Conference on Modelling Fluid Flow (CMFF'22)
The 18th International Conference on Fluid Flow Technologies
Budapest, Hungary, August 30 -September 2, 2022

ONE-DIMENSIONAL MODELLING OF THE ARTERY NETWORK USING THE METHOD OF CHARACTERISTICS WITH A LUMPED HEART

Richárd WÉBER[1], Dániel GYÜRKI[2], György PAÁL[3]

[1] Corresponding Author. Department of Hydrodynamic Systems, Faculty of Mechanical Engineering, Budapest University of Technology and Economics. Bertalan Lajos u. 4 – 6, H-1111 Budapest, Hungary. Tel.: +36 1 463 3484, E-mail: rweber@hds.bme.hu
[2] E-mail: dgyurki@hds.bme.hu
[3] E-mail: paal@hds.bme.hu

ABSTRACT

Since circulatory diseases are leading causes of death in high-income countries, and such disorders often originate from hemodynamic effects, arterial network modelling, which can support diagnostics or medical treatment, is relevant. Human blood circulation can be described using a one-dimensional (1D), distributed parameter model for the arterial network, and lumped models for the heart and peripheral effects. An extended method of characteristics is applied here to solve the momentum and mass conservation equations and the Poynting-Thomson model mimicking the material properties of arterial walls. The heart and the peripheral lumped models are solved with a general zero-dimensional (0D) linear solver handling the boundary conditions, i.e. characteristic equations from the 1D model. The 0D heart model consists of the left chamber and left atrium with mitral and aortic valves. While a capacitance-varying compliance element represents the elastance of the heart, the valves are modelled as check valves with diodes and resistances. A three-element Windkessel model is applied to approximate the effect of the different organs (e.g. kidney, spleen, liver) and peripheries. The model parameters are based on the literature suggestions and by validating the output data from the literature to obtain physiologically relevant results.

artery modelling, lumped heart, method of characteristics, one-dimensional

NOMENCLATURE

D	$[m]$	diameter
E	$[Pa]$	elastic modulus
p	$[Pa]$	static pressure
t	$[s]$	time
u	$[m/s]$	axial velocity
x	$[m]$	spatial coordinate
δ	$[m]$	wall thickness
ε	$[-]$	radial strain
η	$[Pas]$	viscoelastic damping factor
ν	$[m^2/s]$	kinematic viscosity
ρ	$[kg/m^3]$	density

Subscripts and Superscripts

0	nominal values
L	left characteristic point
R	right characteristic point
P	new characteristic point

1. INTRODUCTION

In understanding cardiovascular diseases in modern medicine, numerical simulation of blood flows is relevant. There are two directions of these simulations. Three-dimensional blood flow simulations concentrate on local flow structures in the vessels; many of these are in vessel wall anomalies, like aneurysms or stenoses. The advantage of these simulations is to understand better the small details of the flow and the effects of the malformation. The other direction is a low-dimensional modelling of the whole arterial system. These simulations can model the effect of local illnesses for the entire system or can create boundary conditions for the 3D simulations.

During the last decades, several journal papers were published by our research group on the modelling of the arterial system in one dimension, see, e.g. [1]. This research aims at continuing this low-level modelling approach and extend the modelled cardiovascular system with detailed peripherals, especially the heart. The new model creates more opportunities to analyse circulatory system diseases, such as heart malfunctions or organ failures.

2. MATHEMATICAL MODELLING

2.1. Method of characteristics

The traditional equations (mass balance and momentum) of fluid dynamics are solved, that is, the conservation of mass and momentum. Besides, the arterial wall is considered viscoelastic, described with a Poynting-Thomson model. Overall, Eq. (1) shows the modified mass balance equations.

$$2\frac{\partial \varepsilon}{\partial t} + 2u\frac{\partial \varepsilon}{\partial x} + (2\varepsilon + 1)\frac{\partial u}{\partial t} = 0 \tag{1}$$

The momentum equation is Eq. (2), assuming laminar flow and a circular cross section.

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x} + \frac{1}{\rho}\frac{\partial p}{\partial x} + \frac{32\nu}{D^2}u = 0\,. \tag{2}$$

The equation of the Poynting-Thomson model can be found in Eqs. (3) to (5).

$$\frac{pD_0}{2\delta_0}(2\varepsilon + 1) = E_1\varepsilon_1 \tag{3}$$

$$\frac{pD_0}{2\delta_0}(2\varepsilon + 1) = E_2\varepsilon_2 + \eta_2\frac{d\varepsilon_2}{dt} \tag{4}$$

$$\varepsilon = \varepsilon_1 + \varepsilon_2 \tag{5}$$

While the "1" notation stands for the linear element of the Poynting-Thomson, the "2" indicates the viscoelastic property. The partial differential equations (PDE) are traditionally solved by the finite volume method in fluid mechanics, or nowadays, the finite element method is also applied. However, during this research, the method of characteristics has been used that transforms the PDEs to ordinary differential equations (ODE) along the characteristic lines. The ODE system can be transformed to algebraic equations with the explicit Euler technique, see Eqs. (6) and (7). The detailed description of the transformation can be found in [1].

$$v_P - v_L + \frac{1}{\rho a_L}(p_P - p_L) = -\Delta t J_L \tag{6}$$

$$v_P - v_R - \frac{1}{\rho a_R}(p_P - p_R) = -\Delta t J_R \tag{7}$$

The J_L and J_R are the source terms containing the geodetic height differences, the laminar pressure loss, and the viscoelastic vessel wall effect. The two unknown variables are at the P level, since every quantity is known at the L and R time level. For notations, see Fig. 1. The time step Δt is predefined by the characteristic lines, depending on the vessel's exact geometry (mainly the length) and the number of inner calculation points. Since a real arterial system contains numerous vessels, the common time step is the minimum amongst all the inner time steps.

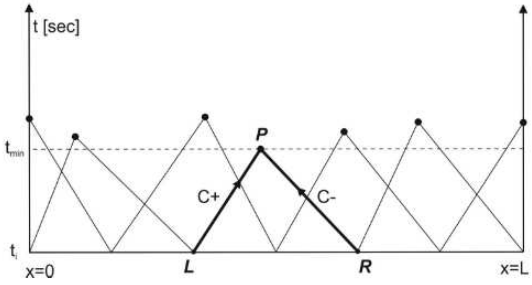

Figure 1. The method of characteristic [1].

Boundary conditions must be defined at each end of an edge, as there is only one characteristic line, and additional information is required from the surrounding of the vessel. For the inner nodes in the arterial system, see Fig. 3 white points, the continuity equation is solved. The peripherals and the heart are handled with lumped, zero-dimensional models.

2.2. Lumped Solver

As there are different models required to mimic the behaviour of different organs, such as the heart or the peripherals, a general lumped or zero-dimensional (0D) solver is relevant. Table 1 shows the type of elements a 0D model might include. All the components are linear, and either an algebraic equation or an ODE must be solved. The ODEs are transformed to algebraic ones using the explicit Euler formula.

Table 1. Elements of the 0D model with their continuous equation and parameter

Type	Parameter	Equation
Resistor	R	$\Delta p = Rq$
Capacitor	C	$q = C\frac{d\Delta p}{dt}$
Elastance	E_{min}, E_{max}	$q = \frac{d\left(\frac{\Delta p}{E(t)}\right)}{dt}$
Inductor	L	$\Delta p = L\frac{dq}{dt}$
Diode	R	$\Delta p = Rq$ and $q > 0$
Battery	V	$\Delta p = V$
Node	-	$\sum_{in} q_i - \sum_{out} q_i = 0$
Ground	-	$p = 0$

A speciality is the elastance for modelling the effect of the heart contraction. The elastance is the function of time, and it describes the pressure-volume relationship of the left chamber. It is general, as it is valid for every healthy adult regardless its age, weight or living habits [2]. Fig. 2 depicts the shape

as a function of time. There are only two parameters: the peak E_{max} and the minimum E_{min} [3].

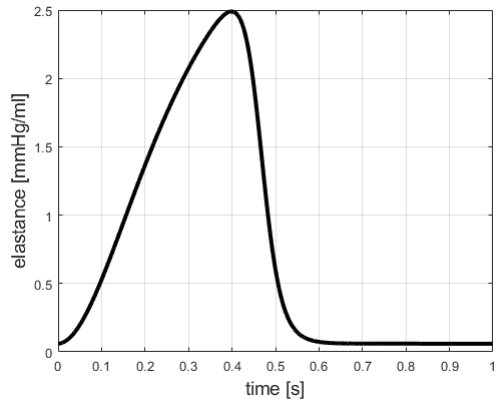

Figure 2. The time history of the elastance.

Although the elastance seems mathematically convenient: continuous, smooth and also differentiable multiple times, numerical instabilities occur during the solution of its equation (see Tab. 1). The appearance of the dE(t)/dt over E(t) lies behind this issue as the minimum value of E(t) is low, while the derivative can be high, especially at the beginning of the cycle. The matrix of the linear equation system would have a high conditional number, making it numerically unstable. One possible solution is to eliminate this term by introducing a new variable, that is $y=\Delta p/E$, as in [3].

The process of the combined 1D-0D solver is briefly the following. First, every inner point and inner boundaries are calculated in the 1D model, also the time step is determined. Second, the neighbouring characteristic equations from the 1D are added to the corresponding 0D equations and are solved together with 0D elements in one step.

3. MODEL BUILDING

3.1. Topology

The topology of the arterial system is based on the system presented in [4]. Beside the main arterial vessels, the brain is introduced in detail, see Fig. 3 top side. The circle of Willis is considered with most of the neighbouring arteries. Moreover, the spine and the arteries of the organs around the spine are also part of the model. The heart and the peripherals are built using 0D models according to [4,5], see Fig. 4.

The standard, three-element Windkessel model is applied for mimicking the peripheral effects of the arterial system. Although the structure of the heart is more complex, the mitral and aortic valves are modelled with an ideal diode and a resistance in series. While the diode serves the role of the check valve effect, i.e. it does not allow any backflow, the resistor causes the pressure loss at the valve. The two inductors mimic the inertia of the fluid. The left atrium pressure is prescribed by a battery or "voltage" component. In low-dimensional modelling, it is an acceptable approximation to keep the left atrium pressure constant. The elastance, i.e. a capacitor with variable parameters, is responsible for mimicking the contraction effect of the heart. Interestingly, it is the same function in time for all patients regardless of their age, weight or race.

3.2. Parameter Identification

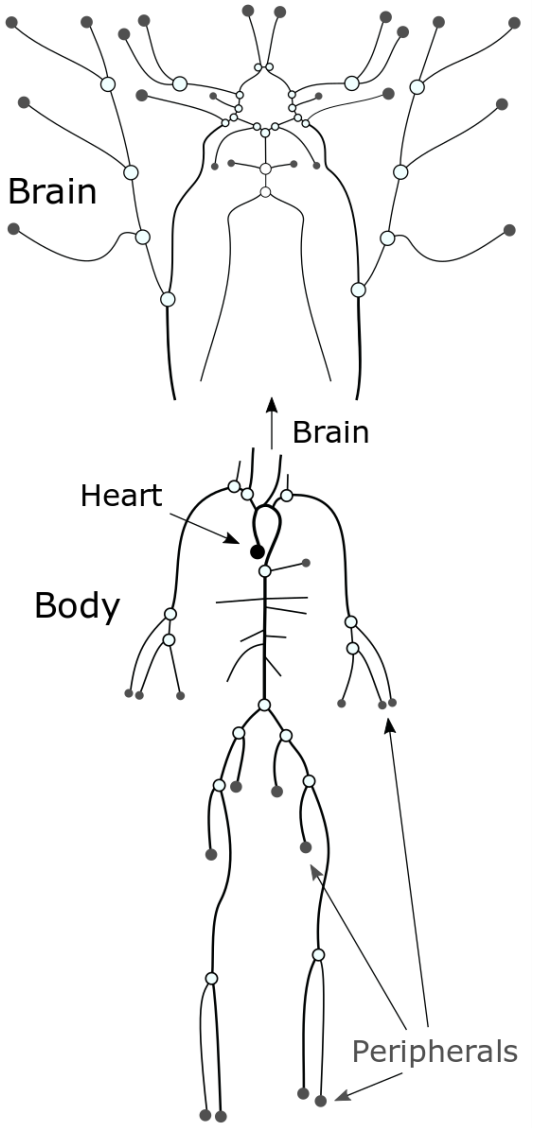

Figure 3. Main body and brain arteries. [4]

The biggest challenge in building a proper model for describing the arterial system is not the topology but determining the countless parameters. Each artery has five geometrical (length, proximal and distal diameter, proximal and distal thickness), three material wall parameters (two elasticities, damping factor), and the number of inner division points in the nine parameters. Moreover, the peripherals are modelled with a three-element Windkessel model; there are three independent parameters. Finally, the

heart is built using two resistors, one capacitor, one elastance and two inductors; overall, it means seven independent parameters. There are 99 arteries and 44 peripherals, meaning the number of all parameters is 99*9+44*3+7=1030. The question is how to choose these to have physiologically relevant outputs?

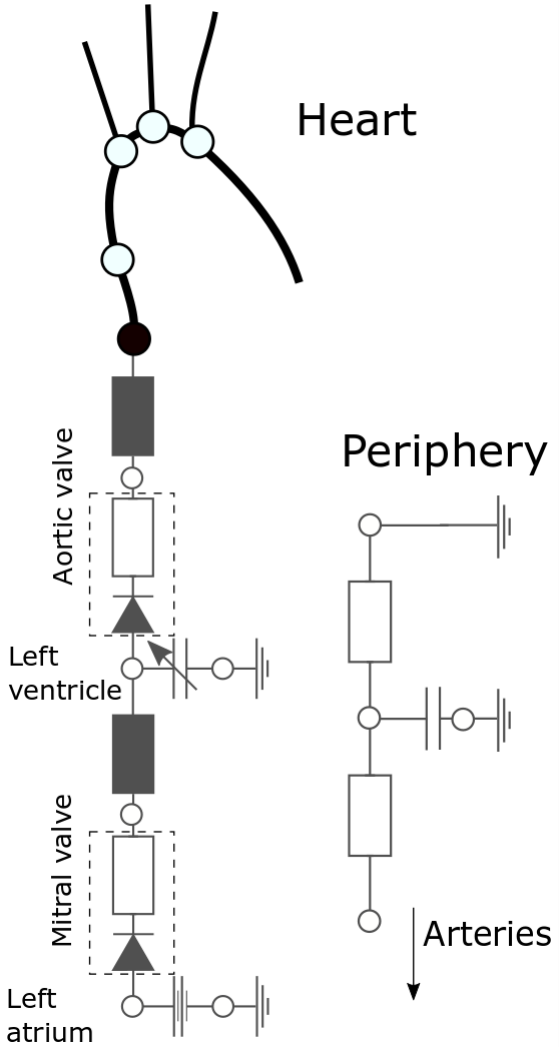

Figure 4. Lumped models: heart and three-element Windkessel at peripherals.

With the help of imaging tools (e.g. CT or MRI), physicians can measure the geometrical parameters with high accuracy. The data found in [4] are used in the current research. Since the literature barely contains information about the wall thickness, it is set to 10% of the diameter for all vessels. The value of the elasticities is critical, as it determines the wave propagation speed (Eq. (8)), thus the pulse wave velocity (PWV) also.

$$a_0 = \sqrt{\frac{E_1 \delta_0}{\rho D_0}} \tag{8}$$

[6] presented a study recently about the measured PWV values from the literature as a function of the nominal (or lumen) diameter. A fitted approximation was used to keep the physiologically proper PWV values (see Fig. 5). The equation mathematically is a power-law function, which was also proposed in [6]. On the one hand, from the Eq. (8) and the definition of the pulse wave velocity, one elasticity value can be calculated; on the other hand, the elasticity parallel to the damping E_2 is set to 1.8 times of E_1 according to previous studies [1].

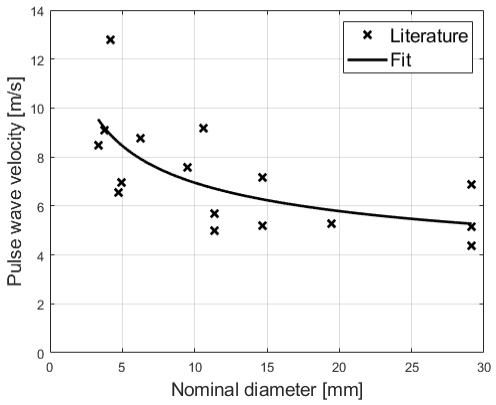

Figure 5. Pulse wave velocity as a function of lumen diameter in the literature from [6] and a fitted power-law function.

The number of division points along a vessel can be determined by manually tuning and checking the average time step values in each vessel. The primary purpose is to accurately approximate the governing fluid dynamical equations, but a secondary goal is to obtain a uniform time step distribution. The lowest value determines the common time step of the model; thus, an exceptionally low time step might inhibit the computational efficiency. The time step is influenced primarily by the length of the artery, the number of division points and the PWV. Since the PWV is typically in the range of 5 to 9 *m/s*, see Fig. 5, it can be assumed to be equal for all vessels at this point. Overall, the proportion of the division points and the length should be uniform, and the sum of the division points is decreased until the approximation error is negligible.

Determining the parameters of the lumped models, such as the heart or the peripherals, is critical and challenging. None of the parameters can be directly measured or estimated. The only suitable method here is tuning the parameters to have a physiologically proper output, e.g. aortic pressure or cardiac output. The exact values for this research is coming from [4] for the peripherals, while the heart parameters are from [2].

4. RESULTS

One of the most crucial hemodynamic quantities in low-dimensional fluid mechanical modelling is the volume flow rate of the heart in time. Fig. 6 depicts the results from the simulation and the literature [6]. The first peak indicates the heart contracting and releasing blood from the left

ventricle. After the peak, it decreases back to zero, but as the signal from the literature suggests, it might undershoot, causing a reverse flow rate or backflow. The simulation cannot catch such details as it is built using perfectly operating diodes (or check valves).

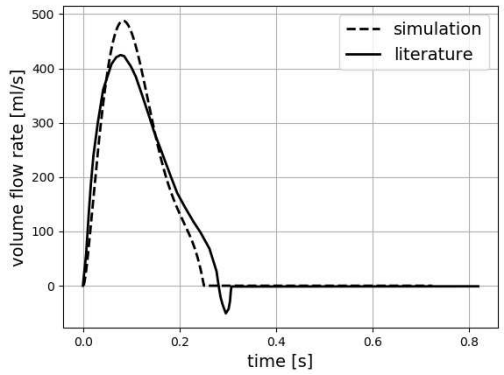

Figure 6. Volume flow rate of the heart from the literature [6] and the simulation over time.

Another relevant and diagnostically important physiological output is the aortic pressure; see Fig. 7 for the results from the simulation and the literature. Although the overall shape is similar and the amplitude is in the same range, the distal and proximal values are not well approximated by the simulation.

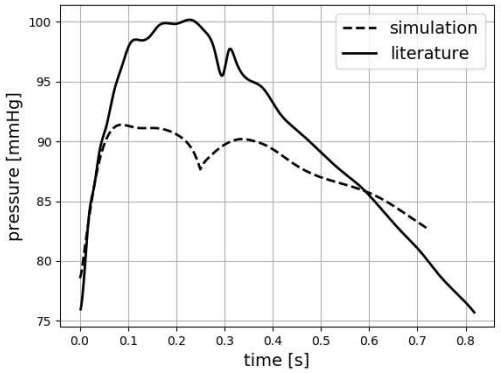

Figure 7. Aortic pressure in time from the literature [6] and the simulation over time.

Similar observations can be drawn based on Fig. 8, which indicates the femoral pressure. The general nature of the signals is identical; however, the diastolic and systolic values differ. Fig. 9 depicts the velocity from the exact location. Slightly more significant differences appear as the simulation constantly underestimates the velocity.

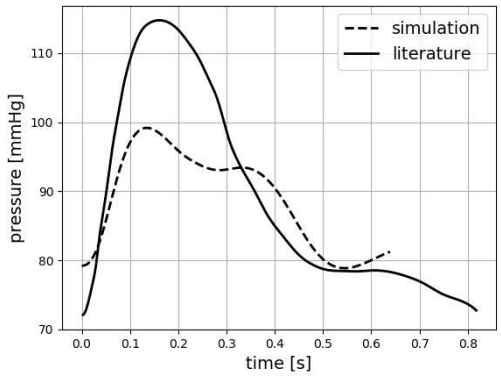

Figure 8. Femoral pressure in time from the literature [6] and the simulation over time.

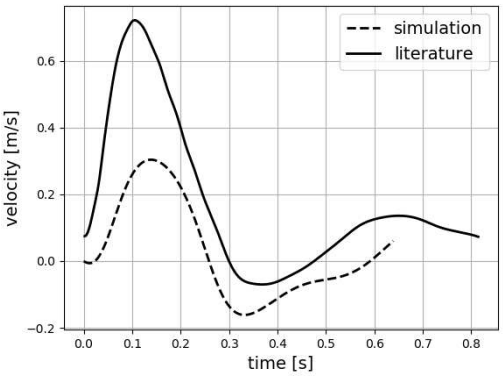

Figure 9. Femoral velocity in time from the literature [6] and the simulation over time.

5. DISCUSSION

The prediction of the proposed model for the pressure and volume flow rates is qualitatively accurate. However, to increase the precision of the model, some additional parameter calibration is inevitable. Even though the model is low-dimensional with low computational time, it contains more than one thousand independent parameters; thus, the question is how to decrease their number. Recently [7] proposed a grouping based on the location of the arteries, then assigned the same multiplier factor to each parameter within the group. The question is whether this (or any other) grouping technique reduces the size of the search space, i.e. it might decrease the accessibility of the optimal solution.

The flow rate of the heart gives an excellent approximation to the literature data; however, catching the reverse flow is not possible with the current model due to the ideal check valves. One possible solution is introducing a hysteresis to the diodes modelling the heart valves. Again, how to choose the parameters correctly to achieve the physiologically relevant solution emerges. The direct

measurement of this phenomenon is not possible, and also, the literature lacks modelling such features.

Considering the femoral outputs, the exact location of the signals is a question. The femoral artery is one of the longest vessels in the human body; it can be over 400 mm. The location of the literature data and the simulation may differ, causing some additional discrepancy. Further analysis should find the exact location of such data or designate points which position is precisely prescribed.

The combined, general 1D-0D solver is the most valuable result of this research, as it creates numerous opportunities. Still, the model can be extended with additional 0D elements of the cardiovascular system. Recently [8] introduced a 0D model to describe the cardiopulmonary circulation. Moreover, a lumped model was proposed in [9] for modelling the venous system. Such parts of the circulatory system can be easily added to our model as the 1D-0D solver in general.

6. SUMMARY

This research introduced a 1D-0D modelling approach for the arterial system, the heart and the peripherals. The main arterial branches are resolved in 1D, and the governing fluid mechanical equations are solved using the method of characteristics. The material of the vessels is considered viscoelastic and described with the Poynting-Thomson model. The heart and the peripherals are handled with 0D models. The critical point is to set the model parameters correctly, which are set based on the data from the scientific literature.

The overall quality of the simulation data is acceptable, as it gives a satisfactory approximation of literature results, even without additional calibration of the parameters. Further investigation is necessary in the direction of input parameter calibration, which carries significant uncertainties (e.g. peripheral resistances, elasticities or heart parameters). Moreover, the model extensions are available, as the combined 1D-0D solver is general, and any model topology can be accurately solved. Pulmonary, venous or coronary circulation elements can be added in further research.

ACKNOWLEDGEMENTS

Project no. NKFI-K129277 (“Evaluation of cerebrovascular events in patients with occlusive carotid artery disorders based on morphological and hemodynamic features”) has been implemented with the support provided by the Ministry of Innovation and Technology of Hungary from the National Research, Development and Innovation Fund.

The project was also funded by the National Research, Development, and Innovation Fund of Hungary under Grant TKP2021-EGA-02.

REFERENCES

[1] Bárdossy, G., Halász G. 2013, „A " backward" calculation method for the estimation of central aortic pressure wave in a 1D arterial model network”, *Computers and Fluids*, Vol. 73, pp. 134-144

[2] Ferreira, A., Chen S., Simaan M., et al., 2005, „A nonlinear state-space model of a combined cardiovascular system and a rotary pump”, *CDC-ECC '05*, Vol. 1, pp. 897-902

[3] Gul R., Shahzadi S., 2019, „Beat-to-beat sensitivity analysis of human systemic circulation coupled with the left ventricle model of the heart: A simulation-based study”, *European Physical Journal Plus*, Vol. 134., pp.1-23

[4] Reymond, P., Merenda, F., Perren, F., et al., 2009, “Validation of a one-dimensional model of the systemic arterial tree”, *American Journal of Physiology - Heart and Circulatory Physiology*, Vol. 297, pp. 208-222

[5] Kim, H., Vignon-Clementel, I., Coogan J., et al., 2010, „Patient-specific modeling of blood flow and pressure in human coronary arteries”, *Annals of Biomedical Engineering*, Vol. 38, pp. 3195-3209

[6] Charlton, P., Harana, J., Vennin, S., et al., 2019, “Modeling arterial pulse waves in healthy aging: a database for in silico evaluation of hemodynamics and pulse wave indexes”, *American Journal of Physiology - Heart and Circulatory Physiology*, Vol. 317, pp. H1062-H1085

[7] Jones, G., Parr, J., Nithiarasu, P., et al. 2021, “A physiologically realistic virtual patient database for the study of arterial haemodynamics”, *International Journal for Numerical Methods in Biomedical Engineering*, Vol. 37, pp. 1-28

[8] Li, B., Wang, H., Li G., et al. 2020, “A patient-specific modelling method of blood circulatory system for the numerical simulation of enhanced external counterpulsation”, *Journal of Biomechanics*, Vol. 111, pp. 1-12

[9] Zhang, H., Fujiwara, N., Kobayashi, M., et al. 2016, „Development of a Numerical Method for Patient-Specific Cerebral Circulation Using 1D–0D Simulation of the Entire Cardiovascular System with SPECT Data”, *Annals of Biomedical Engineering*, Vol. 44, pp. 2351-2363