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# Multi-component model of intramural hematoma

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ABSTRACT

A novel multi-component model is introduced for studying interaction between blood flow and deforming aortic wall with intramural hematoma (IMH). The aortic wall is simulated by a composite structure submodel representing material properties of the three main wall layers. The IMH is described by a poroelasticity submodel which takes into account both the pressure inside hematoma and its deformation. The submodel of the hematoma is fully coupled with the aortic submodel as well as with the submodel of the pulsatile blood flow. Model simulations are used to investigate the relation between the peak wall stress, hematoma thickness and permeability in patients of different age. The results indicate that an increase in hematoma thickness leads to larger wall stress, which is in agreement with clinical data. Further simulations demonstrate that a hematoma with smaller permeability results in larger wall stress, suggesting that blood coagulation in hematoma might increase its mechanical stability. This is in agreement with previous experimental observations of coagulation having a beneficial effect on the condition of a patient with the IMH.



## 1. Introduction

Aortic dissection is a serious medical condition characterized by tearing of the inner layer of the aorta which results in blood surging through the tear causing the inner and middle layers of the aorta to separate. Rupture of the outside aortic wall of the blood-filled vessel often results in fatal aortic dissection. Aortic intramural hematoma (IMH) is an atypical form of aortic dissection. It results from the rupture of the vasa vasorum and the formation of a hemorrhage in the aortic wall. Its distinguishing feature is an absence of the tear in the inner layer that characterizes classic aortic dissection. However, since the aortic wall weakens after the formation of an IMH, the rupture within the wall might propagate and lead to dissection (Sawhney et al., 2001; Tsai et al., 2009). An aortic IMH was found in 5–20% of patients who had shown signs of acute aortic syndromes. Improvement is observed in approximately 10% of patients, while progression to classic dissection occurs in 29–47% of patients and carries a risk of wall rupture in 20–45% of patients (Tsai et al., 2009). Even though levels of morbidity and mortality due to IMH are as high as in case of classic dissection, the appropriate treatment of the IMH is neither well defined nor understood.

Mathematical modeling and numerical simulations have been recently used for predicting variety of patient specific health conditions which currently cannot be determined based only on experimental data. In particular, numerical simulations have been used to determine stress distribution in the aortic wall under different conditions. For example, wall stress in an aorta with a growing aneurysm has been investigated by Thubrikar et al. (1999) as a possible mechanism for the development of transverse intimal tears in aortic dissections. A patient specific study was performed by Nathan et al. (2011) where local thoracic aortic wall stress was estimated using finite element analysis. The influence of the aortic root movement on the aortic wall stress was investigated by Beller et al. (2004) using a single layer model of the wall. The computed wall stress was used to explain why aortic dissection occurred more often in this location. Stress distribution in each layer of the aorta was investigated by Gao et al. (2006a,b). However, since the aortic outlet zero pressure was applied, the effects of the downstream flow were not taken into account. A similar study was performed by Khanafer and Berguer (2009) with imposed physiologically relevant pressure values. A hemodynamics-based model was proposed by Menichini and Xu (2016) to study the formation and growth of thrombus in the aortic dissection. The model was used for simulating different idealized aortic dissections to investigate the effect of geometric features of the aorta on thrombus formation.

A novel two-dimensional multi-component model is introduced in this paper for studying interaction between pulsatile

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blood flow and aortic wall with the IMH. The model describes dynamics of main three layers of the aortic wall and takes into account differences in their mechanical properties. In contrast with the previous studies, the model describes in detail the dynamics of the IMH and its interaction with the deformed aortic wall. The IMH is caused by the hemorrhage into the aortic wall, and the false lumen is known to cause the formation of blood clots (Tsai et al., 2007). Therefore, we assume that IMH consists of both fluid and solid phases. Hence, we describe the IMH using a poroelastic submodel capable of simulating how different distributions of the coagulated blood within IMH could influence its mechanical properties. The submodel for the aorta with IMH is coupled with the submodel for blood flow in the lumen. The coupling between blood flow and a 2-layer structure model was previously considered by Bukač et al. (2015), but without the presence of the poroelastic material. Furthermore, the coupling between the fluid, a single layer, thin elastic structure and poroelastic material was previously considered by Bukač (2016), but in a different configuration. The poroelastic material in Bukač (2016) was in contact with both fluid and the thin elastic structure. In contrast with the previous work, in our present model the poroelastic submodel representing IMH is fully immersed in the 3-layer aortic wall.

Model simulations of the aortic blood flow under physiological conditions were performed using the finite element method. Finally, model simulations were used to investigate relation between the peak wall stress, hematoma thickness and permeability in patients of different age.

## 2. Methods

The multi-component model describes blood flow in a section of a descending aorta with the IMH (see Fig. 1). Similar to the work by Khanafer and Berguer (2009), an idealized, axially symmetric section of an artery is considered. This assumption is justified by CT observations indicating that IMH often appears in the form of a focal region of a symmetric, homogeneous wall thickening (see Fig. 2). Moreover, cross section images show that the IMH maintains a constant circumferential relationship with the aortic wall (Rajiah, 2013; Herlinger et al., 2001; Nienaber et al., 1995; Kazerooni and Gross, 2004).

### 2.1. Blood flow

Blood is a mixture of plasma, red and white blood cells, proteins, lipoproteins and ions, and hence it is not homogeneous or Newtonian. However, in medium-to-large vessels, the size of individual elements becomes negligible with respect to the blood vessel diameter, and the Navier–Stokes equations for an incompressible, viscous fluid have been extensively used for simulations (Nakamura et al., 2006; Vasava et al., 2012; Sankaran et al., 2012):

$$\rho_f(\partial_t \boldsymbol{v} + \boldsymbol{v}\cdot\nabla\boldsymbol{v}) = \nabla\cdot\boldsymbol{\sigma}^f, \tag{1}$$

$$\nabla\cdot\boldsymbol{v} = 0, \tag{2}$$

where $\boldsymbol{v}$ is the velocity, $\boldsymbol{\sigma}^f = -p_f\boldsymbol{I} + 2\mu_f\boldsymbol{D}(\boldsymbol{v})$ is the fluid stress tensor, $p_f$ is the fluid pressure, $\rho_f$ is the fluid density, $\mu_f$ is the fluid viscosity and $\boldsymbol{D}(\boldsymbol{v}) = (\nabla\boldsymbol{v} + (\nabla\boldsymbol{v})^T)/2$ is the strain rate tensor. At the beginning of the simulation, the fluid domain is defined as a rectangle of length $L = 10$ cm and height $R_{lumen} = 1$ cm, where the height is the vessel radius. This is consistent with the experimental measurements of the aortic diameters reported by Wolak et al. (2008) and O'Rourke and Nichols (2005). The blood density is taken to be $\rho_f = 1\,\mathrm{g/cm^3}$ and viscosity $\mu_f = 0.035\,\mathrm{g/cm\,s}$. We impose symmetry boundary conditions at the fluid bottom boundary $\Gamma_b^f$ (see Fig. 1) (Khanafer and Berguer, 2009; Bukač et al., 2015).

A physiological, pulsatile velocity $v_D(t)$ is imposed at the inlet of the fluid domain $\Gamma_{in}^f$, while a pressure waveform $p_{out}(t)$ is imposed at the outlet $\Gamma_{out}^f$ (see Fig. 3) (Mills et al., 1970). The boundary conditions are implemented in the following way:

$$\boldsymbol{v} = \left(v_D(t)\frac{R_{lumen}^2 - r^2}{R_{lumen}^2}, 0\right) \quad \text{on } \Gamma_{in}^f \times (0,T), \tag{3}$$

$$\boldsymbol{\sigma}^f\boldsymbol{n}^f = -p_{out}(t)\boldsymbol{n}^f \quad \text{on } \Gamma_{out}^f \times (0,T), \tag{4}$$

where $\boldsymbol{n}^f$ denotes the outward normals to the fluid boundary.

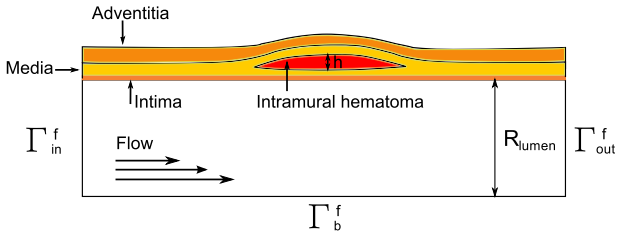


**Fig. 1.** Diagram of an idealized, axially symmetric model of an aorta. $R_{lumen}$ denotes the lumen radius and $h$ denotes the maximum IMH thickness.

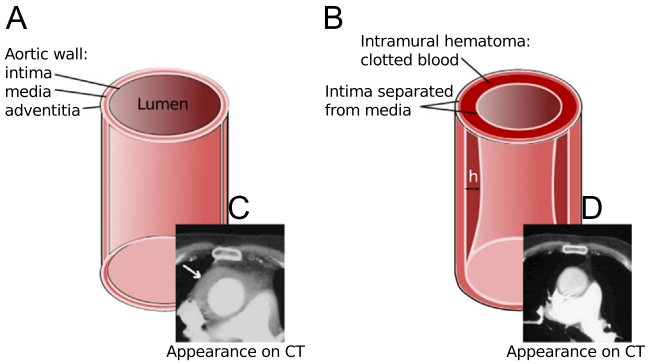


**Fig. 2.** A. Normal aorta. B. Aorta with IMH. C. CT image of normal aorta. D. CT image of aorta with IMH. CT images in panels C and D are taken from Masip (2004).

The velocity waveform $v_D$ corresponds to the Reynolds number of 1200, in which case the flow can be categorized as laminar (Stokes, 1851; Holman, 2001). To circumvent the issues related to the motion of the fluid domain due to the fluid–structure interaction, we use the Navier–Stokes equations in the ALE form (Donea, 1983; Duarte et al., 2004; Bukač et al., 2015).

### 2.2. Aortic wall

Aortic walls are represented as a composite material consisting of three main layers: tunica intima, media and adventitia. We model the aortic wall using a composite structure model, taking into account mechanical properties of each layer. Average thickness ratio of intima/media/adventitia was shown to be 13/56/31 (Schulze-Bauer et al., 2003). Hence, we model the intimal layer as a thin, elastic structure using the Koiter shell model (Bukač, 2013)

$$\rho_i h_i \partial_{tt}\eta_z^i - \frac{h_i E_i}{1-\sigma_i^2}\partial_{zz}\eta_z^i - \frac{h_i}{R_i}\frac{E_i\sigma_i}{1-\sigma_i^2}\partial_z\eta_r^i = f_z^i \tag{5}$$

$$\rho_i h_i \partial_{tt}\eta_r^i + \frac{h_i E_i}{R_i^2(1-\sigma_i^2)}\left(1 + \frac{h_i^2}{12R_i^2}\right)\eta_r^i - \frac{h_i^3}{6}\frac{E_i\sigma_i}{R_i^2(1-\sigma_i^2)}\partial_{zz}\eta_r^i + \frac{h_i}{R_i}\frac{E_i\sigma_i}{1-\sigma_i^2}\partial_z\eta_z^i = f_r^i. \tag{6}$$

The intimal displacement is denoted by $\boldsymbol{\eta}^i = (\eta_z^i, \eta_r^i)$, where $\eta_z^i$ denotes the longitudinal and $\eta_r^i$ the radial displacement. The intimal density is denoted by $\rho_i$, the thickness by $h_i$, and the force applied to the wall by $\boldsymbol{f}_i$. The parameters $E_i$ and $\sigma_i$ are Young's modulus and Poisson's ratio of the intimal layer, respectively.

The deformation of the media/adventitia complex is modeled using the 2D equations of linear elasticity (Quaini, 2009; Bukač et al., 2015):

$$\rho_{ma}\partial_{tt}\boldsymbol{\eta}^{ma} = \nabla\cdot\boldsymbol{\sigma}^s, \tag{7}$$

where $\boldsymbol{\eta}^{ma}$ is the structure displacement and $\rho_{ma}$ is the density. One of the common simplifying assumptions in models of blood flow interaction with the vessel wall is treatment of the biomechanical material properties of the vessel as linearly elastic, homogeneous material (Thubrikar et al., 2001; Hua and Mower, 2001; Scotti et al., 2005; Giannoglou et al., 2006). Some authors even claim that although human arterial tissue acts like a non-linear material, at pressures above 80 mmHg (10.67 KPa) the aorta behaves more like a linearly elastic material (Giannoglou et al., 2006; Thubrikar et al., 2001). Hence, we assume that arterial wall behaves as isotropic, linearly elastic Saint Venant–Kirchhoff material. Thus,

$$\boldsymbol{\sigma}^s = \frac{E_{ma}}{1+\sigma_{ma}}\boldsymbol{D}(\boldsymbol{\eta}^{ma}) + \frac{E_{ma}\sigma_{ma}}{(1+\sigma_{ma})(1-2\sigma_{ma})}(\nabla\cdot\boldsymbol{\eta}^{ma})\boldsymbol{I},$$

where $E_{ma}$ and $\sigma_{ma}$ are Young's modulus and Poisson's ratio, respectively.

Based on the experimental measurements from Xie et al. (1995) and Fischer et al. (2002), we assume that Young's moduli of the intima, media and adventitia are, 2, 6 and 4 MPa, respectively. Poisson's ratio of all three layers is set to 0.45 (Khanafer and Berguer, 2009). Thicknesses of intima, media and adventitia are