BUILDING COUPLED 3D–1D–0D MODELS IN COMPUTATIONAL HEMODYNAMICS

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ABSTRACT

Nowadays, in the computational hemodynamics field there is an increasing need for reproducing accurately physiological blood flow regimes encountered in the cardiovascular system, as well as to simulate coupled global/local phenomena, with the purpose of retrieving as much information as possible from the numerical simulations. In this context, the present work is concerned with the variational derivation of the so-called geometrical multi-scale models which provide mainly two advantages aligned to the needs identified above, giving rise to the 3D–1D–0D coupled models. Also, two examples of application to show the potentialities of these dimensionally-heterogeneous coupled models in computational hemodynamics are provided.

Key Words: computational hemodynamics, multi-scale modelling, variational approach, 3D–1D–0D models, physiological regimes.

1 INTRODUCTION

In last years, numerical simulation have played a main role in the field of computational hemodynamics. Several approaches can be identified in this field. We can work, on one hand, with simplified mathematical models to model the global behavior of the arterial system [1,6]. On the other hand, full three-dimensional simulations help researchers to analyze the local behavior of blood flow [3,8].
With these two alternatives at hand, other questions emerged for which 3D or 1D standalone models might not give accurate answers. Studying the interaction between local and global phenomena under real physiological regimes led researchers to integrate simple and complex representations. Thus, coupled 1D and 3D models yielded new mathematical challenges and new possibilities [2, 4, 5, 9]. Specifically, in this work we present a consistent variational derivation of a 3D–1D–0D multi-scale model of the cardiovascular system taking into account for the local circulation (3D), global circulation (1D) and peripheral circulation (0D). Then, we use the coupled model in applications to show the potentialities of the technique. Section 2 presents some remarks about the use of coupled models, as well as the mathematical formulation. Section 3 is devoted to the applications. Final comments are drawn in Section 4.

2 MATHEMATICAL FORMULATION

A multi-scale approach to this problem is able to couple several elements of interest in the analysis: (i) all the complexity of 3D blood flow circulation in complex arterial districts such as bifurcations, tortuous vessels, valves among others; (ii) all the complexity of the systemic response such that, for a given heart beat (input) we obtain the conformation of the cardiac pulse (the output); (iii) all the influence of peripheral beds, taking into account the peripheral resistance and compliance that determines the overall state of the arterial network as well as rules the blood flow distribution. In what follows the multi-scale model of the arterial tree will be developed (see [2] for more details).

Let a domain \( \Omega \) split into three parts \( \Omega = (\Omega_0 \cup \Omega_1 \cup \Omega_3)^c \) through two coupling interfaces (\( \Gamma_{01} \) and \( \Gamma_{13} \)) for the coupling between the 0D and 1D, and the 1D and 3D respectively. The fluid velocity and pressure are denoted by \((u_0, p_0)\), \((u_1, p_1)\) and \((u_3, p_3)\) for the 0D, 1D and 3D correspondingly. The kinematical hypotheses considered for each sub-domain are the following \( u_0 = u_0 e_2, u_1 = u_1(z) e_2 \) and \( u_3 = u_3(x) \) where \( e_2 \) is the unit axial vector. As a consequence, it is \( p_0 = p_0, p_1 = p_1(z), p_3 = p_3(x) \). Over the lateral boundary of \( \Omega_3 \) we consider adherence of the fluid flow, while in its free outlet we take homogeneous Neumann boundary conditions. Over the inlet of \( \Omega_0 \) we consider a Neumann boundary condition \( p_i \). The blood is a Newtonian fluid and the area in the 0D and 1D models is denoted by \( A_0 \) and \( A_1 \) respectively. The length of the 0D model is \( L_0 \), and the 1D interval is \( I_1 \).

The extended variational principle is the following: find \((u_0, u_1, u_3), (p_0, p_1, p_3), t_{01}, t_{13}\) \( \in U_d \times P_d \times T_d' \) such that

\[
\begin{align*}
(A_0 \rho L_0 \frac{du_0}{dt} + 8 \pi \mu L_0 u_0) v_0 + \int_{I_1} \left[ A_1 \rho \frac{\partial u_1}{\partial t} v_1 + A_1 \rho u_1 \frac{\partial u_1}{\partial z} v_1 - A_1 p_1 \frac{\partial u_1}{\partial z} - p_1 \frac{\partial A_1}{\partial z} v_1 + 8 \pi \mu u_1 v_1 \right] dz \\
+ \int_{\Omega_3} \left[ \rho \frac{\partial u_3}{\partial t} v_3 + \rho (\nabla u_3) u_3 \cdot v_3 - p_3 \text{div} v_3 + 2 \mu \varepsilon (u_3) : \varepsilon (v_3) \right] dx = \\
- A_0 p_0 t_{01} + t_{01} (v_0 - v_1) \bigg|_{t_{01}} - s_{01} (u_0 - u_1) \bigg|_{t_{01}} \\
+ t_{13} (v_1 - \frac{1}{\mu} \int_{\Gamma_{13}} v_3 \cdot e_3 d\Gamma) \bigg|_{t_{13}} + s_{13} (u_1 - \frac{1}{\mu} \int_{\Gamma_{13}} u_3 \cdot e_3 d\Gamma) \bigg|_{t_{13}} \\
\forall ((v_0, v_1, v_3), s_{01}, s_{13}) \in V_d \times T_d',
\end{align*}
\]

with proper initial boundary conditions and \( A_1|_{\Gamma_{13}} = A|_{\Gamma_{13}} \). Note that \( p_0 \) is not a known value in the 0D model, but the pressure drop, that is \( A_0 \Delta p_0 = t_{01} - A_0 p_i \). Also, \( U_d = \{(u_0, u_1, u_3) \in \mathbb{R} \times H^1(I_1) \times \mathbb{R}\}; u_{3|\Gamma_{13}} \) satisfies \( \text{b.c.} \), \( P_d = L^2(I_1) \times L^2(\Omega_3) \), \( V_d \) is the space associated to the linear manifold \( \mathcal{U}_d \), and \( T_d' = \mathbb{R} \times \mathbb{R} \). The area \( A_1 \), and the domain \( \Omega_3 \) are unknowns, so problem (1) is closed with the following constitutive relations for the arterial walls

\[
\begin{align*}
p_1 &= p_r + \frac{E \pi R_h e_2}{A_1} \left( \sqrt{\frac{A_1}{A_r}} - 1 \right) + \frac{E \pi R_h e_2}{2 \sqrt{A_1 A_1}} \frac{\partial A_1}{\partial t} & \text{in } I_1, \\
p_3 &= p_r + \frac{E h e_2}{\tau_3} s_3 + \frac{k_h e_2 \partial s_3}{\tau_3} & \text{over } \Gamma_{L3},
\end{align*}
\]
where \( r \) indicates reference values, \( E \) is the Young modulus, \( k \) the viscoelastic coefficient, \( R \) the radius of curvature, \( h \) the thickness, \( A_1 \) the area of the 1D domain and \( \zeta_3 \) the displacement of the arterial wall in the direction of the normal \( n_3 \) in the 3D model over \( \Gamma_{L,3} \). Then, the velocity over \( \Gamma_{L,3} \) is obtained by doing \( u_3|_{\Gamma_{L,3}} = \frac{\partial \zeta_3}{\partial t} n_3 \). For the derivation of the Euler–Lagrange equations and for the approximation of the problem see [9].

3 EXAMPLES OF APPLICATION

In all the examples presented here we make use of the 1D model given in [1] and the heart cardiac ejection used is given in [7]. Firstly, a 11 cm-long vessel district corresponding to the iliac bifurcation was embedded in the 1D model of the arterial tree. In this example we analyze the physiological blood flow at the iliac bifurcation for different spatial discretizations (case (i): 41000 nodes, case (ii): 77000 nodes, case (iii): 208000 nodes) and put attention in both the global and local solutions. In figure 1 we see how the global solution (mean pressure and flow rate) is affected by the 3D spatial discretization in the case (i), mainly at the proximal location in the flow curve.

![Figure 1: Solution at the global scale (1D model) over the coupling interfaces.](image)

(a) Abdominal aorta (proximal location).  
(b) Left iliac artery (distal location).

When looking at the OSI index (see Figure 2) we see that the differences are small, and the main characteristics are the same in the three cases.

![Figure 2: OSI indicator (from left to right cases (i), (ii) and (iii)).](image)

Secondly, we study the influence in local hemodynamics, again under physiological regimes, to the size of a cerebral aneurysm. We employ three different sizes. In this case, we aim at comparing the intra-aneurismal blood flow behavior, comparing the values of the WSS index. This is done for two time instants. In Figure 3, we see that the blood flow produces a larger value of the shear stress in the region just after the aneurysm when the pathology is more critical.
4 CONCLUSIONS

In this work we have presented a closed framework to deal, in a variational context, with multi-scale computational hemodynamics. Thus, we have presented the model as well as examples of application that give evidences about the performance and potentialities of coupled 3D–1D–0D models with the aim of simulating the blood flow in 3D arterial vessels under the physiological conditions resulting from systemic interaction.

REFERENCES