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A Lumped Parameter Model of the Cardiovascular Circulation

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Introduction

Cardiovascular diseases cause the majority of deaths in the developed countries. They are strongly interlinked with hemodynamics of the cardiovascular system (CS); thus it is important to study blood flow under normal and pathological conditions. Hemodynamic models of CS can be classified as lumped parameter, one-dimensional, two-dimensional and three-dimensional models. The simplest lumped parameter models are attractive for teaching purposes as well as for clinicians, since they describe the whole CS with a small number of parameters (in terms of compliance, resistance and inertance) having a clear physiological meaning. The results of such a model include pressure and volume time variations of observed compartments and also flow rates between compartments (arterial and vein trees are considered as compartments, too). Upgraded with a short-term regulatory system model (arterial baroreflex system, cardiopulmonary baroreflex system and neural control of the heart rate) and resting physiologic perturbations model, the resulting lumped parameter model is capable of generating pretty realistic results, and can be used for the research on the CS [1,2]. In the CVsim model [2] six compartments are used for the teaching version and 21 compartments in the research version, while the CircAdapt model [3] consists of eight compartments. The valves are considered to be ideal check valves in most models, and valve dynamics is modeled in [4]. A lumped model of the heart was developed in combination with one-dimensional model of blood vessel tree [5], and further there is a model with oxygen transport [6].

The goal of this work is to develop a simple hydrodynamic lumped parameter model of the cardiovascular system which can realistically describe time varying pressure in system compartments and flows through the valves in the given physiological state of the system. The model includes four heart compartments (left and right atria and ventricles) and systemic and pulmonary circulation (modeled by arterial and venous compartments). Heart contractility is modeled by time varying elastance [7] (frequently defined as the universal one for all subjects) in most existing models, while in this work the formulation with activation function is used for this purpose. Activation function is defined by parameters that can be obtained by Doppler Echocardiography specifically for each subject.

Mathematical model

A simplified CS (Fig. 1) is reduced to a system of eight chambers, as follows: pulmonary veins (PV), left atrium (LA), left ventricle (LV), systemic arteries (SA), systemic veins (SV), right atrium (RA), right ventricle (RV) and pulmonary artery (PA). Each chamber is characterized by volume (V) and pressure (p), while the blood flowrate (Q) is defined by eight connections between the chambers. These eight connections are: entrance from pulmonary veins into the left atrium (la), mitral valve (mv), aortic valve (av), systemic capillaries (sc), entrance to the right atrium (ra), tricuspid valve (tv), pulmonary valve (pv) and pulmonary capillaries (pc). For the sake of simplicity, it is assumed that pressure disturbances spread at infinite speed, resulting in uniform pre-

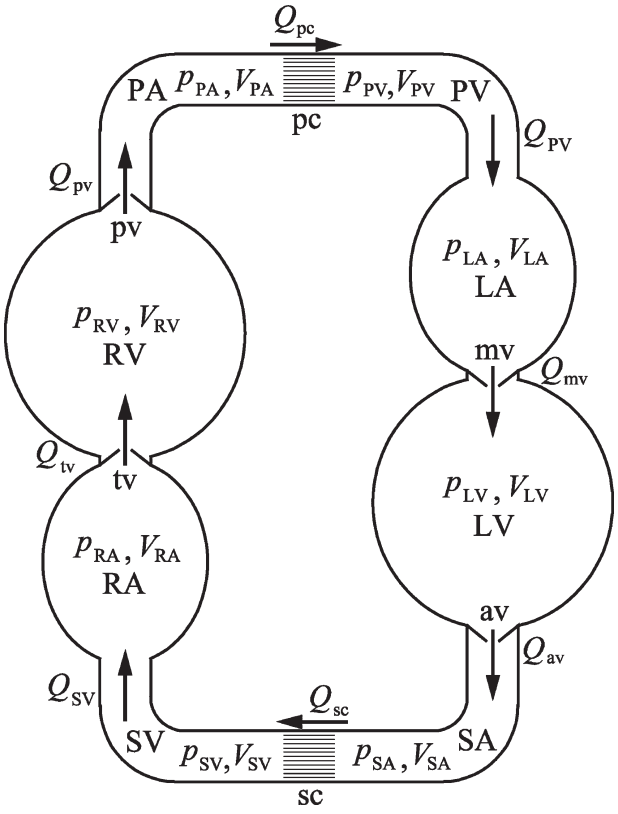

Fig. 1. Scheme of the cardiovascular system consisting of eight chambers and eight interconnections. Abbreviations: PV/SV = pulmonary/systemic veins, LA/RA = left/right atrium, LV/RV = left/right ventricle, SA/PA = systemic/pulmonary arteries, sc/pc = systemic/pulmonary capillaries, mv = mitral valve, av = aortic valve, tv = tricuspid valve, pv = pulmonary valve

ssure in each chamber is uniform. Arteries and veins are considered to be passive chambers (not adding energy to the blood flow), and the left and right heart chambers (atria and ventricles) are active chambers that add energy to the blood flow by their contraction. Blood is considered to be an incompressible fluid of constant density $\rho = 1050\ \mathrm{kg/m^3}$.

The continuity equation defines the rate of chamber volume change:

$$\frac{\mathrm{d}V}{\mathrm{d}t} = Q_{\mathrm{in}} - Q_{\mathrm{out}}\,, \tag{1}$$

where Q_{in} and Q_{out} are the inlet and outlet flow rate, respectively. For example, for pulmonary veins (PV) in Fig. 1 the continuity equation reads: $\mathrm{d}V_{\mathrm{PV}}/\mathrm{d}t = Q_{\mathrm{pc}} - Q_{\mathrm{PV}}$, for the left atrium (LA) it is: $\mathrm{d}V_{\mathrm{LA}}/\mathrm{d}t = Q_{\mathrm{la}} - Q_{\mathrm{mv}}$, and so on. Flow rate between two chambers is defined by the modified Bernoulli equation, which in the case of laminar fluid flow through the pipe of length L and diameter D, takes the form:

$$M\frac{\mathrm{d}Q}{\mathrm{d}t} = p_{\mathrm{in}} - p_{\mathrm{out}} - RQ - rQ^2\,, \tag{2}$$

where M is inertance coefficient, $M = \rho L/A$ ($A = D^2\pi/4$), $R = 32\mu L/\left(D^4\pi\right)$, μ is blood viscosity, $r = 8\rho K/\left(D^4\pi^2\right)$, and K is minor loss coefficient. It is convenient to neglect minor losses ($r = 0$) for the flows through systemic and pulmonary capillaries where friction losses are large, while friction loses are negligible ($R = 0$) with respect to minor losses in flows through valves. All valves are considered to be an ideal check-valve: the valve opens instantaneously for positive flow direction, and it closes instantaneously when flow direction tends to be negative.

Pressure-volume relationships

Veins and arteries models

Veins are modeled as vessels with elastic wall. If we define pressure as a pressure difference of inner vessel and interstitial pressure, then the pressure-volume relationship for veins reads:

$$p = E_0\left(V - V_0\right), \tag{3}$$

where E_0 is venous wall elastance and V_0 is blood volume in veins at zero pressure.

Arteries are modeled as vessels with a visco-elastic wall, and the pressure volume relationship, according to the Voigt model is:

$$p = E_0\left(V - V_0\right) + \eta\frac{\mathrm{d}V}{\mathrm{d}t}\,, \tag{4}$$

where η is arterial wall resistance.

Models of atria and ventricles

The walls of atria and ventricles contain muscles which contract after activation, and in that way they provide the driving force for blood flow. We distinguish two states of the wall: passive and active. The passive (or diastolic) state is modeled by a nonlinear passive pressure – volume relationship ($p_{\mathrm{d}} - V$):

$$p_{\mathrm{d}} = E_0 V_k\left(e^{\frac{V-V_0}{V_k}} - 1\right)\,, \tag{5}$$

where E_0 and V_0 are wall elastance and volume at zero pressure, respectively, and V_k is volume constant. In this model wall elastance (E_{d}) is volume (or pressure) dependent and thus it holds:

$$E_{\mathrm{d}} = \frac{\mathrm{d}p_{\mathrm{d}}}{\mathrm{d}V} = E_0 e^{\frac{V-V_0}{V_k}}\,. \tag{6}$$

The activation of muscles in the ventricular or atrial wall results in an additionally developed pressure, which should be added to passive pressure, resulting in total pressure:

$$p = p_{\mathrm{d}} + \alpha\left(p_{\mathrm{s}} - p_{\mathrm{d}}\right), \tag{7}$$

where α is time dependent activation function in the range from zero to one ($\alpha = 0$ denotes passive state and $\alpha = 1$ the end of systole). p_{s} is usually considered to be linear ESPVR (End Systolic Pressure-Volume Relationship), and here we added a quadratic term; thus, the expression for p_{s} is:

$$p_{\mathrm{s}} = E_0\left(V - V_0\right) + E_V V^2\,, \tag{8}$$

where E_V is constant coefficient.

During systole, when heart muscles contract, activation function rises from zero (at the beginning of systole) to one (at the end of systole), and after that it goes back to zero (relaxation). Here, we defined $t = 0$ at the beginning of systole, and we modeled activation function with a piecewise function:

$$\begin{aligned}
&\alpha^{\mathrm{I}} = A_1 t^2 \exp\left[A_2\left(t - t_{\mathrm{eivc}}\right)\right] && \text{for } 0 < t \le t_{\mathrm{eivc}}\\
&\dot{\alpha}^{\mathrm{II}} = A_3 + \left(1 - \frac{t - t_{\mathrm{eivc}}}{t_{\mathrm{es}} - t_{\mathrm{eivc}}}\right)\left(\dot{\alpha}_{\mathrm{eivc}} - A_3\right)\exp\left(A_4\left(t - t_{\mathrm{eivc}}\right)\right) - \\
&\quad - A_3\frac{t - t_{\mathrm{eivc}}}{t_{\mathrm{es}} - t_{\mathrm{eivc}}}\exp\left(A_4\left(t - t_{\mathrm{eivc}}\right)\right) && \text{for } t_{\mathrm{eivc}} < t \le t_{\mathrm{es}}\\
&\dot{\alpha}^{\mathrm{III}} = \frac{\alpha_{\mathrm{ee}}\left(t - t_{\mathrm{es}}\right)}{t_{\mathrm{ee}} - t_{\mathrm{es}}}\exp\left(A_5\left(t - t_{\mathrm{ee}}\right)\right) && \text{for } t_{\mathrm{es}} < t \le t_{\mathrm{ee}}\\
&\alpha^{\mathrm{IV}} = \alpha_{\mathrm{ee}}\exp\left(\frac{t_{\mathrm{ee}} - t}{\tau}\right) && \text{for } t > t_{\mathrm{ee}}
\end{aligned} \tag{9}$$

At time $t = 0$ (the beginning of the isovolumic contraction) $\alpha = 0$ and its time derivative $\dot{\alpha} = \mathrm{d}\alpha/\mathrm{d}t = 0$. Isovolumic contraction lasts up to time t_{eivc} (eivc = end of isovolumic contraction), when the aortic valve opens and activation function, and its time derivative take values $\alpha = \alpha_{\mathrm{eivc}}$ and $\dot{\alpha} = \dot{\alpha}_{\mathrm{eivc}}$, respectively. At the time of end-systole (t_{es}), interpolation function reaches its maximum value $\alpha = 1$, $\dot{\alpha} = 0$. After that, activation function decreases and at the time of end-ejection (t_{ee}) it gets a value of $\alpha = \alpha_{\mathrm{ee}}$. At t_{ee} time the aortic valve closes, and after that the isovolumic relaxation of the left ventricle starts. It is broadly accepted that during isovolumic relaxation the pressure falls according to the exponential law [8]: $p = p_{\mathrm{ee}}\exp(-t/\tau)$, where τ denotes isovolumic relaxation time constant, so it is reasonable to accept that activation function also follows the exponential law.

In Eq. (9) five unknown constants (A_1 to A_5) and two additional constants (A_6 and A_7) appear after integration expressions for $\dot{\alpha}^{II}$ and $\dot{\alpha}^{III}$. These seven constants are defined by seven conditions of continuity of α, $\dot{\alpha}$ and $\ddot{\alpha}$ as follows:

$$\begin{aligned}
&\alpha^{I}(t_{eivc}) = \alpha_{eivc} \\
&\alpha^{II}(t_{eivc}) = \alpha_{eivc} \\
&\dot{\alpha}^{I}(t_{eivc}) = \dot{\alpha}^{II}(t_{eivc}) \\
&\alpha^{II}(t_{es}) = 1 \\
&\alpha^{III}(t_{es}) = 1 \\
&\dot{\alpha}^{III}(t_{ee}) = -\alpha_{ee}/\tau \\
&\ddot{\alpha}^{II}(t_{es}) = \ddot{\alpha}^{III}(t_{es})
\end{aligned} \quad .$$

Once the unknown constants are resolved, the interpolation function is uniquely defined by the following seven parameters: t_{eivc}, α_{eivc}, $\dot{\alpha}_{eivc}$, t_{es}, t_{ee}, α_{ee} and τ.

The described activation function is used for ventricles and atria with a note that atrial contraction precedes ventricular contraction by time t_{av} (also known as PR interval in ECG). Panel A in Fig. 2 shows typical activation functions for ventricles (black line) and atria (blue line), and time derivative of ventricular activation function (red line). Here, we assume the same activation function for the left and for the right ventricle and the same activation function for the left and for the right atrium.

Numerical procedure

The mathematical model (the set of 24 ordinary differential equations) is integrated by the fourth order Runge-Kutta method. Initially, all flow rates are set at zero, and the volumes of all chambers set at values at the expected average pressure for each chamber. The integration is performed over multiple heart periods (usually ten periods is enough), in order to achieve cycle-to-cycle periodicity, and the results of the last cycle are taken. Integration time step was 1 ms.

Computational results and discussion

Typical results of the described model are presented in Fig. 2. The given results are for a normal subject with an arterial blood pressure of 120 / 80 mmHg, (green line in panel B). Left ventricular and atrial pressures are shown in black and blue line, respectively. There is no incisure in the arterial pressure wave form that is normally seen in invasively obtained measurements, because we did not model aortic valve dynamics. Panel C shows velocity profiles through the aortic valve (black line), mitral valve (blue line) and pulmonary veins (red line). Velocity peaks and profiles are in close agreement with the observations obtained by Doppler Echocardiography in normal subjects. In pulmonary veins flow we can see three distinct waves: positive S and D-wave as well as the negative A-wave. Panel D shows the time variation of left ventricular (black line) and atrial volume (blue line). It is visible that the range of change of atrial volume is much smaller than the ventricular one, since the atrium simultaneously fills and empties.

Fig. 3 shows pressure-volume loops for the left atrium (top panel) and ventricle (bottom panel). Pressure variation in the left atrium is within the physiological range and the shape of p-V loop correctly reflects events in the left atrium during one cycle. The same is valid for the left ventricle.

In the proposed formulation of heart systolic function (defined by Eqs. (7) to (9)) the needed parameters can be well estimated for each subject specifically, in subjects having mitral (and tricuspid) regurgitation. For this purpose, we use blood velocity profiles through the aortic (v_{av}) and mitral (v_{mv}) valve obtained by Doppler Echocardiography during systole. Since the opening in mitral valve during systole is small, the inertial effect and line friction losses are negligible ($M = 0$ and $R = 0$) and minor loss coefficient $K = 1$, it follows from Eq. (2) that

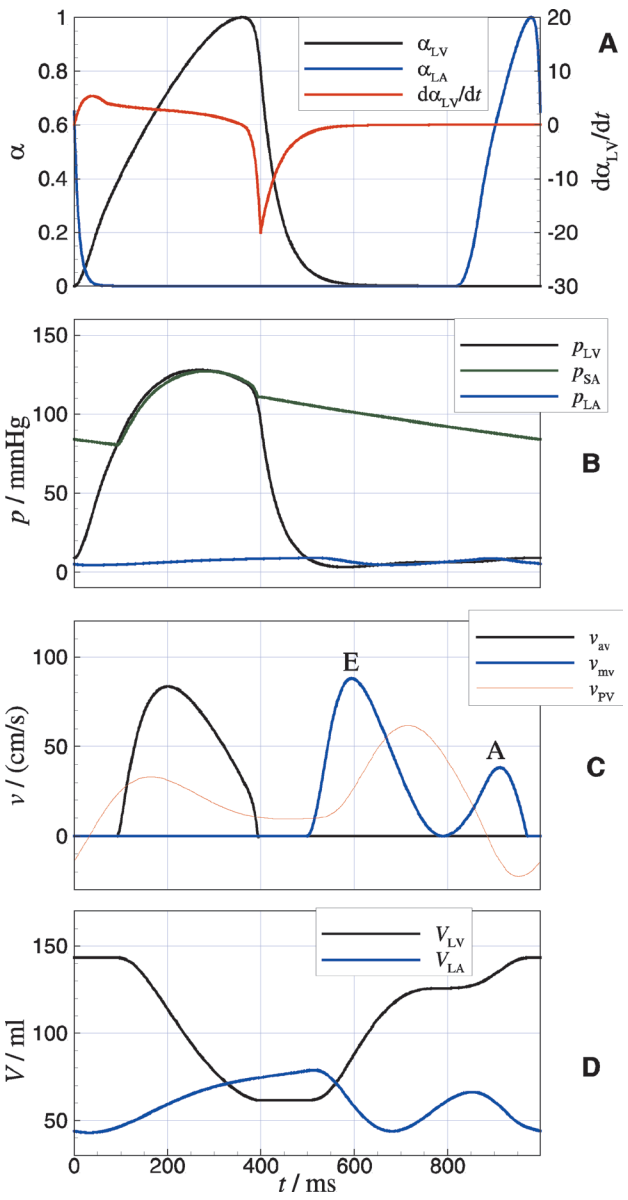

Fig. 2. Results from the proposed model. Panel A: activation function for ventricles and atria, and time derivative of left ventricular activation function; Panel B: Time variation of left ventricular, left atrial and systemic artery pressures; Panel C: time variation of aortic and mitral valve and pulmonary vein velocities; Panel D: Time variation of left ventricular and left atrial volume

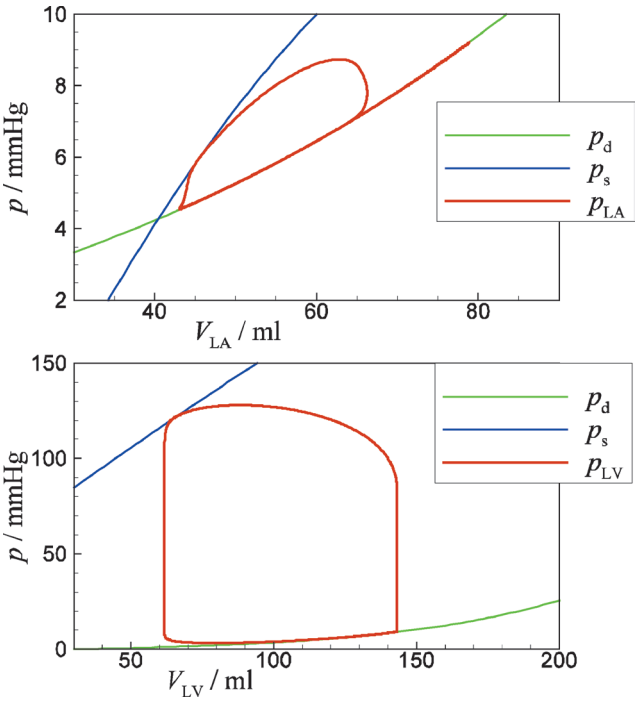

Fig. 3. Pressure-volume loop for left atrium (top) and left ventricle (bottom)

$$p_{LV} = p_{LA} + \frac{\rho}{2} v_{mv}^2 . \quad (10)$$

In Eq. (10) p_{LA} is small and can be either neglected or replaced by its average value. In Eq. (7) the passive pressure p_d is much smaller than p_s, and after neglecting it we have:

$$p_{LV} = \alpha(t) p_s(V_{LV}) . \quad (11)$$

We obtain the stroke volume by integration of v_{av} during the ejection time T_{ej}

$$V_{stroke} = \frac{D_{av}^2 \pi}{4} \int_0^{T_{ej}} v_{av} \, dt , \quad (12)$$

where D_{av} is the left ventricular outflow tract diameter, also measured by Doppler Echocardiography. End diastolic volume is $V_{ed} = V_{stroke} / E_f$, where E_f is the ejection fraction which can be estimated by the Teicholz echo method. The time variation of V_{LV} is then

$$V_{LV}(t) = V_{stroke} - \frac{D_{av}^2 \pi}{4} \int_0^t v_{av}(t') \, dt' . \quad (13)$$

For the given $\alpha(t)$ and V_{LV} we calculate p_s from Eq. (11) and find the coefficients in Eq. (8) by parabolic curve fitting.

There are seven parameters defining the activation function, and two of them (t_{eivc} and t_{ee}) are measured, t_{es} should be in the range defined by the time of the maximum p_{LV} and t_{ee}, and the rest four should satisfy some conditions at the end of isovolumic contraction and at the beginning of isovolumic relaxation. It follows from Eq. (11) that at constant V_{LV} it holds:

$$\frac{dp_{LV}}{dt} = p_s \frac{d\alpha}{dt} . \quad (14)$$

When we apply Eqs. (11) and (14) at $t = t_{eivc}$ when $V_{LV} = V_{ed}$ and $p_{LV} = p_{dia}$ (p_{dia} is the arterial diastolic pressure) and $t = t_{ee}$ when $V_{LV} = V_{es} = V_{ed} - V_{stroke}$ and $p_{LV} = p_{ee}$ (p_{ee} is the ventricular aortic valve closing pressure), we have

$$p_{dia} = \alpha_{eivc} p_s(V_{ed}) , \; p_{ee} = \alpha_{ee} p_s(V_{es}) ,$$
$$\left.\frac{dp_{LV}}{dt}\right|_{eivc} = p_s(V_{ed}) \dot{\alpha}_{eivc} \text{ and } \left.\frac{dp_{LV}}{dt}\right|_{ee} = p_s(V_{es}) \dot{\alpha}_{ee} . \quad (15)$$

If we accept exponential decay low for pressure and activation function, then we have two additional relations

$$\left.\frac{dp_{LV}}{dt}\right|_{ee} = \frac{p_{ee}}{\tau} \text{ and } \dot{\alpha}_{ee} = \frac{\alpha_{ee}}{\tau} . \quad (16)$$

Since we can estimate the left ventricular pressure and also its time derivative at $t = t_{eivc}$ and $t = t_{ee}$ from the mitral regurgitant flow, we use the above relations to find parameters defining activation function.

In the following paper we discuss the method of parameter estimation for pulmonary circulation.

There is still more room for improvements of the proposed model, since:

1) The model does not include valve dynamics. It is known that valve closing causes water hammer, i.e. incisure in the pressure profile.
2) Also, excursion of the annular planes of tricuspid and mitral valve may have an impact on the heart hemodynamic in different phases of cardiac cycle, so it would be of interest to include it into the model.
3) We use the four-element Windkessel model for the arterial systems. Windkessel models with more elements will represent the heart afterload more accurately.
4) Coupling of the lumped parameter model for the heart and one-dimensional model for arteries would provide more information of interest for clinicians.

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Non-invasive Method for Parameter Identification in a Lumped Parameter Model of Pulmonary Circulation

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Introduction

Understanding the function of the right heart and pulmonary circulation becomes more and more important in the treatment of cardiac and pulmonary diseases [1]. Hemodynamic data for examination of pulmonary circulation are usually obtained invasively, what is unacceptable in healthy subjects. That is why we need a non-invasive clinical method for the estimation of pulmonary circulation function that would be suitable for all subjects (with normal function and with cardiorespiratory diseases).

The aim of this work is to develop a lumped parameter model of pulmonary circulation and to develop a method for parameter identification of this model, based on non-invasive (Echocardiography) measurements of velocity profiles through heart valves.

Mathematical model

Fig. 1 schematically shows the right heart, pulmonary artery (PA), lungs, pulmonary veins (PV) and left atrium (LA) as well as the electrical analogue scheme of the proposed lumped model of pulmonary circulation. Resistor R models the total pulmonary vascular resistance, capacitors C and C_1 model arterial compliances of proximal and distal parts, L represents the inertial effects within arteries, η is the wall resistance of the proximal part (the Voigt model) and Z models the impedance of the rest of the system. For the given model parameters and for input pulmonary valve flow ($Q_{pv} = v_{pv} A_{pv}$), it is possible to calculate pulmonary root pressure (p_{PA}). When p_{PA} is measured, it is possible to find optimal values of model parameters which minimize the RMS error between measured p_{PA} and p_{PA} calculated with the model.

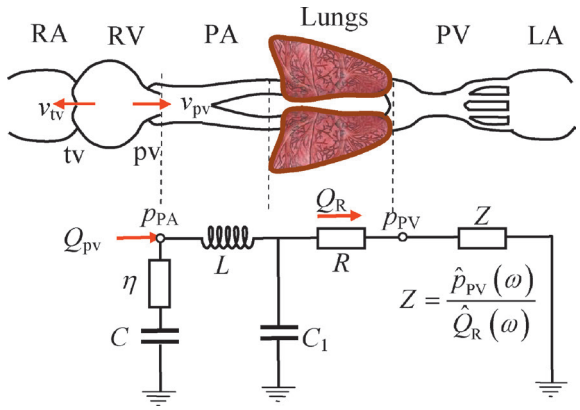

Fig. 1. Schematics of the pulmonary circulation and electrical analogue scheme of its lumped mathematical model. RA/RV = right atrium/ventricle, PA/PV = pulmonary arteries/veins, LA = left atrium, tv/pv = tricuspid/pulmonary valve, Z = impedance, ω = circular frequency

Measurements

By using Doppler Echocardiography, it is possible to measure pulmonary valve (v_{pv}) and tricuspid regurgitant blood velocity (v_{tv}). In each particular case, several measurements were recorded and the average data profiles were calculated. The "measured" p_{PA} is obtained from unsteady Bernoulli equation

$$p_{PA} = p_{RA} + \frac{1}{2}\rho v_{tv}^2 - \frac{1}{2}K\rho v_{pv}^2 - \rho l \frac{dv_{pv}}{dt}, \qquad (1)$$

where K and l are minor loss coefficient and inertial length through the pulmonary valve, respectively, p_{RA} is the average right atrium pressure, which is estimated from the width of vena cava inferior. Similarly, the average pressure in pulmonary veins (p_{PV}) is estimated from the mitral inflow pattern. The stroke volume calculated from the pulmonary and aortic valve velocity should be the same

$$V_{stroke} = \int_0^{T_{ej}} v_{pv} A_{pv} dt = \int_0^{T_{ej}} v_{av} A_{av} dt, \qquad (2)$$

where T_{ej} is ejection time. Since the aortic valve area can be measured more precisely, we use Eq. (2) to calculate A_{pv}.

Parameter identification

First, the pulmonary artery input impedance $Z_{in} = \hat{p}_{PA}/\hat{Q}_{pv}$ (the ratio of the pressure and flow phasors defined by the Fourier series) is calculated, and then p_{PA}^{WK5} is obtained based on measured Q_{pv}. This pressure is compared with the p_{PA} defined by Eq. (1), and the error defined as

$$RMSE = \sqrt{\frac{1}{N}\sum_{i=1}^{N}\left(p_{PA}^{WK5} - p_{PA}\right)^2} \qquad (3)$$

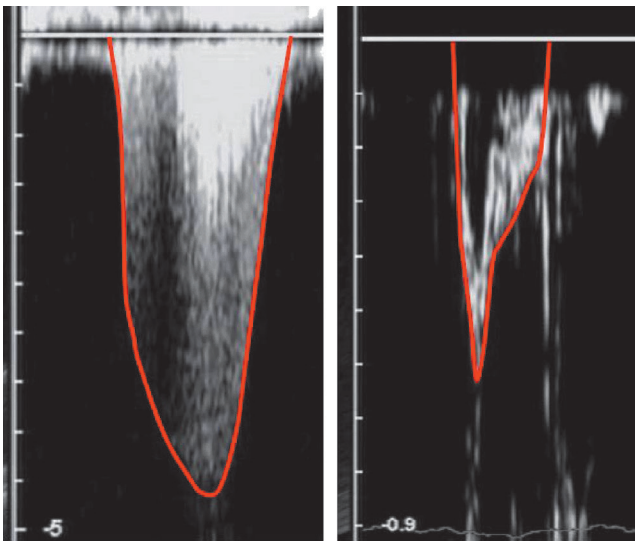

Fig. 2. Example of Doppler regurgitant tricuspid (left) and pulmonary velocity (right). Red lines are plotted for a digitization purpose

(where N is the number of points within T_{ej}) is minimized.

Results and Remarks

The method was applied to an elderly patient with pulmonary arterial hypertension using the following data: Cardiac Output: 5 l/min, Heart Rate 78 beat/min, pulmonary valve diameter D_{pv} = 2.73 cm, isovolumic contraction of RV time t_{eivc} = 34.4 ms, p_{RA} = 5 mmHg, p_{PV} = 10 mmHg, $l = 1.53D_{pv}$, $K = 1$. Fig. 3 shows the measured pulmonary flow, and the comparison of the measured and calculated pressure during T_{ej}. p_{PA}^{WK5} from the model describes the "measured" pressure very well, and shows incisure immediately after pulmonary valve closing. Fig. 4 shows the pulmonary input impedance and the values of model parameters that minimize RMSE. The absolute value of Z_{in} shows its minimum value and zero crossing frequency of the phase angle is 5.4 Hz, what is in good agreement with the observations of elderly subjects.

The proposed method is capable to accurately identify PA model parameters and input impedance of pulmonary circulation by using the pressure data from the ejection time window only.

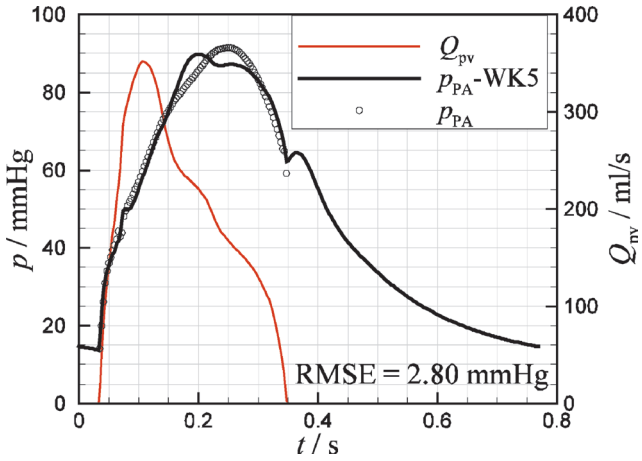

Fig. 3. Velocity through the pulmonary valve (thin red line), "measured" pulmonary root pressure from Eq. (1) (circles), and pulmonary root pressure from the five element lumped model (thick black line)

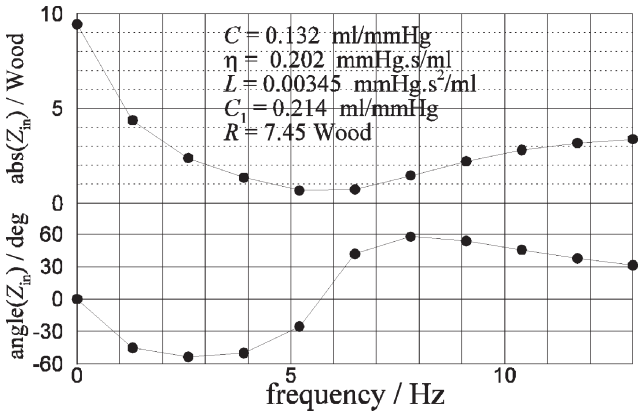

Fig. 4. Absolute value of pulmonary input impedance (upper part) and its phase angle (lower part)

The method is limited to the subject with nicely obtainable tricuspid regurgitant velocity and pulmonary valve flow.

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Mathematical Model of Blood Flow Through the Aortic Valve

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Introduction

In lumped models of circulatory system the aortic flow is governed by unsteady Bernoulli equation. The aortic valve is usually considered as the idealized check valve. This means that the valve opens instantaneously for the positive flow and closes instantaneously preventing the negative (reverse) flow from arteries to the left ventricle. The invasive measurements show that during the valve closing phase the negative flow always occurs. The negative flow cannot be obtained by the idealized valve model. Here we have proposed a model of the aortic valve that could also predict the negative aortic flow.

Mathematical model and numerical method

The characteristic opening and closing phases of the aortic valve are shown in Fig.1. Initially (at the beginning of systole) there is no blood flow through the aortic valve, and the valve leaflets are at rest (panel A in Fig. 1). When the left ventricle (LV) pressure exceeds the arterial pressure (due to LV contraction), the unsteady Bernoulli equation holds

$$M\frac{\mathrm{d}Q}{\mathrm{d}t} = p_{\mathrm{lv}}^{M} - p_{\mathrm{sa}}^{\mathrm{M}} - \frac{K\rho}{2A_{\mathrm{av}}^2}\left(Q - Q_{\mathrm{L}}\right)^2, \qquad (1)$$

where Q is the absolute blood flow through the aortic root, M is inertance coefficient, $\rho = 1050$ kg/m^3 is blood density, A_{av} is the aortic root area, $p_{\mathrm{lv}}^{\mathrm{M}}$ and $p_{\mathrm{sa}}^{\mathrm{M}}$ are measured left ventricle and arterial pressure, respectively, K is minor loss coefficient and Q_{L} is the flow rate that defines the volume V_{L} swept by valve leaflets

$$\frac{\mathrm{d}V_{\mathrm{L}}}{\mathrm{d}t} = Q_{\mathrm{L}}. \qquad (2)$$

During the first opening phase, leaflets move into the arterial space with $Q_{\mathrm{L}} = Q$, but there is no orifice. The orifice occurs after the leaflets have swept a certain volume (see panel B in Fig. 1) $V_{\mathrm{L0}} = \alpha A_{\mathrm{av}}\sqrt{4A_{\mathrm{av}}/\pi}$, where α is the model parameter. During the second opening phase the orifice increases from zero to A_{av}, and leaflets sweep an additional volume (see panel C in Fig. 1) $V_{\mathrm{L1}} = \beta A_{\mathrm{av}}\sqrt{4A_{\mathrm{av}}/\pi}$, where β is the model parameter. In this phase $Q_{\mathrm{L}} = \left(1 - A/A_{\mathrm{av}}\right)Q$, where A is the orifice area which is related to V_{L} as $A = \left[\left(V_{\mathrm{L}} - V_{\mathrm{L0}}\right)/V_{\mathrm{L1}}\right]^2$, see [1]. During these two phases the inertance coefficient is defined as $M = \left(2 - A/A_{\mathrm{av}}\right)M_0$, where $M_0 = \rho L/A_{\mathrm{av}}$ and L is inertance length. After the flow has reached its maximum, the slow leaflets closing phase starts with $Q_{\mathrm{L}} = Q - Q_{\max}A/A_{\mathrm{av}}$. During this phase V_{L} decreases from $V_{\mathrm{L0}} + V_{\mathrm{L1}}$ to V_{L0} (see panels D and E in Fig. 1), and at a certain moment Q becomes negative. For negative Q the inertance coefficients is defined as $M = \delta M_0$, where δ is model parameter. At the end of this phase (for $V_{\mathrm{L}} = V_{\mathrm{L0}}$, see panel E in Fig. 1) leaflets coapt and equation (1) does not hold anymore. Measurements suggest that in the last rapid closing phase (after the leaflets coapt) the leaflets behave as a dumped oscillating system defined by

$$\frac{\mathrm{d}^2Q}{\mathrm{d}t^2} + 2\xi\frac{\mathrm{d}Q}{\mathrm{d}t} + (\omega^2 + \xi^2)Q = 0, \qquad (3)$$

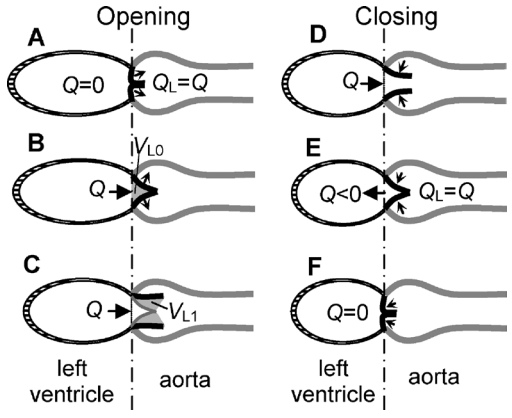

Fig. 1. Scheme of aortic valve opening (panels A to C) and closing (panels D to F)

where ξ and ω are constant parameters. During this phase $Q_{\mathrm{L}} = Q$, and V_{L} should decrease from V_{L0} to zero. If we introduce another parameter $\gamma = \xi/\omega$, parameters ξ and ω are uniquely defined by V_{L0} (or α) and γ.

For the given measured left ventricular and arterial pressures and the set of model parameters: A_{av}, L, K, α, β, γ and δ, the set of equation (1) or (3) and (2) is solved numerically by the fourth order Runge-Kutta method.

Results and conclusions

The proposed model was applied to the measured data in humans and pigs. The measured left ventricle and arterial pressure was used as input, and the calculated aortic flow was compared with the measured ones in Figs. 2 and 3.

There is a very good agreement of the model results with the measured ones, with model parameters in physiological range. Time varying aortic valve orifice A shows two closing phases: a slow one followed by a rapid one as it

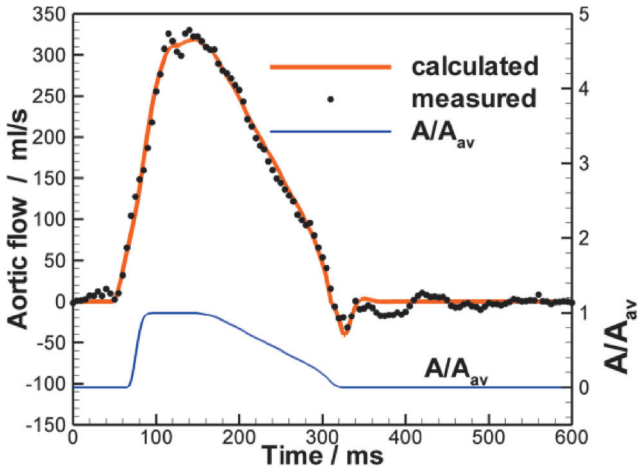

Fig. 2. Model results and measured data [2] in human (model data were $A_{\mathrm{av}} = 2.9$ cm^2, $L = 3$ cm, $K = 1$, $\alpha = 0.15$, $\beta = 0.4$, $\gamma = 0.8$, $\delta = 10$)

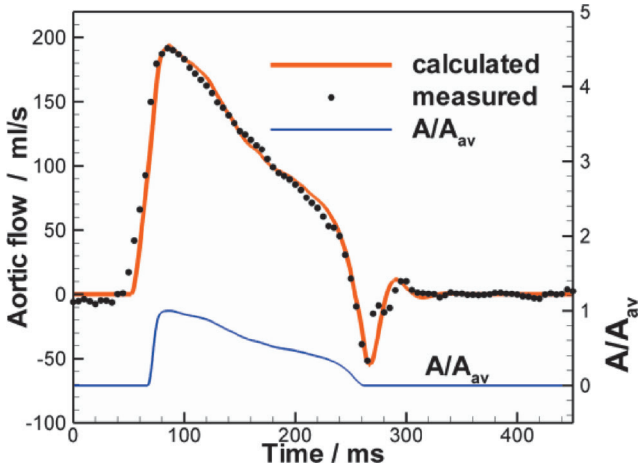

Fig. 3. Model results and measured data [3] in pig (model data were $A_{\mathrm{av}} = 1.6$ cm^2, $L = 1$ cm, $K = 1$, $\alpha = 0.5$, $\beta = 0.6$, $\gamma = 0.5$, $\delta = 3$)

is in experimental observations. The model indicates that the leaflets coapt before the maximal back-flow occurs.

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