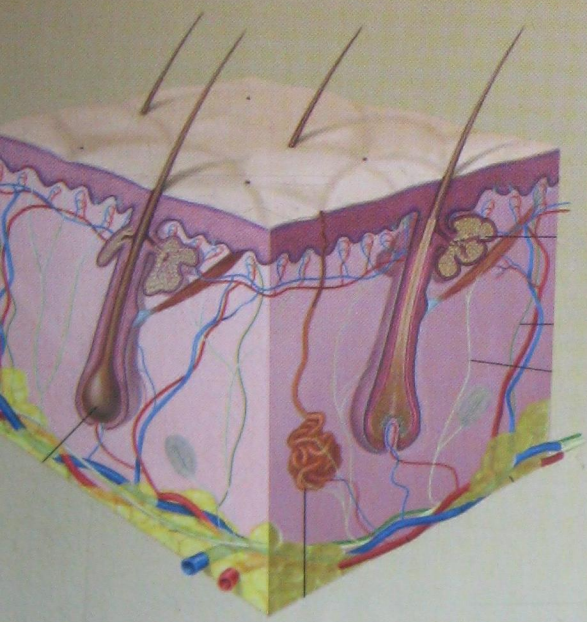
DERMATOLOGY VENEREOLOGY

Under the editorship of Prof. V.I. Stepanenko



MINISTRY OF HEALTH OF UKRAINE

BOGOMOLETS NATIONAL MEDICAL UNIVERSITY

DERMATOLOGY VENEREOLOGY

Under the editorship of Prof. V.I. Stepanenko

Recommended by the Ministry of Public Health of Ukraine as a textbook for students of higher medical institutions of IV accreditation level

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D36 **Dermatology, Venereology.** Textbook / under the editorship of Prof. V.I. Stepanenko. – Kyiv.: KIM, 2013. – 560 p., 257 pic.

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The Textbook has been prepared by employees of the Dermatology and Venereology Subdepartment of Bogomolets National Medical University and leading dermatovenerologists of related field subdepartments of Ukrainian higher medical educational institutions, under the general editorship of Prof. V.I. Stepanenko, the Head of the Base Dermatovenereology Subdepartment, Ministry of Public Health of Ukraine.

The Textbook presents modern concepts of skin and skin adnexa etiology and pathogenesis. These diseases are mainly transmitted sexually. Clinical views and courses of numerous skin diseases and infections transmitted, mainly, sexually are described in the book along with newest methods of their diagnostics and treatment.

The Textbook has been prepared based on the Typical Programs for Students of IV Accreditation Level Higher Medical Educational Institution Medical and Dental Departments, such as Skin and Venereal Diseases (Ministry of Public Health of Ukraine, 2007) and General Family Medicine Practice (Ministry of Public Health of Ukraine, 2010) according to Bologna Process requirements. The Textbook includes subjects mandatory for studying taking into account plans of lecture and hands-on training topics. It also includes a number of other dermatology and venereology topics necessary for understanding differential diagnosing of nosologic skin pathology forms. Additionally, the Textbook includes topics of Medicine Cosmetology and Skin Neoplasms elective courses. The book includes pictorial material prepared based on clinical observations of M.M. Shupenko, a co-author of the Textbook, Associate Professor of the Dermatology and Venereology Subdepartment, Bogomolets NMU, along with individual pictures of exclusive items exhibited at the ceroplasty museum of the subdepartment.

For Students of IV Accreditation Level Higher Medical Educational Institutions of Ukraine, interns, and medical practitioners.

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GENERAL ISSUES on DERMATOLOGY and VENEREOLOGY

SPECIAL DERMATOLOGY

SEXUALLY TRANSMITTED INFECTIONS (VENEREAL DISEASES)

PREFACE

Dermatology and Venereology is a section of clinical medicine including dermatology (derived of Greek *δερμα*, skin, and *λογοζ*, teaching), a theory of skin structure, functioning, and diseases, and venereology (after Venus, a love goddess in Roman mythology), a venereal disease science. The two different sciences were combined based on the fact that enanthesis is a symptom of some venereal diseases.

In the last decade, the «venereal diseases» term is often replaced with «diseases of primary sexual transmission» or «sexually transmitted diseases», which is caused by considerable growth of diseases where possibility of sexual agent transmission was identified.

Currently, the number of benerial disease nosologies and clinical forms is about two thousand. Besides, over 20 diseases are known to be transmitted sexually.

The Textbook includes considerations, from the modern point of view, of skin disease etiopathogenesis, clinical view, clinical course, and classification, along with diseases transmitted sexually, existing, including innovative, diagnosis methods and criteria of differential diagnostics for these diseases, modern methods of their treatment and prophylaxis.

Learning dermato-venereology basics by students of medical and dental departments on the forth year of their study in IV accreditation level higher medical educational institutions of Ukraine is a mandatory step of the general system of studying clinical medicine by future doctors, especially of internal and infectious diseases, urology, obstetrics, and gynaecology. Regarding child's dermato-venereology, it is close associated with neonatology and pediatry.

As a separate clinical subject, dermato-venereology is subdivided into separate and special ones. Its studying presupposes acquiring knowledge of etiology, pathogenesis, clinical symptoms of various dermatologic pathologies as well as mastering modern approaches to their diagnosis treatment, and prophylaxis. When studying dermato-venereology, medical student must master their skills of identifying, exactly and I due time, clinical symptoms of various skin diseasesskin diseasesskin desiases and sexually transmitted infections, deontologically correct communication with patients, and proper caring for them.

Mastering the dermato-venereology study program set forth in this book is expected to form clinical and prophylaxis thinking of medical students – specifically, understanding medical and medico-social significance of dermatovenereology problems – as well as grounding the necessity of specific prophylaxis measures.

The purpose of this Textbook is to ensure fulfillment of the Bologna Process requirements, specifically to provide modern theoretical knowledge and help students to master practical skills to be formed following the principle of independent elaboration of every subject and lection at extracurricular studying and hands-on training.

The Textbook material is set forth on modern professional level with elements of inter-subject integration. Taking into consideration rather large number of skin diseases and infections transmitted sexually, the Textbook makes it possible to imprint knowledge of theoretical material and assess students' ways of thinking based on answering self-control questions using the acquired skills.

In conclusion, I feel it my pleasant duty to express my deep gratitude to senior laboratory assistants of the Dermatology and Venereology Subdepartment, Bogomolets National Medical University, Cand. Sc. (Biology) V.T. Gorgol and L.A. Naumova.

We ask all the Textbook users to address their comments and suggestions to Bogomolets National Medical University, address: Dermatology and Venereology Subdepartment, 13 Taras Shevchenko Boulevard, Kyiv, 01601.

Prof. V.I. Stepanenko

ABSTRACT
TO THE «DERMATOLOGY, VENEREOLOGY» TEXTBOOK

The Textbook has been prepared by employees of dermatology and venereology subdepartment, Bogomolets National Medical University, and leading dermato-venerologists of related field subdepartments of Ukrainian higher medical educational institutions under the general editorship of Prof. V.I. Stepanenko, the Head of the Base Dermatovenereology Subdepartment, Ministry of Public Health of Ukraine.

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The edition makes it possible for students to orientate themselves in various clinical symptoms of skin and infectious diseases of primarily sexual transmission, provide differential diagnosing, administer proper therapy, using modern drugs, and provide patients with recommendations aimed to prevent diseases and relapses of a number of inflammation processes along with prophylaxis of their further spread.

The authors of the Textbook took into consideration modern teaching requirements and new, creative approach to independent students' working taking into account the necessity of transfer to European education principles and control of theoretical knowledge, skills, and practical experience, taking for the base the longstanding experience of teaching this subject in higher medical educational institutions of Ukraine.

The Textbook is purposed for students of IV Accreditation Level Higher Medical Educational Institutions of Ukraine, interns, and medical practitioners.

V.I. Stepanenko

1 TOPIC

Introduction to Dermatovenerology

Dermatovenerology is a clinical medical science to study objective and subjective features of a variety of clinical manifestations of human skin and mucous membrane lesions in their unity and interaction with internal organs and systems, as well as environment in general.

TRAINING AND EDUCATIONAL PURPOSES

- To analyze the stages of formation of dermatovenerology as an individual clinical science
- To define the main achievements of global and domestic dermatovenerology

TO KNOW:

- the value and place of dermatovenerology in residency training;
- definition of dermatological pathology as a medical-psychological problem;
- the general description of historical development of dermatovenerology (the role of works by Hippocrates, Cornelius Celsus, Avicenna, Mercurialis, Whelan, etc.);
- data on formation and development of various foreign schools of scientific dermatovenerology in the XIX–XX centuries (French and German schools of dermatology, their founders and prominent representatives – Jean Alibert, F. Gebre, K. Gibert, M. Kaposi, etc.);
- the contribution of the founders of Russian school of dermatovenerology – Polotebnov A.V., Tarnowski V.M., Pospelov A.I. and others in development of dermatovenerology;
- significant contribution of Ukrainian school of dermatovenerology (Stukovenkov M.I., Nikolsky P.V., Zelenev I.F. and others) in global dermatology.

TO BE ABLE TO:

- distinguish the main phases of development of dermatology and venereology;
- evaluate evolutionary breakthroughs of global and domestic science towards development of diagnostic methods and pathogenic therapeutic regimens of dermatovenerologic patients;
- manage basic deontological principles of dermatologist.

Dermatology: Stages of Development

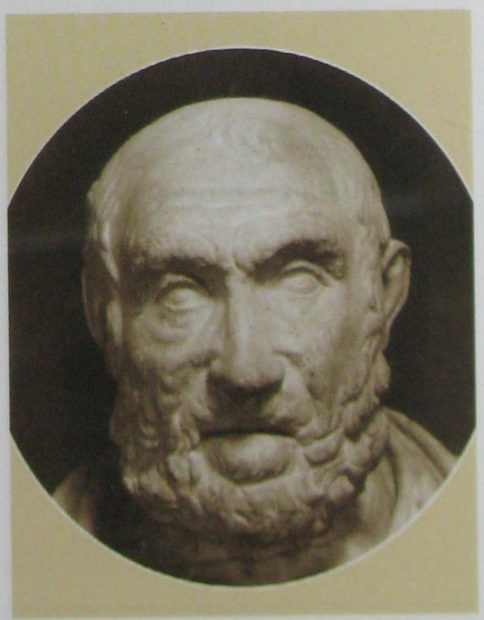
Dermatology is the science to study skin. Dermatology studies skin structure and functions in normal condition and upon incurrence of any skin problems from different points of view: anatomical, physiological, clinical, biochemical, histochemical, immunological and others.

Dermatology is closely related to other areas of clinical medicine: venereal diseases, infectious diseases, medical diseases, pediatrics, endocrinology, neuropathology, psychiatry, etc. At present, the fact that skin is a body linked to the whole organism with circulatory, lymphatic, endocrine and nervous systems is considered an axiom. In addition, skin is an important part of the immune system. Respectively, visceral diseases affect skin condition to any extent. As the outer body covering, skin is affected by a variety of harmful environmental factors that can manifest itself in a barely noticeable to severe form.

As science, dermatology originated in XVIII century. Though, a number of skin diseases are known to mankind since ancient times. Development of dermatology is closely linked to advancement of natural science and social systems, which allows studying the history of dermatology in stages.

The first phase refers to the time of primitive society and formation of the slave-owning system. Definitely, primitive man paid attention to various skin lesions, responded to itching caused by different pathological processes and noticed that some plants helped to reduce, while others just increased irritation. From generation to generation, people accumulated observations over the course of various skin diseases and means to treat them.

With the advent of writing, the accumulated experience made its appearance in medical treatises. The first data on skin diseases is recorded in ancient China, India, Egypt and Greece. In particular, a doctor known as Goang-Tee systematized medical knowledge of that time in China in 2637 BCE. The diseases described in his treatise involved some cases of dermatopathy: mange, furuncle, carbuncle, erysipelas, impetigo, ichthyosis, eczema, leprosy, alopecia, vitiligo, and others. The work also reported on methods of treatment of these diseases.



Hippocrates
(460-370 years. BCE)

Ancient Indian medical treatises (2000-700 years. BCE) described numerous skin diseases, as well as over seven hundred of different plants used to treat them.

Egyptian papyrus dated by 1700 BCE contains a list of drugs for various diseases, including scabies, as well as a large number of recommendations on hygienic skin care. Dated

by 1500 BCE, so-called books of Moses describe leprosy (*lepra*), loss of skin pigment (*vitiligo*) and hair loss (*alopecia*).

In ancient Greece and Rome, people also studied dermatology. The greatest of Greek physicians of the time, Hippocrates (460-370 yrs. BCE), is the founder of scientific medicine. He summarized and systematized known data on skin diseases, described leprosy, scabs, alopecia, herpes and other dermatoses (more than 40 clinical entities). Hippocrates was the first to focus on the link of skin diseases with changes of internal organs and influence of environmental factors. In his view, human skin diseases associated with some pathology of internal organs were predetermined by the «spoiled juices» of the body (humoral theory).

Aulus Cornelius Celsus
(25 BCE – 50 CE)

In ancient Rome, science in general, including medicine, reached a higher level. Descriptions of some skin diseases become more detailed, while new dermatological terms appear; some of them are used to date.

The 1st century BCE allocates an outstanding physician, Aulus Cornelius Celsus, who left a detailed description of many skin diseases, including furunculus, carbuncles, barber's rash, psoriasis, erysipelas. In treatment of eczema and similar lesions, Celsus advised to use lead and sulfur ointment. His successor, Galen (2nd century BCE), attempted to study skin structure. He proposed a classification of skin diseases based on their location: diseases of head, arms, legs and other body parts.

Avicenna (Abu Ali Hussain
Ibn Abdallah Ibn Sina)
(980-1037 yrs.)

In the following century of Great Migration, general science, including medicine, is falling into decay. The second stage in the development of medicine, particularly dermatology, belongs to the period of Middle Ages. Instead of slave-owning system, European countries welcome feudalism. The center of medical research is moving to the East: to Arab countries. On the border of the X-XI centuries CE, the world received the most famous work of the outstanding scientist and physician, Abu Ali Ibn Sina, better known as Avicenna. His «Canon of Medicine» was the first paper to successfully encyclopedize and

summarize the medical knowledge of the time. Avicenna devoted considerable attention to the study of skin diseases. He described pemphigus, leishmaniasis, and hives; knew how to differentiate leprosy from elephantiasis, pruritus of scabies; addressed the issues of cosmetology.

In the second half of the XVI century, Europe welcomed the third stage in the development of medicine, particularly dermatology. This period is associated with the decay of feudal society, which is replaced by capitalist socio-economic system. There comes development of natural sciences, physics, chemistry, medical science, especially anatomy and physiology. During this period, the first books of skin diseases appear. The work of an Italian scientist Jerome Merkurialis (1572) was considered most popular paper of the time. Girolamo Mercuriale provided all previously described skin conditions, but failed to classify them.

Jerome Merkurialis
(Girolamo Mercuriale)
(1530-1606 yrs.)

As an independent scientific medical discipline, dermatology was allocated in the second half of the XVIII century. Morphological direction comes to the fore. This fact involves the need to organize and unify the description, as well as interpret manifestations of skin diseases: morphological elements of the rash. There appears a set of textbooks, particularly a book by an Austrian doctor, Plenk (1772), in which he attempts to classify skin diseases basing on morphological characters; a textbook by a French doctor, Lorrie (1777), in which he described the anatomy and physiology of skin and tried to identify the etiology and pathogenesis of some skin diseases; a textbook by an English doctor, Wheellen (1798), who succeeded to classify skin diseases by primary morphological features.

Jean-Louis Alibert
(1768-1837 yrs.)

In the early nineteenth century, first dermatological schools are formed. Establishment of dermatology as a separate field of clinical medicine is completed. Formed in 1801 in Paris and based on the Saint-Louis Hospital, French Dermatovenereological School was the first institution for patients with skin diseases. Jean-Louis Aliber (1768-1837

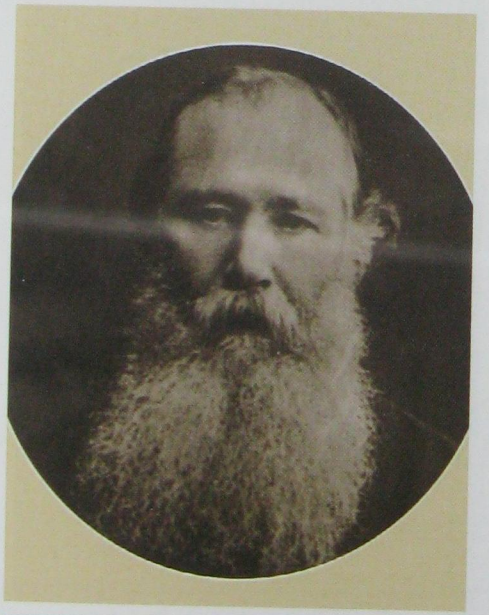
yrs.) is justly considered the school's founder. He introduced the terms of «dermatitis», «syphilide» and «ichthyosis». Talented students and followers of Aliber, particularly Biett (1781-1840 yrs.), Cazenave (1795-1877 yrs.), Gibert (1796-1866 yrs.), Vidal (1825-1893 yrs.), Bazin (1807 -1878 yrs.), Hardy (1811-1893 yrs.) are also to be noted. The concept based on the fact that the main cause of skin diseases is presented by humoral factors, so-called dyscrasia and diathesis, is typical for French dermatological school.

Ferdinand von Hebra
(1816-1880 yrs.)

Vienna (German) Dermatologic School was formed in the 40s of the XIX century. Ferdinand von Hebra (1816-1880 yrs.) is considered the school's founder. F.Hebra believed that postmortem skin studies were of biggest importance in the study of skin diseases. He created an appropriate classification of dermatoses. Considering the majority of skin diseases as local processes mainly caused by external factors, Hebra reduced the value of domestic factors in the etiology of dermatoses to a minimum.

Divergent views on the nature of skin diseases caused long controversy between the French and Viennese dermatological schools, which ended only in the early twentieth century.

Among talented members of the Vienna (and hence German) dermatological school of the nineteenth century, the following scientists are to be noted: Simon (described *Demodex folliculorum*), Kaposi (investigated idiopathic hemorrhagic sarcoma, known as Kaposi's sarcoma), Neumann (identified vegetating pemphigus), Auspitz (determined a triad of symptoms characteristic of psoriasis), Koebner (established the phenomenon of isomorphic reaction in psoriasis).



Polotebnov A.G.
(1838-1907 yrs.)

In other countries, development of dermatology was based on views and attitudes of the French or Vienna dermatological schools and featured no significantly independent, original peculiarities.

In the Russian Empire, formation of scientific Dermatology and Venereology took place in parallel with evolution of the university

medical education. As an independent discipline, dermatology emerged in the 80-ies of the XIX century. By that time, it was a part of general medicine, while treatment of skin and venereal diseases was given to physicians and surgeons. In universities, students studied skin diseases rather formally, while individual lectures on this discipline were prelected by professors of different departments. In particular, since in 1827 the St. Petersburg Medical-Surgical Academy provided lectures on skin diseases by pharmacists and physicians; the data about sexually transmitted diseases was provided within surgical curriculum. Only in 1863, when a new charter was approved by universities, the curriculum of medical schools included a course of skin (at the Department of Special Pathology and Therapy) and sexually transmitted (at the Department of Theoretical Surgery) diseases. In the University of Kiev, the course of skin diseases was prelected by the associate professor Goretsky L.K. from 1864 to 1880, while the same course was prelected by the associate professor Naydenov D.I. at Moscow State University from 1869 to 1884. In St. Petersburg Medical-Surgical Academy, a course of skin diseases was started by Podkopaev F.V. in 1869; in 1871, the course was continued by the associate professor Polotebnov A.G.

In 1876, Polotebnov A.G. became a professor and the first academic dermatologist in Russia. He is considered the founder of the Russian scientific dermatology. Professor Polotebnov A.G. treated skin lesions strongly correlated with the injuries of visceral organs and nervous system, as well as the influence of environmental factors. He paid much attention to unification of morphological and functional studies in dermatology, which is still important at the present day.

In 1869, Tarnovsky V.M., who worked at the St. Petersburg Medical-Surgical Academy along with Polotebnov A.G., introduced a course of syphilology. He is considered the founder of Russian scientific venereology. When Polotebnov A.G. retired in 1894, Tarnovsky V.M. headed the Joint Department of Dermatology and Syphilitic Diseases. Within the period from 1898 to 1924, Professor Pavlov T.P. headed the Department. He became one of the founders of functional approach in dermatology.

The representatives of the St. Petersburg (Leningrad) Dermatovenerological School that made a significant contribution to this field of medicine also include: Peterson O.V. (the study of leprosy, chancroid and leishmaniasis), Pavlov S.T., Podvysotskaya O.N., Aravyisky A.M., Kashkin P.G., Zverkova F.A., Kozhevnikov P.V., Shaposhnikov O.K., Samtsov V.I. etc.

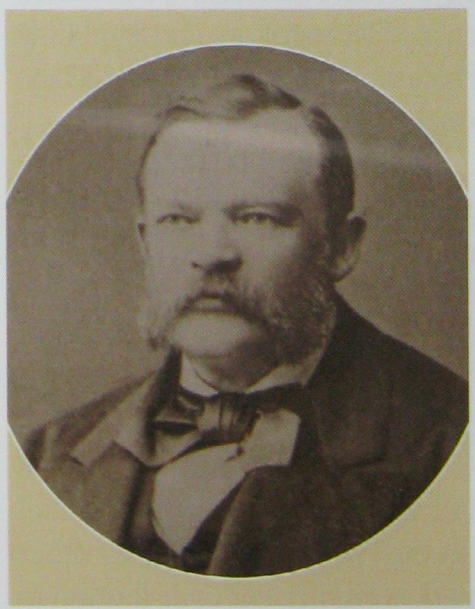
At the University of Moscow, dermatology was taught as a readership course since 1869. Professor Mansurov M.P. was the first head of the independent Department of Skin and Venereal Diseases (1884). From 1892 to 1910, the department was headed by Professor Pospelov A.I., who is considered the actual founder of the Moscow School of Dermatology. In the future, a number of other members of the Moscow School of Dermatology made a significant contribution to the development of dermatology, in particular Meshchersky G.I. (described acute erythema centrifugum, early kidney syphilis), Grigoriev P.S. (conducted in-depth

Pospelov A.I.
(1846-1916 yrs.)

studies of experimental syphilis, published a textbook on skin and venereal diseases), Rakhmanov V.O. (developed methods for prevention of occupational skin diseases), etc.

Founded in Moscow in 1921, Central Research Institute of Skin and Venereal Diseases was considered the leading specialized scientific, methodological and organizational center of dermatology in the Soviet Union. This institute featured the country's leading dermatovenereologists. The list includes Demyanovich M.P. (developed an original method for rapid treatment of scabies), Mashkilleyson L.N. (contributed to the study of dermatophytes, described the symptoms of «hidden» peeling in guttate parapsoriasis), Smelov M.S. (described primary reticulosis of skin), Arievich A.M. (made a significant contribution to development of mycology), Sheklakov N.D. (deeply researched cystic dermatosis), Ovchinnikov N.M. (developed the questions of laboratory diagnosis of sexually transmitted diseases), as well as Studnitsyn A.A., Turanov N.M., Mordovtsev V.N., Kalamkaryan A.A., Delektorskiy V.V., Bednova V.N., Borisenko K.K., Skripkin Y.K. etc.

The Ukrainian Dermatological School is also notable. Being developed in close connection with Russian, and eventually general Soviet dermatological school, it made a significant contribution to development of scientific and practical dermatology.



Ludwig Kazimirovich Goretsky
(1826-1885 yrs.)

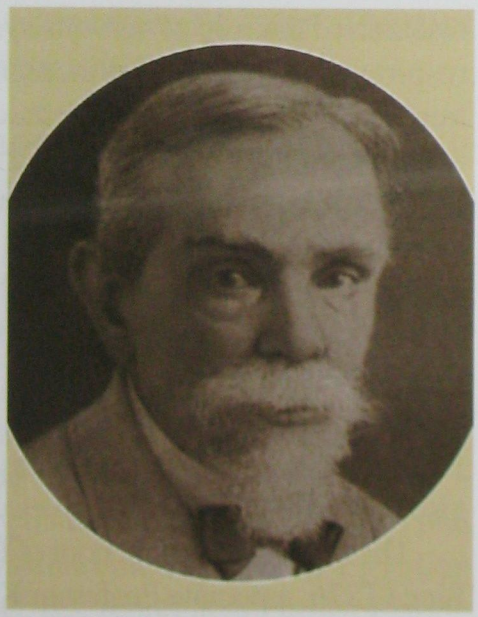
In 1863, the establishment of the Charter of the Russian Universities facilitated evolution of academical medical science, including scientific dermatology. In 1863, the academic specialized clinic of dermatology was formed on the basis of the Kiev Military Hospital. Goretskiy L.K., the assistant professor of the Department of Special Treatment of the Kiev University of St. Vladimir, became a head of the clinic and curator of the skin disease course. Headed by Goretskiy L.K., the institution was virtually the first clinic of dermatology in the Russian Empire. Headed by Professor Gyubbenet Kh.Y., syphilitic clinic was a part of surgical clinic. In 1864, associate professor Goretskiy L.K. gave a course of lectures in skin diseases to medical students. Students of the 9th and 10th

semesters attended classes twice a week. Since 1873, Goretskiy L.K. was asked to teach not only the course of skin diseases, but also give lectures on syphilis. He was able to properly combine theoretical and practical training of students. At the end of the 60ies of the XIX century, Professor Polotebnov A.G. wrote that of all medical departments of Russian universities the University of Kiev was the only institution to provide systematic teaching of skin diseases.

In 1884, the readership course of skin and syphilitic diseases was redeveloped into a separate department. Within the period from 1884 to 1897, Professor Stukovenkov M.I. became the first head of the department. He is justly considered the founder of the Ukrainian (Kiev) School of Dermatology. The school created by Stukovenkov M.I. stood for a patient's comprehensive examination, rather than just studying of visible manifestations of a skin disease. Thus, the members were to reveal the inner connection of dermatoses with the general body condition, as well as detect the causes of the lesions. Stukovenkov M.I. devoted special attention to studying of nervous system of patients with skin diseases. First in the Russian Empire, he acquainted physicians with foliaceous pemphigus, mycosis fungoides, rhinoscleroma and idiopathic Kaposi's sarcoma in his publications. His original researches of treatment of syphilis with mercurial preparations are of particular significance. Stukovenkov M.I. provided the world's first scientific proof of therapeutic doses of mercury drugs in treatment of syphilis infection.

Stukovenkov M.I.
(1842-1897 yrs.)

The best representatives of the national school of dermatovenerologists established by Stukovenkov M.I. became well known scientists and headed the specialized departments of medical faculties in some universities, in particular Nikolsky P.V. (Warsaw, Rostov-on-Don), Borovsky V.K. (Kiev), Zelenev I.F. (Kharkiv), and others. Professor Nikolsky P.V. became world famous thanks to the fact he was the first to describe the symptom of exfoliation in pemphigus verus, which entered into the world diagnostic practice under his name. In turn, Nikolsky P.V.



Nikolsky P.V.
(1858-1940 yrs.)

established a scientific school that created future professors Glukhenky T.T., Kozhevnikov P.V., Lavrov A.P., Shiryaev F.I., etc.

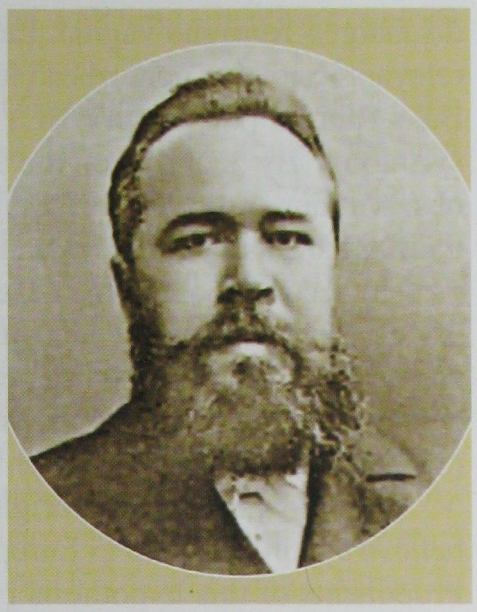
Within the period from 1898 to 1916, professor Tomaszewski S.P. replaced Stukovenkov M.I. as a head of the university department. He lobbied for women's higher medical education and became one of the organizers of the Kiev Medical Women's Institute, founded in 1916. In 1900, Tomaszewski S.P. established the Scientific Association of Dermatology and Venereology of Kiev.

In the future, chairholders and employees of the science department of the University of Kiev kept with the best traditions to enrich national and global Dermatovenereology with their scientific achievements. So, Professor Terebinsky V.I. studied the histopathology of skin and ichthyosis, worked on the clinical diagnosis of syphilis; Professor Borovsky V.K. improved treatment and prevention of syphilis, Professor Tyzhnenko A.M. actively prepared scientific and pedagogical staff, researched skin cancer, red and tubercular lupus, hyperkeratosis. Associate professor Bogdanovich S.N. was a talented clinician and gifted diagnostician of skin diseases. He developed and introduced the expurgatory mode of treatment of skin allergic diseases into clinical practice, worked out diagnostics of skin tumors. Scientific and practical developments and achievements of Professor Kuznets M.M. (studied the histological changes in skin aging, malnutrition, vitamin deficiency) and Professor Kartamyshev A.I. (introduced additional diagnostic test for the diagnosis of pemphigus – to determine the level of chloride in the urine, actively prepared scientific and pedagogical staff, created a tutorial «Skin and Venereal Diseases», which has been repeatedly published and republished in Russian, Ukrainian and Chinese) are also especially noteworthy.

Professor Pototsky I.I. is considered an opinion leader in medical-scientific schools of Ukraine and former Soviet Union. His works are devoted to a deep research of the role of nervous system in development of some chronic dermatoses, respiratory function of skin, skin lesions in leukemia, as well as improvement of treatment of syphilis. His methods of zonal ultraviolet skin irradiation in psoriasis and oxygen therapy in a number of dermatoses are well known. He trained a large group of talented scientists, most of whom were heads of departments of dermatology in various medical schools or heads of departments in professional research institutes, in particular Kolyadenko V.G. (Kiev), Lyashenko I.N. (Vinnitsa), Levkovsky N.M. (Kiev), Tsirkunov L.P. (Kiev), Khasabov L.M. (Rostov-on-Don), Grebennikov V.A. (Rostov-on-Don), Rodin A.Y. (Simferopol), Karagezyan M.A. (Krasnodar), Tseraidis G.S. (Kharkiv) and others. Professors Karysheva K.O., Kalantaevskaya K.A., Glukhenky B.T., Kalyuzhnaya L.D. are also well-known representatives of the school of Dermatovenereology of Kiev.

The dermatological school of Kharkov is considered a strong Ukrainian institution. Since 1876, associate Professor Kuznetsov A.K. lectured the course of skin diseases, while Professor of surgery Zarubin I.K. lectured a course of syphilology to students of the medicine faculty of Kharkov. An independent chair of skin and venereal diseases

was founded in Kharkov in 1885. Until 1897, this department was headed by Professor Brunev O.Y. From 1897 to 1911, the position was given to Professor Zelenev I.F., the founder and editor of the «Russian Journal of Skin and Venereal Diseases», published from 1901 to 1918 in Kharkov, and then in Moscow. Renowned scientists that headed the Department of Skin and Venereal Diseases at the Kharkov Medical Institute included Professor Sobolev L.A., Professor Popov I.S. (developed a microtest-tissue method to research mushrooms, described new clinical forms of pyodermas). In addition, Popov I.S. prepared a number of talented scientists and organizers of dermatovenereological field, among which professors Zadorozhny B.Y. (conducted fundamental research of psoriasis and several other severe dermatoses) and Pyatikop A.I. are to be mentioned.



Zelenev I.F.
(1860-1918 yrs.)

An important role in development of the Ukrainian School of Dermatovenereology belongs to the first Ukrainian Scientific Research Institute of Dermatovenerology, established in 1924 in Kharkov. This institute was headed by famous dermatovenereologists: professors Fedorovsky A.N., Krichevsky A.M., Pyatikop A.I., Mavrov I.I.

From 1961 to 1976, Professor Torsuev N.A., the Head of the Department of Skin and Venereal Diseases of the Medical Institute of Donetsk was considered an outstanding representative of the Ukrainian dermatological school. He implemented systematization of occupational skin diseases. A comprehensive study of leprosy conducted by Torsuev N.A. received worldwide recognition.

Venerology: Stages of Development

Venereology is the science of sexually transmitted diseases (diseases transmitted primarily through sexual contact). These infectious diseases feature specific pathogens. Therefore, venereology studies the epidemiology, pathogenesis, clinical manifestations, diagnosis, treatment and prevention of related infections.

The data about sexually transmitted diseases (diseases transmitted mainly through sex) came down to us from ancient times. However, until the 30s of the XIX century the understanding of these diseases was fairly simplistic.

The first stage in development of venereology dates back to ancient times and continued until the end of the XV century. Historical monuments of Egyptian medicine, particularly the Ebers Papyrus (1700 BC. E.), report about inflammation of the urinary canal (urethra). Hippocrates (V century BC) provided a sufficiently detailed clinical picture of inflammation of the urethra. He also described a «white

discharge» from the vagina. Later, Galen (II century AD) proposed qualifying relevant lesions as gonorrhea (*γονοζ* – seed, *ρεω* – flow). Arabic physician Abu-Bakr-Rhazes (IX century BC) proposed to treat urethritis with lead solution syringing. By the end of the XV century, scientists accumulated numerous observations of these diseases, in particular diagnosed their infectiousness and transmission, as well as and identified the means of prevention.

The second phase of the development of venereology belongs to the end of the XV century, when Europe faced a pandemic of a previously unknown disease, which was first called *«lues»* (punishment). In the future, the disease received the names of some saints (St. Jorgen's disease), as well as that of the countries it came from (Spanish, French, German, Polish disease). Later, the disease was called «syphilis.»

Until now, the issue of origin of syphilis remains debatable. Some scientists believe syphilis existed in Europe, Asia and the Middle East since ancient times. Proponents of this hypothesis point out that excavations of Neolithic burials at various places of Eurasia revealed bones of people with signs of past specific syphilitic gummatous osteomyelitis and gummatous osteoperiostitis. In addition, to prove this hypothesis they reference to the ancient Chinese, Indian and Egyptian treatises that provide descriptions of diseases in the genital area, anus, mouth and nose, as well as bones, which can be considered as manifestations of syphilis. We also know that Hippocrates (V c. BC. E.), Celsus (I c. BC. E.) and Galen (II c. BC. E.) described condylomatous rash on the genitals in combination with diseases of the larynx and bone, which are also possible manifestations of syphilis. In his treatises (X-XI centuries, BC. E.), Avicenna described diseases similar to the clinical manifestations of syphilis.

Ex another hypothesis by some modern scholars, syphilis first appeared in Africa. They suggest that pathogens of tropical treponematoses, including frambesia, carate, bejel, as well as and the causative agent of syphilis represent different versions of once common primary treponema. The early Neolithic age is considered the period when the disease of human treponematosis emerged. In addition, there is further evidence for this hypothesis: there is observed some morphological and biological identity of Treponema pallidum (the causative agent of syphilis) and treponemes that cause tropical treponematosis with a similar clinical course of these infections and cross-immunity.

However, the hypothesis of American origin of syphilis features the greatest number of adherents. It is indicated that the disease was brought to Europe by the sailors from the expedition of Christopher Columbus, after the discovery of America. Sailors were infected from the natives of Haiti, where the disease was known long before the arrival of Europeans. It is important to note that authoritative domestic epidemiologist Gromashevskiy L.V. also supported the hypothesis of an American origin of syphilis and suggested that the disease arose as a result of sexual spirochetosis of South American llamas and moved to the local people as a result of bestiality. In further, the locals infected the sailors of Columbus. The first data on the

disease appeared in Spain (1494), then – in Italy, and with time – in other countries of Europe and Asia.

In 1530, Italian physician, philosopher and poet Girolamo Fracastoro named the disease as syphilis, after shepherd Syphilus (from *sys* – pig, *philos* – friend), the hero of the poem «Syphilis, or the Gallic disease.» The essence of the disease remained unknown. Some doctors referred formerly known gonorrhea to syphilis. With the progress in microbiology, a number of pathogens of sexually transmitted diseases were identified. This marked the beginning of the third stage in development of venereology. In 1836, Donne A. first discovered trichomonads in a woman's vagina. Nearly a century after that, they were considered harmless inhabitants of the vagina. Though, in 1870 Kharkov scientist Lazarevich I. wrote that the presence of trichomonads in vagina is accompanied by inflammatory manifestations and foamy discharge. In 1879, German scientist Neisser A. discovered the causative agent of gonorrhoeae – gonococcus. In 1905, Schaudinn F. and Hoffmann E. found a specific causative agent of syphilis – Treponema Pallidum.

In later years, it was found that the so-called abacterial urethrides can be caused by viral, chlamydia, mycoplasma and ureaplasma infections. Implementation of methods of serodiagnosis was a significant event in the development of syphilology. In 1906, *Wassermann, Neisser, Bruck* suggested a complement fixation test, which gradually improved. In the future, specific reactions for serological diagnosis of syphilis were proposed: treponema pallidum immobilization test (R. Nelsen, M. Mayer, 1949), immunofluorescence test (Weller, Coons, 1954). At the present stage, the method of diagnosis of syphilis using enzyme multiplied immunoassay is applied.

To speed up the diagnosis of syphilis, flocculation tests by Kahn, Sachs-Witebsky and indicative rapid test method of microreaction on glass were offered. In parallel, considerable attention was paid to the further study of the causative agent of syphilis, its ultra structure and interaction with cells of the patient, variability of L-forms of Treponema pallidum; the possibility of persistence of Treponema in poly-membranous phagosomes of cells was established (Ovchinnikov N.M., Delektorsky V.V., 1965).

Throughout the history of syphilology, researchers focused on a comprehensive study of clinical manifestations of the disease.

Fundamental studies of Ricord Ph., Fournier A., Tarnowski V.M., Grigoriev P.S., Astvatsaturov K.S. and many other scientists refined various features of manifestations of syphilis infection and improved quality of the disease clinical diagnosis. In recent decades, the study of clinical picture of syphilis by scientists from research institutes of dermatovenerology, centers and departments of dermatology in several countries, including the Russian Federation and Ukraine, revealed some features of syphilis symptoms (oligosymptomatic forms, prolonged latent time of infection after introduction of infection, etc.). Syphilologists always focused on the problem of innate and latent syphilis.

Doctors paid special attention to treatment of syphilis. At the end of the XV – beginning of the XVI century, right after Europe faced pandemic syphilis, the studies

of methods and treatments that have been used in medicine at that time (diuretic, laxative, diaphoretic drugs, bleeding, etc.) were started, but they failed to be efficient.

As evidenced by numerous domestic and foreign medical chronicles, mercurial were one of the oldest antiluetic therapeutic agents. Since the end of the XV century, rubbing mercury ointment was the most common method of syphilis treatment used in Europe...

In 1894, the founder of the Ukrainian (Kiev) Research School of Dermatovenereologists Prof. Stukovenkov M.I. was the first in the world to scientifically prove the methods of treating syphilis with mercurials and clearly defined therapeutic daily doses through a proposed protein-based methodology for identification of mercury in biological fluids.

In 1909, an entirely new approach to treatment of syphilis appeared. German physician and researcher P. Ehrlich proposed to treat the disease with a triad arsenic drug – Salvarsan. In 1912, an improved product based on arsenic was synthesized – *Neosalvarsan*. Given a sufficiently high therapeutic effect, Neosalvarsan was used in syphilological practice for decades.

In 1921, Levaditi K. and Sazerac Z. offered to introduce bismuth compounds into syphilological practice. Biiochinolum, Bismoverolum, Pentabismolum were often used for intramuscular administration. From the 30ies of the XX century, the treatment of syphilis widely used iodine drugs (potassium iodide, sodium iodide, Lugol's solution). Iodine was administered orally mainly in advanced syphilis, as well as for trial treatment with diagnostic purpose to differentiate syphilitic inflammations in visceral organs from those non-specific.

With the 40ies of the XX century, a new era in treatment of syphilis began, after introduction of penicillin in medical practice. In terms of antibacterial effect, this antibiotic exceeded all known at the time antiluetic medicines. Until the 60ies of the XX century, the method of syphilis treatment included the use of intermittent scheme (combined prescription of penicillin antibiotics and bismuth drugs). Later, a continuous (permanent) scheme of treatment with penicillin group of drugs and no bismuth drugs was used.

Basic modern medicines for etiotropic treatment of syphilis are presented by penicillin and its repository drugs. Tetracyclines, macrolides and cephalosporins are reserve drugs of etiotropic treatment of this infection.

In recent decades, methods of treatment of chlamydia, mycoplasma, ureaplasma, herpes and other urogenital infections are developed and improved. Studies are still in progress.

Development of Venereology is closely related to the control of infections that are primarily transmitted through sexual contact. Practical and scientific solution to the problem was significantly contributed by Stukovenkov M.I., Zabolotny D.K., Mechnikov I.I., Tarnowski V.G., Tomaszewski S.P., Zelenev I.F., Polotebnov A.G., Pospelov A.I., Rakhmanov V.A., Grigoriev P.S., Tyzhnenko A.M., Torsuev N.A., Pototski I.I., Turanov N.M., Ovchinnikov N.M., Porudominsky I.M., Ilyin I.I., Mavrov I.I. and others.

GENERAL ISSUES ON DERMATOLOGY AND VENEREOLOGY

- Morphology, physiology and pathomorphology of skin and its appendages
- Introduction to Dermatology and Venereology
- Principles of Treatment applied in Dermatology and Venereology

Morphology, physiology and pathomorphology of skin and its appendages

Introduction to Dermatology and venereology

Principles of Treatment applied in Dermatology and Venereology

2 TOPIC

TRAINING AND EDUCATIONAL PURPOSES

- To explain physiological and morphological characteristics of skin, its appendages and mucous membranes in normal functioning of the body and in various disease states;
- To disclose the concept of «rash» and its role in setting appropriate dermatologic diagnosis;
- To know basic common and special methods of examination and principles of treatment applied in Dermatology and Venereology.

2.1 Morphology, Physiology and Pathomorphology of Skin, its Appendages and Oral Mucosa

TO KNOW:

- The anatomy of skin, its appendages and oral mucosa;
- Histological structure of skin, its appendages and oral mucosa;
- The nature of functional properties of skin, their age and sex characteristics;
- The function of cells, tissues, organs and physiological systems in health and in various disease states;
- Histopathological changes in the skin and its appendages and oral mucosa of mouth in health or in various pathological conditions.

TO BE ABLE TO:

- Distinguish between the structure of different layers of epidermis, dermis, subcutaneous fat and oral mucosa;
- Distinguish between the histological structures of skin appendages;
- Recognize different types of histopathological skin changes.

Skin (*cutis*) covers the entire surface of the body and transfers to the mucous membrane in the areas of natural openings i.e. mouth, nose, urinary tract and anus. The total area of the skin is 1.5 m^2. There are many folds, depressions, elevations on the surface of the skin. The skin is striated with furrows of different texture, which divide the surface into a number of fields, mostly triangular or diamond-shaped. Rough skin furrows include facial wrinkles, folds of the palms, scrotal folds and furrows on the extensor surfaces of the joints.

The color of the skin is defined as flesh-colored. It includes colors of all tissues that compose the skin and mainly depends on the color of blood in the capillary vessels and skin pigment. Healthy skin is dull in appearance.

Morphology of skin and its appendages

The skin consists of three layers i.e. the epidermis, dermis and subcutaneous tissue (hypodermis). These layers differ in embryonic origin: the epidermis is derived from ectoderm, while dermis and hypodermis – from mesoderm.

Important anatomical constituents of skin include its appendages, which consist of sweat, sebaceous glands, hair and nails.

Epidermis. Histologically epidermis (*epidermis*) is presented by keratinized stratified squamous epithelium. There are five layers of cells separated from the dermis by basement membrane. Directly on the membrane there is a layer of cylindrical cells located palisade and perpendicular to it. This layer is called the basal or primary (*stratum basale*), as well as malpighian (*germinal*), as this is the place, where mitotic cell division that ensures epidermis regeneration takes place.

Above the basal layer there is a ribbed layer (*stratum spinosum*) containing several rows of polygonal cells that are becoming flatter progressively as they are approaching the next layer.

Granular layer (*stratum granulosum*) is represented by one or two (sometimes four) rows of elongated spindle-shaped cells located along the surface of the skin. The cell nuclei are poor in chromatin and therefore are bright. The protoplasm contains keratohyalin grains, keratin precursor protein grains and grains of main keroid of skin and its appendages.

These layers of the epidermis are sometimes combined under the name of Malpighian layer.

Translucent layer (*stratum lucidum*) is located above the granular layer and consists of one or two rows of flat non-nuclear cells. Protoplasm of these cells contains a protein eleidin, which is an intermedium in the formation of keratin.

Horny layer (*stratum corneum*) is the most superficial layer of the epidermis consisting of flat thin horny plates that lie on the top of each other in several rows. Horny plates are completely dead cells that have lost their nuclei. Keratinization is achieved by substituting the protoplasm with keratin protein. The thickness of the horny layer in different parts of the skin varies greatly. Maximum thickness of the horny layer is on the palms and planta. On the surface of this layer, horny plates are

less dense and exfoliate gradually. Their gradual exfoliation occurs constantly and is called physiological desquamation.

The epidermis enters dermis with more or less developed processes called dermal ridges. Derma enters the space between the crests of the epidermis with projections, which are called dermal papillae.

Dermis. Under the epidermis there is the second layer of the skin called the actual skin or dermis (*derma*). It is rich for connective tissue fibers, which form bundles interwoven in different directions. There are rather few cells in the connective tissue of the dermis (fibroblasts, fibroclasts, melanocytes, macrophages, mast cells, mesenchymal cells).

There are three types of connective tissue fibers, which include collagen, elastic and argentophilic fibers. The gaps between fiber bundles are filled with the main amorphous substance, which plays an important role in metabolism and in the protective functions of the skin.

Argentophilic fibers form the basement membrane at the boundary between the epidermis and dermis, cover sebaceous and sweat glands, hair follicles and skin muscles with fine reticulum. By weaving in different directions, collagen and elastic fibers continuously distribute in the dermis, thereby dividing dermis into two layers – papillary and reticular.

Papillary dermis is located immediately below the epidermis. The bundles of connective tissue fibers in the papillary layer are quite thin and interweave in different directions. Many bundles are perpendicular to the skin surface and enter the papillae.

Reticular layer of the dermis consists of thicker fiber bundles, which when combined, form a dense reticulum. Much of these bundles are parallel to the skin surface. Such structure of the dermis ensures its great strength and elasticity.

The thickness of dermis in different areas of the skin varies from 0.5 to 4 mm. Dermis with no clear boundaries moves into the subcutaneous fat layer (hypodermis).

Hypodermis. The subcutaneous fat layer (subcutaneous adipose tissue, hypodermis) (*hypoderma*) also consists of bundles of interwoven connective tissue fibers. These bundles are the continuation of connective tissue bundles of the dermis; they are loose and form glomerular reticulum (*retinaculum cutis*). The nests of these reticulum contain fat lobules i.e. accumulation of fat cells. Subcutaneous fat layer plays an important role in fat metabolism being one of the most important depots of fat in the body. The thickness of hypodermis in different parts of the body is not the same, thus it is more significant in the abdomen, thighs and buttocks.

Dermis and hypodermis are scattered with different cells, which in various stages of differentiation, are divided into connective tissue cells and white blood cells.

Dermis and hypodermis include skin glands, hair, as well as blood vessels, nerves and muscles.

Blood and lymphatic vessels in the skin. The skin has a well-developed system of blood vessels. Blood vessels in the skin can make up to 1/5 of the total human body blood mass. In the process of circulation in the body, that is regulated by central nervous system, the skin acts as one of the major depots.

Arterial trunks penetrate into the subcutaneous fat layers from deeper located tissues. Here, they give branches that feed the hypodermis and at the border of dermis form arterial plexus, which is called deep dermal spider veins. From deep dermal spider veins there are vessels that rise up into the dermis. From these vessels and deep dermal spider veins there are arterial branches feeding dermis, sweat and sebaceous glands, hair, muscles and nerves. On the border of the papillary and reticular layers there is the second arterial plexus called superficial skin spider vein. Therefrom arteriole goes into every single papilla. Terminal arterial branches are divided into skin capillaries, which gradually merge with each other and give rise to the skin veins. Venous skin vessels run parallel to the blood vessels.

The lymphatic system of the skin begins with intercellular gaps of epidermis and numerous lymph slots of dermis. Lymphatic vessels are located along blood vessels. Lymphatic vessels, like blood vessels, form superficial and deep spider veins. Skin blood vessels can quickly change their clearance, which means that they can expand or narrow reflectory under the influence of stimulation of nerve endings, which can be caused by the action of heat, cold, mechanical action (friction, hit) and chemicals. Reflectory expansion or narrowing of blood vessels can also occur due to a variety of neuropsychiatric emotions i.e. joy, fear, anger etc.

Nervous system of the skin. Nerves form the main plexus in the subcutaneous layer, wherefrom numerous trunks, which give rise to new plexus, go to derma. Particularly dense plexus is formed in the papillary layer. Nerve fibers extending therefrom, give rise to numerous nerve endings in the connective tissue and in the epidermis, thus making the skin sensitive. In the subcutaneous fat layer there are Pacinian and Ruffini's corpuscles; in the papillary dermis there are Meissner's, Golgi-Mazzoni's corpuscles and Krause's bulbs and in the epidermis there are Merkel's menisci.

In addition to the sensory nerves there are secretory glands and nerve plexuses in the skin, which are located along the vessels.

Pacinian corpuscle (Lamellar corpuscles) is a complex encapsulated nerve receptor. It consists of the processes of altered cells of ciliated epithelium with sensory cilia, which are in contact with cell membrane of nerve process end. Cytosomes are separated from contact zone by the capsule consisting of several longitudinally oriented glial cells. Sensory cells cilia are located between external and internal capsule, thus contacting with the inner surface of outer capsule.

Pacinian corpuscle acts as:

- mechanoreceptor (due to a change of curvature of the outer surface of the capsule, mechanical effect is transmitted to the sensory cells cilia that generate nerve impulses);
- chemoreceptor (via sulcate channel that is present in the area of corpuscle pole different substances penetrate into the space between the inner and outer capsules, thus inducing nerve impulses);
- baroreceptor (change of blood pressure in the network of blood capillaries in the space between the inner and outer capsule alters the state of the sensory cells, thus inducing nerve impulses).

Pacinian corpuscles have a large receptive field, i.e. represent a rough sensitivity.

Meissner's corpuscle (tactile corpuscles) is a receptor, which is an encapsulated nerve ending present in the skin dermis, most often on the tips of fingers, soles, nipples, eyelids, lips and genitals. It is round. In its center there is a gyrate basket of myelin fiber, which passes through the transverse oval cells resembling Schwann's cells of nervous membrane. From the outside the corpuscle is covered with a connective tissue capsule.

Meissner's and Pacinian corpuscles belong to receptors that are rapidly adapting, i.e. they fix the skin pressure force.

Ruffini's corpuscle is a spindle-shaped receptor containing the inner bulb with a dense network of branched nerve cells and supportive lamellocytes. From the outside the corpuscle is covered with a connective tissue capsule consisting of several layers of flattened fibroblasts. Between the inner bulb and the capsule there is a capsule space filled with liquid. Ruffini's corpuscles are skin stretching receptors that are slow to adapt. There is an assumption that they are also heat thermoreceptors.

Golgi-Mazzoni's corpuscles are thick myelin fibers, «wrapped» around groups of collagen tendinous fibers and surrounded by a connective tissue capsule. Likewise Ruffini's corpuscles, they react to the tension, but their sensitivity threshold is higher.

Tactile Merkel's meniscus (disk) is a set of Merkel's cell with nerve ending. Tactile Merkel's cells are round or elongated cells, which are located among the epithelial cells and are larger than the latter. These cells are connected to the epithelial cells by desmosomes and form a contact with reticulated branched nerve endings.

Merkel's menisci are slow to adapt (to fix the duration of touch) and have small receptive fields, i.e. fine sensitivity.

Krause's bulbs are encapsulated nerve endings, which are composed of terminal branches of sensitive nerve fiber, inner glial bulb and outer connective tissue capsule. They are located in the connective tissue of mucous membranes and in the dermis, mainly on the hairless areas. They are considered to be cold thermoreceptors.

Hair. Hair *(pili)* is divided into: 1) long (head hair, beards, mustaches, armpits hair, hair in the area of external genital organs); 2) setaceous (eyebrows, eyelashes, hair in nose nostrils, in the external ear canal); 3) vellus (in all areas of the skin except for the so-called hair-free sites, in particular on the palms, soles, vermilion zone, nipples, breasts, labia, balanus and the inner layer of the foreskin).

The hair consists of the area freely located over the skin i.e. the hair shaft *(scapus)* and the area hidden in the skin i.e. the hair root *(radix)*. The root ends with extended part, which includes hair follicle, wherefrom the hair grows. From the connective tissue of the dermis the hair follicle is penetrated by dermal papilla, which contains blood vessels that feed the follicle.

The hair shaft consists of three layers: the medulla, cortex and cuticle.

The medulla is a hair marrow and consists of keratinized polygonal cells. There is no hair marrow in vellus.

The cortex is composed of extended cells with elongated nucleus or its fragments. These cells contain pigment melanin that defines the color of the hair. In gray hair pigment is absent, while silver color is achieved by air bubbles that appear in the cortex.

The cuticle is the outer layer of the hair represented by plane dead cells, which are arranged in a single layer leaning on one another like shingles.

Hair root is located in the hair follicle *(folliculus pili)*, which opens as a small hole in the skin *(ostium)*. At the boundary of the inner and middle thirds of the hair the hair follicle is entered by excretory duct of sebaceous gland.

Hair follicle is composed of connective tissue and epithelial parts. Starting from the confluence of sebaceous glands duct the connective tissue part is the most developed in the lower part of the root. Epithelial portion of the hair follicle represents an invagination of the epidermis. From the skin to the mouth of the excretory ducts of sebaceous glands (so called hair funnel – *infundibulum*) one may clearly see all the layers of the epidermis. Then, horny layer disappears, and funnel epithelium goes into outer root sheath epithelium, which is composed of cells similar to the cells of the basal and spinous layers. From the mouth of sebaceous glands duct and lower between the inner sheath and the hair cuticle there is the inner root sheath. There are three layers of inner root sheath: the inner – the inner root sheath cuticle (one row of dead skin cells), medium – Huxley's layer (one to three rows of semi-dead cells with pyknotic nuclei or completely devoid of them) and external – a layer of Henle (one row of dead skin cells). In the course of desquamation, the cells of all three layers mix with sebum near the mouth of the sebaceous gland.

All elements of the component parts of the hair and of the inner root sheath directed to the hair follicle have the cores and at the follicle they blend into the germinal zone of increased cell division, wherefrom the hair grows.

Hair life span is from several months to 4 years.

Skin muscles. Hair is associated with muscular system *(musculi arrectores pilorum)*, which consists of a strip-smooth muscles, one end of which is attached

via short tendon to the reticular layer of the dermis, and the other end – to the outer root sheath of hair just below the confluence of sebaceous glands duct. When contracting, muscles raise hair (the effect of the so-called «goose bumps») and, thus squeezing the sebaceous gland, induce the release of their secret.

Striated muscles are present only in the face skin *(musculi faciales)*. They are called mimic, as their contraction makes face movable and expressive and displays changes in mental state of a person.

Sebaceous glands. Sebaceous glands *(glandulae sebaceae)* are alveolar glands. They are predominantly open in hair follicles. A small number of sebaceous glands open directly on the surface of the skin i.e. on the balanus, the foreskin, on the labia lips, nipples and lips.

By secretion type, sebaceous glands are holocrine, which means that secretion is associated with destruction of adenocytes. In secretory regions, adenocytes are arranged in several rows. External cells make up a germ layer where mitosis takes place and deeper rows' cells accumulating fat droplets are formed. Cell maturation is accompanied by filling of the cell with large drops of fat, by pyknotic changes in the nucleus, which disappears with time, thus resulting in the destruction of the whole cell. Cell fragments, mixed with fat, fill in the gland and secrete via ducts to the skin surface.

Sweat glands. In terms of structure, sweat glands *(glandulae sudoriferae)* are simple tubular glands, which consist of a long ductless and secretory region, twisted into a ball, located deep in the reticular dermis at the border with subcutaneous fat layer. Terminal sections are lined with cuboidal epithelium, followed by a series of longitudinally arranged contractile cells (myoepithelial cells) which lie on the basement membrane. Duct in the dermis is straight and is lined with two rows of cells, and within the epidermis, it is corkscrew and is an extension of the intercellular spaces of the epidermis.

By the type of secretion sweat glands are divided into eccrine (merocrine), in which secretion occurs without destruction of adenocytes, and apocrine, where secretion is accompanied by destruction of the apical parts of adenocytes. Apocrine glands are bigger than eccrine ones, have less tightly curled ball and tend to appear in the hair funnel.

Eccrine glands are evenly arranged across the whole surface of the skin (to exclude vermilion zone, balanus and the inner layer of foreskin). They secrete lubrication for horny layer, are involved in thermoregulation and in the selection of products of nitrogen metabolism.

Apocrine glands are found primarily in the armpit, around the anus, on pubis and abdomen skin, below omphalus and on the labia lips. They develop at puberty. The secret of apocrine sweat gland has a specific smell and contains sex attractants (pheromones).

Nails. Nails *(unguis)* are dense horny quadrate plates located on the back surface of distal phalanges of fingers and toes. They lie on the so-called nail bed.

Their purpose is to protect terminal phalanges from damage. The front edge of the nail plate is free, and its rear and side edges are surrounded by skin fold and go deep into it. The upper part of the skin fold comes over the nail plate, thus nail folds (rear and side) are formed. The nail has its body *(corpus unguis)* and the root *(radix unguis)*. Nail root is the posterior part of the nail plate, which lies deep in the folds of skin at the back of the rear nail fold. Only a small part of the root of nail protrudes from nail fold in the form of white semilunar area (lunula – *lunula unguis*). It is better seen on the thumbnails. The lunula is covered with thin horn rim i.e. nail skin *(eponychion)*, which is a continuation of the rear nail fold. Nail root is located on the back of the nail bed, which is called matrix *(matrix)*. Matrix is a place where the nail plate is formed. It consists of epithelial cells in character resembling the cells of basal and spinous layers of epidermis. Spinous layer contains onychoblasts i.e. cells that form the nail and which turn into horny nail plates. Nail plate itself corresponds to translucent and horny layers of the epidermis. Nails grow slower than the hair. In an average, fingernails grow by 1 mm per week, while feet nails by 0.25 mm per week.

Skin Functions

The state of the skin depends on a number of functions it performs, namely protective, immune, melanin-forming, thermoregulatory, secretory, excretory, metabolic, receptor, sorption, respiratory, repository etc.

Protective function of the skin. Skin is a barrier on the way of various environmental agents that affect the body i.e. mechanical, physical, chemical and biological.

Mechanical impact is prevented by epithelium density, the elasticity of fibrous structures of the dermis and shock-absorbing properties of subcutaneous fat layer. The reduction of any of these components can cause sharp weakening of protective properties of the skin. For example, atrophy of intercellular processes in spinous layer of the epidermis, which is typical of acantholytic pemphigus, results in an extreme vulnerability of epidermis.

The presence of natural sebum (sebaceous matter) on the surface protects skin against drying out and the impact of a number of chemical substances. Only the impact of depleting (strong acids, alkalis and some keratolytics) and fat-soluble substances can weaken protective features of the horny layer.

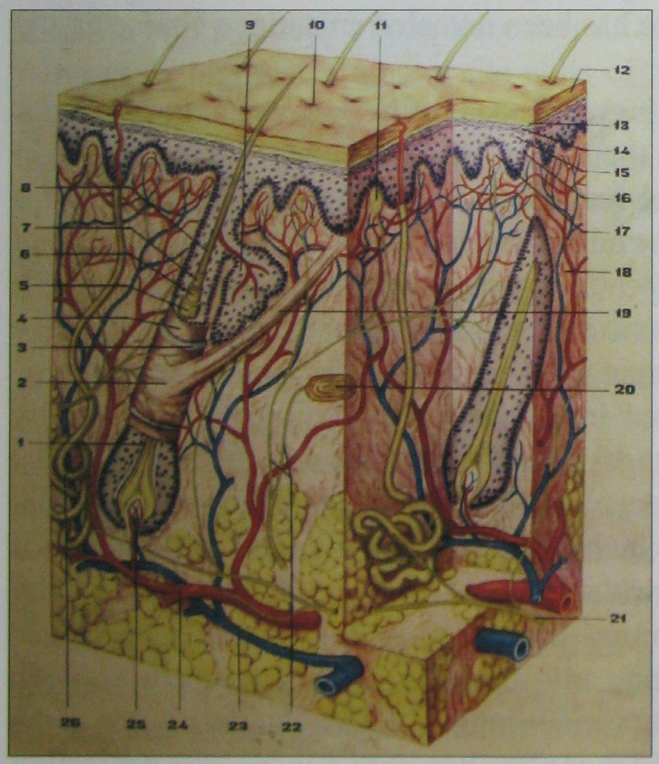
Undamaged epidermis is a poor conductor of electric current. When moistening and reducing fat body substances, skin resistance to electric current greatly decreases.

The protective function of the skin as concerns light is expressed in the action of pigment (melanin) skin system.

Immune function of the skin. The skin plays an important role in the immune system. The main elements of the immune system of the skin are keratinocytes, Langerhans' cells, epidermal T-lymphocytes. Keratinocytes favor the maturation of

T-lymphocytes by impacting thereon with deoxyribonucleotidyl transferase enzyme (DRNT). The majority of human skin T-lymphocytes is in the dermis, usually around the post-capillary venules and skin appendages. They are able to recognize exogenous and endogenous antigens only after their presentation by Langerhans' cells, which are antigen presenting cells. T-cells recognize antigen only in a single structure of MHC (*Major Histocompatibility Complex*). To be recognized by T-helper lymphocytes (CD4 +) antigen should be presented in conjunction with II class MHC (HLA-DR, DP, DQ), whereas the majority of T-suppressor cells (CD8 +) recognize antigen in association with I class MHC molecules (HLA- A, B, C). In the course of immune response to exogenous or endogenous antigens Langerhans's cells involved in antigen presentation are exposed to phenotypic and functional changes, leave the epidermis and enter the lymphatic vessels of the dermis, and then migrate to lymph nodes paracortex. At this stage, Langerhans' cells represent a complex antigen i.e. MHC T-cell antigen receptor on the surface of helper or suppressor. Antigen-specific T-cell response involves the formation of T-lymphocytes blast forms, which return to the skin regions containing the antigen.

Immune disorders play a pathogenic role in various diseases of the skin, including bullous dermatosis, allergodermatosis, psoriasis, T-cell malignant lymphoma of the skin.



Healthy skin and intact mucous membranes are a barrier for most microorganisms, except for those who have a special penetration device. Previously such protective function of the skin has been explained only by mechanical factors i.e. the presence of horny layer, water-lipid mantle, high elasticity and the existence of polyunsaturated fatty acids. Today, however, there is information on the immunological activity of basic structures of the skin that ensure immune response of epidermis,

Fig. 2.1. Human skin structure:
1 – hair cuticle; 2 – connective tissue layer; 3 – vitreous membrane; 4 – outer coat; 5 – layer of Henle; 6 – Huxley's layer; 7 – cuticle; 8 – hair shaft; 9 – sebaceous gland; 10 – sweat gland duct; 11 – Meissner's corpuscle; 12 – horny layer; 13 – translucent layer; 14 – granular layer; 15 – spinous layer; 16 – basal layer; 17 – papillary layer; 18 – reticular layer; 19 – muscle lifting the hair; 20 – Pacinian corpuscle; 21 – motor nerve; 22 – sensory nerves; 23 – veins; 24 – artery; 25 – hair follicle papilla; 26 – sweat-gland.

dermis and subcutaneous fat. One had proved anatomical, molecular and functional similarity of epidermal keratinocytes with epithelial cells of the thymus. Keratinocytes produce epidermal thymocyte activating factor (ETAF), interleukins-1, 2 (T-cell growth factors), interleukin 3 (mast cells proliferation and degranulation factor), natural killers activating factor (NKAF) and epidermal factor of granulocytes activity. Besides these, keratinocytes produce a number of non-specific mediators, biologically active factors that are involved in immune and inflammatory reactions of the skin. Amongst them the most studied are fatty acid metabolites (prostaglandins, leukotrienes, fat hydroxides) and plasminogen activator and inhibitor.

Keratinocytes induce maturation of T-cells by the action of deoxynucleotidyl transferase enzyme (DRNT). Epithelial cells are able to induce the expression of this enzyme, as well as the secretion of thymopoietin in T-lymphocyte differentiation.

The similarity of keratinocytes with epithelial cells of the thymus is confirmed by common heterogenic antigens detected in the basal cells of epidermis and thymus hormone epithelium. Common morphological features of these bodies have been established in the cultivation of thymus epithelium. It was found that thymus cells, when cultured, turn into typical epidermis keratinocytes. In thymus corpuscle (Hassall's corpuscles) receptors there has been revealed an antigen characteristic for the basal layer cells of epidermis.

In dermis immunological activity is conditioned by the presence of lymphocytes around post-capillary venules of the superficial vascular plexus and skin appendages. Immunomorphological methods have found that T-lymphocytes represent 90% of all cells of the skin and are located mainly in the epidermis and papillary dermis. B-lymphocytes are located in the reticular dermis. Endothelial cells of post-capillary venules of the upper vascular plexus and macrophage system play one of the major roles in the development and formation of skin immune responses.

Macrophage system is presented in the dermis and subcutaneous fat by fibroblasts, phagocytic macrophages (histiocytes) and dendritic cells. Morphologically, differentiated histiocyte is a dendritic cell with lots of microvilli. Macrophage system of the skin also includes mast cells involved in migration of T-lymphocytes, in antigen-antibody reactions based on immediate hypersensitivity type. The implementation of immune processes in the skin also involves participation of blood cells migrating into the skin (monocytes, eosinophils, neutrophils, basophils) and performing various immunological functions, one of the basic of which is the interaction of T-lymphocytes with nonspecific protection factors.

Immune function is also given to dendritic epidermocytes representing a modified variant of tissue macrophages population. Likewise mast cells and macrophages, these cells do not have immunological specificity, but when activated by antigens or cytokines, they exhibit physiological activity by secreting biologically active substances.

Melanotic skin system. Melanin (Greek μελας – black) is a class of organic compounds found in plants, animals, protozoa and bacteria, where they mainly play the role of pigments. Chemically, these compounds are derivatives of amino acid tyrosine. The most common in humans are:

- eumelanin – brown-black polymer of dihydroxyindole, dihydroxyindolerodizonic acids and their reduced forms;
- pheomelanin – red-brown polymer of benzothiazine responsible for red hair and freckles.

The synthesis of melanin in the skin i.e. melanogenesis is stimulated by ultraviolet radiation impact on the DNA. Photochemical properties of melanin make it a good photoprotector. It absorbs harmful ultraviolet radiation and converts it into energy in a safe amount of heat in a process known as ultrafast internal conversion. Due to this property melanin absorbs 99.9% of UV light and keeps free radicals at a minimum, thus preventing DNA damage.

Melanogenesis occurs in melanocytes i.e. specialized dendritic cells. During embryonic development melanocytes precursors arise from the neural crest and actively migrate to the peripheral zones. As it has already been noted epidermis basal layer cells contain melanin pigment grains. These grains are also present in melanin pigment cells of papillary dermis and they are called melanoblasts. The number of pigment grains in the cells of the basal layer of epidermis and of melanoblasts in dermis in diverse people differs quite significantly. The more pigment inclusions, the darker the color of the skin. In people with dark skin, Asians and Afro-Americans, pigment inclusions are found in the cells of spinous and granular layers.

Thermoregulatory role of the skin. Thermoregulatory processes in the human body are controlled by central nervous system. The skin is involved in this process via nervous apparatus, blood vessels and sweat glands. Moreover, horny and especially subcutaneous fat layer are bad heat conductors both from external and internal environments. The release of heat in the body results from a number of metabolic processes, such as oxidation, muscle activity etc.

The heat is emitted to the environment via blood vessels and sweat glands.

Blood vessels in the skin react quickly to changes in ambient temperature. When ambient temperature rises, skin blood vessels extend, thereby increasing the amount of blood per time unit flowing through their lumen. This leads to an increase in skin heat emission to the environment. When the temperature goes down, the reverse phenomenon is observed. This process is done by reflex.

The body emits heat to the environment in one of the following ways: radiation, emission, convection and evaporation.

Radiation is a way the surface of the human body emits heat to the environment in the form of infrared rays (λ = 5-20 μm). The amount of heat the body radiates to the environment is proportional to the surface of radiation area and to the difference between the mean values of skin and environment

temperature. The surface radiation area is the total surface area of body parts that contact the air. At 20°C ambient temperature and 40-40% of relative humidity an adult body radiates nearly 40-40% of the total emitted heat. Elimination of heat by radiation increases with a decrease in ambient temperature and decreases with its increase. Under constant ambient temperature, body radiation increases with increase in skin temperature and decreases when it is down. If the average temperature of skin surface and environment are aligned, elimination of heat by radiation becomes impossible. It is possible to reduce elimination of heat by radiation via reduction of the surface of radiation area («winding oneself into a ball»). If the ambient temperature is higher than the average temperature of the skin, the human body is warmed up via absorption of infrared rays emitted by surrounding objects.

Emission is a way to eliminate heat when human body contacts with other physical bodies. The amount of heat eliminated by the body to the environment in this way is proportional to the difference between average temperatures of contacting bodies, to the area of contacting surfaces, to the time of heat contact and thermal conductivity of the body in contact. Dry air and adipose tissue are characterized by low thermal conductivity and, thus are considered to be thermal insulators. The use of clothing made of fabrics containing many small stationary bubbles of air between the fibers (e.g. wool fabric) enables the human body to reduce heat dissipation. Wet, water-vapor saturated air and water are characterized by high thermal conductivity. Therefore, human's staying in a high humidity environment at low temperature is accompanied by increased body heat loss. Wet clothes also lose its insulating properties.

Convection is a way the body eliminates heat by means of transferring heat via moving particles of air or water. To dissipate heat by means of convection body surface shall be airflowed at a temperature that is lower than the temperature of the skin. At that, air layer contacting with the skin warms up, decreases its density, rises and is replaced by cooler, denser air. At 20°C air temperature and 40% relative humidity adult body dissipates into the environment nearly 25% of heat by means of emission and convection (basic convection). By increasing the speed of the air flow (wind, ventilation) heat emission increases significantly as well (forced convection). The heat loss via emission, convection and radiation, which are collectively referred to as «dry» heat loss, becomes ineffective when aligning average temperatures of the body surface and the environment.

Evaporation is a way the body dissipates heat to the environment by its evaporation via sweat or evaporation of moisture from the skin and respiratory tract mucous membranes of («wet» heat loss). A man is constantly experiencing the process of sweating via skin sweat glands («perceivable» or glandular loss of water) and moistening via respiratory tract mucous membranes («unperceivable» loss of water). At that, "perceivable" loss of water impacts the total amount of heat that is given off via evaporation more dramatically than the «unperceivable» one.

Secretory skin function. Sweating is the process of sweating carried out reflexively by sweat glands. Receptors responsible for sweating reflexes are located in the skin, mucous membranes and muscles. The centers of sweating are located in the cerebral cortex of the brain, the hypothalamus, medulla and spinal cord.

The composition and volume of sweat liquid can vary within wide limits, depending on the environmental conditions. On average, at room temperature, the human body releases 400-600 ml of sweat per day. In hot weather, and as a result of heavy physical work, sweating may amount to 12 liters per day. The sweat consists of mineral metabolism products i.e. sulfuric compounds, phosphates, potassium chloride, calcium, calcium salts and protein metabolism products, which include urea, lactic acid, uric acid, ammonia and some amino acids. Also sweat contains volatile fatty acids. Sweat reaction is acidic: pH 3.8-6.2, which contributes to the bactericidal action of the skin.

Sweating plays an important role in thermoregulation of the body, water-salt metabolism and, partially, in separation of metabolic products from the body.

Skin sebaceous excretions. Sebum is the grease of the horny layer of epidermis. It softens the friction between skin folds, prevents the formation of cracks and protects skin from maceration. Sebum is a complex mixture of various lipids, comprising glycerides (40%), free fatty acids (16%), wax and cholesterol esters (25%), squalene (12%) and cholesterol. Sebum triglycerides are decomposed by bacteria to free fatty acids. Some of them form volatile fatty acids, which give the skin a certain smell. Sebum itself is odorless.

On the skin surface fat is a part of the so-called protective mantle – the layer formed by the skin glands secret, its microflora and exfoliated horny scales. Due to the presence of acidic medium on the skin surface (pH 4-5.5), it is also called «acid mantle.»

Sebum performs several functions. It prevents water loss and drying of the skin and protects it from damage by acidic or alkaline medium, thus preventing the penetration of bacteria.

It is believed that sebaceous glands of the skin secrete from 100 to 300 g of secretion per week. Most fat is released on the wings of the nose, the chin and ears. The function of the sebaceous glands is under the direct influence of neuroendocrine system.

Metabolic function of the skin. The skin is involved in the general metabolism of the body – water, mineral, nitrogen, carbohydrate, vitamins. The skin accumulates and releases water, fat, glycogen and salts.

The skin takes special place in water exchange process, as it is the second after muscles reservoir that collects water introduced into the body. Water content in the skin is 62-71%, but in the subcutaneous fat it is much smaller – up to 10%. In children's skin there is much more water than in the adults' skin, and its volume decrease with the age. Mineral components in the skin make 1% of its weight. The skin holds not only water but also sodium chloride (kitchen salt), and at some acute skin diseases its content increases to dangerous levels.

In mineral metabolism an important role is played by potassium and calcium, potassium and sodium ratios, which vary depending on the diet.

Nitrogen metabolism of the skin is mainly caused by proteins. Of the proteins, the skin is rich for collagen (which makes up to 98% of all the proteins of the skin and is in the dermis) and keratin (found in the horny layer of epidermis, hair and nails).

Carbohydrate metabolism in the skin is characterized mainly by the content of glucose. For a healthy person the norm is 50-70 mg%. The amount of glucose in the skin is not equal to its content in the blood. ***Receptor function of the skin.*** The skin, being the peripheral unit of skin analyzer, is a large receptor field that perceives a number of sensations from outside and transmits them to the central nervous system.

There are the following types of skin sensitivity:

- tactile (sense of touch and pressure);
- pain;
- temperature (feeling of cold and heat).

Sense of touch (pressure) is perceived by tactile corpuscles, the number of which varies on different parts of the skin. The sense of deep pressure is perceived by Pacinian corpuscles.

Pain is perceived mostly by free nerve endings located in the epidermis and dermis.

Temperature sensation, perception of heat and cold, is of great importance for the reflexive processes that regulate body temperature. It is assumed that thermal stimuli are perceived by Ruffini's corpuscles, while cold is perceived by terminal Krause's bulbs. There are much more cold points on the entire surface of the skin than the heat once.

Suction (absorption) function. Water and dissolved salts are not absorbed by the skin as translucent and horny layers of the epidermis contain lipids preventing the penetration of water into the skin. However, water-soluble substances can be absorbed through the hair follicles and sweat gland ducts. Alternately, fat-soluble substances are easily absorbed through the epidermis.

Respiratory function of the skin involves the exchange of gases i.e., the uptake of an oxygen and the emission of carbon dioxide and water vapor.

The skin plays small role in the exchange of gases: nearly 1/180 of the oxygen penetrates into the body through the skin and from 1/90 to 1/65 of carbon dioxide is released via lungs. Via sweat glands skin releases up to 800 g of water vapor per day, thus surpassing the lungs for more than twice.

Gas exchange of the skin is qualitatively identical to pulmonary gas exchange and is limited to the diffusion between blood capillaries of the skin and the surrounding atmospheric air.

Repository function. At room temperature, most of the skin blood vessels are in semi-contracted state, which means that the rate of blood flow in the capillaries is negligible. With increasing temperature, dilated blood vessels of the dermis can hold

up to 1 liter of blood (reposition of the blood), and their proliferation may lead to significant changes in the circulatory system.

Morphology of oral mucosa

Oral cavity *(cavitas oris)* is the initial unit of the digestive tract. In front and on the sides, it is limited by lips and cheeks, on the top – by hard and soft palate, at the bottom – by the mouth bed. With mouth lips closed, the mouth has fissure shape, while with mouth lips open – it has round shape.

Oral cavity consists of two sections: front or vestibule of mouth *(vestibulum oris)*, and rear – oral cavity itself *(cavitas oris propria)*. In front and on the sides, vestibule of the mouth is limited by lips and cheeks, from behind and inside – by teeth and mucous membrane upper and lower jaws alveolar processes. Oral cavity is connected with pharyngeal cavity via pharynx.

Vestibule and oral cavity itself are covered with mucous membrane.

Oral mucosa *(tunica mucosa oris)* consists of three layers – epithelial, proper mucous plate *(lamina propria)* and submucous layer.

Epithelial layer. Oral mucosa is covered with stratified squamous epithelium. Its structure is not the same in different parts of the oral cavity. The epithelium on the lips, cheeks, soft palate and mouth bed is normally not coarsen and consists of basal and spinous layers, while on the hard palate and gingiva, it is exposed to keratinization. Thus, besides the aforementioned layers, it has granular and horny layers. It is believed that epithelium keratinization is the result of its response to the influence of the stimulus, especially mechanical.

Between basal layer cells there are individual leucocytes. Through the epithelium, gingival sulcus epithelium in particular, they can penetrate to the oral cavity, and, thus, appear in the oral fluid. In some parts of the epithelium one may find melanocytes i.e. the cells that make melanin. At the border of the epithelial layer and the lamina propria there is a basal membrane consisting of fibrous structures.

Proper mucous plate *(lamina mucosa propria)*, which contains a layer of epithelium, consists of a dense connective tissue. On the border with the epithelium, it forms numerous processes, namely papillae that protrude into the epithelial layer at different depths. The connective tissue is represented by fibrous structures i.e. collagen and reticular fibers and cellular elements i.e. fibroblasts, mast and plasma cells and segmented leukocytes. Proper mucous plate of cheeks and lips is the richest for cellular elements.

Macrophages that perform protective function phagocytize bacteria and dead cells. They are actively involved in inflammation and immune reactions. Labrocytes (mast cells), which are characterized by the ability to produce biologically active substances i.e. heparin and histamine, ensure microcirculation and vascular permeability. Labrocytes are involved in delayed type of hypersensitivity reactions.

Proper mucous plate smoothly goes to submucosa *(tunica submucosa)* formed by loose connective tissue. It has small vessels and minor salivary glands. The intensity of submucosa determines the mobility of tunica mucosa of the mouth.

Physiology of oral mucosa

Due to anatomical and histological features, mucosa performs a number of functions, which include protective, plastic, sensory and suction.

Protective function. This function is ensured by a number of mucosa properties. Microorganisms and their metabolic products together with the scales of epithelium are removed from the mucosal surface. An important role in the protective function is played by leucocytes that penetrate into the oral cavity through the epithelium of tooth-gingival attachment (gingival sulcus). Normally, 1 ml of saliva contains 4 000 leukocytes. In case of diseases of mucous membranes of the mouth (gingivitis, stomatitis) the number of white blood cells in the oral fluid increases dramatically.

Plastic function. This function is ensured by high mitotic activity of the epithelium, which, as some people believe, is by 3-4 times higher than mitotic activity of skin cells. This accounts for high regenerative capacity of oral mucosa, which is often exposed to some kind of damage.

Sensory function. This function is carried out due to the presence of cold, heat, pain, taste, touch receptors. Oral mucosa serves as a reflexogenic zone of glands and gastrointestinal tract muscles. It has been found out that stimulation of taste receptors alters the function of the digestive tract, affects the composition of blood and cardiovascular system. Changes in the level of sensitivity occur not only by increasing or decreasing the sensitivity threshold, but, as conducted studies showed, through the mobilization or demobilization of functional receptors.

Suction function. Oral mucosa has the ability to absorb a number of organic and inorganic compounds, including drugs and toxic substances. It has been defined that the level of suction can be changed. Tanning agents reduce the intake of substances, while the impact of physical factors (electrophoresis, ultrasound, phonophoresis) increases it.

Major skin pathological changes

A significant number of skin diseases are of an inflammatory character. Clinically the inflammatory process is divided into acute and chronic. Such division of inflammatory processes, which is based on their duration, is rather arbitrary and mainly has clinical significance. They believe that pathologic feature of the process transition into chronic type is the proliferation of connective tissue in the area of inflammation, which in average takes place in two months after the beginning of inflammatory process.

Inflammation is a reflex reaction of the body that occurs under the influence of various pathogenic stimuli. Inflammation is mostly a local manifestation of the general reaction of the body. Due to the impact of the pathogenic agent, which excites the nervous system, there appears a complex of pathological phenomena i.e. local

damage to the tissues and organs, breach of circulatory and metabolic processes therein. These processes are interdependent and interconditioned and reflect reactive properties of the body the latter had acquired in the course of evolution.

From the pathologic point of view, inflammation is the result of interaction between damaging agent and the body. The combination of these interactions can vary dramatically, yet in the microscopic picture of inflammation they always single out the following changes: alteration – manifestations of tissue damage (degeneration and necrosis of tissue elements), exudation – changes in the blood vessels (emergence of liquid and formed elements from vessels) and proliferation – multiplication of tissue elements.

Peculiar pathological processes occur in the epidermis, which is related to anatomical features of its structure.

There are three main types of inflammatory changes in the epithelium.

The first type of serous inflammation of the epithelium is ***intracellular edema***, granular degeneration of cells *(alteratio cavitatis),* which is characterized by the appearance of malpighian layer of vacuoles in the cytoplasm of cells, which are located near or around the nucleus, and, thus push it to the periphery. The nucleus is deformed and often has all signs of degeneration and pyknosis. The increasing of intracellular edema can lead to the complete disintegration of epithelial cells. Less commonly, the vacuoles are localized in the nucleus of cells. This phenomenon is called vacuolar (hydropic) degeneration. The nucleus swells, chromatin disappears. The nucleus is transformed into a round vial filled with a liquid, where sometimes nucleolus floats.

The second type of serous inflammation is ***intercellular edema*** of the epidermis *(status spongioides)*, which is characterized by the fact that the exudate separates individual epithelial cells and stretches intercellular bridges. The progression of the process leads to the rupture of intercellular bridges, loss of intercellular communication and the beginning of formation of epithelial vesicle.

The third type of inflammation of the epithelium is ***ballooning degeneration*** *(degeneratio balloon),* which is a necrobiotic process. In addition to the most profound changes in the epithelial cells, intercellular bridges are destroyed. The cells lose their mutual connection and take the form of spherical formations freely floating in the intercellular spaces.

Inflammatory skin diseases are mainly accompanied by the combination of intra- and intercellular serous edema of the epidermis.

Besides serous inflammatory phenomena, there is a number of other pathological processes that can develop in the epidermis. They include as follows:

acanthosis, which is characterized by increased proliferation of spinous layer cells, that results in elongation and expansion of epithelial ridges;

acantholysis, in which atrophy of intercellular epithelial bridges takes place, a strong connection between epithelial cells is disrupted and the cells easily shift to one another, which leads to the detachment of more or less significant layers of the

epithelium. For the first time, acantholysis phenomena have been described in pemphigus by Nikolskyi P.V.;

hyaline degeneration of cells, which is characterized by the appearance of dense homogeneous translucent vitreous substance in the cells that is called the hyaline;

hyperkeratosis as a thickening of the horny layer;

granulosis as a thickening of the granular layer of the epidermis;

parakeratosis, wherein stained nuclei are found in the cells of the horny layer; granular layer is absent;

epidermis atrophy, which is observed in a number of skin diseases. The number of epidermis layers is reduced to a minimum, the cells decrease in volume. Atrophy may extend to the entire epidermis, when there is also an atrophy of epidermis ridges i.e. smoothing of the border between the epidermis and dermis or, of their individual layers.

2.2 Introduction to Dermatovenereology. General Issues of Etiology and Pathogenesis of Dermatoses

In the course of the disease most of the changes in the body is a manifestation of its dynamic response to the influence of pathogenic agent. The state of the organism, in its turn, is closely associated with social conditions of existence, social conditions of life and work of a man, which is the basis to define the disease as a social phenomenon.

Etiology studies the causes of diseases. These causes may be of exogenous and endogenous nature. The term «pathogenesis» refers to the mechanism of occurrence and development of the disease and the ways of its spreading throughout the organism.

Exogenous and endogenous factors of skin diseases occurrence

Exogenous factors of dermatoses (i.e., skin diseases) include those which affect body and skin from outside. There are physical factors (mechanical, thermal, impact of radiant energy), chemical and biological (viruses, bacteria, fungi and fungous organisms, algae sometimes, some arthropods, plants, etc.).

Endogenous factors are very diverse, to include the impact of almost all organs and systems on the skin.

Thus, endocrine disorders as an etiological factor in the occurrence of skin diseases are diverse and have a variety of clinical manifestations, to include the disorder in the anatomical structures, alteration of skin glands function, appearance of autonomic and vasomotor symptoms, disorders of pigment etc.

The disorder in lymph, blood circulation and blood diseases give rise to elephantiasis, acrocyanosis, rosacea, leukemia skin manifestations etc.

Autointoxication caused by the absorption of abnormal food from gastrointestinal tract, the disorder in excretory capacity of kidneys and other organs also leads to a number of diseases (urticaria, pruritus etc.).

Many chemicals and nutrients that enter the body can lead to a number of toxic erythema and itching dermatoses.

Microorganisms, which originally are localized in internal organs or blood, could also cause a number of diseases, such as septic erythema, secondary skin actinomycosis etc.

The clinical picture of skin and sexually transmitted diseases is a compound complex of symptoms consisting of various manifestations. First of all, these manifestations include those that are localized on the skin and visible mucous membranes. We differentiate between subjective and objective symptoms. The subjective symptoms are those that are felt by the patient and for the most part directly touch the skin. Objective symptoms include changes that the doctor notes to the patient during checkup or other physical examination of the skin.

It is important to remember that dermatoses are often accompanied by symptoms relating to the whole organism. These include fever, anemia, weakness and unstable mental status. However, skin diseases may occur due to the diseases

of individual organs or systems, in particular lesions of the gastrointestinal tract, nervous and endocrine systems, blood formation organs, blood and lymph vessels.

Subjective and objective symptoms of skin diseases

Subjective symptoms. Subjective symptoms of skin diseases largely depend on the individual peculiarities of the patient. Some of them react vigorously and vividly on relatively minor manifestations of the disease, while others are quietly even to rather pronounce manifestations of dermatosis. An example is the itching sensation. This feeling deprives some patients of sleep and disrupts their overall health, while others do not perceive itching as discomfort and almost do not notice it.

Subjective symptoms are different in character and strength, depending on personal feelings of the patient. Such feelings mostly include different sensory disturbances in the form of hyperesthesia (itching, burning and pain), hypo-and anesthesia, paresthesia (formication, contraction and numbness). These symptoms are felt by the patient, and only some of them can be marked by a doctor: disorder of skin sensitivity and itching sensation, which the doctor can notice from skin excoriations (scratching) available on the patient's skin.

Objective symptoms. To study objective symptoms in the patients, one should study the condition of the entire skin, wherefore you should use visual impressions and tangential sensations. The study of the entire condition of the skin includes the study of its color, luster, turgor, dryness or humidity, temperature reaction and local changes. When studying local changes of the skin it is very important to know the elements of skin rashes that cause skin lesion.

Diagnostic stages

To correctly build the diagnostic process it is necessary to know its three stages – morphological, clinical and ethiopathogenetic.

Morphological stage concludes with the establishment of preliminary diagnosis. It can be made analytically or synthetically. The first type of morphological stage is more typical for Viennese dermatological school. In this case, first of all, you should determine all the morphological elements of the patient, i.e. make tests. First determine whether the element rises above the level of the skin, whether it has a cavity, how the element's regression goes on – disappears without a trace, leaves crust, flakes, scars etc.

Further analysis is performed as follows. If the element is not raised above the surface of the skin, it's a macule (the only primary element, not protruding above the skin). In that case, you need to determine the nature of this macule (see below). If the element is raised above the surface of the skin, it is necessary to conduct a survey for the presence or absence of a cavity therein, and then analyze primary cavitary and non-cavitary elements. Besides examination, the nature of the morphological element is also established on the basis of palpation, vitropression, scraping etc.

Synthetic type of the morphological stage of diagnostics is more characteristic for dermatologists sticking to French school teaching foundations. In this case they first estimate the entire rash, determine the interdependence of individual elements, their prevalence, localization, grouping, mono-or polymorphism. Thus, for example, in Gibert's disease (pityriasis rosea) oval rash elements are located along Langer's lines on the chest skin and the overall picture resembles a Christmas tree, while in psoriasis the lesions are often localized on the scalp, elbows and knees and lower back. This allows us to make a preliminary diagnosis and then proceed to the analysis of the nature of individual rash elements and use additional methods of examination.

After completion of the morphological stage and establishing a preliminary diagnosis, one proceeds to the *clinical stage* of diagnostics. At this stage, first of all they associate the results of morphological analysis with disease anamnesis, and then use additional studies (vitropression, Brocq scraping, determination of dermographism etc.). In this case, they eliminate the diseases of those that have been suspected, when setting preliminary diagnosis, starting from the least possible. This stage ends up with the establishment of clinical diagnosis, which, e.g., can be eczema, psoriasis.

The top stage of diagnostics is *ethiopathogenetic*, which ends up with the establishment of final diagnosis. At this stage, they define the form of nosologic unity of the disease, its etiologic and pathogenetic nature with all the features typical of the body of a particular patient. At this stage of diagnostics they define the presence of concomitant diseases and their relationship with dermatosis, thus making it possible to assign a rational combination therapy.

Morphological elements of rash on the skin and mucous membranes

Rash or efflorescence *(eruptio, efflorescentia)* are clinical and morphological changes in the skin (exanthema) and mucous membranes (enanthema) that develop under the influence of various endogenous and exogenous factors and are a major symptom of skin and a variety of infectious diseases. The peculiarities of morphological elements of the rash, their color, shape, texture, location and other attributes coupled with the general properties of the skin (e.g. color, elasticity, secretory activity) are critical to the diagnostics of skin diseases.

Any skin rashes consist of certain morphological elements that make up the alphabet of dermatology. It is impossible to describe any skin changes without proper knowledge of this alphabet.

There are primary and secondary rash elements. Primary elements are those that arise as a direct consequence of pathological process in the skin. Secondary elements develop from the primary as a result of their spontaneous evolution or arising in the course of treatment. One should consider the relativity of division of efflorescence into primary and secondary elements. This is related to the fact that, in

certain circumstances, primary elements can act as secondary i.e. be a consequence of the evolution of an element, and secondary elements, in some diseases, may be characterized as primary efflorescence. Below, to characterize individual elements we will give examples of how primary elements act as secondary and vice versa.

Primary morphological rash elements

Primary morphological rash elements are divided into non-cavitary (a spot, papule, tubercle, nodule, blister) and cavitary (vesicle, bulla, pustule).

A spot *(macula)* is an element having no relief, but with changed color of skin. Spots are divided into vascular, pigmented and hemorrhagic.

Vascular macules can be inflammatory and non-inflammatory. Inflammatory vascular spots are characterized by temporary dilation of blood vessels in the dermis. When pressing the affected area of the skin, blood vessels fall down and intensity of macules color becomes lesser or a macule disappears at all, but when stopped pressing, the color of the macule restores. The color of inflammatory macules ranges from bright red to bluish-red. When self-resolved, inflammatory macules may be covered with scales, leave secondary pigment changes in skin color for a long period of time or disappear without a trace.

By the size, inflammatory macules are divided into *roseola* (up to 2 cm in diameter) and *erythema* – of a larger diameter.

Non-inflammatory vascular macules are associated with improper development of blood vessels in the skin. They are divided into congenital (e.g., vascular nevi – *naevi vasculosi*) and those acquired as a result of persistent expansion of surface vessels (e.g. telangiectasia – *teleangiectasiae*).

Pigmented macules may have high (hyperpigmentation), low (hypopigmentation) content of pigment or have not it at all (depigmentation). They can be congenital and acquired. The examples of congenital hyperpigmented spots are pigmented nevi (*naevi pigmentosi*), of acquired hyperpigmented macules – freckles (*ephelides*), of congenital depigmented macules – albinism (*albinismus*) and of acquired depigmented macules – *vitiligo*.

Hemorrhagic macules appear due to bleeding into the skin when blood vessels are injured or permeability of their walls is increased. When pressing on such macules, their color remains almost unchanged. The coloring gradually changes from red to blue, green and yellow (due to the formation of degradation products of hemoglobin). As a rule, such macules disappear completely in 1-2 weeks.

By the size, hemorrhagic macules are divided into petechiae (*petechiae*) – punctate hemorrhage, purpura (*purpura*) – up to 2 cm and ecchymosis (*ecchymosis*) – of a larger size. Separately, they distinguish linear hemorrhage (vibex, pl. *vibices*) and extensive bruising *(sugylatio)*.

There are also *macules arising from the introduction of foreign substances into the skin* (such as tattoos).

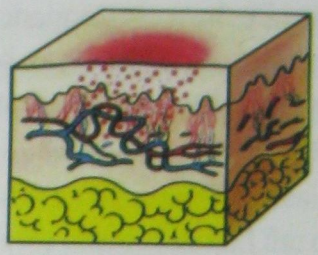


Fig. 2.2. Schematic representation of inflammatory macule *(macula inflammatoria)*.

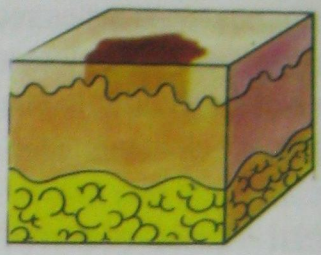


Fig. 2.3. Schematic representation of hyperpigmented macule *(macula hyperchromica)*.

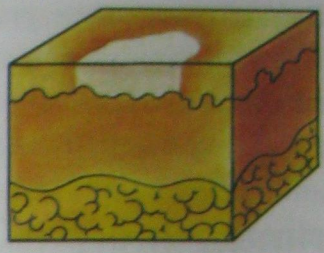


Fig. 2.4. Schematic representation of depigmented macule *(macula achromica)*.

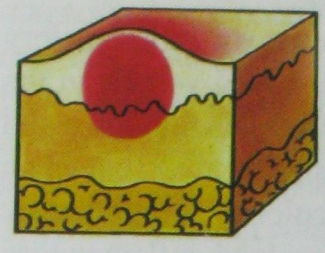


Fig. 2.5. Schematic representation of papula *(papula)*.

Papule is primary non-cavitary morphological element, which is characterized by changes in the skin surface, often its color, texture and usually disappears without a trace. According to the depth of occurrence there are epidermal papules, located within the epidermis (flat warts); dermal, which are located in the papillary dermis (*papular syphilides*) and epidermodermal (papules of psoriasis, lichen planus, atopic dermatitis). Papules can be inflammatory or non-inflammatory. The latter are formed as a result of acanthosis-type sprawl of epidermis (warts), papillomatosis-type sprawl of dermis (papillomas) or concentration of metabolic products in the skin (xanthoma). Inflammatory papules are much more common in psoriasis, secondary syphilis, lichen planus, eczema etc. At that, in the epidermis one may observe acanthosis, granulosis, hyperkeratosis, parakeratosis, while in the papillary dermis – a concentration of cellular infiltrate. Depending on the size, papules can be miliary (1-3 mm in diameter), lenticular (0.4-0.9 cm in diameter) and nummular (1-3 cm in diameter). In some dermatoses there occurs a peripheral growth of papules and their merge, which leads to the formation of larger elements – *plaques* (e.g. in psoriasis). Papules can be round, oval, polygonal in line, flat, semi-circular, conical (with pikes) in shape and thick, densely elastic, pasty and soft by consistency. Sometimes a vesication appears on the surface of papules. Such elements are called papulo-vesicles.

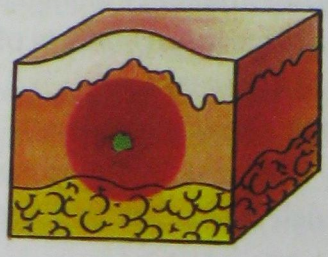


Fig. 2.6. Schematic representation tubercle *(tuberculum)*.

Tubercle (*tuberculum*) is a primary non-cavitary infiltrate morphological element that occurs in the reticular dermis and is characterized by small size (0.5 to 1 cm in diameter), change in color of the skin, its topography and texture and leaves behind a lasting trace in the form of scar or scar atrophy.

There are inflammatory and non-inflammatory tubercles. The first are formed mainly in the reticular dermis due to the formation of infectious granuloma. Clinically, they are quite similar to papules. The main difference is that tubercles usually tend to ulcerate and leave behind scars. Tubercle may also expand omitting ulcer stage with the transition into scar atrophy of the skin. Inflammatory tubercles are observed in leprosy, skin tuberculosis, leishmaniasis and tertiary syphilis. Non-inflammatory tubercles are benign growths of the dermis (fibroids, leiomyoma).

***Nodule** (nodus)* is a primary non-cavitary infiltrate morphological element occurring deep in the dermis and hypodermis and having big sizes (from 2 to 10 cm and over in diameter). To the extent the pathologic process is in progress, in most cases nodule undergo ulceration followed by scarring. They distinguish inflammatory nodule, such as syphiloma, boils and non-inflammatory nodes that result from benign deposition of fat in the skin (lipomas) or malignant proliferative processes (lymphoma). Sometimes nodule can completely resolve (e.g. boils at a formative stage), group in calcium salts or fibrous tissue (syphiloma in the areas of the joints).

Fig. 2.7. Schematic representation of a nodule *(nodus)*.

***Vesicle** (vesicula)* is a primary cavitary morphological element to 0.5 cm in diameter, which has a bottom, a tegmentum and a cavity filled with serous or sero-hemorrhagic matter. Vesications are found in the epidermis (intraepidermaly) or under it (subepidermaly). They can occur on the background of unmodified skin (with dyshydrosis) or on an erythematous background (cold sores). They are often formed due to spongiosis (eczema, atopic dermatitis) or ballooning degeneration (in simple and herpes zoster). The opening of vesications causes the formation of multiple oozing erosions, which eventually are epithelized and leave no permanent change in the skin. There are unicameral (eczema) or multicameral (with herpes) vesications.

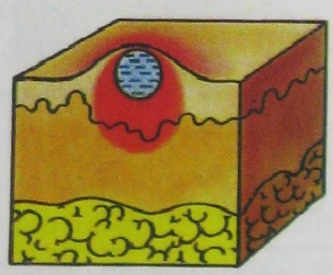


Fig. 2.8. Schematic representation of vesicle *(vesicula)*.

***Bulla** (bulla)* is a primary cavitary morphological element consisting of a bottom, a tegmentum and a cavity filled with serous or sero-hemorrhagic matter. The tegmentum can be hard or loose, thick or thin. The bulla differs from vesication by much larger size – from 0.5 to a few centimeters in diameter. The elements can be found both on intact skin and on inflamed one.

Bulla may appear as a result of acantholysis and be placed intraepidermaly (in acantholytic pemphigus), or as a result of swelling of the skin that leads to the

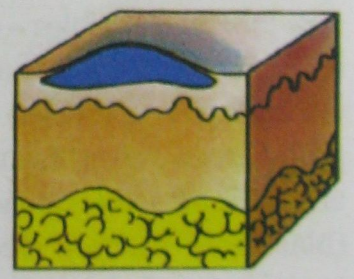


Fig. 2.9. Schematic representation of bulla *(bulla)*.

detachment of epidermis from dermis and be placed subepidermaly (simple contact dermatitis). In places of open bulla there appear erosive surfaces, which eventually are epithelized and leave no permanent change in the skin.

***Pustule** (pustula)* is a primary cavitary morphological element filled with purulent matter. According to the location on the skin they distinguish superficial and deep, follicular (usually staphylococcal) and non-follicular (usually streptococcal) pustules. Superficial follicular pustules are formed at the mouths of the follicle or cover up to two thirds of its length, which means that they are located in the epidermis or papillary dermis. They have a conical shape, often permeated with hair in the central part where one may see yellowish purulent matter. Their diameter is about 1-5 mm. At pustules regression, a purulent matter may shrivel into a yellowish-brown crust, which then disappears. At the site of follicular surface pustules there are no any persistent skin changes; only temporary hypo-or hyperpigmentation is possible. Superficial follicular pustules are observed in ostiofolliculitis, folliculitis and sycosis usual. Deep follicular pustules (deep folliculitis) cover the entire hair follicle and are located within the entire dermis and often hypodermis.

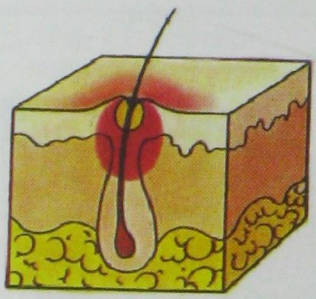


Fig. 2.10. Schematic representation of superficial follicular pustule *(ostiofolliculit)*.

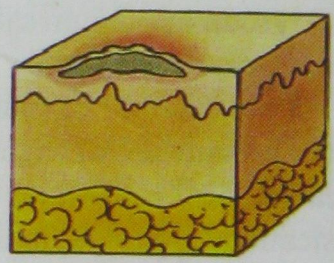


Fig. 2.11. Schematic representation of superficial non-follicular pustule *(impetigo streptogenes)*.

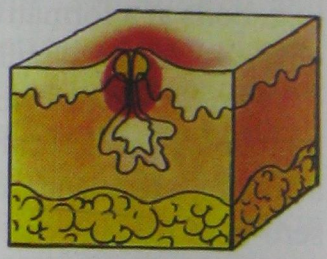


Fig. 2.12. Schematic representation of a blackhead *(acne)*.

Superficial non-follicular pustules – phlyctenule *(phlyctaena)* have a tegemntum, the bottom and a cavity filled with cloudy matter, surrounded by hyperemia aureole. They are located in the epidermis and outwardly look like pustule with a noticeable content. Observed in impetigo. At pustule regression, the exudate shrinks in crust, after the rejection of which there remains temporary de-or hyperpigmentation. Deep non-follicular pustules – ecthymas *(ectyma)* form ulcers with purulent bottom, which are observed in chronic ulcerative pyoderma etc. They remain scars. Ecthyma that is covered with a layered crust resembling snails shell is called rupia *(rupia)*. The pustules can appear around the excretory ducts of sebaceous glands (acne vulgaris), and since sebaceous glands open into the hair follicle, they are also of follicular nature (blackhead – *acne*). Deeper pustules that are formed around excretory ducts of apocrine sweat glands, in spiradenitis, open via fistulous tract and leave behind scars.

***Blister** (urtica)* is a primary non-cavitary exudative morphological element, resulting from the limited acute inflammatory edema in the papillary dermis and

differing by ephemeral nature (exists from few minutes to several hours, disappears without a trace). Usually, blister occurs as an immediate, sometimes delayed allergic reaction to the endogenous or exogenous stimuli. It is observed with insect bites, hives, drug reaction and Dьhring dermatitis. Clinically, blister is a thick elevated element of round or irregular shapes, pink in color, sometimes with a whitish tinge in the center accompanied by itching and burning. It leaves no any (even temporary) secondary changes on the skin.

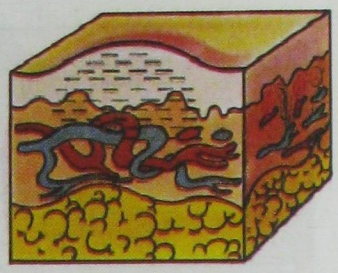


Fig. 2.13. Schematic representation of blister *(urtica)*.

Secondary morphological rash elements

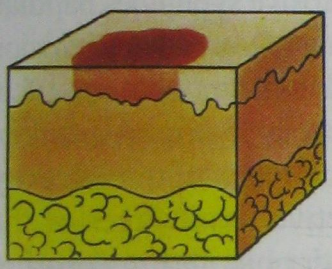


Fig. 2.14. Schematic representation of hyperpigmentation *(hyperpigmentatio)*.

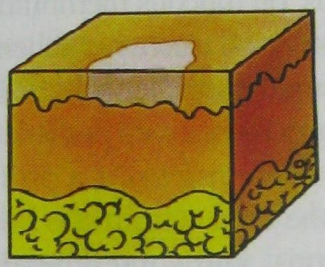


Fig. 2.15. Schematic representation of hypopigmentation *(hypopigmentatio)*.

Secondary morphological rash elements include secondary spot, a crack, excoriation, erosion, ulcer, a scale, scar, tripe, lichenification, vegetation.

Secondary hypo-hyperpigmentation *(hypo-seu hyperpigmentatio secundaria)* is a change of color in the place of primary (papules, pustules etc.) or secondary (erosions, excoriations) elements. For example, in places of former papules in psoriasis there are often depigmentation areas that exactly match the contours of former primary elements (psoriatic pseudoleukoderma), while in regression of planus papules, usually, there is a hyperpigmentation persisting for several weeks and even months.

A crack is a secondary morphological element representing a linear breach of skin integrity caused by weakening of skin elasticity. Cracks are divided into superficial *(fissura)* (which are located within the epidermis; epitelize and regress without a trace, e.g., in mycosis, neurodermatitis etc.) and deep *(rhagas, pl. rhagades)* localized within the epidermis and dermis, often bleeding with formation of hemorrhagic crusts, regressing to the formation of a linear scar, e.g. in congenital syphilis).

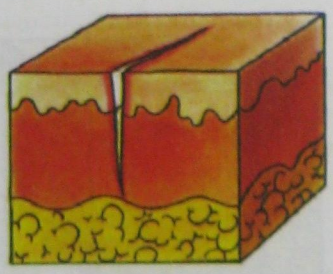


Fig. 2.16. Schematic representation of deep cracks *(rhagas)*.

Excoriation *(excoriatio)* ***or scratching*** is manifested by breach of skin integrity caused by mechanical damage resulting from injuries and scratching.

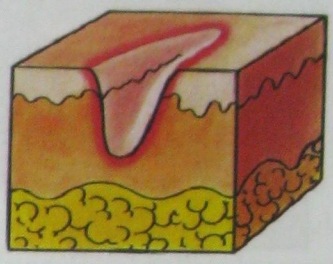


Fig. 2.17. Schematic representation of excoriation *(excoriatio)*.

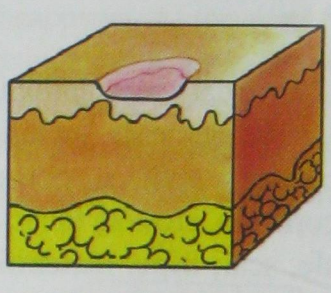


Fig. 2.18. Schematic representation of erosion *(erosio)*.

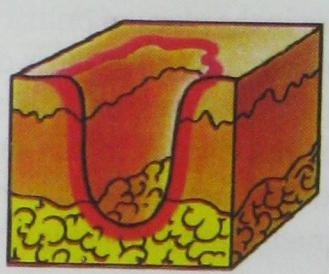


Fig. 2.19. Schematic representation of ulcer *(ulcus)*.

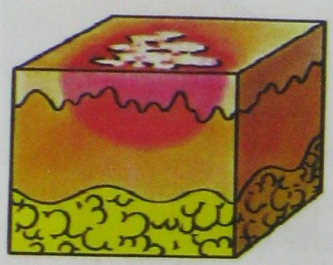


Fig. 2.20. Schematic representation of scale *(squama)*.

Excoriations are most often linear or match the contours of the damaged primary rash element. Depending on the depth of damage to the skin, excoriation may regress without a trace or with a scar, hypo-or hyperpigmentation. Excoriations are objective evidences of itching in patients.

Erosion *(erosio)* occurs at the opening of superficial primary cavitary morphological elements or oozing lesion of inflammatory papules and represents a breach of skin or mucous membranes integrity within the epidermis (epithelium). Erosions appear on the places of vesicles, blisters or superficial pustules and have the same shape and dimensions as primary elements. Sometimes erosion may occur on papular rash, especially when it is localized in the folds, on mucous membranes (erosive syphilides papular, erosive and ulcerative lichen planus). Regression of erosion occurs by epithelialization and ends up with complete disappearance with possible temporary hyperpigmentation.

Ulcer *(ulcus)* is a profound breach of the integrity of the skin within the connective tissue layer of the dermis, and sometimes hypodermis and underlying tissues. It occurs at the opening of tubercles, nodules or deep pustules. The ulcer has the bottom and edges, which can be benign (tuberculosis) or firm (skin cancer). The bottom may be smooth (syphilitic chancre) or uneven (chronic ulcerative pyoderma) covered with a variety of discharge, necrotic plaque or granulation. The edges can be undermined, steep and soucer-shaped. Ulcers always remain scars.

Scale *(squama)* represents horny plates, which, when rejected, result in desquamation *(desquamatio)*. Physiological desquamation occurs constantly and generally is unnoticed. In pathological processes (hyperkeratosis, parakeratosis) desquamation becomes much more pronounced. Depending on the size of scales, desquamation is divided into furfuraceous (scales are small, soft, as if powdering the skin), lamellar (large scales) and gross lamellar (layered rejection of horny layer). Furfuraceous desquamation is observed in pityriasis versicolor, rubrofitii; lamellar – in psoriasis, while gross lamellar – with erythroderma. The scales are loosely held, easily removable (in psoriasis) or sit tight and are separated with difficulty (in lupus).

White scales are typical of psoriasis, yellow – of seborrhea, dark – of some types of ichthyosis. In some cases, the scales are soaked with exudate, thus forming scale crusts (in exudative psoriasis).

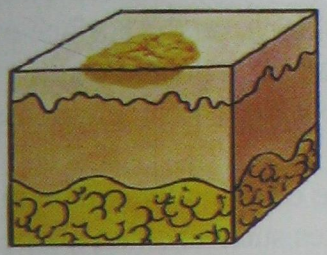


Fig. 2.21. Schematic representation of serous crust *(crusta serosa)*.

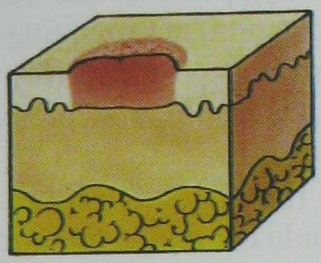


Fig. 2.22. Schematic representation of hemorrhagic crust *(crusta haemorragica)*.

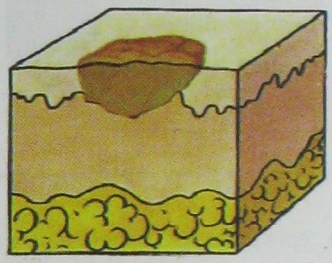


Fig. 2.23. Schematic representation of purulent crust *(crusta purulenta)*.

***Crust** (crusta)* occurs when the contents of vesications, blisters, pustules dries out. Depending on the type of fluid, crusts may be serous, hemorrhagic, purulent or mixed. The shape of crusts is often wrong, although its contours match with the contours of primary rash.

***Scar** (cicatrix)* arises in the healing of ulcers, deep pustules, cracks and excoriations. It is a newly formed coarse-fibered connective tissue (collagen fibers). In terms of elation to the surface of the skin, scars can be superficial and deep, atrophic or hypertrophic. There are no skin appendages (hair, sweat and sebaceous glands) in the scars; epidermis is smooth and translucent. The color of fresh scars is red, then it becomes pigmented and finally – white.

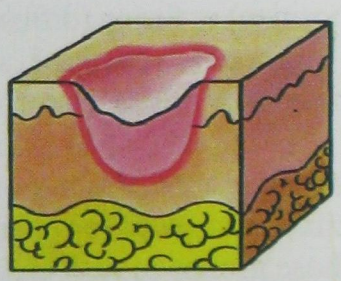


Fig. 2.24. Schematic representation of a scar *(cicatrix)*.

Cicatrical atrophy *(atrophia cicatricans)* is a quantitative and qualitative reduction of all layers of the skin that occurs with resorption of deep dermal infiltrate without prior ulceration. In this case, the skin is thinned, devoid of normal pattern, often impressed if to compare to the surrounding intact areas, and looks like tissue paper, which is particularly noticeable in the side of compression. Similar changes are observed with lupus, scleroderma etc.

***Lichenification** (lichenificatio,* or *lichenisation – lichenisatio)* is characterized by increased skin pattern, thickening and densification of skin due to papular infiltration and hyperkeratosis. In lichenification the skin resembles shagreen skin. Such changes are often formed in chronic itching dermatoses, which appear in the form of papular efflorescence (atopic dermatitis, neurodermatitis, chronic eczema).

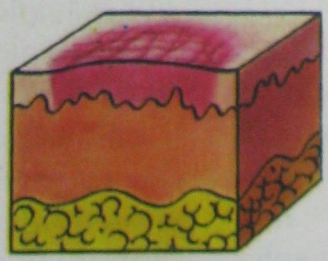


Fig. 2.25. Schematic representation of lichenification *(lichenificatio)*.

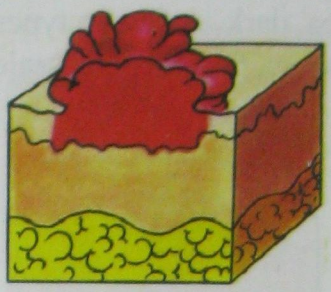


Fig. 2.26. Schematic representation of vegetation *(vegetatio)*.

Vegetation *(vegetatio)* is characterized by the growth of the papillary dermis, has a villous surface resembling a cauliflower or a cock's comb. Vegetation often appears at the bottom of erosive ulcers (wet vegetation) at vegetating pemphigus, on the surface of primary papular rash (dry vegetation) at genital warts.

Determination of morphological rash elements properties

Having established the type of rash elements, you should determine their properties such as color, shape, configuration, consistency, borders, interrelation, placement, location, monomorphism or polymorphism.

The color is described by conventional terms, and it is important to identify the shades of color, which is of great diagnostic value.

The boundaries of rash elements can be clear and fuzzy.

Configuration of rash elements can be round, oval or polyhedral (polygonal).

The consistency of rash elements can be soft or dense.

Interrelation of morphological elements is characterized by their isolation (focal position) or fusion (diffuse location).

There are grouped, systematic or random, symmetric or asymmetric types of placement of rash elements.

Grouped rash is a rash placed in a limited area in the form of certain right or wrong groups. Right groups of elements resemble geometric shapes – a circle, half circle and oval, for example in secondary recurrent syphilis. Wrong groups are those groups of rash elements, which are placed randomly on the site, not making regular geometric shapes. For example, these can be Cuspal syphilides.

Ordered rash is located on the nerve trunks, blood vessels, such as the placement of vesications in herpes zoster.

Disorderly arrangement of rash is characterized by the absence of any patterns in the distribution of elements as in secondary syphilis.

Symmetrical placement of rash is defined by its presence on the left and right parts of the body. It has a diagnostic value, e.g., in determining true eczema.

The localization of rash can be typical (favorite) and atypical (negative).

In a typical localization, rash in whatsoever disease, is observed in definite areas of the skin. For example, in psoriasis, these areas are extensor surfaces of elbows and knees. With atypical localization, rash is found in those areas where, in whatsoever dermatoses, it usually does not occur. For example, in scabies and conventional acne, rash is absent on the palms and soles.

Rash is also divided into monomorphic and polymorphic.

Rash monomorphism is characterized by the presence of primary morphological elements of the same kind. An example is the rash of lichen planus.

Polymorphic rash consists of various elements – either only primary (true polymorphism), or primary and secondary, which are derivatives from primary ones (false or evolutionary polymorphism).

2.3 The Patient's Examination Practice Applied in Dermatovenereology

Examination of the patient in the dermatology clinic is held under the general plan, but has a number of features.

When questioning the patient they establish the time of presence of this or that rash, draw initial picture of the development of the disease, study the clinical course of the disease – whether there were any remissions or relapses, learn about other previous diseases, about the nature and circumstances of the patient's working and living conditions, as well as previous treatment.

Having collected the anamnesis and clarified all the patient's complaints, the latter should be offered to show the areas that disturb him the most. One should examine the entire skin and visible mucous membranes. General examination of the patient often allows setting a correct diagnosis based on such symptoms of the skin to which the patient may or may not pay enough attention. At that, it is very important to spare the feelings of shame of the patients, especially women, do not force them to undress completely, but gradually open individual areas of the skin. Quite often, even at such examination of the lesion and of the entire skin it is possible to set the diagnosis, having dully assessed the existing skin and visible mucous changes.

Pay attention to the color of the skin, which in some cases allows establishing the presence of lesions of gastrointestinal tract, cardiovascular and respiratory systems and blood system. This helps to identify the presence of comorbidities, determine the etiology of the disease and thereby clarify the dermatological diagnosis.

In some cases there is a need to apply a number of additional studies (both, special physical examinations and laboratory and instrumental checks).

The diagnostics of skin and venereal diseases (diseases, primarily, transmitted via sexual contact) is an important step in diagnostic and treatment work of a doctor, as establishment of correct diagnosis is the key to a successful choice of tactics to further manage the patient. In most cases skin diseases assume the appearance of a variety of visceral disorders on its surface, which represent one of the features specific to a particular common disease. Many infectious diseases are accompanied by skin symptoms that allow setting correct diagnosis.

Common and special examination procedures in Dermatology and Venereology

In Dermatology and Venereology they use both common and special examination procedures (vitropression, determination of dermographism, dermoscopy etc.). For example, a general study of the morphology of blood helps to diagnose dermatoses, which are based on organic or functional disorders of hematopoiesis; the definition of ESR and sigma ESR allows you setting the activity of the inflammatory process and clarifying the diagnosis.

Diascopy (vitropression). *Vitropression* (from Greek *δια* – through and *πκπρπy* – I see) is a pressing on the affected area of the skin using diascope

(special transparent plastic plate) or a glass slide (vitropression). With this method it is possible to determine the nature of macule element (vascular, hemorrhagic or pigmented): thereat, vascular spots disappear, while hemorrhagic and pigment macules remain. Vitropression is also used to diagnose lupus: when pressing, tubercles change their color to yellow (the phenomenon of «apple jelly»).

Determination of dermographism. Dermographism (from Greek *δερμα* – skin and *γραφω* – I write) is a vasomotor response to mechanical stroked stimuli that are used to evaluate the tone of the autonomic nervous system based on cutaneous autonomic reflexes.

White dermographism arises in 8-20 seconds after stroked irritation with sharp object in the form of white stripes, which stay from 1 to 10 minutes. Boosted white dermographism witnesses of increased excitability of skin vasoconstrictors, which receive sympathetic innervation, and of the sympathetic tonus of skin vessels. Particularly clear white dermographism is detected on the hips. The sympathicotonia of skin vessels is obvious from a symptom called «white macules», which is similar to white dermographism that occurs when you press on the skin for about 3 seconds, provided that such symptom lasts longer than 2-3 seconds. White dermographism is usually observed in atopic dermatitis.

Red dermographism arises in 5-11 seconds (after stroked irritation of skin) as a red band, which stays from 1.5 min. to 2 hours. Usually, red dermographism is a normal phenomenon. To conclude on the predominance of parasympathetic excitability, only a very diffuse or too long (persistent) dermographism is of relative importance.

More convincing for such a conclusion is an *elevated dermographism* that appears in 1-2 minutes after stroked irritation in the form of edematous skin swelling (in urticaria and other allergodermatoses).

Skin biopsy. A *biopsy* (from Greek *βιοζ* – life and *οψη* – look) is a method of tissue sampling (biopsy) for pathologic study. Histology of biopsy is the support to clinical diagnosis. Therefore, material forwarded to pathological examination should be accompanied by a detailed description of the clinical disease and in severe cases, by the results of differential diagnostics. A biopsy is a small surgery and should be performed in compliance with aseptic and antiseptic requirements, and with the use of anesthesia.

A biopsy is divided into excisional (by excision of the fragment of tissue) and puncture (by puncturing with special tool). The advantage of puncture biopsy is minor injuries, though sometimes it is contraindicated as it may cause activation of processes in tumors. The quantity of material which can be obtained by this method is minimal.

Excisional biopsy can be traditional and electrosurgical. The latter, despite the absence of bleeding after a diagnostic intervention, has the disadvantage that marginal zones obtained by biopsy are exposed to coagulation at quite large depth and, therefore they become unsuitable for research.

The traditional excisional biopsy is performed with intrusion into the biopsy sample of affected and apparently healthy skin. Biopsy sample should include the subcutaneous fat layer. It would be reasonable to sample the material from several affected areas of the skin.

The procedure involves the following steps. Having treated the surgical field, they excise affected skin area together with apparently unchanged skin area, conduct hemostasis and put 2-3 stitches. The biopsy sample is then placed in a normal (10% neutral formaldehyde solution) or exclusive (Fleming's, Chabot's liquids etc.) retainer. The resulting material can be stored up to 4 months.

If you suspect nevus malignancy, the biopsy should be performed in oncology centers Only.

Dermoscopy. *Dermoscopy* (from Greek – *δερμα* – skin and *σκοποσ* – I see) is a non-invasive diagnostic method for visual evaluation of skin lesions by means of a special device called dermatoscope (epiflourescence microscope), allowing more thoroughly to study the surface of the skin and subepidermal structures. This diagnostic tool allows identifying morphological structures that are invisible to the naked eye, thus providing new details of clinical and morphological characteristics of skin lesions. The method involves the study of the skin or hair with a special camera equipped with a magnifying lens that is connected to computer. To test the skin and hair one should use lenses with different magnification. The resulting information is transmitted to a computer and processed via special software. Thus, the process resembles a simple scanning of skin and hair. The patient's skin can be checked on the subject of its:

- type;
- elasticity;
- humidity;
- relief;
- availability and depth of vascular lesions;
- pore size;
- pigmentation;
- sensitivity;
- depth of wrinkles.

The study is conducted with due account for age parameters, since with the increase of age moisture, elasticity and fatness of the skin may change. As concerns hair, one can measure its thickness, size of pores; determine type of scalp and condition of hair shafts.

The diagnostics allows thoroughly and clearly determining the condition of the skin using scientific methods of measurement and choosing professional beauty treatments and products for home care with due account for individual needs of the skin and hair. Application of dermatoscope allows recording primary skin changes in the computer's memory, observing the dynamics of treatment effect, discussing the results with the patient and in obscure diagnostic cases, consulting with other professionals using recorded data.

Determination of psoriatic phenomena. *Psoriatic phenomena* (Auspitz's triology) are almost constant symptoms in progressive and stationary stages of psoriasis. As a result of scraping psoriatic elements (papules, plaques), desquamation increases and scales become white resembling a drop of grinded stearin (the phenomenon of «stearic spot»). When keeping scraping up to the granular layers of epidermis, scales fall down, thus revealing a pink wet film (the phenomenon of «terminal» or «psoriatic» films). If to continue scraping (to the papillary dermis), tiny drops of blood will appear on the surface of the film. Their number depends on the number of capillaries within the papillae damaged during scraping (the phenomenon of «pinpoint bleeding» or «bloody dew»).

Baltzer's Iodine Test. *Iodine test* is an indicator of latent desquamation and is used to diagnose tinea versicolor. The macules are greased with 2-5% alcoholic solution of iodine. Due to loosening of horny layer, the solution is better absorbed within rash area, thus making the spot intensely colored than the surrounding healthy skin. If you have no iodine solution, you may use any aniline dyes. With leukoderma that remains after tinea versicolor, Baltzer's test is negative, especially in persons, who were exposed to UV radiation. This test is also positive in Fox's impetigo.

Skin examination under Wood's lamp. The technique is based on the ability of hair affected by *Microsporum* fungus, to give a bright green fluorescence when exposed to a short part of ultraviolet rays. The source of the latter is a portable specially designed mercury-quartz lamp. In order to restrain the long-wave radiation they use Wood's filter i.e. a glass impregnated with nickel salts. This method allows identifying head hair or smooth skin affected by fungi basing on their characteristic glow. Luminescence of lesions can be weak or disappear, and its color may change after the application of a 5% solution of iodine or ointments. In such cases it is advisable to wash your hair with soap and repeat procedure in 3-4 days. Reliability of the results of this method should be confirmed by the microscopy taken from hair lesions. In favus, one may observe a much darker glow resembling malachite. Skin areas affected by blastomycosis glow with pink-orange, in vitiligo one may see bright white areas dramatically pigmented on the perimeter. Lupus erythematosus, when localized on the red border of lips , gives snow-white glow in hyperkeratosis, whitish – in atrophy and blue – in acute process. Microsporia gives bright green glow, *lichen versicolor* (on the head) – a brick red and erythrasma – coral-red, which is more pronounced on the periphery.

Microscopic diagnostics of pathogenic fungi. To investigate smooth skin mycosis (trichophytosis, microsporia, tinea pedis, keratomycosis, candidiasis) you should take scales from peripheral lesion by scraping with scalpel. With patients suffering from feet, hands dyshydrosis, use scissors or razor blade to cut vesication tegmentum or desquamated epithelium fimbriae, and apply test material to glass slide. In dermatomycosis the material is taken from damaged long hair and vellus using epilation forceps, sometimes sharp side of scalpel, if hair is broken off at the level of the skin («black spots»). To investigate infiltrative-purulent processes, you

need to take hair floating in pus from the periphery using Volkmann's spoon and carry it over into a Petri dish or watch glass. Use the dissecting needle to catch the affected hair and carry it over to a glass slide.

Crush the affected hair, nail plate, thick horn mass and scales on a slide using heated scalpel and then add 2 drops of 10% solution of alkali (NaOH). For clarification purposes, the drug is heated in a spirit-fire, without boiling, until white ring appears on the periphery and then, by pressing a little bit, is covered with cover glass. The affected nail plates, thick horn mass or biopsy material is better investigated by Chernohubov's method of enrichment. The material is treated for 20-30 minutes in 20% alkali solution with a double boiling. For microscopy, use sludge remaining after centrifugation.

The drug is then examined under conventional or phase-contrast microscope, first at low and then at high magnification using a concave mirror, covered diaphragm or lowered condenser.

When studying scales, horny masses and nails physician assistant can only point whether he has or has not found mycelium or other agent of mycosis («mycelium found», «groups of budding cells found»)

Determination of Nikolsky's symptoms. *Nikolsky's Symptom* is one of the leading factors of diagnosing acantholytic pemphigus. There are three types of symptoms – marginal, interbulla, remote. The presence of any of them is of prognostic value.

Marginal symptom – the epidermis exfoliates as a result of loss of communication between the cells of spinous epidermis layer when pulling the bits of bulla tegmentum.

Interbulla symptom – the epidermis exfoliates between bulla when mechanically impacting the apparently intact skin.

Remote symptom – the epidermis exfoliates when mechanically impacting apparently intact skin distant from the lesions.

Study of smears for acantholytic cells. First cytological method of diagnostics of bullous dermatoses was proposed by A Tzank. This method is indispensable in the differential diagnostics of acantholytic pemphigus, pemphigoid and dermatosis herpetiformis (Duhring disease).

Using a piece of gum (eraser) sterilized by boiling, one lightly presses on the bottom surface of fresh bubble. The material is carried over to the fat-free, sterile glass slide, fixed for 1 min. in methanol and dried at room temperature and Gimsa stained (for 20-25 minutes by azure-eosin). The preparation is examined under a microscope at 10x40.

Acantholytic cells are degenerative dystrophic cells found in horny layer of the epidermis. They are smaller than normal epithelial cells, round, with large nucleus, occupying nearly the entire cell and intensely stained (hyperchromic). Two or more lighter nucleoli can be seen inside the nucleus. Cytoplasm is visible on the periphery in the form of the rim. It is abruptly basophilic and light blue – closer to the core.

Jadassohn's skin test. *Jadassohn's test* is performed to confirm the diagnosis of dermatosis herpetiformis. If the result of the first test is doubtful, you need to gradually perform three stages of Jadassohn's test, if necessary.

First stage. On the area free of rashes, apply 1 cm^2 of 50% ointment of potassium iodide to be placed under compress. To control the test, Vaseline compress is applied to the symmetric part of the skin. If test is positive, in 24 hours after application of potassium iodide there appears rash accompanied by itching.

Second stage. Potassium iodide compress is applied to the area of the skin where there is already a rash. If test is positive, previous eruptions become brighter, itching enhances greatly.

Third stage. Throughout the day, 2-3 times, the patient is administered 1 tablespoon of 3% aqueous solution of potassium iodide. If test is positive, new rash appears on any part of the skin accompanied by intense itching.

Sampling of genitourinary discharge for tests. Study of scrapings and discharge from genitourinary organs are mostly performed with inflammatory lesions (gonorrhea, trichomoniasis, chlamydiosis etc.). Test material shall be taken by a doctor. First, using a cotton swab moistened with isotonic sodium chloride solution wipe the external genitalia. With men, if pus does not freely flow, easily push on the back of the urethra squeezing a drop of discharge on a glass slide and evenly smear it with another glass. If there is no discharge, the material is sampled with a loop or blunt Volkmann's spoon after 5-8 hours of refrain from urination. Materials (scraping) for immunofluorescence studies are taken by Volkmann's spoon of smallest diameter in 15-20 minutes after urination, which prevents contamination of scraping with mucus and microflora secretions.

In women they usually study discharge from the cervix, vagina and urethra. Before taking tests it is prohibited to conduct any hygiene throughout the day. Cusco's mirror is introduced into the vagina. Using sterile loop, material is first taken from cervical canal, then from vagina and urethra. Cervical scrapings are informative provided that they are taken in the middle of menstrual-ovulatory cycle (not earlier than five days after the period and not later than five days prior to them). The material is taken by blunt Volkmann's spoon or special disposable instruments. In microbial cervical erosion, scrapings can also be taken from cervix.

Treponema pallidum dark-field study. This study is performed to confirm the diagnosis of primary seronegative syphilis. The technique is used in a dark field with the application of paraboloidal condenser or cardioid condensers and electric light.

The surface of chancre is twice-washed with isotonic sodium chloride solution, then a lotion with saline solution is periodically applied for 12-24 hours. Clean chancre surface is gently stroked with bacteriological loop until the serum, which is then carried over to a thin glass slide, mixed with a drop of warm saline, covered with watch glass and investigated under microscope.

If the chancre is complicated by secondary infection or phimosis, you should puncture regional lymph node. This is done by a 5 ml syringe. The node is captured with fingers, the needle is introduced into one of node poles and then the needle is gradually withdrawn and tissue juice is sucked with syringe. A drop of the resulting tissue juice is mixed with a drop of warm isotonic sodium chloride solution and investigated under microscope.

Treponema pallidum in the dark field looks like thin a little bit sparkling spirals with 8-12 uniform curls. Their movements can be translational (forward and backward), pendulous, around their axis, flexuous and are characterized by slowness and regularity.

2.A Principles of Treatment Applied in Dermatology and Venereology

The main task of treatment of patients with skin and venereal diseases is to eliminate the causes and favoring events that cause disease, and to increase the resistance of the patients' organisms. You should take into account the results of diagnostics, pharmacological properties of medications and dosage forms. Also it is necessary to take into account the etiology and pathogenesis of the disease, comorbidities, complications, tolerance of drugs and their effectiveness in the case of previous treatment. Accordingly, there is causal therapy that tackles the cause (agent) of the disease, pathogenetic therapy that is aimed at eliminating some of the pathogenesis, especially if etiology of the disease has not been established, and symptomatic therapy that is aimed at eliminating symptoms that the patient are most concerned about. It is also important to take into account the patient's psychological response to the disease it has.

There is general and local treatment of dermatoses and venereal diseases (diseases transmitted primarily through sexual contact).

General Treatment

General therapy involves systematic use of drugs (oral or parenteral) with their delivery to the lesions via blood. This treatment is used for disseminated, extensive skin lesions, infections and parasitic dermatoses, as well as diseases transmitted primarily through sexual contact.

Antibacterial agents. Antibacterial agents are substances that selectively suppress the activity of microorganisms. Under the selective effect one should understand the action only on microorganisms, maintaining the viability of the host cells and the effect on not all, but on some genera and species of microorganisms. For example, fusidic acid has a high activity against staphylococci, but has no effect on Pseudomonas aeruginosa.

Traditionally, antibiotics are divided into natural (actually antibiotics, such as penicillin), semi-synthetic (natural molecules modified products, such as Amoxicillin or Cefazolin) and synthetic (e.g. sulpha drugs, nitrofurans). At the present stage, this division has lost its relevance, because a number of natural antibiotics is produced by synthesis (chloramphenicol or laevomycetin), and certain drugs that are called antibiotics (fluoroquinolones) are actually synthetic compounds.

You should distinguish between antibiotics and antiseptics, which do not selectively impact on microorganisms and are used for their destruction in living tissues, and disinfectants intended for non-selective killing of microorganisms outside live organism (for care items, etc.).

Antibiotics are the most numerous group of remedies. All antibiotics, despite the differences in chemical structure and mechanism of action, are united by a number of unique qualities.

Antifungal agents (antimycotics). As fungal diseases (mycoses) are infectious diseases the main way of their treatment is causal treatment that assumes application of antifungal agents (antimycotics). Elimination of the pathogen leads to the

disappearance of all symptoms. Despite the large number of existing antimycotics, their application in treatment is characterized by a number of common principles, which are listed in Table. 2.1.

By chemical composition antifungal agents can be divided into the following main groups:

Azole derivatives:

- Imidazole derivatives (Ketoconazole, Clotrimazole, Econazole, Miconazole, Bifonazole);
- Triazole derivatives of (Fluconazole, Itraconazole, Terconazole).
- Allylamines derivatives (Naftifine, Terbinafine)
- Antifungal antibiotics (Nystatin, Griseofulvin, Amphotericin B, Natamycin)
- Miristamin derivatives (Miramistin)
- Karbamotioat derivatives (Tolciclate, Tolnaftate)
- Undecylenic acid derivatives (Mikoseptin)
- 5-fluorocytosine (Flucytosine)
- Morpholine (Amorpholine)

Table 2.1
General principles of causal treatment of mycoses (by Yu.V. Sergeev and A.Yu. Sergeev)

General principles		*Compliance means*
Efficiency	Drug's compliance with infection etiology	Knowledge of infection etiology: • attribution of causative agent to the species • drug sensitivity test Knowledge of the drug spectrum of action and its indication
	Drug's compliance with the form of disease	Knowledge of drug pharmakokinetics: • rational way of administration • drug's distribution in the lesion • time for effective concentration
Safety	Prevention of severe side and toxic effects	Knowledge of drug's side and toxic effects: • comparison of the benefits and risks of treatment • determination of contraindications • selection of appropriate dose Treatment control: • regular examination • corrective treatment
	Prevention of undesired drug-drug interaction	Knowledge of interaction and compatibility of drugs: • exclusion of non-compatible drugs • correction of a dose and prescription regimen

Of course, not all of the groups of drugs are equally well used at present. Specific indications for the use of different groups of antifungal agents are considered in the description of individual clinical forms of fungal infections.

Antihistamines. Antihistamines include drugs that prevent the interaction of histamine with histamine receptors (antihistamines) and inhibit the release of histamine from tissues that are involved in the biosynthesis and deposition of histamine and mast cells (by stabilizing their membrane). In dermatology the usage of antihistamines prevails.

The mechanism of action of antihistamines is based on competition with histamine for histamine receptors, which are of two types – H1 and H2. H2-receptors are found in the stomach and their blockade inhibits production of hydrochloric acid and pepsin by parietal cells. The term «antihistamine agents» is often referred to H1-receptor blockers, which are in the walls of blood vessels. Their blockade attenuates histamine-induced hypotension, smooth muscle spasms, reduces capillary permeability, eliminates the risk of histamine edema, prevents the development and facilitates the course of allergic manifestations. Antihistamines are divided into three generations (Table 2.3).

First generation includes so-called sedating antihistamines, which also blockade cholinergic muscarinic and serotonin receptors. They have a marked sedative effect, which limits their prescription if you need to do works requiring sustained attention, rapid and coordinated mental and physical reactions. *Second generation of antihistamines* differs by the absence of sedative properties due to selective effect on H1-receptors. Due to the ability to block potassium channels they may affect the cardiac rhythm.

Third generation is represented by active metabolites of the first and second generation of medications. They selectively affect H_1-receptors and are almost free of cardiotoxic and sedative effects.

Glucocorticosteroids. Glucocorticoid hormones (GCS) are one of the most common groups of drugs that are used to treat a variety of inflammatory dermatoses. Local administration of glucocorticoid drugs with strong positive impact on different components of pathogenesis of diverse dermatoses allows directly affecting the lesions of damaged tissue and thus arrest inflammation process in the skin without the use of systemic treatment. On the other hand, even local hormone therapy requires relative caution due to possible development of adverse effects and resistance to conventional therapeutic agents.

The success of treatment with topical GCS (local, topical) is largely determined by correct choice of the preparation and adequate use of its dosage forms in each individual case. Ever-increasing incomings of new drugs to the pharmaceutical market, makes the choice of corticosteroid agents for the practitioner rather difficult, thus requiring constant updating of its knowledge.

Today topical corticosteroids by their strength are divided into four classes (Table 2.3).

Table 2.2
Antihistamine agents' generations

Antihistamines		
I generation	**II generation**	**III generation**
Diphenhydramine (Dimedrolum, Benadryl, Allergin)	Acrivastine (Sempreks)	Cetirizine (Tsetrin, Zyrtec)
Clemastine (Tavegil)	Astemizole (Gismanal)	Fexohenadine (Telfast)
Doxylamine (Dekaprin, Donormil)	Dimetinden (Phenistil)	Desloratadine (Aerius)
Diphenylpyraline	Oxatomide (Tinset)	
Bromodiphenhydramine	Terphenadine (Bronal, Histadin)	
Dimenhydrinate (Dedalon, Dramamine)	Azelastine (Allergodil)	
Chloropyramine (Suprastin)	Levocabastine (Histimet)	
Pyrilamine	Mizolastine	
Antazoline	Loratadine (Claritin)	
Mepyramine	Epinastine (Alezion)	
Brompheniramine	Ebastin (Kestine)	
Chlorpheniramine	Bamipin (Soventol)	
Dexchlorpheniramine		
Pheniramine (Avil)		
Mebhydrolin (Diazolinum)		
Kviphenadin (Fenkarol)		
Sekviphenadin (Bikarphenum)		
Promethazine (Phenergan, Diprazinum, Pipolphen)		
Trimeprazine (Terale n)		
Oxomemazin		
Alimemazin		
Cyclizine		
Hydroxyzine (Atarax)		
Meclizine (Bonine)		
Cyproheptadine (Peritol)		

Table 2.3
Division of topical glucocorticoids by their strength

Strength	International pharmaceutical name	Invented name
Weak	Hydrocortisone acetate 0,1%, 0,25%, 1%, 5%	Hydrocortizone (ointment and cream)
Moderate	Prednisolone 0,25%	Prednisolonum 0,25%
	Clobetazone butyrate 0,1%	Emoveyt
	Mazipredone hydrochloride 0,25%	Deperzolone
	Triamcinolone acetonide 0,1%	Phtorocortum.Tricortum. Polcortolon
	Flumetasone pivalate 0,02%	Lorinden. Locacoten
	Fluocinolone acetonide 0,025%	Flucinar.Sinalar. Sinaflanum
	Fluocortolon 0,025%	Ultralan
	Prednicarbate 0,25%	Prednitop
Strong	Betamethasonedipropionate 0,1%	Diproderm
	Betamethasone valerate 0,1%	CelestodermV. Betnoveytiun
	Hydrocortisone butyrate 0,1%	Laticort. Locoid
	Mometasone 0,1%	Elocom. Moleskine. Elozone. Momederm
	Methylprednisolone aceponate 0,1%	Advantan
	Halomethazone monohydrate 0,005%	Sicorten
	Budesonide 0,025%	Apulein
	Dexamethasone 0,025%	Esperson
	Flucazone propionate 0,05%	Cutivate
Very strong	Clobetazole propionate 0,1%	Dermovate. Delor. Clovate
	Halcinonide 0,1%	Halciderm

Hydrocortisone acetate preparations belonging to the first generation and due to having the mildest action are not used in dermatological practice today. More often they use topical PSL medications, which belong to the second generation and produce a moderate effect. The third generation is represented by numerous mainly halogenated topical corticosteroids, which have moderate, strong or very strong anti-inflammatory properties due to poor percutaneous adsorption. During prolonged use of topical corticosteroids it is necessary to consider some features of the mechanism of action of halogenated steroids, which can lead to adverse

events. Side effects can be divided into local, i.e. arising in the area of application, and systemic i.e. arising as a result of penetration of drug into systemic circulation.

Local side effects include skin atrophy, striae (atrophic scars), telangiectasia, perioral dermatitis, steroid acne, hypertrichosis, activation of viral, fungal or bacterial infections, impaired trophic skin, delayed recovery, congestive hyperemia (rosacea-like dermatitis), hemorrhagic purpura, reactive dermatitis (transient burning sensation, itching, or tingling), withdrawal state (maculopapular rash in the area of application), achromatosis, development of photosensitivity, colloidal pseudomilium, local ischemia, etc.

Systemic effects may occur only at very long-term use of topical corticosteroids in large areas of the skin. Systemic absorption of corticosteroids may suppress the hypothalamic-pituitary-adrenal system. This suppression causes reduction in generation of endogenous corticosteroids and may lead to the breach in metabolism of carbohydrates and unstable blood pressure. Prolonged suppression may result in the atrophy of adrenal cortex and Cushing's syndrome. In children, adrenocortical insufficiency leads to slower growth. However, the therapeutic effect of topical steroids, if properly selected and used, is much stronger than the impact of side effects.

Recently created topical corticosteroids of fourth generation are the best in application as they successfully combine all the positive features of their predecessors, which means that they have high activity comparable with the potency of fluorinated corticosteroids and minimal adverse effects typical of hydrocortisone acetate. The fourth generation of topical steroids includes strong GCS, which do not contain fluorine atom in their structure.

The mechanism of action of topical corticosteroids is based on their interaction with steroid receptors of skin cells resulting in inhibition of the inflammatory response due to vasodilator effect, inhibition of proliferation of epidermal cells, inhibition of the release of inflammatory mediators from eosinophils and neutrophils, inhibition of hyaluronidase activity, stabilization of lysosomal membranes of epidermal cells and the impact on connective tissue (inhibition of mitotic activity of fibroblasts, reduction of production of acid mucopolysaccharides, basophilic regeneration of collagen and elastic fibers).

Topical corticosteroids have anti-inflammatory, antipruritic, anti-proliferative and immunosuppressive properties that determine their use in dermatological practice. Most often, these medications are prescribed in simple and allergic dermatitis, toxicodermatoses, insect bites and various forms of erythema, psoriasis, eczema, neurodermatitis, discoid lupus erythematosis, urticaria, strofulyuse, atopic dermatitis, pemphigus erythematosus, seborrheic dermatitis, lichen planus, cheilitis, Dьhring's dermatosis, hypertrophic and keloid scars, Alopecia areata.

In some cases, to treat acute inflammatory reactions topical corticosteroids may temporarily be used in combination with other drugs for pyoderma, mycosis, herpes zoster, preulcerous eczematous process.

Topical corticosteroids can penetrate into the skin in two ways – directly through epidermis or through open hair follicles, sebaceous and sweat glands. Transepidermal penetration is the main way for topical GCS to penetrate into the skin. However, you should use medications with caution in areas of significant hair distribution, as penetration via hair follicles accelerates penetration of steroids into the microcirculatory bed.

Penetration of corticosteroids through skin depends on six main factors, which include the place of application of the drug, the patient's age, the properties of active ingredients, the basics of the drug, its method of application, stage of disease (skin condition).

General rules for the application of topical corticosteroids:

1. Before using external GCS you should accurately set the diagnosis and define the stage of inflammatory process.
2. It is recommended to start the treatment with weak drugs (to exclude hyperkeratotic forms of dermatoses). If there is no effect after 2-3 weeks of treatment, you should switch to a more powerful drug, and after the effect, you may return to weaker corticosteroids.
3. The medication is applied to clean skin with a thin layer 1-2 times a day (depending on the properties of GCS), do not massage the skin and rub the drug.
4. In chronic processes it is advisable to use the ointment, while in acute – creams and emulsions (sometimes lotion and spray).
5. It is advised to treat scalp with lotion, gel or emulsion.
6. In case of secondary infection of the lesions you should use a combination of drugs with antimicrobial agents, while in hyperkeratotic processes – with keratolytic agents.
7. Less potent corticosteroids should be used on thin skin areas (face, folds), as well as in children and elderly people.
8. During pregnancy it is not recommended to use corticosteroids for long period of time and on large areas of the skin.
9. You should take into account all the environmental factors (temperature, humidity) that contribute to a more rapid absorption of the drug.
10. It is not recommended to use drug under occlusive dressing, except for some forms of hypertrophic dermatoses. Tightly compressing clothes and children's swaddling may serve as occlusal factor and increase the permeability of the skin.
11. You should pay attention to the properties of active components and the basis that itself can cause local inflammation.

Local treatment

Local treatment of dermatoses and STD involves the use of external resources that directly affect the lesion.

Lotions. Lotions are prescribed for acute inflammatory processes in the skin, which are accompanied by the formation of erosive and moist areas (eczema, dermatitis), burning, itching, as well as the presence of bruised and bleeding sites in the skin.

The therapeutic effect of lotions is based on the effect of cold wet and, therefore, cold (icy) medicinal solutions (potassium permanganate in the ratio of 1: 2000, 3.2% of boric acid solution and Furacilin solution in a ratio of 1:10,000 etc.) are required.

Wet-to-dry dressings. The action of wet-to-dry dressings is based on the action of moist heat, and are applied in a number of diseases (eczema, neurodermatitis etc.) that are characterized by the development of limited cutaneous lesions with marked infiltration and oozing. Apply 10-15 layers folded gauze soaked in a drug solution and then squeezed on the affected skin. On top of gauze, put somewhat larger compress paper (without wool) and tape it up. Wet-to-dry dressings should be changed after 4-6 hours to the extent of their drying. Pustular skin diseases and acute generalized inflammation are considered to be contraindications to the use of wet-to-dry dressings.

Compress dressing *(hot compress)*. Application of compress bandages is based on the prolonged action of moist heat. They are used as an aid contributing to the resorption of limited infiltration of the skin, subcutaneous fat and muscles in chronic diseases of the joints and ligaments and in a number of limited inflammation processes.

Contraindications include the breach of the integrity of skin, pustular skin diseases (impetigo, ulcer pyoderma, abrasions etc.) and acute inflammation accompanied by weeping.

A nurse prepares a bandage, gauze, compress paper, cotton wool, medical sterile tray, tweezers and all the necessary substances that are used in compress (leaden water, 5% solution of alcohol, camphor oil). These substances must be of room temperature, except for oil which is preheated to 38-39°C.

Compress dressing should be changed twice a day.

Complications with compress dressings include possible appearance of maceration of the skin, dermatitis, pruritus, pustular skin diseases. However, these phenomena rapidly disappear (with the exception of pustular disease).

Salve dressings. Salve dressings are used to get deeper penetration of the drug contained in ointments or pastes inside the skin, as well as with hygienic purpose (protection of clothes and the surrounding healthy skin from penetration of applied drugs thereon). Under dressings they often apply paste or ointment.

Pastes. A paste is a mixture of equal parts of fat base and powdered substances. They are added with various medicines (naphthalene, ichthyol, tar acids etc.). Pastes have a drying effect and are applied to surface inflammations (eczema, neurodermatitis, dermatitis etc.).

Ointment. Unlike pastes, ointments are less dense in consistency because they contain fatter base. Ointments are widely used with flakes, peels, cracks, chronic inflammatory processes etc.

Apply paste or ointment using a spatula and stripe-smear it on the lesion making light movements of spatula. On the surface area of lubricated site, put sterile gauze and tape it up (no more than 2-3 rounds of roll gauze). In case of massive crusts, the ointment is covered with compress paper, which is tightly fixed with a bandage (ointment compress dressing). This promotes more rapid loosening and rejection of crusts. In parasitic diseases that affect large areas of skin (scabies etc.), an ointment, previously applied on palm, is rubbed into the skin. Some potent substances (Naftalan oil, tar, ihtiola) are sometimes used in pure form, without any basis. Thus, using a gauze or a cotton swab apply these substances only within the lesion. Usually lubrication of lesions with a paste or ointment is applied 1-2 times a day, and with clean tar ihtiola – once in 1-2 days. Contraindications to the use of ointment dressings are acute inflammation of the skin accompanied by oozing. Frequent use of ointments or their misuse (rubbing instead of light lubrication, etc.) can worsen the disease. In case of skin irritation i.e. redness, swelling, itching or pain, the procedure should be stopped.

The paste should not be applied to those areas of the body that are densely covered with hairs.

Talcum. Talcum is used with acute inflammatory processes that are not accompanied by oozing, and in the folds of the skin with chafing. Talcum applied to the skin absorbs moisture from the skin and increases surface evaporation by many times. This increases skin heat emission and, thus causes anti-inflammatory effect.

Creams. Creams are a dosage form consisting of fat mixed with water. Skin cooling creams are used in chronic inflammation.

Shacked-up mixtures *(«magma»).* Shacked-up mixtures that contain powdered substances (usually zinc) in oil or boric-alcohol solution are used for dermatitis, erythroderma, eczema etc. These mixtures dry quickly and requiring no bandaging. Before use, they are shaken and, then using a cotton or gauze, are applied to the affected area of the skin followed by talc or zinc oxide sprinkle. It is not allowed to apply these mixtures with abundant oozing, excessive dryness of skin lesions and localization of lesions on hairy areas.

Plaster. Plaster is a sticky ointment base with thick consistency covered with impermeable matter that may contain active drug substance. Plaster containing no drugs is used to fix dressings on the skin and in the treatment of venous ulcers.

Plaster containing drugs (mercury, salicylic acid, urea etc.) has an intensive deep tissue effect and is used for boils, onychomycosis, warts and bounded hyperkeratosis. Contraindications to the use of plaster are eczema, dermatitis and taxidermy. Before applying plaster the skin is thoroughly degreased with alcohol or medical gasoline; the hair should be shaved off. To fix the bandage, apply usual plaster. It is cut into strips of desired length and then applied so that the central part of the cut strip was pressed against the adhesive bandage, and ends – against degreased skin. If plaster badly sticks to the skin, it should be slightly warmed up.

As concerns complications, the application of plaster may cause dermatitis.

1. Which layer of European people epidermis contains melanocytes?

A. Translucent
B. Spinous
C. Basal
D. Horny
E. Granular

2. Sweat glands can be:

A. Eccrine
B. Border
C. Apocrine
D. Glomerular
E. All answers are correct

3. Which nerve receptors in the skin are responsible for the sensation of cold?

A. Krause's bulbs
B. Ruffini's corpuscles
C. Meissner's corpuscles
D. Pacinian corpuscles
E. Merkel's corpuscles

4. Which pathomorphological process in the skin causes appearance of bulla?

A. Parakeratosis
B. Acanthosis
C. Acantholysis
D. Hyperkeratosis
E. Spongiosis

5. What histopathological changes underlie the concept of «hyperkeratosis»?

A. Irregular proliferation of dermal papilla
B. Intensive growth of the granular layer of the epidermis
C. Thickening of horny layer of epidermis due to the enhanced keratinization and delayed rejection of horny scales
D. None of the aforesaid
E. Intensive growth of spinous layer of epidermis

6. What types of morphological elements constitute monomorphic rash?

A. One type of primary elements
B. One primary and several secondary types of elements
C. Several types of primary elements
D. Several types of secondary elements
E. Elements accompanied by skin defects

7. Clinically, a crust differs from the scale by the fact that it is:

A. Transparent
B. Microscopic
C. Thick and non-transparent
D. Appears as a result of evolution of hemorrhagic macules
E. Appears as a result of evolution of a nodule

8. Erosion is the second morphological elements that:

A. Epithelizes without traces
B. Remains scars
C. Appears as a result of evolution of pigmented macules
D. Appears as a result of scratching
E. Appears exclusively on the physiological lines of skin tension

9. What technique allows distinguishing between vascular inflammatory macules and other types of spots?

A. Angioscopy
B. Vitropression
C. Scratch test
D. Scraping
E. Brocq test

10. What diagnostic technique allows studying functional state of neurovascular system?

A. Diascopy
B. Angioscopy
C. Determination of dermographism nature
D. Luminescent diagnostics
E. Vitropression

Task 1. Pathomorphological examination of biopsy material taken from affected skin cells of a patient has revealed a change in the spinous layer of epidermis and the absence of intercellular connections.

a) What kind of histopathological processes is this?

A. Parakeratosis
B. Acanthosis
C. Hyperkeratosis
D. Spongiosis
E. Acantholysis

b) What morphological element of rash appears thereby?

Task 2. An individual papilla of skin biopsy material contains a structural element, which is an encapsulated nerve receptor and consists of the processes of modified cells of ciliary epithelium with sensitive cilia that are in contact with cell membrane of the nerve end of process. Body cells are separated from contact zone by capsule consisting of several longitudinally oriented glial cells. The cilia of sensitive cells are located between outer and inner capsule, thus contacting with inner surface of outer capsule.

a) What kind of nerve receptors is that?

A. Krause's bulbs
B. Ruffini's corpuscles
C. Meissner's corpuscles
D. Pacinian corpuscles
E. Merkel's corpuscles

b) What function the receptors perform?

Task 3. The patient's skin has a redness of a palm size, which disappears when applying diascopy.

a) What kind of element is that?

A. Telangiectasia
B. Roseola
C. Petechia
D. Chloasma
E. Erythema

b) How the described element is resolved?

Task 4. The patient's skin of upper and lower extremities is covered with numerous non-cavitary (infiltrated) rash elements with clear-cut boundaries of a coin size covered with scales.

a) What kind of elements is that?

A. Vesication
B. Tubercle
C. Nodule
D. Bulla
E. Papulle

b) Outline the stages of evolution of described rash element.

Task 5. The examination of the patient has revealed that skin is covered with translucent polygonal papules of various sizes with impression in the middle. Koebner's phenomenon is observed on the postoperative scar. The diagnosis is lichen planus.

a) What stage of diagnostics is that?

A. Morphological
B. Functional
C. Clinical
D. Anamnestic
E. Ethiopathogenetic

b) The setting of which diagnosis is finished with this type of diagnostic process?

Answers to the quiz of the first level of complexity
1 – C; 2 – E; 3 – A; 4 –C; 5 – C; 6 – A; 7 – C; 8 – A; 9 – B; 10 – C
Answers to the quiz of the second and third levels of complexity
1a – E; 2a – D; 3a – E; 4a – E; 5a – C

3 TOPIC Ectoparasite Infections

Ectoparasite Infestions – skin diseases caused by human and animal parasites. There are two groups of ectoparasite infections, these are diseases caused by the penetration of the parasites into the skin (scabies, demodecosis), and diseases caused by parasites bites (lice infestation) and insect stings.

TRAINING AND EDUCATIONAL PURPOSES

- To determine the ways and possible conditions of ectoparasites infection of the patients
- To explain general course and clinics of scabies
- To determine the clinical varieties of pediculosis
- To generalize the clinics of typical demodecosis manifestations
- To generalize the principles of therapy and prevention of skin infestation

3.1 Scabies

Scabies is a parasitic contagious skin infestation caused by the itch mite *Sarcoptes scabies.*

TO KNOW:

- etiological, pathogenetic and epidemiological features of scabies;
- clinical forms of scabies and peculiarities of disease course;
- diagnostic criteria of skin manifestation during scabies and its complications;
- notion of acarophobia and its manifestations which may arise due to post-scabies itch development;
- methods and principles of treatment, prophylaxis and clinical examination of scabies patients.

TO BE ABLE TO:

- correctly collect anamnesis of scabies patients;
- run laboratory diagnostics (traditional, express-diagnostics, by alkaline skin preparation method) in order detect itch mite;
- make a differential diagnostics in comparison with dermatoses which have similar clinical presentation picture;
- prescribe rational treatment for scabies patient and to correctly prescribe medicines for eternal therapy taking into account the patient's age;
- recommend the necessary preventive measures of scabies.

Etiology. Human scabies is caused by the itch mite *Sarcoptes scabiei* of *Sarcoptidae* family. The size of the male mite is 0.2 mm in length, 0.14-0.19 mm in width. Female is somewhat larger, it is 0.4-0.45 mm in length and 0.25-0.35 mm in width. Under the microscope itch mite is like turtle. After fertilization the male dies, and female burrows into the epilayer of the epidermis, where makes tunnels in parallel with the surface of the skin, in which it lays eggs. A mite can be removed from the itch tunnel with an injection needle or by means of thin sections of stratum corneum in the places of itch tunnels made by the female. For the period from one and a half to two months of its life, the female lays up to 50 eggs, of which three or four days later the mite larvae hatch. After two weeks, the mites become mature.

Epidemiology. Annually up to 300 million cases of scabies morbidity are registered in the world. Fluctuation of scabies morbidity is of sinuous character with rise and fall rates periods in 15-30 years. Mainly, morbidity rate increase is caused by the worsening of sanitary conditions, poverty, migrations, economic crisis, natural disasters, population crowding, especially during the wars. It is observed the parallelism of increase of scabies cases and diseases transmitted predominantly through sexual contact. The source of infection is ill person. The main way of disease transmission is family and domestic. The infection occurs through the direct contact with the sick person, through bed sheets, clothing, gloves, socks and other items that he used. The children often become infected through shared toys in the kindergarten.

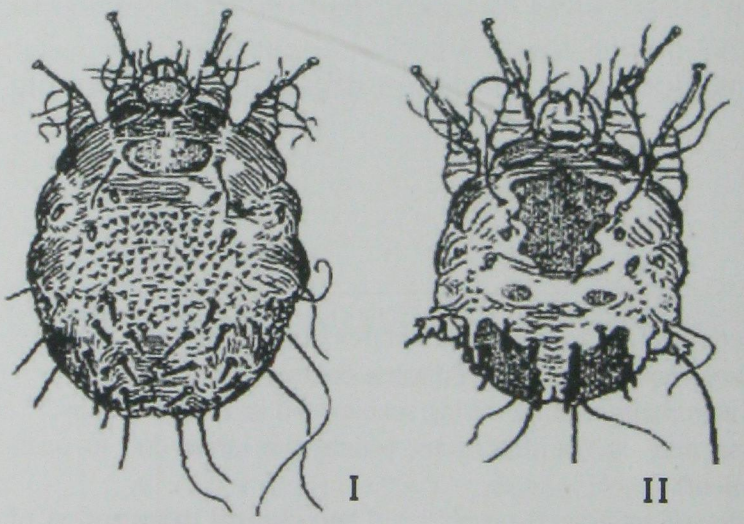


Fig. 3.1. Schematic representation of the itch mite:
I – female, II – male.

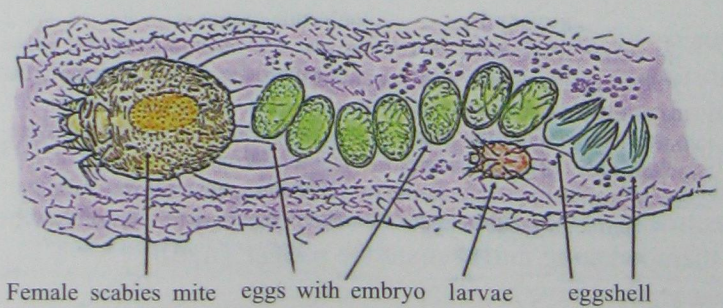


Fig. 3.2. Schematic representation of itch tunnel
in epidermis of the human skin.

Clinical picture. The clinic of scabies is based on the peculiarities of agent parasitizing, skin reaction and topical distribution of itch mites on the host. The incubatory period at the introduction of infection by the female is virtually absent, as it immediately borrows into the epilayer of the epidermis and starts to gnaw through tunnel and lay

eggs, that is, there is the main clinical symptom of the disease. At the introduction of infection by larvae one can talk about the incubation period, which corresponds to the period of larva transformation into the mature female (approximately two weeks). The clinical symptoms of the scabies are itching, which increases in the evening time, the presence of borrows, polymorphism of skin (papulovesicles, papules, scratches, bloody crust), characteristic localization of the rash.

The first subjective sign of the disease is intense itching in the places of borrows made by the female. The itching becomes worse in the evening and at night, that is due to the biological female characteristics, *(period of agent activity, salivation and substances present in the excrements; irritation of the nerve endings at movement of the itch)*, tunneling mainly at night, and the development of organism sensitization to parasites and their waste products. Itch can be localized and generalized. Its intensity varies with different people and depends upon the number of rashes and distribution process.

On the place of the female penetration into the skin a small papulovesicle (rarely papule) appears, which has infiltrative basis. At the distance of 2-7 mm from this element another papulovesicle appears (that is the exit site of the female onto the skin surface), these are so called conjugated freckles. Between the papulovesicles one can see itch tunnels, which have the form of thin strips of grey color, either straight or curved in the form of S letter. By means of light palpation one can find the tunnel relief, this is symptom Cezary. In the result of scratching there appear excoriations or small erosions with bloody crusts.

Typical places of scabies localization are areas of the hands between the fingers, flexor surface of the brachium and elbow joints, the front and rear edges of axillary cavities, lateral surface of the chest and abdomen, nipples areola breast of women, navel area, buttocks and inter-gluteal folds, internal surface of the femora and external genitalia, these are areas with thin stratum corneum. The symptom of Hardy-Gorchakov helps in the diagnostics; this is the presence of pustules (impetigo, rarely ecthyma) and purulent crusts on elbows. The eruptions in view of impetigenous elements, papulovesicles, crusts in the area of inter-gluteal fold skin with the transition to the lumbus, were called the triangle symptom or the rhomb of Michaelis. Scabies, as a rule, is not localized on the skin of the head, neck, back, axillary cavities, palms and plantae (except for children and persons with mental disabilities), that is an

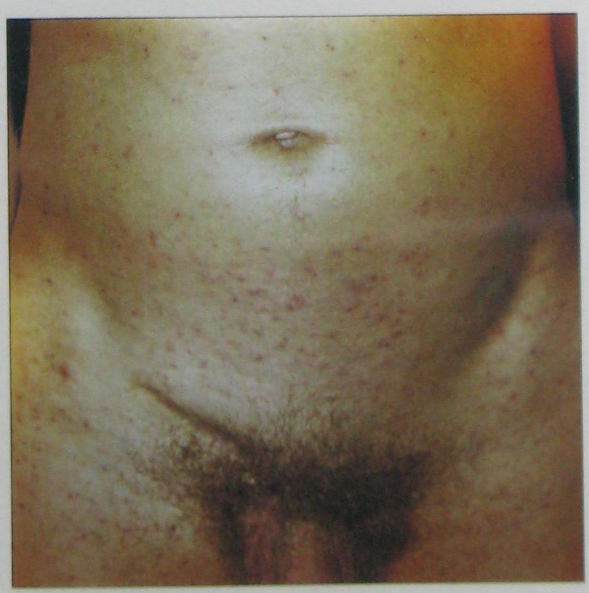


Fig. 3.3. Scabies.

important feature for the differential diagnostics with some other dermatoses. Clinical presentations of scabies on hand skin can be absent or slightly observable as to neat people and persons working with mineral oils, fuel oil (drivers, fitters, turners and others), turf and asphalt.

The peculiarities of clinical presentation of scabies nowadays are light itching and inconsiderable in number eruptions during continuous course. There can be urticarial elements, the absence of rash on the hands due to frequent contact with detergent agents, pastes, chemicals and others.

Scabies is often complicated by the secondary pyococcus infection in the results of the scratching (impetigo, ecthyma, folliculitis, boils), and by microbial eczema, especially in the area of breasts as for women. At eczematization there appears rash, which is typical to eczema; on the erythematous ground there appear small vesicles, oozing lesion, excoriations, and crusts. Eczematization, as a rule, develops as a result of irrational therapy or intolerance to some local agents (of brilliant green, benzyl benzoate, furacilin and others), less frequently as a result of sensitization of mite waste products.

Morbid anatomy. Histologically borrows look like tunnels in the horny layer of epidermis. Cephalic reminds a funnel and slightly opens outwards and the cavity is visualized in the caudal section of the borrow, where the female is situated. Small vasodilatations with a little cellular infiltration around them take place in papillary dermis. Sometimes acanthosis and spongiosis are observed in the epidermis.

Diagnostics. The diagnosis is based on the characteristic clinical picture of the disease and the identification of scabies mite in the laboratory research. With the traditional method the material for the study is obtained by means of the needle from the papulovesicles, located in the end of the borrow, or by means of superficial slice of the epidermis with a razor in the location of borrow. After putting

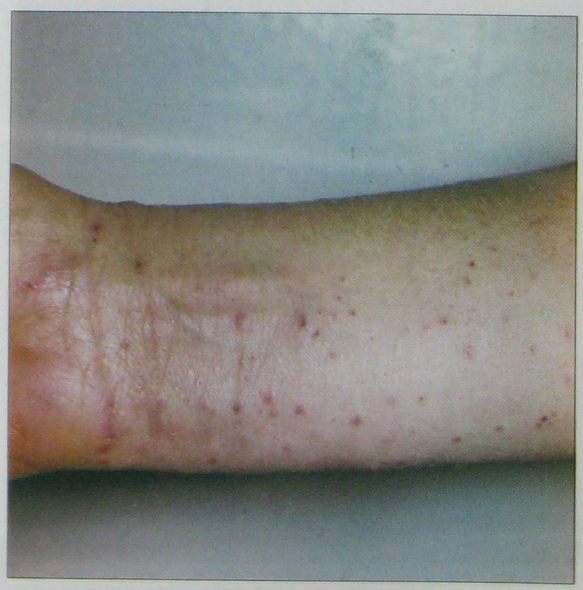


Fig. 3.4. Scabies.

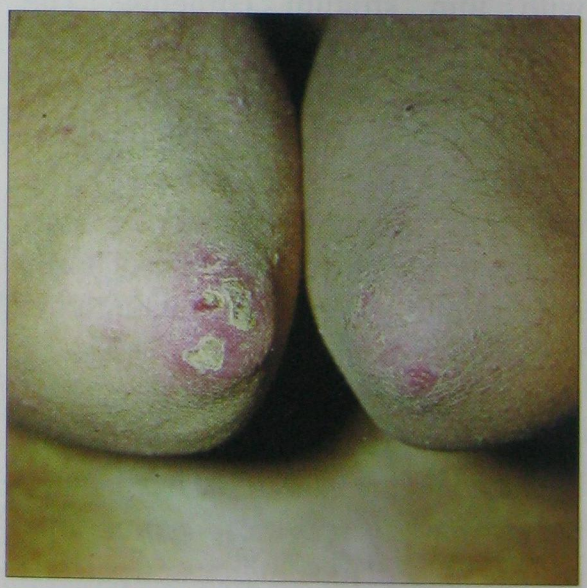


Fig. 3.5. Scabies. Symptom Hardy-Gorchakov.

the obtained material on a glass slide in a drop of 10-20% of alkali solution, the specimen is examined at law magnification under a light microscope.

Differential diagnostics. Most frequently it is necessary to differentiate scabies from skin prurigo, wherein intense itching and papular elements are observed as well. In contrast to scabies, in indicated disease itching worries patient both in the daytime and at night, and more often it takes priority of rash. The popular elements are not conjugated, are located randomly and can appear on face skin, the itch borrows are absent. Indirect evidence in favor of scabies can be itching and similar rash of family members of the patient.

Scabies affection of the penis skin, especially in the form of ecthyma, can be crucial for the suspected syphilitic solid chancre or popular rash, specific to the secondary period of syphilis. The presence of specific carnification at the heart of syphilitic solid chancre, the revealing of pale treponemes in serum of chancre or in lymph node aspirates, as well as the absence of scabies signs on other parts of the body and positive serological reactions allow diagnosing syphilis.

One type of neglected stage of scabies is so-called Norwegian scabies, which is observed in persons with mental illness and in patients suffering from syringomyelia, multiple sclerosis, leprosy. The skin at this time is covered with thick, as bark, crusts of dark green colour, resembling shell. Under the crusts after their removal on erythematous background a lot of white dots can be seen these are scabies mites.

Treatment. In order to kill off the scabies mites, local antiparasitic agents, disintegrating the cornel layer of epidermis and killing parasites, are applied. Appropriate antiparasitic agents as ointments, solutions and sprays are necessary to rub in all skin integuments, except for face and scalp, for adults. As infants and young children can have scabies manifestations on the face, scalp plantae and palms, accordingly, the indicated parts of the body subject to treatment with antiparasitic agents as well.

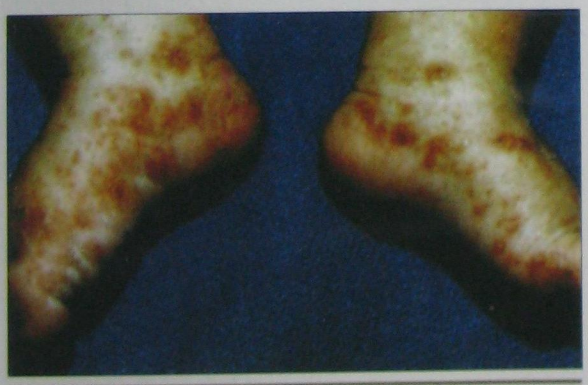


Fig. 3.6. Scabies in nursing infant.

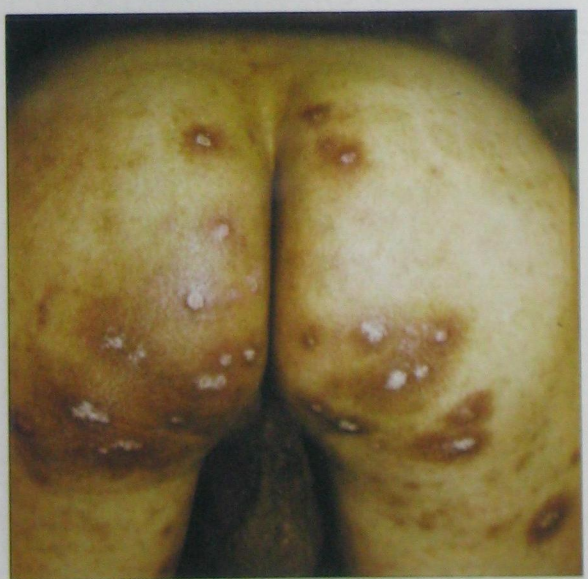


Fig. 3.7. Scabies complicated by pyoderma.

For scabies treatment benzyl benzoate is used. This method differs from the others in such a way that along with high efficiency, there is no unpleasant smell that allows the patient to be at work, in public transport etc. Benzyl benzoate (benzyl-benzene carboxylate in form of 20% suspension in soap solution: 20 g of benzyl benzoate, 2 g of green soap and 78 ml of water) is rubbed in with a cotton swab after that the patient puts on clean clothes and change linen. Infrictions are repeated during the period of two days more without washing. In three days after end of treatment the patient takes a shower and changes clothing. Nowadays, 25% benzyl benzoate cream is used more often than suspension.

The application of the above methods of therapy of scabies, taking into account the toxicity of antiparasitic medicines, is often accompanied by skin irritation (contact dermatitis). In this case, the patients are recommended desensitizing agents and antihistamines, zinc oxide powders, indifferent water magmas, steroid creams and ointments. In case of bacillosis overlay, antibiotics both oraly and topically are used temporarily, and lesion focuses are salved with aniline dyes and 2% salicyl alcohol, as well. If scabies is accompanied by eczema, then 24 hours before the antiscabious therapy, the affected area must be salved with one of the topic corticosteroids. The patients with constant itching after the treatment for a long time are recommended mitigatory and local anti-itching therapy.

Prophylaxis and antiepidemic measures for scabies patients are carried out accounting the epidemiology of disease. Early diagnostics and active case detections are of great importance here. Control of curability is executed in three days after the ending of therapy, and then in every ten days during a month and a half. The effectiveness of therapy mostly depends on thoroughness of sanitary and preventive measures.

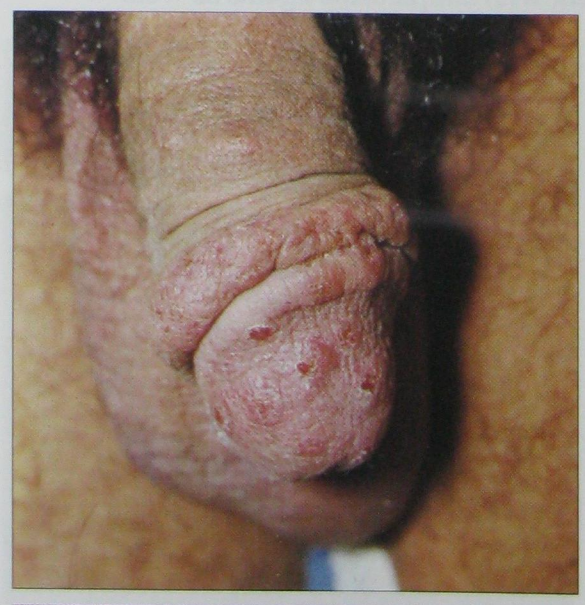


Fig. 3.8. Scabies manifestations in the area of glans.

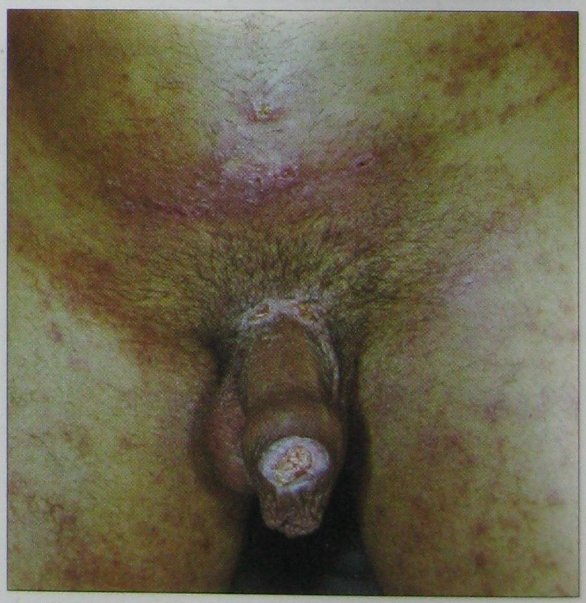


Fig. 3.9. Scabious ecthyma.

3.2 Demodecosis

Demodecosis is a parasitic skin disease caused by the mites-Demodex, these are *Demodex folliculorum* and *Demodex brevis.* Most of researchers consider that mites-Demodex live in the skin of healthy people, and only under certain conditions they reveal their pathogenic features.

Aetiopathogenesis. The mites-*Demodex folliculorum* and *Demodex brevis* of *Demodecidae* family. They are in the follicles and sebaceous glands of the face mainly, as well as of the ear and auditory canals, palpebrae, breasts and genitals. There are two species of Demodex, which parasitize on human body; these are *Demodex folliculorum* and *Demodex brevis*. Both species of the mites are very small in size (0.2–0.3 mm), with fusiform body, short legs and piercing-sucking mouthparts. The mites-Demodex causes illness under the common name demodecosis. More frequently these are people of young or middle age who suffer from a disease, especially in the presence of oily seborrhea. Factors contributing to the development of the disease are gastrointestinal and hormonal disorders, autonomic nervous disorders, menopause, dysmenorrhoea, and work in conditions of sharp change in temperature.

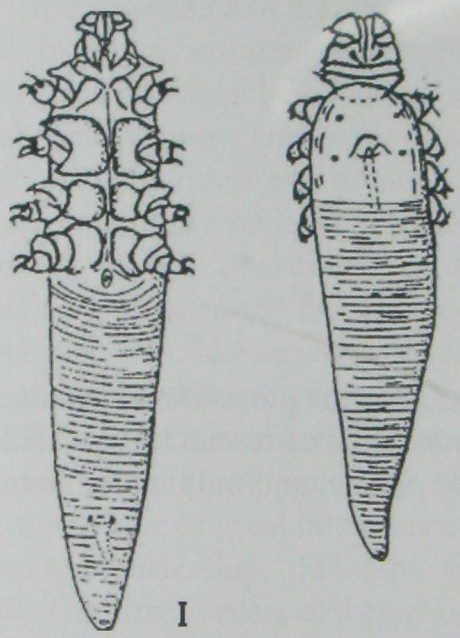


Fig. 3.10. Schematic presentation of the mite *Demodex folliculorum:* I – female, II – male.

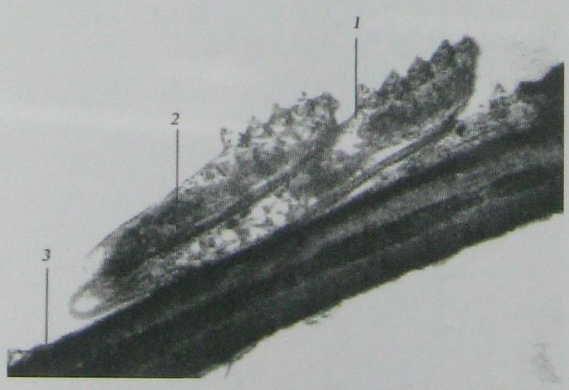


Fig. 3.11. Female and male of the mites-*Demodex folliculorum* in the hair follicle ostium of the eye lash hair:
1 – female, 2 – male, 3 – hair shaft.
Microscopy, 20X magnification.

Clinical picture. It predominantly involves the facial region (cheeks, forehead, and chin). There appear erythematous spots, telangiectasia, focal or fuse infiltration, peeling, as well as follicular papules of pink or red color, papulovesicles, papulopustules or separate macropustules. All these morphological manifestations of the demodecosis are accompanied by a number of subjective sensations, such as itching from minor to insufferable; sensation of skin heat and tightening; reduction of its elasticity and softness; sense of "screwing" something into the skin and "pins and needles" on its surface.

Nowadays there are several classifications of clinical forms of demodecosis. In particular, one defines erythematous-squamous, papular, pustular, rosacea-like, combined and oligosymptomatic forms. With erythematous-squamous form of demodecosis peeling secondary to skin redness is observed in the affected

areas. Disease, as a rule, occurs in spring and summer time. Its clinical symptoms exist up to two months, and then they gradually disappear or manifest less intensively. The relapse of disease occurs again in spring of the next year.

Papular form is the most prevalent form of demodecosis. Except the face, the other areas of the skin can also be affected, such as ears, neck, back and abdomen. The main features of this clinical form are popular or papulovesicular rash, always follicular. The sizes of the papules range from 0.5 to 2 mm in diameter. The color of the papules can be of different intensity from pink to intense red. Silver squamosa or microvesicles appear on the surface of some papules, which can transform into micropustules.

With the pustular form of demodecosis, the clinical picture resembles follicle, acne, rosacea-like or combined form of lesion, but with pustular rash dominating.

Clinical manifestations of rosacea-like form of demodecosis look like manifestations of true rosacea. The peculiarities of this form are stable erythema on face, mainly in the area of nose, as well as the presence of telangiectasia with periodic presentation of flare. An important differential diagnostic criterion of rosacea-like form of demodecosis, as distinct from true rosacea, is the discovery of a large number of the mites-*Demodex* in the lesion areas during the laboratory research. With this clinical form of demodecosis the postulation can be due to parasitizing and activity of the mites-*Demodex*, or the result of action of associated impetiginous bacterial population.

With combined form of demodecosis the rash elements appear which are characteristic for all other clinical forms. The combined form appears in the setting of any other clinical form of demodecosis, but it can develop also as independent form from the beginning of the sickness.

Oligosymptomatic form of demodecosis is characterized by the presence of erythematous spots, small follicular peeling, isolated small papulovesicles and vesicle-pustules.

Depending on the extension of inflammatory process the following forms of demodecosis are recommended to define: limited form with localization of the rash, mainly in the folds of the face skin and near the angles of the eyes; diffusive form which

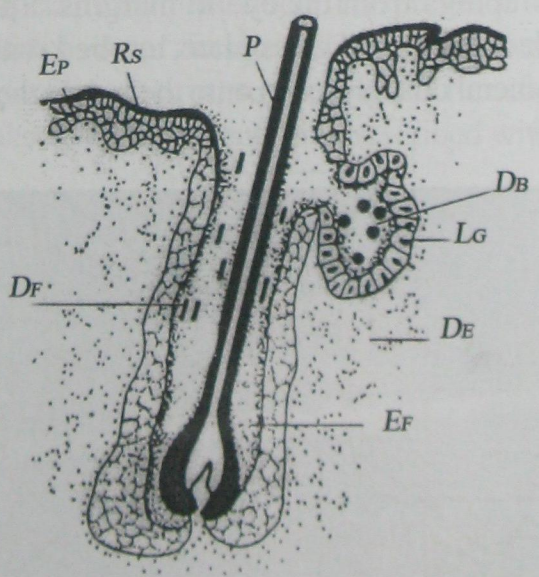


Fig. 3.12. Scheme of mites-*Demodex folliculorum* and mites-*Demodex brevis* localization in the hair follicle:
Df – mite-*Demodex folliculorum*, **Db** – mite-*Demodex brevis*, **De** – derma, **Ef** – follicle epithelium, **Ep** – epidermis, **Lg** – sebaceous gland, **Rs** – corneal layer of skin, **P** – hair.

affects the entire skin of the face; extensive form in which the inflammatory process extends beyond the face.

At the present stage it is also recommended to define the primary and secondary forms of demodecosis. The criterion of such dividing is that the primary demodecosis appears on the unchanged skin in appearance; and the secondary demodecosis is the complication of the principal disease, such as rosacea, perioral dermatitis and some other ones.

Diagnostics. The diagnostics of the demodecosis is in microscopic study of pathological material taken from the lesions with aim of detection of the mites-*Demodex*.

A variety of clinical forms of demodecosis defines specific material sampling for study for the presence of the mites-*Demodex*. With erythematous form of inflammatory process the material is taken from different areas of skin lesion by superficial scraping of squamosa. With papular, pustular and combined forms of demodecosis a puncture is made by means of scalpel and the content of pustules or sebaceous glands is received by extracting. It is also recommended to use comedone extractor or cyclodialysis spoon for extracting of follicle content. The obtained material is transferred onto the glass plate, treated with 20% solution of potassium hydroxide (kerosene, glycerol), covered with a cover slide, and examined under the microscope at low and high magnification in 10-20 minutes.

The laboratory diagnostics of demodecosis of the eyes is also based on the detection of the mites-*Demodex* in the content of hair follicles of the eye lashes or scrapings from the eyelid margins. Epilated 4-6 eyelashes from each eyelid are placed onto the glass plate, applied with 1-2 drops of clear liquid (water, plant or mineral oils, glycerin) onto them, and then covered with cover slide.

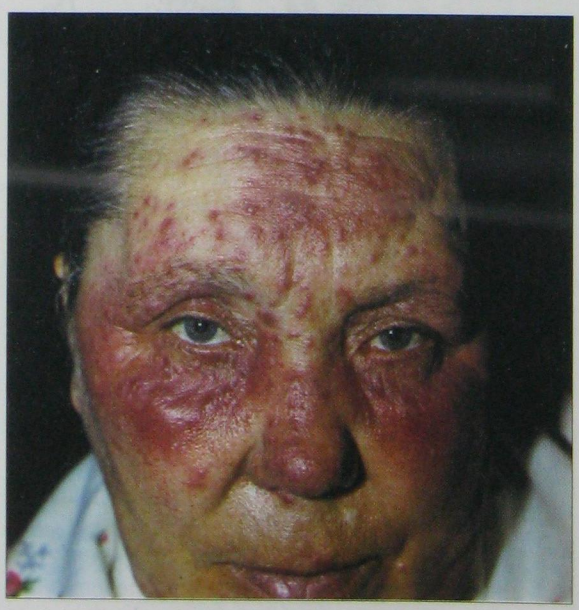


Fig. 3.13. Demodecosis.

It should be noted that up to the present moment there is no generally accepted methodology of collection of samples to determine the number of the mites-*Demodex* in a limited area of affected skin. Besides, the point of significance of identified number of the mites-*Demodex* for setting nosological diagnosis "demodecosis" remains to be debating.

Individual researchers recommend defining the number of the mites-*Demodex* in the affected skin area of 1 cm^2. In this case the detection of the mites-*Demodex* in an amount of more than 5 mites per 1 sm^2 is considered a diagnostic criterion for setting diagnosis

"demodecosis". A lower concentration of these parasites is regarded as carriage, which does not require antiparasitic therapy for the patient to be conducted.

At the same time, the other researchers to determine the diagnosis "demadecosis" offer to take into account the number of the mites-*Demodex (D. folliculorum, D. brevis)* in the structure of one hair follicle in the area of affected face skin, that methodologically is performed by means of histological examination. The detection of the mites-*Demodex* in an amount of 10 mites and more in the structure of one hair follicle is offered to regard as diagnostically significant for setting diagnosis "demodecosis". Herewith, during the quantitication both mature species of the parasites (imago) and the presence of the eggs, larvae and nymphs must be accounted.

Demodecosis should be differentiated from rosacea, acne vulgaris, seborrheic dermatitis, perioral dermatitis, bacterial folliculitis.

Treatment.

Taking into account the proven significance of a number of endogenous factors in the pathogenesis of demodecosis, it is recommended to hold a comprehensive examination of these patients with aim of defining foci of chronic infection, pathology of gastrointestinal tract, liver, pancreas, as well as endocrine and immune disorders.

Treatment of demodecosis requires a complex therapy with accounting of the stage of inflammatory process and presence of associated pathology.

In case of acute facial oedema and oozing lesion antihistamines and dehydration agents, as well as topical anti-inflammatory therapy are prescribed. The presence of pustular elements of rash requires prescription of one of the tetracycline antibiotics, in particular, doxycycline or macrolides (azithromycin) and others. After the elimination of oozing lesion and fresh pustular rash the patients are prescribed with topical antiparasitic therapy.

For the treatment metronidazole (oral) is used as well. Antiparasitic topical therapy can be conducted with use of 20% suspension of benzyl benzoate, 33% sulfur ointment.

The medicines of pyrethroid group, which are synthetic analogues of natural pyrethrins, known for their insecticidal and acricide action, are also used. Concerning appropriate medicines with synthetic pyrethroid-permethrin, it should be noted that the permethrin concentration of the preparation "Spregal" (spray) is higher in comparison with analogues. Besides, the preparation formula of "Spregal" consists of piperonyl butoxide, which contributes to prolongation and strengthening of permethrin action.

During the treatment the diet and avoiding of hot and spicy food is important. To normalize the metabolic processes the treatment of the revealed pathology of other organs and systems is recommended.

Prophylaxis. In order to prevent exacerbation of demodecosis prolonged sun exposure and excessive use of cosmetics should be avoided.

3.3 Pediculosis

Pediculosis *(pediculus)* is an infestation of the skin and hair, caused by the parasites of three kinds, these are head lice *(Pediculus capitis)*, body lice *(Pediculus corporis)* and pubic or crab lice *(Pediculus pubis)*.

Head lice *(Pediculus capitis)* are spread through direct head-to-head contact or due to sharing hair combs, hats and others. The size of the male is 2-3 mm, the female is 2.4-4 mm, and both are of grey color with black dots on the belly edges. Inhabiting the scalp, the female lays eggs (nits) of grayish-white color with 0.75-0.8 mm in length, which are attached to the hair with help of chitinous membranes. In neglected cases the lice can infest the eyebrows, mustache and beard. The nits are attached to the hair and can be seen as white spot formations.

Clinical picture. Due to bites of the lice, there occurs intense pruritus (itching), which causes the appearance of excoriations, covered with hemorrhagic crusts. Often due to pyococcus infection in lesions there appear sycoses, follicles, and impetigo vulgaris. Abscesses and eczematous foci may develop. In complication of the pediculosis with pyoderma, the hair in the affected areas is glued in form of thick bundles, so called mats, at the same time, the regional lymph nodes can increase and become painful. In the back of the head, especially of the girls and women with long hair, there appears grainy pyodermia *(impetigo granulata)*.

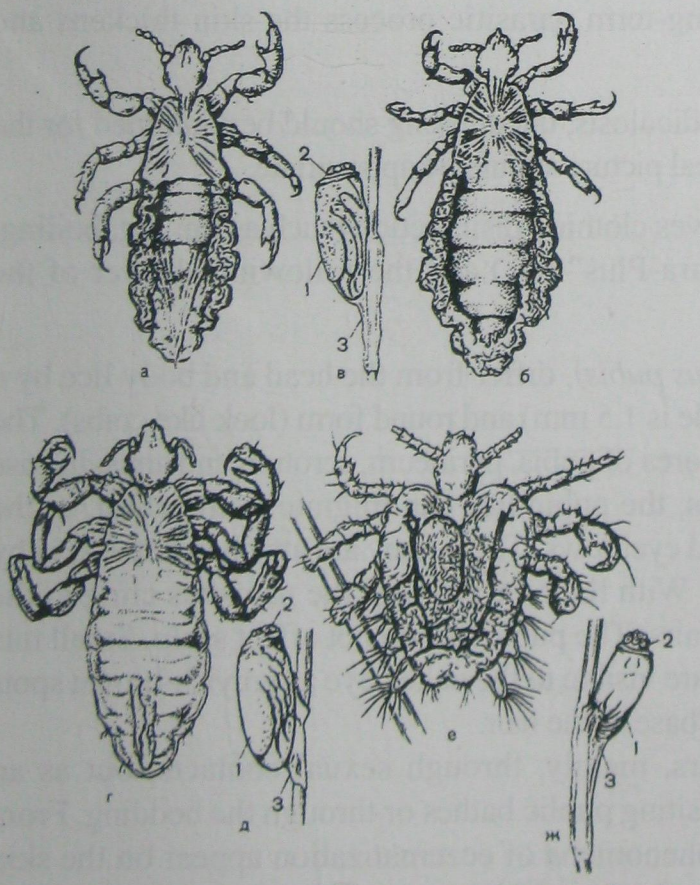


Fig. 3.14. Schematic presentation of the human lice:
a – head louse, male; *b* – female; *c* – nit (egg) of the head louse;
d – body louse, male; e – nit of the body louse;
f – pubic louse; *g* – nit of the pubic louse;
h – egg; 2 – tectorium; 3 – adhesive.

Diagnostics. To diagnose infestation, the scalp should be examined for the presence of the living parasites and nits.

Treatment. For the treatment of scalp pediculosis, 10% water-soap emulsions of benzyl benzoate and such shampoos as Pedilin, Nittiform are used, for treating the skin and hair of the head for 10-15 minutes, thereafter they are washed with hot water and soap. The use of Para-Plus spray is effective. The preparation is sprayed onto the scalp skin and the entire length of the hair; herewith the tip of the spray should be at a distance of 3 cm from the hair. After spraying the preparation it is necessary to wait for 10 minutes. During the exposure one should not cover a head with a towel or scarf. After use of preparation "Para-Plus" it is

necessary to thoroughly wash a head with a shampoo. The dead nits, remained on the hair, should be combed out. All textile products, which the patient contacted with, such as towels, pillows, hats and others, must be treated with the preparation "Para-Plus" to prevent the secondary infestation. In case of dermatitis or eczematization inflammations are eliminated with neutral lotions, which contain corticosteroids.

Body lice *(Pediculus corporis, seu vestimenti)* are rare nowadays on the territory of Ukraine; mainly people without a permanent residence have them. They are somewhat larger than head ones, with size 3-5 mm in length, of white color, without black spots on the abdomen. Lifetime of the parasites is 30-45 days; they well tolerate cold and can starve for several days, die immediately in boiling. Body lice carry saprotyphus and relapsing fever. Lice live and lay eggs in the folds of the clothes, especially in areas adjacent to the neck, shoulders and lower back.

Clinical picture. The lice bites cause intense itching; sometimes the eruption in form of urticarial elements can appear. In result of itching in the areas of lice parasitizing the scratches covered with bloody crusts appear, which are often complicated by the secondary pyococcus infections (impetigo, follicles, boils, abscesses), eczema. During long-term parasitic process the skin thickens and become dry (lichenification).

Diagnostics. To diagnose pediculosis, the dressing should be examined for the presence of the lice and the clinical picture should be appropriate.

Treatment. Treatment involves clothing disinfection (such as ironing, boiling, use of the spray "A-PAR", "Para-Plus" etc.) and the following shower of the patient.

Pubic or crab lice *(Pediculus pubis),* differ from the head and body lice by a smaller size (male is 1 mm, female is 1.5 mm) and round form (look like crabs). The places of parasitizing are mainly area of pubis, perineum, scrotum and anus. In case of people with excessive pilosis, the pubic lice can migrate to the chest, in the axillary cavities, on the beard and eyebrows. There, they are attached to the skin by means of craw-like formations. With their curved legs the parasites cling to the lower parts of the neighboring hairs. The pubic lice do not affect scalp. Small nits are found on the hair. Pubic lice are visible to the naked eye as greyish-brown spots with size of a pin's head near the base of the hair.

Pubic lice infestation occurs, mainly, through sexual contacts, but as an exception, it can occur during visiting public bathes or through the bedding. From time to time in pubic lice, the phenomena of eczematization appear on the skin (especially on the thighs) in the result of scratching and use of irritating solutions and ointments.

Clinical picture. Pubic lice cause intense itching with their bites, which is the reason of excoriation and bloody crusts appearance. Blue spots *(maculae*

cocruleae) with a diameter up to 1 cm of round or oval form appear in view of the line in the places of lice bites on skin of the abdomen, inner thighs and sides of the chest. The appearance of the spots is the result of the mixing of the patient's blood and lice saliva when biting. The blue spots do not disappear during diascopy, which is a significant sign in differential diagnostics from roseolous syphilides and saprotyphus.

Diagnostics. Pediculosis is determined on the basis of the presence of the parasites and nits, as well as blue spots, in the places of the bites on the skin of the abdomen, inner thighs and sides of the chest.

Treatment. Treatment of pubic pediculosis is in the removing of the hair from the affected areas and rubbing of the 33% sulfuric ointment, or 25% emulsion (cream) of benzyl benzoate during the following 3-4 days, after that the patient should take a shower with a soap and change clothes. Last time the sprays containing permethrin are successfully used for the treatment of the pubic pediculosis.

1. What agent causes scabies?

A. *Pediculus corporis*
B. *Sarcoptes scabiei*
C. *Demodex folliculorum*
D. *Pediculus capitis*
E. *Pityrosporum orbiculare*

2. What is incubation period for scabies?

A. 1–3 days
B. 3 months
C. 7–14 days
D. 2 months
E. 1 month

3. What is the character of eruptions in case of scabies?

A. Asymmetric-monomorphic
B. Asymmetric-polymorphic
C. Symmetric-monomorphic
D. Symmetric-polymorphic
E. Diffuse

4. Which of the following symptoms is uncharacteristic for scabies?

A. Sezary symptom
B. The triangle symptom or the rhomb of Michaelis
C. Hardy's symptom (Hardy-Gorchakov symptom)
D. Typical Hebra's borrow
E. Auspitz' symptom

5. What is not clinically typical for scabies?

A. Monomorphic character of rash
B. Presence of papulovesicular elements
C. Presence of itching borrows
D. Specific localization of rash (for adults and children)
E. Itching (in the evening, at night)

6. Rash elements in case of demodecosis are localized on?

A. Extensor surface of extremities
B. Flexor surface of extremities
C. Lumbus
D. Palms, plantae
E. Face

7. Which forms (stages) of disease are uncharacteristic for demodecosis?

A. *Rosacea erythematosis*
B. *Rosacea papulosa*
C. *Rosacea bullosa*
D. *Rosacea pustolosa*
E. *Rosacea hypertrophtica*

8. Scalp pediculosis is diagnosed on the following signs, except:

A. Excoriation
B. Itching
C. Eczematization
D. Secondary pyoderma
E. Presence of bullous elements

9. To treat pediculosis one uses:

A. Emulsion of benzyl benzoate
B. Celestoderm
C. Elocom
D. Herpevirum
E. Nizoral

10. To treat scabies all following preparations are used, except:

A. Benzyl benzoate
B. Solutions of sodium thiosulfate and hydrochloric acid
C. Common sulfur ointment
D. Permethrin
E. Aciclovir

Task 1. A man, who attended a medical appointment with a three-year-old son, is complaining that they both have skin itching which becomes worse at night. *Status localis*: there are eruptions in form of conjugated papulovesicles, a lot of crusts of the brown color and excoriations on the skin of the flexor surface of extremities, body, in the area of lumbus, on abdomen, but child also on the skin of the palms, plantae, face and the hair part of the head.

a) What diagnosis will you set?
- **A.** Scabies
- **B.** Allergic dermatitis
- **C.** Neurodermatitis
- **D.** Psoriasis
- **E.** Eczema

b) Call the methods of treatment and preventive measures of scabies for adults and children.

Task 2. A woman at the age of 35 is complaining of intense itching in the area of pubis and inguinal folds. She caught an illness a few days ago after work travel. *Status localis:* there are multiple scratches and hemorrhagic spots on the skin of abdomen (below), inner thighs.

a) Which diagnosis should be suspected?
- **A.** Scabies
- **B.** Pediculosis
- **C.** Eczema
- **D.** Epidermophytosis
- **E.** Dermatitis

b) Give the grounds for diagnosis.

Task 3. A teenager at the age of 13 has multiple conjugated papulovesicles, borrows, excoriations on the anterolateral surface of the trunk, in the lumbar area, on the buttocks, arms and in the area of radiocarpal joints. *Status localis*: skin itching at night. In view of the data of clinical and laboratory study, "scabies" is diagnosed.

a) Choose acaricide means for patient's treatment:
- **A.** 2% boric acid solution
- **B.** Aniline dyes
- **C.** 0.5% tannin solution
- **D.** 20% benzyl benzoate solution
- **E.** 6% hydrochloric acid solution

b) Call the specific clinical manifestations of scabies.

Task 4. A patient at the age of 50 is complaining about the eruption on the skin of nasolabial triangle and forehead, burning sensation and little itching in the rash areas. *Status localis:* there are papulopustular eruptions associated with erythema on the skin of nasolabial triangle and forehead. In the result of laboratory study the mite-*Demodex folliculorum* has been detected in the scrapings of rash elements.

a) Set the clinical diagnosis:
- **A.** Demodecosis
- **B.** Seborrheic dermatitis
- **C.** Erythema centrifugum
- **D.** Dermatitis
- **E.** Psoriasis

b) Conduct the differentiate diagnostics of the given disease.

Task 5. A patient at the age of 50 was admitted in gastroenterological department with gastritis and biliary dyskinesia. She was prescribed with adequate treatment. The patient was directed to the gastroenterologist for the consultation and treatment by the dermatologist, who set the diagnosis "scabies" to the patient after clinical and laboratory study.

a) Which laboratory study was executed to confirm the diagnosis?
- **A.** Identification of acantholytic cells
- **B.** Identification of *Sarcoptes scabiei*
- **C.** Identification of pathological fungi
- **D.** Identification of *Demodex folliculorum*
- **E.** Identification of *Pediculus vestimenta*

b) How is the laboratory diagnostics of demodecosis performed?

Answers to the quiz of the first level of complexity
1 – B; 2 – C; 3 – D; 4 – E; 5 – A; 6 – E; 7 – C; 8 – E; 9 – A; 10 – E

Answers to the quiz of the second and third levels of complexity
1a – A; 2a – B; 3a – D; 4a – A; 5a – D

4 TOPIC

Bacterial skin infections – pyoderma

Bacterial skin infections or pyodermae are a group of diseases, caused by microorganisms inducing purulent inflammation of skin.

TRAINING AND EDUCATIONAL PURPOSES

- To determine the ways and possible conditions of infection by pyogenic flora
- To generalize classification and general characteristics of pyogenic lesions of skin and mucous membranes
- To explain the role of different factors promoting its development
- To determine the general course and clinic of pyoderma
- To distinguish peculiarities of pyogenic lesions of mucous membranes
- To distinguish characteristic clinical peculiarities of staphyloderma
- To classify typical manifestations of streptoderma
- To define the principles of therapy and prophylaxis of pyogenic lesions

TO KNOW:

- modern views on etiology and pathogenesis of different clinical types of pyoderma;
- factors promoting developments and progression of pyoderma;
- principle of classification of impetiginous lesions of skin and its appendages;
- symptomatology of the main clinical types of pyoderma;
- the main approaches to the general and local treatment of pyoderma;
- the peculiarities of preventive measures of impetiginous lesions of the skin and its appendages.

TO BE ABLE TO:

- correctly collect anamnesis and carry out examination of the patient with pyoderma;
- run diagnostic tests and use additional examination methods, which confirm the diagnosis;
- make a differential diagnostics with the diseases with the similar clinical presentation picture;
- make a plan of recommendations for treatment and prophylaxis of pyoderma patients.

Epidemiology, etiology, pathogenesis of pyoderma

Epidemiology. In economically developed countries the patients with pyoderma compose 1/3 of patients, suffering from infectious diseases. The morbidity rate in children is higher than in adults, it composes 25-60% of the total number of cases of dermatoses. Pyoderma is the most common among the worker of such industries as metalworking, metal mining, coal mining, timber manufacturing, transportation, and various branches of agriculture as well.

Etiology. Most frequently the agents of pyoderma are staphylococcus and streptococcus. Different types of pustular skin diseases can occur initially as separate nosologic entities or as a complication of other dermatoses (scabies, eczema, atopic dermatitis etc.).

Staphylococci under the microscope have got rather correct round shape (their accumulations are often similar to a bunch of grapes) with a diameter of about 0.8-0.9 microns. The most virulent is Staphylococcus aureus. Staphylococci are also presented in form of spherical formations, joining in long chains. The diameter of one coccus varies from 0.5 to1 micron. Pycocci occur on the skin in the form of avirulent microorganisms in 90-92% of healthy people, and it is possible to detect their pathogenic forms only in 8-10% of population. Saprophytes can acquire pathogenicity under certain conditions and their virulence can increase under the action of alkaline reaction of the skin or in case of joining of other agents, such as fungi.

In case of Gram-stained pus smear, both staphylococci and streptococci are well stained in blue that means they are Gram-positive. The toxins, released by pyococcy, are highly toxic and are capable to lyse erythrocytes, leukocytes.

Pathonesis. Virulence of pyococci plays an important role in the occurrence of pyoderma. A number of factors, such as the acid reaction of the horny layer of epidermis, sebaceous glands, enzymatic activity of the skin etc., counteract the increased virulence of staphylococci and streptococci. A number of exogenous and endogenous factors contribute to the development of pustular lesions of skin. The most frequent exogenous factors include the excessive skin contamination with gasoline, oil, dust particles (coal, cement and other), micro injuries (insect bites, excoriations, needlesticks and other), maceration of horny layer (long dish-washing, doing laundry), hypothermia and hyperthermia.

The endogenous factors, contributing to the development of pyoderma, are low-calorie food, hypovitaminosis, chronic debilitating diseases, intoxications (alcoholism, narcomania), physical and nervous strains, diabetes, immunodeficiency etc.

There is no innate immunity against the pyococcus infections, but unstable infectious immunity can appear in the course of pyoderma, the intensity of which varies in wide range. The frequent for pustular diseases are the allergic reactions appearing in the result of sensitization to metabolic products of their agents, whereof the positive intracutaneous tests with the corresponding allergens (vaccines) indirectly testify.

The character of pyoderma development in clinical respect depends on the place of influence of agent. Staphylococci more often affect hair follicles, whereas streptococci mainly parasitize on the smooth skin. Favorite localization of panaritium, for example, is periungual walls, of ectymas is shins, furuncles and carbuncles are more common on the buttocks, lower back etc.

Besides pyococci, the pustular skin lesions can be caused also by collibacillus, proteus vulgaris, fungi, pseudomonas aeruginosa, pneumococci etc.

Depending on the agent, pyodermae are divided into staphylococcus, streptococcus and mixed; depending on the deepness of lesion they can be superficial and deep, on the character of the course they are acute and chronic, on the origin they are primary and secondary.

Staphylococcal skin infections

Staphylodermae are characterized by the development of inflammatory process, mainly in the area of appendages location, such as cutaneous and hair follicles, sebaceous and sweat glands. As a rule, a hair or opening of sebaceous gland is in the center of pustules. Much less frequently, staphylococci cause the lesions of the surface layers the smooth skin that is mainly observed in children. Newborns and infants have got the connection of epidermis with derma insufficiently developed due to the weakness of the basal membrane and dermal papillae smoothness that is why during the staphylococci penetration, the morphological elements such as bubbles and phlyctenas develop.

The following forms of staphylodermae are distinguished; these are surface – ostiofolliculitis and deep – folliculitis, furuncle, carbuncle, hydradenitis, sycosis, vesicle-pustulosis in children, neonatal impetigo, multiple abscesses of children (pseudofurunculosis), exfoliative dermatitis, and neonatal bullous impetigo.

Ostiofolliculitis is characterized by the appearance of small pustule with the size of grain of millet or pinhead, of greenish-yellow or milky-white colour, of spherical shape, surrounded by acute inflammatory hyperemic circle. A pustule is localized in hair follicle and pierced with unaltered hair in the center. The accumulation of pustules is observed in a small area of skin; they do not increase due to peripheral enlargement and do not merge. The process is most often localized on the skin of face, neck, forearms, lower legs, hips. The patients feel little itching. The pustules shrivel forming greyish-yellow crusts in 4-8 days. After crusts falling, insignificant pigmentation remains on the skin, which soon disappears. Ostiofolliculitis appear under the influence of minor skin irritations such as shaving, friction, maceration due to excessive sweating.

In some cases, certain ostiofolliculitis can grow at periphery or deathward, becoming deep folliculitis, furuncles and carbuncles. Growing at periphery ostiofolliculitis eventually reaches the size of a pea, especially in children and scabies patients with wrists lesion. These ostiofolliculitis are called ***Bockhart's impetigo.***

The diagnosis of ostiofolliculitis is set in presence of small tight conical pustules, in their center pierced with a hair and surrounded by hyperemic circle.

Hystologically, a small cavity, bounded above by the horny layer, and bounded below by the callous cells of epidermis, is located in the ostium of the hair follicle. The cavity is filled by the conglomerate mass of polymorphonuclear leukocytes, some lymphocytes, and staphylococci as well. An edema appears around the hair follicle in the derma and capillaries broaden, around which perivascular infiltrate from lymphoid and polymorphonuclear cells is observed.

For the treatment of ostiofolliculitis, the pustule is pierced with a sterile needle, the tectum and pus is removed, the affected areas are anointed by the 1–2% alcohol solutions of aniline dyes (methylene blue, brilliant green, Castellani liquid). After that the skin is wiped with 2% salicylic or boric spirits and sprinkled with 5-10% of boric powder. With large crusts the affected surface is salved with antibiotic ointments (Fusiderm, Bactroban, Altargo, neomycin, tetracycline, and erythromycin). The topical application of Zineryt lotion (erythromycin zinc complex), OXI, Ugrinum lotions, and Diacneal cream is also effective.

Profound folliculitis *(folliculitis profunda)* is a purulent inflammation of the entire hair follicle and the adjacent adipose tissue, arising in the result of pathogenic staphylococci penetration in the depth of follicle. Initially a painful red papule (nud) of size from a pea to a cherry appears on the skin around the ostium of hair follicle. A hair is located in the center of a papule. In a few days, the papules tauten, becoming a follicular pustule of conic form with base induration. A pustule dries out after some time forming the crust, in some cases connective tissue necrosis and intense suppuration can occur. A pustule in the center ulcerates gathering to matter of greenish-yellow or white colour. Profound folliculitis remains pigmented scar. The reasons for the appearance of profound folliculitis are the same as for ostiofolliculitis.

Hystologically, in profound folliculitis there is infiltrate consisting of neutrophils and lymphocytes around the hair follicle. A hair follicle melts itself, and a cavity filled with matter, forms in its place.

The treatment of folliculitis is the same as of ostiofolliculitis.

Furuncle *(furunculus)* is acute purulonecrotic inflammation of hair follicle and its surrounding connective tissue, caused by pathogenic staphylococci. A furuncle develops in the presence of low immunological reactivity of organism. The contributing factors in furuncle appearing are the cutaneous

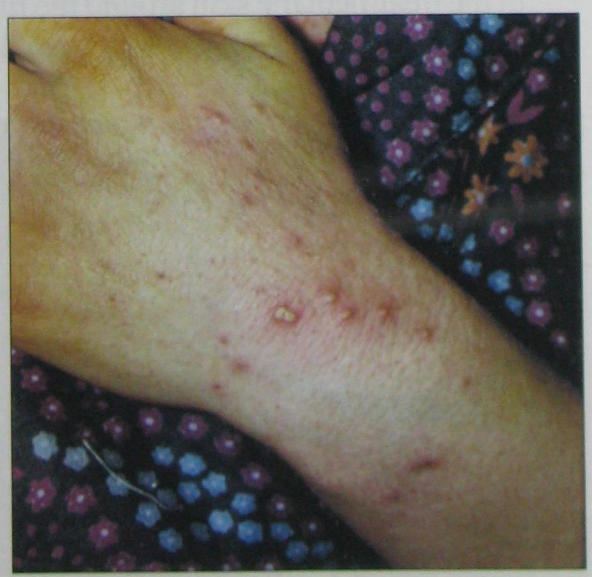


Fig. 4.1. Ostiofolliculitis.

injuries, scratching of allegrodermatosis, catarrhal and infectious diseases, vitamin deficiencies, hypothermia and other. A typical localization of furuncle is the areas of friction between skin and clothes, such as neck, lower back and buttocks.

The symptoms of furuncle develop gradually. In the majority cases, the process develops in the setting of ostiofolliculitis, which spreading depthward, leads to the formation of the node of acute inflammatory character. During the period of 5 to 7 days the furuncle becomes soft in the center, the fluctuation appears. The infiltrate ulcerates discharging matter prolifically. In the center of the burst there appears necrotic tissue of green colour (necrotic core). After its separation there is a deep crater-like ulcer. The ulcer bottom is covered with granulations, gradually discharging from the matter, thus the ulcer cicatrizes.

The development of the furuncle is accompanied by the pains and burning sensation, and sometimes high temperature.

In the case of relapses of furuncle several times in different places they say about the chronic furunculosis. The development of furunculosis is promoted by the dysfunction of internal organs and nerve system, depletion of immunological reactivity, anemia, diabetes, infectious diseases, hypo and avitaminosis, the presence of chronic foci of infection, the use of corticosteroids and other. In children the furunculosis develops with gastro-intestinal disorders, hypotrophy, and rickets.

The typical clinical picture is a massive infiltration, conical shape, redness, pain, the presence of a hair and necrotic core in the center enables quite easy to set a diagnosis of furuncle.

Hystologically, a massive infiltration, consisting of polymorphonuclear leukocytes, lymphocytes, fibroblasts and a small number of plasma cells, forms in derma and subcutaneous fat. Collagen and elastic fibers break down completely with hair follicle. In the focus of lesion, a massive argentophil grid appears which forms the membrane, impeding penetration of infection from the focus into the patient organism.

In the treatment the skin around the furuncle is disinfected with alcohol or ether, after that a hair is pulled out gently from the center of infiltrate with a sterile forceps, pure ichthyol is put on the furuncle, covering it with a thin layer of cotton wool. The procedure is repeated twice a day, until in the center of the follicle the opening forms, on which the gauze folded in several times and moistened with hypertonic solution of sodium chloride is put. After discharge of the core, the ointment with antimicrobial effect and stimulating tissue regeneration is

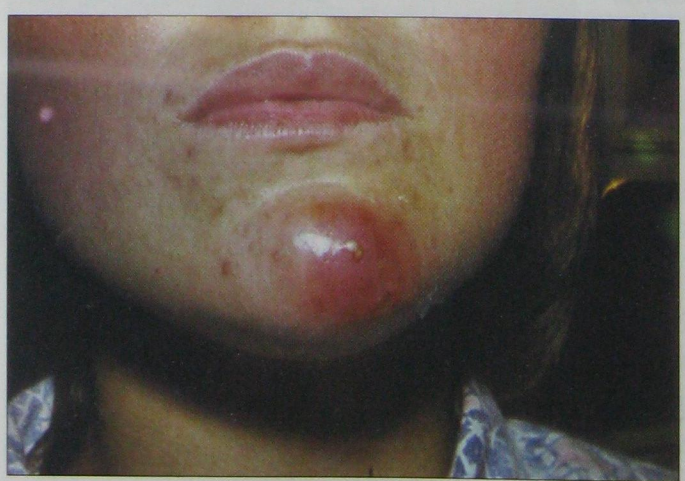


Fig. 4.2. Furuncle.

applied, such as (Fusiderm, Bactroban, Altargo), and skin around the furuncle is wiped with 2% boric and salicylic spirits.

In the case of multiple furuncles, furuncles in the area of face, hairy part of the head, as well as chronic relapsing furunculosis, antibiotic therapy should be applied in order to prevent the occurrence of septicophlebitis of cerebral vessels and general sepsis. Currently, it is recommended to use the broad spectrum antibiotics from the group of cephalosporins, tetracyclines, macrolides etc. in adequate doses during 7 to 10 days with an obligatory determination of pyococcus sensitivity to antibiotics. In severe cases the antibiotics are administrated parenterally. Concurrently antihistamines, such as suprastin, cetrine, claritine, aerius etc. are prescribed.

Carbuncle *(carbunculus)* represents a severe inflammatory process, covering several hair follicles, sebaceous glands and subcutaneous fat. As a rule, a carbuncle develops in the result of dissemination of purulent process with numerous profound folliculitis or in conjugating of close furuncles that leads to extensive skin necrosis. Carbuncle is caused by the pathogenic stains of staphylococci.

Endogenous factors first of all play an important role in the development of carbuncles.

The general condition of the carbuncle patient is usually compromised, there is high temperature, headache etc.

Carbuncles are most often localized on the skin of hindhead, back, lumbus, i.e. in the places of friction and irritation of the skin and its frequent pollution.

At the beginning of the process there are several openings on the skin surface above infiltrate, and infiltrate is surrounded by edema. After the cores separation and intense discharging of matter, mixed with blood, necrotic mass of green colour can be seen on the infiltration site. After purification of the openings from the necrotic masses, there appear deep ulcers, sometimes reaching up to fasciae or muscles. In the result of filling the ulcers with granulations, their cicatrization occurs, and the process ends with formation of retracted stellar scars.

In advanced age, as well as with exhaustion, neuropsychic defatigation and diabetes, the course of the disease acquires malignancy, neuralgic pains and deliration appear and sepsis and erysipelatous inflammation can occur.

Hystologically, in carbuncle there is deep necrosis of all layers of derma and subcutaneous fat. Around the necrotic area there is a massive infiltrate, consisting of neutrophils and a small number of lymphocytes.

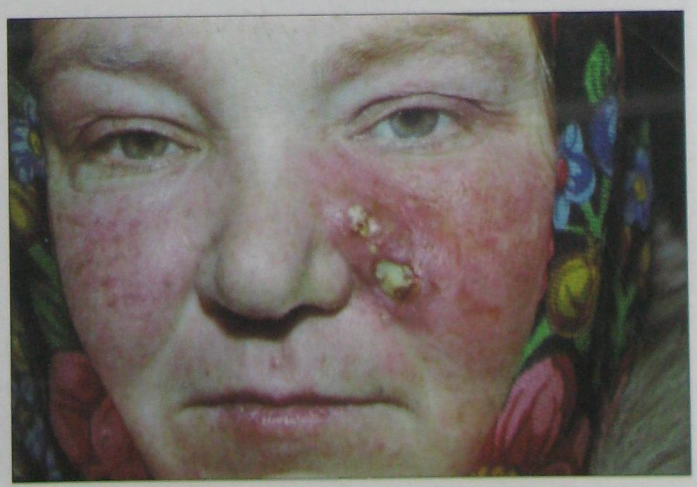


Fig. 4.3. Carbuncle.

Carbuncle treatment requires an obligatory use of antibiotics and other systemic drugs, so as in treatment of severe forms of furuncle and chronic furunculosis. A topical treatment is the same as in furuncle.

Hydratenitis is a purulent inflammation of apocrine sweat glands, located in axillary cavities, on labia majora, mammillae, anal area. It is caused predominantly by staphylococcus aureus. The contributing factor of development of hydratenitis is excessive sweating in axillary folds and perineum, consumption as a result of hypotrophy, infectious, nervous and endocrine diseases. From the external factors, the significant are excessive contamination of skin, microtraumas and cuts resulting from shaving hair in the axillary cavities.

Hydratenitis in children is observed only in senior age, when apocrine sweat glands attain full growth and begin to function (period of puberty).

Hydratenitis develops gradually. At the beginning the feeling of discomfort and insignificant soreness appear at the sites of localization of apocrine glands. The skin is not changed, and in palpation it is possible to detect small indurated formations. Later the skin at the affected sites becomes red, painful pea-sized nodes begin to extrude, which rapidly increasing reach the size of pigeon egg. Sometimes separate nodes can merge. In one to two days the nodes acquire soft consistency and are perforated with formation of openings, from which a large amount of matter is discharged. Gradually, the abscess cavity is filled with granulations, and the process ends with formation of retracted scar.

Maturation of hydratenitis is accompanied by high temperature, severe pains and general weakness. Disease can become chronic, when new nodes are developing one by one. The relapses are especially frequent in people with heavily pronounced sweating, insufficiently observing hygiene. The relapses occur mainly in summer in hot period.

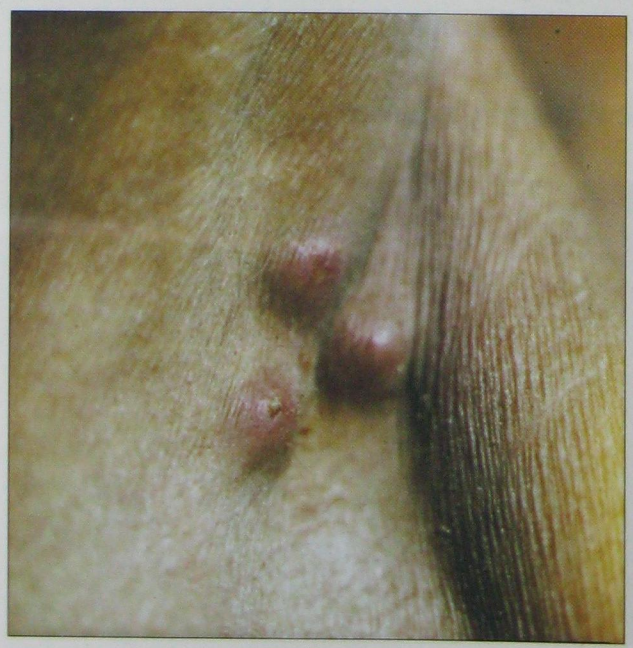


Fig. 4.4. Hydratenitis.

Hystologically, the pathological process in hydratenitis develops in the deep layers of the skin around the body and excretory ducts of apocrine gland, where there is an infiltrate from neutrophilic leukocytes and purulent fusion of sweat gland. The profound destructive changes of vessels occur in the center of necrosis.

The disease is so typical by localization and clinical picture, that the diagnosis is set without much difficulty.

The treatment does not differ from the similar treatment of

numerous furuncle, furunculosis and carbuncle; it includes timely application of antibiotics, immunomodulators, local and symptomatic agents.

Sycosis *(sycosis vulgaris, sycosis simplex, sycosis staphylogenes)* is a chronic staphylococcal skin lesion of face in the area of beard and mustache. Sycosis is observed almost exclusively in men. It can rather rare affect also internal surface of nose, eyebrows and pubic area. It is characterized by chronic course, relapsing eruptions of ostiofollicular pustules on the indicated areas of the skin. Contributing factors in the development of sycosis are integrity violation of epidermis, chronic rhinitis, nervous and endocrine diseases, in the first place, hypofunction of testes.

The development of sycosis begins with the appearance on the skin in the area of beard and mustache of small pustules the size of a millet grain, placed in the follicle ostium. In a few days, the purulent process affects the entire hair follicle (profound folliculitis). Gradually the skin of the affected site infiltrates, gets bluish discoloration, is covered with mixed crusts, swells. The site of the lesion focus enlarges peripherally due to the formation of new folliculitis. When pressing on the infiltrate, droplets of yellow green pus are discharged from the extended hair follicles.

After dropout of crusts, at the sites of lesions small erosions and ulcers appear, from which seropurulent liquid is released.

The general state of health of sycosis patients is not altered, sometimes the patients can complain about light itching, burning sensation and insignificant sickliness. Prolong course of the disease and its localization on the open skin area sometimes leads to oppression of mental state of patients.

Sycosis is rather easy to diagnose on basis of typical localization of lesion areas, chronic course, and presence of infiltrate with follicular pustules. Parasitic sycosis (profound barber's rash) differs by more severe course; the pathogenic fungi are detected during laboratory testing.

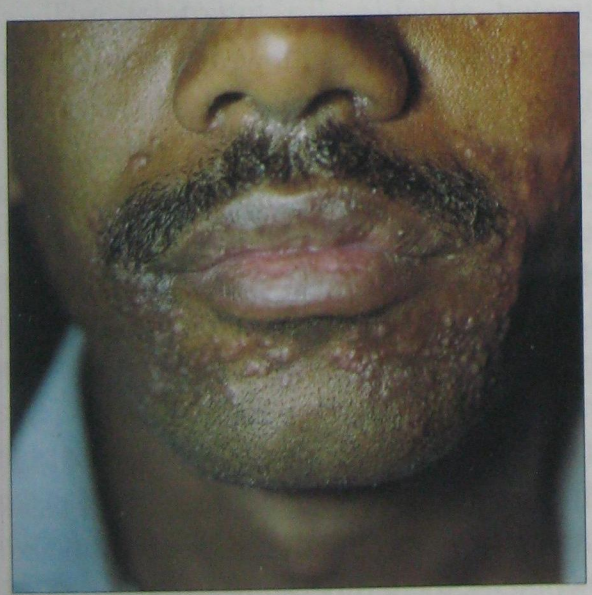


Fig. 4.5. Sycosis staphylococcal.

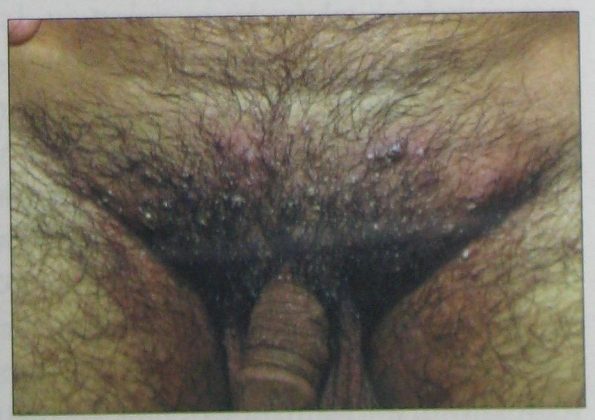


Fig. 4.6. Sycosis staphylococcal in pubic region.

Hystologically, in the area of hair follicle, the abscesses appear, filled with matter, the infiltrate consists of polymorphonuclear leukocytes and fibroblasts, a small number of lymphocytes and plasma cells. The edema, degeneration of hair follicle and surrounding connective tissue is observed in epidermis and derma.

In the acute period of sycosis development the broad spectrum antibiotics are administrated, including tetracyclines or macrolides (azithromycin, doxycyclinum, tetracyclinum etc.).

The topical therapy of sycosis should be started with removal of all crusts from the surface under purulent process, by softening them with plant oil, then the lotions of 1% solution of resorcinol, 0.1% solution of rivanol or furacilin are put on. The affected hairs are tweezed, and the surrounding tissues are wiped daily with 70% ethyl alcohol. Later, the antibiotic and corticosteroid ointments are applied (Fusiderm, Oxycortum, Betaderm, Bactroban, Flucinar N).

Vesiculopustulosis in children is a widespread purulent disease, which appears in the first years of life. In the ostium of the sweat glands numerous pustules appear, filled with white yellow matter, the size of a pin head to a small pea, they do not merge with each other and are surrounded by bright edematous circle. Vesiculopustulosis is localized at the sites of the greatest sweating and skin maceration. Premature infants of asthenic constitution mainly suffer from disease.

Diagnosis is set on grounds of typical clinical presentation and process localization. The disease should be differentiated from scabies, in which the papulovesicles are paired.

Epidemic neonatal pemphigus *(pemphigus neonatorum epidemicus)* is a cute infectious disease, differentiating from other pyodermae by very high contagiousness. Often the infection is transmitted to children from the adults (especially from medical personnel), suffering from pyodermae or quinsy, or through household articles. Sometimes, there are epidemic outbreaks of neonatal pemphius in maternity hospitals or day nursery.

The disease appears in 7-10 days after the birth. In the setting of erythematous patches, the blisters the size of pea are forming, filled with serous contents, they are rapidly increasing in the periphery, reaching the size of nut and becoming less stressed. The content changes from serous into purulent.

The blisters can be placed over the entire the skin cover, especially often on the abdomen near the umbilicus, on the buttocks, hips, back, chest and extremities. They rather quickly go into wet erosions, on the periphery of which the remains of blisters i.e. scraps of epidermis overhang. Drying up, the erosions do not leave crusts and regress with the formation of pink-brown pigment spots without scars.

The general condition of the patients is not altered; the disease mostly lasts 4 to 5 weeks. The process in weakened children can rapidly spread, covering new skin areas by autoinoculation (infection transmission from affected skin areas to the health). Children condition significantly worsens, the temperature rises up to 38–39°C, dyspeptic phenomena join. As concerns blood there are leucocytosis,

eosinophilia, increased ESR. In some cases the disease can be complicated by conjunctivitis, otitis and even sepsis that sometimes lead to the death of a child.

Exfoliative dermatitis *(dermatitis exfoliativa)* presents especially severe form of epidemic neonatal pemphigus. The disease begins with prodromes, such as nausea and temperature rise. Bright erythema appears in the folds of skin, around the mouth, umbilicus, anus and genitalia. In the setting of erythema there appear rather big tense blisters, which rapidly erode. In exfoliative dermatitis the positive Nikolsky's sign is observed, i.e. in case of friction of skin at the visible-healthy sites, it flakes off, forming the erosions; when pulling the scraps of blister with the tweezers, the epidermis exfoliates on the surrounding skin areas (the presence of acantholysis). The disease is accompanied by high temperature, dyspeptic phenomena. For several days, the process affects the entire skin cover, and sepsis develops, often with fatal outcome.

The diagnosis of epidemic neonatal pemphigus is set on the basis of appearance in children on erythematous acute inflammatory background of stressed blisters, which rapidly erode. The disease is necessary to differentiate mainly from syphilitic neonatal pemphigus and congenital epidermolysis bullosa. Both of these diseases are already observed at childbirth, when epidemic neonatal pemphigus develops only on the 7^{th} to 10^{th} day after the birth. In case of syphilitic pemphigus, the blisters affect the skin of palms and feet, which is not observed in case of epidemic pemphigus. In case of congenital syphilis in children it is possible to observe at one time syphilitic rhinitis, diffuse popular infiltration of Hochsinger, osteochondritis, and in the process of microscopy of blisters content in the dark field of view there are a large number of causative agents of syphilis such as pale treponems, classic serological reactions of blood, treponemal immobilization test and fluorescent antibody test are strong positive. Congenital epidermolysis bullosa is characterized by intrauterine appearance of blisters, which occur most frequently at the sites of birth injuries, such as hairy part of the head, buttocks, upper and lower extremities.

The treatment of epidemic neonatal pemphigus involves high-priority administration of antibiotic therapy in order to prevent complications and development of sepsis.

Multiple abscesses in children *(abscessus multiplex infantum),* or ***pseudofurunculosis***, is observed in neonates and infants. The disease begins with the appearance of superficial pustules in the ostium of sweat glands (periporihis). The agent is staphylococcus aureus, which penetrates in the depth of the sweat gland and causes the forming of deep indurated painful nodules. The skin over the nodules is not altered at the beginning, but soon it acquires reddish-brown color. The nodules the size of a pea to a nut soon suppurate and dense yellowish-green matter is discharged onto the surface of the skin. The nodules are very similar to furuncles, but they have not got core in the center (hence the name pseudofurunculosis) and are not connected with pilosebaceous apparatus. Multiple nodules are located on the trunk, hairy part of the head, buttocks, lower and upper extremities. After discharge of a matter, the process ends with cicatrization.

The course of the disease is rather soft, often relapses, is accompanied by high temperature, leukocytosis, increased ESR, and can be complicated with phlegmon, septicemia.

In the pathogenesis of multiple abscesses appearance a significant role is played by unhygienic maintenance of children, increased sweating, malnutrition, the presence of dyspepsia and enteritis, rickets, tuberculosis and other infectious diseases.

The diagnosis is set on grounds of appearance in children of persistent recurrent abscesses not connected with hair follicles and sebaceous glands.

Hystologically, the appearance of purulent abscesses, connected with sweat glands and their ducts, are observed in derma and subcutaneous fat.

The treatment includes the administration of antibiotic injections. Topically, unmixed ichthyol is applied at the affected sites or the abscess is pierced with a sterile needle after skin disinfection with the following anointment by alcoholic solutions of aniline dyes. After ulceration the antibiotic ointments are applied at these sites.

Bullous impetigo *(impetigo bullosa)* is referred by one authors to streptoderma, and the others to the pyoderma, caused by staphylococci (benign form of staphyloderma). Clinically, the disease is characterized by the appearance of blisters or phlyctenas the size of a pea to a pigeon's egg, filled with serous nebulous liquid or matter. The elements are not stressed; they are surrounded with hyperemic circle, and quickly erode. The erosions with wet surfaces can enlarge at periphery; they are surrounded with the scraps of epidermis. Bullous impetigo is localized on the trunk, back of the hands, less often on the feet and lower legs. The course of the disease is benign; the general state of the children is satisfactory. The treatment consists of anointment of erosions with the alcoholic solutions of aniline dyes and antibiotic ointments.

Streptococcal infections of skin – streptoderma

Streptoderma are caused by streptococci, which unlike staphylococci, do not affect pilosebaceous apparatus and sweat glands and do not infect derma and subcutaneous fat with the following development of necrosis. Streptococci mainly cover the smooth skin, and the diseases caused by them are of superficial character and in majority of cases manifest as elements in the form of blisters or phlyctenas, filled with clear or slightly nebulous contents. Most frequently, streptodermae are observed in women and children due to the fact that their skin is more delicate.

The following forms of streptodermae are distinguished, these are streptococcal impetigo, streptococcal intertrigo, syphiloid papular impetigo, superficial panaritium, perleche, lichen simplex, ecthyma vulgaris, and superficial chronic diffuse streptoderma.

Streptococcal impetigo *(impetigo streptogenes)* is caused by streptococcus and localized mainly on the face, extremities, sometimes on the trunk. In the pathogenesis of streptococcal impetigo the skin injuries are of certain importance, as well as its unhygienic conditions, metabolic disorder, reduced immunological reactivity. Impetigo is especially common in children.

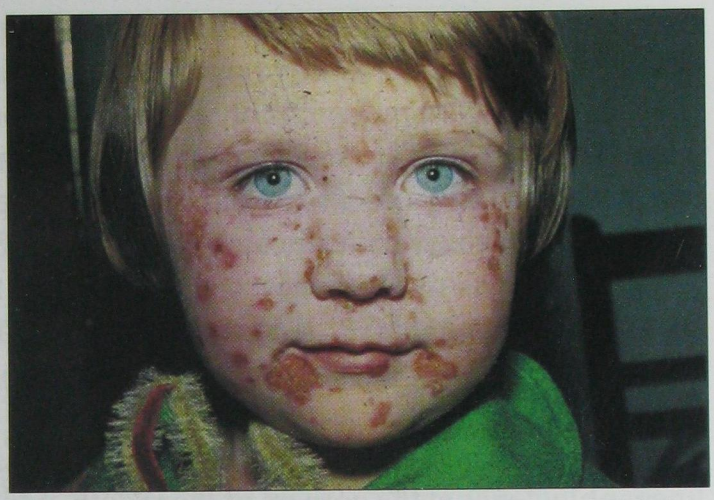


Fig. 4.7. Streptococcal impetigo.

The blisters or phlyctenas with the size of a pea to a nut, not stressed and filled with serous or slightly nebulous liquid appear on the hyperemic, lightly swollen skin, and rapidly increase in size. There is a hyperemic circle on the periphery of phlyctenas. Phlyctenas quickly erode (during the period of several hours) and are covered with thin straw-yellow crust. After the falling of crust away, the pink spot remains and after some time it disappears without any trace. A patient suffers from itching, which sometimes can become intense. In some cases regional lymphadenitis can develop. Phlyctenas are located independently, but sometimes they can merge due to peripheral growth, forming arcs, rings, garlands (ring-shaped impetigo). Streptococcal impetigo has benign course and it ends in full recovery after 7-8 days.

Intertriginous streptoderma, or ***streptococcal inertrigo*** *(intertrigo streptogenes)* is mostly observed in children, especially overfed children, with excessive sweating and gastrointestinal disorders. In the pathogenesis of the disease the dermatoses, accompanied by itching, and diabetes are also important. Intertriginous streptoderma is localized in the skin folds, such as inguinal-scrotal, gluteal, in axillary cavities, behind the ears, under the breasts in women, and in the folds of the abdomen in obese people. This disease is characterized by the appearance of erosive wet surface of bright pink colour, strictly bounded from the adjacent skin and surrounded by epidermal collarette. In the depth of the folds the bleeding folds appear. Subjectively, the patients notice itching and burning sensation. It is possible to notice at the close skin sites the pustules at different stages of development. The disease has long-term course.

Syphiloid papulose impetigo *(impetigo papulata syphiloides)* develops predominantly in infants and is localized on the buttocks, posterior surface of femora and lower legs. On the hyperemic surface the phlyctenas appear, at the base of which there is papulous infiltrate. Phlyctenas erode very quickly, leaving erosive papules. Clinically the disease is analogous to papulo-erosive syphilide. For differential diagnostics it is necessary to carry out the analysis of erosion discharge for the presence of treponema pallidum, and serological study of the patient as well.

Hystologically, in all forms of streptococcal impetigo there is formation of cavity under the horny layer of epidermis. The cavity is filled with serous exudate with some amount of neutrophilic leukocytes and separate epithelial cells. Spongiosis occurs in

the spinous layer of epidermis. Vascular distention with perivascular infiltrate, consisting of neutrophils and lymphocytes, occurs in derma.

Superficial panaritium, is predominantly observed in adults. The phlyctenas appears on the hands around the nail plates, which contain at the beginning serous, and then nebulous purulent discharge. The disease is associated with injuries of the fingers, burrs, which create favorable conditions for the penetration of streptococci. The affected phalanx of a finger swells and hurts.

After breaking of phlyctena there appears erosion, covering the nail wall like horseshoe. The process can lead to the rejection of nail plate. Sometimes there are lymphangitis, general uneasiness and fever.

Angular impetigo *(angulus infectiosus)* is characterized by the appearance of linear phlyctenas at the corners of the mouth, which quickly erode, and the cracks appear on their place, which are especially painful when the mouth is opened. The disease can be localized in the corners of the eyes, in the places of adhesion of ear conches. On the skin around the crack of the angle of mouth there appear melichrous crusts, and maceration of epidermis on the edges of the cracks. The contributing factors to the perleches development are frequent lips licking, dental prostheses wearing, i.e. maceration of the corners of the mouth sites with saliva. The patients complain about itch, pain while eating.

Angular impetigo should be differentiated with yeast affection of the corners of mouth, in case of which the process is not so vivid and there are no crusts. It must be taken into account the possibility of affection of the corners of mouth with erosive syphilitic papules, which certainly are based on tight elastic infiltrate; eruptions of syphilides are observed on the other parts of the body, as well as positive serological reactions.

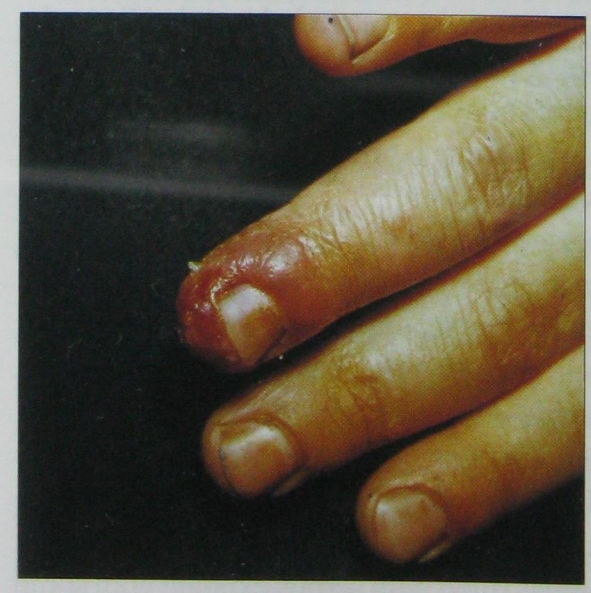


Fig. 4.8. Superficial panaritium.

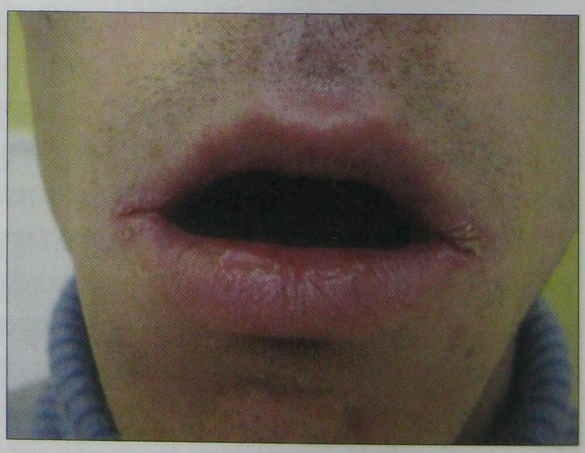


Fig. 4.9. Angular impetigo.

The treatment of different forms of impetigo, and angular impetigo is generally external. In the case of presence of crusts the ointments with disinfectants or antibiotics are applied.

On the erosive wet surfaces the lotions with disinfectants (0,25% solution of silver nitrate, 2% solution of resorcinol etc.) are applied. The healthy skin around the lesion foci is regularly wiped with the 2% salicyl alcohol in order to prevent autoinoculation of infection. At the same time, it is necessary to treat the diseases, which cause the appearance of streptoderma, and eliminate the promoting factors.

Ecthyma vulgaris *(ecthyma vulgare)* refers to profound pyodermae, caused by streptococcus, though there are a number of publications, which indicate that ecthyma can be of staphylococcal etiology as well. Micro injuries and excoriations contribute to the penetration of the agents from the external environment. Nervous and mental stresses, diseases of liver and blood (anemia, leucosis), diabetes, thrombophlebitis, vasculitis, vitamin deficiency etc. are of certain importance in the pathogenesis of the disease is played.

The disease develops gradually, beginning from the appearance on the skin of usual phlyctena the size of pea to a nut, filled with serous contents, which later acquire mattery hemorrhagic character. Phlyctena is located in the setting of erythematous infiltrate, its contents dry up very quickly in the crust of yellowish-brown colour, which has got multi-layer characteristic. Under the skin, there is deep tissue necrosis, covering not only derma, but subcutaneous fat as well. The crust drops off in several days, and the ulcer with soft uneven congested hyperemic edges and bleeding bottom, covered with dingy pultaceous detritus. After two-three weeks the ulcer is slowly cicatrizing. The scar is surrounded by the zone of hyperpigmentation.

There are, as a rule, several ecthymae, and they are most frequently localized on the skin of the lower legs, buttocks, femoris, lumbus, less often they are on the upper extremities. Ecthymae can recur, causing lymphangites and lymphadenites, phlebitis, deep necroses.

In the severe course of the disease so-called *ecthyma terebrans* can appear. Most frequently it occurs in undernourished children, suffering from anemia or rackets. Some authors consider blue pus bacillus to be the agent of ecthyma terebrans. In this case infiltrate and ulcerative defect spread depthward. The disease begins with the appearance on the skin of the buttocks, lower extremities, hairy part of the head of blisters, rapidly evolving into pustules and deeply ulcering. Such ecthymae

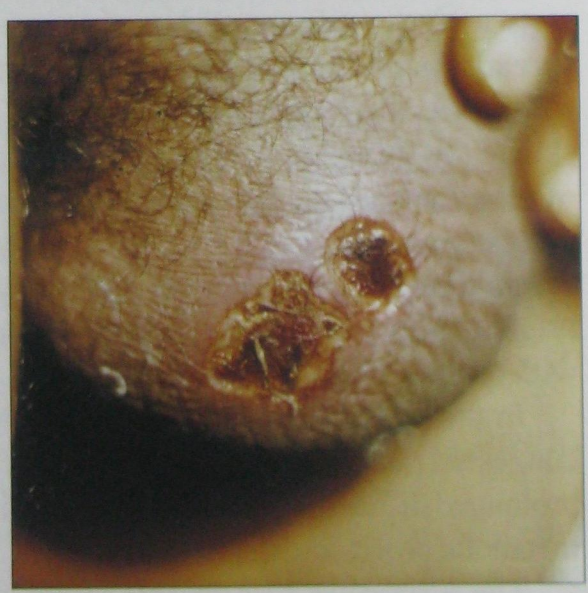


Fig. 4.10. Streptococcal scrotal ecthyma.

cause severe pains. Ecthymae terebrans have got malignant course, are complicated by sepsis, and can be fatal.

Ecthyma vulgaris most frequently should be differentiated with syphilitic ecthyma, which has not got acute inflammatory nature. There are no impetiginous multiple small elements near ecthyma, and the syphilides (especially papulous) can be detected at the other sites of the body. The final diagnosis is defined by the positive serological reactions. In case of scabies, complicated by ecthyma, military-papular rash appears on the other typical areas of skin, the borrows are typical; the patient complains about strong itching, especially at night.

Hystologically, in derma and subcutaneous fat there is necrosis of tissue. The focus is surrounded with indurated infiltrate, consisting of neutrophilous leukocytes and lymphocytes. Edema and vasodilatation appear around the infiltrate.

On the initial stages of the development of ecthyma vulgaris, the phlyctenae are pierced with a needle for removing their contents, the crusts are softened with application of 2% salicyl ointment. In case of formed ecthyma or ecthyma terebrans, antibiotic therapy in accordance with the results of antibiogram, A vitamins and B group vitamins, and disinfecting ointments for topical treatment are put on. The skin around erythema is wiped with 2% salicyl alcohol.

Superficial chronic diffuse streptoderma *(streptodermia chronica diffusa superficialis)* is a chronic streptococcal disease, which is characterized by diffuse lesion of significant sites of skin covering. Most frequently it affects the lower extremities, the process can be also localized on the opistheners. The affected areas have got largely-scalloped lines, sharply bordered from the surrounding healthy skin by the rim of exfoliative epidermis. The skin of the affected areas is sharply hyperemic, of congestive bluish colour, slightly infiltrative, the surface is eroded in the form of wet disk-like sites. The erosions are covered with many thin lamellate crusty scales of yellowish or greenish colour. After removal of the crusts the surface wets with the release of dense serous or serous-yellow exudate. The process spreads over the periphery. With time, staphylococcal infection joins to streptococcal, though there is no lesion of hair follicle and sebaceous gland. The skin of the entire lower leg can be affected on the lower extremities. In the process of development, the lesion area is epithelialized and covered with large lamellate scales. Superficial chronic diffuse streptoderma is often complicated with eczematization, especially on separate skin areas, where against the background of bright red erythema there appear military papules, microvesicles, small erosions with release of the drops of serous fluid.

In the pathogenesis of the disease development a significant role belongs to congested phenomena in the lower extremities, varicose symptom complex, i.e. prolonged disturbed circulation, development of tissue hypoxia and derangement of metabolic processes in skin.

The disease has got a chronic course, with often relapses, especially around non-healing wounds and trophic ulcers.

Superficial chronic diffuse streptoderma should be differentiated with eczema, when the erythema is bright red, the foci are without distinct borders, oozing lesion is punctate, there are no crusty scales, and the process has got symmetric character.

Hystologically, in the erosion places there are no horny and granular layers. At the sites of epidermis continuity there are parakeratosis, spongiosis, intensively pronounced acanthosis. Vascular distention with perivascular infiltrate, predominantly lymphocytic occurs in derma.

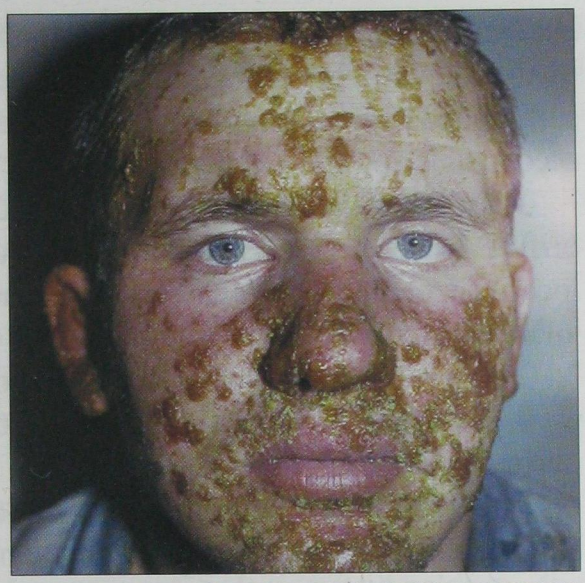


Fig. 4.11. Superficial diffuse streptoderma.

In case of acute course of superficial chronic diffuse streptoderma antibiotic therapy in accordance with the results of antibiogram is put on. For topical treatment the lotions of disinfecting and astringent solutions(5% tannic or 2% boric acid, 1% solution of resorcinol, 0,25% solution of silver nitrate) are applied. At the same time, it is necessary to carry out curative interventions, aiming at the elimination of factors contributing to disease development.

Strepto-staphylococcal skin infections – mixed pyoderma

Mixed pyoderma combine a number of chronic skin diseases of pyogenic nature, mainly polymicrobial one. Their main etiological cause is combined streptococcal and staphylococcal flora. Possible is the participation in the genesis of these diseases other microorganisms as well, such as collibacillus or blue pus bacillus, Proteus vulgaris etc..

In the pathogenesis of mixed pyoderma the essential role belongs first of all to sharp decrease of immunological reactivity of organism and the appearance of sensitization to byproducts of pyoderma agents, especially in children with allergic dermatitis, malnutrition, vitamin deficiency, metabolic disorders, endocrine dysfunctions etc..

Mixed pyoderma include impetigo vulgaris, chronic ulcerative vegetating pyoderma, chancriform pyoderma and botryomycoma.

Impetigo vulgaris *(impetigo vulgaris)* is preceded by the prodromes, such as high temperature and itching at the sites of the following rash appearance.

The disease appears at the beginning as streptococcal impetigo with the appearance at erythematous and slightly infiltrated background of phlyctenas, the contents of which due to overlay of staphylococcal infection get muddy quickly,