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

Author: Yuri Kamnev (Article of one author)

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.

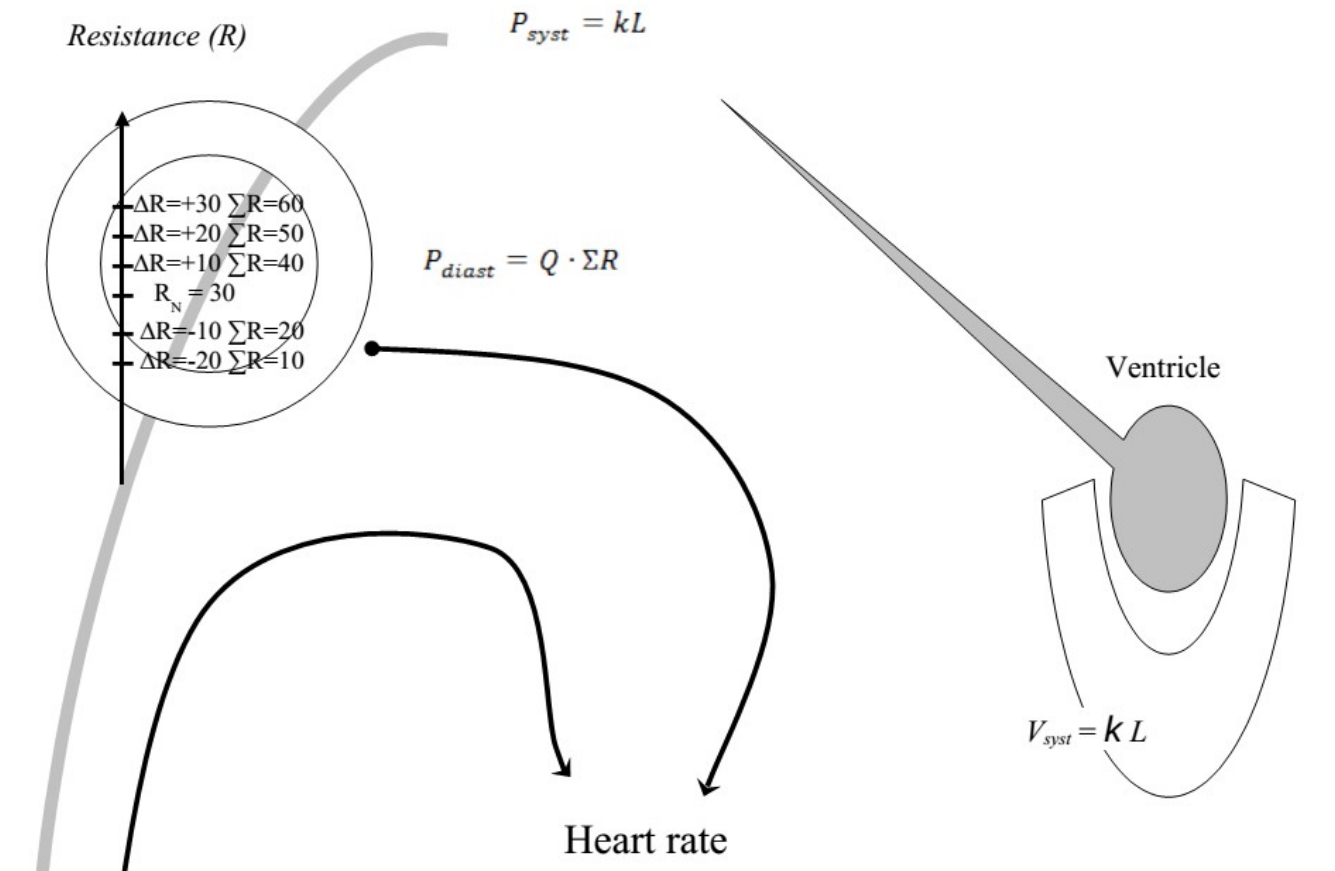
Introduction

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 to 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 fioor 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 conceived 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 which concedes a right for control. 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 $\left[m^3\right]$ is a deformation of the volume, P is a pressure (stress) and M is a coefficient of internal friction (or coefficient of dynamic viscosity) with dimensions of quantity $\left[\frac{n\cdot s}{m^5}\right]$. Hence $L=\frac{1}{M}Pt$ is the formula simplified for arithmetic calculations. 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 is the coefficient of dynamic viscosity $\left[\frac{n\cdot s}{m^5}\right]$ which can be approximated to the viscosity of water (M = 1 $\left[\frac{n\cdot s}{m^2}\right]$ at temperature = 20° C) at present model. 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. Therefore, 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 venuous 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 converts end-diastolic volume into systolic volume which can be considered directly proportional 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, $\sum R$ 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 $\sum R$ is shown; different values of $\sum R$ influence 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 $\sum R$.

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 pressure at the initial part of arterial pipe, - the part which is located between the ventricle and arterial resistive vessels and is presented by elastic artery with constant resistance (the wall of such artery is approximated to the firm wall), - can be defined as product of volume flow rate and the resistance. The resistance is constant and, consequently, the pressure at this part of arterial pipe is directly proportional to volume flow rate. Any systolic volume which participates at Frank-Starling's law is being ejected from the ventricle during the identical time interval and, consequently, the range of volume flow rates (different systolic volumes per identical time interval) is directly proportional to the range of end-diastolic volumes which participate at Frank-Starling's law. Therefore, the pressure that is originated at the initial part of arterial pipe, - and which we may call systolic pressure, - depends on end-diastolic volume via direct proportion: $P_{syst} = k\,L$, where k is a coefficient of proportionality with dimensions of quantity $\left[\frac{n}{m^5}\right]$; consequently, P_{syst} can be substituted instead of V_{syst} but coefficient of proportionality ought to be changed; hence, k must be substituted instead of k ; furthermore, k will be assumed equal to 1 at present model in order to simplify arithmetic calculations.

We have just connected deformation, - i.e. end-diastolic volume, L, - and pressure, - i.e. pressure at the moment, or period, of ejection (systolic pressure), - but now it is pertinent to simulate pulsatile pattern of circulation. Pulsatile flow comprises a lot of characteristics but at that model we need to trace the only one

parameter, - pressure. Firstly, let us primitively simulate pulsatile regime by means of introduction of cardiac cycle; the simplest way is to introduce the beginning and the end; in other words, it is necessary to introduce pairs of time intervals and then connect two different values of pressure with the early and the late parts of cardiac cycle. Secondly, let us get rid of pulsatile flow in veins by means of introduction of damper at the end of arterial portion of pipe (capillary damper) and let us neglect its resistance because we may consider such a resistance constant; consequently, the pressure which we connect with the flow after the damper, - i.e. at the venous portion of the pipe, - lacks pulsatile behavior because the division by pairs is liquidated. Therefore, we may determine three values of pressure: the two values are connected with two periods of cardiac cycle at the arterial portion of the pipe, and the one value is not connected to any of two periods of cardiac cycle because the value relates to the venous portion of the pipe.

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: $R = \frac{P_1 - P_2}{Q}$. Let us associate the pressure at the beginning of the pipe with the pressure connected to the early period of cardiac cycle and this pressure will correspond to systolic pressure, $P_{syst} = k\,L$; the pressure at the end of the pipe let us associate with pressure that has no connection with periods of cardiac cycle and this pressure relates to the venous portion of the pipe, i.e. it is a venous pressure, P. Hydrodynamic resistance, R, is equated to the total peripheral resistance $\sum R$ $\left[\frac{n \cdot s}{m^5}\right]$ which is variable due to the work of arterial resistive vessels. It is relevant to repeat that we neglect the non-variable part of total peripheral resistance, - resistance of capillary basin, which is represented by capillary damper, - and constant resistance of elastic artery (between the ventricle and arterial resistive vessels). Hence, we call $\sum R$ "the resistance" keeping in mind that the resistance has its normal value, R_N, and has the scope of variability. Therefore, $P_{syst} - P = Q \cdot \Sigma R$, 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 – the period which exists at the arterial portion of the pipe; in other words, this pressure is a diastolic pressure: $P_{diast} = Q \cdot \Sigma R$.

Even now 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: $P_{syst} - P_{diast} = P$. Besides, the subtrahend consists of two components ($\sum R$ 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 artery) and if we also possess 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, $\sum R$ is numerically equal to diastolic pressure $P_{diast}=Q\cdot\Sigma R=1\cdot\Sigma R$ $\left[\frac{n}{m^2}\right]\cdot$ and, consequently, diastolic pressure indicates two situations: the normal resistance is maintained $\Sigma R=R_N$ or it is changed as far as $\Sigma R=R_N+n\cdot\Delta R$, where n is the number of steps of elevation or drop of resistance, i.e. ΔR can be positive (elevation) or negative (drop).

The quanta of columns and lines of the table at **Fig.1** were arbitrary chosen but it was convenient to frame 12×12-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 middles 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 numerical difference between R_N (formally multiplied by Q = const = 1) and normal ventricular end-diastolic deformation (which will further be converted into systolic pressure) equal to 6. Normal end-diastolic deformation is the product of normal venous pressure and normal duration of ventricular diastole, and taking into account that M is equal to 1 and k is equal to 1 either, we get $\frac{1}{1}(6\times 6)=36$; after the deduction of 30 (since Q = 1 and hence $P_{diast}=Q\cdot R_N=1\cdot 30=30$) 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 was found and this equilibrium characterizes the normal, non-deviated mode of circulation, - i.e. one-loop circulation that we are going to scrutinize by means of the model.

Two reflex arcs carry out operations 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}, i.e. this reflex measures pressure inside arterial pipe at the end of cardiac cycle (at the second period of cardiac cycle consisting of two periods), - although we have mentioned that in general case the direct information from the muscles of resistive arteries is indispensable; 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 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 the 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 and variations of systolic pressure. In other words, the degree of inertness of converting of arterial pressure (combination of systolic pressure and the resistance, i.e. diastolic pressure) into venous pressure, - inertness generated 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 exceed the elevated resistance, $\sum R = 40$. 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 the only

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:

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

L_1 we considered 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,

$$\frac{1}{M}P_i\times t_d=\frac{1}{M}P_n\times t_{na} \ , \qquad \{1\}$$

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

The value of deformation L_2 produces systolic pressure which overcomes elevated resistance $\sum R$:

$$kL_2-Q\cdot\Sigma R=P_n \quad \text{, as far as} \quad P_{syst}=kL \quad \text{and} \quad P_{diast}=Q\cdot\Sigma R \ .$$

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

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

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, $\sum R$, (producing deviation) and venous pressure, P, - conserve their changed levels. It is clear that $\sum R$ 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 to 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 - in order to achieve the change of end-diastolic deformation by means of venous pressure only). 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 heightening 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 getting information directly from the muscles of arterial resistive vessels, $\sum R$, - the pair which is necessary for calculation of volume flow rate, - this problem will be scrutinized below) and, secondly, solves the equation {3}, substituting $t_N = 6$, $\sum R = 40$ and $P_i = P_N = 6$ into {3}; hence, carotid reflex calculates the value of elongated diastole (i.e. decelerated rhythm):

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

Let us complete tracing the whole pathway from the command of rising of the resistance to the command of declining of it in order to return the system to the initial condition – normal resistance, normal venous pressure and normal rhythm (following the directions of the most thin indissoluble arrows at the table on **Fig.1**). Thus, decelerated rhythm was found ($t_{ch} = 8$) and it must be multiplied by the value of the normal venous pressure $P_i = P_N = 6$ taking into consideration that $\frac{1}{M}=1$ at formula $L=\frac{1}{M}Pt$; the

product (i.e. end-diastolic deformation $L = 48$) generates $P_{syst} = 48$ ($k = 1$ at $P_{syst} = k\,L$) which overcomes the elevated resistance $\sum R = 40$ ($Q = 1$ in formula $P = P_{syst} - Q \cdot \Sigma R$) and the residual pressure (venous pressure) becomes 48 – 40 = 8, i.e. venous pressure becomes equal to 8 (new $P_i = 8$). Horizontal arrow points to the left: the initial acting cell 6_px6_t transforms into 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 will be in need to picture the calculated values of *t* and P_i at the cells with whole indexes $_t$ and $_p$ which are closest 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:

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

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: the arrow points upwards and to the right – i.e. from the cell 6_px8_t 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 (P_{diast}, respectively) is increased 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: the imbalance (elevated resistance) makes the system to react immediately and restore equilibrium by changing of the point of balance ($t_N = 6$ was changed to $t_{ch} = 8$) but then the counterbalance appears (elevated venous pressure) and it compels the system to restore the previous point of balance ($t_N = 6$).

The way backwards begins from the command to decrease the resistance $\Delta R = -10$; obviously the resistance will drop from $\sum R = 40$ to $R_N = 30$. Baroreceptors of carotid reflex detect the falling of P_{diast} and begin to solve the equation {3} in order to find t_{ch} which, for the present instance, denotes the acceleration of rhythm; the value of P_i that must be substituted to {3} is equal to 8 and calculated value of the shortened ventricular diastole (accelerated rhythm) is $t_{ch} = 4.5$; so the acting cell of the table is now 8_px4.5_t (located inside the cell 8_pX5_t at **Fig.1**): horizontal arrow points to the right from the previous acting cell 8_px6_t. The product, i.e. end-diastolic deformation, is $L = 8 \times 4.5 = 36$ (for $\frac{1}{M} = 1$ at formula $L = \frac{1}{M} Pt$). Diminished deformation generates the diminished systolic pressure $P_{syst} = kL = 1 \cdot 36 = 36$ which overcomes $R_N = 30$; 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 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. The calculated value of t_{ch} is equal to 6 and it is the value of normal ventricular duration, i.e. $t_{ch} = t_N$; the arrow points downwards and to the left indicating that the acting cell is again the cell $6_p X 6_t$; the value of P_i is now equal to 6, i.e. $P_i = P_N$. Therefore, the initial equilibrium based on normal parameters is restored.

The two observations concerning this primary example of violation of balance and restoration of balance must be mentioned.

- 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? But it is a particular case and, moreover, the particular case comprises the deviations when the certain value of normal rhythm is fixed at some other numerical value but not at $t_N = 6$. The general case will appear when we begin to vary volume flow rate, Q, and in that case the recurrence to normal rhythm will be not so primitive; so the computing work of Bainbridge's reflex will be obvious and consequently information of measured values of venous pressure will be indispensable.

- It is noticeable that both participants of regulation, - carotid reflex and Bainbridge's reflex, - 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 one more, - factually, the last, - independent variable, the volume flow rate, Q. But we have already mentioned that Q can be found quite easily when information from baroreceptors (P_{diast}) is compared to the information from the muscles of resistive arteries (direct information about $\sum R$).

On the whole, we may affirm that the solution (i.e. calculation of t_{ch}), - carried out either by carotid reflex or by Bainbridge's reflex, - concerning the restoration of balance, - 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 declinations 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, 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. As far as K is not able to be varied we exclude the variability of its component, $\sum R$, but instead of it the variability of the resistance is delegated to P_i : standard increments appear at the scale of P_i and, moreover, each value of P_i gains symmetrical value according to its involvement into increasing or decreasing of resistance (P_i must now characterize the direction of deviation), - this can be seen as graphs framed by working cells at the table of **Fig.1** (strictly we must take into consideration only values of t). Formula $t_{ch}=K\cdot\frac{1}{P_i}$ is a formula of inverse proportion and, consequently, we may speak about symmetrical hyperbolic functions of t_{ch} while P_i works like independent variable. Symmetry axis is the line of acting cells $(2, 4\ldots12)_p$X6_t where 6_t is the duration of normal ventricular diastole (normal rhythm), i.e., in terms of the determining of direction of process, symmetry axis denotes the equipoise at each level of P_i 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} or, in other words, into K ; 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.

	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 $t_N = 3$) which is balanced by means of $P_N = 15$ (starting cell $15_p X 3_t$); the reaction to the 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 compressed by the sides.

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 mode of deviating of the system is the same as the initial one (which is shown nearby), 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 normal heart rate maintains very high levels of venous pressure when the resistance is growing.

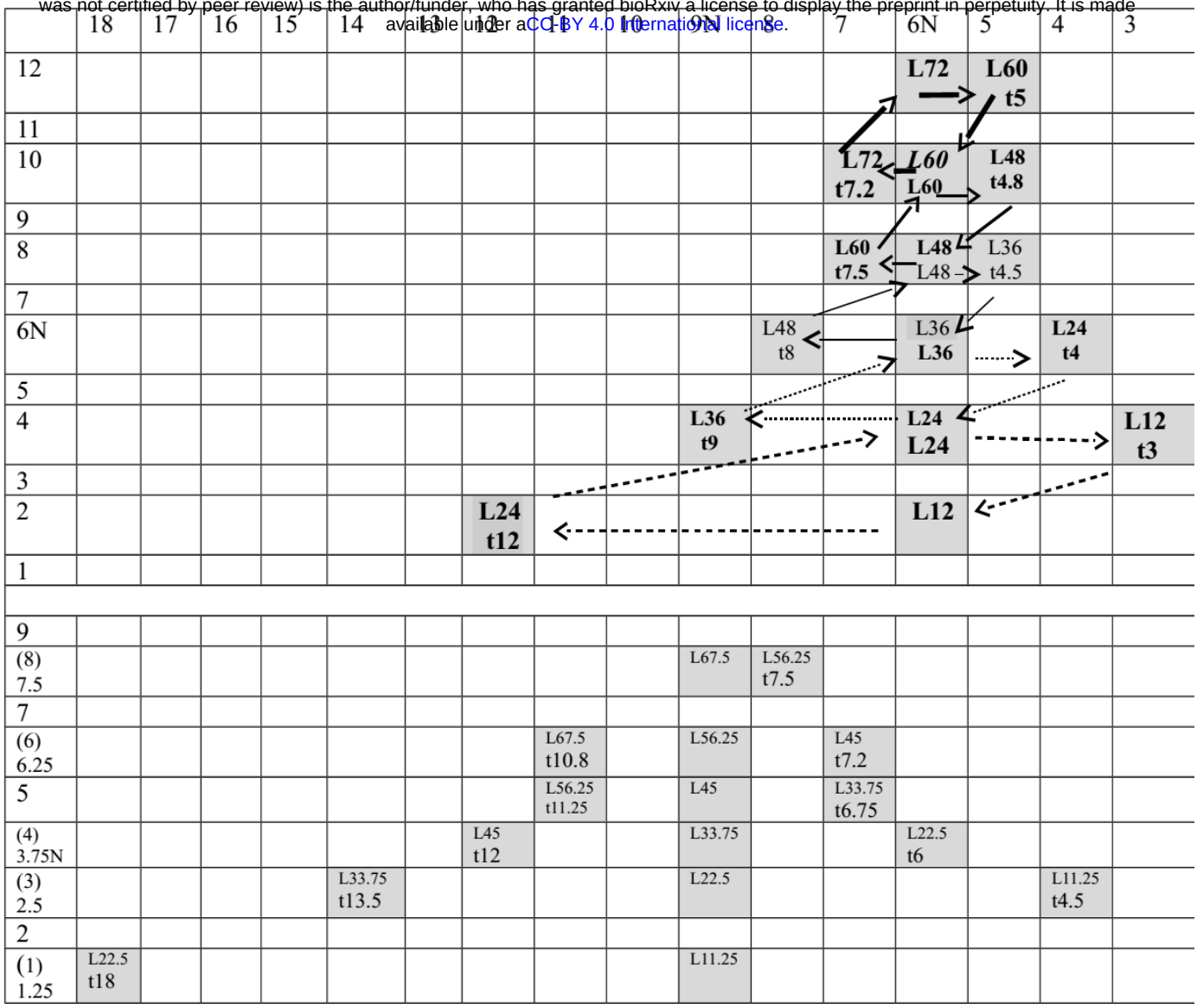


Fig.3. The picture is analogous to **Fig.2:** here the example for comparison from **Fig.1** is situated at the upper part of the picture and at the bottom there is the mode of circulation with brady-rhythm as normal (normal duration of ventricular diastole $t_N = 9$) which is balanced with $P_N = 3.75$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

Fig.3 presents the case with normal rhythm which is slower; the respective value of normal duration of diastole (corresponding to $t_N = 3$ at **Fig.2**) is $t_N = 9$, i.e. the normal duration of ventricular diastole is much longer. 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 it 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.

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 this case t_N and Q are 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 defines rather general linearity that exists, firstly, between end-diastolic volume of the ventricle and contractile force, and, secondly, between contractile force and true systolic pressure; the latter can be approximated to systolic pressure which, in terms of present model, denotes pressure at the beginning of the pipe; therefore, the model is even in need of invariability of k .) The next step includes variation of the parameter t_N – but simultaneously the parameter $\sum R$ 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 the constancy 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. $P_{diast} = Q \cdot \Sigma R$, where P_{diast} is the information taken from carotid baroreceptors and ΣR is the information taken from muscles of resistive arteries; consequently, $Q = \frac{P_{diast}}{\Sigma R} = 1$ is case when two sources give identical information, and if the fracture is equal to 1.2, for instance, it means that the discrepancy was caused by the command of some upper level of regulation and that the command has a goal to force the volume flow rate.

Fig.4 shows the pattern with rapid normal rhythm and high venous pressures taken from **Fig.2**; the point for the intrusion is equilibrium based on normal duration of diastole and normal venous pressure – i.e. acting cell $15_p X3_t$ ($t_N = 3$, $P_i = P_N = 15$ 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 $\boldsymbol{t_N}$ and $\boldsymbol{P_N}$ for new equilibrium. First of all let us calculate the new value of duration of diastole that will appear after the increasing of Q; let us denote it as $\boldsymbol{t_{ch}}$ and realize the calculation by substitution of all the parameters of acting cell $15_p X3_t$ into formula {3} with the excepting of Q = 1 which now is Q = 1.2:

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

In fact, this calculation is done not by us but by carotid reflex as far as information about ΣR and P_{diast} originates from the sources which start the arc of carotid reflex; therefore, carotid reflex has decelerated rhythm in response to forcing of Q.

Thus, after multiplication $\boldsymbol{t_{ch}} = 3.6$ by $P_N = 15$ we get the value of end-diastolic deformation $L = 54$; now acting cell is $15_p X3.6_t$ (lower arrow directed to the left); then we deduce $R_N = 30$ from $L = 54$ and get the value of residual pressure (venous pressure) $P_i = 24$. Bainbridge's reflex detects the elevation of venous pressure and begins to calculate by means of {3} the normal duration of the diastole. This operation can be considered formal at all previous deviations (as it was mentioned above) because Bainbridge's reflex is programmed to restore normal rhythm which value it is aware of, - i.e. the calculation with the help of formula {3} will always result in banal restoration of normal duration of diastole. Now Bainbridge's reflex is also programmed to restore $t_N = 3$ but the result does not lead to restoration of $t_N = 3$:

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} = \frac{1.2 \cdot 30 \cdot 3}{24\left(\frac{1}{1} 3 - 1\right)} = 2.25 = \boldsymbol{t_N}$$

Have we got the rhythm for equilibrium? End-diastolic deformation is the product of $P_i = 24$ and $\boldsymbol{t_N} = 2.25$, i.e. $L = 54$; the deduction of $R_N = 30$ from $L = 54$ results in a value of venous pressure equal to 24; consequently, we deal with equilibrium. As far as the equilibrium is achieved we may denote the venous pressure calculated previously as venous pressure which is normal for circulation with $Q = 1.2$, i.e. $\boldsymbol{P_N} = 24$; the acting cell now becomes $24_p X2.25_t$ (arrow shows to it - upwards and to the right from the previous acting cell) and this cell represents the balanced circulation with forced volume flow rate.

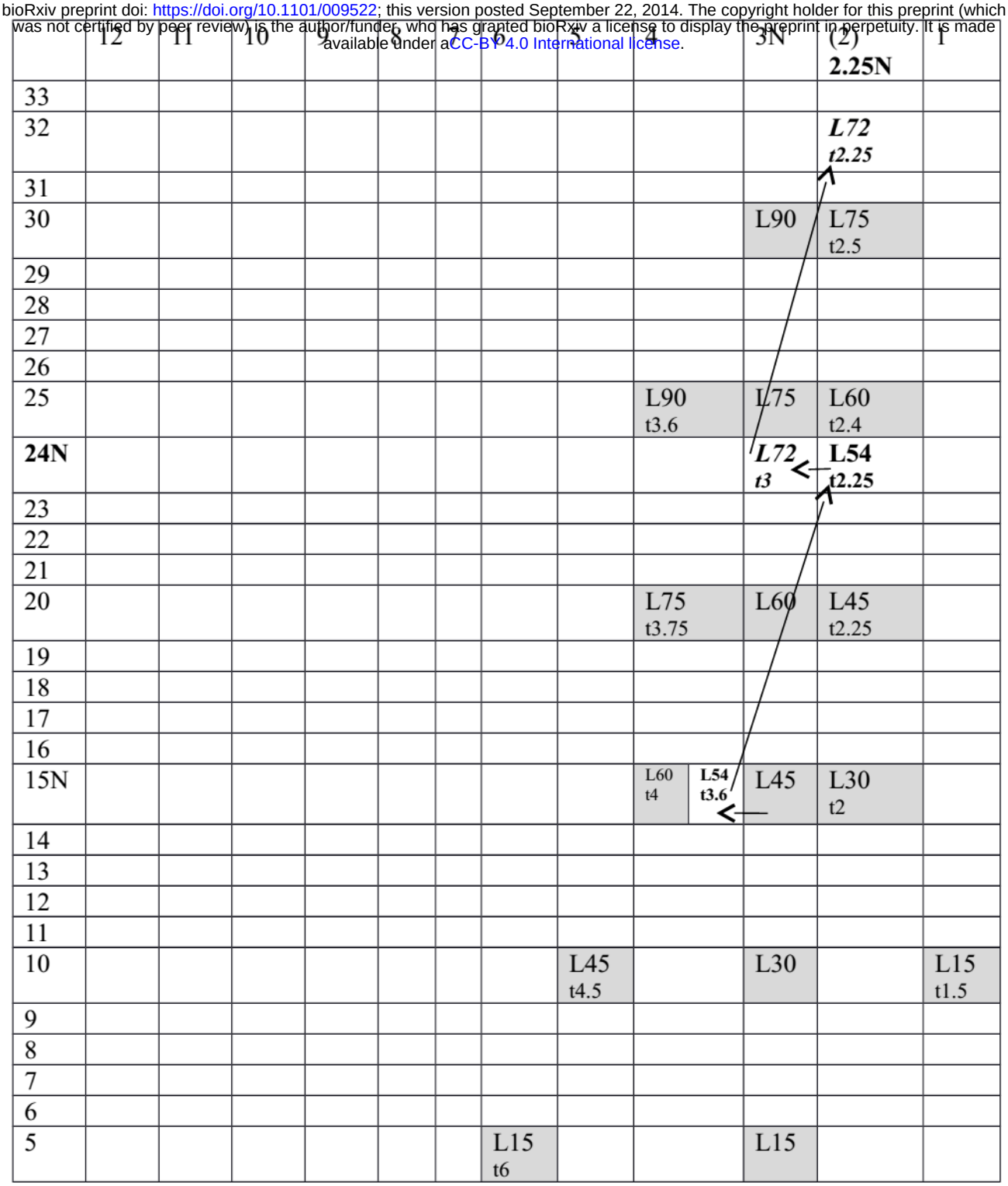


Fig.4. The background is the tachy-mode of circulation taken from **Fig.2**. The primary variation is the forcing of volume flow rate; starting cell $15_p X3_t$ which characterizes circulation at R_N and $Q = 1$ is transformed into the cell $24_p X2.25_t$ which balances circulation at R_N and $Q = 1.2$ and organizes new normal values $\boldsymbol{t_N = 2.25}$, $\boldsymbol{P_N = 24}$ (horizontal arrow directed to the left and then the arrow directed upwards and to the right). Next variation is the one-step elevation of resistance ($\Delta R = 10$, $\sum R = 40$) which results in balancing circulation at $32_p X2.25_t$ (horizontal arrow directed to the left and the next arrow directed upwards and to the right). The system solves the problem

of forcing Q and elevation of $\sum R$ mostly by rising of venous pressure and only slightly by acceleration of rhythm.

Resuming the recent calculations we may notice the dramatic moment: Bainbridge's reflex tried to restore rhythm it had been programmed beforehand, so reflex have sent the command to accelerate rhythm, - i.e. to shorten the diastole from $\boldsymbol{t_{ch}} = 3.6$ down to $t_N = 3$, - but the pace maker shortened diastole down to $\boldsymbol{t_N} = 2.25$; therefore, the heart rate becomes more rapid and, consequently, Bainbridge's reflex is not primitively programmed to restore normal rhythm but Bainbridge's reflex carries out calculations which need gathering the current information. In terms of equilibrium (imbalance, point of balance, counterbalance) such way of changing of the normal rhythm is the changing of point of balance but the method differs from direct variation of t_N as we have carried out before, - here we come across with the case of indirect varying of t_N due to the change of the value of Q.

And now it is time to raise the resistance: $R_N = 30$, $\Delta R = 10$, hence $\sum R = 40$. Carotid reflex detects the increasing of $\sum R$ and decelerates rhythm – being also programmed, like Bainbridge's reflex, that $t_N = 3$. Hence,

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

End-diastolic deformation is $L = 3 \times 24 = 72$, therefore, acting cell is $24_p \times 3_t$ (upper horizontal arrow directed to the left). The residual pressure (venous pressure) is $P_i = 72 - 40 = 32$ and Bainbridge's reflex tries to restore $t_N = 3$ (although the working duration of diastole is already $\mathbf{t_{CH}} = 3$) and calculates:

$$t_{ch} = \frac{Q \cdot \Sigma R \cdot t_N}{P_i\left(\frac{k}{M} t_N - 1\right)} = \frac{1.2 \cdot 40 \cdot 3}{32\left(\frac{1}{1} 3 - 1\right)} = 2.25 = \boldsymbol{t_N} \quad .$$

Now acting cell is $32_p \times 2.25_t$ (arrow directed upwards and to the right from previous acting cell); it is easy to check up that it is a new equilibrium of circulation (with forced volume flow rate and elevated resistance): $L = 2.25 \times 32 = 72$, $P_i = 72 - 40 = 32$. We have seen how the system maintains new normal rhythm ($\boldsymbol{t_N} = 2.25$) calculating it as if normal rhythm is still the essential $t_N = 3$.

Fig.5 visually describes the analogous forcing of volume flow rate and further rise of resistance – but variations are applied to the mode of circulation with slow rhythms and low venous pressures (the pattern was taken from **Fig.3**). The starting point is equilibrium with normal duration of ventricular diastole and normal venous pressure, - i.e. acting cell is $3.75_p \times 9_t$ ($t_N = 9$, $P_i = P_N = 3.75$ when $\sum R = R_N = 30$ and $Q = 1$). After the increasing of Q up to 1.2 the duration of diastole will be changed due to the work of carotid reflex (which finds out the discrepancy between the data concerning P_{diast} and $\sum R$ and inserts $Q = 1.2$ in formula {3}):

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

Multiplication of $\boldsymbol{t_{ch}}$ = 10.8 by P_N = 3.75 results in the value of end-diastolic deformation L = 40.5; now acting cell is $3.75_{\mathrm{p}}\mathrm{X}10.8_{\mathrm{t}}$ (lower arrow directed to the left); then we deduce R_N = 30 from L = 40.5 and get the value of residual pressure (venous pressure) P_i = 10.5. 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):

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i\left(\frac{k}{M}t_N-1\right)}=\frac{1.2\cdot 30\cdot 9}{10.5\left(\frac{1}{1}9-1\right)}\approx 3.86=\boldsymbol{t_N}\quad ,$$

which obviously differs from t_N = 9. End-diastolic deformation is the product of P_i = 10.5 and $\boldsymbol{t_N}$ = 3.86, i.e. L = 40.5; the deduction of R_N = 30 from L = 54 results in a value of venous pressure equal to 10.5; consequently, equilibrium is achieved. That is why we may denote that venous pressure calculated just now is the venous pressure which is normal for circulation with Q = 1.2, i.e. $\boldsymbol{P_N}$ = 10.5; the acting cell now becomes $10.5_{\mathrm{p}}\mathrm{X}3.86_{\mathrm{t}}$ (arrow shows to it - upwards and to the right from the previous acting cell) and this cell represents the balanced circulation with forced volume flow rate.

Now we are going to superimpose the minimal rise of resistance upon circulation with forced volume flow rate: R_N = 30, ΔR = 10, hence $\sum R$ = 40. Carotid reflex detects the increasing of $\sum R$ and decelerates rhythm – being also programmed, like Bainbridge's reflex, that t_N = 9. Hence,

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i\left(\frac{k}{M}t_N-1\right)}=\frac{1.2\cdot 40\cdot 9}{10.5\left(\frac{1}{1}9-1\right)}\approx 5.14=\boldsymbol{t_{CH}}\quad .$$

End-diastolic deformation is L = 5.14 X 10.5 ≈ 54: therefore, acting cell is $10.5_{\mathrm{p}}\mathrm{X}5.14_{\mathrm{t}}$ (upper horizontal arrow directed to the left). The residual pressure (venous pressure) is P_i = 54 – 40 = 14 and Bainbridge's reflex tries to restore t_N = 9 (although the working duration of diastole is already $\mathbf{t_{CH}}$ = 5.14) and calculates:

$$t_{ch}=\frac{Q\cdot\Sigma R\cdot t_N}{P_i\left(\frac{k}{M}t_N-1\right)}=\frac{1.2\cdot 40\cdot 9}{14\left(\frac{1}{1}9-1\right)}\approx 3.86=\boldsymbol{t_N}\quad .$$

Now acting cell is $14_{\mathrm{p}}\mathrm{X}3.86_{\mathrm{t}}$ (the arrow directed upwards and to the right from previous acting cell); it is easy to check up that it is a new equilibrium of circulation (with forced volume flow rate and elevated resistance): L = 3.86 X 14 ≈ 54, P_i = 54 – 40 = 14. We confirm that the system maintains new normal rhythm ($\boldsymbol{t_N}$ = 3.86) calculating it as if normal rhythm is still essential t_N = 9.

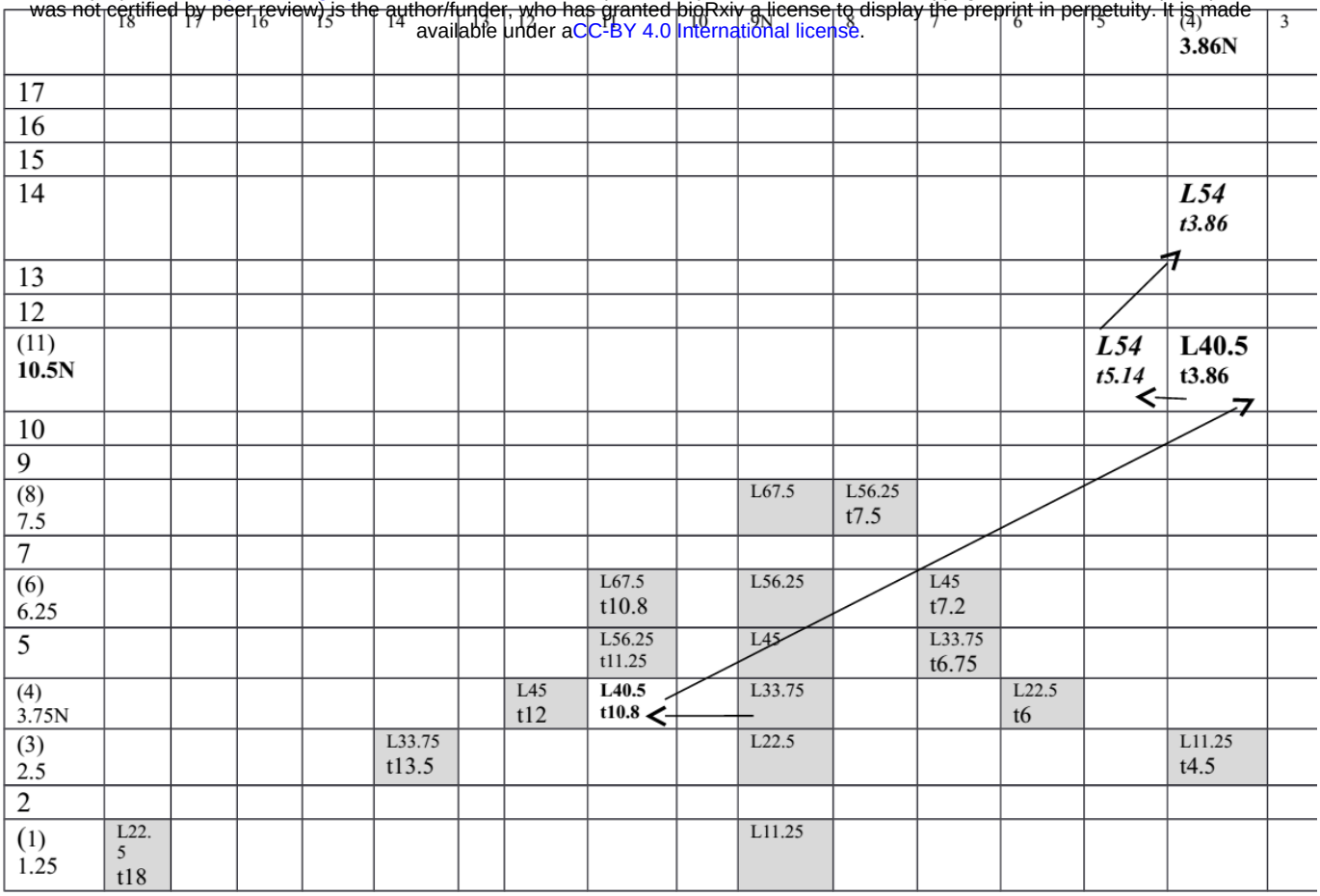


Fig.5. The background is the brady-mode of circulation taken from **Fig.3**. The primary variation is the forcing of volume flow rate; starting cell 3.75_pX9_t which characterizes circulation at R_N and $Q = 1$ is transformed into the cell 10.5_pX3.86_t which balances circulation at R_N and $Q = 1.2$ and organizes new normal values $\boldsymbol{t_N = 3.86}$, $\boldsymbol{P_N = 10.5}$ (horizontal arrow directed to the left and then the arrow directed upwards and to the right). Next variation is the one-step elevation of resistance ($\Delta R = 10$, $\sum R = 40$) which results in balancing circulation at 14_pX3.86_t (horizontal arrow directed to the left and arrow directed upwards and to the right). The system solves the problem of forcing Q and elevation of $\sum R$ mostly by acceleration of rhythm and by moderate rising of venous pressure.

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 and weak acceleration of rhythm. Brady-mode has another starting point - slow rhythm ($t_N = 9$) and low venous pressures ($P_N = 3.75$); brady-mode reacts with significant acceleration of rhythm and moderate rising of venous pressure. Nevertheless, final acceleration of rhythm by brady-mode ($t_N = 3.86$) provides much

slower rhythm than final acceleration of rhythm by tachy-mode (t_N = 2.25); besides, the highest value of venous pressure of brady-mode is 14 when then analogous highest value of tachy-mode is 32. We know from previous examples that restoration of resistance, at first, accelerates rhythm and then decelerates it down to normal value (following by the diminishing of venous pressure); consequently, tachy-mode needs to accelerate very rapid rhythm (t_N = 2.25) just more when the resistance turns back to its normal value. It means that tachy-mode - being imbalanced – is able to restore equipoise with much more difficulties then brady-mode which has maximally accelerated rhythm only t_N = 3.86.

Therefore, brady-mode can be considered more favorable as some standing mode which is ready to reorganize circulation when intensification of regime of circulation happens, - and it will reorganize it without approaching to extreme conditions which may appear irreversible. Extremity and possible irreversibility relates not only to rapid rhythm (as it is illustrated above) when the system is looking for new point of balance (situation caused by increasing of Q), - i.e. it relates not only to point of balance shifted so dangerous way, - but also it relates to high level of venous pressure which plays the role of counterbalance and which is compelled to be "very heavy" to equipoise rather insignificant imbalance ($\sum R = R_N + \Delta R = 30 + 10$; see **Fig.4**) and the material of venous vessels may not be prone to such pressures.

And what the choice of brady- or tachy-mode is based on? 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 we must emphasize on two conclusions which pretend to be the fundamental principles, a sort of: firstly, the normal rhythm must exist as some postulated (programmed) constant; secondly, such normal rhythm, which generates the standing mode of circulation, 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 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 norm 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 implements balancing of one-loop circulation.

References

1. Shi Y, Lawford P, Hose R (2011). Review of Zero-D and 1-D Models of Blood Flow in the Cardiovascular System. *Biomed Eng Online* **10**/33. Published online 2011 April 26. doi: 10.1186/1475-925X-10-33

2. Kibble J., Halsey C. Medical Physiology: The Big Picture, McGraw-Hill, 2009, 448 p.