

Basic criteria of finding the markers of hemodynamic self-regulation mechanism on ECG and Rheogram and analysis of compensation mechanism performance in maintaining hemodynamic parameters at their normal levels

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Aims

Basic criteria of finding the markers of the hemodynamic self-regulation mechanism on an ECG and a Rheogram are described herein. The basics of an analysis of the compensation mechanism responsible for the maintenance of the hemodynamic parameters within the norms are presented in this paper.

Materials and methods

A change in the form of an ECG phase segment reflects qualitatively the actual contraction performance of the respective cardiac muscle part involved. In case when the contraction of a cardiac muscle part is weak, an adjacent part undertakes the required compensation function in order to adjust the process. The causality analysis allows identifying the primary cause of a pathology which makes impossible to maintain the proper phase-related blood volumes within the norms.

Results

An evaluation of the hemodynamic self-regulation mechanism on the basis of the analysis of the compensation mechanism responsible for the maintenance of the hemodynamic parameters within the norms allows revealing the primary cause of the cardiovascular system pathology.

Conclusion

We offer an innovative methodology that is easy to use and very efficient, therefore, it can be widely applied in practice.

Keywords

Cardiometry • Cardiology • ECG • Rheogram • Hemodynamics self-regulation • Pathology of cardiovascular system

Imprint

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Introduction

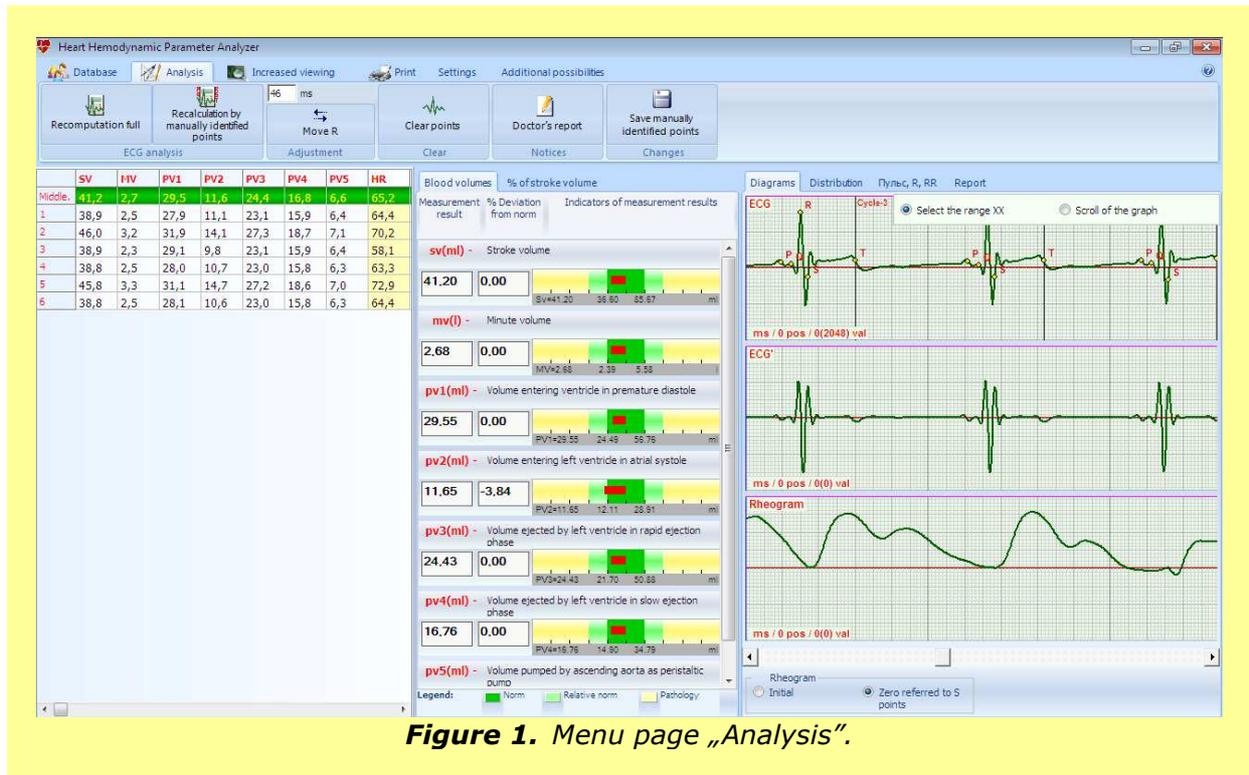
The discovery of the mechanism of the hemodynamic self-regulation was possible owing to the development of a new mathematical model of hemodynamics proposed by the authors hereof. This mechanism has been never described before. Closer examination of this mechanism established that the required hemodynamic parameters are maintained by a re-distribution of the contraction performance between the individual cardiac muscle parts. So, if the contraction amplitude of the septum decreases, that is reflected in the amplitude of one of the ECG waves, namely, the R wave, then it is compensated by an increase in the myocardium muscle contraction amplitude, that is represented by the S wave. It results in the normal value of the stroke volume SV. Therefore, any ECG curve change can show the "price to be paid" by our organism in order to maintain the hemodynamic parameters within their norms. The phase analysis of a Rheogram makes possible to trace pressure changes in the aorta. In general, all mentioned above allows us to identify the primary cause of a disease and monitor efficacy of treatment [1,2].

Materials and methods

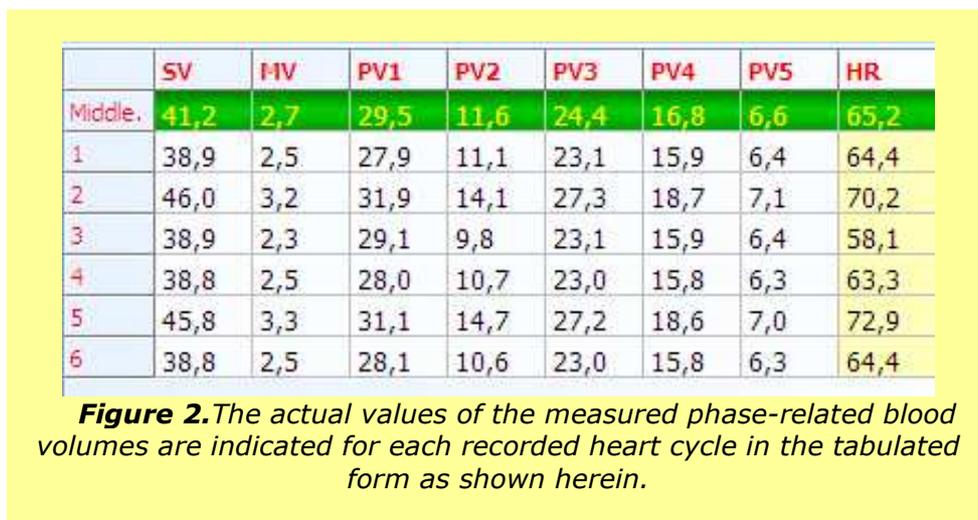
Principles of evaluation of the mechanism of the hemodynamic self-regulation

For this purpose, only the medical device Cardiocode can be used. The identifying and the tracing of the self-regulation mechanism in the cardiovascular system performance are beyond the capabilities of any existing medical equipment because it is only Cardiocode that can provide synchronously recording of an ECG and a Rheogram of the ascending aorta. This device is furnished with its own proprietary software.

Stage one of the evaluation procedure delivers results of the actual phase-related blood volumes measured with the above device. To obtain the measured value, used is the software menu page „Analysis" (s. Fig.1 below).



In the field on the left side of the screen we can find tabulated data on the actually measured stroke, minute and other phase-related blood volumes including heart rates. Such complete data sets are indicated for each heart cycle recorded (s. Fig.2 below). The top line in the table shows averaged values of each parameter, respectively.



In order to make an analysis quick and easy and reveal some “weak points” in hemodynamics, utilized are bar charts which represent the results of the actual measurements

(s. Fig.3). Each scaled red bar in each evaluation line reflects the actual value of the respective parameter. The green bar field indicates the respective hemodynamic norm. If the red bar is located within the green bar field, the hemodynamic value is O.K. The light-green bar field left or right to the green bar field exhibits a transitional status when the norm is found to be at its limit. If the red bar is located outside the light-green field, it shows a deviation from the norm, which exceeds 30% and which should be treated as pathology. Such a deviation is evidence that the compensation mechanism is no longer capable of maintaining the normal hemodynamics.

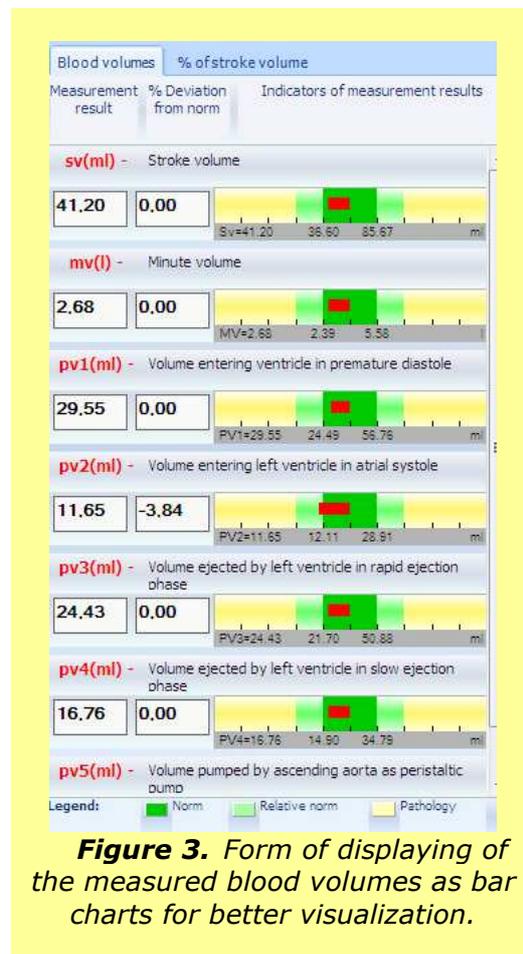
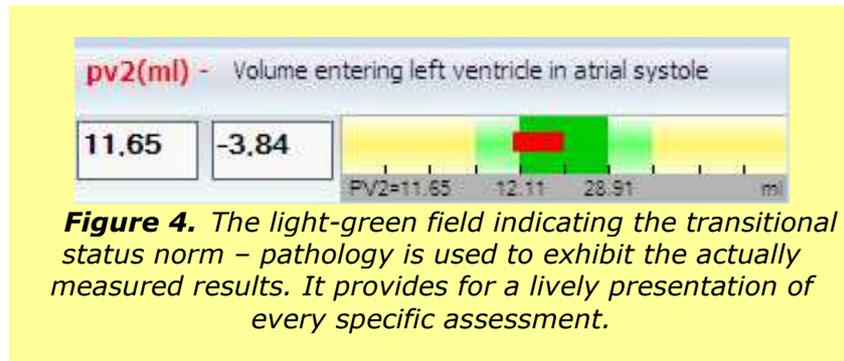


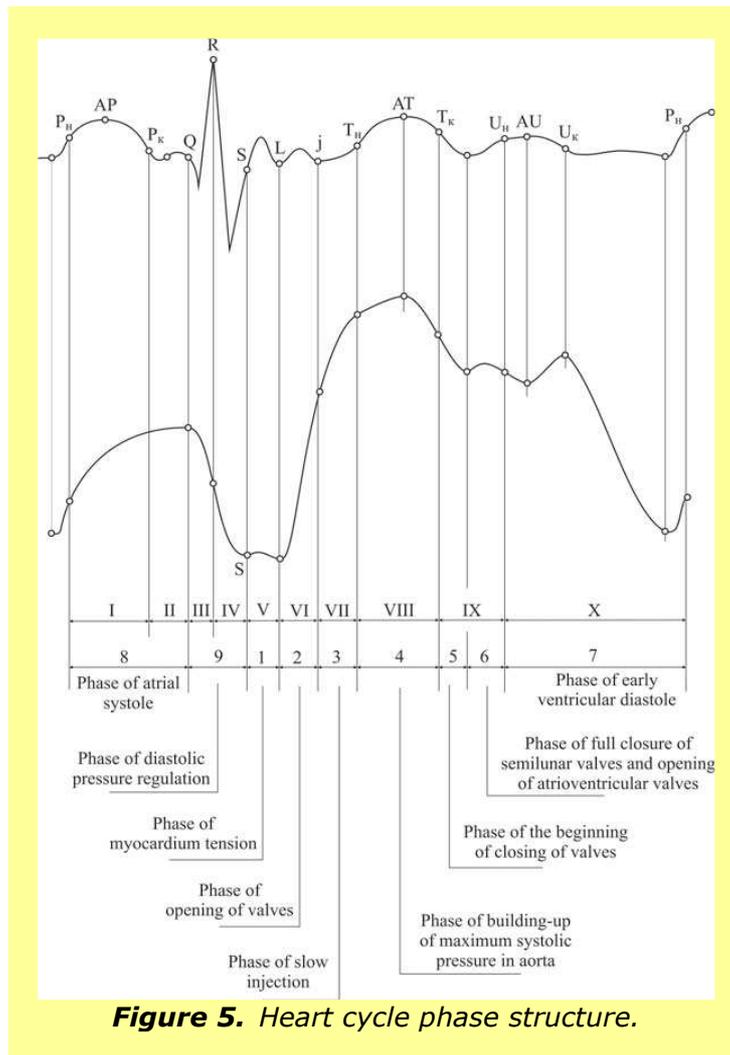
Fig.4 illustrates in more detailed way how the parameter PV2 from the above data set is interpreted. It is evident that the deficit of the volume of blood entering the ventricle in the atrial systole is -3,84%. The scaled red bar is located within the light-green bar field. It means that the next step should be an assessment of the compensation mechanism in those phases, which provide for the atrial systole, in order to identify the primary cause of an imbalance of the self-regulation mechanism in hemodynamics.



Criteria of finding the markers of the compensation mechanism responsible for maintaining hemodynamic parameters within the norm

The compensation mechanism is responsible for the maintenance of the hemodynamic parameters within their norms and operates as follows: the contraction function of the muscles of one segment of the cardiovascular system is increased, if and when the function of the other adjacent segment shows a decrease. It is reflected on ECG and RHEO in the phase structure of the heart cycle.

An illustration of the heart cycle phase structure is given in Fig. 5 below.



Algorithms of the analysis of the compensation mechanism may be different. We describe below some major algorithms utilized both for the identification of the primary cause of pathology and control of the therapy efficacy.

It should be noted that any amplitude change on an ECG reflecting the operation of the compensation mechanism can be graphically presented in the same way as it is the case with the measured parameters (Fig. 4). Fig.6 illustrates those informative zones of the ECG which are involved in the operation of the compensation mechanism for maintaining the normal hemodynamic parameters. This figure shows also ranges of amplitude changes in the said zones. The green field code is used in the same way as it is the case with indicators of actually measured blood volumes (s. Fig.4). The green-color coding is utilized to indicate the range of the norms. The light-green color coding shows a norm-pathology boundary.

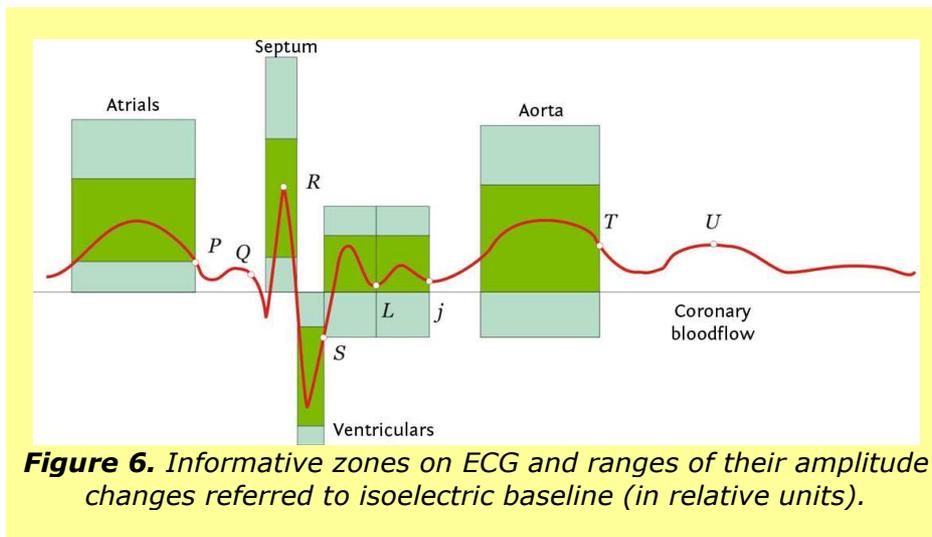


Table 1 illustrates the amplitude ranges of ECG phases for waves: P, R, S, L, T.

Table 1. Coefficients of increase in ECG waves amplitudes

Amplitude compensating the decreased contraction function of heart adjacent segment	+ 2,0	+2,0		+ 2,0	+2,0
Amplitude is within the norm	+ 1,5	+1,5	- 0,5	+1,5	+1,5
Norm	+ 1	+ 1	- 1	+ 1	+ 1
Amplitude is within the norm	+ 0,5	+ 0,5	- 1,5	- 0,5	- 0,5
Amplitude of muscle contraction below the norm (except S wave)	+ 0,25	- 1,5	- 2,0	- 1,5	- 1,0

Basic definitions of the operation of the compensation mechanism used in practice to identify the primary cause of pathology

1. Atria

1.1. Process biophysics

General

The atria provide for the mechanism of the closure of the atrioventricular valves. It is required for the further performance of the heart under the conditions of the diastolic pressure regulation. The atria are responsible for pressure balancing by applying the pressure to the internal space of the ventricles till the above valves are fully closed.

1.2. Functional purpose: closure of atrioventricular valves.

1.3. Atria characteristics:

a) Function of contraction of atrial muscles.

b) Phase volume PV_2 is a volume of blood entering the ventricle in the atrial systole that characterizes the contraction function of the atrium, ml.

1.4. Characteristics evaluation criteria:

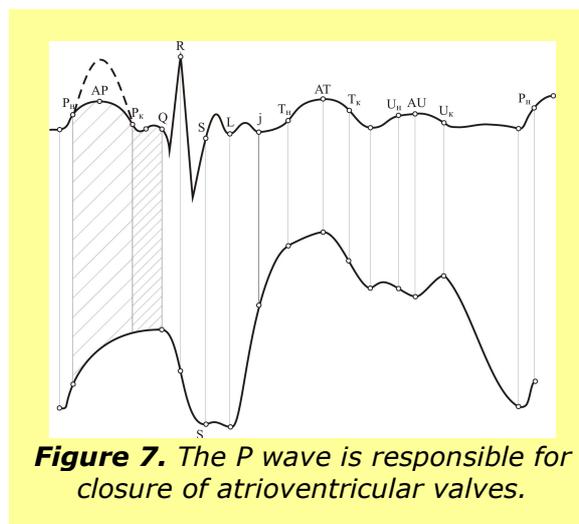
a) An amplitude of the P wave on the ECG represents the contraction function of the atrium.

b) PV_2 is a volume of blood entering the ventricle in the atrial systole phase, ml.

1.5. Markers of the compensation mechanism in the cardiovascular system performance found on ECG:

a) With residual volumes of blood in the ventricles, a no-P-wave effect can be detected. If it occurs periodically, it may be treated as a marker of the compensation mechanism;

b) Increased amplitude of the P wave in case of weak relaxation of the myocardium muscles.



2. Mechanism of diastolic pressure regulation

2.1. Process biophysics

General

The blood circulation must not be stopped even with the heart valves closed. The sequentially activated contraction of the septum and the myocardium and the original anatomic design of the valves arrangement provide for the directed blood flow in all chambers of the heart. It is a mandatory condition, because the available flow energy in case of a blood flow interruption would not be sufficient to re-start the blood circulation after the valve opening. Blood leakage through the closed valves begins only under the conditions, if an excessive volume of blood in the ventricles is available. This excess is displaced into the aorta to provide a condition for an increase in the static (diastolic) pressure required to overcome the increased resistance of the blood vessels which displaced this volume. Later on, the stroke volume is added thereto.

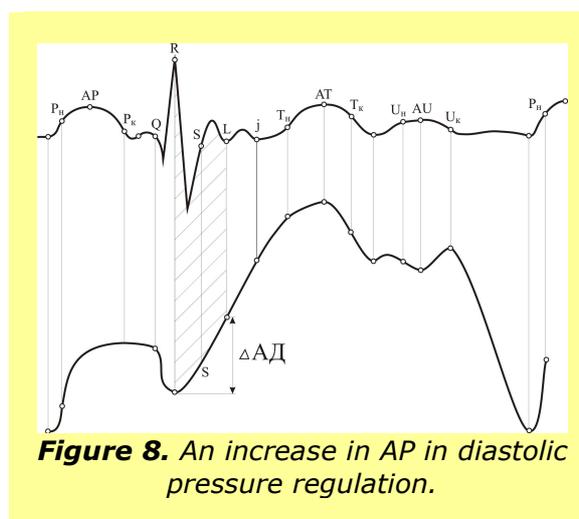
2.2. Functional purpose: to provide the required diastolic pressure level

2.3. Mechanism characteristics:

- a) Function of contraction of septum;
- b) Function of contraction of myocardium muscles.

2.4. Characteristics evaluation criteria:

- a) Amplitude of the R wave of an ECG represents amplitude of the contraction of the septum.
- b) Amplitude of the S wave on an ECG represents amplitude of the contraction of the myocardium.
- c) The elevation segment of the RHEO curve in phases R – S – L reflects an arterial pressure rise (AP increase) (s. Fig.8). It corresponds to the volume of blood displaced into the ventricles by the blood vessels due to the narrowing of the latter.



2.5. Markers of compensation mechanism in cardiovascular system performance on ECG
Under the diminished function of the myocardium contraction in phase R – S (S wave amplitude = 0), the compensation mechanism is marked as a bifurcation of the R wave. It enables the blood flow to maintain the flow velocity in the ventricles which is required for flow energy keeping.

3. Mechanism of systolic pressure regulation

3.1. Process biophysics

General

The mechanism of the regulation of the systolic pressure operates in combination with another mechanism which is responsible for the formation of the blood flow pattern required for the maintenance of the elevated fluidity mode. In order to provide the specific blood flow pattern, which represents alternating rings of blood plasma and corpuscles, used are the straight segment of the ascending aorta, the arch of the aorta supported by its elasticity (stretching ability).

3.2. Functional purpose: to provide the required systolic pressure level.

3.3. Mechanism characteristics:

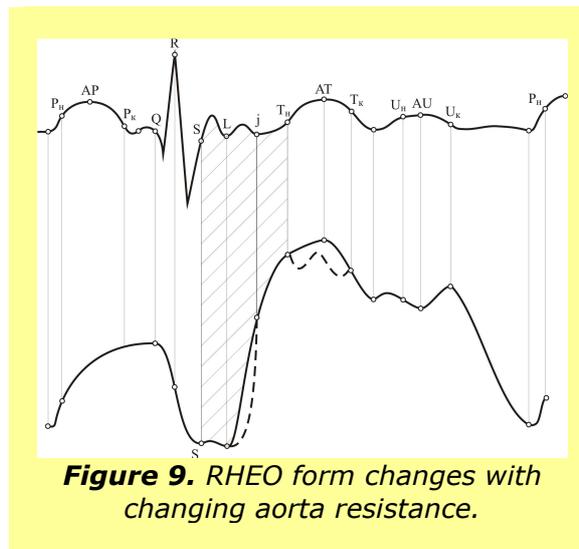
- a) Function of myocardial tension in phase S – L;
- b) Increase in AP in rapid ejection phase L – j;
- c) Increase in AP in slow ejection phase j – T_H and in a T_H-to-T-peak wave segment;
- d) Phase volume PV3 is a blood volume ejected by the ventricle in the rapid ejection phase, ml;
- e) Phase volume PV4 is a blood volume ejected by the ventricle in the slow ejection phase, ml;
- f) Stroke volume – SV, ml.

3.4. Characteristics evaluation criteria:

- a) Amplitude of the L wave on ECG is an indirect indicator of the strength of the myocardial tension;
- b) The slope ratio of the RHEO segment in phase j – T_H is an indication of the ejection velocity;
- c) PV3 is a volume of blood ejected by the ventricle in the rapid ejection phase, ml;
- d) PV4 is a volume of blood ejected by the ventricle in the slow ejection phase, ml;
- e) SV – stroke volume, ml.

3.5. Markers of compensation mechanism in cardiovascular system performance on ECG

- a) An increase in the T-wave amplitude is an indication of produced reduction in the resistance to the blood flow under weak heart;
- b) A delayed opening of the aortic valve prevents the blood flow from entering the aorta in full, reducing in such a way the systolic pressure.



4. Coronary artery filling

4.1. Process biophysics

General

The mechanism of blood filling of the coronary arteries operates in combination with another mechanism which is responsible for the filling of the ventricles with blood. The procedure of filling of the coronary arteries and that of the filling the ventricles start simultaneously. The difference is that the filling of the coronary arteries is completed within a shorter time, since their volume of blood constitutes about 5% of the total volume of blood within the circulation system. The filling starts with the beginning of the valve closure procedure. The aortic valves being located close to the mouth of the aorta are responsible for opening of the coronary artery to provide the start of blood supply to the coronary arteries.

4.2. Functional purpose: to supply the coronary arteries with the required blood volumes.

4.3. Filling characteristics:

a) In interval T - U_H, the sloping of this RHEO segment depends on the actual resistance of the coronary arteries. Under greater resistance, that may occur in case of atherosclerosis, the blood flow rate is less.

4.4. Characteristics evaluation criteria:

a) Sloping of the RHEO segment in interval T - U_H (s. Fig.10).

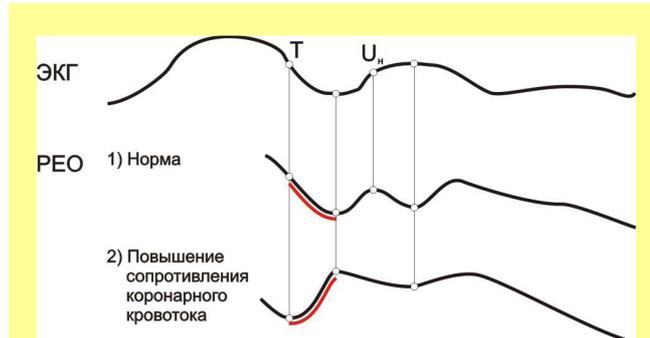


Figure 10. Changes in the RHEO segment configuration during the filling of the coronary arteries in interval T – U_n. The sloping of this RHEO segment depends on the actual resistance of the coronary arteries.

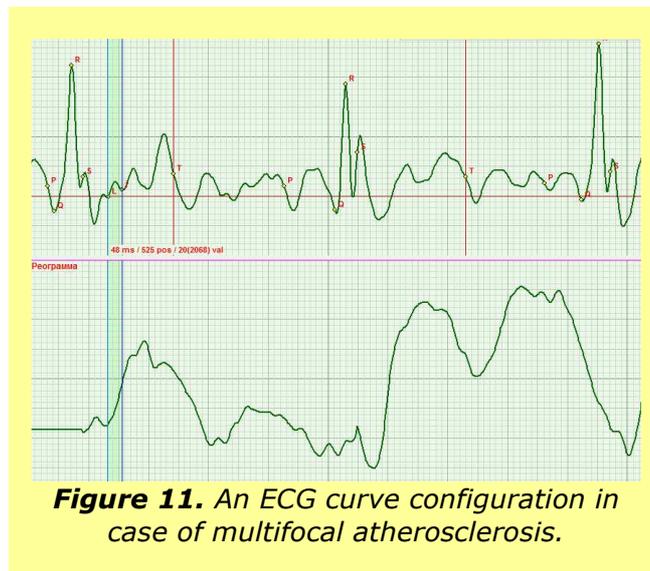
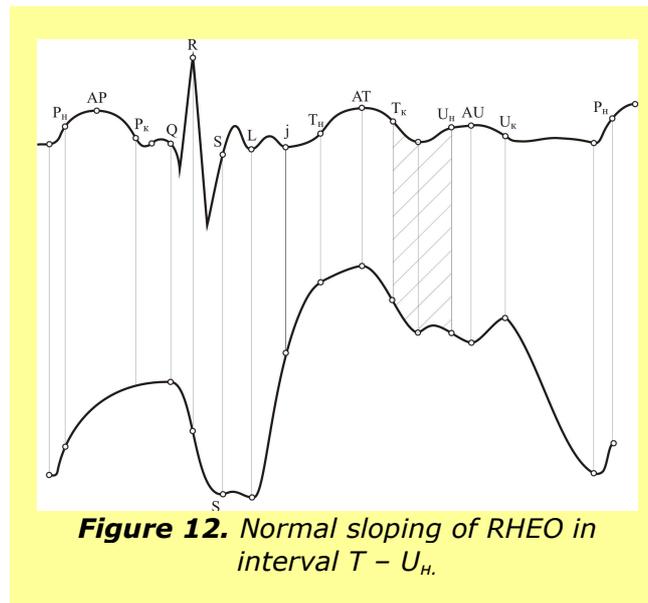


Figure 11. An ECG curve configuration in case of multifocal atherosclerosis.

4.5. Markers of compensation mechanism in cardiovascular system performance on ECG
The compensation mechanism cannot be traced in this interval. The only hindrance during the filling of the coronary arteries might be an arterial occlusion due to atherosclerosis.



5. Filling of ventricles

5.1. Process Biophysics

General

The ventricles are filled with blood owing to their pumping function. During the relaxation of the muscles, the space of the ventricles is increased, and blood enters them through the atria. The aortic valve is closed at this time. The ventricles are anatomically designed in such a way that the chordae tendineae, connecting the ventricle walls and the atrioventricular valves, open the valves with the ventricle expansion, releasing the delivery of blood to the ventricles. The atrial systole is required for closing the atrioventricular valves.

5.2. Functional purpose: filling the ventricles with blood in early diastole and atrial systole

5.3. Filling characteristics

- a) Function of relaxation of myocardium;
- b) Phase volume PV1 is a volume of blood entering the ventricle in the early diastole phase, which is the characteristic property of the suction function of the ventricle, ml;
- c) Phase volume PV2 is a volume of blood entering the ventricle in the atrial systole, which is the characteristic property of the contraction function of the atrium, ml.

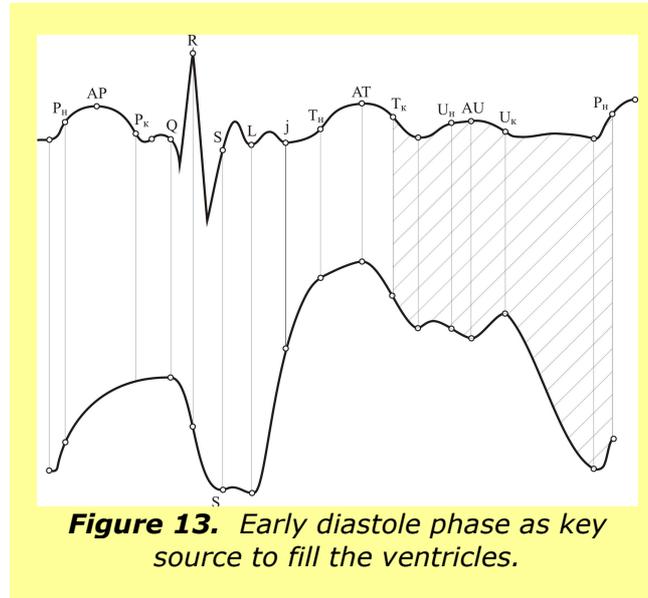
5.4. Characteristics evaluation criteria

- a) PV1 is a volume of blood entering the ventricle in the early diastole, ml;
- b) PV2 is a volume of blood entering the ventricle in the atrial systole, ml;
- c) The duration of phase $P - Q$ depends on elasticity of the myocardial muscles and the septum.

With reduced elasticity, it takes more time to close the atrioventricular valves.

5.5. Markers of compensation mechanism in cardiovascular system performance on ECG.

An increase in amplitude of the P wave that is required to increase the force of pumping blood into the ventricles.



6. General energetics in the heart performance to maintain the proper hemodynamics

6.1. Process Biophysics

General

The processes of general energetics of the heart may be traced when the energy reaches its lower levels. So, the QRS amplitude is decreased due to abnormal performance of the muscle cells, as a rule, caused by mitochondrial cardiomyopathy that represents a reduction in the myocardial contraction range. In this case, cardiac insufficiency occurs that makes impossible to transfer adequate energy to the blood flow to overcome the resistance of the aorta.

6.2. Functional purpose: the total energy to provide contraction of myocardium and septum

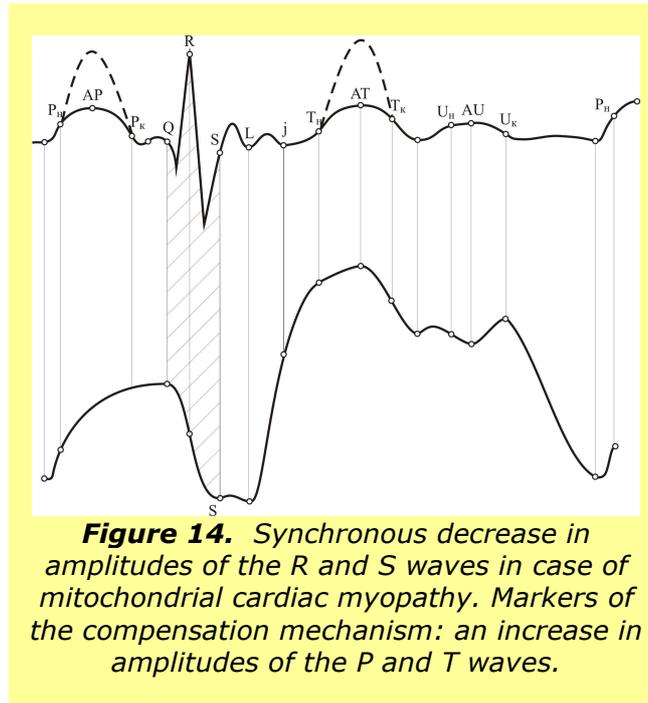
6.3. Characteristics: synchronous decrease of energy of contraction of myocardium and septum

6.4. Characteristics evaluation criteria:

- a) Synchronous decrease in amplitude of the R and S waves;
- b) MV – minute volume, l/min;
- c) PV5 is a volume of blood (an SV fraction) which is pumped by the ascending aorta as peristaltic pump and which is the characteristic feature of the aorta tonus, ml.

6.5. Markers of compensation mechanism in cardiovascular system performance on ECG

- a) An increase in amplitude of the T-wave that leads to an expansion of the aorta and a reduction in the aorta resistance to the blood flow;
 - b) An increase in amplitude of the P wave that leads to an increase in blood volumes pumped into the ventricles because the heart is no more capable of delivering the required volumes;
 - c) An increase in parameter PV5 which is the characteristic feature of the aorta performance.
- This provides a means to discharge a weak heart.



Results

This study results in the development of a new method of evaluation of the self-regulation mechanism in hemodynamics on the basis of the analysis of the compensation mechanism responsible for the proper maintenance of the hemodynamic parameters at their normal level which allows identifying the primary cause of pathology of the cardiovascular system. The basic criteria of finding the markers of the hemodynamic self-regulation mechanism on ECG and Rheogram and the analysis of compensation mechanism performance involved in maintaining hemodynamic parameters within the norm provide a tool for an assessment of the actual functioning of each cardiovascular system segment.

Discussion and conclusions

The mechanism of the hemodynamic self-regulation has been never described before, its discovery became possible owing to the scientific research conducted by the authors. The discovery of the mechanism of blood flow in the vessels enabled measuring blood volumes in every phase of the cardiac cycle. The collected statistical data allowed to observe that phase blood volumes are maintained within the norm even in case of the existing pathologies, with significant ECG changes are observed therewith. The authors studied the biochemical reactions taking place in the cardiovascular system. Assuming the normal functioning of the cardiovascular system it is possible to obtain the ECG forms ranging from normal to extremely abnormal. The authors succeeded in discovering the hemodynamic self-regulation mechanism and the compensation mechanism responsible for maintaining the hemodynamic parameters within the norm. The described criteria of evaluation of the compensation mechanism contribute to significant improvement of the quality of diagnostics performed in functional diagnostics.

Statement on ethical issues

Research involving people and/or animals is in full compliance with current national and international ethical standards.

Conflict of interest

None declared.

Author contributions

All authors contributed extensively to the work presented in this paper.

M.Y.R. read and met the ICMJE criteria for authorship. All authors read and approved the final manuscript.

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