conscious	находящийся в сознании
consciousness	сознание
drive	импульс
flush	покраснение
herpes	герпес
labial	губной
meningitis	1) менингит; 2) лептоменингит
nasal	носовой, назальный
neuron	нейрон, нервная клетка, нейроцит

Pickwickian syndrome	пиквикский синдром (сочетание выраженного ожирения с легочной гиповентиляцией, малоподвижностью, сонливостью)
pneumotaxis	регуляция внешнего дыхания
pontine = pontile	относящийся к варолиеву мосту
somatotype	тип конституции, тип телосложения
somnolence = somnolency	1) сонливость; 2) полубессознательное состояние somnolence
state of nourishment	степень упитанности
stroke	внезапный приступ; припадок
uremia	уремия, мочекровие
uremic	уремический (относящийся к уремии, обусловленный уремией)

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

EXAMINATION of a patient starts with visual inspection.

Visual examination may reveal objective dyspnea, cyanosis or a marked flush on the cheeks. In croupous pneumonia, flush is more pronounced on the side of lesion. It has been noticed that the people with long eyelashes are more liable to tuberculosis. Feverish flush and eye glitter may be present in both tuberculosis and thyrotoxic patients. Skin may not always have not obligatory bluish color, but is characterized by pallor.

Pallor is common in exudative pleuritis or in pulmonary hemorrhage. Herpes labialis et nasalis commonly occurs in pneumoniae.

Skin and mucous membranes cyanosis is characteristic of hypoxemia in pneumoniae, chronic bronchitis, purulent processes in the lungs. The decompensated cor pulmonale produces dramatic cyanosis with almost black skin. In Ayerza' disease, atherosclerotic pulmonary artery stenosis, the cyanosis is equally outstanding. It is accompanied by marked dyspnea, aggravated at the slightest efforts. Acute development of cyanosis is typical of pulmonary artery branches thromboembolism, as well as pneumothorax and foreign body within a bronchus.

The patients' posture in pulmonary pathology may be forced; some of them sit with their hands leaning on the bed (in bronchial asthma attack); others lie in a horizontal position (in cor pulmonale) or with their affected side down (in unilateral exudative pleuritis, in this way providing more effective respiratory excursions for the healthy side).

Some chronic processes in the bronchopulmonary system (e. g. bronchiectases) are accompanied by formation of drumstick fingers and watch glass nails.

A visual examination enables the doctor to estimate the patient's general condition, which may be satisfactory or heavy, down to comatose status. Visual examination makes it possible to determine the patient's somatotype and state of nourishment. It has been observed that people with an asthenic somatotype and poor nourishment state are more inclined to pulmonary pathology. But severe obesity also may be accompanied by pulmonary ventilation disorders with cyanosis, dyspnea and marked somnolence (a patient may fall asleep when eating or driving). This is referred to as Pickwickian[1] syndrome (a variety of cor pulmonale).

Visual examination can enable the doctor to diagnose the presence and type of dyspnea and respiratory rhythm disorder. Periodic Biot's[2] respiration mentioned above is characterized by several normal in-breaths, followed by apnea of various duration. This type of respiration may reflect a decreased respiratory centre excitability or even a none-functional state of its upper unit — pneumotaxic centre or pontine respiratory group, with a shift of pacemaker activity downwards to apneustic centre (this may occur in meningitis, brain tumors, strokes, cerebral injury and coma).

Cheyne-Stokes[3] respiration is also characterized by periods of apnea and hyperapnea, but during hyperapnea breath rate and depth gradually increase, reaching a certain level, then, vice versa, decrease down to a new apnea pause. This is repeated several times. During provisional apnea loss of consciousness, bradycardia, and pupil's narrowing may occur. This type of respiration is considered by some internists to be unfavorable for the prognosis, reflecting deep disorders of central respiratory regulation (it may occur in cranial trauma, stroke, heavy cerebral atherosclerosis, various comae, e. g., uremic coma). But many pathophysiologists insist that Chayne-Stokes type of dyspnoe is not always associated with severe irreversible disorders. It results from decrease of carbogen dioxide sensitivity in respiratory neurons and from weak carotid drive for inhaling, and may be observed in sleeping healthy infants or very old persons. It can also be manifested in severe alcoholic intoxication.

Grocco — Frugoni[4] dissociated respiration is related to discoordination of the phrenic muscle and intercostal muscles contractions. In extreme cases it may look as a paradox of simultaneous in-breath

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[1] Pickwickian syndrome is a disorder that was named not after medical doctor, or patient named Pickwick, but after "Joe, the fat, red faced boy" in Charles Dickens' novel "The Pickwick Papers". This hero of the novel repeatedly falls asleep.

[2] Biot C. (1850–1918), French physician.

[3] See footnotes 2, pp. 161, 202.

[4] Pietro Grocco, Italian physician, born June 27, 1856, Albonese (Pavia); died February 12, 1916, Courmayeur, Val d'Aosta. Cesare Frugoni, Italian physician, born May 4, 1881, Brescia; died January 6, 1978.

movement of upper chest and out-breath movement of abdomen. This is also an evidence of a bad prognosis (in meningitis, brain tumors, brain abscesses, uremia and diabetic coma). It is worth nothing that Biot' and Cheyne-Stokes' respirations may be observed in fully conscious patients with marked cerebral atherosclerosis.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What can visual examination reveal?*
2. *In what diseases is pallor common?*
3. *What kind of people are more liable to tuberculosis in terms of appearance?*
4. *In what patients can feverish flush and eye glitter be present?*

4. Read the task card below.

Describe the main peculiarities of visual examination following the plan:

What may visual examination reveal?
With what information does supply a doctor?
What lung diseases can visual examination enable a doctor to diagnose?

5. Now, using the prompts from exercise 4, talk on the topic: VISULAL EXAMINATION.

6. Work in pairs. Act out with your partner the dialogue between physician and patient:

AT THE DOCTOR'S

Patient. Good morning. I don't like to going to doctors. But I had to go to see you today.

Doctor. How can I help?

Patient. Well, doctor, I haven't been able to breath normally for over a month because of the cough.

Doctor. Was it you who has been coughing all over the polyclinic? You probably have been disturbing other patients, haven't you?

Patient. Yes, I have. You're right, doctor. I'm always disturbing my family coughing day and night. Because of me my family don't sleep.

Doctor. You probably, smoke a lot, don't you? Far away you can smell tobacco smoke on you. By the way, I've never smoked and the smell of tobacco isn't really pleasant to me.

Patient. Can I stay here for a short while? I'll leave soon. I've been smoking heavily since I was at school. I can't give up smoking. I've got a stressful job so smoking helps me to calm down. The main reason I'm here to see you today is that recently I've seen blood streaks in my phlegm. I've never been on a chubby side. Nevertheless I've been loosing weight lately and I've started to get breathless easily. But what if it is lung cancer? I know from mass media that smokers have this disease twice as often as non-smokers.

Doctor. Well, this information is a bit outdated. Nowadays world statistics shows that smokers get lung cancer 50 times more often than non-smokers! It seems to me that even Marlboro himself died of lung cancer. That is why anti-smoking campaigns are carried out all over the world. There is even international anti-smoking day. Heavy smokers often develop bronchitis. It can cause breathlessness, a stroke, bronchiectasic pneumosclerosis and lung cancer eventually. By the way, you've got breathlessness already and your face is grey. And your fingers have an interesting shape. What do your fingers make you think of?

Patient. They look like drumsticks. And the nails are kind of convex. I remember I used to have a drum with drumsticks just like my fingers when I was a child.

Doctor Yes, you're right. The symptom of drumstick fingers arises at bronchial lesions, in smokers especially. Charred lungs and constant shortage of oxygen in your organism are one of the reasons of nails of such a form. Please tell me if you're suffering from a lot of phlegm.

Patient. I've got a lot of it, doctor. In the morning I produce half a glass of phlegm literally.

Doctor. There are a lot of different symptoms for a variety of illnesses. I can see some symptoms of a disease in you. If a patient like you has got a cough with profuse phlegm, hemoptysis and drumstick fingers, we can talk of an old and sure symptom of bronchiectasic disease. In this situation a smoker's bronchi change so strongly that a set of pocket-shaped enlargements appear. They're called bronchiectasias. During the night phlegm accumulates in bronchiectasias. A lot of phlegm is produced in the morning especially. Diseased bronchi can be damaged by a strong cough which is accompanied by hemoptysis. Bronchiectasias gather rather frequently and then a serious disease called an abscess or an apostem of a lung develops. In this situation lung cancer isn't an exception either. This will result in you needing an operation and could be followed by disability and therefore cut short your career. So I would advise you to stop smoking if you don't want to become a disabled person. In the end this is the question of life and death.

Patient. Well, doctor, you've convinced me. What can I do?

Doctor. First of all it is necessary to check my hypothetical diagnosis. You have to have an X-ray of lungs. I'll give you an appointment

card for this examination. If they don't diagnose a tumor in your lungs you will have to undergo a course of out-patient treatment with a pulmonologist. You must promise me to stop smoking, otherwise the treatment won't be effective. Good-bye. Next, please.

Unit 2.2
VISUAL EXAMINATION OF CHEST

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

ankylosis	анкилоз, неподвижность суставов
asthenic	астенический
atelectasis	ателектаз, спадение легкого
barrel chest	эмфизематозная грудная клетка, бочкообразная грудная клетка
Botallo's foramen	овальное отверстие межпредсердной перегородки
clavicle	ключица
costovertebral	реберно-позвоночный
emphysematous	эмфизематозный; содержащий воздух или газ, газовый (о гангрене)
Hodgkin's disease	лимфогранулематоз, хронический злокачественный лимфоматоз (*устар.*), фибромиелоидный ретикулез (*устар.*), болезнь Ходжкина
humpback	патологический кифоз, кифотическое искривление позвоночника, горб
infraclavicular	подключичный
intrathoracic	внутригрудной, интраторакальный
joint	сустав, синартроз, синовиальное соединение
keeled breast = keeled chest	килевидная грудная клетка, куриная грудь
kyphotic	1) кифотический; 2) страдающий кифозом
mammary gland	молочная железа
Marie-Strümpell-Bekhterev disease	анкилозирующий спондилоартрит, болезнь Бехтерева–Штрюмпеля–Мари
myasthenia	миастения, болезнь Эрба–Гольдфлама
nipple	1) сосок (молочной железы); 2) выпячивание, выбухание (напр. дуги аорты); выступ
pleuropulmonary	плевролегочный
pterygoid	крыловидный, птеригиумоподобный
pulmonary heart	правые отделы сердца
rib fracture	перелом ребра
sagittal	сагиттальный, стреловидный
scoliosis	сколиоз (боковое искривление позвоночника)

spondylarthritis	спондилоартрит (воспаление межпозвоночных суставов)
supraclavicular	надключичный
transverse	поперечный
unilateral	односторонний (о локализации патологического процесса)
visceroptosis = visceroptosia	спланхноптоз, висцероптоз, симптомокомплекс Гленара

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Visual examination enables to determine not only the shape of the thorax (whether it be normal, emphysematous, pterygoid), but also its deformations, both derived from bone pathology and pleuropulmonary pathology.

A pterygoid (alar) chest is usually elongated, flattened and narrowed in both transverse and saggital dimensions. Shoulders are drooping, scapulae are wing-shaped and sticking out of the the thorax, intercostal corner is sharp, supraclavicular and infraclavicular pits are protruding, intercostal spaces are drawn in during inhalation. Such changes may be due to tuberculosis or some organic inflammatory process in the lungs and pleura with pneumosclerosis and wrinkled lungs.

An asthenic chest closely resembles the pterygoid type . It is not a result of a disease, but a sign of borderline constitution, often combined with general weakness, pallor, anemia, poor state of nourishment, weak muscles, visceroptosis etc.

An emphysematous chest is typical of an increase of the saggital dimension, position horizontal of ribs, dilated intercostal spaces, intercostal corner exceeding 45 degrees. Old physicians use to call it a "barrel chest." The pits over and below the clavicles and the intercostal spaces may be not observable and even protruding. The barrel chest looks as if it was in a state of constant inhalation. This fixed shape of the thorax may result from loss of lung elasticity (in emphysema or chronic bronchitis), and is mostly acquired, but sometimes congenital. There may also be registered partial deformation of the chest: one half protruded and some parts drawn in. Unilateral protrusion may be caused by air or fluid collected in the corresponding pleural cavity. It will disturb the mammary gland nipples symmetry, cause the rise of the shoulder of the damaged side and scoliosis, convexity towards the damaged side. Intercostal spaces will be broadened and protruded. If one side of the chest

lags behind during respiration and has some portion(s) drawn in, it may be an evidence of unilateral pleuropulmonary lesion, pulmonary atelectasis, dry pleuritis, croupous pneumonia, rib fracture, rough pleural adhesion. Severe unilateral chest pain may lead to reflex muscular contraction of the damaged side and produce asymmetry.

Besides the deformations which are already described, there may be a funnel, keeled, kyphotic chest or gibus cardiacus. These deformations may provoke the development of so called thoraco-phrenic pulmonary heart, and due to this are also significant in diagnostics.

The deformation of cardiac humpback may occur in some congenital heart diseases (Botallo'[1] duct open). Measurements and visual evaluation may register the absence of proper chest expansion on inhalation. It may result from respiratory muscles disorders (myastheniae), in costovertebral joints ankylosis (early and quite characteristic symptom of Marie-Strümpel-Bekhterev's[2] disease or chronic progressive autoimmune spondyloarthritis).

Visual examination of the chest may register vein broadening, resulting from either any initial stage of congestion in the lesser circulation, or from vein compression by intrathoracic tumors or enlarged lymph nodes. The latter may occur in pulmonary tuberculosis or cancer and in Hodgkin's[3] disease .

The presence of a net of small swollen veins in the upper third of the chest, especially in posterior interscapular area, is referred to as a positive Frank's[4] symptom. This is connected with bronchial lymph nodes enlargement and subsequent compression of veins by these nodes. Frank's symptom is believed to be one of diagnostic signs of tuberculosis, in particular, in children.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What can be determined by visual examination?*
2. *How does pterygoid chest look like in both transverse and saggital dimensions?*

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[1] See footnote 1, p. 190.

[2] Pierre Marie, French neurologist, born September 9, 1853, Paris; died April 13. 1940, Paris; Ernst Adolf Gustav Gottfried von Strümpell, Russian (later German) neurologist, born June 29, 1853, Estate Neu-Autz, Russia; died 1925, Leipzig, Germany; Vladimir Mikhailovich Bekhterev, Russian neurologist and psychiatrist, born January 20th, 1857, in Sorali, Vyatskaya region, Russia; died December 24th 1927, Moscow, U.S.S.R.

[3] Thomas Hodgkin, English physcian and pathologist, born August 17, 1798, in Pentonville, St. James Parish, Middlesex; died April 5, 1866, Jaffa, Palestine.

[4] Frank A. E., born 1884, German physician.

3. *What may cause the changes in shoulders, scapulae, intercostal corner, supraclavicular and infraclavicular pits, intercostal spaces?*
4. *What does an asthenic chest resemble?*

4. Read the task card below.

Describe the main peculiarities of visual examination following the plan:

What kinds of deformation of the chest does visual examinations reveal?
What deformations may a chest have?
What may provoke the development of so called thoracophrenic pulmonary heart?

5. Now, using the prompts from exercise 4, talk on the topic: CHEST DEFORMATIONS REVEALED BY VISUAL EXAMINATION.

Unit 2.3
PHYSICAL INVESTIGATION: PALPATION

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

cavern	1) полость, впадина; 2) каверна
cutaneous	кожный
dry rales	сухие хрипы
empyema	эмпиема (скопление гноя в плевральной полости)
fracture	1) перелом; ломать, сломать; 2) разрыв; трещина; излом
hyperalgesia = hyperalgia	гипералгезия (повышенная болевая чувствительность)
ossification	1) окостенение, оссификация; 2) образование костного вещества
rigidity	ригидность, оцепенелость (обусловленная напряжением мышц); тугоподвижность; окоченение
trapezium	кость-трапеция (запястья)
venepuncture	венопункция, венепункция
vertebrosternal	позвоночно-грудинный
vocal fremitus	голосовое дрожание

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Palpation is an informative method of physical pulmonological diagnostics. It gives the possibility to localize the chest pain and evaluate its spreaded and probable location (cutaneous hyperalgesia, intercostal neuralgia) or its more profound (costal trauma, vertebrosternal lesion) cause. In costal fracture there is a characteristic crepitation over the lesion. Palpable crepitation of subcutaneous fat due to air bubbles in it (hypodermic emphysema) produces an impression which is difficult to forget. It may occur after pneumothorax, lung wounds, accidental lung puncture during subclavicular venepunction.

Palpation helps to estimate the extent of chest elasticity or its resistance. Exudative pleuritis, pleural tumors, ossification of the costal cartilages in senescence may result in increased thoracic resistance and rigidity.

The palpation of the apex areas and musculus trapezius may be bothering and even painful in patients with apical dry pleuritis and pleural tuberculosis.

Sometimes palpation may reveal pulsation (in purulent pleuritis — empyema pulsans) when heartbeats are palpable through purulent pleuritic exudate juxtaposed to the heart. Tumors in close proximity to the heart also may pulsate. Some dry r?les and pleura friction murmurs are also palpable.

Finally, palpation makes it possible to investigate so called "fremitus (thrembling) vocalis seu pectoralis" (vocal fremitus). This is trembling of the thoracic wall produced by vocal vibrations. It is well recognized by the palms, attached symmetrically to the chest. The palms delineate the fremitus more precisely than the fingers do. In order to register the fremitus ask the patient to pronounce loudly, when palpating, some words and phrases requiring most vocal vibration (like the Russian numeral 33 — "treed-zat' tree"). Probably, English words like "deteriorate" "territory" may present some analogues. The stronger the voice and the thinner the thoracic wall the more robust will be the phenomenon of fremitus. In females, in children, dropsy and obese patients vocal fremitus is weakened, as well as in exudative pleuritis. If the lung tissue is infiltrated by inflammatory exudate and consolidated, the fremitus vocalis is strengthened. Pulmonary caverns and large bronchiectases also increase the trembling. In emphysema, vice versa, fremitus is greatly weakened. The right bronchus is shorter and broader than the left one, thus normally causing more fremitus from right side.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What is palpation?*
2. *What opportunity does it give?*
3. *Why does palpable crepitation give an impression which is difficult to forget?*

4. Read the task card below.

Describe the main peculiarities of palpation following the plan:
What results does the palpation bring?
What sensation may a patient with apical dry pleuritis and pleural tuberculosis have while being palpated?
How can the phenomenon of vocal fremitus be revealed?

5. Now, using the prompts from exercise 4, talk on the topic: PALPATION.

Unit 2.4

PHYSICAL INVESTIGATION: TOPOGRAPHIC PERCUSSION

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

abdominal cyst	абдоминальная киста
axillary region = axillary space	подмышечная область, подкрыльцовая область
cervical vertebra	шейный позвонок
cognizant	ощутимый
comparative percussion	сравнительная перкуссия
delimitation	предотвращение распространения (патологического процесса), купирование распространения (патологического процесса)
high phrenic position	высокое положение диафрагмы
marginal	предельный, критический
plessimeter finger	палец-плессиметр
pregnancy	беременность
topographic percussion	топографическая перкуссия
trapezium	кость-трапеция (запястья)

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

Percussion is a comprehensive method of lung physical examination. Percussion may give information about the properties and extent of aerial content in the organs lying under the point of percussion.

The patient is to be percussed in the standing or sitting position. The upper muscles of the chest and arm must be relaxed to avoid the distortion of the percussion sound. Weak, severely ill persons must be supported by nurses or relatives during the percussion procedure. If a patient can by no means be kept in a vertical posture, the doctor has to limit the procedure to in the lying position only, which, of course, is not desirable. In the lying position only comparative percussion of the anterior thoracic and armpit areas is possible. The doctor's position must be most convenient for that.

Percussion is divided into *topographic* and *comparative*.

Topographic percussion is designated for the delineation of the lower lung borders. The lower lung border along the posterior scapular line from both sides will normally be over the 10th rib. In emphysema the borders are expanded down to the 12th rib. From the anterior surface of the chest the low border of the left lung is not determined due to the proximity of cardiac dullness and Traube'[1] space (gaseous bubble of the stomach). Topographic percussion reveals the excursion of the lung margins and makes it possible to estimate its symmetry. Lung margin excursion is determined by the difference between the lower lung border on maximal inspiration and maximal expiration. Normally, it is 6–8 cm along the axillary lines. Pleural adhesion, severe pain in dry pleurisy, marked emphysema — limit the lung margin excursion.

In a normal lung the interlobar borders are not recognizable by percussion. But in some kinds of pathology (croupous pneumonia, interlobar exsudative pleurisy) the interlobar borders are clearly detectable by percussion. It is well known that from behind only 2 lobes are tangible on both sides, while in frontal examination 3 lobes are discernible in the right lung (upper, middle and lower).

The rules of topographic percussion are as follows:

1) plessimeter finger must be located in parallel to the border of the dullness checked.

2) Percussion proceeds from clear sound to dull one.

[1] See above footnote 1, p. 161.

3) Lung borders should be determined by smooth percussion.

4) The side of the finger nearest to the lung locates the border.

The height of the lung apex is checked from the front over the clavicle, from behind — over spina scapulae. The smoothest percussion after J. Pleisch[1] is used for it. The normal protrusion of the lung apex from the front is 3–5 cm over the clavicle and at the back is up to the level of the 7th cervical vertebra.

When checking the apex of each lung, it is useful to determine Kronig's[2] fields. They are delineated by means of percussion along the upper edge of M. trapezius from the middle point of the muscle to both sides. The normal length of Kronig's fields is 4–8 cm. But the symmetry of Kronig's fields is much more important than its size, which varies greatly. Kronig's fields asymmetry may result from unilateral apical fibrosis or consolidation, which is especially significant in diagnostic of upper lobe tuberculosis.

A high position of the lower lung border may result from the high phrenic position due to ascites, pregnancy, flatulence, severe obesity, large abdominal cysts and tumors.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What kind of method is percussion?*
2. *What kind of information can percussion supply?*
3. *In what position is the patient to be percussed?*
4. *What is the initial state of the upper muscles of the chest and the arm to avoid percussion sound distortion? Read the task card below.*

4. Read the task card below.

Describe the main peculiarities of percussion following the plan:
What is percussion?
In what position is a patient is to be percussed?
What is topographic percussion?
What is comparative percussion?

5. Now, using the prompts from exercise 4, talk on the topic: PERCUSSION.

[1] See above footnote 2, p. 207.

[2] G. Krönig, German physician (1856–1911), the fields described in 1907.

Unit 2.5

PHYSICAL INVESTIGATION: COMPARATIVE PERCUSSION, SOUND TYPES

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

adhesion	1) адгезия, слипание; склеивание; соединение; сцепление; 2) сращение; спайка; спайкообразование; 3) заживление (раны); 4) молекулярное притяжение
apex	1) верхушка; 2) верхушка легкого; 3) верхушка корня зуба
backbone	позвоночник, позвоночный столб
bandbox sound = bandbox resonance	коробочный перкуторный звук
bronchopneumonia = bronchopneumonitis	очаговая пневмония, бронхопневмония
cracked-pot sound	симптом «треснувшего горшка»
Ellis-d'Amoiseau line	линия Эллиса — д'Амуазо
Garland's triangle	треугольник Гарланда
Grocco-Rauchfus-Korányi triangle	треугольник Грокко-Раухфуса-Кораньи
hepatization	опеченение, гепатизация
hydropneumothorax	гидропневмоторакс
laryngeal	ларингеальный, относящийся к гортани
lien = spleen	селезенка
liver	печень
lung abscess	абсцесс легкого
mediastinum	средостение
mesothelioma	мезотелиома, целотелиома
oscillation	осцилляция, колебание
pneumopericardium	пневмоперикард (наличие газа в перикардиальной полости)
pulmonary edema	отек легких
pulmonary hemorrhage	легочное кровотечение
pulmonary infarction	инфаркт легкого
pulmonary tuberculosis	туберкулез легких
resolution	1) растворение; 2) разложение; расщепление; 3) рассасывание (напр. опухоли); разрешение (напр. воспалительного процесса)
resonance	перкуторный звук
Škoda's tone	тон Шкоды

Traube's space	пространство, промежуток Траубе
tympanic	тимпанический (о перкуторном звуке)

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo. After that read the text aloud, trying to imitate the intonation.

The aim of comparative (symmetric) percussion is to obtain information about the physical (anatomic) status of the lung. It is always performed exactly at symmetric points. The precise symmetry is essential because even an imperceptible difference in the shape or status of the thorax, the slightest curvature of the backbone or various muscular developments may change the character of the sounds.

Comparative percussion proceeds in the following standard sequence: Apex, the anterior surface along the intercostal spaces, the lateral surfaces (a patient must keep both hands on his/her head), the posterior surface (over the scapulae, the interscapular area, below the scapular corner). In the interscapular area the plessimeter finger is positioned parallels to the backbone, below the scapulae — along the intercostal spaces.

J. Škoda[1] has classified percussion sounds as follows:

1. Clear and loud; 2. Dull (dullish); 3. Full or prolonged; 4. Empty or short; 5. High; 6. Low; 7. Tympanic resonance; 8. Non-tympanic.

|1| is produced by percussion over normal lung tissue. Its timbre is usually not musical. Its pitch and amplitude depend on age, fat and muscle development and individual properties of the thoracic wall.

|2| is sometimes called "femoral sound" because it is produced by percussion of the thigh. It is audible during percussion over the inner organs adjacent to the thoracic wall (heart, liver, lien — which corresponds to cardiac, liver, and spleen dullness). Dull sounds over the lung result from decreased air content or complete absence of air in some portion of the lung.

Dull sounds may be of various pulmonary and extrapulmonary origin:

Pulmonary nature of dull lung percussion sound are as follows:

1. Bronchopneumonia; 2. Croupous pneumonia in hepatization phase; 3. Pulmonary tuberculosis; 4. Lung abscess; 5. Large pulmonary infarc-

[1] Josef Škoda (1805–1881), Bohemian clinician, born December 10, 1805, Pilsen, Bohemia; died June 13, 1881, Vienna, Austria.

tion and pulmonary hemorrhage; 6. Lung tumors; 7. Pulmonary edema, especially over lower lobes; 8. Atelectasis.

Dull sounds may be of pleural origin: due to fluid or tumor in pleural cavity, massive adhesions of pleural layers, exudative pleuritis, hydrothorax, mesothelioma even if the lung itself is not involved. Scar tissue in the lung also produces dullness of percussion sound.

The upper border of the dullness caused by an intrapleural fluid has a peculiar shape, referred to as Ellis-D'amoiseau[1] line. This line is created by negative pressure in the pleural cavity. As a result of mediastinum displacement by an exudate, the zones of less resonance loss may be formed. They are called Garland's[2] triangle and Grocco–Rauchfus–Korányi[3] triangle. If the pleural cavity contains some air combined with fluid (hydropneumothorax), the oblique Ellis -Damoiseau line on percussion is not detectable. Instead, the upper border of dullness forms a horizontal line along the fluid level.

Percussion sound altitude depends on the number of oscillations in percussion. Expanded lungs are responsible for low tones (in emphysema, bronchial asthma). High tones are produced on account of a limited area of infiltration within the lung tissue (initial stage of apical tuberculosis).

An empty or short sound (4) is produced by the percussion of the portion with less air content (infiltration). Principally, dullness and shortness of percussion sounds are not identical.

A tympanic sound (the term traced to "tympani drum") is close to musical as it is possible to evaluate its pitch and even initiate it by the voice.

The shorter is the column of vibrating air in percussion, the higher and more musical will be the tympanic sound. Such sound is produced over the areas of chest, adjacent to air-containing cavities. Normally it is audible only over Traube's space (see above).

In pathologic conditions tympanic sound may occur if:

1. Fluid collected in the pleural cavity and the lung rises over the exudate (Škoda's tone)
2. The exudate in pleuritis presses on the lung, but its layer is thin.
3. In relaxation of the upper lobe due to inflammatory infiltrate of the lower lobe.
4. Simultaneous presence of air and fluid in the alveoli due to edema of the lungs or croupous pneumonia in a rush phase without resolution.

Percussion may produce tympanic sounds over air cavities with smooth walls, if the diameter of the cavity is not smaller than the size of

[1] Richard White Bernhard Ellis (1902–1966), English paediatrician, born August 25, 1902, Leicester; died 1966, London. Louis Hyacinthe Céleste Damoiseau, French physician, 1815–1890. Paris.

[2] George Minott Garland, American internist, born 1848; died March 2, 1926.

[3] Pietro Grocco — see above footnote 4, p. 246. Karl Andreyevich Rauchfuss — Russian paediatrician and laryngologist, born November 27/December 9, 1835, St. Petersburg; died November 14, 1915, St. Petersburg (see p. 281, below). Friedrich von Korányi, Hungarian physician, born December 20, 1828, Nagy-Kállo died 1913.

a walnut. The walls of the cavity must be resilient enough to produce a drum-like effect.

If the air cavity is deeply seated, it is disclosed by percussion only if its size is large (physiologic cavities: laryngeal, tracheal, stomach, intestinal, the latter may be in the thorax in big phrenic hernias; pathologic cavities: caverns, pneumothorax, bronchiectases).

Biermer's bandbox sound is a transient between tympanic and non-tympanic. It closely resembles the sound produced by knocking on the empty box. It may be observed in loss of lung elasticity (emphysema, bronchial asthmatic attack).

Laennec's[1] metallic tinkling is produced over smooth-wall caverns more than 6 cm in diameter, and also in pneumothorax and pneumopericardium. Laennec also described another percussion sound: a "cracked-pot" sound or, in French: "bruit de pot fêlé." It is reproduced by beating the knee with the palms joined together. It may be normal in small children during the crying or forced expiration. In pathologic condition this phenomenon is typical for lung cavities joined with bronchus through narrow aperture or pneumothorax communicating with bronchus. This sound characteristically becomes clearer when a patient opens his/her mouth. The sound may acquire a metallic overtone. It is a jingling sound produced by the air moving through the narrow aperture, portion by portion, with forced pushes.

3. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner. After that add more questions of your own and practice them with your partner.

1. *What is comparative (symmetric) percussion aimed at?*
2. *How is it performed?*
3. *Why is the precise symmetry essential?*
4. *In what sequence is comparative percussion performed?*

4. Read the task card below.

Describe the main peculiarities of percussion sounds following the plan:

What is comparative percussion used for?
How are percussion sounds classified?
In what pathologic conditions may they occur?

5. Now, using the prompts from exercise 4, talk on the topic: PERCUSSION SOUNDS.

[1] René-Théophile-Hyacinthe Laennec, French physician, born February 17, 1781, Quimper, Bretagne; died August 13, 1826, Kerlouarnec, Bretagne.

Module 3

AUSCULTATION FROM THE HISTORY OF AUSCULTATION

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

auscultation	аускультация, выслушивание
bed-patient	лежачий больной
binaural	бинауральный (относящийся к обоим ушам)
bronchial respiration	бронхиальное дыхание
bubbling rales	влажные хрипы
carnification = carneous degeneration	карнификация (патологическое изменение легочной ткани, при которой она приобретает консистенцию и вид сырого мяса)
crepitation	крепитация (ощущение похрустывания или потрескивания, возникающее при пальпации или аускультации)
dry rales	сухие хрипы
examination	обследование
exhalation	выдох
faint	обморок, синкопе
hyperventilation	гипервентиляция легких, гиперпноэ
hypocapnia	гипокапния (пониженное содержание двуокиси углерода в крови)
inspiration	вдох
internals	внутренние органы
Leonardo da Vinci phenomenon = Da Costa syndrome	феномен Леонардо да Винчи = синдром Да Коста
orthostatis	ортостаз
pediatric	педиатрический
pleural rub	шум трения плевры
post-mortem	вскрытие трупа, аутопсия
respiratory	дыхательный
souffle	нежный дующий шум (при аускультации)
stethophonendoscope	стетофонендоскоп
stethoscope	стетоскоп

unconscious state patient	больной, находящийся в бессознательном состоянии
vertigo	головокружение
vesicular respiration	везикулярное дыхание

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

As a matter of fact **auscultation** means listening to sound phenomena connected with the activities of internals. Auscultation is used for the diagnostics of the derangements of the activities of internals. In tissues the act of breathing causes vibrations reaching the surface of the human body. Usually these vibrations are not audible at a distance, but they can be audible provided the ear is placed against a patient's body in the case immediate auscultation or on condition that special devices of various construction named stethoscopes are used in case of mediate auscultation.

It was R. T. H. Laennec who described the methods of auscultation in 1816. Later he introduced auscultation into clinical practice in 1819. He also invented the stethoscope. R. T. H. Laennec substantiated the clinical value of auscultation on the basis of comparing the data obtained by auscultation with the data obtained by post-mortem examinations. He described nearly all auscultatory symptoms. He also gave them the names which are used in medicine even to the present day, for example dry rales, bubbling rales, vesicular respiration, bronchial respiration, crepitation, pleural rub etc. Since then auscultation has been an obligatory method in the diagnostics of lung diseases and heart diseases.

Methods of auscultation have been practiced in Russia since 1824. Step by step the stethoscope was improved by P. A. Piorry, F. G. Yanovsky and other scientists. One of the inventors of binaural stethoscope was a Russian pediatrician N. F. Filatov. Later stethophonendoscopes were invented. They are binaural acoustic devices supplied with flexible sound conductors which are simple and practical in auscultating patients, especially bed-patients and unconscious state patients.

Stethophonendoscopes for the auscultation of adults and stethophonendoscopes for the auscultation of children, or pediatric stethophonendoscopes, differ from each other. Pediatric stethophonendoscopes have specific acoustic properties and are supplied with a head of a smaller diameter than usual stethophonendoscopes which are used for

the auscultation of adults. There are also electronic stethoscopes which are not so widely used as they do not have any noticable advantages in comparison with ordinary stethoscopes and are difficult to manage too.

You can auscultate your patient under examination in any appropriate position. Nevertheless a sitting position of a patient with his or her hands on the lap is the best. Such position provokes the maximum relaxation of respiratory muscles. It is possible to auscultate your patient in a standing position but you have to remember that hyperventilation and hypocapnia develop more often in orthostatis. This situation can cause vertigo and even faints owing to the oppression of the respiratory center (Leonardo da Vinci[1] phenomenon, also known as a component of Da Costa's[2] syndrome). If you want the stethoscope to be pressed to the skin closer, you should support your patient the on opposite side with the free hand.

At auscultation the duration and the force of the souffle both at inspiration and at exhalation are estimated and then comparative auscultation (comparative percussion analogue) is carried out in the similar points of the opposite part.

3. Do the following statements agree with the information given in the text?

Write

TRUE *if the statement agrees with the information*
FALSE *if the statement contradicts the information*

1. Auscultation means listening to sound phenomena connected with the activities of lungs.
2. Auscultation is used for the diagnostics of the derangements.
3. In tissues the act of breathing causes vibrations reaching the surface of the lungs.
4. Usually these vibrations are not audible at all, but they can be audible provided the ear is placed against a patient's body.
5. The stethoscope was invented by R. T. H. Laennec.
6. Nearly all auscultatory symptoms were described by P. A. Piorry.
7. Dry rales, bubbling rales, vesicular respiration, bronchial respiration, crepitation, pleural rub are different types of respiration.
8. Auscultation is a method that can be used in the diagnostics of lung diseases and heart diseases.
9. Methods of auscultation have been practiced in Russia for about 200 years.

[1] Leonardo di ser Piero da Vinci (April 15, 1452, village of Anchiano near town of Vinci, Italy — May 2, 1519, Amboise, France) was an Italian Renaissance polymath: painter, sculptor, architect, musician, scientist, mathematician, engineer, inventor, anatomist, geologist, cartographer, cryptographer, botanist and writer.

[2] Jacob Mendez Da Costa, was an American internist, born February 7, 1833, St. Thomas, Danish West Indies; died 1900, USA.

10. It is stethoscopes which are especially useful while auscultating patients, especially bed-patients and unconscious patients.
11. Stethophonendoscopes for the auscultation of adults have a head of smaller diameter than pediatric stethophonendoscopes.
12. Nowadays electronic stethoscopes are widely used.
13. A sitting position of a patient with his or her hands on the lap provokes the maximum relaxation of respiratory muscles.
14. It is never possible to auscultate your patient in a standing position.

4. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner.

1. *What is auscultation?*
2. *What is auscultation used for?*
3. *In what conditions can the vibrations in tissues be audible?*
4. *Who was the first to describe the methods of auscultation?*
5. *Who invented the stethoscope?*
6. *What role did Laennec play in terms of theory and practice of auscultation?*
7. *How long have the methods of auscultation been practiced in Russia?*
8. *How do the stethophonendoscopes for the auscultation of adults and the stethophonendoscopes for the auscultation of children differ?*
9. *In what position is a patient auscultated best of all? Why?*

5. Match the words and expressions in column A with the words and expressions in column B.

A	*B*
sound	diseases
clinical	rub
post-mortem	muscles
dry	rales
vesicular	stethoscope
pleural	practice
heart	conductors
binaural	respiration
sound	phenomena
respiratory	examinations

6. Complete the sentences below.

1. Auscultation means listening to sound **p**_____ connected with the activities of **i**_____.

2. Auscultation is used for the diagnostics of the **d**_____. of the activities of internals.
3. The act of breathing causes **v**_____ reaching the surface of the human body.
4. R. T. H. Laennec substantiated the clinical **v**_____ of auscultation and described nearly all auscultatory **s**_____.
5. R. T. H. Laennec gave auscultatory symptoms the names which are used in medicine now, for example dry rales, **b**_____ rales, vesicular respiration, **b**_____ respiration, crepitation, **p**_____ rub and some others.
6. One of the inventors of **b**_____ stethoscope was a Russian pediatrician N. F. Filatov.
7. Stethophonendoscopes are simple and useful for auscultating **b**_____ and unconscious state patients.
8. Stethophonendoscopes for the auscultation of adults and **p**_____ stethophonendoscopes differ.
9. A sitting position of a patient at auscultation provokes the maximum relaxation of **r**_____ muscles.
10. At auscultation the duration and the **f**_____ of the souffle both at inspiration and at **e**_____ are estimated.

7. Unjumble the words below.

cnasutatioul
einrnalst
tspteoscoeh
tcepiotrain
ihocappyna
toroshtatis
erotigv

8. Read the task card below.

Describe the main peculiarities of auscultation following the plan:
What is auscultation?
What historical background does auscultation have?
How is the process of auscultation performed?

9. Now, using the prompts from exercise 8, talk on the topic: AUSCULTATION.

10. Translate into English.

1. Аускультация — это выслушивание звуковых феноменов, связанных с деятельностью внутренних органов, применяемое для диагностики ее нарушений.
2. Методику аускультации первым описал Лаэннек в 1816 году, а в 1819 году ввел ее в клиническую практику.

3. Лаэннек описал и дал название почти всем аускультативным симптомам, используемым во всем мире и в наши дни: сухие и влажные хрипы, везикулярное и бронхиальное дыхание, крепитация, плевральные шумы и др.
4. С 1824 г. аускультация стала применяться в России. Русский врач-педиатр Н. Ф. Филатов — один из изобретателей бинаурального стетоскопа.
5. Позже появились стетофонендоскопы — бинауральные акустические приборы с гибким звукопроводом, которые просты и удобны при выслушивании больных, особенно лежачих, постельных больных, или больных, находящихся в бессознательном состоянии.
6. Педиатрические стетофонендоскопы обладают специфическими акустическими свойствами и снабжаются головкой меньшего диаметра, чем у стетофонедоскопа для выслушивания взрослых лиц.
7. Выслушивать больного можно в любом положении, но лучше в положении «сидя» с положенными на колени руками. Такое положение способствует максимальному расслаблению дыхательной мускулатуры.
8. Чтобы стетоскоп плотнее прилегал к коже, целесообразно свободной рукой придерживать больного с противоположной стороны.
9. При аускультации оценивают характер, продолжительность и силу дыхательных шумов как на вдохе, так и на выдохе, а затем проводят сравнительную аускультацию (аналогично сравнительной перкуссии) в аналогичных точках противоположной стороны.

RESPIRATORY SOUNDS AND SOME TYPES OF RESPIRATION

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

afferent	приводящий
alveolar respiration	альвеолярное дыхание
alveole	альвеола
atrophy	1) атрофия (органа, ткани); 2) истощение
bronchial (laryngotracheitic) respiration	бронхиальное (ларинготрахеальное) дыхание
bronchial asthma	бронхиальная астма
bronchiole	бронхиола
bronchus (pl. bronchi)	бронх
compliance	изменение объема легких при колебаниях давления

croupous pneumonia	крупозная пневмония
edema	отек
emphysema	эмфизема (растяжение органа или ткани воздухом)
infiltration	инфильтрация
interalveolar septa	межальвеолярная перегородка
interrupted (saccadic) respiration	прерывистое (саккадированное) дыхание
lobe	доля (легкого)
lumen	просвет; полость трубчатого органа
mucous	слизистый
pleural effusion	плевральный выпот
pleural exudate	плевральный экссудат
pleural friction rub	шум трения плевры
pneumothorax	пневмоторакс (наличие избытка воздуха или газа в плевральной полости)
puerile (children's) breath	пуэрильное (детское) дыхание
pulmonary tissue	легочная ткань
respiratory sound	дыхательный шум
rough respiration	жесткое дыхание
secretion	секреция, секрет (желез)
shoulder-blade = scapula (*pl.*: scapulae)	лопатка
site	участок
thorax	грудная клетка
throat	горло, глотка
thyrotoxic tremor	тиротоксический тремор
trachea	трахея
tuberculosis	туберкулез
tumor	опухоль
vesicular respiration	везикулярное дыхание

2. Without looking into the text listen to the recording.

Say what information you have gathered.
Listen to the text again.
Now, read the text silently, trying to grasp all the details of the contents.
Then, read it simultaneously with the speaker, trying to catch up with the tempo.
After that read the text aloud, trying to imitate the intonation.

First of all it is necessary to pay attention to ***the basic respiratory sounds*** such as ***vesicular, or alveolar, respiration*** which is audible above the lungs, and to ***bronchial, or laryngotracheitic respiration,*** which is

audible above the throat, the trachea and above large bronchi. The best way to provide for the best audibility of the basic respiratory sounds is to ask your patient to breathe through the nose the mouth closed.

At pulmonary pathology alongside with the basic respiratory sounds some additional or secondary sounds such as rales, crepitation and pleural friction rub can be audible. Secondary sounds are better audible when your patient breathes through the open mouth.

The respiratory sound in alveoles is named ***vesicular respiration.*** It is formed when taking a breath accompanied with gradual consecutive filling a great number of pulmonary alveoles, blended in the form of a souffle audible during the whole phase of inspiration. It reminds of a sound of drinking tea from a saucer with sucking up the liquid with lips. This sound can also continue at the beginning of exhalation, but then it fades quickly so that it can not be heard during the subsequent two thirds of the phase of exhalation. Vesicular respiration in norm is best audible on the front surface of the thorax below the second rib as well as in axillary areas and also under the corners of shoulder-blades, i. e. in the areas which contain most of the pulmonary tissue. Therefore, in the area of the apexes of the lungs vesicular respiration is always weakened. It is necessary to take into account that in norm at comparative auscultation, the expiration from the right is longer and louder, since the right bronchus is wider and shorter than the left one.

Vesicular respiration can be both weakened and strengthened. It may grow louder without changing its character, and the bigger the lung compliance is, the stronger vesicular respiration becomes. Therefore, vesicular respiration is weaker over those areas of a lung which have lost the ability to extend at taking a breath and to contract at exhalation on account of some pathologic processes, or when the process of inspiratory filling of the corresponding part of a lung with air is complicated or rendered impossible due to the narrowing and congestion of bronchi. Pathological weakening of vesicular inspiration can be connected with significant reduction of the quantity of alveoles as a result of their atrophy and gradual destruction of interalveolar septa and the formation of larger vesicles which cannot contract at exhalation.

In the case of lung emphysema vesicular respiration is normally weakened, because respiratory vibrations of a lung which has been overextended for a long time are rather subdued. Weakened vesicular respiration is often audible in severe bronchitis and in bronchial asthma, while it is not audible at all above separate sites of a lung where afferent bronchi are obstructed with secretion. Vesicular respiration is completely absent when obstructing bronchi are obstructed by tumors or foreign bodies above particular areas of a lung.

In tuberculosis vesicular respiration above diseased sites, e. g., above the top, is often weakened and may be absent. The weakening of vesicular respiration, and its absence above pleural exudates, can be an important indication of pleural effusion, as exudates are poor sound conductors and, having pushed a lung aside from the thorax, they render this lung motionless. Above the pneumothorax vesicular respiration is alto-

gether absent. In adhesive pleuritis vesicular respiration is often weakened owing to the reduction of the lung compliance.

Physiological shift of vesicular respiration towards weakening or strengthening always occurs simultaneously and equally in both halves of the thorax. In a pathological state vesicular respiration changes either simultaneously in both lungs, or in one of the lungs only, or in a limited site of one lobe of the lung. The change of vesicular respiration depends on the quantity of active alveoles, the quality of their walls, on the speed of their filling with air, and also on the duration and force of a phase of inhalating or exhaling.

A weakened vesicular respiration is observed at the initial stage of croupous pneumonia, since in this case alveolar walls are swallen and the amplitude of their vibrations during inhalation.

Vesicular respiration can be ***interrupted or saccadic***. This is a kind of vesicular respiration in which a phase of taking a breath consists of separate interrupted inhalations with small pauses between, them which are mostly synchronous with the heart beat. In this situation exhalation does not usually change. This can be observed when auscultating a patient in a cold room, in the case of nervous trembling or excitement of a patient and in thyrotoxic tremor. If ***saccadic*** breath appears in a limited area of the lung, this is an indication that there is an inflammatory process in small bronchi. This kind of breath often occurs in tuberculous infiltration in the top area lungs apexes.

In children vesicular respiration in norm is louder than in adults. It is called puerile (children's) respiration.

A coarser vesicular respiration is called rough. This kind of respiration is observed in abrupt non-uniform narrowing of small bronchi lumen and bronchioles caused by the inflammatory edema of their mucous, which is observed in bronchitises and connected with the turbulence of the passing air stream. It is rather rough respiration from bronchial one that is audible in bronchitis. Nevertheless one will often hear students mistakenly declaring: "Once this patient has bronchial respiration he has bronchitis".

3. Do the following statements agree with the information given in the text?

Write

TRUE *if the statement agrees with the information*
FALSE *if the statement contradicts the information*

1. Vesicular respiration and alveolar respiration are audible in the lungs.
2. Bronchial respiration is audible above the lungs.
3. The best way to provide for the best possible audibility of the basic respiratory sounds is asking your patient to breathe through the mouth open.

4. Rales, crepitation and pleural friction rub are additional or secondary respiratory sounds can be audible.
5. Secondary sounds are better audible on condition that your patient breathes through a nose with his or her mouth closed.
6. The respiratory sound in alveoles is named vesicular respiration.
7. Vesicular respiration in norm is better audible on the front surface of the thorax below the third rib.
8. The bigger the lung compliance is, the weaker the vesicular respiration becomes.
9. Pathological weakening of vesicular inspiration can be connected with a significant increase of the quantity of alveoles.
10. A weakened vesicular respiration is audible in severe bronchitis and in bronchial asthma.
11. In tuberculosis vesicular respiration above diseased sites is often weakened.
12. The strengthening of vesicular respiration can be an important indication of pleural effusion.
13. Above pneumothorax vesicular respiration is not present at all.
14. In pathological state vesicular respiration changes either simultaneously in both lungs or in one of the lungs only or in the limited site of one lobe of the lung.
15. Vesicular respiration can be interrupted or saccadic.
16. In adults vesicular respiration in norm is louder, than in children.

4. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner.

1. *What are the basic respiratory sounds?*
2. *What is the best way to provide to provide for the best audibility?*
3. *What additional or secondary sounds can be audible alongside with the basic respiratory sounds in pulmonary pathology?*
4. *What respiratory sound is named vesicular respiration?*
5. *How is vesicular respiration formed?*
6. *In which area is vesicular respiration best audible?*
7. *What does the strength of the vesicular respiration depend on?*
8. *What can pathological weakening of vesicular respiration be connected with?*
9. *Why is vesicular respiration normally weakened in lung emphysema?*
10. *What is characteristic of vesicular respiration in tuberculosis?*
11. *What is characteristic of physiological shift of vesicular respiration towards weakening or strengthening?*
12. *What kind of vesicular respiration is called interrupted or saccadic?*
13. *What is characteristic of vesicular respiration in children?*

14. What is characteristic of rough vesicular respiration?

5. Match the words and expressions in column A with the words and expressions in column B.

A	*B*
alveolar	breath
pulmonary	pleuritis
comparative	breath
lung	auscultation
interalveolar	pneumonia
bronchial	respiration
pleural	tremor
adhesive	pathology
croupous	septa
thyreotoxic	effusion
saccadic	compliance
inflammatory	asthma
puerile	process
inflammatory	edema

6. Complete the sentences below.

1. Vesicular or **a**_____ respiration and bronchial or **l**_____ respiration are basic **r**_____ sounds.
2. Additional or secondary respiratory sounds are rales, **c**_____ and pleural friction **r**_____.
3. The respiratory sound in **a**_____ is named **v**_____ respiration.
4. Vesicular respiration in norm is best audible on the front surface of the **t**_____ below the second rib.
5. The bigger the lung **c**_____ is, the stronger vesicular respiration becomes.
6. In lung **e**_____ vesicular respiration is normally weakened.
7. Above **p**_____ vesicular respiration is not present at all.
8. In adhesive **p**_____ vesicular respiration is often weakened.
9. Weakened vesicular respiration is observed at the initial stage of **c**_____ pneumonia.
10. Vesicular respiration can be interrupted or **s**_____.
11. In children vesicular respiration is called **p**_____ respiration.

7. Unjumble the words below.

inesriratop
taraceh
reals
usoflef
ealationxh

toxrha
brohuncs
hempsemay
tasham
uplritise
eeamd

8. Read the task card below.

Describe the main peculiarities of respiratory sounds and some types of respiration following the plan:

What are the basic respiratory sounds?
What is vesicular respiration?
How is vesicular respiration characterized?

9. Now, using the prompts from exercise 8, talk on the topic: RESPIRATORY SOUNDS AND SOME TYPES OF RESPIRATION.

10. Translate into English.

1. Основные дыхательные шумы лучше выслушивать, если больной дышит через нос при закрытом рте.
2. Везикулярное или альвеолярное дыхание выслушивается над легкими, а бронхиальное или ларинготрахеальное дыхание выслушивается над гортанью, трахеей и над крупными бронхами.
3. Побочные шумы лучше выслушиваются при более глубоком дыхании больного через открытый рот. При легочной патологии наряду с основными дыхательными шумами могут выслушиваться дополнительные или побочные шумы: хрипы, крепитация и шум трения плевры.
4. Дыхательный шум, возникающий в альвеолах, называется ***везикулярным дыханием***. Везикулярное дыхание образуется на вдохе при постепенном последовательном заполнении огромного количества легочных альвеол, что суммируется в виде продолжительного нарастающего мягкого дующего шума, который можно выслушать на протяжении всей фазы вдоха.
5. Везикулярное дыхание может быть как ослабленным, так и усиленным. Оно становится громче при глубоком дыхании и тем сильнее, тем больше растяжимость легких. Патологическое ослабление везикулярного дыхания может быть связано со значительным уменьшением количества альвеол в результате их атрофии и постепенно гибели межальвеолярных перегородок и образования более крупных пузырьков, которые не могут спадаться на выдохе.

6. При эмфиземе легких везикулярное дыхание обыкновенно ослаблено, так как дыхательные колебания длительно и чрезмерно растянутого легкого весьма незначительны.
7. Физиологическое изменение везикулярного дыхания в сторону ослабления или усиления всегда происходит одновременно и одинаково в обеих половинах грудной клетки. Изменение везикулярного дыхания зависит от количества сохранившихся альвеол, качества их стенок, от скорости заполнения их воздухом, а также от продолжительности и силы фазы вдоха или выдоха. Ослабленное везикулярное дыхание наблюдается в начальной стадии крупозной пневмонии, так как при этом набухают альвеолярные стенки, и уменьшается амплитуда их колебаний во время вдоха.
8. Везикулярное дыхание может быть ***прерывистым или саккадированным.*** Это везикулярное дыхание, фаза вдоха которого состоит из отдельных коротких прерывистых вдохов с малыми паузами между ними.
9. У детей везикулярное дыхание в норме громче, чем у взрослых и называется ***пуэрильным (детским) дыханием.***
10. Более грубое по характеру везикулярное дыхание называется **жестким.** Такое дыхание наблюдается при резком и неравномерном сужении просвета мелких бронхов и бронхиол вследствие воспалительного отека их слизистой, что наблюдается при бронхитах и связано с завихрением проходящей струи воздуха.

FOCUS ON THE TYPES OF RESPIRATION

1. Listen to the recording. Repeat each medical term during the pause in the recording. Mind the stress.

abscess	абсцесс, гнойник
amphoric respiration	амфорическое дыхание
aperture	апертура, щель
apex	верхушка (легкого)
atelectasis	ателектаз, спадение легкого
bifurcation	бифуркация (разделение трубчатого органа на две ветви)
bronchotracheal respiration	бронхотрахеальное дыхание
carnification	карнификация
cavern	каверна
cavernous tuberculosis	кавернозный туберкулез
condensation	уплотнение (легочной ткани)
confluent pneumonia	сливная пневмония
glottis	голосовая щель
humming rales	жужжащие хрипы

interscapular	межлопаточный
lobar pneumonia	долевая пневмония, лобарная пневмония, лобит
metal respiration	металлическое дыхание
pleuropneumonia	плевропневмония
pneumosclerosis	пневмосклероз, пневмофиброз, склероз легких
presternum	рукоятка грудины
pulmonary infarction	инфаркт легких
pulmonary tuberculosis	туберкулез легких
souffle	нежный дующий шум (при аускультации)
stenotic respiration	стенотическое дыхание
sternum	грудина
the angle of Louis	угол Людовика
thoracic vertebrae	грудной позвонок
timbre	тембр
uncertain respiration	неопределенное дыхание
uneven respiration	неравномерное дыхание

2. Without looking into the text listen to the recording.

Say what information you have gathered.

Listen to the text again.

Now, read the text silently, trying to grasp all the details of the contents.

Then, read it simultaneously with the speaker, trying to catch up with the tempo.

After that read the text aloud, trying to imitate the intonation.

Respiratory sounds which are perceived to as abnormally loud and high-pitch against noiseless respiration are called ***rough respiration***. Such kind of respiration occurs out mainly during exhalation, and it is coarser and longer than respiration in healthy people. ***Rough respiration*** is the characteristic of initial or incomplete condensation of pulmonary tissue. If it is audible above one of the apexes of the lungs this can be treated as an early symptom of pulmonary tuberculosis. It should be born in mind that above the right apex, in norm rough breath is regularly observed. Thus, rough respiration is a variant of vesicular respiration, but it is coarser when the phase of breath and the phase of exhalation are reinforced.

Bronchial respiration audible in auscultation reminds of a sound originating from an abrupt pronunciation of the Russian letter "Х". It is characterized by very fast (high) vibrations with the frequency from 1000 up to 3000 units per second. In breathing in it appears in the trachea and in the throat at the time when the air is flowing through the narrow glottis and into the wider trachea where whirls appear. Such a sound propagates with air all over the bronchial tree. During exhala-

tion, when the glottis is more narrowed, than in taking a breath, the sound becomes stronger, longer and even coarser as the air flow gets into a wider throat area, where the whirls are also formed. This kind of respiration is called ***laryngotracheitic*** after the place of its formation. Bronchial respiration differs from vesicular respiration not by its force but by the character of its sounds which are higher. Healthy people do not have pure bronchial respiration above their lungs, since bronchi are totally surrounded by pulmonary tissue tending to weaken and muffle high sound vibrations.

In norm, bronchial respiration is well audible above the throat, the trachea and in the places of tracheal bifurcation projection to the thorax (in the area of the presternum and at the place where the presternum is fixed with the body of sternum; this place, which located in the interscapular space at the front and at the level of the third and the fourth thoracic vertebrae at the back, is called the angle of Louis).

Pathological bronchial respiration can be audible above the lungs only under certain conditions. The main condition is the condensation of pulmonary tissue particularly in the area where the lung has become airless. Remaining practically unchanged, bronchial respiration, which appears in the trachea and the throat, penetrates through the condensed tissue. Certainly, bronchial respiration can be audible above the lungs, provided that adductor bronchi are patent and not obstructed with slime, neoplasms or a heterogeneous body. In the latter cases bronchial respiration will not be audible above condensed pulmonary tissue and the souffle might not be audible at all (the "mute" lung). Therefore, in pneumonia bronchial respiration is not audible above all the area of inflammatory infiltration, in some sites respiration is weakened and may be entirely absent. Thus, if in the area above the lungs pure bronchial respiration is audible, it is a sure sign that the lung is airless and condensed. Therefore bronchial respiration is typical of every infiltration process located directly in the chest or near it (croupous pneumonia, tuberculosis, pulmonary infarction even at caverns adjacent to the chest wall, but surrounded by inflamed and condensed pulmonary tissue). On the contrary, if the cavern or the focus of condensation are located deep in the lungs and surrounded by aerial pulmonary tissue, when not only bronchial respiration but even vesicular respiration can be audible in this area. Condensation of the lung can be caused by the formation of the connective tissue in the lungs (pneumosclerosis, pulmonary lobe carnification appeared owing to croupous pneumonia).

Depending on the degree and the area of condensation of lungs bronchial respiration can be unequal in terms of force and timbre. In the case of superficial location and massive condensation of a site of pulmonary tissue, bronchial respiration, perceived as though it appears under a doctor's ear, is audible. It is a typical situation for the second stage of croupous pneumonia, or pleuropneumonia, or lobar pneumonia, when the whole lobe of a lung is affected. Nevertheless, bronchial respiration can also be audible in focal pneumonia (bronchial pneumonia) when some

inflammatory infiltration centers are located closely, or even combined with the formation of a larger focus of condensation (confluent pneumonia).

In compressive atelectasis lung bronchial respiration can be very gentle. Weakened bronchial respiration is audible when pleural effusion is present and the lung behind it is condensed, which is intensified by an exudate (compressive atelectasis). Bronchial respiration can also be audible above the lung abscess or the caverns which have emptied themselves of their content and communicate with bronchi. Thus the caverns themselves can play the role of resonators strengthening the timbre of bronchial respiration because air turbulence also occurs while the air is coming coming in through relatively narrow bronchi into wider caverns.

If, as a result of an abscess or cavernous tuberculosis, a smooth-surface cavern of the size not less than a walnut, communicating with a large bronchus, appears in a lung, then there may appear very high sonorous overtones similar to these that can be imitated if you blow through a narrow neck of an empty glass or clay vessel (a bottle or a jug) as a result of bronchotracheal respiration and strong resonance. Such kind of respiration is called amphoric (from a Greek word "amphora" that means "a clay vessel with a narrow throat"). It corresponds to the metal tone at percussion.

When a trachea or a large bronchus is narrowed (for example in case of a tumor) laryngotracheitic respiration, which is strengthened considerably, is called ***stenotic.***

There is also ***metal respiration,*** characterized by a very high timbre and a loud sound reminding of a sound appearing at striking metal. Metal respiration can be audible in open pneumothorax when the air in the pleural cavity communicates with the outer air through an aperture.

Historically the authors used to classify respiration of uneven ("mild") character of vesicular type, but more discontinued, as ***uneven respiration.*** This kind of respiration is transient towards humming rales which is often observed at bronchitis and sometimes at the initial stage of pulmonary tuberculosis.

Traditionally they classified respiration as ***uncertain*** if it was impossible to define either as vesicular or as bronchial. Such respiration appears above the beginning or incomplete infiltrations of pulmonary tissue, where small focuses of condensation and pulmonary tissue containing air alternate, i. e., in the area where the sites of bronchial respiration and vesicular respiration alternate. This type of respiration would be worth concidering in the case of pleural exudates, where above it and at loud rales, respiration is too weak for its character to be defined.

3. Do the following statements agree with the information given in the text?

Write

TRUE *if the statement agrees with the information*
FALSE *if the statement contradicts the information*

1. Rough respiration is found out mainly in breathing.
2. Rough respiration is shorter than respiration in norm.
3. Rough respiration which is audible above one of the apexes of the lungs can be an early symptom of infarction.
4. Bronchial respiration is characterized by very slow vibrations.
5. Bronchial respiration differs from vesicular respiration by its force.
6. Both healthy and unhealthy people have pure bronchial respiration above their lungs.
7. In norm bronchial respiration is well audible above the throat, the trachea and in the places of the tracheal bifurcation projection to the thorax.
8. Pathological bronchial respiration can always be audible above the lungs.
9. In pneumonia bronchial respiration is not audible above all the area of the inflammatory infiltration.
10. Bronchial respiration is typical of some infiltration processes occuring directly at the chest or near it.
11. Bronchial respiration is equal in terms of force and timbre.
12. Weakened bronchial respiration is audible when pleural effusion is present and the lung behind it is condenced.
13. Amphoric respiration corresponds to the metal tone at percussion.
14. Laryngotracheitic respiration, which is strengthened considerably, is called metal.

4. Listen to the following questions and repeat them. Mind the intonation in general and special questions. Then answer the questions and practice them with your partner.

1. *What sounds are called rough respiration?*
2. *What state of pulmonary tissue does rough respiration characterize?*
3. *What sound does bronchial respiration remind of?*
4. *What kind of respiration is called laryngotracheitic?*
5. *In which area is bronchial respiration well audible in norm?*
6. *Under what conditions can pathological bronchial respiration be audible above the lungs?*

7. *Why is bronchial respiration not audible above all the area of the inflammatory infiltration in pneumonia?*
8. *What does the force and the timbre of bronchial respiration depend on?*
9. *What is characteristic of bronchial respiration at compressive atelectasis?*
10. *Under what conditions can weakened bronchial respiration be audible?*
11. *What kind of respiration is called amphoric?*
12. *What other kinds of respiration can you mention?*
13. *How can you characterize them?*

5. Match the words and expressions in column A with the words and expressions in column B. Then listen, check and repeat.

A	*B*
pulmonary	pneumonia
thoracic	wall
inflammatory	rales
chest	vertebrae
vesicular	tuberculosis
connective	atelectasis
lobar	tuberculosis
compressive	tissue
cavernous	respiration
humming	infiltration

6. Complete the sentences below.

1. Rough respiration is found out mainly at **e**_____.
2. Rough respiration is the characteristic of initial or incomplete condensation of **p**_____ tissue.
3. If rough respiration is audible above one of the apexes of the lungs, this can be an early symptom of pulmonary **t**_____.
4. At a breath **b**_____ respiration appears in the trachea and in the throat.
5. In norm bronchial respiration is well audible above the throat, the **trachea** and in the places of the tracheal **b**_____ projection to the thorax.
6. Bronchial respiration can be audible above the lungs provided **a**_____ bronchi are patent and not obstructed with slime, **n**_____ or a heterogeneous body.
7. The condensation of the lung can be caused by the developing of the **c**_____ tissue in the lungs (pneumosclerosis, pulmonary lobe **c**_____ appeared owing to croupous pneumonia).

8. Depending on a degree and the area of the condensation of lungs bronchial respiration can be unequal in terms of force and t_____.

7. Unjumble the words below.

condensation
tuberculosis
glottis
bifurcation
presternum
vertebra
slime
infarction
timbre

8. Read the task card below.

Describe the main peculiarities of types of respiration following the plan:

What is rough respiration?
What is bronchial respiration?
What other types of respiration do you know?

9. Now, using the prompts from exercise 8, talk on the topic: FOCUS ON THE TYPES OF RESPIRATION.

10. Translate into English.

1. ***Жестким дыханием*** называются такие дыхательные шумы, которые выслушиваются как ненормально громкие и высокие. Жесткое дыхание является признаком начинающегося или неполного уплотнения легочной ткани. Если оно выслушивается над одной из верхушек легких, то может быть ранним признаком легочного туберкулеза.
2. ***Бронхиальное дыхание*** при аускультации напоминает звук, возникающий при резком произношении русской буквы «Х». На вдохе он возникает в трахее и гортани во время прохождения струи воздуха через узкую голосовую щель в более широкую трахею, где возникают вихревые движения. По месту образования такое дыхание называют ***ларинготрахеальным.***
3. От везикулярного дыхания бронхиальное дыхание отличается не своей силой, а характером звука, то есть большей высотой тонов. У здоровых лиц над легкими нигде нет чистого бронхиального дыхания, так как бронхи повсюду окружены легочной тканью, которая ослабляет и заглушает звуковые колебания высоких тонов.
4. Патологическое бронхиальное дыхание выслушивается над легкими только в определенных условиях, основным из кото-

рых является уплотнение легочной ткани, то есть там, где легкое стало безвоздушным, когда через уплотненную ткань практически в неизмененном виде проводится бронхиальное дыхание, возникающее, как указывалось, в гортани и трахее.

5. При пневмонии бронхиальное дыхание выслушивается не над всей областью воспалительной инфильтрации, при этом над некоторыми участками дыхание ослаблено и может вообще отсутствовать. Таким образом, если в каком-либо месте над легкими выслушивается чистое бронхиальное дыхание, то это — верный признак того, что здесь легкое безвоздушно и уплотнено.
6. В зависимости от степени и площади уплотнения легких бронхиальное дыхание может быть неодинаковым по силе и по тембру. При поверхностном расположении и массивном уплотнении участка легочной ткани выслушивается особенно громкое и высокое по тембру бронхиальное дыхание, возникающее как бы под самым ухом выслушивающего. Это типично доля второй стадии крупозной пневмонии (её синонимы — плевропневмония, долевая пневмония), когда поражается целая доля легкого. Но бронхиальное дыхание может выслушиваться и при очаговой пневмонии (бронхопневмонии), если несколько воспалительных очагов инфильтрации располагаются близко или даже сливаются с образованием более крупного очага уплотнения («сливная пневмония»).
7. При компрессионном ателектазе легкого бронхиальное дыхание может быть очень тихим. Ослабленное бронхиальное дыхание выслушивается тогда, когда имеется плевральный выпот, а лежащее за ним легкое уплотнено, то есть поджато экссудатом (компрессионный ателектаз). Над абсцессом легкого или кавернами, которые свободны от содержимого (опорожнились) и сообщаются с бронхами, также можно выслушать бронхиальное дыхание. При этом сами полости могут играть роль резонаторов, усиливающих тембр бронхиального дыхания, так как в момент поступления воздуха через относительно узкий бронх в более широкую кавернозную полость также происходит завихрение воздуха.
8. Если в легком в результате абсцесса или кавернозного туберкулеза образуется гладкостенная полость размером не меньше грецкого ореха, сообщающаяся с крупным бронхом, то в результате бронхотрахеального дыхания и сильного резонанса возникают дополнительные очень высокие звучные обертоны, которые можно имитировать, если дуть над узким горлышком пустого стеклянного или глиняного сосуда (бутыль, кувшин). Такое дыхание называется амфорическим (греч.: amphora — глиняный сосуд с узким горлом). Оно соответствует металлическому тону при перкуссии.
9. Если сужена трахея или сужен крупный бронх (например, при опухоли), то ларинготрахеальное дыхание значительно усиливается и носит название ***стенотического.***

10. Выделяется также ***металлическое дыхание,*** отличающееся очень высоким тембром и громким звуком, напоминающим звук, возникающий при ударе по металлу. Металлическое дыхание можно выслушать при открытом пневмотораксе, когда воздух в плевральной полости сообщается отверстием с воздухом внешним.

Medicine Through Biographies

Read the text below, pausing after every paragraph to find the main fact (or idea) around which it is centered.

Then, read the text again and prepare a list of questions covering the main periods of Karl Andreyevich Rauchfuss's life. Using this list, discuss the facts with a partner/partners.

Write a summary of Rauchfuss's most significant contribution to the medical theory and practice.

Discuss interesting facts from Rauchfuss's life. What personal features these facts witness for?

Karl Andreyevich Rauchfuss (1835–1915): the first doctor who took on the white gown

Karl Andreyevich Rauchfuss, an outstanding Russian paediatrician and organizer of children health care was born in St. Petersburg and graduated from the famous Petrischule. In 1852 he was enrolled to the Medical Surgical Academy (now — the Military Medical Academy) and graduated from it in 1857. Later he specialised in Paediatrics and Otorhinolaryngology. Since 1858 till 1868 Rauchfuss was prosector and physician at the Findelhaus, an Imperial orphanage of St. Petersburg, which had a small pediatric hospital inside. That time paediatric infections were highly spread and represented very serious problem for children health care. Some of them (diphtheria, whooping cough, and influenza) involved respiratory system and produced dangerous complications, like croup (stenotic laryngitis). Of great help for Rauchfuss's patients were his surgical skills in laryngology. Once he had to perform an instant tracheotomy in a sick child, who suffered from croup and asphyxia. But the parents were categorically against this urgent surgical operation. The doctor decided not to persuade them, but gave a command to the hospital attendants: "Tie them up with a rope!" The shouting parents

were secured, and the doctor saved the child. Instead of gratitude, they took Rauchfuss to court and their lawyer accused him of double crime: deprivation of parental freedom and damage to the child. But the doctor was justified. In 1869 Rauchfuss became director and physician-in-chief at the newly establshed St. Petersburg Children's Hospital named for Prince Peter von Oldenburg. The hospital was the largest and best in Europe, equipped with all the latest achievements of that time. From the very beginning of its construction doctor Rauchfuss was one of the key figures in this innovative project. Collaborating with the architect Y. Kavos, Rauchfuss was in charge of the building and equipping of the Prince Peter von Oldenburg Children's Hospital (1867 to 1869). Later he reproduced a similar project in Moscow (St. Vladimir Children's Hospital, constructed from 1875 to 1876). At both hospitals he maintained the principle of strict isolation of contagious cases, both at the hospital wards and at the outpatient department. Even the first examination of incoming patients proceeded in complete isolation, at special rooms, communicated with admissions department and with outside. For that epoch it was a great advance. From 1875 Rauchfuss headed the paediatric clinic at the Higher Medical Courses for Women (nowadays — I. P. Pavlov First Medical University), and from 1876 till his last day he was imperial paediatrician in ordinary to the Royal Family. Rauchfuss was the founder of the Society of Paediatricians in St. Petersburg and the all-Russian welfare committee for maternity and childhood. Dr. Rauchfuss is world known among medical professionals not only because of the percussion phenomenon, which he described in 1904 (so called Rauchfuss-Korány-Grocco triangle.

He was the first physician in the history of medicine, who introduced white dressing gowns for doctors and nurses — a tradition now accepted all over the world. In 1905 Russian paediatrician was elected as honorary member of the German Paediatric Society. His wife, Polina Karlovna, was the founder of Froebel Society for early and pre-school children education, now acting worldwide. The hospital, constructed under Dr. Rauchfuss's supervision, still functions at St. Petersburg and is now named after him.

After half a century of successful work, in 1907 K.A. Rauchfuss wrote:

«I am happy that my first meetings in a field of medical career were a Mother and a Child. In the orphanage where I have directed my first steps, there was a cradle of a child. And at the same time it was a cradle of my happiness. I became a pediatrician by a lucky occasion, but this accident was in full accordance with all construction of my soul. Even in the first days and nights when I stood at the bed of a sick child, divided sufferings and a grief of its mother for her beloved one, the soul of the baby welcomed me and a wonderful look of the grateful mother, giving to me the completeness of happiness and opened in me desire to continue this work and to achieve perfection.

And since then I have learned to love and respect in the woman — Mother and in the child — its Future».

VOCABULARY

a

ad oculus	визуально
AB	1) [apex beat] верхушечный толчок; 2) [abdominal bloating] метеоризм, вздутие живота; 3) [aspiration biopsy] аспирационная биопсия
abdominal	абдоминальный, относящийся к животу
abdominal cyst	абдоминальная киста
abscess	абсцесс, гнойник
Addison-Biermer anemia = Addison-Biermer disease	пернициозная анемия, мегалобластическая анемия с первичным аутоиммунным атрофическим гастритом, болезнь Аддисона–Бирмера
adenoid(s)	аденоид; аденоидный; аденоиды, аденоидные разращения, аденоидные вегетации
adhesion	1) адгезия, слипание; склеивание; соединение; сцепление; 2) сращение; спайка; спайкообразование; 3) заживление (раны); 4) молекулярное притяжение
adhesive pericarditis	спаечный перикардит, адгезивный перикардит, слипчивый перикардит
afferent	приводящий
aggravator	фактор, вызывающий обострение
AIDS (acquired immune deficiency syndrome)	СПИД (синдром приобретенного иммунного дефицита)
airways	дыхательные пути
alcohol abuse = alcohol addiction	алкоголизм
alcoholic intoxication	алкогольная интоксикация
allergic rhinitis	аллергический ринит
alopecia	алопеция, облысение
alveolar	альвеолярный
alveolar respiration	альвеолярное дыхание
alveole	альвеола
alveolitis	альвеолит (1. альвеолярная пневмония; 2. воспаление стенок альвеолы зуба)
amphoric respiration	амфорическое дыхание
analgesic	аналгезирующее средство, болеутоляющее средство, аналгетик
anamnesis morbi	анамнез morbi (совокупность сведений о болезни, относящихся к периоду до обращения к врачу)
anasarca	анасарка (распространенный отек подкожной клетчатки)
anatomical axis	анатомическая ось
anemia	анемия, малокровие
aneurysm	аневризма (расширение просвета кровеносного сосуда или полости сердца вследствие патологических изменений их стенок)
angina	1) ангина; 2) стенокардия, грудная жаба

ankylosis	анкилоз, неподвижность суставов
anticoagulant	антикоагулянт, противосвертывающее средство
antipyretic	жаропонижающее средство, антипиретик; жаропонижающий, противолихорадочный
aortic	аортальный
aortic aneurysm	аневризма аорты
aortic insufficiency	недостаточность клапана аорты, аортальная недостаточность
aortic stenosis	стеноз устья аорты, аортальный стеноз
aperture	1) апертура, отверстие, щель; 2) диаметр объектива микроскопа
apex	1) верхушка; 2) верхушка легкого; 3) верхушка корня зуба
aphonia	афония (отсутствие звучности голоса)
apnea	апноэ (временная остановка дыхания)
apneustic breath	1) апнейстическое дыхание (разновидность патологической одышки)
aponeurosis	апоневроз
appendicitis	аппендицит (воспаление червеобразного отростка слепой кишки)
arrhythmia	аритмия (сердца)
arterialization	артериализация (превращение венозной крови в артериальную)
arteritis	артериит (воспаление стенки артерии)
ascending	восходящий, афферентный
ascites	асцит, брюшная водянка
asphyxia	асфиксия (гипоксемия, сопровождаемая гиперкапнией), удушье
asthenic	астенический
asthma	астма; удушье
atelectasis	ателектаз, спадение легкого при нарушенной его вентиляции и сохранной перфузии
atherogenic	атерогенный, способствующий развитию атеросклероза
atherosclerosis	атеросклероз
atherosclerotic cardiosclerosis	атеросклеротический кардиосклероз
atrial	предсердный
atrioventricular	атриовентрикулярный, предсердно-желудочковый
atrium	1) полость; пазуха; синус; 2) предсердие, преддверие; 3) отверстие бронхиолы (открывающееся в альвеолы); 4) преддверие; 5) входные ворота инфекции
atrophy	1) атрофия (органа, ткани); 2) истощение
atropine	атропин
auscultation	аускультация, выслушивание
autoimmune	аутоиммунный
autopsy	аутопсия, вскрытие
axillary	подмышечный, подкрыльцовый

axillary region = axillary space	подмышечная область, подкрыльцовая область
Ayerza's disease	первичный атеросклероз легочной артерии, синдром Айерсы

b

backbone	позвоночник, позвоночный столб
backflow	1) обратный ток (жидкости), противоток; 2) рефлюкс; обратное забрасывание; ретроградный кровоток
bacterial endocarditis	инфекционный, септический эндокардит, бактериальный эндокардит
bandbox sound = bandbox resonance	коробочный перкуторный звук
barrel chest	эмфизематозная грудная клетка, бочкообразная грудная клетка
basal	базальный, расположенный у основания
beat	1) систола (сердца); 2) систолический шум; 3) пульс; 4) толчок (верхушечный, сердечный)
bed-patient	лежачий больной
belching = eructation	отрыжка
belladonna	белладонна, красавка обыкновенная
bending	1) сгибание; изгибание; 2) изгиб; кривизна; изогнутость
bifurcation	бифуркация (разделение трубчатого органа на две ветви)
bigeminy	бигеминия, (разновидность экстрасистолии, форма аллоритмии)
binaural	бинауральный (относящийся к обоим ушам)
blisters	буллы, буллезное поражение
Botallo's duct patency	зияние Боталлова протока (артериальный проток)
Botallo's foramen	Боталлово отверстие, овальное отверстие межпредсердной перегородки
brachial	плечевой, брахиальный
bradycardia	брадикардия (пониженная частота сердечных сокращений)
breathlessness (dyspnea)	одышка (диспноэ)
bronchial asthma	бронхиальная астма
bronchial (laryngotracheitic) respiration	бронхиальное (ларинготрахеальное) дыхание
bronchiectasis = bronchiectasia	бронхоэктаз (расширение ограниченных участков бронха)
bronchiole	бронхиола
bronchitis	бронхит
bronchopneumonia = bronchopneumonitis	очаговая пневмония, бронхопневмония
bronchopulmonary	бронхолегочный
bronchospasm	бронхоспазм

bronchotracheal respiration	бронхотрахеальное дыхание
bronchus (*pl.*: bronchi)	бронх (*мн.*: бронхи)
brucellosis	бруцеллез, мальтийская лихорадка
bubbling râles	влажные хрипы
buccal	1) буккальный, относящийся к щеке; щечный; трансбуккальный (о методе введения лекарственного средства); 2) внутриротовой

C

cachexia	кахексия, общая атрофия
calcinosis	кальциноз, обызвествление, кальцификация
cancer	рак, карцинома, злокачественная эпителиома
capillary pulse	капиллярный пульс, прекапиллярный пульс Квинке, симптом Квинке
carbon dioxide acidosis	газовый ацидоз, дыхательный ацидоз, респираторный ацидоз
carcinogen	канцероген, канцерогенное вещество, онкогенное вещество
cardiac	1) сердечный; 2) кардиальный
cardiac apex	верхушка сердца
cardiac conduction system	проводящая система сердца
cardiac dropsy	сердечный отек, также: гидроперикард, водянка перикарда
cardiac humpback	сердечный горб
cardiac muscle	миокард, сердечная мышца
cardialgia	кардиалгия (боль в сердце)
cardiomyopathy	кардиомиопатия
cardioneurosis	кардионевроз, невроз сердца (*устар.*)
cardiorespiratory	кардиопульмональный, сердечно-легочный
carditis	кардит (воспаление каких-либо структур сердца)
carnification = carneous degeneration	карнификация (патологическое изменение легочной ткани, при которой она приобретает консистенцию и вид сырого мяса)
carotid (artery)	сонная артерия
cartilage	хрящ
case history, case file	история болезни
cavern	1) полость, впадина; 2) каверна
cavernous tuberculosis	кавернозный туберкулез
cerebral	церебральный, мозговой
cerebral (craniocerebral) injury	черепно-мозговая травма
cervical	цервикальный (1. относящийся к области шеи; 2. относящийся к шейке какого-либо органа)
cervical vertebra	шейный позвонок

Cheyne-Stokes respiration	дыхание Чейна-Стокса
cholecystitis	холецистит (воспаление жёлчного пузыря)
cholelithiasis	жёлчнокаменная болезнь, холелитиаз
cholinolytics	холинолитики (противодействующие эффекту ацетилхолина препараты)
ciliary arrhythmia	мерцательная аритмия
claudication	хромота
clavicle	ключица
clonic convulsion	клоническая судорога
coarctation	коарктация (аорты), сужение, стеноз; стриктура (сосуда, канала, отверстия)
cognizant = perceptable	ощутимый, чувствительный
collapse	коллапс (острая сосудистая недостаточность)
coma	кома, коматозное состояние; глубокое бессознательное состояние (с расстройством жизненно важных функций)
comatose	коматозный, находящийся в состоянии комы
combined dyspnea	комбинированная (смешанная) одышка
comparative percussion	сравнительная перкуссия
compliance	изменение объема легких при колебаниях давления
complication	осложнение
compression	компрессия, сдавление; сжатие
condensation	уплотнение (легочной ткани)
confluent pneumonia	сливная пневмония
congenital	врожденный
congestion	застой (напр. крови, желчи)
congestive heart failure	застойная сердечная недостаточность
conjunctive tissue, connective tissue	соединительная ткань
conscious	находящийся в сознании
consciousness	сознание
constipation	констипация, запор, обстипация
contagious	контагиозный, заразный
contusion	1) ушиб; контузия; 2) закрытая травма
cor pulmonale	легочное сердце, гипертрофия и недостаточность правого сердца при заболеваниях легких и гипертензии малого круга кровообращения
cornea	роговица
corneal	роговичный, корнеальный
costovertebral	реберно-позвоночный
cracked-pot sound	симптом «треснувшего горшка»
crepitation	крепитация (ощущение похрустывания или потрескивания, возникающее при пальпации или аускультации)
croup	круп (острый ларингит или ларинготрахеит, сопровождающийся явлениями спазматического стеноза гортани)
croupous pneumonia	крупозная пневмония

crural	голенный, находящийся на голени
cutaneous	кожный
cyanosis	цианоз, синюха (*устар.*) (синюшний оттенок кожи и слизистых оболочек, обусловленный повышенным содержанием восстановленного гемоглобина)

d

dehydration	обезвоживание, дегидратация
delimitation	отграничение, предотвращение распространения (патологического процесса), купирование распространения (патологического процесса)
diabetes mellitus = sugar diabetes	сахарный диабет
diagnosis	1) диагноз; 2) диагностика
diagnostics	учение о диагностике заболеваний
diapedesis	диапедез
diaphragmatic = phrenic	диафрагмальный
diastolic	диастолический
dicumarol	дикумарол
dilatation = dilation	дилатация, расширение
dilate	растягивать
diphtheria	дифтерия
distension	растяжение, вздутие (живота)
diuretic	мочегонное средство, диуретическое средство, диуретик; мочегонный, диуретический
dizziness = vertigo	головокружение
doppler ultrasonography	допплеровская ультрасонография, эхография, допплеровское УЗИ
Dressler's syndrome	постинфарктный синдром, синдром Дресслера
drive	импульс
dropsy	водянка (скопление жидкости в какой-либо полости тела)
dry rales	сухие хрипы
duct	проток; канал; ход; проход
dullness = dulness	тупость (перкуторного звука); приглушенность, притупленность
Dupuytren's contracture (palmar sclerosis)	контрактура ладонного апоневроза, контрактура Дюпюитрена, синдром Дюпюитрена
Duroziez double murmur	двойной шум Дюрозье
dyspepsia	диспепсия (расстройство пищеварения)
dysplasia	дисплазия (нарушение формирования ткани или органа)
dystonia	дистония, нарушение тонуса
dystrophic	страдающий дистрофией
dystrophy	дистрофия, гипобиоз, дегенерация, перерождение, истощение (алиментарное)

e

ECG [electrocardiogram]	электрокардиограмма, ЭКГ
ectomorphic	астенического телосложения
edema (*pl.*: edemata, edemas)	отек
Ellis-d'Amoiseau line	линия Эллиса–д'Амуазо
embolism	эмболия (закупорка кровеносного сосуда эмболом)
emphysema	эмфизема (понижение эластических свойств легкого с увеличением его воздушности за счет остаточного объема), *тж.*: вздутие органа или ткани за счет воздуха
emphysematous	эмфизематозный; содержащий воздух или газ, газовый (о гангрене)
empyema	эмпиема (скопление гноя в полости, чаще всего — плевральной)
encopresis	энкопрез, недержание кала
endocarditis	эндокардит (воспаление эндокарда)
endomorphic	пикнического типа телосложения
ENT [ear, nose, throat]	ЛОР (*букв.*: ухо, нос, горло) сокращение обозначает оториноларингологию
ENT doctor, ENT specialist	ЛОР-врач, врач-оториноларинголог, ухогорлонос
enterovirus	энтеровирус
enuresis	недержание мочи, энурез; ночное недержание мочи
ephedrine	эфедрин (гидрохлорид эфедрина — сосудосуживающее и бронхорасширяющее средство)
epigastric pulse	надчревная пульсация
epigastrium	надчревье, надчревная область, эпигастрий
epinephrine	адреналин, эпинефрин
erethism	эретизм (болезненное состояние возбуждения или раздражения)
ergometry	эргометрия; динамометрия
erysipelas	рожа; рожистое воспаление
erythema	эритема (ограниченная гиперемия кожи)
erythrocytosis	эритроцитоз, полицитемия
esophagus	пищевод
etiology	этиология
exacerbation	обострение болезни
examination	обследование
excursion	движение, подвижность, экскурсия
exhalation	выдох
exhaustion	истощение; утомление, крайняя усталость
expectoration	мокрота, отхаркивание
expiratory dyspnea	экспираторная одышка
extracardiac	некардиальный

extraneous	инородный, посторонний
extrasystole	экстрасистола
extreme fatigue	состояние крайнего утомления
extremities	конечности
exudative	экссудативный, выпотной
exudative pericarditis	экссудативный перикардит, выпотной перикардит
exudative pleuritis	экссудативный плеврит, выпотной плеврит

f

facet	небольшая суставная поверхность, грань
failure	недостаточность, декомпенсация; нарушение; расстройство
faint	обморок, синкопе
false croup	ложный круп, псевдокруп
farmer's lungs	«легкие фермера», экзогенный плесневый аллергический альвеолит
fatigability = fatiguability	утомляемость
femoral	бедренный
fenilin	фенилин
fibrinous pericarditis	фибринозный перикардит
findings	данные
flatulence	метеоризм (вздутие живота вследствие скопления газов в кишечнике)
flush	покраснение
foramen (*pl.*: foramina)	отверстие
fracture	1) перелом; ломать, сломать; 2) разрыв; трещина; излом
functional	функциональный

g

gangrene	гангрена; вызывать гангрену
Garland's triangle	треугольник Гарланда
gastric	гастральный, желудочный
general sickness	общее недомогание
general state	общее самочувствие
gingiva (*pl.*: gingivae)	десна
gland	железа
Glisson's capsule	фиброзная капсула печени, глиссонова капсула
glottis	голосовая щель
goiter	зоб, струма
gout	подагра
Graves' disease	диффузный тиротоксический зоб, фон Базедова болезнь, болезнь Грейвса, болезнь Парри, болезнь Флаяни

grippe	грипп
Grocco-Rauchfus-Korányi triangle	треугольник Грокко–Раухфуса–Кораньи
gross	макроскопический (в морфологии)
gut	кишка, желудочно-кишечный тракт

h

hay fever = hay catarrh	поллиноз, сенная лихорадка
health care	здравоохранение
heart disease	(клапанное) сердечное заболевание; порок сердца
heart failure	сердечная недостаточность
helminthiasis	гельминтоз, глистная инвазия
hematologic	гематологический
hemoglobin	гемоглобин
hemoptysis	кровохарканье
hemorrhage	1) кровотечение; 2) кровоизлияние
hemorrhagic	геморрагический, относящийся к кровотечению
hepatic	печеночный
hepatic cirrhosis	цирроз печени
hepatization	опеченение, гепатизация
hernia	грыжа
herpes	герпес
herpes zoster	опоясывающий герпес, опоясывающий лишай
heterotopic	отраженный
high phrenic position	высокое положение диафрагмы
His' bundle	пучок Гиса
Hodgkin's disease	лимфогранулематоз, хронический злокачественный лимфоматоз, фибромиелоидный ретикулез (*устар.*), болезнь Ходжкина, лимфома Ходжкина
Horton's disease	гистаминовая цефалгия, синдром Хортона, мигрень Хортона
humming râles	жужжащие хрипы
humpback	патологический кифоз, кифотическое искривление позвоночника, горб
hydropericardium = hydropericarditis	гидроперикард, водянка перикарда
hydropneumothorax	гидропневмоторакс
hydrothorax	гидроторакс
hyperalgesia = hyperalgia	гипералгезия (повышенная болевая чувствительность)
hypercapnia	гиперкапния (повышенное парциальное напряжение двуокиси углерода в крови)
hypercapnic	гиперкапнический

hypercholesterolemia = hypercholesteremia	гиперхолестеринемия
hyperemia	гиперемия, полнокровие
hyperlipoproteinemia = hyperlipoproteidemia	гиперлипопротеинемия=гиперлипопротеидемия
hypersthenic	гиперстенический (характеризующийся гиперстенией)
hypertension	гипертензия, гипертония
hyperthyroidism	гипертиреоз, гипертиреоидизм (синдром, обусловленный избыточным действием гормонов щитовидной железы)
hypertonicity	1) гипертоничность; 2) повышение осмотического давления
hypertrophic	гипертрофический
hypertrophy	гипертрофия (увеличение органа или его части без увеличения числа клеток в них)
hyperventilation	гипервентиляция легких, гиперпноэ
hypocapnia	гипокапния (пониженное парциальное напряжение двуокиси углерода в крови)
hypopituitarism	гипопитуитаризм (недостаточность функции гипофиза)
hypotension	гипотензия, гипотония
hypotensive	больной с постоянно сниженным артериальным давлением, гипотоник
hypothyroidism	гипотиреоз
hypoxemia	гипоксемия, аноксемия (пониженное парциальное напряжение кислорода в крови)
hypoxemic	гипоксемический

i

iatrogenic = jatrogenic	ятрогенный (о заболевании)
idiosyncrasy	идиосинкразия, повышенная чувствительность (неаллергической природы), индивидуальная непереносимость
immunization	1) иммунизация, предохранительные прививки, профилактические прививки; 2) иммунизация, вакцинация
impairment	ухудшение (в результате травмы или болезни)
impedance pletismography	импедансная плетизмография, реоплетизмография, электроплетизмография, реография
induration	индурация, уплотнение (органа или ткани); затвердение, отвердение
infiltration	инфильтрация
infraclavicular	подключичный
inspiration	вдох
inspiratory dyspnea	инспираторная одышка

insufficiency	недостаточность
interalveolar septa	межальвеолярные перегородки
intercostal	межреберный
interlobar	интерлобарный, междолевой, находящийся между долями
internals	внутренние органы
interrupted (saccadic) respiration	прерывистое (саккадированное) дыхание
interscapular	межлопаточный
interventricular	межжелудочковый
intracranial	интракраниальный, внутричерепной
intrathoracic	внутригрудной, интраторакальный
ischemic heart disease	ишемическая болезнь сердца

j

jaundice	желтуха
joint	сустав, синартроз, синовиальное соединение, объединенный, совместный
jugular	1) яремный; 2) яремная вена
jugular fossa	яремная ямка, надгрудинная ямка

k

keeled breast = keeled chest	килевидная грудная клетка, куриная грудь
kyphoscoliosis	кифосколиоз
kyphotic	1) кифотический; 2) страдающий кифозом

l

labial	губной
labor	роды, родовой акт
laryngeal edema	отек гортани; отек подсвязочного пространства
laryngeal	ларингеальный, гортанный, относящийся к гортани
laryngitis	ларингит (воспаление слизистой оболочки гортани)
larynx	гортань
left ventricular failure	левожелудочковая недостаточность
Leonardo da Vinci phenomenon, Da Costa syndrome	феномен Леонардо да Винчи, синдром Да Коста (потеря сознания при гипервентиляции и гипокапнии)
lesion	повреждение, поражение, патологическое изменение
leukemia	лейкоз, лейкемия, белокровие
lid	веко, крышка

lien = spleen	селезенка
liver	печень
lobar pneumonia	долевая пневмония, лобарная пневмония, лобит (*устар.*)
lobe	доля (легкого)
lumen	просвет; полость трубчатого органа
lung abscess	абсцесс легкого
lupus erythematosus, LE	красная волчанка, СКВ
lymph node	лимфатический узел, лимфоузел

m

malady	болезнь, заболевание
malaria, jungle fever	малярия
mammary gland	молочная железа
marginal	предельный, критический
Marie–Strümpell–Bekhterev disease	анкилозирующий спондилоартрит, болезнь Бехтерева–Штрюмпеля–Мари
measles	корь
mediastinal	медиастинальный, средостенный
mediastinum	средостение
medioclavicular	средне-ключичный, относящийся к срединной части ключицы
meningitis	1) менингит; 2) лептоменингит
meninx (meninges)	мозговая оболочка головного и спинного мозга
mesaortitis	мезаортит (воспаление мышечной оболочки аорты)
mesenteric	мезентериальный, брыжеечный
mesomorphic	мезоморфного типа телосложения
mesothelioma	мезотелиома, целотелиома
metabolite	метаболит (продукт метаболизма)
metal respiration	металлическое дыхание
metastasis	1) метастаз; 2) метастазирование
methemoglobin	метгемоглобин, ферригемоглобин
microangiopathy	микроангиопатия
misdiagnosis	ошибочный диагноз
mitral insufficiency	митральная недостаточность, недостаточность левого предсердно-желудочкового клапана
mitral stenosis	стеноз левого атриовентрикулярного отверстия, митральный стеноз
mitral valve	левый предсердно-желудочковый клапан, левый двустворчатый клапан, левый митральный клапан
moniliform	монилиформный, бусообразный, бусовидный
morbidity rate	коэффициент заболеваемости

MSH (melanocyte-stimulating hormone)	МСГ (меланоцитстимулирующий гормон)
mucous	слизистый
mucous membrane	слизистая оболочка
murmur	шум, аускультативный феномен
musculus sternocleidomastoideus	грудино-ключично-сосцевидная мышца
Musset's sign	симптом Мюссе (синхронное с ритмом сердца покачивание головы вперед-назад; признак недостаточности клапана сердца)
myasthenia	миастения, болезнь Эрба–Гольдфлама
(multiple) myeloma, myelomatosis	миеломная болезнь, множественная миелома, болезнь Рустицкого — Калера (лимфома из плазматических клеток)
myocardial infarction	инфаркт миокарда
myocarditis	миокардит (воспаление сердечной мышцы)
myositis	миозит (воспаление мышцы)
myxedema	микседема; гипотироидный отек, синдром Галла

n

nailbed	ногтевое ложе
narcotic	наркотизирующий, вызывающий наркоз
nasal	носовой, назальный
nasolabial	носогубной
nasopharynx	носовая часть глотки, носоглотка, эпифаринкс
navel	пупок
nephritis	нефрит (воспаление почек)
nervus	нерв
neuralgia	невралгия
neurocirculatory	нейроцикуляторный
neurocirculatory dystonia = somatoform autonomic dysfunction (see footnote 1, p. 170)	нейроцикуляторная дистония, вегетососудистая дистония, вегетоневроз (*Термин употребляется только в СНГ. Английские эквиваленты — см. сноску 1, стр. 170*)
neurons	нейрон, нервная клетка, нейроцит
neurosis	невроз (*в настоящее время термин исключен из МКБ-10 и трактуется как типовые изменения поведения, вызванные дистрессом*)
neurotic	невротик, человек, страдающий неврозом, имеющий вызванные дистрессом изменения поведенческих реакций
nipple	1) сосок (молочной железы); 2) выпячивание, выбухание (напр. дуги аорты); выступ

nodose = nodous = nodular	нодозный, узелковый; узловатый
noncompensated	некомпенсированный
normosthenic	нормостенический

o

obesity	ожирение
occlusion	обтурация; окклюзия; закупорка
occupational bronchitis	профессиональный бронхит
occupational hazards	профессиональные вредности
opening snap	тон открытия (клапана сердца), щелчок открытия (клапана сердца)
organic	1) относящийся к органу или органам; 2) связанный с жизнью организма; органический
orifice	отверстие; устье; вход; проход
orthostatis	ортостаз
oscillation	осцилляция, колебание
Osler's disease	истинная полицитемия, эритремия, болезнь Вакеза–Ослера
ossification	1) окостенение, оссификация; 2) образование костного вещества
osteochondrosis	остеохондроз
ostium	отверстие (вход в полый орган или канал)
ovary	яичник
oxyhemoglobin	оксигемоглобин, оксигенированный гемоглобин

p

pallor	бледность
palmar	ладонный
palpation	пальпация, ощупывание, прощупывание
palpitation	учащенное сердцебиение
palsied	1) парализованный; 2) дрожащий, трясущийся
pancarditis	панкардит (воспаление всех слоев стенки сердца)
pancreatitis	панкреатит (воспаление поджелудочной железы)
panhypopituitarism	пангипопитуитаризм, в частности — болезнь Симмондса)
pansystolic	пансистолический (о шуме сердца)
papillary	сосочковый, папиллярный
paralysis, palsy	паралич
paroxysmal	пароксизмальный
patency	зияние, раскрытое состояние сосуда, канюли
paediatric = pediatric	педиатрический

pediatrician = paediatrician	педиатр, детский врач
pelvic	тазовый
percussion	перкуссия, выстукивание
periarteritis	периартериит (воспаление адвентиции артерии)
pericardial = pericardiac	перикардиальный, относящийся к перикарду
pericarditis	перикардит (воспаление перикарда)
periostitis	периостит (воспаление надкостницы)
peritoneal	перитонеальный, брюшинный
phalanx (*pl.*: phalanges)	фаланга пальца
phrenic	диафрагмальный, грудобрюшинный
phthisiologic	фтизиатрический
Pickwickian syndrome	пиквикский синдром, синдром Пиквикского клуба, (сочетание выраженного ожирения с легочной гиповентиляцией, малоподвижностью, сонливостью)
pigeon breeder's lung	болезнь (легкое) голубеводов, птичниц (аллергический плесневый альвеолит)
pilosis (in females — hirsutism)	избыточное оволосение (у женщин — гирсутизм)
pitting edema	образование углублений при надавливании на область отека
pituitary necrosis	гипофизарный некроз
plantar	подошвенный, плантарный
plaque	1) бляшка; 2) тромбоцит, кровяная пластинка, бляшка Бицоцеро
plethora	плетора, гиперволемия
pleural effusion	плевральный выпот
pleural exudate	плевральный экссудат
pleural friction rub	шум трения плевры
pleurisy = pleuritis	плеврит (воспаление плевры)
pleuropericardial	плевроперикардиальный
pleuropneumonia	плевропневмония
pleuropulmonary	плевролегочный
plessimeter finger = plexor	палец-плессиметр
pneumoconiosis	пневмокониоз (название профессиональных болезней органов дыхания, обусловленных хроническим воздействием производственной пыли)
pneumonia	пневмония, воспаление легких
pneumopericardium	пневмоперикард (наличие газа в перикардиальной полости)
pneumosclerosis	пневмосклероз, пневмофиброз, склероз легких
pneumotaxis	регуляция внешнего дыхания
pneumothorax	пневмоторакс (наличие воздуха или газа в плевральной полости)

pollutant	загрязняющее вещество, загрязнитель; примеси (в воздухе)
polyp	полип
pontine = pontile	относящийся к варолиеву мосту
popliteal	подколенный
portocaval shunt	портокавальный анастомоз
posterior	задний
post-mortem	посмертное (иследование), вскрытие трупа, аутопсия
pregnancy	беременность
presternum	рукоятка грудины
presystolic	пресистолический
prodromal stage	продромальный период
protrusion	выступание вперед, выпячивание; протрузия, выбухание
pseudocirrhosis	1) сердечный фиброз печени; 2) псевдоцирроз, перикардитический цирроз печени
pseudotuberculosis	псевдотуберкулез
pterygoid	крыловидный, птеригиумоподобный
puerile (children's) breath	пуэрильное (детское) дыхание
pulmonary	легочный, пульмональный
pulmonary edema	отек легких
pulmonary embolism	эмболия сосудов легких; эмболия легочной артерии
pulmonary heart	правые отделы сердца
pulmonary hemorrhage	легочное кровотечение
pulmonary infarction	инфаркт легкого
pulmonary tissue	легочная ткань
pulmonary tuberculosis	туберкулез легких
pulmonary tumor	опухоль легкого
pulsation	пульсация, биение
pulse intermission	перебои пульса
purulent	гнойный; гноящийся
purulent pleuritis	гнойный плеврит

r

radial	1) лучевой (напр. о кости); 2) радиальный (напр. о расположении волокон)
rash	сыпь (быстропроходящая, мимолетная)
RBC 1) [red blood cell]; 2) [red blood count]	1) эритроцит; 2) количество эритроцитов
recurrence	рецидив
recurrent	рецидивирующий, повторяющийся, возвратный
regurgitate	1) течь в обратном направлении; 2) срыгивать

regurgitation	1) регургитация (перемещение содержимого полого органа в направлении, противоположном физиологическому, в результате сокращения его мышц); 2) отрыжка; срыгивание
relative cardiac dullness border	относительная граница сердечной тупости
remittent	перемежающийся (о лихорадке); ремиттирующий (о течении болезни)
renal dropsy	почечный отек
renal failure	почечная недостаточность
resolution	1) растворение; 2) разложение; расщепление; 3) рассасывание (напр. опухоли); разрешение (напр. воспалительного процесса)
resonance	перкуторный звук
respiration rate	частота дыхания
respiratory	дыхательный
respiratory sound	дыхательный шум
reticulum (*pl.*: reticula)	1) ретикулум, (тонкая) сеть, сеточка, сетчатое строение; 2) нейроглия; 3) ретикулярная ткань; 4) остатки РНК в ретикулоцитах при окрашивании бриллиантовым крезиловым голубым
retraction	1) втяжение; западение; 2) ретракция; стягивание, сокращение
retrosternal	загрудинный
rheumatic	ревматический
rheumatic fever	ревматическая атака
rib fracture	перелом ребра
rigidity	ригидность, оцепенелость (обусловленная напряжением мышц); тугоподвижность; окоченение
rough respiration	жесткое дыхание
rupture	разрыв; прободение; перфорация

S

sacral	крестцовый, сакральный
sagittal	сагиттальный, стреловидный
sc [subcutaneous]	п/к [подкожный] *(в частности, путь введения лекарства)*
scapula (*pl.*: scapulae)	лопатка
sclerosis	1) фиброзное уплотнение; 2) склероз
sclerotic	склеротический
sclerotic coat	склера
scoliosis	сколиоз (боковое искривление позвоночника)
scrotal	мошоночный
scrotum	мошонка

secretion	секреция, секрет (желез)
septal	септальный, перегородочный
septic	септический, относящийся к сепсису
septum (*pl.*: septa)	перегородка
shock	шок
shoulder-blade = scapula	лопатка
silicosis	силикоз, силикатоз (форма пневмокониоза, возникающая вследствие вдыхания кремний-содеращей пыли)
sinoatrial node	синусно-предсердный узел, синусный узел, узел Киса–Флака (*прав.*: Флэка)
sinoauricular block	синоаурикулярная блокада
site	участок
Škoda's tone	тон Шкоды
skull	череп
somatotype	тип конституции, тип телосложения
somnolence = somnolency	1) сонливость; 2) полубессознательное состояние
souffle	нежный дующий шум (при аускультации)
spondylarthritis	спондилоартрит (воспаление межпозвоночных суставов)
spondylitis	спондилит (воспаление всех или некоторых структурных элементов позвоночника)
sputum	мокрота
staphylococcus (*pl.*: staphylococci)	стафилококк
state of nourishment	степень упитанности
stenocardia, angina pectoris	стенокардия, грудная жаба
stenotic	стенозированный, вызванный стенозом, характеризующийся сужением
stenotic respiration	стенотическое дыхание
sternal	стернальный, грудинный
sternum	грудина
stethophonendoscope	стетофонендоскоп
stethoscope	стетоскоп
stomatologic	стоматологический
stool	стул
strain	напряжение, нагрузка
stratification	расслоение
streptococcal	стрептококковый
Streptococcus (*pl.* streptococci)	стрептококк
stridor	стридор (свистящее дыхание)
stroke	внезапный приступ; припадок
stupor	1) помрачение сознания; оглушение; 2) ступор
subclavicular	подключичный
subcostal	подреберный
subcutaneous	подкожный
subfebrile	субфебрильный

subicteric	субиктеричность (легкая желтушность)
supraclavicular	надключичный
sympathetic	симпатический (относящийся к симпатической нервной системе)
sympathicotonia	симпатикотония
sympathomimetic(s)	симпатомиметическое средство; симпатомиметический(е)
syncope	синкопе, обморок
syndrome	синдром, симптомокомплекс
syphilis	сифилис, люэс (*устар.*)
syphilitic	сифилитический
systole	систола сердца
systolic	систолический

t

tachycardia	тахикардия
tachycardia attack	приступ тахикардии
Takayasu's disease	болезнь отсутствия пульса, синдром (болезнь) Такаясу
telangiectasia	телеангиэктазия (локальное чрезмерное расширение мелких сосудов)
temporal	1) височный; 2) временный, преходящий
tetralogy of Fallot	тетрада Фалло, тетралогия Фалло
the angle of Louis	угол Людовика
thoracic	торакальный, грудной
thoracic vertebrae	грудной позвонок
thoracic wall	торакальная стенка, стенка грудной клетки
thoracophrenic	торакодиафрагмальный
thorax	грудная клетка
thread pulse	нитевидный пульс
throat	гортань
thromboembolism	тромбоэмболия (эмболия вследствие тромбоза)
thrombophlebitis	тромбофлебит (воспаление вены с ее тромбозом)
thrombotic	тромботический
thyroid	щитовидная железа; щитовидный
thyrotoxic	тиротоксический, тиреотоксический (обусловленный крайне повышенной функцией щитовидной железы)
thyrotoxicosis	тиротоксикоз, тиреотоксикоз
thyrotoxic tremor	тиротоксический тремор
timbre	тембр
tonic convulsion	тоническая судорога
tonsillitis	1) ангина; 2) тонзиллит, амигдалит
topographic percussion	топографическая перкуссия
trachea	трахея

tracheitis	трахеит (воспаление слизистой оболочки трахеи)
tracheobronchial	трахеобронхиальный
transverse	поперечный
trapezium	кость-трапеция (запястья)
Traube's double tone	двойной тон Траубе, двойной симптом Траубе
Traube's space	пространство Траубе
trauma	травма, повреждение
tricuspid	трехстворчатый (о клапане)
tricuspid disease	порок трехстворчатого клапана сердца
tricuspid valve	правый предсердно-желудочковый клапан, правый трехстворчатый клапан
trophic	трофический
Trypanosoma	трипаносома
tuberculosis	туберкулез, чахотка (*устар.*)
tumor	опухоль
tympanic	тимпанический (о перкуторном звуке), барабанный

u

ulceration	1) образование язвы; 2) язва, язвы
ulorrhagia	десневое кровотечение
umbilical	пупочный, умбиликальный
uncertain respiration	неопределенное дыхание
unconscious state patient	больной, находящийся в бессознательном состоянии
uneven respiration	неравномерное дыхание
unilateral	односторонний (о локализации патологического процесса)
upstream	вверх по течению (для кровеносных сосудов)
urate	урат, соль мочевой кислоты
uremia	уремия, мочекровие
uremic	уремический (относящийся к уремии, обусловленный уремией)

v

vagus = vagus nerve	блуждающий нерв
valvular	вальвулярный, клапанный
valvular heart disease	порок клапана сердца
varicosity	1) варикозно расширенная вена; 2) варикозное расширение вен, варикоз
vascular	васкулярный, сосудистый
vasculitis	васкулит, ангиит (воспаление стенок кровеносных сосудов)
vasopathy	вазопатия
vena cava superior	верхняя полая вена

venepuncture	венопункция, венепункция
ventricle	желудочек (сердца)
ventricular hypertrophy	гипертрофия желудочка; гипертрофия желудочков
vermiform	червеобразный
vertebrosternal	позвоночно-грудинный
vertigo	головокружение
vesicular respiration	везикулярное дыхание
visceral	висцеральный (относящийся к внутренним органам)
visceroptosis = visceroptosia	спланхноптоз, висцероптоз, симптомокомплекс Гленара
viscous	вязкий; липкий, клейкий; тягучий
visual examination	визуальный осмотр
vocal cords	голосовые связки
vocal fremitus	голосовое дрожание
vocal ligaments palsy	паралич голосовых связок

W

warfarin	варфарин
whooping cough	коклюш

X

xanthelasma	(плоская) ксантелазма, плоская ксантома
xanthoma	ксантома (патологическое образование из перегруженных липидами макрофагов в коже, сухожилиях и др. локализации при нарушении липидного обмена)
xanthomatosis	ксантоматоз, экстрацеллюлярный холестериноз (*устар.*), болезнь Керля — Урбаха
xiphoid process = xiphoid appendix = metasternum	мечевидный отросток (грудины)

Y

yeast	дрожжи, дрожжевой
yellow fever (*sl.*: yellow jack)	желтая лихорадка

Z

Zahn's thrombus	тромб Цаана, белый тромб
zygote	зигота
zygomatic	скуловой(ая)
zymosan	зимозан

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His research interests spread in the fields of Neuroendocrinology, Cardiology, Gastroenterology, Medical Ethics, Adolescent Medicine and Geriatrics. He has published (in co-authorship with L.P. Churilov) a unique volume: Manual in Adolescent Endocrinology (2004), and also several other monographs and textbooks on Internal Medicine, Pathology and Endocrinology, totally over 400 academic papers, more than 50 dedicated to thyroid pathology.

He writes also on the subjeets of History of Medicine, Literature and Art Science. His essays on the role of A.M. Gorkiy in the organization of the first act of humanitarian aid in the world history, on the verses by A.S. Pushkin and "Amber Chamber" at Tsarskoye Selo, on the possible prototypes of Bazarov, a hero of Turgenev's novel "Fathers and Sons" – are known not only in medical community, but also among art scientists.

Under his supervision 4 Ph. D. Theses in Medicine successfully defended. He is participant or co-organizer of many international, all-Union and All-Russian scientific congresses, conferences and symposia, co-editor of Russian edition of "Diabetes A to Z" Encyclopedia of American Medical Association, member of the Board of Russian Cardiologic and Botkin's Russian Therapeutic Societies, Laureate of Diplomas and Medals of the USSR for medical innovations and scientific achievements, including pioneering works in infrared medical monitoring. Certificate of honors of St. Petersburg State University for scientific contribution (2006).

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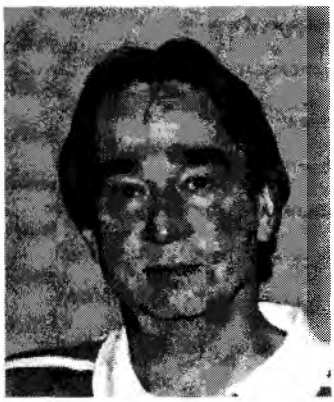
Steven Huneycutt was born at Cleburne, Texas. He is a specialist in Family, Internal and Emergency Medicine. His training took place in three countries, the United States of America, the Russian Federation and Finland, which gave him a broad perspective in treating illnesses and good knowledge of different health care systems and medical traditions through Europe and North America. In 1975 he graduated from University of Texas (Chemistry major), in 1977 – from San Jacinto Junior College, Pasadena, Texas (Biology major) and in 1979 – from Texas Chiropractic College, Pasadena, Texas, where he was conferred upon Doctor of Chiropractic degree. During 1979–1993 he practiced as a chiropractor at Burleson, Texas, then entered M.D. Program in Russia (1993–1997). In 1997 he obtained M.D. degree from St. Petersburg State Pediatric Medical Academy and completed European part of his medical education with Family Practice internship at Lapeenranta, Finland. Later in 1999–2002 he accomplished Family Practice residency with Michigan State University and became an Urgent Care physician for Covenant HealthCare (2002–2003), and later Family Practitioner for Freeman HealthCare (2003–2005). In 2005–2008 he worked as Medical Director for the Management Services Organization at Covenant HealthCare, in 2008–2011 – as an Emergency Room doctor for Borgess of Woodbridge Hills IMC. During work on this book Dr. Huneycutt was employed as a full time family medicine physician by Banner Health and at the same time was medical director for the cardiovascular rehabilitation department. In November, 2011 he opened private family medicine clinic at Loveland, Colorado.

Dr. Huneycutt has authored 3 academic papers (2 of them – in Russian). He has some experience of medical teaching as an Assistant clinical professor in Family Medicine for Michigan State University College of Human Medicine at Saginaw Campus (1999–2002). He is Guest Faculty (2008) for Health Care Compliance Association (HCCA) and fellow of the American Academy of Family Physicians (FAAFP) (2005), also.

Dr. Huneycutt was a Member (1999–2009) of American Medical Association (AMA). He holds a certificate of American Board of Family Practice (2003–2013). He has always bon politically active and responsible as a citizen: served as a county water board member in the State of Texas and later as a city mayor of his home city. In addition he served as a police department administrator. His hobbies are aviation, restoring antique autos and spending time with family wife and 2 children.



Scoggins William Gene, M.D., D.C. (1948–2010)

William Scoggins was born in the 8th January 1948. He spent his childhood in a small farming community in Visalia, in the beautiful San Joaquin Valley in California. On leaving college with a degree in biology, his love of adventure and travel brought him to the oil fields of Alaska. He lived for many years in this rugged environment where he worked with a survey crew for oil exploration.

His interest in chiropractic treatment developed when he suffered a work-related injury. The effective treatment he received from a chiropractor brought about a complete recovery and impressed him so much that he decided to pursue a career in chiropractic care.

In 1988 he graduated from the University of California with a Bachelor of Arts Degree. He studied at the National College of Chiropractic in Lombard, Illinois, where he graduated in 1991 with a Doctor of Chiropractic Degree. In that same year he joined the Murphysboro Chiropractic Center in Southern Illinois. He moved to Alabama in 1992 to continue his practice. He was a respected and popular chiropractic physician and, over the following years, Dr Scoggin's interest in travel was to bring him far beyond his native homeland.

By 1996 he was living and practicing as a chiropractor in Finland. From there he moved to St Albans in England where he settled in 1998. In that same year he received his MD degree from the St Petersburg Paediatric Medical Academy in Russia.

He made his final home in Dublin, Republic of Ireland's capital city in 2000. Over the following six years he practiced with Owen Dennis and Associates in their Finglas and Clontarf Chiropractic Clinics. He established a reputation for his expertise both as a chiropractor and physiotherapist, and was widely respected for his diagnostic abilities. Dr Scoggins (known to us all as Bill) was a highly valued member of staff and a wonderful friend to everyone who worked in the clinics.

In October 2006 he opened his own chiropractic clinic in Raheny, Dublin. His love of learning never waned and he soon developed an interest in Cold Laser Therapy, and its benefits in reducing pain and inflammation. To study and increase his knowledge of this therapy, he returned to Russia, where he had made many friends among his professional colleagues when he was studying for his MD degree. He graduated from postgraduate program in Laser Medicine in Moscow (State Research Centre of Laser Medicine). On his return to Raheny, he provided this treatment to his patients, and his Chiropractic/ Physiotherapy/ Laser clinic was recognised as a centre of excellence.

Tragically, his productive and eventful life came to an end on the 20th October 2010. But his determination to fight his cancer was so strong that he managed to run his clinic and administer to his patients up until the final two weeks before he passed away.

Dr Scoggins was known for his sense of humour, his interest in writing, reading, learning foreign languages and classical music. Feodor Dostoyevsky was his favourite writer, whom he was able to read in the original language. He also enjoyed the Scottish bagpipes which he had learned to play when he was working at the oil pipeline in Alaska.

But he is remembered mostly for his kindness to his patients, friends and professional colleagues. He was unstinting in his work, always eager to acquire new medical information and new methods of treatments that he could use to alleviate the suffering of others. He was much loved and is missed by all of us who had the pleasure of knowing him.

LIST OF ABBREVIATIONS

English

D.C. – Doctor of Chiropractic
F.A.A.F.P. – Fellow of American Association of Family Practitioners
GB – Great Britain
lat. – Latin
M.D. – Doctor of Medicine, from lat.: Medicinae Doctor
MA – Massachusetts
pl. – plural
Ph.D. – Philosophy Doctor, lat.: Philosophiae Doctor
sl. – slang
syn. – synonym(s)
TB, TBC – tuberculosis
USA – United States of America

Русские

букв. – буквально
греч. – греческое
лат. – латинское
мн. – множественное
напр. – например
прав. – правильнее
рус. – русское
СНГ – Содружество Независимых Государств
тж. – также
устар. – устаревшее
франц. – французское

БИБЛИОГРАФИЯ

Ниже приведены источники, использованные при создании авторских текстов для данной книги и источники адаптированных текстов.

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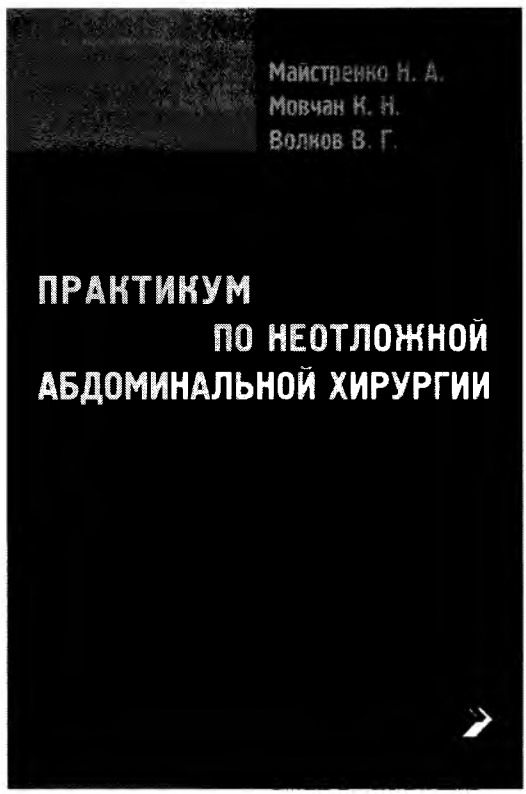
Основной проблемой подготовки студентов медицинских вузов по хирургии является недостаточный уровень их знаний в вопросах оказания медицинской помощи больным и пострадавшим при острых заболеваниях и травмах органов брюшной полости. Сведения по данному вопросу, представленные в учебниках по частной хирургии, не позволяют обучающимся концентрированно сосредоточиться на изучении этого раздела хирургии на фоне нарастающего потока информации. Предлагаемый практикум предусматривает дополнение содержания учебников, не повторяя его, а акцентируя внимание обучающихся на тактических и технических принципах при лечении больных, оказании хирургической помощи больным и пострадавшим при острых заболеваниях и травмах органов брюшной полости.

Практикум рассчитан на студентов медицинских вузов, а также врачей первого уровня последипломного обучения.



Зубик Т. М., Жданов К. В., Ковеленов А. Ю., Левшанков А. И.

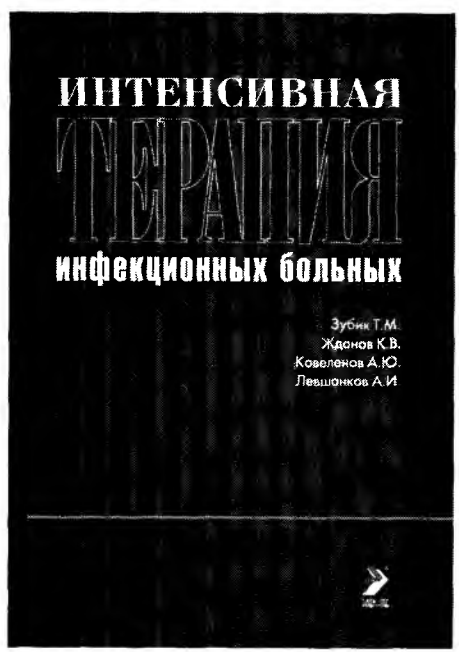
Интенсивная терапия инфекционных больных

Руководство для врачей.

СПб.: ЭЛБИ-СПб., 2010. — 304 с.: ил.
ISBN 978-5-93979-260-8

В книге представлены семиотика критических состояний в инфекционной патологии, а также свойственные им функциональные изменения, нарушения гомео- и гемостаза. Изложены организационные принципы интенсивной терапии инфекционных больных, общие мероприятия по уходу, питанию, коррекции нарушений внутренней среды организма. Приведены основные этиологические факторы, патогенез и патофизиология, клиника, диагностика и лечение инфекционнотоксического шока, инфекционнотоксической энцефалопатии и церебральной гипертензии (отека набухания головного мозга), острого обезвоживания, острых дыхательной, печеночной и почечной недостаточности при тяжелом, критическом течении различных нозологических форм инфекционных заболеваний. Представленные в книге сведения подчинены, прежде всего, задачам ранней диагностики и интенсивной терапии больных в условиях как специализированных (инфекционных) отделений, так и ОРИТ.

Книга предназначена для инфекционистов, реаниматологов, врачей скорой помощи, терапевтов и невропатологов, а также слушателей курсов усовершенствования врачей по соответствующим специальностям.

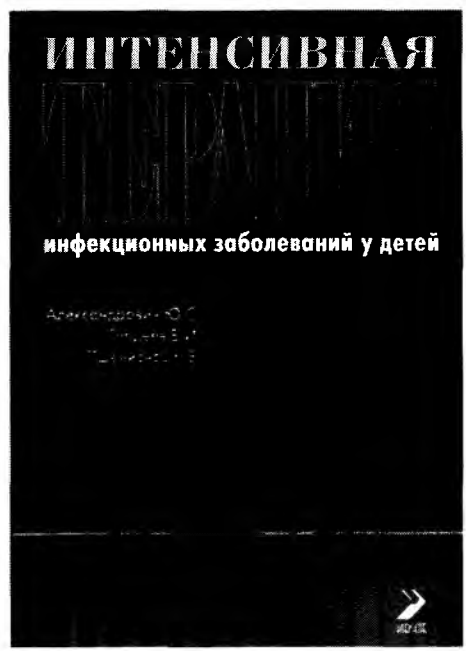


Александрович Ю. С., Гордеев В. И., Пшениснов К. В.

Интенсивная терапия инфекционных заболеваний у детей

СПб.: ЭЛБИ-СПб, 2010. — 320 с.
ISBN 978-5-93979-247-9

В предлагаемом издании представлены основные принципы диагностики и интенсивной терапии критических состояний у детей, обусловленных инфекционной патологией. Рассмотрены современные классификации сепсиса, которые имеют не только научное, но и большое практическое значение, поскольку позволяют существенно улучшить качество интенсивной терапии сепсиса и септического шока у детей. Особое внимание уделено вопросам диагностики и интенсивной терапии наиболее часто встречающихся инфекций детского возраста, даны четкие практические рекомендации по терапии жизнеугрожающих состояний, обусловленных инфекционными заболеваниями и сепсисом. Одним из достоинств книги является и ее многогранность, поскольку она рассматривает все ключевые вопросы интенсивной терапии в педиатрической инфекционной клинике. Книга предназначена для анестезиологов-реаниматологов, педиатров, инфекционистов, врачей "скорой медицинской помощи", а также будет полезна всем специалистам, оказывающим помощь детям, и научным работникам, занимающимся проблемами сепсиса у детей.



Колобов А. В., Цинзерлинг В. А., Смирнова Е. А., Рощупкина И. А.

Плацента человека. Морфофункциональные основы

Учебное пособие.

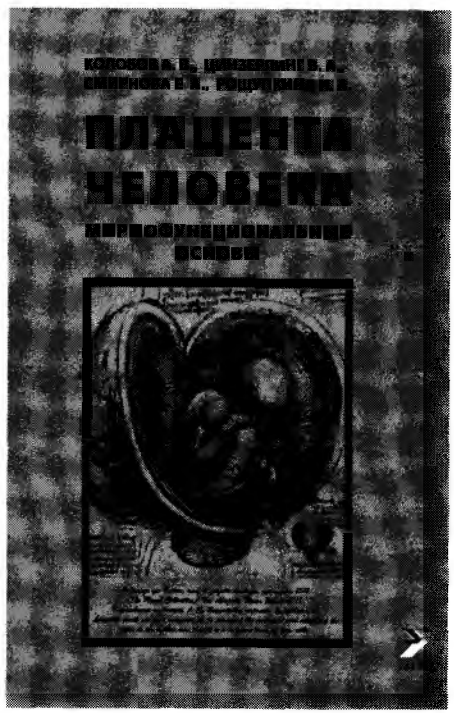
СПб.: ЭЛБИ-СПб, 2011. — 80 с.: ил.
ISBN 978-5-93979-025-7

Книга посвящена уникальному органу репродуктивной системы человека — плаценте.

Учебное пособие содержит современные сведения о макроскопическом и микроскопическом строении плаценты человека, основных особенностях ее развития. Преимуществом пособия является не только морфологическое описание ткани плаценты, но и авторский иллюстративный материал, а также подробное описание функциональных характеристик различных клеток плаценты. Кроме того, проводится сопоставление морфологических и эхографических данных.

Приведенные в книге сведения об эндокринной функции ткани плаценты и особенностях иммунных реакций, протекающих в плаценте, позволяют считать ее одним из важных отделов диффузной иммуно-нейроэндокринной системы. В книге также представлены современные сведения об основных патологических процессах в ткани плаценты.

Учебное пособие предназначено для студентов-медиков, но может представлять интерес и для студентов биологических специальностей, а также для всех, интересующихся репродуктологией.



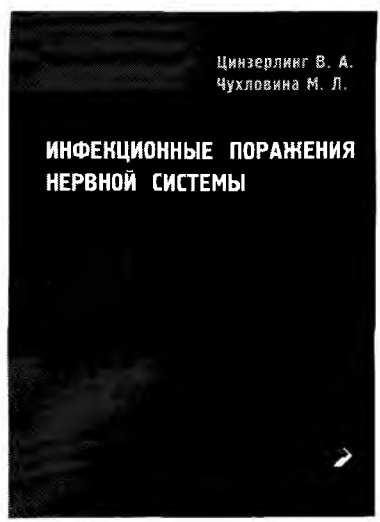
Цинзерлинг В. А., Чухловина М. Л.

Инфекционные поражения нервной системы. Вопросы этиологии, патогенеза и диагностики

Руководство для врачей. — СПб.:

СПб.: ЭЛБИ-СПб, 2011. — 584 с.
ISBN 978-5-91322-027-1

В руководстве систематизирован многолетний опыт клинической и морфологической диагностики инфекционных процессов в центральной и периферической нервной системе, а также модельных экспериментах на животных. На основании сопоставления результатов собственных, во многом приоритетных исследований, разнообразных по этиологии и топографии патологических процессов у пациентов во всех возрастных группах (новорожденных, детей, взрослых) и у плодов с данными современной литературы дается развернутая клинико-морфологическая характеристика всех актуальных для России в настоящее время нозологических форм. Подробно обсуждаются вопросы патогенеза нейроинфекций, принципы их диагностики и формулировки диагноза. Приводятся некоторые доступные из открытых источников информации и результатов собственного анализа статистические данные. Вопросы патоморфологии различных поражений нервной системы, вызванных биологическими возбудителями, в столь полном виде в мировой литературе излагаются впервые. Книга иллюстрирована 116 оригинальными рисунками и 54 содержащими фактические данные таблицами. Важным отличием данного руководства от других изданий является подробное рассмотрение наряду с классическими нейроинфекциями и менее известных, но часто встречающихся процессов, вызванных различными вирусами, микоплазмами и хламидиями. В руководстве приведено значительное количество примеров из клинической и патологоанатомической практики.



Во второе издание внесены существенные дополнения в большинство глав, базирующиеся как на данных полученных авторами в последние годы, так и литературных источниках.

Руководство может быть рекомендовано для широкого круга врачей и в первую очередь специалистов связанных с патологией нервной системы: неврологов, инфекционистов, терапевтов, педиатров, неонатологов, семейных врачей, патологоанатомов, судебно-медицинских экспертов, организаторов здравоохранения. Оно может быть полезным, как для студентов, так и в системе последипломного образования по перечисленным специальностям.

Колбанов В.В.

Валеологический практикум

Учебное пособие

СПб.: ЭЛБИ-СПб, 2011. 224 с.
ISBN 978-5-93979-280-6

Валеологический практикум предназначен прежде всего для освоения методов самоконтроля, экспресс-диагностики и коррекции различных компонентов здоровья и образа жизни.

Практикум знакомит читателя с основными методическими подходами к изучению типологических особенностей, текущего функционального состояния, работоспособности и здоровья не с общепринятых позиций клинической медицины (отрицания болезни), а в плане позитивного анализа наличного потенциала здоровья и уровня благополучия человека. В тематике практических занятий предусмотрено знакомство педагогов с основными мероприятиями, направленными на формирование, сохранение и укрепление здоровья субъектов образовательного процесса.

Предлагаемое учебное пособие содержит разработки практических заданий по экспресс-диагностике, текущему контролю и самоконтролю здоровья, функциональных состояний и образа жизни человека с учетом его возрастных и индивидуальных особенностей. Практикум построен в соответствии с направлениями работы педагога-валеолога по обеспечению здоровья субъектов образовательного процесса.

Материалы практикума могут быть использованы руководителями образовательных учреждений, педагогами-валеологами, педагогами других специальностей, студентами педагогических вузов и колледжей, а также всеми, кто интересуется проблемами формирования собственного здоровья и образа жизни.

Шостак В.И., Лытаев С. А., Березанцева М. С.

Психофизиология

Учебное пособие

СПб.: ЭЛБИ-СПб, 2007. – 352 с.
ISBN 978-5-93979-186-1

В последнее десятилетие в нашей стране явно возрос интерес к психологии и, соответственно, к психологическому образованию. Это способствовало появлению и развитию новых форм обучения, существенно отличающихся от традиционных, классических методов. В частности, на базе высшего психологического образования (гуманитарного, естественнонаучного, медицинского, технического и др.) функционируют циклы переподготовки, повышения квалификации, ускоренного второго высшего образования. Имеющаяся учебная литература, особенно по естественно-биологическим дисциплинам, не всегда является адекватной для таких форм обучения, что ставит обучающихся в затруднительное положение. На основании большого опыта, накопленного на факультете психологии СПбГУ и в Республиканском гуманитарном институте СПбГУ авторы предприняли попытку создания учебного пособия по психофизиологии в соответствии с планами подготовки указанных форм обучения и доступного всем категориям обучающихся. Этими соображениями объясняется структура учебного пособия, посвященного морфо-функциональной характеристике нервной системы, физиологии сенсорных систем и физиологии высшей нервной (психической) деятельности. Учебное пособие предназначено для студентов психологических факультетов, может быть использовано студентами медицинских вузов при изучении соответствующих разделов физиологии, будет полезным клиницистам-неврологам, психиатрам, клиническим психологам.

Под редакцией Г.А. Софронова, М.В. Александрова

Экстремальная токсикология

Учебник

СПб.: ЭЛБИ-СПб, 2012. — 256 с.
ISBN 978-5-93979-288-2

Учебник разработан в соответствии с программой по токсикологии и медицинской защите в рамках учебной дисциплины «Медицина катастроф и безопасность жизнедеятельности».

На современном уровне изложены общие механизмы токсического действия химических веществ. Дана токсикологическая характеристика актуальных веществ, представляющих опасность массовых поражений населения при чрезвычайных ситуациях мирного времени, в результате ведения боевых действий.

Учебник рекомендован студентам медицинских вузов. Может быть использован при дополнительном и послевузовском образовании врачей для изучения основ токсикологии.

Пособие основано на преподавании языка через профессиональный предмет медицины. Оно содержит авторские тексты, представляющие собой фрагменты учебников патофизиологии и пропедевтики внутренних болезней.

В книге используется аудиоматериал, озвученный носителями языка – врачами профессионалами, изнутри знающими как англо-американское, так и отечественное здравоохранение и медицинское образование.

Пособие написано живо и интересно, содержит много исторических и медицинских фактов, биографий известных англоязычных и русскоязычных ученых–медиков, которые используются в качестве обучающего материала, и может с успехом применяться при занятиях английским языком для медиков, получивших образование в России и планирующих или продолжающих свою карьеру в англоязычных странах.

Руководство
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