Viscous deformation of relaxing ventricle and pulsatile blood propelling (numerical model)

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Abstract

The numerical model of one-loop circulation exploits viscous deformation as mechanism of ventricular filling. Mathematical advantage of viscous deforming is a possibility to present the ventricular filling as the function of two independent variables (stress and time); two-dimensional argument frames a table which permits to calculate end-diastolic ventricular volume due to information about measured venous pressure and duration of ventricular diastole. The equation was deduced which balances the system while varying of such parameters as arterial resistance, the value of normal rhythm and volume flow rate. The model explains the phenomenon of asymmetrical position of normal value of rhythm, i.e. the model explains why the range of working brady-rhythms is much narrower than the range of working tachy-rhythms.

Introduction

The opinion that ventricular relaxation is a viscoelastic process can be found mentioned [1] but complexity of material adds nothing comprehensible for connection of such a specific behavior of material to the controlling of circulatory parameters. Kelvin-Foight's model is putting in action the viscous and the elastic processes in parallel; it results in the delayed reaction of the elastic material (so called, "creep") which gives no development in understanding of ventricular filling. Maxwell's model of complex material, which combines the two components for operating in series, can be considered justified if the elastic component plays, for example, the role of safety device preventing the excessive dilatation. Nevertheless the working range of dilatation (i.e. the range of filling itself) needs a description in terms of isolated viscous deforming because it possesses two advantages which ought not to be contaminated by combination with elastic deformation. Firstly, as far as viscous deformation demonstrates behavior of liquid all the information about the ventricular filling with blood (liquid) transmits to the relaxing myocardium if it also behaves like liquid (imitates the behavior of liquid during relaxation). Secondly, viscous deformation depends on the duration of process (unlike elastic one) and, consequently, pulsatile mode of propelling of blood, - which proposes invariable duration of systole and changeable duration of diastole, - must have affinity to the time-dependent process of filling because in that case the bond between deformation and rhythm appears (and, consequently, deformations become controllable). Simple numerical model of one-loop circulation that is presented here solves the problems of balancing of several basic physiological parameters in response to deviations of the system. Finally the model explains why normal rhythm is

located asymmetrically between diapason of working brady-rhythms (which is quite narrow) and diapason of working tachy-rhythms (which is much broader).

Methods

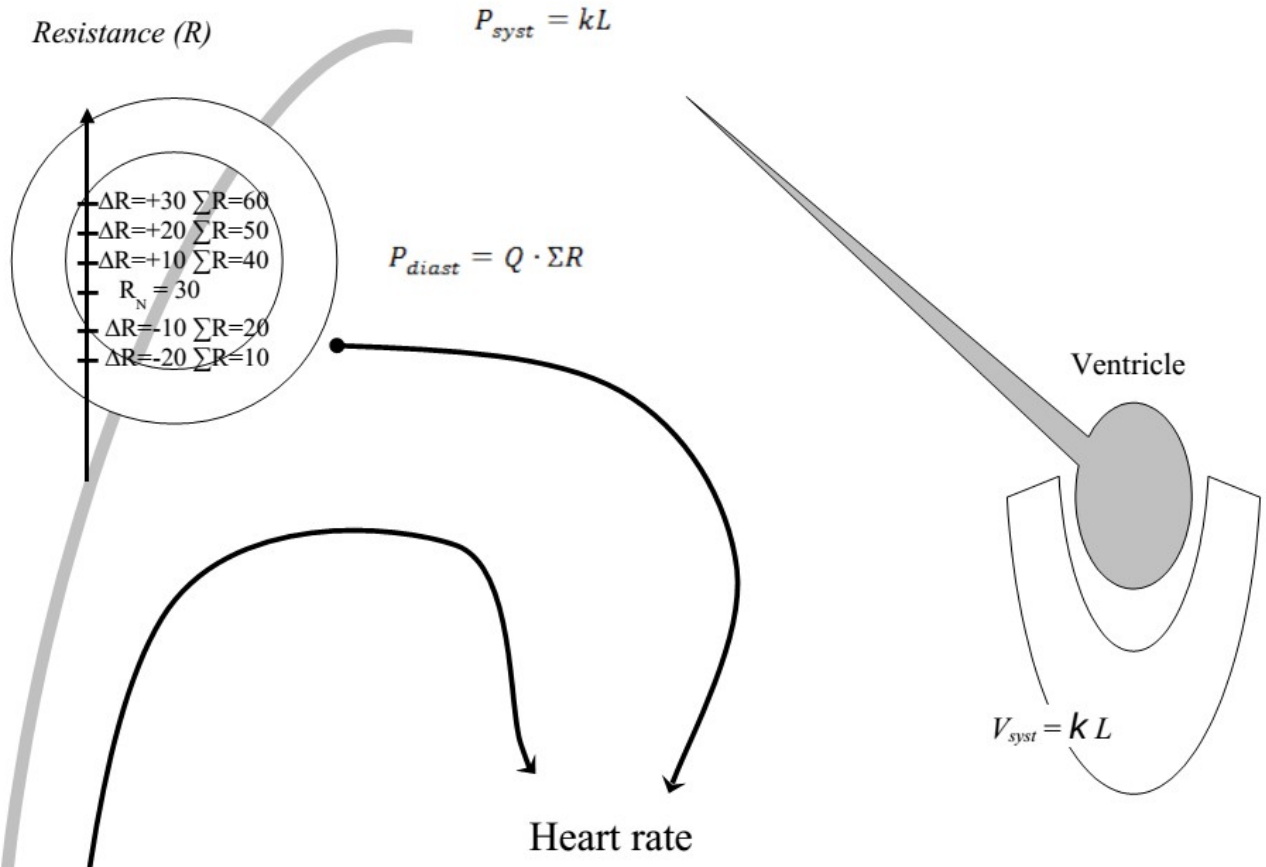
The general definition of viscous deformation is the following: tensor of stresses is a linear function of tensor of strain rates of elementary volume of liquid. We may use more simple definition (Newton's law for internal friction): $\frac{d}{dt}L=\frac{P}{M}$, where L is a deformation, P is a pressure (stress) and M is the coefficient of dynamic viscosity with dimensions of quantity $\left[\frac{n\cdot s}{m^2}\right]$; hence, $L=\frac{1}{M}Pt$ is the formula simplified for arithmetic calculations. L is the relative viscous deformation and has no dimensions of quantity $\left[\frac{m^2\cdot n\cdot s}{n\cdot s\cdot m^2}\right]$; if we want to operate with absolute viscous deformation, i.e. L gains the dimensions of volume $\left[m^3\right]$, the coefficient of dynamic viscosity M must change its dimensions of quantity to $\left[\frac{n\cdot s}{m^5}\right]$ and must change its physical interpretation. Absolute viscous deformation L $\left[m^3\right]$ depends on $\frac{1}{M}$ $\left[\frac{m^5}{n\cdot s}\right]$ and, consequently, L will grow not only due to the lower viscosity of liquid but also due to the larger volume of empty room which the definite geometry of space can place at our disposal; in other words, we must take into consideration that some outer restriction can produce the correction of the volume that we are expecting to observe according to the law of viscous deformation acting at some ideal geometry of space.

The comprehension of this relationship is more clear in terms of hydrodynamics. The parameter with dimensions of quantity $\left[\frac{n\cdot s}{m^5}\right]$ corresponds to hydrodynamic resistance R determined by standard equation $R=\frac{P_1-P_2}{Q}$, where Q is a volume flow rate and P_1-P_2 is the difference of pressures at the beginning and at the end of the pipe. We may represent Q as $\frac{V}{t}$, where V is a volume and t is a time, and then rewrite hydrodynamic equation: $V=\frac{1}{R}\left(P_1-P_2\right)t$. Compare it with the law of viscous deformation

$L=\frac{1}{M}Pt$ and the interpretation of M $\left[\frac{n\cdot s}{m^5}\right]$ will be readable due our understanding of hydrodynamic resistance R $\left[\frac{n\cdot s}{m^5}\right]$: the resistance is the compound parameter that includes either viscosity of the liquid or the friction over the wall of the pipe and the length and diameter of the pipe. All characteristics of the pipe can be called the restrictions originated from definite spatial geometry. Therefore, when we use coefficient M $\left[\frac{n\cdot s}{m^5}\right]$ we operate not only with viscosity but also with some spatial restrictions – external conditions, or medium, where the viscous deformation takes place.

For simplification of modeling the dynamical viscosity itself can be approximated to the viscosity of water (M = 1 $\left[\frac{n\cdot s}{m^2}\right]$ at temperature 20° C), and, respectively, the "external restrictions" - i.e. the another component of M $\left[\frac{n\cdot s}{m^5}\right]$, - can be assumed negligible. Therefore, we have clarified the position concerning the parameters we are dealing with: firstly, it is the absolute viscous deformation L $\left[m^3\right]$ and, secondly, it is the coefficient M $\left[\frac{n\cdot s}{m^5}\right]$ with physical interpretation different from the coefficient of dynamic viscosity M $\left[\frac{n\cdot s}{m^2}\right]$, although numerically we assume both coefficients equal.

Thus, L is the ventricular volume which is enlarging during ventricular diastole, P denotes the venous pressure, t – the duration of ventricular diastole, and M $\left[\frac{n\cdot s}{m^5}\right]$ is the coefficient we have discussed above. Viscous deformation permits to construct a table – a two-dimensional structure, - because viscous deformation is dependent of two variable parameters, P and t (i.e. stress and duration of stress); viscous deformation differs fundamentally from elastic deformation which depends on the value of stress only and is independent of the duration of stress. The table at **Fig.1** is framed on the basis of formula $L=\frac{1}{M}Pt$ described above.



duration of ventricular diastole, t

P venous pressure

	12	11	10	9	8	7	6N	5	4	3	2	1
12							**L72**	**L60 t5**				
11												
10						**L72 t7.2**	***L60*** **L60**	**L48 t4.8**				
9												
8						**L60 t7.5**	**L48** L48	L36 t4.5				
7												
6N					L48 t8		L36 **L36**		**L24 t4**			
5												
4				**L36 t9**			**L24 L24**			**L12 t3**		
3												
2	**L24 t12**						**L12**					
1												

$$L = \frac{1}{M} Pt$$

Fig.1. End-diastolic deformation of the ventricle (L) is generated by two variables due to the law of viscous deforming – venous pressure (P) and duration of ventricular diastole (t). Frank-Starling's law directly proportionally converts end-diastolic volume into systolic volume which is assumed numerically equal to systolic pressure that originates hydrodynamics in the pipe according to formula $P_{syst} - P = Q \cdot \Sigma R$, where Q is a volume flow rate in the arterial pipe, ∑R is a resistance proceeded from the work of arterial resistive vessels ($Q \cdot \Sigma R$ can be interpreted as diastolic pressure). Two reflexes (black curves begin from zones of measuring of pressure, - arterial or venous, - and both end at pace maker) regulate the responses. The scale of variation of ∑R is shown; different values of ∑R effect the correction of t jointly with P according to the deduced equation (see text); line№6 and column№6 denote normal venous pressure and normal duration of ventricular diastole, respectively. Each type of arrows forms a circle which depicts deviation and restoration of the system at each level of ∑R.

The calculated deformation L is the end-diastolic volume which is directly proportional to the systolic volume due to Frank-Starling's law [2], i.e. $V_{syst} = k$ L, where k is a coefficient of proportionality without dimensions of quantity. The significant pressure at the initial portion of arterial pipe, - the portion which is located between the ventricle and arterial resistive vessels and is presented by elastic artery with constant resistance (neglected at the present model), - exists only during period of ejection of blood from the ventricle, - as we also assume at present model. In other words, we assume that pressure – at that portion - has a substantial value only during ventricular systole but has a negligible value during ventricular diastole. It is a first application of the approach we suggest as a method of simulation of pulsatile blood flow: the principle is the following - it is necessary to combine three parameters (to construct the triple parameter concerning pressure):

- anatomical region of the pipe (i.e. we substitute the sequence of regions instead of flow travel, i.e. we simulate a movement),

- the relation to the beginning, systole, or the end, diastole, of cardiac cycle (i.e. we simulate pulsation),

- the pressure.

Therefore, at the initial portion of arterial pipe we may speak about pressure as a substantial value only during ventricular systole – and this pressure we call systolic pressure. *It is the object of investigation of the next publication – to describe in detail and to prove (by means of referring to some physiological phenomenon) the above approach in respect of arterial basin.* Now we suggest only to assume the transition from systolic volume to systolic pressure as numerical equalization but the dimensions of quantity must be specified. Consequently, if $V_{syst} = k$ L and k is a coefficient of proportionality without dimensions of

quantity we need to change the coefficient in $P_{syst}=k\,L$, where k now is a coefficient of proportionality with dimensions of quantity $\left[\frac{n}{m^5}\right]$. Utmost simplification claims that k is equal to 1, and, therefore, we assume that systolic pressure is numerically equal to end-diastolic deformation and that systolic pressure, as a parameter, is bound to the portion of arterial pipe between the ventricle and resistive arteries.

Let us continue the attaching of pressures to anatomical portions of pipe and to cardiac cycle. The function that determines the bond between the pressure difference at the beginning and the end of a pipe, P_1-P_2 , the volume flow rate, Q, and hydrodynamic resistance, R, is standard $R=\frac{P_1-P_2}{Q}$. It can be represented as $P_{syst}-P=Q\cdot\Sigma R$, where $Q\cdot\Sigma R$ has the dimensions of quantity of pressure and so let us associate this pressure with the pressure that we observe during the late period of cardiac cycle and let us assume that it can be registered, as a parameter, only at the basin of arterial resistive vessels; therefore, we speak about diastolic pressure: $P_{diast}=Q\cdot\Sigma R$. The final portion of the pipe, - the vein, - possesses the pressure that can be called the residual pressure $P=P_{syst}-P_{diast}=P_{syst}-Q\cdot\Sigma R$ and it is attached neither to systole nor to diastole (or is attached to both ones) due to the introduction of capillary damper which is located after the basin of resistive arteries. The resistance of capillary damper can be considered constant and at the present model we assume it negligible; we remind that the resistance of the initial elastic artery is neglected also and, consequently, the general resistance of the whole pipe consists only of the arterial resistance which is changeable and controllable.

We may interpret diastolic pressure as a subtrahend that takes away some part from initial pressure, i.e. it converts systolic pressure into venous pressure: $P_{syst}-P_{diast}=P$. Anatomically the subtraction takes place between the initial elastic artery which is supplemented with the minuend (systolic pressure) and the region of resistive arteries which is supplemented with the subtrahend (diastolic pressure which is represented via the resistance multiplied by the volume flow rate). It is pertinent to emphasize that we can not speak about systolic pressure inside the region of resistive arteries because systolic pressure disappears here (minuend transforms into remainder); situation is symmetrical to the one at the region of initial elastic artery, - we can not speak about diastolic pressure because the substantial values of pressure are presented here only during ventricular systole. Certainly, the capillary damper must be added to the operation of subtraction – and at the present model capillary damper does not increase the resistance but only liquidates the division systole-diastole. Therefore, the remainder (venous pressure) appears behind all these operators and is associated with the final portion of pipe (vein) and together with lack of pulsatile flow.

Besides, the subtrahend consists of two components (∑R and Q) and it permits to be aware of the volume flow rate if we know the value of diastolic pressure (pressure at the end of cardiac cycle which can

be measured by baroreceptors in resistive arteries) and if we also possess the direct information from muscles of resistive arteries about the extent of narrowing of the pipe. For the most part of further variations we consider Q of a constant value and equal to 1; in other words, the information from baroreceptors and the information from muscles of resistive arteries will coincide. Such a condition is not groundless as far as it reflects the existence of some optimal capillary flow which corresponds to stability of biochemical processes of cell metabolism. Nevertheless, it does not mean that Q is not able to be increased, and such deviation will also be carried out and scrutinized at the final part of testing of the model. Therefore, ∑R is numerically equal to diastolic pressure when Q = 1, i.e. $P_{diast} = Q \cdot \Sigma R = 1 \cdot \Sigma R \quad \left[\frac{n}{m^2}\right]$.

The quanta of columns and lines of the table at **Fig.1** were arbitrary chosen but it was convenient to frame 12X12-table for making use of minimal whole numbers denoting increments of venous pressure and duration of ventricular diastole (although later, while deviating the system, the advantage of making operations with whole numbers will be lost); the normal value of venous pressure and the normal value of the duration of ventricular diastole were placed in the middle of the scales (line №6 and column №6, respectively). Now it is necessary to determine the state of equilibrium based on normal parameters of: 1) venous pressure, 2) duration of ventricular diastole and 3) the resistance. Since $P = P_{syst} - Q \cdot \Sigma R$ and $P_{syst} = k\,L = \frac{k}{M} P \cdot t$ it is convenient to assume that normal resistance, R_N, is equal to some number of 30 because it permits to get the remainder (normal venous pressure) equal to 6 – after the subtraction of the value of diastolic pressure (R_N multiplied by Q = 1) from the value of normal ventricular end-diastolic deformation (which is numerically equal to systolic pressure). This quantity of 6 (normal venous pressure) participates in calculation of normal end-diastolic deformation – which is the product of normal venous pressure and normal duration of ventricular diastole, - and if we choose quantity of 6 either for the normal value of the duration of ventricular diastole (and taking into account that M is equal to 1 and k is equal to 1 either) we get $L = \frac{1}{1}(6 \times 6) = 36$. After subtraction of 30 ($P_{diast} = Q \cdot R_N = 1 \cdot 30 = 30$) from 36 (P_{syst} is numerically equal to L) we get the quantity equal to 6. This quantity of 6 $\left[\frac{n}{m^2}\right]$ turns back to the table as the number of line that denotes the normal venous pressure $P_N = 6$. The numerical expression of equilibrium of three parameters is found and this equilibrium characterizes the normal, non-deviated circulation (one-loop circulation).

Two reflex arcs carry out the controlling and both begin from the reception of pressure: the one (analog of carotid reflex) decelerates rhythm in response to elevation of diastolic pressure, P_{diast} , and accelerates rhythm in response to descending of P_{diast} ; this reflex measures pressure inside resistive artery at

the end of cardiac cycle (diastolic period), - although we have mentioned that in general case the direct information from the muscles of resistive arteries is indispensable, i.e. the parallel pathway transporting such information must exist; the other reflex (analog of Bainbridge's reflex which existence is hardly proved but the term is eligible) accelerates rhythm in response to elevation of venous pressure, P, and decelerates rhythm in response to descending of P.

Results

Let us trace the reorganization of the system after some minimal, additional to normal, heightening of the resistance, $\Delta R = 10$; hence, the new, i.e. deviated, value of the resistance is the following: $\Sigma R = R_N + \Delta R = 30 + 10 = 40$. The elevated resistance needs the augmentation of contractile force, and the increasing of systolic pressure, respectively, - because the previous one, $P_{syst} = 36$, will not overcome the new value of diastolic pressure, $P_{diast\ new}$:

$$P_{syst} = \frac{k}{M} P \cdot t = \frac{1}{1}(6 \times 6) = 36 \quad ,$$

$$P_{diast\ new} = Q \cdot \Sigma R = 1 \times 40 = 40 \quad .$$

The only way to achieve the prevalence of $P_{syst\ new}$ over $P_{diast\ new}$ is to create higher value of systolic pressure my means of elongating of the duration of ventricular diastole, t, because the venous pressure is still normal, P = 6, and there is no direct influence upon it; moreover, we do not determine the duration of the delay of changing of venous pressure – the delay that inevitably appears due to the work of capillary damper.

So it is necessary to introduce one more assumption (condition) pertinent for this model: the reaction of carotid reflex upon the change of the resistance is extremely quick (within one heart beat); this assumption is indispensable for conservation of the value of venous pressure that was registered just before the change of the resistance inside the arterial basin. We substitute extreme quickness of the reaction instead of determination of the delay of change of venous pressure in response to the events happening inside the arterial basin – variations of the resistance (coinciding with diastolic pressure when Q = 1) and systolic pressure. In other words, the degree of inertness of converting of systolic pressure into venous pressure, - i.e. the speed of subtraction of P_{diast} from P_{syst} supplemented with liquidation of division systole-diastole, - i.e. factually the inertness generated mostly by capillary damper, - is omitted in the model.

Thus, quick reaction of carotid reflex is the following: the detection of the rise of $P_{diast\ new}$ results in deceleration of rhythm. But what will be the new value of rhythm?

The answer needs some speculations. Variation of rhythm can be equalized (with inversion) to the variation of the duration of ventricular diastole because the duration of ventricular systole can be considered constant at any rhythm. Elongation of the ventricular diastole, – multiplied by the value of present venous pressure (which is considered normal), – will result in expanding of deformation; the expanded deformation

will generate augmented contractile force (systolic pressure, respectively) which will permit to overcome the elevated resistance ∑R = 40. If the residual pressure (venous pressure) is still P = 6 (i.e. $P_{syst\,new} - P_{diast\,new} = P_{syst\,new} - Q \cdot \Sigma R = P_{syst\,new} - 1 \times 40 = 6$) it means that the mechanism of response is aimed to maintain venous pressure at constant value; in other words, the mechanism of response involves only the one variable factor - duration of ventricular diastole, - into the process of expanding of viscous deformation, and the another factor, venous pressure, remains not involved. Obviously such an approach leads to early depleting of the ability to react: few steps of the resistance rising will lead to the unreal values of deceleration of the heart rate. On the contrary, the approach with both factors participating in expanding of deformation permits to reorganize circulation in response to broader diapason of rising and falling of the resistance. Therefore, the residual pressure (venous pressure) must be found on some higher level (7 or higher, for the present instance). Certainly the Bainbridge's reflex will respond to: firstly, the reflex will detect the elevation of the venous pressure and, secondly, it will accelerate rhythm. The product of new P, which is higher then normal, and newly accelerated rhythm (newly shortened t), - i.e. the product that creates end-diastolic deformation of ventricle (which then will be transformed by the work of myocardium into systolic volume), - is aimed to retain the chosen higher level of venous pressure but we still have no criterion concerning what is "higher". We may only assume that this higher level must be the nearest - unless we lose all the advantage of second factor – but what is the nearest level? Or maybe there is the optimal new level? At least we may frame the equation based on the following equalities:

$$\frac{1}{M} P_{initial} \times t_{decelerated} = L_1 \quad ; \quad \frac{1}{M} P_{new} \times t_{newly\,accelerated} = L_2 \quad ; \quad L_1 = L_2 \quad .$$

We considered L_1 equal to L_2 because we deal with the same deformation – the one is achieved by elongation of diastole (P is constant) and another is achieved by new higher level of P combined with restoration of initial duration of diastole, - which was registered before the elevation of the resistance, - and which we call as "newly accelerated" naming it in terms of rhythm; hence,

$$\frac{1}{M} P_{i} \times t_{d} = \frac{1}{M} P_{n} \times t_{na} \quad , \qquad \{1\}$$

where $_i$ – initial, $_d$ – decelerated, $_n$ – new, $_{na}$ – newly accelerated.

The value of deformation L_2 produces systolic pressure which overcomes elevated resistance ∑R :

$$k L_2 - Q \cdot \Sigma R = P_n \quad , \text{ as far as } \quad P_{syst} = k L \quad \text{and} \quad P_{diast} = Q \cdot \Sigma R \quad .$$

Hence, taking into account that $k L_2 = \frac{k}{M} P_n \cdot t_{na}$, we use the right part for substitution:

$$\frac{k}{M} P_n \cdot t_{na} - Q \cdot \Sigma R = P_n \quad , \text{ and after transforming we obtain } \quad P_n = \frac{Q \cdot \Sigma R}{\frac{k}{M} t_{na} - 1} \quad .$$

Then we substitute this formula to {1} and reach the final expression:

$$t_d = \frac{Q \cdot \Sigma R \cdot t_{na}}{P_i\left(\frac{k}{M} t_{na} - 1\right)} \qquad \{2\}$$

Consequently we must know beforehand what is the value of rhythm which is "newly accelerated" (t_{na}), i.e. we must know the value of rhythm which is ought to be restored, and, as the way out, we must introduce (postulate) the concept of normal heart rate. (Certainly the normal rhythm may be chosen according to stipulation – because there is no constant numerical value of t_{na} in {2}, - and our abstract calculations can be related to any rhythm considered as normal.) Therefore, we have postulated that among three parameters that are responsible for equilibrium (imbalance, point of balance and counterbalance) – the one is automatically reversible, i.e. it always recurs to its normal level, whereas two other parameters, - resistance, ∑R, (producing deviation) and venous pressure, P, - conserve their changed levels (or, better to say, - conserve the equipoise achieved by the changed levels). It is clear that ∑R is the source of imbalance; consequently, it is necessary to have at disposal: a point of balance and a counterbalance. Postulation of recurrence to normal rhythm is the introduction of the point of balance; respectively, venous pressure can play the role of counterbalance.

Formula {2} was deduced for the description of process of increasing of arterial resistance but evidently the process of decreasing is symmetrical with the process of increasing and, respectively, we must change indexes taking into consideration that now we need more general ones. Thus, index d (decelerated) must be replaced by ch (changed, i.e. decelerated or accelerated due to the rise or drop of the resistance) and index na (newly accelerated) must be replaced by N (normal, because we postulated the restoration of normal rhythm – after elevation or decreasing of the venous pressure). Index $_i$ (initial) at P_i remains the same because, in general case, the level of venous pressure that participates at formula {2} is the level registered before the forthcoming change of the resistance, i.e. P_N is the particular case of P_i. So, we are to rewrite {2}:

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} \qquad \left[\frac{m^3 \cdot n \cdot s \cdot s \cdot m^2}{s \cdot m^5 \cdot n \cdot \frac{m^5 \cdot n \cdot s}{n \cdot s \cdot m^5}} = s\right] \qquad \{3\}$$

We have stopped tracing the reaction of the system in response to minimal elevation of the resistance ($\Delta R = 10$) when we have stumbled upon the question: what is the new value of rhythm (decelerated rhythm)? Now we can get the answer. Carotid reflex, firstly, detects the resistance by means of measuring of P_{diast} and in parallel information about ∑R comes directly from the muscles of arterial resistive vessels, - the pair which is necessary for calculation of volume flow rate (the problem of discrepancy between P_{diast} and ∑R,will be scrutinized below). Secondly, carotid reflex, - or some "calculative center", - solves the equation {3}, substituting $t_N = 6$, $\sum R = 40$ and $P_i = P_N = 6$ into {3}; hence, the value of elongated diastole appears and let it be denoted as t_{shift} because the change of duration of diastole takes place when the level of venous pressure is not changed yet, i.e. we observe the shift of acting cell along the same line (in the table of **Fig.1**):

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i(\frac{k}{M}t_N-1)}=\frac{1\cdot 40\cdot 6}{6(\frac{1}{1}6-1)}=t_{shift}=8$$

Thus, decelerated rhythm was found ($t_{shift} = 8$) and it must be multiplied by the value of the normal venous pressure $P_i = P_N = 6$ according to $L=\frac{1}{M}Pt$ (and taking into consideration that $\frac{1}{M}=1$); the product is the end-diastolic deformation L = 48 which generates systolic pressure numerically equal to L, i.e. $P_{syst} = 48$. As far as $P_{diast} = Q \bullet \Sigma R = 1 \bullet 40 = 40$ it is possible to find residual pressure (venous pressure) according to formula $P=P_{syst}-Q\cdot\Sigma R$. Hence, P = 48 – 40 = 8, i.e. the new $P_i = 8$. Horizontal thin non-doted arrow points to the left: the initial acting cell 6_pX6_t passes on to the new acting cell 6_pX8_t .(Here and below: $_p$ marks the line, i.e. P; and $_t$ marks the column, i.e. t. When decimal fractures for denoting t and P_i appear we are in need to picture the calculated values of t and P_i inside the cells with whole indexes $_t$ and $_p$ which are closest, - or graphically convenient, - to calculated values of t and P_i).

Bainbridge's reflex, firstly, detects the level of venous pressure and, secondly, solves the same equation {3} that previously was solved by carotid reflex:

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i(\frac{k}{M}t_N-1)}=\frac{1\cdot 40\cdot 6}{8(\frac{1}{1}6-1)}=t_N=6$$

Therefore, Bainbridge's reflex accelerates rhythm, i.e. it shortens the duration of diastole down to $t_N = 6$. In other words, Bainbridge's reflex makes normal heart rate recurred: thin non-doted arrow points upwards and to the right, - i.e. the acting cell 6_pX8_t passes on to the cell 8_pX6_t . Now circulation is stabilized in the respect of heart rate (the normal pulse is restored according to postulated condition that the system must be aware in advance about the value of rhythm the system must be returned to) but other measurable parameters are changed: the resistance (and P_{diast}, respectively) is elevated from 30 up to 40, P_{syst} is increased from 36 up to 48 and the venous pressure is also increased from 6 up to 8. Thus, we now observe new equilibrium that has replaced the previous one which was poised within normal parameters only; this new equilibrium demonstrates the existence of two steps of searching the balance: 1) the violation of the balance by elevated resistance (imbalance) makes the system to react immediately and restore equilibrium by changing of the point of balance ($t_N = 6$ was changed to $t_{shift} = 8$) and it is transitory step; 2) then the counterbalance appears (raised venous pressure) and it is factually the repeated violation of balance (transitory balance), i.e. raised venous pressure compels the system to restore the previous point of balance ($t_N = 6$).

The way backwards begins from the command to decrease the resistance, i.e. $\Delta R = -10$; obviously the resistance will fall from $\sum R = 40$ to $R_N = 30$ and the muscles of resistive arteries will inform the pacemaker (or "calculative center"of the pacemaker) about the drop of the resistance. In parallel the baroreceptors of carotid reflex will detect the decline of diastolic pressure which coincides with the

decreasing of the resistance (due to the fact that Q = 1), - and "calculative center" begins to solve the equation {3} in order to find t_{ch} (exactly, t_{shift}) which, for the present instance, denotes the acceleration of rhythm:

$$t_{ch}=\frac{Q\cdot \Sigma R\cdot t_N}{P_i(\frac{k}{M}t_N-1)}=\frac{1\cdot 30\cdot 6}{8(\frac{1}{1}6-1)}=t_{shift}=4.5$$

The acting cell is now $8_pX4.5_t$ (located inside the cell 8_pX5_t at **Fig.1**) – horizontal thin non-dotted arrow points to the right from the previous acting cell 8_pX6_t. The product, i.e. end-diastolic deformation, is L = 8 • 4.5 = 36. Diminished deformation generates the diminished systolic pressure, - which we consider numerically equal to L, - i.e. P_{syst} = 36. This value of systolic pressure overcomes $P_{diast} = Q \bullet R_N = 1 \bullet 30 =$ 30; the residual pressure (venous pressure) is falling: $P=P_{syst}-Q\cdot \Sigma R=P_{syst}-Q\cdot R_N=36-1\cdot 30=6$, and the decline of it will be detected by receptors of Bainbridge's reflex (venous pressure descends from 8 to 6). Then Bainbridge's reflex solves the equation {3} in order to find how intensively the rhythm must be decelerated:

$$t_{ch}=\frac{Q\cdot \Sigma R\cdot t_N}{P_i(\frac{k}{M}t_N-1)}=\frac{1\cdot 30\cdot 6}{6(\frac{1}{1}6-1)}=t_N=6$$

Thin non-dotted arrow points downwards and to the left; it indicates that the acting cell is again the cell 6_pX6_t ; the value of P_i is now equal to 6, i.e. $P_i = P_N$, and , therefore, circulation has restored its initial parameters (which were balanced before the deviation).

Two observations concerning this primary example of violation of balance and restoration of balance ought to be mentioned.

1) It seems that Bainbridge's reflex does not need to calculate t_{ch} by means of equation {3} because Bainbridge's reflex, as it seems, has the only one function - to normalize rhythm. What is it worth of measuring venous pressure then? The venous pressure can be elevated and can be declined as a consequence of perturbations at the arterial basin, i.e. it is the passive behavior. It is pertinent to ask, - can the fluctuation of pressure exist without controlling the limits of elevation and falling of pressure? Obviously, it can not exist, - first of all, due to the restricted characteristics of material of the wall of the vein. Hence, some mechanism of active controlling of venous pressure suggests itself. Firstly, such mechanism must be able to measure the level of venous pressure; secondly, such mechanism must possess some effector which influences on the venous pressure (and the role of muscles of the venous pipe can be excluded immediately, although the tonicity of veins is a changeable value). The present model introduces Bainbridge's reflex as, firstly, the receptor of venous pressure and, secondly, as effector influencing on the heart rate. While varying of the arterial resistance (and later such deviation will be superimposed on the forcing of volume flow rate) the role of Bainbridge's reflex is to restore equipoise by means of normalizing of rhythm; nevertheless, Bainbridge's reflex has access to calculations based on all the parameters of balanced circulation and finally

effects rhythm. Taking into consideration that postulate of constancy of normal rhythm, as a point of balance, does not claims the conservation of some numerical value (once and forever) but permits to switch the mode of circulation (choosing more rapid or more slow normal rhythm) we may suppose that Bainbridge' reflex (exactly, the mechanism standing behind it) pretends to be the regulator of venous pressure (and, consequently, to be the "conductor" of circulation).

2) It is noticeable that both participants of regulation, - carotid reflex (together with parallel pathway bringing information from muscles of resistive arteries) and Bainbridge's reflex, - both used the same formula {3} for calculating t_{ch} and, consequently, carotid reflex must be aware about venous pressure, P_i, and, consequently, Bainbridge's reflex must measure venous pressure and grant the data to carotid reflex; respectively, Bainbridge's reflex must be aware of the resistance, $\sum R$. Besides, proper computing of t_{ch} claims the knowledge of the volume flow rate, Q , and we have already mentioned that Q can be found quite easily by means of comparison the information from baroreceptors (P_{diast}) and information from the muscles of resistive arteries (direct information about $\sum R$). On the whole, we may emphasize that calculation of t_{ch} needs the existence of process of communication between arterial source of information and venous one.

Thus, we have traced the reorganization of circulation caused by minimal rising of the resistance and further restoration of normal value of the resistance. **Fig.1** shows the examples of three more elevations of the resistance with increment $\Delta R = 10$ and two declines of resistance with decrement $\Delta R = -10$ (each step of rising or falling of resistance is accompanied by the recurrence to the initial state, i.e. by the recurrence to the previous step) and the reader can calculate by himself the parameters depicted in the acting cells of the table: L – end-diastolic deformation numerically equal to systolic pressure, t – duration of ventricular diastole (the value of t_N in column №6 is omitted because it is always equal to 6, i.e. it is normal).

Now let us have a look at all the responses shown at **Fig.1** and determine the type of function between venous pressure, P_i, and duration of ventricular diastole, t_{ch}. We may represent formula {3} the following way: $t_{ch} = K \cdot \frac{1}{P_i}$, where $K = \frac{Q \cdot \Sigma R \cdot t_N}{\frac{k}{M} t_N - 1}$ is the compound factor. This factor is variable due to the variability of $\sum R$ only and, consequently, the numerator is represented by one variable value. The denominator is presented by P_i and it is also the variable value. These two variable values are not independent – if we speak about both of them, - and one is functionally bound to another (with no difference what is the independent variable). Moreover, we may say that such function is linear because increments of $\sum R$ and P_i are steady: $\Delta R = 10$ and $\Delta P_i = 2$ (see the table at **Fig.1**). Therefore, the type of function $t_{ch} = K \cdot \frac{1}{P_i}$ depends of quantitative ratio of nominator and denominator: if growth of nominator prevails the function resembles to direct proportion (linear function), and if growth of denominator prevails the

function resembles the inverse proportion (non-linear function). In the table at **Fig.1** we may see the two series of values of t_{ch} (exactly, t_{shift}) which frame two symmetrical graphs (visualized by working cells in the table) and which look very much like hyperbole (respectively, the values of duration of ventricular diastole, t, form non-linear sequence of quantities). Symmetry axis is the line of acting cells $(2, 4\ldots12)_pX6_t$ where 6_t is the duration of normal ventricular diastole, i.e. symmetry axis denotes the equipoise at each level of P_i and $\sum R$ which does not belong either to hyperbole that was formed while the increasing of the resistance or the hyperbole that was formed while the decreasing of the resistance.

Fig.2 and **Fig.3** show additional deviations that are provoked by substitution of some other values of normal duration of ventricular diastole (normal rhythm) instead of $t_N = 6$ into formula {3}; the change of t_N entails the change of normal venous pressure, P_N, because normal resistance, R_N, is still equal to 30. In other words, when we have shifted the position of the point of balance we observe that counterbalance (venous pressure) changes its value upon standard variation of the resistance. The graph from **Fig.1**, - where deviations of the resistance is shown at $t_N = 6$, - is supplemented to **Fig.2** and **Fig.3** for comparison.

At **Fig.2** the new normal rhythm is more rapid, i.e. the duration of ventricular diastole is shorter and it is equal to 3 (3N at abscissa); the calculation of equipoise for standard normal resistance $R_N = 30$ results in changing of values of normal end-diastolic deformation of ventricle (L = 45) and normal venous pressure ($P_N = 15$ at ordinate). The regime of deviating of the system is the same as the initial one (which is shown nearby as a duplicate of graph from **Fig.1**), so we decided not to overload the construction with arrows indicating each step of deviation and further restoration of equilibrium, - we only marked out the acting cells of the table (this is true for **Fig.3** either). There are also two symmetrical hyperbolic graphs but general configuration is more stretched and compressed by the sides; it means that diapason of working rhythms is much narrower than the one at the example shown for the comparison below, but diapason of working values of venous pressure is broader. The parameter that mostly changed is venous pressure: $P_i = 70$ at the highest point comparing to $P_i = 12$ at the highest point at the picture below; therefore, circulation that is based on rapid heart rate maintains very high levels of venous pressure when the resistance is growing.

	12	11	10	9	8	7	6N	5	4	3N	2	1
30										L90	L75 t2.5	
29												
28												
27												
26												
25									L90 t3.6	L75	L60 t2.4	
24												
23												
22												
21												
20									L75 t3.75	L60	L45 t2.25	
19												
18												
17												
16												
15N									L60 t4	L45	L30 t2	
14												
13												
12												
11												
10								L45 t4.5		L30		L15 t1.5
9												
8												
7												
6												
5							L15 t6			L15		
12							**L72**	**L60 t5**				
11												
10						**L72 t7.2**	***L60*** **L60**	**L48 t4.8**				
9												
8						**L60 t7.5**	**L48** L48	L36 t4.5				
7												
6N					L48 t8		L36 **L36**		**L24 t4**			
5												
4				**L36 t9**			**L24 L24**			**L12 t3**		
3												
2	**L24 t12**						**L12**					
1												

Fig.2. At the bottom there is a part repeated from **Fig.1**: the mode of circulation with normal values $t_N = 6$ and $P_N = 6$ (starting cell $6_p X 6_t$) which reacts to standard scale of variations of resistance. The upper part of the picture shows the mode of circulation with tachy-rhythm as normal (normal duration of ventricular diastole $t_N = 3$) and $P_N = 15$ (starting cell $15_p X 3_t$); the reaction to standard scale of variations of $\sum R$ (arrows are omitted) is the following: diapason of venous pressures is broadened and shifted towards high values, diapason of rhythms (durations of ventricular diastole) is narrowed.

Fig.3 presents the case with normal rhythm which is slower; the value of normal duration of diastole is $t_N = 9$, i.e. it is longer then $t_N = 6$ (norm-mode) and much longer then $t_N = 3$ (tachi-mode) and, respectively, this mode of circulation can be called – the brady-mode). New equilibrium, - while normal resistance is conserved ($R_N = 30$), - results in change of value of normal venous pressure and value of normal end-diastolic volume: $P_N = 3.75$ and $L = 33.75$, respectively. Configuration of the analogous symmetrical pair of hyperbolic graphs is flattened comparing to the configuration shown in the picture above (example with $t_N = 6$); it means that diapason of working rhythms is broader but diapason of working values of venous pressure is narrower and the latter is located at the low-pressure region. Therefore, this mode of circulation (where bradycardia was chosen as normal rhythm) is energetically favorable because such circulation can overcome standard growth of resistance exploiting low pressures.

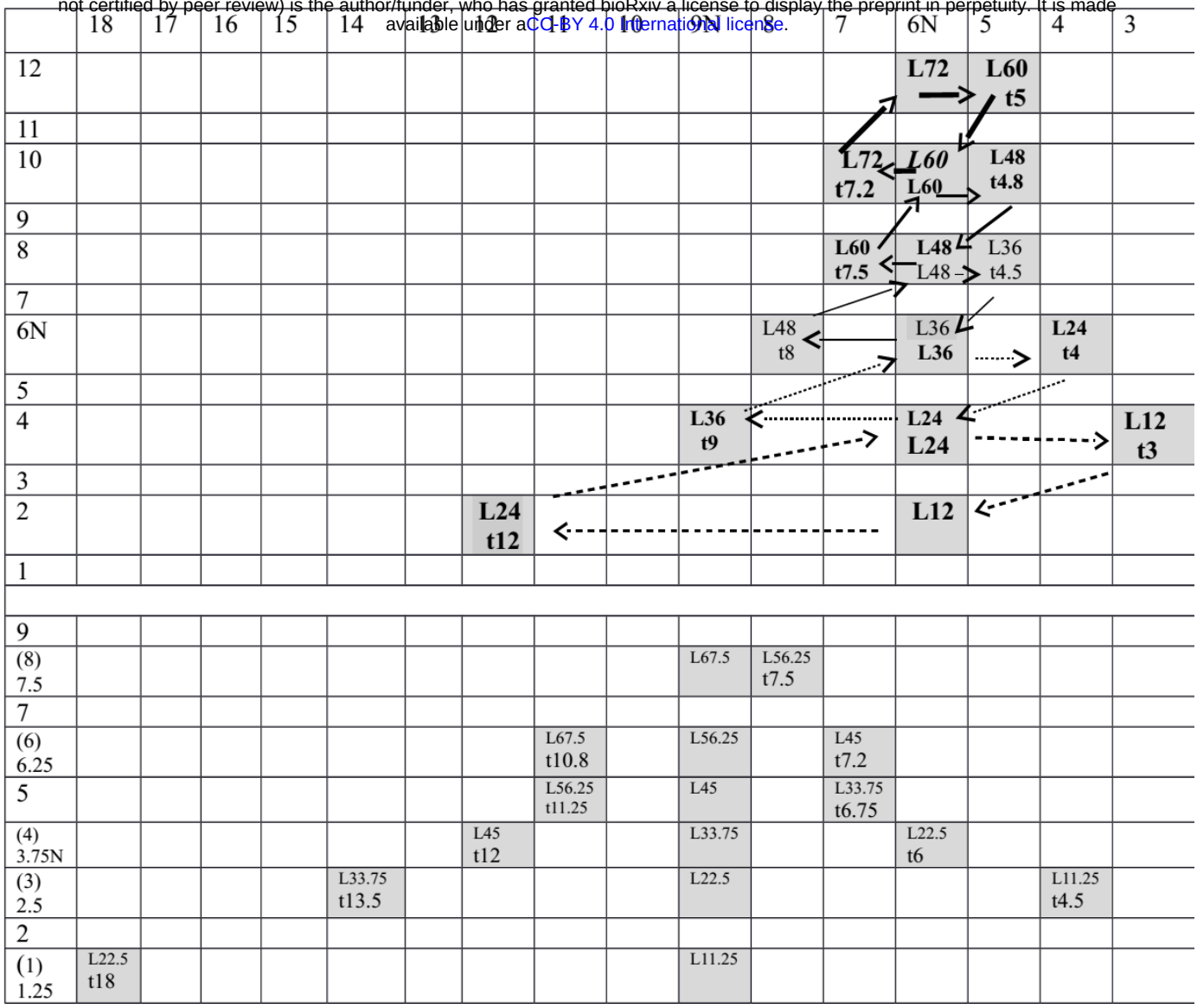


Fig.3. The picture is analogous to **Fig.2:** here the example for comparison from **Fig.1** is situated at the upper part of the picture; the mode of circulation with brady-rhythm as normal is placed at the bottom: $t_N = 9$, $P_N = 3.75$ (starting cell $3.75_p X 9_t$); the reaction to standard scale of variations of $\sum R$ (arrows are omitted) is the following: diapason of venous pressures is narrowed and shifted towards low values, diapason of rhythms (durations of ventricular diastole) is broadened.

Thus, let us have a look at formula {3} again. At first we have varied parameter $\sum R$ and have obtained two symmetrical hyperbolic curves as the response to the deviation of the system; at that case t_N and Q were considered constant. Coefficients M and k are considered invariable at this model. M is a coefficient of dynamic viscosity of relaxing myocardium; M lacks variability until the description of some mechanism which prevents rupture of extremely dilated ventricle improves the model; at present model such mechanism is omitted. Coefficient k is assumed equal to 1 (as simplification) and it defines the linearity that exists, firstly, between end-diastolic volume and systolic volume (Frank-Starling's law) and, secondly, between systolic volume and systolic pressure – the latter bond, we repeat, is taken for granted

now as numerical equalization of these two parameters *(the nature and approximate linearity of this bond will be scrutinized at the next publication)*; therefore, we may speak about invariability of k . The next step includes variation of the parameter t_N – but simultaneously the parameter ∑R repeats its previous varying, - and, as the result, we have found two different modes of circulation which contrast significantly while overcoming standard stepwise changes of resistance. The only one parameter from the compound

factor $K=\frac{Q\cdot\Sigma R\cdot t_N}{\frac{k}{M}t_N-1}$ that has not been undergone to procedure of perturbation is the volume flow rate, Q. Previously we considered Q constant and equal to 1 keeping in mind that stability of cell metabolism provides the feedback for maintaining stability of Q; we are not going to specify this feedback but imaginary intensification of metabolism, - due to physical exertions, for instance, - must force the volume flow rate of blood. So it is quite reasonable to raise the value of Q and trace the changes ensuing; for this aim Q will be intensified in the form of one-step forced value of Q. New value of Q will be applied to the two different modes of circulation (which have been revealed while varying the values of normal heart rate). The new value of Q was chosen 1.2 instead of 1 for the convenience of visual proof only. We may remind that the information about intensification of volume flow rate is a calculable value: when the data coming from carotid baroreceptors does not coincide with data coming from muscles of resistive arteries the value of volume flow rate differs from 1. As far as $P_{diast}=Q\cdot\Sigma R$, - and P_{diast} is the information taken from carotid baroreceptors and ΣR is the information taken from muscles of resistive arteries, - the equation

$Q=\frac{P_{diast}}{\Sigma R}=1$ determines the case when two sources give identical information; if the fracture is equal to 1.2, for instance, it means that the discrepancy was caused by the command of some upper level which deliberately overstate P_{diast} and that the command has a goal to force the volume flow rate.

Final part of the article contains two examples of double deviation; firstly, we have chosen most contrast modes of circulation (tachy-mode and brady-mode); secondly, we have forced the volume flow rate and, thirdly, we have undergone the both modes of circulation (with forced Q) to three-stepped elevation and two-stepped decline of the resistance; we excluded the returns from the steps to its starting positions, - as it was carried out when we have varied only the resistance (**Fig.1**, **Fig.2** and **Fig.3**), - because it would overload the procedure.

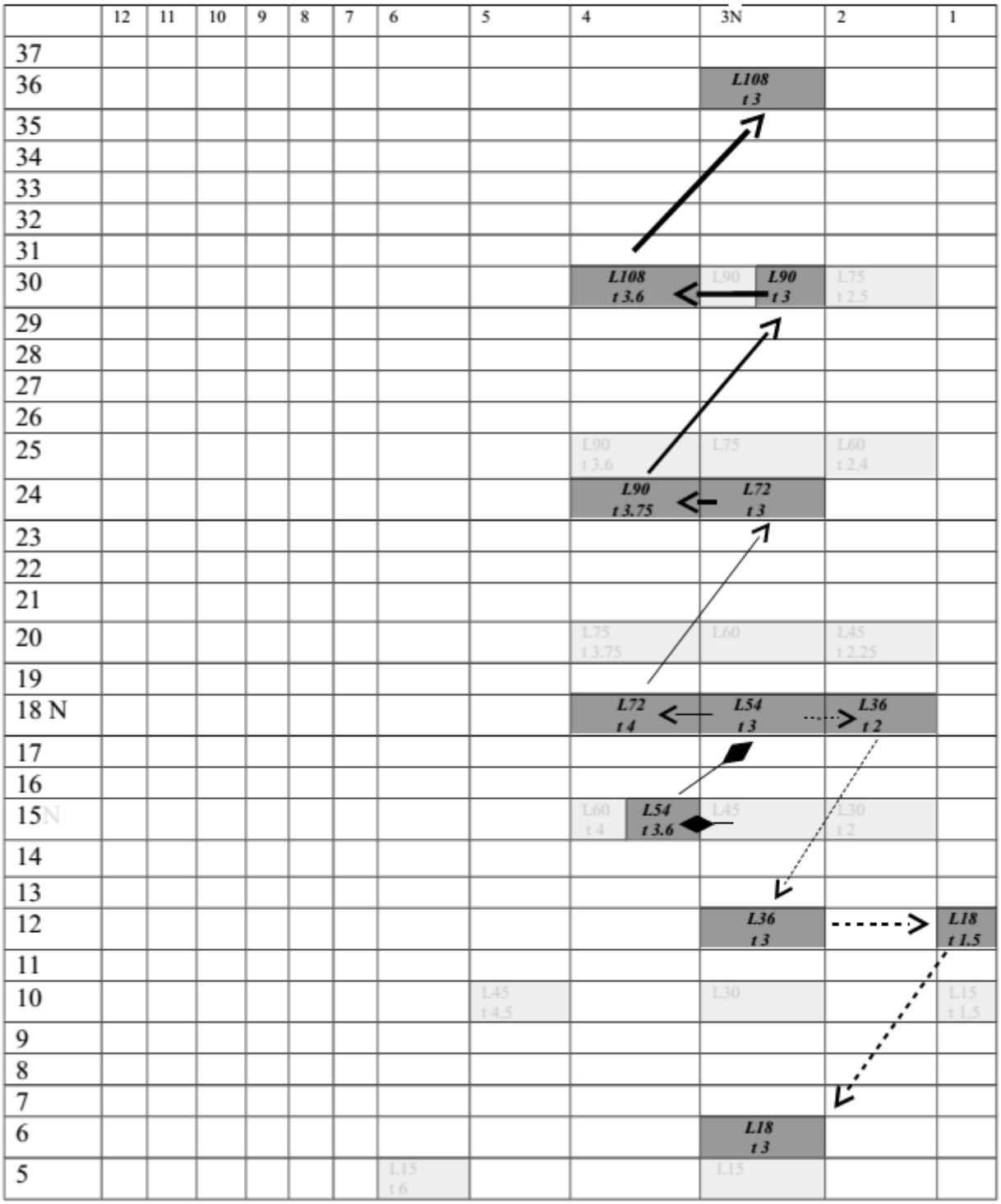


Fig.4. The background is the tachy-mode of circulation taken from **Fig.2**. The primary variation is the forcing of volume flow rate; starting cell 15_pX3_t which characterizes circulation at R_N and Q = 1 passes on to the cell 18_pX3_t which balances circulation at R_N and Q = 1.2 (rhombic arrows). Next variation is the elevation and decline of resistance; first step of elevation of the resistance ($\Delta R = 10$, $\sum R = 40$) results in balancing of circulation at 24_pX3_t (thin arrows). Three steps of elevation and two steps of decline of the resistance is represented.

Fig.4 shows the pattern with rapid rhythm taken from **Fig.2**; the point for the intrusion is equilibrium based on normal (for tachy-mode) duration of diastole and normal (for tachy-mode) venous pressure – i.e. acting cell 15_pX3_t ($t_N = 3$, $P_i = P_N = 15$ when $\sum R = R_N = 30$ and $Q = 1$). The new equilibrium will not affect $R_N = 30$ for the beginning but will only force volume flow rate ($Q = 1.2$ instead of $Q = 1$); so we are to find the values of t_N and P_N for new equilibrium. First of all let us calculate the new value of duration of diastole that will appear after the increasing of Q; it can be calculated as t_{shift} (venous pressure remains the same but duration of diastole will be changed) and realize the calculation by substitution of all the parameters of acting cell 15_pX3_t into formula {3} with the excepting of $Q = 1$ which now is $Q = 1.2$:

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} = \frac{1.2 \cdot 30 \cdot 3}{15\left(\frac{1}{1} 3 - 1\right)} = 3.6 = t_{shift}$$

Till now we have considered that information from carotid baroreceptors, – but not from muscles of resistive arteries, - was decisive, and may be it was partly true when $Q = 1$. Moreover, now when the resistance is not changed ($\sum R = R_N = 30$, i.e. the information from muscles is not changed) the command to reorganize circulation can be originated from carotid baroreceptors only; evidently that it were not baroreceptors but "calculative center" which introduces the false information as if P_{diast} is already elevated (and, consequently, as if Q is already forced). The command results in calculation of t_{shift} and the prolonged diastole enlarges L and, hence, P_{syst} will be increased, - being numerically identical to L, - and, consequently, P_{diast} will be observed elevated because the resistance was not changed, - i.e. Q becomes forced the way it was planned by "calculative center".

Thus, after multiplication $t_{shift} = 3.6$ by $P_N = 15$ we get the value of end-diastolic deformation $L = 54$; now acting cell is $15_pX3.6_t$ (rhombic arrow directed to the left). Previously, when diastolic pressure numerically coincided with the resistance ($Q = 1$), we might subtract $R_N = 30$ from $L = 54$ and get the value of residual pressure (venous pressure) $P_i = 24$; but now $Q = 1.2$ and, consequently, $P_{diast} = Q \bullet \Sigma R = 1.2 \bullet 30 = 36$; hence, we subtract 36 from 54 and get $P_i = 18$. Bainbridge's reflex detects the elevation of venous pressure and begins to calculate by means of {3} the normal duration of the diastole, – i.e. Bainbridge's reflex restores normal rhythm $t_N = 3$.

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} = \frac{1.2 \cdot 30 \cdot 3}{18\left(\frac{1}{1} 3 - 1\right)} = 3 = t_N$$

Now the working cell is 18_pX3_t (rhombic arrow - upwards and to the right) and this cell presents the balanced circulation with forced volume flow rate $Q = 1.2$ and normal resistance $R_N = 30$; the indexes of the cell show that the normal (for tachy-mode) duration of diastole is the same but the level of venous pressure, - which is now considered normal, - is a new level ($P_N = 18$), and that it is higher comparing to the level of

venous pressure of the initial tachy-mode of circulation ($P_N = 15$). Therefore, this cell is the new equilibrium which denotes a new starting point for further elevation (or decline) of the resistance.

And now it is time to raise the resistance: $\Delta R = 10$, $\sum R = 40$. The information from muscles of resistive arteries about the elevation of $\sum R$, - conducted along the pathway in parallel with the arc of carotid reflex, - resulted in deceleration of rhythm, i.e. this reflex is also programmed, like carotid reflex and Bainbridge's reflex, that $t_N = 3$:

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} = \frac{1.2 \cdot 40 \cdot 3}{18\left(\frac{1}{1} 3 - 1\right)} = 4 = t_{shift} \quad .$$

Hence, acting cell is $18_p X 4_t$ (horizontal thin arrow directed to the left); end-diastolic deformation is now $L = 18 \bullet 4 = 72$ and, therefore, acting cell is $18_p X 4_t$ (horizontal thin arrow directed to the left). $P_{diast} = Q \bullet \Sigma R = 1.2 \bullet 40 = 48$; then we subtract 48 from 72 and get $P_i = 24$. As far as venous pressure is detected (by Bainbridge's reflex) elevated, - after the retardation of rhythm, - it means that equipoise is imbalanced again and Bainbridge's reflex is going to restore $t_N = 3$ by means of accelerating rhythm and, consequently, calculates:

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} = \frac{1.2 \cdot 40 \cdot 3}{24\left(\frac{1}{1} 3 - 1\right)} = 3 = t_N \quad .$$

Now acting cell is $24_p X 3_t$ (thin arrow directed upwards and to the right) and we may check up that it is a new equilibrium of circulation (with forced volume flow rate and elevated resistance): $L = 24 \bullet 3 = 72$; $P_i = 72 - 40 \bullet 1.2 = 24$.

Further reorganizations of circulation as the responses to step by step elevation (increment $\Delta R = 10$) or decline (decrement $\Delta R = -10$) of the resistance, - for tachy-mode of circulation, - can be easily calculated and traced with the help of **Fig.4**.

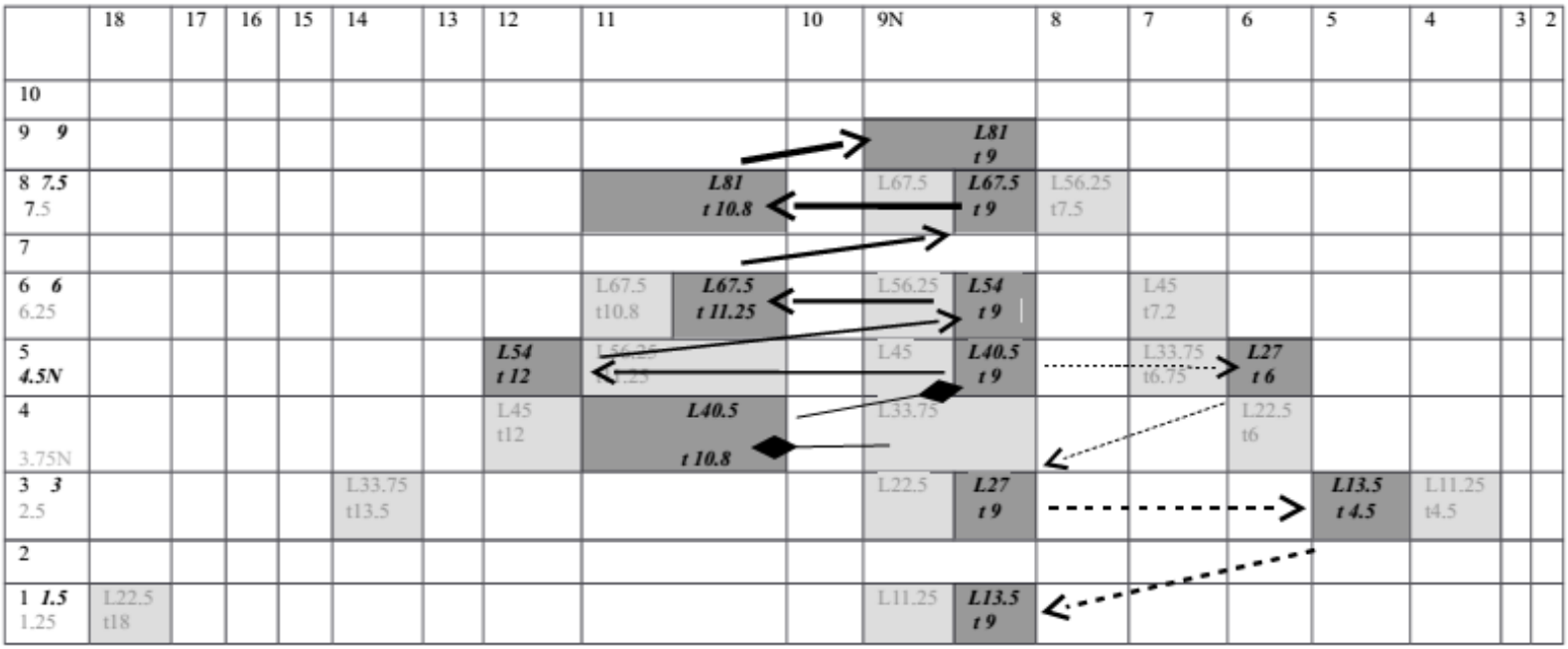


Fig.5. The background is the brady-mode of circulation taken from **Fig.3**. The primary variation is the forcing of volume flow rate; starting cell 3.75_pX9_t which characterizes circulation at R_N and Q = 1 passes on to the starting cell 4.5_pX9_t which balances circulation at R_N and Q = 1.2 (rhombic arrows). Next variation is the one-step elevation of resistance ($\Delta R = 10$, $\sum R = 40$) which results in balancing of circulation at 6_pX9_t (thin arrows). Three steps of elevation and two steps of decline of the resistance is represented.

Fig.5 visually describes the analogous forcing of volume flow rate and further rise and drop of the resistance – but variations are applied to the mode of circulation with slow rhythms (the pattern was taken from **Fig.3**). The starting point is the equilibrium with normal (for brady-mode) duration of ventricular diastole and normal (for brady-mode) venous pressure, - i.e. the acting cell is 3.75_pX9_t ($t_N = 9$, $P_i = P_N = 3.75$ when $\sum R = R_N = 30$ and Q = 1). Carotid reflex (or better to say - "the calculative center") organizes the discrepancy between the data concerning P_{diast} and $\sum R$ and inserts Q = 1.2 in formula {3}:

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} = \frac{1.2 \cdot 30 \cdot 9}{3.75\left(\frac{1}{1} 9 - 1\right)} = 10.8 = t_{shift}$$

Now acting cell is $3.75_pX10.8_t$ (rhombic arrow directed to the left). Multiplication of $t_{shift} = 10.8$ by $P_N = 3.75$ results in the value of end-diastolic deformation L = 40.5 which we numerically equate with systolic pressure; then it is necessary to find the value of P_{diast} which now differs from $R_N = 30$ due to the fact that Q = 1.2; consequently, $P_{diast} = Q \bullet \Sigma R = 1.2 \bullet 30 = 36$, and then we subtract $P_{diast} = 36$ from the value of systolic pressure numerically equal to L = 40.5 and get the value of residual pressure (venous pressure) $P_i = 4.5$. Bainbridge's reflex detects the elevation of venous pressure and begins to calculate by means of {3} the normal duration of the diastole, $t_N = 9$ (restoration of normal rhythm):

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i(\frac{k}{M}t_N-1)}=\frac{1.2\cdot 30\cdot 9}{4.5(\frac{1}{1}9-1)}=9=t_N \quad .$$

The acting cell now is 4.5_pX9_t (rhombic arrow - upwards and to the right); this cell presents the balanced circulation with forced volume flow rate Q = 1.2 and normal resistance $R_N = 30$; the indexes of the cell show that the normal (for brady-mode) duration of diastole is the same but the level of venous pressure, - which is now considered normal, - is a new level ($P_N = 18$), and that it is higher comparing to the level of venous pressure of the initial brady-mode of circulation ($P_N = 15$). Therefore, this cell is the new equilibrium which denotes a new starting point for further elevation (or decline) of the resistance. .

Now we are going to superimpose the minimal rise of resistance ($\Delta R = 10$, $\sum R = 40$) upon brady-mode of circulation with forced volume flow rate. The information from muscles of the resistive arteries about the elevation of $\sum R$, - conducted along the pathway in parallel with the arc of carotid reflex, - resulted in deceleration of rhythm, i.e. this reflex is also programmed, like carotid reflex and Bainbridge's reflex, that $t_N = 9$:

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i(\frac{k}{M}t_N-1)}=\frac{1.2\cdot 40\cdot 9}{4.5(\frac{1}{1}9-1)}=12=t_{shift} \quad .$$

The acting cell now is 4.5_pX12_t (horizontal thin arrow directed to the left). The end-diastolic deformation is L = 4.5 • 9 = 54 and it is numerically equal to systolic pressure; $P_{diast} = Q \bullet \Sigma R = 1.2 \bullet 40 = 48$; then we subtract 48 from 54 and get $P_i = 6$ and Bainbridge's reflex succeeded in restoration of $t_N = 9$ by means of calculation:

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i(\frac{k}{M}t_N-1)}=\frac{1.2\cdot 40\cdot 9}{6(\frac{1}{1}9-1)}=9=t_N \quad .$$

Now acting cell is 6_pX9_t (thin arrow directed upwards and to the right) and we may check up that it is a new equilibrium of circulation (with forced volume flow rate and elevated resistance): L = 6 • 9 = 54; P_i = 54 – 40 • 1.2 = 6.

Further reorganizations of circulation as the responses to step by step elevation (increment $\Delta R = 10$) or decline (decrement $\Delta R = -10$) of the resistance, - for brady-mode of circulation, - can be easily calculated and traced with the help of **Fig.5**.

What difference is evident when comparing tachy-mode (**Fig.4**) and brady-mode (**Fig.5**) of circulation - although both are undergone to standard forcing of Q and further elevation of the resistance? Tachy-mode starts with rapid rhythm ($t_N = 3$) and high level of venous pressure ($P_N = 15$); tachy-mode reacts upon the intensification of regime of circulation by means of significant rising of venous pressure ($P_{max} – P_N = 36 – 15 = 21$) and insignificant amplitude of fluctuation of rhythm (deceleration: $t_{max} - t_N = 4 – 3 = 1$). Brady-mode has another starting point - slow rhythm (t_N =9) and low venous pressures (P_N = 3.75); brady-

mode reacts with significant amplitude of fluctuation of rhythm (deceleration: t_{max} - t_N = 12 – 9 = 3) and moderate rising of venous pressure (P_{max} - P_N = 3.75 – 9 = 5.25). Tachy-mode demonstrates 4-fold higher elevated venous pressure comparing to the elevation of venous pressure of brady-mode (21 : 5.25 = 4); brady-mode demonstrates 3-fold wider amplitude of fluctuation of rhythm comparing to the amplitude of fluctuation of rhythm demonstrated by tachy-mode (3 : 1 = 3). It means that tachy-mode has difficulties with venous pressure and brady-mode has difficulties with rhythm. What is it easier to overcome? Let us analyse what are these difficulties in the flesh. Firstly, we may add to analysis the values of systolic pressures of tachy-mode (P_{max} - P_N = 108 – 45 = 63) and brady-mode (P_{max} - P_N = 81 – 33.75 = 47.25) as far as the value of end-diastolic volume L was assumed to be numerically equal to systolic pressure. Hydrodynamics with higher pressures spends more energy and, consequently, tachy-mode is not energetically favorable. As regards to brady-mode – we have no criterion what diapason of retardation (or acceleration) of rhythm is appropriate to the whole mechanism of cardiac relaxation and contraction. Imagine that such a criterion exists (and it is obviously exists in terms of physiology) and, consequently, the choice of brady-mode is evident. However, the above criterion mostly relates not to the value of postulated (fixed) normal rhythm for brady-mode but to the consequence – i.e. to maximal value of diapason of deceleration. In other words, bradicardia is restricted by amplitude of diapason of retardation but not of acceleration – although diapason of retardation of rhythm is symmetrical with the diapason of acceleration.

And how can the choice of brady- or tachy-mode be implemented? It is based on programming of the value of normal rhythm (normal duration of ventricular diastole) which must be substituted to the equation {3}. So, firstly, we ought to repeat that normal rhythm exists as some postulated (programmed) constant and, secondly, we may conclude that such normal rhythm must be located much closer to brady working rhythms than to tachy working rhythms.

We may apply this knowledge (about different ranges of working rhythms situated at both sides of some normal value of heart rate) to the phenomenon which lacks explanation within any modern theory of circulation (so far as we know). It is just the observing phenomenon that the normal pulse, – as one understands it while measuring it at some comfort and calm conditions, - is located asymmetrically between the range of rhythms which circulatory system is used for deceleration and the range of rhythms which circulatory system is used for acceleration; such normality is much closer to bradycardia then to tachycardia and the range of working brady-rhythms is much narrower than the range of working tachy-rhythms. If we admit the coincidence of the conclusion with the real phenomenon we must admit the whole chain of testimony including the main statement the testimony has been proceeded from. The main statement is that relaxing ventricle imitates viscous deformation (deformation that has two independent variables, stress and time) and this statement has permitted to deduce the equation which successfully balances one-loop circulation.

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