Contributions from the modeling of arterial circulation to the simulation of wall shear rate in blood flow within large arterial vessels

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Introduction

One important aspect in computational fluid dynamic (CFD) modeling is the use of accurate inlet and outlet conditions for the flow geometry and obtaining these values are quite the challenge for blood flow within a specific component of the arterial network. In many previous works, the boundary conditions for CFD modeling would rely upon the use of constant pressure profiles being applied across the geometry and losing the transient/elastic nature that is contained by the remaining vessels of the vascular network. In order to better approximate flow data for blood vessel simulations, it is important to better provide better boundary conditions characteristic to vessels with transient flow patterns and elastic effects, which may cause dampening or delays to flow patterns.

Model Development

An approximate model capable of providing estimates for the time-dependent blood pressure and flow profiles in all of the vessels in the human arterial network has been developed for that purpose. Using a linear superposition approximation, the flow and pressure is broken into a series of various harmonics, which are solved in the Fourier domain [1].

$$Q = \bar{Q} + \text{Real}\left(\sum_{m=1}^{\infty} A_m^Q e^{im\omega_0 (t - z/c)} + \sum_{m=1}^{\infty} B_m^Q e^{im\omega_0 (t + z/c)} \right) \tag{1}$$

$$\Delta P = \overline{\Delta P} + \text{Real}\left(\sum_{m=1}^{\infty} A_m^P e^{im\omega_0 (t - z/c)} + \sum_{m=1}^{\infty} B_m^P e^{im\omega_0 (t + z/c)} \right) \tag{2}$$

The model had been developed using previous literature developed by other pioneers in the field, such as Womersley, West, Olufsen, and Zamir, [1-4] and building upon their works with techniques that emulate similar traits to physiological phenomena and provide extra details not explored yet. West proposes an impedance model [2]:

$$Z = \frac{c_0^2 \rho}{\pi r^2 c} \tag{3}$$

where ρ is the density of blood, r is the radius of the vessel, c_0 is the Moens-Korteweg wave velocity given by:

$$c_0 \equiv \sqrt{\frac{Eh}{2\rho r}} \tag{4}$$

and c is the complex wave velocity.

$$c = c_0 \sqrt{\left(1 - \frac{2J_1(\alpha_c)}{\alpha_c J_0(\alpha_c)}\right)} \tag{5}$$

Additional terms are the modulus of elasticity, E, and the thickness of the vessels, given by h. J_1 and J_0 are Bessel functions of the first kind, and α_c is the complex dimensionless Womersley number, given by:

$$\alpha_c \equiv i^{3/2} r \sqrt{\frac{2\pi f \rho}{\mu}} \tag{6}$$

Where f is the frequency of pulsations for flow and μ is the viscosity of the fluid.

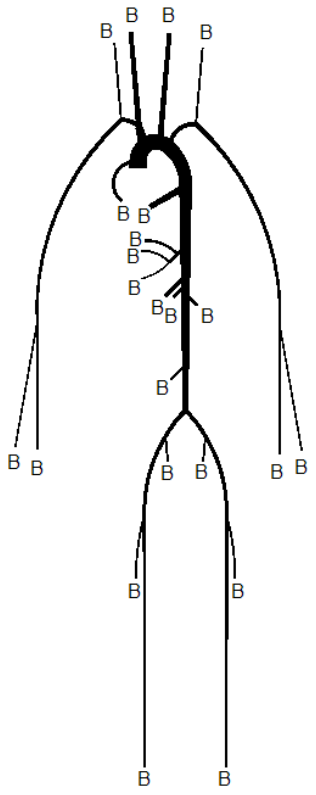

Figure 1: Model vascular network. The network is the same as that of Olufsen [3]. The network includes 45 arteries, each characterized by its length and upstream and downstream radii. The network also includes 23 branching terminal sub-networks denoted by "B"s.

Personalization of the model can be performed by inputting an aortic pressure wave, geometric dimensions of large arteries, (available, for example by MRI) various blood properties, vessel elasticity etc.. With the features of freely inputting data from other potential models, one can make comparisons of pressure and flow profiles for various individuals. A key feature of the model is that it takes into account the full circulatory network from the largest artery to the smallest capillary. The model only requires 45 key large arteries to be determined, and then uses a physical scaling model described by Geoffrey West [2] to bifurcate daughter vessels of the 45 main arteries all the way to capillaries. With this feature, over twenty-four million vessels total are included in the whole model, but there are 45 main arteries that are necessary to input for the model.

The model takes the effects of the pressure and flow and breaks them into steady state and transient portions. Separating the two allows complex phenomena to be applied to the steady portion of the model, while still maintaining the wave transduction and reflection that occurs in the vessels at bifurcations due to transient flow. Improvements with the model include tapering of the blood vessels, and using a lubrication approximation to account for additional effects to the Poiseuille resistance. The rheology of blood is accounted for using both a Fahraeus-Lindquist effect, which takes into account the relationship of local vessel diameter and hematocrit on viscosity of the blood [5].

$$\frac{Hct}{Hct_D} = Hct_D + (1 - Hct_D)\left(1 + 1.7e^{-0.415D} - 0.6e^{-0.011D}\right) \tag{7}$$

The local hematocrit (Hct) can be correlated as a function of the discharge hematocrit (Hct_D) and the local vessel diameter (D – microns for Eq. 7). The effect on viscosity is:

$$\eta_{rel} = 1 + \frac{e^{Hct \cdot a - 1}}{e^{0.45 \cdot a - 1}}\left(110e^{-1.1424D} + 3 - 3.45e^{-0.035D}\right) \tag{8}$$

where the term a is given as

$$a = \frac{4}{1 + e^{-0.593(D - 6.74)}}\,. \tag{9}$$

Also, a yield-stress relationship is implemented with the Casson expression [6], which accounts for the formation and breaking of red blood cell rouleaux during flow. The Casson equation is:

$$\tau_w^{1/2} = \tau_0^{1/2} + \left(\mu_N \dot{\gamma} \lambda\right)^{1/2} \tag{10}$$

where the wall shear stress is given by τ_w, and τ_0 is the yield stress. The viscosity at high shear rates is given by μ_N, while $\dot{\gamma}$ is the shear rate, and λ is a viscous time constant. The yield stress is given by the following empirical formula in [CGS]:

$$\tau_0 \sim \left(Hct - Hct_c\right)^3, \qquad (11)$$

The critical hematocrit (Hct_C) below which no yield stress is observed, for blood is given as 0.04 (i.e. 4%).

Model Predictions

This work is related and compared alongside previous literature results, while allowing easy modifications to customize to other physical parameters. The results of the one-dimensional boundary condition model show decent agreement in how total peripheral resistance of the network at the aorta has been measured [7, 8], in addition it also shows a pressure peaking phenomena observed in previous literature and similar quantitative peaking effects [9].

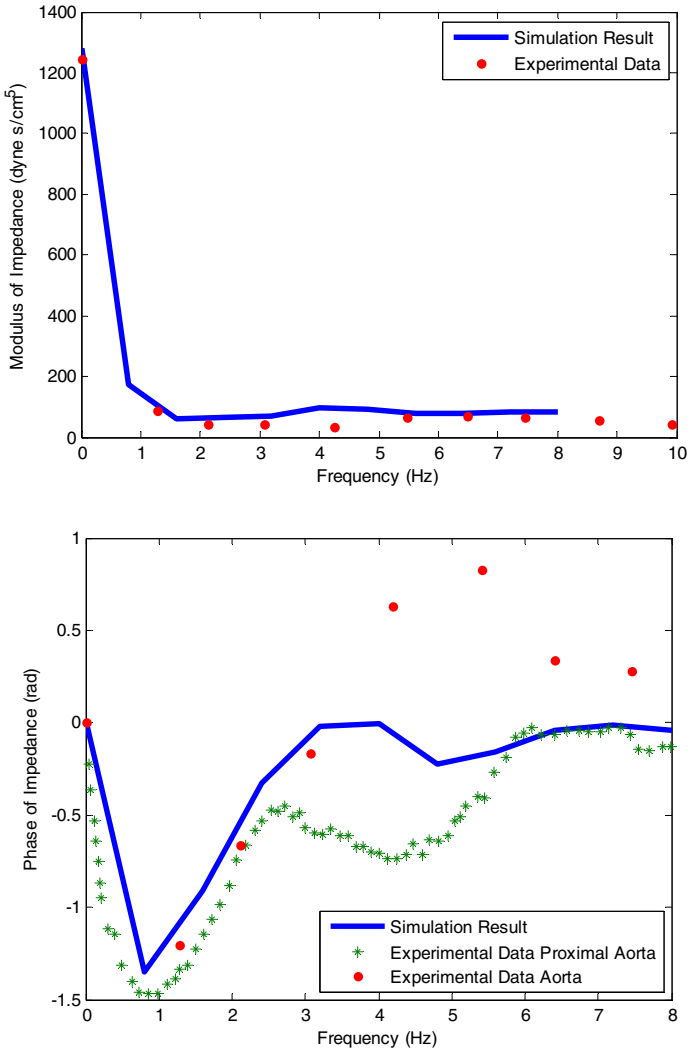

Figure 2: Aortic input impedance (modulus and phase) for model network

Locally in vessels, the pressure waveform is preserved, but as the pulse moves further from the heart, the waveform develops subsequent oscillations beyond the original

pulse. The pressure transforms with a larger peak further away from the heart, and the integration of reflected waves transforms the pulse into multiple peaks at further vessels.

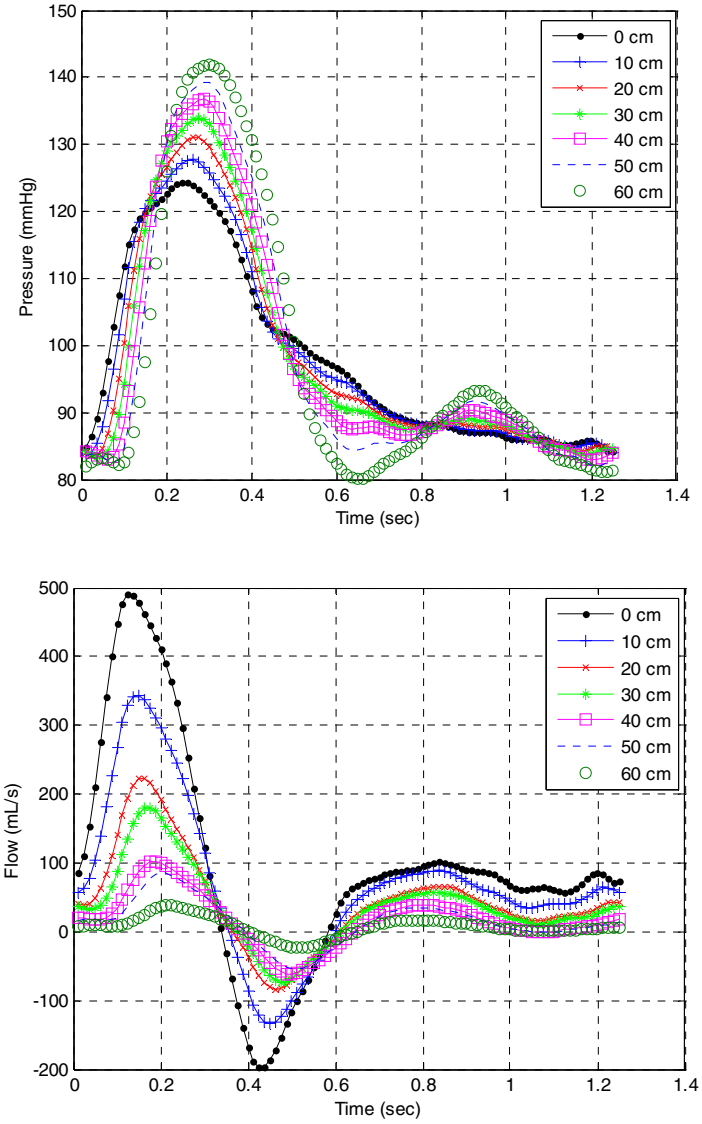

Figure 3: Model predictions for A. pressure and B. flow in the aorta trunk. Distance notes axial position down the aortic trunk from the heart. More distal arteries have delayed pressure waves and show pressure wave amplification

Model Applications

The model has been developed through a multidisciplinary collaboration [10], and is supplied in a user friendly Matlab based format, and it allows for the incorporation of the specific relationship that emerges between the pressure and flow profiles. With the ease of accessing the pressure flow, it makes the application of the model as the outlet condition within more elaborate CFD simulation packages, such as Fluent, easier. More accurate flow profiles can be applied to the CFD simulation of localized regions of the vascular

network, and local flow conditions can be better investigated by having the simulation performed more accurately.

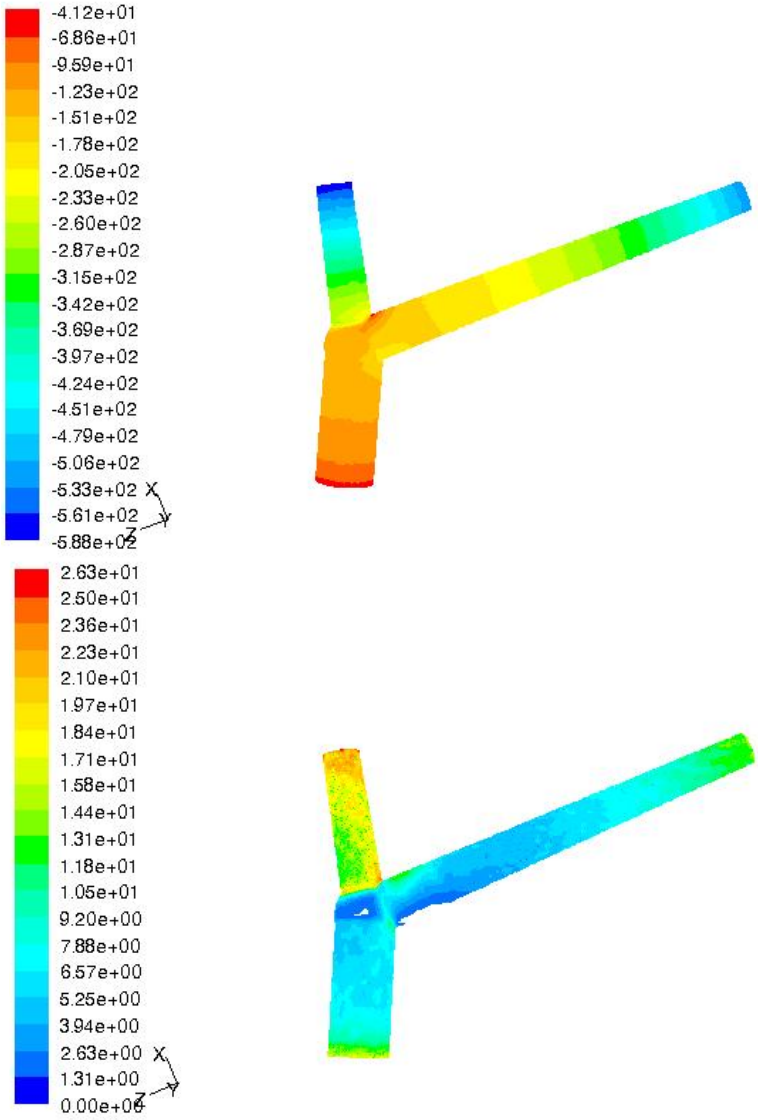

Figure 4: Static Pressure and Wall Shear Stress Contour Plots of Left Main Coronary Artery bifurcating into Left Anterior Descending and Left Circumflex Arteries (Relative Pressure)

Conclusions

Overall, stationary network performance at different points in the network qualitatively agrees with previous computational results, as well as with experimental data. The steady state results confirm a phenomenon similarly observed with the proportional Poiseuille conditions, in that the flow of blood through bifurcations has a compensatory effect which allows increased flow resistances within one component to be compensated with a reduction in network resistance. This model has been used to obtain more physiological relevant outlet conditions in modeling simulations of the left main coronary

artery (near the bifurcation of the left anterior descending and left circumflex coronary arteries) using the commercial software, Fluent. Results are compared against more traditional outlet conditions for a normal and atherosclerotic case (developing in the left anterior descending).

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Acknowledgements

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