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Study of cardiovascular function using a coupled left ventricle and systemic circulation model

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ABSTRACT

To gain insight into cardio-arterial interactions, a coupled left ventricle-systemic artery (LV–SA) model is developed that incorporates a three-dimensional finite-strain left ventricle (LV), and a physiologically-based one-dimensional model for the systemic arteries (SA). The coupling of the LV model and the SA model is achieved by matching the pressure and the flow rate at the aortic root, i.e. the SA model feeds back the pressure as a boundary condition to the LV model, and the aortic flow rate from the LV model is used as the input for the SA model. The governing equations of the coupled system are solved using a combined immersed-boundary finite-element (IB/FE) method and a Lax–Wendroff scheme. A baseline case using physiological measurements of healthy subjects, and four exemplar cases based on different physiological and pathological scenarios are studied using the LV–SA model. The results of the baseline case agree well with published experimental data. The four exemplar cases predict varied pathological responses of the cardiovascular system, which are also supported by clinical observations. The new model can be used to gain insight into cardio-arterial interactions across a range of clinical applications.

1. Introduction

Understanding the interaction between the heart and arteries can provide valuable information for clinical diagnosis and treatment in cardiovascular disease (Cecelja and Chowienczyk, 2012; Chirinos, 2013; Ky et al., 2013). However, current mathematical approaches tend to focus on either the arteries (Müller and Toro, 2014; Qureshi et al., 2014), or the heart alone (Perktold and Rappitsch, 1995; Gerbeau et al., 2005). In the former, ventricular function is either prescribed (Urquiza et al., 2006; Olufsen et al., 2000; Figueroa et al., 2006), or simplified using lumped-parameters (Kim et al., 2009; Arts et al., 2005). In the latter, a lumped systemic circulation model is usually used as boundary condition (Žáček and Krause, 1996; Sun et al., 1997; Arts et al., 2005; Lumens et al., 2009; Peskin and McQueen, 1989; Watanabe et al., 2002; Lee et al., 2009; Gao et al., 2014a; Kerckhoffs et al., 2007; Krishnamurthy et al., 2013; Baillargeon et al., 2014).

As changes in arterial properties can alter the heart function and vice versa (Noguchi et al., 2011), in this paper, we focus on the coupling of the heart and the arteries, by combining a models of a 3D left ventricle (LV) with a systemic arteries (SA) model that uses a structural tree description of the vascular beds containing the smaller arteries. The 3D LV is reconstructed from a dataset of in vivo magnetic resonance imaging (MRI) of a healthy volunteer (Gao et al., 2015a), which includes fluid–structure interaction (FSI). The SA model is based on the development by Olufsen (1999) and Olufsen et al. (2000), which includes both large arteries and remote vascular beds.

2. Methodology

The coupled left ventricle and the systemic artery (LV–SA) model is shown in Fig. 1. The methodologies for the 3D LV and the 1D SA models have been published elsewhere (Gao et al., 2014a; Olufsen et al., 2000, 2012), but are briefly described here to explain the coupling procedure.

2.1. The LV model

The LV model consists of the valvular and inflow/outflow tracts (assumed passive), and the active LV region. The model is solved using a combined immersed boundary finite element (IB/FE) method. Let $\Omega \subset \mathbb{R}^3$ denote the physical domain occupied by the fluid–structure interaction (FSI) system, in which $\mathbf{x} = (x_1, x_2, x_3) \in \Omega$ are fixed Eulerian coordinates. Let $U \subset \mathbb{R}^3$ denote the reference configuration of the immersed solid, in which $\mathbf{X} = (X_1, X_2, X_3) \in U$ are Lagrangian coordinates. $\chi(\mathbf{X}, t)$ describes the physical position of

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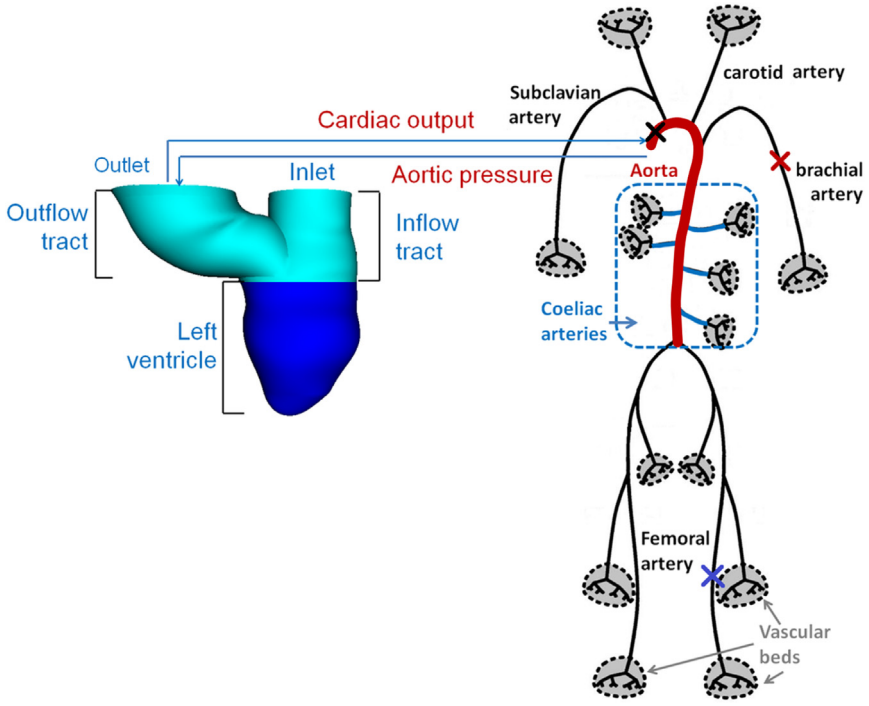

Fig. 1. Schematic illustration of the coupled 3D LV and the 1-D systemic circulation model. The 3D LV is immersed in a 16.5 cm × 16.5 cm × 16.5 cm fluid box, and the LV is meshed with 138k tetrahedral elements. Pressure and flow rate are obtained in the LV and at midpoints of each vessel in three groups of the large arteries, i.e. aorta (red), coeliac arteries (black), and other long arteries (black). '×' denotes the midpoints of the ascending aorta (thicker curve), brachial, and femoral arteries.

the material point $\mathbf{X}$ at time t, and then the physical region occupied by the immersed solid at time t is $\chi(U,t)=\Omega_s(t)\in\Omega$, and the region occupied by the fluid at time t is $\Omega_f(t)=\Omega\backslash\Omega_s(t)$ ('\' is the setminus operator). The formulation of the FSI equations is (Griffith, 2012; Gao et al., 2014b)

$$\rho\left(\frac{\partial \mathbf{u}}{\partial t}(\mathbf{x},t)+\mathbf{u}(\mathbf{x},t)\cdot\nabla\mathbf{u}(\mathbf{x},t)\right)=-\nabla p(\mathbf{x},t)+\mu\nabla^2\mathbf{u}(\mathbf{x},t)+\mathbf{f}^s(\mathbf{x},t), \quad (1)$$

$$\nabla\cdot\mathbf{u}(\mathbf{x},t)=0, \quad (2)$$

$$\frac{\partial \chi}{\partial t}(\mathbf{X},t)=\int_\Omega \mathbf{u}(\mathbf{x},t)\delta(\mathbf{x}-\chi(\mathbf{X},t))\,d\mathbf{x}, \quad (3)$$

$$\mathbf{f}^s(\mathbf{x},t)=\int_U \nabla_{\mathbf{X}}\cdot\mathbb{P}^s(\mathbf{X},t)\delta(\mathbf{x}-\chi(\mathbf{X},t))\,d\mathbf{X} - \int_{\partial U}\mathbb{P}^s(\mathbf{X},t)\cdot\mathbf{N}(\mathbf{X})\delta(\mathbf{x}-\chi(\mathbf{X},t))\,dA, \quad (4)$$

in which μ is the fluid viscosity, $\mathbf{u}(\mathbf{x},t)$ is the fluid velocity of the blood, and $p(\mathbf{x},t)$ is the pressure, $\mathbf{N}(\mathbf{X})$ is the exterior unit normal to U, and $\delta(\mathbf{x})$ is the three-dimensional Dirac delta function. We assume that the fluid and the solid have the same density ρ. $\mathbb{P}^s(\mathbf{X},t)=\det(\mathbb{F})\boldsymbol{\sigma}^s\mathbb{F}^{-T}$ is the first Piola–Kirchhoff stress tensor, and $\boldsymbol{\sigma}^s$ is the structure Cauchy stress tensor,

$$\boldsymbol{\sigma}^s=\boldsymbol{\sigma}^p+\boldsymbol{\sigma}^a, \quad (5)$$

where $\boldsymbol{\sigma}^a=T_0\,T(\mathbf{f}\otimes\mathbf{f})$ is the active stress, while the contractile tension T is described by the myofilament model of Niederer et al. (2006), which is triggered by a prescribed intracellular calcium transit (Hunter et al., 1998), as shown in Fig. 3. T_0 is introduced to make the contraction patient-specific. The passive stress $\boldsymbol{\sigma}^p$ is determined through the Holzapfel–Ogden strain energy function (Holzapfel and Ogden, 2009), as detailed in the Appendix A.

Equations (1) and (2) are discretized using a finite-difference method, and Eqs. (3) and (4) are discretized with a finite-element method. The material parameters in (12) are determined inversely by fitting the measured end-diastolic volume and myocardial strains using a multi-step optimization procedure (Gao et al., 2015b). T_0 is determined by matching the measured stroke volume (Gao et al., 2014a). The valvular region is modelled as a neo-Hookean material, with the shear modulus adjusted so that its maximum displacement agrees with MRI measurements. The inflow/outflow tracts are both assumed to be rigid, with the inlet and outlet annuli fixed in space.

2.2. The systemic arterial model

The SA model consists of 24 large arteries modelled as a one-dimensional cross-sectional-area-averaged flow and pressure. Each terminal vessel in the network of the large arteries is coupled with a group of small arteries (the vascular bed), which are modelled as an asymmetric structured-tree to provide outflow boundary conditions (Olufsen, 1999; Olufsen et al., 2000, 2012).

The governing equations for the SA model are

$$\frac{\partial Q}{\partial x}+\frac{\partial A}{\partial t}=0, \quad (6)$$

$$\frac{\partial Q}{\partial t}+\frac{\partial}{\partial x}\left(\frac{Q^2}{A}\right)+\frac{A}{\rho}\frac{\partial P}{\partial x}=-\frac{2\pi\nu R}{\delta^*}\frac{Q}{A}, \quad (7)$$

$$P(x,t)-P_0=\frac{4}{3}\frac{Eh}{r_0}\left(1-\sqrt{\frac{A_0}{A}}\right), \quad (8)$$

where Q is the volumetric flow rate, A is the cross-sectional area, P is the averaged cross-sectional pressure, P_0 is the constant external pressure, ρ is the density, ν is the kinematic viscosity, R is the radius of the vessel. A_0 and r_0 are the cross sectional area when $P=P_0$, δ^* is the width of the boundary layer ($\delta^*\ll R$), h is the wall thickness, and E is the arterial Young's modulus, computed as,

$$\frac{Eh}{r_0}=k_1\exp(k_2\,r_0)+k_3, \quad (9)$$

where k_i ($i=1$–3) are material constants (Olufsen et al., 2000).