

SIMULATION OF CONTRAST MEDIUM PROPAGATION BASED ON 1D AND 3D PORTAL HEMODYNAMICS

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Abstract: *The mathematical modelling of 3D blood flow coupled with contrast medium propagation in patient-specific human vessels ranks among the most computationally-demanding tasks, especially if a large vessel network is considered. With this in mind, some measure of simplification for potential use in clinical practice is usually inevitable and often also desired. In the present study, the main interest lies in the quantitative comparison between 3D and 1D models of portal hemodynamics and contrast medium propagation in two hepatic portal vein networks reconstructed from CT scans provided by the courtesy of the University Hospital Pilsen. To approximate the flow resistance of the downstream vascular bed and hepatic tissue, the mathematical model of Newtonian blood flow is coupled with the three-element Windkessel model. The numerical simulations of 3D and 1D blood flow and contrast medium propagation under average flow conditions are carried out using own in-house software. The obtained results show that, although the 1D model can never completely imitate the computational capabilities of the 3D model, its easy implementation, time-saving model preparation and almost no demands on computer technology dominate as advantages over obvious but moderate modelling errors arising from the dimensional reduction.*

Keywords: Patient-specific model, Blood flow, Contrast medium, Finite volume method, Windkessel model.

1. Introduction

In the last two decades, medicine has experienced a boom in the field of computer-aided imaging methods. Now the efforts of the bioengineering community are directed toward computational software that would aid surgeons during difficult surgeries or help them find an optimal surgical solution that would increase the chance of complete recovery for the patient. Compared to the numerical simulations performed in industry, where a computation may take days or even weeks, the clinical practice, however, requires results within a short time period and with minimal computational demand. With such strict requirements in mind, a development of clinical software is not easy and it is only natural that some model simplifications are usually inevitable.

This study, which is focused on quantitative comparison between 3D and 1D models of blood flow and contrast medium propagation in realistic hepatic portal vein networks, is a result of research performed at the University of West Bohemia in close co-operation with the University Hospital Pilsen. The main objective of this co-operation is the development of software for microstructurally-oriented multiscale modelling of tissue perfusion (Rohan et al., 2012), which would accelerate liver resection and make it more exact. In this regard, it is crucial to understand the impact of model simplification on the overall simulation accuracy, especially if the importance of blood supply to each part of the liver through the hepatic portal vein is taken into account. Fig. 1 shows selected preliminary data of contrast medium propagation in a patient-specific liver model, where the networks of the hepatic portal vein and hepatic veins are modelled by means of the 1D model.

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The main objective of this study is to determine the differences between the 3D and 1D network models by adopting the assumptions of impermeable and inelastic vessel walls and blood as an incompressible Newtonian fluid with density of 1050 kg/m^3 and dynamic viscosity of $0.00345 \text{ Pa}\cdot\text{s}$.

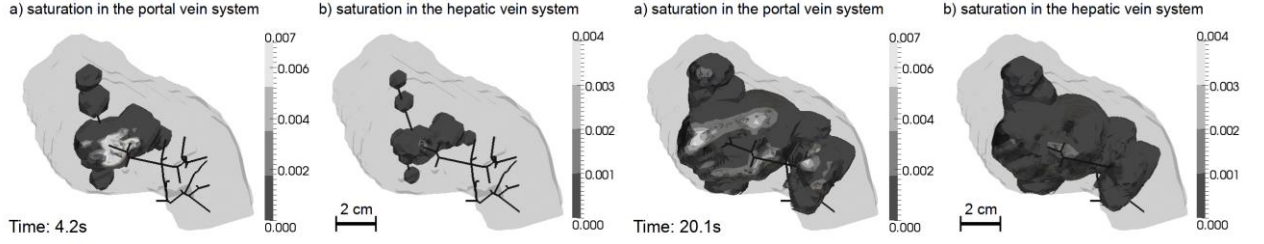


Fig. 1: Example of contrast medium propagation in patient-specific model of human liver at two selected time instants: $t_1 = 4.2 \text{ s}$ (left) and $t_2 = 20.1 \text{ s}$ (right). The results, which are based on the modeling approach introduced in (Rohan et al., 2012), show the local concentration of the contrast medium in the: a) portal and b) hepatic vein systems of the hepatic tissue. Here, the vessels involved in the tissue perfusion and contrast medium propagation are represented by 1D venous networks (black lines).

2. Methods and Results

2.1. Patient-specific hepatic portal vein networks

In this study, the numerical simulations of blood flow and contrast medium propagation are carried out in two realistic portal vein geometries with different levels of complexity (9 outlets vs. 39 outlets), Fig. 2 (left). Both models are reconstructed from CT scans provided by the courtesy of the University Hospital Pilsen. For the reconstruction process, we employ the semi-automatic segmentation software DICOM2FEM (Jiřík & Lukeš, 2013), which is currently being developed at the University of West Bohemia, in combination with the well-known Taubin smoothing algorithm. The computational meshes for the two 3D hepatic portal vein models are generated with the help of the software package HyperMesh v11.0 (Altair Engineering, Troy, USA). The number of tetrahedral cells required to model the simple and complex venous networks is 816,547 and 2,042,156, respectively.

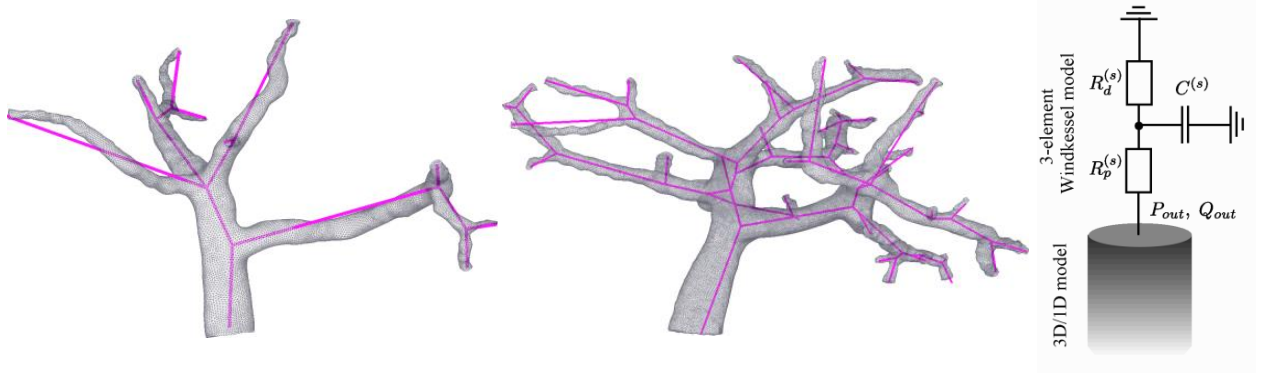


Fig. 2: Left – 1D and 3D reconstructions of simple (9 outlets) and complex (39 outlets) hepatic portal vein networks. Right – The three-element Windkessel model as an outflow boundary condition. For more details on the principle of this lumped model, see, for example, (Westerhof et al., 2009).

2.2. Blood flow in 3D and 1D models

For the description of the blood flow in the 3D venous networks, the non-linear system of Navier-Stokes equations for an incompressible Newtonian fluid is used

$$\frac{\partial v_j}{\partial x_j} = 0, \quad (1)$$

$$\frac{\partial v_i}{\partial t} + \frac{\partial}{\partial x_j} (v_i v_j) + \frac{1}{\rho} \frac{\partial p}{\partial x_i} = \frac{1}{\rho} \frac{\partial^2 v_i}{\partial x_j \partial x_j}, \quad i, j = 1, 2, 3, \quad (2)$$

where t is the time, v_i is the i -th component of the velocity vector \mathbf{v} corresponding to the Cartesian component x_i of the space variables vector \mathbf{x} , p is the pressure, and ρ is the density. The mathematical model is numerically solved using our own computational algorithm based on a stabilised variant of the projection method in combination with the cell-centred finite volume method formulated for hybrid unstructured tetrahedral grids. The principle of this algorithm, which we have successfully implemented for the solution of various hemodynamical problems in the past, is described in (Vimr et al., 2013).

Compared to the 3D flow problem, we assume the blood flow in one inelastic segment of the 1D venous network to be governed by the continuity equation and the Bernoulli equation

$$A_{i-1}u_{i-1} = A_i u_i, \quad (3)$$

$$\frac{1}{2}\rho u_{i-1}^2 + p_{i-1} = \frac{1}{2}\rho u_i^2 + p_i + e_i^{\text{loss}}, \quad (4)$$

where A_i is the cross-sectional area of the i -th segment and u_i is the velocity in this segment. To approximate the losses originating from the viscous resistance, the Bernoulli equation (4) is completed with the term e_i^{loss} corresponding to friction loss in inelastic tubes, which is known to be proportional to the local velocity magnitude. In general, the description of blood flow in a 1D venous network results in a system of non-linear algebraic equations, which is solved with the help of the Newton method.

The numerical simulations of 3D and 1D blood flow are carried out for steady boundary conditions. At the inlet of the hepatic portal vein, we prescribe an average physiological velocity of 0.325 m/s. Because of the difficulties associated with clinical determination of physiological pressure in portal vein networks, each outlet of the two models considered in this study is coupled with a well-known lumped model – the three-element Windkessel model, schematic drawing of which is shown in Fig. 2 (right). Compared to other modelling approaches such as the prescription of one constant outlet pressure, the Windkessel model is able to approximate the flow resistance of the downstream vascular bed and to provide a physiological value of pressure at all network outlets. For one outlet, the Windkessel model is mathematically described by the following two equations for unknown pressures p_d and p_o :

$$\frac{d}{dt}p_d(t) + \frac{p_d(t)}{CR_d} = \frac{1}{C}Q_o(t), \quad p_o(t) = p_d(t) + R_p Q_o(t), \quad (5)$$

where p_o and Q_o are the pressure and flow rate determined at this specific outlet of the 3D/1D models and p_d is the distal pressure representing the pressure in arterioles and capillaries of the downstream vascular bed. Note that the remaining parameters known as the lumped parameters of proximal R_p and distal R_d resistance and capacitance C have to be calculated for each outlet prior to the numerical simulation.

Selected outflow results for the simple and complex venous networks are listed in Tab. 1 with corresponding cross-sectional areas (CSA) and absolute Δ [ml/s] and relative errors σ [%] defined as

$$\Delta = |Q_{3D} - Q_{1D}|, \quad \sigma = \frac{|Q_{3D} - Q_{1D}|}{Q_{3D}} \cdot 100\%, \quad (6)$$

where Q_{3D} and Q_{1D} are the flow rates in the 3D and 1D models, respectively. In general, it is possible to say that the flow rates computed with the 1D blood flow model sufficiently approximate the ones determined using the 3D model.

Tab. 1: Overview of selected outlet results for the simple (left) and complex (right) portal vein networks. Here, the abbreviation CSA stands for cross-sectional area of the outlet.

outlet No.	CSA [mm ²]	abs error Δ [ml/s]	rel error σ [%]	outlet No.	CSA [mm ²]	abs error Δ [ml/s]	rel error σ [%]
1	6.23	0.63	12.95	1	1.39	0.21	15.32
3	6.67	0.57	8.85	9	1.51	0.09	4.54
5	8.36	0.79	12.01	10	1.46	0.24	12.10
6	4.97	0.34	7.11	20	1.03	0.05	6.64
9	4.15	0.32	9.77	36	1.92	0.10	3.52

2.3. Contrast medium propagation

For the purpose of this study, the propagation of the contrast medium dissolved in the blood and transported through the venous network is modelled in analogy to the mathematical model introduced in Rohan et al. (2012). Taking into consideration only the convection of the contrast medium, the local concentration of the tracer expressed by the saturation S is described as

$$\frac{\partial S}{\partial t} + \frac{\partial}{\partial x_j}(S v_j) = 0, \quad (7)$$

where t is the time. The numerical solution of Eq. (7) in the 3D portal vein model is based on the upwind cell-centered finite volume scheme formulated for unstructured tetrahedral grids in combination with the two-stage Runge-Kutta method of second order accuracy in time. To simulate the propagation of the contrast medium based on the portal hemodynamics computed previously, we apply an external source saturation given in the form of a time bolus. Selected numerical results for the simple venous network are shown in Fig. 3. From the graph in Fig. 3 (right), it can be noted that the difference between the 1D and 3D outlet tracer rates is non-zero (22% at $t = 2.5$ s) because of tracer accumulation near the walls of the 3D model.

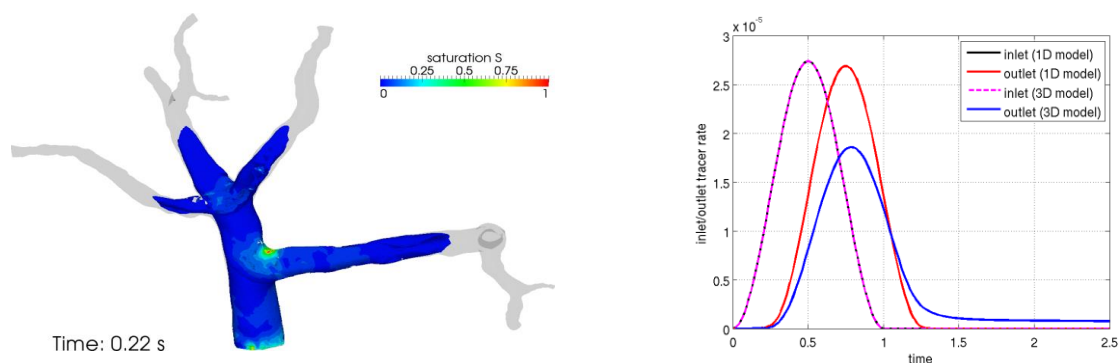


Fig. 3: Left – Propagation of the contrast medium in the 3D model with 9 outlets at the time $t = 0.22$ s. Right – Time development of inlet and outlet tracer rates in the simple 3D and 1D venous networks.

3. Conclusions

Comparing not only the results obtained for both the 3D and 1D models, several advantages and disadvantages of each modelling approach can be noted. First of all, there is the matter of model preparation: Before a 3D venous network can be used for any numerical simulation, quite many time-consuming preparation steps are necessary (reconstruction from CT scans, geometry 'cleaning', remeshing etc.), whereas the 1D network requires only one step – the 'cleaning' (removal of non-anatomical branches and loops). Another issue is the computational demand of the 3D and 1D models: In the case of the 1D network, the simulation of blood flow and contrast medium propagation takes only seconds to complete and requires no special computer technology. On the other hand, the 3D simulation is computationally very demanding even assuming up-to-date computer technology is available. Thus, despite the existing result differences between both models, the benefits of the 1D approach clearly outweigh its slight inaccuracy.

Acknowledgement

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