УДК 616.1-07

ОСМОТР ПРИ СЕРДЕЧНО-СОСУДИСТЫХ ЗАБОЛЕВАНИЯХ

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Санкт-Петербургский государственный университет. 199034, Санкт-Петербург, Университетская наб., д. 7–9.

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РЕЗЮМЕ. Данная публикация посвящена вопросам пропедевтики внутренних болезней, в первую очередь — на материале сердечно-сосудистой, эндокринной и бронхолёгочной патологии. Пропедевтика толкуется авторами широко, как введение во внутреннюю медицину, поэтому статья содержит и терапевтический, и клинико-патофизиологический материал. Публикация сопоставляет достижения и традиции отечественной терапевтической школы с принципами преподавания внутренней медицины, сложившимися в практике зарубежного медицинского образования. В статье рассматривается методология физикального обследования и интерпретации его данных применительно к визуальному осмотру при сердечно-сосудистой патологии.

КЛЮЧЕВЫЕ СЛОВА: сердечно-сосудистые заболевания; физикальное обследование; осмотр; поза больного; сознание; состояние больного; конституция; цвет кожи; отек; гиппократовы пальцы; верхушечный толчок; пульсация сосудов; набухание вен; стигмы гиперлипопротеинемий; контрактура Дюпюитрена.

VISUAL EXAMINATION IN CARDIOVASCULAR DISEASES

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ABSTRACT. This publication continues a series of authorial lectures devoted to questions of Propaedeutics of Internal Diseases, primarily based on the material of cardiovascular, endocrine and bronchopulmonary diseases. Propaedeutics is widely interpreted by authors as an Introduction to Internal Medicine; therefore, these lectures also contain clinical pathophysiological material. The lectures compare the achievements and traditions of Russian classical therapeutic school with the principles of Internal Medicine that have evolved in the practice of foreign medical education. The third lecture is dedicated to methodology of physical examination and its data interpretation related visual inspection in cardiovascular pathology.

KEYWORDS: cardiovascular diseases; consciousness; medical state; patient's position; physical examination; somatotype; visual inspection; skin color; edema; nail clubbing; apex beat; vascular pulsation; vein swelling; stigmas of hyperlipoproteinemia. palmar sclerosis.

The objective physical investigation in cardiovascular patients commonly proceeds according the following sequence: Visual examination, palpation, percussion, and auscultation [2]. Both heart and blood vessels should be carefully examined.

PATIENT'S POSITION

During *visual examination* at first turn medical doctor has to inspect patient's position. In compensated state of their diseases cardiovascular patients live with common and normal life. The patients can walk deliberately and usually, have no complaints [6]. In overt heart failure the patients instinctively prefer to keep their bed in *Fowler's position*[1], *half-risen, almost half-sitting, get-*

[1] Fowler, George Ryerson — an American surgeon, born December 25, 1848, New York City; died February 6, 1906, Albany, New York, described the semi-sitting position in 1900 [here and everywhere below biographies and historical data are given according [12, 14].

ting from this posture some relief. As a rule, while being in such status, they avoid lying on their left side, escaping the unpleasant feeling in heart.

Severe breathlessness makes patients sitting on the bed with their feet on the floor. This position facilitates the blood deposition in lower extremities and relieves the lesser circulation congestion. Position like this is helpful also for ascites patients due to easier diaphragmatic respiratory movements. In severe heart failure the patients use to spend all day and night long sitting in such position and it may last even for weeks, if proper therapy is not applied. The patients with left ventricular failure (caused by myocardial infarction, noticeable cardiosclerosis, left ventricular postinfarction aneurysm, valvular heart diseases etc) also keep similar position.

Typically the patients with right ventricular failure («cor pulmonale») keep this pose rarely and commonly prefer normal laying position, in spite of dyspnea.

In acute vascular failure (in shock, collapse) patients also keep horizontal position, because an attempt of rising provokes insufficiency of cerebral circulation and syncope. In certain cardiac diseases patients have to keep some peculiar forced positions, producing a relief. E. g., in exsudative pericarditis a patient may keep on all fours or lies on his/her tummy, sometimes sits holding the knees. In noticeable heart dilatation the patients commonly lie on their right side [3]. In thrombophlebitis and other diseases of the veins of lower extremities the patients may keep the legs in raised position, trying to diminish pain and tension in low extremities. The posture with foot of the bed risen is called «Trendelenburg's position»[2].

MEDICAL STATE AND LEVEL OF CONSCIOUSNESS

Next thing to evaluate is the patient's general *medical state*. It may be either satisfactory (fair) or serious (heavy), or even critical (extremely heavy). In fair state patient usually can normally walk, does not keep some forced position, can easily serve himself/herself. Patient's vital signs are stable and within normal range. Patient is conscious, although may be uncomfortable. His/her immediate prognosis in fair condition is favorable [16].

In serious state (Russian case histories use the term «heavy» instead) [1], someone, as a rule, has to look after the patient, which is unable to serve himself/herself or voluntary change his/her posture. Vital signs may be unstable and beyond normal limits, immediate forecast is doubtful.

Examining, doctor checks also patient's level of consciousness. In Medicine consciousness is just a normal wakeful status of brain — no more, nor less, without any philosophical interpretations peculiar to humanitarian sciences. Level of consciousness may be different [1, 15]. It is *clear*, when a patient takes part in conversation and answers the questions, being properly oriented in time and circumstances. Clear consciousness is a «state of wakefulness, awareness, or alertness in which most human beings function while not asleep» [16]. The slightest disorder of consciousness level is *obnubilation* (in English medical texts: *Clouding of consciousness*). It is «a very mild form of altered mental status in which the patient has inattention and reduced wakefulness» [16], like being slightly stunned or a little bit tipsy. In stunned (or staggered somnolent) state the patient hardly can get his bearings as regarding time and circumstances and may be inadequate in his answers or hesitates with them. This state can be graded in depth. In *stupor* state patient's consciousness is deeply reduced, a patient is severely stunned, motionless, displays mutism and decreased sensitivity, drowsy but not yet sleeping. In *sopor* state patient is insensible, does not answer any questions, but responds the strong stimuli (e. g. injections), however being able to realize all the physiological reflexes, usually keeping stools and urine. German and Russian medical texts usually distinguish spoor and stupor [1, 7, 10], whereas English ones often do not [10, 15].

In state of *coma* consciousness is absolutely lost, normal reflexes are absent, pathologic ones may arise, which are not normally observable. Coma may have various aetiology, but always is related to key pathogenetic link: Tissue hypoxia of the brain. Tonic or (more often) clonic convulsions may accompany coma. The comatose state can be registered in cerebral form of myocardial infarction. The patients with rheumatic heart diseases may develop coma after cerebrovascular thrombotic embolism or in serious cerebral vasculitis of different aetiology. Commonly, comatose patients are unable to keep stools and urine and have involuntary enuresis and encopresis.

Evaluating the general medical state of a patient, physician also can make a statement of case dynamics and immediate prognosis (is the case improving, worsening or stable).

SOMATOTYPE

Visual examination is to determine patient's somatotype (body constitution). It may be of *normosthenic, asthenic or hypersthenic* (picnic) types. M. V. Chernorutsky[3] introduced these terms in Russian medical practice. In English-speaking countries analogous division was later suggested by W. H. Sheldon[4], who used similar criteria (ectomorphic, endomorphic and mesomorphic somatotypes). Human constitution is quite important in cardiovascular diseases spreading and diagnosis. The persons of hypersthenic somatotype genetically are more susceptible to arterial hypertension and atherosclerosis, asthenic ones — to dysautonomia and arterial hypotension.

SKIN COLOR

[2] Trendelenburg, Friedrich — a German surgeon, born May 24, 1844, Berlin; died December 15, 1924, Nikolassee near Berlin, described the position in 1890.

[3] Chernorutsky, Mikhail Vasil'evich — born 1884, Meleuz, Russian Empire; died 1957, Leningrad, USSR) — Russian and Soviet internist, suggested his gradation of somatotypes in 1928.

[4] Sheldon, William Herbert — born November 19, 1898 at Pawtuxet, USA, died September 17, 1977 at Cambridge, USA, an American psychologist, eugenicist and numismatist, suggested somatotyping method and gradation of somatotypes in 1936–40.

The color of the skin is of utmost significance in cardiovascular diagnosis. Simultaneously physician must check the color of mouth mucous membranes. Visually, doctor can disclose not only color changes, but also hemorrhagic rush, telangiectases, the manifestations of venous congestion in low extremities, resulted from chronic heart failure (of varying severity, up to the skin ulceration). In chronic and acute thrombophlebitis local hyperemia may be revealed, diabetes mellitus is characteristic for skin atrophy, dystrophic nails and pallor of the shins and feet. Skin hemorrhagic rush may occur in septic conditions (e. g. bacterial endocarditis), rheumatic attacks (with annular erythema over joint areas), in vasculitides (Schönlein-Henoch purpura[5]). Pay your attention to the localization of the rush and presence or absence of its symmetry [1, 5, 13].

Cyanosis, which is bluish color of skin and mucosa membranes, — often occurs in cardiovascular diseases. In extreme degree both patient's face and trunk are looking almost black (severe non-compensated cases of pulmonary heart). On initial stage cyanosis is more noticeable at the places with thin skin: nose, lips, chicks, hands (fingers). Progressing cyanosis spreads on the body and extremities. Usually cyanosis marks the elevated blood level of reduced hemoglobin, which has darker color as compared to oxyhemoglobin. The reasons of cyanosis are different. In central cyanosis the blood arterialization is disturbed, as a result of bronchopulmonary diseases and/or poor content of oxygen in the air. In peripheral one blood circulation is retarded, thus facilitating the oxygen diffusion into tissues. As a result, venous blood is enriched with reduced hemoglobin. The patients with cor pulmonale of different aetiology usually have most pronounced central cyanosis (either in bronchopulmonary or thoracic-diaphragmatic and vascular reasons). Different cardiac diseases commonly produce peripheral cyanosis due to circulation disturbance. Cyanosis may be either local (in thrombophlebitis of lower extremities and mechanical compression of the veins by enlarged lymph nodes), or extended (spread). The last is especially noticeable in severe congenital heart diseases, like Fallot's tetralogy[6] or in pulmonary artery sclerosis (Ayerza's disease[7]). The cyanoderma may be permanent (chronic heart failure) or sets in abruptly (pulmonary artery branches embolism, pneumothorax) [8]. If vena cava superior is compressed (by the tumor) isolated cyanosis of the head and neck may occur. In poisoning with certain drugs and toxins, cyanosis may be a result of excessive methemoglobin or sulfohemoglobin production. The efforts and exercises usually increase in such patients both cyanosis and dyspnea, oxygen breath gives them a relief (if the aetiology of hypoxemia is not vascular shunting one).

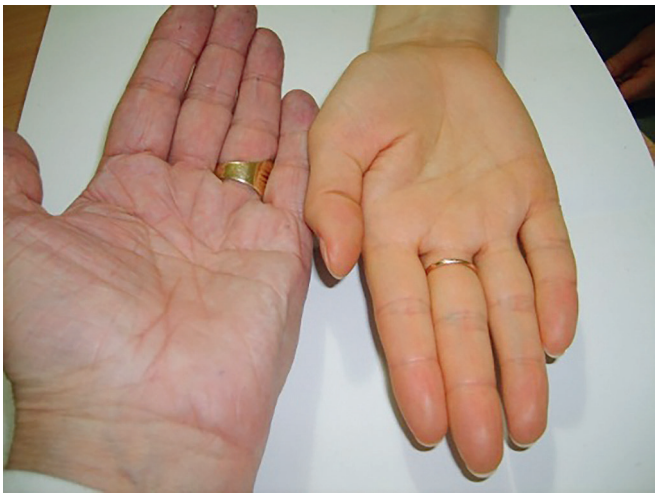

Fig. 1. Right: False carotinemic jaundice in a patient with chronic autoimmune thyroiditis. Left: hand of healthy person. Observation by Y. I. Stroev

Unusual skin and mucous membranes pallor without anaemia may be observed in stenotic aortal heart disease, while mitral heart diseases are always accompanied by so called «mitral face»: Cyanosis plus typical appearance — bluish lips and chicks and pale nasolabial triangle. Pallor in aortal stenosis is caused by insufficient blood ejection into arterial system from left ventricle [7, 9].

But, pallor may accompany the aortal insufficiency as well, due to large portion of regurgitated blood and decrease of peripheral blood supply. The patients with bacterial endocarditis are particularly pale; this hue of skin is called «white coffee».

The noticeable skin pallor is typical for hypopituitarism, including panhypopituitarism and so called Sheehan's[8] syndrome (due to declined melanocyte stimulating hormone production). Sheehan's syndrome may develop after difficult labor in high blood pressure. It is suggested to be caused by pituitary necrosis due to either tromboembolism of supplying artery or because of intrapituitary hemorrhage. There are also signs of autoimmune lymphocytic hypophysitis in this disease. It may complicate the treatment with checkpoint inhibitors for immune stimulation in oncologic patients as an adverse effect, because pituitary and lymphoid cells share common CTLA-4 antigen. Paleness is also characteristic for the patients with essential arterial hypertension, as well as in collapse, shock and syncope.

Yellowish skin and mucous membranes are regarded as frequent manifestations of advanced chronic heart failure because of liver swelling and secondary hepatic dysfunction. Yelowish sclerae are common finding in this case. Subicteric color of skin may be observed in diabetes mellitus and in hypothyroidism (in last

[5] Schönlein, Johann Lukas (30 November 1793, Bamberg, Germany — 23 January 1864, Bamberg, Germany) was a German physician and naturalist. He described this disease in 1837.

Henoch, Eduard Heinrich — German paediatrician, born June (30: July) 16, 1820, Berlin, Germany; died 1910, August 25, Dresden, Germany. He added the description of disease in 1868.

[6] Fallot, Ethienn-Louis Arthur — French physician, born September 29, 1850, Sète outside Marseille; died April 30, 1911. Tetralogy was described in his paper of 1888.

[7] Ayerza, Abel — an Argentine physician, 1861–1918. Buenos Aires, described «chronic cyanotic disease» in 1900.

[8] Sheehan, Harold Leeming, born 1900 at Carlisle, UK, died 1988 — a British pathologist. He described this syndrome in 1937.

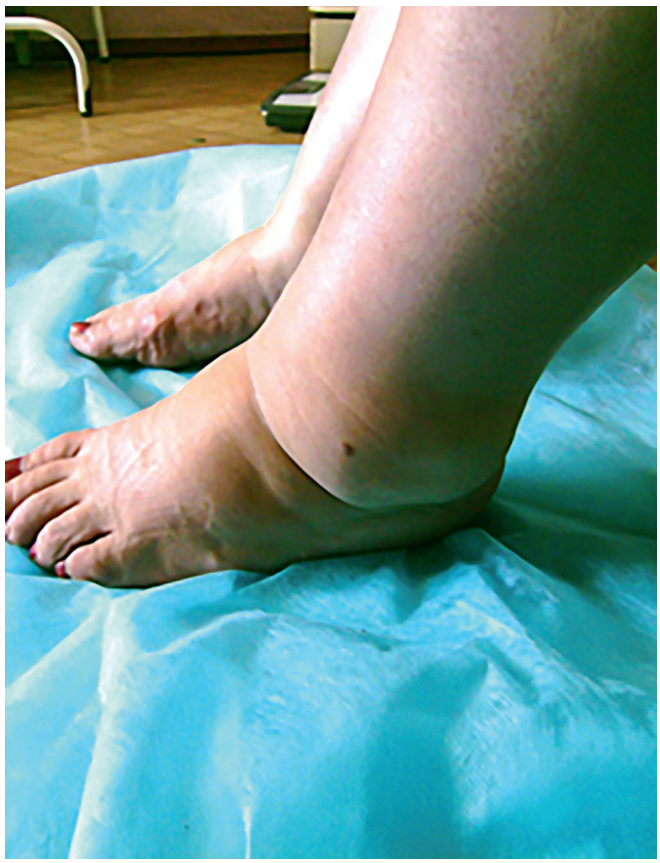

Fig. 2. Myxoedematous lower extremities in a patient with advanced autoimmune thyroiditis. Observation by Y. I. Stroev

case it is a result of false jaundice, caused by lack of carotinase activity in hypothyroidism) [fig. 1].

Constrictive pericarditis provokes both functional and morphologic changes within the liver (due to congestion in venae cavae) known as Niemann-Pick[9] pseudocirrhosis. The noticeable jaundice is observable in this case.

Examining the hair, doctor mast pay attention to the degree of pilosis. Early and frequent baldness (alopecia) is noticeable for atherosclerotic patients. Certain types of arterial hypertension (e. g. post-contusion one) may bring in early canities [5, 7, 10].

EDEMATA

During the examination ad oculus, doctor determines the existence of dropsy and its location (face, extremities, and trunk). The edemata are common in cardiovascular patients. When patients use to walk and stand much, dropsy is usually located on low extremities. If a patient spends major part of time lying in bed, edema may be observed on his back, sacral area, or right side, when he use to lay right side down. Both cardiac and renal dropsy give the pit at the place of finger pressing, slowly flattening out after taking the doctor's finger away (pitting oedema). Only one disease always gives dropsy without observable pits after pressure — it is hypothyroidism [5], known for myxoedema [fig. 2].

Extremely manifested total edema of subcutaneous fat in abdominal and chest area plus fluid collection within the peritoneal, pleural and pericardial cavities is called anasarca. The fluid congestion in pleural cavity is designated as *hydrothorax* (left, right, or bilateral one); similar accumulation of transcellular fluid in abdominal cavity — is known as *ascites*, and in pericardial cavity — as *hydropericardium*. Left hydrothorax may be caused by heart failure or Dressler's[10] postinfarction autoimmune syndrome (polyserositis-polysinovitis), right side one — by compression of vena cava superior. Hydropericardium may be a manifestation of anasarca or pericarditis (exsudative one, with tuberculosis, rheumatic, oncologic either autoimmune aetiologies).

The isolated ascites, not accompanied by the signs of heart failure or hepatic cirrhosis, is very suspicious for some oncologic disease (most frequently, tumor of ovaries with peritoneal metastases).

In edema of low extremities the skin is smooth, pale and tightens, it has waxen appearance. Chronic edema is manifested in skin structure changes; skin becomes firm, stiff, non-elastic and dark, sometimes with shin ulceration. The vascular diseases may cause also local edemata, for example in venous hyperemia due to some vein compression by either tumor, or enlarged lymph nodes.

Improper renal function in cardiovascular diseases always contributes into edema formation. It is necessary to remember, that cardiac dropsy, unlike renal one, is always submitted to hydrostatic laws, locating at lowest possible places under the action of gravitation. So, in renal diseases facial edema is common, although in chronic heart failure feet dropsy is typical. The development of edema is, mostly, gradual. But, dropsy also may set in quite rapidly, for example in case of long paroxysmal tachycardia attack, quickly disappearing after the stopping of its episode. Internal edema usually goes in parallels with the external one, although former is not visible from outside. So, dyspepsia in cardiac diseases may be related to hepatic and gastrointestinal dropsy. In cerebral stroke doctor may observe edema of palsied extremities. Before the introduction of diuretics into broad clinical practice, the physicians not infrequently encountered the extreme degrees of edemata, which manifested in cutaneous bullae (blisters), filled with plasma. The generalized edema in males usually involves scrotum, so it is essential to examine it, because scrotal edema may hurt patient.

NAIL CLUBBING

The phenomenon of «drumstick fingers» or Hippocratic fingers (after the name of the father of Western Medicine, ancient Greek physician Hippocrates (circa 460 B. C. — circa 370 B. C.) who has described them as a morbid sign long ago) — may be revealed

[9] Niemann, Albert (23 February 1880, in Berlin — 22 March 1921, in Berlin) was a German physician; Pick, Ludwig (August 31, 1868 — February 3, 1944) was a German pathologist

[10] Dressler, William (1890–1969) — an American (born and started his medical career in Poland) cardiologist, described the syndrome in 1956.

when examining a cardiologic patient both on hands and feet. The symptom of nail clubbing is not entirely caused by edema and rather is related to chronic hypoxia and proliferation of soft tissues around a nail. It is frequent for bronchoectases, various chronic lung diseases (cancer, tuberculosis, sarcoidosis, idiopatic pneumofibrosis), occurs in mesothelioma, but exists also in severe congenital heart diseases (like Fallot's tetralogy) or develops in prolonged course of bacterial endocarditis. It is somehow associated with inappropriate increase of vasopressin production. Interestingly enough, it occurs in some autoimmune diseases also, like Crohn disease and autoimmune thyroid disease. In last case very similar sign is known as thyroid acropathy. There are cases of nail clubbing, which appear to be irrelative to any disease: Thus, drumstick fingers are not infrequent among healthy black Africans. However, there are two nosological entities with obligatory presence of nail clubbing symptom. First one is hereditary primary hypertrophic osteoarthropathy, or Touraine–Solente–Golé[11] syndrome. This condition has been linked to mutations in the gene on the fourth chromosome (4q33-q34) coding for the enzyme 15-hydroxyprostaglandin dehydrogenase; with decreased breakdown and raised concentration of its substrate — prostaglandin E2 [15]. Another one is a hypertrophic pulmonary osteoarthropathy, or Bamberger — Pierre Marie[12] syndrome (in this disorder drumstick fingers are combined with thickening of periosteum and synovium. The general habitus of such patients may resemble acromegalic signs. Unlike acromegaly, related to excessive effects of growth hormone, nail cubbing phenomena hypothetically are caused by hyperproduction in lungs and other sources some paracrine growth factors and their aberrant systemic effects. At least some cases of this phenomenon may be paraneoplastic, which requires from physician observing this symptom recommendations of profound oncologic screening in such a patient.

VISUAL EXAMINATION OF CARDIAC AREA: APEX BEAT

Cardiac area should be examined under the light going from aside, because it may allow to reveal protrusion or some movements there. The protrusion of the whole cardiac area is common for congenital heart diseases, manifested since early childhood (Botallo's duct[13] patency and others) and as a rule it exists all lifelong. But, the «cardiac hump» may be unrelated to heart pathology (it presents in congenital anomalies of skeleton, chest and rib tumors). In adults some protrusion of cardiac area is observable in exsudative pericarditis. The protrusion above the cardiac area may be caused by aortic aneurysm [5, 9, 11]. Anterior mediastinal and thoracic tumors also may cause cardiac hump. Severe chest deformations of different origin (e. g. in tuberculosis spondylitis) may lead to thoraco-phrenic cor pulmonale.

The healthy non-obese individuals always have visible rhythmic pulsation over cardiac area, caused by the impacts of beating cardiac apex and thoracic wall. The conventional term for this phenomenon is *apex (cardiac) beat* (AB). Naturally, apex beat is situated a little bit upper and to the medium from the real anatomic position of cardiac apex, which is covered by lungs. Apex beat is produced by left ventricle contractions.

In individuals of normosthenic somatotype AB should be located in 5th intercostal space, 1,5–2 cm to the medium from medioclavicular line [1–2]. In asthenic persons AB is one intercostal space lower, and in hypersthenics individuals — one space upper and a little bit shifted to the left. Some cardiologists consider *apex* beat to be normal phenomenon, and use the term «*cardiac* beat» instead — only in case of disease accompanied by right ventricular hypertrophy (cor pulmonale, mitral stenosis, and tricuspid valve disease) [2, 8, 13]. These conditions produce rising pulsation in the area of left edge of sternum bone, resulted from movements of hypertrophic right ventricle. The pulsation may be observed also in the area to the right of sternum — this occurs in aneurysm of ascendant portion of aorta, or in aortal valve insufficiency. Cardiac aneurysm produces pathologic pulsations in 3rd–4th left intercostal space. In small children before age of 5 years old, AB may be situated in 4th space laterally from medioclavicular line. Changing of body position moves the point of AB 3–5 cm from its usual location. AB and the whole heart are moved aside in unilateral hydrothorax, pneumothorax or hydropneumothorax. Mind, that the heart in these cases is displaced contra laterally — to the opposite side from the side involved. In left hydrothorax AB is invisible/non-palpable. It is hardly recognizable in obesity and in marked pulmonary emphysema. Sporadically, the chest wall retraction (retrograde motion) may be observed over cardiac area instead of protrusion. This is so called *negative* AB, characteristic for adhesive pericarditis, due to adherence and joining of sticky pericardium layers together.

VISUAL EXAMINATION OF THE AREAS OVER LARGE VESSELS

The area of epigastrium also is to examined attentively. There may be three kinds of epigastric pulsation: From up — downwards (in right ventricle hypertrophy); from right — to left (in liver enlargement due to tricuspid valve insufficiency) from back — forward (derived from abdominal aortic pulsation, as a rule in slim patients).

The pulsation of peripheral vessels is, normally, invisible. Under pathologic conditions the vessels may display abnormally strong pulsation and some visually registered changes. The vermiform bending of temporal arteries is observed in essential hy-

[11] Touraine, Albert (1883–1961); Solente, Gabriel. (1890–1986); Golé, Laurent. (1903 — after 1975) — the French dermatologists

[12] Von Bamberger, Eugen — a German physician, born September 5, 1858, Würzburg; died October 1921. His description of the syndrome is dated 1889; Marie, Pierre — a French internist, neurologist and pathologist, born September 9, 1853, Paris; died April 13. 1940, Paris. He described the syndrome in 1891, There was also earlier description of 1868 by German internist and neurologist Nikolaus Friedrich (1825–1882) and first description of familiar cases (Mankowsky's syndrome) by Russian neuropathologist Boris Nikitovich Man'kowsky (11 March 1883, Kiev, Russian Empire — 24 November 1962, Kiev, USSR).

[13] Botallo, Leonardo — an Italian anatomist (1530–1600) to whom this eponym was erroneously attributed, never described the duct, which was first described in 1564 by another Italian anatomist and surgeon — Giulio Cesare Aranzi, born 1529/1530, Bologna; died April 7, 1589, Bologna.

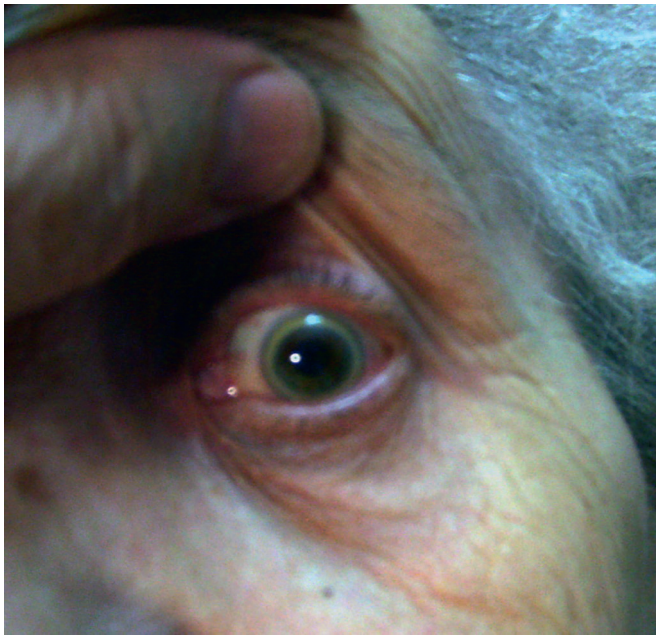

Fig. 3. Corneal lipid arch in precautious senescence due to hypothyroidism and accelerated atherosclerosis. Observation by Y. I. Stroev

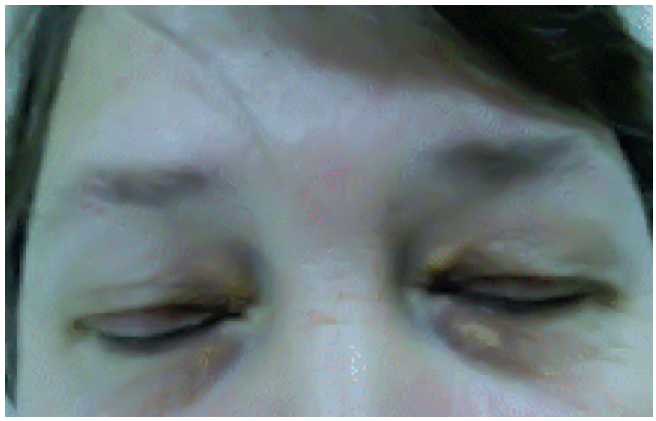

Fig. 4. Xanthelasms of eyelids in a patient with autoimmune thyroiditis and accompanying hyperlipoproteinemia. Observation by Y. I. Stroev

pertension, or in severe atherosclerosis. The moniliform (beaded) temporal arteries may be visualized in some autoimmune diseases, like periarteriitis nodosa and Horton's disease[14] (temporal or giant cell arteriitis), both have positive blood tests for inflammatory markers (C-reactive protein and erythrocyte sedimentation rate acceleration). The Horton's disease is much more common among the persons of Scandinavian — Baltic descent.

In neck area of healthy persons only carotid arterial pulsation may be observed, for example in efforts, usually synchronous with AB. However, in aortic insufficiency abnormal noticeable pulsation of cervical arterial vessels is observed. The patient may even nod involuntary, synchronously with the pulse beats (de Musset's symptom[15]) [7]. Nodding or bobbing of head may accompany aortic aneurysm as well. In these conditions other big vessels also may pulsate (brachial, radial, popliteal etc), which is characteristic for aortic insufficiency and atherosclerosis. In aortic insufficiency capillary pulsation in nail areas is also typical. Examining the cervical area, a physician should mind possible visual thyroid gland enlargement, because it is helpful in diffuse toxic goiter diagnosis if tachycardia also presents [5]. Jugular pit pulsation is observable in aortic aneurysms of various aetiology. This may be accompanied by rhythmic pulsation over thyroid cartilage or trachea (Oliver- Cardarelli's symptom)[16] [12, 14].

The veins may display on examination the picture of dilatation, congestive overfilling and even pulsation. Cervical venous pulsation is peculiar exclusively for tricuspid valve insufficiency due to blood regurgitation into right atrium and, consequently, into venae cavae in right ventricle systoles. In such a case (figuratively named in Latin: «Homo pulsans») venous pulsation may be noticeable even on peripheral veins (for example, that of hands). There exists also simultaneous liver pulsation (sometimesit may be visible ad oculus and always palpable).

Swelling of cervical veins may result from heart congestion, but also from tumor compression of the vein (s) or their thrombotic (throbmoembolic) occlusion. Commonly it is accompanied by local edema and peripheral cyanosis. In congestive liver (portal venous congestion) the net of distended umbilical veins is observable in navel area due to opening of porto-caval anastomoses («caput Medusae»)[17]. To distinguish between arterial and venous cervical pulsations, remember that former is situated to the medium from musculus sternocleidomastoideus, but later — to the exterior from this muscle. In leg vien diseases varicosity and venous swelling with the signs of inflammation are usually obvious. After rising of low extremities the veins usually abate due to improvement of blood outflow conditions.

LOOKING AT THE FACE…

[14] Horton, Bayard Taylor (6 December 1895, Gate City, USA — 6 July 1980, Rochester, USA) — an American physician, described the disease in 1932.

[15] de Musset-Pathay, Alfred Louis Charles; 11 December 1810, Paris, France — 2 May 1857, Paris, France) was a French play writer, poet of Romanticism, and novelist. He suffered from severe aortic valve insufficiency, and the symptom was first described in his case by his senior brother and biographer, French writer Paul de Musset (1804–1880).

[16] Oliver, William Silver — an English military surgeon, born 1836; died 1908, Farnborough, Great Britain; Cardarelli, Antonio — an Italian physician, born March 29, 1831, Civitanova del Sannio; died January 8, 1926. Both autors described similar signs in 1878.

[17] Medusa Gorgona — a monster of Greek mythology (Μέδουσα — in Greek means "guardian, protectress", the word Gorgona is derived from γοργός, which means "grim, dreadful") was generally described as a winged human female with living venomous snakes in place of hair. The view of swollen convoluted veins resembled that snakes to Italian surgeon, anatomist and chess player Marco Aurelio Severino (November, 1580, Tarsia, Italy — 12 July 1656, Naples, Italy), who coined this term in 1632.

Examining the patient's eyes, mind the presence of corneal lipid arch (senescent arch of cornea), typical for certain atherogenic hyperlipoprotieinemiae (especially, that of type V) and accompanying the atherosclerosis [Fig. 3].

The xanthomas of eyelids (xanthelasmas) may be revealed in atherosclerosis, especially in familiar inherited hypercholesterolemia (HLP IIa type, according Fredrickson's classification[18]) [Fig. 4].

Xanthomatosis with eruptive, plane or tendinous xanthomata of various locations is peculiar to hyperlipoproteinemiae of I, IIa, IIb, III, IV either V types, because of phagocytosis of excessive pathological lipoprotein particles by macrophages and formation of granulomata in connective tissues. These visual symptoms registered during physical examination are good reasons for profound lipidological laboratory screening in a patient. Do not neglect them!

Sometimes the face of a cardiologic patients looks so typical that it corresponds to certain well-known pattern, described in medical literature and even designated by professionals with some commonly known eponym [12]. Thus, in metabolic syndrome with hyperlipoproteinemiae of type IV–V typical patient's pear-shaped obese face with thick neck is known as «*Louis Philippe's face*»[19] [fig. 5].

Corvisart's face[20] — is a term used for typical face of a chronic patient with severe relapsing heart failure. It is a flabby face with a sleepy look, acrocyanosis against a pale yellowish skin, with crimson, slightly bulging lips and half-open mouth, of a breathless patient catching a portion of air.

The nasal or buccal vascular mesh may be observed in patients, suffering from essential hypertension or diabetes mellitus, complicated by diabetic microangiopathy.

This phenomenon is observable also in alcoholism and should be distinguished from signs of a parasitic skin disease — facial demodicosis. Scouring reveals numerous pigmented skin plaques with candle wax constituency, not removable by cleaning, commonly they are revealed in senior persons with atherosclerosis.

Fig. 5. «Portrait of king Louis Philippe I in uniform» by unknown French painter from the circle of François Pascal Simon Gérard. Oil on canvas, 78,8 x 64,8 cm. [URL http://touslesroisdefrance.fr/louisphilippei/]

BODY MASS AND THE REST OF THE VISUAL SIGNS

Finally, visual general examination must enable doctor to conclude, if a patient has excess of body mass. It is of great significance, because obesity is epidemiologically related to higher frequency of cardiovascular maladies, metabolic disorders and diabetes mellitus. Check, if possible, patient's growth and weight by measuring, in order to calculate the exact surplus of body mass by means of special tables (DuBois' tables[21] or American insurance companies' tables). Palmar and plantar surfaces of a patient are also subdued to careful examination. The thickening and squeezing of palmar or plantar aponeuroses (Dupuytren's contracture)[22] [Fig. 6] may be non-direct sign of the atherosclerosis, non-insulin dependent diabetes mellitus, or other metabolic disorders. It often occurs in advanced autoimmune thyroiditis, witnessing for acceleration of metabolic senescence. The sign is related to effects of a cytokine transforming growth factor β and may be associated with marfanoid habitus of an individual.

The palmar xanthomata may present in certain atherogenic hyperlipoproteinemiae (see above). Joint deformations may be related to systemic autoimmune disease of connective tissue or gout, which both involve cardiovascular system or accelerate the development of comorbid cardiovascular diseases.

For home reding in this field authors recommend the following guides [1–7, 9–11, 13, 16]. The next lecture will be dedicated to

[18] Fredrickson (also spelled in few papers as Frederickson), Donald Sharp — born 8 August 1924, Canon City, USA; died 7 June 2002, Bethesda, USA — an American biochemist and pathophysiologist

[19] After the name of autocratic and unpopular French king Louis Philippe I (6 October 1773– 26 August 1850), who was an object of broadly known cartoons by progressive artist and journalist Charles Philippon (1800–1861) — in revolutionary France of 1848, when the king was dismissed.

[20] Corvisart-Desmarets, Jean-Nicholas (15 February 1755, Dricourt, France — 18 September 1821, Paris, France) — a French internist.

[21] DuBois, Eugen Floyd, June 4, 1882; Staten Island, USA — February 12, 1959, New York, USA — an American pathophysiologist and endocrinologist

[22] Baron Dupuytren, Guillaume — 5 October 1777, Pierre-Buffière, France — 8 February 1835, Paris, France, a French military surgeon and anatomist. Palmar sclerosis was described by him in 1831, although earlier, in 1614 first mentioned by Swiss physician Felix Platter (1536–1614).

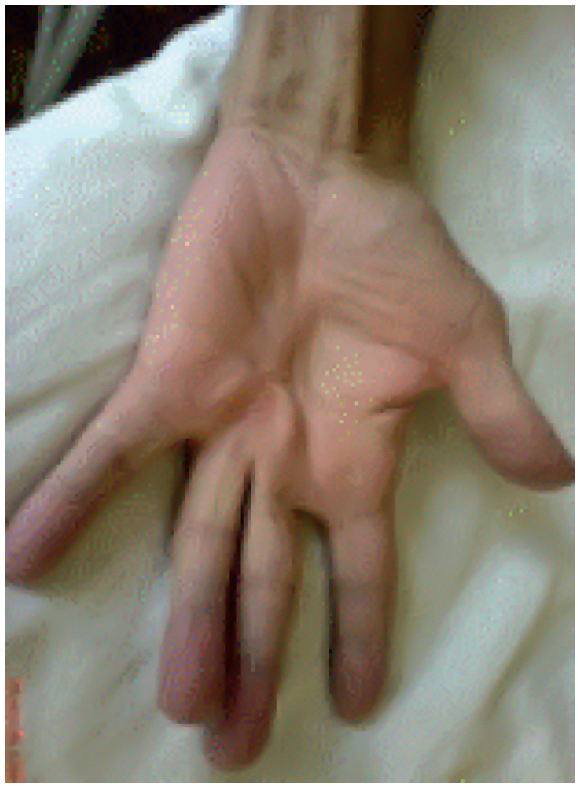

Fig. 6. Palmar sclerosis of II degree of severity in a patient with metabolic syndrome and autoimmune thyroiditis. Observation by Y. I. Stroev

methodology of palpation and medical interpretation of the palpatory revealed findings.

ACKNOWLEGEMENT

Authors are grateful to their American students for whom this project was primarily intended, especially to Dr. S. G. Huneycutt M. D., F. A.A. F.P., D. C. and to untimely deceased Dr. William G. Scoggins M. D., B. A., D. C. (1948–2010) for their valuable help in preparation of these lectures. No conflict of interest to declare. The authors equally contributed to the text, all photos are given with informed personal consent of patients.

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