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

Author: Yuri Kamnev (Article of one author)

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);

- 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 organize the diastolic pressure. Consequently, we ought to determine where the portion of pipe which can be associated with diastole 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 portions of pipe lack the resistance, - would never represent differences of pressures inside these portions of pipe, - at the beginning and at the end of each portion of pipe (i.e. $P_{1\,venous}-P_{2\,venous}$ and $P_{1\,syst}-P_{2\,syst}$), - in the form of $Q\cdot R$ because R is negligible at these portions of pipe. Hence, the difference of pressures inside the vein, - if we change the negligible resistance inside the vein to minimal value, - appears small as far as Q is not rapid, and this corresponds to the observable flow of blood in the vein.

Inside the "aortic" portion of pipe we expect that the same operation, - to change negligible R to some minimal value, - will result in appearance of significant difference of pressures as far as Q is very rapid. Nevertheless, the difference of pressures between the aortic valve and down to the region of resistive arteries ("aortic" portion of pipe) hardly exists during systole, and so situation must be investigated in detail. Thus, we know that inside the "aortic" portion the 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 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 rising of the level of pressures $(P_1 - P_2)_{syst}$ (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} .

Let us develop our understanding of interrelation between the end-diastolic volume L and systolic volume V_{syst} [1]. 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. Certainly, numerical correlation between the speed of the piston and the ejected volume depends of the size of the syringe.) 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).

The reader may have noticed that some low pressures were introduced to the "aortic" portion of pipe and these pressures were associated with diastole, - meanwhile the "aortic" portion had been already associated with systole and with high pressures. For the search of 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 V_{syst} ;
- **the artery between ventricle and arterial resistive vessels** ("aortic" portion of pipe); this portion of pipe demonstrates high level of pressure during ventricular systole and this pressure hardly differs at the beginning and the end of this portion of pipe; the volume flow rate here is rapid and it varies 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 the volume flow rate per standard duration of ventricular systole, 2) conversion of the variable and rapid volume 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 directly proportionally bind V_{syst} (and L, respectively) with P_{syst} (*and permissibility of such substitution is the task of present investigation*); besides, "aortic" pipe demonstrates the

pressure of the low level during ventricular diastole and this pressure also hardly differs at the beginning and the end of this portion of pipe; Q during diastole is negligible at this portion of pipe and, - as far as we do not know the origin of the above low pressures, - we can not associate these pressures with negligible Q.

- **the resistive arteries** where the rapid and variable volume flow rate (which comes from "aortic" pipe during shortly but constantly lasting systole) transforms into some constant value which we associate with diastole and the new Q is maintained as a constant value due to substantial variable resistance ΣR concentrated here (instead of negligible R in "aortic" pipe); in other words, we see the permutation: rapid and variable Q from "aortic" pipe during systole is converted into steady Q that we observe here, in the region of resistive arteries, during diastole, - and negligible (constant) resistance which exists in "aortic" pipe during systole converts here, in the region of resistive arteries, into substantial variable resistance ΣR during diastole; therefore, diastolic pressure is organized ($Q \cdot \Sigma R$) which we associate with diastole in the region of resistive arteries but systolic period still exists here and we do not know what level of pressure (previously it were high levels of systolic pressures in "aortic" pipe) will be observed in the region of resistive arteries.

- **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 turn back to the low level of pressures (in "aortic" pipe during the diastole) – which we have nothing to associate with: the only way out is to consider that the "aortic" portion of pipe, - where we observe the low pressures, - is not "factually" an "aortic" portion of pipe, - i.e. we see here (in "aortic" portion of pipe) the picture reflected from another portion of pipe – from the portion of resistive arteries, - and, consequently, these low pressures are the pressures organized by steady Q and variable ΣR ($Q \cdot \Sigma R$) in the portion of resistive arteries during the diastole. The similar explanation can be given to the levels of pressure during systole at the region of resistive arteries: it is a branch of "aortic" portion of pipe inside the region of resistive arteries – the branch that appears each systolic period. The difference between the filial of "aortic" pipe (with systolic pressure – high pressure) inside the region of resistive arteries during systole and the filial of the region of resistive arteries (with diastolic pressure – low pressure) inside "aortic" pipe during the diastole is the following (although symmetry is evident): systolic pressure comes across with significant resistance ΣR in the region of resistive arteries but diastolic pressure comes across with negligible resistance in "aortic" pipe.

Results

At present model we have equalized numerically the end-diastolic volume (which is a calculable value) and systolic pressure, - more strictly, we have supposed direct proportionality (with coefficients of proportionality equal to1) between L, V_{syst} and P_{syst}, 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 – once again for practical purpose, - how can we measure systolic and diastolic pressures at one territorial (anatomical) point, i.e. at the same portion of the pipe?

The answer is partly given above. The "aortic" portion of pipe "disappears" during the diastole as a conduit adjusted for high level of systolic pressures and we observe inside the "aortic" portion of pipe the reflection of pressure that exists during diastole in the portion of pipe where the resistive arteries are located (diastolic pressure). Certainly, the period of systole at the region of resistive arteries must also "disappear" - symmetrically – as the producer of diastolic pressure; the resistive arteries will only conduct high pressure flow. However, as we have mentioned, the resistances are different,- in "aortic" pipe it is negligible and in the region of resistive arteries it is significant; consequently, the measuring of diastolic pressure in "aortic" pipe (i.e. at elastic or huge muscular-elastic arteries) precisely reflects the diastolic pressure originated in the region of resistive arteries – but the measuring of systolic pressure somewhere at the region of resistive arteries (procedure which is not easy in practical way) will show the diminished values comparing to the data from "aortic" portion of pipe.

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 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
	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**	$\Sigma R = 10$ $P_{diast} = 10$	$\Sigma R = 20$ $P_{diast} = 20$	$R_N = 30$ $P_{diast} = 30$	$\Sigma R = 40$ $P_{diast} = 40$	$\Sigma R = 50$ $P_{diast} = 50$	$\Sigma R = 60$ $P_{diast} = 60$	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**	ΣR = 10 $P_{diast} = 12$	ΣR = 20 $P_{diast} = 24$	$R_N = 30$ $P_{diast} = 36$	ΣR = 40 $P_{diast} = 48$	ΣR = 50 $P_{diast} = 60$	ΣR = 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**	ΣR = 10 $P_{diast} = 12$	ΣR = 20 $P_{diast} = 24$	$R_N = 30$ $P_{diast} = 36$	ΣR = 40 $P_{diast} = 48$	ΣR = 50 $P_{diast} = 60$	ΣR = 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 .