# The conversion of systolic volume into systolic pressure (the development of numerical model)

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## Abstract

The direct proportional trend of the transition from systolic volume to systolic pressure can be proved theoretically with the help of circulatory numerical model. The model operates with compound parameter (anatomical portion of pipe, relation to the phase of cardiac cycle and pressure) which permits to adjust steady flow hydrodynamics to the description of the flow with interrupted indraft of pressure. The outstripping growth of systolic pressure (when diastolic pressure is rising with constant increment) is deduced and the above phenomenon can be observed if tracing systolic, diastolic and pulse pressures according to clinical gradation of arterial hypertension.

## Introduction

The present article ought not to be considered the independent theoretical investigation because it just branches out from the previous, basic article and develops the only one section of the model. This section needs the detailed description and special proof ensuing; it would overload the main demonstration of the basic article [1] and we decided to represent the section separately but in close connection with the methods worked out previously – hence, the references to [1] will appear continually. The topic itself concerns the transition from systolic volume to systolic pressure in terms of hydrodynamics adapted to pulsatile propelling of blood. The approach for the adjustment of steady flow hydrodynamics to the description of the flow interrupted periodically by cessation of the indraft of pressure was introduced already within terms of the simple numerical model [1]. Now the development of the above approach is presented which, to our opinion, entails the validity of the model on the whole.

Methods

At the beginning we would remind the suggested method of simulation of pulsatile blood flow [1]. 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 inception (systole) or to the end (diastole) of cardiac cycle – or to no one of them (i.e. we simulate pulsation or ignore pulsation);

- the pressure.

This approach seems evident when it concerns vein (which is a final portion of the pipe), venous pressure (which undoubtedly is a residual pressure) and the lack of the bond of venous pressure with systole or diastole. Hence, the necessity appears to divide the rest part of the pipe into two in order to bind one part with systole and another – with diastole. We use trivial transformation of standard hydrodynamical law $R=\frac{P_1-P_2}{Q}$ into $P=P_{syst}-P_{diast}=P_{syst}-Q\cdot\Sigma R$ , where P is venous pressure, Q – the volume flow rate, ΣR – general resistance; respectively, the two latter parameters are connected to diastolic pressure only. Consequently, we ought to determine where the portion of pipe which can be connected with diastole (as time interval of cardiac cycle) is located and where all the resistance is concentrated. The concentration of resistance is very useful approximation. Let all the resistance be considered belonged to arterial resistive vessels supplemented with capillary damper which liquidates the division systole-diastole (the resistance of the latter is considered negligible for simplification). The "aortic" portion of artery (artery located between the ventricle and resistive arteries) and also the vein, – as far as both these portions of pipe lack the resistance, - would never represent pressures inside in the form of Q • ΣR because ΣR is negligible. Nevertheless, volume flow rate and pressure are the observable parameters of the flow inside these portions of pipe. This is not a contradiction – this is a consequence of the usage of the above hydrodynamic law which describes the pipe with the resistance distributed evenly. Moreover, we have divided the pipe into three portions, firstly, due to concentration of the resistance in the middle part and, secondly, by attaching the initial portion of pipe to the initial part of cardiac circle (systole) and the middle portion – to the latest portion (diastole). And what about venous portion? What part of cardiac cycle can it be considered attached to? We may say that the venous flow can be considered either connected to both phases or to none of them. But can we represent such an answer by means of our triple parameter? The only

comprehensible way out is a spatial representation of this triple parameter, i.e. it is necessary to introduce the normal (as mathematical structure) and, as a consequence, to represent the orthogonal location of each component. It can be called non-parametric 3-dimensional vector and it permits to use negligibility, - as a zero of 3rd dimension (belonging to systole or diastole), - for determination of the belonging of venous flow to cardiac cycle; but the parameter remains 3-dimensional. It is the way out to avoid contradiction but we are not going to operate with vectors at the present model of one-loop circulation; it is sufficient to mention, - or, better to say, to postulate, - that the response of carotid reflex (see the previous article [1]) upon the variation of diastolic pressure (coinciding with the resistance at Q = 1) is extremely quick, i.e. the value of venous pressure which is involved in calculations will not be changed until the solution of the change of rhythm is found; but after the change of rhythm (following by the change of systolic pressure) the level of venous pressure will react immediately (this situation was partly conditioned and partly considered obvious at [1]). It is just a realization of the above zero-point (of the dimension determining the belonging to cardiac cycle) of triple parameter, - zero-point which characterizes venous pressure and venous portion of pipe. (Vector representation of triple parameters could be useful while supercomputer modeling, especially of the two-loop circulation.)

Let us remind the practical significance of triple parameter, - i.e. how have we applied it to calculations within arithmetic model. We associate $P_{syst}$ with the duration of systole (and with "aortic" portion of pipe) and we associate subtraction of $Q \bullet \Sigma R$ from $P_{syst}$ with the duration of the diastole (and with the portion of pipe represented by resistive arteries and capillary damper). All necessary arithmetic operations which accompany the basic equation were described at [1] in detail; but the transition from systolic volume to systolic pressure was taken for granted as the numerical equalization.

Was it permissible – to introduce the direct proportionality with coefficient of proportionality approximated to 1 as the transition from end-diastolic volume (which was primarily undergone to Frank-Starling's direct proportional transition to systolic volume) to systolic pressure? Evidently the preciseness of the answer is achievable within determination of the trend only.

But firstly it is necessary to understand what is a systolic pressure – in terms of present model (and triple parameter)?

The calculated deformation L is the end-diastolic volume which is directly proportional to the systolic volume ($V_{syst}$) due to Frank-Starling's law, i.e. $V_{syst} = k\,L$, where $k$ is a coefficient of proportionality without dimensions of quantity which we consider equal to 1. Any systolic volume which participates at Frank-Starling's law is being ejected from the ventricle during the identical time interval (duration of ventricular systole) and, consequently, the volume flow rate

(systolic volume per identical time interval) is directly proportional to the respective end-diastolic volume which participates at Frank-Starling's law. (Imagine a syringe. When you eject 1 ml per 1 sec. you move the piston with linear speed A, and when you eject 2 ml per 1 sec. you move the piston with linear speed 2A. Therefore, 1-dimensional parameter absolutely correlates with 3-dimensional process when the parameters of 2nd and 3rd dimensions are being not changed.)

Till now we have spoken about the two sides of one process, – two functions of one object (ventricle), - diastole and systole, which are the alternative processes in respect of the direction of the flow. When we deal with the vessel (and propelling of blood inside the vessel) we see only the reflection of work of ventricle, - the reflection of these two functions, - upon the hydrodynamic law $R=\frac{P_1-P_2}{Q}$ which depicts the steady flow, i.e. the flow synchronized with ventricular systole and the flow synchronized with ventricular diastole, - and both are of the same direction. Now let us apply this hydrodynamic law to the portion of the pipe which is located between the ventricle and arterial resistive vessels and is presented by elastic artery with constant resistance and which we call "the aortic portion" (the wall of such artery is approximated to the firm wall). We have mentioned already, - when we have applied the above formula to the pipe of the whole loop and conditioned that the resistance is concentrated in the middle part of the pipe, - that it is impossible to represent the pressure here via $Q\cdot R$ when the resistance trends to zero. Nevertheless the substantial level of pressure and volume flow rate is observable here (at "the aortic portion" of pipe); formally the difference of pressures at the beginning and the end of this initial part of arterial pipe can be defined as product of volume flow rate and the resistance (the latter trends to zero) in $P_1-P_2=Q\cdot R$, and such formula characterizes the steady flow. Now we need to reflect, - to superimpose, - the division of the flow into pairs (systolic and diastolic periods which have appeared in the pipe) upon this formula. We know that volume flow rate Q is significant only during systole and is negligible during diastole; now imagine that the resistance R is negligible: $P_1-P_2$ during systole and $P_1-P_2$ during the diastole, i.e. both differences of pressures, will be equalized as far as both will trend to zero. Now let us analyse situation when $P_1-P_2$ during systole is equal to $P_1-P_2$ during diastole: equal differences between pressures can be observed either when both values of pressure are within diapason of high pressures or both values of pressure are within diapason of low pressures. Therefore, left parts of the equations $(P_1-P_2)_{syst}=Q\cdot R$ and $(P_1-P_2)_{diast}=Q\cdot R$ can be considered equal despite of the absolute levels of pressure. Thus, $P_1-P_2$ during systole and $P_1-P_2$ during diastole are not only equal but both are

negligible, i.e. left parts of equations trend to zero. Respectively, right parts of these two equations will produce the trend to zero by means of three following ways: 1) Q is negligible and R is substantial; 2) Q is substantial and R is negligible; 3) both Q and R are negligible. Variant 2) may correspond to the period of ventricular systole; variant 3) may correspond to the period of ventricular diastole and variant 1) must be given up as far as it contradicts with the primary condition – that resistance R is negligible. Obviously variant 2) must be connected with negligible difference of pressures achieved at high level of pressures; respectively, variant 3) must be connected with negligible difference of pressures achieved at low level of pressures. The only one parameter which is substantial among other parameters of two equations is Q in the equation describing the flow in the pipe during the period of ventricular systole $(P_1 - P_2)_{syst} = Q \cdot R$ ; therefore, the fact that value of Q is substantial indicates that the level of pressures is high. The dependance between the values of Q and levels of high pressures during systole (pressures that do almost not differ one from another - at the beginning and at the end of "aortic" portion of the loop pipe) can be the object of further theoretical investigation within terms of present model. Nevertheless we can state even now, - according to the laws of conservation, - that the directly proportional growth of Q, as a response to the growth of ventricular volume per standard time interval of ventricular systole, will result in the rising of the level of pressures participating in formation of negligible difference between $P_1$ and $P_2$ (which both belong to the range of high pressures). We may state this because no resistance participate here and no difference of pressures appear, - i.e. the volume flow rate which really exist here during systole determines the level of pressure without any loss of energy. We may only assume for simplification that the dependance between growth of Q and rising of level of high pressures, - which now we can call "systolic pressures", - permits us numerically equate the end-diastolic volume L and systolic pressure $P_{syst}$ . Hence, numerical value of L can be substituted instead of $P_{syst}$ in equation $P_{syst} - P = Q \cdot \Sigma R$ which now relates to the pipe of the whole loop (P is a venous pressure, $Q \cdot \Sigma R$ determines the diastolic pressure which we observe – *during diastole* - at the portion of pipe where the whole resistance is concentrated and $P_{syst}$ is a systolic pressure which we observe in "aortic" pipe *during systole*). But why we have so easily associated the pressure described by $Q \cdot \Sigma R$ with diastolic period?

For the explanation it is pertinent to enumerate the structures the whole loop is combined of and summarize what operations each structure is responsible for:

- **the ventricle** converts end-diastolic volume L into systolic volume;
- **the artery between ventricle and arterial resistive vessels** ("aortic" portion of pipe);

this portion of pipe possesses high level of pressure during ventricular systole and this pressure hardly differs at the beginning and the end of the portion of pipe; the volume flow rate here is

varying due to to the different systolic volumes and standard duration of ventricular systole; the resistance inside this portion of pipe is negligible; this portion of pipe is the place where systolic pressure is organized by means of: 1) direct proportionality between systolic volume and volume flow rate per standard duration of ventricular systole, 2) conversion of variable substantial flow rate into systolic pressure – according to the law we are not aware of but which, we assume, lacks the energy loss, and that is why we numerically equalize $V_{syst}$ (and L, respectively) with $P_{syst}$ (*and permissibility of such substitution is the task of present investigation*); "aortic" pipe possesses the negligible level of pressure during ventricular diastole and this pressure hardly differs at the beginning and the end of this portion of pipe; Q during diastole is negligible at this portion of pipe and, consequently, diastolic pressure trends to negligible value – here, in "aortic" pipe;

- **the resistive arteries** where the variable volume flow rate (which comes from "aortic" pipe during shortly but constantly lasting systole) transforms into some constant value and is maintained as a constant value due to the appearance of substantial variable resistance ΣR (instead of negligible R in "aortic" pipe); the variable Q and the constant Q can not exist simultaneously and that is why it is necessary to associate the variable Q with one separate time interval (systole) and variable Q – with another separate time interval (diastole); it is identical to the fact that negligible resistance and substantial resistance can not be associated with the same portion of pipe, hence, the negligible (and obviously constant) resistance we really can associate with one portion of pipe and substantial (concentrated and variable) resistance we can associate with another portion of pipe; therefore, diastolic pressure is organized here as a product of *constant Q connected with diastolic period* and *variable substantial resistance ΣR connected with the region of resistive arteries*; this anatomical region plays the role of operator that converts systolic pressure into residual (venous) pressure, - as far as we excluded the resistance of capillary damper; hence, at present model the region of resistive arteries can not be characterized by systolic pressure and venous pressure.

- **the capillary damper** which converts pulsatile flow to the steady flow (but the resistance of capillary damper is considered negligible at present model for simplification);

- **the vein** with venous pressure which is determined as the remainder (minuend is a systolic pressure and subtrahend is diastolic pressure; the operation of subtraction mostly belongs to the region of resistive arteries but it is supplemented with operation of liquidation the division systole-diastole, i.e. with the operation which mostly belongs to capillary damper).

It is pertinent to emphasize that there are two divisions – in space (three portions of pipe: "aortic" portion, resistive arteries with capillary damper and the vein) and in time (systole, diastole and independence with respect to cardiac cycle), and that the uniting in pairs claims the impossibility of permutation.

Results

At present model we have equalized numerically the end-diastolic volume (which is a calculable value) and systolic pressure, and we are in need now to prove if such a substitution is permissible – i.e. if some process described by means of the model (where the above substitution works) corresponds to some physiological phenomenon. Practically it means that the parameter that reveals the work of the model (and it can be the elevation and falling of the resistance only), firstly, must be observed as registered parameter, and, secondly, the calculated parameter – the end-diastolic volume L (which can not be observed) - must behave identically (or approximately the similar way) to systolic pressure $P_{syst}$ (which can be observed and measured). Physiological parameter that combines the resistance and systolic pressure is, so called, arterial pressure $P_{syst}/P_{diast}$, where $P_{diast}$ denotes the variable resistance ΣR (which we use in calculations) multiplied by constant value of Q and which can be considered identical with $P_{diast}$ in case when the volume flow rate is equal to 1. The derivative parameter is a pulse pressure (the difference between systolic and diastolic pressures) which is convenient being expressed by one number when we are going to trace the sequence of values of arterial pressure (pairs of numbers) as a response to series of ΣR (= $P_{diast}$ ) with constant increment or decrement.

In the light of recent speculations we ought to ask ourselves – how can we measure systolic and diastolic pressures at one territorial (anatomical) point when we have postulated the inadmissibility of permutation of elementary parameters between triple parameters? Firstly, we must mention that the belonging of each type of pressure to the phase of cardiac cycle is maintained strictly. Secondly, the question reveals the characteristics of the flow of liquid which, in one case, are applicable to our artificial division of the pipe into three discrete parts (the conversion of the ordinary flow into capillary flow and again to ordinary flow in the pipe – the operation that winds up the subtraction of one pressure from another) and, in the second case, characteristics of the flow are hardly applicable to our division (the flow conducts the pressure along the whole arterial basin – i.e. the subtraction of one pressure from another can not be revealed connected strictly to the region of resistive arteries). Therefore, the attachment of diastolic pressure to resistive arteries and the attachment of systolic pressure to “aortic” portion of pipe remains very strict theoretically (for model which does not introduce the description of flowing liquid) but practical measuring of pressure, - which inevitably deals with blood flowing, - encounters with immediate conduction of pressure along arterial basin and, consequently, encounters with territorial spreading of minuend along the whole arterial basin

and subtrahend – also along the whole arterial basin. That is why the value of couple of pressures (arterial pressure) can be obtained either near the aortic valve or somewhere near arteriole.

**Tab.1** is based on data from Fig.1 [1]; we have used the data that regard to the steps of reorganization of norm-mode of circulation ($t_N$ =6, $P_N$ =6, Q=1.0) as a response upon the rising and falling of the resistance but excludes the restoration of each step to the previous one. Three steps of response of L (which is assumed numerically equal to $P_{syst}$ ) upon the rising of the resistance from the point of normal value $R_N$ = 30 (increment ΔR = 10) and two steps of response of L upon the falling of the resistance (decrement ΔR = -10) are represented. **Tab.1** shows the series of ΣR which are numerically identical with the values of $P_{diast}$ shown either in the line "Resistance" or in the line "Arterial pressure", and the series of L from Fig.1[1] is numerically identical with the values of $P_{syst}$ which are shown in the line "Arterial pressure". It is evident that pulse pressure is not a fixed value when the resistance is changing; pulse pressure grows linearly with increment of growth equal to 2.

| **norm-mode** $t_N = 6$ $P_N = 6$ **Q = 1.0** | ΣR = 10<br>$P_{diast}$ = 10 | ΣR = 20<br>$P_{diast}$ = 20 | $R_N$ = 30<br>$P_{diast}$ = 30 | ΣR = 40<br>$P_{diast}$ = 40 | ΣR = 50<br>$P_{diast}$ = 50 | ΣR = 60<br>$P_{diast}$ = 60 | Resistance |
|---|---|---|---|---|---|---|---|
| | 12/10 | 24/20 | 36/30 | 48/40 | 60/50 | 72/60 | Arterial pressure |
| | 2 | 4 | 6 | 8 | 10 | 12 | Pulse pressure |
| | 2 | 2 | 2 | 2 | 2 | | Increment of growth of pulse pressure |

**Table 1.** The steady rising of the resistance (which is identical to diastolic pressure when Q = 1.0) results in growth of pulse pressure; the represented norm-mode of circulation (which basic normal rhythm is not tachy-rhythm or brady-rhythm) is shortly described by information in the upper left cell; the gray column indicates the state of circulation when it is not deviated by shift of the resistance.

**Tab.2** is based on data from Fig.2 [1]; we have used the data that regard to the steps of reorganization of tachy-mode of circulation ($t_N$ =3, $P_N$ =15, Q=1.0) as a response upon the rising and falling of the resistance but excludes the restoration of each step to the previous one. Three steps of response of L (which is assumed numerically equal to $P_{syst}$ ) upon the rising of the resistance from the point of normal value $R_N$ = 30 (increment ΔR = 10) and two steps of response of L upon the falling of the resistance (decrement ΔR = -10) are represented. **Tab.2**

shows the series of ΣR which are numerically identical with the values of $P_{diast}$ shown either in the line "Resistance" or in the line "Arterial pressure", and the series of L from Fig.2 [1] is numerically identical with the values of $P_{syst}$ which are shown in the line "Arterial pressure". It is evident that pulse pressure is not a fixed value when the resistance is changing; pulse pressure grows linearly with increment of growth equal to 2.

| **tachy-mode** $t_N = 3$ $P_N = 15$ **Q = 1.0** | ΣR = 10 $P_{diast}$ = 10 | ΣR = 20 $P_{diast}$ = 20 | $R_N$ = 30 $P_{diast}$ = 30 | ΣR = 40 $P_{diast}$ = 40 | ΣR = 50 $P_{diast}$ = 50 | ΣR = 60 $P_{diast}$ = 60 | Resistance |
|---|---|---|---|---|---|---|---|
| | 15/10 | 30/20 | 45/30 | 60/40 | 75/50 | 90/60 | Arterial pressure |
| | 5 | 10 | 15 | 20 | 25 | 30 | Pulse pressure |
| | 5 | 5 | 5 | 5 | 5 | | Increment of growth of pulse pressure |

**Table 2.** The steady rising of the resistance (which is identical to diastolic pressure when Q = 1.0) results in growth of pulse pressure; the represented tachy-mode of circulation is shortly described by information in the upper left cell; the gray column indicates the state of circulation when tachy-mode is not deviated by shift of the resistance (it is the starting point, i.e. the normal state for tachy-mode).

**Tab.3** is based on data from Fig.3 [1]; we have used the data that regard to the steps of reorganization of brady-mode of circulation ($t_N$ =9, $P_N$ =3.75, Q=1.0) as a response upon the rising and falling of the resistance but excludes the restoration of each step to the previous one. Three steps of response of L (which is assumed numerically equal to $P_{syst}$ ) upon the rising of the resistance from the point of normal value $R_N$ = 30 (increment ΔR = 10) and two steps of response of L upon the falling of the resistance (decrement ΔR = -10) are represented. **Tab.3** shows the series of ΣR which are numerically identical with the values of $P_{diast}$ shown either in the line "Resistance" or in the line "Arterial pressure", and the series of L from Fig.3[1] is numerically identical with the values of $P_{syst}$ which are shown in the line "Arterial pressure". It is evident that pulse pressure is not a fixed value when the resistance is changing; pulse pressure grows linearly with increment of growth equal to 1.25.

| **brady-mode** $t_N = 9$ $P_N = 3.75$ **Q = 1.0** | ΣR = 10 $P_{diast}$ = 10 | ΣR = 20 $P_{diast}$ = 20 | $R_N$ = 30 $P_{diast}$ = 30 | ΣR = 40 $P_{diast}$ = 40 | ΣR = 50 $P_{diast}$ = 50 | ΣR = 60 $P_{diast}$ = 60 | Resistance |
|---|---|---|---|---|---|---|---|
| | 11.25/10 | 22.5/20 | 33.75/30 | 45/40 | 56.25/50 | 67.5/60 | Arterial pressure |
| | 1.25 | 2.5 | 3.75 | 5 | 6.25 | 7.5 | Pulse pressure |
| | 1.25 | 1.25 | 1.25 | 1.25 | 1.25 | | Increment of growth of pulse pressure |

**Table 3.** The steady rising of the resistance (which is identical to diastolic pressure when Q = 1.0) results in growth of pulse pressure; the represented brady-mode of circulation is shortly described by information in the upper left cell; the gray column indicates the state of brady-mode when circulation is not deviated by shift of the resistance (it is the starting point, i.e. the normal state for brady-mode).

As far as, - at the general case, - we do not know if the measured diastolic pressure is accompanied by Q = 1.0 or not, it is pertinent to investigate also the extreme modes of circulation (tachy-mode and brady-mode) with forced volume flow rate (Q = 1.2) . We are going to find out – how the increment of growth of pulse pressure is influenced by the forcing of volume flow rate.

**Tab.4** , the main part, is based on data from Fig.4 [1]; the upper small table is the central part of **Tab.2** and it is used for comparison (tachy-mode with volume flow rate not forced, i.e. Q = 1.0); the main table reflects the steps of reorganization of tachy-mode of circulation with forced volume flow rate ($t_N$ =3, $P_N$ =18, Q = 1.2) as a response upon the rising and falling of the resistance which now differs from $P_{diast}$ . Three steps of response of L (data from Fig.4 [1]; L is assumed numerically equal to $P_{syst}$ ) upon the rising of the resistance and two steps of response of L upon the falling of the resistance are represented as values of systolic pressure (see the line "Arterial pressure"). **Tab.4** shows the series of ΣR which now differs from the values of $P_{diast}$ (both are represented in the line "Resistance"); the values of $P_{diast}$ can also be found in the line "Arterial pressure"; the series of L (L18, L36, L54, L72, L90, L108) from Fig. 4 [1] is numerically identical with the values of $P_{syst}$ which are shown in the line "Arterial pressure". It is evident that pulse pressure is not a fixed value when the resistance (and diastolic pressure) are changing; pulse pressure grows linearly with increment of growth equal to 6. When comparing to the increment of growth of pulse pressure which persists at tachy-mode with Q = 1.0 (i.e. the quantity of 5 from **Tab. 2)**, we may notice that new quantity of 6 (**Tab.4**) is higher; it means that pulse pressure is growing still linearly but the slope of the function is more steep.

| tachy-mode $t_N = 3$ $P_N = 15$ **Q = 1.0** | $R_N = 30$ $P_{diast} = 30$ | Resistance |
|---|---|---|
| | 45/30 | Arterial pressure |
| | 15 | Pulse pressure |

| tachy-mode $t_N = 3$ $P_N = 18$ **Q = 1.2** | $\Sigma R = 10$ $P_{diast} = 12$ | $\Sigma R = 20$ $P_{diast} = 24$ | $R_N = 30$ $P_{diast} = 36$ | $\Sigma R = 40$ $P_{diast} = 48$ | $\Sigma R = 50$ $P_{diast} = 60$ | $\Sigma R = 60$ $P_{diast} = 72$ | Resistance |
|---|---|---|---|---|---|---|---|
| | 18/12 | 36/24 | 54/36 | 72/48 | 90/60 | 108/72 | Arterial pressure |
| | 6 | 12 | 18 | 24 | 30 | 36 | Pulse pressure |
| | 6 | 6 | 6 | 6 | 6 | | Increment of growth of pulse pressure |

**Table 4.** The steady rising of the resistance (which differs from diastolic pressure when Q = 1.2) results in growth of pulse pressure. The upper small table is the central part from the **Tab.3** (for comparison); the main table represents tachy-mode of circulation with forced volume flow rate (basic parameters are given at the upper left cell); the gray column indicates the state of circulation when tachy-mode with Q = 1.2 is not deviated by shift of the resistance (it is the starting point, i.e. the normal state for tachy-mode with Q = 1.2).

**Tab.5** , the main part, is based on data from Fig.5 [1]; the upper small table is the central part of **Tab.3** and it is used for comparison (brady-mode with volume flow rate not forced, i.e. Q = 1.0); the main table reflects the steps of reorganization of brady-mode of circulation with forced volume flow rate ($t_N$ =9, $P_N$ =4.5, Q = 1.2) as a response upon the rising and falling of the resistance which now differs from $P_{diast}$ . Three steps of response of L (data from Fig.5 [1]; L is assumed numerically equal to $P_{syst}$ ) upon the rising of the resistance and two steps of response of L upon the falling of the resistance are represented as values of systolic pressure (see the line "Arterial pressure"). **Tab.5** shows the series of ΣR which now differs from the values of $P_{diast}$ (both are represented in the line "Resistance"); the values of $P_{diast}$ can also be found in the line "Arterial pressure"; the series of L (L18, L36, L54, L72, L90, L108) from Fig. 5 [1] is numerically identical with the values of $P_{syst}$ which are shown in the line "Arterial pressure". It is evident that pulse pressure is not a fixed value when the resistance (and diastolic pressure) are changing; pulse pressure grows linearly with increment of growth equal to 1.5. When comparing to the increment of growth of pulse pressure which persists at brady-mode with Q = 1.0 (i.e. the quantity of 1.25 from **Tab.3)**, we may notice that new quantity of

1.5 (**Tab.5**) is higher; it means that pulse pressure is growing still linearly but the slope of the function is more steep.

| **brady-mode** $t_N = 9$ $P_N = 3.75$ **Q = 1.0** | $R_N = 30$ $P_{diast} = 30$ | Resistance |
|---|---|---|
| | 33.75/30 | Arterial pressure |
| | 3.75 | Pulse pressure |

| **brady-mode** $t_N = 9$ $P_N = 4.5$ **Q = 1.2** | $\Sigma R = 10$ $P_{diast} = 12$ | $\Sigma R = 20$ $P_{diast} = 24$ | $R_N = 30$ $P_{diast} = 36$ | $\Sigma R = 40$ $P_{diast} = 48$ | $\Sigma R = 50$ $P_{diast} = 60$ | $\Sigma R = 60$ $P_{diast} = 72$ | Resistance |
|---|---|---|---|---|---|---|---|
| | 13.5/12 | 27/24 | 40.5/36 | 54/48 | 67.5/60 | 81/72 | Arterial pressure |
| | 1.5 | 3 | 4.5 | 6 | 7.5 | 9 | Pulse pressure |
| | 1.5 | 1.5 | 1.5 | 1.5 | 1.5 | | Increment of growth of pulse pressure |

**Table 5.** The steady rising of the resistance (which differs from diastolic pressure when Q = 1.2) results in growth of pulse pressure. The upper small table is the central part from the **Tab.3** (for comparison); the main table represents brady-mode of circulation with forced volume flow rate (basic parameters are given at the upper left cell); the gray column indicates the state of circulation when brady-mode with Q = 1.2 is not deviated by shift of the resistance (it is the starting point, i.e. the normal state for brady-mode with Q = 1.2).

Therefore, five examples demonstrate the linear growth of pulse pressure while diastolic pressure is growing with steady increment. The dependency is based on calculation of end-diastolic deformation L (end-diastolic volume) which outstrips the steady elevation of the resistance (according to the equation {3} of the model [1]). The transition from L to systolic volume is a direct proportionality (Frank-Starling's law) and, consequently, only the transition from systolic volume to systolic pressure is unknown. The question is: either the latter transition duplicates the direct proportionality of Frank-Starling' law (maybe with slightly different coefficient of proportionality) or the transition (from systolic volume to systolic pressure) distorts the above direct proportionality of Frank-Starling's law? We have supposed that the transition is a directly proportional function with coefficient of proportionality equal to1, - i.e. we have numerically equalized systolic volume (and end-diastolic volume, respectively, as far as coefficient of proportionality of Frank-Starling's law was assumed equal

to 1) with systolic pressure. As a result – we see the series of paired values of pressures (arterial pressure) and series of values of pulse pressure which all demonstrate the one specific feature: the outstripping of systolic pressure (the increment of growth of pulse pressure is constant and differs from zero). If we observe something of the kind in reality it means that, firstly, calculations on the basis of the equation {3} [1] are true and, secondly, that the transition from systolic volume to systolic pressure is linear (or maybe that circulation uses only quasi-linear portion of the function which generally is not linear, - just like Frank-Starling's law does when it avoids extreme non-linear portions of its own function). The mostly simple way is to draw the attention to clinical practice of measuring of arterial pressure. What combinations of systolic and diastolic pressures, - which we consider normal either at calm conditions or at physical exertions, - are we get used to observe? **Tab.6** proposes the data which is trivial and approximate but the information reflects the trends which are undoubtedly observable, first of all, within sports medicine.

| | Diastolic pressure | Increment of growth of diastolic pressure | Pulse pressure | Increment of growth of pulse pressure |
|---|---|---|---|---|
| "Low arterial pressure" 90/60 | 60 | | 30 | |
| Normal arterial pressure 120/70, 120/80 | 70-80 | 10-20 | 40-50 | 10-20 |
| Heightened arterial pressure 140/90, 150/90 | 90 | 10-20 | 50-60 | 10 |
| High arterial pressure 160/100 – 200/110 | 100-110 | 10-20 | 60-90 | 10-30 |

**Table 6**. The ordinary clinical gradation of arterial hypertension (no connection with renal pathology); quantified values are approximate but quite observable (either within essential arterial hypertension or within responses to physical exertion).

The "low arterial pressure" is considered 90/60 (pulse pressure is equal to 30). The average normal arterial pressure is 120/80 or 120/70 (pulse pressure is 40-50), - i.e. the raise of diastolic pressure by 10-20 units results in elevation of pulse pressure by 10-20 units. The heightening of arterial pressure begins from 140/90, 150/90 and, consequently, the diastolic pressure raises by 10-20 comparing to normal scope (70-80); simultaneously pulse pressure reaches the values of 50-60, - i.e. it elevates from 40-50 up to 50-60 (with increment of growth

equal to 10 units). The next step of heightening of arterial pressure is stipulated by the observing values of diastolic pressure within 100-110; the values of systolic pressure that accompanies such level of diastolic pressure are within range of 160-200 (or higher). Therefore, standard raise of diastolic pressure by 10-20 units results in the increasing of pulse pressure up to 60-90 and it exceeds the previous values of 50-60 by 10-30, and it is quite close to the previous increments of growth of pulse pressure (which were equal to 10-20 and 10). Certainly something happens at the upper extreme portion of functions that are responsible for transition: 1) from diastolic volume to systolic volume and 2) from systolic volume to systolic pressure, - i.e. some non-linearity appears when volumes and pressures exceed some diapason, - but on the whole we have obtained the results (from such primitive source as approximate observations available to each physician) that basically correspond to the above calculations. The coincidence of theoretical prognostication and practical data is just in favor of the suggested model of one-loop circulation (described in [1] and developed at present work) which can be exploited for more complex simulations – especially supported by supercomputer modeling.

Reference

1. Yuri Kamnev. Viscous deformation of relaxing ventricle and pulsatile blood propelling (numerical model). doi: http://dx.doi.org/10.1101/009522 .