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A dimensionally-heterogeneous closed-loop model for the cardiovascular system and its applications

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ABSTRACT

In the present work a computational model of the entire cardiovascular system is developed using heterogeneous mathematical representations. This model integrates different levels of detail for the blood circulation. The arterial tree is described by a one dimensional model in order to simulate the wave propagation phenomena that take place at the larger arterial vessels. The inflow and outflow locations of this 1D model are coupled with lumped parameter descriptions of the remainder part of the circulatory system, closing the loop. The four cardiac valves are considered using a valve model which allows for stenoses and regurgitation phenomena. In addition, full 3D geometrical models of arterial districts are embedded in this closed-loop circuit to model the local blood flow in specific vessels. This kind of detailed closed-loop network for the cardiovascular system allows hemodynamics analyses of patient-specific arterial district, delivering naturally the appropriate boundary conditions for different cardiovascular scenarios. An example of application involving the effect of aortic insufficiency on the local hemodynamics of a cerebral aneurism is provided as a motivation to reproduce, through numerical simulation, the hemodynamic environment in patients suffering from infective endocarditis and mycotic aneurisms. The need for incorporating homeostatic control mechanisms is also discussed in view of the large sensitivity observed in the results, noting that this kind of integrative modeling allows such incorporation.

1. Introduction

The structural and functional behavior of the cardiovascular system can be considered as the result of the interaction among different levels of detail that describe the blood circulation. The integration of these levels of detail is mandatory in order to gain insight about the way in which local and global phenomena are inter-related.

When modeling the blood circulation in the cardiovascular system, the following levels of detail are identified (also called levels of integration): (i) the hemodynamics of large arteries, (ii) the local circulation in specific vessels, (iii) the peripheral circulation, (iv) the venous circulation and (v) the cardiac/pulmonary circulation. Such levels of circulation sometimes refer to a certain geometrical scale (for instance the blood flow in large arteries, or in specific districts), and sometimes refer to a given vascular entity (the heart, or a given peripheral bed).

There have been several approaches to integrating different levels of circulation in the sense introduced above. Models based on lumped representations have been employed to accomplish this task [1–5]. As well, the use of distributed models for simulating the blood flow in compliant vessels has been matter of research [6–13]. Particularly, the 1D model employed in Ref. [8] contains 55 arterial segments and a lumped representation (0D model) for the peripheral/venous/pulmonary and cardiac circulations. In Ref. [14], phenomenological models of the cardiac valves are proposed to accurately account for the opening and closing phases of the valves, being capable of modeling certain pathological conditions like regurgitation and stenosis.

When modeling blood flow in specific vessels, several contributions have dealt with heterogeneous representations in order to couple local and global hemodynamics phenomena. This has been mostly carried out using 3D and 1D (or 0D) models to couple blood flow in complex arterial geometries with either full or partial geometrical descriptions for the systemic dynamics [15–23].

This work presents a computational model of the entire cardiovascular system which accounts for specific vessels, systemic arteries, peripheral circulation, systemic veins, pulmonary and heart circulation and complex valve functioning. The arterial tree is described by a one dimensional model with 128 arterial segments [7] to represent the wave propagation phenomenon in the larger

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arterial vessels including the wave reflections occurring at branching sites, bifurcations, and at any other location with impedance mismatch interfaces. At each outflow point the peripheral circulation in arterioles and capillaries is incorporated by using 0D three-element Windkessel models [12]. In turn, the whole peripheral circulation converges to the venous system through the upper and lower parts of the body [8]. These two main compartments are represented using lumped models for the venules, veins and cavas (inferior and superior). The right and left heart circulation as well as the pulmonary circulation, are also modeled by means of 0D models. Particularly, a non-linear model for the heart valves is considered [14], which allows for the regurgitation phase during the valve closing. Finally, the 0D model of the left ventricle is coupled with the inflow boundary in the 1D model, closing the cardiovascular system. The entire 0D model which performs the coupling between the outflow and inflow points in the arterial tree consists of 14 compartments. In addition, full 3D models are considered in order to account for all the complexity of blood flow in specific vessels of interest [15].

Despite the fact that the model of the cardiovascular system presented in this work borrows the most important features of the different models available in the literature, the novelty of our contribution resides in the integration of all these models in a detailed closed-loop of the entire cardiovascular system. The integration of such models was carried out following an extended non-classical variational formulation also proposed by the authors (see Ref. [15] for details). Among other applications, the use of this integrated model can be applied to put in evidence the way in which a pathology in one region of the cardiovascular system can affect local hemodynamics at a different site, therefore assessing the effects of the local-global hemodynamics coupling in the analysis of patient-specific cardiovascular diseases.

Concerning applications, it has been widely recognized the importance of the coupling between local and global hemodynamics phenomena [24] (and references therein). Particularly, one specific example is the hemodynamic analysis on patients suffering from cerebral aneurisms and at the same time from aortic regurgitation. This scenario is especially important because regurgitation in some cases (in acute regurgitation more than in chronic cases) is related to infective endocarditis. In turn, the presence of cerebral aneurisms that turn out to result infectious (mycotic aneurisms) may yield fatal consequences. Rupture in mycotic aneurisms without warning is not unusual in patients with infective endocarditis [24–26]. Hence, the model developed in this work is employed to simulate the effect of aortic insufficiency on the local hemodynamics in a cerebral aneurism. This example aims at showing the descriptive capabilities of this kind of models to simulate cardiovascular scenarios of clinical interest. However, it is worthwhile to remark the need for a new class of models capable to integrate not only all these advances but also allowing the interaction between the cardiovascular system and systems such as autonomous nervous, respiratory, digestive, endocrine and lymphatic [27], which influence the behavior of the cardiovascular system under normal or altered conditions due to diseases or human intervention. Specifically, this integration between the present model and the baroreflex mechanism has been recently reported by the authors in Ref. [28]. Moreover, the accuracy provided by the different components that form the proposed closed-loop model was already described in the literature (see for example Refs. [8,11,14] and references there in). Concerning patient-specific hemodynamic analyses, this integrated model provides: (i) the boundary conditions for the 3D arterial district (se for example Refs. [16,17,29]); (ii) the framework to adjust model parameters that govern the integrated model such that pressure and flow waveforms match patient-specific records (see for example Refs. [11,30]); and (iii) adequate descriptive capabilities to reproduce a wide range of physiological and pathophysiological conditions, making possible to deliver modeling-based diagnoses, as well as more accurate and appropriate therapy and surgical planning.

This work is organized as follows. Section 2 presents all the modeling tools used for the different levels of circulation. Section 2.7 provides the data upon which the present model is set up. In Section 3 the model is employed to simulate different cardiovascular scenarios. The discussion of the results is given in Section 4.

2. Methods

In this section the mathematical models employed for the different vascular entities considered in the closed-loop representation of the cardiovascular system are described. Finally, an account of the iterative method used to effectively couple the heterogeneous models is presented.

2.1. 1D model for the systemic arteries

Blood flow in the systemic arteries is modeled using a 1D model derived from the Navier–Stokes equations by introducing suitable assumptions. This procedure yields, for a generic 1D segment $\mathcal{I}$, the following equations (see Ref. [31] for its derivation)

$$\begin{cases} \dfrac{\partial Q}{\partial t} + \dfrac{\partial}{\partial x}\left(\beta \dfrac{Q^2}{A}\right) = -\dfrac{A}{\rho}\dfrac{\partial P}{\partial x} - \dfrac{\pi D}{\rho}\tau_o & \text{in } \mathcal{I}, \\ \dfrac{\partial A}{\partial t} + \dfrac{\partial Q}{\partial x} = 0 & \text{in } \mathcal{I}, \end{cases} \tag{1}$$

where $\tau_o = f_r \frac{\rho \tilde{u}|\tilde{u}|}{8}$, $Q = \tilde{u}A$, A is the cross sectional area of the artery (D its diameter), $\tilde{u}$ the mean value of the axial velocity, x the axial coordinate, P the mean pressure, ρ the blood density, τ_o the viscous shear stress acting on the arterial wall, f_r a Darcy friction factor (in this work a fully developed parabolic velocity profile is considered) and β is the momentum correction factor ($\beta = 1$ is considered here).

The system is closed by introducing a constitutive law which establishes a relation between the pressure and the cross sectional area. Here a non-linear visco-elastic model [32] is used

$$P = P_o + \frac{h_o E_e}{R_o}\varepsilon + \frac{K h_o}{R_o}\dot{\varepsilon} \quad \text{in } \mathcal{I}, \tag{2}$$

where $\varepsilon = \sqrt{\frac{A}{A_o}} - 1$, R is the radius of the artery, E_e is the effective Young modulus of the elastin, K is the effective viscosity of the wall, h is the thickness of the arterial wall and the subscript 'o' denotes quantities evaluated at the reference pressure P_o.

2.2. 0D model for the arterioles and capillaries

The peripheral circulation is represented through Windkessel models [4,12]. The Windkessel behavior is determined by a resistance R_c to represent the capillaries, in series with the model of arterioles characterized by a resistance R_a and a capacitor C_a connected in parallel. Here, P_i and P_o are the pressures at the input and output of the compartment, respectively, Q_i is the blood flow into the Windkessel element. The balance equation for this model is the following

$$\frac{dQ_i}{dt} = \frac{1}{R_c R_a C_a}\left[R_a C_a \frac{d}{dt}(P_i - P_o) + (P_i - P_o) - (R_c + R_a)Q_i\right], \tag{3}$$

Since the Windkessel element plays the role of an interface between arterial and venous systems, P_i represents the pressure from the arterial side and P_o is the pressure from the venous side, more precisely at the venules.