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AMBULATORY ELECTROCARDIOGRAPHY

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Анотація

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The book is devoted to ambulatory ECG monitoring from technical issues to clinical use with examples of reports and interpretation of the obtained results. For students of medical faculties, functional diagnostics specialists, cardiologists, doctors of other specialties who use ambulatory electrocardiography in their work.

Contents

Don't believe what you hear just because you heard it.

Do not follow traditions just because many followed them before you.

Do not follow your teachers just out of respect for their authority and age.

Only after studying everything, only knowing for yourself that your action will serve the good of others, then accept it and live for it.

The Buddha

Abbreviation

BP - blood pressure
ACTH - adrenocorticotropic hormone
ANS - autonomic nervous system
ATP - adenorin triphosphate
AECG - ambulatory ECG (ambulatory electrocardiography)
ANS - autonomic nervous system
HRV - heart rate variability
CHD - ischemic heart disease
VT - ventricular tachycardia
MVP - mitral valve prolapse
PSVT - paroxysmal supraventricular tachyarrhythmias
PSNS - parasympathetic nervous system
LPV - late potentials of the ventricles
LPA - late potentials of the atria
PT - paroxysmal tachycardia
SB - sinus bradycardia
SVT - supraventricular tachycardia
The SNS is the sympathetic nervous system.

From the origins to the present day

Ambulatory electrocardiography (AECG) is an integral part of the diagnostic complex of modern clinical practice.

The origins of AECG date back to the late 1930s and are associated with the name of the talented scientist-experimenter N. Holter. He not only expressed, but also implemented the idea of recording human biological signals in natural conditions.

N. Holter with Ya.A. Gingerelli was the first to stimulate and record the electrical activity of the rat brain at a distance with the transmission and reception of signals through implanted electrodes by radio.

N. Holter, together with the physicist V. Glasscock, created the first electrocardiograph with a radio transmitter, which transmitted electrical signals from the heart to a distance of an entire city block. The dimensions (only weight 38.5 kg) did not allow using the electrocardiograph in practice, and therefore were reduced, but with the possibility of broadcasting the electrocardiographic signal to a distance of several meters. With a further reduction in size and the addition of a recording decoding system, the first full-fledged AECG was released.

The official birth date of AECG is considered to be 1961, when N. Holter's article "New method of heart research" was published in Science magazine. The first commercial AECG appeared the following year.

However, it took almost 40 years to summarize the results of the clinical use of AECG in the form of guidelines issued in 1999 by the American College of Cardiology and the American Heart Association.

Today, the AECG undergoes revolutionary changes in the hardware, methods and means of telemetric transmission of electrocardiographic signals, possible parameters of the number of registered channels, quality and duration of their registration, software products with the possibility of computer interpretation of electrophysiological phenomena, as well as many other aspects of service, about which more not so long ago you could only dream.

AECG today means more than simply recording an ECG in relation to a subject's natural behavior; studying the frequency of heart rhythm disturbances and their intervals, daily changes in heart rate variability (HRV), control of pharmacotherapy and implanted pacemakers, but also solving this variety of tasks on the sound philosophical basis of chronobiology and chronomedicine.

Historically, several names have been used for the AECG method, including ambulatory electrocardiography, dynamic electrocardiography, ambulatory and 24-hour ECG monitoring, and Holter monitoring.

AECG today is a long-term continuous (from several minutes and days, to months and years) and event (event recorder) dynamic recording of an electrocardiogram, including with wireless transmission, on a digital medium, with automatic and semi-automatic decoding), special devices, in including implanted.

Despite the fact that AECG has reached maturity, not everything is so simple "under the moon". If the technique of AECG is a matter of technique, then everything is much more difficult to decipher the results and you cannot do without a good assistant.

This book focuses on all aspects of AECG, from technical aspects to examples of clinical application, and thus, we hope, lives up to the expectations of its writing. This is a revised, corrected and supplemented edition, but now in Ukrainian and English.

The authors

Not just electrocardiography

AECG is not just electrocardiography. It contains information about the biological clock, neurohumoral regulation and related electrophysiological processes in the heart. Everything that is registered with the AECG in connection with the electrophysiological processes in the heart, concerns not one heart, but the circulatory system as a whole, its integration in a whole organism with its extensive connections with the (internal and external) world.

The source of electrophysiological processes in the heart

AECG is based on recording the electromagnetic field of the heart. Its source is the contracting myocardium and the conducting system of the heart, organized into a single spatio-temporal structure. Through neurohumoral regulation, this structure is integrated in the body and responds to changes in the world with the help of sensors of higher nervous formations.

Spatial-temporal organization of the myocardium

Functional "bricks" of the myocardium - cardiomyocytes - have a number of important physiological properties.

Contractility refers to the ability of cardiomyocytes to contract. Actomyosin coupling is the basis of shortening.

Excitability refers to the ability of cardiomyocytes to generate electrical action potentials in response to irritation.

Automatism is the ability of cardiomyocytes to produce self-wave electrical impulses, under the influence of which (even when isolated) they can be in a state of rhythmic contraction. This property is most developed in the conducting system of the heart, primarily the sinoatrial and atrioventricular nodes.

Conduction refers to the transmission of excitation by the conduction system from its source (in physiological conditions, the sinoatrial node) to contracting cardiomyocytes.

When, as a result of (spontaneous) depolarization, the transmembrane potential reaches the limit level, action potentials are generated by cardiomyocytes.

With the development of an action potential in each new cardiomyocyte, the wave of depolarization spreads to the adjacent ones. The transmembrane potential arising in them reaches the threshold level and is realized in the action potential. As a result, there is an avalanche-like spread of the action potential along the walls and chambers of the heart in accordance with the topology of the conduction system.

The connection between electrical impulses of the conducting system of the heart and active deformations of cardiomyocytes (and the entire myocardium) is supported by calcium ion flows. This process is energy-dependent and is provided by ATP with the transformation of chemical energy into the energy of active deformations of cardiomyocytes.

The sodium-potassium pump maintains at a stable level high values of the resting potential, which is of crucial importance in ensuring the contractile function of the myocardium.
The period of time during which the cardiomyocyte is unable to generate a propagating excitation in response to stimulation of any force is called the effective refractory period. When, in the process of repolarization, the transmembrane potential reaches 60 mV, the development of excitation spreading through the myocardium becomes possible, but the action potential occurs only in response to stronger (superthreshold) stimuli, and the speed of propagation of excitation through the myocardium is reduced. This period of time is called the relative refractory period and corresponds to the second half of the final repolarization phase of the action potential.

When repolarizing cardiomyocytes exit the refractoriness state and their conductivity is restored, the myocardium becomes heterogeneous in refractoriness and loses electrical stability. This interval was called the vulnerable period, because it is the source and direct cause of many ectopic heart rhythm disturbances.

The functions of cardiomyocytes and myocardium are regulated by hormones and neurotransmitters using various mechanisms. This is the action potential, energy supply system of actomyosin coupling, change in the number and capacity of calcium channels.

The activity of cardiomyocytes in the whole myocardium is synchronized by the conducting system of the heart and neurohumoral mechanisms.

As a result, the heart is integrated into a whole organism structurally and functionally.

Conduction system of the heart

The conduction system of the heart is represented by two nodes and numerous fibers.

The main, sinoatrial, node in physiological conditions is the driver of the rhythm. The action potentials arising spontaneously in it spread along the fibers of the conduction system to the atria and the atrioventricular node, from which they are transmitted with some delay to the fibers of the

conduction system of the ventricles (His bundle, right and left legs of the His bundle, their peripheral branches - fibers).

The conduction system cyclically generates and transmits electrical impulses to the contracting myocardium. These impulses trigger an action potential in the cardiomyocytes of the contracting myocardium. As a result, the latter are reduced.

All elements of the conduction system are automatic.

The speed of conducting electrical impulses through the conduction system is 2-5 m/s (more in large, less in small trunks). It is 10 times higher than in the atrioventricular node, as well as cells of the contracting myocardium.

The high speed of propagation of excitation along the fibers of the conduction system and its branched structure ensure that the myocardium of the atria and ventricles is almost instantly covered by the wave of excitation.

Both in the atria and in the ventricles, the excitation wave spreads from the endocardial surface to the epicardial, so their inner layers contract earlier than the outer ones.

Earlier, the apical sections of the ventricles are excited and contracted.

The conduction system, like the activity of the entire heart, is controlled by neurohumoral system.

Management of the conduction system is carried out through the interface of the sinoatrial and atrioventricular nodes, as well as other formations with the autonomic sympathetic and parasympathetic nerves and the heart's own nerves.

Regulation of the heart

Extracardiac and intracardiac regulation contours are conventionally distinguished. The first is represented by the sympathetic and parasympathetic branches of the so-called autonomic nervous system (ANS), the second by the intracardiac reflex ring.

The heart is directly innervated by the vagus nerves from the bulbar and sympathetic nerves from the thoracolumbar autonomic center.

The extracardiac department regulates the heart in accordance with the body's requests, the intracardiac along with other mechanisms ensures its functional integrity.

The ANS has a multi-level hierarchical organization with multi-sided non-linear intra- and inter-level direct and feedback connections, both within its boundaries and with the central and somatic nervous system. The highest level of the ANS is the highest vegetative centers of the cortex of the large hemispheres.

The sympathetic nervous system is part of the sympathoadrenal system, which additionally includes the medulla of the adrenal glands and other clusters of chromaffin cells, including SIF cells of the myocardium.

The influence of sympathetic and parasympathetic nerves on the biomechanics of the heart is to some extent antagonistic. Activation of sympathetic nerves increases, parasympathetic - decreases the speed of conduction of impulses through the conduction system, the contractility of the myocardium of the atria and ventricles, heart rate.

Humoral link of regulation – biologically active substances synthesized by specialized organs, tissues and cells, supplied to the myocardium by liquid media, including blood flow and intercellular ultracirculation. The vast majority of these substances are synthesized in the medulla of the adrenal glands. Their most studied representatives are norepinephrine and adrenaline.

A number of active substances are synthesized directly in the heart tissue - atrial natriuretic hormone, components of the renin-angiotensin-aldosterone system, cytokines, etc. They participate in the regulation not only of the activity of the heart, but also of the entire circulatory system.

The central nervous system controls the sense organs and the transmission of neurohumoral mechanisms of target organs, in particular blood circulation, which is open to any internal and external influences.

Regulation of R.M. Baevsky recommends considering it as a two-circuit model with central and autonomous circuits with direct and reverse control. The central circuit is the most complex multi-level system of regulation of physiological functions, which includes numerous links from the centers of the medulla oblongata to the hypothalamic-pituitary level of autonomic regulation and the cerebral cortex. Its structure is schematically represented by three levels that do not correspond to such anatomical and morphological structures of the brain as individual functional systems or levels of regulation.

The first level organizes the interaction of factors with the external environment (adaptation to external influences) - the central nervous system with cortical regulation mechanisms.

The second level ensures the systemic balance of the body and intersystemic homeostasis - higher autonomic centers (including the hypothalamic-pituitary system).

The third level ensures intrasystemic homeostasis in various systems, in particular in the cardiorespiratory system - subcortical nerve centers with a vasomotor center as part of the subcutaneous cardiovascular center, which exerts a stimulating or depressing effect on the heart through sympathetic nerve fibers.

The central circuit is identified with sympathoadrenal effects characterized by slow heart rate components and associated with nonrespiratory sinus arrhythmia. The working structures of the vegetative circle are the sinus node, the vagus nerve and its nuclei in the medulla oblongata (parasympathetic regulation). The respiratory system along the contour is perceived as an element of feedback.

A direct connection between the contours is created through nervous (mainly sympathetic) and humoral connections. The reverse direction is provided by afferent impulses from baroreceptors of the heart and blood vessels, chemoreceptors and large receptor zones of various organs and tissues. The baroreflex response is the main cause of heart rate variability (HR) in the frequency range of 0.04-0.40 Hz.

Each of the levels of regulation determines its characteristic period of fluctuations of regulated functions. The higher it is, the longer the periods of oscillatory processes, which is due to the greater number of its elements.

A drop in hemodynamics below a critical level threatens with loss of internal consciousness and the risk of sudden death. Therefore, regulatory systems duplicate and insure one, ensuring their high stability and safety. The latter are manifested not only in conditions of physiological stress, such as, for example, a change in body position, rhythm and depth of breathing, daily fluctuations in the level of blood hormones or environmental temperature, physical exertion, as well as in many, even the most important, pathological effects during distress.

A sign of high-quality regulation is a deviation of blood pressure during the transition from a horizontal position to a vertical one (active orthostatic test) by no more than 5 mm Hg. with an increase in heart rate of not less than 3 and not more than 10 (bpm). The factor of body position ceases to influence the regulation of blood circulation when gravity disappears in space conditions.

Systems and procedures

Modern commercial AECG systems consist of a set of recorders, a personal computer, a transfer unit from the recorder to the computer, including telemetry methods, software, analysis protocols and report generation. This allows pre-processing of the recording in real time, analyzing the whole set of electrophysiological phenomena, including late ventricular potentials, monitoring the work of the artificial pacemaker. Recorders in these systems independently test the quality of the signal during the research process and perform a number of other functions. If necessary, the results of registration can be transmitted through Internet channels through modems for the purpose of consulting, forming databases, etc.

Categories of registrars

There are 2 categories of AECG recorders: with permanent recording and "eventful".

Recorders with permanent recording usually have 3-12 leads, are equipped with hard media for recording in digital form, and allow continuous recording for at least 72 hours. Their weight is rapidly decreasing and in the latest models is about 100 g (Fig. 1).

Fig. 1. CardioSens ECG recorder. Weight 130 g, dimensions 80x60x18 mm3, power supply 1.2 V, 1 AA cell.

An obligatory element of any recorder is the activation button, through which the examinee records the current AECG moment in accordance with the diary protocol (taking medication, occurrence of clinical symptoms, other events). Registration and analysis of the record are separated by time.

For example, in fig. 2 presents the CardioSens AECG system with permanent recording. It provides continuous recording of the ECG signal of diagnostic quality for up to 72 hours with digital recording on a flash card.

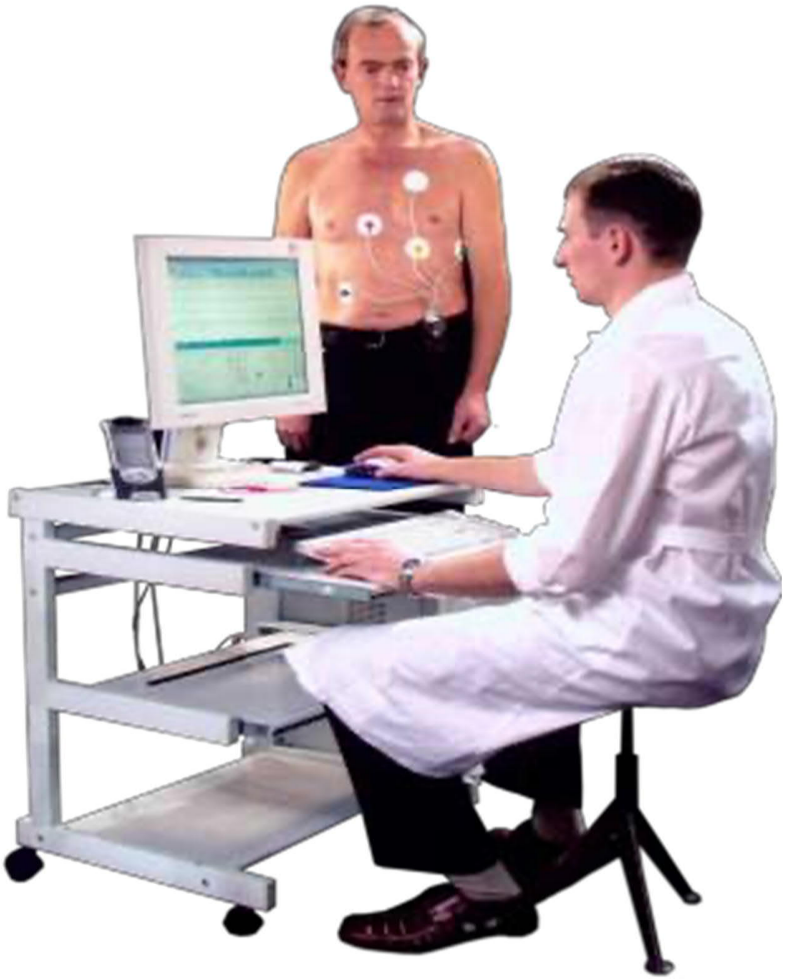


Fig. 2. CardioSens AECG computer system - CardioSens.

CardioSens was developed by the employees of the National Aerospace University "KHAI" with the participation of the employees of the medical faculty of the Kharkiv National University named after V. Karazin and is produced by the "Khaimedika" enterprise. CardioSens implements many ideas of the authors of the book.

Event recorders allow you to record individual events of limited duration in time. There are two types of recorders with minor differences. The first type is activated by the patient for a short period of time with the appearance of symptoms, and the second type is a loop (Reveal), which records the ECG continuously, but sends short recording periods (from 5 to 3000) to memory. seconds) when activated by the patient.

Event recorders allow you to transmit recorded recordings over telephone channels. They are used for a long time (many weeks) to detect rare events.

Maintenance

Maintenance is carried out with the support of representatives of manufacturing companies in accordance with warranty obligations and contracts.

Registration is the first responsible stage of AECG. Its components:

- a working recorder (elements: amplifier, cables, contacts, connections, solid-state media),

- suitable power elements,

- full charge of power cells,

- correct installation of power elements in the recorder with observance of polarity,

- high-quality disposable electrodes with an optimal conductive contact surface,

- carefully prepared skin of the examinee in the locations and fixation of the electrodes,

- choosing the place of application of the electrode on the least mobile area of the skin without natural skin folds,

- applying electrodes to dry skin and their high-quality contact with it,

- quality control of the use of electrodes by measuring the resistance between them, which should not exceed 8 KOM,

- excellent dynamic characteristics in the patient-recorder system (with unlimited patient movement),

- correctly selected number and position of leads, duration of registration,

- AECG control recording in a number of standard positions and during several standard stress tests (lying, standing, on the side, after hyperventilation, etc.),

- during the period of activity, fixation of the recorder on the side in adults and in the middle of the back in children under 5 years of age (to minimize the restriction of free activity),

- during the rest period, in order to prevent possible tension of the wires or their winding on the neck during involuntary changes in the position of the patient's body during sleep, the location of the recorder should be next to them at the gentle end of the bed,

- detailed instruction of the patient before the examination regarding the equipment,

- a high-quality diary (according to Yabluchanskyi),

- staying outside strong electromagnetic fields during the examination period,

- if the signal quality is inadequate, repeat the skin treatment, replace the electrodes and find their best location for a quality ECG signal,

- inform the patient that restarting the recorder must be accompanied by turning on the calibration signal generator,

- after completing the AECG, re-checking the condition of the electrodes and finding out from the patient whether the recorder was restarted.

Recording quality

A recording in which artifacts do not exceed 10% of its total duration is considered suitable for analysis and diagnostic conclusions. The analysis of records with a total duration of artifacts up to 30% of the total duration of the record is allowed, if they do not capture time intervals that are important (according to the diary) for diagnosis. A record suitable for analysis can be rated as high-quality, and an unsuitable one, accordingly, as low-quality.

Duration of registration

One rule for the duration of AECG recordings cannot be found for all cases. Thanks to the AECG, today we know that "disruptions" of the rhythm is the norm of a healthy person, moreover, with high daily variability.

The situation in clinical practice is even more complicated. For example, during the AECG, the patient was found to have 1,000 extrasystoles, an antiarrhythmic drug was given, and the next day, when the AECG was repeated, their number fell to 200. The question is whether to rejoice in success or not, if the number of extrasystoles in the AECG the day after tomorrow is 4,000. Should the medication be canceled or the recording repeated? Is it a proarrhythmogenic effect or the lack of effect of the drug, or is it simply the initial high daily variability of the number of extrasystoles in the patient, even unrelated to the disease?

You can answer the question by understanding that health and illness are philosophical categories, and that there are more norms in illness than in health. In addition, the number of extrasystoles, like the extrasystoles themselves, is a surrogate indicator for the quality of health.

The question of what should be the duration of the ECG should be supplemented with the question of how to organize this recording.

Our proposal is a long-term study in accordance with the task, but with the limitations of the Yabluchanskyi diary (see below).

According to the indications, the duration of the recording can be 6 hours or less when conducted at the required time of day.

When assessing the circadian fluctuations of the studied indicators, the recording time should not be less than a day.

In case of rare regular violations (1-2 times a week), at least a three-day record is required. More rare violations require transtelephone monitoring, event and even implanted recorders.

The time of the beginning of the study is important because if attention is focused on the recording of the second half of the day, it is better to start the AECG in the morning hours, and when the attention is focused on the recording of the first half of the day, it should be started from the afternoon.

Artifacts

Artifacts cannot be avoided with AECG. They are inevitable.

The results of AECG due to the registration of ECG signals during the unrestricted free activity of the patient are significantly determined by technical circumstances. The most important are the quality of electrode placement, the condition of the connections between the electrodes and the recorder, the condition of the power elements and the recorder itself.

Deformation of electrodes and detachment of their active surface from the skin, drying of conductive paste due to increased resistance and polarization potential, influence of cables as mechanical factors and peculiar antennas are an important source of distortions of the ECG signal of the heart.

These distortions concern the entire amplitude-frequency characteristics of the ECG signal of the heart, up to the oscillations of the isoelectric line, accompany the entire recording in each patient and persistently persist from study to study.

Artifacts created by the electrical fields of skeletal muscles (muscle potentials) are associated with the patient's physical activity, which manifests as high-frequency oscillations of various amplitudes and levels of regularity.

External electric fields, most often the household electrical network, are manifested by sinusoidal signals with an oscillation frequency of 50 Hz. An important contribution is static electricity from the patient's clothing. The influence of these fields increases in case of contact violations in the registrar system.

As for the artifacts generated by equipment and equipment malfunctions, the most problematic link is the lead cables. Cables have a limited useful life and must be changed in a timely manner.

The standard AECG involves a retrospective analysis of the recording, which is why the conclusion about its quality and, accordingly, the suitability of the study of the conducted research is also made retrospectively.

With modern recorders, there are practically no situations when the performed AECG turns out to be unsuitable for analysis. On the other hand, there is no case where artifacts are not observed in the recordings.

Artifacts "can't be brushed aside" and forgotten about "like a terrible dream." Careful handling of artifacts prevents false conclusions based on AECG results.

AECG leads

Modern AECG systems allow recording from 3 to 12 leads. Before the start of the study, the most informative lead system for the patient is selected, considering anatomical relationships and electrophysiological processes in the heart, as well as the pathological process. Only special cases require more than 1-2 leads.

At the same time, the ventricular ECG complexes should be of sufficiently large amplitude on at least one of the leads. Such a lead (reference) is most often close to V5. For an adequate assessment of heart rhythm disturbances, it is necessary to clearly visualize the P wave, and the right chest leads are most suitable for this. The correct selection of AECG leads is facilitated by pre-registered ECG in 12 standard leads.
У виборі відведень та накладення електродів орієнтуються на полярність і приналежність кабелів до конкретних каналів, чому є схема розведення каналів на реєстраторі.

Modified chest leads (CM – chest modified) simulate standard chest leads: CM 1 simulates V1, CS 1 – V2, CS 2 – V 4 and CM 5 – V1:

CM1 - "negative" electrode at the left edge of the handle of the sternum and "positive" - in the standard position of V1;

CC1 - "negative" electrode at the left edge of the handle of the sternum and "positive" - in the standard position of V1;

CC2 - "negative" electrode at the left edge of the handle of the sternum and "positive" - in the standard V2 position;

CM 5 - "negative" electrode at the right edge of the handle of the sternum and "positive" in the standard V5 position.

Other frequently modified chest electrodes:

CH 5 - "negative" electrode on the head and "positive" in the standard V5 position;

CS 5 – "negative" electrode in the right subclavian fossa and "positive" in the standard V5 position;

CC 5 - "negative" electrode in the V6 R position and "positive" in the standard V5 position;

CB 5 - "negative" electrode in the lower corner of the right scapula and "positive" in the standard V5 position;

CR 5 - "negative" electrode - on the right and "positive" in the standard position of V5.

Lead CM 5 ensures the registration of the QRS complex of the largest amplitude. Lead CM 1 is aimed at registering the atrial P wave, which is not always successful. Leads CS 1 and CS2 are aimed at monitoring ischemic changes of the myocardium (Fig. 3). They are also used to record the activity of the artificial pacemaker.

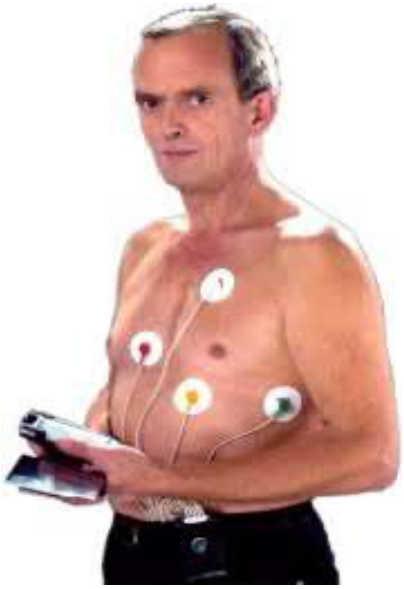


Fig. 3. Electrode stacking scheme when using a 5-electrode cable.

Indications

Any method has its indications. This also applies to AECG. They are most widely set forth in the Guidelines of the American College of Cardiology and the American Heart Association (ACC/AHA) on ambulatory electrocardiography (Ambulatory Electrocardiography Guidelines Review Committee), developed with the participation of the North American Society of Pacing and Electrophysiology (hereinafter "Recommendations").

According to the Recommendations, 3 classes of indications are distinguished:

Class I – for the benefit of which there is evidence and/or general agreement of experts,

Class II - on the benefits of which the evidence is contradictory and/or experts adhere to fundamentally different points of view:

Subclass IIa – benefit prevails,

Subclass IIb - the benefit is not obvious,

Class III - there is evidence and/or general agreement of experts regarding the lack of benefit and the possibility of harm from the use of AECG.

The recommendations contain 7 groups of indications.

The first group concerns the assessment of symptoms associated with heart rhythm disorders:

Class I: unconsciousness of an unclear nature, preconscious states, dizziness, repeating heartbeats;

Class II: episodes of difficulty breathing, pain in the chest, fatigue that do not find other explanations; neurological symptoms with presumed transient atrial fibrillation; fainting and conditions close to them; palpitations, the nature of which is established and not related to arrhythmia, but there is no effect of specific treatment;

Class III: fainting and conditions close to it, palpitations, the nature of which is established; neurological symptoms that are clearly not related to arrhythmia.

The second group is aimed at determining the prognosis based on the detection of asymptomatic arrhythmia:

Class I: no evidence;

Class IIb: previous myocardial infarction with left ventricular ejection fraction (LVEF) less than 40%, heart failure, hypertrophic cardiomyopathy;

Class III: arterial hypertension with LV hypertrophy, previous myocardial infarction with LV FI over 40%, preparation for extracardiac surgical interventions; sleep apnea and valvular heart disease.

The third group pursues the determination of the prognosis considering the detection of asymptomatic arrhythmia with the help of heart rate variability (HRV) technology:

Class I: no evidence;

Class IIb: previous myocardial infarction with LVEF less than 40%, heart failure, hypertrophic cardiomyopathy;

Class III: previous myocardial infarction with LVEF of more than 40%, assessment of neuropathy in diabetes, rhythm disturbances that do not allow for HRV analysis.

The fourth group aims to evaluate antiarrhythmic therapy, which is carried out:

Class I: assessment of the proarrhythmic effect of antiarrhythmic drugs in patients with baseline rates and reproducibility of arrhythmic events;

Class IIa: detection of proarrhythmic effects of drugs;

Class IIb: heart rate control in atrial fibrillation and flutter; detection of asymptomatic episodes of arrhythmia against the background of antiarrhythmic therapy in outpatient settings;

Class III: no evidence.

The fifth group is aimed at evaluating the function of implanted electrophysiological devices (IEFD):

Class I: assessment of clinical symptoms and syndromes (fainting, palpitations) to identify their relationship with possible disorders, such as muscle inhibition, pacemaker tachycardia; software control of frequency adaptation and automatic mode switching; detection of potential violations when other methods have proven ineffective; evaluation of drug therapy with frequent justified inclusions of an implanted cardioverter-defibrillator;

Class IIb: monitoring of patients immediately after implantation surgery and assessment of the frequency of supraventricular arrhythmias;

Class III: evaluation of the function of the IEFD with other methods of detecting violations of their work, planned control during long-term observation of asymptomatic patients.

The sixth group determines the indications for the assessment of myocardial ischemia:

I class: suspicion of variant angina pectoris;

Class IIb: pain syndrome and future operations on the vessels of patients when it is impossible to perform physical exercises, atypical pain syndrome in diagnosed coronary heart disease (CHD);

Class III: initial examination of patients with chest pain who are able to exercise; routine screening of asymptomatic patients.

The seventh group determines the conditions of research in children:

Class I: syncopal states and dizziness in children with detected heart pathology, previously documented arrhythmias and dependence on a pacemaker; syncopal conditions associated with exercise, the cause of which has not been established by other methods; hypertrophic and dilated cardiomyopathy; suspected or documented long QT syndrome; heart palpitations in children operated on for congenital heart defects and with preserved residual hemodynamic disorders; assessment of drug antiarrhythmic therapy during the period of rapid somatic growth; asymptomatic congenital complete atrioventricular block (without EKS);

IIa class: syncopal states and constant heartbeat of unknown origin in children without obvious signs of cardiac pathology; assessment of heart rhythm during antiarrhythmic therapy, especially if it is associated with a high risk of proarrhythmic effects, as well as after atrioventricular block as a result of heart surgery or catheter ablation; evaluation of the frequency-adaptive function or physiological electrocardiostimulation according to the relevant symptoms;

IIb class: examination of asymptomatic patients operated on for congenital heart defects, especially with hemodynamic disturbances and a high risk of late postoperative arrhythmias; examination of patients under 3 years of age with previous episodes of tachyarrhythmia in order not to miss asymptomatic relapses; suspicion of continuous recurrent or permanent atrial tachycardia; complex forms of ectopic activity of the ventricles at rest or during physical exertion; atypical pain syndrome with diagnosed coronary artery disease;

Class III: syncopal states and dizziness of clearly extracardiac origin, chest pain in the absence of cardiac pathology; scheduled examination for admission to sports; short heart contractions in a healthy heart, asymptomatic WPW.

According to the recommendations, AECG is not indicated for assessing the risk of life-threatening arrhythmias in patients of the following categories:

- stable angina without heart rhythm disturbances and symptoms of heart failure;

- in patients with asymptomatic mitral valve prolapse;

- in asymptomatic patients who have heart rhythm disturbances that cause loss of consciousness during professional work, which may pose a danger to the people around them.

In these cases, the probability of registering rhythm disturbances during 24-hour ECG monitoring is so small that the research result does not allow reliable conclusions to be drawn.

It is not very convenient to use lengthy recommendations, as evidenced by attempts, often successful, to present them more compactly.

Supporting the recommendations, we consider it important to pay attention to another class of indications related to chronobiology, or the so-called violations of the circadian organization (periodicity) of heart activity - over dipper, dipper, non-dipper, night peaker.

Protocol

The protocol is a complete conclusion of the conducted AECG.

The protocol should contain:

- passport data of the patient,

- diagnostics,

- The aim of the study,

- the name of the AECG recorder,

- used leads,

- total recording duration,

- the total duration of records with artifacts and their main causes,

- conclusions regarding the possibility of analyzing AECG results by artifacts and recording quality,

- ECG report: full documentation of tables, trends, all samples of normal and atypical ECG, rhythm disturbances, graphs, numerical indicators of used additional options, etc.,

- conclusion from the comments of the doctor who performed the AECG regarding individual provisions of the protocol.

Appendix to the protocol:

- output AECG record,

- diary,

- edited AECG record,

- the results of the analysis of the edited ECG according to the diary data.

The AECG protocol is a voluminous document and can reach 40 printed pages of A4 format or more.

Diary

There is no AECG without a diary.

Why exactly at this moment the rhythm becomes rarer and signs of vegetative balance shift towards the fast link of regulation? Was the patient sleeping? Did he take beta-blockers, muscarinic receptor stimulators? Just "relaxed" after a decent physical, if you didn't come up with anything, tension? These questions explain why it is impossible to conduct an AECG without a diary.

There is an opinion that a standard form of a diary is impossible: patients are different and you can't put on "the same pants" for everyone. Therefore, it is recommended that during the study the behavior of the subject should be free with the obligatory display of details of the behavior in the diary. This includes "walking, driving, studying, stress, etc.," as well as eating times, medications, symptoms occurring, with as much detail as possible.

We believe that the problems of the clinical use of AECG are largely related to the lack of a standard form of the diary, and we offer Yabluchanskyi's diary. It is based on the correct understanding and assimilation of the AEKG category of freedom as a conscious necessity.

We offer a diary according to Yabluchanskyi in a form that allows re-examination of the AECG patient according to a single plan, when with each new examination the day and night will be lived in the same way as during the first: sleep, get up, do physical exercises, have breakfast, take medicine, take tests at the same time.

DIARY OF AECG
Patient_____ Date of birth _____ Weight_____Height_____ Address: _____ Phone: _____ Study start date _____ Study start time _____ Purpose _____ Behavior plan (according to lifestyle) and planned actions for the period of AECG (reproduce in repeated studies).

Hours	Activities								**Name of the type of activity (according to the plan), event (disorder in the state of health) (indicate the hours and minutes)**
	1	2	3	4	5	6	7	8	
06-07									
07-08									
08-09									
09-10									
10-11									
11-12									
12-13									
13-14									
14-15									
15-16									
16-17									
17-18									
18-19									
19-20									
20-21									
21-22									
22-23									
23-24									
24-01									
01-02									

02-03								
03-04								
04-05								
05-06								

Where are the types of activity: 1 – sleep, 2 – eating, 3 – medication, 4 – rest, 5 – emotional stress, 6 – physical stress, 7 – stress test(s), their types, 8 – events (heartbeat, heartache, dizziness, headache, etc.).

It is important not to refuse to include in the diary experiments with physical, emotional and other types of stress characteristic of this patient, which form the basis of his life. Priority is given to experiments with the highest probability of events for which an AECG is performed. The only limitation is that they are all repeated in each study at short intervals and before triggering events that increase the likelihood of symptoms to assess the risk associated with them. It is dangerous to provoke uncontrolled experiments outside the hospital.

The standard of the diary according to Yabluchanskyi is an individual, but necessarily repeatable plan of the patient's behavior for each AECG.

AECG is the work of the patient, and therefore the diary is accepted by the doctor from his patient with a detailed report of the day lived during the study, so that unclear moments of the recording can be clarified before confirming that the study was successful and re-recording is not necessary.

Diagnostic and treatment procedures

AECG often coincides with other diagnostic and treatment procedures. This coincidence may even be planned, especially regarding the control of treatment procedures. However, there may be urgent diagnostic and treatment tasks that either cannot be performed with an overhead recorder, or where there is a high probability of artifacts and even damage to the recorder. An example can be resuscitation measures, getting the patient into the zone of influence of magnetic fields, such as, for example, MRI-tomography, cardioversion, etc. The issue of compatibility of AECG and such studies and procedures is decided individually.

There are more and more AECG diagnostic procedures compatible with it and aimed at increasing its informativeness, and it is necessary to refer to them as often as possible.

In a hospital or family doctor's clinic, if there is a treadmill, a stress test can be performed. Not even one, but several per day or for several days, before and during the intervention stages. In this way, it is possible to evaluate how, for example, the action of certain drugs manifests itself, to choose their effective dosages and schemes for the natural living conditions of this particular patient.

In the absence of a treadmill, it can be replaced by simple exercises such as walking. It is desirable to control the rhythm of exercises, for example, with the help of a watch with a second

hand. It is worth knowing that changes in physical load tolerance are valid only if the average load capacity increases by at least 20 %.

«Smart» AECG

Fully automatic analysis of AECG results is currently a fantasy, due to the variety of artifacts that cannot be avoided. Without analysis and processing of artifacts, you cannot count on a benign conclusion. Therefore, a "smart" AECG is an automatic analysis of recording results with the mandatory support of a doctor.

Signal-averaged AECG of high quality

Such AECG is aimed at identifying late potentials of the ventricles as risk factors in patients with ventricular arrhythmias, primarily after a myocardial infarction, as well as in patients with a history of ventricular fibrillation.

Physiological norms of AECG

With AECG, specialists have become more tolerant of electrophysiological and other phenomena in the circulatory system. Thus, thanks to the AECG, the physiological norms of heart rate have become much wider, and many electrophysiological phenomena that were perceived before it as manifestations of pathology are today common findings in a healthy person and acquire clinical significance only when it comes to a compromised heart.

Therefore, the AECG norms set out below make sense only when applied to the heart of a healthy person.

The lower limit of heart rate (bpm), most often during sleep:

- newborns - 70

- Children under 1 year - 65

- Children under 11 years old - 45

- teenagers under 16 years old - 40

- persons over 18 years old - 35

Upper heart rate limit (bpm):

- newborns - 220

- Children under 11 years old - 200

- teenagers under 16 years old – 190

persons older than 18 years - 220 - number of years

Elevation of the ST segment:

- up to 1 mm from the age of 10

- more than 1 mm lasting less than 1 minute

Wide fluctuations in the amplitude of the T wave in the positive range

Short periods of change in the amplitude of the P wave
Pauses (most often during REM sleep) lasting up to:

- 1000 ms in newborns,

- 1750 ms in adults

The second criterion is that they do not exceed twice the previous RR interval.

Prolongation of the QT interval regardless of heart rate to:

- 400 ms up to 1 year,

- 430 ms up to 3 years,

- 480 ms up to 15 years,

- 500 ms older than 15 years.

Non-sinus ectopic rhythms:

- supraventricular

- knotty

Extrasystoles:

- supraventricular single,

- supraventricular group in adults,

- single ventricles up to 60 years old,

- paired ventricles older than 60 years.

Paroxysm of tachycardia in adults:

- supraventricular

- ventricular

Atrial fibrillation

"Night" AV blockade of degrees I and II with "Samoilov-Wenkebach" periods.
Extrasystoles, supraventricular and ventricular, are found in every second. In old age, the frequency of their detection increases to 100%. The frequency of complex ventricular arrhythmias increases with age. In the general population, it makes up 10-15% of cases.

Short-term, up to 5 complexes, supraventricular tachycardias during AECG are observed in every fifth person. If more than 5, then only every 20th. At a heart rate of up to 120 bpm, attacks remain unnoticed or are easily tolerated.

Short-term episodes of ventricular tachycardia with a frequency of up to 180 bpm occur in 5% of cases in all age groups. They occur mainly at night.

On conventional electrocardiography, a prolongation of the AV conduction time of 200 ms or more with a fixed or "floating" duration is considered to require intervention. AECG expanded the boundaries of the physiology of atrioventricular conduction and showed that these blocks also occur in healthy people.

Therefore, an episode outside of clinical manifestations, registered during routine electrocardiography, cannot be considered a clear sign of pathological disorders. Although atrioventricular blockade of the II degree still requires control. Even if we are not talking about cases with a pronounced clinical picture of cardiovascular, vegetative and other accompanying disorders.

It should be noted that the frequency of the listed blockades in healthy people decreases with age from 10% in childhood and adolescence to 3% in old age. They are usually associated with periods of rest, most often occur at night during sleep and are associated with an increase in parasympathetic activity.

The prognostic value of pauses registered on AECG remains unclear. Although pauses longer than 3 seconds are considered critical, many subjects do not experience syncope and maintain normal hemodynamics even with longer pauses.

There are statistics of the frequency of various electrophysiological phenomena detected during AECG, but it has epidemiological significance.

A specific patient will naturally require a specific diagnosis.

AECG is much more powerful than traditional ECG

AECG means expanding the boundaries of traditional ECG and is an important tool for evaluating regulatory systems in the everyday dimension, the use of which is a difficult task.

Norms of regulatory systems do not simply "gravitate" to gender, age, environmental conditions, but also have a pronounced seasonal character, changing during the day in connection with natural activity. They are uniquely individual, as each of us is individual with the peak of individuality in the soul "I".

Heart rate variability (HRV) is one of the AECG tools that allows for the assessment of regulatory systems.

Norms of HRV indicators in healthy people:

- high individuality in calm and transitional processes;

- an increase in the total power of the HRV spectrum during the modulation of breathing and a drop in orthostatic reactions due to the predominant change in the power of the high-frequency component;

- lack of sexual (gender) differences in higher heart rate in women;

- a decrease in the total power of the HRV spectrum with age due to a faster decrease in the power of the high-frequency component;

- lower weight – greater, greater weight – lower total power and the power of the high-frequency component of the HRV spectrum with higher and, accordingly, smaller responses to metronomization of breathing and active orthostasis;

- circadian changes in the spectral indicators of HRV with an increase in the total power of the spectrum due to an increase in the high-frequency component at night and a decrease due to a relative increase in the power of the low- and medium-frequency components due to a drop in the high-frequency component;

- an increase in the total power of the HRV spectrum with an increase in the power of the high-frequency component during an active physical life with a focus on cyclical exercises;

a drop in the total power of the HRV spectrum with a decrease in the power of the high-frequency component during active mental activity in combination with light physical exertion.

Testing the biological clock

AECG manuals focus on the analysis of arrhythmias, ST, QRS, QT behavior and, among other minor points, heart rate variability (HRV). As for rhythm disturbances, each of them is described in detail, as on a standard electrocardiography (ECG). But the AECG is more than a standard EKG because it is primarily a biological clock testing tool.

About the biological and astronomical clock

We live in a world governed by the astronomical clock. Even the most important events are measured using an astronomical clock. But we know that individual age is not always equal to astronomical age. One is young in body and soul. Another is young in body, but the rest of the signs show that they is deeply old, or vice versa.

Individual age is a biological clock, but it is set to astronomical. When this process goes well - there are no health problems, when it's bad - health deteriorates, diseases get boring.

AECG creates a picture of electrophysiological processes in the heart with the help of the astronomical clock, but these processes themselves are under the control of the biological clock. The cardiac cycle, one of the internal clocks of the cyclic organization of cardiac activity, is the result and manifestation of the biological clock, and its daily organization is the result of setting the biological clock to the astronomical one.

The biological clock is a subject of study of chronobiology as a science of the temporal organization of biological systems and processes.

Basic concepts and indicators of the biological clock

• biological rhythm – a chain of repeated changes in a certain sequence of some biological indicator,

• cycle period – cycle duration or time interval between two repeating starting points of the rhythmic curve,

• frequency – the number of cycles per unit of time,

• amplitude – the difference between the maximum deviation and the average value of the rhythmic value,

• phase – any separate time part of the cycle,

• phase difference – the difference in time of the corresponding phases of two rhythms in fractions of the period or degrees,

• acrophase – the time of the maximum deviation of the measured rhythmic value during the period,

• mesor – the median of the measured rhythmic quantity,

• base level – the mode of the smallest values of the absolute values of the measured rhythmic value.

Normative chronomap of indicators

Each of the indicators of the biological clock has norms, called normative chronomap or chronodesma. Chronodesma considers the characteristics of the spectral composition of the biorhythms of the indicator and its general variability, as well as the age-related features of its chronostructure. The norms take into account age, gender, daily routine, as well as climatic features of the region of residence.

The human body is a container of an infinite number of biorhythms or biological clocks. As for AECG, we should talk about biorhythms or a biological clock generated by the cyclic activity of the heart, as well as the cyclic organization of regulatory systems affecting it, starting with the simplest local reflex rings, sympathetic and parasympathetic influences, and ending with integrative cortical functions.

Regulatory systems are open to the world, through them the astronomical clock "captures" the biological clock.

Biorhythms

Biorhythms are one of the main properties of living systems. Physiological rhythms operate in physiological conditions. Pathological conditions are a more serious matter. On the one hand, this is a violation of physiological biorhythms, or, even more often, their adjustment to the pathological process in order to provide the best possible solution (read our principle of optimality of the disease), on the other hand, the appearance of additional rhythms caused by the pathological process.

Main physiological biorhythms

• ultradian (up to 20 hours),

• circadian (two-day - 20-28 hours),

• infradian (28 hours – 10 days):

- circaseptan (3.5-10 days).

To date, about 500 functions and processes with circadian periodicity have been identified in humans..

Examples of physiological biorhythms

- ultradian – 40- and 90-minute rhythms of norepinephrine concentration in the blood plasma;

- circadian - the rhythm of replication of deoxyribonucleic acid with an acrophase of about 15 hours, the rhythm of mitotic activity of the bone marrow with an acrophase in the evening, the rhythm of daily HRV changes with an acrophase. Note that in most patients with coronary heart disease, the 12-hour rhythm of HRV changes prevails;

- circaseptan - rhythm of mitotic activity of cells, rhythm of the level of 17corticosteroids (17-CS) in urine, twelve-day fluctuations of HRV.

The whole set of biorhythms

Chronome is an invention of Halberg F. - derived from "chronos" (time) and "nomos" (rule). As the genome includes the entire set of genes, the chronome includes the entire set of biorhythms of the body.

The central place among the rhythms is the circadian rhythm, which combines the rhythms of subcellular, cellular, tissue, organ and system processes into a single coordinated temporal structure.

Some typical characteristics of the circadian period of a healthy person - body weight reaches its maximum at 18-19, heart rate - at 15-16, blood pressure - at 15-18, body temperature - at 18-00.

The amplitude of circadian fluctuations in healthy young people for body temperature is 3%, pulse – 30%, blood pressure – 25%. It is prone to the influence of social factors (depression, stress of various nature).

Today, the AECG usually lasts from 1 to 3 days, allowing determination of ultradian, circadian and, partially, infradian rhythms.

The watchman at the gate

The reaction of the human body, its systems, primarily cardiovascular, to the stress of one force and nature, other things being equal, is determined by the moment of exposure to stress. At some moments of time the reaction is positive, and at others it can be diametrically opposite, negative.

The biological clock is a gatekeeper that determines the body's response to stress at the appropriate time.

In the early morning hours, when the biological clock switches from night to daytime astronomical time, there is a conditional absolute transition from the "kingdom of the vagus nerve" to the "kingdom of the sympathetic nerve", both blood coagulation and the probability of malignant arrhythmias increase. A healthy person does not care about all this, but for a sick person, these are risks, especially fatal conditions.

Mechanisms of formation and regulation of biorhythms

Biorhythms are endogenous and possibly genetically coded. They are stable undamped oscillations with individual amplitude-frequency characteristics. Chronomes interact with the structures of the environment with a similar time periodicity, acting as external synchronizers.

The length of day and night is important for a person - the synchronizer of daily and annual biorhythms (in the first case - the absolute values of the length of day and night, in the second - their changes during the year).

All "salt" is in transitional processes

Biological rhythms, despite their exceptional stability, do not have a rigid structure. Being clearly "tied" to external synchronizers, they have a spectrum of stable states and when the frequency characteristics of the synchronizers change, they "drift" between the latter, that is, they go from one stable state to another. The transition is carried out through transitional processes. For the circadian rhythm, the duration of the transition process can be from 5 to 40 days.Transition processes create the greatest probability of violations of biological rhythms, which have received the general name of desynchronosis. It should be noted that desynchronosis is much more frequent than we imagine and is one of the clinical syndromes of most diseases.

Circadian rhythms and the "central" clock

Each cell of the human body contains genes that determine the circadian periodicity of vital activity. These intracellular "clocks" are adjusted to the periods of the change of light and dark time of the day. In other words, they have internal (endogenous) and external (exogenous) components.

Exogenous rhythmicity is superimposed on endogenous rhythmicity and thereby significantly increases the amplitude of the circadian rhythm and masks the endogenous rhythm. The intensity of physical and mental activity during the waking period, as well as the quality and structure of sleep, are significantly reflected in the amplitude of the circadian rhythm.

Even under strictly standardized conditions, the period of the intracellular clock is not strictly equal to 24 hours and is most often in the interval of 20-28 hours, which is recorded by the term "circadian" - round-the-clock. The circadian endogenous clocks are adjusted by external periodic synchronizers to the 24-hour period of the Earth's day. They can, however, be stretched and compressed over a wide range of up to 48 and 16 hours.

The human body, as a whole system, can exist normally only under the conditions of temporal coordination of all its functions, which implies the existence of a "central" clock. The "central" clock is located in the suprachiasmatic nucleus of the thalamus of the brain. It is here that nerve fibers from the optic nerve come, and it is here that various hormones (primarily melatonin) are brought with the blood, which carry out the complex "adjustment" of the clock to external synchronizers.

The regulation functions of the "central" clock are largely under the control of the pineal gland.

Circadian rhythmicity is shown by all the variety of multilevel nervous and humoral regulatory physiological processes, including autonomic nervous, hypothalamic-pituitary-adrenal and renin-angiotensin-aldosterone systems.

Each of the processes is synchronized both by external synchronizers and by mutual synchronization. Under physiological conditions, the activity of the sympathetic nervous system (SNR) predominates in the light, and the parasympathetic (PSNS) - in the dark. The concentration of norepinephrine, adrenaline, adrenocorticotropic hormone (ACTH), cortisol, endothelin in human blood plasma in circadian fluctuations shows acrophase in the morning. The acrophase of excretion of catecholamines with urine falls on the 15th hour.

Sleep

A person spends a third of his life in sleep. The quality of sleep determines the general level of health and quality of life. Sleep disturbance causes their significant decrease. More significantly, they affect the health of a sick person and become the cause of fatal disorders.

Wakefulness and sleep are related: disturbed wakefulness is often the cause of sleep disorders, just as poor sleep determines poor wakefulness.

What is a sleep?

Sleep is a physiological state that occurs mainly in the dark and is characterized by inhibition of active interaction with the environment. Conscious mental activity stops completely. Outwardly, sleep is manifested by certain stereotypical body positions, minimal motor activity, reduced reactions to stimuli, reversibility and characteristic cyclical changes in brain functions.

Cyclic organization of sleep

Sleep is a cyclical process. Each cycle consists of phases of slow and rapid (paradoxical) sleep. The phase of slow sleep is NREM sleep (without rapid eye movements) and the phase of rapid sleep is REM sleep (with rapid eye movements). In the NREM phase of sleep, slow activity is determined on the electroencephalogram, vegetative activity subsides, the pulse slows down, and blood pressure decreases. This is the phase of energy recovery. In the phase of REM sleep, rapid rhythmic activity is registered in the brain, rapid movements of the eyeballs occur, vegetative and endocrine storms occur; blood pressure, breathing rate, heart rate fluctuate; and dreams are seen at this time. The phase of NREM sleep occupies 75-80% and REM sleep - 25-20% of the duration of one cycle, which generally lasts about 60-90 minutes. From 3 to 6 cycles are observed during sleep. The last cycles in sleep can be interrupted by very short episodes of wakefulness. During sleep, natural changes occur in the brain, accompanied by corresponding reactions of vegetative and humoral regulation. They are associated with psychological processing of experience, stabilization of the psycho-emotional sphere, synchronization of systems and processes, restoration of energy potential. All this is aimed at ensuring effective wakefulness.

The need for sleep

The need for sleep varies from 4-6 to 8-10 hours or more. It is established in early adolescence and changes little during life. Physiological determinants of sleep - age, daily periodicity of day and night and its disturbances, geomagnetic influences, physical and emotional stress, etc. The depth of sleep is determined by the property of NREM sleep.

Display of sleep in AECG

With AECG, the sleep of a healthy person is represented by phases of slow and rapid sleep, respectively, with low and high HRV, respectively, with a higher and lower HR. Normally, awakening occurs in the REM sleep phase, the HR gradually increases.

Wakefulness

A person usually does not sleep during daylight hours. Waking is all active life. Everyone has their own style.

Structure is an important determinant of wakefulness. Its components are the time of onset and duration of each of the elements of wakefulness: awakening, exercise, breakfast, medication, physical and mental activity, rest, free time, preparation for sleep, etc.

The biological clock adjusts to this style, and therefore, in order to better understand the results of the AECG, it is desirable to understand in detail the style of sleep and wakefulness. Yabluchanskyi's diary contributes to this.

"Aging" of the biological clock

Genetic coding and external synchronization ensure high resistance of the biological clock to disturbing factors. With age, the amplitudes of some rhythms increase, the acrophase and other characteristics may change. In any case, it is necessary to pay special attention to the biological clock during AECG, especially in people of older age groups.

Circadian index

The simplest circadian rhythm indicator is the circadian index (CI). It is defined as the ratio of the average daytime to the average nighttime HR and is considered the main characteristic of the structure of the daily rhythm.

In healthy people older than 3 years, the CI has no sex-age differences and is in the range of 1.24 - 1.44 units. On average, in the adult population, it is estimated at 1.32±0.08 u. These CI values correlate well with the prevailing sympathetic activity during the day and parasympathetic activity during the night, as well as circadian changes of slower humoral regulation.

The daily structure of the heart rhythm in healthy people is resistant to changes in periods of sleep and wakefulness.

"Pitfalls" of CI are in arrhythmias. Tachy- and bradyarrhythmias, if they are frequent enough and are excluded from the determination of the average daily and average HR, introduce a systematic error into it. As a result, the assessment of CI is shifted, which may actually be within the physiological values of a healthy person.

Clinical significance of biorhythms

Violations in the chronoinfrastructure of biorhythms are clinically extremely important, but their role has not yet been completely revealed, and it is up to the pioneers.

Physiological norms

Norms of biorhythms are exclusively individual. To some extent, this is taken into account in the chronobiological types that are conditionally distinguished - "larks", "owls", "doves".

In "larks", the entire spectrum of physiological activity (meaning also mental) falls on the first half of the day. "Larks" go to bed early and wake up early.

In "owls", the entire spectrum of physiological activity occupies the second half of the day. "Owls" go to bed late and wake up late.

"Pigeons" are similar to "larks" in terms of their physiological activity.

Weak link

Weak link - transitional processes, which are associated with a significant change in the state of a person - psycho-emotional, physical distress.

In terms of biorhythms, it is natural to mention the transitional processes associated with their infrastructure. For circadian rhythms, it's falling asleep and waking up. An example is the morning increase in HR in patients with atherosclerosis, taking into account the localization of "problematic" atherosclerotic plaques, which is in phase with the circadian peak frequency of angina attacks, myocardial infarctions, hemorrhagic and ischemic strokes, and sudden cardiac death.

The value of changes in circadian indicators

Daily HR fluctuations are largely related to vegetative balance. The greater the sympathetic activity and, accordingly, the HR during the day, and the greater the parasympathetic activity and, accordingly, the lower HR at night, the greater the CI, and vice versa.

In sick children of the first year of life, CI is often lower than physiological standards. For example, below 1.15 in children under 3 months and below 1.2 in children 6-12 months. The reason for this is the incomplete formation of vegetative nervous regulation before this period of life, which results in a lower overall level of health. Early perinatal mortality associated with respiratory distress syndrome is well known. Not the last role in its causes belongs to incompleteness, or even defects in the formation of the autonomic nervous system.

As for adults, when the rigidity of the daily HR rhythm increases with a decrease in CI below physiological standards, it is necessary to think about diabetic neurovegetopathy, diseases of the connective tissue system, prolonged QT interval syndrome, progressive heart failure, coronary heart disease, arterial hypertension, etc. Those who died from coronary heart disease and hypertrophic cardiomyopathy had a lower CI than those who survived - 1.03 versus 1.09.

An increase in CI above physiological standards is characteristic of trained athletes. In the clinic, it is found in patients with chronic lesions of the nucleus tractus solitarius, in pathological conditions associated with blockade of afferent parasympathetic impulses with high sensitivity to efferent sympathetic stimulation, etc.

Seasonal changes in circadian parameters are observed in many, in accordance with the seasonality of exacerbations. For example, in patients with arterial hypertension and ischemic heart disease.

On the other hand, the circadian rhythm of HR is preserved in such a rare disease as fatal familial insomnia with selective degeneration of hypothalamic nuclei, which is another evidence of its partial independence from the sleep-wake rhythm.

Desynchronosis

Acute and chronic desynchronosis are distinguished. It is acute during transcontinental transmeridian flights, and in clinical practice - during sleep-wake circadian rhythm disturbances. Acute desynchronosis can transform into chronic. Chronic desynchronosis is a pathological condition caused by permanent desynchronization of the physiological functions of the human body. AECG with desynchronosis has no fundamental features and is performed in accordance with standard protocols.

Today, the following main types of desynchronization are distinguished:

- increase, decrease in amplitude;

- lengthening, shortening of the period;

- positive, negative acceleration of characteristics;

- perversion of daily periodicity;

- occurrence of ectopic events;

- a combination of the listed violations in different combinations.

Desynchronizations are early harbingers of early disorders and a mandatory component of pathological conditions.

The value of the biological clock in AECG.

The clinical value of the biological clock is not limited to desynchronization.

In AECG, it is more important to pay attention to the time of occurrence of unnatural electrophysiological phenomena (ectopic contractions, slowing of conduction of various levels of the conduction system, QT prolongation, ST shift, etc.) from the point of view of "critical" hours.

We are talking about the hours when the danger of developing paroxysmal conditions with a high risk of myocardial infarction, cerebral stroke, other disasters, including sudden death, increases.

It is known that there is no difference in the circadian profile of arrhythmias without organic damage and with organic damage to the heart - the fundamental importance of circadian disturbances for arrhythmias.

The maximum of attacks of paroxysmal tachycardia in the population will occur at 3-7 p.m. hours and the minimum at 4 a.m. Ventricular fibrillation is most likely at 4-10 a.m. and 5-8 p.m. Paroxysms of atrial fibrillation most often occur at 0-2 a.m., 8-9 a.m. and 2-4 p.m. In children of the first year of life, nocturnal paroxysms of arrhythmias prevail over daytime ones, children often die in the second half of the night. Sudden death of young athletes in 63% of cases occurs between 3-7 p.m. "Nocturnal" tachyarrhythmias are most often associated with the REM sleep phase.

About quasi-stationary and transient processes

AECG is an important carrier of information about the temporal development of cyclic electrophysiological processes in the heart, the concentrated expression of which are cyclic changes in the duration of RR intervals or, more conveniently, heart rate.

In quasi-stationary conditions (rest, rhythmic physical and/or mental activity), the duration of RR intervals (HR) fluctuates around some average value. These fluctuations are called heart rate variability (HRV).

When transitioning from rest to stress (physical stress, mental stress, including in various combinations), from health to illness, from one phase of disease development to another, in phases of recovery, if possible, during exacerbations and remissions usually the duration of the RR intervals is shortened or, accordingly, the HR increases. These changes are called transitional processes.

Fluctuations in the duration of RR intervals (HR) around some average value in quasi-stationary conditions, changes in the characteristics of these fluctuations, the direction and character of changes in transient processes are the result, and therefore, a carrier of information about the state of the regulatory functions of the human body.

Heart rate variability is a "window" into the body's regulatory processes

HRV indicates the integrative regulatory functions of the human body, the core of which is regulation. Regulation is qualitative - everything is fine with integrative functions.

HRV of an isolated and/or denervated heart, for example, during transplantation, is characterized by extremely small cycle-by-cycle fluctuations in the duration of RR intervals and heart rate, less than 1% of their average values. These fluctuations are chaotic and have the character of noise. These are the properties of HRV of the heart of a human fetus before its connection to autonomic nervous regulation. It acquires the same properties in diabetic vegetative neuropathy, some other pathological conditions, one way or another connected with its partial denervation. In all these cases, the heart rhythm becomes extremely regular and is called pendulum-like, or embryocardia.

Pendulum-like rhythm (embryocardia) in an adult, as written in textbooks on propaedeutics of internal diseases, reflects significant disturbances in heart regulation and is characteristic of severe patients that fade away.

The situation is significantly different with a heart with preserved regulation. Cycle-by-cycle fluctuations in the duration of RR intervals and heart rate here are not only much larger, they are characterized by a certain structure, far from noise.

HRV of a person with formed and preserved regulation - the result and reflection of the quality of regulation. Most often, the regulation is qualitative, if arrhythmias are rare, there are no structural deviations of the heart from physiological standards, and HRV is in the range of 3-5%.

On the "other side of the coin" to the metronomized rhythm is the HRV of more than 10%, when the regulation is in "panic" and does not calm down in any way.

Please note that HRV in the assessment of regulation in the system of existing Euro-American and national, for example, Ukrainian or Russian standards should be evaluated in quasi-stationary conditions.

The importance of evaluating transient processes

Transient processes are the result of physiological stress, pathological conditions that caused them, in connection with regulation.

In healthy people, in the physiological range of loads, the transition processes are efficient - fast and with minimal energy losses for the transition from one functional state to another.

A drop-in speed, other deviations in the transition, an increase in energy costs for the transition are signs of, if not pathological, then at least borderline conditions and require assessment and correction.

In AECG, as well as conventional ECG, the quality of regulation is assessed (should be assessed) only with respect to quasi-stationary processes or conditions in accordance with currently existing restrictions on HRV.

With transition processes, everything is more difficult, since there are still no standard methods for evaluating the transition. Some of the possible methods we offer in this book.

Separating quasi-stationary and transient processes

In the recording of the duration of RR-intervals or HRV heart rate obtained by AECG, the first step in solving the tasks of assessing the quality of regulation is its division into time intervals corresponding to quasi-stationary and transient processes.

This is helped by a diary carefully planned with the patient and implemented by him.

It is better when the AECG system is equipped with software for identifying quasi-stationary and transient processes.

HRV technology

When using HRV technology in AECG, it is necessary to take into account that HRV is, first of all, the result of the current (at the moment) state of regulation, and if we consider it as an average..., not even daily, but day or night characteristics, it is the same as testing a doll, which is not real, not alive, only a person in appearance.

In AECG, as in conventional ECG, HRV should be evaluated only at quasi-stationary intervals.

It is important to note, however, that one quasi-stationary time interval usually represents a quasi-stationary process of one type, another time interval a quasi-stationary process of another type,

and so on. In this case, one quasi-stationary process is "not related" to another, since another quasi-stationary process reflects a different state of regulation, which corresponds to a different state of the patient.

Evaluating the results of HRV at various quasi-stationary intervals in terms of the corresponding functional states of the patient, and comparing them with each other, the doctor receives much more information about the state of the patient's regulatory systems in the HRV protocol.

HRV methods

HRV research is based on the measurement of RR-intervals with the construction and further analysis of the constructed numerical series by mathematical methods.

The most used HR parameters: mean heart rate (mHR) and its standard deviation (SDNN), percentage of the number of pairs that differ by more than 50 milliseconds, consecutive normalized RR intervals for the entire recording period (PNN50), coefficient of variation (CV) , the total power of the HRV spectrum (TR) and the power of its spectral components - a) ultra low (ULF), b) very low (VLF), c) low (LF) and d) high frequencies (HF). The mentioned spectral components in humans are usually attributed to the following frequency intervals: ULF: 0-0.0033 (Hz), VLF: 0.0033-0.04 (Hz), LF: 0.04-0.15 (Hz), HF: 0.15-0.4 (Hz).

To standardize short recordings, the recommended length of RR intervals is 5 minutes, unless the nature of the study dictates otherwise. VLF interpretation should be avoided when processing short recordings of less than 5 min. For HF assessment, a recording of at least 1 minute and LF – at least 2 minutes is required.

In long recordings (daily, day, night, etc.), only the power of the ultra-low-frequency component (ULF) of the HRV spectrum is evaluated.

Interpretation of HRV indicators

There are many indicators, and the task of choosing arises, the decision of which is up to the user. Our rule is a minimum of indicators, however, in the aggregate, which characterize the state of regulation in general and in detail. In our work, we limit ourselves to the total power of the HRV spectrum and the powers of its spectral components.

The isolated spectral components of the total HRV power are associated with the powers of the regulatory influences of the humoral and autonomic nervous systems. Efferent vagal activity is considered an important component of HF, but it is significantly influenced by the respiratory center (cardiorespiratory arrhythmia), and direct subordination to cortical functions mediates direct central effects on the cardiac spectrum. In the same way, LF is often considered as a marker of sympathetic modulation, associated, to a large extent, with baroreceptors. In reality, it also depends on vagal and humoral influences, since functionally regulation is a single indivisible orchestra. The LF/HF ratio is often viewed as a marker of sympathetic balance, but this must be done in light of the limitations noted. In the existing system of ideas, VLF is associated with thermoregulation, humoral, to a large extent, metabolic processes and sympathetic tone. There is evidence that VLF and LF are sensitive indicators of metabolic process control and well reflect energy deficit states.

Connecting the spectral components of HRV with regulation, it is more natural to talk, however, not about its vegetative parasympathetic and sympathetic, humoral and other links, but about fast, slow, very slow and ultraslow regulation.

The interpretation of frequently used indicators of HRV is summarized in the tables 1-3.

Table 1.

Indicators of heart rate variability in the time domain.

Indicator	Dimensionality	Name	Physiological interpretation
HR	1/min	Heart rate	The average frequency during the study period as an integral characteristic of the level of functioning of the circulatory system
mRR	ms	The average length of the RR interval	The average length of cardiac contraction during the study period, which integrally evaluates the level of functioning of the circulatory system
sdRR	ms	Standard deviation of the mean length of the RR interval	A measure of the power of high-, low-, and ultra-low-frequency effects on short recordings and the entire spectrum of neurohumoral effects on long recordings
rMSSD	ms	The square root of the root-mean-square deviations of successive RR intervals	A measure of the power of high-frequency neurohumoral influences, often associated with the activity of the parasympathetic link of the autonomic nervous system
pNN50	%	Number of consecutive RR intervals differing by more than 50 ms as a ratio to the number of all RR intervals	A measure of the power ratio of high-frequency and low-frequency neurohumoral effects, often associated with the ratio of the activity of the parasympathetic and sympathetic links of the autonomic nervous system
HRVTi	-	Triangular index as an integral of the density of the distribution divided by the maximum of the density of the distribution of RR-intervals	A measure of the power of the effects of neurohumoral regulation
CV	%	Coefficient of variation	A measure of the power of the effects of neurohumoral regulation

MxDMn		The difference between the maximum and minimum values of cardio intervals	The range of effects of neurohumoral regulation
Mo		Mode	Most often, the value of the level of functioning of the cardiovascular system
AMo		Amplitude of mode	Most often, the value of the influence of the sympathetic link of regulation
SI		Stress index of power of regulatory systems	The degree of tension of the regulatory systems (the degree of predominance of the activity of the central mechanisms of regulation over autonomous ones)

Table 2.

Indicators of heart rate variability in the frequency domain.

Indicator	Dimension ality	Name	Physiological interpretation
TP	ms^2	The total power of the HRV spectrum	A measure of the power of the effects of neurohumoral regulation
ULF	ms^2	The power of the ultra-low-frequency domain of the daily HRV spectrum	A measure of the power of ultra-low-frequency effects of neurohumoral regulation, the exact origin of which is not established, is associated with periodic
VLF	ms^2	Very low frequency domain power	A measure of the power of very low-frequency effects of neurohumoral regulation, associated with thermoregulation, the renin-angiotensin system and the sympathetic nervous system
LF	ms^2	The power of the low-frequency domain	A measure of the power of low-frequency effects of neurohumoral regulation, associated mainly with sympathetic and partially parasympathetic links of regulation
LFnorm	%	Normalized LF to LF +HF	The relative level of the low-frequency link of neurohumoral regulation is associated with the relative level of the sympathetic link
HF	ms^2	The power of the high-frequency domain	A measure of the power of high-frequency effects of neurohumoral regulation, associated mainly with the parasympathetic link of regulation

HFnorm	%	Normalized HF on LF +HF	The relative level of the high-frequency link of neurohumoral regulation is associated with the relative level of the parasympathetic link
LF/HF	-	Power ratio of low and high frequency domains	A measure of the balance of low- and high-frequency links of regulation, often considered as a measure of sympathovagal balance
VLFav	ms^2	The average value of the power spectrum of the very low-frequency domain	The average level of activity of very low-frequency effects of neurohumoral regulation is associated with the effects of suprasegmental divisions of the sympathetic link of autonomic regulation
LFav	ms^2	The average value of the power spectrum of the low-frequency domain	The average level of activity of low-frequency effects of neurohumoral regulation is associated with the effects of the activity of the vasomotor center
HFav	ms^2	The average value of the power spectrum of the high-frequency domain	The average level of activity of high-frequency effects of neurohumoral regulation is associated with the effects of the parasympathetic link of autonomic regulation
IC	-	Index of centralization	The degree of centralization of heart rhythm control (predominance of the activity of the central circuit of regulation over the autonomous one)

Table 3.

Indicators of autocorrelation analysis.

Indicator	Dimensionality	Name	Physiological interpretation
CC1	-	The value of the first coefficient of the autocorrelation function	The degree of activity of the autonomous regulation circuit
CEO	-	The number of shifts of the autocorrelation function before obtaining the value of the correlation coefficient is less than zero	The degree of activity of the central regulation circuit

Removal of restrictions

The strict limitations of sinus rhythm HRV technology according to existing standards have led to its use in heart rhythm disorders, especially atrial fibrillation and flutter, only in the period between attacks. Despite the fact that even in this sense it is extremely effective, the limitation is not a dogma. Below, we will show that with constant atrial fibrillation and flutter, other arrhythmias, HRV indicators behave like sinus rhythm, and this technology is quite applicable in their study when additional restrictions are imposed.

Methods of transient processes

The study of transient processes makes it necessary to consider the sequence of RR-intervals from the standpoint of dynamic nonlinear systems, the central place of which is occupied by the concept of stability of the system and its measure - Lyapunov exponents. Since the application of the latter is associated with difficulties caused by a significant stochastic component, the method of local Lyapunov indices - M-indices has been developed. The method allows the analysis of AECG recording sections of arbitrary length and degree of non-linearity and gives statistically significant results resistant to the stochastic component.

Indexes are determined using the method:

M0 is the average value of the largest local Lyapunov exponent in the studied section of HRV. The sign of the index M0 shows what prevails in the studied area of HRV: "+" - acceleration, "-" - braking. And the value is the degree of nonlinearity of the HRV plot against the function exp(t). If the studied segment of the site is linear, then M0 =0. For stationary recordings of HRV, M0 will be close to zero.

M 1 is an index that allows you to evaluate the temporal changes of the HRV record: whether the movement in the phase space leads to the expansion or contraction of the phase flow. The sign of the index M 1 shows what happens to the phase flow in the studied area of HRV: + - expansion, - compression. And the value is the degree of change of the phase flow in the studied area of HRV against the function exp(t). When the studied segment is stationary or the changes are strictly periodic in nature, M1 =0.

M is a composite index characterizing the non-linearity and non-stationarity of the transient process in the studied section of the HRV recording. The "+" sign means a non-linear movement with a predominant increase in heart rate; "-" means non-linear movement with a predominant decrease in heart rate. The size of the M index shows the degree of non-linearity and non-stationarity of the transient HRV process.

Examples of transitional processes with explanations:

Transition exp(t) (Fig. 1.): M0 =1; M1 = 1; M=1 is a positive accelerating transition with pronounced nonlinearity and expansion of the phase space. Nonlinear unstable transition. If a random component is added to the main transition function exp(t), for example, a Gaussian random process with a maximum amplitude of ±10% of the value of the main function (Fig. 2) is imposed, the values of the M-indices will change only within 3-5%, which means about the resistance of the selected event to noise. For fig. 2.: M0 = 0.97; M1 = 0.97; M = 0.95%.

An example of a weakly unstable but nonlinear transient process is given in fig. 3. This is the result of the composition: exp(t)+10% random component+sine wave half-period. For such a

transient process M0 = -3.5; M1 = 0.025; M = 0.1, that is. a positive retarding transition with significant nonlinearity and a weak stretching of the phase space or a strongly nonlinear weakly unstable transition.

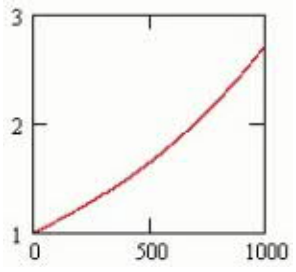


Fig. 1.

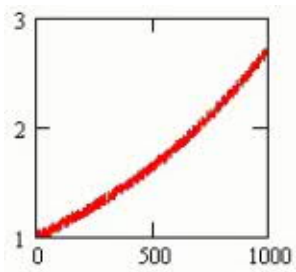


Fig. 2.

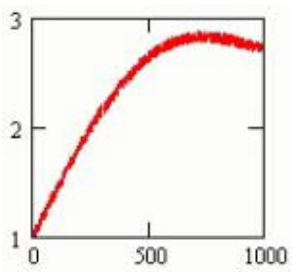


Fig. 3.

Fig. 4 shows a transition in the form of a composition of a linear function with a 10% random component, for which M0 =0.004; M1 = 0.74; M=0.0031 is a positive linear transition with phase expansion. A linear indifferent stable transition. Examples of stable transient processes are shown in fig. 5. - 6.

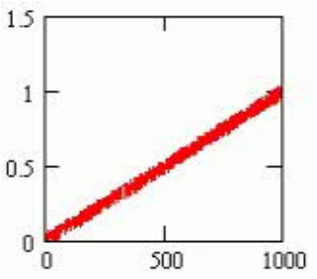


Fig. 4.

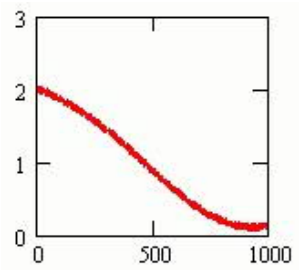


Fig. 5.

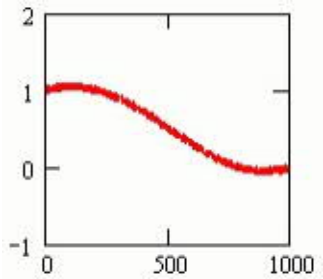


Fig. 6.

Linear transition +10% random component + half-period of cosine (Fig. 5.): M0 =-0.045; M1 = -0.83; M=-0.036 – negative weakly inhibiting and almost linear transition with phase compression. Weakly nonlinear close to an indifferently stable transition.

Linear transition + exp(-2t)+10% random component+half period of cosine (Fig. 6): M0 =-0.83; M1 = -0.67; M=-0.57 – negative retarding nonlinear transition with phase compression. Non-linear transition.

Additional help in describing the transition process can be provided by its general geometric and time characteristics (according to Fig. 7.):

- HxT (c*n) – the size of the transition process curve. It characterizes the duration and scope of the transition process;
- h(c) is the value of the transition level. It characterizes the average change in frequency during the transient process. The sign h determines a positive, negative or indifferent transition: - h>>0 – positive; h<<0 – negative; |h|<0.01 - indifferent.

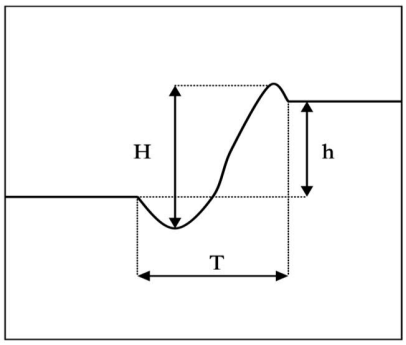


Fig. 7.

A separate item should discuss the method of using the proposed M-indices. First of all, this is a direct application for the description of transient processes. However, it is equally important to use them to assess the limit of application of the standard spectral analysis of HRV and thereby increase the reliability of the analysis results.

Obviously, the calculation of M-indexes for the entire HRV recording will allow us to establish whether the recording is stationary. If not, the results of the spectral analysis for the entire recording cannot be considered correct, and in this case the M-indexes will help to find areas of the rhythmogram where the spectral analysis can be applied. An example of such automatic division of the rhythmogram is shown in fig. 8 for the daily interval of the Holter HRV recording (79% of the recording is quasi-stationary; 21% is non-stationary):

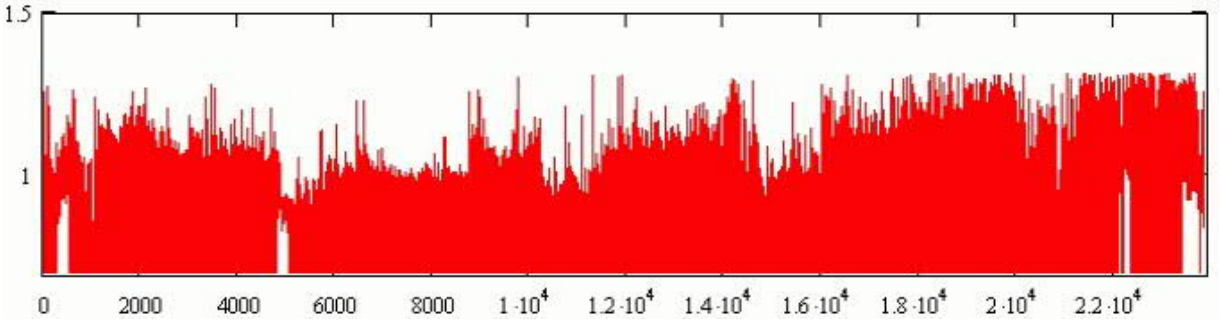


Fig. 8. Allocation of stationary (79%) and non-stationary (21%) sites.

In the daytime recording of the VRR and the night interval of the VRR recording (Fig. 9.): 95% of the record is quasi-stationary; 5% - non-stationary:

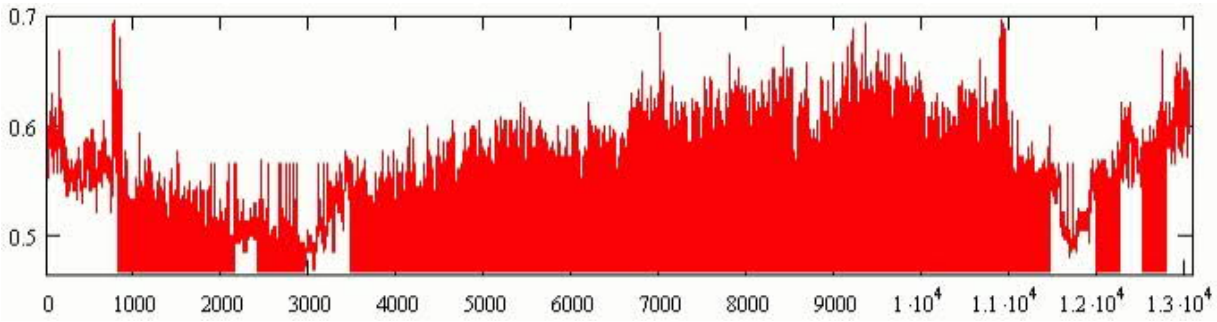


Fig. 9. Selection of stationary (95 %) and non-stationary (5 %) areas in the night HRV recording.

Standard conclusion protocol

Assessment of regulation based on HRV and transients research data is not a simple task and requires deep knowledge and considerable experience.

It is natural for beginners to resort to the help of standard protocols, which, on the one hand, formalize the conclusion and, on the other hand, ensure its completeness and internal consistency. These protocols, however, are also useful to the experienced practitioner.

We have tested and use a protocol that can be presented in a generalized form as follows: quality (influences) of regulation, power (influences) of regulation, balance (influences) of regulation; physiological nature of transient processes when escaping from a quasi-stationary state, when returning to a quasi-stationary state; risk of fatal conditions.

The most important indicators of the quality (impact) of regulation are the power and power balance of their branches. The strength of regulatory influences is estimated by TR HRV at quasi-stationary epochs. The balance of medium and fast branches of regulation on them is evaluated in relation to LF/HF. It is also possible to find the balance of other frequency ranges of adjustment.

The physiological nature of transient processes is assessed by M-indexes at the stages of evading the quasi-stationary state and returning to it by conducting and analyzing the results of functional tests (stress tests).

This point can have a different degree of completeness: from exclusion from the protocol to its filling with the results of many functional, drug, mental and other stress tests determined by the specific conditions of the study.

The quality of transitional processes and their resistance to external and internal stress factors, the possibility of choosing interventions that have the necessary effects on regulation, and the evaluation of the effectiveness and correctness of the selected patient management tactics are determined.

The choice of a specific type of influence on the physiological functions of the subject's body is determined by their content and tactical tasks.

The same applies to the study of mental influences on regulation, which acquire exceptional importance when testing mentally dependent individuals.

The risk of fatal arrhythmias and states is assessed by the indicators of the time domain and TR HRV, as well as by the ratio of the powers of the spectral components that form it, for quasi-stationary epochs of the studied physiological functions.

A useful conclusion layout:

1. Quality (impacts) of regulation: a. Power (influences) of regulation: high, moderate, low. exhaustion b. Balance (influences) of regulation: developed regulation: predominance of slow, slow and medium, slow and fast, medium, medium and fast, fast regulation.

2. Physiological stress reactions of regulation (active and passive tilt test, modulated breathing, manual dynamometry, Valsalva test, etc.). Reactions to stress (including in acute

pharmacological tests) and the return to a quasi-steady state after stress are evaluated by the methods of studying transient processes based on the above indicators.

3. Risk of fatal arrhythmias and conditions. Low, moderate, high, very high. It is calculated by indicators, as well as the quality (impacts) of regulation.

For the first case, it is possible to recommend the implementation of the conclusion layout as shown in Table 1.

Table 1

Diagnostic algorithm for functional assessment of the state of the regulation system.

A fragment of the conclusion formula	Indicator and value limits
1. Power 2. висока 3. moderate 4. low 5. critically low	TP More 3000, 1500-3000, 300-1500 Less 300
2. Balance of regulatory links Norm Predominance of slow (long-frequency link) regulation Predominance of slow and medium (medium-frequency link) regulation Predominance of slow and fast (high-speed link) regulation Predominance of medium and fast with balance Medium regulation is preferred Fast regulation is preferred	VLF, LF/HF 0.4-0.6, 1.3-1.9 More 0.6, 1.3-1.9 More 0.6, over 1.9 More 0.6, Less 1.3 Less 0.4, 1.3-1.9 Less 0.4, More 1.9 Less 0.4, less 1.3

3. Physiology of transient processes - reaction to stress - Return to the previous quasi-stationary state	Classification criteria based on M-index values:|M|<0.03 – linear, stationary; 0.3>|M|>0.03 - weakly nonlinear; 3>|M|>0.3 - non-linear; |M|>3 is highly nonlinear. Interpretation of the transition process:M0>0 - accelerate; M0<0 deaccelerate; M1>0 - expand; M1<0 - shrink; M>0 - unstable; M<0 - stable.
4. Risk of fatal conditions 4.1 Low 4.2 Moderate 4.3 High 4.4 Very high	TP, LF/HF More than 0.025, less than 1.9 0.015-0.025, less than 3.5 Or any, from 3.5 to 15.0 0.010-0.015, less than 1.0 and more than 3.5 or any, more than 15.0 less than 0.0

The main thing is in quasi-stationary and transient processes

Quasi-stationary and transient processes are the result and "mirror" of regulation. Regulations tend to change, so general conclusions about its state can only be made in terms of fuzzy sets.

AECG and medical interventions

AECG is primarily a diagnostic tool. Diagnosis is the first step in medical interventions. The next step is the control of medical interventions.

A person is an individuality, and there is no way to measure it with standard measures, and therefore you can't stock up on recipes for all cases, and it remains to apply recommendations only with smears.

A fairy tale is a lie, but there is a hint in it

A fairy tale is a biological clock, a hint that it should be used, and not only in diagnostics, but also in the control of medical interventions in the first place. The most is done here in pharmacotherapy, which is commonly called chronotherapy.

Chronotherapy methods:

- preventive – adaptation of the intervention (the moment of administration of the drug) to the time of reaching the acrophase of the indicator, the functions in which the intervention is carried out,

- imitative - compliance with the patterns of daily fluctuations of indicators, functions that are interfered with,

- "imposing" a rhythm - intervention (introduction of a drug) every time at a certain time of the day to impose on the patient's body a rhythm of changes in the indicator and/or function, as close as possible to the set rhythm (note that to the rhythm of a healthy person in a sick person striving is far from always correct - the principle of disease optimality).

In the cardiology clinic, the indicators by which physiological functions are evaluated are most often systolic and diastolic blood pressure (BP), stroke volume and/or ejection fraction of the left ventricle, HR, and some others.

An example of a preventive intervention is the administration of an antihypertensive drug so that its maximum effect occurs before the patient's blood pressure begins to rise. An example of simulated intervention is the morning appointment of glucocorticoids in accordance with the patient's body's natural morning increase in the secretion of its own steroids. An example of "imposing" a rhythm is the introduction of a melatonin drug that mimics the natural hormone of the pineal gland, which is involved in the regulation of the biological clock, at the same time, to adjust the patient's biological clock (very often in astronauts).

There are many advantages of chronotherapy. The first thing is to achieve a clinical effect with smaller course and daily doses with the minimization of side effects of the drug. The most important are patient discipline, aiming to achieve higher treatment results. It is impossible to overestimate the decrease in depressive moods, the determination to recover, the strengthening of a positive attitude towards life, and the restoration of social contacts.

Regulation control

In health, even more so in illness, qualitative regulation is important, one for health, and one for illness, in exact accordance with the Principle of Optimality of Disease.

Regulation in the disease is carried out through the great adaptation syndrome of H. Selye.

If everything is fine with the regulation, we monitor and do not intervene, if something is wrong, we optimize.

With a decrease in the power of regulation, primarily rapid, which is assessed using HRV technology, disturbances in transient processes with tachycardic HR reactions and hypertensive blood pressure reactions, beta-blockers are the means of first choice.

Angiotensin-converting enzyme (ACE) and angiotensin receptor (ARA) inhibitors, unlike beta-blockers, reduce the power of regulation, while having a similar effect on blood pressure as beta-blockers.

Each drug has a regulatory effect, so in their use it is advisable to also evaluate how they affect the regulation, so as not to worsen.

If you have such a tool as an AECG in your hands, the best advice is to choose the medicine, dose, moment, frequency and terms of appointment.

Measure seven times, you may not have to cut

We are talking about arrhythmias, intervention in which is required, if only "pathological", and, first of all, due to the pathological process that gave rise to them. Control of arrhythmias using AECG is a difficult task due to their significant daily fluctuations. Therefore, for example, Gieca et al.

suggest that antiarrhythmic treatment be considered effective if and only if the conditions for reducing the number of ES by more than 75% are met; decrease in the number of paired and early (R on T) ECs by more than 90; reducing the number of monomorphic types of polymorphic extrasystoles to a maximum of 2; disappearance of episodes of ventricular tachycardia.

Table 1 may also be useful, which lists the criteria for evaluating the effectiveness of antiarrhythmic therapy in terms of changes in the number of ventricular EC depending on the time that has passed since its initiation.

Table 1,

Criteria for evaluating the effectiveness of antiarrhythmic therapy by the number of reductions in ventricular extrasystoles.

Day from the start of therapy	The degree of reduction in the number of extrasystoles		
	the total number of		the total number of
1-7	63	90	95
8-90	79	94	98
91-365	92	98	98
More 365	98	99	99

It is important to note that AECG is not performed in patients with paroxysms of sustained ventricular tachycardia in the anamnesis with the number of ventricular ECs less than 60 per day. Here, to control the effectiveness of therapy, ventricular stimulation should be used before and after taking the tested drugs.

It is important to make a caveat that effective antiarrhythmic therapy indicates only it, because the obtained antiarrhythmic effect does not mean a decrease in the probability of paroxysmal rhythm disturbances. An example is the results of the CAST study, when flecainide or encainide reliably reduced the number of arrhythmias, but the frequency of sudden death in patients increased. The price is too expensive.

Any antiarrhythmic drug can increase the activity of an existing arrhythmia or provoke a new one. The criteria for the proarrhythmogenic effect of antiarrhythmic agents are:

- at least a fourfold increase in the total number of ventricular ES per day;

- at least a tenfold increase in the number of paired ECs and episodes of unstable ventricular tachycardia;

- appearance of previously unregistered persistent ventricular tachycardia or its new persistent morphological form.

The presence of just one criterion is considered sufficient for a conclusion about the proarrhythmogenic effect of the medicinal product being tested in the patient. It is this drug that is being tested, because another, even from this group, may turn out to be completely antiarrhythmic without any adverse reactions. Here, as in dermatoglyphics, everything is extremely individual.

The risk of proarrhythmogenic effect of the drug is especially high in cases of:

- prolongation of the QT interval;

- primarily inherited predisposition;

- paroxysms of ventricular tachycardia or ventricular fibrillation in history;

- very low the ejection fraction of the left ventricle;

- during treatment with cardiac glycosides.

Stigmas of the proarrhythmogenic effect of an antiarrhythmic drug are:

- return of arrhythmia in the form of atrial flutter with conduction to the ventricles 1:1 in the treatment of AF;

- appearance of pirouette-tachycardia (torsades de pointes);

- emergence or strengthening of existing dysfunction of the sinus node;

- development or strengthening of atrioventricular blockade;

- development of intraventricular blocks.

Extremes are not justified

Inactivity during paroxysms and persistent AF tachycardia is not justified. Antiarrhythmic therapy of benign arrhythmias is not justified. A calm attitude towards malignant and detected malignant ventricular arrhythmias is also not justified.

The task of therapy of these arrhythmias is not only in their application, which is not always possible, as, for example, in permanent AF, but also in improving the prognosis and increasing the patient's quality of life with a guarantee of treatment safety.

Time to rewrite scrolls

Modern recommendations with enviable scrupulousness clone the classification of antiarrhythmic drugs by E.M. Wanghan Williams in the modification of J.C. Harrison, which divides them into four classes:

Class I - blockers of sodium channels,

Class II - blockers of beta-adrenergic receptors,

Class III - drugs that increase the duration of the action potential and myocardial refractoriness,

Class IV - blockers of calcium channels.

Some classes consist of subclasses with a significant list of antiarrhythmic drugs, and a false impression is created that not only arrhythmias, but also all antiarrhythmic drugs without exception should be known perfectly. The false impression is that at least Class I drugs have lost their former appeal. Large clinical studies have repeatedly revealed and confirmed a significant increase in overall mortality and the frequency of sudden death in patients with organic heart lesions on the background of taking class I antiarrhythmic drugs. They effectively eliminated the current arrhythmia, but caused malignant ventricular arrhythmias. All is not well with drugs of class IV. They can be used in emergency situations. But they are no less effective, and most importantly safe, with class II and III drugs.

Conclusion - the main classes of antiarrhythmic drugs are II and III. At the same time, in the II class, the absolute priority belongs to cardioselective beta-blockers, and the leader in the III class is amidarone. It's time to rewrite the scrolls.

"Superior" drug

Amiodarone belongs to class III antiarrhythmic drugs. Its antiarrhythmic effects are not limited to increasing the duration of the action potential and myocardial refractoriness. Classification is schematization, and its authors wrote in their preambles that each of the drugs has a range of actions, and that the classification records the most important of them.

Turning to pharmaceutical reference books, we find that amiodarone is not only able to block potassium channels and prolong the action potential, slowing repolarization, but also, like class I antiarrhythmics, inactivates fast sodium channels, and, like class IV, can block slow calcium channels. It also has a non-competitive inhibitory effect on alpha- and beta-adrenergic receptors (class II), showing a sympatholytic effect. It turns out that amiodarone has the properties of all four classes of antiarrhythmic drugs. But even this is not enough. The drug inhibits the synthesis of thyroxine in the thyroid gland and its conversion to triiodothyronine, and therefore affects metabolic pathways in general. The structure of amiodarone is similar to thyroid hormones, the iodine content in it is about 37% of the molecular weight.

In the same reference books, we will find that amiodarone reduces the myocardial oxygen demand, causes the expansion of coronary arteries and has an anti-anginal effect. Unlike most antiarrhythmics, its negative inotropic effect is insignificant.

The exceptional place of amiodarone among antiarrhythmic drugs allows us to consider that it is a "superior" drug.

It is important to consider that the triggering and supporting mechanisms of tachyarrhythmia are often far outside the heart. Research on HRV during daily ECG monitoring has convincingly shown that tachyarrhythmia paroxysms are preceded by cataclysms in regulatory systems, such as episodes of autonomic sympathetic and parasympathetic storms. The sources of these cataclysms can be formed at different levels of hierarchically organized neurohumoral regulation with an incredible interweaving of vertical and horizontal connections, modifying, individualizing, to be extremely precise, the same tachyarrhythmia in different patients.

If the mechanisms of tachyarrhythmias are innumerable, and the implementation of a specific one is a matter of chance, the most optimal here is the "superclass" antiarrhythmic drug amiodarone. Learn to use amiodarone skillfully, and it will not let you down.

Malignant and potentially malignant ventricular arrhythmias recorded in a patient with heart disease are evidence of a high probability of their recurrence with a high risk of sudden cardiac or, rather, arrhythmic death. The first episode of AF is a sign of impending chronic AF with its own problems. The first preventive medication here is amiodarone.

In patients of older age groups with severe organic heart changes (atherosclerotic, post-infarction, post-myocardial cardiosclerosis; severe myocardial hypertrophy in hypertension, etc.), distress, such as, for example, surgical interventions, can cause the development of malignant and potentially malignant diseases. Their prevention consists in prophylactic administration of amiodarone for the appropriate period (perioperative).

Malignant and potentially malignant arrhythmias, like AF paroxysms, are much easier to prevent than to treat. And not only because their appearance threatens urgent situations that are not easy to deal with, but also because once they have arisen, they have the ability to arise again. At the most inconvenient time, under the most inconvenient circumstances.

In international and national recommendations for cardiopulmonary resuscitation, emergency cardiac care, treatment of AF, malignant and potentially malignant ventricular arrhythmias, amiodarone is the drug of first choice. It is recommended to limit the use of other antiarrhythmic drugs due to their proarrhythmogenic and depressant effects on cardiac biomechanics and systemic hemodynamics.

Priority belongs to injectable amiodarone in a dose of 5-7 mg/kg with intravenous infusion over 30-60 minutes.

Despite the fact that amiodarone is the first treatment for AF, malignant and potentially malignant arrhythmias, in clinical practice there is a need to combine it with other antiarrhythmic drugs. These are, first of all, cardioselective beta-blockers with high bioavailability. The best among them is betaxolol, which has no intrinsic sympathomimetic activity and vasodilatory effects and which can be prescribed only once a day.

Adding beta-blockers to amiodarone significantly reduces the risk of death. As for the beginning of heart rate treatment, the combination has a greater effect.

The problem for the doctor when he is "not in order" with amiodarone and does not use its antiarrhythmic power to its full potential is tachycardiac AF.

We are taught that AF is chaos, disorganized chaotic contractions of groups of atrial cardiomyocytes with incomplete atrioventricular block. Consequences of chaos - arrhythmogenic dilation of the heart, developing and progressing heart failure, thrombus formation in the atria, thromboembolism, stroke, sudden death.

Our team proved that AF is deterministic chaos.

Have you wondered why patients with AF, if there is no heart failure of high degrees, do not complain of shortness of breath even during serious physical exertion?

The answer is not in seven seals - in AF, the regulation is preserved, and patients, as with sinus rhythm, show full-fledged frequency-adaptive reactions of the heart.

We also showed that with AF, amiodarone not only controls the HR, but also restores the heart's rate-adaptive reactions to physical, psychoemotional, and other stress. This is the whole point of its positive impact on the quality of life of patients.

Schemes of clinical use of amiodarone are many, and each is good in its own way. The most standard and proven one is laid out in the instructions for use, and it is most natural for a doctor with little personal experience to focus on it.

Our advice is to be closer to the patient, cooperate with the patient, listen to the "call" of his heart, and you will find the scheme that is the most suitable for a specific clinical situation. No less individual than your patient's fingerprints.

Amiodarone is the first and most reliable drug in pharmacological cardioversion of acute and paroxysmal persistent AF, control of HR in persistent AF, its prevention in the perioperative period in older patients, in malignant and potentially malignant.

If you are interested in atrial fibrillations

The optimal resting HR in patients with sinus rhythm of 60-80 per 1 min is not always a guideline for the level of the controlled rate of ventricular contractions in AF. It is usually taken for a frequency of ventricular contractions of 80-90 per 1 minute.

The reason is clear - ineffective diastole of the heart in AF due to the loss of the atrial systole phase, and, as a result, the hemodynamic need for a higher frequency of ventricular contractions in heart failure, which accompanies it like a shadow.

In AECG, additional criteria are added - the average hourly frequency of ventricular contractions should not be more than 80 per 1 minute, and there should not be episodes with an average frequency of ventricular contractions over 1 hour of more than 100 per 1 minute.

In the control of the frequency of ventricular contractions in atrial fibrillation, it is important that during physical exercise it increases in the physiological range, and its increase in level corresponds to the degree of exercise.

Also, arrhythmias

We are talking about blockades that are treated not only by surgical, but also by therapeutic methods.

AECG allows monitoring the results of both surgical and therapeutic treatment. It plays an important role in monitoring the effectiveness of pacemakers.

Examples of standard AECG reports

The examples of a healthy volunteer and patients with various forms of AF on the background of cardiac disease are given, with an assessment of the effectiveness of therapy. The first two examples with full graphical results, and the rest only with the display of daily HR changes.

Example 1

Full name: S-ov V., male, 21 years old. Diagnosis: healthy. The purpose of the study: daily changes of the ECG in a healthy volunteer. 3 Duration of observation: 24 hours and 10 minutes.

The total duration of the artifacts: 3 hours 04 minutes (12%) Conclusions regarding the possibility of analyzing the AECG results: the total duration of the artifacts does not exceed the critical one, the analysis is possible.

Report of ambulatory ECG monitoring

Hourly summary of Holter monitoring.

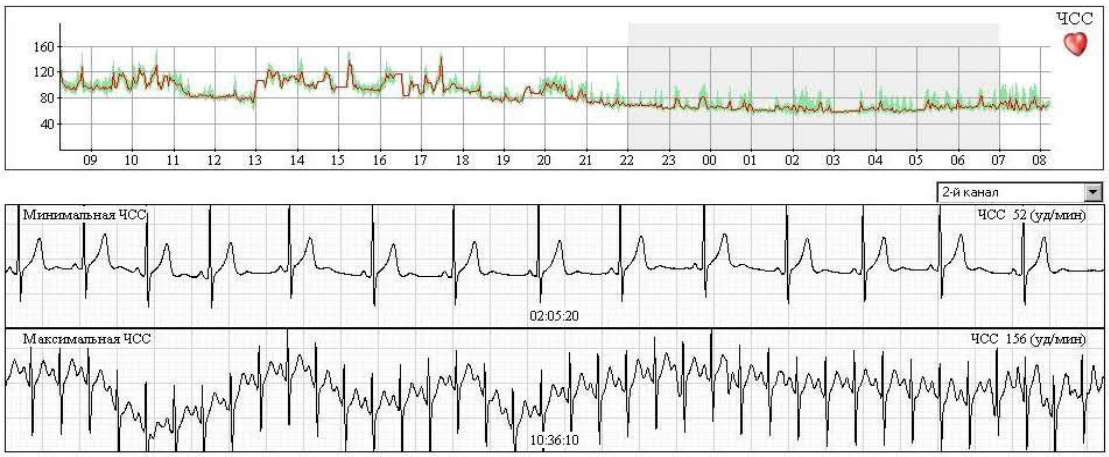
General characteristics of the rhythm		*Ventricular extrasystole*	
Average HR during the day	89 bpm	Average HR during the day	89 bpm
Average HR at night	65 bpm	Average HR at night	65 bpm
Max. HR	156 bpm at10:36	Max. HR	156 bpm at10:36
Min. HR	52 bpm at 02:05	Min. HR	52 bpm at 02:05
Max. hourly HR	110 bpm at 13:14	Max. hourly HR	110 bpm at 13:14
Min. hourly HR	61 bpm at 03:04	Min. hourly HR	61 bpm at 03:04
Disturbance of rhythm		*Supraventricular extrasystole*	
Tachycardia	139 in 28900 s.	Tachycardia	139 in 28900 s.
Bradycardia	Not found	Bradycardia	Not found
Pauses	Not found	Pauses	Not found
Max. pause	Not found	Max. pause	Not found

Hourly summary of Holter monitoring.

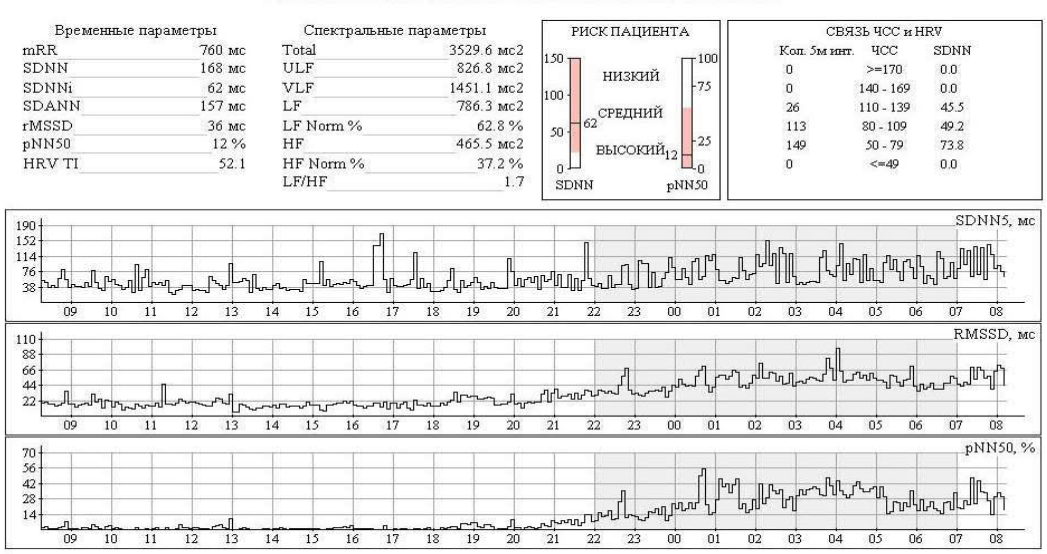
Time	HR, bpm			VE	Bigem.	Coup.	VE runs	SVE	SV coup.	SVT	Tachy	Brady	Pause	STj episode		
	min.	avg.	max.											1к	2к	3к
8.14	85	101	150	0	0	0	0	0	0	0	14	0	0	2	3	4
9.00	82	101	138	2	0	0	0	0	0	0	22	0	0	1	4	4
10.00	91	109	156	1	0	0	0	0	0	0	2	0	0	0	4	6
11.00	76	88	122	0	0	0	0	0	0	0	7	0	0	0	6	7

12.00	63	81	114	0	0	0	0	0	0	0	4	0	0	0	0	0
13.00	92	110	134	0	0	0	0	0	0	0	0	0	0	0	3	3
14.00	85	103	142	0	0	0	0	0	0	0	4	0	0	1	2	3
15.00	82	99	153	0	0	0	0	0	0	0	20	0	0	1	4	4
16.00	79	101	135	0	0	0	0	0	0	0	5	0	0	0	9	3
17.00	82	100	149	0	0	0	0	0	0	0	12	0	0	1	3	3
18.00	73	84	122	0	0	0	0	0	0	0	11	0	0	0	5	7
19.00	73	83	113	0	0	0	0	0	0	0	8	0	0	0	0	0
20.00	72	90	119	0	0	0	0	0	0	0	15	0	0	0	8	9
21.00	58	72	115	0	0	0	0	0	0	0	2	0	0	0	4	2
22.00	61	68	95	0	0	0	0	0	0	0	0	0	0	0	2	2
23.00	58	69	103	0	0	0	0	0	0	0	2	0	0	0	0	0
0.00	58	66	97	0	0	0	0	0	0	0	1	0	0	0	0	0
1.00	54	63	82	0	0	0	0	0	0	0	0	0	0	0	0	0
2.00	52	63	99	0	0	0	0	0	0	0	0	0	0	0	1	0
3.00	56	61	91	0	0	0	0	0	0	0	0	0	0	0	0	0
4.00	55	62	99	0	0	0	0	0	0	0	1	0	0	0	0	0
5.00	57	66	94	0	0	0	0	0	0	0	1	0	0	0	0	0
6.00	59	68	96	0	0	0	0	0	0	0	2	0	0	0	0	0
7.00	56	68	104	2	0	0	0	0	0	0	5	0	0	0	0	0
8.00	59	67	78	0	0	0	0	0	0	0	0	0	0	0	0	0
Avg.		79														
Total				5	0	0	0	0	0	0	139	0	0	6	58	57

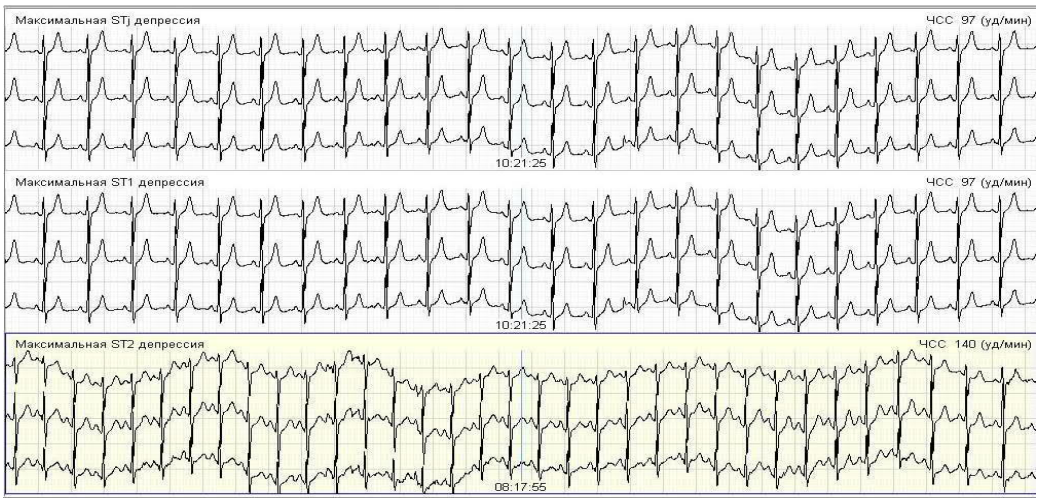
Dynamics of daily heart rate, minimum and maximum heart rate.



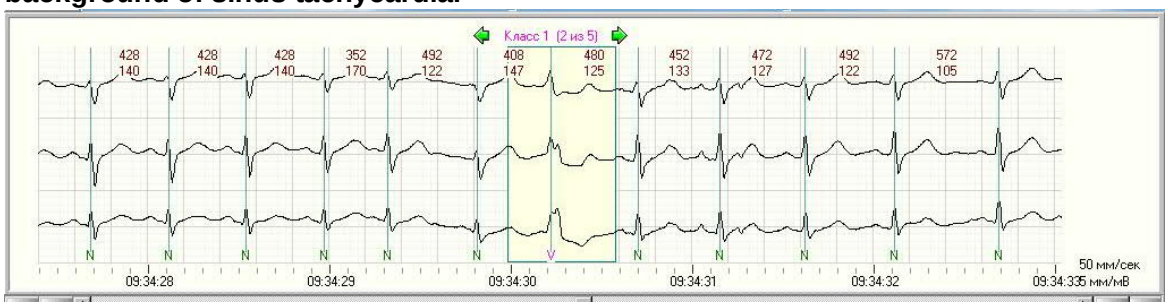
Temporal characteristics of heart rate variability.



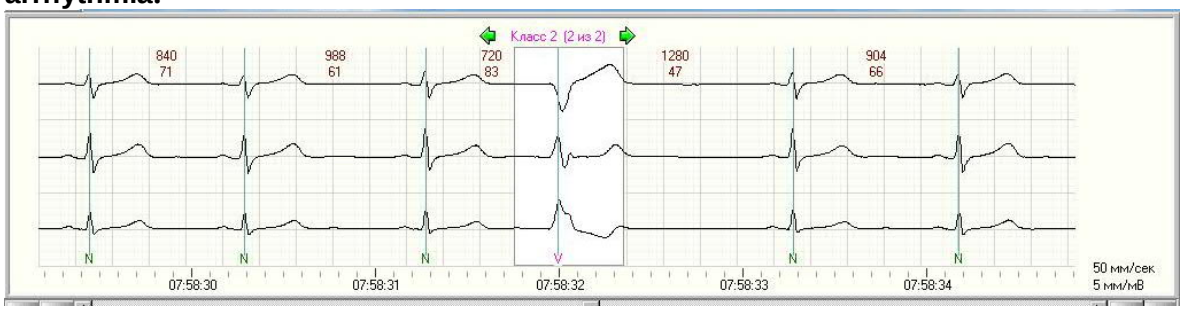
Fragments of maximum STj, ST1 and ST2 depression.



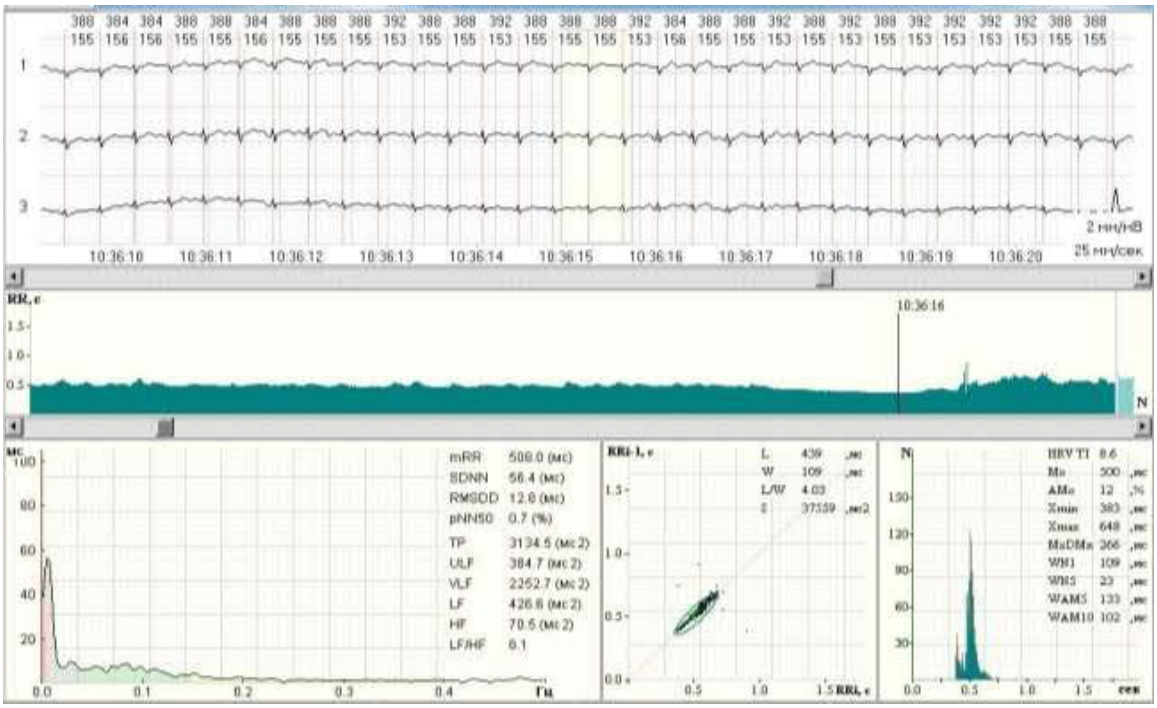
Fragment of an ECG with a single interpolated ventricular extrasystole on the background of sinus tachycardia.



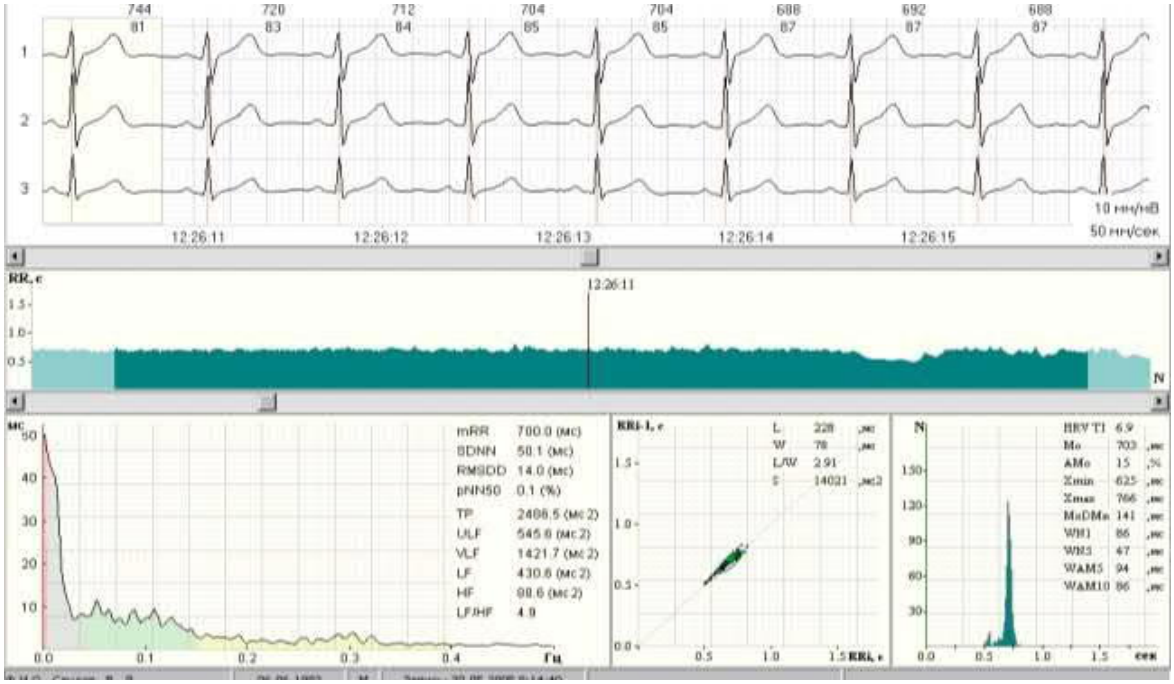
Fragment of an ECG with a single ventricular extrasystole on the background of sinus arrhythmia.



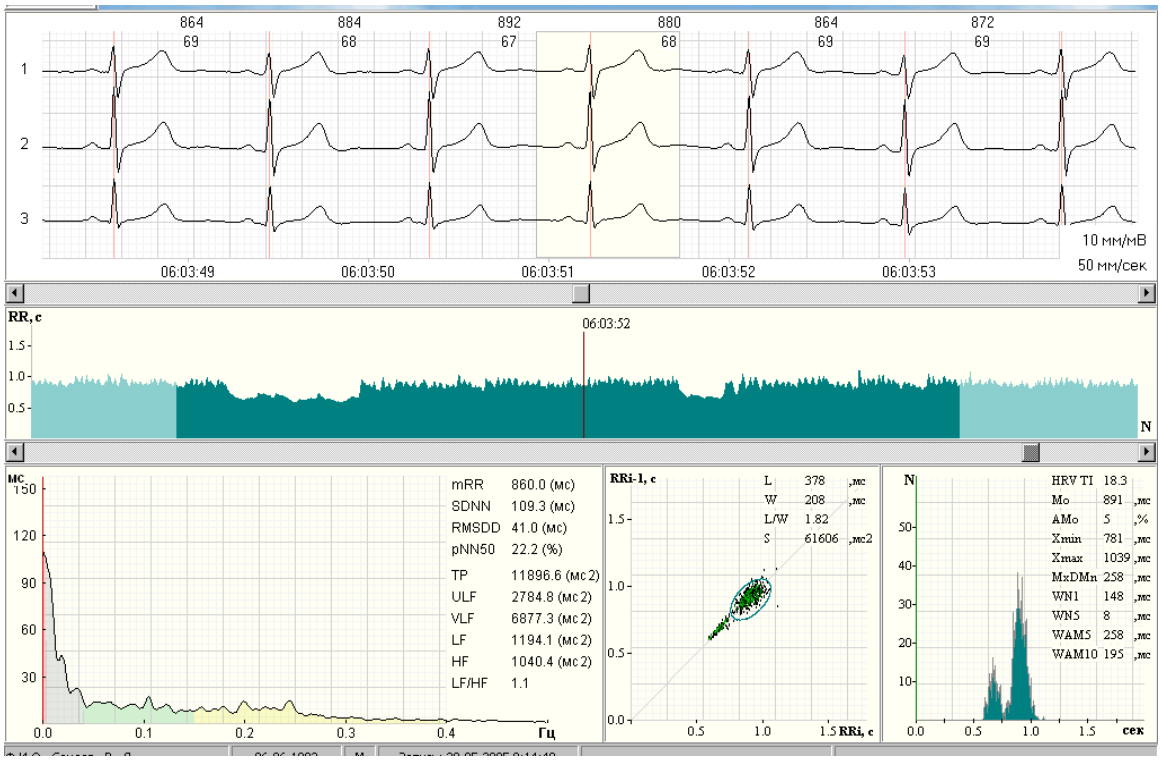
A fragment of the ECG, rhythmogram and HRV indicators during normal physical activity: TR - 2486.5 ms2, LF/HF - 4.9.



Fragment of ECG, rhythmogram and HRV indicators during significant physical exertion: TR - 3134.5 ms2, LF/HF - 6.1.



Fragment of ECG, rhythmogram and HRV indicators during sleeping: HR – 67-69 bpm, LF/HF - 1.1



Conclusion

1. Heart rate

Average heart rate values at the upper limits of the norm (89 beats/min during the day and 65 beats/min during sleep) with preserved circadian periodicity (CI - 1.37). A total of 139 tachycardias with a total duration of 28,900 seconds were registered. from 1 to 22 per hour. Most tachycardias are an adequate response to physical and emotional stress. The maximum heart rate (156 beats/min at 10-36 hours) does not exceed the submaximal one for this age (norm of 159 beats/min) and was registered during physical exertion (according to the diary - climbing at a moderate pace to the 6th floor). During sleep (10:30 p.m. to 7:20 a.m.), 7 episodes of tachycardia with an increase in heart rate not exceeding 107 bpm were recorded.

2. Rhythm

Against the background of sinus rhythm, 5 ventricular ECs (L0 and L1 class) were registered with a pre-ectopic interval of 513-775 msec. (on average 668 msec.), associated with emotional stress and physical exertion.

3. HRV

Borderline low TR and increased LF/HF during normal exercise (2486.5 ms 2 and 4.9, respectively), increased TR and LF/HF during significant exercise (3134.5 ms 2 and 6.1, respectively) and increased TR with a decrease LF/HF during night sleep (11896.6 ms2 and 1.1, respectively) – 3134.5 ms2, LF/HF – 6.1).

4. S-T segment

Ischemic S-T changes during observation were not detected. Episodes of depression of the S-T segment during the day are caused by tachycardia. At night there is a "vagus" S-T elevation (02:08) (see Examples).

Conclusions

Borderline average daytime and average nighttime HR with preserved circadian variability. Non-fatal rhythm disturbances: ES of low gradations according to Lown and Wolf. Normal frequency-adaptive responses to physical and emotional stress. Low power of the HRV spectrum with increased LF/HF as a manifestation of daytime sympathicotonia. Adequate response of the ANS during nighttime sleep.

Example 2

Full name: R-ch R., male, 57 years old.

Diagnosis: Arterial hypertension of the 1st stage, 2nd degree of severity. The first episode of atrial fibrillation, tachysystolic form. HF stage I, FC IV with preserved systolic function of the left ventricle.

The purpose of the study: to determine the nature and clinical significance of heart rhythm disorders.

AECG registrar: "CardioSens" (Kharkov).

Leads: CM-5, CC-1, CC-3.

Duration of observation: 18 hours 24 minutes.

Total duration of artifacts: 31 min. (1.7 %).

Conclusions about the possibility of analyzing the AECG results: the total duration of the artifacts is much shorter than the critical one, the analysis is possible.

Report of ambulatory ECG monitoring

Hourly summary of Holter monitoring.

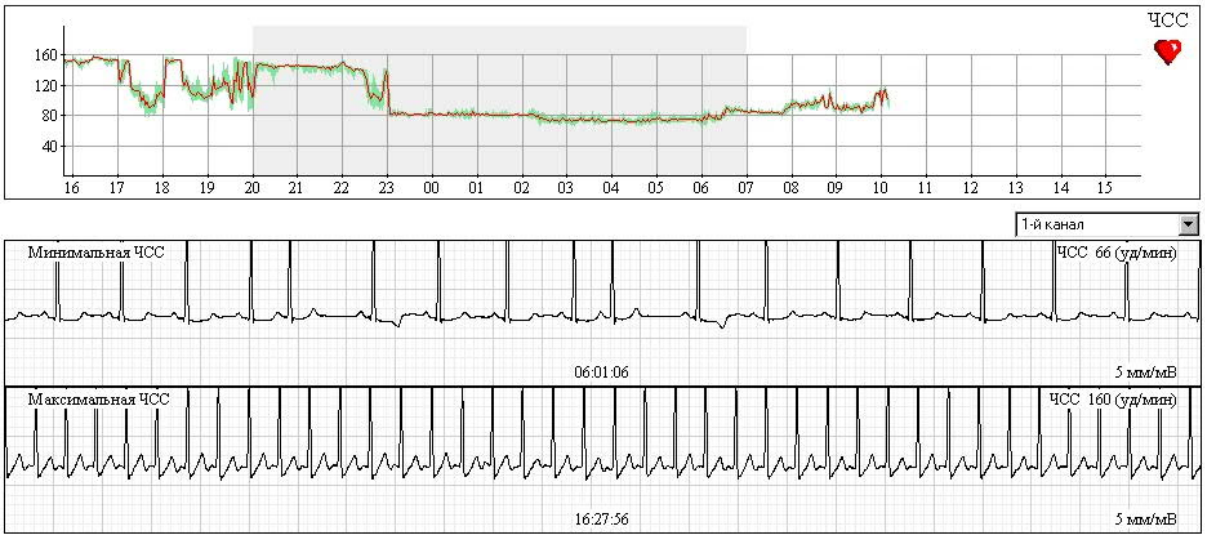
General characteristics of the rhythm		*Ventricular extrasystole*	
Average HR during the day	110 bpm	Average HR during the day	110 bpm
Average HR at night	88 bpm	Average HR at night	88 bpm
Max. HR	160 bpm at 16:27	Max. HR	160 bpm at 16:27
Min. HR	66 bpm at 06:01	Min. HR	66 bpm at 06:01
Max. hourly HR	153 bpm at 16:17	Max. hourly HR	153 bpm at 16:17
Min. hourly HR	73 bpm at 04:05	Min. hourly HR	73 bpm at 04:05
Disturbance of rhythm		*Supraventricular extrasystole*	
Tachycardia	33 in 17854 c	Tachycardia	33 in 17854 c
Bradycardia	Not found	Bradycardia	Not found
Pauses	Not found	Pauses	Not found
Max. pause	Not found	Max. pause	Not found

Hourly summary of Holter monitoring.

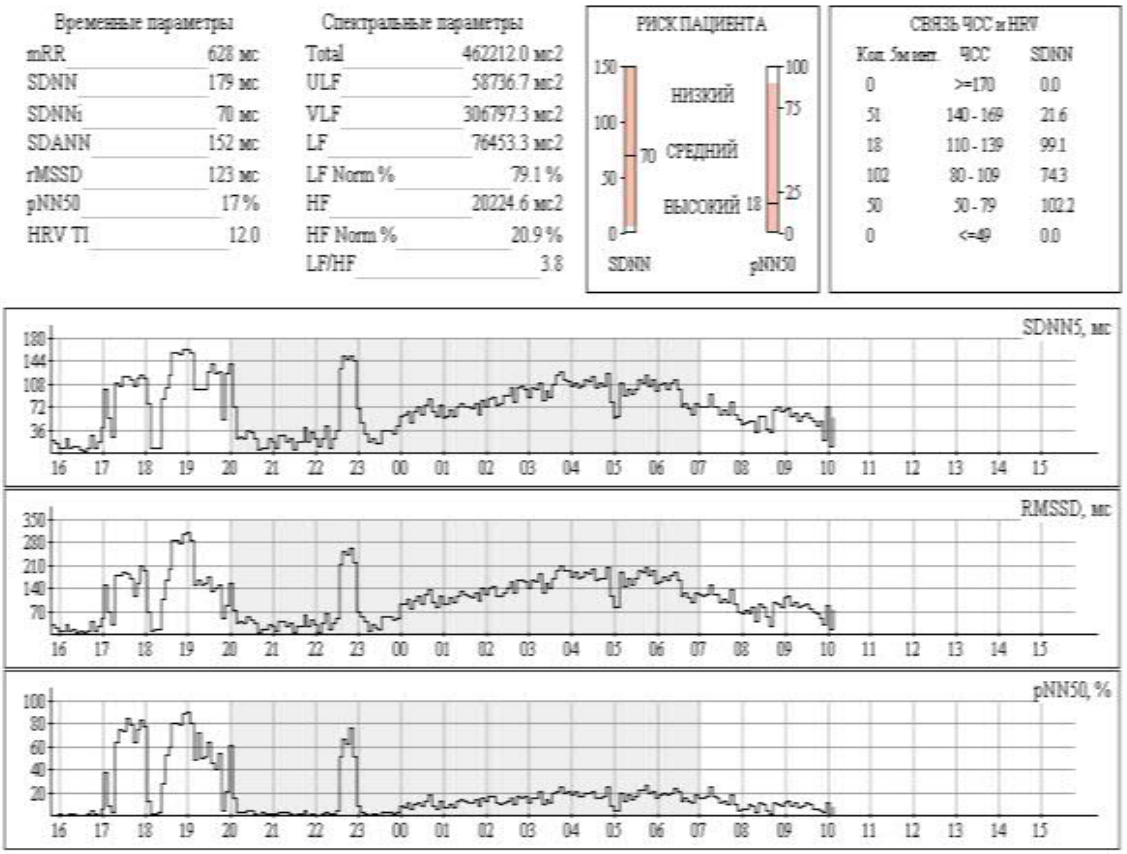
Time	HR, bpm			VE	Bigem.	Coup.	VE runs	SVE	SV coup.	SVT	Tachy	Brady	Pause	STj episode		
	min	avg	max											1к	2к	3к

15.47	143	152	156	2	0	0	0	0	0	0	1	0	0	1	1	2
16.00	140	153	160	8	0	0	0	0	0	0	0	0	0	7	7	5
17.00	78	112	153	155	0	0	0	0	0	0	1	0	0	14	3	0
18.00	91	123	156	65	0	0	0	0	0	0	6	0	0	10	3	2
19.00	86	118	156	58	0	0	0	0	0	0	19	0	0	7	3	1
20.00	93	143	150	22	0	0	0	0	0	0	1	0	0	7	13	2
21.00	132	144	150	65	0	0	0	0	0	0	0	0	0	1	4	0
22.00	87	125	152	62	0	0	0	0	0	0	3	0	0	6	7	1
23.00	77	83	136	6	0	0	0	0	0	0	2	0	0	4	3	1
0.00	75	82	90	1	0	0	0	0	0	0	0	0	0	0	0	0
1.00	77	81	87	0	0	0	0	0	0	0	0	0	0	0	0	0
2.00	66	77	89	0	0	0	0	0	0	0	0	0	0	0	0	0
3.00	66	74	85	2	0	0	0	0	0	0	0	0	0	0	0	0
4.00	66	73	86	0	0	0	0	0	0	0	0	0	0	0	0	0
5.00	69	75	83	0	0	0	0	0	0	0	0	0	0	1	0	0
6.00	66	82	93	1	0	0	0	0	0	0	0	0	0	0	0	0
7.00	79	85	96	3	0	0	0	0	0	0	0	0	0	0	0	0
8.00	85	96	115	39	0	0	0	0	0	0	0	0	0	10	0	0
9.00	81	92	115	23	0	0	0	0	0	0	0	0	0	3	2	0
10.00	88	103	117	9	0	0	0	0	0	0	0	0	0	3	0	0
Avg.		97														
Total				521	0	0	0	0	0	0	33	0	0	74	46	14

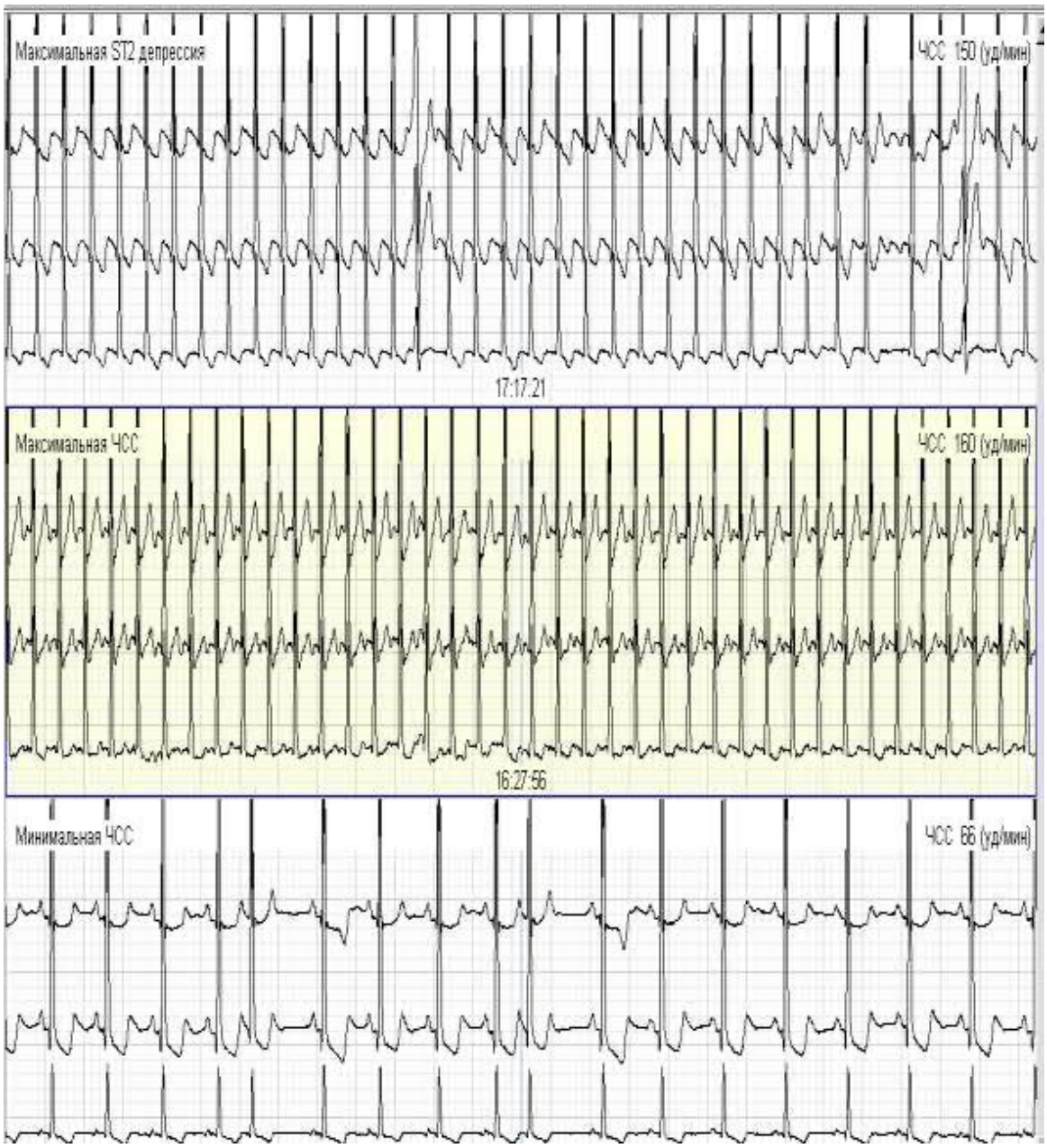
Dynamics of daily heart rate, minimum and maximum heart rate.



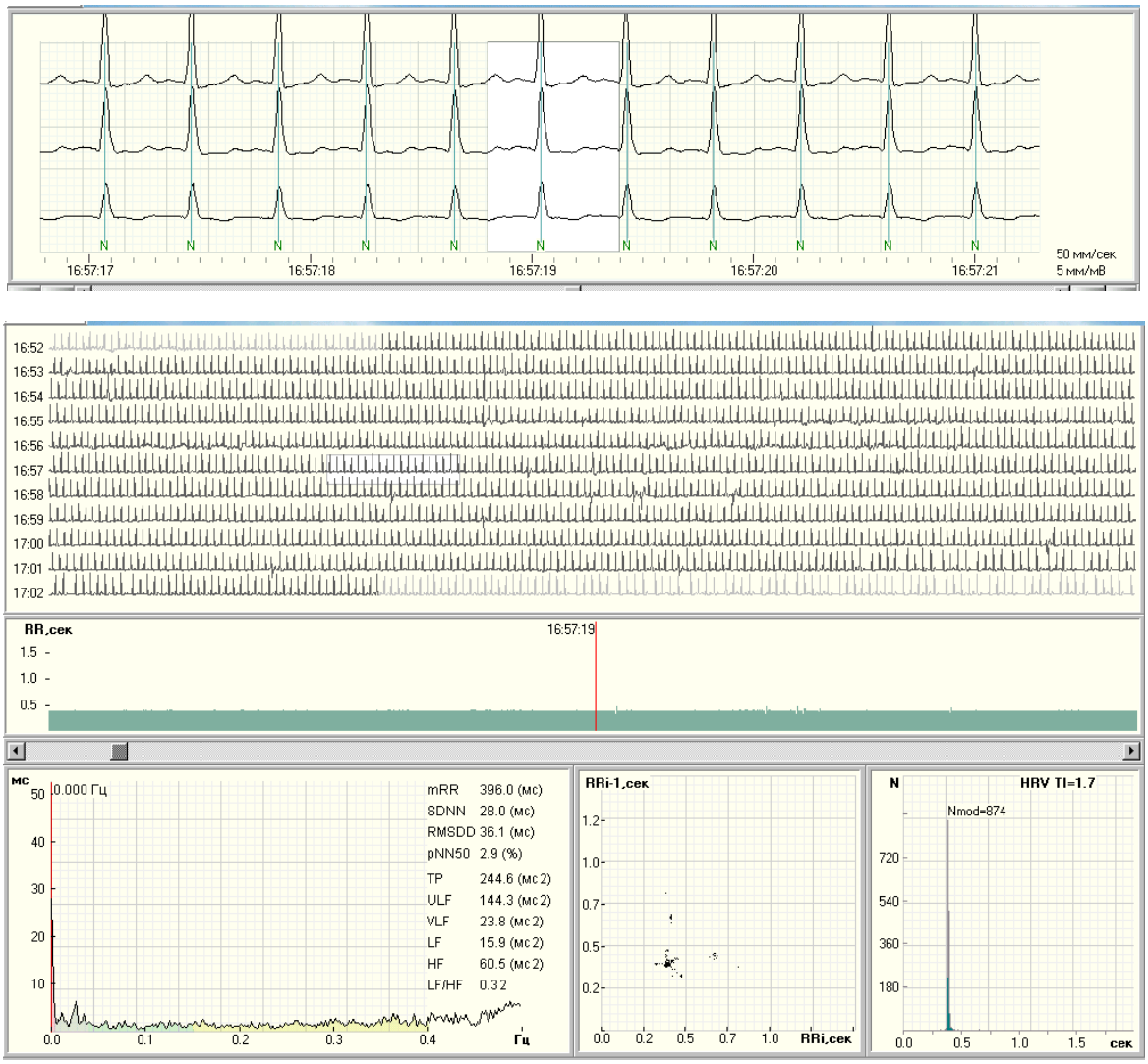
Temporal characteristics of heart rate variability.



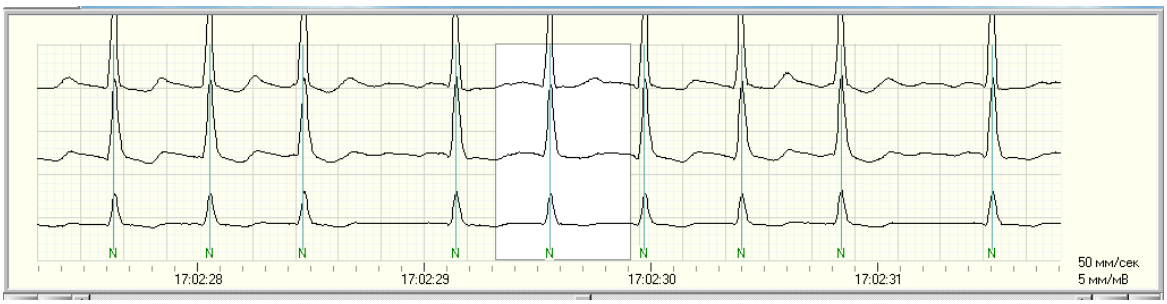
Fragments of maximum STj, ST1 and ST2 depression.

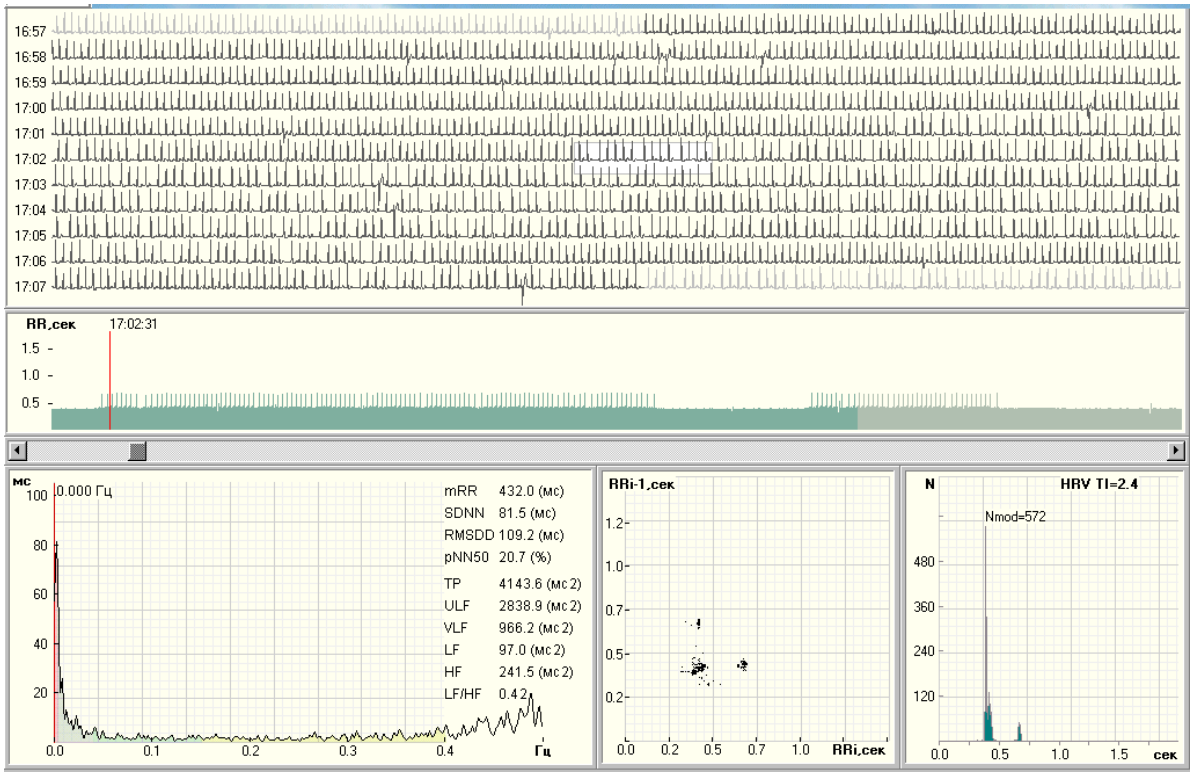


Fragment of ECG, rhythmogram and HRV indicators during sinus tachycardia.

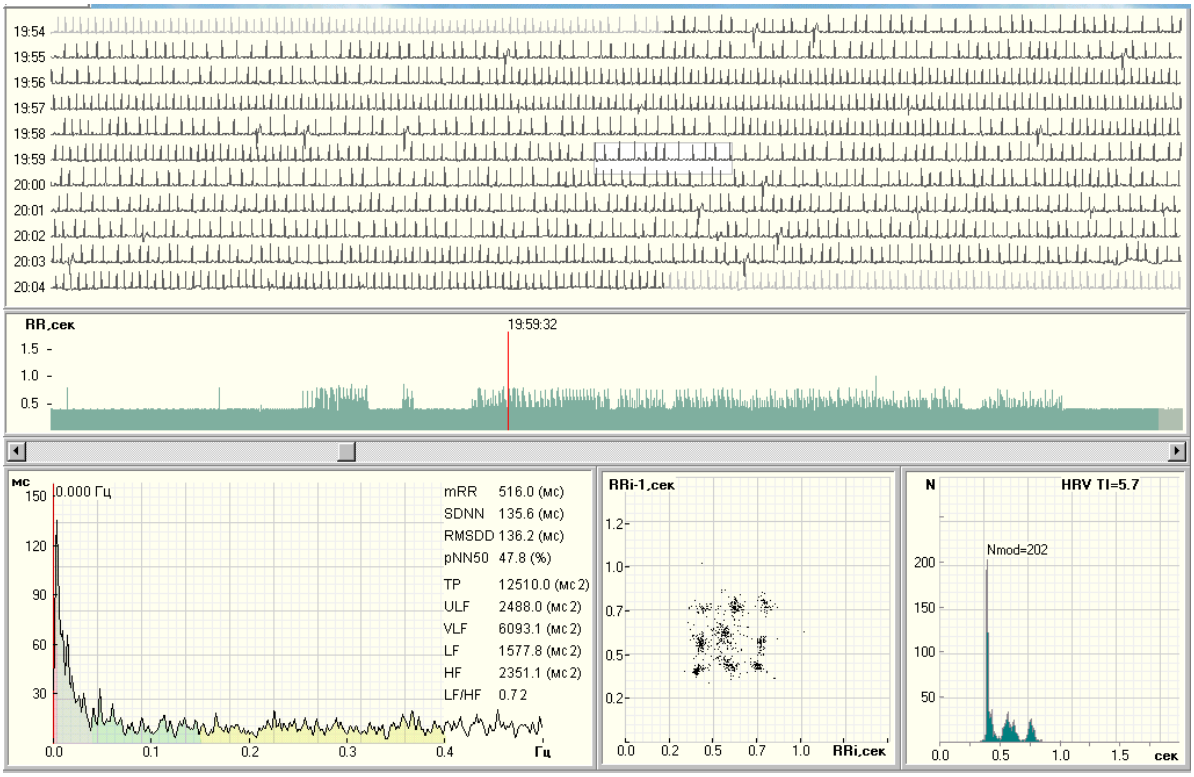


Fragment of ECG, rhythmogram and HRV indicators during atrial flutter with different AV conduction.

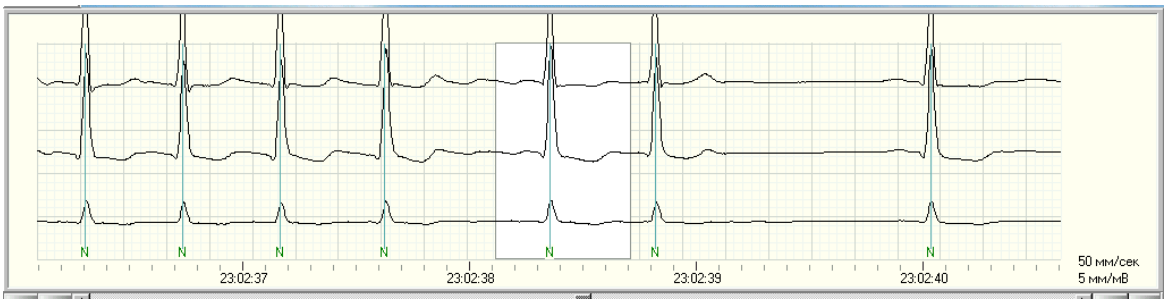




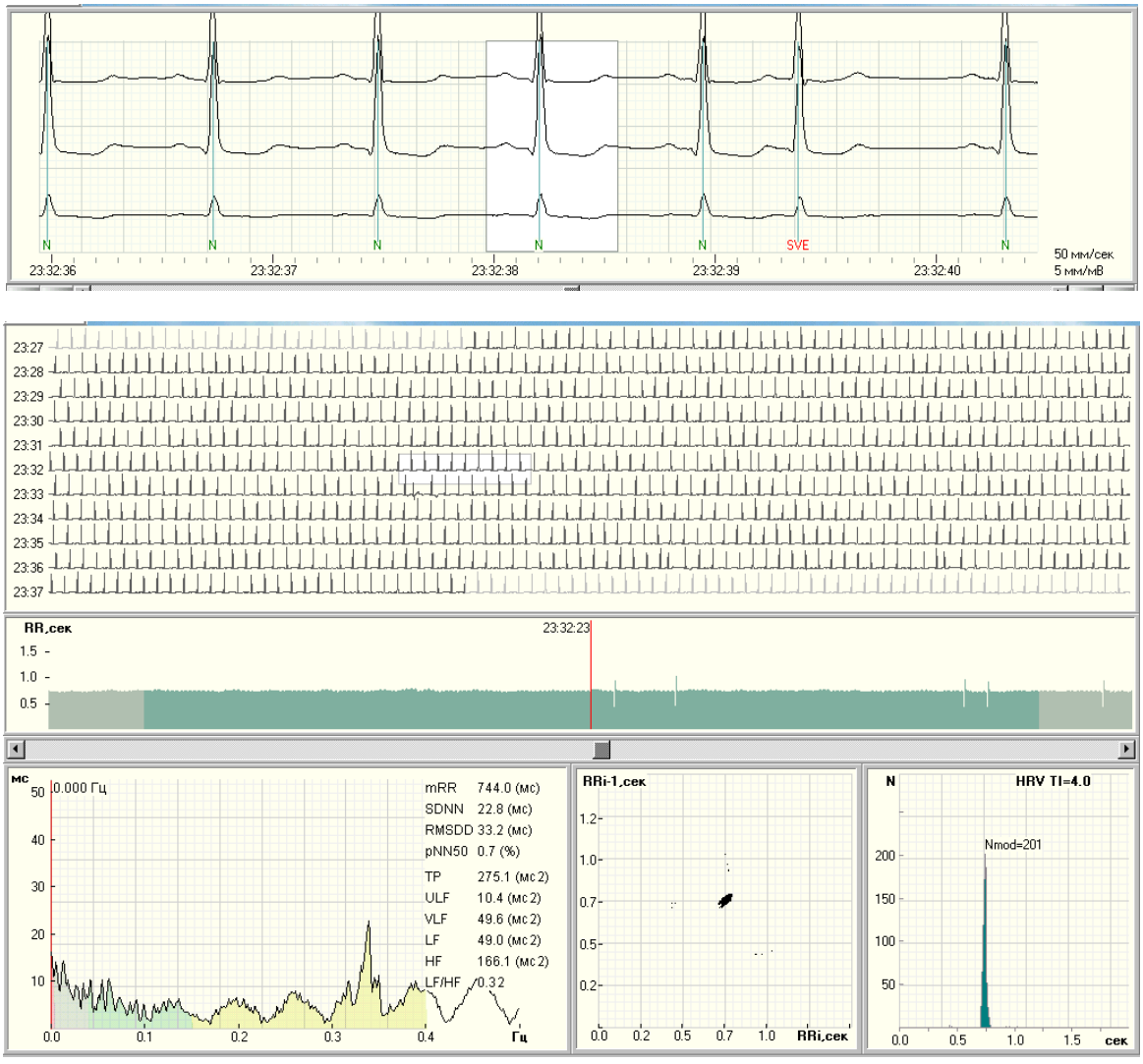
A fragment of the ECG, rhythmogram and HRV indicators during the transition of paroxysmal tachycardia to atrial fibrillation.



A fragment of the ECG during the restoration of sinus rhythm



Fragment of ECG, rhythmogram and HRV indicators during sinus rhythm with single supraventricular extrasystoles.



Conclusion

1. Heart rate

High average day (110 beats/min) and night (88 beats/min) HR with preserved circadian periodicity (CI - 1.25). At rest and during moderate exercise, 33 episodes of tachycardia with a total duration of 17854 seconds were registered. (The first 8 hours from the start of ECG registration). During the period of tachycardia, the average hourly HR is 118,153 beats/min. with a maximum HR of 160 bpm. All episodes of tachycardia occurred with minimal or moderate physical exertion. Periods of bradycardia and pauses were not detected.

2. Rhythm

A total of 521 ventricular extrasystoles with a pre-ectopic interval of 485-674 ms were registered. (623 ms on average). Before the restoration of sinus rhythm, extrasystoles of L2 class (from 58 to 155 per hour) and L1 class (from 2 to 22 per hour) were detected for 5 hours. After rhythm restoration, extrasystoles of L2 class (39 per hour), 7 hours of L1 class (from 1 to 23 per hour) and 4 hours of L0 class were registered.

In the period from 15:57 to 23:02, short-term paroxysms of atrial flutter and fibrillation were detected against the background of sinus tachycardia. At 23:02, sinus rhythm was restored with a HR of 80 beats/min with adequate physical exertion, HR up to 117 beats/min. diary).

3. HRV

ECG sections with sinus rhythm are characterized by low total spectrum power (TP) and LF/HF ratio (275.1 ms 2 and 0.32, respectively), which indicates severe disturbances in the regulatory system. Short ECG periods with atrial flutter and fibrillation had HRV indicators characteristic of these arrhythmias, characterized by an increase in TR during flutter by an order of magnitude (4143.6 ms2) and fibrillation - by two orders of magnitude (12510 ms2) higher than sinus 1 (0.42 and 0.72 respectively).

4. S-T segment

No significant episodes of myocardial ischemia were found.

Conclusions

Pathologically high average day and night heart rate with preserved circadian variability. Complex rhythm disturbance: frequent episodes of atrial flutter/fibrillation of the tachystolic form with a positive reaction to cordarone, rare single extrasystoles of low gradations according to Lown and Wolf. Tachycardiac rate-adaptive responses to minimal and moderate stress. Low power of the HRV spectrum with low LF/HF as a manifestation of parasympatheticotonia.

Example 3 Full name: N-va L., female, 60 years old.

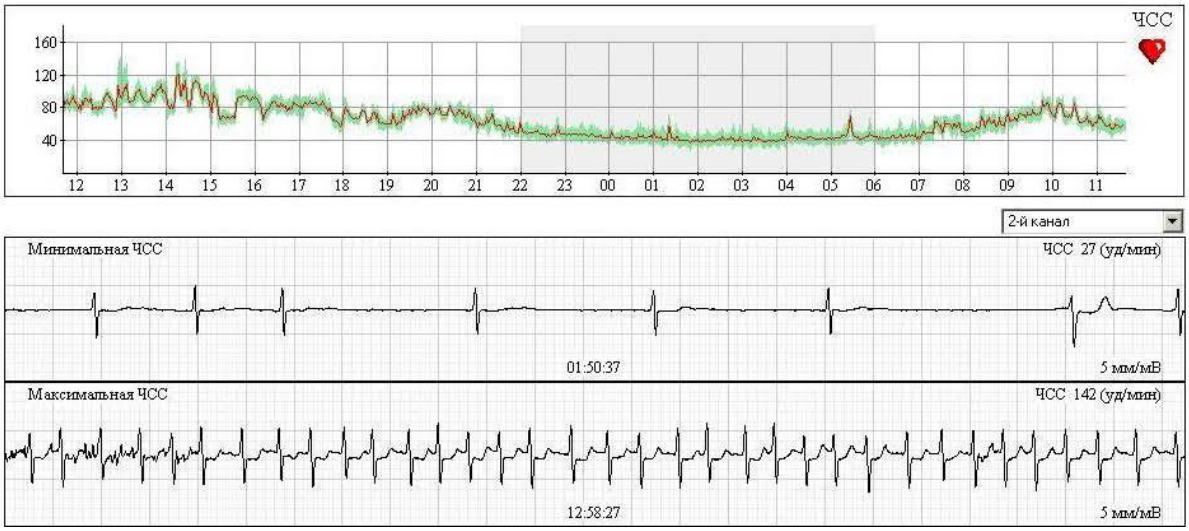
Diagnosis: Arterial hypertension II stage, II degree of severity. Permanent atrial fibrillation, tachysystolic form. CH II-A stage, III FC with systolic dysfunction of the left ventricle.

The purpose of the study: determination of the daily dynamics and frequency-adaptive response of the HR, the nature and clinical significance of HR disorders, the evaluation of the effectiveness of antiarrhythmic therapy. ECG recorder: "CardioSens" (Kharkov) Leads: CM-5, CC-1, CC-3

RESEARCH 1. Duration of observation: 24 hours.

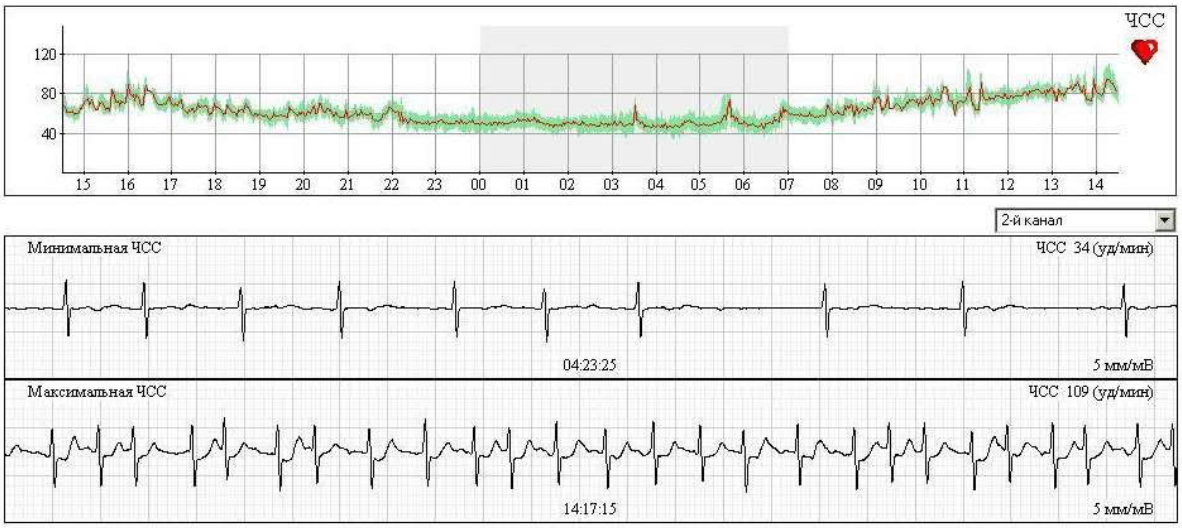
Total duration of artifacts: 3 hours 44 minutes. (14.3%).

Conclusions regarding the possibility of analyzing the AECG results: the total duration of the artifacts does not exceed the critical one, the analysis is possible.



RESEARCH 2.

Duration of observation: 24 hours Total duration of artifacts: 1 h 20 min (5%) Conclusions about the possibility of analyzing the AECG results: the total duration of artifacts is much shorter than the critical one, the analysis is possible The therapy carried out: locren 20 mg once a day, enap 2 / Daily, aspirin 325 mg per day.



Conclusion

1. Heart rate

At baseline, average daytime HR (69 beats/min) and low average nighttime (44 beats/min) HR with increased circadian periodicity (CI - 1.57). During the day, 6 episodes of tachycardia with a total duration of 250 seconds were registered. During the period of tachycardia, the average hourly HR is 86-95 beats/min. with a maximum heart rate of 142 bpm. All episodes of tachycardia occurred with minimal or moderate physical exertion. At night, 33 episodes of bradycardia (from 1 to 37 per hour) with a duration of 3288 seconds were registered. with an average hourly HR of 40-47 beats/min and 250 pauses (over 2000 seconds) from 1 to 58 per hour with a minimum heart rate of 27 beats/min.

In the course of therapy, with the preservation of a small number of tachycardias (5 episodes lasting 121 seconds), the average daytime HR moderately decreased (66 vs. 69 bpm), the average night heart rate increased (50 vs. 44 bpm), and the circadian frequency of the HR normalized - 1 .32 vs. 1.57), the maximum HR during submaximal exercise decreased to a normal value (109 vs. 142 bpm) and the minimum HR increased (34 vs. 27 bpm). With an increase in the total number of bradycardia (heart rate less than 56 bpm) at night (339 vs. 130), the number of pauses decreased almost 5 times (51 vs. 250).

2. Rhythm

Against the background of constant atrial fibrillation, a total of 1007 ventricular ECs with a pre-ectopic interval of 345-624 ms were registered. (523 ms on average). During 7 hours, ECs of L1 class (less than 30 per hour) were detected, during the remaining 14 hours – L2 (from 36 to 100 per hour) in combination with L3b (bigeminy), L3a (paired ECs) and L4b (more than three QRS complexes in a row) .

In the course of therapy, the number of ventricular ECs decreased by 90% (104 vs. 1007), episodes of bigeminy decreased by more than 95% (1 vs. 16), paired ventricular ECs and ventricular runs disappeared. 104 ventricular ECs were registered: during 4 hours – L0 class (EC absent), 1 hour – L3b class (1 bigeminy) and 19 hours – L1 class (from 1 to 16 ECs).

3. S-T segment

No significant episodes of ischemia were detected in both studies.

Conclusions :

At baseline, the average daytime and extremely low average nighttime HR with increased circadian variability. Complex rhythm disturbance: pathological ventricular ES of high gradations according to Lown and Wolf., pronounced bradyarrhythmia at night. Tachycardiac rate-adaptive responses of HR to minimal and moderate exercise. A positive reaction to locren therapy (20 mg per day). Normalization of average daily and average HR with restoration of circadian variability. Non-fatal rhythm disturbances: transition of ventricular ES of high gradations to low according to Lown and Wolf, preservation of bradyarrhythmia at night. Normalization of frequency-adaptive heart rate responses to moderate exercise.

Example 4.

Full name: Z-kyi V., male, 46 years old.

Diagnosis: CHD. Stable angina pectoris of tension I FC. Arterial hypertension II stage, I degree of severity. Persistent atrial fibrillation, tachysystolic form. HF first stage, FC II with preserved systolic function of the left ventricle.

The purpose of the study: determination of the daily dynamics and frequency-adaptive response of the heart rate, the nature and clinical significance of HR disorders, the evaluation of the effectiveness of antiarrhythmic therapy.

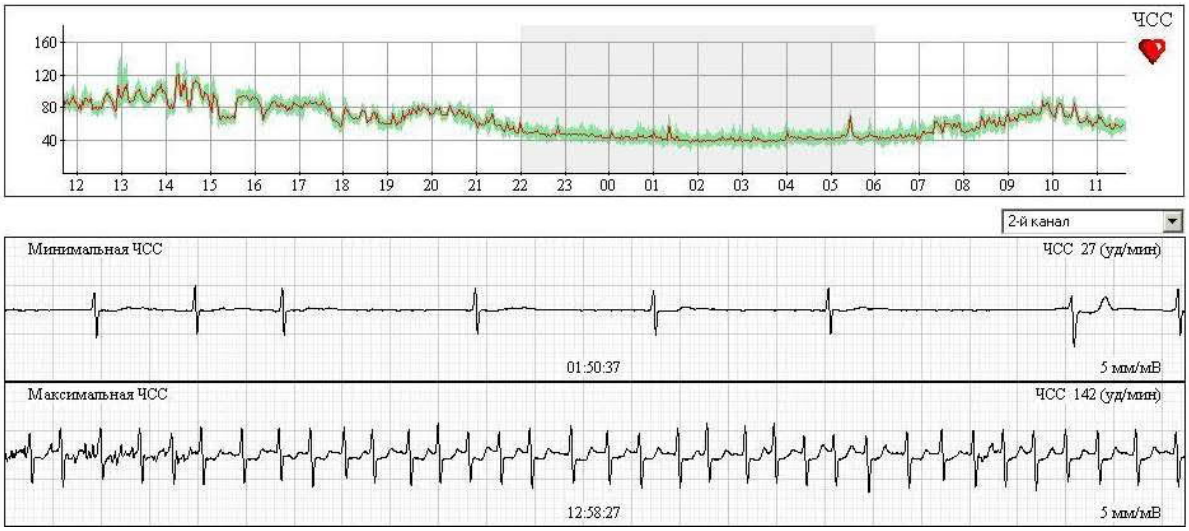
AECG registrar: "CardioSens" (Kharkov).

Leads: CM-5, CC-1, CC-3.

RESEARCH 1.

Duration of observation: 24 hours 17 minutes.

Total duration of artifacts: 0:00 00 min (0 %).



Conclusions regarding the possibility of analyzing the AECG results: there are no artifacts, the analysis is possible.

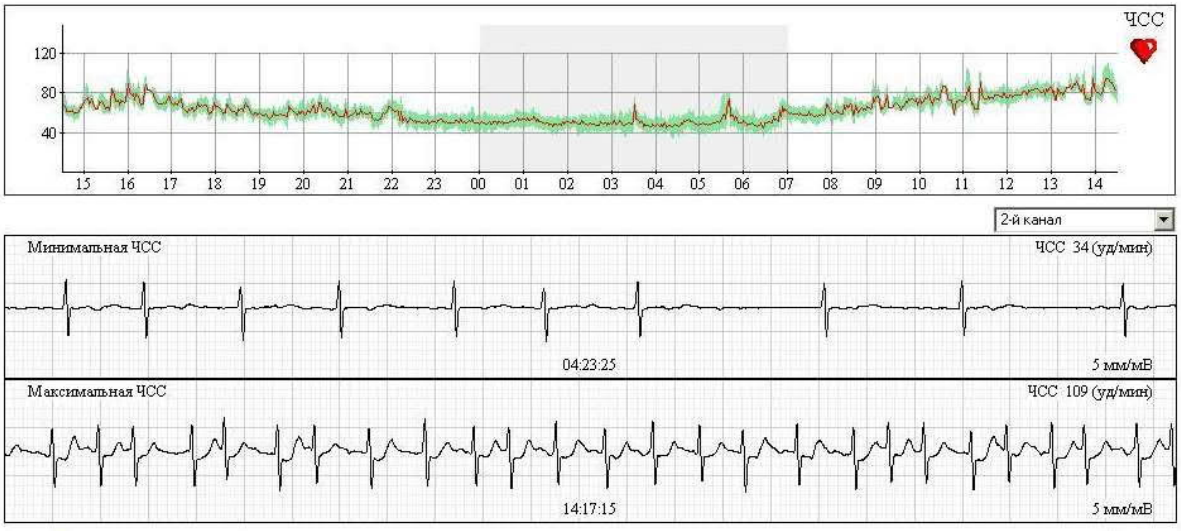
RESEARCH 2.

Duration of observation: 24 hours.

Total duration of artifacts: 1 hour 20 minutes (5%).

Conclusions about the possibility of analyzing the AECG results: the total duration of the artifacts is much shorter than the critical one, the analysis is possible.

Therapy carried out: cordarone 200 mg 3 times a day, enap 10 mg 2 times a day.



Conclusion

1. Heart rate

At baseline, average daytime HR (69 beats/min) and low average nighttime (44 beats/min) HR with increased circadian periodicity (CI - 1.57). During the day, 6 episodes of tachycardia with a total duration of 250 seconds were registered. During the period of tachycardia, the average hourly HR is 86-95 beats/min. with a maximum heart rate of 142 bpm. All episodes of tachycardia occurred with minimal or moderate physical exertion. At night, 33 episodes of bradycardia (from 1 to 37 per hour) with a duration of 3288 seconds were registered. with an average hourly HR of 40-47 beats/min and 250 pauses (over 2000 seconds) from 1 to 58 per hour with a minimum HR of 27 beats/min.

In the course of therapy, with the preservation of a small number of tachycardias (5 episodes lasting 121 seconds), the average daytime HR moderately decreased (66 vs. 69 bpm), the average night heart rate increased (50 vs. 44 bpm), and the circadian frequency of the HR normalized - 1 .32 vs. 1.57), the maximum HR during submaximal exercise decreased to a normal value (109 vs. 142 bpm) and the minimum heart rate increased (34 vs. 27 bpm). With an increase in the total number of bradycardia (heart rate less than 56 bpm) at night (339 vs. 130), the number of pauses decreased almost 5 times (51 vs. 250).

2. Rhythm

Initially, a total of 1007 ventricular ECs with a pre-ectopic interval of 345-624 ms were registered against the background of persistent atrial fibrillation. (523 ms on average). During 7 hours, ECs of L1 class (less than 30 per hour) were detected, during the remaining 14 hours – L2 (from 36 to 100 per hour) in combination with L3b (bigeminy), L3a (paired ECs) and L4b (more than three QRS complexes in a row) .

In the course of therapy, the number of ventricular ECs decreased by 90% (104 vs. 1007), episodes of bigeminy decreased by more than 95% (1 vs. 16), paired ventricular ECs and ventricular runs disappeared. 104 ventricular ECs were registered: during 4 hours – L0 class (EC absent), 1 hour – L3b class (1 bigeminy) and 19 hours – L1 class (from 1 to 16 ECs).

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Conclusions :

At baseline, the average daytime and extremely low average nighttime heart rate with increased circadian variability. Complex rhythm disturbance: pathological ventricular ES of high gradations according to Lown and Wolf., pronounced bradyarrhythmia at night. Tachycardiac rate-adaptive responses of HR to minimal and moderate exercise. Positive reaction to cordarone therapy (200 mg 3 times a day). Normalization of average daily and average HR with restoration of circadian variability. Non-fatal rhythm disturbances: transition of ventricular ES of high gradations to low according to Lown and Wolf, preservation of bradyarrhythmia at night. Normalization of frequency-adaptive heart rate responses to moderate exercise.

Main clinical syndromes and diseases

Great adaptation syndrome

The syndrome of great adaptation is identified with regulation, its quality. Balanced regulation is the key to health and successful recovery after illness. Violation of regulation - health weakens and diseases arise.

The regulation does not reflect the specificity of the disease, if it is not about the diseases of the regulation, but about its compliance or non-compliance with the disease, and therefore it is not worth looking for something specific for a particular disease in it. if only because there are many diseases, but there is only one big adaptation syndrome.

With AEKG, as in clinical practice in general, sufficient attention should be paid to the syndrome of great adaptation.

With AECG, the power of regulation is estimated through the total power of the HRV frequency spectrum.

If the total power of the HRV spectrum is within the limits of normal reactions to the disease, then the prerequisites of the disease are more favorable than in any other case. If the situation goes beyond the normal reaction to the disease, its course worsens.

If the total power of the HRV spectrum exceeds the norm, there is a high probability of vegetative cataclysms, which means a pathogenetic and not a sanogenetic component of the disease.

When the total power of the HRV spectrum decreases, control of the body's systems over the development of the disease decreases. The disease develops sluggishly, sanogenetic mechanisms are delayed.

Balance (of humoral and nervous autonomic links), or, from the point of view of spectral analysis of HRV, slow, medium and fast regulation is important. The balance is assessed by the power ratio of the corresponding ranges (domains) of the HRV frequency spectrum, regardless of the method of their assessment.

If the TP of the HRV frequency spectrum exceeds the response norm or even the norm, then the dominant slow regulation generates or exacerbates a long-term hyperreactive distress with persistent and even increasing pathogenetic reactions. If the average regulation prevails, the pathogenetic reactions are the same, but with a stronger "rocking". With the predominance of rapid regulation, there is a chance for stability, but only if it is not erroneous (with suffocation, rapid regulation begins to prevail due to irritation of the nuclei of the vagus nerves by the closely located respiratory nuclei of the brain stem).

In the reduction of the TP of the HRV frequency spectrum, the first violin belongs to the drop in the power of the fast (high-frequency) link. A decrease in the TP of the HRV spectrum with redistribution processes in favor of the slow (low-frequency) or medium (medium-frequency) link is the same as its deeper decrease or degeneration. An increase in the power of the fast component has a stabilizing effect, but as the TP of the HRV spectrum further decreases, the price of this effect becomes less and less.

An important characteristic is the physiological nature of regulation reactions and the degree of its violations. They are evaluated in functional and pharmacological tests by the degree of deviation of its indicators from the values characteristic of the initial quasi-stationary state, as well as by the characteristics of transient processes.

When working with a specific patient, it is necessary to select samples and their combinations that provide a more complete diagnosis of regulation, and the most reliable control method here is AECG.

Arrhythmias

AECG has expanded the understanding of the physiology and pathology of heart rhythm. We have shown above that with AECG in healthy people it is possible to find most of the arrhythmias that are found in patients. Despite the fact that arrhythmias are one of the most important indications for AECG, the most difficult and responsible part of it is their interpretation.

In connection with AECG, two types of tasks in the study of arrhythmias should be distinguished.

The first type of tasks arises when it was possible to register the clinical picture of arrhythmias and/or the arrhythmias themselves. Registered arrhythmias with the expected arrhythmic nature of the observed clinical signs confirm it. When there is an arrhythmia, but there are no clinical signs, it is necessary to determine its practical significance.

The second type of tasks arises in cases where the arrhythmia is not registered and its clinical signs are not diagnostically interesting. In other words, when the AECG should be performed again. Monitoring during the next week increases the possibility of detecting the connection of arrhythmias with clinical signs up to 70% and within a month - up to 90%. Here's the only question, should we strive to indefinitely increase the duration of AECG studies, or resort to other methods?

With the detection of arrhythmias, the task of assessing their nature and mechanisms arises.

Arrhythmias are many, different, such as, for example, functional neurogenic (hyper- and hypo-, adreno- and cholinergic, mixed), dyshormonal, dyselectrolyte, toxic, medicinal, and also associated with organic damage to the heart (organic).

Hyperadrenergic arrhythmias are due to the predominance of sympathetic influences against the background of suppression of the functional reserves of the main pacemaker with a tendency to higher values of the average HR during metronomization of the heart rhythm. Most often, they are associated with psycho-emotional stress, alcohol consumption, spicy food, smoking.

Hypoadrenergic arrhythmias may be associated with a deficiency of norepinephrine deposition in the endings of sympathetic nerves and SIF cells of the myocardium.

Hypercholinergic arrhythmias are due to the predominance of parasympathetic influences with a tendency to lower values of the average HR and a tendency to bradycardia at night with paroxysms of arrhythmia with increased sympathetic activity, which more often appear during sleep, wedge stasis, after eating. They are also observed in diaphragmatic hernias, esophageal diverticula, gastroesophageal reflux, biliary tract dyskinesia, flatulence.

There are also various options for combining disturbances in the ratio of sympathetic and parasympathetic influences.

Organic causes of arrhythmias are focal and diffuse dystrophic, inflammatory, including coronarogenic, diseases and syndromes in other, primarily systemic diseases, as well as their results in the form of local and widespread cardiosclerosis; cardiomyopathy, heart defects. It is more difficult to name a disease when arrhythmias never occur.

The classification of arrhythmias is always conditional by nature, and there is actually a superposition of functional and organic factors. This rule is unconditional for arrhythmias of organic origin, and therefore it is natural to talk about functional and mixed arrhythmias.

Idiopathic arrhythmias certainly have a cause. The reasons related to the problems of the central nervous system, vegetative regulation of the heart, extra- and intracardial sensory reception, especially regarding pathological reactions to stress (disruption of transient processes as the main manifestation of distress), today are solved by the methods of the quasi-transient and transient processes described by us.

However, it is not always possible to determine the exact electrophysiological mechanism of arrhythmias during AECG, and, apparently, it is not always necessary. This task requires special methods, for example, transesophageal electrophysiological examination of the heart, which, however, do not always need to be performed.

Assessment of arrhythmias is important and necessary, but correct. Gone are the days when they were intervented with at every opportunity.

Arrhythmias with problems of the great adaptation syndrome, rather than organic changes on the part of the heart, are conveniently classified as caused by blood circulation disorders that are exacerbated, do not significantly affect blood circulation and are aimed at compensating for its disorders.

First of all, compensatory rhythms should include regular and irregular tachycardic non-critical rhythms, regular and irregular normocarditic hemodynamically effective rhythms with ineffective atrial systole, late mono-(oligo-)topic extrasystoles (EC) with a compensatory pause. All of them either provide compensation for hemodynamic disturbances with low cardiac output, or prevent syncopal and severe conditions with a drop in heart rate.

A high degree of bradycardia and a low frequency of adaptation to physical activity are also not always signs of a "non-physiological" rhythm and may be compensatory. With asymmetric hypertrophy of the interventricular septum, the low-frequency rhythm registered in basal conditions often shows a small increase in heart rate in samples with physical exertion. The doctor is alarmed by this and stops the test. If a supraesophageal electrophysiological examination of the heart is performed with ultrasound control, it can be found that obstruction of the left ventricular outflow tract occurs at high stimulation frequencies. Nature, it turns out, acted wisely by imposing a ban on increasing the heart rate beyond the values when the development of obstruction is potentially possible.

Compensatory arrhythmias are short-term and long-term. The first occur for a short time in acute and in different periods of chronic diseases. They are compensatory for this period and lose these properties at other times. Others can exist for months and years, the entire life of the patient.

Most often, arrhythmias of short-term compensation are observed in severe forms of acute myocardial infarction, myocarditis, myocardial dystrophies, acute intoxications of various genesis, prolapse, etc.

Arrhythmias of long-term compensation occur in cardiosclerosis, valvular heart defects, cardiomyopathies, that is, organic changes in the myocardium.

Approaches to short-term and long-term compensation of arrhythmias are different. With short-term compensation, let's assume a greater deviation of the heart rate from the physiological norm. When restoring hemodynamics, it is necessary to correct short-term compensatory rhythms due to the loss of their role. If hemodynamically effective atrial fibrillation (AF) is a mechanism for local reduction of preload on the heart in acute widespread myocardial infarction, it loses this property in the subacute period.

An example of long-term compensation is the mentioned frequency-dependent obstruction of the outflow tract of the left ventricle with asymmetric hypertrophy of the interventricular septum.

Most often, long-term compensation arrhythmias develop during inflammatory and sclerotic processes in the heart. Even if changes in the conduction system are not taken into account, inflammation and cardiosclerosis require rhythm adaptation, the first sign of which is a change in basal heart rate.

Higher rates of basal heart rate in the elderly (within the normal range of fluctuations for a healthy person!) are a risk of critical paroxysmal conditions.

In chronic heart failure at the stages of subcompensation and decompensation, normosystolic AF, as in acute myocardial infarction (AMI), has a compensatory nature and is a local mechanism for reducing preload on the heart.

Often, sclerosis as a result of many chronic processes spreads to the conduction system, and then there are changes in the work of the heart that are interpreted as pathological. But they can also have a compensatory nature. Their elimination is possible only by prosthetics of the conducting system. An example of the consequences of such processes is the syndrome of weakness of the sinus-atrial node and the occurrence of replacing rhythms. The latter are compensatory. They cannot be suppressed, but must be corrected. Including the period before implantation of an artificial pacemaker.

Not everything is simple with arrhythmias and in traditional philosophy. So, no matter how thin the hypotheses about the local mechanisms of AF may be, they are an integral part of the general mechanisms. Thanks to AECG, for example, we know that AF paroxysm is triggered in some cases by sympathic tonic, in others by parasympathetic tonic, and in others by mixed between them and other types.

Let's take simple diagnostically monotopic, 100% from the same source, which are exactly the walls of the heart, extrasystoles, and, I would like to think, there are no problems with them. But something is completely wrong if, for example, they respond to extracardiac influences, such as psychotropic drugs. Why yes, the answer is simple - the heart cannot be "torn" out of a whole organism. Topical diagnosis, even in such a seemingly completely understandable case, is clearly lacking.

AECG leads to the origins of arrhythmias, their correct understanding. Correct understanding follows the same treatment.

A word about Heart Rate

Heart rate is extremely individual. With its norms of rest, reactions to stress, recovery after stress, and even greater values and changes in pathological conditions.

In healthy people, it is generally accepted that the lower and upper limits of resting HR are 50-100 beats per minute. It is smaller in hypersthenics, larger in asthenics. In a world where the exception to the rule is trivial, a lower resting heart rate is often the result of slower and a higher resting heart rate is the result of faster metabolic pathways.

The natural response of the HR to stress is an increase. The recovery period of HR after stress in healthy people is short. Even after high loads, it reaches the upper limits of the initial level in the first ten minutes. Slowing and increasing HR at rest, impaired reactions to stress and restorative changes are evidence of the existence of pathological conditions.

The changes found in HR at rest and under stress are not a problem of the HR, but a problem of the condition that led to them. These changes in HR may correspond to it, and then that is fine in any case, but may not correspond, which is an indication of more serious disorders.

Hyperthermic syndrome, hyperthyroidism should be accompanied by an increase in HR. But it can be not only adequate, but also insufficient and excessive. Heart rate in this guise is a criterion for the quality of regulation in these pathological conditions, how correctly and incorrectly their intimate mechanisms are organized.

Significant bradycardia with high blood pressure is a forced measure. It's a pressure problem, not HR. Atrial tachycardia with a wide post-infarction aneurysm is a mechanism for maintaining systemic blood circulation. It's an aneurysm problem, not a HR problem.

An extremely low HR is a danger of syncopal states, an extremely high HR is the development of acute left ventricular failure, and syncopal states as well.

We see high and low HR as circumstances of pathological conditions, we solve the problem of how adequate they are for these conditions and how safe they are for the patient's health as a whole.

It is also important that the HR is determined by the quality of life. It is less common in physically active and mentally strong people, with a calmer response to stress and faster recovery. Detrained, mentally weak people are characterized by large values and reactions of HR to stress with a longer recovery period. By the way, these are important factors that reflect the state of a person's biological rhythms, and with them the resources of his health and prospects for the quality of his future life.

Nodules

AECG is not a variant of ECG. Its background and capabilities are much broader and are not limited to electrocardiographic and related phenomena. AECG is a window into the regulatory systems and the mechanisms provided by them in general in health and disease, which are related to the functioning of the heart, and even more electrophysiological processes in it, are a

drop. It is such an AEKG that raises the diagnosis to the level with which medical care is directed to the patient, but not to his diseases. Therefore, we hope that precisely in these colors, and this is how we intended our book, you will catch our elevated view of AEKG.

Cheers to you in your processional activities, colleagues, and when using AECG, including.

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