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Control of Cardiovascular System

Mikhail Rudenko, Olga Voronova, Vladimir Zernov,
Konstantin Mamberger, Dmitry Makedonsky,
Sergey Rudenko and Sergey Kolmakov
Russian New University,
Russia

1. Introduction

The main method of cognition of the performance of biological systems is their mathematical modeling. The essence of this method should reflect the principle of optimization in biology[9]. Any biosystem cannot function if its energy consumption is inadequately high.

The same is applicable to the blood circulatory system. Its main function is to transport blood throughout the body in order to maintain the proper gaseous exchange, deliver important substances to viscera and tissues in living body and remove decay products. It is impossible to study this function without due consideration of hemodynamic features. But how is the blood circulation provided? It is a question of principle, and so far no unambiguous answer has been given thereto.

The conventional interpretation of blood circulation is that blood flows through blood vessels under laminar flow conditions to which Poiseuille's law is applicable. But it is a matter of fact that this conventional interpretation concept is inadequate because it is not in compliance with the above principle of optimization in biology, according to which all processes in bio systems show their best performance, i.e., their highest efficiency. It is just the compliance with this principle that is the major criterion to be used for evaluation of adequacy of any theoretical models describing various systems in living body and their interactions both with each other and their external environment.

Significant progress in understanding of such phenomena is made after G. Poyedintsev and O.Voronova discovered the so called mode of elevated fluidity, i.e., the third flow conditions that show lesser losses of energy to overcome friction and that is noted for lesser friction losses and specific pattern of the flow[4].

It has been proved that the blood flow through the blood vessels is provided in “the third” flow mode that is the most efficient and therefore fully in compliance with the said principle of optimization.

The theory of the third mode is a foundation for the development of new mathematical models describing the performance of the blood circulation system. In addition, new methods of quantitative determination of a number of hemodynamic parameters and

qualitative evaluation of some processes occurring in the system have been elaborated. The application of these methods in practice allows filling a lot of gaps in theoretical cardiology and creates at the same time a system of analysis of the functions of the cardiovascular system taking into account the relevant cause-effect relationship.

The detailed description of this theory is given in our book "Theoretical Principles of Heart Cycle Phase Analysis"[3]. Our intention is to outline herein the general principles of the performance of the cardiovascular system only.

2. Biophysical processes of formation of hemodynamic mechanism

2.1 Special features of hemodynamics and its regulation. Hemodynamic volumetric parameters

There two types of liquid flow conditions described in the classical fluid mechanics: the first type is the laminar flow, and the second one is the turbulent flow mode. In the 80th last century, a new theory of a specific liquid flow mode was developed by G.M. Poyedinstev and O.K. Voronova that was defined by them as the "elevated fluidity mode"[4]. Another name "the third flow mode" was given by the above discoverers to differ it from the two other modes well-known before. Being experts in solving technical problems of fluid mechanics, the authors succeeded in modeling the above elevated fluidity mode in a rigid pipe. For this purpose, hydraulic pulsators of specific design were used. It was established that the energy used to transport liquid in the third flow mode is several times less than it is the case under the laminar flow conditions[3]. Moreover, an efficiency of this process could be considerably increased when liquid is pumped under certain conditions through an elastic piping. The subsequent researches demonstrated that the physical processes producing the elevated fluidity mode and those in the blood circulation are identical. The mathematical tools used to describe "the third" flow mode was applied to describe the hemodynamic processes.

It was established by the authors that there are processes which are always observed in a rigid pipe at the initiation of a liquid flow from a quiescent state, as mentioned below. Whilst particles of liquid are starting their moving in the rigid pipe due to a difference in the static pressure, there a set of concentric waves of friction in the boundary layer is generating, the front of propagation of which is directed towards the pipe axis[3] (Fig. 1). Amplitudes of these waves depend on the diameter of the pipe, acoustic velocity in liquid and an initial difference in pressures at the pipe ends. The length of these traveling waves during this complex process continuously increases. The waves travel towards the axis of the pipe and degenerate. Finally, there a single wave remains only close to the pipe wall, the profile of which becomes parabolic that is typical for the laminar flow (s. Fig. 2 herein).

It should be noted that it is just within this short period of time, i.e., starting from the moment of the motion initiation from a quiescent state till the moment of formation of the laminar flow (s. positions E and F in Fig. 2 herein), when liquid flows in its optimum mode of elevated fluidity, considering it from the point of view of energy consumption (s. positions A, B, C, D in Fig 2 herein). The energy consumption under the laminar flow conditions to transport liquid in the pipe is significantly higher due to increase in the flow resistance.

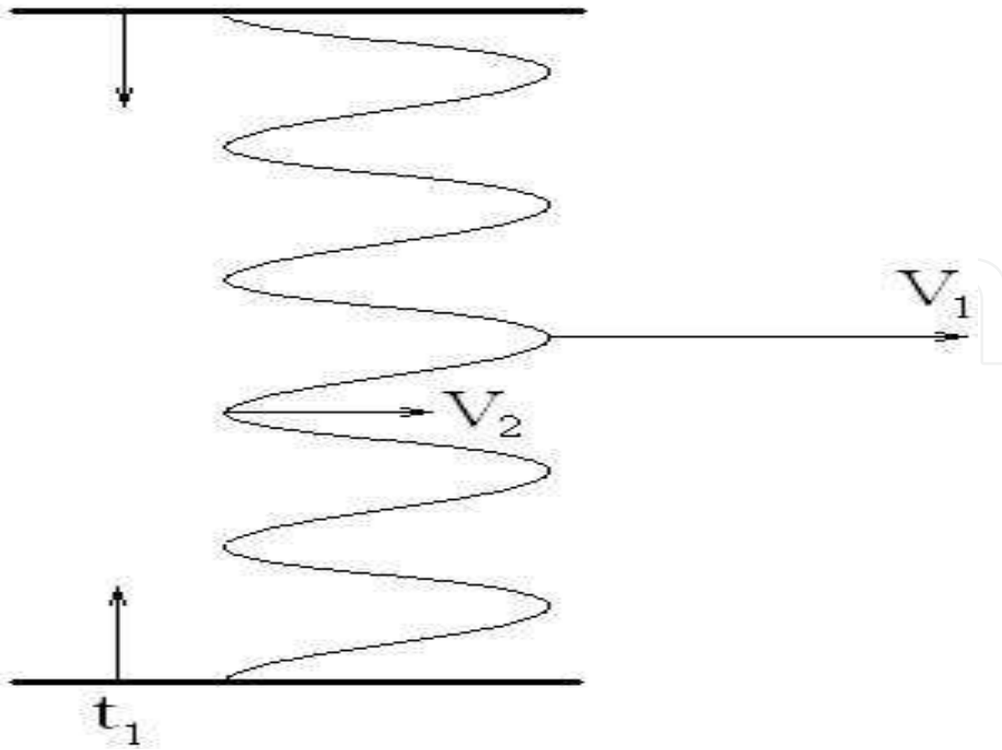


Fig. 1. Formation of concentric waves of friction at initiation of flow in a pipe (according to G.M. Poyedintsev and O.K.Voronova); t_1 - moment of pressure difference formation; V_1 - velocity of plasma in stagnated layers; V_2 – velocity of blood elements in accelerated layers

There is another phenomenon typical for the "third" flow mode. If liquid contains suspended particles similar to those in blood, during the development of the above mentioned wave process the particles are concentrated at the wave maxima, and the particle-free liquid is delivered to their minima, correspondingly[3]. When the liquid, patterned in such a way, flows along the pipe axis, the velocity of the concentric particle-loaded layers is twice what the liquid pattern-free layers reach. Vectors of velocity are parallel to the axis of the flow. And it is just a prerequisite to elevated fluidity of liquid with reduced friction between the liquid layers and the pipe wall. Figure 2 herein shows the locations of erythrocytes in the blood flow referring to each flow formation stage as mentioned above. At the beginning of the formation of the "third" flow mode, there ring-shaped alternating layers of the blood elements and plasma are available, while in the laminar mode all elements are accumulated in the center of the flow. In this case they are located very close to each other forming a thick mass. This process may result in an aggregation of erythrocytes and hemolysis. In order to avoid such pathological consequences, it is a must to manage the blood transportation in the "third" mode of flow, avoiding its transformation into a laminar one.

The theory gives a clue that it can be obtained when transporting liquid in a pulsating mode through an elastic pipe. According to this theory, the pipe clear width and the liquid flow velocity should be changed with every impulse under certain laws[3]. The laws of increasing in the pipe clear width and decreasing in the flow velocity with every impulse take the form as follows[4].

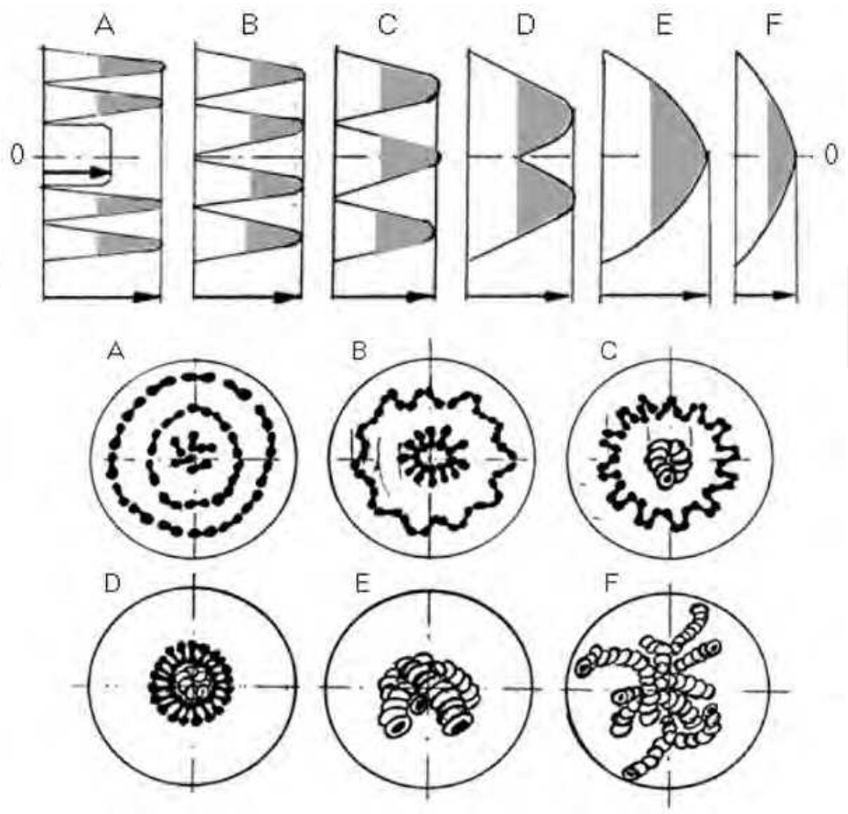


Fig. 2. Formation of two-phase pattern at the initiation of the flow from a quiescent state (according to G.M. Poyedintsev and O.K. Voronova), A-F – flow structure in corresponding sections

$$r_t = r_0 \left(\frac{t}{t_0} \right)^{1/5} \tag{1}$$

$$W_t = W_0 \left(\frac{t_0}{t} \right)^{2/5} \tag{2}$$

where r_t – current radius of the pipe increasing;
r_0 – initial radius (at $t = t_0$);
t - current time ($t \geq t_0$);
t_0 – time of acceleration of flow velocity up to maximum velocity in an impulse;
W_t – current value of liquid flow velocity;
W_0 – maximum value of velocity in an impulse (at $t = t_0$).

It is proved by the authors of this theory that the above conditions are met in the blood circulation system.

This is provided by changing in the clear width of blood vessels in every cardiac cycle and arterial pressure pulsating. The shape of the arterial pressure wave is given herein in Fig. 3 below.

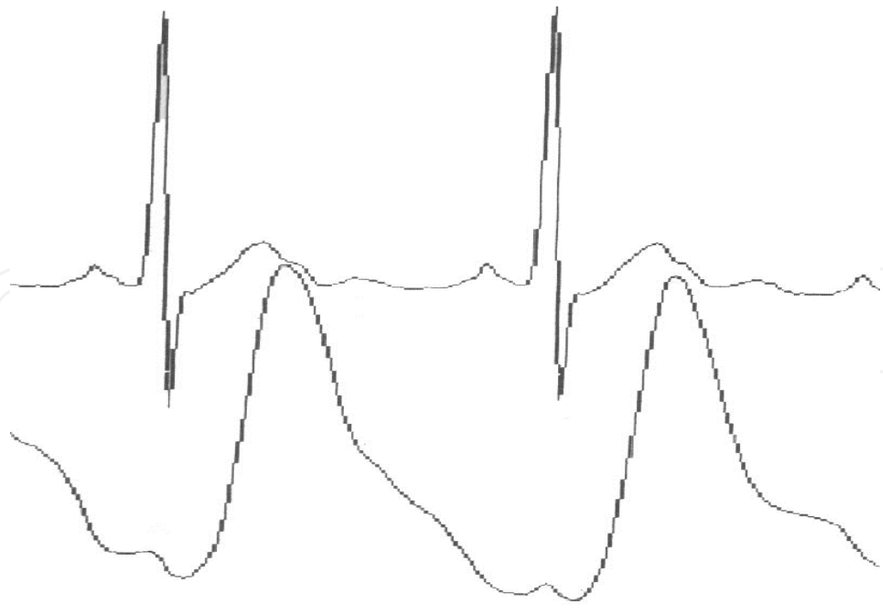


Fig. 3. Arterial pressure wave shape reography-recorded. ECG recorded simultaneously with Rheogram.

The foundation of hemodynamics is the phase mode of the heart performance. In one beat the heart changes its shape ten times that corresponds to the heart cycle phases[4].

The most efficient way is to evaluate the status of hemodynamics not only by values of integral parameters, i.e., stroke and minute volumes, but also phase-related volumes of blood entering or leaving the heart in the respective phase in a cardiac cycle.

So, the final formulae for calculation the volumes of blood in the phase of rapid and slow ejection, symbolized as PV3 and PV4, respectively, are as follows:

$$PV3=S\cdot(QR+RS)^2\cdot f_1(\alpha)\cdot[f_2(\alpha)+f_3(\alpha,\beta,\gamma,\delta)] \quad \text{(ml)}; \tag{3}$$

$$PV4=S\cdot(QR+RS)^2\cdot f_1(\alpha)\cdot f_4(\alpha,\beta,\gamma,\delta) \quad \text{(ml)}, \tag{4}$$

where S - cross-section of ascending aorta;
QR – phase duration according to ECG curve;
RS – phase duration according to ECG curve;

$$f_1(\alpha)=\frac{22072{,}5[(5\alpha-2)^3-27]}{(5\alpha-2)^5-243};$$

$$f_2(\alpha)=\frac{\alpha^5-1}{2};$$

$$f3(\alpha,\beta,\gamma,\delta)=\frac{1}{8}[\frac{10}{3}(4\alpha^2-\delta^2)(\beta^3-\alpha^3)+5\chi\delta(\beta^4-\alpha^4)-2\chi^2(\beta^5-\alpha^5)];$$

$$f_4(\alpha,\beta,\gamma,\delta)=\frac{1}{8}[5(\delta^2-\frac{8}{3}\alpha^2)(\beta^3-\alpha^3)+7{,}5\chi\delta(\beta^4-\alpha^4)+3\chi^2(\beta^5-\alpha^5)];$$

$$\alpha=(1+\frac{Em}{QR+RS})^{0,2};$$

$$\beta=(1+\frac{Em+Er}{QR+RS})^{0,2};$$

$$\chi=\frac{2(\alpha-1)}{\beta-\alpha};$$

$$\delta=\alpha(2+\chi).$$

Stroke volume SV is calculated by an equation as given below:

$$SV = PV3+ PV4=S\cdot(QR+RS)^2\cdot f_1(\alpha)\cdot[f_2(\alpha)+f_3(\alpha,\beta,\gamma,\delta)+f_4(\alpha,\beta,\gamma,\delta)] \quad (ml) \quad (5)$$

The minute stroke is computed as follows:

$$MV = SV\cdot HR \qquad (l/min) \tag{6}$$

In similar way calculated are other phase-related volumes of blood as listed below:

PV1 – volume of blood entering the ventricle in premature diastole;
PV2 – volume of blood entering the ventricle in atrial systole;
PV5 – volume of blood pumped by ascending aorta as peristaltic pump.

So, the main parameters in hemodynamics are 7 volumes of blood entering or leaving the heart in different heart cycle phases. They are as follows: stroke volume SV, minute volume MV, two diastolic phase-related volumes PV1 and PV2, two systolic phase-related volumes PV3 and PV4, and PV5 as volume of blood pumped by the aorta.

The authors of this theory in their researches utilized relative phase volumes denoted by RV. Each relative phase volume is that expressed as a percentage of stroke volume SV. These relative parameters demonstrate contributions of each phase process to the formation of the stroke volume in general.

The above hemodynamic parameters should be used mainly in order to evaluate eventual deviations from their normal values, if any. The limits of normal values of hemodynamic parameters are not conditional, and they have their respective calculated values.

With respect to the normal values (the required parameters) in hemodynamics, they have been taken on the basis of the known data on ECG waves, intervals and segments for adults from the literature sources as given below:

1. The upper and lower limit of the QRS complex values:

QRS_{max} = 0.1 s. ; QRS_{min} = 0.08 s.

2. The upper and lower limit of the RS complex values:
   RS_{max} = 0.05 s. ; RS_{min} = 0.035 s.
3. The normal value of interval QT in every specific cardiac cycle is determined from the Bazett formula as follows:

$$QT = 0.37\ RR^{0.5}, s \quad \text{(male)}; \tag{7}$$

$$QT = 0.4\ \ RR^{0.5}, s \quad \text{(female)}. \tag{8}$$

4. Normal value $PQ_{cer.}$ is calculated from a formula as indicated below:

$$PQ_{cer.} = 1 / (10^{-6}\ \ 638{,}44\ \ HR^2 + 9{,}0787)\ s \tag{9}$$

This equation has been produced according to the method of approximation of normal values $PQ_{cer.}$, as known from the sources, considering their dependence on heart rate (HR).

These values are used as initial values for calculations of an individual range of normal values of volumetric parameters in hemodynamics considering individual patient cases. In practice, for a better visualization of the data, it should be recommended to present them not only numerically but also graphically, as bar charts, as shown in Figure 4 herein. In the latter case, it is convenient to indicate the deviations from the normal value limits of the actually calculated values of hemodynamic parameters as percentage.

For example. On Figure 4 a), b), c) the result of hemodynamic parameters PV2 measuring - volume of blood entering the ventricle in atrial systole- is displayed as follows. Figure 4 a) in column "Blood volumes" shows the result of measuring 18,31 (ml). The second column "% of stroke volume" shows the deviation from the norm. It is 0% here. For quick associative perception of both these values and rapid highlighting of going beyond the bounds of norm parameter, there exists a dark green field with red light indicator to the right of this number in the column "indicators of measurement results". On the left and right sides of the dark green field we see the values of individual range of this hemodynamic parameter, calculated using equation 7, 8, 9. In this case, it is from 15.26 to 35,13 ml. Measured parameter of 18,31 ml is in the middle of the range, which corresponds to the 0% deviation from the norm. And the red light indicator that corresponds to this value is on the dark green background. Light green field - is a bound of "norm - pathology". Sides of this field correspond to excess or deficiency of more than 30% of norm. More than 30% excess requires special attention to the patient. As a rule, such patients needs hospital care. Figure 4 b) shows another patient's result, PV2 = 12,85 ml, and this result goes 15.84% beyond patient's individual norm 15,26 ... 35.13 ml. In this case red light indicates lack of blood volume, rather than redundancy. Lower (upper) than 30% value, but lower (upper) than normal value corridor, denotes further out-patient treatment for this patient. Fig. 4 c) shows a third patient with PV2 = 47,00 ml value, which goes 76.91% beyond his individual norm 10,72 ... 26.13 ml. Red light indicates the redundancy of blood volume. This patient should be examined by cardiac cycle phase analysis to identify the root causes of the disease. It's possible to identify these causes using ECG and RHEO for phase compensation mechanism of the cardio-vascular system determination.

a) b) c)

Fig. 4. Displayed measured phase-related values and their qualitative representation as bar charts, with reference to normal values. This figure gives three different measuring cases

The values of phase-related blood volumes are influenced by the mechanism of compensation existing in the cardiovascular system[6]. This mechanism is responsible for the maintenance of the hemodynamic parameters within their respective norms. If any parameter goes far beyond its norm, it means that it is an indication of physiological problems of the respective phase process. In this case, the function in the next phase compensates for the changes in the functioning of the problematic phase[6]. It is the just the case with sportsmen whose cardiovascular system shows the proper performance.

Physical exercise may cause a deficiency in diastolic volumes of blood by more than 500 %.[4] Under the circumstances, the systolic phases undertake to compensate for the above deficiency. For this purpose, the mechanisms may be involved, the manifestations of which cannot be found even in a pathology case. Upon stress relieving, 1 minute later, all phase-related volumes are normalized again. This kind of the performance of the cardiovascular system hinders an identification of the cause of pathology at early stages for those who are not professional athletes.

As a rule, deviations due to pathology exceed the norm by more than 30 %. Patients, who receive their treatment at cardiology hospital, show sometimes deviations of 50 % and over. The only way to find the primary cause of any pathology, based on the manifestation of the compensation mechanism, can be a thorough analysis of the actual cause-relationship in every individual case.

The phase-related volumetric parameters in hemodynamics are the most informative characteristics of the performance of the cardiovascular system since they are capable of

reflecting the coordinated operation of the heart and the associated blood vessels. Knowing their ratios and considering the actual anatomic and functional status of the heart and the blood vessels in every phase, we can produce very reliably a diagnosis of the actual status of the blood circulation system, reveal pathology and control the efficiency of therapy, if required.

The above mentioned evidence is really of fundamental importance. It should be taken into account when making diagnosis.

2.2 Mechanism of regulation of systolic pressure

The above mentioned main volumetric parameters should be complemented by another one: it is arterial pressure (AP). The cardiovascular system has its own mechanism to provide separate regulation of the systolic and diastolic pressures (AP)[8]. A narrowing in sectional areas of the blood vessels in total leads to a displacement of a certain volume of blood that is symbolized by ΔV. The displacement volume enters the ventricles in premature diastole phase T – P. During myocardium contraction phase R – S, the same volume is displaced via the closed aortic valve into the aorta. Actually, before the ejection of stroke volume SV into aorta, the total of displacement volume ΔV enters the aorta. Therefore, it is that the R – S phase, when ΔV can be ejected into the aorta, is preceded by that phase when the motion of the entire mass of blood is actuated, and this preceding phase is the Q – R interval, when the contraction of the septum occurs. It is just the phase when the blood flow becomes its directed vortex motion within the ventricle. Displacement volume ΔV contributes to moving against the total increased resistance of the blood vessels in the next phase which shows rapid blood ejection.

The blood circulation scheme is shown in Figure 5 herein. The anatomy of the heart is designed in such a way so that the displacement blood can penetrate without hindrance through the closed arteric valve into the aorta. It is determined not only by the configuration of the valves but also the mechanism of the contraction of the heart chambers that consists of three phases. Phase one among them is the contraction of the septum. Phase two provides for the contraction of the ventricle walls. Phase three is the phase of tension. The processes occurring therein are responsible for spinning the blood flows so that the penetration of the displacement blood through the closed valves into the aorta is assisted. Under normal conditions, when there is no displacement volume ΔV available, and, as a consequence, no penetration is required, upon completion of the phase of tension, stroke volume SV residing in the heart is supplied into the aorta. In this case, volume SV added to the volume of blood residing in the aorta creates the systolic pressure that produces a difference in pressures between the aorta and the periphery. Such mechanism required to overcome an increased blood flow resistance operates cyclically till the cause of blood vessel constriction disappears. The processes described above are typical for the mechanism of regulation of the diastolic arterial pressure. Various Rheogram curve shapes reflect this mechanism.

The anatomy design of the heart is determined by the phase mechanism of hemodynamics, i.e., the mechanism of the regulation of the diastolic pressure. This mechanism is responsible for elimination of general vasoconstriction difficulties in blood circulation. Causes of the said vasoconstriction cannot be diagnostically identified in this case.

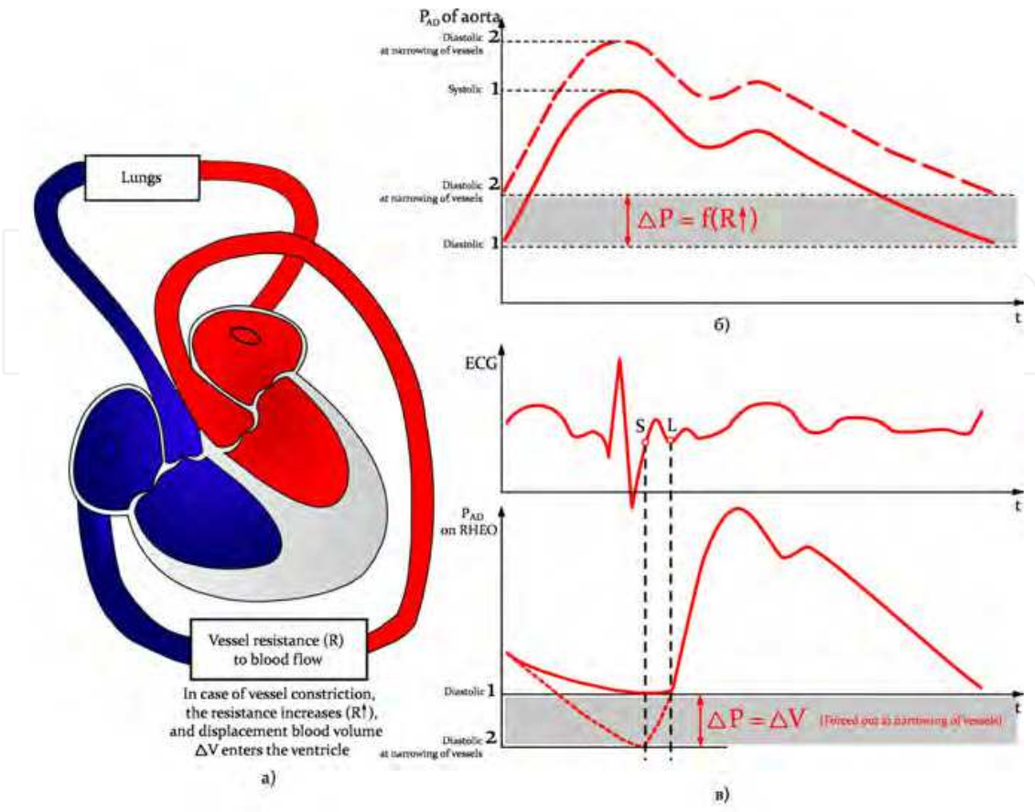


Fig. 5. a) Blood circulation scheme considering changes in blood vessel resistance. b) AP changes in aorta; c) Changes in AP identifiable on Rheo curve in phase of tension S-L, in proportion to displacement volume ΔV in blood vessel constriction

With synchronous recording of an ECG and a Rheo from the ascending aorta, provided that they are synchronized at wave point S on the ECG curve, the process of the regulation of diastolic pressure may manifest itself as an early AP rise on the respective Rheo curve in phases R – S and S – L.

2.3 Mechanism of regulation of systolic pressure

The mechanism of regulation of the systolic pressure differs significantly from that responsible for the regulation of the diastolic pressure. It has the function to provide a pre-requisite to the blood circulation in the blood vessels due to a difference in pressures between the aorta and veins and manage the transportation of an oxygen quantity as required by tissues and cells. For these purposes, several biophysical processes are engaged.

First and foremost, we should mention the process of myocardium contraction in tension phase S – L. The tension created in this phase presets the velocity of the blood flow during the blood ejection phase. Therefore, the initial velocity of the blood flow in the aorta depends on the degree of the myocardium tension.

The second important process is the phenomenon of an increase in the systolic pressure during the propagation of the AP wave throughout the arteries[1]. The systolic pressure in the aorta and that in the brachial artery may considerably differ from each other. On the

normal conditions, the pressure increase is provided by the pumping function of the blood vessels and their increasing resistance.

An additional point to emphasize is that there is another biophysical phenomenon connected with hemodynamics. It is cavitation in blood that promotes blood volume expansion[2]. It may spread over very quickly within one heart cycle and is capable of considerably expanding the blood volume.

The cause of the systolic pressure buildup is a reduction in blood supply of some viscera. The pressure buildup is aimed at elimination of hindrances in blood supply in order to maintain the proper blood circulation. The blood supply mechanism of some viscera provides for protection from arterial overpressures. In the first place, the protection of the cerebral blood supply system should be mentioned. The cerebral blood vessels are anatomically connected with veins. During an increase in AP, the venous drainage is hindered, affecting the blood vessel constriction and limiting in such a way an excessive AP increase.

If for some reason a viscus is not sufficiently supplied with blood, it leads to a systolic AP growth. The venous drainage will be hindered. The first symptoms of this problem could be edema of legs. To solve this problem, required should be elimination of the cause of the improper blood supply to the affected viscus that should decrease the AP and, subsequently, normalize the venous drainage.

3. Phase structure of heart cycle according to ECG curve

Every heart cycle consists of 10 phases. Each phase undertakes its own functions[7].

The complete phase structure of an ECG is shown in Figure 6 herein.

Phase of atrial systole $P_н$ – $P_к$;
Phase of closing of atrioventricular valve $P_к$ – Q;
Phase of contraction of septum Q – R;
Phase of contraction of ventricle walls R – S;
Phase of tension of myocardium S – L;
Phase of rapid ejection L – j;
Phase of slow ejection j - $T_н$;
Phase of buildup of maximum systolic pressure in aorta $T_н$ - $T_к$,;
Phase of closing of aortic valve $T_к$ - $U_н$;
Phase of premature diastole of ventricles $U_н$ - $P_н$.

Each phase serves its purpose. But the phases may be grouped in a manner as follows:

Group of diastol4ic phases which are responsible for blood supply to the ventricles:

Phase of premature diastole of ventricles $U_н$ - $P_н$;
Phase of atrial systole $P_н$ – $P_к$;
Phase of closing of atrioventricular valve $P_к$ – Q.

The phase of premature diastole contains a period of time equal to the duration of wave U which reflects an intensive filling of the coronary vessels with blood. It occurs in synchronism with filling of the ventricles.

The diastolic phases are described as hemodynamic values PV1 and PV2.

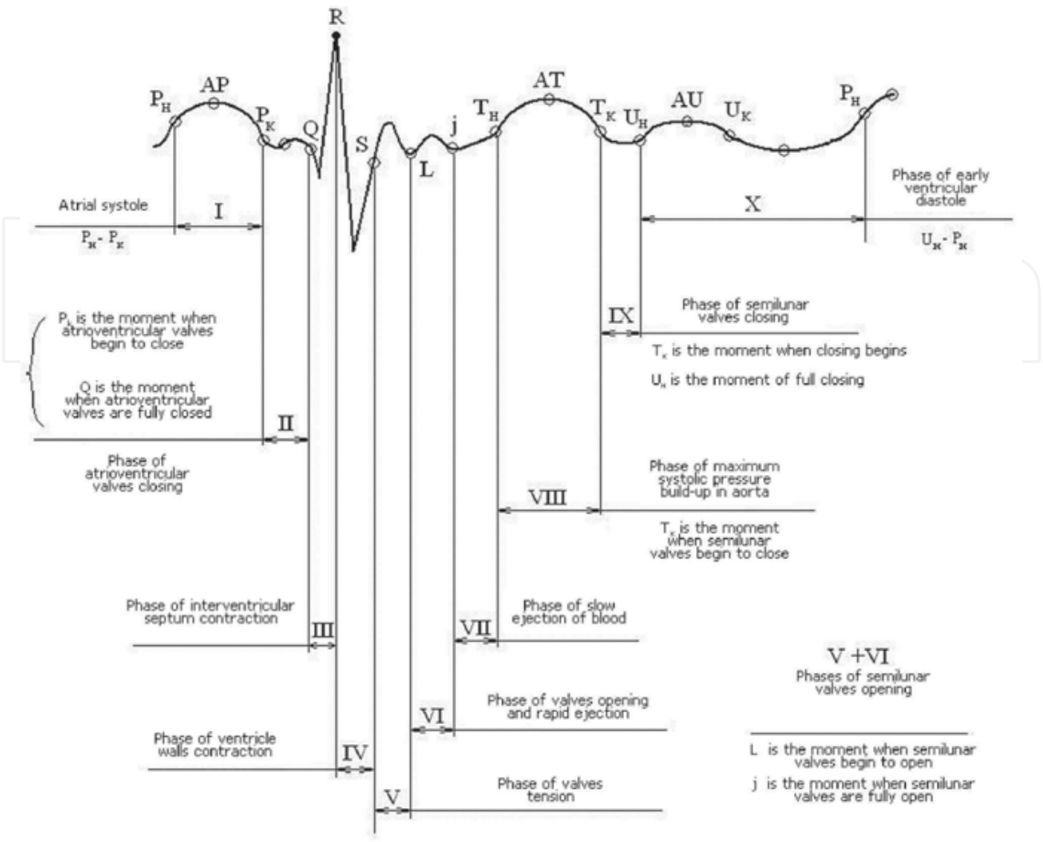


Fig. 6. Phase structure of ECG recorded from ascending aorta; Phase of atrial systole $P_н$ – $P_к$; Phase of closing of atrioventricular valve $P_к$ – Q; Phase of contraction of septum Q – R; Phase of contraction of ventricle walls R – S; Phase of tension of myocardium S – L; Phase of rapid ejection L – j; Phase of slow ejection j - $T_н$; Phase of buildup of maximum systolic pressure in aorta $T_н$ - $T_к$,; Phase of closing of aortic valve $T_к$ - $U_н$; Phase of premature diastole of ventricles $U_н$ - $P_н$

Group of systolic phases which provide for the conditions for the proper blood circulation. They can be divided into subgroups undertaking certain functions as given below:

Subgroup responsible for diastolic AP regulation:

Phase of contraction of septum Q – R;
Phase of contraction of ventricle walls R – S;
Phase of tension of myocardium S – L (partially).

Subgroup responsible for systolic AP regulation:

Phase of tension of myocardium S – L,
Phase of rapid ejection L – j.

Subgroup responsible for aorta pumping function control:

Phase of slow ejection j - $T_н$;
Phase of buildup of maximum systolic pressure in aorta $T_н$ - $T_к$,;

Phase of closing of aortic valve $T_к$ - $U_н$;

The given systolic phases are characterized by hemodynamic values PV3, PV4 and SV.

Hemodynamic value MV is an indication of a blood flow rate.

Hemodynamic parameter PV5 shows what share of blood is pumped by the aorta operating as a peristaltic pump during the ejection of blood from the ventricles.

It should be noted that phase of slow ejection j - $T_н$ is a time when the stroke volume of blood is distributed throughout the large blood vessel, i.e., the time of the aorta expansion. As our investigations demonstrate, in case of improper elasticity of the aorta this period of time is prolonged.

4. Phase structure of heart cycle on RHEO curve

An electrocardiogram reflects the most important hemodynamic processes. According to an ECG curve, it is possible to identify an intensity of the contraction of the muscles of the respective segment in the cardiovascular system by analyzing inflection points in the respective heart cycle phase and considering the respective phase amplitudes. However, it is required to understand how the flow of blood changes. For this purpose, rheography should be used. A rheogram shows changes in the arterial pressure. An ECG and a RHEO are produced by using signals of different nature. To record an ECG used is electric potential, and for RHEOgraphy employed are changes in amplitudes of high-frequency AC under the influence of changing blood volumes in blood circulation, which produce changes in the conductivity within the space between the recording electrodes.

There is no AP increase in myocardium tension phase S – L. The aortic valve opens at the moment denoted as L. The slope ratio of RHEO in phase of rapid ejection L – j is descriptive of the velocity of stroke volume travel, and, finally, decisive in governing the systolic AP.

5. Criteria for recording phases on ECG, Rheo and their derivatives

When considering an ECG as a complex signal, it should be pointed out that it consists of a number of single-period in-series sinusoidal signals connected. It is referred to a re-distribution of energy in bio systems in a not a stepwise, but sinusoidal way, showing half-periods as follows: energy increase, retardation, attenuation and development. Transition points of these processes should be at the same time the points of inflection of energy functions which are shown by the first derivative at their extrema. Similar processes occur in the cardiovascular system control. Figure 8 represents a schematic model of an ECG comprising the said in-series single-period sinusoidal waves.

Should an ECG curve be differentiated, 10 extrema on the derivative can be identified which correspond to the boundaries of the respective phases of the heart cycle. It should be mentioned that each phase shall be determined by the same criterion, i.e., by the respective local extremum on the derivative curve. Since a wavefront steepness varies, the respective amplitudes of the derivative extrema differ. The ECG phases are equivalent to those of energy variations responsible for the heart control. For illustration purposes, it is better to use graphic differentiation.

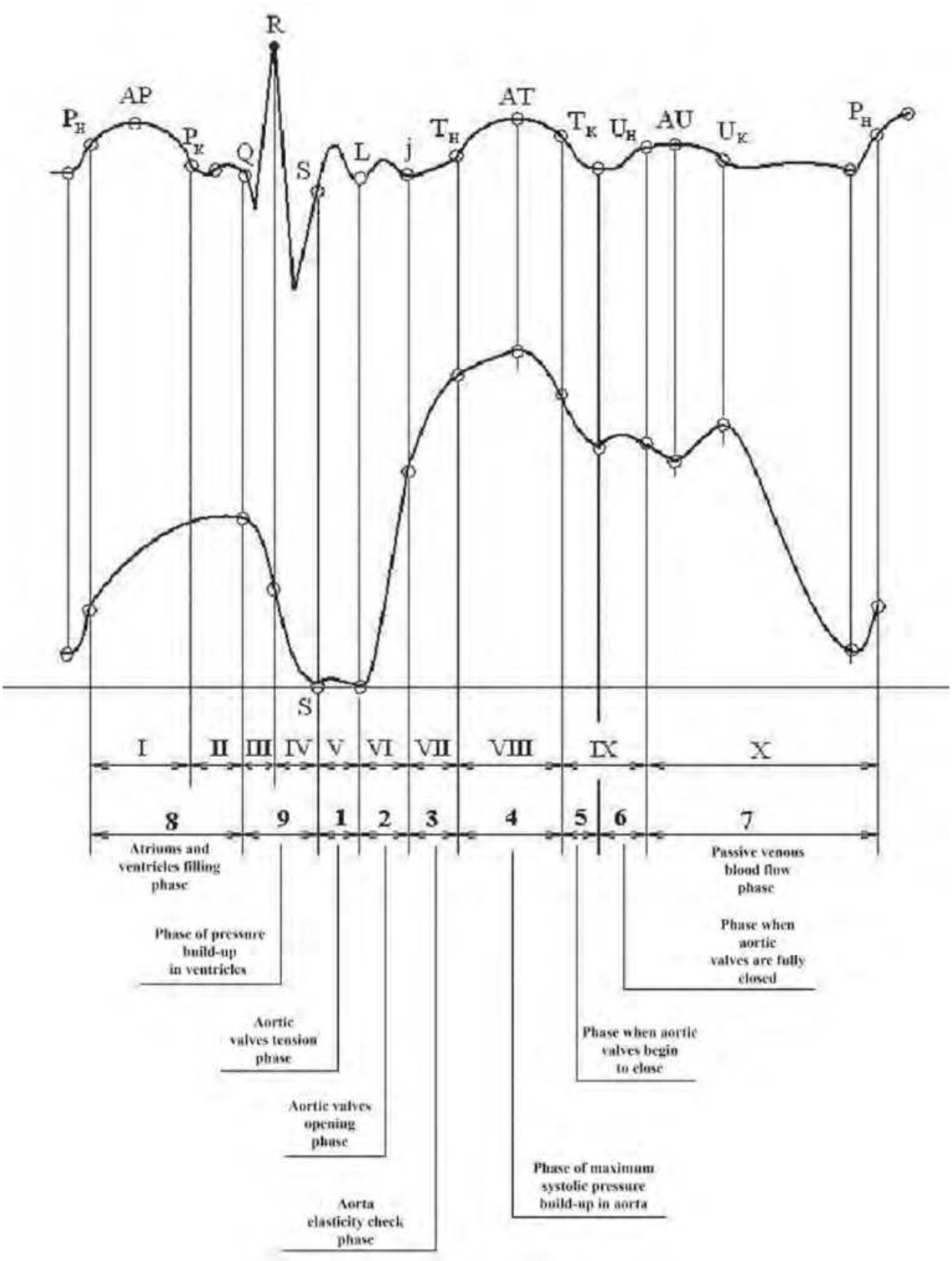


Fig. 7. Phase structure of RHEO recorded from ascending aorta

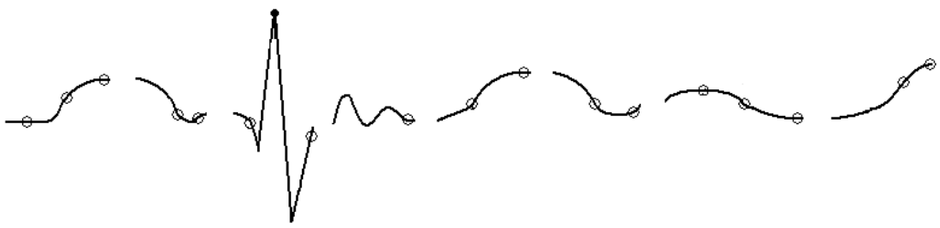


Fig. 8. Schematic model of ECG comprising in-series single-period sinusoidal variations

It is just the graphic differentiation that is capable of clearly illustrating all specific points of such complex signal like an ECG signal. Whereas it is practically impossible to detect visually on an ECG curve the inflection points, they can be easy identified on the derivative by local extrema without error. Figure 9 gives an ECG curve and its first derivative. It is evident that point P on the ECG curve corresponds to point P on the derivative that is found by the respective local extremum. In the same way point T should be identified. It is of great importance to localize point S. There are no other methods capable of identifying this point.

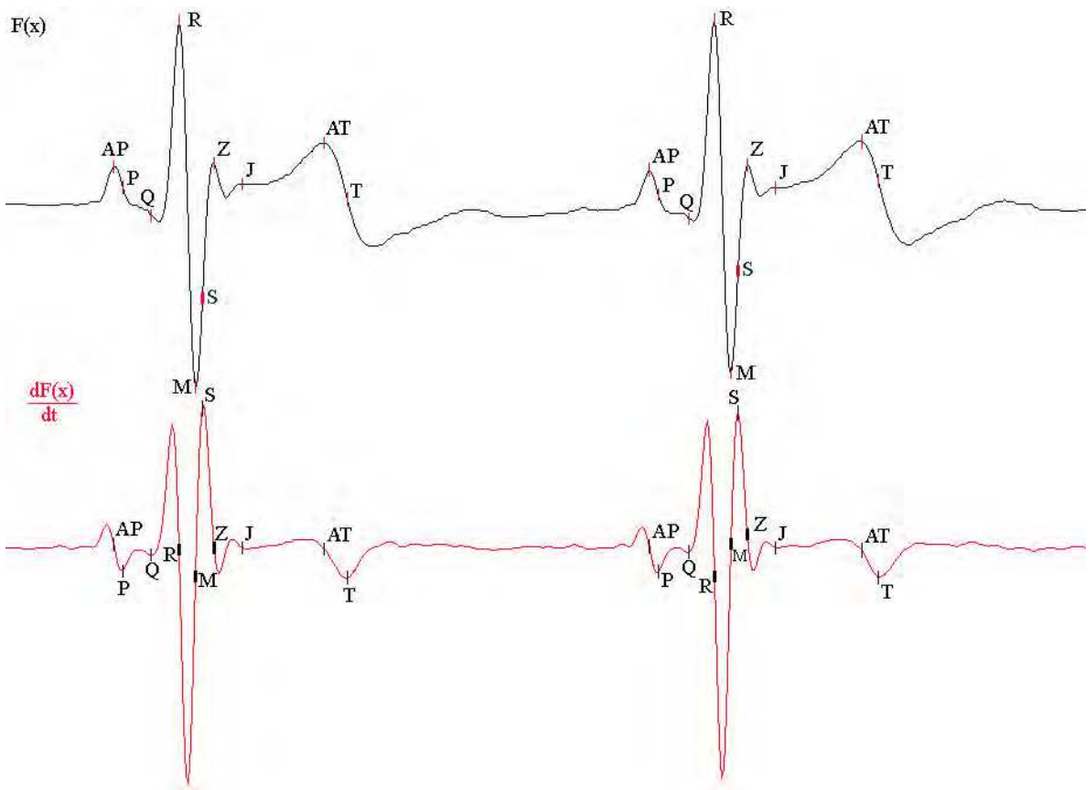


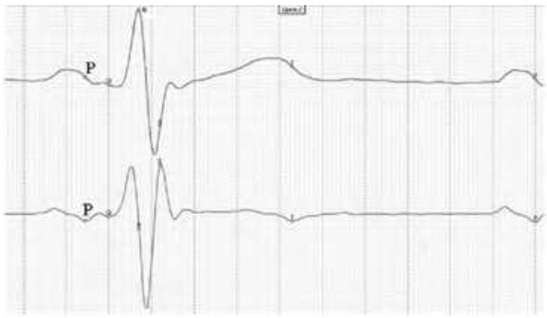
Fig. 9. Graphic differentiation of ECG curve. Shown are an ECG and its first derivative. Wave points on the ECG curve are its inflection points that correspond to the local extrema on the derivative

It is just the derivative that is capable of recognizing point S very clearly by the respective local positive extremum. The proposed procedure of identifying the above mentioned key points makes possible to develop a computer-assisted technology for measuring durations of every heart cycle phase.

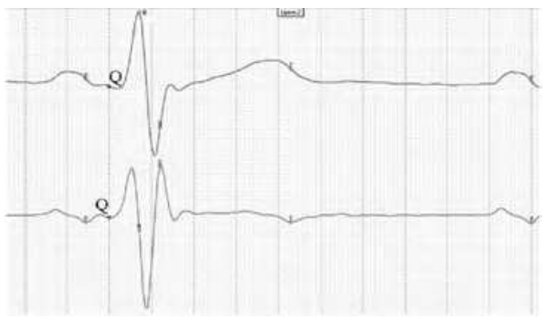
For the same purpose, the second derivative may be used, too, but in this case there is no need to do it since the informative content of the heart cycle phase identifiable criteria with utilization of the first derivative is quite sufficient.

Some real ECG curves recorded from the aorta are given in Figure 10 herein. Wave points P, Q, S and T are marked on the curves which are reliably found according to the first derivative.

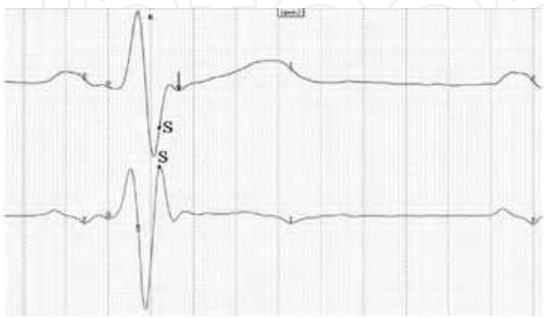
Figure 11 herein illustrates real ECG signals and the first derivative of this ECG. The ECG shape shown in this Figure is close to an ideal one. It is the matter of fact that in practice we deal with such ECG curves that significantly differ from the ideal ECG type represented herein. Therefore, it is the differentiation only that can very reliably identify the boundaries of every phase in every heart cycle.



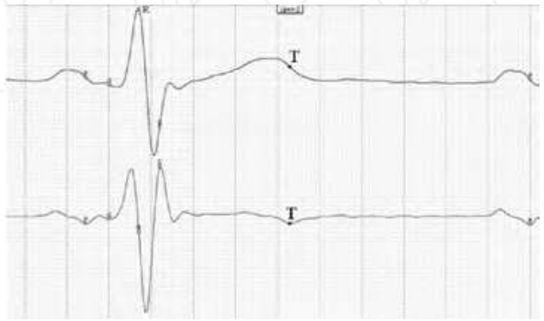
point P



point Q



point S



point T

Fig. 10. Key points P, Q, S and T on ECG curve, characterizing the respective phases of the heart cycle and corresponding to the respective local extrema on the derivative

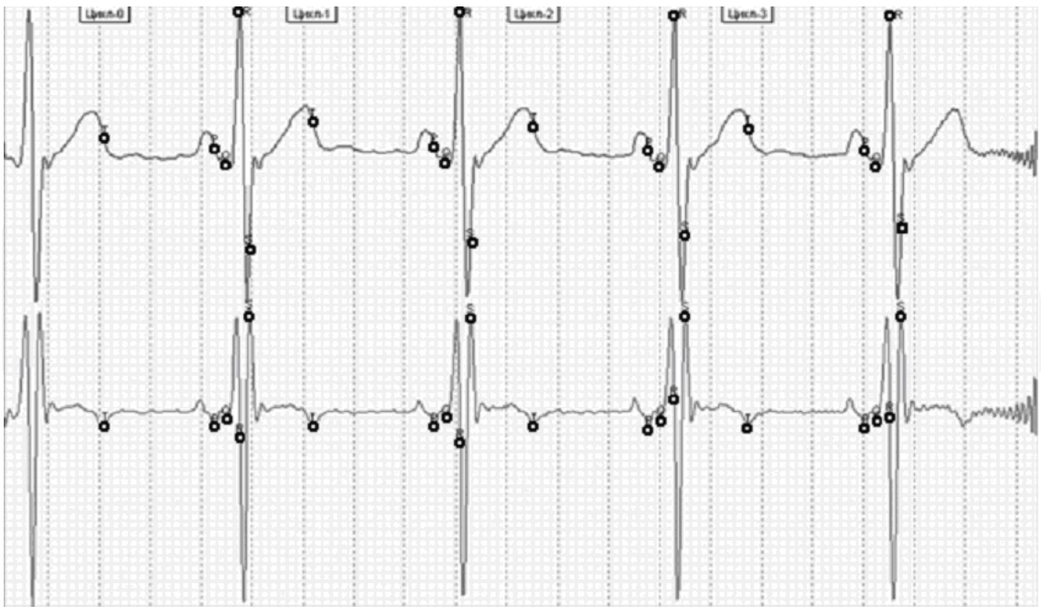


Fig. 11. Identification of phases on an ECG curve with use of the first derivative graph

6. Functions of cardiovascular system to be evaluated on the basis of heart cycle phase analysis

The complex of the functions of the cardiovascular system is a combination of the functions in every individual heart cycle phase. There is a certain logic design available explaining this. Every phase has its own significance but the basis of all phases is the mechanism of contraction or relaxation of muscles. Should metabolic disturbance in a muscle occur, its contraction or relaxation will be diminished. In this case, every next phase will undertake to compensate for this malfunction by enhancing its activity. The phase analysis gives us a clue to clearly identifying such imbalances.

In this connection, the following functions of the cardiovascular system should be mentioned:

№	Function	Regulated parameter
1	Contraction of septum	diastolic AP in the aorta
2	Contraction of myocardium;	diastolic AP in the aorta
3	Tension of myocardium muscles	systolic AP in the aorta
4	Elasticity of aorta	Maintain blood flow structure
5	condition of venous flow	
6	condition of pulmonary function	
7	whether pre-stroke conditions are available or not	
8	problems with coronary blood flow	

Table 1. Main functions and regulated parameters of cardiovascular system

Figure 12 given below demonstrates the relations between the heart cycle phases on an ECG & RHEO and the respective functions of the cardiovascular system. Although it seems that the hemodynamic mechanism as a whole and the performance of the cardiovascular system are very complicated, the heart cycle phase analysis allows establishing of cause-effect relationship of any pathology in every individual case within the shortest time. It is very important that it makes possible to detect the primary cause of a cardiac disease.

Figure 13 displays anatomic segments of the heart and their respective functions in every heart cycle phase.

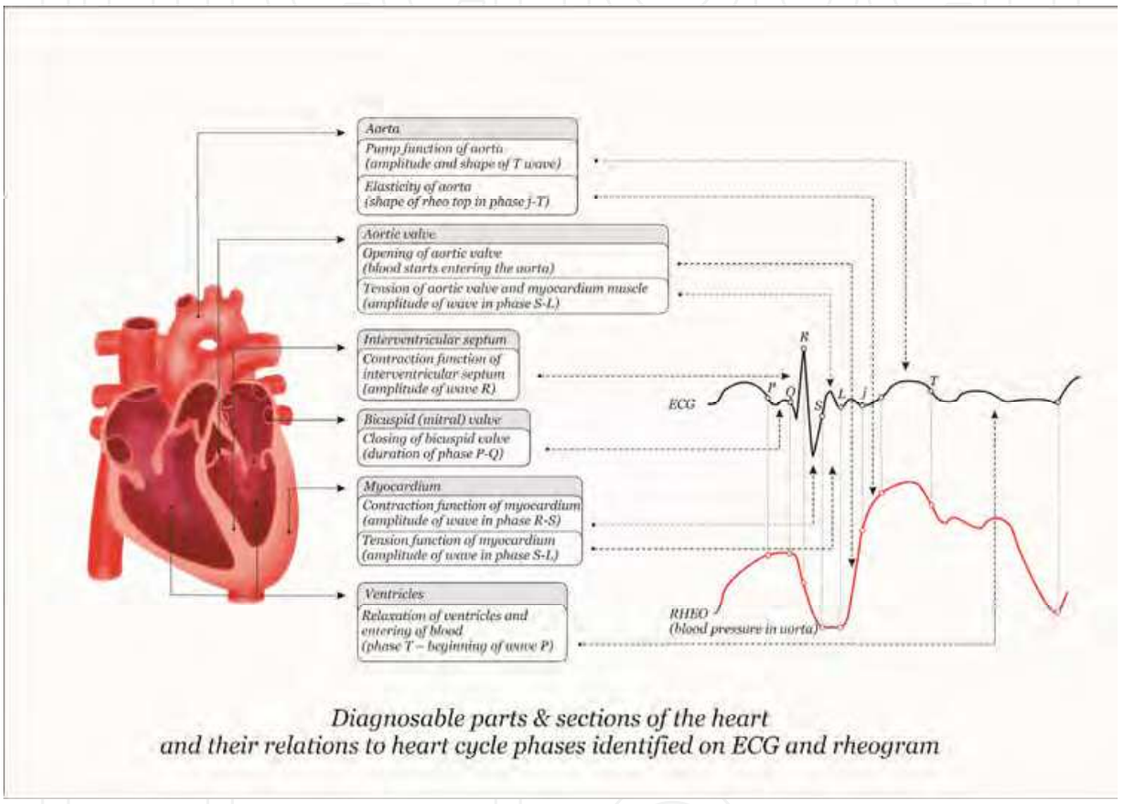


Fig. 12. Diagnosable heart segments with their functions and their relations to heart cycle phases on ECG and RHEO

7. Conclusion

Making progress in research of biophysical processes of the formation of the hemodynamic mechanism is possible only when theoretical models are tested for their compliance in practice, i.e., a model to be validated should show in practice its compliance with the requirements for all simulated functions. The results of many years' researches accumulated by our R & D team made it possible not only to develop an innovative, radically new theory of the heart cycle phase analysis but also provide metrology for such field of medical science as cardiology[4]. We have succeeded in solving the problem of indirect measuring technologies for hemodynamic parameters, including phase-related volumes, by the mathematical modeling.

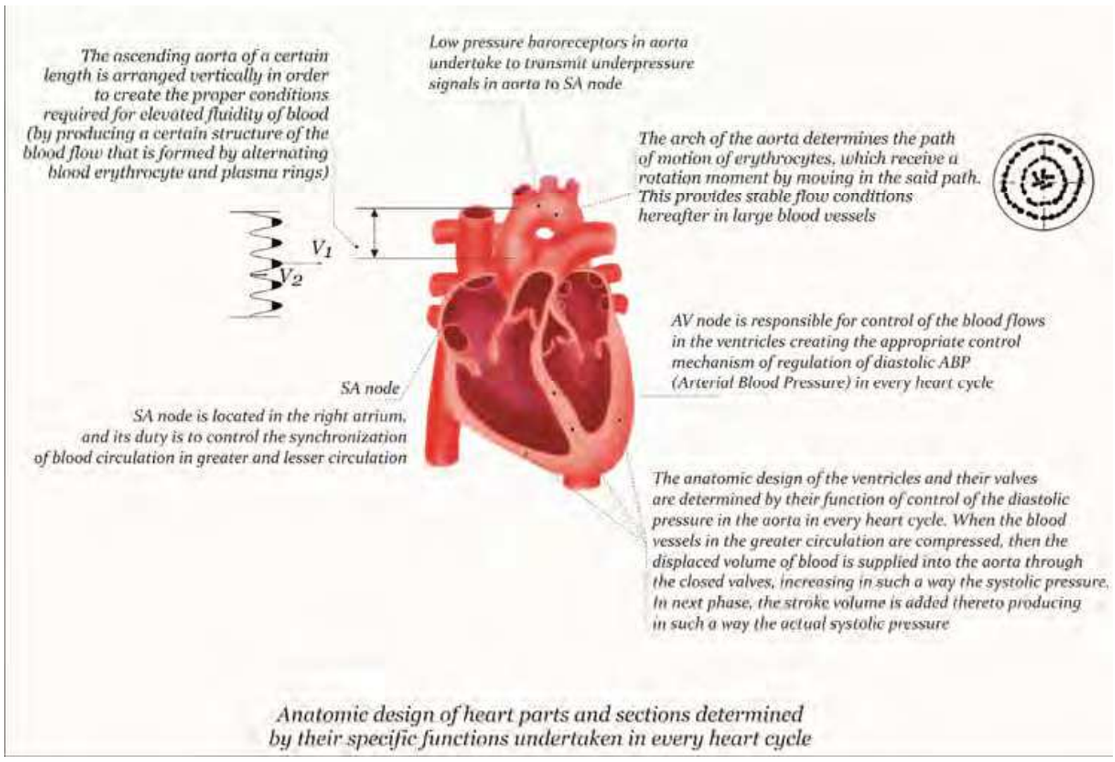


Fig. 13. Anatomical design of the heart predetermined by the required functions in every heart cycle

Our clinical studies offer a clearer view of how many difficult issues associated with biochemical reactions responsible for the stable maintenance of the hemodynamic and the entire performance of the cardiovascular system can be answered. This is a pre-requisite to developing and validating of new high-efficient therapy methods.

Hereby the authors would like to express their hope that within the nearest future we shall deal with a new research field, which is cardiometry. The basis of this science should create mathematical modeling and instrumentation technology.

8. Acknowledgements

The well-known recipe for success in any work is to create a team of like-minded researches working for the same cause. If the concept of their work is that the point of life is work, and if the work results encourage and motivate them, then success is assured. But our life is able to make its corrections. We regret to say that, one of the authors of our discovery, who originated the idea of the "third" mode of flow, died. We speak about Gustav M. Poyedintsev, a great mathematician and scientist. Our last book *Theoretical Principles of Heart Cycle Phase Analysis* published in 2007 was devoted to the memory of him and his work.

The other sad news has been received by us when we were working on this Chapter: Jaana Koponen-Kolmakova, another member of our R & D team, has departed this life. She was really an outstanding person! She was the General Manager of the Company CARDIOCODE-Finland. She remains in our memory for ever.

During our work we meet a lot of people who dedicate their life to science. We always enjoy communicating with them. This is a sort of people who deserve our special recognition and respect.

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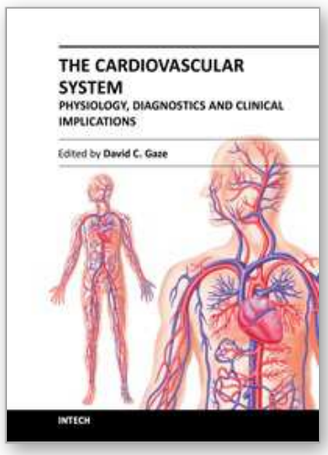

The Cardiovascular System - Physiology, Diagnostics and Clinical Implications

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The cardiovascular system includes the heart located centrally in the thorax and the vessels of the body which carry blood. The cardiovascular (or circulatory) system supplies oxygen from inspired air, via the lungs to the tissues around the body. It is also responsible for the removal of the waste product, carbon dioxide via air expired from the lungs. The cardiovascular system also transports nutrients such as electrolytes, amino acids, enzymes, hormones which are integral to cellular respiration, metabolism and immunity. This book is not meant to be an all encompassing text on cardiovascular physiology and pathology rather a selection of chapters from experts in the field who describe recent advances in basic and clinical sciences. As such, the text is divided into three main sections: Cardiovascular Physiology, Cardiovascular Diagnostics and lastly, Clinical Impact of Cardiovascular Physiology and Pathophysiology.

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
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