WACBE World Congress on Bioengineering 2007
Bangkok, THAILAND

# A ONE-DIMENSIONAL NUMERICAL MODEL FOR THE SIMULATION OF THE BLOOD FLOW ON ARTERIES.

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According to WHO estimates, 17 million people around the globe die of CardioVascular Disease (CVD) each year. About 600 million people with high blood pressure are at risk of heart attack, stroke and cardiac failure. In 1999 CVD contributed to one-third of global deaths. Low and middle-income countries contributed to 78 percent of CVD deaths. By 2010 CVD is estimated to be the major cause of death in developed countries. This huge impact has motivated the development of numerical models for arterial behavior in order to study cardiovascular pathologies and interventions.

## 1. INTRODUCTION.

The reason why one-dimensional models for the blood flow may be attractive is that full 3D computer simulations of the whole cardiovascular system and specific surgical interventions are quite prohibitive in terms of computational calculus. The aim of this work is to create and validate a new numerical scheme with experimental measures of the flow profile obtained by high resolution magnetic resonance and ultrasound techniques

## 2. METHODOLOGY.

### 2.1. Mathematical model.

We introduce a non-linear one-dimensional model to describe the flow motion in arteries and its interaction with the wall displacement. The governing system of equations (Eq.1-Eq.2-Eq.3) results from conservation of mass and momentum applied to a 1-D impermeable and deformable tubular control volume of incompressible and Newtonian fluid. The model has been deduced by making the following assumptions: axial symmetry, radial displacements, and constant pressure on each section, no body forces and dominance of axial velocity. Also, in the absence of branching, a short section of an artery may be considered a cylindrical compliant tube.

$$\frac{\partial A}{\partial t}+\frac{\partial Q}{\partial z}=0 \qquad \text{Eq.1}$$

$$\frac{\partial Q}{\partial t}+\frac{\partial}{\partial z}\left(\alpha\frac{Q^2}{A}\right)+\frac{A}{\rho}\frac{\partial P}{\partial z}+K_R\left(\frac{Q}{A}\right)=0 \qquad \text{Eq.2}$$

where the unknowns are A(z,t) is the cross-sectional area of the vessel, Q(z,t) is the mean flux, P is the average internal pressure over the cross-section, α is the momentum-flux correction coefficient,

z is the axial coordinate along the vessel, t is the time, ρ is the density of the blood taken here to be 1040 Kg·m3 and $K_R$ is the friction force per unit length, which is modelled as $K_R = 2\pi\nu(\gamma+2)$ according to Formaggia[1], with ν the viscosity of the blood taken here to be 4.5 mPa·s. In order to close the system equations (Eq.1-Eq.2), we provide a relation for the pressure completed with a pressure–area (Eq.3) relation previously. It assumes a thin, homogeneous and elastic arterial wall and it takes the form.

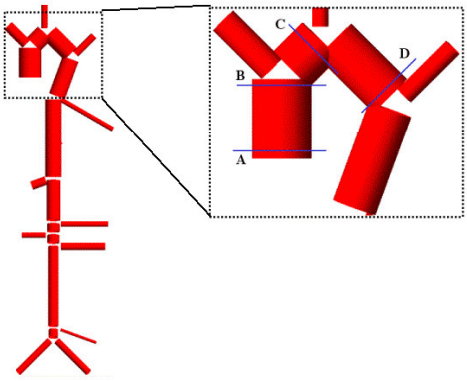
$$P = P_{ext} + \psi(A; A_o, \beta) \Rightarrow \psi = \beta \frac{\sqrt{A} - \sqrt{A_o}}{A_o} \Rightarrow \beta = \frac{E h_0 \sqrt{\pi}}{1 - \xi^2} \qquad \text{Eq.3}$$

where $A_0$ and $h_o$ are the sectional area and wall thickness at the reference state ($P_0$,$U_0$), with $P_0$ and $U_0$ assumed to be zero, E is the Young's modulus and ξ is the Poisson's ratio, typically taken to be ξ=0.5, since biological tissue is practically incompressible. The parameter β is related to the speed of pulse wave propagation, c (Eq.4), through Formaggia[1],

$$c = \frac{\beta}{3\rho A_o} A^{\frac{3}{2}} \qquad \text{Eq.4}$$

**2.2 Boundary conditions and branching.**

The hyperbolic system of partial differential equations (Eq.1-Eq.2-Eq.3) is solved in each arterial segment of Fig.1 with the following boundary conditions. At the proximal end of the ascending aorta (Fig1.Left.(A)) we enforce a real inflow rate Q(t) gives for the MRI measures in a cardiac cycle and for the rest of the arteries we impose the terminal resistance according to the reference Wang and Parker[2][Table1]. For the all branches the technique used is the domain-bifurcation of Quarteroni [3], the boundary conditions of the arterial segments joining at junctions are prescribed by enforcing conservation of mass and continuity of the total pressure $P + \frac{1}{2}\rho\left(\frac{Q}{A}\right)$.



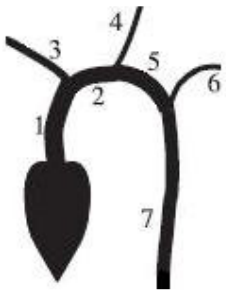




Figure 1: Schematic representation of the arterial network simulated, which includes the aorta, brachial, carotid, femoral and the main thoracic arteries (left). Numbers refer to names of the main arteries in the Table 1 of the area of study.

### 2.3 Numerical Scheme.

Eqs.(1-2-3) are solved using a second-order discontinuous Taylor-Galerkin spatial-discretisation scheme and a four-order explicit Runge-Kutta time-integration scheme, with a adaptive stepsize control. This scheme is suitable for the 1-D formulation because it can propagate waves of different frequencies without suffering from excessive dispersion and diffusion errors.The Taylor-Galerkin requires a time step limitation in order to keep the solution stable, the stabilization adopted is the method CFL (Courant-Friedrichs-Levy)[4]

$$\Delta t \leq \mathrm{CFL} \min_{0 \leq i \leq N} \left[ \frac{l_i}{\max(\lambda_{1,i}, \lambda_{2,i})} \right] \qquad \text{Eq.5}$$

where the $\lambda_{1,i}$ means the eigenvalue $\lambda_1$ at the mesh in the node i and CFL=0.577[4]

### 2.4 Physiological Data.

The physiological parameters used in each arterial segment are given in the Table1. Our model aims at representing the arterial geometry and elasticity of a healthy young adult. In the absence of detailed knowledge of these properties, the geometry and wall elasticity of the arteries are based on data published in Wang and Parker [2].

| | Name | L (cm) | r (cm) | h (cm) | E ($10^6$Pa) | c (m/s) | $R_t$($10^9$Pa·s/m$^3$) |
|---|---|---|---|---|---|---|---|
| **1** | **Ascending Aorta** | 4.0 | 1.470 | 0.163 | 0.4 | 4.67 | |
| **2** | **Aortic Arch I** | 2.0 | 1.263 | 0.126 | 0.4 | 4.43 | |
| **3** | **Brachiocephalic** | 3.4 | 0.541 | 0.067 | 0.4 | 4.93 | |
| **4** | **Left. Ext Carotid** | 17.7 | 0.334 | 0.042 | 0.8 | 7.02 | 13.9 |
| **5** | **Aortic Arch II** | 3.9 | 1.195 | 0.115 | 0.4 | 4.35 | |
| **6** | **Left Subclavian I** | 3.4 | 0.474 | 0.066 | 0.4 | 5.23 | 6.01 |
| **7** | **Thoracic Aorta** | 5.2 | 1.120 | 0.110 | 0.4 | 4.39 | |

Table 1: Physiological data used in the model

## 3.VALIDATION OF RESULTS.

We have presented a 1-D model of pulse wave propagation that is able to capture the main wave propagation features observed in vivo in the aorta and the arteries supplying them from the aorta. From the origin of the aorta, Fig 1 (Left) position A, our model predicts a continuous blood flow towards the arch aortic, Fig 1 (Left) positions B,C,D during all the cardiac cycles, which is in agreement with in vivo measurements (Fig 2),using terminal and no terminal resistance. In order to validate the flow distribution between the different arteries, we also compare the flow simulated with physiological data obtained a correct distribution. Table[2] (1/3 of the volume flow is coming out from the heart to the upper-aortic circulation).

| | Vessel name | Q(ml/sec) | % of the total Flow |
|---|---|---|---|
| 3 | Brachiocephalic | 105 | |
| 4 | Left. Ext Carotid | 26 | |
| 6 | Left Subclavian I | 40 | |
| | **Total Upper-Aortic** | 171 | 34% |
| 7 | Thoracic Aorta | 350 | 66% |
| | **Total Flow** | 521 | |

Table 2: Mean volume rates (in ml/sec) at the outlet of the arteries studied.

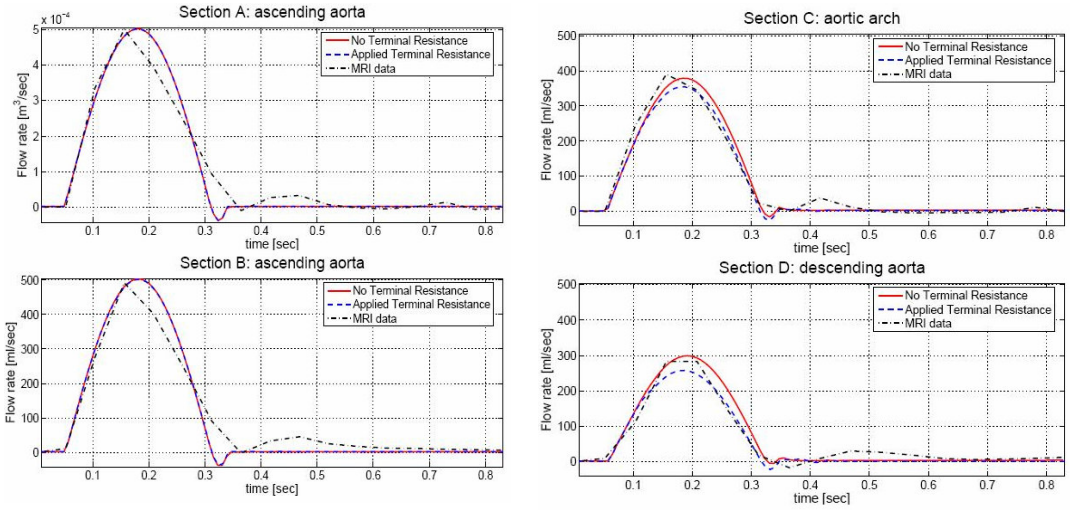



Figure 2: Flow velocity over one cardiac cycle in points A,B,C,D to the figure1.

## 4.CONCLUSION.

The possibility of developing fast and powerful one-dimensional models to understand perfectly how the flow patterns and distributions is propagated throughout all the cardiovascular system within a prescribed geometry, can be useful to set both inflow and outflow boundary conditions for 3D finite elements problems and could be helpful for the early detection, diagnosis and prevention of related arterial diseases.

## 5.ACKNOWLEDGEMENTS

The authors would like to acknowledge Dr. Francesç Carreras for his contribution, and the Hospital de la Santa Creu i Sant Pau for the support gives us to do this work.

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